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What is special about social cognition?

Ralph Adolphs

California Institute of Technology

Address:

Ralph Adolphs

HSS 228-77

Caltech, Pasadena, CA 91125

radolphs@hss.caltech.edu

(626)-395-4486

"In the distant future I see open fields for far more important researches. Psychology will be based on a new foundation, that of the necessary acquirement of each mental power and capacity by gradation." Charles Darwin, On the Origin of Species.

When Charles Darwin wrote the above quote, at the end of his famous book, "On the Origin of Species," (Darwin, 1859) he was presciently advocating the extension of evolutionary theory as applied to morphology—the topic of his book—to the evolution of the mind. Sociobiology and Evolutionary Psychology have indeed taken up the baton, albeit with agendas that not even Darwin could have anticipated. The detailed claims of these disciplines remain debated, but their central question remains as clear and urgent as ever. What distinguishes the human mind? What makes us essentially human?

It is easy to construe these questions so vaguely as to admit of little progress towards an answer. However, it is equally easy to point out some specific facts that provide purchase. Take the observation by Richard Passingham (1982) that, "Our species is unique because, in only 35,000 years or so, we have revolutionized the face of the earth." That observation is patently correct. Homo sapiens has changed the face of the earth, and constructed societies on a scale never before seen on this planet. In so doing, we have also become uniquely capable of destroying the majority of species, including ourselves. And all of that happened on a timescale far too rapid to have incorporated evolution by natural selection as Darwin had in mind. So how has this dramatic change been possible?

The answer, of course, lies with a different form of evolution than the one based on genes: the evolution of culture. Comparative psychologists and anthropologists have long pointed out the dramatically accelerated accumulation of information that is possible with cultural, rather than genetic transmission. Unsurprisingly, the claim has been made that culture is indeed what distinguishes humans from all other species. Thus we find Michael Tomasello, in his book The Cultural Origins of Human Cognition (Tomasello, 1999) writing,

"The basic fact is thus that human beings are able to pool their cognitive resources in ways that other species are not…made possible by a single very special form of social cognition, namely, the ability of individual organisms to understand conspecifics as beings like themselves who have intentional and mental lives like their own."

While the claim that culture is unique to humans has recently been challenged by studies in apes (Whiten et al., 1999), the idea that human social cognition is what makes the human mind special remains a popular view. One idea has been that intra- or inter-group competition amongst early hominids fueled a need to anticipate and predict others' behavior. Both tactical deception (Whiten & Byrne, 1997) and social cooperativity are behavioral consequences of such a mechanism, and while precursors of both are found in other animals, they are not found remotely to the same extent as in humans. Could these mechanisms have fueled the expansion of the human brain and our distinctive cognitive abilities (Dunbar, 1998)?

Tomasello's conjecture certainly has plausibility. We humans think of other people as having minds, having experiences, feelings, emotions, beliefs and intentions, and as having a view on the world as we ourselves do. Broadly, this capacity has been dubbed "Theory of Mind", and there has been continuous debate regarding whether or not nonhuman primates

might possess something similar (Povinelli & Vonk, 2003; Premack & Woodruff, 1978; Tomasello, Call, & Hare, 2003) --an issue, interestingly, on which Tomasello himself has recently changed his mind). Despite its prima facie plausibility, this idea still leaves unresolved the question of this Chapter. Granted that our social cognitive abilities are special: are they the reason our minds are special, or are there more general capacities to which social cognition is derivative?

There is no shortage of examples of the latter possibility. Our ability to conceive of other minds could, for example, be part of a more general ability to flexibly adopt other points of view. Thus, Suddendorf and Corballis have put forth the "mental time travel hypothesis" (Suddendorf & Corballis, 1997), the idea that only humans are capable of re-experiencing the past and imagining the future. Conceiving of ourselves from alternate points of view, conceiving of others as like ourselves—all these are arguably merely specific instances of much more general metacognitive abilities. It is not clear that they are uniquely social in any sense, nor that they evolved solely as a consequence of social pressures.

