Chapter 3 Early trauma as conditioning of psychopathology in adult women

Capítulo 3 Trauma temprano como condicionante de la psicopatología en mujeres adultas

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Abstract

Violence is a public health problem, with severe negative consequences on the mental and physical health of people, female primarily; one of five women reports more child abuse or maltreatment than each one of 13 men; women who have suffered some form of child abuse, have up to four times the risk of developing symptoms of depression in adult life. According to the WHO, between 1990 and 2013, people with depression or anxiety increased by 50%, from 416 million to 615 million. About 10% of the world's population is affected, and mental disorders account for 30% of the global burden of non-fatal disease. Humanitarian emergencies and conflict increase the need to expand therapeutic options. Major Depressive Disorder is characterized by severe mood alteration, displeasure and these affect the social, work, and personal areas; the persistence of the disorder could cause distress and physical and functional disability. The women are at twice the risk of suffering depression and suffer more severe depressive symptoms. In addition, women who have been victims of physical or sexual abuse have higher rates of mental health problems. Exposure to child abuse is associated with a markedly increased risk of psychiatric and medical disorders; The hypothalamic-pituitary-adrenal (HPA) axis is one of the main signaling pathways activated in response to stress and trauma. Alterations in the HPA axis or allostatic load are a psychophysiological condition due to the chronic persistence of child abuse, increasing the risk of suffering from mental disorders in adulthood. It is necessary to emphasize the importance of early trauma when psychiatric diagnosis, offer better treatment options, and stop the family chain of adverse events.

Early trauma, Allostatic load, Psychopathology, Women

Resumen

La violencia es un problema de salud pública, con graves consecuencias negativas en la salud mental y física de las personas, principalmente de las mujeres; una de cada cinco mujeres denuncia más abuso o maltrato infantil que cada uno de los 13 hombres; las mujeres que han sufrido alguna forma de abuso infantil, tienen hasta cuatro veces más riesgo de desarrollar síntomas de depresión en la vida adulta. Según la OMS, entre 1990 y 2013, las personas con depresión o ansiedad aumentaron un 50%, pasando de 416 millones a 615 millones. Alrededor del 10% de la población mundial está afectada, y los trastornos mentales representan el 30% de la carga mundial de enfermedades no mortales. Las emergencias humanitarias y los conflictos aumentan la necesidad de ampliar las opciones terapéuticas. El Trastorno Depresivo Mayor se caracteriza por una severa alteración del estado de ánimo, el malestar y estos afectan a las áreas social, laboral y personal; la persistencia del trastorno puede causar angustia y discapacidad física y funcional. Las mujeres tienen el doble de riesgo de padecer depresión y sufren síntomas depresivos más graves. Además, las mujeres que han sido víctimas de abusos físicos o sexuales presentan mayores tasas de problemas de salud mental. La exposición al maltrato infantil se asocia a un riesgo notablemente mayor de trastornos psiquiátricos y médicos; El eje hipotálamo-hipófisis-suprarrenal (HPA) es una de las principales vías de señalización que se activan en respuesta al estrés y al trauma. Las alteraciones del eje HPA o carga alostática son una condición psicofisiológica debida a la persistencia crónica del maltrato infantil, aumentando el riesgo de padecer trastornos mentales en la edad adulta. Es necesario enfatizar la importancia del trauma temprano a la hora del diagnóstico psiquiátrico, ofrecer mejores opciones de tratamiento y detener la cadena familiar de eventos adversos.

Trauma temprano, Carga alostática, Psicopatología, Mujeres

3.1 Introduction

In modern psychology, there is great interest in the relationship between early trauma due to adverse experiences in childhood and psychopathology in adult women; first, we will present in this chapter the definition of adverse experiences in childhood, their classification, the outcome, and how this could bring socio-emotional and physiological alterations. Second, we will define early trauma and its effects on physical and mental health; then, we will focus on trauma in women. We will explore the statistics; the studies focused on post-traumatic stress in women and the impact of life stress on psychosis and attenuated psychosis. We will address the impact of early trauma and neuronal affectations due to child abuse and the relationships with an inappropriate or prolonged activation function of the hypothalamic-pituitary-adrenal axis (HPA).

We will also address schizophrenia and the impact of stress and early trauma on the development of this disease; the allostatic load and psychosis will be related too. Finally, we will talk about the public policies that exist in México to prevent early trauma.

