

Addiction, Personality and Motivation

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It is suggested that addictive behaviour, so called, fits into a *psychological resource* model. In other words, the habits in question are acquired because they serve a useful function for the individual, and the nature of the functions they fulfil is related to the personality profile of the 'addict'. For some people this resource function develops into a form of addiction, and it is suggested that the reason this occurs is related to excessive *dopamine functioning*. This in turn is used to suggest the nature of the *addictive personality*. Excessive dopamine functioning is related to the personality dimension of *psychoticism*, and evidence is cited to the effect that psychoticism is closely related to a large number of addictions. The precise reasons for the addictive effects of dopamine are still being debated, but clearly there is a causal chain linking personality and biological factors together in the production of addictive behaviour. © 1997 by John Wiley & Sons, Ltd.

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INTRODUCTION: THE RESOURCE MODEL OF ADDICTION

The term 'addiction' is widely used to characterize the tendency to indulge in certain types of behaviour to an unusual and possibly harmful extent, addicts often finding it difficult or impossible to terminate such behaviour without outside help, or even with it. Such behaviour often involves drugs (alcohol, amphetamine, cocaine, heroin, etc.), but not necessarily so; in popular parlance one can be addicted to sex, sport, pornography, travel, or work (workaholics). There are two major *models* of addiction, the *medical* or *chemical* (physical addiction) and the *psychological* (resource model). As Gilbert (1995) and Warburton (1990) have pointed out, the term 'addiction' has little scientific meaning, being employed in different ways by different writers, and having no agreed interpretation or underlying theory. It is not even known whether addictions (using the term in its widest, common sense meaning) is *specific* to one substance or activity, or *general*, i.e. covering several different areas. Often the term is used in a *pejorative sense* to suggest that the behaviour in question is a form of disease, requiring medical intervention. Voss (1992) has given a list of the criteria for distinguishing between habituation (or resource use) and true or medical addiction;

these run as follows: (1) want; (2) freedom of choice; (3) psychological dependence; (4) physical dependence, increased tolerance, escalation of dosage, withdrawal, craving; (5) moral deterioration; (6) intellectual reduction; (7) mental dissolution; (8) social collapse. He points out that while alcohol and drugs fit all but one of these (freedom of choice), smoking does not; nor does it remove freedom of choice. This does not remove the possibility that alcohol and drugs may also have a resource component; the glib use of the term 'addiction' for habituation serves no useful purpose. It may have some meaning if applied to certain drugs, and to certain people. No generalization should be offered without specific proof covering Voss's eight points. These cover what we might call 'genuine' or 'medical chemical' addictions; in this paper we are using the term in a much broader, non medical sense.

The view taken here is that the term 'addiction' refers to certain types of behaviour that can be interpreted as constituting a *resource* for the person concerned; in other words, the behaviour confers certain benefits on that person, and hence the behaviour in question is continued even though there may be certain unwanted consequences, usually occurring only in a statistical fashion (risk ratios), and after a considerable period of

time. As an example, consider smoking. I have argued that nicotine has a biphasic action, increasing cortical arousal in smaller doses, and decreasing tension in larger doses (Eysenck, 1980). These effects can be reinforcing, the former in extraverts attempting to *raise* their abnormally low level of cortical arousal, the latter in emotionally unstable people attempting to *lower* their tenseness. This analysis suggests that smoking may be related to personality, in the sense that people high on extraversion or neuroticism are more likely to smoke than people low on either or both these personality traits (Eysenck, 1980). As Gilbert (1995) has shown, both propositions have found considerable support in a number of empirical studies.

It would seem to follow that if people smoke to receive certain benefits from smoking (resource theory), they would continue in this behaviour because it was reinforcing, and it would be difficult to wean them away from it. The problems encountered by most 'quit smoking' programmes bear testimony to this; initial successes are usually followed by large scale returns to smoking by many subjects of such trials. It would also seem to follow that if we could offer smokers alternative ways of obtaining the type of satisfaction they obtain from smoking, e.g. by teaching high neuroticism scorers relaxation methods to reduce tenseness, the effect on smoking would be stronger and more lasting. O'Connor and Stravinski (1982) have demonstrated that this is indeed so, thus giving strong support to the resource theory.