I. Social Cognition and Modularity

Social cognition encompasses the processing of information relevant to guide social behavior, our interactions with other people. Of course, in some sense nearly all information is "relevant" for social behavior, making social cognition so defined a term too general to be useful. Researchers have generally restricted the term to that information directly perceived

from, or inferred about, other people. A further restriction has focused the domain on those aspects of cognition closely linked to emotion.

Social cognition, then, concerns the perception, attention to, memory for, and thinking about other people, and in a way that involves emotional and/or motivational processing. Examples include the processing of other people's faces and voices, of judging their personality, predicting their likely behavior, and planning our own interactions with them. In one typical example, it begins with the sensory processing of social information (visual perception of someone's face or body posture, for example), proceeds to the formation of inferences and judgments about the social meaning and significance of this information, based on innate and acquired memories, current context, and future goals and plans, and culminates in the modulation of essentially all aspects of cognition and behavior. While pervasive in the sense that it has the potential to influence essentially all aspects of cognition and behavior, social cognition draws upon a circumscribed domain of information.

The degree to and manner in which different species engage in social interaction varies considerably. Even among primates, a highly social group of mammals, there are large differences ranging from the essentially solitary existence of orang-utans to the close alliances and groups seen in chimpanzees, to very large-scale human societies. No less variable is the occurrence of social cognition within an individual: we regularly engage in social interactions; but we also regularly drive our car, memorize phone numbers, and plan the decorating of our house, all of which are complex and uniquely human activities yet none of which is essentially social in nature.

A natural question then arises regarding how social cognition fits into cognitive processing more generally construed. Is social cognition a particular type of cognition? If so,

what distinguishes it, and how does it interact with non-social cognitive processes? In thinking about this question, people are often driven to one of two extremes: that social cognition is a way of processing information that is entirely distinct from non-social information processing; or that social cognition is exactly like cognition in general, only applied to the domain of social stimuli.

The above distinction has been formalized in the concept of modularity. First detailed by the philosopher Jerry Fodor (Fodor, 1983), modularity has come to mean several things, and its current usage differs considerably from earlier ones (Coltheart, 1999; Cowie & Woodward, 2004). Without going into detail, we can itemize the following attributes of modular processes, as the term will be used here. Modular processes are

--to some extent specialized for processing certain kinds of information (that is, they are domain-specific, rather than domain-general)

--evolved towards the above function

--best understood, from the view of cognitive psychology, as a distinct class of processes

--best understood, from the view of neuroscience, as relying on a distinct set of brain structures

--as a consequence of the prior two properties, can be disproportionately impaired following psychiatric or neurological disease

--show computational features indicating that they are specialized

In conjunction with the above, incomplete, list of attributes of modular processes, there are also certain properties that we think it is important NOT to ascribe to them. Modular processes

--do not necessarily have a larger innate component than any other set of processes

--do not necessarily depend less on experience and development than any other set of processes

--are not limited in their application to the domain of information for which they are specialized or for which they evolved (teleologically speaking)

--depending on the details, may be quite powerful and efficient in processing kinds of information for which they are not specialized, insofar as those make suitable computational demands (that is, they may be pre-adapted for certain kinds of information processing)

--may interact extensively with other (non-social) cognitive processing (that is, they are not informationally encapsulated, to use Fodor's original term)

While it seems clear that social cognition is not modular in a strong sense, if all of social cognition is considered together, there are certainly components of social cognition that appear strongly modular. Pheromonal signaling via the vomeronasal system (a pathway that prominently includes the amygdala), for instance, would appear paradigmatically modular. Language appears highly modular. All of these are components, but the entire collection of social cognitive abilities fails to be modular for the simple reason that it draws on processes that are too "central"—what Fodor termed "horizontal". That is, pheromone reception and speech perception might both be modular insofar as they are inputs to social cognition; it is once we actually start judging and reasoning about such social information that the modularity breaks down.

