This paper appeared in In Tager-Flusberg, H, (ed) Neurodevelopmental Disorders.
MIT Press (1999)

The extreme-male-brain theory of autism

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Acknowledgements: I am grateful for support from the MRC (UK), the Wellcome Trust, and the Gatsby Foundation, during the preparation of this work. Parts of this chapter are reprinted from Baron-Cohen and Hammer (in press a), and Baron-Cohen and Swettenham (in press), with permission.

1. Autism

Autism is widely regarded to be the most severe of the childhood psychiatric conditions (Rutter, 1983; Frith, 1989; Baron-Cohen, 1995). It is diagnosed on the basis of abnormal social development, abnormal communicative development, and the presence of narrow, restricted interests, and repetitive activity, along with limited imaginative ability (DSM-IV, 1994). Such children fail to become social, instead remaining on the periphery of any social group, and becoming absorbed in repetitive interests and activities, such as collecting unusual objects or facts. It is a tragedy for their families who work tirelessly to attempt to engage with and socialize their child, mostly with very limited results.

In this chapter, I begin by summarizing psychological findings from studies of autism. A brief review of genetic evidence appears next, as a bridge into the next section, where a recent notion is introduced: the "male brain". Evidence for biologically-based psychological sex differences is presented, and the "male brain" is defined. Finally, I relate this notion to autism, summarizing our new theory (Baron-Cohen and Hammer, 1996a) that autism is an extreme form of the male brain. This theory makes a number of predictions possible, and the current evidence relevant to these predictions is presented.

2. Psychological theories of autism

In this section, evidence for three psychological theories is reviewed: the mindblindness theory, the central coherence theory, and the executive dysfunction theory.

The mindblindness theory

Our early theory of autism suggested that the social and communicative abnormalities in this syndrome could be the result of an impairment in the development of a "theory of mind", or the capacity for "mindreading". This is defined as the ability to attribute mental states to oneself and others, and make sense of and predict behaviour on the basis of mental states. This is held to be important to autism simply because it is arguably the main way in which the normal individual succeeds in understanding and participating in social relationships and communication.

Wimmer and Perner (1983) devised an elegant paradigm to test when normally developing children show evidence of possessing a theory of mind - specifically, when they are aware of another person's beliefs. The child was presented with a short story, with the simplest of plots. The story involves one character not being present when an object is moved, and therefore not knowing that the object is in a new location. The child being tested is asked where the character thinks the object is. Wimmer and Perner called this the False Belief test, since the focuses on the subject's ability to infer a story character's mistaken belief about a situation. These authors found that normal 4 year olds correctly infer that the character thinks the object is where the character last left it, rather than where it actually is. This is impressive evidence for the normal child's ability to distinguish between their own knowledge (about reality) and someone else's false belief (about reality).

When this test was given to a sample of children with autism, with mild degrees of mental retardation, a large majority of them 'failed' this test by indicating that the character thinks the object is where it actually is (Baron-Cohen, Leslie, and Frith, 1985). That is, they appeared to disregard the critical fact that, by virtue of being absent during the critical scene, the character's mental state would necessarily be different to the child's own mental state. In contrast, a control group of children with Down Syndrome, with moderate degrees of mental retardation, passed this test as easily as the normal children. The implication was that the ability to infer mental states may be an aspect of social intelligence that is relatively independent of general intelligence (Cosmides, 1989), and that children with autism might be specifically impaired in the development of a theory of mind.

Of course, simply failing one test would not necessarily mean that children with autism lacked awareness of the mind. There might be many reasons for failure on such a test. (Interestingly, control questions in the original procedure ruled out memory, or language difficulties, or inattention as possible causes of failure). The conclusion that children with autism are indeed impaired in this domain only becomes possible because of the convergence of results from widely differing experimental paradigms. These are reviewed in detail in an edited volume (Baron-Cohen, Tager-Flusberg, and Cohen, 1993) and for that reason are only briefly summarized here, next.

Summary of results from studies of mind-reading in children with autism [1]

[1] In the following list of studies, all of the tests mentioned are at the level of a normal 4 year old child.

The majority of children with autism

(i) are at chance on tests of the mental-physical distinction (Baron-Cohen, 1989a). That is, they do not show a clear understanding of how physical objects differ from thoughts about objects. For example, when asked which can be touched: a biscuit, or a thought (about a biscuit), young normal 3 year olds rapidly identify the former, whereas most children with autism respond at chance levels.

(ii) They also have an appropriate understanding of the functions of the brain, but have a poor understanding of the functions of the mind (Baron-Cohen, 1989a). That is, they recognize that the brain's physical function is to make you move and do things, but they do not spontaneously mention the mind's mental function (in thinking, dreaming, wishing, deceiving, etc.,). Again, contrast this with normal 3 year old children who do spontaneously use such mental state terms in their descriptions of what the mind is for (Wellman and Estes, 1983).

(iii) Most children with autism also fail to make the appearance-reality distinction (Baron-Cohen, 1989a), meaning that, in their description of misleading objects (like a red candle in the shape of an apple), they do not distinguish between what the object looks like, and what they know it really is. For example, the normal 4 year old child will say of an ambiguous object, when asked what it looks like, and what it really is, that "It looks like an apple, but really it's a candle made of wax" (Flavell, Flavell, and Green, 1983). In

contrast, children with autism tend to refer to just one aspect of the object (e.g., saying "It looks like an apple, and it really is an apple").

(iv) Most children with autism fail a range of first-order false belief tasks, of the kind described in the previous section (Baron-Cohen et al, 1985, 1986; Perner, Frith, Leslie, and Leekam, 1989; Swettenham, 1996; Reed and Petersen, 1990; Leekam and Perner, 1991). That is, they show deficits in thinking about someone else's different beliefs.

