



# Autism Spectrum Disorders and ADHD: Overlapping Phenomenology, Diagnostic Issues, and Treatment Considerations

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## Abstract

**Purpose of Review** Autism spectrum disorder (ASD) and attention deficit/hyperactivity disorder (ADHD) are both increasing in prevalence and commonly co-occur with each other. The goal of this review is to outline what has been published recently on the topics of ASD, ADHD, and the comorbid state (ASD+ADHD) with a particular focus on shared phenomenology, differential diagnosis, and treatment considerations.

**Recent Findings** ASD and ADHD have shared genetic heritability and are both associated with shared impairments in social functioning and executive functioning. Quantitative and qualitative differences exist, however, in the phenotypic presentations of the impairments which characterize ASD and ADHD. For ASD interventions to be maximally efficacious, comorbid ADHD needs to be considered (and vice versa).

**Summary** The research on ASD and ADHD suggests some overlap between the two disorders yet enough differences to indicate that these conditions are sufficiently distinct to warrant separate diagnostic categories.

**Keywords** ADHD · Autism · Autism spectrum disorder · Neurodevelopmental disorder · Comorbidity · Diagnosis · DSM-5

## Introduction

Attention deficit/hyperactivity disorder (ADHD) and autism spectrum disorder (ASD) are both neurodevelopmental disorders which typically onset in childhood. ADHD is defined by the presence of impairing symptoms of inattention and/or hyperactivity–impulsivity that onset before age 12, is present across two or more settings, and cannot be better explained by another condition [1]. ASD is characterized by enduring and impairing social communication and interaction deficits that occur across multiple contexts along with the presence of restricted, repetitive behaviors, interests or activities, or sensory symptoms [1].

Prior to the *Diagnostic and Statistical Manual for Mental Disorders – 5th edition* (DSM-5) [1] in 2013, clinicians were unable to make an ADHD diagnosis in the context of ASD. It

was presumed that any symptoms of inattention and/or hyperactivity–impulsivity were secondary to ASD and not due to an additional ADHD diagnosis [2]. With this exclusionary criterion lifted in the DSM-5, it is not surprising that a vast literature has been published within the past several years on the topic of ADHD, ASD, and ASD+ADHD. (Please see Fig. 1 for graphical representation of this increase in research activity.)

Both ADHD [3] and ASD [4] are increasing in prevalence and the symptoms and impairments of both conditions often persist into adulthood [5•]. When considered in the context of the substantial impairments and societal costs (e.g., reduced parental quality of life [6]) associated with ADHD [7], ASD [8], and the amplification of those negative outcomes in the comorbid condition (ASD+ADHD) [9–14], it is clear that ADHD, ASD, and ASD+ADHD represent a public health problem. For example, children with ASD constitute 8% and children with ADHD represent 13% of all youth receiving school-based services under the Individuals with Disabilities Education Act [15]. Thus, these two conditions alone account for nearly one-fourth of all children receiving school-based services.

ASD and ADHD often co-occur [16]; 13% of youth in a large epidemiological ADHD study were diagnosed with

