J Autism Dev Disord (2016) 46:2821–2829
DOI 10.1007/s10803-016-2814-5

COMMENTARY

Social (Pragmatic) Communication Disorder and Its Relation to the Autism Spectrum: Dilemmas Arising From the DSM-5 Classification

Yael Brukner-Wertman[1,2] · Nathaniel Laor[2,3,4] · Ofer Golan[1,2]

Published online: 26 May 2016

Abstract DSM-5 introduced two diagnoses describing neurodevelopmental deficits in social communication (SC); Autism Spectrum Disorder (ASD) and Social (Pragmatic) Communication Disorder (SPCD). These diagnoses are differentiated by Repetitive and Restricted Behaviors (RRB), required for an ASD diagnosis and absent in SPCD. We highlight the gaps between the research into SPCD and DSM-5's diagnostic criteria, and discuss the clinical implications of this diagnostic decision. We argue that DSM-5's demand for full manifestation of both SC and RRB axes when diagnosing ASD, prematurely forced a categorical view on the continual nature of the potentially dependent SC and RRB phenotypes. We conclude by highlighting the implications of this differential diagnostic decision on public health policies, designated therapy, and the need for further research regarding SPCD.

Keywords Social communication disorder · Autism spectrum disorder · Phenotype · Differential diagnosis · Treatment · Services

✉ Yael Brukner-Wertman
yaelbrukner@gmail.com

✉ Ofer Golan
ofer.golan@biu.ac.il

[1] Department of Psychology, Bar-Ilan University, Ramat-Gan, Israel

[2] Association for Children at Risk, Givat Shmuel, Israel

[3] Sackler Faculty of Medicine, Tel-Aviv University, Tel-Aviv, Israel

[4] Yale Child Study Center, New-Haven, CT, USA

Introduction

In his seminal paper, Kanner defined autism for the first time as a collection of characteristics that included deficits in social interaction and communication together with restricted and repetitive behaviors (RRB) (Kanner 1943). This triad of impairments was later used by the psychiatric nosology to characterize the diagnosis of autistic disorder, allowing for different levels of symptom expression at each category (American Psychiatric Association 1987). The recently published DSM-5 introduced several significant changes in this classification, collapsing the communication and social interaction categories into one category defined as social communication (SC) deficits, and describing, for the first time, two different diagnoses related to these characteristics: A diagnosis of Autism Spectrum Disorder (ASD), which includes RRB symptoms in addition to SC deficits, and a separate diagnosis of Social (Pragmatic) Communication Disorder (SPCD), which is characterized only by impairments in SC. The decision of the DSM-5 editors is associated with two issues which researchers have been examining for a long time: The first regards the continuous nature of autism, and the second regards the dependence/independence of the SC and RRB categories. The clinical and research implications of these changes in the diagnostic classification system are significant.

In this paper, we seek to examine the research findings addressing the questions of continuity of the autism spectrum and the independence of its two symptom categories. In addition, we examine the diagnostic and therapeutic implications of the diagnostic change. We conclude by discussing some potential effects of the separation of ASD from SPCD in DSM-5.

Continuity of the Autism Spectrum

In its early days, the diagnosis of autism required the complete and unequivocal existence of both SC and RRB symptoms, in what later came to be known as "classical autism" or "Kanner's autism". At the same time, Kanner himself reported behavioral characteristics among the parents of diagnosed children and first and second order relatives that resembled those of their children. Like the diagnosed children, these relatives were obsessive and disinterested in others, but their symptoms were partial or less severe (Kanner 1943). In other words, at the very early stages of the description of autism it was already clear that its expression goes beyond the clinical boundaries of the diagnosis.

The pioneering work of Folstein and Rutter (1977), dramatically changed the understanding of the etiology of ASD. It identified autism as a genetically based disorder, and provided further support for the association between partial autism symptomatology and the full-blown diagnosis. Folstein and Rutter's work highlighted the possible existence of partial expressions of autism in family members of diagnosed children. This characterization, later referred to as the Broader Autism Phenotype (BAP), describes unique personality attributes in the context of SC difficulties and RRB, presumably underlined by autism related genotype (Constantino et al. 2006; Rutter 2000). The existence of this milder form of autism symptomatology has been validated since then in many studies (e.g., Georgiades et al. 2013; Piven 2001; Constantino et al. 2006; Skuse et al. 2005; Bailey et al. 1998; Sucksmith et al. 2011).

The partial expression of autism symptomatology has also been studied in the general population, with no apparent familial pattern (Wing and Gould 1979). Wing and Gould's epidemiological study is a significant milestone in supporting the view of Kanner's autism as a disorder on a spectrum. This spectrum included different combinations of poor social interaction, poor verbal and nonverbal communication, and restricted repetitive behavioral patterns. It included both clinical impairments, and sub-clinical expressions in the general population. The existence of a genetically based spectrum of autistic traits, with unclear boundaries and various manifestations both in family members of diagnosed individuals and in the general population has since received extensive support (e.g., Chakrabarti et al. 2009; Robinson et al. 2011; Lundstrom et al. 2012; Skuse et al. 2005; Leekam et al. 2011; Happé and Frith 2006; Constantino and Todd 2003).

