A paradigm shift for addiction science?

¿Un cambio de paradigma para la ciencia de la adicción?

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Abstract

This article assumes that a radical transformation is currently occurring in addiction science and asks whether this can usefully be seen as a Kuhnian 'paradigm shift'. The puzzle of addiction is that people labelled as addicts persist in behaving in ways they know cause harm to themselves and others. In the currently dominant 'normal science', the answer to this puzzle is that addictive behaviour represents a kind of compulsion caused by a disease of the brain. However, this is contradicted by anomalous findings from several types of evidence that addictive behaviour is not automatic and compelled but is voluntary and intentional. The emerging paradigm is therefore based on the assumption that addictive behaviour is a disorder of choice. How the puzzle addiction can be solved is the first task to be addressed under this new paradigm but some possibilities are suggested. If it is believed that evidence from neuroimaging is sufficient proof that addiction must be a brain disease, reasons are provided for why such a belief is unfounded. Implications for the treatment and prevention of addiction arising from the new paradigm are explored. The article concludes by pointing out that the existence of the Addiction Theory Network shows that the author is not alone is believing that a paradigm shift in addiction science is possible and by alerting the reader to a forthcoming multi-authored book in which the validity of the brain disease model of addiction is comprehensively examined.

Keywords

Addiction, Science, Paradigm shift, Brain disease, Compulsion, Voluntary and intentional behavior, Disorder of choice.

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Resumen

Este artículo parte de la base de que en la actualidad se está produciendo una transformación radical en la ciencia de las adicciones y se pregunta si puede considerarse útil como un "cambio de paradigma" kuhniano. El rompecabezas de la adicción es que las personas etiquetadas como adictas persisten en comportarse de manera que saben que se dañan a sí mismas y a los demás. En la "ciencia oficial" actualmente dominante, la respuesta a este enigma es que el comportamiento adictivo representa una especie de compulsión causada por una enfermedad del cerebro. Sin embargo, esto se contradice con los hallazgos anómalos de varios tipos de pruebas que indican que el comportamiento adictivo no es automático y obligado, sino voluntario e intencionado. Por tanto, el paradigma emergente se basa en la suposición de que el comportamiento adictivo es un trastorno de elección. Cómo se puede resolver el rompecabezas de la adicción es la primera tarea que debe abordarse bajo este nuevo paradigma, pero se sugieren algunas posibilidades. Si se cree que la evidencia de la neuroimagen es prueba suficiente de que la adicción debe ser una enfermedad cerebral, se ofrecen razones de por qué tal creencia es infundada. Se exploran las implicaciones para el tratamiento y la prevención de la adicción derivadas del nuevo paradigma. El artículo concluye señalando que la existencia de la Red de Teoría de la Adicción (Addiction Theory Network) demuestra que el autor no es el único que cree que es posible un cambio de paradigma en la ciencia de la adicción y alertando al lector sobre un próximo libro de varios autores en el que se examina exhaustivamente la validez del modelo de enfermedad cerebral de la adicción.

Palabras clave

Adicción, Ciencia, Cambio de paradigma, Enfermedad cerebral, Compulsión, Comportamiento voluntario e intencional, Trastorno de elección.

Since its introduction by Thomas Kuhn over 60 years ago (Kuhn, 1962), the concept of 'paradigm shift' has become one of the most overused in our vocabulary, both lay and scientific. During my time in the field of addiction studies, there have been several innovations that have been proclaimed to be paradigm shifts in the scientific explanation of addiction but which have turned out to be no such thing, either absorbed into the existing mainstream or overtaken by other developments. Kuhn's main examples of paradigm shifts from the history of science are the renaissance transition from a geocentric to a heliocentric view of the universe and the replacement of Newtonian by Einsteinian physics at the beginning of the 20th Century, high standards to aspire to for anyone wishing to announce a paradigm shift for addiction science. Nevertheless, I do believe that a profound change in the scientific understanding of addiction is currently taking place in our field and my main objective in this article is to summarise the nature of this alleged transformation. I am not concerned here to argue definitively, one way or the other, whether this change qualifies as a paradigm shift; I suggest it rather as an interesting possibility that may be helpful in considering the current state of addiction science.

