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Theory of Mind in Autism and Schizophrenia

A Case of Over-optimistic Reverse Engineering

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CONSTRAINTS ON MODULAR THEORIES: HORIZONTAL INTEGRATION, VERTICAL INTEGRATION, AND THE THEORY-OF-MIND MODULE

Although autism and schizophrenia present as widely differing disorders, there are intriguing connections between them, which have led to a number of speculative attempts at theoretical unification. First, the term *autism*, now confined to autistic subjects, was first coined by Eugene Bleuler in the 19th century to capture the social isolation and lack of impetus to engage with the world characteristic of schizophrenics experiencing what today would be called "negative symptoms." Second, both autistics and schizophrenics have sensory-motor disorders including perceptual abnormalities, stereotypy and disorganization, unusual patterns of affect, attentional and executive deficits, and social problems. Third, these are both disorders where the range of symptoms extends in a characteristic pattern across many cognitive domains, although general intelligence is sometimes spared.

Explaining these disorders, either separately or together, poses a two-staged challenge of horizontal and vertical integration (Robbins, 1997). *Horizontal*

integration means accounting for and so unifying a pathological set of behavioral and phenomenological symptoms by reference to a cognitive model of normal function. *Vertical integration* refers to the way in which the symptoms thus unified are linked to neurobiology by a theory that shows how the relevant cognitive function is normally implemented, hence pathologically manifested, in human neurobiology. Thus cognitive models initially constructed by abstracting from neurobiology at the stage of horizontal integration are ultimately mapped to neurobiological function at the stage of vertical integration via an implementation theory. This two-staged project of horizontal and vertical integration is complicated by the developmental dimension of neurocognitive disorders (Karmiloff-Smith, 1998; Thomas & Karmiloff-Smith, in press). In disorders acquired during adulthood, theorists can be more confident of the ways in which distinctive abnormalities result from damage to, or disruption of, normally articulated cognitive systems, whereas the distinctive abnormalities of developmental neurocognitive disorders inevitably reflect a long history of organismic adaptation and compensation.

In this chapter, we examine a theory of cognitive function that aims to resolve these difficulties for both autism and schizophrenia. This theory proposes, first, a cognitive account of the core symptoms of each disorder, thereby meeting the goal of horizontal integration. Second, it suggests a way of mapping a range of diverse symptoms onto a highly specific neural substrate, thereby meeting the goal of vertical integration. The theory, originally proposed by Simon Baron-Cohen and collaborators for autism and extended by Chris Frith for schizophrenia, is that both disorders result from the malfunction of a single cognitive system, incorporating, most importantly, the "theory-of-mind" module (or ToMM) (Baron-Cohen, Leslie, & Frith, 1985; Frith, 1992). According to these theorists, the ToMM is required for an understanding of intentional agency because it underwrites autistic peoples' capacity to conceptualize mental states, specifically beliefs and desires, as part of a theory of behavioral explanation. In brief, ToMM theorists suggest that if this specific cognitive module fails to develop properly (perhaps due to failure of subsidiary cognitive modules that play an important role in triggering ToM), the result is autism; if it fails in maturity, the result is schizophrenia. The idea is that the autistic child's specifically social failures and the schizophrenic person's disrupted sense of agency may have a common causal-cognitive structure basis in malfunction of a module specialized for the representation of mental-state concepts. There are, of course, many differences between these disorders despite the similarities just noted. However, as Christopher Frith reminds us, this need not tell against the project of finding a unified theory encompassing both. Given that autism is a developmental disorder, whereas schizophrenia usually occurs first in adulthood, we might reasonably expect significant (although cognitively related) differences in their typical symptoms. As Frith put it:

> It is likely that the cognitive deficit in autism is present from birth, although not reliably detectable until about the third year (Schopler & Mesibov, 1988). As a consequence the whole course of development must be abnormal. There is evidence that a proportion of schizophrenic patients show signs of social abnormalities during childhood (Castle, Wessely, & Murray, submitted). However in most cases of schizophrenia development appears to be entirely normal until the first breakdown, typically in the early 20s. My proposal is that people with schizophrenia resemble people with autism in that they too have impairments in the mechanism that enable them to empathize. However in most cases this mechanism was functioning adequately until their first breakdown. Given these very different developmental histories this defect will be manifest in different ways. The autistic person has never known that other people have minds. The schizophrenic knows well that other people have minds but has lost the ability to infer the contents of these minds: their beliefs and intentions. They may even lose the ability to reflect on the contents of their own mind. However they still have available ritual and behavioural routines for interacting with people which do not require inferences about mental states. (Frith, 1992, p. 121)

Despite its initial appeal, we argue that the ToMM theory of autism and schizophrenia fails. There is no single ToM module responsible for successful social reasoning and behavior in normal subjects; hence there is no dedicated module, realized in neural substrate, that fails to develop in autism or breaks down in schizophrenia. In other words, we argue that the ToMM theory fails at the first stage of horizontal integration.

CONSTRAINTS ON MODULAR THEORIES

Cognitive Domains: Actual or Virtual

Our objection to the ToM modularity hypothesis does not hinge on evidence for or against neural localization. We accept that cognitive functions can be realized in distributed neural architectures. In our view, the only structural constraint modularity hypotheses must meet is the following: In order for there to be an *actual* cognitive domain subserved by a dedicated module for processing information specific to that domain, it must be realized in a functionally specific neural assembly—that is, an assembly that serves no other cognitive function(s). If this constraint cannot be met, then even supposing a particular disorder comprises a pathological behavioral domain, the domain specificity of the underlying cognitive disorder is merely *virtual*: There may be an appearance of modularity but the appearance is misleading. In both autism and schizophrenia, the *apparent* ToM deficits are a consequence of a disunified array of cognitive and (possibly) noncognitive malfunctions. This implies that whatever unity

obtains in the domain of social cognition is merely virtual, the result of a number of interacting subsystems whose interaction is not governed by the operations of a single "theory-of-mind" mechanism.

Cognitive Capacities: Developmentally Set or Developmentally Constructed

To say that a cognitive domain is actual—that is, subserved by a dedicated module—is a synchronic hypothesis about cognitive architecture. It does not depend on claims about the diachronic history of cognitive development. Thus objections to modular nativism should be disentangled from objections to modularity per se (Karmiloff-Smith, 1998; cf. Elman et al., 1996). A cognitive capacity may be modular in the sense of functionally discrete without such modularity being genetically predetermined. If so, there can be failure of typical modularization without failure of a genetically specified module.

To illustrate this difference, consider two alternative explanations of specific language impairment (SLI), a deficit in language ability that spares other cognitive capacities. The classical approach to this disorder, on which nativist theories of ToM deficits are modeled, is one of straightforward reverse engineering: In order to account for the unified and specific behavioral deficit, classical theorists posit a genetically prespecified dedicated language module for deriving grammatical rules that fails to mature normally. Evidence of heritability is often taken as further support for the innate modular theory (Gopnik & Goad, 1997; Van der Lely, 1997).

Another possibility is that SLI is not the result of failure in a genetically prespecified modular language capacity at all. It is, rather, the absence of a modularized capacity—hence, a specific language impairment—due to the developmental impact of a subtle hearing deficit (Donnai & Karmiloff Smith, 2000; Tallal, 1985, 1988; Thomas & Karmiloff-Smith, in press). In order to read linguistic structure into the acoustic stream, children need to be able to detect significant acoustic variation in that stream, but the SLI subject is unable to do this. For example, the SLI child might just hear "ough" instead of "D" "O" "G" where "dog" comes in the middle of an acoustic stream. Wright, Lombardino, Puranik, Leonard, and Merzenich (1997) found that in order for SLI subjects to distinguish linguistically significant variations against the masking effects of surrounding sounds, they needed to be amplified 45 dB above the surrounding stream (see also Tallal, Miller, & Fitch, 1995, on the ameliorating effects of lengthening phonemic transitions). The hearing of these subjects would not show up as abnormal in standard tests because it is their ability to overcome masking effects that is the problem, not auditory function per se. (Subjects can hear the sounds perfectly well as long as they are not masked by preceding and succeeding sounds.) If this explanation is right, then the link between SLI as a behavioral domain and the underlying cognitive domain is not, as it initially

appears, the result of an impairment in native linguistic capacity. Instead SLI is the product of a developmental cascade based on a subtle perceptual deficit which has nothing to do with language per se (Wright et al., 1997). Thus, theses like that of Stephen Pinker that SLI is evidence for the presence of a genetically specified module devoted to syntactic processing are undermined (Gerrans, 2002; Karmiloff-Smith, 1998; Karmiloff-Smith et al., 2003; Thomas & Karmiloff-Smith, in press). This explanation will naturally complicate the project of vertical integration for SLI as a developmental disorder: Instead of searching for the neural substrate of a specific grammatical capacity in early stages of linguistic development, theorists ought to be looking for the neural substrate of a cognitive system dedicated to processing phonological distinctions.

