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The role of social buffering on chronic disruptions in quality of care: evidence from caregiver-based interventions in foster children

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Abstract

There is growing evidence that social support can buffer the physiological stress response, specifically cortisol reactivity. We use a developmental framework to review the importance of social buffering in early childhood, a period of heightened plasticity for programming of the hypothalamic-pituitary-adrenal (HPA) axis. The social environment, in which parents play the largest role in early life, is a critical agent in the developmental trajectory of the HPA axis. A prevailing model of social buffering primarily focuses on the role of social support in the context of acute stressors and cortisol response. This review expands this model to provide evidence of the mechanism of social buffering, or lack thereof, across periods of chronic stress by applying the social buffer model to children involved in the child welfare system. We also highlight current interventions that capitalize on the mechanism of social buffering to modify HPA axis functioning across childhood. Last, we synthesize our findings using the social buffering framework to inform future targeted interventions.

Keywords

Neglect; social support; development; cortisol; intervention

Overview

Early life is a critical period in the programming of the hypothalamic-pituitary-adrenal (HPA) axis, which is essential for maintaining homeostatic balance in the face of stressors (Lupien, McEwen, Gunnar, & Heim, 2009). The social environment plays a crucial role in HPA axis development during this period of heightened plasticity (Lupien et al., 2009). In infancy and childhood, the social environment typically comprises a primary caregiver who is the key external regulator of HPA axis activity. “Social buffering” refers to the ability of social factors (i.e., the primary caregiver) to regulate or buffer (i.e., dampen) the

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physiological response to a stressor. When a caregiver's ability to serve as a social buffer is compromised, child HPA axis development is affected (Hostinar, Sullivan, & Gunnar, 2014).

Research about social buffering may explicate effects of the early caregiving environment on typical and atypical development of the HPA axis; however, much of the work to date has focused on the effect of social support on cortisol reactivity to an acute stressor (Hostinar et al., 2014). This work has greatly increased our understanding of the mechanistic specificity by which caregiver support buffers against stressful early experiences but is also limited in its ability to model the full range of adverse experiences to which children are exposed. In addition to highlighting the effects of acute stressors, research has also shown that the production of basal cortisol is dampened by social support (Hostinar et al., 2014). This review expands the current social buffering framework to incorporate evidence of the mechanistic role of social support in diurnal cortisol patterning in environments of chronic stress. Investigations of stress regulation and the effects of social buffering across time enable us to characterize diurnal cortisol patterning in the home environment in relation to ongoing parenting behaviors.

This review focuses on the influence of caregiver support, and lack thereof, on diurnal cortisol patterning for young children in foster care, many of whom experience early and ongoing adversity (see Figure 1). The goals of this review are to (a) review the literature about diurnal cortisol patterning in foster children, with an emphasis on the common experience of neglect (i.e., lack of a social buffer); (b) review evidence that caregiver-based interventions that target responsive parenting strategies affect HPA axis functioning in foster children; and (c) synthesize our findings using the social buffering framework to inform future targeted interventions.

Caregiver quality and HPA axis patterning

In addition to providing a social safety net for maltreated children whose birth parents are unable to provide a safe environment, from a scientific perspective, foster care also represents a valuable "natural experiment" for examining the effects of limited social buffering on children's HPA axis development. Although foster children experience a range of chronic stress, neglect is the hallmark experience leading to out-of-home placement: In a 2013 report, neglect was the most commonly reported type of maltreatment (79.5%; US DHHS, 2015). "Neglect" refers to a caregiver's failure to provide in terms of a child's physical, medical, emotional, and/or supervisory needs (US DHHS, 2015). Within the social-buffering framework, neglect can be conceptualized as the lack of, or absence of, a reliable social buffer during a developmental period when external regulation by caregivers is critical, with consequences for the typical development of the HPA axis and the health and behavioral outcomes influenced by HPA axis activity.

