A Closer Look at ADHD (Referenced)

Introduction

This guide is the referenced companion to the two ADHD patient documents — the adult and child guides, each of which combines the explanation with practical guidance. It sets out the evidence and reasoning behind them, so that interested patients and clinicians can read in more depth and look at the original sources. It is based on my own experience and reading of the evidence base and is not intended to be exhaustive or a definitive guide. It is what I have found helpful to understand, and what I would share if I had unlimited time. Different clinicians and patients may come to different conclusions based on their own experience.

Part One: Understanding ADHD

What kind of diagnosis ADHD is

ADHD is a widely recognised diagnosis.[1] When clinicians use agreed criteria they tend to agree on it, and it picks out a group at higher risk of problems at school and work, with the law, and with health.[2,3] But it is not a disease with a well-defined physical fault behind it. There is no blood test, brain scan or genetic test that correlates with the diagnosis.[2] It is best understood as a description of a pattern: a group of traits — inattention, restlessness, impulsivity and strong emotional reactions — that tend to go together and cause difficulty in some settings.[3,4]

That puts ADHD in the same bracket as much of psychiatry, and as parts of general medicine such as migraine, where we diagnose from a recognisable pattern rather than an objective test. This can lead to strong and polarised views in the wider cultural discourse and media.[5,6] The position I take, based on practice and reading the evidence, is that the label is useful because it can validate a patient's experience and point towards support — while still being a description, not an explanation.

I find this important because misunderstanding the nature of ADHD can lead patients to pin all hope on a future "cure" that doesn't really fit what is going on. That can increase distress, and it is exacerbated by long and increasing waiting times.

A trait spread across a spectrum, with the line drawn by judgement

The traits behind ADHD are spread right across the population — they are not something people simply have or do not have. ADHD sits towards the end of that range where the traits are strong and persistent enough to cause real difficulty, and the line is drawn by agreed criteria and clinical judgement, not by a natural boundary.[2,3]

The relative-age effect shows this clearly. In many countries the youngest children in a school year are much more likely to be diagnosed and medicated than the oldest in the same year. In a Canadian study of nearly a million children, boys born in the month before the cut-off were about 30% more likely to be diagnosed than those born just after it, and girls about 70% more

likely.[7] The simplest explanation is that ordinary immaturity is being read as a disorder. That does not necessarily make those diagnoses wrong — many of these children do struggle — but it shows how much a child's age and setting affect where the line ends up.

What the biology does and does not show

Before the findings, I find it helpful to consider the logic. Every brain is different — if you scanned the whole population you would find variation everywhere, because no two people are alike. So if you select a group who share a set of traits and compare their scans with everyone else's, finding some average difference is close to inevitable. It tells you the group differs on average; it does not tell you that you have found the cause of the traits, still less a fault. And an average difference between groups says almost nothing about any individual within them.

The imaging studies bear this out. The largest study of its kind, the ENIGMA mega-analysis of 1,713 people with ADHD and 1,529 controls, found smaller volumes in several subcortical structures — but the effects were tiny. The biggest, for the amygdala, was a standardised difference of 0.19; the others ranged from 0.10 to 0.15.[8] To put that in perspective, at an effect of that size, if you were handed one person's scan and asked which group they came from, you would be right barely more often than chance. The distributions overlap almost entirely. In addition, the differences were largest in children, were not statistically significant in adults at all, and were not correlated with symptom severity or stimulant use.[8]

The genetics research shows similar findings. Twin studies put heritability high — around 74%, with family and twin estimates ranging into the high 80s.[2] But when you look for the actual common variants, the evidence is less clear: the first well-powered genome-wide study found 12 significant loci across more than 20,000 cases, and all measured common variation together accounted for about 22% of the variance in liability.[9] Each individual variant contributes a fraction of a percent. There is no "ADHD gene," and polygenic scores are not at all useful for diagnosing an individual. The variants are also strikingly non-specific — dozens of other traits and conditions show significant genetic overlap with ADHD.[9]

One finding deserves particular attention, because it relates directly to the moving line discussed below. When the same group repeated the analysis with a broader case definition — nearly doubling the sample by including, for instance, people identified through having been prescribed ADHD medications — they found more loci (27), but the proportion of variance explained by common variants fell, from about 22% to about 14%.[10] The most likely reason is that the broader definition captured a more heterogeneous group. Widening the diagnostic boundary diluted the genetic signal. It is empirical support for the idea that the diagnostic threshold is key, and that it is based on reported experience rather than any objective testing.

None of this means the difficulties are not real, or that biology is irrelevant — ADHD is clearly heritable and clearly involves the brain in the same way all behaviours and traits do. It means biology gives us context, not a boundary or a different "type of brain," and not an explanation of any particular person. The honest line for patients is that there is no scan, no blood test and

no genetic test that correlates reliably with ADHD, and no different type of brain in the scientific sense. The difficulties associated with their traits, especially in the modern environment, can be genuinely disabling, and can be recognised, validated and supported without relying on a biological framing that the evidence doesn't really support.

