Not in our brains: on the complex relations between biology and behaviour

Abstract

Ordinary behaviour that is not the result of a brain disease has a complex relationship to biological factors. It may be influenced by them, but is not caused by them. In contrast, brain diseases can directly cause certain behaviours, or patterns of behaviour. Mental disorders are best thought of as variants of ordinary behaviour, not as brain diseases.

Awais Aftab’s response to my paper is informative because it expresses what I suspect is a widespread but confused inclination among psychiatrists to ‘have their cake and eat it’. On the one hand they do not want to be branded as reductionists, so they deny they are equating mental disorder with brain disease or brain mechanisms. Yet, they want to maintain a role for physical causation in mental disorder, because without it the link between psychiatry and medicine starts to look tenuous.

Reductionism is the implication of what Aftab is saying, however. By denying any important distinction between behaviour produced by a brain disease and other, non-disease driven behaviour (which I refer to here as ‘ordinary behaviour’), he reveals himself to be, fundamentally, a neurodeterminist.

Aftab emphasises that intentional behaviour and neurological processes can co-exist, but of course no one would argue with this. It is how they relate to each other that is at issue. I take the view espoused by philosopher Peter Hacker, following Aristotle, who emphasises how organisms have ‘powers’ or capacities that depend on the nature of their bodies. Human beings have particularly sophisticated ‘mental’ capacities that enable us to respond in an
intricate and flexible way to the world around us. According to this view, we have agency or individuality, which is a consequence of the particular biological makeup of the human organism (Hacker, 2010).

I am not suggesting, therefore, that self-determined (as opposed to disease-determined) behaviour is immune from biological influence. What I am doing is distinguishing brain disease, which is a specific situation in which there is a direct causal link between a biological process and behaviour, from ordinary behaviour. Although there is a complex interaction between biology and agency, it is wrong to think of ordinary behaviour being caused by a biological process in the same way. Aftab, in contrast, wants to blur this distinction, calling it a ‘false binary.’

I devoted a good proportion of my original paper to the reasons why behaviour is the central issue. I am not denying that we can and do have personal experiences that do not manifest in behaviour, including ordinary sensations, thoughts and moods and less familiar ones such as hallucinations and extreme moods. These are conceptually inextricably linked to outward behaviour, however, in that the constitutive criteria for the application of mental concepts are behavioural. We recognise what we call mental disorders on the basis of particular patterns of behaviour, including speech and communication.

Aftab is correct that brain diseases rarely eliminate agency altogether. The man with the brain tumour I used as an example is still making decisions and undertaking complex actions, just like people with advanced dementia. The disease does not fully determine each decision they make, but it circumscribes these decisions. It causes the individual to show an uncharacteristic pattern of behaviour, which is the result of the brain disease and not an expression of the individual’s agency or character.
To the extent that a behaviour, or pattern of behaviour, is caused by a biological mechanism, it is not intentional or meaningful. Something that is produced by a biological process cannot simultaneously be ‘caused by’ psychological factors (incidentally reifying the ‘psychological’ and speaking of it as a ‘cause’ is part of the conceptual confusion, as I explained in the original paper). Biological processes do not have meaning. They are ‘governed by predictable regularities which… have nothing to do with the desires and purposes of individual human beings’ (Moncrieff, 2020, P 173).

Although Aftab dismisses it as ‘semantic’, much hinges on what is meant by ‘cause,’ and on the nature of the relationship between ordinary behaviour and biology. We are organisms with certain sorts of bodies that have physiological needs and appetites, including hunger, pain and sexual desire. These physiological states are different from meaningful states like beliefs, emotions and moods, however, although meaning may be superimposed onto physiological states, as when we love or care about the object of our sexual desire.

Physiology influences our ordinary behaviour in complex and varied ways.

