

# Persistent Fear and Anxiety Can Affect Young Children's Learning and Development

WORKING PAPER 9

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The National Scientific Council on the Developing Child, housed at the Center on the Developing Child at Harvard University, is a multi-disciplinary collaboration designed to bring the science of early childhood and early brain development to bear on public decision-making. Established in 2003, the Council is committed to an evidence-based approach to building broad-based public will that transcends political partisanship and recognizes the complementary responsibilities of family, community, workplace, and government to promote the well-being of all young children. For more information, go to [www.developingchild.net](http://www.developingchild.net).

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# The Issue

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ENSURING THAT YOUNG CHILDREN HAVE SAFE, SECURE ENVIRONMENTS IN WHICH TO GROW, LEARN, and develop healthy brains and bodies is not only good for the children themselves but also builds a strong foundation for a thriving, prosperous society. Science shows that early exposure to circumstances that produce persistent fear and chronic anxiety can have lifelong consequences by disrupting the developing architecture of the brain. Unfortunately, many young children are exposed to such circumstances. While some of these experiences are one-time events and others may reoccur or persist over time, all of them have the potential to affect how children learn, solve problems, and relate to others.

All children experience fears during childhood, including fear of the dark, monsters, and strangers. These fears are normal aspects of development and are temporary in nature. In contrast, threatening circumstances that persistently elicit fear and anxiety predict significant risk for adverse long-term outcomes from which children do not recover easily. Physical, sexual, or emotional abuse; significant maltreatment of one parent by the other; and the persistent threat of violence in the community are examples of such threatening circumstances in a child's environment.

Studies show that experiences like abuse and exposure to violence can cause fear and chronic anxiety in children and that these states trigger extreme, prolonged activation of the body's stress response system. In studies with animals, this type of chronic activation of the stress system has been shown to disrupt the efficiency of brain circuitry and lead to both immediate and long-term physical and psychological problems. This is especially true when stress-system overload occurs during sensitive periods of brain development. While much of the evidence for the effects of stress on the development of brain architecture comes from animal studies, strong similarities in the processes of brain development across species indicate that experiences of persistent fear and chronic anxiety likely exert similarly adverse impacts on the developing brain in humans. Thus, stress-system overload can significantly diminish a child's ability to learn and engage in typical social interactions across the lifespan.

Many policymakers, educators, and even medical professionals are unaware of the potentially significant, long-term risks of exposure to fear-provoking circumstances in children and

lack information about the prevalence of these situations in their communities. Critically, 1 in every 7 children, and nearly 1 out of every 40 infants, in the United States experience some form of maltreatment, including chronic neglect or physical, emotional, or sexual abuse.<sup>1,2</sup> Child maltreatment has been shown to occur most often in families that face excessive levels of stress, such as that associated with community violence, parental drug abuse, or significant social isolation.<sup>3</sup> Research also tells us that nearly half of children living in poverty witness violence, or are indirectly victims of violence.<sup>1</sup> Clearly, for children in these circumstances, the frequent and repetitive threats around them

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create the potential for heightened fear and chronic anxiety.

Behavioral neuroscience research in animals tells us that serious, fear-triggering experiences elicit physiological responses that affect the architecture of the brain as it is developing. These experiences cause changes in brain activity and have been shown to have long-term, adverse consequences for learning, behavior, and health. Studies show that solutions for children are available through programs that effectively prevent specific types of fear-eliciting events, such as physical or sexual abuse. The timely

implementation of such interventions can prevent and treat the harmful effects of exposure to extreme, fear-eliciting circumstances. In addition to these preventive measures, there also are effective treatments for children experiencing high levels of anxiety or chronic fear that

result from serious emotional trauma. Despite this rapidly increasing knowledge base, however, significant gaps continue to exist in how society responds to the developmental needs of children who regularly experience serious, fear-inducing events.

## What Science Tells Us

SOME TYPES OF FEAR ARE NORMAL ASPECTS OF development. Infants begin to experience feelings of fear and differentiate them from other emotions between 6 and 12 months of age.<sup>4,5</sup> Over the course of the early childhood period, toddlers and preschoolers typically express fear of a wide variety of events or individuals. For example, it is not unusual for a young child to react with wariness or distress when greeted by an unfamiliar adult. Such responses are often called “stranger anxiety” and typically first emerge at around 9 or 10 months of age. This hesitancy toward unfamiliar people generally continues throughout childhood, but diminishes over time, as children’s social worlds expand and they interact with increasing numbers of caregivers, relatives, neighbors, and other familiar adults.

Later in early childhood, it is common for children to express fear of both imagined and real circumstances. The emergence and development of imagination, for example, may lead to fear of monsters or the dark. These reactions are

for why children outgrow these normative fears. Many fears are a result of the difficulty young children have in distinguishing between the real and the imaginary. As they get older, children get better at understanding what is real and what it means for something to be “make believe.” At the same time, they develop a growing sense of control and predictability over their immediate environment, so that even very young children are less frightened by events if they have some control over them. For example, a toy that scares 12-month-olds because it is loud and unpredictable will elicit less fear if the children are shown how to turn it on and off and are allowed to do so.<sup>6</sup> As they get older, children develop the cognitive and social skills needed to better understand predictability in their environment and, therefore, gain a greater sense of control. As these developmental capacities are mastered, many of the normal fears of childhood begin to disappear. Thus, the emergence and course of typical childhood fears are different from the fears and anxiety elicited by traumatic situations such as physical or sexual abuse or exposure to violence: While typical fears disappear with age, the fear and anxiety elicited by maltreatment and other threatening circumstances do not.

