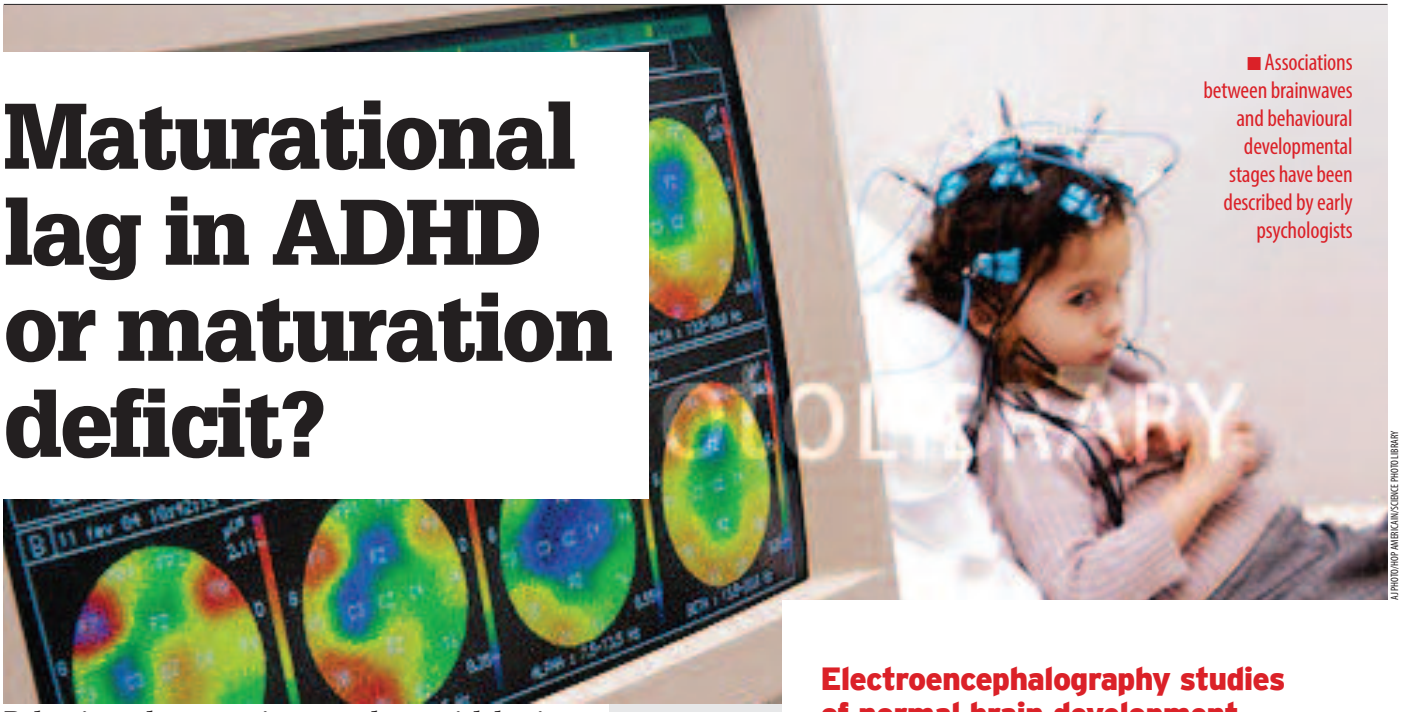
ADHD IN PRACTICE 2014; Vol 6 No 1

Maturational lag in ADHD or maturation deficit?



■ Associations between brainwaves and behavioural developmental stages have been described by early psychologists

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Beverley Steffert PhD Psychologist and Director, Learning Recovery, Cambridge

Tony Steffert BSc MA QEEG Technician, Computing and Communications Department, The Open University, Milton Keynes, UK

Behavioural maturation correlates with brain-based development. Maturation is not a linear story, there is an ebb and flow that has to be precisely timed for normal development – too fast can be just as damaging as too slow. In fact, a slower development of the prefrontal cortex can result in a cortical thickness that has been associated with a higher intelligence quotient (IQ) and 'late bloomers',[1] while a rapid development of the brain is associated with autism.[2] Progressive increases in the thickness of pyramidal cells and synaptic density, peak and ebb over childhood.[3] Brain weight and head circumference increase and plateau at various ages.

