EVALUATING THE BRAIN DISEASE MODEL OF ADDICTION

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26
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26
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Debates about the etiology of addiction among researchers and policymakers have a long history and continue to the present day. In contemporary societies within a range of nations, the brain disease model of addiction (BDMA) has received strong support. This has come, in particular, from US agencies such as the National Institute on Drug Abuse (NIDA) and American Society of Addiction Medicine. Moreover, there continues to be a significant investment in addiction neuroscience research globally. However, the views of addiction treatment providers about the BDMA, and its clinical impact and relevance, are often relegated to the background when lively debates led by public health researchers and neuroscientists dominate discourse about the neurobiology of addiction. In this chapter, we start by providing a brief history of the biomedicalization of addiction. Moving beyond the binary question of 'Is addiction a brain disease, or not?', we then summarize addiction treatment providers' views about the BDMA and its impact on clinical practice and care. We turn next – and in view of treatment providers' variable engagements with the BDMA – to discuss how the delivery of addiction treatment may be affected. Further, drawing on recent critical drug studies scholarship, we critique how a simplistic, linear 'bench-to-bedside' model of addiction neuroscience translation elides the role treatment providers play in translating neuroscience. Finally, we consider the effects of how the enactment of addiction as a brain disease within policy impacts treatment, and how addiction might be enacted in other ways in future policy frameworks.

The biomedicalization of addiction

The framing of addiction as a 'disease' in general – and a 'brain disease' more specifically – has been longstanding across many nations. As Campbell (2007) argues, a disease framing not only brings alcohol and other drug problems under the purview of medicine, but also raises a number of complex questions about the ontology of addiction. In particular, is addiction a disease of the will, a disease of desire, a problem of social contagion, a biochemical imbalance, or a brain disease? Is addiction chronic, acute, lifelong, or episodic? These often-competing concepts, or *governing images* (Room, 2001) that influence how addiction is viewed and treated have come into (and out of) focus since the late 18th century. Moreover, the very existence of the book in which this chapter appears reconfirms how controversial questions about the nature of addiction remain.

Within addiction treatment settings, the evolution and circulation of the disease concept has a long history (Campbell, 2007; Courtwright, 2012). After witnessing the damaging effects of alcohol in the early 19th century, physicians, including Benjamin Rush in the USA and Thomas Trotter in England, developed what is now understood as the

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foundation of the modern disease model of alcoholism (Berridge, 2013; Edwards, 2012; Levine, 1985). For Rush, alcoholism was a 'disease of the will' that resided within the individual and impaired their ability to control the consumption of alcohol (Berridge, 2013; Levine, 1985). This early disease model formulation occurred at a time when there was a cultural emphasis on abstinence and temperance. It was not until the late 19th century that the disease concept gathered momentum among clinical and scientific audiences, when early clinical studies sought to gather evidence to prove just what type of disease addiction might be (Campbell, 2007).

A well-developed historiography has focused on how the 20th century USA witnessed, at first, a radical *de*-medicalization of addiction. This included the replacement of medical approaches to 'inebriety' and opiate abuse with legal tools such as prohibition and the criminalization of drug use (Campbell, 2007; Gerstein & Harwood, 1990; Levine, 1985; Musto, 1999). In the mid-20th century, the repeal of national alcohol prohibition, and a 'rediscovery' of the disease concept, were attributed to a confluence of social and political factors. Among these were: (i) the growth of the Alcoholics Anonymous movement founded in 1935; (ii) the establishment of the Yale Center of Alcohol Studies in the 1930s; and (iii) the publication of Jellinek's (1960) widely cited text *The Disease Concept of Alcoholism*, which was heavily influenced by Alcoholics Anonymous (Conrad & Schneider, 2010; Levine, 1985).

In the 1960s and 1970s, an emerging "neurobiological style of thought" (Vrecko, 2010, p. 56) increasingly pervaded addiction medicine within the USA and a range of other countries. Addiction research and treatment became concentrated on the brain and its dysfunction (Vrecko, 2010). Continuing into the 1980s, this reflected a wider ascendancy of neurotechnologies and of neurobiological ways of knowing the self and others (Pickersgill, 2010). Neuroscience was commonly cast by social actors within and beyond biomedicine as representing an optimistic future of medical discovery, with then-US President George H. W. Bush signing a presidential declaration to commit the 1990s to be the 'Decade of the Brain' (Goldstein, 1994).

