9 A Model of the Mindreading System: Neuropsychological and Neurobiological Perspectives

Simon Baron-Cohen
Institute of Psychiatry, University of London, UK
Howard Ring
Institute of Neurology, University of London, UK

INTRODUCTION

There is mounting evidence that ToMM (the Theory of Mind Mechanism) may be a modular system (Baron-Cohen, 1992; Leslie, 1991; Leslie & Roth, 1993). ToMM is the name Leslie gives to the system underpinning our everyday theory of mind. ToMM's modularity rests on two arguments:

1. Children with autism are impaired in their understanding of epistemic mental states (such as beliefs: Baron-Cohen, Leslie, & Frith, 1985; Perner, Frith, Leslie, & Leekam, 1989), but are unimpaired in their understanding of nonmental representations (such as photographs, drawings, maps, and models: Charman & Baron-Cohen, 1992; 1993; Leekam & Perner, 1991; Leslie & Thaiss, 1992); and
2. Understanding mental states requires the processing of a special kind of representation. Leslie and Roth (1993) call these *M-Representations*[1], and suggest they have the following structure[2]:

[Agent-Attitude-"Proposition"].

[1] They adopt this term in order to avoid the confusions arising from the earlier term 'metarepresentations' (Perner, 1993).

[2] In recent writings, Leslie suggests M-Representations have a 4th term, to express an anchor in reality (e.g. see Leslie, German, & Happé, 1993).

Examples of what could fill these three slots are:

[John-thinks-"The money is in the biscuit tin"], or
[Mary-believes-"The moon is made of green cheese"].

In these examples, the whole M-Representation can be true even if the proposition is false (i.e. even if the money is not in the biscuit tin, or if the moon is not made of green cheese). For example, the M-Representation can be true if John indeed *thinks* the money is in the biscuit tin, or if Mary really *believes* the moon is made of green cheese. The usefulness of M-Representations is that they allow prediction of an Agent's future action. For example, they lead us to predict that John will go to the biscuit tin if he wants the money. M-Representation also allow one to make sense of an Agent's behaviour. For example, they help make sense of why John looks disappointed when he opens the biscuit tin.

The evidence from autism, showing that such children have specific difficulties in predicting an Agent's behaviour on the basis of beliefs and other mental states, in combination with Leslie's account of ToMM's unique class representations, satisfy two criteria for a system to be modular: the possibility of neural dissociation, and the existence of specific, dedicated representations (Fodor, 1983; Jackendoff, 1987).

According to Leslie (1987), ToMM comes on line in the middle of the second year of life, and its arrival is marked by the production and comprehension of pretence. Building on Leslie's model, Baron-Cohen (in press a; in press b) argues for the existence of three developmentally earlier modules:

1. ID (the Intentionality Detector),
2. EDD (the Eye-Direction Detector), and
3. SAM (the Shared Attention Mechanism).

As will be explained, SAM is held to play a crucial role in triggering ToMM to function. In this chapter, we begin by briefly summarising the neuropsychology of these mechanisms. We then shift levels to consider the possible neurobiology of these mechanisms. In doing this, we seek to integrate Baron-Cohen's (in press a; in press b) psychological theory with Brothers' (1990) neurobiology theory.

THE NEUROPSYCHOLOGY OF THE MINDREADING SYSTEM

Figure 9.1 shows the four postulated components in the Mindreading System[3]. Here, we review each of the four mechanisms in turn.

[3] Further details of each of them can be found elsewhere (Baron-Cohen, in press a; in press b).

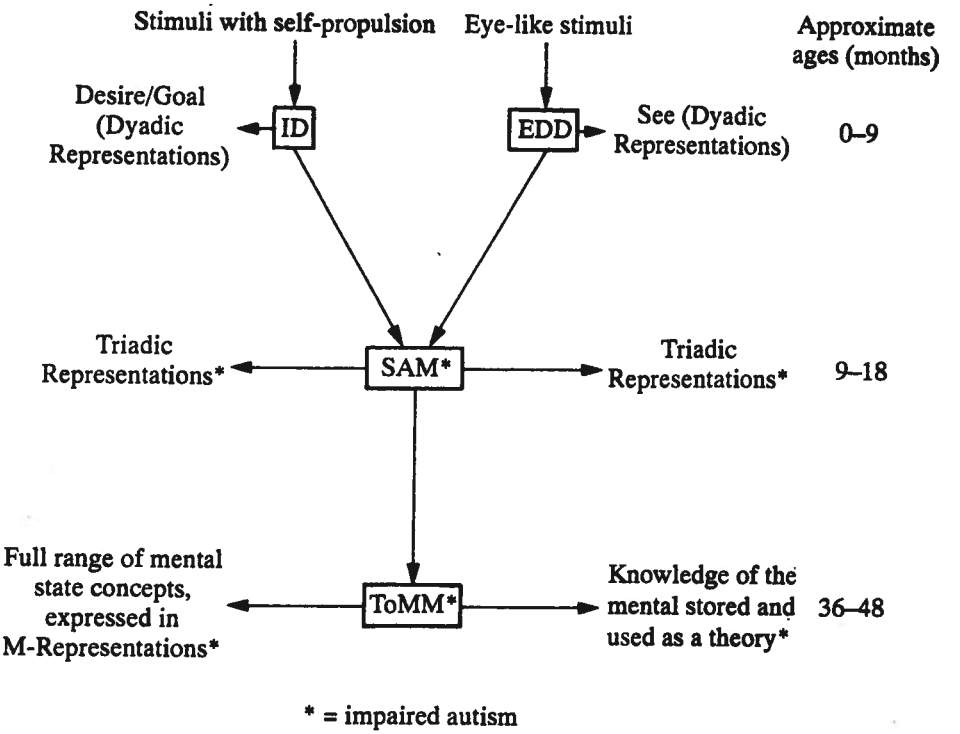

FIG. 9.1 The four components of the Mindreading System (adapted from Baron-Cohen, in press a).

The Intentionality Detector

ID is a primitive perceptual mechanism that is amodal, and that interprets self-propelling stimuli in terms of that stimulus' goal or desire. That is, it reads directional stimuli as volitional. In this system, "goal" is defined as the target of an action, and "desire" is defined as a movement towards or away from a target. ID is very similar to Premack's (1990) notion of a module that is hand-wired into our visual system to detect intentionality—though in the case of ID, the mental states of goal and desire are read into a wide range of stimuli with *direction* (a touch, a push, a jump, a shout, any movement that occurs without an apparent external cause, etc). ID builds *dyadic* representations of behaviour, of the form:

[Agent–Relation–Object], or
[Agent–Relation–Self], or
[Self–Relation–Object], or
[Self–Relation–Agent].

Examples of these are:

[The man-has goal-the door], or
[The mouse-wants-the cheese],
[I-want-the cup].

The Eye-direction Detector

EDD is a system that functions to detect the presence of eyes (or eye-like stimuli), and then builds dyadic representations of eye behaviour. These are built, for example, every time EDD detects that another's eyes are "looking at me." EDD's dyadic representations have an identical structure to ID's, but whereas for ID the Relation slot is filled with a term like "goal" or "desire," in the case of EDD the Relation slot is filled with a term like "see," "look," etc. That is, eyes or eye-like stimuli are read as visual and informational. In the case of both ID and EDD, their representations are dyadic because there are only two entities, connected by a Relation term.

Examples of EDD's representations are:

[Mother-sees-me], or
[The cat-sees-the mouse], or
[I-see-the man].

The Shared Attention Mechanism

SAM is a mechanism that functions to check if you and another organism are attending to the same object. To do this, SAM builds *triadic* representations[4]. These are more complex than dyadic representations. They have a structure that is expressed by something like the following:

[Agent–Relation–(Self–Relation–Object)], or
[Self–Relation–(Agent–Relation–Object)].

Examples are:

[Mother-sees-(I-see-the car)], or
[I-see-(Mother-looking at-the door)].

