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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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Researchers found 13.9% (51) of the children with ADHD had CNVs compared with 7.4% (78) of the non-ADHD controls. The average recorded IQ of the 366 children with ADHD was 86, 14 points below the general population average of 100. Furthermore, when 33 intellectually impaired 'ADHD children' (IQ lower than 70) were excluded from the ADHD cohort, only 11.4% (38) of the remaining 333 had CNVs. In total, 39% (13) of the 33 children with ADHD and an IQ below 70 had CNVs. This evidence is more suggestive of a relationship between the presence of CNVs and intellectual disability than ADHD. The authors should have controlled for IQ given its disproportionate impact on CNV levels, but chose not. This is worse than junk science, as the authors have misled the medical community in their conclusions. Authors such as Foreman have a responsibility to read such research more carefully before simply repeating the authors' unsupportable conclusions. A more scientifically grounded (rather media-attention seeking) reading of these results suggests that CNVs probably have no association with ADHD *per se*. With genetics then, the cupboard is empty and the null hypothesis stands: there is no characteristic identifiable genetic abnormality/profile associated with ADHD.

Neuroimaging

As with genetics, the neuroimaging evidence put forward by Foreman is just as inconclusive. Having explained there is no 'conclusive' account of the neurological basis of ADHD (which he suggests is for a variety of reasons such as small sample sizes, but neglects to mention the most obvious possibility – that there is no conclusive evidence because there is no characteristic 'ADHD' abnormality), he cites a few references that he suggests implicate 'right-lateralised ventral', 'the fronto-parietal', 'fronto-striatal', and 'ventral striatum'. He could have chosen more potential areas from other studies. This picture of consistently inconsistent findings, which are statistical deviations (the brains would not be recognised by radiologists as being clinically abnormal), come from small sample size studies, don't control for IQ level, and most don't control for the effects of medication (to mention a few obvious issues) is simply not commented on. Foreman displays again his ability to take other people's findings at face value without a more critical analysis in order to reach his favoured conclusion. Here too then the cupboard is also empty. No one has come near to finding a characteristic abnormality and as a result there is no biological marker or brain scan used to diagnose ADHD. The null hypothesis stands: there is no characteristic identifiable neurological abnormality/profile associated with ADHD.

Treatment

Well so much for the shoddy science. What about the important question of treatment? Amazingly, Foreman suddenly discovers his capacity for more careful scientific thinking when he reviews 'non-drug' treatments for ADHD, noting issues such as publication bias, study quality, un-blinded studies, concluding (correctly I think) that the evidence on the effects of specific non-drug treatments on 'core ADHD symptoms' is not particularly impressive. Unfortunately, he didn't ask himself how meaningful to most children's life getting rid of 'core ADHD symptoms' as a goal is.

Unsurprisingly, Foreman returns to form when reviewing drug-based treatments. The recent Cochrane systematic review (Storebø *et al*. 2015), is quoted by him to handpick data that supports his favoured position for viewing medication as the main or only sure fire effective intervention. He doesn't mention their conclusion that because *all the trials* were considered poor quality and un-blinding was likely to be common (due to frequency of adverse effects) that they could not conclude that taking methylphenidate will improve the lives of children and adolescents with ADHD. This is a startling admission that, despite decades of research, established practice cannot be considered firmly evidence based even with short-term prescriptions. Foreman says nothing about the long-term outcome data, an omission that is unacceptable given that prescriptions are usually given for many years. As I pointed out in my article the available evidence suggests that young people who stay on medication are doing no better or have worse long-term outcomes (in areas as diverse as their physical health and academic outcomes) than those who don't take medications regardless of initial severity of symptoms.

Unfortunately Foreman is in good company in his unwillingness to address serious concerns about established practice. As Moncrieff and I have previously pointed out (Moncrieff & Timimi, 2012), UK National Institute for Health and Care Excellence (NICE, 2008) recommended using stimulant medication as a first line treatment in severe ADHD (but not for mild or moderate ADHD) on a faulty basis. To support this recommendation they referred to only one study that reports on a re-analysis of data from the largest trial comparing medication and behavioural treatments. This paper concluded that a subgroup of children with more severe ADHD symptoms showed a larger decrease in symptoms with medication than with behaviour therapy (Santosh *et al*. 2005). This study used data that was gathered at the 14-month point of the study. Data obtained after 3 years' follow-up did not reveal beneficial long-term effects of medication over behaviour therapy, even in those with more severe symptoms at the start (Jensen *et al*. 2007). The

recommendation of the NICE guideline to use medication as a first line treatment for adult ADHD was based on only three studies with study duration of 21–45 days (Moncrieff & Timimi, 2012). If this is the bias and lack of objectivity in national guidelines, perhaps it's not surprising to see such a one-sided coverage of the evidence base in Foreman's article.

Good therapy is not about treating symptoms

The therapeutic point about understanding the evidence and not getting seduced by wishful and magical thinking into believing there is a knowable biological category out there called ADHD for which there is a specific and (pseudo)medical treatment, is that practice based on such insecurity (about our identity as doctors) may limit our imaginations, whilst culturing us and others into a narrow view about what really matters in young people's lives. In my experience (not a scientific position I know) most young people who could be diagnosed with ADHD do fine with short family/school-based interventions that focus less on symptoms and more on meaningful outcomes, context, and relationships. I fear that in this age of what I call the 'McDonaldisation' of children's mental health (Timimi, 2010) such blinkered avoidance of robust engagement with the evidence is leading to a profession not only corrupted by pharmaceutical industry involvement, but where we are losing our ability to offer that unique perspective to medicine that comes from a thorough grounding in understanding context, systems, and the nature of change (development in all its guises).

I will leave the last words of my rebuttal to one of the most productive and influential child psychiatrists of the modern era – Leon Eisenberg. In the 1960s Eisenberg was one of the leading proponents for recognising hyperactivity and then ADHD and putting these concepts on the map by arguing for their inclusion in diagnostic manuals. Later he came to regret this. Shortly before his death, in an interview with Der Speigel in 2012 (see http://www.spiegel.de/spiegel/print/d-83865282.html) he stated, 'ADHD is a prime example of a fabricated disease' arguing instead that child psychiatrists should be much more thorough in identifying the psychosocial reasons that could lead to behavioural problems. Eisenberg was by the end of his career arguing vociferously that kids cannot be understood and helped by isolating them into individualist units that work or don't work according to some narrow neoliberal definition of 'normality', 'The epidemiology of the mental and physical health of children and adolescents the world over reflects: the genomes they inherit (and the modifications those genes undergo in utero); the pregnancies that led to their births, whether their mothers survive those pregnancies, and whether their births were welcome; the parents, the neighbours, and the neighbourhoods they 'inherit' along with their genomes; when and where they live (by cohort, by country, and by province); the air they breathe; the water they drink; what and how much they eat; the schools they attend (and by whom they are taught what and for how long); the energy they expend; the family status in the social order; the friends they have; and last the amount and kind of medical and psychiatric care they receive' (Eisenberg & Belfer, 2009: 26). Amen to that.

Conflicts of Interest

None.

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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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