Part III

Human disorders of social behaviour and cognition

8 Autism and the origins of social neuroscience

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"Social neuroscience" is something of a new phrase, and the editors of this volume are to be congratulated for collecting together the fragmented work, and thereby helping the creation of a new field. In their introduction to this book, they cover the history of this idea; but for me there are some themes—even lessons—worth highlighting.

Lessons from history

Nonsocial neuroscience

When I started in psychology some 20 years ago, there was almost no hint of social neuroscience. Cognitive neuroscience was alive and well, so this was not a reflection of a lack of activity in the wider field. Rather, it reflects that studies of the brain were for the most part *non*social. We had, for example, Blakemore's and Weizcrantz' classic studies of the visual system in kittens, monkeys, and humans to tell us which (nonsocial) features of the environment were perceived and how. We had Luria's and Shallice's classic studies of the (nonsocial) control of action to reveal not just a "central executive" for planning in the brain, but a syndrome of executive dysfunction. We had a wealth of other studies investigating conditions such as amnesia and agnosia to tell us how memory and knowledge of information *in general* worked in the brain. Even Wernicke's and Broca's classic studies of the language system in brain-damaged patients focused for the most part on the production and comprehension of words *in general* by the normal brain. But such aphasias were lexical or syntactic or semantic, and ignored the social aspects of communication: pragmatics.

Why was this? After all, cognitive neuroscientists then were not fools. They knew then, just we know now, that the human brain, and indeed most primate brains, exists *first and foremost* in a social world. Primates do not sit in solitary, solipsistic universes. So why did they treat the brain as if it had no special interest in the social world?

My guess is that there are (at least) two explanations for this. First, cognitive neuroscience followed a parsimonious approach of assuming that the brain is

a *general* information processor. Whether tacitly or explicitly, the assumption has been that the visual system, or the auditory system, or the memory systems, or the planning system, work on input of a general kind, where content plays little role. Of course, distinctions have been drawn, such as visual versus auditory memory, but, within a given system, it was held to not matter whether the visual input is a tree or a car: The search was to identify the general operating principles of the visual system. The same applies to memory. It matters little if we are studying memory of cars or of animals. We should still be able to identify the general operating principles of the memory system.

Such a content-free approach was laudable in its parsimony, as the danger otherwise was that neuroscience could have ended up positing a very large number of specialized circuits for different classes of information, and then the whole enterprise of understanding the basic laws of the brain would have been thrown off course. However, throughout this enterprise, there were always cracks appearing in this "brain-as-a-general-processor" theory. Just one example was the case of prosopagnosia, where some clinicians claimed that some patients could recognize any kind of object *except* faces. And the publication of Fodor's landmark book (1983) on *modularity* still stands as a major challenge to such a general theory.

The second possible explanation relates to cognitive neuroscientists as natural scientists. The nature of natural science is to try to isolate variables in a system under controlled conditions. The ultimate model for natural scientists is physics, and it is no surprise that even in the study of the human brain, the dominant approach has been to study how the brain responds to the manipulation of elementary features of the input. Is a vertical edge detected by the same assembly of neurons as a horizontal edge? Is a regular verb processed in the same way as an irregular verb? Again, such a focus on controllable, simple stimuli or features is laudable, since in this way one can make inferences about how the system works. If one were dealing with the complexity of the social world, how on earth could one begin to isolate what was causing what?

This is not intended as a criticism of cognitive neuroscience in adopting a general or a nonsocial approach. The natural science methodology has reaped great benefits and has reappropriated the study of the mind from the hands of psychoanalysts and social scientists, who ignored the brain and biology for decades. We have much to be grateful for. But there may be a set of parallels that emerge from a range of fields within psychology that show a similar disregard of the special nature of the social world. The lessons have been learned late.

