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Editors' choice

Post-humanism, addiction and the loss of self-control: Reflections on the missing core in addiction science☆

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ABSTRACT

The core criterion of addiction is the loss of self control. Ironically enough, however, neither the social nor the biomedical sciences of addiction have so far made any measurable headway in linking drug use to a loss of self control. In this essay I begin by demonstrating the limitations in this regard suffered by the social and bio-medical sciences. Whereas the social sciences have variously reduced addicted drug use to deviant, but nonetheless self-governed, behaviour or discourses thereof, the bio-medical sciences have completely failed to adequately specify, let alone empirically analyse, how we might distinguish addicted from self-governed behaviour. I then show how these limitations can be very easily overcome by the adoption of a post-humanist perspective on self control and the various afflictions, including addiction, to which it is regarded heir. This argument provides occasion to acquaint readers with post-humanist scholarship concerning a spectrum of relevant topics including the human body, disease, drug use and therapeutic intervention and to show how these lines of investigation can be combined to provide an innovative, theoretically robust and practically valuable method for advancing the scientific study of addiction specifically as the loss of self control. The essay concludes with a discussion of some of the more important ramifications that follow from the adoption of this post-humanist approach for drug policy studies.

Introduction

There is a curious void at the centre of addiction science. Despite widespread acknowledgement that the core criterion of addiction is the loss of self-control (cf. Levine, 1978; O'Brien, Volkow, & Li, 2006; Reinarman, 2005; Valverde, 1998; West, 2006), nowhere has anyone succeeded in scientifically distinguishing controlled drug use from the loss of self control. Whereas the social sciences have invariably construed addiction as either deviant, but nonetheless self-governed, behaviour or discourses thereof, the biomedical sciences have failed even to adequately specify, let alone empirically analyse, how we might distinguish self-governed from addicted behaviour. This oversight stems from the overwhelming tendency to conceptualise human biology and human social life dichotomously as two, and only two, wholly discrete and independently integrated ontological domains. While mainstream addiction science allows for research that combines categories from each side of the boundary between "the social" and "the biological," it is uniformly resistant to considering this boundary as itself diversely drawn, provisional and in flux. Because post-humanism has been precisely concerned to problematise this understanding of the relationship between "the social" and its ontological others (e.g. "the natural," "the biological," "the mental," "the metaphysical"), and insists that particular configurations of the social and its ontological others are intrinsically mediated through one another, it offers an immensely valuable opportunity to overcome the strange pandemic disability one finds throughout addiction science to account for its own core phenomenon.

In the first section I demonstrate that neither the biomedical nor the social sciences have ever managed to adequately link drug use with a loss of self control. I then show how this limitation can be very easily overcome by the adoption of a post-humanist perspective on self control and the various afflictions, including addiction, to which it is regarded heir. This argument provides occasion to acquaint readers with post-humanist scholarship concerning a spectrum of relevant topics including the human body, disease, drug use and therapeutic intervention and to show how these lines of investigation can be combined to provide an innovative, theoretically robust and practically valuable method for advancing the scientific study of addiction specifically as a loss of self-control. The article concludes with a discussion of two of the more important ramifications that follow from the adoption of the proposed post-humanist approach to the study of addiction for drug policy studies. The first is a less generic and more nuanced regard for the particular constellations of challenges faced by specific policy makers,

☆ Some passages in this article have been variously adapted from Weinberg (1997, 2002).

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service providers and service users, not least of which is the various combination of factors that can influence the phenomenology of addiction as affliction. Second, precisely because it enhances our appreciation of the phenomenology of addiction as affliction, the proposed post-humanist orientation to addiction also provides for an empirically richer and more nuanced consideration of the ethics of intervention. Scholarly debate on the ethics of intervention has suffered from a too starkly dichotomous characterisation of drug use as either a freely exercised prerogative or a symptom of biological pathology. Post-humanism helps us to more exactly appreciate the continuum between freedom and affliction as well as the full range of empirical factors that influence how drug related behaviour is specifically located on this continuum in any given instance.

Where in addiction science is the loss of self-control?

A brief history of biomedical approaches to addiction

A few early modern authorities dabbled with the idea that inebriety may be a disease (cf. Levine, 1978; Porter, 1985), but sustained biomedical interest did not emerge until the middle of the nineteenth century. By the late nineteenth century there was a fairly well established two tiered medical understanding of addiction (Courtwright, 2001). Those who could afford private care were ordinarily diagnosed with the so-called disease of neurasthenia, literally nervous exhaustion, and prescribed temporary respite from the complex demands of modern life. Those who could not afford to pay were consigned to state sponsored institutions also staffed by medical doctors but designed to manage the more pessimistic diagnosis of degeneracy. Degeneracy followed either from a hereditary predisposition or a dissolute life and while it could be prevented, few medical men thought it could be reversed. Rather than seeking to return the patient to a former state of non-addiction, the medical treatment of degeneracy was focused more on limiting the havoc degenerate addicts might wreak upon their wider communities.

