REVIEW ARTICLE

Turkish Journal of Psychiatry 2024;35(2):137–149

https://doi.org/10.5080/u26852

Etiopathogenesis in the Development of Borderline Personality Characteristics in Children and Adolescents

Onat YETİM[1], Özhan YALÇIN[2]

ABSTRACT

The validity and clinical significance of the characteristics of borderline personality disorder (BPD) in adolescents are increasingly being recognized. The persistence of BPD characteristics in adolescence is high and is associated with negative interpersonal, academic, professional, and financial outcomes. In the literature, BPD characteristics observed in children and adolescents are explained with psychodynamic theories, developmental models, and evolutionary approaches. Emotional dysregulation, interpersonal dysfunction, impulsivity, and self-harming behavior, negative life events, temperament characteristics, neuropsychological dysfunctions, neuroanatomical, genetic, hormonal, and immunological factors may play a role in BPD etiopathogenesis. This review aims to address different approaches and relevant factors for the development of BPD. The articles published between 1968-2021 in the PubMed database were reviewed, and prominent studies were selected for evaluation. The importance of invalidating environment, epistemic freezing and hypermentalization, complex or attachment trauma is emphasized in psychodynamic and developmental literature. In the evolutionary approach, on the other hand, romantic relationships and the onset of reproduction are emphasized as the reason for the emergence of symptoms during adolescence, and it is argued that BPD is related to the rapid life history strategy. It is stated that a decrease in volume in the orbitofrontal cortex and anterior cingulate cortex, which are involved in top-down emotional processing, and an increase in the activity of thalamus and hippocampus regions, which are involved in bottom-up emotional processing are observed in adolescents with BPD characteristics when compared to healthy controls. It is thought that the increase in activation in the superior temporal gyrus and precuneus observed in adolescents with BPD features is a neural indicator of hypermentalization, and the increase in activation in the insula is a neural indicator of social pain. It has been reported that the decrease in resting heart rate and the increase in heart rate variability observed in adolescents with BPD symptoms are associated with the activation of the parasympathetic system. BPD in adolescents is a disorder that challenges clinicians in terms of diagnosis, differential diagnosis, and treatment. It is crucial to evaluate the factors related to etiopathogenesis in BPD in a multifaceted and detailed manner.

Keywords: Borderline Personality Disorder, Difficulty in Emotion Regulation, Mentalization, Trauma, Self-harming Behavior, Temperamental Characteristics

INTRODUCTION

Borderline personality disorder (BPD) has been determined to be a common mental disorder with rates varying between 0.7% and 1.8% in adults in population-based studies (Lieb et al. 2004, Winsper et al. 2020). The validity and clinical importance of BPD characteristics in adolescents is increasingly recognized (Sharp and Fonagy 2015, Winsper et al. 2016). Approximately 1% to 4% of adolescents meet the diagnostic criteria for BPD (Kaess et al. 2014). It is observed at a higher rate in girls during adolescence, especially in clinical samples (Grilo et al. 1996, Sharp et al. 2018). Prodromal features for BPD and features of BPD appear in early adolescence and their incidence gradually decreases after young adulthood. Therefore, BPD is thought to be a disorder with neurodevelopmental aspects (Chanen and Kaess 2012, Chanen et al. 2014, Kaess et al. 2014, Sharp et al. 2018).

Studies in adults and adolescents show that the descriptive features of BPD are best characterized by a phenotype that includes difficulty in emotion regulation, affective instability, interpersonal dysfunction, emotional interoception-sensation problems, hyper-reactive emotional activity, attachment problems, hypersensitivity to abandonment-rejection, acting out, self-harming behavior (SIB), suicide attempts and threats, chronic anxiety, decision-making and choice disorders, predisposition to psychotic and dissociative reactions, identity and selfhood problems, sexual problems, aggression and impulsivity (Gunderson 2007, McCloskey et al. 2009).

Received: 22.08.2021, **Accepted:** 25.04.2022, **Available Online Date:** 12.10.2023

[1]Psychiatrist, Ersin Arslan Research and Educational Hospital, Child and Adolescent Mental Health, Gaziantep, [2]Assoc. Prof., Private Practice, Child and Adolescent Psychiatrist Antalya, Turkey.

e-mail: onatyetim@gmail.com

Despite strong evidence supporting the benefits of diagnosing BPD during adolescence (Zanarini et al. 2018), stigmatization concerns still pose an obstacle to early diagnosis in clinical practice (Chanen et al. 2008a, Griffiths 2011, Kaess et al. 2014, Laurenssen et al. 2013). Among the debates about the diagnosis of BPD during adolescence are the developmental characteristics of adolescence and the problems of diagnostic continuity (Laurenssen et al. 2013). However, not being diagnosed brings the risks of misdiagnosis of adolescents with BPD features (Paris 2013) and missed early intervention opportunities. In addition, the "Diagnostic and Statistical Manual of Mental Disorders" (DSM-5) facilitated the diagnosis of personality disorder for those under the age of 18 (Newton-Howes et al. 2015). Studies have shown that "symptoms continue over time" for adolescents with BPD characteristics; He proposes three different trajectories in which "symptoms fade over time" and "other psychiatric disorders develop" (Winsper et al. 2015b). It is stated that the diagnostic continuity of BPD in adolescence is similar to that in adulthood, and that BPD symptoms continue to be seen in a significant portion of adolescents even after 20 years (Winsper et al. 2015a, 2015b). In a twin follow-up study of 10 years, young people with BPD features were evaluated 4 times at the ages of 14, 17, 20 and 24, and rank order stability was found to be 0.53-0.73 in this study (Bornovalova et al. 2009). It is stated that personality disorder characteristics of adolescents also show moderate continuity over time, as in adult samples (Sharp et al. 2018).

The aim of this study is to review the theoretical approaches and empirical findings for the explanation of the characteristics associated with borderline personality disorder features, which are becoming increasingly important in children and adolescents. For this purpose, studies related to borderline personality disorder were searched in PubMed index between 1968-2021. In this review, priority was given to studies aimed at children and adolescents.

Psychodynamic Theories

It is stated that the main developmental task of adolescence is the emergence of a "consistent sense of identity", which can be defined as "internal and interpersonal continuity", along with the existence of an autonomous and self-directing personality (Erikson 1968, McAdams, Olson 2010). The formation process of this identity and sense of self is characterized by inconsistency and confusion in some adolescents, in which case symptoms of personality disorder may occur (Gunderson et al. 2018).

Kernberg's (1984) model has common features with current developmental models. In addition, considering the severity of the symptoms, BPD stands between internalizing / externalizing disorders and psychotic disorders. Kernberg (1976) argues that the hallmark of BPD is the lack of integration in object representations. In borderline personality organization, patients use primitive and maladaptive defense mechanisms, such as splitting, for emotional regulation, and thus are unable to form holistic object representations. Kernberg (1976) argues that the split defense mechanism develops as a result of avoiding relational conflict with the caregiver.

Kohut (1977), the founder of selfhood psychology, focused more on the inadequacy of the caregiver. In Kohut's definition of "restoration of the self", it is stated that in the development of the sense of self, the individual develops the feelings of "self-worth", "self-consistency" and "self-continuity" by relying mainly on others (self-objects). However, when caregivers respond to their babies with anger or fear instead of mirroring, children internalize a foreign self - the "False Self" (Fonagy and Luyten 2009). Emotions conveyed by the caregiver through mirroring play an important role in the development of an integrated self.

Erikson (1986) draws attention to the mutual interaction of two dynamic elements ("identity synthesis" and "identity confusion") in the process of identity development. Contrary to identity synthesis in identity confusion, a consistent and functional sense of self is not formed and different identity-related features cannot be brought into a coherent whole. The widely accepted ground for the self in young people with BPD features is "identity confusion" (Kernberg 1983, Westen and Cohen 1993). Identity is generally assessed by self-narratives, which are indicative of people's ability to consistently integrate their autobiographical past and imagined future (McAdams and Mclean 2013). The causal and thematic coherence skills that develop during adolescence lead to the linking and integration of events in life, enabling self-narratives (Bluck and Habermas 2001). In adolescents with "identity confusion" and BPD characteristics, inconsistencies in self-narratives, fear of abandonment and insecurity are observed (Lind et al. 2019).

As a result, within the framework of psychodynamic theories, the importance of self and identity problems is revealed in the development of BPD in adolescents. Among these problems, "formation of holistic object representations", "development of sense of self and its integrity", "negative effects of caregivers on the self", "identity confusion" stand out.

Temperament Traits and Associated Neurobiological Factors

Inherited biology-based differences contribute to individuals' emotional and behavioral response tendencies, in short, to temperament (Bates et al. 2014, Davidson et al. 2000). When exposed to environmental risks, these reaction tendencies can turn into symptoms of psychopathology (Kagan 2013). In a twin follow-up study with 1116 children, low self-control

and high impulsivity levels at the age of 5 were found to be predictors of BPD characteristics at the age of 12 (Belsky et al. 2012). Children with persistent high impulsivity generally display a reactive, rapid, uncontrolled approach to rewarding stimuli without appropriately considering the potential negative consequences (Eisenberg et al. 2010, Martel 2013). Studies show that the interaction between temperamental characteristics such as impulsivity, which is persistent in the development of BPD, and environmental risks, such as parental maltreatment, stands out (Sharp et al. 2015). Children with problematic temperaments are more likely to be abandoned, neglected or abused by attachment figures who are also impulsive and hypersensitive to social stimuli (Crawford et al. 2009, Lyons-Ruth and Jacobvitz 2008).

