Borderline Personality Disorder, Complex Trauma, and Problems with Self and Identity: A Social-Communicative Approach

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Abstract

Borderline personality disorder (BPD) is a relatively highly prevalent psychiatric disorder that is associated with very high personal and socioeconomic costs. This paper provides a state-of-the art review of the relationship between complex trauma and key features of BPD, with a focus on problems with self-coherence and self-continuity. We first review evidence for the high prevalence of complex trauma in BPD patients. This is followed by a discussion of emerging knowledge concerning the biobehavioral mechanisms involved in problems related to self and identity in BPD. We emphasize three biobehavioral systems that are affected by complex trauma and are centrally implicated in identify diffusion in BPD: the attachment system, mentalizing or social cognition, and the capacity for epistemic trust—that is, an openness to the reception of social communication that is personally relevant and of generalizable significance. We formulate a new approach to personality and severe personality disorders, and to problems with self and identity in these disorders, rooted in a social-communicative understanding of the foundations of selfhood. We also discuss how extant evidence-based treatments address the abovementioned biobehavioral systems involved in identity diffusion in BPD and related disorders, and the supporting evidence. We close the paper with recommendations for future research.

Keywords: Borderline personality disorder, personality, self, identity, trauma.

Introduction

Borderline personality disorder (BPD) is a relatively highly prevalent disorder that is associated with high personal and socioeconomic costs. In the context of this special issue focusing on self and identity, it is particularly important to point out that BPD is associated with very high levels of self-harm and suicidality (Leichsenring, Leibing, Kruse, New, & Leweke, 2011). In combination with the high levels of emotional and physical pain (Holm & Severinsson, 2008; Sansone & Sansone, 2012), and hypersensitivity to social exclusion (Bungert et al., 2015; De Panfilis, Riva, Preti, Cabrino, & Marchesi, 2015), a focus on impairments in the sense of self and identity in these patients seems appropriate. Indeed, studies show that up to 10% of patients with BPD die by suicide (Skodol et al., 2002).

Both diagnostic (American Psychiatric Association, 2013) and theoretical (Beck, Freeman, & Davis, 2004; Fonagy, Luyten, & Allison, 2015; Kernberg & Caligor, 2005) approaches have focused on impairments in self and identity as a central factor in BPD. These impairments in the self-structure so typical of BPD patients has been linked to other key features of the disorder such as affect dysregulation, dissociation, impulsivity, and problematic interpersonal relationships (Gunderson & Lyons-Ruth, 2008; Kernberg & Caligor, 2005; Richetin, Preti, Costantini, & De Panfilis, 2017).

Both clinical practice and research suggest high rates of early adversity, at least in a subsample of patients, with most of these patients having a history of attachment trauma or so-called complex trauma (Ball & Links, 2009; Chanen & Kaess, 2012; de Aquino Ferreira, Queiroz Pereira, Neri Benevides, & Aguiar Melo, 2018; Stepp, Lazarus, & Byrd, 2016). Complex trauma in particular has been suggested to play a key role in explaining the severe problems with self and identity in BPD patients.

This paper provides a state-of-the art review of the relationship between complex trauma and key features of BPD, with a focus on problems related to self and identity. We

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first review evidence for the high prevalence of complex trauma in BPD patients. This is followed by a discussion of emerging knowledge concerning the biobehavioral mechanisms involved in problems related to the self in BPD. We emphasize three biobehavioral systems that are affected by complex trauma and are centrally implicated in identify diffusion in BPD: the attachment system, mentalizing or social cognition, and the capacity for epistemic trust. We formulate a new approach to personality and severe personality disorders, and to problems with self and identity in these disorders. We also discuss the implications of these views for current evidence-based treatments for BPD, and directions for future research.

Specifically, we discuss two major related changes in our views on complex trauma. First, whereas in the past we have always emphasized the negative impact of (complex) trauma on the capacity to form and maintain attachment relationships, and on the capacity for reflective functioning or mentalizing, there has been a notable shift in our thinking concerning complex trauma. Indeed, we now consider the impact of trauma within a broader bioecological, evolutionary framework, and argue that the negative impact of complex trauma should be considered within a broader framework emphasizing continuous interactions between environmental factors (not limited to the attachment environment, but also considering broader environmental factors such as peers and the sociocultural context) and biological factors (see Figure 1). Second, as depicted in Figure 1, we now argue that these interactions between environmental and biological factors disrupt the evolutionarily pre-wired human capacity for social learning and salutogenesis (i.e., the capacity to benefit from positive social input) by detrimental effects on the capacity for epistemic trust—that is, openness to the reception of social communication that is personally relevant and of generalizable significance. Because epistemic trust is disrupted by trauma, impairments in the capacity for attachment and the associated capacity for social cognition or mentalizing ensue, closing the individual off from the social world and thus the possibility of learning and the

social recalibration of the mind. Hence, the central idea underlying our current approach to complex trauma and selfhood is that trauma becomes complex when it undermines the individual's capacity to learn from the social environment, as depicted in Figure 1, by inhibiting, and in the extreme even totally halting, the evolutionarily adaptive capacity for epistemic trust that facilitates social learning and salutogenesis by opening up the individual to the social world through the development of secure attachment relationships and mentalizing. A breakdown in communication and social learning generates the experience of isolation that is associated with complex trauma, and is so typical of individuals with BPD, and further closes off the capacity for accessing the adaptive functioning of the social imagination in relation to the intersubjective self. As depicted in Figure 1, instead of moving forward, trauma moves the system in the opposite direction.

[Insert Figure 1 here]

Problems with Self and Identity in BPD: Quo Vadis?

Although there has been a continuous increase in the development and empirical evaluation of treatments for BPD patients, recent years have also seen considerable controversy regarding the nature of BPD (Bender, Morey, & Skodol, 2011; Shedler et al., 2010; Skodol, 2012; Spitzer, First, Shedler, Westen, & Skodol, 2008). On the one hand, BPD is often diagnosed using a polythetic combination of descriptive features as in the *Diagnostic and Statistical Manual of Mental Disorders* (DSM; American Psychiatric Association, 2013). Given the polythetic diagnostic approach in DSM, theoretically speaking, 256 combinations of the symptoms are possible. BPD thus defined is therefore extremely heterogeneous, as individuals meeting criteria for the disorder may have very different clinical presentations. Studies have identified various subtypes or clusters of BPD patients (Smits et al., 2017).

Yet, at the same time, recent studies also suggest a strong common factor in BPD (Sharp et al., 2015; Smits et al., 2017), which has recently also been referred to as a general psychopathology or "p" factor (Caspi et al., 2014; Fonagy et al., 2015). But what is this common factor underlying the various clinical presentations of BPD? This question has led to a second tradition in research concerning the nature and diagnosis of BPD, emphasizing commonalities among BPD patients and patients with personality disorder more generally. Historically, several theoretical approaches have invoked the concept of a disturbed sense of self or identity (Blatt & Auerbach, 1988; Bradley & Westen, 2005; Jorgensen, 2006; Kernberg & Caligor, 2005; Livesley, 2008). As noted, impairments in the sense of agency or self-directedness—also referred to as identity diffusion—have been consistently identified as a key feature of the disorder (Adler, Chin, Kolisetty, & Oltmanns, 2012; Barnow, Ruge, Spitzer, & Freyberger, 2005; Bender & Skodol, 2007; Jørgensen et al., 2012; Richetin et al., 2017). Furthermore, these impairments have been closely associated with problems in the capacity to form and maintain interpersonal relationships, and other characteristics typically associated with BPD, such as high levels of impulsivity, feelings of dissociation, and a strong sense of inner pain prompted by experiences of rejection, isolation, or abandonment (Gunderson & Lyons-Ruth, 2008). Consistent with these findings, different levels of impairments in levels of self or identity and interpersonal functioning have been proposed as central dimensions underlying personality disorders, and BPD specifically, in future editions of DSM (Bender et al., 2011).

BPD can indeed be considered to be perhaps the most prototypical disorder in terms of problems related to self and identity and associated problems with attachment and relatedness. From this perspective, instability is what is stable in BPD (Schmideberg, 1959). This also makes individuals with the disorder "hard to reach", with studies suggesting that they show considerable delays in seeking treatment, high levels of dropout from treatment,

and typically a succession of unsuccessful and often brief treatments (Fonagy, Luyten, Allison, & Campbell, 2017a; Leichsenring et al., 2011).

The starting point of the present paper is perhaps somewhat paradoxical in this regard. In our opinion, it has been insufficiently emphasized that, while BPD is characterized by self—other diffusion and the marked instability and fluidity that is associated with these features, at the same time it is also associated with marked rigidity. Of course, what we are saying is not completely new. The rigidity of BPD patients has been a central component in many theories of BPD and has been invoked to explain the temporal and cross-situational stability of personality traits related to BPD (Fonagy et al., 2015). Yet, there is a clear risk of reification and circularity here, as we need to understand the mechanisms responsible for the absence in these patients of the capacity for adaptation and change, and the developmental roots of this absence.

In this context, notable theorists have described adaptive personality development in terms of the capacity to respond appropriately to ever-changing circumstances. Carl Rogers (Rogers, 1961; Rogers & Dymond, 1954), for instance, described healthy personality functioning as being characterized by openness to experience, flexibility, adaptability, and spontaneity. Aaron Beck (Beck et al., 2004) similarly described flexibility of cognitive-affective schemas as a key feature of the structural qualities of schemas, besides their breadth and density. Even closer to the present approach, both attachment approaches and object relational theorists have described rigidity as a central feature of personality pathology (McWilliams, 2011). Specifically, Blatt and colleagues (Blatt & Luyten, 2009; Luyten & Blatt, 2013) have emphasized that while adaptive personality development is characterized by the capacity to constantly re-evaluate the sense of self and relatedness in the course of development, psychopathology involves a lack of the capacity to move flexibly around either polarity, leading to an exaggerated emphasis on either identity and autonomy, or attachment

and relatedness. Both contemporary interpersonal theories (Benjamin, 2005; Horowitz et al., 2006) and attachment theory (Mikulincer & Shaver, 2007) share a similar emphasis on the need for flexibility. Within attachment theory, for instance, insecure individuals are thought to be characterized by marked rigidity in terms of applying the same templates from the past to new attachment relationships, in that they fear either the loss of autonomy (i.e., self) and/or the loss of affection of their attachment figure (i.e., relatedness) (Mikulincer & Shaver, 2007). Crucial here for our argument is that the insecurely attached individual has a strong tendency to hold on to this template even when it is not confirmed by others; there is a closing off of the flow of information provided by others concerning attachment relationships.