Nonsocial psychology

The study of child development began with a disproportionate focus on the nonsocial aspects of cognition. Piaget's classic studies of object permanence

and what is really "folk physics" predominated until the 1980s, when Bruner (in Oxford) and his students reminded the field that children have minds that are trying to make sense of a social world, and not just a physical world. Indeed, the shift of focus to the pragmatics of communication (and away from traditional, Chomskian approaches to language acquisition), and the "discovery" of the developing child's "theory of mind" (Astington, Harris, & Olson, 1988; Wellman, 1990), owes a lot to Bruner's repeated concern that we were treating the child-as-scientist and ignoring the child-in-relationships (Bruner, 1983).

The same history unfolded in the field of intelligence. Almost all the early and classical IQ tests sought to assess the person's nonsocial IQ: David Wechsler's nonverbal subtests of object assembly or block design, or his verbal subtests of vocabulary or digit span, or Raven's matrices taught us an enormous amount about the predictive power of IQ (Raven, 1956; Wechsler, 1939), but virtually ignored what today is called "social intelligence" or "emotional intelligence" (Goleman, 1995). Equally, cognitive psychology focused in large part on the nonsocial aspects of cognition, with the new field of "social cognition" only coming in quite late in the twentieth century (Shantz, 1983).

If we look at the field of primatology, we can see a similar pattern. The attempt to understand the evolution of intelligence and the evolution of the brain focused on humans-as-tool-users and general problem solvers (or early hominids as "folk physicists") as the driving force behind the evolution of a larger, more powerful brain. It was rather late in the twentieth century when it was asked, "Does the chimpanzee have a theory of mind?" (Premack & Woodruff, 1978), and when it was proposed that the driving force behind the evolution of intelligence and the brain may have been the need to socially outwit competitors (the "Machiavellian intelligence hypothesis" (Whiten, 1997).

I lay out this brief and partial history because I think there may be lessons to be learnt. Naturally, there is a risk of painting the history as too black and white; too nonsocial when all along there was a streak of social neuroscience running through it. We know that Piaget's concept of "egocentrism" applied not only to the child's folk physics (to explain the child's errors in understanding conservation of mass, for example) but also to the child's folk psychology (to explain the child's errors in communication). And we know that Charles Gross's classic single-cell recording studies were identifying cell assemblies that fired not only in response to nonsocial aspects of the visual environment, but also in response to specifically social features such as hands and faces (Gross, Rocha-Miranda, & Bender, 1972). We know that Harry Harlow's, Robert Hinde's, and John Bowlby's classic studies of the attachment system in monkeys and humans progressed despite this history, and indeed ethology never lost sight of its social context (Bowlby, 1969). But these exceptions to the rule do not, I think, invalidate the broad picture I have painted. Rather, they were the seeds for the new field of social neuroscience.

From nonsocial to social accounts of autism

The study of autism has followed a similar history. The psychological theories of autism before the 1980s were for the most part nonsocial. The child's social difficulties were either attributed to a failure to generalize (Rimland, 1964) or were seen as secondary to a language disorder (Rutter, 1978), or thought to reflect a failure to process meaning (or semantics) (Hermelin & O'Connor, 1970), for example. For this reason, the proposal (by my colleagues and me in the 1980s) that the social and communication difficulties that are the hallmark of autism might reflect a *specific* deficit in an aspect of *social cognition* was treated as quite novel. Our idea was that there might be specific brain regions or neural circuits that underpinned social understanding, and ultimately social behaviour. We opened this area of investigation by asking, "Does the autistic child have a theory of mind?" (Baron-Cohen, Leslie, & Frith, 1985). A related investigation into emotion recognition in autism (Hobson, 1986) was also regarded as new and important. Later in this chapter, I summarize how this work has unfolded in the subsequent 20 years.

The social brain

My own theoretical and empirical work was greatly enriched by Leslie Brothers' important proposal of a network of neural regions that comprise the "social brain" (Brothers, 1990). She suggested that this included areas of the prefrontal cortex (orbital and medial areas particularly), the superior temporal sulcus, and the amygdala. Since the neurodevelopmental condition of autism involves deficits in what today I refer to as "empathizing" (Baron-Cohen, 2002), it is plausible that autism may be caused by an abnormality in one or more of these brain areas.

