3 Animal cognition and human personality

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Reinforcement Sensitivity Theory (RST) is based, both in terms of data and concepts, on the experimental analysis of (non-human) animal behaviour. Some workers in emotion and human personality research question its validity on this count alone. But, since Galileo and Copernicus, our world has clearly not been the centre of the wider universe. Likewise, since Darwin, biology has accepted the essential continuity between our own and other species (with chimpanzees, genetically speaking, being 98 per cent human), where the human form and characters have not been the goal of evolution. In this chapter we evaluate the claim that emotion and personality, nonetheless, remain distinct from the rest of biology; that with them it is still the case that 'the only proper study of mankind is man'. This will clarify the foundations on which RST rests.

Emotion and personality can be approached from more 'cognitive' (e.g., Matthews, chapter 17) or 'biological' (McNaughton and Corr, chapter 2) perspectives. But these are really different sides of the same coin. There are cases where cognitive or biological factors may seem relatively more important. But both kinds of case exist. This requires a true theoretical and empirical integration to take a combined 'biocognitive' perspective. Each approach fills the gaps left by the other. Their combination leads to a richer picture and a deeper understanding.

To many this is so obvious that to argue is to attack a straw man. But resistance to the idea of non-human cognition, or that human emotion and personality are just like other animals, while tacit is widespread. It is fuelled by a range of anthropocentric fallacies that die hard. But neural and behavioural homologies across species have illuminated much of human cognition, emotion and personality – perhaps nowhere more so than with RST.

A focus on language or surface aspects of cognition can blind us to this fact. But, after many years in the scientific wilderness, emotion (which in terms of everyday application is the most important aspect of

psychology) is now receiving due attention and highlighting biological factors. But many assumptions and practices still remain that prevent integration of cognitive and biological constructs. RST provides a framework for such integration.

The question 'What is an emotion' (James 1884) is now tractable. Biological, and particularly evolutionary, analyses (McNaughton 1989) explain human emotions through the immense commonalities in internal and skeletal behaviours as well as through illuminating differences. Darwin (1872, reprinted 1965) devoted a whole book to the functional analysis of emotion and much more is known now.

We expose, below, fallacies that are a barrier to the wholesale transfer of the biological analysis of emotion and personality to the human species. For many these are so obvious that our treatment will seem otiose. But there are still others who view the role of 'pure cognition' as so central to human psychology as to make biology irrelevant, or at least a second-best level of analysis. But, even were cognition independent of emotion, it is not hardware-independent. Perceptual illusions result from processes such as lateral inhibition that can be exemplified in organisms as different from us as the horseshoe crab *Limmulus*. Human cognitive processes depend on neural mechanisms and are evident in behavioural data that are also general to non-human species. Pharmacology and imaging technologies (and on occasion more invasive techniques) confirm this generality directly (Gray and Braver 2002).

Neural mechanisms can be too low a level of analysis for some behaviours in any species. The psychology of non-human cognition can often provide a clearer picture of the psychological fundamentals of human cognition than analysis restricted to humans and particularly analysis restricted to the verbal channel. Indeed, Charles Darwin distrusted human verbal behaviour and preferred observation of non-human animals as being 'less likely to deceive us' (Darwin 1965).

'Behaviour analysis', or at least radical behaviourism, can also be blamed for a lack of interest in non-human animal cognition. Simplified artificial laboratory environments and limited ranges of measured behaviour made the rules underlying behaviour easier to discover. Even if we allow that animals could have cognitions, many of us were brought up with the idea that, 'according to behaviourism, the job of psychology is to specify the relationship between some physical event and the response, without reference to mental processes' (Dickinson 1980). But to insist that you should only use behaviour as *evidence* is not the religious aberration of radical behaviourism. Rather, this held that all you

could *talk* about was behaviour – an obvious error to either a cognitive or a biological psychologist. But most theorists now believe that observing behavioural changes does not commit one to a strict behaviourist perspective, radical or otherwise. Mental processes can, and should, be inferred from behaviour (Dickinson 1980). Scientific psychology, except where it embraces neuroscience, must draw conclusions from behavioural observations, verbal and non-verbal. Where it does embrace neuroscience it is embracing even more the doctrine that we can use external observables to deduce 'what is going on inside the head'.

Conversely, an aversion to radical behaviourism can also blind one to the fact that the behaviour analysts' own data is often the best evidence for changes in internal cognitive structures (see Fallacy 8 below). More importantly, the very artificiality of the procedures used was specifically designed to eliminate species-specific behaviours and discover species general rules. So, despite the behaviourists' distaste for cognitive constructs, they were in fact analysing species-general cognitive processes. Such processes would be expected, given phylogenetic continuity, to form the basis of human cognition whatever species-specific additional processes might also be involved. 'Cognitivism', then, can ignore the importance both of behaviour (or neural activity) as fundamental evidence and comparative analysis as the key to unlocking function.

The focus of this chapter, then, is on how far human cognition can be expected to conform to commonalities in non-human cognition. Given the assumption that the human species will have conserved functions that are generally conserved across other species, the details of such clear specific differences (e.g., colour vision), as can be found when selected pairs of species are compared, can be dealt with later as special cases.

Fallacies: biology or cognition

We suspect that the following fallacies underlie the separation of biological and cognitive constructs in psychology in general and personality psychology in particular. Each may seem a straw man but, inside them, we believe are the bones of strongly-held implicit positions that are seldom enunciated, but frequently distort reasoning – and so the type and direction of research carried out in the psychology of emotion and personality.