3.2 Methodology

One search in PubMed NCBI about “childhood trauma”, “psychopathology” and “women” has deployed 145 articles and 90 of them have been included. The following topics were defined:

> Adverse childhood Experiences, Early trauma, Early trauma in women, Impact of early trauma, Neuronal alterations’ due to childhood maltreatment, Child maltreatment and the hypothalamic-pituitary-adrenal axis (HPA), Allostatic load and mental pathology, Schizophrenia, The impact of stress and early trauma on the development of schizophrenia and Public Policies in México for the prevention and care of early trauma

3.3 Topics

3.3.1 Adverse childhood experiences

Adverse Childhood Experiences (ACEs) act as stressors and risk factors that impact well-being during adulthood. Although these factors are commonly reported in Western countries, little data focuses on low- and middle-income countries. However, studies show that the prevalence of these experiences is frequently higher than those reported in studies from more developed nations. These data indicate social disparities between the world's communities. The greater the adverse experiences in childhood, the greater the risk of chronic diseases, the more significant the social and emotional impact in later years. There is an association between ACEs and the risk of diseases including diabetes, obesity, and heart diseases. Given that ACEs carry devastating impacts until adulthood, it is imperative to focus on the prevention of these experiences in vulnerable populations, developing programs of detection and prevention, as well as an early warning and contingency system (Chu and Chu, 2021).

Abuse, as well as emotional neglect, leads to an increased risk of mood and anxiety disorders, substance and alcohol abuse, and certain other medical disorders in adulthood. Negligence is defined as the failure of a parent or another caring person, to implement the responsibility or supervision and don't provide food, clothing, shelter, and necessary medical attention to minors (Lippard and Nemeroff, 2020).

Abuse and emotional neglect lead to an increased risk of mood and anxiety disorders, substance and alcohol abuse, and certain other medical disorders in adulthood. Negligence is defined as the failure of a parent or another caring person to implement responsibility or supervision and do not provide food, clothing, shelter, and necessary medical attention to minors (Amores-Villalba and Mateos-Mateos, 2017).

Recent studies have shown that exposure to childhood maltreatment, anywhere stage of development, could trigger disease that perdures all life. Childhood maltreatment is associated whit a higher risk of psychiatric and physical disorders. Alterations on the Hypothalamic-Pituitary-Adrenal axis (HPA) and inflammatory cytokines due to childhood maltreatment could contribute to the vulnerability of depression (Lippard and Nemeroff, 2020). Furthermore, there is evidence about a maltreatment history is associated with specific depressive symptoms in adulthood, more severe, more chronic, and a worse response to pharmacological therapy (Vitriol, et al., 2017).

3.3.2 Early trauma

According to World Health Organization, 66.2% of adults report traumatic experiences before 18 years old and they have pointed multiplex traumas in an average of two or more (Dennis et al., 2009). Trauma is the effect on the human psyche caused by a traumatic event. We can consider a traumatic event as an event that is perceived as threatening or aversive, and that could damage self-security and self-integrity and cause fear and inability feelings, so this triggers physiologic arousal by stress reaction (DSM-5). Supposing this event occurs in early childhood when physical and psychological coping is lower, and resources for fighting or flying are low. In that case, the psychological impact will be more significant and chronic, and as a result, it will damage the physiological response on adolescence and adulthood front life stressors.

Trauma can be classified as a) physical abuse, defined as physical violence, b) sexual abuse is a force to the child to do sexual activities, c) psychologic and emotional abuse which includes, threats, intimidation, discrimination, rejection and ridicule, and d) neglect about health, education, nutrition, and safe living of child (Martins et al., 2011; World Health Organization [WHO], 2006).

Early trauma is associated with a higher risk of many medical disorders such as ischemic cardiopathy, diabetes, irritable bowel syndrome, asthma, and others. Also, early trauma induces poor health, depression, anxiety, and sudden death in the adulthood (Brown et al., 2009; Danese and McEwen, 2012; Danese et al., 2009; Dennis et al., 2009; Dube et al., 2009; Openshaw et al., 2015). Hence, we can consider the ACEs as a powerful predictor of health state in adulthood. The impact of traumatic experience on psychological development depends on many factors, including age at the first event, frequency, and active rolling of care people (Antonopoulou et al., 2017). Family history of chronic illness is well related to the effects on the physical and mental health of the child; nevertheless, there are few studies about the effects of abuse and negligent infant on physical and mental childhood health. This highlights the complex interactions between environment, experience, and biological factors. To understand these complex biopsychosocial phenomenon, multidisciplinary efforts are mandatory (Nemerof, 2016).

