accurately, irrespective of the animal's location. By contrast, place cells might mediate the association of grids to the environment, allowing the selection of grid cells that overlap at a given location to be associated with the specific sensory input available at that location via connections with a specific place cell [8].

The finding of grid cells opens up many avenues of enquiry, for example, how do grid cells and place cells together contribute to phenomena such as remapping (see Box 1), or the encoding of environmental shape [9,10]? What implications do grid cells have for the temporal (e.g. [8]) and non-spatial (e.g. [11]) processing of information in this brain region? Most importantly, the discovery of grid cells serves to deepen further the question of how these brain structures contribute to episodic memory, which remains the most obvious and yet mysterious function of this network in humans.

References

1 Tolman, E.C. (1948) Cognitive maps in rats and men. *Psychol. Rev.* 40, 40–60
2 O'Keefe, J. and Nadel, L. (1978) *The Hippocampus as a Cognitive Map*, Clarendon Press
3 Hafting, T. *et al*. (2005) Microstructure of a spatial map in the entorhinal cortex. *Nature* 436, 801–806
4 Etienne, A.S. and Jeffery, K.J. (2004) Path integration in mammals. *Hippocampus* 14, 180–192
5 McNaughton, B.L. *et al*. (1996) Deciphering the hippocampal polyglot: the hippocampus as a path integration system. *J. Exp. Biol.* 199, 173–185
6 Zhang, K. (1996) Representation of spatial orientation by the intrinsic dynamics of the head-direction cell ensemble: a theory. *J. Neurosci.* 16, 2112–2126
7 Hargreaves, E.L. *et al*. (2005) Major dissociation between medial and lateral entorhinal input to dorsal hippocampus. *Science* 308, 1792–1794
8 O'Keefe, J. and Burgess, N. (2005) Dual phase and rate coding in hippocampal place cells: theoretical significance and relationship to entorhinal grid cells. *Hippocampus* 15, 853–866
9 O'Keefe, J. and Burgess, N. (1996) Geometric determinants of the place fields of hippocampal neurons. *Nature* 381, 425–428
10 Muller, R.U. and Kubie, J.L. (1987) The effects of changes in the environment on the spatial firing of hippocampal complex-spike cells. *J. Neurosci.* 7, 1951–1968
11 Wood, E.R. *et al*. (1999) The global record of memory in hippocampal neuronal activity. *Nature* 397, 613–616
12 Hori, E. *et al*. (2003) Representation of place by monkey hippocampal neurons in real and virtual translocation. *Hippocampus* 13, 190–196
13 Rolls, E.T. (1999) Spatial view cells and the representation of place in the primate hippocampus. *Hippocampus* 9, 467–480
14 Ekstrom, A.D. *et al*. (2003) Cellular networks underlying human spatial navigation. *Nature* 425, 184–188
15 Taube, J.S. (1998) Head direction cells and the neurophysiological basis for a sense of direction. *Prog. Neurobiol.* 55, 225–256

1364-6613/$ - see front matter
doi:10.1016/j.tics.2005.11.002

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

Corresponding author: Stone, V.E. (stone@psy.uq.edu.au).
Available online 28 November 2005

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

1 Apperly, I.A., Samson, D., and Humphreys, G.W. (2005) Domain-specificity and theory of mind: evaluating neuropsychological evidence. *Trends Cogn. Sci.* 9, 572–577

2 Charman, T. *et al*. (2000) Testing joint attention, imitation and play as infancy precursors to language and theory of mind. *Cogn. Dev.* 15, 481–498

3 Dawson, G. *et al*. (2004) Early social attention impairments in autism: social orienting, joint attention and attention to distress. *Dev. Psychol.* 40, 271–283

4 Gerrans, P. (2003) The theory of mind module in evolutionary psychology. *Biol. Philos.* 17, 305–321

5 Stone, V.E. (2005) Theory of mind and the evolution of social intelligence. In *Social Neuroscience: People Thinking About People* (Cacciopo, J., ed.), MIT Press

6 Suddendorf, T. (1999) The rise of the metamind. In *The Descent of Mind: Psychological Perspectives on Hominid Evolution* (Corballis, M.C. and Lea, S., eds), pp. 218–260, Oxford University Press

7 Corballis, M. (2003) Recursion as the key to the human mind. In *From Mating to Mentality: Evaluating Evolutionary Psychology* (Sterelny, K. and Fitness, J., eds), pp. 155–171, Psychology Press

8 Schultz, R.T. (2005) Developmental deficits in social perception in autism: the role of the amygdala and fusiform face area. *Int. J. Dev. Neurosci.* 23, 125–141

9 Baron-Cohen, S. *et al*. (1999) A mathematician, a physicist, and a computer scientist with Asperger Syndrome: performance on folk psychology and folk physics tests. *Neurocase* 5, 475–483

10 Baron-Cohen, S., Leslie, A. and Frith, U. (1985) Does the autistic child have a theory of mind? *Cognition* 21, 37–46

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

Evidence for infants' understanding of false beliefs should not be dismissed

Gergely Csibra and Victoria Southgate

School of Psychology, Birkbeck College, University of London, Malet Street, London WC1E 7HX, UK

In their response to Leslie [1], Ruffman and Perner (R&P) reiterate their position that there is no need to explain Onishi and Baillargeon's (O&B) recent findings [2] with 15-month-olds in terms of attributing false beliefs (FB). Here we put forward three reasons why their points do not explain the infants' performance.

(1) We are not surprised that Leslie [3] did not respond to Perner and Ruffman's 'neurological' argument [4], according to which 'cells in the brain code for configurations of persons relating to objects'. To support their argument, they cited: (i) a neural-network model [5], which *hypothesized* rather than demonstrated the forming of associations in the prefrontal cortex between two rather than three stimulus features; and (ii) a neurophysiological study [6] showing that cells in the rat's hippocampal region are activated differently for novel and familiar arrangements of pictures, without demonstrating that those cells coded for *episodes* rather than familiarity of arrangements *per se*. Although these studies suggest that brains could form such associations, to present them as evidence for 15-month-olds forming the particular episodic three-way associations that Perner and Ruffman's account requires is unconvincing.

(2) Although we certainly agree with R&P that teleological understanding [7] could account for many examples of early competence demonstrated in infants, this is in fact a red herring in this debate. The teleological model can only take into account actual states of reality, and is therefore unable to explain O&B's result. In fact, Gergely and Csibra [7] explicitly stated that as soon as the teleological interpretation is applied to fictional states (as required by this result), it has been upgraded to mentalistic understanding.

(3) R&P ask why infants would not default to answering in terms of reality on O&B's task if, as Leslie suggests, this is what children failing the traditional FB task do. The answer to this question seems straightforward: in the looking version of the FB task [2], infants are not

Corresponding author: Csibra, G. (g.csibra@bbk.ac.uk).
Available online 28 November 2005