After the mid-1990s, a number of prominent US research agencies – most notably NIDA – enrolled insights from neuroscience (especially neuroimaging) to stake the claim that alcohol and other drug addiction was a chronic, relapsing *brain disease* (Leshner, 1997; Chapter 2, this volume; Volkow, 2005). Whereas previously viewing addiction as a disease had located the problem of addiction within the individual, without providing a unitary mechanistic explanation, the BDMA specifically located the mechanistic cause of addiction within the brain (Volkow et al., 2016; see Chapter 3, this volume). During the 1990s, NIDA funded more than 85% of global research on alcohol and other drug addiction (US Government Printing Office, 1998); in this time, and subsequently, it has been a vocal advocate of the BDMA. Organizations representing treatment providers, including the American Society of Addiction Medicine (2011) and the Australian Medical Association (2017), have also been proponents of the BDMA.

NIDA's strong policy support for the BDMA rekindled an impassioned debate about the etiology of addiction (Hall et al., 2015; Chapter 10, this volume; Volkow et al., 2016). Debates between researchers, clinicians, and policymakers concerning the underlying etiology of addiction that played out in the first days of the 'disease model controversy' (Shaffer, 1990), were revived and emboldened through the 'brain disease model controversy' (Barnett, 2020). These continue to the present day. For example, in the public sphere we have seen TED Talks by prominent addiction clinicians, such as psychiatrist Sally Satel (2019), and best-selling books by Gabor Maté (2011) and Marc Lewis (2015), arguing against the notion that addiction is a brain disease (see also Chapter 13, Chapter 19, and Chapter 37, this volume). Leading advocates of the BDMA, including Alan Leshner (1997), and Nora Volkow and colleagues (2016), have been opposed by others arguing for a public health approach to addiction treatment (e.g., Hall et al., 2015; Heyman, 2009; Kalant, 2010). A two-volume special issue of *Neuroethics* (see Snoek & Matthews, 2017) was dedicated to the debate, and newly formed research groups

have galvanized to oppose the BDMA, exemplified by the Addiction Theory Network (Heather et al., 2018).

In Amsterdam, on January 9, 2018, two of the most prominent advocates for and against the BDMA, Nora Volkow and Marc Lewis, held a lively public discussion about its merits (SA Drug Policy Week, 2019). For some, this had the potential to be a crescendo moment, when opposing parties could resolve (or at least address head on) central concerns within the debate about the BDMA. Finally, Volkow, the head of NIDA, could explain, advocate for, and defend NIDA's long standing, strategic support for the BDMA, against counter arguments led by Lewis and other vocal clinicians and researchers. Of course, it was no such moment of resolution. Like other debates of its kind, it replayed familiar themes of 'What is a disease?', and included an exchange of views about whether the neuroscientific evidence supported viewing addiction as a brain disease or other concern, such as a neurodevelopmental disorder. As Edwards (1970, p. 161) reflected half a century ago about alcoholism:

> Much of the necessary evidence on which to make a decision as to whether alcoholism is a disease is not yet available, and when all the relevant information on the causes of abnormal drinking has been gathered in, the decision as to alcoholism being a disease will still rest very much on the definition of "alcoholism" on the one hand and of "disease" on the other.

In much the same way, debates about the BDMA are likewise deadlocked by the definition of 'disease' in general, and 'brain disease' specifically, and arguments about the underlying neuroscience of addiction. There has been a tendency among many to frame the debate about the BDMA in binary terms: 'Is addiction a brain disease, or not?'

Such a dichotomy risks eliding how and why treatment providers – e.g., physicians, nurses, social workers, case workers, workers with lived experience – adopt different models of addiction (and, indeed, of disease) and engage with neuroscience in diverse ways in practice. Consequently, the aims of this chapter are twofold. First, we summarize treatment providers' views about the disease and brain disease models of addiction, and their impact on clinical practice and care. Second, we reflect on the implications of treatment providers' engagements with the BDMA for addiction treatment, research, and policy.

