

Emanuel Miller lecture: Confusions and controversies about Asperger syndrome

Uta Frith

UCL Institute of Cognitive Neuroscience, UK

Background: Hans Asperger drew attention to individuals who show the core symptoms of autism in the presence of high verbal intelligence. **Methods:** A review of the literature explores current issues concerning the diagnosis and nature of Asperger syndrome. **Results:** The behavioural and neurophysiological evidence to date suggests that Asperger syndrome is a variant of autism typically occurring in high-functioning individuals, and not a separate disorder. One of the problems of diagnosis is that the typical impairment of social communication may be difficult to identify in early childhood, and can be camouflaged in adulthood by compensatory learning. The range and nature of the social impairments in Asperger syndrome are still in need of investigation, but appear to be less severe than in autism. Experimental evidence suggests that individuals with Asperger syndrome may lack an intuitive theory of mind (mentalising), but may be able to acquire an explicit theory of mind. Brain imaging studies pinpoint a network that links medial prefrontal and temporal cortex as the neural substrate of intuitive mentalising. This network shows reduced activation and poor connectivity in Asperger syndrome. While some individuals with Asperger syndrome have written eloquently about their lives, their ability to talk about their own emotions appears to be impaired (alexithymia). This impairment may be linked to depression and anxiety, which is common in adulthood. Little is as yet known about the often considerable cognitive strengths in Asperger syndrome, or about the difficulties observed in higher-level executive skills. **Conclusions:** Studies are needed that define the developmental course of the disorder and the nature of the strengths and weaknesses in both social and non-social domains. This requires more sensitive assessment instruments than are currently available. Questions about the prevalence of Asperger syndrome, about associated and secondary features, and about optimal education and management, urgently call for such studies. **Keywords:** Autistic disorder, emotion, executive function, mentalising impairment, social cognition, theory of mind.

Emanuel Miller's (1893–1970) biographical details suggest some significant parallels to Hans Asperger (1906–1980). Both men were among the first child psychiatrists in their respective countries, Great Britain and Austria, and founded lasting institutions that care for children's mental health. Both emphasised the critical importance of social work and education. From all accounts both were Renaissance men who built up large libraries testifying to their wide interests from medicine to philosophy and the arts. Asperger's working life, as well as Miller's, was deeply marked by the historical cataclysm of the first half of the 20th century. The wars that wrecked Europe had psychiatric consequences of a kind that clinicians had hardly experienced before. Just as in London, where Emanuel Miller treated shell-shocked patients returning from the First World War, so in Vienna, Erwin Lazar, Hans Asperger's teacher, set up centres for severely deprived children, and devoted his clinical expertise to alleviating their condition. A tradition of remedial education was started, which Asperger continued throughout his life.

However, what Hans Asperger is chiefly remembered for is that he was one of the two farsighted clinicians who identified and labelled the condition that is now known as autism. Leo Kanner, who was working in Baltimore, published his seminal paper on autistic disturbances of affective contact in 1943,

while Hans Asperger published his paper on autistic psychopathy in 1944. Neither of them knew the other, and they appear to have kept a professional distance. Each appeared to believe that the other was writing about a different type of child. However, a close analysis of the case descriptions demonstrates a great deal of overlap (Wing, 1991). Asperger's most famous cases had high intellectual ability and precocious language, while no such case was reported by Kanner, but he also described others who had low ability and poor language. In this sense Hans Asperger anticipated the autism spectrum (Wing, 2000). Asperger viewed autism as a constitutionally given personality type, albeit a pathological one. His term 'autistic psychopathy' strikes us as odd today, but at the time it merely indicated a stable condition as opposed to a progressive disorder. Asperger pointed out that improvement often occurred over the course of development, in contrast to the deterioration typically seen in psychotic patients. Kanner too reported improvements with development in his original cases, but gave a less optimistic picture (Kanner, 1971).

Hans Asperger sought to increase awareness of autism since he believed that autistic individuals were likely to suffer from tragic misunderstanding and consequent maltreatment by others. He was not only concerned about the children he met in his

paediatric clinic, but also about adults, including some of the parents, who had problems of isolation and employment that stemmed from their condition. He advocated an educational approach, where teachers would work with them rather than against them, building on their strengths and circumventing their weaknesses. As these children did not function well in a group, he argued that individual attention was necessary. He was convinced they learned best when guided by their own special interests. He observed that teachers who were objective, and to some extent detached, in their methods often obtained good results. Asperger persuasively argued that individuals with autistic disorder could not only be successful in their chosen profession, but were of high value to society. He believed that the education of children with autism must be based on a deep understanding of the condition. These insights are still relevant today.

The rise of Asperger syndrome

Hans Asperger's work led to the recognition of children and adults with good verbal ability who appeared to have autism in a milder form. These individuals had previously been almost invisible, being considered amongst eccentrics, obsessives, or borderline and schizotypal personalities. One of the reasons that they are now visible is precisely because they can be given the label Asperger syndrome. The label Asperger syndrome began to come into use after Lorna Wing's influential paper published in 1981, which brought Asperger's contribution to the attention of the English-speaking world. However, Asperger's original paper was not translated into English until 1991 (Frith, 1991).

In her 1981 paper Wing argued that autism could not be captured by a few static criteria, but that there were striking individual differences and changes with development. She suggested that autism could be manifest in socially odd behaviour just as much as in aloofness. Wing found that the children with 'odd' rather than aloof social behaviour were often highly talkative and reminiscent of some of Asperger's cases. Wing did not wish to imply that these children were not autistic. Instead she proposed a spectrum of autistic disorders with different degrees of severity along each of the dimensions of social impairment, communication impairment and restricted repetitive behaviour. The assumption of an autism spectrum made sense of behavioural changes over time, often from aloof to socially odd, and made sense of the fact that genetically related individuals can show very different manifestations of autistic disorder (e.g., Burgoine & Wing, 1983; Le Couteur et al., 1996).

Somewhat surprisingly, the label Asperger syndrome rapidly became widely used. This was despite the fact that many people were uncertain as to how

to pronounce the name (the *g* in Asperger is pronounced as in *get* and not as in *gem*). Clinicians who were already aware of unusual children, sometimes described as 'loners' (Wolff, 1995), and had not hitherto connected them to autism, saw the relationship to Asperger syndrome. The label high-functioning autism also began to come into use and was championed by some authorities to refer to the increasingly recognised group of able children with autism, in reference to either verbal or non-verbal intelligence. Adult psychiatrists too became interested and started to reclassify some of their most puzzling patients on the borders of personality disorder and schizophrenia as cases of Asperger syndrome (e.g., Tantam, 1991).

Descriptions of Asperger syndrome in the media created strong resonance in the general public. Many people thought they knew somebody who might have Asperger syndrome. Furthermore, many sufferers who had been searching for an explanation of their problems were now beginning to diagnose themselves. From being virtually unknown only twenty years ago, Asperger syndrome has now become almost a household word. A comprehensive volume edited by Klin, Volkmar, and Sparrow (2000), however, demonstrates that what we know about Asperger syndrome is still very scant and controversies about the nature of the disorder are far from resolved. Notwithstanding this uncertainty, there appears to be a pressing demand for practical help and guidance, as reflected in a large number of websites (see, for instance, tonyattwood.com.au).

Questions about the diagnosis of Asperger syndrome

Clinical and practical knowledge about Asperger syndrome is accumulating, but we do not yet know how frequent Asperger syndrome is. Of course, we can only know how many cases there are if we know what a case is! There is reason to believe that in current clinical practice the label Asperger syndrome is used rather indiscriminately. Asperger syndrome has a special cachet that hints at superior intelligence and perhaps even genius. The label high-functioning autism, because it is sometimes applied to individuals who are only relatively high functioning, possibly carries less of such an implication. In reality, the label is given to many children and adults on the autism spectrum who are simply atypical in their presentation, talkative rather than withdrawn, but not necessarily of high ability. Asperger syndrome, or high-functioning autism as identified in many clinics today, comprises a far too heterogeneous group, including cases of well below average ability and poor social adaptation, as well as those of superior intelligence and good social adaptation.

Do the diagnostic manuals help in the definition of Asperger syndrome? Only up to a point. In fact it has

been argued that Asperger syndrome is logically impossible to diagnose according to DSM-IV (Mayes, Calhoun, & Crites, 2001) and future editions will undoubtedly see revisions. The currently most workable distinction between a diagnosis of Asperger syndrome and a diagnosis of autism is that early language and cognitive function should not be delayed in Asperger syndrome. This requirement (that single words are used by age 2 and communicative phrases by age 3 years) allows a reasonably consistent diagnostic differentiation, but is not without problems. It does not necessarily mean that language acquisition was normal. First, it relies on retrospective reports. Second, some of these reports imply that acquisition was atypical. The vocabulary of children who are later diagnosed with Asperger syndrome is often described as precociously adult, meaning that it contains rare words, which are not normally used by children. Third, communicative phrases, even if present at age 3, do not guarantee good language understanding. Obviously, gross delay in language and cognitive development can be ruled out with retrospective questioning, but contemporaneous evidence would be better. Given that such evidence is as yet lacking, it is by no means clear whether the presence of speech by age 2 to 3 should remain a touchstone for differential diagnosis. Another possibility would be to make verbal ability, measured in later childhood or adulthood, a discriminating criterion.