3.3.3 Early trauma in women

In our society, the woman is the quintessential caretaker of children and adult patients, whether at home or in hospitals, family, friends, or professional work. Women's role as caregivers, both within the health system and at home, can put them at greater risk and may raise concerns about the disease. Approximately 70% of health workers and welfare worldwide are women (WHO, 2019), including frontline health workers. Women are also more likely to care for sick children or other relatives (Wenham et al. 2020).

Gender-based differences are related to the frequency of occurrence of child abuse experiences, while 1 of 5 adult women report having suffered some abuse in childhood, only 1 in 13 men report it; in the same way, women report a greater intensity of emotional abuse than men (Edwards et al., 2003; WHO, 2018). In addition, there is a higher prevalence of post-traumatic stress in women than in men, besides the discordance in the types of traumas experienced between women and men; to unravel the gender-related alteration in these phenotypes because of abuse, more research is needed (Seligowski et al., 2020).

On the other hand, ethnicity also influences the coping response to stress in women; in a recent study, it was found that Hispanic women and African American women had higher levels of hair cortisol than non-Hispanic white women, which could be evidenced by the participation of genetic mechanisms, lifestyles, and social differences (Schreier et al., 2015). Furthermore, ethnicity also influences the coping response to stress in women; in a recent study, it was found that Hispanic women and African American women had higher levels of hair cortisol than non-Hispanic white women, which could be evidenced by the participation of genetic mechanisms, lifestyles, and social differences (Schreier et al., 2015). These differences begin in early life for women, continue into adolescence, and persist into late adulthood; therefore, during women's reproductive and productive years, they present a greater risk of mental illness.

Other interesting aspect to consider is, that psychosocial and biological factors impact mental illness' course and in its treatment response. Recently, it has been revealed that biological processes involved in the predisposition of female depression, including genetically determined vulnerability, hormonal fluctuations, and excessive sensitivity to hormones in the brain system, are mediated by depressive states. The authors also found that psychosocial events such as stress, ACEs, victimization, socialization, cultural beliefs related to gender, coping styles including internalization of stress, and a disadvantaged social status increase women's vulnerability (Sassarini, 2016). In this line, recent studies show that women who had experienced trauma in early life perceive their social networks as less useful, conflictive, and more unpredictable; in general, they perceive less social support than those who did not experience early trauma (Anand et al., 2018; Light et al., 2019).

In addition, neuropsychological problems secondary to child abuse are related to the difficulties these girls encounter when adjusting to school or interacting with her classmate, either due to academic intellectual issues or socio-emotional problems (Amores-Villalba and Mateos-Mateos, 2017).

3.3.4 Impact of early trauma

Recently, researchers have begun to examine the impact of life stressors on psychosis and attenuated psychosis. All psychosis patients who have experienced childhood trauma report increased negative emotions and psychotic symptoms in response to life stress than patients with psychosis, but without early trauma (Collip et al., 2011; Lardinois et al., 2011). Adolescents with Posttraumatic Stress Disorder (PTSD) also developed psychosis at a high rate of 21% in just 2.5 years. This group has also reported many stressful, independent, and undesirable life events than healthy adolescents, and they report more distress in response to daily complaints (Tessner et al., 2011). Additionally, the frequency of daily stressors predicted an increase in positive symptoms one year later.

On the whole, this confers Clinical High Risk (CHR); CHR is a measure used to evaluate the predisposition to develop psychopathologies in later ages (French and Morrison, 2004). It has been found that young people with CHR show an elevated dopaminergic response after stress exposure compared to healthy controls and patients with schizophrenia not treated with antipsychotics (Mizrahi et al., 2012). We can suggest that trauma-derived stress increases the reactivity of the HPA axis in adolescents with attenuated and complete psychosis, triggering fluctuations in dopamine transmission and the severity of psychotic symptoms; this could be influenced by genetic predisposing and environmental factors combined with ACEs.

It is well known that CHR patients report higher stress levels than first-episode psychosis patients (Pruessner et al., 2011). Hence, reducing stress and improving coping is a critical goal for developing preventive interventions for schizophrenia.

