HHS Public Access

Author manuscript

Soc Neurosci. Author manuscript; available in PMC 2015 October 24.

Published in final edited form as:

Soc Neurosci. 2015 October ; 10(5): 479–488. doi:10.1080/17470919.2015.1070747.

The Social Buffering of the Hypothalamic-Pituitary-Adrenocortical Axis in Humans: Developmental and Experiential Determinants

Megan R. Gunnar and
Institute of Child Development, University of Minnesota

Camelia E. Hostinar
Institute for Policy Research, Northwestern University

Abstract

Social buffering, a subset of social support, is the process through which the availability of a conspecific reduces the activity of stress-mediating neurobiological systems. While its role in coping and resilience is significant, we know little about its developmental history in humans. This brief review presents an integrative developmental account of the social buffering of hypothalamic-pituitary-adrenocortical (HPA) stress reactivity in humans, from infancy to adulthood. During infancy, parents are powerful stress-regulators for children, but child temperament also plays a role and interacts with parenting quality to predict the magnitude of stress responses to fear or pain stimuli. Recent work indicates that parental support remains a potent stress buffer into late childhood, but that it loses its effectiveness as a buffer of the HPA axis by adolescence. Puberty may be the switch that alters the potency of parental buffering. In Beginning in middle childhood, friends may serve as stress buffers, particularly when other peers are the source of stress. By adulthood romantic partners assume this protective role, though studies often reveal sex differences that are currently not well understood. Translational research across species will be critical for developing a mechanistic understanding of social buffering and the processes involved in developmental changes noted in this review.

Keywords

social buffering; HPA axis; cortisol; development

Supportive social relationships contribute to health and well-being throughout life (Cohen, 2004). Social partners not only reduce stress by providing material help, but their presence can provide a psychological buffer against stress (Taylor, 2011). The stress buffering effect of social partners has been documented in many species (Hennessy, Kaiser, & Sachser, 2009). A critical stress mediating system, the hypothalamic-pituitary-adrenocortical (HPA) axis (Lupien, McEwen, Gunnar, & Heim, 2009), has been the focus of much of the work on

Corresponding Author: Megan R. Gunnar, University of Minnesota - Institute of Child Development, 51 East River Road, Minneapolis, Minnesota 55455 United States, gunnar@umn.edu.

Disclosure: Neither Megan Gunnar nor Camelia Hostinar have any relevant financial or nonfinancial relationships to disclose.

social buffering (Hostinar, Sullivan, & Gunnar, 2014). The HPA axis is of special note when studying social buffering from a developmental perspective. This is because its hormonal product, cortisol in humans, is a gene transcription factor that impacts numerous aspects of neurobehavioral development (Gunnar & Vazquez, 2006). The present paper reviews the developmental research on the role of the presence and availability of social partners in buffering the HPA axis in human development. As will be discussed, this work is closely aligned with research on parent-child attachment relationships. However, while attachment security is important to buffering the HPA axis in children, we do not fully understand the mechanisms underlying the power of the parent's presence to block cortisol elevations, nor do we know the mechanisms explaining developmental changes in social buffering during development.

Social Stress Buffering During Infancy and Early Childhood

Over two decades ago the first author conducted the first study of social buffering of the HPA axis during human development robot (Nachmias, Gunnar, Mangelsdorf, Parritz, & Buss, 1996). This study followed puzzling findings showing that while newborns exhibited a robust adrenocortical response to stressors as mild as being undressed, weighed and measured, by the end of the first year of life even a stressor like being stuck twice with a needle, once in each thigh, for childhood inoculations ceased to produce elevations in cortisol in most babies, even though all babies cried vigorously when receiving these injections (Gunnar, 1992; Gunnar, Brodersen, Krueger, & Rigatuso, 1996; Jacobson, Bihun & Chiodo, 1999) (see figure 1). What was puzzling was whether this phenomenon should be viewed as the emergence of a stress hypo-responsive period, similar to that seen in very young rodents (Suchecki, Rosenfeld, & Levine, 1993), or as the emergence of the parent's presence as a powerful buffer of the HPA axis, similar to what had been observed in non-human primates (Gunnar, Gonzales, Goodlin, & Levine, 1981). This was a difficult puzzle to solve because to test whether the parent's presence buffered the axis when children were exposed to a stressor, children needed to be exposed to a stressor with and without the parent present. However, by the end of the first year of life, infants show a robust stress response to separation (Spangler & Grossmann, 1993), so it would be difficult to differentiate parental buffering from separation stress.

To get around this problem, rather than asking about presence and absence of the parental stress buffer, instead we asked about whether the security of the parent-infant relationship influenced the potency of the buffer. We found that a secure attachment relationship with the parent who was present during stressors like exposure to strange objects and events –e.g., live clown, loud mechanical toys (Nachmias et al., 1996) or receiving childhood inoculations (Gunnar, Brodersen, Nachmias, Buss, & Rigatuso, 1996) completely blocked increases in cortisol, while elevations were still noted for children who were insecurely attached to the parent who was with them. Notably, in these studies it was the temperamentally fearful children who benefited the most from secure relationships as these were the children who otherwise showed the most marked elevations in cortisol (see Figure 2 a and b).

