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Aggression in Borderline Personality Disorder: A Multidimensional Model

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This article proposes a multidimensional model of aggression in borderline personality disorder (BPD) from the perspective of the biobehavioral dimensions of affective dysregulation, impulsivity, threat hypersensitivity, and empathic functioning. It summarizes data from studies that investigated these biobehavioral dimensions using self-reports, behavioral tasks, neuroimaging, neurochemistry as well as psychophysiology, and identifies the following alterations: (a) affective dysregulation associated with prefrontal-limbic imbalance, enhanced heart rate reactivity, skin conductance, and startle response; (b) impulsivity also associated with prefrontal-limbic imbalance, central serotonergic dysfunction, more electroencephalographic slow wave activity, and reduced P300 amplitude in a 2-tone discrimination task; (c) threat hypersensitivity associated with enhanced perception of anger in ambiguous facial expressions, greater speed and number of reflexive eye movements to angry eyes (shown to be compensated by exogenous oxytocin), enhanced P100 amplitude in response to blends of happy versus angry facial expressions, and prefrontal-limbic imbalance; (d) reduced cognitive empathy associated with reduced activity in the superior temporal sulcus/gyrus and preliminary findings of lower oxytocinergic and higher vasopressinergic activity; and (e) reduced self-other differentiation associated with greater emotional simulation and hyperactivation of the somatosensory cortex. These biobehavioral dimensions can be nicely linked to conceptual terms of the alternative *Diagnostic and Statistical Manual of Mental Disorders*, fifth edition (*DSM-5*) model of BPD, and thus to a multidimensional rather than a traditional categorical approach.

Keywords: alternative *DSM-5* model of BPD, borderline personality disorder, neurobiology, personality dimension, reactive aggression

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Aggression can be defined as any behavior directed toward another individual that is carried out with the proximate intent to cause harm (Anderson & Bushman, 2002). The high prevalence of aggression in borderline personality disorder (BPD) is demonstrated by data showing that 73% of individuals diagnosed with BPD have engaged in aggressive behaviors over the course of a year (Newhill, Eack, & Mulvey, 2009), 58% have been "occasionally or often" involved in physical fights, and 25% have used a weapon against others at some point in their lives (Soloff, Meltzer, & Becker, et al., 2003, p. 154). Additionally, individuals with BPD constitute a major proportion of aggression-prone populations such as prison inmates, with prevalence rates of 30% (Black et al., 2007). Studies have found enhanced aggression in BPD compared with healthy and clinical controls irrespective of whether the inclusion was based on categorical *Diagnostic and Statistical Manual of Mental Disorders*, third edition/fourth edition (*DSM–III/IV*) diagnosis (e.g., Gardner, Leibenluft, O'Leary, & Cowdry, 1991; McCloskey et al., 2009; Soloff, Kelly, Strotmeyer, Malone, & Mann, 2003) or on dimensional severity scores of BPD traits (e.g., Hines, 2008; Ostrov & Houston, 2008; Raine, 1993; Whisman & Schonbrun, 2009). Therefore, aggression has been regarded as a core feature of BPD (e.g., Siever et al., 2002; Skodol et al., 2002).

Aggression is most widely classified into instrumental and reactive forms (e.g., Berkowitz, 1993). Instrumental aggression refers to planned, goal-directed behavior, whereas reactive aggression is usually triggered by threats, frustration, or provocation and is strongly associated with negative emotions, particularly anger (e.g., Barratt & Felthous, 2003; Poulin & Boivin, 2000). In BPD, aggression is typically of the reactive type (Blair, 2004; Gardner, Archer, & Jackson, 2012; Herpertz et al., 2001). This has also been confirmed by laboratory tests of aggression, in which BPD patients have been repeatedly found to react more aggressively to provocations of a (fictitious) opponent compared with healthy individuals (Dougherty, Bjork, Huckabee, Moeller, & Swann, 1999; McCloskey et al., 2009; New et al., 2009). There is broad evidence indicating that aggression in BPD is tightly linked to interpersonal dysfunction, with negative interpersonal events (Herr, Keenan-Miller, Rosenthal, & Feldblum, 2013) and interpersonal problems (Stepp, Smith, Morse, Hallquist, & Pilkonis, 2012) predicting subsequent aggressive behavior in subjects scoring high on BPD traits. Additionally, BPD-associated aggression has been shown to

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primarily emerge in close relationships, with most aggressive acts directed against significant others or acquaintances (Newhill et al., 2009). BPD traits were also linked to intimate partner violence in young to late middle-aged individuals (Holtzworth-Munroe, Meehan, Herron, Rehman, & Stuart, 2000; Ross & Babcock, 2009; Weinstein, Gleason, & Oltmanns, 2012).