New research areas are beginning to focus on the natural plasticity of the HPA axis during adolescent development to implement effective interventions that promote resilience to stress during the prodromal phase. For example, cognitive-behavioral therapy approaches help people to re-evaluate the interpretation of stressful stimuli more adaptively. Behavioral stress reduction techniques teach people to take an active role in reducing their exposure to stressors. Family therapy reduces stressful interactions and increases family support. Cognitive training exercises improve the accuracy and rationality of processing cognitive and socio-affective stimuli and increase the brain's "cognitive resilience" to data in its environment.

3.3.5 Neuronal alterations' due to childhood maltreatment

Child sexual abuse at critical stages of the development carries with substantial alterations at neuronal function, and therefore structural, since abuse and neglect are, for the biological system, a stressful event chronic, these affect the current and subsequent operation of the central and peripheral nervous system and the individual (De Bellis, 2005; Mesa-Gresa and Moya-Albiol, 2011; Amores-Villalba and Mateos-Mateos, 2017).

A previous study showed that exposure during sensitive child periods resulted in a steep dose-response function. The severity of adversity experienced at age 10–11 contributed to larger right but not left amygdala volume in adulthood. These results provide preliminary evidence that the amygdala may have a developmentally sensitive period in preadolescence (Pechtel et al., 2014).

Early child abuse could be inhibiting neurogenic processes, causing a decrease in gray matter in the hippocampus, in ventral, rostral and dorsolateral areas of prefrontal, in the insular cortex, and in the pre-limbic area, as well as delays in myelination of the white matter, which affects neuronal connectivity and hypo functionality (Kirsch, 2021). Also, alterations in the normal process of neuronal pruning can be observed during critical periods (De Bellis, 2005; Mesa-Gresa and Moya-Albiol, 2011).

Neurobiological correlates on mental disorders in women with a history of child abuse or maltreatment is expected since the more significant the child abuse, the greater the increase in prodromal symptoms in adult life (Kirsch, 2021). Previous studies have reported a lower hippocampal volume in adult women with major depression, which was found exclusively in those women with a severe and prolonged history of sexual or physical abuse in childhood (Vythilingam et al., 2002). However, these deficits and neurobiological alterations may not be evidenced during childhood, but in adult life, there is a failure to develop the individual's neurocognitive potential (Capilla et al., 2007).

3.3.6 Child maltreatment and the hypothalamic-pituitary-adrenal axis (HPA)

The hypothalamic-pituitary-adrenal axis (HPA) is the main signaling pathway activated in response to stress and trauma.

Suppose the cerebral cortex interprets environmental stimuli as dangerous. In that case, it communicates to the amygdala, which projects signals to the paraventricular nucleus of the hypothalamus, resulting in a Corticotropin-Releasing Hormone (CRH) and Arginine Vasopressin Protein (AVP). Already bound to their receptors (CRHR1 and AVPR1A, respectively), they activate the release of adrenocorticotropic hormone (ACTH) by the pituitary gland; this hormone acts on the adrenal cortex, releasing glucocorticoids such as cortisol, in humans, to the blood system (Lupien and McEwen, 1997; Rivier and Plotsky, 1986; Šimić et al., 2021).

Cortisol exerts adaptive functions to respond to adverse biological stimuli due to adverse experience, such as glucose mobilization, increased muscle activity, and cardiovascular tone, and gastric acid secretion. Once the dangerous situation has passed, cortisol itself exerts a negative feedback mechanism to return to the basal state and restore the body's homeostasis; however, the repetition of the event generates a sensitization of the CRH regulation system, and therefore, the feedback system is altered. The persistent sensitization of this system constitutes a biological substrate to develop a greater vulnerability to suffering alterations in response to subsequent stress, damage to the cognitive-emotional system, and consequently, acquire a long-term mental disorder (Cerda-Molina et al., 2017).

It is well-knowing that early violence and chronic stress induce an altered response of CRH secretion and other neurotransmitters involved in the reward system. Regarding the alteration of cortisol reactivity (CR) in cases of early violence, previous studies confirm that this persistent activation of the HPA axis during childhood leads to post-traumatic stress disorder (PTSD), depressive and anxiety episodes (Juruena 2014). Adult women who suffered from depression and had been victims of sexual abuse in childhood showed hyperreactivity of the HPA axis and, therefore, increased cortisol secretion in an acute stress test. In contrast, women who suffered abuse but did not present depressive symptoms had a moderate cortisol secretion pattern. In contrast, Juul et al. (2016) described those adult women with reports of sexual abuse and other trauma during childhood decreased their CR levels.

