Dopamine, appetitive reinforcement, and the neuropsychology of human learning: An individual differences approach

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Personality and Neuropsychology

Personality research is usually perceived as lying towards the "softer" end of psychological inquiry, seemingly far from the "harder" neural mechanisms, processes, and neurological lesions which are the daily stuff of neuropsychology. As biologically-oriented personality psychologists, however, we have always tried to couch our proposals in brain-behavioural terms and have even offered suggestions as to the "neuropsychology" of personality traits such as anxiety and their related clinical conditions (e.g., Gray, 1982). Furthermore, several other personality theorists have also drawn from the rich materials of behavioural neuroscience (e.g., Cloninger, Svracik, & Przybeck, 1993; Depue & Collins, 1999; Zuckerman, 1991). In this chapter we continue to apply our neuropsychological approach to personality: specifically focusing on the effects of personality traits on learning tasks.

The usual reaction of our more traditional neuropsychology colleagues is to express a varying degree of scepticism about any attempt to explore the neuropsychology of personality. It is therefore perhaps worth reflecting at the start of this chapter on the possible reasons for such a reaction.

One possible concern is that of *severity*. The subjects who participate in a personality study are healthy after all and so it might be expected that the range of variation of brain functioning that they would display would be relatively small. There are many studies of the effects of personality on cognitive task performance, for example those focusing on trait anxiety (Eysenck, 1992). These studies show that significant personality-task correlations can be readily obtained. Furthermore, we believe that new neuroscience methods will enable researchers to illuminate the neurobiological bases of these differences between individuals.

Another concern, which is potentially more serious, is that of *specificity*. Neuropsychology traditionally attempts to detect relationships between specific brain functions and the specific brain structures that are damaged in the patients being studied. Personality traits, even when they have a strong biological basis, may reflect mechanisms that are distributed over a wide neural network of structures (e.g., Gray, 1982; Depue & Collins, 1999; Johnson et al, 1999). However, the idea that specific brain structures may act more or less independently, and that the corresponding psychological functions may be empirically isolated, is an oversimplification that does not reflect the richly interconnected nature of the brain. In keeping with this, there has been a detectable shift away from notions of strict localisation of function in contemporary neuropsychology. This trend has been encouraged by connectionist models, which have emphasised distributed processing (e.g., Rumelhart & McClelland, 1986), and by the often widely dispersed patterns of neural activation revealed by functional neuroimaging studies (see Posner & Raichle, 1994).

A final area of disquiet probably stems from distrust of the self-report questionnaires used to measure personality traits and of "correlational" research in general. Careful psychometric development ensures that, whatever entities are measured by personality questionnaires, they are measured reliably. No-one's research exemplifies the careful approach to the construction and validation of personality measurement instruments, across

several decades, more than that of Jan Strelau (e.g., De Pascalis, Zawadzki, & Strelau, 2000; Friedensberg & Strelau, 1982; Newman, Clark, Crawford, & Strelau, 1997; Strelau, Angleitner, Bantelmann, & Ruch, 1990; Strelau & Zawadzki, 1995). Personality researchers, who aspire to biological, reductionist explanations of the traits in question, should remember that their neuropsychological investigations of personality can be only as good as the instruments with which they measure their primary constructs. Researchers like us, who have contributed little to the development of specific personality instruments, therefore owe a considerable debt to the careful psychometric groundwork by people like Strelau and Eysenck.

As a result of this kind of careful instrument development, there is currently a certain "bullishness" amongst researchers about the status of personality traits and their measurement (e.g., Matthews & Deary, 1998). Moreover, finding correlations with cognitive task performance is not self-fulfilling or circular, as the questionnaires only very rarely contain items which directly ask the subject to rate their own cognitive abilities. Finally, it should be recalled that traditional neuropsychology, and the currently highly popular techniques of functional neuroimaging, are also correlational methods. In these cases the experimenter records either the cognitive deficits which correlate with the presence or absence of a particular lesion, or records the neural activations which correlate with the tasks undertaken in the scanner.

In view of the above points we would argue that there is little reason for neuropsychologists to be so sceptical about, or hostile to, the use of personality studies to understand human cognition. There are also some advantages to this approach. A major issue for neuropsychological research arises from the need to demonstrate specific deficits. Patients often show widespread deficits across tasks, particularly when one is studying groups with neurodegenerative (e.g., Alzheimer's or Parkinson's disease) or neuropsychiatric conditions (e.g., schizophrenia). The research has to disentangle a proposed specific deficit on a particular task, which can be linked to specific brain pathology, from generalised problems on other tasks that cannot be linked, in a straightforward way, to particular brain structures. These generalised problems may arise from a host of other factors: psychoactive medication; hospitalisation and ensuing institutionalisation; diffuse cortical atrophy etc. etc. Most, if not all, of these generalised problems will be absent in the healthy subjects who would participate in a personality study, and so it may be more easily possible to see any specific deficit in such studies (Claridge, 1987; Patterson & Newman, 1993; Pickering & Gray, 1999).

Bearing in mind the above discussion, this chapter will begin a neuropsychological analysis of mechanisms through which individual differences in personality might influence some kinds of human learning. In addition we will suggest that these mechanisms may have a biological basis in factors affecting dopaminergic neurotransmission. We will therefore begin to enhance our analysis by constructing biologically-constrained neural network models of some of the processes concerned.

Possible Personality-Sensitive Processes Affecting Learning

To pre-empt what will follow we are going to suggest that certain forms of human learning, involving the formation of new associations between stimuli and responses, are ripe for study within an individual differences framework. Such stimulus-response (S-R), or habit, learning is currently the focus of considerable neuropsychological interest (e.g.,

Ashby, Alfonso-Reese, Turken, & Waldron, 1998) and we hope that an input from individual differences research in this area will provide further insights.

In particular we will consider two processes affecting S-R learning that may be reliably affected by personality trait variation. First, and in most detail, we will stress the (subject's reaction to the) reinforcement which follows a response (using ideas from reinforcement sensitivity theory; Gray, 1970; Gray, 1981; Pickering, Corr, Powell, Kumari, Thornton, & Gray, 1997). Second, we will stress (individual differences in) the processes by which a stimulus feature is deemed salient (Gray, Kumari, Lawrence, & Young, 1999; Schmajuk, Buhusi, & Gray, 1998). An ability to detect and attend to salient stimuli may be particularly relevant in S-R learning tasks in which the subject has to learn which stimulus features are predictive of the responses required, and learn to distinguish them from other stimulus features that are not predictive.

Reinforcement Sensitivity Theory: Neurophysiological and Theoretical Considerations

Reinforcement sensitivity theory (RST) is an account of fundamental human personality traits in terms of individual differences in the sensitivity/reactivity of basic brain-behavioural systems that respond to reinforcing (and other) stimuli. The details of the theory, and the empirical evidence that has accumulated from attempts to test it, have been described over the last three decades (Corr, 2001; Gray, 1970, 1981, 1982; Matthews & Gilliland, 1999; Pickering, Díaz, & Gray, 1995; Pickering et al, 1997; Pickering & Gray, 1999). We will reprise only those parts that are relevant for the current chapter.

One of the basic systems discussed in RST is known as the behavioural activation system (BAS). It is suggested that the BAS responds to specific inputs; in particular, stimuli associated with reward. When a person's BAS is activated by these specific inputs there are *motivational* consequences: the arousal of the person increases, energising any ongoing approach behaviour, and this might commonly be seen when the person is involved in approach behaviour towards the BAS-activating stimulus itself. For the purposes of this chapter we will emphasise the *reinforcing* consequences of BAS activation below: that is, the consequences for the learning of a response when that response has the effect of eliciting a BAS-activating stimulus from the environment (i.e., when a response leads to BAS activation). The personality component of the theory simply proposes that the biological basis of a fundamental personality dimension corresponds to inter-individual variations in the sensitivity/reactivity of the BAS to those stimuli which activate it. For the moment, we shall refer to the personality dimension concerned as the "BAS-related" trait; later in the chapter we shall consider what this trait may actually be. It is suggested, therefore, that subjects who have high levels of the BAS-related personality trait have a highly responsive BAS, and they are predicted to experience stronger motivational and reinforcing consequences of BAS activation than subjects with lower levels of the BAS-related trait. We have suggested (e.g., Gray, 1987b; Pickering & Gray, 1999) that the BAS is located, in part, within brain regions that are richly innervated by ascending dopaminergic projections; thus, the biological basis of the BAS-related trait is proposed to be partly dopaminergic. Other personality theorists have proposed fundamental personality traits based on the functioning of an explicitly BAS-like system (e.g., Cloninger et al, 1993; Depue & Collins, 1999; Zuckerman, 1991), and they have each stressed that the biological basis of such traits is likely to involve variations in dopaminergic neurotransmission. An area of disagreement (see below) centres over precisely which of the major personality dimensions (and their associated measurement instruments) offers the best characterisation for the BAS-related personality trait.

Two aspects of the original BAS formulation are worthy of note because we are going to suggest that they may need revision. First, we will consider the proposal that BAS-activating stimuli are stimuli *associated* with reward, along with the associated notion that the BAS is not activated by primary rewards themselves. It has been noted in the literature for some time that this aspect may have led to confusion (e.g., Zinbarg & Revelle, 1989, p. 304). In fact, cell recording studies in monkeys have shown that dopaminergic cells fire phasically in response both to primary rewards (e.g., an unsignalled drop of fruit juice placed into the animal's mouth) and to conditioned visual or auditory stimuli that have become valid predictors of reward. After learning has established the conditioned stimulus as a reward-predictor, the primary reward no longer elicits phasic firing in the dopaminergic cell (see Brown, Bullock, & Grossberg, 1999, and Schultz, Romo, Ljungberg, Mirenowicz, Hollerman, & Dickinson, 1995, for details and references).

In relation to these findings, it did not matter whether the dopaminergic cells being recorded from were located in the substantia nigra (such cells project predominantly to the dorsal striatum, i.e. the caudate or putamen), or were located in the ventral tegmental area (such cells project to a wide range of structures, but within the striatum they project predominantly to ventral targets, i.e. to the nucleus accumbens). These dopamine (DA) projection pathways are referred to as the *nigrostriatal* and *mesolimbic* DA systems respectively[1]. If firing of the ascending nigrostriatal or mesolimbic DA projection cells represents a key part of BAS functioning, as is widely supposed, then it seems likely that the BAS will respond both to primary rewards and stimuli associated with these rewards (although perhaps at different stages of learning).

A second issue to consider is the proposal that the BAS controls an individual's response to a stimulus associated with reward, but is not involved in the conditioning processes by which the stimulus became associated with the reward in the first place. We have counselled for some time that this distinction is a grey area within RST (Gray, 1987a) and have noted elsewhere that some data indicate an influence of potentially BAS-related personality traits on the conditioning process itself (see the discussion of the study by Corr, Pickering, & Gray, 1995, in Pickering et al, 1997). Zinbarg and Mohlman (1998) have responded to the uncertainties over this point by distinguishing between the standard ("motivational") version of RST and an "associative" version in which BAS-related personality traits are predicted to correlate with the strength of the association formed between a conditioned stimulus and a reward.

The recording data from DA cells, noted earlier, also speak to this second issue. However, some detailed background must be explored first in order to make the point. There are now quite a large number of similar computational models that attempt to simulate the DA cell recording data (e.g., Brown et al, 1999; Houk, Adams, & Barto, 1995; Salum, Roque Da Silva, & Pickering, 1999; Brown et al, 1999, also review a number of other models). A common feature of the models is the use of a so-called "three-factor" learning rule to describe the long-term modifications of synapses that underpin learning. The three-factor rule is so named because it proposes that three separate components are necessary for synaptic modification to occur. Let us consider this learning rule specifically in relation to the synapses which form the junction between cortical input terminals and the dendritic spines of the striatal neurons receiving those inputs. Synaptic strengthening is proposed to occur only when: (i) the *presynaptic* terminal from the cortical input is activated; (ii) the

[1] In this chapter we shall often refer generically to striatal neurons. This term is meant to cover neurons in both the dorsal striatum (e.g., in the caudate and putamen, often referred to as the neostriatum) and ventral striatum (e.g., in the nucleus accumbens).

postsynaptic striatal neuron is strongly depolarised (i.e. beyond the threshold for this cell to fire); and (iii) an appropriately-timed *reinforcement* signal has arrived at the synapse. It is widely suggested that the mesolimbic and nigrostriatal DA projections (i.e., those projecting to the striatum and other brain regions) carry the reinforcement signal.

