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REVIEW

Understanding the Relationship Between Sleep Problems in Early Childhood and Borderline Personality Disorder: A Narrative Review

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Objective: Recent research indicates that sleep problems in childhood precede the development of borderline personality disorder (BPD) symptoms, but the mechanisms by which sleep problems associate with BPD are still unknown. This narrative review aims to provide some potential explanations for how early sleep problems might associate with BPD.

Methods: We used the biosocial developmental model of BPD as a framework to discuss how sleep problems may associate with BPD. Articles were identified via PubMed and Embase, and papers published between January 1991 and April 2021 were extracted. Authors made a series of literature searches using the following keywords: Sleep problems, Insomnia, Nightmares, Hypothalamic–Pituitary–Adrenal Axis (HPA), Prefrontal Cortex, Family Psychopathology, Disrupted Attachment, Child Maltreatment, Impulsivity, Emotion Regulation, Internalizing, Externalizing, Rumination, Childhood, Adolescence, Young people. The inclusion criteria were published in peer-reviewed journals; human studies or reviews; published in English. The exclusion criteria were commentaries; abstracts from conferences; studies with animal samples. A total of 96 articles were included for the purpose of this review.

Results: The evidence from this review suggests that some biological factors and core features of BPD act as potential mechanisms mediating the associations between early sleep and subsequent BPD, while some family-related factors might constitute common risk factors for sleep problems and BPD.

Conclusion: The biosocial developmental model of BPD provides a plausible characterization of how sleep disruption might lead to subsequent BPD. Further research on new developmental and early intervention approaches to understand how sleep in early stages associates with BPD could have significant clinical impact on these patients and could inform targeted therapeutic interventions.

Keywords: sleep, borderline personality disorder, biosocial developmental model, childhood, adolescence, mechanisms

Introduction

Borderline Personality Disorder (BPD) is characterized by emotional instability, impulsivity, disturbed cognition, sleeplessness and states of high inner tension. Sleep problems are common in BPD and exacerbate some core symptoms, such as emotional dysregulation (ie, the inability to flexibly respond to and manage emotions).[1] However, the nature of the associations between sleep problems and BPD needs exploration and is also characterized by lack of specificity (eg, sleep also associates with other mental disorders, such as depression).[2] Therefore, further

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research is required to understand the specific role of sleep in the development and exacerbation of BPD.

It is possible that sleep problems may precede the development of BPD. Findings from two recent longitudinal studies have provided support for the existence of prospective associations between sleep problems in childhood and subsequent BPD symptoms in adolescence. First, it was shown that persistent nightmares in childhood (ie, at 2.5, 3.5, 4.8 and 6.8 years of age) were associated with more BPD symptoms in adolescence.[3] Second, shorter sleep duration at 3.5 years was prospectively associated with the development of BPD symptoms in adolescence.[4] Therefore, emerging evidence suggests that nightmares and specific sleep problems occurring in early childhood are prospectively associated with the development of BPD symptoms in adolescence, at least in the general population. However, the potential mechanisms/factors underlying the associations between early sleep problems and BPD are still unknown.

The biosocial developmental model of borderline personality[5] provides a framework, which helps to understand the development of BPD. In this model, the authors suggest one probable pathway that leads to BPD, which begins with early vulnerability, expressed initially as impulsivity and followed by heightened emotional sensitivity. These vulnerabilities are then potentiated across development by environmental risk factors that give rise to more significant and functionally damaging emotional, behavioral, and cognitive dysregulation. In this model, several biological correlates, psychosocial risk factors and etiological hypotheses are presented. Therefore, we will use this model as a framework to discuss how sleep problems might associate with the development of BPD. More specifically, we will focus on the factors (ie, biological, psychosocial and core features) from the model with the strongest evidence for the development of BPD and that also have links with early sleep problems. Therefore, among the biological correlates, we will focus on the hypothalamic–pituitary–adrenal axis (HPA) and prefrontal cortex. Among the psychosocial risk factors, we will focus on family psychopathology, disrupted attachment and childhood maltreatment. Finally, among the core features of BPD, we will focus on impulsivity, emotion regulation, and internalizing (eg, sadness, anxiety or loneliness) and externalizing (eg, aggression or hyperactivity) symptoms. Thus, this article reviews the links between early sleep and each of these factors, as potential mechanisms and/or contributing factors explaining the association between sleep problems and subsequent BPD, and aims to answer the following research questions:

(1) What is the association between sleep and HPA in adolescence and childhood?

(2) What is the impact of sleep on the prefrontal cortex in young adulthood, adolescence and childhood?

(3) What is the association between sleep and family psychopathology in early childhood?

(4) How do sleep and disrupted attached interact in early childhood?

(5) What is the impact of childhood maltreatment on sleep in childhood, adolescence and young adulthood?

(6) What is the link between sleep and impulsivity in young adulthood, adolescence and childhood?

(7) What is the association between sleep and emotional regulation in young adulthood, adolescence and childhood?

(8) How do sleep and internalizing/externalizing symptoms associate in adolescence and childhood?

Method

A literature search was conducted via PubMed and Embase for studies published between January 1991 and April 2021. IMM and BD conducted a series of literature searches, using the following keywords in indexed fields: 'Sleep problems', "Insomnia", "Nightmares", "HPA", "prefrontal cortex", "family psychopathology", "disrupted attachment", "child maltreatment", "impulsivity", "emotion regulation", "internalizing", "externalizing" and "rumination", together with "childhood", "adolescence", "young people". The keywords of sleep problems OR insomnia OR nightmares AND childhood OR adolescence OR young people, together with one of the correlates, separately, were used in all possible permutations, and abstracts from the results of searches were assessed. Selected papers had to meet the following criteria: (1) published in peer-reviewed journals; (2) human studies or reviews on the related topic; (3) published in English. The exclusion criteria were (1) commentaries; (2) abstracts from conferences; (3) studies with animal samples. In the next step, the full text of eligible articles was obtained and assessed. After a full-text assessment of all articles by the research team, their inclusion was subjected to open discussion by the research team. Finally, a total of 96 articles were included for the purpose of this review. See Figure 1 for details of the search and selection strategy.

Overlapping Biological Correlates of BPD and Sleep

Sleep and Hypothalamic–Pituitary–Adrenal Axis

The HPA axis represents a key stress response system, and growing evidence suggests that it is dysfunctional in the BPD population.[6] Further, sleep, and in particular deep sleep, has an inhibitory influence on the HPA axis, whereas activation of the HPA can lead to sleeplessness.[7]

What is the Association Between Sleep and HPA in Adolescence and Childhood?

In adolescents, the existing studies on the topic have focused on actigraphy-based measures. For instance, in a cross-sectional study with 19-year-old healthy adolescents (N = 119), longer sleep duration was related to steeper decline in cortisol.[8] Another study conducted in 265 participants with a mean age of 12.3 years old reported that actigraph-based sleep duration and sleep quality were both related to diurnal cortisol levels in boys, but not in girls.[9] Further, a recent actigraphy-based cross-sectional study conducted with 55 healthy individuals aged 8–16 years found that increased HPA activity was associated with longer duration of staying awake as well as poorer sleep efficiency and greater number of awakenings.[10]

Further cross-sectional research has been conducted in younger ages, using both objective and subjective sleep measures. In an actigraphy-based study in eight-year-old children (N = 282), shorter average sleep duration and lower sleep efficiency were both associated with higher levels of salivary cortisol.[11] Another study using sleep-electroencephalography in 113 children aged 6–10 years found that morning cortisol secretion was negatively

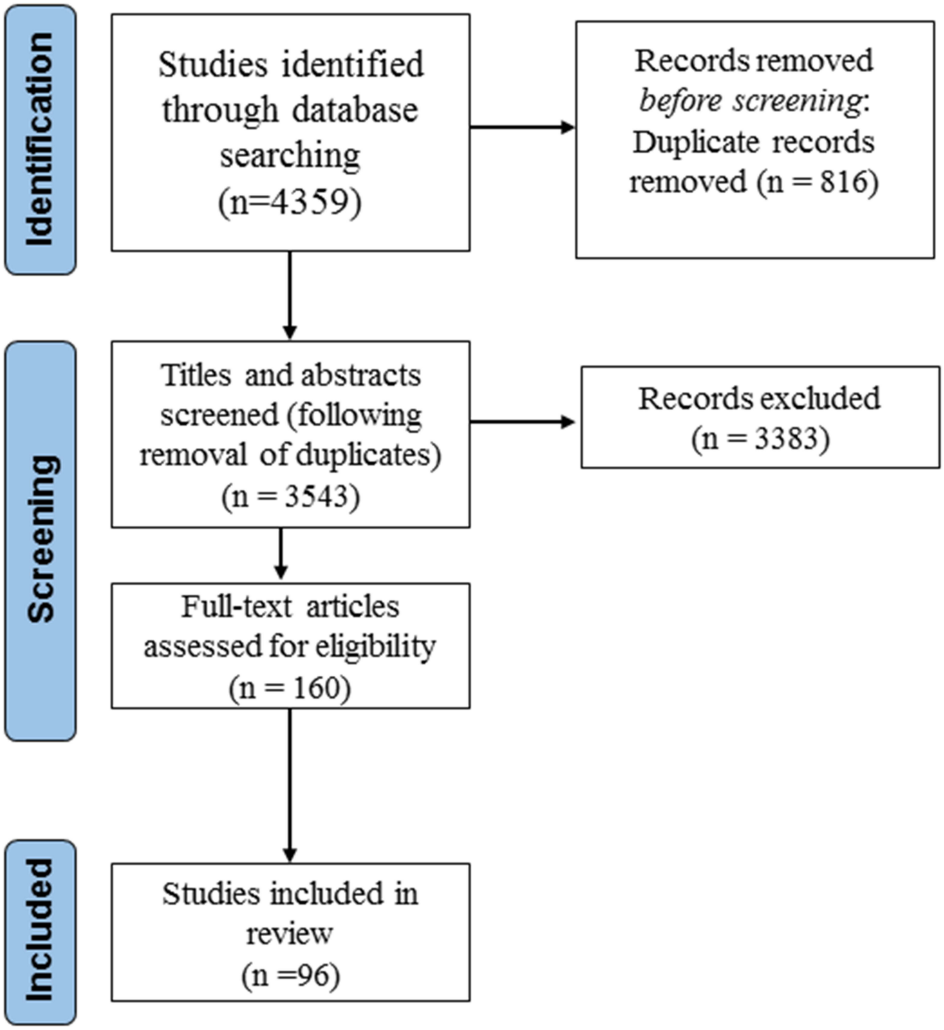

Figure 1 Flowchart outlining the search and selection strategy. This figure describes the search and selection for strategy for the studies included in this narrative review. Initially, 4359 studies were identified through database searching; then, 3543 studies out of them were screened for titles and abstracts; and out of these 3543 studies, 160 full-text articles were assessed for eligibility. Finally, 96 studies were included in this narrative review.