What makes these representations triadic is the inclusion of an embedded dyadic representation (e.g. [Self-Relation-Object] or [Agent-Relation-Object]). This is required in order for the triadic representation to specify that both Self and Agent are looking at the *same* object, and that one or

[4] The distinction between dyadic and triadic relations stems from Bakeman and Adamson (1984). They are also key distinctions that Gomez (1991) draws. Hobson (this volume) brings out the importance of such relations in talking about the "triangle." In Baron-Cohen's (in press a; in press b) theory, these terms describe types of *representation*.

both of the Agents has detected this. Perhaps a more accurate way of expressing this is via the diagram in Fig. 9.2.

SAM is a central mechanism that depends on ID and EDD for its input. That is, it can only build triadic representations out of dyadic ones. Because these are more easily constructed out of EDD's dyadic representations, EDD and SAM are held to have a privileged relationship: EDD sends its output to SAM, as is shown in Fig. 9.1.

SAM can do a few more things. First, it connects ID to EDD. This means that in the triadic representations that SAM builds, the Relation slot can be filled by either visual terms ("see," "look," etc.) or volitional terms ("goal," "desire," etc.). This allows SAM to read eye-direction in terms of an Agent's goals and desires. Secondly, SAM passes its triadic representations to the fourth of the components in the Mindreading System, ToMM. This is held to be necessary to trigger ToMM to function (Baron-Cohen, 1989; 1991a; 1993; in press a; in press b).

The Theory of Mind Mechanism

As briefly mentioned in the Introduction to this chapter, we follow Leslie's description of this component. That is, ToMM is held to represent something like M-Representations, employing the full range of mental state concepts in the Attitude slot. These include the epistemic states (believe,

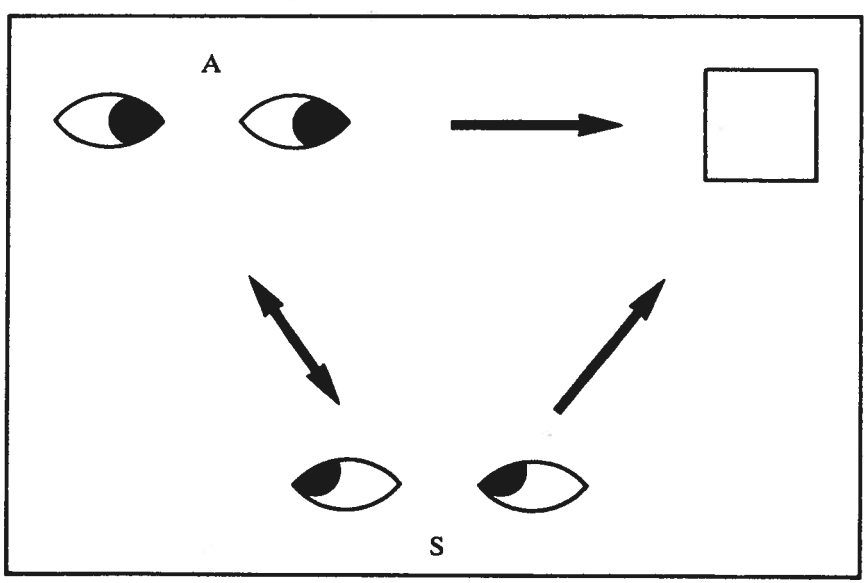

FIG. 9.2 A triadic representation (adapted from Baron-Cohen, in press a). S = Self, and A = Agent.

think, know, pretend, imagine, dream, etc.). ToMM's second function is to organise mental state knowledge into a useful and coherent theory of action, such that it becomes both explanatory and predictive (Premack & Woodruff, 1978; Wellman, 1990).

So much for the background. What of the neuropsychology of these four mechanisms? Here, we examine both the normal developmental timetable of these four mechanisms, and the possible dissociability between them in cases where development goes wrong. Specifically, we focus on two kinds of pathology: autism, and congenital blindness[5].

Normal Development

ID is probably functional from early in infancy (Premack, 1990). Rather than infants operating like behaviourists at any point in development, they probably read movement in terms of goal-directed action from the outset. Certainly, by the time they speak they refer to goal-directed action and desires (Wellman, 1990).

EDD is clearly functioning in infancy, as shown by infants' selective attention to other people's eyes (Maurer & Barrera, 1981). This selective attention to the eyes appears to have a long evolutionary heritage: From the reptiles, to the birds, to the primates one can find selective attention to the eyes of another organism (Baron-Cohen, in press a; in press b). The fact that human infants smile when they receive eye-contact (Wolff, 1963) is evidence that they can detect when "eyes are directed at me." Certainly by preschool age, when they can be tested verbally, they have no difficulty in correctly responding to the test question "Which one is looking at you?" when presented with pairs of photographs of faces like those in Fig. 9.3 (Baron-Cohen & Cross, 1992).

SAM is held to come on line slightly later than the two previous mechanisms: around 8–12 months of age. The clearest expressions of this are spontaneous gaze-monitoring (Butterworth, 1991; Scaife & Bruner, 1975) and spontaneous gaze-directing behaviours such as protodeclarative pointing (Bates, Camaioni, & Volterra, 1975). In both phenomena, the infant alternates his or her own gaze back and forth between the adult's eyes and the object to which they are both attending, or at which the infant is directing the adult's gaze (Baron-Cohen, 1989; 1991a; Gomez, 1991).

Regarding SAM's function of inferring goal from eye-direction, Phillips, Baron-Cohen, and Rutter (1992) investigated this with normal toddlers ranging from 9–18 months. The child was presented either with an ambiguous or an unambiguous action. One ambiguous action comprised *blocking* the

[5] We follow Hobson (1990) in drawing this comparison, though we use it to emphasise the differential dissociation of the four components of the Mindreading System.

FIG. 9.3 "Which one is looking at you?" (from Baron-Cohen & Cross, 1992, reproduced with permission).

child's hands during manual activity, by the adult cupping her hands over the child's. A second ambiguous action comprised offering an object to the child, but then at the last minute *teasingly* withdrawing it, just as the child began to reach for it. The unambiguous action simply comprised *giving* or presenting an object to the child. Phillips et al. found that, on at least half of the trials, 100% of the infants responded to the ambiguous actions by instantly looking at the adult's eyes (within the first 5 seconds after the tease or the block), whereas only 39% of them did so following the unambiguous action, using the same criteria. This suggests that under conditions in which the goal of an action is uncertain, the first place young children (and indeed adults) look for information to disambiguate the goal is the eyes.

A further study demonstrated that it is indeed eye-*direction* that children use to infer the mental state of goal (Baron-Cohen, Campbell, et al., 1993). Since this paradigm was verbal, young three-year-olds were tested. The child was asked "Which chocolate will Charlie take?" after being shown a display of four sweets and a cartoon character (Charlie's face) looking at one of these (see Fig. 9.4). Subjects tended to pick the one Charlie was *looking at* as the goal of his next action, and this was statistically significant. Regarding evidence for SAM's function of inferring desire from eye-direction, Baron-Cohen, Campbell, et al. (1993) also presented normal three- to four-year-olds with the display of the four sweets, with Charlie's eyes pointing towards one of the four sweets, randomly selected (as in Fig. 9.4), and asked the

