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The cognitive neuroscience of autism: Implications for the evolution of the male brain

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Abstract

An evolutionary approach to the cognitive neuroscience of autism generated the “theory of mind” (ToM) hypothesis. Most of its predictions of selective deficits in this domain have been confirmed. Current work is attempting to isolate the brain basis of the ToM deficits in autism. The ToM hypothesis has considerable explanatory power in relation to the “triad symptoms” of autism (social, communication, and imagination abnormalities), but has little relevance to the non-triad symptoms (attention to detail, islets of ability, and obsessions). An evolutionary hypothesis to account for these in terms of superior folk physics is discussed.

Autism: An Introduction

This chapter illustrates how an evolutionary approach has generated a key hypothesis in the cognitive neuroscience of autism. It then re-introduces the evolutionary approach to generate a new hypothesis for those features that are not yet well understood. But first, what is autism?

Autism is considered to be the most severe of the childhood neuropsychiatric conditions. It is diagnosed on the basis of abnormal development of social behaviour, communication, and imagination, often in the presence of marked obsessional, repetitive, or ritualistic behaviour (APA, 1994). In an attempt to understand the so-called ‘triad’ impairments in autism (social, communication, and imagination abnormalities), my colleagues and I adopted an evolutionary framework. We asked the following questions: (1) Might mechanisms for understanding and interacting with the social world be specialized adaptations, universal both to hominids today and ancestrally? (2) If so, what might such ancient cognitive mechanisms be? (3) Could such mechanisms become selectively impaired as a result of a genetic factor? (4) Might autism be such a case of genetic caused impairment to specialized social-cognitive mechanisms?

To answer these questions we turned to the philosophical, primatological, and human developmental literatures as a pointer to the prerequisites for hominid social interaction. Three key texts led to the same clear conclusion: human social life is characterized by the necessary adoption of the ‘intentional stance’ (Dennett, 1987), that is, understanding action by ascribing mental states (beliefs, desires, intentions, etc.) to agents; humans appear to do this universally and yet chimpanzees (or other non-human primates) only do this in a very

limited way, if at all ((Premack & Woodruff, 1978); and in the normal case even a 4 year old child can pass a shockingly complex test of social intelligence or mental state ascription, namely, a test of understanding *false beliefs* (Wimmer & Perner, 1983). This fundamental and apparently uniquely human ability has been called a theory of mind (ToM). My colleagues and I therefore set out to test the ToM hypothesis of autism - that such children might for genetic reasons have a selective deficit in this most essential of neurocognitive mechanisms (Baron-Cohen, Leslie, and Frith, 1985). The relevant evidence is summarized next.

1. The ToM hypothesis: experimental evidence

First order ToM tests

First-order tests involve inferring what one person thinks, knows, intends, desires, etc. There is a good deal of experimental evidence to review here, so this section is necessarily concise. For clarity, different cognitive tests used are underlined.

The majority of children with autism are at chance on tests of the mental-physical distinction (Baron-Cohen, 1989a). They have also been shown to have an appropriate understanding of the functions of the brain, but have a poor understanding of the functions of the mind (*ibid*). That is, they do not spontaneously mention the mind's mental function (in thinking, dreaming, wishing, deceiving, etc.,). They also fail to make the appearance-reality distinction (*ibid*). They fail a range of first-order false belief tasks, that is, distinguishing between their own current belief, and that of someone else (Baron-Cohen, Leslie & Frith, 1985; Baron-Cohen, Leslie & Frith, 1986; Leekam & Perner, 1991; Perner, Frith, Leslie & Leekam, 1989;

Reed & Peterson, 1990; Swettenham, Baron-Cohen, Gomez & Walsh, 1996) (Swettenham, 1996). They also fail tests assessing if they understand the principle that "seeing leads to knowing" (Baron-Cohen & Goodhart, 1994; Leslie & 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. (See Figure 1). 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.