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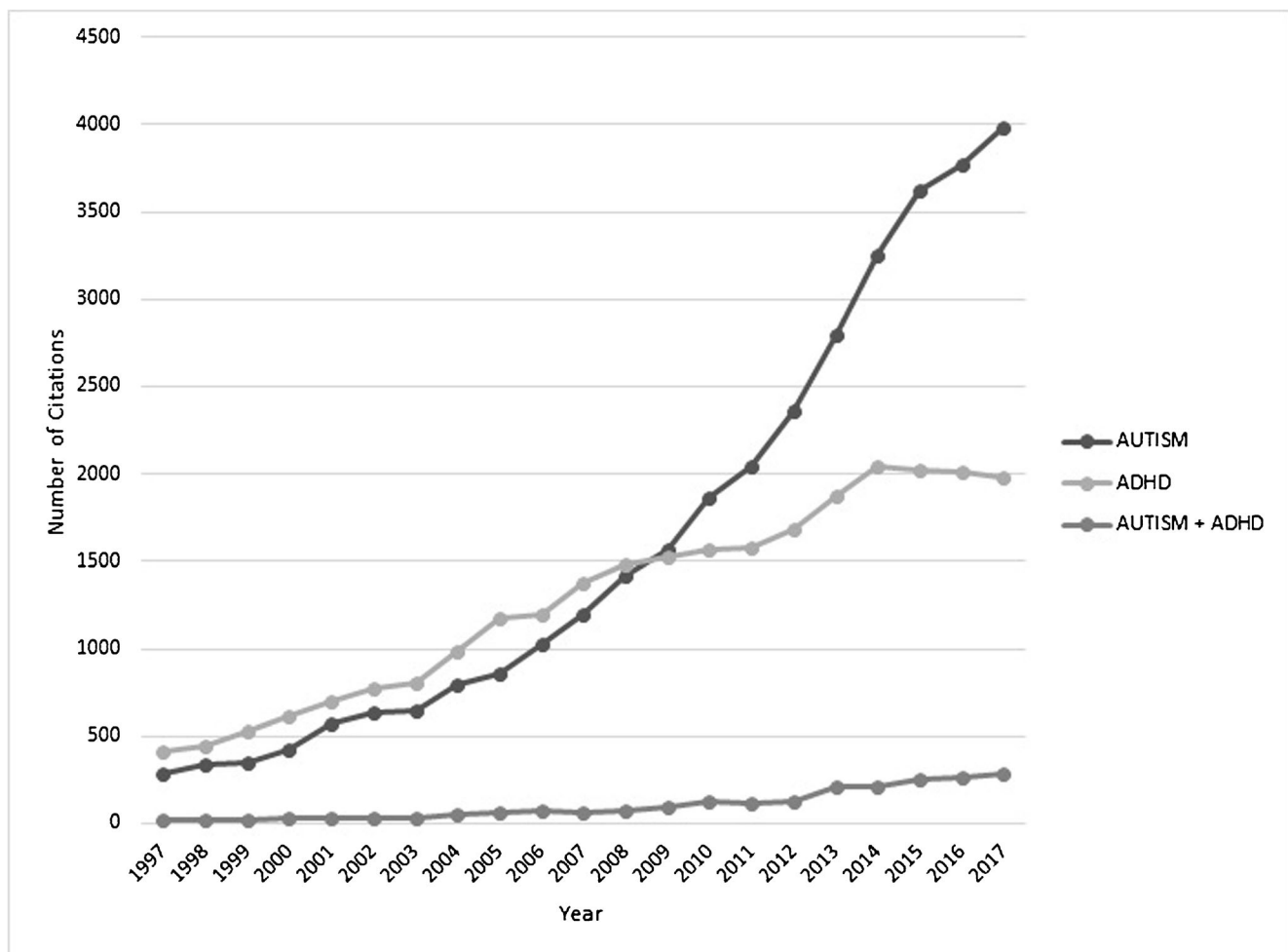


Fig. 1 Number of PubMed citations for ASD, ADHD, and ASD+ADHD

comorbid ASD [17]. Others have similarly reported that approximately 1 in 8 youth with ADHD have ASD [18]. Conversely, ADHD is the most common comorbidity in children with ASD with comorbidity rates in the 40–70% range [19–22]. The substantial overlap between ADHD and ASD presents clinicians with difficult differential diagnostic [23] and treatment [24•] considerations. For example, youth with ADHD are diagnosed with ASD approximately 2 years later than children with ASD without a pre-existing ADHD diagnosis [25, 26]. Similarly, youth with ADHD who do not have an ASD diagnosis still have elevated levels of ASD symptoms [27] and vice versa [28••]. Given all of the above, understanding the phenotypic expression of ADHD, ASD, and ASD+ADHD is an important clinical goal [29].

