Chapter 11

Caregiver roles in children's threat and safety learning

Neuroscientific evidence and real-world implications

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Caregiver-child attachment is foundational to children's fear development (Bowlby, 1969). The attachment bond is thought to serve as a base from which the developing child can begin to learn about the external world, including which aspects of the environment are safe and which pose a threat (Tottenham, 2014). Neuroscientific research draws on threat and safety learning paradigms to chart the development of both normative fear and pathological anxiety. In this chapter we adopt an attachment perspective, leveraging data from neuroscience, evolutionary biology, and experimental psychology to detail how caregivers modulate threat neurocircuitry and associated anxiety trajectories in their children. In doing so, we elucidate the role of caregiving in children's developing understanding of threat, both in the context of laboratory-based experimental paradigms and in the face of real-world threat exposure in the form of ethnic-racial discrimination experiences.

Threat and safety learning

Laboratory threat learning paradigms provide a powerful translational platform for investigating the neural underpinnings of both developmental processes and stress-related disorders, such as anxiety disorders. Fear conditioning, a form of associative learning, is one such widely used experimental paradigm for investigating the psychophysiological processes and neural mechanisms subserving threat learning in a range of mammalian species (Shechner et al., 2014). In classical fear conditioning paradigms, a neutral conditioned stimulus (CS, e.g., light) is repeatedly paired with an aversive unconditioned stimulus (US, e.g., shock). These repeated pairings yield a CS-US association, whereby the previously neutral stimulus is now processed as a threat cue and begins to produce a conditioned response (CR, e.g., freezing behavior). Some paradigms also probe safety learning processes via *two* CSs, one paired with the US (CS+) and another unpaired (CS-; Michalska et al., 2016; Mullins et al., 2021). When the CS+, but not the CS-, elicits a CR, an organism's ability to respond adaptively to future similarly threatening events while maintaining an understanding of situations that remain safe is enhanced. Extinction, on the other hand, is a process during which the CS+ is presented repeatedly in the absence of the US, leading to an attenuated CR. Of

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note, extinction does not eradicate the initial learned association between the CS+ and the US. Rather, it creates a new learned association by which the CS+ is now associated with the *absence* of the US (Bouton, 2004). Finally, *extinction recall* occurs when the extinguished CS+ is presented again at a later time, with the general consensus that low levels of fear expression reflect successful extinction recall and high levels of fear expression reflect poor extinction recall (Glenn et al., 2020; 2021; Michalska, et al., 2019).

Associative threat learning processes like conditioning, extinction, and extinction recall are adaptive when executed at a level proportionate to both the likelihood and severity of the potential threat (Fanselow, 2018). However, these forms of learning can also become a source of pathology when they go awry and fear becomes so pervasive that it interferes with normal functioning (Rosen and Schulkin, 1998). A common feature across anxiety disorders is aberrant and excessive anticipatory responding under conditions of threat uncertainty (Grupe and Nitschke, 2013; Michalska et al., 2022) whereby anxious individuals may appraise the unknown probability of a particular outcome (i.e., whether a CS will predict an aversive US) as overly likely (Baker and Galván, 2020). Perturbations in threat learning can occur when, for example, fear conditioned responses are triggered in the absence of any CS-US contingency, or when an individual is impaired in recognizing safety cues, particularly ones resembling previously learned threat cues (Lissek et al., 2005). Understanding the neural mechanisms underlying associative learning of threat and safety can elucidate processes shaping the development of both normative fear and pathological anxiety, the most prevalent form of child psychopathology (Kessler et al., 2012) with diagnostic rates known to increase as children enter adolescence (Canino et al., 2004).

