10 Neuropsychoanalytic notes on addiction

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Introduction

Freud had remarkably little to say about addiction, save for this one observation, made early in his psychological work (and often repeated thereafter):

> It has dawned on me that masturbation is the one major habit, the 'primal addiction' and that it is only as a substitute and replacement for it that the other addictions – for alcohol, morphine, tobacco, etc., – come into existence. (Freud, 1897:272)

A minor elaboration of this view is the following one, also made early in Freud's work:

> Not everyone who has occasion to take morphia, cocaine, chloral-hydrate, and so on, for a period, acquires in this way an 'addiction' to them. Closer enquiry usually shows that these narcotics are meant to serve – directly or indirectly – as a substitute for a lack of sexual satisfaction.' (Freud 1898:276)

In other words, during development masturbation is normally replaced by adult sexual satisfaction, but where it is not it may be substituted by (another) addiction.

These are quintessentially Freudian claims, museum-quality examples of the type. For that reason, perhaps, they are all too easily dismissed by addiction researchers today. If readers are willing to suspend judgment for a moment, though, and permit us to unpack the meaning of these claims and reconsider them in the light of contemporary neuroscience, their patience will be amply repaid.

For all of his failings, Freud had an uncanny ability to identify the essence of a psychological problem. The fundamental problem of addiction exposed by the parallel that Freud drew with masturbation is that substance abuse (like masturbation) is an affectively rewarding activity that serves no biological purpose. Substance abuse generates hedonically positive affects in the brain (or reduces negative ones; for full discussion of this 'opponent process' issue in addiction research, see Khantzian, 2003) but does not sustain reproductive fitness. In fact, it does the opposite. Addiction therefore cannot be an evolutionarily proper use of the brain mechanisms in question, but it is psychologically compelling nonetheless.

Substance abuse vs masturbation

Substance abuse appears to employ brain mechanisms that were evolutionarily designed to reward biologically useful activities (like copulation, as opposed to masturbation) (see also

Chapter 5). This is what pleasurable affects are *for*, evolutionarily speaking; they reward biologically useful actions, and thus motivate animals to perform the work that is necessary to achieve them (Solms & Nersessian, 1999; Damasio 1999). The word 'abuse' in 'substance abuse' refers to the fact that the pleasure is attained without the natural effort and persistence it was designed to reward. The motivation to perform the effortful work to achieve biologically useful goals in an indifferent and even hostile world is substituted by mere self-administration of pleasure-producing (or unpleasure-reducing) substances. This requires hardly any effort at all, aside from acquiring drugs that are not legally available, and nothing biologically useful is achieved by it. Part of the affective prize is thus attained without actually taking part in the social pursuit and competition for gratification. The biological purpose of reward is cheated. A cheap form of affect has been traded for reproductive fitness.

Herein presumably lies the root of our moral condemnation of both masturbation and addiction alike.

This conclusion seems to be the implication of the second Freud quotation above: substance abuse (like masturbation) is an inferior *substitute* for 'real' sources of satisfaction (like copulation). Substance abuse, like masturbation, represents a failure to negotiate the transition from infantile self-soothing to mastery of the real external world – the arena of all the competitions that we simply have to enter in order to survive and reproduce.

This begs the question: why and how do we normally negotiate this transition? What enables us to give up effortless masturbatory/addictive pleasures in favour of real ones, in favour of the biologically (and morally) wholesome ones? If we can trace the mechanism by which we normally traverse this transition, we will have identified the pivotal locus of the biological failure (of the psychopathology) called addiction.

It seems unlikely that evolution would have left this important task to moral persuasion (education or learning) alone. There must be some intrinsic mechanism that motivates us to forego empty pleasures in favour of the more difficult and risky business of engagement with the real world. And there does appear to be just such an intrinsic mechanism, the identification of which reveals important shortcomings in many contemporary conceptualisations of the neural basis of reward.

If the biology of reward entailed a unitary brain mechanism, such as the one that many eminent researchers still seem to claim for the mesocortical-mesolimbic dopamine (DA) 'reward system' (Haber & Knutson, 2009; Rolls, 1999), then it is difficult to imagine why animals would ever bother to negotiate the task just mentioned. Why not just go straight for the reward, and bypass the effort and risk involved in the activities that 'properly' generate them? It comes as no surprise to us to learn, therefore, that the mammalian brain does contain a more complex reward mechanism which drives us to seek our pleasures in the external world. This latter mechanism is indeed the mesocortical-mesolimbic DA system, but it turns out to not be a simple 'reward' system at all – despite the fact that *almost all drugs of abuse* (like all forms of appetitive behaviour) *do indeed massively increase DA activity in this system*.

