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Narrative review

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Emotional behavioral disorders research: a narrative review

Abstract

This narrative review includes brief summaries of the five most common emotional behavioral disorders including oppositional defiant disorder (ODD), intermittent explosive disorder (IED), conduct disorder (CD), disruptive mood dysregulation disorder (DMDD) and social (pragmatic) communication disorder (SCD). ODD risk factors/ predictors were self-regulation problems, social difficulties, internet gaming, suicidal ideation and physical health problems. Neural synchrony was a potential underlying biological mechanism. IED risk factors/ predictors were self-regulation problems, emotional dysregulation, callous-unemotional traits and suicide attempts. The amygdala and the orbital frontal cortex were studied as potential underlying biological mechanisms. CD risk factors/predictors include callous-unemotional traits, social anxiety, approach-avoidance behavior, grandiose-manipulative and daring-impulsive traits. Potential underlying biological mechanisms include low surface area in 26 brain regions and cortical thinning as well as less volume of the amygdala, nucleus accumbens, thalamus and hippocampus. DMDD risk factors/ predictors include lower competence and social skills. Low heart rate variability was studied as a potential underlying biological mechanism. SCD was said to have verbal and nonverbal limitations as risk factors/predictors. Methodological limitations of this literature include the similar diagnostic criteria used for the different disorders, the lack of comparisons between the different disorders, and the infrequent mechanism and intervention studies.

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Introduction

Narrative review

Emotional behavioral disorders (EBD) in the Diagnostic and Statistical Manual of Mental Disorders (DSM-5)[1] have been characterized by persistent and significant difficulties in emotion regulation, behavior control and social interactions. The five most common emotional behavioral disorders are oppositional defiant disorder, intermittent explosive disorder, conduct disorder, disruptive mood dysregulation disorder and social (pragmatic) communication disorder.

Oppositional defiant disorder is defined as a pattern of angry or irritable mood, defiant behavior and vindictiveness. Intermittent explosive disorder is characterized as recurrent episodes of impulsive aggression during which the individual may verbally or physically harm others. Conduct disorder is more severe than oppositional defiant disorder involving a persistent pattern of violating societal rules such as aggression, theft and deceit. Disruptive mood dysregulation disorder is a chronic and persistent mood of irritability, anger and frequent temper outbursts. Social (pragmatic) communication disorder is characterized by difficulties understanding and using social cues, resulting in impaired social relationships.

The specific diagnostic criteria for EBD vary but generally involve persistent and significant symptoms that interfere with daily functioning. Several factors are considered in making the diagnoses including symptoms that are not attributable to other mental health conditions or developmental factors and symptoms that meet specific age- appropriate thresholds. EBDs are relatively common in children and adolescents, affecting an estimated 3 to 5% of the population. These disorders can have a significant impact on academic performance, social relationships and family functioning.

In this narrative review, brief summaries are given on studies that were published in 2024-2025 on the five most common emotional behavioral disorders. This research was found on PubMed and PsycINFO by entering the specific emotional behavioral disorder and the years 2024-2025. Exclusion criteria included proposed protocols, pilot studies and non-English papers. The current literature on these emotional behavioral disorders is primarily focused on the symptoms and/or correlates of the disorders that are also the focus of this narrative review.

Oppositional defiant behavior disorder (ODD)

Oppositional defiant disorder has been conceptualized as a self-regulation disorder. It is usually diagnosed in childhood and consists of negative reactions to authority, noncompliance, irritable mood and negative attention-seeking behaviors. Like other behavioral disorders, ODD is often comorbid with anxiety and mood disorders as well as ADHD.

Risk factors/predictors

Several risk factors/predictors for ODD have appeared in this literature (see Table 1). These include self- regulation, emotion regulation, social difficulties, internet gaming disorder, physical health problems and lower neural synchrony.

Table 1 Oppositional defiant disorder risks and interventions (and first authors)

Risks	First authors
Self-regulation problems	Zhang
Emotion regulation problems	Obakht
Social difficulties	Braengan
Internet gaming disorder	Keshky
Physical health problems	McDowell
Lower neural synchrony	Chang
Intervention	
Parent management training	Dedousis-Wallace

In a study on 504 grade school children (ages 6-11 years), parents and classroom teachers completed questionnaires and were followed up for an assessment six months later.[2] Using moderation network analysis, the relationship between **self-regulation** and ODD symptoms was explored. Self-regulation including emotion regulation, self-control and emotional lability/ negativity were bidirectionally related to ODD symptoms. These relationships were significantly weaker in girls than boys, likely because ODD is less prevalent in girls.