In a follow-up study conducted with 2282 children and adolescents, it was found that high levels of negative affect, shyness and impulsivity, which are temperament traits, predicted the development of BPD (Stepp et al. 2014a). Persistent impulsivity temperament trait in children with "low level of responsibility-compatibility" personality traits; shyness and negative affect temperament traits are associated with "high level of neuroticism" (Sharp et al. 2015). In studies conducted with adults with BPD, a combination of "five-factor personality" features with "high level of neuroticism" and "low level of agreeableness and responsibility" is observed (Trull et al. 2003). It has been reported that there are significant correlations between BPD characteristics and "disinhibition" and "antagonism" temperament dimensions in adolescents (Jovev et al. 2013). Disinhibition is, like impulsivity, associated with "low level of responsibility" in adolescents; and antagonism is associated with "low level of compatibility". Among the five-factor personality traits, "low level of responsibility" reported in BPD includes impatient, impulsive individuals who have difficulty in organizing; on the other hand, "low level of compatibility" defines individuals who are insecure, utilitarian-Machiavellian or ruthless in interpersonal relationships (Stepp et al. 2014a). Temperament features such as impulsivity and anxiety are associated with individual differences in the functioning of two basic motivational systems in the brain. These systems, called the "behavioral activation system" (BAS) and "behavioral inhibition system" (BIS), regulate approaching and avoiding environmental stimuli (Barros-Loscertales et al. 2010, Fowles 1980). BIS is supported by neural structures including the amygdala and septo-hippocampal system and is mainly stimulated by serotonergic (5-HT) pathways. It is believed that this system prevents approaching-under-threat behaviors and mediates the anxious temperament characteristics (McNaughton and Corr 2004). It is stated that anxious temperament characteristics in childhood may reflect an internalizing trajectory towards self-harming behavior and BPD in adolescence in connection with depression and anxiety disorder (Beauchaine 2015, Neuhaus and Beauchaine 2013). BAS is supported by the mesolimbic dopamine system, which includes the ventral tegmental area, nucleus accumbens, and ventral striatum. It is thought that hypodopaminergic state and low-level BAS activation are associated with impulsive and irritable temperament features (Laakso et al. 2003, Sagvolden et al. 2005). The heterotypic comorbidity in BPD is thought to be related to the hypodopaminergic state (Beauchaine 2012). In a study, it was suggested that the temperament dimensions of harm avoidance and novelty seeking being high while reward dependence is low constitutes a biological vulnerability in terms of BPD development (Kaess et al. 2013). Harm avoidance is thought to be related to the serotonergic system (BIS), and novelty seeking is thought to be related to the dopaminergic (BAS) system (Cloninger et al. 1993).

As a result, it is observed that some temperamental characteristics of children and adolescents stand out in BPD. Among these, high impulsivity, high neuroticism, disinhibition and antagonism are the general characteristics emphasized in research.

Parental Attitudes and BPD Characteristics

The individuals with whom children and adolescents interact play an important role in reinforcing the negative affect of children. These established relationships are characterized by emotional devaluation, in which the emotional needs of the child are ignored or rejected (Crowell et al. 2009, Linehan 1993a). Linehan emphasizes the effect of the "invalidating environment", in which the infant's emotional experiences are not approved and supported by the caregiver, on the development of BPD characteristics (Linehan 1993a). The devaluing environment, compulsive and traumatic behaviors of parents and negative reinforcement processes contribute to the continuity of emotion regulation difficulties in vulnerable youth (Beauchaine et al. 2019, Crowell et al. 2009). Negativity in the family environment and maladaptive parental behaviors predict BPD characteristics in 11-year-old children (Winsper et al. 2012). In population-based follow-up studies, it was determined that maternal BPD diagnosis predicted BPD characteristics in adolescents (Barnow et al. 2013, Reinelt et al. 2014, Zalewski et al. 2014). Negative mother-child interactions such as rejection of the child or the parent's overprotective attitudes, the mother's oscillation between not caring for the child and clinging to the child, separation-individuation being perceived as a threat by the mother, not allowing/punishing separation-individuation/exploitation of feelings (intergenerational transmission of separation-individuation pathologies), taking normal developmental difficulties personally contribute to the transition of BPD symptoms from the mother to the

adolescent. It is possible that these mothers also experienced traumatic separation-individuation processes with their own mothers (Reinelt et al. 2014). However, the negative attitude of parents to children is mutual in nature. (El-Sheikh and Erath 2011). In studies on the development and parental attitudes of children with difficult temperaments, it has been found that difficult temperament has a reinforcing effect on negative parental attitudes and that these children have a high sensitivity towards caregivers and the environment (Boyce and Ellis 2005, Burt et al. 2005). It is thought that mutually negative and inconsistent behaviors may be effective in ending the conflict temporarily (Crowell et al. 2014a). However, these behaviors, which are reinforced over time, contribute to interpersonal problems, emotional regulation difficulties and the development of psychopathologies.

Cognition and Neuropsychological Findings in BPD

Individuals with BPD use more negative attitudes about themselves and others, blame themselves and others more in interpersonal relationships and show resentment, perceive the environment more hostile, and use non-adaptive regulation strategies. These findings are associated with difficulty in emotion regulation and impulsivity as a general attitude. According to neuropsychological studies; cognitive flexibility, changing the setup, shifting attention, making decisions/choices, sustained and selective attention, problem solving, self-suppression/inhibition skills, planning, strategy development, working memory are among the cognitive functions reported to be impaired in individuals with BPD (Massó Rodriguez et al. 2021). These neuropsychological impairments are associated with low stress coping skills, susceptibility to ruminative thoughts, impulsivity, and poor emotion regulation skills.

Social Cognition, Mentalization and Its Neurobiology in BPD

Mentalization is defined as the ability to understand the mental states of others (for example, emotions, intentions, attitudes and goals) (Han and Northoff 2009, Northoff et al. 2006). Hypermentalization mediates the relationship between attachment style and BPD characteristics in adolescents (Sharp et al. 2016). "Hypermentalization" is a social-cognitive process that involves making assumptions about other people's mental states that go beyond observable data (Sharp 2014). It therefore involves overloading mental states and misinterpreting them. Early onset, repetitive, chronic, namely cumulative trauma causes the child to be constantly alert and hypermentalization to occur in developmental process. Hypermentalization has a significant effect on individuals with BPD who have serious problems in maintaining continuity and consistency, especially in interpersonal relationships. "Epistemic freezing", on the other hand, refers to the tendency to defend existing knowledge structures even if they are false or misleading (Fiske and Taylor 1991). Such a defensive strategy, also called "cognitive closure", is observed especially in individuals who grew up in a traumatic environment (ie, high-risk environment for cumulative trauma). Current psychodynamic approaches, for example Fonagy et al.'s (2017) psychodynamic, socially oriented BPD model, places "epistemic freezing" and "mentalization" at the center of early-developing BPD characteristics. The development of the self is closely related to the capacity for reflective functioning or mentalization.

Theory of mind skills are necessary for correct empathy in childhood and for an abstract, symbolic social thinking in adolescence (Baron-Cohen et al. 2008, Frith 2007). Activation of the fronto-temporo-parietal regions and mirror neurons during childhood supports the understanding of the emotions and behaviors of others (Satpute and Lieberman 2006). Socially sensitive and consistent parental attitudes in the relationship between the child and the caregiver ensure that children have a high social processing capacity (Bornstein and Bornstein 2007). Neural development supported by these positive experiences in early childhood paves the way for secure attachment and healthy social communication behaviors (Malenka and Siegelbaum 2001). Negativities in the relationship with the caregiver in early childhood and subsequent relational traumas negatively affect the ability to perceive and respond to social cues (Fonagy et al. 2011). In a study related to theory of mind, a significant negative correlation was found between the ability to attribute first-degree and second-degree false beliefs at a 5-year-old and BPD characteristics at a 12-year-old (Belsky et al. 2012). Midline structures of the brain such as anterior cingulate cortex, dorsomedial prefrontal cortex, precuneus, posterior cingulate cortex, superior temporal gyrus, and temporoparietal regions play a role in an individual's understanding of themselves and others' mental states (Herpertz et al. 2017). The increase in activation in the superior temporal gyrus and precuneus in the mentalization network in adolescents with BPD features may be a neural indicator of hypermentalization (Sharp et al. 2011). The superior temporal gyrus is involved in perceiving the gestures and mimics of others, while the precuneus and insula are involved in establishing emotional empathy. In adolescents with BPD characteristics, an activity increase is observed in the insula pertaining to negativities in interpersonal relationships (Krauch et al. 2018). Adolescents with BPD characteristics have more emotional empathy and less cognitive empathy. While emotion regulation difficulties in adolescents with BPD were associated with an increase in emotional empathy; hypermentalization is associated with a decrease in cognitive empathy (Kalpakci et al. 2016).

In addition, social functioning problems are accompanied by white matter abnormalities in adolescents with BPD features. There is evidence that the developmental peak cannot be reached in white matter structure in adolescents with BPD features (New et al. 2013). Attention bias towards negative emotional stimuli can be observed in the presence of depressed mood in adolescents with BPD characteristics. Adolescents with BPD characteristics need more intense expressions in order to perceive emotions accurately.

Attachment and BPD Features

In a "good enough" environment, the infant's experiences are properly understood by the caregiver and the infant's needs are sensitively responded to. Parents' sensitivity towards their children is shown through a non-verbal communication that includes a clear reflection of the baby's emotions and ostensive cues (Fonagy et al. 2000). It is suggested that the use of demonstrative-observable cues (eg eye contact) initiates epistemic openness to knowledge in the infant (Csibra and Gergely 2009). There is a similar emphasis on "epistemic freezing" in both contemporary interpersonal theories (Benjamin 2005, Horowitz et al. 2006) and attachment theory (Mikulincer and Shaver 2007).