Wickens and Kotter (1995), in an excellent review, summarise much of the relevant information: the evidence implicating mesolimbic/nigrostriatal DA neurons in reinforcement mechanisms; the theoretical development of the three-factor rule and its utility in explaining reinforcement processes; and the electrophysiological evidence concerning the role of DA in synaptic plasticity in the striatum. Finally, Wickens and Kotter propose a specific set of neurophysiological mechanisms by which the firing of mesolimbic/nigrostriatal DA cells may act as a reinforcement signal within the three-factor learning rule framework. The full details are beyond the scope of this chapter and so only a brief summary is given here.

A critical component is the binding of DA at a specific subtype of postsynaptic DA receptor (the D-1 subtype), located on the dendritic spines of striatal neurons. Binding at these receptors is driven by phasic bursts of firing in the ascending DA cells (and recall that just these phasic bursts of firing occur in response to rewards, or to conditioned stimuli associated with reward). These dopaminergic events (factor three of the three-factor rule) interact with processes occurring at other synapses located on the same dendritic spines of the striatal neurons (specifically the corticostriatal synapses at the junctions between terminals of cortical input neurons and the striatal dendritic spines). Firing of the cortical neurons (factor one) releases glutamate at these corticostriatal synapses and this binds at postsynaptic N-methyl-D-aspartic acid (NMDA) receptors on the dendritic spines. This NMDA receptor activation leads to depolarisation of the striatal cell (factor two) and an increase in calcium concentration within the dendritic spine. The interaction between these DA- and glutamate-activated processes is proposed to lead to a long-term strengthening (or "potentiation") of the corticostriatal synapses.

Finally, it is suggested that if the glutamate-activated processes occur (factors one and two) but are not followed, at the appropriate time, by DA cell firing (and the associated changes at striatal dendritic spines; factor three), then the corticostriatal synapses may undergo long-term weakening (or "depression"). These mechanisms of synaptic potentiation and depression will play a role in the computer simulations of learning presented later.

How does all the above discussion of the neurophysiology of synaptic modification in the striatum relate to RST, and in particular to the question of whether the BAS may play a role in the formation of reward-based associations? The various computational models of DA cell firing, noted above, suppose that the DA cells, in the substantia nigra pars compacta (SNc) and ventral tegmental area (VTA), are activated by primary rewards. Brown et al (1999) provide the most specific account (see Figure 1).

Brown et al suggest that primary reward signals arise initially in the lateral hypothalamus, and the resulting hypothalamic output activates both the ventral striatum and the pedunculopontine tegmental nucleus (PPTN). The PPTN is also indirectly activated by outputs from the ventral striatum: these outputs inhibit the ventral pallidum and thereby disinhibit the PPTN. The PPTN then projects to the SNc and there produces an increase in DA cell firing. However, the DA cells project to both dorsal and ventral parts of the striatum. Brown et al further propose that, in the ventral striatum, the resulting DA release acts as a reinforcement signal that allows the conditioning of sensory stimuli associated with the occurrence of reward. Specifically, they propose that cortical neurons, processing the sensory stimuli, synapse onto the dendritic spines of the ventral striatal neurons, and these corticostriatal synapses are strengthened (by the mechanisms suggested by Wickens & Kotter, 1995) when DA firing in the SNc is triggered by a primary reward. In this way the

conditioned stimulus, by itself, becomes able to activate the ventral striatal neuron and then, via the ventral striatum -> ventral pallidum -> PPTN -> SNc pathway, can elicit conditioned firing of the DA cells in the SNc.

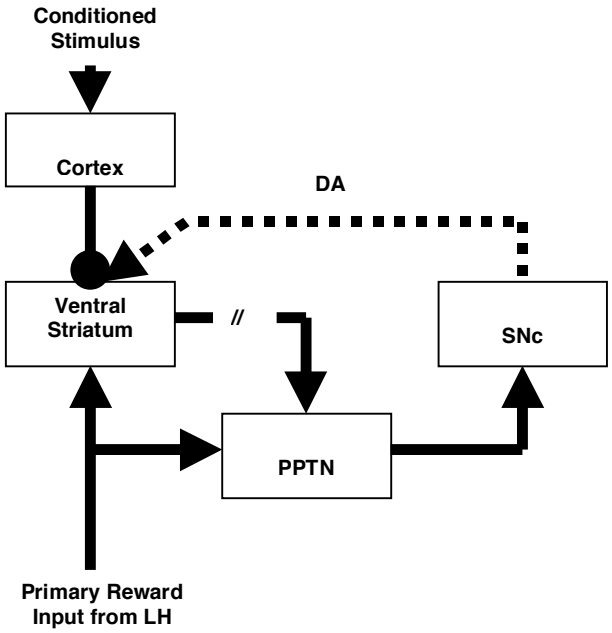

Figure 1:
A box diagram showing some of the neural components of the model by Brown et al (1999). The dotted arrow shows the dopaminergic projections to ventral striatum. All the pathways shown by solid continuous arrows are excitatory. The projection from ventral striatum to PPTN is depicted as non-continuous to reflect the fact that the pathway inhibits neural activity in the ventral pallidum thereby reducing the tonic pallidal inhibition of PPTN. The round arrowhead in the pathway from cortex to ventral striatum indicates a set of modifiable synapses. *Abbreviations*: DA dopamine; SNc substantia nigra pars compacta; LH lateral hypothalamus; PPTN pedunculopontine tegmental nucleus.

In summary, then, the DA cells which are activated by primary rewards are also involved in the strengthening of the neural pathways that enable these same cells to respond to conditioned reward stimuli. If the functioning of the BAS depends on the firing of these DA cells, as is widely supposed, then variations in the functioning of the BAS across individuals should influence the extent to which stimuli become associated with rewards.

A simple speculation as to how BAS functioning may vary across individuals would be to suggest that there is variation in the number and/or functioning of the D-1 DA receptors

on the dendritic spines of the striatal neurons. As already outlined, these receptors are widely believed to be responsible for the reinforcement function of the ascending DA projection systems. Therefore, high BAS reactivity might result from greater numbers, and/or increased DA-binding affinity, in these receptors. Genotypic variations in D-1 DA receptor structure have been identified (e.g., Cichon, Nothen, Erdmann, & Propping, 1994), and these polymorphisms have been found to be linked to potentially BAS-related behaviours such as smoking and gambling (Comings et al, 1997). The structural differences caused by D-1 DA receptor polymorphisms could alter receptor functioning, thereby potentially leading to the genetic variations in an individual's sensitivity to positive reinforcers.

Of course, there are several other components in the dopaminergic systems, underlying BAS function, which may show functional variation across individuals. For example, the DA cells themselves might be more reactive to their incoming inputs (from the PPTN, according to Brown et al's model), in high BAS trait, relative to low BAS trait, individuals. High BAS reactivity would then be manifest as a more intense DA cell firing in response to primary or secondary positive reinforcers. This increased firing could potentially boost positive reinforcement effects, through elevating the level of DA binding to the postsynaptic D-1 DA receptors. In terms of reinforcing consequences the end result could therefore be the same as that which might be produced by increased numbers, or increased binding potential, of the D-1 DA receptors themselves.

Predictions from RST about the Reinforcing Effects of BAS Activation

The previous section argued that the BAS is activated both by stimuli associated with rewards, and by rewards themselves. These stimuli are referred to as secondary and primary positive (or appetitive) reinforcers, respectively. This explains why the general theory is referred to as "Reinforcement" Sensitivity Theory. By definition, positive reinforcers increase the probability of any response upon which they are contingent (Gray, 1975). If a hungry animal's response is followed by a food reward (or a tone previously paired with food), then it will be more likely to emit that response again under similar circumstances. If a young child chooses to share his or her toy with a friend, and this action is followed by an approving smile from a parent, then the child will be more likely to share his/her toys in future. The change in behaviour represents learning under positive reinforcement.

As we have seen, RST is concerned with individual differences in the reactivity/ sensitivity of the BAS to positive reinforcers. It follows, therefore, that the learning of highly BAS-reactive individuals should be more strongly affected by positive reinforcements given during learning than would the learning of individuals with a less reactive BAS. The natural expectation is therefore that, the more BAS-reactive an individual is, the more quickly and strongly they should learn associations under conditions of positive reinforcement. This is a basic prediction of RST: the higher an individual's level of the BAS-related personality trait, the better they should learn under positive reinforcement. In the literature, this prediction has been tested a number of times, with very modest success, as we shall see below. The computer modelling work presented later casts light on why evidence in support of this basic prediction of RST may have proved so elusive. Before considering the past studies, however, we must address the issue that we have so far sidestepped: what personality factor corresponds to the BAS-related personality trait?

Characterising the BAS-Related Personality Trait

The original presentation of RST (Gray, 1970) suggested that neurotic extraverts were more "susceptible to reward" than stable introverts. In terms of the terminology of this chapter, this means that the BAS-related personality trait was considered to be a combination of neuroticism and extraversion. Note that, from the outset, the theory proposed an additive combination of extraversion (E) and neuroticism (N), with a greater weighting for E than for N[2]. Our position on this question has evolved: we proposed that the BAS-related trait might also contain a contribution from Eysenck's Psychoticism (P) scale (Eysenck & Eysenck, 1975b), with the high end of the P factor relating to high BAS-reactivity (Gray, 1987b), and we have also suggested that the weighting of the contribution from N might be effectively zero (e.g., Díaz & Pickering, 1993). In terms of a single verbal label for the BAS-related trait, we originally proposed that *impulsivity* or *trait impulsiveness* might well capture the dimension running from stable introverts (at the low end) to neurotic extraverts (at the high end). However, psychometric measures of impulsiveness (a tendency to act quickly without deliberating) tend to show high correlations with other traits such as sensation seeking, and antisocial or nonconformist tendencies. All these elements are present, to some extent, within the items that comprise Eysenck's P scale. In light of this we suggested recently (Pickering & Gray, 1999) that *impulsive sensation seeking* might be a better label for the BAS-related trait. However, to reflect the antisocial characteristics we shall, in this chapter, adopt the term *impulsive antisocial sensation seeking* (ImpASS) as a working label for the BAS-related personality dimension. Of course, this characterisation may be wrong, and only some elements of the ImpASS cluster of traits may relate to BAS functioning. Our view is that this is a matter to be determined empirically, once behavioural correlates of BAS functioning, which are reliably associated with personality measures, can be established.

The position just outlined is broadly in line with that of some other theorists who have proposed a personality dimension based on the inter-individual functional variations of a BAS-like system (e.g., Cloninger et al, 1993; Zuckerman, 1991). However, other theorists, who propose a BAS-based personality trait, argue that extraversion is a better label for the dimension (e.g., Depue & Collins, 1999).

The issue is complicated because there is a wide range of self-report questionnaires that are used to measure these personality dimensions and their constituent facets, and the measures tend to be positively correlated (see Table 1 for the widely-used ImpASS and E scales that are mentioned in this chapter; Depue & Collins, 1999, give a much fuller list of E scales). Depue and Collins graphically depicted the relationship between E and ImpASS measures, as revealed by multivariate statistical techniques, and this revealed that E and ImpASS measures tend to lie at 45º to one another, although the angle may grow towards 90º for ImpASS measures with a more pronounced antisocial component.