The relationship between ODD and self-regulation was supported by data on a sample of two birth cohorts from Norway (N= 1042 youth ages 4 to 14 years).[3] ODD increased from age 4 to 6 and from age 8 to 10 and then started to wane as children entered adolescence. Increased **emotion regulation** predicted a reduced number of ODD symptoms across early development. This prediction was strong for both the angry/ irritable and the argumentative/defiant dimensions of ODD.

In research on neuropsychological mechanisms of **social difficulties** in oppositional defiant disorder, children with ODD (N=39, mean age = 10) completed neuropsychological tests measuring social cognition including theory of mind and face emotion recognition.[4] Two thirds of the children with ODD showed clear difficulties with the theory of mind and executive function tasks (cognitive flexibility, inhibition, and working memory).

Oppositional defiant disorder has also been related to **internet gaming disorder** and suicidal ideation based on the Suicidal Ideation Scale, the Internet Gaming Disorder Scale and the Oppositional Defiance Scale (N=273 adolescents, mean age =15).[5] Oppositional defiant disorder and internet gaming disorder were positively related to suicidal ideation. Self-control was found to mediate this relationship, and this relationship was stronger in females compared to males.

ODD has also been associated with **physical health problems**. In a study entitled "Examining the covariation between symptoms of physical health and ODD across adolescence to young adulthood", the participants (N= 662) from the Victoria Health Youth Survey were assessed for 10 years from the ages 12 to 30.[6] Individuals with more ODD symptoms reported more physical health symptoms.

Only one study could be found in this literature that addressed potential underlying biological mechanisms for ODD. In this research, interpersonal neural synchrony between mothers and children was measured as the temporal similarity of brain signals during their interactions. In this study (N= 35 children with ODD and 37 healthy children), neural activity was explored in the frontal, parietal and temporal regions. The results showed **lower neural synchrony** in the ODD group compared to the control group and the synchrony was negatively associated with ODD symptoms.

Interventions

In the only intervention that could be found for ODD in this current literature, **parent management training** was provided for Australian parents of children between the ages 7 and 14 (N= 145).[7] Assessments were conducted at baseline, post-intervention and at a six-month follow-up. High pretreatment levels of conduct problems, lagging skills and inconsistent discipline predicted poor treatment outcomes.

Intermittent explosive disorder

Intermittent explosive disorder (IED) has been associated with several risk factors/predictors that are similar to the risk factors/ predictors of ODD and similar to each other (see Table 2). These include an inability to control aggressive impulses, self-control problems, emotional dysregulation and suicide attempts.

Table 2 Intermittent explosive disorder risks and potential underlying biological mechanisms (and first authors)

Risks	First authors
Inability to control aggressive impulses	Liu
Self-control problems	Rad
Emotional dysregulation	Ciesinski
Suicide attempts	Timmins
Potential mechanisms	
Amygdala and orbitofrontal cortex	Paliakkara

Risk factors/predictors

One of the most common risk factors/predictors of IED is repeated **inability to control aggressive impulses**. In a paper entitled "Angry without borders: global prevalence and factors of intermittent explosive disorder: a systematic review and meta-analysis", 29 studies were analyzed (N= 182,112 participants across 17 countries).[8]

The prevalence of IED varied significantly across subgroups with higher rates in clinical samples (11%), as might be expected. Other risk factors included male gender, younger age, trauma exposure and psychiatric comorbidities (mood, anxiety and substance use disorder).

Self-control problems have been illustrated by research that compared an IED group (N = 54), a psychiatric control group (N= 59), and a healthy control group (N= 62).[9] The participants completed computerized neurocognitive tasks including the color-word Stroop task, the emotional Stroop task, the go-no go task and the stop signal task. The IED group had poorer performance than the psychiatric and healthy control groups on cognitive inhibition, response inhibition and increased emotional interference. The authors concluded that IED control problems were related to deficient cognitive, emotional and behavioral inhibitions.

