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**Mental Capital and Wellbeing:
Making the most of ourselves in the 21st century**

**State-of-Science Review: SR-D10
Autism Spectrum Conditions**

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Summary

The psychology of autism spectrum conditions is reviewed, with particular focus on the difficulties of empathy alongside the strong drive to systemise. This is followed by a review of the atypical neurology, and the evidence for genetic and endocrine factors in the aetiology of autism spectrum conditions. Next, the need for early diagnosis and early intervention is discussed. Finally, we look ahead to future horizons and highlight some key priorities: mental health in those with Asperger Syndrome (AS); ‘normalising’ autism spectrum conditions; understanding the difference between AS and classic autism; meeting the cost of the autism spectrum; the need for integrative research; and the need to put intervention on a rational basis.

1. Introduction

Autism is a *spectrum* condition, meaning that it is manifested to varying degrees of severity. At one extreme, a person may have no social skills, no language, and major learning difficulties. At the other extreme, the individual may have average or even above average IQ, precocious vocabulary (though a lack of interest in small-talk or chatting), and odd social skills (being one-sided or extremely self-centred). The former would receive a diagnosis of *classic autism*. The latter would receive a diagnosis of *Asperger Syndrome (AS)*. Both of these are subgroups on the autistic spectrum. Both also share a strong preference for routines and repetition, and ‘obsessional’ interest in highly specific topics. Up to 1% of the population are somewhere on the autistic spectrum.

2. Psychological aspects

The *empathising-systemising (E-S) theory* (Baron-Cohen, 2002) proposes that there are empathising deficits in autism, whilst systemising is either intact or superior. *Empathy* involves imagining another person’s thoughts and feelings, and having an appropriate emotional reaction to those feelings. Children and adults with AS show their empathising deficits on age-appropriate tests of emotion recognition, theory of mind, and spontaneous empathy (Baron-Cohen et al., 1999a; Baron-Cohen et al., 2001a).

Theory of mind (ToM) is the ability to attribute mental states to oneself or others and is regarded as the cognitive component of empathy. Emotion recognition is sometimes regarded as part of ToM because emotions are mental states. Often emotion-recognition deficits only appear if ‘complex’ emotions are tested, though in some individuals with autism the deficit is evident even when ‘basic’ emotions are tested. This deficit can make sense of the difficulties in social and communicative development, and in imagining others’ minds.

Systemising is the drive to analyse a system in terms of underlying rules, in order to understand and predict its behaviour. People with autism spectrum conditions show precocious understanding of systems, relative to their mental age, on tests of intuitive physics or questionnaires assessing how interested a person is in different types of systems (maps, train timetables, machines, syntax, etc) (Baron-Cohen et al., 2001b; Baron-Cohen et al., 2003; Lawson et al., 2004). The unusually strong repetitive behaviour, the strong desire for routines, and the ‘need for sameness’, can be seen as the result of a strong drive to systemise. Systemising also requires excellent attention to detail, and people with autism and AS are faster on visual search tasks (Plaisted et al., 1998). Strong systemising can therefore explain the strengths that people with autism and AS have.

There are two other major psychological theories: Executive Dysfunction and Weak Central Coherence. The Executive Dysfunction theory claims that people with autism have difficulty with planning or attention-

shifting (Russell, 1997). Although this is true of a subgroup of people on the autistic spectrum, there are people with AS who have no planning difficulties (in that they can, for example, excel in areas like mathematics) and no problem with attention-shifting (in that they can, for example, juggle).

Equally, the Weak Central Coherence theory claims that people on the autistic spectrum have trouble with integration of information (Frith, 1989). Whilst there is evidence for a failure to use context to the same extent, there are individuals who can integrate well enough to achieve a global grasp of a whole system (e.g. a computer or car engine). These latter two theories cannot, therefore, explain all the features of the condition.

3. Neurological aspects

Anatomical abnormalities have been identified in different brain regions in autism. These are not found in every case, and there are inconsistencies between studies, such that sometimes overgrowth is found, and sometimes undergrowth. The brain regions that have been reported to be atypical include the cerebellum (Murakami, 1989), corpus callosum (Egaas et al., 1995), hippocampus (Bauman and Kempner, 1985), and the amygdala (Aylward et al., 1999).

