Biology, childhood trauma, and murder: Rethinking justice

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Abstract

This article reviews recent findings in the developmental neurophysiology of children subjected to psychological trauma. Studies link extreme neglect and abuse with long-term changes in the nervous and endocrine systems. A growing body of research literature indicates that individuals with severe trauma histories are at higher risk of behaving violently than those without such histories. This article links these two research areas by discussing how severe and protracted child abuse and/or neglect can lead to biological changes, putting these individuals at greater risk for committing homicide and other forms of violence than those without child maltreatment histories. The implications of these biological findings for forensic evaluations are discussed. Based on new understanding of the effects of child maltreatment, the authors invite law and mental health professionals to rethink their notions of justice and offender accountability, and they challenge policymakers to allocate funds for research into effective treatment and for service delivery.

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1. Introduction

For centuries health and legal professionals have debated the motivational dynamics that propel individuals to kill. Since the 1800s, explanations for murder have fortunately moved beyond demonic possession to include a variety of psychological theories, including psychoanalytic, behavioural, existential, and attachment. Attention has also focused on sociological and economic theories that examine environments and circumstances that enhance criminal and violent offending.

Advances in science, particularly during the past 10 to 20 years, have led to biological investigations into the underpinnings of violent behaviour. Early investigations of homicide offenders looked at those referred for psychiatric and neurological evaluations. In a pioneering study, Raine et al. (1994) used positron emission tomography (PET) brain
imaging to investigate dysfunction of the prefrontal cortex in murderers pleading not guilty by reason of insanity or incompetent with a group of matched controls. Their results suggested that deficits localized in the prefrontal cortex may be associated with violent behaviour in a particular group of offenders. Subsequently, Raine, Buchsbaum, and LaCasse (1997) identified dysfunction in several areas of the brain in murderers pleading not guilty by reason of insanity. They suggested that “a network of abnormal cortical and subcortical brain processes” may increase the risk of violence.

Researchers in another study reported a high rate of organic impairment among murderers referred by the court for competency and criminal responsibility evaluations (Frierson, Schwartz-Watts, Morgan, & Malone, 1998). In a study of murderers referred by their attorneys for a neurological examination, neuropsychological testing revealed abnormalities in all subjects. The authors reported that there was a confirmed history of profound and enduring physical abuse in 26 of these 31 cases. They concluded that prolonged and severe physical abuse likely interacts with neurological brain dysfunction and contributes to violent behaviour (Blake, Pincus, and Buckner, 1995).

Scientific evidence showing the important role played by biology in human behaviour has had some impact on law and public policy in the United States. The United States Supreme Court abolished the death penalty for juvenile offenders in March 2005. Research findings indicating that the brain does not complete its development until post-adolescence influenced the Court’s ruling. The Court cited medical and social-science evidence that adolescents are not mature enough to be held accountable for their crimes to the same extent as adults. At the time of this ruling 20 states still permitted capital punishment for offenders younger than age 18 (Roper v. Simmons 543 U.S.).

Recent research findings provide convincing evidence that the effects of normal biologically based immaturity on adolescent’s thinking and behaviour are further compromised by child maltreatment. Severe abuse and/or neglect, particularly during early childhood, is a form of Type III trauma, extreme trauma characterized by multiple traumatic experiences that typically begin at an early age and may be perceived as life-threatening (Solomon & Heide, 1999). Young children who are maltreated form insecure attachments with their caretakers leading to developmental disruptions that can have enduring consequences.

Traumatic stress caused by child neglect and/or abuse compromises homeostasis and leads to a constellation of long-term biological changes involving the nervous and endocrine systems. These changes affect physiological, emotional, cognitive, and social function, including the ability to regulate, affect, relate to other people, and develop empathy. When confronted with stressful situations, Type III trauma survivors often have difficulty accessing higher cortical centers, the areas of the brain essential for thinking logically and formulating appropriate decisions. Instead, their responses are driven by limbic and brain stem activity, often resulting in socially inappropriate behaviour. This primitive response mode results in a variety of problems including difficulty regulating affective impulses and inappropriate expression of anger.

We are just beginning to understand the complex biological effects of traumatic stress associated with neglect and/or abuse that can contribute to adolescents and adults behaving violently. At the same time, there has been a renewed interest among criminologists in the psychological, neuropsychological, and physiological correlates of serious antisocial behaviour by adolescents. Findings from a recent study revealed that neurophysiological and autonomic factors distinguished delinquents who engaged in serious offending from those who engaged in acts of minor delinquency (Cauffman, Steinberg, & Piquero, 2005). Researchers who maintain that serious and persistent offending is associated with inherited or acquired neurophysiological liabilities recognize that the environment in which the child is raised often exacerbates the child’s biological vulnerabilities. Child abuse, poor and inadequate parenting, and disrupted family bonds can clearly contribute to a child’s biological liabilities and augment their effects (Lewis et al., 1985; Lewis, Lovely et al., 1988; Lewis, Pincus et al., 1988; Lewis, Yeager, Gidlow, & Lewis, 2001; Moffitt, 2003). In this article the authors examine the biological effects of child maltreatment and how these changes can affect individuals, particularly when they are operating under stress. The implications for trauma survivors who have killed and faced charges in the criminal justice system are discussed.

