ORIGINAL CONTRIBUTION

Social Fear in US Infants: The Roles of Hair and Salivary Cortisol

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Elevated social fear in infancy poses risk for later social maladjustment and psychopathology. Hair cortisol concentration (HCC), an index of cumulative cortisol exposure, and diurnal salivary cortisol slope, a biomarker of acute stress regulation, have been associated with social fear behaviors in childhood; however, no research has addressed their relations in infancy. Elucidating potential biomarkers of infant social fear behaviors, as well as environmental factors associated with these biomarkers, may grant insights into the ontogeny of fear behaviors that increase risk for internalizing and externalizing psychopathologies later in life. The current study used multiple linear regression to examine if infant HCC, infant diurnal cortisol slope, and income-to-needs ratios (ITN) were differentially associated with observed social fear responses to a Stranger Approach task at 12 months. Using a sample of 90 infants (M_{age} = 12.26m, SD = 0.81m, 50% female), results indicated that increased infant HCC was associated with increased distress vocalizations during the Stranger Approach task, while steeper diurnal cortisol slope was associated with fewer distress vocalizations. Ordinary least squares path analyses did not reveal group differences between economically strained and non-strained infants in how cortisol measures and social fear responses related. Findings underscore very early psychobiological correlates of fearfulness that may increase risk for fear-related disorders and adverse mental health symptomology across childhood.

INTRODUCTION

Early social fear reactivity shapes physiological, behavioral, and affective processes that contribute to physical and mental health long-term [1,2], particularly during infancy when humans rely on caregivers for protection from social and nonsocial threats [3]. However, while infant fear behaviors may serve to optimize survival potential [4], augmented or contextually inappropriate fear responses may devolve into dysregulated emotional reactivity that confers risk for socioemotional problems across development [5].

Heightened social fear responses in early life are linked to internalizing [3,6] and externalizing problems

in childhood [7], reduced social engagement [8], and increased attention bias towards threat [9]. These associations are likely related to the ecological importance of fear for infant survival, though they also reflect adverse outcomes that may stem from being overly or under sensitive to environmental threat cues [4]. Infant social fear is also tied to physiological regulation [10], which holds implications for social adjustment [11] and maladaptive patterns of psychobiological stress regulation later in life [6]. Importantly, increased social fear behaviors, rather than those in response to nonsocial threats, impede interpersonal bonding [12] and increase risk for social phobias and anxiety disorders [13]. Thus, analyzing the ways in which infants respond to fear-eliciting social stimuli may clarify early regulatory differences that hold implications for socioemotional development beyond the infant years. Additionally, considering the psychobiological mechanisms underlying infant fear behaviors, as well as the environmental factors that shape these mechanisms, may grant insight into the ontogeny of fear reactivity and factors that mark very early risk for maladaptive outcomes longitudinally.

The Development of Early Social Fear

Social fear responses emerge and evolve during the first year of life, becoming measurably pronounced at 6 months and increasing throughout the first postnatal year [14,15]. Evolutionarily, social fear holds adaptive significance for increasing individual fitness [16]. Although general temperamental fearfulness has some foundations in genetic heritability [17], social fearfulness may also be a product of experience [16]. Studies have linked early adversity, such as lower socioeconomic status (SES), to increased distress to novelty in infancy [18], which predicts social anxiety later in life [19]. Additionally, lower family income and reduced parental education are associated with difficulty disengaging from fearful faces in 9- to 12-month-old infants [20]. These relations suggest that contextual variation may play a role in shaping infant social fearfulness through persistent environmental exposure to psychosocial stress, which may be related to the evolutionary significance of learned fear responses to threat more generally.

Social fear behaviors increase from 6 to 36 months [15] and demonstrate relatively stable trajectories across the toddlerhood [21,22] and childhood years [23]. These early trends have proven to be important markers of individual differences in fear responses that predict socioemotional outcomes [6]. For example, infants who demonstrate steeper increases in stranger-related social fear behaviors from 6 to 36 months are at increased risk for anxiety problems and social phobias at age 8 [6]. Further, parent-reported infant fearfulness is correlated with

childhood internalizing symptoms, such that heightened fearfulness in the first year of life is associated with increased risk for depression and anxiety at age 4 to 8 years [7]. High-intensity fear behaviors at 15 months have also been linked to behavior problems in first grade [24], and elevated toddler fear responses such as facial fear, freezing, and seeking proximity to caregivers during social fear paradigms predict more socially anxious behaviors in kindergarten [25].

One common social fear paradigm is the Stranger Approach task, a procedure used in the infant version of the Laboratory Temperament Assessment Battery (Lab-TAB) [26]. In this paradigm, an unfamiliar adult engages with the infant in a series of epochs that evoke emotional responses to being approached and handled by a stranger [26]. The Stranger Approach paradigm reliably elicits social fear behaviors in infants as young as 6 months [26], which are indexed through observed affective responses across epochs [27]. The Stranger Approach paradigm has not only been linked to longitudinal differences in internalizing problems and social inhibition [6,28], but also has been studied in relation to acute stress reactivity and regulation [29]. While the Stranger Approach task does not appear to elicit immediate changes in biological stress reactivity in infants [29], this paradigm has been linked to broader indices of physiological regulation [6]. Specifically, higher levels of fear during the Stranger Approach in infancy and toddlerhood predict dysregulation in diurnal stress regulation in middle childhood, such as lower waking cortisol levels at age 6 [30] and flatter diurnal cortisol slopes at age 8 [6]. Thus, examining variable measures of psychobiological processes underlying observable responses to the Stranger Approach is important for understanding very early relations between types of physiological regulation and social fear behaviors that may compromise socioemotional adjustment over time.