In this review, we consider the recent empirical literature that has been published on ASD, ADHD, and, when applicable, ASD+ADHD. Our goal is to cover the overlapping phenomenology between ASD and ADHD from a biopsychosocial perspective. We then consider issues and complexities associated with diagnosing ASD and ADHD

and conclude with treatment considerations relevant to ASD and ADHD.

## Overlapping Phenomenology

**Biology** Both ADHD and ASD are considered to be neurodevelopmental disorders that onset in childhood, and although causal links are currently unknown, both disorders are highly heritable with approximately 70–80% of both phenotypes being accounted for by genetics [30, 31]. Further, when one dyad of a twin pair has ASD, there is a much higher likelihood that the unaffected twin will have symptoms of ADHD [32]. Hyperactive–impulsive symptoms correlate strongly ( $r = .56$ ) with restricted and repetitive behaviors in ASD [33] and family members of individuals with ASD have elevated rates of ADHD diagnoses [32, 34, 35] and vice versa [36]. For example, siblings of probands with ASD have nearly a 4-fold increased risk for ADHD compared to matched controls [37•].

All of the above has led to the speculation that ASD and ADHD have shared genetic heritability [32]. As of yet, no specific gene variations have been identified that link these disorders together, but current research examining genome-wide copy number variations (CNV's) have identified increases in rare CNV's at similar loci among those with ADHD, ASD, intellectual disability, and schizophrenia, providing preliminary evidence of shared genetic pathways between these disorders [38, 39].

Beyond genetics, there is also little overlap in findings from neuroimaging studies between those with ADHD and those with ASD with respect to either resting state connectivity or functional network activations. However, methodological complications and limitations might account for a large proportion of the lack of consistency. These limitations include, among others, the heterogeneity of symptom presentations both within and across disorders, the small sample sizes of these often expensive and intensive studies, differences in methodologies across studies, and the wide range of ages used within and across studies that complicate the interpretation of findings given the developmental nature of both disorders.

In spite of these limitations, several research groups have been at the forefront of this work in the last decade or so. The findings from their studies [40•, 41], which are corroborated by recent meta-analyses [42, 43], suggest that ADHD and ASD are characterized as disorders of large-scale connectivity but with little overlap in the specific regions that are under or over connected, with one exception. Children with ADHD and those with ASD and ADHD symptoms, but not those with ASD without ADHD symptoms, showed connectome-wide dysconnectivity in the precuneus, an area considered to be a hub of the default mode network, involved in mind-wandering [40•]. The findings from this study suggest a shared neural atypicality in the impact of ADHD symptoms among those with and without a comorbid ASD diagnosis, and support the importance of future research in this area.

**Social Function in ADHD and ASD** Social difficulties are a hallmark of ASD and are required for a diagnosis. Although deficits in social function are not explicitly required for a diagnosis of ADHD, they factor into several diagnostic criteria that include the following: “often has difficulty waiting in line,” “often blurts out answers,” and “often interrupts or intrudes on others,” and these are commonly reported by parents of youth with ADHD as causing social impairment [44]. The social impairments of ADHD seem to reflect impulsivity or hyperactivity, but might also reflect more general social dysfunction. Recent work has provided some clues as to the nature of these impairments by focusing on the mechanisms underlying their expression in ASD and ADHD.

**Social Cognition** Determining the mechanisms underlying social impairment may provide important clues to the nature of

the overlap between those with ADHD and those with ASD. For example, social perception abilities, as measured by the reading of the Eyes Test, in which participants determine an individual's mental state on the basis of black and white pictures of eyes, varied upon a continuum among those with neurodevelopmental disorders that included ASD, ADHD, and obsessive-compulsive disorder (OCD) in relation to typically developing comparison participants. Those with OCD trended towards having better social perception than even the typically developing groups, while those with ASD had the greatest deficits, with the performance of those with ADHD being intermediate [45]. In addition to examining group differences, the authors also examined the contribution of ADHD and social communication symptoms to task performance across diagnostic groups. They found that across groups, hyperactivity, but not inattention exerted a negative effect on social perception scores, and that when controlling for social communication scores, all group differences disappeared. Together, these findings suggest that social communication skills/deficits appear to impact social perception similarly, irrespective of diagnostic labels, underscoring it as an important transdiagnostic mechanism underlying levels of social impairment.

