Neuroethics (2017) 10:71–78
DOI 10.1007/s12152-016-9285-4

ORIGINAL PAPER

What Is Wrong with the Brains of Addicts?

Edmund Henden · **Olav Gjelsvik**

Received: 30 August 2016 / Accepted: 24 October 2016 / Published online: 15 November 2016

Abstract In his target article and recent interesting book about addiction and the brain, Marc Lewis claims that the prevalent medical view of addiction as a brain disease or a disorder, is mistaken. In this commentary we critically examine his arguments for this claim. We find these arguments to rest on some problematical and largely undefended assumptions about notions of disease, disorder and the demarcation between them and good health. Even if addiction does seem to differ from some typical brain diseases, we believe contrary to Lewis, that there are still good reasons to maintain its classification as a mental or behavioral disorder.

Keywords Addiction · Disease · Disorder · Dysfunction · Irrationality · Normativity

The answer neuroscientist Marc Lewis gives to this question is: *nothing*. In his recent interesting book about addiction and the brain, he argues that the prevalent medical view according to which addiction is a chronic, relapsing brain disease, is mistaken. Even if addiction changes the brain both functionally and structurally, these changes do not imply the presence of disease. Rather, they are the same changes we see in normal learning and development when we repeatedly pursue highly attractive goals. Brains always change with new experiences, whether it's meeting a lover, visiting Paris or taking heroin, and those changes stabilize and consolidate the more the experience is repeated. What is special about addictive goods is that the intensity of the attraction that motivates us to pursue them gives rise to what Lewis calls "deep learning." That is, they lead to the formation of habits – neural and behavioral – that are more deeply entrenched than other, less compelling habits and therefore more difficult to extinguish. Even if they are bad for addicts – they become increasingly compulsive and difficult to control – they do not indicate that there is anything wrong with their brains.

Lewis' book *The Biology of Desire: Why Addiction is Not a Disease* (on which his target article is based) explains in an accessible and engaging manner how addiction changes the brains of addicts. It is a strength of Lewis's approach that he aims to integrate scientific knowledge of changes to the brain into a broader understanding of addiction as a complex human problem requiring multiple levels of analysis, from the neuronal to the environmental and social. Lewis moves beyond the standard dichotomy of a choice model that views addiction as a rational choice (at least in the short run) in response to harmful social or psychological circumstances, and a medical model which sees it rather as exemplifying compulsive and irrational behavior caused by a diseased brain. The problem with the former, Lewis says, is that "it throws out the brain with the bathwater," thus depriving itself of

E. Henden
Centre for the Study of Professions, Oslo and Akershus University College of Applied Sciences, PO Box 4, St. Olavs Plass, Oslo, Norway

E. Henden (✉) · O. Gjelsvik
Centre for the Study of Mind in Nature (CSMN), University of Oslo, Oslo, Norway
e-mail: Edmund.Henden@hioa.no

the necessary resources to explain what is special about addiction, which according to Lewis, is precisely the loss of control and compulsivity, just as proponents of the medical model have argued. This does not mean, he claims, that the medical model gets it right: the problem with the latter is that it assumes that *disease* explains these special features, while in fact they are the outcome of experience and normal learning. Lewis's book can be seen as an important contribution to the dismantling of the problematic dichotomous views of addiction as either choice or compulsion, a dichotomy that has come under increasing criticism in the recent theoretical literature.