Cortisol

The hypothalamic-pituitary-adrenocortical (HPA) axis, a biological stress system that governs fear reactivity, is vulnerable to contextual influences in infancy [31,32]. The HPA axis produces cortisol, a hormone that indexes biological stress exposure and regulation [33]. Chronic exposure to environmental stressors, also known as allostatic load, results in HPA overactivation and cortisol overproduction, saturating the infant's internal regulatory systems [34,35]. Over time, prolonged periods of augmented cortisol levels result in dysregulation of homeostatic psychobiological systems [31,34], such as elevated or blunted patterns of cortisol production [36] and HPA hyporeactivity later in life [37,38]. Additionally, chronically high levels of circulating cortisol lead to greater activation of glucocorticoid receptors and miner-

alocorticoid receptors in the amygdala, which over time may shape the development of amygdala function, increasing amygdala excitability and synaptic connectivity [39]. In this way, chronic biological stress exposure can become ingrained in regulatory responses to environmental threats, social encounters, and adjustment to novelty, often with long-term ramifications for socioemotional and physical health [31]. Alternatively, these associations may be bidirectional, with contextually augmented or inappropriate behavioral responses to threat contributing to HPA dysregulation over time [40].

Cortisol can be used to assess both acute and chronic psychobiological stress exposure. Daily cortisol production and regulation follows a circadian rhythm, in which salivary cortisol levels are high in the morning and decrease throughout the day [41]. Infants are born with no diurnal cortisol rhythm [41], though evidence suggests that rhythmic patterns begin to emerge as young as 2 months of age [42]. By 8 months, infant diurnal cortisol rhythms represent stable patterns of daily cortisol regulation that resemble those of an adult [42]. Daily cortisol rhythms are often quantified through calculating diurnal cortisol slope from morning to evening, with steeper slopes indicating better cortisol regulation through sharper decreases throughout the day [43]. In contrast, flatter diurnal cortisol slopes may reflect HPA dysregulation and an inability to adequately regulate cortisol in tandem with typical circadian patterns [34].

Another method of measuring cortisol is hair cortisol concentration (HCC), or accumulated deposits of cortisol in human hair [44]. HCC is a particularly useful biomarker of chronic stress, as it grants longitudinal insights into individual differences in psychobiological stress experiences through cortisol build-up over time [45]. HCC offers several advantages over salivary measures of cortisol, as it is less susceptible to daily emotional lability and acts as a cumulative measure of cortisol that is not interrupted by intermittent sampling periods [46]. Although higher HCC has been associated with daily levels of cortisol exposure, HCC and diurnal salivary cortisol slope are uncorrelated [47]. HCC and diurnal slope are also influenced by different environmental factors, suggesting that the psychobiological mechanisms underlying these measures are distinct [47]. Thus, assessing HCC and diurnal cortisol slope concurrently may support a more comprehensive approach to understanding the dynamics between acute and chronic biological stress, the early experiences that shape them, and resulting behavioral outcomes that may prove detrimental to physical and mental health throughout childhood.

In childhood, as in adulthood [44], increased HCC suggests greater persisting psychobiological stress [46]. HCC can be assessed in infants as young as 30 days old [48], with longitudinal evidence suggesting that increased HCC is a reliable biomarker of augmented psychobiological stress exposure across development [49]. Increased infant HCC is associated with prolonged exposure to distressing environments [48] and socioeconomic adversity [47,50,51]. While these findings suggest that context and experience contribute to chronic cortisol exposure across infancy, research examining HCC and its associations with infant behavioral development is scarce. This gap limits empirical understanding of the role of HCC in infant functioning and socioemotional wellbeing, which is important for elucidating the ways in which psychobiological stress may contribute to developmental psychopathologies and fear-related disorders.

Cortisol and Social Fear

Cortisol and social fear have been associated through multiple metrics in childhood [45,52,53]. Increased social fear, measured through parent reports, is related to heightened salivary cortisol concentration in 2-year-olds [52]. Elevated cortisol reactivity during social stressors is also linked to longitudinal increases in social anxiety across childhood, particularly when children have parents who also demonstrate high stress-reactivity [53]. Further, increased childhood HCC is associated with greater temperamental fearfulness, suggesting that chronic biological stress may factor into early social and nonsocial fearfulness [45].

Infant research with cortisol and social fear is less prevalent. Existing studies on these relations often measure acute cortisol reactivity in response to a social stressor, with most finding null results [29]. Although this method targets immediate physiological responses, measuring the change in cortisol levels before and after a stressor does not give insights into daily cortisol rhythms [33]. That is, while cortisol reactivity is one measure of acute psychobiological stress that does not appear to be related to infant fear, diurnal slope allows for a broader view of daily regulatory patterns that may grant alternative insights into whether and how infants are equipped to manage social stressors [33]. However, to our knowledge, only a few studies have examined diurnal salivary cortisol and social fear in infancy [6,10]. For example, Watamura and colleagues showed that flatter diurnal cortisol slopes among infants (M_{age} = 10.4 months) and toddlers (M_{age} = 28.8 months) in childcare are associated with increased teacher-reported social fearfulness [10]. Van Hulle and colleagues also showed that steeper increases in infant observable social fear responses between the ages of 6 and 36 months predict flatter diurnal cortisol slope at age 8 [6]. While these studies suggest associations between diurnal cortisol slope and infant social fear, more research is necessary to enhance our understanding of the mechanisms underlying these links.

Other measures of cortisol, such as HCC, may help

to clarify relations between psychobiological stress and social fear in infancy. Infant research with nonhuman primates has shown that increased HCC is associated with reduced social affiliation in infant rhesus monkeys [54]. However, no research has extended analyses of HCC to social fear behaviors in human infants. Considering that HCC and diurnal cortisol slope are uncorrelated [47], but each may contribute to early social fear behaviors [10,54], examining HCC and diurnal slope together may offer a more exhaustive examination of early biomarkers of social fear. Further, extending animal models linking HCC and social fear to human infants is an important step for delineating whether and how chronic psychobiological stress and fearfulness share relations in young humans.

