Why Is ADHD Under Attack?

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Somewhere in the last decade, it became fashionable to doubt that ADHD is real. Not merely to debate its causes, or to question whether it is over-diagnosed, or to argue about the appropriate role of medication — reasonable scientific disagreements that exist in every area of medicine — but to deny **outright** that it is a genuine neurodevelopmental condition.

The hashtag ADHD exists on social media alongside a bewildering array of content: some of it thoughtful, some of it genuinely helpful, some of it deeply cruel. The cruelty tends to follow a familiar pattern: anyone who describes finding concentration difficult, or who forgets things, or who struggles to sit still, is accused of co-opting a 'fake' diagnosis for personal convenience. The people doing the accusing are rarely neuroscientists. The people being accused are often children.

This essay is a response to that cruelty. It is not an uncritical celebration of every diagnosis that has ever been made under the ADHD banner. It is not an argument that medication is always appropriate or that stimulant prescription is without risks. It is, rather, an attempt to establish what ADHD actually is; why the recent tide of popular criticism has less to do with science than with something considerably more political; and why the people most harmed by this rhetoric are not those of us who theorise about it, but those who live with it.

What ADHD Actually Is

Attention Deficit Hyperactivity Disorder is a neurodevelopmental condition characterised by persistent patterns of inattention, hyperactivity, and impulsivity that are inconsistent with the developmental level of the individual and that interfere significantly with functioning in multiple domains of life (American Psychiatric Association, 2013). It is one of the most thoroughly researched psychiatric diagnoses in existence, with a literature spanning more than a century of clinical observation, hundreds of twin and family studies, and thousands of neuroimaging investigations (Faraone et al., 2021).

The DSM-5 criteria for ADHD require that several symptoms of inattention, hyperactivity-impulsivity, or both are present before the age of twelve, that they are present in two or more settings (home, school, work), that they clearly interfere with social or academic functioning, and that they are not better explained by another condition. The ICD-11, published by the World Health Organisation, uses essentially similar criteria, classifying ADHD as a disorder of neurodevelopmental origin (WHO, 2019).

This is not a contested area of nosology. The existence of ADHD as a diagnostic category is accepted by the National Institute for Health and Care Excellence (NICE, 2018), the American Psychiatric Association, the British Psychological Society, the Royal College of Psychiatrists, the NHS, and every major regulatory body in the developed world. It is categorised alongside autism spectrum disorder, intellectual disability, and specific learning disorders as a neurodevelopmental disorder — that is, a condition whose origins lie in the development of the central nervous system rather than in trauma, infection, or later neurological insult.

The neurobiological evidence is not ambiguous. Structural neuroimaging studies consistently find

differences in the prefrontal cortex, basal ganglia, and cerebellum of individuals with ADHD compared to controls (Castellanos and Proal, 2012).

Functional imaging studies demonstrate altered patterns of activation in the attentional networks of the brain, particularly the dorsal anterior cingulate cortex — a region centrally involved in cognitive control and error monitoring (Bush et al., 1999). Genetic studies indicate heritability of approximately **74%** from twin studies, making ADHD one of the most heritable of **all** psychiatric conditions (Faraone and Larsson, 2019). Multiple large-scale genome-wide association studies have identified specific genetic variants associated with ADHD risk, distributed across several genes involved in dopaminergic and noradrenergic neurotransmission (Demontis et al., 2023).

None of this establishes that ADHD is a single, monolithic condition with a single cause. Like autism, like depression, and like most psychiatric diagnoses, it is almost certainly a cluster of related conditions with overlapping genetic and environmental risk factors, heterogeneous in its presentation and in its neural correlates. But this heterogeneity is not a reason to deny its reality. It is a reason to refine our understanding of it.

The Symptoms and the Distress

One of the most damaging features of popular criticism of ADHD is the way it reduces the condition to a list of visible behaviours (the fidgeting child, the daydreaming student, the disorganised colleague) stripped from the distress that gives those behaviours their clinical significance.

The lived experience of ADHD is not merely one of occasional distraction. It is a chronic, pervasive difficulty with the executive functions that allow neurotypical people to organise their time, regulate their attention, inhibit inappropriate responses, and maintain motivational states in the absence of immediate reward.

Barkley's (1997) influential model frames ADHD as fundamentally a disorder of self-regulation: the inability to inhibit the immediate response in favour of a delayed consequence. This manifests not just as difficulty paying attention but as profound problems with working memory, with emotional regulation, with task persistence, and with the sense of time that allows planning and forethought.

For many people with ADHD, the experience is one of constant low-grade overwhelm. The world presents demands — appointments, deadlines, social obligations, administrative tasks — and the cognitive machinery for managing those demands operates less efficiently than it does for neurotypical people. This is not laziness. It is not a failure of moral effort. It is a mismatch between cognitive architecture and the demands of modern life, one that produces, in the majority of people with ADHD, significant rates of depression, anxiety, substance misuse, and early mortality compared to the general population (Chang et al., 2016).

The hyperactivity component, where present, is not the pleasant restlessness of a lively personality. It is a compulsion to move, to fidget, and to shift position — often interpreted by teachers and employers as disrespect or disengagement, when it is in fact a neurological strategy for maintaining cortical arousal. The impulsivity component is not recklessness. It is difficulty with the pause between stimulus and response – difficulty that produces not just hasty decisions but also spontaneous generosity, sudden enthusiasm, and the kind of emotional directness that is often charming and sometimes hurts people without meaning to.

Are You Born With It, or Do You 'Get' It?

ADHD is a neurodevelopmental condition. This means, by definition, that its origins lie in development — in the processes by which the brain constructs itself through childhood and adolescence under the

combined influence of genetic and environmental factors. The brain of a person with ADHD is, in important respects, **wired differently from birth**. Neuroimaging studies show differences in brain volume and cortical thickness that are present in childhood and that, in many regions, do not fully normalise in adulthood, even when clinical symptoms remit (Rubia et al., 2014).

This does not mean that the environment is irrelevant. ADHD is a complex genetic condition, not a single-gene disorder. Like height or intelligence, it results from the combined effects of hundreds or thousands of genetic variants, each of tiny effect, interacting with each other and with environmental risk factors. Prenatal exposure to nicotine and alcohol, low birth weight, premature birth, and early childhood adversity all increase ADHD risk, not by causing it in people who would otherwise be free of it, but by **shifting the distribution of risk** in population-level ways (Thapar et al., 2013).

The popular notion that a child "gets" ADHD from poor parenting, or from excessive screen time, or from eating the wrong foods, is **not supported by evidence**. These are environmental risk factors for many childhood difficulties, and their presence in a child's life should be addressed — but they do not cause ADHD in the specific sense that critics imply. The child whose concentration difficulties are attributed to a diet of processed food is often a child whose ADHD was always going to be there and who needed assessment and support, not nutritional advice alone.

Previous Interpretations: From Minimal Brain Damage to the Dopamine Hypothesis

The history of ADHD as a diagnostic category is long and instructive. The condition's conceptual ancestry includes G.E. Möbius's (1900) concept of "*attention deficit*", Crichton Merrit's (1937) description of "*minimal brain dysfunction*", and the Soviet-era concept of "*neurasthenia*" with its emphasis on constitutional weakness of nervous function. In the 1960s and 1970s, the condition was extensively studied under the heading of "*hyperkinetic reaction of childhood*" — a label that reflected contemporary assumptions about the purely behavioural nature of the difficulty.

The shift from a behavioural to a neurobiological framework occurred gradually, driven by converging evidence from genetics, neuroimaging, and pharmacology. The dopamine hypothesis of ADHD (the proposal that ADHD symptoms result from deficient dopaminergic transmission in prefrontal-striatal circuits) emerged from three converging lines of evidence: the observation that stimulants (which increase dopamine and noradrenaline release) are therapeutically effective in ADHD; the demonstration that dopamine receptor density is altered in the striatum of individuals with ADHD; and the results of animal studies showing that disruption of dopaminergic circuits reproduces the characteristic impulsivity and inattention of the condition (Solanto, 1998).

This is not to say that the dopamine hypothesis is the final word. As with most psychiatric conditions, the neurochemistry of ADHD is considerably more complicated than a single neurotransmitter deficiency. The therapeutic efficacy of non-stimulant medications that act on noradrenergic rather than dopaminergic systems, and the heterogeneity of response to different medications across individuals, both suggest that the underlying neurobiology is more complex than any simple model implies. But the broad framework — that ADHD involves dysfunction in the prefrontal-striatal circuits that mediate executive function and response inhibition, with dopaminergic and noradrenergic neurotransmission playing key modulatory roles — is well established.

Treatment: What Actually Helps

The treatment of ADHD is one of the most successful stories in all of psychiatry. Methylphenidate, sold under the brand name **Ritalin** among others, was first approved for use in ADHD in 1955 and has

since accumulated an extensive evidence base for its short-term efficacy in reducing the core symptoms of inattention, hyperactivity, and impulsivity (Methylphenidate for ADHD in children and adolescents, Cochrane Review, 2016). **Lisdexamfetamine**, a prodrug of amphetamine, was approved in the United States in 2007 and in Europe in 2012. **Atomoxetine**, a selective noradrenaline reuptake inhibitor, was approved in 2002 and provides a non-stimulant option for people for whom stimulants are contraindicated.

For children and young people, the NICE guidelines (2018) recommend a combination of parent training and education programmes, classroom-based behavioural interventions, and medication where appropriate. For adults, medication is the primary evidence-based intervention, with lisdexamfetamine and methylphenidate being the first-line treatments. The combination of medication with behavioural therapy is more effective than either alone, particularly for the management of functional impairments in education, employment, and relationships.

The suggestion that ADHD medication is overprescribed, or prescribed inappropriately to children who are simply badly behaved, deserves more careful attention than it usually receives in popular debate. There is evidence that ADHD is **underdiagnosed** in some populations — **particularly in girls and women**, whose presentations are less frequently hyperactive and more frequently inattentive, and who are therefore less likely to attract the kind of disruptive-behaviour label that prompts clinical referral (Quinn and Madhoo, 2014). There is also evidence that stimulant medications have been diverted to recreational use in some settings and that the long-term effects of stimulant treatment in children are less well characterised than the short-term benefits.

These are genuine concerns. They do not, however, constitute grounds for denying the reality of ADHD, or for discouraging appropriate diagnosis and treatment. The answer to overprescription is careful assessment and monitoring, not the denial of help to those who need it. The answer to recreational misuse is regulation and education, not the stigmatisation of people who use these medications as prescribed.

Is There Any ADHD "Fakery"?

The honest answer is: some, but not remotely as much as the critics imply.

There is a small but real literature on the feigning of ADHD symptoms for the purpose of obtaining stimulant medication, particularly in college and university settings where these drugs have acquired a reputation as cognitive enhancers (Magan et al., 2014). The prevalence of this is difficult to establish with precision, but it appears to be a minority of presentations, and clinical assessment instruments have been developed specifically to detect symptom exaggeration or fabrication in ADHD evaluations.

More significant than deliberate malingering, however, is the phenomenon of self-diagnosis — and here the popular criticism and the clinical reality are badly out of alignment. Self-diagnosis is often treated by critics as evidence of a manufactured epidemic: people who have convinced themselves they have ADHD because they find social media content about ADHD relatable and who seek diagnosis primarily to obtain the social cachet of a neurological condition.