We call a cognitive capacity *developmentally set* insofar as it is achieved by a module whose cognitive architecture is genetically prespecified. By contrast, a cognitive capacity is *developmentally constructed* if the mechanism or mechanisms that achieve it are progressively structured in consequence of an organism's environmentally interactive developmental history. Thus it counts against a dedicated modularity hypothesis as a developmental thesis about high-level cognitive capacities such as syntax or ToM if it can be shown that such specialized competencies fail to develop as a result of the way organisms interact with their environment in consequence of lower order abnormalities. However, as in the case just described, it may be that some cognitive capacity is still an actual cognitive domain, capable of being selectively impaired in adulthood, even if it depends developmentally on mediating systems that are cognitively unrelated to the capacity in question.

Cognitive Versus Mechanical Explanations: Guarding Against Methodological Bias

Explaining any cognitive disorder begins with its behavioral profile: the distinctive pattern of disabilities and spared (or sometimes superior) abilities. From there, theorists hypothesize underlying structures that account for this behavioral profile, testing these hypotheses against further external and/or internal sources of evidence (e.g., cognitive-behavioral and/or neurological studies, as well as computational models), and then modifying or replacing their theories. This methodological practice of reverse engineering is indispensable yet introduces a bias in explanation toward theories that postulate dedicated higher order cognitive structures. This bias is not necessarily a bad thing, because the theories it favors may be on the right track. However, we note that this methodologically induced bias can easily slide into a reverse-engineering fallacy if alternative explanations are not appropriately considered. This is particularly true if alternatives seem theoretically more complicated and empirically less tractable when it comes to experimental design. Still, although it may be more elegant to postulate an X-module to explain a set of symptoms, the truth for any

particular disorder may often lie in a complex multiplicity of overlapping cognitive and noncognitive causes. To keep the full range of conceptual possibilities in mind, we think a distinction between cognitive and purely mechanical explanations of cognitive function can be usefully clarified.

Cognitive Explanations. An explanation for a disorder counts as genuinely cognitive if it makes essential or theoretically ineliminable reference to a system's design by way of invoking a malfunctioning cognitive mechanism or mechanisms (Dennett, 1978). For example, we lose explanatory power if we explain visual neglect purely in neural terms without reference to the visual task the neural system implicated normally performs. Of course, because cognitive mechanisms are realized in neural substrate, cognitive malfunctions are inevitably problems at the level of neuronal functioning. However, a cognitive theory initially abstracts from neural realization in identifying a cognitive function and then maps that function to its neural substrate via an implementation theory. Consider an analogous case: A computer malfunction is correctly explained at the design or programming level (analogous to the cognitive level) if its occurrence is contingent upon some flaw in the program no matter how that program is physically realized—differently, as it may be, in two different computer systems (cf. below note 1).

Thus, in terms of the taxonomy introduced earlier, a cognitive module, putatively identified as the cause of a disorder, may be actual or virtual. If it is actual, then there is selective damage to a dedicated cognitive mechanism, making the reverse engineering inference from behavior to mechanism relatively straightforward. In this case, the module implicated in the disorder is architecturally real (see Figure 12.1). By contrast, the postulated unifying cognitive function may be virtual: It gives the appearance of being achieved by a dedicated mechanism, but the underlying architectural reality is of a number of interacting, possibly lower order, quasi-independent cognitive subsystems. The proposed cognitive function is descriptively too abstract to serve as an implementation theory and, in this sense, the modularity hypothesis misdescribes the organization of the agent's cognitive system (see Figure 12.2).

What kind of evidence could distinguish between these two alternatives? One important piece of evidence exploits the putative link between autism and schizophrenia. For, should it turn out as we suspect in schizophrenia, that postdevelopmental abnormalities in lower order systems suffice to produce the pathological symptoms, this is good reason to question the existence of a module specialized for cognizing that domain, hence one that could be differentially affected in the course of autistic development.

Mechanical Explanations. So far, we have claimed that reasoning backward from a behavioral profile may misleadingly invite high-level modularity hypotheses for specific cognitive disorders, either diachronically, as a developmental

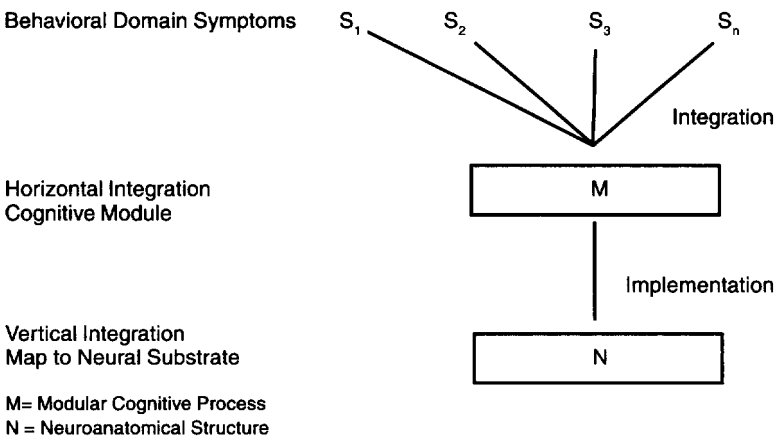

FIGURE 12.1. Reverse engineering: Mapping symptoms to neural substrate via a cognitive model.

hypothesis, or synchronically, as a hypothesis about contemporary architectural organization. However, the postulation of dedicated cognitive entities can also mislead if it turns out that the disorder is not cognitive at all. This is the case with structureless neuropathologies such as Alzheimer's disease. The interest of this type of case for our purposes is that structureless neuropathology can sometimes produce behavioral outcomes that misleadingly invite cognitive unification at higher levels and so constitute architecturally virtual domains.

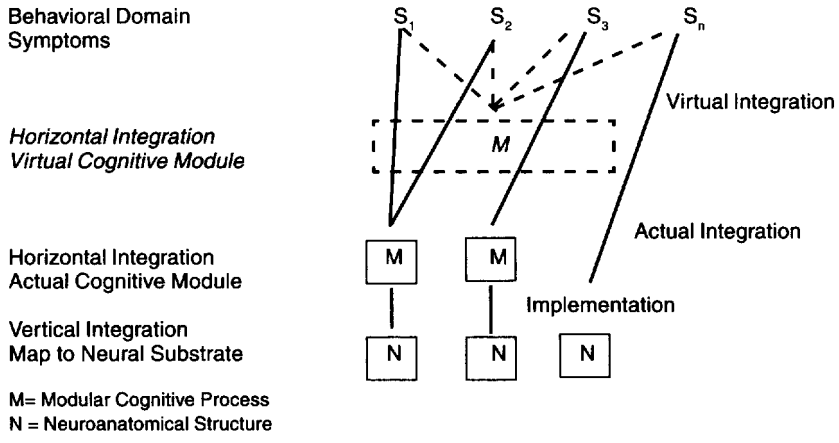

FIGURE 12.2. Reverse engineering fallacy 1: Lower level cognitive explanation.

Consider, for instance, the disorder *phenylketonuria* (PKU). Although PKU has many symptoms that are similar to autism, it is produced not by the failure of a specific cognitive module, but by the absence of an enzyme that synthesizes an amino acid. In this case, postulating the failure of a higher order mechanism is ultimately mistaken, not because the postulated module fractures into a subset of cognitively or developmentally more basic ones, but because the cause of the disorder is essentially noncognitive: It does not arise through the breakdown of any cognitive system *qua* cognitive system. Because there is no cognitive story to tell in this case, the correct approach for achieving vertical integration is bottom up: We explain the neural malfunction and treat the resultant behavior as the outcome of haphazard interference with the development of a number of arbitrarily involved cognitive functions. The correct explanation, as illustrated in Figure 12.3, is entirely at the level of neural mechanism.[1]

Autism and schizophrenia are interesting cases precisely because their diversity of symptoms and lack of uniformity from case to case continue to encourage the idea, popular among pioneers of neurology, that the deficits in question may be essentially mechanical rather than cognitive. Nonetheless, clinicians are reluctant to endorse this conclusion because the disorders do seem to form a pathological domain, rather than a random collection of symptoms. Hence, unifying cognitive theories, such as ToMM, continue to be attractive. However, ToM theorists go too far in proposing that high-level processing of social information constitutes a developmentally set or, indeed, architecturally real cognitive system whose malfunction is implicated in both autism and schizophrenia.

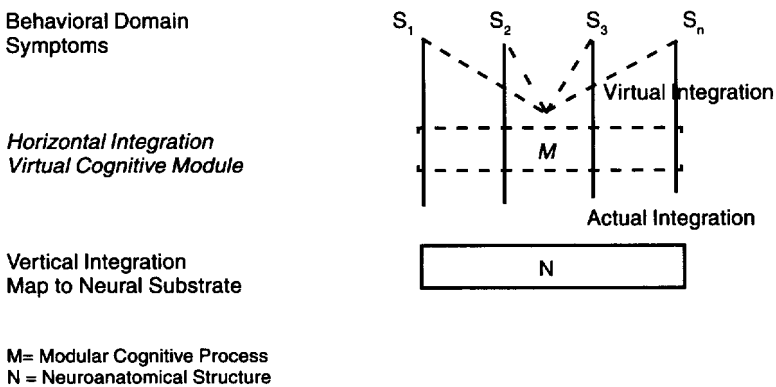

FIGURE 12.3. Reverse engineering fallacy 2: Mechanical explanation.