To define Asperger syndrome, can we find guidance from Asperger's original paper? Again, only up to a point. Asperger's first descriptions included intellectually able and verbally articulate individuals but also cases that did not meet these criteria. Thus, not all cases described by Hans Asperger had what we would now classify, according to DSM-IV, as Asperger's Disorder (Miller & Ozonoff, 1997; Hippler & Klicpera, 2003).

How easy is it to separate Asperger syndrome from autism?

In a black and white world, two alternatives present themselves: Asperger syndrome is an entirely separate disorder from autism with a different genetic, neurophysiological and cognitive basis. This is contrasted with the idea that Asperger syndrome is the same disorder, but a variant, with essentially the same contributory causes. In between these extremes the fact must be accommodated that Asperger syndrome and autism are both extremely heterogeneous disorders. One possibility is that, in some cases at least, Asperger syndrome provides a model of 'pure' autism where intellectual disability does not confound the clinical picture. It is conceivable that a more circumscribed type of brain abnormality is less detrimental to the development of other brain systems. This would imply a seemingly

'milder' disorder because it is not accompanied by other problems, such as speech impairment or motor dyscoordination. With compensatory learning, such a disorder could remain hidden for a long time. However, as we shall see below, Asperger syndrome, even in relatively pure form, may be neither mild nor hidden. Furthermore, associated learning disabilities in Asperger syndrome appear to be common and throw doubt on such a simple picture.

The alternative view that Asperger syndrome is a separate developmental disorder is also subject to doubt. A number of studies, recently reviewed by Reitzel and Szatmari (2003), have looked for differentiating neuropsychological profiles which would point to a different brain basis. These studies invariably found that high-functioning autism groups achieved on average poorer verbal scores than Asperger syndrome groups. However, this seems a potentially circular finding, considering that the diagnosis of Asperger syndrome is likely to select for high verbal ability. On the other hand, studies are not consistent in finding poorer non-verbal scores, greater spatial or motor difficulties in Asperger compared to high-functioning autism groups. The idea that Asperger syndrome could map onto the psychometrically defined group of children with non-verbal learning difficulties (NVLD) was pursued by Klin and colleagues (Klin, Volkmar, Sparrow, Cicchetti, & Rourke, 1995). Specific difficulties in maths, as well as in perceptual and motor tasks, a pattern thought to be typical for NVLD children, are often present in Asperger syndrome, but not necessarily so.

Several arguments speak for the possibility that Asperger syndrome has the same aetiology as autism. First, there is the genetic argument. Cases of Asperger syndrome and cases of autism may occur in siblings and presumably the same genetic predisposition gave rise to both disorders. The assumption is that the biological cause is similar even if the manifestation of the disorder is very different. Second, there is the outcome argument. Older individuals with high-functioning autism share so many features with individuals with Asperger syndrome that they are often difficult to tell apart (Szatmari et al., 2000; Howlin, 2003). Indeed, in adulthood a proportion of individuals previously diagnosed as suffering from autism and showing early language delay can become almost indistinguishable in their behaviour from those with high-functioning autism who did not have this delay (Gilchrist et al., 2001; Mayes & Calhoun, 2001). Third, there is the neuro-anatomical argument, for which a sparse amount of evidence exists. An examination of the cellular structure in selected brain areas (Casanova, Buxhoeveden, Switala, & Roy, 2002) suggests abnormalities in the minicolumnar organisation similar to those reported in autism. Specifically, minicolumns were smaller, and their component cells were more dispersed than normal. Bauman and Kemper (2003) concluded

that there are certain features of brain pathology in autism that are present regardless of IQ.

Thus, the currently prevailing view is that Asperger syndrome is not an essentially different disorder from autism, but a variant of autism, and located at the milder end of the spectrum of autistic disorders. This view is consistent with the incomplete, but growing information that we have about genetic, neurophysiological, cognitive and behavioural information: autism and Asperger syndrome are highly related subtypes of disorders of the autism spectrum. On the other hand, we are far from a consensus. Rhinehart, Bradshaw, Brereton, and Shaw (2002), for instance, believe that, despite the demonstrable clinical overlap between Asperger syndrome and autism, it is still premature to rule out the possibility that these disorders may be clinically and neurobiologically separate.

Even if the prevailing view favours a continuum, it does not follow that we should abandon the label Asperger syndrome and instead talk of 'mild autism' or 'autism with high verbal ability'. At present, the label 'high-functioning autism' is much used and is often interchangeable with Asperger syndrome. However, it does not require the absence of significant delay in early language and cognitive function. Time will tell whether both labels will remain in use in the future. Wing (1981) proposed the label Asperger syndrome for purely pragmatic reasons, to raise awareness of this particular form of autistic disorder. These reasons are important. Given the question of educational placement, there is clearly a need to differentiate verbally able children who have strong academic interests from those who have severe problems with language and show other evidence of learning disability. Abandoning the label Asperger syndrome would lose the historical context and the wealth of information that has now been accumulated around it. The term has contributed significantly to an increase in the awareness of autistic disorder in the general public. One reason that the label was so keenly taken up was that it helped ordinary people to understand what might be the matter with the strange person with narrow obsessive interests and social ineptness whom they might have come across in their everyday environment. It makes sense to provide a special label to the least severe and least handicapping form of autistic disorder. It is less stigmatising and allows for the fact that at least some individuals are able to cope with minimal supervision or specialist help.

Questions about the developmental course of Asperger syndrome

A necessary step towards clarifying the nature of Asperger syndrome is the study of the developmental course of the disorder. An important clue is offered by the fact that, on average, the diagnosis of Asper-

ger syndrome is made much later than the diagnosis of autism. Howlin and Ashgarian (1999) wrote to 614 parents of children with autism and 156 with Asperger syndrome. The average age when a diagnosis of autism was confirmed was around 5.5 years compared to 11 years for Asperger syndrome. Parents of children with a diagnosis of autism were generally aware of problems in their child's development by 18 months of age; but in the Asperger group only from around 30 months of age. Initial worries in both groups centred on abnormal social development, but parents of children with Asperger syndrome were less likely to have noted communication problems. Why does the disorder seem to be hidden in infancy and early childhood? Do we simply lack sensitive tests? Alternatively, do the problems arise later and only when the demands of the social world become too heavy?

It is possible that even at an early age the problems of Asperger syndrome are obscured by the specific strengths that are often associated with the disorder. The child who shows 'adult' or precocious language, who has special interests and excellent memory, is unlikely to make parents rush to a clinic, even if this child does not interact with peers. Parents may overlook this sign when the child at first seems more advanced than potential playmates. It is unknown whether children later diagnosed with Asperger syndrome present behaviour problems that are similar to those that many young children with autism present, such as temper tantrums, resistance to toilet training, restricted food preferences, and sleep disturbance. Even if it turns out that behaviour problems are not a major issue, this may not mean that these children are 'easy'.

Just as puzzling as the fact that Asperger syndrome leads a somewhat hidden existence in early childhood is the fact that it can be hidden again in adulthood, but usually not forever. This is particularly true in cases where intellectual ability is high and where environmental support is good. In these cases a short encounter or routine interaction will not reveal anything unusual. However, over time and in unexpected situations, it appears that the façade of normality cannot be kept up. A number of spouses of Asperger syndrome individuals tell how they were taken in by their partners' superficially normal presentation and how they arrived only later at the difficult discovery that life with an Asperger syndrome individual is very different from normal (e.g., Slater-Walker & Slater-Walker, 2002).

Is Asperger syndrome a 'mild' disorder?

Some individuals with Asperger syndrome lead near normal lives and show excellent adaptation. Others can hardly cope and need constant supervision. Clearly, not all cases show good compensation and clearly, there are different degrees of severity. How-

ever, in some cases the disorder can *appear* to be more severe, not necessarily because the core symptoms *are* more severe, but because additional clinical disorders aggravate the picture (Gillberg & Billstedt, 2000).

In children with Asperger syndrome, additional motor coordination difficulties, as well as specific learning difficulties in maths and reading have been documented. For instance, Green et al. (2002) found motor impairment in every single case they assessed. Reitzel and Szatmari (2003) reported that out of a group of 27 children with Asperger syndrome, assembled for a follow-up study at age 4 to 6, from six centres in Southern Ontario, 21% had specific reading difficulties and 46% specific maths difficulties, when aged between 9 and 13 years. Specific difficulties were defined in terms of a 15-point discrepancy with non-verbal IQ (which had to be above 80). It is likely that additional learning difficulties impede academic progress and need specialist remediation.