DSM-5 offers two concepts of BPD: In Section II (diagnostic criteria and codes), *DSM-5* provides a broad analogy to the categorical polythetic classification of BPD in *DSM–IV* (American Psychiatric Association, 2013, p. 663). The alternative model of BPD in Section III (emerging measures and models) uses a hybrid approach, which still conceptualizes BPD categorically, but in terms of multiple personality dimensions: All BPD individuals are located on a spectrum of impairments in personality functioning (differentiated into self and interpersonal functioning) and pathological personality traits on the domains of negative affectivity, antagonism, and disinhibition (American Psychiatric Association, 2013, p. 761; see, e.g., Skodol, 2012, for a review).

Our review begins with significant biobehavioral dimensions, meaning fundamental behavioral facets that have been shown to be associated with neurobiological alterations in BPD. The following dimensions have been discussed to underlie aggression in BPD: *affective dysregulation*, *impulsivity*, *threat hypersensitivity*, and *empathic functioning*. Our approach is to summarize the data related to these biobehavioral dimensions comprising self-reports, behavioral tasks, neuroimaging, neurochemistry, and psychophysiology.[1] We will then provide a working model of aggression in BPD, which illustrates that these biobehavioral dimensions can be nicely linked to conceptual terms of the alternative *DSM-5* model of BPD, and thus to a multidimensional rather than a traditional categorical approach. The alternative *DSM-5* model could therefore serve as a suitable starting point for future research on aggression in BPD.

Self-Reports and Behavioral Tasks

In this section, we will summarize studies that used self-reports and behavioral tasks to investigate the biobehavioral dimensions in the context of aggression in BPD.

Affective dysregulation plays a key role in theories of reactive aggression, as they regard reactive aggression to be a result of insufficiently controlled negative affect, predominantly anger (Berkowitz, 2003; Davidson et al., 2000; Siever, 2008). Links between *affective dysregulation* and aggression in BPD can be seen in studies revealing associations between BPD traits and guilt-, resentment-, and irritation-related self-reported forms of aggression that are described as the "more emotional aggressiveness subdimensions" (Fossati et al., 2004, p. 168). BPD traits also correlated positively with maladaptive forms of emotional coping, such as blaming oneself and venting emotions, which in turn mediated reactive aggression (Gardner, Archer, & Jackson, 2012). Two prospective studies revealed that *affective dysregulation* fully mediated the relationship between BPD traits and subsequent aggressive behavior in a mixed clinical and community sample (Scott, Stepp, & Pilkonis, 2014) and in a solely clinical sample of BPD patients (Newhill, Eack, & Mulvey, 2012). This emphasizes the high relevance of *affective dysregulation* for aggression in BPD. *Affective dysregulation* might also predispose the individual to increased experiences of anger or hostility. This was empirically demonstrated by Trull et al. (2008), who used experience sampling methodology (i.e., ambulatory and real-time data collection) to show that BPD patients more frequently experienced extreme spikes of hostility than a clinical control group of depressive patients. The experience of anger may ultimately result in aggressive behavior, as recently illustrated in a study showing that the expression of anger, for example, arguing with other people, predicted subsequent aggressive behaviors in individuals scoring high on BPD traits (Stepp et al., 2012). The relevance of anger for aggression in BPD was also supported by the identification of a subgroup of BPD patients in whom anger proneness and aggression were found to co-occur (Hallquist & Pilkonis, 2012).

