J Autism Dev Disord (2009) 39:1560–1567
DOI 10.1007/s10803-009-0798-0

ORIGINAL PAPER

Qualitative or Quantitative Differences Between Asperger's Disorder and Autism? Historical Considerations

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Published online: 23 June 2009

Abstract The histories of autism and Asperger's Disorder (AD), based on original contributions by Kanner and Asperger, are reviewed in relation to DSM-IV diagnostic criteria. Their original articles appear to have influenced the distinction between AD and autism made in the DSM-IV. Based on up-to-date empirical research, however, it appears that AD and autism are not qualitatively distinct disorders, but are different quantitative manifestations of the same disorder. The differences between AD and autism may be a function of individual variability in these areas, not the manifestation of qualitatively distinct disorders. The DSM-IV criteria for AD and autism need to be considered with their historical developments, and based on empirical evidence, the DSM-IV diagnostic criteria may be subject to critical review.

Keywords Autism · Asperger's Disorder · History

In 1943, Leo Kanner described a disorder similar to, but distinct from childhood schizophrenia. This disorder, true to its name, was referred to as autism, and was included in the Diagnostic and Statistical Manual of Mental Disorders, 3rd Ed. (DSM-III). In 1981, Hans Asperger's account of Autistic Psychopathy (1944) was introduced to North America (Frith 1991). This led to the apparent discovery of a new disorder similar to autism. Named after Asperger, it was included in the DSM-IV as a qualitatively distinct disorder from autism. Since its inclusion in DSM-IV, however, there has been a great deal of research examining whether Asperger's Disorder (AD) should be considered a disorder on an autistic spectrum, or whether its characteristics warrant it as related to but distinct from autism.

Asperger's Disorder and autism are highly related, and are both considered Pervasive Developmental Disorders (PDD). Determining whether a qualitative distinction exists between AD and autism specifically, and autism generally, has several clinical and research implications. If both disorders are distinct they may also differ in etiology, which may impact early identification and biological markers for the disorder(s), or prevention through physiological means. In addition, prognoses and interventions would likely differ between Asperger's and autism if they were qualitatively distinct (Macintosh and Dissanayake 2004).

Leo Kanner

In his landmark paper in 1943, *Autistic Disturbances of Affective Contact*, Leo Kanner described 11 children who demonstrated obsessive and repetitive behaviors, social deficits, and echolalia. Although the children's behaviors were somewhat consistent with childhood schizophrenia, they seemed different from other recorded incidences of childhood schizophrenic patients. Children with schizophrenia demonstrated average development prior to the onset of their disorders, yet, according to Kanner, his patients seemed to exhibit "extreme aloneness" from birth (p. 248) (although research at present has not identified autism in children at birth, factors in the first year of life, such as intense interest toward objects, has been shown retrospectively in children with autism, i.e., Maestro et al. 2006). In addition, Kanner's patients seemed peculiar because they related obsessively with objects but avoided any affective contact with people. Relating with people

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was tolerated by the children by focusing on the person's hand or foot as a detached object, with social reciprocity apparently lacking entirely.

Kanner's descriptions of the 11 children in his article appear consistent with the present diagnosis of autism, and his paper was arguably influential on the development of the diagnostic criteria in the long run. Rutter, whose writings more directly impacted the development of diagnostic criteria in DSM-III, frequently cited Kanner's writings well over 30 years after they were published (1978; Rutter and Schopler 1987). Nine years after Kanner's seminal paper, the first edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM) was published (APA 1952), and the second edition published in 1968 (APA). In these first two editions of the DSM, autism was not categorized as a disorder. However, elements of modern-day diagnostic criteria for autism were present in the criteria for schizophrenic disorders, most notably Schizoid Personality and Schizophrenia—Childhood Type. Under Schizoid Personality, these manuals establish "autistic thinking" (i.e., coldness, emotional detachment, aloofness) as criteria (p. 42, 1968; p. 35, 1952). DSM-II describes Schizophrenia—Childhood Type as a condition manifested by "autistic, atypical, and withdrawn behavior" (p. 35, 1968). The inclusion of autistic-type thinking and behavior, as we know it today, were considered schizophrenic symptoms well before the onset of DSM (Frith 1991), which may explain the time lag between the first published clinical description of the disorder in 1943 and its inclusion in DSM-III in 1980.