### **The emergence and course of typical childhood fears are different from the fears and anxiety elicited by traumatic situations such as physical or sexual abuse or exposure to violence.**

typical and usually peak between 4 and 5 years of age. Generally speaking, normal preschool fears do not disrupt a child’s life, and they dissipate by age 7 or 8. That is, while children may express these fears at certain times (e.g., bedtime) or in response to certain events (e.g., being surprised by a clown at a birthday party), their overall behavior does not otherwise suggest that they are generally fearful or distressed.

Scientific research provides an explanation

**Early exposure to extremely fearful events affects the developing brain, particularly in those areas involved in emotions and learning.** A large and growing body of research, including animal studies as well as recent neuroimaging studies of human adults, has revealed groundbreaking insights into the brain circuitry that underlies how we learn to be afraid<sup>7,8</sup> and how we come to associate a specific event or experience with negative outcomes.<sup>9,10</sup> Two extensively studied structures located deep in the brain—the amygdala and the hippocampus—are involved in fear conditioning.<sup>9,10</sup> The amygdala detects whether a stimulus, person, or event is threatening<sup>9,10</sup> and the hippocampus links the fear

response to the context in which the aversive stimulus or threatening event occurred.<sup>11</sup> Studies also show that both the amygdala and the hippocampus play an important role in how the body then responds to this threat. Elevated stress hormones such as cortisol have been shown to affect the growth and performance of the hippocampus and the activity of the amygdala in rodents and non-human primates, and early and persistent activation of the stress response system adversely affects brain architecture in these critical regions.

Beyond its impact on these two brain structures, heightened stress has also been shown in animals to impair the development of the prefrontal cortex, the brain region that, in humans, is critical for the emergence of executive functions—a cluster of abilities such as making, following, and altering plans; controlling and focusing attention; inhibiting impulsive behaviors; and developing the ability to hold and incorporate new information in decision-making. These skills become increasingly important throughout the school years and into adulthood. Behavioral neuroscience research in animals tells us that the prefrontal cortex is highly sensitive to the detrimental effects of excessive stress exposure and that its developing architecture is vulnerable to the negative effects of chronic fear.<sup>12</sup>

**When young children experience serious fear-triggering events, they learn to associate that fear with the context and conditions that accompanied it.** Very young children can actually *learn* to be fearful through a process called “fear conditioning,” which is strongly connected to the development of later anxiety disorders.<sup>13,14,15,16</sup> In the typical circumstances of early childhood, fear responses are activated quickly and then dissipate. However, when young children are chronically exposed to perceived or real threat, fear-system activation can be prolonged. In research studies, fear conditioning involves the pairing of a neutral stimulus (e.g., a tone or a light) that normally does not elicit a negative emotional response with an aversive stimulus (e.g., pain) that produces fear. As this conditioning evolves, it solidifies the relation between the two stimuli and then generalizes the fear response to other neutral stimuli that may share similar characteristics with the aversive stimulus. Conditioned fear is apparent when individuals come to experience and express fear

within *the context in which the learning occurred*. For example, a child who is physically abused by an adult may become anxious in response to both the person and the place where the fear

**For young children who perceive the world as a threatening place, a wide range of conditions can trigger anxious behaviors that then impair their ability to learn and to interact socially with others.**

learning occurred. Over time, the fear elicited and the consequent anxiety can become generalized, and subsequent fear responses may be elicited by other people and places that bear sometimes only small resemblances to the original conditions of trauma. Consequently, for young children who perceive the world as a threatening place, a wide range of conditions can trigger anxious behaviors that then impair their ability to learn and to interact socially with others. The extent to which these problems affect physical and mental health is influenced by the frequency of the stressful exposure and/or the emotional intensity of the fear-eliciting event.

**Science tells us that unlearning fear is a fundamentally different process from fear learning.**

The process of unlearning conditioned fear is called “extinction” and actually involves physically separate and distinct areas of the brain’s architecture from those into which fear responses are first incorporated. Generally speaking, the unlearning process involves activity in the prefrontal cortex, which decreases the fear response by regulating the activity of the amygdala.<sup>17,18,19,20</sup> Research tells us that fears are not just passively forgotten over time, but they must be actively unlearned. Studies show that fear *learning* can occur relatively early in life,<sup>21,22,23</sup> whereas fear *unlearning* is only achieved later, when certain structures in the brain have matured.<sup>24,25</sup> Consequently, early fear learning can have a significant impact on the physical and mental health of a young child that can take years to remediate.

This understanding of how fear unlearning occurs can be helpful in designing interventions for anxious and fearful children. For example, research has shown that unlearning negative fear responses to specific stimuli such as



animals, insects, heights, or social situations can be accomplished successfully by presenting the aversive stimulus or circumstance at a low level of intensity while the fearful individual is in a safe context. This therapeutic approach is called cognitive behavioral therapy. Providing additional explanations for anxious behavior during these controlled exposures has proven to be particularly successful for reducing anxiety in older children with excessive fears, as their ability to understand these explanations develops. Such interventions work well with specific phobias, as well as social or generalized anxiety, but are not effective in remediating the effects of abuse or neglect.

