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AUTISM SPECTRUM (A RICHDALE AND L HOLLIER, SECTION EDITORS)

# Trends in the Overlap of Autism Spectrum Disorder and Attention Deficit Hyperactivity Disorder: Prevalence, Clinical Management, Language and Genetics

T. May[1,2,3] · A. Brignell[2] · Z. Hawi[4] · A. Brereton[1] · B. Tonge[1,5] · M. A. Bellgrove[4] · N. J. Rinehart[1]

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## Abstract

**Purpose of Review** To review recent literature on the overlap of autism spectrum disorder (ASD) and attention deficit hyperactivity disorder (ADHD), now both conditions can be dually diagnosed in the Diagnostic and Statistical Manual of Mental disorders 5th edition.

**Recent Findings** There is a high comorbidity with rates of comorbid ASD and ADHD ranging from 40 to 83%. Multidisciplinary assessment and management of the combined presentation is thus required. Language difficulties are a common comorbidity in both ASD and ADHD with around 60% of children with ASD and 40% of those with ADHD having language problems. Twin studies show up to 72% of the co-variance of ADHD, and ASD symptoms can be explained by shared additive genetic factors providing a genetic basis for the observed clinical overlap.

**Summary** There are still many gaps in our knowledge with limited research exploring well-defined groups of children with ASD only, ADHD only and ASD with ADHD. Clinicians should thoroughly assess ADHD symptomatology in children with ASD and vice versa to understand the challenges for these children and inform treatment planning.

**Keywords** Autism spectrum disorder · Attention deficit hyperactivity disorder · Comorbidity · Treatment · Language

## Introduction

Two of the most common disorders of child development are attention deficit hyperactivity disorder (ADHD) and autism spectrum disorder (ASD). These disorders frequently present together, resulting in challenges for both clinicians and researchers. Clinical decision-making requires differentiating which clinical symptoms drive the different functional impairment. It can be difficult from a clinical perspective to understand which aspects drive the different impairments an individual experiences and the best approach to their clinical management. The overlap of the two conditions also offers potential insights into underlying aetiology.

ADHD affects around 5–7% of children and is characterized by levels of inattention and/or hyperactivity and impulsivity that are developmentally inappropriate [1, 2]. Inattentive symptoms may present at difficulties sustaining attention on tasks, being easily distracted and disorganization. Hyperactive/impulsive symptoms may range from high levels of activity, difficulty sitting still, excessive talking to acting impulsively without considering the consequences. ADHD emerges during childhood with some cases remitting during adolescence. Around 2–3% of adults experience clinically significant ADHD [3, 4].

ASD affects around 1–4% of the population and symptoms include deficits in social communication, social interaction and restricted, repetitive, stereotyped and odd behaviour

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✉ T. May
tamara.may@deakin.edu.au

[1] Deakin Child Study Centre, School of Psychology, Faculty of Health, Deakin University Geelong, Geelong, VIC, Australia

[2] Murdoch Childrens Research Institute, Parkville, VIC, Australia

[3] Deakin University, 221 Burwood Hwy, Burwood, Victoria 3125, Australia

[4] Monash Institute for Cognitive and Clinical Neurosciences (MICCN), School of Psychological Sciences, Monash University, Melbourne, Australia

[5] Centre for Developmental Psychology and Psychiatry, Monash University, Notting Hill, VIC, Australia

patterns [1, 5]. Social symptoms range through a broad spectrum from making minimal social overtures and having no motivation to engage with others, to having social motivation but lacking the skills to successfully navigate social situations and make and keep friendships. Repetitive behaviours may also range from obvious unusual fixations to narrow but more socially appropriate obsessions, or hypo- or hyper-sensitivity to sensory stimulation. The onset of ASD is in early childhood and it is a lifelong disorder, although interventions may result in symptom reduction over time for some individuals.

Both conditions affect more males than females (ASD M:F ratio is 3:1 [6], ADHD 2:1 [1]) and both are associated with significant clinical impairment in social, academic, occupational and emotional functioning. Both ASD and ADHD have high levels of comorbid psychiatric disorders but frequently, the two conditions co-occur together. This comorbid (or dual) diagnosis has only recently been allowed in one of the common diagnostic guidelines, the Diagnostic and Statistical Manual of Mental disorders 5th edition (DSM-5) [1].

