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Review

Conceptualisation Paradigms for Childhood Autism and ADHD Co-occurrence: A Brief Review and Clinical Implications

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ABSTRACT

Objectives: 'Neurodevelopmental disorders' (NDD) and their related emotional, behavioural, and intellectual disorders (NDEBIDs) include ASD, ADHD and tic disorders. Co-occurrence of NDEBID with each other and with other mental health disorders is the norm. We aimed to review recent research up to March 2024, reporting on the comorbidity of discrete NDEBIDs in general and focus more specifically on the co-occurrence of ADHD and ASD.

Methods: This is a narrative review of recent research up to March 2024, reporting on the comorbidity of discrete NDEBIDs in general and focus more specifically on the co-occurrence of ADHD and ASD.

Results: Four alternative hypotheses are reviewed to conceptualise how co-occurring NDDs are described. These are concepts of Neuroconstructivism, 'Neurodevelopmental Disability', 'Overarching Spectrum of ASD-ADHD disorder' and 'Severe ASD Spectrum'.

Neuroconstructivism argues that the brain gradually acquires specialised functions over its developmental period. The term 'Neurodevelopmental Disability' has been proposed as an alternative generic term for all NDEBIDs instead of separate discrete diagnoses.

An 'overarching Spectrum of ASD-ADHD disorder' has been proposed for children and young people (CYP) presenting with range of symptoms across both diagnostic categories. Other researchers have conceptualised the idea of co-occurring ASD and ADHD as a form of 'severe Autism' with a unique attentional trait and associated functional impairments, rather than two separate diagnoses.

Conclusion: The clinical and therapeutic implications for the ADHD-ASD co-occurrence are discussed. A unified description of NDEBID would buttress their common aetiologies. This would also encourage practitioners not to restrict individuals into constrained diagnostic boxes but to consider the full range of their difficulties, even if they do not meet the seemingly arbitrary threshold levels for diagnosis of the individual conditions.

Further research is needed to help analyse important peculiar neuropsychological features among individuals with the co-occurring ASD/ADHD features.

Keywords: Neurodevelopmental disorders, emotional, behaviour, intellectual disorders, comorbidities, co-occurrence, Autism, ADHD, Neuroconstructivism, 'Neurodevelopmental Disability', 'Overarching Spectrum of ASD-ADHD disorder', 'Severe ASD Spectrum', paradigm.

INTRODUCTION

Neurodevelopmental disorders (NDD) constitute a group of conditions with impairment of brain development in early life, resulting in functional impairment in cognitive, muscular, language and other developmental domains [1]. Attention-Deficit/ Hyperactivity Disorder (ADHD) and Autism Spectrum Disorder (ASD) are two of the commonest neurodevelopmental disorders in childhood. Other NDD include tic disorders, developmental motor, language and intellectual disabilities [2].

Co-occurrence of NDD and related emotional, behavioural and intellectual disabilities, associated with multi-domain functional impairment is recognised as the norm rather than the exception. Researchers are increasingly drawing attention to the almost ubiquitous occurrence of comorbidity among patients with NDD. This has led to the conceptualisation of ESSENCE (Early Symptomatic Syndromes Eliciting Neurodevelopmental Clinical Examinations) to enhance a more holistic and integrated care approach [3].

Other authors have coined a generic term of Neurodevelopmental, Emotional, Behavioural and Intellectual disorders (NDEBID), to capture the common co-occurrence of a wide range of conditions under the same umbrella [1]. The concept of NDEBID as a unifying spectrum implies that each NDD (such as ADHD, ASD, tics, learning disability etc) is often comorbid with other NDD, other physical and psychiatric problems. For example, a recent meta-analysis of 340 published studies has shown that 87% of patients with Autism experience at least one comorbid psychiatric, physical or neurological conditions and up to 40% experience two or more comorbidities, including ADHD, anxiety, bipolar disorder, depression, Tourette syndrome, developmental coordination and sleep disorders [4,5].

Main Points

- We trace a brief history of ADHD and ASD diagnosis both as individual and comorbid conditions.
- We examined a few conceptual hypotheses about the ASD-ADHD co-occurrences, which may also be applicable to the wider spectrum of other NDEBID conditions.
- We conclude that a more holistic conceptualization of all NDDs by Clinicians would provide additional benefits to the affected CYP and their carers, helping them to receive a more integrated early support interventions tailored to the peculiar needs of each individual, irrespective of their diagnostic labels, or none.

ADHD is the most frequently diagnosed neuro-behavioural disorder among children and young people (CYP), with estimated prevalence between 5% and 12% in high income nations [2]. Its characteristic core symptoms consist of developmentally inappropriate levels of hyperactivity, impulsivity and inattention [2]. ASD is characterised by significant impairment of communication, social interaction and stereotypical behaviours with restricted or exaggerated interests [6]. The prevalence of both ASD and ADHD has been rising steadily over the past two decades[4].

Most NDD present with a wide range of symptoms beyond the boundaries of their categorical diagnostic criteria, which are often arbitrary and modified over time by consensus among researchers and clinicians [7].