PERSONALITY AND ADDICTION

Can we extend such a personality type theory to the problem of addiction? Obviously some people find it easier than others to give up addictive sources of gratification. Many US soldiers acquired the habit of smoking opium in Vietnam, but had no difficulty giving it up on their return; others became hopeless addicts. One possible difference may be found in the circumstances encountered by the people concerned. Many people took up smoking during the war because of the stress involved, and had no difficulty in giving it up after the war, because the stress was removed. A resource model can easily explain such examples of quitting made easy by changing circumstances. However, clearly this is not enough, because 'addictive' people remain wedded to their addiction in spite of changing circumstances. This raises the possibility that there may exist an 'addictive personality',

i.e. a type of person who is readily addicted to certain types of behaviour which are reinforcing, and will continue to indulge in these behaviours even after the circumstances giving rise to them have changed. It is this possibility that is being discussed in this paper.

In this sense of there existing an 'addictive personality', we would expect genetic factors to play an important role, because genetic factors are known to be a major determinant of practically all known personality traits, and because the major dimensions of personality implicated in addiction in particular are known to have high heritabilities (Eaves *et al.*, 1989). Turner *et al.* (1995) have discussed genetic approaches in behavioural medicine in detail, and there seems to be little doubt about the involvement of genetic factors in alcoholism (Cardoret *et al.*, 1985; Searles, 1988; McGue, 1995), and smoking (Eaves and Eysenck, 1980; Rowe and Linver, 1995; Heath and Madden, 1995). Eating disorders and obesity, too, have been shown to have a genetic basis (Cardon, 1995; Meyer, 1995; Spelt and Meyer, 1995). There is no direct evidence that identical genes are involved in different types of addiction, but if they are, then similar personality factors should appear in connection with each.

What is meant by 'personality' here is much more than just a characterization of a person in terms of traits of one kind or another. Figure 1 will make it clear that psychometric traits do indeed fill the centre of the picture, but such trait characterization is only part of a much larger nomological network (Eysenck and Eysenck, 1985). There is much evidence that all aspects of personality are strongly determined by *genetic factors* (Eaves *et al.*, 1989). DNA cannot, of course, affect behaviour directly, and hence we have *biological intermediaries* (proximal antecedents) linking DNA and behaviour. Theories of personality can be tested in the experimental laboratory (proximal consequences), and finally give rise to predictions involving *social behaviour* (distal consequences). The alleged 'addictive behaviours' would fall into this last category, and hence would require not only a link with psychometric personality traits, but also with biological antecedents. We have tried to go some way towards filling in the various parts of such a systemic view.

The first step in such a search for causal connections must be an inductive one, namely a search for personality correlates of addiction. There are three major dimensions of personality, P (psychoticism),

