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What is Reinforcement Sensitivity? Neuroscience Paradigms for Approach-avoidance Process Theories of Personality

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Abstract

Reinforcement sensitivity is a concept proposed by Gray (1973) to describe the biological antecedents of personality, and has become the common mechanism among a family of personality theories concerning approach and avoidance processes. These theories suggest that 2–3 biobehavioural systems mediate the effects of reward and punishment on emotion and motivation, and that individual differences in the functioning of these systems manifest as personality. Identifying paradigms for operationalising reinforcement sensitivity is therefore critical for testing and developing these theories, and evaluating their footprint in personality space. In this paper I suggest that, while traditional self-report paradigms in personality psychology may be less-than-ideal for this purpose, neuroscience paradigms may offer operations of reinforcement sensitivity at multiple levels of approach and avoidance processes. After brief reflection on the use of such methods in animal models—which first spawned the concept of reinforcement sensitivity—recent developments in four domains of neuroscience are reviewed. These are psychogenomics, psychopharmacology, neuroimaging and category-learning. By exploring these paradigms as potential operations of reinforcement sensitivity we may enrich our understanding of the putative biobehavioural bases of personality. Copyright © 2008 John Wiley & Sons, Ltd.

Key words: reinforcement sensitivity; approach; avoidance; emotion; motivation; psychogenomics; psychopharmacology; neuroimaging; category-learning; neuroscience

> *Only once you have directly measured human variation in reinforcement sensitivity can you then ask how it corresponds to any personality questionnaire.*
>
> Neil McNaughton (Personal communication, 5th December 2006)

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WHAT IS REINFORCEMENT SENSITIVITY?

In the last few decades there has been increasing convergence among biologically oriented psychologists in terms of the theories put forward to explain individual variation in personality. These theories suggest that personality partly reflects variation in the functioning of 2–3 biologically-based systems concerned with motivation and emotion processes (Carver, Sutton, & Scheier, 2000; Cloninger, 1987; Davidson, 1998; Depue, 2006; Elliot & Thrash, 2002; Fowles, 1987; Gray, 1973; Gray & McNaughton, 2000; Tellegen, 1985; Zuckerman, 1994). The two broad processes which are distinguished by all of these theories (as well as the basic emotion and motivation literature; see Bradley, 2000, for a review) are *approach* and *avoidance*. Some theories focus primarily on these processes in terms of motivation (e.g. behavioural activation/inhibition; Fowles, 1987), while others emphasise their relevance to emotion (e.g. positive/negative affectivity; Tellegen, 1985). Some theories further distinguish between multiple, distinct kinds of approach and avoidance processes (e.g. Gray & McNaughton, 2000), and some focus primarily on one process (e.g. Zuckerman, 1994). But all of these theories agree that approach and avoidance processes are engaged by reinforcing stimuli in the environment—rewards and punishments, threats and incentives—and that personality reflects inter-individual variation in sensitivity to these reinforcing stimuli. It is for this reason that the theory put forward by Gray (1973), which has perhaps had the most profound influence on this area, has been termed '*Reinforcement Sensitivity Theory*' (RST; see Corr, 2008, for a detailed volume reviewing this theory and its impact on personality psychology). The present review was influenced by RST in particular, however the focus is on reinforcement sensitivity more generally, the central mechanism of almost all approach-avoidance theories of personality.

Approach processes concern sensitivity to rewarding stimuli, and are mediated by a Behavioural Approach System (BAS; Gray, 1987; Pickering & Gray, 2001), which is also known as a Behavioural Facilitation System (Depue, 2006), a Behavioural Activation System (Cloninger, 1987; Fowles, 1987; Gray, 1987; Pickering & Smillie, 2008), or simply 'reward circuitry' (Knutson & Cooper, 2005). The neurobiology of the BAS is primarily located in the basal ganglia, with a central role played by mesolimbic dopamine (DA) projections from the ventral tegmental area (VTA) to the ventral striatum (a key component of which is the nucleus accumbens), and also mesocortical DA projections to prefrontal cortex (Bozarth, 1991; Depue & Collins, 1999; Knutson & Cooper, 2005; McCLure, York, & Montegue, 2004; Pickering & Gray, 1999). Phasic activity of DA neurons increases in response to unpredicted reward, decreases in response to unpredicted non-reward, and is sustained when rewards are fully predicted (Day, Roitman, Wightman, & Carelli, 2007; Pickering & Smillie, 2008; Schultz, 1998, 2007). This suggests that DA communicates reward prediction error, and that BAS activation is triggered by unpredicted reward and sustained by predicted reward. Tonic levels of DA may also be relevant more generally for enabling behavioural activation (Schultz, 1998, p. 20). BAS output is thought to increase positive affect and motivate behavioural approach of the stimulus. Inter-individual variation in the typical activity of the BAS—how sensitive an individual generally is to rewards—is thought to manifest as a major personality trait, *extraversion* (or in other trait models, *positive emotionality*; Tellegen, 1985) perhaps being the most agreed-upon candidate today.

Avoidance processes concern sensitivity to punishment and threat stimuli, and in RST are mediated by two biobehavioural emotion and motivation systems. The Fight-Flight-Freeze System (FFFS; Gray & McNaughton, 2000) is activated by all punishment

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stimuli, the emotional consequence of which is fear, and the motivational consequence of which is defensive avoidance (McNaughton & Corr, 2004). The observable attributes of defensive avoidance are constrained by ecological factors, such that highly proximal threat will elicit defensive attack (the 'fight' response), while more distant threat will elicit escape (the 'flight' response) or, if escape is impossible, freezing (Blanchard & Blanchard, 1990a, 1990b). Stimuli of mixed valence, leading to *conflict* between goals (such as when a threatening stimulus must be approached, leading to approach-avoidance conflict), activates a separate Behavioural Inhibition System (BIS; Gray, 1981; Gray & McNaughton, 2000).[1] The emotional output of the BIS is anxiousness, accompanied by a repertoire of risk assessment and caution referred to in motivational terms as defensive approach (McNaughton & Corr, 2004). The qualitatively distinct emotion and motivation processes mediated by the FFFS and BIS are thought to contribute more generally towards sensitivity to punishment (Corr, 2004, p. 319). In neurophysiological terms, the BIS consists of the septo-hippocampal system and amygdala, while the FFFS comprises a hierarchical system spanning the periaquedital grey (intense/proximal threat), medial hypothalamus, amygdala and anterior cingulate cortex (distal threat). Threat stimuli are understood to trigger the release of serotonin (5-HT) and noradrenalin (NA), which project to all levels of the avoidance systems (Graeff, 1994; Gray & McNaughton, 2000, p. 111–119; McNaughton & Corr, 2004). General reactivity to such stimuli is thought to manifest over time and situations as trait *fearfulness* (FFFS-mediated) and *anxiety* (BIS-mediated). However, most approach-avoidance models of personality refer more generally to punishment sensitivity, and relate this to high-bandwidth personality dimensions such as *neuroticism* (or in other trait models, *negative emotionality*; Tellegen, 1985).

