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RESEARCH ARTICLE

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Emotion regulation moderates the association between parent and child hair cortisol concentrations

Katie Kao[1] | Charu T. Tuladhar[2] | Jerrold S. Meyer[3] | Amanda R. Tarullo[2]

[1]Harvard Medical School, Brazelton Touchpoints Center, Division of Developmental Medicine, Boston Children's Hospital, Boston, Massachusetts

[2]Department of Psychological and Brain Sciences, Boston University, Boston, Massachusetts

[3]Department of Psychological and Brain Sciences, University of Massachusetts, Amherst, Massachusetts

Correspondence
Katie Kao, Brazelton Touchpoints Center, Division of Developmental Medicine, Boston Children's Hospital, Boston, MA.
Email: katiekao@bu.edu

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Abstract

Successful emotion regulation facilitates children's coping with everyday stress. It develops rapidly in the early preschool period. However, no work has been done to investigate the potential buffering role of emotion regulation from cumulative physiological effects of stress. In this study, we examined hair cortisol concentration (HCC), an early marker of chronic physiological stress, socioeconomic status (SES), parental sensitivity, and emotion regulation and reactivity in a sample of 3.5-year-old children (N = 86). Emotion regulation and emotional reactivity were independent of child HCC. However, emotion regulation moderated the relationship between parent and child HCC. For children with better emotion regulation, there was no association between parent and child HCC, suggesting that emotion regulation skills buffered the transgenerational effects of chronic physiological stress. Emotional reactivity moderated the relationship between SES and child HCC, and attenuated the association between parental sensitivity and child HCC. Taken together, these findings demonstrate that children who were less emotionally reactive were less susceptible to their environments. Results provide support that child emotion regulation and emotional reactivity can reduce or strengthen the relationship between established risk factors and levels of chronic physiological stress in early childhood.

KEYWORDS
chronic stress, emotion regulation, hair cortisol, preschool, socioeconomic status

1 | INTRODUCTION

Early childhood exposure to psychosocial stressors has been linked to later health outcomes. One proposed mechanism to help explain this association is chronic overactivation of the hypothalamic–pituitary–adrenocortical (HPA) axis, of which the end product is the stress hormone cortisol (Karlén et al., 2015; King & Hegadoren, 2002). Long-term exposure to cortisol elevations may contribute to dysregulation of interconnected physiological systems, referred to as allostatic load (McEwen & Stellar, 1993; Wosu, Valdimarsdóttir, Shield, Williams, & Williams, 2013). Allostatic load predicts long-term physiological functioning, morbidity, and mortality (Danese & McEwen, 2012; Taylor, Way, & Seeman, 2011). More research is needed to understand the mechanisms through which early accumulation of stress can lead to poor health outcomes throughout life.

Hair cortisol concentration (HCC) has been established as an early biomarker of cumulative exposure to physiological stress (Russell, Koren, Rieder, & Van Uum, 2012). Cortisol is deposited in the hair shaft as it grows. Thus, when taking into account the rate of growth, a hair sample can provide a timeline of long-term cortisol exposure (Meyer & Novak, 2012; Russell et al., 2012). While salivary cortisol is often used as a biomarker of an individual's acute stress or diurnal rhythm, HCC is a chronic measure which indexes both basal circulating cortisol levels and cumulative physiological

reaction to stress over time. Elevated child HCC has been related to increased stressful life events (Vanaelst et al., 2013). Prior studies have also established that higher parent HCC, lower socioeconomic status (SES), and poor parenting quality are related to increased levels of cortisol in young children (Flom, St. John, Meyer, & Tarullo, 2017; Kao, Doan, St. John, Meyer, & Tarullo, 2018; Karlén, Frostell, Theodorsson, Faresjö, & Ludvigsson, 2013; Ouellette et al., 2015; Tarullo, St. John, & Meyer, 2017; Vaghri et al., 2013; Vliegenthart et al., 2016). More research in early childhood is needed to understand the mechanisms for how early accumulation of stress throughout life can lead to poor health outcomes. The HPA axis is still developing in the first few years of life and is sensitive to early experiences (Gunnar & Talge, 2008). Ecobiodevelopmental frameworks for childhood adversity (Shonkoff, 2010; Shonkoff & Garner, 2012) posit that early life stressors in the child's environment are incorporated into the developing child's biological functioning through processes of physiological adaptation or disruption, and that this altered biological functioning then contributes to long-term health disparities. Critically, however, ecobiodevelopmental frameworks also posit an interplay between a child's behavioral and emotional functioning and biological functioning (Shonkoff & Garner, 2012). Yet, despite the growing empirical literature linking early life stressors to child hair cortisol, indexing chronic physiological stress, this potential interplay with child emotional functioning is largely unexamined. Thus, it is essential to investigate individual child characteristics that may attenuate or strengthen risk factors for chronic physiological dysregulation, as indexed by hair cortisol.

This paper focuses on understanding the role of emotion regulation and emotional reactivity in children's chronic physiological stress, including as potential moderators of the associations between early identified risk factors and children's chronic physiological stress. In the following sections, we (1) briefly review the literature on preschooler's emotion regulation development and discuss why emotion regulation may be relevant to cortisol function; (2) explore parent HCC, SES, and parent sensitivity as early risk factors for children's HCC; and (3) propose a model to test emotion reactivity and emotion regulation as potential moderators of the associations between these distinct risk factors and child HCC.

1.1 | Child emotion regulation and emotional reactivity

Two components to consider when addressing children's emotional capacities are emotion regulation and emotion reactivity. Emotion regulation are internal and external processes that modulate emotional experiences to meet situational demands and achieve goals (Cole, Martin, & Dennis, 2004; Gross & John, 2003). Emotional reactivity reflects the characteristic threshold, intensity, and duration of affective arousal (Rothbart & Bates, 2006). While related, emotion regulation and emotional reactivity uniquely contribute to the prediction of children's psychosocial functioning (Rydell, Berlin, & Bohlin, 2003). Conceptually, emotion reactivity refers to individual differences in how easily and intensely emotions are aroused (Murphy, Eisenberg, Fabes, Shepard, & Gunthrie, 1999) while emotion regulation involves successful management of emotional arousal (Calkins, 1994; Underwood, 1997). The two processes are related but separable at the empirical level, while both contributing to the production of behavior (Kagan, 1994). Under this assumption, children who are more emotionally reactive may also have a harder time regulating their emotions. From a developmental perspective, emotion reactivity and emotion regulation have different developmental courses. Early in development, infants demonstrate different frequencies and intensities of emotionality, which in part reflect temperamental characteristics (Rothbart, 1989). Emotion reactivity is moderately stable across early childhood, though influenced by caregiving experiences (Hane, Henderson, Reeb-Sutherland, & Fox, 2010). Emotion regulation, by contrast, depends on cognitive domains such as theory of mind and executive function, and therefore begins to emerge in in the early preschool period as children make strides in these key cognitive domains (Carlson, Moses, & Claxton, 2004; Denham & Kochanoff, 2002).

Particularly during the preschool period, children undergo rapid changes in emotion regulation and reactivity (see for review Cole et al., 2004). Parenting quality is a likely source of individual differences in the development of emotion regulation and reactivity (Morris, Silk, Steinberg, Myers, & Robinson, 2007; Thompson & Meyer, 2007). Studies with older children implicate the important contribution of parental sensitivity on children's ability to self-regulate their emotions (Cassano, Perry-Parrish, & Zeman, 2007; Jaffe, Gullone, & Hughes, 2010; Morris et al., 2002; Yap, Allen, & Ladouceur, 2008) as well as the influence of parenting quality on children's negative reactivity and emotionality (Duncombe, Havighurst, Holland, & Frankling, 2012). Parents who display positive affect, express affection, and respond appropriately to their child's cues serve as effective emotion coregulators in early childhood. These parents provide external soothing and support to guide children directly as they cope with frustrating events and model self-regulatory strategies that children internalize to support self-soothing (Dennis, 2006). Between 2 and 5 years of age, advances in cognitive, motor, social, and language development occur which enable the child to develop the capacity to regulate their own emotions (Kopp & Neufeld, 2003). However, during this early preschool age when emotion regulation capacities are just beginning to emerge, the extent to which children's emotional functioning reflects parenting quality is less understood.

1.2 | Child emotion regulation: implications for socioemotional functioning and stress physiology

It is well established that maladaptive patterns of emotion regulation and dysregulated emotional expression can compromise socioemotional functioning, which may result in symptoms of psychopathology (Eisenberg et al., 2001; Kim & Cicchetti, 2009). Research suggests that the greatest risk for poor psychosocial functioning occurs when children are highly reactive in emotionally arousing situations, show more negative rather than positive emotional expressions, and have maladaptive emotion

regulation strategies (Eisenberg, Fabes, Gunthrie, & Reiser, 2000). Furthermore, children who are more adept at regulating the experience of emotion depending on contextual demands have better coping skills when faced with everyday stressors (Lengua, 2002). Thus, it is conceivable that more adaptive emotion regulation may be associated with lower cortisol levels over time. However, to our knowledge, no studies have examined the direct relationship between emotion regulation and HCC.

While deficits in emotion regulation and higher levels of negative emotional expression are related to behavioral socioemotional difficulties (Keenan, 2006; Shipman, Schneider, & Brown, 2004), the role of emotion regulation and reactivity with HCC is less clear. Emotion regulation and reactivity may be particularly important among children who experience sociocontextual stressors, such that emotion regulatory abilities may attenuate the relationship between known risks factors and chronic physiological stress. Children with better emotional coping skills may be better able to utilize regulatory strategies and down-regulate negative physiological arousal when exposed to enduring stressors. Work is needed to identify the extent to which children's emotion regulation and reactivity contribute to the interplay of sociocontextual and biological stress.

1.3 | Parent and child hair cortisol

Growing evidence indicates that parent and child HCC are associated in infancy (Flom et al., 2017; Karlén et al., 2013), the preschool period (Kao et al., 2018), and later childhood (Ouellette et al., 2015). There are several possible explanations for the intergenerational transmission of parent and child HCC. Parents and young children may be exposed to similar environmental stressors, resulting in more similar cumulative cortisol exposure (Stenius et al., 2008). It is also possible that parents and children have related HCC because HCC is genetically influenced. Twin studies have demonstrated the heritability of basal salivary cortisol (Bartels, Geus, Kirschbaum, Sluyter, & Boomsma, 2003; Ouellet-Morin et al., 2008, 2009) and animal studies have indicated the heritability of HCC in monkeys (Fairbanks et al., 2011). Recently, the first genetic study examining the role of genetic variation of long-term cortisol exposure revealed that genetic factors accounted for about half the variation in HCC (Tucker-Drob et al., 2017). Thus, the relation of parent and child HCC may be due, in part, to shared genetic alleles.