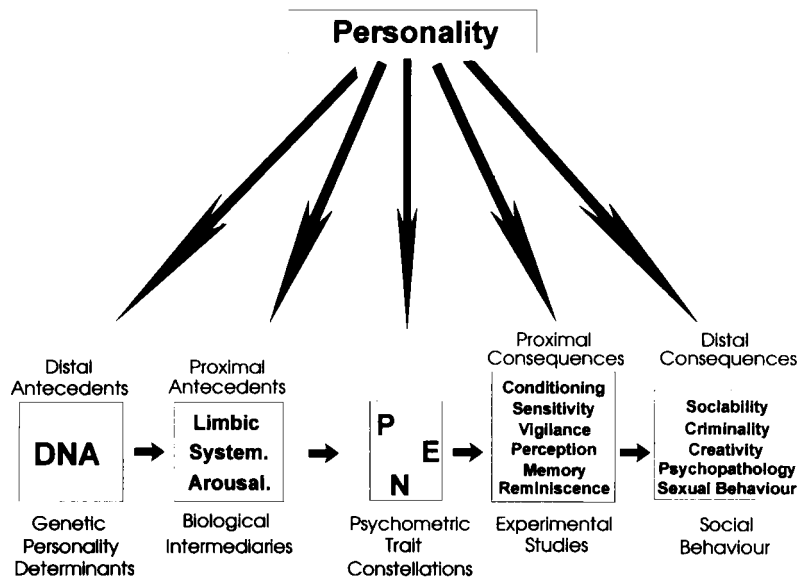


Figure 1. Nature of personality structure

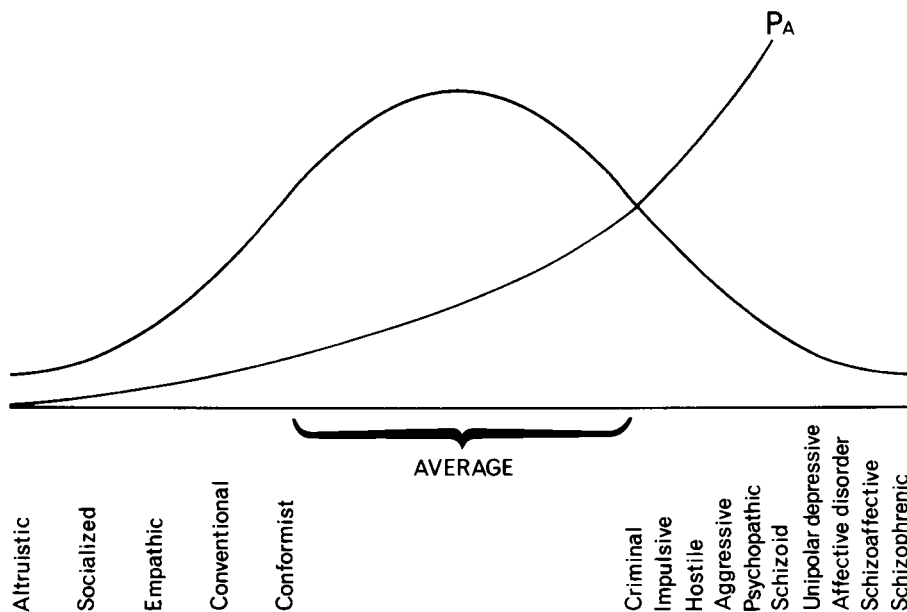


Figure 2. The psychoticism continuums, with P_A denoting increasing probability of functional psychosis with higher psychoticism scores

E (extraversion) and N (neuroticism); these are uncorrelated with each other, and cover different areas of personality (Eysenck and Eysenck, 1985). As we shall see, it is particularly the psychoticism dimension that has been found to be correlated with addictive behaviour, and hence a few words

may be useful in introducing it. The underlying theory states that there is a dimension of personality which relates to a person's liability to functional psychosis, as shown in Figure 2 (Eysenck, 1992). Psychoticism measures a dispositional variable; P has to be combined with stress to produce

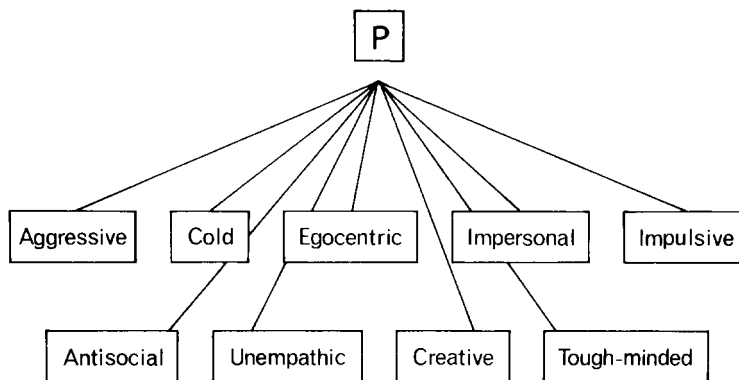


Figure 3. Traits characteristic of psychoticism

actual psychiatric symptoms. We are dealing throughout with non-psychotic individuals in our studies, but of course the biological substratum of P would have to be similar to, or identical with, that of schizophrenia to make the theory acceptable. Gray *et al.* (1991) have argued that there is indeed such a similarity as we shall discuss presently.

The actual traits which intercorrelate together to make up the higher-order factor of psychoticism are shown in Figure 3; the evidence for the existence of such a factor, and the evidence for its identification as psychoticism, are given elsewhere (Eysenck, 1992). Is it true that addictive behaviour is largely determined by P, and to a smaller extent by N (neuroticism)? Early studies by Gossop (1978) and Teasdale *et al.* (1971) showed that drug-dependent groups had typically high levels of psychoticism, together with elevated scores on neuroticism; they also had somewhat lower levels of extraversion than controls.

A larger and more detailed study comparing drug addicts and controls was carried out by Gossop and Eysenck, (1980) who found that for both males and females high-P was an important discriminant, with high neuroticism (N) also important, but less so for women than for men. They also suggested that the high-N scores might have been inflated for various reasons. Low extraversion (E) scores were also again characteristic of drug addicts. The test used also contained a Lie Scale (L) which essentially measures conformist behaviour, and usually correlates negatively with P; low L scorers were characteristic of the drug addicts. On the basis of these results, the authors constructed an addiction scale consisting of the 32 most discriminating items (all at $p < 0.001$). On

this scale, addicts had mean scores almost twice as high as controls (Gossop and Eysenck, 1980).