Over the years, the clinical diagnostic system had also recognized that partial manifestation of SC or RRB could in fact represent a clinical diagnosis on the autism spectrum. A partial manifestation was acknowledged by the DSM-IV classification and termed PDD-NOS (American Psychiatric Association 1994). In parallel, the ICD 10 classification termed it Pervasive Developmental Disorder, Unspecified, or Atypical Autism (World Health Organization 1992). Researchers highlighted the limitations of these undifferentiated diagnostic categories (Towbin 2005; Walker et al. 2004; Mandy et al. 2011), stressing the need to improve the definition of partial states of autism. Solutions in the form of characterization of the clinical autistic expressions of various subgroups as dimensions rather than as diagnostic categories have been proposed and discussed in the process of formulating the new classification of ASD in the DSM-5 (e.g., Volkmar et al. 2009). Eventually, the authors of DSM-5 adopted the view of autism as a spectrum. This is reflected in the name of the diagnosis, as well as in the requirement to define the level of support required by the individual (corresponding to the formerly defined "level of functioning"). Nonetheless, in practice, the criteria set in DSM-5, require significant impairments in both SC *and* RRB for a diagnosis of ASD. Hence, partial expressions of these categories have effectively been taken out of the ASD diagnosis. SC impairments with sub-clinical RRB manifestations should now be labeled SPCD, while RRB impairments with sub-clinical SC symptoms have no defined diagnostic category.

As a result, in setting the boundaries of the ASD diagnosis, the psychiatric nosology has forced a categorical division of attributes that appear to be on a continuum. The contrast between DSM-5 terminology and the continual nature of autistic symptomatology raises several issues, which we discuss below.

Independence of the SC and RRB Categories

A separate, though related issue regards the distinction between the SC and RRB categories. It has been assumed for many years that the behavioral symptoms of ASD have a common genetic, neurological and cognitive source. Wing and Gould (1979) found that all children with social impairments exhibited stereotypical repetitive behaviors and almost all of them showed deficiencies or abnormalities in language and symbolic activities, supporting the co-existence of these symptoms. Constantino et al. (2004) examined the factor structure of autistic traits using data from 226 child psychiatric patients with and without PDD, employing cluster analysis of data from the Autism Diagnostic Interview-Revised (Lord et al. 1994) and factor analysis of data from the Social Responsiveness Scale (Constantino 2002). Their findings revealed a single, continuously distributed, underlying factor of ASD traits. Similarly, Hoekstra et al. (2008), administered the Autism-

Spectrum Quotient (Baron-Cohen et al. 2001) to a group of typically developing adults. They found a high correlation between four of the five categories comprising the questionnaire (social skills, communication, attention switching and imagination) and proposed they should be clustered together. In a recent study, Frazier et al. (2014) administered the Social Responsiveness Scale 2 (SRS2: Constantino and Gruber 2012) to children and adults from the general population. In contrast to previous studies, factor analysis supported the separate existence of the SC and RRB categories, although the two were strongly correlated. It should be mentioned that correlation between SC and RRB are often much weaker in studies conducted only with diagnosed participants. However, such studies are likely to underestimate the magnitude of the association between the features of ASD (Murray et al. 2014).

The contrasting approach argues that SC and RRB are independent categories and are only weakly correlated. A population study, examining 3000 pairs of twins aged 7–9, found an average to low correlation between SC and RRB categories (Ronald et al. 2006). This study found that many of the children, who exhibited a high level of symptom severity in one domain, did not necessarily exhibit a high level of symptom severity in the other. For example, 59 % of the children exhibited only social impairments. These findings led the researchers to conclude that there is a separate genetic etiology for the two symptom categories, suggesting they should be seen as separate.

Similar findings emerged from the Twins Early Development Study (TEDS), which examined 189 children diagnosed with autism using the Development and Well-being Assessment (DAWBA; Goodman et al. 2000). In a statistical analysis of cross-twin cross-trait correlations, weak correlations were found between RRB and SC components, supporting the separation of the ASD categories (Dworzynski et al. 2009). Similarly, Mandy et al. (2014) used the Developmental, Diagnostic and Dimensional Interview (3Di; Skuse et al. 2004) to test 948 participants, some diagnosed with ASD, and some exhibiting BAP characteristics, according to the Autism Genetic Resource Exchange (AGRE; www.agre.org) criteria. Results indicated that among people with ASD the major symptoms can be divided into two domains that are only moderately related. Some studies have shown that people may have only one component of the SC and RRB dyad without the other (Mandy et al. 2011; Pooni et al. 2012; Ronald et al. 2011; Kolevzon et al. 2004; Robinson et al. 2012) These findings support a genetic, etiological, and phenomenological distinction between the components of ASD (Happe et al. 2006).

These conflicting findings stress the importance of further in-depth examination of the independence of the SC and RRB axes in neurodevelopmental disorders (Rutter 2014). Indeed, several studies discuss the manifestation of the SC and RRB clusters in various contexts, including, for example, genetic versus environmental influences on the diagnostic features (Robinson et al. 2012), the cognitive manifestations of the SC and RRB categories (Brunsdon and Happé 2014), and the likelihood of co-morbid affective conditions (Mazefsky et al. 2008). Mandy and Skuse (2008) have suggested that, at the current stage of knowledge, a separate classification of the SC and RRB categories should not be adopted, since their independence has not yet been sufficiently supported. Therefore, drawing conclusions about nosological changes may be premature. Leekam et al. (2011) suggested that even if the independence of SC and RRB can be proven, their combination under the autism diagnosis generates a specific pattern that should be studied separately.