All NDD often present with common neurodevelopmental features outside their diagnostic criteria such as communication, language and motor disorders. Neurocognitive executive functional impairments such as impaired working memory and planning are also almost invariably universal components of many NDD, particularly ADHD and ASD. Emotional difficulties including mood lability and irritability used to be considered an integral aspect of ADHD [8] but are no longer required as its defining criteria. These emotional features are also common features of other neuro-behavioural disorders (eg, anxiety, depression, or oppositional defiant disorder).

Comorbidity Concept

The term "comorbidity" is a relatively novel concept introduced in the 1970s by Feinstein [9]. It's a somewhat controversial paradigm and is still contested in scientific research [10,11]. For example, the term 'comorbid' appeared zero times in the Diagnostic and Statistical Manual of Mental Disorders (3rd version) or DSM-III but more than 600 times in DSM-5 [12].

First (2005) argued that psychiatric comorbidity is 'artifactual' and a direct consequence of the design of diagnostic manuals 'to "split" diagnostic entities into numerous specific narrowly defined disorders' [13]. The concept of comorbid diagnoses also present with some practical problems such as unwarranted polypharmacy [14], preventing clinicians from seeing a patient

as whole and unified person, making it more difficult for patients to undergo the process of self-understanding as individuals who need to develop their own unique protective or compensatory strategies to deal with their spectrum of peculiar vulnerabilities [11]. A preferred terminology used in this article is co-occurrence of ASD and ADHD (ASD-ADHD) that have been previously diagnosed separately based on taxonomic classification standards.

Aetiology and Risk Factors of NDEBID Disorders

Most NDEBID conditions have unknown aetiology and rarely present with unique psychopathology. The diagnosis is typically based on clinical assessment of psychological symptoms [11]. They often share common symptoms and presentations that are not mutually exclusive to any one diagnostic category.

In the absence of known exact aetiologies of NDEBID conditions, there is ample evidence for a complex interaction between many genetic and adverse environmental factors that increase the risk of developing any of these disorders [1]. The risk factors include heredity, maternal perinatal physical and psychiatric pathology, parental history of psychiatric disorders, prematurity and foetal exposure to smoking, psychotropic drugs or insecticides (Figure 1). Different combinations of these factors have all been linked to higher risk of different NDD [4,15].

There is very limited knowledge about potentially modifiable risk factors (eg, prenatal and early life environmental enrichment and social influences) that could be exploited to optimise neurodevelopmental outcomes of CYP [7].

History of Classification Models and Category Conceptualisation

The commonest diagnostic manuals for NDEBID and other mental health disorders are the DSM (published by the American Psychiatric Association) and the International Classification of Diseases (ICD) published by the World Health Organisation (WHO). Most NDEBID have no clear aetiologies and their taxonomic classification has been problematic and controversial, especially for Autism and its relationship with ADHD.

Both DSM and ICD have been published as serial editions where the criteria for Autism diagnosis and its subcategories, have undergone a number of alterations. Definitions of the age and type of onset, co-occurring conditions and the severity of its typical symptoms have been modified over the past few decades. For example, DSM-IV and the ICD-10 did not include the term "autistic spectrum disorder" [16].

The history of Autism recognition has been tortuous and interesting. It originated from Leo Kanner's [17] masterpiece report of infantile autism cases (1943) and Hans Asperger's publication (1944) of boys with significant social difficulties, unusual restricted interests, despite age-appropriate verbal skills [18]. This led to the rationality of the diagnostic model first included in DSM-III as the Pervasive Developmental Disorders (PDDs). It also included a 'subthreshold' concept of atypical PDD or PPD not otherwise specified (NOS) [19]. DSM-IV introduced the addition of Asperger's disorder as a different diagnostic category. DSM-5 [12] led to elimination of Autism sub-categories and introduction of a unified category differentiated by multiple dimensions.