(v) They also fail tests assessing if they understand the principle that "seeing leads to knowing" (Baron-Cohen and Goodhart, 1994; Leslie and Frith, 1988). For example, when presented with two dolls, one of whom touches a box, and the other of whom looks inside the box, and when asked "Which one knows what's inside the box?", they are at chance in their response. In contrast, normal children of 3-4 years of age correctly judge that it is the one who looked, who knows what's in the box.

(vi) Whereas normally developing children are rather good at picking out mental state words (like "think", "know", and "imagine") in a wordlist that contains both mental state and non-mental state words, most children with autism are at chance (Baron-Cohen, Ring, Moriarty, Shmitz, Costa, and Ell, 1994). In contrast, they have no difficulty in picking out words describing physical states.

(vii) Nor do most children with autism produce the same range of mental state words in their spontaneous speech (Tager-Flusberg, 1992; Baron-Cohen et al, 1986). Thus, from

about 18-36 months of age, normally developing children spontaneously use words like "think", "know", "pretend", "imagine", "wish", "hope", etc., and use such terms appropriately (Wellman, 1990). In contrast, such words occur less frequently, and are often even absent, in the spontaneous speech of children with autism.

(viii) They are also impaired in the production of spontaneous pretend play (Baron-Cohen, 1987; Wing, Gould, Yeates, and Brierley, 1977; Lewis and Boucher, 1988). Pretend play is relevant here simply because it involves understanding the mental state of pretending. The normal child of even 2 years old effortlessly distinguishes between when someone else is acting veridically, versus when they are "just pretending" (Leslie, 1987). Sometimes mommy is actually eating (putting a real spoon with real food into her mouth), whilst at other times mommy is just pretending to eat (holding a pen to her lips, and making funny slurping noises, in between her smiles).

Young normal children make rapid sense of such behaviour, presumably because they can represent the latter case as being driven by the mental state of "pretending". They also spontaneously generate examples of pretence themselves, and do not show any confusion as they switch back and forth between pretence (the mental world), and reality (the physical world). In contrast, most children with autism produce little pretence, and often appear confused about what pretence is for, and when someone is or is not pretending.

(ix) Whilst they can understand simple causes of emotion (such as reactions to physical situations), the majority of children with autism have difficulty understanding more mentalistic causes of emotion (such as beliefs) [Baron-Cohen, 1991a; Baron-Cohen, Spitz, and Cross, 1993]. For example, they can understand that if Jane actually falls over and cuts her knee, she will feel sad, and that if John actually gets a present, he will feel happy. But they are poor at understanding that if John thinks he's getting a present (even if in reality he is not), he will still feel happy. In contrast, normal 4 year old children comprehend such belief-based emotions.

(x) Most children with autism also fail to recognize the eye-region of the face as indicating when a person is thinking and what a person might want (Baron-Cohen and Cross, 1992; Baron-Cohen, Campbell, Karmiloff-Smith, Grant, and Walker, 1995). Children and adults without autism use gaze to infer both of these mental states.

For example, when presented with pairs of photos like those in Figure 1, normal 3-4 year olds easily identify the person looking upwards and away as the one who is thinking. Children with autism are less sure of this. And when shown a display like the one in Figure 2, normal 4 year olds identify the candy that Charlie is looking at as the one he wants. Children with autism mostly fail to pick up that gaze can be an indicator of what a person might want.

insert Figures 1 and 2 here

In addition:

(xi) Many children with autism fail to make the accidental-intentional distinction (Phillips, 1993). That is, they are poor at distinguishing if someone "meant" to do something, or if something simply happened accidentally.

(xii) They also seem unable to deceive (Baron-Cohen, 1992; Sodian and Frith, 1992), a result that would be expected if one was unaware that people's beliefs can differ and therefore can be manipulated. In contrast, normal children of 4 begin to be quite adept at lying, thus revealing their awareness of the mental lives of others.

(xiii) Most children with autism also have disproportionate difficulty on tests of understanding metaphor, sarcasm, and irony - these all being statements which cannot be decoded literally, but which are only meaningful by reference to the speaker's intention (Happe, 1994). An example would be understanding the phrase "the drinks are on the house", which one adult with autism (of above average IQ) could only interpret literally. This suggests that children with autism are aware of the physical (the actual words uttered), but are relatively unaware of the mental states (the intentions) behind them.

(xiv) Indeed, most children with autism fail to produce most aspects of pragmatics in their speech (reviewed in Baron-Cohen, 1988; and Tager-Flusberg, 1993), and fail to recognize violations of pragmatic rules, such as the Gricean Maxims of conversational cooperation (Surian, Baron-Cohen, and Van der Lely, 1996). For example, one Gricean

Maxim of conversation is "Be relevant". If someone replies to a question with an irrelevant answer, normal young children are very sensitive to this pragmatic failure, but most children with autism are not. Since many pragmatic rules involve tailoring one's speech to what the listener expects, or needs to know, or might be interested in, this can be seen as intrinsically linked to a sensitivity of another person's mental states.

(xv) Crucially, most children with autism are unimpaired at understanding how physical representations (such as drawings, photos, maps, and models) work, even while they cannot understand mental representations (such as beliefs) [Charman and Baron-Cohen, 1992, 1995; Leekam and Perner, 1991; Leslie and Thaiss, 1992]. To the extent that both types of task require understanding of representation, this suggests there is something special about understanding mental representations that causes problems in autism.

(xvi) They are also unimpaired on logical reasoning (about the physical world) even though they have difficulty in psychological reasoning (about the mental world) [Scott and Baron-Cohen, 1996).

This long list of experiments provides strong evidence for children with autism lacking the normal understanding of mental states. For this reason, autism can be conceptualized as involving degrees of mindblindness (Baron-Cohen, 1990, 1995).