Autistic disorder first appeared as a disorder distinct from schizophrenia in the DSM-III, under the name Infantile Autism (APA 1980), which was later changed to Autism in the revised DSM-III in 1987. The a uthors indicated that some camps still considered autism a schizophrenic disorder, and that infantile autism was the earliest form of schizophrenia. Research in family studies, however, suggested that both were distinct disorders. The DSM-III criteria for infantile autism included deficits in the development of language, atypical patterns of speech when present, lack of responsiveness to others, unusual attachments and interests, and resistance to change, with an onset before 30 months of age. These criteria are remarkably similar to the diagnostic criteria for autism in the DSM-IV, which are qualitative impairments in social interactions and communication, and restricted repetitive and stereotyped patterns of behavior, interests, and activities, with onset prior to age three (APA 2000).

Kanner provided many clinical descriptions in his study that are congruent with DSM-III and DSM-IV criteria, even in terminology. The title of Kanner's publication, *Autistic Disturbances of Affective Contact*, provided the name autism for the disorder. The terms "autistic" and "autism" are derived from the Greek word "autos", meaning "self", and was used to describe some characteristics of schizophrenia (Frith 1991). In the context of autism as a disorder, it is meant to convey an apparent disconnect from the social world outside of themselves. Kanner described some of his child patients, "like in a shell", "acting as if people weren't there", and "perfectly oblivious to everything about him" (1943, p. 242), which seems to describe the apparent self-absorption associated with the term "autistic". Kanner also described features of communicative impairment; "As far as the communicative functions of speech are concerned, there is no fundamental difference between the eight speaking and the three mute children" (p. 243); and repetitive behavior such as echolalia; "language was deflected in a considerable measure to a self-sufficient, semantically and conversationally valueless or grossly distorted memory exercise…When sentences are finally formed, they are for a long time mostly parrot-like repetitions of heard word combinations." (p. 243) The DSM-IV contains four specific symptoms in each of the diagnostic categories of impairments in social interaction and communication, and stereotyped patterns of behavior, totaling twelve symptoms. A careful reading of Kanner's article will uncover reference to each of those twelve symptoms in his clinical descriptions. Kanner also noted in his cases that the children's development was stable, not regressive like in childhood onset schizophrenia. This is reflected in the diagnostic criteria of onset of symptoms prior to 30 months in DSM-III, or before 3 years in DSM-IV. The only diagnostic criteria for autism that cannot be related back to Kanner's article involves differential diagnosis, in that the symptoms are not better accounted for by Rett's Disorder or Childhood Disintegrative Disorder, and there is not much Kanner could have contributed in that respect since those disorders were not yet recognized.

Although Kanner describes at length the deficits associated with "autistic disturbances", he does point out some apparent and potential strengths of the children. He noted the children displayed good cognitive potential and strong episodic memory, and that those who spoke demonstrated an excellent vocabulary. The relative strengths that these children demonstrated in memory and vocabulary (or in DSM-type language, the lack of deficits in these areas) were not incorporated into the DSM-III criteria for autism.

Hans Asperger

In 1944, Hans Asperger published his account, *Autistic Psychopathy in Childhood*, which is remarkably similar to Kanner's publication a year earlier (Frith 1991). In it, Asperger describes in his children the same deficits of

social interaction and stereotyped behavior that Kanner described. In Frith's translation of Asperger's article (1991), it is noted that "relations with the outside world are extremely limited" (p. 42), that a patient "could not engage in the lively reciprocity of normal social interaction" (p. 45), and that the children had "abnormal fixations" with objects (p. 81). Like Kanner, Asperger also noted that the autistic differed from the schizophrenic in that cognitive and social development was not regressive but stable in the former. Qualitative impairments in communication, unlike Kanner's publication, are not apparent in Asperger's work. In fact, Asperger reported some extraordinary abilities in mathematics and relative strengths in some communicative skills, noting that some children spoke like adults, though in a rigid stereotyped fashion.