Improper or prolonged activation of the HPA axis is energetically costly and has been associated with numerous physiological and psychological states of illness (Danese and McEwen, 2012). Some results suggest that it is possible to consider alterations in cortisol secretion as a biomarker for designing interventions and pharmacological treatments for people suffering from PTSD and depression because of early violence (Cerda-Molina, 2017).

3.3.7 Allostatic load and mental pathology

Derived from the allostasis concept, McEween and Stellar (1993) developed the concept of Allostatic load. This concept links the cost-of-living chronic stress and the probability of developing certain diseases. *The allostatic load* (AL) can be defined as " The wear and tear of the body in the face of sustained resistance to chronic stressors, such as the presence of frequent adverse events." Hence, this concept reflects the impact of life experiences and a series of biopsychosocial factors of reference and response that generate patterns of behavior and physiological reactivity for life. The allostatic load causes physiological changes that eventually lead to biological or mental illnesses. This perspective offers many opportunities to handle affected people through medical approaches and psychosocial strategies, helping them restoring the baseline state (before physiological stress activation), emphasizing general health, fitness, and control of thoughts and perceptions.

Some research confirms that high levels of allostatic load in adulthood are related to adverse childhood experiences, including child abuse and maltreatment (Carroll et al., 2013). Studies focused on adults with PTSD after suffering early life traumas revealed a relationship between neuroendocrine biomarkers of allostatic load and early life stress and subsequent PTSD development (Thayer et al., 2017).

The primary mediators of the allostatic load or overload are cortisol, dehydroepiandrosterone (DHEA), epinephrine, norepinephrine, because of their immediate correlation with adrenal function (Seeman et al., 1997;2001). Considering these biomarkers along with others such as body mass index, cholesterol, triglycerides, among others; authors can calculate the rate or index of allostatic load. This index has been positively associated with patients at first-episode psychosis and multiple-episode schizophrenia patients, and with more severe manic symptoms (Berger et al., 2018a, b); the former study also found that AL index predicted lower social and occupational functioning (Berger et al., 2018a).

A recent study showed that the rate of allostatic load was significantly higher in patients with psychosis. This association was greater linked in those with childhood trauma and sexual abuse (Piotrowski 2020). Authors also concluded that the greater belief of social support and social interactions following adverse events could alleviate the biological deregulations associated with trauma during childhood.

3.3.8 Schizophrenia

Schizophrenia is a highly heritable disorder (80% to 85%) (Brown 2011). Genetic factors interact with environmental injuries, which begin in the pre and perinatal period, increasing risk and vulnerability to several environmental stressors. Although the prevalence of this disease is equal between men and women, it is women the most affected by the burden of life stress, which could increase the risk of disease, especially in developing countries (Thara and Kamath, 2015).

This interaction between genes and the environment leads to aberrations in brain development and the origination of neural networks, which will become evident until adolescence or early adulthood, when brain maturation is almost complete (Andreasen 2010; Hoffman and McGlashan 1997). Cognitive models of positive and negative symptoms of schizophrenia could link genetic vulnerability, early experience, and environmental stressors later in life with schizophrenia upset (Batinic, 2019). In the beginning, the individual experiences a break in reality and psychotic symptoms, almost always t in a mild or attenuated manner, but if they do not receive the treatment, it will progress to a psychotic episode. Schizophrenia can be considered a neurodevelopmental disorder characterized by decreased efficiency and regular connectivity in cortical and subcortical neural networks. This fact makes them particularly vulnerable to the harmful effects of stress. It leads us to understand how interventions aimed at an adaptive response to vitally stressful stimuli or how treating the abnormal processing of the nervous system can improve the early course of schizophrenia.

