LITERATURE REVIEW

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Introduction

The purpose of this literature review is to provide an overview of the clinical phenomenon known as Asperger syndrome, one of a group of conditions known as the pervasive developmental disorders (PDDs), also known as the autistic spectrum disorders (ASD). Diagnostic convention holds that individuals with autism with normal IQ are labelled as having "high-functioning autism" (HFA). If an individual meets the criteria for HFA yet does not have a history of language delay or abnormality in communication, they are classified as having Asperger syndrome (American Psychiatric Association, 1994). Because there is no clearly defined distinction between the diagnostic categories of HFA and AS, this paper employs the protocol of autism spectrum conditions (ASC). This review has adopted Baron-Cohen's (1999) convention of "autism spectrum conditions" (ASC) in describing the participants, choosing to remove the pejorative "disorder" from the descriptor.

There is some evidence that the diagnosis of ASC is becoming more widely used in child psychiatry. This phenomenon may be traced to a number of factors: first, ASC may be more common than previous research indicated; second, child health professionals may be using the Asperger Syndrome diagnosis to spare the family distress and the implied poor prognosis that a diagnosis of autism may incur. The result of the greater frequency in diagnosis has meant child health services need to become more acquainted with the most important controversies and theories about ASC. There is very little information available readily accessible by parents, spouses, siblings, teachers, school counsellors, school administrators, vocational rehabilitation specialists, and even mental health professionals. The Internet is replete with information about ASC, mainly through webrings and personal web sites.

This review offers a summary of the research literature regarding the core features of the the autistic spectrum conditions (ASC); a history of the construct; recent epidemiological studies; etiological factors influencing its development, including neurobiological, genetic, and environmental causes; the neuropsychology of ASC; and finally the outcome

for adults with ASC, and treatment of AS, including psychological and pharmacological therapies. For more comprehensive reviews, the reader's attention is drawn to the following excellent summaries by Bailey *et al.* (1996), Filipek *et al.* (1999), and Gillberg (1998), as well as the edited books by Klin, Volkmar, and Sparrow (2000) and Schopler, Mesibov, & Kunce (1998), which provide up to date, comprehensive discussions on ASC from a number of theoretical and professional perspectives.

Computerised literature searches of the Medline (National Library of Medicine) and PsychInfo (American Psychological Association) databases in English using the term "autism OR asperger" produced over 2,000 articles published in the last 10 years, a figure that excludes books and book chapters, which run into the hundreds. Limiting the search to the term "asperger" for the years 1940 - July 2001 found 219 references: 5 references before 1980, 8 more in the next ten years, 51 between 1990-1995, 108 between 1995-2000, and 57 between January 2000 and July 2001. Most papers on Asperger Syndrome have referred to children and adolescents, while there have been proportionately more papers on adults with high-functioning autism, possibly because Asperger Syndrome is a "newer" diagnosis, applied to "newer" (i.e. younger) populations (Gillberg & Ehlers, 1998).

Historical Perspective

Leo Kanner (1943), an émigré German child psychiatrist in the United States, first identified a syndrome of "autistic disturbances" among a group of children who shared patterns of behaviour including social incapability, obsessiveness, stereotypy, and echolalia (Frith, 1991). He wrote of a group of patients who appeared aloof or indifferent to other people, resisted change, and engaged in repetitive activities. As these children grew older, he observed the following commonalities: a conspicuous absence of make-believe play; a fascination with objects which were often skillfully handled; mutism or language which seemed to lack communicative intent; and "islets of ability" or special skills which were expressed in remarkable feats of rote memory, calculation, or some other isolated skill. Shortly after Kanner's paper appeared in the United States, the

Austrian paediatrician Hans Asperger (1991/1944) published a report in a German journal, in which he described four boys with "autistic psychopathy", who displayed deficits in social interaction and milder autistic behaviours, despite apparently adequate verbal and cognitive skills. Asperger emphasized that the four children were representative of a much larger sample of children in the population. These boys were cognitively within the normal range yet they demonstrated deficits that resembled a milder, or "higher-functioning" form of autism. Not only did his patients have egocentric preoccupations that interfered with acquiring skills in other areas, but they tended to intellectualise their feelings, had poor empathy, and struggled to grasp social cues. Asperger also noted that they were clumsy and uncoordinated, with an odd posture and gait, which prevented their involvement in group sports. Further, he described them as like "little professors" who talked at length about their own area of interest, but whose nonverbal and pragmatic communication was impaired. He left no doubt that their egocentric demeanour, overarching restricted interests, combined with a reduced capacity for empathy, poor social understanding and peer relationships led to aggression and other disruptive behaviours. In his original paper, Asperger (1991/1944) was optimistic about the outcome, citing both the usefulness of their special talents in employment and the existence of similar traits in their well-functioning parents. However in later work, Asperger was less convinced of a rosy outcome (Volkmar *et al.*, 2000).

Although initially unaware of each other's work, both Kanner and Asperger used the term "autistic" to characterize the disturbances that they observed. This term had been introduced by Eugen Bleuler in 1911 to describe the extreme withdrawal from the outside world into the self, which he identified as the basic disturbance in schizophrenia (Frith, 1991). Indeed, a number of practitioners in the field of abnormal child development began to describe a group of children whom they classified as having symptoms that were part of a syndrome of childhood psychoses. Both Kanner and Asperger recognized that, in contrast to Bleuler's schizophrenia, the difficulties of patients in entering affective relationships with others seemed to be present from the beginning. Unlike the "autism" of schizophrenia (typified by a progressive loss of contact with the external world), Kanner's and Asperger's patients exhibited their difficulties early in life and with a

consistent and chronic, rather than progressively deteriorating, course. There are certain characteristics of the children that Asperger described that differ from Kanner's descriptions of autistic children. Asperger described children who developed speech and language by the time they entered school, typically with large vocabularies and good grammatical skills. They tended to seek the company of peers, but did not know how to do so appropriately. Asperger also described these children as having original thoughts and strange obsessions (Frith, 1991, p. 96-97). In contrast, Kanner (1943) described children who typically did not speak or, if they did speak, they possessed a limited vocabulary. The children that Kanner described did not seek the company of peers, but were characterised by "an extreme autistic aloneness that, whenever possible, disregards, ignores, shuts out anything that comes to the child from the outside" (p. 242).

Asperger believed that the disorder had neurobiological causes, and he placed emphasis on the stability of the clinical picture throughout the lifespan, noting that neither environment nor education affected the major characteristics of the syndrome. He recognized that although the symptoms and problems change over time, the overall problem is seldom outgrown and the essential aspects of the problem remain unchanged. In early childhood there are the difficulties in learning simple practical skills and in social adaptation. These difficulties arose out of the same disturbance which at school age causes learning and conduct problems, in adolescence job and performance problems, and in adulthood social and marital conflicts.

Both Wing (1998) and Klin (1994) have interpreted "psychopathy" benignly, as an "abnormality of personality" or "personality disorder". Schopler (1998) takes an opposing view, asserting that Asperger's paper is unequivocal in its use of contemporary understandings of psychopathy. For example, Asperger (1991/1944) wrote that "[e]ven when treated affectionately they respond with malice and cruelty. They delight in malice, bordering on actual sadism, directed either physically or psychologically" (p. 81). Schopler pointed out that Wing (1981) identified eight attributes of the condition in her review of Asperger's work, but did not include "characteristics given equal weight by Asperger such as sadism, antisocial behaviour, tendency to homesickness, and so on" (p.

387). If psychopathy is the main distinguishing feature between the autism of Kanner and Asperger, then perhaps Asperger was describing a very different group of children than his colleague in the United States.