**Chronic and intense fear early in life affects the development of the stress response system and influences the processing of emotional memories.**<sup>26,27</sup> When an individual is confronted with

a threat, stress systems are activated and elevate the levels of several different stress chemicals that are circulating throughout the body.<sup>28,29</sup> An increase in one of those chemicals, cortisol, can have a dramatic impact on how memories are processed and stored.<sup>29,30</sup> The production of cortisol and adrenalin (as well as noradrenaline in the brain) in a normal stress response leads to memory formation for events and places that generate danger. More specifically, elevated cortisol levels can strengthen the formation of memories of emotional events,<sup>31,32</sup> block the ability to unlearn fear memories,<sup>33</sup> and enhance the formation of memories of the surrounding context in which the fearful event occurred.<sup>34</sup> Interestingly, too much cortisol can also have the opposite effect and actually impair memory and learning in non-threatening contexts.<sup>35</sup> Thus, the biological response to stress is intimately involved in both fear learning and unlearning.

## Fear and Anxiety Affect the Brain Architecture of Learning and Memory

### PREFRONTAL CORTEX

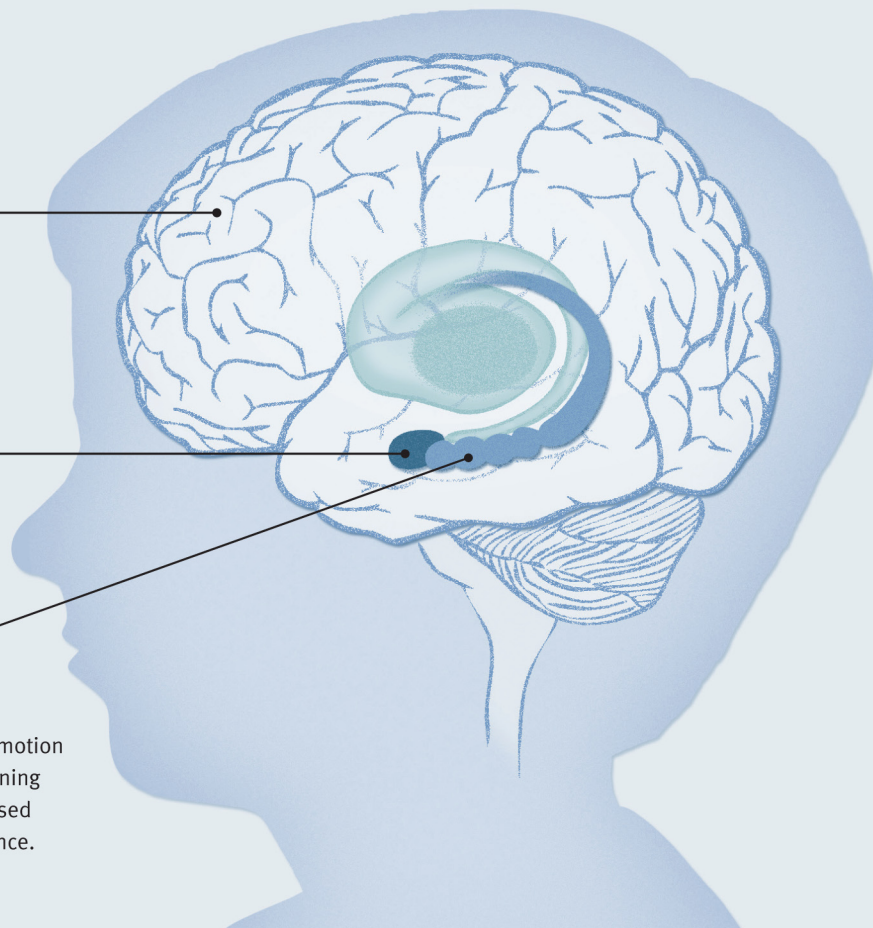
Center of executive functions; regulates thought, emotions, and actions. Especially vulnerable to elevation of brain chemicals caused by stress. Matures later in childhood.

### AMYGDALA

Triggers emotional responses; detects whether a stimulus is threatening. Elevated cortisol levels caused by stress can affect activity. Matures in early years of life.

### HIPPOCAMPUS

Center of short-term memory; connects emotion of fear to the context in which the threatening event occurs. Elevated cortisol levels caused by stress can affect growth and performance. Matures in early years of life.



Fear learning can form emotional memories that are extremely powerful and long lasting. These memories are relived by individuals who experienced a traumatic event when cues in the environment activate those memories. This repeated recall or retrieval of the memory makes emotional memories both more easily activated and more resistant to being forgotten.<sup>29,30</sup> The repeated recall of a traumatic event can lead to additional release of cortisol, even in the absence of the actual event. Behavioral neuroscience research with animals has shown that chronic elevation of cortisol can have a number of detrimental effects, including increased damage to brain cells in areas that support learning, thereby leading to increased impairment in subsequent memory formation.<sup>30,31</sup>

