The complex interrelation between dissociation and borderline personality disorder: A review of the role of responses to aversive events

Dana BICHESCU-BURIAN[1]

Abstract: Borderline personality disorder (BPD) is a serious mental disorder associated with extensive psychopathology and treatment utilization. Pathological dissociation belongs to the central features of this disorder. Severe dissociative symptoms and disorders among BPD patients pose serious obstacles against effective treatment. Delivering meaningful treatment strategies requires extension of etiological knowledge. Recent findings in clinical and neurophysiological research point to the significant role that trauma-related dissociation among aetiological factors of BPD. This paper analyses evidence and discuss aetiological relevance concerning the role of adverse antecedents in the occurrence of pathological dissociation and other clinical problems in BPD. After several introductive remarks on the psychopathology and functioning in BPD and on the concept of dissociation, I review findings on the clinical significance of pathological dissociation in BPD. Subsequently, I present evidence regarding the association between adverse events and dissociation in general and the pathogenic role of trauma-related dissociation in BPD. Finally, I present several theoretical models attempting to explain the role of trauma-related dissociation in the aetiology of BPD.

Key words: Pathological Dissociation, Borderline Personality Disorder (BPD), Aversive Events, Peritraumatic Dissociation, Clinical and Neurobiological Findings

Introduction

BPD is a serious mental disorder associated with extensive pathology, comorbidity, mortality, treatment utilization, and public health costs (Bohus, 2007). It is characterised by a severe pattern of instability of affect self-image, and social relationships as well as significant impulsivity (Lieb, Zanarini, Linehan, & Bohus, 2005). Pathological dissociation has been described as a fundamental feature of BPD (Zanarini, Ruser, Frankenburg, Hennen, & Gunderson, 2000; Zweig-Frank & Paris, 1997). Severe dissociation is clinically considered an indicator of high psychopathology. Such facts highlight the high clinical and health policy relevance of BPD. Yet etiological knowledge is currently still insufficient to understand BPD and guide its treatment (Paris, 2009). The need for further investigation of factors underlying disorder-specific comorbidity has been particularly emphasized (Grant et al., 2008).

Dissociation as a concept has been addressed in very different ways. Dissociation is generally viewed an altered state of consciousness characterised by

[1] University of Ulm, Germany; Knowledge Based Society, Iaşi, Romania; DanaMaria.BichescuBurian@ZfP-Zentrum.de

lacking integration of cognition, emotion, and experience into the stream of consciousness leading to a reduced awareness of environmental events (Carlson et al., 1993; Foa, Keene & Freidman, 2000). Since dissociative phenomena comprise a large spectrum from common experiences such as absorption until highly pathological disintegration of memory and identity, we agree with the view of differentiating between pathological and non-pathological dissociation, which is sustained by research findings up to date (Spitzer, Barnow, Freyberger, & Grabe, 2006; Spitzer, Barnow, Freyberger, & Grabe, 2007). This paper aims at gaining insight as to how aversive events potentially lead to pathological dissociation during the developmental process of persons with BPD. To this end, I explicitly refer to well-defined notions of dissociation: peritraumatic dissociation (Marmar, Weiss & Metzler, 1998) and dissociative symptoms and disorders according to ICD-10. This paper focuses on the dissociative reactions to stress and pathological dissociation and presents clinical and neurobiological research findings on the association between BPD, severe pathological dissociation, and aversive events.