It is important to mention that a small minority of children or adults with autism pass first-order false belief tests. (First-order tests involve inferring what one person thinks).

However, these individuals often fail second-order false belief tests (Baron-Cohen, 1989b), that is, tests of understanding what one character thinks another character thinks. Such second-order reasoning is usually understood by normal children of 5-6 years of age, and yet these tests are failed by individuals with autism with a mental age above this level.

We can therefore interpret these results in terms of there being a specific developmental delay in mind-reading at a number of different points (Baron-Cohen, 1991b). Some individuals with autism who are very high functioning (in terms of IQ and language level), and who are usually adults, may pass even second-order tests (Bowler, 1992; Ozonoff, Pennington, and Rogers, 1991; Happe, 1993). Those who can pass second-order tests correspondingly also pass the appropriate tests of understanding figurative language (Happe, 1993). However, their deficit shows up on tests of adult mind-reading (Baron-Cohen, Jolliffe, Mortimore, and Robertson, in press). Thus, being able to pass a test designed for a 6 year old when you are an adult may mask persisting mind-reading deficits by ceiling effects.

In summary, there appears to be an impairment in the development of a theory of mind in the majority of cases with autism. This finding has the potential to explain the social, communicative, and imaginative abnormalities that are diagnostic of the condition, since being able to reflect on one's own mental states (and those of others) would appear to be essential in all of these domains. This deficit has been found to correlate with real-life social skills, as measured by a modified version of the Vineland Adaptive Behaviour

Scale (Frith, Happe, and Siddons, 1994). In the next sections, we turn to consider the neural and developmental origins of this cognitive deficit.

The brain basis of theory of mind

One possibility arising from these studies is that there may be a particular part of the brain which in the normal case is responsible for our mindreading ability, and which is specifically impaired in autism. If this view is correct, the assumption is that this may be for genetic reasons, since autism appears to be strongly heritable (see Santangelo and Folstein, chapter 17, this volume). The idea that the development of our theory of mind is under genetic control in the normal case is consistent with evidence from cross-cultural studies: Normally developing children from markedly different cultures seem to pass tests of 'mind-reading' at roughly the same ages (Avis and Harris, 1991).

Quite which parts of the brain might be involved in this is not yet clear, though candidate regions include right orbito-frontal cortex, which is active when subjects are thinking about mental state terms during functional imaging using SPECT (Baron-Cohen, Ring, et al, 1994); and left medial frontal cortex, which is active when subjects are drawing inferences about thoughts whilst being PET scanned (Fletcher, Happe, Frith, Baker, Dolan, Frackowiack, and Frith, 1995; Goel, Grafman, Sadato, and Hallett, 1995). Other candidate regions include the superior temporal sulcus and the amygdala (for reasons explained later). These regions may form parts of a neural circuit supporting theory of mind processing (Baron-Cohen and Ring, 1994).

Developmental origins of theory of mind

In an influential article, Alan Leslie (1987) proposed that in the normal case, the developmental origins of mind-reading lie in the capacity for pretence; and that in the case of children with autism, the developmental origins of their mindblindness lies in their inability to pretend. In his model, pretence was the 'crucible' for theory of mind, as both involved the same computational complexity. Thus (according to Leslie), in order to understand that someone else might think "This banana is real", or pretend "This banana is real", the child would need to be able to represent the agent's mental attitude towards the proposition - since the only difference between these two states of affairs *is* the person's mental attitude. One idea, then, is that mindreading is first evident from about 18-24 months of age, in the normal toddler's emerging pretend play.

However, there is some evidence that this abiility might have even earlier developmental origins. Soon after the first demonstrations of mindblindness in autism, Marian Sigman and her colleagues also reported severe deficits in joint attention in children with autism (Sigman, Mundy, Ungerer, and Sherman, 1986). Joint attention refers to those behaviours produced by the child which involve monitoring or directing the target of attention of another person, so as to coordinate the child's own attention with that of somebody else (Bruner, 1983). Such behaviours include the pointing gesture, gaze-monitoring, and showing gestures, most of which are absent in most children with autism.

This was an important discovery because joint attention behaviours are normally well-developed by 14 months of age (Scaife and Bruner, 1975; Butterworth, 1991), so their absence in autism signifies a very early-occurring deficit. This was also important because the traditional mind-reading skills referred to above are mostly those one would expect to see in a 3-4 year old normal child. Deficits in these areas cannot therefore be the developmentally earliest signs of autism, since we know that autism is present from at least the second year of life (Rutter, 1978), if not earlier.

Implicit in the idea of joint attention deficits in autism was the notion that these might relate to a failure to appreciate other people's point of view (Sigman et al, 1986). Bretherton, McNew, and Beeghly-Smith (1981) had also suggested joint attention should be understood as an "implicit theory of mind" - or an implicit awareness of the mental. Baron-Cohen (1989c,d, 1991c) explicitly argued that the joint attention and mind-reading deficits in autism were no coincidence, and proposed that joint attention was a precursor to the development of mind-reading. In that study (Baron-Cohen, 1989c), young children with autism (under 5 years old) were shown to produce one form of the pointing gesture (imperative pointing, or pointing to request) whilst failing to produce another form of pointing (declarative pointing, or pointing to share interest).