Although Asperger's description of autistic disorder may have been recognized in Germany, his work was not introduced to North America until 1981, by Wing. This came a staggering 37 years after his article was first published, and 1 year after the publication of the DSM-III. Despite a strong interest in the disorder in North America, his original article was not translated into English until 10 years after it was introduced (Frith 1991). In this respect, it is astounding to think that Asperger's and Kanner's articles, though nearly identical in description, were not directly compared until nearly 50 years after their publication dates! In fact, Asperger published 1 year after Kanner but may have been engaged in the investigation of autism well before him (Lyons and Fitzgerald 2007). Their accounts of their patients were virtually parallel in both description and timing, yet culminated in two distinct, though similar disorders in the DSM-IV. This may be in part related to the fact that they were introduced to the English-speaking world decades apart.

The relative focus on patients' verbal strengths in Asperger's report seems to have had a direct impact on the DSM-IV criteria for Asperger's Disorder (AD). The diagnostic criteria for AD differs most notably from autism in that communication delay is not present. Interestingly, similar verbal strengths were identified by Kanner in some, but not all of his cases (1943). Yet this distinction was not made in the DSM-III (before Asperger's work reached North America), probably due to the orientation of the DSM to focus on deficits rather than strengths. When Asperger's cases were introduced to North America, they may have been perceived as novel because they were recognized much later than Kanner's work, and the orientation of Asperger's article focused more so on children's strengths. In retrospect, it may be that a new disorder or a variant of autism was not introduced to North America as was thought at the time. Rather, the autism Hans Asperger discussed may have been the same autism that Kanner referred to. Wing, who first introduced Asperger's work to North America, indicated that there was no evidence that any qualitative differences existed between the two disorders, but this assertion was largely ignored (2000).

These apparent, though restricted verbal strengths from both Kanner's and Asperger's accounts seem consistent with High Functioning Autism (HFA) and/or Asperger's Disorder (according to DSM), which at present is a topic of diagnostic controversy and the central theme of this paper. In DSM-IV, AD was distinguished from autism, but apart from the absences of communicative impairment and cognitive delay, Asperger's Disorder is virtually identical to autism diagnostically. HFA does not appear in the DSM as a diagnosis per se, but is considered to be autism absent of cognitive delay, or in other words, an IQ above 70 (Ghaziuddin and MountainKimchi 2004). The distinction, if any, between HFA and AD is blurry.

AD/HFA Research

Research publications since 2000 comparing AD and HFA were searched using PsychINFO and reference citations. Inclusion of articles were restricted to cognitive, social, and language/communication domains, which appear to be the central distinguishing features of autism and AD from a DSM-IV framework. Since the introduction of AD to North America in 1981, there has been substantial research (and resulting controversy) regarding differences, if any, between AD and HFA. Early research in this area suggested that children with AD do exhibit greater verbal ability than those with HFA (i.e., Ozonoff et al. 1991). There were, however, some problems with much of the research. Many studies that found qualitative differences between AD and HFA were conducted before formalized diagnostic procedures were created for AD (i.e., DSM-IV). When studies were replicated using DSM-IV criteria, the differences were often no longer present. Even when using DSM-IV criteria in comparison studies, finding differences between AD and HFA groups did not necessarily mean that both disorders were distinct (Macintosh and Dissanayake 2004). For example, DSM-IV distinguishes HFA from AD as having a presence of language delay. Therefore, if a researcher partitions participants into HFA and AD groups based on DSM-IV criteria, the HFA group will necessarily present with language delay relative to the AD group. Finding group differences in verbal ability between the groups should not be surprising, nor does it necessarily present evidence of a distinction between HFA and AD, because language ability is the basis of how they were partitioned. In other research, AD and HFA participants were not matched for IQ, and patients with higher IQ tended to receive a diagnosis of AD versus HFA. In addition, the majority of studies evaluated participants in

childhood or early adolescence; few studies evaluated late adolescence and adulthood, and those that did tended to find no differences between AD and HFA groups (Howlin 2003).

