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Rebuttal to Timimi's article 'Attention-deficit hyperactivity disorder: a critique of the concept'

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The empirical claims made in this critique have already been refuted in my initial position paper, so I focus here on the philosophical assumptions it makes.

Are medical and psychiatric diagnoses fundamentally different?

The critique makes a very strong claim about the ordinary medical diagnosis. Specifically

> In the rest of medicine therefore, my diagnosis explains and has some causal connection with the behaviours/symptoms that are described.

The word 'explains' can, dependent on usage, have three distinct meanings: making something clear, giving a reason, and proposing a cause. Thus, the critique requires that, whatever else a diagnosis does, it must make some comment about causation. We shall see below that this constraint does not apply to medical diagnosis, either historically or currently.

A historical perspective on medical diagnosis

The oldest diagnostic system is from Ancient Egypt and is recorded in the Edwin Smith Papyrus (*Edwin Smith's Surgical Papyrus*, nd). It gives details of assessment and treatment for 48 cases, grouped by three diagnoses: 'An ailment which I will treat'; 'An ailment with which I will content'; and 'An ailment not to be treated'. Thus, the roots of diagnosis lie not with cause, but with prognosis. Even at this early stage, as the case studies show, it was well understood that the purpose of understanding aetiology was to improve treatment, but was not necessary to make a diagnosis. Sir William Osler, who transformed the practice of medicine in the 19th Century, and to whom is attributed such aphorisms as 'there are three important things in medicine: diagnosis, diagnosis, and diagnosis' emphasised that the basis of diagnosis was the direct observation of the patient (Andrews, 2002). He and his colleagues were entirely happy making diagnoses, such as progeria, where the aetiology was completely unknown to them. Instead, the emphasis was on recognition and classification, and this continues to this day. Aetiology is an assistant to treatment, not a diagnosis.

Medical diagnoses of unknown cause

A very common example of such a diagnosis is 'Pyrexia of Unknown Origin'. This has precise diagnostic criteria, which specifically include failure to reach another diagnosis following 1 week of inpatient investigations (Chan-Tack & Bartlett, 2017). Though many alternative diagnoses may be found with further investigation, reports suggest that anything between 5% and 50% of cases have no aetiology identified (Horowitz, 2013). However, the identification is of prognostic value as such cases usually have a good outcome. Rarer examples include complex regional pain syndrome (Borchers & Gershwin, 2014): in this latter case, there is much less clarity over even descriptive characteristics of the diagnosis than with attention-deficit hyperactivity disorder (ADHD), but no-one argues that the disorder does not exist.

Conclusion: psychiatric diagnoses are medical diagnoses

In the World Health Organisation's International Classification of Diseases, psychiatric disorders are merely one chapter, albeit published separately for convenience (World Health Organisation, 1993). There is no fundamental difference of approach: both psychiatric and physical diagnoses are deduced from combinations of signs and symptoms, which are observed in patients. The critique is right in pointing out that psychiatric diagnoses are not perfect, but many medical diagnoses are not either, as anyone who has had a diagnosis of 'viraemia' from their GP will confirm. Sick people are universally recognised as being unable to meet all their usual obligations, and it seems unfair to say that, when a sickness is manifested in thought or behaviour, and we do not know its cause for certain, then the sufferer is not entitled to the same societal latitude as those who have equally obscure

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illnesses which do not affect their mental state. It is therefore discriminatory, as well as incorrect, to selectively deprive psychiatric patients of diagnoses, which, despite their imperfections, have served medicine well for thousands of years.

Diagnosis and 'caseness'

The second philosophical error the critique makes is to claim that dependence on description makes empirical case ascertainment impossible. Provided a threshold can be reproducibly defined, there is no reason for that threshold not to be based on the description. As ever, there are uncontested physical equivalents: pyrexia, obesity, and hypertension are based on cut-offs for temperature, weight, and blood pressure, which are all clinical observations. Psychiatric questionnaires behave similarly and lead to similar questions about service delivery at a population level (Foreman, 2015). Genetic studies can be alternatively understood as validations of descriptive case definitions by differential heritability or (more recently) DNA analysis. The extended discussion of this, in my initial position paper, points out that, even if construct validity is changed by recent advances, a diagnosis may still retain predictive validity, and may therefore guide our next steps as clinicians. There is nothing new or threatening to diagnosis in such changes: once phthisis and Pott's disease were regarded as different conditions; now they are both understood as manifestations of tuberculosis, but the signs and symptoms of each continue to be of diagnostic utility. So long as observation-based identification of caseness correctly tells us what we should do next, it retains its value.