The personality patterns of criminals are similar to those of drug addicts, particularly in having high P and N scores (Eysenck and Gudjonsson, 1989). Gossop and Eysenck (1983) tested 221 drug addicts and over 1000 criminals on the P, E, N and L scales. They found addicts higher on P, lower on E, higher on N (particularly the women), and lower on L. In other words, the differences in personality patterns are similar to those obtained with normal controls.

These studies were done with traditional drug takers. Smokers, if we are willing to consider them 'addicted' in the sense of continuing to smoke cigarettes in spite of many health warnings, have been found to have high-P scores, and it may be noted that nicotine is an indirect dopamine agonist (Spielberger and Jacobs, 1982; Gilbert, 1995). The relevance of this point will be made clear below; just note that dopamine plays an important part in the Gray *et al.* (1991), as well as in other theories of schizophrenia.

As far as alcoholism goes, two dimensions appear to be relevant to its aetiology (Sher, 1991; McGue, 1995). The first resembles psychoticism, with characteristics like impulsivity, inattention and character disorders. The second is neuroticism, or 'negative emotionality', with a tendency to experience negative moral states and psychological distress.

Rather more interesting and unusual is work with bulimics who have been suggested to share many similarities with addicts (Garrow *et al.*, 1975). The outcome was a clear confirmation of the hypothesis, with patients having higher P and N scores, and lower E and L scores, than controls

(Feldman and Eysenck, 1986). The study was repeated by Silva and Eysenck (1987), with similar results, comparing 59 female patients suffering from anorexia nervosa with 122 bulimics; the bulimics score significantly higher than the anorexics on P and N, and lower on L. On the addiction scale they also scored significantly higher. Another unusual sample was made up of gamblers (Blaszczynski *et al.*, 1985), who had significantly higher P and N scores than controls.

Observed personality characteristics of drug addicts are not culturally determined but can be observed in other cultures as well as in Europe. A Saudi Arabian group of drug addicts was tested by Abu-Arab and Hashem (1995), showing again the same high P-high N patterns observed in European subjects. These authors also refer to another study by Abu-Arab (1987), showing similar correlations with alcoholism (see also Hurlburt *et al.*, 1982).

In a recent study, Mann *et al.* (1995) used the NEO Personality Inventory (Costa and McCrae, 1991), which has two scales (A — agreeableness and C — conscientiousness) which have a high negative correlation with P; they also have scales for E and N. They compared a group of addicts with controls, and found the expected differences, with addicts lower on A and C, and also on E, but higher on N. I have been unable to find any studies of addiction that found results in a direction opposite to that indicated, i.e. high P, high N, low L and possibly low E. Sex differences do not change this pattern, but women have less elevated N differences. Particularly impressive are the universally high P scores of addicts, as demanded by our theory.

These are just a few of the early studies using the Eysenck Personality Questionnaire scales. Francis (1996) has listed all available studies for addiction to alcohol, opium, heroin, benzodiazepines, etc.; in all, he found 19 studies specifically linking P and addiction, and 23 linking N and addiction. (The larger number of studies using N is due to the fact that many more investigators used N scales than P scales). Extraversion gave 10 negative and two positive correlations with addiction, as well as 12 studies without significant results. The Lie Scale shows seven studies giving negative correlations with addiction, two with positive, and three with insignificant correlations.

Francis summarized his survey of addictive behaviour by saying that the literature 'confirms that psychoticism is a key personality factor in this area'. Furthermore, 'the majority of studies also

confirms a clear relationship between neuroticism and the use of drugs and alcohol'. However, 'the relationship between extraversion and the use of drugs and alcohol is much less clear'. Francis adds his own rather novel investigation of personality and attitude towards substance use among 13–15-year-old children, using a large sample of 19 349 subjects. A negative attitude to drug usage correlated 0.34 with P, +0.33 with L, 0.16 with E and 0.03 with N. Controlling for sex slightly raised the correlation for P, L and E, leaving that for N unchanged. In so far as attitude is predictive of use and abuse, these figures support findings on addiction, except for the low values for N.

