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# Emotional Dysregulation and Cognitive Disengagement Syndrome: Exploring Their Relationship Through the Lens of Twin Studies

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**Abstract:** Cognitive Disengagement Syndrome (CDS) is a clinical construct characterized by symptoms such as excessive daydreaming, mental confusion, slowed behavior, and reduced cognitive and motor activity. Increasing evidence suggests a potential overlap between CDS and Emotional Dysregulation (ED), a transdiagnostic construct associated with difficulties in regulating emotional responses. This narrative review synthesizes current empirical findings and theoretical perspectives on the co-occurrence of CDS and ED, with a particular focus on insights provided by behavioral genetics—especially twin studies. We describe the core principles and models used in twin research and evaluate how they have been applied to disentangle genetic and environmental contributions to these phenotypes and their overlap. While some studies support a shared etiology between CDS and ED, particularly through non-shared environmental influences, research in this area remains limited and conceptually fragmented. The review identifies critical knowledge gaps, including the lack of longitudinal studies, inconsistent definitions of ED, and limited exploration of developmental trajectories. We argue that future twin studies are essential for clarifying these unresolved issues. Nonetheless, limitations include the scarcity of twin-based studies directly examining the CDS–ED association and methodological inconsistencies across the existing literature.

**Keywords:** cognitive disengagement syndrome; emotional dysregulation; twin studies; narrative review



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## 1. Introduction

Previously referred to as Sluggish Cognitive Tempo, Cognitive Disengagement Syndrome (CDS) is characterized by a constellation of symptoms affecting both cognitive and motor functioning [1]. More specifically, CDS encompasses a range of developmentally atypical behaviors, including slowed cognitive processing, frequent daydreaming and mind-wandering, mental fogginess or confusion, intrusive or task-irrelevant thoughts, difficulty initiating and maintaining effort, low motivation, drowsiness, and pronounced hypoactivity [2–4]. These symptoms can substantially interfere with a child's daily activities and overall functioning across various domains [1,4].

Interest in studying this condition among researchers and clinicians emerged in the late 1980s, following factor analytic studies that began differentiating CDS from the inattentive subtype of Attention-Deficit/Hyperactivity Disorder (ADHD) [1]. Initially, CDS was regarded as synonymous with the inattentive presentation of ADHD [5]. However, later research has increasingly emphasized the distinctiveness of these two constructs, and CDS is now recognized as a separate nosological entity, although it has not yet been included in current diagnostic classification systems [6]. In recent years, an increasing body of research has examined the association between Cognitive Disengagement Syndrome (CDS) and Emotional Dysregulation (ED). ED is characterized by heightened emotional reactivity, including pronounced irritability, frequent outbursts, mood instability, low frustration tolerance, and increased emotional sensitivity [7]. The growing interest in investigating these two phenotypes together arises from their transdiagnostic nature and their shared associations with both internalizing and externalizing disorders [4,8]. However, despite promising findings, research on their co-occurrence remains in its early stages, and current evidence raises more questions than it answers, particularly regarding the etiological mechanisms underlying this comorbidity.

Given these premises, it is crucial to emphasize the importance of addressing this issue through the framework of Quantitative Genetics, which provides a powerful methodological approach for disentangling the genetic and environmental factors underlying psychological disorders [9]. Quantitative genetic studies, particularly twin studies, are widely utilized in developmental psychopathology research and have substantially advanced our understanding of the etiological foundations of comorbidities between phenotypes [9].

In light of the above, this review aims to explore the potential value of twin studies in elucidating the genetic and environmental foundations of the co-occurrence of ED and CDS manifestations. Following an introductory overview of research designs in quantitative behavioral genetics, the review will examine the core characteristics of both CDS and ED, along with the existing literature on their co-occurrence. The discussion will then highlight the importance of employing twin study designs to deepen our understanding of the comorbidity between these two phenomena.

## 2. Behavioral Genetics

Behavioral Genetics offers a crucial framework for understanding the co-occurrence of Cognitive Disengagement Syndrome (CDS) and Emotional Dysregulation (ED), by disentangling the genetic and environmental contributions to these overlapping yet distinct psychopathological dimensions. Emerging in the 1960s, the field was originally designed to investigate the interplay of heredity and environment in shaping behavior, temperament, and psychopathology [9,10]. It encompasses several subfields, including Qualitative Genetics, Epigenetics, and particularly Quantitative Genetics, which has been instrumental in clarifying the etiological architecture of co-occurring traits such as CDS and ED in developmental populations.

A central tenet in Behavioral Genetics is that genes and environment do not operate in isolation, but rather interact dynamically to influence individual variability, including susceptibility to emotional and cognitive dysregulation [11]. This is especially relevant for constructs like CDS and ED, where both heritable predispositions and individual-specific environmental experiences (e.g., adverse peer relations and family stress) are likely to jointly contribute to their manifestation and interrelation [12]. Research has also highlighted that commonly assessed environmental variables—such as family socioeconomic status, parenting style, or life events—may themselves be partially shaped by genetic factors, a phenomenon known as genotype-environment correlation (rGE) [13–15]. For instance, children with a genetically driven tendency toward cognitive disengagement may elicit

specific environmental responses (e.g., negative teacher or peer interactions), which could in turn exacerbate emotional regulation difficulties. Understanding such transactional mechanisms is vital to unraveling whether the observed association between CDS and ED reflects shared heritable pathways, common environmental risk factors, or both.