2. Complex PTSD

Child maltreatment can lead to Post-traumatic Stress Disorder (PTSD). Three defining indicators of PTSD are (1) intrusive thoughts, images, sensory experiences, memories, and dreams; (2) avoidance of reminders of the traumatic event and/or emotional numbing; and (3) physiological hyperarousal (American Psychiatric Association, 2000). Individuals with PTSD are always on alert, ready to respond to real or perceived danger. During a traumatic experience, individuals are typically overwhelmed by their pain and by intense feelings, and may feel powerless and helpless.
When children experience ongoing abuse, they feel helpless and alone. They may learn to dissociate from their experience. Later, they try to avoid any reminders of the experience.

Long-term childhood maltreatment can lead to complex PTSD, a chronic form of PTSD with an additional constellation of symptoms. In complex PTSD, trauma survivors also commonly experience poor self-concept, depression, dissociation, and difficulty with affective regulation. They tend to be impulsive, aggressive toward themselves or others, and suffer from chronic feelings of shame and self-blame. They have low self esteem and experience difficulty relating to other people (van der Kolk, 2002a,b). Our clients with complex PTSD typically do not maintain long-term relationships and often have only superficial connections with others.

Clinically, individuals with complex PTSD appear anxious, hyperalert, and startle easily. They often report having difficulty sleeping and report disturbing nightmares. Long-term childhood maltreatment can result in recalibration of components of the body’s stress response system. Because they are chronically physiologically hyperaroused, many individuals with complex PTSD are constantly in survival mode. Others rapidly shift into trauma mode when confronted with stressful situations or triggered by sounds, sights, or smells that remind them of traumatic events. They instantly respond physiologically for fight or flight. If neither of these options is available, they freeze.

Individuals with complex PTSD have difficulty regulating affect and in order to avoid intense feelings, they have learned to dissociate from their feelings and become numb. A common strategy is to numb intense feelings with alcohol, drugs, or food. These maladaptive coping strategies often impair social and occupational functioning. Obviously, these trauma-related mechanisms for surviving perceived danger can contribute to antisocial behaviour. Alcohol and some drugs can impair judgment and reduce inhibition, making violent acting out an even more likely event for individuals with complex PTSD who have ingested these substances than individuals who have not. Violent outcomes are also more probable events for individuals who lack a basic sense of attachment to human beings than for those who feel connected to others.

3. Child maltreatment and attachment

The foundation for all human interaction is learned during the first year of life when parents (or other caregivers) meet the infant’s basic needs for food, physical comfort, and human contact. A good caregiver is attuned to the infant’s needs and shifting physiological and emotional states. When the baby is hungry, wet, cold, or in pain, the caregiver acts to relieve her discomfort. When the baby is frightened, sad, or angry, the adult soothes her and provides safety. By calming a crying or frightened baby, the caretaker regulates the baby’s affect (which the baby cannot yet do for itself). Through frequent, close interaction, infant and caregiver learn to read each other’s signals and respond appropriately. If all goes well, the infant develops a secure attachment, which represents a “secure base” in the world (Bowlby, 1988). The development of a secure attachment bond between infant and caregiver is the foundation for all future emotional and social connections between the child and other human beings (Magid & McKelvey, 1987; Schore, 2003c). The quality of early attachment between child and caretaker affects brain development and behaviour (Main, 1995).

Child neglect can be more devastating to a child than abuse by itself (Cicchetti & Toth, 1995; Hildyard & Wolfe, 2002). Neglect typically begins at an early age. Because the caregiver does not meet the baby’s needs, the baby cannot develop a secure attachment. The absence of a consistent, nurturing caregiver who forms a healthy relationship with the infant and takes care of the infant’s needs is developmentally traumatic. Babies whose physical, emotional, and medical needs are not met learn that they cannot count on other human beings to respond to their needs and to comfort them. Their attachment to their caretaker is insecure and they exhibit disorganized and/or disoriented behaviour in the caretaker’s presence. These children become distrustful and may remain disconnected from other humans. Deep down they may harbour intense rage and hatred. Their pain and anger is a direct result of the parents’ failure to nurture and care for them (Magid & McKelvey, 1987; Schore, 2003c). Compared to physically abused children, neglected children exhibit more severe cognitive deficits and fare worse academically. They are more likely to withdraw socially, and they internalize problems, rather than externalize them (Hildyard & Wolfe, 2002). Maternal rejection at age 1 has been shown to predict violent crime at age eighteen (Raine, Brennan, & Sarnoff, 1994).

Like neglected children, those who have been abused typically develop disorganized, insecure attachments to their caregivers. Without this most basic relationship, children do not develop a healthy self-concept. The developmental deficits resulting from poor attachment negatively impact their future ability to relate well to others and to regulate
emotion effectively (van der Kolk & Fisler, 1994; Schore, 2002). Unattached children often do not develop compassion and empathy for others. Accordingly, these individuals are more prone to acting out violently in relationships because they do not form attachments to other people (Schore, 2003a, b).

4. Biological effects of childhood trauma

Recent studies have contributed to our understanding of the physiological impact of traumatic events and the long-term biological consequences of those experiences on the nervous and endocrine systems. Body systems are closely linked and when one system is adversely affected, a domino effect occurs that causes changes in many other organs and systems of the body. Findings regarding the biological effects of childhood trauma are discussed in the next sections.