A latent class analysis of cognitive-affective heterogeneity of IED individuals revealed four different groups based on the level of **emotional dysregulation**.[10] The two moderate emotion-dysregulated groups endorsed **callous-unemotional traits** and low empathy. They also differed on severity of outcomes (e.g. aggression, suicide attempts, antisocial behavior, global function and comorbidities).

Self-control problems have also been related to **suicide attempts**. In a study entitled "Self-regulation in adults with intermittent explosive disorder and a history of suicide attempts", adult community participants (N = 735) completed diagnostic assessments and self-report measures on suicidal behaviors, aggression, trait anger, impulsivity and affect regulation.[11] Participants with IED plus suicide attempts had the highest scores on all outcome variables. Those with IED had higher aggression and anger scores than those with suicide attempts but did not differ on general impulsivity and affect regulation.

Not unlike the other impulse control disorders, intermittent explosive disorder has been notably comorbid with other conditions. In a study on psychiatric, neurological and somatic comorbidities, 96% of the individuals with IED had another psychiatric diagnosis (N= 30,357 individuals with IED and 30,357 demographically matched controls).[12] The comorbidities included neurodegenerative diseases and neurological conditions as well as somatic diseases including obesity, hypertension and hyperlipidemia. Methodological limitations of this study related to the reliance on medical records and the low diagnostic rates of IED, questioning the generalizability of these findings and underscoring the need for further research.

Potential underlying biological mechanisms

Only one paper could be found on potential underlying biological mechanisms. In this systematic review of 7 databases on the etiology and neurobiology of intermittent explosive disorder, the roles of the **amygdala and orbitofrontal cortex** in emotion regulation and impulse control were highlighted.[13] The research also showed that interventions were targeting serotonergic signaling.

Conduct disorders

Conduct disorders (CD) are among the most frequent psychiatric disorders in children and adolescents. The worldwide prevalence has been estimated as 2 to 4%.[14] The prevalence of diagnosed CD has differed 31- fold between countries with .1% in Denmark, .3% in Norway, 1.1% in the USA and 3.1% in Germany with a male / female ratio of 2- 2.5. The rate of psychiatric comorbidity has ranged from 70 to 86% with attention deficit hyperactivity disorder being the most common. Between 4% of youth with a CD diagnosis in Germany and 12% in the US were prescribed antipsychotic medications, and 1.2% in Norway and 12.5% in Germany underwent psychiatric hospitalizations.

Risk factors/predictors

Several risk factors/predictors have been identified for conduct disorders in this current literature (see table 3). They include callous-unemotional traits, social anxiety, approach-avoidance behavior, grandiose-manipulative traits and daring-impulsive traits.

Callous-unemotional traits, social anxiety and approach-avoidance behavior have been reported for conduct disorder.[15] In this study on 40 male adolescent inpatients with conduct disorder and 30 typically developing controls (mean age= 13), a virtual reality task was given in which the participants approached virtual age mates with different facial expressions. While the control group showed significantly greater interpersonal distances to angry classmates than happy classmates, the conduct disorder patients did not differ on their approach to happy and angry classmates. This behavior was associated with high scores on social anxiety and callous-unemotional traits. The authors suggested that this lack of adjustment to social cues might contribute to the problems those with conduct disorder have with their peers.

Grandiose-manipulative and daring-impulsive traits have been identified in individuals with conduct disorder in addition to the callous-unemotional traits. In a sample of detained boys with a conduct disorder diagnosis (N = 286, ages 16 to 17 years), all three specifiers were correlated with CD symptom scores.[16] In this sample, the grandiose-manipulative and daring-impulsive traits added significantly to the callous-unemotional traits in classifying the detained boys with conduct disorder. The authors referred to this as the multi-specifier model for conduct disorder.

Potential underlying biological mechanisms

A few potential underlying biological mechanisms have been the focus of research in this current literature on conduct disorders (see Table 3). They include cortical thinning, lower surface areas and smaller volumes in several regions of the brain.

In a sample of boys with conduct disorder (N=50 with impulsive aggression, 26 with premeditated aggression and 69 healthy controls) structural MRI scans were conducted.[17] The impulsive group showed significant **cortical thinning** in the right frontal cortex while the premeditated group demonstrated deficits in the bilateral superior parietal cortex. Both types of aggression were negatively correlated with volume in the left amygdala.