Depue and Collins (1999) recently developed their case by arguing that the E dimension was directly related to markers of dopaminergic neurotransmission, as one would predict for a BAS-related trait. In addition they suggested that ImpASS measures can sometimes be found to be correlated with DA markers, but only because ImpASS measures can stand as proxies for the E dimension (with which, as already noted, they correlate). Depue and Collins concluded that associations between E and DA markers should be stronger, and reported more consistently in the literature, than the associations between ImpASS and DA

[2] Some authors appear to have been confused into believing that we were proposing a multiplicative combination. A multiplicative relationship is more in line with the related, but distinct, position adopted by Newman and his colleagues (see Pickering, Corr, & Gray, 1999, for a discussion).

markers, if E were reflecting variations in dopaminergic neurotransmission more directly than ImpASS. They claimed that the literature showed just such a pattern of inter-relationships.

Table 1:
Some examples of widely-used measures of Extraversion and Impulsive Antisocial Sensation Seeking (ImpASS) personality traits.

Measures of Extraversion		
Scale	***Originator(s)***	***Inventory***
Extraversion	Eysenck	Eysenck Personality Inventory/Questionnaire (EPI/EPQ; Eysenck & Eysenck, 1975a;b)
Extraversion	Costa and McCrae	Neuroticism Extraversion Openness Personality Inventory (NEO-PI; Costa & McCrae, 1992)
Positive Emotionality	Tellegen and Waller	Multidimensional Personality Questionnaire (MPQ; Tellegen, 1982; Tellegen & Waller, 1992)
Measures of ImpASS		
Scale	***Originator***	***Inventory***
Novelty Seeking	Cloninger	Tridimensional Personality Questionnaire (TPQ; Cloninger, 1989)
P Scale	Eysenck	EPQ (Eysenck & Eysenck, 1975b)
Impulsiveness and Venturesomeness	Eysenck	The I_7-Impulsiveness Scale (S.B.G.Eysenck, Pearson, Easting, & Allsop, 1985b)
Sensation Seeking Scale	Zuckerman	Sensation Seeking Scale (SSS; Zuckerman, 1979)
Behavioral Activation System Scale	Carver and White	Behavioral Inhibition System and Behavioral Activation System (BIS-BAS) Scales (Carver & White, 1994)

By contrast, we would argue that, taking the published data in the round, there is no clear indication that DA-E relationships are stronger, or more consistently obtained, than DA-ImpASS relationships. To emphasise this, we begin by noting some of the reported relationships between DA and ImpASS measures, before considering evidence for dopaminergic involvement in E.

Clinicians have anecdotally observed an association between Parkinson's disease and personality characteristics at the low pole of the ImpASS dimension (i.e., stoicism,

industriousness, and inflexibility), after the onset of symptoms. The anecdotal observations also extend, more interestingly, to reports of premorbid personality from the patient or spouse. Menza and colleagues (Menza, Forman, Goldstein, & Golbe, 1990; Menza, Golbe, Cody, & Forman, 1993) have documented low levels of Cloninger's Novelty Seeking scale (Cloninger, 1989) in Parkinson's disease patients, and have argued that damage to the mesolimbic dopaminergic system in Parkinson's disease is responsible for the patients' characteristic (low ImpASS) personality profile.

There have been a few neuroimaging studies which cast light on the biological basis of ImpASS personality traits. In our laboratory, a single photon emission tomography (SPET) study in a small group of healthy volunteers (N.S.Gray, Pickering, & Gray, 1994) used a specific radioligand for dopamine D2 receptors (^{123}I-IBZM). We correlated the left and right hemisphere measures of ligand binding in the striatum with scores on the EPQ. We found significant negative correlations between the P scale from the EPQ (EPQ-P; Eysenck & Eysenck, 1975b) and the D2 binding index in each hemisphere, but no significant associations with other personality measures from the EPQ, *including extraversion* (EPQ-E). The binding index, lower in subjects with high EPQ-P scores, probably indexes D2 receptor density. However, it is possible that there may have been compensatory up-regulation of receptor sensitivity in subjects with a reduced number of receptors, so dopaminergic activity at individual synapses in such subjects may be enhanced.

A research group in Sweden (Farde, Gustavsson, & Jönsson, 1997) has independently reported on the personality correlates of D2 receptor binding in a larger group of healthy volunteers. Receptor density (in right and left striatum combined) was measured using positron emission tomography (PET) with a different radioligand, and personality was assessed via the Karolinska Scales of Personality (KSP). Significant negative correlations were obtained for the dimensions of detachment and irritability, which index some aspects of the ImpASS personality dimension. (Note that it might equally well be argued that these scales relate inversely to aspects of E.) The correlations with other scales from the KSP were noted to be non-significant (after correction for multiple comparisons), but were not reported in detail. It would have been particularly interesting to know the size of the relationships between D2 receptor binding and the impulsiveness and monotony avoidance scales of the KSP, as these measure other aspects of ImpASS personality.

A further PET study in a small group of Parkinson's disease patients (Menza, Mark, Burn, & Brooks, 1995) looked at the relationship between Cloninger's Novelty Seeking and striatal uptake of [^{18}F]dopa. Uptake of the ligand in the left caudate, but not other measured regions, was significantly correlated with Novelty Seeking. These workers related these findings to their earlier studies (Menza *et al*, 1990; 1993), which had showed low levels of Novelty Seeking in Parkinson's disease patients.

In recent years, the first evidence has begun to emerge, in human personality research, of an association between scores on a specific personality index and structural variations in the subjects' DNA (e.g., Benjamin *et al*, 1996; Ebstein *et al*, 1996). This research is germane to the present chapter as the index concerned (Cloninger's Novelty Seeking) measures ImpASS personality traits, and the gene with which it was associated codes for a subtype of the dopamine receptor (the D-4 subtype).

The dopamine D-4 receptor (DRD4) marker consists of alleles with from 2 to 8 repeats of a 48 base-pair sequence in exon III of the gene on chromosome 11 that codes for the dopamine D-4 receptor. The number of repeats changes the length of the third cytoplasmic loop of the receptor. Various *in vitro* studies (e.g., Asghari *et al*, 1994) have shown that the shorter alleles (2 to 5 repeats) code for a receptor that is somewhat more efficient in binding dopamine than the larger alleles (6 to 8 repeats). For this reason, DRD4 genotypes have

usually been analysed by comparing individuals who have two short alleles (about two-thirds of genotypes) versus those with one or two long alleles (one-third of genotypes).

In the original studies, individuals with at least one long-repeat DRD4 allele showed significantly higher Novelty Seeking scores than individuals without a long-repeat allele. The other three Cloninger temperament scales (Reward Dependence, Persistence, Harm Avoidance; Cloninger et al, 1993) showed no significant differences between the two groups. These associations have subsequently been investigated in a large number of studies. Although these subsequent reports have produced mixed results (see Plomin & Caspi, 1999, for a partial review), the overall pattern seems to indicate a link between DA receptor structure and ImpASS personality traits, particularly those measured by the Novelty Seeking scale. It should be noted that Benjamin et al (1996) actually found a significant association between DRD4 allele type and scores on both E and Conscientiousness (measured using the NEO Personality Inventory; Costa & McCrae, 1992). The relationship between DRD4 alleles and Novelty Seeking was based on subjects' *predicted* Novelty Seeking scores, calculated using a regression equation with the NEO scores as predictors. Clearly, the association with E fits with Depue and Collins' position, but the association with Conscientiousness (which is inversely related to ImpASS, but unrelated to E) does not.

Psychological and physiological responses to dopaminergic drug challenges, as a function of personality scores, offer further insights. Netter, Hennig, and Roed (1996) tested male smokers in a placebo-controlled design after they had abstained from smoking for 2.5 hours. When the subjects had been given a dopamine agonist (lisuride), or dopamine anatgonist (fluphenazine), drug-induced effects on prolactin secretion were observed. These effects were not mediated by either of two ImpASS measures used (the Disinhibition and Experience Seeking [ES] subscales from Zuckerman's Sensation Seeking Scale). However, other effects were mediated by ImpASS traits. Relative to placebo treatment, cigarette craving increased under lisuride for high ES subjects but was reduced under the same drug for low ES subjects. General wellbeing was increased after fluphenazine, and decreased after lisuride, to a significantly greater extent for high ES subjects than for low ES subjects.

Corr and Kumari (2000) looked at the mood-inducing effects of amphetamine as a function of two measures of ImpASS (Cloninger's Novelty Seeking scale and Eysenck's P scale), along with Eysenck's Extraversion scale. In a double-blind, placebo-controlled design, there were no mediating effects of E or Novelty-Seeking on the mood changes (in energetic arousal, tense arousal and hedonic tone) which they measured. In the placebo-treated groups, energetic arousal and hedonic tone increased, while tense arousal decreased, with increasing P scores. These personality-mediated effects in the placebo-treated subjects (presumably due to anticipation of drug effects) were significantly weaker in the amphetamine-treated groups.

The evidence just presented makes a moderately strong case that ImpASS personality measures are associated with variations in dopaminergic neurotransmission and, in some cases (N.S.Gray et al, 1994; Corr & Kumari, 2000), measures of E did not show a similar association. Depue and Collins' evidence comes from their own studies: this work also investigates the personality variables which mediate the psychophysiological effects induced by dopaminergic drug challenges. Their results tend to demonstrate associations with measures of E rather than with measures of ImpASS.

In the first study, Depue, Luciana, Arbisi, Collins, & Leon (1994) studied a sample of 11 healthy women. They correlated personality scores (from the Multidimensional Personality Questionnaire; Tellegen & Waller, 1992) against two psychophysiological indices that respond to a dopamine agonist challenge (they used the drug bromocriptine). The psychophysiological indices (measures of prolactin secretion and spontaneous blink rate)

were positively, significantly, and strongly associated with Positive Emotionality (a measure of extraversion) but not with Constraint or Negative Emotionality. Their findings were replicated in a larger study of 80 healthy young male and female subjects, which used a randomised crossover, double-blind protocol (see Depue, 1995; 1996; Depue & Collins, 1999). The association between prolactin secretion and Positive Emotionality was again strong and positive, although the positive relationship with Eysenck's E scale (EPQ-E) was a non-significant trend, and was slightly weaker than the (non-significant negative) association observed for the P scale. In line with the findings of Netter et al (1996), other measures of ImpASS (e.g., those from Zuckerman's Sensation Seeking scale) generally did not correlate with prolactin secretion, although those with a positive affective component (e.g., Eysenck's Venturesomeness scale; S.B.G.Eysenck et al, 1985b) did show a modest but significant positive correlation.

In summarising this evidence, we reiterate our earlier point: it seems premature to conclude that measures of E are more strongly and/or more consistently associated with DA functioning than measures of ImpASS. The discrepancies between the findings of Depue's group, and the other findings reviewed above, may have arisen because Depue and colleagues have typically used a different measure of E (Positive Emotionality rather than EPQ-E). The most convincing associations with ImpASS may be those involving the EPQ-P (which just failed to reach significance in Depue's research, with the correlation being in the opposite direction to that for Positive Emotionality). These data raise the possibility that there may be more than one personality factor that is associated with variations in dopaminergic neurotransmission.

Past Studies of BAS-Mediated Personality Effects on Learning

We have previously described an idealised experiment for testing for RST's personality predictions relating to the motivational consequences of BAS activation, and argued that few published studies have come close to satisfying the conditions needed to test RST adequately (Pickering et al, 1997). In this section we will carry out a similar exercise for RST's predictions relating to the reinforcing consequences of BAS activation.

For an idealised reinforcement experiment several conditions must be met. First, the relevant personality traits must be measured. In light of the controversy regarding the true nature of the BAS-related personality trait (see above) the study should include measures of extraversion or impulsive antisocial sensation seeking (or both). Second, in order to explore personality associations with reinforcement effects, the dependent variable must index *learning* rather than general task performance or psychomotor speed (which is more likely to be affected by motivational processes). Third, although reinforcement effects are traditionally considered to apply to operant conditioning paradigms, this chapter argued earlier that (the associative version of) RST can also make predictions for the influence of the BAS-related personality trait on the formation of associations between conditioned stimuli and rewards (i.e., the BAS may affect classical conditioning as well). Therefore, studies need not be restricted to operant procedures.