Seeking reward

The mistaken attribution of simple reward functions to the mesocortical-mesolimbic DA system was first appreciated by Panksepp (1982, 1986). He observed that artificial

stimulation of this (D2-receptor-mediated) motivational DA system does not generate pleasurable feelings and blissful satiation in mammals, as *consummation* of a need normally should. Rather it impels the animal to excitedly *seek more of the stimulation* (in other words, it actually increases the *appetitive* drive that *leads* us to rewards, rather than the consummation behaviour that actually *delivers* them). This and subsequent research led Panksepp to rename the DA 'reward' system a 'SEEKING' system (Panksepp 1998; capitals in the original). The SEEKING system motivates animals to engage with the world – to eagerly forage, to curiously explore, to optimistically expect – in short, *to look to the outside world for attaining pleasurable experiences*. The SEEKING system is stimulated into action internally by medial hypothalamic 'need detector' mechanisms, and externally by enticing opportunities in the world. And when it is activated it impels the animal to engage with the real objects that satisfy its inner needs. This *in turn* generates pleasurable experiences via PLEASURE-LUST (and other) systems, which utilise the SEEKING system for their hedonic ends but which are not themselves mediated by DA alone. These systems will be discussed below.

Berridge and Robinson (1998) were driven to very similar conclusions by their own research findings, which eventually led Berridge (2007) to draw an analogous (but as we shall see, not identical) distinction between the brain mechanisms for 'wanting' and 'liking' in mammals, with 'wanting' being just one step removed from SEEKING. (A full history of the diversity of views in this theoretical hornet's nest is summarized in Alcaro et al, 2007, and Panksepp & Moskal, 2008.)

It is not difficult to see, in light of our reflections on Freud's observations, why an instinctual SEEKING or 'wanting' mechanism would have evolved alongside the PLEASURE or 'liking' mechanism. It is a sad but incontrovertible fact that all our needs cannot be met narcissistically, by mere *feeling* of reward versus *actual achievement* of reward. Biological needs represent a true *lack* in the organism that can only be rectified by an object (and usually a rather specific object) in the outside world. This is one of the great facts of life, and an ultimate source of all its struggles.

The distinction between the brain's appetitive and consummatory 'reward' mechanisms helps to make sense of the fact that addicts do not generally find their substance-induced DA surges to be pleasurable; at times they do not even *like* the objects of their addictive *wants* (Kassel, 2010).

Most recent addiction research into the 'wanting' aspect of the brain's so-called reward mechanisms has, however, not been interpreted within Panksepp's framework as summarised above. It has been interpreted on the view that 'wanting' is equivalent to something quite different from simple SEEKING, something called 'incentive salience' (Robinson & Berridge, 2003). On this view, DA activity is said to *predict* which objects are likely to produce pleasurable experiences (incentive), and thereby to motivate the animal to selectively attend to such objects (salience). Addiction researchers who follow this view (eg, Volkow et al, 2009) accordingly argue that drug-induced DA surges make addicts over-incentivise the drugs that generate such surges – and associated environmental cues – to consider them excessively important (inappropriately salient).

A moment's thought, however, reveals a significant conflation in this view. Drug-induced surges in salience attribution should incentivise the addict to pay extra attention to the *pleasure-generating* things they come across while high, not to the thing (the DA agonist)

that merely induced the high. On Berridge's own theory, the DA agonist is not intrinsically affect-generating. The foraging behaviours (and the associated curious, interested, expectant, optimistic feelings) that DA actually induce, merely increase the chances of the animal encountering the real objects of their needs – the objects that they 'like'. This, a second stage that can only follow the primary 'SEEKING/wanting' process, is what is actually rewarding.

The incentive salience mechanism that Berridge and others attribute to the mesocortical-mesolimbic DA system seems to be a higher-order process in the complex cascade of appetitive eagerness. Firstly, the animal has to (1) be driven to seek the objects of its biological needs in the outside world, surely an invigorating hedonic activity, before it can (2) experience the pleasurable rewards that such objects generate, which in turn enables the animal to (3) learn from such experiences – ie, associate specific objects with the pleasurable relief of each need. Only then can the animal have any basis for predicting biologically appropriate pleasures from the sight or smell of specific objects (ie, attribute incentive salience). In short, incentive-salience has to rely on past learning, while SEEKING is an intrinsic emotional-affective system that allows learning to occur.