The puzzle of addiction

In order to compare and evaluate competing accounts of addiction, it is first necessary to clarify what it is that any satisfactory theory of addiction must seek to explain. I suggest, and many would agree, that this is the fact that people we call addicts persist in behaving in ways that they know cause harm to themselves and often to others. Note that this does not refer merely to behaviour that *others* believe to be causing harm; it is essential that the individual in question *recognises* that their behaviour is harmful and, moreover, wishes on frequent occasions to quit. It follows that people suffering from what we call addiction try often to change their behaviour but fail to do so. While we know that many do eventually succeed in changing without professional help (e.g., Lopez-Quintero et al., 2011), it is because of this difficulty that others seek help, either from formal treatment or mutual aid organisations; they wish to change their behaviour but are unable to achieve this change by themselves and seek assistance to do so. (This characterisation of addiction applies as much to so-called behavioural addictions, like 'compulsive' gambling, gaming etc., as to conventional substance addictions, but that is another story.)

But how can it be that someone persists in behaving in a way they *know* is doing them harm? If one knows that behaving in a certain way is harmful and causes pain and distress, why does one not simply desist? The fact that some people do not desist under these circumstances is the essential irrationality of addiction. It may also be called the puzzle of addiction (Pickard, 2019) that any scientific account must attempt to solve.

Normal science: addiction as disease

Assuming that a paradigm shift is underway, what is the nature of the 'normal science' that is under threat. It is, in broad terms, scientific activity based on the assumption that addiction is a disease or, more precisely, that addictive behaviour – repetitive drug seeking and drug consumption despite knowledge of adverse consequences - is a symptom of an underlying disease. As is now well known, the idea that addiction is a disease arose around the end of the 18th and beginning of the 19th Century specifically in relation to alcohol (Levine, 1978). 'Habitual drunkenness' was portrayed as a 'disease of the will' which rendered the individual powerless to resist drinking. This concept of powerlessness was later transferred to opiates and thence to other substances and activities during the 20th Century. Thus, the general answer provided by the disease paradigm to the puzzle of addiction is that addicts continue to engage in addictive behaviour despite their awareness of adverse consequences because they are *compelled* to do so; they cannot choose to refrain from damaging behaviour because, unlike those not suffering from the disease of addiction, they have no choice in the matter.

In more recent times, the disease concept of addiction has taken on a particular form based on the rapid advance at the end of the 20th Century of neuroscience as a scientific discipline. This is, of course, the *brain disease model of addiction* (BDMA), vigorously promoted by the *National Institute of Drug Abuse* in the USA (see, e.g., Volkow et al, 2016). Since the 1990s when it was first advanced, the BDMA has come to dominate scientific, professional and policy discourse on addiction, especially in the USA but to varying extents in other countries of the

world too. There is no question that it represents the dominant and 'normal' paradigm for the study of addiction in world science (see Heather et al., 2021b).

In relation to the puzzle of addiction, however, no matter how theoretically sophisticated and technically advanced it has become, the BDMA continues to rely on the notion of compulsion. When the exact nature of this modern, neurobiological view of compulsion is examined more closely, it appears to vary among versions of the brain disease model and to be somewhat confused, particularly in relation to whether some automatic process is involved or whether it is the old-fashioned notion of 'irresistible desire' that is key to compulsion (see Heather, 2017). But whatever specific version of compulsion is considered, and whatever nuances it might convey, the answer to the puzzle of addiction offered by the BDMA is that addicts are compelled to behave as they do.

