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ADHD Newsletter 5 Evolving Concepts of Adult ADHD

Taken from Shaw, P. *Am J Psychiatry*,
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Concepts of ADHD in adulthood have changed greatly. Initially it was held that ADHD both began and essentially ended, in childhood. However, in the 1990's a series of prospective studies showed that around 15-20% of children continue to show the full syndrome into adulthood, and symptoms of inattention persist in a further 50%. Most recently, a series of studies found that the syndrome could have its onset in adolescence, or even adulthood - herein referred to as late-onset ADHD - challenging the accepted view of ADHD as a neurodevelopmental disorder with an invariable childhood onset.

The multimodal treatment study of ADHD suggested an onset in adolescence (ie after the accepted cut off from age 12) of 40% with a further 20% in adulthood. However, this startling prevalence of apparent late-onset ADHD plummeted to 3.3% as soon as three sensible constraints were implemented. First it was ensured that the symptoms caused impairment across multiple contexts.

Second, the symptoms were not better explained by substance misuse or another mental disorder. Finally, the exact chronology of symptom course was mapped to ensure that the onset was indeed after age 12. The prevalence of late-onset ADHD fell further to 2% if those with childhood symptoms that fell near the diagnostic threshold were excluded, suggesting that their ADHD was as much late- recognised as late-onset.

This work makes it clear that there is a small group of genuine late-onset ADHD cases. The prevalence is between 2 and 3.3% which is close to the 2.7% estimate from the Dunedin birth cohort study conducted in New Zealand. A prevalence of 2-3% is hardly trivial, even if the late onset syndrome frequently arises in the context of complex psychopathology.

The lesson for us as clinicians, as is so often the case in medicine, is to keep an open mind. Adults lives and hopes for the future can be transformed by the correct recognition of the

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condition and the implementation of treatment which has few side effects and no major complications as long as cardiovascular parameters are monitored.

Taken from Faraone, S. et al, Nature Review, vol 1, 2015

ADHD is a persistent neurodevelopmental disorder that affects 5% of children and adolescents and 2.5% of adults worldwide. Throughout an individual's lifetime, ADHD can increase the risk of other psychiatric disorders, educational and occupational failure, accidents, criminality, social disability and addictions. No single risk factor is necessary or sufficient to cause ADHD. In most cases ADHD arises from several genetic and environmental risk factors that each have a small effect and act together to increase susceptibility. The multifactorial causation of ADHD is consistent with the heterogeneity of the disorder, which is shown by its extensive psychiatric comorbidity, its multiple domains of neurocognitive impairment, and the wide range of structural and functional brain anomalies associated with it.

The diagnosis of ADHD is reliable and valid when evaluated with standard criteria for psychiatric disorders. Rating scales and clinical interviews facilitate diagnosis and aid screening. The expression of symptoms varies as a function of patient developmental stage and social and academic contexts.

Although there are no curative treatments for ADHD, evidence-based treatments can markedly reduce its symptoms and associated impairments. For example, medications are efficacious and normally well tolerated, and various non-pharmacological approaches are also valuable. Ongoing clinical and neurobiological research holds the promise of advancing diagnostic and therapeutic approaches to ADHD

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