This review explores prevalence, clinical management, language and genetics by focusing on research in the years since the comorbid diagnosis of ASD and ADHD has been permitted. The prevalence relating to the overlap will be reviewed as will management of the comorbid condition. Language difficulties, another common comorbidity in both ASD and ADHD, will be explored to highlight the complexity of comorbidity. Recent updates and limitations in understanding the genetic overlap of the two conditions will also be explored. Finally, the review draws together the challenges in this area as well as possible directions of future research.

## Prevalence: ADHD and ASD Comorbidity

There is no doubt that there is an increasing recognition of the comorbidity of ASD and ADHD. Both conditions are being identified and diagnosed more often than in the past which may relate to an increased awareness, changes in diagnostic criteria and reporting practices particularly for ASD [7]. Previous editions of DSM [8] did not allow clinicians to diagnose ASD in a child with ADHD. However, in 2013, DSM-5 [1] removed this prohibition in response to research that has provided clear evidence that the two conditions can co-exist. Despite the earlier prohibition, clinical reports of comorbidity have been published in the decade prior to DSM-5. ADHD has been reported to be the most common psychiatric condition diagnosed in children with high-functioning ASD (IQ > 70) [9]. Other studies reported that 59 to 83% of youth with ASD also present with ADHD [10, 11]. Research subsequent to DSM-5 examining ASD/ADHD in children [12, 13] reports estimates of comorbid ASD/ADHD that concur with previous findings that ADHD is the most common psychiatric comorbidity in children with ASD with comorbidity rates of 40–70%. Around one third of 4–18-year olds with ADHD have clinically elevated levels of ASD symptoms [14].

More recently, attention has been paid to the prevalence of ASD/ADHD in 6–17-year olds. Joshi et al. [15] found a high rate of comorbid ADHD in youth with ASD (up to 83%) that concurs with earlier findings that ADHD is the most common psychiatric condition in referred populations of youth with ASD [16, 17]. Mansour [18] recently reported that higher levels of ADHD symptom severity, rather than severity of ASD symptoms, were associated with a greater number of comorbid psychiatric diagnoses in school-aged children with ASD. Joshi et al. [15] also reported that in a sample of youth with ASD, rates of ADHD were equally high and evenly distributed among subtypes of ASD, that is those with intellectual disability (IQ < 70) and those without intellectual disability (> 70). In children with ADHD, increasing levels of autistic symptoms have been associated with more oppositional, conduct and anxiety symptoms as well as lower IQ [19].

However, despite the robust presentation of ADHD symptoms, a significant proportion of youths with ASD/ADHD fail to receive ADHD-specific treatment. Hartman et al. [20] commented on the biased focus of research on children with ASD/ADHD rather than older individuals and reported findings from their study that investigated individuals aged from birth to 84 years. ASD/ADHD symptoms were at their highest during adolescence and lower in early childhood and old age. They recommend that a lifespan approach is necessary to understand the symptomatology of comorbid ASD and ADHD and further, that in light of their finding that adolescents with ASD/ADHD were more severely affected, that they should be a focus for future research and the development of treatment options.

## Management of ADHD Symptoms in ASD

The co-occurrence of developmentally excessive symptoms of inattention, hyperactivity, distractibility and impulsiveness in children with ASD adds considerably to the level of emotional and behavioural disturbance in the child and the stress and burden for parents and teachers that occurs for either condition separately [21]. Further, children with ASD are more likely to have other comorbid neurodevelopmental, emotional and behavioural difficulties which even further complicate their presentation including anxiety and mood disorders, obsessive-compulsive disorder, problems regulating their emotions with irritability and aggression, epilepsy, disturbed sleep and other chronic or current medical conditions, and intellectual disability. Therefore, the necessary and essential first step in planning the management of comorbid ADHD is a comprehensive assessment of the presenting symptoms, mental state and behavioural observation, developmental and family history, cognitive/educational/neuropsychological