insert Figure 1 here

Children with autism are at chance on a test of recognizing mental state words (like "think", "know", and "imagine") in a wordlist (Baron-Cohen et al., 1994). Nor do they produce the same range of mental state words in their spontaneous speech (Baron-Cohen et al., 1986; Tager-Flusberg, 1992). They are also impaired in the production of spontaneous pretend play (Baron-Cohen, 1987; Lewis & Boucher, 1988; Wing & Gould, 1979) (Ungerer et al, 1981). Pretend play is relevant here because it is thought to involve understanding the mental state of pretending. Whilst they can understand simple causes of emotion (such as situations and desires), the majority of children with autism have difficulty on tests of understanding more complex causes of emotion (such as beliefs) (Baron-Cohen, 1991a; Baron-Cohen, Spitz & Cross, 1993).

They also fail tests of recognizing the eye-region of the face as indicating when a person is thinking and what a person might want (Baron-Cohen, Campbell, Karmiloff-Smith, Grant & Walker, 1995; Baron-Cohen & Cross, 1992). Children and adults without autism use gaze to

infer both of these mental states. They fail a test of being able to monitor their own intentions (Phillips, Baron-Cohen & Rutter, in press). That is, they are poor at distinguishing if they "meant" to do something, or if they did something accidentally.

They also have problems on tests of deception (Baron-Cohen, 1992; Sodian & Frith, 1992; Yirmiya, Solomonica-Levi & Shulman, 1996), a result that would be expected if one was unaware that people's beliefs can differ and therefore can be manipulated. They also fail tests of understanding metaphor, sarcasm, and irony - these all being intentionally non-literal-statements (Happe, 1993). Indeed, they fail to produce most aspects of pragmatics in their speech (Baron-Cohen, 1988; Tager-Flusberg, 1993), and fail to recognize violations of pragmatic rules, such as the Gricean Maxims of conversational cooperation (Surian, Baron-Cohen & Van der Lely, 1996). Since many pragmatic rules involve tailoring one's speech to what the listener needs to know, or might be interested in, this can be seen as intrinsically linked to a theory of mind. Most children with autism also have difficulties in tests of imagination (Scott & Baron-Cohen, 1996), for example, producing drawings of impossible or totally fictional entities such as two-headed men. This could reflect a difficulty in thinking about their own mental state of imagination, or reflect difficulties in flexible behaviour (Leevers & Harris, 1998). Supporting an imagination deficit, they also do not show the normal facilitation effect of imagination on logical reasoning (Scott, Baron-Cohen & Leslie, 1996), unlike normally developing children. Performance on ToM tasks by children with autism has been found to correlate with real-life social skills, as measured by a modified version of the Vineland Adaptive Behaviour Scale (Frith, Happe & Siddons, 1994).

Second-order, adolescent, and adult ToM tests

A small minority of children or adults with autism pass first-order false belief tests. 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 (Sullivan, Zaitchik & Tager-Flusberg, 1994), and yet these tests are failed by individuals with autism with a mental age above this level. This suggests there can be a *specific developmental delay* in theory of mind at a number of different points. 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 (Bowler, 1992; Happe, 1993; Ozonoff, Pennington & Rogers, 1991). Those who can pass second-order tests however may have difficulties in understanding stories in which characters are motivated by complex mental states such as bluff and double bluff (Happe, 1994). Equally, such able subjects have difficulties in decoding complex mental states from the expression in the eye-region of the face (Baron-Cohen, Jolliffe, Mortimore & Robertson, 1997; Baron-Cohen, Wheelwright & Jolliffe, 1997). Examples of the Eyes Test are shown in Figure 2. Again, this suggests that the mindreading deficit may only be detectable in such high-level, older subjects using sensitive, age-appropriate tests.

insert Figure 2 here

Similarly, children with Asperger Syndrome (AS) may pass first- and second-order ToM tests, but fail to detect “faux pas” in speech (Baron-Cohen, O’Riordan, Jones, Stone & Plaistead, submitted). Detecting faux pas, of course, is all about detecting who said the ‘wrong’ thing - that is, detecting who said something that the listener should not *know* about.

