Early Adversity, Socioemotional Development, and Stress in Urban 1-Year-Old Children

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Objective To determine demographic, maternal, and child factors associated with socioemotional (SE) problems and chronic stress in 1-year-old children.

Study design This was a prospective, longitudinal, community-based study, which followed mother-infant dyads (n = 1070; representative of race, education, and income status of Memphis/Shelby County, Tennessee) from midgestation into early childhood. Child SE development was measured using the Brief Infant-Toddler Social and Emotional Assessment in all 1097 1-year-olds. Chronic stress was assessed by hair cortisol in a subsample of 1-year-olds (n = 297). Multivariate regression models were developed to predict SE problems and hair cortisol levels.

Results More black mothers than white mothers reported SE problems in their 1-year-olds (32.9% vs 10.2%; $P < .001$). In multivariate regression, SE problems in blacks were predicted by lower maternal education, greater parenting stress and maternal psychological distress, and higher cyclothymic personality score. In whites, predictors of SE problems were Medicaid insurance, higher maternal depression score at 1 year, greater parenting stress and maternal psychological distress, higher dysthymic personality score, and male sex. SE problem scores were associated with higher hair cortisol levels ($P = .01$). Blacks had higher hair cortisol levels than whites ($P < .001$). In the entire subsample, increased hair cortisol levels were associated with higher parenting stress ($P = .001$), lower maternal depression score ($P = .01$), lower birth length ($P < .001$), and greater length at 1 year of age ($P = .003$).

Conclusion Differences in maternal education, insurance, mental health, and early stress may disrupt SE development in children. Complex relationships between hair cortisol level in 1-year-olds and maternal parenting stress and depression symptoms suggest dysregulation of the child's hypothalamic-pituitary-adrenal axis. *(J Pediatr 2013;163:1733-39)*.

Child socioemotional (SE) development is defined as "the developing capacity of the child from birth through 5 years of age to form close and secure adult and peer relationships; experience, regulate, and express emotions in socially and culturally appropriate ways; and explore the environment and learn—all in the context of family, community, and culture."[1] Complex interactions of biological, environmental, social, cultural, and community factors operating from preconception through childhood shape neurodevelopment, and thereby SE development, in early life.[2] SE competence facilitates cognitive development and school readiness,[3] improved school performance,[4] and subsequent lifespan outcomes, such as adult health[5] and employment success.[6]

Socioeconomic variables, such as family income and wealth, parent education and occupation,[7] and related factors such as home and neighborhood[8] influence child SE development. Maternal depression or anxiety are associated with diminished SE competence in children.[9-11] Stressful life events, such as intimate partner violence or chronic stress related to poverty or neighborhood crime and instability, can diminish a parent's ability to interact and nurture the developing child.[10,12,13]

The physiological changes involved in the response to recurrent stressors alter the regulation of the hypothalamic-pituitary-adrenal (HPA) axis, often referred to as "allostatic load."[14] HPA axis responses in early life are particularly detrimental because of the wide-ranging effects during the sensitive periods of brain development, as well as the encoding of impaired ability to respond to future stressors.[15] Early adverse experiences lead to dysregulation of the HPA axis, characterized by exaggerated or ineffective responses, prolonged reactivity, and

BITSEA	Brief Infant-Toddler Social and Emotional Assessment
BSI	Brief Symptom Inventory
CANDLE	Conditions Affecting Neurocognitive Development and Learning in Early Childhood
EPDS	Edinburgh Postnatal Depression Scale
HPA	Hypothalamic-pituitary-adrenal
SE	Socioemotional
TEMPS	Temperament Evaluation of Memphis, Pisa, Paris, and San Diego

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delayed recovery after stress. Exposure to direct or indirect (ie, maternal) stressors in infancy seems to promote emotional or cognitive problems in toddlerhood,[16] later childhood,[17] and adolescence.[18] Although maternal depression,[17] poverty, and related stress are often associated with a dysregulated HPA axis in offspring,[19] how these factors affect early SE development is not clear. Chronic or cumulative stress can be measured by hair cortisol level, a better measure of chronic stress than cortisol level in plasma, saliva or urine.[20] We hypothesized that adverse maternal and socioeconomic risk factors may lead to cumulative stress in infancy, manifested in elevated hair cortisol levels,[21] which may influence early SE outcomes.