The idea that social understanding might be independent of general intelligence comes from three sources:

(1) There are individuals who are capable of considerable understanding of the nonsocial world (as in physics, maths, and engineering) but who readily admit to finding the social world confusing (Baron-Cohen, Wheelwright, Stone, & Rutherford, 1999; Sacks, 1994).
(2) The opposite type of individual also exists: people who have no difficulty interacting with the social world but who find nonsocial problem solving confusing (Karmiloff-Smith, Grant, Bellugi, & Baron-Cohen, 1995).
(3) Certain kinds of brain damage (as to the amygdala) can cause selective impairment in social judgement (Damasio, Tranel, & Damasio, 1990) without any necessary loss to general problem solving ability. Loss of social judgement can, of course, coexist with memory and executive dysfunction (Tranel & Hyman, 1990), but the functional double dissociation between empathizing and nonsocial aspects of intelligence suggests their neural independence.

In the remainder of this chapter, I review the evidence for the normal development of empathizing. I then review the literature suggesting autism involves an empathizing deficit. Finally, I end with a summary of the evidence for the role of the amygdala in empathy. The evidence for the social function of the orbito- and medial prefrontal cortex, and the superior temporal sulcus, is reviewed elsewhere (Baron-Cohen & Ring, 1994; Baron-Cohen, Ring, Moriarty, Shmitz, Costa, & Ell, 1994).

The empathizing theory of autism

Autism is diagnosed when a child or adult has abnormalities in a "triad" of behavioural domains: social development, communication, and repetitive behaviour/obsessive interests (American Psychiatric Association, 1994; ICD-10, 1994). Asperger syndrome (AS) was first described by Asperger (1944). The descriptions of the children he documented overlapped considerably with the accounts of childhood autism (Kanner, 1943). Little was published on AS in English until relatively recently (Frith, 1991; Wing, 1981). Current diagnostic practice recognizes people with AS as meeting the same criteria as for high-functioning autism (HFA), but with no history of language delay, and with no cognitive delay.

The mind-blindness theory of autism (Baron-Cohen, 1995), and its extension into empathizing theory (Baron-Cohen, 2002) proposes that in autism spectrum conditions there are deficits in the normal process of empathizing, relative to mental age. These deficits can occur by degrees. The term "empathizing" encompasses the following earlier terms: "theory of mind", "mind-reading", and taking the "intentional stance" (Dennett, 1987).

Empathizing involves two major elements: (1) the ability to attribute mental states to oneself and others, as a natural way to understand agents (Baron-Cohen, 1994; Leslie, 1995; Premack, 1990); (2) having an emotional reaction that is appropriate to the other person's mental state. In this sense, it includes what is normally meant by the term "theory of mind" (the attributional component), but it goes beyond this, to also include having some affective reaction (such as sympathy).

The first of these, the mental state attribution component, has been widely discussed in terms of being an evolved ability, the response of a cognitive system to a universe that can be broadly divided into two kinds of entities: those that do and those that do not possess intentionality (Brentano, 1970). The mental state attribution component is effectively judging whether this is the sort of entity that might possess intentionality. Intentionality is defined as the capacity of something to refer or point to things other than itself. A chair cannot point to anything. It just is. In contrast, a rabbit can "look" at a carrot, it can "want" the carrot, it can "think" that this is a carrot, etc. Essentially, agents have intentionality, but nonagents do not. This means that when we observe agents and nonagents move, we construe their motion as having different causes (Csibra, Gergely, Biro, Koos, & Brockbanck, 1999;

Gelman & Hirschfield, 1994). Agents can move by self-propulsion, which we naturally interpret as driven by their goals and desires, but nonagents cannot.