While in the case of children issues of developmental delay are prominent, in adults a variety of psychiatric problems can occur, although at present the frequency of such problems is unknown. Problems include obsessional compulsive disorder, anxiety and depression, and are likely to have severe repercussions on the ability to cope with everyday demands. The relevant signs and symptoms are not always reported by the sufferers and recognised by their families, but specialist treatment is necessary and can alleviate the symptoms.

When compared to the devastating effects of language impairment and learning disability in autism, Asperger syndrome indeed appears to be 'mild'. However, it cannot be regarded as a 'mild' disorder. Many examples exist to demonstrate that it can impose a heavy burden on the sufferer, the family and the wider community.

Conjectures about the social impairments in Asperger syndrome

Just like other types of autistic disorder, the hallmark of Asperger syndrome is a failure in social learning and social awareness. But exactly how should one characterise this failure in Asperger syndrome? In contrast to people with autism, these individuals often stand out by their desire for social interaction and their wish to have friends and spouses.

One way to describe the social impairment in Asperger syndrome is as an extreme form of egocentrism with the resulting lack of consideration for others. The self-absorption and disregard of others is not like the strategy that a normal selfish person might deliberately adopt and flexibly use according to what is currently in his or her best interest. Autistic egocentrism, by contrast, appears to be non-deliberate and not determined by what might currently be in the best interest of the individual.

This egocentrism seems to present a huge difficulty in forming successful long-term interpersonal relationships. Spouses and family members can experience bitter frustration and distress. They are baffled by the fact that there is no mutual sharing of feelings, even when the Asperger individual in question is highly articulate. One obstacle seems to be an inability on the part of the person with Asperger syndrome to put themselves into another person's shoes and to imagine what their own actions look like and feel like from another person's point of view.

Another way to describe the social impairment is as a failure of empathy, involving a poor ability to be in tune with the feelings of other people. Baron-Cohen and colleagues compare lack of empathising with an excess of systemising, which refers to a particular engagement with the physical world (e.g., Baron-Cohen, Richler, Bisarya, Gurunathan, & Wheelwright, 2003). Tantam and others have long emphasised a lack of a basic emotional resonance with other individuals, which is often perceived as callousness and coldness by others (Tantam, 1991).

Is there a basic deficit in recognition of emotions? Would such a deficit account for a lack of emotional resonance as well as lack of empathising and egocentrism? The results from existing studies are inconsistent. One study (Howard et al., 2000) showed impairment in fear recognition, but another did not (Adolphs, Sears, & Piven, 2001). Critchley et al. (2000) reported that Asperger syndrome and high-functioning individuals with autism, in contrast to controls, did not activate a cortical face area when explicitly appraising expressions, and showed less activation in the left amygdaloid and cerebellar regions when implicitly processing emotional facial expressions. Shamay-Tsoori and colleagues (Shamay-Tsoori, Tomer, Yaniv, & Aharon-Peretz, 2002) described two cases of adolescents with Asperger syndrome who showed extreme deficits on measures of empathy, but did not have significant impairments in their ability to recognise emotions or the ability to create a mental representation of another person's knowledge. However, both adolescents were unable to integrate the emotional content with mental representations and deduce the other person's emotional state. The authors suggest that impaired empathy in individuals with Asperger syndrome may be due to impaired integration of the cognitive and affective facets of the other person's mental state.

Impairments in the understanding of complex emotions are generally held to be typical in Asperger syndrome. Baron-Cohen, Jolliffe, Mortimore, and Robertson (1997) showed that the recognition of complex emotions from the eye region is impaired in high-functioning people with autism and autistic disorder. These individuals were unimpaired when recognising gender from the eye region of the face, and when recognising emotions from the whole face. The authors suggest that this provides evidence for specific empathising deficits (that is, difficulty in

attributing feelings and other mental states to other people). Blair (2003) concluded in his recent review of the field that only the perception of complex emotions is impaired in autism, and that this impairment can be seen as part and parcel of an impairment in understanding others' mental states.

Apart from a poor understanding of one's own and other people's inner states, problems are also evident in other aspects of social understanding. One important and robust finding is the poor recognition of faces (e.g., Critchley et al., 2000; Schultz et al., 2003). Klin and colleagues (Klin, Jones, Schultz, Volkmar, & Cohen, 2002) have demonstrated the dramatically different eye gaze pattern when watching social scenes in persons with high-functioning autism. Thus, these individuals tended to look at mouth rather than eye regions in faces. Moreover, when shown emotionally laden interactions in a movie (the movie shown was *Who is afraid of Virginia Wolf?*), their eye gaze was liable to stray off course and into the background rather than follow the tense dialogues between people by looking at their faces. It remains to be seen whether abnormal looking patterns and poor face recognition are separable components of social impairment. Klin, Schultz and colleagues suggest that face processing abnormalities might be a developmental consequence of failure to look preferentially at faces. While most children quickly become 'face experts', children with autism may not process faces as special stimuli.

Communication impairments, which are evident even in highly verbal individuals (e.g., Ziatas, Durkin, & Pratt, 2003), also call for systematic analysis and explanation. Anecdotally, people with Asperger syndrome have often far superior written than spoken language, and often state that they prefer to communicate in writing. Presumably one advantage is that this avoids the constant strain of having to decode another person's verbal and non-verbal communicative signals. Furthermore, in this typically off-line situation there is time to think and to use an explicit theory of mind to compute effects on the recipient of the message.

The diagnosis of communication impairment in individuals with high verbal ability is not easy. We still lack practical tools for assessing the use of gesture and voice. Shriberg, Paul, McSweeny, Klin, and Cohen (2001) documented inappropriate vocalisations in the domains of phrasing, stress, and resonance in Asperger syndrome. They reported that while speakers with Asperger syndrome were significantly more voluble than speakers with high-functioning autism, there were otherwise few statistically significant differences between the two groups.

The conjectures about the social impairments in Asperger syndrome vary widely, but agreement cannot be reached until the precise nature of this impairment has been clarified. However, clarification is hampered by a lack of psychometrically validated tests. The experimental tasks in current use suggest

that there are large individual differences in the social competence of individuals with Asperger syndrome, overlapping with those of normally developing individuals. Some surface behaviours that index poor social competence will look identical in both groups, for instance poor face recognition, lack of empathic resonance, avoidance of eye contact, and preference for written over spoken communication. However, their causes are likely to differ for the two populations. It would be strange to assume that these behaviours must necessarily be caused by a faulty neuro-cognitive mechanism. Such an assumption is only justified for the case of autistic disorder, which has a biological cause, and where ultimately a biological marker will validate the behavioural diagnosis.

A failure of intuitive mentalising

It is likely that failure in a number of different neuro-cognitive mechanisms underlie the range of social impairments in Asperger syndrome. One such mechanism, which enables the automatic attribution of mental states to others, has been extensively tested over the past 20 years (see reviews in Baron-Cohen, Tager-Flusberg, & Cohen, 1993, 2000). The empirical work includes high-functioning individuals with autism and Asperger syndrome (e.g., Baron-Cohen et al., 1997; Happé, 1994; Baron-Cohen, O'Riordan et al., 1999; Klin, 2000; Jolliffe & Baron-Cohen, 1999; Rutherford, Baron-Cohen, & Wheelwright, 2002). There has been some success in identifying the brain basis of this mechanism. Furthermore, functional brain imaging studies of mentalising have been carried out with Asperger syndrome individuals.

The empirical studies of mentalising failure in autism suggest that it can be detected very early, that is from around 18 months (Baron-Cohen et al., 1996; Charman et al., 1997). This age is considered to be the earliest for unequivocal evidence of implicit mentalising in normally developing children, as manifest in sophisticated joint attention and pretend play. Mentalising failure is seen in the everyday inability of young children with autism to tell lies, keep secrets and predict the behaviour of others on the basis of mental states. An example of a laboratory test with relevance to real-life behaviour is the penny hiding game. Typically, the child with autism can hide the penny perfectly well in a closed hand, but will give the hiding place away, for instance by leaving the other hand open (Baron-Cohen, 1992).

What is the evidence for mentalising impairments in Asperger syndrome? Unlike in the case of autism, screening at age 18 months for the presence of joint attention and pretend play did not reliably pick out children later diagnosed with Asperger syndrome (Baird et al., 2000; Cox et al., 1999). Comparisons of mentalising tasks in older children show that

children with Asperger syndrome show impairment, but to a lesser degree than children with autism (Ziats, Durkin, & Pratt, 1998). Studies in adults too tend to indicate only subtle impairments as revealed in inconsistent and slow responses on advanced and naturalistic tests (e.g., Roeyers, Buysse, Ponnet, & Pichal, 2001; Kaland et al., 2002; Channon et al., 2001).