Neural processing of threat

Threat and safety learning involves processing sensory information about the CS and the US. Because these stimuli are frequently presented in distinct sensory modalities (e.g., visual light and tactile shock), they activate different sensory cortices (Shechner, 2014). Neuroimaging research in both animals and humans further implicates a network of regions in threat and safety learning including the amygdala, the medial prefrontal cortex (mPFC), and the hippocampus (Maren, 2011). The amygdala, an almond-shaped structure in the medial temporal lobe, forms the core of the neural network that processes threatening stimuli, including detecting threat and activating fear behaviors in response to dangerous stimuli (Fanselow and LeDoux, 1999). The hippocampus is a highly interconnected region that contributes to the regulation of threat responding by segmenting information about an environmental stimulus and distributing this information to various regions in the brain (Meyer et al., 2019). Given the key role of the hippocampus in disambiguating cues that have different meanings in different contexts (Maren et al., 2013) and that its projections modulate amygdala-prefrontal function by providing information about the extent of threat and safety in the environment (Fanselow, 2000),

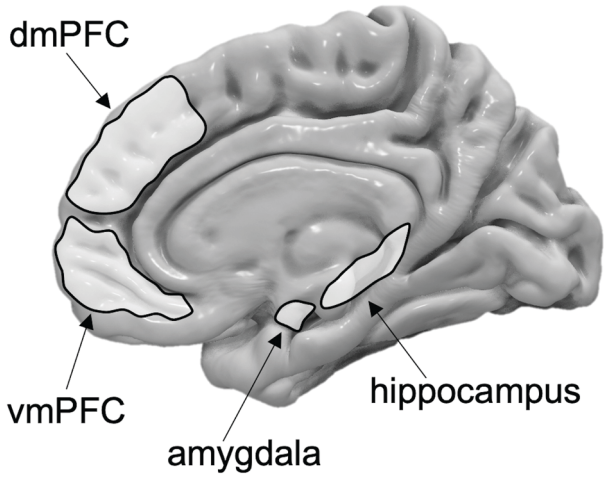

Figure 11.1 Neurocircuitry underpinning threat and safety learning.

the hippocampus is thought to be central for conditioned inhibition. The mPFC receives input from subcortical structures, like the amygdala and hippocampus, which enables the encoding of threat-relevant information to determine behavioral outputs, including the expression and regulation of fear (Alexandra Kredlow et al., 2022) (Figure 11.1).

Animal models of threat and safety learning

A potential fear circuitry in the brain has been elucidated in rodent models, suggesting that information about the CS and the paired US initially converges in the amygdala which is likely involved in both the acquisition and expression of acquired fear (Maren et al., 1996). In other words, sensory information from sensory cortical regions (e.g., visual cortex, auditory cortex) is received by the amygdala which in turn projects to targets in the brainstem that mediate CRs (McDonald et al., 1996). At first, the neutral CS produces weaker amygdala activation relative to the US, but this response is strengthened with repeated CS-US pairings, reflecting a learned association. The *prelimbic region* of the mPFC in rodent brains, homologous to the dorsomedial prefrontal cortex (dmPFC) in human brains, is thought to enhance the expression of fear conditioning via excitatory projections to the amygdala (Sierra-Mercado, Padilla-Coreano, and Quirk, 2011). Importantly, once associations are formed, the CS can elicit a strong amygdala response even in the absence of the US, sending subsequent projections to the brainstem and motor areas that control the expression of behavioral (e.g., freezing), autonomic (e.g., skin conductance response), and endocrinergic (e.g., hormone release) fear responses (LeDoux, 2000). Conversely, during extinction, inhibitory circuits in the

amygdala *prevent* neuronal excitation (Royer and Paré, 2002). Simultaneously, the *infralimbic region* of the rodent mPFC, homologous to the ventromedial prefrontal cortex (vmPFC) in humans, attenuates the expression of fear responses through connections with these inhibitory circuits within the amygdala (Quirk and Mueller, 2008). Further contributing to threat and safety learning, the hippocampus facilitates context-specific learning and extinction recall (Corcoran and Maren, 2004). Given the complementary roles the amygdala, mPFC, and hippocampus play in successful conditioning and extinction, disruptions among these networks are consequential for threat and safety learning and anxiety, more broadly.