The second of these, the affective reaction component, is closer to what we ordinarily refer to by the word "empathy". Thus, we not only attribute a mental state to the agent in front of us (for example, the man is in pain), but we also react to his emotional state with an appropriate emotion ourselves (we feel sorry for him). Empathizing thus essentially allows us to make sense of the behaviour of other agents we are observing, predict what they might do next, and imagine how they might feel. And it allows us to feel connected to another agent's experience, and respond appropriately to them.

The normal development of empathizing

Empathizing develops from human infancy (Johnson, 2000). In the infancy period, it includes

- being able to judge whether something is an agent or not (Premack, 1990)
- being able to judge whether another agent is looking at you or not (Baron-Cohen & Goodhart, 1994b)
- being able to judge whether an agent is expressing a basic emotion (Ekman, 1992), and, if so, what type
- engaging in shared attention, as by following gaze or pointing gestures (Mundy & Crowson, 1997; Scaife & Bruner, 1975; Tomasello, 1988)
- showing concern or basic empathy at another's distress, or responding appropriately to another's basic emotional state (Yirmiya, Sigman, Kasari, & Mundy, 1992)
- being able to judge an agent's goal or basic intention (Premack, 1990).

Empathizing can be identified and studied from at least 12 months of age (Baron-Cohen, 1994; Premack, 1990). Thus, infants dishabituate to actions of "agents" who appear to violate goal-directedness (Gergely, Nadasdy, Gergely, & Biro, 1995; Rochat, Morgan, & Carpenter, 1997). They also expect agents to "emote" (express emotion), and expect this to be consistent across modalities (between face and voice) (Walker, 1982). They are also highly sensitive to where another person is looking, and by 14 months will strive to establish joint attention (Butterworth, 1991; Hood, Willen, & Driver, 1997; Scaife & Bruner, 1975). By 14 months, they also start to produce and understand pretence (Bates, Benigni, 1979; Leslie, Bretherton, Camaioni, & Volterra, 1987). By 18 months, they begin to show concern at the distress of others (Yirmiya et al., 1992). By 2 years old, they begin to use mental state words in their speech (Wellman & Bartsch, 1988).

Empathizing of course develops beyond early childhood, and continues to develop throughout the lifespan. These later developments include:

- attribution of the range of mental states to oneself and others, including pretence, deception, and belief (Leslie & Keeble, 1987)
- recognizing and responding appropriately to complex emotions, not just basic ones (Harris, Johnson, Hutton, Andrews, & Cooke, 1989).
- linking mental states to action, including language, and therefore understanding and producing pragmatically appropriate language (Tager-Flusberg, 1993)
- making sense of others' behaviour, predicting it, and even manipulating it (Whiten, 1991)
- judging what is appropriate in different social contexts, based on what others will think of our own behaviour
- communicating an empathic understanding of another mind.

Thus, by 3 years of age, children can understand relationships between mental states, such as that seeing leads to knowing (Pratt & Bryant, 1990). By 4 years, they can understand that people can hold false beliefs (Wimmer & Perner, 1983). By 5–6 years, they can understand that people can hold beliefs about beliefs (Perner & Wimmer, 1985). By 7 years, they begin to understand what not to say in order to avoid offending others (Baron-Cohen, O'Riordan, Jones, Stone, & Plaisted, 1999). With age, mental state attribution becomes increasingly more complex (Baron-Cohen, Joliffe, Mortimore, & Robertson, 1997; Happe, 1993). The little cross-cultural evidence that exists suggests a similar picture in very different cultures (Avis & Harris, 1991).

These developmental data have been interpreted in terms of an innate module being part of the infant cognitive architecture. This has been dubbed a theory of mind mechanism (ToMM) (Leslie, 1995). But, as we have suggested, empathizing also encompasses the skills that are involved in normal reciprocal social relationships (including intimate ones) and in sensitive communication. Empathizing is a narrowly defined domain, namely, *understanding and responding to people's minds*. Deficits in empathizing are referred to as degrees of mind-blindness.