Dissociative symptoms and disorders in BPD

Transient stress-related severe dissociation is one of the BPD diagnostic criteria within the DSM-IV. Dissociative symptoms are highly prevalent in BPD (up to 76.2 %), as shown by larger epidemiological surveys (e.g. Korzekwa, Dell, Links, Thabane & Fougere, 2009; Stiglmayr et al., 2008; Zanarini, Gunderson & Frankenburg, 1990). Severe dissociative mechanisms in BPD disturb emotional and cognitive processing (Ebner-Priemer et al., 2009) and therefore may seriously impede successful psychological treatment. Indeed, clinical trials have identified dissociative and posttraumatic pathology in the aftermath of childhood abuse as high-ranking predictors for negative treatment outcome in BPD (Paris, 2009; Spitzer et al., 2006; Spitzer et al., 2007). Moreover, dissociative symptoms are very difficult to treat (Foa & Rothbaum, 1998). Clinical practice show that particularly in severely traumatised BPD patients, failures in treatment may aggravate dissociative states, leading to self-harm, suicide attempts and readmissions (Steinert, Tschöke & Uhlmann, 2010).

Dissociative pathology and BPD are complexly interrelated. Some BPD patients exhibit merely minor dissociative symptoms while others fulfil the criteria for one or several dissociative disorders (DDs; Eaton et al., 2010; Korzekwa et al., 2009). Unlike patients exclusively diagnosed with DDs, BPD patients with comorbid DDs are additionally characterised by increased morbidity and impairment (Dell, 1998; Horevitz & Braun, 1984). Dissociative identity disorder (DID) is diagnosed among 10% to 27% of the BPD patients (Ross, 2007; Sar, Akyuz, Kugu, Ozturk & Ertem-Vehid, 2006). Moreover, BPD is diagnosed in 30% to 70% of the DID patients (Dell, 1998; Horevitz & Braun, 1984; Sar et al., 2006). Based on such findings, Korzekwa et al. (2009) postulate the existence of several categories of persons with BPD: besides the highly impaired patients with most severe traumatic history and highly prevalent DDs and DID, there are also patients

with a disorganised attachment style and a significant traumatic history and others with rather minor dissociative symptoms and little adverse antecedents.
The present state of art with regard to the association between dissociation and BPD support the assumption that dissociative pathology in BPD is a psychological marker of more severe symptoms and impairments.

Self-harm in BPD patients is associated with a child abuse history and dissociative pathology

There is another body of evidence regarding the interrelation between dissociation and another specific psychopathological feature of BPD, namely self-harming behaviours. Up to date, ca. 70 % BPD persons use self-harming behaviours to reduce dysphoric states (Bohus et al., 2000).

Several studies have addressed the association between self-harming behaviour and dissociation in the general and clinical population samples (Briere & Gil, 1998; Feeny, Zoellner, Fitzgibbons & Foa, 2000; Gratz, Conrad & Roemer, 2002; Low, Jones, MacLeod, Power & Dugan, 2000; van der Kolk, Perry & Herman, 1991; Wachter, Murphy, Kennerley & Wachter, 2009; Zlotnick, Mattia & Zimmerman, 1999). In the study of Gratz et al. (2002), dissociation was the most significant predictor of self-harming behaviours. Several studies have consistently demonstrated that psychiatric patients with self-harming behaviours are characterised by higher levels of dissociation (Briere & Gil, 1998; Kisiel & Lyons, 2001; Low et al., 2000; van der Kolk et al., 1991; Wachter et al., 2009; Zlotnick et al., 1999). In both healthy persons and patients, self-harm is associated with childhood abuse and is apparently resorted to decrease dissociation, emotional distress, and posttraumatic symptoms (Briere & Gill, 1998).

Several studies on psychiatric patients with self-harming behaviours have demonstrated that the majority of these patients are having a BPD diagnosis and a history of childhood trauma (Stanley, Gameroff, Michalsen, & Mann, 2001; Starr, 2004; Goodman et al., 2010). Goodman et al. (2010) have shown that a higher score on pathological dissociation as measured by DES-T (Dissociative Experiences Scale – Taxon; Waller & Ross, 1997) was significantly associated with a diagnosis of BPD and with a history of self-mutilation and suicide attempt. Starr (2004) has pointed out that such behaviour is used as an attempt of emotional regulation.