In attachment theory, it is emphasized that individuals with insecure attachment patterns continue their past patterns with a prominent rigidity in new attachment relations. The reason for this is the fear of loss of autonomy (loss of self) or loss of love of the attachment figure (loss of relatedness) (Mikulincer and Shaver 2007). Individuals with insecure attachment patterns have a strong tendency to maintain these patterns even if they are not approved by others. This shows the loss of the information flow that new attachment relationships provide to the individual, namely "epistemic freezing". In cross-sectional studies with adults, it is already known that there is a significant correlation between insecure attachment style and BPD (Levy 2005). In follow-up studies, it was determined that insecure attachment style in infancy and adolescence predicted the development of BPD (Carlson et al. 2009, Lyons-Ruth 2008). Adolescents with insecure attachment patterns are more prone to interpersonal hostility (Allen et al. 2007). Insecure attachment style is observed in 92-96% of individuals with BPD, and separation anxiety symptoms can be observed in childhood as a result of intergenerational transmission in both attachment and separation-individuation in individuals who develop BPD later on (Levy 2005). BPD features are more common in adolescents with an anxious-resistant-preoccupied attachment style (Rosenstein and Horowitz 1996). Studies have emphasized the findings that the correlation between insecure attachment patterns and BPD begins in childhood and continues into adulthood.

Childhood Adverse Life Events and BPD Characteristics

A history of physical, sexual abuse and emotional neglect was found to be more common in adults and adolescents with BPD than in psychiatric controls (Horesh et al. 2008, Ogata et al. 1990, Salzman et al. 1993, Stepp et al. 2014b, Zanarini et al. 1997). Studies have found that patients with BPD have a high rate of "early attachment trauma" or "complex trauma" (Ball and Links 2009, Chanen and Kaess 2012). "Complex trauma" describes long-term and cumulative childhood traumatic experiences, often involving neglect and/or abuse. (Asnes and Leventhal 2011). "Complex trauma"; it is often referred to as "Type II trauma", "attachment trauma", "relational trauma" or "developmental trauma". (Herman 1992 and Terr 1991).

Some researchers emphasized similar aspects of BPD with the diagnosis of "complex post-traumatic stress disorder" and suggested that complex trauma plays a role in the persistence of emotion dysregulation and causes the development of BPD (Ford and Courtois 2014, Trippany et al. 2006, van der Kolk et al. 1996). It is recommended to establish "Developmental Trauma Disorder" as a separate psychiatric diagnosis for the developmental effects of complex trauma in children and adolescents. The definition of trauma in "developmental trauma disorder" is limited as interpersonal and attachment trauma and the importance of attachment patterns in children and adolescents is emphasized. Diagnostic criteria in "developmental trauma disorder" are evaluated under 3 different categories. These are "somatic symptoms and difficulty in emotion regulation", "attention and behavioral problems", "self-regulation and interpersonal dysfunctions" (D'Andrea et al. 2012, Ford 2009). Diagnostic criteria for "developmental trauma disorder" in children and adolescents are highly correlated with BPD characteristics (Schmid et al. 2008). In line with the proposed diagnosis, insecure and disorganized attachment patterns are observed in a significant number of children exposed to abuse or neglect (Hipwell et al. 2000, Kim and Cicchetti 2004, Van Ijzendoorn et al. 1999, Weinfield et al. 2000).

It is believed that exposure to sexual abuse during childhood is one of the most important factors in the etiology of BPD (Westen et al. 1990). Exposure to both abuse and emotional neglect during childhood is associated with BPD characteristics observed in adolescents; however, it is stated that the relationship between exposure to emotional neglect and BPD characteristics is stronger compared to abuse (Zanarini et al. 2020).

"Complex trauma" does not usually occur in isolation; it is typically part of the broader 'risk environment' (Cicchetti, Toth 2005). A recent review of thirty-nine follow-up studies found that exposure to different types of traumas

such as emotional abuse and neglect, physical and sexual abuse; typically occurs in a broad context such as parental maltreatment, parental psychopathology, low socioeconomic status, domestic violence. This risky environment is closely related to the development of BPD (Stepp et al. 2016).

Studies on negative life events and their developmental effects in children reveal the importance of developmental timing (APA 2010). The first years of life, when rapid and important changes in brain development are experienced, are considered to be a sensitive period (Gee and Casey 2015). The caregiver-child interaction in this sensitive early childhood period is very important for the development of BPD. It is stated that the effect of maternal absence in early childhood on BPD symptoms cannot be explained by exposure to abuse alone (Lyons-Ruth et al. 2013). In a study, it was determined that 84% of individuals with BPD were exposed to neglect and emotional abuse by their parents before the age of 18 (Zanarini 2000). The chronicity of caregiver maltreatment is thought to be associated with developing BPD features. (Hecht et al. 2014). In addition, it was found that maltreatment in twins who were followed up from birth to 12 years of age strongly predicted BPD characteristics at the age of 12 in children with a family history of psychiatric disorders (Belsky et al. 2012).

Being bullied in childhood poses a risk for the development of BPD in early adolescence (Antila et al. 2017). In a 12-year follow-up study involving 6050 mothers and children, it was determined that continuous exposure to peer bullying during childhood was a risk factor for the development of BPD symptoms at the age of 12 (Wolke et al. 2012). In addition, broader environmental and sociocultural factors such as social inequality are thought to play an important role in explaining vulnerability for BPD. In societies with the highest levels of economic inequality, problems that may be associated with BPD, such as substance use disorder and adolescence pregnancies, are more common (Wilkinson and Pickett 2009). Studies have found that poverty requiring public assistance predicts BPD characteristics in adolescence (Stepp et al. 2014a, 2016).

"Trauma spectrum disorders" such as BPD in particular, attention deficit hyperactivity disorder (ADHD), dissociative experiences, aggression, post-traumatic stress disorder (PTSD), anorexia nervosa, bulimia nervosa, anxiety disorders, depression and substance use disorder, disinhibited social engagement disorder, reactive attachment disorder; the development of SIB which are associated with childhood traumatic experiences (Yildiz et al. 2020a, 2020b); are related to the interaction of many genetic, neurobiological, psychosocial and environmental factors, and they play an important role in the intergenerational transmission of trauma-related experiences and psychopathologies.

Evolutionary Approach

An important "behavioral theory of ecology" called the "life history theory" emphasizes the differential allocation of the organism's resources to physical growth and reproduction. Life history strategies are generally conceptualized as a slow or fast continuum (Grisevicius et al. 2011, Promislow and Harvey 1990). Each strategy manifests itself with different sexual, psychological and behavioral characteristics (Del Giudice 2009, Kaplan and Gangestad, 2005). "Slow life history strategies" are defined by a greater allocation of resources to growth, development, and parenting. In a slow life history, individuals begin to reproduce at a later age, establish consistent and long-lasting romantic relationships, and invest more in their children. "Fast life history strategies", on the other hand, are characterized by a greater allocation of resources to mate-finding efforts. In the rapid life history, individuals start to reproduce at a younger age, establish many and short-term romantic relationships, and invest less in their children (Egan et al. 2005, Weiss et al. 2004).

Studies have shown that there is a significant correlation between early environmental risk and rapid life history strategies. For example, individuals who are exposed to environmental risks have early sexual experiences, a tendency to have multiple and short-term romantic relationships, and impulsive behaviors (Belsky et al. 2012, Simpson et al. 2012). Children exposed to environmental cues such as harsh parenting attitudes, violence or abuse are more likely to develop an 'internal working model' in which future resource availability is unpredictable (Brüne 2016). This leads to a tendency to maximize short-term benefits in interpersonal relationships, namely the rapid life history strategy (Belsky et al. 1991).

It has been suggested that BPD characteristics are associated with the rapid life history strategy. The high rate of association of BPD symptoms such as impulsivity, difficulty in emotion regulation and self-harming behavior with negative childhood life events supports this view (Brüne 2016). Self-harming behavior in a devaluing environment is thought to be a strong signal to increase parental care (Fonagy et al. 2000). Threats to end children's lives are also a danger to the parents' own biological fitness and to the continuation of the lineage. However, psychiatric comorbidities associated with rapid life history strategies such as ADHD, substance use disorder and bulimia nervosa are observed at a high rate in individuals with BPD (Del Giudice 2014). Studies have shown that women with BPD have earlier sexual experiences and have more sexual partners than controls (Sansone et al. 2008, 2011). In addition, unplanned pregnancies are more common in adolescents with BPD features (De Genna et al. 2012). It is thought that the internalization symptoms observed in young people with BPD features may serve to decrease the metabolic rate, increase the fat ratio, and thus lead to earlier

menarche (Belsky et al. 1991). However, there is not enough evidence for physical maturation such as early menarche age in adolescents with BPD features (Brüne et al. 2010). However, symptoms that are indicators of a rapid life history strategy such as risky behaviors, impulsivity and self-harm in BPD gradually decrease with age (Morgan et al. 2013). Rapid strategies aiming to maximize reproductive success early in life, such as in adolescence, become dysfunctional with age, and both symptoms and rapid strategies decrease considerably in the postmenopausal period.

