WHAT HAS (NEURO)PSYCHOLOGY TOLD US ABOUT THE MIND (SO FAR)? A REPLY TO COLTHEART (2006)

Richard Henson

(Medical Research Council – MRC, Cognition and Brain Sciences Unit, Cambridge, UK)

Coltheart (2006, this issue) asks a practical question about whether any neuroimaging studies to date have successfully distinguished between competing psychological theories. His conclusion is that they have not. I think this conclusion is wrong, mainly because I think he and I mean different things by "distinguish".

Coltheart finds faults with each of the neuroimaging studies that I cited in a previous paper (Henson, 2005). I do not contest some of these faults. Coltheart also presents two examples of competing theories that he believes extant psychological and neuropsychological data do distinguish. Here, I do contest his conclusions, namely that these data provide unequivocal support for one of the alternative theories. In other words, I will argue below that Coltheart's examples are not free from faults either. However, the more important point I wish to make is that I believe that datasets rarely, if ever, provide unequivocal evidence for a theory.

Perhaps I was unwise to use the word "distinguish" in the context of competing theories (Henson, 2005). I don't think (psychological) theories are refuted, in a Popperian sense, by any one datum (experiment). A cursory glance at the history of experimental psychology would appear to support this claim. Rather, theories seem to compete via a form of natural selection, in which theories that amass the most empirical support tend to be favoured (though other criteria, e.g., parsimony, clearly also apply). In this context, I see psychological, neuropsychological and neuroimaging data as equally valid contributions to this evidence, *provided* one is prepared to make certain bridging assumptions about each, such as the "transparency" assumption in neuropsychology (e.g., Caramazza, 1986), or what I called the "systematic function-structure mapping" assumption in neuroimaging (Henson, 2005).

Thus I am not going to defend each neuroimaging example in this paper (I will only discuss one of my original neuroimaging examples below, though if the reader would like detailed responses to each of Coltheart's criticisms, I would be happy to provide them in a personal communication). I am not surprised that one can *re*-interpret the findings of

these studies. What I will do, first, is address Coltheart's two examples of behavioural data that he believes do distinguish two theories. I will argue that there is not the kind of uniformity of opinion concerning these data as Coltheart might think. Thus I do not think his examples necessarily have any better "evidential" status than my neuroimaging examples. Secondly, I will correct Coltheart's description of the Remember/Know neuroimaging data of Henson et al. (1999), and clarify how subsequent neuroimaging studies have themselves re-interpreted that data. Thirdly, I will briefly re-consider the "systematic function-structure mapping" assumption, which Coltheart accepts (at least, for the purposes of his paper), and which would appear necessary for neuroimaging data to make any contact with psychological-level theories. For me, this assumption is the more worrisome aspect of the argument I made in Henson (2005), rather than the status of individual examples. In other words, while the "in practice" question that Coltheart deals with is a useful one, I am more worried about the "in principle" question of whether neuroimaging data could *ever* inform psychological theories. I will finish by mentioning a possible organisation of the mind/brain that casts doubt on this "in principle" question, not simply in terms of the function-structure mapping assumption, but also the basic logic of experimental dissociations that underlie the majority of behavioural, neuropsychological and neuroimaging research.

COLTHEART'S NEW EXAMPLES

Coltheart's first example concerns serial *versus* parallel processing in reading. According to theory T_a, print is converted to speech in parallel, whereas according to T_b, part of this conversion operates serially from left-to-right. In support of T_b, Coltheart cites: 1a) reading latencies are a function of the left-right position of grapheme-phoneme irregularities within a word, 1b) the facilitation in naming the ink-colour of nonwords is a function of the position of a phoneme in the nonword that matches the first phoneme of the colour name, 1c) priming of naming latency is a function of the

position of a phoneme-match between the (masked) prime and target words.

