Neurodevelopmental Disorders

edited by Helen Tager-Flusberg

A Bradford Book
The MIT Press
Cambridge, Massachusetts
London, England

17 The Extreme Male-Brain Theory of Autism

Simon Baron-Cohen

Autism is regarded widely 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 [Diagnostic and Statistical Manual of Mental Disorders (fourth edition) (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. Their isolation is a tragedy for their families, who work tirelessly to attempt to engage with and socialize their child, mostly with very limited results.

This chapter begins with a summary of psychological findings from studies of autism. A brief review of genetic evidence appears next, as a bridge into the next section, wherein a recent notion is introduced: the "male brain." Evidence for biologically based psychological gender differences is presented, and the "male brain" is defined. Finally, this notion is related to autism, summarizing the author's new theory (Baron-Cohen & Hammer, 1997a) 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.

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 "mind-reading." This is defined as the ability to attribute mental states to oneself and others and to

make sense of and predict behavior 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 given a short story with the simplest of plots. The story involves one character's 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*, because it 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 normal children'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 and 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 et al., 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 necessarily would be different from the child's mental state. In contrast, a control group of children with Down syndrome and moderate degrees of mental retardation passed this test as easily as did 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 impaired specifically 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. Many reasons might account 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 et al., 1993) and for that reason are summarized only briefly here.

Results from Studies of Mind-Reading in Autistic Children[1] The majority of children with autism 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 autistic children respond at chance levels.

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). 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 & Estes, 1986).

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 et al., 1986). In contrast, autistic children tend to refer to just one aspect of the object (e.g., saying "It looks like an apple, and it really is an apple").

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

They also fail tests assessing their understanding of the principle that "seeing leads to knowing" (Leslie & Frith, 1988; Baron-Cohen & Goodhart, 1994). 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 to 4 years of age correctly judge that the one who looked knows what is in the box.

Whereas normally developing children are rather good at picking out mental-state words (e.g., *think, know,* and *imagine*) in a word list that contains both mental-state and non-mental-state words, most autistic children are at chance (Baron-Cohen et al., 1994). In contrast, they have no difficulty in picking out words describing physical states.

Also, most children with autism do not *produce* the same range of mental-state words in their spontaneous speech (Baron-Cohen et al., 1986; Tager-Flusberg, 1992). Thus, from perhaps 18 to 36 months of age, normally developing children spontaneously use such words as *think, know, pretend, imagine, wish, hope,* and the like and use such terms appropriately (Wellman, 1990). In contrast, such words occur less frequently and often are even absent in the spontaneous speech of children with autism.

Such children also are impaired in the production of spontaneous pretend play (Wing et al., 1977; Baron-Cohen, 1987; Lewis & Boucher, 1988). Pretend play is relevant here simply because it involves understanding the mental

state of *pretending*. The normal child of even age 2 effortlessly distinguishes between someone's acting veridically and "just pretending" (Leslie, 1987). Sometimes mommy actually is eating (putting a real spoon with real food into her mouth), whereas at other times, mommy is just pretending to eat (holding a pen to her lips and making funny slurping noises between her smiles).

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

Though they can understand simple causes of emotion (such as reactions to *physical* situations), the majority of children with autism have difficulty in understanding more *mentalistic* causes of emotion (such as beliefs; Baron-Cohen, 1991a; Baron-Cohen et al., 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. However, 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.

Most children with autism also fail to recognize the eye region of the face as indicating that a person is *thinking* and what a person might *want* (Baron-Cohen & Cross, 1992; Baron-Cohen et al., 1995). Children and adults without autism use gaze to infer both of these mental states.

For example, when presented with pairs of photos such as those in figure 17.1, normal 3- to 4-year-olds easily identify the person looking upward and away as the one who is thinking. Children with autism are less sure of this. When shown a display such as the one in figure 17.2, normal 4-year-olds identify the candy that Charlie is looking at as the one he wants. Autistic children mostly fail to intuit that gaze can be an indicator of what a person might want.

In addition, many children with autism fail to make the accidental-intentional distinction (Phillips, 1993); they are poor at distinguishing whether someone "meant" to do something or whether something simply happened accidentally.

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

Most children with autism also have disproportionate difficulty on tests of understanding metaphor, sarcasm, and irony, all statements that cannot be decoded literally but are meaningful only by reference to the speaker's *inten-*

Figure 17.1 Which one is thinking? (Reproduced from Baron-Cohen & Cross, 1992, with permission.)

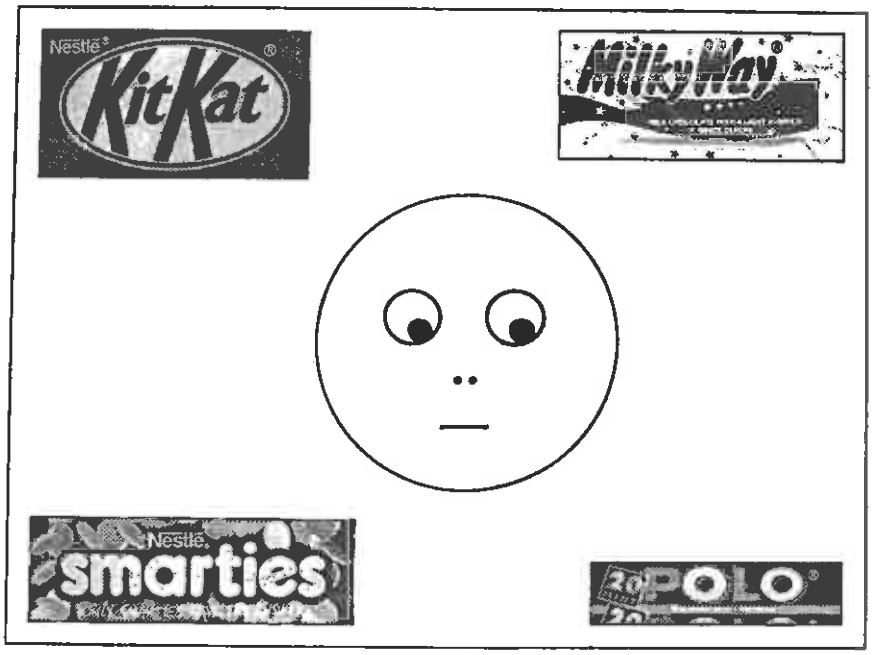

Figure 17.2 Which candy does Charlie want? (Reproduced from Baron-Cohen et al., 1995, with permission.)

tion (Happe, 1994). An example would be understanding "The drinks are on the house," a statement that one adult with autism (and above-average IQ) could interpret only 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.

Indeed, most children with autism fail to produce most aspects of pragmatics in their speech (see review 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 et al., 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. As 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 linked intrinsically to a sensitivity of another person's mental states.

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

Autistic children also are unimpaired in logical reasoning (i.e., about the physical world), even though they have difficulty in psychological reasoning (i.e., about the mental world; Scott & Baron-Cohen, 1996).

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

Importantly, 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): tests of understanding what one character thinks another character thinks. Such second-order reasoning usually is understood by normal children of age 5 to 6, and yet these tests are failed by autistic individuals with a mental age above this level.

Therefore, we can interpret these results in terms of a specific developmental delay in mind-reading at a number of different points (Baron-Cohen, 1991b). Some autistic individuals who are very high-functioning (in terms of IQ and language level) and usually are adults may pass even second-order tests (Ozonoff et al., 1991; Bowler, 1992; 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 et al., 1997).

Thus, being able as an adult to pass a test designed for a 6-year-old may mask persisting mind-reading deficits by ceiling effects.

In summary, an impairment seems apparent 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, because being able to reflect on one's own mental states (and those of others) would appear to be essential in all these domains. This deficit has been found to correlate with real-life social skills, as measured by a modified version of the Vineland Adaptive Behavior Scale (Frith et al., 1994).