Table 3 Conduct disorder risks and potential underlying biological mechanisms and interventions (and first authors)

Risks	First authors
Callous-unemotional traits	Derks
Grandiose-manipulative and daring-impulsive traits	Colins
Potential mechanisms	
Cortical thinning	Jiang
Lower surface area brain regions	Koerner, Gao
Smaller volume brain regions	Gao
Fewer amygdala responses to facial expressions	DeBrito
Interventions	
Multisystemic and functional family therapy	Lee
Treatment engagement	Delamillieure
Completers/dropouts	Hauschild

Brain structure has also been explored in a study comparing CD (N= 291) and healthy controls (N= 379) who varied in age between 9 and 18 years (mean age = 14).[18] Structural MRI scans suggested **lower surface area across frontal, temporal and parietal regions** in those with CD as well as lower total surface area. No differences were noted in brain maturation.

Lower surface area was also noted in those with CD in an analysis of structural MRI data from 15 international cohorts (N=1185 with CD and 1253 healthy controls, mean age = 14 years).[19] The conduct disorder group had **lower surface area in as many as 26 cortical regions** and lower total surface area. Cortical thickness differed in the caudal anterior cingulate cortex and the superior temporal sulcus. The conduct disorder group also had **smaller volumes in the amygdala, nucleus accumbens, thalamus and hippocampus.** Those with numerous callous-unemotional traits showed more widespread differences compared to those with a few callous-unemotional traits. These findings provide additional evidence that brain alterations might contribute to conduct disorder.

Differences in brain responses during face processing have also been noted in conduct disorder as a function of callous- unemotional traits.[20] In a sample of CD (N= 161) and typically developing youth (N = 241) age 9 to 16 years, those with conduct disorder showed **fewer amygdala responses to facial expressions** (angry, fearful and neutral faces) which were related to callous-unemotional traits.

Interventions

Three intervention studies were found in this current literature on conduct disorder. Only one of them explored the effects of therapy. The others assessed the differences between those who were engaged and those not engaged in therapy and the differences between completers and dropouts.

Multisystemic and functional family therapy effects were explored in a scoping review of the literature on 25 randomized controlled trials on family-based interventions for youth (10-17 years-old).[21] Approximately one-third of these trials were effective which was not surprising given that these youth had severe and persistent conduct problems.

Treatment engagement and its relationships to callous-unemotional and grandiose-manipulative traits were explored in a sample of adolescents (N=286 boys and 69 girls).[22] Consistent with the previously discussed data on callous-unemotional traits, boys with

those traits were less engaged in treatment. Surprisingly, girls with grandiose-manipulative traits were more engaged in treatment.

Intervention completers were also compared to **dropouts** in a small sample of adolescents (N=32 including 21 completers and 11 dropouts).[23] Only lack of intimacy rather than aggressiveness played a crucial role in the adolescents who dropped out.

Disruptive mood dysregulation disorder

Disruptive mood dysregulation disorder (DMDD) is characterized by severe irritability and outbursts primarily in children and adolescents (see Table 4). Although this is a relatively new diagnosis, a group of researchers and clinicians (N=23) have reached consensus on the diagnosis of DMDD.[24]

Table 4 Disruptive mood dysregulation risks, potential underlying biological mechanisms and interventions (and first authors)

Risks	First authors
Less overall competence and adaptive functioning	Coldevin
Less social skill-related temperament trait	Aburso
Greater levels of parenting stress	Coldevin
Potential mechanisms	
Low heartrate variability	Leal
Interventions	
Pharmacological	Chang
Cognitive behavioral	Soleimani-Rad

Risk factors/predictors

A few risk factors/predictors have been identified for disruptive mood dysregulation disorder. They include less overall competence and adaptive functioning, less social skill-related temperament and higher levels of parenting stress.

In a sample of Norwegian children referred to a mental health clinic (N=218, 6 to 11- years-old), as many as 24% met the diagnostic criteria for DMDD.[25] Those children were more likely to be male, living in poverty, having multiple mental health diagnoses and lower functioning based on the Children's Global Assessment Scale. The parents and teachers of the children reported **lower overall competence and adaptive functioning** of those children. This select sample of children from a mental health clinic is, of course, not representative of children in the community and this sample experienced many problems that confounded the effects of their DMDD.