In evolutionary approaches, attention is drawn to the onset of romantic relationships and reproduction with sexual maturation as the reason for the emergence of BPD symptoms during adolescence (Brüne 2016). It is stated that individuals with BPD have sexual experiences at an earlier age and tend to have short-term romantic relationships (Sansone et al. 2008, 2011). These findings are thought to be related to the negative attitudes or absence of the opposite sex parent. It was determined that the transition to puberty and sexual maturation were earlier in girls who grew up in a house without male caregivers (Belsky et al. 1991). Early menarche age, which is observed as a result of environmental risks such as the absence of the opposite sex parent, means more resources are allocated to mate selection together with the ability to start reproduction (Belsky et al. 1991, Ellis 2004). Thus, young people may tend to have many and short-term romantic relationships (Egan et al. 2005, Weiss et al. 2004).

Biosocial Development Model

The "biosocial development model" (Crowell et al. 2009), which is the developmental model for BPD, is based on Linehan's (1993a) biosocial theory, which emphasizes biologically based emotional vulnerability. Biosocial theory together with dialectical behavioral theory forms the basis of a cognitive-behavioral therapy approach that emphasizes the role of emotion regulation difficulties in the development and persistence of BPD (Linehan 1993a, 1993b). In the biosocial theory, it is stated that difficulty in emotion regulation is at the center of BPD-related symptoms such as SIB, suicidal behavior and impulsive behaviors (i.e. substance use, risky sexual behavior). In addition, Linehan (1993a) emphasizes the "invalidating environment" effect on the development of BPD characteristics, in which the infant's emotional experiences are not approved and supported by the caregiver. In the biosocial theory, it is stated that the temperament characteristics of the child and the devaluing environment mutually affect each other (Chapman 2019). For example, when children with high impulsivity are exposed to devaluing parental attitudes, they are under a high risk of developing emotional dysregulation (Winsper et al. 2017).

Difficulty in Emotion Regulation in BPD, Neurobiology, Neuroimaging Findings

In individuals with BPD, exaggerated emotional reactions to interpersonal difficulties, frequently experiencing feelings of guilt-shame, experiencing extreme positive and negative emotions, decreased emotional awareness, tendency to blame oneself and others, catastrophizing, inability to exhibit appropriate individual/autonomous coping skills, and ability to compromise and adapt. Emotional dysregulation is common due to retardation (Rodriguez et al. 2021). Emotion regulation capacity depends on the gradual development of the fronto-striatal circuit from early childhood to young adulthood (Rubia 2013, Silvers et al. 2014). This development is strongly influenced by environmental factors such as cultural socialization processes, quality of interaction with caregivers and peers (Baarendse et al. 2013, Whittle et al. 2016). Stressful life events and exposure to trauma in childhood negatively affect the development of neural regions responsible for emotion regulation. It is thought that exposure to stressful life events causes gray matter volume loss in the amygdala and thus increases amygdala activation (Schulze et al. 2016). Areas that may be associated with difficulty in emotion regulation in BPD include amygdala, hippocampus, insula, rostral anterior cingulate cortex (emotion regulation difficulties) / dorsal anterior cingulate cortex, orbitofrontal cortex, ventrolateral and dorsolateral prefrontal cortex, thalamus, and hippocampus (emotion regulation, emotion recognition, impulsivity, sensitivity to immediate and late rewards, decision making) / inferior frontal gyrus (anger and aggression) / uncinate and occipito-frontal fascicles (fibers connecting the limbic system to the frontal cortex-decreased top-down control of emotional and aggressive responses) (Buchheim et al. 2013, Chanen et al. 2008b, Goodman et al. 2011, Guyer et al. 2016, Jovev et al. 2013, Krauch et al. 2018, Leichsenring et al. 2011, Mühlberger et al. 2010, New et al. 2013, Schmahl et al. 2003, Schulze et al. 2016, Stanley et al. 2018, Tabibnia et al. 2011, Takahashi et al. 2009, Whittle et al. 2009).

A decrease in volume was detected in the anterior cingulate cortex region in adolescents with BPD characteristics compared to healthy controls (Goodman et al. 2011, Whittle et al. 2009). This decrease in volume, which leads to a decrease in top-down control, was found to be significantly associated with parasuicidal behaviors (Whittle et al. 2009). A decrease in orbitofrontal cortex volume was found in adolescents with BPD characteristics compared to healthy controls (Chanen et al. 2008b). However, contrary to the adult literature (Schmahl et al., 2003), volume differences in the hippocampus and amygdala were not detected in adolescents with BPD characteristics compared to controls. Findings suggest that changes in these regions may occur in the later stages of the disorder due to decreased volume in the orbitofrontal cortex

(Chanen et al. 2008b). Decreased volume in the orbitofrontal cortex negatively affects emotion regulation and learning skills for socially appropriate behaviors (Buchheim et al. 2013). Contrary to studies conducted with adults with BPD (Schulze et al. 2016), no increase in amygdala activation was observed in case of interpersonal rejection in adolescents with BPD characteristics (Krauch et al. 2018). However, similar to studies conducted with adults with BPD (Schulze et al. 2016), an increase in activation in the left posterior insula was found in case of interpersonal rejection in adolescents with BPD features (Krauch et al. 2018). These neurobiological factors may also be involved in ADHD and specific learning disability, which pose a risk for the development of BPD.

Impulsivity-Self-Injurious Behavior and Associated Neurobiological Factors in BPD

The high level of psychiatric symptoms in adolescents with SIB is associated with BPD characteristics and exposure to trauma (Slavin-Stewart et al. 2018). In developmental approaches, it is suggested that self-harming behavior is a precursor of BPD (Homan et al. 2017, Gratz et al. 2014). Similar to studies conducted with adults (Silbersweig et al. 2007), both gray and white matter abnormalities are observed in the fronto-limbic areas involved in impulse control in adolescents with BPD features (Bozzatello et al. 2019). In studies on SIB, it was found that the pain threshold values of adult and adolescent patients with BPD were higher than those of healthy controls (Ludäscher et al. 2015, Schmahl et al. 2004). Nociceptive stimulation leads to a decrease in stress levels in individuals with BPD (Willis et al. 2017). This increased pain threshold is explained by two mechanisms. In the first of these, it is emphasized that the functional connection between the amygdala and the superior temporal gyrus is impaired as a result of amygdala deactivation (increased top-down inhibition) observed together with the activation of the medial prefrontal cortex. In individuals with BPD, this impaired connection improves after self-harming behavior (Reitz et al. 2015). In the second mechanism, it is stated that the increased functional connection between the posterior insula (pain-hurt associated with emotion) and the dorsolateral prefrontal cortex observed in individuals with BPD leads to hypoalgesia (Niedtfeld et al. 2012). Endogenous opioid system disorders may also be associated with depersonalization, hypoalgesia and dissociation during SIB (Fossati 2012, Herpertz and Bertsch 2015, Stanley and Siever 2010).

According to a recent review, the decreased connectivity between the precuneus and right fronto-parietal regions observed in BPD may lead to problems in self-referencing and self-seeing/evaluation skills. According to some researchers, this situation disrupts the integration of internal representations with stimuli from the environment, which may be related to impulsivity and SIB (Rodriguez et al. 2021).

Recently, the number of studies on "Heart Rate Variability" (HRV) in BPD and self-harming behavior has been increasing. The "Polyvagal Theory Model" provides important information about the neurobiology of social behavior (Porges 2009). In this model, vagal tone represents the functioning of the neurovegetative system based on the parasympathetic/sympathetic system balance. Increasing vagal tone both slows down the heart and makes the heart rate more variable (i.e., there is more beat variation between heartbeats) (Bourvis et al. 2017). Increased activation of the parasympathetic system is observed in adolescents with BPD features (Koenig et al. 2017).

BPD and Genetics

An average heritability of 49% is estimated for personality traits (Polderman et al. 2015). In passive gene-environment correlation, it is emphasized that parents both present their children with an environment related to their own genotype and transmit their own genotypes (Kendler and Baker 2007). Genes that cause vulnerability for BPD development also increase the possibility of exposure to environmental risk factors (Stanley et al. 2018). It has been reported that there is a higher correlation in monozygotic twins (0.66) than dizygotic twins (0.29) in terms of BPD characteristics, and genetic factors are responsible for 66% of the variance in BPD symptoms (Belsky et al. 2012). Among the aforementioned genes associated with BPD; oxytocin receptor gene (OXTR), HPA axis-related FK 506 binding protein gene (FKBP5), 5-HTTLPR serotonin transporter gene stand out. In addition, dopamine-related genes, brain-derived neurotrophic factor (BDNF), vasopressin receptor 1A gene, sodium channel voltage-gated type IX alpha subunit gene, dihydropyrimidine dehydrogenase (DPYD) gene 1, Plakophilin-4 (PKP4) gene, serine incorporator 5 The (SERINC5) gene and the mu-opioid receptor gene (OPRM177G), which is also involved in the regulation of attachment and separation behaviors, may also be associated with the development of BPD (Cicchetti et al. 2014, Plomin 2013, Hankin et al. 2011, Perez-Rodriguez et al. 2018), Sela and Barbaro 2018).

Hormones, Neuropeptides, Immunological Responses and BKB

An increase in pituitary gland volume and changes in plasma dehydroepiandrosterone sulfate (DHEA-S) levels have been reported among hormonal, immunological and neuropeptide-related changes pertaining to BPD characteristics (Conti et al. 2013, Jovev et al. 2008). In BPD, chronic stress in childhood and adolescence initially causes HPA axis hyperactivity; however, it is thought to cause HPA axis hypoactivation (low cortisol response) in the stress response later in life (Miller et al. 2007). In the neuropeptide model of BPD, it is suggested that abnormalities in attachment and affiliative

systems modulated by neuropeptides such as oxytocin and endogenous opioids related to interpersonal relationships may be the cause of impulsive behaviors after social rejection or abandonment (Fossati 2012, Herpertz and Bertsch 2015, Stanley and Siever 2010).