In view of these unresolved questions, DSM-5 seems to have adopted both approaches: on the one hand, it requires both SC and RRB symptoms for a diagnosis of ASD, whereas on the other hand, it acknowledges an independent SC disorder (with no RRB component), by creating the SPCD diagnosis. Following this line of thought calls for a separate RRB disorder (with no SC component), which is missing in the DSM-5.[1] We believe that this fact represents the current state of inconclusive evidence regarding the relations between SC and RRB. Whereas such an open debate is welcomed within a research context, its implementation in a clinical diagnostic manual has serious implications for individuals who would have previously been diagnosed with PDD. Some of the clinical implications of this chosen diagnostic classification are discussed below.

Clinical Implications of ASD and SPCD Separation

In this section, we discuss two major clinical implications of the decision to create the new clinical diagnosis of SPCD which is separate from ASD: The provision of a positive diagnosis of SPCD, based on current DSM-5 criteria, and the provision of an optimal therapy to individuals with SPCD.

[1] Partial representation of the RRB category may be found in tic disorders that are often accompanied by social deficits due to the social isolation caused by the tic behaviors. However, the current definition of tic disorders fails to fully cover the RRBs manifested in ASD.

SPCD Diagnosis and Differential Diagnosis Difficulties

According to DSM-5, SPCD is characterized by pragmatic language deficits. However, clinicians who wish to assess pragmatic abilities face a shortage of tools, which reflect the complexity of the pragmatic features of language (Adams 2015). The one-on-one, structured nature of most diagnostic evaluations often fails to capture the complex and dynamic nature of the social environment (Perkins et al. 1999) where the pragmatic deficits of individuals with SPCD would be more evident. Hence, the diagnostic assessment procedure may be limited in picking up pragmatic impairments (Adams 2002; Volden et al. 2009). Moreover, assessing the components of pragmatics, such as turn taking, eye contact and the use of humor, often requires special awareness and sensitivity to cultural aspects (Carter et al. 2005). Consequently, there are very few standardized tests for the assessment of the pragmatic components of communication, compared to tests assessing the more structured components of language, namely vocabulary and syntax (Norbury and Sparks 2013; Adams 2015). Moreover, these tests are usually not part of the canonical psychological or psychiatric evaluations.

Currently, there are several instruments available for the assessment of pragmatic skills, which, alongside with their strength, have various limitations. These instruments include parent's and teacher's report questionnaires, such as the Children's Communication Checklist 2 (CCC-2, Bishop 2003) that are used as screening tools, rather than as diagnostic instruments (Norbury et al. 2004). There are tests of pragmatic skills, such as the pragmatic assessment modules of the Assessment of Comprehension and Expression (Adams et al. 2001); the Clinical Evaluation of Language Fundamentals (Semel et al. 2000); the Test of Language Competence (Wiig and Secord 1989); the Test of Pragmatic Language (2nd Ed.; Phelps-Terasaki and Phelps-Gunn 2007), and the Social Learning Developmental Test (Bowers et al. 2008). However, these are administered in a formal assessment framework and may therefore be limited in their ability to capture social situations that are open and varied in context (Adams 2002). The Targeted Observation of Pragmatics in Children's Conversation (TOPICC; Adams et al. 2012a) is a more direct observational tool, which benefits from a more ecologically valid assessment protocol, but it has so far received little attention in terms of its psychometric properties in general, and its diagnostic sensitivity and specificity in particular (Norbury 2014). Other observations such as the Pragmatic Rating Scale (PRS) (Landa et al. 1992) and the The Yale in vivo Pragmatic Protocol (Y*i*PP) (Schoen-Simmons et al. 2014) are more commonly used in research, than in clinical settings. Therefore, it seems that the clinical field still awaits standardized, valid, and sensitive instruments for the diagnostic assessment of SPCD (See Norbury 2014 and Adams 2015 for detailed reviews of existing instruments).

Another unanswered challenge is the provision of clear specificity criteria, allowing for the differential diagnosis of SPCD from other conditions. This is especially relevant when attempting to differentiate between SPCD and the category it may have previously belonged to-ASD. The differential diagnostic challenge focuses both on the SC cluster, which is supposedly common to both diagnoses, as well as on the RRB cluster, that is expected only in ASD. Allegedly, a diagnosis of SPCD is meant to represent the SC cluster of ASD and to be diagnosed by the same criteria. However, the description of SPCD's criteria uses a different jargon than that used to describe the SC cluster of ASD. It focuses almost exclusively on the subject of language pragmatics, which is not directly mentioned in the ASD description of SC. Furthermore, unlike ASD, the diagnosis of SPCD does not include a reference to the level of impaired pragmatic skills. This definition gap leaves the clinician to decide whether the SC impairments in SPCD should be as intensive as those observed in ASD, or, perhaps, milder.

Another challenge for the differential diagnoses between SPCD and ASD lies in the definition of the RRB cluster. While the diagnosis of ASD in DSM-5 requires at least 2 characteristics from the RRB cluster, a SPCD diagnosis should be considered "only if the developmental history fails to reveal any evidence of restricted/repetitive patterns of behaviors, interests, or activities" (American Psychiatric Association 2013; p.49). These criteria leave a gray area where individuals with SC deficits have *some* RRB features. These features may exist at a low level of severity, either quantitatively (i.e., fewer features) or qualitatively (e.g., inflexible thinking without evidence of behavioral repetition). However, if these features do not reach the 2 RRBs threshold, required for an ASD diagnosis, such individuals would be deprived of *both* ASD and SPCD diagnoses, according to DSM-5, despite their significant difficulties.

