DOMESTIC VIOLENCE, CHILDREN, AND TOXIC STRESS

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"It is easier to build strong children than to repair broken men."
Frederick Douglass

"The child is the father of the man."
William Wordsworth

I. INTRODUCTION

It is estimated that approximately 1.5 million women and 830,000 men experience physical or sexual assault annually in the United States by intimate partners, commonly called domestic violence.[1] In the United States, more than 15 million children live in families in which domestic violence occurs and almost half of these children witness severe violence in assaults of a parent.[2] As individuals and as a society, we need to face the reality that exposure of children to domestic violence undermines healthy physical and emotional development for the child, which places the child at increased risk of physical and emotional diseases and disabilities as adults.[3] The effects of domestic violence on children include social consequences, physical consequences, and behavioral or neuroendocrine consequences that may result in transient, long-lasting or permanent harm to the child.[4] The purpose of this discussion is to provide a medical background for the urgency for appropriate intervention in situations where domestic violence occurs in the presence of children.

II. OVERVIEW OF THE EFFECTS OF DOMESTIC VIOLENCE ON CHILDREN

The social consequences for children include those related to the violence directed at their mother and the common association of domestic violence and child abuse. Studies indicate that families with either child abuse or

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[1] Jonathan D. Thackeray et al., *Clinical Report–Intimate Partner Violence: The Role of the Pediatrician*, 125 PEDIATRICS 1094, 1094-95 (2010), http://pediatrics.aappublications.org/content/125/5/1094.

[2] Megan Bair-Merritt et al., *Silent Victims–An Epidemic of Childhood Exposure to Domestic Violence*, 369 NEW ENG. MED. 1673, 1673 (2010), http://www.nejm.org/doi/full/10.1056/NEJMp1307643.

[3] Thackeray, *supra* note 1, at 1095; Bair-Merritt, *supra* note 2, at 1674.

[4] Thackeray, *supra* note 1, at 1095.

domestic violence have a 30-60% chance of having both types of violence occurring in the home.[5] When the abuser, which oftentimes is the father or father figure, is removed from the household, another abusive father figure unfortunately may step in, which in turn leads to severe family disruption. Legal and social interventions may result in the removal of the abuser from the home or the removal of the children from the home for variable periods of time.

The physical consequences of domestic violence include direct injury to the child associated with a domestic violence incident and indirect injury from the victim of domestic violence being unable to provide a safe, nurturing environment for the child. Direct injury may involve the fetus.[6] Pregnancy may increase the risk of domestic violence for women and domestic violence in this setting is associated with prematurity, low birth weight, and perinatal death.[7] More commonly, we see direct injury to the child who is present with their mother during a domestic violence episode.[8] Infants may be injured when the mother holds the infant in her arms as the abuser physically attacks her. The mothers may attempt to provide protection for the infants by holding them, or may think they can use the infant as a type of "shield" to stop the abusers physical assault. In either case, the child may suffer physical injuries. Some younger children may simply be unable to get out of the way and are struck by physical blows or thrown objects during the domestic conflict. Older children and adolescents may try to intervene and are injured while trying to prevent injury to one of both of the adults. Any child in a violent environment may be injured as a proxy to the adult "victim" as the abusive adult tries to control the victim by hurting the child, which threatens to produce even greater injury to the child if the victim resists the abuser's control.[9]

The behavioral and neuroendocrine consequences are the most significant consequences because these include generally poor social, emotional, and developmental growth of the child with associated poor overall physical and mental health during adulthood.[10] These consequences in infants may be manifested as increased irritability, increased emotional arousal, and less responsiveness to adults.[11] In the school-aged child, the effects are often manifested as anxiety, depression, attention problems, aggression, and withdrawal.[12] Adolescents may exhibit similar symptoms as school-aged children, and they may show bullying and risk-taking behavior and experience dating violence.[13]

