86 The Cognitive Neuroscience of Autism: Evolutionary Approaches

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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. Currently attempts are being made 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 nontriad symptoms (attention to detail, islets of ability, and obsessions). An evolutionary hypothesis to account for these in terms of superior folk physics is discussed.

This chapter illustrates how an evolutionary approach has generated a key hypothesis in the cognitive neuroscience of autism. It then reintroduces 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 behavior, communication, and imagination, often in the presence of marked obsessional, repetitive, or ritualistic behavior (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 current hominids 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 pointers to the prerequisites for hominid social interaction. Three key texts led to the same clear conclusions: 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, whilst chimpanzees (or other nonhuman primates) only do this in a very limited way, if at all (Premack and 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 and 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.

The theory of mind hypothesis: Experimental evidence

FIRST-ORDER THEORY OF MIND TESTS *First-order* tests involve inferring what one person thinks, knows, intends, or desires. There is a good deal of experimental evidence to review, so this section is necessarily concise. For clarity, different cognitive tests used are in italics.

Most children with autism are at chance on tests of the *mental-physical distinction* (Baron-Cohen, 1989a). They also have been shown to 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 do not spontaneously mention the mind's mental function (in thinking, dreaming, wishing, deceiving, etc.). They also fail to make the *appearance-reality distinction* (Baron-Cohen, 1989a). They fail a range of *first-order false belief* tasks, that is, they fail to distinguish between their own current belief and that of someone else (Baron-Cohen, Leslie, and Frith, 1985; Baron-Cohen, Leslie, and Frith, 1986; Leekam and Perner, 1991; Perner et al., 1989; Reed and Peterson, 1990; Swettenham et al., 1996) They also fail tests assessing whether they understand the principle that "*seeing leads*

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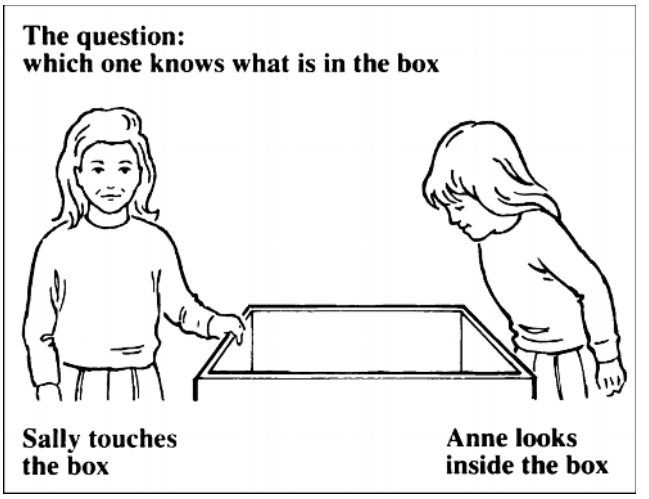

FIGURE 86.1 The "seeing leads to knowing" test.

to knowing" (Baron-Cohen and Goodhart, 1994; Leslie and Frith, 1988). For example, when presented with two dolls, one of which touches a box and the other of which *looks inside* the box, then asked "Which one *knows* what's inside the box?" they are at chance in their response (figure 86.1). In contrast, normal children of 3 to 4 years of age correctly judge that it is the one who looked who knows what's in the box.

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). They also do not produce the same range of mental state words in their spontaneous speech (Baron-Cohen, Leslie, and Frith, 1986; Tager-Flusberg, 1992). They also are impaired in the production of spontaneous *pretend play* (Baron-Cohen, 1987; Lewis and Boucher, 1988; Wing and Gould, 1979; Ungerer et al., 1981). Pretend play is relevant here because it is thought to involve understanding the mental state of pretending. Although they can understand simple causes of emotion (such as situations and desires), most children with autism have difficulty on tests of understanding more *complex causes of emotion* (such as beliefs) (Baron-Cohen, 1991a; Baron-Cohen, Spitz, and 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 et al., 1995; Baron-Cohen and 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, and Rutter, 1998). That is, they are poor at distinguishing whether they "meant" to do something or whether they did something accidentally.

They also have problems on tests of *deception* (Baron-Cohen, 1992; Sodian and Frith, 1992; Yirmiya, Solomonica-Levi, and 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 nonliteral 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, and Van der Lely, 1996). Because 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 and 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 behavior (Leevers and Harris, 1998). Supporting an imagination deficit, they also do not show the normal facilitation effect of imagination on logical reasoning (Scott, Baron-Cohen, and Leslie, in press), 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, Happ, and Siddons, 1994).