The gap described above calls for a more comprehensive diagnostic coverage of the spectrum which includes both ASD and SPCD. In addition, the differential diagnosis between the two should be backed by more robust assessment instruments (Leekam et al. 2011). In ASD, the evaluation of RRB relies on instruments such as the Autism Diagnostic Interview-Revised (ADI-R) (Lord et al. 1994) which was designed according to DSM-IV criteria, and may not acknowledge more subtle manifestations of RRB even within the ASD diagnosis. The second edition of the Autism Diagnostic Observation Schedule (ADOS-2) (Lord et al. 2012) combines SC and RRB symptoms together and may therefore fail to distinguish SPCD from ASD. Other

diagnostic instruments like the Diagnostic Interview for Social and Communication Disorders (DISCO) (Carrington et al. 2015) and the Dimensional and Diagnostic Interview (3di) (Mandy et al. 2012) have already demonstrated an ability to separately evaluate the SC and RRB axes of ASD, but have not yet reported on its use for the differential diagnosis of ASD and SPCD.

At present the diagnostic assessment of an individual suspected of having SPCD requires first ruling out the possibility of ASD. Only if the individual's impairments are below the threshold for a diagnosis of ASD will the question of SPCD be examined. Even then, in the absence of agreed-upon tools for assessing pragmatics, the diagnosis will likely be determined by clinical judgment (Grove and Meehl 1996). Such an approach may lead to SPCD becoming a residual category for 'not-quite' ASD, rather like the previous PDD-NOS category (Skuse 2012).

To conclude, if DSM-5 editors wish to back up the independence of SC and RRB by providing separate diagnostic labels, then clearer, well validated, assessment instruments may be needed.

Therapeutic Implications of the SPCD Diagnosis

The separation of SPCD from ASD is also likely to have major implications for therapeutic interventions. According to Ozonoff (2012), a diagnosis of SPCD will serve as an umbrella category for children with social communication impairments without stereotypical repetitive behaviors. It will encompass both children who were previously diagnosed with PDD–NOS and those who exhibited pragmatic impairments, but received no diagnosis in DSM-IV. Grouping children with pragmatic difficulties under one diagnostic category should help in constructing an appropriate therapeutic response to their specific needs, focusing on the social-communication domain (Adams et al. 2012b). However, a focus on social communication alone raises concerns that other deficits, which may be relevant to individuals with SPCD, are therapeutically overlooked.

For example, if the SC impairments in the SPCD diagnosis are similar to those found in ASD, it is possible that individuals with SPCD will have hampered abilities to assess the emotional and mental states of others (Baron-Cohen et al. 2008) as well as their own (Williams 2010). Such deficits have implications for broader psychological aspects that have been documented in ASD, such as impaired awareness of psychological aspects of the self (Lind 2010; Frith and Happe 1999) and deficits in one's sense of agency (Farley et al. 2010). More specifically, it has been suggested that a deficit in narrative skills (a.k.a., "story telling"), which is a diagnostic criterion in SPCD, may affect self-identity formation (Losh and Capps 2003). Besides its communicative role, the narrative ability organizes, reflects and reconstructs events and experiences in one's life and forms them into a coherent theme. Hence, the narrative ability is instrumental in creating the generalized meaning of who we are, out of the fragmented events of life (Ochs and Capps 1997). If the narrative ability shapes personal identity (Polkinghorne 1991), the difficulties individuals with SPCD have in this domain may affect their sense of self (Ochs and Capps 1997), which clearly bears important therapeutic implications.

Another aspect of adjustment that might not be addressed when the focus of treatment in SPCD is placed on SC alone is Emotion Regulation (ER). Adaptive ER functioning also requires socio-communicative skills, and appropriate comprehension of emotions (Weiss et al. 2014). Since individuals with SPCD may have difficulties in these aspects of functioning, they may be prone to ER difficulties, which are similar to those associated with ASD (Mazefsky et al. 2013).

Finally, focusing on SC alone risks that subtle RRB manifestations that might exist in SPCD, such as inflexible thinking or difficulties with change (Christ et al. 2010; Sumiyoshia et al. 2011), will be overlooked and untreated. Hence, an intervention which focuses on the SC component without considering additional RRB features may result in a specific rather than a global effect (Murray et al. 2014). On the other hand, existing ASD therapeutic interventions may not be fine-tuned enough for these subtle RRB characteristics. The appropriateness of evidence based interventions addressing RRBs in ASD, to the needs of individuals with SPCD should be further examined.

The above examples demonstrate why providing interventions for individuals with SPCD which focus solely on social communication skills may be insufficient. These arguments should be taken into consideration when setting up support packages for children with SPCD. In the meantime, it is suggested that children with SPCD who would have been diagnosed with PDD according to DSM-IV criteria, should receive interventions provided for individuals with ASD (Kim et al. 2014).

Concluding Remarks

Conceptualization of autism as a diagnostic entity has been a dynamic and ongoing process. More than seventy years after autism was first described, the diagnosis is still developing and is marked by the challenge of converting complex and heterogeneous phenomena into formal diagnostic criteria. The most recent decision, to separate social communication impairments into two diagnoses, based upon the presence or absence of RRB components, formed the basis for the introduction of SPCD as a new diagnostic category.

As discussed in this article, the new diagnosis is problematic in view of its controversial scientific foundation, both with respect to the characterization of autism as a continuum (or a spectrum) and with respect to the questionable independence of the SC and RRB dimensions. In addition, the vague nature of the diagnostic characteristics of the disorder and the absence of appropriate, valid, assessment measures are problematic. Finally, we raised questions, which should be addressed, regarding the kind of therapeutic support individuals with SPCD may need.