The putative role of reinforcement sensitivity in emotion, motivation and personality is depicted in Figure 1, while the biobehavioural architecture of reinforcement sensitivity, as described by RST in particular, is depicted in Figure 2. It is important to note that these

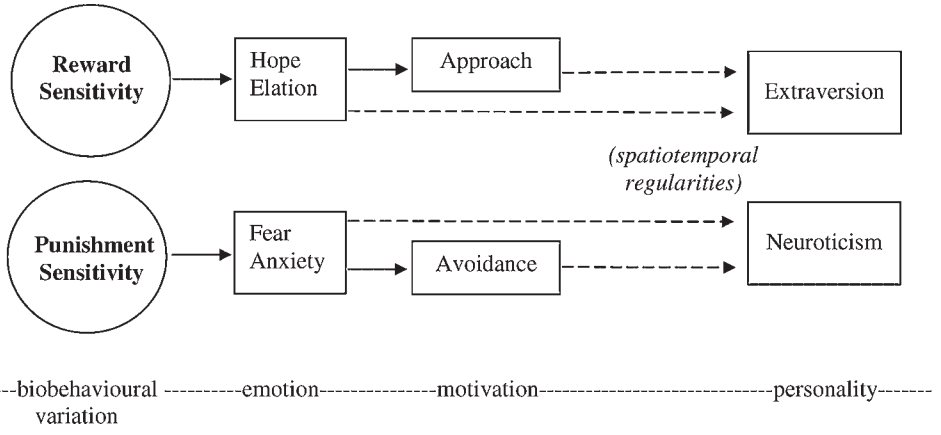

Figure 1. Conceptual illustration of approach-avoidance processes and their putative relationship with personality. Punishment sensitivity underlies negative emotional responses to punishment, which motivates avoidance of the threat, and manifests over time and situations as Neuroticism. Reward sensitivity underlies positive emotional responses to reward, which motivates approach of the incentive, and manifests over time and situations as Extraversion.

[1]Note that goal conflict may occur between *any* mutually incompatible goals, even two approach goals (e.g. competing rewards).

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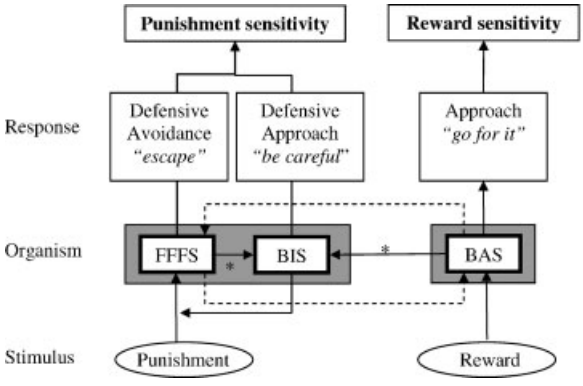

Figure 2. Biobehavioural architecture comprising reinforcement sensitivity as understood from the perspective of RST. BAS activation by reward inhibits the FFFS while FFFS activation by punishment inhibits the BAS. The BIS is activated if and only if the FFFS and BAS are jointly activated, signalling goal conflict (e.g. approach-avoidance). BIS activation biases BAS-FFFS competition in favour of the FFFS by weighting the inputs to the FFFS. Reward sensitivity is represented by inter-individual variation in BAS functioning, while punishment sensitivity is represented by inter-individual variation in both BIS and FFFS functioning. Although not a general feature of approach-avoidance models of personality, RST distinguishes between two avoidance processes, BIS-mediated defensive approach and FFFS-mediated defensive avoidance.

depictions are not unequivocally agreed-upon representations of RST in particular or approach-avoidance process theories in general. There is divided opinion on many issues, such as the specific traits and states which arise from approach and avoidance systems (e.g. Carver, 2004; Depue & Collins, 1999, p. 497), and the degree of their interdependence (e.g. Corr, 2002; Pickering, 1997; Smillie, Pickering, & Jackson, 2006). There are also many fundamental questions which have not been fully addressed, such as the role of reinforcement sensitivity in complicated, dynamic or long-term goals.[2] Similarly, the distinction between fear- and anxiety-related avoidance processes which emerged from a major revision of RST (see Gray & McNaughton, 2000) is shared by only a minority of related theories (see Davis & Young, 1998). Alternatively, while RST suggests a somewhat monolithic conception of approach motivation traits (e.g. extraversion), others argue for pluralism (e.g. agentic vs affiliative processes; see Depue, 2006). Perhaps the only aspect of these theories on which there is unanimous agreement is that personality traits reflect individual differences in reactivity to reinforcing stimuli. The importance of reinforcement sensitivity as a central explanatory mechanism in these theories can therefore not be overstated. Indeed, the potential for these theories only to explain personality is dependent upon the identification of paradigms to operationally define reinforcement sensitivity.

Presently, the dominant paradigm for assessing reinforcement sensitivity is psychometric; a large number of self-report questionnaires are routinely used to assess trait reward and punishment sensitivity and predict experimental criteria (see Caseras, Avila, & Torrubia, 2003, for a comparative investigation). For instance, Boksem, Tops, Wester, Meijman, and Lorist (2006) employed Carver and White's (1994) BIS/BAS scales 'to

[2]For example, Pickering and Corr (in press) note that attainment of long-term appetitive goals may require navigation through a landscape of sub-goals; the extent to which approach and avoidance systems may control this 'sub-goal scaffolding' is unknown.

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assess dispositional BIS and BAS sensitivities' [p. 98], against which an Event Related Potential (ERP) paradigm could be validated, such that, for instance, 'if the error-related ERP components are related to responsiveness of a dopaminergic reward system, we would expect a relationship between these components and individual differences in BAS scores' [p. 94]. Such wording appears to suggest that we can validate a potential neural signature of reward sensitivity by correlating it with a questionnaire. Similar suggestions can be widely found in the RST literature (alas, my own work is no exception!). It seems likely that a good case can be made for the usefulness of purpose-built reinforcement sensitivity questionnaires. Nevertheless, it is curious to observe so strong a reliance upon self-report measures in the area of personality psychology which has perhaps delved deepest into neuroscience paradigms and biologically driven perspectives.

There are a number of concerns one could raise about questionnaire operationalisation of reinforcement sensitivity. First, approach-avoidance theories such as RST are (mostly) bottom-up theories of personality, in that reinforcement sensitivity is postulated *a priori* as a biobehavioural antecedent of variation on trait measures. Framing trait measures as predictors of biobehavioural variables seems therefore to be proceeding in the opposite direction. In fact, it emulates the Eysenckian top-down strategy of beginning with 'gold standard' trait dimensions and searching for their biological correlates—the very strategy which was criticised by individuals such as Gray (1981). Second, although we might sidestep this first issue by viewing purpose-built reinforcement sensitivity questionnaires as proxies for underlying biobehavioural functions (instead of trait-like constructs), roughly 20 years of research has failed to produce decisive evidence that they in fact do this (Pickering, 2004). Third, the lack of convergence among these measures suggest that 'BAS questionnaires', for example, are not all measuring the same thing (Caseras et al., 2003; Quilty & Oakman, 2004), and various psychometric problems cast doubt on their basic measurement properties (e.g. Cogswell, Alloy, van Dulmen, & Fresco, 2006; Cooper, Smillie, & Jackson, 2008; Gomez, Cooper, & Gomez, 2005). More broadly, if such questionnaires are validated against a biobehavioural index in some studies, and then used to validate a biobehavioural index in other studies, we risk a circular and potentially vacuous understanding of reinforcement sensitivity. Finally, it seems biologically implausible to suggest that individuals can introspect directly about their reinforcement sensitivity—that is, consciously access the operational parameters of the BAS, BIS and FFFS—and report this on a personality questionnaire (Pickering, 2008; Smillie et al., 2006). Self-report questionnaires may partly describe functional outcomes of approach-avoidance systems (i.e. the various emotional and motivational *consequences* of approach and avoidance processes), but they cannot themselves be treated as proxies for the operational parameters of these systems.

While the psychometric tradition has dominated in approach-avoidance theories of personality along with most other areas of individual differences, basic neuroscientific research—much of it entirely unconcerned with personality—has yielded a number of paradigms which may facilitate a more concrete understanding of reinforcement sensitivity. These paradigms are no more 'biological' than questionnaires—even self reports must be at least partly caused by functioning of the brain (Corr, 2004, p. 322)—but they might be argued to more directly and objectively index those functions than self-reported introspections. Four main areas are discussed: psychogenomic methods may provide a window onto the genetic variations which influence brain structures and processes involved in reinforcement sensitivity. Psychopharmacologic methods might be used to manipulate reinforcement sensitivity experimentally through their effects on relevant neurochemical

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systems. Neuroimaging techniques provide potential temporal and spatial signatures of approach and avoidance system structure and function. And, of course, behavioural paradigms such as decision-making and category-learning may provide operationalisations of reinforcement sensitivity which complement the ethological focus of approach-avoidance theories. In drawing attention to such research, my objective is to encourage more direct, functional measurement of reinforcement sensitivity through the use of the valuable tools that neuroscience has made available for all of us. Before discussing each of these methods, I begin with an overview of how various techniques such as these have been used within animal paradigms to delineate the neuropsychology of reinforcement sensitivity, as described above and illustrated conceptually in Figure 2.