Panksepp has always emphasised the intrinsically 'objectless' quality of the SEEKING instinct, calling it 'a goad without a goal' (Panksepp, 1971). Learning comes later, and involves much more than instinct. Learning is a by-product of the encounter between instinct and environment. Learning would never occur if it were not for the existence of a primary instinct toward engagement with the environment – with all the effort, frustration and risk that this entails.

Incentive salience – a learning-based mechanism – therefore reveals itself to be (in Freudian terms) an 'ego' rather than an 'id' mechanism. Ironically, this is also where moral persuasion (the 'superego') may enter the equation. It is no accident that recent research on the role of incentive salience in addiction has strongly emphasised the contribution in addictive pathogenesis of aberrant *inhibitory controls* on the underlying mescortical-mesolimbic DA activity (Baler & Volkow, 2006). All the influences exerted by parents, upbringing, education and the like have their effects on these higher ('learning') aspects of the transition from self-soothing to object mastery. We should therefore be on our guard against overly reductionist attempts to elucidate the basic molecular mechanisms of incentive salience conditioning (Chen, Chen & Chiang 2009) as if it were the same as the primary-process motivation employed in SEEKING resources. The variability contributed to any psychopathology by higher-order environmental factors such as parenting, upbringing and education, as Freud taught us, can be very complicated indeed.

It is to the first two steps in the three-step process outlined above that we must return, then, if we wish to elucidate the hard-wired, instinctual ('id') mechanisms leading from self-soothing to object mastery. There are very special hedonic feeling components to the instinctual systems that support those aspects of survival which require a confrontation with the real world.

We have said already that the first step in the process must be a basic SEEKING tendency, which drives the animal to 'forage' in the outside world, triggered by its detection of an inner need. Why, then, is this simple and primary DA-activated process so heavily implicated in addiction? After all, SEEKING is the step in the putative process that leads the animal *away*

from narcissistic self-soothing (from Freud's 'masturbation'). Why then do substances of abuse *increase* activation of this DA mechanism? Does this not reveal a contradiction in the parallel that Freud drew between masturbation and addiction (which he saw as a fixation upon or regression to self-soothing)?

It certainly would be a contradiction if it were not for the important finding (made in relation to cocaine addiction in the 1980s, and in subsequent studies in relation to methamphetamine, alcohol and heroin, too) that D2 receptors are consistently *decreased* in addicts, even long after the resolution of acute withdrawal effects (Volkow et al, 1990, 1993, 2004). Recent research has also shown that relatively decreased D2 receptors *precede* the development of an addiction – that it may in fact be an important biological marker of addictive vulnerability (Nader et al, 2006; Volkow et al, 2006, 2007). From an affective point of view, this condition would be the opponent-process of the appetitive-SEEKING reward that temporary (ie, artificial pharmacological) arousal of dopamine systems promotes (Johnson, 2008; Khantzian, 2003; Koob & LeMoal, 2001).

These findings are currently being interpreted to mean that individuals with blunted capacity to attribute 'incentive salience' gradually come to learn that only substances that can produce massive surges of D2-mediated activity are salient. But in light of our differences with salience attribution theory in this context, especially at a primary-process level, a better interpretation might be that individuals with blunted SEEKING capacities come to learn (especially if not otherwise helped by parents, educators and the like) that substances which produce massive surges of D2-mediated activity enable them to *gain access to pleasurable experiences and objects in the outside world* that would otherwise be relatively inaccessible to them. The object of the addiction would then not just be the stimulant substance itself – as incentive salience theory suggests – but rather the possibility (or expectation, or even hope) of gaining social, sexual and other biologically useful rewards that the substance artificially evokes. This alternative explanation of the link between reduced D2 receptivity and addiction has important clinical implications, and we believe it deserves careful consideration in future research.

For now, however, coming full circle, it appears that the main focus of the addiction process must still fall back, to a substantial degree, on the pleasurable aspects of rewards. This, and not just the primary SEEKING instinct or the drugs that stimulate it (or the paraphernalia associated with those drugs), seems to remain the ultimate psychological object of addiction, at least at the higher levels of the mental apparatus. Thus, pleasure (or its habituation, and hence relief from the ensuing pain) must remain big-ticket items in addiction research.