standardized assessments, speech and language assessment, standardized parent and teacher behavioural questionnaire reports and various medical and neurological imaging investigations when indicated. This is of necessity a specialist and multidisciplinary process. For example, the management of a child with ASD and ADHD in the setting of fetal alcohol spectrum disorder who is living in a socially and economically disadvantaged and fragmented family might be different to that of a child with ASD and ADHD who also suffers from significant separation anxiety within a cohesive and supportive wider family in which the mother is suffering from a recurrent depressive illness. Cultural expectations regarding the behaviour of children and approaches to their care will also influence a family's response to management suggestions. Therefore, the treatment of ADHD in a child with ASD must always occur in the context of a wider approach to management focused on addressing the various symptoms of ASD such as delayed and abnormal social communication, non-functional ritualistic behaviour and other comorbid problems such as generalized anxiety, motor dyspraxia or language problems. The decision to also treat symptoms of ADHD would be based on their severity and the significance of their contribution to the child's functional impairment. The salience of the extra disability created by symptoms of ADHD in a child with ASD means that their successful treatment can lead to significant improvement in the child's adaptation and quality of life, even though symptoms of ASD remain.

Approaches to the treatment of ADHD in ASD are based on the best practice management of young people with ADHD in general. These are described in a number of clinical guidelines including the NICE Clinical Guideline no. 72 [22], SIGN Clinical Guideline 112 [23] and the American Academy of Pediatrics Clinical Practice Guideline [24]. Initial approaches to management include an evidence-based, parent education and skills training program [25], educational and behavioural management strategies [26] and treatment of other comorbid problems such as speech therapy for language disorder. Currently, evidence is lacking on the benefits or harms of elimination and supplementary diets, and naturopathic and physical interventions to endorse their use, apart from the adverse stimulating effect of some food colourings such as tartrazine on child behaviour [27, 28]. Consultation with the teacher regarding classroom management, including approaches to the management of associated learning and social interaction difficulties, is helpful.

If psychosocial and environmental approaches to management are ineffective, then treatment with medication is indicated. In general, for children with ADHD who do not have ASD, psychostimulants (dexamphetamine or methylphenidate) are the first choice given consistent evidence that they reduce symptoms of inattention and hyperactivity, improve learning outcomes and school adjustment and improve family and social interactions in the medium term of up to 3 years [24]. However, the evidence for the same level of effectiveness and benefit for their use in children with ADHD and ASD is less robust with response being more variable and troublesome and unacceptable side effects occurring more commonly [29–32]. There are no clear indicators of response, with some children showing no response and others showing significant therapeutic benefit [33]. The increased risk of adverse side effects includes appetite suppression, increased emotionality such as anxiety and irritability, tics, sleep disturbance and increased hyperactivity [30, 31]. Given the possibility of a positive therapeutic response to treatment with stimulant medication, it is reasonable to offer this but parents must be warned of the more limited chance of a therapeutic response and the increased risk of side effects [34]. It is best to initiate treatment with a low dose of around 0.125 mg/kg, three times a day (breakfast, midday and early afternoon) increasing slowly if indicated [35]. The use of standardized symptom checklists and parent and school reports is necessary to follow the response to medication and the medication should be stopped if there is no therapeutic response or if symptoms worsen or problematic side effects develop.

There are a number of other medications with evidence for their efficacy in the treatment of ADHD in young people who do not have ASD but the evidence for their use with comorbid ASD is limited or lacking. There is some evidence in a small open-label trial that atomoxetine (noradrenergic reuptake inhibitor) might be of some therapeutic benefit in children with ASD who have ADHD [36] although it might be more effective in young people with ASD who have less severe symptoms of autism [37]. The use of atomoxetine might also be indicated when tics and anxiety are problematic side effects from the use of stimulant medication and when a once-a-day-only dose is desirable, particularly for compliance. Suicidal thinking and adverse effects on liver function are potential side effects, which should be monitored. There is good evidence for the effectiveness of the neuroleptic risperidone in the treatment of disruptive behaviour and irritability in young people with ASD and this might include symptoms of hyperactivity [38]. Aripiprazole is another neuroleptic medication with similar benefits to risperidone in the treatment of disruptive behaviour, including hyperactivity in young people with ASD [39]. Side effects with these neuroleptic medications can be considerable to the extent that they must be ceased because of weight gain and associated metabolic abnormalities and dystonic effects [40]. Some limited controlled studies of clonidine and guanfacine indicate a therapeutic response for ADHD symptoms in young people with ASD although this response might attenuate with time [41, 42]. There is no evidence for the benefit on ADHD symptoms of some other medications used in ASD such as selective serotonin reuptake inhibitors or anticonvulsants with mood-stabilizing effects such as sodium valproate [43]. Clonidine might also have the advantage of a sedative effect when used at night time.