We find ourselves in broad agreement with Lewis's description of addiction as a complex human problem in need of multiple levels of analysis, that neither rules out choice and experience nor impaired control and compulsivity, and which importantly involves brain changes that stabilize into deeply entrenched habits that are difficult to extinguish [1–3]. In this commentary we focus on his claim that the view of addiction emerging from this description, *rules out* a disease-based conception of addiction. In defense of his claim, Lewis refers to arguments of a more conceptual than scientific nature since they do not dispute any of the neuroscientific evidence that has led many researchers to claim that addiction is a disease (the disease model is not "so far off base scientifically" as Lewis remarks ([4]:12)) . Rather, what he disputes is the interpretation of this evidence by proponents of the disease model as, in his words, "implying" that something is wrong with the brain ([4]: 7). Of course, "implication" may seem too strong a word in the context since the concept of disease is normative (a point to which we return below) while the relevant neuroscientific and behavioral evidence is couched in non-normative terms. It would thus be a fallacy simply to deduce from this evidence that addiction is a disease. Nevertheless, this does not mean that the evidence cannot in fact *support* the view that addiction is a disease. Whether it does depends, in part, on what the criteria are for something to be a disease, and whether addiction meets them. It is from this perspective we discuss Lewis's arguments against the disease model of addiction.

Lewis' Argument against the Disease Model of Addiction

Discussing his opponents' view of addiction as a brain disease, Lewis cites the principle reason for their conviction: *it messes up brain wiring*. Without delving into the details (which are fascinating enough), what this means according to Lewis, is, roughly, two things: first, addiction leads to a loss of grey matter volume (reduced synaptic density) in the dorsolateral prefrontal cortex (dlPFC), the region of the brain responsible for discrimination, judgment, and conscious self-control – a loss that seems due to a functional decoupling of this region from the striatum, or the "motivational core," i.e., the region responsible for pursuing rewards. This loss corresponds with reduced capacity to engage cognitive control. Second, addiction causes a shift in activation from the ventral to the dorsal striatum (consisting of the growth of fibers from the former to the latter area) in response to drug-associated cues. This corresponds to drug-seeking behavior becoming more compulsive and less impulsive in character. Together, these brain changes explain the loss of control and compulsivity characteristic of addiction, and are taken by proponents of the disease model to be "the golden proof" that addiction is a brain disease ([5]: 168).

Lewis does not dispute the science behind these claims. On the contrary, they give an accurate description of how repeated uptake of dopamine in response to drugs changes the brain, he thinks. So, why are these brain changes *not* evidence of brain disease? Lewis' main argument is based first on the concept of "neuroplasticity," a term used to refer to the brain's capacity to reorganize itself by creating new neural pathways in response to experience, and second a rejection of what he calls "the principle of neuronormativity," whereby "the brain is a normative thing that can go wrong and then be repaired" ([4]: 13). Looked at from the perspective of neuroplasticity, the problem with the disease model is that it takes what is in fact a perfectly natural process of neural change and stabilization in response to the stimulation of learning and experience to be *pathological*, that is, as implying that there is something *wrong* with the brain. But neither reduced synaptic density in the dlPFC, functional decoupling between dlPFC and the striatum, nor ventral-to-dorsal shift in striatal activation, are abnormal features of brains. They are causally involved in all kinds of activities that start off as highly rewarding and end up as behavioral habits, whether they involve becoming absorbed in a sport, joining a political movement, or falling in love. In fact, they can even be seen as *adaptive* since automatization of behavior improves overall cognitive efficiency or, as Lewis puts it,

"[w]e need habits in order to free our minds for other things" ([4]: 7). Research also shows that the brain changes that characterize addiction can reverse over several months of abstinence, and that spontaneous recovery from addiction is common, something Lewis takes as further evidence of the correspondence between these changes "with variations in experience, not disease" ([4]: 9). "There is no clear dividing line between addiction," he concludes, "and the repeated pursuit of other attractive goals, either in experience or in brain function" – even if addictive habits can be more deeply entrenched and cause more suffering than many other habits ([4]: 6). Since "disease" and "normality," he claims, are vague, overlapping categories, there is, therefore, "little benefit in calling addiction a disease" ([5]: 164). Lewis extends this reasoning to rule out a conception of addiction as disorder since, as he notes, the concept of disorder is "a close cousin" of the word "disease," and such labeling only puts us on a slippery slope to classifying all sorts of normal activities "as diseases or disorders" ([5]: 8, 23).

