Letters

Does the normal brain have a theory of mind?

Valerie E. Stone[1] and Philip Gerrans[2]

[1]School of Psychology, University of Queensland, McElwain Bldg, St Lucia, QLD 4072, Australia
[2]Department of Philosophy, University of Adelaide, Australia

Apperly, Samson and Humphreys [1] have elegantly detailed the empirical standards necessary to claim that theory of mind (ToM) is domain-specific. They argue convincingly that current evidence from research with neurological patients under-determines domain-specific claims, but do not describe the alternative to the domain-specific view. We sketch an explicit alternative, describing computational architecture that could support ToM inferences without requiring a specific ToM module. We argue that this view integrates evidence from both autism and neuropsychology more convincingly than the modular view.

ToM abilities depend on the interaction (both developmental and on-line) of domain-general abilities with lower-level cognitive mechanisms for representing social information: face processing, gaze monitoring, tracking of intentions and goals, and joint attention [2–5]. These lower-level mechanisms are *domain-specific* – restricted to social stimuli and dependent on specific neural circuitry [5]. Their normal functioning is an essential precursor to normal ToM performance [2,4,5]. However, they are not sufficient by themselves for sophisticated ToM (belief-state) inferences. The outputs of these lower-level mechanisms are used for inferences by higher level *domain-general* mechanisms: executive function, metarepresentation and recursion [4–7]. Executive function allows us to keep the elements of a social interaction in mind, and inhibit our own knowledge of the state of reality when asked about someone else's mental state [5]. Metarepresentation operating on information about eye gaze and attention (who saw or was attending to what) allows us to represent others' knowledge states (who knew what) [5]. Recursion operating on metarepresentations of mental states allows us to reason about not just others' thoughts, but others' thoughts about thoughts [7].

On our view, ToM is no more than what happens when these domain-general mechanisms interact with lower-level, domain-specific mechanisms to process social information [4,5]. Deficits on ToM tasks can result from deficits in low-level social input systems (e.g. joint attention) *or* in higher-level domain-general capacities. On this view, it should be impossible to find a pure ToM deficit occurring independently of other deficits.

Indeed, there is currently no evidence for a pure ToM deficit. Children with autism have deficits not only on ToM tests, but also in face-processing, gaze monitoring and joint attention [3,8]. Failures in low-level inputs to ToM computations could account for their deficits on these tests. Without co-morbid intellectual disability, individuals with autism seem to have intact capacities for metarepresentation and recursion, as indexed by false-photograph tests and mathematical ability [4,9]. All known cases of patients with ToM deficits arising from brain lesions involve deficits in either low-level social input systems *or* higher-level domain-general abilities. Orbitofrontal patients with deficits on ToM tasks have lower-level social deficits in face-processing and tracking intentions [5]. As Apperly *et al.* detail, medial frontal and temporoparietal junction (TPJ) patients have either executive function deficits, general metarepresentational deficits, or no ToM deficits [1].

When Baron-Cohen, Leslie and Frith published their original paper 'Does the autistic child have a theory of mind?', they argued that ToM is '*one* of the manifestations of a basic metarepresentational capacity' ([10], p. 37; emphasis added). We think it is time to recapture the insights of their original proposal, and abandon the quest for the neural substrate of the fabled ToM module. Apperly *et al.*'s analysis of TPJ patients' performance shows that it might be more promising to focus on the domain-general and uniquely human ability of metarepresentation.

References

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Corresponding author: Stone, V.E. (stone@psy.uq.edu.au).

 1364-6613/$ - see front matter doi:10.1016/j.tics.2005.11.010