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Interpersonal Trauma Exposure and Cognitive Development in Children to Age 8 Years: A Longitudinal Study

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Abstract

Background—Childhood trauma exposure has been associated with deficits in cognitive functioning. The influence of timing of exposure on the magnitude and persistence of deficits is not well understood. The impact of exposure in early development has been especially under-investigated. This study examined the impact of interpersonal trauma exposure (IPT) in the first years of life on childhood cognitive functioning.

Methods—Children (N= 206) participating in a longitudinal birth cohort study were assessed prospectively for exposure to IPT (physical or emotional abuse or neglect, sexual abuse, witnessing maternal partner violence) between birth and 64 months. Child intelligent quotient scores (IQ) were assessed at 24, 64, and 96 months of age. Race/ethnicity, gender, socioeconomic status, maternal IQ, birth complications, birthweight, and cognitive stimulation in the home were also assessed.

Results—IPT was significantly associated with decreased cognitive scores at all time points, even after controlling for sociodemographic factors, maternal IQ, birth complications, birthweight, and cognitive stimulation in the home. IPT in the first two years appeared to be especially detrimental. On average, compared to children not exposed to IPT in the first two years, exposed children scored one-half standard deviation lower across cognitive assessments.

Conclusion—IPT in early life may have adverse effects on cognitive development. IPT during the first two years may have particular impact, with effects persisting at least into later childhood.

Keywords

cognitive development; IQ; trauma; child abuse; domestic violence

Competing Interest: None declared.

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¹As previously described,[12] investigators determined that verbal abuse was the most subjective form of maltreatment and had high overlap with other maltreatment types (especially physical abuse); also, no standardized assessment procedure was available to code verbal abuse at 64 months as had been available at 24 months. Therefore, verbal abuse was not coded after 24 months.

Childhood exposure to traumatic events has significant effects on long-term cognitive development, as evidenced by negative associations with intelligence quotient (IQ) scores, language development, and academic achievement.[1,2, 3] The impact of timing of exposure is not well understood, though current knowledge regarding brain development suggests that the type, magnitude, and persistence of effects depends on when in development exposure occurs.[1,4] In early development, particularly from birth to age two years, the brain undergoes rapid growth and reorganization, a process heavily influenced by environmental factors.[5, 6] Structural and functional reorganization that occurs during this sensitive period may become permanent, influencing subsequent development, even after environmental conditions change. Therefore, early childhood trauma may have considerable and enduring effects on cognitive development, though empirical evidence in this area is needed.[1]

The goal of the current study was to examine the impact of a specific type of early trauma exposure, interpersonal trauma (IPT) involving the primary caregiver, on child cognitive outcomes. IPT, including maltreatment and interparental violence, is a particularly potent stressor for young children, given the critical role of the attachment relationship in shaping the developing nervous system.[7] Furthermore, maltreatment and interparental violence are often chronic events.[1] Though previous research has associated such exposures with impairments in cognitive domains (e.g., IQ, executive functioning, reading and math abilities)[1, 2, 3, 8, 9, 10] and brain structure (e.g., reduced volumes of the cerebral cortex, hippocampus, and corpus callosum) and functioning (e.g., event-related potential and electroencephalography abnormalities),[1, 4, 6] the immediate and long-term effects of such exposures specifically in the first years of life have not been well researched.

The current study addresses this gap by examining the impact of IPT exposure from birth to age 5 on longitudinal assessments of cognitive functioning in a prospective birth cohort sample. Analyses distinguished between exposures from 0 to 24 months and from 24 to 64 months and controlled for several potential confounders, including sociodemographic factors, maternal IQ, birth complications, birthweight, and quality of cognitive stimulation in the home.

METHODS

Participants

Participants (N = 206) were from the Minnesota Longitudinal Study of Parents and Children (MLSPC), a prospective examination of adaptation in low-income families.[11] Englishspeaking pregnant women were recruited during the third trimester from the Minneapolis Department of Public Health Clinic and the Hennepin County General Hospital between 1975 and 1977. Mothers were eligible if the pregnancy was their first (primiparous) and if they qualified for public assistance for prenatal care and delivery (i.e., their income was below the official poverty line); 267 women consented and were enrolled. An additional 147 mothers were approached but declined to participate (mother's partner refused to allow participation, too busy) or were unable to participate (planned to move, delivered prior to prenatal assessment, infant adopted out, infant died at delivery, mother did not speak English fluently enough to complete study measures). There were no significant differences between families who did and did not consent to study participation on maternal age, education, occupation, or clinic staff assessment of family risk. All procedures were approved by the Institutional Review Board of the University of Minnesota. Mothers provided written informed consent. Participant attrition from 267 to 206 occurred largely during the first two years of the study. There were no differences between enrolled families who did and did not complete the current study activities on maternal marital status, education level, age, or socioeconomic status (SES) at the child's birth or child race/

ethnicity, gender, or birthweight. Table 1 summarizes participant demographic characteristics.