Taking the DSM-IV criteria of Asperger's at face value, a delay in language or cognitive functioning (IQ < 70) should not be present in an AD diagnosis. In HFA, cognitive delay should also be absent (Ghaziuddin and MountainKimchi 2004), but a general delay in language should be present. Therefore, matching for cognitive functioning, patients with AD should perform significantly better in language functioning than those with autism, *and* on par with controls. However, research in this area indicates that this appears to not be the case. Preschool children with AD were found to demonstrate better language comprehension (Iwanaga et al. 2000) and language skills (Szatmari et al. 2003) than those with HFA. This result, however, is expected because patients with AD are distinguished from autism largely by virtue of their language ability anyway, based on DSM-IV criteria. The preschool children in these studies, however, were not compared to controls, which would help determine whether a language delay was absent or present. Shriberg et al. (2001) found that patients with AD had more voluble speech than those with HFA. However, AD participants were no different than HFA, and significantly lower than controls, in articulation distortion errors and inappropriate or uncodable utterances. Patients with AD appear on par with HFA groups in cognitive and some language abilities, but significantly lower than controls in language expression and comprehension, especially in studies that use older children, adolescents, and adults as the sample (Howlin 2003).

Differences in cognitive capacity between AD and autism may not be indicative of qualitative distinctions between the disorders. Mayes and Calhoun (2004) compared children with AD and autism on IQ and other variables, such as frequency of autistic symptoms and social concerns. Although they found that lower IQ was related to more autistic symptoms and social problems, these effects were nullified when IQ and age were statistically removed. This suggests that autistic symptoms and social problems were attributable to IQ and age, rather than differences intrinsic to autism. This result seems consistent with research suggesting that differences between AD and HFA diminish as children get older (Howlin 2003; Szatmari et al. 2000).

Although full-scale IQs of children with AD and HFA are similar in many studies, their intellectual profiles appear to differ on the whole. Ghaziuddin and MountainKimchi (2004) compared the intellectual profiles of AD and HFA patients using the Wechsler scales. They found that patients with AD had verbal IQs (VIQ) almost 11 standard score points higher than performance IQs (PIQ), which difference was highly significant. In addition, the VIQ of patients with AD were usually higher than the VIQs of patients with HFA. The IQ profiles of HFA patients were varied, with an equal amount showing relative strengths in VIQ and PIQ. In addition, the patients with AD performed significantly better on the verbal subtests Arithmetic, Information, and Vocabulary than the HFA patients, whereas there were no difference on the nonverbal subtests Block Design and Object Assembly. Similar results were described by Koyama et al. (2007), who found that children with AD performed significantly better on VIQ and the verbal subtests Vocabulary and Comprehension. These results suggest that patients diagnosed with AD are likely to perform better on verbal tasks than performance tasks, and that their verbal abilities tend to be stronger than the verbal abilities of HFA. Again, this is not surprising because AD and HFA groups were already partitioned by language ability as part of the diagnostic process. Ghaziuddin and Mountain-Kimchi note, however, that both AD and HFA groups had individuals whose profiles were typical of the other group. This suggests that although verbal ability may be greater in AD as a whole, that the diagnostic utility of VIQ and PIQ for differentiating the disorders is questionable.

Another problematic area in distinguishing AD from HFA is the diagnostic criteria for Asperger's of a lack of general delay in language, because language delay is not clearly nor objectively defined. In addition, many people diagnosed with AD present with language delay later in life; conversely, many with autism do not (Macintosh and Dissanayake 2004). Bennett et al. (2008) evaluated the use of structural language impairment for discriminating between populations on the autism spectrum. Structural language impairment refers to deficits in grammar or syntax, not semantic or pragmatic uses of language. This is more specific and measurable than general language delay noted in DSM-IV. Participants were tested for language skills at ages 4–6 and again 2 years later. Subsequently, children's autistic symptoms and adaptive functioning were assessed once every 2 years until ages 15–17. In this way, the researchers were able to evaluate the long-term outcomes of the participants in relation to their language skills early in life. The researchers found that structural language impairment at ages 6–8 was more predictive of autistic symptoms and adaptive dysfunction in adolescence than was a diagnosis of AD or autism based on DSM-IV criteria. This result suggests that impaired use of syntax and grammar, at ages 6–8, is a better predictor of autistic tendencies than general delay in language by age 3, as reported retrospectively by parents. In addition, structural language impairment at ages 6–8 was more predictive of autistic behavior and adaptive functioning than the same impairments at ages 4–6, suggesting a "catch-up" effect as

children reach primary school age. This catch-up may make an analysis of language delay prior to age 3 meaningless (Woodbury-Smith et al. 2005).