Finally, parents of children with AS, at least one of whom presumably carries the genes for AS, also show difficulties in attributing mental states when just the eye-region of the face is available (Baron-Cohen & Hammer, 1997).

For this reason, autism has been conceptualized as involving “mindblindness” *to varying degrees* (Baron-Cohen, 1990; Baron-Cohen, 1995).

Universality

To test if the ToM deficit is universal in autism, more complex, subtle or age-appropriate ToM tests may have to be used. When these have been used, ToM deficits do appear to be universal in autism and Asperger Syndrome (Baron-Cohen et al., 1997). A different approach to addressing the universality question is to adopt Uta Frith’s suggestion (Frith, 1989) that there should be no cases of someone with an autism spectrum condition who passes a ToM test at the right age or mental age. (Thus, even a child with AS, if tested at 4 years old, should fail a false belief test, even if they can pass it when re-tested at 6 years, for example. Such a pattern would fit the specific developmental delay hypothesis (Baron-Cohen, 1989b).

Validity

Some authors (Waterhouse, Fein, & Modahl, 1996) have claimed that no correlation between ToM deficits and social skills is found. In fact, when the *relevant* social skills are assessed (namely, social skills involving mentalizing) strong correlations are found (Frith et al., 1994). A further question surrounding validity is that since ToM skills only appear in the preschool

years, and yet autism develops by the end of the first year of life, this may mean that ToM deficits cannot be a core of the condition (Rutter and Bailey, 1993). This argument is wrong simply because it ignores the work on infancy precursors to ToM, reviewed next.

Infancy precursors to ToM

(Leslie, 1987) proposed that in the normal case, a theory of mind is already evident in the capacity for pretence; and that in the case of children with autism, an early manifestation of the theory of mind deficit lay in their inability to pretend. Why? In his view, in order to understand that someone else might pretend "This banana is real", the child (according to Leslie) would need to be able to represent the agent's mental attitude towards the proposition. One idea, then, is that theory of mind is first evident from about 18-24 months of age, in the normal toddler's emerging pretend play.

However, there is some evidence that theory of mind might have *even earlier* developmental origins. There are severe deficits in joint attention skills in children with autism (Sigman, Mundy, Ungerer & Sherman, 1986). Joint attention skills are 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. Joint attention behaviours are normally fully developed by about 14 months of age (Butterworth, 1991; Scaife & Bruner, 1975), so their absence in autism signifies a very early-occurring deficit.

What is the evidence for lack of joint attention in autism? One study (Baron-Cohen, 1989d) found that young children with autism (under 5 years old) produced 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 (Charman, Swettenham, Baron-Cohen, Cox & Baird, 1997; Leekam, Baron-Cohen, Brown, Perrett & Milders, 1997; Phillips, Baron-Cohen & Rutter, 1992; Phillips, Gomez, Baron-Cohen, Riviere & Laa, 1996). Absence of joint attention, in combination with an absence of pretend play, at 18 months of age, is a very strong predictor of autism, both in a high risk study of siblings who are undiagnosed (Baron-Cohen, Allen & Gillberg, 1992), and in a random population study (Baron-Cohen et al., 1996). In the latter study, 16,000 children at 18 months of age were screened by their health visitors for these behaviours, using the CHAT (Checklist for Autism in Toddlers). Just 12 children out of the total population lacked joint attention and pretend play, of whom 11 were discovered to have an autism spectrum disorder¹. The joint attention deficit in autism is received a great deal of research attention and is now one of the best validated cognitive deficits in the condition (Mundy, 1995; Mundy & Crowson, 1997; Mundy, Sigman & Kasari, 1990; Mundy, Sigman & Kasari, 1994).

The finding of both joint attention and theory of mind deficits in autism may be no coincidence, if joint attention is a precursor to the development of a theory of mind. This is

¹ In the paper reporting this result the rate was 10 out 12 affected children. Subsequent follow-up of these cases reveals the rate is now 11 out 12.

plausible because joint attention involves attending to another person's mental state, of attention (Baron-Cohen, 1989c; Baron-Cohen, 1989d; Baron-Cohen, 1991b).