The anomaly: addictive behaviour is voluntary and intentional

According to Kuhn (1962), scientific revolutions occur when periods of paradigm-regulated normal science are disrupted by a novel finding, one which cannot be predicted from within the existing paradigm or be made to find a place within it. Preoccupation with these anomalies, as Kuhn calls them, lead to a state of crisis in which the technical, puzzle-solving activity of normal science breaks down and is replaced by a re-examination, often involving considerable acrimony, of the fundamental assumptions that have up to then been in force. The response of the scientific community is invariably one of polarisation, with some defending the old paradigm and others urging its replacement by the new one. In time, the outlines of the new paradigm emerge and the scientific discipline in question enters its next normal period.

If this description applies to what is now happening in addiction science, what is the crucial anomaly that subverts the old paradigm and will eventually lead to its downfall? In this case, it is not a single finding but rather a collection of findings that have the same overall implication: that addictive behaviour, rather than being automatic, compelled, involuntary or 'against the will', is in fact voluntary and goal-directed, intentional behaviour. The first powerful evidence for this conclusion goes back to a considerable body of experimental evidence collected during the 1960s and 1970s showing that the drinking of even the most chronic and severe alcohol addicts was *operant* behaviour, i.e., behaviour that is determined by its consequences (see Heather, 2017). The particular reinforcement contingencies applying to the drinking behaviour of chronic alcohol addicts obviously show marked differences from those applying to non-alcohol addicts, but what these findings clearly demonstrated is that, rather than being qualitatively different and 'compelled', alcohol addicts' drinking behaviour follows the same general laws that govern normal, goal-directed behaviour of any kind. The same conclusion can be drawn more recently from experiments with crack cocaine addicts recruited from the general community (Hart et al., 2000) and from research on nicotine addiction (see Heather, 2017).

Moving from the experimental laboratory to the real world of treatment for addictive disorders, the most efficacious method of treatment according to meta-analysis of a large number of randomised

controlled trials (Dutra et al., 2008) is *contingency management* (CM). This evidence further supports the conclusion that addictive behaviour is operant behaviour largely dependent on its consequences. Studies of CM programmes with physicians, airline pilots and other professional groups have reported remarkably successful results, but similarly high rates of recovery have been obtained among less privileged groups (DuPont & Humphreys, 2011).

There is a range of other types of evidence that is consistent with and strengthens the conclusion that addictive behaviour is operant, and thus voluntary and intentional, behaviour (see Heather, 2017; Satel; & Lilienfeld, 2014). Perhaps most convincing is results from a follow-up of veterans of the Vietnam War. Towards the end of the war, the US government became alarmed about reports that a large proportion of American servicemen in Vietnam were addicted to heroin or other drugs. A team of researchers was commissioned to interview a large sample of men in Vietnam to determine the extent and characteristics of their drug use, and then to follow them up on their return to the US after discharge in 1971. Against all expectations, the great majority had simply 'given up' addiction. In the first year after return, only 5% of those who had been addicted in Vietnam were addicted in the US; and despite reports of withdrawal symptoms, 88% had not resumed regular use of opiates at a three-year follow-up. This did not occur because drugs were unavailable after return home; interviewees reported that they knew how to obtain heroin and some had occasionally, but not regularly, used (see Robins et al., 1974). This evidence is of course inconsistent with the idea that addictive behaviour is the result of compulsion or with any the notion that it is the expression of an underlying disease, including a brain disease.

Evidence from the Vietnam veterans' follow-up is now well-known, certainly by most scientists and professionals in the addiction field. The same is true to some extent of the other types of evidence that are inconsistent with the assumption that addiction is a disease based on compulsion. So what is the response to this evidence of those who support the disease theory and the BDMA in particular? Unfortunately, it is simply to ignore it and carry on as though it never existed. If it is true that a paradigm shift is occurring in addiction science, they will not be able to ignore this evidence forever.

The new paradigm

Assuming again that it is helpful to think in these terms, what is the nature of the new paradigm that will replace the disease paradigm of addiction? Since the new paradigm is, by definition, only emerging, it is impossible to be precise about all its features. However, it is already clear that expressions of the new paradigm are to be found in what is frequently called the *choice* perspective on addiction (Henden et al., 2013).