Howlin (2003) obtained similar findings in her study, where adults with PDD were separated into an autism group (early language delay present), and an AD group (early language delays absent), and were matched for age, nonverbal IQ, and gender. Howlin found that there were no significant differences on the Autism Diagnostic Inventory—Revised (ADI-R) between the autism and AD groups, suggesting no differences in autistic symptomology based on the presence or absence of early language delay. This result indicates that early language development may not be a good indicator of autistic symptoms later in life, and that differences between autism and AD are ambiguous. However, Matson and Wilkins (2008) note that the inclusion of an instrument specific to AD would have provided a broader view of differences between the autism and AD groups.

Another discriminating factor between AD and autism in DSM-IV is onset of language delay. A child will be given an AD diagnosis only if there is no language delay *prior* to a certain age. For example, a child who used single words by age 2 years will meet onset criteria for AD. However, a diagnosis of autism takes precedence where there is a conflict of criteria, such as if that child used single words by age 2 years, but did not use communicative phrases by age 3 years. This approach is problematic in that diagnoses are slanted toward autism on the basis of development. In addition, information regarding a child's language development is usually obtained retrospectively, which presents with a number of issues. Dates of developmental milestones may not be remembered, minor developmental delays may be inflated, and present diagnostic realities may distort parents' memories of their children's development (Woodbury-Smith et al. 2005).

The supposition that children with AD would be free of language delay is somewhat mysterious, as Asperger described language and communication dysfunction amongst his participants (Frith 1991). This apparent absence of language delay in early years may be tied to the supposed absence of qualitative impairments in communication, which also distinguishes AD from autism in DSM-IV. However, research has been unequivocal in demonstrating deficits in social communication for AD (Howlin 2003; Woodbury-Smith et al. 2005), and Bennett et al.'s findings (2008) suggest that communication impairment is in fact present for AD. With recent research suggesting homogeneity of language and communication dysfunction between AD and HFA, qualitative distinctions between the disorders become questionable.

A DSM-IV diagnosis for either AD or autism requires the presence of qualitative impairments in social interaction. However, because several diagnostic criteria for AD in the DSM-IV have come into question, research comparing AD with HFA in social skills and interaction is needed to determine whether there are differences in these areas. Because communication impairment in AD is supposed to be absent, most researchers comparing social skills hypothesize that children with AD will demonstrate better social functioning. Barbaro and Dissanayake (2007) compared children with AD and HFA on self-presentational display rules, where they were evaluated for their ability to regulate their outer expressions of emotion. AD and HFA groups did not differ in their uses of self-presentational display rules, which were less effective than those used by typically developing children. This result indicates that children with AD and HFA seem to be on par in the regulation of outward emotional expression.

Social skills deficits can sometimes lead to behavioral challenges. Macintosh and Dissanayake (2006a) compared children with AD and HFA in levels of cooperation, assertion, responsibility, and self-control. They found no differences between the AD and HFA groups, but found that both groups showed deficits compared with typically developing children. They also noted that both AD and HFA groups were at increased risk of co-morbid disorders, such as depression.