Treatment providers' perspectives on the disease and brain disease models of addiction

A review of treatment providers' perspectives on the (brain) disease model of addiction identified 34 studies published from 1969–2015 (Barnett, Hall et al., 2018a). These were mainly conducted in the USA, and focused on views about the disease model, with only a few recent studies exploring perspectives on the brain disease model specifically. The motivations for the research reviewed by Barnett and colleagues were wide-ranging. For example, in the USA during the 1970s, many returning Vietnam War veterans were experiencing concerns with alcohol use. Research regarding US treatment providers' views on whether alcoholism was a disease with a medical solution, or a moral failing requiring a legalistic remedy, had implications for how veterans returning home would be treated (e.g., Knox, 1971). More recently, there have been studies that have explored the extent to which the disease concept is supported among treatment providers in countries beyond the USA, where the disease concept has not been central to addiction policy (e.g., for work in Sweden, see Palm, 2004; see also Chapter 36, this volume).

High-quality studies using validated measures largely indicate that treatment providers may support the disease concept and moral, free-will or social models simultaneously (Barnett, Hall et al., 2018a). Others (e.g., Karasaki et al., 2013) have suggested that different models, including the (brain) disease, moral, or psychosocial models, may be supported simultaneously by treatment providers as part of a 'hybrid approach' to understanding drug use and addiction. This mirrors the ontological bricolage noted for other professional groups, as they patch together different – and sometimes ostensibly

incompatible – models of self and others to better understand their work and interpersonal relationships (Pickersgill et al., 2011). In certain clinical circumstances, strategically deploying a disease model, for example by making a person's addiction problem an issue of brain dysfunction, rather than an issue of moral turpitude, may be viewed by treatment providers as a valuable instrument. Framing a client's condition as a disease may indeed 'de-responsibilize' a client for their condition via the adage of 'It's not *me* that's the problem, but rather, my *brain*' (cf., Rotgers, Chapter 16, this volume).

Shedding light on the complexity of views across clinical workforces about the etiology of addiction, there are patterns between treatment providers' demographics and backgrounds, and their perspectives on what underlies addiction (Barnett, Hall et al., 2018a; Barnett, O'Brien et al., 2020). In particular, support for the disease model has been associated with treatment providers' age, year of qualification, being in recovery from an addiction problem, and, especially, whether treatment providers had a personal history of attending 12-step programs. The disease concept predominates within 12-step programs such as Alcoholics Anonymous and Narcotics Anonymous (Levine, 1985); this ubiquity means that clinicians in recovery and who have themselves attended 12-step programs potentially import the disease concept into their own clinical practice.

There is growing evidence to suggest that viewing addiction as a brain disease is higher among US treatment providers in comparison to their counterparts in many other countries (Barnett, O'Brien et al., 2020; Russell et al., 2011). We speculate that there are several reasons that account for much of this difference. Historically, the (brain) disease model of addiction has been anchored in US approaches to research and treatment, and a cultural focus on the effects of alcohol and other drug use on the individual. Relatedly, higher support for the BDMA in the USA may be explained in part by the strong support for it in US drug and treatment policy (e.g., American Society of Addiction Medicine, 2011). High levels of exposure to policy messages in support of the BDMA might be expected to enjoin US treatment providers to view the brain disease model as offering some of the best prospects for treatment and client outcomes (although we would of course not expect that these messages would straightforwardly and unilaterally *determine* treatment providers' perspectives).

Treatment providers' perspectives on the clinical impact of viewing addiction as a brain disease

In addition to exploring treatment providers' perspectives on whether addiction is a disease, their views on the impact of the BDMA for client behavior and clinical practice have also been explored. The perspectives of treatment providers about the potential clinical implications for clients of framing addiction as a brain disease vary (Barnett & Fry, 2015; Bell et al., 2014). On the one hand, some providers consider that, if clients understand their addiction as a brain disease, it might reduce the stigma they experience by framing their behavior as a medical rather than moral issue, reduce guilt, increase insight into drug use, and increase treatment-seeking behavior. On the other hand, viewing addiction as a brain disease is viewed by others as potentially increasing stigma (by, for example, characterizing people as dangerous or out of control), and undermining a client's sense of personal responsibility or increasing their sense of helplessness in recovery (cf., Morris, Chapter 18, this volume).