Despite these controversies and the still open questions concerning the new diagnosis, DSM-5 editors have decided to define SPCD as a diagnosis which is independent of ASD. Whereas a DSM-5 ASD diagnosis is offered to all *existing* individuals with a DSM-IV diagnosis of PDD-NOS, *new* referrals for diagnostic evaluation would no longer be able to rely on DSM-IV classification. These nosological decisions bring about significant policy changes for these individuals, whose special needs were previously acknowledged, and served, under the umbrella of ASD. Since the diagnosis of autism is associated with a well-established network of organizations engaged in public health, education, employment, economic benefits and research, excluding SPCD from this network raises the question of how will the official systems deal with it (Tanguay 2011). Hence, with the new diagnosis in place, individuals with SPCD are at risk of losing the public support which would have previously been granted to them. In addition, separating SPCD from ASD may deprive individuals with SPCD of the social support and the positive self-identity ("autistic pride") that this community provides (Davidson 2008; Bertilsdotter Rosqvist et al. 2013).

It still unclear how many individuals who would have been diagnosed with PDD, now fit a SPCD diagnosis (Norbury 2014). The current estimates vary from 6.2 % (Kim et al. 2014) to nearly 20 % (Wilson et al. 2013). In addition, preliminary reports on the prevalence of SPCD in a population sample are nearing 0.5 % (Kim et al. 2014). Since the actual prevalence of SPCD is still under research, the extent of the financial toll imposed on the public is not yet clear. With the current uncertainty around SPCD, public officials, nongovernmental organizations and professional disciplines may not assume responsibility for this new diagnosis. Hence, clinical decision making, as well as the financial burden of supporting individuals with SPCD may fall on their families.

Under these circumstances, the decision to define sequential features, whose interrelationship is unclear, as categorical diagnosis entities is problematic. In addition, in the absence of appropriate instruments for clear characterization of SPCD, beyond the rather general DSM-5 criteria, it is possible for children to be misclassified and unsuitably treated.

Despite these limitations, it is possible to understand the rationale behind the definition of ASD as a diagnosis incorporating both SC and RRB features and the resultant creation of the SPCD diagnosis. Furthermore, creation of the SPCD diagnosis finally highlights partial states of autism that DSM-IV has previously overlooked. These may now receive more attention both in research and in therapeutic intervention. Nonetheless, as long as the therapeutic needs of individuals with SPCD are not fully characterized, they should, in our opinion, continue to benefit from the public support packages allocated to individuals with ASD. We believe that a time period should be set for the examination of the needs of those marked by partial characteristics of autism. Only after such an examination, can a decision be made regarding the separation of the ASD and SPCD diagnoses and particularly regarding the therapeutic and support mechanisms required by individuals with SPCD.

Acknowledgments We are grateful to Dr. Raya Ariel for her useful comments on a previous version of this paper.

Funding The first and last authors received funding for this study by the Israeli Ministry of Science, Technology, and Space (Grant Number 10842).

Author Contributions The authors have each made substantial contributions to the creation of this commentary. The last author read and approved the final manuscript.

Compliance with Ethical Standards

Conflict of interest All authors declare that they have no conflict of interest.

Ethical Approval This article does not contain any studies with human participants or animals performed by any of the authors.

References

Adams, C. (2002). Practitioner review: The assessment of language pragmatics. *Journal of Child Psychology and Psychiatry, 438*, 973–987.

Adams, C. (2015). Assessment and intervention for children with pragmatic language impairment. In D. A. Hwa-Froelich (Ed.), *Social communication development and disorders* (pp. 141–170). New York: Psychology Press.

Adams, C., Cooke, R., Crutchley, A., Hesketh, A., & Reeves, D. (2001). *Assessment of comprehension and expression 6–11 (ACE 6–11)*. Windsor: NFER Nelson.

Adams, C., Lockton, E., Freed, J., Gaile, J., Earl, G., McBean, K., & Law, J. (2012a). The Social communication intervention project: A randomized controlled trial of the effectiveness of speech and language therapy for school age children who have pragmatic and social communication problems with or without autism spectrum disorder. *International Journal of Language and Communication Disorders, 47*(3), 233–244.

Adams, C., Lockton, E., Gaile, J., Earl, G., & Freed, J. (2012b). Implementation of a manualized communication intervention for school aged children with pragmatic and social communication needs in a randomized controlled trial: The social communication intervention project. *International Journal of Language and Communication Disorders, 47*(3), 245–256.

American Psychiatric Association. (1987). *Diagnostic and statistical manual of mental disorders 3rd edition revised (DSM-III-R)*. Washington: American Psychiatric Association.

American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders 4th edition revised (dsm-iv)*. Washington: American Psychiatric Association.

American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders 5th edition text revision (DSM-V)*. Washington: American Psychiatric Association.

Bailey, A., Palferman, S., Heavey, L., & Le Couteur, A. (1998). Autism: The phenotype in relatives. *Journal of Autism and Developmental Disorders, 28*(5), 369–392.

Baron-Cohen, S., Golan, O., Chakrabarti, B., & Belmonte, M. K. (2008). Social cognition and autism spectrum conditions. In C. Sharp, P. Fonagy, & I. Goodyer (Eds.), *Social cognition and developmental psychopathology* (pp. 29–56). Oxford: Oxford University Press.

Baron-Cohen, S., Wheelwright, S., Skinner, R., Martin, J., & Clubley, E. (2001). The autism spectrum quotient (AQ): Evidence from asperger syndrome/high functioning autism, males and females, scientists and mathematicians. *Journal of Autism and Developmental Disorders, 31*, 5–17.