The connections between the functional and structural brain areas develop a covering sheath (myelination), which occurs at different rates, with some parts (the reticular formation) completely myelinated by eight to ten years[4] of age. The frontal lobes are the last to become myelinated and are, therefore, only then functionally mature; sometimes this does not happen until adulthood. These areas are the most heavily involved in executive function, so a lack of myelination of the nerve fibres means less efficient connection between frontal lobes and other areas of the brain and lower levels of executive functioning. The areas that develop with age are the ventral medial prefrontal cortex, the cingulate cortex, the ventral striatum, insular, inferior temporal gyrus and posterior parietal cortex, all with implications for learning and behaviour.[5] Postpubertal development is characterised by a 'pruning back' of excess grey matter and the number of synaptic connections in a posterior to frontal manner. Inefficient myelination is associated with several disorders, including neurodevelopmental conditions.[6]

Electroencephalography studies of normal brain development

The electroencephalography (EEG) reflects these developmental studies in that various measures of frequency, recorded as an EEG wave, increase with age, from low frequency (four to seven Hertz per second) to higher (15 to 20 Hertz per second). As this happens, the ability to maintain alertness, attention and memory increases. There is a point where higher frequency (>21 Hertz) is associated with over-arousal states, such as anxiety or anger. Functional areas of the brain that are connected by myelinated pathways show a coherence in the EEG. Hudspeth and Pribram[7] and Thatcher[8] have shown associations between various stages of normal brain development, including brainwaves and the behavioural developmental stages described by early psychologists, such as Piaget.[9]

When normal development is compromised

This normal pattern of development can be compromised by many factors from genetics to environmental toxins, pre- and postnatal nutrition, birth traumas, head injuries and lack of normal experiences to stimulate developmental trajectories. When these or other inhibitors compromise normal development, a neurodevelopmental disorder is diagnosed; the new *Diagnostic and Statistical Manual for Mental Health*, 5th edition (DSM-5)[10] now classifies ADHD as a neurodevelopmental disorder.

A neurodevelopmental disorder implies that the ADHD brain will show early signs of anomalous development and Oades[11] does indeed show a right frontal impairment in children diagnosed with ADHD, especially when they are engaging in stop and delay tasks (inhibitory).[12] Oades suggested this to be evidence for a dislocation in prepubertal

developmental of sensory, spatial, holistic, memory and language processes, which all need efficient division between their homologous areas in the opposite hemispheres. Castellanos *et al*[13] showed a disruption in the circuitry involved in the inhibitory control base of the prefrontal cortex and the emotional and motor areas based within the basal ganglia and cerebellum. All these areas were 3–4% lower in volume than controls. Thus, the inhibitory areas of the brain cannot control the hyperactivity and impulsivity of the subcortical structures. Of course, other aspects of executive functioning, such as planning and organising, would also be affected. The lower volume of brain matter would be expressed in electrochemistry. Using a large sample size, Barry *et al*[14] described just that; ADHD diagnosed children have either an excess of low frequency EEG or an abnormal ratio between low and high frequency. Monastra *et al*[15] have shown an abnormality of brainwaves with an excess of low frequency, which can co-exist with a deficit of high frequency. Kropotov *et al*[16] identified and suggested a further third subtype of ADHD, defined by an excess of extremely high frequency (>21 Hertz). Low frequency correlates with low arousal and lethargy, while higher frequency is associated with alertness and concentration in all mammals. Thus, the prevailing consensus has been that there is a continuum of arousal from low to high in brainwave frequency, with higher frequencies increasing with age and that this is abnormal in ADHD. However, Arns *et al*[17] more recently showed that in six to 13-year-old children, this ratio, when measured over the last decade, declines in significance and may be the result of the 'winner's curse'; that is, fewer studies find less evidence of the original finding as time goes on. Among other possibilities, this could be due to more precise measurement as equipment becomes more accurate and, possibly, although the authors do not suggest this, better understanding and training of ADHD symptoms in the last decade. Nevertheless, there are well-validated brain-based differences between ADHD and non-ADHD brains, which would be reflected in the EEG. Dupuy *et al*[18] outline several topographical differences between ADHD typologies, although there was not a global difference in the EEG of the ADHD combined versus the inattentive ADHD type, probably due to the small sample size. But these are still meaningful differences showing individual variation. However, there were sufficient EEG differences between boys and girls to suggest all further research into ADHD should not be based on mixed-sex studies.