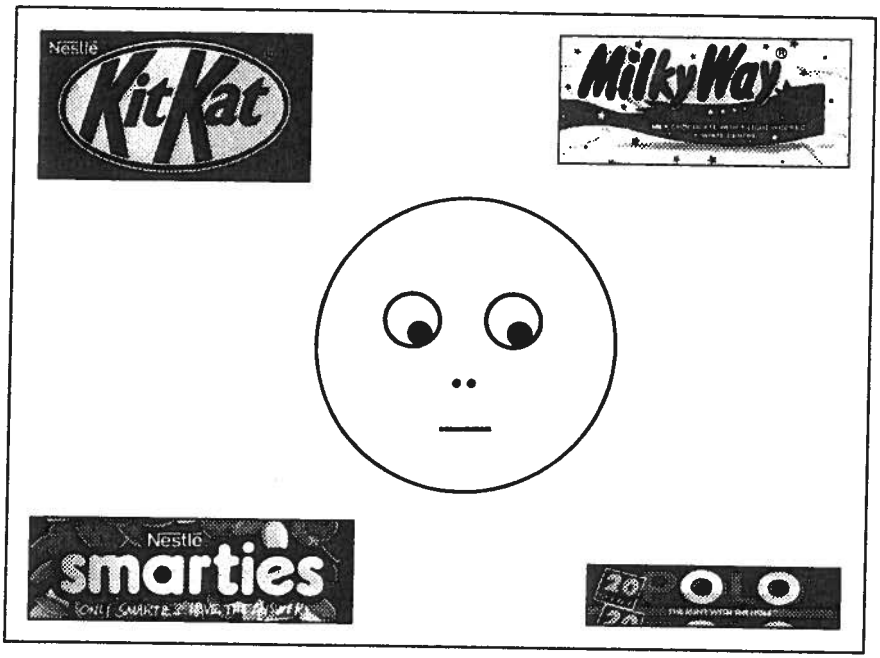

FIG. 9.4 The Four Sweets Display (adapted from Baron-Cohen, Campbell et al., 1993).

subject "Which one does Charlie *want*?" Children of this age had no difficulty at all in inferring Charlie's desire from his eye-direction.

ToMM, as Leslie (1987) suggests, probably makes its first appearance with pretence, around 18–24 months of age. By 3 years of age, preschoolers are able to understand aspects of what people know (Pratt & Bryant, 1990), and by 4 years of age they can distinguish true and false beliefs (Wimmer & Perner, 1983). Their mental-state knowledge is also highly organised into a coherent "theory" which the child uses for both explanation and prediction of behaviour (Wellman, 1990).

Autism

Both ID and EDD appear to be intact in children with autism. Evidence for these claims includes the following: Such children use words referring to goal-directed action and desire in their spontaneous speech (Baron-Cohen, Leslie, & Frith, 1986; Tager-Flusberg, 1993), they can predict emotions on the basis of a person's desire (Baron-Cohen, 1991b), and they can detect when the eyes of another person are directed at them (Baron-Cohen, Campbell, et al., 1993). However, SAM appears to be impaired in autism. Key evidence for this is that they show little if any spontaneous gaze

monitoring (Leekam et al., 1993; Sigman, Mundy, Ungerer, & Sherman, 1986) or spontaneous protodeclative pointing to direct another's gaze to share interest in something, as an end in itself (Baron-Cohen, 1989).

If SAM is impaired in autism, then children with autism should also have difficulties in reading eye-direction in terms of mental states such as goal and desire. There is some evidence that supports this prediction. Phillips et al. (1992) tested very young children with autism for their ability to use eye-direction to detect a person's goals, using the ambiguous and unambiguous actions described earlier. However, these children did not use eye-contact to disambiguate the ambiguous actions, looking as little in both conditions (less than 11% looking, in each). Baron-Cohen, Campbell, et al. (1993) went on to test children with autism to see if, on the Four Sweets Task, they were able to use eye-direction to infer the mental states of want and goal. They found significant impairments in the use of eye-direction in inferring both of these.

Autism and Congenital Blindness

SAM and ToMM, as so far described, are independent systems with a special relationship: ToMM is activated by taking SAM's triadic representations as input. This is a testable claim about a certain kind of precursor relationship[6] between the two systems in development. The claim is that SAM is a necessary (though not sufficient) condition for the development of ToMM. Note, though, that although SAM usually builds triadic representations using EDD's dyadic representations, this need not be the case. For example, since children with congenital blindness lack EDD, SAM must be restricted to building triadic representations specifying joint-attention via touch or audition (e.g. [Mother-touches-(I-touch-the apple)]). So, in their case, SAM must be building triadic representations using ID's dyadic representations. These kind of triadic representations are likely to be considerably more difficult to build than those derived from EDD.

To summarise, the key claims can be elaborated as follows:

1. SAM's triadic representations are necessary to trigger ToMM's development.
2. SAM exploits EDD as the system of choice for building triadic representations.

[6] Gopnik, Slaughter, and Meltzoff (this volume) also pursue the argument that understanding perception is a precursor for understanding belief, though for them this is couched in terms of theory-change. Butterworth (this volume) similarly argues that joint-attention is one of the important routes towards an understanding of mind in the developing child. Our account is compatible with both of these accounts, but as the reader will gather, ours is both a modular and a neurobiological thesis.

3. In most cases of autism, SAM is impaired whilst EDD is not.
4. In congenital blindness, EDD is impaired but SAM is not.

Regarding (2), this is not only because joint attention can be established on distal objects more easily via vision than it can via touch or audition, but also because eye-direction is a more reliable source of information about a person's goals, interest, etc., than is (say) body-orientation. Hence the assumption that EDD and SAM have been prioritised in evolution for solving the problem of detecting a person's mental states of attention, desire, and goal (Baron-Cohen, in press a; in press b).

Given the evidence presented earlier, that most children with autism fail to develop a fully functioning SAM, it follows from the theory outlined here that in these children this would have the knock-on effect of not activating ToMM. In principle, however, the theory predicts two subgroups of autism:

Subgroup 1. Both SAM and ToMM are impaired, as explained by the knock-on hypothesis. There is considerable evidence suggesting that many subjects with autism fall into this group (see Baron-Cohen, 1993, for a review)[7].

Subgroup 2. SAM is intact, whereas ToMM is impaired in its own right. This group remains to be properly investigated. One possibility is that this group corresponds to those children with autism who are reported to have a period of normal development up to the age of 18 months (Derek Ricks, personal communication, 1985; Volkmar & Cohen, 1989), and then show clear signs of autism.

Finally it follows from this theory that in children with congenital blindness, since SAM is intact, ToMM should also develop, although a slight delay in this would not be surprising given the need for SAM to use ID instead of EDD. Here too, we await the relevant tests of ToMM in children with congenital blindness.

THE NEUROBIOLOGY OF THE MINDREADING SYSTEM

In this section we present a neurobiological model. This model is of necessity speculative, as many pieces of the jigsaw are not yet in place. However, we feel that there are enough clues to begin to sketch the model. Our model is based on an integration of our work (Baron-Cohen, in press a; in press b;

[7] Consistent with this, absence of joint attention and pretend play is one predictor of autism at 18 months of age (Baron-Cohen, Allen, & Gillberg, 1992).

Baron-Cohen, Ring, et al., 1993) with that of Brothers (Brothers, 1990; Brothers & Ring, 1992). The model proposes:

1. that the superior temporal sulcus (STS) is responsible for EDD's functions;
2. that since SAM uses EDD, the STS may be responsible for aspects of SAM too;
3. that the orbito-frontal cortex (OFC) is responsible for some of ToMM's functions;
4. that the amygdala may be involved in several components of the Mindreading System; and
5. that these three brain areas form a circuit via important neural interconnections.

These three brain regions are shown in Fig. 9.5. Frith (1992) proposes a similar model, also based on Brothers (1990). We turn to consider the evidence for these assumptions.

The STS: The Seat of EDD and SAM?

Specific "face" cells were identified by Bruce, Desimone, and Gross (1981) and Perrett, Rolls, and Caan (1982) in the STS. Later studies demonstrated the role of specific cells which respond selectively to *individual* familiar faces (Bayliss, Rolls, & Leonard, 1985; Desimone, Albright, Gross, & Bruce, 1984; Kendrick & Baldwin, 1987; Perrett et al., 1984; Yamane, Kaji, & Kawano, 1988), and to the orientation of the head (Perrett et al., 1985). Perrett's group found that some cell types respond selectively to the left profile, others to the back of the head, etc.