Environmental factors that may play a role in infant regulation and fear behaviors are also important to consider with regards to infant fear development. SES is one measure of early life stress that has been linked to infant diurnal salivary cortisol [55], infant HCC [47], and fear in children and adolescents [56]. Lower income is associated with fearful temperament [57], decreased sociability [58], and flatter infant diurnal cortisol slopes [59]. Twelve-month-old infants from poorer socioeconomic homes also demonstrate higher HCC than their higher SES counterparts regardless of diurnal salivary cortisol slope [47]. Furthermore, early chronic exposure to poverty and lower SES predict flatter diurnal salivary cortisol slopes in young adulthood [43], indicating that these regulatory differences may become ingrained into long-term physiological health and regulatory processes.

Synchrony between parent and child cortisol across levels of socioeconomic strain suggests heritability in psychobiological regulatory capacities [60,61]. Socioeconomically disadvantaged children whose parents have heightened HCC are also more likely to have higher HCC [61]. Similarly, low-income infants of parents with higher diurnal cortisol output are more likely to have higher salivary cortisol output themselves [55], and a recent genetic study indicated that genetic factors account for approximately half the variability in HCC [62]. However, parent-child concordance may also indicate that shared environmental factors, like SES, shape caregiver biological stress levels in addition to those of the developing infant [47,63,64]. Alternatively, these links may be a result of maternal responses to contextual adversity that then shape infant regulation [63,64]. Increased maternal psychosocial stress during pregnancy is linked to greater infant social fearfulness between the ages of 14 and 19 months [65]. Additionally, interventions targeting foster parent-reported stress result in better foster child diurnal cortisol regulation longitudinally [66]. Thus, parental stress levels may be another contextual factor shaping early stress regulation and fear responses. However, gaps in the literature persist in that no studies have examined socioeconomic variability in relation to parent and infant measures of cortisol and infant behavioral social fear concurrently during the first postnatal year.

The first aim of the current study was to examine whether and how infant diurnal salivary cortisol slope, infant HCC, and parent HCC related to social fear responses in 12-month-old infants. We did this by using a behavioral Stranger Approach paradigm and multiple measures of parent and infant cortisol to assess the relations between acute and chronic stress and fear responses to social novelty. We used multiple linear regressions to examine the unique associations between cortisol and infant fear behaviors at 12 months. Our second aim was to test if economic strain moderated the relations between cortisol measures and infant fear responses. Considering the importance of socioeconomic variability for both HCC and diurnal slope [47], as well as noted differences in fearfulness for lower income children [57], our exploration of economic strain as a moderator aimed to unveil if observed relations between cortisol and social fear responses were due, in part, to socioeconomic disadvantage.

Based on previous literature [10,45,64], we hypothesized that flatter infant diurnal slope, increased infant HCC, and increased parent HCC would be associated with increased infant social fear responses during the Stranger Approach paradigm. Additionally, based on studies that have revealed socioeconomic differences in childhood fear responses [56], as well as infant and parent cortisol measures [47], we expected that economic strain would moderate the relations between infant cortisol measures and infant fear behaviors. Specifically, we hypothesized that flatter infant diurnal slope and increased infant HCC would be associated with more fear responses for infants from lower income homes. Findings highlight early links between biological stress and social fear behaviors in infancy that offer promising avenues for future research addressing risk for maladaptive mental health outcomes in childhood.

MATERIALS AND METHODS

Participants

A total of 90 infants (M_{age} = 12.26m, SD = 0.81m, 50% female) were recruited as part of the Stress Hormone Influences on Early Learning and Development Study, a cross-sectional study recruiting 12-month-old infants and their families in the Greater Boston Metropolitan Area. Inclusionary criteria at the time of recruitment included infants without any known neurological, developmental, or physical disorders. Participants were recruited using a departmental participant database, community recruitment events, hospital birth records, and online advertisements, resulting in a racially and socioeconomi-

Table 1. Demographic Information

Child Sex	
Male	N = 45 (50%)
Female	N = 45 (50%)
Child age (months)	
M (SD)	12.26 (0.81)
Income-to-Needs Ratio	
M (SD)	4.09 (2.89)
Child Ethnicity	
Non-Hispanic White	51.1%
Hispanic	21.1%
African American	12.2%
Multiracial	11.1%
Asian American	7.8%

cally diverse sample in which 51.1% of parents identified their child as Non-Hispanic White, 21.1% identified as Hispanic, 12.2% identified as African American, 11.1% identified as Multiracial, and 7.8% identified as Asian American. Economic strain was defined as income-to-needs ratios (ITN) calculated in relation to the cost of living in Boston, Massachusetts, resulting in 42.4% of families being defined as lower income for falling below 3.00 times the federal poverty designation for household size ($M = 4.09$, $SD = 2.89$). Demographic information is shown in Table 1.

Procedure

Families were recruited when infants were 12 months old to participate in a lab visit lasting 1.5 hours at Boston University. Following informed consent, parents completed questionnaires on infant hair habits and family demographic information. Parents were then trained in home saliva collection procedures and hair samples were collected from both parents and infants. Next, infants participated in an observational social fear paradigm designed to elicit emotional responses to interactions with an unfamiliar adult. All study procedures, compensation, and participant travel reimbursements were approved by the Institutional Review Board at Boston University.