Methods

The Conditions Affecting Neurocognitive Development and Learning in Early Childhood (CANDLE) study has enrolled 1503 healthy 16- to 40-year-old women in their second trimester of pregnancy in Shelby County, Tennessee, and will follow them until their children reach age 54 months. Women were enrolled at an urban hospital obstetric clinic and at community obstetric practices. Exclusion criteria included existing chronic disease requiring medication (eg, hypertension, diabetes, sickle cell disease), known pregnancy complications (eg, complete placenta previa, oligohydramnios), and plans to deliver at a nonparticipating hospital. The **Figure** shows the participants available for this investigation. Informed consent was provided by the participants or their legally authorized representatives. A financial incentive of a $100 gift card was offered to each participant for each visit. The study was approved by the University of Tennessee Health Science Center's Institutional Review Board.

Demographic and Socioeconomic Measures

Participants provided information on health insurance, race, ethnicity, marital status, parity, and household composition via self-administered questionnaires. Prepregnancy height and weight were based on self-report and used to calculate prepregnancy body mass index as weight in kilograms divided by height in meters squared.

Of the 1390 participants scheduled for a 1 year follow-up examination, 1070 (77%) completed the examination (**Figure**). A smaller proportion of blacks than whites attended the follow-up examination (74% vs 84%). Compared with eligible black mothers who did not attend the examination, those who did so were older, more likely to have private insurance (27% vs 14%), and to have completed more than 12 years of education (37% vs 18%). Similarly, white mothers who attended the examination were older and more likely to have completed more than 12 years of education (74% vs 59%) compared with those who did not. The subsample of 297 children with hair cortisol level data were more likely to be female, on Medicaid, and from a 2-parent family ($P < .01$ for all comparisons) compared with the 773 children without hair cortisol data, but there were no differences between the 2 groups in maternal depression score at 12 months, maternal age, or maternal education.

Maternal Psychosocial Measures

The Brief Symptom Inventory (BSI),[22] a 53-item self-report scale measuring the nature and intensity of psychological symptoms, was completed by mothers during the third trimester of pregnancy and at the 1-year child visit. The BSI Global Severity Index T score served as an overall measure of the severity of psychological symptoms.

Maternal depression was screened using the 10-item Edinburgh Postnatal Depression Scale (EPDS)[23] at 4 weeks postpartum and the 1-year visit. Continuous EPDS scores were analyzed.

Maternal temperament was measured during gestation with the 84-item Temperament Evaluation of Memphis, Pisa, Paris, and San Diego (TEMPS)[24] to identify the following personality styles: hyperthymic (intrusive, cheerful, overoptimistic or exuberant, extroverted, overly talkative), irritable (angry, impulsive, snapping or cursing often, unpleasant), cyclothymic (lethargy alternating with activity, marked unevenness in quantity and quality of productivity, mental confusion alternating with sharp and creative thinking), or dysthymic (gloomy, pessimistic, incapable of fun, self-critical, self-derogatory, brooding, worried, feelings of inadequacy).[24] Continuous TEMPS scores were used for this analysis.

Mothers' reports of parenting stress were assessed at the 1-year visit using the 36-item Parenting Stress Index Short Form to identify parents at risk for dysfunctional parenting and parenting characteristics not promoting typical development in children.[25] Total parenting stress percentiles were used in this analysis.

The risk for physical child abuse was assessed at the 1-year visit using the 160-item Child Abuse Potential Inventory parent questionnaire. A score $\geq$166 was considered to indicate a mother at risk for abuse.[26]

Child Measures

Gestational age was determined by ultrasound or by the mother's report of the last menstrual cycle. Age- and sex-specific percentiles for weight, length, and head circumference were used in this analysis.

Cognitive development was assessed at 1 year using the Bayley Scales of Infant and Toddler Development, Third Edition screener.[27] Cognitive development was classified as at risk (<2nd percentile), emerging (2nd to <25th percentile), or competent (>25th percentile). Because only 12 of the 1-year-olds were deemed at risk, children with at-risk and emerging scores were combined in this analysis.