Bertilsdotter Rosqvist, H., Brownlow, C., & O'Dell, L. (2013). Mapping the social geographies of autism—online and off-line narratives of neuro-shared and separate spaces. *Disability and Society, 3*(28), 367–379.

Bishop, D. V. M. (2003). *Children's communication checklist-2nd edition*. London: Psychological Corporation.

Bowers, L., Huisingh, R., & LoGiudice, C. (2008). *Social learning developmental test: elementry*. East Moline: Linguisystems.

Brunsdon, V. E., & Happé, H. (2014). Exploring the 'fractionation' of autism at the cognitive level. *Autism, 18*(1), 17–30.

Carrington, S., Leekam, S., Kent, R., Maljaars, J., Gould, J., Wing, L., & Noens, I. (2015). Signposting for diagnosis of autism spectrum disorder using the diagnostic interview for social and communication disorders (DISCO). *Research in Autism Spectrum Disorders, 9*, 45–52.

Carter, J. A., Lees, J. A., Murira, G. M., Gona, J., Neville, B. G., & Newton, C. R. (2005). Issues in the development of cross-cultural assessments of speech and language for children. *International Journal of Language & Communication Disorders, 40*(4), 385–401.

Chakrabarti, B., Dudbridge, F., Kent, L., Wheelwright, S., Hill-Cawthorne, G., Allison, C., & Baron-Cohen, S. (2009). Genes related to sex steroids, neural growth, and social–emotional behavior are associated with autistic traits, empathy, and Asperger syndrome. *Autism Research, 2*(3), 157–177.

Christ, S. E., Kanne, S. M., & Reiersen, A. M. (2010). Executive function in individuals with subthreshold autism traits. *Neuropsychology, 24*(5), 590–598.

Constantino, J. N. (2002). *The social responsiveness scale*. Los Angeles: Western Psychological Service.

Constantino, J. N., & Gruber, C. P. (2012). *The social responsiveness scale, 2nd edition (SRS-2)*. Los Angeles: Western Psychological Services.

Constantino, J. N., Gruber, C. P., Davis, S., Hayes, S., Passanante, N., & Przybeck, T. (2004). The factor structure of autistic traits. *Journal of Child Psychology and Psychiatry, 45*, 719–726.

Constantino, J. N., Lajonchere, C., Lutz, M., Gray, T., Abbacchi, A., McKenna, K., et al. (2006). Autistic social impairment in the siblings of children with pervasive developmental disorders. *The American Journal of Psychiatry, 163*(2), 294–296.

Constantino, J. N., & Todd, R. D. (2003). Autistic traits in the general population: Twin study. *Archives of General Psychiatry, 60*, 524–530.

Davidson, J. (2008). Autistic culture online: Virtual communication and cultural expression on the spectrum. *Social and Cultural Geography, 9*(7), 791–806.

Dworzynski, K., Happé, F., Bolton, P., & Ronald, A. (2009). Relationship between symptom domains in autism spectrum disorders: A population based twin study. *Journal of Autism and Developmental Disorders, 39*(8), 1197–1210.

Farley, A., López, B., & Saunders, G. (2010). Self-conceptualisation in autism: Knowing oneself versus knowing self-through-other. *Autism, 14*(5), 519–530.

Folstein, S. E., & Rutter, M. L. (1977). Infantile autism: A genetic study of 21 twin pairs. *Journal of Child Psychology and Psychiatry, 18*, 297–321.

Frazier, T. W., Ratliff, K. R., Gruber, C., Zhang, Y., Law, P. A., & Constantino, J. N. (2014). Confirmatory factor analytic structure and measurement invariance of quantitative autistic traits measured by the social responsiveness scale-2. *Autism, 18*(1), 31–44.

Frith, U., & Happe, F. (1999). Theory of mind and self-consciousness: What is it like to be autistic? *Mind and Language, 14*(1), 1–22.

Georgiades, S., Szatmari, P., Zwaigenbaum, L., Bryson, S., Brian, J., Roberts, W., et al. (2013). A prospective study of autistic-like traits in unaffected siblings of probands with autism spectrum disorder. *JAMA Psychiatry, 70*(1), 42–48.

Goodman, R., Ford, T., Richards, H., Gatward, R., & Meltzer, H. (2000). The development and well-being assessment: Description and initial validation of an integrated assessment of child and adolescent psychopathology. *Journal of Child Psychology and Psychiatry and Allied Disciplines, 41*, 645–655.

Grove, W. M., & Meehl, P. E. (1996). Comparative efficiency of informal (subjective, impressionistic) and formal (mechanical, algorithmic) prediction procedures: The clinical–statistical controversy. *Psychology Public Policy and Law, 2*(2), 293.

Happé, F., & Frith, U. (2006). The weak coherence account: detail-focused cognitive style in autism spectrum disorders. *Journal of Autism and Developmental Disorders, 36*, 5–25.

Happe, F., Ronald, A., & Plomin, R. (2006). Time to give up on a single explanation for autism. *Nature Neuroscience, 9*, 1218–1220.

Hoekstra, R. A., Bartels, M., Cath, D. C., & Boomsma, D. I. (2008). Factor Structure, reliability and criterion validity of the autism-spectrum quotient (AQ): A study in dutch population and patient groups. *Journal of Autism and Developmental Disorders, 38*, 1555–1566.

Kanner, L. (1943). Autistic disturbances of affective contact. *The Nervous Child, 2*, 217–250.