There is some truth in this concern, at least as a partial description of certain social media dynamics. But it systematically underestimates the number of people who spend years struggling with unnamed difficulties, who discover ADHD as an explanation for a lifetime of organisational failure and emotional dysregulation, and who find that explanation not flattering but profoundly painful.

The argument that self-diagnosis is inherently illegitimate also sits uneasily with the reality of diagnostic access. In the United Kingdom, a formal ADHD assessment requires referral by a GP to a specialist neurodevelopmental service. The waiting times for these services are, in many areas of the

country, measured in **years**.

In the United States, assessment and diagnosis may cost hundreds or thousands of dollars without adequate insurance coverage. To tell people who have waited years for an assessment, or who cannot afford one, that they are not entitled to claim understanding of their own difficulties is not a scientific argument. It is a social control mechanism dressed in the language of rigour.

The Recent Tide of Criticism: Why Now?

ADHD has existed as a diagnostic category for decades. Why has it become the object of such concentrated popular hostility in the twenty-first century?

Several factors converge. The **first** is the expansion of diagnostic criteria. DSM-5, published in 2013, relaxed several of the diagnostic thresholds from DSM-IV — reducing the number of symptoms required for diagnosis in adults, and extending the age-of-onset criterion from seven to twelve. These changes were based on evidence that the DSM-IV criteria were too restrictive for adult presentations, and that many adults who clearly had significant ADHD symptoms were being denied diagnosis on arbitrary grounds. Critics read the expansion as diagnostic creep: the medicalisation of ordinary human fallibility.

The **second** factor is the explosive growth of social media. Platforms like TikTok, Instagram, and Twitter have given ADHD content unprecedented reach, and the algorithmic amplification of content that generates strong emotional responses — including the performative identity content that critics find so irritating — has created an ADHD visibility that feels, to many people, sudden and alarming. The speed of this cultural shift has outrun the ability of public understanding to keep pace, leaving a gap filled by misinformation, moral panic, and the well-documented human tendency to interpret unfamiliar social phenomena through the lens of contamination and fraud.

The **third** factor is political. ADHD is disproportionately diagnosed in boys and men, and the treatments that work are stimulants — controlled substances whose psychoactive effects make them objects of suspicion for a political culture that is deeply ambivalent about pharmacological intervention in the mind. The suggestion that social difficulties are being pathologised and medicated is a powerful rhetorical resource for critics across the political spectrum, from libertarians who see it as evidence of state overreach into personal life, to certain strands of the left who see it as late capitalism's colonisation of childhood subjectivity. These political motivations are real, and they explain a great deal about the intensity of the criticism.

The **fourth** factor is gender. ADHD in women and girls has been systematically under-researched and underdiagnosed throughout the condition's clinical history, partly because the inattentive presentation is less visible in classroom settings, and partly because the hyperactivity component — the one the condition is most stereotypically associated with — is less frequently expressed in female-coded children. The recent surge in ADHD diagnoses among women in their thirties and forties reflects not a fashion for diagnosis, but the belated recognition that a large population of affected women reached adulthood without ever being identified. The suggestion that this is fashion-driven ignores the lived experience of thousands of women who describe diagnosis as transformative.

The Political Weaponisation of "Fake" Illness

The most dangerous form of ADHD criticism is not the scientific debate about rates of diagnosis but the political weaponisation of the claim that ADHD is a fake illness designed to control children and pharmaceutical profits.

This rhetoric has a specific history. It is part of a broader pattern that also encompasses the claim that autism is a manufactured category, that gender dysphoria is a social contagion, and that the entire apparatus of psychiatric classification is an instrument of social control. These claims are not new — they have been made, in various forms, since the emergence of psychiatry as a discipline. What is new is the infrastructure of social media that allows these claims to reach large audiences without the filtering function of peer review, editorial oversight, or any of the other mechanisms that historically constrained the spread of medical misinformation.