This latter view brings us a step closer to an explanation for the marked rigidity that characterizes individuals with personality disorder (particularly BPD), but it does not provide a sufficient explanation for what appears to be typical of patients with marked BPD features. As noted above, comparable concepts such as personality, a sense of self and identity, internal working models of attachment, and cognitive schemas of self and others have been invoked to explain the rigidity typical of BPD patients. But these are hypothetical constructs, and, although they attempt to bridge the interface between the individual and his/her social environment, they need to be explained themselves, otherwise there is a risk of reification and circularity. The self, and particularly the sense of self-coherence and self-continuity over time, is an illusion (Bargh, 2011, 2014) that is the product of the capacity for reflective functioning or mentalizing—that is, the human capacity to understand oneself in terms of intentional mental states (i.e., feelings, wishes, attitudes, and goals) (Han & Northoff, 2009; Northoff et al., 2006). Stated otherwise: the self (and feelings of self-coherence and selfcontinuity) is always created "on-line", as it were. BPD patients seem to have severe problems in the process of creating this feeling of continuity and coherence, particularly in high-arousal contexts such as interpersonal relationships. This is also demonstrated by studies

showing that BPD patients with low levels of self-concept clarity in particular tend to engage in body modification (e.g., piercing, scarification, cosmetic surgery) and self-harm (Scala et al., 2018). In the next section, we summarize the latest iteration of our evolving understanding of these problems, which is rooted in evolutionary-based theories and findings related to the origins of the capacity for epistemic trust and epistemic petrification in particular, and the function of social communication, particularly when faced with traumatic events. As discussed in more detail elsewhere, our views come closest to those of Kruglanski and colleagues (Kruglanski, 1989; Kruglanski & Webster, 1996; Pierro & Kruglanski, 2008). According to these authors, *epistemic freezing* refers to a tendency to defend existing knowledge structures even when they are incorrect or misleading (see also Fiske & Taylor, 1991). Such a defensive strategy, also referred to as cognitive closure, may indeed be adaptive when the individual is first confronted with trauma, particularly when the trauma is part of a broader traumatizing environment. Consistent with this view, we redefine personality disorder as a disorder of social communication resulting in marked impairments in the sense of self-coherence and self-continuity because the individual is unable to benefit from the organizing influence of social communication and social recalibration of the mind in particular. Finally, although the focus in this paper is on complex trauma in relation to BPD, we will argue that adversity is neither a necessary nor a sufficient condition for the epistemic petrification that is typical of BPD patients. In this context, we will focus on the ostensible role of genetic factors and hypersensitivity to social information more generally.

BPD and (Complex) Trauma

Complex trauma usually refers to prolonged early negative life experiences involving neglect and/or abuse, typically within an attachment/caregiving context, meaning that caregivers who are supposed to protect and care for the child are at the same time a source of

anxiety, threat, neglect, and/or abuse (Asnes & Leventhal, 2011). Complex trauma is also often referred to as Type II trauma, attachment trauma, early relational trauma, or early developmental trauma, as opposed to Type I trauma, which refers to experiencing or witnessing a single extreme or life-threatening event that is often impersonal in nature, such as a natural disaster (Herman, 1992; Meichenbaum, 1994; Terr, 1991). Complex trauma is sometimes further divided into Type II trauma, involving prolonged and repeated negative events (e.g., sexual abuse, domestic violence, torture, genocide) typically involving conflict, abuse, and/or neglect by others, and Type III trauma, defined as (early) abuse and neglect within a caregiving context (Mendelsohn et al., 2011; Solomon & Heide, 1999). The distinction between these types of trauma and their relationship with (early) adversity is, however, not always that clear, as complex trauma does not tend to occur in isolation but is typically part of a so-called broader "risky environment" (Cicchetti & Toth, 2005). For instance, McLaughlin et al. (2012) found that 60% of adolescents in a US national survey reported multiple childhood adversities. Similarly, Green et al. (2010) found that all types of adversity assessed in the National Comorbidity Survey-Replication, a populationrepresentative study in the US, were highly inter-correlated. Moreover, these authors found that a general maladaptive family functioning factor, consisting of different types of childhood adversity (including parental mental illness, substance abuse disorder, criminality, family violence, physical abuse, sexual abuse, and neglect), was the most robust predictor of onset of mental disorders (Green et al., 2010). These findings are consistent with the notion that complex trauma should always be considered within a broader socioecological framework (Bronfenbrenner, 1977; Cicchetti & Toth, 2005).

Given the high rates of complex trauma in BPD (discussed in detail below) and the well-demonstrated pernicious effects of trauma on the development of feelings of self and identity, the capacity to form interpersonal relationships, and emotion regulation, it is highly

tempting to relate problems with self-coherence and self-continuity in BPD to trauma, and particularly complex trauma. Although there is a need for more research, and prospective studies in particular, recent reviews converge to suggest that there is fairly consistent evidence suggesting that trauma—and complex trauma in particular—is causally linked to increased vulnerability for BPD (Ball & Links, 2009; Chanen & Kaess, 2012; de Aquino Ferreira et al., 2018; Stepp et al., 2016). A recent review of 39 prospective studies, reflecting 24 unique samples, suggests that exposure to different types of trauma, such as emotional abuse and neglect, and physical and sexual abuse, typically occurring within a broader context characterized by neglect and maltreatment (i.e., poor parenting, parental psychopathology, lower socioeconomic status, violence), is associated with increased risk for BPD (Stepp et al., 2016). Indeed, rates of (complex) trauma in BPD are often as high as 90%, and patients with BPD tend to fairly consistently report the highest levels of trauma compared with individuals with chronic depression (Brakemeier et al., 2018)—another disorder closely associated with trauma—and other types of personality disorder. Based on these findings and common associated biobehavioral disturbances, some authors have even proposed reformulating BPD, or at least a subtype of BPD, in terms of a disorder of complex trauma or complex post-traumatic disorder—an option that is under consideration for the new version of the International Classification of Diseases (for a recent overview, see Giourou et al., 2018).

However, there are several caveats that warn against such a simple interpretation of this body of literature. First, despite the high observed rates of (complex) trauma in BPD, not all patients with BPD have a history of early adverse experiences. Between 8% (Kingdon et al., 2010) and 70% (Afifi et al., 2011) of individuals with BPD do not report early abuse or neglect.

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Second, an emphasis on complex trauma could easily lead to simplistic etiological models that unjustly focus on early parent-child relationships to the neglect of peer relationships, the broader environment, and the mediating and moderating role of later experiences, as has been typically found in longitudinal studies (Carlson, Egeland, & Sroufe, 2009; Carlson, Sroufe, & Egeland, 2004; Salvatore, Haydon, Simpson, & Collins, 2013). In the E-Risk study, a longitudinal twin study of over 2,200 children and families, lower mentalizing capacities, as assessed with a theory of mind task, were found in individuals with emergent BPD in early adolescence (Belsky et al., 2012). These impairments were clearly related to problems in peer relationships—particularly becoming a victim, being a bully, and being a bully-victim (i.e., a bully who has a history of being a victim) (Shakoor et al., 2012). Similarly, in the Avon Longitudinal Study of Parents and Children, the risk of BPD symptoms increased with increased risk of bully-victimization (Winsper, Hall, Strauss, & Wolke, 2017). Furthermore, broader environmental and sociocultural factors such as social inequality appear to play an important role in explaining vulnerability to BPD. As demonstrated by Wilkinson and Pickett (2009), countries with the highest levels of income inequality also show higher levels of mental health problems that are typically linked to BPD, such as substance abuse and teenage births. Furthermore, the prevalence of BPD can be relatively well predicted from the ratio of the average income of the richest 20% to that of the poorest 20% of the population. Hence, a general lack of social concern for equality may be directly related to the prevalence of BPD.

A third important caveat is that there is strong and consistent evidence for the role of individual difference variables implicated in vulnerability to BPD. These include genetic factors and temperamental differences, and these factors may be particularly important in the context of considerations concerning the role of (complex) trauma in BPD. With regard to genetic factors, although molecular genetic studies in BPD so far have not led to replicable

results (Fraley, Roisman, Booth-LaForce, Owen, & Holland, 2013; Luijk et al., 2011), heritability estimates of BPD range between 40% and 50% (Bornovalova, Hicks, Iacono, & McGue, 2009; Distel et al., 2008; Kendler et al., 2008; Torgersen et al., 2000). In addition, there is increasing evidence for gene—environment interactions in BPD, emphasizing the need to develop multifactorial interactional models of BPD. Distel et al. (2011), for instance, in a twin study of over 5,000 twins and almost 1,300 siblings, reported that the unique environmental variance in explaining BPD features increased linearly with the number of traumatic life events to which an individual had been exposed (from 54% with no events to 64% with six events). Likewise, the proportion of variance accounted for by genetics declined with exposure to adversity (from 46% in those exposed to no traumatic life events to 36% in those who report six or more types of trauma) (Distel et al., 2010).

Similarly, Belsky et al. (2012) studied a nationally representative birth cohort of over 1,100 families with twins, finding only a weak association between maltreatment and BPD symptoms. However, when genetic vulnerability, operationalized as any family history of mental disorder, was introduced, maltreatment was highly associated with BPD: of those with a family history of psychopathology and maltreatment, 47% were in the "extreme" BPD group. By contrast, in the absence of a family history of psychopathology, maltreatment was reported by only 7% of individuals with BPD. Hence, differential susceptibility to the environment (Belsky & Fearon, 2008; Ellis, Boyce, Belsky, Bakermans-Kranenburg, & van Ijzendoorn, 2011) may play an important role in the development of BPD.

This brings us to another factor that is in our opinion important to consider in the ongoing discussion of the role of complex trauma in BPD. Child temperament (which may itself be in part genetically determined), may play an important role in moderating the effects of genes and environment. For instance, high levels of impulsivity/aggression and hypersensitivity to social information have been implicated in BPD (for a review, see Fonagy

& Luyten, 2016), and may play an important role in developmental pathways to BPD in combination with, but also in the absence of, trauma. For instance, individuals with BPD have been reported to show elevated levels of emotional and physical pain in response to negative experiences (Holm & Severinsson, 2008; Sansone & Sansone, 2012), and hypersensitivity to social exclusion (Bungert et al., 2015; De Panfilis et al., 2015). One study reported that individuals with BPD feel excluded even when they are actually socially included by others (De Panfilis et al., 2015). This would also be consistent with research suggesting that evocative person-environment correlations play a major role in explaining developmental trajectories (Klahr & Burt, 2014; Marceau et al., 2013). The effects of maltreatment in the development of BPD might often reflect gene-gene effects mediated through trauma (wherein genetic vulnerability of the attachment figure influences genetic vulnerability in the child) and evocative gene-environment correlations (wherein the child unwittingly increases the likelihood of neglect and abuse by the parent), rather than simple environmental effects. Stated somewhat schematically: children with a "difficult" temperament are, on average, more likely to be abandoned, neglected, or abused by attachment figures who are themselves more impulsive and hypersensitive to social information (Crawford, Cohen, Chen, Anglin, & Ehrensaft, 2009; Lyons-Ruth & Jacobvitz, 2008). As a result, and as explained in more detail below, it is highly likely that neglect and abuse will cause these children to miss out on the organizing influence of early parenting on the development of the self and self-coherence, self-worth, and self-continuity in particular, as these children are increasingly likely to begin to alternately blame their attachment figures and themselves for the neglect and abuse, leading to splits in their self-representation as either deserving or unworthy of love and attention, depending on the emotional state they are in. In the next section, we discuss the impact of (complex) trauma on attachment and mentalizing and on the development of self and identity specifically.

Hence, as depicted in Figure 1, both biological and (broader) environmental factors, and their interactions (as expressed, for instance, in gene–environment correlations and interactions, and in epigenetic effects) should be included in any theoretical model of complex trauma.

Complex Trauma in BPD and Impairments in Self and Identity: The Role of Attachment and Mentalizing

Trauma and Attachment

The impact of trauma, particularly attachment trauma, on the attachment and mentalizing biobehavioral systems has been relatively well documented at both the behavioral and the neurobiological level. The attachment system is a biobehavioral system that is activated in response to threat (Bowlby, 1973; Mikulincer & Shaver, 2007). Activation of the attachment system typically leads the individual to seek proximity to attachment figures, which, at least in the context of secure attachment (i.e., when attachment figures are available and responsive), leads to down-regulation of feelings of distress and discomfort.