Despite this established link between parent and child chronic physiological stress, less is known about how this association may differ as a function of individual characteristics, such as children's emotion regulation and reactivity. Children who have better emotion regulation strategies might have better coping skills in the context of high parental stress, potentially buffering them from elevated levels of cumulative cortisol exposure. Conversely, children who are highly reactive in emotionally arousing situations may not have the emotional control to adaptively respond in the context of high parental stress, potentially leaving them more vulnerable to physiological dysregulation.

1.4 | Sociocontextual risk and child hair cortisol

Sociocontextual stressors have been linked with physiological dysregulation in adults and children, such as chronic overactivation of the HPA axis (Evans & English, 2002; Juster, McEwen, & Lupien, 2010). Low SES often refers to income levels below federal poverty levels or other risk factors such as lower education levels or occupational status. Lower SES (consisting of parent education, parent occupational prestige, and income-to-needs ratio) was related to higher HCC in 1-year-olds (Flom et al., 2017). Poorer maternal education and lower neighborhood SES have been correlated with higher HCC in preschool and older children (Vaghri et al., 2013; Vliegenthart et al., 2016). While SES has been examined more extensively with *salivary* cortisol, the results are complex. For example, lower SES has been associated with higher basal salivary cortisol (e.g., Clearfield, Carter-Rodriguez, Merali, & Shober, 2014; Lupien, King, Meaney, & McEwen, 2000) as well as dampened diurnal salivary cortisol levels in children (Flom et al., 2017). Adults and children who experienced previous trauma had blunted basal salivary cortisol levels compared to those who did not experience trauma (Bevans, Cerbone, & Overstreet, 2008; Klaassens, Giltay, van Veen, Veen, & Zitman, 2010). Nonetheless, this research collectively suggests that low SES is a risk factor for early physiological dysregulation, indexed by both chronic and acute cortisol levels. While we expect a negative association between SES and child HCC, this pattern may differ for children in low-SES environments with better emotional coping abilities. Children in poverty are more likely to be exposed to stressors such as residential instability, higher levels of neighborhood and family violence, and psychological distress among caregivers which can compromise children's psychological and physiological functioning (Gershoff, Aber, Raver, & Lennon, 2007). Children's ability to cope with these stressors may dampen or strengthen the relationship between SES and children's HCC. Examining the moderating role of emotion regulation and reactivity on the interplay of environmental and chronic physiological stress may help elucidate risk within the child in the context of poverty.

1.5 | Parental sensitivity and child HCC

The HPA axis is immature at birth and young children depend on sensitive caregiving to shape healthy stress physiology. In the absence of sensitive care, children's cortisol function may become dysregulated (Gunnar & Talge, 2008). While parental sensitivity has not been explicitly examined with child HCC, several studies show the association between parenting quality and measures of salivary cortisol. For example, low parental sensitivity (i.e., poor ability to perceive children's signals and accurately vary behavior appropriately) has been related to higher basal salivary cortisol across the day and higher total salivary cortisol output overnight (Letourneau, Watson, Duffett-Leger, Hegadoren, & Tryphonopoulos, 2011; Philbrook et al., 2014). Thus, parental sensitivity is a likely factor that influences children's chronic physiological stress, indexed by HCC. Given the evidence that poor

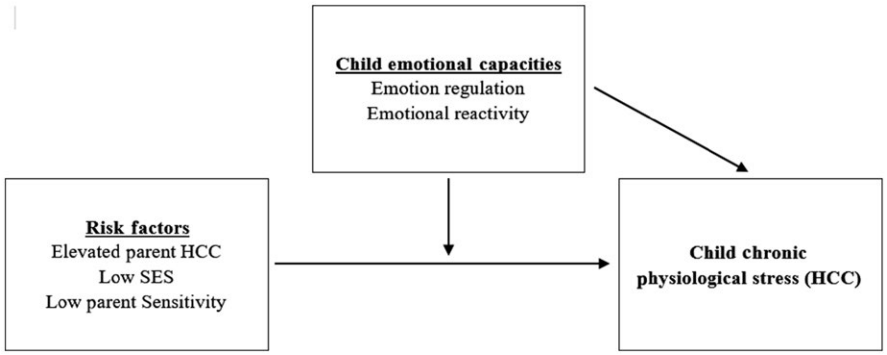

FIGURE 1 Conceptual model tested in this paper, proposing that child emotional capabilities both have a direct association with child chronic physiological stress (child hair cortisol concentration [HCC]) and moderate the association between risk factors (elevated parent HCC, low socioeconomic status, and low parent sensitivity) and child HCC

parenting quality is related to dysregulated cortisol responses in children, examining emotion regulation and reactivity as potential moderators of this relationship may help us better understand individual characteristics that may change whether children's HCC levels are at risk when exposed to environmental stressors such as insensitive parenting.

1.6 | The current study

Early chronic physiological stress has enduring impacts on children's physical and mental health outcomes. Children's early environments can be risk factors for elevated cumulative cortisol exposure during the preschool years. Not yet explored, however, are emerging child emotion characteristics, such as emotion regulation and reactivity, which may directly relate to HCC levels or may contribute to the interplay of sociocontextual factors and chronic physiological stress. Our hypotheses in the current study are informed by ecobiodevelopmental frameworks and by the existing literature on how emotion regulation and negativity reactivity relate to adaptive behavioral functioning. As discussed above, emotion regulation and reactivity are directly related to socioemotional well-being (Kim & Cicchetti, 2009) and also modulate the impact of psychosocial stressors (Lengua, 2002). We hypothesize a parallel role for these emotional capacities in child chronic physiological stress, relating directly to child HCC and also moderating the association of risk factors with child HCC. The objective of the current study was to test this conceptual model, shown in Figure 1. Specifically, we aimed (1) to examine emotion regulation and emotional reactivity as potential correlates of child HCC, and (2) to explore whether emotion regulation and emotional reactivity moderated the relationships of identified risk factors (elevated parent HCC, low SES, and low parental sensitivity) of child HCC. We hypothesized that poorer emotion regulation and increased emotional reactivity would relate to elevated levels of child HCC. We also predicted that the associations between child HCC with parent HCC, SES, and parental sensitivity would differ as a function of emotion regulation and emotional reactivity. Given the detrimental health outcomes related to allostatic load, it is crucial to identify individual child characteristics that may buffer children from the enduring effects of sociocontextual stressors on chronic physiological stress.

2 | METHOD

2.1 | Participants

Ninety-one children were enrolled in the study, but five children did not provide useable hair cortisol values and were excluded from analysis. This resulted in a final sample of 86 children (44 female) aged 3.5 years old (M = 3.54 years, SD = 0.13 years) and their primary caregiver (78 mothers and 8 fathers). Participants were from the greater Boston metropolitan area and were recruited from a department-maintained database of families who had expressed interest in participating in research, from online advertising, and from community recruitment events. Participating children were 60.5% European American, 5.8% African American, 9.3% Asian, 7.0% multiracial, and 17.4% Hispanic. All children were full-term singletons who had no known auditory, visual, neurological, or developmental disorders. Our sample included a wide range of SES levels with the top 25% of our participants making over $150,000 and the bottom 24% qualifying for public assistance based on income (see Table 1 for demographics).

2.2 | Procedure

This study was approved by the University Institutional Review Board. Upon arrival, the primary caregiver provided informed

TABLE 1 Demographic information

Child age (years)	
M (*SD*)	3.54 (0.13)
Child ethnicity	
European American	60.5%
Black	5.8%
Asian	9.3%
Hispanic	17.4%
Multiracial	7.0%
Participating parent education (% with at least a 4-year college degree)	82.6%
Non-participating parent education (% with at least a 4-year college degree)	77.7%
Annual income (% household income over $60,000)	69.4%

consent. Hair cortisol samples were collected from both the child and parent and the parent filled out questionnaires. Parent–child dyads participated in a 12-min interaction that included a 5-min free play, 5-min structured play with a challenging wooden puzzle (Hammond, Müller, Carpendale, Bibok, & Liebermann-Finestone, 2012), and 2-min clean-up.

2.3 | Measures

2.3.1 | Hair cortisol

Hair cortisol measurement procedures followed our validated methods (Davenport, Tiefenbacher, Lutz, Novak, & Meyer, 2006; Meyer, Novak, Hamel, & Rosenberg, 2014). Hair samples were collected from both parent and child and were then assayed to determine HCC. Samples of 3 cm length from the scalp and weighing 15–30 mg were cut from the posterior vertex of the head. Because washing, hair coloring, or hair straightening may affect HCC (Hoffman, Karban, Benitez, Goodteacher, & Laudenslager, 2014), parents were asked about their own and their child's hair history including the frequency that the hair got wet. Human scalp hair grows at approximately 1 cm per month (LeBeau, Montgomery, & Brewer, 2011), so the 3 cm sample indexed cortisol output over the past 3 months. Hair samples were stored in plastic tubes labeled with subject ID, and were frozen at −20°C until cortisol analysis. Hair samples were weighted, washed twice with isopropanol to remove contaminants, dried, and ground into a fine powder. Cortisol was extracted into methanol, which was then evaporated, and the residue was reconstituted in assay buffer. Reconstituted extracts were analyzed for cortisol using a sensitive and selective commercially available enzyme immunoassay (Salimetrics, LLC; Carlsbad, CA). Assay readout was converted to pg cortisol per mg of dry hair weight. Both intra- and interassay coefficients of variation were approximately 10%.