4.1. The brain does not complete its development until adulthood

Research findings during the past several years have provided evidence that the brain continues to develop throughout childhood, adolescence, and into early adulthood. The brain does not complete its development until the early 20s and, perhaps, not until about age 25 (Beckman, 2004). The gray matter in the lateral region of the prefrontal cortex is responsible for executive functions such as controlling impulses, reasoning, and making decisions. Interestingly, the prefrontal cortex is one of the last regions of the brain to mature (Giedd, 2004).

During its development, the brain produces more neurons and forms more neural connections than it needs. This overproduction, the subsequent pruning of these neurons, and the formation of new synapses (connections between neurons that allow neurons to form neural pathways) contribute to neural plasticity, which allows the nervous system to change its structure and function in response to experience. The neural fibres that organize to form neural tracts continue to mature through childhood and adolescence. These fibres increase in diameter and become myelinated (covered by myelin, an insulating fatty substance) (Paus et al., 1999). A positive correlation has been reported between fibre organization and/or density and cognitive function (Schmithorst, Wilke, Dardzinski, Scott, & Holland, 2005).

During the first 2 years of postnatal life the right side of the brain is in a critical growth period. Its normal development depends on healthy attachment (Schore, 1994, 1996, 2003c). The infant’s relationship with its primary caregiver has a direct effect on the wiring of neural circuits in the developing brain.

4.2. Childhood trauma interferes with normal brain development

Childhood trauma interferes with the normal development of the brain. Traumatic stress interferes with the normal death of neurons, development of neural pathways, and with synaptic pruning. Many neural circuits affected by early experience connect areas of the brain critical for emotional, physiological, psychological, and social development. Some of these neural circuits are responsible for the ability to cope adaptively in emotional and stressful situations (Schore, 2003c).

The right hemisphere of the brain is dominant for processing information related to emotion, social interaction, and physiological states. Healthy attachment is critical in the development of key areas of the right brain including the orbitofrontal cortex (a region of the prefrontal cortex), anterior cingulate cortex (ACC; part of the medial prefrontal cortex), and amygdala (part of the limbic system important in assessing emotional experience), and for the neural circuits that connect them (Fig. 1).

The chronic traumatic stress caused by child abuse and neglect compromises right brain development, resulting in neuron damage and atrophy. Neural circuits that connect cortical and subcortical areas are modified in ways that have major psychological consequences. For example, impairment of the orbitofrontal cortex and the circuits connecting it with subcortical areas can diminish the child’s sense of self and result in disconnection from other people.

The orbitofrontal cortex links important centers in the cerebral cortex, the limbic system, hypothalamus, and brain stem. It has a critical role in integrating many important processes and helps regulate emotional states and responses. For example, by way of its connections with the hypothalamus and limbic system, it regulates autonomic responses to social stimuli and mediates emotionally attuned communication.

The orbitofrontal cortex is important in regulating mood and in social adjustment and responsibility. These qualities are important components of an individual’s personality. This part of the cortex helps us understand other people’s emotional experience and thus is important in the capacity to respond empathically. The orbitofrontal cortex normally
inhibits areas in the hypothalamus that are associated with aggression, and is therefore central in the regulation of aggressive impulses.

The ACC is important in experiencing the affective component of pain both for ourselves and for others. This region of the brain apparently allows us to understand how our actions affect another person and to appreciate the feelings of other people. This ability is the basis for empathy. In severely maltreated children, neural pathways in the right brain are compromised. These children do not cope well with stress and do not develop the ability to regulate the intensity and duration of their affect (Schore, 2002; Streeck-Fischer & van der kolk, 2000; van der Kolk and Fisler, 1994). Not surprisingly, they do not understand other people’s feelings and may not develop empathy. Without empathy, children cannot reach higher stages of moral judgment (Kohlberg, 1969).

4.3. Childhood trauma can cause long-term changes in the brain

Research findings demonstrate differences between traumatized and normal brains. Long-term effects appear to be most dramatic in individuals who have endured Type III trauma. During traumatic events, the central nervous system (which includes the brain and spinal cord) is overstimulated, and the physiological changes that occur can cause long-term, perhaps permanent, changes in brain structure and function. Such changes can affect memory, learning, ability to regulate affect, and social development.

Long-term changes in the brain associated with child maltreatment include significantly smaller total brain and cerebral volumes (Carrion et al., 2001; De Bellis, Keshavan et al., 1999), electroencephalogram (EEG) abnormalities, decreased size of the corpus callosum (a large bundle of neurons that transmit information between the two hemispheres), impaired function of the cerebellar vermis (the central region of the cerebellum; helps inhibit limbic system structures), decreased function of pathways in the right brain and limbic system (Ito et al., 1993; Teicher et al., 2003), and changes in neurotransmitter concentration and function (De Bellis, Keshavan et al., 1999). We will describe some of these changes in the following sections.