Is mentalising impaired in Asperger syndrome but less than in autism? It is also possible that the impairment is not less, but is camouflaged (e.g., Frith, 2003). Highly verbal individuals can give well-reasoned answers in theory of mind tasks, perhaps because their intelligence allows them to use logical inferences. For instance, they can succeed in understanding complex scenarios, such as tracking where Mary believes that John thinks he can buy a coat (Bowler, 1992). However, displaying an explicit theory of mind does not necessarily imply an intuitive mentalising ability.

The distinction made here is similar to that made by Tager-Flusberg (2001) between a 'social perceptive' and a 'social cognitive' component of theory of mind. The claim would be that in Asperger syndrome only the social perceptive component is impaired, but in autism both would be impaired. This hypothesis is in need of testing. Perhaps it could explain why in Asperger syndrome the social communication failure is more evident in real life than in the laboratory. In real life a high premium may be set on the perceptive component because usually cues are ambiguous and responses are required to be fast; in the laboratory, the social cognitive component can come into its own if there is enough time to apply analytic reasoning.

A theory proposed by Klin and colleagues (Klin, Jones, Schultz, & Volkmar, 2003) explains the discrepancy between real-world impairment and laboratory success as a result of the high complexity of the social world that is never retained in laboratory tests. Thus Klin and colleagues suggest that the salience of stimuli is fundamentally different in autism, and this is revealed in abnormal responses to social stimuli, for instance deviant patterns of eye gaze and inability to recognise faces. This would apply to Asperger syndrome as much as to other forms of autistic disorder.

Intuitive mentalising tasks have also been used in the laboratory, and on these seemingly simpler tasks individuals with Asperger syndrome seem to be if anything more impaired than on some of the complex tasks mentioned earlier. One example is the automatic comprehension of animated shapes as interacting agents. Heider and Simmel (1944) showed that observers feel compelled to attribute mental states to animated shapes in line with their movement patterns. Klin (2000) showed such geometric shapes to individuals with high-functioning autism and Asperger syndrome. In Klin's study, the participants with autistic disorder identified about a

quarter of the social elements in the story, while a third of their attributions were irrelevant. They used pertinent mental state terms very infrequently, and were also unable to derive psychologically based personality features from the shapes' movements. Individuals with Asperger syndrome improved their performance slightly compared to those with autism when provided with more explicit verbal information, but not significantly so. Another study with animations, conducted with different materials and different scoring criteria by Abell, Happé, and Frith (2000) also showed that children with autism were significantly less able to attribute appropriate mental states than control participants. Using the same stimuli, Castelli, Frith, Happé, and Frith (2002) found that this was true even for very high-functioning adults with autism and Asperger syndrome.

Brain imaging studies of mentalising in autism

There is an increasing number of brain imaging studies in which normal volunteers have been asked to perform simple tasks that require them to take into account other people's mental states (for a recent review see Frith & Frith, 2003). In each of these studies the critical tasks are compared with tasks that are similar in every respect except that they do not require mentalising. The results to date are remarkably consistent. Regardless of presentation and response mode, similar regions of the brain are active when volunteers make inferences about the mental states of protagonists, as opposed to physical or behavioural states. These regions are strongly interconnected, and it is likely that they constitute part of the 'social brain' that includes frontal (medial prefrontal; orbitofrontal) and temporal (superior temporal sulcus; inferior basal temporal cortex; temporal poles; amygdala) regions, all highly interconnected (Brothers, 1997).

A small handful of studies have been carried out with high-functioning individuals with autism and those with Asperger syndrome (Happé et al., 1996; Baron-Cohen, Ring et al., 1999; Castelli et al., 2002; Nieminen-von Wendt et al., 2003). The control groups were normally intelligent adults of the same age and educational level. In all these studies, regardless of the task used (visual and auditory stories, photographs of eyes, geometric animations), able individuals with autism or Asperger syndrome showed reduced brain activation in components of the mentalising system. Figure 1 shows the results from the study by Castelli et al. (2002) using geometric animations. Here 10 high-functioning individuals took part, who were diagnosed according to DSM-IV as having either autism or Asperger syndrome. These two subgroups did not differ on current social behaviour or on experimental and psychometric tests. They were compared with 10 normal volunteers matched for age and intelligence.

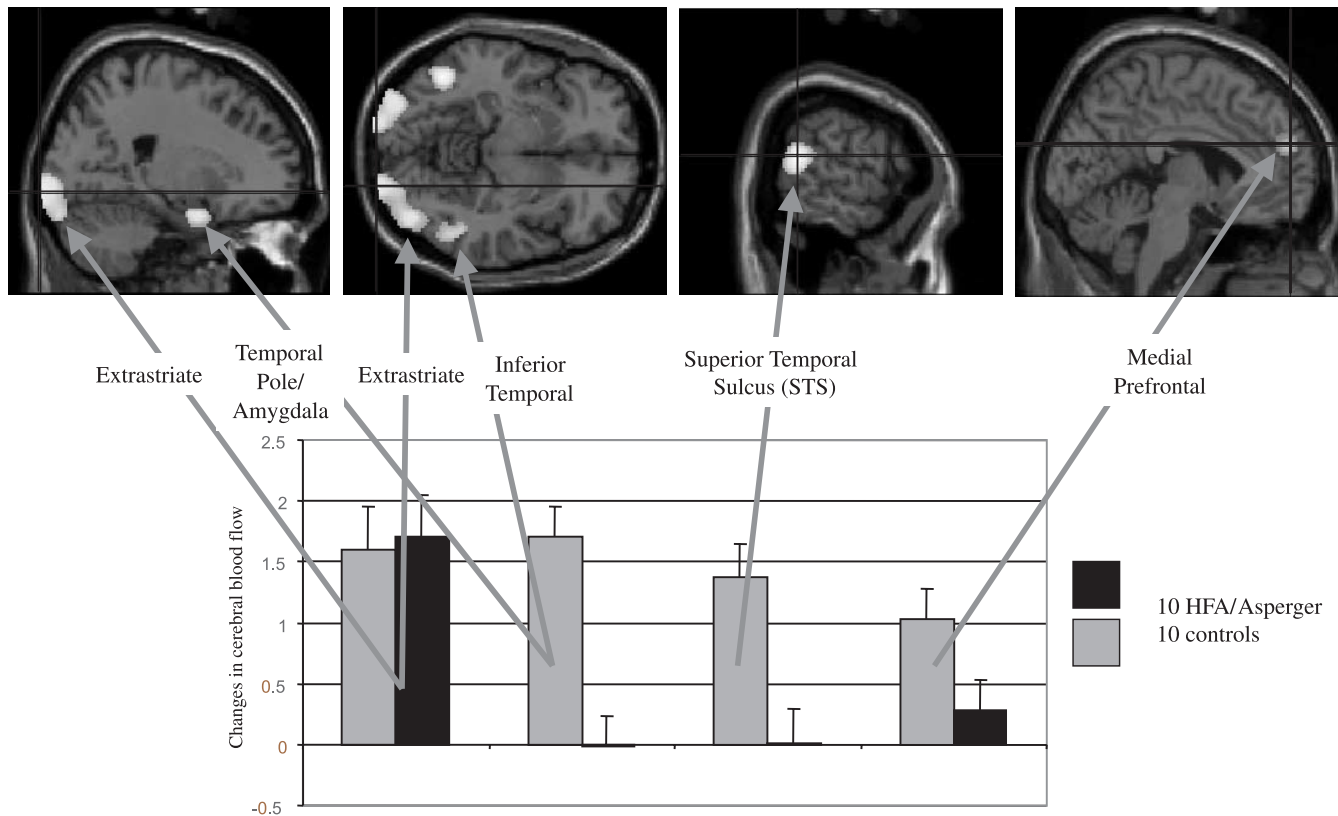


Figure 1 Reduced activation in the mentalising system in Asperger syndrome when watching scripted vs. random animations (based on data from Castelli et al., 2002)

As Figure 1 shows, the three main regions of the mentalising system showed significantly less activation in the autism/Asperger syndrome group.

