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EDITORIAL

Towards addiction as relationship

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Abstract
In this article, we wish to offer some thoughts on the taken-for-granted connotations of the term 'addiction', and on the limits of the disease model which these assumptions support, and by way of comparison with parallel developments in the field of 'schizophrenia' research, to suggest some avenues for conceptual development.

Keywords: *Addiction, meaning, systems, consequences, diagnosis*

Introduction

It is now a convention of critical and historical analyses of psychology to observe that lay understandings and academic understandings are mutually constitutive (e.g. see Rose, 1997). The everyday vocabulary *and experience* of our psychological world is shaped by professional vocabularies and practices (i.e. experiences and claims are made available to all of us by these vocabularies). Equally, as social scientists, we can never entirely separate our 'academic' understandings of psychological phenomena from those experiential and cultural presuppositions which we bring to the topic.

We wish to begin by commenting upon three particular levels of meaning attached to 'addiction'. At the first of these levels, the term retains pejorative

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and stigmatising connotations, which evoke moral failings, and which position 'addicts' at the margins of 'normal' society. The references to the language of 'dope fiends' and '*speed freaks*,' made by Shaffer in a 1986 paper may seem a little anachronistic to the contemporary ear, but this is merely because the vocabulary continues to evolve. Shaffer's summary of the significance of this imagery remains accurate: ''These labels euphemistically and pejoratively identify a society's counter-culture: the dependent, morally corrupt and socially impoverished.'' (Shaffer, 1986, p. 287). It is not easy to challenge such labels directly, but it ought to be possible for us to act and speak in ways which allow those people experiencing distress to take up more acceptable positions for themselves. This might mean rethinking the way that way we use the term 'addiction,' and in particular the consequences of adhering to diagnostic categories.

At the second level, 'addiction' has acquired a pathological connotation, developing as it has done through Jellinek's (1960) work, into a concept which is often understood as a 'disease.' The disease model may have represented a kind of ethical improvement on the preceding moral model (e.g. see Brickman, Rabinovitz, Karuza & Coates, 1982), but in many ways it simply situates the source of the stigma elsewhere. There is a parallel here with the field of schizophrenia research. The power of 'diagnosis' and the domain of the medical establishment appear to leave both 'addict' and 'society' with relatively few opportunities to express agency and responsibility, whether in terms of the development of the problem, or the provision of strategies and resources for supporting coping and change.

Szasz (1974) has suggested that initially addiction may have simply been *likened* to a disease, and with progressive motives. Unfortunately, simile hardened into metaphor, and then into 'fact.' The disease model pervades addictions research and practice, despite evidence that addictive behaviour is not inevitably progressive, degenerative, or incurable (e.g. Robins, Helzer & Davis, 1975); not unrelated to complex sociocultural and psychological factors (e.g. see Peele, 1990, 1996); and that addictive behaviours are often similar to other behaviours (comparable forms of expressing agency and identity through unconventional activity) which do not carry the same 'diagnosis' (e.g. see Larkin & Griffiths, 2004). Taken together, these observations ought to undermine its validity, but the disease concept has become taken-for-granted. In a recent editorial in *American Journal of Psychiatry*, for example, Kalivas begins his first sentence with the *de facto* statement that ''addiction is a disease of the brain and [...] can in part be defined as having little control over craving and relapse'' (2004, p. 193). The first part of the statement by Kalivas ('addiction is a disease of the brain') might well be considered to be the 'hard core' (after Lakatos, 1970; and Boyle, 1990) of addictions science, and we return to two of its major supporting assumptions in a moment.

The latter part of Kalivas' statement ('having little control over craving and relapse') is interesting because it leads us to the third level of meaning attached to 'addiction': ''What an addict learns in his failed attempts to quit, is that he is what his culture labels an addict. To be addicted is to be unable to