Empathizing in autism spectrum conditions

Since the first test of mind-blindness in children with autism (Baron-Cohen et al., 1985), there have been more than 30 experimental tests. The vast majority of these have revealed profound impairments in the development of their empathizing ability. These are reviewed elsewhere (Baron-Cohen, 1995; Baron-Cohen, Tager-Flusberg, & Cohen, 1993) but include deficits in the following:

- joint attention (Baron-Cohen, 1989b)
- use of mental state terms in language (Tager-Flusberg, 1993)
- production and comprehension of pretence (Baron-Cohen, 1987; Wing & Gould, 1979)

- understanding that "seeing-leads-to-knowing" (Baron-Cohen & Goodhart, 1994b; Leslie & Frith, 1988)
- distinguishing mental from physical entities (Baron-Cohen, 1989b; Ozonoff, Pennington, & Rogers, 1990)
- making the appearance–reality distinction (Baron-Cohen, 1989a)
- understanding false belief (Baron-Cohen et al., 1985)
- understanding beliefs about beliefs (Baron-Cohen, 1989c)
- understanding complex emotions (Baron-Cohen, 1991)
- showing concern at another's pain (Yirmiya et al., 1992).

Some children and adults with AS show their empathizing deficits only on age-appropriate adult tests (Baron-Cohen, Jolliffe, Mortimore, & Robertson, 1997; Baron-Cohen, Wheelwright, Hill, Raste & Plumb, 1997; Baron-Cohen, Wheelwright, & Jolliffe, 2001), or on age-appropriate screening instruments such as the empathy quotient (EQ) (Baron-Cohen, Richler, Bisarya, Gurunathan, & Wheelwright, 2003).

Evidence for the contribution of the amygdala in the social brain and in autism

There are several important lines of evidence implicating the amygdala in primate social behaviour. Extensive reviews exist elsewhere (Kling & Brothers, 1992). We also know that the human amygdala is activated in humans when decoding signals of social importance, such as gaze, expression-recognition (especially of fearful faces), and body movements) (Baron-Cohen, Ring et al., 1999; Bonda, Petrides Ostry, & Evans, 1996; Kawashima et al., 1999; Morris et al., 1996; Whalen et al., 1998; Wicker, Michel, Henaff, & Decety, 1998). But there are six lines of evidence for an amygdala deficit in autism.

Post-mortem evidence

A neuroanatomical study of autism at post-mortem found microscopic pathology (in the form of increased cell density) in the amygdala, in the presence of normal amygdala volume (Bauman & Kemper, 1994; Rapin & Katzman, 1998).

An animal model of autism

The only animal model of autism involves ablation of the amygdala (in rhesus monkeys) (Bachevalier, 1991). There are obviously limits to any animal model of autism, given that the syndrome involves deficits in higher-order cognition, but Bachevalier makes the case that the effects of amygdala lesions in monkeys resemble some of the symptoms of autism. In particular, the Klüver–Bucy syndrome seems a fairly good animal model of autism (Hetzler & Griffin, 1981).

Similarities between autism and patients following amygdalotomy

Patients with amygdala lesions show impairments in social judgement (Adolphs, Tranel, Damasio, & Damasio, 1994; Young, Hellawell, De Wal, & Johnson, 1996) that have been likened to "acquired autism" (Stone, 2000). The age of onset of deficits in acquired versus idiopathic cases is likely to mean that the two syndromes also differ in many ways, too. Similarly, patients with autism tend to show a similar pattern of deficits to those seen in patients with amygdala lesions (Adolphs, Sears, & Piven, 2001).

The effects of temporal lobe tubers

In cases of tuberous sclerosis, autistic comorbidity is determined by hamartomata in the temporal lobe (Bolton & Griffiths, 1997)[1].

Structural neuroimaging

A structural magnetic resonance imaging study of autism reported reduced amygdala volume (Abell et al., 1999).