This may assist with the insomnia that can occur as a symptom of ADHD but also as a side effect of stimulant medication. If there is limited or no therapeutic response to a medication and there are no adverse side effects, then the dose can be gradually increased to the maximum tolerable dose. If there is then no evidence of therapeutic response, confirmed using behaviour checklists, the medication should be ceased and the treatment plan re-evaluated [44].

## Language Development in ASD and ADHD

Difficulties with understanding and using aspects of language such as syntax, semantics and morphology are not a core diagnostic feature of ASD or ADHD, yet they commonly co-occur. It is estimated that around 63% of individuals with ASD have language difficulties [45, 46], with around 25–30% remaining minimally verbal [47]. Estimates of the co-occurrence of ADHD and language difficulties vary widely, ranging from 8 to 90%, depending on study methodology (see Redmond 2016 [48] for a review). Community-based studies report that 40–45% of children with ADHD have co-occurring language problems [46, 49, 50] and the risk for language difficulties is around three times higher than it is for children without ADHD [49, 50].

Some of the key themes investigated with regard to the language problems observed in ADHD and ASD include whether each condition has a unique language phenotype or whether language profiles are shared with other conditions such as specific language impairment (SLI). Further key areas include possible overlap in aetiological and neurocognitive pathways, whether the diagnostic features of each condition (e.g. difficulties with joint attention or inattention) may exacerbate language development, and limitations with regard to available measurement tools and differential diagnosis [48, 51–56].

There is substantial heterogeneity in language abilities in children with ASD. Delay in the onset of talking and difficulties with the content and form of language (specifically syntax, semantics and morphology) commonly occur in ASD [52, 56–58]. Features of language reported to be more unique to ASD include early regression in communication skills at around 2 years of age [59–61], use of stereotyped language or echolalia (echoing words) beyond a developmentally appropriate age [62–64]), pronoun reversal (e.g. using 'you' instead of 'I' [62, 65]) and relative weakness in receptive compared to expressive language ability [66, 67]. Recently, the extent to which these difficulties are specific to ASD has been challenged (see Gernsbacher 2016 [54] for a review). For example, there are reports of children without ASD demonstrating echolalia [68], pronoun reversal [69] and loss of language between 1 and 2 years of age [70]. Furthermore, a meta-analysis of 74 studies found despite children with ASD demonstrating delays in language (1.5 SD below same age peers), there was no evidence of expressive over receptive language advantage [71]. Although some children with ASD may demonstrate the aforementioned features, there appears to be limited support for their use as reliable diagnostic markers of ASD.

Possible language subgroups in ASD have also been examined. One study identified two distinct language profiles in children with ASD [72]. Children with higher verbal skills were similar to children with typical development (TD) on most language measures (vocabulary size, morphosyntax and wh-question complexity), but children with ASD who had low verbal skills were slower to develop on most language measures. Other studies have investigated putative predictors of language outcomes. These include differences in neural substrates [73] and a range of environmental and behavioural factors such as maternal education, play, imitation, joint attention, intervention and cognition, (e.g. [74, 75]). Non-verbal IQ and early language ability appear to be the most consistent predictors of language outcomes in children with ASD [76, 77]. However, there is mixed evidence about the extent to which social communication or ASD symptom severity impact language development, particularly whether there are sensitive periods in development where ASD symptoms have a greater influence over language relative to other periods [74, 77].