In a systematic review and meta- analysis on the prevalence of DMDD in community- based and clinical populations, 14 studies were included.[26] The prevalence in community-based samples was 3% while the prevalence in clinical populations was significantly higher at 22%, as might be expected. Anxiety, depressive disorders and ADHD were the most frequent comorbid conditions that occurred with DMDD. Although these conditions have frequently been comorbid, comparisons have rarely been made between the conditions. In one exception, a comparison between DMDD and MDD (major depression disorder) in children and adolescents (N = 233) showed that the DMDD group had significantly **lower social skill-related temperament traits** including empathy, compassion and helpfulness.[27] They also exhibited higher levels of impulsivity, disorderliness and self-acceptance.

In a recent study on parental stress and attachment styles of parents of children with disruptive mood dysregulation disorder (N = 208 parents of children 6 to 12-years-old who had been referred to mental health clinics), the parents of children with DMDD experienced **higher levels of parenting stress** related to the children's problems.[25] They also reported insecure adult attachment styles.

Potential underlying biological mechanisms

Low heart rate variability is the only potential underlying biological mechanism that has been noted in this literature on DMDD. In a study on children's attachment styles, a group of children with DMDD was compared to a control group on their attachment style and their heart rate variability (vagal tone) after a frustration task (N = 30).[28] The group of children with DMDD had a greater prevalence of avoidant attachment styles and lower vagal tone during the frustration task.

Interventions

Interventions have included both **pharmacological and behavioral therapies**. In a recent systematic review and meta-analysis, six pharmacological and five non- pharmacological studies were reviewed.[29] **Decreased irritability** was noted following the drug interventions. Stimulants combined with other drugs and behavioral therapy significantly reduced irritability. The authors suggested that large scale randomized controlled trials were needed, especially those combining pharmacological and non-pharmacological interventions.

While a low level of consensus on pharmacological interventions has been reported for a group of clinicians and researchers (N=23), the use of **behavior-focused therapies and family therapies** received a high degree of consensus.[24] **Cognitive behavioral therapy (CBT)** has been supported by at least one study in this literature. In this randomized controlled study, a group of children was compared to a waitlist control group (N= 53).[30] The children in the CBT group received 15 weekly sessions. At a three-month follow-up assessment, children in the CBT group showed less aggressive behavior, anger outbursts and irritability which was largely maintained through the follow-up period.

Social (pragmatic) communication disorder

Social communication disorder has been difficult to differentiate from autism spectrum disorder and developmental language disorders.[31] The basic problem has been the lack of screening measures for differentially diagnosing social (pragmatic) communication disorder. A recent factor analysis of the Social Communication Disorder Screener, which is a 14- item parent report measure based on the DSM-5 diagnostic criteria (N = 500 parents of children 5 to 18-years-old), 3 subscales were noted to reflect overall **verbal and nonverbal communication limitations**.[32]

In a recent comparison between children with autism spectrum disorder and social communication disorder (N = 232 with ASD and 90 with SCD), children with social communication disorder had better adaptive functioning across all areas of the Vineland Adaptive Behavior Scale.[31] The most significant differences were observed on communication and social skills.

Methodological limitations of this literature

Several methodological limitations can be noted for this current literature on emotional behavioral disorders. They include unequal sample sizes for comparisons between those with the disorder and healthy controls, the conditions having similar diagnostic criteria and comorbidities, the lack of studies on comparisons between the disorders, and the rare biological mechanism and intervention studies.

Several of the disorder studies were comprised of clinical samples, and the comparison healthy control groups were often a different

sample size. Several of the disorders have similar diagnostic criteria including self-regulation difficulties, callous-unemotional traits and suicidality. Although many of the papers were based on systematic reviews and meta- analyses, no studies could be found on comparisons between the disorders.

Only a few potential underlying biological mechanism studies appeared in this literature except for CD, and intervention studies were rare except for CD and DMDD. Despite these methodological limitations, this literature can be informative for future mechanism and intervention studies. The severity of these disorders and the infrequent underlying mechanism and intervention studies highlight the importance of continuing research on emotional behavioral disorders.[33–44]

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Conflicts of interest

The author declares that there are no conflicts of interest.

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