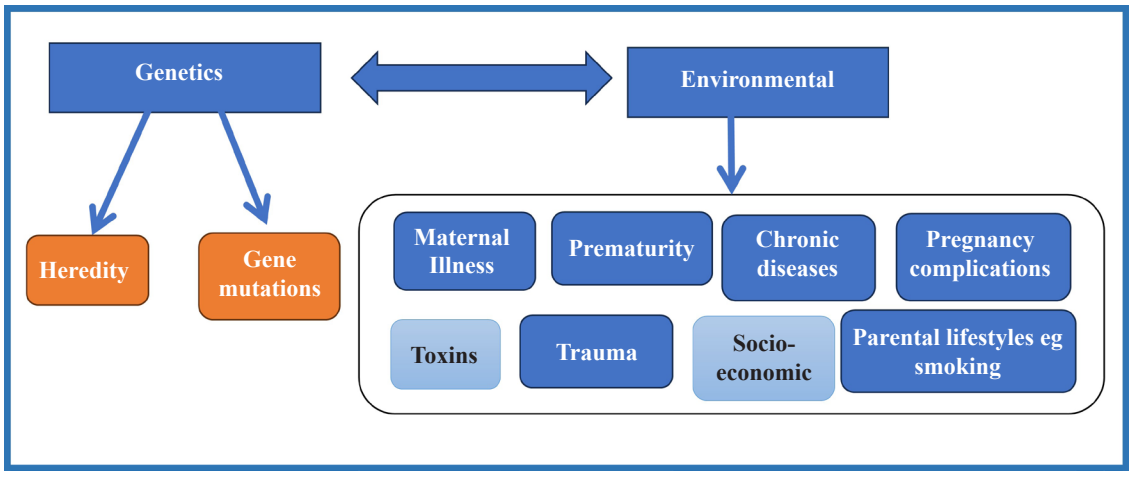

Figure 1. Depicting the multiple aetiological risk factors associated with NDEBID conditions.
Legend: NDEBID: Neurodevelopmental, emotional, behavioural and intellectual disorders.

The ICD 10th edition [20] followed a slightly different pathway and explicitly recognized other related disorders including Asperger syndrome, Rett's disorder and childhood disintegrative disorder [21]. ICD-11 [22] used the umbrella term of Autism spectrum disorder with eight subcategories along the spectrum determined by differences in developmental history (such as regression), intellectual and language skills [19].

The DSM-5 [12] deviated from the tradition of the previous editions which did not allow the diagnosis of both ASD and ADHD, as Autism diagnosis was seen as covering the symptoms of ADHD. Research has shown that ASD and ADHD are characterised by some overlapping symptoms and the DSM-5 has followed the line of evidence suggesting that there are sufficient unique symptoms for each of the conditions to recognise them as distinct diagnostic entities [23].

The history of ADHD and evolution of its clinical classification is equally intriguing. Sir Alexander Crichton's 1798 book "On Attention and its Diseases" appears to be the earliest clinical description of ADHD [24]. Sir George Frederic Still was a British physician who subsequently published series of reports on a number of children characterised by a "defect of moral control" in the 1900s. About three decades later, two physicians Kramer and Pollnow reported about children with "hyperkinetic disease of infancy" in the 1930s including features recognisable as typical symptoms of ADHD such as overactivity, emotional instability and lack of focus [24].

ADHD first appeared in the 1968 edition of DSM, as "Hyperkinetic Reaction of Childhood" and was subsequently changed to "Attention Deficit Disorder" in DSM-III (1980). ADHD terminology first appeared in with DSM-III-R (1987) followed by the DSM-IV edition which described three subtypes of "predominantly inattentive", "predominantly hyperactive-impulsive", and "combined" [24]. It also introduced a new related diagnostic category of "disruptive mood dysregulation disorder" (DMDD) [25].

MATERIALS AND METHODS

This narrative review is mainly informed by a systematic search (from 2000 and up to March 2024) using the PubMed, PsycINFO, Ovid, Database of Abstracts and Reviews, and other online sources, using a combination of keywords including "comorbid", "co-occurrence", "childhood", "adolescent", "behaviour", "disorders", "autism", "ADHD" or "problems". After a careful review of the abstracts, 210 out of 1431 papers were found to be relevant for this review. No abstract was excluded on the basis of publication language if sufficient relevant information was included. The subsequent selection of 210 relevant papers was based on more in-depth review of the titles and abstracts of the retrieved articles, as well as relevant references within the selected papers. This review aims to explore the published literature and provide Clinicians a better understanding of an alternative paradigm of more holistic and integrated care approach in the diagnosis and treatment of CYP with coexisting ASD and ADHD.

Themes identified for this review include prevalence of co-occurring ASD and ADHD, similarities and peculiar vulnerabilities of co-occurring ASD-ADHD, neurobiology studies, four alternative explanations /Hypotheses for co-occurring NDD, clinical and therapeutic implications for co-occurring ASD-ADHD.

We acknowledge there are potential limitations in the selection of relevant papers due to the various combinations of searched keywords. We supplemented the direct search results with relevant referenced articles within the full text of the papers identified from the search engines to ensure a more comprehensive appraisal of the subject.

RESULTS

Prevalence of co-occurring ASD and ADHD:

Since the publication of DSM-5 and permissible separate diagnosis of both ASD and ADHD, the co-occurrence of both conditions has been recognised as quite common. The co-occurrence of ASD-ADHD has long been identified as clinical presentations even before the official changes in DSM-5 was published.

There are also often several other non-medical perspectives and motivations that may drive the demand for one or more diagnostic labels. Many parents and carers have faced the sad reality that choices in classification of a child's condition can have practical implications for social care remuneration in many healthcare systems and might determine if a child can access certain services such as special educational funding.

