DOES THE STUDY OF AUTISM JUSTIFY MINIMALIST INNATE MODULARITY?

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ABSTRACT: Innate modularity is a central question for cognitive neuroscience. One proposal is that a "theory of mind" is a species-specific (human) example of an innate module. The genetic, neurodevelopmental, psychiatric condition of autism is considered in terms of the innate modularity claim. At the opposite extreme, explanations of autism in terms of deficits in a general learning mechanism are considered. It is concluded that both of these theories may be untenable, and instead there may be some justification for an intermediate model of social perception: minimalist innate modularity.

The study of autism has been the battleground for modularity and anti-modularity theorists. On the side of modularity are scientists who view the theory-of-mind deficit in autism as evidence for some form of modularity (Baron-Cohen, 1995; Happe & Frith, 1996; Leslie, 1991). On the side of anti-modularity are those who only recognize the brain as a general learning mechanism (Mackintosh, McLaren, Plaisted), or those who view the theory of mind deficit in autism as part of a broader deficit in higher-order cognitive processes such as executive function (Russell, 1997a; Pennington et al., 1998; Shallice, 1988). In some senses, this battle is simply an example of the wider battle in cognitive neuroscience over whether modules exist, or what if anything is innate in the cognitive system. Although this article focuses on the arguments in relation to theory of mind being modular (using evidence from autism), readers can easily apply these arguments in relation to

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language (using evidence from language disorder) or in relation to other domains. First, we introduce autism and the theory of mind hypothesis.

BACKGROUND: AUTISM AND THE THEORY OF MIND HYPOTHESIS

Autism is considered to be the most severe of the childhood neuropsychiatric conditions. It is diagnosed on the basis of abnormal development of social behavior, communication, and imagination, often in the presence of marked obsessional, repetitive, or ritualistic behaviour (American Psychiatric Association, 1994). The theory of mind hypothesis states that children with autism are impaired in the development of the ability to appreciate their own and other people's mental states—such as their beliefs, desires, intentions, knowledge, pretence, and perception—and the links between mental states and action. Because of the importance of a theory of mind (henceforth ToM) in normal social interaction, social understanding, imagination, and communication, a deficit in the development of a ToM is a powerful way to explain abnormalities in these areas in autism. First-order theory of mind tests involve inferring what one person thinks, knows, intends, desires, etc. The majority of children with autism are at chance on such tests; 21 such tests are summarized concisely in the list overleaf.

By way of illustration, one of these is described in more detail, namely the "see-

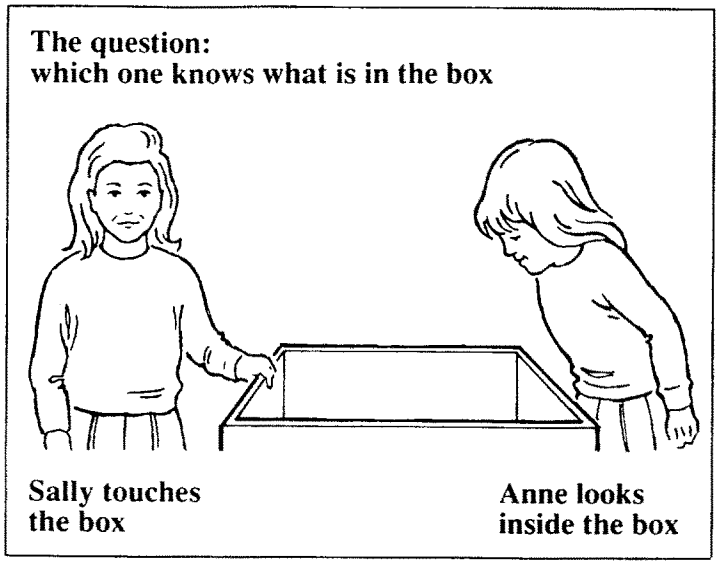

FIGURE 1
Illustration from the "seeing-leads-to-knowing test" developed by Pratt and Bryant (1990).

ing-leads-to-knowing" test. This was originally developed by Chris Pratt and Peter Bryant (1990), who presented a story (using dolls) to 3–4-year-old normally developing children. The story is short and is illustrated in Figure 1. Thus, Sally and Anne are introduced, and the subject is told that Sally touches the box, while Anne looks inside the box. The subject is then asked, "Which one knows what is in the box?" Pratt and Bryant found that normal 3–4-year-olds could correctly identify the character who knows what's in the box (in this example, Anne). If you ask the normal child of this age why he or she chose Anne, the child will typically say, "Well, she knows, because she looked." In passing this test, children are thus demonstrating their understanding of a cornerstone of their theory of mind, namely the principle that seeing-leads-to-knowing.