Further, there is a growing body of empirical research examining the impact of neuroscientific framings of addiction on how people view their own and others' sense of self-efficacy and free will. For example, Australian smokers who agreed smoking was a brain disease were more likely to report an intention to use cessation medicines and had higher quitting self-efficacy than those who disagreed (Morphett et al., 2017). Another study found that being exposed to addiction neuroscience information may have only modest effects on how people view other addicted individuals' levels of free will and responsibility for their behavior (Racine et al.,

2017). Related research exists which examines the association between biogenetic explanations and stigma toward people with mental illness and addiction (Kvaale et al., 2013; Lebowitz & Ahn, 2012). For instance, although more of the public sampled in the US between 1996 and 2006 embraced a neurobiological understanding of mental illness (including alcohol dependence), a neurobiological understanding was not related to a reduction in stigma toward people with mental illnesses (Pescosolido et al., 2010). Future empirical research examining the effects of viewing addiction as a brain disease on people's sense of free will, responsibility, and the stigma they may experience will be important to ascertain whether treatment providers' concerns regarding the BDMA's effect on client behavior are realized in the future.

Treatment providers' own perspectives about the etiology of addiction may also affect how treatment begins, what it looks like, and how it is limited or enabled. There is evidence that providers who support the disease model of alcoholism are more likely to: (i) insist on abstinence as the only treatment goal (Hshieh & Srebalus, 1997); (ii) refer clients to Alcoholics Anonymous (Casswell & McPherson, 1983); and, (iii) impose their own treatment goals on clients rather than incorporating those of the client (Moyers & Miller, 1993). They are also less likely to consider controlled drinking as a treatment option (Moyers & Miller, 1993). Treatment providers in Australian research have also raised the concern that practice informed by a brain disease model may ignore psychological and social factors that require attention during treatment (Barnett & Fry, 2015; Bell et al., 2014).

Importantly, though, treatment in clinical practice is rarely – if at all – viewed exclusively through the lens of neurobiology or a brain disease (at least in Australian treatment settings; Barnett, Pickersgill et al., 2020; Meurk et al., 2016). Rather than being informed by a 'brain disease view', our qualitative work in Australia found that treatment providers engage in a process of 'selective neurologization' (Barnett, Pickersgill et al., 2020). Treatment providers are strategic and selective in their deployment of neuroscientific representations with clients, in order, for example, to foster client optimism or reduce self-blame, enact drug use as a high-risk practice, or to explain complex psychopharmacology.

Rather than overtly discussing addiction in terms of a 'brain disease', neuroscientific terms are often communicated using highly metaphorical storytelling. For example, we traced how the concept of the brain potentially becoming 'hostage' to drug use was strategically deployed in an attempt to discourage youth from using alcohol and other drugs. In another example, a 'lock and key' metaphor was viewed as having explanatory value when describing the complex psychopharmacology underlying pharmacotherapies (e.g., opioid replacement therapy) (Barnett, Pickersgill et al., 2020). These processes of 'neural imagining' (Buchbinder, 2015) afford flexibility in the way the brain is represented to clients and allow treatment providers to tailor science communication for therapeutic gain in different circumstances and for varying audiences. Hence, rather than care being straightforwardly neuroessentialist and narrowly focused on pathology through the prism of the BDMA, our work has highlighted how treatment providers in Australia are agile in invoking neuroscience for what they perceive to be therapeutic gain.

Having considered treatment providers' perspectives on the BDMA, its clinical impact, and how they engage with neuroscience in practice, we now consider the implications of these views for three domains: addiction treatment, research, and policy.