Raw HCC levels for children (M = 35.27, SD = 80.63) and parents (M = 13.57, SD = 24.40) were natural log-transformed because the data were not normally distributed. Most parents in this sample provided useable HCC values (95.6%). Of those without useable HCC, one parent declined hair cortisol collection and three samples were lost during processing. Most children also had useable HCC (94.5%). Of those without useable HCC, one sample was excluded due to biologically implausible HCC values (1,218.0 pg/mg), one parent declined child hair cortisol collection, and three samples were lost during processing. Parent's HCC was unrelated to frequency of washing (Spearman's rho = 0.05, p = 0.674), color treatment ($t(84)$ = 0.24, p = 0.814), or hair straightening ($t(84)$ = −0.20, p = 0.839). Frequency of washing was also unrelated to children's HCC (Spearman's rho = 0.15, p = 0.171). Therefore, it was not necessary to correct for variation in hair care habits.

2.3.2 | SES composite

The following three indicators were standardized and averaged to create a comprehensive SES composite.

2.3.3 | Income-to-needs ratio

Parents reported their annual household income and the number of family members currently living in the household. To calculate income-to-needs, we divided the total family income by the federal poverty threshold based on the number of household members. Three cases were statistical outliers and therefore winsorized to within 3 *SD*s to restore normality of distribution.

2.3.4 | Parent education

The highest level of education from both parents was coded on a scale from 1 to 10 (1 = no education to 10 = graduate school). Codes were standardized and averaged to create a combined parent education composite.

2.3.5 | Occupational prestige

Occupational prestige was coded for each parent using the Job Zone coding scheme from the Occupational Information Network (O*NET, http://www.onetonline.org/help/online/zones), which ranks U.S. Census-based occupational categories on a 1–5 scale based on the education, experience, and training required. Codes were standardized and averaged to create a combined parent occupational prestige composite.

2.3.6 | Parental sensitivity

Video records of parent-child interactions were coded using the sensitivity subscale from the Emotional Availability (EA) Scales (Pipp-Siegel & Biringen, 1998). The EA scales capture the capacity of a dyad to share an emotionally healthy relationship and assess the affect and behavior of both the child and caregiver during an interaction (Biringen, Derscheid, Vliegen, Closson, & Easterbrooks, 2014). Specifically, the scores for the sensitivity subscale are based on the parent's ability to read the child's cues so that responses to child behaviors are considered and sensitivity is interpreted in relation to the child. This includes clear, accurate perceptions of emotions, responsiveness, ability to handle conflictual situations, and awareness of timing (Biringen & Easterbrooks, 2012). Higher scores indicate optimal sensitivity while lower scores reflect emotional detachment. Two certified coders who completed the EA training program coded the videos and 20% of the sample was double coded for reliability. Intraclass correlation coefficients (ICC) were calculated to assess inter-rater reliability. The ICC for parental sensitivity was 0.81.

2.3.7 | Emotion regulation behaviors

The primary caregiver completed the Emotion Regulation Checklist (ERC; Shields & Cicchetti, 1997) to report on their child's emotion regulation and reactivity. The ERC has been validated for preschool children (Cohen & Mendez, 2009; Graziano, Reavis, Keane, & Calkins, 2007; Izard et al., 2008). The measure is composed of 24 items rated

on a 4-point Likert scale that indicates the frequency of emotion related behaviors from 1 (never) to 4 (always), and yields two subscales: negativity/lability and emotion regulation. The negativity/lability subscale, indexing both negative emotional reactivity and general reactivity, includes 16 items that assess children's tendency to display emotional responses, and includes items such as "is easily frustrated" and "exhibits wide mood swings." The emotion regulation subscale includes eight items that refer to children's ability to modulate emotional arousal, and includes items such as "displays appropriate negative affect in response to hostile, aggressive, or intrusive acts by peers" or "can say when s/he is feeling angry." Negatively weighted items were reverse-scored and items corresponding to each subscale were averaged to yield two total scores for emotion reactivity (α = 0.74) and emotion regulation (α = 0.70), where higher scores reflected greater emotional reactivity and better emotion regulation respectively.

2.4 | Analysis plan

In preliminary analyses, gender was tested as a potential covariate to determine whether it needed to be controlled for in subsequent analyses. Group differences between male and female children in HCC, emotional reactivity, and emotion regulation were assessed using independent samples *t* tests. Group differences between male and female parents in HCC were also assessed using independent samples *t* test. We also examined whether there were group differences in European American and non-European American children and parents in HCC. Pearson correlations were conducted between the child emotion variables (i.e. emotion regulation and emotion reactivity) and the environmental factors (i.e. SES and parenting).

In the main analyses, Pearson correlations were conducted to test the hypothesized direct association of child emotional variables (emotion regulation and emotional reactivity) with child HCC. Next, we used Pearson correlations to examine relations of child HCC to the risk factors (elevated parent HCC, low parental sensitivity, and low SES). Finally, to examine the extent to which children's emotion regulation and emotional reactivity attenuated the relationship between risk factors and child HCC, we tested emotion regulation and emotional reactivity as moderating factors in the associations of parent HCC, SES, and parental sensitivity with child HCC. Moderation analyses were conducted using ordinary least squares path analysis (Hayes, 2013) where significant effects were estimated using bias-corrected bootstrap confidence intervals at the 95% level and based on 5,000 samples. All variables were entered in the moderation models as continuous variables.

3 | RESULTS

3.1 | Preliminary analyses

Natural log-transformed values of HCC were used in all analyses (see Table 2 for descriptive statistics). Child gender was not associated with child HCC, emotion regulation, or emotionally reactivity, and thus was not included as a covariate in further analyses. Parent's gender was not associated with parent HCC levels and was also not included as a covariate. There was a difference between European American and non-European American children in HCC, such that non-European American children had higher HCC, $t(50.52) = -2.97$, $p = 0.004$. There were no racial differences in parent HCC. Emotion regulation and emotion reactivity were not related to SES or parental sensitivity. See Table 3 for correlations among all variables.

TABLE 2 Descriptive statistics for study variables

Variable name	*M* (*SD*)	Min	Max	*N*
Child HCC (natural log)	2.74 (111)	0.00	6.38	86
Parent HCC (natural log)	1.99 (1.02)	−1.20	5.01	85
SES (*z*-score)	−0.004 (0.79)	−2.08	1.63	86
Child emotion regulation	3.24 (0.40)	2.13	4.00	85
Child emotion reactivity	1.78 (0.30)	1.13	2.67	85
Parental sensitivity	5.48 (1.16)	3.00	7.00	86

HCC: hair cortisol concentration; SES: socioeconomic status.

3.2 | Emotion regulation, emotional reactivity, and child HCC

Child HCC was not related to emotion regulation, $r(85) = 0.07$, $p = 0.53$ or emotional reactivity, $r(85) = 0.10$, $p = 0.36$. Consistent with prior work (Blandon, Calkins, Keane, & O'Brien, 2008), emotion regulation and emotional reactivity were related but distinct constructs, $r(90) = -0.34$, $p = 0.001$.

3.3 | Moderating role of emotion regulation and emotional reactivity on parent and child HCC

Higher parent HCC was related to higher child HCC, $r(84) = 0.58$, $p < 0.001$ (see Figure 2a). To understand whether children's emotion regulation or emotional reactivity moderated the association between parent and child HCC, ordinary least squares path analysis (Hayes, 2013) was conducted. Parent HCC was the independent variable, emotion regulation and reactivity were entered in the model as potential moderators, and the dependent variable was child HCC. As shown in Table 4 and Figure 3a, a conditional process model ($F(5, 78) = 9.44$, $p < 0.001$) yielded a main effect of emotion regulation on child HCC ($b_2 = 1.53$, $p = 0.03$, CI: 0.16–2.90), and a significant parent HCC × emotion regulation interaction ($b_4 = -0.77$, $p = 0.02$, CI: −1.43 to −0.11), revealing a conditional effect of parent HCC on child HCC. The parent HCC × emotion regulation interaction accounted for 4.3% of the variance and significantly improved the model, $F(1, 78) = 5.40$, $p = 0.02$. Examination of estimated marginal means revealed that for children with high levels of emotion regulation, there was no relationship between parent and child HCC. However, at lower levels of emotion regulation, there was a positive association between parent and child HCC. Emotional

TABLE 3 Bivariate correlations among measures

	1	2	3	4	5	6
1. Child HCC	—	0.58**	−0.39**	0.07	0.10	−0.27*
2. Parent HCC		—	−0.21	−0.05	0.25*	−0.17
3. SES			—	0.19	−0.11	0.28*
4. Child emotion regulation				—	−0.36**	0.09
5. Child emotion reactivity					—	−0.08
6. Parental sensitivity						—

HCC: hair cortisol concentration; SES: socioeconomic status.
*p <0.05, **p <0.01.

reactivity was not a significant moderator in the model. Thus, the relationship between parent and child HCC varied depending on children's emotion regulation skills but not on levels of emotional reactivity. Figure 4a is an illustration depicting the interaction using estimated marginal means to visually highlight the interaction effect, and all variables were continuous.

3.4 | Moderating role of emotion regulation and emotional reactivity on SES and child HCC

Higher SES was correlated to lower child HCC, $r(85) = -0.39$, $p < 0.001$ (see Figure 2b). Ordinary least squares path analysis (Hayes, 2013) was again conducted to examine emotion regulation and emotional reactivity as potential moderators of the association between SES and child HCC. This time, SES was the independent variable, emotion regulation and reactivity were entered in the model as potential moderators, and child HCC was the dependent variable. As shown in Table 5 and Figure 3b, a conditional process model ($F(5, 79) = 4.84$, $p = 0.001$) yielded a significant SES x emotion reactivity interaction ($b_4 = -1.15$, $p = 0.02$, CI: −2.13 to −0.18), revealing a conditional effect of SES on child HCC. The SES × emotional reactivity interaction accounted for 5.3% of the variance and significantly improved the model, $F(1, 79) = 5.51$, $p = 0.02$. Examination of estimated marginal means revealed that for children who showed more emotional reactivity, lower SES related to higher child HCC. However, there was no relationship between SES and child HCC for children who were less emotionally reactive. Emotion regulation was not a significant moderator in the model. Thus, the relationship between SES and child HCC varied depending on children's emotional reactivity. Figure 4b is an illustration depicting the interaction using estimated marginal means to visually highlight the interaction effect, and all variables were continuous.