STRONG AND WEAK TOMM HYPOTHESIS

In both autism and schizophrenia we propose that the symptoms unified under theToMM hypothesis can be explained in either one of two ways: (a) as a result of malfunction at sensorimotor or perceptual levels (i.e., in terms of malfunction of a lower order cognitive module), or (b) as a result of purely mechanical malfunction.

In the case of autism, we remain neutral between the two alternatives because determining whether the type of sensorimotor disorder we identify as a developmental precursor to autism is low-level cognitive or mechanical is not a simple matter. However, we do think that a developmental cascade consequent on early sensorimotor malfunction is a sufficient explanation of mentalizing abnormalities, especially once the role of social interaction, modulated by sensory experience, is given its due in characterizing the development of higher order social cognition. In the case of schizophrenia, there are two well-developed alternatives to a ToMM account that fit the pattern we outlined. In keeping with option (a), the motor control account locates the malfunction that produces a significant set of symptoms in a lower order cognitive subsystem whose neural substrate is the premotor cortex. In keeping with option (b), the misconnection account treats schizophrenia as a mechanical malfunction with diffuse cortical and subcortical effects.

These arguments are directed at a strong version of the ToMM hypothesis, which claims that a malfunctioning ToM module accounts for all significant symptoms of these disorders. The ToMM theorist may also defend a weaker claim that autism and schizophrenia involve a congery of cognitive capacities of which only an essential core, typically having to do with social cognition, are ToMM deficits. However, this weaker claim is open to the following objections: First, if the affected capacities are genuinely independent, then there should be some evidence of dissociation between so-called core and peripheral symptoms occurring in both these disorders. This has yet to be established. But even were there convincing evidence of dissociation, the ToMM hypothesis could no longer play an explanatory role in autism (or schizophrenia) per se, but only for so-called core (i.e., ToM) aspects of the disorder. This only postpones the question of why these "core" problems are often enough conjoined with other problems to constitute an autistic (or schizophrenic) behavioral type. Moreover, if the core deficits can be produced in either of the ways we suggest, then there is no reason to save the ToMM hypothesis at all. This in effect replays the argument against strong ToMM for the more restricted set of "core" symptoms.

In light of these difficulties, the weak ToMM theorist may revert to the claim that all symptoms, core and peripheral, are connected even though they are not dependent on the same module. But this is to concede that however specific the cognitive abnormalities seem in autism (or schizophrenia), the disorder involves a number of subsystems linked by neural architecture or neuro-

chemical modulation. In other words, as in the case of PKU or "misconnection" explanations of schizophrenia, the correct level of explanation is mechanical.

In sum, the weak ToMM hypothesis is not a stable fallback position. But the strong ToMM hypothesis—that all significant symptoms of these disorders are in fact cognitively unified by the ToM module—is far too strong to remain defensible, even in the eyes of some of its main proponents. We conclude that although the ToMM hypothesis has generated much valuable research, it is in the end an instance of overly optimistic reverse engineering.

THE CASE OF AUTISM[2]

Autistic individuals share a distinctive triad of impairments in social, communicative, and imaginative capacities (the latter demonstrated by the absence of pretend play in childhood and restricted interests and activities that persist throughout life) (Rutter & Schopler, 1987; Wing & Gould, 1978, 1979). Although 75% of individuals diagnosed with autism are intellectually handicapped in a general way (as reflected in low IQ scores), the remaining 25% have normal to high IQs and often perform well, and sometimes better than average, on reasoning tasks that do not require any understanding of the mental life of agents. In contrast, on so-called "theory-of-mind" tests, these "high-functioning" autistic children are significantly impaired when compared with normal children and even those with Down's syndrome who are matched with them by mental age (for a review of research, see Baron-Cohen, 2001). For instance, on first-order false-belief tasks, which require subjects to predict another's behavior on the basis of attributing them to a false belief, children will normally pass by a mental age of 4 to 5 years (Wimmer & Perner, 1983).[3] Autistic individuals, if they pass at all, only do so when they are considerably older: on average, at a verbal mental age of 9 (Happé, 1995).

A prime example of this dissociation between social and nonsocial reasoning skills involves the Zaitchik "false-photograph" task, which is modeled on the standard false-belief task except insofar as it tests children's ability to reason about physical (photographic) instead of mental representation (Zaitchik, 1990). Using a simplified version of this task, Leekam and Perner (1991) tested a group of high-functioning autistic teenagers in two conditions, one testing false-belief understanding and the other, photographic "misrepresentation." In both conditions, participants were shown a doll (Judy) wearing a red dress. In the false-belief condition, a second doll (Susan) sees Judy in the red dress and then leaves the room. Judy's dress is changed from red to green, and subjects are asked: "What color does Susan think that Judy is?" In the false photograph condition, a Polaroid photo is taken of Judy in the red dress. While the photo is developing, her dress is again changed from red to green, and subjects are asked: "In the picture, what color is Judy?" Only 25% of autistic participants were correct on the false-belief question, but almost all those tested passed the false-

photograph question (Leekam & Perner, 1991). Similar results were obtained by Leslie and Thais (1992). This pattern of dramatically failing false-belief while passing false-photograph tasks does not occur in normal 4-year-olds.[4]

Results like these strongly suggest that autistic individuals are not generally impaired in their reasoning abilities, but rather have a specific inability to reason about, and perhaps even conceptualize, mental states and processes—hence the idea that autism results from the developmental failure of a so-called ToM module, an "innate, isolable component of the mind which embodies a *theory* of the nature and the operations of mind" (Carruthers, 1996, p. 258; see also Baron-Cohen, 1995; Leslie & Thaiss, 1992). Moreover, as an important extension of their theory, ToMM theorists argue that a dysfunctional ToM module can account not just for the deficits identified experimentally, but for the clinical profile collected under the headings of social, communicative, and imaginative abnormalities.[5]

Consider, for instance, the characteristic social abnormalities associated with autism. These might easily be connected with an inability to attribute mental states to others, especially if these abnormalities reflect an apparent indifference or insensitivity to what others are thinking and feeling. Thus, autistic children show no interest in, and even a positive aversion to, meeting another's eyes. They show no tendency to engage in social referencing behaviors, that is, directing another's attention toward an object in order to share their interest in it or gather information about it. They show little understanding of how their actions affect others or how others' actions are meant to affect them. They may often be confused by what other people do, but show little capacity to be hurt by intentionally malicious behavior, or touched by intentionally kind behavior whether or not the behavior is experienced as beneficial. They may be amused by other people's physical "antics," even when those antics betray extreme distress or pain. They understand sabotage, but are blind to deceit and other forms of slyness.

Communicative abnormalities may also be rooted in this mentalizing deficit. Language skills vary widely across the autistic population. But even among those who develop fair linguistic capacity, typical problems remain. These are connected in particular with communicative and pragmatic aspects of language use that depend on the speaker's awareness of the conversational situation, including especially the listener's point of view: abnormal prosody (rhythm, stress, tone), abnormal shifts in topic, inability to give and receive conversational cues, abnormal accompanying gestures and facial expressions, pronoun reversals ("I" for "you"), idiosyncratic use of words, abrupt interruptions and terminations of conversation, insensitivity to taboos on personal topics, and so forth. Autistic individuals also tend toward extreme literal-mindedness—showing an insensitivity to metaphor, irony, sarcasm, even idioms as idioms: To autistic individuals, "he went the whole nine yards" means, literally, "he went nine whole yards." There is little or no understanding that others may intend to convey by their words something more or other than just what their words mean.

Some of these communicative abnormalities are closely related to the final element in this triad of deficits: autistic lack of imagination. From early childhood, autistic individuals show a notable absence of spontaneous pretend play, as if it never occurs to them to think about things (represent them) other than as they are. Instead, they will engage in repetitive, stereotyped activities such as sorting objects or lining them up in rows. They also tend to show limited or absent interest in the larger meaning of things (function, associations, symbolic properties) but focus instead on superficial details, with obsessive interests that are circumscribed accordingly. It may be memorizing bus routes, timetables, birth dates, or even door colors. Many autistic individuals are notable for their rote memory skills, even though they show little concern with focusing on what's worth remembering for other cognitive purposes. Perhaps this is because they have a limited capacity for imagining what those purposes might be, hence a limited capacity for opportunistic planning (for a discussion of planning deficits as connected with theory-of-mind capacities, see Currie, 1996; for an alternative perspective, see Russell, 1997).

So prima facie ToMM is a very attractive unifying hypothesis, but we should note that there are other autistic abnormalities that seem to have little to do with "theory-of-mind" capacities. These include sensory-motor problems: for example, extreme and unusual physical sensitivities and insensitivities; slowed orienting of attention; oddities of posture and gait; tics, twitches, and unusual mannerisms; and stereotypies such as rocking, hand-flapping, spinning, thumb-twiddling, and echolalia. They also include abnormalities in perceptual processing, leading to a characteristic autistic profile of assets and deficits on various perceptual tasks: for example, insusceptibility to certain perceptual illusions, superior performance on finding embedded figures within a larger design, superior visual memory and capacity for rendering scenes in precise detail, perfect pitch, difficulties with "gestalt" perception—seeing whole figures or scenes as opposed to their parts, absence of perceptual "switching" with ambiguous figures such as the duck-rabbit, and so on.