In our commentary, there are three assumptions of this argument we want to focus on. First, what appears to be an underlying normative assumption about the concept of disease. Second, the assumption that if there is no clear dividing line between addiction and some other conditions which are not diseases, addiction cannot be a disease. Third, the assumption that there is no important difference between classifying addiction as a disorder and classifying it as a disease. We find these assumptions to be problematic and largely unmotivated.

Normativity and the Brain

What would have to be the case in order for addiction to be a brain disease? According to Lewis, that "the *kind* (or extent or location) of brain change characteristic of addiction is nothing like what we see in normal learning and development" ([4]: 4). Since, however, the brain change underlying addiction *is like* what we see in normal learning and development, it does not imply the presence of disease, he concludes. But even if a brain change is nothing like what we see in normal learning and development that is not in itself sufficient to show that it is diseased. In the literature on the concept of disease, most theorists see "dysfunction" as its core criterion.[1] That is, they believe one (at least necessary) condition for some physiological change to imply the presence of disease is that it is associated with or involves some "dysfunction." Since "dysfunction" depends on the concept of function, which is a normative concept (in the minimal sense that it determines the way a particular organ or mechanism is supposed to work), the concept of disease is also a normative concept. Something is a heart disease, for example, because it interferes with what the heart is supposed to do (provide a continuous flow of blood throughout the body), and hence deviates from the norms governing the function of hearts.[2] Without norms of one kind or another there would consequently be no way to talk about *diseases*, including *brain* diseases.[3] Now, presumably Lewis does not want to deny the existence of brain diseases (e.g., that Alzheimer's disease, schizophrenia or stroke are brain diseases). What he denies, as we understand it, is a certain interpretation of the norms assumed by his opponents to show that *addiction* is a brain disease, namely the interpretation according to which it is a brain disease because the underlying brain changes by deviating from norms of neural function and standard neural architecture (presumably norms which are intrinsic to the brain and can be specified in purely neuronal terms) constitute some neural abnormality. Lewis's point is that there is no way to determine what these norms are. Moreover, the idea that there even exist such norms seems ruled out by neuroplasticity since this concept implies that the brain is an "open system that can develop in a multitude of directions, integrating the meaning of experience according to its own proclivities" ([4]: 13).

[1] For one very influential version of the dysfunction view, see especially [6].

[2] It should be noted here that there are different ways of interpreting the normativity of the concept of function, ranging from evolutionary to causal-mechanistic ones. For a recent version of the latter, see e.g., [7].

[3] Without norms there would be no way to talk about "learning" either, at least not in the ordinary language sense of the term, as learning in this sense presupposes normative notions like knowledge and skill. Consequently, whether something counts as learning (in the ordinary sense) it will not be something that can be determined purely at a descriptive level, whether neural or behavioral. From this perspective, acquiring an addiction, rather than seeming a case of learning, may seem more like a case of *un*learning or of losing various skills, abilities and knowledge (e.g., skills, abilities and knowledge associated with self-governance).

There are, however, difficulties with concluding from this line of reasoning that addiction is not a brain disease. Rather than showing that the disease model of addiction is mistaken, it might be showing that it is *a particular version* of this model that is mistaken, a version that might depend on an incomplete or even flawed interpretation of the norms that determine what counts as a brain disease. This means that, given a correct and complete interpretation of the relevant norms, it might still be consistent with the available evidence that addiction is a brain disease. In fact, there may be many reasons to be skeptical of a purely "neuronal" interpretation of the relevant norms, reasons which have nothing to do with the plasiticity of the brain. Let us briefly consider two such reasons here.