ANIMAL MODELS: REINFORCEMENT SENSITIVITY IN THE RAT

Personality differences described in humans appear to also exist in a range of non-human animals, from chimpanzees (Pederson, King, & Landau, 2005) to domestic dogs (Gosling, Kwan, & John, 2003). However, the role of animal models in personality theory remains a contentious subject (see Gosling & John, 1999, for a review). Some (e.g. Matthews, 2008) doubt the utility of a comparative approach, given the clear role of higher cognitive information-processing and semantic constructs in personality. Of course, there are many aspects of personality which animal models have never presumed to explain (Gray, 1973, p. 413, see also footnote 3), and potential advantages of cognitive models for some purposes say nothing about the usefulness of animal-based biological models for other purposes. Indeed, there are strong grounds for studying human personality through the lens of comparative psychology, particularly where individual differences in emotion and motivation are concerned. This is owing to wide agreement in the literature that such processes in humans are phylogentically old, and should therefore be at least partly functionally invariant across the different species to which humans are closely related (Gray, 1973; Gray & McNaughton, 2000; Ibanez, Avila, Ruiperez, Moro, & Ortet, 2007; LeDoux, 1998; Panksepp, 1998). If animal models can further our understanding of emotion and motivation per se, then they are also relevant to explanations of personality which are based upon emotion and motivation processes.

Gray (e.g. 1973) was probably the first to explicitly introduce animal models to personality theorists (although he credited those less famed for doing so at an earlier date; especially Teplov's, 1964 account of Pavlov). Strictly speaking, the animal (rat) models which influenced the concept of reinforcement sensitivity provided windows to emotion *states* rather than personality traits; Gray's RST is in many respects a state theory, where traits are inferred out of states which manifest themselves robustly over time and space. Consistent with the animal learning theorist approach, Gray (1973, p. 417–422) conceptualised emotions in terms of the (inferred) subjective condition of the animal during behavioural reactions to reinforcing stimuli. In other words, emotions were treated as organising concepts for regularities in behavioural responding to reinforcing stimuli. While the emotional consequence of reinforcement is inferred from behaviour, motivation describes the *purpose* of behaviour (e.g. to avoid a threat stimulus), and as such emotion can be viewed as the precursor to motivation (e.g. fear → avoidance). When combined with certain experimental techniques, this view of emotion and motivation offers very specific operational definitions in experiments. For instance, 'defensive approach' behaviour (chiefly, risk assessment) is elicited by incompatible goals or stimuli of mixed valence (e.g.

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food paired with intermittent shock). The inferred emotion in this case is anxiety and the motivation is to avoid the shock which could result from eating the food.[3] This 'anxious' behaviour is reduced or eradicated by administering anxiolytic drugs or making lesions to the septo-hippocampal system (Gray, 1970, 1976; Gray & McNaughton, 2000). We can therefore define anxiety, or 'conflict sensitivity', as the *process* that is reduced by such experimental manipulations (as noted by Fowles, 2006, p. 7).[4] Further, by studying the brain structures and circuitry affected by these manipulations—the contents of the BIS—we can determine the neuropsychology of that process. Shifting from states to traits then simply involves a shift in focus from a single observation of punishment sensitivity to the long-term patterning of this process. In doing so, we arrive at the primary hypothesis of approach-avoidance theories of personality; that there exist stable, biologically based individual differences in reinforcement sensitivity, and that this has a strong bearing on our personality.

If emotion and motivation processes correspond to brain structure and function then they must be under genetic control. In animal models, this has been investigated through selective breeding experiments. By outbreeding rats according to certain behavioural criteria, and carefully controlling for environmental influences (e.g. through cross-fostering techniques, see Gray, 1987, p. 43–46), one can safely attribute most behavioural/emotional/motivational differences in the descendent pups to genetic factors. One of the most important chapters in this research is the selective breeding experiments that gave rise to the 'Maudsley Strains' (Broadhurst, 1975; Gray, 1987, p. 43–51; Gray & McNaughton, 2000, p. 343–345). These rodents were outbred according to a defecation criterion (number of faecal boli produced in a threatening environment), used as an index of fearfulness (the emergent emotion of the FFFS; Figure 2). After 15 generations of genetic separation between Maudsley-reactives ('fearful' rats) and Maudsley-non-reactives ('fearless' rats), successive generations showed dramatic differences in defecation score. More compellingly, differences between these strains were also observed on a wide range of fear- and avoidance-related behavioural and physiological measures (Broadhurst, 1975). From this one can conclude that inter-individual variation in behavioural sensitivity to punishment is heritable.

Specific effects of pharmacologic and genetic manipulations on cohesive classes of behaviour, organised in terms of approach and avoidance systems, indicate that these systems correspond to physical structures and processes in the brain. Our knowledge of *which* structures and processes are involved, as described in the introduction to this review, has also been largely driven by animal models. One particularly exciting and relatively recent example of this concerns what is possibly the first animal model of individual differences in reward sensitivity, helping to confirm the neuropsychological contents of the BAS (Figure 2). Dalley et al. (2007) outbred Lister hooded rats on the basis of anticipatory

[3]Non-biologically oriented specialists in emotion and motivation will almost certainly perceive limitations of this approach to their domain. However it is important to remember that theories such as RST do not presume to explain complex emotions or desires, such as those which have been highly interpreted or cognitively enriched. In the hierarchical model of affect by Ortony, Norman, and Revelle (2005), it is suggested that the most basic level of affect, 'proto-affect', simply involves the assignment of value (positive or negative) to stimuli, and drives basic behavioural tendencies (motivations) to approach or avoid (p. 179–182). This model may be a useful way of describing the level at which theories concerning reinforcement sensitivity operate, in comparison with theories which work at higher levels of information processing.

[4]Note that this operational definition is not tautological: the naming of 'anti-anxiety' drugs is based upon subjective human reports, while our understanding of the behavioural repertoire which anxiolytics influence is based upon ethological studies of non-drugged animals (as noted by Gray & McNaughton, 2000, p. 85).

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responding to a visual cue for a food reward. The resulting strains were then shown to differ on a range of relevant behavioural and neuropsychological measures. Specifically, the reward sensitive strain again showed increased anticipatory responding to reward, and also, when trained to respond for intravenous injection of cocaine (a DA-activating psychostimulant), higher rates of responding. Pre-drug-exposure Positron Emission Tomography (PET) scanning also revealed lower D2/D3 receptor availability in the ventral striatum of the reward sensitive strain. Such research has helped to confirm the causal role that midbrain DA has in inter-individual variation in reward sensitivity.

If there were yardsticks against which all operations of reinforcement sensitivity should be evaluated (or, at least, with which they should be compared), then these surely must come from animal models. This is particularly the case for RST, which was entirely based upon animal research and then applied as a bottom-up model for human personality (Gray, 1973). Knowledge gained through animal research provides a starting point in the search for paradigms to operationally measure or elicit reinforcement sensitivity in humans.