There is, of course, a famous example that purports to resist this negative conclusion, and to claim support the Evolutionary Psychology's controversial idea that the mind is massively modular through and through. That example is the Wason selection task (Barkow, Cosmides,

& Tooby, 1992). In this task, content effects have been found, such that people reason differently depending on the nature of the material they are reasoning about, even if the logical structure remains the same. In particular, there is the idea that we reason about social content in ways that reflects modules for detecting the violation of social contracts. The mechanism has even been linked to specific regions of the brain (Stone, Cosmides, Tooby, Kroll, & Knight, 2002). There remains debate about this particular example; but, the Wason selection task aside, there are plenty of counterexamples where certain quite circumscribed aspects of social cognition turn out, upon further investigation, to lose the domain-specificity that they initially appeared to support. We will review some of this evidence in detail below.

2. The Neuroscience of Social Cognition

Two good examples of current study in cognitive neuroscience are the social cognitive functions of the prefrontal cortex, and of the amygdala. Both structures can be disproportionately activated in certain social tasks in functional imaging studies, and lesions in both can results in disproportionate impairments in social behavior. Nonetheless, there is also good evidence that both structures participate prominently in processes that have nothing to do with social cognition as such. Are they examples of systems that evolved for social cognition that have been co-opted for other functions? Or are they examples of domain-general systems on which social cognition also happens to draw? A similar question arises in regard to simulation, a mechanism much studied in relation to how we construe others, how we pick up

their emotions, and how we generate empathy. Again, the basic mechanism appears to be derivative to more general aspects of motor control and imagery.

a. The Prefrontal Cortex.

Let us begin with the prefrontal cortex. At the purely structural level, there is already some evidence that certain features of the human prefrontal cortex distinguish it from that of any other animal. For instance, particular sectors, such as frontal polar cortex (Broadman's area 10) are disproportionately larger in humans than in any other ape (Semendeferi, Armstrong, Schleicher, Zilles, & Van Hoesen, 2001). It is thought that this region of the brain subserves long-term planning, metacognition, self-relevant processing—in short, precisely those competences that Tomasello, Corballis, and others had claimed unique to humans (see above). The prefrontal cortex of humans, specifically anterior cingulate and frontoinsular cortex (lateral orbitofrontal cortex) contains morphologically specialized cells, so-called spindle cells, that are found only to a much lesser degree in other apes, and not at all in other primates (Allman, 2002; Nimchinsky et al., 1999). One conjecture is that these cells serve rapid signaling of social information during error detection.

Lesions of the prefrontal cortex have famously been known to impair social functioning ever since the classic case of Phineas Gage (Damasio, 1994). Patients with damage centered in ventral and medial sectors of the prefrontal cortex have disproportionate difficulty in their social behavior—their social decision-making is poor, their social relationships with others are severely compromised, they fail to detect social faux pas, and there is the suggestion that they may have a specific impairment on social or emotional "EQ" with sparing of the standard

cognitive “IQ” (Bar-On, Tranel, Denburg, & Bechara, 2003)cf. Bechara this volume). In trying to get a handle on this issue, perhaps one of the most informative studies is the one by Barrash et al., which asked relatives of such patients to fill out a detailed questionnaire of changes in personality and behavior after sustaining such a lesion (Barrash, Tranel, & Anderson, 2000). Several key attributes achieved statistical significance in being endorsed as different after damage to ventromedial prefrontal cortex, including the following list:

--lack of insight

--lack of initiative

--social inappropriateness

--poor judgment

--lack of persistence

--indecisiveness

--blunted emotional experience

--apathy

--inappropriate affect

--lack of planning

Are all of these social? What might they all share in common? The claim has been made that this constellation of impairments all are derivative to impaired emotional processing, and perhaps especially impaired processing of social emotions. On the other hand, it is conceivable that more general defects, such as a failure in response inhibition or a global lack of motivation, could underlie the social impairments.