Children with ADHD have a high prevalence of language difficulties, as measured by screening tools (e.g. [49]), parent-report measures (e.g., [78]), language assessments (e.g. [79, 80]) and specific language tasks (e.g. [81]). They are at increased risk for delayed onset of first words and word combinations and lower performance on language assessments of syntax, vocabulary, reading, narrative and short-term memory tasks [50, 82, 83]. There is also evidence that children with ADHD have pragmatic language difficulties (e.g. more stereotyped conversations, problems with conversational rapport and problems with social relationships) [78, 83, 84]. A recent review and meta-analysis of studies investigating language difficulties in ADHD found children with ADHD had significantly lower functioning compared with controls across most language domains including overall, receptive, expressive and pragmatic language [80].

Specific features of language have not been described in children with ADHD to the extent they have in ASD; however, comparison with conditions other than ASD has been made. At the conversation level, utterance formulation measures differentiated children with ADHD from those with typical development (TD) and specific language impairment (SLI) in one study [85]. However, children with ADHD were comparable to children with TD on measures of vocabulary diversity, average sentence length and morphosyntax [85]. In addition, children with ADHD and co-occurring language impairment (LI) were found to be comparable to children with

SLI on measures of tense marking, non-word repetition and sentence recall [86]. Language difficulties in ADHD have been found to adversely impact academic performance in areas such as reading, math computation and academic competence [49, 82] but there is little evidence they are associated with children's social functioning [49]. Interestingly, one study found ADHD did not have an independent aggravating impact on children's LI, suggesting children with ADHD + LI may not experience a 'double deficit' by having both conditions [48].

Direct comparison studies of language ability between children with ADHD and ASD are rare. In one study that used a parent-report measure of language, no group differences were found between ASD, ADHD and TD in syntax, speech and semantics [78]. Children with ASD had more severe difficulty in pragmatic language relative to children with ADHD (specifically in the use of context, use of non-verbal communication and quality of social relationships); however, the two groups demonstrated similar profiles when compared to the TD children, with both having greater difficulty with pragmatic language than the structural aspects of language [78]. Miniscalco et al. [87] found children with language delay had difficulty with narrative language tasks irrespective of additional comorbidity (i.e. ASD, ADHD) but children with ASD and ADHD scored low on a Freedom from Distractibility Task relative to children with language delay only. Further, group comparison studies are crucial if we are to understand what aspects of language development may be distinctive and the patterns of strength and weakness relative to each condition [88, 89]. This will help us better understand risk indicators and causes (e.g. neurobiological, genetic) and tailor the most appropriate interventions.

The mechanisms by which language difficulties co-occur in ADHD and ASD are highly complex. Whether language impairment is a risk factor on the pathway to ADHD/ASD or the reverse is true is not well understood [48, 49]. Moreover, the extent to which the overlap between language and ASD/ADHD reflects separate, partial or shared causal pathways continues to be debated [48, 50, 53, 55, 56]. It is hoped future research, some of which is currently underway (e.g. [52, 73, 90]), linking genetic and neurobiological underpinnings of language in ASD/ADHD to our current understanding of the conditions will provide further important insights.

## Genetic Risk Factors for ASD and ADHD

The contribution of environmental factors to the aetiology of both ASD and ADHD is well established [91–94]. However, increasingly a strong genetic component is recognized. Approximately, 70–90% of the phenotypic variance for either disorder can be explained by additive genetic factors [95, 96]. Crucially, twin studies using questionnaire-based data show that up to 72% of the co-variance of ADHD and ASD symptoms can be explained by shared additive genetic factors [97], thus providing a genetic basis for the observed clinical overlap. The results of segregation analysis in family-based designs indicate that a polygenic mode of inheritance is likely for both conditions [98, 99].

The last 15 years of linkage, candidate gene and genome-wide association studies have identified several chromosomal regions of interest to ASD and ADHD. Although no study has directly contrasted the molecular genetic drivers of ASD to those of ADHD, overlap is inferred from the co-occurrence of genetic association signals across studies. For example, there is strong evidence for an ADHD linkage peak on chromosome 16p13 [100] that overlaps with findings of three genome-wide scans for autism at 16p13 [101–103]. Further, ADHD-specific candidate genes have also been reported to be associated with ASD. Specifically, there is replicated evidence of association with the genes encoding dopamine beta hydroxylase (DBH) and the serotonin transporter (SLC6A4) with both ASD and ADHD [100, 104, 105].