In normal and healthy neural development, peak synapse proliferation and peak synaptic density occur at approximately two years of age, followed by a decrease in this synaptic density during childhood and a decline in adolescence (Spear, 2003). This process has been defined as synaptic pruning, which is especially essential in the prefrontal cortex regions, to increase the efficiency of neural processes and neurocognitive functions (Spear 2003). Once the synaptic pruning in the prefrontal cortex has been carried out, a reestablishment of the functions happens, such as an increase in the ability to solve abstract and complex problems, increased reasoning, planning, flexible thinking, emotional control, among other executive functions (Giraldo-Chica et al., 2018; Spear 2003). Several studies suggest that excessive pruning or abnormalities in the pruning process are related to the establishment of psychosis in adolescence (Andreasen et al., 2011; McGlashan and Hoffman 2000); for example, individuals with schizophrenia exhibit decreased dendritic spine density, but the number of neurons is intact (Glantz and Lewis 2000; Selemon et al., 1995). Specific genes are known to contribute to abnormalities in synaptic pruning and synaptic functioning in schizophrenia. However, the primary effect is a reduction in the volume of gray matter in cortical and subcortical regions key to the development of the disease (frontal and temporal areas) (Fusar-Poli et al., 2012).

Another critical process is the increase in density in white matter. Beginning in adolescence and continuing into early adulthood, the hippocampus and frontal lobe undergo a substantial myelination process, mainly driven by brain mechanisms of experience-dependent plasticity (or simply, adaptive learning). Recent evidence suggests that this process is disrupted in schizophrenia; the net result is that decreased white matter density hinders the rapid and efficient integration of information processing within and across cortical zones and contributes to cognitive decline (Dwork et al., 2007).

In a longitudinal study covering the changes in brain volume over time in a cohort of patients with schizophrenia, the results confirm a decrease in several regions of gray matter and white matter, which is more pronounced two years after the first episode of psychosis (Andreasen et al., 2011).

Cascade alterations in the functioning of neural networks in schizophrenia are evident, including a reduction in synaptic density and reducing neural integrity; all may result from one or more abnormalities in brain plasticity mechanisms, leading to maladaptive changes (Balu and Coyle, 2011). The reduced functional connectivity between crucial nervous systems and reduced efficiency in their socio-cognitive operations suggests that clinical attention must be focused on correcting this abnormal functioning. If possible, before the individual experiences maladaptive changes that can be irreversible to improve the picture of schizophrenia.

3.3.9 The impact of stress and early trauma on the development of schizophrenia

A stressful environment interacts with neurocognitive vulnerability, increasing the risk that triggers the establishment of psychosis (Walker and Diforio 1997; Nuechterlein and Dawson 1984). Many studies show an association between the magnitude of the stressor and psychosis in individuals with schizophrenia; evidence of the link between stress and attenuated psychosis during the prodromal phase continues increasing. The main proposed mechanism that outlines this relationship is the dysregulation of the hypothalamic-pituitary-adrenal (HPA) axis, which turns out to be the central stress response system in mammals.

In this line, recent research has found the interaction between mineralocorticoids (MR) and glucocorticoids receptors (GR) with genes implicated in neuroplasticity processes, therefore, in learning and memory. Moreover, some of these genes are also involved in mental disorders such as schizophrenia, among others (Mifsud et al., 2021).

Chronic stress and the elevation of glucocorticoids in response to stress can cause stress sensitization, resulting in a reduction of hippocampal neurons, glucocorticoid receptors in the hippocampus, and a suppression of long-term potentiation (LTP) (Pavlides et al., 1993; Sapolsky 1985; Sapolsky et al., 1990; Stein-Behrens et al., 1994), which impairs the consolidation of declarative memory. Chronic stress damages hippocampal functions and increases and conditions thoughts about threats that affect the amygdala, which persist after recovery from stress (Conrad et al., 2004; Vyas et al., 2004). In animal models, prolonged stress causes dysregulation in the negative biofeedback circuit, whereby the damaged hippocampus fails to properly modulate the activity of the HPA axis (Meaney et al. 1989; Sapolsky et al. 1990) and increases glutamate both in the prefrontal cortex as well as in the hippocampus, which can result in altered levels of dopamine (Moghaddam 2002).