Other studies have found that specific cells respond selectively to direction of gaze (Perrett et al., 1985; 1990). For example, Fig. 9.6 shows significantly more excitation from a cell in the STS of an observer when looking at eyes which are directed forward. (Other cells in the STS show the opposite pattern.) Perrett et al. (1985) found that 64% of cells in the STS responsive to the face or profile views of the head are also selective for the direction of gaze. Other evidence consistent with the notion of the STS being the brain basis of EDD comes from neuropsychology: Lesions in the STS produce an impairment in the ability to discriminate gaze direction by monkeys (Campbell et al., 1990), and by some patients with prosopagnosia (Campbell et al., 1990; Heywood & Cowey, 1991; Perrett et al., 1990). Perrett and his colleagues, in their most recent publications (Perrett et al., 1990; 1991), refer to the cells which respond to gaze-direction as cells responsive to the state of *attention* of the other individual, and which have the primary function of detecting if another individual is "looking at me" or not. In our model, such

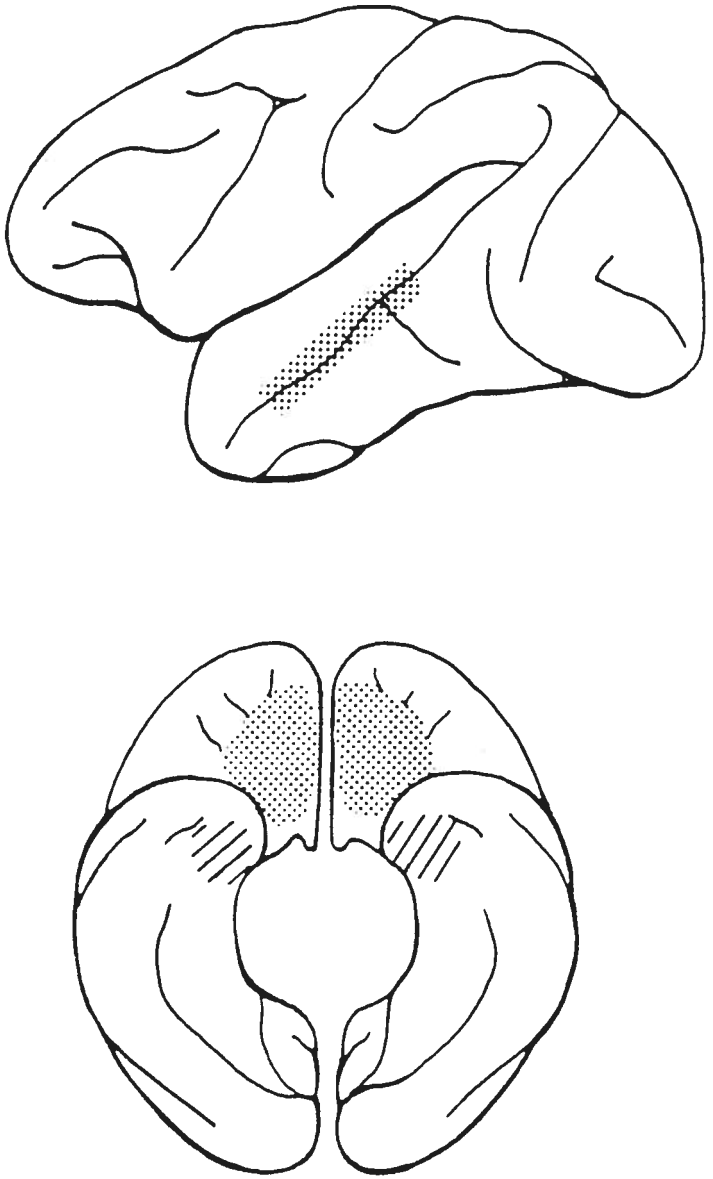

FIG. 9.5 The orbito-frontal cortex (OFC: lower illustration: basal view) and the superior temporal sulcus (STS: upper illustration: lateral view) in the macaque brain. The shaded regions show medial portions of the amygdala (from Brothers, 1990, reproduced with permission).

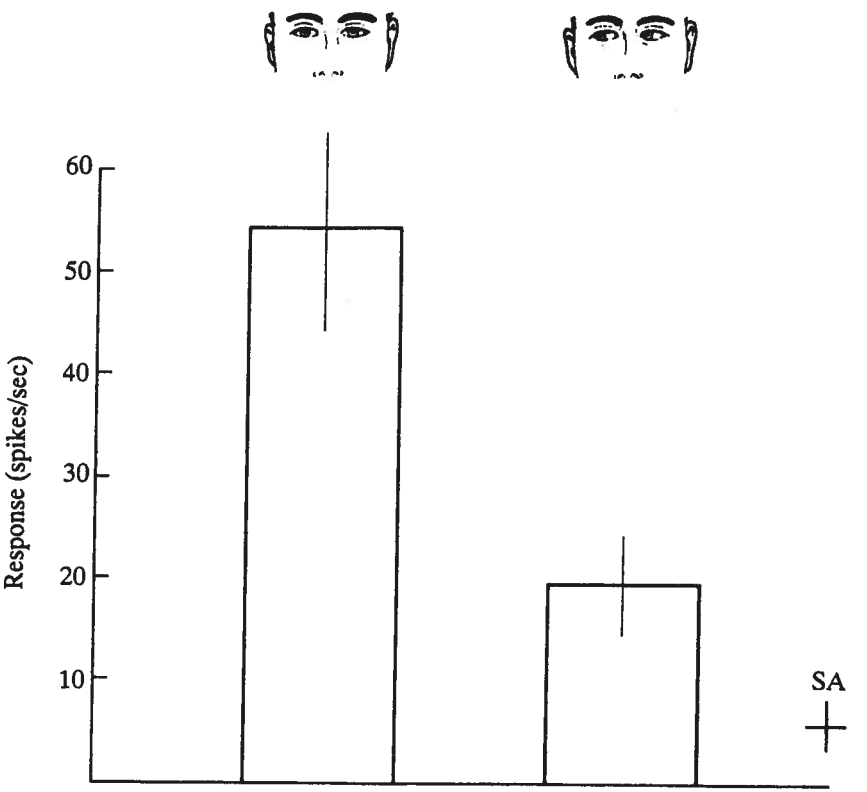

FIG. 9.6 Responsivity of cell M047 in the STS to eye-direction (adapted from Perrett et al., 1990, reproduced with permission).

cell assemblies serve EDD, and we speculate that since SAM works so closely with EDD, such cell assemblies in the STS may serve aspects of SAM's functioning as well. SAM's link with ToMM, however, requires the connection from STS to OFC. OFC receives direct inputs from rostral temporal cortices, including the depths of the STS (Barbas, 1988).

The OFC and ToMM

Damasio (1984) argues that the prefrontal region can be divided into three distinct functional sectors:

1. Superior mesial cortex (SMC: Brodmann Areas 6 and 24);
2. Dorsolateral cortex (DLC: Brodmann Areas, 4, 6, 8–11, and 43–47); and

3. Orbito-frontal cortex (OFC: Brodmann Areas 10–14, lying on the ventral surface of the frontal lobe).

These distinctions are proposed on the basis that lesions in these three regions produce very different symptoms:

1. Lesions in SMC produce a lack of drive or interest in interacting with the environment, social or nonsocial; and abnormalities in the expression of emotion (Damasio, 1984).
2. Lesions in DLC produce executive function deficits, such as perseveration and impulsivity (Milner, 1964; Shallice, 1988). For example, DLC lesions in juvenile monkeys impair the delayed response, whereas OFC lesions in juvenile monkeys produce no lasting impairment in this (Goldman, 1971; Goldman & Alexander, 1977). This suggests OFC and DLC have separate functions. OFC and DLC also have distinct anatomical connectivity (Fuster, 1989). Additional evidence for the role of DLC (especially Brodmann area 9: the principle sulcus) in delayed responding comes from Diamond and Goldman-Rakic's (1989) study of Piaget's AB task in human infants and rhesus monkeys.
3. Lesions in OFC produce *loss of social judgement* which, we argue, is due to loss of ToMM. Evidence for this is reviewed next.