CONCLUSION

Broad spectrum features such as parent-child relationship pattern, parental psychopathology, child psychopathologies, psychodynamic and attachment-related factors, environmental factors, temperamental characteristics, neurobiological processes, genetic factors, emotion regulation difficulties, childhood traumas and negative life events, exposure to sexual abuse, impulsivity related features, theory of mind deficiencies and hypermentalization, hormonal causes, neurotransmitter and neuropeptide function problems, and epigenetic processes play a role in the development of BPD.

In psychodynamic and developmental approaches to BPD, emphasis is put on interpersonal dysfunction, difficulty in emotion regulation and impulsivity. The invalidating environment (Linehan 1993a), epistemic freezing and mentalization (Fonagy et al., 2017), complex or attachment trauma (van der Kolk et al., 2009) play a role in the development of BPD. The diagnosis of Developmental Trauma Disorder is recommended as a separate psychiatric diagnosis for the developmental effects of complex trauma in children and adolescents (van der Kolk et al., 2009).

In the evolutionary approach, the onset of romantic relationships and reproduction with sexual maturation is emphasized as the reason for the emergence of BPD symptoms during adolescence, and it is argued that BPD is associated with the strategy of rapid life history (Brüne 2016). Rapid strategies aiming to maximize reproductive success early in life, such as in adolescence, become dysfunctional with age, and both symptoms and rapid strategies decrease considerably in the postmenopausal period.

Adolescents with BPD characteristics have decreased volume in the orbitofrontal cortex (Chanen et al. 2008b) and anterior cingulate cortex (Whittle et al. 2009), which are involved in top-down emotional processing compared to healthy controls; an increase in activation is observed in the thalamus and hippocampus regions, which are involved in bottom-up emotional processing (Krauch et al. 2018). However, contrary to the adult literature (Schmahl et al. 2003), volume differences in the hippocampus and amygdala are not observed in adolescents with BPD characteristics compared to controls (Brunner et al. 2010). It is thought that changes in these regions occur in the later stages of the disorder due to decreased volume in the orbitofrontal cortex (Chanen et al. 2008b).

Since our study is limited to a review of BPD, conducting studies that reveal the importance of the findings we have discussed in applied clinical contexts will guide clinicians in terms of diagnosis, differential diagnosis and treatment of BPD. Especially mental health experts working in the field of adolescents should handle the problem considering its different dimensions. Since studies focusing on childhood are important in terms of their effects in predicting BPD during adolescence and young adulthood, it would be beneficial to increase studies for this age group. Experimental studies on various features of BPD and comparisons to be made between samples of adolescents with and without BPD features will help clarify the distinctive features of BPD.

Longitudinal studies following the long-term effects of developmental trauma will contribute to understanding the relationship between trauma and BPD characteristics. This study is based on a review of studies conducted among child and adolescent population in the field of BPD in the last 50 years. Most of the studies we have discussed are of a relational nature. Therefore, the generalizability and effect size of the findings are limited. Studies conducted in the field of BPD in our country will contribute to the literature while supporting the validity of the findings.

REFERENCES

Allen JP, Porter M, McFarland C et al. (2007) The relation of attachment security to adolescents' paternal and peer relationships, depression, and externalizing behavior. Child Development 78:1222-39.

American Psychological Association (2010) Resilience and Recovery After War: Refugee Children and Families in the United States. Washington, DC: Author. Retrieved from http://www.apa.org/pi/families/refugees and prospect, s, 27.

Antila H, Arola R, Hakko H et al. (2017) Bullying involvement in relation to personality disorders: A prospective follow-up of 508 inpatient adolescents. Eur Child Adolesc Psychiatry 26:779-89.

Asnes AG, Leventhal JM (2011) Connecting the dots in childhood and adolescent trauma. Archives of Pediatrics & Adolescent Medicine 165:87–9.

Baarendse PJ, Counotte DS, O'Donnell P et al. (2013) Early social experience is critical for the development of cognitive control and dopamine modulation of prefrontal cortex function. Neuropsychopharmacology 38:1485–94.

Ball JS, Links PS (2009) Borderline personality disorder and childhood trauma: Evidence for a causal relationship. Curr Psychiatry Rep 11:63–8.

Barnow S, Aldinger M, Arens EA et al. (2013) Maternal transmission of borderline personality disorder symptoms in the community-based Greifswald family study. J Pers Disord 27:806–19.

Baron-Cohen S, Golan O, Chakrabarti B et al. (2008) Social cognition and autism spectrum conditions. Social Cognition and Developmental Psychopathology, C Sharp, P Fonagry, I Goodyer (Ed), Oxford, UK: Oxford University Press, s. 29-56.

Barros-Loscertales A, Ventura-Campos N, Sanjuan-Tomas A et al. (2010) Behavioral activation system modulation on brain activation during appetitive and aversive stimulus processing. Soc Cogn Affect Neurosci 5:18–28.

Bates JE, Schermerhorn AC, Petersen IT (2014) Temperament concepts in developmental psychopathology. Handbook of Developmental Psychopathology, 3. Baskı, KD Rudolph, M Lewis, KD Rudolph (Ed), New York, Springer, s.311-29.

Beauchaine TP (2012) Physiological markers of emotion and behavior dysregulation in externalizing psychopathology. Monogr Soc Res Child Dev 77:79-86.

Beauchaine TP (2015) Future directions in emotion dysregulation and youth psychopathology. J Clin Child Adolesc Psychol 44:875-96.

Beauchaine TP, Hinshaw SP, Bridge JA (2019) Nonsuicidal self- injury and suicidal behaviors in girls: The case for targeted prevention in preadolescence. Clin Psychol Sci 7:643-67.

Belsky J, Steinberg L, Draper P (1991) Childhood experience, interpersonal development, and reproductive strategy: An evolutionary theory of socialization. Child Dev 62:647-70.

Belsky J, Schlomer GL, Ellis BJ (2012) Beyond cumulative risk: Distinguishing harshness and unpredictability as determinants of parenting and early life history strategy. Dev Psychol 48:662-73.

Belsky DW, Caspi A, Arseneault L et al. (2012) Etiological features of borderline personality related characteristics in a birth cohort of 12-year-old children. Dev Psychopathol 24:251-65.

Benjamin LS (2005) Interpersonal theory of personality disorders. The structural analysis of social behaviour and interpersonal reconstructive therapy. Major Theories of Personality Disorder, 2.Baskı, MF Lenzenweger, JF Clarkin (Ed), New York, NY, Guilford Press, s. 157-230.

Bluck S, Habermas T (2001) Extending the study of autobiographical memory: Thinking back about life across the life span. Rev Gen Psychol 5:135–47.

Bornovalova MA, Hicks BM, Iacono WG et al. (2009) Stability, change, and heritability of borderline personality disorder traits from adolescence to adulthood: A longitudinal twin study. Dev Psychopathol 21:1335–53.

Bornstein L, Bornstein MH (2007) Parenting styles and child social development. Encyclopedia on Early Childhood Development, RE Tremblay, M Boivin, RDV Peters (Ed), Montreal, Canada, Centre of Excellence for Early Childhood Development and Strategic Knowledge Cluster on Early Child Development, s.1-4.

Bourvis N, Aouidad A, Cabelguen C et al. (2017) How do stress exposure and stress regulation relate to borderline personality disorder?. Frontiers in psychology 8:2054.

Boyce WT, Ellis BJ (2005) Biological sensitivity to context: I. An evolutionary–developmental theory of the origins and functions of stress reactivity. Development and Psychopathology 17:27-301.

Bozzatello P, Bellino S, Bosia M et al. (2019) Early detection and outcome in borderline personality disorder. Frontiers in psychiatry 10:710.

Brüne M, Ghiassi V, Ribbert H (2010) Does borderline personality reflect the pathological extreme of an adaptive reproductive strategy? Insights and hypotheses from evolutionary life- history theory. Clinical Neuropsychiatry 7:3-9.

Brüne M (2016) Borderline personality disorder: Why 'fast and furious'? Evol Med Public Health 1:52-66.

Buchheim A, Roth G, Schiepek G et al. (2013) Neurobiology of borderline personality disorder (BPD) and antisocial personality disorder (APD). Swiss Archives of Neurology and Psychiatry 164:115-22.

Burt SA, McGue M, Krueger RF et al. (2005) How are parent–child conflict and childhood externalizing symptoms related over time? Results from a genetically informative cross-lagged study. Dev Psychopathol 17:145–65.

Carlson EA, Egeland B, Sroufe LA (2009) A prospective investigation of the development of borderline personality symptoms. Dev Psychopathol 21:1311-34.

Chanen AM, Jovev M, McCutcheon LK et al. (2008a) Borderline personality disorder in young people and the prospects for prevention and early intervention. Current Psychiatry Reviews 4:48–57.

Chanen AM, Velakoulis D, Carison K et al. (2008b) Orbitofrontal, amygdala and hippocampal volumes in teenagers with first-presentation borderline personality disorder. Psychiatry Res Neuroimaging 163:116-25.

Chanen AM, Kaess M (2012) Developmental pathways to borderline personality disorder. Curr Psychiatry Rep14:45–53.

Chanen AM, McCutcheon L, Kerr IB (2014) HYPE: A cognitive analytic therapy-based prevention and early intervention programme for borderline personality disorder. Handbook of Borderline Personality Disorder in Children and Adolescents, C Sharp, J Tackett (Ed), New York, Springer, s. 361–83.

