Chapter

Borderline Personality Disorder and Childhood Trauma: The Posited Mechanisms of Symptoms Expression

Maria Uscinska, Nicolo' Gagliano, Andrea Polla Mattiot and Silvio Bellino

Abstract

Traumatic events are reported in a large percentage of the population, however, only in some individuals it will lead to a diagnosable trauma-related disorder. Borderline personality disorder (BPD) is deemed to be a form of acute reaction to childhood trauma. Therein experiences of childhood abuse and neglect take on an important etiological role, generating severely disorganized attachment relationships, which in turn affect the development of emotional regulation systems, and significantly inhibit the development of mentalization and metacognitive skills. Furthermore, the last decade has seen important contribution of neuroscientific research in shedding light on the neurobiological correlates of traumatic experiences. A wealth of scientific literature links the onset of BPD to the combination between genetic and environmental factors (G×E), in particular between biological vulnerabilities and the exposure to traumatic experiences during childhood. Although no research can predict with certainty which trauma will translate into symptoms, there are indications as to who is more at risk of developing a trauma-related disorder. Herein we describe the psychological and epigenetic mechanisms affected by childhood trauma and altered in BPD patients.

Keywords: borderline personality disorder, childhood trauma, psychopathology

1. Historical and conceptual overview

The concept of trauma first entered the DSM-III as a rare, catastrophic stressor outside the range of usual human experience, apt to evoke significant symptoms of distress in most people [1]. To date, it is deemed a silent epidemic [2] defined in DSM-5 as a result of either direct or indirect exposure to actual or threatened serious injury or sexual violence, death or a threatened death [3]. Meaning 'stroke or wound' in Greek, in psychopathology trauma refers to a lesion or an insult to the psychic organism induced by a stressor or a series of noxious events that occur suddenly and in a disruptive way in a subject's life [4].

The impact of trauma on the human psyche has received considerable research attention, starting with the thesis that it is a process taking place within the attachment relationships and separation [5]. Owing to the advent of neuroimaging techniques it is well-established that human experiences, whether traumatic or therapeutic, have

measurable influences on the brain structure and function. As harmful pathways are etched deep into the brain following an exposure to trauma, neuroimaging studies allow to index the extend of associated cerebral damage. Therein, the level of maternal support in childhood was shown to determine hippocampus volume in adulthood [6], whilst childhood poverty was associated with reduced white matter, cortical gray matter, and hippocampal volume [7]. In some cases, the progressive impact of traumatic experience may lead to a diagnosis of debilitating psychiatric disorders such as BPD, complex PTSD or disorders of extreme stress, not otherwise specified (DESNOS) [8]. A wealth of research on neuro-functional alterations associated with trauma-exposure revealed patterns of increased amygdala activity in response to threatening stimuli, and simultaneous decreased activity in prefrontal areas of the cortex that downregulates the amygdala [9–11], as well as hyperactive hippocampus [11]. Furthermore, recent evidence has shown that trauma experience is associated with altered functional connectivity between the amygdala, and medial prefrontal cortex (mPFC), insula, and dCCA [10, 12], furthering the thesis of an interplay between prefrontal regions and limbic structures. With this in mind, the next section follows with an overview of trauma-related psychiatric disorders. In keeping with research trends in developmental neurosciences the main theme of the chapter is pathological pathways from trauma to BPD, focusing on trauma-induced alterations in neurobiological systems.