Reactive aggression has also been considered as a consequence of *impulsivity* (Coccaro, Sripada, Yanowitch, & Phan, 2011; Gollan, Lee, & Coccaro, 2005; Goodman & New, 2000). Data from BPD patients showed that *impulsivity* and aggression were positively correlated (e.g., Hollander et al., 2005; Soloff, Meltzer, et al., 2003) and loaded on the same factor in one (Koenigsberg et al., 2001) but not in all studies (Critchfield, Levy, & Clarkin, 2004). Scott et al., (2014) reported that *impulsivity* did not mediate between BPD traits and aggression. Besides differences in sample characteristics, the latter result might be attributable to the instrument used to assess *impulsivity*, that is, a composite of different subscales of the NEO Personality Inventory (Costa & McCrae, 1992). As this instrument was designed to assess normative personality traits, it may have failed to capture the aspects of *impulsivity* specifically related to aggression. Given the strong correlation between *impulsivity* and anger (García-Forero, Gallardo-Pujol, Maydeu-Olivares, & Andrés-Pueyo, 2009), one might also speculate that the effect of *impulsivity* on aggression varies as a function of anger. That is, poor impulse control might only (or particularly) result in aggressive behavior under circumstances of momentarily experienced anger.

Threat hypersensitivity is a central construct in Blair's model of reactive aggression (Blair, 2004, 2012). Animal studies indicate that aggressive behavior is displayed when a threat is very close and escape is impossible (Blanchard, Blanchard, Takahashi, & Kelley, 1977). In BPD patients, *threat hypersensitivity* has mainly been studied by means of facial recognition tasks. Results indicate a biased or enhanced perception of social threat cues in BPD patients (Domes, Schulze, & Herpertz, 2009): Compared with healthy controls, they overreported fear when presented with neutral faces (Wagner & Linehan, 1999), perceived ambiguous blends of facial expression as more angry (Domes et al., 2008), and focused more initial attention (von Ceumern-Lindenstjerna et al., 2010a) toward and had difficulties in disengaging from negative facial expressions (von Ceumern-Lindenstjerna et al., 2010b). In addition, in a study using eye tracking, BPD patients were found to have more and faster initial reflexive eye movements toward the eyes of very briefly presented angry faces, thus the most threatening and arousing region (Bertsch, Gamer, et al., 2013). These findings suggest that BPD patients misattribute facial emotions, making them susceptible to the experience of threat or provocation, and ultimately to reactive aggression.

[1] See Table S1 in the supplemental materials for a detailed description of the cited studies, including sample characteristics, methodology, and key findings.

Threat hypersensitivity may be one of the sources leading to the interpersonal hypersensitivity of BPD individuals. According to the alternative *DSM-5* model of BPD, interpersonal hypersensitivity is the "prone[ness] to feel slighted or insulted" (American Psychiatric Association, 2013, p. 766). It has been linked to aggression in BPD by findings showing that criticism or blame predicted aggressive behavior in women scoring high on BPD traits (Herr et al., 2013). Furthermore, fearful forms of attachment that are particularly linked to interpersonal hypersensitivity (Gunderson & Lyons-Ruth, 2008) have been associated with reactive aggression in BPD patients (Critchfield, Levy, Clarkin, & Kernberg, 2008). *Threat hypersensitivity* might also interact with fundamental assumptions of BPD patients, such as seeing the world and others as dangerous and malevolent, which may lead to feelings of threat and could provoke reactive aggression (Arntz, Dreessen, Schouten, & Weertman, 2004; Arntz, 1994; Pretzer, 1990).

Empathic functioning can be differentiated into *cognitive empathy*, *affective empathy*, and supplemental regulatory mechanisms. Analogously to mentalization or theory of mind, *cognitive empathy* is regarded as the capacity to infer the mental states of others. *Affective empathy* captures the emotional engagement with other individuals' emotional displays. The regulatory mechanisms allow the distinction between emotional reactions of the self and the other, hereafter called *self-other differentiation* (see, e.g., Decety, 2011; Jeung & Herpertz, 2014, for reviews). Impaired *empathic functioning* is fundamental to the evolution of human aggression (de Waal, 2012; Decety, 2011; Fonagy, 2003). In empirical terms, *cognitive empathy* in particular has been associated with reactive aggression (Fossati et al., 2009; Jolliffe & Farrington, 2004; Renouf et al., 2010; van Langen et al., 2014). In BPD patients, reduced *cognitive empathy* was found in studies using self-reports (Harari, Shamay-Tsoory, Ravid, & Levkovitz, 2010; New et al., 2012) or advanced behavioral tasks that approximate real-life social interactions (Dziobek et al., 2011; Preißler, Dziobek, Ritter, Heekeren, & Roepke, 2010; Ritter et al., 2011). The latter tasks—also referred to as ecologically valid tasks— use, for instance, short film clips displaying social interactions, after which participants have to evaluate the intentions, emotions, and thoughts of the interaction partners. Contrary to the finding of reduced *cognitive empathy*, and despite some inconsistencies (Dziobek et al., 2011), *affective empathy* seems to be intact in BPD patients (Harari et al., 2010; Mier et al., 2013; New et al., 2012).