Anxiety, for example, is a normal feeling that arises in response to a situation we find challenging or threatening. When we experience anxiety, physiological changes occur that are part of what we think of as the ‘feeling’ of anxiety. Anxiety is not initiated by the individual; people do not chose to feel anxious. But it is not caused by our physiology either. Anxiety, like other emotions, is a meaningful response to our environment- it occurs because of our intelligent evaluation of a situation. The way people behave when they are anxious is influenced by the physiological changes that accompany anxiety - difficulty concentrating due to increased arousal, for example. Yet the behaviour arises from the individual and expresses the individual’s beliefs and inclinations. It is not adequately accounted for by the physiological reaction.
People have different proclivities to anxiety and varying capacities for managing their feelings. This variation may be determined in part by our genes and also by our past experiences. This complex relationship between our physiology and our behaviour and the variation between individuals is all part of the nature of ordinary behaviour. It is different from the relationship that pertains between a brain disease and the behaviour that results from it.

Thus, although our biological make-up may influence our behaviour, it does not cause it in the way that the brain tumour causes the abnormal sexual behaviour in the example I used, or the way that dementia changes behaviour. In the case of a brain disease, there is a direct mechanical link between the disease, and its impacts on the structure or function of the brain, and the resulting behaviour. In contrast, ordinary, intentional behaviour comes from the person. It is a manifestation of the individual’s character, that is of their tendencies, susceptibilities, values and purposes. Ordinary behaviour is realised through neurological and physiological mechanisms, and may be impacted by them, but it is not initiated by them or attributable to them.

Even if you want to think of people as having organically determined predispositions to certain inclinations, such as Aftab’s idea that ‘ordinary’ paedophilic behaviour is partially determined by ‘functional configurations of neurological mechanisms’ (a contentious proposition), we still want to distinguish between this and paedophilic behaviour caused by a brain tumour. We might find a neurological network that predisposes people to touch other people’s breasts uninvited, for example, but we would still want to distinguish most incidents of such behaviour from the disinhibited behaviour that can be caused by a neurological disease or a brain injury.
As I said in the original paper, it is perfectly coherent to argue that mental disorders are brain diseases that are different from ordinary behaviour, although there is little evidence or justification for this position. It is common, though in my view incoherent, to argue that all behaviour is causally determined by neurological events, and that human freedom, rationality and responsibility are simply illusions.

It is not clear what view Aftab is espousing, but I think he wants to straddle the two positions, and argue that although mental disorders are not fully fledged brain diseases, they are different from ordinary behaviour, too, in that they are more strongly determined by biological mechanisms. If this is the case, he needs to specify how the relationship between mental disorders and biology is different from the complex relations between biology and ordinary behaviour. If he is suggesting that mental disorders, along with some other situations (possibly those characterised by compulsion, including paedophilia) are distinctive because there is a direct causal association between a specific neural ‘configuration’ and the mental state or behaviour, then it seems to me he is conceptualising them as brain disease. If the association is not causal and specific, then it is not clear how it can be distinguished from the ways in which ordinary behaviour is related to biological and neurological states.

Aftab criticises me for decoupling agency from moral responsibility in relation to mental disorder, though, again, he does not set out his own position. This is indeed a point that needs much fuller exposition. Agency is a complicated concept. In severe cases of mental disturbance, there seems to be agency of a sort, but not necessarily the sort we would credit as rational or ‘competent’ agency. Yet neither, in my view, is it agency that is simply distorted by a physical process, such as a brain disease, or drug intoxication.

I am grateful to Aftab for enabling me to clarify that psychiatrists need to come off the fence. Psychiatric disorders are either brain diseases or they are variants of ordinary behaviour -
‘part of the range of ways that human beings live within, and interact with, their world’ (Moncrieff, 2020, P 178). No other possibility has been put forward by Aftab or anyone else. People with what we call mental disorders are trying to negotiate their individual circumstances in various human ways. They are not walking representations of ‘neurological mechanisms’.

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Footnotes

A reference to ‘Not in Our Genes’ (Rose et al, 1990)

References:


Moncrieff, J. (2020). “It Was the Brain Tumor That Done It!” Szasz and Wittgenstein on the Importance of Distinguishing Disease from Behavior and Implications for the Nature of

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