A sizable literature demonstrates the impact of early-life neglect on HPA axis functioning in foster children. Though this literature clearly suggests that experiences of neglect affect the HPA axis, the specific nature of this effect varies. The most commonly documented effect of neglect on HPA axis functioning is a blunting of diurnal cortisol (sometimes referred to as *hypocortisolism*; Badanes, Watamura, & Hankin, 2011; Gunnar & Vazquez, 2001). When

the association between neglect and hypocortisolism was first observed, findings were inconsistent with the prevailing consensus in the field that experiences of stress lead to elevated cortisol (Gunnar & Vazquez, 2001). It is important to note that hypocortisolism does not confer the same benefits as blunted cortisol reactivity following successful social buffering. In fact, a peak in diurnal cortisol in the morning has numerous benefits, while a blunted response has been associated with deleterious effects (for full review, see Gunnar & Quevedo, 2007). The consistent finding of hypocortisolism across populations whose early adverse experience largely comprised neglect, such as previously institutionalized children and foster children, challenged the field to consider negative effects on HPA axis functioning specific to the absence of responsive caregiving in addition to effects associated with experiences of abuse (i.e., omission as opposed to commission; Bruce, Gunnar, Pears, & Fisher, 2013; McLaughlin et al., 2015).

Research by our group and others has demonstrated a relationship between neglect and hypocortisolism in young foster children. In a sample of preschool-age foster children, our group found the severity of experienced physical neglect to be associated with lower morning cortisol levels (Bruce, Fisher, Pears, & Levine, 2009). In addition, when compared with a low-income community control sample, these foster children were significantly more likely to show low morning cortisol levels (approximately 30% of foster vs. 10% of control children; Bruce et al., 2009). Dozier and colleagues (2006) found foster infants and toddlers, approximately 86% of whom were in care because of neglect, to be more likely to exhibit atypically low or high cortisol patterning at any point across the day than were community controls. Notably, foster children had significantly lower morning cortisol specifically, again pointing to the consistency of the neglect–hypocortisolism relationship.

Much of the initial research on cortisol in foster children has been cross-sectional in nature. More recent longitudinal analyses have shown that stability of HPA axis functioning over time may be an additional indicator of early stress effects in foster children. Evidence suggests that diurnal cortisol levels are not only lower but also more variable over time in foster children than in controls (Laurent, Gilliam, Bruce, & Fisher, 2014).

A key theoretical link for the extension of the social buffer model from acute stress response contexts to chronic stress response exists in the relationship between acute cortisol reactivity and diurnal cortisol functioning (see Figure 1). Few human studies have directly examined the link between diurnal and acute HPA activity. Recent work from our group that is particularly relevant to our working extension of the social buffering model demonstrates that diurnal cortisol levels measured in a group of children with varying levels of risk (i.e., CWS involvement vs. low income) at regular intervals during the preschool years predicted cortisol response to a laboratory stressor during middle childhood (Laurent, Gilliam, Wright, & Fisher, 2015). Though this study does not specifically address the influence of particular early caregiving environments on the relationship between diurnal cortisol and subsequent response for a stressor, it does serve as a critical first step in the demonstration of this relationship upon which future studies can build to test the moderating effects of early caregiving over time.

The exact mechanism through which the social environment affects the HPA axis is challenging to determine because dysfunction could exist at multiple entry points of the system. In humans, measurement of HPA function is typically restricted to cortisol, the end product of HPA activity, due to the level of invasiveness inherent in measuring other products of the system. Animal models have been critical in providing insight into different candidate mechanisms. The HPA axis operates by means of a hormonal cascade: corticotropin-releasing hormone (CRH) is released from the hypothalamus, stimulating the pituitary to release adrenocorticotropin hormone (ACTH), which in turn stimulates the release of cortisol from the adrenal gland. Regulation of the HPA axis is governed by both a direct and indirect negative feedback mechanism; under typical conditions, increased cortisol concentrations directly inhibit the release of CRH from the hypothalamus and ACTH from the anterior pituitary. However, the input into the hypothalamus is multidetermined and includes input from somatosensory, limbic, and higher cortical regions; therefore, glucocorticoid concentrations can indirectly inhibit HPA activity by binding to these glucocorticoid receptor (GR) dense brain structures (Strüber, Strüber, & Roth, 2014). Strüber et al. (2014) propose two pathways through which early life stress can differentially alter cortisol functioning based on quality and quantity of maternal care, suggesting early life stress accompanied by high maternal care results in increased GR expression, whereas early life stress in the absence of high maternal care (e.g., neglect, poor quality care) can result in chronic elevated glucocorticoid concentrations, which in turn leads to a downregulation of GR expression (for full review, see Strüber et al., 2014). Though the measurement of receptor densities requires a level of invasiveness not feasible in humans, the evidence from animal model reviewed above suggests that the differential density of GR expression in brain regions impacting the HPA is a likely candidate mechanism for social environment effects on cortisol functioning.