The root source of many of these fallacies is Cartesian dualism. This still reigns (albeit silently) in many areas of psychological science. Dualism allows a disregard for the central tenets of Darwinian biology

and promotes an implicit anthropocentrism that supports major theoretical divides in psychology. The arguments advanced here adhere to two fundamental beliefs in biology: the continuity of species implied by Darwinian evolution; and the mapping of mind to brain as different levels of description of the same fundamental entity. Mind is not here identical to brain. It is a property of brain *processes*.

Fallacy 1: primary anthropocentrism

Although seldom nowadays stated in its raw form, anthropocentrism is often at the core of many of the other reasons given for treating human beings as categorically different in some specific way from 'the animals'. Explicable in terms of ingroup-outgroup bias or human egocentrism, it is no better a basis for modern psychological science than it was for astronomy in Galileo's time.

The human species is, of course, unique. But, for scientific purposes, all species are unique (at least in some important respects). The general argument against anthropocentrism is that there is no combination of characters that would truly set us apart from all 'the animals'. Specific more or less unique characters can, of course, be found individually on a species-by-species basis. But no character sets us apart from other animals in a way that other characters do not set each species apart from all others (Macphail and Bolhuis 2001).

The general argument for inclusivity is Darwinian. Genetically and ontogenetically, human beings are just one part of a massive radiation of species in time where unique characters are dwarfed by massive cross-species communalities in the majority of characters. Species-specific differences can be important – these are often much more marked along the phylogenetic scale (e.g., echo-location in the dolphin) – but such differences are best characterized *after* a proper scientific account of cross-species communalities. Such communalities are particularly strong for the systems controlling the fundamental emotions, and individual differences in these systems (ex hypothesi, personality). Cognition is just one (albeit important) component of these systems.

> Love and hatred are common to the whole of sensitive creation, [and] their causes ... are of so simple a nature that they may easily be suppos'd to operate on mere animals. (David Hume, (1739/1985) *A Treatise on Human Nature* (London: Penguin)).

Truly unique characters (as opposed to character combinations) are also extremely rare and the number of nominally unique human characters much fewer than is commonly believed (McNaughton 1989; Macphail

1996; Cahill, McGaugh and Weinberger 2001). For example, tool use (Tebbich, Taborsky, Fessl and Blomqvist 2001); language capability (Macphail 1996); and, even culture, a higher order externalization of our cognitions (Rendell and Whitehead 2001; Pepperberg and Lynn 2000; Whiten 2001) all appear to occur in other species. Chimpanzee cultures reveal 'patterns of variation far more extensive than have previously been documented for any animal species except human' (Whiten *et al.* 1999, p. 682); and, as a result, chimpanzee cultures cannot be cleanly separated from human cultures (Boesch and Tomasello 1998). In this respect, the orang-utan appears truly 'the old man of the forest' (Smuts 2004). Cross-species continuity seems to be the rule; it is not the exception.

Fallacy 2: cognitive anthropocentrism

We may accept that humans are 'just an improved form of ape' but feel that for emotion and personality the nominal 2 per cent difference between human and chimpanzee genomes is sufficient justification for a belief that we have cognition and they do not – or, at least, a sufficiently developed cognitive system useful for understanding human data. To disavow anthropocentrism may seem, then, to reject a role for cognition in emotion and personality. To the contrary, cognition is seen as central to emotion and personality in all species. Arguably the role of cognition will be better understood (especially in terms of mechanism) when it is tied to biological, and particularly neuropsychological, constructs. A cognitive psychology without neurobiology lacks any connection to the rest of science – and little chance of making sense of underlying processes. But this is not to argue for a pure reductionism. A neurobiology without cognition would have little relevance to psychology – and indeed little way of making global sense of its detailed findings. What is required, then, is parallel, interlinked study at the cognitive and neural levels. For many phenomena, translation between them will require an additional sub-cognitive level (Smolensky 1988) of which 'neural network' models are the most obvious example. One of the strengths of RST is its explicit attempt to achieve this goal (see Pickering, chapter 16).

Of course, some specific human cognitions may be qualitatively different from those of *all* non-human species. But this is just the standard caveat for all generic biological research that each species differs somewhat from others. Human cognitions depend on the same fundamental neural machinery as those of other animals. There are massive similarities in organization, components, pharmacological responses and many other features of the brains of mammals as a class.

Certainly, there are constraints on cognition that derive directly from biology (you cannot comprehend the aesthetics of colour if you are born completely colour blind), but the influence of biology goes beyond mere constraints to the very structure and processes of cognition.

In a fundamental sense it can never be proved that different species have the same kinds of cognitions, however close their behaviour or neural machinery is shown to match. But to argue this is little short of a counsel of despair. Using this criterion, we can never prove that your cognition is the same as mine, or indeed that you have cognitions as such at all. On such a view there can be no 'cognitive science'. One might counter this by referring to the similarities in neural structure and behavioural phenomenology between individuals. But this argument for commonality holds equally well for the relations *between* species.

To take a biological perspective is not to imply that all aspects of cognition can be understood biologically. Some aspects of the cognitions of *any species* can only be understood in terms of their historical and social context. Many anthropologists would argue that different peoples have different cognitions by virtue of their different cultures. For example, the Yanomamo Indians, who live in Southern Venezuela and the adjacent portions of northern Brazil, like to be described as the 'fierce people' and are often at war with neighbouring tribes (Chagnon 1977). They have their own customs, marriage system, hierarchy, etc. and their lack of material culture is more than compensated by the richness and complexity of their theological concepts, myths and legends. Their ideas of the cosmos, origin myth, the soul and shamans are very unlike that of the high-rise Manhattan dweller. There is no merit in trying to explain these differences of cognition in biological terms.