Studies investigating self-harm among BPD patients (Brodsky, Cloitre & Dulit, 1997; Dubo, Zanarini, Lewis, & Williams, 1997; Zweig-Frank et al., 1994) have showed that such behaviour is associated with dissociation and a history of childhood abuse and neglect. Findings that BPD patients often experience self-harm as painless (Bohus at al., 2000; Kemperman et al., 1997) are also indications that it takes place in a dissociative state.

Taken together, previous findings indicate a strong association between dissociation and self-harming behaviours in general and in BPD patients in

particular (see Figure 1). Most studies indicate that dissociation is the strongest predictor of self-harming behaviours in persons with a history of childhood abuse and neglect. Moreover, dissociative BPD patients are frequently having a history of childhood abuse and are at particularly high risk for self-harm, severe clinical problems, and utilization of psychiatric treatment.

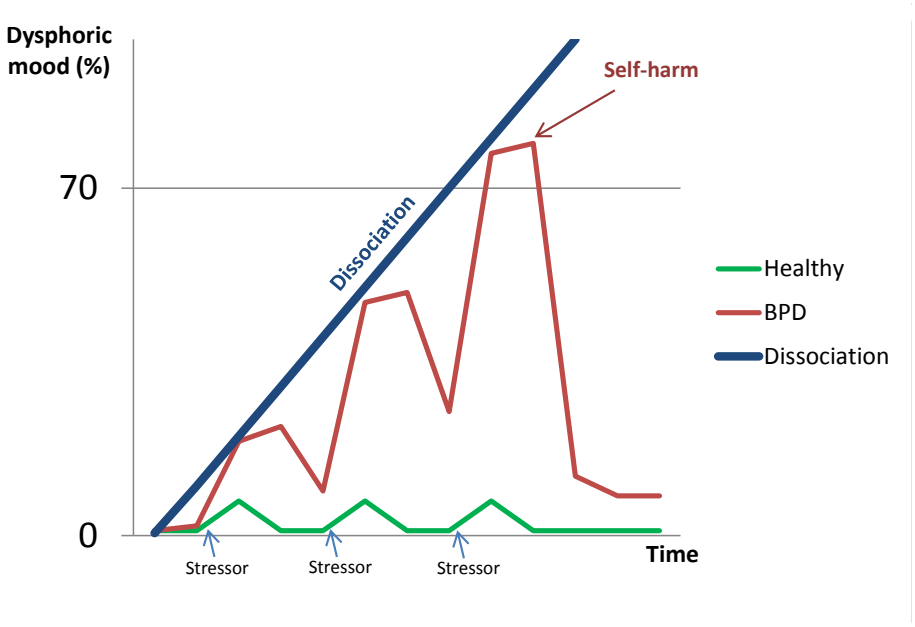


Figure 1. Schematic illustration of the BPD psychophysiological deficits of the emotional regulation in response to every day stressors as compared to healthy stressors and of the correlation between the level of dysphonic mood and dissociation as shown by previous findings (e.g. Stiglmayr et al., 2008).

The general interrelation between dissociation and aversive events: biological and clinical perspectives

The growing attention to the severe dissociative pathology comes from the awareness of the psychological sequelae of aversive events, especially chronic childhood abuse and sexual abuse (Butzel et al., 2000; Chu, Frey, Kisiel, & Lyons, 2001; Ganzel & Matthews, 1999; Low et al., 2000; Pearson, 1997). The probability of dissociative pathology increases with the prolonged and repeated exposure to trauma.

According to evolutionary theories, dissociative states are hard-wired biological defence mechanisms developed along with the classic Freeze-Flight-

Fight reactions. Although involving risks for bodily injuries, dissociation is the remaining survival strategy to reduce extreme affect and facilitate adaptive behaviour to inescapable threat by a superior aggressor (Sierra & Berrios, 1998). In neurobiological terms, dissociation arises when high anxiety causes the medial prefrontal cortex to inhibit emotional processing within the limbic system, thus reducing sympathetic output (Sierra & Berrios, 1998).