A mesocorticolimbic dopaminergic circuit also known as the reward system, in concert with the stress system, underpins this sequence of events. Mesolimbic pathways in the reward system originate from the ventral tegmental area and project to ventral striatal regions, the hippocampus, and the amygdala. Mesocortical pathways, in turn, involve projections to the prefrontal cortex and anterior cingulate cortex (Pizzagalli, 2014; Russo & Nestler, 2013; Spear, 2000). Dopamine, oxytocin, vasopressin, opioids, and cannabinoids are key biological mediators in the reward system, and have been shown to modulate the pain and distress associated with social loss and rejection (Hsu et al., 2015; Panksepp & Watt, 2011; Spear, 2000). The attachment system, and the reward system underpinning it, thus plays a fundamental role in both the development and the continuing regulation of the stress

system (Hostinar, Sullivan, & Gunnar, 2014; Strathearn, 2011; Swain et al., 2014). Securely attached individuals are able to turn to others in times of stress and adversity. For these individuals, attachment experiences are rewarding; hence, these individuals turn to others in times of need, and proximity to attachment figures (either in reality or by the activation of representations of interactions with attachment figures) typically leads to down-regulation of distress and discomfort—thus buffering the effects of stress and adversity. Securely attached individuals are fundamentally driven by the expectation that others will be there for them and will support, understand, and validate them.

By contrast, repeated experiences of insecurity in attachment relationships are associated with increased vulnerability to stress and adversity, mediated through disturbances in stress regulation systems—the hypothalamic—pituitary—adrenal (HPA) axis and the sympathetic nervous system in particular—and associated immune and pain regulation systems (Gunnar & Quevedo, 2007). These individuals function as if they are constantly in a fight/flight or freeze state, and typically show hypersensitivity to stress and adversity. Insecurely attached individuals, particularly those with complex trauma, have the expectation that others will not be there to provide support and comfort. Furthermore, the chronic stress and arousal that is associated with attachment trauma may initially lead to a state of HPA axis hyperactivity, but this may switch to hypoactivity because of the wear and tear on physiological systems of such chronic high arousal states (Miller, Chen, & Zhou, 2007). This may also explain the high comorbidity between (complex) trauma and other stress-related syndromes, such as depression, posttraumatic stress disorder (PTSD), chronic pain and chronic fatigue conditions, and physical disease (Anda et al., 2006).

These "programming" effects of early adversity may be particularly pronounced in socalled sensitive or critical periods, which in humans probably last into early adulthood (Heim & Binder, 2012; Lupien, McEwen, Gunnar, & Heim, 2009). This is also the time when attachment trauma, particularly abuse and neglect, characteristically occurs, which helps to explain the particularly pernicious effects of early maltreatment. Yet, it is often forgotten that this critical window extends into adolescence and early adulthood. Brain structures involved in the stress and attachment systems (as well as the mentalizing system; see below) undergo key structural changes and functional reorganization at this time (Lupien et al., 2009; Mutlu et al., 2013; Pervanidou & Chrousos, 2012; Shaw et al., 2008). It is therefore not surprising that studies have relatively consistently found structural and functional abnormalities in neural systems underlying the stress, attachment, and mentalizing systems in individuals with a history of (complex) trauma across diagnostic categories (Gobinath, Mahmoud, & Galea, 2014; Serafini et al., 2014).

The example of oxytocin, one biomediator in the attachment system, is particularly pertinent in this regard. Studies suggest that in securely attached individuals, the administration of oxytocin typically leads to an increase in affiliative behavior and trust in others, at the same time reducing behavioral and neuroendocrinological responses to stress via down-regulation of the HPA axis (Feldman, Vengrober, & Ebstein, 2014; Neumann, 2008). By contrast, studies have found decreased basal oxytocin levels in individuals with attachment trauma, and negative effects of oxytocin administration on cooperation, trust, and the stress response (Bartz, Zaki, Bolger, & Ochsner, 2011). At the behavioral level, this is typically expressed in terms of severe impairments in the capacity to seek help from others when faced with stress and adversity. Individuals with a history of complex trauma typically find themselves in a catch-22 situation: the very same caregivers who should provide support, comfort, and understanding are the source of conflict, abuse, and/or neglect (Teicher & Samson, 2013).

As a result, attachment hyperactivating or deactivating strategies—or a combination of both, as observed in individuals with disorganized attachment—develop. There is

increasing consensus that these so-called secondary attachment strategies should be seen in terms of adaptations to a particular environment characterized by marked inconsistency in the availability of attachment figures and/or downright abuse and/or neglect by these figures (Ein-Dor, Mikulincer, Doron, & Shaver, 2010; Ellis et al., 2011). Attachment deactivating strategies typically tend to develop in response to the (perceived) unavailability of attachment figures, whereas attachment hyperactivating strategies typically develop as an often desperate attempt to elicit care and support from attachment figures who are unavailable or inconsistently available. Particularly when attachment figures are also the cause of abuse and neglect, the child feels trapped in an approach—avoidance conflict. This is typically thought to be reflected in disorganized attachment (Holmes, 2004; Main & Hesse, 1990), which is expressed in marked approach—avoidance and idealization—denigration cycles in relation to others. The feeling of confusion with regard to both the self and others that is characteristic of individuals with attachment disorganization—particularly those who are prone to depersonalization and dissociation—most closely resembles the identity diffusion that is typical of individuals with complex trauma and BPD. This assumption is consistent with studies reporting high rates of disorganized attachment in individuals with BPD (Fonagy & Luyten, 2016; Levy, Beeney, & Temes, 2011). More generally, a meta-analysis emphasized the particularly pernicious impact of complex trauma (Cyr, Euser, Bakermans-Kranenburg, & Van Ijzendoorn, 2010). In this meta-analysis, studies of children at risk (including, but not limited, to children with a history of maltreatment) showed less secure attachment, and more disorganized attachment in particular, compared with children growing up in low-risk families, with moderate to large effect sizes (Cohen's d = 0.67 and d = 0.77, respectively). Yet, very large differences in effect size were found when the researchers considered only the set of studies of maltreatment: children with a history of maltreatment were less secure (d =2.10) and more disorganized (d = 2.19) compared with other high-risk children (d = 0.48 and

d = 0.48, respectively). Hence, in individuals with complex trauma, particularly those with disorganized attachment, both the self and others may be felt as extremely positive or attractive, or as extremely negative or repulsive, depending on the context. This poses a serious challenge for the traumatized individual to develop a feeling of self-coherence and self-continuity. As noted, genetic vulnerability, including temperamental factors such as high levels of aggression or hypersensitivity to social information, may further complicate the development of a stable sense of self.

Trauma, Attachment, and Mentalizing

Research has amply shown an inverse relationship between stress or arousal and mentalizing (Arnsten, 1998; Mayes, 2000). As stress and arousal increase, there is a switch from relatively slow, controlled, and nuanced mentalizing, mainly underpinned by prefrontal executive functions, to more rapid, automatic, and typically biased mentalizing mediated by posterior cortical and limbic structures (Luyten & Fonagy, 2015). Hence, with the loss of controlled mentalizing, rapid (fight/flight/freeze) but biased mentalizing comes online.

Although this switch is adaptive from an evolutionary perspective in response to discrete trauma—as in such circumstances fast and automatic responses are much more efficient than slow, serial processes—when faced with chronic trauma, the individual is increasingly left with what we have termed "unmentalized frightening experiences" that lead to a constant pressure to externalize these experiences (in self-harm or re-enactments; see below, see also Table 1), leading to a constant feeling of dread and threatening disintegration of the self. Consistent with these assumptions, research has shown that the threshold for the switch from controlled to automatic mentalizing is related to exposure to early adversity as well as to the use of attachment deactivating and hyperactivating strategies (Luyten & Fonagy, 2015).

[insert Table 1 about here]

At the behavioral level, this means that complex trauma disrupts the capacity to frame and reframe adverse experiences. As individuals with complex trauma typically have severe problems relying on others, their access to the process of what we have termed "relational referencing" is very limited or sometimes completely absent. Relational referencing refers to the process by which a person can begin to meaningfully frame and reframe traumatic experiences. The result of this process is that the individual is able to mentalize what could not be mentalized before. This process hence enables what Kohut (1977) has referred to in a related context as a "restoration of the self", that is, the individual is able to develop a sense of self-worth, self-coherence, and self-continuity by relying on others (so-called self-objects) as a basis for the development of a sense of self.

From a mentalizing perspective, an experience thus becomes traumatic only when it leads to a feeling that one's mind is alone (Allen, 2012): that one is unable to "think the unthinkable" and "feel what one cannot feel". Being able to turn to a trusted other not only provides a sense of security and support, consistent with the attachment "secure base" notion, but it also enables one to recalibrate one's own mind. This typically involves a process of marked mirroring, and thus requires another person. Developmental studies indeed suggest that children—and adolescents and adults—experience a strong need for marked mirroring when they are faced with unexpected events and with adversity in particular (Fonagy, Gergely, & Target, 2007). Marked mirroring involves the process by which another person reflects back in a marked (i.e., digested) way what the other person cannot, or can only partially, mentalize. The notion of marking involves a process by which what is reflected back is modulated by another person in a caring and validating way. This is a trial-and-error process, just as in normal development, which thus largely depends on the mentalizing

capacity of the other person (typically an attachment figure). It is this experience of being held in mind by someone else that we see as crucial in restoring a sense of agency and control, and ultimately a sense of selfhood.

However, (complex) trauma typically disrupts this process and leads to the reemergence of non-mentalizing modes of experiencing both the self and other. There are three
non-mentalizing modes that are relevant here. First, psychic equivalence is highly
characteristic of traumatized individuals, as they begin to experience thoughts and feelings as
if they were real: thinking about trauma leads the person to relive the trauma. As a result,
individuals with complex trauma feel stuck in the painful past: they have been abused and
neglected, there is nothing anyone can do about this, there is no hope or solution, particularly
as they expect that others will abuse or neglect them again (an instance of psychic
equivalence, see also below). So-called "toxic" feelings of shame characteristic of individuals
with attachment trauma further emphasize the painfulness of traumatic experiences. These
findings are consistent with studies showing that the combination of trauma and impairments
in mentalizing are typical of patients with BPD (Fonagy et al., 1996), and are further
compounded by toxic shame, leading to a what has been called a shame-prone self (Hawes,
Helyer, Herlianto, & Willing, 2013; Rusch et al., 2007).

Because these feelings are often unbearable, a second non-mentalizing way of experiencing the self and others emerges, the teleological mode of functioning. In this mode, only actions may bring relief, and thus real (i.e., observable) changes have to be made in relation to the self and or others in a desperate attempt to find relief, support, or both. In teleological mode, studies suggest (Luyten & Fonagy, 2019), self-harm (e.g., cutting or excessive drinking) may be seen as an attempt to regulate extremely painful feelings, or promiscuity may result from desperate attempts to find love and care. Finally, pretend mode functioning may ensue: the individual loses contact with reality and becomes immersed in

continuous rumination about events from the recent or distant past, which in the extreme may give rise to feelings of dissociation as a defense against feelings of inner badness, emptiness, or worthlessness in a proportion of patients with BPD. Indeed, a recent meta-analysis of 10 studies, totaling 2,035 participants, found that levels of dissociation in BPD patients were significantly higher than in individuals diagnosed with other psychiatric disorders, but slightly lower than in patients with PTSD or dissociative disorders, mainly because of the large heterogeneity of dissociation in BPD patients (Scalabrini, Cavicchioli, Fossati, & Maffei, 2017).