4.3.1. Electroencephalogram (EEG) abnormalities

Seventy-two percent of children and adolescents with histories of severe abuse exhibit electroencephalogram (EEG) abnormalities (sharp waves and spikes) in the frontal and temporal regions of the left hemisphere (Ito, Teicher, Glod, &
Ackerman, 1998). Among our adult Type III trauma clients, many report that they have been prescribed dilantin and other drugs used to treat epilepsy and other seizure disorders. When explored, these pharmacological interventions were based on abnormal EEGs mistakenly suggesting that these individuals had seizure disorders. The EEG abnormalities seen in trauma survivors have been associated with increased frequency of violence toward self or others.

4.3.2. Effects on the corpus callosum

The corpus callosum consists of most of the neurons connecting the right and left cerebral hemispheres. Child neglect has been associated with a 15–18% reduction in the size of several corpus callosum regions, particularly in boys (Teicher et al., 2004). In contrast, sexual abuse was the strongest factor associated with reduced corpus callosum size in girls. Size decrease in the corpus callosum may decrease integration between right and left hemispheres. Consequently, individuals with a smaller corpus callosum would be less able to use the logic and reason of the left hemisphere to inhibit the rage expressed by the right hemisphere. They would have greater difficulty regulating emotion and thinking things through before acting on their feelings.

4.3.3. Effects on the limbic system

The limbic system is an action system of the brain that helps regulate emotion, memory, and behaviour. This system consists of a group of interconnected nuclei (neural centers) located in the cerebral cortex and hypothalamus. The limbic system mediates the response to unpleasant stimuli in both healthy individuals and in those suffering from the effects of trauma. Certain parts of the limbic system, however, are activated only in individuals with PTSD. Reliving a traumatic experience increases limbic system activation, especially in the right hemisphere.

The amygdala is a part of the limbic system that filters incoming sensory information and interprets it in the context of emotional needs and survival. The amygdala is important in assessing our experience in emotional terms and helps decide whether or not a stimulus is dangerous. When it perceives danger, the amygdala sends information to other parts of the brain so that appropriate responses can be made. Studies have demonstrated that the amygdala is 8–10% smaller in abuse survivors (Teicher et al., 2003). Decreased size of the amygdala has been associated with depression, irritability, and hostility.

Another part of the limbic system, the hippocampus, functions in the formation and retrieval of verbal and emotional memories. The hippocampus places our experience in categories and stores it along with similar memories. The volume of the hippocampus is reduced in trauma survivors, and the extent of hippocampal size decrease has been correlated with the chronicity of abuse.

The smaller hippocampus may account for lower performance on neuropsychological measures involving memory. Trauma survivors also have difficulty in attention and concentration, and many do not remember details about important life experiences. The hippocampus is one of the few parts of the brain that continues to produce new neurons after birth. Exposure to stress hormones, however, inhibits this process and also damages and destroys existing neurons. A recent animal study demonstrated that early maternal separation resulted in a decrease in synaptic development in the hippocampus (Anderson & Teicher, 2004). This condition could directly affect attention and ability to store information (memory).

4.3.4. Effects on neurotransmitters

A number of neurotransmitters (chemical compounds released by neurons that signal other neurons, muscles, or certain glands) are affected by traumatic experience. The principal known brain neurotransmitters are norepinephrine, dopamine, serotonin, glutamate, and gamma-aminobutyric acid (GABA). Several years after suffering severe abuse, children, who were otherwise medically healthy, exhibited significantly higher concentrations of norepinephrine, epinephrine, and dopamine than non-abused anxious controls (De Bellis, Baum et al., 1999). The results correlated positively with duration of the trauma. Furthermore, PTSD symptoms, including intrusive thoughts, avoidance, and hyperarousal correlated positively with high urine concentrations of norepinephrine and dopamine.

Many individuals suffering from PTSD turn to alcohol or drugs, especially heroin, to feel better. Alcohol and benzodiazepines (e.g., Valium, Xanax) stimulate GABA, which inhibits glutamate. This inhibition appears to decrease the frequency with which traumatic memories break through into consciousness.

Serotonin and norepinephrine are important in encoding emotional memory. Serotonin has a critical role in the maturation of neural systems in the brain that regulate emotion in the adult (Ansorge, Zhou, Lira, Hen, & Gingrich, 2004). Early disruption of serotonin function affects adult emotional behaviour. Low serotonin concentration has
been linked to some moods and behaviours associated with complex PTSD including depression, impulsivity, and aggressive behaviour. Low concentration appears to be related to decreased ability to inhibit inappropriate behaviour.

Genetics plays a role in neurotransmitter concentration. Several gene mutations have been linked to low levels of serotonin (resulting in depression). For example, a variant of a gene that controls the rate of serotonin synthesis has been identified that reduces the expression of serotonin in the brain by 80% (Caron & Duke, 2004). Another gene has been located that transports serotonin, and a variant of this gene was also associated with depression. This gene may help moderate the negative effects of maltreatment (Caspi et al., 2002). Men who carry a variant of the gene have a higher risk for being aggressive, impulsive, and even violent if they were abused as children or if they drink alcohol.

4.3.5. Traumatic memories get stuck in the limbic system

Memories of ordinary experiences are temporarily stored in the limbic system as episodic memories, which are memories of personal experience and events. Episodic memories are autobiographical; they include a sense of time and self. The cognitive aspects of memories are temporarily stored in the hippocampus, whereas the associated emotion is stored in the amygdala. As the brain processes these memories over time, aspects of them are abstracted and transferred to the neocortex, particularly the association areas of the frontal lobes, for long-term storage. These stored memories are referred to as semantic, or factual, memories.