Measures

Infant Social Fear. Infant social fear behaviors were assessed at the in-lab visit using the Lab-TAB Stranger Approach paradigm, a commonly used measure of infant fear behaviors validated for use with infants as young as 6 months old [26]. The Stranger Approach paradigm was completed in a series of epochs. First, infants were strapped in a highchair while their parents were seated behind them out of view. When infants were calm, an un-

familiar female research assistant entered the room and paused at the door, approximately four meters from the infant (10s). The research assistant then approached two meters closer to the infant and addressed him/her with a neutral expression, verbalizing that she was about to approach the infant and lift him/her from the highchair. The research assistant then walked within 0.3 meters from the infant (10s) and paused while standing over the infant (10s) before kneeling in front of the highchair, unstrapping the infant, lifting him/her, and holding him/her for 10 seconds. Infants were then replaced in the highchair and the research assistant left the room. Total task duration across all epochs lasted 65 seconds. In accordance with previous literature [27], trained research assistants coded participant videos for intensity of negative affective responses, including escape behavior and distress vocalizations. Intensity was determined as the greatest observed emotional response in each epoch, resulting in six intensity scores per behavior per infant. Intensity of escape behavior was coded on a four-point scale (0 = no escape behavior or looking to parent for guidance; 1 = mild fleeting escape behavior, turn towards parent, gaze aversion; 2 = moderate escape behavior, twisting or leaning away; 3 = vigorously trying to escape lasting more than three consecutive seconds, pushing or hitting stranger). Intensity of distress vocalizations was coded on a six-point scale (0 = no distress; 1 = mild sound that does not indicate interest/happiness; 2 = whimpering for 1-2 seconds; 3 = longer whining, fussing, mild protest, or low intensity cry; 4 = non-muted crying without screaming; 5 = full intensity cry or scream). Twenty percent of cases were double coded to guard against drift in research assistant coding consistency. Both distress vocalizations (ICC(3,4) = 0.691, $p < .001$) and escape behavior (ICC(3,4) = 0.692, $p < .001$) returned acceptable internal reliabilities at 95% confidence. Composite intensity scores were calculated as aggregate means of response intensity across task epochs ($M_{\text{Escape Behavior}} = 0.703$, $SD_{\text{Escape Behavior}} = 0.470$, $M_{\text{Distress}} = 0.537$, $SD_{\text{Distress}} = 0.707$). In total, 16 research assistants were used as strangers. All research assistants acting as strangers were thoroughly trained in Stranger Approach protocols, including maintaining neutral verbal and facial affect during the task. No differences in infant escape behavior (F(15) = 1.46, *ns*) nor distress vocalizations (F(15) = .598, *ns*) were found across strangers.

Infant Diurnal Cortisol Slope. Parents collected saliva samples from their infants at home at three time points over 3 consecutive days in which infants followed their normal schedule and were not sick, resulting in a total of nine saliva samples per infant. Sampling dates were scheduled during the in-lab visit. If infants became sick over the course of the predetermined sampling dates, saliva sampling was delayed to a week after the onset of illness. Saliva samples were collected using Salimetrics

infant swabs [67] that were placed in the infant's mouth for 60 seconds immediately after waking, early afternoon (at least an hour past midday feeding or before midday feeding), and at bedtime (before final feeding). Parents were given all materials to collect each sample, were trained during the in-lab visit, and were provided with written instructions to follow at home. Parents also recorded information about sampling times, feeding times, infant sleep schedules, co-sleeping, and other factors that may interfere with salivary cortisol levels. Home saliva samples were collected within 3-87 days after the in-lab visit (M_{Days} = 13, SD = 13.69) and within a week of data completion, with longer post-lab visit delays resulting from infant sickness, parent work obligations, unanticipated life events, or parents forgetting to complete saliva collection. Parents were instructed to store samples in the back of their freezer until pickup, and samples were transported to the lab in freezer bags to avoid thaw. Collected samples were stored in freezer bags at -20°C in lab freezers at Boston University to remain intact until being sent to Trier Laboratories in Germany for cortisol assay. Samples were shipped to Germany in two batches, with the longest storage time for any given sample being 528 days. Given that sleep patterns may impact diurnal cortisol [68], cortisol values at waking were log-transformed and corrected for time-since-wake, calculated from an actigraphy measure of waking and MEMS cap time stamps, prior to analyses. Slope steepness was calculated as the difference in salivary cortisol concentration from waking to bedtime as an index of diurnal cortisol regulation across the day. Infant salivary cortisol data was excluded if parents reported infant feeding within 60 minutes of cortisol collection to control for the confounding influence of feeding on salivary cortisol concentration [69].

Infant and Parent HCC. Hair samples were collected from both infants and parents during the in-lab visit by a trained research assistant. Hair samples were taken from the 3 centimeters closest to the scalp at the posterior vertex of the head to represent cumulative cortisol output over several months prior to sampling [70]. Hair samples weighed between 15 and 30mg. Parent reports of hair hygiene habits were also collected for both parents and infants due to the confounding influence of hair washing and other factors promoting the loss of accumulated cortisol [71,72]. Collected hair samples were stored in freezer bags in lab freezers at -20°C until processed for cortisol analysis as described in Meyer et al. (2014) with minor modifications [73]. Briefly, hair samples were weighed, washed twice with isopropanol to remove contaminants, dried, and ground into powder using a bead mill. Cortisol was then extracted into methanol, reconstituted in assay buffer, and spin-filtered to remove any residual particulate material. Reconstituted extracts were assessed for cortisol levels in duplicate along with standards and quality con-

trols using a selective and sensitive immunoassay (Arbor Assays DetextX Cortisol ELISA kit, Ann Arbor, MI), and HCC was calculated by converting assay readout to pg cortisol per mg of dry hair weight. Raw infant cortisol levels and raw parental cortisol levels were natural log-transformed to account for nonnormal distributions. Both intra- and inter-assay coefficients of variation were less than 10%. Of the 90 participating infants, four infants did not have usable cortisol data. Of the remaining 86 infants, four had biologically implausible HCC (> 1,500 pg/mg), one did not provide a hair sample but did provide saliva samples, one used topical scalp cream on the day of hair sampling, and two used steroid medications within 3 months prior to the in-lab visit. Infant HCC was not related to hair washing frequency, and thus correcting for hair washing habits was not necessary (r = 0.085, *ns*). Of the 90 participating parents, three did not have usable HCC data. Parent HCC was not related to hair washing frequency (r = -0.100, *ns*), hair straightening (r = 0.031, *ns*), hair coloring (r = .122, *ns*), or application of topical scalp cream (r = 0.015, *ns*) and thus correcting for these hair habits was not necessary.