Pleasure and opioids

As we have already noted, the PLEASURE-LUST, or 'liking', aspect of the reward process is not just DA-mediated (although DA has a role to play in it). It is generally accepted on current knowledge that the main aspect of the PLEASURE process is mediated by opioids (acting on mu and delta receptors in the basal forebrain region in particular; see Panksepp, 1998; Berridge, 1996). These are very ancient brain molecules, which are thought to have evolved in the brain initially for their *hedonic* properties, but they also served endogenous

analgesic functions and therefore came later to ameliorate the behaviourally more complex *pain of social loss* (Panksepp, 1998). This latter instinctual mechanism – which Panksepp calls the PANIC-GRIEF system – is especially highly developed in mammals, which are exquisitely social animals (but it must have evolved earlier, as it is also present in birds). This system has its epicentre in a neuronal network that courses between anterior cingulate gyrus, various dinecephalic nuclei and the dorsal periaqueductal grey.

The hedonic, analgesic and social-soothing properties of opioids are difficult to separate entirely, especially when considered from the lived viewpoint of what a substance abuser is trying to achieve. A diagnostic differentiation of this kind would certainly be clinically important, as indeed is the more basic distinction we have already drawn between those who are seeking DA stimulation and those who are seeking opioid-mediated euphoria or relief (cf. 'uppers' versus 'downers'). But now we must finally consider the opioid systems as a whole in relation to the Freudian formulation of addiction that we are considering here.

It is easy to see the link between an opiate-induced hedonic fog and the narcissistic delights of masturbation. We have likewise already provided an answer to the question as to why animals take the trouble to transcend masturbation, and engage instead with the outside world in pursuit of pleasure and relief from pain. The answer was found in the fact that a primary SEEKING instinct exists, alongside various PLEASURE-LUST instincts. This implies that masturbatory pleasure, while satisfying the second of these, leaves the first of them (the object-seeking instinct) dampened for a while but, in the final accounting, unsatisfied. All at once, this insight throws the pivotal role of the other opioid-mediated instinct, PANIC-GRIEF, into sharp relief.

As already mentioned, this system evolved in order to foster social bonds – first and foremost between infants and their mothers, then between sexual mates, and ultimately between social groups of all kinds (including families and clans). It is easy to see the adaptive advantages of such an instinct, which attaches mothers (and, to a lesser extent, fathers) to their genetic offspring, the offspring to their major sources of survival care, and genetically related conspecifics more broadly to each other. The price we have to pay for this evolutionary advantage, though, is the pain of social loss: separation distress (PANIC), sadness and despair (GRIEF). The avoidance of such pain is what keeps us together: neurochemically speaking, we cling to our mothers and lovers in order to keep our mu-opioid receptor activity contentedly high.

Now, it is of the utmost importance to note that the 'attachment' processes initiated by this instinctual system *have all the hallmarks of addiction*. Consider for a moment the following tabulation of the similarities between substance addiction/withdrawal and social attachment/loss (see Table 10.1). The analogies are extremely striking. It comes as no surprise to learn, therefore, that opiates were historically the first line of treatment for depression (for summary, see Tenore, 2008). Why, then, did we stop using them for this purpose? For the simple reason that they are so addictive!

So, *attachment is a primary form of addiction*. Anyone who has fallen in love knows the truth of this statement. Being in love with someone is almost indistinguishable from being addicted to them. This, surely then, is the major biological endophenotype that is hijacked by substance abuse.

Table 10.1: Summary of the major similarities between the dynamics of opioid dependence and key features of social attachments

SIMILARITIES BETWEEN	
OPIATE ADDICTION &	SOCIAL DEPENDENCE
1) Drug dependence	1) Social bonding
2) Drug tolerance	2) Estrangement
3) Drug withdrawal	3) Separation distress
a) PSYCHIC PAIN →	a) LONELINESS
b) LACRIMATION →	b) CRYING
c) ANOREXIA →	c) LOSS OF APPETITE
d) DESPONDENCY →	d) DEPRESSION
e) INSOMNIA →	e) SLEEPLESSNESS
f) AGGRESSIVENESS →	f) IRRITABILITY

Source: Panksepp (1998)