associated with sleep duration and slow wave sleep and positively associated with the relative amount of Stage 2 sleep during the preceding night.[12] Further, El-Sheikh et al reported in a study where they combined actigraphy and self-reported sleep measures in a group of nine-year-old children (N = 64) that higher levels of cortisol were related to increased subjective sleep problems and objective shorter sleep duration and poorer sleep quality.[13] Pesonen et al[14] examined 284 eight-year-old children and found that boys with parent-reported sleep problems (eg, difficulties in initiating and maintaining sleep, excessive somnolence, or nightmares) had lower levels of diurnal salivary cortisol. But these findings were unrelated to actual sleep duration measured by actigraphy. Finally, another study combining parent-reported insomnia symptoms with polysomnography-based sleep duration in 327 children aged 5–12 years old showed that parent-reported insomnia with objective short sleep duration was associated with higher cortisol levels.[15]

The existing studies in preschoolers have mainly used objective sleep measures. The first of these studies was done using polysomnography-based sleep recordings in 67 preschool-children aged 5.34 years and showed that the level of cortisol secretion was significantly lower in good sleepers,[16] while poor sleepers[16] displayed a significantly increased cortisol secretion.[17] Another cross-sectional study conducted with 82 preschoolers (mean age = 4.91 years) using actigraphy showed that children with high cortisol secretion had extended sleep-onset latency and decreased sleep efficiency.[18]

Finally, few longitudinal studies have been conducted in infants and toddlers using parent-reported sleep measures. In a study of 322 infants aged 12–20 months, infants with a flatter diurnal cortisol slope, which is considered maladaptive, and those with a more marked morning cortisol rise had shorter nighttime sleep duration.[19] Further, a longitudinal study in 51 mothers and their 18–20-month-old toddlers showed that blunted cortisol secretion patterns predicted risk for early emerging sleep problems.[20]

Therefore, the existing evidence suggests that associations between sleep problems and HPA axis dysfunction are evident in young ages, from adolescence to infancy. And this evidence has been supported using both subjective and objective sleep measures. More specifically, it seems that sleep difficulties are associated with increased cortisol levels, and consequently with increased HPA axis activity, in early stages of the development. However, further longitudinal studies are still needed and there is still some inconsistency concerning the associations between objective sleep duration and cortisol levels. In addition, little is known yet about the directionality of these associations. Within the context of the associations between early sleep problems and subsequent BPD symptoms, which is the focus of this review, sleep difficulties in early childhood might increase HPA axis activity, which would consequently lead to BPD symptomatology. However, further specific studies on this topic are required to test this pathway.

Sleep and Prefrontal Cortex

A number of studies have described abnormal prefrontal cortex in individuals with BPD.[21] For example, reduced glucose metabolism in the prefrontal cortex has been reported in BPD patients (mean age = 34.2 years old).[22] Further, BPD patients (aged 20–43 years old) demonstrated decreased metabolism in anterior cingulate and prefrontal cortex.[23] In addition, some adolescents with BPD had structural alterations in frontolimbic regions.[24] Concerning the role that sleep problems may have in the prefrontal cortex, there is evidence that the prefrontal cortex is particularly sensitive to sleep deprivation and benefits from good sleep practices.[25] However, most of the existing evidence comes from studies in adults with a limited number of studies focusing on young people.

What is the Impact of Sleep on the Prefrontal Cortex in Young Adulthood, Adolescence and Childhood?

A cross-sectional study of 20 young subjects (age range 17–23) using functional magnetic resonance imaging (fMRI) showed that sleep deprivation associated with deficits in the bilateral inferior frontal gyrus, left subthalamic nucleus and left lingual gyrus.[24] Further, a recent cross-sectional study with 25 healthy young adults and adolescents (age range = 12–22) using actigraphy and resting-state fMRI showed that irregular sleep patterns are associated with increased network connectivity in the default mode network– specifically in the right and left lateral parietal lobule.[26] In adolescents, three studies have examined the associations between sleep and brain structure. In one of these studies using structural MRI, the brain areas in which higher volume was correlated with longer sleep duration included the orbitofrontal cortex, prefrontal and temporal cortex, precuneus, and supramarginal gyrus.[27] Another cross-sectional study conducted in 177 adolescents (mean age 14.4 years), the medial prefrontal cortex correlated both with bedtime and wake-up times.[28] A third

study conducted in forty-six adolescents using a functional MRI scan during which they completed a cognitive control and risk taking task found that adolescents who reported poorer sleep also exhibited less recruitment of the dorsolateral prefrontal cortex during cognitive control.[29]

So far, only two studies have examined the associations between sleep and brain areas in children. In a cross-sectional study in 15 male children (ages 7–11 years), shorter sleep duration contributed to neural alterations of brain regions involved in the regulation of emotion and reward processing (including prefrontal areas).[30] In a longitudinal study from the Generation R cohort, mothers reported sleep disturbances in 720 children at ages 2 months, 1.5, 2, 3, and 6 years and T1-weighted MRI images were used to assess brain structure at 7 years. The authors reported that more adverse developmental course of childhood sleep disturbances was associated with smaller grey matter volumes and thinner dorsolateral prefrontal cortex.[31] Therefore, there is still limited research examining the specific effects of sleep in prefrontal brain areas in young ages. Among the existing evidence, the studies support those findings from adult research and suggest that sleep problems have an impact on the prefrontal cortex. However, further studies in this area are still needed and especially longitudinal studies and manipulation studies in young children and infants. The current evidence, and specially the one that comes from sleep deprivation studies, supports the notion that early sleep problems might have an impact in prefrontal areas, and subsequently, impairments at this cortical level might underlie the development of BPD symptoms.

Overlapping Psychosocial Risk Factors for BPD and Sleep

Sleep and Family Psychopathology

There is convincing evidence that the families of patients with BPD have greater rates of psychopathology (eg, depressive, substance abuse, or antisocial disorders) than the families of healthy controls.[32] This may be due to the familial risk and heritability observed in BPD patients[33] or due to the maladaptive parenting strategies frequently experienced by BPD patients.[34] Research investigating the links between offspring sleep and parental psychopathology has been limited. Most studies in this area are related to maternal depression, and more specifically in the perinatal and postnatal period. Further, all these studies have mainly focused on the effects of parental mental health on their offspring's sleep during childhood or infancy, and thus for this specific section, we will focus here on these stages.

What is the Association Between Sleep and Family Psychopathology in Early Childhood?

Concerning the effect that parental mental health has in sleep development in early childhood, and in relation to the perinatal period, depressed pregnant women seem to have newborns exhibiting higher levels of disrupted sleep.-[35] Similarly, prenatal maternal anxiety and depression predict more sleep problems in toddlers aged 18–30 months,[36] and mothers with prenatal depression report more night awakenings in their one-year-old infants.[37] There is also evidence that both mother's prenatal and early postpartum depression symptoms associate with the emergence of infant's sleep problems.[38] With respect to postpartum depression only, depressed mothers report more infants' sleep difficulties in the first weeks,[39] and in the first year.[40] Further, maternal report of infant sleep problem can be considered a significant predictor of maternal postnatal depression.[41] Beyond the infancy period, in a clinical sample of mothers with mood disorders,[42] sleep problems are more frequent, severe and persistent in infants of depressed mothers. Similarly, maternal symptoms of anxiety and depression during the first year of the child's life are associated with nocturnal awakening at six and 18 months.[43] Finally, infant sleeping problems relate to maternal depressive (and comorbid anxiety) disorders irrespective of maternal parity.[44]

Among the effects that maternal depression has in toddlers' and/or children's sleep functioning, most evidence supports the negative effect of maternal depression. In a large cross-sectional study using a large representative sample of 2–3 years old children, self-reported mother depressive symptomatology contributed to parent-reported sleep problems in toddlers.[45] Similarly, in another cross-sectional study in 6000+ 3-to-4 years old twin pairs, maternal depression was associated with sleep problems.[46] Another cross-sectional study conducted in 4–9 years old children (N=1391) corroborated the associations between maternal depression and children's sleep disturbances also in preschoolers and school-aged children.[47] Only one study, so far, has examined the effects of maternal depression in their offspring sleep using polysomnography in 64 healthy infants and showed that altered sleep structure was observed in children born from depressed mothers.[48]

Few studies have also examined other maternal mental health problems, rather than maternal depression. Previous research suggests that infant sleep problems are associated with poorer health in both parents.[49] In a study conducted by Warren et al,[50] the authors examined four- and 14-month-old infants of mothers with panic disorder (PD) and showed that infants from the four-month cohort with PD mothers had more fragmented sleep at eight months. In another study, in a sample of cocaine-exposed seven-month-old infants,[51] the findings showed that exposed infants who had mothers with higher levels of anxiety showed higher levels of sleep problems. Finally, a recent study reported that several maternal risk factors during pregnancy were related to sleep difficulties in infants.[52] In relation to children, in a study conducted including 182 families, bedtime behavior and daytime sleepiness seemed to be greatly affected in those children of mothers with mental health problems.[53]

To sum up, the existing literature has been mainly focused on maternal depression, especially perinatal and postnatal depression and mostly in children and infants. Also, most of these studies have been conducted using parent-reported sleep measures and scarce research has used objective-based sleep measures. Further, some attempts have been made to investigate the role of other maternal mental disorders on infants' sleep. The existing evidence suggests that mother mental health problems may lead to subsequent sleep problems in their children, although there is also some evidence suggesting the opposite effect (ie, the impact of sleep on parent's psychopathology). In the context of the development of BPD and the role of sleep, this evidence suggests that parental mental health problems could be a common risk factor for sleep problems and BPD symptoms.