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[5] Thackeray, *supra* note 1, at 1095.
[6] *Id.*
[7] *Id*.
[8] *Id*.
[9] *Id.*; *see also* Bair-Merritt, *supra* note 2, at 1673-74.
[10] Thackeray, *supra* note 1, at 1095.
[11] *See id*.
[12] *Id.*
[13] *Id.*

III. THE LIFELONG CONSEQUENCES OF CHILDHOOD TRAUMA AND ADVERSITY

Over the last two decades we have learned a lot about the effects of trauma on children. This knowledge includes the effects seen in the developing child and subsequent, life-long effects found among adults who were subjected to abuse, neglect and domestic violence as children. Trauma is cumulative and dose-related – the greater the number of episodes of trauma experienced over time, the worse the effect.[14] Early childhood trauma appears to have the greatest impact.[15] Trauma early in life affects the child's development of attachment, emotional regulation, and impulse control.[16] Traumatized children act differently than non-traumatized children.[17] Traumatized children have lower social competence; they have less empathy for others, can't recognize their own emotions, and can't recognize other's emotions, resulting in impaired interpersonal relationships.[18] The cumulative dose-related effect of childhood trauma can last a lifetime. Traumatized children are at increased risk of adverse health effects including physical and psychological health.[19]

Generally, we have learned that childhood adversity has lifelong consequences, and specifically, that adversity is strongly associated with unhealthy adult lifestyles and poor physical and mental health years later.[20] In the mid-1990s, Drs. Felitti and Anda began a study of more than 17,000 adults, largely considered to be of middle class, who received heath care through the Kaiser Permanente Health System. These adults clearly demonstrated that Adverse Childhood Experiences (ACEs) can contribute significantly to negative adult physical and mental health outcomes.[21] In their initial and subsequent studies, their list of adverse childhood events included: physical abuse, sexual abuse, emotional abuse, physical neglect, emotional neglect, domestic violence in home, household substance abuse, household mental illness, parental separation or divorce, and incarcerated household member.[22] They found ACEs are common, with approximately

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[14] *See* John Stirling, Jr. et al., *Understanding the Behavioral and Emotional Consequences of Child Abuse,* 122 PEDIATRICS 667, 668 (2008), http://pediatrics.aappublications.org/content/pediatrics/122/3/667.full.pdf.

[15] *See id*. at 669-70.

[16] *Id*.

[17] *Id*.

[18] *Id*.

[19] Vincent J. Felitti et al., *Relationships of Childhood Abuse and Household Dysfunction to Many of the Leading Causes of Death in Adults: The Adverse Childhood Experiences (ACE) Study,* 15 AM. J. PREVENTATIVE MED. 245, 251 (1998), http://www.ajpmonline.org/article/S0749-3797(98)00017-8/pdf.

[20] *Id*.

[21] *Id*. at 245.

[22] Shanta R. Dube et al., *Childhood Abuse, Household Dysfunction, and the Risk of Attempted Suicide Throughout the Life Span: Findings from the Adverse Childhood Experience Study,* 286 JAMA PEDIATRICS 3089, 3090 (2001), http://jama.jamanetwork.com/article.aspx?articleid=194504.

two-thirds of their patients had at least one ACE and one-fifth reported three or more ACEs.[23] Adults who reported exposure to domestic violence during childhood were 6 times more likely to also experience emotional abuse, 4.8 times more likely to also experience physical abuse, and 2.6 times more likely to also experience sexual abuse.[24] As the investigators reviewed the health of their patients, they found the effects of the ACES were "dose-related"; the more ACEs the individual reported the worse were their physical and mental health outcomes.[25] A number of the poor outcomes appeared related to increased risk taking behaviors demonstrated by patients with ACEs, including substance abuse, alcoholism, smoking, and risky sexual behaviors.[26] Of particular importance was the association of higher ACE scores with increased risk of being either a victim or perpetrator of interpersonal violence.[27] Less intuitive were negative health outcomes that appear to be less directly mediated through health risk behaviors including chronic heart disease, liver disease and autoimmune disorders.[28] Over the course of the last two decades, it has become clear that the negative health outcomes associated with ACEs are mediated through a combination of increased heath risk behaviors, the effects of chronic stress on the brain, the endocrine system, the immune system and the function of genes.[29] Adverse childhood experiences are associated with poorer physical and mental health as adults. Thus, in order to produce healthier adults, we need to make healthier children, and to do that we need to address the role of chronic stress.