**Social Interactions** Low levels of reciprocal friendships are another shared feature between ADHD and ASD [46]. The magnitude of the effect between typically developing peers and those with ADHD on measures of peer regard ( $r = .27$ ) is larger than the effect sizes for other social domains such as social cognition and social behavior [44]. Youth with ASD likewise have significantly low levels of reciprocated friendship [47] and typically developing peers across multiple age groups are less willing to engage with individuals with ASD, often making a decision within 10 seconds of exposure to an individual with ASD [48]. Children with ADHD have intact social knowledge yet impaired social interactions, suggestive of a performance deficit [49]. Conversely, youth with ASD have knowledge deficits [50] and are more likely than those with ADHD to respond to clinic-based social skills training interventions that often teach social skills [51].

The social difficulties of individuals with ASD appear more due to the absence of positive behaviors (e.g., social approach, eye contact) rather than the presence of negative behaviors [52]. Conversely, the social difficulties of individuals with ADHD are more likely due to the presence of negative behaviors such as interrupting and intruding on conversations [53] suggesting differences in the nature of social impairment across diagnosis.

Although social impairment is clearly implicated in both diagnoses, it is not the only domain that has been considered key to understanding the overlap between both disorders. Several studies have compared the cognitive, linguistic, and executive function profiles of both groups, in an attempt to

characterize similarities and differences, as well as search for shared underlying mechanisms between these disorders.

**Psychological** The psychological profiles of both ADHD and ASD are complex, and comparisons between disorders are complicated by the large heterogeneity of cognitive abilities among those with ASD. Comparisons between ADHD, who generally show average cognitive function, and ASD are often focused on those with ASD and higher cognitive abilities. As such, it is important to note that commonalities between the two groups cannot be generalized to the entire autism spectrum. Nonetheless, interesting patterns emerge with respect to one broad psychological function, executive function.

**Executive Functions** Executive function (EF) is broad term that encompasses multiple domains of function including inhibition, cognitive shifting, planning, working memory, and concept formation. Once transdiagnostic executive function impairments are controlled for, ADHD and ASD have their own specific profile of executive dysfunction. While ASD is generally considered a more severe condition, executive dysfunction is more pervasive and severe in ADHD [54]. Others have recently suggested that ADHD and ASD share overlapping, yet unique, executive function profiles [55]. Furthermore, the association between EF and ADHD symptoms remains after controlling for ASD symptoms. This suggests an additive nature for the comorbid condition (ASD+ADHD) [56].

Executive function has been studied extensively in both ADHD and ASD, with consistent findings of deficits relative to both age- and IQ-matched typically developing participants in both groups [57–59]. However, there appears to some differentiation between the two diagnostic categories. Specifically, individuals with ADHD appear to struggle most clearly with inhibition, the ability to withhold a pre-potent response, and planning/problem solving, while those with ASD struggle most with cognitive flexibility, which requires holding and switching between multiple perspectives rapidly [58]. Further, age-related improvements are less clear for those with ADHD than ASD, and task performance is positively correlated with parent-reported social and communication abilities, and negatively correlated with hyperactivity for TD and ASD groups, but not those with ADHD [58]. These findings suggest that EF is more impaired in ADHD than in ASD, that those with ADHD tend not to improve with age, and further corroborates the notion that profiles of performance do not overlap considerably between the groups.