Caregiver-based interventions on HPA axis patterning

Caregiver-based interventions to improve outcomes for young foster children may function by affecting the quality of the social buffer in the child's life. Strikingly, the interventions reviewed here have also been shown to affect HPA axis functioning. Though these interventions were not explicitly designed to target social buffering, these studies offer examples of opportunities to probe the plasticity of systems such as the HPA axis that we know to be affected by social buffering. In addition, though the specific physiological mechanisms by which these interventions affect HPA functioning remain unclear because of the complexity of this system mentioned previously, these intervention studies demonstrate that the HPA axis is indeed malleable and sensitive to consistent, responsive parenting. Evidence for the effectiveness of these interventions that target consistent, responsive parenting is reviewed in the following paragraphs, with an emphasis on the ways in which this evidence maps onto the social buffering framework.

Multidimensional Treatment Foster Care for Preschoolers (MTFC-P) is a multicomponent intervention aimed at improving outcomes for preschool-age foster children by increasing caregivers' positive reinforcement and consistent, nonharsh limit setting (for more details, see Fisher, Ellis, & Chamberlain, 1999). From a social buffering perspective, a mechanism of MTFC-P's action is to increase the experience of reliable and consistent caregiving while

in a foster placement and across transitions in caregivers. Results of a randomized, controlled trial showed a preventative impact of the intervention on the HPA axis; specifically, the intervention group did not display the same flattening of diurnal cortisol across time as did the comparison group of children in regular foster care, and the intervention group had cortisol trajectories similar to those of a low-income community control of nonmaltreated children. These findings suggest that MTFC-P protected against this flattening of diurnal cortisol associated with neglect and chronic stress exposure (Fisher, Stoolmiller, Gunnar, & Burraston, 2007).

Further investigation of the potential mechanisms underlying this prevention effect revealed the critical influence of caregiver stress, an important correlate of caregiver responsiveness. In particular, foster parents in the MTFC-P condition experienced a sustained decrease in stress associated with child behavior, while the foster parents receiving services as usual experienced an increase during the year-long study period (Fisher & Stoolmiller, 2008). Critically, in families receiving services as usual, higher levels of foster parent stress associated with child problem behavior predicted more blunted cortisol levels in foster children, a relationship not present in the MTFC-P group. From a social buffering perspective, these findings suggest that supportive interventions, such as MTFC-P, may affect HPA axis functioning by bolstering the resources available for foster parents to help them deal with challenging behavior. MTFC-P has also been shown to mitigate the negative effects of a placement change, which can be conceptualized as a shift in the social buffer, on HPA axis functioning by focusing services on providing a consistent caregiving environment in foster care and in permanent placements (Fisher, Van Ryzin, & Gunnar, 2011).

Others have focused on foster care interventions in infancy, and as such, have taken an attachment-based approach. A focus on attachment is particularly logical early in life given the critical role of the child–caregiver relationship during this time. The Attachment and Biobehavioral Catch-up (ABC) intervention aims to increase sensitive and responsive parenting following child distress, an aim in line with improving the caregiver's capacity to serve as an effective buffer for the child. Evidence suggests that the ABC intervention is effective at normalizing HPA axis functioning in children with a history of Child Protective Services involvement because of neglect (Bernard, Dozier, Bick, & Gordon, 2015), and that these effects are maintained even 3 years following the intervention (Bernard, Hostinar, & Dozier, 2015).

Summary and future directions

Overall, these studies provide evidence that experiences of chronic adversity across childhood (i.e., neglect and caregiver instability/stress associated with placement in foster care) and diurnal cortisol patterning can be integrated into the current social buffering framework. These studies are a critical starting point for the field's exploration of how interventions designed to influence the quality of caregiving can alter HPA axis functioning. The interventions took a large-scale, multifaceted approach to provide initial evidence for which systems are malleable and which correlates of social support can in fact disrupt (and/or improve) future cortisol trajectories. These data suggest that although the specific mechanisms of interaction between daily parent–child relational quality and daily HPA axis

patterning are unknown, interventions designed to improve consistent, responsive, and supportive care demonstrate pliability in this system and are a means by which to normalize HPA axis functioning. And, these findings provide further evidence that the preschool years are a particularly plastic developmental period for social support's influence on HPA axis calibration.