IV. THE ROLE OF CHRONIC STRESS OR TOXIC STRESS

It is important to recognize that not all stress is bad. There are three categories of stress experience: *positive stress*, *tolerable stress*, and *toxic stress*.[30] *Positive stress* is involved in many common experiences and

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[23] Dube, *supra* note 22, at 3092.

[24] Thackeray, *supra* note 1, at 1095.

[25] Felitti, *supra* note 19, at 250.

[26] Robert F. Anda et al., *The Enduring Effects of Abuse and Related Adverse Experiences in Childhood: A Convergence of Evidence From Neurobiology and Epidemiology*, 256 EUR. ARCHIVES OF PSYCHIATRY & CLINICAL NEUROSCIENCE 174, 181 (2006), http://link.springer.com/article/10.1007/s00406-005-0624-4.

[27] *Id.* at 178-79.

[28] Felitti, *supra* note 19, at 251.

[29] Comm. on Psychosocial Aspects of the Child and Family Health et al., *Early Childhood Adversity, Toxic Stress and the Role of the Pediatrician: Translating Developmental Science Into Lifelong Health*, 129 PEDIATRICS e224, e225 (2011), http://pediatrics.aappublications.org/content/early/2011/12/21/peds.2011-2662; Jack P. Shonkoff et al., *The Lifelong Effects of Early Childhood Adversity and Toxic Stress*, 129 PEDIATRICS 1, e235-e237 (2012); Sara B. Johnson et al., *The Science of Early Life Toxic Stress for Pediatric and Advocacy*, 131 PEDIATRICS 319, 325 (2013), http://pediatrics.aappublications.org/content/131/2/319; Andrew S. Garner, *Home Visiting and the Biology Of Toxic Stress: Opportunities to Address Early Childhood Adversity*, 132 PEDIATRICS S65, S69 (2013), http://pediatrics.aappublications.org/content/132/Supplement_2/S65.

[30] Shonkoff, *supra* note 29, at e325.

interactions.[31] The body's response to positive stress is a brief increase in heart rate and mild transient elevation of stress hormone level.[32] These responses tend to increase our focus or concentration and improve performance, and in certain cases, they can be protective from physical trauma.[33] *Tolerable stress* involves more serious and more prolonged activation of stress hormones in response to a stressful situation, but the effects of the stress are buffered by supportive relationships with family, friends or other contacts.[34] *Toxic stress* involves prolonged activation of the stress response systems in the absence of protective relationships.[35] Prolonged elevation of the stress response can result in changes to the brain and other organs, and the ability of the body to regulate the response to stress.[36] Toxic stress in children who experience traumatic events is often called child traumatic stress.[37]

So how do we define child traumatic stress? Child traumatic stress is "the physical and emotional responses of a child to events that threaten the life or physical integrity of the child or of someone critically important to the child (such as a parent or sibling)."[38] These traumatic events overwhelm a child's capacity to cope and elicit feelings of terror, powerlessness, and out-of-control physiological arousal of the body's stress response.[39] The effects or impact of the trauma are dependent on factors related to the child, the family and the situation. Traumatic stress may be an acute response to a single traumatic event (natural disasters, illness, accident) or a chronic response to recurring or cumulative traumatic events (war, abuse, neglect).[40] A critical factor for the developing child is that events that threaten their safety or are frightening can lead to changes in the way the body reacts initially, and these changes may persist.[41] The critical issue regarding traumatic stress is whether it produces a longstanding physiologic change in the individual.[42]

So to summarize:

1. Three interrelated categories of ACEs – child abuse (physical, sexual, and emotional), neglect (physical and emotional), and exposure to domestic violence/interpersonal violence – are sources of traumatic stress for children.[43]

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[31] Shonkoff, *supra* note 29, at e325.
[32] *Id.*
[33] *Id.*
[34] *Id.*
[35] *Id.* at e235-36.
[36] *Id.* at e236.
[37] *See* AM. ACAD. OF PEDIATRICS, HELPING FOSTER ADOPTIVE FAMILIES COPE WITH TRAUMA 4 (2013), https://www.aap.org/en-us/advocacy-and-policy/aap-health-initiatives/healthy-foster-care-america/Documents/Guide.pdf.
[38] *Id.*
[39] *Id.*
[40] *Id.*
[41] Shonkoff, *supra* note 29, at e326.
[42] *See id.* at e237-e238.
[43] *Id.* at e326.

2. Traumatic stress becomes toxic stress when it makes persisting changes in the way the body responds to stress.[44]
3. Children are more susceptible to these changes, and these changes play a role in physical and mental health outcomes later in life.[45]
4. Toxic stress produces changes in multiple body systems. Major changes can be found in social/behavioral functions, the neuroendocrine system (brain structure and stress hormone regulation), and in molecular function (genetics, epigenetics, and immune function). These systems are inter-related and changes in one system can result in changes in another. Resilience, or the ability to recover from a stress, may not be the same across all systems.[46]

A. Effects of Toxic Stress on the Social/Behavioral System

The social/ behavioral systems are often the most easily detected changes. The three major behavioral goals of child development are *attachment, regulation, and cognition*.[47] *Attachment* is the ability to develop a relationship with at least one individual.[48] This primary attachment opens the door to establishing relationships with others. *Regulation* requires learning to effectively control your feelings.[49] *Cognition* is required to allow for the development of an informational base and to use it for decision making and problem solving.[50]

Impaired attachment to the parents or caretaker during early childhood promotes poor connection to, relationships with, and distrust of all adults.[51] Early childhood development involves back and forth exchanges between parent and child, each adjusting their responses to the other. Regulation of exchanges is impaired by lack of normal parent-child interactions, and in the face of persistent fear or alert states induced by toxic stress, the children develop less empathy for others and have difficulty in recognizing their own and other's emotions.[52] Cognition is impaired, as toxic stress results in the child having a poor working memory, an inability to control impulsive behavior, and poor cognitive flexibility, causing impaired organizational skills.[53] When something frightening happens, the brain makes certain you do not forget it, and we remember these events in a special way. We remember the pattern of sensations involving the sights, the sounds, the

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[44] Shonkoff, *supra* note 29, at e238.

[45] Garner, *supra* note 29, at s68-69.

[46] AM. ACAD. OF PEDIATRICS, *supra* note 37, at 2-3.

[47] Kristine Jentoft Kinniburgh et al., *Attachment, Self-Regulation, and Competency*, 35 PSYCHIATRIC ANNALS 424, 429.

[48] *Id.*

[49] *Id.*

[50] *Id.*

[51] Stirling, *supra* note 14, at 669-70.

[52] *Id.*

[53] CHILD WELFARE INFO. GATEWAY, *Understanding the Effect of Maltreatment on Brain Development* 6-8 (2015), http://www.childwelfare.gov/pubs/issue-briefs/brain-development/.

smells and our feelings all mixed together.[54] Exposure to any one of these "trigger" sensations can make the child feel like the frightening experience is happening again. Common behaviors seen in children living with toxic stress include: "hair trigger" emotional responses; hyper-vigilance; frequent aggression; poor impulse control; difficulty regulating their arousal; reluctance to turn to others for help (distrust); inability to discuss their feelings; detachment; withdrawal; and insecurity over food, safety; or relationships and therefore are very hard to parent.[55] Furthermore, these children are labeled "behavior problems" and may end up being social pariahs and difficult to educate.[56]