First, it might be argued that deviation from the norms of neural function and standard neural architecture is at best *insufficient* for determining whether some form of brain change is evidence of disease. Take Alzheimer's for example (a brain disease if there ever was any!). It consists in the buildup of amyloid plaques and neurofibrillary tangles in the brain. But why are these neural changes evidence of *disease*? Presumably, an important part of the answer is that they correspond with mental decline. That is, by working backwards from mental decline to what turns out to be the neural basis of that decline, we can determine whether the neural changes associated with and causally involved in Alzheimer imply the presence of disease [8]. This suggests that whether some brain changes imply the presence of disease is not just a neuronal question (even if it cannot be understood without reference to the neurological). It also depends on norms of good *mental* functioning. Moreover, according to some theorists, any physical state or condition is only evidence of disease if it is harmful for its owner. Judgments of harm, however, cannot be made without reference to *social* norms and values [9]. Whether addiction is a brain disease or not is not, therefore, something that can be determined solely on the basis of evidence from neuroscience. Yet, this appears to be the only evidence Lewis considers to be of relevance.

Second, it might be argued (more strongly) that deviation from norms of neural function and standard neural architecture is *not necessary* for determining whether some brain change is evidence of disease. Thus, some of those who seem to think that addiction is a brain disease appear to base their view on what they take to be evidence of the brain changes underlying addiction deviating from norms of natural (biological) function. According to such aetiological views, functions are determined by the course of natural evolution, by means of variation and selection, and dysfunction occurs when some organ or mechanism fails to play the role for which it was selected in the course of natural evolution. Thus, based on the claim that the dopaminergic system evolved to play a particular role in the organism's adaption to its environment, it has been argued that addiction exemplifies *dysfunction* in this system by causing it to fail to play that role (for discussion, see [10]). There is no need to go into the details of these views here (Lewis touches upon several of them in his book). Suffice it to say that *if* – as seems plausible – natural (biological) dysfunctions need not imply any deviation from norms of neural function and standard neural architecture (that is, natural dysfunctions can occur in *the absence* of neural abnormalities), there might be an alternative route to the conclusion that addiction is a brain disease, a route Lewis doesn't really consider but that does not appear to be vulnerable to his objections against the disease model of addiction.

The point of mentioning these considerations here is only to suggest that determining whether the brain changes underlying addiction imply the presence of disease may involve other norms (as well as values) than those Lewis seems to think are embodied in the principle of neuronormativity. In fact, the way he presents his opponents view, we find it unclear to what extent they are relying on this principle at all. What they appear *to say* is that the brain changes characteristic of addiction imply the presence of disease because they are associated with or involve a "cognitive dysfunction" which they call "impaired response inhibition" ([4]: 3). This, of course, is just another description of the loss of control seen in addictive behavior. However, saying that these brain changes are diseased because they are associated with or involve "cognitive dysfunction" is not the same as saying they are diseased because they deviate from (unspecified) norms of neural function and standard neural architecture. Our point here is not that dubbing the dysfunction "cognitive" shows that addiction *is* a brain disease. Rather, it is that it is unclear whether Lewis, by showing that the principle of neuronormatvity should be rejected (because of neuroplasticity), succeeds in showing that the disease model of addiction is mistaken.

The Demarcation Problem

One of Lewis's prominent objections to the disease model is that "there is no clear dividing line between addiction and the repeated pursuit of other attractive goals, either in experience or in brain function" ([4]: 6). Whether it is the amount of dopamine released, the degree of specificity in what we find rewarding, or the (lack of) availability of top-down cognitive control, these are continuous dimensions, Lewis points out ([5]: 164). They do not, therefore, lend themselves to "two distinct categories like disease versus good health." An example he uses to illustrate this point is falling in love. Looked at from both an experiential and neuroscientific point of view, love has a lot in common with addiction: like addiction, it can easily become compulsive, difficult to control, and overly focused on the immediate, with little regard for long-term consequences. Moreover, mesolimbic dopamine (particularly in the nucleus accumbens region) appears to be a major contributor in both. If addiction is a disease, then so is apparently love, Lewis claims.[4] Spelled out, his argument here seems to be that, because addiction is not clearly distinct (either experientially or neurologically) from some other conditions that are not diseases, addiction cannot be a disease.