Chapman AL (2019) Borderline personality disorder and emotion dysregulation. Dev Psychopathol 31:1143-56.

Cicchetti D, Toth SL (2005) Child maltreatment. Annu Rev Clin Psychol 1:409–38.

Cicchetti D, Rogosch FA, Hecht KF et al. (2014) Moderation of maltreatment effects on childhood borderline personality symptoms by gender and oxytocin receptor and FK506 binding protein 5 genes. Dev Psychopathol 26:831-49.

Cloninger CR, Svrakic DM, Przybeck TR (1993) A psychobiological model of temperament and character. Arch Gen Psychiatry 50:975-90.

Conti E, Nacinovich R, Bomba M et al. (2013) Diazepam binding inhibitor and dehydroepiandrosterone sulphate plasma levels in borderline personality disorder adolescents. Neuropsychobiology 69:19–24.

Crawford TN, Cohen PR, Chen H et al. (2009) Early maternal separation and the trajectory of borderline personality disorder symptoms. Dev Psychopathol 21:1013–30.

Crowell SE, Beauchaine TP, Linehan MM (2009) A biosocial developmental model of borderline personality: Elaborating and extending Linehan's theory. Psychol Bull 135:495–510.

Crowell SE, Baucom BR, Yaptangco M et al. (2014) Emotion dysregulation and dyadic conflict in depressed and typical adolescents: Evaluating concordance across psychophysiological and observational measures. Biol Psychol 98:50–8.

Csibra G, Gergely G (2009) Natural pedagogy. Trends in Cognitive Sciences 13:148–53.

D'Andrea W, Ford JD, Stolbach B et al. (2012) Understanding interpersonal trauma in children: Why we need a developmentally appropriate trauma diagnosis. Am J Orthopsychiatry 82:187- 200.

Davidson RJ, Jackson DC, Kalin NH (2000) Emotion, plasticity, context, and regulation: Perspectives from affective neuroscience. Psychol Bull 126:890–909.

De Genna NM, Feske U, Larkby C et al. (2012) Pregnancies, abortions, and births among women with and without borderline personality disorder. Womens Health Issues 22:e371-e7.

Del Giudice M (2014) An evolutionary life history framework for psychopathology. Psychological Inquiry 25:261-300.

Del Giudice M (2009) Sex, attachment, and the development of reproductive strategies. Behav Brain Sci 32:1-67.

Egan V, Figueredo AJ, Wolf P et al. (2005) Sensational interests, mating effort, and personality: evidence for cross-cultural validity. Journal of Individual Differences 26:11-9.

Eisenberg N, Spinrad T, Eggum N (2010) Emotion-related self-regulation and its relation to children's maladjustment. Annu Rev Clin Psychol 6:497–525.

Ellis BJ (2004) Timing of pubertal maturation in girls: An integrated life history approach. Psychol Bull 130:920-58.

El-Sheikh M, Erath SA (2011) Family conflict, autonomic nervous system functioning, and child adaptation: State of the science and future directions. Dev Psychopathol 23:703–21.

Erikson EH (1968) Identity, Youth and Crisis. New York, Norton Company, s.36.

Erikson EH (1986) Childhood and Society. New York, W. W. Norton, s.40.

Fiske ST, Taylor SE (1991) Social Cognition (2nd ed). New York, NY, McGraw-Hill, s.44.

Fonagy P, Target M, Gergely G (2000) Attachment and borderline personality disorder. A theory and some evidence. Psychiatr Clin North Am 23:103-22.

Fonagy P, Luyten P (2009) A developmental, mentalization-based approach to the understanding and treatment of borderline personality disorder. Dev Psychopathol 21:1355-81.

Fonagy P, Luyten P, Strathearn L (2011) Borderline personality disorder, mentalization, and the neurobiology of attachment. Infant Ment Health J 32:47-69.

Fonagy P, Luyten P, Allison E et al. (2017) What we have changed our minds about: Part 2. Borderline personality disorder, epistemic trust and the developmental significance of social communication. Borderline Personal Disord Emot Dysregul 4:9.

Ford JD (2009) Neurobiological and developmental research: Clinical implications. Treating Complex Traumatic Stress Disorders: An Evidence-Based Guide, CA Courtois, JD Ford (Ed), New York, Guilford Press s. 31-58.

Ford JD, Courtois CA (2014) Complex PTSD, affect dysregulation, and borderline personality disorder. Borderline Personal Disord Emot Dysregul 9:9.

Fossati A (2012) Adult attachment in the clinical management of borderline personality disorder. J Psychiatr Pract 18:159–71.

Fowles DC (1980) The three arousal model: Implications of Gray's two- factor learning theory for heart rate, electrodermal activity, and psychopathy. Psychophysiology 17:87–104.

Frith CD (2007) The social brain? Philos Trans R Soc Lond B Biol Sci 362:671-8.

Gee DG, Casey BJ (2015) The impact of developmental timing for stress and recovery. Neurobiol Stress 1:184-94.

Goodman M, Hazlett EA, Avedon JB et al. (2011) Anterior cingulate volume reduction in adolescents with borderline personality disorder and co-morbid major depression. J Psychiatr Res 45:803-7.

Gratz KL, Dixon-Gordon KL, Tull MT (2014) Self-injurious behaviors in adolescents with borderline personality disorder. Handbook of Borderline Personality Disorder in Children and Adolescents, C Sharp, JL Thackett (Ed), New York, Springer, s. 195-210.

Griffiths M (2011) Validity, utility and acceptability of borderline personality disorder diagnosis in childhood and adolescence: Survey of psychiatrists. The Psychiatrist 35:19-22.

Grilo CM, Becker DF, Fehon DC et al. (1996) Gender differences in personality disorders in psychiatrically hospitalized adolescents. Am J Psychiatry 153:1089-91.

Griskevicius V, Tybur JM, Delton AW et al. (2011) The influence of mortality and socioeconomic status on risk and delayed rewards: A life history theory approach. J Pers Soc Psychol 100:1015-22.

Gunderson, JG (2007) Disturbed relationships as a phenotype for borderline personality disorder (commentary). Am J Psychiatry 164:1637-40.

Gunderson JG, Herpertz SC, Skodol AE et al. (2018) Borderline personality disorder. Nat Rev Dis Primers 4:18029.

Guyer AE, Silk JS, Nelson EE (2016) The neurobiology of the emotional adolescent: from the inside out. Neurosci Biobehav Rev 70:74-85.

Han S, Northoff G (2009) Understanding the self: A cultural neuroscience approach. Prog Brain Res 178:203-12.

Hankin BL, Barrocas AL, Jenness J et al. (2011) Association between 5-HTTLPR and borderline personality disorder traits among youth. Frontier in Psychiatry 2:1-7.

Hecht KF, Cicchetti D, Rogosch FA et al. (2014) Borderline personality features in childhood: The role of subtype, developmental timing, and chronicity of child maltreatment. Dev Psychopathol 26:805–15.

Herman J (1992) Trauma and Recovery. New York, NY, Basic Books, s.9.

Herpertz SC, Bertsch K (2015) A new perspective on the pathophysiology of borderline personality disorder: A model of the role of oxytocin. Am J Psychiatry 172:840-51.

Herpertz SC, Nagy K, Ueltzhöffer K et al. (2017) Brain mechanisms underlying reactive aggression in borderline personality disorder-sex matters. Biol Psychiatry 82:257–66.

Hipwell AE, Goossens FA, Melhuish EC et al. (2000) Severe maternal psychopathology and infant-mother attachment. Dev Psychopathol 12:157-75.

Homan KJ, Sim LA, Fargo JD et al. (2017) Five-year prospective investigation of self-harm/suicide-related behaviors in the development of borderline personality disorder. Personal Disord 8:183-8.

Horesh N, Ratner S, Laor N et al.(2008) A comparison of life events in adolescents with major depression, borderline personality disorder and matched controls: A pilot study. Psychopathology 41:300–6.

Horowitz LM, Wilson KR, Turan B et al. (2006) How interpersonal motives clarify the meaning of interpersonal behavior: A revised circumplex model. Pers Soc Psychol Rev 10:67–86.

Jovev M, Garner B, Phillips L et al. (2008) An MRI study of pituitary volume and parasuicidal behavior in teenagers with first-presentation borderline personality disorder. Psychiat Res Neuroim 162:273-7.

Jovev M, McKenzie T, Whittle S et al. (2013) Temperament and maltreatment in the emergence of borderline and antisocial personality pathology during early adolescence. J Can Acad Child Adolesc Psychiatry 22:220–9.

Kaess M, Resch F, Parzer P et al. (2013) Temperamental patterns in female adolescents with borderline personality disorder. J Nerv Ment Dis 201:109–15.

Kaess M, Brunner R, Chanen A (2014) Borderline personality disorder in adolescence. Pediatrics 134:782–93.

Kagan J (2013) Behavioral inhibition as a temperamental vulnerability to psychopathology. Child and Adolescent Psychopathology, 2.Baskı, TP Beauchaine, SP Hinshaw (Ed), Hoboken, NJ, Wiley, s. 197–226.

Kalpakci A, Vanwoerden S, Elhai JD et al. (2016) The independent contributions of emotion dysregulation and hypermentalization to the 'double dissociation' of affective and cognitive empahty in female adolescent inpatients with BPD J Pers Disord 30:242–60.

Kaplan HS, Gangestad SW (2005) Life history theory and evolutionary psychology. The Handbook of Evolutionary Psychology, D. M. Buss (Ed), Hoboken, NJ, Wiley, s. 68–95.