The evidence is more confusing yet when we consider functional imaging studies. Literally thousands of paradigms have been found to activate regions of prefrontal cortex. Some of these paradigms are specifically social, others are not. Two regions of considerable recent interest are the insula, and the anterior cingulate cortex. The insula has been proposed to be specialized in primates for the explicit representation of interoceptive information, the substrate of conscious feelings (Craig, 2002); the anterior cingulate for error detection and response monitoring (Paus, 2001). In each case, recent social experiments have resulted in activation in these structures, during empathy or social rejection, for instance. Yet the reasons for the activation in the social experiments have been presumed to be the utilization by social cognition of more general cognitive processes. The anterior cingulate cortex is activated by the pain of social rejection (Eisenberger, Lieberman, & Williams, 2003), or by observing someone else in pain (Singer et al., 2004), yet plays a general role in pain processing—and in fact, a very general role in detecting any salient stimuli that might require interruption of ongoing processing, of which pain may be an instance. The insula is activated by empathy for others (Singer et al., 2004), by observing others express disgust, as well as by pain, disgust, and other interoceptive information in oneself.

b. The Amygdala

The second structure mentioned, the amygdala, presents us with a parallel story. Back in the 1930s, Kluver and Bucy (Kluver & Bucy, 1937, 1939) pointed to the impairments in social behavior in primates with complete amygdala lesions, an emphasis that has continued

in several studies of the consequences of amygdala lesions on behavior in the real world. Kling and others found that monkeys with bilateral amygdala lesions were so severely affected in their social behavior that they often died if left in the wild (Dicks, Myers, & Kling, 1969; Kling & Brothers, 1992). More recent studies by Amaral and others have made more focal amygdala lesions, and have found impairments in social behavior that are more subtle, as well as impairments in non-social behaviors such as approach tendencies towards novel objects (Emery et al., 2001). These results are broadly consistent with the data from humans.

Several lesion studies (Adolphs, Tranel, Damasio, & Damasio, 1994; Anderson & Phelps, 2000; Anderson, Spencer, Fulbright, & Phelps, 2000; Calder et al., 1996; Young, Hellawell, Van de Wal, & Johnson, 1996), complemented by functional imaging studies (Breiter et al., 1996; Morris et al., 1996; Whalen et al., 2001), all demonstrate that the human amygdala is critical for normal judgments about the internal states of others from viewing pictures of their facial expressions. Detailed analyses of some of the lesion studies suggested that the impairment was disproportionately severe for the recognition of expressions of fear, a conclusion supported also by some functional imaging studies.

The initial impression that amygdala damage results in a disproportionate impairment in recognition of fear needs to be tempered by more recent studies. A first flag was raised by many other studies of impairments in facial emotion recognition in a large number of pathological cases, including psychiatric illnesses and brain damage of various kinds: a common pattern across all the studies is the finding that fear is typically the emotion whose recognition is the most severely impaired, and it is also the most difficult emotion to recognize on some tasks for normal individuals (cf. (Rapcsak et al., 2000)). A second flag was raised by finding that some patients with complete bilateral amygdala damage appeared to perform

normally on a task on which other patients with such damage were impaired (Adolphs et al., 1999; Hamann et al., 1996; Schmolck & Squire, 2001). A third and related flag was the finding that, when the data from several patients with bilateral amygdala damage were put together and analyzed in detail, the pattern that emerged was that they were indeed all impaired in their ability to make normal judgments regarding the basic emotions shown in facial expressions; but they were not all impaired in the same way, on the same tasks, or on the same emotions (Adolphs, 1999; Adolphs et al., 1999; Schmolck & Squire, 2001).

The amygdala's role is not limited to making judgments about basic emotions, but includes a role in making social judgments. This fact was already suggested by earlier studies in nonhuman primates, such as (Kling & Brothers, 1992; Kluver & Bucy, 1937; Rosvold, Mirsky, & Pribram, 1954), which demonstrated impaired social behavior following amygdala damage. They have been corroborated in recent times by studies in monkeys with more selective amygdala lesions, and by using more sophisticated ways of assessing social behavior (Emery & Amaral, 1999; Emery et al., 2001), and they have been shown now also in humans. Building on these findings, some recent studies suggest a general role for the amygdala in so-called "theory of mind" abilities: the collection of abilities whereby we attribute internal mental states, intentions, desires, and emotions to other people (Baron-Cohen et al., 2000; Fine, Lumsden, & Blair, 2001). Three sets of studies from our laboratory corroborate the view that the amygdala is important for generating social attributions: studies of judgments of trustworthiness, studies of social attributions to visual motion displays, and studies of the recognition of social emotions from faces and eyes.