The label of 'choice' clearly derives from the demonstration, as we have seen, that addictive behaviour is voluntary and intentional, since that is what we mean when we say that behaviour represents a choice. But there is an obvious problem here. How does regarding addictive behaviour as a choice address the puzzle of addiction? If not by compulsion, how can it be explained, under this new perspective on addiction, that people choose to do what they know is harmful to them? One kind of response to this question is simply to deny the puzzle of

addiction and insist that addictive behaviour signifies a simple choice, no different in nature from the ordinary, everyday choices we make all the time (e.g., Schaler, 2000). This is *not* the position taken here. Rather, crucial to the validity of any new way of explaining addiction is the assumption that addiction is a *disorder* of voluntary and intentional behaviour – or, in other words, *a disorder of choice* (Heyman, 2009).

The exact nature of this disorder is perhaps the first problem to be addressed by conceptual analysis and investigation under the new paradigm, and only a few passing comments may be made here. One possibility is that it is a disorder of choice *over time* or, in other words, a kind of failure to make consistent choices over time. Although addicts respond to incentives and are free to choose to engage or not engage in addictive behaviour at any one time, their autonomy is impaired when their pattern of choices is considered over time (Levy, 2006). From this perspective, rather than compulsion, the hallmark of addiction is inconsistency, ambivalence, vacillation and conflict (cf., Orford, 2001). More generally, perhaps the leading explanatory perspective on addiction under the new paradigm derives from the discipline of *behavioural economics* (Vuchinich & Heather, 2003; Acuff et al., 2021). Aside from behavioural economics, an alternative way of explaining addiction is the biased choice model proposed by Verschure and Wiers (2021). There are indeed many accounts of addiction in the existing literature that may be classified as variations of the disordered choice approach and that eschew the BDMA and the idea of compulsion. To attempt to cover them here would go beyond the aims of this article but some may be found in Heather et al. (2021a).

One more important point should be made before moving on. In addition to the implications of the evidence summarised above that addictive behaviour is voluntary and intentional, the most damaging criticism of the BDMA and disease theory of addiction in general is that it ignores economic, social and cultural variables – economic and cultural poverty, lack of social capital, high levels of drug availability, absence of opportunities for alternative rewards, etc. – in addiction (Reinarman & Granfield, 2015). Supporters of the BMDA often mention social factors in their defence of the model (e.g., Leshner, 1998) but this is merely to point to factors that affect the expression of an underlying disease, in much the same way that ignorance and prejudice might be said to affect the observed expression of epilepsy. But this criticism of the BDMA goes further than this by maintaining that socio-economic and cultural variables are implicated in the *causes* of the disorder - as much as, and in systematic interaction with, neurobiological factors (Borsboom et al., 2019).

The relevance of this to the present discussion is that it is only by regarding addictive behaviour as voluntary and intentional that socio-economic and cultural causation can come into play in our explanations of it. If it were an automatic, compulsive phenomenon, it is difficult to see how addictive behaviour could by influenced by economic, social and cultural factors; once it is seen as voluntarily chosen, it can immediately be seen as subject to influence by social norms, learned expectations, cultural traditions etc.. This enormously extends the range of potential causative factors that have to be considered in a novel understanding of addiction beyond the disease tradition. (For clarity, it should be noted that it is not being suggested here that automatic processes

play no part in addiction; drug cravings and urges, for example, are automatic in origin. But craving and urges do not lead inevitably to drug seeking and use without the intervention of psychological, social and cultural variables that, it is argued here, must be taken into account in any satisfactory theory of addiction.)

But what about changes to the brain?

An objection to the possibility of a paradigm shift for addiction can be anticipated. This is the belief that addiction must be a brain disease because of evidence from neuroimaging (fMRI or PET) that addiction is brought about by the prolonged effects of drug consumption on the brain. It follows that other kinds of evidence, including that might suggest the need for a paradigm shift, are subordinate to this crucial observation because this *proves* that addiction is a brain disease. This 'proof' is frequently accepted by members of the general public and by many practitioners and scientists in the addiction field as well. We shall see, however, that it is unfounded.