How can ToMM theorists account for these additional symptoms? As we saw in the introduction, a weak ToMM view would involve conceding that these abnormalities are fundamentally unrelated to the core ToMM deficit, so that autism is, in effect, an association of relatively independent disorders resulting from multiple failures across a variety of distinct neurological systems. However, if these neurological systems are genuinely independent, then either we should expect some evidence of dissociable breakdown or, in lieu of that, some explanation for why breakdowns in multiple systems co-occur. The weak ToMM theorist encounters trouble either way: The first possibility seems empirically unvindicated and the second undercuts the explanatory power of the weak ToMM hypothesis.

Consider the first possibility: If ToM deficits are essentially unrelated to other characteristic symptoms in autism, then we should expect to see a relatively pure ToM-impaired autistic type. Such autistics would most nearly re-

semble individuals who have developed normally but show impairments in social cognition because of acquired neurological damage in their frontal or temporal lobes. Yet there seems to be no evidence of such specific abnormalities in the autistic population. Whatever range of symptoms autistics manifest, their profile is quite unlike that of brain-damaged adults, especially with regard to sensory-motor and perceptual difficulties. However, as Tager-Flusberg reminded us, these differences in profile should not be surprising given that autism is a neurodevelopmental disorder:

> Interestingly, neurodevelopmental disorders are more often associated with *diffuse* cortical damage, which suggests the impact of such disorders is more widespread, affecting complex neural systems rather than simple localized areas. Furthermore, across a range of developmental syndromes, we find that not only are particular cortical systems affected but often associated atypical subcortical structures are involved as well. For example, in autism both the cerebellum and limbic system show significant abnormalities. These findings suggest deviations in brain development that begin early in embryology and cannot be easily classified and interpreted as later acquired focal lesions. Our theories of structural brain abnormalities in neurodevelopmental disorders will have to incorporate these kinds of developmental complexities rather than relying on more established studies from work with adults. (Tager-Flusberg, 1999, p. 3)

More likely, then, is the second possibility: Autism involves multiple failures across various distinct neurological systems that co-occur for a reason. If these systems are functionally unrelated, as the weak ToMM theorist avers, then it seems the only kind of account that would make sense of this multiple failure is a mechanical one. As with PKU, autistic behavior might be the result of haphazard interference with the development of a number of arbitrarily involved cognitive functions, including those supporting social cognition. If so, hypothesizing a particular deficit in the putative ToM module does no explanatory work in autism, whatever it may do for explaining acquired disorders in social cognition. (This is not to say we endorse such a hypothesis, only that it plays no role in the explanation of autism even under the assumption that social cognition is normally accomplished by a modularized neural system.)

Faced with these difficulties, the ToMM theorist might well consider the stronger claim: Many if not all "peripheral" autistic symptoms can be explained in terms of a malfunctioning ToM module, in which case they are not peripheral to defects in social cognition at all, since they too are produced by failure to metarepresent mental states. Uta Frith and Francesca Happé (1999) recently made this suggestion,[6] building on an idea repeatedly emphasized by Alison Gopnik: that a ToM capacity implies no asymmetry between first- and third-person ascriptions of mental states.[7] Thus, the autistic subject's understanding of her own mind would be just as impoverished as her understanding of other minds.

Frith and Happé speculated that because autistic subjects cannot metarepresent their sensory and perceptual processes as states of their own mind, they are at the mercy of them in a way that could produce the characteristic profile of autistic abnormalities. For instance, on motor tests involving monitoring and correction of action, autistics perform poorly when compared with normal controls (Russell & Jarrold, 1998, 1999). Frith and Happé offered the following ToM explanation: "without self-awareness, an individual might not know how she is going to act until she acted, nor why she acted as she did. . . . A person who lacks self-consciousness may be unable to distinguish between her own willed and involuntary actions" (Frith & Happé, 1999, p. 8). Alternatively, there might be improved performance on tasks where action without in-depth conscious reflection is superior to consciously performed action. This might explain autistic individuals' relatively good capacity to perform routinized action coupled with a poor capacity to act flexibly and imaginatively (ibid., p. 10). It might also account for autistic insusceptibility to certain visual illusions, such as the Titchener circles (or Ebbinghaus illusion), where subjects' "superior performance in verbal response is not contaminated by conscious reflection" (ibid., p. 10). [In support of this contention, recent evidence shows that when even normal subjects respond motorically (and apparently unconsciously) in their reaching behavior to the correct size of the circles, nevertheless they continue to (consciously) judge the size of the circles incorrectly (Aglioti, DeSouza, & Goodale, 1995)].

One notable and particularly salient characteristic of autism is extreme and unusual sensory experience. Frith and Happé relate this to a dysfunctional ToM as follows:

> If low-functioning autistics are unable to reflect on their inner experiences, then they would be unable to develop over time the richly connected semantic and experiential associations which normally pervade our reflective consciousness. Observation by parents suggests that the awareness of sensations and experiences may be peculiar in children with autism. Anecdotal reports of abnormal sensory and pain experiences are on occasion quite extreme. . . . One anecdotal example is the case of a young girl with autism who was found to have suffered acute appendicitis, but had not complained of pain and, when asked how she felt, did not report anything wrong. Abnormal response to heat and cold, as well as hypo- and hyper-sensitivity to sound, light or touch are frequently reported. . . . Such responses might be expected if there was an inability to reflect on inner experiential states. Of course, normal pain perception is greatly affected by attribution and expectation. These individuals might feel immediate pain in the same way as everyone else, but would not be able to attribute to themselves the emotional significance that normally accompanies pain. This might explain why they do not complain about it. We may speculate that the self-conscious person reflects not only on the pain but also on the experience of pain. This person is feeling "misery" in addition to feeling pain. (Frith & Happé, 1999, pp. 10–11)

Frith and Happé's view implies that autistic subjects would be rather unreflective about their sensory experiences. But, as they themselves noted, autistic hypersensitivity is as dramatic as autistic hyposensitivity. In fact, both third-person observation and first-person report indicate that autistic subjects are abnormally aware of their sensory experiences, and aware of them mainly because their sensory experiences are extreme and persistently captivating. Furthermore, they are aware of them *as* mental experiences. It's hard to see how this can be accounted for in terms of a general deficit of self-consciousness stemming from a dysfunctional ToM mechanism. And, of course, if there is no general deficit of self-consciousness; accounting for autistic motor and perceptual abnormalities in these terms becomes equally suspect (as opposed to accounting for them in terms of local motor and perceptual system dysfunctions).

Our conclusion is that Frith and Happé's strong ToMM hypothesis encounters these difficulties because it reverses, temporally and conceptually, the correct direction of explanation between lower and higher order cognitive capacities. Reverting to the SLI case, it is as if someone tried to explain auditory failures and related behavior as the consequence of defective grammatical processing rather than defective grammatical processing as the consequence of early auditory difficulties. The higher order cognitive difficulties of autism and other developmental disorders are far more likely to be the cascading effects of relatively early, lower order sensory and perceptual abnormalities than lower order abnormalities are to be the result of a failure in higher cognition (Karmiloff-Smith, 1998; cf. Tager-Flusberg, 1999). If this developmental connection makes theoretical (and empirical) sense, then the explanatory difficulty of integrating the various symptoms of the disorder disappears.

To make theoretical sense of this approach, we need to consider how early sensory and perceptual disturbances could generate the autistic profile of social deficits. The first step involves restoring the emphasis, sometimes lost in reverse engineering reasoning, of the role intersubjective encounters play in developing a child's capacity for normal agency. Reciprocal, affectively patterned interactions with others are important determinants of infant experience and a necessary condition for the development of higher order cognitive capacities, especially those relating to "mind reading" (here, minimalistically conceived as a capacity for understanding others as psychological agents like oneself). In our view, developing a capacity for "mind reading" goes hand in hand with developing the capacity to *be minded* like others—that is, with acquiring habits of psychological self-regulation and behavior that conform to shared norms of sensible (predictable, rational) agency. If the infant's sensory-motor and perceptual systems are abnormal, his or her motivation and ability to engage with others in the types of interaction that lead to developing such capacities of agency will be disrupted from the very beginning. Hence, the difference between autistic and normal individuals may not be so much the lack of some specifically social capacity as the failure to develop their capacities within an intensely social context

(for more detailed and somewhat complementary developments of this view, see Gerrans, 1998; McGeer 2001).

This fits with, although does not precisely repeat, a theme emphasized by Hobson and other theorists that what matters to a child's normal social cognitive development is the affective quality of his or her intersubjective experience (Hobson, 1991; Stern, 1985; Trevarthen, 1979; Trevarthen & Hubley, 1978). That is to say, the initial innate bridge between self and other is sustained by perceiving and reproducing the bodily expressed feelings of others: smile for smile, frown for frown, fearful look for fearful look. This makes others potentially significant for the infant in two respects at once: Not only do they provide information about the world and human experience; they also serve as a critical source of sensory-affective regulation. Thus, for instance, a mother may comfort a distressed child by, first, adopting in face and voice expressions that are recognizable to the child as mirroring its own distress, then modulating these in a way that expresses the easing of distress. The child, carried along by its innate proclivities for imitation, will often follow the direction of the mother's expressive modulation, experiencing the easing of its own distress in consequence (Gergely & Watson, 1995). Indeed, the regulative benefits of imitation may be so critical to an infant's well-being that it is they, rather than any direct epistemic rewards, that drive the infant's interactions with responsive others.