**Persistent fear can distort how a child perceives and responds to threat.** Fear learning typically takes place in specific contexts and results in those fears becoming associated with the places where the learning occurred. Children may also express fear in response to situations that are similar (not identical) to those initially learned or to situations that are similar to the contexts in which the original learning occurred. These are called “generalized” fear responses, and they are thought to underlie the expression of later anxiety disorders, including post-traumatic stress disorder (PTSD).<sup>15,36,37</sup> Thus, although all individuals display a heightened fear response when faced with threatening contexts,<sup>36,38</sup> individuals with anxiety disorders show this same increased fear response when faced with similar contexts that are known to be safe.<sup>36,38,39</sup> Indeed, children who have had chronic and intense fearful experiences often lose the capacity to differentiate between threat and safety. This impairs their ability to learn and interact with others, because they frequently perceive threat in familiar social circumstances, such as on the playground or in school. These responses inhibit their ability to learn and often lead to serious anxiety disorders.<sup>40,41</sup>

Young children who have been exposed to traumatic circumstances also have difficulty identifying and responding to different expressions of emotions, and, therefore, have trouble forming healthy relationships.<sup>42,43,44,45,46</sup> These deficits lead to general problems with social interaction, such as understanding others’ facial expressions and emotions. For example, children raised in physically abusive households show heightened sensitivity (compared with

non-abused children) to angry faces, which negatively affects their brain function and behavior.<sup>47,48,49,50</sup> Learning to identify anger—quickly and successfully—in order to avoid being harmed is a highly adaptive and appropriate response to an abusive environment. However, an increased tendency to assume someone is angry when his or her facial expression is ambiguous can be inappropriate and maladaptive

## Children who have had chronic and intense fearful experiences often lose the capacity to differentiate between threat and safety.

in a typical, non-threatening social setting and even dangerous in unfamiliar social settings.<sup>51</sup> This “attention bias” to threat is associated with interpreting ambiguous information in a negative fashion, and it is linked to greater vulnerability to stress and anxious behaviors as well as to a greater likelihood to respond aggressively as a form of self-defense in neutral circumstances that are erroneously viewed as threatening. Thus, the extent to which children with a heightened attention bias to threat view the world as a hostile and threatening place can be viewed as both a logical adaptation to an abusive environment and a potent risk factor for behavior problems in later childhood, adolescence, and adult life.

**Early exposure to intense or persistent fear-triggering events affects children’s ability to learn.** There is extensive and growing scientific evidence that prolonged and/or excessive exposure to fear and states of anxiety can cause levels of stress that can impair early learning and adversely affect later performance in school, the workplace, and the community. Multiple studies in humans have documented problems in cognitive control and learning as a result of toxic stress.<sup>52,53</sup> These findings have been strengthened by research evidence from non-human primates and rodents that is expanding our understanding of the brain mechanisms underlying these difficulties.

The brain region in animals that appears highly vulnerable to adversity in this regard is the prefrontal cortex, which is the critical area for regulating thought, emotions, and actions as well as for keeping information readily

accessible during the process of active learning. For example, researchers have found that elevations in brain chemicals like noradrenaline, an important neurotransmitter, can impair functions that are controlled by the prefrontal region by altering the activity of neurons in that area of the brain. In a related fashion, humans

experiencing chronic stress have been shown to perform poorly on tasks related to prefrontal cortex functioning (such as working memory or shifting attention), and their ability to control their emotions is typically impaired.<sup>12</sup>

## Correcting Popular Misrepresentations of Science

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THERE ARE A NUMBER OF WIDESPREAD MISCONCEPTIONS about how children experience, respond to, and learn fear. Many of these assumptions derive from overgeneralizations of what fears are typical at specific developmental stages as well as misunderstandings about what children can simply “outgrow” as they mature. Being afraid of strangers and monsters are common examples of typical fears. In contrast, research has demonstrated convincingly that excessive fear and anxiety caused by experiences such as abuse and neglect can affect the developing child in very different ways from the fear experiences that characterize a typical childhood.

**Contrary to popular belief, serious fear-triggering events can have significant and long-lasting impacts on the developing child, beginning in infancy.** Science tells us that young children *can* perceive threat in their environment but, unlike adults, they do not have the cognitive or physical capacities to regulate their psychological response, reduce the threat, or remove themselves from the threatening situation. Research also shows that very young infants can learn to fear certain places, events, or people. These learned fear responses may disrupt the physiology of the stress response system, making it more difficult for the body to respond appropriately to typical, mild stress in everyday contexts later in life. Furthermore, when fear is learned, normal situations and circumstances can elicit responses that are harmful to a child’s development.

**Children do not naturally outgrow early learned fear responses over time.** Fear learning early in life can often be adaptive—think about how a young child learns to stay away from hot surfaces. Thus, fear learning and associated

memories that occur early in life get built into our brain architecture and do not dissipate with age. During typical development, children learn to regulate their responses to mild threats and stresses. However, if young children are exposed to persistent fear and excessive threat during particularly sensitive periods in the developmental process, they may not develop healthy patterns of threat/stress regulation. When they occur, these disruptions do not naturally disappear.

**Simply removing a child from a dangerous environment will not by itself undo the serious consequences or reverse the negative impacts of early fear learning.** There is no doubt that children in harm’s way should be removed from a dangerous situation. However, simply moving a child out of immediate danger does not in itself reverse or eliminate the way that he or she has learned to be fearful. The child’s memory retains those learned links, and such thoughts and memories are sufficient to elicit ongoing fear and make a child anxious. Science clearly shows that reducing fear responses requires active work and evidence-based treatment. Children who have been traumatized need to be in responsive and secure environments that restore their sense of safety, control, and predictability—and supportive interventions are needed to assure the provision of these environments. Thus, it is critical that communities be equipped to address the sources of fear in children’s lives. Where indicated, children with anxiety can benefit from scientifically proven treatments, such as cognitive behavioral therapy, which have been shown to reduce anxiety and fear.