Contrary to Coltheart's statement that none of these effects are consistent with theory T_a, Zorzi (2000) simulated the reading latency results (1a) with a connectionist model, in which the spelling-sound conversion occurred in parallel. This model suggested that the critical factor is the consistency of the grapheme-phoneme mapping rather than the position-of-irregularity, as the two are naturally correlated (though see Roberts et al., 2003, for a counter-argument). Regarding the "masked onset priming effect" (1c), Kinoshita and Woollams (2002) have argued that it reflects speech preparatory effects (i.e., articulatory planning), which occur after the mapping from spelling to sound (i.e., after phonological assembly). An analogous masked onset priming effect would therefore presumably be predicted when naming pictures, rather than words. That is, the seriality arises as part of normal articulation, rather than during the translation from orthography to phonology. I suspect that the same articulatory contribution could also explain the nonword colour-naming results (1b).

I do not seek to argue that the data support T_a over T_b instead. Coltheart (personal communication, May, 2005) has since pointed out further evidence for T_b [e.g., the position of bivalence effect (Havelka and Rastle, 2005), the length by lexicality effect (Weekes, 1997), the length by language effect (Perry and Ziegler, 2002), and the 'whammy' effect (Rastle and Coltheart, 2000)]. It is also worth noting that there do not appear to be any data inconsistent with T_b (though there is a larger question of the resolvability of serial *vs.* parallel debates; Townsend, 1972). However, my argument is simply that there are 1) instantiations of T_a that appear to be able to explain some of the data (1a) used to support T_b, and 2) some of the data (1b+1c) used to support T_b may have causes of seriality that do not relate to reading per se. Thus, it is not obvious to me that the behavioural data "strongly favour T_b over T_a" (Coltheart, 2006, this issue).

Coltheart's second example concerns the role of semantics in reading. According to theory T_a, low-frequency, irregular words require access to their meanings in order to be read aloud, whereas according to T_b, irregular-word reading can be accomplished without accessing meaning. In support of T_b, Coltheart cites: 2a) four single-case studies of patients with impaired semantics but normal irregular-word reading. Yet data from 48 patients with semantic dementia studied by our group in Cambridge show a highly reliable correlation between the degree of semantic deficit and problems with irregular word reading (Woollams et al., 2005). This correlation is predicted by a model in which correct reading of irregular words requires the assistance of semantics, an instantiation of Coltheart's T_a (Plaut et al., 1996, simulation 4). Furthermore, though a subset of three patients initially showed normal irregular word reading despite detectable semantic deficits, like the cases cited by Coltheart (2006, this issue), longitudinal study of these patients showed that, as their semantic impairment increased, their irregular word reading became abnormal (Woollams et al., 2005). This study therefore also demonstrates the danger of extrapolating from small numbers of single-cases (if they conflict with the modal behavioural of a larger group). In any case, I would therefore argue that the neuropsychological data actually favour T_a over T_b; the opposite of Coltheart's claim.

In summary, given that we disagree over their interpretation, it is questionable whether the behavioural data from either normal adults or neuropsychological patients offered by Coltheart (2006, this issue) have "successfully distinguished" the psychological theories he describes. Note however that I would not use my criticisms of Coltheart's arguments to question the inherent value of (neuro)psychological data. In the same manner, I do not think one should use Coltheart's criticisms of my neuroimaging examples to question the inherent value of neuroimaging data. Rather, both behavioural and neuroimaging data have value, since they are both observations about the system we are trying to understand (Henson, 2005). There will probably always be competing theories that interpret those data in different ways; the most successful are likely to be those that can interpret both types of data simultaneously.

THE REMEMBER/KNOW NEUROIMAGING EXAMPLE

One neuroimaging example that I discussed at length in Henson (2005) was an event-related functional magnetic resonance imaging (fMRI) study of Remember (R)/Know (K) judgments in a recognition memory task (Henson et al., 1999). It is true that this study was not originally reported in terms of the debate between single- *versus* dual-process models of recognition memory. I applied these data to the debate subsequently, because I think the debate is a particularly pertinent example of situation where behavioural experiments have had problems distinguishing between theories: viz., those that postulate two distinct memory processes (e.g., recollection and familiarity; Yonelinas, 2002) and those that assume only a single continuum of memory strength, on which subjects place different response criteria (i.e., signal detection theory – SDT; e.g., Heathcote, 2003). As Dunn (2004) and others have shown, dissociations using the R/K paradigm that are often used to support dual-process theories can also be explained by SDT (as can other recognition memory data, like asymmetrical receiver operating characteristic –

ROC – curves, provided 'old' and 'new' distributions have different variances).