SECOND-ORDER, ADOLESCENT, AND ADULT THEORY OF MIND 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 believes another character thinks. Such second-order reasoning usually is understood by normal children of 5 to 6 years of age (Sullivan, Zaitchik, and Tager-Flusberg, 1994), and yet individuals with autism with a mental age above this level may fail these tests. This suggests that 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 intelligence quotient [IQ] and language level), and who are old enough, may pass even second-order tests (Bowler, 1992; Happe, 1993; Ozonoff, Pennington, and 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,

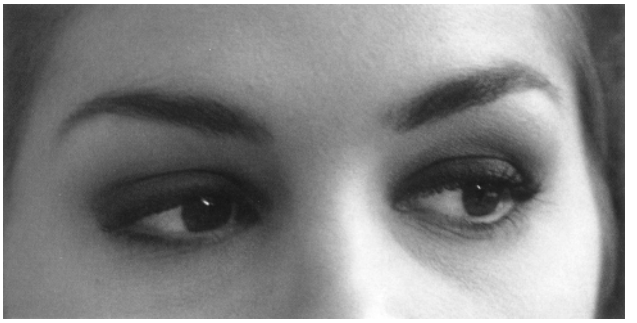

FIGURE 86.2 An item from the "reading the mind in the eyes" test. Is this face concerned or unconcerned?

Mortimore, and Robertson, 1997; Baron-Cohen, Wheelwright, and Jolliffe, 1997). Examples of the *Eyes Test* are shown in figure 86.2. Again, this suggests that the mind-reading deficit may only be detectable in such high-level, older subjects using sensitive, age-appropriate tests.

Similarly, children with Asperger's syndrome (AS) may pass first- and second-order ToM tests but fail to detect *faux pas* in speech (Baron-Cohen et al., in press). 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 and Hammer, 1997).

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

UNIVERSALITY To test whether 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 AS (Baron-Cohen, Joliffe, 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 chronological or mental age. (Thus, even a child with AS, if tested at 4 years old, should fail a false-belief test, even if he or she can pass it when retested at 6 years, for example. Such a pattern would fit the specific developmental delay hypothesis [Baron-Cohen, 1989b].)

VALIDITY Some authors (Waterhouse, Fein, and 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, Happe, and Siddons, 1994). A further point surrounding validity is that because 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, which is reviewed next.

INFANCY PRECURSORS TO THEORY OF MIND Leslie (1987) proposed that normally, a theory of mind already is evident in the capacity for pretense, and in children with autism, an early manifestation of the ToM deficit lies in their inability to pretend. Why? In his view, to understand that someone else might *pretend* that "this banana is real," the child (according to Leslie) would need to be able to represent the agent's *mental attitude* toward the proposition. One idea, then, is that ToM is first evident from approximately 18 to 24 months of age in the normal toddler's emerging pretend play.

However, there is some evidence that ToM might have *even earlier* developmental origins. There are severe deficits in *joint attention* skills in children with autism (Sigman et al., 1986). Joint attention skills are those behaviors produced by the child that 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 behaviors include the pointing gesture, gaze-monitoring, and showing gestures, most of which are absent in most children with autism. Joint attention behaviors normally are fully developed by about 14 months of age (Butterworth, 1991; Scaife and 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 (younger than 5 years old) with autism produced one form of the pointing gesture (imperative pointing, or pointing to request) while 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 et al., 1997; Leekam et al., 1997; Phillips, Baron-Cohen, and Rutter, 1992; Phillips et al., 1996). Absence of joint attention at 18 months of age, in combination with an absence of pretend play, is a very strong predic-

tor of autism, both in a high-risk study of siblings who are undiagnosed (Baron-Cohen, Allen, and 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 behaviors using the Checklist for Autism in Toddlers (CHAT). Just 12 children of the total population lacked joint attention and pretend play, 11 of whom were discovered to have an autism spectrum disorder.[1] The joint attention deficit in autism has received a great deal of research attention and currently is one of the best validated cognitive deficits in the condition (Mundy, 1995; Mundy and Crowson, 1997; Mundy, Sigman, and Kasari, 1990, 1994).

The finding of both joint attention and ToM deficits in autism may not be a coincidence if joint attention is a *precursor* to the development of a ToM. This is plausible because joint attention involves attending to another person's mental state of attention (Baron-Cohen, 1989c, 1989d, 1991b).