This was good evidence that infants and toddlers in secure relationships were experiencing a robust buffering of the HPA axis when their attachment figure was present; it was not clear whether the parent had to actually be present for the effect to be observed. According to attachment theory, children form internal working models of their attachment relationships and over time these models influence the child's ability to regulate their emotional state even when the parent is not immediately available (Cassidy, 1994). Thus it was conceivable that securely attached children would be buffered against stress even when the attachment figure was not available, and experience greater reductions in cortisol production than would insecurely attached children. Studying children's adaptation to child care allowed an opportunity to address this question. Certainly when children first start child care we might expect them to show a separation response, but over time they should adapt. Would the securely attached children show that they were capable of making a more rapid adaptation? Ahnert and colleagues (Ahnert, Gunnar, Lamb, & Barthel, 2004) studied 12- to 24-month-olds in Berlin, Germany who were starting center-based child care for the first time. Cortisol was assessed during four periods: (1) at home before beginning child care, (2) on several days when the mothers came with the children to child care in order to help them to adjust, (3) on the first, fifth and ninth day after the mothers left their children at the center, and (4) at 5 months. The results were quite clear. Although the children in secure relationships had lower cortisol when their mothers were present, their cortisol levels rose to those of children from insecure attachment relationships as soon as the mothers were no longer there. Securely attached children did not adapt or return to baseline levels faster than insecurely attached children. Thus, for young children it appears that the parent needs to be present for buffering to work.

However, how does this buffering work? What are the psychological and physiological mechanisms through which social buffering operates in infants and very young children? In most of our adult models of social buffering we assume that what is being buffered are neural systems involved in threat processing. Indeed, there is good evidence that among adults, attachment figures operate as safety signals, activating brain regions that block or reduce the perception of pain and threat (Eisenberger et al., 2011). Certainly, there is good evidence that young children interpret events differently in the presence of their attachment figures. A stranger playing peek-a-boo with a baby may elicit crying, while a parent doing so elicits smiles and laughter (Sroufe, Waters, & Matas, 1974). Likewise attachment figures are described as providing a secure base from which to explore the world (Bowlby, 1969). However, it is striking that during the infant and toddler period, parental social buffering has been demonstrated precisely under circumstances in which the parent's presence is not sufficient to prevent the child from displaying high degrees of behavioral fear and distress (Gunnar, Brodersen, Nachmias, et al., 1996; Nachmias et al., 1996). Thus what is observed is a marked dissociation between behaviors reflecting fear and distress and activity of the HPA axis. The parent is buffering the HPA axis, even though the child remains frightened or distressed by the stressor.

These findings raise the possibility that in infants and toddlers the attachment figure's presence is operating in the hypothalamus to prevent the hormonal cascade from beginning even if higher brain regions are sending signals that would otherwise trigger a response. There is evidence of this in rat pups for whom it has been shown that the presence of the

mother blocks neural activity in the form of NE release in the hypothalamus thus preventing the cascade of events leading to elevations in the corticosterone, the rodent version of cortisol (Moriceau & Sullivan, 2006). In a similar vein, oxytocin stimulated by the attachment figure's presence is known to operate at the hypothalamic and pituitary level to reduce the HPA response (Gibbs, 1986). Thus, early in development attachment figures as social buffers may be capable of operating fairly directly on the HPA axis to reduce or prevent cortisol elevations to threatening stimuli (Hostinar et al., 2014). Whether this is true and whether these more direct impacts on the axis also operate when social buffering alters corticolimbic processes involved in threat perception and emotional reactivity requires further exploration.

Parental Social Buffering in Middle Childhood

Contact with the attachment figure continues to be a powerful stress buffer into middle childhood. However, by middle childhood and possibly earlier, the parent does not have to be physically present to reduce the HPA axis response. A recent study (Seltzer, Ziegler, & Pollak, 2010) randomly assigned 7- to 12-year-old girls to recover from the Trier Social Stress Test either (a) with their mothers, (b) with an experimenter but the girls could call and talk with their mothers on the phone, or (c) with only the experimenter. They found that oxytocin production was elevated both by being with the mother and talking to her on the phone. Cortisol returned to baseline fastest when the mother was fully present, but talking to her on the phone also lowered cortisol compared to the condition in which the child had no access to the mother during the recovery period. In a subsequent study, they showed that typing and receiving instant messages with the mother via a computer-based program did not have any buffering effect, with both cortisol and urinary oxytocin levels in this condition being comparable to those of children who had no contact with their mother afterwards (Seltzer, Prososki, Ziegler, & Pollak, 2012).

This suggests that the visual, verbal and likely physical cues from the parent are more powerful safety signals than a simple informational exchanges with the parent.

In studying social buffering among adults, Kirschbaum and colleagues (Kirschbaum, Klauer, Filipp, & Hellhammer, 1995) also used the Trier Social Stress Test but rather than having the social support provided during recovery, they had the social partner present during the period of time when the participant prepares to enter the room with the judges and give their speech. The support person then would leave and the participant would perform the task. Using this paradigm, we examined whether having the attachment figure present compared to preparing with an adult female stranger would buffer the HPA axis to the Trier Social Stress Test among 9- and 10-year olds (Hostinar, Johnson, & Gunnar, 2015). All of the children in both groups came to the assessment with their parent. The parent was with them during the period of consent and assent when the children learned about the task and during the recovery period after the task. Thus, the only difference between conditions was the short period of speech preparation when one group had the parent present and the other did not. Even so, we obtained a significant increase in cortisol for the children who prepared with the female stranger and no elevation for children who prepared with their attachment figure (see figure 3, panel A). At the end of the session we used the Lang Self-Assessment

Manikin to help the children rate how stressed they felt during each segment of the assessment. All children reported an increase in stress through the verbal math segment of the assessment and then a calming down once the speech/math performance was over. Having the parent present during the preparation did not reduce their self-reported stress. Of course, the parent was not present while the children were giving their speech and doing the math. Nonetheless, her presence and help during the preparation period blocked activation of the axis.