The importance of Asperger's work went largely unrecognized until 1981, when Lorna Wing published a review of his wartime work as well as a number of case studies of her own. Kanner, on the other hand, lived in America and published in English. Although he discussed the congenital nature of autism, Kanner proposed that parental psychopathology was strongly associated with the development of the disorder. Tragically, clinical wisdom held that parents were the underlying cause of the autistic child's delayed and atypical development. In keeping with the psychoanalytic *zeitgeist* within psychiatry and psychology, the environmental model of aetiology and approaches to treatment prevailed throughout the middle years of the 20th century (e.g., Bettelheim, 1967)

Differential Diagnosis

No formal set of diagnostic criteria for autism was developed until the work of Rutter and Hersov (1977), and until the term pervasive developmental disorder (PDD) was first included in the third edition of the Diagnostic and Statistical Manual (DSM-III, 1980) of the American Psychiatric Association. In 1992, the World Health Organisation included Asperger Syndrome in its tenth edition of ICD, and DSM-IV, published in 1994, included Asperger's Disorder (rather than Asperger Syndrome) under the PDD rubric. In both classification systems there are five diagnoses included under the PDD or Autistic Spectrum heading: autistic disorder/childhood autism, Rett's disorder/syndrome, childhood disintegrative disorder (CDD), Asperger's disorder/syndrome, and pervasive developmental disorder not otherwise specified (PDD-NOS) or PDD, unspecified. As with any other psychiatric diagnosis, a reliable nosology for an "autistic spectrum" depends on two conditions: differentiating between the subtypes of the autistic conditions and separating the subtypes from non-autistic disorders. In the first case, Asperger Syndrome is the most controversial of the autistic spectrum diagnoses. Researchers have

debated whether children on the autism spectrum can be reliably classified into different sub-groups, or whether autism varies in severity and type, from most to least socially withdrawn and non-communicative. It is by no means clear whether Asperger Syndrome differs in some fundamental manner from other autism spectrum conditions in its natural history, course, adult outcome, family history, genetic component, or neuropsychological profile (Volkmar *et al.*, 2000). In heuristic terms, the qualitative impairments in social interaction, and restrictive and repetitive patterns of behaviours and activities of children with Asperger Syndrome are identical to those for autistic disorder, although highly circumscribed and all-encompassing interests, whilst typical, are not required for diagnosis of Asperger Syndrome. As it is currently defined, the onset of Asperger Syndrome is somewhat later than in autism, and the diagnosis can be made in the absence of a "clinically significant" language delay, i.e., single words by 2 years of age, and communicative phrases by age 3 years. This requirement probably contributes to Asperger Syndrome being identified later than the other autistic conditions. Diagnostic criteria requires that normal or near-normal IQ (FSIQ >70) is also necessary, as well as normal adaptive behaviour (outside social interaction), such as self-help skills and curiosity about the environment. It is impossible to confer a dual diagnosis of Asperger Syndrome and autistic disorder, as autistic disorder must take precedence in both diagnostic systems. High-functioning autism or HFA, like AS, usually refers to children with autism who have an IQ score over 70; which is close to 25% of the autistic population (American Psychiatric Association, 1994). Frith (1991) considered Asperger Syndrome to be a subcategory of autism, and on the same continuum as autism. Szatmari, Bartolucci, and Bremner (1989) also held the view that Asperger Syndrome is "on the autistic spectrum" (p. 559). These authors suggested that Asperger Syndrome and autism "share a common aetiology but differ primarily on severity" (p. 554). Frith (1989) stated that Asperger Syndrome should "be reserved for the rare intelligent and highly verbal, near-normal autistic child" (p. 8).

Wing's (1981) conceptual framework of autism as a triad of impairments in social interaction, social communication, and imagination, typically accompanied by stereotypic or repetitive behaviour, has driven research interest in the area, such that Rutter (1999)

has argued that autism is the most validated psychiatric disorder in childhood. However, DSM diagnostic criteria have been shown to be less reliable in diagnosing individuals whose symptoms resemble "classic autism", but who may not meet the criteria in one of the three domains, or because their symptoms are not markedly impairing (Tanguay, Robertson, & Derrick, 1998). For example, some more able children can grow to become less socially isolated, more competent in communication, and develop intense interests in more abstract concepts, rather than concrete objects (Szatmari, 2000). Gillberg (1998) has consistently argued for a developmental perspective in the diagnosis, arguing for the possibility that "in some individuals it might be appropriate to diagnose autism at one point in time and Asperger Syndrome at another (p. 201)." The stigma surrounding autism is another important social factor in arriving at a diagnosis. Because of the negative connotations of the diagnosis for many parents, clinicians may tend to give a diagnosis of Asperger Syndrome rather than high functioning autism (HFA), as an alternative, more acceptable, "A" word (Filipek *et al.* 1999). Frith (1991) has described children with Asperger Syndrome as having "a dash" of autism.

It is likely that there may be multiple underlying subtypes and mechanisms behind the broad clinical picture of Asperger syndrome. This leaves room for some confusion regarding diagnostic terms and it is most likely that children and adults with similar behavioural and neurocognitive characteristics are being diagnosed with Asperger Syndrome, HFA, atypical autism, or PDD-NOS, depending upon by whom, or where, they have been assessed. To highlight the uncertainty around the construct, if Asperger were to return from the grave he would have trouble diagnosing the disorder that bears his name. A growing number of researchers has cited the lack of research support for the Asperger Syndrome diagnosis, with Lorna Wing herself arguing in 1998 that HFA and Asperger Syndrome are indistinguishable conditions. Miller and Ozonoff (1997) concluded that under current DSM-IV diagnostic criteria, Asperger should have diagnosed his four patients as having Autistic Disorder (Kanner's autism), and not Asperger Syndrome. A recent study of 157 children diagnosed with autism spectrum conditions found that, on clinical review using criteria from DSM-IV, all of the children met criteria for autistic disorder, and none of the children could be assigned a diagnosis

of Asperger Syndrome (Mayes *et al.* 2001). It was an "impossible" diagnosis because all the children possessed communication impairments, such as an inability to initiate or sustain a conversation or idiosyncratic language use, which was evident prior to the age of three years. The authors concluded that clinicians may be disregarding the DSM-IV's criteria and are using "definitions influenced by literature and popular belief" (p. 268).

Of course, fuzzy diagnostic boundaries have not prevented children with significant problems in social interaction (but without the full clinical picture of Kanner's autism) from presenting to health professionals, and these individuals have been assessed according to the individual clinician's own area of expertise. For example, such children have been labelled as having a "schizoid personality" (Wolff & Barlow, 1979); "nonverbal learning disability" (Rourke, 1995); "developmental learning disability of the right hemisphere" (Denckla, 1983); "semantic-pragmatic disorder" (Bishop, 1989); multiple complex developmental disorder (Ad-Dab'Bagh & Greenfield, 2001); and "developmental receptive-language disorder" (Howlin, Mawhood, & Rutter, 2000). Although there appears to be significant symptom overlap in the different populations, it remains uncertain how best to integrate these findings into a coherent diagnostic entity (Volkmar *et al.*, 2000). The problems in diagnosing autism represent a more general problem when an attempt is made to categorically differentiate the autistic spectrum, or psychiatric disorders in general.

Although the current diagnostic systems were deliberately formulated as "atheoretical" taxonomic systems, a number of specific theoretical assumptions underlie the projects. According to Robert Spitzer, the chair of the DSM-III and DSM-IIIR work groups, a "mental disorder" exists as "merely a subset of medical disorder" (Spitzer & Endicott, 1978, p. 16), implying that psychopathology is best understood as a disease or illness entity manifesting as dysfunction within individuals. DSM's categorical "deficit" model of psychopathology has successfully achieved a medicalisation of human behaviour, in which symptoms are stripped of their political, social, or evolutionary meanings. Psychiatry has employed clients' subjective experiences to confirm that symptoms are manifestations of an underlying biological disorder. Prevailing psychiatric discourse

theorises psychopathology as residing within the individual's "dysfunctional", requiring amelioration through primarily pharmacological intervention. Psychiatric definitions of mental disorders rest on a notion of abnormality or functional impairment. While the diagnostic system has contributed to the development of numerous treatments and invaluable research into human behaviour, the assumption that psychopathology can be understood in an objective scientific manner separate from sociocultural factors has been vigorously rejected by many authors. Although the DSM includes social conditions as important for consideration, intraorganismic dysfunction has been posited as the "core" of mental disorders, with social factors "providing a superficial overlay" (Thacker, Ward, & Strongman, 1999, p. 846).