First results indicate that BPD patients try excessively to interpret other people's mental states and/or overattribute the intentions of others, which suggests impairments in *self-other differentiation* (Sharp et al., 2011). In tasks that can be performed via cognitive or affective empathic involvement, such as the "Reading the Mind in the Eyes task" (Baron-Cohen, Jolliffe, Mortimore, & Robertson, 1997), BPD patients outperformed healthy controls (Fertuck et al., 2009; Frick et al., 2012), possibly through (compensatory) overmobilizing affective empathic strategies, such as emotional simulation (Gallese & Goldman, 1998). In line with the theory of greater emotional simulation in BPD, Matzke et al. (2014) found that compared with healthy controls, BPD patients exhibited higher electromyographic activity of the frowning muscle while viewing negative facial expressions of others such as anger, sadness, and disgust. Exaggerated emotional simulation, however, impacts negatively on the capability of *self-other differentiation*, resulting in an affect-dominated and unmediated perception of others, known as emotional contagion (Fonagy & Luyten, 2009; Schmahl & Herpertz, 2014). This leaves BPD patients "vulnerable to losing a sense of self" (Fonagy & Luyten, 2009, p. 1362) and to being increasingly overwhelmed by others' mental states. In the context of dysfunctional intimate relationships, which provoke negative emotions of despair, jealousy (Costa & Babcock, 2008), fear of abandonment (Gunderson, 1996), mistrust, and/or shame (Rüsch et al., 2007), this could result in experiences of anger (Peters, Geiger, Smart, & Baer, 2014), threat, frustration, and eventually reactive aggression.

Interestingly, the pattern of deficient *empathic functioning* of BPD patients is the opposite of that found in individuals with other aggression-prone personality disorders, such as antisocial or psychopathic individuals. The latter show intact *cognitive empathy* but impaired *affective empathy* (Blair, 2013), favoring instrumental forms of aggression (Blair, Peschardt, Budhani, Mitchell, & Pine, 2006) which have been shown not to be abnormally enhanced in BPD (Herpertz et al., 2001). However, studies directly investigating the relationship between *empathic functioning* and aggression in BPD are still lacking, and further research is needed to disentangle the differential contributions of *empathic functioning* to aggression in BPD and related disorders.

In the attempt to capture these findings (hereafter related to alterations of the biobehavioral dimensions) within the alternative *DSM-5* model of BPD, they can be subsumed under both impairments of personality functioning and pathological personality traits (American Psychiatric Association, 2013, pp. 766 –767), and thus the two descriptive levels provided by this classification. *Affective dysregulation* is closely related to emotional lability, a trait facet of the personality domain negative affectivity. *Impulsivity* is directly addressed as a trait facet of the personality domain disinhibition. *Threat hypersensitivity* can be related to both descriptive levels: to the trait facets of emotional lability and hostility on the one hand, and to impairments in interpersonal functioning on the other. Within the latter, it can be attributed to the empathic deficit of BPD patients, namely to interpersonal hypersensitivity and a negatively biased perception of others. The reduced *cognitive empathy* is encompassed in the description of the empathic deficit of the alternative *DSM-5* model. However, reduced *self-other differentiation*, regarded as a facet of poor empathy in neuroscientific models (Decety & Jackson, 2004), is not directly addressed as an empathic deficit in the alternative *DSM-5* model. Instead, it is linked to the "markedly impoverished, poorly developed, or unstable self-image" mentioned as reflecting disturbances of BPD patients' self-identity.