Studies examining the shared EF profiles of co-occurring ADHD and ASD have found that those with an ASD and ASD+ADHD both have cognitive flexibility and planning impairments while those with ADHD and ASD+ADHD have response inhibition difficulties. Compared to youth with ASD, those with ASD+ADHD are more impaired in working memory on emotional recognition tasks and have higher levels of

parent-reported anxiety [60], suggesting an additive nature to the comorbid condition.

Given the consistent findings of social impairments and executive dysfunction in both ASD and ADHD, it is not surprising that the diagnostic overlap between ADHD and ASD peaks in adolescence, possibly due to the increased demands for social adaptation and executive functioning that is present during this developmental period [5•]. Overall psychological profiles of individuals with ADHD, ASD, and co-occurring ADHD an ASD suggest some overlap between the two disorders yet enough differences to suggest that these conditions are sufficiently distinct to warrant separate diagnostic categories.

## Diagnostic Issues

Gold standard diagnostic measures have been developed for both ASD and ADHD and include the Autism Diagnostic Interview-Revised [61] and the Autism Diagnostic Observation Schedule – 2nd edition [62] for ASD and the use of standardized ADHD rating scales, structured interviews such as the KSADS-PL [63], global impairment measures, and behavioral observations for ADHD [64].

**ADHD in ASD** One aspect that is critically and clinically relevant is the validity of using diagnostic scales that are considered best practices for ASD or ADHD to diagnose comorbidities between disorders. Although 40–70% of individuals with ASD have clinically significant ADHD symptoms [19–22], and 20–60% of those with ADHD experience social impairments similar to those reported in ASD [44], the diagnoses could not, prior to this iteration of the DSM, be provided comorbidly. This recent change has prompted a closer examination of inattention, hyperactivity/impulsivity, and social function in these two diagnoses, but several complications have arisen.

The most diagnostically relevant of these is that although individuals with ASD may indeed meet the diagnostic criteria for ADHD as outlined in the DSM-5, the presence or absence of specific symptoms is usually based on parent (and teacher) reports. It is unclear, however, whether parent and teacher reports of symptom endorsement truly represents the presence of both disorders in an individual, which disorder parents are attributing specific symptoms to, as well as whether the constructs measured on ADHD scales measure the same constructs among those with ASD and vice versa. For example, the use of a popular ADHD symptom measure, the ADHD Rating Scale-IV failed to separate inattention and hyperactivity/impulsivity in ASD in a sample of 386 youth with ASD (with normal intellectual function) [65]. The authors recommended that clinical interviewing follows the use of the ADHD Rating Scale-IV to separate ASD symptoms from hyperactivity/impulsivity and inattention [65].



Clinician report of psychiatric comorbidity diagnoses in ASD, especially ADHD, is lower than diagnoses generated by a structured parent interview. For example, mental health clinicians reported that 36% of the youth with ASD that they were treating had comorbid ADHD compared to the 78% of these same youth who met diagnostic criteria based upon a structured interview with a parent [66]. No child characteristics predicted ADHD diagnostic agreement between clinician and parent.

**ASD in ADHD** Both the ADI-R and ADOS-2 have gone through extensive psychometric testing and have adequate sensitivity and specificity. Since ADHD could not be diagnosed in individuals with ADHD, the validation samples of the ADI-R and ADOS-2 did not include participants with ADHD, and as such the discriminant validity of the instruments was not assessed. More recent work suggest that clinicians need to be cautious when using the ADOS-2 and the ADI-R in individuals with ADHD [23]. Although few individuals with ADHD (approximately 11%) met the diagnostic cutoff on both the ADOS-2 and the ADI, 21% of these children met cutoff on the ADOS-2, and 30% met on the basis of the ADI-R. Further, only four items on the ADOS-2 and only a single item on the ADI-R adequately differentiated between those with ADHD and those with ASD. These findings are troubling, even more so given that few clinicians are trained in either the ADOS-2 or the ADI-R, let alone both.