Given these complex interdependencies, quantitative genetics provides rigorous methodologies for parsing the relative influence of each component on observed behaviors and psychological traits including the nuanced behavioral phenotypes of CDS and ED.

### 2.1. Quantitative Genetics

Quantitative genetics allows for the decomposition of phenotypic variance into three core components: additive genetic effects (A), shared environmental influences (C), and unique environmental factors (E) [16]. These parameters are particularly informative for examining whether the overlap between CDS and ED reflects common genetic underpinnings (A), environmental exposures shared among family members (C), or individual-specific experiences (E). For example, evidence of high A and low C components in both CDS and ED would suggest that their association is partially due to shared heritable liability. Two classic research strategies in this field are adoption and twin studies [9]. While adoption designs have offered foundational insights, they are now limited by practical and methodological constraints, such as sample representativeness and reduced availability [16].

#### 2.1.1. Twin Studies

Twin studies, by comparing monozygotic (MZ) and dizygotic (DZ) twins, offer a robust method for studying the genetic and environmental contributions to phenotypic traits and are widely recognized as a valid methodology in quantitative genetics studies [11,17]. MZ share 100% of their genetic material, as they are conceived by the fertilization of a single zygote, which makes them genetically identical. This genetic similarity allows MZ twins to serve as a model for investigating the genetic underpinnings of phenotypic traits, as any phenotypic differences observed between them are primarily attributable to environmental factors [18]. In contrast, DZ twins resulting from the fertilization of two separate eggs share approximately 50% of their genetic material, similar to non-twin siblings [18]. Although DZ twins differ genetically, they share the same intrauterine environment, which makes them an ideal comparison group for evaluating the relative contributions of genetic and environmental factors to phenotypic variance [18]. The comparison between MZ and DZ twins, therefore, enables a more accurate assessment of the genetic influence on a trait while accounting for environmental factors that both twin types may share. These studies are aimed at addressing the percentage of variance on traits explained by A, C, and E in order to allow researchers to understand whether the emergence of a given trait is mostly influenced by genetics, shared environmental factors, and unique environmental components [9]. To do so, twin studies estimate the correlations for a given phenotype between MZs and DZs, assuming that if the emergence of a specific trait is mostly influenced by genetics, correlations between MZs should be higher than those between DZs [19]. However, the validity of twin studies is strictly linked to the meeting of the Equal Environment Assumption (EEA). The EEA fundamentally assumes that MZs and DZs share similar environmental factors relevant to the phenotypic expression of the studied trait [9,16]. If this condition is not met, due to potentially greater shared environmental influences among MZ twins, the validity of comparisons between MZ and DZ twins would be compromised. Consequently, this could lead to an overestimation of genetic influences, as highlighted by Fagnani et al. [20]. Twin studies have historically faced criticism regarding their potential lack of representativeness. Nevertheless, longitudinal research has consistently demonstrated that twins

offer a valid and generalizable framework for investigating various traits of interest within bio-psycho-social disciplines [11].

However, it is worth mentioning that the primary limitation of this research design regards the substantial difficulties associated with participant recruitment. Achieving an adequately powered sample typically necessitates the establishment of comprehensive national twin registries, a process that can span several years of rigorous effort. Furthermore, a significant proportion of twin studies have predominantly employed male-only cohorts, potentially limiting the generalizability of the findings and failing to account for the full spectrum of genetic and environmental influences across genders. Another important limitation to consider when discussing findings from twin studies concerns the impact of assessment instruments on twin data. Specifically, diagnostic questionnaires for children and adolescents can be administered to parents or teachers, or directly to the individuals themselves [21]. Evidence from the twin study literature indicates that third-party reports tend to overestimate both genetic (A) and shared environmental (C) influences, whereas self-reports are more likely to emphasize the role of non-shared environmental (E) factors [21,22]. In particular, studies relying on parent informants consistently report inflated estimates of shared environmental influences, likely due to parents' tendency to perceive their children's experiences within the family context as highly similar [22].

Beyond variance estimates, the choice of informant also affects the symptom scores obtained from diagnostic scales, especially with respect to internalizing and externalizing disorders [21]. Research has shown that parents and teachers tend to assign higher externalizing scores to children or students compared to the self-reports of the individuals themselves, whereas the reverse pattern is observed for internalizing symptoms. This discrepancy is likely attributable to the intrinsic nature of the symptoms: externalizing behaviors are more overt and observable to others, whereas internalizing symptoms are more subtle, subjective, and closely tied to intrapsychic processes [21].