Adolescence and Parental Buffering

Adolescence is a time when children begin to shift their reliance on parents as their sources of security towards friends and later romantic partners (Allen & Miga, 2010; Collins, Welsh & Furman, 2009). Adolescents begin to spend more and more time with their peers and their peer networks become an increasingly important focus of their lives (Brown & Larson, 2009). In a very real sense, in adolescence youth begin to leave home emotionally, if not yet physically. We know relatively little about social stress buffering during adolescence, although adolescent perceptions of social support from family and friends is frequently measured and correlate positively with social and academic outcomes (Smetana, Campione-Bar, & Metzger, 2006). As an initial study, we asked whether parents were still effective stress buffers of the HPA axis for adolescents. In the same study in which we examined parental stress buffering among 9- and 10-year-old children we performed the same procedures for 15- and 16-year-old adolescents (Hostinar et al., 2015). The results were clear and are shown in figure 3, panel B. Basal cortisol levels were higher in adolescence, as expected (see for review, Gunnar & Vazquez, 2006). In addition, both adolescents who prepared for the speech with their attachment figure and those preparing with the female stranger exhibited a significant and similar cortisol response to the stressor task. In observations of parent-child interaction during the preparation period, parents were equally supportive of children and adolescents. Adolescents reported that they had more conflicts in general with parents than did the children, but variation in conflict reports did not predict cortisol increases. Thus, it appeared that parents had lost their potency as buffers of the HPA axis response to this type of stressor in adolescence.

When between 10 and 15 years did do parents lose their HPA axis, stress-buffering potency? Puberty is expected to open another period of neurobiological plasticity (Dahl & Gunnar, 2009). With increasing pubertal stage, there is an increase in the production of steroid hormones, including cortisol (Gunnar & Vazquez, 2006). We are in the process of examining whether puberty might be the biological switch that turns off the parent's availability as a potent HPA axis stress buffer (Doom, Hostinar, VanZomeren-Dohn, & Gunnar, 2015). To address this question we needed to be able to differentiate pubertal stage from age. These variables are generally too highly correlated to do so. However, in early adolescence youth start and traverse pubertal development at different times and rates. By focusing on early adolescence one can pre-select participants so as to uncouple age and pubertal stage. This is what we did. We chose a limited age range of youth from 11 years through 14 years. When we contacted the families by phone, we conducted a brief pubertal assessment and then recruited them based on a crossing of sex, age and pubertal stage. Once they came in for the laboratory portion of the assessment we reassessed puberty by asking

the youth to complete the Petersen Pubertal Development Scale (Petersen, Crockett, Richards, & Boxer, 1988). We assigned roughly half of the younger and older and pre/early and mid/late pubertal stage youth to prepare for the speech with their attachment figure and half with a female stranger. To focus on activation of the HPA axis, in this study we used landmark registration to differentiate reactivity from recovery of the axis. Our preliminary results indicate that the effects of pubertal stage were specific to the activation or reactivity phase of the response. For 11- to 14-year-old youth who were in the pre- or early stages of puberty the parent's presence buffered reactivity, while for the same age youth who were mid- to late-pubertal the parent's presence had no effect.

Combined, both of these studies (Doom et al., 2015; Hostinar et al., 2015) suggest that the transition from childhood to adolescence involves a loss of a critical stress buffer. The parent-child attachment relationship no longer operates to shield the developing brain from the effects of elevated cortisol. This does not mean that adolescents are no longer attached to their parents. Indeed, there is good evidence that adolescents remain emotionally attached and that the quality of their attachment relationships continues to be a major predictor of their self-reported well-being (Greenberg, Siegel, & Leitch, 1983). Nor does it mean that loss of the parental HPA stress buffer is a threat to development. During the latter part of gestation, cortisol levels rise and help mature organ systems in preparation for a transition from intra- to extrauterine life (Moisiadis & Matthews, 2014). Recently, evidence has accumulated that brief exposure to stressors which produce marked elevations in glucocorticoids are good for the brain, fostering the development of neural systems that promote resilience (Lee, Buckmaster, Yi, Schatzberg, & Lyons, 2014). Thus, it may be that for most children the loss of a potent parental buffer of the HPA axis is simply a part of normal development and enhances the child's capacity to develop mature patterns of stress regulation. However, it is noteworthy that the timing of this loss corresponds to the period in development when a rise in affective disorders is observed (Maughan, Collishaw, & Stringaris, 2013). While most of the attention in explaining this rise with puberty has focused on the role of sex steroids, particularly estrogens because girls are more vulnerable to depression than boys, after years of study the results are still equivocal (Balzer, Duke, Hawke, & Steinbeck, 2015). The present findings raise the possibility that the loss of the parental social buffer of the HPA axis may also play a role, particularly as dysregulation of the HPA axis is associated with the emergence of depression in adolescents (Adam et al., 2010; Halligan, Herbert, Goodyer, & Murray, 2007).

Friends and Social Buffering in Childhood and Adolescence

Under extraordinary circumstance, children can form primary attachment relationships with other children, such as those that Anna Freud documented for the children who lost parents during the Second World War (Freud & Burlingham, 1943). Similarly, non-human primates reared only with peers will also use peers as primary sources of security (Suomi, 1991). However, these relationships do not appear to function as effectively as parent-offspring relationships as this type of rearing is associated with significant problems in managing stresses and challenges later in life (Suomi, 1991). Likewise, in non-human primates there is evidence that attachment to peers in peer-reared monkeys is not as strong as that observed among mother-reared infants (Meyer, Novak, Bowman, & Harlow, 1975).

