Dr Anthony MANDER

ADHD Newsletter 7

Course, Treatment Response and Neuroscience of ADHD in Adults

Course of Condition

Lecendreux et al (2019) have followed up nearly 500 families nine years after the first interview which screened for symptoms of a number of conditions including ADHD. At follow up the mean age was 18. Nearly 28% of those initially diagnosed with ADHD had continuing symptoms at follow-up. A further 1% not diagnosed at baseline now met the criteria. Persistence and new onsets were predicted by several baseline features including family history. Many children met subthreshold, rather than the full criteria for the condition. This needs to be considered by clinicians. There was some support for the concept of adult-onset ADHD.

Treatment Response

Stimulant medications have been used for hyperactive individuals for over 80 years. While the short-term effectiveness is well established, few longer-term studies beyond a year have been conducted. Hence, we cannot reliably establish the long-term benefits, or answer questions such as whether a person has 'grown-out' of their ADHD.

Two recent reports highlight the difficulty of conducting studies that can answer such important questions.

The Multimodality Treatment of ADHD study found that after 14 months, structured medication was superior to other treatment methods. The most recent follow-up examined medication usage, ADHD outcome and growth at age 25. Only 7% of the sample took their medication consistently. Because of this, no relationship could be shown between ADHD outcome and medication usage.

Matthijssen et al (2019) have reported on the first double-blind placebo-controlled discontinuation study in ADHD patients continuously treated with methylphenidate for at least 2 years. There were difficulties in recruiting enough patients because "many patients we approached did not want to participate". They argued they "knew it still worked". During the 7-week trial over 40% of those that discontinued worsened compared to under 16% of those who continued medication. This confirms the ongoing positive effect of medication. But what about the 60% who did not worsen when medication was stopped? Clinicians working in this area are very familiar with the slower relapse that occurs with many patients. This is presumably because well learnt routines under treatment are often maintained for a period without medication. Many individuals slowly slip into past chaotic behaviours.

Multiple other studies using large national or insurance databases are beginning to establish the long-term benefits of medication for those under treatment. Treated patients have fewer motor vehicle accidents, a lower risk of traumatic brain injury, less likelihood of engaging in criminal activity, and lower rates of suicidal behaviour and substance abuse. So, is there a long-term benefit of medication? Emphatically **YES!**

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Neuroscience of ADHD

The catecholamine hypothesis of ADHD centres around dopamine, adrenaline and noradrenaline. Dopamine is a precursor of noradrenaline. These neurotransmitters affect attention, alertness and arousal through a crucial balance essential for maintaining functioning and allowing response to acute demand. A hypothesis suggesting "too much" or "too little" of a single neurotransmitter does not explain the diversity or complexity of ADHD symptoms.

ADHD involves impaired neurotransmitter activity in:

- Frontal cortex responsible for executive function
- Limbic system responsible for emotional regulation
- Basal ganglia, which regulates communication within the brain. Deficiencies here lead to inattention or impulsivity
- Reticular activating system which is a major relay area where deficiencies lead to inattention, impulsivity or hyperactivity

ADHD may be the result of problems in one or more of these areas and may be due to a lack of adrenaline or noradrenaline (or its chemical constituents dopa and dopamine).

Catecholamines not only facilitate attention, they are essential to executive function. The pre-frontal cortex directs behaviours, thoughts, and feelings represented in working memory.

This representational knowledge is essential to fundamental cognitive abilities that allow us to:

- Inhibit inappropriate behaviours
- Regulate attention
- Monitor actions
- Plan and organise for the future

Deficits in these functions account for many common behavioural symptoms.

Stimulants work by causing the brain to synthesise more noradrenaline, whereas non-stimulants slow the rate at which it is broken down. There are important differences between methylphenidate and dexamphetamine.

Methylphenidate increases the synaptic concentration of dopamine by blocking pre-synaptic dopamine uptake.

Dexamphetamine also blocks dopamine reuptake but also increases production of both dopamine and noradrenaline. While both medications increase attention, the noradrenergic effects of dexamphetamine may contribute to energy and enhanced executive function.

Dopamine may be more essential to attaching attention; Noradrenaline may contribute more to executive function.

Strattera, through acting primarily on noradrenaline, has secondary effects of increasing pre-frontal cortical dopamine

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