Does the ToM deficit imply modularity?

One possibility is that there may be a particular part of the brain which in the normal case is responsible for understanding mental states, and which is specifically impaired in autism. This may be modular, as in Leslie's proposal of an innate theory of mind mechanism (Leslie, 1991; Leslie & Roth, 1993; Leslie, 1987). Leslie (see also Chapter x, this volume) suggests the function of such a mechanism is to represent information in a data-structure of the following form: [**Agent-Attitude-"Proposition"**] - e.g.: [Fred-thinks-"the safe is behind the Picasso"]. Such a proposal is sufficient to allow representation of the full range of mental states, in the Attitude slot. Leslie's computational analysis has been widely accepted, though the innate modularity claim is more controversial (Carruthers, 1996; Russell, 1997b). Future work needs to focus on testing this claim against alternatives. For example, (Baron-Cohen, 1994) suggests lower level social-perception mechanisms (an Eye-Direction Detector, or EDD, an Intentionality Detector, or ID, and a Shared Attention Mechanism, or SAM) provide input to ToM, so that what is innate may be an attentional bias to relevant social information (faces, actions, eyes). Russell (Russell, 1997a) argues that the ToM deficit can be produced by non-modular, executive dysfunction.

Note that the modularity thesis of ToM has been tested in a series of single cases of neurological patients: (1) A patient with severe SLI (specific language impairment) but with intact ToM demonstrates the potential independence of language and ToM (Van der Lely,

1997). (2) A patient with impaired executive function (EF) but intact ToM demonstrates the potential independence of EF and ToM. Some patients with Tourette Syndrome meet this criteria (Baron-Cohen, Robertson & Moriarty, 1994). (3) A patient with intact EF but impaired ToM also suggests the independence of these two cognitive domains (Baron-Cohen, Stone, Wheelwright, and Rutherford (1998). (4) A person with very high IQ but ToM impairments demonstrates the existence of pure deficits in social intelligence, independent of general intelligence (ibid). Finally, (5) patients with low IQ but intact ToM prove the same point. Patients with Williams Syndrome fulfil this criterion (Tager-Flusberg, Boshart & Baron-Cohen, in press)².

It is entirely possible that the ToM deficit in autism occurs for genetic reasons, since autism appears to be strongly heritable (Bailey et al., 1995; Bolton & Rutter, 1990; Le Couteur et al., 1996). The idea that the development of theory of mind is under genetic/biological 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 theory of mind at roughly the same ages (Avis & Harris, 1991). Which brain areas might be involved in ToM?

The neural basis of ToM

Quite which part of the brain might subservise ToM is not yet clear, though candidate regions include the following: (i) right orbito-frontal cortex, which is active when subjects are thinking about mental state terms during functional imaging using SPECT (Baron-Cohen et

² Caution is needed to distinguish the innate modularity thesis of ToM from an acquired modularity thesis of ToM (Karmiloff-Smith, 1992?).

al., 1994); (ii) left medial frontal cortex, which is active when subjects are drawing inferences about thoughts whilst being PET scanned (Fletcher et al., 1995; Goel, Grafman, Sadato & Hallett, 1995). The first PET study to look at adults with autism/Asperger Syndrome during a ToM task shows that such patients do not show the same patterns of neural activation when thinking about other minds (Happe et al, in press). (iii) Other candidate regions include the amygdala (Baron-Cohen and Ring, 1994). Ongoing studies suggest adult patients with acquired amygdala lesions have difficulties with advanced (or adult level) theory of mind tasks (Andy Young, personal communication), and a recent fMRI study of ToM using the Eyes Task (described earlier) found that whilst normal controls used areas of the fronto-temporal cortex and the amygdala, high functioning adults with autism or AS did not activate the amygdala during this task (Baron-Cohen et al., submitted). (iv) Finally, 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 a theory of mind (Baron-Cohen & Ring, 1994).