Other studies have found differences in social skills between children with AD and HFA. Ghaziuddin (2008) classified characteristics of social interaction of children, with IQ above 70, into three categories: first, aloof participants, who were indifferent toward most social situations and other children; second, passive participants, who responded to questions appropriately but did not initiate social contact; and third, active but odd participants, who spontaneously initiated social interaction but did so in an inappropriate manner. Ghaziuddin found that the majority of children diagnosed with autism were described as aloof and passive, and that the majority of children with AD were described as active but odd. This result, while not necessarily demonstrating differences in social skill, do suggest differing social characteristics between AD and autism. Macintosh and Dissanayake (2006b) also found differences in characteristics of social interaction between AD and HFA. Observing children's social interactions in everyday settings, the researchers found that children with AD and HFA demonstrate similar social behaviors in spontaneous peer interactions, social competence, and time spent interacting, which were all below that of typically developing children. However, the authors did find that children with AD demonstrated more overt attempts of initiating social interaction and more conversation during social interaction, suggesting increased social motivation and increased expressive language. In addition, Klin et al. (2005) found that social phobia was present in fewer

individuals with AD than PDD-NOS, but not fewer than those with autism. Taken together, the findings of Ghaziuddin, Macintosh and Dissanayake, and Klin et al. suggest differences in social preferences or personality, not necessarily social skill. For example, extraverted children with autistic symptoms may be more likely to be diagnosed with AD because communication is more apparent, though not necessarily impaired. Conversely, introverted children of similar IQ may be more apt to receive a diagnosis of autism, because the presence of communication and language skill is less apparent.

Discussion

Up to now, there seems to be little consistent evidence of significant differences in symptomology, or social, emotional, or psychiatric problems between AD and HFA. Frith (2004) asserts that Asperger Disorder appears to be a variant of autism typically diagnosed in patients that are verbally and/or cognitively higher-functioning, not a separate disorder. Recent empirical research appears to support this claim.

The recognition of HFA calls into question the validity of DSM-IV criteria of absence of cognitive delay for AD. If AD is a distinct disorder from autism, there is little use for this cognitive criterion because many patients with autism are not cognitively delayed (based on an IQ above 70).

The AD diagnostic criteria in DSM-IV regarding absence of language delay can be problematic for a number of reasons. First, language delay is neither specific nor well-defined. Examples of language delay in the text include "single words used by age 2 years, communicative phrases used by age 3 years" (p. 84). But those abilities do not necessarily preclude the presence of a language delay. For example, an echolalic child may use single words by age 2 but with no communicative meaning or intent. Second, the presence of language delay in the first 3 years of life does not necessarily translate into lifelong language impairment, nor does the apparent absence of language delay in toddlers necessarily lead to lifelong verbal ability within the average or functional range. Differences in verbal ability between AD and HFA have disappeared as early as primary school age in some studies (Howlin 2003). Third, because recognition of language delay is age-sensitive (i.e., by age 2 years) the burden is in part left on the parent to determine whether the child demonstrated a language delay. If, for example, a child vocalizes advanced terminology relative to age, the parent may perceive relative verbal strength, even if that child speaks the term inappropriately or as a stereotyped pattern of behavior. Fourth, because language delay is age-sensitive, clinicians must rely on retrospective reports of language development, which may not accurately represent true language functioning. As it stands in DSM-IV, the presence or absence of "language delay" seems to be the primary (if not only) discriminating variable between AD and HFA. Language delay, however, lacks definitive meaning and is subject to invalid retrospective reporting, and is likely not well-fit as a discriminating variable between disorders (Macintosh and Dissanayake 2004).

What may be difficult to conceptualize is that verbal deficit is considered a primary characteristic of autism, and when an apparently autistic child shows relatively enhanced verbal ability we might perceive the need to reclassify that child. In addition, verbal ability seems highly relatable to cognitive functioning (e.g., a person with higher VIQ may give a first impression of precociousness compared to a person with higher PIQ). Therefore, when a child appears precocious at first glance, there may be an assumption that the child has strong verbal ability (therefore, not autistic), which may not be the case. A child can "speak like an adult" (i.e., children in Kanner's and Asperger's studies), but still show deficits in verbal ability, such as the use of grammatical rules and syntax (Bennett et al. 2008). This long-standing impression of verbal deficits in autism may have skewed our impressions of the actual disorder. Language impairment, for example, may be a secondary characteristic of social abstinence. Further research is needed in this area to better understand the fundamental aspects of autism, and to determine whether our current framework best fits the actual disorder.