Kim, Y. S., Fombonne, E., Koh, Y., Kim, S., Cheon, K., & Leventhal, B. L. (2014). Developmental disorder and DSM-5 autism spectrum disorder prevalence in an epidemiologic sample. *Journal of the American Academy of Child and Adolescent Psychiatry, 53*(5), 500–508.

Kolevzon, A., Smith, C. J., Schmeidler, J., Buxbaum, J. D., & Silverman, J. M. (2004). Familial symptom domains in monozygotic siblings with autism. *American Journal of Medical Genetics Part B Neuropsychiatric Genetics, 129*(1), 76–81.

Landa, R., Piven, J., Wzorek, M., Gayle, J., Cloud, D., Chase, G., et al. (1992). Social language use in parents of autistic individuals. *Psychological Medicine, 22*, 246–254.

Leekam, S. R., Prior, M. R., & Uljarevic, M. (2011). Restricted and repetitive behaviors in autism spectrum disorders: A review of research in the last decade. *Psychological Bulletin, 137*(4), 562–593.

Lind, S. E. (2010). Memory and the self in autism: A review and theoretical framework. *Autism, 14*(5), 430–456.

Lord, C., Rutter, M., DiLavore, P. C., Risi, S., Gotham, K., & Bishop, S. (2012). *Autism diagnostic observation schedule: ADOS-2*. Los Angeles, CA: Western Psychological Services.

Lord, C., Rutter, M., & Le Couteur, A. (1994). Autism diagnostic interview-revised: A revised version of a diagnostic interview for caregivers of individuals with possible pervasive developmental disorders. *Journal of Autism and Developmental Disorders, 24*(5), 659–685.

Losh, M., & Capps, L. (2003). Narrative ability in high-functioning children with autism or asperger's syndrome. *Journal of Autism and Developmental Disorders, 33*(3), 239–251.

Lundstrom, S., Chang, Z., Rastam, M., Gillberg, C., Larsson, H., Anckarsater, H., & Lichtenstein, P. (2012). Autism spectrum disorders and autistic like traits, similar etiology in the extreme end and the normal variation. *Archives of General Psychiatry, 69*(1), 46–52.

Mandy, W., Charman, T., Gilmour, J., & Skuse, D. (2011). Toward specifying pervasive developmental disorder—not otherwise specified. *Autism Research, 4*(2), 121–131.

Mandy, W., Charman, T., Puura, K., & Skuse, D. (2014). Investigating the cross-cultural validity of *DSM-5* autism spectrum disorder: Evidence from Finnish and UK samples. *Autism, 18*(1), 45–54.

Mandy, W. P., Charman, T., & Skuse, D. H. (2012). Testing the construct validity of proposed criteria for DSM-5 autism spectrum disorder. *Journal of the American Academy of Child and Adolescent Psychiatry, 51*(1), 41–50.

Mandy, W. P., & Skuse, D. H. (2008). Research review: what is the association between The social-communication element of autism and repetitive interests, behaviours and activities? *Journal of Child Psychology and Psychiatry, 49*, 795–808.

Mazefsky, C. A., Goin-Kochel, R. P., Riley, B. P., & Maes, H. H. (2008). Genetic and environmental influences on symptom domains in twins and siblings with autism. *Research in Autism Spectrum Disorders, 2*(2), 320–331.

Mazefsky, C. A., Herrington, J., Siegel, M., Scarpa, A., Maddox, B. B., Scahill, L., & White, S. W. (2013). The role of emotion regulation in autism spectrum disorder. *Journal of the American Academy of Child and Adolescent Psychiatry, 52*(7), 679–688.

Murray, A. L., McKenzie, K., Kuenssberg, R., & O'Donnell, M. (2014). Are we under-estimating the association between autism symptoms?: The importance of considering simultaneous selection when using samples of individuals who meet diagnostic criteria for an autism spectrum disorder. *Journal of Autism and Developmental Disorders, 44*(11), 2921–2930.

Norbury, C. F. (2014). Practitioner review: Social (pragmatic) communication disorder conceptualization, evidence and clinical implications. *Journal of Child Psychology and Psychiatry, 55*(3), 204–216.

Norbury, C. F., Nash, M., Baird, G., & Bishop, D. (2004). Using a parental checklist to identify diagnostic groups in children with communication impairment: A validation of the children's communication checklist–2. *International Journal of Language and Communication Disorders, 39*, 345–364.

Norbury, C. F., & Sparks, A. (2013). Difference or disorder? Cultural issues in understanding neurodevelopmental disorders. *Developmental Psychology, 49*, 45–58.

Ochs, E., & Capps, L. (1997). Narrative authenticity. *Journal of Narrative and Life History, 7*, 83–89.

Ozonoff, S. (2012). Editorial: DSM-5 and autism spectrum disorders–two decades of perspectives from the JCPP. *Journal of Child Psychology and Psychiatry, 53*(9), e4–e6.

Perkins, L., Crisp, J., & Walshaw, D. (1999). Exploring conversation analysis as an assessment tool in aphasia: The issue of reliability. *Aphasiology, 13*, 259–281.

Phelps-Terasaki, D., & Phelps-Gunn, T. (2007). *Test of pragmatic language* (2nd ed.). Austin: Pro-Ed.

Piven, J. (2001). The broad autism phenotype: a complementary strategy for molecular genetic studies of autism. *American Journal of Medical Genetics, 105*(1), 34–35.

Polkinghorne, D. E. (1991). Narrative and self-concept. *Journal of Narrative and Life History, 1*(2&3), 135–153.