Fourth, the prevailing reinforcement context must be exclusively (or predominantly) rewarding. The reason for this stems from the (mutually) inhibitory interactions between the BAS and another brain-behavioural system (the behavioural inhibition system, or BIS) proposed in RST. The BIS is thought to be activated by stimuli associated with punishment and therefore may be active in tasks where punishing reinforcers are delivered. The BIS and BAS can be thought of as competing for exclusive control over behaviour, and we have shown that interactions between these systems, when both are activated, can considerably

complicate the behavioural predictions made by RST (see Pickering, 1997). In essence, this means that, in learning experiments with both punishments and rewards (rather than rewards alone), we cannot be sure which system will predominate for any given individual. Therefore, to study the effects of the BAS one needs an experiment in which the only explicit reinforcers are rewarding. We should keep in mind these conditions when reviewing the main published studies below.

One major series of studies (carried out in India by combinations of B.S.Gupta, U.Gupta, & S.Gupta) used a verbal operant conditioning procedure (see S.Gupta, 1990, for a review). The study by S.Gupta (1990) is typical. Subjects had to generate sentences using one of five pronouns (I; We; He; They; or You) and a series of specific verbs typed onto cards. The first 20 cards provided a baseline for the rate of use of "I" and "We" pronouns. For the next 60 cards (the conditioning phase) the experimenter reinforced the use of the "I" and "We" pronouns but offered no reinforcement or feedback of any kind for the other pronouns. For subjects in the positive reinforcement condition the experimenter said "good" to their responses that included the target pronouns and, for subjects in the punishment condition, said "poor" to responses including the target pronouns. A post-test phase on a further 20 cards allowed an index of learning to be computed. The change in "I" and "We" usage, produced by the conditioning phase, was measured by subtracting the number of target responses produced in the baseline from the number produced in the post-test phase.

Three groups of subjects varying in impulsivity were tested in the positive reinforcement condition, and three matched groups were tested in the punishment condition. The subjects were drawn from a very large sample, and were selected because they had high, medium, or low scorers on the Hindi version of the Eysenck Personality Inventory. Six further groups of 20 subjects (high, medium, or low sociability crossed with the two reinforcement conditions) were selected on the basis of their sociability scores from the same personality inventory.

In the positive reinforcement condition, subjects with high impulsivity scores showed more learning (i.e., a bigger increase from baseline to post-test in use of the reinforced pronouns) than subjects with either medium or low impulsivity scores. The same pattern was found for sociability. However, the study also revealed significant decreases in the use of the reinforced pronouns (between baseline and post-test) for subjects in the punishment condition. This effect was greater for subjects in the low impulsivity group, than for subjects with either medium or high impulsivity scores. (Learning in the punishment condition did not vary as a function of sociability scores.) Thus, impulsivity and sociability (extraversion) measures did appear to mediate operant conditioning under positive verbal reinforcement, but impulsivity also mediated conditioning under punishing verbal reinforcement. The latter effect is not predicted by RST. Furthermore, when our group tried to replicate these effects with subjects in London, we were unsuccessful (Hernaiz-Sanders, 1991).

Another important series of studies used variants of a go/no-go discrimination learning paradigm. The typical design involved presenting subjects with items drawn from a small set of between 8 and 12 randomly selected two digit numbers, with half being randomly designated as targets (to which the subject should respond; i.e. go items) and the remainder serving as distractors (to which the subject should not respond; i.e. no-go items). Typically, separate analyses have been conducted using the numbers of omission errors for targets, and the numbers of false positives made to distractors. Most of these studies have been carried out using so-called "mixed incentive" conditions, in which the correct responses to targets are rewarded and false positive responses to distractors are punished. This approach has been adopted because mixed-incentive conditions are most pertinent to the theory of disinhibition advanced by Newman and his colleagues (see Patterson & Newman, 1993).

For the reasons noted earlier, mixed-incentive conditions are not ideal for testing predictions based on RST. One study (Hagopian & Ollendick, 1994) did test a reward-only condition in which correct responses to targets were rewarded with auditory feedback and a 10-point increase in the subject's score, displayed on the screen. False positives received no reinforcement and neither did correct rejections of distractors, or omission errors made to targets. Hagopian and Ollendick also tested subjects under a mixed-incentive condition, in which punishment of false positives (loss of points) was used in addition to the rewards for correct responses. (Hagopian and Ollendick distinguished these conditions in terms of what reinforcement was applied to the false positives, with the results that the reward-only condition was confusingly referred to as "non-reward", and the mixed-incentive condition was referred to as "punishment".)

Unfortunately, Hagopian and Ollendick did not test for the effects of possible BAS-related personality traits, but looked instead at the effects of differences in anxiety. RST specifically predicts that anxiety relates to the functioning of the BIS, rather than the BAS. As such, anxiety should not affect learning under the predominantly positive reinforcement conditions prevailing in Hagopian and Ollendick's non-reward condition. However, they reported that low anxiety subjects made significantly more responses overall, and made significantly more false positives, than high anxiety subjects. By finding a significant personality effect that is specifically predicted by RST *not* to occur, then this result appears to contribute to the lack of fit between data and theory (rather like S.Gupta's significant effects of impulsivity on learning under punishment reinforcement).

However, there are reasons to suppose that the high and low anxiety groups in Hagopian and Ollendick's study did *not* differ in learning. In these go/no-go discrimination tasks subjects' performance can be affected by contributions from their ability to discriminate between the targets and distractors (called "sensitivity") and the psychological criterion they adopt for making a response. The placement of the response criterion determines the subject's "bias" towards responding or not, and can be completely independent of the subject's sensitivity. A difference in learning between groups of subjects should be revealed by a change in sensitivity, and not by a change in criterion placement (response bias). Separate analyses of correct target responses and false alarms, which is the standard approach for the go/no-go task, cannot separate sensitivity from criterion placement changes.

We therefore applied standard decision theory methods to the group average data reported by Hagopian and Ollendick. For the reward-only condition we found that the low and high anxiety groups had almost identical mean sensivity indices (d-prime=0.84 for low anxiety; d-prime=0.86 for high anxiety). However, the mean criterion placement index differed substantially between the groups (C=-0.89 for low anxiety; C=-0.43 for high anxiety; C=0 indicates no response bias, and the more negative the C index the greater the bias towards responding).

It is interesting to note that RST might predict response bias effects, in experimental tasks, as part of the motivational (rather than the reinforcing) consequences of BAS activation. If the general experimental context were rewarding, and activated the BAS, then any ongoing approach behaviour during the experiment might be (indiscriminately) enhanced. It is not inconceivable, on tasks where the subject has to decide whether to respond or not, that a BAS-induced enhancement in approach behaviour tendencies could manifest itself as a more liberal criterion placement. The result would be an (indiscriminately) increased number of responses, which could occur independently of the subject's level of learning concerning when that approach behaviour should be most effectively applied. This still leaves the puzzle, from Hagopian and Ollendick's data, of why

low anxiety subjects should show a sizeable increase in response bias under predominantly rewarding conditions.

Two other go/no-go discrimination learning studies (Zinbarg & Revelle, 1989; Zinbarg & Mohlman, 1998) merit further attention. Zinbarg and Revelle carried out 4 similar go/no-go experiments on a total of 310 subjects. They manipulated a number of variables across these experiments (time of day; task difficulty; subjects' expectations about the probability that a go response would be needed). They used letters (or letter pairs) as targets and distractors in the discrimination task and, within each experiment, employed two types of target (called "go cues") and two types of distractor (no-go cues). Targets are stimuli for which a go response is correct; distractors are stimuli for which a no-go response is correct. For one target type (approach cues), responses were rewarded (by gaining points visually displayed on the screen); and for the other target type (active avoidance cues) responses avoided the punishment (loss of points) that occurred when no response was made to these cues. For one type of distractor (omission cues) no-go responses were rewarded (and this reward opportunity was therefore lost when a false positive response was made to these cues); and for the other distractor type (passive avoidance cues) false positives were punished, so that the punishment could be passively avoided by not responding to these distractors.

Zinbarg and Revelle argued that RST predicts that the go cues should activate the BAS (and the no-go cues should activate the BIS), although because both cues were always present within an experiment, the study suffers the usual uncertainty of mixed-incentive conditions (i.e., whether BAS or BIS will be dominant for a particular subject). Zinbarg and Revelle analysed performance for go and no-go cues separately, an approach that supposes that the system which is dominant will keep changing as different items are presented during the experiment. There must be some uncertainty surrounding whether the BAS and BIS can be turned on and off (as Zinbarg and Revelle implicitly suppose) by an intermixed series of BAS and BIS-activating stimuli of this kind, presented at a fairly rapid rate.

Zinbarg and Revelle extracted learning measures by looking at the linear trend for the number of responses made by a subject across each experiment (experiments were divided into four blocks for this purpose). In the statistical analyses of their data Zinbarg and Revelle included cue type (go vs. no-go) as a repeated-measures factor. The influence of this factor on the slope of the linear response trend reflects subjects' ability to discriminate go from no-go cues. However, at best this would therefore reflect a combination of two separate effects on learning: an effect of BAS activation (for the go cues) and of BIS activation (for no-go cues).

Zinbarg and Revelle used high and low scoring impulsivity groups to look at BAS-related personality effects. The subjects were divided into these groups according to their scores on an impulsivity measure derived from the Eysenck Personality Inventory (Eysenck & Eysenck, 1975a). The impulsivity groups were crossed with high and low scoring anxiety groups (allocated according to scores on the Trait Anxiety scale of Spielberger's State-Trait Anxiety Inventory; Spielberger, Gorsuch, & Lushene, 1970). The anxiety groups were included to look at BIS-related personality effects.

The findings obtained were consistent with our earlier comments that both BAS and BIS effects would be likely to combine to affect Zinbarg and Revelle's learning measures. Only one personality effect was consistent across their 4 experiments (significant in two experiments, with a similar trend in the other two): the anxiety by impulsivity by cue type (go vs. no-go) interaction. Specifically, among low impulsivity subjects, they found that the discrimination between go and no-go cues was learned better by high anxiety subjects, relative to low anxiety subjects, while the effect of anxiety was in the opposite direction for

high impulsivity subjects. An attempt to explain this interaction in terms of the dynamics of the competition between BAS and BIS could be attempted, but would be entirely *post hoc*.

Zinbarg and Mohlman (1998) reported two experiments based on the standard digit-based version of the go/no-go discrimination task. While this study shared some of the problems with other go/no-go studies (such as the fact that mixed-incentive conditions were used once again), some interesting new elements were added. In the first experiment, correct responses to targets were rewarded with 25 cents, and false positive responses to distractors were punished with the loss of 25 cents. No feedback was given when a no-go response was erroneously made to a target or was correctly made to a distractor.

Following the approach used by Zinbarg and Revelle (1989), Zinbarg and Mohlman looked at learning via the slopes of the linear trends of responses (to targets and distractors separately) across blocks of trials. In such an analysis, the targets are construed as reward cues, which activate the BAS selectively, and the distractors are construed as punishment cues, which activate the BIS selectively. We have already noted our concern about the assumption, implicit in this experimental logic, that the BIS and BAS can be turned on and off independently in response to rapidly intermixed presentations of stimuli of this kind.

In their first experiment, Zinbarg and Mohlman used the same personality measures of anxiety and impulsivity as were used in the earlier Zinbarg and Revelle experiments. There were no significant correlations between personality scores and the slope of the increasing linear trend of responses to reward cues (targets) across blocks.

There was, however, a significant interaction of anxiety by impulsivity on the slope of the decreasing trend of responses to punishment cues (distractors) across blocks. This anxiety by impulsivity interaction was also found by Zinbarg and Revelle (1989). However, for Zinbarg and Mohlman the interaction arose because high levels of impulsivity helped high anxious subjects to more rapidly decrease their number of responses to punishment cues, whereas for Zinbarg and Revelle high impulsivity impaired the ability of high anxious subjects to learn the discrimination between BAS-activating and BIS-activating cues.