It should not be surprising to observe greater cognitive ability and verbal acumen among some people with autism compared with others with autism; such differences in verbal ability exist within typically developing populations. Szatmari et al. (2000) seemed to recognize that variation in development of patients with autism does not mean that a different diagnostic category is necessarily needed. These researchers suggest that AD and autism may "represent parallel and potentially overlapping developmental trajectories" (p. 1980). Therefore, it may be more accurate to think about patients with Asperger's as patients with autism that are precocious and/or verbally gifted relative to other patients with autism.

Apparent strengths in verbal ability may be designated to children with AD, relative to HFA, because they are more apt to be heard. Similar deficits in communication have been demonstrated for children with AD and HFA (Bennett et al. 2008), as well as deficits in social skills (Barbaro and Dissanayake 2007; Macintosh and Dissanayake 2006a). However, social motivation (Macintosh and Dissanayake 2006a) and a propensity to speak (Ghaziuddin 2008) have been shown to be greater in AD. This may not represent a distinct diagnostic difference, rather differences in personality. Children more likely to be diagnosed with

AD rather than autism may be more extraverted than other autistic children. Differences in extraversion/introversion have been well-established in personality theory and in typically developing populations; it should not be surprising that some children with HFA would be more extraverted than others.

Based on up-to-date empirical research, AD and autism may be different quantitative manifestations of the same disorder, not qualitatively distinct. The differences in cognitive, language, and social ability between AD and autism may be a function of individual variability in these areas, not necessarily the presence of unique disorders. Because individual variability is apparent in typically developing populations, such variability could be found within the autistic population as well. A child with autism may have relatively high verbal ability and cognitive functioning, and/or may present with an extraverted personality. Because the DSM-IV criteria at present distinguish AD as a lack of language delay and cognitive functioning, such a child may be more likely to be diagnosed with AD. Other children with autism may present with lower cognitive and language functioning, and receive a diagnosis of autistic disorder. Children with higher relative cognitive functioning but an introverted personality may be deemed as HFA. More research is needed to understand whether individual variation accounts for the apparent differences in children and adults with AD and autism, and whether AD belongs on the autistic spectrum.

References

American Psychiatric Association. (1952). *Diagnostic and statistical manual of mental disorders*. Washington, DC: American Psychiatric Association.

American Psychiatric Association. (1968). *Diagnostic and statistical manual of mental disorders* (2nd ed.). Washington, DC: American Psychiatric Association.

American Psychiatric Association. (1980). *Diagnostic and statistical manual of mental disorders* (3rd ed.). Washington, DC: American Psychiatric Association.

American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th ed., TR). Washington, DC: American Psychiatric Association.

Barbaro, J., & Dissanayake, C. (2007). A comparative study of the use and understanding of self-presentational display rules in children with high functioning autism and Asperger's disorder. *Journal of Autism and Developmental Disorders, 37*(7), 1235–1246.

Bennett, T., Szatmari, P., Bryson, S., Volden, J., Zwaigenbaum, L., Vaccarella, L., et al. (2008). Differentiating autism and Asperger syndrome on the basis of language delay or impairment. *Journal of Autism and Developmental Disorders, 38*(4), 616–625.

Frith, U. (1991). *Autism and Asperger syndrome*. Cambridge: Cambridge University Press.

Frith, U. (2004). Emanuel Miller lecture: Confusions and controversies about Asperger syndrome. *Journal of Child Psychology and Psychiatry, 45*(4), 672–686.

Ghaziuddin, M. (2008). Defining the behavioral phenotype of Asperger syndrome. *Journal of Autism and Developmental Disorders, 38*(1), 138–142.

Ghaziuddin, M., & MountainKimchi, K. (2004). Defining the intellectual profile of Asperger syndrome: Comparison with high-functioning autism. *Journal of Autism and Developmental Disorders, 34*(3), 279–284.

Howlin, P. (2003). Outcome in high-functioning adults with autism with and without early language delays: Implications for the differentiation between autism and Asperger syndrome. *Journal of Autism and Developmental Disorders, 33*(1), 3.

Iwanaga, R., Kawasaki, C., & Tsuchida, R. (2000). Brief report: Comparison of sensory-motor and cognitive function between autism and asperger syndrome in preschool children. *Journal of Autism and Developmental Disorders, 30*(2), 169–174.