Additional Covariates. Additional potential covariates were selected *a priori* to test for possible confounding influences of demographic variables and parent HCC on infant cortisol measures and social fear behaviors. Specifically, we tested if infant age, sex, race (ie, Hispanic, Non-Hispanic White, Asian American, African American), ITN, and parent HCC were correlated with infant HCC, infant diurnal slope, infant escape behavior, and infant distress vocalizations. Each racial group was defined as a dichotomous dummy variable (0 = non-identification, 1 = racial group identification), with Multiracial infants included as racial non-identification for all dichotomous racial group variables. Included covariates were defined according to results from bivariate correlations and independent samples t-tests and are included in the results.

Data Analytic Plan

Zero-order correlations between parent and infant HCC and measures of hair habits were assessed to determine potential confounding influences on hair cortisol measures. Next, we examined zero-order correlations and t-tests between potential covariates (ie, racial group membership, ITN, infant age, infant sex, parent HCC) and infant HCC, infant diurnal cortisol slope, and infant fear responses. Covariates were defined as measures that were significantly related to cortisol measures or fear outcomes and were included in subsequent models containing those variables of interest to control for confounding influence over model results.

After establishing appropriate model covariates, multiple linear regressions were run in IBM SPSS Sta-

tistics for Windows, Version 27.0 [74] to examine the relations between cortisol and infant social fear. Infant average distress vocalizations and average intensity of escape behavior were regressed onto infant HCC, infant diurnal cortisol slope, and relevant covariates in two separate models. To control for overlap in social fear behaviors, each outcome was included as a covariate in the regression predicting the other fear behavior.

To examine the role of economic strain in the relations of infant cortisol measures to infant fear responses, moderation analyses were conducted in IBM SPSS Statistics for Windows, Version 27.0 [74] using ordinary least squares path analysis [75], where significant effects were based on 5,000 samples and estimated using bias-corrected bootstrap confidence intervals at the 95% level. Interactions were tested in independent models. For moderation analyses, the continuous ITN variable was divided into a dichotomous variable (0 = economically strained, 1 = not economically strained) at 3.00 times the federal poverty designation for household size to examine the moderating influence of economic strain specifically rather than a continuous measure of poverty. This allowed for targeted assessments of grouped differences between economically strained and non-strained infants. All direct effect and interaction models were sufficiently powered using G*Power [76].

RESULTS

Preliminary Analyses. Relevant covariates were defined as demographic variables that related to cortisol variables or fear outcomes. Bivariate correlations to establish relevant covariates revealed that infant HCC was negatively correlated with ITN ($p = .012$) and Non-Hispanic White group membership ($p = .02$), such that higher infant HCC was associated with lower ITN and reduced likelihood of identifying as Non-Hispanic White. Infant HCC was positively correlated with parent HCC ($p < .001$) such that increased infant HCC was associated with increased parent HCC. Parent HCC was also positively correlated with escape behavior ($p = .015$), such that increased parent HCC was associated with increased intensity of infant escape behavior during the Stranger Approach. Average distress vocalizations were positively correlated with Asian American group membership ($p = .004$) such that Asian American infants were more likely to produce distress vocalizations. Infant age, African American group membership, and Hispanic group membership were not correlated with any predictor or outcome, and additional t-tests examining infant sex as a potential covariate were not significant. Thus, ITN, Asian American group membership, Non-Hispanic White group membership, and parent HCC were retained as covariates in subsequent analytic models, while infant age, infant

Table 2. Zero-order Correlations Between Model Predictors and Fear Outcomes

	1	2	3	4	5	6	7	8	9
ITN	-								
African American	-.31**	-							
Asian American	.12	-.11	-						
Non-Hispanic White	.48**	-.38**	-.30**	-					
Hispanic	-.44**	.06	-.15	-.53**	-				
Infant HCC	-.28*	.18	.09	-.26*	.13	-			
Infant diurnal slope	.02	.23*	-.10	-.01	.05	0.05	-		
Average escape intensity	-.21	.11	.12	-.09	.00	0.17	-0.12	-	
Average distress vocalizations	-.07	-.01	.32**	-.09	-.07	0.29*	-0.31*	0.50**	-
N	85	90	90	90	90	83	75	83	83
Mean	4.09	0.12	0.08	0.51	0.21	3.81	0.00	0.70	0.54
Standard Deviation	2.89	0.33	0.27	0.50	0.41	1.21	0.99	0.47	0.71