Many studies have verified dysregulation of the stress response system in schizophrenia, primarily using cortisol assays to access the integrity of the HPA axis (Walder et al., 2000). Salivary cortisol is related to symptom severity in a sample of patients with schizophrenia, affective disorders, or no history of mental illness; for example, baseline cortisol and ACTH levels are higher in newly diagnosed chronic schizophrenia patients than in healthy subjects (Bradley and Dinan 2010). Some studies suggest that the stress response and the functioning of the HPA axis are dysregulated during the prodromal phase of schizophrenia, which is related to the level of symptoms and the transition to psychosis (Aiello et al., 2012; Holtzman et al. 2012). In a study with adolescents with schizotypal personality disorder, salivary cortisol levels were associated with the severity of schizotypal symptoms and transition to psychosis at a 12–24-month follow-up (Walker et al., 2001; Walker and Bollini 2002). Salvaging the concept of High clinical Risk (HCR), Yee et al. (2007) found that patients with High Clinical Risk (HCR) showed higher basal cortisol levels than healthy controls. They also found that, after a social stress test in the laboratory, the magnitude of the cortisol response of patients with schizotypal personality disorder was lower than in controls, but they showed a response to stress with a delayed recovery pattern of cortisol compared to controls (Yee et al., 2007). Other studies have also shown that over-activation of the HPA axis is more common during the prodromal phase (Büschlen et al., 2011; Garner et al., 2005; Pantelis et al., 2009).

Interestingly growing evidence has documented higher rates of childhood trauma in patients with schizophrenia than controls, suggesting an environmental influence that increases the stress response in schizophrenia (Matheson et al., 2013; Sideli et al., 2012). The relationship between early adversity due to trauma and later psychiatric symptoms has been well investigated for depressive disorders and substance abuse (Cerda-Molina et al., 2017; Laucht et al., 2013).

3.3.10 Public Policies in México for the prevention and care of early trauma

Violence is a public health problem, with severe negative consequences and alterations in the mental and physical health of the population, especially of the female gender (Krug et al., 2002). Women who have suffered abuse in childhood are up to four times greater risk of developing symptoms of depression in adult life (Heim et al., 2000); furthermore, the magnitude of the trauma correlates significantly with the severity of depressive symptoms (Mullen, et al., 1996).

An agenda of attention to child abuse is urgently needed since spending on care and treatment of depression, and anxiety costs the world economy 1 trillion US$ a year and, according to the WHO, common mental disorders are on the rise throughout the world. Between 1990 and 2013, the number of people with depression or anxiety has increased by 50%, from 416 million to 615 million. About 10% of the world's population is affected, and mental disorders account for 30% of the global burden of non-fatal disease. Humanitarian emergencies and conflict increase the need to expand therapeutic options. The WHO estimates that 1 in 5 people is affected by depression and anxiety (WHO, 2016). It is a fact that countries should invest in rigorous national studies on the prevalence of child maltreatment and obtain detailed information on its nature (Mathews et al., 2020); this can help improve treatment and prevention strategies (Lippard and Nemeroff, 2020). Child sexual abuse continues to be a severe public health problem in all countries worldwide, as confirmed by the prevalence data. The terrible consequences that child sexual abuse tends to have outweighed the necessary efforts required in the changes and adjustments of public policies (Castro et al., 2019). The adjustments include the prevention of child abuse or neglect, early detection with the use of instruments with high sensitivity and specificity, and timely treatment. Mental health professionals need to recognize that childhood trauma is directly associated with the presence and severity of psychiatric symptoms, attempts, and ideation suicide, especially in women. Finally, optimal treatment of psychiatric patients must consider the history of child abuse and alterations in the stress response. The proposal implies that the Mexican health system invests in studies of the prevalence of early trauma and recognizes the crucial role in the presence and severity of mental disorders. This latest could help implement effective care strategies to compensate for the allostatic burden that already exists, mainly when it affects women, who become potential caretakers and abusers.

3.4 Conclusions

Early trauma carries severe adverse consequences for the mental health of individuals, especially for women, who have up to four times the risk of suffering from a mental disorder if they have a history of sexual or physical abuse in childhood. Allostatic load promotes inadequate responses to acute stressors. This condition would be a consequence of constant exposure to chronic stressors, mainly if the experiences occurred in childhood (before 15 years old). The younger the individual, the greater the neurological damage and the greater the risk of suffering from a psychiatric disorder. Some alterations are only visible until adulthood, when the individual presents misadjusted behaviors in the work, social, and family spheres; presenting, in addition, violent or negligent behaviors that perpetuate the chain of adverse events in the minors in their care. The research focused on the prevalence of child abuse in Mexico, its consequences, and regulatory factors are essential and influence public policies for prevention and offering optimal treatment to psychiatric patients who consider the history of childhood trauma, especially in women.

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