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Denial in Addiction

Hanna Pickard

Abstract: I argue that denial plays a central but insufficiently recognized role in addiction. The puzzle inherent in addiction is why drug use persists despite negative consequences. The orthodox conception of addiction resolves this puzzle by appeal to compulsion; but there is increasing evidence that addicts are not compelled to use but retain choice and control over their consumption in many circumstances. I argue that denial offers an alternative explanation: there is no puzzle as to why drug use persists despite negative consequences if these consequences are not straightforwardly known. I describe the nature of the causal knowledge that one's drug use is causing negative consequences; map the conceptual landscape of denial and explain how it can block such knowledge; and explore some of the processes and mechanisms that have been studied by philosophy and the cognitive sciences and which may underpin denial in addiction, including well-established information-processing biases, motivational influences on belief formation and self-deception, and cognitive deficits with respect to insight and self-awareness. I conclude by suggesting that addiction is as much a disorder of cognition as a disorder of conation.

Keywords: Addiction, choice, compulsion, cognition, denial, motivated belief, self-deception

The orthodox conception of addiction is a chronic, relapsing neurobiological disease characterised by compulsive drug use despite negative consequences.¹ This conception is endorsed by the medical establishment, including the American National Institute of Alcohol Abuse and Alcoholism, The American National Institute of Drug Abuse, The American Medical Association, the British Medical Association, and the World Health Organization; as well as reflected in the clinical description and diagnostic criteria in the Diagnostic and Statistical Manual of Mental Disorders Fifth Edition and the International Classification of Diseases and Health Problems. It is also widely shared outside of it.

¹ I include alcohol, as well as all common illicit and pharmaceutical drugs of abuse, in the reference of the term 'drugs'. There are evident similarities but also differences between drug and behavioural addictions; I leave open the extent to which both the orthodox conception of addiction and the alternative conception developed below apply equally to behavioural addictions. For discussion, see Foddy forthcoming.

Philosophical and popular portrayals of addicts alike typically give expression to the orthodoxy, by casting addicts as subject to irresistible desires, over which they have literally no control.² In addition, neurobiology is often claimed to lend support to it. Our increasing knowledge of the acute and chronic effects of drugs on the brain, especially in relation to aberrant learning and reward, offers an understanding of some of the mechanisms that may underpin addictive behaviour, potentially explaining both the phenomenology of cravings and the notorious strength of the motivation to use drugs.³

Yet, there is a striking omission from this orthodox conception and the surrounding depiction of addicts in both the philosophical and scientific literature. *Denial* is a prominent and notorious feature of addiction. Indeed, the first step of *Alcoholics Anonymous* is to admit that you have a problem, in the face of a form of denial that is on the one hand common, yet on the other so extreme, that it is described as nothing less than *insanity* (Alcoholics Anonymous 2001, 37). What then is the role of denial in the orthodox conception of addiction and corresponding portrayals of addicts? Arguably, there is none.⁴ Addiction is characterised as a disorder of *compulsion*, with limited if any recognition of the fact that, in many instances, it may be as much if not more a disorder of *cognition*.⁵

² William James famously describes addicts thus: ‘The craving for a drink in real dipsomaniacs, or for opium or chloral in those subjugated, is of a strength of which normal persons can form no conception. “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 the rum”; “If a bottle of brandy stood at one hand and the pit of hell yawned at the other, and I were convinced that I should be pushed in as sure as I took one glass, I could not refrain”: such statements abound in dipsomaniacs’ mouths’ (1890, 543). Harry Frankfurt’s depiction of addiction is a more contemporary rendition of the same idea: addiction is a ‘physiological condition’ that means a person ‘inevitably succumbs’ to the desire to use, which is ‘too powerful . . . to withstand’ and results in that person potentially being ‘helplessly violated by [their] own desires’ (2003 [1971], 328).

³ The scientific literature is vast, but for some helpful reviews of at least some of the relevant findings, see Robinson and Berridge 2008; Hyman 2005; Koob and Le Moal 1997; Koob and Volkow 2010; Montague et al. 2004; Redish et al. 2008. Louis Charland is a philosopher who endorses the orthodox conception and explicitly links neurobiology with irresistibility of desire with respect to heroin addiction: ‘the compulsive drug-taking that defines [heroin] addiction is a direct physiological consequence of dramatic neuroadaptations produced in the reward pathways of the brain’ (Charland 2002, 40–41). For this reason, according to Charland, ‘decisions that relate to heroin use are susceptible to powerful physiological and psychological compulsions that usually nullify any semblance of voluntary choice’ (Charland 2002, 41).

⁴ But note that the final diagnostic criterion out of the polythetic set used by both the DSM-5 (APA 2013) and the ICD-10 (WHO 1992) is persisting in using despite either knowledge of the negative consequences of use (DSM-5 criterion for substance use disorder); or clear evidence of negative consequences of use that the addict is, or could be expected to be, aware of (ICD-10 criterion for syndrome dependence).

⁵ Exceptions include Rita Goldstein and her colleagues, who have repeatedly characterised addiction as a cognitive disorder (Goldstein et al. 2009; see Section 4). In addition, although different from a focus on denial, research into decision-making in addiction, including temporal discounting and judgment-shift models, also implicitly acknowledges a cognitive dimension to addiction (temporal discounting is briefly discussed in Section 1; for discussion of addiction and judgment-shifts see Levy forthcoming; for a review of decision-making in addiction, see Redish et al. 2008). Within the philosophical literature, both Crowther forthcoming and Segal 2013 contain discussions of denial.