Implications for treatment

An important consideration for those responsible for designing treatment systems is how differences in treatment providers' perspectives on the etiology of addiction affect healthcare delivery and clients' experience of care. Let us assume that providers' divergent views on the causes of addiction shape their care practices, with the result that some treatment approaches are favored over others (e.g., pharmacotherapies, social approaches). As a result, clients accessing treatment may be presented with

multiple, even contradictory, views about the factors underlying their drug problems and how to treat them. One avenue to address treatment providers' multiple ontologies of addiction is for policymakers responsible for service design to consider standardizing care via the implementation of an overarching, universal addiction treatment model (Barnett, Hall et al., 2018a, 2018b). One way to implement this may be to: (i) use standard intake and assessment systems across a treatment sector; (ii) standardize interventions and map client treatment pathways; and (iii) ensure treatment approaches are harmonized across a treatment sector (e.g., by following a harm reduction approach).

However, this type of harmonization of addiction treatment practices potentially has a range of negative, unforeseen consequences. Savic and Lubman (2018) have made a compelling case against the concept of treatment 'standardization' based on three factors: (i) attempts to translate a standardized treatment model into practice would be challenging; (ii) a 'one-size-fits-all' approach would not account for people's complex needs in treatment; and (iii) implementing an overarching model would paternalistically rely on 'expert' knowledge at the expense of consumer participation and client-centered care.

Informed by debates and practices in Sweden, Storbjörk (2018) also advocated for the maintenance of multiple treatment models in practice when arguing that different conceptions of addiction within treatment settings may facilitate better matches between service users with different needs and treatment providers. Storbjörk discussed the contested medicalization of addiction in the 'non-medical stronghold' of the Nordic countries (see Chapter 36, this volume). As she detailed, in Sweden, key stakeholders within addiction treatment have resisted a wholly biomedical understanding of alcohol and other drug problems, with social models of addiction historically engrained within treatment settings. Taking a US perspective, Schmidt (2018) argued that moralized formulations of the disease concept were present in Benjamin Rush's original formulation of alcoholism as a 'disease of the will' (Levine, 1985; Schmidt, 1995), and continue to pose problems for clients because they have never fully dropped away (e.g., leading to stigma which deters people from accessing treatment). Schmidt argued this was problematic because addiction treatment stakes its legitimacy on evidence-based medical science rather than ideological systems of belief which remain pervasive within treatment.

Consequently, as treatment settings across the world design the best services they can (often with reduced funding) to reduce harms from alcohol and other drug use, this "persistent contradiction" (Schmidt, 2018, p. 723) in the ways treatment providers view the etiology of addiction remains contentious. We might ask, though: does this *really* matter? One answer to this question is that different treatments and responses based on diverse social, psychological, and biomedical models can (and need to) co-exist, particularly given that biopsychosocial factors are so intertwined for people experiencing addiction problems. Clients in treatment may benefit both from social interventions (e.g., built on a Social Identity of Model of Recovery approach: see Best et al., 2016, and Chapter 43, this volume), and biomedical interventions when required (e.g., pharmacotherapies). Indeed, tailoring and combining treatment offerings according to client need is considered important in articulations of holistic, person-centered, and integrated care (Savic, Best et al., 2017). Going forward, it is vital to invest in research about how to integrate complex, contemporary addiction neuroscience into practice – regardless of whether treatment providers support the BDMA.

Implications for research

The global pipeline of addiction neuroscience research has advanced understandings of the effects of alcohol and other drugs on the brain considerably over the past few decades. From the 1980s to the beginning of the 21st century, there was a rapid increase in the number of published addiction neuroscience studies (Netherland, 2011). However, it has been argued that the translation of neuroscientific knowledge to practice has been, at best, underwhelming (Kalant, 2010). In NIDA's 2016–2020

strategic plan, the failure to translate neuroscience to practice is explicitly acknowledged as a "bench to bedside gap" (National Institute on Drug Abuse, 2016, p. 5).