When this same test was given to a group of children with autism, and a second group of children without autism but with mental handicap,[1] both of whom had a mental age (MA) above 4 years old, the group with mental handicap passed the test in line with their MA, whereas the children with autism were significantly impaired on the task. Indeed, they were as likely to choose Anne as they were to choose Sally, suggesting they did not grasp this principle, and were mostly guessing. Because this deficit occurred in the presence of sufficient MA, it is taken as evidence of a *specific* ToM deficit. Most of the other results in the following list reveal a similar pattern of autism-specific, ToM-specific deficit.

LIST: 21 TESTS OF THEORY OF MIND IN PEOPLE WITH AUTISM

1. The *mental–physical distinction* (Baron-Cohen, 1989a)
2. Understanding of the *functions of the mind* (*ibid.*)
3. The *appearance–reality distinction* (*ibid.*)
4. *First-order false-belief tasks* (Baron-Cohen, Leslie, & Frith, 1985; Baron-CoThen, Leslie, & Frith, 1986; Leekam & Perner, 1991; Perner, Frith, Leslie, & Leekam, 1989; Reed & Peterson, 1990; Swettenham, Baron-Cohen, Gomez, & Walsh, 1996) (Swettenham, 1996)
5. *"Seeing-leads-to-knowing"* tests (Baron-Cohen & Goodhart, 1994; Leslie & Frith, 1988)
6. Tests of *recognizing mental-state words* (like "think," "know," and "imagine") in a word list (Baron-Cohen et al., 1994a)
7. Tests of *production* of the same range of *mental-state words* in their spontaneous speech (Baron-Cohen et al., 1986; Tager-Flusberg, 1992)
8. Tests of the production of spontaneous *pretend play* (Baron-Cohen, 1987; Lewis & Boucher, 1988; Wing & Gould, 1979; Ungerer et al., 1981)
9. Tests of understanding more *complex causes of emotion* (such as beliefs) (Baron-Cohen, 1991; Baron-Cohen, Spitz, & Cross, 1993)
10. Tests of recognizing *the eye-region of the face* as indicating when a person is thinking and what a person might want (Baron-Cohen, Campbell, Karmiloff-Smith, Grant, & Walker, 1995; Baron-Cohen & Cross, 1992)

11. Tests of being able to *monitor their own intentions* (Phillips, Baron-Cohen, & Rutter, 1998)
12. Tests of *deception* (Baron-Cohen, 1992; Sodian & Frith, 1992; Yirmiya, Solomonica-Levi, & Shulman, 1996)
13. Tests of *understanding metaphor, sarcasm, and irony*
14. Tests of *pragmatics* in their speech (Baron-Cohen, 1988; Tager-Flusberg, 1993)
15. Tests of recognition of violations of pragmatic rules (Surian, Baron-Cohen, & Van der Lely, 1996)
16. Tests of *imagination* (Scott & Baron-Cohen, 1996)
17. Performance on ToM tasks by children with autism has been found to correlate with real-life social skills, as measured by a modified version of the Vineland Adaptive Behaviour Scale (Frith, Happe, & Siddons, 1994).
18. A small minority of children or adults with autism pass first-order false-belief tests. However, these indivduals often fail *second-order false-belief tests* (Baron-Cohen, 1989b). This suggests there can be a specific developmental delay in theory of mind at a number of different points.
19. Some individuals with autism who are very high functioning (in terms of IQ and language level), and who are usually adults, may pass even second-order tests (Bowler, 1992; Happe, 1993; Ozonoff, Pennington, & Rogers, 1991). Those who can pass second-order tests, however, may have difficulties in *understanding stories* in which characters are motivated by complex mental states such as bluff and double-bluff (Happe, 1994).
20. Such able subjects have difficulties in decoding complex mental states from the expression in the eye-region of the face (Baron-Cohen, Jolliffe, Mortimore, & Robertson, 1997a; Baron-Cohen, Wheelwright, & Jolliffe, 1997b). Again, this suggests that the mind reading deficit may only be detectable in such high-level older subjects using sensitive, age-appropriate tests.
21. Parents of children with Asperger syndrome (AS), at least one of whom presumably carries the gene for AS, also show difficulties in attributing mental states when just the eye-region of the face is available (Baron-Cohen & Hammer, 1997)

This list of experiments provides strong and converging evidence for there being a theory of mind deficit in autism. For this reason, autism has been conceptualized as involving "mindblindness" *to varying degrees* (Baron-Cohen, 1990; Baron-Cohen, 1995).