While not a gold standard ASD assessment tool, the Social Communication Questionnaire (SCQ) has been used to differentiate ASD from ADHD and ASD+ADHD. Both ASD groups had higher SCQ total and domain scores than youth with ADHD only. A cut score of 13 on the SCQ differentiated between ADHD and ASD [67]. The Autism Mental Status Examination (AMSE) has demonstrated adequate abilities to detect ASD in children with ADHD. Using a cut score of 5 on the brief, clinician-rated instrument (which correlates highly  $r = .67$  with the ADOS-2) resulted in sensitivity (.83) and specificity (.90) for detecting ASD in youth with ADHD [68].

**ASD+ADHD** Three separate pathways explaining the comorbidity between ADHD and ASD have been demonstrated using structural equation modeling. These pathways are from impulsivity to social information processing difficulties, from hyperactivity to restricted and repetitive behaviors and a pairwise pathway between inattention, verbal IQ, and social information processing difficulties [69].

A latent class analysis study reported that 77.5% of a combined clinical and population-based sample could be placed into a concordant category (low ASD, low ADHD; 10.1%), (medium ASD, medium ADHD; 54.2%), and (high ASD, high ADHD; 13.2%). Conversely, two discordant classes emerged, one with higher scores on the ADHD traits (ADHD > ASD; 18.3%), and one with higher scores on the ASD trait (ASD > ADHD; 4.2%) [70]. These data and others

have led some to opine that it is not possible to determine if ADHD symptoms in ASD represent ASD, comorbid ADHD, or a separate condition entirely [71]. At this point, clinical judgment remains the deciding factor in determining which diagnosis is/are the most appropriate for a given individual.

The base rate of ADHD symptoms for children, adolescents, and adults with ASD has never been firmly established. Without base rate data on ADHD symptoms in ASD, we still do not know which ADHD symptoms and thresholds may enhance the predictive and discriminant validity of our ADHD diagnostic instruments.

## Treatment Considerations

There are well-researched and effective interventions that are available for both ASD and ADHD and vary according to the age of the child. For school-aged children with ASD, a focus on social, adaptive, and academic skills acquisition is recommended while in adulthood, the developmental of vocational and adaptive living skills becomes more integral to ASD management [72]. For school-aged children with ADHD, organizational interventions and parent/teacher training in contingency management is recommended while in adolescents and adults, the use of cognitive behavioral treatment is effective [73]. While no medications are FDA-approved for treating the core symptoms of ASD, the use of stimulants, atomoxetine, and alpha-2 agonist medications has FDA approval for managing ADHD [74]. Thus, there exists a wide range of effective treatment options for individuals with ASD and ADHD.

Despite these evidence-based options, we know far less about what constitutes an effective intervention for individuals with ASD+ADHD. This is surprising given the significant overlap between the two conditions as well as the increased impairment associated with the comorbid condition. The presence of ADHD in ASD is associated with increased ASD severity and a significantly increased risk for a third condition, especially anxiety and mood disorders [75]. Likewise, increasing ADHD severity, yet not increasing ASD severity, is associated with the number of additional comorbid psychiatric diagnoses in children with ASD [76•]. Below, we review what has been recently published about treating the comorbid condition, ASD+ADHD.

**Pharmacological** Eighty-six percent of youth with ASD+ADHD have been prescribed a medication for ADHD symptoms [77]. The presence of ADHD increases the risk for polypharmacy in ASD, an outcome observed in roughly one in four individuals with ASD treated with medications [78]. Psychiatrically referred youth with ASD often receive polypharmacy regimens (mean number of psychotropic medications =  $3 \pm 1.5$ ) [79]. Other population-based data have

indicated that psychotropic medication use (especially stimulants) in ASD occurs in just under 70% of the ASD population (40% are prescribed two or more psychotropic medications concurrently) and poly-pharmacy is associated strongly with age [80]. Over 85% of prescribers treating youth with ASD+ADHD routinely prescribed psychotropic medications for their patients. The most common target for psychotropic medication was aggression reduction with hyperactivity-impulsivity being second most frequently targeted [81].