Implicit in the notion of *clinical translation* is the idea that science should form the basis of clinical practice (Martin et al., 2008). However, scientific knowledge is socially organized and embedded within local epistemic communities, and – often messily – intertwines with pre-existing clinical practice, with the work of the clinic also powerfully shaping research agendas (Martin et al., 2008). Our own work has also contested the oversimplified notion that addiction neuroscientific knowledge linearly forms the basis of clinical understandings. For example, treatment providers' representations of the brain to clients using techniques like 'neural imaginaries' (Buchbinder, 2015) are applied for strategic purposes (Barnett, Pickersgill et al., 2020). Neuroscience is only occasionally – and strategically – discussed by treatment providers with clients in order to create optimism about recovery, while at other times the relevance of neuroscience for clients is questioned. Future aspirational narratives of 'bench-to-bedside' translation run the risk of ignoring the complex ways in which addiction treatment providers engage with neuroscience and represent the brain in the clinic. They also risk eliding the multiple ways in and degrees to which neuroscientific ideas instantiate in practice with different effects.

Critical drug studies scholarship drawing on perspectives from science and technology studies (STS) provides fruitful new ways of critically engaging with neuroscientific ideas and interventions, and their translation into clinical practice (e.g., Farrugia & Fraser, 2017; Fraser, 2013; Fraser et al., 2014; Fraser et al., 2018; see also Keane et al., Chapter 35, this volume). Broadly speaking, STS is concerned with how science, knowledge, and technology are produced, and how they shape – and are shaped by – the world. STS scholarship consequently provides the theoretical tools for attending to how neuroscience – or any forms of knowledge – circulate and materialize in practice.

One useful STS conceptual tool in this regard is what has been coined an *evidence-making intervention approach* (Rhodes & Lancaster, 2019). Rather than viewing evidence (or ideas for that matter) as being adopted into practice in a simplistic linear fashion (as imagined in evidence-based medicine), the two key aims of the evidence-making intervention approach are: (i) to understand how an intervention (e.g., a therapy; biomedical treatment) is constituted through frictions between the different forms of knowledge that make it; and (ii) to make visible the multiple lived effects of health interventions in how they form local bio-social subjectivities and how they shape localized 'ecologies of care' (including those potentially unforeseen by an intervention's evidencing elsewhere). Applying the principles of an evidence-making intervention approach (Rhodes & Lancaster, 2019), neuroscientifically informed techniques of addiction medicine arise from the intersection of different discourses (e.g., neuroscientific, recovery, moral), practices, and human and non-human actors, and emerge in varying ways in different local settings (Barnett, Dilkes-Frayne et al., 2018). Furthermore, neuroscientific enactments of addiction – and how they give rise to different bio-social subjectivities – differ in varying clinical contexts. We found that when the brain is discussed selectively and in different ways with clients (Barnett, Pickersgill et al., 2020), the types of client subjectivities that emerge may vary. For example, discussing the purportedly damaging effects of alcohol highlighted clients' future risk of pathology and acted as a deterrent to drinking. In other examples, deploying the concept of neuroplasticity had the effect of producing optimism about recovery after stopping or reducing drug use. Just as Rhodes and colleagues (2016) observed that addiction science as a biomedical object does not have stable, universal effects in all settings, neither does addiction neuroscience as a neurobiological object have stable effects in local care settings.

In the context of NIDA's acknowledged 'bench-to-bedside' translational failure of addiction neuroscience to practice, future research may benefit from drawing upon theoretical toolkits provided within

STS literature, such as an evidence-making intervention approach (Rhodes & Lancaster, 2019). An evidence-making intervention approach provides the theoretical springboard for an analysis that is sensitive to how different actors (e.g., treatment providers, patients, education resources, policy instruments) engage with the neurosciences, with what effect, in different care settings. The current, narrow focus on simplistic linear translational models means that the many potential unintended uses and effects of addiction neuroscience within clinical practice continue to remain underexplored. Moreover, in unraveling how the neurosciences instantiate within, and impact, clinical practice, the critical STS literature has thus far predominantly focused on countries, including Australia, Canada, and Sweden (e.g., Barnett, Pickersgill et al., 2020; Farrugia & Fraser, 2017; Fraser et al., 2014; Fraser et al., 2018; Meurk et al., 2016). This has led to a gap in the critical literature on how the neurosciences impact care, particularly in the USA, where the brain disease model remains at the forefront of treatment policy and a vast investment in addiction neuroscience prevails.