Nevertheless there is evidence that children can use peers as stress buffers under certain circumstances. This is particularly the case when other peers are the source of stress. Known as the friendship protection hypothesis, children and youth with at least one supportive friend are less negatively impacted by being bullied than children without any friends (Kendrick, Jutengren, & Stattin, 2012). At least by grade 4, friends appear to be able to buffer the HPA axis to experiences of peer rejection (Peters, Riksen-Walraven, Cillessen, & de Weerth, 2011) and other negative experiences at school (Adams, Santo, & Bukowski, 2011). Thus it is possible that as the potency of parental buffering of the HPA axis wanes, the presence and availability of friends may be able to provide youth with an effective buffer of the HPA axis. However, the quality of the friendship is likely to be critical. For example, in a study of recovery from the Trier Social Stress Test, youth were paired with their best friend and told to discuss their performance with this friend (Calhoun et al., 2014). Youth who described their friendship as more supportive and friends who were more positively responsive to their friend as she or he talked with them about their performance returned to baseline cortisol levels faster. As in other work, friends who encouraged youth to ruminate on negative aspects of their performance prolonged the stress response (Stewart, Mazurka, Bond, Wynne-Edwards, & Harkness, 2013). Thus the availability of friends, their supportiveness at times of adversity and the coping strategies they encourage likely will influence their effectiveness as buffers of HPA axis stress responses.

Social Stress Buffering Among Adults and Gender Differences

Attachment relationships with parents in childhood are associated with the quality of relationships individuals form with romantic partners in adulthood (Holland & Roisman, 2010). This and the work on friends as social buffers would suggest that romantic partners would certainly be capable of buffering the HPA axis, especially if the relationship was secure. However, the evidence so far is that this may well be true for men, but the evidence for women is equivocal. Among adult men, support by a romantic partner or best friend markedly reduces the cortisol response to the Trier Social Stress test when the partner was present and supportive during speech preparation (Heinrichs, Baumgartner, Kirschbaum, & Ehlert, 2003; Kirschbaum et al., 1995). Importantly, social buffering by a best friend was as effective as nasal oxytocin in buffering the HPA axis among men (Heinrichs et al., 2003). However, in this work researchers have found relatively little evidence that for women the HPA axis is buffered by the presence and verbal support of an opposite sex romantic partner (Kirschbaum et al., 1995). Only when that partner provided physical touch (neck and shoulder massage) was the presence of a woman's opposite-sex partner a buffer for the HPA axis (Ditzen et al., 2007). Notably, this was not because physical touch elevated oxytocin while verbal support did not. Both verbal and physical touch by the partner during speech preparation elevated oxytocin equally, while only touch reduced the HPA response. Findings such as these raise questions about how critical oxytocin is in mediating social buffering of the HPA axis for women.

In contrast to the work on the HPA axis, many of the studies on cardiovascular measures of stress and social buffering have been done using women as the participants. Here there is evidence that the presence of another woman, whether she is a friend or a stranger, can reduce women's cardiovascular reactions to social evaluative stressors (Fontana, Diegnan,

Villeneuve, & Lepore, 1999). However, in these cases it is critical that the woman providing social support is unable to hear the performance of the target woman as this removes any evaluative component from the social support (Kors, Linden, & Gerin, 1997). When the threat of social evaluation is allowed by permitting the person present to be privy to the quality of the participant's performance, not only can the person's presence not reduce autonomic reactivity, but sometimes it increases (Gramer & Reitbauer, 2010). Autonomic activity, which the cardiac measures reflect, is sensitive to how much effort the person puts into his or her performance. Thus, it is likely that rather than social facilitation effects influencing effort and are masking any social buffering effect of a peer or romantic partner's presence when autonomic measures are used. Unlike the autonomic nervous system, the HPA axis has been shown to more closely track the participant's sense of threat (Ursin, Baade, & Levine, 1978), making HPA axis measures more likely to reflect the social buffering of stress than autonomic measures when the stressor context involves performance measures that can be influenced by effort.

Nonetheless, the presence of gender differences in the effectiveness of social buffering to reduce cardiovascular responses in men (effective) and women (less effective), does seem to mirror gender differences in social buffering of the HPA axis among adults. Furthermore, this gender difference extends to the effects of oxytocin as a proxy for social support. Specifically, nasal oxytocin reduced cardiovascular reactivity to a social stressor for men, but increased it for women (Kubzansky, Mendes, Appleton, Block, & Adler, 2012). We do not know at what age these sex differences in the stress buffering effects of friends and romantic partners emerge in development. Thus far sex differences in HPA axis stress responses to social stressors, such as peer rejection and public speaking, have not been reported. For example, although men respond more strongly to instrumental achievement stressors like the Trier Social Stress Test and women respond more strongly to a rejection stressor (Stroud, Salovey, & Epel, 2002), this is not observed in childhood and adolescence (Stroud et al., 2009). When we have observed sex differences in response to the Trier Social Stress Test they could be explained by differences in pubertal maturation (Gunnar, Wewerka, Frenn, Long, & Griggs, 2009). In addition, in none of our studies of parental buffering of the HPA axis have we noted significant sex differences; however, this may have been a power problem as the cell sizes were small when the possibility of interactions with sex were also included in our analyses (Doom et al., 2015; Hostinar et al., 2015; Nachmias et al., 1996). In terms of understanding both the neurobiological and psychological processes involved in the social buffering and its role in development, a better understanding of gendered processes will be necessary.

Mechanisms: The Future Direction

As noted earlier, we have yet to fully understand the mechanisms underlying the social buffering effects described in this paper. Nor is it clear precisely how these mechanisms change with development. There are a number of putative mediators of social buffering at different levels of the neuraxis from those operating at the level of the hypothalamus to those at higher levels influencing perceptions of threat and emotion regulation (Hostinar et al., 2014). Understanding these mechanisms, their development, and their capacity to be shaped by experiences as we grow up is an important frontier in developmental social

neuroscience. Addressing these questions will require a synthesis of research in nonhuman animals and humans as many of the mechanistic questions will be too invasive to address in typically developing children and adolescents. However, given the importance of stress and its regulation to healthy development (Shonkoff, Boyce, & McEwen, 2009), this work holds the promise of helping to foster understanding of the ontogeny of emotional disorders in children and adolescents and provide insights for the design of more effective interventions.