Beyond main intervention effects, it is important to acknowledge individual variability in outcomes. For example, it is likely that the level of social buffering required to influence HPA axis patterning exists on a continuum that varies for different individuals. Our review suggests that the absence of a social buffer does not permanently exclude the system from appropriately responding, although it may render the system more vulnerable to downregulation when under stress. Moreover, minimizing parental stress specifically in response to child behaviors and not to overall stress and increasing the stability of the caregiver may be meaningful next steps to probe how the caregiver can buffer HPA axis patterning. However, the large-scale design of these interventions also renders us unable to draw causal inferences about specific mechanisms of change. Future interventions should seek to test these theories by targeting specific components of social buffering to probe the mechanisms of change involved in interventions.

One example of this targeted intervention approach is a promising new intervention developed in our lab called Filming Interactions to Nurture Development (FIND). FIND directly targets and aims to increase behaviors that enable caregivers to be more reliable buffers to children. FIND uses the concept of "serve and return" (Schindler, Fisher, & Shonkoff, in press) in the context of strength-based video coaching to demonstrate to caregivers the ways in which they are already effectively responding to their child in developmentally appropriate ways. The intervention includes several elements that are particularly relevant to social buffering, including showing the caregiver instances during which they provided external regulation when the child is distressed. Investigations of FIND's effectiveness and underlying mechanisms are underway.

It is essential to note the larger system in which the HPA axis exists and functions as well as methodologies that index chronic stress (i.e., hair cortisol). The HPA axis serves a critical regulatory role in the function and activation of several systems, including brain regions sensitive to levels of glucocorticoids (i.e., amygdala, hippocampus, and prefrontal cortex), immune health, oxidative stress, and the gut–brain axis (Bellavance & Rivest, 2014; Dinan & Cryan, 2012; Lupien et al., 2009). Promising evidence from animal studies and emerging human studies literature suggests that these associations may be multiple entry points into the system for intervention, and that a social buffering framework is a useful means to direct future intervention research and efforts. Future work should continue to seek greater clarity about the mechanisms that link acute and chronic stress and the relative role of social buffering to update and refine our working conceptual extension model.

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Disclosure statement

Dr. Fisher is the developer of the MTFC-P intervention described herein, and a co-owner of Treatment Foster Care Consultants, Inc., which provides consultation on the implementation of MTFC-P.

References

Badanes LS, Watamura SE, Hankin BL. Hypocortisolism as a potential marker of allostatic load in children: Associations with family risk and internalizing disorders. Development and Psychopathology. 2011; 23(3):881–896. [PubMed: 21756439]

Bellavance MA, Rivest S. The HPA–immune axis and the immunomodulatory actions of glucocorticoids in the brain. Frontiers in Immunology. 2014; 5

Bernard K, Dozier M, Bick J, Gordon MK. Intervening to enhance cortisol regulation among children at risk for neglect: Results of a randomized clinical trial. Development and Psychopathology. 2015; 27:829–841. [PubMed: 25156798]

Bernard K, Hostinar CE, Dozier M. Intervention effects on diurnal cortisol rhythms of Child Protective Services–referred infants in early childhood preschool follow-up results of a randomized clinical trial. JAMA Pediatrics. 2015; 169(2):112–119. [PubMed: 25436448]

Bruce J, Fisher PA, Pears KC, Levine S. Morning cortisol levels in preschool-aged foster children: Differential effects of maltreatment type. Developmental Psychobiology. 2009; 51(1):14–23. [PubMed: 18720365]

Bruce J, Gunnar MR, Pears KC, Fisher PA. Early adverse care, stress neurobiology, and prevention science: Lessons learned. Prevention Science. 2013; 14(3):247–256. [PubMed: 23420476]

Dinan TG, Cryan JF. Regulation of the stress response by the gut microbiota: Implications for psychoneuroendocrinology. Psychoneuroendocrinology. 2012; 37(9):1369–1378. [PubMed: 22483040]

Dozier M, Manni M, Gordon MK, Peloso E, Gunnar MR, Stovall-McClough KC, Levine S. Foster children's diurnal production of cortisol: An exploratory study. Child Maltreatment. 2006; 11(2): 189–197. [PubMed: 16595852]

Fisher PA, Ellis BH, Chamberlain P. Early intervention foster care: A model for preventing risk in young children who have been maltreated. Children's Services: Social Policy, Research, and Practice. 1999; 2(3):159–182.