3.5 | Moderating role of emotion regulation and emotional reactivity on parental sensitivity and child HCC

Higher parental sensitivity was related to lower child HCC, $r(86) = -0.27$, $p = 0.01$ (see Figure 2c). Ordinary least squares path analysis (Hayes, 2013) was again conducted to examine emotion regulation and emotional reactivity as potential moderators of the association between parental sensitivity and child HCC. Parental sensitivity was the independent variable, emotion regulation and emotional reactivity were entered in the model as potential moderators, and child HCC was the dependent variable. As shown in Table 6 and Figure 3c, a conditional process model ($F(5, 79) = 3.23$, $p = 0.01$) yielded a significant parental sensitivity x emotional reactivity interaction ($b_4 = -0.95$, $p = 0.006$, CI: −1.61 to −0.28), revealing a conditional effect of parental sensitivity on child HCC. The parental sensitivity x emotional reactivity interaction accounted for 8.5% of the variance and significantly improved the model, $F(1, 79) = 8.07$, $p = 0.006$. There remained a main effect between parental sensitivity and child HCC after accounting for the interaction ($b_1 = 2.64$, $p = 0.05$, CI: 0.02–5.26), but this relationship was attenuated for children who were less emotionally reactive. Examination of estimated marginal means revealed that for children who showed more emotional reactivity, poorer parental sensitivity related to higher child HCC. Emotion regulation was not a significant moderator in the model. Thus, the relationship between parental sensitivity and child HCC varied depending on children's emotional reactivity. Figure 4c is an illustration depicting the interaction using estimated marginal means to visually highlight the interaction effect, and all variables were continuous.

4 | DISCUSSION

In a sample of preschool children, we examined the roles of emotion regulation and emotional reactivity in chronic physiological stress in early childhood, as indexed by HCC. Although neither emotion regulation nor emotional reactivity were correlated with child HCC, we found that emotion regulation moderated the relationship between parent and child HCC. For children with better emotion regulation skills, the intergenerational transmission between parent and child HCC was mitigated. Furthermore, emotional reactivity moderated the relationship between environmental risks (i.e., SES and parental sensitivity) and child HCC. For children who were less emotionally reactive, the relationship between SES and child HCC diminished and the relationship between sensitive parenting and child HCC was attenuated. Given

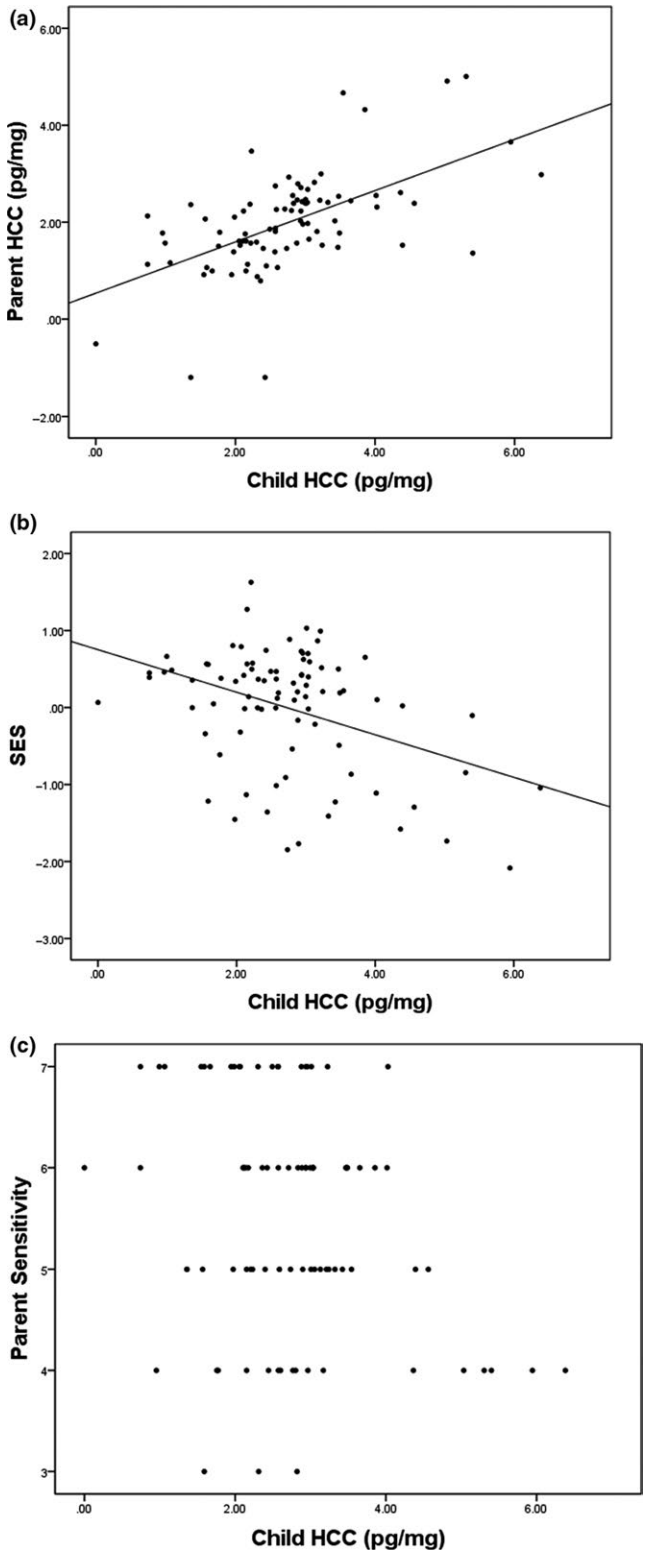

FIGURE 2 (a) Scatterplot showing the relationship between parent hair cortisol concentration (HCC) and child HCC, $r(84) = 0.58$, $p < 0.001$. (b) Scatterplot showing the relationship between socioeconomic status and child HCC, $r(85) = -0.39$, $p < 0.001$. (c) Scatterplot showing the relationship between parental sensitivity and child HCC, $r(85) = -0.27$, $p = 0.01$

TABLE 4 Model coefficients and summary information for moderation model of parent hair cortisol concentration (HCC) and child HCC

		Child HCC		
Antecedent		**Coeff.**	***SE***	***p***
Parent HCC	b_1	2.66	1.50	0.08
Emotion regulation	b_2	1.53	0.69	0.03
Emotion reactivity	b_3	−0.61	0.87	0.49
Parent HCC × emotion regulation	b_4	−0.77	0.33	0.02
Parent HCC × emotion reactivity	b_5	0.24	0.34	0.48
Constant		−2.38	3.39	0.49
		$R^2 = 0.38$		
		$F(5, 78) = 9.44$, $p < 0.001$		

the detrimental health effects of long-term elevated cortisol exposure, findings highlight that emotion regulation skills and emotional reactivity can weaken or strengthen the relationship between risk factors and chronic physiological stress in early childhood.

The current study adds to the growing evidence that parent and child HCC are associated in early childhood (Flom et al., 2017; Kao et al., 2018; Ouellette et al., 2015), and extends these findings by identifying that emotion regulation moderated this association. The strong correlation between parent and child HCC was only present for children with poor emotion regulation skills and not for children with more adaptive emotion regulation abilities. Findings suggest that children's own capacity of emotion regulation buffered the transgenerational effects of chronic physiological stress. In other words, for children with better emotion regulation skills, the concordance between parent and child HCC was mitigated.

Consistent with prior research (Flom et al., 2017; Vaghri et al., 2013; Vliegenthart et al., 2016), we found that children from lower SES households had higher HCC. Building on these findings, we provide evidence that this link is moderated by children's emotional reactivity. The relationship between SES and child HCC was only significant for children who were more emotionally reactive, suggesting SES matters less for chronic physiological stress in the context of better emotional stability. While emotion reactivity does not itself directly relate to chronic physiological stress, it appears to matter for the incorporation of SES context into biological stress functioning. Furthermore, children who generally showed less emotional intensity and exhibited fewer mood swings were not as susceptible to higher levels of chronic physiological stress even when exposed to poverty-related stressors. Cumulative stress, lack of resources, shifting work schedules, and single parenthood account for higher levels of chaos in low-income households compared with more economically advantaged households (Ackerman & Brown, 2010). Children who are less emotionally reactive may not be as emotive or attentive to negative characteristics of their surroundings, which could be protective in highly chaotic households. While