However, such salient differences in superficial content mask deeper similarities that are taken for granted: they have a marriage system, they have hierarchies, they have an origin myth, and they have shamans. These in turn mask even greater underlying similarities in processes: in how cultural information is transmitted; how facts about the world are learned; and how facts and beliefs can be distorted. In the specific case of associative learning, the basic underlying processes of neural plasticity are likely to be identical across all species. In what *fundamental* ways are the Yanomamo-Manhattan cultural differences qualitatively distinct from the cultural differences (learned patterns of behaviour) observed in isolated populations of chimpanzees? Or, for that matter, do they differ in their means of transmission and implementation from the cultural differences observed in killer whales? (Whales teach their young the best method to ride the surf onto, and off, the beach; they teach them to

attack prey, and to recognize the characteristic sound of engines of tuna-trawlers for an easy meal.) They *must* have the mental processes that we like to refer to as cognitions in human beings.

Of course, the particular stimulus (perhaps something you just read) that causes your emotion will not produce the same result in other animals. But that is equally true of human animals who speak a different language, or of any individual of any species that merely had different life experience, or indeed yourself on different occasions. Despite such variations in the effects of eliciting stimuli, however, brain activation is similar across individuals and occasions with different elicitations of the same basic perceptual or emotional state. Importantly, the brain activation associated with any particular state in human animals occurs in homologous parts of the brain in non-human ones.

Our concern should not, then, be with these superficial differences, which are often highly salient in the human animal, but are dependent on individual history. Our concern should be with the more general underlying cognitive processes that are common to cultures and that are tied to neurobiological processes. The stimulus that elicits your fear may be arbitrary but the processes required to make that stimulus fearful are quite general and the fear itself is a stereotyped compound of cognitive, affective and bodily tone. At this level of description, the marked similarities between non-human and human animals are obvious.

Fallacy 3: language is cognition

It is possible to accept that the neural organization of cognitive centres and so cognitive processes is the same across species but nonetheless argue that the content of those cognitions is categorically different because of some emergent property of human cognitive processing – in particular, language.

However, this view is a result of the fact that the *discussion* of cognition is necessarily verbal and that any inherently non-verbal aspects of cognition cannot easily be shared. Cognitions are also often thought to be most easily manipulated and assessed in humans using the verbal channel. (It should be noted, however, that delusions and phobic cognitions are more frequently altered by guided experience or drugs than by rational argument or persuasion.) Cognitions tend, therefore, to be perceived by those who analyse them as necessarily or at least substantially verbal – and nothing approaching our level of verbal ability appears to be present in the standard laboratory subject, the rat.

When linking panic disorder with the periaqueductal grey, one of us (McNaughton 2002) found it convenient to say that 'where [panic disorder] does present, the most dramatic associated cognition, 'I am going to die' is what we would expect if a normal person were very close to a predator. This type of situation provides an animal analogue, e.g., a rat faced with a cat, that is sensitive to panicolytic drugs'.

For one neurobiologist of our acquaintance, at least, this labelling of the rat's cognition as 'I am going to die' looked like a joke or at least a poorly-chosen metaphor.

Certainly, a rat does not have the equipment to verbalize its cognition. But many would agree that the rat nose-to-nose with a cat would, in a sense *feel* very much like you or I would if we were nose-to-nose with a lion. The rat, like us, would experience an immense compulsion to leave the vicinity, coupled with palpitations, release of adrenaline, etc. But, equally importantly, if you have ever been in a truly threatening situation you should agree that, like the rat, you would not verbalize sub-vocally 'I'm going to die'; indeed verbalization of any sort would be blocked by the racing of your pure thoughts. 'I'm going to die' is merely how we interpret and describe the feeling, post-mortem as it were. The phrase is then, in our language, the closest we can get to what would be running through our heads at that precise moment. But, what was running through the animal's head would not be language, irrespective of whether the animal is human or non-human. *Talk* about emotion is not the same thing as the emotion itself.

Nor is emotion necessary to demonstrate the uncoupling of thought and language. We can certainly verbalize silently, and run verbal arguments through our head. But a moment's consideration will usually allow us to realize that the thoughts behind a normal argument run much faster than the words used to express it. Indeed, multiple strands of possible upcoming lines of argument can be run through our mind in the time it takes for one line to be verbalized. There is even imaging evidence that we make decisions hundreds of milliseconds before we are consciously aware of them (Gray 2004) and this conscious awareness must be more hundreds of milliseconds ahead of verbal translation of the associated cognition.

It is also worth noting that there are mental operations that cannot be verbalized, which we would label as 'cognitive'. Mental rotation is one example. One can describe the phenomenon to someone else: it involves deciding which of two mirror images matches a copy of one of them displayed rotated with respect to them. But *doing* mental rotation does not involve verbalization. The evidence is that it involves the actual

rotation, in the brain, of the test image – a rotation that occurs at a fixed rate and is totally unconcsious.

Modern cognitive psychology does not, of course, equate cognition with words. But we have dealt with this fallacy at length because so much manipulation of human cognition is based on word usage. For example, *cognitive*-behavioural therapy not only stresses the importance of thoughts, attributions, etc., but typically operates by trying to alter attributions via verbal routes (but not, be it noted, by logical argument). Arguments for the uniqueness of humans also very seldom focus on non-verbal examples.