This dissociation was interpreted in terms of the declarative form of pointing alone being an indicator of the child monitoring another person's mental state - in this case, the mental state of "interest", or "attention". More recent laboratory studies have confirmed

the lack of spontaneous gaze-monitoring (Leekam, Baron-Cohen, Brown, Perrett, and Milders, in press; Phillips, Baron-Cohen, and Rutter, 1992; Phillips, Gomez, Baron-Cohen, Riviere, and Laa, 1995). Early diagnosis studies have also borne this out (Baron-Cohen, Allen, and Gillberg, 1992; Baron-Cohen, Cox, Baird, Swettenham, Drew, Nightingale, and Charman, 1996). The demonstration of a joint attention deficit in autism, and the role that the superior temporal sulcus in the monkey brain plays in the monitoring of gaze-direction (Perrett et al, 1985) has led to the idea that the superior temporal sulcus may be involved in the development of mind-reading (Baron-Cohen, 1994, 1995; Baron-Cohen and Ring, 1994). Brothers (1990) also reviews evidence suggesting the amygdala contains cells sensitive to gaze and facial expressions of mental states.

Whilst there is now considerable evidence for the theory of mind deficit in autism, it is also clear that this is not the only cognitive deficit in autism. Two others have emerged as important in the last 5 years. First, children with autism fail tests of "executive function". Secondly, they also fail tests of "central coherence". We briefly review each of these next. This is important, because whilst the theory of mind deficits may account for aspects of the social, communicative, and imaginative abnormalities, there are other symptoms (such as their repetitive behaviour, and unusual perception) that are not easily explained by this cognitive deficit.

Central coherence and autism

The second cognitive deficit in autism that we review is in what Frith (1989) calls "central coherence". This is a slippery notion to define. The essence of it is the normal drive to integrate information into a context, or “Gestalt”. Frith argues that the superior ability on the Embedded Figures Test seen in autism (Shah and Frith, 1983), and on an unsegmented version of the Block Design subtest in the WISC and WAIS (Shah and Frith, 1993), arises because of a relative immunity to context effects in autism. Happe (in press) also reports a failure to use context in reading, by people with autism, such that homophones are mispronounced. for example, "there was a tear in her eye" might be misread so as to sound like "there was a tear in her dress". A recent study has shown that children with autism are equally good at judging the identity of familiar faces in photographs, whether they are given the whole face or just part of the face. Non-autistic controls show a `global advantage' on such a test, performing significantly better when given the whole face, not just the parts of the face (Campbell, Baron-Cohen, and Walker, 1995). The central coherence account of autism is attractive in having the potential to explain the non-holistic, piece-meal perceptual style characteristic of autism; and the unusual cognitive profile seen in this condition (including the islets of ability).

A strong version of the central coherence account cannot be correct, however, since children with autism perform in line with their mental age on a range of tasks that would seem to involve integration across context. These include: (i) Transitive inference tests [A>B, and B>C, therefore A?C] (Scott and Baron-Cohen, 1996); (ii) Analogical reasoning tests [A is to B as C is to ?] (Scott and Baron-Cohen, 1996); and (iii)

Counterfactual syllogistic reasoning tests [eg. All cats bark, Rex is a cat, therefore Rex ?] (Scott, Baron-Cohen, and Leslie, 1996).

Finally, Happe (in press) reports that some very high functioning people with autism who pass second-order theory of mind tasks nevertheless fail tasks of central coherence, such as the homophone task, mentioned earlier. This dissociation implies theory of mind and central coherence may be relatively independent processes (Frith and Happe, 1994). Whether both deficits in autism in fact reduce to a more basic deficit is still the subject of controversy. In sum, a weak form of central coherence theory seems likely to be correct, disabling individuals with autism from making full use of context. Whether this can account for islets of ability in autism (and even in Savant Syndrome) remains to be investigated in detail.

Executive function and autism

This is the third and final area of psychological studies for which claims have been made of impairments in autism. Executive function is the postulated mechanism which enables the normal person to shift attention flexibly, inhibit prepotent responses, generate goal-directed behaviour, and solve problems in a planful, strategic way (see Shallice, 1988; Baddeley, 1991). The basic idea, developed by Norman and Shallice (1980), is that without a "central executive", or a "Supervisory Attentional System" as it is also called, actions are controlled by the environment, such that the organism simply responds to cues which elicit behaviour. Without a Supervisory Attentional System, action schemas

or motor programs 'contend' between themselves for execution. This takes place in a system known as the Contention Scheduling System. Shallice's notion is that the Contention Scheduling System is broadly a basal-ganglia function, whilst the Supervisory Attentional System is basically a frontal lobe function. The Supervisory Attentional System allows inhibition of routine actions. The claim that the Supervisory Attentional System is a frontal function derives from the evidence that patients with frontal lobe damage fail tests of this (or executive) function.

Tests of executive function include the following:

(i) The Wisconsin Card Sorting Test (Milner, 1964) in which the subject has to shift card-sorting strategies flexibly.

(ii) The Tower of Hanoi (and its modified version, the Tower of London [Shallice, 1982]), in which the subject has to solve problems by planning before acting.

(iii) The Verbal Fluency Test (or F-A-S test: see Perret, 1974) in which the subject has to generate novel examples of words beginning with a given letter, in a fixed time period.

(iv) The Detour Reaching Test (Diamond, 1991), in which the subject has to inhibit reaching straight for a visible goal, and instead take a detour route to the goal.

Patients with frontal lobe damage fail on these tasks (reviewed in Shallice, 1988), and so do people with autism (Rumsey and Hamberger, 1988; Prior and Hoffman, 1990; Ozonoff, Pennington, and Rogers, 1992; Hughes and Russell, 1993; Hughes, Russell, and Robbins, 1994). This has led to the conclusion that children with autism might have frontal lobe damage. One suggestion arising from this is that they might fail theory of

mind tests listed earlier because they cannot "disengage from the salience of reality" (Hughes and Russell, 1993).