Models of anxiety emphasize exaggerated associative learning of environmental cues and aversive outcomes (Lissek et al., 2005). As noted above, the amygdala is central to the formation of CS-US associations, particularly in the context of aversive stimuli (LeDoux, 2000). This enhanced learning serves an adaptive function in the case of real threats, but when it persists in neutral or secure contexts, it can result in excessive fear and avoidance, core features of anxiety. In rodents, activity in the amygdala reflects a cue's associability with threat (Holland and Gallagher, 2006) and inhibition of this region is required to prevent freezing responses to a CS+ and allow appropriate avoidance of the US (Moscarello and LeDoux, 2013). This suggests that amygdala hyperactivity can interfere with effective safety learning and exacerbate anxiety and is consistent with work in non-human primates that finds resting amygdala metabolism predicts trait-like anxiety (Fox et al., 2008). Of note, primates with extreme anxious temperament exhibit hyperactivity even in the security of their own homes. Similarly, in mice exposure to neutral tones elicits greater amygdala activity and anxious behavior, particularly when the timing of these tones is unpredictable (Herry et al., 2007). Thus, over-anticipation of even *non-aversive* events implicates amygdala activity and anxiety.

The rodent mPFC-amygdala circuit is involved in learning about and responding to safety in potentially threatening contexts. Electrical stimulation of the rodent vmPFC reduces the expression of amygdala-mediated conditioned fear responses (Milad and Quirk, 2002), while inactivation of this region impairs the acquisition and recall of fear extinction (Sierra-Mercado et al., 2011). Further, animals without a functional hippocampus are unable to contextualize their fear and extinction memories and, instead, respond according to their net experience with the CS (Maren, 2011). This promotes the generalization of fear across multiple contexts, a key symptom of anxiety disorders (Jasnow et al., 2017).

Human studies of threat and safety learning

Complementing animal models of threat and safety learning, human neuroimaging studies likewise show that the amygdala plays a central role in enhancing responsivity to threat and safety (Delgado, Olsson, and Phelps, 2006), while the hippocampus facilitates contextual conditioning and CS-US contingency awareness. Patients with amygdala lesions, for instance, report impairments in fear conditioning (Weike et al., 2005) and amnestic patients with damage to the hippocampus

but an intact amygdala show increased autonomic reactivity during threat and safety learning paradigms, despite an inability to explicitly report the CS-US contingency (Fried, MacDonald, and Wilson, 1997). In contrast, patients with damage to the amygdala demonstrate awareness of the CS-US contingencies but fail to show elevated autonomic arousal in response to the CS+ (Phelps, 2006). Similar to patterns of activation observed in rodents during extinction recall, studies in humans also show inhibition of the amygdala coupled with activation of the vmPFC can facilitate regulatory processes crucial to safety learning (Quirk and Beer, 2006). Just as in animals, perturbations in these networks can disrupt threat and safety learning processes and, consequently, maintain or exacerbate anxiety symptoms.

Threat and safety learning in anxious individuals

Theoretical and empirical models posit a central tenet of anxiety in humans is an intolerance of uncertainty (Grupe and Nitschke, 2013; Michalska et al., 2022), defined as the perception of uncertainty as inherently threatening, regardless of the true possibility of threat (Tanovic, Gee, and Joormann, 2018). Individuals who find uncertainty less tolerable exhibit similar amygdala activity in response to both threat and safety during early trials of extinction (i.e., safety learning), whereas individuals who are more tolerant of uncertainty exhibit greater amygdala activity to threat cues compared to safety cues (Morriss, Christakou, and van Reekum, 2015). Anxious individuals thus appear to have select difficulty discriminating between threat and safety and their responses to threat may generalize to stimuli that in fact denote safety (Glenn et al., 2020). This suggests that highly anxious individuals continue to express fear in response to previously learned threat stimuli, despite the absence of threat, possibly as a result of difficulty inhibiting fear expression via elevated amygdala activity and reduced flexibility of amygdala-vmPFC circuitry.