2. The interface between trauma and mental illness

Mental illness is often broadly defined, encompassing disturbances across domains of functioning in the emotional, cognitive, and/or behavioral realms. Accordingly, the biopsychosocial model of disease causation and treatment brought about a new way of conceptualizing mental health difficulties resulting from biological, psychological, and social factors [13]. In this view, adverse life events interact with genetic susceptibility, personality and social context to co-determine individual vulnerability to clinical expression of mental illness, its severity and course. In quest to unravel how different factors and processes translate into a psychiatric disturbance, researchers and clinicians have sought to understand the progressive and developmental impact of trauma experience on an individual. A wealth of research reveals that exposure to adversities, stressors and neglect can chronically and pervasively alter biological, cognitive, psychological, and social development, giving rise to disturbances in impulse and affect regulation, alterations in attention, consciousness, attribution and schema, as well as interpersonal difficulties [14–16]. On the neurobiological level, exposure to trauma triggers a surge of neurochemical factors, potent enough to interfere with integrative capacity and the ordinary process of neurodevelopment [17, 18]. Therein appraisal of adverse events and associated emotions might give rise to the stress response, prompting a cascade of biological events that alter various essential processes, namely neurogenesis, synaptogenesis, migration and neurochemical differentiation. Indeed, research exists to support that a persistent traumatic event might induce a permanent neurobiological modification of the subject's stress response [19] evidenced in elevated urinary epinephrine, norepinephrine, and dopamine, increase or decrease in baseline heart rate, alterations of alpha-2-adrenergic receptors, limbic and cortical abnormalities, and altered development of some cortical areas [18]. Elevated stress in turn has been shown to downregulate the process of hippocampal neurogenesis [20]. Notably, peritraumatic stress reactions within the first hours are predictors of the development of a trauma-related disorder, and therefore constitute a critical window for interventions for prevention of trauma-related disorders [21, 22]. This is presumably due to the fact that memory consolidation occurs during the first night's sleep following the exposure [23]. Conversely, it has been shown that

an adequate resilience capacity, that is the ability to adapt to an adverse situation is a crucial protective factor against the occurrence of a trauma-related disorder [24].

3. Trauma-related disorders in DSM-5

Unlike previous editions, DSM-5 has introduced a number of modifications, regrouping disorders that appear to be etiologically related to one another [3]. In this view, trauma- and stress-related disorders are separated from anxiety disorders and obsessive–compulsive disorder, and classified in a specific chapter clustering disorders characterized by the occurrence of one or more traumatic or stressful events in which the subject is involved. The cluster includes Post-Traumatic Stress Disorder (PTSD), Acute Stress Disorder (ASD), Attachment Disorders, Disinhibited Social Engagement Disorder (DSED) and Adjustment Disorder. Whilst Attachment Disorders and DSED originate in childhood, the remaining diagnoses refer to trauma experience in the adult population [3].

PTSD is the most complex disturbance of the cluster, characterized by intrusive re-experiencing symptoms and avoidant, numbing, and hyperarousal symptoms that manifest themselves after an insult to the person's physical integrity or event that has caused a serious injury to the subject or to the other close to him/her [3, 18]. Unlike previous versions, DSM-5 lists specific criteria of PTSD symptoms for patients under 6 years of age, which may not be particularly manifest or verbally expressed, nevertheless a careful observation of the child's behavior can identify the impact of trauma on interpersonal difficulties, insecure disorganized attachment style, or episodes of aggression and difficulty in affective regulation [25].

ASD encompasses a broad array of immediate, transient reactions to a sudden impact of trauma that typically subsides within 48 hours. The disorder is characterized by intrusive memories, negative mood, dissociation, avoidance, and/or hyperarousal experienced during the first month after a traumatic event. Since the introduction of ASD into DSM-IV in order to identify those at risk for developing PTSD, concerns have been raised whether the diagnosis reliably predicted PTSD and whether it pathologized normal reactions to trauma. Furthermore, little empirical evidence exists to support the thesis that an individual with ASD endorses at least three dissociative symptoms. In response to this criticism, the ASD diagnostic criteria were changed with the publication of DSM-5 excluding the dissociative symptoms requirement resulting in a stronger predictive power.

Adjustment disorder has been described as the linchpin between normalcy and psychiatric disturbance as it characterizes a severe emotional reaction to an identifiable stressor that does not meet criteria for other more specific disorder [26]. The symptoms can vary among diagnosed individuals and include hopelessness, anhedonia, sadness, irritability, sleep problems, avoidance, diminished performance, aggression and so forth.

Attachment disorders are characterized by a disrupted attachment related to early social deprivation, maltreatment or neglect and are differentiated into disinhibited and inhibited or reactive attachment disorders. Whilst the former is associated with the absence of early attachment relationships and indiscriminate sociability, the latter is etiologically linked to neglect and abuse.