Procedures and Measures

Interpersonal Trauma Exposure (IPT)—IPT events included experiencing child maltreatment and witnessing partner violence against the mother. Two dichotomous (yes/no) scores were derived based on timing of IPT exposure: exposed in infancy (0-24 months); exposed in preschool (24-64 months). All IPT assessments were made blind to the cognitive results.

<u>Child maltreatment:</u> Child maltreatment was identified prospectively based on the following: home observations at 7-10 days, 3, 6, and 9 months and twice at 12 months; laboratory observations at 9, 12 (two visits), 18, 24, and 42 months; maternal interviews throughout assessment periods; and reviews of medical records and child protection records at 24 and 64 months. Children were classified as maltreated between 0 and 24 months if there was evidence of any of the following: (a) physical abuse, defined as parental acts resulting in physical damage (e.g., bruises, cuts, burns); (b) psychological maltreatment, defined as verbal abuse (e.g., constant harassment or berating, chronically finding fault, harsh criticism) or psychological unavailability (e.g., interacting only as necessary, emotional unresponsiveness); or (c) neglect, defined as incompetent and irresponsible management of the child's day-to-day care, inadequate nutritional or health care, or dangerous home environment due to insufficient supervision. Between 24 and 64 months, sexual abuse was added as a maltreatment category and was defined as genital contact between the child and a person 5 years older than the child (all perpetrators were adolescents or adults).[12] Psychological unavailability but not verbal abuse was rated as a form of psychological maltreatment due to conceptual and methodological difficulties¹ in assessing verbal abuse during this time period.[12] Project staff conferenced and classified each child into the above categories, reaching near perfect agreement regarding classification. Validation for the identification of maltreatment cases has been previously reported.[12, 13]

Exposure to maternal partner violence: Child exposure to partner violence against the mother was based on maternal interviews and questionnaires and interviewer observations of the families at 12, 18, 24, 30, 42, 48, 54, and 64 months.[14] Inter-rater reliability was calculated at each time point on the basis of 50 ratings completed by two developmental psychology graduate students (*rs*=.93-.99). Children exposed to severe maternal partner violence were classified as IPT-exposed, given prior research showing a similar magnitude of IQ effects among children exposed to high levels of domestic violence as among maltreated children.[10]

Potential Confounders

Sociodemographics: Factors previously associated with cognitive development[15, 16] were considered as potential confounders: child gender; child race/ethnicity; SES, assessed during pregnancy and when the child was 42 and 96 months old. SES was based on the mean standardized scores from at least two of three sources: the revised Duncan Socioeconomic Index household score,[17, 18] maternal education, and household income. Greater scores indicate higher SES.

Birth-related factors: A variable reflecting the presence or absence of birth complications was based on data extracted from labor and delivery records. Presence of any of the following problems were coded as positive for a birth complication: maternal heart massage required; Rh-/fetal blood incompatibility; fetal tachycardia, bradycardia, asphyxia, or

Maternal IQ: Maternal IQ was estimated by summing scores on the Comprehension, Similarities, and Block Design subscales of the Wechsler Adult Intelligence Scale (WAIS), [19, 20] ascertained when the child was 48 months of age. The measure is a reasonable estimate of maternal IQ throughout the child's life, given the stability of IQ in adulthood[21] and the high reliability, validity, and stability of the WAIS.[22]

fluid retained in the lungs); gestational age < 37 weeks or 42 weeks. Birthweight was also

extracted from medical records.

Quality of cognitive stimulation: The Home Observation for Measurement of the Environment (HOME) was administered at 30 months to assess the support available to promote the child's social and cognitive development. The scale has demonstrated moderate stability across time, [23] and prior research has documented associations between child IQ and HOME subscales. [24] A cognitive stimulation scale (HOME) was created by summing standardized scores from the subscales focused on cognitive support, including organization of the physical and temporal environment, provision of appropriate play materials, and opportunity for variety and daily stimulation. Higher scores indicate greater stimulation.