But where does this leave Freud's claim regarding masturbation? In our opinion, it encourages us to take a deeper view of what masturbation actually entails. Although it is difficult to know what is going on in the mind of a masturbating infant (it is important to remember that Freud included pre-genital pleasures – thumb-sucking, etc – under this heading), it seems unlikely that it entails pleasurable sensations alone, devoid of representational contents. Certainly this applies to the masturbatory fantasies of adolescents and adults. What excites the pleasurable sensations is not manual stimulation alone but almost invariably the presence of an imaginary partner, or at least of another person of some kind. In short, what distinguishes masturbation from actual copulation is not so much an absence of object-seeking as a *frustration* of object-seeking. One masturbates for *lack* of an object (whatever the reason for that lack might be). This is why masturbation is considered inferior to copulation, not only by society, but by the masturbator too. Masturbation is ultimately an empty source of pleasure, in a very literal sense. Masturbation involves frustration of the SEEKING instinct, plus satisfaction of the PLEASURE-LUST instinct, which equals empty (objectless) pleasure, pleasure without attachment, or worse: substitutive pleasure in the absence of a specific longed-for object (ie, object of affection). This formulation fits perfectly with the understanding of addiction outlined above. Addiction, like masturbation, is a substitute and replacement not only for general mastery of the object world, but specifically for *the attainment of a secure love object*.

This blindingly obvious insight has massive clinical relevance, today as much as ever. Moreover, the distinction between the abuse of DA-activating substances to buttress object-seeking (to increase social confidence, etc), and the abuse of opiates to actually replace the object, suggests that the latter may be a more malignant (less hopeful) form of addiction.

Despite all Freud's quaint and sometimes misleadingly idiosyncratic language, we trust that readers will agree that his conception of addiction as *a substitute for mature sexual attachment*, and therefore an equivalent of masturbation, still describes the pivot of the problem. The deeper and more detailed insights (and potentially powerful new therapeutic tools) provided by modern neuroscientific approaches to the underlying mechanisms around which the problem of addiction revolves makes it all the more imperative that we do not lose sight of this wood for the trees. The implications of this deepening understanding apply equally to new psychological and pharmacological treatment possibilities.

Conclusion

To avoid misunderstanding, we will summarise our argument as succinctly as possible. Addiction involves (1) a primary appetitive process called SEEKING, plus (2) a primary consummatory process called PLEASURE-LUST, which rewards the SEEKING activity and thereby allows learning to occur, plus (3) a primary social process called attachment, which is mediated by the PANIC-GRIEF system. Once an attachment is established, reunion with the object of attachment is the specific pleasure that the addict seeks. Our argument is that addiction researchers who apply 'incentive salience' theory conflate 1 and 2, and they overemphasise this aspect of addiction without recognising the importance of 3, which in our view is the big-ticket item. It is the big-ticket item for the simple reason that the real object of processes 1 and 2 – what they really 'want' – is 3. Many addiction researchers today seem to think that what the addict wants is a drug (DA-mediated). We, by contrast, think that what the addict really wants is to restore lost attachments (mu opioid-mediated). The SEEKING followed by PLEASURE learning-processes that these researchers prioritise are typically in the service of this particular type of 'wanting'. In other words, addicts, like masturbators, are not really looking for a sensory reward; the substance abuse is a self-soothing substitute for what they really want.

The second of the two Freud quotes cited above ends like this:

> [The success of a treatment for addiction] will only be an apparent one, so long as the physician contents himself with withdrawing the narcotic substance from his patients, without troubling about the source from which their imperative need for it springs … Whenever normal sexual life can no longer be established, we can count with certainty on the patient's relapse. (Freud, 1898:276)

This conclusion still rings true more than a century after it was first reached. We would only add 'social attachment' – primal mother–infant bonding – to Freud's use of the term 'sexual life', since Freud implicitly included almost all other rewarding aspects of loving interaction under his broad use of the word 'sexual'.

Indeed, recent animal research has indicated that maternal CARE urges reduce the brain's tendency to find cocaine attractive (eg, Ferris et al, 2005). The substitutive attachment aspects of addiction probably go a long way to explaining why 12-step programmes are among the most effective ways to break addictive cycles. They return participants into an emotionally engaged and ultimately satisfying social network, which is so patently lacking in

the lives of many addicts. For those who seek new pharmacological therapies for addiction, we might suggest that they evaluate the paradoxical prediction (by current standards) that the non-addictive opioid receptor agonist-antagonist buprenorphine may be efficacious in reducing amphetamine and cocaine addictions.

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