The question is, do these observed, brain-based differences remediate with age? Burke and Edge[19] separated EEG studies into developmental deviation and developmental lag. The EEGs that result from central nervous system functioning abnormalities are considered to be in the deviation category and, therefore, unlikely to mature, while EEG patterns similar to that of younger children (increased slow activity with decreased fast wave activity, increased frontal theta, and increased delta in temporal and parietal lobes) are considered maturational. This seems a logical step, but some of these deviations are classified by Mueller *et al*[20] and Steffert and Steffert[21] as a further subtyping of ADHD for example, the alpha subtype; there seems little basis for categorising these EEG patterns as deviant. Burke and Edge's conclusion was that there is support for both models.

Maturation lag or maturational deviancy hardly matters at school since low arousal, characterised by an excess of low frequency, especially in the frontal lobes, is not the state needed for school, and explains the constant refrain in teacher's reports along the lines of 'needs to concentrate', 'must stop rushing his work', 'must complete projects/hand them in' and an array of complaints describing the lack of mental energy ADHD children have when the task is repetitive, difficult, lengthy or in the child's words, 'boring'.

There are well-validated brain-based differences between ADHD and non-ADHD brains

Such pressure to change is a stress in itself and leads to the argument that an immature brain, with underlying prefrontal cortex pathophysiology, is likely to react to stress, which itself produces abnormal neurotransmission.

Certainly, anomalies in neurotransmission, which have an influence on ADHD symptomology, have long been shown and the deficits in the dopamine system have led to the widespread use of methylphenidate, which increases dopamine turnover. Other neurotransmitter systems, such as the norepinephrine and serotonin systems, have also been shown to be deficient, with drugs developed to target these systems. Nakao *et al*[22] surveyed research to show that both medication and age independently normalise the EEG in that dopamine enhancement increases activation in the basal ganglia, as well as functional connectivity with frontal-cortical and cerebellular regions. As a consequence, it is suggested that the activation of these compromised areas by methylphenidate helps the individual use and, thus, repair those areas. The authors do admit drawbacks in this claim in terms of sample size, uneven groups, lack of randomisation, different terminology and measures in some of the studies they surveyed. Thus, the suggestion that drugs can help repair compromised brains is eschewed by holistically minded therapists in favour of

therapies designed to teach long-term coping mechanisms. Questions to be asked here are: what happens when the drug is terminated; what side effects are there from long-term use; and why do stimulants only work for 76%[23] of ADHD diagnosed individuals? Antle *et al*[24] note that decreased appetite and sleep are widespread side effects. It is also the case that some ADHD subtypes will become worse with medication – for example, stimulant medication increases beta brainwaves, so would be contraindicated for the excess of beta types of ADHD. Woodard[25] explicitly notes contraindication for those who are anxious, agitated or tense.

The big question

The question researchers, parents and children themselves would like answered is: is ADHD a developmental lag and will the affected children grow out of it, or is this a developmental deficit that has to be coped with using medical or psychological interventions? The developmental lag supporters point to a brain and behaviour that would be quite normal in a child two or three years younger.[26,27] This means that as the child ages, increasing levels of higher brainwave frequency would progress naturally to the brainwaves mediating attention, alertness and awareness.

There are patterns of cortical activity and abnormality in some ADHD children that are not normal at any age

An interesting correlation, suggested by Arns *et al*,[17] is the decline in sleep duration with age observed in ADHD diagnosed children. They suggest a study of the theta/beta ratio and sleep duration in normal children would give some insight, since sleep problems, onset and duration are the most commonly noted symptoms of children diagnosed with ADHD.[28]

But there are patterns of cortical activity and abnormality in some ADHD children that are not normal at any age and, therefore, not likely to be 'grown out of'. Rubia *et al*[12] reviewed the brain imaging literature on ADHD to conclude that there is support for minor structural changes and reduced functional activation in frontal and caudate areas, especially on the right side of the brain. Berger *et al*, showed that in a continuous performance test, some aspects of performance of ADHD children matched that of normal controls, who were one to three years younger. The inhibitory control aspect showed a different trajectory.[27]

Steffensson *et al*[29] analysed a large twin sample suggesting the existence of at least two pathways to ADHD, one through a predisposition to a maturational lag and another specific to ADHD, which may be more vulnerable to negative environmental influences and have more of the troubling symptoms (to parents and teachers) of hyperactivity and impulsivity.

