Lecture 5: 'Addiction: Compulsion or Choice?'

I. Recap

Last week we looked at akrasia, and some philosophical responses to the question: is action against one's better judgement possible?

We looked at Davidson's famous solution to the problem of akrasia which focuses on action against 'conditional', or prima facie, judgements, and examined how the philosophical literature post-Davidson can be framed as a debate between so-called 'internalist' solutions (which deny - some think, implausibly - the reality of action against ones unconditional evaluative judgement) and externalist solutions to the problem of akrasia (which accommodate the reality and prevalence of akrasia, but at the cost of positing a divergence between our evaluative judgements and motivation of action).

We then went on to examine Holton's analysis of weakness of the will, as distinct from akrasia, in terms of an over-ready revision of intentions, or resolutions, to act.

II. Two Models of Addiction

The Moral or 'Ordinary Choice' Model of Addiction:

1. The addict *chooses* her behaviour (i.e. it is intentional, voluntary).
2. The addict is therefore morally responsible—and thus potentially blameworthy—for the outcomes of such choices.

The Disease Model of Addiction:

> National Institute on Drug Abuse: "Addiction is defined as a chronic, relapsing brain disease that is characterized by compulsive drug seeking and use, despite harmful consequences."
>
> William James' alcoholic: Were a keg of rum in one corner of a room, and were a cannon constantly discharging balls between me and it, I could not refrain from passing before that cannon in order to get at the rum (Holton and Berridge 2013).

III. Important Preliminaries

1. There is no theory-neutral way to define addiction.

- Most medical bodies *define* addiction as compelled.
- Many advocates of the moral model deny that addiction exists.
- This has resulted in a proliferation of different 'models' of addiction which often talk past one another.

2. Addiction is *not* equivalent to repeated drug use.

- It is obvious why lots of people take lots of drugs lots of the time: improved social interaction, improved cognitive performance, coping with psychological stress, self-medication, sensory curiosity, euphoria and hedonia (Miller and Schumann 2011).

- The puzzle of addiction: to explain persistent drug use despite the recognition of severely negative consequences (Pickard 2018).

3. Drug addiction is severely stigmatised around the world.

- Common language: "junkies", "crack moms", "getting clean".
- The possession and trafficking of many psychoactive drugs are almost universally criminalised.

4. Addiction plausibly extends beyond drug use.

- "Behavioural addictions" to, e.g., gambling, videogames, pornography, sex, food, exhibit a similar behavioural and neurological profile to "substance addictions" (Lewis 2017; Brewer and Potenza 2008).

IV. The Disease Model of Addiction

1. Addiction can be *extremely* self-destructive. Explanation: because people would not *voluntarily choose* such bad outcomes for themselves, their behaviour must be compelled.

2. Dissociation between the addict's behaviour and her evaluation of its worth.

- Addicts often claim that they get no pleasure from drugs, that they want to stop, that they have lost control, etc.
- Recall Davidson's principle: if an agent engages in behaviour radically dissociated from her evaluative judgements, it *cannot* be intentional.

3. Addiction has a strong heritable component.

4. The neuroscience of addiction. Addiction changes the brain. These changes are often interpreted as: (1) neurological *dysfunction* or impairment; and (2) removing the addict's control.

1. Incentive salience theory: drugs artificially increase dopamine release, which creates pathological "wanting," i.e. intense and long-lasting desire for the drug that is (i) triggered either by the drug or drug-related cues and (ii) dissociated from pleasure ("liking") or even the expectation of pleasure (see Robinson and Berridge 2003; Holton and Berridge 2013).
    - Explains relapse after sustained abstinence. Explains the self-reports of addicts.
    - Note: strong implication that Davidson's principle is wrong: incentive salience desires are neither caused by nor responsive to an agent's evaluation of their objects.
2. (Partial) functional disconnection of dorsolateral prefrontal cortex (dlPFC) from motivational areas of the brain. dlPFC is critical for self-control, delayed gratification, etc. This leads to "impaired response inhibition" in the context of desire for drugs (Robinson and Berridge 2003).
    - Addiction partially removes the capacity for self-control in the face of the desire to consume the drug.

5. Animal studies. Rats left in an isolated cage given access both to normal water and to drug-laced water (cocaine or morphine) will escalate use of drugs, neglecting food and water, sometimes even until self-induced death (Bozarth and Wise 1985).

6. The Disease Model *avoids stigma*. "Calling addiction a disease not only mitigates massive volumes of stigma and guilt but also aims to provide accessible avenues for addicts to get help."

Revisiting the question from the perspective of the disease model:

- It's *not* the case that all that's wrong with addicts is that they do not want to stop badly enough.
- Addicts suffer from neurological dysfunction and impairment which removes their capacity to control their behaviour.
- Insofar as their behaviour is determined by their desires (what they *want*), these desires are not responsive to their reflective cost-benefit analyses and are impossible—or at least severely difficult—to control.

V. Addiction and Choice: Problems for the Disease Model

1. Does irrationality and self-destruction = compulsion = pathology?

"It seems fair to say that who cites selfishness and myopic choices as evidence of pathology (e.g. "she had to be sick because she bought drugs rather than groceries") naively misread human nature" (Heyman 2013, p.3).

2. Data against compulsion:

- Most addicts quit without any clinical intervention in their late twenties and early thirties (Heyman 2009).
- Many quit by going "cold turkey"
- Addicts are responsive to incentives (Pickard 2017; Heyman 2013).
- If we reduce the strength of our concept of compulsion, this reduces the explanatory power of compulsion in relation to the puzzle of continued use in the face of severe negative consequences (Pickard 2022).