#### 2.1.2. Twin Studies: Statistical Methods and Models

Twin studies primarily employ two statistical approaches, univariate and multivariate models, depending on the specific research objectives. The univariate model is implemented to estimate the relative contributions of genetic and environmental factors to the variance of a single phenotype, whereas the multivariate model is applied when analyzing the interrelationships between two or more phenotypes [20]. In the univariate model, the analysis involves computing and comparing intraclass correlations between MZ and DZ twins. In contrast, multivariate models primarily focus on cross-twin/cross-trait correlations, which assess the relationships between two or more phenotypes observed in each twin within a pair [20]. Structural Equation Models (SEM) are used to assess the influence of genetic and environmental factors on phenotypic variance and covariance [23]. In these models, the phenotypes under investigation are treated as observed variables, while A (additive genetic factors), C (shared environmental factors), and E (non-shared environmental factors) function as latent variables. Three primary models are commonly employed for parameter estimation: the Cholesky model, the Independent Pathway model, and the Common Pathway model. The Cholesky model [Figure A1, Appendix A] proposes that correlations among traits arise from both genetic and environmental influences [18,20]. When analyzing n variables, the Cholesky decomposition incorporates n distinct genetic and environmental factors: the first factor influences all traits, the second factor affects all traits except the first, the third factor impacts all traits except the first two, and so on [18,20]. The Independent Pathway model [Figure A2, Appendix A] posits that common genetic and environmental latent factors directly influence all traits under investigation. These shared factors contribute to the associations among traits, while a set of distinct latent

variables accounts for the unique variability of each individual trait [24]. Finally, the Common Pathway model [Figure A3, Appendix A] assumes that genetic and environmental influences contribute to a single underlying latent variable, which directly impacts the observed traits. Additionally, similar to the Independent Pathway model, this framework includes trait-specific latent factors that account for the unique variance components not shared across traits [24]. In statistical analyses, model selection is based on comparisons using chi-square tests, following the principle of parsimony. Under this principle, models with fewer latent variables are chosen over more complex ones as long as they do not lead to a substantial decline in data fit [24].

## 3. Emotional Regulation, Dysregulation, and Twin Studies

### *3.1. Emotional Regulation and Emotional Dysregulation: Definitions and Comorbidities*

Emotion Regulation (ER) refers to the set of processes through which individuals influence the onset, intensity, duration, and expression of emotional states to adapt to environmental demands and internal goals [25,26]. According to Gross' process model of ER [26,27], regulation strategies are categorized based on when they intervene in the emotion-generative process. Antecedent-focused strategies, such as Cognitive Reappraisal, modify the interpretation of a situation before the emotional response is fully generated. In contrast, response-focused strategies, such as Emotion Suppression, attempt to alter the expression of emotion after it has already been elicited [28,29].

Empirical findings suggest that Cognitive Reappraisal is generally associated with more adaptive outcomes, such as increased positive affect and lower levels of psychopathology, while Emotion Suppression is often linked to heightened physiological stress responses, negative affect, and impaired social functioning. However, a growing body of research emphasizes the need for contextual flexibility in the use of ER strategies [25]. In this view, neither reappraisal nor suppression is inherently adaptive or maladaptive; rather, it is the ability to flexibly shift between strategies in response to changing contexts that determines emotional well-being. Moreover, ER and Executive Functions (EF)—which include processes such as inhibitory control, cognitive flexibility, and working memory—are strongly interrelated. These domains co-develop during childhood and adolescence and support adaptive goal-directed behavior [30]. While some studies suggest that ER scaffolds the development of EF, others propose a bidirectional relationship [31,32]. For example, children with better ER capacities in early development often show more efficient EF later on, whereas those with poor EF are less capable of implementing effective ER strategies. This dynamic interplay may be particularly relevant for understanding emotion–cognition interactions in neurodevelopmental conditions. Difficulties in Emotion Regulation can lead to a condition known as Emotional Dysregulation (ED), which refers to a persistent pattern of emotional experiences that interfere with an individual's ability to maintain goal-directed behavior and interpersonal functioning [7,33]. ED is characterized by several core features: heightened emotional sensitivity, intense and prolonged emotional responses, difficulty returning to baseline after an emotional reaction, and an overall lack of regulatory control. Common manifestations include irritability, mood instability, emotional lability, low frustration tolerance, and aggressive outbursts [7,33]. ED is not merely a transient difficulty but represents a pervasive dysfunction that affects multiple domains of functioning. It has been consistently associated with impaired academic performance, interpersonal difficulties, and a higher risk of psychiatric comorbidities [34–36]. Individuals with ED are more likely to experience peer rejection, family conflict, and difficulties forming and maintaining social relationships. These disruptions often contribute to a negative feedback loop, where social and environmental stressors further compromise regulatory abilities. Importantly, ED is increasingly recognized as a transdiagnostic construct, meaning that

it plays a central role across a wide range of mental health conditions rather than being confined to a specific diagnosis. Historically, ED was viewed primarily in relation to externalizing disorders like ADHD and oppositional defiant disorders, or internalizing disorders such as anxiety and depression. However, mounting evidence indicates that ED contributes broadly to the development, maintenance, and severity of both types of psychopathology [8,37]. For instance, Cai et al. [8] demonstrated that ED levels significantly predicted internalizing (e.g., depression and anxiety) and externalizing (e.g., aggression and rule-breaking) symptomatology in both neurotypical and neurodivergent adolescents. These effects were independent of diagnostic labels, suggesting that ED reflects a general liability toward emotional and behavioral dysregulation. As a result, ED has been included in the Research Domain Criteria (RDoC) framework as a core dimension in understanding psychopathology [38]. The negative impact of ED extends into long-term developmental outcomes. Children and adolescents with high ED are more likely to exhibit academic underachievement, school dropout, and social withdrawal. In adulthood, ED is associated with lower socioeconomic status, reduced occupational functioning, poor physical health, and increased rates of suicidal ideation and self-harm [36].