[a]HCC = Hair Cortisol Concentration [b]ITN = Income-to-needs ratio [1]**Bold text** indicates significant correlations; $p \leq .05^*$, $p \leq .01^{**}$
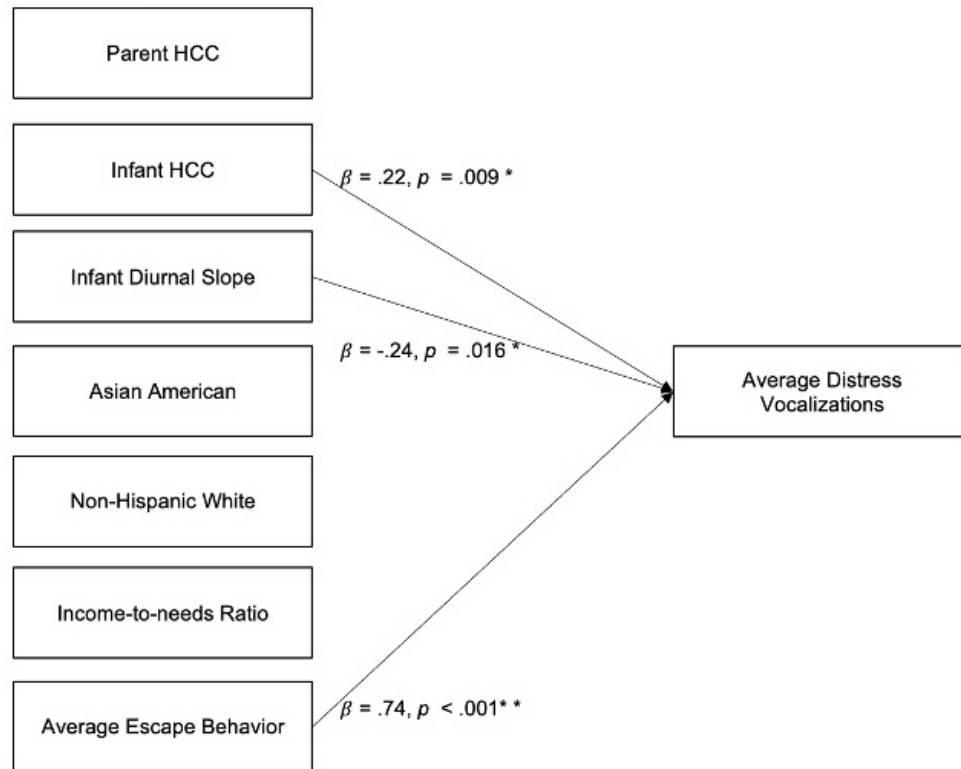
Figure 1. **Direct effects of cortisol measures and covariates on infant average distress vocalizations during the Stranger Approach Paradigm**. $p \leq .05^*$, $p \leq .01^{**}$, $p \leq .001^{***}$. Multiple linear regression and standardized estimates of the relations between parent HCC, infant HCC, infant diurnal cortisol slope, and relevant covariates and infant average distress vocalizations during the Stranger Approach Paradigm. 47.3% of the variance in infant distress vocalizations was explained by this regression model.

sex, African American group membership, and Hispanic group membership were dropped from further analyses given their lack of significance with cortisol measures or outcomes.

Table 2 shows additional bivariate correlations between infant cortisol measures and fear outcomes. Infant HCC was positively correlated with average distress vocalizations during the Stranger Approach task ($p = .011$), such that increased infant HCC was associated with more distress vocalizations. Further, infant diurnal cortisol slope was negatively correlated with average distress vocalizations ($p = .011$) such that steeper diurnal cortisol slope was associated with fewer distress vocalizations.

Cortisol measures associated with social fear. Direct effects of model variables on infant distress vocalizations were assessed by regressing average distress vocalizations on infant HCC, infant diurnal slope, and relevant covariates. Results from the distress vocalizations multiple linear regression indicated that 47.3% of the variance in distress vocalizations was explained by our model ($F(7, 51) = 6.53$, $p < .001$). Results revealed main effects of infant HCC and infant diurnal slope on average distress vocalizations. Specifically, distress vocalizations were positively associated with infant HCC ($B = 0.22$, $\beta = 0.30$,

$p = 0.009$) and negatively associated with infant diurnal slope ($B = -0.24$, $\beta = -0.27$, $p = 0.016$), such that increased HCC and flatter diurnal slope were associated with more distress. Additionally, average escape behavior was positively associated with distress vocalizations, such that more escape behavior was associated with more distress vocalizations ($B = 0.74$, $\beta = 0.44$, $p < 0.001$). Significant pathways associated with average distress vocalizations are depicted in Figure 1.

Our second model examined direct effects of model variables on average escape behaviors. Results from the escape behavior multiple linear regression indicated that 35.7% of the variance in escape behavior was explained by our model ($F(7, 51) = 4.05$, $p = .001$). Results revealed a main effect of distress vocalizations on escape behavior, such that more distress vocalizations were associated with more escape behaviors ($B = 0.32$, $\beta = 0.54$, $p < 0.001$). No other main effects were found in the associations between average infant escape behavior and covariates or variables of interest. All significant and nonsignificant direct effects for both regression models are shown in Table 3. Significant pathways associated with average escape behaviors are depicted in Figure 2.

Table 3. Direct Associations Between Cortisol Measures and Infant Social Fear

	Average Distress Vocalizations					Average Escape Intensity				
Direct Effects	B	SE	β	p	CI (95%)	B	SE	β	p	CI (95%)
Covariates										
ITN	.02	.03	.06	.64	(-.05, .08)	-.03	.02	-.15	.26	(-.07, .02)
Asian American	.46	.32	.18	.16	(-.19, 1.10)	.06	.22	.04	.78	(-.37, .49)
Non-Hispanic White	-.07	.21	-.04	.76	(-.49, .36)	.15	.14	.16	.27	(-.12, .43)
Parent HCC	-.19	.14	-.16	.16	(-.47, .08)	.16	.09	.22	.08	(-.02, .33)
Main Effects										
Infant HCC	**.22**	**.08**	**.30**	**.009**	**(.06, .38)**	-.03	.02	-.15	.59	(-.15, .08)
Infant diurnal slope	**-.24**	**.10**	**-.27**	**.016**	**(-.43, -.05)**	.00	.07	.00	.98	(-.13, .14)