It should be noted that there are considerable methodological problems associated with the neuroimaging research in question. These include lack of replication of findings, small sample sizes and low statistical power, inappropriate selection of control groups, failure to control for pre-existing differences between experimental and control groups, questionable interpretation of results, and lack of demonstrated relationships between neurological and cognitive/behavioural measures (see, e.g., Button et al., 2013; Grifell & Hart, 2018). However, for the sake of this argument, let us leave these objections aside and assume that existing research has demonstrated reliable and valid differences between the brain structure or function in addicts and non-addicts.

The first important question is whether these differences can be interpreted as indicating the *cause* of addictive behaviour. For this inference to be made, it is obviously necessary that a relationship between brain changes and behaviour can be demonstrated to exist over time. Unfortunately for this causal hypothesis, however, nearly all the existing neuroimaging evidence refers only to a single point in time; the brains of addicts are compared with those of non-addicts on a single occasion but not thereafter. Even stout defenders of the BDMA like Heilig et al. (2021) conclude that "none of the brain imaging findings are sufficiently specific to distinguish between addiction and its absence, and … are typically obtained in cross-sectional studies that can at best establish correlative rather than causal links" (p.5). As might be expected, Heilig and colleagues believe that improvements in brain imaging techniques will eventually be able to distinguish between addiction and its absence, and that such differences will eventually be shown to have causal significance. Be that as it may, it would be helpful if scientists who share this suitably cautious interpretation of evidence from neuroimaging studies would inform the general public, and some of their scientific colleagues too, that proof that addiction is a brain disease is not yet available and possibly may never be.

But let us assume, again for the sake of this argument, that some causal link between changes in the brains of addicts and observed addictive behaviour has been demonstrated. Does this make addiction unequivocally a brain disease? A clear demonstration that changes to the brain need not betoken brain disease comes from a famous

study of the brains of London taxi-drivers (Maguire et al. 2006). To qualify as a taxi-driver in London, one must acquire 'The Knowledge' of tens of thousands of streets in the city and their layouts. Maguire and her colleagues analysed structural MRIs of the brains of licensed taxi drivers and compared them to those of control subjects who did not drive taxis. Their main finding was that the posterior hippocampi of taxi drivers were significantly larger than those of the controls. (The posterior hippocampus is known to be the area of the brain responsible for storing a spatial representation of the environment.) Hippocampal volume was correlated with the amount of time spent as a taxi driver. The investigators concluded that "… there is a capacity for local plastic change in the structure of the healthy adult human brain in response to environmental demands" (p.4398). It may also be concluded that changes to the structure of the brain in themselves are insufficient grounds to warrant the attribution of brain disease (unless one wishes to regard acquisition of 'The Knowledge' as a disease!). Some other grounds in addition to demonstrated brain changes are necessary for the attribution of a brain disease.

To return to addiction, Marc Lewis is a neuroscientist who accepts that repeated, long-term ingestion of psychoactive substances changes the brain but who contends that these changes reflect deep learning rather than neuropathology (see Lewis, 2018). In any case, the crucial question here, as Heyman and Mims (2017) point out, is not whether drugs change the brain but whether they change the brain so that drug use is no longer voluntary and intentional. In other words, it is the question whether evidence on brain changes in addiction from neuroimaging research solves the puzzle of addiction that was posed at the beginning of this article by demonstrating that those brain changes remove the possibility of choice and make drug seeking and use compulsive. According to Heyman and Mims, "To determine whether drug addicts are compulsive drug users, we need to know what influences drug use in those who meet agreed-upon criteria for addiction. If the factors are similar to those that affect voluntary actions, then drug use in addicts remains voluntary, albeit irrational and self-destructive" (p. 389).