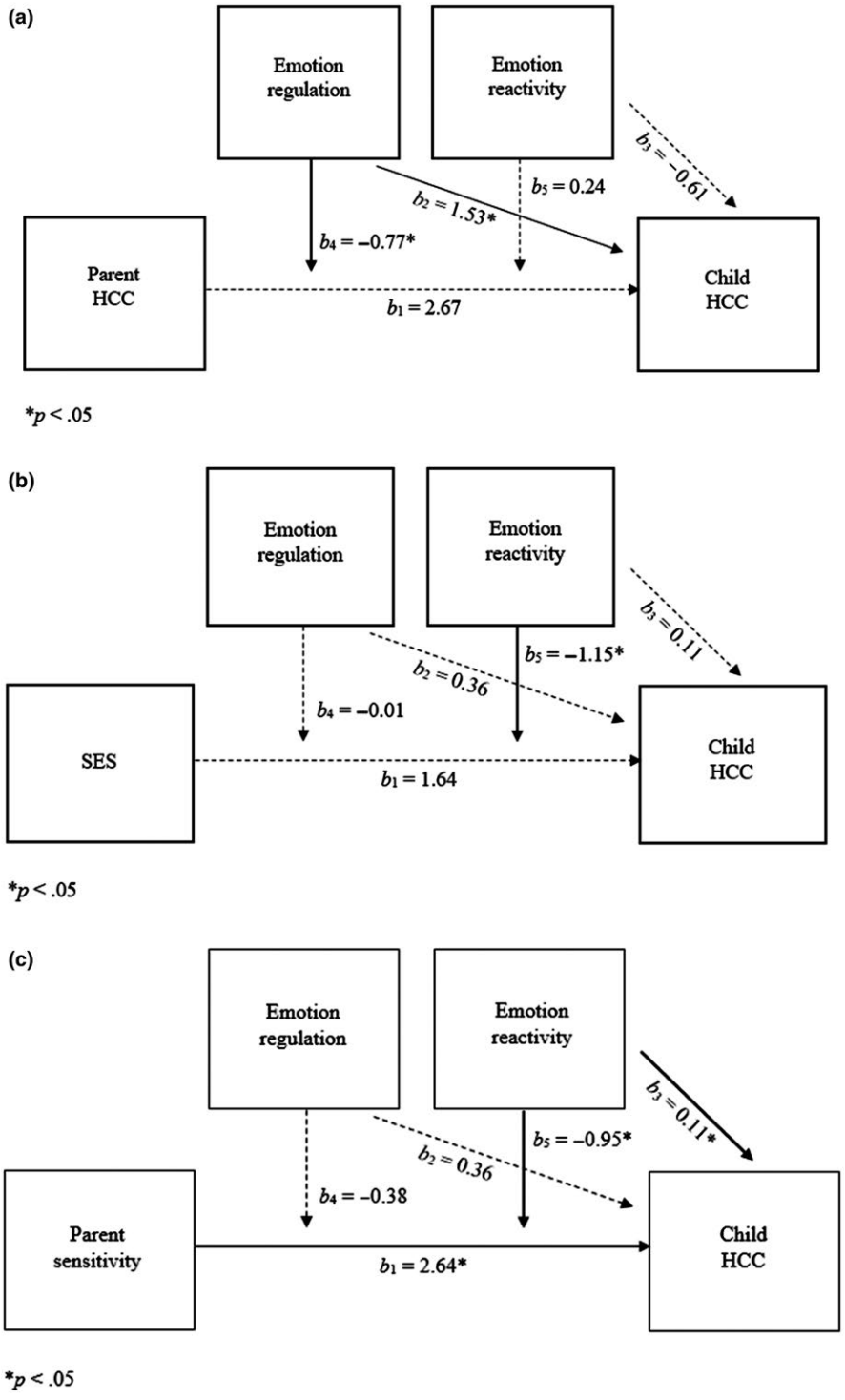

FIGURE 3 (a) A conditional process model between parent and child hair cortisol concentration (HCC), ($F(5, 78) = 9.44$, $p < 0.001$), yielded a main effect of emotion regulation on child HCC ($b_2 = 1.53$, $p = 0.03$, CI: 0.16–2.90), and a significant parent HCC × emotion regulation interaction ($b_4 = -0.77$, $p = 0.02$, CI: −1.43 to −0.11). (b) A conditional process model between socioeconomic status (SES) and child HCC, ($F(5, 79) = 4.84$, $p = 0.001$), yielded a significant SES × emotion regulation interaction ($b_4 = -1.15$, $p = 0.02$, CI: −2.13 to −0.18). (c) A conditional process model between parental sensitivity and child HCC, ($F(5, 79) = 3.23$, $p = 0.01$), yielded a main effect of parental sensitivity on child HCC ($b_1 = 2.64$, $p = 0.05$, CI: 0.02–5.26), a main effect of emotion reactivity on child HCC ($b_3 = 5.25$, $p = 0.003$, CI: 1.81–8.70), and a significant parental sensitivity × emotion regulation interaction ($b_5 = -0.95$, $p = 0.006$, CI: −1.61 to −0.28)

data in the current sample does not suggest that better emotional stability is an overall protective factor for chronic physiological stress, it does suggest that being less emotionally reactive may protect children from negative physiological effects linked to low SES.

Finally, we found that less sensitive caregiving was related to higher child HCC. To our knowledge, we are the first to report an association between parental sensitivity and child HCC. Furthermore, this relationship was amplified by children's emotional reactivity. Findings suggest that children who are more emotionally reactive are more vulnerable to insensitive parenting, such that the association between low parental sensitivity and higher child HCC is stronger than for children who are less emotionally reactive. Taken together, findings indicate that emotionality influences the extent to which children's cumulative physiological stress exposure is impacted by environmental risks. Being more emotionally stable may be particularly helpful if children are exposed to adverse environments, such as poor parenting quality. These results speak to the complex interplay of caregiving

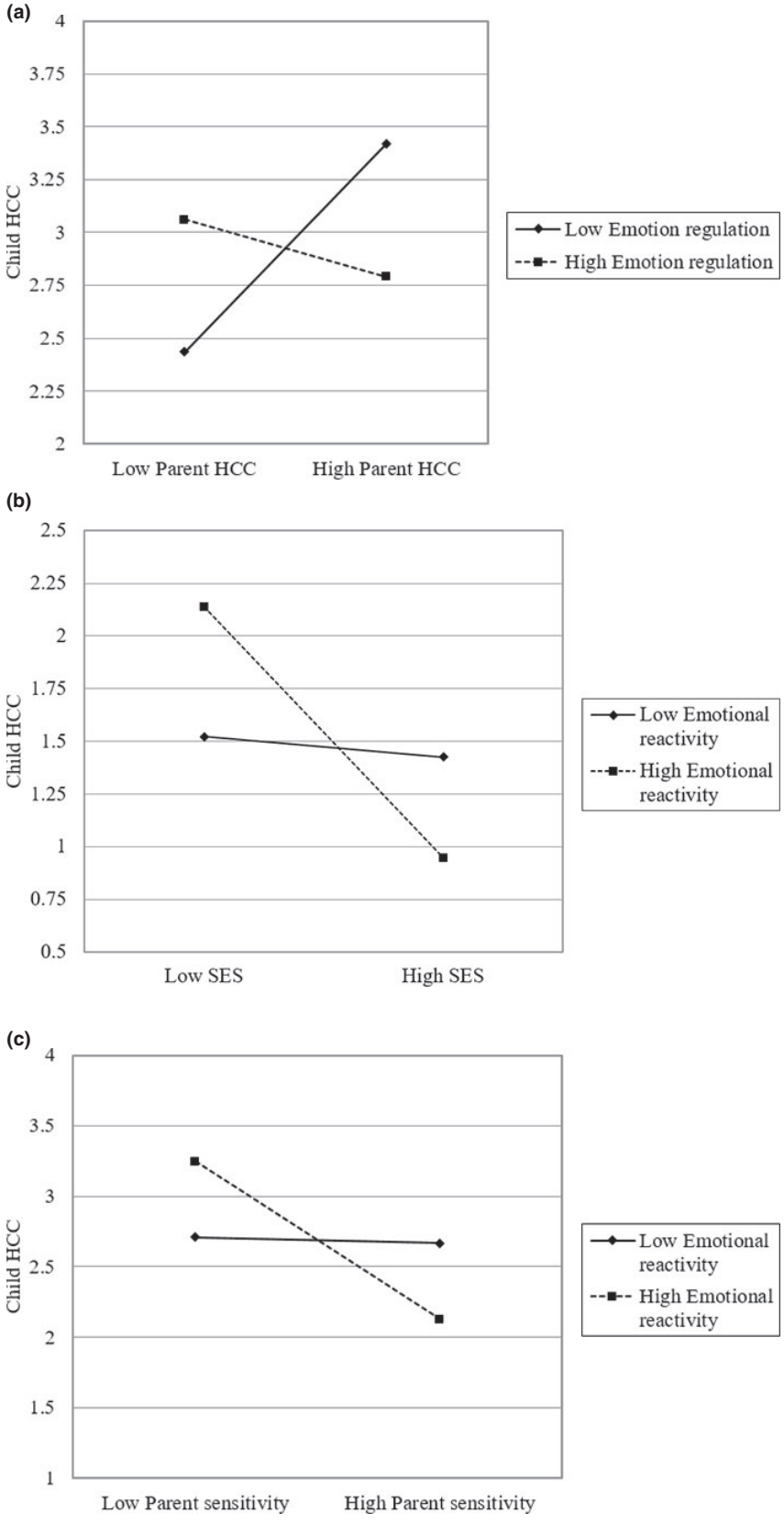

FIGURE 4 (a) Estimated marginal means for child hair cortisol concentration (HCC) by emotion regulation as a function of parent HCC, 1 *SD* below and 1 *SD* above the mean. Among children with poorer emotion regulation skills, child HCC was closely linked to parent HCC, while for children with better emotion regulation skills, the slope was flatter. (b) Estimated marginal means for child HCC by emotion reactivity as a function of socioeconomic status (SES), 1 *SD* below and 1 *SD* above the mean. Among children with higher emotional reactivity, child HCC was closely linked to SES, while for children with less emotional reactivity, the slope was flatter. (c) Estimated marginal means for child HCC by emotion reactivity as a function of parental sensitivity, 1 *SD* below and 1 *SD* above the mean. Among children with higher emotional reactivity, child HCC was closely linked to parental sensitivity, while for children with less emotional reactivity, the slope was flatter

and behavioral and physiological aspects of regulation in early childhood.

It is interesting that emotion regulation uniquely moderated parent and child HCC while emotional reactivity uniquely moderated the relationships between SES and child HCC, as well as parental sensitivity and child HCC. Although related, emotion regulation and emotion reactivity make distinct contributions to children's psychological functioning and researchers continue to

TABLE 5 Model coefficients and summary information for moderation model of socioeconomic status (SES) and child hair cortisol concentration (HCC)

		Child HCC		
Antecedent		**Coeff.**	***SE***	***p***
SES	b_1	1.64	1.82	0.37
Emotion regulation	b_2	0.36	0.30	0.22
Emotion reactivity	b_3	0.11	0.40	0.79
SES × emotion regulation	b_4	−0.01	0.39	0.99
SES × emotion reactivity	b_5	−1.15	0.49	0.02
Constant		1.31	1.40	0.35
		$R^2 = 0.24$		
		$F(5, 79) = 4.84$, $p = 0.001$		

TABLE 6 Model coefficients and summary information for moderation model of parental sensitivity and child hair cortisol concentration (HCC)

		Child HCC		
Antecedent		**Coeff.**	***SE***	***p***
Parental sensitivity	b_1	2.64	1.32	0.05
Emotion regulation	b_2	2.21	1.47	0.14
Emotion reactivity	b_3	5.26	1.73	0.003
Parental sensitivity × emotion regulation	b_4	−0.38	0.29	0.20
Parental sensitivity × emotion reactivity	b_5	−0.95	0.33	0.006
Constant		12.37	6.61	0.06
		$R^2 = 0.17$		
		$F(5, 79) = 3.23$, $p = 0.01$		

emphasize that these two dimensions should be treated as separate constructs (Derryberry & Rothbart, 1997). One possibility of the current differential findings may be attributed to the different developmental timing of emotion regulation and emotion reactivity. Emotion reactivity is considered to be closely aligned with early temperamental characteristics (Rothbart, 1989). Emotion regulation, while evident in the first year of life, becomes more complex later in the preschool period, a time when cognitive skills are rapidly coming online (Carlson, Mandell, & Williams, 2004; Garon, Bryson, & Smith, 2008). In other words, emotion regulation skills are still maturing in the early preschool period while emotion reactivity may already be established. Thus, in the context of high levels of parental chronic physiological stress, low emotion reactivity alone may be insufficient to buffer a child—it may take the more sophisticated skill of emotion regulation. On the other hand, being less emotionally reactive in general meant that children's physiological stress systems were less sensitive to SES and parenting quality, while emotion regulation did not play a role in these associations. Part of emotional reactivity is the degree of emotional lability from the child. Thus, if the child is less emotionally labile, they may not be as vulnerable to environmental instability since their temperamental disposition is more stable in terms of emotionality. Taken together, findings highlight that the extent to which differential emotional capacities attenuate the relationship between risk factors and child HCC may be context specific. Future studies should further examine whether emotion regulation or emotional reactivity moderate the impact of other risk factors for increased HCC.