BROTHERS' SOCIAL BRAIN THEORY

In an important article in 1990, Leslie Brothers suggested that social intelligence should be distinguished from other kinds of intelligence, and that social intelligence might have its own dedicated neural circuitry (involving the amygdala, orbitofrontal cortex [OFC], and superior temporal sulcus [STS]). Evidence for Brothers' proposal included the existence of brain-damaged patients who seemed to show selective impairments in social judgment, lesion studies of the nonhuman animal brain selectively affecting social behavior, and single-cell recording studies identifying

cells that fire only in response to social stimuli (such as faces and hands). Brothers referred to these three neural regions as comprising the "social brain."

LESLIE'S MODULARITY CONTENTION

Alan Leslie, independently of Brothers, suggested that there may be a particular part of the brain that in the normal case is specifically responsible for understanding *mental states*, and which is impaired in autism. He proposed this may be modular, and he coined the term "theory of mind mechanism" (ToMM) as shorthand for this (Leslie, 1991; Leslie & Roth, 1993; Leslie, 1987). The ToMM may be part of the social brain (Baron-Cohen, 1995). Note that the term "module" in this theory is not necessarily defined using all of Fodors' nine criteria (Fodor, 1983); rather, varieties of modularity are conceptualized. But critically for Leslie and others, at a minimum such modules are in part innate, and computationally dedicated or specific to a particular class of input. Leslie suggests the function of the theory of mind mechanism is to represent information in a data-structure of the following form: [Agent-Attitude-"Proposition"]—e.g.: [Fred-thinks, "The safe is behind the Picasso"]. Such a proposal is sufficient to allow representation of the full range of mental states, in the Attitude slot. Such representations allow one to make sense of why Fred, a burglar in this example, is seen as lifting the Picasso painting off the wall, or would enable one to predict where Fred would look in the house. Leslie's computational analysis has been widely accepted, but the innate modularity claim is more controversial (Carruthers, 1996; Russell, 1997b).

ANTI-MODULARITY ARGUMENTS IN RELATION TO THEORY OF MIND

Arguments against the ToM deficit in autism being proof of modularity are as follows:

1. *Circularity.* One cannot use autism as both the proof of a ToM deficit and proof of this being modular in the normal case. One needs independent lines of evidence to prove or disprove the modularity thesis. For example, finding a universal pattern of ontogenesis to the development of a theory of mind in normal development, or double dissociations between theory of mind and executive function in neurological patients, would bolster the modularity position. In fact, there is some evidence for universal ontogenetic similarity (Avis & Harris, 1991). Double-dissociation evidence is also available (reviewed later).
2. *Profligacy.* While it may seem parsimonious to explain all the cognitive evidence in autism by positing a ToM module, can this be justified given how complex such a ToM module would have to be? For example, it would have to store the complete set of mental-state concepts in advance. Anti-nativists should rightly be concerned that too much may be being built in at the innate level, with insufficient support for this.
3. *Ignoring the role of learning.* We know normal infants and toddlers spend many long hours engaged in social interaction. Is this merely for reasons for attachment and vocabulary learning, or might this be necessary experience for ac-

quiring a ToM too? If so, might ToM (modular or otherwise) be acquired rather than be innate, or (more likely) an interaction of both innate and environmental factors? The fact that blind children may be delayed in the theory of mind development (Brown, Hobson, Lee, & Stevenson, 1997), or that there are birth-order effects in this domain (Perner, Ruffman, & Leekam, 1994), suggests environmental input is important.

ALTERNATIVE MODULARITY THEORIES

1. Baron-Cohen's Minimalist Innate Modularity Theory. Leslie's claim is not the only modular theory, and we need to consider the alternatives. For example, Baron-Cohen (1994) suggests lower-level perceptual mechanisms extract relevant social information, which provide critical inputs to developing a ToM. These mechanisms include an Eye Direction Detector (or EDD), which grabs the infant's attention to the eye-region of faces and thus provides opportunities for the infant to learn the significance of gaze as a clue to a person's mental states; an Intentionality Detector (or ID), which grabs the infant's attention to animate actions, providing opportunities for the infant to learn about goal-directedness; and a Shared Attention Mechanism (or SAM), which takes inputs from the previous two mechanisms to enable the infant to work out if he or she and another person are attending to the same thing, thus ensuring that shared foci or common topics are a central experience for the developing infant. In this model, ToMM is conceived of either being a more mature development of SAM, or is triggered by SAM. We might label Baron-Cohen's model a *minimalist innate modularity theory*. Rather than having to postulate ToMM coming fully prepackaged as an innate module, this minimalist alternative specifies less that is innate—but still specifies some innate social-information mechanisms.