From a mechanistic perspective, ED may mediate the relationship between environmental stressors—such as trauma, neglect, or peer victimization—and later psychiatric outcomes. Several studies suggest that early adversity disrupts normative emotion regulation development, increasing vulnerability to depression, anxiety, and behavioral problems [34,35]. The chronic activation of stress-response systems in the absence of effective regulation may result in maladaptive patterns that persist over time. Given its central role across psychopathologies and developmental trajectories, ED has become a key target for clinical interventions. Treatments aimed at improving emotional regulation—such as Dialectical Behavior Therapy (DBT), emotion-focused therapy, and mindfulness-based cognitive therapy—have shown efficacy across a range of conditions in both youth and adults. These approaches typically focus on increasing emotional awareness, reducing reactivity, and enhancing flexible use of regulation strategies. Nevertheless, despite its clinical importance, the conceptualization and measurement of ED remain inconsistent across studies. Researchers often focus on isolated components of ED (e.g., emotional impulsivity or mood instability) rather than adopting an integrative framework. This heterogeneity hampers the ability to generalize findings and underscores the need for more refined theoretical models and psychometric tools. In summary, ED represents a complex, multidimensional construct with far-reaching implications for mental health and functioning. It not only interferes with emotion-related processes but also compromises cognitive development, behavioral regulation, and social adaptation. As such, it should be considered a central focus in both developmental research and intervention design.

### *3.2. Twin Studies on Emotional Dysregulation*

To date, only a limited number of studies have employed twin study methodologies to investigate the genetic underpinnings of ED. Consequently, its etiology remains poorly understood from a quantitative genetics' perspective. The absence of a widely accepted definition of ED further contributes to inconsistencies across studies, leading to substantial variability in its conceptualization and phenotypic characterization [7]. Coccaro et al. [39] investigated the genetic and environmental factors contributing to Affect Intensity and Affect Lability. Affect Intensity is defined as the predisposition to experience emotions with high magnitude, while Affect Lability refers to the tendency to undergo rapid and unpredictable emotional shifts. To explore these constructs, the authors administered the Affective Lability Scale (ALS) [40] and the Affect Intensity Measure (AIM) [41] to 182 MZ and 119 DZ male twins from the Vietnam Era Twin Registry. Model-fitting analyses

were subsequently conducted to portion the variance into genetic and environmental components. Their findings highlight that additive genetic factors accounted for 40% of the variance in Affect Intensity and 25% of the variance in the ALS subscale assessing mood shifts between anxiety and depression [39]. Additionally, non-additive genetic influences were identified for ALS subscales measuring shifts from normal mood to depression (29%) and from normal mood to anger (27%) [39]. Evidence for shared environmental influences on affect-related measures was negligible, while non-shared environmental factors accounted for between 52% and 74% of the variance across all the scales [39]. Therefore, these results suggest that individual variability in Affective Lability and Affective Instability is primarily attributable to additive genetics influences and unique personal experiences, rather than shared environmental factors [39].

Further research has focused on exploring the genetic and environmental influences on ER strategies, such as Cognitive Reappraisal and Suppression, in a sample of 448 MZ and 295 DZ twin pairs [42]. Their findings suggest that non-shared environmental factors seem to play a significant role in explaining most of the variance in Cognitive Reappraisal (80%) and Suppression (65%), while additive genetic factors accounted for 20% and 35% of the variance, respectively. Consistent with the findings of Coccaro et al. [39], shared environmental factors were found to have a negligible effect on these phenotypes in their sample [42]. More specifically, Reappraisal was found to be less heritable than Suppression and was primarily influenced by non-shared environmental factors, highlighting the importance of context in shaping functional ER strategies [42]. Moreover, this study examined the covariance between these variables through a Cholesky Decomposition, highlighting an overlap between the non-shared environmental factors influencing Reappraisal and the broader construct of adaptive emotional functioning, as measured by the Brief Risk-Resilience Index for Screening (BRISC) [43].

In examining the role of ED in psychopathology, Mikhail et al. [44] investigated the genetic and environmental factors underlying the mediating role of ER in the comorbidity between Internalizing Disorders and Disordered Eating in a sample of 688 adult female twins. The analyses revealed significant genetic overlap between Internalizing Disorders and ED, with approximately 50% of the genetic variance in internalizing symptoms attributable to ER difficulties, and 17% of the genetic variance in Disordered Eating also linked to these challenges [44]. Notably, while internalizing symptoms shared substantial genetic influences with ER, Disordered Eating showed a distinct genetic contribution that did not overlap with either internalizing symptoms or ER difficulties [44]. These findings suggest that, although genetic factors connect internalizing symptoms and Disordered Eating through ER difficulties, the pathways leading to Disordered Eating involve additional unique genetic influences, highlighting the importance of considering distinct developmental trajectories in the understanding of eating disorders in relation to internalizing psychopathology. This study underscores ER as a critical shared mechanism in these conditions, suggesting potential targets for intervention and treatment strategies [44].

Expanding on the role of genetic and environmental influences in ED, recent research has also investigated its association with neurodevelopmental disorders, which are commonly characterized by ED. In this context, Astenvald et al. [45] examined the association between ADHD and ED using a co-twin control design with 389 twin pairs aged 8–31 years. Their findings revealed a significant association between ADHD and ED, even after adjusting for age, sex, and other mental health conditions. Within-pair analyses showed that twins with ADHD exhibited significantly higher levels of ED compared to their co-twins without ADHD. This association remained significant in DZ twins but was non-significant in the MZ subsample, as evidenced by non-overlapping confidence intervals between the DZ and MZ estimates. The differential pattern observed between DZ and MZ twins

suggests a genetic contribution to the association between ADHD and ED. These results align with prior findings emphasizing the heritability of ED and further suggest that ADHD may represent an additional pathway through which genetic and environmental factors shape ED [45].