The Brain Basis of Theory of Mind One possibility arising from these studies is the presence of a particular part of the brain that in the normal case is responsible for our mind-reading ability and specifically is impaired in autism. If this view is correct, the assumption is that this may be for genetic reasons, as autism appears to be strongly heritable (see chapter 18). 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 & Harris, 1991).

Exactly which parts of the brain might be involved in this is not yet clear, though candidate regions include 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 left medial frontal cortex, which is active when subjects are drawing inferences about thoughts while being scanned with positron emission tomography (Fletcher et al., 1995; Goel et al., 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 & Ring, 1994).

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

However, some evidence suggests that this ability might have even earlier developmental origins. Soon after the first demonstrations of mindblindness in autism, Sigman et al. (1986) also reported severe deficits in *joint attention* in children with autism. Joint attention refers to those behaviors produced by the child and involving 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.

This discovery was important because joint attention behaviors are normally well developed by age 14 months (Scaife & Bruner, 1975; Butterworth, 1991), so their absence in autism signifies a very early occurring deficit. This was important also because the traditional mind-reading skills already discussed are mostly those one would expect to see in a 3- to 4-year old normal child. Deficits in these areas cannot, therefore, be the developmentally earliest signs of autism, because 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 et al. (1981) also had suggested that 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 age 5) were shown to produce 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 (Leekam et al., 1997; Phillips et al., 1992; Phillips et al., 1995). Early diagnosis studies also have borne this out (Baron-Cohen et al., 1992; Baron-Cohen et al., 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 & Ring, 1994). Brothers (1990) also reviewed evidence suggesting that the amygdala contains cells sensitive to gaze and facial expressions of mental states.

Though now considerable evidence supports the theory of mind deficit in autism, clearly 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." This is important, because though the theory of mind deficits may account for aspects of the social, communicative, and imaginative abnormalities, other symptoms (such as their repetitive behavior and unusual perception) are not explained easily 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." Defining this notion is slippery. The essence of it is the normal drive to integrate information into a context, or "Gestalt." Frith argued that the superior ability on the embedded figures test seen in autism (Shah & Frith, 1983) and on an unsegmented version of the block design subtest in the Wechsler Intelligence Scale for Children and the Wechsler Adult Intelligence Scale (Shah & Frith, 1993) arises because of a relative immunity to context effects in autism. Happe (1997) also reported a failure by autistic people to use context in reading, such that homophones are mispronounced. For example, "There was a *tear* in her eye" might be misread as "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 entire face or just part of the face. Nonautistic controls show a "global advantage" on such a test, performing significantly better when given the entire face, not just the parts of the face (Campbell et al., 1995). The central coherence account of autism is attractive in having the potential to explain the nonholistic, piecemeal 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, because 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 (1) transitive inference tests (A > B, and B > C, therefore A?C; Scott & Baron-Cohen, 1996); (2) analogical reasoning tests (A is to B as C is to?; Scott & Baron-Cohen, 1996); and (3) counterfactual syllogistic reasoning tests (e.g., all cats bark; Rex is a cat; therefore Rex?; Scott et al., 1995).

Finally, Happe (1997) reported 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 that theory of mind and central coherence may be relatively independent processes (Frith & 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

Regarding this third and final area of psychological studies, claims have been made of impairments in autism. Executive function is the postulated mechanism that enables a normal person to shift attention flexibly, inhibit prepotent responses, generate goal-directed behavior, 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 an organism simply responds to cues that elicit behavior. Without a supervisory attentional system, action schemas or motor programs "contend" among themselves for execution. This contention 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, whereas the supervisory attentional system is basically a frontal lobe function. The supervisory attentional system allows inhibition of routine actions. The claim that this 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 (1) the Wisconsin Card Sorting Test (Milner, 1964), in which the subject has to shift card-sorting strategies flexibly; (2) 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; (3) the verbal fluency test (or FAS 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; and (4) the detour-reaching test (Diamond, 1991), in which the subject has to inhibit reaching directly for a visible goal and instead has to take a detour route to the goal.

Patients with frontal lobe damage fail on these tasks (reviewed in Shallice, 1988), as do people with autism (Rumsey & Hamberger, 1988; Prior & Hoffman, 1990; Ozonoff et al., 1991; Hughes & Russell, 1993; Hughes et al., 1994). This has led to the conclusion that children with autism might have frontal lobe damage. One suggestion arising from this finding is that they might fail theory of mind tests listed earlier because they cannot "disengage from the salience of reality" (Hughes & Russell, 1993).

Little doubt remains that in autism an executive dysfunction is likely to be a sign of frontal pathology. However, an important note is that executive dysfunction occurs in a large number of clinical disorders, and in this respect, it is not specific to autism. Thus, all the following eight patient groups show impairments on different tests of executive function:

- Schizophrenia (Frith, 1992; Elliot et al., 1995)
- Treated patients with phenylketonuria (Pennington et al., 1985; Welsh et al., 1990; Diamond, 1994)
- Obsessive-compulsive disorder (Head et al., 1989; Zelinski et al., 1991; Christensen et al., 1992)

- Gilles de la Tourette syndrome (Bornstein, 1990, 1991; Baron-Cohen & Robertson, 1995)
- Attention deficit with hyperactivity disorder (Chelune et al., 1986; Gorenstein et al., 1989; Loge et al., 1990; Grodzinsky & Diamond, 1992)
- Parkinson disease (Downes et al., 1989)
- Frontal lobe syndrome (Owen et al., 1991)
- Children and adults with mental handicap (Borys et al., 1982)

Studies demonstrating executive dysfunction across different populations suggest the absence of any specific mapping between psychiatric classification and the concept of what Baddeley and Wilson (1988) call a *dysexecutive syndrome* (Baron-Cohen & Moriarty, 1995). That all these conditions involve an executive impairment and yet do not lead to autism strongly suggests that, by itself, an impairment in executive function cannot explain autism. Note that examples of patients or disorders that show a double dissociation between executive function and theory of mind would be the strongest test of the independence of these processes.[2]

Possibly, as presently construed, the concept of executive function is too broad a level of analysis. The model suggests that this has several component processes (generativity, attention shifting, disengaging, etc.), and possibly 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 resides a deficit in "disengaging from the salience of reality." However, this cannot be correct in its strong form because, in a number of studies, subjects have to perform just such a task, and yet children with autism *pass* such tests as the following:

- Visual perspective taking (Hobson, 1984; Baron-Cohen, 1989c, 1991b; Tan & Harris, 1991): In these tasks, a child has to infer what others can see from their spatial position, even if that differs from what the child currently **sees.**
- False photograph tests (Leekam & Perner, 1991; Leslie & Thaiss, 1992; Swettenham et al., 1996): In these tasks, a child has to infer where something will be in an outdated photograph of reality, when they know that reality has been changed such that the object is actually in a new position.
- False map tests (Leekam & Perner, 1991; Leslie & Thaiss, 1992): These tasks test the same ability as that in the false photograph task but use a map rather than a photograph.
- False drawing tests (Charman & Baron-Cohen, 1992): These tasks also test the same ability as that in the false photograph task but use a drawing rather than a photograph.
- False model tests (Charman & Baron-Cohen, 1995): These tasks test the same ability as that in the false photograph task but use a model rather than a photograph.

• Intellectual realism tests in drawing (Charman & Baron-Cohen, 1993): In these tasks, a subject is asked to draw an object that is partially occluded (e.g., drawing a coffee mug in which the handle is out of view).

Children with autism show "intellectual realism" at the same mental age as that in nonautistic children (i.e., below a mental age of perhaps 6 years) in that they include the occluded object even though it is out of view. For example, they draw the handle of the coffee mug, even when it is not visible. (It is not until after a mental age MA of nearly 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, theory of mind probably is not reducible to executive function. Executive function deficits in autism may instead co-occur with theory of mind deficits because of their shared frontal origin in the brain. Despite these provisos, the executive hypothesis of autism is important, as an attraction of the account is its potential to explain the perseverative, repetitive behaviors in this condition, behaviors not explained by the theory of mind hypothesis. Perseveration and repetitive behaviors are symptomatic of frontal lobe syndrome, in which executive dysfunction also is seen (Shallice, 1988). On this view, the two cognitive deficits may be separately responsible for different types of abnormal behavior.