The major innovation added by Zinbarg and Mohlman (for half the subjects in Experiment 1, and all the subjects in Experiment 2) was to get subjects to provide expectation ratings for each stimulus, at the start of the experiment and at the end of each block of training trials. The ratings were essentially confidence judgments about the expected outcome of making of a response to each stimulus (on a 9-point scale from 1, which meant subjects were "absolutely certain that [responding] will lead to losing money", through 5, meaning they were "completely uncertain", to 9, meaning that they were "absolutely certain that responding will lead to winning money"). Learning of these expectancies was assessed via the slopes of the linear trends over blocks, computed separately for reward (target) and punishment (distractor) cues.

In their first experiment, there were no significant personality correlations with reward expectancy learning (i.e., the slope of the increasing trend of expectancies to reward cues over blocks). However, there was a significant correlation between anxiety and punishment expectancy learning (i.e., the slope of the decreasing trend of expectancies to punishment cues over blocks). This correlation reflected the fact that more anxious subjects acquired negative expectancies (about losing money) for the punishment cues more quickly than less anxious subjects. This result is consistent with the similar finding reported by Corr et al (1995) using a different measure of anxiety.

In Zinbarg and Mohlman's second experiment all the subjects provided expectancy ratings. Half the subjects were tested under financial incentives as before, but the other half had potentially more ego-involving reinforcement: gaining estimated IQ points for correct response to targets and losing estimated IQ points for false positives. They added further

measures of anxiety and impulsive sensation seeking. The BAS Drive and BAS Reward Responsiveness scales were used for impulsive sensation seeking, and the BIS scale was used for anxiety (these scales were developed by Carver & White, 1994).

None of the predicted personality effects on the slopes of the linear trends of responses, to either reward or punishment cues, was significant in either the ego-reinforcement or monetary-reinforcement conditions. This included the anxiety by impulsivity interaction terms which had previously been significant (in Experiment 1, for punishment cues). However, both anxiety measures (Spielberger's Trait Anxiety and the BIS scale) were significantly positively correlated with the slope of the linear trend of responses to the reward cues in the monetary reinforcement condition. This correlation is not expected to be significant under RST.

In the ego-reinforcement condition, there were no significant personality correlations with reward expectancy learning. However, there was a significant correlation between the BIS scale anxiety measure and punishment expectancy learning. This correlation indicated that high anxious subjects acquired the negative expectancies more rapidly than low anxious subjects. (In the monetary reinforcement condition, the corresponding correlation was nonsignificant and in the opposite direction.)

In the monetary reinforcement condition, there was a significant positive correlation of the BAS Reward Responsiveness scale with reward expectancy learning (in the ego reinforcement condition, the corresponding correlation was actually -- nonsignificantly -- negative). In the monetary reinforcement condition, the only significant personality effect on punishment expectancy learning was with Spielberger's Trait Anxiety scale. However, this correlation reflected a faster acquisition of negative expectancies in lower anxiety subjects. This result was thus in the opposite direction both to the effect observed in the first experiment, and to that seen (for the BIS scale) in the ego-reinforcement condition of the second experiment.

In summary, Zinbarg and Mohlman's experiment found very little evidence for correlations between BAS-related personality traits (impulsivity, sensation seeking) and learning under rewarding reinforcement. They did find several significant associations with the BIS-related personality trait (anxiety), but these seemed to raise more questions than they answered. Why were the effects significant but sometimes in the wrong direction? Why were they sometimes significant for learning about reward cues (for which anxiety is predicted to have no effect)? Why were they not replicable across monetary vs. ego-involving modalities of reinforcement, or across similar experiments? These problems are not unique to this study; we have noted similar problems in much of the other literature relevant to RST (see Pickering et al, 1997).

Our own laboratory has attempted to make a contribution to the literature on BAS-related effects on learning with two studies (Corr et al, 1995; Pickering et al, 1995). Both these studies did use a reward-only reinforcement condition. Pickering et al (1995) used a maze-learning task, but failed to find any significant correlations between a number of potential BAS-related personality traits and rate of learning of the maze under reward. They did report significant personality effects (of Eysenck's Venturesomeness scale; S.B.G.Eysenck et al, 1985b) on the speed of the subjects' movements across the maze, but this seems likely to be a motivational effect of BAS activation. Corr et al's (1995) instrumental learning task also used response time as its main dependent variable, and so is also likely to be influenced by any motivational effects that were produced by BAS activation. However, there were no significant effects of potential BAS-related personality trait measures on asymptotic response speed in the reward condition (in fact, as discussed by Pickering et al, 1997, there were significant effects of trait anxiety).

The final study we will review is a complex experiment by Ball and Zuckerman (1990). In many ways this represents the best experiment looking at the influence of BAS-related personality traits on learning. They tested 140 undergraduate subjects who scored either in the upper or lower decile of the Sensation Seeking Scale (Zuckerman, 1979). They used a concept-learning task (which can be thought of as a series of simultaneous, rather than go/no-go, discrimination problems).

The visual stimuli were presented in a pair on each trial, with one stimulus on each trial being the target, to which the subject had to respond, and the other stimulus serving as a distractor. The position of the target stimulus varied from left to right across trials. The stimuli were compounds of elements drawn from eight binary dimensions (i.e., a stimulus contained either a letter T or an X; it had either one or two borders around the letter etc.). Two dimensions (letter shape and border shape) were critical dimensions (and particular values on those dimensions, counterbalanced across subjects, designated the target stimulus). On each trial, the target stimulus in the pair contained target values on both the critical dimensions (e.g., letter T and square border); the distractor stimulus contained the complementary values (e.g., letter X and circular border). Across the 16 stimulus-pairings used in the experiment, each value, from each of the other six non-critical dimensions, appeared eight times in a target stimulus and eight times in a distractor stimulus[3]. (The target and distractor stimuli in a pair were complementary to each other on all eight dimensions.)

Ball and Zuckerman used four different reinforcement conditions, formed by crossing two between-subjects factors: verbal vs. monetary reinforcement; and reward-only vs. punishment-only conditions. No feedback was given for errors during the reward-only condition, nor for correct responses in the punishment-only condition. (The authors also used subject gender as a factor in their analyses, but this need not concern us here.) Although the paper reported response latency data, the primary results for the present chapter concern the learning measures. Ball and Zuckerman report the number of trials that each subject took to reach a criterion of five consecutive correct responses. The 16 stimulus-pairs were presented once each in a training epoch. After each epoch was completed, training continued immediately with the next epoch, in which all stimulus-pairings were again used once. On reaching the criterion, there was a non-reversal shift (i.e., two previously non-critical dimensions became the critical dimension and the previously critical dimensions became non-critical). This shift was not signalled to the subjects, other than through the reinforcement they received, and subjects continued the task until they reached the same learning criterion after the shift.

When the trials to criterion data were analysed with a repeated-measures factor (phase of learning: pre- vs. post- shift), there were no interactions of any of the between-subjects factors with the phase of learning. We will therefore mostly concentrate on the results from the pre-shift phase. The main finding was that the high sensation seeking group of subjects learned the task significantly faster (i.e. reached the criterion in fewer trials) than the low sensation seeking group. A high extraversion group learned faster than a low extraversion group, and a group with high P scale scores learned faster than a group with low P scores (in the post-shift phase). These other groupings were based on scores from the EPQ (Eysenck & Eysenck, 1975b). However, the findings for the other personality scales are of dubious interest given that the subject groups were selected for extreme sensation seeking scores. This means that the groupings for other, correlated, personality scales (such as extraversion

[3] This was true for the "Random condition" of their experiment; half the subjects were tested in a so-called "Correlated condition" in which the values for one of the six non-critical dimensions had a 75% concordance with the target values from the critical dimensions. This factor did not affect the findings most relevant for the present chapter.

and the P scale) are going to be confounded with sensation seeking scores. (Ball and Zuckerman reported the results for the other personality scales after covarying out sensation seeking scores, as an attempt to reduce this problem. However, this attempted solution is statistically dubious: see Tabachnick & Fidell, 1996.)

The other key finding was that the effect of sensation seeking group on learning did not interact with reinforcement condition (and neither did the effect of extraversion or P group). Ball and Zuckerman, following RST, had predicted that the beneficial effects on learning of sensation seeking, and other BAS-related traits, would have been restricted to the reward-only reinforcement conditions. *These data raise the possibility that BAS-related personality traits may affect learning through mechanisms that are not connected with sensitivity to positive reinforcers.*

Ball and Zuckerman offered two possible explanations for why high sensation seekers might have learned this task faster than low sensation seekers, irrespective of reinforcement condition. First, they suggested that, relative to low sensation seekers, high sensation seekers might have adopted a beneficial risk-taking cognitive strategy during the early trial-and-error stages of the task. Second, Ball and Zuckerman argued that high sensation seekers might have superior selective attention abilities to low sensation seekers, and thus be better able to focus in on the relevant stimulus dimensions rather than the irrelevant ones. Ball and Zuckerman noted existing data (Martin, 1985) that were consistent with their attentional account. We will return to this issue below, where we shall expand on the attentional explanation.

To summarize this section, we would suggest that the literature reviewed gives pretty weak support for RST's predictions concerning BAS-related personality effects on learning. However, we also feel that the studies concerned have rarely tested the theory adequately, often failing to meet one or more of the four conditions we outlined earlier. Nonetheless, the study by Ball and Zuckerman seemed to contain all the necessary elements, and it produced significant associations between BAS-related personality measures (i.e., measures of ImpASS) and learning. Unfortunately, the correlations did not depend on the presence of rewarding reinforcement.

As Ball and Zuckerman suggest, we might conclude that ImpASS traits can affect learning through psychological mechanisms unrelated to a subject's responsivity to positive reinforcement. At present we take this possible conclusion seriously, because we have a series of unpublished findings which replicate Ball and Zuckerman's results with different ImpASS measures and slightly differing learning tasks. A key feature is that our learning tasks have been designed to minimise, and in one case to completely remove, any contribution from environmentally-provided positive reinforcement.

From a neuropsychological perspective, we might tentatively believe that ImpASS traits are associated with variations in dopaminergic neurotransmission, in accordance with the data reviewed earlier. If other psychological mechanisms, unrelated to reinforcement processes, are responsible for ImpASS-learning correlations, then it is conceivable that they will depend, in some way, upon dopaminergic neurotransmission.

The position just outlined still leaves us with a lack of evidence, from learning studies at least, in support of RST's BAS-related personality predictions. It is ironic that the evidence base for BAS-mediated personality effects is so weak, given the widespread belief in a BAS-like brain-behavioural system, along with an associated fundamental personality trait that is partly dopaminergic in nature (see the peer commentaries on the major recent review by Depue and Collins, 1999). Against this *zeitgeist*, one must consider the question of how much longer one should continue to test a theory which seems so unable to generate consistent support. On reflection, we think it would be premature to abandon the concept of

the BAS in personality research just yet. Perhaps the research on learning (and other) behavioural processes, which are predicted to correlate with BAS functioning, would benefit from having a more precise theoretical formulation to generate experimental predictions.

In light of the above, we decided to formalise the predictions of RST, concerning BAS-related reinforcement effects on learning, in the form of a biologically-constrained neural network model. This approach to the neuropsychology of reinforcement effects is possible, given our growing appreciation of the role of DA in reinforcement learning, and the many models of DA-related reinforcement which now exist (as discussed above). In the next section we present the results of such modelling and the surprising insights we have started to gain from it.