There seems little doubt that in autism there is an executive dysfunction, and that this is likely to be a sign of frontal pathology. However, it is important to note that executive dysfunction occurs in a large number of clinical disorders, and in this respect it is not specific to autism. Thus, the following 8 patient groups all show impairments on different tests of executive function: schizophrenia (Frith, 1992; Elliot, McKenna, Robbins, and Sahakian, 1995; see Elliot and Sahakian, 1995, for a review); treated patients with PKU (Diamond, 1994; Pennington, van Doorninck, McCabe, and McCabe, 1985; Welsh, Pennington, Ozonoff, Rouse, and McCabe, 1990); obsessive-compulsive disorder (Christensen, Kim, Dysken, and Hoover, 1992; Head, Bolton, and Hymas, 1989; Zelinski, Taylor, and Juzwin, 1991); Gilles de la Tourette Syndrome (Bornstein, 1990, 1991; Baron-Cohen and Robertson, 1995); Attention Deficit with Hyperactivity Disorder [Chelune, Ferguson, Koon, and Dickey, 1986; Gorenstein, Mammato, and Sandy, 1989; Grodzinsky and Diamond, 1992; Loge, Staton, and Beatty, 1990]; Parkinson's disease (Downes, Roberts, Sahakian, Evenden, Morris, and Robbins, 1989); frontal lobe syndrome (Owen, Roberts, Polkey, Sahakian, and Robbins, 1991); and children and adults with mental handicap (Borys, Spitz, and Dorans, 1982).

This implies that there is no specific mapping between psychiatric classification and the concept of what Baddeley and Wilson (1988) call a "dysexecutive syndrome" (Baron-Cohen and Moriarty, 1995). Since all of these conditions involve an executive

impairment, and yet do not lead to autism, it follows that, by itself, an impairment in executive function cannot explain autism. Note that examples of patients or disorders which show a double dissociation between executive function and theory of mind would be the strongest test of the independence of these processes[2].

It may be that, as presently construed, the concept of executive function is too broad a level of analysis. The model suggests this has several component processes (generativity, attention-shifting, disengaging, etc.,), and it may be that specificity of deficit will be more apparent at this more fine grain level of analysis. One example of a component process hypothesis is that in autism there is a deficit in "disengaging from the salience of reality". However, this cannot be correct in its strong form. This is because there are a number of studies in which subjects have to do just this, and yet children with autism pass these tests. These include the following:

(i) visual perspective taking (Baron-Cohen, 1989c, 1991b; Hobson, 1984; Tan and Harris, 1991). In these tasks, the child has to infer what someone else can see from their spatial position, even if this is different to what the child currently sees.

(ii) False photograph tests (Leslie and Thaiss, 1992; Leekam and Perner, 1991; Swettenham, Baron-Cohen, Gomez, and Walsh, 1996). In these tasks, the child has to infer where something will be in an out-dated photograph of reality, when they know that reality has been changed such that the object is actually in a new position.

[2] A further confound is that many tests of theory of mind involve some attention shifting, and many tests of executive function involve taking into account one's own mental states, such as one's plans and thoughts.

(iii) False map tests (Leslie and Thaiss, 1992; Leekam and Perner, 1991). These tasks test the same ability as the false photograph task, but using a map instead of a photograph.

(iv) False drawing tests (Charman and Baron-Cohen, 1992). These tasks test the same ability as the false photograph task, but using a drawing instead of a photograph.

(v) False model tests (Charman and Baron-Cohen, 1995). These tasks test the same ability as the false photograph task, but using a model instead of a photograph.

(vi) Intellectual realism tests in drawing (Charman and Baron-Cohen, 1993). In these tasks, the subject is asked to draw an object that is partially occluded - for example, drawing a coffee mug in which the handle is out of view. Children with autism show "intellectual realism" at the same mental age as do children without autism (ie: below a mental age of about 6 yrs old), in that they include the occluded object even though this is out of view. For example, they draw the handle of the coffee mug, even when this is not visible. (It is not until after an MA of about 6 years has been achieved that subjects (with or without autism) show "visual realism", drawing only what they see, not what they know about). This task is relevant in that if children with autism were `prisoners' of reality, they should show precocious visual realism, which they do not.

For these reasons, the executive function hypothesis remains in need of considerable clarification. In addition, it is unlikely that theory of mind is reducible to executive function. Executive function deficits in autism may instead cooccur with theory of mind deficits because of their shared frontal origin in the brain. Despite these provisos, the executive hypothesis of autism is important, since an attraction of the account is its potential to explain the perseverative, repetitive behaviours in this condition, which are not accounted for by the theory of mind hypothesis. Perseveration and repetitive

behaviours are symptomatic of frontal lobe syndrome in which executive dysfunction is also seen (Shallice, 1988). On this view, the two cognitive deficits may be separately responsible for different types of abnormal behaviour.

3. Genetics and autism

Santangelo and Folstein (this volume, chapter 17) provides a thorough review of the genetics of autism, to which the interested reader is referred. However, as a bridge between the psychological evidence reviewed above, and the new model of autism discussed later in the chapter, the key evidence for genetic factors in autism is briefly summarized here.

Autism (and Asperger Syndrome) appear to be strongly heritable. Here is the heritability evidence. First, family studies have shown that first degree relatives of people with autism have a raised risk of autism, compared to population baseline levels (Folstein and Rutter, 1988). For example, whilst estimates of autism in the general population range from 1 in 2500, to 1 in 1000 (Wing and Gould, 1979), the sibling risk rate in families with a child with autism is 3%. This is therefore significantly higher than the population baseline rate. Such family data could imply an environmental or hereditary cause. However, twin studies implicate a genetic aetiology more persuasively. The concordance rate for autism among monozygotic (MZ) twins is as high as 60% whilst the concordance rate among dizygotic (DZ) twins is no higher than

the sib risk rate (Folstein and Rutter, 1988; Bolton and Rutter, 1990). Steffenberg et al (Steffenberg et al, 1989) found an even stronger difference between MZ and DZ concordance rates (91% vs 0%). Whilst such twin studies are not watertight evidence for hereditary factors, they are strongly suggestive of it.