GENETICS AND AUTISM

Chapter 18 provides a thorough review of the genetics of autism, to which the interested reader is referred. However, as a bridge between the psychological evidence already reviewed and the new model of autism discussed later, the key evidence for genetic factors in autism is briefly summarized.

Autism and Asperger syndrome appear to be strongly heritable according to 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 & Rutter, 1988). For example, though estimates of autism in the general population range from 1 in 2500 to 1 in 1000 (Wing et al., 1977), the sibling risk rate in families with an autistic child is 3%. Therefore, this is significantly higher than the population baseline rate. Such family data could imply an environmental or hereditary cause. However, twin studies implicate a genetic etiology more persuasively. The concordance rate for autism among monozygotic (MZ) twins is as high as 60%, whereas the concordance rate among dizygotic (DZ) twins is no higher than the sibling risk rate (Folstein & Rutter, 1988; Bolton & Rutter, 1990). Steffenberg et al. (1989) found an even stronger difference between MZ and

DZ concordance rates (91% versus 0%). Though such twin studies are not watertight evidence for hereditary factors, they strongly suggest it.

Autism also is 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 male-female ratio is 4:1; (Rutter, 1978). If one takes just the "pure" cases of autism (also sometimes designated as having Asperger syndrome) whose IQs are in the normal range, the male-female ratio is even more dramatic: 9:1 (Wing, 1981).[3] Without doubt, then, autism and Asperger syndrome bear a strong relationship to being male. Precisely what this relationship is has received little research attention. The following discussion outlines a model to explain the connection between autism and being male.

THE MALE-BRAIN THEORY OF AUTISM

Our new model of brain development may have considerable relevance for our understanding of autism (Baron-Cohen & Hammer, 1997a). The model depends on the possibility of a "male brain" (defined psychometrically). The relevant background for this notion comes from the long history of research into gender differences in cognition.

Differences in Male and Female Cognition

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

- Language tasks (e.g., the verbal fluency task calling for listing as many words as possible, beginning with the letter *L*. Female subjects also show a faster rate of language development and a lower risk for specific language impairment. (See Hyde and Linn, 1988, regarding gender differences in language, and Bishop, 1990, regarding language disorder.)
- Tests of social judgment (Argyle & Cooke, 1976; Hall, 1977; Halpern, 1992)
- Measures of empathy and cooperation (Hutt, 1972)
- Rapid identification of matching items (also known as *perceptual speed*; Kimura, 1992)
- Ideational fluency (e.g., list as many things as possible of the same color; Kimura, 1992)
- Fine-motor coordination (e.g., placing pegs in pegboard holes; Kimura, 1992)
- Mathematical calculation tests (Kimura, 1992)
- Pretend play in childhood (Hutt, 1972)

In contrast, men (as a group) are superior to women in the following areas:

- Mathematical reasoning, especially geometry and mathematical word problems (Johnson, 1984; Mills et al., 1993; Steinkamp et al., 1985; Marshall & Smith, 1987; Lummis & Stevenson, 1990; Stevenson et al., 1990). Benbow and Stanley (1980, 1983), for example, reported that at high-level mathematics, the male-female ratio is 13:1.
- The embedded figures task (i.e., finding a part within a whole; Witkin et al., 1971).
- The mental rotation task (i.e., imagining how an object will look when it is rotated or how a sheet of paper will look when it is folded; Masters et al., Sanders, 1993; Kalichman, 1989).
- Some (but not all)[4] spatial skills: mostly Euclidean geometrical navigation (Witelson, 1976; Linn & Petersen, 1985; Gilger & Ho, 1989; Law et al., 1993; Voyer et al., 1995). Spatial superiority in male individuals is found even in childhood (Kerns & Berenbaum, 1991).
- Target-directed motor skills, such as guiding or intercepting projectiles, irrespective of the amount of practice (Buffery & Gray, 1972; Kimura, 1992).

Prenatal Determination of Male and Female Brain

After conception, the embryo undergoes cell differentiation. In a male embryo, the XY genotype controls the growth of testes; at approximately 8 weeks' gestational age, the testes are not only formed but release bursts of testosterone. Testosterone frequently has been proposed to have a causal effect on subsequent fetal brain development,[5] such that by birth, clear gender 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 to 10 (Rhees et al., 1990). At birth, human female babies attend for longer to social stimuli, such as faces and voices, whereas male babies will attend for longer to nonsocial, spatial stimuli, such as mobiles (Goodenough, 1957; McGuiness & Pribam, 1979; Eibl-Ebelsfeldt, 1989). Levels of prenatal testosterone (as assessed during amniocentesis) predict spatial ability at follow-up at age 7 (Grimshaw et al., 1995).[6] One suggestion is that the release of testosterone at this stage of fetal life may determine aspects of brain development, leading to either the male or female brain type.

Defining the Male and Female Brain

Aforementioned evidence points to the notion that during fetal life, endocrine factors shape the brain as either "the male-brain type" or vice versa, 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*).

Table 17.1 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

Folk psychology is broadly "mind reading," and folk physics is broadly understanding physical objects (and this includes mechanical, constructional, mathematical, and spatial skills; Pinker, 1998). In our model, we operationally define the male-brain type as individuals whose folk physics skills are in advance of their social folk psychology skills. That is, they show a folk physics > folk psychology discrepancy. This is regardless of one's chromosomal gender. Similarly, we define the female-brain type as individuals whose folk psychology skills are in advance of their spatial, folk physics skills[7] (i.e., they show a folk psychology > folk physics discrepancy). Again, this is regardless of one's gender. 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 call this third possibility the *cognitively balanced brain type*. Autism and Asperger syndrome arguably are extreme forms of the male-brain type; the folk physics > folk psychology discrepancy is even larger than in the normal male-brain type. These types of brain are summarized in table 17.1.

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) reviewed evidence for differences in cerebral lateralization. In particular, she reviewed evidence that at birth, the right hemisphere cortex in the human male fetus is thicker than the left is. Some reports also show that the corpus callosum is larger in female subjects (De Lacoste-Utamsing & Holloway, 1982), though reports are conflicting (Wittelson, 1989, 1991; Dennenberg et al., 1991; Habib et al., 1991). Hines (1990) reviewed 13 studies and concluded that the female 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, evidence suggests that aspects of folk physics, such as spatial ability, are affected by hormonal changes. For example, exposure to androgens prenatally increases spatial performance in female humans and females of other species (Resnick et al., 1986; Hines & Green, 1991; Halpern, 1992), and castration of the rat decreases spatial ability (Williams et al., 1990). The

neuroendocrine evidence may be consistent with the notion that a male or female brain type is a function of the levels of circulating male or female hormones during critical periods of neural development.[8]

Any consideration of neurocognitive gender differences should also investigate the voluminous literature on cerebral lateralization. Geschwind and Galaburda's (1987) well-known model assumes the presence of a "standard dominance pattern" (strong left hemisphere dominance for language and handedness and strong right hemisphere dominance for such nonlinguistic functions 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, for peer commentary regarding their review), but certainly, important connections have been demonstrated between lateralization, gender, 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 (approximately 5% of cases). In left-handed people, lateralization of language to the right hemisphere is more common (some 25%). In his extensive review, Bryden (1988) concluded that left-handers show reduced language-laterality effects (i.e., they show a smaller difference in how quickly they respond to stimuli presented to their right or left 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. Male subjects have a rate of left-handedness much higher than that in female subjects (Halpern, 1992). Thus, when Bryden analyzed the same data by gender, he found that 81% of male (but only 74% of female) subjects showed a right-ear advantage. He concluded that, in general, female subjects have a more bilateral organization of cognitive abilities than do male subjects. Hines (1990) expressed the same idea differently: The degree of left-hemisphere dominance is greater in male than in female subjects.