Rather, it is evidence from brain measures – particularly event-related potentials recorded with electroencephalography (EEG) – that appear to provide better evidence in favour of dual-process models (see, e.g., Rugg and Yonelinas, 2003). One reason for this may be that SDT is a theory about decisions – not memory per se – and so does not rule out the possibility that qualitatively different types of memory retrieval occur, and the subject simply combines these into a single "strength of evidence" in order to make a *n*-way decision. Thus this is a situation where brain measures may surpass behavioural measures, since they have the potential to reveal qualitatively different types of process occurring prior to (in time or space) the behavioural response, which can not be teased apart using properties of the response alone. In other words, since behavioural responses reflect the final output of a number of cognitive/neural processes, they may offer less scope for finding dissociations as a consequence of experimental manipulations.

Given this perspective, I wanted an example of a haemodynamic imaging (fMRI) experiment that addressed this debate. The argument was simply that, if R and K judgments produce a qualitatively different pattern of activity over the brain, then this is easier to explain in terms of dual-process than single-process theories[1]. The double dissociation between activity in right prefrontal and left parietal cortices that I described supports such a qualitative difference. Thus the Henson et al. (1999) study provided a particularly appealing example with which to illustrate the logic of the type of inference one can draw from neuroimaging data (what I called a 'function-to-structure deduction' inference; Henson, 2005).

Coltheart (2006, this issue) observes that in a subsequent fMRI study (Henson et al., 2000), we described a modified "single-process" model that could explain data from that study, which used confidence judgments, as well as the data from our previous R/K study. The basic idea was that, while the parietal activity might reflect memory strength, the prefrontal activity actually reflects "monitoring" or "checking" (Burgess and Shallice, 1996) that occurs when memory strength is close to response criterion, which is more likely for low than high confidence judgments, and for K than R judgments (given that R judgments are normally associated with high confidence). Coltheart (2006, this issue) mentions this subsequent study only because I noted it in my original paper (Henson, 2005).

However what Coltheart does not note is that this modified "single memory process" theory is yet another, new theory (T_c): it still has two processes, it's just that only one is specific to memory. It does not correspond to the original T_b, in Coltheart's definition. Rather, it represents the type of extension that a single memory-process theorist might appeal to, in order to explain the neuroimaging findings. Given that my main point in this article is that no dataset is definitive (see above), the fact that an alternative theory can be constructed to explain the original R/K example is not surprising. In this situation – where the data previously explained by one theory is also explained by a new, alternative theory – the solution is simply to design yet further experiments that directly contrast the old and new theory. This iteration characterises experimental science. In this particular context of recognition memory, a number of yet further neuroimaging studies have dissociated prefrontal activation (which, indeed, appears to reflect decision processes; e.g., Herron et al., 2004), perirhinal deactivation (believed to reflect familiarity, and not apparently influenced by factors like confidence that affect decisions; Henson et al., 2003) and hippocampus, posterior cingulate and inferior parietal activations (believed to reflect recollection; Yonelinas et al., 2005). These latter dissociations between perirhinal cortex and hippocampal/parietal cortices, having controlled for decision processes, continue to favour the original, dual-*memory*-process theory (T_a) over the single-process theory (T_b), or various extensions thereof (T_c).