This evolutionary view of dissociation as a survival strategy in extreme situations complies with the records of peritraumatic dissociation among acute reactions to psychological trauma in form of automatic derealisation and depersonalisation experiences that occur during exposure to threat for purposes of emotional disconnect from distressing, aversive information (Marmar et al., 1998). While this detached state is adaptive in the short term, it may be highly impairing if it persists over time, as in DDs. Thus, the disintegration process of sensation, emotion, and cognition into the particular declarative memory during overwhelming threat may result in disorders of the trauma spectrum including BPD, posttraumatic stress disorder (PTSD), DDs and other disorders (e.g. Ehlers & Clark, 2000; Elbert, Rockstroh, Kolassa, Schauer, & Neuner, 2006; Marmar et al., 1998). There is substantial evidence that peritraumatic dissociation robustly predicts mental health problems including PTSD (e.g. Birmes et al., 2003; Ozer, Best, Lipsey & Weiss, 2003), as well as later dissociative symptoms and BPD (Sar, Akyuz, Kundakci, Kiziltan, & Dogan, 2004).

Evidence on the role of aversive events in the occurrence of dissociative pathology in BPD

A similarity between dissociative symptom patterns occurring in both BPD and PTSD patients has been noted. The concept of "complex PTSD" (Disorders of Extreme Stress, not Otherwise Specified, DESNOS) has been discussed as a comprehensive diagnosis for the appropriate classification of the consequences of severe neglect, emotional, physical and sexual violence seen in certain BPD patients (e.g. Herman, Perry & van der Kolk, 1989). A strong relationship between pathological dissociation and psychological trauma in BPD is empirically supported, with the level of dissociation as strongly predicted by early severe, repetitive trauma (e.g. abusive and neglect experiences; Bernstein & Putnam, 1986; Dutra, Bureau, Holmes, Lyubchik, & Lyons-Ruth, 2009; Gunderson & Sabo 1993; Herman et al., 1989).

BPD patients report numerous childhood adverse events (abusive experiences, neglect; Zanarini et al., 2002) and substantially more than patients with other personality disorders (Yen at al., 2002). High rates of comorbid PTSD ranging from 60% to 70% (Golier et al., 2003; Zanarini et al., 1998) are being reported in BPD. Developmental psychopathology research understands disturbances in emotion regulation and interpersonal problems as repeated

childhood trauma affecting cognitive, emotional, and social development (e.g. Cook at al., 2005). Anyway, no direct association between these adverse experiences and the psychopathological alterations in adulthood has been found (Fossati, Madeddu, & Maffei, 1999; Paris, 2007). Therefore, this association may be mediated by other factors such as dissociation. More recent findings (Wagner, Baskaya, Lieb, Dahmen, & Tadić, 2009) favour the interaction between genetic and negative environmental factors (e.g. adverse childhood events) as an aetiological supposition (Gabbard, 2005).

Neurobiological findings support the idea of trauma-related dissociation in BPD

A main neuropsychological finding on prolonged, severe traumatisation refers to their damaging effects on cognition, memory, and brain structures such as hippocampus and amygdala (Karl et al., 2006). With respect to the neurobiological correlates of peritraumatic dissociative reactions, an inhibitory effect of the limbic system on the emotional processing through the activation of the medial prefrontal cortex and a reduced psychophysiological reactivity determined by the predominant parasympathetic activation is assumed (Sierra & Berrios, 1998), as already mentioned above. This activation pattern has been already proved to appear during dissociative states in PTSD patients (Lanius et al., 2010).