4. Trauma and borderline personality disorder

The interface between childhood trauma and Borderline Personality Disorder (BPD) has been a topic of discussion and controversy in clinical research. This

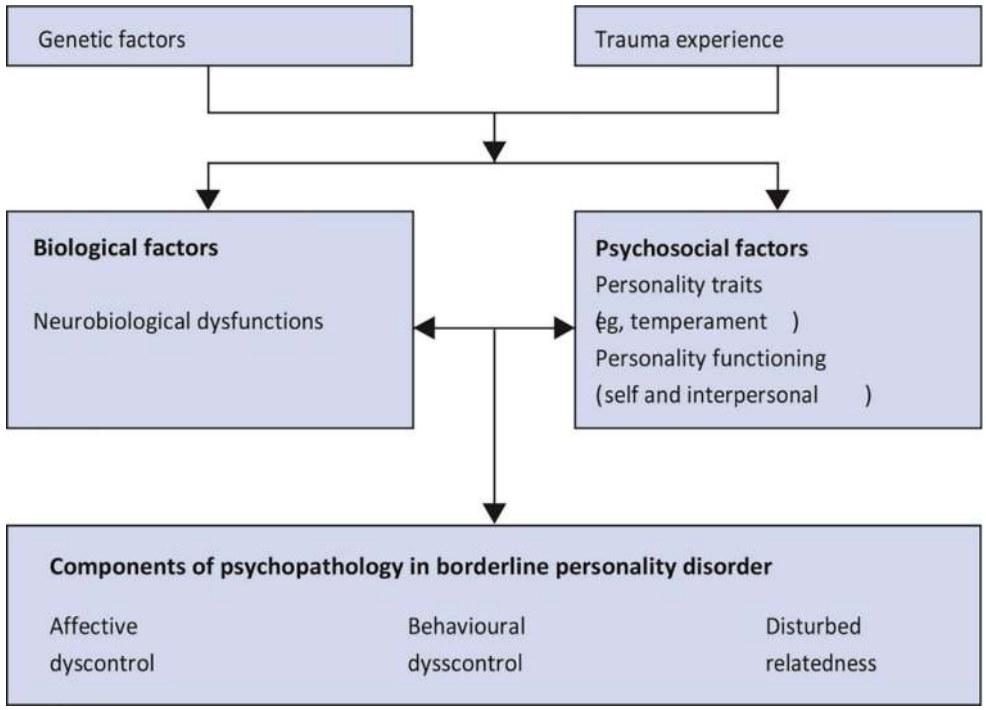

Figure 1.
The biopsychosocial model of borderline personality disorder.

severe psychiatric disorder is characterized by a pervasive pattern of instability in affect regulation, impulse control, self-image, cognition and interpersonal relationships [3]. Frequent self-damaging and impulsive behaviors, such as suicide, self-harm or substance abuse exacerbate the severity and morbidity of the disorder [27, 28]. Although the diagnosis of BPD does not require a history of traumatic event, childhood trauma is considered the main environmental factor contributing to the etiology and severity of the disorder [29–31]. By way of example, in a prospective study 500 children who had suffered physical and sexual abuse and neglect were found to be significantly more likely to meet criteria for BPD in adulthood than matched controls [32]. Although childhood trauma does not always lead to psychopathology [31] there is an empirical consensus that the interaction between childhood trauma and temperamental traits constitutes the basis for the etiology and severity of BPD [33, 34]. Accordingly, the biopsychosocial model of BPD (**Figure 1**) posits that the disorder results from the interaction between biologically based temperamental vulnerabilities and adverse experiences in childhood [35–38].

5. Clinical pathways from trauma to BPD

5.1 Psychodynamic psychopathology and attachment

From the standpoint of contemporary clinical psychology there are three perspectives firmly rooted in the attachment theory that best describe the complex psychopathology of BPD, namely the processes of mentalization, the theory of interpersonal motivational systems and the model of affective regulation.

5.1.1 Mentalization

A trauma within the bond of attachment goes to generate a repeated and continuous activation of this system. Whilst looking for closeness of an attachment

figure that has been traumatizing, the child will reencounter traumatic experiences. The prolonged activation of the attachment system will produce specific inhibitory responses to the mentalization, as well as those related to the physiological increase of emotional arousal. The child, in an attempt to obtain some form of control over the aggressor, might try to identify with the aggressor, internalizing the intent of the aggressor and thus generating a part of the dissociated self, so-called the alien Self.

Over time, the destructive intent of the abuser will be perceived as coming from within own self and not from another person, leading to experimenting a strong sense of hatred towards oneself by child that might persist to adulthood. To deal with severe trauma and abuse a defensive operation to avoid the reflection on the content of the caregiver's mind is triggered, thereby preventing the resolution of the experiences of abuse [39].