However, when interpreting these findings, it is essential to consider potential limitations to their cross-cultural generalizability. For instance, Chen et al. [46] demonstrated that the relative contributions of genetic and environmental factors to psychological traits differ between Chinese and Western populations, with genetic influences on traits being less pronounced among Chinese adolescents compared to their Western counterparts.

## 4. Cognitive Disengagement Syndrome (CDS) and Twin Studies

### *4.1. CDS: Symptoms, Impairments, and Differentiation with ADHD*

Cognitive Disengagement Syndrome (CDS) can be defined as a cluster of symptoms impacting cognitive function and its underlying processes [1]. The core characteristics of CDS include excessive daydreaming, difficulties in sustaining and directing attention, and deficits in information processing, particularly in terms of accuracy and speed [2]. Furthermore, CDS is associated with motor dysfunction, manifesting as hypoactivity, prolonged sedentary behavior, and slowed, reduced, or delayed motor responses [3,4]. These impairments contribute to significant deficits in academic performance, social interactions, and overall adaptive functioning across both youth and adulthood [1,4]. This condition was formerly named Sluggish Cognitive Tempo (SCT), a designation that emphasized cognitive sluggishness, mental fogginess, and slowed cognitive and motor processes [4]. However, this terminology shift from SCT to CDS reflects both conceptual and terminological advancements. The former label primarily underscored cognitive slowness while failing to encompass the broader symptomatology, which includes inattention, excessive daydreaming, hypoactivity, and psychomotor slowing. Additionally, the term "sluggish" carried a stigmatizing connotation, whereas "disengagement" offers a more neutral and clinically precise characterization. The revised nomenclature more accurately captures the multifaceted nature of the disorder and aligns with contemporary research emphasizing its complex clinical presentation [4,47,48].

The impact of CDS extends beyond cognitive and motor deficits, influencing broader functional domains such as academic performance and social interactions [4]. Research has increasingly focused on distinguishing CDS from related conditions, particularly ADHD, by examining their distinct neurocognitive profiles. Empirical findings indicate that ADHD and CDS exhibit notable differences in neurocognitive functioning. Krone et al. [49] identified significant differences in EF deficits among adults with comorbid ADHD and CDS compared to those diagnosed solely with ADHD. Specifically, CDS appears to be primarily associated with deficits in processing speed, which manifest as delayed responses and difficulties in information processing under increased cognitive demands. These impairments hinder the effective implementation of strategic approaches in working memory tasks. The distinct cognitive profile of CDS is characterized by slower response times and specific patterns of cognitive strategy use, differentiating it from ADHD. Consequently, the presence of processing speed deficits suggests that CDS imposes an additional cognitive burden on individuals with both ADHD and CDS compared to those with ADHD alone. Given these findings, EF performance emerges as a critical dimension for distinguishing CDS from ADHD. Krone et al. [49] propose that, as CDS is associated with more pronounced EF deficits than ADHD, EF impairments could serve as a key discriminative factor between the two conditions. A comprehensive distinction between CDS and ADHD is widely acknowledged, not only due to differences in neurocognitive profiles but also in relation to symptom manifestation, potential pharmacological interventions, and longitudinal trajectories. More

specifically, the inattention observed in CDS seems to differ qualitatively from that seen in ADHD. In CDS, the difficulty in maintaining attentional focus is predominantly triggered by internal stimuli. Conversely, in ADHD, inattention is more strongly influenced by external stimuli from the environment [50,51]. This distinction is further supported by evidence suggesting that the presence of CDS as the primary subtype of ADHD is associated with poor adherence to and limited efficacy of methylphenidate treatment. These findings reinforce the hypothesis that a clear differentiation can be made between the two conditions, particularly with regard to treatment outcomes [52]. Moreover, CDS and ADHD exhibit distinct patterns of longitudinal stability. Symptoms of hyperactivity/impulsivity and inattention in ADHD tend to remain stable over time, whereas the symptomatology of CDS generally shows a declining trajectory with development, indicating lower longitudinal stability compared to ADHD [53].

### 4.2. CDS: Comorbidities

This syndrome is frequently found in comorbidity with a range of other conditions, including neurodevelopmental, emotional, and behavioral disorders. CDS has been strongly associated with depression in both children and adults [1,54], with recent studies also highlighting its predicting role in increasing depressive symptoms in youths [55–57]. Moreover, CDS is closely linked to somatic disorders, such as physical complaints and lethargy [56,58–60]. In contrast, its association with externalizing disorders appears to be more limited. While small but significant correlations have been observed between CDS and conditions such as oppositional defiant disorder and conduct disorder, these associations typically become non-significant or even negative when ADHD symptoms are accounted for [1]. Beyond ADHD, CDS frequently co-occurs with other neurodevelopmental disorders, including learning disabilities, intellectual disability, and autism spectrum disorder. Individuals with high CDS levels are more likely to receive a diagnosis of dyslexia and dysgraphia compared to those without elevated CDS [2,61]. Additionally, meta-analyses suggest a small but significant correlation between CDS and lower intellectual ability [1]. CDS symptoms are also frequently observed in children with autism. Studies indicate that 13–16% of children with high CDS symptoms also meet the criteria for an autism diagnosis, while 30–49% of children with autism present with elevated CDS symptoms [62–65]. Furthermore, CDS has been associated with deficits in emotional self-regulation when measured with self-reports from both children and adults [55,66]. However, findings based on parental assessments seem to be less consistent [2,61]. However, it still remains unclear whether CDS is specifically associated with heightened emotional distress, reduced emotional responsiveness, or a more generalized difficulty in self-regulation [4].