In one study, we asked subjects to judge how much they would trust, or how much they would want to approach, an unfamiliar person (Adolphs, Tranel, & Damasio, 1998). We found

that three subjects with bilateral damage to the amygdala were specifically impaired in their ability to judge the untrustworthiness and unapproachability of these stimuli; they performed normally when judging people who looked very trustworthy and approachable. The performances given by the subjects with bilateral amygdala damage became progressively more impaired, the more untrustworthy or unapproachable the face was normally judged to be. These findings have now been corroborated also using functional imaging in normal individuals (Winston, Strange, O'Doherty, & Dolan, 2002). As expected, when normal individuals are shown faces of people judged to look untrustworthy, they show an increased amygdala activation, compared to the activation seen when they look at people judged to look trustworthy. Moreover, this pattern of activation was obtained regardless of whether the subjects in the scanner were making explicit judgments about trustworthiness or making an unrelated judgment. Thus, the amygdala's role in evaluating the trustworthiness of unfamiliar people, as its role in making other social judgments, may be fairly automatic, rapid, and obligatory.

An even broader role for the amygdala in making social attributions comes from a recent study in which subjects were shown stimuli developed by the social psychologist Fritz Heider, who designed short video clips depicting geometric shapes moving on a white background. Although the only cue available is visual motion (the stimuli don't otherwise look social), normal subjects immediately make social attributions to such stimuli (Heider & Simmel, 1944). The stimuli are interpreted as having intentions, emotions, and personalities, attributions assigned to them by the viewer in order to provide a compact, coherent, and relevant social description of the stimulus. It is in fact easier, and subjects normally cannot help but to see the stimulus in social terms. By contrast, a subject with selective bilateral amygdala damage (SM)

gave a spontaneous description of the stimulus in purely geometric terms that lacked any social attribution (Heberlein & Adolphs, 2004). The finding was particularly striking because giving such a geometric description is normally more difficult than giving the social description. For our purposes, the finding is interesting because such a complex social judgment is at least not obviously based on simpler motivational processes.

The above studies demonstrate that the human amygdala is important not only for processing information about basic emotions, but also about complex social states, intentions, and relationships. But what is the evidence that the amygdala might be disproportionately important for processing explicitly social information? It is difficult to know how to go about answering this question, but a recent study (Adolphs, Tranel, & Baron-Cohen, 2002) provides one approach. There are certain classes of emotional states, the so-called social emotions, that require knowledge about complex social relationships and that do not make sense independently of a social context. At least some "basic" emotions, such as fear or disgust, certainly apply to non-social situations; but "social" emotions, such as jealousy, pride, or embarrassment, necessarily require a social context and require a concept of a social self that is situated within a social group. In one recent study, two subjects with complete bilateral amygdala damage, as well as 30 with unilateral amygdala damage, were found to be impaired in their ability recognize social emotions from the face, as well as from just the eye region of the face. A further analysis demonstrated that the impairment was disproportionately severe for recognizing social emotions, as compared to basic emotions, a dissociation that held up either for faces as a whole, or for just the eye region of the face.

But does the amygdala play a disproportionately important role in processing social stimuli, as opposed to non-social stimuli? In many mammals, such as rodents, it seems likely that the

answer to this last question is negative. There is really no good evidence to support the idea that the rat amygdala, for instance, is more important to guide responses to other rats than it is to guide responses to food, water, or electric shock and the non-social stimuli with which these reinforcers can be associated. However, in primates, and especially in humans, the question is more intriguing. As reviewed above, humans with bilateral amygdala damage do have pronounced impairments in making social judgments, and in some cases there is evidence that their impairments in making complex social judgments are worse and more pervasive than their impairments in making simpler emotional judgments. It is thus a reasonable hypothesis, albeit one that needs to be made more precise, that throughout phylogeny the role of the amygdala in processing specifically social stimuli has become progressively more elaborated, in tandem with the elaboration of social behavior. Perhaps the human amygdala does not play a role in social cognition merely and derivatively in virtue of its role in mediating general (non domain-specific) motivational processes; perhaps the human amygdala is relatively specialized to process those stimuli (arguably especially visual stimuli) that have an explicitly social significance. (Of course, insofar as all social stimuli are motivational, this is still consistent with the notion that the amygdala processes the motivational value of social stimuli, as opposed to some non-motivational property). There are some preliminary arguments both in favor of and against this view.