The British Association for Psychopharmacology consensus guidelines do not recommend routine use of medications for managing core ASD symptoms. However, the group recommended the use of methylphenidate, atomoxetine, and guanfacine (in that order) for ADHD management in individuals with ASD [82]. A Cochrane review similarly concluded that methylphenidate reduces hyperactivity-impulsivity symptoms in youth with ASD (less robustly impacting inattention) yet has no impact upon core ASD symptoms [24•]. Compared to those prescribed a low dose, those prescribed a medium dose demonstrate more clinically significant improvements in ADHD symptoms [83].

While effective for managing ADHD symptoms in ASD, a meta-analysis [84] reported effect sizes associated with methylphenidate in ASD ( $ES = .67$ ) are lower than those reported for treating ADHD (without ASD) ( $ES = 1.03$ ) [85]. Moreover, methylphenidate is associated with higher rates of side effects such as social withdrawal, depression, and irritability when used in ASD [84]. Nonetheless, stimulants such as methylphenidate remain the front-line intervention for managing ADHD symptoms in ASD.

While the stimulants are recommended as a front-line pharmacological therapy for ADHD in ASD, atomoxetine and guanfacine also have demonstrated efficacy. For example, in individuals with ASD and an intellectual disability, atomoxetine was efficacious for reducing ADHD symptoms in 43% of the children [86]. Atomoxetine is sleep neutral in youth with ASD, neither negatively nor positively impacting parent-reported sleep levels in their child [87]. An 8-week trial of extended-release guanfacine in youth with ASD+ADHD resulted in significant reductions in parent-reported oppositional behaviors compared to placebo. Nonetheless, no differences were found between the placebo and extended-release guanfacine groups for parent-reported anxiety and sleep problems [88].

**Non-pharmacological** While some data suggest that the addition of a psychosocial intervention to medication treatment does not add incremental benefit [89], most professional practice parameters recommend a combination of medication and psychosocial interventions for managing ADHD in the context of ASD (see *Clinical Practice Pathways for Evaluation and Medication Choice for ADHD symptoms in ASD* [90]). Likewise, in addition to the use of medication, the British Association for Psychopharmacology consensus guidelines

recommended social communication interventions for children and adolescents with ASD and social skills training for adolescents with ASD [82]. While efficacious in ASD [51], social skills training has not proven efficacious or effective in children and adolescents with ADHD [91]. Similarly, the effects of parent training in children with ASD is moderated by the presence of ADHD with effects being observed more readily in children without ADHD [92]. Thus, it remains unclear the extent to which these ASD evidence-based interventions are efficacious in individuals with ASD+ADHD.

**Future Treatment Directions** Unlike depression and anxiety, digital health interventions (e.g., computer-assisted therapy, smartphone apps) are largely ineffective for ADHD and ASD [93]. Conversely, exercise has small to moderate effects on several aspects of cognition in individuals with ADHD and ASD, especially simple learning tasks and response inhibition; approximately 62% of individuals with ADHD and ASD respond favorably to exercise interventions [94]. Others have concluded that dietary interventions such as food additive exclusion diet, gluten-free/casein-free diet, and oligoantigenic diet are worthwhile to investigate and proposed microbiome–gut–brain axis as the putative mechanism [95]. Future research should continue to investigate dietary, exercise, and digital health interventions.