This typically leads to a negative cascade of events in interpersonal relationships, as the mind of others interacting with the traumatized individual quickly tends to "freeze" as well. There is a huge body of literature that has empirically documented secondary or vicarious traumatization in mental health professionals working with traumatized individuals. Such responses typically include PTSD-like features and hostile—helpless states of mind in the mental health professional (Newell, Nelson-Gardell, & MacNeil, 2016). A meta-analysis of 38 published studies suggests that such reactions are most likely to occur among those with the highest caseload, a personal trauma history, and a poor social support network, although these associations were modest at best (Hensel, Ruiz, Finney, & Dewa, 2015). Similarly, strong countertransference responses (including anger and disgust) have been empirically demonstrated among mental health professionals working with (traumatized) BPD patients, regardless of the professionals' theoretical orientation (Gelso & Hayes, 2002). As a result, the traumatized person not only feels confirmed in his/her view that both the self and others are bad, but increasingly feels isolated and beyond help. The self becomes dominated by a feeling of nameless dread and threatens to disintegrate.

Research findings have shown that the capacity for mentalizing of attachment figures may be an important moderator of the relationship between (complex) trauma and interpersonal and interpersonal functioning (see Luyten & Fonagy, 2019, for a review).

The tendency of children with a history of maltreatment to inaccurately ascribe anger to others (Camras, Sachs-Alter, & Ribordy, 1996; Cicchetti & Curtis, 2005) is also particularly relevant. A meta-analysis suggests a similar bias in BPD patients, together with a bias toward perceiving neutral faces as negative (Daros, Zakzanis, & Ruocco, 2013). It is this bias, rooted in a sense of the self as bad, evil, neglected, or unworthy as a result of complex trauma, that allows us to better understand the tendency for re-enactment that is highly prevalent in individuals with complex trauma and in BPD patients. At least three types of reenactment of trauma can be distinguished. Revictimization is the best-known and most extensively documented type of re-enactment associated with complex trauma (Cloitre, Scarvalone, & Difede, 1997; Widom, 1999). While revictimization may be difficult to understand from a trait personality perspective, from an attachment and mentalizing perspective it is simply a repeat of the attachment template. Besides the anxiety, anger, and conflict, the abuser is also a source of care, love, or support. Furthermore, the anxiety this generates further intensifies the individual's attachment needs, paradoxically strengthening the relationship with the abuser (Allen, 2001). Hence, revictimization typically repeats the unsolvable approach—avoidance conflict typical of the disorganized/disoriented attachment pattern (Main & Hesse, 1990).

The re-enactment of neglect typically continues a pattern of neglect in earlier relationships. These individuals are hypersensitive to any type of perceived or real (emotional) neglect. This may lead to a state of righteous vindication in the traumatized individual, who may feel he/she has every reason to retaliate because of the apparent neglect by others (Clarkin, Kernberg, & Yeomans, 1999). Furthermore, the resulting aggression helps

to achieve a sense of coherence and continuity of the self ("I am the one who is always neglected by others"), and thus these individuals may become "addicted" to others who are neglectful or abusive (either in reality or as perceived). Finally, a substantial proportion of individuals with complex trauma tend to become perpetrators themselves and become involved in child maltreatment, bullying, and/or intimate partner abuse. For these individuals, observing certain features in others (e.g., aggression or happiness in their children or spouse) typically triggers their own past experiences of neglect and/or abuse. Evidence for the intergenerational transmission of trauma also comes from studies finding high rates of concordance of unresolved/disorganized attachment status in parents and infant disorganization (Cyr et al., 2010).

Communication, Epistemic Trust, and Impairments in Self and Identity in BPD

Although attachment and mentalizing approaches to complex trauma provide a powerful lens through which to view the impact of (complex) trauma on vulnerability to BPD and impairments in these individuals' sense of self in particular, there has been a recent transition in our thinking in this context in response to two sets of recent and striking findings. First, recent studies concerning the structure of psychopathology have identified a general psychopathology (or 'p') factor underlying common psychopathologies. Furthermore, models containing such a higher order p factor provide a better fit to the data than models with three high-order factors (internalizing, externalizing, and thought disorder). Since Caspi et al.'s (2014) seminal study in this area, several studies have replicated this higher order factor (Del Giudice, 2015; Laceulle, Vollebergh, & Ormel, 2015; Lahey et al., 2015; Murray, Eisner, & Ribeaud, 2016) and have found that the p factor increases the chance of most types of common mental health problems and negatively influences the course of these problems, similar to (complex) trauma (Nanni, Uher, & Danese, 2012; Scott, McLaughlin, Smith, &

Ellis, 2012; Teicher & Samson, 2013). But what is this p factor, and what are the mechanisms underpinning its association with all these other factors?

This brings us to a second set of recent findings, namely that resilience or so-called minimal impact resilience appears to be the normative response to trauma (Fonagy et al., 2017a; Fonagy, Luyten, Allison, & Campbell, 2017b). This notion refers to the observation that most people experience only temporary distress after single traumatic events (Type I trauma, as described earlier in this paper) (Bonanno & Diminich, 2013; Southwick, Bonanno, Masten, Panter-Brick, & Yehuda, 2014). This has been demonstrated in victims of terrorist attacks, individuals who have experienced unemployment, divorce, bereavement, a natural disaster, a life-threatening medical procedure, or been deployed in military operations (for a review, see Bonanno & Diminich, 2013). Hence, human beings seem to have a remarkable capacity for resilience. Interestingly, prospective studies in this context suggest that individuals who show chronic maladjustment after such events tend to have a history of previous trauma, poor social support, maladaptive emotion regulation strategies, or a combination of these factors (Denckla et al., 2018; Orcutt, Bonanno, Hannan, & Miron, 2014).

Both sets of findings on the p factor and the ubiquity of minimal impact resilience have led us to reverse the question that has largely dominated research on trauma—that is, it is not a focus on the development of vulnerability factors that may be implicated in explaining the relationship between trauma and maladjustment that will further elucidate the mechanisms involved in explaining the effects of trauma, but rather the absence of resilience. Kalisch, Mueller, and Tuscher's (2015) comprehensive positive appraisal style theory of resilience is particularly helpful in this context. According to this model, resilience involves three central mechanisms: (a) positive situation classification, (b) retrospective reappraisal of threat, and (c) inhibition of retraumatizing triggers. From a mentalizing perspective, all three

mechanisms involve the capacity to recalibrate one's mind when faced with a traumatic experience.

But where and when does this capacity to recalibrate one's social imagination in relation to others originate? Recent evolutionary and developmental accounts of the origins of social communication—and the capacity for epistemic trust in particular—provide important clues. Social imagination is instrumental in an intersubjectively generated selfhood in adaptively maintaining psychological functioning. This ongoing social imaginative process often goes awry—as evidenced by the prevalence of emotional distress and psychiatric disorder—but this potentially risky capacity for novelty and unruliness also allows the human mind to be creative and flexible. Mentalizing is the basic social cognitive tool that individuals use to constantly update and adaptively sketch out their imagined selves and the imagined minds of others.

However, in order for accurate and flexible mentalizing to become fully available to an individual, the underpinning mechanism of epistemic trust needs to be stimulated. Yet, epistemic trust is not a given: epistemic vigilance is a valuable adaptation that can protect the individual from misinformation, whether motivated by ignorance or the intention to deceive (Mascaro & Sperber, 2009; Sperber et al., 2010). In order for communication to be accepted as meaningful and relevant, epistemic trust between the "teacher" and the "learner" needs to be established (Wilson & Sperber, 2012).

Developmentally, attachment figures play a key role in this context, as they are the first to use particular communicative signals, known as ostensive cues, to put the infant into a so-called learning mode. It has been shown that humans display a species-specific sensitivity to specific nonverbal cues, such as eye contact, turn-taking contingent reactivity, being called by their name, and the use of a special tone of voice ("motherese") (Csibra & Gergely, 2011). These ostensive cues specifically trigger a *pedagogic stance* in the recipient—that is, they

signal that forthcoming communications are personally relevant and significant. As a result, the recipient feels recognized as a subjective, agentive self, which opens up a channel for the fast and efficient transmission of knowledge typical of human learning. We suggest that trauma becomes complex when it undermines the individual's epistemic trust and thus his/her sensitivity to ostensive cues and capacity to learn from their social environment, hampering the normative, evolutionary-based functions of attachment and mentalizing—that is, to facilitate social learning and salutogenesis (see Figure 1). In a study by Hanson et al. (2017), participants aged 12–17 who had experienced physical abuse were less able than their peers, who had no history of maltreatment, to correctly learn which stimuli were likely to result in reward, even after repeated feedback. The group who had experienced adversity also made less use of information about known rewards, and made decisions earlier in the learning process, apparently in the expectation of a less consistent and more random system of reward.

In a "good enough" caregiving environment, the infant finds their self experience represented accurately by the other, in the first instance through having their needs sensitively responded to. Experimental studies have suggested that the use of ostensive cues indeed triggers epistemic openness to knowledge in the infant (Csibra & Gergely, 2009). Ostensive cues communicate that the attachment figure recognizes the autonomy and agency of the recipient of the communication. The use of ostensive cues also signals that the knowledge that is conveyed is of use to the recipient (i.e., personally relevant and generalizable to other contexts and situations). Hence, ostensive cues provided by an attachment figure (and, later on, by other individuals who are perceived as trustworthy) provide a much-needed counterweight to epistemic vigilance.

Adversity and deprivation can generate chronic mistrust by inhibiting imagination, creating an overarching avoidance of mentalizing and an almost phobic avoidance of mental states, leaving the individual deeply vulnerable in most social situations. Even in the absence

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of such a pervasive failure of imagination, inadequate mentalizing may lead the traumatized individual to be biased in their perception of social reality and to misrepresent how others represent them, leading them to feel persistently misunderstood and to experience an intense and consistent sense of (epistemic) injustice. Further, the long-term outcome of epistemic isolation secondary to the failure of imagination as described here may create problems for individuals who have distorted personal narratives that generate inaccurate views of the self, so that even an accurate perception of one's personal narrative by others is not experienced as a match, and a painful experience of interpersonal alienation persists. Conversely, in yet other instances, deprivation and trauma may generate inappropriate trust. We understand such excessive *epistemic credulity* as being triggered by a hyperactive or unmoored imagination generating a personal narrative that is too diffuse to provide an accurate sense of differential awareness of others' capacity to perceive oneself. Excessive credulity results, as all personal narratives feel as if they "fit" sufficiently for trust to be generated, making the person vulnerable to exploitation.

Again, although epistemic trust and mentalizing are typically first acquired in the context of attachment relationships, we are mindful not to revert to a naive environmental position. As depicted in Figure 1, genetic predisposition and other individual differences as well as broader environmental factors (such as poverty and inequality) may make individuals more or less receptive to ostensive cues. What we are arguing is that because features of individuals with a history of complex trauma appeared to be remarkably stable, and even appeared to reflect a marked rigidity, laypersons, scientists, and clinicians alike tended to attribute these characteristics to the individual rather than to features of their own relationship to the individual. Hence, these features were seen as reflecting the patient's personality, self, or identity. The approach taken in this paper, however, suggests that the "personality" or "self' that characterizes these individuals is actually an adaptation strategy, and that the

"personality disorder" or "distorted sense of self or identity" reflects a disorder of social communication. Hence, the "personality" or "personality disorder" of these individuals is stable only insofar as the mechanisms that underpin its stability in place and active. This opens up important new perspectives for interventions and for conceptualizing the mechanisms of change involved in the treatment of individuals with complex trauma.