Kendler KS, Baker JH (2007) Genetic influences on measures of the environment: a systematic review. Psychol Med 37:615-26.

Kernberg O (1976) Technical considerations in the treatment of borderline personality organization. J Am Psychoanal Assoc 24:795–829.

Kernberg O (1983) Object relations theory and character analysis. J Am Psychoanal Assoc 31:247-72.

Kernberg OF (1984) The couch at sea: psychoanalytic studies of group and organizational leadership. Int J Group Psychother 34:5-23.

Kim J, Cicchetti D (2004) A longitudinal study of child maltreatment, mother-child relationship quality and maladjustment: the role of self-esteem and social competence. J Abnorm Child Psychol 324:341–54.

Koenig J, Rinnewitz L, Parzer P et al. (2017) Resting cardiac function in adolescent non-suicidal self-injury: The impact of borderline personality disorder symptoms and psychosocial functioning. Psychiatry Res 248:117-20.

Kohut H (1977) The Restoration of the Self. Madison, WI, International Universities Press, Inc, s.7.

Krauch M, Ueltzhöffer K, Brunner R et al. (2018) Heightened Salience of Anger and Aggression in Female Adolescents With Borderline Personality Disorder-A Script-Based fMRI Study. Front Behav Neurosci 12:57.

Laakso A, Wallius E, Kajander J et al. (2003) Personality traits and striatal dopamine synthesis capacity in healthy subjects. Am J Psychiatry 160:904–10.

Laurenssen EMP, Hutsebaut J, Feenstra, DJ ve ark (2013) Diagnosis of personality disorders in adolescents: a study among psychologists. Child Adolesc Psychiatry Ment Health 7:3.

Leichsenring F, Leibing E, Kruse J et al. (2011) Borderline personality disorder. Lancet 377:74-84.

Levy KN (2005) The implications of attachment theory and research for understanding borderline personality disorder. Dev Psychopathol 17:959- 86.

Lieb K, Zanarini MC, Schmahl C et al. (2004) Borderline personality disorder. Lancet 364:453-61.

Lind M, Vanwoerden S, Penner F et al. (2019) Inpatient adolescents with borderline personality disorder features: Identity diffusion and narrative incoherence. Personal Disord 4:389-93.

Linehan M (1993a) Cognitive-Behavioral Treatment of Borderline Personality Disorder. New York, Guilford Press s.35.

Linehan M (1993b) Skills Training Manual for Treating Borderline Personality Disorder. New York, Guilford Press s.30.

Ludäscher P, von Kalckreuth C, Parze P et al. (2015) Pain perception in female adolescents with borderline personality disorder. Eur Child Adolesc Psychiatry 24:351–7.

Lyons-Ruth K, Jacobvitz D (2008) Attachment disorganization: Genetic factors, parenting contexts, and developmental transforma- tion from infancy to adulthood. Handbook of Attachment: Theory, Research, and Clinical Applications, 2. Baskı, J Cassidy, PR Shaver (Ed.), New York, NY, Guilford Press s. 666–97.

Lyons-Ruth K (2008) Contributions of the mother-infant relationship to dissociative, borderline, and conduct symptoms in young adulthood. Infant Ment Health J 29:203-18.

Lyons-Ruth K, Bureau JF, Holmes B et al. (2013) Borderline symptoms and suicidality/self-injury in late adolescence: prospectively observed relationship correlates in infancy and childhood. Psychiatry Res 206:273-81.

Martel M (2013) Sexual selection and sex differences in the prevalence of childhood externalizing and adolescent internalizing disorder. Psychol Bull 139:1221–59.

Malenka RC, Siegelbaum SA (2001) Synaptic plasticity: Diverse targets and mechanisms for regulating synaptic efficacy. Synapses, W M Cowan, TC Sudhof, CF Stevens (Ed.), Baltimore, MD, Johns Hopkins University Press s. 393-454.

Massó Rodriguez A, Hogg B, Gardoki-Souto I et al. (2021) Clinical features, neuropsychology and neuroimaging in bipolar and borderline personality disorder: A systematic review of cross-diagnostic studies. Front Psychiatry 12:681876.

McAdams DP, Olson BD (2010) Personality development: continuity and change over the life course. Annu Rev Psychol 61:517- 42.

McAdams DP, McLean KC (2013) Narrative identity. Current Directions in Psychol Sci 22:233- 8.

McCloskey MS, New AS, Siever LJ et al. (2009) Evaluation of behavioral impulsivity and aggression tasks as endophenotypes for borderline personality disorder. J Psychiatr Res 43:1036-48.

McNaughton N, Corr, PJ (2004) A two-dimensional neuropsychology of defense: Fear/anxiety and defensive distance. Neurosci Biobehav Rev 28:285-305.

Mikulincer M, Shaver PR (2007) Attachment in Adulthood: Structure, Dynamics and Change. New York, NY, Guilford Press.

Miller GE, Chen E, Zhou ES (2007) If it goes up, must it come down? Chronic stress and the hypothalamic pituitary adrenocortical axis in humans. Psychol Bull 133:25-45.

Morgan TA, Chelminski I, Young D et al. (2013) Differences between older and younger adults with borderline personality disorder on clinical presentation and impairment. J Psychiatr Res 47:1507-13.

Mühlberger A, Wieser MJ, Gerdes AB et al. (2010) Stop looking angry and smile, please: start and stop of the very same facial expression differentially activate threat-and reward-related brain networks. Soc Cogn Affect Neurosci 6:321-9.

Neuhaus E, Beauchaine TP (2013) Impulsivity and vulnerability to psychopathology. Child and Adolescent Psychopathology, 2. Baskı, TP Beauchaine, SP Hinshaw (Ed.), Hoboken, NJ, Wiley, s. 197–226.

New AS, Carpenter DM, Perez-Rodriguez MM et al. (2013) Developmental differences in diffusion tensor imaging parameters in borderline personality disorder. J Psychiatr Res 47:1101-9.

Newton-Howes G, Clark LA, Chanen A (2015) Personality disorder across the life course. Lancet 385:727–34.

Niedtfeld I, Kirsch P, Schulze L et al. (2012) Functional connectivity of pain-mediated affect regulation in Borderline Personality Disorder. PLOS One 7:e33293.

Northoff G, Heinzel A, de Greck M et al. (2006) Self-referential processing in our brain—a meta-analysis of imaging studies on the self. Neuroimage 31:440-57.

Ogata SN, Silk KR, Goodrich S et al. (1990) Childhood sexual and physical abuse in adult patients with borderline personality disorder. Am J Psychiatry 147:1008- 13.

Paris J (2013) Personality disorders begin in adolescence. J Can Acad Child Adolesc Psychiatry 22:195–6.

Perez-Rodriguez MM, Bulbena-Cabré A, Bassir Nia A et al. (2018) The neurobiology of borderline personality disorder. Psychiatr Clin North Am 41:633-50.

Plomin R (2013) Commentary: Missing heritability, polygenic scores, and gene–environment correlation. J Child Psychol Psychiatry 54:1147-9.

Polderman TJC, Benyamin B, de Leeuw C et al. (2015) Meta-analysis of the heritability of human traits based on fifty years of twin studies. Nature Genetics 47:702-9.

Porges SW (2009) The polyvagal theory: new insights into adaptive reactions of the autonomic nervous system. Cleve Clin J Med 76: 86-90.

Promislow DEL, Harvey PH (1990) Living fast and dying young: A comparative analysis of life-history variation among mammals. Journal of Zoology 220:417-37.

Reinelt E, Stopsack M, Aldinger M et al. (2014) Longitudinal transmission pathways of borderline personality disorder symptoms: from mother to child? Psychopathology 47:10-6.

Reitz S, Kluetsch R, Niedtfeld I et al. (2015) Incision and stress regulation in borderline personality disorder: neurobiological mechanisms of self-injurious behaviour. Br J Psychiatry 207:165-72.

Rosenstein DS, Horowitz HA (1996) Adolescent attachment and psychopathology. J Consult Clin Psychol 64:244-53.

Rubia K (2013) Functional brain imaging across development. Eur Child Adolesc Psychiatry 22:719–31.

Sagvolden T, Aase H, Johansen EB et al. (2005) A dynamic developmental theory of attention-deficit/hyperactivity disorder (ADHD) predominantly hyperactive/impulsive and combined subtypes. Behav Brain Sci 28: 397–468.

Salzman JP, Salzman C, Wolfson AN et al.(1993) Association between borderline personality structure and history of child- hood abuse in adult volunteers. Compr Psychiatry 34:254–7.

Sansone RA, Barnes J, Muennich E et al. (2008) Borderline personality symptomatology and sexual impulsivity. Int J Psychiatry Med 38:53–60.

Sansone RA, Chu JW, Wiederman MW (2011) Sexual behaviour and borderline personality disorder among female psychiatric inpatients. Int J Psychiatry Clin Pract 15:69–73.

Satpute AB, Lieberman MD (2006) Integrating automatic and controlled processes into neurocognitive models of social cognition. Brain Res 1079:86–97.

Schmahl C, Vermetten E, Elzinga BM et al. (2003) Magnetic resonance imaging of hippocampal and amygdala volume in women with childhood abuse and borderline personality disorder. Psychiatry Res 122:193–8.

Schmahl CG, Elzinga BM, Ebner UW et al. (2004) Psychophysiological reactivity to traumatic and abandonment scripts in borderline personality and posttraumatic stress disorders: a preliminary report. Psychiatry Res 126:33–42.