Let us take more complex emotional phenomena, compulsions and obsessions, which when they are excessive in frequency or dysfunctional in type, allow diagnosis of Obsessive Compulsive Disorder (OCD). Many people would grant that a rat could have a compulsion (e.g., highly repetitive risk assessment behaviour in the absence of a predator); but could the rat have an obsession? Obsessions are 'a bridge too far', even for some strictly neurobiological psychologists. However, much of the rat's information processing could be 'obsessive' in the same sense that a human being's would be. The rat would attend selectively to possible sources of danger; other potentially important stimuli in the environment would be ignored; and usual motor plans would be disrupted. All of this could occur in the absence of overt behaviour. The parallel between rat and human seems fairly obvious with functional obsessions (such as the checking of the whereabouts of offspring). But even dysfunction is not specific to humans. Experimental neurosis can be produced in other species in much the same way as post-traumatic stress disorder in human beings. In a rat, this would disrupt cognitive processing producing a mental life that was 'obsessed' with threat. The fact that the rat cannot talk about being obsessed is irrelevant: if we are to restrict cognitive processes to verbalizations then we would have to eliminate, as we have seen above, many of the most complex human cognitions! Certainly, with OCD, the source of the obsession in the initial stages of the disease appears senseless, intrusive and non-verbalizable. It is only when we try and analyse the condition that words become involved. Even here, words are usually chosen as the cheapest means of assessment rather than the best.

Thus cognitions proper are internal and silent despite the need for verbalization *or some other behaviour* as evidence for their nature and existence. As evidence of internal states, verbal behaviour may be no better and no worse, in principle, than any other kind of behaviour. Internal verbalization may even work to deceive us about the true nature of our own cognitions, especially when those are pathological.

Cognitions are pre-verbal and indeed, often, pre-conscious (Velmans 1991) and pre-volitional (Libet 1985).

Fallacy 4: cognitions are emotionally neutral

Even if one is not thinking in terms of verbalization, cognition (and particularly 'reason') can seem peculiarly human. Cognitions are seen as pure, valence-free, ideas. We would not expect rats to indulge in mental arithmetic. But, because we cannot use the verbal channel, non-human animals must be judged by their behaviour and this can be viewed as solely the result of emotion. This allows us to believe that humans are the only possessors of pure, emotion-free, cognition.

But perception and action are more mixed in the brain than this account allows. Ironically, it is the study of the perception and production of language that has produced some of the clearest evidence for perception and action being quite generally intertwined (MacKay 1987).

It also appears that language originated more as an efficient way of communicating emotions rather than pure ideas. Monitoring of conversations in a university cafeteria, peopled by university students and academic staff, has shown that the vast majority of language is used to determine who did what and with what and to whom and not to discuss emotionally neutral concepts (Dunbar 1992). The world still awaits a TV channel that screens only neutral information (all 'information' channels are contaminated by infotainment).

Verbalization, then, appears to have evolved as a specialization of emotional expression – essentially a form of grooming (Dunbar 1992) – that has only then been exapted to allow less emotionally laden communication. This idea is consistent with the fact that the brain areas devoted to the understanding and production of language in the dominant hemisphere are devoted to the understanding and production of emotional expression in the non-dominant hemisphere. The latter are also fully capable of supporting language if the dominant hemisphere is damaged early in development (Ogden 1988, 1989).

Now, this is not to deny the existence of pure thoughts ('$2 + 2 = 4$') that can be essentially motivation-free. But these are relatively rare and their proper manipulation requires considerable training. Indeed, as academics, we know all too well that formal education takes many years, often unsuccessfully, to attempt to develop such motivation-free thoughts. In both the uneducated and highly educated, thoughts are typically embedded in a motivational framework. In any case, the cognitions involved in emotion *are* embedded in a motivational framework. This said, even, rats can have 'pure thoughts' (see Fallacy 8).

Fallacy 5: cognitions are unconstrained

We have so far dealt with what cognitions are not. We have not yet dealt with what we mean by 'cognition'. Many cognitive scientists believe that cognitions are knowledge-level mental representations that are not constrained (apart from in an obviously trivial sense) by the brain. When applied to emotion and personality, these scientists believe that personal meaning, values and other cognitions are the only important constructs as they are hardware-independent (see below). Most forms of cognitive therapy are predicated on the belief that disordered emotion results from 'irrational' thinking (irrationality implying less a lack of logic than the presence of self-defeating cognitions).

In its most general form, a true lack of constraint would imply that an intact brain can have an infinite number of different cognitions. But there are obvious storage and processing limitations: we cannot possibly remember the correct sequence of the 3 billion base pairs that comprise the human genome.

But what of flexible cognitive content (the human equivalent of the data stored in a digital computer)? What about qualitative rather than quantitative constraints? A few moments thought should show that cognitions must be constrained within a particular person or species. In the same way that perception of colour is impossible for the colour blind, cognitions of certain sorts are impossible for those with other neural birth defects or with focal brain dysfunction. Focal brain dysfunction is particularly interesting as it shows gaps in cognition and cognitive performance that can be remarkably selective. For example, a person may be incapable of naming only one particular class of object (e.g., living as opposed to non-living). Equally, schizophrenia (involving loose connection of thought streams), delusions (involving distorted conceptions of reality), mania (involving distorted decision-making) and paranoia (involving distorted attribution of motives) all show that so called 'normal' thinking depends on neural and chemical systems that can suffer from quite selective dysfunctions.