# The Science-Policy Gap

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**Advances in the science of child development tell us that significant fear-eliciting experiences early in life can disrupt the typical development of stress regulation as well as learning, memory, and social behavior, yet there is still widespread resistance in the policy arena to fully addressing the needs of young children who have been traumatized.** Building on decades of evidence from behavioral research, it is now abundantly clear that young children who are exposed to circumstances that produce persistent fear are at heightened risk for anxiety disorders and other mental health problems that persist into adulthood. Concurrently, a variety of prevention and early intervention programs have been developed to address the needs of young children who have been exposed to such fearful situations as physical abuse or family violence. The limited availability of these kinds of programs for very young children represents a striking failure to relieve immediate distress as well as prevent serious and costly long-term disability.

**The lack of availability of adequate health insurance to cover the cost of therapeutic treatment for young children who are experiencing persistent fear and chronic anxiety represents a significant lost opportunity to ameliorate preventable impairments in physical and mental health that can have lifelong implications.** The science of child development points the way toward effective approaches to the treatment of children with excessive anxiety and fear. These methods, if administered early, can reduce the incidence of anxiety disorders in children and prevent the kinds of elevated stress responses that lead to physical and mental health impairments later in life. Addressing the current gaps among what science knows about effective treatments, what is available in health care and early childhood settings, what is covered by health insurance, and the availability of coverage for all children needs to be an important policy priority.

## Policy Implications

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**Programs and policies that are designed to address domestic violence, substance abuse, and mental health problems in adults who have (or are expecting) children would have considerably stronger impacts if their focus also included the children's developmental needs, beginning in the prenatal period.** Extensive scientific evidence shows that significant mental health problems in parents can be a source of fear and stress in children and have negative effects on a child's development. Through reduced caregiving capacities, the co-occurrence of child neglect or abuse, and exposure to other sources of fear and stress, parental mental health conditions have direct consequences for the health and well-being of their children. The fear that abuse and neglect elicits in children can lead to serious dysregulation of their emotions and behavior control. That said, there are promising interventions that have been shown to be effective in preventing abuse and neglect. Prenatal home visiting for

first-time mothers provided by trained nurses is one example of a program whose effectiveness has been documented by randomized controlled trials in multiple locations.<sup>54,55</sup> Other promising approaches include specific training for

**Extensive scientific evidence shows that significant mental health problems in parents can be a source of fear and stress in children and have negative effects on a child's development.**

professionals who work with families experiencing trauma and the incorporation of developmental interventions for young children in programs that address domestic violence.<sup>56</sup> When hard evidence of program effectiveness is available, the imperative of providing appropriate

preventive or intervention services is clear. When program evaluation data on successful prevention or interventions for specific threats to child well-being are limited or nonexistent, the compelling evidence of potential harm to children's development calls for serious investment in the design and testing of new strategies for prevention and treatment that are grounded in sound scientific principles, subjected to rigorous evaluation, and improved continuously over time.

**Child welfare policies and programs that are mandated to assess and intervene in cases of suspected and/or confirmed abuse or neglect must address the full range of children's developmental needs, not just focus on their physical safety.** All states have established systems that require the reporting of suspected child maltreatment and the provision of protective services for children whose health or well-being

is threatened. These services focus largely on issues related to physical safety, reduction of repeated injury, and child custody. Advances in neuroscience now indicate that evaluations of maltreated children that rely solely on physical examination and screening for broken bones are insufficient and must be augmented by comprehensive developmental assessments and appropriate intervention by skilled professionals as needed. To this end, it is important to note that early intervention programs for children with developmental delays or disabilities have the expertise to provide many of the services needed by maltreated children, and these programs are already available in all states under a federal entitlement specified in the Individuals With Disabilities Education Act (IDEA). Moreover, the most recent reauthorizations of the relevant federal legislation for both the child welfare and early intervention systems (the Keeping Children and Families Safe Act and IDEA,

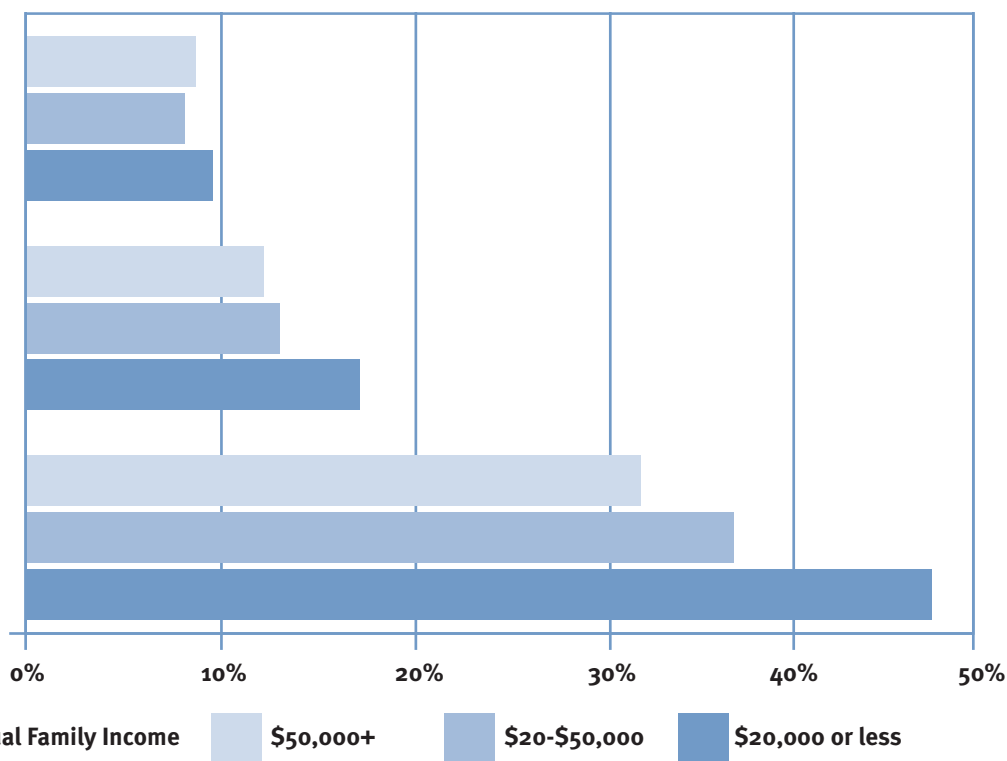
### Fear-Inducing Events Disproportionately Affect Children in Low-Income Environments