In a study of prefrontal cortex activation during threat appraisal, the point during the recollection of extinguished fears when participants report how afraid they are of a presented CS, anxious adults exhibit reduced activation in the vmPFC relative to non-anxious adults (Britton et al., 2013), suggesting reduced neural engagement during emotional regulatory processes. Interestingly, anxious children exhibit a U-shaped pattern of activation in response to the most extreme CS+ and CS-, suggesting heightened sensitivity to both threat *and* safety conditions and a decreasing ability to regulate in the presence of increasingly similar stimuli (Michalska et al., 2019). Impaired hippocampus-dependent associative learning may be an additional vulnerability factor for anxiety (Lambert and McLaughlin, 2019). Specifically, humans with dysregulated hippocampal function may have difficulty remembering the details of an aversive event, which could contribute to anxiety stemming from the anticipation of a similar event in the future.

Perturbations within and among key neural regions, namely the amygdala, vmPFC, and hippocampus, can disrupt threat and safety learning and subsequently elicit, maintain, or even exacerbate anxiety symptoms. Childhood, in particular, is a period characterized by rapid development of this neurocircuitry (Gogtay et al.,

2004; Wierenga et al., 2014), as well as the emergence of individual differences in threat anticipation and anxiety symptoms (Michalska et al., 2019). Importantly, findings on the neurobiology of fear and anxiety reviewed so far have come from research studying threat or defense responses in isolation from their social context. But as every caregiver knows, children do not acquire knowledge about what is threatening and what is safe in a social vacuum. To more fully characterize such knowledge, it is imperative to consider how caregivers, who play an outsized role in the lives of their children during this time, may regulate children's threat neurobiology and shape their understanding of safety.

Caregiver roles in children's threat and safety learning

Caregiver-child attachment is foundational to children's fear development. Even the earliest psychological theories of attachment posit that a primary driver of attachment formation is a caregiver's ability to modulate fear in their child (Bowlby, 1969). The routine presence of the caregiver, coupled with high levels of warm caregiving, promotes attachment formation (Ainsworth and Bell, 1970; Anisfeld et al., 1990), with sensitive and responsive caregiving fostering secure attachment relationships. The attachment bond is thought to serve as a base from which the developing individual can begin to learn about the external world, including which aspects of the environment are safe and which pose a threat (Tottenham, 2014).

Attachment formation

The attachment bond is a foundation from which future environmental exploration is built, which implies that the formation of this bond is a key precursor to the development of threat and safety learning. In rodents, the threat system is quiescent in early life and neural circuits developing postnatally are biased toward supporting attachment learning and proximity seeking *over* threat learning (Callaghan et al., 2019). For instance, amygdala-dependent learning does not occur in infant rats younger than 10 days of age (Sullivan et al., 2000), despite pups' ability to readily detect aversive stimuli (Collier and Bolles, 1980). It is thought that fear behaviors are not learned or expressed because the amygdala is not actively engaged in contingency learning at this time. Likewise, in human infants, the amygdala is not responsive to threat cues during postnatal development (Graham, Fisher, and Pfeifer, 2013).

In the absence of amygdala-dependent fear learning, competing systems instead produce *preference* behaviors for learned associations, likely supporting pup-mother attachment. These competing systems are the same as those engaged when pups are learning their mother's natural odor (Perry et al., 2016) and begin to orient toward her scent to facilitate attachment formation (Landers and Sullivan, 2012). During this developmental period, threat conditioning thus fails to engage the neural substrates for learning fear responses, and, instead, engages the mechanisms for forming an attachment to a caregiver. This is especially noteworthy because

even though attachment has historically been considered innate, such more recent neurobiological evidence indicates a significant amount of learning that activates a biologically predisposed attachment circuit used to initiate and maintain the attachment bond. This work also helps to explain why postnatal infants, both human and nonhuman, readily learn attachments to their caregivers, regardless of the quality of care (Perry, Blair, and Sullivan, 2017). As offspring exit this developmental period, however, variations in caregiving begin to predict differences in threat and safety learning (Callaghan et al., 2019).