B. Effects of Toxic Stress on the Brain and Neuroendocrine System

The behaviors observed in children exposed to toxic stress originate from the changes induced in the structure and function of the brain and the neuroendocrine system.[57] To understand how this happens, we must first consider how the brain normally grows. Brain growth is very rapid during the first two years of life, achieving 75-80% of its adult weight by age two, and reaches 90-95% of its adult weight by age eight.[58] The plasticity of the brain, the ability of the brain to change structurally in response to experience, is greatest during the first two years of life.[59] At birth, the brain contains most of the neurons or "nerve cells" it will ever contain.[60] Neurons typically "grow" by increasing the connections between neurons, adding additional axons and dendrites.[61] These axons and dendrites produce a type of "wiring" that links one cell to one or more additional cells allowing communication between the cells.[62] Stimulated connections grow, unused connections are cut off.[63] Other types of cells add to the growing brain. Specialized cells that act as "insulation" between the "wires" (axons and dendrites) increase in number through a process called myelination.[64] Increasing number of glial cells provide a framework or structural support for neurons and blood vessels grow in number and length, forming supply lines to deliver oxygen and nutrients to the neurons.[65]

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[54] Stirling, *supra* note 14, at 668.

[55] *Id*.

[56] *Id.* at 669.

[57] Shonkoff, *supra* note 29, at e326.

[58] Rhosel K. Lenroot & Jay N. Giedd, *Brain Development in Children and Adolescents: Insights from Anatomical Imaging*, 30 NEUROSCIENCE & BEHAV. REVS. 718, 720 (2006).

[59] CHILD WELFARE INFO. GATEWAY, *supra* note 53, at 2-5.

[60] Danya Glaser, *The Effects of Child Maltreatment on the Developing Brain*, 82 MED.-LEGAL J. 97, 100 (2014).

[61] *Id.*

[62] *Id.*

[63] *Id.*

[64] Lenroot & Giedd, *supra* note 58, at 719.

[65] *Id*.

Childhood traumatic stress alters the process of brain growth and development.[66] The effects are greatest during early childhood, but development continues throughout adolescence and into the early adult years.[67] Both genders experience these changes, but the effects of trauma on the brain appear to be greater for boys than girls.[68] Traumatized children have smaller brains for their age and body size than children who have not experienced trauma.[69] Some brain regions reduced in size/activity (anterior cingulate, corpus callosum, prefrontal cortex), while others increased in activity (dorsolateral cortex, amygdala).[70]

Although childhood traumatic stress can affect many regions of the brain, the primary effects are in structures whose function is to regulate emotion.[71] The orbital prefrontal cortex and dorsolateral cortex are both involved in planning and judging the value of actions.[72] Traumatic stress produces decreased activity of the orbital prefrontal cortex and increased activity of the dorsolateral cortex, resulting in defective attention, planning and judgment.[73] The anterior cingulate is involved in making quick judgments, morality, empathy for others, and impulse control.[74] Traumatic stress produces decreased activity of the anterior cingulate, resulting in poor impulse control and lack of empathy.[75] The corpus callosum allows for transfer of information regarding emotions between the right and left sides of the brain.[76] Traumatic stress decreases activity within the corpus callosum and this correlates with symptoms associated with post-traumatic stress disorder.[77] Increased activity within the amygdala, the fear and rage center in the brain, occurs due to traumatic stress and is associated with poor emotional control.[78] The amygdala, in cooperation with the hypothalamus, the pituitary gland, and the adrenal glands, is responsible for what most people recognize as a "stress response."[79]

We recognize the stress response as certain changes in body sensations, including tensing of muscles, dilated pupils, increased sweating, decreased saliva production, increased heart and respiration rate, and unusual sensations in the abdomen.[80] This stress response prepares the body to respond to a