Wakefield (1992) has also criticised DSM for the overinclusiveness of its diagnostic criteria, and its failure to distinguish psychiatric conditions resulting from internal dysfunctions (either in brain structure, neurochemical transmission, or cognitive distortions) from conditions that are brought about because of societal disapproval. Wakefield introduced the notion of harmful dysfunction to the definition of mental disorders, that is, a disorder should not only interfere with individual function, but cause some form of harm, according to societal standards. Wakefield has not argued for the abandonment of diagnosis, rather he advocated for an approach based on the DSM's internal logic of the concept of disorder as used in the medical sciences as a whole. One of the goals of the DSM-III task force was to arrange mental disorders in clearly defined categories, to facilitate greater reliability among clinicians. However, although more reliable, many authors (e.g., Butler, 1999) have contended that it has come at a cost to validity and that the DSM system encourages clinicians to see the diagnostic label as explanatory, rather than descriptive, thus obscuring the meaning of the symptoms to the individual.

In recent years, a number of researchers have attempted to place the study of psychological disorders within a framework of evolutionary theory, prompting the development of the new field of evolutionary psychopathology (Gilbert, 1998; Murphy & Stich, 2000). That is, the mind is best understood according to its evolved adaptation to

the environment (Pinker, 1997). Murphy and Stich (2000) have outlined a bimodal distinction between disorders that arise from a malfunction of a module of the mind and those that arise due to our minds functioning in environments that are very different from those in which they evolved. The mismatch between current and ancestral environments can produce mental disorders, despite there being nothing wrong with those minds except that they were in play too late, in an evolutionary sense. The proposal has important implications for the classification of mental disorders. The question remains: why have the obvious disadvantages associated with the autism genes not removed their presence from the human population? Crow's (1995) application of evolutionary approaches to psychosis has furnished psychologists with a framework with which to understand autism. Autism is similar to psychosis in that both conditions are relatively "invariant with respect to substantial variations in climactic, social and industrial environments; ...[and they occur] at approximately the same incidence in populations separated by thousands of years" (p. 12).

Clinical Features

Onset and characteristics of early development

Professional consensus has identified three core sets of symptoms in the autism spectrum conditions: impaired reciprocal social interaction, delayed language, and aberrant activities (such as restricted range of interests and rigid adherence to routine). Impairments in socialization, communication, and imagination are present in different forms at all stages of development (Frith, 1991). Characteristic (but not core) symptoms of autism include mental retardation, uneven cognitive profiles, savant abilities, and abnormalities of attention (Rutter, 1999). One of the earliest signs thought to be specific to autism is a lack of pointing and looking to share interest and attention with another person, and there is some evidence from the use of a general practitioner checklist that a lack of protodeclarative pointing is one of the factors predictive of a later diagnosis of autism (Baron-Cohen *et al.* 1996). However, in children with global developmental delays, this behavior would also be expected to emerge later, and hence would lack

specificity to autism. Losche (1990) reviewed early home movies of autistic and nonautistic subjects and concluded that the timing and sequence of developmental gains differs between normal and autistic children after the first year of life. Travis and Sigman (2000) stated that "the first clear signs of autism involve the failure to show the patterns of social, communicative, and emotional responses that typically emerge [at the end of the first year]" (p. 643-644). They report that children with autism exhibit the following deficits: an impaired ability to jointly attend to a third object, poor aid-eliciting to obtain objects, difficulty maintaining ongoing interaction with another, and an inability to use social referencing, that is, using emotional expressions of others to gain knowledge about the world. There is no question that children with autism establish attachment relationships (Sigman & Ungerer, 1984), although they tend to be less likely to spontaneously initiate play, typically choosing to play alone. It may, therefore, be difficult to make the diagnosis of autism with confidence prior to two or three years of age (Frith, 1991). During the preschool years a more recognizable pattern of behavior difficulties emerge. Language may be delayed, precocious, or otherwise highly idiosyncratic (Wing, 1991). Some children show an early fascination with numbers and letters. Hyperlexia may be evident in which the child is able decode towords, but with little or no comprehension of meaning. Deafness is often suspected because these children seem unaware of what is going on around them. Social interaction is noticeably impaired, make-believe play is absent, and instead, the child may become fixated on simple repetitive activities or rituals. In young children deficits in "mentalising" (i.e., the ability to attribute mental states such as thoughts, feelings, and motives) to others and to oneself may be evident. As they mature, children with ASC may pass mentalising tasks in formal test situations while continuing to show deficits in applying these abilities in real-life settings (Ozonoff *et al.* 1991).

In conclusion, Bailey, Phillips, and Rutter (1996) reported that most cases of autism manifest by the age of 18 months, and it may even be evident by 12 months, although diagnostic signs may be more easily missed at the younger age. In a child with milder cognitive and social deficits, identification may be significantly more difficult, and

"normal" language development in the latter part of the second year probably contributes to missed diagnosis.

Language

Although current clinical practice excludes any clinically significant language delay in diagnosing AS, there are usually some observable differences in how children use language. First, their prosody (those aspects of spoken language such as volume of speech, intonation, inflection, rate, *inter alia*) is frequently unusual. Inflection and intonation typically are not as rigid and monotone as in autism, but a restricted range of intonation patterns may result in utterances in which tone of voice is inconsistent or unrelated to the content and communicative intent. Their speech can be pedantic and poorly modulated, with a concrete and literal tone to conversation. Second, pragmatic, or conversational, language skills often are weak because of problems with turn-taking, a tendency to revert to areas of special interest, or difficulty sustaining the subtle flow of conversations (Bishop, 1989). Therefore, speech may seem tangential and disordered, yet it is more often a reflection of their egocentric conversational approach and failure to censor output which accompanies internal thoughts. This may be evident in monologues on the topic of consuming interest (e.g., geography, railway schedules, computer software); failure to integrate what the listener can be expected to know in terms of background information; as well as difficulty implementing the rules of conservation, such as turn-taking and topic transitioning. Impoverished nonverbal pragmatic or communication skills may also be present. A third characteristic of communication among individuals with ASC is verbosity, such as the tendency to launch into monologues on their favorite topic (with apparent disregard for the listener's interest, nonverbal signals, or background knowledge), not understanding that this interferes with their interactions with others and puts others off. Sometimes the language sounds overly formal or pedantic, idioms and slang may not be used or are misused, and language comprehension tends toward the concrete. Many children with ASC have difficulties dealing with humor, tending not to "get" jokes, laughing at the wrong time, or "missing the point", which has led some researchers to conclude that people diagnosed with

pervasive developmental disorders are humourless. This is despite the fact that quite a few individuals show an interest in humor and jokes, particularly puns or word games (Attwood, 1999).

Narrative language and autism spectrum conditions

Loveland and Tunali (1999) proposed that narratives should be treated as kinds of communicative acts, i.e., examining not only the content of the narrative but how they function for the speaker and the listener. They listed four categories of narratives as a basis for analysis and research. A story-narrative is an "organised series of causally-related event-descriptions that deal with some topic or lead to some point" (p. 249). They may be fictional or anecdotal, both requiring an understanding of event causality and of linguistic tools for describing events. Script narratives are narrative accounts of generalisations of events and represent information about commonalities of events over time. Informative/didactic narratives are produced to convey specific information to someone. Recitations and performances are narratives when an individual recites learned narrative speech.