2. Johnson and Morton's Even More Minimalist Innate Modularity Theory. However, once one considers more minimalist versions of the innate modularity theory, one is forced to take seriously even more minimalist possibilities. Thus, Morton and Johnson (1991) do not even go as far as to suggest the existence of innate mechanisms as complex or as specific as EDD, ID, and SAM. Instead, they simply postulate a mechanism called CONSPEC, which grabs the infant's attention to look at face-like stimuli. A second innate mechanism (CONLERN) then steers the infant's attentional system to learn all about faces. On this argument, our strong intuition that looking at eyes is innate does not reflect an EDD, but simply reflects that eyes are parts of the face that move a lot, and CONLERN ensures we track such changeable social stimuli in order to learn what they predict. On this model, if we have modules like EDD, SAM, and ToMM, these are acquired, not innate, modules.

INTERIM SUMMARY

If we pause at this point, we can summarize the debate thus far: Leslie's proposal of a rich innate module (ToMM) may be simply the end result of a set of simpler innate modules (EDD, ID, and SAM; Baron-Cohen, 1995), but that even

these may be the end result of yet simpler innate modules (CONSPEC and CONLERN; Morton & Johnson, 1991). Finally, anti-nativists would argue that all of the domains of knowledge we have discussed so far (knowledge about eyes, intentional actions, theory of mind) may be modular in the sense of being domains of expertise in the normal system, but these may just as plausibly be acquired modules, not innate ones (Karmiloff-Smith, 1992).

SO WHY NOT SAY THERE'S NO MODULARITY AT ALL?

Those readers who are still engaged by this point might reasonably ask: Why stop this train of argument at an innate CONSPEC or CONLERN? Instead, one could postulate that even these may be mere end points arising out of a general learning mechanism that is working on input from this particular planet. That is, maybe there is nothing innate in cognition, and that apparent similarities in development within and across cultures simply reflect that we are all taking as input into a general learning mechanism information about the same sort of environment. Our environment, in every culture, is populated by people with faces, eyes, actions, etc.; thus, unmysteriously, this is what we end up learning about. This view is certainly proposed by the learning theorists (Mackintosh, McLaren, Plaisted). If one really wanted to strive for parsimony, is not this model of the mind (no innate structure) surely the most attractive option?

Not quite. For one thing, the no-innate modules view has a hard time accounting for cases of pure social deficit. Why should someone with good general intelligence (a good general learning mechanism?) excel in the domain of physics, or math, or engineering, and yet have difficulties in the social domain? Cases of Asperger syndrome (Asperger, 1944) may fit this characterization (Baron-Cohen et al., in press).

Further Problems for the General Learning Mechanism Theory. The general learning mechanism theorist might also have difficulty accounting for the following kinds of neurological patients:

1. Patients with severe SLI (specific language impairment) but with intact ToM suggest the potential independence of language and ToM. Van der Lely has reported one such patient (Van der Lely, 1997).
2. Patients with impaired executive function (EF) but intact ToM suggest the potential independence of EF and ToM. Some patients with Tourette syndrome meet this criteria (Baron-Cohen, Robertson, & Moriarty, 1994b).
3. Patients with impaired ToM but intact EF. Baron-Cohen, Stone, Wheelwright, and Rutherford, in press, describe a single case of a University academic with Asperger syndrome who had exactly this profile.
4. Patients with low IQ but intact ToM. Patients with Williams syndrome can show this profile (Karmiloff-Smith, Grant, Bellugi, & Baron-Cohen, 1995; Tager-Flusberg, Boshart, & Baron-Cohen, in press).
5. Patients with acquired neurological lesions who lose only mentalizing skills. Some patients with amygdala or orbitofrontal cortex lesions have been reported to show this pattern (Stone, Baron-Cohen, Young, & Calder, 1998).

The above cases strongly suggest modularity of ToM, but they are silent on the question of whether this is innate or acquired modularity.

TESTING FOR MINIMALIST INNATE MODULARITY IN THE SOCIAL DOMAIN

How might this debate be resolved through experimental investigation? The following sorts of evidence would all disprove the general learning mechanism view of the mind.