PSYCHOGENOMIC MARKERS OF REINFORCEMENT SENSITIVITY

Psychogenomics is a broad term referring to the study of genetic factors involved in psychological processes (Corr, 2006, p. 365). Until relatively recently, such investigations were limited to behavioural genetics, which statistically partitions individual differences into genetic and environmental components. A significant chapter in this literature was *The Minnesota Study of Twins Reared Apart*, which demonstrated that 50% of the variance in personality could be attributed to genetic factors (Bouchard, Lykken, McGue, Segal, & Tellegen, 1990; see also Eaves, Eysenck, & Martin, 1989, whose estimates are basically identical). One might suggest from this that the 50% of heritable variance in personality questionnaire scores may principally reflect stable interindividual differences in the biobehavioural systems represented in Figure 1. However, behavioural genetics cannot identify the qualitative nature of genetic influences (e.g. genotypes which modulate catecholamine function) and therefore is unable to speak to this issue. That is not the case for molecular genetics, which analyses the structure and function of genetic variation at the molecular level. Resultantly, that is this methodology which has had the most impact in defining reinforcement sensitivity in genetic terms.

In the last decade or so of molecular genetic research, much has been learned about genetic variation within the 5-HT system in particular, and the influence this has on avoidance processes, particularly negative emotionality (see Hariri & Holmes, 2006, for a review). Of particular interest is the 5-HT transporter (5-HTT), which mediates the reuptake of 5-HT following release, and therefore influences the reactivity, in terms of strength and duration, of the 5-HT system. A relatively common variant of the 5-HTT polymorphic region, the 5-HTTLPR genotype, appears to partly determine the extent of 5-HT reuptake via the behaviour of 5-HTT; individuals carrying the long form of this allele have been found to have a twofold increase in reuptake relative to those with the short allele (Heils et al., 1995, 1996). Genomic imaging experiments have related the 5-HTTLPR genotype to functional variation in the specific brain regions which form the neural architecture of punishment sensitivity. For instance, using Functional Magnetic Resonance Imaging (fMRI), Hariri et al. (2002) observed substantially greater activity of the amygdala for short allele during perceptual processing of fearful and angry facial expressions. This genetic marker is also predictive of personality: Lesch et al. (1996) reported significant

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relations with the NEO-PI-R Neuroticism scale (Costa & McCrae, 1992), the 16PF Anxiety scale (Catell, 1946), and Harm Avoidance, from Cloninger's Tridimensional Personality Questionnaire (TPQ; Cloninger, 1986)—all of which have been identified as potential trait manifestations of punishment sensitivity.[5] This body of research represents the first confirmation of specific gene involvement in the brain structures and personality traits linked with avoidance processes and modelled in the 'Maudsley-reactive' strain.

It must be noted from this research that only around 20% of variance in amygdala activity and 8% of genetic variance in personality traits is explained by 5-HTTLPR. This should not cause surprise or dismay, as it is almost certain that any continuously distributed phenotypic variation results from multiple interacting genotypic effects (see Plomin, DeFries, McClearn, & McGuffin, 2001, pp. 28–40; indeed, 5-HT is only one of the two primary neurotransmitters linked with avoidance processes earlier in this paper). As such, 5-HTTLPR may influence punishment sensitivity traits in combination with other genetic variants such as those connected with NA function. In fact, such gene interactions have been recently discovered in relation to panic disorder, which RST suggests is a result of extreme reactivity of the FFFS, and may also develop from an anxiety or phobic disorder connected with reactivity of the BIS (Gray & McNaughton, 2000, p. 297–303). Freitag et al. (2006) demonstrated increased statistical likelihood of panic disorder as a consequence of interactions between 5-HT and NA polymorphisms which appear to impact upon the availability of the associated neurotransmitters. Despite many questions currently unanswered, this burgeoning area is already providing clues regarding specific genetic markers of punishment sensitivity.

In the case of approach processes, psychogenomic studies have focussed on genotypes which are associated with variation in DA function, such as genes which code for D2 and D4 receptors (which both inhibit DA release; Arnsten, 1998; Girault & Greengard, 2004). Cohen, Young, Baek, Kessler, and Ranganath (2005) compared individuals with and without the A1 allele of the Taq1A polymorphism of the DRD2 gene on a reinforced gambling task. Presence of A1 allele is associated with a 30–40% reduction in the density of D2 receptors, and therefore one would expect greater reward sensitivity in such individuals owing to lower inhibition of DA release. Indeed, relative activation magnitudes for rewarded versus non-rewarded trials were greater in several regions of interest (orbitofrontal cortex, nucleus accumbens, amygdala) for A1 carriers. Furthermore, activation magnitudes in the same brain region were significantly correlated with scores on the Big Five Personality Inventory measure of Extraversion (John, Donahue, & Kentle, 1991). Similar results have been reported by Canli (2006) in relation to the 7-repeat allele of the DRD4 gene, the presence of which was associated with both NEO-PI-R Extraversion and activation magnitudes contrasting positive versus neutral facial expressions. In a recent meta-analysis, however, while a single nucleotide polymorphism (SNP) of the DRD4 gene (C-521T) was found to be reliably associated with lower scores on traits such as TPQ Novelty-Seeking and Extraversion from the Eysenck Personality Questionnaire (EPQ; Eysenck & Eysenck, 1991), the 7-repeat allele was not (Munafò, Yalcin, Willis-Owen, & Flint, 2008). Furthermore, some research has found associations between Taq 1A and Neuroticism but not Extraversion (Wacker, Reuter, Hennig, & Stemmler, 2005). As such,

[5]Note that this does not validate these questionnaires as *measures* of punishment sensitivity. Rather, it supports the hypothesis that punishment sensitivity, operatonalised in terms of the 5-HTTLPR genotype, may manifest as or partly underlie traits such as Neuroticism, anxiety and harm avoidance.

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while this literature provides some early support for the role of DA-related genes in reward sensitivity, effects on personality have not always confirmed expectations.

As was noted for genetic correlates of punishment sensitivity, it is almost certain that the genetic antecedents of reward sensitivity are multiple and interactive. For instance, recent research has focussed on the interaction of DA-related genotypes with the catecho-l-o-methyltransferase (COMT) gene. COMT metabolises catecholamines and has a known role in the breakdown of DA. A SNP in the COMT gene resulting in a coding substitution of methionine rather than valine reduces this metabolic activity fourfold for those homozygous for methionine rather than valine (i.e. met/met versus val/val; Weinshilboum, Otterness, & Szumlanski, 1999). Consistent with the logical prediction from this, Yacubian et al. (2007) observed a monotonic increase of activation magnitude in the striatum (specifically, the ventral putamen) and prefrontal cortex during reward anticipation as a function of COMT genotype (met/met > val/met > val/val). Further, when predicting activation in the ventral striatum there was a significant interaction between variants of COMT and the DA transporter gene, DAT, which regulates DA reuptake in the same way that 5-HTT regulates 5-HT reuptake. Similar gene–gene interactions have also been associated with a trait measure of reward reactivity (Reuter, Schmitz, Corr, & Hennig, 2006). An important potential limitation of such research, however, is the small number of observations per cell which is often unavoidable in the study of gene–gene interactions.

As a research tool, molecular genetics has been described as the greatest achievement of science in the 20th century (Dawkins, 2003, p. 127); indeed it has answered more questions about genetic aspects of approach-avoidance personality processes in the last 5–10 years than has ever been possible before. It does not seem overly ambitious to suggest that this area of research may soon be able to identify gene combinations which are analogous to the rat strains engineered by selective breeding. However, our understanding of specific genes variants and their consequences is still juvenile, and it seems clear that the search for genetic markers of reinforcement sensitivity will be long and arduous. In order to choose a candidate gene for reinforcement sensitivity, such as DRD2 or 5-HTT, one must first have some idea of its function in general. This knowledge may come from basic research into approach and avoidance processes, but such investigations are perhaps unlikely to be driven by personality theorists. Possibilities may also be suggested from genome-wide association studies, increasingly used in medical research to explore correlations between genotypes and phenotypes, however high costs and potential unreliability (type 1 errors) may pose formidable difficulties (Shriner, Vaughan, Padilla, & Tiwari, 2007).