Sleep and Disrupted Attachment

Problems in secure attachment formation are associated with both BPD psychopathology[54] and child sleeping problems.[55] Considering that attachment has been mainly studied in infancy and childhood, the studies reported in this section will cover these stages.

How Do Sleep and Disrupted Attached Interact in Early Childhood?

In a meta-analysis study conducted in 2017, the authors reported positive associations between secure infant-mother attachment and sleep efficiency and insecure-resistant attachment and sleep problems.[56] Most of the studies on the topic have been conducted using a longitudinal approach. For instance, in a study using objective measures of sleep and attachment in 62 middle-class families, children with secure attachment subsequently slept more and had higher sleep efficiency than those with insecure attachment.[57] In another longitudinal study using parent-reported sleep measures in 134 mother–child dyads between six and 36 months,[58] it was shown that children with disorganized attachment had prospectively shorter sleep duration, later bedtime, and more awakenings. In another longitudinal study where attachment at 18 months and children's actigraph-based and parent-reported sleep quality at 24 months were examined in 55 families,[59] higher resistance predicted longer wake duration at night.

Longitudinal studies in infants have also reported the association between attachment and sleep.[60] For instance, information on night wakings in 93 infants showed that attachment at 12 months was related to infant night waking patterns in the first six months of life. In another longitudinal study where attachment and sleep were measured from seven to 14 months of child's life, infants with a secure attachment had lower number of night wakings over time.[61] Further, there is also evidence of the impact of sleep difficulties in subsequent disruption of attachment in early childhood. For instance, infants with insecure-resistant attachments at 15 months have greater numbers of night wakings and longer night-waking episodes at six and 15 months.[62] Similarly, sleep problems in infants at three months were associated with disorganized attachment at 18 months.[63]

In whole, the existing evidence suggests that disruptive attachment leads to sleep difficulties in infancy and childhood, but there is also evidence that early sleep problems might impact later attachment styles, suggesting a bidirectional association between sleep and attachment. In relation to the associations between early sleep and BPD, one explanation could be that disrupted attachment may be a common risk factor for both sleep problems and BPD. But also, other potential explanation could be that early sleep problems would lead to disrupted attachment, which consequently leads to the development of BPD symptoms. Further longitudinal research on how disrupted attachment, early sleep difficulties and BPD symptoms interact with each other is still needed.

Sleep and Childhood Maltreatment

BPD associates with history of maltreatment in childhood.[64] Further, childhood abuse could be also a

risk factor for sleep difficulties. Although the effects of childhood maltreatment can also lead to sleep problems in adulthood,[65] for this review, we will only focus on those relevant studies investigating the associations between childhood maltreatment and sleep in childhood, adolescence and young adults.

What is the Impact of Childhood Maltreatment on Sleep in Childhood, Adolescence and Young Adulthood?

In a recent study conducted in 18–22 years old young adults, childhood emotional neglect predicted insomnia symptoms.[66] Further, in a recent study conducted including 181 college students, those who reported more childhood maltreatment also reported significantly lower quality sleep and felt less rested upon awakening.[67] In adolescence, a study conducted in 73 subjects found that more severe childhood maltreatment was related to increased sleep disturbances.[68] In another longitudinal study using 2910 adolescents aged 14–17 years old, sexual abuse, physical abuse, intimate partner violence, emotional maltreatment and physical neglect in childhood were all associated with several sleep problems in adolescence.[69] Another recent study conducted in 153,547 adolescents found that physical abuse, emotional abuse, sexual abuse, physical neglect or emotional neglect associated with increased risk of sleep disturbance.[70] In 1997, a study using 44 pre-pubertal children found that abused children had prolonged actigraphy-based sleep latency and decreased sleep efficiency.[71]

In a large population-based sample of adolescents (N = 9582), youth exposed to childhood adversity were more likely to have insomnia, with a larger risk for those exposed to interpersonal violence during early childhood or adolescence, and a clear dose–response relationship between exposure to multiple types of adversity and risk for insomnia.[72] Another longitudinal study in 147 female adolescents showed that sexually abused participants reported greater rates of sleep disturbances.[73] Finally, in a recent longitudinal cohort study of 2491 children at 5–9 and 10–16 years old, having an accumulation of childhood adversities significantly associated with sleep disturbances in youth 10–16 years, but not in younger children.[74]

Therefore, the existing evidence suggests that childhood maltreatment may lead to sleep difficulties in young adulthood, adolescence and childhood. However, most of this research has been conducted using subjective sleep measures and further research using objective-based sleep measures is needed. Within the context of the associations between early sleep and BPD, childhood maltreatment could be a common risk factor for sleep and BPD symptoms.

Core Features of BPD

Sleep and Impulsivity

Impulsivity in BPD is a central symptom.[75] Further, although inadequate sleep has been linked to poor impulse control in adults, little research has investigated the role of sleep on impulsive behaviors in younger ages.

What is the Link Between Sleep and Impulsivity in Young Adulthood, Adolescence and Childhood?

In relation to the associations in young adults, one study conducted in 373 young adults (18–29 years) found that higher levels of sleepiness associated with several types of impulsivity.[76] Another recent study in 13- to-19-year-olds diagnosed with bipolar disorder (BD; n = 33, 16.2 years), the change in sleep duration between school days and weekends was associated with increased impulsivity.[77] Further, one study conducted in 19 young adults showed that acute sleep restriction caused deregulation of cognitive control that may manifest in increased impulsivity.[78]

In children and adolescents, there is also some research on the associations between sleep and impulsivity. In one study conducted in 87 healthy participants (8.6 to 15.8 years),[79] sleepiness following acute sleep restriction was not sufficient to produce deficits in impulsivity and sustained attention. Further, another study conducted in 1180 children (7–10 years) showed that children with sleep disorders had greater predisposition to impulsivity.[80] A randomized trial using a sample of 34 typically developing children aged 7 to 11 years reported that a cumulative restriction of sleep of 54.04 minutes was associated with detectable deterioration in restless-impulsive behavior scores.[81]

Therefore, the existing scarce evidence from studies in young people and adolescents suggests that sleep might play a causal role in the development of impulsive behaviours, while further research is still needed to confirm whether this is also true in children. However, with the existing evidence we can hypothesize that impulsivity could be considered a mediating factor in the associations between early sleep and BPD symptoms; however, further research is still needed.

Sleep and Emotion Regulation

Emotion dysregulation is often considered a core characteristic of BPD.[82] Further, there is also evidence about the

associations between emotion regulation and sleep, such as the developmental cascade model in which sleep problems have a persistent negative effect on emotional regulation, which in turn contributes to ongoing sleep problems and poorer attentional regulation in children over time.[83] In recent years, there has been also an increasing research interest in understanding the bidirectional relationship between emotion and sleep. The relationship between inadequate sleep and mood has been well established in adults. However, further studies are still needed in younger ages, and especially to investigate the potential role of sleep in emotion regulation.

What is the Association Between Sleep and Emotion Regulation in Young Adulthood, Adolescence and Childhood?

In a recent cross-sectional study conducted in young people (20 years old), distraction and reappraisal (ie, two emotion regulation strategies) were impaired by sleep deprivation.[84] Another sleep deprivation study with 50 healthy adolescents, aged 14–17, confirmed that several nights of sleep restriction adversely affects adolescents' ability to regulate their emotions.[85] In another manipulation study, but this time in 32 children (8–12 years), modest differences in sleep duration, accumulated over few days, affected critical emotional functions in children.-[85] Further, a recent study showed that after two nights of sleep restriction conducted in a sample of 53 children aged 7–11 years old alterations in children's affect, emotional arousal, facial expressions, and emotion regulation appeared.[86] In young children, in a sleep restriction study conducted in 10 healthy toddlers (30–36 months),[87] sleep restriction resulted in an increase in negative emotional responses.