The co-occurrence of ADHD and ASD leads to experience of additional vulnerability and complexity for the affected children and young people (CYP). It also presents new challenges for

the Clinicians, including timing of appropriate diagnosis, given that the diagnosis of ASD tends precede the diagnosis of ADHD by a period of about 2 years in children with ASD-ADHD co-occurrence [26]. An observational population study of 1496 CYP with co-occurring ASD-ADHD from the USA reported about 80% of them receiving ASD diagnosis either before or concurrent with of ADHD, while those with earlier ADHD diagnosis alone led to delay of ASD co-diagnosis by an average of 3 years [27]. The co-occurrence is known to peak in adolescence compared to early childhood and later adulthood, mostly because of deficits in social adaptation and executive function skills at a time when social demand is greatest during transition to independent adulthood [28].

Several studies reporting ADHD symptoms among children with ASD have published a wide range of co-occurrence rates, with higher prevalence generally reported among clinical samples (53 – 78%) compared to population-based samples (26 to 45.3%), examples of which are listed in table 1 [27, 28]. On the other hand, there are fewer studies reporting the prevalence of ASD in CYP with ADHD diagnosis and the rates are generally lower: (2-19% in community vs 24% in clinical samples). Hollingdale et al (2020) reported a pooled rate of 21% for the co-occurrence of ASD symptoms in young people from both clinical and community ADHD samples from a meta-analysis of 22 studies (total N = 61 985) [31]. This comparative analysis suggests that ASD is the more severe condition and more likely to co-exist with other NDDs.

The need for new guidance for Clinicians working with children, young people and adults who present with co-occurring ADHD and ASD has been recognised and addressed by a consensus specialist publication [32].

Similarities and Peculiar Vulnerabilities of co-occurring ASD-ADHD

There are many psychopathological symptoms that are shared between ASD and ADHD, as well as many other NDD. There. is evidence that executive function deficits based on both frontoparietal and salience/ventral attention networks and weaker connectivities linked to ADHD symptoms are also found in CYP with ASD [33]. A recent systematic review of 58 studies of Executive function measurements in CYP showed similar profiles among individuals with ASD and ADHD across 45 performance metrics from 24 different tasks [34].

There are similar but qualitatively diverse symptoms of hyperactivity/restlessness, attention deficits, emotional dysregulation and social difficulties described in both ADHD and ASD patients (Table 2). Previous research also suggests that co-occurring ADHD symptoms may exacerbate externalizing behaviour problems associated with ASD [35].

Table 1. Published prevalence of co-occurring ADHD and ASD in different samples

Author(s)	Community samples	Clinical samples	Pooled samples	Comments
	ADHD symptoms among ASD Patients			
Lee & Ousley 2006 [67]		78%		N=83, DSM-IV
Simonoff et al 2008 [30]	28.2%			N=112 (10-14 yrs), DSM-IV (40% had one and 70% 2 or more psychiatric comorbidities)
Sinzig et al 2009 [29]		53%		N=83, DSM-IV
Gordon-Lipkin et al 2018 [37]	45.3%			N=3319, DSM-5
Mutluer et al 2022 [68]	26%			Meta-analysis of 39 studies
Micai et al 2023 [5]			45%	Meta-analysis of 44 studies (22% in adults from 13 studies)
Canals et al 2024 [69]	32.8%			N=781, DSM-5
	ASD symptoms among ADHD Patients			
Hanson et al 2013 [70]	2% to 16%			N=1838, DSM-IV-TR
Hollingdale et al 2020 [31]	19%	24%	21%	Meta-analysis of 22 studies (p>0.05)
Canals et al 2024 [69]	9.8%			N=781, DSM-5

Table 2. Qualitative differences between ASD and ADHD Domains

DOMAIN	ADHD	ASD
DOPAMINE PATHWAY	Deficient Dopamine receptor and Dopamine / Noradrenaline transmitter activities in the fronto-limbic pathways.	Overactive Dopaminergic pathways involving the basal ganglia nuclei (basis for stereotypies) and mesolimbic system (interpersonal and perceptual deficits) pathways
HYPERACTIVITY	Hyperactivity/impulsivity symptoms predominant in younger children	Restlessness and Aggression is reported in all age groups
SOCIAL ATTENTION	Inattentiveness becomes increasingly more disabling in older CYP [71].	Social communication and reciprocity impairments, with or without normal language skills [72].

CYP with co-occurring ASD and ADHD have been reported to experience greater burdens of psychiatric, behavioural and developmental comorbidities [30,36,37]. For example, they have increased risk for psychiatric hospitalization, experience greater degrees of multi-domain functional impairment, and require higher levels of mental health services [35]. Some studies have reported that children with both co-occurring ASD and ADHD appear to have higher deficits in recognition of facial affect compared to those diagnosed with either ASD or ADHD alone [38].

Subtle or 'mild' presentations, tendencies to 'camouflage' symptoms by compensatory strategies as well as 'masking' of difficulties by other associated psychiatric conditions, represent unique clinical challenges in both conditions, especially in later adulthood [39].

Neurobiology Studies

Chantiluke et al studied functional Magnetic Resonance (fMRI) images among four groups: ASD, ADHD, comorbid ASD and ADHD, or neither (controls). They compared prefrontal functioning during a temporal discounting task among all the groups. They identified unique features for each of the three non-control groups, as well as some brain changes common to the 3 clinical groups. The co-occurring ASD-ADHD group showed distinctly greater impairments involving the brain areas of lateral and ventromedial prefrontal cortex, ventral striatum, and anterior cingulate cortex [40].