Eslinger and Damasio (1985) reported the case of a patient (EVR) who underwent surgery for an OFC meningioma at the age of 35, with the consequent loss of all of the right OFC, part of the left OFC, and some damage to adjacent areas. Before his operation, the patient appears to have been socially normal: He held a senior position in a firm of accountants, was active in the local church, was married, and was a father of two. After his surgery, his family reported that he began to make decisions that suggested he had lost his social judgement: He went into business with a man of questionable reputation, against the advice of his friends and family; he then invested all of his savings into the new partnership, only (predictably to everyone else) to end up bankrupt. Similar difficulties led to a breakdown in his marriage. He married again, but this second marriage ended after just two years, again, quite predictably to everyone else.

Eslinger and Damasio's comment on EVR's decline is that his social judgement had become markedly impaired[8]. They describe him (1985, p.

[8] Of interest, he has also become markedly obsessional (e.g. refusing to part with useless possessions such as old telephone directories, taking two hours to wash and shave in the morning) and unable to form plans (taking several hours to decide which restaurant to go to, etc.). The interest of this association stems from the co-association of social impairment and "obsessional" behaviour in autism.

1737) as a case of "acquired sociopathy," to convey how post-operatively EVR had lost (1985, p. 1739) his "social sense." They note the similarity with Ackerley and Benton's (1948) case (JP) of a "primary social defect" following early, bilateral prefrontal lobe atrophy damage. Damasio, Tranel, and Damasio (1990) report another case with a similar social deficit from OFC lesions.

Although these neurological cases constitute at least suggestive hints for OFC involvement in ToMM, Price, Daffner, Stowe, and Mesulam (1990) report the only explicit test of a theory of mind deficit in two patients with acquired early frontal damage (at birth in one case, and at age four in the other). Both of their patients showed poor empathy in their social behaviour, and failed (what would today be seen as) a theory of mind test (a test of Flavell et al.'s, 1968). Further converging evidence comes from Kaczmarek (1984), who reported that damage to OFC produces more disturbance to narrative or discourse skills than does damage to DLC. This was mirrored in a report by Alexander, Benson, and Stuss (1989). Since discourse/pragmatic skills depend on ToMM (Baron-Cohen, 1988a; 1988b; Happé, 1993; Tager-Flusberg, 1993), this evidence is consistent with the OFC theory of ToMM.

On the basis of these strong clues for OFC involvement in ToMM, Baron-Cohen, Ring, et al. (in press) carried out a neuroimaging study of ToMM with normal adult subjects, using SPECT (Single Photon Emission Computed Tomography). Subjects were asked to detect the mental state terms in a word list that was played through headphones. We predicted significant OFC activation. A significant increase in cerebral blood flow in the right OFC was indeed found during this task, relative to a control (non-ToMM) task. No specific increase in activation occurred in an adjacent frontal lobe region (right frontal polar). To the extent that this mental state term detection task taps ToMM, this implies that at least some functions of ToMM require the right OFC.

Of the major three prefrontal regions (OFC, SMC, and DLC), OFC is the richest region in terms of limbic system connectivity (Damasio, 1984). The connections come from, among other centres, the amygdala (Nauta, 1962; 1964). Extending Brothers' (1990) model once again, we turn next to consider the role of the amygdala in a possible ToMM circuit.

The Amygdala: The Seat of ToMM's Affective Function?

The STS projects to the lateral nucleus of the amygdala (Aggleton, Burton, & Passingham, 1980). The OFC receives significant inputs from the medial structures of the amygdala (Aggleton, 1985; Amaral & Price, 1984; Porrino, Crane, & Goldman-Rakic, 1982; Van Hoesen, 1981). These observations

suggest an *STS-amygdala-OFC circuit* (Brothers, 1990). Like the STS, both the medial and lateral nuclei of the amygdala also have face-sensitive cells (Leonard, Rolls, Wilson, & Bayliss, 1985; Nakamura, Mikami, & Kubota, 1992). Since face-responsive units in the amygdala fire at a longer latency than do those in the STS, it is thought that they probably occur "downstream" from the STS (Leonard et al., 1985). Furthermore, like the STS, the amygdala also has cells sensitive to aspects of facial expression and gaze direction (Brothers, Ring, & Kling, 1990). Lesions in the amygdala alone can produce the Kluver-Bucy syndrome[9] (Schreiner & Kling, 1953). More recent accounts of the effects of amygdalectomy (Kling & Brothers, 1992) include not only social isolation, but also difficulties in social perception (e.g. an inability to respond selectively to different social ranks such as enemies versus friends; Kling, Lancaster, & Benitone, 1970), and a failure to attach emotional significance to sensory stimuli. The precise relationship between ToMM and the amygdala remains to be investigated, but certainly it seems plausible, on the basis of both its connectivity (to OFC and STS) and its importance in social and emotional perception, that it plays a role in supporting EDD, SAM, and ToMM.

The Relationship Between ToMM and Executive Function

We have already mentioned that OFC is implicated in ToMM, whereas DLC is implicated in executive function (planning and response inhibition). Additional evidence that ToMM and executive function are neurally very close to each other comes from other studies. First, children with autism show an impairment in both ToMM and executive function (Hughes & Russell, 1993; Ozonoff, Pennington, & Rogers, 1991; Prior & Hoffman, 1990; Rumsey & Hamburger, 1988). Secondly, measures of empathy and "cognitive flexibility" (an executive function index) tend to correlate in adults with acquired cerebral lesions (Grattan, Bloomer, Archambault, & Eslinger, 1990). For example, both EVR (Eslinger & Damasio, 1985) and JP (Ackerley & Benton, 1948) showed the association of loss of social sense and lack of cognitive flexibility. Grattan and Eslinger (1991) review a range of other similar cases. Such studies are consistent with the notion that the frontal lobes can be "fractionated" (Grattan & Eslinger, 1991, p. 299) and may possess modularity (Damasio, 1984; Pribam, 1987). However, since not

[9] In Kluver and Bucy's (1939) description of this syndrome, the symptoms occurred following bilateral removal of the temporal lobes in monkeys, and included (a) loss of the ability to discriminate animate and inanimate objects; (b) excessive oral exploration of objects; (c) a tendency to react to every visual stimulus; (d) a failure to vocalise towards other monkeys; (e) unusual movements; (f) changes in, or complete absence of, emotional reactions; and (g) hypersexuality.

all patients with executive function impairments have a ToMM impairment, it is possible that ToMM and the executive function are connected, but independent, systems. Anatomical connections from OFC to DLC have been demonstrated by Van Hoesen, Pandya, and Butters (1975). See also Ozonoff et al. (1991) for a discussion of the different possible relationships between ToMM and executive function.

The Neurobiology of Autism and the Mindreading System

We now wish to clarify the circuit-based model of the Mindreading System discussed earlier. To reiterate, Brothers (1990) proposed that a major neural circuit exists, linking OFC, STS, and the amygdala. We extend this idea as follows: first, the Mindreading System may be distributed across all three centres. Second, we propose that lesions in any one of these three centres, *or* in any one of the three connections between these three centres, could produce autism; and that the different subgroups of autism we discussed earlier would be determined by where in the circuit the lesion was.

Our theory of autism is most closely related to Frith's (1992), which itself is based on Brothers (1990); however, we elaborate this to incorporate the theory of four components of the Mindreading System. Our theory is also related to Damasio and Maurer's (1978), which also postulates damage to a frontal-temporal area, though in their case this is relatively wide (incorporating the supplementary motor area, cingulate gyrus, entorrhinal area, perirhinal area, parahippocampal gyrus, and subicular and presubicular regions). Our model is more specific than this, in focusing on the OFC and STS components. It is unclear as yet what the relationship is between our theory and those of Bachevalier (1991) and DeLong (1992), both of which propose an amygdalo-hippocampal dysfunction.