Acknowledgments

This work was supported by the Canadian Institute for Advanced Research's Program on Child and Brain Development and NIMH Center grant MH078105 to MRG and NICHD award number F32HD078048 to CEH. The content is solely the responsibility of the authors and does not necessarily represent the official views of the National Institutes of Health.

References

Adam EK, Doane LD, Zinbarg RE, Mineka S, Craske MG, Griffith JW. Prospective prediction of major depressive disorder from cortisol awakening responses in adolescence. Psychoneuoendocrinology. 2010; 35:921–931.

Adams RE, Santo JB, Bukowski WM. The presence of a best friend buffers the effects of negative experiences. Developmental Psychology. 2011; 47:1786–1791. [PubMed: 21895364]

Ahnert L, Gunnar MR, Lamb ME, Barthel M. Transition to child care: Associations with infant-mother attachment, infant negative emotion, and cortisol elevations. Child Development. 2004; 75(3):639–650. [PubMed: 15144478]

Allen JP, Miga EM. Attachment in adolescence: A move to the level of emotion regulation. Journal of Social and Personal Relationships. 2010; 1:181–190. [PubMed: 20431705]

Balzer BW, Duke SA, Hawke CI, Steinbeck KS. The effects of estradiol on mood and behavior in human female adolescents: a systematic review. European Journal of Pediatrics. 2015; 174:289–298. [PubMed: 25567794]

Bowlby, J. Attachment and Loss: Attachment. Vol. 1. New York: Basic Books; 1969.

Brown, BB.; Larson, J. Peer relations in adolescence. In: Lerner, RM.; Steinberg, L., editors. Handbook of Adolescent Psychology: Contextual Influences on Adolescent Development. Vol. 2. Hoboken, New Jersey: Wiley and Sons; 2009. p. 74-103.

Calhoun CD, Helms SW, Heilbron N, Rudolph KD, Hastings PD, Prinstein MJ. Relational victimization, friendship, and adolescents' hypothalamic-pituitary-adrenal axis responses to an in vivo social stressor. Development and Psychopathology. 2014; 26:605–618. [PubMed: 25047287]

Cassidy J. Emotion regulation within attachment relationships. Monographs of the Society for Research in Child Development. 1994; 59(2–3):228–283. [PubMed: 7984163]

Cohen S. Social relationships and health. American Psychologist. 2004; 59:676–684. [PubMed: 15554821]

Collins WA, Welsh DP, Furman W. Adolescent romantic relationships. Annual Review of Psychology. 2009; 60:631–652.

Dahl RE, Gunnar MR. Introduction to Special Section on Heightened stress responsiveness and emotional reactivity during pubertal maturation: Implications for Psychopathology. Development & Psychopathology. 2009; 21:1–6. [PubMed: 19144219]

Ditzen B, Neumann ID, Bodenmann G, von Dawans B, Turner RA, Ehlert U, Heinrichs M. Effects of different kinds of couple interaction on cortisol and heart rate responses to stress in women. Psychoneuroendocrinology. 2007; 32:565–574. [PubMed: 17499441]

Doom JR, Hostinar CE, VanZomeren-Dohn, Gunnar MR. The roles of puberty and age in explaining the diminished effectiveness of parental buffering of HPA reactivity and recovery in adolescence. Under review. 2015

Eisenberger NI, Mastera SL, Inagakia TK, Taylor SE, Shirinyan D, Lieberman MD, Naliboff BD. Attachment figures activate a safety signal-related neural region and reduce pain experience. Proceedings of the National Academy of Sciences. 2011; 108:11721–11726.

Fontana AM, Diegnan T, Villeneuve A, Lepore SJ. Nonevaluative social support reduces cardiovascular reactivity in young women during acutely stressful performance situations. Journal of Behavioral Medicine. 1999; 22:75–91. [PubMed: 10196730]

Freud, A.; Burlingham, DT. War and children. Oxford, England: Medical War Books; 1943.

Gibbs DM. Stress-specific modulation of ACTH secretion by oxytocin. Neuroendocrinology. 1986; 42:456–458. [PubMed: 3010155]

Gramer M, Reitbauer C. The influence of social support on cardiovascular responses during stressor anticipation and active coping. Biological Psychology. 2010; 85:268–274. [PubMed: 20673842]

Greenberg MT, Siegel JM, Leitch CJ. The nature and importance of attachment relationships to parents and peers during adolescence. Journal of Youth and Adolescence. 1983; 12:373–386. [PubMed: 24306358]

Gunnar MR. Reactivity of the hypothalamic-pituitary-adrenocortical system to stressors in normal infants and children. Pediatrics. 1992; 90(3):491–497. [PubMed: 1513614]

Gunnar MR, Brodersen L, Krueger K, Rigatuso J. Dampening of adrenocortical responses during infancy: Normative changes and individual differences. Child Development. 1996; 67(3):877–889. [PubMed: 8706532]

Gunnar MR, Brodersen L, Nachmias M, Buss KA, Rigatuso J. Stress reactivity and attachment security. Developmental Psychobiology. 1996; 29(3):191–204. [PubMed: 8666128]

Gunnar MR, Gonzales C, Goodlin C, Levine S. Behavioral and pituitary-adrenal responses during a prolonged separation period in infant Rhesus macaques. Psychoneuroendocrinology. 1981; 6(1): 65–75. [PubMed: 7195597]

Gunnar, MR.; Vazquez, D. Stress neurobiology and developmental psychopathology. In: Cicchetti, D.; Cohen, D., editors. Developmental Psychopathology:Developmental Neuroscience. 2nd ed.. Vol. 2. New York: Wiley; 2006. p. 533-577.