Cognitive Outcomes—The cognitive battery conforms to recent suggestions for assessing cognitive trajectories in longitudinal studies of children.[25]

Bayley Mental Development Scale (BMD): At 24 months, children were administered the Bayley Scales of Infant Development, [26] which provide standardized scores (M= 100, SD = 15) on scales of infant mental and motor development. For these analyses, the mental development scale (BMD) was used, which assesses the following: sensory/perceptual acuities, discriminations, and responses; acquisition of object constancy; memory learning and problem solving; vocalization and beginning of verbal communication; basis of abstract thinking; habituation; mental mapping; complex language; mathematical concept formation.

Wechsler Preschool and Primary Scale of Intelligence (WPPSI): At 64 months, children were administered a short form of the WPPSI,[27] including the Vocabulary, Block Design, and Animal House subtests. Prorated IQ scores (M = 100, SD = 15) were derived using Sattler's formula.[20] The short form has high reliability and validity and correlates highly with full-scale IQ scores.[20, 24]

<u>Wechsler Intelligence Scale for Children-Revised (WISC-R)</u>: At 96 months, children were administered a short form of the WISC-R,[28] including the Vocabulary, Similarities, and Block Design subtests.[20] Prorated IQ scores (M = 100, SD = 15) were derived using Sattler's formula.[20] The short form has high reliability and validity and correlates highly with full-scale IQ scores.[20, 24]

Data Analytic Plan

Differences among children never exposed to IPT, children exposed to IPT in infancy only, children exposed to IPT in preschool only, and children exposed to IPT in both infancy and preschool on cognitive scores were examined separately at 24, 64, and 96 months via one-way ANOVAs followed by pairwise *t*-tests if the overall *F* test was significant. Associations among the study variables were tested in bivariate correlational analyses. To test whether IPT exposure was associated with cognitive scores from 24 to 96 months, mixed effects

models with a random intercept were implemented. These models accounted for repeated cognition measures within participants, took advantage of the longitudinal nature of the data, increased statistical power (when compared to analyzing separately at each time point), and enhanced possible inferences regarding associations between IPT exposure and cognitive functioning over time. Longitudinal models were possible given that all of the cognitive measures are standardized to have the same mean (M=100) and standard deviation (SD=15). An indicator variable was included in the model that allowed IPT in preschool to affect cognitive outcomes only at 64 and 96 months. This ensured that IPT in preschool could not affect 24-month cognitive outcomes.

In the first step of the model, a 3-way interaction term among IPT in infancy, IPT in preschool, and time of cognitive testing (i.e., 24, 64, or 96 months) and all possible 2-way interaction and main effect terms were included. The 3-way interaction term tested whether the effect of IPT in infancy on cognitive scores differed over time depending on the presence or absence of IPT exposure in preschool (IPT in infancy*time*IPT in preschool). Once found non-significant, the 3-way interaction was removed, and the remaining 2-way interactions were tested. The two-way interactions tested (a) whether the effect of IPT in one time period (infancy or preschool) on cognitive scores differed by whether IPT in the other time period (preschool or infancy) was experienced (IPT in infancy*IPT in preschool), (b) whether the effect of IPT in infancy on cognitive scores changed over time (IPT in infancy*time), and (c) whether the effect of IPT in preschool on cognitive scores changed over time (IPT in preschool*time). Any non-significant two-way interaction terms were removed from the model. In the final step, covariates were added to test the effects of IPT exposure independent of other known risk factors, including child gender, race, SES, maternal IQ, birthweight, birth complications, and cognitive stimulation in the home. SES was included as a time-varying covariate in the model, with SES during pregnancy used for predicting 24-month cognitive scores, SES at 42 months for predicting 64-month cognitive scores, and SES at 96 months for predicting 96-month cognitive scores. In addition, a main effect term for time was included to test whether there were differences in mean cognitive scores over time. For all analyses, a *p*-value < 0.05 was considered statistically significant.

A set of multiple regression analyses were also run using comparable models to predict cognitive scores separately at each age (24, 64, and 96 months). Results from these analyses produced similar conclusions and are available on-line.