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[66] Shonkoff, *supra* note 29, at e236.
[67] *Id.*
[68] Felitti, supra note 19, at 251.
[69] Garner, *supra* note 29, at S69.
[70] *Id.*
[71] CHILD WELFARE INFO. GATEWAY, *supra* note 53, at 5-6.
[72] *Id.*
[73] *Id.*
[74] *Id.*
[75] *Id.*
[76] *Id.* at 6.
[77] CHILD WELFARE INFO. GATEWAY, *supra* note 53, at 6.
[78] *Id.*
[79] Michael D. De Bellis, *The Psychobiology of Neglect*, 10 CHILD MALTREATMENT 150, 155 (2005).
[80] *Id.*

threat and to be ready for fight or flight.[81] The relationship of the amygdala to the hypothalamic-pituitary-adrenal axis (HPA axis) is complex, but essentially, a sensory message is sent to the amygdala for interpretation.[82] When the amygdala recognizes the message as a threat, it sends the message along to the hypothalamus, which in turn sends a message to the pituitary and adrenal glands.[83] The principal response of the adrenal glands is the release of cortisol, the primary stress hormone, which prepares the body to encounter (fight) or to get away from (flight) the situation causing the stress.[84] The secondary stress hormone epinephrine, or adrenalin, is also released.[85] Once the stress is lessened, the high level of cortisol in the blood feeds back to the pituitary and the hypothalamus and the further release of cortisol is stopped.[86] In an acute stress situation, the response turns on as needed and turns off when no longer needed.[87] In an acute situation, if you encounter a bear in the woods, the bear runs away or you run away, and the response turns off after you feel safe. By contrast, in the chronic stress situation, the response does not turn off—the bear is always there, and the system runs out of control.[88]

C. Effects of Toxic Stress at the Molecular and Genetic Level

Childhood traumatic stress also makes alterations at the molecular and genetic levels.[89] Genes contain DNA coded "instructions" for building proteins and directing the function of individual cells and organs, as well as total body function.[90] The DNA instructions are read or "transcribed" in cells.[91] Although each cell in the body contains the whole genome (complete set of instructions), individual cells may repeatedly use relatively few "pages" of instructions that relate to the specific function of that cell in the body.[92] Naturally occurring gene variations (alleles) affect our risk of developing certain diseases, but they also influence how we respond to adverse events.[93] Genes are our biological pre-programming, representing our "Nature" and the function of the genes are affected by the environment (Nurture).[94] The expanding science of epigenetics defines and studies the effect of the

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[81] De Bellis, *supra* note 79, at 155.
[82] *Id.*
[83] *Id.*
[84] *Id.*
[85] Shonkoff, *supra* note 29, at e326.
[86] *See id.*
[87] *Id.* at e235-36.
[88] *Id.*
[89] Garner, *supra* note 29, at S68.
[90] *See* NAT'L INST. OF GEN. MED. SCI., U.S. DEPT. OF HEALTH & HUMAN SERVS., NIH PUB. NO.10 - 662, THE NEW GENETICS 4 (2010).
[91] *Id.*
[92] *Id.*
[93] Garner, *supra* note 29, at S67.
[94] *Id.*

environment on gene function.[95] Gene expression or gene function can be modified by experience without changing the actual DNA in the gene.[96] Transcription, or the reading of specific genes, can be "turned off" by attachment of methyl groups (methylation) to the gene.[97] Transcription can also be blocked by the folding of genes around molecular structures known as histones.[98] The good news is that these modifications allow cells to differentiate (change in function) and allow genes themselves to "learn" how to function in response to certain environmental changes.[99] The bad news is that this process may lead to unwanted learning or unwanted functional changes.