[a]*HCC* = Hair Cortisol Concentration; [b]*ITN* = Income-to-needs Ratio; [1]**Bold text** indicates significant pathways in the regression model
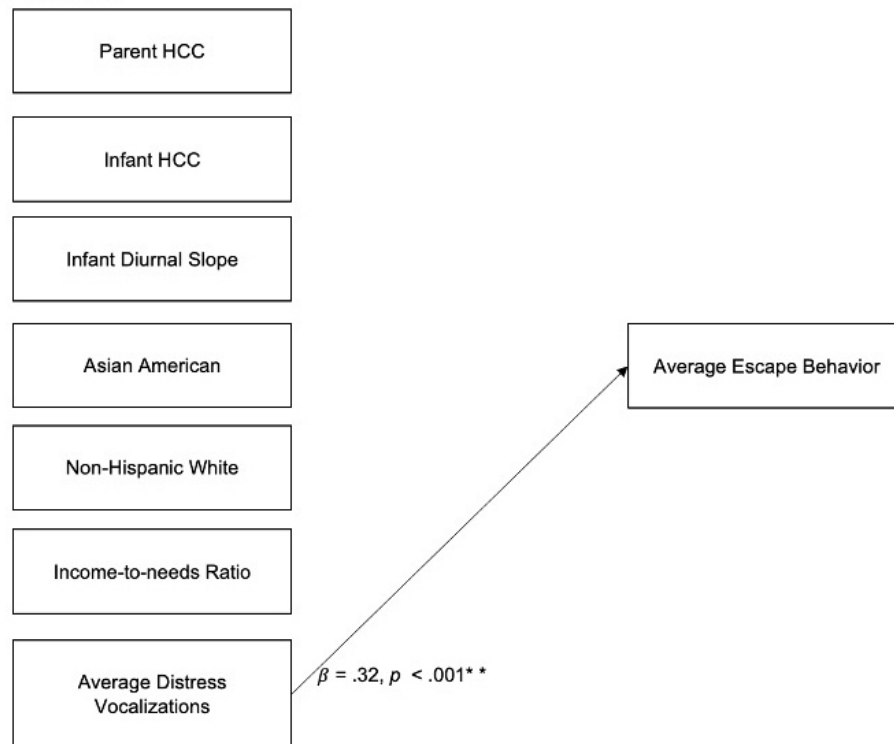

Figure 2. **Direct effects of cortisol measures and covariates on infant average escape behaviors during the Stranger Approach Paradigm**. $p \le .05^*$, $p \le .01^{**}$, $p \le .001^{***}$. Multiple linear regression and standardized estimates of the relations between parent HCC, infant HCC, infant diurnal cortisol slope, and relevant covariates and infant average escape behavior during the Stranger Approach Paradigm. 35.7% of variance in infant escape behavior was explained by this regression model.

Socioeconomic differences in the relation of biological stress and social fear. Ordinary least squares path analyses using the dichotomous ITN variable revealed no socioeconomic group differences in the relations between cortisol measures and infant social fear. Specifically, we tested interactions between infant HCC, parent HCC, infant diurnal slope and the dichotomous ITN variable in relation to distress vocalizations and average intensity of escape behavior, with no models yielding significant group differences in fear outcomes between economically strained groups.

DISCUSSION

The current study is the first to assess the relations of infant chronic and diurnal psychobiological stress to infant social fear behaviors. Using the Stranger Approach paradigm with 12-month-old infants, we found that flatter infant diurnal cortisol slope and increased infant HCC were associated with more distress vocalizations. These findings indicate that increased exposure to chronic psychobiological stress and dysregulated circadian cortisol regulation are independent in their associations with increased infant social fear. There were no socioeconomic differences in the relations between infant cortisol measures and distress vocalizations nor escape behavior during the Stranger Approach task. These findings suggest homogeneity in social fear behaviors across socioeconomic strain and implicate multiple measures of psychobiological stress as unique correlates of infant social fear behaviors that may inform future research on socioemotional development.

The current results expand upon previous research suggesting that increased fearfulness, as indexed by teacher reports, is associated with flatter diurnal cortisol slopes in infants in childcare [10] by using observational measures of social fear. Others have examined infant diurnal salivary cortisol slope as a longitudinal predictor of socially anxious behaviors in childhood, implicating infant diurnal slope as an early biomarker that potentially confers risk for maladaptive patterns of social adjustment later in life [6]. Our findings extend these results by showing that diurnal cortisol rhythms are linked to observed social fear behaviors in infancy above and beyond infant chronic psychobiological stress. Thus, our results offer evidence that blunted infant diurnal cortisol rhythms, which reflect HPA dysregulation, could help identify infants at risk for heightened fear responses to social stressors. Additionally, our findings add to previous research showing null relations between cortisol reactivity and fear behaviors in infant social stress tasks [29] by demonstrating that other measures of acute biological stress (ie, diurnal salivary cortisol slope) may pose unique risk for early fearfulness. Although we did not collect salivary cortisol reactivity data during the in-lab Stranger Approach task, and thus could not include cortisol reactivity in our models, our findings suggest that dysregulation in daily regulatory patterns, rather than cortisol reactivity to a specific stimulus, could be an indicator of infant ability to manage social stressors.