Summary

The ToM deficit in autism is present to varying degrees in individuals with autism or AS of different ages. It can be seen at least as early as 18 months of age, in the form of an absence of joint attention and pretend play; and that this is not only of interest to basic science, but is of clinical significance, in improving early diagnosis. Future research will need to explore the first year of life, using prospective studies of high risk populations, to better understand the ontogenesis of both ToM and autism. Note that the ToM hypothesis is successful in

explaining the ‘triad’ symptoms of autism (social, communication, and imagination deficits), but has almost no relevance to the ‘non-triad’ symptoms (attention to detail, strong obsessions, islets of ability). What might explain these?

2. Re-introducing the evolutionary framework to explain the non-triad symptoms of autism

Existing attempts to account for the non-triad symptoms of autism essentially paint these symptoms in terms of deficits (central coherence theory is said to be “weak” (Frith, 1989), or executive control is said to be “dysfunctional” (Russell, 1997)). In this section I rethink the non-triad features in such a way as to keep evolutionary considerations central, and to emphasize these features as reflecting superior abilities, not deficits.

The evolutionary view of cognition is in terms of domain-specificity. A number of theorists have suggested that rather than adopting the traditional ways of carving-up of cognition, one should instead study cognitive development in terms of a small set of ‘core domains of cognition’, motivated by an evolutionary framework (Carey, 1985; Gelman & Hirschfield, 1994; Pinker, 1997; Sperber, Premack & Premack, 1995; Wellman & Gelman, in press). By this is meant domains of knowledge that develop very early in human infancy, with a universal pattern of ontogenesis, and an initial state that is likely to be in part innate. The universalist approach here immediately underlines that these aspects of cognition may be fundamental and result from evolutionary selection pressures at least as old as early hominids, if not older.

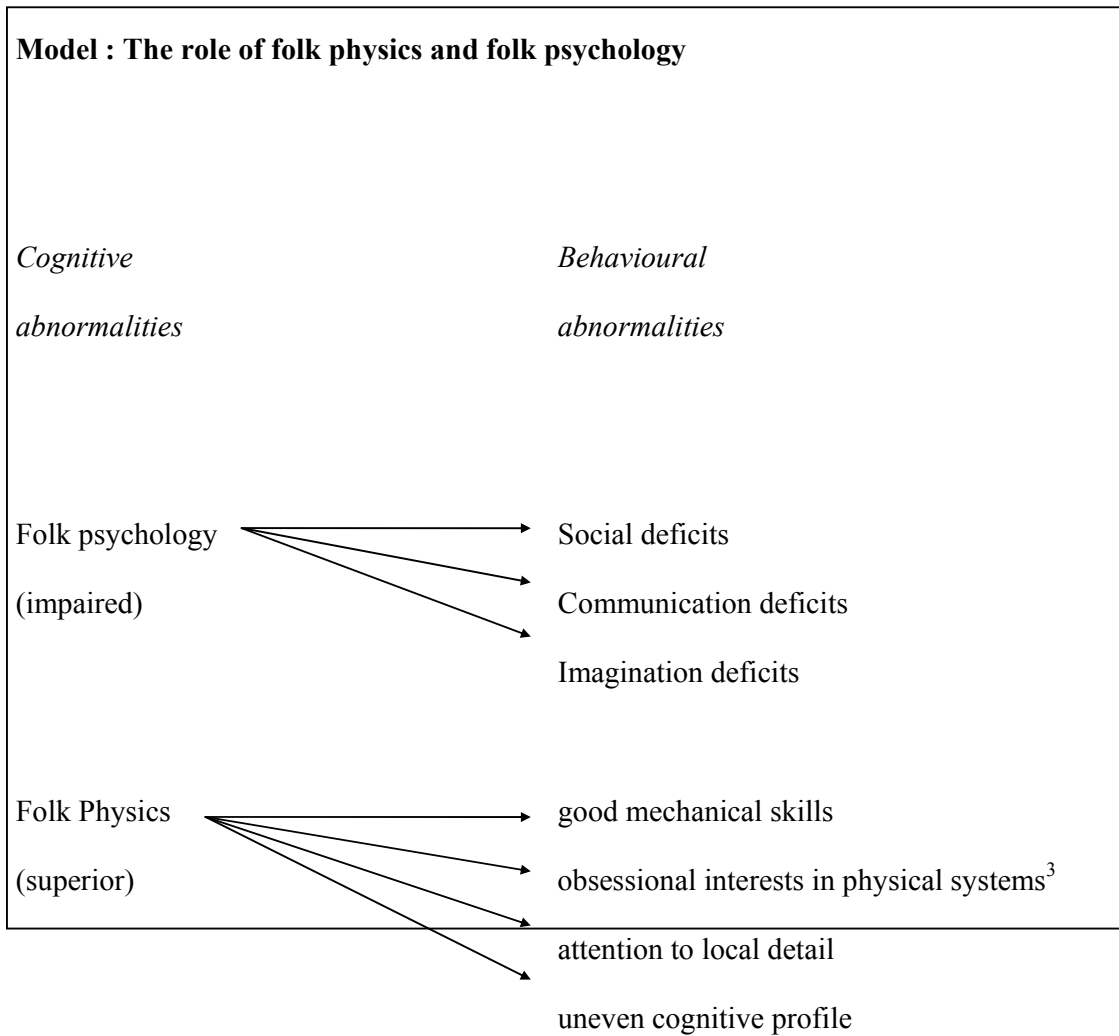
A consensus among these researchers is that two such core domains of cognition are **folk psychology** and **folk physics**. The term ‘folk’ here is intended to emphasize that this knowledge develops without any formal teaching. Some authors also use the term ‘intuitive psychology’ and ‘intuitive physics’. Folk psychology is our everyday ability to understand and predict an agent’s behaviour in terms of intentional states like goals, beliefs, and desires. It is what we considered earlier, under the heading of theory of mind. Folk physics is our everyday ability to understand and predict the behaviour of inanimate objects in terms of principles relating to size, weight, motion, physical causality, etc.