THE SYSTEMATICITY ASSUMPTION

Note that Coltheart's criticisms of my neuroimaging examples were phrased at the psychological level, concerning, for example, limitations in the particular experimental conditions compared, or precise predictions of the relevant theories. Another way to question the value of neuroimaging data would be to question the "systematic structure-function mapping" assumption (Henson, 2005), namely that the same psychological process cannot be associated with different patterns of brain activity within an experiment (what I called the 'weak' form of systematicity), or that there is a one-to-one mapping between a psychological process and a specific network of regional activities (what I called the 'strong' form of systematicity). In other words, one could also question my examples in terms of how psychological functions are implemented in the brain, giving reasons why the neuroimaging data are not systematically related to the psychological processes engaged (even those processes according to Coltheart's re-interpretations of the experimental comparisons).

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[1]Contrary to what Coltheart (2006, this issue) states, the single-process theory, T_b in this case, does not predict that patterns of brain activity will be identical. Rather, though brain regions associated with memory strength in a single-process model might be more active for R than K judgments, this would correspond to a quantitative, rather than qualitative, difference over the brain: a distinction I defined in some detail (Henson, 2005).

My argument (Henson, 2005) was that it may be impossible to validate the systematic structure-function mapping assumption with neuroimaging data alone (though the combination of neuroimaging data with neuropsychological data, or data from transcranial magnetic stimulation – TMS –, may be more fruitful in this regard). Rather, it becomes a working assumption, whose validity is best judged by the success of the neuroimaging field as a whole (analogous to the working assumptions in neuropsychology, e.g., that the behavioural deficits following brain damage reflect the dysfunction of one or more components of the 'normal' cognitive system, rather than, say, compensatory strategies adopted by the patient, or the re-wiring of a completely new system). Such assumptions are not uncommon in science (Henson, 2005).

However, there are additional assumptions underlying my original argument that I did not make explicit. These concern the basic logic of most psychological experiments, specifically the logic of experimental dissociations (as has been considered at length in neuropsychology; see, e.g., Shallice, 1988). Though not unique to neuroimaging, it is these assumptions that I think are most vulnerable to criticism, particularly when one considers the brain as a complex, nonlinear dynamical system.

THE BRAIN AS A NONLINEAR DYNAMICAL SYSTEM

Most psychological theories, or cognitive models, are based on some form of modularity. Variants of this assumption, and its consequences, have been discussed by many people (Simon, 1969; Posner, 1978; Chomksy, 1980; Marr, 1982; Fodor, 1983; Shallice, 1988; Farah, 1994). For present purposes, modularity can be viewed in terms of several claims. The first is that behaviour is decomposable into a set of hypothetical component processes (the basis of cognitivism). A second claim is that these components can, in principle, be modulated independently, e.g., by an experimental manipulation, or by a brain lesion. A third, related claim is that modulation of one component does not directly affect other components (the 'locality assumption' rejected by Farah, 1994), or at least does not affect their basic functionality, even if it reduces the efficiency of their functioning and hence that of the system as a whole (what Posner, 1978, and Shallice, 1988, call 'isolable subsystems'). A fourth claim (often rejected explicitly, but assumed implicitly) is that information flows in one direction through these components, e.g., from stimulus to response [there may be other desirable criteria, such as 'domain-specificity', 'information encapsulation', 'innateness' (Fodor, 1983), but these are less relevant here)].

I think cognitive psychology would find it difficult to make headway, as an experimental science, without the first or second claims, so they are assumptions I am willing to make. I find the third and fourth claims more worrisome, given that the brain is clearly a highly interconnected system, including immediately recurrent connections. Nonetheless, my response is, as mentioned above, a pragmatic one: the validity of these assumptions is best ascertained by the success of the field as a whole, i.e., whether cognitive psychology converges on a number of successful theories.

