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Autism:
Research into causes and intervention

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Autism is diagnosed when a child or adult has abnormalities in a 'triad' of behavioural domains: social development, communication, and repetitive behaviour/obsessive interests (APA, 1994; ICD-10, 1994). Autism can occur at any point on the IQ continuum, and IQ is a strong predictor of outcome (Rutter, 1978). Autism is also invariably accompanied by language delay (no single words before 2 years old). Asperger Syndrome (AS) (Asperger, 1944) is a subgroup on the autistic spectrum. People with AS share many of the same features as are seen in autism, but with no history of language delay and where IQ is in the average range or above. The main cognitive theories of autism are summarised next:

Cognitive theories

a. The Mindblindness theory

The mindblindness theory of autism (Baron-Cohen, 1995) proposed that in autism spectrum conditions there are deficits in the normal process of empathizing, relative to mental age. These deficits can occur by degrees (relative to mental age). The term 'empathizing' encompasses a range of other terms: 'theory of mind', 'mind-reading', 'empathy', and taking the 'intentional stance' (Dennett, 1987). Empathizing involves two major elements: (a) the ability to attribute mental states to oneself and others, as a natural way to make sense of agents (Baron-Cohen, 1994; Leslie, 1995; Premack, 1990); and (b) having an emotional reaction that is appropriate to the other person's mental state. In this sense, it goes beyond what is normally meant by the term 'theory

of mind' (Wellman, 1990) to include having some affective reaction (such as sympathy).¹

Since the first test of mindblindness in children with autism (Baron-Cohen, Leslie, & Frith, 1985), there have been more than 30 experimental tests. The vast majority of these have revealed profound impairments in the development of their empathizing ability. These are reviewed elsewhere (Baron-Cohen, 1995; Baron-Cohen, Tager-Flusberg, & Cohen, 1993). Some children and adults with AS only show their empathising deficits on age-appropriate adult tests (Baron-Cohen, Jolliffe, Mortimore, & Robertson, 1997; Baron-Cohen, Wheelwright, Hill, Raste, & Plumb, 2001; Baron-Cohen, Wheelwright, & Jolliffe, 1997). This deficit in their empathizing is thought to underlie the difficulties such children have in social and communicative development (Baron-Cohen, 1988; Tager-Flusberg, 1993), and in the imagination of others' minds (Baron-Cohen, 1987; Leslie, 1987).

b. The Empathizing-Systemizing (E-S) theory

Systemizing is the drive to analyze and build systems, in order to understand and predict the behaviour of non-agentive (inanimate) events. Systems are all around us in our environment, and include technical systems (such as machines and tools); natural systems (such as biological and geographical phenomena); abstract systems (such as mathematics or computer programs); and even social systems (such as profits and losses in a business, or a football league table). The way we make sense of any of these systems is in terms of underlying rules and regularities, or specifically an analysis of

¹ Sympathy is considered here as a special case of empathy, where the observer's affective response to another's affect is to want to alleviate suffering in some way (Baron-Cohen & Wheelwright, in press).

input-operation-output relationships (Baron-Cohen, 2002). The E-S theory holds that alongside the empathizing deficits in autism, systemizing is either intact or superior (Baron-Cohen, Wheelwright, Griffin, Lawson, & Hill, 2002). The evidence for deficits in autism was reviewed earlier, but studies suggest systemizing in autism is at least in line with mental age, or superior (Baron-Cohen, Leslie, & Frith, 1986; Baron-Cohen, Richler, Bisarya, Gurunathan, & Wheelwright, 2003; Baron-Cohen, Wheelwright, Scahill, Lawson, & Spong, 2001).

c. Executive function theory

People with autism spectrum conditions show “repetitive behaviour”, a strong desire for routines, and a “need for sameness”. To date, the only cognitive account to attempt to explain this aspect of the syndrome is the executive dysfunction theory (Ozonoff, Rogers, Farnham, & Pennington, 1994; Pennington et al., 1997; Russell, 1997a). This paints an essentially negative view of this behaviour, assuming that it is a form of ‘frontal lobe’ perseveration or inability to shift attention. People with autism who have additional learning disabilities are more likely to show executive deficits (Russell, 1997b). But the fact that it is possible for people with AS to exist with no demonstrable executive dysfunction whilst still having deficits in empathizing and talents in systemizing (Baron-Cohen, Wheelwright, Stone, & Rutherford, 1999) suggests that executive dysfunction cannot be a core feature of autism spectrum conditions.