We think there are several problems with this argument. It seems to suggest that the distinction "disease versus good health" somehow exists "in nature," independently of normatively identified function, a view we believe is highly problematic. Concepts of function and dysfunction clearly allow for gradation along several dimensions. That is, in some cases it might be difficult to determine whether some mechanism is functioning "normally" or is falling below this level and should count as dysfunctional, i.e., a (possible) pathological condition. Different kinds of norms, values or contextual factors (including social norms and circumstances) might influence this decision. What it suggests is that concepts of function and dysfunction often and typically refer to continuous dimensions, just like "amount of dopamine released" or "(lack of) availability of top-down cognitive control." If "dysfunction" is a core criterion of disease, the implication seems, in other words, to be that there is no exact line between disease and good health. That is, there are *bound* to be grey areas in which some physical changes might not be indisputable disease nor indisputable good health. This seems to be a reflection of the nature of these concepts. The fundamentally important point here is that it may not matter at all. It is simply a version of the "sorites" paradox much discussed in philosophy. If there is no clear dividing line between what is and what is not a heap of sand, are there any heaps of sand? Even if there are things we do not know are heaps of sand or not, this doesn't mean that there are no heaps of sand, nor many things that are not heaps of sand. The requirement of an exact dividing line is misplaced as long as we have very clear cases on either side of a grey area. Even cancer, it might be argued, is like this, as there are many conditions that are neither clearly cancer nor clearly *not* cancer. This does not mean there is no cancer or that cancer cannot be a proper diagnosis. The existence of cancer does not depend on an absolutely exact demarcation. What kind of demarcation criteria to use is ultimately a contextual and pragmatic question, governed by the goal of the demarcation. We cannot, for this reason, see that the absence of "a clear dividing line" between addiction and some other conditions which are not clearly addictions, can provide any evidence that addiction is not a brain disease.

What Is Addiction?

Addiction, Lewis writes, describes "the repeated pursuit of highly attractive goals and the brain changes that condense this cycle of thought and behavior into a well-learned habit" ([4]: 7). We don't disagree. Consistent with this description, one factor that arguably might distinguish addiction from some typical brain diseases is that unlike the latter, *the symptoms* of addiction (such as compulsive drug seeking behavior, obsessive thoughts related to drug-taking etc.) appear to be within the capacity of most addicts to influence through their own decision-making processes, even if doing so might be very difficult ([12]). In contrast to this, it seems well beyond the capacity of most victims of, say, Alzheimer's disease, to volitionally influence the symptoms of *their* condition once it has taken hold (or, at least, it is

[4] How much love really has in common with addiction depends on one's view of what love is. There has been considerable debate about this question in philosophy, e.g., whether love is a moral emotion [11]. A suspicion one might harbor is that when Lewis and fellow neuroscientists talk about love, what they really have in mind is what many participants in this debate would describe, rather, as infatuation or sexual drive, which they distinguish from love. We put this issue aside here.

reasonable to suppose it is substantially *more* difficult for them to do so than it is for addicts).

We are not claiming that this criterion provides a necessary and sufficient condition for distinguishing disease from good health – as already pointed out, we don't think there exists any exact line here, and besides, there are bound to be grey areas when it comes to an individual's capacity to volitionally influence the symptoms of their condition too, regardless of whether the condition is classified as a disease or not (e.g., the victims of some diseases can, to some degree, volitionally influence the symptoms of their disease). What it plausibly does is to distinguish addiction from typical brain diseases like stroke, schizophrenia, or Alzheimer's. In fact, Lewis himself seems to have something like this in mind when he remarks that while effective treatment of addiction "target[s] cognitive and motivational processes such as self-determination, insight, willpower, and self-forgiveness […] no disease […] can be arrested by tapping such processes" ([5]: 169).