A Neural Network Model of BAS-Related Personality Effects on Learning

In order to construct a biologically-constrained network model in this section, we will need to draw upon some of the neural circuitry that we considered earlier (see Figure 1). We will present simulations of Ball and Zuckerman's concept formation experiment (the random condition with positive reinforcement), partly because it is the best published test of RST's predictions regarding personality and positive reinforcement effects on learning, but also because the task they used is essentially a category learning task. Category learning tasks involve mapping multiple novel stimuli into a smaller number of separate response classes (such as arbitrary category labels or, in the case of Ball and Zuckerman's study, left- or right-sided button presses). The neuropsychology of category learning tasks is currently being intensively researched and there is a growing understanding of the underlying neural circuitry. It is especially pleasing, from our current perspective, to observe that category learning tasks are believed to depend on the dopaminergically-innervated striatal circuits implicated in the functioning of the BAS.

In an excellent and wide-ranging review, Ashby et al. (1998) describe the importance of the structures shown in Figure 2 for category learning. For a visual task, a high-level visual representation of the stimulus would be computed in extrastriate visual areas (e.g., inferotemporal cortex) and this information would project to the tail of the caudate in the striatum. There is feedback from the striatum to the prefrontal cortex via a complex set of striato-pallido-thalamo-cortical loops, and the prefrontal cortex completes the circuit by projecting back to the (head of the) caudate and the thalamus. Ashby et al suggest that units in the prefrontal cortex are associated with one of the category responses required, and project cortico-cortically to the appropriate motor execution units in premotor and/or motor cortex. The nigrostriatal DA pathway is shown projecting from the substantia nigra pars compacta (SNc) to the striatum. The mesolimbic DA pathway is shown projecting from the ventral tegmental area (VTA) to the prefrontal cortex.

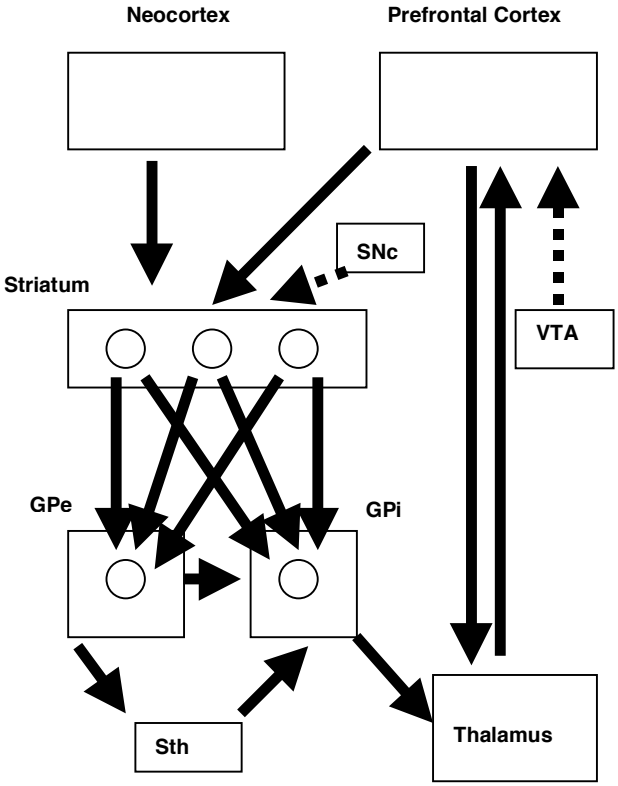

Figure 2
A box diagram of a cortico-striato-pallido-thalamo-cortical processing loop. Dotted arrows show the dopaminergic projections to striatum and prefrontal cortex. *Abbreviations*: SNc substantia nigra pars compacta; VTA ventral tegmental area; GPe external segment of the globus pallidus; GPi internal segment of the globus pallidus; Sth subthalamic nucleus.

Ashby et al (1998) review the evidence on category learning in various subject groups who have impairments to the above circuitry. Perhaps the most compelling data relate to patients with Parkinson's disease, a condition characterised by extensive dopaminergic cell loss in the nigrostriatal DA pathway. These patients been shown to be severely impaired at a probabilistic category learning task while showing good memory for the details of the computer screen layout and stimulus materials used (Knowlton, Mangels, & Squire, 1996).

In our model we simplified the circuitry of Figure 2 into an equivalent three-layer network that is depicted in Figure 3. There is a "stimulus pattern" (SP) layer (extrastriate visual cortex) providing the input to the "S-R representation" (SRR) layer (striatum); this in turn projects to, and receives projections back from, a "response decision" (RD) layer (prefrontal cortex). Note that the (corticostriatal) synapses between the SP and SRR layers

are modifiable (shown as round arrowheads), under the influence of a dopaminergic positive reinforcement signal (arising in SNc), using a 3-factor learning rule of the kind described earlier. The synapses between the SRR and RD layers are modifiable in a similar way, under the influence of a dopaminergic positive reinforcement signal (arising in VTA). We can think of these latter synapses as being thalamo-cortical, i.e. the last synapse in the pathway from striatum via globus pallidus and thalamus to the prefrontal cortex (although several of the synapses in that pathway might be modifiable). Figure 3 also depicts modifiable synapses in the reciprocal pathway between the RD and SRR layers (there are round arrowheads at each end of the connections between the SRR and RD layers). In the simulations presented in this chapter this reciprocal pathway is omitted for simplicity.

Finally, Figure 3 shows lateral inhibitory connections between units in both the SRR and RD layers (horizontal double-headed arrows). These mutual inhibitory interactions are used to select a single active neuron in each layer when input is passed to the network (so-called "winner-takes-all" competition). Mutual inhibitory connections like this are thought to exist in the striatum, because the striatal neurons have extensive axon collaterals contacting other striatal cells, and firing of the striatal neurons releases the inhibitory neurotransmitter γ–amino butyric acid (GABA) at synapses on their target neurons (see Groves, Garcia-Munoz, Linder, Manley, Martone, & Young, 1995, pp. 68-69).

The inputs to the network comprised the pairs of stimuli used on each trial of Ball and Zuckerman's task. Each pair of stimuli was coded as a vector of 32 binary neurons. The values of the vector elements can be thought of as representing the output firing (1=firing; 0=not firing) of the SP layer (i.e., visual cortical) neurons processing the stimulus-pairs. 16 neurons coded for the stimulus on the left on a particular trial, and 16 corresponding neurons coded for the stimulus on the right. There was one pair of neurons, in each set of 16, that coded for each of the 8 stimulus dimensions (e.g., neurons 1 and 2 coded for the letter shape of the left stimulus, while neurons 17 and 18 coded for the letter shape of the right stimulus). In each pair of neurons the pattern {01} was used to code for one value on the dimension (e.g., letter shape=T), and the complementary pattern {10} was used to code for the complementary value on the dimension (e.g., letter shape=X). Thus, if the left stimulus had a letter T in it, and the right stimulus had an X in it, then SP layer neurons 1, 2, 17 and 18 had the output values {01…..10……}. The same coding approach was used across all eight stimulus dimensions.

Two stimulus dimensions were designated to be critical, and particular values on those two dimensions, determined randomly for each subject, defined the target stimulus within a stimulus-pair. As we simulated the so-called "random" condition from Ball and Zuckerman, each of the values on each of the noncritical stimulus dimensions appeared eight times in a target stimulus and eight times in a distractor stimulus, across the set of 16 stimuli. Two RD layer neurons were used in the simulations. If the left stimulus was the target, then activation of the first RD neuron was considered to be the correct response; when the right stimulus was the target the activation of the other RD neuron was considered correct. (We did not model the processes that pass activity from the response decision neurons to the motor execution neurons in premotor and motor cortex.)

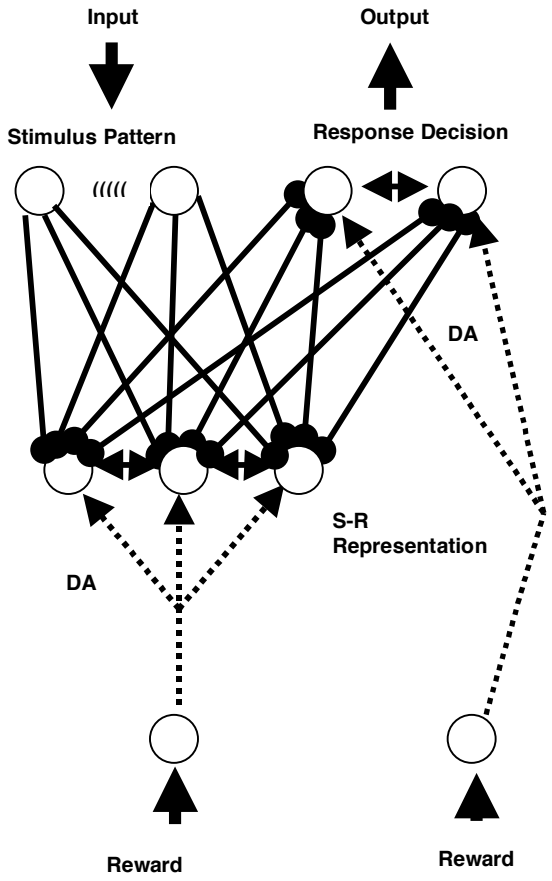

Figure 3:
A schematic architecture for the neural network model with 3 layers (Stimulus Pattern; S-R Representation; Response Decision). Dotted arrows indicate dopaminergic reinforcement projections arising from cells that are activated by primary and secondary reward stimuli. Double-headed horizontal arrows indicate inhibitory interactions within the S-R Representation and Response Decision layers of the model. Round arrowheads indicate modifiable synapses, and where these are double-headed reciprocal connections between layers are included. Only a subset of neurons in the Stimulus Pattern and S-R Representation layers are depicted for clarity. *Abbreviations*: S-R stimulus-response; DA dopaminergic reinforcement signals.

When a correct response was made by the network, a positive reinforcement signal (with a strength represented by parameter R^P) was used as part of the learning rule (see below). The parameter R^P had a value of zero when there was no positive reinforcement (as occurred after an error in Ball and Zuckerman's positive reinforcement conditions). We did not model the processes by which the positive reinforcement signal could arise, as a result of the positive reinforcement provided to the subject. We could have done so, in a relatively straightforward fashion, by adding elements from Brown et al's (1999) model to our current simulation.

Ten SRR layer (striatal) neurons were used in the simulations. There was 100% feedforward connectivity between the 32 neurons of the SP layer and the 10 neurons of the SRR layer, and 100% connectivity between the 10 neurons of the SRR layer and the 2 neurons of the RD layer. The initial weights of these connections were drawn from random normal distributions (mean=0.3 and s.d.=0.065 for SP layer to SRR layer; mean=0.5 and s.d.=0.125 for SRR layer to RD layer; any values randomly falling below 0 were set to 0).

The simulations created 100 simulated subjects and each subject had a unique random weight initialisation according to the above parameters. A training epoch was a set of 16 trials in which each stimulus pair was presented once. Each epoch used a different random order of the stimuli, and the orders for each epoch were randomised for each subject individually. A critical element of the simulation was the value taken by the positive reinforcement parameter (R^P) when the response was followed by positive reinforcement (R^P was 0 when responses were wrong). The value was varied across simulated subjects to reflect the individual sensitivities of the BAS to reinforcing stimuli, but the same value was used for all the positive reinforcements experienced by a particular subject. High values reflected large reactions to positive reinforcements delivered in the experiment, and therefore any simulated subject with a high value was considered to have high levels of BAS-related personality traits. (The R^P values for positively reinforced trials were drawn from a random normal distribution with mean=2 and s.d.=0.3) Thus, there were only three factors which varied across subjects: the initial weight randomisation; the random order of the training items; and the value of the reinforcing effect produced by a positive reinforcer.

Training continued until the criterion of five consecutive correct responses was reached and the number of trials required for each subject was recorded. A nonreversal shift was not used in the simulation; instead training continued until five epochs were completed, by which time all subjects had comfortably reached the learning criterion.