Regarding the link between lateralization and folk physics, Benbow (1986) reported an elevated incidence of that left-handedness in children gifted mathematically. Hassler and Gupta (1993) also found that 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 ambidexterity in children with exceptional drawing ability. Kimura and D'Amico (1989) found that non-right-handed university science students have higher spatial ability than that in right-handed controls. Sanders et al., (1982) found in their family study that left-handed men score higher than do right-handed men on spatial tasks (though left-handed women scored lower than did right-handed women). Indeed, elevated rates of left-handedness occur in those working in the visuospatial arts (Peterson, 1979;

Mebert & Michel, 1980), in architecture, and in engineering (Petersen & Lansky, 1974)—all aspects of folk physics.[9] Direction of handedness appears to be strongly familial (McManus, 1985).

The foregoing review therefore suggests that the male-brain type (as defined earlier) is likely to involve complex gender-by-laterality interactions. Halpern (1992) summarized some of the evidence for this. Right-handed male subjects perform better on spatial tests but worse on verbal tests, relative to left-handed male subjects. Right-handed female subjects perform worse on spatial tests but better on verbal tests, relative to left-handed female subjects. This 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.

EVIDENCE FROM AUTISM FOR THE EXTREME MALE-BRAIN THEORY

Some of the evidence from autism 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.

Consistency with the Male-Brain Theory

Certain evidence from autism is consistent with the male-brain theory. For example, normal male subjects are superior in spatial tasks as compared to normal female subjects, and people with autism or Asperger syndrome are even better on spatial tasks, such as the embedded figures test (Jolliffe & Baron-Cohen, 1997). A strong male bias exists in the gender ratio of autism or Asperger syndrome. Also, normal male subjects are slower to develop language than are normal female subjects, and autistic children are even more delayed in language development (Rutter, 1978).

Normal male individuals develop socially more slowly than do normal female individuals, and autistic people are even more delayed in social development (Baron-Cohen et al., in press). Additionally, normal female subjects are superior to male subjects in mind-reading tasks, and people with autism or Asperger syndrome are severely impaired in mind-reading (Baron-Cohen et al., 1996).

Parents of children with autism or Asperger syndrome (who can be assumed to share the genotype of their child) also show superior spatial abilities and relative deficits in mind-reading (i.e., a marked male-brain pattern; Baron-Cohen & Hammer, 1997b). Normal male persons have a smaller corpus callosum than that in normal female persons, and in people with autism or Asperger syndrome they are even smaller (Egaas et al., 1994).

Left-handedness is more common among male subjects, and people with autism or Asperger syndrome show an elevated incidence of left-handedness. Fein et al. (1984) found an 18% incidence of left-handedness in autism. Satz

et al. (1985) and Soper et al. (1986) found a very similar picture: In their autistic sample, 22% were left-handed.[10]

In the normal population, the male brain is heavier than is the female brain, and people with autism have brains even heavier than those in normal male subjects. (Piren et al., 1995). In the normal population, more male persons are found in mathematical, mechanical, and spatial occupations than are female persons. Parents of children with autism or Asperger syndrome are disproportionately represented in such occupations (Baron-Cohen et al., 1997). All these occupations require good folk physics, though not necessarily requiring equally developed folk psychological skills.

Inconsistencies with the Male-Brain Theory

Some evidence from autism raises inconsistencies with the male-brain theory. Because males are more strongly lateralized than are 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 direction opposite to those predicted by the theory. Thus, Prior and Bradshaw (1979) found that children with autism show no clear right-ear advantage in dichotic listening tasks, and Dawson et al. (1986) found that autistic children did not show the asymmetry of evoked response to auditory speech, unlike that of normal controls. The most recent relevant study is a SPECT neuroimaging investigation of autism reporting a lack of normal hemispheric asymmetry (Chiron et al., 1995). Satz et al. (1985) concluded that children with autism are less strongly lateralized, as compared to normal children. This is not consistent with the extreme male-brain theory of autism. However, this may have arisen because these studies looked at lateralization of language in children with autism plus significant language delay. Interesting future studies might look for lateralization of spatial abilities, in cases of "pure" autism or Asperger syndrome to test the extreme male-brain theory further.

CONCLUSIONS: THE CONTINUUM OF MALE- AND FEMALE-BRAIN TYPES

An important assumption of the aforementioned 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 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); slightly more than this (a touch of Asperger syndrome); or markedly still (frank Asperger syndrome); or in an extreme way (classic autism). Such a model encompasses 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 psychometrical ways. The foregoing psychological studies are consistent also with the claim that autism (including Asperger syndrome) is an extreme form of the male brain. Currently, the neurobiological basis of such a model remains unclear.

ACKNOWLEDGMENTS

I am grateful for support from the Medical Research Council (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 (1997a) and Baron-Cohen and Swettenham (1997), with permission.

NOTES

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

2. A further confounding variable is that many tests of theory of mind involve some attention shifting and that many tests of executive function involve accounting for one's mental states, such as one's plans and thoughts.

3. Such individuals are described as having either "high-functioning autism" or "Asperger syndrome" (after Hans Asperger, 1944, who first described such a group of children). A difference may exist between these two conditions (Ozonoff et al., 1991) but, for the present purposes, we consider them as one group.

4. Kimura (1992), for example, reported that men are not superior to women in measures of recall of landmarks from a route.

5. Perhaps the best known formulation of the testosterone model is that of Geschwind and Galaburda (1987). Their model is far-ranging, including predictions that testosterone in fetal life will affect immune status, cerebral lateralization, handedness, risk for neurodevelopmental disorder, and many other factors. Evidence for it is mixed. See Bryden et al. (1994) for a critical review, and see 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 et al., (1995) and Fitch and Dennenberg (1996).

6. In the Grimshaw et al. (1995) study, an association was found only between prenatal testosterone and spatial ability in girls, not in boys. The authors of that study 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.

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

8. Precisely when these critical periods occur is left open here, though these likely occur during fetal and early infant stages of development.

9. See Martino and Winner (1995) for a recent study of this area.

10. It should be noted though that anomalous handedness is present also in children with general developmental delay (irrespective of whether they have autism; see Bishop, 1990). What

remains to be seen, then, is whether the anomalous handedness in autism is specific to this condition or is secondary to general developmental delay present in two-thirds of children with autism.

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).

REFERENCES

Argyle, M., & Cook, M. (1976). *Gaze and Mutual Gaze.* Cambridge: Cambridge University Press.

Asperger, H. (1944). Die "Autistischen Psychopathen" im Kindesalter. *Archiv fur Psychiatrie und Nervenkrankheiten, 117,* 76–136.

Avis, J., & Harris, P. (1991). Belief-desire reasoning among Baka children: Evidence for a universal conception of mind. *Child Development, 62,* 460–467.

Baddeley, A. (1991). *Human memory: Theory and practice.* Hillsdale, NJ: Erlbaum.

Baddeley, A., & Wilson, B. (1988). Frontal amnesia and the dysexecutive syndrome. *Brain and Cognition, 7,* 212–230.

Baron-Cohen, S. (1987). Autism and symbolic play. *British Journal of Developmental Psychology, 5,* 139–148.

Baron-Cohen, S. (1988). Social and pragmatic deficits in autism: Cognitive or affective? *Journal of Autism and Developmental Disorders, 18,* 379–402.

Baron-Cohen, S. (1989a). Are autistic children behaviorists? An examination of their mental-physical and appearance-reality distinctions. *Journal of Autism and Developmental Disorders, 19,* 579–600.

Baron-Cohen, S. (1989b). The autistic child's theory of mind: A case of specific developmental delay. *Journal of Child Psychology and Psychiatry, 30,* 285–298.

Baron-Cohen, S. (1989c). Perceptual role-taking and protodeclarative pointing in autism. *British Journal of Developmental Psychology, 7,* 113–127.

Baron-Cohen, S. (1989d). Joint attention deficits in autism: Towards a cognitive analysis. *Development and Psychopathology, 1,* 185–189.