Clinical Implications: Toward a Generic Treatment for Complex Trauma?

There is increasing consensus that complex trauma has a negative impact on treatment outcomes (Fonagy et al., 2015; Teicher & Samson, 2013). If our assumption is correct, that is, that individuals with a history of complex trauma are characteristically closed off from the capacity for social recalibration of the mind, it means that most of these patients, as in normal development, will need another caring and sympathetic human mind as a critical prerequisite for change in order to foster (and often to establish) epistemic trust and the capacity for salutogenesis—the capacity to benefit from positive features in one's environment. In this context, we have described three systems of communication that seem to be at work, regardless of the type of psychosocial intervention, in bringing about therapeutic change in patients with complex trauma.

Communication System 1: The Teaching and Learning of Content

All effective psychosocial interventions for patients with complex seem to offer a convincing model of the mind to these patients—that is, a model that describes how their mind has been affected by trauma and how these problems can be resolved. Importantly, this model of the mind needs to be felt by the patient as mirroring, in a marked way, what they are experiencing. If it does not, nothing will happen, and mentalizing and epistemic trust will not be generated. To return to the concept of the imagined self, this stage of treatment consists of

the therapist working to show they are seeking to recognize the personal narrative of that individual patient and, by doing so, acknowledge the patient's individual agency and their imagined stance in relation to the social environment. Further, by elaborating on their particular therapeutic model, the therapist is working to reflect back to the patient their understanding of the patient's imagined self, and how it can be aligned with a sympathetic and functional social environment in the form of the specific method of treatment.

Communication System 2: The Re-Emergence of Robust Mentalizing

The model of the mind offered, insofar as it increases the understanding and framing of the trauma the patient has experienced, fosters the patient's sense of agency and subjectivity. Critical in this process appears to be the patient's ability to recognize that their personal narrative, or "imagined self" has been accurately represented back to them in their therapist's social imagination. In an individual with a very diffuse, loosely conceived imagined self, or one whose sense of self is, for example, persecutory and self-punishing, establishing this communication system represents significant mentalizing challenges for the therapist. But, once this alignment between the patient's self-narrative and the therapist's conception of it is achieved on a sufficiently benign and consistent basis, epistemic trust is stimulated in the patient. It thus becomes possible for the patient to think about their own mind in relation to the therapist's mind, and their therapist's mind in relation to their own, in a process of collaborative social communication, as occurs in normal development. In this process, balanced mentalizing can emerge and be rehearsed as the patient explores their own mind and their therapist's mind in relation to it. As the communication system becomes embedded, the capacity of the patient to reflect in increasingly sophisticated and differentiated ways about him/herself and others develops.

Communication System 3: The Re-Emergence of Social Learning

More robust mentalizing, together with increased epistemic trust, typically fosters the capacity for salutogenesis. The unfolding of this communication system largely takes place outside the therapeutic relationship, and different modalities widely differ in the extent to which they are able to attend to the wider social environment in this way. This is a stage of treatment that necessarily recognizes the limitations of therapy and challenges any notion of therapeutic omnipotence. However, the therapeutic relationship can have an ongoing role here, in creating an environment in which benign and accurate representations of the patient's self can be reflected back to the patient when the outside world undermines or distorts the patient's perception of the social environment and their position within it.

Conclusions and Directions for Future Research

This paper offers a novel approach to the relationship between BPD and (complex) trauma with a focus on impairments in self and identity. We argue that trauma—in particular, repeated, chronic attachment trauma— disrupts not just the attachment behavioral system and the capacity for mentalizing, but first and foremost the capacity for epistemic trust and the closely associated ability to benefit from the environment in recalibrating one's mind. The disruption of this capacity typically leads to a state of isolation, where the individual is cut off from interpersonal experiences that allow the recalibration of the mind when faced with traumatic experiences. The individual comes to be perceived and experienced by others, including healthcare professionals, as "difficult to treat" or "hard to reach." Personality psychologists who asses the temperamental and personality features of these individuals over time argue that these individuals are characterized by marked rigidity because of the high temporal stability of their personality features. Hence, their rigidity and stability are attributed to the individual, rather than to features of the type of relationship these individuals have with

others and their environment more generally. In contrast, this paper argues that the stable/rigid "personality" or "self" that is purportedly observed and objectively measured is in fact an adaptation strategy. In this view, a personality disorder is better conceptualized in terms of a disorder of social communication, and the purported rigidity and stability are only observed insofar as the mechanisms that underpin them are active.

These views not only suggest important new avenues for future research, but also open up important new perspectives for intervention. At this point, three strands of research may be particularly relevant to the views expressed in this paper. First, longitudinal studies are needed that trace the interactions among complex trauma, epistemic trust, and the development of resilience. Indeed, although a focus on the role of vulnerability factors (such as genetic predisposition and temperament) may be an important component of these studies, we believe that a focus on factors promoting resilience in the face of adversity at multiple levels of analysis (ranging from genes to behavior and sociocultural context) may prove to be a more productive avenue to understanding the long-term sequelae of trauma. Second, experimental research is needed to more firmly establish assumptions concerning causality that are part of the views expressed in this paper. Finally, sophisticated, multi-wave process—outcome studies are needed to model and test the purported interactions among the proposed three communication systems in the treatment of individuals with complex trauma, across treatment approaches. We hope that this paper will contribute to such research.

References

Adler, J. M., Chin, E. D., Kolisetty, A. P., & Oltmanns, T. F. (2012). The distinguishing characteristics of narrative identity in adults with features of borderline personality disorder: An empirical investigation. *Journal of Personality Disorders*, 26, 498-512. doi: 10.1521/pedi.2012.26.4.498

- Afifi, T. O., Mather, A., Boman, J., Fleisher, W., Enns, M. W., Macmillan, H., & Sareen, J. (2011). Childhood adversity and personality disorders: Results from a nationally representative population-based study. *Journal of Psychiatric Research*, 45, 814-822. doi: 10.1016/j.jpsychires.2010.11.008
- Allen, J. G. (2001). *Traumatic relationships and serious mental disorders*. Chichester, UK: Wiley.
- Allen, J. G. (2012). Mentalizing in the development and treatment of attachment trauma. London, UK: Karnac Books.
- American Psychiatric Association. (2013). *DSM-5: Diagnostic and statistical manual of mental disorders, fifth edition*. Arlington, VA: American Psychiatric Press.
- Anda, R. F., Felitti, V. J., Bremner, J. D., Walker, J. D., Whitfield, C., Perry, B. D., . . . Giles, W. H. (2006). The enduring effects of abuse and related adverse experiences in childhood. A convergence of evidence from neurobiology and epidemiology.
 European Archives of Psychiatry and Clinical Neuroscience, 256, 174-186. doi: 10.1007/s00406-005-0624-4
- Arnsten, A. F. T. (1998). The biology of being frazzled. *Science*, 280, 1711-1712. doi: 10.1126/science.280.5370.1711
- Asnes, A. G., & Leventhal, J. M. (2011). Connecting the dots in childhood and adolescent trauma. *Archives of Pediatrics & Adolescent Medicine*, 165, 87-89. doi: 10.1001/archpediatrics.2010.241
- Ball, J. S., & Links, P. S. (2009). Borderline personality disorder and childhood trauma: Evidence for a causal relationship. *Current Psychiatry Reports*, 11, 63-68. doi: 10.1007/s11920-009-0010-4
- Bargh, J. A. (2011). Unconscious thought theory and its discontents: A critique of the critiques. *Social Cognition*, 29, 629-647. doi: 10.1521/soco.2011.29.6.629

- Bargh, J. A. (2014). Our unconscious mind. *Scientific American*, *310*, 30-37. doi: 10.1038/scientificamerican0114-30
- Barnow, S., Ruge, J., Spitzer, C., & Freyberger, H. J. (2005). Temperament und Charakter bei Personen mit Borderline-Personlichkeitsstorung. [Temperament and character in persons with borderline personality disorder]. *Nervenarzt*, 76, 839-848. doi: 10.1007/s00115-004-1810-8
- Bartz, J. A., Zaki, J., Bolger, N., & Ochsner, K. N. (2011). Social effects of oxytocin in humans: Context and person matter. *Trends in Cognitive Sciences*, 15, 301-309. doi: 10.1016/j.tics.2011.05.002
- Beck, A. T., Freeman, A., & Davis, D. D. (2004). *Cognitive therapy of personality disorders*. New York, NY: Guilford Press.
- Belsky, D. W., Caspi, A., Arseneault, L., Bleidorn, W., Fonagy, P., Goodman, M., . . . Moffitt, T. E. (2012). Etiological features of borderline personality related characteristics in a birth cohort of 12-year-old children. *Development and Psychopathology*, 24, 251-265. doi: 10.1017/S0954579411000812
- Belsky, J., & Fearon, R. M. P. (2008). Precursors of attachment security. In J. Cassidy & P.R. Shaver (Eds.), *Handbook of attachment theory and research* (2nd ed., pp. 295-316). New York, NY: Guilford Press.
- Bender, D. S., Morey, L. C., & Skodol, A. E. (2011). Toward a model for assessing level of personality functioning in DSM-5, part I: A review of theory and methods. *Journal of Personality Assessment*, 93, 332-346. doi: 10.1080/00223891.2011.583808
- Bender, D. S., & Skodol, A. E. (2007). Borderline personality as a self-other representational disturbance. *Journal of Personality Disorders*, 21, 500-517. doi: 10.1521/pedi.2007.21.5.500

- Benjamin, L. S. (2005). Interpersonal theory of personality disorders. The structural analysis of social behaviour and interpersonal reconstructive therapy. In M. F. Lenzenweger & J. F. Clarkin (Eds.), *Major theories of personality disorder* (2nd ed., pp. 157-230). New York, NY: Guilford Press.
- Blatt, S. J., & Auerbach, J. S. (1988). Differential cognitive disturbances in three types of borderline patients. *Journal of Personality Disorders*, 2, 198-211. doi: 10.1521/pedi.1988.2.3.198
- Blatt, S. J., & Luyten, P. (2009). A structural-developmental psychodynamic approach to psychopathology: Two polarities of experience across the life span. *Development and Psychopathology*, 21, 793-814. doi: 10.1017/S0954579409000431
- Bonanno, G. A., & Diminich, E. D. (2013). Annual Research Review: Positive adjustment to adversity—trajectories of minimal-impact resilience and emergent resilience. *Journal of Child Psychology and Psychiatry*, *54*, 378-401. doi: 10.1111/jcpp.12021
- Bornovalova, M. A., Hicks, B. M., Iacono, W. G., & McGue, M. (2009). Stability, change, and heritability of borderline personality disorder traits from adolescence to adulthood: A longitudinal twin study. *Development and Psychopathology*, 21, 1335-1353. doi: 10.1017/S0954579409990186
- Bowlby, J. (1973). Attachment and loss, Vol. 2: Separation: Anxiety and anger. New York, NY: Basic Books.
- Bradley, R., & Westen, D. (2005). The psychodynamics of borderline personality disorder: A view from developmental psychopathology. *Development and Psychopathology*, 17, 927-957. doi: 10.1017/S0954579405050443
- Brakemeier, E. L., Dobias, J., Hertel, J., Bohus, M., Limberger, M. F., Schramm, E., . . .