Before presenting the results of the simulations, we will give the equations used in the model. We can define e_j as the excitatory input to the jth neuron in the SRR layer. The equation for e_j is standard in neural networks:

$$e_j = \Sigma_i \, w_{ij} * x_i^{\text{out}} \qquad \ldots (1)$$

where x_i^{out} is the output from ith SP layer neuron and w_{ij} is the weight from ith SP layer to the jth SRR layer neuron. The SRR layer neuron with the largest excitatory input was then deemed the winner of the competition within the SRR layer, and had its output set to a value of one; the outputs of all the other SRR layer units were set to zero. The output from the active SRR layer neuron activated the RD layer neurons (according to an equation identical to Equation 1 above) and the RD layer neurons then entered a competition from which one emerged as the winner. It was assumed (but not modelled) that the active RD neuron then triggered its associated response, and then the environment provided positive reinforcement if the response was correct.

After the response was made, and any positive feedback was received, the network underwent learning according to a three-factor learning rule. Neural activities and outputs were reset between trials, but the weight values evolved, according to the learning rule, from trial to trial, without any decay from one trial to the next. The learning rule specifies the change in weight brought about by a single trial. We can write the following equation describing Δw_{ij}, which is the change for a synapse between the *i*th SP and *j*th SRR neurons:

$$\Delta w_{ij} = y_j^{\text{out}}(\lambda_1 R^P[x_i^{\text{out}} - \lambda_2 w_{ij}] - \lambda_3 x_i^{\text{out}} w_{ij}) \qquad \ldots (2)$$

where y_j^{out} is the output from the *j*th SRR layer neuron and λ_1, λ_2, and λ_3 are learning rate constants (0.05, 0.75, and 0.3 respectively). This is a three-factor learning rule because y_j^{out} is the postsynaptic term (factor 1), x_i^{out} is the presynaptic term (factor 2), and R^P is the positive reinforcement term (factor 3). When each of these three factors is present there is long-term strengthening (potentiation) of the weights. Note also that when factors 1 and 2 are present, but are not followed by positive reinforcement (i.e., when R^P=0, following an erroneous response), the synaptic weights are weakened at a rate determined by the constant λ_3. This is in keeping with the evidence, noted earlier, that lack of positive reinforcement may lead to long-term synaptic depression in the striatum. An identical equation (with identical learning rate constants) was used to modify the weights between the SRR layer and RD layer neurons.

Results of Simulations of the Random Condition from Ball and Zuckerman (1990)

Ball and Zuckerman (1990) did not give the mean number of trials to criterion that their subjects required to learn the pre-shift task. From their graphs, however, it can be seen that the criterion for the post-shift task was reached after an average of around 17.5 trials. The choice of the above learning rate parameters (and other network features) was constrained so that we obtained an average number of trials to criterion for the pre-shift task that was under 20 trials (in fact the average for the 100 simulated subjects was 18.9). Figure 4 shows the mean number of correct responses per epoch for the 5 complete epochs that were simulated. This figure shows that almost every subject was responding perfectly to all stimuli by the third training epoch.

From Equation (2) the rate of learning (i.e., Δw) is directly proportional to the positive reinforcement parameter, R^P. We therefore felt certain that learning performance would show a simple direct correlation with an individual's R^P value. In this task simulation, where learning was measured via the number of trials to criterion (a measure which gets smaller as learning is faster), it was predicted that a negative correlation would be found. Indeed, when presenting the model at a scientific meeting, one of us (AP) was so sure of the result that he didn't bother to carry out the simulations, claiming it was self-evident that the expected correlation would emerge (and no-one in the audience, including some experienced modellers, suggested otherwise). *We were therefore extremely surprised when it proved very difficult to find sizeable correlations between trials to criterion and R^P*. Figure 5 shows the results for the parameter settings described above. This graph does depict a very modest (but significant) negative correlation (r=-0.25, p<0.02), associated with the best-fitting regression line shown in the figure.

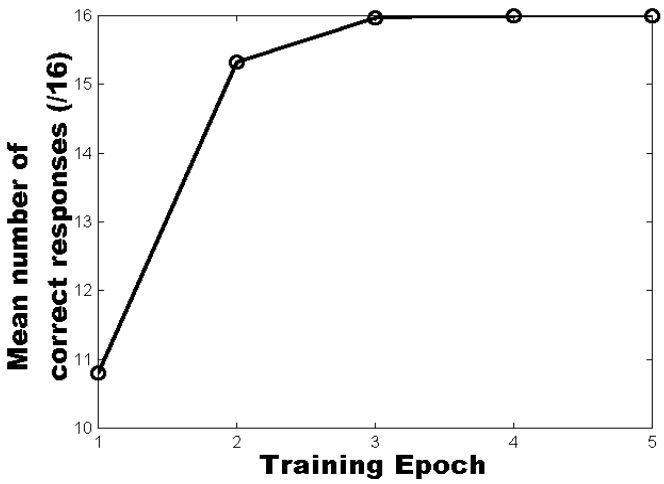

Figure 4:
The mean number of correct responses per training epoch made by 100 simulated subjects. The simulation was of the task used by Ball and Zuckerman (1990; random condition with positive reinforcement). There are 16 training stimuli presented once each per training epoch.

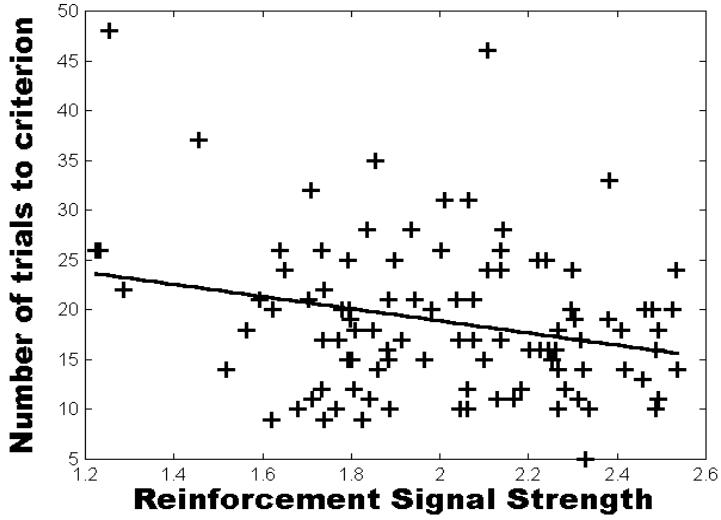

Figure 5:
Scatterplot of the number of trials taken by a simulated subject to reach the learning criterion against the value of the positive reinforcement signal which followed a correct response. The best fitting linear regression line is shown (R^2=0.06).

We chose to report the results for these parameter settings because they produced a correlation typical of the significant correlations we obtained. Many other simulations we ran, using alternative parameter values and different network properties, failed to produce a significant correlation between learning performance and R^P values. We varied almost every aspect of the model including: (a) the values of the learning rate constants (i.e., we allowed values which resulted in the mean number of trials to criterion across the 100 simulated subjects being greater than 20); (b) the numbers of neurons in the SRR layer; (c) the number of winning neurons in the SRR layer (i.e., we allowed more than one winning neuron); (d) the degree of connectivity between the SP and SRR layers (i.e., we allowed each SP neurons to have synapses with only a proportion of the SRR neurons); and (e) the mean (and s.d.) of the distribution from which individual subjects' R^P values were sampled. We also tried adding a number of other reasonable features to the model that might enhance the size of the correlation. In each simulation we also looked at whether other measures of learning performance might be more sensitive to the correlation. Nothing we did gave us a correlation substantially bigger than the one reported above; the correlations were almost all between 0 and –0.35.

Even if the small correlation reported above approximates the "correct" value for this model, we are confident that it would usually be *impossible* to detect the influence of such a correlation in a real experiment, akin to the one conducted by Ball and Zuckerman (1990). First, our simulation restricted very severely the number of individual differences that might contribute to a subject's performance (individual simulated subjects varied in their R^P value, the randomisation of their initial weight settings, and the randomisation for the order of stimuli used across training trials). Real subjects' performance is almost certain to be affected by a host of other individual noise factors, which would act to reduce any correlation. Second, even if RST were correct in essence, the BAS-related personality trait measures are themselves likely to have a far from perfect correlation with the underlying biological variables that give rise to the variations in reinforcement sensitivity. Our simulated correlation is directly between variations in learning performance and variations in degree of biological reinforcement experienced. By contrast, the correlation with personality, in a real experiment, would be diluted further by the correlation between the reinforcement variable and the BAS-traits measures being used.

The second point in the foregoing paragraph is not damaging to all computational models of this kind: we were quite easily able to construct a non-reinforcement model which was able to generate correlations of 0.8 (in 100 simulated subjects) between learning performance and the critical parameter that varied across subjects. We feel that simulated correlations need to be of this order of magnitude in order to be detectable in real experiments using personality trait measures. Incidentally, these other simulations, which do not use reinforcement learning processes, increase our confidence that a non-reinforcement model may, in principle, be able to account for the significant learning-personality correlations reported by Ball and Zuckerman.

It is important to note that our surprise finding for the reinforcement learning model holds for other kinds of learning task, and is not limited to the Ball and Zuckerman procedure. We have used the same reinforcement model to simulate other well-known category learning tasks, including ones which take more trials to master, and have similarly found a great difficulty in obtaining significant correlations between R^P and learning. Ongoing work is attempting to understand the reason for this general lack of correlation.

We feel that the findings from this modelling study, although negative in one sense, may be of considerable importance. The results might indicate a general explanation for the difficulty, which emerged from the literature reviewed above, in obtaining consistent

personality-learning correlations in experiments testing the predictions of RST. Even if RST were true with respect to positive reinforcement and personality, the simulations suggest that it may be almost impossible to detect the predicted associations for many learning tasks. The model suggests the surprising result that, although positive reinforcement may be vital for learning to occur (and there would be no learning in our model if R^P were zero for positively reinforced trials), variations in the strength of reinforcement experienced across subjects may often contribute little to the observed variance in learning performance. Going further, one might derive a practical recommendation from these simulation findings: before conducting any future studies of BAS-related personality traits and learning under positive reinforcement, it might therefore be appropriate to build a computational model of the task to see if that learning task is likely to be sensitive to variations in reinforcement across subjects.

These conclusions strengthen the view, outlined earlier, that impulsive antisocial sensation seeking personality traits may correlate with learning performance for reasons that are unrelated to positive reinforcement processes. In the next section we consider these ideas further.

Alternative Accounts for the Effects of ImpASS Personality Traits on Learning

Ball and Zuckerman's (1990) suggestion, that subjects with high ImpASS personality scores might have superior selective attention abilities to subjects with lower scores, was noted earlier in this chapter. This claim, if true, could explain superior learning performance for subjects with high, rather than low, ImpASS scores, at least on tasks requiring the selective learning of relationships involving some stimulus features, rather than others. Ball and Zuckerman's own task was of this kind, because successful learning depended on focusing on the two critical stimulus dimensions that were associated with response categories, and ignoring the six other nonpredictive distracting dimensions.

This idea resonates with suggestions we have previously made (Gray et al, 1999) concerning the functions of one of the DA systems implicated in the operation of the BAS. We presented evidence that the mesolimbic dopaminergic projection pathway causes release of DA in the nucleus accumbens (in the ventral striatum) in response to the *salience* of a stimulus, with higher release reflecting greater stimulus salience. By this account both rewards, and stimuli associated with reward, would cause DA release, because they are either biologically or associatively salient. However, the account proposes that other stimuli should activate DA release (novel stimuli, punishments, or stimuli associated with punishment), even though these other stimuli are unconnected with positive reinforcement. It is conceivable that ImpASS personality traits reflect inter-individual variations in the functioning of a system involved in stimulus salience processing: we will consider the relevant evidence below.

Latent inhibition (LI) is a task which we have argued is critically dependent upon stimulus salience processing operations in the nucleus accumbens. In LI tasks with animals, the subjects are preexposed to a stimulus that has no significant consequences. After an adequate period of such pre-treatment, it is thought that the preexposed stimulus loses salience and becomes "selected out" from the focus of attention. This is shown in the second phase of the task in which the stimulus contingencies are changed so that the preexposed stimulus is now predictive of a biological reinforcer. Because the preexposure phase has caused the stimulus to lose salience, the subsequent learning of the association between the stimulus and the reinforcer is retarded (relative to subjects who have not undergone the preexposure phase). This retardation of learning is the LI phenomenon.