Caregiver presence and practices

In childhood, previous goals of attachment formation and proximity seeking are gradually replaced by goals of increasingly independent exploration. In humans, these changes are accompanied by elevated amygdala activity and maturation of the hippocampus and prefrontal cortex, enabling fear learning capacities (Gabard-Durnam et al., 2014; Silvers et al., 2017; Uematsu et al., 2012). Similarly, in young rats, stress hormone (i.e., cortisol) release facilitates amygdala activation allowing fear conditioning to emerge (Moriceau and Sullivan, 2006). At this time, the presence and proximity of a caregiver can predict differential responsivity to threat, in a process known as caregiver or social "buffering", a phenomenon where a caregiver or other significant social figure attenuates stress hormone release by blocking the hypothalamic pituitary adrenal (HPA) axis. One of the most powerful effects of social buffering is maternal social buffering of offspring, whereby the mother acquires the ability to serve as a safe haven or signal safety for the child. In the laboratory, children exhibit lower amygdala reactivity and more mature prefrontal connectivity when viewing pictures of mothers' faces, than when viewing pictures of strangers' faces (Gee et al., 2014). Children with greater attachment security exhibit the most effective amygdala suppression, suggesting secure caregiver attachment supports adaptive threat regulation. Relatedly, children's ability to appropriately inhibit fear-potentiated startle (reflexive eye blinking) is enhanced when mothers are more physically accessible (i.e., just outside the testing room versus down the hall; van Rooij et al., 2017). Animal models arrive at similar conclusions, notably that stress reduction in the parent's presence can block fear conditioning in rat pups through attenuation of amygdala learning-induced plasticity (Moriceau and Sullivan, 2006).

As reviewed above, among rodents, the mere presence of the mother during fear learning causes the infant to approach rather than avoid threat cues. Rodent caregiver deprivation, on the other hand, can result in the early emergence of adult-like fear learning via alterations in fronto-amygdala circuitry (Callaghan and Richardson, 2011) and earlier emergence of amygdala function (Moriceau et al., 2006) and structural maturation (Ono et al., 2008). Neural connectivity is similarly affected in human children experiencing early maternal deprivation (Gee et al., 2013), indicating that maternal deprivation accelerates the development of the threat learning system involving the amygdala. Compared to youth raised by their

biological parents, previously institutionalized youth exhibit broader amygdala-hippocampal-PFC network connectivity during threat conditioning, providing further evidence that caregiver absence can alter threat neurocircuitry and threat and safety learning processes (Silvers et al., 2016).

Threat neurocircuitry is not only impacted by caregiver presence, but also by the quality of care received. Children of mothers who exhibit high levels of caregiver warmth display reduced amygdala responsivity to facial emotions relative to children of mothers who endorse low levels of caregiver warmth (Stevens et al., 2021), suggesting maternal warmth helps attenuate threat responsivity in children. Even later in development, adolescents who report receiving more parental support show dampened amygdala reactivity to threat cues (Romund et al., 2016). Conversely, chronic harsh parenting has profound adverse consequences for brain development including reduced amygdala-insula connectivity and less effective deactivation of the medial temporal lobe to threat versus safety stimuli (La Buissonnière-Ariza et al., 2019). Rat pups reared with an abusive mother demonstrate disrupted engagement of the infralimbic cortex, homologous to the human vmPFC, during conditioning and the mother's ability to buffer fear responses is compromised (Robinson-Drummer et al., 2019). Human children suffering physical and sexual abuse exhibit reduced amygdala and hippocampal volume and alterations in physiological responsivity to threat (McLaughlin et al., 2016). Caregiver influences may even extend beyond severe forms of abuse and neglect to more mildly negative caregiving practices. A recent study of fathers and daughters, for instance, suggests that high levels of criticism can subtly impact safety learning in anxious youth (Mullins et al., 2021). Together, these findings illustrate neural mechanisms through which attachment security, caregiver presence and accessibility, and the quality of caregiving practices jointly shape threat and safety learning in children. Of note, the vast majority of this empirical work, is conducted in controlled laboratory settings. How caregiving similarly implicates the neurocircuitry subserving children's understanding of real-world threats is less clear.