Why, then, is it not obvious that there are gaps in our cognitive armoury? Well, first, it should be noted that the gaps are often obvious, but only when they are in someone else's armoury. Agraphia, aphasia and various agnosias or even everyday misattribution are striking when we see them in others. 'But of course' we, the general community, do not suffer from these by definition.

But, we should note that the comparison can also be made in the reverse direction. Autistic savants can carry out mental operations that are starkly incomprehensible to the rest of us. They thus define mental operations of

which a human brain could be capable but that seem available only at the expense of other faculties. Likewise, where differences can be determined in general between male and female functions and brain organization, there is evidence of trade-offs (Kimura 1992). Specific capacities brought to a high pitch appear to reduce or translocate other capacities within the brain and removal of the non-dominant half of the brain results in migration of dominant functions (Ogden 1988, 1989). Even in the intact adult brain, expansion of cortical representations of some entities are at the expense of the representations of adjacent ones (Nudo, Milliken, Jenkins and Merzenich 1996). Different people may have had their cognitions constrained in different ways but their very differences demonstrate that cognition is constrained.

It is also important to consider the constraints that are imposed on higher-order cognition by perception. A cognition, defined as the representation of some fact about the world within some symbology of which a mind is capable, is a deduction or inference. Any percept can be thought of as a cognition (although some may prefer to distinguish individual percepts from clusters of percepts or rules relating to them). Yet even the simplest percept is an inference from available evidence based on a coherent picture of the world. It melds bottom-up and top-down information. What we see is not the world. It is subject to illusions that result from the detailed physiology of your peripheral systems. It is also subject to illusions based on our expectations, including socially derived expectations. Thus, even 'veridical perception' is a distortion of the world that in turn will affect those cognitions that derive from that perception.

Fallacy 6: cognitions are hardware-free

There is a subtler variant of Fallacy 5 that deserves separate consideration. Some cognitive theorists hold the belief that cognitive processes cannot, in principle, be related to neural processes since they are different philosophical things (Matthews 2000; Matthews, Derryberry and Siegle 2000; Matthews 1997). Important in this regard is the hardware-software distinction which most cognitive psychologists would endorse as marking cognition apart from neural processes. As one prominent cognitive theorist in personality, Gerald Matthews, stated clearly:

> There is a remaining difference regarding the hardware-software distinction, which I (along with most cognitive psychologists) do not see as a straw man. Philosophically, everything is doubtless the same brain/mind stuff, but development of coherent and empirically testable models requires choices to be made about which of the different descriptive languages of hardware and software (and

intentionality) is appropriate for the research problem at hand. A conservative position is that these languages are not readily compatible with one another, and trying to describe software constructs in hardware terms is an arduous task (though connectionism may help). I have some sympathy with the more radical Pylyshyn-Newell position that there are some emergent software phenomena that cannot be reduced to neural constructs ... [There is also] the cognitive science argument that rule-bound computational systems ('software') are not readily reducible to physical processes ('hardware'). One wouldn't try to explain the operation of MS Word in terms of silicon, so is it really wise to apply hardware explanations to brain software? (Matthews, Personal Communication, 26 May 1999).

Note that this point of view does not require either limitless hardware, or even limitless possible encodings of information within the hardware. All it requires is that the flexibility of programming of the machine is sufficiently great that the output from it (while constrained to what may be a considerable extent) is much more determined by the software currently loaded than the hardware into which it has been loaded.

There is a risk in over-applying the silicon computer metaphor. Certainly, your mother tongue cannot be predicted from the structure of your brain. Nor is a neural analysis appropriate if we want to find out from you what you had for breakfast. In respect to the specific details of much current content, your mind is like the computer. But the computer is designed to be a truly general machine. The brain and mind, by contrast, co-evolved and both have been shaped by Darwinian processes – much of the 'programming' is therefore 'hardwired' and much more like a robot that has been designed to perform some specific set of tasks than a general-purpose computer. Genes operate on the developing brain (in interaction with the environment) to deliver mental characteristics that interact with the environment to generate behaviours that are then selected for. In some cases the selection of such genes has given rise to quite specific 'innate' reactions to equally specific classes of external stimuli. This has been particularly studied with a rat's fear of a cat or a duck's fear of a hawk. But the human species has similar innate selectivities. Separation anxiety, fear of the dark and fear of spiders are among a host of 'cognitively' unlikely emotional reactions. They attach selectively to particular objects (spiders even in countries where these are not venomous) and not others (guns). These selectivities are best explained by in-built neural tendencies rather than by current developmental history (although developmental triggers are often required for the behaviours to appear).

Returning to the computer analogy, there is one way that the brain is quite unlike a digital computer. You cannot suddenly reprogram the

visual cortex for auditory processing, but you could easily load MicroSoft PowerPoint© into memory locations that just a moment ago stored MicroSoft Word©. The software of the computer can run from any part of the memory segments of the hardware:[1] the brain and mind are very different in this important respect.[2] This difference is not trivial, because relatively hardwired emotion processes seem designed to respond to any adequate eliciting stimuli in a highly structured manner and the triggers for these emotion processes are specific classes of cognition – the identity of which must be similarly hardwired. The highest cognitive levels, found in the cortex, allow specific identification of arbitrary signals. But these are then assigned meanings that exercise control over lower brain stem and limbic structures. Cognitive-behavioural therapy works precisely because it operates on cognitions to alter the assignment of stimuli as inputs to the lower brain systems of emotion.