Memories of moderately disturbing experiences apparently remain in the right limbic system for a longer period of time than memories of neutral events. We process disturbing memories by thinking, talking, and sometimes dreaming, about the experience. As the brain slowly processes the memory, it is transferred into the left cerebral cortex where it is filed away along with other memories and becomes part of the story of one’s life. We can retrieve the stored information when we need it to understand new experiences.

Traumatic events overwhelm the brain’s capacity to process information. The episodic memory of the experience may be stored in the right limbic system indefinitely. These dysfunctionally stored memories may generate vivid images of the traumatic experience, terrifying thoughts, feelings, body sensations, sounds and smells. Such unprocessed traumatic memories can cause cognitive and emotional looping, anxiety, maladaptive coping strategies, depression, and many other symptoms of complex PTSD. Because the episodic memory is not processed, a relevant semantic memory is not stored. Consequently, the trauma survivor has difficulty using knowledge from the past experience to guide future action. An individual who cannot learn from his experience is at greater risk of behaving inappropriately, even violently.

4.3.6. The limbic system remains on red alert

The limbic system mediates the response to unpleasant stimuli in both healthy individuals and in those suffering from the effects of trauma. Flashbacks of traumatic events appear to be orchestrated by the amygdala. During a traumatic episode, the amygdala becomes programmed to remember the smells, sounds, and sensations that are part of the experience. From that time on, stimuli associated with the traumatic experience can act as triggers. When a trauma survivor is triggered, the limbic system (especially in the right hemisphere) is activated, and the survivor may relive the traumatic experience.

The body instantly responds to sensory input that triggers memories of a traumatic experience by going into prepare-for-danger mode. Physiological hyperarousal, distress, and anxiety may lead to intense emotional reactions. The exaggerated responses of the amygdala to harmless stimuli perceived as threatening is dissociated from cortical (medial frontal) activation (Rauch et al., 2000). In fact, the frontal cortex is not activated. Consequently, responses are generated by the limbic system and are accordingly rooted in emotion rather than in rationality and judgment.

Higher cortical areas normally modulate limbic system responses. For example, the ACC normally inhibits the amygdala so that it does not respond too strongly. Early trauma compromises the normal development of neural circuits between the prefrontal cortex (especially the orbitofrontal and ACC) and the amygdala. Impaired development of the orbitofrontal cortex and its neural connections with the limbic system decreases capacity to regulate affect.

A study using functional magnetic resonance imaging (fMRI) (Shin et al., 2001) demonstrated that triggers decrease the normal response of the ACC, so that the amygdala is not effectively inhibited. As a result, blood flow increases to the amygdala causing the amygdala’s responses to be exaggerated. Thus, the survivor’s emotional reactions are fueled by the traumatic experience.
Abnormal development of the neural circuits linking the orbitofrontal cortex and ACC with the amygdala interferes with normal inhibition of rage responses. When aggressive impulses are not inhibited, an individual may act out violently. This lack of inhibition is part of the pattern of sociopathy (Best, Williams, & Coccaro, 2002; Schore, 2003c).

In PTSD, abnormal functioning of areas of the hippocampus and the prefrontal cortex leads to memory problems and dysregulation of emotion. Individuals with decreased hippocampal function are more likely to respond to emotionally charged information with aggression and to have difficulty learning from emotional experience.

4.3.7. Remembering traumatic experiences can cause speechless terror

Broca’s area, a region in the posterior part of the left frontal cortex, is critically involved in generating words, and thus, in the expression of language. Neuroimaging studies using positron emission tomography (PET) show greater deactivation of Broca’s area in individuals with PTSD. In response to exposure to detailed accounts of their traumatic experiences, activity in Broca’s area decreases in trauma survivors (Pitman, Shin, & Rauch, 2001; Rauch et al., 1996). This decrease in activity may be the physiological basis for the difficulty that trauma survivors have describing their experience in words. Survivors have described flashbacks of traumatic experience as being in a state of “speechless terror.” Without words, it is difficult to process and resolve a traumatic experience.

4.3.8. Trauma survivors have difficulty modulating autonomic arousal

In addition to adversely affecting brain function, early exposure to severe stress can cause long-term changes in the body’s sympathetic nervous system and hormonal response to stress. When danger threatens, the body’s immediate response is sympathetic nervous system activation. The cerebral cortex sends neural messages to autonomic centers in the hypothalamus, activating the hypothalamic–adrenal medullary response. Sympathetic nerves transmit messages from the hypothalamus to the adrenal medullae. These endocrine glands flood the body with epinephrine and norepinephrine, in preparation for fight or flight. Epinephrine and norepinephrine act on many organs in the body, including the brain, heart, and blood vessels.

Sympathetic nervous system activity in trauma survivors with PTSD is greater than in normal individuals, and epinephrine and norepinephrine levels remain elevated even in the absence of danger (Schnurr, Friedman, & Bernardy, 2002). Early exposure to stress programs this stress-response system to respond more rapidly and more intensely. In a hyperaroused state, responses are driven by the limbic system and by brain stem activity. Increased difficulty accessing higher cortical centers, areas of the brain essential for formulating appropriate decisions, increases the risk for impulsive and aggressive behaviour.