This relationship between sleep and emotion regulation has been also tested in large representative samples of adolescents. For instance, in a nationally representative sample (N = 10,148; 13–18 years), differential relations between sleep problems and certain regulation strategies were found.[88] Finally, some attempts have been conducted to specifically investigate the role of sleep problems and emotion regulation in BPD. In a study conducted in 450 undergraduates (23 years old), BPD characteristics capturing emotional dysregulation showed the strongest correlations with poor sleep quality.[89] Further, a recent study conducted in 217 adolescent inpatients with self-reported measures of sleep disturbance, emotional dysregulation and BPD symptoms showed that BPD features and emotion dysregulation were significantly related to indices of sleep disturbance, but there was no empirical support for the mediating role of emotion regulation in the relation between sleep and BPD.[90] Also in relation to sleep problems and emotion regulation in BPD, one study examined the level of dream disturbances (ie, nightmares) in connection with measures of emotional instability in 23 subjects diagnosed with BPD and 23 controls, and found an association between dream disturbances and levels of emotional instability in BPD patients.[91]

Therefore, the existing evidence supports the notion that sleep problems might have a role in the development of emotional dysregulation. In relation to the topic of this review, sleep problems in early childhood might lead to emotional dysregulation, which subsequently would lead to BPD symptoms. Therefore, emotional dysregulation could be treated as a mediating factor in the associations between early sleep and subsequent BPD. However, the potential mediation role of emotion dysregulation in the association between sleep and BPD should be further investigated, preferably using longitudinal approaches for large sample size of individuals.

Sleep and Internalizing/Externalizing Problems

BPD is highly comorbid with internalizing and externalizing psychopathology.[92] Concerning sleep in childhood, one common finding is the links with internalizing and externalizing difficulties. Some studies have focused on internalizing and externalizing problems separately, while others have included both constructs together.

How Do Sleep and Internalizing/Externalizing Symptoms Associate in Adolescence and Childhood?

Concerning the associations between sleep and internalizing problems, in a study in 300 eight-year-old twin pairs, several sleep difficulties were associated with depression, and the overall association was largely influenced by genes.[93] Following the research on the genetic contribution, a recent study conducted with 5111 adolescent twin pairs reported also a large genetic overlap between sleep problems and internalizing symptoms.[94] Some other studies have focused on the prospective associations between sleep and internalizing symptoms. In a recent longitudinal study among 319 youth (aged 11–26 years), dysregulated sleep was bidirectionally associated with clinician-rated anxiety and depression symptom severity.[95] In another recent study in 993 and 736 participants (longitudinal cohort entry age of 10/11 years), sleep duration was not

longitudinally associated with internalizing problems but it was in the cross-sectional analyses.[96] Further, a longitudinal study conducted in 2,510 adolescents reported the effect of sleep duration and cigarette smoking on later internalizing problems.[97] Another longitudinal study conducted in 1,089 children found that bedtime but not nighttime waking problems significantly predicted adolescent internalizing problems.[98] In younger ages, a recent longitudinal study conducted in 35,075 mothers of children between 1.5 and 8 years old showed a bidirectional association between subjective sleep and internalizing symptoms from toddlerhood to middle childhood.[99] Further, and using actigraphy measures, a recent study conducted in 119 children assessed longitudinally at 30, 36, 42, and 54 months reported that sleep variability and late sleep timing in toddlerhood were associated with higher levels of internalizing problems.[100]

Regarding the research on the associations between sleep and externalizing problems, in a longitudinal study by Muratori et al in 227 children (6–10 years),[101] increased sleep problems associated with worsening of inattentive and hyperactive behavioral problems one year later. Similarly, in a cross-sectional study in 8950 preschoolers, shorter nighttime sleep duration was associated with higher likelihood of parent-reported externalizing symptoms.[102] Also in preschoolers, a study of 510 children aged 2 to 5 years reported that parent-reported short sleep was associated with more externalizing problems.[103] Further, in a recent study conducted in 196 children aged 34–77 months externalizing problems rather than cognitive development were associated with sleep difficulties in preschool children with developmental disability.[104]

Most of the studies on the topic have examined the effects of sleep with both internalizing and externalizing symptoms. In adolescents, in a longitudinal study in 555 young adolescents (Mean age = 13.96), sleep problems were precursors of substance use, internalizing and externalizing problems in adolescence.[105] Concerning studies in childhood, in a cohort study using data obtained every 2 years in 4983 children aged 4–5 years, bidirectional associations were found between sleep problems and externalizing but not with internalizing symptoms.[106] Further, in a longitudinal study including 270 children with ADHD, aged 5–13 years, the main findings showed a weak evidence of a bidirectional relationship between sleep problems and internalizing/externalizing problems.[107]

In younger children, in a study conducted in 297 families with 5–6-year-old preschoolers, both short sleep duration and sleeping difficulties were associated with children's behavioral symptoms.[108] Childhood sleep problems have also been related to maternal ratings of internalizing and externalizing problems during adolescence for both boys and girls in a study with 292 boys and 94 girls from a community sample of high-risk families.[109] Further, a recent study conducted in 48 boys and 74 girls aged 7–11 years reported that parental reports of sleep disturbances can be used to identify children at increased risk for internalizing and externalizing problems.[110] Another study conducted in a sample of 285 elementary school students found that self-reported time in bed and sleep quality was negatively associated with internalizing and externalizing symptoms.[111] In a recent longitudinal study, Williamson et al examined 4517 children recruited at birth and found that children with increased middle childhood sleep problems experienced greater internalizing and externalizing symptoms at 10–11 years.[112]

Also, in a recent longitudinal study with more than 1000 infants and toddlers,[113] short sleep duration and more night wakings in infancy were associated with internalizing symptoms at 24 months; and more night wakings and longer sleep-onset latency in infancy with externalizing problems at 24 months. Another study in 123 toddlers reported that short sleep predicted greater externalizing behaviors a year later.[114] In a 2017 study, later bedtimes and less total sleep predicted social-emotional problems in infants and toddlers.[115] A larger longitudinal study conducted in 32,662 children reported that short sleep duration and frequent nocturnal awakenings at 18 months significantly predicted both concurrent and later incidence of emotional and behavioral problems at 5 years.[116] In a cross-sectional study among a community sample of children and adolescents (N = 175), depressive symptoms showed a greater association with sleep problems among adolescents, while anxiety symptoms were generally associated with sleep problems in all youth.[117] Similarly, a longitudinal study in 1001 toddlers from 24- to 36-months found specific relations between sleep problems and internalizing and externalizing problems during toddlerhood.[118]

To sum up, these findings suggest that there is a robust association between sleep problems and both internalizing and externalizing problems at different stages from infancy to young adulthood. Several studies suggest the existence of a bidirectional association, but more evidence supports the notion that sleep affects internalizing/externalizing symptoms, rather than the opposite direction. However, further research is still needed. In the context of this

review, we suggest that early sleep problems might underlie the development of internalizing/externalizing problems which subsequently may lead to BPD symptoms. However, another approach could be that internalizing/externalizing symptoms are a common factor of sleep and BPD symptoms, thus further investigation is required.

Table 1 describes the key features of each of the studies included in this narrative review.

Conclusion

There are several pathways that could explain why sleep problems and subsequent BPD symptoms associate (see Figure 2). In this review, we used the biosocial developmental model of BPD as a framework to discuss how sleep problems might associate with the development of BPD including some of the correlates, risk and/or etiological factors described in this model. For instance, here we present evidence that supports the link between early sleep and some of the most relevant features that characterize BPD (ie, dysfunction of the HPA axis and prefrontal cortex, family psychopathology, disrupted attachment, childhood maltreatment, impulsivity, emotion regulation, and internalizing/externalizing symptoms). Therefore, this suggests that some of these factors could act as mediating factors in the associations between sleep and subsequent BPD symptoms (ie, HPA, prefrontal functioning, emotional dysregulation, impulsivity and internalizing/externalizing symptoms), while some other factors might constitute a common risk factor (ie, family psychopathology, disrupted attachment and childhood maltreatment). Other potential explanations using the biosocial developmental model of BPD could relate to the fact that early sleep problems could be an indicator of early difficulties with regulation, which in turn would predict higher levels of co-developing internalizing and externalizing symptoms which subsequently lead to the development of BPD psychopathology in adolescence.[119] In addition, there is some evidence which suggests that sleep disturbance might play a causal role in some of these processes. To ascertain this causal role of sleep, experimental designs such as sleep manipulation are required.[120] For instance, there is evidence for a causal link between sleep deprivation and deficits at the level of the prefrontal cortex, as well as increased impulsivity and/or dysregulation problems, using cross-sectional studies. Further, longitudinal studies partially support the fact that early sleep problems might potentially cause disruptions in the development of secure infant-mother attachment and externalizing and internalizing symptoms in childhood. These studies report that sleep problems precede the development of subsequent child and family-related difficulties, and although longitudinal designs cannot fully determine causality, their analyses meet some of the Bradford Hill criteria for causation, such as temporality and plausibility.[121] However, there is still a lack of studies investigating the potential mechanisms by which sleep disturbances might lead to some of these processes. There is a need for further research on the potential underlying mechanisms such as family factors, parenting strategies, or biochemical changes in the central nervous system.

A strength of the literature included in this review is the inclusion of a variety of processes potentially associated with sleep and which could explain the link between sleep and BPD. Therefore, this provides evidence of a number of potential mechanisms underlying these associations and allows us to advance on the current knowledge of the topic. Further, to do so, this narrative review uses as a framework the biosocial development model of BPD, which is a well-tested model of BPD. Other strength of this narrative review is the inclusion of both subjective and objective sleep measures. Further, we also included not only cross-sectional studies but also longitudinal and sleep manipulation studies, which allows to decipher the potential causal role of sleep in some of these processes. However, we only focused here on some of the processes presented in the biosocial development model of BPD, while some other potentially relevant mechanisms are not discussed in this narrative review, which constituted a disadvantage of the present work. Further, other limitations of the current review of the literature are (i) the small sample size used in some of the studies; (ii) many of the studies were cross-sectional and thus causality cannot be inferred; and (iii) a relatively short follow-up period in most of the longitudinal studies included. In addition, due to the nature of the current review (ie, narrative review), this was limited by the subjective nature of its methodology, rather than presenting the operationalization of the variables, the magnitude of effect sizes, and/or the sample sizes, which is characteristic of a systematic review. Further, we only included two databases as search engines and there was no critical appraisal of the included studies. Future studies should consider conducting a systematic review and/or meta-analysis to reveal patterns in the current literature that could not be detected through a narrative review.