In these early days, reigning theories still reflected the influences of humoral medicine in prioritising attention to moderate habits and self-regulation over anatomical structure and physiological function. And while it would be unfair to blithely reduce nineteenth century addiction medicine to no more than dressed-up social prejudices, it was undeniably more deeply informed by the perceived character of the patient than the perceived character of his or her putative disease (Baumohl & Room, 1987; Courtwright, 2001). In short, insofar as addiction medicine had not yet fully distinguished medical pathology from the social marginality it was meant to explain, it had as yet no clear separation between what a Foucauldian might call the biomedical and the socio-cultural gaze. Nor, more specifically, did it provide any way of medically linking drug use with a loss of self-control. Neurasthenia cast addiction as a form of fatigue not biological dysfunction and, likewise, degeneracy yielded an understanding of addiction as atavism not affliction. Neither could empirically distinguish self-control from its loss because in neither case was anything other than the self of the supposed addict implicated as a proximal cause of his or her behaviour.

As the nineteenth century came to a close, addiction medicine entered a protracted period of doldrums. Theories of degeneracy and neurasthenia were eventually dismissed by a new generation of medical scientists and the pall of prohibitionist sentiment and then legislation both minimised the availability of funding for addiction research and dissuaded most medical professionals from entering the field. Those who did occupied two camps. The first embraced psychodynamic theories that retained a view of addicts as intrinsically inferior beings (cf. Acker, 2002). The second focused on physiological withdrawal, arguing that addiction did not belie underlying deficits like degeneracy or psychopathy but was a normal physiological response to which anyone might succumb (Campbell, 2007). Because they seemed to legitimate medical maintenance of addicts' drug supply, physiological withdrawal based theories did not enjoy much approval amongst policy makers committed to prohibition but did slowly gain sway in the medical community as psychodynamic psychiatry fell from favour. Physiological withdrawal symptoms appeared to provide a specific, universally applicable, biomedically identifiable marker by which addicts might be categorically distinguished from non-addicts. They thereby introduced an apparent path to scientific respectability insofar as the aetiology and identity of addiction could now be categorically specified in strictly biomedical terms. Those substances that produce physiological withdrawal symptoms were classed as genuinely addictive. Those that did not were categorically denied that status. However, once again, addiction science had plainly failed to link drug use with a loss of self-control. Demonstrating that a substance causes withdrawal symptoms does not indicate how these symptoms, in turn, cause a loss of self-control rather than just a change and narrowing of personal priorities.[1] Indeed, using drugs to stave off the pains of withdrawal could be seen to exhibit a perfectly reasonable cost–benefit analysis.

Other, better noticed, anomalies began to accumulate too. One can perfectly understand how someone might remain in a perpetual cycle of withdrawal symptom avoidance for as long as withdrawal symptoms actually loom. But why is it, some asked, that the many medications that ease or altogether eliminate physiological withdrawal symptoms have had such a dismal record of getting people permanently off drugs? Perhaps even more perplexing, why are those who have actually suffered the ravages of cold turkey not uniformly chastened by this experience? One would think that such a profoundly nasty ordeal might discourage people from returning to the use of physically addictive drugs. But, too often, it does no such thing. Conversely, why do so many people who become physiologically dependent seem to have so few, if any, qualms about stopping? Finally, it has grown progressively more difficult to argue that only gross physiological withdrawal symptoms[2] cause addiction. Drugs like crack cocaine or nicotine and activities like sex, gambling, and eating – none of which produce such symptoms – appear capable of inducing behavioural patterns every bit as damaging as those induced by alcohol and opiates. It is in no small part due to this accumulation of anomalies that interest turned to our most recent paradigm in biomedical addiction science (cf. Leshner, 1997, p. 46), what the historian David Courtwright (2010) dubbed the "NIDA Brain Disease Paradigm."

The brain disease paradigm is first and foremost anchored in the priority given to basic science (Campbell, 2010; Vrecko, 2010). This has largely meant confining research to basic biology conceived as a primordial, discrete and independently integrated ontological domain. Brain disease scientists argue that people ingest chemicals like heroin, cocaine, alcohol or nicotine because they biologically cause euphoria by promoting the release of neurotransmitters, preventing their re-uptake, or mimicking their effects (cf. Koob, 2006). But what of addiction? Many studies have noted after prolonged use the positive effects of drug use are often eclipsed by the negative (cf. Koob, Stinus, LeMoal, & Bloom, 1989). Some heavy users even

[1] Though one hears banter of "workaholics" and the like, in truth mere dedication to a form of activity or consumption is an extremely dubious stand in for a genuine loss of self-control. The questions of whether this dedication is to a form of relief or pleasure, and whether it is virtuous or vicious have absolutely no bearing on the question of whether it is voluntary or involuntary.

[2] By gross withdrawal symptoms I mean symptoms like vomiting, cramping, delirium tremens, runny nose, itchy eyes and so on which implicate specific physiological effects of withdrawal. This is in contrast to more diffuse effects like anxiety or headaches which are less clearly reducible to such specific physiological effects.