Given that P is the major element in addiction, it may be worthwhile to enquire about the prevalence of addiction in two large groups characterized by high P scores, namely criminals (Eysenck and Gudjonsson, 1989) and creative people and geniuses (Simonton, 1994). It is hardly necessary to discuss in detail the close relationship between addiction and criminality; this is too well known to require elaboration. As regards creative artists and scientists, the evidence has been reviewed by Simonton (1994). Note also that excessive dopamine functioning has also been found in criminals (Raine, 1993; Masters and McGuire, 1994). These links are at present merely correlational, and would surely deserve closer study, particularly as concerns causal mechanisms.

BIOLOGICAL ANTECEDENTS OF ADDICTION

We must now turn to the *biological antecedents* which characterize addiction; if our theory cannot accommodate such findings, then clearly it cannot serve the unifying function hoped for it. In what follows I shall rely very much on a theory put forward by Joseph *et al.* (1996), based as it is on much empirical work. As they point out, drugs often associated with abuse and addiction characteristically share the feature of being able to *increase neurotransmission in the mesolimbic dopamine system*. This system ascends from the neural tegmental area in the midbrain to the limbic areas associated with emotions, including the *nucleus accumbens* (NAc) and the *amygdala*. Di Chiara and Imperato (1988) have shown that various addictive drugs, such as amphetamine, cocaine, nicotine, morphine and alcohol affect extracellular dopamine levels in the NAc of the rat. The data suggest innervation from the AIO nucleus in the ventral

tegmental area, and leave little doubt that all of these drugs of abuse can activate the mesolimbic system at low doses. This is so although these common effects are brought about by diverse pharmacological action of the drugs in question.

The relationship between addiction and dopamine activity is given further support by a recent study (Fowler *et al.*, 1996). They report that brains of living smokers show a 40 per cent decrease in the level of monoamine oxidase relative to non smokers, or former smokers. They draw attention to the fact that MAO is involved in the breakdown of dopamine. 'MAO inhibition is therefore associated with enhanced activity of dopamine' (p. 733). The authors remark on the prevalence of smoking in psychiatric disorders, and of course low MAO levels are associated with psychopathological behaviour (Zuckerman, 1991). What is important for our theory is that dopamine functioning has been linked experimentally with high P scoring (Gray *et al.*, 1994); this is the fundamental link between personality and biological reality.

Von Knorring and Oreland (1985) lend further support to the involvement of MAO in addiction in a paper which studied the smoking habits of an unselected series of 1129 18-year-old men from the general population. 'Regular smokers were found to be extraverts, sensation seekers who were easily bored and with a strong tendency to avoid monotony. They also had a lower than average intellectual level, and were more prone to the abuse of alcohol, glue, cannabis, amphetamine and morphine. Furthermore, they had a low platelet MAO ... Ex smokers had personality traits, intellectual levels and platelet MAO of the same magnitude as non smokers: this may be the reason why they were able to give up smoking' (p. 327). This study strongly supports the hypothesis of a *generally addictive person*, and connects this addictive personality to low platelet MAO. The personality traits involved pertain to P as much or more than to extraversion; it is regrettable that the P scale was not used in this study.

These data suggest the existence of a general basis for addiction, in enhanced dopamine functioning, regardless of what substance is involved. One possible hypothesis to explain this generalization is based on the fact that the mesolimbic dopamine system is involved with the mechanism of *reward* and *reinforcement*. Studies involved in this type of research relate to experiments on self stimulation, drug self administration, and conditioned place preference (Fibiger and Phillips,

1988; Stolerman, 1992). What renders these results interesting is that NAc activity is also increased during and ends shortly after food reward, water reward, and sexual activity in male rats. These are all rewarding, in the sense that they reinforce (increase) the likelihood of behaviour associated with them — in other words, animals will work to obtain access to these reinforcers. Stimuli associated with these reinforcers also acquire secondary reinforcing properties through a simple Pavlovian conditioning process. Such secondary rewards are also associated with dopamine activity in the NAc (Damsma *et al.*, 1992; Phillips *et al.*, 1993). This combination of findings might suggest a simple resource theory: drugs of abuse produce an increase in dopamine in the NAc. Addiction occurs because the drugs involved produce *stronger* reinforcing effects in the brain systems of high-P (dopamine active) people than those of low-P (dopamine inactive) people.