In the area of BPD, recent research deliver evidence of similar affected brain areas in early traumatised BPD patients (Irle, Lange, Sachse, & Weniger, 2010) and similar modulation patterns of dissociative responding is by frontolimbic neural processing (Ebner-Priemer et al., 2009; Ludäscher et al., 2010). Ludäscher et al., 2010 have found that such activation is associated with lower pain sensitivity. This may also explain the interrelation between dissociation and self-harming in BPD. The study of Ebner-Priemer et al. (2009) has additionally revealed that BPD patients with high dissociative states exhibit diminished acquisition of differential aversive delay conditioning with respect to emotional aspects of learning. The review of neuropsychological studies on BPD (Irle et al., 2010) indicates that early traumatised BPD patients with chronic, severe DDs present with a particular structural feature of increased superior parietal cortices.

So far, findings on neurobiological correlates of dissociation in BPD favour the notion of trauma-related dissociation by two main findings: (1) a processing pattern consisting of parasympathetic activation and emotional modulation mediated by the inhibitory influence of the medial prefrontal cortex on the limbic system is characteristic of dissociative states and (2) early traumatised patients with chronic, severe dissociation exhibit a structural specificity consisting of increased parietal volumes.

Aetiological theories and models

The classical aetiological models of BPD have been elaborated by Linehan

(1996) and Bohus (2002), constituting the basis on which Dialectical Behavior Therapy (DBT; Linehan, 1996) has been elaborated. Figure 2 offers a synthetic presentation of these models.

Anyway, the etiological mechanism of early aversive events resulting in dissociative experiences associated with serious mental illness in adulthood remains indefinite in detail. Animal models have showed that deprivation experiences negatively influence (Brown & Bogerts, 2001) the neuronal and synaptic plasticity in the limbic areas resulting in disturbed social and emotional control and learning. Such negative influences of early stress-experiences are stable over time and they are supposedly mediated by epigenetic changes in gene expression (Champagne & Curley, 2009). This may explain the reduced resilience and impairment in managing even moderate stress situations (Oizl, Champagne, van der Veen, & Kloet, 2010; Stiglmayr et al., 2008).

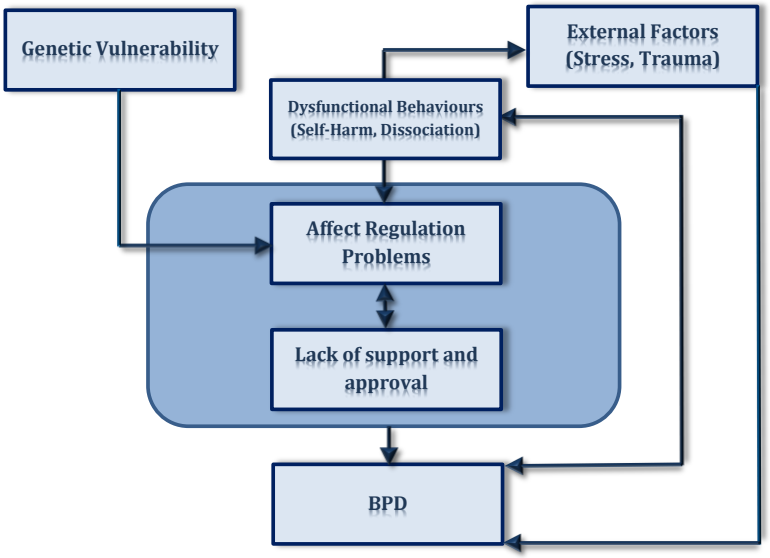


Figure 2. *Biosocial aetiological model of BPD (according to Bohus, 2002; Linehan, 1996).*