Table 1 Description of the Studies Included in the Narrative Review

	Study Design	N	Age/Age Grouping	Sleep Measure		Main Findings
				Objective	Subjective	
Sleep and HPA						
El-Sheikh et al (2008)[13]	Cross-sectional	64	Mean=8.75 years Age grouping: childhood.	Actigraphy: total sleep minutes, sleep efficiency, minutes awake after sleep onset, and sleep activity	The Sleep Habits Survey: sleepiness and sleep/wake problems.	Cortisol measure: children came to the laboratory to provide saliva samples, which were used to assess cortisol. There was a significant relationship between higher levels of cortisol and increased subjective sleep problems, poorer sleep quality and shorter sleep duration.
Fernandez-Mendoza et al (2014)[15]	Cross-sectional	327	Range=5–12 years Age grouping: childhood.	Polysomnography: sleep duration	Pediatric Behavior Scale: insomnia symptoms of difficulty initiating and/or maintaining sleep.	Cortisol measure: children provided evening and morning saliva samples to assay for cortisol. There was a significant association between parent-reported insomnia symptoms with objective sleep duration and higher cortisol levels in the evening and morning.
Hatzinger et al (2008)[17]	Cross-sectional	67	Mean=4.91 years Age grouping: childhood.	Polysomnography: REM sleep time, number of awakenings after sleep onset, and sleep efficiency	N/A	Cortisol measure: for baseline HPA-activity assessment, saliva samples were collected immediately after awakening, whereas saliva samples before, while and after a psychological challenge were used to assess the HPA-activity under stress conditions. "Poor" sleepers showed significantly increased morning cortisol levels. An elevated number of awakenings after sleep onset were significantly associated with increased cortisol levels under stress conditions.
Hatzinger et al (2010)[18]	Cross-sectional	82	Mean=4.91 years Age grouping: childhood.	Actigraphy: sleep onset latency, awakening time, number and times of awakenings after sleep onset, sleep period time and sleep efficiency	Daily log of their child's sleep: bedtime, wake-up time, sleep duration, awakenings.	Cortisol measure: on the first and on the last morning of sleep registration, the activity of the HPA axis was assessed via the amount of cortisol in the saliva. Children who have poor sleep patterns with high HPA activity showed extended sleep onset latency and sleep efficiency.
Kiel et al (2015)[20]	Longitudinal	51	Mean=18.96 months Age grouping: toddlers.	N/A	Infant-Toddler Social and Emotional Assessment: Sleep scale (5 items).	Cortisol measure: Mothers were trained by laboratory staff to gather saliva from their toddlers using a cotton dental roll. High morning levels of cortisol predicted increasing sleep problems from age 2 to age 3.

(*Continued*)

Table 1 (Continued).

	Study Design	N	Age/Age Grouping	Sleep Measure		Main Findings
				Objective	Subjective	
LaVoy et al (2020)[10]	Cross-sectional	55	Range=8–16 years Age grouping: adolescence.	Actigraphy: minutes awake, sleep efficiency, sleep duration, sleep onset latency and night awakenings	Sleep diaries, The School Habits Survey; sleep duration, night awakenings, wake-up and bedtime, sleepiness and sleep/wake behaviour problems.	Cortisol measure: participants donated first waking saliva samples, which were later assayed for α-amylase (sAA), cortisol, interleukin (IL)-6, and IL-1β. Morning cortisol levels were associated with objective sleep variables such as sleep efficiency, minutes spent awake the night before and night awakening the night after.
Lemola et al (2015)[12]	Cross-sectional	113	Mean=8.3 years Age grouping: childhood.	Sleep-electroencephalography: sleep duration, sleep continuity, and sleep architecture.	N/A	Cortisol measure: The parents of the children were instructed to collect four saliva samples on the following morning, with the first one after awakening and 10, 20, and 30 minutes later. Cortisol secretion was negatively associated with sleep duration in school age children and slow wave sleep and positively associated with the stage 2 sleep during the preceding night.
Pesonen et al (2012)[14]	Cross-sectional	248	Mean=8.1 years Age grouping: childhood.	Actigraphy: sleep duration.	Sleep Disturbance Scale for Children: sleep-wake transition, arousal, excessive daytime somnolence, sleep hyperhidrosis.	Cortisol measure: salivary cortisol was sampled throughout one day at home and during the Trier Social Stress Test for Children (TSST-C) in clinic. Boys with parent-reported sleep problems had lower levels of diurnal salivary cortisol levels. Girls with sleep problems had unaltered diurnal salivary cortisol.
Pesonen et al (2014)[9]	Longitudinal	305	Mean=123 years Age grouping= Adolescence	Actigraphy: sleep duration, awakening times, temporary pauses in actigraph registration, sleep quality or quantity.	N/A	Cortisol measure: Saliva was obtained during a 2-day period at awakening and 15, 30, 45, 60 minutes after awakening, at 12:00, at 17:00 and at bedtime. Long sleep duration in boys associated with higher cortisol awakening response (CAR). Lower sleep quality in boys was associated with lower CAR. In girls, no significant associations were found.

(*Continued*)

Table 1 (Continued).

	Study Design	N	Age/Age Grouping	Sleep Measure		Main Findings
				Objective	Subjective	
Räikkönen et al (2010)[11]	Cross-sectional	282	Age=8 years Age grouping: childhood.	Actigraphy: sleep duration and efficiency	Sleep log: bed and awakening times.	Cortisol measure: diurnal salivary cortisol and salivary cortisol and α-amylase (a sympatho-adrenal-medullary system marker) responses to the Trier Social Stress Test for Children (TSST-C) were measured. Children with short average sleep duration displayed higher salivary cortisol awakening response. Children with low sleep efficiency displayed higher diurnal cortisol levels.
Saridjan et al (2017)[19]	Longitudinal	322	Range=12–20 months Age grouping: infancy/ toddlers.	N/A	Parental report, Child Behavior Checklist: Sleep duration and sleep behaviour.	Cortisol measure: the diurnal cortisol rhythm was determined by calculating the area under the curve (AUC), the cortisol awakening response (CAR), and the diurnal slope. Infants with a flatter cortisol slope and those with a more marked morning cortisol rise had shorter nighttime sleep duration.
Zeiders et al (2011)[8]	Cross-sectional	119	Mean=19.01 years Age grouping: young adults.	Actigraphy: hours of sleep and wake times	Sleep diary: hours of sleep and wake times.	Cortisol measure: participants provided six cortisol samples (wake-up, +30 minutes, +2 hours, +8 hours, +12 hours, and bedtime) on each of three consecutive days while wearing an actigraph. The hours of sleep of the previous night predicted steeper diurnal slopes the next day. Greater waking cortisol levels and steeper slopes predicted more hours of sleep and a later wake time the next day.
Sleep and Prefrontal Cortex						
Cheng et al (2020)[27]	Cross-sectional	11,067	Range=9–11 years Age grouping: childhood.	N/A	Parent Sleep Disturbance Scale for Children: sleep duration.	Higher volume in orbitofrontal cortex, prefrontal and temporal cortex, precuneus and supramarginal gyrus was correlated with longer sleep duration.
Kocevska et al (2017)[31]	Longitudinal	720	From 2 months old until 7 years. Age grouping: From toddlerhood until childhood	N/A	Parental reports of postal questionnaires, parental reports of the Child Behavior Checklist: sleep disturbance.	Sleep disturbances from age 2 onwards were associated with smaller grey matter volumes and thinner dorsolateral prefrontal cortex.

(*Continued*)

Table 1 (Continued).

	Study Design	N	Age/Age Grouping	Sleep Measure		Main Findings
				Objective	Subjective	
Lunsford-Avery et al (2020)[26]	Cross-sectional	25	Mean=18.08 years Age grouping: young adults.	Actigraphy: Sleep regularity	N/A	Irregular sleep patterns were significantly associated with increased path length within the default mode network, specifically in the right and left lateral parietal lobule.
Reidy et al (2016)[30]	Cross-sectional	15	Range=7–11 years Age grouping: childhood.	N/A	Parental report of child's sleep duration.	Sleep duration linked positively to amygdala-prefrontal functional connectivity.
Telzer et al (2013)[29]	Cross-sectional	46	Mean=15.23 Age grouping: Adolescence.	N/A	Pittsburgh Sleep Quality Index: poor sleep quality.	Adolescents showed less dorsolateral prefrontal cortex (DLPFC) activation during cognitive control, greater insula activation during reward processing, and reduced functional coupling between the DLPFC and affective regions.
Urrila et al (2017)[28]	Cross-sectional	177	Mean=14.4 years Age grouping: adolescence.	fMRI	Adolescents report on habitual bedtimes and wake-up times.	The medial prefrontal cortex was significantly associated both with bedtime and wake-up times during weekends, also with poor school performance.
Sleep and Family Psychopathology						
Bat-Pitault et al (2017)[48]	Cross-sectional	64	Age=<37 weeks Age grouping: infancy.	Polysomnography: Sleep macro-structural parameters	N/A	Maternal psychopathology measurement: being born to mothers diagnosed with depression. Altered sleep structure was observed during the first months of life in infants born from depressed mothers.
Dennis & Ross (2005)[39]	Longitudinal	505	Age= Newborns Age grouping: infancy.	N/A	Maternal report of infant's sleep patterns: number of night awakenings, sleep duration, sleep quality, and enough sleep.	Maternal psychopathology measurement: Edinburgh Postnatal Depression Scale. Infant sleep patterns and maternal fatigue were strongly associated with a new set of depressive symptoms in the postpartum period.
Dias & Figueiredo (2020)[38]	Longitudinal	164	Age= Newborns Age grouping: infancy.	N/A	Children's Sleep Habits Questionnaire: sleep anxiety, daytime sleepiness and bedtime resistance.	Maternal psychopathology measurement: Edinburgh Postnatal Depression Scale. Both mother's prenatal and postpartum depression symptoms predicted infant's sleep difficulties.