Autism is also predominantly a *male* condition. If one takes the population of autism as a whole (75% of whom not only have autism but also have mental handicap), the sex ratio is 4:1 (m:f) (Rutter, 1978). If one takes just the 'pure' cases of autism (who are also sometimes referred to as having Asperger Syndrome), whose IQs are in the normal range, the sex ratio is even more dramatic: 9:1 (m:f) (Wing, 1981)[3]. Without doubt, then, autism (and Asperger Syndrome) has a strong relationship with being male. Precisely what this relationship is has received little research attention. Sections 4-6 below outline a model to explain the connection between autism and being male.

4. The male brain theory of autism

In the final part of this chapter, a new model of brain development is outlined which may have considerable relevance for our understanding of autism. The model depends on the notion of there being a "male brain", defined psychometrically . The relevant background

[3] Such individuals are either described as having "high functioning autism" or "Asperger Syndrome", after Hans Asperger (1944) who first described such a group of children. There may be a difference between these two conditions (Ozonoff, Rogers, and Pennington, 1991), but for the present purposes we will consider them as one group.

for this notion comes from the long history of research into sex differences in cognition. This is briefly summarized next.

Evidence that males and females differ in cognition

Some of the key findings (for reviews, see Buffery and Gray, 1972; Kimura, 1992; Halpern, 1992; McGee, 1979; Geary, 1996) are that (as a group) women are superior to men on:

(a) language tasks (such as the Verbal Fluency Task - eg: list as many words as you can, beginning with the letter 'L'). Females also show a faster rate of language development, and a lower risk for specific language impairment. (See Hyde and Linn, 1988, on sex differences in language; and Bishop, 1990, on language disorder);

(b) tests of social judgement (Hall, 1977; Halpern, 1992; Argyle and Cooke, 1976);

(c) measures of empathy and cooperation (Hutt, 1972);

(d) rapid identification of matching items (also known as "perceptual speed": Kimura, 1992);

(e) ideational fluency (eg: list as many things as you can that are the same colour: Kimura, 1992);

(f) fine-motor coordination (eg: placing pegs in pegboard holes: Kimura, 1992);

(g) mathematical calculation tests (Kimura, 1992);

(h) pretend play in childhood (Hutt, 1972).

In contrast, men (as a group) are superior to women on:

(i) mathematical reasoning, especially geometry and mathematical word problems (Lummis and Stevenson, 1990; Stevenson et al, 1990; Marshall and Smith, 1987; Steinkamp et al, 1985; Johnson, 1984; Mills, Ablard, and Stumpf, 1993). Benbow and Stanley (1980, 1983) for example reports that at high-level mathematics, the male-female ratio is 13:1;

(ii) the Embedded Figures Task (ie: finding a part within a whole) (Witkin et al, 1971).

(iii) the Mental Rotation Task (ie: imagining how an object will look when it is rotated, or how a sheet of paper will look when it is folded: Masters and Sanders, 1993; Kalichman, 1989);

(iv) some (but not all[4]) spatial skills - mostly Euclidean geometric navigation (Linn and Petersen, 1985; Gilger and Ho, 1989; Law, Pellegrino, and Hunt, 1993; Voyer, Voyer, and Bryden, 1995; Witelson, 1976). Spatial superiority in males is even found in childhood (Kerns and Berenbaum, 1991).

(v) target-directed motor skills, such as guiding or intercepting projectiles - irrespective of the amount of practice (Kimura, 1992; Buffery and Gray, 1972).

Evidence that the male and female brain are determined prenatally

[4] Kimura (1992) for example reports that men are not superior over women on measures of recall of landmarks from a route.

Post conception, the embryo undergoes cell differentiation. In a male embryo, the XY genotype controls the growth of testes, and at approximately 8 weeks gestational age, the testes are not only formed but release bursts of testosterone. Testosterone has frequently been proposed to have a causal effect on subsequent foetal brain development[5], such that by birth, clear sex differences are evident. In rats, the 'masculinizing' effects are confined to a critical or sensitive period of testosterone release, around gestational day 17 and postnatal days 8-10 (Rhees, Shryne, and Gorski, 1990). In humans, at birth, female babies attend for longer to **social** stimuli, such as faces and voices, whilst male babies will attend for longer to non-social, **spatial** stimuli, such as mobiles (Goodenough, 1957; Eibl-Ebelsfeldt, 1989; McGuiness and Pribam, 1979). Levels of prenatal testosterone (as assessed during amniocentesis) predict spatial ability at follow-up at age 7 (Grimshaw, Sitarenios, and Finegan, 1995[6]). One suggestion is that the release of testosterone at this stage of foetal life may determine aspects of brain development, leading to either the male or female brain type. This is defined next.

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[5] Perhaps the best known formulation of the testosterone model is by Geschwind and Galaburda (1987). Their model is far ranging, including predictions that testosterone in fetal life will impact on immune status, cerebral lateralization, handedness, risk for neurodevelopmental disorder, and many other factors. Evidence for it is mixed. See Bryden, McManus, and Bulman-Fleming (1994) for a critical review, and the commentaries on their target article for full debate. For more recent review of the role of both male and female sex hormones in development, see Grimshaw, Sitanerios, and Finegan, (1995), and Fitch and Dennenberg (1996).

[6] In the Grimshaw et al (1995) study, an association was only found between prenatal testosterone and spatial ability in girls, not boys. The authors of that paper interpret this finding in the context of the claim by Gouchie and Kimura (1991) that high levels of prenatal testosterone might have a *curvilinear* relationship with spatial ability.