Emotion processes also drive cognitions. Neurotic people are hypervigilant to threat and assign more threatening meanings to motivationally ambiguous stimuli. These biases arise because of the specific neurochemistry of inputs to crucial structures involved in monitoring the environment. We can change these neurochemical processes and so the person's cognitions by appropriate drug treatment.

Fallacy 7: radical behaviourism

The aberration of radical behaviourism has led many behavioural scientists to throw out the baby with the bath water. This is because the radical behaviourist revolution in psychology has generated a common residual fallacy, peculiarly afflicting only scientists and not the general public. Many personality theorists feel that biopsychology, learning theory and other analyses of non-humans are solely concerned with stimulus-response relationships. As such, these disciplines are held to be irrelevant to analysis of the sophisticated products of human cognition.

Simple behavioural observations, quite devoid of the ghost of introspection, make clear that behaviour is controlled by cognition. The nature of a goal is held in the animal's mind: its behaviour is not simply

[1] Note that the software is not indifferent to the hardware. An IBM program will not run (without recompilation) on a MAC and will not run, or often fit, in an older IBM machine. A program cannot run on a hard disk, it needs a CPU.

[2] The brain has a certain degree of plasticity, and reprogramming is possible on a longer timescale. For example, in those blind from birth the spatial processing units normally driven by vision may acquire auditory input; and, in animals like the blind mole rat, evolutionary rewiring of other inputs to what was visual cortex is complete (Catania 2002). But this is much more like rebuilding the machine than reprogramming it.

controlled by some chain of stimulus-response connections (McNaughton 1989; Cahill, McGaugh and Weinberger 2001). Animals immediately produce completely novel response sequences to achieve their unchanged internal goal when their original response is blocked (Towe and Luschei 1981; Hinde 1966). Internal representations of the external world are not unique to the human species.

Cognition also plays a deeper role in the observed actions. Physiological evidence shows that the cognitive restructuring that is the primary 'response' to 'conditioning' is the generation of an appropriate novel mental structure and not simply the recurrence of an older mental structure. Conditioning can result in physiological reactions that are quite different from those elicited by the conditional or unconditional stimulus before conditioning (Brady 1975a, 1975b) but that are entirely appropriate to the *anticipation* of the arrival of the unconditional stimulus. Learning does not, then, always (or necessarily) involve stimulus substitution[3] (Pavlov 1927). The behavioural changes observed by learning theorists, consequently, are evidence for new cognitions.

Of course, given their relatively larger brain, we would expect human beings to experience more elaborate and richer cognitions than the rat. We also know that our visual world is richer, being in colour whereas the rat's is not. Conversely, the rat's olfactory world is much richer than ours. But this does not mean that rat vision (or rat cognition during panic, or rat olfaction) is fundamentally different from the human variety, except in the trivial sense that the rat's cognition will be different from the chimpanzee's – we use the word 'trivial' to emphasize the much more important cross-species continuities.

Fallacy 8: silent cognitions

A weaker form of the radical behaviourist fallacy derives from the fact that cognitions, per se, are behaviourally silent. We know that we have thoughts that do not lead to behaviour. While our only evidence for *another* person's cognitions is behaviour (including verbalization as behaviour), we infer silent cognitions in them from the combination of this behaviour and our own experience. But we resist making this inference with non-human animals. Indeed, strictly, we should not make it even with other people on the basis solely of our own experience. It is tempting, therefore, to see non-human animals as not having any mental

[3] Stimulus substitution: the acquisition by a previously neutral conditional stimulus of responses elicited by a motivationally significant unconditional stimulus.

processes that are behaviourally silent and hence as not having cognitions in the 'true human' form.

Simple behavioural observations, nonetheless, can show that the knowledge that ultimately guides the behaviour of a rat or pigeon can be behaviourally silent while it is being learned and become evident only later, as in demonstrations of latent learning (Kimble 1961; Mackintosh 1974) and 'sensory preconditioning' (Dickinson 1980). Sensory preconditioning[4] is something of a misnomer, with the term 'preconditioning' reflecting the radical behaviourist fallacy that conditioning (i.e., learning) can only have occurred when a response changes. 'Silent conditioning' would better describe the fact that a rat can learn that a tone follows a light as easily as the fact that a shock follows the light. All conditioning would clearly be the same if we did not require behavioural evidence for its occurrence. Likewise, the same behaviour results whether a particular cognition is elicited by a conditioned or by an innate stimulus.

Fallacy 9: the cortex is the seat of cognition

This is a fallacy linked to a particular view of neuroanatomy. The idea is that the neocortex is the primary engine of cognition, the seat of our self-awareness. Indeed there are those who have argued that fish, for example, can have no perception of pain simply because they lack a neocortex (Rose 2002). Animals with relatively less neocortex, it is argued, cannot have our kind of cognitions. But, if we look at the cytoarchitecture of the cortex we find that the most recently evolved, most 'neo', neocortical cells are those closest to the peripheral inputs and outputs (Pandya and Barnes 1987). Each new level of the system has evolved by being interposed between the pure sensory periphery and a core of ancient essentially noncortical material (Nauta and Feirtag 1986).

[4] Sensory preconditioning is demonstrated with three experimental phases, the latter two allowing inferences to be drawn about processes occurring in the first:

Phase 1: Stimulus A (a light) is paired with stimulus B (a tone) in a series of classical (Pavlovian) conditioning-like trials. Neither A nor B produces any observable response, before or after the conditioning-like trials.