 Normann, C. (2018). Childhood maltreatment in women with borderline personality

- disorder, chronic depression, and episodic depression, and in healthy controls. *Psychotherapy and Psychosomatics*, 87, 49-51. doi: 10.1159/000484481
- Bronfenbrenner, U. (1977). Toward an experimental ecology of human development.

 *American Psychologist, 32, 513-531. doi: 10.1037/0003-066x.32.7.513
- Bungert, M., Koppe, G., Niedtfeld, I., Vollstadt-Klein, S., Schmahl, C., Lis, S., & Bohus, M. (2015). Pain processing after social exclusion and its relation to rejection sensitivity in borderline personality disorder. *PLoS One*, 10, e0133693. doi: 10.1371/journal.pone.0133693
- Camras, L. A., Sachs-Alter, E., & Ribordy, S. C. (1996). Emotion understanding in maltreated children: Recognition of facial expressions and integration with other emotion cues. In M. D. Lewis & M. Sullivan (Eds.), *Emotional development in atypical children* (pp. 203-225). Mahwah, NJ: Lawrence Erlbaum Associates.
- Carlson, E. A., Egeland, B., & Sroufe, L. A. (2009). A prospective investigation of the development of borderline personality symptoms. *Development and Psychopathology*, 21, 1311-1334. doi: 10.1017/S0954579409990174
- Carlson, E. A., Sroufe, L. A., & Egeland, B. (2004). The construction of experience: a longitudinal study of representation and behavior. *Child Development*, 75, 66-83. doi: 10.1111/j.1467-8624.2004.00654.x
- Caspi, A., Houts, R. M., Belsky, D. W., Goldman-Mellor, S. J., Harrington, H., Israel, S., . . . Moffitt, T. E. (2014). The p factor: One general psychopathology factor in the structure of psychiatric disorders? *Clinical Psychological Science*, 2, 119-137. doi: 10.1177/2167702613497473
- Chanen, A. M., & Kaess, M. (2012). Developmental pathways to borderline personality disorder. *Current Psychiatry Reports*, *14*, 45-53. doi: 10.1007/s11920-011-0242-y

- Cicchetti, D., & Curtis, W. J. (2005). An event-related potential study of the processing of affective facial expressions in young children who experienced maltreatment during the first year of life. *Development and Psychopathology, 17*, 641-677. doi: 10.1017/S0954579405050315
- Cicchetti, D., & Toth, S. L. (2005). Child maltreatment. *Annual Review of Clinical Psychology*, 1, 409-438. doi: 10.1146/annurev.clinpsy.1.102803.144029
- Clarkin, J. F., Kernberg, O. F., & Yeomans, F. (1999). *Transference-focused psychotherapy* for borderline personality disorder patients. New York, NY: Guilford Press.
- Cloitre, M., Scarvalone, P., & Difede, J. (1997). Posttraumatic stress disorder, self- and interpersonal dysfunction among sexually retraumatized women. *Journal of Traumatic Stress*, 10, 437-452. doi: 10.1002/jts.2490100309
- Crawford, T. N., Cohen, P. R., Chen, H., Anglin, D. M., & Ehrensaft, M. (2009). Early maternal separation and the trajectory of borderline personality disorder symptoms.

 *Development and Psychopathology, 21, 1013-1030. doi: 10.1017/S0954579409000546
- Csibra, G., & Gergely, G. (2009). Natural pedagogy. *Trends in Cognitive Sciences*, 13, 148-153. doi: 10.1016/j.tics.2009.01.005
- Csibra, G., & Gergely, G. (2011). Natural pedagogy as evolutionary adaptation.

 *Philosophical Transactions of the Royal Society of London. Series B, Biological Sciences, 366, 1149-1157. doi: 10.1098/rstb.2010.0319
- Cyr, C., Euser, E. M., Bakermans-Kranenburg, M. J., & Van Ijzendoorn, M. H. (2010).
 Attachment security and disorganization in maltreating and high-risk families: A series of meta-analyses. *Development and Psychopathology*, 22, 87-108. doi: 10.1017/S0954579409990289

- Daros, A. R., Zakzanis, K. K., & Ruocco, A. C. (2013). Facial emotion recognition in borderline personality disorder. *Psychological Medicine*, 43, 1953-1963. doi: 10.1017/S0033291712002607
- de Aquino Ferreira, L. F., Queiroz Pereira, F. H., Neri Benevides, A. M. L., & Aguiar Melo, M. C. (2018). Borderline personality disorder and sexual abuse: A systematic review. *Psychiatry Research*, 262, 70-77. doi: 10.1016/j.psychres.2018.01.043
- De Panfilis, C., Riva, P., Preti, E., Cabrino, C., & Marchesi, C. (2015). When social inclusion is not enough: Implicit expectations of extreme inclusion in borderline personality disorder. *Personality Disorders: Theory, Research and Treatment, 6*, 301-309. doi: 10.1037/per0000132
- Del Giudice, M. (2015). The life history model of psychopathology explains the structure of psychiatric disorders and the emergence of the p factor. *Clinical Psychological Science*, *4*, 299-311. doi: 10.1177/2167702615583628
- Denckla, C. A., Mancini, A. D., Consedine, N. S., Milanovic, S. M., Basu, A., Seedat, S., . . . Koenen, K. C. (2018). Distinguishing postpartum and antepartum depressive trajectories in a large population-based cohort: The impact of exposure to adversity and offspring gender. *Psychological Medicine*, 48, 1139-1147. doi: 10.1017/S0033291717002549
- Distel, M. A., Middeldorp, C. M., Trull, T. J., Derom, C. A., Willemsen, G., & Boomsma, D. I. (2011). Life events and borderline personality features: The influence of gene-environment interaction and gene-environment correlation. *Psychological Medicine*, 41, 849-860. doi: 10.1017/S0033291710001297
- Distel, M. A., Trull, T. J., Derom, C. A., Thiery, E. W., Grimmer, M. A., Martin, N. G., . . . Boomsma, D. I. (2008). Heritability of borderline personality disorder features is

- similar across three countries. *Psychological Medicine*, *38*, 1219-1229. doi: 10.1017/S0033291707002024
- Distel, M. A., Willemsen, G., Ligthart, L., Derom, C. A., Martin, N. G., Neale, M. C., . . . Boomsma, D. I. (2010). Genetic covariance structure of the four main features of borderline personality disorder. *Journal of Personality Disorders*, 24, 427-444. doi: 10.1521/pedi.2010.24.4.427
- Ein-Dor, T., Mikulincer, M., Doron, G., & Shaver, P. R. (2010). The attachment paradox:

 How can so many of us (the insecure ones) have no adaptive advantages?

 Perspectives on Psychological Science, 5, 123-141. doi: 10.1177/1745691610362349
- Ellis, B. J., Boyce, W. T., Belsky, J., Bakermans-Kranenburg, M. J., & van Ijzendoorn, M. H. (2011). Differential susceptibility to the environment: An evolutionary—neurodevelopmental theory. *Development and Psychopathology, 23*, 7-28. doi: 10.1017/S0954579410000611
- Feldman, R., Vengrober, A., & Ebstein, R. P. (2014). Affiliation buffers stress: Cumulative genetic risk in oxytocin-vasopressin genes combines with early caregiving to predict PTSD in war-exposed young children. *Translational Psychiatry*, 4, e370. doi: 10.1038/tp.2014.6
- Fiske, S. T., & Taylor, S. E. (1991). *Social cognition* (2nd ed.). New York, NY: McGraw-Hill.
- Fonagy, P., Gergely, G., & Target, M. (2007). The parent-infant dyad and the construction of the subjective self. *Journal of Child Psychology and Psychiatry*, 48, 288-328. doi: 10.1111/j.1469-7610.2007.01727.x
- Fonagy, P., Leigh, T., Steele, M., Steele, H., Kennedy, R., Mattoon, G., . . . Gerber, A. (1996). The relation of attachment status, psychiatric classification, and response to

- psychotherapy. *Journal of Consulting and Clinical Psychology*, 64, 22-31. doi: 10.1037/0022-006X.64.1.22
- Fonagy, P., & Luyten, P. (2016). A multilevel perspective on the development of borderline personality disorder. In D. Cicchetti (Ed.), *Developmental psychopathology*. *Vol 3: Maladaptation and psychopathology* (3rd ed., pp. 726-792). New York, NY: Wiley.
- Fonagy, P., Luyten, P., & Allison, E. (2015). Epistemic petrification and the restoration of epistemic trust: A new conceptualization of borderline personality disorder and its psychosocial treatment. *Journal of Personality Disorders*, 29, 575-609. doi: 10.1521/pedi.2015.29.5.575
- Fonagy, P., Luyten, P., Allison, E., & Campbell, C. (2017a). What we have changed our minds about: Part 1. Borderline personality disorder as a limitation of resilience.

 *Borderline Personality Disorder and Emotion Dysregulation, 4, 11. doi: 10.1186/s40479-017-0061-9
- Fonagy, P., Luyten, P., Allison, E., & Campbell, C. (2017b). What we have changed our minds about: Part 2. Borderline personality disorder, epistemic trust and the developmental significance of social communication. *Borderline Personality Disorder and Emotion Dysregulation*, 4, 9. doi: 10.1186/s40479-017-0062-8
- Fraley, R. C., Roisman, G. I., Booth-LaForce, C., Owen, M. T., & Holland, A. S. (2013).
 Interpersonal and genetic origins of adult attachment styles: A longitudinal study from infancy to early adulthood. *Journal of Personality and Social Psychology*, 104, 817-838. doi: 10.1037/a0031435
- Gelso, C. J., & Hayes, J. A. (2002). The management of countertransference. In J. C. Norcross (Ed.), *Psychotherapy relationships that work* (pp. 267-283). Oxford, UK: Oxford University Press.

- Giourou, E., Skokou, M., Andrew, S. P., Alexopoulou, K., Gourzis, P., & Jelastopulu, E. (2018). Complex posttraumatic stress disorder: The need to consolidate a distinct clinical syndrome or to reevaluate features of psychiatric disorders following interpersonal trauma? *World Journal of Psychiatry*, 8, 12-19. doi: 10.5498/wjp.v8.i1.12
- Gobinath, A. R., Mahmoud, R., & Galea, L. A. (2014). Influence of sex and stress exposure across the lifespan on endophenotypes of depression: Focus on behavior, glucocorticoids, and hippocampus. *Frontiers in Neuroscience*, 8, 420. doi: 10.3389/fnins.2014.00420
- Green, J. G., McLaughlin, K. A., Berglund, P. A., Gruber, M. J., Sampson, N. A., Zaslavsky,
 A. M., & Kessler, R. C. (2010). Childhood adversities and adult psychiatric disorders in the national comorbidity survey replication I: Associations with first onset of DSM-IV disorders. *Archives of General Psychiatry*, 67, 113-123. doi: 10.1001/archgenpsychiatry.2009.186
- Gunderson, J. G., & Lyons-Ruth, K. (2008). BPD's interpersonal hypersensitivity phenotype:

 A gene-environment-developmental model. *Journal of Personality Disorders*, 22, 22-41. doi: 10.1521/pedi.2008.22.1.22
- Gunnar, M., & Quevedo, K. (2007). The neurobiology of stress and development. *Annual Review of Psychology*, 58, 145-173. doi: 10.1146/annurev.psych.58.110405.085605
- Han, S., & Northoff, G. (2009). Understanding the self: A cultural neuroscience approach.