PSYCHOPHARMACOLOGIC MANIPULATION OF RST SYSTEM FUNCTION

While the psychogenomic approach may one day allow us to measure reinforcement sensitivity at the genetic level in humans, ethical concerns will obviously prohibit the genetic *manipulations* that are possible in animal models (e.g. selective breeding, genetic engineering). One of the most direct methods for manipulating reinforcement sensitivity is through the administration of pharmacologic agents which are known to influence the behaviour of specific neuroreceptors. This technique is in most important respects identical to the administration of anxiolytic drugs to block avoidance processes in experimental animals (Gray & McNaughton, 2000, p. 72–82). The interest for approach-avoidance

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theories of personality lies therefore in the potential for these drugs to manipulate reinforcement sensitivity in humans. Given stable individual differences reinforcement sensitivity prior to drug exposure, one should expect corresponding differential effects of these psychoparmacologic agents. Such differences provide potential indices of reinforcement sensitivity which can then be explored in terms of their relationship to major personality traits.

Two pharmacologic agents which have received much attention, particularly in the context of clinical neuroscience and addiction research, are the psychostimulant drugs cocaine and amphetamine (see Erinoff & Brown, 1994). Cocaine is known to block NA, 5-HT and DA reuptake, while amphetamine additionally stimulates monoamine release (Groppetti, Zambotti, Biazzi, & Mantegazza, 1973). Although not specific in their neurochemical actions, these substances have been argued to influence approach processes (e.g. Koob, Caine, Markou, Pulvirenti, & Weiss, 1994), as both have been shown to increase approach behaviour and reward learning in rodents (Taylor & Jentsch, 2001) and incentive processing in humans (Knutson, Bjork, Fong, Hommer, Mattay, & Weinberger, 2004). Furthermore, individual differences in response to cocaine and amphetamine administration have been found to predict personality. White, Lott, and de Wit (2006) found that amphetamine-induced increases in positive mood were associated with the MPQ scales of fearlessness and social potency. The substantial evidence linking DA function with MPQ Agentic Extraversion (Depue, 2006; Depue & Collins, 1999), of which social potency is a lower-order facet, suggests that a DA mechanism may underlie this finding. Complimentary results were obtained by Oswald et al. (2007) who found higher positive mood and lower right ventral striatal DA release following amphetamine administration in impulsive subjects (defined as those scoring above the median on a NEO-PI-R-derived measure of impulsivity On the other hand, while cocaine appears to robustly increase positive affect (Foltin, Ward, Haney, Hart, & Collins, 2003) amphetamine has been shown to increase positive *and* negative mood (Oswald et al., 2007), and effects concerning personality are occasionally opposite to those expected (Corr & Kumari, 2000). Furthermore, amphetamine has a range of effects on attention, vigilance, perceptual speed, and psychomotor function (Silber, Croft, Papafotiou, & Stough, 2006), suggesting that emotion systems are perhaps not directly or specifically targeted by this agent.

Interpretation of psychopharmacologic manipulations is potentially more straightforward when drugs with highly specific actions are employed. One group in Marburg, Germany, have recently reported intriguing findings in relation to the selective D2/D3 receptor antagonist sulpiride (see Chavanon, Wacker, Leue, & Stemmler, 2007; Wacker, Chavanon, & Stemmler, 2006). Sulpiride is known to leave D1 and D4 receptors unaffected, along with other neurotransmitter systems including 5-HT and NA (Perrault, Schoemaker, & Scatton, 1996). It blocks D2/D3 receptors primarily in limbic structures, where important BAS-related structures such as the nucleus accumbens are found. Wacker et al. (2006) and Chavanon et al. (2007) observed pronounced differences between extroverts and introverts when assessing performance on a working memory task (N-back task) and EEG measures of working memory (frontal vs parietal theta and alpha), both of which have a known DA basis. Further, under sulpiride these effects were completely reversed. Highly complementary results to these were reported by Cools, Sheridan, Jacobs, and D'Esposito (2007) using bromocriptine, a selective D2 agonist. Such findings clearly suggest that manipulations of the reward system have differential effects on behaviour, and that these differences are related to personality. An important caveat, however, is that cognitive rather than affective or motivational processes were examined in these studies. DA

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processes relating to working memory may be directly involved in reward sensitivity—for instance, through the maintaining and updating of approach goal representations (Miller & Cohen, 2001)—or these processes may be quite separable (as implied by research discussed later, e.g. Ashby & Ell, 2001). In either case, the more ideal experiment from the perspective of approach-avoidance process theories would examine how DA challenges modulate the effects of reinforcing stimuli on emotional or motivational criteria.

An example of such an experiment examined motivational reactions in abstinent male smokers (Reuter, Netter, Toll, & Hennig, 2002). At one-week intervals, following a 3.5 hour period of abstinence from smoking, participants received a DA agonist, a DA antagonist, and a placebo. The agonist was lisuride, which decreases the release of prolactin (a peptide hormone which inhibits DA release), and the antagonist was fluphenazine, which increases prolactin release. Participants were classified as 'responders' or 'non-responders' depending on whether or not (a) prolactin release decreased more after lisuride than placebo, and (b) prolactin release increased more after fluphenazine than placebo. Craving was assessed using a computerised task which presented various monetary trade-offs for a cigarette, to which the participant had to respond for each trade-off whether they would choose the money or the cigarette. 'Fluphenazine responders' sacrificed more money for a cigarette when given the DA agonist, while 'lisuride responders' sacrificed less money for a cigarette when given the DA antagonist. Furthermore, fluphenazine responders scored significantly higher on the EPQ Extraversion scale, while lisuride responders scored significantly higher on an 'Experience Seeking' subscale of trait Sensation Seeking (Zuckerman, Eysenck, & Eysenck, 1978). These findings demonstrate that individual differences in hormonal reactivity associated with DA function correspond to individual differences in motivational reactions to reward cues, and that these individual differences relate to personality. More generally, Reuter et al.'s strategy for classifying participants as 'responders' or 'non-responders' to a pharmacologic challenge seems an exemplary approach to operationalising reinforcement sensitivity.

In evaluating the contribution of psychopharmacology to the understanding of avoidance processes and sensitivity to punishment, one quickly surmises that 5-HT theories of impulsivity (see Arche & Santisteban, 2006; Evenden, 1999) have dominated this area. This makes it difficult to comment decisively on the ability for 5-HT agents to operationalise punishment sensitivity in humans in an analogous way to anxiolytic drugs in animals. On the other hand, some of these experiments may be of relevance to the RST view of BIS-mediated anxiety, specifically where impulsiveness is conceptualised as the inverse of anxiety (i.e. high anxiety constrains impulsive behaviour, low anxiety releases it; see Carver & Miller, 2006). Many experiments have operationalised impulsiveness in terms of response disinhibition (e.g. stop-signal task), which one might tentatively equate to (low) behavioural inhibition. However, some reviews suggest that the 5-HT view of impulsiveness has not been robustly supported when operationalised using such paradigms. For instance, Chamberlain and Sahakian (2007) concluded from a recent review that, while NA manipulations affect response-inhibition, SSRIs and central 5-HT depletion does not (see also Carver & Miller, 2006; Clarke, Roiser, Cools, Rubinsztein, Sahakian, & Robbins, 2005). Such data may provide mixed support for the neurochemical basis of punishment sensitivity posited in approach-avoidance theories, or it may not speak strongly to this issue at all. In either case it seems difficult to draw firm conclusions from this literature regarding psychopharmacologic operations of punishment sensitivity as it has been driven by somewhat separate theoretical foci.