The presence of ASD symptoms in children with ADHD is associated with negative impacts upon the family quality of life including more negative emotional impacts and impacts upon the family and time. Parents of youth with ADHD and elevated ASD symptoms reported lower parenting self-efficacy than parents of youth with ADHD alone [96]. Similarly, a large population-based study in Denmark reported that having a child with ADHD, ASD+ADHD, and to a lesser

**Table 1** Suggested future research directions

1. How best to support the transition to adulthood
2. Including stakeholders more centrally in research topics
3. Analyzing moderators and mediators of treatment outcomes for ASD+ADHD
4. Girls with ASD, ADHD, and ASD+ADHD
5. Understanding subthreshold manifestations of ASD and ADHD
6. Understanding the sensory features in ASD and ADHD
7. Understanding developmental changes and trajectories underlying both phenotypes
8. Research Domain Criteria (RDoC) initiatives and dimensionality of ASD and ADHD
9. Early identification and intervention
10. Improving ecologically valid assessment (use of ecological momentary assessments)
11. Integrating technology into intervention designs

ASD autism spectrum disorder, ADHD attention deficit/hyperactivity disorder

**Table 2** Frameworks for understanding ASD and ADHD comorbidity

Model	Support	Recent citations
Comorbid ASD and ADHD is due to chance (random)	-	[16–22, 27, 28••]
Comorbid ASD and ADHD reflects sampling biases	-	[16, 18, 20, 28••]
One syndrome is an early manifestation of the other (precursor)	-	[16, 17, 27, 28••]
ASD and ADHD are not distinct entities but represent phenotypic variability of the same disorder (lumper)	~	[43, 52, 53]
ASD and ADHD share common vulnerabilities (e.g., genotype, environmental) (multifinality)	~	[31–36, 37•, 103]
ASD and ADHD are distinct and separate entities (splitter)	+	[9–14, 43, 52, 53, 56, 60, 103]
Comorbid ASD and ADHD represents a distinct subtype within a heterogeneous disorder (subgroup)	+	[69–71]
Development of one syndrome increases the risk for the other (potentiation)	+	[25–27, 28••, 44]

ASD autism spectrum disorder, ADHD attention deficit/hyperactivity disorder. Support levels (-, no support; ~, some support; +, support)

extent ASD is associated with increased risk for parental separation/divorce compared to typically developing children. By the age of 11, 50% of the ADHD families had separated, compared with 37% of ASD families and 25% of control families. Most marital dissolutions occurred when the proband was between ages 3 and 5 years old [97]. In children with ASD, having an older sibling is associated with a lower risk for ADHD, anxiety, and depression [98]. All of the above suggest that while family-level interventions have not been investigated in ASD+ADHD, it seems important to consider the family unit as a mechanism of change.

A large Medicaid claims study reported that significantly higher percentage of children with ASD (52%) received school-based mental health services compared to children with ADHD (8%) [99]. Nonetheless, children with ASD are perceived by their parents to have greater unmet occupational, physical, and speech therapy service needs than those with ADHD [100]. Future research should continue to investigate school-based interventions. (Please see Table 1 for additional research considerations which seem important to pursue in the next several years.)

## Conclusions

There are a variety of models that have been developed to explain the comorbidity of psychiatric disorders [101]. (Please see Table 2 for a list of several explanatory models and our opinions about how comorbid ASD and ADHD might be understood within each framework.) At this time, we believe that the accumulated knowledge base suggests that ASD and ADHD are related conditions yet sufficiently distinct to be considered separate disorders. Using an analogy, we consider ASD and ADHD to be “cousins” to each other, possibly even siblings. However, we do not believe that ASD and ADHD should be considered twins.

The comorbid state represents an “additive” profile of two conditions in our view. There are both quantitative and qualitative differences between the two conditions. Nonetheless, we agree with the call to investigate transdiagnostic, more dimensional considerations which might explain the etiological overlap and shared impairments and outcomes [102]. As indexed in Fig. 1, research interest in ASD, ADHD, and ASD+ADHD has grown exponentially over the past 20 years. We know far more about both ASD and ADHD yet continue to know less about the comorbid condition. Increases in prevalence for both conditions suggest that understanding the comorbid state will be a particularly important agenda for future researchers and clinicians.

## Compliance with Ethical Standards

**Conflict of Interest** Kevin M. Antshel reports a grant from Shire Pharmaceutical Company and personal fees from Arbor Pharmaceutical Company. Natalie Russo declares no potential conflicts of interest.

**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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