The executive account has also traditionally ignored the *content* of “repetitive behaviour”. The empathizing-systemizing theory in contrast draws attention to the

fact that much repetitive behaviour involves the child's 'obsessional' or strong interests with mechanical systems (such as light switches or water faucets) or other systems that can be understood in terms of rules and regularities. Rather than these behaviours being a sign of executive dysfunction, these may reflect the child's intact or even superior interest in systems. One study suggests that autistic obsessions are not random with respect to content (which would be predicted by the content-free executive dysfunction theory), but that these tend to cluster in the domain of systems (Baron-Cohen & Wheelwright, 1999).

d. Central coherence (CC) theory

The normal brain is held to show 'strong' central coherence (or Gestalt processing), i.e. a preference for global over local processing. 'Weak' central coherence (Frith, 1989; Happe, 1996) refers to the individual's preference for local detail over global processing. This has been demonstrated in terms of an autistic superiority on the Embedded Figures Task (EFT) (Witkin, Dyk, Faterson, Goodenough, & Karp, 1962) and the Block Design subtest of the Weschler IQ tests (Jolliffe & Baron-Cohen, 1997; Shah & Frith, 1983, 1993). It has also been demonstrated in terms of an autistic deficit in integrating fragments of objects and integrating sentences within a paragraph (Jolliffe & Baron-Cohen, 2000, 2001). The faster and more accurate performance on the EFT and Block Design Test have been interpreted as evidence of good segmentation skills, and superior attention to detail. The latter has also been demonstrated on visual search tasks (Plaisted, O'Riordan, & Baron-Cohen, 1998a, 1998b).

Systemizing requires as a first stage, excellent attention to detail, identifying parameters that may then be tested for their role in the behaviour of the system under examination. So, both the E-S theory and the CC theory predict excellent attention to detail. However, the E-S and CC theories also make opposite predictions when it comes to an individual with autism being able to understand a whole system. The E-S theory predicts that a person with autism, faced with a new system to learn, will show a stronger drive to learn the system, compared to someone without autism, so long as there are underlying rules and regularities that can be discovered. Moreover, they will readily grasp that a change of one parameter in one part of the system may have distant effects on another part of the system. In contrast, the CC theory predicts that they should fail to understand whole (global) systems or the relationships between parts of a system. This has not yet been tested.

Autism and the brain

A neural basis of empathy or social intelligence was first proposed by Brothers (Brothers, 1990). She suggested from animal lesion studies (Kling & Brothers, 1992), single cell recording studies (Brothers, Ring, & Kling, 1990), and neurological studies that social intelligence was a function of three regions: the amygdala, the orbito-frontal cortex (OFC), and the superior temporal sulcus and gyrus (STG). Together, she called these the “social brain”. In this next section, we focus particularly on the role of the amygdala in social intelligence (Adolphs, Baron-Cohen, & Tranel, 2002).

The amygdala

There are two important lines of evidence implicating the amygdala in primate social behaviour. Extensive reviews exist elsewhere (Kling & Brothers, 1992). Here we summarise two main lines of evidence.

a) Lesions of the primate amygdala affect social behaviour

Amygdala-lesioned monkeys become socially isolated. They fail to initiate social interactions and to respond appropriately to social gestures (Kling & Brothers, 1992; Kling & Steklis, 1976).

b) Neuroimaging studies in humans

The human amygdala is activated in humans when decoding signals of social importance, such as gaze, expression-recognition (especially of fearful faces), and body movements) (Baron-Cohen, Ring et al., 1999; Bonda, Petrides, Ostry, & Evans, 1996; Kawashima et al., 1999; Morris et al., 1996; Whalen et al., 1998; Wicker, Michel, Henaff, & Decety, 1998).

There are four lines of evidence for an amygdala deficit in autism (Baron-Cohen et al., 2000).

(a) Post-mortem evidence

A neuroanatomical study of adults with autism at post-mortem found microscopic pathology (in the form of increased cell density) in the amygdala, in the presence of normal amygdala volume (Bauman & Kemper, 1994; Rapin & Katzman, 1998).

(b) Similarities between autism and patients following amygdalotomy

Patients with amygdala lesions show impairments in social judgement (Adolphs, Tranel, Damasio, & Damasio, 1994; Young, Hellawell, De Wal, & Johnson, 1996) that have been likened to “acquired autism” (Stone, 2000). The age of onset of deficits in acquired vs. idiopathic cases is likely to mean that the two syndromes also differ in many ways, too. Similarly, patients with autism tend to show a similar pattern of deficits to those seen in patients with amygdala lesions (Adolphs, Sears, & Piven, 2001).

(c) Structural neuroimaging

A recent structural magnetic resonance imaging study of autism reported reduced amygdala volume (Abell et al., 1999). This is not the only structural abnormality in the brain (see below), but the amygdala abnormality has some potential relevance to the social symptoms observed. It is not yet known why this difference occurs.

(d) Functional neuroimaging

Using single photon emission computed tomography (SPECT), patients with autism spectrum conditions show significant reductions in temporal lobe blood flow. This is not simply an effect of temporal lobe epilepsy (Gillberg, Bjure, Uvebrant, Vestergren, & Gillberg, 1993). In a recent functional magnetic resonance imaging (fMRI) study, adults with High Functioning Autism (HFA) or Asperger Syndrome (AS) showed significantly less amygdala activation during a mentalizing task (Reading the Mind in the Eyes task), compared to normal (Baron-Cohen, Ring et al., 1999).