In learning how to be like others, the infant is learning how to be itself in tolerable contact with the world. Of course, these structured interactions, first with others, then later with objects and situations via the mediation of others, become enormously rewarding on the epistemic front as well, for they allow the growing child to metabolize its experiences in ways that are conducive to developing a picture of the world as a stable, predictable place. The normal child who becomes well-regulated in the manner of other people thus derives a double epistemic benefit from this process: The world, including the progressively more complex and differentiated behavior of other people, is made open to manageable exploration, while, at the same time, other people become known to the child inside and out in a way that underwrites his or her "mind-reading" capacities.

If this is a reasonable sketch of what happens in normal development, it suggests a clear connection between autistic sensory disturbances and their failure to engage with others in ways that lead to developing normal capacities of agency. Autistic individuals need not lack a basic social capacity, or even drive to imitate others, as is sometimes suggested (Meltzoff & Gopnik, 1993). Indeed, some autistics show extraordinary if oddly selective parroting tendencies. Nor need it be true that they have a basic affective disorder, as Hobson and others propose. Their capacity to imitate and so engage in intersubjective encounters would hardly be evoked in a sustained way if autistics find their contact with others, on the whole, far too stimulating to be tolerated. Indeed, in an effort to manage their sensory experiences, autistic individuals might need to shut other people out in a fairly pointed way. But far from indicating that they lack any

specialized machinery for attending to others, this may well show that they *have* such machinery, with the consequence that others constitute a disproportionately powerful source of stimuli that quickly become overwhelming for them. In any case, the devastating effects of finding in others an abnormal source of sensory dysregulation rather than a normal source of helpful regulation are twofold: (1) Autistic individuals would be cast back on their own resources for managing their sensory experiences perhaps by reducing, repeating or drowning out incoming sensory stimuli in ways they can control. This could explain a number of characteristic autistic behaviors that range from being seemingly dull and repetitive to bizarrely self-stimulatory and even self-abusive: lining up blocks, counting and calculating, repetitively flushing toilets, examining grains of sand, chewing things regardless of taste or danger, spinning, hand-flapping, rocking, echolalia, head-banging, biting and slapping oneself, and so forth. (2) Being excluded from the regulative influences of other people, autistics will not develop habits of agency that conform to shared norms of what it is to experience, think, and act in recognizably normal ways. Hence, they will be deprived of the very kinds of interactions that give rise to ordinary capacities of agency, a disability reflected in the perplexing nature of their own behavior as well as in their own perplexity at the behavior of others.

In sum, we think that strong ToMM theorists are right to emphasize the connection between a capacity to know other minds and the sorts of capacities for self-awareness and self-governance that make for normal agency. As against weak ToMM theorists, we also think strong ToMM theorists are right to emphasize the connection between higher order cognitive abnormalities in autism and so-called peripheral symptoms. However, in postulating a single higher order cognitive deficit, they neglect to consider how basic sensory and perceptual problems can have cascading developmental effects, particularly with respect to sociocognitive development, by disrupting an infant's normal environment of regulative interactions with others. From this perspective, autistic mentalizing deficits are part of a more general pattern of deficits that constitute from birth to maturity an abnormal developmental trajectory that leads to a distinctive cognitive style or set of cognitive styles. In some cases, depending on the severity of the initial problems, the child's compensatory abilities, and the kind of environmental supports he or she may find, this developmental trajectory may even produce capacities that are sufficient to pass some, if not all, "theory-of-mind" tasks. Such capacities have been found amongst high-functioning autistics, although they vary widely and do not lead to normal social behavior. Are these autistic individuals then not genuine mentalizers? Certainly they are not mentalizers in the usual sense. However, in our view, this is not because their ToM module is inoperative or only partially operative; rather, it is because the capacities they develop bear only, and to varying degrees, a family resemblance to capacities that characterize the cognitive styles of normally developing individuals.

There are a number of advantages to the bottom-up developmental ap-

proach we suggest. We have discussed two of them and alluded to a third. These include: (1) respecting the distinction between "peripheral" (or lower order) and "core" (higher order cognitive) abnormalities, and yet accounting in an integrated way for their comorbidity; (2) making sense of the neurofunctional differences between a disorder like autism and acquired pathologies consequent upon localized brain damage; and (3) providing a natural way to account for the range of mentalizing capacities found amongst high-functioning autistics without having to posit a late-developing ToM mechanism that is only partially operative.[8]

A fourth and final advantage of our approach bears special mention because it makes sense of an otherwise puzzling phenomenon. If autistic sensory disturbances do indeed lie at the developmental core of later "mindblindness" and other higher order cognitive abnormalities, then other clinical populations with early sensory problems ought to show similar kinds of deficits. And indeed this is the case. Deaf children of hearing parents as well as congenitally blind children show autistic-like abnormalities in social, communicative, and imaginative skills, as well as selective incapacity to pass reasoning tasks with a mentalistic component (Brown et al., 1997; Hobson, 1993; Peterson & Siegal, 1998, 1999; Peterson, Peterson, & Webb, 2000).[9] We think the parallels among these populations are so stunning as to call for a unifying explanation. It follows from the account developed here that any child will be unable to develop mind reading as long as it is impossible for him or her to make good regulative use of other people. This may stem from having a missing sensory avenue to others, as much as it may stem from having one's sensory avenues to others overwhelmed by the overstimulation involved in sustained exposure to them.

In terms of the taxonomy we laid out in our introductory remarks, we have argued that the mentalizing deficits of autism constitute at the very least a developmentally constructed cognitive domain. Whatever neural specialization for social reasoning occurs in normally developing children, we believe this is the outcome of an ongoing process of functional development in which these higher order cognitive capacities depend on a child's normal engagement with others in a structured social environment. Such normal engagement is naturally mediated by a child's sensory-motor and perceptual systems, among other things, so if profound disruptions occur in these systems, it is not surprising that subsequent neurofunctional developmental will be dramatically affected. Do we therefore think that normal development leads to the kind of modularization of "theory-of-mind" capacities that nonnativistic ToMM theorists suggest? Do we think, in other words, that normal ToM capacities constitute an actual cognitive domain, subserved by an architecturally real neural system? Nothing we have said so far argues strongly against this possibility. However, we do think that the ToMM account of an adult-onset disorder like schizophrenia, with symptoms that are interestingly similar to and interestingly distinct from autism, is sufficiently problematic that it points to the general conclusion that social reasoning constitutes an architecturally virtual as well as a developmentally constructed domain.

THE CASE OF SCHIZOPHRENIA

In its strong form, the ToMM theory of schizophrenia hypothesizes that all symptoms of the disorder derive from an acquired deficit in ToM function. It is certainly true that schizophrenics perform very poorly on standard ToM tests (Langdon & Coltheart, 1999) and that their social interactions, especially in cases of severe negative symptoms, are severely impaired. However, the real impetus (and challenge) for the ToMM theory derives less from giving an adequate account of these ToM deficits than to providing an integrated account of the range of disparate symptoms not obviously connected with problems in social cognition. For example, it is not clear how hallucinations and delusions, formal thought disorder, and inappropriate affect and the psychomotor poverty (alexia, avolition, apathy, flat affect) could all be attributable to one underlying cognitive problem.

The first stage in achieving horizontal integration of schizophrenic symptoms is to focus on the nature of the delusions and hallucinations characterizing the disorder. Typically these concern the subject's sense of agency—that is, the feeling that the subject is the author of his or her own thoughts or actions. In some cases the schizophrenic feels as if auditory experiences ("hearing voices") that are, so to speak, "in her mind" are nevertheless not produced by her. She is the owner of these experiences but not the author of them (Gallagher, 2000a, 2000b; Gerrans, 2001). Similarly, schizophrenics may feel as if their occurrent thoughts are being influenced by someone else (someone else is making them think certain thoughts) or actually being inserted into their minds by a kind of psychokinesis. In the reverse case, schizophrenics may feel as if they are inserting their thoughts into the minds of others (thought broadcast).

A similar dissociation between ownership and authorship is characteristic of the schizophrenic experience of action. Schizophrenics may feel as if their body is not under their own volitional control, as if they were a kind of inert marionette moving at the will of someone else. Once again, they own these actions in the sense of knowing that the movements are movements of their own bodies. But because they are not aware of intending to perform the movements or of their voluntary control, they do not feel as if they author them. Voluntary control of action can also fail in a way that is analogous to thought broadcast. In these cases the schizophrenic may feel as if the movements of others, or in some cases of objects in the external world, are caused by the schizophrenic herself. Bovet and Parnas (1993) reported a case of a subject who thought that his urinations launched bombing raids, and the delusion that the universe is expanding or contracting according to the subject's own bodily peristalsis is well known to clinicians.

These phenomena are all "first rank" (core) symptoms of schizophrenia described by Carl Schneider as a "loss of ego boundaries." These Schneiderian symptoms concern the subject's awareness of her own cognitive or practical

agency, which seems to affect her ability to correctly discern who is the author of particular experiences/thoughts or actions.