5. Childhood trauma causes long-term changes in the endocrine system

The hypothalamic–pituitary–adrenal axis is a major stress response system, an important back-up system for the adrenal medullary system. When activated by stressors, the cerebral cortex signals the hypothalamus to release corticotropin-releasing factor (CRF). CRF stimulates the pituitary gland to increase its output of adrenocorticotropic hormone (ACTH). This hormone acts on the adrenal cortex, stimulating cortisol secretion. Cortisol activates metabolic processes that provide adequate fuel supplies for the cells when they are stressed. In this way, cortisol provides important back-up for the adrenal medulla, permitting the individual to respond effectively.

Recent studies have demonstrated that hyperactivity of the hypothalamic–pituitary–adrenal system is a long-term consequence of child abuse (Heim, Ehlert, & Hellhammer, 2000). In trauma survivors, the hypothalamus increases its output of CRF. Neurons that secrete CRF have also been located in nuclei in the brainstem that project to the cerebrum and hypothalamus. These neurons may also increase their CRF output. Elevated levels of CRF appear to contribute to anxiety and depression. Too much CRF also appears to decrease appetite, disrupt sleep patterns, increase heart rate and blood pressure, and increase the startle response to noise.

In abused children, cortisol concentrations are above normal levels. However, in neglected children, cortisol concentrations eventually become low, despite high CRF levels (Gunnar & Vasquez, 2001). In adult trauma survivors cortisol levels are lower than would be expected (Newport & Nemeroff, 2000). The body appears to adapt to long-term trauma (and to chronic stress) by decreasing its cortisol output. Exposure to new stressors, then, elicits lower levels of cortisol secretion than in non-traumatized adults.
Because normal amounts of cortisol are necessary to modulate physiological responses to stress, a traumatized child becomes more vulnerable to stressors later in life. These hormonal changes increase an adult’s vulnerability even to mild stressors, contributing to an increased risk of mood and anxiety disorders, as well as to immune-related disorders and chronic pain syndromes (Heim et al., 2000). Insufficient amounts of cortisol also appear to predispose individuals to using maladaptive coping strategies. Interestingly, violent offenders with Antisocial Personality Disorder have low levels of cortisol (Virkunen, 1985).

As we have discussed, early traumatic stress directly affects the developing limbic system. In neglected children, the hypothalamus is affected, resulting initially in physiological hyperarousal. Later, hypoarousal occurs (Schore, 2003c). In the adult, this hypoarousal, which typically includes low heart rate and blood pressure, may be associated with a pattern of acting out rage in predatory violence. In fact, physiological under-arousal is characteristic of sociopaths. Recent findings show that violent juvenile offenders have a low resting heart rate (Cauffman et al., 2005).

Fig. 1 summarizes the biological effects of severe abuse and neglect. This diagram illustrates the critical interconnections of nervous and endocrine systems in the development of an abnormal physiological state that can lead to antisocial behaviour.

5.1. A positive feedback loop intensifies aggressive behaviour

Recent studies in rats have demonstrated that a positive feedback loop between the aggression center in the brain and the release of stress hormones by the adrenal cortex intensifies aggressive behaviour. Thus, stress and aggression form a rapid positive feedback loop (Kruk, Halasz, Meelis, & Haller, 2004). When stress increases, aggression increases. Conversely, aggressive behaviour stimulates the release of stress hormones. When a certain area of the hypothalamus (called the “attack center”) is stimulated in rats, the stress hormone corticosterone (similar to cortisol) is rapidly released even when no opponent rat is present. This mechanism may explain how stressors rapidly generate and exacerbate violent behaviour. For example, the authors have evaluated a number of homicide offenders who stabbed their victims multiple times (57 in one case). Once in this positive feedback loop, the offender continues to act out violently until his or her rage has dissipated.

It is important to note that while some survivors of abuse and neglect act out against others, many direct their aggressive impulses against themselves. We have worked clinically with many trauma survivors who engage in overt acts of violence against themselves, such as cutting or burning their own bodies. Others develop more subtle patterns of self-defeating behaviour. Both those who act out against others and those who harm themselves often turn to alcohol or drugs, or develop other maladaptive coping strategies in attempts to dampen their painful and aggressive feelings.

5.2. Trauma causes a disconnect between the mind and body

During traumatic experiences, the body is overwhelmed and the mind dissociates from body sensations, as well as from affect. When children experience long-term abuse and/or neglect, splitting off their feelings and their body sensations often becomes a way of being. In our clinical practices, we see many type III survivors who are so disconnected from their bodies that they do not experience touch, pressure, or even pain. Clients have come in to therapy sessions with serious injuries and explained that they were unaware that they had been hurt until they noticed the blood. When they do feel, many trauma survivors have difficulty assigning emotional significance to their sensory experience.