% of children  
ages 2-17 who...

experience sexual  
victimization

experience  
maltreatment

witness violence



Source: Finkelhor, et al. (2005)<sup>1</sup>

respectively) include requirements for regularized referrals of newly established child protective cases from the child welfare agency to the early intervention system for developmental screening. Notwithstanding its strong, science-based rationale, the implementation of this linkage has been limited to date and requires immediate attention. The evidence that significant “fear learning” with long-term consequences can occur as early as the first year of life—and that the capabilities for effective “fear unlearning” do not fully emerge until later—underscores the extent to which the limited child development expertise available within the nation’s child welfare system can no longer be justified.

**Early identification and treatment for anxiety and post-traumatic stress disorders in young children should be routinely available through existing services for families, as they can significantly affect the future mental and physical health of children.** Advances in neuroscience, behavioral and developmental studies, and clinical research have all converged to contribute to a shared understanding of both the reality of early childhood mental health impairment and the parameters of successful preventive intervention and effective treatments. Early in infancy and childhood, intervention and treatment should focus on programs that provide families with necessary services, supports, and expertise, while later in development, supports should be focused more on children themselves.

The critical importance of intervening early in the lives of young children who experience excessive fear and anxiety is evident in two domains: the need to relieve current suffering and the opportunity to prevent enduring impairment that can lead to a lifetime of poor mental and physical health, diminished economic productivity, and antisocial behavior. With these high stakes in mind, all of society

would benefit from a greater capacity to address the problem of excessive fear and anxiety in young children across a broad array of service systems, including health care, child welfare programs, school- and child care-based health services, and the foster care system, among others.

**Policies with a broad mandate to reduce poverty and neighborhood violence would likely have greater long-term impacts if they also included explicit and focused attention on the prevention of fear and anxiety overload in young children.** Children who live in violent communities have been shown to have more behavior problems, greater evidence of post-traumatic stress

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disorder, and increased physical symptoms such as headaches and stomachaches, as well as lower capacity for empathy and diminished self-esteem.<sup>57</sup> Programs focused on the reduction of poverty, domestic violence, substance abuse, and neighborhood violence are examples of the kinds of community-based services whose impacts could be enhanced by incorporating targeted interventions to explicitly address the emotional needs of young children living under these conditions. When delivered effectively, such interventions could have a multiplier effect into the next generation by reducing both the individual and societal costs of the negative developmental effects of persistent fear, including mental health impairments, antisocial behavior, physical disease, and violent crime.