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

Klin, A., Pauls, D., Schultz, R., & Volkmar, F. (2005). Three diagnostic approaches to asperger syndrome: Implications for research. *Journal of Autism and Developmental Disorders, 35*(2), 221–234.

Koyama, T., Tachimori, H., Osada, H., Takeda, T., & Kurita, H. (2007). Cognitive and symptom profiles in Asperger's syndrome and high-functioning autism. *Psychiatry and Clinical Neurosciences, 61*(1), 99–104.

Lyons, V., & Fitzgerald, M. (2007). Asperger (1906–1980) and Kanner (1894–1981), the two pioneers of autism. *Journal of Autism and Developmental Disorders, 37*(10), 2022–2023.

Macintosh, K. E., & Dissanayake, C. (2004). The similarities and differences between autistic disorder and Asperger's disorder: A review of the empirical evidence. *Journal of Child Psychology and Psychiatry and Allied Disciplines, 45*(3), 421–434.

Macintosh, K., & Dissanayake, C. (2006a). A comparative study of the spontaneous social interactions of children with high-functioning autism and children with Asperger's disorder. *Autism, 10*(2), 199–220.

Macintosh, K., & Dissanayake, C. (2006b). Social skills and problem behaviours in school aged children with high-functioning autism and Asperger's disorder. *Journal of Autism and Developmental Disorders, 36*(8), 1065–1076.

Maestro, S., Muratori, F., Cesari, A., Pecini, C., Apicella, F., & Stern, D. (2006). A view to regressive autism through home movies: Is early development really normal? *Acta Psychiatrica Scandinavica, 113*(1), 68–72.

Matson, J. L., & Wilkins, J. (2008). Nosology and diagnosis of Asperger's syndrome. *Research in Autism Spectrum Disorders, 2*, 288–300.

Mayes, S. D., & Calhoun, S. L. (2004). Influence of IQ and age in childhood autism: Lack of support for DSM-IV Asperger's disorder. *Journal of Developmental and Physical Disabilities, 16*(3), 257–272.

Ozonoff, S., Rogers, S. J., & Pennington, B. F. (1991). Asperger's syndrome: Evidence of an empirical distinction from high-functioning autism. *Journal of Child Psychology and Psychiatry, 32*(7), 1107–1122.

Rutter, M. (1978). Diagnosis and definition of childhood autism. *Journal of Autism & Childhood Schizophrenia, 8*(2), 139–161.

Rutter, M., & Schopler, E. (1987). Autism and pervasive developmental disorders: Concepts and diagnostic issues. *Journal of Autism and Developmental Disorders, 17*(2), 159–186.

Shriberg, L. D., Paul, R., McSweeny, J. L., Klin, A. M., Cohen, D. J., & Volkmar, F. R. (2001). Speech and prosody characteristics of adolescents and adults with high-functioning autism and asperger syndrome. *Journal of Speech Language & Hearing Research, 44*(5), 1097–1115.

Szatmari, P., Bryson, S. E., Boyle, M. H., Streiner, D. L., & Duku, E. (2003). Predictors of outcome among high functioning children with autism and Asperger syndrome. *Journal of Child Psychology and Psychiatry and Allied Disciplines, 44*(4), 520–528.

Szatmari, P., Bryson, S. E., Streiner, D. L., Wilson, F., Archer, L., & Ryerse, C. (2000). Two-year outcome of preschool children with autism or Asperger's syndrome. *American Journal of Psychiatry, 157*(12), 1980–1987.

Wing, L. (1981). Asperger's syndrome: A clinical account. *Psychological Medicine, 11*, 115–129.

Wing, L. (2000). Past and future of research on Asperger syndrome. In A. Klin, F. K. Volkmar, & S. S. Sparrow (Eds.), *Asperger syndrome* (pp. 418–432). New York: The Guildford Press.

Woodbury-Smith, M., Klin, A., & Volkmar, F. (2005). Asperger's syndrome: A comparison of clinical diagnoses and those made according to the ICD-10 and DSM-IV. *Journal of Autism and Developmental Disorders, 35*(2), 235–240.