Phase 2: Stimulus B (the tone) is next paired with a food in a series of conditioning trials. Initially the subject salivates when the food is presented, after a number of trials, they salivate when B is presented.

Phase 3: Stimulus A is now presented to the subject without any previous pairing of A with food. In experiments of this type it is usually found that the subject will salivate when A is presented.

A has never been paired with food. Before Phase 2 it did not produce salivation. In Phase 3 it produces salivation showing that during Phase 1 the animal learned the relation between A and B. But, because neither was motivationally significant at the time of learning, it did not demonstrate this change in its cognitions by any change in behaviour.

The highest order processing (perhaps self or consciousness) therefore occurs in the oldest centres with the most archaic cytoarchitecture. This suggests that such 'top-end' processing exists, in however primitive a form, in the earliest creatures that can be said to have a telencephalon. Indeed, treating the end product as integration between modalities and combining perception and action into a unitary whole, it must have existed in a limited form primordially with increases in brain capacity only increasing the number and resolution of the sensory filters and motor mechanisms attached to it.

Before we turn to the specifics of emotion and personality research, we must stress that we are not arguing here that all relevant cognitive processes in human beings can be readily found and understood in non-human animals. The rat with its relatively small brain cannot have values, beliefs or expectations of the same order as you or I. But the nature of the rat's processes can inform us about human processes. In some cases, it should do so more readily because of the relative simplicity of the fundamental cognitions of an animal reared and tested in an impoverished environment. In other cases it may do so more readily because basic processes are not interacting with additional more complex ones. Our argument is, then, that both biological and cognitive constructs derived from work on non-human animals are relevant to an analysis of human emotion and personality. The human processes include and will often be largely like the non-human. That does not mean they all have to be exactly like.

Nor, even if all processes could be shown to be the same in all species, would we want to say that there could be nothing left to explain in a qualitative way in human beings. But the relevance of this uniqueness pales into insignificance once the considerable cross-species similarities are appreciated, especially in basic emotional processes.

Biology *and* cognition

Science has not yet reached a point where it can be shown that cognitive and biological explanations of emotion and personality *must* always be intertwined. But it would seem to us sensible to entertain this possibility seriously. This section turns to a positive path of identifying the integral links between biology and cognition, showing how personality research, and much of psychology in general, requires what we might term a *biocognitive* perspective.

Even at this early stage of scientific development, a common language would help to unite cognitive and biological levels of explanation. This does not imply that we may not have purely cognitive or neural theories

within limited phenomenological domains. But it would seem wise to try to develop constructs that, in theoretical and operational terms, may be translated across the various levels of analysis. In the specific areas of emotion and personality RST goes a long way to achieving this aim.

Brain-mind relations

It is given in modern cognitive and behavioural neuroscience (although still contested in some areas of psychology) that if you change the activity of the brain, you change the mind. There are already neural prosthetics that deliver auditory input to the deaf, visual input to the blind and control of the world to the paralysed. Less exotically, drugs can produce global changes in perception, mood and cognition. Above all, genetics, development and physical trauma, such as closed head injury or brain-splitting operations, can produce changes in neural circuits sufficient to produce massive, and usually consistent, changes in specific mental capacity and predisposition.

More recently, imaging techniques have led to a wealth of studies that show that if you change the mind (via external perceptual input) you change the activity of the brain in consistent ways. This activity usually changes in those structures previously implicated by invasive brain manipulation in the control of the relevant mental processes. For example, visual input activates the striate cortex and lesions of the striate cortex produce functional blindness. Faces showing fear activate the amygdala (Morris, Frith, Perrett *et al.* 1996; Morris, Friston, Büchel *et al.* 1998; Breiter, Etcoff, Whalen *et al.* 1996) and amygdala lesions produce impaired recognition of fearful expressions (Broks, Young, Maratos *et al.* 1998; Adolphs, Tranel, Damasio and Damasio 1994, 1995). There is also a wealth of other data implicating the amygdala in the control of fear responses more generally (Aggleton 1993; Davis 1992; LeDoux 1994, 1998).

To treat the mind as a property of brain activity is not, however, to give physiological events causal primacy over psychological events. Certainly, any particular internal psychological change will be represented by a physiological change. For example, learning of a particular fact will be represented by altered strengths of particular synapses and blocking this alteration will prevent learning (LeDoux 1993, 1994; Gewirtz and Davis 1997; Lee and Kim 1998). But it is the *information* encoded in external events, not the physical nature of the neural changes, that is critical for the details of the underlying mental and physical changes. The same can be true even when there are traumatic physical changes in the brain. Post-traumatic Stress Disorder (PTSD)

results in major physical changes in the brain, including loss of neurones in areas like the hippocampus (Bremner, Randall, Scott *et al.* 1995; Nadel and Jacobs 1996). Yet the disorder is in many cases the result of an *interpretation* of a pattern of light energy (visual images) where the light cannot itself damage the brain physically. Thus, what is best thought of as an internal mental event (a perception of threat) can produce physical damage in the brain (of course, 'mental' events are no less neural events). Nor need psychologically induced changes in brain morphology be pathological. Rats exposed to complex and interesting environments develop larger cortices.