Maternal report of child SE problems was elicited at 1 year using the Brief Infant-Toddler Social and Emotional Assessment (BITSEA). A total BITSEA problem score $\leq$25th percentile for age was considered to indicate an SE problem.[28]

Hair Cortisol Measurement

Hair samples (5-50 mg) were collected from the posterior vertex of 1-year-olds; 1-3 cm hair samples were cut as close

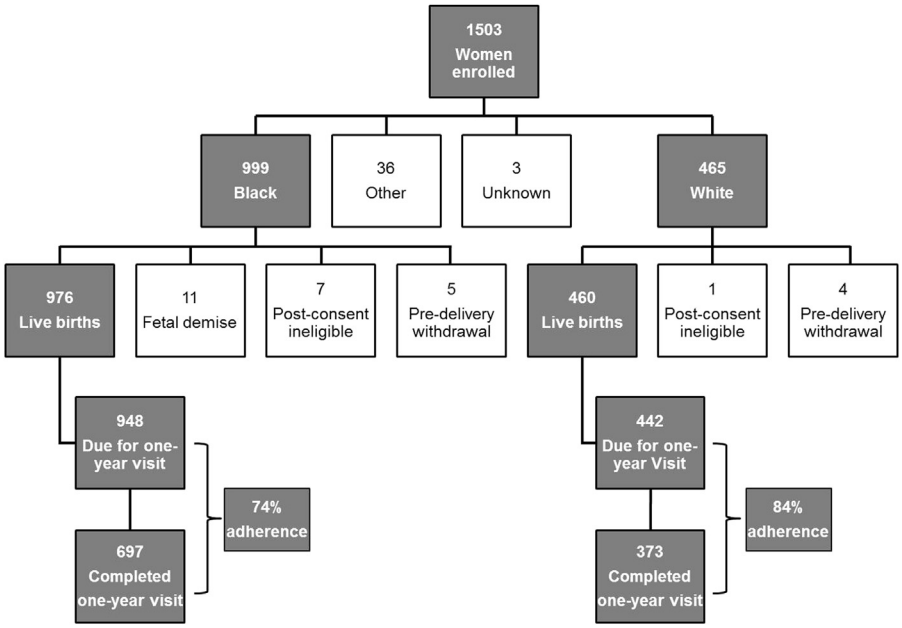

Figure. Flowchart of participants in the CANDLE study.

to the scalp as possible, sealed, and weighed. Each hair sample was minced and gently shaken in 1 mL of methanol overnight at 52°C; this procedure was repeated 2-3 times for each sample. Pellets from the samples were resuspended in 150 μL of phosphate-buffered saline. Cortisol was measured using an enzyme-linked immunosorbent assay kit (Alpco Diagnostics, Salem, New Hampshire) following the manufacturer's instructions. All calibrator, control, and test specimens were measured in duplicate. Assay plates were read using an Epoch plate reader (BioTek Instruments, Winooski, Vermont) set at 450 nm. Gen51.11 software (BioTek Instruments) was used to quantify cortisol expression in unknown specimens measured against a standard curve. Intra-assay and interassay coefficients of variability were <7% and <10%, respectively.

Statistical Analyses

We used descriptive statistics to summarize demographic, maternal, and child characteristics in the data by SE problem and hair cortisol quartiles in blacks and whites. The outcomes of children in quartile 1 (low cortisol) were compared with those in quartiles 2-4 (higher cortisol). The 2-sample *t* test or Wilcoxon rank-sum test was used to examine the differences in means or medians, respectively, for continuous variables in SE problem groups or hair cortisol quartile groups. The χ^2 test was used to examine race-specific associations between SE problems or cortisol quartiles and other categorical variables. The Spearman correlation coefficient was used to assess the relationship between hair cortisol level and other factors. A *P* value <.05 was considered statistically significant.

Multivariate logistic regression analyses were performed to identify the demographic, maternal, and child factors associated with SE problems and elevated hair cortisol level for the entire sample and for black and white children separately. Factors in the bivariate analyses with $P < .10$ were included in the regression models. Inclusion of a factor in the final model was based on backward elimination at $P < .05$. For each categorical variable, the OR indicated the likelihood of having SE problems for the given category relative to the reference category, controlling for other independent variables. The fit of the logistic model was assessed using the Hosmer-Lemeshow goodness-of-fit test[29] and the likelihood ratio test of the global null hypothesis that the model parameters were zero. Adjusted ORs and corresponding 95% CIs were reported. Analyses were performed using SAS version 9.3 (SAS Institute, Cary, North Carolina).