Although no one single-nucleotide polymorphism (SNP) association with either disorder has attained genome-wide significance (GWAS significance level $p \leqslant 5 \times 10^{-8}$), a quantitative trait loci (QTL) analysis identified a risk variant at cadherin 13 (CDH13) associated with inattentive symptoms in children with ADHD [106]. Although no common CDH13 SNPs have been reported to associate with autism, a genome-scan linkage analysis identified substantial evidence of linkage signal at 16q23 which harbours the CDH13 gene [107]. The potential importance of CDH13 in ASD was further supported by findings from rare recurrent de novo copy number variations (CNVs) at 16p13.2 encompassing the genes USP7 and C16orf72 and CDH13 in ASD [108]. A GWAS locus for ASD (rs4307059) was also mapped between cadherin 9 (CDH9) and 10 (CDH10) [109], further indicating the importance of cadherin gene superfamily in both conditions.

More recently, GWAS meta-analysis of SNP associations in five major disorders including schizophrenia (SZ), bipolar disorder (BPD), major depressive disorder (MDD), ADHD and ASD involving 33,332 cases and 27,888 controls of European ancestry reported interesting findings [110]. Three SNP loci demonstrated cross-disorder associations. These SNPS were mapped to regions on chromosomes 3p21 and 10q24. The first was rs2535629, which mapped to intron 7 of the inter-alpha-trypsin inhibitor heavy chain 3 (ITIH3). However, two other inter-alpha-trypsin inhibitor genes, the ITIH1 and ITIH4 were also mapped within this genomic region. The association signal may have resulted from any of these genes. The two other association signals were mapped to rs11191454 and rs2799573 on chromosome 3. The first SNP

was located to arsenite methyltransferase (AS3MT) whereas the second was mapped to the calcium voltage-gated channel auxiliary subunit beta 2 (CACNB2) gene. Finally, SNPs with evidence of cross-disorder association were enriched for brain expression quantitative trait loci (eQTL) markers. Evidence of GWAS associations with both ASD and ADHD were also observed at several loci such as neuron navigator 2 (NAV2), integrin alpha 11(ITGA11) and synaptotagmin XVII (SYT17) [111].

CNV studies have also shown that several ASD susceptibility loci (1p36, 1q21.1, 15q11.2-q13.1, 15q13.3, 16p11.2 and 22q11) were enriched for CNVs in an ADHD population [111]. Overall, emerging evidence shows that ASD and ADHD may share overlapping genetic substrates. However, these findings were derived from samples which were ascertained during the era when the hierarchical rule of DSM-IV precluded dual diagnosis of ASD with ADHD. To further understand the genetic overlap between ASD and ADHD, direct comparison of DNA changes in samples of ASD children without ADHD, ADHD children without ASD and children with features of both disorders is now required.

## Conclusions

A substantial body of research evidence on the overlap of ASD and ADHD resulted in removal of the DSM-IV-TR prohibition for dual diagnosis in those with ASD in the DSM-5 released in 2013. Research on the overlap of these two conditions since this time is well advanced in some domains, such as understanding the prevalence. However, there is a lack of research on the comorbid condition in others such as language profiles, pharmacological treatment and understanding the presentation and impacts of the comorbid condition throughout the lifespan. Since genetic studies of the overlap of ASD and ADHD have largely used populations ascertained prior to DSM-5, the exact extent of the genetic overlap remains unclear. Exploration of well-characterized diagnostic groups with ASD, ADHD and ASD with ADHD is needed across the above areas. Given the well-established overlap of ASD and ADHD, clinicians should thoroughly assess ADHD symptomatology in children with ASD and vice versa to better understand the challenges for these children and inform treatment planning.

## Compliance with Ethical Standards

**Conflict of Interest** The authors declare that they have no competing interests.

**Human and Animal Rights and Informed Consent** This article does not contain any studies with human or animal subjects performed by any of the authors.

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