Castelli et al. (2002) suggested that weak connectivity between the components could be a possible reason for the reduced activation. This was confirmed by a connectivity analysis of the regions associated with differences in blood flow. There was a significantly weaker relationship between activations in occipital extrastriate regions and the fronto-temporal regions of the mentalising system in the autism/Asperger group than in the normal group. The extrastriate regions of the brain are concerned with visual-spatial processing. As illustrated in Figure 1, they showed equally enhanced activity in both groups, while watching the movement of shapes that evoked mental state attribution (relative to random movements). However, this appropriately enhanced visual spatial processing was not accompanied by enhanced processing in more anterior regions of the system. Why not? One reason could be lack of top-down modulation. Normally, top-down signals select the important, unexpected or salient aspects of the information and thus information overload is avoided. We can sometimes experience this top-down effect when we suddenly understand the meaning of a complex stimulus, which a moment before seemed uninterpretable. In the case of the animations, we understand the meaning of the complex movements as particular social interactions. It could be this as-

pect of perceptual processing that is not functioning well in people with Asperger syndrome.

This suggestion opens up a link to another problem, at the cognitive level, which is frequently proposed as characteristic of autistic disorders: a difficulty in high-level executive functions, involved in forward planning and flexibility. These can also be described as the effects of a lack of top-down control.

Cognitive strengths and weaknesses in non-social domains

In contrast with the social difficulties, it is notable that people with Asperger syndrome often have extremely good understanding of the non-social world (e.g., Baron-Cohen, Wheelwright et al., 1999). Reitzel and Szatmari's (2003) review of studies that compare Asperger syndrome with that of other forms of autistic disorder concludes that an unevenness of the cognitive profile is highly typical of all forms of autism. It has long been known that the Wechsler IQ scales show similar peaks and troughs in very able individuals with autistic disorder as in those who are much less able, and this has been confirmed in recent studies (Goldstein, Beers, Siegel, & Minshew, 2001).

How is the unevenness to be explained? One possibility is that the cognitive style of weak central coherence facilitates painstaking analysis of per-

ceptual and verbal detail and thus can lead to peak performance (Happé, 1999). Weak central coherence appears to apply to Asperger syndrome just as much as to other forms of autism if it is seen as intimately linked to the pursuit of narrow interests. Another possibility is that as a result of lacking top-down control, bottom-up processes are unusually strong and incoming information is processed to a high degree (Frith, 2003). Recent empirical studies in this area have provided evidence for enhanced perception in autism in auditory and visual modalities, at no cost to the global level of information processing (Plaisted, Saksida, Alcantarà, & Weisblatt, 2003; Mottron, Burack, Iarocci, Belleville, & Enns, 2003).

Baron-Cohen explains the cognitive strengths and weaknesses by proposing the concept of 'systemising', which refers to the tendency to amass facts about the physical world in a systematic fashion. He considers this the other side of poor 'empathising', which clearly applies only to the social but not to the physical world (Baron-Cohen, 2002). In this sense, Baron-Cohen is attempting to offer a unified account of both social and non-social features of autistic disorder.

Some cognitive weaknesses in Asperger syndrome, in both social and non-social domains, can be explained by a specific neuropsychological abnormality, namely executive function deficits. Such impairments have been explored widely in lower-functioning children with autism (see chapters in Russell, 1997), but they are also present in higher-functioning children (Turner, 1999). However, there has been much less research with adults with Asperger syndrome. Observations of perseveration in movements, in choice of topic, in both work and leisure activities, suggest that the top-down control of action is problematic. Top-down control, as used here, is the opposite of stimulus-driven behaviour. It allows modulation of attention and action in the service of long-term aims and avoidance of distraction by accidentally present attention-grabbing stimuli. How this cognitive process relates to top-down processing in neurophysiological terms has still to be explored. The presence of top-down control in normal individuals may explain why certain stimuli are salient in certain contexts, and why responses can be very different even if the stimulus remains the same. Thus, the same stimulus has different meaning according to expectation and context. If in the case of autistic disorder this control were weak, then one would expect that the same stimulus would always be salient and have the same meaning, regardless of context.

It is doubtful whether impairments in executive functions will be found to be milder in Asperger syndrome than in autism. Likewise, it remains to be seen whether the strengths due to attention to detail, as postulated by weak central coherence theory, will be less extreme. Asperger individuals are often

known for their meticulous work whether it is in crafts, art or science and their ability to identify hitherto overlooked details. Likewise, anecdotes suggest that difficulties tend to arise from the fact that people with Asperger syndrome see situations in fixed and absolute terms, rather than relative to context. Furthermore, they often need someone to remind them when they should do something that they intended to do, and to tell them what the appropriate action should be in slightly unexpected situations. They appear to find it hard to override routine responses. They are well known for being rigid and tend to take any change of plan or routine very badly. They crave sameness just like individuals with other forms of autism, and they thrive if there is clear structure in the environment and they need external prompts to carry out infrequent and unusual tasks.

Only a small amount of evidence from brain imaging is available so far and it supports the notion of frontal dysfunction in Asperger syndrome. A structural study of the brain of Asperger syndrome individuals (McAlonan et al., 2002) found abnormalities in fronto-striatal pathways. The authors propose that these may result in defective sensorimotor gating, and consequently characteristic difficulties inhibiting repetitive thoughts, speech and actions. Murphy and colleagues (2002) found a correlation between abnormalities in the neuronal integrity of the prefrontal lobe and obsessive behaviour.

Much more work is needed to clarify the neurophysiological and cognitive basis of the problems and interests in Asperger syndrome in the non-social domain. It is as yet unknown how these features relate to the social features, and whether all cases have all of the features, or only some. It is possible that some people will be characterised by mild social problems but severe executive problems, and other people with severe social problems but mild executive problems. Does weak central coherence characterise only a subgroup? And is enhanced perceptual processing an advantage in acquiring certain academic and artistic skills? Future research in this area could have considerable diagnostic and therapeutic implications.

Imagination and originality

Hans Asperger alluded in several of his cases to their high originality of thought but also imagination. For instance, Ernst K. was reported to tell fantastic stories. An important sign of what Asperger termed 'autistic intelligence' was the ability to coin apt and original verbal expressions. This is different from savant skills, whose association with autism is well documented, while their association with Asperger syndrome is as yet unknown.

Anecdotally, in children and adults with Asperger syndrome, there is much evidence for the ability to role-play and to create fictitious worlds in words and

pictures (Tantam, 1991). Biographical accounts sometimes tell of extensive and wide-ranging imaginary activities. The artist Gilles Trehin provides one stunning example of creative imagery (see Figure 2). Gilles has created an imaginary city, Urville, over many years since his childhood, with its own history and population, and his own inventive names of streets and buildings (see <http://urville.com/index.html>). On his website Gilles explains how the idea of the city grew in his mind, stimulated by a visit to New York as a child, and that he first started to construct a city with Lego bricks. Having been frustrated by the impossibility to build a whole city in his bedroom in this way, he was delighted by the realisation (at the age of 12 or 13) that he could imagine the city in his mind and draw parts of it on paper. Gilles was diagnosed as autistic in early childhood, but as an adult he perfectly fits the clinical picture of Asperger syndrome.

In most cases of autism, pretence and imaginative play have been described as absent or delayed, and, if present, they are often described as restricted, stereotyped and repetitive. This assumption may not hold in the case of Asperger syndrome. Indeed, children with Asperger syndrome were found to be more able to produce creative narratives than children with autism (Craig & Baron-Cohen, 2000).

However, even in the outstanding case of Gilles it is not clear to what extent his imaginative activities are separable from obsessive interests. Some of the highly specialised interests found in people with autistic disorder focus on imaginary people, while others focus on concrete objects, and yet others on more abstract concepts, such as calendar dates.

Listening to people with Asperger syndrome

Many individuals with Asperger syndrome are prolific writers. One need only look at the catalogues of Jessica Kingsley, a publisher who specialises in this literature. These writings certainly give important insights but they do not necessarily add up to a consistent picture. Many forcefully point out that they are not patients, do not feel affected and are not suffering from any disorder, but instead they have different personalities, different needs and different views from those they like to call 'neurotypicals'. Researchers and clinicians can agree with this to some extent. However, they may point out that a peculiar lack of insight and an egocentric viewpoint are typical of the syndrome, throwing doubt on at least some of the self-assessments of needs and expectations. One problem with the autobiograph-



Figure 2 Place de l'Île de Beauté, Urville, Drawing by Gilles Trehin

ical literature is that the authenticity of the diagnosis is not guaranteed. As mentioned earlier, there are concerns about an overextension of the diagnosis, so that disorders, yet to be identified, might currently be classified as Asperger syndrome.

Individuals with Asperger syndrome give ample proof that they have the intellectual resources to make sense of the physical world through often excellent perception and prodigious memory. This strength in general information processing capacity is likely to act as a means for learning about the social world as well. These individuals can find a different developmental pathway. The autobiographies of individuals with Asperger syndrome indicate a high degree of retrospective self-analysis that came with adulthood. This can be seen, for instance, in Gunilla Gerland's autobiography (1997) and in Clare Sainsbury's collection of over twenty individuals' reminiscences of their school years (2000). These works suggest that self-knowledge and sharing of knowledge with others was poor in childhood.