At present, although there are few consistent findings in the neurobiology of autism (Bailey, 1993; Bishop, 1992), there are some neuroimaging studies that are compatible with frontal lobe dysfunction in this group. For example, Piven et al. (1990) reported evidence of abnormal neuronal migration in the frontal lobes of three subjects with autism. Such abnormalities would be consistent with either executive function deficits (from a DLC lesion) or theory of mind impairments (from an OFC lesion), or both, as Ozonoff (in press) points out. Horwitz, Rumsey, Grady, and Rapoport's (1988) PET study found significantly lower correlations of metabolic activity between frontal and parietal regions in subjects with autism. Ozonoff speculates that this might reflect an abnormality in integrating the processing between these two lobes, hinting that frontal lesions might be leading to a relative over-reliance on parietal processing. L'Hermitte (1984) suggests that the frontal lobes may exert an inhibiting

control over the parietal lobes[10]. If this is correct, then Horwitz et al.'s results might indicate a dysfunction in this frontal inhibitory control over the parietal lobes. Further studies of this kind are needed.

There are also some studies that are compatible with a temporal lobe dysfunction in autism. Thus, EEG abnormalities from the temporal lobes have been reported (DeLong, 1978; Hauser, DeLong, & Rosman, 1975), as has enlargement of the temporal horn of the lateral ventricles (Hauser et al., 1975). Hetzler and Griffin (1981) give a good review of the arguments for temporal lobe involvement in autism, in particular noting some of the overlap between autism and the Kluver-Bucy syndrome, which as mentioned earlier can be produced by bilateral removal of the temporal lobes (Kluver & Bucy, 1939; see footnote 9). Finally, there are some studies that are compatible with an amygdala lesion in autism (Bauman & Kemper, 1985; 1988).

If autism was produced by an OFC lesion, what would one expect to see? The symptoms associated with OFC lesions consist of the following:

1. Impaired social judgement (in humans: Eslinger & Damasio, 1985).
2. Utilisation behaviour (in humans: L'Hermitte, 1984).
3. Pragmatic/discourse breakdown (in humans, of course: Kaczmarek, 1984).
4. Diminished aggression (in humans: Mateer & Williams, 1991)
5. Increased indifference (in monkeys: Meyer, 1972).
6. Decreased appreciation for dangerous situations (in humans: Case JB[11]: Mateer & Williams, 1991).
7. Hyper-olfactory exploration (in monkeys: Thorpe, Rolls, & Maddison, 1983).
8. Diminished response to pain (in humans: Goldman-Rakic, 1987).
9. Excessive activity (in monkeys: Ferrier, 1886).

All of these nine features are commonly seen in autism. In addition, damage to frontal regions adjacent to OFC would risk producing deficits in Broca's area (language production) and in DLC (executive function). An OFC lesion thus provides one way of accounting for the association between these impairments and ToMM deficits in autism.

[10] L'Hermitte suggests that loss of this inhibitory control may account for "utilisation behaviour" (the tendency to pick up and use objects, even when this is inappropriate). L'Hermitte reports this behaviour in some of his patients with frontal damage, and interestingly this behaviour has also been reported in children with autism (Fox & Tallis, in press).

[11] Note that JB not only received OFC damage, but also temporal damage. It is therefore unclear if this symptom is unique to the OFC syndrome.

Similarly, if autism was produced by a dysfunction in the amygdala, what would one expect to see? The effects of amygdalectomy were reviewed earlier, but are elaborated here:

1. Abnormalities in social perception (Kling et al., 1970).
2. Failure to attach emotional significance to stimuli (Kluver & Bucy, 1939; Weiskrantz, 1956).
3. Diminution in aggression and fear (Kling & Brothers, 1992).
4. Decrease in affiliative behaviour (Kling & Brothers, 1992).

Again, all of these four features are commonly seen in autism.

Finally, if the lesion was in the STS, this would not only impair EDD (and thus SAM), which are postulated to require the STS, but depending on the extent of temporal lobe damage, could risk receptive language (Wernicke's) areas being impaired. Again, this provides a possible account for the association between autism and language disorder. The pragmatics of language, as was mentioned earlier, are likely to be independent of Wernicke's area, and to require OFC involvement (Kaczmarek, 1984).

CONCLUSIONS

In this chapter we have elaborated the theory of the Mindreading System (Baron-Cohen, in press a; in press b) by focusing on both the neuropsychological and the neurobiological level. In particular, we have elaborated Brothers' (1990) model of the "social brain" to suggest that EDD, SAM, and ToMM lie in a three-node circuit between the STS, the OFC, and the amygdala, and that if this circuit is broken at any point, autism can be produced. We have not speculated about any possible localisation of ID, as there are currently no clues as to where this might be found. However, we have equated the neurobiological connections between the STS and the OFC with the psychological relation between EDD, SAM, and ToMM. The evidence for these claims has been summarised, though it is noted that many empirical gaps remain to be filled. Nevertheless, we hope that our model suggests new directions for research: It contains a number of testable predictions about the precursor status of SAM in triggering ToMM, and the proposed sites of neural dysfunction in autism.

ACKNOWLEDGEMENTS

We are indebted to Leslie Brothers, who helped us think through the neurobiological ideas in this paper, and guided us through the literature. Peter Mitchell and Charlie Lewis also gave us excellent editorial advice. The authors were supported by the Medical Research Council, the Mental Health Foundation, the Nuffield Foundation, and the Raymond Way Trust during the preparation of this work.

In Figure 9.4: KitKat, Smarties, and Polo are all registered trademarks, © Société des Produits Nestlé S.A.; and MilkyWay is a registered trademark, © Mars.

REFERENCES

Ackerley, S., & Benton, A. (1948). Report of a case of bilateral frontal lobe defect. *Association for Research in Nervous and Mental Diseases, 27*, 479–504.

Aggleton, J. (1985). A description of intra-amygdaloid connections in old world monkeys. *Experimental Brain Research, 57*, 390–399.

Aggleton, J., Burton, M., & Passingham, R. (1980). Cortical and subcortical afferents to the amygdala of the rhesus monkey (Macaca Mulatta). *Brain Research, 190*, 347–368.

Alexander, M., Benson, D., & Stuss, D. (1989). Frontal lobes and language. *Brain and Language, 37*, 656–659.

Amaral, D., & Price, J. (1984). Amygdalo-cortical projections in the monkey (Macaca fascicularis). *Journal of Comparative Neurology, 230*, 465–496.

Bachevalier, J. (1991). An animal model for childhood autism. In C. Tamminga & S. Schulz (Eds.), *Advances in neuropsychiatry and psychopharmacology, Vol. 1: Schizophrenia Research*. New York: Raven Press.

Bailey, A. (1993). The biology of autism. Editorial. *Psychological Medicine, 23*, 7–11.

Bakeman, R., & Adamson, L. (1984). Coordinating attention to people and objects in mother–infant and peer–infant interaction. *Child Development, 55*, 1278–1289.

Barbas, H. (1988). Anatomic organization of basoventral and mediodorsal visual recipient prefrontal regions in the rhesus monkey. *Journal of Comparative Neurology, 276*, 313–342.

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

Baron-Cohen, S. (1988b). Without a theory of mind one cannot participate in a conversation. *Cognition, 29*, 83–84.

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

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

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

Baron-Cohen, S. (1992). On modularity and development in autism: A reply to Burack. *Journal of Child Psychology and Psychiatry, 33*, 623–629.

Baron-Cohen, S. (1993). From attention-goal psychology to belief-desire psychology: The development of a theory of mind, and its dysfunction. In S. Baron-Cohen, H. Tager-Flusberg, & D.J. Cohen (Eds.), *Understanding other minds: Perspectives from autism*. Oxford: Oxford University Press.

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

Baron-Cohen, S. (in press b). *EDD and SAM:* Two cases for evolutionary psychology. In C. Moore & P. Dunham. (Eds.), *The role of joint attention in development*. Hillsdale, NJ: Lawrence Erlbaum Associates Inc.