 Progress in Brain Research, 178, 203-212. doi: 10.1016/S0079-6123(09)17814-7
- Hanson, J. L., van den Bos, W., Roeber, B. J., Rudolph, K. D., Davidson, R. J., & Pollak, S.
 D. (2017). Early adversity and learning: Implications for typical and atypical behavioral development. *Journal of Child Psychology and Psychiatry*, 58, 770-778.
 doi: 10.1111/jcpp.12694

- Hawes, D. J., Helyer, R., Herlianto, E. C., & Willing, J. (2013). Borderline personality features and implicit shame-prone self-concept in middle childhood and early adolescence. *Journal of Clinical Child and Adolescent Psychology*, 42, 302-308. doi: 10.1080/15374416.2012.723264
- Heim, C., & Binder, E. B. (2012). Current research trends in early life stress and depression:

 Review of human studies on sensitive periods, gene-environment interactions, and epigenetics. *Experimental Neurology*, 233, 102-111. doi: 10.1016/j.expneurol.2011.10.032
- Hensel, J. M., Ruiz, C., Finney, C., & Dewa, C. S. (2015). Meta-analysis of risk factors for secondary traumatic stress in therapeutic work with trauma victims. *Journal of Traumatic Stress*, 28, 83-91. doi: 10.1002/jts.21998
- Herman, J. (1992). Trauma and recovery. New York, NY: Basic Books.
- Holm, A. L., & Severinsson, E. (2008). The emotional pain and distress of borderline personality disorder: A review of the literature. *International Journal of Mental Health Nursing*, 17, 27-35. doi: 10.1111/j.1447-0349.2007.00508.x
- Holmes, J. (2004). Disorganized attachment and borderline personality disorder: A clinical perspective. *Attachment and Human Development*, *6*, 181-190. doi: 10.1080/14616730410001688202
- Horowitz, L. M., Wilson, K. R., Turan, B., Zolotsev, P., Constantino, M. J., & Henderson, L. (2006). How interpersonal motives clarify the meaning of interpersonal behavior: a revised circumplex model. *Personality and Social Psychology Review*, 10, 67-86. doi: 10.1207/s15327957pspr1001_4
- Hostinar, C. E., Sullivan, R. M., & Gunnar, M. R. (2014). Psychobiological mechanisms underlying the social buffering of the hypothalamic-pituitary-adrenocortical axis: A

- review of animal models and human studies across development. *Psychological Bulletin*, *140*, 256-282. doi: 10.1037/a0032671
- Hsu, D. T., Sanford, B. J., Meyers, K. K., Love, T. M., Hazlett, K. E., Walker, S. J., . . .
 Zubieta, J. K. (2015). It still hurts: altered endogenous opioid activity in the brain during social rejection and acceptance in major depressive disorder. *Molecular Psychiatry*, 20, 193-200. doi: 10.1038/mp.2014.185
- Jorgensen, C. R. (2006). Disturbed sense of identity in borderline personality disorder. *Journal of Personality Disorders*, 20, 618-644. doi: 10.1521/pedi.2006.20.6.618
- Jørgensen, C. R., Berntsen, D., Bech, M., Kjolbye, M., Bennedsen, B. E., & Ramsgaard, S. B. (2012). Identity-related autobiographical memories and cultural life scripts in patients with borderline personality disorder. *Consciousness and Cognition*, 21, 788-798. doi: 10.1016/j.concog.2012.01.010
- Kalisch, R., Muller, M. B., & Tuscher, O. (2015). A conceptual framework for the neurobiological study of resilience. *Behavioral and Brain Sciences*, 38, e92. doi: 10.1017/S0140525X1400082X
- Kendler, K. S., Aggen, S. H., Czajkowski, N., Roysamb, E., Tambs, K., Torgersen, S., . . . Reichborn-Kjennerud, T. (2008). The structure of genetic and environmental risk factors for DSM-IV personality disorders: A multivariate twin study. *Archives of General Psychiatry*, 65, 1438-1446. doi: 10.1001/archpsyc.65.12.1438
- Kernberg, O. F., & Caligor, E. (2005). A psychoanalytic theory of personality disorders. In
 M. F. Lenzenweger & J. F. Clarkin (Eds.), *Major theories of personality disorder*(2nd ed. ed., pp. 114-156). New York, NY: Guilford Press.
- Kingdon, D. G., Ashcroft, K., Bhandari, B., Gleeson, S., Warikoo, N., Symons, M., . . . Mehta, R. (2010). Schizophrenia and borderline personality disorder: Similarities and differences in the experience of auditory hallucinations, paranoia, and childhood

- trauma. *Journal of Nervous and Mental Disease*, *198*, 399-403. doi: 10.1097/NMD.0b013e3181e08c27
- Klahr, A. M., & Burt, S. A. (2014). Elucidating the etiology of individual differences in parenting: A meta-analysis of behavioral genetic research. *Psychological Bulletin*, 140, 544-586. doi: 10.1037/a0034205
- Kohut, H. (1977). *The restoration of the self*. New York, NY: International Universities Press.
- Kruglanski, A. W. (1989). Lay epistemics and human knowledge: Cognitive and motivational bases. New York, NY: Plenum Press.
- Kruglanski, A. W., & Webster, D. M. (1996). Motivated closing of the mind: "Seizing" and "freezing". *Psychological Review*, 103, 263-283. doi: 10.1037/0033-295X.103.2.263
- Laceulle, O. M., Vollebergh, W. A. M., & Ormel, J. (2015). The structure of psychopathology in adolescence: Replication of a general psychopathology factor in the TRAILS study. *Clinical Psychological Science*, *3*, 850-860. doi: 10.1177/2167702614560750
- Lahey, B. B., Rathouz, P. J., Keenan, K., Stepp, S. D., Loeber, R., & Hipwell, A. E. (2015).
 Criterion validity of the general factor of psychopathology in a prospective study of girls. *Journal of Child Psychology and Psychiatry*, 56, 415-422. doi: 10.1111/jcpp.12300
- Leichsenring, F., Leibing, E., Kruse, J., New, A. S., & Leweke, F. (2011). Borderline personality disorder. *Lancet*, *377*, 74-84. doi: 10.1016/S0140-6736(10)61422-5
- Levy, K. N., Beeney, J. E., & Temes, C. M. (2011). Attachment and its vicissitudes in borderline personality disorder. *Current Psychiatry Reports*, *13*, 50-59. doi: 10.1007/s11920-010-0169-8

- Livesley, J. (2008). Toward a genetically-informed model of borderline personality disorder. *Journal of Personality Disorders*, 22, 42-71. doi: 10.1521/pedi.2008.22.1.42
- Luijk, M. P., Roisman, G. I., Haltigan, J. D., Tiemeier, H., Booth-Laforce, C., van Ijzendoorn, M. H., . . . Bakermans-Kranenburg, M. J. (2011). Dopaminergic, serotonergic, and oxytonergic candidate genes associated with infant attachment security and disorganization? In search of main and interaction effects. *Journal of Child Psychology and Psychiatry*, *52*, 1295-1307. doi: 10.1111/j.1469-7610.2011.02440.x
- Lupien, S. J., McEwen, B. S., Gunnar, M. R., & Heim, C. (2009). Effects of stress throughout the lifespan on the brain, behaviour and cognition. *Nature Reviews Neuroscience*, *10*, 434-445. doi: 10.1038/nrn2639
- Luyten, P., & Blatt, S. J. (2013). Interpersonal relatedness and self-definition in normal and disrupted personality development: Retrospect and prospect. *American Psychologist*, 68, 172-183. doi: 10.1037/a0032243
- Luyten, P., & Fonagy, P. (2015). The neurobiology of mentalizing. *Personality Disorders:*Theory, Research and Treatment, 6, 366-379. doi: 10.1037/per0000117
- Luyten, P., & Fonagy, P. (2019). Mentalizing and trauma. In A. Bateman & P. Fonagy (Eds.),

 *Handbook of mentalizing in mental health practice (2nd ed.). Washington, DC:

 *American Psychiatric Publishing, Inc.
- Lyons-Ruth, K., & Jacobvitz, D. (2008). Attachment disorganization: Genetic factors, parenting contexts, and developmental transformation from infancy to adulthood. In J. Cassidy & P. R. Shaver (Eds.), *Handbook of attachment: Theory, research, and clinical applications* (2nd ed., pp. 666-697). New York, NY: Guilford Press.
- Main, M., & Hesse, E. (1990). Adult lack of resolution of attachment-related trauma related to infant disorganized/disoriented behavior in the Ainsworth strange situation:

- Linking parental states of mind to infant behavior in a stressful situation. In M. T. Greenberg, D. Cicchetti & E. M. Cummings (Eds.), *Attachment in the preschool years: Theory, research, and intervention* (pp. 339-426). Chicago, IL: University of Chicago Press.
- Marceau, K., Horwitz, B. N., Narusyte, J., Ganiban, J. M., Spotts, E. L., Reiss, D., & Neiderhiser, J. M. (2013). Gene-environment correlation underlying the association between parental negativity and adolescent externalizing problems. *Child Development*, 84, 2031-2046. doi: 10.1111/cdev.12094
- Mascaro, O., & Sperber, D. (2009). The moral, epistemic, and mindreading components of children's vigilance towards deception. *Cognition*, *112*, 367-380. doi: 10.1016/j.cognition.2009.05.012
- Mayes, L. C. (2000). A developmental perspective on the regulation of arousal states. Seminars in Perinatology, 24, 267-279. doi: DOI 10.1053/sper.2000.9121
- McLaughlin, K. A., Green, J. G., Gruber, M. J., Sampson, N. A., Zaslavsky, A. M., & Kessler, R. C. (2012). Childhood adversities and first onset of psychiatric disorders in a national sample of US adolescents. *Archives of General Psychiatry*, 69, 1151-1160. doi: 10.1001/archgenpsychiatry.2011.2277
- McWilliams, N. (2011). Psychoanalytic diagnosis: Understanding personality structure in the clinical process (2nd ed.). New York, NY: Guilford Press.
- Meichenbaum, D. (1994). A clinical handbook/practical therapist manual for assessing and treating adults with post-traumatic stress disorder (PTSD). Waterloo, Canada: Institute Press.
- Mendelsohn, M., Herman, J. L., Schatzow, E., Coco, M., Kallivayalil, D., & Levitan, J. (2011). *The trauma recovery group: A guide for practitioners*. New York, NY: Guilford Press.

- Mikulincer, M., & Shaver, P. R. (2007). *Attachment in adulthood: Structure, dynamics and change*. New York, NY: Guilford Press.
- Miller, G. E., Chen, E., & Zhou, E. S. (2007). If it goes up, must it come down? Chronic stress and the hypothalamic-pituitary-adrenocortical axis in humans. *Psychological Bulletin*, *133*, 25-45. doi: 10.1037/0033-2909.133.1.25
- Murray, A. L., Eisner, M., & Ribeaud, D. (2016). The development of the general factor of psychopathology 'p factor' through childhood and adolescence. *Journal of Abnormal Child Psychology*, 44, 1573-1586. doi: 10.1007/s10802-016-0132-1
- Mutlu, A. K., Schneider, M., Debbane, M., Badoud, D., Eliez, S., & Schaer, M. (2013). Sex differences in thickness, and folding developments throughout the cortex.