To our knowledge, this is the first study to examine emotion regulation behaviors in relation to HCC. Our proposed model for the interplay of emotional capacities and child chronic physiological stress was partially supported. Contrary to what we expected, child HCC was not correlated directly with emotion regulation or emotional reactivity. Instead, we found that children's chronic physiological stress was influenced only when emotion regulation and reactivity was paired with risk factors, in this case parental chronic physiological stress and SES. It is particularly striking that despite not having evidence of an overall association, emotion regulation and emotional reactivity were significant moderators. At a global level, the lack of an association between emotional capacities and child HCC may mislead one to think that at this early age, emotion regulation and emotional reactivity do not yet matter for physiological functioning. However, our findings suggest that emotion regulation and emotional reactivity, while still emergent skills at age three, do matter in the context of risk. Children with high emotion regulation skills seem less influenced by low SES context in regards to cumulative cortisol exposure. Children rely on their environments to provide socioemotional support through sensitive parenting or socioeconomic resources. Thus, when these environmental supports are not in place, having better emotional regulatory capacities may reduce vulnerability to certain stressors.

Prior work has found that executive function is related to salivary cortisol measures in young children (Blair, Granger, & Razza, 2005; Blair et al., 2011). Likewise, emotion regulation may be related to acute cortisol levels in this early preschool period. Thus, it may not be until later childhood when regulatory capacities are more established, that emotion regulation would have direct associations with cumulative cortisol exposure. Future studies are needed to empirically test these relationships. Current findings highlight an important contribution, that emergent emotional capacities do have implications for children's chronic physiological stress during a period of rapid cognitive and socioemotional development, particularly in the context of risk.

While others report found a relationship between parenting quality and emotion regulation in older children (Garner & Spears, 2000; Raver, 2004), we did not find this association in our sample of 3.5-year-olds. This suggests that emergent emotion regulation capacities may initially be independent of broad parenting qualities at this young age. One possibility for this null finding is that we measured parental sensitivity during a free-play interaction, which captures more global parenting quality. It is possible that what matters for emotion regulation at this age is not broad parenting quality but a

specific aspect of caregiving that we did not measure explicitly (e.g., emotion talk, or how the parent responds to child distress). Second, it may be that there is something unique about emergent emotion regulation skills that is not fully explained by parenting quality, for example, it could depend on other environmental factors we did not measure. Perhaps genetics or other environmental factors that were not included in this study play a role in individual differences in children's early emotion regulation capacities. However, our findings suggest that individual differences in emergent emotion regulation and early emotionality are not fully explained by a global measure of parental sensitivity.

The role of emotion regulation, and self-control more generally, in psychosocial and biological health outcomes is likely complex and may well vary with developmental stage, with the degree of environmental risk, and/or with the specific outcomes assessed. For example, Miller, Yu, Chen, and Brody (2015) reported that among low-SES adolescents, self-control predicted better psychosocial functioning but this came at a physiological cost, with more rapid immune cell aging. Thus, while the current cross-sectional study offers preliminary evidence that emotion regulation in early childhood may buffer children from chronic physiological stress in the face of environmental risks, much more work will be needed to describe the role of emotion regulation in physiological stress and other health outcomes across development.

While findings from our study contribute to the expanding field of psychosocial stressors and HCC, there are some limitations. First, we cannot determine directionality from our findings. While parent behavioral and physiological regulation (i.e., parental sensitivity and HCC) both related to child physiological regulation (i.e., HCC), there may well be bidirectional relations such that having a physiologically dysregulated child may make parents more behaviorally and physiologically dysregulated themselves. Relatedly, the experience of parenting a highly reactive, labile child may be highly stressful to the parent, impacting both parental sensitivity and parent stress physiology. An important future direction will be to unpack these potential bidirectional interactions, perhaps through a longitudinal study with repeated measures of both parent and child. Additionally, while the aim of this study was to explore and identify candidate variable that matter for biological embedding of stress, it would be valuable to also determine unique effects of each risk factor (e.g., low parental sensitivity, elevated parent HCC, low SES) by including them in one model. Future studies should make sure to include a large enough sample size to insure enough power to be able to tease out individual relationships of risk factors of HCC.

While the ERC is a well-established, widely used instrument of emotion regulation and emotional reactivity in preschool children (Chang, Schwartz, Dodge, & McBride-Chang, 2003; Cohen & Mendez, 2009; Izard et al., 2008), it was a parent-report measure. It would be beneficial for future studies to include an observational measure of emotion regulation in addition to parental report to assess children's emotion regulatory capacities at a behavioral level. We had a rather well-educated, diverse socioeconomic sample that represented a range of SES levels. However, it would be interesting to examine whether results would generalize within extreme ends of the SES gradient or in other areas that have varying ranges of education levels. Furthermore, the current data suggests that the role of emotion regulation in the interplay of environmental risks and physiological stress varies by SES, with emotion regulation playing a particularly salient role in low SES contexts. Therefore, it would be valuable for a future study to recruit a primarily low-SES sample to further elucidate the role of emotion regulation in this context, with attention to multiple SES-linked environmental risks. SES is itself a proxy for multiple risks. Now that we know about this moderating role of emotion regulation by SES, a next step would be to look at these associations in a high poverty sample to further understand the role of emotion regulation and emotional reactivity in how specific environmental risks relate to chronic cortisol exposure.

Additionally, race and SES were confounded in our sample. While we report the difference, we do not control for it because being and SES are so confounded and because we do not have enough within specific ethnic groups. Our study focused on a narrow age range of 3.5-year-olds, when emotion regulation capacities are just emerging, and it would be interesting to explore if the pattern of associations might vary in different developmental periods. Finally, the participating parents were mostly mothers. Future studies should try to incorporate equal numbers of mothers and fathers to compare whether the relationship between parent and child HCC differed depending on the parent's sex.

5 | CONCLUSIONS

Chronic physiological stress can have detrimental health outcomes. Therefore, it is essential to examine how certain traits, such as emotion regulation, can attenuate the relationship between risk factors and physiological functioning. Results from the current study provide empirical support that in early childhood, adaptive emotional capacities allow children to be less dependent on their environment. Findings suggest that in the context of risk, that is, for children exposed to increased parental chronic physiological stress, lower SES and poor parenting, better emotion regulation skills may buffer children from chronic physiological stress. Especially when environmental supports are not in place early in childhood, our study underscores that individual emotion regulation capacities can play a crucial role in pathways of resilience at a biological level. Preschool children may have differential outcomes in the face of adversity depending on their emotion regulation abilities. More research is needed to examine the nuanced interplay of acute and chronic physiological stress with emotion regulation in early childhood.

ORCID

Katie Kao https://orcid.org/0000-0003-0697-5071

REFERENCES

Bartels, M., de Geus, E. J., Kirschbaum, C., Sluyter, F., & Boomsma, D. I. (2003). Heritability of daytime cortisol levels in children. *Behavior Genetics*, *33*, 421–433. https://doi.org/10.1023/A:1025321609994

Bevans, K., Cerbone, A., & Overstreet, S. (2008). Relations between recurrent trauma exposure and recent life stress and salivary cortisol among children. *Development and Psychopathology*, *20*(1), 257–272. https://doi.org/10.1017/S0954579408000126

Biringen, Z., Derscheid, D., Vliegen, N., Closson, L., & Easterbrooks, M. A. (2014). Emotional availability (EA): Theoretical background, empirical research using the EA Scales, and clinical applications. *Developmental Review*, *34*(2), 114–167. https://doi.org/10.1016/j.dr.2014.01.002

Biringen, Z., & Easterbrooks, M. A. (2012). Emotional availability: Concept, research, and window on developmental psychopathology. *Development and Psychopathology*, *24*(1), 1–8. https://doi.org/10.1017/S0954579411000617

Blair, C., Granger, D., & Razza, R. P. (2005). Cortisol reactivity is positively related to executive function in preschool children attending Head Start. *Child Development*, *76*(3), 554–567. https://doi.org/10.1111/j.1467-8624.2005.00863.x

Blair, C., Granger, D. A., Willoughby, M., Mills-Koonce, R., Cox, M., Greenberg, M. T., … Fortunato, C. K. (2011). Salivary cortisol mediates effects of poverty and parenting on executive functions in early childhood. *Child Development*, *82*(6), 1970–1984. https://doi.org/10.1111/j.1467-8624.2011.01643.x

Blandon, A. Y., Calkins, S. D., Keane, S. P., & O'Brien, M. (2008). Individual differences in trajectories of emotion regulation processes: The effects of maternal depressive symptomatology and children's physiological regulation. *Developmental Psychology*, *44*(4), 1110–1123. https://doi.org/10.1037/0012-1649.44.4.1110

Calkins, S. D. (1994). Origins and outcomes of individual differences in emotion regulation. *Monographs of the Society for Research in Child Development*, *59*(2–3), 53–72. j.1540-5834.1994.tb01277.x

Carlson, S. M., Mandell, D. J., & Williams, L. (2004). Executive function and theory of mind: Stability and prediction from ages 2 to 3. *Developmental Psychology*, *40*(6), 1105. https://doi.org/10.1037/0012-1649.40.6.1105

Carlson, S. M., Moses, L. J., & Claxton, L. J. (2004). Individual differences in executive functioning and theory of mind: An investigation of inhibitory control and planning ability. *Journal of Experimental Child Psychology*, *87*(4), 299–319. https://doi.org/10.1016/j.jecp.2004.01.002