## References

1. Finkelhor, D., Ormrod, R., Turner, H., & Hamby, S. L. (2005). The victimization of children and youth: A comprehensive, national survey. *Child Maltreatment*, 10(1), 5-25.
2. U.S. Department of Health and Human Services, Administration on Children, Youth, and Families. (2009). *Child maltreatment 2007*. Washington, DC: U.S. Government Printing Office.
3. Centers for Disease Control and Prevention (2009). *Understanding child maltreatment*. Retrieved from <http://www.cdc.gov/violenceprevention/pdf/CM-FactSheet-a.pdf>
4. Lewis, M. & Michalson, L. (1983). *Children's emotions and moods: Developmental theory and measurement*. New York: Plenum Press.
5. Nelson, C. A., & De Haan, M. (1996). Neural correlates of infants' visual responsiveness to facial expressions of emotion. *Developmental Psychobiology*, 29(7), 577-595.
6. Gunnar-von Gnechten, M. R. (1978). Changing a frightening toy into a pleasant toy by allowing the infant to control its actions. *Developmental Psychology*, 14, 157-162.
7. Phelps, E. A., & LeDoux, J. E. (2005). Contributions of the amygdala to emotion processing: From animal models to human behavior. *Neuron*, 48, 175-187.
8. Delgado, M. R., Olsson, A., & Phelps, E. A. (2006). Extending animal models of fear conditioning to humans. *Biological Psychology*, 23, 39-48.
9. LeDoux, J. E. (2000). Emotion circuits in the brain. *Annual Review of Neuroscience*, 23, 155-184.
10. LeDoux, J. E. & Phelps, E. A. (2008). Emotional networks in the brain. In M. Lewis, J. M. Haviland-Jones, & L. F. Barrett, (Eds.), *Handbook of emotions* (pp. 159-179). New York: Guilford Press.
11. Kim, J. J., & Fanselow, M. S. (1992). Modality-specific retrograde amnesia of fear. *Science*, 256, 675-677.
12. Arnsten, A. F. (2009). Stress signaling pathways that impair prefrontal cortex structure and function. *Nature Reviews Neuroscience*, 10, 410-422.
13. Watson, J. B., & Rayner, R. (1920). Conditioned emotional reactions. *Journal of Experimental Psychology*, 3(1), 1-14.
14. Pavlov, I. (1927). *Conditioned reflexes*. London: Oxford University Press.
15. Grillon, C., & Morgan, C. A. I. (1999). Fear-potentiated startle conditioning to explicit and contextual cues in Gulf War veterans with posttraumatic stress disorder. *Journal of Abnormal Psychology*, 108, 134-142.
16. Pine, D. S. (1999). Pathophysiology of childhood anxiety disorders. *Biological Psychiatry*, 46, 1555-1566.
17. Quirk, G. J., Garcia, R., & Gonzalez-Lima, F. (2006). Prefrontal mechanisms in extinction of conditioned fear. *Biological Psychiatry*, 60, 337-343.
18. Sotres-Bayon, F., Bush, D. E. A., & LeDoux, J. E. (2009). Emotional perseveration: An update on prefrontal-amygdala interactions in fear extinction. *Learning & Memory*, 11, 525-535.
19. Morgan, M. A., Romanski, L. M., & LeDoux, J. E. (1993). Extinction of emotional learning: Contribution of medial prefrontal cortex. *Neuroscience Letters*, 163, 109-113.
20. Phelps, E. A., Delgado, M., Nearing, K., & LeDoux, J. (2004). Extinction learning in humans: Role of the amygdala and vmPFC. *Neuron*, 43(6), 897-905.
21. Sullivan, R.M., Landers, M., Yeaman, B., & Wilson, D. A. (2000). Neurophysiology: Good memories of bad events in infancy. *Nature*, 407, 38-39.
22. Prather, M.D., Lavenex, P., Mauldin-Jourdain, M. L., Mason, W. A., Capitanio, J. P., Mendoza, S. P., & Amaral, D. G. (2001). Increased social fear and decreased fear of objects in monkeys with neonatal amygdala lesions. *Neuroscience*, 106(4), 653-658.
23. Rudy, J. W. (1993). Contextual conditioning and auditory cue conditioning dissociate during development. *Behavioral Neuroscience*, 107, 887-891.
24. Carew, M. B., & Rudy, J. W. (1991). Multiple functions of context during conditioning: A developmental analysis. *Developmental Psychobiology*, 24, 191-209.
25. Kim, J. H., & Richardson, R. (2008). The effect of temporary amygdala inactivation on extinction and reextinction of fear in the developing rat: Unlearning as a potential mechanism for extinction early in development. *Journal of Neuroscience*, 28, 1282-1290.
26. Sanchez, M. M., Ladd, C. O., & Plotsky, P. M. (2001). Early adverse experience as a developmental risk factor for later psychopathology: Evidence from rodent and primate models. *Development and Psychopathology*, 13(3), 419-449.
27. Nemeroff, C. B. (2004). Neurobiological consequences of childhood trauma. *Journal of Clinical Psychiatry*, 65(1), 18-28.
28. McEwen, B. S. (2007). Physiology and neurobiology of stress and adaptation: Central role of the brain. *Physiological Reviews*, 87, 873-904.
29. de Kloet, E. R., Karst, H., & Joëls, M. (2008). Corticosteroid hormones in the central stress response: Quick-and-slow. *Frontiers in Neuroendocrinology*, 29(2), 268-272.
30. Wiegert, O., Joels, M., & Krugers, H. J. (2008). Corticosteroid hormones, synaptic strength and emotional memories: Corticosteroid modulation of memory a cellular and molecular perspective. *Progress in Brain Research*, 167, 269-271.
31. Roozendaal, B., Barseggyan, A., & Lee, S. (2008). Adrenal stress hormones, amygdala activation, and memory for emotionally arousing experiences. *Progress in Brain Research*, 167, 79-97.
32. McGaugh, J. L., Cahill, L., & Roozendaal, B. (2006). Involvement of the amygdala in memory storage: Interaction with other brain systems. *Proceedings of the National Academy of Sciences USA*, 93, 13508-13514.
33. Yang, Y. L., Chao, P. K., Ro, L. S., Wo, Y. Y. P., & Lu, K. T. (2007). Glutamate NMDA receptors within the amygdala participate in the modulatory effect of glucocorticoids on extinction of conditioned fear in rats. *Neuropsychopharmacology*, 32, 1042-1051.
34. Brinks, V., de Kloet, E. R., & Oitzl, M. S. (2008). Strain specific fear behaviour and glucocorticoid response to aversive events: Modelling PTSD in mice. *Progress in Brain Research*, 167, 257-261.
35. Roozendaal, B., McEwen, B. S., & Chattarji, S. (2009). Stress, memory, and the amygdala. *Nature Reviews Neuroscience*, 10, 423-433.