Research in clinical neuroscience offers greater insight into psychopharmacologic operations of punishment sensitivity. Specifically, the use of selective serotonin reuptake

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inhibitors (SSRIs) and selective noradrenaline reuptake inhibitors (SNRIs) has been explored in considerable depth for the treatment of anxiety disorders (for recent reviews see Sheehan & Sheehan, 2007, and van Ingen Schenau & Wisman, 2007). The (state) anxiety-reducing influence of SSRIs and SNRIs in humans (along with other anxiolytics, such as those which influence GABA function) unequivocally confirms the animal literature concerning the effects of anxiolytics on behaviour (Gray & McNaughton, 2000, chapter 4). From this we might conclude that such drugs can be used in experiments to manipulate punishment sensitivity; indeed, punishment sensitivity is widely understood to lie at the heart of anxiety disorders, and to be principally affected by drug treatments (Blier & de Montigny, 1999). However, this is still an inferential leap which might require less effort if more stepping stones were more available, in the form of dedicated experiments focusing on affective and motivational effects of punishment.

To the author's knowledge, only one study has specifically examined the role of 5-HT in punishment sensitivity in humans in a manner which interfaces directly with approach-avoidance personality theories. Cools et al. (2005) examined the effect of central 5-HT depletion, via acute tryptophan depletion (ATD), on processing of fearful facial expressions. (Tryptophan is a precursor to 5-HT and is depleted through consumption of an amino acid supplement; Young, Smith, Pihl, & Ervin, 1985.) Following ATD, fMRI revealed enhanced activation in the amygdala and hippocampus in response to fearful and neutral faces, relative to happy faces. However, this effect only reached significance when a measure of anxiety (in the form of Carver & White's, 1994, BIS scale) was taken into account. This experiment compliments the psychogenomic investigation by Hariri et al. (2002) summarised above, in which greater amygdala activity was observed during processing of fearful and angry facial expressions for individuals who are genetically predisposed (in terms of the 5-HTTLPR genotype) toward greater 5-HT availability. Furthermore—similar to the literature summarised above in relation to DA, reward sensitivity and extraversion—it argues for a pharmacologic operation of punishment sensitivity that is associated with personality.

To summarise this section, psychopharmacologic manipulation of neurotransmitter systems implicated in approach and avoidance may have the potential to provide operations of reinforcement sensitivity. A particular attraction is the potential to experimentally vary reinforcement sensitivity, and to do so in a way which builds a direct paradigmatic bridge with the relevant animal literature. Also, the immediate foci of psychopharmacology paradigms are changes in emotional or motivational *states*, which are the building blocks of personality traits according to theories such as RST. Some clear challenges include the fact that many candidate substances do not have sufficiently specific neurochemical effects. Furthermore, even if we were able to selectively target the DA, NA and 5-HT systems individually, it is difficult to account for their subsequent interactions with one another and with other neurochemicals and brain processes (although this is a fairly general problem with experimental manipulation in the neurosciences).

NEUROIMAGING: DIRECT MEASUREMENT OF REINFORCEMENT SENSITIVITY?

Operations of reinforcement sensitivity using neuroimaging techniques may enable us to predict personality from relevant measures of brain structure and function. For instance, Barros-Loscertales et al. (2006) used structural MRI to assess grey matter volumes in the

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hippocampus and amygdala—the core of avoidance processes—and these were positively predictive of scores on a 'Sensitivity to Punishment' questionnaire (Torrubia, Avila, Motlo, & Caseras, 2001). While such findings are encouraging, one might argue that *dynamic* changes in the brain in response to rewarding or punishing events might provide better operationalisation of reinforcement sensitivity or *reactivity*. This requires functional neuroimaging techniques such as fMRI, which uses MRI to measure haemodynamic (blood oxygenation-level dependent; BOLD) responses to experimental manipulations. It is not uncommon in the broader neuroscience literature to assess the rewarding or punishing effects of stimuli in terms of activity elicited in the brain regions associated with approach and avoidance systems. For instance, Harbaugh, Mayr, and Bughart (2007) compared the rewarding effects of voluntary versus taxation-based charitable contributions in terms of BOLD response in the ventral striatum. The ideal extension of such research, from the perspective of personality, is to then determine in what way such indices relate to interindividual variation in scores on personality questionnaires. Such studies might be argued to decisively test the relevance of approach-avoidance brain system functions to personality. This section will summarise some recent advances in the identification of neural signatures of reinforcement sensitivity which may be useful in this respect.

There is now considerable evidence that anxiety and fear processes can be observed in terms of amygdala activity (e.g. Delgado, Olsson, & Phelps, 2006; Juranek, Filipek, Berenji, Modahl, Osann, & Spence, 2006; Morgan, 2006; Stein, Simmons, Feinstein, & Paulus, 2007). Furthermore, some of this research has linked such neural signatures of avoidance processes with personality traits (e.g. Reuter et al., 2004). An interesting recent example here is a study by Haas, Omura, Constable, and Canli (2007) using an emotional conflict task in which emotionally positive, neutral or negative faces are presented together with a congruent or incongruent word. Emotionally incongruent trials juxtaposed positive and negatively valenced stimuli (e.g. a sad face presented with the word 'party') and, as such, may constitute BIS-activating 'goal conflict' (as discussed by Gray & McNaughton, 2000). Indeed, fMRI revealed activation of the amygdala in response to emotional conflict trials, and activation magnitude was dependent on trait Neuroticism. Given Gray and McNaughton's view that conflict sensitivity is specifically related to trait anxiety, while Neuroticism perhaps reflects punishment sensitivity more broadly, it is particularly noteworthy that amygdala activity was significantly related only to the anxiety subscale of Neuroticism. This research demonstrates a relationship between another promising index of reinforcement sensitivity, comprising relatively direct measurement of the relevant brain areas, with personality.

In the case of the reward system or BAS, neuroimaging studies in humans have confirmed results of animal data concerning the role of the DA-innervated brain regions in reward processing (e.g. Knutson & Cooper, 2005; McClure et al., 2004). The knowledge that DA neurons communicate reward-prediction-error (RPE) has had a strong influence on the search for potential neural signatures of reward sensitivity. Specifically, paradigms have been devised which manipulate expectations regarding the probability of a reward, and examine neural activity in BAS-related regions of interest in response to unpredicted reward and non-reward. Cohen (2007) examined behavioural data and BOLD response to two decision options which differed in risk but not in expected utility. The high-risk choice offered a 40% chance of \$2.50 or 60% chance of \$0.00, while the low-risk choice offered an 80% chance of \$1.25 or 20% chance of \$0.00. A reinforcement learning model in which learning was driven by the size of the RPE (i.e. large non-predicted reward = large positive influence on learning) predicted trial by trial choice behaviour and neural response in the

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ventral striatum. Importantly, the best fitting model was one which permitted individual differences in the RPE-driven learning parameters. Cohen is firm in his conclusion from this model that individual differences *must* be incorporated in models of brain-behaviour processes concerning reactions to reward. One might further predict that these individual differences relate to approach motivation, positive emotion states, and, more distally, to personality traits such as extraversion.

The economic and logistic costs of fMRI make it difficult to study the large participant numbers which are often required for individual differences research. A less resource-intensive alternative is electrophysiological methods such as electroencephalogram recording (EEG) and the ERP technique. In comparison to fMRI, these provide more limited spatial resolution and source localisation, but far superior temporal resolution. Potts and colleagues (e.g. Martin & Potts, 2004; Potts, Martin, Burton, & Montague, 2006) have recently published a series of experiments which identify a potential ERP index of RPE occurring approximately 200–300 ms after delivery or non-delivery of a reward. In their associative learning paradigm, one cue is followed by a reward on 90% of trials (therefore reward almost always expected) while another cue is followed by reward on 10% of trials (therefore reward is almost never expected). Echoing the single-cell recordings in which DA neurons show enhanced firing after a non-predicted reward and a sharp depression after a non-predicted non-reward (see Schultz, 1998), Potts et al. (2006) observed a positive component (P2a) when a reward followed a cue associated with non-reward, and a negative component (close to N3) when a non-reward followed a cue associated with reward. These components were localised to the medial prefrontal-cortex, to which ascending DA pathways from the VTA are known to project. Although as yet unverified by evidence to corroborate the putative DA basis of this component (e.g. alteration by a selective DA agent or variation with DA-related genetic markers), this does seem a promising electrophysiological operationalisation of reward sensitivity (an attempt to link psychogenomic and behavioural data with the neural responses in this paradigm is forthcoming; Smillie, Cooper & Pickering, in preparation).