Epigenetic changes can affect many different types of environmental stimuli and can affect genes controlling a number of different body systems.[100] Several animal studies have demonstrated that the interactions of the parents with offspring early in life can produce changes related to stress hormone regulation, resulting in dysregulation of the HPA axis.[101] In his study, Dr. Meaney intentionally traumatized a group of mother rats and observed that the traumatized mother rats groomed their pups differently when compared with non-traumatized mother rats.[102] Their pups (baby rats) grew up anxious and generally hyperresponsive to stimuli, acting as if they were responding to chronic stress.[103] Different methylation patterns were found on the corticotropin receptor gene in these pups.[104] This gene is involved in stress hormone regulation, specifically in the recognition that cortisol levels are elevated.[105] When cortisol levels are elevated and the elevation cannot be recognized, the message is sent to release even more cortisol, resulting in persistent elevation of cortisol.[106] The behaviors of the pups were caused by methylation uncoupling the normal negative feedback, leading to a persistence of the stress response.[107]

Dr. Murgatroyd, in another study, repeatedly separated baby mice from their mothers, and this resulted in similar behaviors in the baby mice caused by sustained HPA axis activity associated with methylation abnormalities of

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[95] Bao-Zhu Zang et al., *Child Abuse and Epigenetic Mechanisms of Disease Risk,* 44 AM. J. PREVENTATIVE MED. 101, 101 (2013).

[96] Sarah E. Romens et al., *Associations Between Early Life, Stress and Gene Methylation in Children,* 86 CHILD DEV. 303, 303 (2015).

[97] *See id.*

[98] *See* Garner, *supra* note 29, at S68.

[99] *See id.*

[100] *See* Romens et. al., *supra* note 96, at 303.

[101] Johnson et. al., *supra* note 29, at 323.

[102] Michael J Meaney & Moshe Szyf, *Environmental Programming of Stress Responses Through DNA Methylation: Life at the Interface Between a Dynamic Environment and a Fixed Genome,* 7 DIALOGUES CLINICAL NEUROSCIENCE. 103, 106 (2005).

[103] *Id.*

[104] *See id.*

[105] *See id.*

[106] *Id.*

[107] *See id*. at 116.

another regulatory gene involved in the stress response.[108] In a third study, Drs. Brunton, Donadio, and Russell intentionally stressed pregnant female mice during late gestation.[109] The male offspring grew up to be more anxious as adults than the sons of non-stressed mothers.[110] This study demonstrates that the stress of the mother during fetal development permanently altered the methylation of the corticotropin receptor gene in these offspring, resulting in sustained hyperactivity of the stress response.[111] In the first study, the mothers' stress behaviors caused the offspring to undergo epigenetic alteration to their environment during early life.[112] In the second study, stress of offspring removed from their mother's care resulted in similar epigenetic changes.[113] In the third study, prenatal stress in the mother resulted in permanent epigenetic alteration of the stress hormone response in male offspring.[114] Therefore, through epigenetic "learning," stresses in a parent or the offspring during early life may result in uncontrolled release of stress hormones in the offspring. Stresses during pregnancy may also result in similar epigenetic changes in male offspring.

To bring this back to the topic of domestic violence, domestic violence toward a mother with young offspring, affecting her ability to raise her offspring, and domestic violence directed towards a pregnant mother may result in uncontrolled stress response in the offspring. In humans, several studies have documented similar effects on stress hormone regulation due to lack of appropriate caregiving among children raised in institutional care.[115] One study looked at suicides in humans exposed to child abuse and demonstrated methylation of another gene involved in the regulation of the stress response.[116] This gene also plays a part in turning off the production of cortisol when blood levels are elevated, resulting in persisting, uncontrolled stress response.[117] Early care-giving also plays a pivotal role in the maturation of immunity, and the HPA axis is important to the immune response.[118] Cytokines are chemical messengers that play an important role in immunity, and proinflammatory cytokines activate the HPA axis.[119] Cortisol produced in response to HPA axis activation helps turn off the

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[108] Chris Murgatroyd et al., *Dynamic DNA Methylation Programs Persistent Adverse Effects on Early-Life Stress*, 12 NATURE NEUROSCIENCE. 1559, 1559 (2009).

[109] Paula J. Brunton et al., *Sex Differences in Prenatally Programmed Anxiety Behavior in Rats: Differential Corticotropin-Releasing Hormone Receptor mRNA Expression in the Amygdaloid Complex*, 14 STRESS 634, 635 (2011).

[110] *Id.* at 638.