A final point about neuroimaging evidence and neuroscience in general should be made before ending this topic. This is the frequently-encountered conflation of neuroscientific research on addiction with support for the BDMA. Conversely, it is the conflation of criticism of the BDMA with criticism of neuroscience itself. That the brain is the basis of all experience and behaviour is a truism that no-one in their right mind could possibly deny. This does not mean, however, that any research demonstrating the role of neural mechanisms in addictive behaviour has shown it to be a brain disease, nor that all criticisms of the BDMA are criticisms of neuroscientific research as a whole. To be as clear as possible, to criticize the BDMA and call for its replacement by an improved understanding of addiction does not itself imply a criticism of neuroscience or an underestimation of its role in achieving a fuller understanding of the nature and causes of addiction and recovery from it.

Implications for treatment and prevention

It is difficult to anticipate clearly what changes to the treatment and prevention of addiction might occur after a paradigm shift

but a few generalisations can be attempted. First, there is the point made by Wayne Hall and colleagues (2015) that the BDMA has not helped to deliver more effective treatments for addiction, as had been promised, and that its effect on public policies on addiction has been modest at best. Hall et al. argue that the focus on the neurobiology of a minority of severely addicted individuals has undermined the implementation of effective and cost-effective population-level policies targeted, for example, at discouraging people from smoking tobacco and heavy alcohol consumption. They also question the pursuit of high technology interventions aimed directly at the brain when most people with addiction continue to lack access to psychosocial and drug treatments of proven effectiveness, such as contingency management, cognitive-behavioural therapy, motivational interviewing, nicotine replacement therapy and methadone maintenance. Thus the demise of the BDMA would redirect resources towards the dissemination of treatments that are known to work, as well as an increased investment in population preventive policies.

The main practical advance in treatment anticipated by BDMA supporters is the development of new pharmacological substances and other invasive medical interventions to correct the alleged brain malfunction responsible for the brain disease. There is no question that pharmacotherapy has a role to play in addiction treatment, mainly by making possible a period of stability in which problems in relationships, accommodation, livelihood etc., may be addressed. But relying mainly on pharmacotherapy as the permanent solution to an addictive disorder leaves the *self*-regulation of behaviour largely untouched. And, as already noted, responding to addiction as if it were a disease of the brain ignores social, environmental and cultural influences that must be addressed if lasting recovery is to be achieved. It also ignores and is unable to comprehend unique, human-level, histories and individual differences which must be taken into account in the attempt to forge a new way of life and identity (Hammersley, forthcoming). To use what is perhaps another overused term, the BDMA *dehumanises* addiction treatment. This is not to say that no current treatment is sensitive to the human level and or takes account of social/environmental circumstances; no doubt it does. But if the BDMA were to become increasingly dominant, the fear is that these essential qualities of treatment will be washed away in a preoccupation with biotechnical solutions. Treatment responsive to the possible paradigm change outlined here would avoid these deficiencies of treatment based on the BDMA.

There is also the charge that the disease concept of addiction and the treatment based on it have the effect of reducing addicts' chances of recovery by telling them that they are powerless to change without special help. Indeed, Peele (2017) alleges that treatment founded on the idea that addiction is a chronic, relapsing brain disease, implying thereby the conceptual and treatment goal of eliminating choice in addiction and recovery, is "not only futile, but iatrogenic" (p.97). The future of treatment for addiction under a new paradigm would be centred on the opposite goal of encouraging choice and empowering people to change. There is much theory and research to support this argument. Efficacy expectancies, our belief in our ability to master a specific change in behaviour, are the most important determinant of successful therapeutic and self-initiated change according to the domi-

nant theory of behaviour change during the second half of the 20th Century (Bandura, 1997). And increased self-efficacy is an essential component of in the most influential model of relapse prevention (Marlatt & Donovan, 2007).