Fisher PA, Stoolmiller M. Intervention effects on foster parent stress: Associations with child cortisol levels. Development and Psychopathology. 2008; 20(3):1003–1021. [PubMed: 18606041]

Fisher PA, Stoolmiller M, Gunnar MR, Burraston BO. Effects of a therapeutic intervention for foster preschoolers on diurnal cortisol activity. Psychoneuroendocrinology. 2007; 32(8–10):892–905. [PubMed: 17656028]

Fisher PA, Van Ryzin MJ, Gunnar MR. Mitigating HPA axis dysregulation associated with placement changes in foster care. Psychoneuroendocrinology. 2011; 36(4):531–539. [PubMed: 20888698]

Gunnar M, Quevedo K. The neurobiology of stress and development. Annual Review of Psychology. 2007; 58:145–173.

Gunnar MR, Vazquez DM. Low cortisol and a flattening of expected daytime rhythm: Potential indices of risk in human development. Development and Psychopathology. 2001; 13(3):515–538. [PubMed: 11523846]

Hostinar CE, Sullivan RM, Gunnar MR. Psychobiological mechanisms underlying the social buffering of the hypothalamic–pituitary–adrenocortical axis: A review of animal models and human studies across development. Psychological Bulletin. 2014; 140(1):256–282. [PubMed: 23607429]

Laurent HK, Gilliam KS, Bruce J, Fisher PA. HPA stability for children in foster care: Mental health implications and moderation by early intervention. Developmental Psychobiology. 2014; 56(6): 1406–1415. [PubMed: 24889670]

Laurent HK, Gilliam KS, Wright DB, Fisher PA. Child anxiety symptoms related to longitudinal cortisol trajectories and acute stress responses: Evidence of developmental stress sensitization. Journal of Abnormal Psychology. 2015; 124(1):68–79. [PubMed: 25688433]

Lupien SJ, McEwen BS, Gunnar MR, Heim C. Effects of stress throughout the lifespan on the brain, behaviour and cognition. Nature Reviews Neuroscience. 2009; 10(6):434–445. [PubMed: 19401723]

McLaughlin KA, Sheridan MA, Tibu F, Fox NA, Zeanah CH, Nelson CA. Causal effects of the early caregiving environment on development of stress response systems in children. Proceedings of the National Academy of Sciences. 2015; 112(18):5637–5642.

Schindler H, Fisher PA, Shonkoff J. From innovation to impact at scale: Lessons learned from a cluster of research-community partnerships. Child Development. (in press).

Strüber N, Strüber D, Roth G. Impact of early adversity on glucocorticoid regulation and later mental disorders. Neuroscience & Biobehavioral Reviews. 2014; 38:17–37. [PubMed: 24216122]

U.S. Department of Health and Human Services, Administration for Children and Families, Administration on Children, Youth and Families, Children's Bureau. Child maltreatment 2013. 2015. Retrieved from https://www.acf.hhs.gov/sites/default/files/cb/cm2013.pdf