Gray et al (1999) review the key evidence in support of this salience-based account of LI, concentrating on the evidence for the vital role of DA release in the nucleus accumbens. In particular, LI in animals and human beings is sensitive to treatment by DA agonists and antagonists. By defining "salience" carefully in a neural network model, we have been able to model the complex effects of these drugs on LI tasks (Schmajuk et al, 1998). To achieve this, it was necessary to equate the pharmacologically induced changes in accumbens DA release with changes to the value of the salience parameter (in the modelling work, salience has been termed "novelty").

Several LI tasks suitable for human subjects have been developed, mostly based around operant learning procedures (see Lubow & Gewirtz, 1995). There has now been a large number of studies looking at the personality traits (and other individual differences factors) associated with LI performance. A partial review of the studies is provided by the information summarised in Table 2 (but note that Table 2 does not contain an exhaustive list of all the studies.)

There is some evidence that LI may be associated with many individual differences variables (e.g., Peterson and Carson, 2000), but two principal types of personality scale have been used in these studies summarised in Table 2. One type attempts to measure schizotypal personality traits, i.e. those traits in healthy subjects that reflect tendencies towards schizophrenic-like behaviours, emotions and cognitions (see Vollema and van den Bosch, 1995, for a review of schizotypal personality measures in general). The other type measures ImpASS personality characteristics (the P scale has been the most widely used; see Table 2).

The focus on schizotypal personality arises because LI is reduced in certain types of schizophrenic patients (see Gray et al, 1999) and there is interest in seeing whether a similar change in LI can be observed in subjects who, although healthy, score highly on schizotypal personality traits. In fact, from reading the papers in Table 2 it is clear that the P scale has been included in many LI studies (including our own: Baruch et al, 1988) because it was construed as a schizotypal personality scale (in keeping with Eysenck' original designation of the scale as a "Psychoticism" scale). Careful psychometric work within mainstream personality theory (see Gibbons & Rammsayer, 1999, for a review) has shown that the P scale clusters strongly with measures of impulsivity and sensation seeking. In multivariate analyses of schizotypal personality scales, which have often included P, it is clear that the P scale forms the major part of a fourth "asocial" or "impulsive-nonconformist" schizotypal factor. This factor does not map onto a syndrome that can be seen within schizophrenia (unlike the other three "true" schizotypal factors that emerge from these analyses) but instead resembles features of borderline, antisocial, and schizoid personality disorders (see Vollema and van den Bosch, 1995, for a review).

It seems clear from Table 2 that measures of schizotypal personality and ImpASS personality traits both affect LI, in the majority of the studies, although neither type of measure accounts for a clear majority of the significant findings. Subjects who score highly on these scales show reduced LI. When in the preexposure condition of the LI experiment, these high scoring subjects learn associations with the preexposed stimulus (during the second phase of the LI task) largely as if they had not undergone the preexposure phase. Unlike low-scoring subjects, subjects with high scores on schizotypal or ImpASS personality traits appear to be treating the preexposed stimulus as if it were still salient.

Table 2.
A partial summary of studies relating LI task performance to impulsive antisocial sensation seeking and/or schizotypal personality variables.

Personality Studies of LI			
Study	*Nature of LI Task*	*Personality Scales Used*	*Significant Effects Seen*
Baruch et al (1988)	Auditory operant learning (OL)	P scale[1]; STA scale[a]; Launay-Slade Hallucination scale[b]	P and LI interaction* (low P subjects, but not high P subjects, show LI)
Lubow et al (1992)	Expt. 1: Auditory OL	P scale; STA scale	P and LI interaction (low P subjects, but not high P subjects, show LI); similar interaction for STA
	Expt. 2: Visual OL	P scale; STA scale	Same interactions as for Expt. 1
Lipp & Vaitl (1992)	Pavlovian Differential Electrodermal Conditioning	Revised P scale[2]; STA scale; Launay-Slade Hallucination scale	High STA subjects show significantly more differential conditioning than low STA in preexposure conditions; both groups show equivalent conditioning in control condition
De la Casa et al (1993; Expt. 1)	Visual OL	Psychosis-proneness (PP) groupings based on specific MMPI scale scores[c]	Low PP subjects showed LI but high PP subjects did not (with 6 mins preexposure for preexposure group); no LI for either personality group (with 3 mins preexposure); LI for both groups (with 15 mins preexposure)
Lipp et al (1994)	Expt. 3: Visual OL	Revised P scale; STB scale[3]; STA scale; Launay-Slade Hallucination scale; VSS[d]; SPQ[c]; a subset of the Chapman scales[c]	No significant personality and LI interactions
Lipp et al (1994)	Expt. 4: Pavlovian Differential Electrodermal Conditioning	Same scales as for Expt. 3	STA and LI interaction (high STA subjects in preexposure condition; and low STA subjects in control condition show significant differential conditioning); similar interactions for STB and Launay-Slade scales
Allan et al (1995)	Auditory OL	P scale; STB scale; STA scale	STA and STB scales were both associated with LI (high scores reduce LI) independently of smoker/nonsmoker grouping; P scale effect on LI not independent of smoking group
Gibbons & Rammsayer (1999)	Visual OL	P scale; Sensation Seeking scale (SSS)[4]; I_7–Impulsiveness[5] (here all scales used were German versions)	P and LI interaction (low, but not high, scorers show significant LI); similar effect for Disinhibition subscale of SSS

Personality Studies of LI			
Study	*Nature of LI Task*	*Personality Scales Used*	*Significant Effects Seen*
Hofer et al (1999)	Visual OL	STA scale	STA and LI interaction (low, but not high, STA groups show significant LI)
Peterson & Carson (2000)	Auditory OL	Revised P scale; Extra-version and NEO PI scales¶	Higher P scores were significantly associated with faster learning performance for subjects in the preexposure condition, but not for subjects in the control condition; the P effect in the preexposure condition was no longer significant after partialling out extraversion and "openness to experience" scores

Note: *All the studies included used a between-subjects LI procedure with preexposure and control (non-preexposure) conditions. In these cases, interactions between personality and LI therefore refer to a differential between-subjects LI effect for high and low scoring personality groups. ImpASS personality measures used: [1]EPQ-P (Eysenck & Eysenck, 1975b); [2]Revised EPQ-P (S.B.G.Eysenck et al, 1985a); [3]STB (Claridge & Broks, 1984); [4]Zuckerman (1979); [5]S.B.G.Eysenck et al (1985b). Schizotypal personality measures used: [a]STA (Claridge & Broks, 1984); [b]Launay & Slade (1981); [c]See article for details; [d]Venables, Wilkins, Mitchell, Raine, & Bailes (1990); [e]Raine (1991). We include the STB scale as a measure of ImpASS traits because it was designed to index personality features similar to those found in Borderline Personality Disorder, and items from ithe STB scale load (with P scale items) onto the fourth "asocial" schizotypal factor, which we argue is an ImpASS factor (see text).
We do not consider any of these scales (S.B.G.Eysenck et al, 1985a; Costa & McCrae, 1992) to measure either schizotypal or ImpASS personality traits directly.

The evidence for an effect of ImpASS traits on LI is consistent with the suggestion that these traits may index inter-individual variations in stimulus salience processing, and increases the plausibility that this suggestion might underlie the correlations between ImpASS traits and performance on other kinds of learning tasks. Given the robust effects on LI, which can be obtained by manipulations of dopaminergic neurotransmission, then one might argue that the ImpASS-LI correlations provide a further line of support for the claim that ImpASS traits are associated with variations in dopaminergic neurotransmission. The other evidence in line with this claim was reviewed earlier. We have also noted the specific proposal (Gray et al, 1999; Schmajuk et al, 1998) that it is the mesolimbic DA projection to nucleus accumbens which is critically involved in processing the salience of a stimulus. This idea might then lead to the more refined suggestion as to the localisation of the dopaminergic substrate of ImpASS traits.

Nonetheless, it should be noted that some measures of ImpASS traits (e.g., the P scale) show moderate, positive, and significant correlations (e.g., between 0.4 and 0.6 in our data from student samples) with measures of "true" schizotypal personality factors such as the Unusual Experiences and Cognitive Disorganisation scales developed by Mason and Claridge (Mason, Claridge, & Jackson, 1995). This observation would seem to raise three possibilities: (i) that both ImpASS measures and true schizotypal personality measures are independently associated with LI; (ii) ImpASS measures are associated with LI but true schizotypal measures appear to affect LI only by dint of their association with ImpASS measures; or (iii) true schizotypal measures are associated with LI but ImpASS measures appear to affect LI only by dint of their association with true schizotypal measures. If

possibility (iii) were true, then effects of ImpASS traits on learning, such as those reported by Ball and Zuckerman (1990), may actually have been proxies for a real underlying association between schizotypal personality and (certain types of) learning.

We know of no published data that strongly distinguish between the above possibilities. However, the study by Gibbons and Rammsayer (1999) may be suggestive. They showed that both the P scale and the Disinhibition subscale (from Zuckerman's Sensation Seeking scale) were associated with LI. For subjects in the preexposed condition of their LI task, high scorers on these scales showed significantly greater learning, relative to low scorers. The corresponding correlations for subjects in the non-preexposed control condition were not significant. Importantly, regression analyses showed that both *P* and Disinhibition explained significant independent portions of the variance in the learning performance of subjects in the preexposed condition. This result encourages the view -- possibility (i) above -- that correlated personality traits may be independently associated with LI performance. Perhaps the association between LI and P, independent of Disinhibition, reflected an influence of schizotypal personality factors (indirectly assessed by the P scale), while the association between LI and Disinhibition, independent of *P*, reflected the influence of ImpASS traits. Clarification of this issue must await further data, but should be pursued in parallel with studies which try to establish more clearly the nature of the psychological processes (such as salience) that might underlie personality-learning associations across a range of tasks.

Summary

In this chapter we have considered Reinforcement Sensitivity Theory's proposal that certain personality traits reflect variations across people in the sensitivity of the Behavioural Activation System (BAS), which processes positive reinforcing stimuli. We have suggested that such BAS-related traits may depend on dopaminergic neurotransmission, but have as yet come to no firm conclusion about the precise nature of the traits involved. There is evidence to suggest that both extraversion and impulsive antisocial sensation seeking may be linked to functioning of the brain's dopamine systems, and, on this basis, either type of trait plausibly relates to BAS functioning.

We reviewed the published evidence testing the predicted relationship between putative BAS-related traits and learning performance under positive reinforcement conditions. We concluded that the evidence in support of the theory was scant, and found that promising significant effects were usually either unreliable, were less consistent with the theory than they first appeared, were contradicted by other findings, or were complicated by other, unpredicted, significant effects. This conclusion is qualified by our view that few studies have tested the theory adequately. However, some methodologically sound studies (e.g., Ball and Zuckerman, 1990) have found significant correlations between measures of impulsive antisocial sensation traits and learning, but these correlations appear to be independent of positive reinforcement. At the end of the chapter, we briefly considered an alternative account for these findings in terms of individual differences in processing the (relative) salience of stimuli and their constituent features.

During this chapter, we also constructed a simple, but biologically-constrained, neural network model of learning under positive reinforcement. To our great surprise, we found that it was very difficult to obtain significant correlations between the learning performance of simulated subjects and the modelled individual differences in the strength of the positive reinforcement signals that simulated subjects experienced after successful responses during the task. These simulation findings suggest that, even if RST were true, it might be very

difficult in real experiments to obtain the correlations between personality and learning under positive reinforcement that the theory would predict. The fact that the real experiments have indeed so often failed to yield the predicted correlations may therefore be less damaging for RST than would otherwise be the case. In future studies we need to ascertain that performance on the learning tasks used is likely to be affected by individual differences in reinforcement sensitivity. Only then can we confidently try to establish the personality dimensions that may be related to such individual differences. Neural network models may have a role to play in determining our choice of tasks for this future research.

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