Baron-Cohen, S. (1990). Autism: A specific cognitive disorder of "mind-blindness." *International Review of Psychiatry, 2,* 79–88.

Baron-Cohen, S. (1991a). Do people with autism understand what causes emotion? *Child Development, 62,* 385–395.

Baron-Cohen, S. (1991b). The development of a theory of mind in autism: Deviance and delay? *Psychiatric Clinics of North America, 14,* 33–51.

Baron-Cohen, S. (1991c). Precursors to a theory of mind: Understanding attention in others. In A. Whiten (Ed.), *Natural theories of mind.* Oxford: Basil Blackwell.

Baron-Cohen, S. (1992). Out of sight or out of mind: Another look at deception in autism. *Journal of Child Psychology and Psychiatry, 33,* 1141–1155.

Baron-Cohen, S. (1994). How to build a baby that can read minds: Cognitive mechanisms in mindreading. *Cahiers de Psychologie Cognitive/Current Psychology of Cognition, 13*(5), 513–552.

Baron-Cohen, S. (1995). *Mindblindness: An essay on autism and theory of mind.* Boston: MIT Press.

Baron-Cohen, S., Allen, J., & Gillberg, C., (1992). Can autism be detected at 18 months? The needle, the haystack, and the CHAT. *British Journal of Psychiatry, 161,* 839–843.

Baron-Cohen, S., Campbell, R., Karmiloff-Smith, A., Grant, J., & Walker, J. (1995). Are children with autism blind to the mentalistic significance of the eyes? *British Journal of Developmental Psychology, 13,* 379–398.

Baron-Cohen, S., Cox, A., Baird, G., Swettenham, J., Drew, A., Nightingale, N., & Charman, T. (1996a). Psychological markers of autism at 18 months of age in a large population. *British Journal of Psychiatry, 168,* 158–163.

Baron-Cohen, S., & Cross, P. (1992). Reading the eyes: Evidence for the role of perception in the development of a theory of mind. *Mind and Language, 6,* 173–186.

Baron-Cohen, S., & Goodhart, F. (1994). The "seeing leads to knowing" deficit in autism: The Pratt and Bryant probe. *British Journal of Developmental Psychology, 12,* 397–402.

Baron-Cohen, S., Jolliffe, T., Mortimore, C., & Robertson, M. (1997). Another advanced test of theory of mind: Evidence from very high functioning adults with autism or Asperger syndrome. *Journal of Child Psychology and Psychiatry, 38,* 813–822.

Baron-Cohen, S., & Hammer J. (1997a). Is autism an extreme form of the male brain? *Advances in Infancy Research, 11,* 193–217.

Baron-Cohen, S., & Hammer, J. (1997b). Parents of children with Asperger Syndrome: What is the cognitive phenotype? *Journal of Cognitive Neuroscience, 9,* 548–554.

Baron-Cohen, S., Leslie, A. M., & Frith, U. (1985). Does the autistic child have a "theory of mind"? *Cognition, 21,* 37–46.

Baron-Cohen, S., Leslie, A. M., & Frith, U. (1986). Mechanical, behavioral and intentional understanding of picture stories in autistic children. *British Journal of Developmental Psychology, 4,* 113–125.

Baron-Cohen, S., & Moriarty, J. (1995). Developmental dysexecutive syndrome: Does it exist? A neuropsychological perspective. In M. Robertson & V. Eapen (Eds.), *Movement and allied disorders in childhood.* New York: Wiley.

Baron-Cohen, S., O'Riordan, M., Stone, V., Jones, R., & Plaisted, K. (in press). Recognition of faux pas by normally developing children and children with Asperger syndrome or high functioning autism. *Journal of Autism and Developmental Disorders.*

Baron-Cohen, S., & Ring, H. (1994). A model of the mindreading system: Neuropsychological and neurobiological perspectives. In P. Mitchell, & C. Lewis (Eds.), *Origins of an understanding of mind.* Hillsdale, NJ: Erlbaum.

Baron-Cohen, S., Ring, H., Moriarty, J., Shmitz, P., Costa, D., & Ell, P. (1994). Recognition of mental state terms: A clinical study of autism, and a functional neuroimaging study of normal adults. *British Journal of Psychiatry, 165,* 640–649.

Baron-Cohen, S., & Robertson, M. (1995). Children with either autism, Gilles de la Tourette Syndrome, or both: Mapping cognition to specific syndromes. *Neurocase, 1,* 101–104.

Baron-Cohen, S., Spitz, A., & Cross, P. (1993). Can children with autism recognize surprise? *Cognition and Emotion, 7,* 507–516.

Baron-Cohen, S., Tager-Flusberg, H., and Cohen, D. J. (Eds.) (1993). *Understanding other minds: Perspectives from autism.* Oxford: Oxford University Press.

Baron-Cohen, S., Wheelwright, S., Bolton, P., Stott, C., & Goodyer, I. (1997). Is there a link between engineering and autism? *Autism, 1,* 101–109.

Benbow, C., & Stanley, J. (1980). Sex differences in mathematical ability: Fact or artifact? *Science, 210,* 1262–1264.

Benbow, C., & Stanley, J. (1983). Sex differences in mathematical reasoning ability: More facts. *Science, 222,* 1029–1031.

Bishop, D. (1990). *Handedness and developmental disorder.* Oxford: Blackwell.

Bolton, P., & Rutter, M. (1990). Genetic influences in autism. *International Review of Psychiatry, 2,* 67–80.

Bornstein, R. (1990). Neuropsychological performance in children with Tourette syndrome. *Psychiatry Research, 33,* 73–81.

Bornstein, R. (1991). Neuropsychological correlates of obsessive characteristics in Tourette syndrome. *Journal of Neuropsychiatry and Clinical Neurosciences, 3,* 157–162.

Borys, S., Spitz, H., & Dorans, B. (1982). Tower of Hanoi performance of retarded young adults and nonretarded children as a function of solution length and goal state. *Journal of Experimental Psychology, 33,* 87–110.

Bowler, D. M. (1992). Theory of mind in Asperger syndrome. *Journal of Child Psychology and Psychiatry, 33,* 877–893.

Bretherton, I., McNew, S., & Beeghly-Smith, M. (1981). Early person knowledge as expressed in gestural and verbal communication: When do infants acquire a "theory of mind"? In M. Lamb & L. Sharrod (Eds.), *Infant social cognition.* (pp. 333–374) Hillsdale, NJ: Erlbaum.

Brothers, L. (1990). The social brain: A project for integrating primate behavior and neurophysiology in a new domain. *Concepts in Neuroscience, 1,* 27–51.

Bruner, J. (1983). *Child's talk: Learning to use language.* Oxford: Oxford University Press.

Bryden, M. (1988). An overview of the dichotic listening procedure and its relation to cerebral organization. In K. Hugdahl (Ed.), *Handbook of dichotic listening.* Chichester, UK: Wiley.

Bryden, M., McManus, I. C., & Bulman-Fleming, M. (1994). Evaluating the empirical support for the Geschwind-Behan-Galaburda model of cerebral lateralization. *Brain and Cognition, 26,* 103–167.

Buffery, A., & Gray, J. (1972). Sex differences in the development of spatial and linguistic skills. In C. Ounsted & D. Taylor (Eds.), *Gender differences: Their ontogeny and significance.* London: Churchill Livingstone.

Butterworth, G. (1991). The ontogeny and phylogeny of joint visual attention. In A. Whiten (Ed.) *Natural theories of mind.* Oxford: Basil Blackwell.

Campbell, R., Baron-Cohen, S., & Walker, J. (1995). *Do people with autism show a whole face advantage in recognition of familiar faces and their parts? A test of central coherence theory.* Unpublished manuscript, Goldsmith's College, University of London.

Charman, T., and Baron-Cohen, S. (1992). Understanding beliefs and drawings: A further test of the metarepresentation theory of autism. *Journal of Child Psychology and Psychiatry, 33,* 1105–1112.