The narratives of people with autism tend to reflect disordered language, such as difficulties in grammar, word-finding, and semantics. They also may differ in content, including bizarre, or irrelevant material, reflecting an idiosyncratic world-view. Pragmatic errors may reflect a poor understanding of the listener's knowledge (referring to things or persons unknown to the audience, with little explanation) and affective states. It is also likely that the narratives will reflect poor awareness of the thoughts, feelings, and motivations of characters in the narrative. Finally, people with autism do not fully participate in the social and cultural context in which narration takes place.

Social Development

Children with autism pectrum conditions are frequently noted by teachers and parents to be "in their own world" and preoccupied with their own agenda, yet they are seldom as

aloof as children with autism. Autistic individuals below normal IQ may be more apt to be withdrawn and may seem disinterested in relating to others, whereas those with ASC are often quite eager to relate to others but lack the skills to do so (Klin & Volkmar, 1995). In fact, most children with ASC, at least once they get to school age, are aware of other people, and express a desire to fit in socially and have friends. Yet few individuals with ASC are able to form friendships (Filipek et al, 1999). They are often deeply frustrated and disappointed by their social difficulties. Their problem is not a lack of interaction so much as a lack of effectiveness in interactions, seeming to have difficulty knowing how to "make connections" socially. Because their approaches tend to be inappropriate and peculiar, they are often (involuntarily) socially isolated from their peers. Although the individual with ASC may be able to correctly describe other people's intentions, emotions, and conventions, they are unable to execute this knowledge in a spontaneous and useful manner. The lack of spontaneous adaptation is associated with an over-reliance on formalistic rules of behavior. Gillberg (1995) has described this as a "disorder of empathy", the inability to effectively "read" others' needs and perspectives and respond appropriately. As a result, children with ASC tend to misread social situations and their interactions and responses are frequently viewed by others as "odd". Mesibov and Stephens (1990) studied the perception of popularity among adults with a diagnosis of high-functioning autism (HFA) and found that, like their age-mates, they valued humor, attractiveness, intelligence, and athletic ability. However, they did not always agree with their age-mates' perceptions of these attributes.

Restricted Range of Interests, Activities, or Behaviors

The most obvious hallmark of people with autism spectrum conditions is their peculiar, idiosyncratic areas of "special interest". In contrast to autism with mental retardation, where the interests are more likely to be objects or parts of objects, in ASC the interests appear most often to be specific intellectual areas. Often, very young children will show an obsessive interest in an area such as mathematics, aspects of science, reading (some have a history of hyperlexia, or rote reading at a precocious age) or some aspect of history or geography, wanting to learn everything possible about that subject and tending

to dwell on it in conversations and free play. A number of children with ASC focus on maps, weather, astronomy, various types of machinery or aspects of cars, trains, planes or rockets. Interestingly, as far back as Asperger's original clinical description in 1944, the area of transport has seemed to be a particularly common fascination (he described children who memorized the tramlines in Vienna down to the last stop). Many young children with ASC seem to be unusually aware of things such as the route taken on car trips. Sometimes the areas of fascination represent exaggerations of interests common to children in our culture, such as Pokemon, Ninja Turtles, Power Rangers, dinosaurs, etc. Given deficits in the pragmatics of social interaction, the AS individual will readily share this information, at great length, and in considerable detail. The area of special interest may dominate the social interactions and activities of the AS individual (and their families as well). In many children the areas of special interest will change over time, with one preoccupation replaced by another. Klin and Volkmar (2000) have reported that the specific subject area may change every two years or so, but in some children the interests may persist into adulthood, and there are many cases where the childhood fascinations have formed the basis for an adult career, including a good number of college professors (Attwood, 1999).

Motor Deficits

Although not a required criterion for a diagnosis of AS, gross and fine motor problems are often seen in association with autism spectrum conditions, and motor clumsiness is an additional symptom. A number of researchers (e.g., Ehlers & Gilberg, 1993; Ghaziuddin, Butler, Tsai, & Ghaziuddin, 1994) have advocated for the inclusion of motor deficits in the diagnosis of Asperger Syndrome and high-functioning autism, with their research findings suggesting that there are high rates of incoordination among the population with ASC. Unfortunately measuring motor skills is an extremely difficult exercise, and there are few standardised instruments up to the task. In a well-controlled Australian study, Manjviona and Prior (1999) failed to demonstrate that children with Asperger Syndrome had significantly impaired motor functioning, nor that clumsiness was useful for differential diagnosis. However, they found that more than half of the children with

Asperger Syndrome combined with the children with HFA (that is, the children with ASC) had clinically significant levels of motor impairment. For example, they learnt to walk a few months later than expected, motor milestones tended to be delayed, and they had difficulty balancing on one leg, with their eyes closed. Adrien *et al.* (1992) reviewed home movies of children before autism was suspected, and found that blind raters were able to differentiate children with autism from those without, based on measures of motility. Writing from clinical experience, Attwood (1999) noted delays in the acquisition of more complex motor skills such as riding a bike, catching a ball, and climbing playground equipment. He stated that individuals with Asperger Syndrome often display an unusual gait when walking or running, poor manipulative skills, and deficits in visual-motor coordination. Rapin (2001) has concurred, citing apraxia (difficulty programming or imitating complex motor acts) as a feature of autism spectrum conditions. Asperger (1991/1944) noted that one of his patients had great difficulty copying various rhythms. Attwood (1999) has stated that,

> As two people walk side by side they tend to synchronise movements of their limbs, ...their movements have the same rhythm. The person with Asperger's Syndrome appears to walk to the beat of a different drum (p. 108).

Rogers and Pennington (1991) have advanced a theoretical model that posits deficits in imitation as a primary explanation for autism. Research literature has consistently found that people with autism do not readily imitate the actions of others. Tantum (2000) has also placed difficulty in copying the actions of others as central to the disorder. He reported that motor incoordination may actually be idiosyncrasy, as there is little sense of modelling their actions or gait on those of others.

Theory of mind and folk psychology

Over the past fifteen years, researchers have investigated how people with autism explain and predict human behaviour. There are a number of words to describe this uniquely human ability, including theory of mind (Premack & Woodruff, 1978), folk psychology

(Astington, 1994; 1999), and the intentional stance (Dennett, 1987). Folk psychology is the ability to attribute beliefs and thoughts to others, and to understand that others have mentalistic perspectives which are unique and different from our own (Leslie, 1987). Happe & Frith (1995) referred to theory of mind as the capacity to "mind-read", that is the ability that human beings have to interpret their own and others' behaviour in terms of what they know and feel. There is growing evidence that many autistic individuals do not understand that other people have their own plans, thoughts, and points of view (Baron-Cohen, 2000). Furthermore, it appears that they have difficulty understanding other people's beliefs, attitudes, and emotions. Many of the tasks used to test this theory were given to non-autistic children as well as children with mental retardation, and the theory of mind phenomenon appears to be unique to those with autism. Most developmentally normal children gain this understanding by the age of 4, and even children with Down's syndrome reach this level of psychological competence by the time they reach the mental age of 4, based on measures of nonverbal intelligence (Astington, 1999). A large body of empirical work has revealed that most children with autism have a deficiency in this area despite attaining significantly higher mental ages on psychological tests (Baron-Cohen, 1995). In addition, theory of mind appears to be independent of intelligence, even though people with Asperger syndrome exhibit this problem to a lesser degree. Failure in theory of mind may underpin the specific cognitive and interpersonal impairments in areas such as imagination and communication of autistic individuals (Baron-Cohen, 1990).

By not understanding that other people think differently than themselves, or the "unwritten rules of society", many people with autism may have problems relating socially and communicating to other people. That is, they may not be able to anticipate what others will say or do in various situations. In addition, they may have difficulty understanding that their peers or classmates even have thoughts and emotions, and may thus appear to be self-centered, eccentric, or uncaring, although there is nothing in the theory of mind to imply that autistic individuals feel superior to others (Klin, Volkmar, & Sparrow, 2000).