Pooni, J., Ninteman, A., Bryant-Waugh, R., et al. (2012). Investigating autism spectrum disorder and autistic traits in early onset eating disorder. *International Journal of Eating Disorders, 45*(4), 583–591.

Robinson, E. B., Koenen, K. C., McCormick, M. C., Munir, K., Hallett, V., Happé, F., et al. (2011). Evidence that autistic traits show the same etiology in the general population and at the quantitative extremes (5%, 2.5%, and 1%). *Archives of General Psychiatry, 68*, 1113–1121.

Robinson, E. B., Koenen, K. C., McCormick, M. C., Munir, K., Hallett, V., Happé, F., et al. (2012). A multivariate twin study of autistic traits in 12-year-olds: Testing the fractionable autism triad hypothesis. *Behavior Genetics, 42*(2), 245–255.

Ronald, A., Happé, F., Bolton, P., Butcher, L. M., Price, T. S., Wheelwright, S., et al. (2006). Genetic heterogeneity between the three components of the autism spectrum: a twin study. *Journal of the American Academy of Child and Adolescent Psychiatry, 45*(6), 691–699.

Ronald, A., Larsson, H., Anckarsäter, H., & Lichtenstein, P. (2011). A twin study of autism symptoms in Sweden. *Molecular Psychiatry, 16*, 1039–1047.

Rutter, M. (2000). Genetic studies of autism: from the 1970s into the millennium. *Journal of Abnormal Child Psychology, 28*(1), 3–14.

Rutter, M. (2014). Addressing the issue of fractionation in autism spectrum disorder: A commentary on Brunsdon and Happé, Frazier, Hobson, and Mandy et al. *Autism, 18*, 55–57.

Schoen-Simmons, E., Paul, R., & Volkmar, F. (2014). Assessing pragmatic language in autism spectrum disorder: The yale in vivo pragmatic protocol. *Journal of Speech Language and Hearing Research, 57*, 2162–2173.

Semel, E., Wiig, E., & Secord, W. (2000). *Clinical evaluation of language fundamentals–3rd edition (UK)*. Hove: Psychological Corporation.

Skuse, D. H. (2012). DSM-5's conceptualization of autistic disorders. *Journal of the American Academy of Child and Adolescent Psychiatry, 51*(4), 344–346.

Skuse, D. H., Mandy, W. P., & Scourfield, J. (2005). Measuring autistic traits: Heritability, reliability and validity of the social and communication disorders checklist. *The British Journal of Psychiatry, 187*(6), 568–572.

Skuse, D. H., Warrington, R., Bishop, D., Chowdhury, U., Lau, J., Mandy, W., & Place, M. (2004). The developmental, dimensional and diagnostic interview (3di): A novel computerized assessment for autism spectrum disorders. *Journal of the American Academy of Child and Adolescent Psychiatry, 43*(5), 548–558.

Sucksmith, E., Roth, I., & Hoekstra, R. A. (2011). Autistic traits below the clinical threshold: Re-examining the broader autism phenotype in the 21st. *Neuropsychology Review, 21*(4), 360–389.

Sumiyoshia, C., Kawakubob, Y., Sugab, M., Sumiyoshie, T., & Kasaib, K. (2011). Impaired ability to organize information in individuals with autism spectrum disorders and their siblings. *Neuroscience Research, 69*, 252–257.

Tanguay, P. E. (2011). Commentary: Autism in DSM-5. *American Journal of Psychiatry, 168*(11), 1142–1144.

Towbin, K. E. (2005). Pervasive developmental disorder not otherwise specified. In F. R. Volkmar, R. Paul, A. Klin, & D. J. Cohen (Eds.), *Handbook of autism and pervasive developmental disorders* (pp. 165–200). New York: Wiley.

Volden, J., Coolican, J., Garon, N., White, J., & Bryson, S. (2009). Pragmatic language in autism spectrum disorder: Relationships to measures of ability and disability. *Journal of Autism and Developmental Disorders, 39*, 388–393.

Volkmar, F., State, M., & Klin, A. (2009). Autism and autism spectrum disorders: diagnostic issues for the coming decade. *Journal of Child Psychology and Psychiatry, 50*(1–2), 108–115.

Walker, D. R., Thompson, A., Zwaigenbaum, L., Goldberg, J., Bryson, S. E., et al. (2004). Specifying PDD-NOS: A comparison of PDD-NOS, Asperger syndrome, and autism. *Journal of the American Academy of Child and Adolescent Psychiatry, 43*, 172–180.

Weiss, J. A., Thomson, K., & Chan, L. (2014). A systematic literature review of emotion regulation measurement in individuals with autism spectrum disorder. *Autism Research, 7*(6), 629–648.

Wiig, E., & Secord, W. (1989). *Test of language competence–expanded*. Hove: Psychological Corporation.

Williams, D. (2010). Theory of own mind in autism, evidence of a specific deficit in self-awareness? *Autism, 14*(5), 474–494.

Wilson, C. E., Gilan, N., Spain, D., Robertson, D., Roberts, G., et al. (2013). Comparison of ICD-10R, DSM-IV-TR and DSM-5 in an adult autism spectrum disorder diagnostic clinic. *Journal of Autism and Developmental Disorders, 43*, 2515–2525.

Wing, L., & Gould, J. (1979). Severe impairments of social interaction and associated abnormalities in children: Epidemiology and classification. *Journal of Autism and Developmental Disorders, 9*(1), 11–29.

World Health Organization. (1992). *International statistical classification of diseases and related health problems (ICD-10)*. Geneva: WHO.