From the autobiographical writings there appears to be at least a subgroup of Asperger syndrome individuals who develop self-awareness to a heightened degree. They talk about themselves and their experiences, but do not seem to have the same curiosity to know what others think or feel. Some tend to be suspicious of others, even paranoid, and this could be because they continue to find it difficult to read other people's minds (Blackshaw, Kinderman, Hare, & Hatton, 2001). Suspicion is likely to flourish when you do not automatically take into account that other people have different points of view from your own. If you are continually surprised that other people think differently from you, and if it is hard for you to realise that other people may have interests that are incompatible with yours, then you may well feel that they are hostile to you.

The ability to reflect on one's own emotions and to share emotions with others by talking about them is a natural consequence of mentalising. If intuitive mentalising were impaired it would be expected that the awareness of complex emotions is also impaired. Of course, the inability to identify emotions and to talk about them may have many different causes – as, for example, in depression. The term 'alexithymia', meaning 'no words for feelings', has been coined to describe this symptom, which has been investigated in a range of psychiatric patients. Alexithymia can be measured by self-report using standardised questionnaires (e.g., the Toronto Alexithymia Scale; Bagby, Parker, & Taylor, 1994). The following examples may indicate how difficulty identifying and communicating feelings is addressed: 'when I am upset, I don't know if I am sad, frightened or angry'; 'I do not find examination of my feelings useful in solving personal problems'; 'I think it is strange that others analyse their emotions so often.'

In a recent study, 27 individuals with autism spectrum disorders were assessed, of whom 19 had a diagnosis of Asperger syndrome and 8 a diagnosis of high-functioning autism. They obtained significantly elevated scores on this questionnaire, when compared to IQ- and age-matched controls and also when compared to their own relatives (Hill, Berthoz, & Frith, *in press*). They reported greater difficulty in identifying and describing their own feelings, and less interest in the psychological motives behind actions. The ability to reflect on these difficulties shows that a certain amount of insight is present, and the problems reported are consistent with the autobiographical accounts. These treat in detail physical sensations and sensory experiences, but rarely complex emotions and attitudes. Half of the Asperger syndrome group obtained such extreme scores on the Toronto questionnaire that they would have been classified as severely impaired.

In this study the participants were also given the Beck Depression Inventory (Beck, Steer, & Garbin, 1988) in which individuals are asked to indicate by means of specific statements how they have felt over the past week. The participants with Asperger syndrome showed significantly increased scores for depression and about 20% scored so highly that they would have been categorised as clinically depressed. Depression and chronic anxiety in Asperger syndrome are common psychiatric features (Ghaziuddin, Ghaziuddin, & Greden, 2002; Green, Gilchrist, Burton, & Cox, 2000). It is possible to explain these features as secondary reactions to the burden caused by the core problems of autism. For instance, if intuitive mentalising failure makes it hard to predict and interpret other people's behaviour, then the social world cannot be the source of pleasure that it can be for ordinary people. Especially if there is a desire to be part of the social world, as frequently expressed by people with Asperger syndrome, unexplained failure of social interactions may well create or intensify feelings of depression. The inability to cope with the non-routine aspects of everyday life due to executive function problems may also contribute to high anxiety and depression.

Looking towards the future

Asperger syndrome is a category that is desperately needed and hugely important in clinical practice, even if the boundaries are fluid at present. Indeed the need is so great that it is possible that the diagnosis of Asperger syndrome is at present overextended. Perhaps one reason for its current popularity and, potentially, overextension is that social impairment is very common, but current categories for diagnosis are extremely few. Thus Asperger syndrome may act like a magnet for as yet unidentified developmental disorders that affect social interaction and everyday adaptation.

It may well be that eventually subgroups of what is now called Asperger syndrome will be identified, each characterised by specific core problems. In line with Hans Asperger's belief that these individuals have much to offer society, the importance of finding appropriate niches and work support must be recognised. It is ironical that the cognitive strengths can obscure the social difficulties, and that this may lead to less willingness to acknowledge the need for guidance and support.

What can we conclude so far about the needs of people with Asperger syndrome? They carry the burden of a neuro-genetic disorder, however mild it may be in comparison to other forms of autism, and they are likely to need a measure of support throughout their lives. Raising awareness of the condition remains a crucial task. Unfortunately, individuals with Asperger syndrome can be denied recognition and help because they are intellectually bright and may be able to give the impression of a near normal competence in routine interactions. The appearance of normality is deceptive, however, and breaks down when novel or stressful situations arise. Raising awareness is important also in school children, and might well alleviate the distressingly common problem of the bullying of children who stand out as socially odd (Little, 2001).

From the behavioural and neurophysiological studies to date we may assume that the mind and the brain of the person with Asperger syndrome is different from the mind/brain of the ordinary person and not so different from the person with autism. Some potentially handicapping secondary problems in adults with Asperger syndrome can be traced to reduced awareness of their own feelings and those of others. The inability to talk about feelings appears to be associated with clinically significant depression and anxiety. Treatment of these symptoms is a matter of priority.

An astounding fact about Asperger syndrome is that a proportion of individuals can achieve high academic qualifications, and a few, high scientific distinctions (Baron-Cohen, Wheelwright et al., 1999; Fitzgerald, 2002). However, many individuals with Asperger syndrome do not find a vocational niche in adult life. It is generally agreed that highly able children with autistic disorder benefit from specialist education just as much as less able children, and similar teaching approaches may be suitable for both (see chapters in Prior, 2003). Given the great heterogeneity of autistic disorder, individual programmes with one-to-one instruction seem to be an obvious choice. The extraordinary achievements of people with Asperger syndrome demonstrate that compensatory learning can partially overcome neurophysiological deficiency. Compensation is not something that works all by itself. Teaching, and in particular explicit teaching of otherwise implicit rules, is necessary. Despite the bleak message that neurophysiological abnormalities that exist from

birth or before cannot be put right with present means, remarkable improvements in adaptation can occur over the lifespan.

Everyone will agree that much work needs to be done in the fine-grained description of behaviour and in the long-term follow-up of individual cases before we know what Asperger syndrome really is. Meanwhile, there are things to be done to help better recognition and awareness of the disorder. Because Asperger syndrome is often recognised when the child is at school, there is a practical need for assessment instruments that can be used by educational psychologists as well as clinicians. Such instruments could then act as a basis for charting the developmental course of the disorder, which remains one of the most urgent tasks to be done. To be useful in this way the assessment instrument needs to include psychometric tests to estimate general intellectual ability; neuropsychological tests to identify cognitive strengths and weaknesses; and experimental tests of social functioning, including person perception, emotion perception and subtle tests of intuitive mentalising. This is a difficult task, but such an assessment instrument would make a start in documenting the nature and development of some of the features that are typical of Asperger syndrome.

Acknowledgement

This work has been supported by an MRC programme grant. I am grateful to Francesca Happé, Sarah-Jayne Blakemore and Elisabeth Hill for their constructive comments on this paper.

Correspondence to

Uta Frith, UCL Institute of Cognitive Neuroscience, 17 Queen Square, London WC1N 3AR, UK; Email: u.frith@ucl.ac.uk

References

- Abell, F., Happé, F., & Frith, U. (2000). Do triangles play tricks? Attribution of mental states to animated shapes in normal and abnormal development. *Journal of Cognitive Development*, 15, 1–20.
- Adolphs, R., Sears, L., & Piven, J. (2001). Abnormal processing of social information from faces in autism. *Journal of Cognitive Neuroscience*, 13, 232–240.
- Asperger, H. (1944). Die 'autistischen Psychopathen' im Kindesalter. *Archiv für Psychiatrie und Nervenkrankheiten*, 117, 76–136. [Translated by U. Frith in U. Frith (Ed.). (1991). *Autism and Asperger syndrome* (pp. 36–92). Cambridge: Cambridge University Press].
- Bagby, R.M., Parker, J.D., & Taylor, G.J. (1994). The twenty-item Toronto Alexithymia Scale – I. Item selection and cross-validation of the factor structure. *Journal of Psychosomatic Research*, 38, 23–32.