Children's understanding of real-world threats

One salient real-world threat that may rewire threat neurocircuitry is ethnic-racial discrimination, the unfair treatment of individuals due to their ethnicity or race (Carter and Forsyth, 2010). The turn of the century has seen compounding, extensive, and harmful effects of ethnic-racial discrimination on mental health during childhood and adolescence (Priest et al., 2013), with higher rates of exposure associated consistently with elevations in anxiety. Far less work explores the neurobiological mechanisms mediating detriments in mental health, and virtually no studies of ethnic-racial discrimination adopt a threat and safety learning perspective that elucidates how caregivers can protect children from the harmful effects of ethnic-racial discrimination. This is especially surprising given experiences of ethnic-racial discrimination are, in fact, instances of learning that condition how future

racially charged social interactions are experienced. Indeed, the neural structures supporting the physical component of pain are shared with those supporting the experience of social pain that results from rejection, exclusion, and harassment (Eisenberger, 2012). Repeated encounters with and anticipation of ethnic-racial discrimination shape how children understand their experiences, form expectancies about future encounters, and monitor and prepare themselves for social interactions in the social environments they inhabit (Blair and Raver, 2012). Thus, neural circuits involved in threat and safety detection are critical for monitoring the environment for potential social threats and coordinating neurophysiological responses. When these systems are perturbed by chronic stressors like ethnic-racial discrimination, this can engender anxious hypervigilance, or excessive anticipatory threat responding, a core feature of pathological anxiety. Thus, embedding the study of ethnic-racial discrimination into a theoretical framework centered on laboratory-based threat and safety learning can help clarify neurobiological mechanisms by which real-world experiences of ethnic-racial discrimination contribute to anxiety.

Neurobiological consequences of discrimination

Emerging functional and structural neuroimaging evidence documents detrimental and compounding effects of ethnic-racial discrimination on the neural architecture subserving threat and safety learning (Hobson et al., 2022). Individuals who report higher levels of intersectional discrimination exposure (racism, sexism, heterosexism) exhibit heightened spontaneous amygdala activity and greater functional connectivity with neighboring regions during resting state fMRI (Clark, Miller, and Hegde, 2018). Additionally, participants subjected to racially motivated social exclusion demonstrate higher levels of dmPFC and vmPFC activation than during experimental conditions of social inclusion (Masten, Telzer, and Eisenberger, 2011), demonstrating effects of racial bias on neural regions linked to social distress and emotion regulation. Structural investigations posit similarly consequential effects of ethnic-racial discrimination, with one study showing smaller hippocampal volumes in children residing in regions with more prejudicial social policies and attitudes relative to youth living in lower stigma contexts (Hatzenbuehler et al., 2021) and another study documenting larger amygdala volumes in adults exposed to higher levels of sexism, racism, and ageism (Rosario et al., 2020). Further, people at risk of ethnic-racial discrimination are particularly attuned to cues that signal certain social situations as threatening or safe, suggesting that ethnic-racial discrimination exposure also implicates threat-relevant attentional processes (Purdie-Vaughns et al., 2008). This work, while modest in scale, suggests the neural substrates underlying laboratory-based threat learning are a reliable proxy for real-world threats like exclusion, harassment, and discrimination. Examining whether caregivers have the capacity to play an equally influential role in real-world threat learning as they do in the laboratory could inform preventive efforts targeting how the toll discrimination takes on anxiety may be offset.