5.3. Genetics plays a role

Some children who are maltreated do not develop PTSD. Furthermore, many adults who suffered child abuse are law-abiding citizens. Certain genes increase the risk for PTSD; others, for developing Antisocial Personality Disorder. For example, males with a history of child abuse and the genotype for low monoamine oxidase A (MAOA) activity were more likely to exhibit antisocial behaviour (Caspi et al., 2002) (MAOA metabolizes neurotransmitters, including norepinephrine, dopamine, and serotonin). In fact, maltreated males with low MAOA activity were three times as likely to be convicted of violent crimes by age 26 as males who had been maltreated, but had the normal gene for MAOA activity.
6. Implications for offender accountability

The findings synthesized above have important implications regarding how the justice system evaluates the responsibility of juvenile and adult violent offenders with significant trauma histories. We know that child abuse increases the risk of criminality by about 50% (Widom, 1997). Histories of severe childhood maltreatment are common in preteen homicide offenders (Shumaker & Prinz, 2000) and adolescent homicide offenders (Heide, 1992, 1999; Lewis et al., 1985), including those sentenced to death for murder (Beckman, 2004; Lewis et al., 1988), as well as adult homicide offenders (Lewis, 1999; Lewis et al., 2001; Lewis, Yeager, Swica, Pincus, & Lewis, 1997; Pincus, 2001). Many offenders suffer from complex PTSD. In these individuals, a variety of stimuli that are not dangerous keep the body in an almost continuous state of physiological arousal, making it very difficult for these individuals to discriminate between real and imagined danger. Responses to perceived danger are generated at subcortical levels, increasing the probability of such individuals’ reacting irrationally out of fear and anxiety. As van der Kolk (2002a,b) has pointed out, the inability to accurately evaluate the emotional significance of sensory experience makes it difficult for trauma survivors to learn from their experience.

Mental health professionals are often asked to evaluate violent offenders, particularly those who commit homicide (Ewing, 1987, 1990; Lewis, 1999; Malmquist, 1996; Palermo, 2003; Walker, 1989). Our review of recent findings on the biological effects of trauma clearly indicates that forensic evaluations should include a trauma history and careful assessment of the effects of traumatic experience (Heide, 1999; Heide and Solomon, 1997). Our clinical experiences have suggested that trauma-driven neurophysiological changes can dramatically impact the nature, results, and conclusions of forensic evaluations, including those that address competency issues, mental status at the time of the offence, and factors in mitigation.

In assessing competency to stand trial, a key issue is the offender’s ability to assist counsel in his/her defence. The authors have evaluated homicide offenders who were not able to assist their attorneys due to complex PTSD. In several of these cases, when the evaluator raised the issue of the offence, the offender appeared to shift into trauma mode and experienced terror. In this state, these defendants were not able to access information or to continue the discussion.

Recognizing the symptoms of complex PTSD can be a key factor in understanding the defendant’s state of mind at the time of the crime. Awareness that the defendant may be re-experiencing the trauma can help to explain violent behaviour that seems grossly disproportionate to the situation or completely inappropriate and senseless. In some cases, complex PTSD could result in an offender being excused from responsibility for her behaviour, that is, found not guilty by reason of insanity. In other cases, complex PTSD could result in an offender being held less accountable for his behaviour, that is, found guilty possibly of a lesser charge.

Mitigating factors are often presented at sentencing, especially in capital cases. In most states, statutory mitigating factors include the following: (1) The defendant was under the influence of extreme mental or emotional disturbance, and (2) The capacity of the defendant to appreciate the criminal nature of his behaviour or to conform his conduct to the requirements of law was significantly impaired (Melton, Petrila, Poythress, & Slobogin, 1997; Heide, 1992, 1999). Many individuals with complex PTSD are continuously “under the influence of extreme mental or emotional stress.” Others are triggered by stressors and rapidly shift into the trauma mode. In this state, trauma survivors feel overwhelmed by intense feelings of terror, and often helplessness. Our clinical experiences have suggested that the capacity of a defendant in this state to “appreciate the criminal nature of his behaviour or to conform his conduct to the requirements of law” may be significantly impaired.

7. Conclusions

This article has reviewed recent scientific findings showing that Type III trauma interferes with normal development and is often associated with long-term neurophysiological changes involving the brain and endocrine system. Abnormal development of the brain can lead to cognitive deficits; affect dysregulation, lack of empathy, rage, and aggression. Type III trauma survivors are typically diagnosed with complex PTSD. They have difficulty accessing higher cortical centers when in stressful situations, and consequently, they are at greater risk of behaving inappropriately. For example, they are more likely than those without a trauma history to lash out violently at others, to take flight, or to freeze in panic.

Research findings raise the question to what extent individuals with complex PTSD should be held accountable for violently explosive behaviour. Are Type III trauma survivors responsible for inexplicable violence if they are
essentially in a survival mode when they attack others based on past events? Is it morally defensible to find these defendants culpable for their behaviour if scientific findings provide convincing evidence that individuals cannot process and evaluate information when they are in this hyperaroused state?

There is some evidence that affective murderers can be distinguished from predatory murderers, as well as from a control group. Raine et al. (1998) found that, relative to a comparison group, affective murderers had lower right and left prefrontal functioning, higher right hemisphere cortical functioning, and lower right hemisphere prefrontal/subcortical ratios. In contrast, although the predatory murderers also had excessively high subcortical activity, their prefrontal functioning was more equivalent to that of the controls, indicating that they were able to make decisions.