Part of the reason that the question has not been answered is methodological. The grouping of a wide range of ages into a mean, which is then compared with a control group of similar mean but different ranges of age, ignores individual development in brain maturation, which Oades shows can be quite different among similar aged children. He also showed that some brain-based changes went on past 30 years.[11] Taylor[30] makes the point that if only one brain-based step in a behavioural sequence is not developed, others may evolve in a desynchronised way and the entire function may lag behind. For example, the functions of inhibitory control, attention and motor activity may need a step-wise unfolding, which, if not developed normally, may always be subject to some inadequacy, but it would be hard to decide which was the initial lag.

Burke and Edge[19] note that co-morbidities, and even personality problems, are often not taken account of and some children with severe ADHD will, through increasing stress (for example, teacher criticism and parental hostility) develop more ADHD symptoms.

El-Sayed *et al*[31] showed that girls' brains mature sooner than boys', which may alter the concept of maturational lag and perhaps explain the differential diagnosis between girls and boys. It is certainly evidence for maturation of the brain's attention and learning systems, since boys catch up. But different parts of the brain can develop in different sequences, time and age, making a comparison difficult. To add to this, the diagnosis of ADHD is often made on fairly subjective criteria using interviews and checklists with parents and teachers, or sometimes a continuous performance attention test, which can be vulnerable to lack of motivation or anxiety.

If ADHD is not a homogenous disorder with the same underlying neurodevelopmental process, the concept of an overall maturational lag is not appropriate, although the strong genetic basis suggests some similarity. There may be many maturational lags in different parts of the brain. The maturation of the brain is mostly genetically determined but nature has a role both during foetal development and in later life experiences. Longitudinal and cross-sectional studies of ADHD cannot yet capture the individual spurts and lags that all these influences have on development of the brain and behaviour. Perhaps they could be regarded as variances of normality, and therapies really just a cure for childhood.

Do ADHD symptoms decrease with age?

The question becomes which symptom and which lag? Many studies now attest to the clinical observation of remediation or a decrease in some symptoms with age. Figures differ – in Weiss *et al*,[32] a 15-year follow-up study found that 66% of children diagnosed with ADHD still fulfilled the criteria in adulthood, while Mannuzza *et al*[33] found a 42% rate of persistence over 20 years. Barkley[34] found that 72% of children still met the ADHD criteria eight years later. Weiss *et al*,[32] in a careful longitudinal study, found that hyperactivity continues into adulthood, even if it is transformed to restlessness and distractibility. Farone *et al*[35] showed that a third of ADHD children will continue to have their difficulties in adulthood. Similarly, Biederman *et al*[36] found 85% persistence of ADHD symptoms in a four-year follow-up study. This still means some reduction of ADHD symptoms, which is estimated by Hill and Schoener[37] to decline by 50% every five years.

Additionally, some quantitative EEG studies have been able to show a reduction in low frequency brainwaves after treatment, like neurofeedback,[38] which correlates with attentional ability, reduced impulsiveness and the underlying executive function deficits.

If we accept figures of up to a third of ADHD diagnosed children developing normal behaviour in adulthood, with concomitant brain-based correlations towards normality, the question then becomes – which children and which symptoms do or do not remediate, and under what conditions?

Is there a continuum of developmental deviancy, from just EEG excess of slow frequency to smaller frontal cortex, right caudate nucleus and other observed brain differences, or is the EEG a reflection of underlying structural and functional deviances anyway? The above studies suggest that inhibitory control[34] is the main defining feature of ADHD and the symptom least likely to remediate, even if it changes from childhood impulsiveness to delay aversion. Inhibitory control has an electrophysiological correlation; the P300,[21] which is shown to be consistently associated with ADHD diagnoses.