Other Neuroimaging studies have also demonstrated both neuroanatomical features peculiar to ADHD and ASD separately, but also unique neurobiology features for subjects with co-occurring ASD-ADHD, distinct from either ASD or ADHD alone. A recent study of brain imaging using a large, multi-site neuroimaging dataset of over 75,000 individuals, has reported uniquely identifiable brain abnormalities in both autism and ADHD. While ASD individuals demonstrated increased cortical thickness and volume restricted to the superior temporal cortex, ADHD individuals presented with more widespread evidence of increased cortical thickness but reduced cortical volume and surface area in the brain cortex. The co-occurring ASD-ADHD group showed a distinct pattern of global distribution of higher cortical thickness and reduced surface area in specific locations [41].

These physio-pathologic findings suggest that co-existing ASD-ADHD comorbidity does not correspond to a mere combination of both disorders, but may represent a unique phenotypical psychopathology entity,

Four Alternative explanations /Hypotheses for co-occurring NDD

We summarise a few alternative hypotheses that have been published to conceptualise how co-occurring NDD are understood, with particular emphasis on ASD-ADHD (Table 3). These conceptualisation paradigms have significant clinical implications.

Neuroconstructivism

Neuroconstructivism is an hypothesis that depicts specialisation of different brain areas occurring gradually over the course of its development through lived experiences. It views the brain as an inter-related complex system in such a way that any disturbance in a localised area during the developmental period leads to a "cascading effect" which invariably affects the performance of other cognitive functions related to other areas. It provides a basis for the commonly observed co-occurrence of several NDD alongside other related emotional, behavioural and intellectual

disorders. Many NDEBID conditions share several core symptoms of inattention, hyperactivity, impulsivity, emotional dysregulation and executive function deficits, including ASD, ADHD and Learning disability, though their qualitative pattern may vary for each condition [42,43].

This would encourage a more comprehensive view of how NDD are described and managed holistically [44]. This suggests that each patient should be regarded as a unique individual with a complex mixture of psychopathology symptoms and functional impairments that need to be identified and used to determine the range of supportive interventions they require. No artificial categorisation of their symptoms is absolutely necessary and may be misleading. Each child presenting with any neurodevelopmental symptoms requires a comprehensive assessment for both dominant and co-existing conditions within the NDEBID complex [3].

Table 3. A summary of Four Alternative explanations /Hypotheses for co-occurring NDD (with particular reference to ADHD-ASD co-occurrence)

	Descriptions	**Clinical Implication**
Neuroconstructivism	The brain develops as an interacting system where disturbance in one local area in the early stages of development can have a cascading effect on a range of other cognitive domains. Presence of any NDD is an indication that other developmental functioning are likely to be affected.	Many NDEBID conditions share many similar symptoms with varying qualitative patterns. Each child presenting with any neurodevelopmental symptoms requires a comprehensive assessment for both dominant and co-existing conditions [3]
Neurodevelopmental Disability	An alternative generic term to be used for CYP presenting with any features of NDD. "Disability" implies significant impact on daily functioning, and not an abnormality. This obviates the need for separate diagnoses based on seemingly arbitrary symptom cut-off points.	It encourages early identification of specific difficulties and unique strengths of each child described in more detail. Offers a more holistic care approach with early interventions tailored to the peculiar needs of each CYP
Overarching Spectrum of ASD-ADHD disorder	Both disorders have similar but qualitatively diverse underlying neuropsychological "deficits" (Table 4). Common genetic and environmental causal pathways are implied. Both may be conceptualised as extreme manifestations of one spectrum disorder within a continuum distribution.	Clinical presentation of the spectrum range from ADHD alone to co-occurring ASD-ADHD symptoms and severe ASD on the extreme end. All affected CYP are assessed and managed within the same integrated multidisciplinary team and avoid the current fragmented services, often split between child health and mental health teams.
Severe ASD Spectrum with peculiar attentional deficits (mimicking ADHD)	Co-occurring ASD-ADHD may be seen as a form of severe ASD and not two separate diagnoses, causing disabling functional impairments. The unique core characteristic of this entity is a peculiar attentional trait, not equivalent to a separate ADHD, Agitation and restlessness of ASD could be due to joint attention impairment. Dopaminergic hyperactivity along the nigrostriatal and mesolimbic pathways may better explain the basis of restlessness in ASD, rather than deficient dopamine and norepinephrine neurotransmitters in the prefrontal lobe responsible for ADHD symptoms.	CYP with either suspected ASD and or ADHD require a multidisciplinary and multifaceted approach. Stimulants tend to be less well-tolerated and less effective among those with intellectual disability or ASD, and they are more likely to be prescribed antipsychotics and antidepressants. Additional psychological and behavioural treatments are often required, preferably in combination with pharmacological therapy to control ADHD symptoms

Neurodevelopmental Disability

Dorothy Bishop proposed the term 'Neurodevelopmental Disability' as an optional generic term used for identification and description of CYP presenting with any features of NDD, instead of separate diagnoses.