Data Imputation—Participants were included in the analyses if they had data for at least one of the cognitive outcomes; 199 completed the BMD, 174 completed the WPPSI, and 177 completed the WISC-R. Missing data in the predictor, covariate, and outcome variables were imputed using the Markov Chain Monte Carlo method[29] of multiple imputation, implemented in SAS PROC MI, which produces unbiased results if the data are missing at random. Analyses were conducted on the multiple imputed datasets and then summarized for inference purposes according to the rules developed by Rubin,[30, 31] implemented in SAS PROC MIANALYZE. One hundred thirty-one participants had complete data for all variables; 75 had missing data for at least one variable. Ten datasets were imputed, each with 206 observations. Results from the imputed datasets did not differ qualitatively from results from the complete-case dataset, and both sets produced similar conclusions. Results based on imputed data are presented.

RESULTS

By 64 months, 36.5% of the sample had experienced IPT, with 4.8% exposed in infancy only, 13.0% in preschool only, and 18.7% in both infancy and preschool. Table 2 displays mean cognitive test scores by IPT exposure. ANOVAs showed that IPT groups differed

significantly on BMD scores at 24 months (p = .0003), WPPSI scores at 64 months (p < .0001) and WISC-R scores at 96 months (p = .0006). Follow-up pair-wise *t*-tests revealed that children exposed to IPT in infancy only and children exposed in infancy + preschool had lower BMD and WPPSI scores than children exposed in preschool only and unexposed children. Children exposed to IPT in infancy + preschool had significantly lower WISC-R scores than unexposed children. The remaining pairwise comparisons were not significantly different. Table 3 presents the bivariate correlation coefficients among the study variables.

In the mixed effects models predicting cognitive scores from 24 to 96 months, none of the interaction terms were significant, signifying that (a) IPT exposure in infancy had a similar magnitude of effect on cognitive scores at 24, 64, and 96 months (non-significant IPT in infancy*time); (b) IPT exposure in preschool had a similar magnitude of effect on cognitive scores at 64 and 96 months (non-significant IPT in preschool*time); (c) the impact of IPT exposure in one time period (infancy or preschool) on cognitive scores did not vary by the presence/absence of IPT exposure in the other time period (non-significant IPT in infancy*IPT in preschool); and (d) the impact of IPT exposure in infancy on cognitive scores over time did not vary by the presence/absence of IPT exposure in preschool (nonsignificant IPT in infancy*time*IPT in preschool). Therefore, the interaction terms were removed from the final model, summarized in Table 4. In this model, IPT exposure in infancy but not in preschool was significantly associated with cognitive outcomes. Specifically, those with IPT exposure in infancy had cognitive scores that were on average 7.25 points lower than those without exposure in infancy. Additionally, male gender, lower birthweight, lower maternal IQ, and lesser cognitive stimulation in the home predicted lower cognitive scores. The time main effect term was not significant, indicating similar mean scores on the cognitive tests over time.

DISCUSSION

This is the first study to examine prospectively the impact of early IPT—specifically maltreatment or witnessing maternal partner violence—on cognitive functioning from infancy through the early school years with repeated assessments on the same children. The results suggest that IPT in early childhood, particularly during the first two years, has significant and enduring effects on cognitive development, even after adjusting for gender, race/ethnicity, SES, maternal IQ, birth complications, birthweight, and quality of cognitive stimulation in the home. These findings are consistent with other studies documenting vulnerability in the early years to fundamental changes in neural circuitry and brain structure related to social adversity and trauma.[1,6] In contrast to many studies in this area, subjects were drawn from the community rather than mental health clinics or domestic violence shelters or via child protection records, thereby broadening the generalizability of the findings.

Several hypotheses have been proposed to account for the impact of trauma on childhood cognitive development. Certain forms of maltreatment may cause direct injury to the brain, such as physical abuse involving the head or neglect resulting in malnutrition. Other forms of IPT that do not involve frank neurological injury (e.g., sexual abuse, witnessing domestic violence) have also been associated with adverse cognitive development, leading some to hypothesize that trauma affects cognitive outcomes through stress pathways.[1, 10] Extreme stress has been associated with enduring changes in the secretion and processing of numerous stress hormones and neurotransmitters.[1, 8] These responses may be associated with altered neural structure and functioning, particularly in the early years when the brain is undergoing its most rapid phase of growth, differentiation, and synaptic organization.[1] Because early brain organization frames later neurological development, changes in early development may have lifelong consequences.