Sass, Parnas, and Whiting (2000) have argued that philosophical understandings can enrich the study of psychopathology by "helping to refine phenomenological descriptions of, and conceptual discrimination among, the experiential anomalies characteristic of [mental disorders]" (p. 90). In the case of autism, continental traditions in philosophy mitigate against absolutist positions, suggesting that the cognitive element of theory of mind can not be differentiated from the affective component, therefore undermining the traditional Anglo-American philosophical division between belief and desire. Both Asperger and Kanner placed autism as a disorder of a primarily emotional nature. Mishara, Parnas, and Naudin (1998) critiqued theory of mind in autism as "too closely tied to a representational concept of mind... Intersubjective perception does not rest on a theory of mind but on an initial empathy in an emergent (bi-personal) field where self and others have no priority (pp. 570-1)".

The Russian psychologist Lev Vygotsky argued that, in order to understand children's individual mental functioning, psychologists need to examine the social and cultural process in which it develops. Children are not born outside of their culture, and their mental state concepts are derived by means of enculturation. For example, because parents see their infants' spontaneous gestures as intentional communication the infants come to see themselves as having intentions and therefore start to communicate intentionally. As parents communicate to their children in the language of their thoughts, feelings, and desires, the children acquire the ability to see themselves as holding such states. Vygotsky's theories posit linguistic development as fundamental to gaining mental state concepts. Without language, children could not learn about these concepts. Theory of mind, even mind itself, is seen as a cultural invention. Mental states are not privately held within individuals but "subject to interpretation and open to discussion by others" (Astington, 1999, p. 404).

Prevalence

Until ten years ago, the prevailing scientific opinion was that autism was a rare condition. However, recent expansion of the diagnostic criteria to include "milder" cases along a

continuum of core characteristics, has turned this opinion around. For example, taken as a whole, the autistic spectrum disorders are more prevalent in children than cancer, Downs syndrome, or spina bifida (Filipek *et al.* 1999). Although clinical literature on Asperger syndrome is increasing at an exponential rate, epidemiological studies of its prevalence and characteristics have struggled to keep pace. The reasons for this are manifold: poor agreement on diagnostic attribution; the very recent inclusion in the two dominant diagnostic manuals of a range of "autisms" or PDDs; the broadening of the autism spectrum to include individuals previously considered "odd" or eccentric; and the methodological minefield of all epidemiological studies, such as whether to use "point" or "period" prevalence, "cumulative" or "cohort" incidence. Fombonne (1999) conducted a comprehensive review of 23 epidemiological studies of autism spectrum disorders (across ten countries) published in English between 1966 and 1998. Over 4 million people were surveyed, and Fombonne found that 1533 cases of autism were identified. Across the surveys, there was a reported prevalence rate of between 0.7 to 15.5 per 10,000, with a median of 5.2 per 10,000. Prevalence rates increased significantly with publication year, probably due to changes in case definition and improved identification, and he found that the median rate rose to 7.2 per 10,000 for the 11 studies conducted since 1989. He noted that all selected surveys underestimated the population rate (due to a small number of missed diagnoses) and they identified a larger group of children with some shared symptoms of the autistic group. Based on the 11 recent studies, Fombonne concluded that the prevalence for all forms of pervasive developmental disorder was 18.7 per 10,000. Although there are only two epidemiological studies of the prevalence and characteristics of Asperger Syndrome, Fombonne's review of all autism research suggests that Asperger syndrome is considerably more common than "classic" autism. While "classic" autism has traditionally been felt to occur in about 4-5 out of every 10,000 children, Klin and Volkmar (2000) estimate that the prevalence of Asperger Syndrome or atypical autism may be as high as 20-25 per 10,000.

In one epidemiological study that specifically targeted Asperger Syndrome, using very inclusive diagnostic criteria, Ehlers and Gillberg (1993) conducted a prevalence study of

1500 Swedish school-aged children (7 to 16 year-olds). They found a rate of 36 per 10,000 children for those fitting Asperger's (1991/1944) descriptions and a further 35 per 10,000 with marked social impairment but without the full Asperger Syndrome diagnostic criteria. The authors concluded that nearly 0.7% of the study's participants (71 out of 10,000) had a clinical picture either diagnostic of or suggestive of Asperger Syndrome to some degree. However, these estimates are based on a handful of cases (n = 4 and n = 5, respectively), and caution must be exercised in generalising from such a small sample (Fombonne, 1999).

A great deal of research has gone into discovering whether there is an "epidemic", due to factors such as the MMR innoculations and environmental pollution, without conclusive evidence as to their influence. However, the work of Fombonne (1998; 1999) and others indicates that there is evidence that autism spectrum condition rates are rising, because professional organisations have reached an uneasy consensus on diagnostic criteria, health professionals are more willing and able to make the diagnosis (due to better training, more reliable assessment instruments, and increased media coverage of the phenomenon). In turn, parents, teachers, and adults with ASC are more aware of the possibility of the diagnosis. On a cautious note, the diagnosis is subject to over popularization (Schopler, 1998), just as attention deficit hyperactivity disorder (ADHD) was the childhood disorder of the 90s, Asperger syndrome could become the ADHD of the 00's decade. Parents, desperate to assist their children, and wishing to secure services in the school or community, can be duped into believing the psychiatric rhetoric that a diagnosis is the "royal road" to recovery. The problem is that most psychiatric diagnoses require the prescription of psychiatric medications, all too often in the absence of evidence for their usefulness. It seems churlish (but necessary) to indict a society that requires a psychiatric diagnosis to assist in the process of "normal" identity formation.

Sex Ratios

Asperger syndrome appears to be more common in boys than in girls, although the reasons for this remain unclear. Estimates of the male to female ratio vary from 4:1 to

15:1. Fombonne (1998) found that the male/female ratio was 3.8:1, varying according to level of intellectual functioning. Tantum (2000) agreed that sex rates may vary according to the IQ of the individuals under study. He reported that, for those individuals in the very high to superior ranges of intellectual abilities, males are more strongly overrepresented. The excess of autistic boys over girls was noted by both Kanner (1943) and Asperger (1991), although the incidence for females is higher than originally thought. In reviews of 16 population studies of autism, Wing (1993) and Gillberg (1995) found that the male to female ratio was closer to 2-3:1. Again, however, in autism without mental retardation, the ratio is probably higher (Wing & Gould, 1979), and the male to female ratio in autism tends to go down with decreasing IQ (Wing, 1981). At the lowest ability levels the ratio of boys to girls was only 2:1, yet at the highest ability levels, Wing's sample showed a ratio of 15:1.

The four main explanations for the skewed sex ratios among people with autism spectrum disorders have been (1) genetic (linked to sex chromosomes), (2) brain damage among the more cognitively disabled group (Wing, 1981), (3) differences in overall cognitive styles in males and females leading men to have lower empathy and less ability to mask these empathy deficits (Gillberg, 1995), and (4) evolutionary adaptation (Baron-Cohen, 1997a). Just as Asperger (1991/1944) himself described the condition as an "exaggerated" pattern of male personality, one prominent author has argued that autism is an "extreme form" of the male brain. Baron-Cohen and Hammer (1997) argued that maleness and autistic features or traits are inextricably linked, and that boys are, evolutionarily speaking, "hard-wired" for autism. In a climate of declining performance at school, disproportionately higher rates of reading disorders and social impairments, then boys may have difficulty in adapting to the increasing requirement to be "emotionally intelligent", to listen and "intuit" a sense of the intentions of others. Although there is a powerful explanatory framework for human behaviour in presenting evolutionary principles as a fait accompli, Baron-Cohen tends to portray the differences between the sexes as absolute and unproblematic.