(*Continued*)

Table 1 (Continued).

	Study Design	N	Age/Age Grouping	Sleep Measure		Main Findings
				Objective	Subjective	
Field et al (2007)[35]	Longitudinal	253	Age= Newborns Age grouping: infancy.	N/A	Sleep scale: quiet sleep (no REM), active sleep (without REM), REM sleep, drowsy, inactive alert, active awake, crying, indeterminate sleep.	Maternal psychopathology measurement: SCID diagnosis of depression, and self-report measures on depression, anxiety and anger. The infants of the depressed mothers had more sleep difficulties such as less time in deep sleep and more time in disorganised sleep.
Gregory et al (2005)[46]	Cross-sectional	6612	Range=3–4 years Age grouping: childhood.	N/A	Parental report: hard to go to sleep, frequent awakenings, nightmares, and early waking.	Family psychopathology measurement: parent-report of family disorganization and depression. Maternal depression and family disorganization were associated with sleep problems and anxiety in twins.
Halal et al (2021)[36]	Longitudinal	2222	Age=Newborns Age grouping: infancy.	Actigraphy: sleep duration, night awakenings and sleep efficiency.	Brief Infant Sleep Questionnaire: sleep duration and night awakenings.	Maternal psychopathology measurement: Edinburgh Postnatal Depression Scale. Prenatal maternal depression predicted more sleep problems in newborns.
Hiscock & Wake (2001)[41]	Cross-sectional	738	Range=6–12 months Age grouping: infancy.	N/A	Maternal report: infant sleeping in the parent's bed, being nursed to sleep, time to fall asleep, night awakenings, and naps duration.	Maternal psychopathology measurement: Edinburgh Postnatal Depression Scale. Infant sleep problems were associated with maternal postnatal depression scores.
Martin et al (2007)[49]	Cross-sectional	5107 infants 4983 pre-school	Range= Newborns and 4–5 years Age grouping: infancy and children.	N/A	Maternal report: whether their child's sleep was a problem (no, mild, moderate or severe problem).	Parents psychopathology measurement: the Kessler-6. Preschool sleep problems were associated with poor maternal general health.
Morales-Munoz et al (2018)[52]	Longitudinal	1221	Age= Newborns Age grouping: infancy.	N/A	The Brief Infant Sleep Questionnaire: number of nocturnal sleep hours; number of daytime sleep hours; total number of sleep hours per day; and method for falling asleep.	Maternal psychopathology measurement: The Alcohol Use Disorders Identification Test, The Global Seasonality Score, The List of Threatening Experiences, The State-Trait Anxiety Inventory, The Center for Epidemiological Studies Depression Scale and The Adult ADHD Self-Report Scale. Several maternal risk factors, including anxiety, depression, insomnia or ADHD during pregnancy were related to sleep difficulties.

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Table 1 (Continued).

	Study Design	N	Age/Age Grouping	Sleep Measure		Main Findings
				Objective	Subjective	
O'Connor et al (2007)[37]	Longitudinal	11,490	Age= Newborns Age grouping: infancy.	N/A	Parental report: amount of sleep, frequency of awakening, child regularly refuses to go to bed, wakes very early, difficulty going to sleep, nightmares, get up after being put to bed, woke in the night, or got up after few hours sleep.	Maternal psychopathology measurement: Crown-Crisp experiential index and the Edinburgh Postnatal Depression Scale. Higher levels of prenatal maternal depression and anxiety predicted more sleep difficulties at 18 and 30 months.
Petzoldt et al (2016)[44]	Longitudinal	286	Age= Newborns Age grouping: infancy.	N/A	Baby-DIPS: sleeping problems.	Maternal psychopathology measurement: Composite International Diagnostic Interview for Women (CIDI-V) to assess DSM-IV anxiety and depressive disorders. Infant sleeping difficulties were associated with maternal depressive and comorbid anxiety disorders irrespective of maternal parity.
Reid et al (2009)[45]	Cross-sectional	8868	Range=2–3 years Age grouping: toddlers.	N/A	Parental report: trouble falling asleep, particular and long bedtime routine, and waking up several times at night.	Maternal psychopathology measurement: 12-item short-form of the Center for Epidemiologic Studies – depression scale. Maternal symptoms of anxiety and depression during the first year of the child's life were both associated with child nocturnal awakening at 6 and 18 months.
Schuetze et al (2006)[51]	Longitudinal	118	Age= Newborns Age grouping: infancy.	N/A	The Maternal Cognitions About Infant Sleep: five subscales on Limit Setting, Anger, Doubt, Feeding, and Safety.	Maternal psychopathology measurement: The Brief Symptom Inventory. Prenatal exposure to cocaine was significantly associated with more severe sleep difficulties in newborns and maternal anxiety mediated the association.
Seifer et al (1996)[53]	Longitudinal	182	Age grouping: toddlers.	N/A	The Sleep Habits Questionnaire: total sleep time, bedtime struggles, sleep behavior, night waking, morning waking, and daytime sleepiness.	Maternal psychopathology measurement: maternal diagnostic classification. Poorly functioning families had children whose sleep habits were less optimal.
Shang et al (2006)[47]	Cross-sectional	1391	Range=4–9 years Age grouping: childhood.	N/A	The Sleep Habit Questionnaire: early insomnia, late sleep, night wakening, sleep talking, sleepwalking, nightmare, enuresis, bruxism and snoring.	Maternal psychopathology measurement: The Chinese Health Questionnaire. Parental distress, including maternal depression, was significantly associated with children's sleep problems.

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Table 1 (Continued).

	Study Design	N	Age/Age Grouping	Sleep Measure		Main Findings
				Objective	Subjective	
Stoléru et al (1997)[42]	Longitudinal	85	Range=1–12 years Age grouping: infancy, toddlers, childhood, early adolescence.	N/A	The Child Behavior Checklist: sleep problems.	Maternal psychopathology measurement: diagnosis of unipolar and bipolar affective illness. Sleep problems were more frequent and severe in children of affectively ill mothers.
Warren et al (2003)[50]	Cross-sectional	94	Age= Newborns Age grouping: infancy.	Videotaping	The Sleep Habits Questionnaire, Sleep logs: early insomnia, late sleep, night wakening, sleep duration.	Maternal psychopathology measurement: diagnosis of panic disorder. Infants from the 4-month cohort with panic disorder mothers had more awakenings and greater time awake during the night at 8 months.
Ystrom et al (2017)[43]	Longitudinal	14,926	Age= Newborns Age grouping: infancy.	N/A	Parental report: nocturnal awakenings.	Maternal psychopathology measurement: Hopkins Symptom Checklist (SCL-8). Maternal symptoms of anxiety and depression in infants were both associated with child nocturnal awakening at 6 and 18 months.
Sleep and Disrupted Attachment						
Belanger et al (2018)[57]	Longitudinal	64	Mean=25.35 months Age grouping: toddlers.	Actigraphy: sleep duration, sleep efficiency.	Sleep diaries, Child Behavior Checklist: sleep duration, sleep efficiency, sleep difficulties.	Securely attached children subsequently slept more at night and had higher sleep efficiency.
Beijers et al (2011)[60]	Longitudinal	193	Age= Newborns Age grouping: infancy.	N/A	Sleep diaries: night awakenings.	Infants with an insecure-resistant attachment style at 12 months of age woke up more during the night in their first 6 months of life than the other infants.
Bilgin & Wolke (2020)[63]	Longitudinal	105	Age= Newborns Age grouping: infancy.	N/A	Maternal report: symptoms of sleeping problems.	Sleep problems in infants at 3 months were associated with disorganized attachment with the mother at 18 months.
McNamara et al (2003)[62]	Longitudinal	342	Age= Newborns Age grouping: infancy.	N/A	Maternal report: sleep problems.	Infants with insecure-resistant attachments had greater numbers of night wakings and longer mean durations of night waking episodes than infants with insecure-avoidant attachment style.
Pennestri et al (2015)[58]	Longitudinal	134	Age= Newborns Age grouping: infancy.	N/A	Maternal report: duration of sleep, bedtime, awakenings, and periods of time in bed.	Infants with disorganised attachment had more sleep difficulties than infants with secure or ambivalent attachment style.

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Table 1 (Continued).