### 4.3. Twin Studies on CDS

Up to now, only a few studies have employed twin methodologies to investigate the heritability of CDS, highlighting the relevance of non-shared environmental factors in the development of this syndrome [12,67]. However, none of these studies specifically aimed to focus exclusively on the etiology of CDS. Moruzzi et al. [67] examined the etiological relationships among ADHD dimensions, including Inattentive Problems (INP), Hyperactivity-Impulsivity Problems (HIP), and CDS. These findings indicated moderate to substantial intercorrelations among these dimensions, with shared genetic influences ranging from 65% to 83% and shared environmental contributions between 29% and 44%. Despite this overlap, this study highlighted that CDS seems to represent a distinct construct, as it is predominantly influenced by unique non-shared environmental factors (72%). This evidence appears to be further supported. Specifically, Leopold et al. [68] conducted a longitudinal twin study over a 10-year period to assess the (in)stability of these two

constructs. The findings indicated that although CDS and ADHD exhibit a high and stable correlation over time, ADHD–HYP symptoms tend to decrease, whereas ADHD–IN symptoms demonstrate greater stability. Conversely, CDS symptoms showed an increasing trend across different time points. To date, the only twin study that has assessed the etiology of CDS independently from ADHD is the one by Scaini et al. [12]. This study aimed at investigating the genetic and environmental factors underlying the comorbidity between CDS, Somatic Anxiety, and Generalized Anxiety Disorder. Similarly to Moruzzi et al. [67], this study highlights that CDS seems to be predominantly influenced by unique non-shared environmental factors (67%), while the variance explained by unique genetic factors seems to be moderate (29%).

## 5. The Association Between CDS and ED

A growing body of research has focused on the relationship between ED and CDS. Findings suggest that CDS might be associated with lower ER skills both in children and adults [4]. A study by Flannery et al. [69] has addressed this issue by investigating the mediating role of ED in the association between CDS and social impairment in college students. The findings show that participants with high levels of CDS exhibited increased symptoms of ADHD, depression, and anxiety, as well as greater difficulties in ER and social adjustment compared to those without high CDS [69]. Notably, CDS was strongly associated with social impairment, independent of other psychopathological conditions, although it did not predict general interpersonal functioning. Additionally, CDS was associated with ED, even after controlling for the significant relationship between depression and ED. Moreover, ED was found to mediate the relationship between CDS and social impairment [69]. Further research has specifically examined the network structure of the associations between Maladaptive Daydreaming—a core symptom of CDS—and ED in a sample of young adults [70]. This study highlighted a strong interconnection between the two constructs, indicating that poorer ER skills were generally associated with higher severity of Maladaptive Daydreaming [70].

When discussing findings regarding the association between ED and CDS, the importance of age must be considered. Research suggests that the association between CDS and ED is more pronounced in adults rather than in children [71,72]. For example, Barkley [2] highlighted that difficulties in ER were mostly observed in children with both ADHD and CDS compared to those with ADHD alone, CDS alone, or healthy controls. In contrast, Yılmaz and Bahadır [71] have examined the association between CDS, ED, and Alexithymia in a sample of adults with ADHD and/or CDS. The participants were categorized into four groups based on the severity of CDS symptoms and the risk for ADHD. The findings highlighted that individuals with both high CDS symptoms and high risk of ADHD had significantly higher levels of ED and Alexithymia compared to the other groups [71]. Regarding youth populations, Becker et al. [66] found that self-reported CDS in children aged 8 to 13 was associated with specific emotional characteristics. Specifically, children who reported higher levels of CDS exhibited reduced emotional inhibition and a greater tendency to engage in maladaptive emotional expression. Similarly, a study conducted on children and adolescents aged 6 to 17 years revealed a significant correlation between CDS and ED, even when controlling for inattention (IN) and hyperactivity (HI) symptoms, suggesting a direct association between CDS and emotional difficulties [73]. Expanding on these findings, Cano-Crespo et al. [74] examined the mediating role of ED in the relationship between CDS, ADHD, and Internalizing/Externalizing disorders in a sample of children aged 8 to 13 Their findings suggested that ED mediates the relationship between CDS, ADHD, anxiety and depression, whereas no significant predictive relationship was found between CDS and ER difficulties [74].

## 6. Discussion: The Critical Role of Twin Studies in Examining the Association Between CDS and ED

ED is increasingly recognized as a transdiagnostic factor present across a wide range of psychological conditions [7], including internalizing and externalizing disorders [8], as well as neurodevelopmental disorders such as ADHD [37]. Given the well-documented comorbidity between CDS, internalizing disorders, externalizing disorders, and ADHD [4], it is crucial to explore the relationship between CDS and ED. Understanding this relationship could provide valuable insights into the distinct and shared mechanisms underlying these conditions and inform tailored intervention strategies.