Some people however reject all of these claims. Uttal (2001), for example, while accepting Fodor's claim that some "early" sensory and motor processes may be modular, doubts that higher cognitive processes are. van Orden and Paap (1997) criticise what they call the "Doctrine of Single Causes", in which behaviour is explained in terms of the linear supposition of the effect of each component (plus noise). This doctrine entails what they call "domino causality", such that a stimulus causes a response via a sequence of unidirectional causal links between components. They state that isolating single causes (components) by experimental manipulations is only a consequence of assuming their existence in the first place. Instead, they propose that the brain is better understood in terms of "reciprocal causality", in which every component affects every other component, such that their contributions cannot be determined from the behaviour of the system as a whole. In this case, any difference between two experimental conditions that is believed to modulate a single component actually reverberates throughout the system and affects all components (a violation of the 'pure insertion' assumption that I discussed in Henson, 2005). van Orden et al. (2001) extend this argument to question the logic of double dissociations that form the bedrock of much (neuro)psychological theorising.

Another way of phrasing these arguments is that the brain is a complex, nonlinear, dynamical system (e.g., Kelso, 1995). While these arguments have some force, the problem I have is that they would seem to make conventional experimental investigation difficult. At its essence, the experimental method requires manipulation of one variable, while controlling (holding constant) other variables. One assumes that the same result would obtain had the experimental manipulation been performed while the other variables held different values. If one is not prepared to make this assumption for some variables, one can manipulate them factorially and test for interactions [the logic

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[2]Contrary to my perhaps optimistic hopes, van Orden and Paap (1997) also claim that: "Research programs based on this doctrine have failed to converge" (p. 93). Even more depressingly, they state that "... the conclusion of our critique does not require a serviceable alternative to the doctrine of single causes" (p. 93). For a more optimistic view however, see the excellent article by Bechtel (2002).

that Sternberg (1969) popularised for reaction times – RTs]. Clearly the method is limited in that no experiment can manipulate every possible variable (even when restricted to those that are relevant according to current theories). Nonetheless, despite the fact that the world (as well as the brain) is a complex, nonlinear dynamical system in which such context-dependent interactions would be expected, the empirical method has proved successful: for example, in isolating Newton's laws of motion in classical physics. It is only via reducing this complexity of the world by careful experimental control in the laboratory (e.g., dropping objects in a vacuum), that these laws were verified. Or consider an example from the biological sciences: the cell is clearly a complex, non-linear, dynamical system, and yet the first step towards understanding how the cell works was to isolate its components (see Bechtel, 2002, for further philosophical/historical arguments for the success of decomposition in the biological sciences).

In other words, some natural, complex nonlinear dynamical systems would appear amenable to conventional experimentation. Perhaps this is more difficult for the brain, but if so, what are the alternative scientific methods? Clearly mathematical methods exist for making general claims about complex systems, and these have been applied to some neural and behavioural data (such as the fractal dimension of EEG data associated with epileptic activity; e.g., Pijn et al., 1997), or to make general claims about the brain (e.g., Friston, 1997). When analytical solutions to such mathematical descriptions become intractable, one must resort to numerical solutions, i.e., computational modelling. Artificial neural network models are one example, which can incorporate nonlinearity and recurrency (see examples by Zorzi, 2000, and Plaut et al., 1996, cited earlier). However, note that the behavioural data they fit are often collected using conventional dissociation logic. More importantly, the networks tend to have some internal structure, either imposed by the modeller (such as separation of orthography, phonology and semantics in the Plaut et al., 1996, model) or acquired during training. Though these components (different layers of units) are still highly interactive, they at least open the door to separable experimental modulation, or lesion, of just one component (as in the first and second claims associated with modularity above).

To return to Coltheart's paper, I reiterate that the above considerations of modularity apply equally to psychological, neuropsychological and neuroimaging experiments, so cannot be used to criticise neuroimaging independently of cognitive psychology or neuropsychology. I am not certain how to address them satisfactorily, since I have difficulty reconciling basic neurobiology and complex, nonlinear dynamical systems with the modularity assumption and the basic experimental method. But perhaps I best leave such philosophical musings to the professionals (e.g., Bechtel, 2002; Uttal, 2001). Besides, I have experiments to run.

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Richard Henson, Medical Research Council (MRC), Cognition and Brain Sciences Unit, 15 Chaucer Road, Cambridge, CB2 2EF, UK.
e-mail: rik.henson@mrc-cbu.cam.ac.uk