The ERP components studied by Potts are also predictive of personality in a manner consistent with approach-avoidance process theories. Martin and Potts (2004) compared components in the 200–300 ms time window for high and low impulsives, divided into groups on the basis of a median split performed on the Barratt Impulsiveness Scale (Patton, Stanford, & Barratt, 1995), which some research has linked with reward sensitivity and DA (Limosin et al., 2003). In the high-impulsive group, P2a to non-predicted non-reward was significantly lower compared with (in order of increasing magnitude) predicted non-reward, predicted reward, and non-predicted reward. Differences were non-significant in the low-impulsive group. A separate group studying very similar ERP components, however, found almost opposite effects using the Carver and White (1994) BAS scale. In this experiment, Boksem et al. (2006) calculated ERPs in the broad temporal area of the P2a, but only following errors on a flanker task. They observed a positive component following errors that was larger for 'high BAS' scorers than 'low BAS' scorers. A similar but non-significant trend was observed for measures of Extraversion and Novelty-Seeking. While these results do not fit comfortably with those of Martin and Potts, they are perhaps not directly comparable given that Boksem et al. did not focus specifically on *prediction* errors. Both studies however suggest that individual differences in neural activity following reinforcing events are predictive of personality.

Neuroimaging has the potential provide operations of reinforcement sensitivity which reflect activity in the brain regions that mediate approach and avoidance processes.

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Additionally, as was noted for psychopharmacology paradigms, functional imaging focuses on psychobiological states. This is of value because approach-avoidance process theories of personality are specifically concerned with individual differences in the emotion and motivational states elicited by reinforcing stimuli. One could further suggest that functional neuroimaging in particular has the unique potential to provide a 'Rosetta Stone' when translating between different levels of analysis. That is, it can help to associate or dissociate genes, drugs and/or behaviours. With advancing technology and retreating costs, the possibility of directly measuring reinforcement sensitivity in space (e.g. fMRI) and time (e.g. ERPs) seems likely to revolutionise personality research in the same way that imaging techniques have already revolutionised neuropsychology more generally.

BEHAVIOURAL PARADIGMS: CATEGORY-LEARNING

As indicated earlier, theories of reinforcement sensitivity have been strongly driven by animal models, and in such experiments our dependent variables ultimately consist of behaviour. It is not surprising then that the majority of these theories are dominated by behavioural operationalisations of reinforcement sensitivity. A comprehensive overview of the various behavioural paradigms which have been used to operationalise reinforcement sensitivity is well beyond the scope of this review, and readily available elsewhere (e.g. Matthews & Gilliland, 1999, p. 602–615; Pickering, Corr, Powell, Kumari, Thornton, & Gray, 1997). Most paradigms examine individual differences in some task-related behaviour during either a basic reinforcement-learning/conditioning paradigm (e.g. Corr, Pickering, & Gray, 1997; Levey & Martin, 1981; Shiels, Hawk, Kopelowicz, & Gignac, 2007; Smillie, Dalgleish, & Jackson, 2007), or, perhaps more typically, *any* task with a strong, salient reinforcement contingency (e.g. financial gains or losses). An example of the latter is the *Card Arranging Reward Responsivity Objective Test* (CARROT; Powell, al-Adawi, Morgan, & Greenwood, 1996), in which cards bearing five digits have to be accurately sorted into three piles based upon the presence of a 1, 2 or 3 among the five digits. Subjects first perform the task with instructions to maximise speed and accuracy, then again with a financial incentive (10 pence for each 5 cards correctly sorted), and finally once more without the financial incentive. Reward sensitivity is then operationalised as the increase in sorting rate (number of cards per second) under rewarding conditions, relative to the average sorting rate in the first and third (non-rewarded) conditions. Scores on this index are increased by DA agonists (Bromocriptine; Powell et al., 1996) and indirect DA agents (e.g. nicotine and caffeine; al-Adawi & Powell, 1997; McFie & Powell, in preparation), and are predicted by genotypes relating to higher DA activity (Powell, 2007).

Even without such encouraging neuroscientific data, purpose-built behavioural operations of reinforcement sensitivity such as the CARROT have tremendously strong face validity. It seems perfectly logical to expect that the introduction of a reward contingency to *any* experimental task will 'activate' the BAS and thereby yield differential effects on task-related behavioural criteria (i.e. reaction times, learning, response-bias etc) according to individual variation in reward sensitivity. On the other hand, however, surely it is *far more likely* that behaviour will vary as a function of reward sensitivity if the behaviour in question is known to critically depend upon brain reward circuitry. In the case of the CARROT, although evidence suggests that the criterion this task provides has some relationship with DA function, the mechanism underlying card-sorting time is somewhat ambiguous. The financial incentive may affect goal commitment, attentional

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processing or motor efficiency, to suggest a few possibilities. Further, the basis of this process in DA function may be indirect or secondary to some other brain function. Such ambiguities may account for the often weak or inconsistent validity evidence for behavioural paradigms in RST (Matthews & Gilliland, 1999; Pickering et al., 1997; Pickering & Smillie, 2008). For this reason, my colleagues and I argue, the key behaviours in the paradigms we use to operationalise reinforcement sensitivity (e.g. card sorting in the case of the CARROT) should ideally be linked with the functioning of the relevant biological systems.

One broad class of behavioural paradigm which may be useful in this respect is category-learning. Categorisation refers to processes in which novel objects or stimuli are assigned to two or more categories or classes, and the neuropsychological influences on learning of these processes has been the subject of considerable research (as reviewed by Ashby & Maddox, 2005). It is possible to distinguish between various category-learning processes, many of which have been linked with different, specific brain processes. For example, Robbins (2006) reviews several converging bodies of neuroscientific evidence, chiefly involving psychopharmacological manipulations in animal learning studies, which point towards highly differentiated involvement of 5-HT, DA, and NA in category-learning processes. Specifically, DA appears to be involved in 'set formation', which refers to the learning from feedback of explicit categorisation rules (e.g. long lines are As, short lines are Bs). Conversely, 5-HT appears to drive 'reversal learning', in which an explicit rule is learned (e.g. long lines are As, short lines are Bs) and then the stimulus-category contingency is reversed (i.e. long lines are now Bs, short lines are now As). Finally, NA is implicated in 'set shifting' which is similar to reversal learning except that a new contingency is introduced rather than an old contingency reversed. For instance, an example of 'extradimensional shift' would be if the category-predicting stimulus dimension changed from line length to line colour. Tasks involving such learning processes may therefore point towards promising operationalisations of reward and punishment sensitivity, by virtue of their known basis in DA, NA and 5-HT function.

Increasingly specific understanding of DA involvement in category-learning has recently fed into some particularly exciting reward sensitivity operations. Evidence from neuroimaging, psychopathology, behavioural experiments and formal modelling (e.g. Ashby, Alfonso-Reese, Turken, & Waldron, 1998; Ashby & O'Brien, 2005) suggests that we can subdivide DA-based categorisation processes according to two forms of set formation or rule learning. Specifically, DA involvement in the learning of explicit, verbalisable categorisation rules (e.g. red circles are As, blue circles are Bs) appears to be mediated by frontal executive functions such as working memory. Conversely, when two or more stimulus dimensions (e.g. colour, shape, size) must be integrated in a pre-decisional, implicit and non-verbalisable manner (Ashby & Ell, 2001), DA involvement is characterised by procedural, reinforcement-driven learning, directly linked with brain regions such as the nucleus accumbens and ventral striatum (see Maddox, Ashby, & Bohil, 2003; Waldron & Ashby, 2001). On this basis, Pickering and others have proposed that category-learning tasks which require 'information integration' should be particularly effective for devising operations of reward sensitivity (see Pickering, 2004, p. 467–471 for some supportive findings).