Wise and Rompre (1985) have reviewed the huge literature on brain dopamine and reward. They point out that 'the evidence is strong that dopamine plays some fundamental and special role in the rewarding effects of brain stimulation, psychomotor stimulants, opiates, and food' (p. 270); they go on to say that dopamine is not the only reward transmitter, and that dopaminergic neurons are not the final common path for all rewards. They conclude that 'in all likelihood, the dopamine system plays some very general role in mood and movement, a role that is essential to reward function as well as to other aspects of motivated behavior' (p. 221).

There are problems associated with such a theory. In the first place many animal studies have shown that stresses and aversive stimuli of various sorts are also associated with increased dopamine release in the NAc (e.g. Young *et al.*, 1992; Saulskaya and Marsden, 1995). Thus not only *rewarding* but also *punishing* stimuli produce increased dopamine release in the NAc. And, as in the case of rewarding stimuli, secondary aversive properties acquired through a process of Pavlovian conditioning also gain the ability to increase dopamine activity in the NAc (Young *et al.*, 1992). Thus, as Joseph *et al.* (1996) point out, 'while all rewarding stimuli so far studied increase DA activity in the NAc, it is by no means the case that all stimuli which increase DA activity in the NAc are rewarding' (p. 58).

Another problem with a simple reward theory is that neutral stimuli presented in a regular temporal

association, resulting in the formation of a conditioned association, as indexed in the sensory pre-conditioning paradigm, are also associated with increased DA activity in the NAc, while the identical stimuli presented in a non-associated manner are not (Young *et al.*, 1995). Thus, as Joseph *et al.* (1996) summarize the evidence, 'increased DA activity in the NAc is associated with primary and secondary motivational stimuli, whether rewarding or aversive, and with associations between neutral stimuli which result in conditioning. Perhaps the common factor here is that all of these stimuli, or configuration of stimuli, are salient to the animal' (p. 58).

The fact that dopamine has several different effects does not necessarily rule out the possibility that it is its effect on the pleasure centres that is crucial for addictive behaviour. Aversive and conditioning effects may be irrelevant to addiction; the complexity of neurotransmitter activity is well known. But in order to maintain this theory we would require more experimental support than is available at the moment. Possibly conditioning provides another plausible bridge between addiction and dopamine, in that addictive behaviours are *conditioned* behaviours, with the positive effects of the addictive behaviours acting as unconditioned response variables reinforcing these behaviours more strongly in people more likely to form strong conditioned responses.

Joseph *et al.* (1996) put forward a rather different theory, linked with *latent inhibition* (LI) (Lubow, 1989). LI refers to an experimental two-stage arrangement in which a neutral stimulus is presented a number of times without any contingent reinforcement, e.g. a sound is presented randomly while the subject is carrying out an (irrelevant) learning task. Later on this neutral stimulus is used as the CS (conditioned stimulus) in a proper conditioning experiment, and it is found that this pre-exposure impairs the ability of the stimulus to become conditioned to any UCS. Latent inhibition is largely absent in schizophrenia, and is also much reduced in high P scorers (Lubow *et al.*, 1987, 1992; Baruch *et al.*, 1988). Latent inhibition is increased by dopamine antagonists, e.g. haloperidol, and decreased by dopamine agonists, e.g. amphetamine and nicotine (Joseph *et al.*, 1993; Warburton *et al.*, 1994). Given these facts, Joseph *et al.* (1996) argue that the effect of disrupting latent inhibition is to make familiar stimuli *salient*, or perhaps curiosity arousing. This, they suggest, may serve to make a boring life more interesting; salient (apparently

novel) stimuli produce an *orienting reaction* which increases cortical arousal levels. Increased DA release will reduce the probability that stimuli will be ignored as familiar and non productive. Again, as before, we must note that much further work will be required (perhaps by looking at indices of cortical arousal) before this hypothesis is regarded as acceptable. If reduction in latent inhibition does indeed raise cortical arousal, this might account for the fact that low extraversion is sometimes correlated with addiction; low arousal is the major psychophysiological precursor of extraversion, and heightening arousal is regarded as desirable by extraverts. High P scorers, of course, also have low arousal levels.

The different hypotheses mentioned here to account for dopamine action increasing addictive behaviour are not antagonistic to each other. It is quite possible that all are correct, and operate to a differing degree in different people, depending on their position on P, E and N. There seems little doubt that personality plays a prominent part in relation to addiction, regardless of the type of addiction, and that dopamine plays a large mediating role between DNA and personality, particularly P. These facts suggest the direction in which future research may go with advantage; there are plenty of promising hypotheses to keep such research going.

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