	Study Design	N	Age/Age Grouping	Sleep Measure		Main Findings
				Objective	Subjective	
Simard et al (2013)[59]	Longitudinal	55	Age=18 months Age grouping: toddlers.	Actigraphy	Sleep diaries: sleep efficiency, wake duration at night, sleep duration.	Higher resistance attachment behavior predicted longer wake duration at night in children.
Zentall et al (2012)[61]	Longitudinal	46	Age= Newborns Age grouping: infancy.	N/A	Parent reported night waking.	Infants with a secure pattern of attachment with mothers had lower number of night wakings over time.
Sleep and Childhood Maltreatment						
April-Sanders et al (2021)[74]	Longitudinal	2491	Range=5–9 and 10–16 years Age grouping: childhood and adolescence.	N/A	Parent and youth report; prevalence of sleep disturbances.	Childhood adversity was associated with sleep disturbances in youth of 10–16 years of age, but not in 5–9 years of olds.
Glod et al (1997)[71]	Cross-sectional	44	Range=6–12 years Age grouping: childhood and early adolescence.	Actigraphy: nocturnal activity and algorithmic estimation of sleep initiation and continuity.	N/A	Abused children were twice as active at night as normal and depressed children.
Hamilton et al (2018)[66]	Cross-sectional	102	Range=18–22 years Age grouping: young adulthood.	N/A	Sleep diary: sleep duration and insomnia symptoms.	Only childhood emotional neglect significantly predicted higher levels of insomnia symptoms.
Kaubrys et al (2021)[67]	Cross-sectional	181	Age grouping: college students.	N/A	Self-reports on sleep: sleep efficiency, sleep duration, sleep latency, sleep quality, and restful sleep.	Experiencing more daily stressors than usual was significantly associated with delayed sleep latency and lower sleep efficiency. Students who reported more childhood maltreatment also reported significantly lower quality of sleep and feeling less rested upon awakening.
McPhie et al (2014)[68]	Longitudinal	73	Mean=15.8 Age grouping: adolescence.	N/A	Self-reports on current sleep disturbances.	More severe childhood maltreatment was related to increased sleep problems during adolescence. Psychological distress was a significant mediator of the childhood maltreatment-adolescent sleep disturbance association.
Noll et al (2006)[73]	Longitudinal	147	Range=6–16 years Age grouping: childhood and adolescence.	N/A	Subjective reports of sleep: typical sleeping patterns and sleep disturbances.	Sexually abused female adolescents reported significantly greater rates of sleep disturbances than controls.

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Table 1 (Continued).

	Study Design	N	Age/Age Grouping	Sleep Measure		Main Findings
				Objective	Subjective	
Turner et al (2020)[69]	Cross-sectional	2910	Range=14–17 years Age grouping: adolescence.	N/A	Subjective Reports of sleep experiences: time to fall asleep, waking during the night, and hours of sleep.	All types of childhood maltreatment were significantly related to increased odds of taking more than 10 minutes to fall asleep, fewer hours slept on weekdays and waking more often during the night.
Wang et al (2016)[72]	Cross-sectional	9582	Range=13–18 years Age grouping: adolescence and young adulthood.	N/A	The International Classification of Sleep Disorders II: risk of insomnia.	Adolescents exposed to at least one childhood adversity of any type were more likely to experience insomnia than nonexposed children.
Xiao et al (2019)[70]	Cross-sectional	153,547	Mean=15.0 years Age grouping: adolescence.	N/A	The Chinese Version of the Pittsburgh Sleep Quality Index: sleep disturbance.	Sleep disturbance was significantly associated with physical abuse, emotional abuse, sexual abuse, physical neglect, and emotional neglect in adolescents.
Sleep and Impulsivity						
Fallone et al (2001)[79]	Cross-sectional	82	Mean=11.9 years Age grouping: early adolescence.	Actigraphy, Polysomnography: daytime sleep latency.	Multiple sleep latency test, Subjective sleepiness ratings: sleep latency and subjective sleepiness.	Sleepiness following acute sleep restriction was not sufficient to produce deficits on impulsivity and sustained attention.
Gershon et al (2019)[77]	Cross-sectional	59	Range=13–19 years Age grouping: adolescence and young adulthood.	N/A	Self-report questionnaire: bedtime, rise time, and sleep duration.	Greater change in sleep duration between school days and weekends was related to higher levels of impulsivity among adolescents with bipolar disorder compared to controls.
Grant & Chamberlain (2018)[76]	Cross-sectional	373	Range=18–29 years Age grouping: young adulthood.	N/A	The Epworth Sleepiness Scale: sleepiness.	Higher levels of sleepiness were significantly associated with a range of impulsive measures.
Gruber et al (2012)[81]	Cross-sectional	34	Range:7–11 Age grouping: Childhood	Actigraphy: sleep duration, sleep quality	The Modified Epworth Sleepiness Scale	A cumulative extension of sleep duration of 27.36 minutes was associated with improvement in emotional lability and restless-impulsive behaviour and a significant reduction in reported daytime sleepiness. A cumulative restriction of sleep of 54.04 minutes was associated with detectable deterioration on emotion regulation and impulsivity.
Medeiros et al (2005)[80]	Cross-sectional	1180	Range=7–10 years Age grouping: childhood.	N/A	Parental report: sleep disorders and sleep-related respiratory disorders.	Children with non-respiratory sleep disorders had greater predisposition to impulsivity than children without sleep disorders.

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Table 1 (Continued).

	Study Design	N	Age/Age Grouping	Sleep Measure		Main Findings
				Objective	Subjective	
Rossa et al (2014)[78]	Cross-sectional	19	Mean=20.16 years Age grouping: young adulthood.	N/A	Karolinska Sleepiness Scale: sleepiness.	An acute sleep restriction paradigm significantly impacted on affective experiences and increased impulsive and high-risk behaviour in young adults.
Sleep and Emotion dysregulation						
Alfano et al (2020)[86]	Cross-sectional	53	Mean=9.0 Age grouping: Childhood	Polysomnography and actigraphy: sleep duration and sleep quality.	N/A	Deleterious alterations were observed in children's affect, emotional arousal, facial expressions, and emotion regulation after sleep restriction.
Baum et al (2014)[85]	Cross-sectional	50	Range=14–17 years Age grouping: adolescence.	Actigraphy: sleep duration.	Sleep diary: sleep duration.	Several nights of sleep restriction adversely affected adolescents' mood and ability to regulate their emotions.
Berger et al (2012)[87]	Cross-sectional	10	Range=30–36 months Age grouping: toddlers.	Actigraphy: sleep duration.	Children's Sleep Habits Questionnaire, Sleep diary: sleep duration.	Sleep restriction resulted in a reduction in positive responses, and an increase in negative emotion responses.
Grove et al (2017)[89]	Cross-sectional	481	Mean=23 years Age grouping: young adulthood.	N/A	The Pittsburgh Sleep Quality Index: sleep quality.	BPD features related to emotion dysregulation were most strongly associated with poor sleep quality.
Palmer et al (2018)[88]	Cross-sectional	10,148	Range=13–18 years Age grouping: adolescence and young adulthood.	N/A	Self-report: sleep problems.	Adolescents with greater sleep difficulties reported poorer emotion regulation.
Simor et al (2010)[91]	Cross-sectional	46	N/A	N/A	The Dream Quality Questionnaire: dream disturbances.	Dream disturbances were more frequent in patients with BPD than in controls.
Wall et al (2020)[90]	Longitudinal	217	Range= 12–17 Age grouping: Adolescence	N/A	The School Sleep Habits Survey: sleep disturbance.	Borderline personality features and emotion dysregulation were significantly related to sleep disturbances.
Zhang et al (2019)[84]	Cross-sectional	51	Range=18 months-3 years Age grouping: toddlers.	N/A	The Chinese Version of the Pittsburgh Sleep Quality Index: sleep quality.	Emotion regulation strategies (ie, distraction and reappraisal) were impaired by sleep deprivation.
Sleep and Internalising/Externalising disorders						
Alfano et al (2009)[117]	Cross-sectional	175	Mean=11.4 years Age grouping: childhood.	N/A	A sleep scale was created from sleep related items from various measures: sleep problems.	There were significant associations between sleep problems and both anxiety and depressive symptoms.

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Table 1 (Continued).

	Study Design	N	Age/Age Grouping	Sleep Measure		Main Findings
				Objective	Subjective	
Bai et al (2020)[95]	Longitudinal	319	Range=11–26 years Age grouping: childhood, adolescence and young adulthood.	N/A	The Physical Symptom Checklist: dysregulated sleep.	Dysregulated sleep was bidirectionally associated with clinician-rated anxiety and depression symptom severity.
Chang et al (2018)[97]	Longitudinal	2510	Range=11–12 years Age grouping: Adolescence	N/A	Subjective reports of sleep times.	Significant inter-relationships for sleep duration and cigarette smoking trajectories were found during adolescence; all atypical sleep duration trajectories conferred increased risks of cigarette smoking. The effects of sleep duration and cigarette smoking on later internalizing problems were found to vary by sex and trajectory patterns.
Cho et al (2017)[114]	Longitudinal	123	Mean=24.43 years Age grouping: young adulthood.	N/A	Parental report: sleep duration.	Short sleep predicted greater daytime externalizing behaviors a year later.
Conway et al (2017)[118]	Longitudinal	1001	Range=24–36 months Age grouping: toddlers.	N/A	Parental report: trouble getting to sleep, bedtime resistance, sleep problems.	There were reciprocal associations between trouble getting to sleep and internalizing problems, and unidirectional links between externalizing problems and bedtime resistance.
Gregory et al (2006)[93]	Cross-sectional	300	Mean=8.6 years Age grouping: childhood.	N/A	The Child Sleep Habits Questionnaire: sleep problems.	A range of sleep difficulties were associated with depression in children.
Gruber et al (2020)[110]	Cross-sectional	122	Range=7–11 Age grouping: Childhood	Actigraphy: Night-time sleep, sleep schedule, and sleep efficiency	Child Sleep Habits Questionnaire, parental report on daily sleep logs: sleep disturbances.	Children who were above the cutoff score of the Children Sleep Habits Questionnaire were less likely to obtain the recommended amount of sleep for their age, had higher levels of internalizing and externalizing symptoms and a higher prevalence of clinical levels of externalizing and internalizing problems, had lower grades in English and French and were more likely to fail these subjects.

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Table 1 (Continued).