'just quit'.'' (Elster, 2000, p. 132). This is what might be considered the 'cultural hard core' of the addiction concept. That is, there is an idea about 'what it means to describe oneself as addicted' which is 'out there' in the world, and which can not easily be changed. Just as Boyle (1990) and other critics of the disease model in schizophrenia have been keen to state that they do recognise the experiences of people who hear voices, for example, or the distress of people who have persecutory beliefs, so we must point out that 'addiction' does pick up on a meaningful human experience. Some people will find that their relationship to certain appetitive activities become paradoxical, repetitive, and problematic, and are *experienced as* non-volitional (whether they *are* 'non-volitional' is another matter). In such situations, people will tend to describe themselves as 'addicted,' and obviously we wish to be able to engage with them on those terms. For us, *these* are the hard core features of what 'addiction' is, because they emerge as the common features of people's use and understanding of the term (e.g. see Larkin, 2001; Larkin & Griffiths, 2002, 2004). The moral and disease models, on the other hand, remain open to challenge: they attach consequences to having described oneself as 'addicted' which can be stigmatising, self-fulfilling and resistant to change. Functional analyses and deconstructions of this model have been offered before, of course (e.g. Davies, 1992), but the consequences have not been embraced by the wider addictions community. It is our view that more careful use of the term by professionals, and more thorough examination of our assumptions about it, are the first steps towards reducing stigma and pathologisation, and towards providing more flexible and effective support for people with problems.

In comparison to the field of 'schizophrenia' research, addictions researchers have one advantage: they have been relatively successful in developing rich accounts of *what addiction is like* (particularly in the growing use of qualitative research to understand the actions, experiences and contexts of people with addiction problems; e.g. see Taylor, 1993). This is a good start – but these kind of data present a challenge to the 'hard core' of the disease model which has not been taken up.

This problem is particularly pertinent to the very narrow definition used by biological researchers, who generally claim to be studying 'addiction' whenever they look at withdrawal, tolerance, or the neuropsychological effects of psychoactive drugs. Addiction tends to be viewed by the natural sciences as a combination of *withdrawal* and/or *tolerance*, plus *long-term health damage*. Obviously, withdrawal effects can make it much more difficult to achieve moderation and/or abstinence: but they are neither necessary nor sufficient for a description of 'addiction.' There are three reasons for this. First, addictive relationships are observed and experienced with activities and substances which do not produce particularly severe forms of withdrawal and/or tolerance. Second, addictive behaviours are often resumed long after the effects of withdrawal have dissipated, and these returns to use can often be attributed to contextual circumstances. Third, withdrawal has been reported by people experiencing non-chemical addiction problems, such as gambling (e.g. see Griffiths

& Smeaton, 2002). Long-term health damage is an important indicator of the paradoxical and problematic nature of addiction – but it is not the only one.

Animal models still prevail here, too, despite serious questions about their relevance to human behaviour (e.g. see Davies, 1992) and their misuse in distorting the importance of *context* (e.g. Alexander, Beyerstein, Hadaway & Coambs, 1981). This limited conceptualisation of addiction offers us a view of the body as a kind of simple input–output device. Yet, clinical and personal experience, and qualitative and social research, tell us that 'addiction' is a complex and multidimensional phenomenon. This is not to say that an understanding of drug effects is not important, merely that it must be situated within a biopsychosocial model which acknowledges complexity (e.g. see Larkin & Griffiths, 1998). Most importantly, neuropsychological observations must not be conflated with *experiential* effects (e.g. see Moncrieff & Cohen, 2005). We know, for example, that the same substance may be used for more than one purpose, often by the same person, and on the same day (e.g. see Brown, 1993).

To paraphrase Burglass and Shaffer (1984), the important questions are 'why some things and not others?' and 'why some people and not others?' Answers to these questions have been hindered by two common misconceptions about addiction, which to some extent have underpinned the 'hard core' disease concept. These are that addiction somehow resides within: (i) particular types of people or (ii) particular substances, and/or particular kinds of activity. That is, either some people are already 'diseased,' or else some substances/activities *cause* this disease, or both. We examine these, briefly, and in turn.

Particular types of people

There is a belief that some people are destined to become addicted. Typically this is explained in one (or both) of two ways. That some people (i.e., 'addicts') have an addictive personality, and that there is a genetic basis for addiction.