It is also very relevant to this issue that, despite the fact that professionally-delivered addiction treatment is beneficial for many individuals, only a minority of those who recover from addiction-related problems actually receive it. Humphreys (2015) refers to 'the gatekeeper myth' which says that recovery can only be achieved with the assistance of highly-educated specialists in addiction treatment. Evidence shows that this assertion is completely false, yet the myth continues to undermine individual efforts aimed at 'natural recovery' (Klingemann et al., 2007) and downplays the contributions of non-professional sources of help, e.g., mutual-aid groups, pastoral counselling and community-based contingency-management programs operated by the criminal justice system. The myth also creates unrealistic expectations about the effectiveness of formal treatment. These misconceptions would hopefully disappear under a new paradigm in which there would be a radical transformation in communications to the public about addiction, one where they are persuaded to believe that breaking free from addiction is possible and advised about how this can be successfully accomplished.

As for primary prevention and public health, it follows from a recognition that the emergence of addictive behaviours is strongly influenced by environmental factors that, to prevent those behaviours from occurring, we can vary the environmental conditions in question. Behavioural economics is again useful here. Tucker and colleagues (2017) have explained how manipulations of 'the architecture of choice' can help people that make choices that are in their best interests. They maintain that choice architecture strategies implemented within healthcare systems and communities have greater potential for impact on the population that individually-based clinical treatments. Such strategies are entirely consistent with a new paradigm founded on the premise that addictive behaviour reflects biased choices.

The Addiction Theory Network

In case it is thought that the author is a lone voice in thinking that a paradigm shift in addiction science might be possible, the existence of the *Addiction Theory Network* (ATN) shows otherwise. In February 2014, the journal *Nature* published an editorial concerned with the attempt by animal rights activists to close down addiction research laboratories conducting experiments on animals (Animal Farm, 2014). The editorial also stated that drug addiction was "a chronic relapsing disease that changes the structure and function of the brain" and that this was not "particularly controversial, at least among scientists" (p. 5). Derek Heim (2014) wrote a letter to the journal protesting against these assertions and obtained signatures of 94 addiction scholars and researchers from around the world. Heim's letter disagreed with 'the one-dimensional portrayal of addiction' in the editorial and its claim that this was uncontroversial among scientists. He also argued that "substance abuse cannot be divorced from its social, psychological, cultural, political, legal and environmental contexts: it is not simply a consequence of brain malfunction" (p. 40).

Subsequently, with assistance from the present author, Heim contacted the signa-

tories to his letter to Nature to ask whether they would be interested in joining a group, to be known as the Addiction Theory Network, with the aims of opposing the dominant influence of the BDMA and collaborating to develop alternative ways of understanding and responding to addiction. A good proportion agreed and many others have subsequently joined the network. At the time of writing (20 September, 2021), membership stands at 222 from all round the world. The network activity consists mainly of a google group https://groups.google.com/forum/#!forum/addictiontheorynetwork but there has been one pre-pandemic meeting of the network so far, upon which the editorial by Heather et al. (2018) was based. Membership of the ATN is open to scientists, academics, students and practitioners with a *bona fide* interest in addiction and who broadly concur with the aims of the network. Anyone wishing to join should go to Google Groups, search for Addiction Theory Network and apply to join.

The latest development of interest is the production of a book edited on behalf of the ATN and entitled *Evaluating the Brain Disease Model of Addiction* (Heather et al., forthcoming-a). Several chapters from the book have already been mentioned here. All four editors of the book are members of the ATN. Despite this starting position, however, the editors wished to avoid the charge that the book attacks 'a straw man' and aimed to address a readership of all those interested in fundamental issues about addiction, ranging from theoretical to experimental to practical perspectives. Also, while many of those scientifically or professionally involved in addiction already hold firm views on the BDMA, it was thought that there must be others who were unsure what view to take and this agnostic position was represented too. As well as offering arguments for and against the BDMA, the editors wanted the book to be a source of innovative ideas on the nature of addiction and what should be done about it, ideas that went beyond both criticisms and defences of the BDMA. For these reasons, the book was divided into four sections: For, Against, Unsure, and Alternatives. Each section contains reprints of classic articles in the literature relevant to the evaluation of the BDMA but the majority of chapters are original contributions by leading figures in this field of study. Publication is expected in the first half of 2022.

Conflict of interest

The author has no conflict of interest to declare.

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