Description, explanation, and treating the label as a thing

A common mistake is to treat the label as if it were a separate thing acting on the person — what is called reification.[6] "My ADHD made me do that" and "I can't, because of my ADHD" both slip from a description (these difficulties are there) to a cause (a thing that makes me do things).

This reification can shrink a patient's sense of what they can change, making difficulties feel fixed and outside them when many can in fact be worked on.[5,6] It can also obscure individual stories: the label stands in for understanding how this person's traits, physical state, environment and circumstances are combining to cause the difficulty. The same caution applies when a diagnosis is used to explain away or excuse behaviour — it names a pattern, but it does not, by itself, show that someone had no control over what they did. None of this makes the difficulties any less real; the point is to keep the diagnosis as a working description rather than the end of the explanation.

A related error runs the other way, and it influences what patients expect from us. Much of medicine works on a discovery model: the diagnosis identifies an underlying cause, and naming it points to a treatment that corrects it. Type 1 diabetes is a clear example — thirst, weight loss and fatigue are caused by absent insulin, and replacing the insulin corrects them. Patients often import that model into ADHD, and long waits can make this worse: by the time an assessment arrives, considerable hope may be resting on the discovery of something that can be put right. But the assessment is not uncovering a hidden condition beneath the traits; it is a judgement to apply a diagnostic label to a pattern already present.

This is worth saying explicitly and early, because the alternative is a patient who reaches the end of a long wait, receives the diagnosis, then has to wait months or longer to be prescribed medication, and finds that the differences these things make might be smaller than hoped for. The label can be very helpful: it makes sense of a history, facilitates adjustments, opens access to treatment, and often helps validate experience and reduce self-blame after years of struggle. What it cannot do is function as the discovery of a correctable lesion, and expectations set on that basis will not usually be possible to meet.

Culture, environment and the demands on attention

Our capacity for attention has not changed, but the world that influences it has. Modern schooling and work demand sustained focus, organisation and constant task-switching, and phones and screens are built to fragment attention with endless novelty and interruption.[3] This leaves people across the board feeling distracted, restless and overloaded — much like the difficulties ADHD describes.

It helps to see this as an interaction: difficulty often comes from a mismatch between how someone's attention works and what their environment demands, rather than sitting entirely inside the person. That does not deny real differences between people, and that some people naturally have more of the traits that cause ADHD-type difficulties — but it also avoids putting all the difficulty on them as an individual. It also explains why the same traits can be an asset in one setting and disabling in another, and why changing the environment is often as useful as "treating" the person. This way of looking at things is also at the heart of the neurodiversity idea,[11] though how useful that idea is in the clinic is still debated.[12]

Rising recognition and a line that keeps moving

Recognition and diagnosis have risen sharply, especially in adults and in groups missed in the past. Several things are happening at once. The criteria have widened: DSM-5 moved the age by which symptoms must have started from 7 to 12, and dropped the number of symptoms needed in adults from six to five, which brings more people over the line.[4] More awareness, more self-recognition and less stigma mean more people come forward. And the boundary itself tends to drift outwards over time, gradually taking in milder cases — what has been called "concept creep."[13]

This is not to say any particular person is overdiagnosed, or that the difficulties are not real. It is a reminder that prevalence figures shift a lot depending on how and where you measure,[14] that the line moves with the times and the setting, and that the relative-age findings show context affecting who gets diagnosed even within one classroom.[7] For patients, what matters in practice is whether the description helps them get understanding and support.

How common it is, and what it is linked to

About 5% of school-aged children meet the criteria at any one time, with the figures varying more because of how studies are done than because of a real rise of that size.[14] In adults, a global review put childhood-onset ADHD that persists at around 2.6%, and adult ADHD symptoms regardless of childhood onset at around 6.8%, both falling with age.[15]

Across large groups the associated risks are well evidenced: doing less well at school, unstable work, accidents, substance misuse, contact with the criminal justice system, and dying earlier. A Danish study of 1.92 million people found roughly double the death rate, mostly from accidents, and higher the later the diagnosis was made.[16] These are group patterns and do not decide what happens to any one person; many people diagnosed with ADHD do well, especially when recognition and support are provided.

How it develops, and why it shows up at certain points

ADHD is understood to first show in childhood, even though it is often picked up later. Difficulties tend to show up more when demands go up or the structure around someone falls away — moving school, university, work, becoming a parent. The brain's organising and self-control skills develop slowly through childhood and the teens, which is part of why children the

same age differ so much, and part of what drives the relative-age effect.[17] People often cope for a while by over-preparing, by perfectionism, or through anxiety-driven control, and these can hide the difficulties until the demands outstrip them — a common run-up to a later diagnosis and to burnout. Attention and self-control also dip with poor sleep, stress and low mood, which matters both for assessment and for thinking about what else might be going on.