Attempts at ameliorating ADHD symptoms

Studies have shown that temperament (predispositions to anxiety, extroversion or conscientiousness)[39] makes a difference to outcome from preschool to secondary school, which is likely to be carried into adulthood.

Given these heterogeneous studies, it seems that the more 'normal' neurological profile of the AHHD child, with little clinical co-morbidity, is the most likely to be able to 'grow out' of ADHD. However, much depends on the circumstances that seem conducive to success in later life. In an environment where doing things quickly is necessary, novelty abounds; there is variety and change. Plenty of opportunity for hands-on and physical activity would better allow an ADHD profile to remediate, as would the support of an accommodating partner, efficient secretary and other supports.

Diamond's study,[40] giving rats an enriched environment conducive to their exploratory natures, allowed the rats to develop a cortical thickness compared to their unfortunate controls, who were fed and housed adequately, but were not given new toys and wheels everyday; this is the basis for this supposition. Hyperactivity, impulsivity distractibility and lack of attention may, in the right environmental niche, become spontaneity, flexibility, imagination, rapid summarising and an ability to see the big picture unfettered by petty details, that is, the visionary tycoon or Napoleonic general.

This raises the last question – are the ADHD children who 'grow out of it' simply the ones whose circumstances allowed remediation? [AU: Ok?]. This is a new question inviting a new study. It is certainly the case according to Hallowell,[41] that many overworked managers have developed attention deficit traits, simply from the overload and stress they have in keeping focussed, staying organised, setting priorities and managing time in the competitive, capitalistic world they work in. These were normal people who had no earlier diagnosis of ADHD; some were gifted and talented but, in the workplace, were overwhelmed with fear – fear of making mistakes, not meeting deadlines, losing their job or position (which bred exactly the same tendency to difficulty in focussing) and making impulsive judgments that the ADHD individual exhibits. He or she loses flexibility, reverts to 'black and white' thinking, and loses good problem-solving ability and creativity, while mistrust and anger become the dominant emotions. Alcohol and drugs can become necessary props. Health suffers as the brain and body become overwhelmed and now, we have the mirror image of the ADHD child in an adult, who did not previously have any symptoms of ADHD.

Intervention in childhood could and should prevent the ADHD child becoming an ADHD adult

The take home message here is that interventions in childhood could and should prevent the ADHD child becoming an ADHD adult and at least able to use coping strategies. Environmental engineering may be out of the question but there are known interventions that work: cognitive behavioural therapy; behaviour modification with

timers and schedules; modifying classroom environments; one-to-one teaching for core subjects; social and thinking skills programmes; attention and memory training;[42] neurofeedback;[43] judicious medication (even in adults);[22] and nutritional strategies.[44] All have their place and all are needed at one point or another.

Conclusion

While the question of maturational lag versus deviance becomes less relevant than the question of which environment and interventions can best suit the development of the ADHD child, and help them turn their negative traits into positive outcomes, it seems from the present research that the fewer the symptoms and co-morbidities, the more likely the ADHD child will be able to 'grow out' of it. Interventions that increase myelination growth and, therefore, more efficient executive functions, are most likely to help the reduction of the developmental lag of prefrontal cortices.

We suggest that research which may better answer the question of who can overcome ADHD symptoms, and how and why, would use the opposite direction of the quoted studies and would involve a large sample of normally productive people in occupations conducive to ADHD (courier, sports and racing) versus non-conducive (accountancy and bookkeeping), sampling their background for the presence of any ADHD symptoms and how they might have overcome any of them. Historical accounts of famous people who have achieved something and who are judged to have had ADHD suggest this may be a productive approach; there are many in the ADHD pantheon ■

Declaration of interest
The authors declare that there is no conflict of interest.

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Key points

- Functional and structural areas of the brain develop at different rates. This normal brain development can be compromised by many factors, including genetics, environmental toxins and head injuries.
- Current research is trying to establish whether ADHD can be regarded as a lag in maturation of specific brain areas or as a deviance from the norm. Due to various methodological problems, this question is difficult to answer.
- Various studies suggest that children with ADHD who have fewer symptoms and co-morbidities are likely to progress out of ADHD upon reaching adulthood.
- An investigation of adults who were diagnosed with ADHD in childhood could be useful in order to gain an insight into how they used their negative traits positively.