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. (1993). *Are children with autism blind to the mentalistic significance of the eyes?* Unpublished manuscript, Institute of Psychiatry, University of London.

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*, 166–180.

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, behavioural and Intentional understanding of picture stories in autistic children. *British Journal of Developmental Psychology, 4*, 113–125.

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

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

Bates, E., Camaioni, L., & Volterra, L. (1975). The acquisition of performatives prior to speech. *Merrill-Palmer Quarterly, 21*, 205–226.

Bauman, M., & Kemper, T. (1985). Histoanatomic observation of the brain in early infantile autism. *Neurology, 35*, 866–874.

Bauman, M., & Kemper, T. (1988). Limbic and cerebellar abnormalities: Consistent findings in infantile autism. *Journal of Neuropathology and Experimental Neurology, 47*, 369.

Bayliss, G., Rolls, E., & Leonard, C. (1985). Selectivity between faces in the responses of a population of neurons in the cortex in the superior temporal sulcus of the monkey. *Brain Research, 342*, 91–102.

Bishop, D. (1992). Autism and frontal-limbic functions. *Journal of Child Psychology and Psychiatry, 34*, 279–294.

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

Brothers, L., & Ring, B. (1992). A neuroethological framework for the representation of minds. *Journal of Cognitive Neuroscience, 4*, 107–118.

Brothers, L., Ring, B., & Kling, A. (1990). Responses of neurons in the macaque amygdala to complex social stimuli. *Behavioural Brain Research, 41*, 199–213.

Bruce, C., Desimone, R., & Gross, C. (1981). Visual properties of neurones in a polysensory area in superior temporal sulcus of the macaque. *Journal of Neurophysiology, 46*, 369–384.

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

Campbell, R., Heywood, C., Cowey, A., Regard, M., & Landis, T. (1990). Sensitivity to eye gaze in prosopagnosic patients and monkeys with superior temporal sulcus ablation. *Neuropsychologia, 28*, 1123–1142.

Charman, T., & 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). *Understanding photos, models, and beliefs: A further test of the modularity thesis of theory of mind.* Unpublished manuscript, University College, London.

Damasio, A. (1984). The frontal lobes. In K. Heilman & E. Valenstein (Eds.), *Clinical neuropsychology* (Revised). New York: Oxford University Press.

Damasio, A., & Maurer, R. (1978). A neurologic model for childhood autism. *Archives of Neurology, 35*, 777–786.

Damasio, A., Tranel, D., & Damasio, H. (1990). Individuals with sociopathic behaviour caused by frontal lobe damage fail to respond autonomically to socially charged stimuli. *Behavioural Brain Research, 14*, 81–94.

DeLong, G. (1978). A neuropsychologic interpretation of infantile autism. In M. Rutter & E. Schopler (Eds.), *Autism: A reappraisal of concepts and treatment.* New York: Plenum Press.

DeLong, G. (1992). Autism, amnesia, hippocampus, and learning. *Neurobehavioural Review, 16*, 63–70.

Desimone, R., Albright, T., Gross, C., & Bruce, C. (1984). Stimulus selective properties of inferior temporal neurons in the macaque. *Journal of Neuroscience, 8*, 2051–2062.

Diamond, A., & Goldman-Rakic, P. (1989). Comparison of human infants and rhesus monkeys on Piaget's $A\overline{B}$ task: Evidence for dependence on dorsolateral prefrontal cortex. *Experimental Brain Research, 74*, 24–40.

Eslinger, P., & Damasio, A. (1985). Severe disturbance of higher cognition after bilateral frontal lobe ablation: Patient EVR. *Neurology, 35*, 1731–1741.

Ferrier, D. (1886). *Functions of the brain* (2nd edn.). London: Smith & Elder.

Flavell, J., Botkin, P., Fry, C., Wright, T., & Jarvis, P. (1968). *The development of role-taking and communication in children.* New York: Wiley.

Fodor, J. (1983). *The modularity of mind: An essay on faculty psychology.* Cambridge, Mass.: MIT Press.

Fox, N., & Tallis, F. (in press). Utilization behaviour in adults with autism: A preliminary investigation of frontal lobe functioning. *Journal of Clinical and Experimental Neuropsychology.*

Frith, C. (1992). *The cognitive neuropsychology of schizophrenia.* Hove: Lawrence Erlbaum Associates Ltd.

Fuster, J. (1989). *The prefrontal cortex: Anatomy, physiology, and neuropsychology of the frontal lobe.* New York: Raven Press.

Goldman, P. (1971). Functional development of the prefrontal cortex in early life and the problem of neuronal plasticity. *Experimental Neurology, 32*, 366–387.

Goldman, P., & Alexander, G. (1977). Maturation of prefrontal cortex in the monkey revealed by local reversible cryogenic depression. *Nature, 267*, 613–615.

Goldman-Rakic, P. (1987). Circuitry of primate prefrontal cortex and regulation of behaviour by representational memory. In V. Mountcastle, F. Plum, & S. Geiger (Eds.), *Handbook of physiology: Section 1: Nervous system: Part 1.* Bethesda, Maryland: American Physiological Society.

Gomez, J.C. (1991). Primates' reading of other minds. In A. Whiten (Ed.), *Natural theories of mind.* Oxford: Basil Blackwell.

Grattan, L., Bloomer, R., Archambault, F., & Eslinger, P. (1990). Cognitive and neural underpinnings of empathy. *The Clinical Neuropsychologist, 4*, 279.

Grattan, L., & Eslinger, P. (1991). Frontal lobe damage in children and adults: A comparative review. *Developmental Neuropsychology, 7*, 283–326.

Gross, C., Rocha-Miranda, C., & Bender, D. (1972). Visual properties of neurons in the inferotemporal cortex of the macaque. *Journal of Neurophysiology, 35*, 96–111.

Happé, F. (1993). Communicative competence and theory of mind: a test of relevance theory. *Cognition, 48*, 101–119.

Hauser, S., DeLong, G., & Rosman, N. (1975). Pneumographic findings in the infantile autism syndrome. *Brain, 98*, 667–688.

Hetzler, B., & Griffin, J. (1981). Infantile autism and the temporal lobe of the brain. *Journal of Autism and Developmental Disorders, 9*, 153–157.

Heywood, C., & Cowey, A. (1991). The role of the "face cell" area in the discrimination and recognition of faces in monkeys. *Philosophical Transactions of the Royal Society of London: Series B, 335*, 31–38.

Hobson, R.P. (1990). On acquiring knowledge about people and the capacity to pretend: Response to Leslie (1987). *Psychological Review, 97*, 114–121.

Horwitz, B., Rumsey, J., Grady, C., & Rapoport, S. (1988). The cerebral metabolic landscape in autism: Intercorrelations of regional glucose utilization. *Archives of Neurology, 45*, 749–755.

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.

Jackendoff, R. (1987). *Consciousness and the computational mind.* Cambridge, Mass.: MIT Press/Bradford Books.

Kaczmarek, B. (1984). Neurolinguistic analysis of verbal utterances in patients with focal lesions of frontal lobes. *Brain and Language, 21*, 52–58.

Kendrick, K., & Baldwin, B. (1987). Cells in the temporal cortex of conscious sheep can respond preferably to the sight of faces. *Science, 236*, 448–450.

Kling, A., & Brothers, L. (1992). The amygdala and social behavior. In J. Aggleton (Ed.), *Neurobiological aspects of emotion, memory, and mental dysfunction*. New York: Wiley-Liss Inc.

Kling, A., Lancaster, J., & Benitone, J. (1970). Amygdalectomy in the free ranging vervet. *Journal of Psychiatric Research, 7*, 191–199.

Kluver, H., & Bucy, P. (1939). Preliminary analysis of function of the temporal lobe in monkeys. *Archives of Neurology, 42*, 979–1000.

Langdell, T. (1978). Recognition of faces: An approach to the study of autism. *Journal of Child Psychology and Psychiatry, 19*, 225–238.