Charman, T., & Baron-Cohen, S. (1993). Drawing development in autism: The intellectual to visual realism shift. *British Journal of Developmental Psychology, 11,* 171–185.

Charman, T., & Baron-Cohen, S. (1995). Understanding models, photos, and beliefs: A test of the modularity thesis of metarepresentation. *Cognitive Development, 10,* 287–298.

Chelune, G., Ferguson, W., Koon, R., & Dickey, T. (1986). Frontal lobe disinhibition in attention deficit disorder. *Child Psychiatry and Human Development, 16,* 221–234.

Chiron, C., Leboyer, M., Leon, F., Jambaque, I., Nuttin, N., & Syrota, A. (1995). SPECT study of the brain in childhood autism: Evidence for a lack of normal hemispheril asymmetry. *Developmental Medicine and Childhood Neurology, 37,* 849–861.

Christensen, K., Kim, S., Dysken, M., & Hoover, K. (1992). Neuropsychological performance in obsessive compulsive disorder. *Biological Psychiatry, 31,* 4–18.

Cosmides, L. (1989). The logic of social exchange: Has natural selection shaped how humans reason? Studies with the Wason selection task. *Cognition, 31,* 187–276.

Cranberg, L., & Albert, M. (1988). The chess mind. In L. Obler & D. Fein (Eds.), *The exceptional brain: Neuropsychology of talent and special abilities* (pp. 69–83). New York: Guilford.

Dawson, G., Finley, C., Phillips, S., & Galpert, L. (1986). Hemispheric specialization and the language abilities of autistic children. *Child Development, 57,* 1440–1453.

De Lacoste-Utamsing, C., & Holloway, R. (1982). Sexual differences in the human corpus callosum. *Science, 216,* 1431–1432.

Denenberg, V., Kertesz, A., & Cowell, P. (1991). A factor analysis of the human's corpus callosum. *Brain Research, 548,* 126–132.

Diamond, A. (1991). Neuropsychological insights into the meaning of object concept development. In S. Carey & R. Gelman (Eds.), *The epigenesis of mind: Essays on biology and knowledge.* Hillsdale, NJ: Erlbaum.

Diamond, A. (1994). Phenylaline levels of 6–10 mg/dl may not be as benign as once thought. *Acta Paediatrica, 83,* 89–91.

Downes, J., Roberts, A., Sahakian, B., Evenden, J., Morris, R., & Robbins, T. (1989). Impaired extra-dimensional shift performance in medicated and unmedicated Parkinson's disease: Evidence for a specific attentional dysfunction. *Neuropsychologia, 27,* 1329–1343.

Egaas, B., Courchesne, E., & Saitoh, O. (1995). Reduced size of the corpus callosum in autism. *Archives of Neurology, 52,* 794–801.

Eibl-Eibesfeldt, I. (1989). *Human ethology.* New York: Aldine de Gruyter.

Elliot, R., McKenna, P., Robbins, T., & Sahakian, B. (1995). Neuropsychological evidence for frontostriatal dysfunction in schizophrenia. *Psychological Medicine, 25,* 619–630.

Elliot, R., & Sahakian, B. (1995). The neuropsychology of schizophrenia: Relations with clinical and neurobiological dimensions. *Psychological Medicine, 25,* 581–594.

Fein, D., Humes, M., Kaplan, E., Lucci, D., & Waterhouse, L. (1988). The question of left-hemisphere dysfunction in infantile autism. *Psychological Bulletin, 95,* 258–281.

Fitch, R., & Denenberg, V. (1996). A role for ovarian hormones in sexual differentiation of the brain. *Psychology.*

Flavell, J. H., Green, F. L., & Flavell, E. R. (1986). Development of knowledge about the appearance-reality distinction. *Monographs of the Society for Research in Child Development, 51.*

Fletcher, P., Happe, F., Frith, U., Baker, S., Dolan, R., Frackowiak, R., & Frith, C. (1995). Other minds in the brain: A functional imaging study of "theory of mind" in story comprehension. *Cognition, 57,* 109–128.

Folstein, S., & Rutter, M. (1988). Autism: Familial aggregation and genetic implications. *Journal of Autism and Developmental Disorders, 18,* 3–30.

Frith, U. (1989). *Autism: Explaining the enigma.* Oxford: Basil Blackwell.

Frith, U., and Happe, F. (1994). Autism: Beyond "theory of mind." *Cognition, 50,* 115–132.

Frith, U., Happe, F., & Siddons, F. (1994). Autism and theory of mind in everyday life. *Social Development, 3,* 108–124.

Geary, D. (1996). Sexual selection and **sex** differences in mathematical abilities. *Behavior and Brain Sciences, 19,* 229–284.

Geschwind, N., & Galaburda, A. (1987). *Cerebral lateralization.* Cambridge, MA: MIT Press.

Gilger, J., & Ho, H. (1989). Gender differences in adult spatial information processing: Their relationship to pubertal timing, adolescent activities, and sex-typing of personality. *Cognitive Development, 4,* 197–214.

Goel, V., Grafman, J., Sadato, N., & Hallett, M. (1995). Modelling other minds. *Neuroreport, 6,* 1741–1746.

Goodenough, E. (1957). Interest in persons as an aspect of sex differences in the early years. *Genetic Psychology Monographs, 55,* 287–323.

Gorenstein, E., Mammato, C., & Sandy, J. (1989). Performance of inattentive-overactive children on selected measures of prefrontal type. *Journal of Clinical Psychology, 45,* 619–632.

Gouchie, C., & Kimura, D. (1991). The relationship between testosterone level and cognitive ability patterns. *Psychoneuroendocrinology, 16,* 323–334.

Grimshaw, G., Sitarenios, G., & Finegan, J. (1995). Mental rotation at 7 years: Relations with prenatal testosterone levels and spatial play experiences. *Brain and Cognition, 29,* 85–100.

Grodzinsky, G., & Diamond, A. (1992). Frontal lobe functioning in boys with attention deficit hyperactivity disorder. *Developmental Neuropsychology, 8,* 427–445.

Habib, M., Gayraud, D., Olivia, A., Regis, J., Salamon, G., & Khalil, R. (1991). Effects of handedness and sex on the morphology of the corpus callosum: A study with brain magnetic resonance imaging. *Brain and Cognition, 16,* 41–61.

Hall, J. (1977). Gender effects in decoding non-verbal cues. *Psychological Bulletin, 85,* 845–857.

Halpern, D. (1992). *Sex differences in cognitive ability* (2nd ed.). Hillsdale, NJ: Erlbaum.

Hassler, M., & Gupta, D. (1993). Functional brain organization, handedness and immune vulnerability in musicians and non-musicians. *Neuropsychologia, 31,* 655–660.

Happe, F. (1993). Communicative competence and theory of mind in autism: A test of relevance theory. *Cognition, 48,* 101–119.

Happe, F. (1994). An advanced test of theory of mind: Understanding of story characters' thoughts and feelings by able autistic, mentally handicapped, and normal children and adults. *Journal of Autism and Developmental Disorders, 24,* 129–154.

Happe, F. (1997). Central coherence and theory of mind in autism. Reading homographs in context. *British Journal of Developmental Psychology, 15,* 1–12.

Head, D., Bolton, D., & Hymas, N. (1989). Deficit in cognitive shifting ability in patients with obsessive-compulsive disorder. *Biological Psychiatry, 25,* 929–937.

Hines, M. (1990). Gonadal hormones and human cognitive development. In J. Balthazart (Ed.), *Hormones, brain and behavior in vertebrates: I. Sexual differentiation, neuroanatomical aspects, neurotransmitters, and neuropeptides.* Basel: Karger.

Hines, M., & Green, R. (1991). Human hormonal and neural correlates of sex-typed behaviors. *Review of Psychiatry, 10,* 536–555.

Hobson, R. P. (1984). Early childhood autism and the question of egocentrism. *Journal of Autism and Developmental Disorders, 14,* 85–104.

