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Response to commentaries

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The commentaries reinforce our claim that humans' ability to verbalize their desire to change and treatment goals are core features of addiction that cannot be modelled in animals. We agree that preclinical research is important, but we remain skeptical about the value of animal models of addiction, no matter how sophisticated.

An anonymous reviewer of our paper stated: "I hope that addiction researchers who have built (or are building) their career and reputation on developing, testing and using animal models of addiction will take the time to seriously ponder the authors' arguments and appraise them constructively before engaging in the debate". We thank the commentators for doing so, and we found common ground with each of them who highlighted issues that we discussed in our paper. Specifically, the importance of distinguishing animal models of addiction from models of drug instrumentalization (Müller(1)), the possibilities afforded by the 0 / 3 crit model and its combination with alternative reinforcers including social interaction (Deroche-Gamonet (2)), and the important role for preclinical models in delineating brain adaptations associated with chronic drug use or vulnerability to addiction (Perry & Lawrence (3)).

Despite these areas of consensus, our argument that addiction may be a uniquely human phenomenon was not convincingly challenged by any of the commentators. Building on previous work (4-7), we argued (8) that a defining feature of addiction may be the persistent failure to refrain from or reduce drug use despite prior intentions to do so. This construct is dependent on language and therefore impossible to model in non-human animals. Rebuttals to this argument reiterated the features of addiction that *can* be modelled in animals using

the 0 / 3 crit model (2), or the need to apply the 0 / 3 crit model to vulnerable subpopulations of animals (1). Both counterpoints sidestep our argument that animal models may never be able to capture the essential features of addiction in humans, no matter how sophisticated. Indeed, two commentators referred to the role of language in their rebuttals: by acknowledging that drug users "ask for support when their drug taking becomes maladaptive" (2), or by pointing out that humans with addiction may want different things from treatment (complete abstinence versus moderation)(1). Indeed, moderation of drinking is a desirable and achievable goal for many people with alcohol dependence (9), but clinical research depends on patients' ability to verbalize their treatment goal.

Deroche-Gamonet (2) offers the example of molecular and biological research on diabetes, which sits comfortably alongside research on social and commercial determinants of the disease, as a defense of the importance of studying basic processes in preclinical research. Unfortunately this analogy between diabetes and addiction is undermined by network models of *psychiatric disorders* that emphasize the redundancy of any reductionist approach that fails to consider the relations *between subjective symptoms* (10). This may be particularly relevant to addiction (11).

Finally, Perry and Lawrence (3) discuss ways in which animal models can be useful for probing the individual differences that predispose to drug use, or the neural mechanisms that underlie neuroadaptations that occur after chronic drug exposure. We agree. All commentators argued that the future for animal model of addiction looks rosy if one considers more sophisticated models that are able to model multiple features of addiction. Time will tell if this optimistic forecast is justified, but we hope that our paper has stimulated consideration

that a useful animal model of addiction may be unattainable, and the continued pursuit of one can mislead and ultimately be a waste of resources.

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