Schmid M, Schmeck K, Petermann F (2008) Persönlichkeitsstörungen im Kindes- und Jugendalter? Kindheit und Entwicklung 17:190-202.

Schulze L, Schmahl C, Niedtfeld I (2016) Neural correlates of disturbed emotion pro- cessing in borderline personality disorder: A multimodal meta-analysis. Biol Psychiatry 79:97-106.

Sela Y, Barbaro N (2018) Evolutionary perspectives on personality and individual differences. The SAGE Handbook of Personality and Individual Differences: The Science of Personality and Individual Differences, V Zeigler-Hill, TK Shackelford (Ed.), NewYork, Sage Publications s.203-28).

Sharp C, Pane H, Ha C et al. (2011) Theory of mind and emotion regulation difficulties in adolescents with borderline traits. J Am Acad Child Adolesc Psychiatry 50:563-73

Sharp C (2014) The social-cognitive basis of borderline personality disorder: A theory of hypermentalizing. The Handbook of Borderline Personality Disorder in Children and Adolescents, C, Sharp, J Tackett (Ed.), New York, Springer. s.211-25

Sharp C, Fonagy P (2015) Practitioner review: Borderline personality disorder in adolescence – Recent conceptualization, intervention, and implications for clinical practice. J Child Psychol Psychiatry 56:1266–88.

Sharp C, Kalpakci A, Mellick W (2015) First evidence of a prospective relation between avoidance of internal states and borderline personality disorder features in adolescents. Eur Child Adolesc Psychiatry 24:283–90.

Sharp C, Venta A, Vanwoerden S et al. (2016) First empirical evaluation of the link between attachment, social cognition and borderline features in adolescents. Compr Psychiatry 64:4-11.

Sharp C, Vanwoerden S, Wall K (2018) Adolescence as a Sensitive Period for the Development of Personality Disorder. Psychiatr Clin North Am 41:669–83.

Silbersweig D, Clarkin JF, Goldstein M et al. (2007) Failure of frontolimbic inhibitory function in the context of negative emotion in borderline personality disorder. Am J Psychiatry 164:1832–41.

Silvers JA, Buhle JT, Ochsner KN (2014) The neuroscience of emotion regulation: Basic Mechanisms and Their Role in Development, Aging and Psychopathology, cilt 2, KN Ochsner, SM Kosslyn (Ed.), The Oxford University Press s.1-48

Simpson JA, Griskevicius V, I-Chun Kou S et al. (2012) Evolution, stress, and sensitive periods: The influence of unpredictability in early versus late childhood on sex and risky behavior. Dev Psychol 48:674-86.

Slavin-Stewart C, Boylan K, Burke JD (2018) Subgroups of Adolescent Girls With Borderline Personality Disorder Symptoms. J Pers Disord 32:636-53.

Stanley B, Siever LJ (2010) The interpersonal dimension of borderline personality disorder: Toward a neuropeptide model. Am J Psychiatry 167:24–39.

Stanley B, Sher L, Wilson S et al. (2010) Non-suicidal self-injurious behavior, endogenous opioids and monoamine neurotransmitters. J Affect Disord 124:134-40.

Stanley B, Perez-Rodriguez MM, Labouliere C et al. (2018) A Neuroscience-Oriented Research Approach to Borderline Personality Disorder. J Pers Disord 1-39.

Stepp SD, Keenan K, Hipwell AE et al. (2014a) The impact of childhood temperament on the development of borderline personality disorder symptoms over the course of adolescence. Borderline Personal Disord Emot Dysregul 1:18

Stepp SD, Whalen DJ, Scott LN et al. (2014b) Reciprocal effects of parenting and borderline personality disorder symptoms in adolescent girls. Dev Psychopathol 26:361-78.

Stepp SD, Lazarus SA, Byrd AL (2016) A systematic re- view of risk factors prospectively, associated with borderline personality disorder: Taking stock and moving forward. Personal Disord 7:316–23.

Tabibnia G, Monterosso JR, Baicy K et al. (2011) Different forms of self-control share a neurocognitive substrate. The Journal of Neuroscience: J Neurosci 31:4805-10.

Takahashi T, Chanen AM, Wood SJ et al. (2009) Insular cortex volume and impulsivity in teenag- ers with first-presentation borderline personality disorder. Prog Neuropsychopharmacol Biol Psychiatry 33:1395-400.

Terr LC (1991) Childhood traumas: An outline and over- view. Am J Psychiatry 148:10-20.

Trippany RL, Helm HM, Simpson L (2006) Trauma reenactment: Rethinking borderline personality disorder when diagnosing sexual abuse survivors. J Ment Health Couns 28:95-110.

Trull T, Widiger T, Lynam D et al. (2003) Borderline personality disorder from the perspective of general personality functioning. J Abnorm Psychol 112:193–202.

Van Ijzendoorn MH, Schuengel C, Bakermans-Kranenburg MJ (1999) Disorganized attachment in early childhood: Meta-analysis of precursors, concomitants, and sequelae. Dev Psychopathol 11:225-49.

Van der Kolk BA, Pelcovitz D, Roth S et al. (1996) Dissociation, somatization, and affect dysregulation: The complexity of adaption to trauma. Am J Psychiatry 153:83-93.

Van der Kolk BA, Pynoos RS, Cicchetti D et al. (2009) Proposal to include a developmental trauma disorder diagnosis for children and adolescents in DSM-V. Unpublished manuscript, the National Child Traumatic Stress Network Developmental Trauma Disorder Taskforce, University of California Los Angeles, Los Angeles, CA.

Weinfield NS, Sroufe LA, Egeland B (2000) Continuity, discontinuity, and their correlates. Child Dev 71:695–702.

Weiss A, Egan V, Figueredo AJ (2004) Sensational interests as a form of intrasexual competition. Personality and Individual Differences, 36:563-73.

Westen D, Ludolph P, Misle B et al. (1990) Physical and sexual abuse in adolescent girls with borderline personality disorder. Am J Orthopsychiatry 60:55-66.

Westen D ve Cohen RP (1993) The self in borderline personality disorder: A psychodynamic perspective. The Self in Emotional Distress: Cognitive and Psychodynamic Perspectives, ZV Segal, SJ Blatt (Ed.), New York, Guilford Press, s. 334-68

Whittle S, Chanen AM, Fornito A et al. (2009) Anterior cingulate volume in adolescents with first-presentation borderline personality disorder. Psychiatric Research Neuroimaging 172:155–60.

Whittle S, Vijayakumar N, Dennison M et al. (2016) Observed measures of negative parenting predict brain development during adolescence. PLoS One 11:e0147774.

Wilkinson R, Pickett K (2009) The Spirit Level: Why Equality is Better for Everyone. London, UK: Penguin Books.

Willis F, Kuniss S, Kleindienst N et al. (2017) The role of nociceptive input and tissue injury on stress regulation in borderline personality disorder. Pain 158:479-87.

Winsper C, Zanarini M, Wolke D (2012) Prospective study of family adversity and maladaptive parenting in childhood and borderline personality disorder symptoms in a non-clinical population at 11 years. Psychol Med 42:2405–20.

Winsper C, Wolke D, Lereya T (2015a) Prospective associations between prenatal adversities and borderline personality disorder at 11–12 years. Psychol Med 45:1025-37.

Winsper C, Marwaha S, Lereya ST (2015b) Clinical and psychosocial outcomes of borderline personality disorder in childhood and adolescence: a systematic review. Psychol Med 45:2237–51.

Winsper C, Lereya ST, Marwaha S (2016) The aetiological and psychopathological validity of borderline personality disorder in youth: A systematic review and meta-analysis. Clin Psychol Rev 44:13-24.

Winsper C, Hall J, Strauss VY et al. (2017) Aetiological pathways to Borderline Personality Disorder symptoms in early adolescence: childhood dysregulated behaviour, maladaptive parenting and bully victimisation. Borderline Personal Disord Emot Dysregul 4:10.

Winsper C, Bilgin A, Thompson A et al. (2020) The prevalence of personality disorders in the community: a global systematic review and meta-analysis. Br J Psychiatry 216:69–78.

Wolke D, Schreier A, Zanarini MC et al. (2012) Bullied by peers in childhood and borderline personality symptoms at 11 years of age: a prospective study. J Child Psychol Psychiatry 53:846–55.

Yildiz D, Ciftci A, Yalcin O (2020a) The association between trauma history, trauma-related psychopathology and treatment completion at an adolescent inpatient substance abuse treatment center in Turkey. Journal of Child and Adolesence Substance Abuse 29:58-67.

Yildiz D, Ciftci A, Yalcin O (2020b) Substance use patterns, psychosocial traumas, psychiatric comorbidities, and gender differences among adolescent inpatients at an addiction treatment center. Dusunen Adam 33:190-202.

Zalewski M, Stepp SD, Scott LN et al. (2014) Maternal borderline personality disorder symptoms and parenting of adolescent daughters. J Pers Disord 28:541-54.

Zanarini MC, Williams AA, Lewis RE et al. (1997) Reported pathological childhood experiences associated with the development of borderline personality disorder. Am J Psychiatry 154:1101-6.

Zanarini MC (2000) Childhood experiences associated with the development of borderline personality disorder. Psychiatr Clin North Am 23:89–101.

Zanarini MC, Temes CM, Frankenburg FR et al. (2018) Description and prediction of time-to-attainment of excellent recovery for borderline patients followed prospectively for 20 years. Psychiatric Res 262:40-5.

Zanarini MC, Temes CM, Magni LR et al. (2020) Risk Factors for Borderline Personality Disorder in Adolescents. Journal of Personality Disorders 34:17-24.