[111] *Id.* at 635.

[112] *See* Meaney & Szyf, *supra* note 102, at 106.

[113] Murgatroyd, *supra* note 108, at 1565.

[114] Brunton, *supra* note 109, at 638.

[115] Johnson, *supra* note 29, at 323.

[116] *See* PATRICK O. MCGOWAN ET AL., PROMOTER-WIDE HYPERMETHYLATION OF THE RIBOSOMAL RNA GENE PROMOTER IN THE SUICIDE BRAIN 5 (PLoS One, 2008).

[117] *Id.*; *see also* Meaney & Szyf, *supra* note 102, at 106.

[118] Johnson, *supra* note 29, at 322-23.

[119] *Id.*

immune response.[120] Toxic stress produces dysregulation of the HPA axis, and in turn this produces broad effects on the immune system and inflammatory processes related to immunity, including suppression of normal immune responses and persistence of the inflammatory response after it is no longer needed.[121]

V. INTERVENTION FOR CHILDREN EXPOSED TO DOMESTIC VIOLENCE

Children exposed to domestic violence, frequently experiencing concurrent child abuse and neglect, are caught in a vicious cycle associated with toxic stress. The toxic stress occurs while the child is experiencing the episode of violence itself, in the period between the cycles of violence, and persists long after the last episode of violence, affecting their future physical and mental health. Children experiencing toxic stress have major neuroendocrine, epigenetic, and behavioral changes which may persist long after the violent exposures end. In order to create appropriate interventions, we must recognize the role of toxic stress and the additional adverse childhood experiences which are active in both the children and the adults experiencing domestic violence, and the frequently co-occurring abuse and neglect. The parenting skills of both abusive and non-abusive parents in the domestic violence relationship are impaired and affect their ability to be proper parents. Social agencies and family courts must recognize the implications of the effects of toxic stress and the impaired parenting skills and implications for case management, child custody, and foster care needs of these children.[122] Children who experience toxic stress are more difficult to parent than typical children because of their altered behaviors related to neuroendocrine and epigenetic changes.[123]

Unfortunately, some of the damage produced by toxic stress may be permanent and prevention programs are essential to break the cycle.[124] Fortunately, children are remarkably resilient, and reversal or at least control of many of the changes is possible, but earlier intervention and trauma-focused intervention are important to accomplish this "repair." Both the caretakers and those responsible for behavioral and social interventions need to recognize that behavioral and social interventions are needed to begin to reverse the effects of domestic violence. We need to recognize that children can only be treated by proxy. Medications for symptom control and counseling a couple of times a month for the child are not adequate. The children require a trauma-informed and trauma-focused therapy. Caregivers for the children must be involved, safe, and supportive; otherwise interventions are likely to fail. Ensuring that every child has a caretaker who has the skills and support to be a safe, stable, and nurturing source can foster

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[120] Johnson, *supra* note 29, at 322-23.
[121] *Id.* at 321-22.
[122] *See generally* Shonkoff, *supra* note 29, at e228;
[123] Stirling, *supra* note 14, at 668-70; *see* AM. ACAD. OF PEDIATRICS, *supra* note 37, at 11.
[124] *See generally* Johnson, *supra* note 29.

resilience and help transform toxic stress into tolerable stress.[125] The longer we delay the onset of effective intervention, the more difficult the task becomes for society in managing the problems of the children and the adults they will become.

We are guilty of many errors and many faults,
but our worst crime is
abandoning the children,
neglecting the fountain of life.
Many of the things we need can wait.
The child cannot.
Right now is the time
his bones are being formed,
his blood is being made
and his senses are being developed.
To him we cannot answer 'Tomorrow'.
His name is Today.[126]

[125] Johnson, *supra* note 29.

[126] Gabriela Mistral, *His Name is Today*, Chilean educator, diplomat, and poet, Nobel Laureate 1945, reprinted in LEONARD DAVIS, CHILDREN OF THE EAST xv (Janus Pub. Co. 1994).