Gunnar MR, Wewerka S, Frenn K, Long JD, Griggs C. Developmental changes in HPA activity over the transition to adolescence: Normative changes and associations with puberty. Development & Psychopathology. 2009; 21:69–85. [PubMed: 19144223]

Halligan SL, Herbert J, Goodyer I, Murray L. Disturbances in morning cortisol excretion in association with maternal postnatal depression predict subsequent depressive symptomatology in adolescents. Biological Psychiatry. 2007; 62:40–46. [PubMed: 17188253]

Heinrichs M, Baumgartner T, Kirschbaum C, Ehlert U. Social support and oxytocin interact to suppress cortisol and subjective responses to psychosocial stress. Biological Psychiatry. 2003; 54:1389–1398. [PubMed: 14675803]

Hennessy MB, Kaiser S, Sachser N. Social buffering of the stress response: diversity, mechanisms, and functions. Frontiers in Neuroendocrinology. 2009; 30(4):470–482. [PubMed: 19545584]

Holland AS, Roisman GI. Adult attachment security and young adults' dating relationships over time: self-reported, observational, and physiological evidence. Developmental Psychology. 2010; 46:552–557. [PubMed: 20210513]

Hostinar CE, Johnson AE, Gunnar MR. Parent support is less effective in buffering cortisol stress reactivity for adolescents compared to children. Developmental Science. 2015; 18:281–297. [PubMed: 24942038]

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]

Jacobson SW, Bihun JT, Chiodo LM. Effects of prenatal alcohol and cocaine exposure on infant cortisol levels. Development and Psychopathology. 1999; 11:195–208. [PubMed: 16506530]

Kendrick K, Jutengren G, Stattin H. The protective role of supportive friends against bullying perpetration and victimization. journal of Adolescence. 2012; 35:1069–1080. [PubMed: 22464910]

Kirschbaum C, Klauer T, Filipp SH, Hellhammer DH. Sex-specific effects of social support on cortisol and subjective responses to acute psychological stress. Psychosomatic Medicine. 1995; 57:23–31. [PubMed: 7732155]

Kors DJ, Linden W, Gerin W. Evaluation interferes with social support: Effects on cardiovascular stress reactivity in women. Journal of Social and Clinical Psychology. 1997; 16:1–23.

Kubzansky LD, Mendes WB, Appleton AA, Block J, Adler GK. A heartfelt response: Oxytocin effects on response to social stress in men and women. Biological Psychology. 2012; 90:1–9. [PubMed: 22387929]

Lee AG, Buckmaster CL, Yi E, Schatzberg AF, Lyons DM. Coping and glucocorticoid receptor regulation by stress inoculation. Psychoneuroendocrinology. 2014; 49:272–279. [PubMed: 25127085]

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

Maughan B, Collishaw S, Stringaris A. Depression in childhood and adolescence. Journal of the Canadian Academy of Child and Adolescent Psychiatry. 2013; 22:35–40. [PubMed: 23390431]

Meyer JS, Novak MA, Bowman RE, Harlow HF. Behavioral and hormonal effects of attachment object separation in surrogate-peer-reared and mother-reared infant rhesus monkeys. Developmental Psychobiology. 1975; 8:425–435. [PubMed: 817950]

Moisiadis VG, Matthews SG. Glucocorticoids and fetal programming part 2: Mechanisms. Nature Reviews Endocrinology. 2014; 10:403–411.

Moriceau S, Sullivan RM. Maternal presence serves as a switch between learning fear and attraction in infancy. Nature Neuroscience. 2006; 9:1004–1006. [PubMed: 16829957]

Nachmias M, Gunnar MR, Mangelsdorf S, Parritz R, Buss KA. Behavioral inhibition and stress reactivity: Moderating role of attachment security. Child Development. 1996; 67(2):508–522. [PubMed: 8625725]

Peters E, Riksen-Walraven JM, Cillessen AH, de Weerth C. Peer rejection and HPA activity in middle childhood: friendship makes a difference. Child Development. 2011; 82:1906–1920. [PubMed: 22026414]

Petersen A, Crockett L, Richards M, Boxer A. A self-report measure of pubertal status: Reliability, validity, and initial norms. Journal of Youth and Adolescence. 1988; 17:117–133. [PubMed: 24277579]

Seltzer LJ, Prososki AR, Ziegler TE, Pollak SD. Instant messages vs. speech: Hormones and why we still need to hear each other. Evolution and Human Behavior. 2012; 33:42–45. [PubMed: 22337755]

Seltzer LJ, Ziegler TE, Pollak SD. Social vocalizations can release oxytocin in humans. Proceedings of the Royal Society of Biological Sciences. 2010; 277:2661–2666.

Shonkoff J, Boyce WT, McEwen BS. Neuroscience, molecular biology, and the childhood roots of health disparities: building a new framework for health promotion and disease prevention. JAMA. 2009; 301:2252–2259. [PubMed: 19491187]

Smetana JG, Campione-Barr N, Metzger A. Adolescent development in interpersonal and societal contexts. Annual Review of Psychology. 2006; 57:255–284.

Spangler G, Grossmann KE. Biobehavioral organization in securely and insecurely attached infants. Child Development. 1993; 64:1439–1450. [PubMed: 8222882]

Sroufe, LA.; Waters, E.; Matas, L. Contextual determinants of infant affective response. In: Lewis, M.; Rosenblum, L., editors. The origins of fear. New York: Wiley; 1974.