Once the cases are described in this way, ToMM theorists claim that their hypothesis gains plausibility due to the conceptual structure of folk psychological ascriptions of agency (Frith, 1992; Leslie, 1994). According to a philosophical analysis of this structure, agency is the intentional control of bodily movement: What distinguishes an action from mere bodily movement is that the agent's intentions play a causal role in producing the action. So attributing agency involves a cognitive act of attributing intentions to the author of the relevant bodily movement, whether the author is oneself or someone else. Further, because intentions decompose into constituent beliefs and desires, attributing intentions requires a cognitive capacity to attribute beliefs about the world in which an agent's desires are to be realized, and desires to change the state of the world. The desires give the ends, and the beliefs give the means.

Support for this view comes from picture sequencing ToM tests. Schizophrenics, like autistic subjects, are good at completing sequences where the relevant movements can be captured in nonintentional terms (as in someone's being pushed off a seat and starting to cry, a strictly causal chain of events); but they are poor at completing sequences in which the story requires interpreting the characters' intentions, as in stories of deception. Further clinical and experimental data show that the schizophrenic ability to attribute agency, internal (self) or external (other), is very fragile. Perhaps then the schizophrenic makes an observation or has an experience that stands in need of belief/desire interpretation in order to be correctly attributed to an agent but lacks the cognitive capacity to do so. Hence the schizophrenic will be very poor at linking action to its governing intention in both internal and external cases, because the schizophrenic has an acquired deficit in the module that represents the constituents (beliefs and desires) of an intention.

Such a deficit might also explain so-called negative symptoms of the disorder in terms of an inability to generate the requisite intentions or to make use of them to terminate or adjust an action (failures of willed action, perseveration, Parkinsonianism). Finally, and most speculatively, if we conceive of thought itself as intentionally guided, a deficit in the device that monitors intentions might explain formal thought disorder and inappropriate affect. Perhaps the schizophrenic who initiates a train of thought "loses" access to its guiding intention, with the consequence that that sequence of thoughts becomes rambling, incoherent, or fixated and disconnected from the appropriate emotions (Campbell, 1999; Frith, 1992; Gallagher, 2000a).

The foundational idea behind all these speculations is that an agent who cannot (meta)represent beliefs and desires, the task supposedly performed by the ToM module, will be unable either to determine agency in conditions where attribution is an issue or to exert appropriate agential control in cases where she needs access to her original governing intentions. As Christopher Frith put it in

1992, "resolution may be achieved at the theoretical level if we can show that defects of will and defects in inferring the intentions of others reflect a similar cognitive deficit" (Frith, 1992, p. 122). At the time, Frith described this framework as "no doubt overinclusive." But he said that postulating a unifying cognitive domain subserved by the ToM module is the first step in a cognitive explanation of a pathological behavioral domain.

One prominent refinement of this "overinclusive" formulation owes much to Frith himself, and from our point of view, it is interesting that it involves both a weakening of the ToMM hypothesis (a dysfunctional ToM does not account for all schizophrenic symptoms) and a shift of explanatory emphasis from higher order to lower order cognitive systems. Frith now claims that an important subset of Schneiderian symptoms is likely produced at a much lower cognitive level than that initially postulated by ToMM theory: namely, the motor control system. Various "misconnection" or "dysmetric" theories of schizophrenia go even further: They explain the variety of symptoms at a mechanical level, bypassing the need for cognitive explanations at all.

It is worth pointing out that, depending on the actual mechanisms involved, these mechanical and cognitive accounts could be made to fit together. If, for example, the mechanism of misconnection is neurochemical transport between cortical areas, then one consequence might be local over- or underactivation of the premotor cortex, an architecturally discrete subsystem. As we explained in our introduction, we would still consider the ultimate cause of schizophrenic positive symptoms to be a basic noncognitive mechanism that happens to selectively affect one subsystem first en route to its more global disabling effect. Another example might be a dopamine failure in the basal ganglia, characteristic of Parkinson's disease, which, en route to its more global manifestation, produces the unusual grammatical disorders characteristic of Parkinsonians (Pinker, 2000).

We now describe the motor control and misconnection approaches in slightly more detail, showing how they explain important schizophrenic symptoms without implicating a dysfunctional ToM module.

Forward Models and the Sense of Agency

Frith originally argued that delusions of control or influence in schizophrenia are essentially failures to correctly attribute authorship of actions resulting from failures to correctly cognize intentions.

However, a more economical explanation of problems with the attribution of authorship implicates a more basic cognitive subsystem dedicated to the control of action. That subsystem, neurally realized in the premotor cortex, is the forward model for motor control.

The idea of a forward model goes back at least to the 19th century and it is captured in Figure 12.4. This model builds on the idea that any bodily move-

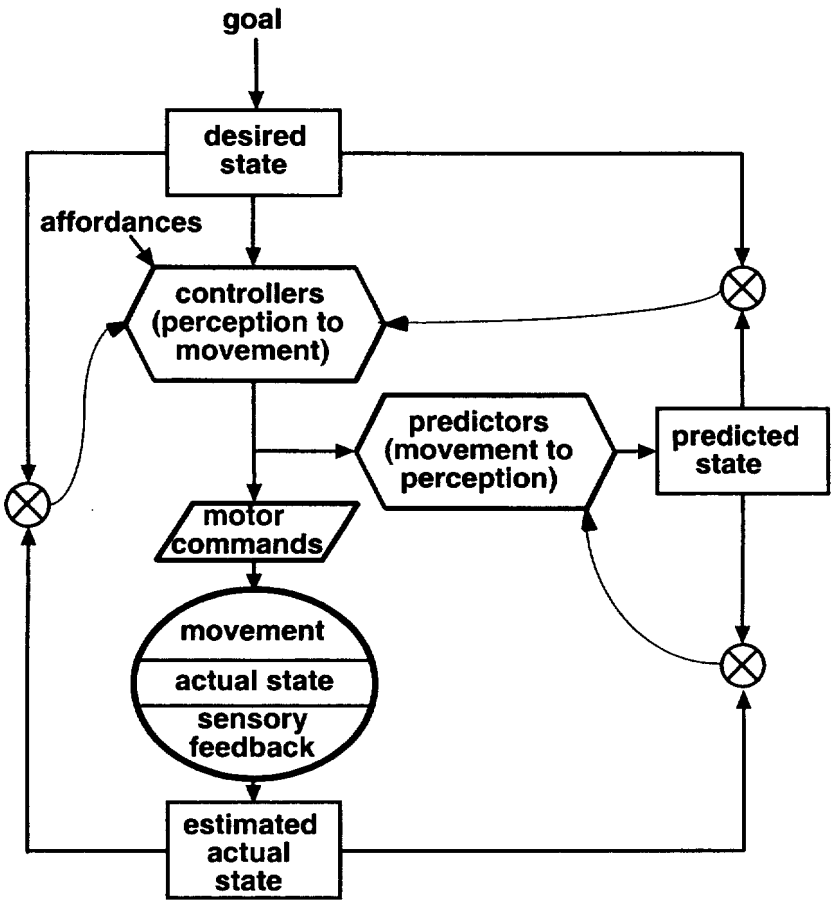

FIGURE 12.4. A forward model for motor control.

ment will generate perceptual and sensory (afferent) feedback. In order to control the movement, the system compares that feedback with a prediction about that feedback generated when the movement was initiated. When the afferent feedback matches the prediction, the system computes that the movement has been successful. When there is a mismatch, the system can send another motor instruction, which, in effect, corrects for the degree of discrepancy sensed. The system operates dynamically and in real time. For example, when someone decides to pick up a glass, the motor system must first calculate the movement(s) required to realize that goal (the desired state). The result of this calculation is the inverse model. The system then produces an instruction to the motor subsystem, which, if successfully completed, will realize the goal. A copy of that instruction (efference copy) is stored together with a prediction of the antici-

pated (bodily) consequences of the movement (for instance, that a movement of the hand 15 degrees to the left will produce a corresponding change in visual and proprioceptive information representing its orientation). This is the forward model. Now if the hand moves too far, say 20 degrees to the left, the reafferent information can be compared to the prediction, thereby yielding an error of 5 degrees. The system then generates a new inverse model, motor instruction, and efference copy to initiate and control a corrective movement of 5 degrees to the right.

The forward model is a computational solution to problems of motor control. Although not all of its elements have been decisively mapped to neural substrates, there is a large body of convergent research to show that the basic idea captures the essential cognitive properties of the motor control system (Blakemore, Goodbody, & Wolpert, 1998; Frith, Blakemore, & Wolpert, 2000; Jahanshahi & Frith, 1998; Decety et al., 1994). In particular, there is good evidence to suggest that current and predicted states of limbs are represented in parietal regions, and that actions are initiated in frontal brain regions. The most likely mechanism for the comparator system, then, is the inhibition of parietal areas by activity in frontal areas (Jeannerod, 1994).

Apart from its empirical credentials, the forward model is theoretically well placed to explain the way in which we establish ownership and authorship of actions. For any action, it will be ours if it corresponds to an efference copy that our motor system has generated (Frith, 1987; Frith & Done, 1989). Schizophrenic misattribution of agency might then arise at this level of processing in one of two ways:

Option 1: The schizophrenic performs an action but the forward model system contains no matching prediction—in such cases the schizophrenic attributes the action to someone else.