This mechanism explains why BPD patients frequently have relationships in which they feel victimized by others perceived as persecutors. Through the defensive process called projective identification, a patient might attempt to "force" a significant person, including the psychotherapist, to assume the character of the "Alien Self/Bad Object".

5.1.2 Attachment and other interpersonal motivational systems

In accordance with the theory of attachment, the experience of ill-treatment in the evolutionary age can have a strong negative impact on the development of the models of representation of the Self, of the figures of attachment and of the relationship between them.

The theory of interpersonal motivational systems [40–42] identifies a series of systems similar to that of attachment, which are also evolutionary-based, and support individuals towards some fundamental goals of existence such as defense, attachment, care, social rank, sexuality, cooperation, predation. According to Liotti, the disorganization of attachment involves both a multifaceted and split representation of oneself and the figure of attachment, and a metaphysical deficit that makes affective regulation difficult. The structuring of an internal disorganized operating model with perceptions of the multiple self-object relationship, inconsistent and not integrated, it seems explainable through the simultaneous and incompatible activation of the attachment and defense system in the child, as the caregiver deputed to respond to the demands of the attachment system is also the figure activating the defense system endangering the child's personal life and safety. Once they reach adulthood, when traumatic memories emerge in the mind of the parents, the pain associated with them activates the attachment system of the figures that should provide care, thus evoking feelings of anger and fear in the caregiver when care responses are needed by the baby.

5.1.3 Affective regulation

The theory of affective regulation [43–47] focuses more on the psychobiological or "primary" aspects of the affective experience rather than cognitive-affective aspects of the experience as indicated in the work of Fonagy and colleagues.

Incorporating contributions of the clinical tradition of psychology of the Self [48] and contemporary infant research [49, 50], the theory proposes the process of tuning, defined as the synchronicity of affective states as the foundation of caregiving practices and the most compromised aspect in the relationships of attachment. Failure to tune between the child's affective states and the caregiver prevents the affective adjustment process within the 'tolerance window' which constitutes the ideal metabolic conditions for neuronal metabolic development.

5.2 Cognitive psychopathology

Beck and collaborators [51] have provided a conceptualization of the BPD according to the cognitive-behavioral perspective, which posits three main dysfunctional nuclear convictions at the basis if the disorder, namely the world is dangerous and malicious, I am powerless and vulnerable, and I am inherently unacceptable. The first two convictions tend to produce a sense of hypervigilance and distrust of others. There are two other central cognitive features: a dichotomic thought and a poorly articulated self-pattern, which determine a weak sense of personal identity. These aspects contribute to creating the emotional and interpersonal behaviors typical of this disorder.

Within the schema therapy framework Young and colleagues [52] stress that borderline subjects tend to present too many schemas and to oscillate between extreme affective states, and it is in the excess and overlap of reference schemas that the symptomatology of the BPD resides.

Dialectical Behavior Therapy (DBT) posits at the center of the borderline disorder a deficit of the system of regulation of the emotions with its consequences among which are: understanding the contextual, relational and transient nature of emotions; building an effective mental model of the relationships between emotions and environmental events; and semantically labeling each emotion properly [53]. This deficit is deemed to be the product of temperamental characteristics, such as emotional vulnerability, which interact with a disabling environment of the person's emotional experiences.

According to the cognitive constructivist model proposed by Dimaggio and Semerari [54] BPD is attributable to specific functional deficits of metacognition capabilities, detectable in the following functions:

- deficit of integration, where different mental states are experimented without reciprocal memory (hence, the tendency to oscillate between them in little chaotic and rapid modulated);

- deficit in emotional regulation, caused by a genetic-temperamental vulnerability that results in hyperreactivity to stimuli, combined with environmental-disabling experiences;

- deficit of differentiation between reality and its representations as a consequence of affective dysregulation.

The relationship between these deficits is manifold, where dysregulation and integration difficulties affect each other, leading to a lack of differentiation.

Conflict of interest

The author has no relevant affiliations or financial involvement with any organization or entity with a financial interest in or financial conflict with the subject matter or materials discussed in the manuscript. This includes employment, consultancies, honoraria, stock ownership or options, expert testimony, grants or patents received or pending, or royalties. No writing assistance was utilized in the production of this manuscript.

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