The evidence for 'addictive personality' rests to a certain extent upon one's faith in the validity of psychometric testing. Setting aside this major hurdle (e.g. see Danziger, 1990; Rose, 1996, 1997) the evidence in this area is still inconclusive and contradictory. First, psychologists have yet to determine which particular personality traits are linked to addiction. Studies have claimed that 'the addictive personality' may be characterised by an absurdly wide range of factors: sensation-seeking, novelty-seeking, and extroversion; certain arousal, emotional, or locus-of-control preferences; obsessive–compulsive disorder; the influence of major traumatic life events, learned behaviours or socialisation effects; general 'substance abuse' or 'multiple addiction' traits; and more recently, sweet-liking. The extent of this range stretches not only the notion of an 'addictive personality' (e.g. see Sutker & Allain, 1988) but also the concept of 'personality' itself. Inevitably, much of this work is correlational in design, and so the interpretation of results is not easily framed in terms of cause and effect. The conceptual difficulty here is a function of over-simplification: the hope of this strain of empiricism is that if we can only divide people up *into the right groups*,

then the explanation will emerge. It may look like a lucrative short-cut, but the diversity of answers being offered seems to suggest otherwise. Of course, the relationship between individual bodies, minds, contexts, and life histories is complex *and* important – but it requires that we approach the matter from a more sophisticated and integrative position (e.g. see Orford, 2001).

The search for a genetic basis for addiction rests upon the notion that some types of individuals are somehow 'biologically wired' to become addicts. Again, we must set aside any doubts about the limited conceptualisation of 'the environment' which often typifies this kind of research, and its combination with epidemiological designs which are largely descriptive (see Kendler, 2005 – this is a problem for the nonmolecular research in this area). In the early 1990s, McGue reviewed the first wave of this research and concluded that there was evidence for 'moderate' heritability in males, and evidence for 'only modest' heritability in females, with the *caveat* that the genetic influences appeared to be heterogeneous and polygenic (McGue, 1993). A review by Tyndale (2003), and a meta-analysis by Walters (2002), pick up where McGue left off, indicating an ''upper limit of 30–36% for the heritability of alcohol misuse'' (Walters, 2002, p. 557) and concluding that ''risk for alcoholism or nicotine dependence is likely to be the result of a large number of genes, each contributing a small fraction of the overall risk,'' (Tyndale, 2003, p. 94). Furthermore, some of these same genes appear to be risk factors for other problems, some of them conceptually unrelated to 'addiction.' The point here is that while these findings do contribute something to our understanding of 'why some people and not others,' they do not adequately or independently explain the range of variation. Therefore the most we can say is that some people are more likely to develop problems under certain conditions, and that given the right conditions *most* people could probably develop an addiction. Emphasis needs to be placed on identifying those 'conditions,' rather than on searching for the narrowest of reductionist explanations.

Particular substances or particular kinds of activity

Substances cannot be described as intrinsically addictive in themselves (unless perhaps one chooses to define 'addictive' in terms of a substance's ability to produce tolerance and/or withdrawal, and to ignore the range of human experience which is excluded by this). Biologists may be able to tell us very valuable things about the psychopharmacological nature of the rewards which particular substances offer to us, and the different kinds of neuroadaptation which they may or may not produce in order to effect tolerance and/or withdrawal. But this, on its own, is not an adequate explanation for addiction. Robins et al. (1975) classic study of heroin-users returning from the Vietnam war is one example of the evidence which refutes this second oversimplification. This study clearly demonstrated the importance of context, and the framework provided by such contexts for making sense of addiction: in a hostile and threatening environment, opiates clearly provide something not usually required by most people; and given

a cultural environment in which opiate use is a commonplace, and opiates are available, then opiate use 'makes sense'. Here is support then, for the earlier assertion that some people are more likely to become addicted under some conditions, and that given the right conditions perhaps many people could understand what it means to be an addict. Indeed, amid more recent reports of normalised and recreational heroin-use, and a more general awareness that drug-users ''may take drugs competently as well as incompetently'' (Davies, 1992, p. 163), the myth of the 'instantly addictive substance' appears to have moved on, having transferred itself to crack cocaine for the time-being (e.g. see Hausen & Krausz, 2001, for a refutation). The cultural function of such narratives is a question for another day.

So, with regard to the question, 'why some things and not others?', we have stated already that the rewards associated with various activities may be qualitatively very different, and may not necessarily be inherent or unique to a particular activity or substance, either. Many rewarding activities are rewarding because they present a person with opportunities to 'shift' one's own subjective experience of oneself (e.g. see Larkin & Griffiths, 2004). Frequently, a range of such opportunities is offered to the experienced user. Shaffer (1996) has pointed out that those activities which can be *most relied upon* to shift self-experience *in a robust manner* are likely to be the most popular – and to be the most frequent basis of problems. So, obviously, our understanding of the available resources for mood-modification must play a major part in understanding addiction: but one must make a careful distinction between describing some substances as being more 'robust shifters of experience' than others (as we would advocate) and describing some substances as 'more addictive' than others (which we would not).