Stewart JG, Mazurka R, Bond L, Wynne-Edwards KE, Harkness KL. Rumination and impaired cortisol recovery following a social stressor in adolescent depression. Journal of Abnormal Child Psychology. 2013; 41:1015–1026. [PubMed: 23553496]

Stroud L, Foster E, Handwerger K, Papandonatos GD, Granger D, Kivlighan KT, Niaura R. Stress response and the adolescent transition: performance versus peer rejection stress. Development & Psychopathology. 2009; 21:47–68. [PubMed: 19144222]

Stroud L, Salovey P, Epel ES. Sex differences in stress responses: social rejection versus achievement stress. Biological Psychiatry. 2002; 52:318–327. [PubMed: 12208639]

Suchecki D, Rosenfeld P, Levine S. Maternal regulation of the hypothalamic-pituitary-adrenal axis in the rat: The roles of feeding and stroking. Developmental Brain Research. 1993; 75(2):185–192. [PubMed: 8261610]

Suomi, SJ. Early stress and adult emotional reactivity in rhesus monkeys; Paper presented at the The childhood environment and adult disease: CIBA Foundation symposium; 1991.

Taylor, SE. Social support: A review. In: Friedman, HS., editor. Oxford Handbook of Health Psychology. New York, New York: Oxford University Press; 2011. p. 189-214.

Ursin, H.; Baade, E.; Levine, S. Psychobiology of stress: A study of moping Men. New York: Academic Press; 1978.

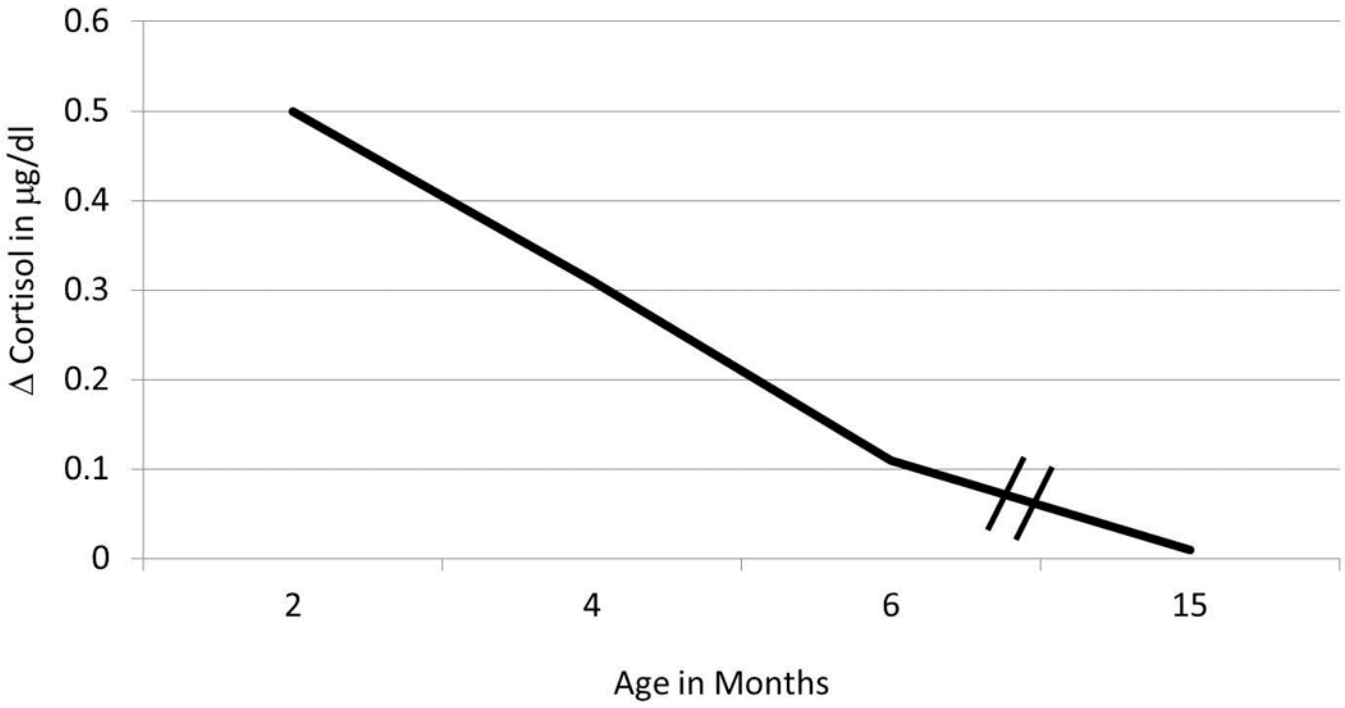

Figure 1.

Decrease in cortisol responses in µg/dl to well-baby examinations and inoculations across the first two years of life. Data adapted from Gunnar, Brodersen, Krueger, and Rigatuso (1996).