Schauer & Elbert (2010) have developed a biologically orientated model of trauma-related dissociation. This is a well-grounded evolutionary-based etiological model of trauma-related disorders that extends the conventional models of defence reactions in threatening situation through the incorporation of a sixth response,

namely "Flag" (see Figure 3). The six defence reactions[2] (''Freeze-Flight-Fight-Fright-Flag-Faint'') are distributed on 2 dimensions in form of an inverted U-shaped defence cascade, which progresses in grades of increasing (sympathetic arousal) and decreasing activation (parasympathetic dissociative responses). Schauer & Elbert (2010) argue that trauma-related responses (emotional, physiological and behavioural) form learned fear associations in the amygdala that are coded in memory as networks of reciprocally exciting components ("fear network" in Lang, Davis & Öhman, 2000). They facilitate prompt danger-focused perception and reaction, but can become pathologically detached from contextual cues (time and location of the danger). The concrete individual peritraumatic defence response will be re-enacted every time the trauma-related fear network is reactivated (i.e. through internal or external triggers, e.g. during exposure therapy), producing at least two major subtypes of clinical symptom profiles (hyperarousal versus dissociation) among persons with trauma spectrum disorders (e.g. PTSD, BPD).

[2] The defence responses have been repeatedly described within the biological and psychological literature (e.g. Bracha, 2004). **Stage 1 "Freeze"** (orienting response) is a "Stop, look, and listen" response (focused attention, and preparedness). Bodily responses are pupillary dilatation, motor inhibition, drop in heart rate. **Stages 2 and 3 "Flight" and "Fight"** (alarm response) consist of attempts to flee and to fight characterised by a discharge of the sympathetic autonomic nervous system and high arousal (increased heart rate, cardiac and muscular vasodilatation, faster breath). **Stage 4 "Fright"** (tonic immobility) consists of high alertness, unresponsiveness for reducing risk of injury and preparing a possible escape. It occurs in cases of direct physical and physical inferiority as compared with the aggressor. Sympathetic and parasympathetic are coactivated (increased muscle tonus, inhibition of efferent pathways). **Stages 5 & 6 "Flag" and "Faint"** (flaccid immobility up to fainting) characterised by marked parasympathetic activation and dissociation (failure of voluntary acts, derealisation, depersonalisation, emotional numbness, memory decline, disgust, reduced heart rate, low cerebral blood supply, declining central information processing and unresponsiveness).

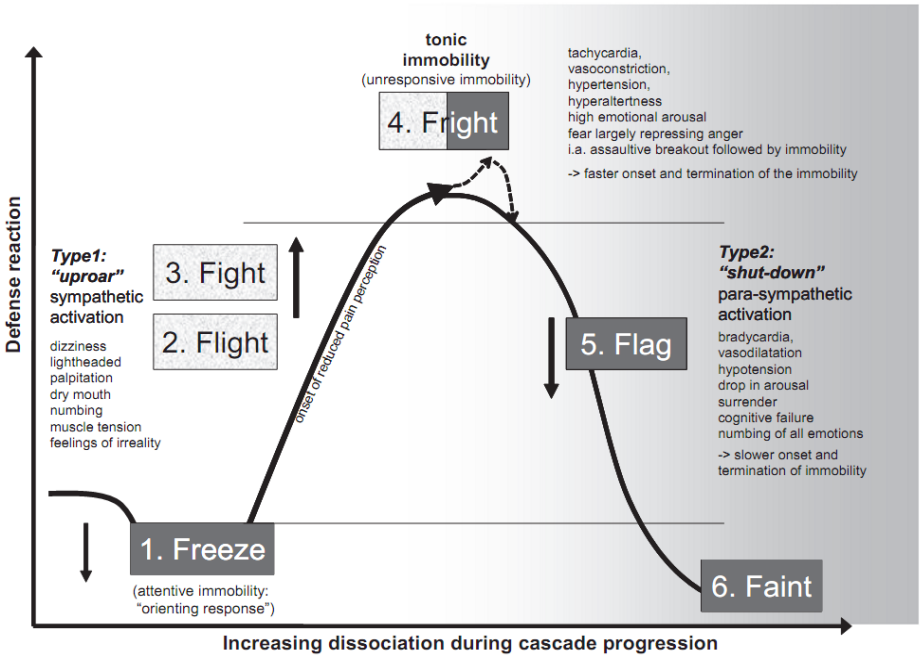


Figure 3. *Schematic illustration of the defence cascade as it progresses along the 6-F course of action. The ''uproar'' sympathetic arousal reaches a maximum at the fright stage, eventually superseded by the onset of dissociative ''shut-down'' (gray area).*