Results

Table I presents characteristics of the mothers and children by SE problem and race. Overall, 32.9% (229 of 697) of black mothers and 10.2% (38 of 373) of white mothers reported SE problems in their 1-year-olds ($P < .001$). The mothers reporting SE problems were younger, less educated, and more likely to have Medicaid insurance compared with those not reporting SE problems. The white mothers reporting SE problems in their children had a higher prepregnancy body mass index than those not reporting SE problems. In black children, those with SE problems had lower birth weight, birth length, and length at 1 year compared with those without SE problems. White children with SE problems were more likely than those without SE problems to have at-risk/emerging, but not competent, cognitive function.

Table II examines the relationships between maternal mental health variables and child SE problems by race. All maternal mental health variables in each race were related to SE problems ($P < .01$ to <.001). Multivariate models

Table I. Characteristics of CANDLE participants (n = 1070) by SE problems at age 1 year

Sociodemographic characteristic	Black, SE problems			White, SE problems		
	Yes (n = 229)	No (n = 468)	*P* value	Yes (n = 38)	No (n = 335)	*P* value
Maternal variables						
Age, years, median (IQR) (range, 16-40)	23 (8)	26 (9)	<.001	27 (8)	29 (7)	.03
Educational attainment, n (%)						
≤high school	171 (74.67)	269 (57.48)	<.001	15 (39.47)	82 (24.48)	.05
>high school	58 (25.33)	199 (42.52)		23 (60.53)	253 (75.52)	
Marital status, n (%)						
Single	142 (62.01)	269 (57.48)	.29	5 (13.16)	27 (8.06)	.35
Married or living with partner	87 (37.99)	199 (42.52)		33 (86.84)	308 (91.94)	
Health insurance status, n (%)						
Medicaid/TennCare	190 (82.97)	318 (67.95)	<.001	15 (39.47)	65 (19.40)	.007
Other (private, employer, military, none)	39 (17.03)	150 (32.05)		23 (60.53)	270 (80.60)	
Body mass index, median (IQR)	26.6 (10.5)	27.5 (10.9)	.16	26.2 (10.9)	23.9 (6.9)	.03
Birth outcomes						
Gestational age, wk, median (IQR)	38.53 (2.05)	38.82 (1.71)	.12	39.22 (1.30)	38.94 (1.41)	.12
Length, sex-specific percentile, median (IQR)	53 (49)	61 (44)	.005	76.5 (33)	72 (34)	.67
Weight, sex-specific percentile, median (IQR)	24 (29)	31 (38)	.002	48 (40)	50(41)	.98
Weight for length, sex-specific percentile, median (IQR)	30.87 (49.13)	28.45 (47.57)	.97	28.89 (44.13)	26.64 (46.17)	.87
Male sex, n (%)	129 (18.51)	231 (33.14)	.09	23 (6.17)	154 (41.29)	.12
Child variables at age 1 year						
Age, months, median (IQR)	12 (2)	12(1)	.08	12.5 (2)	12 (1)	.09
Length, sex-specific percentile, median (IQR)	64.07 (48.65)	71.37 (46.22)	.05	53.13 (49.70)	63.97 (50.28)	.42
Weight, sex-specific percentile, median (IQR)	53.80 (50.45)	55.37 (50.40)	.73	65.41 (31.20)	56.08 (51.36)	.32
Weight for length, sex-specific percentile, median (IQR)	68.30 (59.10)	62.71 (54.22)	.15	76.21 (41.10)	67.04 (45.94)	.07
Cognitive functioning, n (%)						
At risk/emerging	35 (5.03)	67 (9.63)	.20	13 (3.49)	40 (10.72)	<.001
Competent	194 (32.90)	400 (57.47)		25 (6.70)	295 (79.09)	

predicting the likelihood of child SE problems were developed using logistic regression analyses (Table II). Maternal education, parenting stress, maternal psychological distress, and cyclothymic score (TEMPS) were predictive of SE problems in black children. Predictors of SE problems in white children were Medicaid insurance, male sex, lower maternal depression score, greater parenting stress, maternal psychological distress, and dysthymic score.

The Hosmer-Lemeshow tests suggested that the data fit the models for blacks ($P = .23$), whites ($P = .92$), and the entire sample ($P = .49$). The overall logistic regression models had a likelihood ratio P value <.001 for each racial group and the entire sample.