Tharp and Pickering (2007) recently investigated relationships between category-learning and personality, and obtained results which are potentially relevant to this proposal. The stimuli to be categorised were straight lines varying in length, angle and horizontal position. It was possible to attain a maximum of 83% correct by applying a

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simple verbalisible rule to any one of the stimulus dimensions while ignoring the others (e.g. long lines, are As, short lines are Bs, ignore angle and horizontal position), or 100% correct by combining length and angle while ignoring position. Small rewards were given in the form of positive feedback for correct trials, and if a criterion level of performance (90% correct) was attained in the final block of trials, an entry into a lottery. The critical finding was striking differential prediction of performance in terms of an 'Extraversion' factor (a composite of EPQ Extraversion and related measures) and an Impulsivity/Psychoticism factor (a composite of EPQ Psychoticism and related measures). Specifically, a standard deviation increase in Extraversion was associated with being 2.3 times more likely to attain criterion, while a standard deviation increase in Impulsivity/Psychoticism was associated with being 2.5 times *less* likely to attain criterion. Given the salience of rewarding contingencies in this task, one is tempted to suggest that these findings support the view that BAS-function underlies Extraversion rather than Impulsivity/Psychoticism (as has been recently argued; Depue & Collins, 1999). According to the category-learning literature summarised above, however, the viability of such an interpretation may hinge on the involvement of information integration, which did not appear to be the case for this task. Specifically, Tharp and Pickering's formal modelling indicated that Extraverts tended not to use information integration per se, but rather an explicit conjunctive rule (e.g. long, steep lines are As, short, less steep lines are Bs), the neuropsychology of which is unfortunately not well understood. Clearly, more research is needed in this area.

Behavioural paradigms are well represented in the approach-avoidance personality literature, perhaps appropriately so given the (animal) ethological roots of such theories. However, identifying behavioural means of assessing the effects of biobehavioural reward and punishment systems may be less than straightforward. Although in agreement with common sense, plucking any behavioural task from one's test library and introducing rewards or punishments is probably not the most probing means for studying reinforcement sensitivity (Pickering, 2004; Pickering & Smillie, 2008). It may be possible to operationalise reinforcement sensitivity with greater confidence by looking to other literatures—most notably the behavioural, cognitive and clinical neurosciences—for paradigms which have been linked with specific, relevant brain functions. Furthermore, we might surely be more confident if these paradigms were used alongside the neuroscientific methodologies discussed in the previous sections.

REINFORCEMENT SENSITIVITY: TOP-DOWN OR BOTTOM-UP?

Throughout this paper I have attempted to identify paradigms for operationalising reinforcement sensitivity which have emerged from neuroscience research. Some of the studies I have reviewed concern causal influences on the biobehavioural systems depicted in Figure 2 (e.g. psychogenomics) while others focus on their functional consequences (e.g. category-learning). Is this combination of top-down and bottom-up routes to understanding reinforcement sensitivity consistent with the spirit of approach-avoidance theories of personality? In fact, many approach-avoidance theories appear to work both bottom-up and top-down; for instance, Zuckerman (1994) is concerned with the biological explanation of a defined trait, *sensation-seeking* (top-down), but also uses bio-behavioural theory to drive his defining of that trait (bottom-up). However, the theory which has arguably had the strongest influence on this area, and has certainly been the dominant focus of this review, is viewed as a strictly bottom-up model. Specifically, Gray (1973, 1981)

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argued that we should examine the influence of fundamental biobehavioural processes on emotion, motivation and behaviour, and that doing so may lead us to a different understanding of personality than a top-down approach provides. This argument was partly a critique of the use of methods such as factor analysis to determine the nature and number of personality traits (see also Block, 1995). Nevertheless, supposing we agree with Gray's bottom-up approach to understanding *personality*, this does not necessarily imply that our understanding of *reinforcement sensitivity* must be restricted to bottom-up explanation. Although the goal for RST is to extrapolate upward, from reinforcement sensitivity to personality variation (Figure 1), it may also be a considerable drop down to the nuts and bolts which collectively determine reinforcement sensitivity (Figure 2).

There is good reason to suppose that a top-down approach will often be required for identifying promising candidates of reinforcement sensitivity. For instance, while we know that reward processes are driven by DA function, we also know that DA drives multiple, divergent processes (e.g. Arnsten, 1998; Rammsayer, 2004; Robbins, 1997). As such it seems clumsy to equate any and all aspects of DA function to reward sensitivity, and indeed, by focusing on any and all aspects of DA one would simply be studying the DA system, *not* reward sensitivity! The same point could also be made for other neurotransmitters, along with the various other indices of reinforcement sensitivity, by whichever method they are investigated. For this reason, bottom-up approaches to reinforcement sensitivity (e.g. *'This gene influences 5HT function and therefore may provide a useful marker for punishment sensitivity'*) may also require top-down scaffolding (e.g. *'This gene modulates behavioural responses to fear stimuli and therefore may provide a useful marker for punishment sensitivity'*). This is nicely demonstrated by Robinson, Sandstrom, Denenberg, and Palmiter (2005), who devised a maze-learning paradigm to assess whether DA mediated 'liking, wanting, and/or learning about rewards' [p. 5]. In this series of experiments, behaviour of DA-deficient mice was compared both between-subjects (i.e. a control group of non-engineered mice) and within-subjects (i.e. after DA function was restored). Results suggested that DA does not mediate the liking of rewards (in terms of the mean number of rewards consumed each day) or learning from rewards (in terms of the number of correct maze arm entries per 10 days). Rather, DA appears to drive the motivational effect of rewards, in terms of the speed with which rewards are approached. The findings of this research are compelling, and possibly controversial, but the more general point of interest lies in the use of top-down concepts (liking, learning, wanting) as scaffolding for the study of basic biobehavioural processes (DA). Similarly, identifying suitable paradigms for operationalising reinforcement sensitivity, seems strongly dependent upon our conceptual understanding of approach and avoidance processes.

CONCLUSION

Reinforcement sensitivity is the central concept in a broad family of theories which suggest that personality is at least partly caused by the functioning of biobehavioural emotion/motivation systems concerned with approach and avoidance processes. Most of these theories were based upon, or can be otherwise directly liked with, Jeffrey Gray's RST. At the time when reinforcement sensitivity was proposed as a basis for personality, it was unthinkable to operationalise using the paradigms at our disposal today. As such, we have perhaps inherited a fuzzy and ambiguous understanding of reinforcement sensitivity.

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Specifically, despite most approach-avoidance personality theories' having embraced a range of neuroscience methods, reinforcement sensitivity itself is almost always operationalised using questionnaires. Some potential pathways to more concrete, neuroscientific measures and operations of this concept have been opened up in areas such as psychogenomics (especially the candidate gene approach), psychopharmacology, neuroimaging, and category-learning.

There are opportunities but also challenges associated with the paradigms reviewed in this paper. Specifically, psychogenomics has the potential to provide highly reliable, and unequivocally causal, markers of reinforcement sensitivity, however identification of the specific genes involved will be both costly and difficult. Psychopharmacological studies allow us to manipulate reinforcement sensitivity, and therefore conduct fully experimental investigations in this area, however the potential imprecision of these manipulations introduces uncertainty into this approach. Neuroimaging techniques may allow us to measure reinforcement sensitivity in space and time, although the associated economic and skill requirements may still act as an obstacle for many. Finally, we can be more selective in our choice of behavioural paradigms by favouring those whose underlying biological mechanisms are crucially involved in approach or avoidance motivation, such as category-learning tasks (however the biological processes recruited during behavioural paradigms are not always known). If we can overcome the challenges presented by these paradigms perhaps we can move closer toward 'a fully-fledged neuroscience of personality' (Corr, 2004, p. 319).

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