	Study Design	N	Age/Age Grouping	Sleep Measure		Main Findings
				Objective	Subjective	
Hoyniak et al (2020)[100]	Longitudinal	119	30 months of age Age grouping: Childhood	Actigraphy: sleep duration, sleep timing, sleep vulnerability, and sleep activity	Parental report on daily sleep diaries	More variable sleep at 30 months had higher teacher-reported internalizing problems in preschool. Children with later sleep timing at 30 months had poorer cognitive and academic skills at 54 months. Changes in sleep from 30 to 54 months were not associated with any of the domains of adjustment.
Lavigne et al (1999)[103]	Cross-sectional	510	Range=2–5 Age grouping: Childhood	N/A	Parental report on the amount of sleep child obtained	The relationship between less sleep at night and the presence of a DSM-III-R psychiatric diagnosis was significant. Less night sleep and less sleep in a 24 hour period were associated with increased total behavior problems on the Child Behavior Checklist and with more externalizing problems.
Lee et al (2021)[104]	Cross-sectional	196	Mean=59.6 months Age grouping: Childhood	N/A	Children's Sleep Habits Questionnaire: poor and good sleepers.	Externalising problems such as aggressive behaviors and attention problems, rather than cognitive development, are associated with sleep difficulties in preschool children with developmental disability.
Madrid-Valero et al (2020)[94]	Cross-sectional	5111 twin pairs	Mean=16.32 years Age grouping: adolescence.	N/A	The Pittsburgh Sleep Quality Index, The Insomnia Severity Index: sleep quality and insomnia.	Associations between sleep and internalizing problems were moderate and there was a large genetic overlap between these problems.
Mindell et al (2017)[115]	Longitudinal	117	Range=3–18 months Age grouping: toddlers.	N/A	The Brief Infant Sleep Questionnaire: bedtime, sleep duration, sleep latency and night awakenings.	Later bedtimes and less total sleep predicted higher internalising problem scores.
Morales-Muñoz et al (2020)[113]	Longitudinal	936	Age= Newborns Age grouping: infancy.	N/A	The Brief Infant Sleep Questionnaire, The Infant Sleep Questionnaire: sleep duration, sleep latency and night awakenings.	Shorter sleep and poorer sleep quality in infancy were prospectively related to emotional and behavioural symptoms in toddlers.
Mulraney et al (2016)[107]	Longitudinal	270	Range=5–13 years Age grouping: childhood.	N/A	Children's Sleep Habits Questionnaire: sleep problems.	There was a weak bidirectional relationship between sleep problems and emotional problems.
Muratori et al (2019)[101]	Longitudinal	227	Range=6–10 years Age grouping: childhood.	N/A	The Sleep Disturbance Scale for Children: sleep problems.	A significant association was found between increased sleep problems and worsening of inattentive and hyperactive behavioral problems 1 year later.

(*Continued*)

Table 1 (Continued).

	Study Design	N	Age/Age Grouping	Sleep Measure		Main Findings
				Objective	Subjective	
Nunes et al (2020)[96]	Longitudinal	1729	Range=10–11 years Age grouping: childhood.	N/A	Parental and self-reports: sleep duration, difficulties getting to sleep and changes in difficulties getting to sleep.	Sleep duration was not longitudinally associated with internalizing problems, but it was in the cross-sectional analyses.
Paavonen et al (2009)[108]	Cross-sectional	297	Range=5–6 years Age grouping: childhood.	N/A	The Sleep Disturbance Scale for Children: Sleep quality and sleep duration.	Both short sleep duration and sleeping difficulties were significantly associated with children's behavioral symptoms.
Pieters et al (2015)[105]	Longitudinal	555	Mean=13.96 years Age grouping: early adolescence.	N/A	The Adolescent Sleep Wake Scale, The Adolescent Hygiene Sleep Scale: sleep quality and sleep hygiene.	Sleep problems predicted changes in substance use, internalizing and externalizing problems over time, but problematic behaviours did not predict changes in sleep problems.
Reynolds & Alfano (2016)[98]	Longitudinal	1364	Participants were assessed from birth to age 15	N/A	A set of questions was derived from the Children's Sleep Habits Questionnaire: bedtime and nighttime waking problems.	Bedtime but not nighttime waking problems significantly predicted adolescent internalising problems.
Rubens et al (2017)[111]	Cross-sectional	285	Range=8–11 years Age grouping: Childhood	N/A	Self-reports of sleep quality and time in bed.	Sleep quality was negatively associated with anxiety, depressive symptoms, irritability, reactive aggression, and delinquency engagement.
Quach et al (2018)[106]	Longitudinal	4983	Range=4–5 years Age grouping: childhood.	N/A	Parental report: difficulty getting to sleep at night, not happy sleeping alone, waking during night, and restless sleep.	Significant bidirectional associations were found between sleep and externalizing problems, with greater sleep problems associated with later externalizing behaviours.
Scharf et al (2013)[102]	Cross-sectional	8950	Age=4 years Age grouping: childhood.	N/A	Parental report: night-time sleep duration.	Shorter nighttime sleep duration was associated with higher likelihood of parent-reported externalizing behavioral symptoms.
Sivertsen et al (2015)[116]	Longitudinal	32,662	The data were collected at gestational week 17, 18 months and 5 years after birth. Age grouping: Childhood	N/A	Mother reported child sleep duration and nocturnal awakenings.	Short sleep duration and frequent nocturnal awakenings at 18 months significantly predicted both concurrent and later incidence of emotional and behavioural problems at 5 years.
Sivertsen et al (2021)[99]	Longitudinal	35,075	Range=1.5–8 years Age grouping: Childhood	N/A	Child Behavior Checklist: sleep duration and nocturnal awakenings.	Short sleep duration and frequent nightly awakenings at 1.5 years predicted the development of depressive symptoms at 8 years of age. Internalising problems at 1.5 years predicted onset of later short sleep duration.

(*Continued*)

Table 1 (Continued).

	Study Design	N	Age/Age Grouping	Sleep Measure		Main Findings
				Objective	Subjective	
Williamson et al (2020)[112]	Longitudinal	4517	Range=10–11 years Age grouping: childhood.	N/A	Parental report: sleep problems.	Children with increased sleep problems experienced greater internalizing and externalizing symptoms.
Wong et al (2009)[109]	Longitudinal	386	Range=3–11 Age grouping: Childhood	N/A	Maternal reports on Child Behavior Checklist: sleep problems.	Sleep problems at ages 3–8 predicted onset of alcohol, cigarette, and marijuana use among boys and onset of alcohol use among girls. Childhood sleep problems were related to maternal ratings of internalising and externalising problems during adolescence for both boys and girls.

Abbreviations: ADHD, attention deficit hyperactivity disorder; fMRI, functional magnetic resonance imaging; BPD, borderline personality disorder.

Further research should specifically investigate sleep in childhood and subsequent BPD symptoms in combination with some of the relevant features described in this review. Future research should also examine whether this pathway and potential factors described here are specific to BPD or whether this could also apply to other mental health problems, such as psychosis or depression. One hypothesis could be that under the combination of specific core features of BPD, such as the ones described in this model, impairment of sleep at specific stages of the development could lead to the development of subsequent BPD symptoms, instead of other mental health problems. However, further research is still required. This will help us to better understand how early sleep associates with BPD symptoms. This would lead to a better characterization of the potential underlying mechanisms of BPD symptoms and to a better understanding of how sleep disruption in early stages might lead to the development of subsequent BPD symptoms. In addition, further studies should focus on investigating whether sleep in early childhood might play a causal role in the development of BPD symptoms. Investigation of new developmental and early intervention approaches to understand how sleep in early childhood associates with subsequent BPD symptoms could have significant clinical impact on these patients and would aid to design better targeted therapeutic interventions.

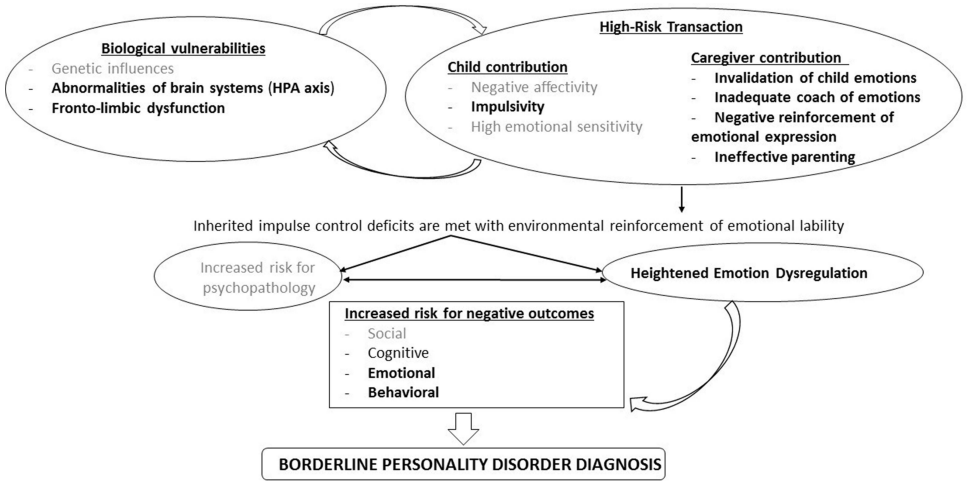

Figure 2 Adaptation of the biosocial developmental model of BPD. In this figure we present the pathways that could explain why sleep problems and subsequent BPD symptoms associate, using the biosocial developmental model by Crowell et al. In bold we have highlighted the processes that could have potential interaction with sleep problems and thus could explain the potential associations between sleep and BPD. These are HPA, prefrontal cortex, impulsivity, family psychopathology, disrupted attachment, childhood maltreatment, emotional dysregulation and internalizing/externalizing symptoms.

Disclosure

The authors report no conflicts of interest in this work.

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