- Baird, G., Charman, T., Baron-Cohen, S., Cox, A., Swettenham, J., Wheelwright, S., & Drew, A. (2000). A screening instrument for autism at 18 months of age: A 6-year follow-up study. *Journal of the American Academy of Child and Adolescent Psychiatry*, 39, 694–702.
- Baron-Cohen, S. (1992). Out of sight or out of mind? Another look at deception in autism. *Journal of Child Psychology and Psychiatry*, 33, 1141–1155.
- Baron-Cohen, S. (2002). The extreme male brain theory of autism. *Trends in Cognitive Sciences*, 6, 248–254.
- Baron-Cohen, S., Cox, A., Baird, G., Swettenham, J., Nightingale, N., Morgan, K., Drew, A., & Charman, T. (1996). Psychological markers in the detection of autism in infancy in a large population. *British Journal of Psychiatry*, 168, 158–163.
- Baron-Cohen, S., Jolliffe, T., Mortimore, C., & Robertson, M. (1997). Another advanced test of theory of mind: Evidence from very high functioning adults with autism or Asperger syndrome. *Journal of Child Psychology and Psychiatry*, 38, 813–822.
- Baron-Cohen, S., O'Riordan, M., Stone, V., Jones, R., & Plaisted, K. (1999). Recognition of faux pas by normally developing children and children with Asperger syndrome or high-functioning autism. *Journal of Autism and Developmental Disorders*, 29, 407–418.
- Baron-Cohen, S., Richler, J., Bisarya, D., Gurunathan, N., & Wheelwright, S. (2003). The systemizing quotient: An investigation of adults with Asperger syndrome or high-functioning autism, and normal sex differences. *Philosophical Transactions of the Royal Society London, B*, 358, 361–374.
- Baron-Cohen, S., Ring, H.A., Wheelwright, S., Bullmore, E.T., Brammer, M.J., Simmons, A., & Williams, S.C. (1999). Social intelligence in the normal and autistic brain: An fMRI study. *European Journal of Neuroscience*, 11, 1891–1898.
- Baron-Cohen, S., Tager-Flusberg, H., & Cohen, D. (1993). *Understanding other minds: Perspectives from autism*. Oxford: Oxford University Press.
- Baron-Cohen, S., Tager-Flusberg, H., & Cohen, D. (2000). *Understanding other minds: Perspectives from developmental neuroscience* (2nd edn). Oxford: Oxford University Press.
- Baron-Cohen, S., Wheelwright, S., Stone, V., & Rutherford, M. (1999). A mathematician, a physicist and a computer scientist with Asperger syndrome: Performance on folk psychology and folk physics tests. *Neurocase*, 5, 475–483.
- Bauman, M.L., & Kemper, T.L. (2003). The neuropathology of the autism spectrum disorders: What have we learned? *Novartis Foundation Symposium* 251, 112–122.
- Beck, A.T., Steer, R., & Garbin, M. (1988). Psychometric properties of the Beck Depression Inventory: 25 years of research. *Clinical Psychology Review*, 8, 77–100.
- Blackshaw, A.J., Kinderman, P., Hare, D.J., & Hatton, C. (2001). Theory of mind, causal attribution and paranoia in Asperger syndrome. *Autism*, 5, 147–163.
- Blair, R.J.R. (2003). Facial expressions, their communicatory functions and neuro-cognitive substrates. *Philosophical Transactions of the Royal Society London, B*, 358, 561–572.
- Bowler, D. (1992). 'Theory of Mind' in Asperger syndrome. *Journal of Child Psychology and Psychiatry*, 33, 877–893.
- Brothers, L. (1997). *Friday's footprint: How society shapes the human mind*. Oxford: Oxford University Press.
- Burgoine, E., & Wing, L. (1983). Identical triplets with Asperger's syndrome. *British Journal of Psychiatry*, 143, 261–265.
- Casanova, M.F., Buxhoeveden, D.P., Switala, A.E., & Roy, E. (2002). Asperger's syndrome and cortical neuropathology. *Journal of Child Neurology*, 17, 142–145.
- Castelli, F., Frith, C.D., Happé, F., & Frith, U. (2002). Autism, Asperger syndrome and brain mechanisms for the attribution of mental states to animated shapes. *Brain*, 125, 1839–1849.
- Channon, S., Charman, T., Heap, J., Crawford, S., & Rios, P. (2001). Real-life-type problem-solving in Asperger's syndrome. *Journal of Autism and Developmental Disorders*, 31, 461–469.
- Charman, T., Swettenham, J., Baron-Cohen, S., Cox, A., Baird, G., & Drew, A. (1997). Infants with autism: An investigation of empathy, pretend play, joint attention, and imitation. *Developmental Psychology*, 33, 781–789.
- Cox, A., Klein, K., Charman, T., Baird, G., Baron-Cohen, S., Swettenham, J., Drew, A., & Wheelwright, S. (1999). Autism spectrum disorders at 20 and 42 months of age: Stability of clinical and ADI-R diagnosis. *Journal of Child Psychology and Psychiatry*, 40, 719–732.
- Craig, J., & Baron-Cohen, S. (2000). Story-telling ability in children with autism or Asperger syndrome: A window into the imagination. *Israeli Journal of Psychiatry and Related Sciences*, 37, 64–70.
- Critchley, H.D., Daly, E.M., Bullmore, E.T., Williams, S.C.R., Van Amelsvoort, T., Robertson, D.M., Rowe, A., Phillips, M., McAlonan, G., Howlin, P., & Murphy, D.G.M. (2000). The functional neuroanatomy of social behaviour – changes in cerebral blood flow when people with autistic disorder process facial expressions. *Brain*, 123, 2203–2212.
- Fitzgerald, M. (2002). Asperger's disorder and mathematicians of genius. *Journal of Autism and Developmental Disorders*, 32, 59–60.
- Frith, U. (1991). Asperger and his syndrome. In U. Frith (Ed.), *Autism and Asperger syndrome* (pp. 1–36). Cambridge: Cambridge University Press.
- Frith, U. (2003). *Autism: Explaining the enigma* (2nd edn). Oxford: Blackwell.
- Frith, U., & Frith, C.D. (2003). Development and neurophysiology of mentalizing. *Philosophical Transactions of the Royal Society London, B*, 358, 459–473.
- Gerland, G. (1997). *A real person. Life from the outside*. London: Souvenir Press.
- Ghaziuddin, M., Ghaziuddin, N., & Greden, J. (2002). Depression in persons with autism: Implications for research and clinical care. *Journal of Autism and Developmental Disorders*, 32, 299–306.
- Gillberg, C., & Billstedt, E. (2000). Autism and Asperger syndrome: Coexistence with other clinical disorders. *Acta Psychiatrica Scandinavica*, 102, 321–330.
- Gilchrist, A., Green, J., Cox, A., Burton, D., Rutter, M., & Le Couteur, A. (2001). Development and current