Hughes, C., & Russell, J. (1993). Autistic children's difficulty with mental disengagement from an object: Its implications for theories of autism. *Developmental Psychology, 29,* 498–510.

Hughes, C., Russell, J., & Robbins, T. (1994). Specific planning deficit in autism: Evidence of a central executive dysfunction. *Neuropsychologia, 33,* 477–492.

Hutt, C. (1972). Neuroendocrinal behavior and intellectual aspects of sexual differentiation in human development. In C. Ounsted & D. Taylor (Eds.), *Gender differences: Their ontogeny and significance*. Edinburgh: Churchill Livingstone.

Hyde, J., & Linn, M. (1988). Gender differences in verbal ability: A meta-analysis. *Psychological Bulletin, 104,* 53–69.

Johnson, E. (1984). Sex differences in problem-solving. *Journal of Educational Psychology, 76,* 1359–1371.

Jolliffe, T., & Baron-Cohen, S. (1997). Are people with autism and Asperger syndrome faster than normal on the embedded figures test? *Journal of Child Psychology and Psychiatry, 38,* 527–534.

Kalichman, S. (1989). The effects of stimulus context on paper and pencil spatial task performance. *Journal of General Psychology, 116,* 133–139.

Karmiloff-Smith, A., Klima, E., Bellugi, U., Grant, J., & Baron-Cohen, S. (1995). Is there a social module? Language, face-processing and theory of mind in subjects with William's syndrome and autism. *Journal of Cognitive Neuroscience, 7,* 196–208.

Kerns, K., & Berenbaum, S. (1991). Sex differences in spatial ability in childhood. Behavior Genetics, *21,* 383–396.

Kimura, D. (1992). Sex differences in the brain. *Scientific American, September,* 119–125.

Kimura, D., & D'Amico, C. (1989). Evidence for subgroups of adextrals based on speech lateralization and cognitive patterns. *Neuropsychologia, 27,* 977–986.

Law, D., Pellegrino, J., & Hunt, E. (1993). Comparing the tortoise and the hare: Gender differences and experiences in dynamic spatial reasoning tasks. *Psychological Science, 4,* 35–40.

Leekam, S., Baron-Cohen, S., Brown, S., Perrett, D., & Milders, M., (1997). Eye-direction detection: A dissociation between geometric and joint-attention skills in autism. *British Journal of Developmental Psychology, 15,* 77–95.

Leekam, S., & Perner, J. (1991). Does the autistic child have a metarepresentational deficit? *Cognition, 40,* 203–218.

Leslie, A. M. (1987). Pretence and representation: The origins of "theory of mind." *Psychological Review, 94,* 412–426.

Leslie, A. M., & Frith, U. (1988). Autistic children's understanding of seeing, knowing, and believing. *British Journal of Developmental Psychology, 6,* 315–324.

Leslie, A. M., & Thaiss, L. (1992). Domain specificity in conceptual development: Evidence from autism. *Cognition, 43,* 225–251.

Lewis, V., & Boucher, J. (1988). Spontaneous, instructed and elicited play in relatively able autistic children. *British Journal of Developmental Psychology, 6,* 325–339.

Linn, M., & Petersen, A. (1985). Emergence and characterization of sex differences in spatial ability: A meta-analysis. *Child Development, 56,* 1479–1498.

Loge, D., Staton, D., & Beatty, W. (1990). Performance of children with ADHD on tests sensitive to frontal lobe dysfunction. *Journal of the American Academy of Child and Adolescent Psychiatry, 29,* 540–545.

Lummis, M., & Stevenson, H. (1990). Gender differences in beliefs and achievement: A cross-cultural study. *Developmental Psychology, 26,* 254–263.

Martino, G., & Winner, E. (1995). Talents and disorders: Relationships among handedness, sex, and college major. *Brain and Cognition, 29,* 66–84.

Marshall, S., & Smith, J. (1987). Sex differences in learning mathematics: A longitudinal study with item and error analyses. *Journal of Educational Psychology, 79,* 372–383.

Masters, M., & Sanders, B. (1993). Is the gender difference in mental rotation disappearing? *Behavior Genetics, 23,* 337–341.

McGee, M. (1979). Human spatial abilities: Psychometric studies and environmental, genetic, hormonal, and neurological influences. *Psychological Bulletin, 86,* 889–918.

McGuinness, D., & Pribam, K. (1979). The origins of sensory bias in the development of gender differences in perception and cognition. In M. Bortner (Ed.), *Cognitive growth and development: Essays in memory of Herbert Birch.* New York: Brunner/Mazel.

McManus, I. C. (1985). Handedness, language dominance, and aphasia: A genetic model. *Psychological Medicine,* Monograph Supplement 8.

Mebert, C., & Michel, G. (1980). Handedness in artists. In L. Herron, (Ed.), *Neuropsychology of left-handedness.* New York: Academic.

Mills, C., Ablard, K., & Stumpf, H. (1993). Gender differences in academically talented young students' mathematical reasoning: Patterns across age and subskills. *Journal of Educational Psychology, 85,* 340–346.

Milner, B. (1964). Some effects of frontal lobectomy in man. In J. Warren & K. Akert (Eds.), *The frontal granular cortex and behavior.* New York: McGraw-Hill.

Moir, A., & Jessel, D. (1989). *Brainsex.* London: Mandarin Press.

Norman, D., & Shallice, T. (1980). Attention to action: Willed and automatic control of behavior. In R. Davidson G. Schwartz & D. Shapiro (Eds.), *Consciousness and self-regulation: Vol 4.* New York: Plenum.

Owen, A., Roberts, A., Polkey, C., Sahakian, B., & Robbins, T. (1991). Extradimensional versus intradimensional set shifting performance following frontal lobe excisions, temporal lobe excisions, or amygdalo-hippocampectomy in man. *Neuropsychologia, 10,* 99–106.

Ozonoff, S., Pennington, B., & Rogers, S. (1991). Executive function deficits in high-functioning autistic children: Relationship to theory of mind. *Journal of Child Psychology and Psychiatry, 32,* 1081–1106.

Ozonoff, S., Rogers, S., & Pennington, B. (1991). Asperger's syndrome: Evidence of an empirical distinction from high-functioning autism. *Journal of Child Psychiatry and Psychology, 32,* 1107–1122.

Pennington, B., van Doorninck, W., McCabe, L., & McCabe, E. (1985). Neurological deficits in early treated phenylketonurics. *American Journal of Mental Deficiency, 89,* 467–474.

Perner, J., Frith, U., Leslie, A. M., & Leekam, S. (1989). Exploration of the autistic child's theory of mind: Knowledge, belief, and communication. *Child Development, 60,* 689–700.

Perret, E. (1974). The left frontal lobe of man and the suppression of habitual responses in verbal categorical behavior. *Neuropsychologia, 16,* 527–537.

Perrett, D., Smith, P., Potter, D., Mistlin, A., Head, A., Milner, A., & Jeeves, M. (1985). Visual cells in the temporal cortex sensitive to face view and gaze direction. *Proceedings of the Royal Society of London, B223,* 293–317.

Peterson, J. (1979). Left-handedness: Difference between student artists and scientists. *Perceptual and Motor Skills, 48,* 961–962.

Peterson, J., & Lansky, L. (1974). Left-handedness among architects: Some facts and speculations. *Perceptual and Motor Skills, 38,* 547–550.

Phillips, W. (1993). *Understanding intention and desire by children with autism.* Unpublished doctoral dissertation, Institute of Psychiatry, University of London.

Phillips, W., Baron-Cohen, S., & Rutter, M. (1992). The role of eye-contact in the detection of goals: Evidence from normal toddlers, and children with autism or mental handicap. *Development and Psychopathology, 4,* 375–383.

Phillips, W., Gomez, J-C., Baron-Cohen, S., Riviere, A., & Laa, V. (1995). Treating people as objects, agents, or subjects: How young children with and without autism make requests. *Journal of Child Psychology and Psychiatry, 36,* 1383–1398.