An argument apparently in favor of the view comes from a recent study by Barton and Aggleton (Barton & Aggleton, 2000), who found a three-way allometric correlation between the volume of the basolateral amygdala (relative to the size of the rest of the brain), the size of visual cortex, and the size and complexity of social groups. A different argument apparently against the idea comes from the observation that monkeys with selective bilateral amygdala

damage, while manifesting abnormal fear and anxiety responses, still appear to have, contra Kluver and Bucy, a normal repertoire of social behaviors, if those can be triggered (Amaral et al., 2003). The same conclusion appears to hold in humans. The social judgments made by subject SM, for instance, are certainly compromised, but in no way abolished. But this latter evidence only shows that social cognition, as one would expect, relies not on a single type of mechanism but on a huge array of different strategies that depend on very many different neural structures, of which the amygdala is one. One strategy relies on recruiting basic motivational and emotional circuitry, but specifically for the domain of socially relevant stimuli, and it is this strategy that depends on the amygdala. Other strategies, perhaps those relying substantially on language and on declarative memory stores, may not rely on the amygdala.

c. Simulation

Rather than trying to establish that the above data argue for the domain-specificity of social cognition, it may be more fruitful to acknowledge that social cognition draws on a host of cognitive resources, and to ask instead in which way social and non-social information processing support one another. A rich example already adumbrated above is our ability to construct models of counterfactual situations, to imagine the impossible, to mentally travel in time. Certainly, one instance of this ability is the ability to imagine what it is like to be another person whom we are observing. Recent interest in the mechanisms whereby we achieve this has focused on the notion of "simulation", the idea that we are able to run off-line some of the

same processes that we would be engaging ourselves from the observation of another person's behavior. The ability may derive from basic motor control adaptations, and yet may have expanded vastly in the service of the need to accurately predict social behavior.

There is considerable empirical evidence that humans obtain knowledge about other people's emotional states, at least in part, via some kind of articulated emulation (cf. (Grush, 2003) for a recent review). Premotor cortices are engaged when we observe others behaving emotionally, as are somatosensory cortices and insula. One interpretation of these findings is that we engage some of the same machinery during emulation as during actual emotions: the body outside the brain. There is good evidence to further support this idea: observing other people express emotions results in some mirroring of the emotional state in the viewer. In this case it seems that the emulator is the same as the system in normal operation, although it may engage only a subset of a hierarchically structured system. The possibility of using the body itself as the emulator when we model another person's emotion would be not only economical, but suggests an interesting way in which actual, analog physical processes—state changes in various parameters of the body that normally comprise an emotional response—can be used in information processing. The body might be thought of as a somatic scratchpad that we can probe with efferent signals. Given the complexity of interaction among multiple somatic parameters, it may not be feasible to emulate this entirely neurally.

Typically, of course, our emulation should be less than the real thing. Thus emotion emulation involves faint somatic changes that are a subset of having the emotion oneself and that involve active inhibition of expression of some of the components. Much the same happens when we dream: as in waking emulation, we construct models that include responses in our bodies, and while there are indeed somatic responses during dreaming, these are actively

inhibited from full expression. It would seem odd to have evolved such efferent processing and inhibition if the body itself did not play an important role in building the models that help us to predict the world.