Our results that increased infant HCC was associated with increased distress vocalizations during the Stranger Approach task extend previous literature examining infant HCC and social fearfulness in nonhuman primates [54] as well as research linking childhood HCC with fearfulness in preschool [45]. Our findings offer evidence that HCC

may be linked to behavioral responses to social stressors in human infants as well. Consistent with prior literature [77], we found that infant and parent HCC were positively correlated. However, our results revealed that infant HCC was uniquely associated with distress vocalizations above and beyond parent HCC, while parent HCC was not associated with either fear behavior. This suggests that infant HCC uniquely contributes to observed variance in specific types of infant fear behaviors – a finding that is particularly striking for elucidating biomarkers of fearfulness in early life. Children exhibiting heightened fearfulness are at greater risk for anxiety disorders [78], social withdrawal [40], and peer rejection [79]. One possible explanation for our results is that persistent infant biological stress could promote more intense negative reactions to social novelty, which may lead to long-term consequences for social adjustment in childhood. Alternatively, increased social fearfulness may contribute to infant chronic biological stress accumulation or impaired daily salivary cortisol regulation, leading to bidirectional relations between social fear and HPA regulation [80]. While more research is needed to clarify links between early HCC and maladaptive social outcomes associated with increased fearfulness and the directionality of these relations, our findings suggest that increased infant HCC may confer unique risk for specific fear behaviors in 12-month-olds.

Our analyses revealed no associations between infant HCC or slope and escape behavior. The null result may reflect granularity in fear responses that could differentially relate to measures of psychobiological stress. Escape behaviors may be more strongly related to immediate biological responses to social and nonsocial stressors, while distress vocalizations may be associated with underlying biological stress exposure and regulation. While some studies have found no associations between cortisol reactivity and social fear more broadly [29], others have suggested that increased salivary cortisol reactivity is specifically linked to more escape behaviors in toddlers [81]. Moreover, children that exhibit greater salivary cortisol reactivity during social stressors also demonstrate more withdrawal, proximity-seeking to caregivers, and activity cessation while in the company of unfamiliar conspecifics [82]. Although we did not collect salivary cortisol reactivity data in the current study, future research could examine distinct psychobiological correlates of specific fear responses in infancy.

Indeed, fine-grained assessments of fear behaviors may be important for understanding the development of fear-related disorders and mental health outcomes. Mouse models have shown that specific fear behaviors, such as freezing, are enhanced in mice selectively bred for increased anxiety-related behaviors in comparison to normal controls, while flinching, jumping, and distress

vocalizations are not [83]. Human research with young children has also established correlations between specific types of fears and coping patterns that increase risk for anxiety disorders [84]. Thus, there may also be variability in the types of infant social fear responses that selectively increase risk for psychopathology. Importantly, although we did not test for differences in nonsocial fear responses, granularity in fear behaviors more generally may also exist given variation in physiological responses in nonsocial fear paradigms [85]. While more research is necessary to parse the mechanisms by which aspects of HPA function relate to granular fear responses in infancy, future analyses could examine how developmental trajectories of specific infant fear behaviors and psychobiological stress predict risk for psychopathology longitudinally. Further, future research may compare social and nonsocial fear behaviors in relation to underlying physiology to examine if differences exist across types of fear responses.

Although lower income has been linked with fearfulness in childhood [57,58], our analyses did not show direct effects between ITN and our social fear outcomes. Additionally, ITN did not moderate the relations between infant cortisol measures and observed infant social fear responses. While lower income infants did have higher chronic biological stress, our results suggest that differences in infant psychobiological stress across socioeconomic status may not be related to early social fear behaviors. We also did not find that ITN was correlated with infant diurnal slope, which is contradictory to existing research linking lower household income to flatter diurnal cortisol slopes in infancy [47,59]. Since higher income infants did not show different levels of social fear than lower income infants, but did show reduced HCC on average, it appears as though the relations between infant chronic psychobiological stress and social fear behaviors may exist independently from socioeconomic strain.

Considering that HCC is genetically heritable above and beyond early life experiences [62,86], our results may be indicative of genetic variation supporting temperamental differences in social fear. Despite this, research has also shown strong associations between socioeconomic inequality and fearful temperament [18] which was not reflected in our findings. Alternatively, chronic psychobiological stress may be influenced by contextual factors not assessed here, such as neonatal cortisol exposure, individual differences in anthropometry [87], parenting styles, or infant-caregiver attachment [88]. Regardless, our results indicate that infant HCC and social fear are linked independently of socioeconomic variation. More research is necessary to elucidate the mechanisms and contextual influences contributing to these associations, particularly using longitudinal data that can aid in determining the directionality of these relations in infant development [89].

Strengths of the current study included our multi-method approach, ecologically diverse sample, and use of objective measures of cortisol. Our use of a well-validated temperamental assessment also enhanced the validity of our results while avoiding potential confounds of parent-reports. Despite these strengths, our measure of economic strain may not be directly generalizable to extremes of poverty, particularly at an international level [90]. Our sample size was relatively small, which constrained our ability to conduct *post-hoc* analyses of other aspects of early experience that may moderate the relations between our infant cortisol measures and observed social fear. Our sample size also limited our ability to conduct three-way interactions between infant race, economic strain, and cortisol measures in relation to fear behaviors, which may be an interesting direction for future research addressing the impact of economic strain on minority infant biological stress. Lastly, our cross-sectional model did not allow for long-term interpretations of directionality, nor did it allow us to examine the potential mediating role of cortisol measures in the associations between economic strain and social fear [89]. Additional research should expand upon our findings through longitudinal research using infant cortisol measures as markers of fear trajectories across childhood and beyond.

CONCLUSION

Heightened social fear behaviors pose risk for maladaptive social outcomes and psychopathology in early life. Thus, it is essential to clarify factors underlying increased social fear behaviors in infancy to elucidate correlates of behaviors that are known to promote adverse mental health symptoms over time. The current study is the first to demonstrate unique associations of both infant diurnal psychobiological stress regulation and chronic psychobiological stress and social fear behaviors. Our results provide initial empirical evidence that better circadian regulation and reduced exposure to chronic stress are linked to fewer social fear behaviors from a very young age, although additional research is necessary to examine these relations longitudinally, in relation to other contextual factors, and cross-culturally. Taken together, our findings underscore biological correlates of fearfulness in infancy that may increase risk for, or buffer against, social maladjustment and fear-related disorders later in life.

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