Part Two: Assessment and management

Assessment

ADHD should be diagnosed after a full assessment by a suitably trained specialist.[1] Questionnaires are useful for screening but cannot make the diagnosis on their own. A good assessment builds up a developmental history (with someone who knew the person early on, where possible), looks at whether difficulties show up across different parts of life, and weighs up other explanations — anxiety, depression, trauma, poor sleep, substance use and ongoing stress all affect attention and often occur alongside a diagnosis of ADHD.[1,3] What counts is real impairment, not just the presence of the traits. In children, information from both home and school is essential, and it is worth taking the child's age within their school year into account explicitly.[7]

Medication: what it does, its limits, and its harms

Stimulants (methylphenidate, lisdexamfetamine) are first choice, with non-stimulants (atomoxetine, guanfacine) as alternatives.[1] Pooling the trials, stimulants come out with the best balance of benefit and tolerability in the short term, across all age groups.[18] They work by boosting dopamine and noradrenaline signalling, and they improve attention and impulse control in anyone who takes them — they are not fixing a known fault.[2]

It is worth being clear about the size of the short-term effect, because it is larger than for many psychiatric drugs. On symptom scales rated by clinicians in children, the average effect is big — a standardised difference of around 0.8 for methylphenidate and around 1.0 for amphetamines against placebo.[18] In responder terms, very roughly 55–75% of children improve clearly on a stimulant, against around 10–25% on placebo, which works out at a number needed to treat of about 2 to 3; most people who do not respond to one stimulant respond to the other.[19] The effect is noticeably smaller when symptoms are rated by teachers or by patients themselves rather than by clinicians.[18]

The short-term evidence is consistent, but not high quality: the main Cochrane review of methylphenidate in children rated the certainty of the evidence as low, and flagged frequent (if minor) side effects and the poor quality of many of the trials.[20] Good long-term trial evidence is lacking. In the MTA study, the early advantage of intensive medication had mostly faded by the 8-year follow-up, with no real differences between the groups on most measures.[21,22] This is often interpreted as evidence that the benefits of medication wash out over time, but another explanation is that by follow-up, treatment use had become mixed and self-selected across all

four arms, with most children taking medication at some point regardless of original assignment.

So most of what we know over the longer term comes from large registry studies. These show fewer adverse outcomes during the periods when people are on medication than when the same people are off it — for example 32–41% fewer criminal convictions in a Swedish study.[23] A fair summary is that medication reliably reduces core symptoms in the short term and is linked to better real-world outcomes across populations, while its long-term effect on the wider shape of someone's life is uncertain and varies from person to person.

Common side effects include reduced appetite, trouble sleeping, a faster heart rate and higher blood pressure, and a sense of emotional flatness; growth needs monitoring in children. Serious problems are uncommon, but stimulants and amphetamines carry a small risk of triggering psychotic symptoms, a little higher with amphetamines.[24] It is worth reviewing regularly, including physical checks, and planning occasional breaks from medication to see whether it is still needed.

Stopping, and missed doses

Stimulants do not cause the kind of discontinuation syndrome seen with antidepressants. Because they are short-acting, a missed dose mostly just means the symptoms return that day, and stimulants can usually be reduced or stopped without a formal taper and without physiological withdrawal at normal doses.[1] A minority notice end-of-dose rebound — a short spell of irritability or restlessness as the dose wears off. And abruptly stopping high doses, or misuse, can produce a temporary "crash" of fatigue, low mood, increased appetite and disturbed sleep, though this is not typical of ordinary therapeutic use.

The alpha-2 agonists are the real exception. Guanfacine and clonidine should not be stopped suddenly: abrupt discontinuation can cause rebound hypertension — occasionally severe — so they need to be tapered, and missed doses can bring symptoms back and raise blood pressure and heart rate.[25] Atomoxetine works gradually over several weeks rather than dose by dose, so a single missed dose matters less, and it is not associated with rebound or withdrawal.[1]

What helps besides medication

Structured routines, organisational and environmental support, explaining the condition clearly, parent training for children, and CBT-style approaches in adults all have a part to play; sleep, exercise, workload and stress all have a real effect on attention.[1,3] Changing the environment is often as important as medication — which fits the interaction point above — and support does not need to wait for a diagnosis.

The service context

Demand for assessment has far outstripped what services can provide, with long NHS waits and a rise in private assessments. When someone has been started on medication privately

and shared care is requested, the usual safeguards apply: an assessment that meets recognised standards, clear monitoring, agreed responsibilities between clinicians, and prescribing only within competence.[1] Local policies and waiting times change, so check the current position with the relevant service.

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