Functional neuroimaging

In single photon emission computed tomography (SPECT), patients with autism spectrum conditions show significant reductions in temporal lobe blood flow. This is not simply an effect of temporal lobe epilepsy (Gillberg, Bjure, Uvebrant, Vestergren, & Gillberg, 1993). In our earlier functional magnetic resonance imaging (fMRI) study, we found that adults with high functioning autism (HFA) or AS showed significantly less amygdala activation during an empathizing task (the "reading the mind in the eyes" task), than normal controls (Baron-Cohen et al., 1999). Adults with HFA or AS, with intelligence in the normal range, show deficits on this task (Baron-Cohen et al., 1997; Baron-Cohen et al., 2001), as do parents of children with autism/AS (Baron-Cohen & Hammer, 1997). Children with Williams syndrome are not impaired on this test, despite their general retardation (Tager-Flusberg, Boshart, & Baron-Cohen, 1998).

Other brain areas that might be abnormal in autism

While this chapter highlights the role that amygdala abnormality plays in autism, we do not suggest that this is the only abnormal neural region. For

1 We emphasize the amygdala theory of autism, though some of the lines of evidence cited here implicate temporal lobe structures, which include the amygdala but also other adjacent mesiotemporal areas. It remains for future work to establish the specificity of an amygdala deficit in autism.

example, the case has been made for anomalous functioning in the cerebellum (Courchesne et al., 1994), hippocampal formation (De Long, 1992), medial frontal cortex (Happe et al., 1996), and frontolimbic connections (Bishop, 1993) in autism. Reduced neuron size and increased cell-packing density has also been found in the limbic system, specifically the hippocampus, subiculum, entorhinal cortex, amygdala, mamillary bodies, anterior cingulate, and septum in autism (Bauman & Kemper, 1988, 1994; Bauman & Kempner, 1985, 1986; Raymond, Bauman, & Kemper, 1996). A full review of neuroimaging of autism may be found elsewhere (Filipek, 1999). Here, we instead follow a line of argument begun by other authors emphasizing an amygdala theory of autism (Bachevalier, 1994; Baron-Cohen et al., 2000; Bauman & Kemper, 1988; Hetzler & Griffin, 1981). In the closing section of this chapter, we briefly turn from brain regions to the neurochemistry, and particularly the neuroendocrinology, of social development.

Foetal testosterone (FT) and brain development

Foetal testosterone (FT) acts on the developing brain to influence cerebral lateralization (Kimura, 1999; Wilson, Foster, Kronenberg, & Larsen, 1998). Evidence for this derives from both animal studies (Harris & Levine, 1962; Arnold & Gorski, 1984; Williams, Barnett, & Meck, 1990), and the effects of abnormal hormonal environments during human pregnancy, such as congenital adrenal hyperplasia or synthetic hormone injections (Collaer & Hines, 1995; Hines & Shipley, 1984).

There is reason to believe that sex hormones are inversely related to social and language development (Geschwind & Galaburda, 1985a, 1985b, 1985c, 1985). Sex differences (female superiority) have been found in studies of normal language and social development (Baron-Cohen, 2002; Connellan, Baron-Cohen, Wheelwright, Ba'tki, & Ahluwalia, 2001; Hyde & Linn, 1988; Maccoby & Jacklin, 1974), and recent studies suggest an inverse correlation between levels of FT as measured in amniotic fluid, and both amount of eye contact measured at 12 months old (Lutchmaya, Baron-Cohen, & Raggatt, 2002a) and vocabulary size at 18 and 24 months old (Lutchmaya et al., 2002b). Geschwind's theory was that FT might accelerate the growth of the right hemisphere at the expense of the left, which is usually dominant in language functions and which may also be of some significance for empathy.

Summary and future work

Social neuroscience is now an important part of cognitive neuroscience. Studies of autism have contributed to this new field, and the literature reviewed earlier hints at the validity of an amygdala theory of autism. Future studies will be needed to test this more extensively. Secondly, future research will need to specify in greater detail which of the 13 nuclei in the amygdala are intact in autism, and which are impaired. Finally, the intriguing possibility

that FT mediates empathy through testosterone receptors in the amygdala and other parts of the "social brain" will be an important hypothesis to test, when methods become available.

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