Hair cortisol was measured in 297 children (175 black and 122 white). The median hair cortisol level was 13.5 ng/mg (IQR, 16.0 ng/mg) for all children, higher for blacks than for whites (median, 16.5 ng/mg [IQR, 14.6 ng/mg] vs 6.6 ng/mg [IQR, 11.3 ng/mg]; $P < .001$). Higher cortisol quartiles for the entire sample were associated with higher BITSEA Total Problem scores ($P = .01$), with no difference between the racial groups. Hair cortisol level was positively correlated with parenting stress ($r = 0.24$; $P = .008$) and maternal depression scores at 4 weeks ($r = 0.22$; $P = .02$) and 1 year ($r = 0.20$; $P = .03$) in whites, but only with the dysthymic score in blacks ($r = 0.19$; $P = .01$). Multivariate linear regression models predicting hair cortisol level (Table III) included variables with a significance of $P < .10$. Lower maternal depression score at 1 year ($P = .01$), greater parenting stress ($P = .001$), shorter birth length ($P < .001$), and greater height at 1 year ($P = .003$) were predictive of hair cortisol levels in the entire sample. Predictors of hair cortisol levels in blacks were lower maternal depression score ($P = .01$), greater parenting stress ($P = .03$), and lower birth length percentile ($P = .04$). Predictors in whites were greater maternal psychological distress ($P = .03$), greater parenting stress ($P = .004$), lower birth length percentile ($P < .001$), and greater length at 1 year ($P = .04$).

Discussion

Our data show a threefold difference in the prevalence of SE problems in black 1-year-olds compared with white 1-year-olds. Poorer SE development in black boys has been observed as early as 9 months of age, with differences extending through preschool age. Adjustment for socioeconomic and demographic variables reduced, but did not eliminate, these gaps.[30] In our study, parenting stress and maternal psychological distress were associated with SE problems in both black and white children. The prominent effects of cyclothymic score in blacks and the dysthymic score in whites also suggest differences in parental responses to the children and their behavior. The variable mood and energy displayed by black mothers with higher cyclothymic scores and the more gloomy, pessimistic mood seen in white mothers with higher dysthymic scores may influence the maternal–infant interactions necessary to support SE development by making interactions more unpredictable and/or less reassuring to the infant.

Higher maternal allostatic load,[14,31] as reflected in maternal psychological distress and parenting stress, was associated with increased risk for SE problems. This association suggests a possible mediating role of maternal stress on dysregulation of the infant's HPA axis. Studies using salivary cortisol

Table II. Maternal mental health characteristics and multivariate models predicting the likelihood of SE problems at age 1 year

Characteristic	Black (n = 697)			White (n = 373)		
	OR (95% CI)*	SD/unit	*P* value	OR (95% CI)*	SD/unit	*P* value
Maternal mental health characteristics						
EPDS						
4 weeks	1.59 (1.35-1.89)	4.31	<.001	1.98 (1.42-2.77)	3.46	<.001
1 year	1.81 (1.53-2.14)	4.26	<.001	1.80 (1.35-2.39)	3.88	<.001
TEMPS (scaled score)						
Cyclothymic	1.68 (1.43-1.98)	2.78	<.001	2.00 (1.54-2.60)	2.68	<.001
Dysthymic	1.32 (1.13-1.54)	1.29	<.001	1.67 (1.20-2.31)	1.43	.002
Hyperthymic	0.72 (0.61-0.84)	1.96	<.001	0.59 (0.43-0.80)	2.35	<.001
Irritable	1.48 (1.26-1.73)	1.54	<.001	1.80 (1.36-2.38)	1.28	<.001
Child Abuse Potential Inventory (reference normal)	3.30 (2.09-5.23)	1	<.001	4.54 (1.74-11.89)	1	.002
Parenting Stress Index	2.39 (2.00-2.84)	31.64	<.001	2.38 (1.67-3.38)	28.31	<.001
Global Severity Index	1.65 (1.38-1.96)	9.64	<.001	2.02 (1.39-2.93)	8.77	<.001
Multivariate models predicting likelihood of SE problems						
Insurance (reference other)	-	-	-	3.44 (1.27-9.37)	1	.002
Education at 1 year (reference >high school)	1.92 (1.24-2.97)	1	.003	-	-	-
Male (reference female)	-	-	-	3.55 (1.29-9.73)	1	.01
EPDS score at 1 year	-	-	-	0.53 (0.29-0.99)	3.86	.05
Parenting Stress Index	1.80 (1.44-2.25)	31.62	<.001	2.53 (1.51-4.24)	28.12	<.001
Global Severity Index	1.30 (1.02-1.65)	10.47	.04	3.61 (1.66-7.85)	10.00	.001
Cyclothymic TEMPS score	1.40 (1.13-1.74)	2.81	.002	-	-	-
Dysthymic TEMPS score	-	-	-	1.66 (1.04-2.66)	1.43	.04