(a) If Neonates Prefer Faces. This has in fact been demonstrated over 40 years ago, and has been replicated many times (Johnson & Morton, 1991). This strongly implies an innate attentional bias toward social information.

(b) If Faces Are Processed by Specific Brain Regions. This could be acquired modularity, but if the neural specificity was also found in neonates, this again implies innate neurocognitive structure to the mind, toward social information, along the lines suggested by Brothers (Brothers, 1990).

(c) If Neonates With Autism Show no Face Preference. This has never been tested, and it would be difficult to do so until we have a biological marker for identifying neonates who will go on to develop autism. But such a finding would be problematic for a general learning mechanism account.

(d) If There Was Strong Heritability for ToM/Social Intelligence. Again, this has not yet been tested, but if confirmed this would imply that the ToM[2] deficit in autism occurs for genetic reasons. This is not implausible; autism appears to be strongly heritable (Bailey et al., 1995; Bolton & Rutter, 1990; Le Couteur et al., 1996). The idea that the development of theory of mind is under genetic/biological control in the normal case is consistent with evidence from cross-cultural studies: Normally developing children from markedly different cultures seem to pass tests of theory of mind at roughly the same ages (Avis & Harris, 1991).

(e) If There Was Neural Specificity to ToM. This could reflect acquired rather than innate specificity, unless such localization was demonstrable in infants. Which part of the brain might subserve ToM is not yet clear, though from Brothers' proposal and related work, candidate regions include the following:

(i) The right *orbitofrontal cortex*, which is active when subjects are thinking about mental-state terms during functional imaging using SPECT (Baron-Cohen et al., 1994a).
(ii) The *left medio frontal cortex*, which is active when subjects are drawing inferences about thoughts while undergoing a PET scan (Fletcher et al., 1995; Goel, Grafman, Sadato, & Hallett, 1995). The first PET study to look at adults with autism/Asperger syndrome during a ToM task shows that such patients do not exhibit the same patterns of neural activation when thinking about other minds (Happe et al., 1996).
(iii) Other candidate regions include the *amygdala* (Baron-Cohen & Ring, 1994).

Ongoing studies suggest adult patients with acquired amygdala lesions have difficulties with advanced (or adult level) theory of mind tasks (Stone et al., 1998), and a recent fMRI study of ToM using the Eyes Task found that, while normal controls used areas of the frontotemporal cortex and the amygdala, high-functioning adults with autism or Asperger syndrome do not activate the amygdala during this task (Baron-Cohen et al., 1999).

(iv) Finally, the demonstration of a joint attention (gaze-monitoring) deficit in autism, and the role that the *superior temporal sulcus* in the monkey brain plays in the monitoring of gaze-direction (Perrett et al., 1985), have both led to the idea that the superior temporal sulcus may be involved in the development of a theory of mind (Baron-Cohen & Ring, 1994).

CONCLUSIONS

The available data allow us to interpret the theory of mind deficit in autism as strong evidence for modularity of social intelligence, but may not justify a rich innate module such as Leslie's ToMM. Rather, a ToMM may be the result of both innate and acquired factors in development. When seen early in development, young children autism do not show joint attention or normal preferential attention to faces and eyes (Baron-Cohen et al., 1996; Charman, Swettenham, Baron-Cohen, Cox, & Baird, 1997), which is at least consistent with a minimalist innate modularity theory (Baron-Cohen, 1995) involving lower-level social perception mechanisms.

There is an outside chance that a general learning mechanism theory could be rescued by arguing that the normal neonatal preference for attending to faces and eyes is not a sign of a social-cognitive module but simply a reflection of the general learning mechanism preferring complexity (faces being arguably more complex stimuli than inanimate objects). This would seem to be empirically testable in principle, by matching stimuli for complexity. Johnson and Morton have, in fact, attempted to control for complexity by matching their stimuli (faces and nonfaces) for spatial frequency. It may be that there are other aspects of stimulus complexity that need to be controlled. Either way, the minimalist innate modularity theorist would predict that an infant with autism may attend to equally complex stimuli from the nonsocial domain but simply not attend preferentially to the social stimuli.

Finally, the anti-modular, general leaning mechanism theorist in contrast would predict that infants with autism would be less interested in any kind of complex stimuli, whether social or nonsocial. Such tests are for the future.

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NOTES

1. Here we use the term "mental handicap" to refer to children with general developmental delay and below-average IQ. In the UK the term "learning difficulties" is preferred,

but in the United States this latter term has a different meaning. Hence, the use of the former term here.

2. The terms *ToM* and *social intelligence* are used coterminously here.

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