As the evidence in Section 1 (above) indicated, an impaired folk psychology characterizing autism now seems beyond any doubt. But it is plausible that in contrast there might be an intact or even *superior* folk physics in autism. In the discussion of this below, we will include the following within folk physics: understanding of objects, machines, physical-causality, and physical systems. How well are predictions from this view of autism confirmed?

There is a range of relevant evidence to consider. First, children with autism certainly understand physical causality (Baron-Cohen et al., 1986). They also seem to understand machines such as cameras (Leekam & Perner, 1991; Leslie & Thaiss, 1992), possibly better than mental age matched controls. In addition, many of their obsessional interests centre on machines and physical systems (Baron-Cohen, 1997).

If impaired folk psychology together with superior folk physics were a good characterization of the cognitive phenotype of autism, then this might also constitute the “broader phenotype” of those 1st degree relatives of children with autism who carry the relevant genes, but express

them to a lesser degree. Recent studies bear this out. First, parents of children with AS show impairments on an adult test of folk psychology (the Reading the Mind in the Eyes Test), together with a superiority on the Embedded Figures Test (Baron-Cohen et al., 1997). Quite what the Embedded Figures Test is a test of is unclear, though at one level it measures how one analyses wholes into their parts, and this may be an aspect of folk physics. Secondly, fathers of children with autism, as well as grandfathers, are over-represented in occupations such as engineering, whilst being under-represented in occupations such as social work (Baron-Cohen, Wheelwright, Stott, Bolton & Goodyer, 1997). Engineering is a clear example of an occupation that requires good folk physics, whilst social work is a clear example of an occupation that requires good folk psychology. Similarly, students in the fields of maths/physics/or engineering are more likely to have a relative with autism than are students in the humanities (Baron-Cohen et al., 1997). These family studies are all consistent with the idea that the autistic spectrum phenotype at the cognitive level involves this *combination* of superior folk physics with impaired folk psychology. This is summarized in the model below relating cognition to symptoms:



³ Physical systems include: classification, spatial information [maps, etc], repeating patterns [eg, astronomy], numbers [eg, calenders], machines, and spinning objects.