Confusing correlation with causation

This well-known philosophical error occurred when the critique considered the evidence for medication on clinical outcomes. The critique mentioned that higher rates of medication use were associated with higher rates of delinquency (Molina *et al*. 2007) but failed to mention that, when these were measured, the provision of medication was not blinded in the groups under study, and could be varied by subject choice. The authors mentioned that causation could not therefore be inferred (people with worse ADHD could both attract higher doses and be more prone to delinquency): the critique did not. It is, of course, true that treatments have side effects, and some drug treatments may have more side effects than non-drug treatments. However, harm also arises from the conditions themselves. Striking the optimal balance to minimise harm is essential in the treatment decision, but errors in interpretation may well lead to the wrong balance being struck.

Conclusions

The critique has made fundamental mistakes in its understanding of diagnosis, caseness, and the relevance of causation. This has meant it has misinterpreted the evidence on ADHD. In particular, it has used false claims of the invalidity of psychiatric diagnosis to claim non-existence of a disorder with significant disability, when identified at diagnosis. The risks of such an approach are clear in the discussion on treatment. While there are questions about the effectiveness of long-term treatment for ADHD, the critique's approach to these is to suggest there is nothing wrong in the first place, when this is clearly not so. In the words of the Edwin Smith Papyrus, I would rather consider ADHD an ailment with which I will contend, rather than an ailment not to be treated.

Conflicts of Interest

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Ethical Standards

The authors assert that all procedures contributing to this work comply with the ethical standards of the relevant national and institutional committee on human experimentation with the Helsinki Declaration of 1975, as revised in 2008.

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Rebuttal to Foreman's article 'Attention-deficit hyperactivity disorder (ADHD): progress and controversy in diagnosis and treatment'

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In his article supporting the notion that attention-deficit hyperactivity disorder (ADHD) represents a knowable, natural biological entity with a characteristic aetiology and response to treatment, Dr Foreman demonstrates a preference for rhetoric over scientific clarity. He believes we should accept that attacks on the concept are 'misguided', because 'it is well established within conventional psychiatry', and therefore he confidently states that 'claims that ADHD is solely a social construct can be conclusively refuted'.

These bold statements are hollow when the evidence put forward by Foreman is analysed within a proper and robust scientific framework. Scientific knowledge develops through disproving a null hypothesis. In this case the null hypothesis that must be assumed, until proven otherwise, is that there is no *characteristic* natural entity that can be elicited and reliably measured/identified that corresponds with ADHD. In the rest of this paper I examine how well (or not) Foreman has been able to demonstrate that this null hypothesis can be disproven.

Foreman concentrates on two lines of evidence to support his conviction that ADHD is a valid and largely biological condition – genetics and neuroimaging.

Genetics

Foreman is convinced that ADHD is strongly heritable with a 'heritability of 0.7–0.8'. The basis of such estimates has been thoroughly debunked as it rests on what is known as the 'equal environment assumption' (EEA), where it is assumed that when a higher percentage of monozygotic (MZ) than dizygotic (DZ) twins share the same disorder, this is due to genetics rather than environmental factors. For this to be the case it is assumed that environmental influences are controlled for, as they share the same environment (i.e. siblings in the same family, etc.). However, it has been long established that EEA isn't supportable as MZ are often treated more similarly (e.g. dressed in same clothes) and experience a unique psychological environment (e.g. swapping roles to confuse others) when compared with DZ twins. Therefore they do not experience equal environments and so the twin study method cannot disentangle genetic from environmental factors (Joseph, 2009). The only way to reliably evidence a specific genetic contribution to ADHD is through molecular genetic studies. Since faster and cheaper whole genome scans have become available the molecular genetic evidence has been accumulating. Foreman concedes that any potential genetic contribution is not showing up as specific but rather as a 'general vulnerability to psychopathology, irrespective of diagnostic type'. However, he shows his lack of scientific credentials by taking at face value that 'unequivocal evidence, unconfounded with potential environmental effects was identified in 2010, when an international population with ADHD was shown to have a greater proportion of Copy Number Variants (CNVs) than controls' (Williams *et al*. 2010). This study is typical of the scientism (a belief that something is 'scientific' because it looks like you are doing 'science') that has infected academic psychiatry. The study involved the comparison of whole genome scans of 366 children 'with ADHD' with those of 1047 'non-ADHD' control children, looking for CNVs (abnormal bits of repeated or deleted genes).

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