and substance use disorders, and the possibility of specific underlying neurobiological mechanisms, stands out to a certain extent as an overly narrow perspective.

The subheading of the paper is 'What can we learn from the brain?', which can be said to be consistent with the content inasmuch as the prospect of connecting dysfunction in specific brain regions with identified symptoms in both depression and substance abuse disorders is a major theme in the paper. A long list of references concerns findings from the use of functional magnetic resonance imaging (fMRI) identifying regions and structures of the brain that have a bearing on depression and substance use disorders. However, in this case one also has the impression that the authors are unwilling to look critically at the evidence that supports their ambition to provide a neurobiological underpinning for the effects of MT on depression and substance use disorders. The interpretation of brain activation maps is by no means a straightforward task due to the complex neuronal interconnections in the brain, which make it difficult to decide whether the identification of activity in a given part of the brain is primary or secondary to activity in some other part [6]. As pointed out by Kalant [7], there are good reasons to believe that neurobiology will never be able to tell us all that we need to know about addiction and we would be well advised to refrain from developing reductionist models of addiction.

Declaration of interest

None.

Keywords Depression, mindfulness, neurobiology, outcome equivalence paradox, specific mechanisms, substance use disorders.

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RESPONSE TO COMMENTARIES

We appreciate the thorough and thought-provoking commentaries offered in response to our For Debate paper [1]. Drs DiClemente [2] and Bergmark [3] raise several important issues.

First, it is argued that the case for the efficacy of mindfulness training (MT) is overstated, as the majority of studies cited have not used active comparison groups. Indeed, more recent meta-analyses have suggested that the referenced paper suggesting that MT may not be effective was 'premature and unsubstantiated' [4]. We agree that more well-controlled trials are necessary, especially for substance use and co-occurring disorders [5,6].

Secondly, both commentators raised the important question of whether the paper's focus on rumination and stress is too narrow. They point to the contexts of distress and affect regulation, and also call attention to other, more wide-ranging effects of MT. We argue that stress and negative affect share function through the common process of associative learning: as stress leads to negative affect, negative reinforcement loops are generated and maintained by repeated engagement in behaviors aimed at decreasing these 'unpleasant' affective states [e.g. stress → negative affect → craving/wanting the unpleasantness to go away → behavior (e.g. drug-taking, self-appraisal/rumination) → reduction of negative affect → associative memory → increased salience of stress, etc.] [7–13]. Thus, commonalities in regional brain dysfunction might point towards a shared dysfunctional pathway that is core to both depression and substance use disorders (SUDs): associative learning. MT targets this associative learning process, which includes not only negative affect, but also craving and stress as core elements [14–16]. Thus, it is unclear if associative learning as a potential mechanistic target of MT should be categorized as narrow or wide-ranging. Nevertheless, as the mechanistic underpinnings of MT are as yet unknown, it is helpful to have a relatively clear focus (e.g. rumination and stress) to begin investigating how it works. Observing how MT affects the different 'spokes' of the associative learning 'wheel' differentially may give insight into their relative effects on the other spokes, as well as the wheel as a whole. On the other hand, maintaining openness and flexibility to incorporate emerging data in modifying models is also important, and additional intermediary phenotypes such as emotional regulation appear important in addiction, depression and their co-occurrence, and in how MT might target these disorders.

Thirdly, the question was raised about the uniqueness of the potential active ingredients of MT, given that other techniques such as transcendental meditation and relaxation imagery have shown similar cardiovascular and neural effects. Indeed, it is possible; in fact likely, that MT shares active ingredients with other techniques. For example, all three practices use directed attention (i.e. breath, mantra or body part). However, MT may be unique in bringing an attentional as well as an acceptance-based focus to not only formal exercises (e.g. meditation) but to everyday activities and states (e.g. mood and cravings), fostering greater awareness and self-control in response to previously problematic situations or states. In fact, a recent study showed that mindfulness training was associated with improved performance on the Stroop task, suggesting that this training helps to bring even basic, automatic reactions under more conscious control [17]. Future studies will be critical for teasing apart potential additional/unique effects of MT above and beyond those shared with other techniques.

Fourthly, the point that reductionism can be misguided was raised. Again, we agree that reductionism is often not helpful. However, finding common core elements or intermediary phenotypes contributing to disorders may give us a starting point from which to make headway towards their underpinnings and treatment.

Declarations of interest

None.

Keywords Addiction, co-occurring disorders, depression, dual diagnosis, mindfulness, substance use treatment.

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