Implications for policy

Agencies designing national drug and alcohol policy, including in Australia, the UK, and the USA, will further benefit by considering how addiction treatment workforces view the etiology of addiction, in particular their views about the BDMA. Support for the BDMA has been particularly prevalent in US national drug policy (Office of National Drug Control Policy, 2016), and in agencies that represent US treatment providers (American Society of Addiction Medicine, 2011). It has also been emphasized by research agencies in the USA that have significant global influence on investment in addiction neuroscience research (e.g., NIDA).

Taking a pragmatic approach, two key questions remain: (i) what are the roles of agencies representing addiction treatment providers across the globe in regards to issues like the BDMA?; and (ii) in the formation of policy positions, should agencies reflect the views of their members (a 'bottom-up' approach), or aim to lead what they believe to be best practice in order to influence their members' practice (a 'top-down' approach)? If agencies representing treatment providers aim to take a bottom-up approach by representing their members' views, agencies in, for instance, Australia and the UK should exercise caution when issuing statements in support of the BDMA, as their members may have less positive views about the brain disease model compared to their US colleagues. Conversely, if agencies aim to take a top-down approach by advocating for what they believe as effective evidence-based policy to influence members' practice, they should be aware that treatment models may be selectively adopted (or avoided) in practice. Moreover, the potential risks and benefits that treatment providers raise about the BDMA (Barnett & Fry, 2015; Bell et al., 2014) for client outcomes and clinical practice must be more readily considered to inform policy design.

Beyond these pragmatic questions, there has been a move within critical drug scholarship to unsettle more conventional notions of alcohol and other drug policy-making and its effects. Productively drawing on Bacchi's policy analysis methods (Bacchi, 2009), it has been argued that rather than responding to a stable, pre-existing 'problem', policies *problematize* (or enact) alcohol and other drug concerns in different ways through proposed responses or 'solutions' (e.g., Fraser & Moore, 2011; Lancaster & Ritter, 2014). Such problematizations within policy may benefit some while harming others, and influence how people are governed. In our own work, informed by STS perspectives and Bacchi's policy analysis toolkit (Bacchi, 2009), we traced how neuroscientific discourses influenced Australian alcohol and other drug treatment (Barnett, Dilkes-Frayne et al., 2018). We found evidence that neuroscientific discourses familiar to the BDMA have the effect of directing treatments to target the brain, rather than encouraging social interventions to address addiction.

Importantly, policy-making has vital, real-world implications (for Bacchi, 2009, p. 15, "lived effects"), including downstream implications for the provision of treatment, access to resources, and clients'

experiences of care. In view of this, policy statements in support of a narrow brain disease view of addiction (e.g., Australian Medical Association, 2017) require a detailed, considered appraisal. These types of policy statements are not merely responding to a problem, but rather enact alcohol and other drug problems in ways that directly influence treatment and the ways in which people who purportedly experience 'problems' are viewed (e.g., as 'addicted', 'abnormal', 'normal'). As others have argued (e.g., Bacchi, 2009; Savic, Ferguson et al., 2017), we suggest that a critical reflexivity in policy-making would be useful to: (i) attempt to avoid potentially harmful lived effects of policies and narrow enactments of addiction as a brain disease; (ii) pay attention to the complex array of biopsychosocial aspects of addiction relevant to treatment; and (iii) consider different and new possibilities for engaging with addiction and constructing novel, holistic treatment systems.

Conclusion

Within a lively ongoing debate about the BDMA, the voices of treatment providers remain underappreciated. Their views have important implications for how treatment-seekers experience care. The oversimplified, linear 'bench-to-bedside' notion of addiction neuroscience translation elides the pivotal roles treatment providers play in engaging with neuroscientific concepts in practice. Furthermore, policymakers shaping and steering national drug policy and treatment provider agencies should consider the variable views of treatment providers about the BDMA and its implications for practice. Scholarship from critical drug studies, including STS approaches, may open up new possibilities to explore how treatment providers' engagements with neuroscience can contribute to the design of holistic treatment systems and effective alcohol and other drug policy.

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