Other brain areas that might be abnormal in autism

Whilst the above section highlights the likely role an amygdala abnormality might play in autism, it is likely that this is not the only abnormal neural region. For example, the case has been made for anomalous functioning in the cerebellum (Courchesne et al., 1994), hippocampal formation (DeLong, 1992), left medial frontal cortex (Happé et al., 1996), and fronto-limbic connections (Bishop, 1993) in autism. Reduced neuron size and increased cell-packing density has also been found in the limbic system, specifically the hippocampus, subiculum, entorhinal cortex, amygdala, mammillary bodies, anterior cingulate, and septum in autism (Bauman & Kemper, 1988; Bauman & Kemper, 1994; Bauman & Kempner, 1985; Bauman & Kempner, 1986; Raymond, Bauman, & Kemper, 1996). A full review of neuroimaging of autism may be found elsewhere (Filipek, 1999).

Genetics

Ultimately, the behavioural, cognitive, affective, and neural abnormalities in autism spectrum conditions are likely to be due to genetic factors. For example, in an epidemiological study of same-sex autistic twins, studying 27 pairs of MZ twins and 20 DZ twins, it was found that 60% of MZ pairs were concordant for autism vs 0% of DZ pairs (Bailey et al., 1995). When this study considered a broader phenotype (of related cognitive or social abnormalities), 92% of MZ pairs were concordant vs. 10% of DZ pairs. The high concordance in MZ twins indicates a high degree of genetic influence. Molecular genetic studies are beginning to narrow down candidate regions on certain chromosomes. The International Molecular Genetic Study of Autism Consortium (IMGSAC) (IMGSAC, 1998) conducted a 2-stage genome search for susceptibility loci in autism in 87 affected sib pairs plus 12 non-sib affected relative-pairs, from a total of 99 families. Regions on 6 chromosomes were identified that generated a multipoint maximum lod score of greater than 1. A region on chromosome 7q was the most significant, with a maximum lod score of 3.55 near markers D7S530 and D7S684 in the subset of 56 U.K. affected sib-pair families, and a maximum lod score of 2.53 in all 87 affected sib-pair families.

There may also be a relationship between autism and specific language impairment (SLI) because genetic studies in each disorder point to a locus on chromosome 7q31 (Folstein & Mankoski, 2000). The IMGSAC's later study (IMGSAC, 2001) analyzed 125 sib pairs meeting stringent inclusion criteria and found a multipoint maximum lod score of 2.15 at marker D7S477, whereas analysis of all 153 sib pairs generated a multipoint maximum lod score of 3.37. Linkage disequilibrium mapping identified 2

regions of association. One was under the peak of linkage, the other was 27 cM distal. A review of published full-genome scans and found that a region of approximately 50 cM on 7q appeared to play a role in the aetiology of autistic disorder (Gutknecht, 2001). It was noted, however, that the finding must be considered with caution because lod score values did not reach the threshold for significant linkage.

Note that chromosome 7q is not the only focus of attention. For example, there has been a reported linkage evidence for a susceptibility gene for autism on chromosome 2 (Buxbaum et al., 2001). That study found a maximum multipoint heterogeneity lod score (hlod) of 1.96 and a maximum multipoint nonparametric linkage (NPL) score of 2.39 on 2q in an analysis of 95 affected-relative-pair families.

As of yet, specific genes for autism have not yet been identified, despite the encouraging possibility of candidate regions on chromosomes. The future of research in this field will be not only to isolate the relevant genes but also to understand the function of these genes, and ultimately the relationships between these different causal levels in autism. It is hoped that during this research endeavour there will also be evaluations of the most promising interventions.

Targeting interventions

The standard interventions for autism spectrum conditions in childhood are behavioural (Applied Behavioral Analysis, or ABA (Lovaas & Smith, 1988), speech therapy (where appropriate), and special education. A meta-analysis of treatment effects suggest that no one approach is better than others, but that interventions are most

successful when they start early, are intensive, and are highly structured (Howlin, 1998). One example of tailoring special education to key areas of cognitive deficit is educational software for emotion recognition (Baron-Cohen & The-Human-Emotions-Team, 2003) which allows people on the autistic spectrum to study emotion recognition using a DVD-ROM, so that they can determine their own pace and developmental level for learning. There is a need for treatment trials to evaluate the relative benefits of such approaches.

Summary

Autism spectrum conditions are neuro-developmental syndromes, with strong heritability. Cognitive theories have had some success in explaining why the cluster of features should co-occur. Empathizing deficits have the potential to make sense of one triad of impairments (social difficulties, communication difficulties, and imagining others' minds), and may have a brain basis in the amygdala and left medial frontal cortex. A strong systemizing drive may account for a distinct triad of strengths (good attention to detail, deep, narrow interests and islets of ability). The brain basis of systemizing is yet to be understood. Family genetics studies suggest that these same cognitive dimensions (reduced empathizing alongside a strong drive to systemize) may also characterize the "broader phenotype" among first-degree relatives. Molecular genetic studies are underway and any candidate genes for autism will ultimately need to be tested in relation to the observed differences in the brain, cognition, and behaviour. The ethics of genetic screening or gene therapy should be thought about well ahead of these becoming available, since there is by no means any consensus that these would be desirable, given the wide range of phenotypic traits, not all of which are disabling. Future research will need to focus on evaluating the extent to which any

form of intervention reduces the triad of impairments whilst supporting the triad of strengths.

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