The first evidence is the skewed sex ratio. For individuals with an IQ in the normal range, the sex ratio may be as high as 40 males to 1 female (Ehlers *et al.*, 1997). The second piece of evidence is the superior folk physics skills in people with autism; skills traditionally associated with being male. Folk physics is a proposed evolutionary cognitive module that refers to a basic knowledge of how the physical world of objects works, contrasted to folk psychology, which is a basic understanding of how the social world works (Baron-Cohen, 1997a). For example, fathers and grandfathers of children with Asperger Syndrome are more than twice as likely to work in the field of engineering than controls (Baron-Cohen *et al*, 1997). The final piece of evidence is that recent studies have found a sex difference (in favour of females) for the development of folk psychology (Baron-Cohen, 2000).

Etiology

Genetic causes

Autism is the most strongly genetic of childhood psychiatric disorders (Bailey, Phillips, & Rutter, 1996). Asperger (1991/1944) emphasised that in his research, similar traits were to be found to some degree in parents and other relatives. In some cases there is a clear genetic component, with one parent (most often the father) showing either the full picture of Asperger Syndrome or at least some of the traits associated with Asperger Syndrome. Temperamental traits such as having intense and limited interests, compulsive or rigid style and social awkwardness or timidity also seem to be more common, alone or in combination, in relatives of Asperger Syndrome children (Gillberg, 1998). Sometimes there will be a positive family history of autism in relatives, further strengthening the impression that Asperger Syndrome and autism are related conditions. Siblings of people with autism have a 3 to 8 per cent chance of being diagnosed with the same disorder, much higher than the 0.16 per cent rate in the non-autistic population, but less than the 50 per cent chance that characterises a genetic disorder caused by a single dominant mutation, or the 25 per cent chance that characterises a single recessive mutation (Rodier, 2000). Evidence of a genetic aetiology for autism has been provided

by numerous epidemiological studies. Estimates for sibling frequency have ranged from 2% to 6 %, or 50 to 150 times the frequency in the general population (Rutter & Bartak, 1971). In a study of a relatively large autistic sample, Ritvo (1989) estimated the overall risk of recurrence to be 8.6%. In Ritvo's sample, if the first autistic child was male, the recurrence risk was 7%; if the first autistic child was female, the recurrence rate was 14.5%. In an overview of these studies, Ciaranello & Ciaranello (1995) concluded that all of the studies may underestimate the recurrence rate due to a tendency for parents to stop having children after the birth of an autistic child. However, the extent to which these stoppage rules apply among parents of autistic children is unknown. Attempts to specify a mode of inheritance for autism has been complicated by factors such as sex-influenced inheritance; reduced penetrance; variable expression of the disorder; diagnostic ambiguities; and stoppage rules. Twin studies completed by a number of researchers (e.g., Ritvo, Freeman, Mason-Brothers, & Ritvo, 1985; Steffenburg, Gillberg, Hellgren, & Anderson, 1989) overwhelmingly support the importance of genetic influences. Monozygotic (MZ) concordance rates are 60-90%, compared to 5% for dizygotic (DZ) twins, indicating a heritability of the underlying liability to autism of 90 per cent. Bailey and others (1996) concluded that a higher MZ:DZ ratio is found using a broader autistic phenotype, which includes "milder" cognitive and social deficits, suggesting that traditional diagnostic boundaries are over restrictive. Family studies provide further evidence of a genetic basis for autism, as there is evidence that language and communication impairments are more common in families of children affected, and that familial loading increases with the severity of the autism (Bolton, Pickles, Murphy, & Rutter, 1998).

It seems likely that for Asperger Syndrome, as for autism, the clinical picture is multiply determined by a cascade of many factors, including the action of several genes, so that there is no single identifiable cause in most cases. Rather than a single gene, several genes acting in combination appear to provide a greater vulnerability to autism (Rutter, 1999). The gene-gene as well as gene-environment interactions contribute the vulnerability to, and expression of, the autistic spectrum disorders. Research into the etiology of autism is complicated by the challenge "to identify which brain dysfunctions

are causally and developmentally primary" (Travis & Sigman, 2000, p. 642) among affected individuals, as brain abnormalities may occur due to "aberrant input from other structures, or as a secondary consequence of aberrant experiences during development" (ibid).

Because autism is so transmissible, another fruitful piece in the puzzle comes via evolutionary psychology, and particularly evolutionary cognitive science. The paramount proponent of this position has been Simon Baron-Cohen, who has not only researched the experience of those living with autism, but he has also created a simple checklist for general practitioners to employ at the 18 month check-up, which can predict a later diagnosis of autism (Baron-Cohen, 1996). He has also begun to question the notion of autism as a disability, preferring to use the concept of "cognitive difference", and advocating for more sensitive understandings of people with autism spectrum conditions.

Nongenetic Causes

The most frequently cited nongenetic cause of autism is prenatal exposure to viral infection (Ciaranello & Ciaranello, 1995). Chess (1977) reported a significantly increased incidence of autism in children born during the 1964 rubella pandemic. These children developed autism along with other birth defects characteristic of congenital rubella syndrome. Although other infectious agents have been associated with autism, these are mostly single cases (Lotspeich & Ciaranello, 1993). Varicella (Knobloch & Pasamanick, 1975), rubella (Rodier, 2000), and prenatal toxoplasmosis and syphilis (Rutter & Bartak, 1971) have been linked to cases of autism. Taken together, these and other case reports provide evidence of a possible link between prenatal infection and the disruption of brain development such that autism occurs.

There is inconsistent evidence regarding prenatal, perinatal, or neonatal trauma in association with autism. In a review of the literature, Nelson (1991) was unable to find any consistent link between maternal history, pregnancy, delivery, or neonatal events

with autism. However, in a study of 46 children diagnosed with high functioning autism (HFA), which used normally developing siblings as controls, it was reported that the HFA subjects had a higher frequency of reported complications during pregnancy, were more likely to have a gestation period over 42 weeks, and were more frequently first born or fourth-or-later born children (Lord, Mulloy, Weendelboe, & Schopler, 1991). In a review of the literature, Ciaranello and Ciaranello (1995) concluded that pre- and perinatal factors seem to play a larger role in cases of autism associated with mental retardation than with people diagnosed with high functioning autism. In a very recent case review of 74 children with autism, Juul-Dam, Townsend, and Courchesne (2001) found that there was a consistent association of unfavourable events in pregnancy, delivery, and the neoenatal phase and the autism spectrum disorders. However, the authors cautioned that interpretation of their results was difficult, as the specific complications that carried the highest risk of autism had no unifying pathological process behind them. They speculated that it was possible their results supported the view that autism, as a genetically transmitted disorder, may cause the complications to occur.

Other nongenetic factors associated with autism include hypothyroidism and other medical conditions in the mother. Gillberg, Gillberg, and Kopp (1992) studied five children with autistic conditions and found that three of the five had congenital hypothyroidism and the remaining two had mothers who were likely to have been hypothyroid during pregnancy. Maternal antibody formation and rejection by embryonic lymphocytes was found in association with autism in a sample of eleven mothers of autistic children (Warren, Cole, & Odell, 1990). Davis, Fennoy, and Laraque (1992) studied 70 mothers who used cocaine or engaged in poly-drug abuse during pregnancy. In this sample, 11.4% of these women gave birth to autistic children and 94% of the children born showed a pattern of delayed language development. Environmental toxins have also been implicated as possible etiologic agents in autistic spectrum disorders (Rodier, 2000).

Neurobiology

The literature on the neurobiology of autism spans four decades and has produced a large body of inconsistent and often contradictory results. Almost every region of the brain has contributed to possible loci for autism. Proposed sites have included the limbic system, brainstem, basal ganglia, vestibular system, and cerebellum. While genetic and psychological studies have furnished a relatively robust set of findings, neurobiological investigations suffer from poor replicability, probably due to methodological limitations and the heterogeneity of subject populations (Bailey, Phillips, & Rutter, 1996). Postmortem and neuroimaging studies have outlined neuroanatomical abnormalities in autism but the extent to which their conclusions can be generalized to ASC is not known (Rutter, 1999). One finding that seems robust is that people with autism have unusually large brains, following up on Kanner's (1943) original note that his patients had very large heads (Rutter, 1999).