Option 2: The schizophrenic might activate the prediction system by observing the action of another. Note that this phenomenon has been established in macaque monkeys as part of the normal observation of action. It seems that merely observing an action can provoke neural activity in that part of the monkey's control system that signals that a movement is an action: namely, the forward model (Gallese, Fadiga, Fogassi, & Rizzolatti, 1996; Gallese, Fadiga, Fogassi, Luppino, & Murata, 1997; Rizzolati, Fadiga, Gallese, & Fogassi, 1996). Due to hypoactivity in the inhibitory system, or lack of connectivity between controllers and predictors, the normal signals that tell the system that the action is not self-initiated are unavailable to the system. The schizophrenic then attributes the observed action to herself (Figure 12.5) (Dapriati et al., 1997; Georgieff & Jeannerod, 1998).

These hypotheses are supported by experiments with schizophrenics who experience delusions of influence. They show unusual patterns of activity and connectivity between frontal and parietal areas and failures of attribution in motor control tasks. For example, schizophrenics report vivid sensations of alien control when asked to move a joystick. In other cases, when provided with am-

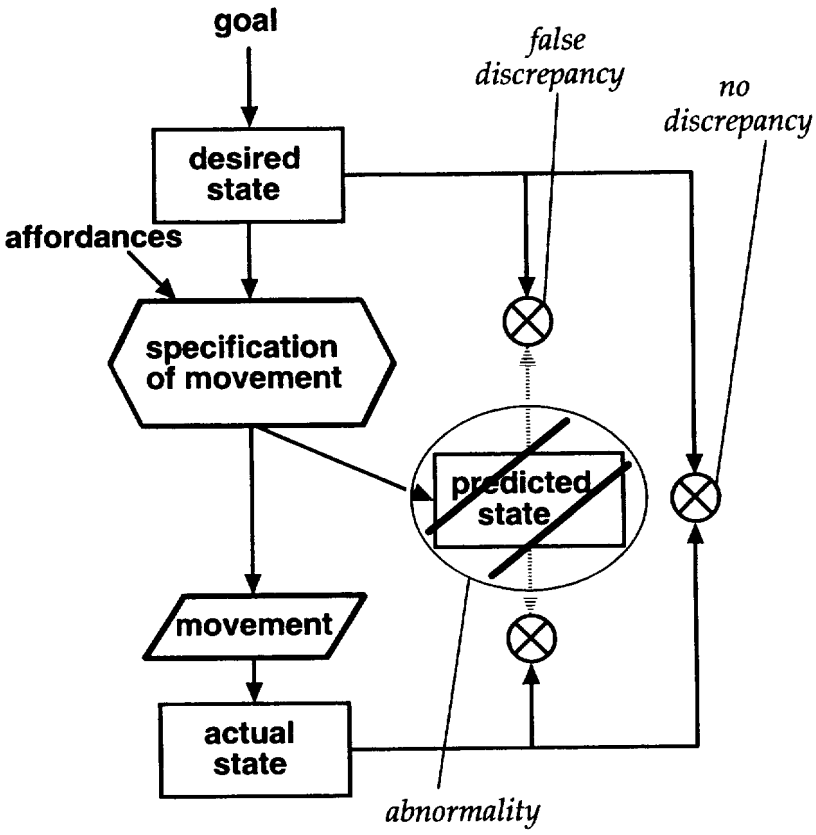

FIGURE 12.5. Delusions of control.

biguous visual feedback concerning bodily movement, schizophrenics that report alien influence are far worse than normal and noninfluenced schizophrenic controls at correctly attributing authorship (Dapriati et al., 1997; Spence et al., 1997).

Motor control theorists of schizophrenic delusion disagree over the precise details of experimental interpretation (Frith et al., 2000; Georgieff & Jeannerod, 1998). However, there is broad agreement on the idea that the primary cognitive failure is of the motor control subsystem. Over- or underactivation of implicated neural areas produces aberrant sensations of volitional control. The schizophrenic feels as if she is not in volitional control of her body or, alternatively, as if she is in volitional control of actions or events external to her. If this is correct, then the source of at least these positive symptoms has nothing to do with a ToM module but results from malfunction in a more basic sensorimotor mechanism.

If the positive symptoms of schizophrenia can be explained in this way, then it seems that a weak ToMM hypothesis has not much role to play in accounting for the disorder. After all, Schneiderian symptoms are generally held to constitute the core of the disorder. Of course, another way to defend a weak ToMM hypothesis (explored by Frith, 1992) is to argue that the core of the disorder is actually the *negative* symptoms (psychomotor poverty, avolition, apathy), now interpreted as *failures of willed action*, and attempt to explain these as a result of ToM malfunction—for example, as failures to form, metarepresent, and act on intentions. However, as we show, the negative symptoms are prime candidates for a mechanical explanation. If so, then the weak ToMM hypothesis is left without any real work to do.

In any case, to repeat our arguments made in the introduction, if positive and negative symptoms are not cleanly dissociable, this suggests that the cognitive system or systems implicated in schizophrenia are affected together. Either this is because there is only one system involved (as the discredited strong ToMM hypothesis suggested), or it is because more than one system is involved (as the weak ToMM hypothesis suggests). But then the problem becomes one of accounting for the connection between them: Why do they fail together? The most obvious hypothesis is one of mechanical failure: Some neurological process causes disruption in a number of cognitive systems, which produces the characteristic array of positive and negative symptoms. If this is genuinely the case (see the misconnection theory described next), then the explanation for schizophrenia is ultimately not cognitive at all, despite the suggestive pattern of cognitive disabilities. Most importantly from our point of view, mechanical failures of this sort dramatically undermine straightforward reverse engineering theorizing. The reason is this: Given the number of systems that are likely to be involved in any such mechanical failure, reasoning backward from a pattern of cognitive and other behavioral disabilities to the functional architecture of the system is an Herculean task. In particular, there is no good reason to assume that seemingly discrete domains at the cognitive level will correspond to actual domains in the architecture of the system. As we put it earlier, these may not be real, but only architecturally virtual cognitive domains.

Misconnection and Dysmetria

Motor control theories of misattributed agency undermine the strong ToMM hypothesis for schizophrenia by accounting for a significant—some say "core"—subset of symptoms in terms of a malfunctioning lower level sensorimotor subsystem, rather than one that is devoted to higher level cognitive processes. A second way of opposing the strong ToMM hypothesis would be more radical still, by seeking to explain schizophrenia as an essentially mechanical disorder. In this case, even if the sensorimotor system is affected, it is not a problem specific to the sensorimotor system per se—that is, a cognitive problem relating

to the generation and use of motor predictions. It is, rather, a noncognitive problem that affects, among a variety of functionally unrelated things, the generation and use of motor predictions.

Some recent theories of schizophrenia take this approach. Although differing in many details, both Nancy Andreasen and Peter Liddle share the view that schizophrenia involves disruption to a circuit integrating functionally diverse cognitive processes whose neural substrates are located in different areas of the brain. As Liddle put it, "the evidence indicates that the abnormality characteristic of schizophrenia is impaired coordination of activity at the diverse sites engaged during mental processing rather than a static loss of function at any site" (Liddle, 2001, p. 72).

For Liddle, the relevant circuit involves a series of linked cortico-striato-thalamo-cortical feedback loops whose activation affects the diverse regions implicated by neuroimaging studies of schizophrenia. He suggests that the cause of this *misconnection* is most likely dopaminergic regulation of this circuit.

For Andreasen, the explanatory strategy is to identify the neural substrates of cognitive malfunction in schizophrenia and investigate possible linking circuitry. The circuit she identifies is a cortico-cerebellar-thalamo-cerebellar-cortical loop. For Andreasen, however, the misconnection is explained as a lack of temporal integration of activation across distributed brain regions. Hence she dubs the misconnection syndrome a case of *dysmetria* (Andreasen, Paradisio, & O'Leary, 1998; Andreasen et al., 1999).

Patients suffering from schizophrenia have a misconnection syndrome that leads them to make abnormal associations between mental representations; they lack the ability to distinguish between self and nonself, and, due to an incapacity to suppress the multiple stimuli normally bombarding human consciousness, they are further unable to distinguish between the important and the trivial. In light of these difficulties, a number of problems follow: Internal representations may be attributed to the external world, leading to hallucinations. Perceptions or other information will be misconnected with inappropriate associations leading to delusional misinterpretations. Online monitoring of language or thoughts will be impaired or mistimed, leading to disorganized speech and disorganized thinking. Behavior will not be adequately monitored, leading to social awkwardness, excessive aggressiveness or shyness, or other abnormalities in behavioral activities. The inability to monitor may also lead to the "freezing" characteristic of catatonic immobility or a failure to inhibit expressed as catatonic excitement. Difficulties in inhibiting or prioritizing may also lead to the various negative symptoms such as avolition or anhedonia, much as a computer locks when it cannot match signals sent at an incorrect rate or to an incorrect place (Andreasen et al., 1999, p. 4).

Andreasen calls her theory "cognitive" rather than "mechanical." But we should note that exactly the same explanation could be offered by someone who focuses on finding the mechanical link between diffusely affected systems. The only thing missing would be the *coda* of cognitive unification provided by sug-

gesting a functional role for the linking circuit that Andreasen proposes is disrupted in schizophrenia.