Defining a male and female brain[7]

Evidence reviewed in the previous section points to the notion that during foetal life, endocrine factors shape the brain as either:

(a) more developed in terms of "folk psychology" and less developed in terms of "folk physics". (Moir and Jessel, 1989, in their popular book, for shorthand call this "the female brain type"); or

(b) or vice versa ("the male brain type").

Folk psychology is broadly "mindreading", and folk physics is broadly understanding physical objects (and this includes mechanical, constructional, mathematical and spatial skills) (Pinker, in press). In our model, we operationally define the male brain type as an individual whose folk physics skills are in advance of his or her social folk pscyhology skills. That is, they show a folk physics>folk psychology discrepancy. This is regardless of one's chromosomal sex. Similarly, we will define the female brain type as an individual whose folk psychology skills are in advance of his or her spatial folk physics skills. That is, they show a folk psychology>folk physics discrepancy. Again, this is regardless of one's sex. Clearly, this suggests that yet other people might have neither the male nor the female brain type, because their folk psychology skills are roughly equal to their folk physics skills. We will call this third possibility the "cognitively balanced brain type". Autism (and Asperger Syndrome), we will argue, are extreme forms of the male

[7] This model should not be used to reinforce traditional occupational and economic inequalities between the sexes. A detailed reading of the model should lead the reader to draw conclusions based on individuals' brain type rather than their sex.

brain type. That is, the folk physics>folk psychology discrepancy is even larger than in the normal male brain type. These types of brain are summarized in Figure 3.

insert Figure 3 here

Neural substrates of the male and female brain

Precisely which structures distinguish these two brain types is still controversial (see Fitch and Dennenberg, 1996, for a review). Kimura (1992) reviews evidence for differences in cerebral lateralization. In particular, she reviews evidence that at birth, in the human male foetus, the right hemisphere cortex is *thicker* than the left: Some reports also show the corpus callosum is larger in females (De la Coste-Utamsing and Holloway, 1982), though reports are conflicting (Wittelson, 1989, 1991; Habib et al, 1991; Dennenberg, Kertesz, and Cowell, 1991). Hines (1990) reviews 13 studies and concludes that in females the corpus callosum is larger, and that this might cause the female superiority in verbal fluency (as a function of better interhemispheric transfer of information).

Finally, there is evidence that aspects of folk physics such as spatial ability are affected by hormonal changes. For example, exposure to androgens prenatally increases spatial performance in human females and females of other species (Resnick et al, 1986; see Hines and Green, 1991; and Halpern, 1992), and castration of the rat decreases spatial

ability (Williams, Barnett, and Meck, 1990). The neuroendocrine evidence may be consistent with the notion of a male or female brain type being a function of the levels of circulating male or female hormones during critical periods of neural development[8].

When considering neurocognitive sex differences, it is important to also consider the large literature on cerebral lateralization. Geschwind and Gallaburda's (1987) well-known model assumes that there is a "standard dominance pattern" (strong left hemisphere dominance for language and handedness, and strong right hemisphere dominance for non-linguistic functions such as visuospatial abilities). Their model predicted that elevated fetal testosterone levels push lateralization away from this standard pattern and toward an "anomalous" pattern. Their model has been criticized on many grounds (see Bryden et al, 1994, with peer commentary on their review), but certainly, important connections have been demonstrated between lateralization, sex, and handedness.

In the normal population, 95% of right handed people have language lateralized to the left hemisphere (as assessed by dichotic listening tasks), and only very rarely to the right (about 5% of cases). In left-handed people, lateralization of language to the right hemisphere is more common (about 25%). Bryden (1988) in his extensive review, concludes that left-handers show reduced language-laterality effects, ie: they show a smaller difference in how quickly they respond to stimuli presented to their right or left

[8] Precisely when these critical periods are is left open here, though these are likely to be during foetal and early infant stages of development.

ear or visual field, relative to right-handers. Thus, he found 82% of right-handers, but only 62% of left handers, show a right-ear advantage in dichotic listening (verbal) tasks. Males have a much higher rate of left-handedness than females (Halpern, 1992). Thus, when Bryden analysed the same data by sex, he found that 81% of males, but only 74% of females, showed a right ear advantage. He concludes that in general, females have a more bilateral organization of cognitive abilities than males. Hines (1990) expresses the same idea differently: the degree of left-hemisphere dominance is greater in males than females.

Regarding the link between lateralization and folk physics, Benbow (1986) reported an elevated incidence of left-handedness in children gifted mathematically. Hassler and Gupta (1993) also found left-handers score higher on a measure of musical talent, and (replicating the earlier work) show reduced right-ear advantage. In addition, Cranberg and Albert (1988) reported an elevated incidence of non-right handedness in high level male chess players. Rosenblatt and Winner (1988) found a very high rate of left-handedness and ambidextrality in children with exceptional drawing ability. Kimura and D'Amico (1989) found that non-right handed science students in university have higher spatial ability than right-handed controls. Sanders, Wilson, and Vandenberg (1982) found, in their family study, that left-handed men score higher than right-handed men on spatial tasks (though left-handed women were worse than right-handed women). Indeed, elevated rates of left-handedness occurs in the those working in the visuo-spatial arts (Mebert and Michel, 1980; Peterson, 1979), in architecture, and in engineering (Petersen

and Lansky, 1974) - all aspects of folk-physics[9]. Direction of handedness appears to be strongly familial (McManus, 1985).