Structural and cellular abnormalities have been found in the medial temporal lobe and related structures, including the hippocampus and amygdala (Bauman & Kemper, 1985). These deficits have been associated with difficulties in socioemotional functioning, sensory processing, and motor planning (Aronowitz, Decaria, Allen, Weiss, & Saunders, 1997). Kemper and Bauman (1993) reported small, densely packed cells in the hippocampus and amygdala and suggested that this reflected an immature pattern of neuronal development in these structures. They observed neurons which appeared immature in the diagonal band of Broca, which projects cholinergic afferents to the hippocampus and amygdala (Ciaranello & Ciaranello, 1995).

Advances in neuroimaging have confirmed earlier postmodern findings that cerebellar abnormalities are an important aspect of autism (Belmonte & Carper, 1999). Although there is no single neuroanatomic defect in autism, there is evidence that implicates neuronal maturation defects, particularly in the cerebellar vermis, cerebellar hemispheres, and limbic structures. These deficits do not appear to be reflected in the size or metabolic activity of these structures. An interesting conjecture is that the cerebellum paves the way for a cascade of events resulting in the development of autistic symptoms. Previous understandings of the cerebellum's function has been limited to its motor operations, but

its role in cognitive development is beginning to be better understood. The cerebellum projects into the limbic system, then onto the prefrontal cortex. The link between the cerebellum-thalamus-prefrontal systems provides a parsimonious explanation for the dysfunctional development of novel cognitive strategies in people with autism. Thus, the evidence implicating the cerebellum and the limbic forebrain, at the cellular level, suggests possible deficits in neuronal migration, maturation, or synaptic connectivity. However, the speculation has outpaced the empirical evidence at this time, and, until cerebral blood flow techniques are suitable for very young children, the neurophysiology field will be confounded by questions of cause and/or effect.

Another fascinating piece in the autism jigsaw is that about one in four individuals diagnosed with autism develop epilepsy, suggesting a fundamental abnormality of brain function (Rutter, 1999). Further evidence implicating structural problems has come from findings that EEG abnormalities are found in 50% of autistic individuals (Bailey, Phillips, & Rutter, 1996). Unspecified abnormal electroencephalogram, auditory brainstem response, and oculomotor findings have been reported in ASC, as well as in low-functioning autism (Gillberg, 1989).

Many studies have attempted to find neurochemical deficits in autism following the hypothesis that autism may be a metabolic disease which arises from a defect in some biochemical pathway. As with neurophysiological investigations, despite almost 40 years of research in the area, there have been no consistent findings implicating a biochemical basis for autism. However, Bailey, Phillips, and Rutter (1996) suggested that raised serotonin levels (hyperserotonemia) may be implicated in autism, possibly in up to one third of people with autism (Tsai, 1999), but again, results suffer from a lack of replication.

Several case studies have documented specific medical disorders in association with autism symptoms including: tuberous sclerosis (Gillberg, Gillberg, & Ahlsen, 1994); Marfan-like syndromes (Tantam, Evered, & Hersov, 1990); Kleine-Levin syndrome

(Berthier, Santamaria, & Encabo, 1992); fragile X syndrome and other chromosomal anomalies (Bailey *et al.*, 1996).

Neuropsychology

Although a diagnosis of autism spectrum conditions is based on clinical symptoms and early history, cognitive assessment has a crucial role in differential diagnosis and planning sensitive interventions. The specific neuropsychological impairments that may be exhibited by children with autism are deficits in explicit memory, in establishing rules for governing rewards, and in working memory, planning, and response inhibition (Dawson, 1996). Minshew and Goldstein (1998) hypothesised that Asperger Syndrome is a selective disorder of complex information processing abilities and as a disorder of multiple primary deficits. Their review of research has shown that people with autism have specific cognitive batteries with normal performance on tasks requiring rote, mechanical, or perceptual processes, and poor performance on tasks requiring higher-order conceptual processes, reasoning, interpretation, integration, or abstraction. However, there is substantial variability in the intellectual profiles of people with autism, and research has not found any cognitive pattern that confirms or excludes a diagnosis of autism.

In terms of differentiating between Asperger Syndrome and other points along the autistic spectrum, normal or near-normal Full Scale IQ is considered a core element in the profile of a person with AS. Klin, Volkmar, Sparrow, Cicchetti, and Rourke (1995) compared the neuropsychological profiles for both diagnosed groups using stringent ICD-10 (World Health Organisation, 1992) research criteria. They found that 11 areas discriminated between the two conditions. They suggested that Asperger Syndrome most resembled a cluster of neuropsychological assets and deficits termed non-verbal learning disabilities (NLD). Study participants (n=21) diagnosed with Asperger Syndrome had a verbal-performance discrepancy, with verbal IQ being consistent universally higher than performance IQ. Study participants (n=19) with high functioning autism (HFA) displayed no difference between verbal IQ and performance IQ scores. However,

Manjiviona and Prior (1999) undertook a similar study, with similar sample size, yet failed to discover any significant verbal IQ superiority over performance IQ among children with Asperger Syndrome. They suggested that the cognitive profiles and consistency of the profile across the groups "argue against the notion of a definitive and distinctive neurocognitive profile in Asperger syndrome" (p. 344). The authors added that the significantly higher full scale IQ level found in the Asperger Syndrome children "suggests that this may be the cardinal feature that clinicians react to when making a diagnosis" (*ibid.*), and that "children with Asperger Syndrome "probably represent the upper end of the spectrum of autistic disorders" (p. 350). In conclusion, the authors add weight to a conclusion that there is little to differentiate between children diagnosed with high functioning autism and Asperger Syndrome.

Outcome for adults with autistic spectrum conditions

As the literature into autistic spectrum conditions escalates, a growing number of researchers and clinicians have noted the rather unspectacular finding that children with ASC grow up to become adults with ASC. One of the most interesting and useful sources of data on outcome comes indirectly from observing those parents or other relatives of children with ASC, who themselves appear to have ASC. From observations of parents with ASC traits, it is clear that ASC does not preclude the potential for a more "normal" adult life. Commonly, these adults will gravitate to a job or profession that relates to their own areas of special interest, sometimes becoming very proficient. The available data does suggest that, compared to other forms of autism spectrum disorders, children with AS are more likely to grow up to be independently functioning adults in terms of employment, marriage and family.

Kanner (1971) noted that adolescents with autism may vaguely realize that they are different from others and that they are excluded from many interpersonal relationships. Although they amass many facts about the world, their knowledge remains fragmented and they continue to have difficulty in the meaningful, integrated execution of their knowledge (Frith, 1991), lacking the "common sense" of the everyday. In adulthood, the

ASC individual may become superficially well adapted but typically remain egocentric and isolated, their use of language and gestures remaining stilted, seldom entering into the natural flow of conversation (Bishop, 2000).

The vast majority of published research on autism has dealt with children, although some recent texts have addressed the lack of interest in adults (e.g., Howlin, 1997; Tantam, 2000). There have been a handful of systematic long-term follow-up studies for children with ASC. Three factors contribute to the lack of information: the high cost of longitudinal research; the recent inclusion of Asperger Syndrome in differential diagnostic systems; as well as the belief among psychologists that the prognosis in autistic spectrum disorders is very poor. Howlin and Goode (1998) reported that studies conducted since 1980 suggest that there have been improvements in levels of functioning of people with autism. De-institutionalisation of people living with mental illness and developmental disabilities provides clear guidelines of what does (and does not) work in supporting individuals of "difference" in our society. Just as rates of institutionalisation have fallen dramatically in the last twenty years, participation in work or training has climbed equally dramatically (Howlin, 1997). It would appear that at least three factors related to early development are associated with later outcome: the development of simple communicative language by age 6; non-verbal scores at, or above, the mildly retarded range, and the presence of additional interests, such as mathematics, computing, or music (Howlin & Goode, 1998). Early diagnosis and appropriate intervention appears to hold the key to improved quality of life among adults with ASC, as appropriate interventions can be put into place to support the child, both educationally and psychologically, as well as through programs to provide and long-term support for the family. Yet we know that this is not the norm. Gillberg (1998) has estimated that perhaps 30-50% of all adults with Asperger Syndrome are never evaluated or correctly diagnosed. These "normal Aspergers" are viewed by others as "just different" or eccentric, or they may be diagnosed with other psychiatric disorders (Perlman, 2000) . It has been suggested that some of these highest functioning and brightest individuals with Asperger Syndrome represent a unique resource for society, having the single

mindedness and consuming interest to advance our knowledge in various areas of science, math, engineering, etc (Tantum, 2000).