The significant association between IPT and child cognitive outcomes may reflect the role of the parent-child relationship in promoting child cognitive development. Child maltreatment represents the most extreme form of poor caregiving, and maltreating parents demonstrate a host of characteristics predictive of various negative child outcomes.[3] Exposure to partner violence may also compromise mothers' caregiving abilities.[32] Poorer quality parent-child interactions, in turn, have been associated with depressed child cognitive development.[24, 33]

Trauma-induced psychological symptoms (e.g., posttraumatic stress disorder [PTSD]) may contribute to cognitive deficits by impeding the ability to engage the environment effectively and learn new skills.[3] Some studies have found that neural/cognitive effects from trauma exposure are evident only among participants with PTSD, whereas others have documented effects regardless of psychiatric status.[6, 8, 9] Conversely, low IQ by age 5 has been identified as a risk factor for the development of PTSD.[34] Therefore, there are likely complex, multi-directional, transactional associations over time among emotional/behavioral symptomatology, cognitive functioning, and brain development that shape children's responses to IPT. Future studies may consider whether psychological disorders such as PTSD mediate or moderate the effects of early trauma exposure on cognitive development.

Of note, short forms of the WPPSI and WISC-R were used, a validated method in developmental research and the recommended approach for assessing child IQ in longitudinal studies.[25] However, trauma exposure may have different effects on the various domains of cognitive functioning, including domains not assessed with the short forms. Therefore, future research should consider use of more comprehensive IQ batteries to examine whether subscales are differentially affected by early trauma exposure.

Limitations

A significant limitation of the current study is the relatively small sample size, particularly the small number of children exposed to IPT in infancy only. Because the large majority of children exposed to IPT in infancy were also exposed in the preschool period, the power to distinguish timing effects from chronicity effects was limited. Previous studies have demonstrated that longer exposure to maltreatment is associated with smaller intracranial volume and greater IQ suppression, though length of exposure is often confounded with age of onset.[6] That IPT exposure in infancy but not in the preschool period emerged as a significant predictor and that exposure in infancy had similar effects on cognitive scores across development and regardless of preschool exposure suggests that the first two years of life may be a period of particular vulnerability to negative, enduring effects of trauma exposure on cognitive development.

Changes to the maltreatment coding scheme after 24 months may have resulted in an underidentification of maltreatment cases during both time periods. Notably, among the identified cases of verbal abuse and sexual abuse, 79% and 64%, respectively, experienced at least one other form of maltreatment. Therefore, the majority of unidentified verbal and sexual abuse cases were likely identified as IPT-exposed for other forms of maltreatment. Given the frequency of overlap among different IPT exposures, examining cognition effects by exposure type was not possible.

Other factors not assessed within the current study may, in part, account for associations between IPT and child cognitive functioning, including maternal prenatal substance use (e.g., alcohol, tobacco, narcotics) and child lead exposure. The current analyses adjusted for birth complications and low birthweight, pathways through which prenatal substance use may affect cognitive outcomes.[35, 36] Analyses also adjusted for SES, which is strongly

associated with lead exposure.[37] Because participants were all low SES, the results may not generalize to higher SES populations.

Conclusion

Each year in the United States, there are approximately 750,000 validated cases of child maltreatment,[38] and 3 to 10 million children witness domestic violence.[39] Children under the age of 4 are most likely to be victimized/exposed, with infants from birth to 1 year especially vulnerable.[38] The current findings suggest that the first years of life is a period of heightened sensitivity to substantial and enduring cognitive effects from such exposures. Depressed cognitive functioning in early development has been shown to result in long-term damaging consequences, including poor academic performance throughout schooling and poor adjustment throughout life.[1, 40] These findings highlight the importance of identifying at-risk families and preventing IPT in early life to promote positive cognitive development throughout childhood.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Table 1

Characteristics of Mother and Child Participants at Child's Birth (N= 206)

Variable	%	Mean	Standard Deviation	Range
Maternal Age		20.67 years	3.78 years	12 - 34 years
Maternal Education				
Less than high school	37%			
High school graduate	40%			
Some college education or greater	23%			
Maternal Marital Status				
Single/Separated/Divorced/Widowed	65%			
Married	35%			
Child Gender, Male	56%			
Birth Complications, Any	30%			
Child Birthweight		3262 grams	545 grams	1580 -4400 grams
Child Race/Ethnicity				
White, Non-Hispanic	65.5%			
Multiracial	17%			
Black	12%			
Native American	4%			
Asian	0.5%			
Hispanic	1%			