DOES THE THEORY OF MIND DEFICIT IMPLY MODULARITY? One possibility is that there may be a particular part of the brain that normally is responsible for understanding mental states that is specifically impaired in autism. This may be modular, as in Leslie's proposal of an innate theory of mind mechanism (Leslie, 1987, 1991; Leslie and Roth, 1993). Leslie (see also chapter 85) suggests that the function of such a mechanism is to represent information in a data-structure of the following form: **[Agent-Attitude-"Proposition"]**–for example: [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, although the innate modularity claim is more controversial (Carruthers, 1996; Russell, 1997b). Future work needs to focus on testing this claim against alternatives. For example, lower level social-perception mechanisms (an Eye-Direction Detector [EDD], an Intentionality Detector [ID], and a Shared Attention Mechanism [SAM]) may provide input to ToM, so that what is innate may be an attentional bias to relevant social information (faces, actions, eyes) (Baron-Cohen, 1994). Russell (1997a) argues that the ToM deficit can be produced by nonmodular, 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 specific language impairment (SLI) 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's syndrome meet these criteria (Baron-Cohen, Robertson, and Moriarty, 1994). (3) A patient with intact EF but impaired ToM also suggests the independence of these two cognitive domains (Baron-Cohen, et al., in press). (4) A person with a very high IQ but ToM impairments demonstrates the existence of pure deficits in social intelligence, independent of general intelligence (Baron-Cohen et al., in press). (5) Patients with low IQs but intact ToM prove the same point. Patients with Williams syndrome fulfill this criterion (Tager-Flusberg, Boshart, and Baron-Cohen, 1998).[2]

It is entirely possible that the ToM deficit in autism occurs for genetic reasons because autism appears to be strongly heritable (Bailey et al., 1995; Bolton and Rutter, 1990; Le Couteur et al., 1996). The idea that the development of ToM normally is under genetic/biological control 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 and Harris, 1991). Which brain areas might be involved in ToM?

THE NEURAL BASIS OF THEORY OF MIND Exactly which part of the brain might subserve ToM is not yet clear, although candidate regions include the following: the right *orbitofrontal cortex*, which is active when subjects are thinking about mental state terms during functional imaging using single photon emission computed tomography (SPECT) (Baron-Cohen et al., 1994); and the *left medial frontal cortex*, which is active when subjects are drawing inferences about thoughts while undergoing positron-emission tomography (PET) scan (Fletcher et al., 1995; Goel et al., 1995). The first PET study to look at adults with autism/AS 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., 1996); other candidate regions include the *amygdala* (Baron-Cohen and Ring, 1994). Ongoing studies suggest that adult patients with acquired amygdala lesions have difficulties with advanced (or adult-level) ToM tasks (Andy Young, personal communication, 1999), and a recent functional magnetic resonance imaging (fMRI) study of ToM using the Eyes Task (described earlier) found that although normal controls used areas of the frontotemporal cortex and the amygdala, high-functioning adults with autism or AS did not activate the amygdala during this task (Baron-Cohen et al., 1999). 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 ToM (Baron-Cohen and Ring, 1994).

SUMMARY The ToM deficit in autism is present to varying degrees in individuals of different ages with autism or AS. 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 this is not only of interest to basic science but is of clinical significance in improving early diagnosis. Future research needs to explore the first year of life, using prospective studies of high-risk populations, to better understand the ontogenesis of both ToM and autism. The ToM hypothesis is successful in explaining the "triad" symptoms of autism (social, communication, and imagination deficits) but has almost no relevance to the "nontriad" symptoms (attention to detail, strong obsessions, islets of ability). What might explain these?

Reintroducing the evolutionary framework to explain the nontriad symptoms of autism

Existing attempts to account for the nontriad symptoms of autism essentially paint these symptoms in terms of deficits (central coherence is said to be "weak" [Frith, 1989], or executive control is said to be "dysfunctional" [Russell, 1997a]). In this section, I rethink the nontriad features 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 and Hirschfield, 1994; Pinker, 1997; Sperber et al., 1995; Wellman and Gelman, 1998). This refers to 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" is intended to emphasize that this knowledge develops without any formal teaching. Some authors also use the terms "intuitive psychology" and "intuitive physics." Folk psychology is our everyday ability to understand and predict an agent's behavior in terms of intentional states such as 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 behavior of inanimate objects in terms of principles relating to physical causality.

