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Parental buffering of fear and stress neurobiology: Reviewing parallels across rodent, monkey, and human models

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Abstract

It has been long recognized that parents exert profound influences on child development. Dating back to at least the seventeenth-century Enlightenment, the ability for parents to shape child behavior in an enduring way has been noted. Twentieth-century scholars developed theories to explain how parenting histories influence psychological development, and since that time, the number of scientific publications on parenting influences in both human and nonhuman animal fields has grown at an exponential rate, reaching numbers in the thousands by 2015. This special issue describes a symposium delivered by Megan Gunnar, Regina Sullivan, Mar Sanchez, and Nim Tottenham in the Fall of 2014 at the Society for Social Neuroscience. The goal of the symposium was to describe the emerging knowledge on neurobiological mechanisms that mediate parent–offspring interactions across three different species: rodent, monkey, and human. The talks were aimed at designing testable models of parenting effects on the development of emotional and stress regulation. Specifically, the symposium aimed at characterizing the special modulatory (buffering) effects of parental cues on fear- and stress-relevant neurobiology and behaviors of the offspring and to discuss examples of impaired buffering when the parent–infant relationship is disrupted.

Keywords

Parents; Social buffering; Fear; Stress; Amygdala; Prefrontal cortex

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PARENTAL SOCIAL BUFFERING

Humans and other altricial species are dependent on parental care during early development. High-quality care not only reduces infants’ distress and buffers their stress physiology (Gunnar & Donzella, 2002; Hennessy, Kaiser, & Sachser, 2009), but also affects the neurobehavioral and cognitive development of the infant (Tang, Reeb-Sutherland, Romeo, & McEwen, 2014), has persistent influences on its gene transcription (Champagne, 2013; McGowan, Sasaki, & Roth, 2014; Meaney & Szyf, 2005), and regulates what it learns about the environment (Sorce, Emde, Campos, & Klinnert, 1985). This is well established, and there are well-developed theories surrounding these phenomena. Furthermore, it is well known that when we are young and dependent on adult care for survival, the mere presence of attachment figures can block the activation of stress physiology even when the infant is expressing strong behavioral distress (Levine, Johnson, & Gonzalez, 1985; Nachmias, Gunnar, Mangelsdorf, Parritz, & Buss, 1996). This effect is known as parental social buffering. What remains less worked out in both humans and other species are the neurobiological underpinnings for these early stress-buffering processes and for the parental modulation of learning and adaptation to the environment.

In adulthood, members of many social species gain emotional relief from the presence of conspecifics, especially if there is previous familiarity with these social partners (Hostinar, Sullivan, & Gunnar, 2014; Kikusui, Winslow, & Mori, 2006). For instance, neuroimaging studies in human adults suggest that romantic partners can dampen neural activation to threat (Coan, Schaefer, & Davidson, 2006) or pain (Eisenberger et al., 2011). It is not clear, however, the extent to which the neurobiology of social buffering of fear and pain in adulthood is similar to such phenomena in infancy, childhood, and adolescence or whether the social buffering of fear undergoes changes with development, similar or related to effects observed with the neurobiology of fear learning/extinction (see below). It is also not known whether studies examining stress responses of the hypothalamic-pituitary-adrenocortical (HPA) and sympathetic-adrenomedullary (SAM) systems are studying the same phenomena as the work on the social buffering of fear emotions. Work on social buffering of these neuroendocrine stress systems among human adults has shown that such buffering may be more effective for men and/or may require physical contact for women (e.g., Ditzen et al., 2007; Kirschbaum, Klauer, Filipp, & Hellhammer, 1995) and can be observed even when participants describe no reductions in emotional responses to the threatening event. To complicate issues even further, there is emerging evidence that cultural norms and practices shape expectations about social support and influence the types of support that are effective in dampening physiological stress responses (Taylor, Welch, Kim, & Sherman, 2007).

Although much of the work on social buffering has focused exclusively either on early development or adulthood, there is emerging evidence that the potency of social buffers, particularly caregivers, changes in predictable ways across development (Hostinar, Johnson, & Gunnar, 2015) and that these shifts may intersect with age-related changes in the neurobiology of fear learning and extinction (Callaghan, Sullivan, Howell, & Tottenham, 2014; Moriceau & Sullivan, 2006) and age-related changes in the HPA axis (Gunnar & Vazquez, 2001; Moriceau & Sullivan, 2004).
DEVELOPMENT OF FEAR LEARNING

Learning to recognize danger is critical to survival and has already been found to be a robust paradigm for cross-species analysis. We suggest that the neurobiology of fear is uniquely suited to the study of social buffering because fear is a relatively well-described neurobehavioral system (see Pattwell, Mouly, Sullivan, & Lee, 2013 for review) and provides a template for assessing the neurobiology of social buffering throughout the lifespan.

Regarding fear learning in humans and other animals, there is evidence that the ventromedial prefrontal cortex (PFC) plays a different role in fear learning in children, adolescents, and adults (Britton et al., 2013). With respect to social buffering, it may be especially important that there are developmental differences in the neural architecture underlying fear extinction. Notably, in younger organisms fear extinction may depend more on the amygdala, whereas with development and into adulthood, there are joint roles in extinction played by the amygdala, ventromedial PFC, dorsal anterior cingulate, and hippocampus (Livneh & Paz, 2012; Shechner, Hong, Britton, Pine, & Fox, 2014). Furthermore, there is recent evidence that the neurocircuitry of fear continues to develop into adolescence with continued developmental changes in the amygdala, PFC, and hippocampus (e.g., Pattwell et al., 2012).