- functioning in adolescents with Asperger syndrome: A comparative study. *Journal of Child Psychology and Psychiatry*, 42, 227–240.
- Goldstein, G., Beers, S.R., Siegel, D.J., & Minshew, N.J. (2001). A comparison of WAIS-R profiles in adults with high-functioning autism or differing subtypes of learning disability. *Applied Neuropsychology*, 8, 148–154.
- Green, D., Baird, G., Barnett, A.L., Henderson, L., Huber, J., & Henderson, S.E. (2002). The severity and nature of motor impairment in Asperger's syndrome: A comparison with specific developmental disorder of motor function. *Journal of Child Psychology and Psychiatry*, 43, 655–668.
- Green, J., Gilchrist, A., Burton, D., & Cox, A. (2000). Social and psychiatric functioning in adolescents with Asperger syndrome compared with conduct disorder. *Journal of Autism and Developmental Disorders*, 30, 279–93.
- Happé, F. (1999). Autism: Cognitive deficit or cognitive style? *Trends in Cognitive Sciences*, 3, 216–222.
- Happé, F., Ehlers, S., Fletcher, P., Frith, U., Johansson, M., Gillberg, C., Dolan, R., Frackowiak, R., & Frith, C. (1996). 'Theory of mind' in the brain. Evidence from a PET scan study of Asperger syndrome. *NeuroReport*, 8, 197–201.
- Happé, F.G.E. (1994). An advanced test of theory of mind: Understanding of story characters' thoughts and feelings by able autistic, mentally handicapped, and normal children and adults. *Journal of Autism and Developmental Disorders*, 24, 129–154.
- Heider, F. & Simmel, M. (1944). An experimental study of apparent behavior. *American Journal of Psychology*, 57, 243–259.
- Hill, E., Berthoz, S., & Frith, U. (in press). Brief report: Cognitive processing of own emotions in individuals with autistic spectrum disorder and their relatives. *Journal of Autism and Developmental Disorders*.
- Hippler, K., & Klicpera, C. (2003). A retrospective analysis of the clinical case records of 'autistic psychopaths' diagnosed by Hans Asperger and his team at the University Children's Hospital Vienna. *Philosophical Transactions of the Royal Society Series B*, 358, 291–301.
- Howard, M.A., Cowell, P.E., Boucher, J., Broks, P., Mayes, A., Farrant, A., & Roberts, N. (2000). Convergent neuroanatomical and behavioural evidence of an amygdala hypothesis of autism. *Neuroreport*, 11, 2931–2935.
- 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, 3–13.
- Howlin, P., & Asgharian, A. (1999). The diagnosis of autism and Asperger syndrome: Findings from a survey of 770 families. *Developmental Medicine and Child Neurology*, 41, 834–839.
- Jolliffe, T., & Baron-Cohen, S. (1999). The Strange Stories Test: A replication with high-functioning adults with autism or Asperger syndrome. *Journal of Autism and Developmental Disorders*, 29, 395–406.
- Kaland, N., Moller-Nielsen, A., Callesen, K., Mortensen, E.L., Gottlieb, D., & Smith, L. (2002). A new 'advanced' test of theory of mind: Evidence from children and adolescents with Asperger syndrome. *Journal of Child Psychology and Psychiatry*, 43, 517–528.
- Kanner, L. (1943). Autistic disturbances of affective contact. *Nervous Child*, 2, 217–250.
- Kanner, L. (1971). Follow-up study of eleven autistic children originally reported in 1943. *Journal of Autism and Childhood Schizophrenia*, 1, 119–145.
- Klin, A. (2000). Attributing social meaning to ambiguous visual stimuli in higher-functioning autism and Asperger syndrome: The Social Attribution Task. *Journal of Child Psychology and Psychiatry*, 41, 831–46.
- Klin, A., Jones, W., Schultz, R., & Volkmar, F. (2003). The enactive mind, or from actions to cognition: Lessons from autism. *Philosophical Transactions of the Royal Society Series B*, 358, 345–360.
- Klin, A., Jones, W., Schultz, R., Volkmar, F., & Cohen, D. (2002). Defining and quantifying the social phenotype in autism. *American Journal of Psychiatry*, 159, 895–908.
- Klin, A., Volkmar, F., & Sparrow, S. (Eds). (2000). *Asperger syndrome*. New York: Guilford Press.
- Klin, A., Volkmar, F.R., Sparrow, S.S., Cicchetti, D.V., & Rourke, B.P. (1995). Validity and neuropsychological characterization of Asperger syndrome: Convergence with nonverbal learning disabilities syndrome. *Journal of Child Psychology and Psychiatry*, 30, 1127–1140.
- Le Couteur, A., Bailey, A., Goode, S., Pickles, A., Robertson, S., Gottesman, I., & Rutter, M. (1996). A broader phenotype of autism: The clinical spectrum in twins. *Journal of Child Psychology and Psychiatry*, 37, 785–801.
- Little, L. (2001). Peer victimization of children with Asperger spectrum disorders. *Journal of the American Academy of Child and Adolescent Psychiatry*, 40, 995–996.
- Mayes, S.D., & Calhoun, S.L. (2001). Non-significance of early speech delay in children with autism and normal intelligence and implications for DSM-IV Asperger's disorder. *Autism*, 5, 81–94.
- Mayes, S.D., Calhoun, S.L., & Crites, D.L. (2001). Does DSM-IV Asperger's disorder exist? *Journal of Abnormal Child Psychology*, 29, 263–271.
- McAlonan, G.M., Daly, E., Kumari, V., Critchley, H.D., van Amelsvoort, T., Suckling, J., Simmons, A., Sigmondsson, T., Greenwood, K., Russell, A., Schmitz, N., Happé, F., Howlin, P., & Murphy, D.G. (2002). *Brain anatomy and sensorimotor gating in Asperger's syndrome*. *Brain* 125, 1594–1606.
- Miller, J.N., & Ozonoff, S. (1997). Did Asperger's cases have Asperger disorder? A research note. *Journal of Child Psychology and Psychiatry*, 38, 247–251.
- Mottron, L., Burack, J.A., Iarocci, G., Belleville, S., & Enns, J.T. (2003). Locally oriented perception with intact global processing among adolescents with high-functioning autism: Evidence from multiple paradigms. *Journal of Child Psychology and Psychiatry*, 44, 904–913.
- Murphy, D.G., Critchley, H.D., Schmitz, N., McAlonan, G., Van Amelsvoort, T., Robertson, D., Daly, E., Rowe, A., Russell, A., Simmons, A., Murphy, K.C., & Howlin, P. (2002). Asperger syndrome: A proton magnetic

- resonance spectroscopy study of brain. *Archives of General Psychiatry*, 59, 885–891.
- Nieminen-von Wendt, T., Metsahonkala, L., Kulomaki, T., Alto, S., Autti, T., Vanhala, R., & von Wendt, L. (2003). Changes in cerebral blood flow in Asperger syndrome during theory of mind tasks presented by the auditory route. *European Journal of Child and Adolescent Psychiatry*, 12, 178–189.
- Plaisted, K., Saksida, L., Alcantarà, J., & Weisblatt, E. (2003). Towards an understanding of the mechanisms of weak central coherence effects: Experiments in visual configural learning and auditory perception. *Philosophical Transactions of the Royal Society B*, 358, 375–386.
- Prior, M.(Ed.). (2003). *Asperger syndrome, behavioural and educational aspects*. New York: Guilford Publications.
- Reitzel, J., & Szatmari, P. (2003). Learning difficulties in Asperger syndrome. In M. Prior (Ed.), *Asperger syndrome, behavioural and educational aspects* (pp. 35–54). New York: Guilford Publications.
- Rinehart, N.J., Bradshaw, J.L., Brereton, A.V., & Tonge, B.J. (2002). A clinical and neurobehavioural review of high-functioning autism and Asperger's disorder. *Australian and New Zealand Journal of Psychiatry*, 36, 762–770.
- Roeyers, H., Buyse, A., Ponnet, K., & Pichal, B. (2001). Advancing advanced mind-reading tests: Empathic accuracy in adults with a pervasive developmental disorder. *Journal of Child Psychology and Psychiatry*, 42, 271–278.
- Russell, J.(Ed.). (1997). *Autism as an executive disorder*. Oxford: Oxford University Press.
- Rutherford, M.D., Baron-Cohen, S., & Wheelwright, S. (2002). Reading the mind in the voice: a study with normal adults and adults with Asperger syndrome and high functioning autism. *Journal of Autism and Developmental Disorders*, 32, 189–194.
- Sainsbury, C. (2000). *Martian in the playground. Understanding the schoolchild with Asperger syndrome*. Bristol: Lucky Duck Publishers.
- Schultz, R.T., Grelotti, D.J., Klin, A., Kleinman, J., Van der Gaag, C., Marois, R., & Skudlarski, P. (2003). The role of the fusiform face area in social cognition: Implications for the pathobiology of autism. *Philosophical Transactions of the Royal Society B*, 358, 415–427.
- Shamay-Tsoory, S.G., Tomer, R., Yaniv, S., & Aharon-Peretz, J. (2002). Empathy deficits in Asperger syndrome: A cognitive profile. *Neurocase*, 8, 245–252.
- Shriberg, L.D., Paul, R., McSweeney, J.L., Klin, A., & Cohen, D.J. (2001). Speech and prosody characteristics of adolescents and adults with high-functioning autism and Asperger syndrome. *Journal of Speech Language and Hearing Research*, 44, 1097–1115.
- Slater-Walker, G., & Slater-Walker, C. (2002). *An Asperger marriage*. London: Jessica Kingsley Publishers.
- 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, 1980–1987.
- Tager-Flusberg, H. (2001). A reexamination of the theory of mind hypothesis of autism. In J. Burack, T. Charman, et al. (Eds.), *The development of autism: Perspectives from theory and research* (pp. 173–193). New Jersey: Lawrence Erlbaum Associates.
- Tantam, D. (1991). Asperger syndrome in adulthood. In U. Frith (Ed.), *Autism and Asperger syndrome* (pp. 147–183). Cambridge: Cambridge University Press.
- Turner, M. (1999). Generating novel ideas: Fluency performance in high-functioning and learning disabled individuals with autism. *Journal of Child Psychology and Psychiatry*, 40, 189–201.
- Wing, L. (1981). Asperger's syndrome; a clinical account. *Psychological Medicine*, 11, 115–130.
- Wing, L. (1991). The relationship between Asperger's syndrome and Kanner's autism. In U. Frith (Ed.), *Autism and Asperger syndrome* (pp. 93–121). Cambridge: Cambridge University Press.
- Wing, L. (2000). Past and future research on Asperger syndrome. In A. Klin, F. Volkmar, & S. Sparrow (Eds.), *Asperger syndrome*. New York: Guilford.
- Wolff, S. (1995). *Loners: The life path of unusual children*. London: Routledge.
- Ziadas, K., Durkin, K., & Pratt, C. (1998). Belief term development in children with autism, Asperger syndrome, specific language impairment, and normal development: Links to theory of mind development. *Journal of Child Psychology and Psychiatry*, 39, 755–763.
- Ziadas, K., Durkin, K., & Pratt, C. (2003). Differences in assertive speech acts produced by children with autism, Asperger syndrome, specific language impairment, and normal development. *Developmental Psychopathology*, 15, 73–94.