3. The extreme male brain theory of autism: a third evolutionary hypothesis

There is one more interesting evolutionary hypothesis to consider, and this picks up an old but undeveloped idea from (Asperger, 1944) who wrote “The autistic personality is an extreme variant of male intelligence....In the autistic individual the male pattern is exaggerated to the extreme” (Frith, 1991). The extreme male brain (EMB) theory is explored in detail elsewhere (Baron-Cohen and Hammer, 1997). One clue to the EMB theory is that in autism the sex ratio is 4 males to 1 female (Rutter, 1978). The 4:1 sex ratio is true of autism when one includes individuals with this condition at all points on the IQ scale. If one restricts it to individuals with autism with an IQ in the normal range (referred to as either ‘high-functioning autism’ or ‘Asperger Syndrome’ (AS)⁴), the sex ratio is even more dramatically biased against males: (Wing, 1988) estimates it as 9:1 (male to female), and (Ehlers et al., 1997) recently documented a ratio of 40:0 (m:f). Since high-functioning autism or Asperger Syndrome may be considered to be “pure autism” (i.e., not confounded by the effects of mental retardation) (Frith, 1989), it may be that these sex ratios are more accurate estimates of how the sexes are differentially affected by this condition.

A second clue to the EMB theory is that superior folk physics skills (seen in autism and AS) is also generally associated with being male: the sex ratio in fields like engineering, maths and physics, remains heavily biased towards males. Whether this reflects biological or social factors has not been established. A third clue is that three recent studies have found sex differences in the rate of development of folk psychology/ToM skills (Baron-Cohen et al.,

⁴ High functioning autism is the term used when an individual of normal IQ meets criteria for autism, including a delay in the onset of speech; Asperger Syndrome (AS) is the term used when an individual of normal IQ meets criteria for autism, but with no delay in the onset of speech (ICD-10, 1994).

1997; Baron-Cohen et al., submitted; Happe, 1995) in all cases showing a female superiority. The implication is that the male brain⁵ involves this combination of impaired folk psychology and superior folk physics to a mild degree, whilst in autism spectrum disorders this combination occurs to a more marked extent. In the final figure below, one can see this notion graphically depicted: all individuals are situated somewhere on the proposed continuum of folk psychology (FΨ) - folk physics (FPhys), some individuals showing no discrepancy between these domains (depicted as zero on the graph), others showing differing degrees of discrepancy in either direction. Normal males tend to be to the right of zero, and normal females to the left of zero. People with autism spectrum conditions tend to be even further to the right of normal males.

insert Figure 3 here

Conclusions

The evolutionary framework used here might help explain why a condition like autism persists in the gene pool: the very same genes that lead an individual to have a child with autism can lead to superior functioning in the domain of folk physics. (Pinker, 1997) argues that the evolution of the human mind should be considered in terms of its evolved adaptedness to the environment. In his view, the brain needed to be able to maximize the survival of its host body in response to at least two broad challenges: the physical

⁵ Note that the theory therefore defines what constitutes the male brain. This cognitive profile does not have to be true of every biological male of course. There will be many individuals who are biologically male who do not have the male brain, as so defined. Equally, there will be some individuals who are biologically female who have the male brain, so defined.

environment and the social environment. The specialized cognitive domains of folk physics and folk psychology can be seen as adaptations to each of these. One possibility is that a cognitive profile of superior folk physics alongside of impaired folk psychology could arise for genetic reasons, in that some brains are better adapted to understanding the social environment whilst other brains are better adapted to understanding the physical environment. Quite why this relationship should occur between these two domains is not clear: Is this neural compensation by one domain over another? Are these two independent domains that can simply dissociate from one another to a greater or lesser extent? And how are such domains constructed in the first place? These must be questions for the future.

Figure Legends

Figure 1: The ‘seeing leads to knowing’ test

Figure 2: The ‘reading the mind in the eyes’ test

Figure 3: A model of the distribution of discrepancies between Folk Psychology (F) and Folk Physics (FPhys).

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