Regarding the integration of social buffering and fear learning, there is ample evidence in rodents that social buffering has profound effects on the fear system throughout the lifespan. For example, early in development when the organism is wholly dependent on adult care, maternal presence blocks pups’ fear learning, presumably to prevent pups from learning to fear the mother or nest (Moriceau, Roth, & Sullivan, 2010). The neural mechanism for this inhibition of fear learning is maternal social buffering blockade of amygdala plasticity (Moriceau & Sullivan, 2006; Shionoya, Moriceau, Bradstock, & Sullivan, 2007). Maternal presence can also alter the development of the PFC. The social source of social buffering also changes: while social buffering cues learned in early life remain important, the effectiveness becomes attenuated and conspecifics gain importance (i.e., cage mates and reproductive partners, Kikusui et al., 2006). While social buffering effects mediated by the amygdala appear consistent during development, the assessment of other brain areas involved in stress reduction and fear has received little attention (Sevelinges et al., 2007, 2011; Sevelinges, Sullivan, Messaoudi, & Mouly, 2008). What is not known and rarely studied is whether this type of integration of social buffering and fear learning holds true in humans and other primates and what implications it has on understanding the basic mechanisms of fear in our highly social species.

DEVELOPMENTAL SWITCHES IN FEAR LEARNING AND STRESS BUFFERING

There is emerging evidence of developmental switches or transition points in the neurobiology underlying fear behaviors (e.g., amygdala–PFC circuitry) that have been identified in rodents, monkeys, and humans (Gabard-Durnam et al., 2014; Gee et al., 2013; Moriceau & Sullivan, 2006; Morin et al., 2015). The neurobiology of these switch/transition points is poorly understood, but they seem to coincide with increasing independence from
caregivers. In humans, there is emerging evidence of developmental switch points when parental social buffering may cease to be effective in reducing stress responses. For example, during adolescence (Hostinar et al., 2015), associated with the midpoint in pubertal development (Doom, Hostinar, VanZomeren-Dohn, & Gunnar, 2015), parental social buffering becomes less potent. Whether peers or other conspecifics become more potent in buffering stress and emotions at that point is not yet known.

Critically, in rodents and possibly in other species including humans and nonhuman primates, it has been suggested that shifts in the potency of parental buffering may correspond to maturational changes in the activity of fear/defensive neural circuits and their regulation (Landers & Sullivan, 2012). The rodent literature, using remote telemetry local field potential recordings of pups naturally interacting with their mother in the nest, suggests that this developmental transition may be related to the functional emergence of the PFC (Sarro, Wilson, & Sullivan, 2014).

**EARLY LIFE STRESS**

Finally, in addition to the fact that there appears to be crosstalk between the neurobiological mechanisms of social buffering and those of fear learning/extinction during development, these intersections have been relatively unexplored. Another motivation for integrating these areas is that early life stress has profound influences on both domains in ways we may never understand unless we study them together. Thus, there is increasing evidence that the most potent form of early life stress is derived from removing or damaging the capacity of caregivers to provide a powerful stress buffer for the developing organism (Gunnar & Herrera, 2013). There is also some evidence that early social deprivation in the form of orphanage rearing interferes with the child’s ability to use the parent to buffer stress when they are later adopted into a supportive home (Fries, Shirtcliff, & Pollak, 2008; McCormack, Newman, Higley, Maestripieri, & Sanchez, 2009). This decrease in buffering may result from premature closure of the amygdala–PFC sensitive periods. Indeed there is evidence that early life stress may speed up the development of fear neurocircuitry, resulting in a premature appearance of adult-like fear retention and extinction (Cowan, Callaghan, & Richardson, 2013). These latter two findings raise the possibility that the timing of maturation of fear learning neural circuits is tied to the effectiveness of the parent or caregiving figures as stress buffers, thus further arguing that these two separate areas of research need to be integrated.

To date, the fields of neuroscience and psychology have yet to offer comprehensive models of stress- and fear-regulation across development. This issue contains papers that each addresses the central role of caregiving in neuroaffective development across three different species. A paper by Gunnar and Hostinar and another from Nim Tottenham present findings to show how these bio-environmental processes operate in the human at the level of the HPA axis and amygdala–PFC cortex circuitry. Sanchez, McCormack, and Howell present a paper describing parallel findings in nonhuman primates, focusing on a rhesus monkey model with naturally occurring disruptions of mother–infant bonds. Sullivan and Perry present rodent data that provide mechanistic explanations for these effects that might generalize up to the human. There is a high degree of convergence across species, and this convergence allows
for designing testable theoretical models that encompass the social environment, neurobiology, and behavioral development. Social relationships can alleviate stress across the lifespan and can also serve as a milieu for learning self-regulation skills; thus the topics proposed here not only serve as catalysts for integrating work across multiple disciplines, but also are directly relevant to the general public and to the well-being of society as a whole.

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