Why, then, does Andreasen accord the CCTCC circuit a functional role in cognition? No doubt it is because schizophrenic symptoms seem to mark a disintegration of the mind, rather than structureless neuropathology. To account for this difference, Andreasen introduces an extra function the brain must accomplish for normal functioning—namely, "synchrony," the neural substrate of which is the proposed CCTCC circuit. Malfunction of this circuit (due to widespread mechanical breakdown) is "dysmetria."

Although we think Andreasen is right to emphasize that schizophrenia involves functional disintegration (and so, effectively, a disintegration of mind), she goes too far in supposing it needs a cognitive explanation. What distinguishes schizophrenia from a structureless neuropathology is the anatomical (rather than functional) integrity of the proposed CCTCC circuit. Schizophrenic symptoms are the result of mechanical damage to the CCTCC circuit, so are not accidentally distributed throughout the brain.

Ultimately, Andreasen's theory is eliminative of higher cognitive and intentional explanations for schizophrenia, not just because dysmetria "cuts across" cognitive explanations, but for a deeper reason shared by all eliminative explanations. It suggests that the anatomical level exhausts explanation of schizophrenic symptoms. Although Andreasen postulates "synchrony" as a cognitive function, she is simply imposing unnecessary higher order unification on functionally disparate symptoms. As a higher order cognitive concept, "synchrony" does not work, not least because it is implicated in all cognitive functions because they all involve the synchronized firing of sets of neurons. In particular, it does not suggest progressive decomposition into finer and more basic cognitive structures terminating in an implementation theory. Instead it leads straight to the mechanical level via a search for a circuit whose *location* would link all the symptoms, and an account of the functioning of that circuit not in cognitive terms but rather in the vocabulary of neuroscience. Hence this is a case where the postulation of cognitive unity is merely virtual, and such unity as the disorder possesses is at a purely mechanical level.

Our purpose here is not to argue for a particular theory of schizophrenia but to point out that persuasive accounts of the origin of schizophrenic symptoms do not require their unification at a high level. Co-occurrence of symptoms is, here, most likely the result of neuroanatomical processes that are essentially noncognitive.

CONCLUSION

Both autism and schizophrenia present a distinctive profile of spared abilities and disabilities, with "core" symptoms that invite unification in terms of discretely impaired cognitive function. By the principles of reverse engineering,

such unification suggests there may be a single underlying cognitive entity that is differentially affected. However, although such reasoning is not always fallacious, it may inhibit research into more basic underlying cognitive and/or mechanical explanations of "core" symptoms. In our view, this research becomes especially important if there is no explanation of a range of further symptoms with which these core symptoms are characteristically comorbid. ToMM theorists have generally recognized the need to widen the explanatory scope of their accounts, but have failed to do so in any convincing way. Admittedly, their proposals are often speculative, awaiting further empirical investigation. But it may be that they persist in this approach partly because it seems unlikely that such distinctive higher order cognitive abnormalities could be explained except by way of selective impairment to dedicated higher order cognitive systems. In this chapter, we have argued that such cognitive abnormalities may in fact be caused—developmentally or, indeed, architecturally—by damage to lower order cognitive mechanisms and/or mechanical malfunction, and there are good reasons to prefer such explanations for both autism and schizophrenia. In any case, we propose that theorists should only retain high-level cognitive hypotheses where these function as theoretically ineliminable constraints on implementation theories for cognitive capacities. In the case of both autism and schizophrenia, continuing research suggests that, even though ToM deficits constitute a clinically interesting set of related symptoms, their explanation will not involve a dedicated ToM module. In fact, our bet is that this module will prove to be eliminable as the deeper cognitive and noncognitive structure of both disorders becomes more fully understood.

NOTES

1. Mirroring our earlier point, because mechanical malfunction inevitably produces cognitive malfunction, some might doubt the viability of distinguishing between mechanical and cognitive explanations for various disorders. Although we think the distinction between PKU and, say, prosopagnosia is clear enough, we reinforce the idea by considering another computer analogy. Suppose some instability in the microcircuitry of a PC causes a particular, resource-hungry, program to crash first. The program is indeed a discrete computational entity identified by its selective malfunction, but the ultimate cause is not computational but mechanical. The reason is simply this: The solution is not to reinstall the program, but to fix the microcircuitry.
2. Some of the material in this section draws heavily on ideas developed in McGeer (2001) and McGeer (forthcoming). For additional arguments against the ToM approach to autism, see Gerrans (1998).
3. There are a number of variations of this task, but one simple version that has been used on autistic populations is the so-called "Sally–Ann" task (Baron-Cohen, Leslie, & Frith, 1985; cf. Wimmer & Perner, 1983): Children are shown two dolls, "Sally" and "Ann." Sally has a basket in which she places a marble. Then, she goes away

leaving her basket behind. Ann takes Sally's marble out of the basket and puts it in a box. Sally returns, and the children are asked "where will Sally look for her marble?" To pass, children must correctly predict that Sally will look in the basket where *she* believes the marble to be, as opposed to the box where they know the marble is themselves.

4. Although some studies indicate normal children may show weak superiority on false-photo tasks (Slaughter, 1998) or dissociation in the opposite direction (passing false belief and failing false photograph; Leslie & Thaiss, 1992; Zaitchik, 1990), more recent studies suggest this may be an artefact of experimental design. Normal 4-year olds do equally well on both tasks once incidental conversational and linguistic differences between them have been eliminated. Autistic subjects, on the other hand, continue to show the dramatic physical–mental dissociation seen in earlier studies (Peterson & Siegal, 1998).
5. For a defence of this perspective and for details of autistic abnormalities from which the following limited summary is culled, see the collected papers in Baron-Cohen, Tager-Flusberg, & Cohen (2000). See also Frith (1989) and Happé (1994).
6. The theoretical perspective advocated in this paper stands in some contrast to other work done by these authors. Although both have been friendly to a ToM account of social deficits, Uta Frith in particular has advocated a "weak central coherence" (WCC) view of autism in contrast to the ToM approach on grounds that the latter cannot account for autistic abnormal capacities in areas unrelated to social cognition (for instance, autistic savant talents). According to WCC, autistics lack a capacity to process incoming information in context, thereby using contextual cues to deliver higher level meanings. For an explanation and defense of this approach, see Frith (1989) and Happé (1999). At one time, Frith seemed to reject the ToM approach on the grounds that autistic assets and deficits are likely to have the same origin. Recently, she seems to have leaned more toward a multiple deficit approach involving WCC, ToM, and executive dysfunction (affecting planning, working memory, impulse control, shifting tasks, and the initiation and monitoring of action; Frith, 1997). Happé herself has also advocated something like a weak ToM (multiple dysfunction) stance in her writing: "To date, the experimental findings suggest that weak central coherence and theory of mind are somewhat independent. . . . However, it is likely that these two aspects of autism interact, and failure to integrate information in context might contribute to everyday social difficulties. Featural processing might play a part in certain social impairments. Piecemeal processing of faces, for example . . . could hamper emotion recognition" (Happé, 1999, p. 220). This theoretical vacillation with respect to ToM may well indicate that the authors have no settled views as yet, due precisely to the kinds of explanatory difficulties we are highlighting in this chapter.
7. This is not to suggest that Gopnik endorses the kind of modularity approach to ToM capacities discussed in this article. In Gopnik's view (and the view of her collaborators), theory of mind is—as the term suggests—a theoretical competence. Its acquisition is to be explained in terms of a child's native capacity for theory building put to work on progressively elaborating an innate proto-theory of agency in the face of new evidence (see, e.g., Gopnik, Capps, & Meltzoff, 2000).
8. As yet ToMM theorists give no explanation for why a late-developing ToM mechanism should not work as reliably as one that develops on schedule, other than its

lateness. Perhaps to avoid this problem, some suggest that high-functioning autistics that are able to pass theory-of-mind tests do so by means of "compensatory mechanisms"—general problem-solving capacities deployed in the social realm. However, it is hard to see how this use of "general problem-solving capacities" would not require some specialized knowledge about psychological matters, in which case the expertise of normal subjects might well be explained by the vast difference in their knowledge base acquired through years of training, rather than by the presence of any specialized mechanism for psychological reasoning.

9. It is interesting to compare these various clinical populations with Down's syndrome children who do pass false-belief tasks at the same mental age as normal children. Hence, Down's syndrome children are developmentally retarded, but they seem to follow a normal developmental trajectory (see, e.g., Baron-Cohen, Leslie, & Frith, 1985). By contrast, congenitally blind children and deaf children of hearing parents follow the same abnormal developmental trajectory as autistic children: They are unusually delayed in passing theory-of-mind tasks compared with non-social reasoning tasks. They also show autisticlike abnormalities in social, communicative, and imaginative abilities; but, as is not the case with autistic children, these abnormalities tend to disappear as they become more able to relate to others through developing skills that overcome their handicaps in a context of able and responsive others (Brown et al., 1997; Peterson & Siegal, 1998). It is also notable that deaf children whose parents are native signers, and who therefore have rich proto-conversational and conversational interactions with others from an early age, do not show any "autistic" social or cognitive abnormalities in the nature of their conversational behavior (Meadow, Greenberg, Erting, & Carmichael, 1981). In particular, they do not have any difficulty passing "theory-of-mind" reasoning tasks. These comparative results are discussed in Peterson and Siegal (1999).

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