Epilepsy also occurs in a proportion of individuals with autism spectrum conditions, though the exact rate is no longer clear. Although in classic autism it is well established that one third of cases develop epilepsy by adolescence, in the Asperger subgroup these rates may be much lower and have not been systematically studied. In terms of neuropathology, the number of Purkinje cells in the cerebellar cortex is abnormally low (Murakami et al., 1989). Abnormalities have also been reported in the density of packing of neurons in the hippocampus, amygdala, and other parts of the limbic system (Bauman and Kempner, 1985). One report suggests a reduction in the size of cortical minicolumns (Casanova et al., 2002), though this remains to be confirmed in independent studies.

We can summarise functional neuroimaging studies of autism spectrum conditions as having found *increased* activity in sensory regions of the brain normally associated with stimulus-driven processing, and *decreased* activity in regions normally associated with higher-cognitive processing (Ring et al., 1999). Abnormalities have also been found in the amygdala (Baron-Cohen et al., 1999b), the orbito- (Baron-Cohen et al., 1994) and medial-frontal cortex (Happé et al., 1996). These atypical patterns of neural activity arise in relation to the empathising deficits.

Using MRI volumetric analysis, or measures of head circumference, some reports suggest the autistic brain involves transient postnatal macrocephaly (Courchesne et al., 2001). For example, neonates later diagnosed with autism have normal head circumference, but by two to four years of age, 90% of these have MRI-based brain volumes larger than average (Aylward et al., 2002). This may reflect an enlargement of cerebellar and cerebral white and grey matter. This increase in volume of cortical grey matter may, in turn, reflect a failure of synaptic pruning, or an excess of synaptogenesis. Again, independent confirmation of these abnormal growth rates is needed.

4. Genetic and hormonal aspects

The sibling risk-rate for autism shows a five- to ten-fold increase over general population rates. It used to be said that the sibling recurrence rate was much higher than this (50- to 100-fold), but this was based on old epidemiological prevalence rates of autism being 4 per 10,000, whereas today we recognise that 1% of children have an autism spectrum condition. The sibling recurrence rate is 5-10%.

Regarding twin studies, when a narrow phenotype is considered, 60% of MZ pairs are concordant for autism versus no DZ pairs. When a broader phenotype is considered, 92% of MZ pairs are concordant as compared to 10% of DZ pairs (Bailey et al., 1995). Molecular linkage genetic studies have led to a number of chromosomal regions being implicated, such as 2q, 7q, and 15q (Barrett et al., 1999; IMGSAC, 2001; Yonan et al., 2003). Loci on the X chromosome have also been implicated in autism, which may explain the sex ratio (markedly biased towards males), though these obviously cannot account for cases of male-to-male transmission.

The marked sex ratio in autism may also reflect hormonal factors. Currently, there are clues that foetal testosterone (FT) may play a role. Girls with congenital adrenal hyperplasia (CAH), a genetic condition involving over-production of FT, show more autistic traits than their unaffected sisters (Knickmeyer et al., 2006a). Within normal development, FT is inversely correlated with frequency of eye contact, rate of vocabulary development, empathy and social skills (Lutchmaya et al., 2002a; Lutchmaya, 2002b; Chapman et al., 2006), and FT is positively correlated with narrow interests and systemising (Auyeung et al., 2006).

In addition, there is preliminary evidence of somatic hypermasculinisation in autism, precocious puberty in boys, and delayed puberty in girls with autism and AS (Tordjman et al., 1997; Knickmeyer et al., 2006b), as well as elevated rates of testosterone-related medical conditions in women with AS and their mothers (Ingudomnukul et al., 2007). A comprehensive study of endocrine function in autism is still needed.

5. Early diagnosis and intervention

The earliest that classic autism has been reliably diagnosed is 18 months of age. This has been shown using a screening instrument (the CHAT, or Checklist for Autism in Toddlers) which tests for the absence of 'joint attention' behaviours such as pointing and gaze following, and the absence of pretend play, all of which are normally present by this age (Baron-Cohen et al., 1996). Population-based studies show that the CHAT has excellent specificity (children who fail on this test have a 83.3% chance of developing autism or a related pervasive developmental disorder), but low sensitivity (it only detects two out of every five cases, mostly missing the Asperger subgroup). Revisions of the CHAT are under way to improve the instrument further.

Asperger Syndrome can be reliably diagnosed by age five years. This has been shown using a screening instrument called the CAST (Childhood Asperger Screening Test) (Scott et al., 2002).