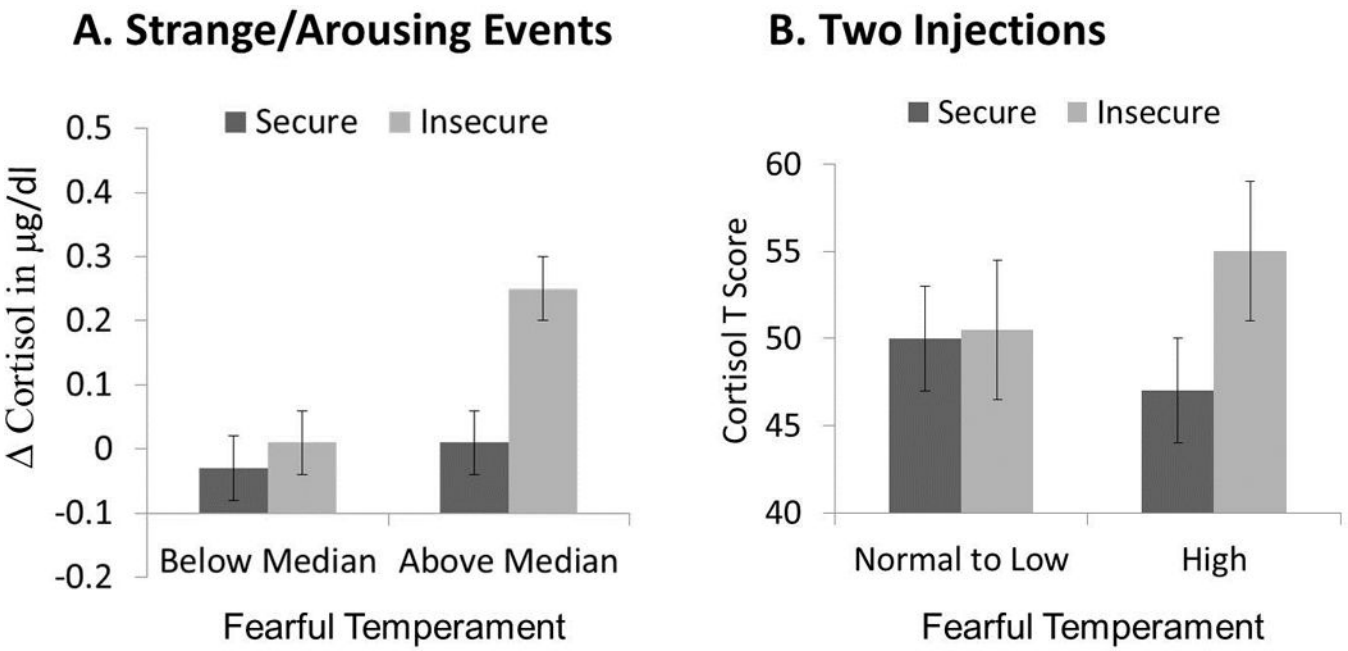

Figure 2.

Panel A: Cortisol increases in µg/dl among 18-month-olds exposed to strange and potentially scary objects and events. Fearfulness was measured as the children's behavioral responses to the events and divided at the median. Attachment security was indexed using the Strange Situation assessment conducted on a different day. Error bars reflect standard error of the mean. Data are adapted from Nachmias et al. (1996).

Panel B: Cortisol responses in responses to well-child visits with inoculations at 15-months of age. Cortisol measures are T scores calculated on standardized residuals after regressing post-test levels on pre-test levels to control for the Law of Initial Values. Attachment security was assessed using the Strange Situation conducted at 18-months. Fearful temperament was assessed through parent report. Bars reflect standard error of the mean. Data adapted from Gunnar, Brodersen, Nachmias et al. (1996).

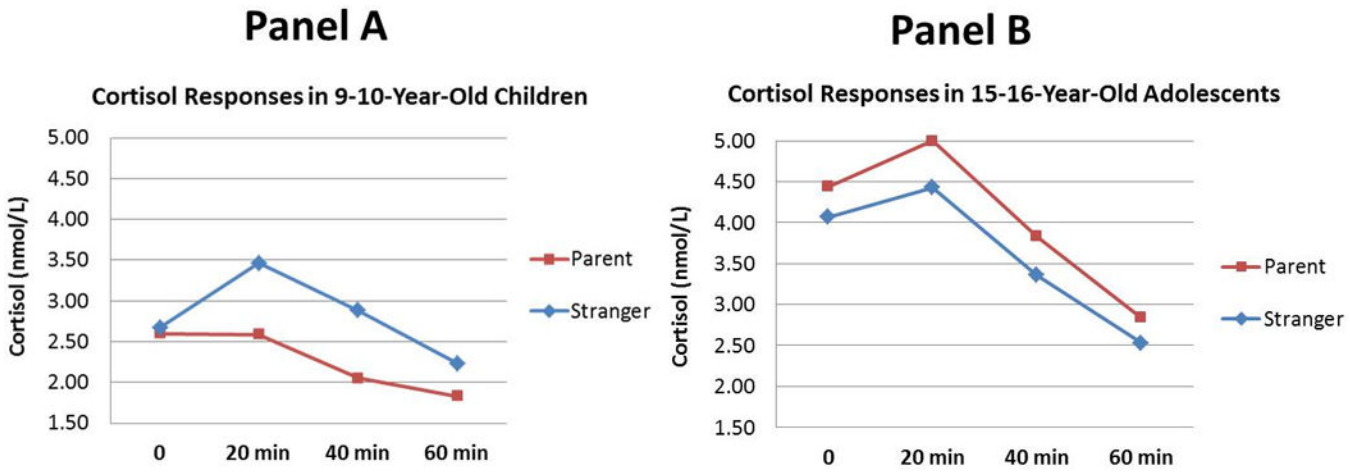

Figure 3.

Panel A: Cortisol levels assessed in nmols/l in response to the Trier Social Stress Test in typically-developing 9- and 10-year-old children. Half of the children of both sexes were tested with the parent and half with a female stranger. Data are estimated growth curves and are adapted and reprinted with permission from Hostinar et al. (2015).

Panel B: Cortisol levels assessed in nmols/l in response to the Trier Social Stress Test in typically-developing 15- and 16-year-old children. Half of the children of both sexes were tested with the parent and half with a female stranger. Data are estimated growth curves and are adapted and reprinted with permission from Hostinar et al. (2015).