Using the term 'Disability', rather than 'Disorder', would better describe the substantial functional impairment in multiple domains of daily living, frequently experienced by the affected individuals, without implying abnormality.

This would encourage practitioners not to restrict individuals into constrained diagnostic boxes but taking into account the full range of difficulties the individual is experiencing regardless of whether they meet the seemingly arbitrary cut-off points for diagnosis required for separate conditions. It would also enhance early recognition of specific difficulties as well as unique strengths of each child, as the basis for informing appropriate types and levels of supportive interventions tailored to each CYP [45]. It is conceivable that this would lead to a more holistic and integrated care approach by a range of multidisciplinary professionals placing the affected CYP in the centre of every management offer.

Overarching Spectrum of ASD-ADHD Disorder

ASD and ADHD are neuropathological disorders presenting with similar but qualitatively diverse underlying neuropsychological "deficits" (Table 4). The reported high rates of co-occurring ADHD and ASD symptoms have led many researchers to suggest common causal pathways and possibly a single neuropathology entity [46]. Both conditions have witnessed a significant concurrent increase in prevalence in the past 20 years. Both ASD and ADHD present with similar impairments in the brain networks that determine executive functioning of the affected individuals [47].

In terms of aetiological factors, there is evidence of a genetic overlap between clinical ASD and ADHD based on several family, twin, and molecular genetic studies [48,49]. They are both associated with similar range of perinatal, environmental and socio-economic risk factors [15]. They have similarly high levels of heritability, high male predominance and often cluster together in families.

ASD and ADHD may be conceptualised as different presentations of a single overarching disorder within a continuum spectrum, characterised by emotional dysregulation as a fundamental component. Though ASD is traditionally regarded as distinct from ADHD, its core symptoms of hyperactivity/restlessness, attention deficits, and emotional lability are also common features of ASD [50]. Social impairment is central to the description and prognosis of both disorders, and many CYP with co-occurring ADHD and ASD often require social skills training interventions [51]. Periods of precipitous transition during human development are also associated with rapid symptom evolution for both ASD and ADHD, such as in the preschool, adolescent or elderly age, taking up parenting, employment or higher educational roles [28].

Table 4. Qualitative differences between ASD and ADHD Symptomatology

SYMPTOMS	ADHD	ASD
Inattention	Short attention span, easy distractibility, flitting	Learning difficulties/defiance, alternate interests, impaired joint attention/ focus shift
Hyperactivity	Motor hyperactivity	Sensory-seeking behaviour, stereotypies
Impulsivity	Impaired inhibition	Reduced empathy
Social Communication	Pragmatic Speech & Language delay	Core impairment
Reciprocal interaction	Social immaturity / inattention, misses cues, annoying	Low social enjoyment
Stereotyped interests/behaviour	Hyper-focus, habitual movements	Compulsive
Emotional disorder	Mood swings	Mood dysregulation / Irritability
Brain structures affected	Prefrontal cortex and sub-limbic system	Orbitofrontal cortex, superior temporal sulcus, fusiform gyrus, amygdala, and cerebellum

ASD and ADHD could be considered as the most extreme presentations of the same disorder on the opposite ends within a continuum spectrum. Both conditions are associated with social functioning difficulties, unique motor skill deficits and sensory features in comparison to normal developing children, with subtle variations in the quality of the impairments [52]. In a recent observational study of 225 Italian CYP, both ASD and ADHD diagnosis was associated with significant social impairment, compared to the non-diagnosed control. The peculiar social deficits in ADHD related to poor social performance, while ASD children manifested greater deficits in social perception and in certain forms of social knowledge [53].

The ASD-ADHD spectrum may present with features spanning from predominantly core ADHD symptoms with scarcely any social difficulties, through combined ADHD and ASD symptoms, up to ASD presenting with predominantly high levels of social and communicative difficulties, which may regarded as the severest end of the spectrum. Some authors have described six subcategories of this hypothesised ASD-ADHD spectrum, using evidence gathered from extant literature and clinical experience [54]. ADHD only is regarded as the milder end of the spectrum while ASD with different degrees of comorbid ADHD symptoms make up the more severe subtypes.

This would imply that both disorders are assessed and managed within the same integrated multidisciplinary team and avoid the current state of disjointed and fragmented services experienced by the affected CYP and the families, usually split between Child Health and Child Mental health teams [55].

Severe ASD Spectrum with peculiar "attentional deficits"

Some researchers have also hypothesised that co-occurring ASD-ADHD may be conceptualised as a form of severe ASD and not two separate diagnoses, characterised by a peculiar attentional deficit, with the associated functional impairments. It has been suggested that peculiar attentional deficits represent a universal presentation in CYP with ASD, which invariably leads to the characteristic social communication impairments. It has been argued that diagnostic categorisation of ASD should include their distinct attentional difficulties. The attentional deficits in ASD is unique and is not equivalent to the classical inattention symptoms of an independent ADHD diagnosis [56]. Though people with ASD may have stronger sustained, focused, and visual search attention than other people, especially when they are focusing on subjects of interest, they experience peculiar deficits in other attention areas. They struggle with disengaging or shifting attention, orienting attention to non-social stimuli, joint attention, filtering out distractors and paying attention to things that don't interest them. These peculiar attention deficits in ASD contrast with the typical 'short attention span' and 'excessive distractibility' in ADHD patients [56].