My aim in this article is to argue that denial has a central role in addiction and ought to be squarely on the map of addiction research. Its place on this map is not only important to clinical purposes, such as diagnosis and, perhaps especially, effective treatment. It also suggests that inter-disciplinary work in philosophy and the cognitive sciences may have something distinctive to offer our understanding of addiction. Denial is a form of motivated belief or self-deception, or perhaps even, as suggested by Alcoholics Anonymous, insanity or outright delusion.⁶ It is therefore possible that, just as our increasing neurobiological knowledge promises insight into the conative impairment characteristic of addiction, our increasing philosophical and psychological knowledge of human irrationality promises insight into the cognitive impairment characteristic of addiction.

I develop this idea in four parts. First, I describe how the orthodox conception of addiction explains a puzzle inherent in addiction, in such a way as to obviate a role for denial. However, the evidence against the orthodox conception is ever-increasing, suggesting that drug consumption is not straightforwardly compulsive but responsive to incentives and so involves choice. I briefly detail this evidence, and then indicate how an appeal to denial can resolve the puzzle of addiction apart from the orthodox conception. Second, I describe the kind of causal knowledge required to know that one's drug use is causing negative consequences and so that one has a drug problem, and why it may be difficult to achieve and hence particularly prone to denial. Third, I describe our folk psychological conception of denial, which has been influenced by elements of psychodynamic theory and clinical practice, and map some of the conceptual landscape, noting the different kinds, means, degrees, and objects of denial that we distinguish within, broadly speaking, our personal-level psychological perspective. In passing, I point out a striking similarity between first-person reports of denial from addicts and first-person reports of denial from people with eating disorders. I end this section by pinpointing more precisely the role of denial in addiction. Fourth and finally, I sketch some of the processes and mechanisms that have been studied by philosophy and the cognitive sciences and which may underpin denial in addiction, including well-established information-processing biases, motivational influences on belief formation and self-deception, and cognitive deficits with respect to insight and self-awareness, linking these where possible to the personal-level psychological perspective articulated in Section 3, and making some schematic suggestions for directions for future research. I conclude by suggesting that we need to move beyond the orthodox conception, and reckon not only with the evidence that drug consumption involves choice, but also with the cognitive distortions in addiction that impact on that choice.

1. The orthodox conception

⁶ For discussion of some of the connections between motivated belief, self-deception, and delusion, see Bayne and Fernandez 2009a.

The prevalence and power of the orthodox conception is no doubt due to a host of varied and complex factors, including historical and socio-political forces.⁷ But it can also in part be explained by its capacity to resolve a striking puzzle inherent in addiction.

Addiction, as characterised by the orthodox conception and of course as is widely known, typically has multiple, and severe, negative consequences. It can destroy people's bodies, causing disease and death. It can damage people's brains, causing cognitive impairment. It can both cause and exacerbate mental health problems, such as depression, anxiety, and psychosis. It ruins relationships. People lose their families and friends, their jobs and social standing, their sense of having a community to which they belong. It can be a source of terrible shame, self-hatred, and low self-worth. Yet common sense suggests that, if people know that an action will bring about negative consequences, and they are able to avoid it, then they do. We act, so far as we can, in our own best interests and the interests of others we care for. This is a basic folk psychological rule of thumb for explaining and predicting human action. But this is what addicts seem not to do. Although addiction has extreme negative consequences, addicts continue to use drugs. This is the puzzle inherent in addiction.

Understanding addiction as a neurobiological disease of compulsion serves to resolve this puzzle. The explanation is that addiction 'hijacks' the brain, to use a common metaphor, so that addicts lose all control over their consumption and cannot help taking drugs, despite the consequences and against their best interests. As Carl Elliott describes it: 'the addiction holds the leash' (2002, 48). Hence the puzzle of why addicts use drugs despite negative consequences can be straightforwardly explained: if addicts could avoid using drugs, they would – but they can't, so they don't.

However, despite its capacity to resolve the puzzle of persistent use in the face of negative consequences, the orthodox conception of addiction as a disorder of compulsion is increasingly untenable. There is mounting evidence that, however hard it is for addicts to control their use, and however important it is for others to recognize and respect this struggle, addicts are not in fact compelled to use but have choice over their consumption in many circumstances.

To briefly review some of the evidence. Anecdotal and first-person reports abound of addicts (including those with a DSM-based diagnosis of dependence) going 'cold turkey'.⁸ Large-scale epidemiological studies demonstrate that the majority of addicts 'mature out' without clinical intervention in their late twenties and early thirties, as the responsibilities and opportunities of adulthood, such as parenthood and employment, increase (for a review of the empirical data see

⁷ For discussion of some of these forces, see Heyman 2009; Satel and Lillienfeld 2014.

⁸ For some first-person reports and discussion thereof, see Heyman 2009 and 2013a.

Heyman 2009; cf. Foddy and Savulescu 2006; Peele 1985; Pickard 2012). Rates of use are cost-sensitive: indeed, some addicts choose to undergo withdrawal in order to decrease tolerance, thereby reducing the cost of future use (Ainslie 2000). There is increasing evidence that Contingency Management treatment improves abstinence and treatment-compliance, compared to standard forms of treatment such as counselling and cognitive-behavioural therapy, by offering a reward structure of alternative goods, such as modest monetary incentives and small prizes, on condition that addicts produce clean urine samples (for a review see Petry et al. 2011). Experimental studies show that, when offered a choice between taking drugs or receiving money then and there in the laboratory setting, addicts will frequently choose money over drugs (Hart et al. 2000; Hart 2013). Finally, since Bruce Alexander's seminal experiment 'Rat Park' first intimated that something similar might be true of rats (Alexander et al. 1978, 1985), animal research on addiction has convincingly demonstrated that, although the majority of cocaine-addicted rats will escalate self-administration, sometimes to the point of death, if offered no alternative goods, they will forego cocaine and choose alternative goods, such as saccharin or same-sex snuggling, if these are available (Ahmed 2010; Zernig et al. 2013; for discussion of some of the more striking features of this research, see Pickard and Ahmed forthcoming). Broadly speaking, the evidence converges to indicate strongly that addicts are able to control their consumption and choose not to use drugs in many circumstances when there is sufficient reason for them to do so. In other words, drug use is not straightforwardly compulsive but rather responsive to incentives.⁹

However, this challenge to the orthodox conception carries an explanatory burden, namely, to offer an alternative resolution to the puzzle. If addicts are indeed able to control their consumption and abstain from drug use when there is sufficient reason for them to do so, why then do they routinely choose instead to continue to use despite negative consequences? Broadly speaking, there are three possible answers to this question that can be extracted from mainstream addiction research.¹⁰

The first draws attention to the fact that the basic folk psychological rule of thumb for explaining and predicting human action that motivates the puzzle – namely, that people act, so far as they can, in their own best interests and the interests of others they care for – is indeed only a rule of thumb. Human psychology also has a decidedly self-destructive streak. In particular, addiction is associated with anxiety, mood, and personality disorders, alongside a vast range of complex social and mental

⁹ For empirically-informed philosophical discussions of how to balance compulsion and choice in a theory of addiction, see Henden et al. 2013; Holton and Berridge 2013. For discussion of addiction in relation to free will, see Pickard forthcoming.

¹⁰ For fuller discussion of these answers, see Pickard and Ahmed forthcoming.

health needs characteristic of psycho-socio-economic adversity and isolation.¹¹ People facing these problems may exhibit patterns of deliberate self-harm and recklessness, and experience high levels of shame and self-hatred, alongside low self-worth. In other words, negative consequences only offer an incentive not to use drugs *if* you value and care about yourself.

The second answer notes that in addition, for people in such circumstances, drugs may offer relief from life's miseries. The 'self-medication' hypothesis has long been a staple of clinical understanding of drug use (Khantzian 1985, 1997; cf. Muller and Schumann 2011 and Pickard 2012). It is common knowledge that drugs offer relief from psychological distress: we 'reach for the bottle' or 'drown our sorrows' when in need. For many chronic addicts, drugs may provide a habitual and, in the short-term, effective way of managing psychological distress, caused by negative emotions alongside other symptoms and problems (which, for addicts, will include the state of unsatisfied drug cravings) typically experienced by people with mental health problems living in impoverished circumstances.

Finally, the third answer builds on the second, by recognizing the temporal dynamics of decision-making in relation to drug consumption. The benefits of drug use may be clear and immediate, while the costs are typically delayed and less certain.¹² The disposition to discount the future relative to the present is a common feature of human psychology, standardly considered rational to the extent that, adjusting for the relative value of the rewards, the present reward is certain while the future reward is uncertain. But, in addition, human discount curves are typically hyperbolic, so that as a reward nears in time, its value increases sharply, creating shifts in preferences over time in response to availability (Ainslie 2001; cf. Heyman 2009). Addicts have steeply hyperbolic discount rates compared to the norm (Bickel and Marsch 2001; Bickel et al. 2014). It is an open question whether such a narrowing of temporal horizon is pathological or rather in part rational, taking into account the life circumstances and options realistically available to many addicts.¹³ But, either way, discounting models can explain why use persists: future consequences provide incentive not to use only *if* they are represented as outweighing present benefits in addictive decision-making.

¹¹ For discussion, see Alexander 2008; Compton et al. 2007; Maté 2009; Regier et al. 1990; Pickard 2012; Pickard and Pearce 2013.

¹² It is an important and often neglected point that drugs are instrumental means to many valuable ends – consumption, as stated above, has evident benefits. Muller and Schumann 2011 delineate the following well-established functions for non-addictive consumption: (1) Improved social interaction; (2) Facilitated mating and sexual behaviour; (3) Improved cognitive performance; (4) Facilitated recovery and coping with psychological stress; (5) Self-medication for mental problems; (6) Sensory curiosity – expanded experiential horizon; and finally, (7) Euphoria and hedonia. Many of these benefits continue to be secured by drug use even when consumption has escalated and the diagnostic criteria for substance use disorder (DSM-5) or syndrome dependence (ICD-10) are met.

¹³ For discussion, see Heyman 2013b; Levy 2006; Pickard forthcoming.

These three answers from mainstream addiction research can help to dispel the puzzle. But there is clearly another possible answer to the question of why addicts persist in using despite negative consequences which should, I hope, be apparent. The folk psychological rule of thumb that people act in their own best interests and the interests of others they care for presumes that the likely consequences of their actions are known.¹⁴ We can only be guided in our decision-making by the outcomes of actions which we can foresee. If addicts do not know that their drug use has negative consequences, then there is no puzzle as to why they continue to use in the face of them. Similarly, if addicts *are in denial* that their drug use has negative consequences, then again there is no puzzle as to why they continue to use in the face of them, as denial blocks straightforward attributions of knowledge (see Section 3 and 4).¹⁵ Given the strength of the evidence challenging the orthodox conception, there is a need to resolve the puzzle without appeal to compulsion. The three answers sketched above are part of the answer. But denial is an additional piece of the puzzle. Hence the need to ensure its place on the map of addiction research.

2. Knowledge of the negative consequences of drug use

To make vivid the point that we can only be guided in our decision-making by the outcomes of actions of which we can foresee, consider first the example of nicotine addiction during the period of US history prior to the discovery that smoking causes disease. During this period, some people may have guessed, but nobody knew, that cigarettes were bad for you. Indeed, some people may have believed that cigarettes were good for you, due to extremely successful albeit misleading advertising campaigns and government lobbying by the industry (Kruger 1996). Of course, during this period, smoking in fact caused disease. But, given that many people enjoyed smoking, there is no puzzle as to why they did it – whether they were addicted to cigarettes or not – despite these negative health consequences *as they did not know about them*.

How then do people learn that their drug use has negative consequences? One way or another, they have to *discover* that it does. The fact that one's drug use is causing negative consequences is not immediately manifest through introspection, but requires acquiring *causal knowledge*.

¹⁴ I make this and related points in relation to knowledge, but one might wonder whether belief would suffice. In some cases, it might; but in other cases the fact that it is knowledge is likely to matter, and in addition it is more colloquial to speak of knowledge as opposed to belief.

¹⁵ Note for clarity that temporal discounting of future negative consequences is not the same as a failure to know the negative consequences of use. On the one hand, some negative consequences may reside in the present; on the other, future costs that are discounted relative to present benefits are nonetheless represented in decision-making processes – the point is that they are not weighed as they would be if they were not at a temporal remove in comparison with benefits. To put it more colloquially, temporal discounting does not standardly involve failing to know, but rather failing to care about, future negative consequences. That said, denial is an umbrella term (see Section 3 and 4), and it is possible that some forms may sometimes involve temporal discounting.

There are at least two kinds of causal knowledge relevant to addiction, typically acquired by two corresponding routes. On the one hand, there are large-scale generalizations, such as the knowledge that smoking causes disease. Acquiring knowledge of large-scale generalizations typically depends on equally large-scale collective research efforts involving data collection and hypothesis testing and confirmation. For example, the causal link between smoking and disease was established by extensive longitudinal comparisons of smoking versus non-smoking populations, and confirmatory evidence from animal models. Once such large-scale generalizations are known in the research community, they can become known to the public at large via the dissemination of expert testimony through channels such as the media and public education initiatives. Crucially, as individuals, we do not have the ability to acquire knowledge of these large-scale generalizations on our own, simply by considering our own experience, but must rely on scientific discovery and its dissemination. But once disseminated, this knowledge is available for use in individual decision-making. The large-scale generalization that smoking causes disease allows individuals to infer that, if they smoke heavily, then they are at risk of smoking-related disease.

On the other hand, there are small-scale individual generalizations, pertaining especially to our own individual actions and their outcomes. We can often acquire this knowledge on the basis of our own experience alone. If we observe an association between two events – such as an action of ours and an outcome – we can test the possibility of a causal relation, by intervening and manipulating the hypothesized cause while monitoring the effect of doing so. For example, although we cannot discover that smoking causes disease on our own, we can potentially discover that, in our own case, smoking causes headache. We can do this by first noticing the association and then testing the hypothesis by controlling our actions: smoke a cigarette, then observe the effects; don't smoke, then observe the effects. Once this causal knowledge is acquired, it can be used in individual decision-making, allowing us to achieve outcomes by means of interventions such as our own actions. So, armed with the knowledge that in one's own case, smoking causes headache, one can choose not to smoke, to avoid headache.

It is extremely important to recognize that causal knowledge of the negative consequences of drug use can be difficult to acquire. With respect to large-scale generalizations such as health risks, individuals are not only dependent on scientific discovery and dissemination, but must also be willing to trust the testimony available. In addition, large-scale generalizations are typically probabilistic, and so require individuals to understand and assess risk, as well as to overcome any tendency to think of themselves as the exception to the rule (see Sections 3 and 4). With respect to small-scale individual generalizations, one's experience may not offer clear confirmation. Given that

the causal network of relations is likely complicated and thickly interwoven, and drugs may well be contributory as opposed to single causes, interventions and manipulations may not yield knowledge. Suppose, for example, that you are an addict who opts not to use drugs on some occasion: you refrain from use. That is unlikely to mean that your problems, including those which may have been initially caused or exacerbated by drugs, disappear. For instance, the damage to your body is unlikely to be immediately reversed; the damage to your relationships is unlikely to immediately heal. Indeed, things may get worse before they get better as, at least in the short to medium term, life without drugs may be more of a struggle and contain more suffering than life with them. So an intervention – foregoing drugs now or in the short to medium term – may not produce the effect – the disappearance of negative consequences of use – which would support the acquisition of knowledge of a causal relationship between them. To take a well-worn example (cf. Segal 2013): if a person's drinking is contributing to ruining their marriage, but they are drinking in part because they are unhappy in their marriage, then stopping drinking will neither immediately fix the marriage nor correspondingly offer clear evidence that drinking is a cause of the problem.

Acquiring causal knowledge of the negative consequences of drug use must therefore be seen as an *achievement*. It can be hard to come by, and the evidence supporting it may be equivocal and even in some cases open to reasonable doubt. Perhaps it is no surprise, then, if it also is a form of knowledge particularly subject to denial.

3. The landscape of denial

Our folk psychological conception of denial has been influenced by elements of psychodynamic theory and clinical practice, where it is considered a core psychological defence mechanism.¹⁶ Psychological defence mechanisms are typically understood, not only to perform the function of protecting a person from experiencing psychological pain and distress, such as is commonly associated with negative emotions and critical self-evaluations, but to do so by design. Within our psychodynamically-influenced folk psychological perspective, denial is commonly understood as a refusal to acknowledge the reality of one's situation, when doing so would cause such psychological pain and distress. It therefore protects a person against this negative experience by 'denying' the reality that would otherwise cause it. As such, denial appears to be a form of motivated belief or self-deception. When psychological pain and distress would result from reckoning with the reality of

¹⁶ Classic discussions of psychological defence mechanisms include Sigmund Freud 1894 and 1896, and Anna Freud 1937. There is no agreed taxonomy, but an indicative although by no means exhaustive list includes: denial, repression, regression, dissociation, projection, introjection, reaction formation, compartmentalization, displacement, sublimation, intellectualization, and rationalization. For discussion of how to retain the insights of psychodynamic theory while rejecting its more outlandish elements, see McKay and Dennett 2009; McKay, Langdon, and Coltheart 2009.

one's situation, aversion to such pain and distress may motivate denial. To bring a little more precision to this common understanding of denial, we can gloss it as a failure to believe the truth of a proposition because doing so would cause psychological pain and distress, and despite evidence in its favour that would ordinarily suffice for its acceptance.

This gloss requires immediate clarification and qualification. First, the term 'ordinarily' can be interpreted in different ways. One possibility is to understand it as invoking something like an *impartial observer test*, similar to that suggested by Al Mele in his analysis of self-deception (2009, 60). Adapted to denial, Mele's basic idea is that, if we make the evidence in favour of the proposition denied available to a group of the denier's *impartial cognitive peers*, then those who would come to believe the proposition would significantly outnumber those who would come to disbelieve it.¹⁷ 'Impartial' in this context will need to be understood so as to exclude not only those peers who would experience pain and distress if they believed the truth of the proposition, but also those who, for any reason, have a preference that it be false. 'Cognitive peers' will need to be understood in a way which not only takes intelligence levels and reasoning capabilities and tendencies into consideration, but is also culturally and contextually sensitive, as evidence that in one culture or context suffices for the acceptance of the truth of a proposition may not do so in another. Broadly speaking, then, we could understand 'ordinarily' to indicate a mismatch in belief between an individual and their (qualified) community, holding the evidence constant. However, an alternative possibility would be to understand 'ordinarily' as invoking a comparison, not to impartial cognitive peers, but to what the person in question would believe based on the available evidence *were it not the case that believing the truth of the proposition would cause them psychological pain and distress*. Both interpretations have their problems and virtues; no doubt there are also other options.

Second, what counts as a 'failure to believe' admits of significant variation. Consider the proposition that an alcoholic's drinking is causing cirrhosis of the liver. Failing to believe this proposition may consist in explicitly rejecting it in favour of propositions that conflict with it. For instance, the alcoholic may believe that nothing is in fact wrong with their liver. Or, they may accept that something is wrong with their liver, but deny that the problem is serious and/or caused by excessive alcohol consumption. Or, finally, they might adopt a sceptical stance, refusing to commit to it not being the case that they have cirrhosis caused by excessive alcohol consumption, and instead withholding judgment either way, taking the view that the evidence is as yet inconclusive.

¹⁷ Mele suggests a further condition, namely, that the impartial cognitive peers must give at least as much consideration to the evidence as does the person in question. However, as denial may proceed via a refusal to consider the evidence at all (see below), I do not include this further condition as part of an interpretation of 'ordinarily' as, in such cases, the impartial cognitive peers could fail to accept the proposition in question and concur with the person in denial.

Psychological processes such as minimization, rationalization, and shifting standards of evidence, often underwrite this variety of denial, where a proposition for which there is evidence that would ordinarily suffice for its acceptance is outright rejected while a conflicting proposition is accepted, or, at least, judgment is suspended.

On the other hand, a ‘failure to believe’ may consist less in outright rejection of a belief, and more in an ongoing process of failing to consider the proposition or the evidence that supports it at all. Rather than explicitly dispute the proposition in question, one simply does everything possible to avoid it. Psychological processes such as distraction, forgetfulness, and repression, may serve this variety of denial, where the proposition that would cause psychological pain and distress were it accepted is dispensed with through cognitive omission, rather than commission.

Third, as should be apparent, the psychological processes involved in a failure to believe may or may not be conscious processes. Minimization and rationalization are typically conscious processes. For instance, an addict might minimize the damage to their liver by reasoning that they’ve known other addicts whose symptoms were worse and who continued drinking for years, so it cannot be that their situation is really so serious. Forgetfulness and repression, on the other hand, are not conscious processes; while shifting the standards of evidence for acceptance of a proposition in relation to its capacity for causing psychological pain and distress may or may not be.

Fourth, people may have more or less insight into the fact that they are in denial. For example, an addict who is in general prone to minimization may recognize this about themselves as well as even recognizing – in some sense or at some level – when actively engaging in minimization that that is what they are doing. On the other hand, there are clearly occasions when addicts have no insight whatsoever into the fact that they are in denial. Alcoholics Anonymous describes how abstinent alcoholics may suddenly experience a sort of ‘mental blank’ (2001, 42) such that they momentarily seem to lack conscious access to any knowledge they have about their alcoholism and its negative consequences before the first drink of a relapse:

‘As I crossed the threshold of the dining room, the thought came to my mind that it would be nice to have a couple of cocktails with dinner. That was all. Nothing more. I ordered a cocktail and my meal. Then I ordered another cocktail.... As soon as I regained my ability to think, I went carefully over that evening... Not only had I been off my guard, I had made no fight whatever against the first drinks.

This time I had not thought of the consequences at all. I had commenced to drink as carelessly as though the cocktails were ginger ale’ (Alcoholics Anonymous 2001, 41, italics in original).¹⁸

Fifth, denial can be directed at different kinds of propositions. Within Alcoholics Anonymous and other 12-step programmes, denial is often taken to be directed at the quite general proposition that a person has a drinking or drug use problem, typically understood as the claim that they are powerless to control the extent of their consumption.¹⁹ However, denial is equally often directed at more specific propositions about the causal impact of drug use in a person’s life. This causal impact is central to a diagnosis of addiction.²⁰ Given the fact that drug use confers many benefits (Muller and Schumann 2011; cf. footnote 12) and that there is no sharp line between heavy use and addiction, the negative consequences of drug use are crucial to establishing that there is indeed a problem – that the costs of use have come to outweigh the benefits. Facts about the causal impact of drug use in a person’s life therefore provide evidence of a drug problem and in principle a reason to desist from consumption – if indeed they are believed rather than denied.

To illustrate, consider the following two first-person reports:

‘[I] drank 2 ½ weeks after an esophageal hemorrhage. Couldn’t bring that into my mind with sufficient force because I had already convinced myself that scar tissue is stronger than normal tissue (they cauterized the wound). Even brought photos home they took with the endoscope to remind myself. I now had a super esophagus and was good to go, so to speak’ (quoted in Segal (2013, 306) who reports the conversation took place after this person’s doctor told him that if he were to continue to drink, he would die).

‘When I was about 8 I developed a keen desire to take drugs. This was in the 1960s, so drugs were all over the media, movies, news reports etc. I started on solvents aged about 11, moved on to various drugs at 14, took them until 20, when I switched to alcohol. I then drank too much more or less every day. I did not until the age of 42 admit that I might be taking any serious risks at all with my health. [But] my denial set in before I had even tried a drug or a drink. Rock stars would appear on

¹⁸ For a philosophical discussion of this feature of AA’s understanding of alcoholism and relapse, see Crowther forthcoming.

¹⁹ Note that, if the evidence adduced above suggesting that addicts can control their consumption is credible, then this proposition, as typically understood in AA and 12-step programmes, is in fact inaccurate. Alcoholics Anonymous advocates total abstinence as opposed to any attempt at moderate drinking or drug use. There is evidence that in some cases of severe alcoholism, this recommendation is not as effective as other forms of treatment, such as a more tolerant attitude towards moderate drinking coupled with the indefinite administration of naltrexone (Sinclair 2001). For a critical discussion of the AA model, see Dodes and Dodes 2014; Glaser 2013; for a sympathetic discussion, see Segal forthcoming.

²⁰ For discussion, see Levy 2013; Pickard and Ahmed forthcoming; Stephens and Graham 2009; for a dissenting view, see Martin et al. 2014.

TV and say “Don’t do this! I tried it and it wrecked my life” sort of thing. Didn’t bother me. Yet I was not a particularly foolhardy kid. I didn’t take abnormal risks in other areas. But I flew straight into drugs, and into hard drugs. Not shooting up, but anything short of that, cocaine and speed at 15 years of age, without a thought of risk. So what was going on in me at 8, 9 years of age? There may have been a very strong desire to escape reality ... which motivated both taking drugs to escape and denial itself ... denial being an escape from reality’ (an anonymous addict, personal communication).

Part of what is striking about these reports is that the denial appears two-pronged. On the one hand, there is denial of the large-scale generalizations that underpin the proposition that consumption carries health risks and other problems. The first addict convinces himself that scar tissue is stronger than normal tissue; the second addict is not bothered by testimony by others that drugs wrecked their lives. On the other hand, there is denial that, even if such large-scale generalizations are in fact true, they apply in the individual case and so are relevant to individual decision-making. The first addict has a ‘super’ esophagus and so is now even better than normal; the second proceeds without a thought of risk. It is as if these addicts reason and act recklessly based on a sort of belief in their own personal exceptionalism (see Section 4), invulnerability, or indeed omnipotence – almost akin to a grandiose delusion. Whatever the risks may be for others, they do not apply in their own case.

Interestingly, this kind of reasoning is not unique to addiction. It is also found in people with eating disorders. To illustrate, consider the following three first-person reports (from Hope et al. 2013, 29):

‘Everyone was telling me “you could die any second,” I mean vomiting is very dangerous at a low weight. I didn’t believe them, I wasn’t gonna die, it wouldn’t happen to me.’

‘I would have rather DIED than have to go through stopping [calorie restricting] because I’d become so fixated on ... that way of living I couldn’t see a way out and I wouldn’t have done it, even though you know I could have died any second I didn’t believe that, I would have carried on until I did die.’

‘I was getting a little bit worried but not much, I mean to me it [the physical danger] wasn’t really important ... I was just too wrapped up in what I was doing at the moment, it just didn’t mean much!’

The similarities in these various first-person reports from people suffering from addiction and people suffering from eating disorders are striking, not only in their denial of or indifference to the risk of death, but in their expression of a belief in personal exceptionalism or invulnerability in relation to

drug use and low weight respectively.²¹ Although our current understanding of eating disorders is relatively limited in comparison to addiction, eating disorders are like addiction in that the relevant behaviour is highly motivated and habitual, an object of psychological fixation and reward. DSM-5 describes them thus: ‘Feeding and eating disorders are characterized by a persistent disturbance of eating or eating-related behaviour that results in the altered consumption or absorption of food and that significantly impairs physical health or psychosocial functioning’ and further notes that some people ‘report eating-related symptoms resembling those typically endorsed by individuals with substance use disorders, such as craving and patterns of compulsive use’ (APA 2013, 329). In both cases, people experience a strong drive to persist in the relevant behaviour, and appear aided in doing so by some variety of denial. This parallel is noteworthy in part because it suggests the possibility that denial may be a common factor in psychiatric disorders characterised by maladaptive agency, and so represents a general tendency in human cognition (see Section 4) that, given the right circumstances, can lead to a variety of forms of pathology.

Returning to addiction, why would acknowledging the negative consequences of drug consumption cause psychological pain and distress? One part of the answer to this question must be that these consequences are themselves frightening and upsetting; another may be that it can be overwhelmingly shaming to acknowledge the harm one’s behaviour has done – especially, perhaps, when it involves harm not only to the self, but equally to others the person cares about.²² However, another part of the answer to the question is surely that the psychological pain and distress is not directly about the negative consequences themselves, but rather about the demand that acknowledging them creates, namely, *to desist from the behaviour causing these consequences* – that is, to quit drugs.

Although addicts retain the capacity to refrain from use in many circumstances (see Section 1), there is no question that the desire for drugs characteristic of addiction is very strong and hard to resist (Robinson and Berridge 2008; Holton and Berridge 2013; Segal 2013); and, moreover, for many addicts, drugs additionally serve valuable functions (Muller and Schumann 2011; Pickard 2012 and forthcoming) as well as offering pleasure (Foddy and Savulescu 2006, 2010). Despite the costs, addicts are nonetheless strongly motivated and highly incentivized to continue to use drugs. They may therefore be in denial about the negative consequences of consumption, not only because of

²¹ There are also of course differences. The eating disorder patients report denial only at the height of their disorder, while the second addict quoted above suggests denial occurred prior to using drugs, as opposed to developing during the process of escalating use. There are also first-person reports of denial being at its strongest not during regular use, but immediately prior to relapse, even after long periods of abstinence, as found in the quote from Alcoholics Anonymous above of the alcoholic who orders a cocktail as if it were ginger ale. For discussion, see Crowther forthcoming.

²² For discussion of shame in relation to addiction, see Flanagan 2014.

the psychological pain and distress that would ensue from recognizing these consequences, but because of the pain and distress that would ensue from quitting drugs. They are in denial about the negative consequences of use precisely because they want to persist to such a degree that it is psychologically painful and distressing even to contemplate desistance – let alone actually doing so.

What then is the role of denial in addiction? The puzzle inherent in addiction is why drug use persists in the face of negative consequences. Once costs outweigh benefits, addicts ought to refrain from consumption, but don't. In absence of compulsion, denial can explain why they don't. If we do not know about the negative consequences of our actions, then these consequences cannot guide us in our decision-making. There are of course many possible causes of ignorance and routes to knowledge-failure. But denial is one of them. If, despite the evidence, addicts are in denial that their drug use is causing negative consequences, then the disincentive to continued consumption that this knowledge offers is effectively removed from their psychology and cannot guide decision-making. Denial explains why drug use persists in the face of negative consequences, despite the fact that addicts respond to incentives, by blocking the straightforward attribution of causal knowledge of the negative impact of use.

4. Motivated belief, self-deception, and cognitive deficits in addiction

In many, although certainly not all, cases of addiction, denial therefore plays a central role. But in offering an answer to the question of how it is possible that drug use persists despite negative consequences, the phenomenon of denial raises many questions of its own. How quite generally is denial possible? And, more particularly, can we make any progress identifying some of the processes and mechanisms underpinning denial in addiction? In this Section, I offer some minimal suggestions that naturally emerge from consideration of philosophy and the cognitive sciences, to begin to sketch how we might explore these questions. As should be evident from the discussion in Section 3, denial is an umbrella term, admitting of different kinds, means, degrees, and objects, and we should expect this diversity to be evident with respect to denial among addicts. I therefore look briefly at the possibility of modelling denial in addiction on theories of motivated belief, self-deception, and cognitive deficits respectively.

Motivated belief. It is now well-established that human psychology is not reliably rational: we are prone to various information-processing biases, such as (to name but a few) the availability heuristic, the attentional bias, the confirmation bias, and the 'better-than-average' effect, which demonstrate

that human procedural norms for belief formation differ markedly from the epistemic ideal.²³ In particular, it is possible that the biases contributing to the ‘better-than-average’ effect, which suggests that the vast majority of individuals have an unrealistically positive self-conception and view themselves, as the name suggests, as better than average, may contribute to the belief in personal exceptionalism manifest in the self-reports quoted in Section 3, and hence may also contribute to the explanation of some cases of denial in addiction.

In addition to these information-processing biases, it is equally well-established that belief-formation processes can be influenced by our desires and emotions: we are motivated to believe that which we want or hope for, and to disbelieve that which we do not want to be the case or fear. Motivated belief may co-opt existing information-processing biases to serve its purposes, or may produce further departures not only from the epistemic ideal but from the procedural norms that govern non-motivated belief. For instance, there is evidence that we are more likely to adopt a sceptical mindset and question the validity and quality of preference-inconsistent than preference-consistent evidence (for a review, see Ditto 2009), leading us to shift standards of evidence for belief formation and retention, in relation to our desires and emotions.²⁴

I suggested in Section 2 that acquiring causal knowledge of the negative consequences of drug use should be seen as *an achievement* even in the absence of motivational influences because it may be hard to come by. The causal network may be complicated and interwoven, and the evidence may be equivocal and indeed in some cases open to reasonable doubt. For these reasons, knowledge that consumption is causing negative consequences is an easy target for motivationally biased belief-formation processes. There are many opportunities for a sceptical mindset to prevail, and for both established and more specific biases to obscure or skew the evidence. The ground is ripe for denial.

Self-deception. Traditional accounts of self-deception model it on interpersonal deception. In prototypical instances of interpersonal deception, the deceiver believes a proposition and intends to cause the deceived to believe the opposite. By analogy, traditional accounts of self-deception claim that, in prototypical instances of self-deception, the self-deceiver believes a proposition and intends to cause themselves to believe the opposite (cf. Davidson 1985). There are thus two components to the psychology of self-deception according to this sort of account: on the one hand, the intention to

²³ This importance of distinguishing procedural from epistemic norms is forcefully made in Bayne and Fernandez 2009b. For a review of some of these findings, see Kahneman 2011; Kahneman et al. 1982; Kurzban 2012; McKay and Dennett 2009. Hazlett 2013 chapter 2 offers an empirical review and philosophical discussion of the ubiquity and value of ‘self-enhancement biases’.

²⁴ For discussion of the varieties of ways motivation may affect reasoning, see Mele 2001; Hazlett 2013.

believe the opposite of what one believes; on the other, the result of this intention, namely, the simultaneous existence of two contradictory beliefs.

Traditional accounts of self-deception therefore raise two paradoxes: how is it possible for an intention to self-deceive to be effective, and how is it possible to simultaneously hold two contradictory beliefs (Mele 2001)? Philosophical approaches to these paradoxes are highly varied, and may involve rejecting one or both of these components of the psychology of self-deception.²⁵ But, however these paradoxes are ultimately resolved, some instances of denial in addiction seem to suggest that addicts do know, in some sense or at some level, that their consumption is causing negative consequences. In other words (and putting the issue of the intention to self-deceive to one side), addicts sometimes appear to be subject to something akin to simultaneous contradictory beliefs.

Consider the first-person reports quoted in Section 3. To be sure, the self-description of the addict whose denial set in before they tried drugs, and who did not admit that their drug use carried any health risks until the age of 42, does not offer evidence of deep or hidden knowledge of the negative consequences of consumption. But this is not the case with the other self-reports. The addict described by *Alcoholics Anonymous* who relapses when he treats a cocktail like a ginger ale knows full well the consequences for him of drinking, but has a ‘mental blank’. The alcoholic who drinks after an esophageal hemorrhage takes the step of bringing photos from the endoscopy home, to bolster his self-serving conviction that he now has a ‘super esophagus’, presumably against the nagging thought that in truth he does not. Similarly, one of the anorexic patients quoted above acknowledges she was getting ‘a little bit worried’ even though this did not affect her calorie-restricting. These various self-reports suggest that addicts in denial may sometimes believe, in some sense or at some level, the proposition they deny. If this is right, then understanding such instances of denial will require a general theory of self-deception which acknowledges and explains the capacity to hold contradictory beliefs at one and the same time.

²⁵ For instance, Al Mele (2001) denies both that self-deception involves an intention to self-deceive and the simultaneous existence of two contradictory beliefs, offering a deflationary account of self-deception; Neil Levy (2009) accepts the simultaneous existence of two contradictory beliefs, but denies that there is a genuine intention to self-deceive, as opposed to more common-or-garden-variety motivational influences on belief formation, perhaps alongside the presence of cognitive deficits; Robert Audi (1982) and Neil Van Leeuwen (2007) deny the simultaneous existence of two contradictory beliefs, so that self-deception involves only one bona fide belief. In addition, more scientifically-focused, accounts of self-deception typically aim to address these puzzles by appeal to the possible evolutionary adaptiveness of self-deception alongside a modular view of mental architecture (cf. Kurzban 2012; Trivers 1985 and 2013; Van Leeuwen 2007b; von Hippel and Trivers 2011; see too Mijovic-Prelec and Prelec 2010 who model self-deception on self-signalling and adduce experimental evidence in support of their theory; for some objections, see McKay and Dennett 2009).

Cognitive deficits. In addition to the possibility that denial of the negative consequences of drug consumption in addiction is a form of motivated belief or self-deception, there is also evidence suggesting that addicts show a domain-general impairment in insight and self-awareness, associated with abnormalities in select brain regions, particularly the rostral anterior cingulate cortex (rACC) which is implicated in the processing of personally-relevant information across a range of disorders (for a review, see Moeller and Goldstein 2014). The basic findings indicate that for severely addicted individuals, on the one hand, drug cues are associated with increased rACC activity compared with controls, suggesting these are tagged as having heightened personal relevance; while, on the other hand, error-monitoring and high-risk decision-making across a range of laboratory tasks are associated with reduced activation in the rACC, suggesting that errors and risks are tagged as of limited personal relevance. A further complementary study suggests that individuals with cocaine-use disorders show insensitivity to both predicted and unpredicted losses in laboratory gambling tasks compared to controls, as measured by feedback-related negativity (Parvaz et al. 2015).

The speculative suggestion in this research is that, in addition to drug cues having special salience, addicts may persist in using in the face of the negative consequences of drug consumption due to an impairment in the ability to process the personal relevance of these very consequences, and hence to learn from and be guided by them in relation to future behaviour. It therefore offers an alternative to motivated belief and self-deception as an explanation of a failure to know the negative consequences of drug consumption. On a cognitive deficit model, addicts lack this knowledge not out of denial, as understood in Section 3, but because of a domain-general impairment in the capacity to monitor errors, risks, and losses associated with their actions, as applicable to their addiction in particular. To put it colloquially, on this account, addicts are more akin to a hearing-impaired person who cannot follow a conversation in a noisy restaurant, than a cancer patient who refuses to believe their prognosis. As a result, a cognitive deficit model of impaired insight and self-awareness in addiction does not straightforwardly have the resources to explain those instances of denial which appear to be evidently motivated or, even more, seem to suggest that the addicts in question know, in some sense or at some level, the very proposition they also deny. For on this model, addicts have an impairment which impedes the acquisition of such knowledge at all. Nonetheless, it can solve the puzzle inherent in addiction and explain how use persists despite negative consequences. For, impaired insight and self-awareness can potentially explain why addicts do not know about these consequences, apart from any disposition towards denial.

It is not uncommon to discover that apparently similar pathological symptoms have different underlying explanations. Given the diversity and breadth evident in our folk psychological

conception of denial, we should be prepared from the outset to discover a variety of underlying processes and mechanisms. The more we can distinguish these in principle and learn to identify in practice which process or mechanism is implicated in an individual addict's persistent use, the more we can target clinical interventions to suit need. But, despite the likely variety and complexity of the details of processes and mechanisms underpinning denial or other routes to knowledge-failure, these latter play a unified and central role in addiction. In many, although not all, cases, denial and its ilk are central to the explanation of why addicts persist in using despite evidence that would, ordinarily, suffice for knowledge that drug consumption is causing negative consequences, and so motivate abstinence. For this reason, we should treat addiction not only as a disorder of conation, but as a disorder of cognition. Of course, these forms of disorder may connect, as the motivational influence on belief formation in addiction may be especially strong precisely because of the strength of the motivation to consume. Nonetheless, it is time to move beyond the orthodox conception of addiction, and reckon not only with the evidence that drug consumption involves choice, but also with the cognitive distortions in addiction that impact on that choice.²⁶

²⁶ A version of this paper was presented at the *Mind & Language* Workshop on Addiction, Self, and Self-knowledge in 2015 and I am grateful to the audience for discussion. Special thanks are due to Tim Bayne, Ian Phillips, and Ema Sullivan-Bissett for extremely helpful comments on a previous draft. Many of the ideas in this paper have developed out of discussion and collaboration with Serge H. Ahmed and it is with genuine gratitude that I acknowledge my debt to him.

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