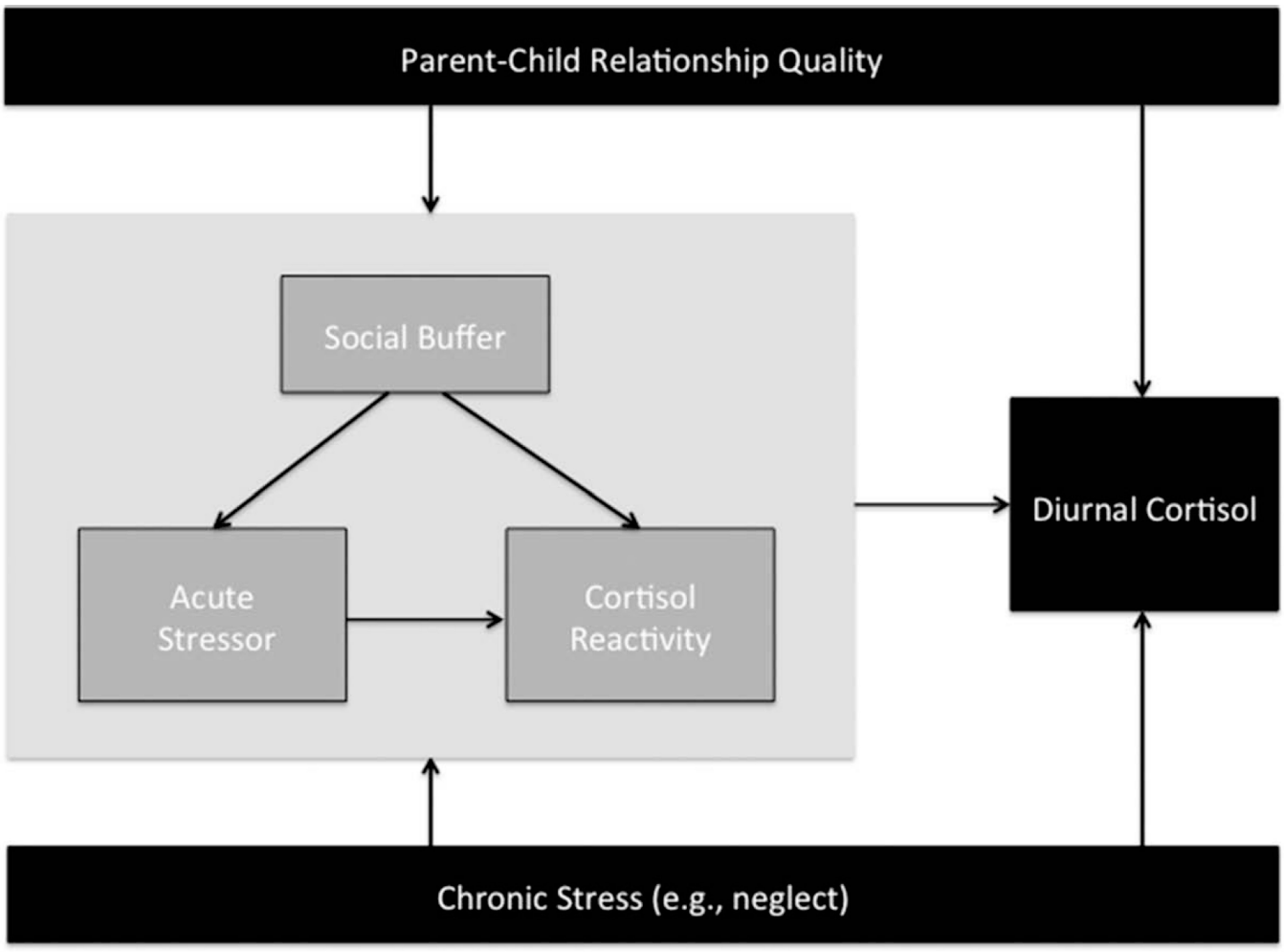

Figure 1. Working conceptual model of social buffer theory extension

An *acute stressor* evokes an increase in *cortisol reactivity*. An effective *social buffer* can moderate (i.e., dampen) this cortisol reactivity. However, the effectiveness of the social buffer during an acute stressor is dependent on the quality of the parent–child relationship over time. Parents who provide consistent, supportive care are more effective social buffers for their children than those who are not. Over time, these processes (depicted in gray) shape overall functioning of the HPA axis, as indicated by *diurnal cortisol*, and are moderated by *chronic stress*, which can take many forms including but not limited to neglect, maltreatment, and socioeconomic adversity. Additionally, we recognize other pathways that likely affect this model and warrant further investigation. The quality of the social buffer may directly impact diurnal cortisol, and the nature of the acute stressor (i.e., lack of food or shelter vs. physical abuse) likely interacts with the quality of the social buffer and social buffering capacity.