Leekam, S., Baron-Cohen, S., Perrett, D., Milders, M., & Brown, S. (1993). Eye-direction detection: A disassociation of geometric and joint attention skills in autism. Unpublished manuscript, Institute of Applied and Social Psychology, University of Kent, Canterbury.

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

Lempers, J., Flavell, E., & Flavell, J. (1977). The development in very young children of tacit knowledge concerning visual perception. *Genetic Psychology Monographs, 95*, 3–53.

Leonard, C., Rolls, E., Wilson, F., & Bayliss, G. (1985). Neurons in the amygdala of the monkey with responses selective for faces. *Behaviour and Brain Research, 15*, 159–176.

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

Leslie, A. (1991). The theory of mind impairment in autism: Evidence for a modular mechanism of development? In A. Whiten (Ed.), *Natural theories of mind*. Oxford: Basil Blackwell.

Leslie, A., German, T., & Happé, F. (1993). Even a theory-theory needs information processing: ToMM, an alternative theory-theory of the child's theory of mind. *Behavioral and Brain Sciences, 16*, 56–57.

Leslie, A., & Roth, D. (1993). What autism teaches us about metarepresentation. In S. Baron-Cohen, H. Tager-Flusberg, & D.J. Cohen (Eds.), *Understanding other minds: Perspectives from autism*. Oxford: Oxford University Press.

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

L'Hermitte, F. (1984). "Utilisation behaviour" and its relation to lesions of the frontal lobes. *Brain, 106*, 237–255.

Mateer, C., & Williams, D. (1991). Effects of frontal lobe injury in childhood. *Developmental Neuropsychology, 7*, 359–376.

Maurer, D., & Barrera, M. (1981). Infants' perception of natural and distorted arrangements of a schematic face. *Child Development, 52*, 196–202.

Maurer, R., & Damasio, A. (1982). Childhood autism from the point of view of behavioural neurology. *Journal of Autism and Developmental Disorders, 12*, 195–205.

Meyer, D. (1972). Some features of the dorsolateral frontal and inferotemporal syndromes in monkeys. *Acta Neurobiologica Experimenta, 32*, 235–260.

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

Nakamura, K., Mikami, A., & Kubota, K. (1992). Activity of single neurons in the monkey amygdala during performance of a visual discrimination task. *Journal of Neurophysiology, 67*, 1447–1463.

Nauta, W. (1962). Neural associations of the amygdaloid complex in the monkey. *Brain, 85*, 505–520.

Nauta, W. (1964). Some efferent connections of the prefrontal cortex in the monkey. In J. Warren & K. Akert (Eds.), *The frontal granula cortex and behavior*. New York: McGraw-Hill.

Ozonoff, S. (in press). Executive functions in autism. In E. Schopler & G. Mesibov (Eds.), *Learning and cognition in autism*. New York: Plenum Press.

Ozonoff, S., Pennington, B., & Rogers, S. (1991). Executive function deficits in high-functioning autistic individuals: Relationship to theory of mind. *Journal of Child Psychology and Psychiatry, 32*, 1081–1105.

Perner, J. (1993). The theory of mind deficit in autism: Rethinking the metarepresentation theory. In S. Baron-Cohen, H. Tager-Flusberg, & D.J. Cohen (Eds.), *Understanding other minds: Perspectives from autism*. Oxford: Oxford University Press.

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.

Perrett, D., Harries, M., Mistlin, A., Hietanen, J., Benson, P., Bevan, R., Thomas, S., Oram, M., Ortega, J., & Brierley, K. (1990). Social signals analysed at the single cell level: Someone is looking at me, something touched me, something moved! *International Journal of Comparative Psychology, 4*, 25–55.

Perrett, D., & Mistlin, A. (1990). Perception of facial characteristics by monkeys. In W. Stebbins & M. Berkley (Eds.), *Comparative perception, Vol II: Complex signals*. New York: John Wiley & Son.

Perrett, D., Oram, M., Harries, M., Bevan, R., Hietanen, J., Benson, P., & Thomas, S. (1991). Viewer-centred and object-centred codings of heads in the macaque temporal cortex. *Experimental Brain Research, 86*, 159–173.

Perrett, D., Rolls, E., & Caan, W. (1982). Visual neurones responsive to faces in the monkey temporal cortex. *Experimental Brain Research, 47*, 329–342.

Perrett, D., Smith, P., Potter, D., Mistlin, A., Head, A., Milner, A., & Jeeves, M. (1984). Neurones responsive to faces in the temporal cortex: Studies of functional organization, sensitivity to identity, and relation to perception. *Human Neurobiology, 3*, 197–208.

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.

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

Piven, J., Berthier, M., Starkstein, S., Nehme, E., Pearlson, G., & Folstein, S. (1990). Magnetic resonance imaging evidence for a defect of cerebral cortical development in autism. *American Journal of Psychiatry, 147*, 737–739.

Porrino, L., Crane, A., & Goldman-Rakic, P. (1982). Direct and indirect pathways from the amygdala to the frontal lobe in rhesus monkeys. *Journal of Comparative Neurology, 198*, 121–136.

Pratt, C., & Bryant, P. (1990). Young children understand that looking leads to knowing (so long as they are looking into a single barrel). *Child Development, 61*, 973–983.

Premack, D. (1990). Do infants have a theory of self-propelled objects? *Cognition, 36*, 1–16.

Premack, D., & Woodruff, G. (1978). Does the chimpanzee have a "theory of mind?" *Behaviour and Brain Sciences, 4*, 515–526.

Pribam, K. (1987). The subdivisions of the frontal cortex revisited. In E. Perceman (Ed.), *The frontal lobes revisited*. New York: IRBN Press.

Price, B., Daffner, K., Stowe, R., & Mesulam, M. (1990). The compartmental learning disabilities of early frontal lobe damage. *Brain, 113*, 1383–1393.

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

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.

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

Schreiner, L., & Kling, A. (1953). Behavioural changes following rhinencephalitic injury in the cat. *Journal of Neurophysiology, 16*, 643–659.

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

Sorce, J., Emde, R., Campos, J., & Klinnert, M. (1985). Maternal emotional signalling: Its effect on the visual cliff behaviour of one-year-olds. *Developmental Psychology, 21*, 195–200.

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.

Tantam, D. (1992). Characterizing the fundamental social handicap in autism. *Acta Paedopsychiatrica, 55*, 83–91.

Thorpe, S., Rolls, E., & Maddison, S. (1983). The orbito-frontal cortex: Neuronal activity in the behaving monkey. *Experimental Brain Research, 49*, 93–115.

Van Hoesen, G. (1981). The differential distribution, diversity, and sprouting of cortical projections to the amygdala in the rhesus monkey. In Y. Ben-Ari (Ed.), *The amygdaloid complex*. Amsterdam: Elsevier.

Van Hoesen, G., Pandya, D., & Butters, N. (1975). Some connections of the entorhinal (area 28) and perirhinal (area 37) cortices of the rhesus monkey, II. Frontal lobe afferants. *Brain Research, 95*, 25–38.

Volkmar, F., & Cohen, D. (1989). Disintegrative disorder or "late onset" autism? *Journal of Child Psychology and Psychiatry, 30*, 717–724.

Volkmar, F.R., Sparrow, S., Rende, R.D., & Cohen, D.J. (1989). Facial perception in autism. *Journal of Child Psychology and Psychiatry, 30*, 591–598.

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

Weiskrantz, L. (1956). Behavioural changes associated with ablation of the amygdaloid complex in monkeys. *Journal of Comparative Physiology and Psychology, 4*, 381–391.

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.

Wolff, P. (1963). Observations on the early development of smiling. In B. Foss (Ed.), *Determinants of infant behaviour, Vol. 2.* New York: Wiley.

Yamane, S., Kaji, S., & Kawano, K. (1988). What facial features activate face neurons in the inferotemporal cortex of the monkey: *Experimental Brain Research, 73*, 209–214.