Now, given what appears to be Lewis's recognition of addiction as a condition needing professional intervention – he sees it as essentially involving loss of control, compulsive behavior and psychological suffering – it is not entirely clear to us why he refuses to accept its classification as a *disorder* (which, of course, is its current medical diagnosis). The reason he gives in support of this view is that "disorder" is a "close cousin" of the word "disease" ([5]: 8). From this premise and the view that addiction does not involve any neural dysfunction and is therefore not a disease, he seems to infer that it neither can be a disorder. If this is indeed his argument, Lewis appears to think that addiction cannot be a disorder based on an assumption that something can be a disorder only if it involves a specific underlying neural dysfunction. This assumption, however, seems implausible to us. There are two things to note. First, mental or behavioral disorders cannot be specified without reference to the *mental* or the *behavioral*. That is, to determine whether a mental or behavioral process is "disorderly," we must compare it with what we take to be "good" mental or behavioral functioning. This requires normative evaluation; more specifically, an assessment of rationality.[5] Second, even if some disorders might involve neural dysfunction (and even *be* brain diseases), it does not seem plausible that something cannot be a disorder unless there is some specific underlying neural dysfunction. It follows that even if addiction is not a brain disease (because it does not involve any specific neural dysfunction), it might still be correctly classified as a mental or behavioral "disorder."

Regarding the first point, Lewis seems to want to rule out irrationality as evidence of disorder: "to say that addiction isn't rational is just stating the obvious," he notes, "[t]hinkers from Homer to Dennett and writers from Shakespeare to Nabokov have made abundantly clear that irrationality is an essential feature of being human" ([5]: 29). But even if irrationality might be "an essential feature of being human," that doesn't mean that *specific forms of irrationality* cannot provide evidence of disorder. In fact, a closer look at the current versions of DSM or ICD reveals that assumptions about rationality and irrationality are an important part of the background for attributions of disorder. Without such assumptions it is hard to see, for example, how to make sense of notions such as "impairment in reality testing," "magical thinking," "suspects without sufficient basis that others are exploiting, harming or deceiving him or her," or "worry about every day, routine life circumstances" etc. [14].

So when does irrationality provide evidence of disorder? One plausible view might be when it takes such forms and reaches such magnitudes that it disrupts the person's ability to interact with other people and lead an ordinary life, e.g., by functioning normally in their social roles and meeting their obligations at school, work, and home [15]. That is, when it causes severe impairment in psychosocial functioning, and professional intervention is needed. There is nothing in this picture that suggests that milder forms of irrationality cannot still be common among people who *don't* suffer from disorders.

Irrationality is, of course, widely believed to be a characteristic of addiction. Addicts find it extremely difficult to revise or abandon their drug-oriented decision-making pattern even if they are given good and sufficient reasons to do so. It is this amazing lack of reasons-responsiveness and drastically diminished ability to change, even if the consequences are severe suffering and impaired functioning, that in our view makes it legitimate to speak of addiction as a "disorder." This brings us to the second point above, the view that something cannot be a disorder unless there is a specific underlying neural dysfunction. This view relies on a questionable form of "reductionism" about the concept

[5] For a defense of the view that mental illness involves violations of rationality, see [13].

of disorder. A familiar analogy illustrates why it is questionable: software problems are neither the same as, nor even dependent upon, hardware problems. If the mental is a matter of information-processing systems, as in the standard picture of cognitive psychology, the mental problems underlying disorders might be due to malfunctions in mental "software," rather than dysfunctions in neural "hardware." Moreover, such mental malfunctions can occur in the absence of dysfunctions in neural "hardware" (which does not have to rule out that the interaction between a particular neurological change and mental or rational contents plays an important causal role!). This analogy relies, of course, on a certain view of the mental (which has been making increasing inroads into psychiatry), but there are other ways to reach the same conclusion which do not rely on this particular view [16]. We cannot address the huge topic of reductionism about the mental (or mental problems) here. For present purposes, what's important to note is that if Lewis's reluctance to classify addiction as a disorder depends on "reductionist" assumptions about the concept of disorder (as it appears to do), we think it rests on shaky foundations. To successfully rule out that addiction is a disorder he has to provide much more by way of support for this kind of reductionism than he does in his book and target article. In our view, given the current status of the theoretical literature on psychological reductionism, the prospect of finding such support is pretty dim.