The most effective interventions for children on the autistic spectrum are special education, such as social skills teaching (Koegel et al., 1998), and Applied Behavioural Analysis (ABA), where appropriate skills and behaviours are taught through principles of reinforcement (Volkmar et al., 2004). The key ingredients for effective early intervention are that the methods are highly structured, intensive, and individualised. Appropriate cognitive interventions are also beneficial for teenagers and adults (Golan et al., 2006b).

Medical treatments are not usual. Indeed, there are ethical issues surrounding the notion of trying to 'cure' autism. Although some aspects of the condition do require help (e.g. the empathy difficulties), other aspects may not (e.g. the systemising talents).

6. Summary and ongoing research

Autism is a set of neurodevelopmental conditions affecting social and communication development and a narrow focus of attention. It affects boys far more often than girls. Its ultimate cause is likely to be genetic. Early diagnosis is possible from 18 months of age. Intervention is educational and psychological, rather than medical. International studies are underway to identify the genes that cause autism. Once these are

identified, there will be a need to understand their function, and determine which genes relate to the different aspects of the neurobiology, psychology, and behavioural manifestations of autism. Intervention research is also needed in order to understand which interventions work best for which subgroup of people with autism or AS.

7. Future horizons: mental health and Asperger Syndrome

Teenagers and adults with AS often suffer from additional mental health problems, the most common being depression. Many also feel suicidal; tragically some are so desperate as to attempt suicide, a proportion of whom actually die. The high levels of depression are not surprising if people with AS feel that they do not fit into society and feel rejected by the majority.

Many people with the condition have had an educational experience that is very negative, being bullied by peers in school, and failing academically because of following their own narrow interests instead of the expected broad range of subjects upon which the present UK national curriculum is based. If social success depends on social skills, then people with AS are always going to be at a disadvantage relative to others. If educational success at school is based on being a generalist rather than a specialist, then again people with AS will be disadvantaged educationally.

One way to avoid the secondary risks of depression and of dropping out of school would be to enable people with AS to feel valued if they are good at one narrow subject (e.g. 'the history of the Battle of Liege, 1914'), even if they have over-specialised to such an extent that they have not covered the whole of the History syllabus, let alone the national curriculum. Valuing people for what they *can* do rather than what they *cannot* do would also enable people with AS to feel positive about their strong, narrowly-focused systemising skills even in the face of their social and empathising difficulties. This requires both a change in educational policy and attitude. Even among the higher-functioning individuals with autism or AS, support in adolescence and adulthood is clearly inadequate. In the UK, according to the National Autistic Society survey, 90% of such adults are unemployed, despite having an average or above average IQ. This high-functioning sub-group, who for the most part attend mainstream schools, are at high risk of being bullied, dropping out prior to GCSEs, being excluded for difficult (non-conformist) behaviour, and (as mentioned above) developing depression because of their social isolation. Such difficulties can be viewed as secondary to the autism or AS and are preventable with the right support.

8. Future horizons: normalising autism spectrum conditions

One new idea is to see the high-functioning autistic spectrum as a dimension of traits that runs right through the population. Using a metric of autistic traits called the Autism Spectrum Quotient (AQ), population studies have confirmed that these show an approximately normal distribution (Baron-Cohen et al, 2001c). This means that, rather than thinking of people with autism or AS as different to everyone else, we can see them on a continuum with everyone else.

The E-S theory recognises that different people show different size discrepancies between E (empathising) and S (systemising). Whilst people with autism and AS show a large discrepancy between these ($S \gg E$), there are others who show smaller discrepancies in the same direction ($S > E$), a profile that is more common among males and prompting the theory that autism may be no more than an extreme of the male brain. Equally, some individuals show a discrepancy in the opposite direction ($E > S$), a profile more common among females, and there are even individuals who show the mirror image of autism ($E \gg S$).

Both the E-S theory and the AQ suggest that society needs to be tolerant of different cognitive styles, rather than expecting everyone to conform to a single (average) profile. Acknowledging that there is no single profile that counts as 'normal' may help change attitudes towards those who show minority profiles and help normalise autism spectrum conditions, again reducing the risk of making people with high-functioning autism and AS feel marginalised.