If indeed excessive subcortical activity predisposes to aggressive behaviour, it would seem that affective murderers, relative to predatory murderers, would be less capable of regulating and controlling violent impulses generated from subcortical structures due to poor prefrontal functioning than would predatory murderers (Raine et al., 1998). Clearly, longitudinal research combining physiological, neurophysiological, and psychological methods would be helpful in elucidating the pathways by which biological functioning contributes to violent behaviour.

The criminal justice system is based on the foundation of a rational man who makes conscious decisions before acting. For hundreds of years, the legal system has been guided by the premise that human beings are to be held accountable for their behaviour because they have choices. What if the range of choices is limited, or severely compromised, in certain situations for some individuals who have sustained severe trauma and have been significantly affected by it?

One could argue that a history of severe trauma must be considered particularly in cases of violence when an offender with a previously non-violent history suddenly erupts into what appears to be a frenzied attack. The viciousness of the attack, that is, “the overkill,” would appear in itself to be an indication of the offender’s inability to regulate emotion and to evaluate the extent of violence needed to neutralize or stop the victim, and/or to get away.

As the literature grows showing that Type III survivors with complex PTSD are more at risk of behaving maladaptively, other questions pertinent to the area of individual responsibility must be considered. To what event should the effects of alcohol, drugs, neurological impairment, intellectual deficits, and/or low personality development be factored into the equation? If individuals with complex PTSD have difficulty thinking clearly and responding appropriately under renewed stress, what happens when these individuals have consumed significant quantities of alcohol or mind-altering drugs? The difficulties in regulating affect and behaviour would seem to be additive under these conditions. Similarly, if these individuals have neurological impairments, intellectual deficits, and/or low personality development, their ability to stop, to evaluate their behaviour, and to decide on a rational course of action would seem to be increasingly compromised.

Clearly, the scientific findings synthesized herein have important implications for the disposition of offenders. As noted by Cauffman and her colleagues in a 2005 seminal article in *Criminology*, the debate is often contentious between those who view serious offenders as individuals whose violent behaviour is biologically determined and those who believe “that the potential plasticity of serious offenders is virtually limitless.” Proponents of the first approach miss the mark by concluding that many serious offenders are “biologically incorrigible” and cannot be helped. Proponents of the second approach err by their refusal to acknowledge that some offenders’ behaviour is indeed “biologically driven” (p. 162).

**7.1. Implications for treatment**

Like Cauffman and her colleagues, we believe that enhanced understanding of violent offenders is needed and that biological, neurophysiological, and psychological factors should be considered in discussions of meaningful assessment and effective intervention. A growing body of literature suggests that trauma survivors can be helped if provided with effective treatment. Unfortunately, many traditional therapies, including cognitive-behavioural therapy (CBT), are not effective in treating complex PTSD because they do not address the physiological issues driving maladaptive behaviour (Solomon & Heide, 2005). For example, CBT teaches clients how traumatic experience has affected them and helps them change irrational beliefs. This approach helps clients gain insight into their thoughts and behaviour, and learn to identify stimuli that trigger them. They gain insight into their maladaptive responses patterns, and may learn to manage disturbing feelings. However, they do not process the traumatic (episodic) memories or resolve their physiological hyperarousal. Consequently, these clients continue to...
be triggered by stimuli that remind their limbic systems of their traumatic experiences, and may continue to respond maladaptively.

Biologically informed psychotherapy addresses the physiological effects of trauma (Solomon & Heide, 2005; van der Kolk, 1996). Clients are helped to process the episodic memories of the traumatic experience. The memory can then be transferred from the limbic system to the cortex and filed away along with other narrative memories. Clients learn to tolerate intense feelings and to release emotion in appropriate ways, and they learn to calm their physiology. This approach helps clients reconnect with their bodies and with their feelings.

Several treatment approaches have been developed that consider the biological aspects of trauma recovery. The one most studied and most widely used is Eye Movement Desensitization and Reprocessing (EMDR). EMDR is an effective combination of body-focused and cognitive-behavioural treatment (Solomon & Heide, 2005). EMDR helps trauma survivors’ access and process traumatic memories so that they can be brought to an adaptive resolution (Shapiro, 2001). At least fourteen empirical studies support the efficacy of EMDR (Servan-Schreiber, 2000; Shapiro, 1999).

7.1.1. Critical questions for policy development and action

Research findings are leading us to unparalleled understanding of the brain and the endocrine system. At the same time, these findings are bringing us to an age old question. To what extent do recent advances in science challenge notions of morality that date back thousands of years? As we learn more about the brain and individual or group differences in our capacity to control feelings, thoughts, and behaviour, will we as a society have the social conscience needed to “re-invent justice”? Will we do what is necessary to ensure that the law is fair, just, and humane to those who are constitutionally more at risk of responding and behaving maladaptively through no fault of their own?

If society is really committed to reducing and preventing violence, including homicides, we will expend the resources to further educate the public about the deleterious effects of child abuse and neglect. We will determine which treatment or combination of intervention strategies is most effective with helping survivors of trauma overcome the negative aspects of their pasts and lead healthy and contributing lives. We will allocate the funds to ensure that survivors of childhood maltreatment have equal access to state-of-the-art treatment.

References


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