Last year, the National Autistic Society in Great Britain published the results of a survey of 1 200 parents of adults with autism or Asperger syndrome (Barnard, Harvey, Potter, & Prior, 2001). For data collection, the authors requested that adults place their children within three categories of functioning: lower, medium, and higher. Higher functioning referred to "the more able end of the autistic spectrum where the true level of disability and vulnerability may be masked by an average or above average intelligence (p. 4)." The report found that fewer than 6 per cent of adults on the higher autistic spectrum were in full time work. Forty-nine per cent of adults who were "high-functioning" or diagnosed with Asperger Syndrome were still living at home with their parents. Seventy per cent of parents felt their son or daughter was unable to live independently without support, while less than 10% of adults with autism or AS could manage most household duties, such as shopping, making meals, washing, and budgeting without help. Of those adults within the higher-functioning group, only 3% of adults were living fully independently, and a further 8% were living independently with regular professional or family support. One third of the parents had reported that their son or daughter had experienced mental health problems, and for those diagnosed after age 30, one half of the parents reported mental health problems. Of those people who experienced mental health problems, more than half had suffered depression, while 8 per cent had reported suicidal thoughts or had attempted suicide.

Psychiatric Comorbidity

There are few studies of comorbid psychiatric disorders in people with autism spectrum conditions. Most studies of comorbidity are limited by small sample size as well as problems in uniformly defining ASC. Although Kanner (1943) believed there to be a link between childhood autism and adult psychosis, there is little evidence to prove this link (Howlin & Goode, 1998). Volkmar and Klin (1997) stated that some studies found an association between Asperger Syndrome and Tourette's Syndrome, a finding that they

were unable to replicate when examining a larger pool of Asperger Syndrome subjects. Other disorders which may accompany ASC include obsessive-compulsive disorder, and Attention Deficit Hyperactivity Disorder. The comorbidity of certain conditions also may vary according to developmental level. For example, ADHD appears to be more common in younger Asperger Syndrome individuals while depression may be more apt to emerge in adolescence and adulthood (Volkmar & Klin, 1997). Researchers have proposed that at least some of the adults previously identified as Schizoid Personality Disorder may actually be displaying the manifestation of Asperger Syndrome in adulthood. Schizoid Personality Disorder is an Axis II diagnosis (APA, 1994) from adult psychiatric nomenclature but may actually represent a form of autism in adults (Wolff & Barlow, 1979). Wolff and Chick (1980) followed children diagnosed with Schizoid Personality Disorder and those diagnosed with Asperger Syndrome into adulthood, concluding that the disorders were essentially identical. However, they noted that the group identified as "Schizoid" demonstrated more distractibility and less perseveration on cognitive tasks than the subjects identified as Asperger Syndrome. Other researchers have objected to the idea of collapsing these diagnostic categories into one entity (Tantum, 1988).

The recent study by the National Autism Society confirmed the empirical and clinical evidence that the most prevalent comorbid conditions are related to anxiety and depression (Bolton *et al.*, 1998). Wing (2000) cited the limited research and her clinical experience to support her belief that adults with Asperger syndrome have high rates of depression and suicidal behaviour; and that the risk for acquiring these disorders grows as the individuals age (Howlin & Goode, 1998). To compound the problem, people with ASC are likely to be misdiagnosed with other mental health problems earlier in life, or not diagnosed at all (Burger & Lang, 1998).

Intervention Strategies

Despite greater use of pharmacotherapy in managing people with ASC, there is little empirical data to support the practice (Martin *et al*, 2000). All of the drug treatments are

not specific to ASC, and are used across the range of child and adult psychiatric disorders. For example, the use of serotonin-related agents such as fluoxetine (Prozac) has shown some effectiveness in treating depressive symptoms and obsessive-compulsive behaviours (Tsai, 1999). One atypical antipsychotic, risperidone, acting on both the dopamine and serotonin receptor sites, has shown positive effects in reducing aggression and hyperactivity among children and adolescents with ASC (Nicholson, Awad, & Sloman, 1998), although there are some reports of tardive dyskenisia among some children taking the medication (Tsai, 1999).

Volkmar and Klin (1997) suggest that skills and concepts be taught in an explicit and rote fashion where possible, employing a parts-to-whole verbal instructional approach. Learning strategies will be most effective when based on the specific pattern of strengths and deficits demonstrated in neuropsychological assessment. If motor and visual-motor deficits are identified, physical and occupational therapies are indicated. Interventions designed to improve communication and social skills may have any or all of the following as its goals, depending on the specific needs of the individual. These include enhanced awareness of one's own nonverbal behaviors (e.g., the use of verbal inflection, eye contact, and gaze in social interaction); verbal decoding strategies for more accurately interpreting the nonverbal behavior of others; more integrated processing of visual and auditory stimuli; as well as improved social awareness and perspective taking.

Treatments for autism spectrum conditions remains primarily symptomatic and supportive. As lifelong disorders, treatment needs and approaches will change with the individual development. Psychoactive medications may be used to target specific symptoms but are generally not indicated in ASC. In high-functioning individuals with symptoms of obsessive-compulsive disorder, anxiety, or depression, pharmacotherapy may be helpful (Wing, 1991). Insight-oriented psychodynamic therapy has been extensively used to treat these disorders with little objective evidence of success (Campbell, Schopler, Cueva, & Hallin, 1996). Tony Attwood (1999) has developed some guidelines for modifying cognitive behavioural therapy for people with Asperger Syndrome. Cognitive behavioural therapy offers some promising opportunities for

treatment, as it is based on education of the client, and can be used to teach social reasoning skills in a "scientific" manner. If people with ASC develop depression due to the sense of not having friends, or being excluded from social relationships, the therapist can give assistance in these domains, rehearsing different scenarios and enhancing opportunities for social interaction. In some respects, the therapist can act as a "cultural interpreter" for the person with ASC, helping him or her to understand why people act the way they do, and suggesting how best to respond in these situations.

Direct teaching of theory of mind skills has proved effective in working with younger children with Asperger Syndrome (Howlin, Baron-Cohen, & Hadwin, 1999), although whether they have improved social competence is less clear. Social skills groups have been run with adolescents, but again, good results achieved within the group have not been shown to generalise to other settings, and have degraded over time (Marriage *et al.*, 1995).

Summary

Community mental health centres, special education services, and private psychiatric providers have been inundated with referrals for assessment and treatment of young and not so young people who could be diagnosed as having an autism spectrum condition. The response is often determined by individual interest and experience in the population. The resources simply do not exist to cope with the unique challenges of an increasing group of people. Diagnostic manuals have proved a mixed blessing; offering greater reliability at the cost of diminished construct validity. Future work needs to concentrate on sophisticated understandings of cognitive difference, providing tailored support to children and their families, as well as newly diagnosed adults. The isolation and despair that can often accompany the condition can be ameliorated by a "life span" developmental approach, identifying children as early as possible, supporting families with information and contact with other parents, initiating individualised educational strategies, using strengths of visual thinking, a commitment to nonviolence at school, and support and assistance in transition from school to the workforce.

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