36. Grillon, C. (2002). Startle reactivity and anxiety disorders: aversive conditioning, context, and neurobiology. *Biological Psychiatry*, 52, 958-975.
37. Davis, M. (2006). Neural systems involved in fear and anxiety measured with fear-potentiated startle. *American Psychologist*, 61(8), 741-756.
38. Lissek, S., Powers, A. S., McClure, E. B., Phelps, E. A., Woldehawariat, G., Grillon, C., & Pine, D. S. (2005). Classical fear conditioning in the anxiety disorders: A meta-analysis. *Behaviour Research & Therapy*, 43(11): p. 1391-1424.
39. Lissek, S., Biggs, A. L., Rabin, S. J., Cornwell, B. R., Alvarez, R. P., Pine, D.S., & Grillon, C. (2008). Generalization of conditioned fear-potentiated startle in humans: Experimental validation and clinical relevance. *Behaviour Research & Therapy*, 46(5), 678-687.
40. Grillon, C., Dierker, L., & Merikangas, K. R. (1998). Fear-potentiated startle in adolescent offspring of parents with anxiety disorders. *Biological Psychiatry*, 44, 990-997.
41. Reeb-Sutherland, B. C., Helfinstein, S. M., Degnan, K. A., Perez-Edgar, K., Henderson, H. A., Lissek, S., Chronis-Tuscano, A., Grillon, C., Pine, D. S., & Fox, N. A. (2009). Startle response in behaviorally inhibited adolescents with a lifetime occurrence of anxiety disorders. *Journal of the American Academy of Child and Adolescent Psychiatry*, 48(6), 610-617.
42. Wismer Fries, A. B., Ziegler, T. E., Kurian, J. R., Jacoris, S., & Pollak, S. D. (2005). Early experience in humans is associated with changes in neuro-peptides critical for regulating social behavior. *Proceedings of the National Academy of Sciences USA*, 102(47), 17237-17240.
43. Zeanah, C. H., Smyke, A. T., & Dumitrescu, A. (2002). Attachment disturbances in young children II: Indiscriminate behavior and institutional care. *Journal of the American Academy of Child and Adolescent Psychiatry*, 41(8), 983-989.
44. Zeanah, C. H., Smyke, A. T., & Koga, S. F. (2005). Attachment in institutionalized and community children in Romania. *Child Development*, 76(5), 1015-1028.
45. O'Connor, T. G., Rutter, M., & The English and Romanian Adoptees (ERA) Study Team (2000). Attachment disorder behavior following early severe deprivation: extension and longitudinal follow-up. *Journal of the American Academy of Child and Adolescent Psychiatry*, 39(6), 703-712.
46. O'Connor, T. G., Bredenkamp, D., Rutter, M., & The English and Romanian Adoptees (ERA) Study Team. (1999). Attachment disturbances and disorders in children exposed to early severe deprivation. *Infant Mental Health Journal*, 20(1), 10-29.
47. Pollak, S. D., Messner, M., Kistler, D. J., & Cohn, J. F. (2009). Development of perceptual expertise in emotion recognition. *Cognition*, 110(2), 242-247.
48. Pollak, S. D. & Kistler, D. J. (2002). Early experience is associated with the development of categorical representations for facial expressions of emotion. *Proceedings of the National Academy of the Sciences USA*, 99(13), 9072-9076.
49. Pollak, S. D., Cicchetti, D., Hornung, K., & Reed, A. (2000). Recognizing emotion in faces: Developmental effects of child abuse and neglect. *Developmental Psychology*, 36(5), 679-688.
50. Pollak, S. D., & Tolley-Schell, S. A. (2003). Selective attention to facial emotion in physically abused children. *Journal of Abnormal Psychology*, 112(3), 323-338.
51. Pollak, S. D. (2008). Mechanisms linking early experience and the emergence of emotions: illustrations from the study of maltreated children. *Current Directions in Psychological Science*, 17, 370-375.
52. National Scientific Council on the Developing Child. (2005). *Excessive stress disrupts the architecture of the developing brain* (Working Paper No. 3). Retrieved from <http://www.developingchild.net>
53. Shonkoff, J., Boyce, W. T., & McEwen, B. S. (2009). Neuroscience, molecular biology, and the childhood roots of health disparities: building a new framework for health promotion and disease prevention. *Journal of the American Medical Association* 301(21), 2252-2259.
54. MacMillan, H. L., Wathen, C. N., Barlow, J., Fergusson, D. M., Leventhal, J. M., & Taussig, H. N. (2009). Interventions to prevent child maltreatment and associated impairment. *Lancet*, 373(9659), 250-266.
55. Donelan-McCall, N., Eckenrode, J., & Olds, D. L. (2009). Home visiting for the prevention of child maltreatment: Lessons learned during the past 20 years. *Pediatric Clinics of North America*, 56, 389-403.
56. Poole, A., Beran, T., & Thurston, W. (2008). Direct and indirect services for children in domestic violence shelters. *Journal of Family Violence*, 23, 679-686.
57. Huth-Bocks, A. C., Levendosky, A. A., & Semel, M. A. (2001). The direct and indirect effects of domestic violence on young children's intellectual functioning. *Journal of Family Violence*, 16(3), 269-290.
58. Danese, A., Moffitt, T. E., Pariante, C. M., Ambler, A., Poulton, R., & Caspi, A. (2008). Elevated inflammation levels in depressed adults with a history of childhood maltreatment. *Arch Gen Psychiatry*, 65(4), 409-415.



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