This hypothesis is strengthened by the observation that CYP with co-occurring ASD-DHD have worse functional outcomes and treatment response to psychostimulants, compared to those without ASD symptoms, especially in the presence of learning disability [33,57]. The ADHD response to atomoxetine in CYP with ASD is also worse than typically expected for CYP with ADHD but without ASD and or intellectual disability [58].

Overactive Dopamine neurotransmitter pathways involving the basal ganglia - nigrostriatum (basis for the stereotypical behaviours) and mesolimbic system connecting the ventral tegmental area (VTA) with the nucleus accumbens, amygdala, hippocampus, and prefrontal cortex (basis for the interpersonal and perceptual deficits) have been hypothesised as the basis for the restlessness and apparent hyperactivity in ASD. This contrasts with the deficient Dopamine /Noradrenaline pathways in the fronto-limbic brain centres typically seen in ADHD patients. This might explain the empirical perception of Dopamine antagonists (such as Aripiprazole or Phenergan) providing better efficacy in the control of many symptoms of ASD in comparison to Dopamine agonists (psychostimulants) [56]. Some studies have shown that children with ASD alone are more likely to be prescribed antipsychotics and selective serotonin reuptake inhibitors (SSRIs) compared to those with ADHD [59].

It may also be hypothesised that "agitation" and physical restlessness in autistic children are by-products of their joint attention deficits, and unrelated to the hyperactivity typical of ADHD. Hyperactivity in ASD could also be related to their emotional dysregulation linked to irritability [56].

The published heritability estimates for ASD is similar to that of ADHD (between 70 and 90%), suggesting shared genetic vulnerabilities. It has been reported that ASD is more frequently associated (in up to 40 percent) with identifiable genetic syndromes, chromosomal defects such as copy number variants of micro-DNA deletion, insertion or duplication, single gene defects and mitochondrial abnormalities leading to metabolic disorders [48]. This suggests that ASD is a more severe entity out

of the two conditions with greater genetic vulnerabilities.

These observations would suggests that CYP with co-occurring ASD-ADHD need additional psychological and behavioural treatments such as parent training, psychoeducation, psychosocial interventions, cognitive remediation, adaptive behaviour and social skills training, preferably in combination with pharmacological therapy to control ADHD symptoms [32,60,61]. This emphasises the need for a multidisciplinary, holistic and integrated care approach to effectively address their complex needs.

DISCUSSION

Clinicians diagnosing and managing CYP with various NDD are conversant with their common co-occurrence with each other and with other physical and psychiatric disorders. The current categorical diagnostic classification based on often arbitrary thresholds of meeting a number of symptom-criteria, may be unsuitable for many affected CYP. A more holistic approach of managing individual symptoms, rather than a cluster of unrelated symptoms, would offer better clinical and psychosocial benefit for a wider range of CYP, including those presenting with subthreshold traits and a complex mixture of psychopathology.

It may be argued that older editions of DSM and ICD which embraced a more traditional categorical classification of psychiatric disorders, in which one overarching diagnosis precludes any further related diagnosis, and restricted the potentially problematic tendency to multiply comorbidity diagnoses are to be preferred for avoiding further service fragmentations for the affected CYP[62], The older editions of the classification manuals were based on the hierarchical descending order of clinical conditions, with the top order diagnoses excluding the lower ranking disorders. This would have been a rational basis for the exclusion of ADHD diagnosis in CYP with ASD in the older editions of the DSM, since many of the ADHD symptomatology could be explained by the index ASD. We believe that this hierarchical model still has clinical utility today to avoid multiplication of comorbid diagnoses, instead of providing individualised patient care based on their range of difficulties and not determined by the number of their separate diagnostic labels.

This review paper has examined a few conceptualisation theories that would help the Clinician to offer a more holistic care for CYP with NDD. Assessment of CYP with either suspected ASD and or ADHD requires a multidisciplinary and multifaceted approach, including use of standardised and semi-standardised validated tools for both symptoms and associated impairments, detailed developmental, clinical and family history, and a current mental state examination, and if necessary, assessment of risk and cognitive ability [32]. This would require the integrated expertise of different professionals including paediatricians, psychiatrists, allied therapists, educationist, social workers and voluntary sector professionals.

This review has highted alternative concepts of viewing wider range of difficulties as inter-related neurodevelopmental spectra, rather than emphasising the individual diagnostic classification labels. Embracing this alternative paradigm of describing and conceptualising neurodevelopmental disorders would encourage practitioners not to restrict individuals into constrained diagnostic boxes and rather see each CYP as a unique individual requiring a comprehensive package of care tailored to their peculiar list of strengths and difficulties.