The same view on triggering situations reactivating biologically predetermined behavioural responses is shared by the more psychologically focused trauma-related model of structural dissociation proposed by van der Hart, Nijenhuis, & Steele (2006). Starting from the Janet's work on dissociation (van der Hart & Rutger, 1989), van der Hart et al. (2006) postulate that these responses are viewed by this model within the previously traumatized personality as "emotional parts of the personality" (EPs), which are carrying the memories of previous traumatic experiences. When people are in their EP, they may be experiencing the flashbacks, emotions or cognitions of the traumatic event. The EPs are dissociated from the so-called "apparently normal parts of the personality" (ANPs), which is that part of personality dealing activities of daily living and mostly fading traumatic memories out. By this organization, reactivation of traumatic memories can be avoided during the daily activities. According to van der Hart et al. (2006), there are three types of dissociation: primary, secondary, and tertiary dissociation (see Table 1). By this view, BPD patients may be understood as persons with an ANP and several EPs as separated personality system parts, which may explain the frequently observed dissociative states in stress situations as well as changes in mood and functioning.

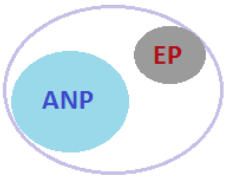
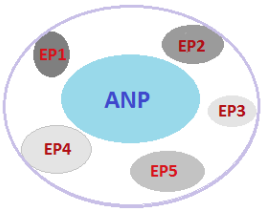
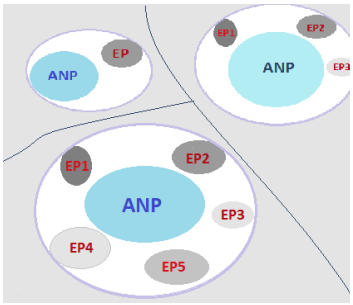
Type of dissociation	Diagnosis	Parts of the previously traumatised personality	
Primary	PTSD	One ANP and one EP experienced as "not me". In EP the trauma-related re-experiencing in form of flashbacks, emotions and cognitions takes place.	
Secondary	BPD DESNOS	One ANP and several EPs. Some EPs include active defence responses (arousal, flight, fight), whereas others exhibit dissociative responses (numbing, derealization).	
Tertiary	DID DDNOS	Several EPs and several ANPs. More separation between parts of personality and reduced awareness from each other. The parts of personalities may be organised in substructures corresponding to particular developmental periods.	

Table 1. *Schematic illustration of the three types of structural dissociation in the aftermath of exposure to aversive events according to the model of van der Hart et al. (2006). Correspondingly, the category of secondary dissociation includes person with complex PTSD and BPD.*

Implications and Conclusions

From a biological and psychological view, dissociation has been regarded as a survival strategy that arises during inescapable threat and may perpetuate in pathological forms. Such views are supported by numerous findings on the role of peritraumatic dissociation on the later development of mental disorders.

Clinical psychological research has shown that the presence of pathological dissociation in persons with BPD is an indicator of more severe clinical problems and functional impairments and aversive events have a major impact of on the

pathogenesis of dissociation. Studies investigating the association between self-harm and dissociation deliver additional evidence in this regard. The neurobiological research in the area of BPD also supports the assumption of trauma-related dissociation in BPD patients with dissociative symptoms and disorders.

Some comprehensive etiological models of stress-related dissociation based on the biological and psychological research findings have been already elaborated. Further research is necessary to empirically test such models and, more generally, the extensive investigation of trauma-related dissociative phenomena in BPD is required in order to better understand the dissociative features of BPD and to support the advance of psychotherapeutic methods.

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