Twin studies offer a particularly valuable approach for disentangling genetic and environmental contributions to this relationship, helping to clarify the role of ED in the co-occurrence of these conditions and its potential as a shared underlying mechanism. However, despite growing interest, the interplay between CDS and ED remains relatively underexplored, particularly in terms of their etiological underpinnings and developmental trajectories. One probable factor contributing to this knowledge gap is the conceptual ambiguity surrounding both CDS and ED [4,75]. Specifically, ED lacks a universally accepted and comprehensive definition, leading researchers to focus on its individual components—such as ER strategies, emotional impulsivity, and emotional lability—rather than addressing its multidimensional nature in a systematic manner [76]. Similarly, there is ongoing debate regarding the nosological status of CDS, with some scholars arguing that it constitutes a distinct clinical entity, while others contend that it represents a phenotypic variation within the broader ADHD spectrum [75].

Emerging theoretical frameworks and empirical findings suggest that certain neurodevelopmental and psychiatric comorbidities may not represent discrete disorders but rather alternative phenotypic expressions of ADHD [75]. This raises critical questions regarding the conceptualization of overlapping clinical phenomena and whether they should be integrated into an expanded ADHD framework. Notably, ER difficulties and CDS are among the most frequently proposed domains within this broader ADHD characterization [75]. Furthermore, empirical evidence indicates a significant association between ED and CDS, underscoring the need for further research into the etiopathogenetic mechanisms underlying their co-occurrence [4].

ED has been increasingly acknowledged as a critical aspect of EF, with deficits in EF commonly observed in individuals with CDS [31,49]. Research suggests that ED may mediate the relationship between CDS and various psychopathological outcomes, particularly Internalizing and Externalizing symptoms [74]. However, understanding how ED operates within this context, especially in relation to developmental trajectories, requires further investigation. Twin studies offer a valuable framework for exploring these mediating mechanisms [44]. By examining the interplay between CDS, ED, and EF through twin designs, gaining a more comprehensive understanding of how ED interacts within the broader spectrum of CDS and its role in the development of psychopathological symptoms would be possible.

Nevertheless, the relationship between CDS and ED throughout development remains poorly understood, as do the developmental trajectories and potential age-related variations in this association. A more comprehensive understanding of the interplay between these two phenotypes is essential, as it would enable further investigation into the etiological basis of the CDS–ED relationship and provide insights into its longitudinal trajectories. This, in turn, could facilitate a more precise characterization of neurodevelopmental and psychiatric conditions and inform targeted intervention strategies.

This would be particularly important from a clinical perspective, given that only a limited number of studies have explored the effectiveness of interventions in reducing

symptoms related to CDS and its associated impairments [4,77]. To date, no intervention has been specifically designed for individuals with CDS [4]. Considering the established association between CDS and ED [2,69–71], treatment protocols for psychiatric disorders involving individuals with high levels of CDS symptoms could benefit from incorporating techniques aimed at enhancing emotional awareness and regulation, such as Dialectical Behavior Therapy (DBT) [78] techniques.

Moreover, the few available twin studies suggest that while both CDS and ED have strong genetic underpinnings, they are primarily influenced by non-shared environmental factors [12,39,42,44,45]. Therefore, it is plausible that psychosocial factors (e.g., peer rejection, adverse school experiences) play a significant role as risk factors for psychological distress and could represent key targets for clinical intervention.

Future research should focus on clarifying the causal mechanisms underlying the relationship between CDS and Emotional Dysregulation (ED), particularly in developmental terms. Becker et al. [4] highlighted the presence of Emotional Dysregulation profiles in youth with CDS, suggesting it may represent a core clinical feature of the syndrome. However, as ED is also frequently observed in individuals with ADHD, future studies should account for the potential confounding effects of CDS–ADHD comorbidity to determine the specificity of this association. Given the current conceptual ambiguity and the early stage of the literature, further efforts are needed to standardize definitions and measurements to enhance reliability across twin and genetically informed studies. In addition, it will be crucial to explore how early interventions may influence developmental trajectories, especially when considering the role of non-shared environmental factors in shaping treatment outcomes. Advancing this line of research will not only refine our understanding of CDS but also support the development of targeted clinical strategies grounded in more precise etiological models.

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## Abbreviations

The following abbreviations are used in this manuscript:

| | |
|---|---|
| ADHD | Attention-Deficit/Hyperactivity Disorder |
| CDS | Cognitive Disengagement Syndrome |
| ED | Emotional Dysregulation |
| rGE | Genotype-Environment correlation |
| A | Additive genetic effects |
| C | Shared environmental effects |
| E | Unique environmental effects |
| MZ | Monozygotic |
| DZ | Dizygotic |

| | |
|---|---|
| EEA | Equal Environment Assumption |
| SEM | Structural Equation Models |
| ER | Emotion Regulation |
| EF | Executive Functions |
| RDoC | Research Domain Criteria |
| ALS | Affective Lability Scale |
| AIM | Affect Intensity Measure |
| BRISC | Brief Risk-Resilience Index for Screening |
| SCT | Sluggish Cognitive Tempo |
| INP | Inattentive Problems |
| HIP | Hyperactivity-Impulsivity Problems |
| IN | Inattention |
| HI | Hyperactivity |
| DBT | Dialectical Behavior Therapy |