3. Brain change does not equal brain disease (see Lewis 2017; Pickard 2018). Partial heritability of addictive traits is irrelevant.

- To deny that addiction is a brain disease, is not to deny that neuroscience can illuminate the nature of addiction - all forms of non-pathological learning are illuminated by neuroscience (Pickard 2022).
- Difference may just signal *diversity*, not disorder (Murphy 2021).
- The question here is wether or not disease is a dysfunction, and this is arguably not settled by existing work (Pickard 2022).

4. Implies that there can be *asymptomatic addiction*, and also, that the pathology present without being the *cause* of the symptoms.

5. Other factors:

- Early trauma and self-medication: "substance abuse among those with PTSD is as high as 60-80%, and the rate of PTSD among substance abusers is 40-60%" (Lewis 2015, p.3).
- Socio-economic circumstances strongly predict addiction (Pickard 2017).

6. Rat park

- When rats are placed in attractive environments, they almost always opt for plain water rather than drug-laced water; when they do take drugs, they do not do it compulsively (Alexander et al.1978; 1985).

Addiction and Choice: Some Considerations:

1. Sometimes persistent drug-use can be a reasonable or at least intelligible response to (perceived) life circumstances, e.g. reducing the psychological harm of trauma, a bad environment, terrible life prospects, etc. (Pickard 2018).

2. Addicts often discount the value of the future at a greater rate than others (Heyman 2009). If you grow up in an extremely volatile environment, this might itself be reasonable.

3. Some addicts' choices are heavily influenced by self-contempt ("I don't deserve a good life"), by their self-identity ("I am a drug addict"), by denial, by an (often understandable) inability to conceive of themselves in alternative situations, and by the difficulty of isolating their drug-use as the or a source of their problems (Pickard 2018).

Note: these are all psychological explanations that can be invoked without resorting to "there is something wrong with the brain" (see Gene Heyman's work, and especially Hannah Pickard's work for this psychological perspective). These psychological explanations may even be compatible with the idea that some addictions are pathological as the disease model describes (Pickard 2022).

Recovery and the belief in self-efficacy:

- "… the only pre-treatment characteristic that predicted relapse, six months after concluding outpatient treatments for alcohol dependence, was "the extent to which clients endorsed disease model beliefs before entering treatment"" (Lewis 2015, p.10; Miller et al 1996).

Revisiting the question from the perspective of a choice model approach:

The choice model: addicts *are* in control of their behaviour, which they choose to engage in.

BUT: this does not necessarily imply the second element of the *moral model* of addiction, because:

- Addicts' choices, beliefs, and desires often result from circumstances beyond their control (e.g. past traumas, socioeconomic conditions, mental illness, etc.).
- Therefore: in many cases, what is "wrong" cannot be attributed to the addict—or solely to the addict—but also *society* (Pickard 2017).

VI. Issues and Questions

1. Does the disease model require the addictive behaviour is literally involuntary? Couldn't neurological dysfunction just make control *extremely difficult*?
2. Might there be degrees of compulsion?
3. Can't evidence of neurological dysfunction co-exist with evidence of the role of choice and agency?
4. Do choice and compulsion provide the right categories for approaching addiction in the first place?

5. Even if addiction *does* involve neurological dysfunction, might it be *useful* for addicts, or *society*, to believe otherwise? (See the first section of Pickard 2021 for a discussion of the mixed data on this).

6. Is the idea that addiction is a single kind of thing a good starting point for the science of addiction, or might we make more progress by viewing addiction as *heterogenous* (Pickard 2022)?

Addiction often results in extremely harmful consequences—both for the addict, and sometimes for those around her. Where should we place blame?

1. The drug?
2. A brain disease?
3. The addict?
4. Society?
5. All of these?
6. None of these?

Important References

Richard, H. Berridge, K. (2013) "Addiction between compulsion and choice." Addiction and Self-Control: Perspectives from Philosophy, Psychology, and Neuroscience», Oxford University Press, New York: 239-268. [good on the neuroscience of addiction].

Lewis, M. (2017) "Addiction and the brain: development, not disease." Neuroethics. 10: 7-18. [good on the neuroscience of addiction]

Robinson TE, Berridge KC. (2003) Addiction. Annual Review of Psychology. 54(1):25–53. [good on the neuroscience of addiction].

Pickard, H. (2017) "Responsibility without blame for addiction." Neuroethics 10.1. 169-180.

Pickard, H (2018): 'The Puzzle of Addiction".

Pickard, H. (2022): 'Is addiction a brain disease? A Plea for agnosticism and heterogenity'. Psychopharmacology.

Heyman, G. (2013) Addiction and choice: theory and new data. *Frontiers in Psychiatry*.

Heyman, G. M. (2009). Addiction: A Disorder of Choice. Cambridge, MA, Harvard University Press.

National Institute on Drug Abuse, "The Science of Drug Abuse and Addiction: The Basics," September 2014, www.drugabuse.gov/publications/media-guide/ science-drug-abuse-addiction.

Murphy, Dominic, "Concepts of Disease and Health", The Stanford Encyclopedia of Philosophy (Spring 2021 Edition), Edward N. Zalta (ed.), URL = <https://plato.stanford.edu/archives/spr2021/entries/health-disease/ >Ducci F, Goldman D. (2012)

The Genetic Basis of Addictive Disorders. The Psychiatric clinics of North America. 35(2):495-519. doi:10.1016/j.psc.2012.03.010.

Bozarth, M. A., and Wise, R. A. (1985) "Toxicity associated with long-term intravenous heroin and cocaine self-administration in the rat," JAMA 254: 81-83.