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	BN	BMD, 24 Months	MPPS	WPPSI, 64 Months	WISC-	WISC-R, 96 Months
	Mean	Mean 95% CI	Mean	Mean 95% CI Mean 95% CI	Mean	95% CI
No exposure	103.73	103.73 100.44, 107.02 108.34 105.90, 110.77 107.08 104.22, 109.94	108.34	105.90, 110.77	107.08	104.22, 109.94
Exposure infancy ^a only	86.62	74.71, 98.52	90.89	81.00, 100.78 95.91 78.27, 113.55	95.91	78.27, 113.55
Exposure preschool ^b only	102.12	94.73, 109.50	104.66	104.66 99.00, 110.32 101.11 94.76, 107.47	101.11	94.76, 107.47
Exposure infancy ^{<i>a</i>} + preschool b 90.25 85.50, 94.99 95.44 90.79, 100.09 93.89	⁵ 90.25	85.50, 94.99	95.44	90.79, 100.09	93.89	87.94, 99.83

b Preschool = 24-64 months.

Table 3

Correlation Coefficients Among Study Variables

Variable	1	7	e	4	n	9	2	×	6	AT	Π	12	13
1. IPT, 0-24 months	1												
2. IPT, 24-64 months	.59 ***	ł											
3. Child gender ^a	10	03	I										
4. Child race/ethnicity b	.14	60:	05	ł									
5. SES, prenatal	20 **	17	07	15*	1								
6. SES, 42 months	16*	25 **	08	04	.67 ***	ł							
7. SES, 96 months	21 **	27 **	.03	12	.55 ***	*** 69.	I						
8. Maternal IQ (WAIS)	17*	26 **	09	15*	.47 ***	.54 ***	.50 ***	I					
9. Birth complications	07	08	05	00 [.]	.14 *	.13	.12	03	I				
10. Child birthweight	02	.03	.03	03	10	06	03	60.	24 ^{**}	ł			
11. HOME, 30 months	37 ***	34 ***	.03	37 ***	.24 ***	.29 ***	.39 ***	.30***	.05	07	I		
12. BMD, 24 months	31 ***	19 **	.19 ^{**}	18**	.19 **	.20 **	.28 ***	.27 ***	06	.21 ^{**}	.35 ***	1	
13. WPPSI, 64 months	-38 ***	26 ***	.15*	16^{*}	.35 ***	.40 ***	.38 ***	.46***	.05	$.16^*$.37 ***	.55 ***	1
14. WISC-R, 96 months	30 ***	27 ***	.04	17*	.27 ***	.29***	.41 ***	.47 ***	.02	.20**	.33 ***	.52 ***	.72***

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exposure; IQ = intelligence ool and Primary Scale of

 $^{a}Male$ children were coded "1"; female children were coded "2."

bWhite, Non-Hispanic children were coded "1"; racial/ethnic minority children were coded "2."

p < 0.05.

p < 0.01.p < 0.01.p < 0.001.

Table 4

Associations between Interpersonal Trauma Exposure and Child Cognitive Scores from 24 to 96 Months: Final Mixed Effects Model

Variable	Parameter Estimate	Standard Error	<i>p</i> -Value
IPT exposure, 0-24 months	-7.25	2.30	0.002
IPT exposure, 24-64 months	-0.37	1.82	0.84
Time ^{<i>a</i>}	-0.04	0.04	0.31
Gender ^b	4.85	1.63	0.003
Race/ethnicity ^C	-0.39	1.81	0.83
SES ^d	0.17	0.07	0.02
Maternal IQ (WAIS)	0.52	0.12	< 0.0001
Birthweight	0.01	0.002	0.0007
Birth complications	0.23	1.78	0.90
Cognitive stimulation in the home	1.27	0.43	0.003

Notes. IPT = interpersonal trauma exposure; IQ = intelligence quotient; SES = socioeconomic status; WAIS = Wechsler Adult Intelligence Scale.

 a The time term tested whether there were differences in mean cognitive scores over time.

 b Males are the reference group.

 $^{\ensuremath{\mathcal{C}}}$ White, Non-Hispanic children are the reference group.

 $d_{\text{SES}}^{}$ during pregnancy was used for predicting 24-month cognitive scores, SES at 42 months for predicting 64-month cognitive scores, and SES at 96 months for predicting 96-month cognitive scores.