The linking of these different types of observation is nicely exemplified in analysis of spatial memory. A spatial mapping task activates the hippocampus (Maguire, Frackowiak and Frith 1997); and lesions of the hippocampus impair spatial learning (Morris, Garrud, Rawlins and O'Keefe 1982). Most interestingly, sufficient use of the cognitive facility of spatial mapping, as shown in London taxi drivers, seems to have the physiological effect of increasing the size of the hippocampus (Maguire, Gadian, Johnsrude *et al.* 2000).

Emotion and personality

Many aspects of cognition, emotion and personality can be understood by studying homologous behavioural and neural phenomena in non-human animals. This position is epitomized in recent higher level applications of the concept of the BIS (Gray and McNaughton 2000, ch 11):

> Inasmuch, then, as the septo-hippocampal system is involved in cognitive and memorial processing, a consequence of our theory is that pathological anxiety itself is likely to result, at least in some cases, from abnormal cognitive and mnemonic processing (McNaughton 1997). This brings the anxiety aspects of our theory quite close to more recent cognitive theories of generalised anxiety (Mathews 1993; Eysenck 1992). 'Cognitive dysfunction' suggests affective neutrality and a focusing of hippocampal processing on cortical information. But when emergency threatens, the messages received from older structures located in the brainstem take precedence and a fundamentally cognitive dysfunction can have, nonetheless, affective consequences ... It has recently been suggested that the critical pathology in this disorder lies in the functioning of cognitive, particularly working memory, *system* (Eysenck and Calvo 1992; Eysenck 1992) or the control of attentional resources (Mathews and MacLeod 1994). While the cognitive processes we invoke are different, our theory has much in common with these views, sharing in particular the idea that generalised anxiety is primarily a cognitive disorder.

Personality, in particular, can be viewed as largely independent of the specific cognition through which we assess its effect at any particular

point in time. Personality acts as a general filter on all evaluation. As noted by Gray and McNaughton (2000, pp. 366–367):

> Neuroticism reflects principally a *perceptual* bias (that is to say, a *cognitive* bias . . . [requiring] interpretation of what is perceived) towards the identification or magnification of threat of all kinds [both fear and anxiety] . . . Anxiolytic drug treatment, in contrast, would alter the increased negative biasing associated with conflict, and so reduce anxiety only . . . Neuroticism [should] amplify the operation of systems that detect threat generally, and thus the entirety of the networks that subserve defence . . . Cognitive behavioural therapy works in the reverse manner, by dampening the operation of these same systems.

Thus RST, despite being predominantly based on data obtained from rats, emphasizes that cognitions, including high-level cognitions, are fundamental to threat processing in general (Neuroticism) and conflict between cognitive goals specifically (Anxiety).

With respect to clinical conditions, there is no question that the cognitions of a specific human individual in a particular situation will be different from those of a non-human animal. But they will also be quite different from those of other human individuals. Indeed it is the extent of certain individual differences that allow us to identify certain thoughts as pathological. However, cognitive biases, personality factors and most obviously the effects of drugs are factors that operate on *broad classes* of stimuli rather than on specific individual stimuli. Understanding of the resultant behaviour (which we need as scientific evidence) depends much more on categorizing such a class in terms of its affective value (e.g., immediate threat) than its cognitive complexity (possibly relating to a disadvantaged childhood and unloving mother). Once it is accepted that non-human animals are also driven by cognition, they can give us a clearer view of the factors driving our own behaviour than will study restricted to human beings.

Conclusion

This chapter starts from the position that emotion and personality researchers *are* divided into cognitive and behavioural (neural) camps (Corr 2001; Matthews 1997; Matthews and Gilliland 1999; Matthews, Derryberry and Siegle 2000). This is likely to be the result, in both cases, of a tacit rejection of a joint cognitive-neural perspective – an either-or type of thinking that portrays these two perspectives as somehow mutually exclusive. We see them, by contrast, as necessarily complementary – each level of analysis filling gaps left by the other.

Amalgamation is also necessary between academic and clinical perspectives. We agree with Luu, Tucker and Derryberry (1998) that

'anxiety may be a necessary component (motivator) of normal planning and regulation of behaviour' (p. 577). In the case of clinical anxiety, the most pronounced feature is the excessive motivational bias towards threat. It is not the specific behaviours or affect which are distinguishable from non-pathological anxiety but their excessive occurrence. Here we come full circle, because it is with extreme clinical conditions that neural analysis and non-human homologies are most apparent. This is true in particular of impaired executive functions (e.g., planning). Clinically, these demonstrate the involvement of the frontal lobes in cognitive processes and, in doing so, also link cognition to emotion. It is difficult to think of a frontal lobe related cognitive impairment that does not also entail an emotional component. The evolution of the neocortex may then not have acted to divorce cognition from emotion but to elaborate on a relationship between perception and action as two sides of the same coin.

This chapter has tried to bring the two sides together – or, at least, to have drained some of the conceptual swamp-land barring the start of this journey. In particular, it has listed a number of fallacies that we believe continue to pervade psychology in general, but especially emotion and personality psychology, which is still re-emerging from the dark days of neglect. As noted by Corr (2004, p. 318):

> Personality has long been the Cinderella of psychology: its scientific potential thwarted by psychoanalysis, social constructivism and statistical indeterminism, and neglected by experimental (cognitive) psychology.

As the contributors to this volume have shown, this dismal state of affairs is fast passing. Taken together with the other chapters in this volume, this chapter suggests that RST provides a general framework for the integration of the biological and cognitive into a biocognitive perspective, but this will be achieved only by putting to rest a number of die-hard fallacies concerning the relevance of non-human animal data and concepts for human emotion and personality

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