"-" indicates nonsignificant variables that were not retained in the model.
*OR for a 1 SD increase in risk factor or for reference risk category. For example, in black mothers, for each SD unit increase (31.64) on the Parenting Stress Index, the OR for SE problems was 2.39 (95% CI, 2.00-2.84). Similar ORs are seen in white mothers.

measurements have reported infant and child cortisol reactivity and self-regulation in response to experimentally induced stressful events.[32] These studies and others support the concept that family context, parental mental health, and parent–infant interaction can alter the long-term regulation of the child's HPA axis.[17-19]

Hair cortisol level is a novel biomarker for chronic stress and may more accurately reflect allostatic load than measurements of salivary or plasma cortisol.[20] It has been validated as a measure of chronic stress in preschool[21] and elementary school children.[33] We found higher hair cortisol levels in black children, which may reflect their exposure to greater poverty, lower maternal education,[21] the neighborhood/home environment, or other factors leading to chronic stress in infancy. Higher basal salivary cortisol levels in black infants and preschoolers have been reported, even when controlling for other aspects of risk.[34] Abundant evidence links household and neighborhood poverty with an increased allostatic load in early childhood,[34] adolescence,[35] and adulthood.[36]

Shorter birth length, possibly reflecting maternal adversity during pregnancy, and parenting stress level at 1 year were associated with higher hair cortisol levels in the entire sample and in both races. This is a remarkable finding, suggestive of fetal programming of the HPA axis. It confirms the findings from animal studies and contributes to the glucocorticoid hypothesis for fetal origins of adult disease.[37] Prenatal maternal stress is known to enhance infant cortisol responses to acute stress just after birth,[38] but responses to chronic stress in infants have not been investigated previously. Similar responses, if occurring repeatedly during infancy,

Table III. Multivariate models predicting hair cortisol levels at age 1 year

Characteristic	Black (n = 175); R^2 = 0.0786			White (n = 122); R^2 = 0.2139			Entire sample (n = 297); R^2 = 0.1082		
	β estimate	SE	*P* value	β estimate	SE	*P* value	β estimate	SE	*P* value
Birth length percentile	−0.421	0.204	.04	−0.519	0.139	<.001	−0.475	0.126	<.001
Length at 1 year percentile	0.358	0.202	.08	0.258	0.123	.04	0.363	0.122	.003
Parenting Stress Index	0.435	0.203	.03	0.449	0.150	.004	0.440	0.135	.001
EPDS at 1 year	−3.611	1.450	.01	−2.500	1.428	.08	−2.493	0.966	.010
Global Severity Index	-	-	-	1.298	0.583	.03	-	-	-
Irritable TEMPS score	-	-	-	−5.951	3.247	.07	-	-	-
Possible SE problems	-	-	-	−26.077	14.851	.08	-	-	-
Cognitive risk at 1 year	-	-	-	-	-	-	18.544	10.470	.078

"-" indicates nonsignificant variables that were not retained in the model.
Based on bivariate analyses, variables entered in the initial model were maternal age, education, marital status, insurance status, temperament (cyclothymic, dysthymic, hyperthymic, and irritable scores), depression (EPDS score at 4 weeks postpartum and 1 year), Global Severity Index (maternal psychological distress on the BSI), total stress percentile on the Parenting Stress Index, and Child Abuse Potential Inventory at 1 year and the child's sex, birth weight and length (sex-specific percentiles); age at evaluation; weight, height, and weight-for-height at 1 year (*z*-scores for age and sex), cognitive risk at 1 year (Bayley Scales of Infant and Toddler Development, Third Edition screener); and SE problems (BITSEA).

would lead to higher hair cortisol levels and possibly impaired SE development in 1-year-olds.[39]