Caregiving in the context of discrimination

A nascent literature articulates the shortcomings of an attachment framework that does not adequately attend to the social context of attachment formation between caregivers and children of color (Stern, Barbarin, and Cassidy, 2022). Families of color both face unique sociocultural stressors that may tax the caregiver-child relationship and also possess rich cultural resources to buffer and counter such stressors. Caregivers of color face the undue burden of providing their children with protection and safety in the face of powerful threats like intergenerational trauma of discrimination, ongoing racist policies that disproportionately harm people of color, and daily experiences of mistreatment based on race and ethnicity. Importantly, for youth of color, caregiver attachment security may be an especially robust predictor of well-being due to greater activation of the attachment system triggered by discrimination-related stress (Parade, Leerkes, and Blankson, 2010). Therefore, if threat and safety learning research is to make meaningful progress in characterizing caregiver roles in children's understanding of real-world threats, like ethnic-racial discrimination, and their potential influence on threat neurocircuitry and anxiety, we must as a field increase our attention on attachment relationships in caregiver-child dyads of color and other historically marginalized groups.

Caregiver socialization efforts are the primary mechanism through which children understand and practice responding to experiences of ethnic-racial discrimination (Smalls-Glover et al., 2013). Importantly, the efficacy of caregiver socialization messages about external threats hinges on the quality of the attachment relationship (Darling and Steinberg, 1993), such that children may respond more positively to caregiver ethnic-racial socialization if they themselves are in a responsive caregiving environment (Smalls, 2009). It is, thus, unsurprising that caregiver efforts to protect children from the deleterious effects of ethnic-racial discrimination are best practiced in the context of positive caregiving practices. Specifically, caregivers who are engaged in warm, supportive relationships with their children tend to provide them with cultural socialization and ethnic-racial pride messages that exhibit the most consistently protective effects (Smalls, 2009). While effects of discrimination on mental health are more severe at higher levels of exposure, nurturant and involved caregiving and caregiver closeness have been shown to attenuate these costs (Brody et al., 2006). For example, high ethnic pride is associated with high parental acceptance, which is, in turn, linked to reduced anxiety in children (Gray, Carter, and Silverman, 2011). Further, children of parents who endorse high levels of cultural pride reinforcement messages have significantly lower anxiety scores relative to children of parents who endorse low levels of these messages (Bannon et al., 2009). Caregivers who practice appropriate monitoring of and involvement with their children also transmit more frequent cultural socialization messages (Murry et al., 2014). Importantly, these ethnic-racial socialization efforts executed in a positive caregiving environment predict better child psychological well-being in the context of ethnic-racial discrimination (Varner et al., 2018). Together, these

findings suggest that secure attachment relationships and high-quality caregiving practices serve to protect children from the costs of ethnic-racial discrimination on mental health. We contend that caregivers who facilitate their children's understanding of and responding to these unique and potent threats place them on a trajectory for adaptive threat and safety learning that may protect against the development of anxiety. Given the speculative nature of our argument, empirical testing represents immediate next steps for future work.

In conclusion, the current chapter draws on data from neuroscience, evolutionary biology, and experimental psychology to make a case for the study of discrimination from a threat and safety learning perspective. Clarifying whether exposure to ethnic-racial discrimination alters the neurocircuitry involved in learning about and responding to threat and identifying aspects of caregiving that play into these processes will provide new insights into the neural mechanisms of ethnic and racial health disparities and ways in which they can be offset. Future work should empirically test altered threat neurocircuitry as a neurobiological pathway by which ethnic-racial discrimination disrupts threat and safety learning and elevates anxiety, and how caregivers can buffer these effects. Shifting political climate and positive momentum notwithstanding, structural inequality and ethnic-racial discrimination are significant, historic barriers with limited short-term solutions. As we work towards systemic structural change, it is imperative we simultaneously identify and leverage proximal means to protect children from the harmful effects of ethnic-racial discrimination.

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