9. Classic autism versus Asperger Syndrome

Whilst the above discussion applies right across the autistic spectrum, it remains the case that those individuals on the autistic spectrum who also have below average IQ (and therefore are at risk of broader developmental delays) are likely to need special support right across their lives. They will remain 'vulnerable' even as adults. The issues and challenges that their low IQ raises are in some respects no different to those raised by low IQ in other individuals (who are not on the autistic spectrum) and are probably best served by the same Learning Disability agencies.

10. The cost of autism spectrum conditions

To date there has only been one economic analysis of the impact of autism on society. Knapp and Jarbrink in 2007 calculated that autism costs the UK £28 billion per annum, with the cost per individual with autism being £2.9 million over their lifetime. Such estimates include the impact on families that can be major, leading to loss of earnings of a carer, and even suicide by a parent. It will be important to establish indices to measure if the cost is reducing using new strategies and interventions.

11. Future horizons: where does science need to focus?

This review highlights that we have had a fair amount of research in disparate areas (genetics, neuroimaging, hormones, neuropathology, psychology, intervention), but almost no research of an integrative kind, which attempts to relate findings in one area (e.g. genetics) to another (e.g. neuroimaging), let alone to a third (e.g. hormones) or a fourth (cognition). Integrative research will ultimately be needed in order for us to make sense of the significance of findings in any one domain. Whilst this has happened in other fields such as depression (a well-known example being how the short version of the serotonin transporter gene affects treatment outcome) or in affective neuroscience (how, for example, polymorphisms in the cannabinoid receptor gene CNR1 affects the brain's response to happy faces) (Chakrabarti et al., 2006), such approaches have not really been tried in autism.

12. Putting intervention on a rational basis

For many years, 'treatment' in autism has proceeded on the basis of an approach that has been tried and tested but without any real rationale for why it should be effective. ABA is one such example. Here, one can see that the principles behind it would enable target skills to be broken down into simpler units to be acquired through shaping and mass practice. Whilst there is some evidence for the effectiveness of ABA, the methods require external reinforcers or rewards to maintain the child's attention and cooperation, suggesting that they are not as autism-friendly as they could be.

Newer interventions, in contrast, are designed to harness individuals' areas of strength and their natural interests as a means for building new skills. Two examples of these are:

- a) Teaching emotion-recognition via computers using the *Mindreading* DVD educational software (Golan et al., 2006a);
- b) Teaching emotion-recognition by presenting emotional expressions on toy vehicles using the *Transporters* animation (Baron-Cohen, 2007).

In the case of the *Mindreading* DVD (www.jkp.com/mindreading), the individual's natural interest in lawful, predictable computers and in information being systematically organised renders the domain of emotions easier to learn about. In the case of the *Transporters* animation (www.thetransporters.com), the child's natural interest in the mechanical, predictable motion of vehicles means they are attending to the film, thereby enabling implicit learning of emotions since these are grafted onto the vehicles (See Figure below). In this way, the domain of emotions is separated from their usual context (real-time social interaction). Rather than expecting the child with autism to join the social world, with all its attendant unpredictability, social information is taken to the child's safer world of computers or mechanical vehicles.



Figure 1: Screen shots from *The Transporters* (www.thetransporters.com)

Such methods of intervention are rational in that they are based on cognitive theory (in this case hyper-systemising). Other examples include Lego-Therapy which also exploits the child's strong interest in systems (in this case, constructional systems) to encourage turn-taking and social communication (LeGoff, 2004; Owens, submitted).

A final example of a 'treatment' approach based on rational principles is the new trial of oxytocin, given that animal research shows this hormone enhances social processing and social interaction (Insel, 1997) and has similar effects in humans (Domes et al., 2007). The key issue will be to ensure that treatments are targeted at the features of autism that are disabling while leaving other features that are assets rather than 'symptoms' untouched and free to develop.

13. Some useful links

- The National Autistic Society is the main charity in the UK for families with a child on the autistic spectrum: www.nas.org.
- The Autism Research Centre, Cambridge University, contains a searchable database of publications and screening instruments such as the CAST, AQ, and CHAT: www.autismresearchcentre.com.
- As interventions are scientifically evaluated, the results of such studies are summarised at www.researchautism.net.

14. Further reading

The following reviews may be useful: *Psychological Aspects* (Baron-Cohen, 2002); *Neurobiological and Genetic Aspects* (Baron-Cohen, 2005a); *Hormonal Aspects* (Baron-Cohen, 2005b)

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