PSYCHOLOGICAL CONSEQUENCES OF CHILD MALTREATMENT

Child abuse or maltreatment affects psychological states with a complex matrix of behavioral, emotional, and cognitive factors [1]. Child abuse also elevates the risk of psychiatric and medical diseases [2, 3] although not all exposed individuals demonstrate the same altered responses, suggesting that the interactions between genetic, epigenetic, and psychosocial-environmental factors modulate the consequences of child maltreatment.

Child abuse substantially contributes to child mortality and morbidity, but it also has long-lasting effects on mental health, drug and alcohol misuse (especially in girls), risky sexual behavior, obesity, and criminal behaviour, which persist into adulthood [4]. In addition, its effects may extend beyond the immediate victim into subsequent generations as a consequence of epigenetic effects transmitted directly to offspring and/or behavioral changes in affected individuals. Exposure to multiple forms of abuse has been said to be associated with very large effect sizes [4, 5]. In fact, most maltreated children are exposed to multiple types of abuse and the number of the different types is a critically important factor [6].

There is an increasing evidence of the effects of childhood trauma in the developing brain of children and adolescents and of its long-lasting neurobiological effects during adulthood [1]. One of the effects of child maltreatment is the acquired inability of the brain to inhibit some negative actions [1]. Child abuse may result in a chronic incapability to modulate emotions, thus augmenting the risk of getting involved in indiscriminate relationships with others in which old traumas may be repeated [1, 7-12]. In fact, abnormalities in the orbitofrontal cortex and amygdala as a consequence of trauma exposure may impair decision-making and may also predispose to act more impulsively in the future [13, 14]. The hippocampus also participates in cognitive processes and seems to be particularly vulnerable to stress [15]. A shrink in hippocampal volume has been described as a major biological consequence of exposure to trauma [16, 17]. Damage or atrophy of the hippocampus impairs the inhibitory role of this cerebral region and leads to more prolonged hypothalamic-pituitary-adrenal (HPA) response to psychological stressor. Moreover, stress may close this loop because it also leads to loss of neurons in the hippocampus and to a decrease in synaptic connectivity [18, 19]. However, some other factors, such as the effect of alcohol and substance abuse, may also affect hippocampal size, because some studies fail to show a reduced hippocampal volume in response to trauma, especially in children and young adults [20].

Additional neurobiological structures may also be affected as a result of childhood abuse [21, 22]: 1) the middle portion of the corpus callosum, thus leading to an increased hemispheric laterality and decreased hemispheric integration; 2) the anterior cingulate cortex, which is especially involved when effort is needed to perform a task such as in early learning and problem solving [23]; 3) the cerebellar vermis, a region of the brain which has the highest density of glucocorticoid receptors during development; and, 4) the maturity of the left hemisphere neocortex.

Multiple neurotransmitters and hormones are also involved in the chronic stress response. Fear traits are said to be much influenced by the amygdala, the serotonergic, the noradrenergic, and the gamma-amino-butyric acid (GABA) systems, while anger seems to be mostly regulated by the nucleus accumbens and the dopaminergic and glutamatergic systems [15]. As neurohormones, neurotransmitters and neuropeptides interact, the dysfunction of one brain neurochemical system affects other systems. Besides glucocorticoids and neurotransmitters, a number of protein hormones, as well as other endogenous and exogenous substances, have been said to intervene in the stress response leading to several psychobiological abnormalities: insulin, insulin-like growth factor (IGF-1), growth hormone, adenosine, caffeine, ghrelin and leptin [15, 18].

With prolonged chronic stress, the HPA axis is hyperactivated, with the resulting release in adrenocorticotropic and cortisol, which involves structural changes, cell atrophy and neuronal loss [15]. Child abuse [24] may lead to a chronic hyperactivation of the HPA axis that finally leads to a blunted cortisol response to diverse stimuli. However, as not all exposed individuals demonstrate altered HPA axis physiology, genetic and complex epigenetic variations together with the effect of environmental factors may be crucial to explain variations in the consequences of trauma exposure [24, 25].

There have been relatively few investigations of the development of the HPA axis during adolescence, although the studies currently available indicate that the effects of stress exposure during adolescence differ, and may be longer-lasting than, effects of the same stress exposure in adulthood [26, 27]. The effects of adolescent stress depend on a number of factors, including: age, gender, duration of stress exposure, type of stressor, and time between stress exposure and testing [26]. Some developmental factors should also be taken into account (e.g. although hippocampus dysfunction has been proved to be a potential consequence of early trauma exposure during adolescence, clear neuroanatomic changes -hippocampal atrophy- is not clearly assessed until adulthood [28]). However, when studying patients with adult borderline...
personality disorder (BPD) [29] those with a history of childhood abuse have smaller pituitary volumes than those BPD patients without history of childhood maltreatment, possibly as a consequence of a HPA axis dysfunction.

Interactions between the developing amygdala and HPA axis underlie critical periods for emotional learning, which are modulated by developmental support and maternal care [30]. Heim et al. [31] found decreased concentrations of oxytocin in the cerebrospinal fluid (CSF) in adult healthy women exposed to childhood maltreatment compared to those who had never endured early life adversities. The neuropeptide oxytocin (OT) plays a key role in mediating social affiliation, attachment, social support, maternal behavior and trust, as well as protection against stress and anxiety.

Moreover, severe stress or trauma can lead to excessive serotonin activation in many regions of the brain [32]. Excessive elevation in serotonin levels appears to eventually result in serotonin depletion if trauma is chronic or persistent [33]. Chronic serotonin activation may result from re-experiencing the trauma and intrusive thoughts, even if the actual traumatic stressor is not continuing to occur. Reduced availability of serotonin then leads to a decreased ability of the central nervous system to dampen emotional responses to later stressors, increasing one’s proneness to dysphoric states and hyperarousal symptoms after trauma exposure. Low serotonin function has been correlated with impulsive and aggressive behaviors in children, adolescents and adults with different psychiatric diagnoses including depression, substance abuse disorders, PTSD, and has also been related to suicidal behavior [34-39].

In summary, child abuse may lead to serious psychobiological consequences that may be persistent through the life course. Complex interactions between genetic, epigenetic, and psychosocial-environmental factors account for the differences observed between individuals that have been exposed to child maltreatment. Although evidence available supports a superior effectiveness of the primary prevention interventions over the second and tertiary strategies, trying to lessen the negative effects of child abuse should encourage new research strategies on the psychobiological treatment of this condition.

REFERENCES: