The phenomenology of craving, and the explanatory overreach of neuroscience

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(response to commentaries)

I would like to thank Owen Flanagan and Douglas Porter for their interesting and insightful commentaries, both of which inspired me to think more deeply about aspects of addictive craving. In this response, I will make some clarifying points, particularly regarding my views on the relationship between neuroscience and phenomenology, and I will expand on my thesis, focusing especially on addiction treatment and the role of testimony.

I will start with two central concerns that Flanagan raises, then I will address three of his additional criticisms. First, Flanagan takes issue with my claim that the dominant neurobiological view of craving does not capture the phenomenology of craving. The worry is that I am asking neuroscience to do something that by its nature it cannot; namely, to capture the way craving is experienced first personally.

To begin, I would like to lay out three things that I do not mean when I use the term phenomenology. First, I am not referring to phenomenology as the basic qualitative character of addictive craving, rather I am using phenomenology in a thicker sense; that is, phenomenology as the first personal understanding of the experience of addictive craving, which is interpretive, motivational and often reflective. Phenomenology in this sense is not always the first iteration of an experience, but one that people typically access on reflection. Second, I do not mean that people in addiction have a perfectly clear and articulate purchase on the nature of their addictive cravings, or that they will always describe craving in the precise terms that I give as examples in the paper. The importance of the first person is that the things people commonly say about their cravings reveals that they are relating their cravings to existential aspects of their lives. And third, I do not think that phenomenological evidence is the only kind of evidence for my theory of addictive craving, or that it is the only kind of evidence that matters. What I do think is that testimony that is narrative, reflective, and interpretive, along with the other kinds of evidence that I present (including behavioral facts and evidence from social and psychological research on addiction), directs us towards the more existential analysis of addictive craving that I defend.

Now, to the concern that I am asking too much of neuroscience. I entirely agree with Flanagan that neuroscience should not and cannot be expected to capture the way things are experienced first personally, not because I am a dualist when it comes to craving (or otherwise), but because, as most everyone would agree, neural descriptions are different from psychological descriptions. The neural description will not convey "what it is like." My point is that the desires the received view of craving explains and the desires I am describing are different mind/brain processes, i.e. the desires they explain at the neural level do not map onto the experiences of craving that I describe. This matters, because the desires I depict play a significant role in overriding attempts to not engage in one's addiction (especially in chronic and poly-addiction), according to people experiencing addiction themselves.

The dominant neurobiological account of craving, including the version defended by Berridge and Holton (2013; 2017) and Berridge and Robinson (1993), does a good job of "capturing" (read: explaining) certain kinds of desires that result from using drugs repeatedly across time. However, they are not explanatory of the more existentially loaded desires that play a central role in addiction. In other words, I am exposing the explanatory overreach of the dominant neurobiological accounts of craving. The neuroscience simply does not offer an explanation of the kind of craving I describe; neuroscience is looking in the wrong place insofar as it has not addressed the way cravings connect to the existential and emotional needs of people in addiction. Thus, I am not trying to dismiss neuroscience, but rather to redirect it.

The second major concern that Flanagan expresses is that my theory of addiction does not give a unified account of *all* addictive craving, across all addictions. But he is expecting more from my theory than I am. I am not trying to cover every case, and I do not intend to give necessary or sufficient conditions for addictive craving. Rather, I am drawing attention to a kind of craving that occurs in addiction, but that does not occur for everyone who uses drugs repeatedly or engages in addictive processes across time. These cravings have been conflated with the reward-learned urges described by the dominant neurobiological view of craving, and they should not be. They are more psychologically complex, and have different (existentially loaded) content. What I describe is a pervasive kind of addictive craving that the neuro-theories do not address. In my view these cravings are prevalent and typical across many cases of addiction, but I am not claiming they are true of every case.

Next I turn to three additional criticisms that Flanagan raises. My replies to his first two major concerns – regarding (1) my use of phenomenology and (2) the disunity of craving – will ground my responses to these additional criticisms.

Flanagan claims that my theory positions neuroscience in competition with psychosocial theories of craving. I agree with Flanagan that neuroscience and social and psychological theories of craving need not be in competition. Following my reply to (1), my aim is to show the limits of the explanatory power of the neuro-level analysis, and to warn against dominant neuro-theories that circumvent social and psychological factors. I reveal ways in which addictive craving needs to be treated at the psychosocial level, and to the aspects of craving that cannot be addressed by pharmacological intervention as a primary treatment approach. While psychosocial theories and neuro-theories are not fundamentally incompatible, historically they have been divisive in theorizing and treating addiction. And in some fields of medicine and in public discourse, the dominant neural models *have* overshadowed the alternatives. (This point will later bridge to my reply to Porter's comments concerning the broader problems of neuroessentialism).

Flanagan also criticizes my paper for oversimplifying the dominant neurobiological view of craving. He is right that neurobiological theories of addiction focus on more than the reward system. He names some of the other parts of the brain that are impacted by addiction. However, as I noted in my response to (2), the purpose of my paper is not to undermine all neuroscience of addiction. Rather, I am focused specifically on how dominant neuro-theories explain *craving*.

It is worth emphasizing at this point that, while there are certainly very careful neurobiological theories of addiction, the "brain disease" definition of addiction that has had the most public uptake and substantially undergirds medical treatment of addiction has not been so nuanced. In terms of competing definitions of addiction, the disease model still takes the popular lead in research, clinical contexts, and media (Lewis, 2015, p. 5; Heyman, 2009, p. 17). The National Institute of Drug Abuse (NIDA), for instance, defines addiction as a chronic disease, and it links advances in addiction treatment to advancing brain research (NIDA, 2018).

Finally, Flanagan argues that his own experience of alcohol cravings does not fit my theory. What his narrative brings out is important: addiction is diverse, highly personal, and changes across time. Moreover, addictive substances and behaviors can play different roles in different people's lives, as well as in different periods of an individual's addiction. However, his description of craving in late-stage alcoholism does not fall outside of my account of addictive craving. What he describes is an intense desire for relief from physical (and psychological, it seems) pain and distress, a desire that outweighs considerations of how continued alcohol use would be importantly detrimental. Flanagan's experience of craving is perhaps a limiting case of an existential construal of craving, but nonetheless, what he describes is a case of a craving to drink in service of avoiding pain.

Late stage alcoholism can produce peculiar forms of physical deterioration that distinguish it from other substance addictions to the extent that it foregrounds the extreme physical risks of withdrawal and detox. In this respect, it makes sense that relief from physical pain and duress may top the list of aims of craving. (However, I would contend that relief from shame and other painful emotions are prominent in most cases of late stage alcoholism as well; that is, the more emotional or existential aims that I have addressed.) Craving aimed at pain relief is not outside the bounds of my theory of craving, and furthermore, it is not captured by the dominant neuro-explanation of intense urges triggered by an encounter with a cue associated with one's addiction.

This takes me to Porter's commentary. Porter focuses on the ways that neuroscience tends to promote a kind of essentialism that is detrimental to individuals experiencing addiction, marginalizes psychosocial approaches to addiction, and perpetuates discriminatory social norms (as well as drug policy that has been used historically – and continues to be used – to criminalize and punish racialized and poor people). This seems entirely right to me. Following on from that, I will make two brief points of clarification. Then, referring to Porter's arguments against neuroessentialism, I will expand on how my theory of craving bears on questions about addiction treatment approaches and how it speaks to the importance of testimony and the epistemic authority of people with addictions.

First, I want to clarify that, while I do intend to push back on the kind of dominant neurobiological accounts of craving that I have described, I am not suggesting that addictive cravings cannot be explained at the level of the brain, or that the phenomenology of addictive craving is separable from the brain. I do not endorse anti-materialism. And fortunately, I do not need to take that metaphysical stance in order to maintain that the received view of craving falls short and casts too wide a net in terms of which phenomena in addiction it successfully explains.

Secondly, I do not think that all neurobiological theories of addiction are dogmatically neuroessentialist, as per the definition provided by Porter; that is, that they endorse the claim that "the brain is the underlying 'essence' of a person" and that "mental disorders have inherent, underlying and determinant biological basis in the brain." I share Porter's concerns about neuroessentialism, but in fairness to neuroscientists, I do not assume they are all neuroessentialists. I presume that Berridge and Holton (2013; 2017) and Berridge and Robison (1993), for example, would take a softer stance (I doubt they would argue that the brain is the essence of a person). I take myself to be agreeing with Porter on this, and I want to echo his well-put point that neuro-theories, and especially the traditional disease model of addiction, feed neuroessentialist biases.

Next I turn to the fact that how we understand the cause and nature of addictive craving has implications for research, funding, public policy, and care and treatment options for individuals dealing with addiction. One of the main worries motivating my paper is that by looking primarily to the impact of drug exposure on the brain to explain the desires driving addiction, we easily end up with a picture of addictive craving that is construed as antithetical to a harm reduction approach to addiction. This is concerning because there is plenty of evidence that harm reduction is highly effective in improving individual wellbeing and supporting recovery for people in addiction, especially people who are socio-economically disenfranchised and face other intersecting forms of oppression. (Relevant studies include Marshall et al., 2011; Uchenhagen et al., 2000; & Wood et al., 2006).

A foundational aim of harm reduction is to find strategies and practices that reduce the negative consequences of addiction (including the harm caused by social stigma and criminalization), while meeting individuals where they are at, in terms of emotional, psychological, social, and material needs. This involves helping individuals pursue their own wellbeing, where they get to define wellbeing for themselves; wellbeing is not equated with abstinence. To put my worry simply, if drugs are taken to be the primary cause of addictive craving, then it is easy to dismiss or at least be highly skeptical of an approach to treating addiction that does not require or even prioritize drug abstinence – though abstinence is not incompatible with the aims of harm reduction. (See, for instance, Maguet, O., Lumann, N., & Debaulieu, C. (Eds.), 2013).

Porter says of my description of the contents of addictive craving—versus "brute intense urge" – that "the modifiable nature of the fulfilment of those desires seems more compatible with hope for recovery." To this point, on my account of addictive craving, supporting someone to be able to resist their cravings will have much more to do with ensuring that their emotional, social, material, and existential needs are being met, and that they have options to fulfill these needs outside of engaging in their addiction, and thus, that drugs and addictive behaviors become one option among others. So, my account of addictive craving, in alignment with a harm reduction model, does not entail abstinence as the solution to craving, or as the means to enabling self-control.

Porter draws attention to the fact that different models of addiction predict different roles for testimony in treatment. If addictive craving can be sufficiently explained by examining the impact of repeated drug use on the brain (as per the dominant neurobiological approach), then it is unclear what role testimony ought to play in research and treatment targeted at reducing the impact of addictive cravings. The concern is that epistemic authority regarding treatment and intervention is granted to scientists and medical experts, not to individuals who have insight into the *experience* of addictive

craving (but typically not into the nitty gritty of the neural mechanisms linked to sustained substance use).

This is especially pressing because people in addiction are often accorded deficient credibility when it comes to reporting on their addiction. "Addict" used as an identity category has accrued stereotypes: intentionally deceptive, or self-deceived (prone to denial, rationalization, self-justification). A plausible contributing factor to the popularity of the neurobiological model of addiction is that these models do not rely on testimony or phenomenological explanation from "unreliable narrators." (Similar ideas are explored in literature on epistemic injustice, medicalization and mental disorders; for a few examples, see Wardrope, 2014; Kidd & Carel, 2017; and Scrutton, 2017.) Additionally, many people with addictions belong to more than one marginalized social identity group; i.e., groups that face identity prejudicial credibility deficits (Fricker, 2007), which only compounds testimonial injustice. The people who are the most affected by addiction, who are the most 'puzzling,' are the people whose testimony is most commonly dismissed as unreliable. According to my account of the contents of craving, the epistemic authority gained from first personal experience is essential to treating and intervening on addictive craving. Thus, testimony will play an indispensable role.

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