 NeuroImage, 82, 200-207. doi: 10.1016/j.neuroimage.2013.05.076
- Nanni, V., Uher, R., & Danese, A. (2012). Childhood maltreatment predicts unfavorable course of illness and treatment outcome in depression: A meta-analysis. *American Journal of Psychiatry*, 169, 141-151. doi: 10.1176/appi.ajp.2011.11020335
- Neumann, I. D. (2008). Brain oxytocin: A key regulator of emotional and social behaviours in both females and males. *Journal of Neuroendocrinology*, 20, 858-865. doi: 10.1111/j.1365-2826.2008.01726.x
- Newell, J. M., Nelson-Gardell, D., & MacNeil, G. (2016). Clinician responses to client traumas: A chronological review of constructs and terminology. *Trauma, Violence, & Abuse, 17*, 306-313. doi: 10.1177/1524838015584365
- Northoff, G., Heinzel, A., de Greck, M., Bermpohl, F., Dobrowolny, H., & Panksepp, J. (2006). Self-referential processing in our brain—a meta-analysis of imaging studies on the self. *NeuroImage*, *31*, 440-457. doi: 10.1016/j.neuroimage.2005.12.002

- Orcutt, H. K., Bonanno, G. A., Hannan, S. M., & Miron, L. R. (2014). Prospective trajectories of posttraumatic stress in college women following a campus mass shooting. *Journal of Traumatic Stress*, 27, 249-256. doi: 10.1002/jts.21914
- Panksepp, J., & Watt, D. (2011). Why does depression hurt? Ancestral primary-process separation-distress (PANIC/GRIEF) and diminished brain reward (SEEKING) processes in the genesis of depressive affect. *Psychiatry*, 74, 5-13. doi: 10.1521/psyc.2011.74.1.5
- Pervanidou, P., & Chrousos, G. P. (2012). Metabolic consequences of stress during childhood and adolescence. *Metabolism*, *61*, 611-619. doi: 10.1016/j.metabol.2011.10.005
- Pierro, A., & Kruglanski, A. W. (2008). "Seizing and freezing" on a significant-person schema: Need for closure and the transference effect in social judgment. *Personality and Social Psychology Bulletin, 34*, 1492-1503. doi: 10.1177/0146167208322865
- Pizzagalli, D. A. (2014). Depression, stress, and anhedonia: Toward a synthesis and integrated model. *Annual Review of Clinical Psychology*, *10*, 393-423. doi: 10.1146/annurev-clinpsy-050212-185606
- Richetin, J., Preti, E., Costantini, G., & De Panfilis, C. (2017). The centrality of affective instability and identity in borderline personality disorder: Evidence from network analysis. *PLoS One*, *12*, e0186695. doi: 10.1371/journal.pone.0186695
- Rogers, C. R. (1961). *On becoming a person: A therapist's view of psychotherapy*. London, UK: Constable.
- Rogers, C. R., & Dymond, R. F. (1954). *Psychotherapy and personality change: Coordinated research studies in the client-centered approach*. Chicago, IL: University of Chicago Press.
- Rusch, N., Lieb, K., Gottler, I., Hermann, C., Schramm, E., Richter, H., . . . Bohus, M. (2007). Shame and implicit self-concept in women with borderline personality

- disorder. *American Journal of Psychiatry*, *164*, 500-508. doi: 10.1176/ajp.2007.164.3.500
- Russo, S. J., & Nestler, E. J. (2013). The brain reward circuitry in mood disorders. *Nature Reviews Neuroscience*, *14*, 609-625. doi: 10.1038/nrn3381
- Salvatore, J. E., Haydon, K. C., Simpson, J. A., & Collins, W. A. (2013). The distinctive role of romantic relationships in moderating the effects of early caregiving on adult anxious-depressed symptoms over 9 years. *Development and Psychopathology*, 25, 843-856. doi: 10.1017/S0954579413000205
- Sansone, R. A., & Sansone, L. A. (2012). Chronic pain syndromes and borderline personality. *Innovations in Clinical Neuroscience*, 9, 10-14.
- Scala, J. W., Levy, K. N., Johnson, B. N., Kivity, Y., Ellison, W. D., Pincus, A. L., . . . Newman, M. G. (2018). The role of negative affect and self-concept clarity in predicting self-injurious urges in borderline personality disorder using ecological momentary assessment. *Journal of Personality Disorders*, 32, 36-57. doi: 10.1521/pedi.2018.32.supp.36
- Scalabrini, A., Cavicchioli, M., Fossati, A., & Maffei, C. (2017). The extent of dissociation in borderline personality disorder: A meta-analytic review. *Journal of Trauma & Dissociation*, 18, 522-543. doi: 10.1080/15299732.2016.1240738
- Schmideberg, M. (1959). The borderline patient. In S. Arieti (Ed.), *American handbook of psychiatry* (pp. 398-416). New York, NY: Basic Books.
- Scott, K. M., McLaughlin, K. A., Smith, D. A., & Ellis, P. M. (2012). Childhood maltreatment and DSM-IV adult mental disorders: Comparison of prospective and retrospective findings. *British Journal of Psychiatry*, 200, 469-475. doi: 10.1192/bjp.bp.111.103267

- Serafini, G., Pompili, M., Borgwardt, S., Houenou, J., Geoffroy, P. A., Jardri, R., . . . Amore, M. (2014). Brain changes in early-onset bipolar and unipolar depressive disorders: A systematic review in children and adolescents. *European Child and Adolescent Psychiatry*, 23, 1023-1041. doi: 10.1007/s00787-014-0614-z
- Shakoor, S., Jaffee, S. R., Bowes, L., Ouellet-Morin, I., Andreou, P., Happe, F., . . .

 Arseneault, L. (2012). A prospective longitudinal study of children's theory of mind and adolescent involvement in bullying. *Journal of Child Psychology and Psychiatry*, 53, 254-261. doi: 10.1111/j.1469-7610.2011.02488.x
- Sharp, C., Wright, A. G., Fowler, J. C., Frueh, B. C., Allen, J. G., Oldham, J., & Clark, L. A. (2015). The structure of personality pathology: Both general ('g') and specific ('s') factors? *Journal of Abnormal Psychology*, 124, 387-398. doi: 10.1037/abn0000033
- Shaw, P., Kabani, N. J., Lerch, J. P., Eckstrand, K., Lenroot, R., Gogtay, N., . . . Wise, S. P. (2008). Neurodevelopmental trajectories of the human cerebral cortex. *Journal of Neuroscience*, 28, 3586-3594. doi: 10.1523/JNEUROSCI.5309-07.2008
- Shedler, J., Beck, A., Fonagy, P., Gabbard, G. O., Gunderson, J., Kernberg, O., . . . Westen, D. (2010). Personality disorders in DSM-5. *American Journal of Psychiatry*, 167, 1026-1028. doi: 10.1176/appi.ajp.2010.10050746
- Skodol, A. E. (2012). Personality disorders in DSM-5. *Annual Review of Clinical Psychology*, 8, 317-344. doi: 10.1146/annurev-clinpsy-032511-143131
- Skodol, A. E., Gunderson, J. G., Pfohl, B., Widiger, T. A., Livesley, W. J., & Siever, L. J. (2002). The borderline diagnosis I: Psychopathology, comorbidity, and personality structure. *Biological Psychiatry*, *51*, 936-950. doi: 10.1016/S0006-3223(02)01324-0
- Smits, M. L., Feenstra, D. J., Bales, D. L., de Vos, J., Lucas, Z., Verheul, R., & Luyten, P. (2017). Subtypes of borderline personality disorder patients: A cluster-analytic

- approach. *Borderline Personality Disorder and Emotion Dysregulation, 4*, 16. doi: 10.1186/s40479-017-0066-4
- Solomon, E. P., & Heide, K. M. (1999). Type III trauma: Toward a more effective conceptualization of psychological trauma. *International Journal of Offender Therapy and Comparative Criminology*, 43, 202-210. doi: 10.1177/0306624X99432007
- Southwick, S. M., Bonanno, G. A., Masten, A. S., Panter-Brick, C., & Yehuda, R. (2014).

 Resilience definitions, theory, and challenges: Interdisciplinary perspectives.

 European Journal of Psychotraumatology, 5 doi: 10.3402/ejpt.v5.25338
- Spear, L. P. (2000). The adolescent brain and age-related behavioral manifestations.

 Neuroscience and Biobehavioral Reviews, 24, 417-463. doi: 10.1016/S0149-7634(00)00014-2
- Sperber, D., Clement, F., Heintz, C., Mascaro, O., Mercier, H., Origgi, G., & Wilson, D. (2010). Epistemic vigilance. *Mind & Language*, 25, 359-393. doi: 10.1111/j.1468-0017.2010.01394.x
- Spitzer, R. L., First, M. B., Shedler, J., Westen, D., & Skodol, A. E. (2008). Clinical utility of five dimensional systems for personality diagnosis: A "consumer preference" study. *Journal of Nervous and Mental Disease*, 196, 356-374. doi: 10.1097/NMD.0b013e3181710950
- Stepp, S. D., Lazarus, S. A., & Byrd, A. L. (2016). A systematic review of risk factors prospectively associated with borderline personality disorder: Taking stock and moving forward. *Personality Disorders: Theory, Research and Treatment*, 7, 316-323. doi: 10.1037/per0000186
- Strathearn, L. (2011). Maternal neglect: Oxytocin, dopamine and the neurobiology of attachment. *Journal of Neuroendocrinology*, 23, 1054-1065. doi: 10.1111/j.1365-2826.2011.02228.x

- Swain, J. E., Kim, P., Spicer, J., Ho, S. S., Dayton, C. J., Elmadih, A., & Abel, K. M. (2014).

 Approaching the biology of human parental attachment: Brain imaging, oxytocin and coordinated assessments of mothers and fathers. *Brain Research*, *1580*, 78-101. doi: 10.1016/j.brainres.2014.03.007
- Teicher, M. H., & Samson, J. A. (2013). Childhood maltreatment and psychopathology: A case for ecophenotypic variants as clinically and neurobiologically distinct subtypes.

 *American Journal of Psychiatry, 170, 1114-1133. doi: 10.1176/appi.ajp.2013.12070957
- Terr, L. C. (1991). Childhood traumas: An outline and overview. *American Journal of Psychiatry*, 148, 10-20. doi: 10.1176/ajp.148.1.10
- Torgersen, S., Lygren, S., Oien, P. A., Skre, I., Onstad, S., Edvardsen, J., . . . Kringlen, E. (2000). A twin study of personality disorders. *Comprehensive Psychiatry*, 41, 416-425. doi: 10.1053/comp.2000.16560
- Widom, C. S. (1999). Posttraumatic stress disorder in abused and neglected children grown up. *American Journal of Psychiatry*, *156*, 1223-1229. doi: 10.1176/ajp.156.8.1223
- Wilkinson, R., & Pickett, K. (2009). *The spirit level: Why equality is better for everyone*. London, UK: Penguin Books.
- Wilson, D., & Sperber, D. (2012). *Meaning and relevance*. Cambridge, UK: Cambridge University Press.
- Winsper, C., Hall, J., Strauss, V. Y., & Wolke, D. (2017). Aetiological pathways to borderline personality disorder symptoms in early adolescence: Childhood dysregulated behaviour, maladaptive parenting and bully victimisation. *Borderline Personality Disorder and Emotion Dysregulation*, 4, 10. doi: 10.1186/s40479-017-0060-x

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