The above review therefore suggests that the 'male brain type' (as defined earlier) is likely to involve complex sex-by-laterality interactions. Halpern (1992) summarizes some of the evidence for this: right-handed males perform better on spatial tests, but worse on verbal tests, relative to left-handed males. Right-handed females perform worse on spatial tests, but better on verbal tests, relative to left-handed females. The above evidence points to the importance of these two variables, but does not yet enable us to draw final conclusions about the brain basis of these different brain types.

5. The extreme male brain theory: Evidence from autism

In this penultimate section of the chapter, some of the evidence from autism that is relevant to the extreme male brain theory of autism is listed. As will be seen, this evidence is largely consistent with the theory, though at least one piece of evidence raises problems for it.

Evidence from autism consistent with the theory

[9] See Martino and Winner, 1995, for a recent study of this area.

(i) Normal males are superior in spatial tasks compared to normal females, and people with autism or Asperger Syndrome are even better on spatial tasks, such as the Embedded Figures Test (Jolliffe and Baron-Cohen, in press).

(ii) There is a strong male bias in the sex ratio of autism or AS.

(iiii) Normal males are slower to develop language than normal females, and children with autism are even more delayed in language development (Rutter, 1978).

(iv) Normal males are slower to develop socially than normal females, and people with autism are even more delayed in social development (O'Riordan, Baron-Cohen, Jones, Stone, and Plaisted, 1996).

(v) Normal females are superior to males on mindreading tasks, and people with autism or AS are severely impaired in mindreading (see Baron-Cohen et al, 1996).

(vi) Parents of children with autism or AS (who can be assumed to share the genotype of their child) also show superior spatial abilities and relative deficits in mindreading (i.e., a marked male brain pattern (Baron-Cohen and Hammer, in press b).

(vii) Normal males have a smaller corpus callosum than normal females, and people with autism or AS have an even smaller one (Egaas, Courchesne, and Saitou, 1994).

(viii) Left handedness is more common among males, and people with autism or AS show an elevated incidence of left-handedness: Fein, Humes, Kaplan, Lucci, and Waterhouse (1984) found an 18% incidence of left-handedness in autism. Satz and colleagues (Satz, Soper, Orsini, Henry, and Zvi, 1985; Soper, Orsini, Henry, Zvi, and Schulman, 1986) found a very similar picture: in their autistic sample, 22% were left handed[10].

[10] It should be noted though that anomalous handedness is also present in children with general

(ix) In the normal population, the male brain is heavier than the female brain, and people with autism have even heavier brains than normal males (Bailey et al, 1994).

(x) In the normal population, more males are found in mathematical/mechanical/spatial occupations than females. Parents of children with autism or AS are disproportionately represented in such occupations (Baron-Cohen, Wheelwright, Bolton, Stott & Goodyer 1996). These occupations all require good folk physics whilst not necessarily requiring equally developed folk psychological skills.

Evidence from autism inconsistent with the theory

Since males are more strongly lateralized than females, people with autism should show strong lateralization. Studies looking at lateralization in autism using dichotic listening tasks and evoked auditory potentials reveal abnormalities, but in the opposite direction to those predicted by the theory. Thus, Prior and Bradshaw (1979) found children with autism show no clear right-ear advantage in dichotic listening tasks; and Dawson, Finley, Phillips, and Galpert (1986) found they did not show the asymmetry of evoked response to auditory speech, unlike normal controls. The most recent relevant study is a SPECT neuroimaging investigation of autism reporting a lack of normal hemispheric asymmetry (Chiron, Leboyer, Leon, Jambaque, Nuttin, and Syrota, 1995). Satz concludes that children with autism are less strongly lateralized, compared to normal children. This is not consistent with the extreme male brain theory of autism. However, this may have

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developmental delay (irrespective of whether they have autism - see Bishop, 1990). It remains to be seen, then whether the anomalous handedness in autism is specific to this condition, or secondary to general developmental delay that is present in two-thirds of children with autism.

arisen because these studies looked at lateralization of language, in children with autism plus significant language delay. It would be interesting for future studies to look for lateralization of spatial abilities, in cases of 'pure' autism or Asperger Syndrome, in order to test the extreme male brain theory further.

6. Conclusions: the continuum of male and female brain types

An important assumption of the model is that all individuals fall on a **continuum** as regards male and female brain types. As stated earlier, we have referred to some individuals as "cognitively balanced", being equally good at folk physics and folk psychology. They show no discrepancy. Other individuals are better at folk physics than they are at folk psychology: this corresponds to the male brain type. People with the male brain type might show this discrepancy just marginally (the normal male brain type), or just more than this (a touch of Asperger Syndrome), or more markedly still (frank Asperger Syndrome), or in an extreme way (classic autism). Such a model encompasses Lorna Wing's (1988) important notion of an autistic continuum, blurring into the normal population.[11] The work reviewed here constitutes preliminary but suggestive evidence for the notion of male and female brain types, defined in psychometric ways. The above psychological studies are also consistent with the claim that autism (and Asperger

[11] It is tempting to surmise that children with Williams Syndrome might have an extreme form of the *female* brain type, (Karmiloff-Smith et al, 1995).

Syndrome) is an extreme form of the male brain. Currently, the neurobiological basis of such a model is still unclear.

Figure 1: Which one is thinking? Reproduced from Baron-Cohen and Cross (1992), with permission.

Figure 2: Which candy does Charlie want? Reproduced from Baron-Cohen et al (1995), with permission.

Figure 3: Summary of the brain types.

BRAIN TYPE	**COGNITIVE PROFILE**
The Cognitively Balanced Brain:	**Folk Physics = Folk Psychology**
The Normal Female Brain:	**Folk Physics < Folk Psychology**
The Normal Male Brain:	**Folk Physics > Folk Psychology**
Asperger Syndrome	**Folk Physics >> Folk Psychology**
Autism	**Folk Physics >>>Folk Psychology**

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