The notion that adults can have attention-deficit hyperactivity disorder (ADHD) has polarised British psychiatry, primarily over ADHD’s diagnostic validity, and whether drug treatment is justifiable and ethical, or merely cosmetic.1,2 However, as this controversy could disadvantage patients, re-examination is merited.

The diagnosis problem

A true diagnosis is a syndrome of signs and symptoms with specific aetiology and pathology producing biological disadvantage, responsive to specific interventions. Attention-deficit hyperactivity disorder may fall short; its apparent symptom cluster could be an artefact of dichotomisation, evidence suggests that ADHD represents one end of a spectrum.3 Neuroimaging studies identify differences, but do not establish whether these represent pathology or extremes of normality. Specific chromosomal variants4 occur in a few cases, but a better model involves multiple genes contributing to quantitative traits.5 Although medications help ADHD, particularly individuals with the most severe, symptomatic ADHD traits,3 they could, arguably, help everyone. Finally, ADHD could potentially confer biological advantage.

This replicates the controversies that surround similarly defined non-psychiatric conditions such as hypertension, particularly ‘pre-conditions’.6 Here, a downwardly drifting threshold applied to the normal spectrum risks pathologising more of normality. The fundamental problem is that thresholds arbitrarily define sections of asymptomatic people as having a diagnosis, implying abnormality and pathology. The logical endpoint is that complete populations are disorderised and require treatment, as illustrated by the concept behind the polypill.7 An unintentional consequence is that refusing or denying treatment is unethical even if the individual is asymptomatic.

Thresholds polarise risk factors, inadvertently obscuring the fact that risks operate across the population (Fig. 1), increasing from one end to other. Attention-deficit hyperactivity disorder traits and their associated risks also vary across the population. Thresholds can provide false security to those below, and false alarm to those above – hypertension does not, for example, guarantee a stroke. Similarly, although ADHD traits are associated with an increased risk of social adversity (for example divorce, erratic employment) and psychiatric disorders (for example anxiety and mood disorders, substance misuse) these are not inevitable. Thus, although around 15% of prisoners have significant ADHD,8 85% do not, and although 70% with ADHD have mental disorders,3 30% do not.

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A threshold implies that having extreme values on the distribution is inherently problematic and should be corrected towards the mean. However, some argue that extremes may confer advantage in certain circumstances and populations. Charities such as AtB and Mindroom, suggest people with ADHD ‘thrive on new challenges with short term deadlines’ and evidence suggests that ADHD-associated genes may be positively selected. Perhaps current societal norms disadvantage those with significant ADHD.

Although those at the very extreme of the distribution are likely to be symptomatic on the basis of their traits alone (extremes of inattention and restless overactivity are usually disabling), and are most likely to benefit from intervention, a threshold’s true function is to guide who may most benefit from risk reduction – that is, from a preventive intervention. However, whereas antihypertensives are mainly primary prevention, ADHD medication is secondary prevention, ‘diagnosis’ requiring significant functional impairment, thereby targeting individuals who have developed secondary problems. It is reassuring that the proposed diagnostic criteria for ADHD in adults explicitly require significant impairment to be present in at least two areas of life, as primary prevention with stimulants (medicating someone who does not incur difficulties despite being particularly impulsive or inattentive) cannot be justified – not only are the inherent risks of stimulants prohibitive, but there is no evidence that stimulants are effective as primary prevention. As with hypertension, non-pharmacological lifestyle and psychological interventions may be more appropriate primary prevention.

The problem of medicating a (psychiatric) risk factor

The possibility of pharmacologically modifying psychiatric risk factors raises novel challenges because it potentially involves modifying personality or temperamental traits using potent drugs. There is continuity of concept between neurodevelopmental disorders (such as ADHD) and personality disorders, both being characterised by extremes of dimensions of personality or temperament present from childhood. Personality disorders may be regarded as risk factors, similar to ADHD. Although Kendall postulated that trait conditions would become accepted diagnoses when effective treatments emerged, this has not happened with ADHD, despite effective pharmacotherapy. Instead, adult ADHD is used as an exemplar in the debate about the growing trend to pharmacologically modify aspects of personality. The available data suggest that ADHD traits have lead to secondary problems.

Conclusions

The recognition of ADHD in adults leads to a number of challenging consequences in terms of our concept of diagnosis in mental health disorders, the role of medication and the ethical questions raised by the possibility of medications being able to modify aspects of personality. The available data suggest that ADHD traits are best regarded as risk factors for a set of negative outcomes. Pharmacologically modifying these ADHD risk factors should be limited to those for whom these traits have led to significant secondary negative outcomes. As treatment is secondary prevention, and the first-line drugs are potentially dangerous, only those willing to engage meaningfully in modifying these risk factors, and willing to accept responsibility for managing their risk factors irrespective of whether or not they take medication, should be prescribed these agents.
Adults with attention-deficit hyperactivity disorder

References