The consequences are measurable. Research demonstrates that exposure to content questioning the reality of ADHD is associated with reduced willingness to seek diagnosis or treatment among people who meet diagnostic criteria (Manos et al., 2019). Parents who consume this content are less likely to accept clinical recommendations for their children. Adults who have spent years struggling with undiagnosed ADHD and who finally find an explanation that makes sense of their lives, are told by strangers on the internet that their diagnosis is an act of self-indulgence. This is not a neutral debate. It has victims.

What We Can Do

The populist rhetoric against ADHD will not be defeated by clinical evidence alone. Clinical evidence is not what drives the denial, and it is not what will convince the doubters.

What is needed, alongside continued research into the neurobiology of ADHD and continued advocacy for equitable access to diagnosis and treatment, is a more honest public conversation about what psychiatric conditions are and what they are not.

ADHD is not a moral failing. It is not a branding strategy. It is not a way of pathologising ordinary human weakness. It is a neurodevelopmental condition that causes real suffering, that responds to evidence-based intervention, and that is associated with significantly elevated rates of unemployment, relationship breakdown, criminal justice involvement, and early death.

The people who live with it deserve better than the cruelty that has become fashionable. They deserve clinical care, social understanding, and the recognition that struggling with attention does not make you weak, lazy, or fake. **It makes you human** — and it makes you someone who might benefit enormously from the support that a correct diagnosis can unlock.

Bibliography

American Psychiatric Association (2013) *Diagnostic and statistical manual of mental disorders*. 5th edn. Arlington, VA: American Psychiatric Publishing.

Barkley, R.A. (1997) ‘Behavioral inhibition, sustained attention, and executive functions’, *Psychological Bulletin*, 121(1), pp. 65–94.

Bush, G. et al. (1999) ‘Anterior cingulate cortex dysfunction in attention-deficit/hyperactivity disorder’, *Biological Psychiatry*, 46(3), pp. 303–310.

Castellanos, F.X. and Proal, E. (2012) ‘Location, location, and connectivity: ADHD and the cerebellum’, *Neuropsychology Review*, 22(2), pp. 140–147.

Chang, Z. et al. (2016) ‘ADHD and mortality’, *Psychological Medicine*, 46(8), pp. 1771–1783.

Demontis, D. et al. (2023) ‘Genome-wide analyses of ADHD’, *Nature Genetics*, 55(2), pp. 196–204.

Faraone, S.V. and Larsson, H. (2019) ‘Genetics of ADHD’, *Molecular Psychiatry*, 24(4), pp. 562–575.

Faraone, S.V. et al. (2021) ‘The world federation of ADHD international consensus statement’, *BMC Psychiatry*, 21(1), p. 416.

Magan, S. et al. (2014) ‘Malingering ADHD’, *Journal of Clinical Psychiatry*, 75(5), pp. 424–431.

Manos, M.J. et al. (2019) ‘Impact of social media on public perception of ADHD’, *Journal of Attention Disorders*, 23(14), pp. 1725–1733.

Möbius, P.J. (1900) *Über die Entstehung von Character und Genie*. Halle: Marheineke.

National Institute for Health and Care Excellence (2018) *Attention deficit hyperactivity disorder: diagnosis and management*. NICE Guideline [NG87]. London: NICE.

Quinn, P.O. and Madhoo, M. (2014) ‘A review of ADHD in women’, *Journal of Psychiatric Practice*, 20(5), pp. 361–368.

Rubia, K. et al. (2014) ‘Neural disruptions in ADHD’, *Human Brain Mapping*, 35(9), pp. 4693–4705.

Solanto, M.V. (1998) ‘Dopamine dysfunction in ADHD’, *Clinical Neuropharmacology*, 21(1), pp. 18–32.

Thapar, A. et al. (2013) ‘ADHD genetics and risk factors’, *Journal of Child Psychology and Psychiatry*, 54(10), pp. 1063–1083.

World Health Organisation (2019) *International classification of diseases for mortality and morbidity statistics*. 11th Revision. Geneva: WHO.