Cassano, M., Perry-Parrish, C., & Zeman, J. (2007). Influence of gender on parental socialization of children's sadness regulation. *Social Development*, *16*(2), 210–231. https://doi.org/10.1111/j.1467-9507.2007.00381.x

Chang, L., Schwartz, D., Dodge, K. A., & McBride-Chang, C. (2003). Harsh parenting in relation to child emotion regulation and aggression. *Journal of Family Psychology*, *17*(4), 598–606. https://doi.org/10.1037/0893-3200.17.4.598

Clearfield, M. W., Carter-Rodriguez, A., Merali, A.-R., & Shober, R. (2014). The effects of SES on infant and maternal diurnal salivary cortisol output. *Infant Behavior and Development*, *37*(3), 298–304. https://doi.org/10.1016/j.infbeh.2014.04.008

Cohen, J. S., & Mendez, J. L. (2009). Emotion regulation, language ability, and the stability of preschool children's peer play behavior. *Early Education & Development*, *20*(6), 1016–1037. https://doi.org/10.1080/10409280903305716

Cole, P. M., Martin, S. E., & Dennis, T. A. (2004). Emotion regulation as a scientific construct: Methodological challenges and directions for child development research. *Child Development*, *75*(2), 317–333. https://doi.org/10.1111/j.1467-8624.2004.00673.x

Danese, A., & McEwen, B. S. (2012). Adverse childhood experiences, allostasis, allostatic load, and age-related disease. *Physiology & Behavior*, *106*(1), 29–39. https://doi.org/10.1016/j.physbeh.2011.08.019

Davenport, M. D., Tiefenbacher, S., Lutz, C. K., Novak, M. A., & Meyer, J. S. (2006). Analysis of endogenous cortisol concentrations in the hair of rhesus macaques. *General and Comparative Endocrinology*, *147*, 255–261. https://doi.org/10.1016/j.ygcen.2006.01.005

Denham, S., & Kochanoff, A. T. (2002). Parental contributions to preschoolers' understanding of emotion. *Marriage & Family Review*, *34*(3–4), 311–343. https://doi.org/10.1300/J002v34n03_06

Dennis, T. (2006). Emotional self-regulation in preschoolers: The interplay of child approach reactivity, parenting, and control capacities. *Developmental Psychology*, *42*(1), 84. https://doi.org/10.1037/0012-1649.42.1.84

Derryberry, D., & Rothbart, M. K. (1997). Reactive and effortful processes in the organization of temperament. *Development and Psychopathology*, *9*(4), 633–652. https://doi.org/10.1017/S0954579497001375

Duncombe, M. E., Havighurst, S. S., Holland, K. A., & Frankling, E. J. (2012). The contribution of parenting practices and parent emotion factors in children at risk for disruptive behavior disorders. *Child Psychiatry & Human Development*, *43*(5), 715–733. https://doi.org/10.1007/s10578-012-0290-5

Eisenberg, N., Cumberland, A., Spinrad, T. L., Fabes, R. A., Shepard, S. A., Reiser, M., … Guthrie, I. K. (2001). The relations of regulation and emotionality to children's externalizing and internalizing problem behavior. *Child Development*, *72*(4), 1112–1134. https://doi.org/10.1111/1467-8624.00337

Eisenberg, N., Fabes, R. A., Guthrie, I. K., & Reiser, M. (2000). Dispositional emotionality and regulation: Their role in predicting quality of social functioning. *Journal of Personality and Social Psychology*, *78*(1), 136–157. https://doi.org/10.1037//0022-3514.78.1.136

Evans, G. W., & English, K. (2002). The environment of poverty: Multiple stressor exposure, psychophysiological stress, and socioemotional adjustment. *Child Development*, *73*(4), 1238–1248. https://doi.org/10.1111/1467-8624.00469

Fairbanks, L. A., Jorgensen, M. J., Bailey, J. N., Breidenthal, S. E., Grzywa, R., & Laudenslager, M. L. (2011). Heritability and genetic correlation of hair cortisol in vervet monkeys in low and higher stress environments. *Psychoneuroendocrinology*, *36*, 1201–1208. https://doi.org/10.106/j.psyneuen.2011.02.013

Flom, M., St John, A. M., Meyer, J. S., & Tarullo, A. R. (2017). Infant hair cortisol: Associations with salivary cortisol and environmental context. *Developmental Psychobiology*, *59*, 26–38. https://doi.org/10.1002/dev.21449

Garner, P. W., & Spears, F. M. (2000). Emotion regulation in low-income preschoolers. *Social Development*, *9*(2), 246–264. https://doi.org/10.1111/1467-9507.00122

Garon, N., Bryson, S. E., & Smith, I. M. (2008). Executive function in preschoolers: A review using an integrative framework. *Psychological Bulletin*, *134*(1), 31. https://doi.org/10.1037/0033-2909.134.1.31

Gershoff, E. T., Aber, J. L., Raver, C. C., & Lennon, M. C. (2007). Income is not enough: Incorporating material hardship into models of income associations with parenting and child development. *Child Development*, *78*(1), 70–95. https://doi.org/10.1111/j.1467-8624.2007.00986.x

Graziano, P. A., Reavis, R. D., Keane, S. P., & Calkins, S. D. (2007). The role of emotion regulation in children's early academic success. *Journal of School Psychology*, *45*(1), 3–19. https://doi.org/10.1016/j.jsp.2006.09.002

Gross, J. J., & John, O. P. (2003). Individual differences in two emotion regulation processes: Implications for affect, relationships, and well-being. *Journal of Personality and Social Psychology*, *85*(2), 348–362. https://doi.org/10.1037/0022-3514.85.2.348

Gunnar, M., & Talge, N. M. (2008). Neuroendocrine measures in developmental research. In L. A. Schmidt, & S. J. Segalowitz (Eds.), *Developmental psychophysiology: Theory, systems, and methods* (pp. 343–364). New York: Cambridge University Press.

Hammond, S. I., Müller, U., Carpendale, J. I., Bibok, M. B., & Liebermann-Finestone, D. P. (2012). The effects of parental scaffolding on preschoolers' executive function. *Developmental Psychology*, *48*(1), 271. https://doi.org/10.1037/a0025519

Hane, A. A., Henderson, H. A., Reeb-Sutherland, B. C., & Fox, N. A. (2010). Ordinary variations in human maternal caregiving in infancy and biobehavioral development in early childhood: A follow-up study. *Developmental Psychobiology*, *52*(6), 558–567. https://doi.org/10.1002/dev.20461

Hayes, A. F. (2013). *Introduction to mediation, moderation, and conditional process analysis: A regression-based approach*. New York: Guilford Press.

Hoffman, M. C., Karban, L. V., Benitez, P., Goodteacher, A., & Laudenslager, M. L. (2014). Chemical processing and shampooing impact cortisol measured in human hair. *Clinical and Investigative Medicine*, *37*, E252–E257. https://doi.org/10.25011/cim.v37i4.21731

Izard, C. E., King, K. A., Trentacosta, C. J., Morgan, J. K., Laurenceau, J.-P., Krauthamer-Ewing, E. S., & Finlon, K. J.. (2008). Accelerating the development of emotion competence in Head Start children: Effects on adaptive and maladaptive behavior. *Development and Psychopathology*, *20*(01), https://doi.org/10.1017/S0954579408000175.

Jaffe, M., Gullone, E., & Hughes, E. K. (2010). The roles of temperamental dispositions and perceived parenting behaviours in the use of two emotion regulation strategies in late childhood. *Journal of Applied Developmental Psychology*, *31*(1), 47–59. https://doi.org/10.1016/j.appdev.2009.07.008

Juster, R.-P., McEwen, B. S., & Lupien, S. J. (2010). Allostatic load biomarkers of chronic stress and impact on health and cognition. *Neuroscience & Biobehavioral Reviews*, *35*(1), 2–16. https://doi.org/10.1016/j.neubiorev.2009.10.002

Kagan, J. (1994). On the nature of emotion. *Monographs of the Society for Research in Child Development*, *59*(2–3), 7–24. https://doi.org/10.1111/j.1540-5834.1994.tb01275.x

Kao, K., Doan, S. N., St. John, A. M., Meyer, J. S., & Tarullo, A. R. (2018). Salivary cortisol reactivity in preschoolers is associated with hair cortisol and behavioral problems. *Stress*, *21*(1), 28–35. https://doi.org/10.1080/10253890.2017.1391210

Karlén, J., Frostell, A., Theodorsson, E., Faresjö, T., & Ludvigsson, J. (2013). Maternal influence on child HPA axis: A prospective study of cortisol levels in hair. *Pediatrics*, *132*, e1333–e1340. https://doi.org/10.1542/peds.2013-1178

Karlén, J., Ludvigsson, J., Hedmark, M., Faresjö, Å., Theodorsson, E., & Faresjö, T. (2015). Early psychosocial exposures, hair cortisol levels, and disease risk. *Pediatrics*, *135*, e1450–e1457. https://doi.org/10.1542/peds.2014-2561

Keenan, K. (2006). Emotion dysregulation as a risk factor for child psychopathology. *Clinical Psychology: Science and Practice*, *7*(4), 418–434. https://doi.org/10.1093/clipsy.7.4.418

Kim, J., & Cicchetti, D. (2009). Longitudinal pathways linking child maltreatment, emotion regulation, peer relations, and psychopathology: Pathways linking maltreatment, emotion regulation, and psychopathology. *Journal of Child Psychology and Psychiatry*, *51*(6), 706–716. https://doi.org/10.1111/j.1469-7610.2009.02202.x

King, S. L., & Hegadoren, K. M. (2002). Stress hormones: How do they measure up? *Biological Research for Nursing*, *4*(2), 92–103. https://doi.org/10.1177/1099800402238334

Klaassens, E. R., Giltay, E. J., van Veen, T., Veen, G., & Zitman, F. G. (2010). Trauma exposure in relation to basal salivary cortisol and the hormone response to the dexamethasone/CRH test in male railway employees without lifetime psychopathology. *Psychoneuroendocrinology*, *35*(6), 878–886. https://doi.org/10.1016/j.psyneuen.2009.11.012.

Kopp, C. B., & Neufeld, S. J. (2003). Emotional development during infancy. In R. J. Davidson, K. R. Scherer, & H. H. Goldsmith (Eds.), *Handbook of affective sciences* (pp. 347–374). New York: Oxford University Press.