Addiction as relationship

The problem implicit in the moral and pathological understandings of addiction becomes salient here. It is still a truism to observe, as Krivanek once did, that ''contrary to all logic there is no relationship between relative acceptability of a drug and its ratio of desirable and undesirable effects'' (1988, p. 38). The negative consequences of 'addiction' may include: the health risks associated with long-term or excessive use of some substances; the further risks associated with contamination of those substances; the physical dangers involved in gaining access to some substances/activities; and the legal, relational, financial and social costs to the user and their family. These consequences are the means by which people identify their own problems, and they do this regardless of the available diagnostic categories, or the legality or normative status of their actions. They provide the basis from which assessment and intervention develop.

We have been querying the viability of two of the major assumptions underpinning the 'hard core' disease model. To dispense with this model is potentially to dispense with a *top–down* diagnostic system for understanding addiction. Indeed, it might be prudent to demolish this now, before the genetic evidence does it for us (Kendler, 2005). An explicitly *bottom–up* approach, focusing upon

negative consequences as they are experienced by the person and by those people around them, provides a viable and responsive alternative. It should be the basis for developing a more flexible and needs-led system of research, theory and practice. Such a focus might be considered as analogous to the evolving preference for non-diagnostic (e.g. see Moncrieff & Cohen, 2005) and symptom (Bentall, 2004; Read, Mosher & Bentall, 2004) approaches in psychosis. It can be reconciled with the meaningful accounts of clients and their contexts, and with the complex modelling of polygenic interactions which may eventually emerge from the molecular genetic work. Indeed, *services* may already be structured in this way – but theory and research continue to be aligned by the disease model.

We remain a step away from providing a basis for integrated and eclectic interventions, which may be what is required in order to provide clients with opportunities to find out 'what works' for them. But by contrast, consider the consequences of offering support on the basis of the two main assumptions of the disease model. Attempts to put 'certain types of persons' in 'certain types of treatment' have been largely unpersuasive (e.g. PROJECT MATCH research group, 1997), and indeed, this may be a direct consequence of design generated from the 'certain types of people' assumption. Service-users' accounts (e.g. Larkin, 2002) seem to suggest a need for greater agency and choice, and for more flexible and systemic resources to support and facilitate change. PROJECT MATCH had to sacrifice these options to the requirements of a controlled design. In terms of the 'certain types of substance' argument, the 'dangers' of addiction are not usually associated with the intrinsic *rewards* of any particular activity – though they may be compounded by its illegality – so it makes little sense to aim all our therapeutic responses *at* particular activities. Some needs overlap, and some do not, of course, and in many cases services are already designed to deal with this situation – but there is little clinical ground to be gained by directing our attention towards the *properties* of a substance, *per se*. It is the *meaning* of these properties to the person which matters most.

Bodies and brains, and their engagement with activities and substances, each provide us with a set of constraints and potentials for self-experience, and for its variation. It is certainly sensible to understand the range of these constraints and potentials, insofar as they can be mapped out. However, it is culture which offers us meaningful opportunities for engagement with these activities and substances, and it is culture which furnishes us with both the motives for doing so, and the resources for making sense of our experiences when we do. 'Addiction' can only exist *in the relationship between* persons (bodies–minds), actions (activities–substances), and culture.

We have argued elsewhere that an effective approach ought to aim to integrate these factors, and to situate 'addiction' within the relationship between them, and this might be characterised as a 'complex systems' approach (see Larkin & Griffiths, 1998; Griffiths & Larkin, 2004). Here, we envisage a dynamic process, in which persons engage in a range of contextually appropriate, mood-modifying activities. The effects of these activities are partly a consequence of

the opportunities available to the person for expressing agency, and claiming identity within the system in which they live, of available patterns of action and meaning. In part, these effects will also be a consequence of the constraints and potentials offered by the person's own phenotype, and by the properties of the activity itself. That is, a number of biological, psychological and sociocultural factors will determine the nature of the process. The *durability* of this process (i.e. its resistance to change), and any other attributes which may be of interest, will depend upon the nature of the relationship between these factors.

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