The idea of using parts of the body as an emulator is in line with theories of situated or enactive cognition—the idea that the mind makes use of processes that include the external environment and an organism's interaction with it. Three interesting open issues remain. First, an emulation approach to understanding other minds is likely to be a very dynamic, iterative process. It seems unlikely that we obtain all the information in a single shot, and more plausible that we run various parts of the emulator, perhaps to different depths of detail, in order to approximate the answer we seek. Second, there is likely to be a collection of emulators rather than a single one—perhaps these are hierarchically structured in some way. One could imagine emotion emulation involving progressive layers of somatic involvement, depending on the detail of the modeling required, much as visual imagery has been shown to involve different levels of the visual hierarchy, depending on the grain of the imagery. And third, we can well imagine extending the modeling outside the bounds of the body. To obtain social information, we may query not only our own bodies, but other people. Clearly, this is the case in a general sense: we probe other people's reactions to initial and often subtle behaviors on own part, and use their response as feedback in constructing a more accurate model of the social world. All of these ways of creating knowledge about the world should probably be seen on a continuum—ranging from simple lookup tables and systems of rules to models entirely internal to the brain or encompassing varying degrees of the body or external environment.

3. What is special about social cognition.

In reviewing the above examples, I wish to draw two conclusions. First, that they cannot support the idea that social cognition is strongly modular. Second, that they do support the idea that there is indeed something special about social cognition. The trick is to reconcile these two apparently disparate conclusions by steering a middle course that draws on both. Social cognition is too broad a capacity, and makes contact with the rest of cognition at too many places, to be considered anything like an encapsulated, impenetrable module. Yet there is reason to think that it comprises computational strategies that evolved specifically to guide social behavior, that some of those social computations are relatively involuntary, automatic, and below the level of our awareness, and that it draws on a restricted set of neural structures that are sufficiently well defined that we can speak of a "neural system" for social cognition.

In redefining the question, it may be necessary to abandon our predilection for reductive explanations—or at least the usual sort of reduction. It is usually assumed, without any particular argument, that knowledge about the social meaning of a stimulus depends in part on knowledge about the basic motivational value of a stimulus, but not conversely. The assumption is that we could impair social knowledge while sparing knowledge about more basic emotional and motivational value; but that impairing knowledge about basic motivational value would necessarily entail impairments in social knowledge. What is puzzling about this picture is not merely that it is reductionist, but rather that we assume the reduction to proceed in

one direction rather than another. Why not suppose that the social cognition is the basic adaptive package, and that motivated behavior in general draws in part on that?

In general, there may be no single answer to the question of the relation between social cognition and non-social cognition. Is one reducible to the other? Well, some components of social cognition may be reducible to some components of non-social cognition. On the other hand, some components of non-social cognition may be reducible to social cognition. It all depends on the particular component, since neither domain is monolithic. These considerations raise a key issue that may provide an answer to the question of this chapter: the level of analysis of a system. At a very molar level, social behavior is by definition special, since it concerns interactions with other people, rather than objects. At a very microscopic level, social cognition is clearly not special, since it depends on the same microphysical processes as do all other biological events. Is social cognition special? It depends on who is asking the question. Psychologically, behaviorally, there are likely to be aspects of it that are indeed quite special and that make our behaviors so different from those of any other animal. Neurobiologically, it will be more difficult to find similar evidence, since the special behaviors emerge out of vast collections of neural mechanisms that are themselves less clearly specialized.

Nonetheless, one might insist, if there is specialization at the behavioral level, there must be a trace of it to be found in all the subvenient levels—we should see the signature of such social specialization in the brain, in the neurons, in the genes. Well, only if we place those in a much broader context within which they contribute to behavior. That context, of course, is the social environment, culture, and social development. Given a long period of development, given a complex social environment, it may indeed be that a particular social cognitive ability, or a particular aspect of social behavior, depends on the contribution made by specific genes

(Paterson, Brown, Gsoedl, Johnson, & Karmiloff-Smith, 1999). But that doesn't mean that there are genes "for" those aspects of social cognition.

In much the same spirit, we should be reluctant to look for neural structures that are "for" social cognition. Rather, we should consider social cognition as emerging from a very complex interplay among many structures, in the context of development, of a particular culture, and considering the brain as a system that generates behavior only through its interaction with the body and the social environment. What is special about social cognition may well be the extent to which it relies on relations among these different components and levels, and the extent to which it requires their integration.

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