Over-focusing on the child's the most prominent difficulties to formulate a discrete diagnosis is likely to misinterpret their entire vulnerabilities and ignore more subtler impairments in other areas of daily functioning.

Managing comorbid NDEBID conditions can be quite challenging both for the Clinician and the patients. The process of assessment, gathering information from multiple sources, formulation of diagnosis, selection of appropriate treatment options and subsequent monitoring and follow up involve complex processes that need to be carefully coordinated and executed with precision. The patients and their families also come with high expectations and demand almost immediate answers to their longstanding concerns. Due to the current traditional models of care based on individual diagnosis, rather than holistic patient-centric need-oriented care, fragmentation of treatment and support services is unfortunately a common experience of patients with NDD and their carers [63]. Considering the almost universal occurrence of comorbidity of several conditions in CYP with NDD, there are strong arguments in favour of greater integration between mental health, physical and neurodevelopmental paediatric services [64].

Interventions for ASD-ADHD co-occurrence may require modifications of standard strategies across multiple settings. Medication treatments may require modifications for optimal outcome in comorbid conditions. More research is needed to

develop novel clinical and neuropsychological approaches to assess important psychopathological features of the concurrent ASD-ADHD patients.

Nordgaard et al [11] have suggested some strategies that could help to reduce the prevalence of comorbid diagnoses and promote a more holistic care. Firstly, by differentiating between comorbidity entities in terms of stability and duration of their symptoms. Conditions that can fluctuate in severity, such as ASD, ADHD, schizophrenia, or personality disorders, should be regarded as co-occurring traits, and be differentiated from the more stable disorders which are usually present for a longer time, over several years as the main diagnosis. Secondly, Practitioners should not immediately make more than one diagnosis at the first meeting with the patient, but rather prioritising diagnosing and treating the condition with most severe impact, to see if the other trait conditions recede with it. While these suggestions do not necessarily embrace the concept of a wider spectrum of related NDEBID conditions, it suggests that each Clinicians would take responsibility for continued care and monitoring of each patient, ensuring early recognition of new or emerging symptoms, and addressing them appropriately.

CONCLUSION

There is increasing published evidence to support the paradigm of making a single diagnosis of severe ASD with co-occurring ADHD symptoms or more generic description of a wider range of symptoms associated with any NDEBID, as highlighted in this review. It is conceivable that this would likely provide a better understanding for many CYP with apparent comorbid diagnoses. Some advantages to this approach include realisation that a modified approach to treatment should be explored and encouragement of further research. There are potential ethical implications for shifting from the current model of multiple comorbid diagnosis to single umbrella terminology concepts. The patients and their families may feel that their complex difficulties are not appropriately and comprehensively appraised. It's possible that additional health, social and educational resources are required to accommodate this new model. Many clinicians currently working in smaller isolated teams may feel overwhelmed by the need to provide additional non-pharmacological interventions to the patients, beyond the limits of their training and competence, in the absence of other supportive allied experts. Clinical and empirical research of this alternative model would help guide the practitioners and commissioners about the best evidence-based model that would provide most cost-effective benefits.

Optimal interventions for CYP with co-occurring ASD-ADHD requires a good comprehension of the fundamental neuropathology and the peculiar deficits involved.

A more unified approach to the description of NDEBID would help to buttress the idea of their common aetiological factors in the early developing brain and emphasise that co-occurrence of recognisable patterns is the norm rather the exception. The affected CYP often have many unmet underlying mental health, physical and social needs, best suited for an holistic integrated care approach [1,64,65]. This would encourage better integration and joint working between paediatricians, psychiatrists, other allied therapeutic and mental health professionals. The potential benefits of this coordinated approach are many both for the staff and service users [55]. The benefits include better patient-centred care, more cost-effective services, improved staff morale and improved health outcomes. Evidence have shown that multi-agency collaboration between services such as education, health and social care leads to more comprehensive assessments and integrated services which promote "holistic development across life domains" [66].

Clinical experience and empirical research evidence have shown that treatment of ADHD symptoms is less effective in CYP with ASD-ADHD co-occurrence, suggesting that the underlying pathophysiological mechanisms for these symptoms are different from those with ADHD alone.

Clinical practice has shown that interventions for co-occurring ASD-ADHD diagnosis may require modifications of standard strategies across multiple settings. Further research is needed to help analyse important peculiar neuropsychological features among individuals with the co-occurring ASD-ADHD features, especially their Attentional deficits.

We argue for a preferred use of the 'co-occurrence' terminology for describing the presence of more than one discrete NDEBID previously diagnosed separately based on taxonomic classification manuals. Future research exploring other unified models of diagnosing and managing CYP with a wide range of NDEBID symptoms would be highly beneficial both for the Clinicians, the affected CYP and their families. This may lead to a more

equitable and holistic care, where interventions are determined mainly by identified needs, rather than non-homogenous discrete diagnoses, which may not necessarily determine the levels of additional support required by the individual patient.

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