Conclusion

Although we agree with Lewis's description of addiction as involving brain changes that stabilize into deeply entrenched habits that are difficult to extinguish, we are not convinced by the arguments he offers against the disease model of addiction. We are even less convinced by the argument he deploys against the view of addiction as a "disorder." This latter argument appears to rely on a particular assumption whereby, for something to be a disorder, there has to be some specific underlying neural dysfunction. It's a problematic idea and seems largely unmotivated. So what is our view of addiction? Addiction is a multi-determined pattern of behavior that varies greatly across individuals in terms of severity and causal influences. At its core, however, is loss of self-control with respect to drug-oriented choices and actions. In line with current diagnostic practices, when an addictive pattern of behavior continues despite adverse consequences, e.g., severe psychological suffering, impairment of social functioning, and despite a strong motivation to stop, there is, we believe, a legitimate reason to use the word "disorder" about it. We are torn on the question of whether addictions that satisfy this condition are also brain diseases. While EH, for reasons hinted at in the previous section, is inclined to think they are not, OG believes the concept of "disease" is itself in need of revision which might allow that they are. Ultimately, whether addiction is a "disease," "disorder," or something else, cannot be decided until we agree on what these crucial terms mean. To this end, further conceptual and philosophical analyses and arguments are needed. At present none of these crucial terms appear to have satisfactory definitions in medicine and psychiatry or indeed the philosophy of medicine and/or psychiatry that are not riddled with controversy.

References

1. Henden, E. 2013. Addictive actions. *Philosophical Psychology* 26(3): 362–282.
2. Henden, E., H.O. Melberg, and O.J. Røgeberg. 2013. Addiction: choice or compulsion? *Frontiers in Psychiatry* 4(77): 1–11.
3. Henden, E. 2016. Addiction, compulsion, and weakness of the will: a dual-process perspective. In *Addiction and choice: rethinking the relationship*, eds. N. Heather and G. Segal. Oxford: Oxford University Press.
4. Lewis, M. 2016. Addiction and the brain: development, not disease, this issue.
5. Lewis, M. 2015. *The biology of desire: why addiction is not a disease*. PublicAffaires New York.
6. Boorse, C. 1997. A rebuttal on health. In *What is disease?* ed. J.M. Humber and R.F. Almeder, 3–143. Totowa, NJ: Humana Press.
7. Lange, M. 2007. The end of diseases. *Philosophical Topics* 35: 265–292.
8. Summers, J. 2012. Review of George Graham, the disordered mind. *Philosophical Psychology* 25(6): 941–944.
9. Wakefield, J.C. 1991. The concept of mental disorder: on the boundary between biological facts and social values. *The American Psychologist* 47: 373–388.
10. Levy, N. 2013. Addiction is not a brain disease (and it matters). *Frontiers in Psychiatry* 4(24): 1–7.
11. Velleman, J.D. 1999. Love as a moral emotion. *Ethics* 109(2): 338–374.
12. Heyman, G.M. 2009. *Addiction, a disorder of choice*. Harvard University Press.
13. Graham, G. 2010. *The disordered mind. An introduction to philosophy of mind and mental illness*. New York: Routledge.

14. Crowe, M. 2000. Constructing normality: a discourse analysis of the DSM-IV. *Journal of Psychiatric and Mental Health Nursing* 7: 69–77.
15. Bolton, D., and N. Banner. 2012. Does mental disorder involve loss of personal autonomy? In *Autonomy and mental disorder*, ed. L. Radoilska, 77–99. New York: Oxford University Press.
16. Davidson, D. 1980. The material mind. In *Essays on actions & events*, 245–259. Oxford: Clarendon Press.