Maternal psychological distress as measured by the BSI was positively correlated with higher hair cortisol levels in white children, implying expected robust responses to ongoing stress transmitted from the mother. This association was not seen in black children, however, suggesting dampened HPA responses to maternal distress. Unexpectedly, a higher maternal depression score on the EPDS was associated with lower hair cortisol levels in their children ($P = .01$ in blacks; $P = .08$ in whites), further supporting a pattern of HPA dysregulation, at least in black children. Such a pattern is associated with increased risk for posttraumatic stress disorder, substance use, and other mental health problems,[40] possibly from "toxic stress" exposures in early life.[2,41] HPA dysregulation signals an increased allostatic load in black mothers and infants, possibly related to lower educational attainment, greater poverty, or single parent status. Urbanization further increases allostatic load.[42] Long-term implications of HPA dysregulation include not only recurring cycles of economic deprivation and poorer physical and mental health, but also genetic/epigenetic changes associated with degenerative, proinflammatory, and metabolic disorders.[43,44]

HPA dysregulation in black children also could result from variables not measured in the present study. The contribution of increased parenting stress or increased maternal psychological distress as a predictor of SE problems is higher in whites than in blacks. These differences suggest that other unmeasured factors (ie, neighborhood, family, biological, or cultural) may contribute to increased SE problems in black infants.[38] Research on the timing and severity of stressors and the effects of multiple stressors on infant/child HPA regulation is needed. Repeated assessments of HPA functioning are important and should include assessment of baseline HPA status as well as moment-to-moment fluctuations.[18]

Maintaining adequate follow-up of participants is important in cohort studies. This is especially challenging in studies of individuals at high social risk. The follow-up rate with complete data of the CANDLE study (77%) compares favorably with the complete data rates of other similar US cohort studies, including Project Viva (53% at 1 year)[45] and the Early Childhood Longitudinal Study–Birth Cohort (70.8% at 9 months).[46] Because mothers without follow-up were younger, less educated, and more likely to be on Medicaid, our present findings may underestimate the true prevalence of SE problems and allostatic load in this population. The subsample with hair cortisol data differed from the overall sample in those demographic factors found not to be significant predictors of hair cortisol level (more likely to be female, on Medicaid, and from a 2-parent household; **Table III**). These differences are unlikely to alter our conclusions.

SE problems were identified based on parental report on the BITSEA. The BITSEA Total Problem score is closely correlated with the Ages and Stages Social-Emotional Questionnaires total score ($r = 0.55$).[47] Although the BITSEA was developed using a representative sample and was clinically validated in a sample including minority and nonminority children,[48] it might not accurately reflect the parents and children residing in Memphis/Shelby County, Tennessee. The black mothers in our sample could be overreporting, and the white mothers could be underreporting. Direct observation of SE development using recently developed methods[49] would be helpful; however, the mother's perspective of the child, independent of observed behavior, is a key feature in parenting.

To limit the effects of known biological confounders in our analyses, our sample excluded mothers with many identifiable health problems before and during pregnancy that can potentially affect child development. Thus, this medically low-risk cohort does not allow for a comprehensive analysis of the effects of maternal health on child development.

The social and biological associations identified in this study should be applicable in settings of a similar demographic profile, especially in southern US cities with high levels of urban black poverty, and also in cities with similar demographics elsewhere. Consideration of community-specific, even neighborhood-specific, variables will be important in identifying critical factors influencing child development and related targets for intervention.

In conclusion, in this prospective cohort study, we found pronounced differences in the prevalence of SE problems in black 1-year-olds compared with white 1-year-olds, with corresponding differences in hair cortisol level and HPA axis regulation, possibly reflecting differential exposures to stress. Future investigations should examine the wide variety of potential factors contributing to SE problems as the child develops. These factors include personal stressors, such as job or housing loss, financial crises, home and household resources, family alcohol and substance use, potentially complex effects of public assistance programs and other social, cultural, and physiological variables. Similarly, positive factors associated with resiliency or potential buffering of family or parent–child interaction stressors merit examination. In this and other cohorts, advanced statistical methods, such as multilevel modeling, should be applied to quantify the complex factors contributing to SE development over time and to identify those factors that offer the greatest potential for intervention in specific infant populations.[41] Doing so could ultimately reduce the heavy burden of chronic diseases and mental health disorders in our urban populations. ■

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