Pinker, S. (1998). *How the mind works.* New York: Norton.

Piren, J., Arndt, S., Bailey, J., Havercamp, S., Andeason, N., & Palmer, P. (1995). An MRI study of brain size in autism. *American Journal of Psychiatry, 152,* 1145–1149.

Prior, M., & Bradshaw, J. (1979). Hemispheric functioning in autistic children. *Cortex, 15,* 73–81.

Prior, M., & Hoffman, W. (1990). Neuropsychological testing of autistic children through exploration with frontal lobe tests. *Journal of Autism and Developmental Disorders, 20,* 581–590.

Reed, T., & Peterson, C. (1990). A comparative study of autistic subjects' performance at two levels of visual and cognitive perspective taking. *Journal of Autism and Developmental Disorders, 20,* 555–568.

Resnick, S., Berenbaum, S., Gottesman, I., & Bouchard, T., Jr. (1986). Early hormonal influences on cognitive functioning in congenital adrenal hyperplasia. *Developmental Psychology, 22,* 191–198.

Rhees, R., Shyrne, J., & Gorski, R. (1990). Onset of the hormone-sensitive perinatal period for sexual differentation of the sexually dimorphic nucleus of the preoptic area. *Journal of Neurobiology, 21,* 781–786.

Rosenblatt, E., & Winner, E. (1988). Is superior visual memory a component of superior drawing ability? In L. Obler & D. Fein (Eds.), *The exceptional brain: Neuropsychology of talent and special abilities.* New York: Guilford Press.

Rumsey, J., & Hamburger, S. (1988). Neuropsychological findings in high functioning men with infantile autism, residual state. *Journal of Clinical and Experimental Neuropsychology, 10,* 201–221.

Rutter, M. (1978). Diagnosis and definition. In M. Rutter & E. Schopler (Eds.), *Autism: A reappraisal of concepts and treatment.* New York: Plenum Press.

Rutter, M. (1983). Cognitive deficits in the pathogenesis of autism. *Journal of Child Psychology and Psychiatry, 24,* 513–531.

Satz, P., Soper, H., Orsini, D., Henry, R., & Zvi, J. (1985). Handedness subtypes in autism. *Psychiatric Annals, 15,* 447–451.

Scaife, M., & Bruner, J. (1975). The capacity for joint visual attention in the infant. *Nature, 253,* 265–266.

Scott, F., & Baron-Cohen, S. (1996). Logical, analogical, and psychological reasoning in autism: A test of the Cosmides theory. *Development and Psychopathology, 8,* 235–246.

Shah, A., & Frith, U. (1983). An islet of ability in autism: A research note. *Journal of Child Psychology and Psychiatry, 24,* 613–620.

Shah, A., & Frith, U. (1993). Why do autistic individuals show superior performance on the block design test? *Journal of Child Psychology and Psychiatry, 34,* 1351–1364.

Shallice, T. (1982). Specific impairments of planning. *Philosophical Transactions of the Royal Society of London, B298,* 199–209.

Shallice, T. (1988). *From neuropsychology to mental structure.* Cambridge, UK: Cambridge University Press.

Sigman, M., Mundy, P., Ungerer, J., & Sherman, T. (1986). Social interactions of autistic, mentally retarded, and normal children and their caregivers. *Journal of Child Psychology and Psychiatry, 27,* 647–656.

Sodian, B., & Frith, U. (1992). Deception and sabotage in autistic, retarded, and normal children. *Journal of Child Psychology and Psychiatry, 33,* 591–606.

Soper, H., Satz, P., Orsini, D., Henry, R., Zvi, J., & Schulman, M. (1986). Handedness patterns in autism suggests subtypes. *Journal of Autism and Developmental Disorders, 16,* 155–167.

Steffenberg, S., Gillberg, C., Hellgren, L., Andersson, L., Gillberg, C., Jakobsson, G., & Bohman, M. (1989). A twin study of autism in Denmark, Finland, Iceland, Norway, and Sweden. *Journal of Child Psychology and Psychiatry, 30,* 405–416.

Steinkamp, M., Harnisch, D., Walberg, H., & Tsai, S. (1985). Cross national gender differences in mathematics attitude and achievement among 13 year olds. *Journal of Mathematical Behavior, 4,* 259–277.

Stevenson, H., Lee, S., Chen, C., Lummis, M., Stigler, J., Fan, L., & Ge, F. (1990). Mathematics achievement of children in China and the United States. *Child Development, 61,* 1053–1066.

Surian, L., Baron-Cohen, S., & Van der Lely, H. (1996). Are children with autism deaf to Gricean Maxims? *Cognitive Neuropsychiatry, 1,* 55–72.

Swettenham, J. (1996). Can children be taught to understand false belief using computers? *Journal of Child Psychology and Psychiatry, 37,* 157–166.

Swettenham, J., Baron-Cohen, S., Gomez, J-C., & Walsh, S. (1996). What's inside a person's head? Conceiving of the mind as a camera helps children with autism develop an alternative to a theory of mind. *Cognitive Neurospychiatry, 1,* 73–88.

Tager-Flusberg, H. (1992). Autistic children's talk about psychological states: Deficits in the early acquisition of a theory of mind. *Child Development, 63,* 161–172.

Tager-Flusberg, H. (1993). What language reveals about the understanding of minds in children with autism. In S. Baron-Cohen, H. Tager-Flusberg, & D. J. Cohen (Eds.), *Understanding other minds: Perspectives from autism.* Oxford: Oxford University Press.

Tan, J., & Harris, P. (1991). Autistic children understand seeing and wanting. *Development and Psychopathology, 3,* 163–174.

Voyer, D., Voyer, S., & Bryden, M. (1995). Magnitude of sex differences in spatial abilities: A meta-analysis and consideration of critical variables. *Psychological Bulletin, 117,* 250–270.

Wellman, H. (1990). *Children's theories of mind.* Cambridge, MA: MIT Press Bradford Books.

Wellman, H., & Estes, D. (1986). Early understanding of mental entities: A reexamination of childhood realism. *Child Development, 57,* 910–923.

Welsh, M., Pennington, B., Ozonoff, S., Rouse, B., & McCabe, E. (1990). Neuropsychology of early-treated phenylketonuria: Specific executive function deficits. *Child Development, 61,* 1679–1713.

Williams, C., Barnett, A., & Meck, W. (1990). Organizational effects of early gonadal secretions on sexual differentiation in spatial memory. *Behavioral Neuroscience, 104,* 84–97.

Wimmer, H., & Perner, J. (1983). Beliefs about beliefs: Representation and constraining function of wrong beliefs in young children's understanding of deception. *Cognition, 13,* 103–128.

Wing, L. (1981). Asperger syndrome: A clinical account. *Psychological Medicine, 11,* 115–130.

Wing, L. (1988). The autistic continuum. In L. Wing (Ed.), *Aspects of autism: Biological research.* London: Gaskell/Royal College of Psychiatrists.

Wing, L., Gould, J., Yeates, S. R., & Brierley, L. M. (1977). Symbolic play in severely mentally retarded and in autistic children. *Journal of Child Psychology and Psychiatry, 18,* 167–178.

Witelson, S. (1976). Sex and the single hemisphere: Specialization of the right hemisphere for spatial processing. *Science, 193,* 425–427.

Witelson, S. (1989). Hand and sex differences in the isthmus and genu of the human corpus callosum. *Brain, 112,* 799–835.

Witelson, S. (1991). Neural sexual mosaicism: Sexual differentiation of the human temporo-parietal region for function asymmetry. *Psychoneuroendocrinology, 16,* 131–153.

Witkin, H., Oltman, P., Raskin, E., & Karp, S. (1971). *A manual for the embedded figures test.* Palo Alto, CA: Consulting Psychologists Press.

Zelinski, C., Taylor, M., & Juzwin, K. (1991). Neuropsychological deficits in obsessive-compulsive disorder. *Neuropsychiatry, Neuropsychology and Behavioral Neurology, 4,* 110–126.