As indicated in the section on the experimental evidence of the ToM hypothesis, an impaired folk psychology characterizing autism seems beyond any doubt. But it is plausible that there might also be an intact or even *superior* folk physics in autism. In the following discussion of this, we 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 and Perner, 1991; Leslie and Thaiss, 1992), possibly better than mental age-matched controls. In addition, many of their obsessional interests center on machines and physical systems (Baron-Cohen, 1997; Baron-Cohen and Wheelwright, in press).

If *impaired folk psychology together with superior folk physics* were a good characterization of the cognitive phenotype of autism, then this also might constitute the "broader phenotype" of those first-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 and Hammer, 1997a). Exactly what the Embedded Figures Test is a test of is unclear, although at one level it measures how one analyzes wholes into their parts, and this may be a prerequisite of folk physics. Second, fathers of children with autism, as well as grandfathers, are over-represented in occupations such as engineering relative to occupations such as social work (Baron-Cohen, Wheelwright, et al., 1997). Engineering is a clear example of an occupation that requires good folk physics, whereas social work is a clear example of an occupation that requires good folk psychology. Similarly, students in the fields of math/physics/engineering are more likely to have a relative with autism than are students in the humanities (Baron-Cohen et al., 1998). These family studies all are 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 in figure 86.3 relating cognition to symptoms.

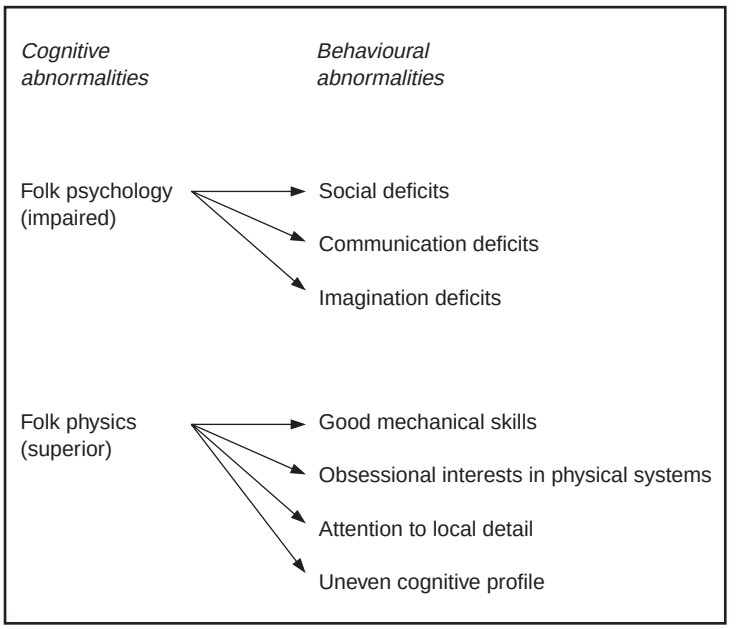

FIGURE 86.3 A model of the relationship between folk psychology and folk physics.

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, 1997b). 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 AS[3]), the sex ratio is even more dramatically biased against males: Wing (1988) estimates it as 9:1 (male:female), and Ehlers and associates (1997) recently documented a ratio of 40:0 (m:f). Because high-functioning autism or AS 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 affected differentially by this condition.

A second clue to the EMB theory is that superior folk physics skills (seen in autism and AS) also generally are associated with being male: the sex ratio in fields like engineering, math, and physics remains heavily biased toward 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, Joliffe, et al., 1997; Baron-Cohen et al., in press; Happe, 1995) in all cases showing a female superiority. The implication is that if the male brain[4] involves this combination of impaired folk psychology and superior folk physics to a mild degree, in autism spectrum disorders this combination occurs to a more marked extent.

Conclusions

The evolutionary framework used 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 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 along with impaired folk psychology could arise for genetic reasons because some brains are better adapted to understanding the social en-

vironment whereas other brains are better adapted to understanding the physical environment. Exactly 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 dissociate from one another to a greater or lesser extent? How are such domains constructed in the first place? These are questions for the future.

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NOTES

1. In the paper reporting this result, the rate was 10 of 12 affected children. Subsequent follow-up of these cases reveals the rate is now 11 of 12.
2. Caution is needed to distinguish the innate modularity thesis of ToM from an acquired modularity thesis of ToM.
3. 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).
4. 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 are many individuals who are biologically male who do not have the male brain, as so defined. Equally, there are some individuals who are biologically female who have the male brain, so defined.

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