LeBeau, M. A., Montgomery, M. A., & Brewer, J. D. (2011). The role of variations in growth rate and sample collection on interpreting results of segmental analyses of hair. *Forensic Science International*, *210*, 110–116. https://doi.org/10.1016/j.forsciint.2011.02.015

Lengua, L. J. (2002). The contribution of emotionality and self-regulation to the understanding of children's response to multiple risk. *Child Development*, *73*(1), 144–161. https://doi.org/10.1111/1467-8624.00397

Letourneau, N., Watson, B., Duffett-Leger, L., Hegadoren, K., & Tryphonopoulos, P. (2011). Cortisol patterns of depressed mothers and their infants are related to maternal–infant interactive behaviours. *Journal of Reproductive and Infant Psychology*, *29*(5), 439–459. https://doi.org/10.1080/02646838.2011.649474

Lupien, S. J., King, S., Meaney, M. J., & McEwen, B. S. (2000). Child's stress hormone levels correlate with mother's socioeconomic status and depressive state. *Biological Psychiatry*, *48*, 976–980. https://doi.org/10.1016/S0006-3223(00)00965-3

McEwen, B. S., & Stellar, E. (1993). Stress and the individual: Mechanisms leading to disease. *Archives of Internal Medicine*, *153*(18), 2093–2101. https://doi.org/10.1001/archinte.1993.00410180039004

Meyer, J. S., & Novak, M. A. (2012). Minireview: hair cortisol: a novel biomarker of hypothalamic-pituitary-adrenocortical activity. *Endocrinology*, *153*(9), 4120–4127. https:/doi.org/10.1210/en.2012-1226

Meyer, J., Novak, M., Hamel, A., & Rosenberg, K. (2014). Extraction and analysis of cortisol from human and monkey hair. *Journal of Visualized Experiments*, *83*, e50882. https://doi.org/10.3791/50882

Miller, G., Yu, T., Chen, E., & Brody, G. (2015). Self-control forecasts better psychosocial outcomes but faster epigenetic aging in low-SES youth. *Proceedings of the National Academy of Sciences of the United States of America*, *112*, 10325–10330. https://doi.org/10.1073/pnas.1505063112

Morris, A. S., Silk, J. S., Steinberg, L., Myers, S. S., & Robinson, L. R. (2007). The role of the family context in the development of emotion regulation. *Social Development*, *16*(2), 361–388. https://doi.org/10.1111/j.1467-9507.2007.00389.x

Morris, A. S., Silk, J. S., Steinberg, L., Sessa, F. M., Avenevoli, S., & Essex, M. J. (2002). Temperamental vulnerability and negative parenting as interacting predictors of child adjustment. *Journal of Marriage and the Family*, *64*, 461–471. https://doi.org/10.1111/j.1741-3737.2002.00461.x

Murphy, B. C., Eisenberg, N., Fabes, R. A., Shepard, S., & Guthrie, I. K. (1999). Consistency and change in children's emotionality and regulation: A longitudinal study. *Merrill-Palmer Quarterly*, *45*, 413–444. https://jstor.org/stable/23092580

Ouellet-Morin, I., Boivin, M., Dionne, G., Lupien, S. J., Arsenault, L., Barr, R. G., … Tremblay, R. E. (2008). Variations in heritability of cortisol reactivity to stress as a function of early familial adversity among 19-month-old twins. *Archives of General Psychiatry*, *65*, 211–218. https://doi.org/10.1001/archgenpsychiatry.2007.27

Ouellet-Morin, I., Dionne, G., Pérusse, D., Lupien, S. J., Arseneault, L., Barr, R. G., … Boivin, M. (2009). Daytime cortisol secretion in 6-month-old twins: Genetic and environmental contributions as a function of early familial adversity. *Biological Psychiatry*, *65*, 409–416. https://doi.org/10.1016/j.biopsych.2008.10.003

Ouellette, S. J., Russell, E., Kryski, K. R., Sheikh, H. I., Singh, S. M., Koren, G., & Hayden, E. P. (2015). Hair cortisol concentrations in higher- and lower-stress mother–daughter dyads: A pilot study of associations and moderators. *Developmental Psychobiology*, *57*, 519–534. https://doi.org/10.1002/dev.21302

Philbrook, L. E., Hozella, A. C., Kim, B. R., Jian, N., Shimizu, M., & Teti, D. M. (2014). Maternal emotional availability at bedtime and infant cortisol at 1 and 3 months. *Early Human Development*, *90*(10), 595–605. https://doi.org/10.1016/j.earlhumdev.2014.05.014

Pipp-Siegel, S., & Biringen, Z. (1998). Assessing the quality of relationships between parents and children: The emotional availability scales. *The Volta Review*.

Raver, C. C. (2004). Placing emotional self-regulation in sociocultural and socioeconomic contexts. *Child Development*, *75*(2), 346–353. https://doi.org/10.1111/j.1467-8624.2004.00676.x

Rothbart, M. K. (1989). Temperament in childhood: A framework. In G. A. Kohnstamm, J. E. Bates, & M. K. Rothbart (Eds.), *Temperament in childhood* (pp. 59–73). Oxford, England: John Wiley & Sons.

Rothbart, M. K., & Bates, J. E. (2006). Temperament. In N. Eisenberg, & W. Damon (Eds.), *Handbook of child psychology: Vol. 3. Social, emotional, and personality development* (6th ed., pp. 99–166). New York: Wiley.

Russell, E., Koren, G., Rieder, M., & Van Uum, S. (2012). Hair cortisol as a biological marker of chronic stress: Current status, future directions and unanswered questions. *Psychoneuroendocrinology*, *37*(5), 589–601. https://doi.org/10.1016/j.psyneuen.2011.09.009

Rydell, A.-M., Berlin, L., & Bohlin, G. (2003). Emotionality, emotion regulation, and adaptation among 5- to 8-year-old children. *Emotion*, *3*(1), 30–47. https://doi.org/10.1037/1528-3542.3.1.30

Shields, A., & Cicchetti, D. (1997). Emotion regulation among school-age children: The development and validation of a new criterion Q-sort scale. *Developmental Psychology*, *33*(6), 906–916. https://doi.org/10.1037//0012-1649.33.6.906

Shipman, K., Schneider, R., & Brown, A. (2004). Emotion dysregulation and psychopathology. In M. Beauregard (Ed.), *Consciousness, emotional self regulation, and the brain* (pp. 61–85). Amsterdam, Netherlands: John Benjamins.

Shonkoff, J. P. (2010). Building a new biodevelopmental framework to guide the future of early childhood policy. *Child Development*, *81*, 357–367. https://doi.org/10.1111/j.1467-8624.2009.01399.x

Shonkoff, J. P., Garner, A. S., Siegel, B. S., Dobbins, M. I., Earls, M. F., Garner, A. S., … Wood, D. l. (2012). The lifelong effects of early childhood adversity and toxic stress. *Pediatrics*, *129*, e232. https://doi.org/10.1542/peds.2011-2663

Stenius, F., Theorell, T., Lilja, G., Scheynius, A., Alm, J., & Lindblad, F. (2008). Comparisons between salivary cortisol levels in six-months-olds and their parents. *Psychoneuroendocrinology*, *33*, 352–359. https://doi.org/10.1016/j.psyneuen.2007.12.001

Tarullo, A. R., St. John, A. M., & Meyer, J. S. (2017). Chronic stress in the mother-infant dyad: Maternal hair cortisol, infant salivary cortisol and interactional synchrony. *Infant Behavior and Development*, *47*, 92–102. https://doi.org/10.1016/j.infbeh.2017.03.007

Taylor, S. E., Way, B. M., & Seeman, T. E. (2011). Early adversity and adult health outcomes. *Development and Psychopathology*, *23*(03), 939–954. https://doi.org/10.1017/S0954579411000411

Thompson, R. A., & Meyer, S. (2007). Socialization of emotion regulation in the family. In J. J. Gross (Ed.), *Handbook of emotion regulation* (pp. 249–268). New York, NY: Guilford Press.

Tucker-Drob, E. M., Grotzinger, A. D., Briley, D. A., Engelhardt, L. E., Mann, F. D., Patterson, M., … Harden, K. P. (2017). Genetic influences on hormonal markers of chronic hypothalamic–pituitary–adrenal function in human hair. *Psychological Medicine*, *47*, 1389–1401. https://doi.org/10.1017/S0033291716003068

Underwood, M. K. (1997). Top ten pressing questions about the development of emotion regulation. *Motivation and Emotion*, *21*(1), 127–146. https://doi.org/10.1023/A:1024482516226

Vaghri, Z., Guhn, M., Weinberg, J., Grunau, R. E., Yu, W., & Hertzman, C. (2013). Hair cortisol reflects socio-economic factors and hair zinc in preschoolers. *Psychoneuroendocrinology*, *38*(3), 331–340. https://doi.org/10.1016/j.psyneuen.2012.06.009

Vanaelst, B., Michels, N., De Vriendt, T., Huybrechts, I., Vyncke, K., Sioen, I., … De Henauw, S. (2013). Cortisone in hair of elementary school girls and its relationship with childhood stress. *European Journal of Pediatrics*, *172*, 843–846. https://doi.org/10.1007/s00431-013-1955-1

Vliegenthart, J., Noppe, G., van Rossum, E. F. C., Koper, J. W., Raat, H., & van den Akker, E. L. T. (2016). Socioeconomic status in children is associated with hair cortisol levels as a biological measure of chronic stress. *Psychoneuroendocrinology*, *65*, 9–14. https://doi.org/10.1016/j.psyneuen.2015.11.022

Wosu, A. C., Valdimarsdóttir, U., Shields, A. E., Williams, D. R., & Williams, M. A. (2013). Correlates of cortisol in human hair: Implications for epidemiologic studies on health effects of chronic stress. *Annals of Epidemiology*, *23*(12), 797–811.e2. https://doi.org/10.1016/j.annepidem.2013.09.006

Yap, M. B., Allen, N. B., & Ladouceur, C. D. (2008). Maternal socialization of positive affect: The impact of invalidation on adolescent emotion regulation and depressive symptomatology. *Child Development*, *79*(5), 1415–1431. https://doi.org/10.1111/j.1467-8624.2008.01196.x

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