## Appendix A

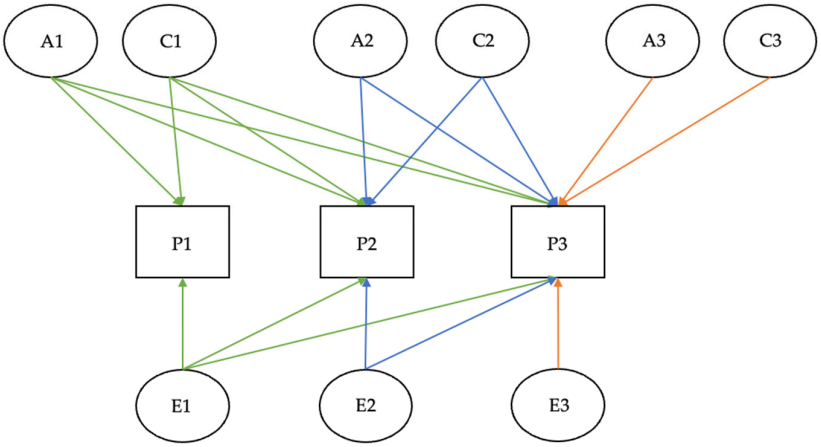


**Figure A1.** Multivariate Cholesky model. Notes. Observed variables are depicted in squares and latent variables in circles. A: additive genetic factors; C: shared environmental factors; E: unique environmental factors. P: Phenotype.

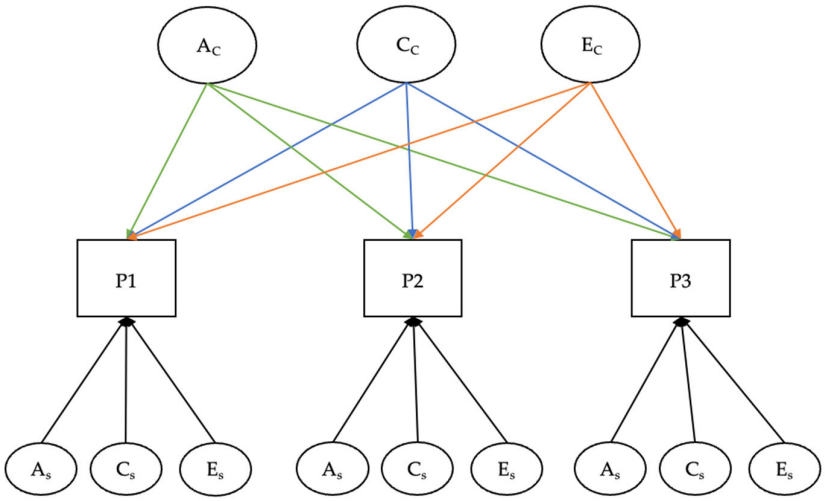


**Figure A2.** Independent Pathway model. Notes. Observed variables are depicted in squares and latent variables in circles. Ac: Additive genetic factors common to the phenotypes; Cc: shared environmental factors common to the phenotypes; Ec: unique environmental factors common to the phenotypes. A: additive genetic factors specific to the phenotypes; C: shared environmental factors specific to the phenotypes; E: unique environmental factors specific to the phenotypes.

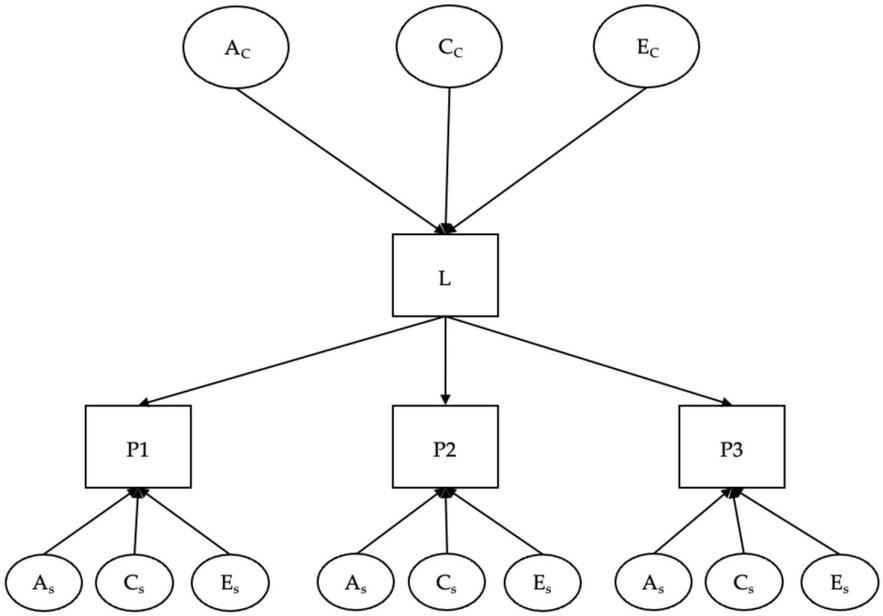


**Figure A3.** Common Pathway model. Notes. Observed variables are depicted in squares and latent variables in circles. Ac: Additive genetic factors common to the phenotypes; Cc: shared environmental factors common to the phenotypes; Ec: unique environmental factors common to the phenotypes; A: additive genetic factors specific to the phenotypes; C: shared environmental factors specific to the phenotypes; E: unique environmental factors specific to the phenotypes; L: common latent susceptibility factor.

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