



ADHD diet and the brain

In the face of mounting ADHD diagnosis and widespread prescription of potent psychoactives, a GP and a psychologist discuss causation and natural treatment options.

The general practitioner: Dr Robyn Cosford

Robyn Cosford, MBBS(Hons), FACNEM, is Conjoint Lecturer, University of Newcastle and a practising integrative GP. This article is an extract from her lecture to the MINDD conference, 19 May, Sydney

ADHD does not exist alone, but is occurring in increasing incidence in our modern children in the context of the changes in modern society and as part of a wider spectrum of children's disorders which includes oppositional defiant disorder (ODD), obsessive-compulsive disorder (OCD), conduct disorder (CD) and autistic spectrum disorder (ASD). A 2001 West Australian study of 4500 children found 14 per cent with a mental-health problem, and attention-deficit disorder in 11 per cent. These figures are echoed in a more recent US study of 76,662 children, which

found one-fifth of the children, most often boys, had a psychiatric disorder, 94 per cent of which were adjustment disorder, ADHD, ODD, CD and depression. Other neurodevelopmental disorders are also increasing: developmental impairment of speech and language is now the most common neurodevelopmental condition among children under five in the UK, affecting 5–8 per cent of the population in 2000 and, interestingly, toddlers with speech delay are more likely to have social and emotional adjustment problems and dysfunction.

Rising recurrent-infection, allergy and asthma incidence

Concurrent with this increase in mental-health and neurodevelopmental disorders has been a general shift in the pattern of children's illnesses over the past two decades. The childhood infectious diseases of the past have been replaced with chronic recurrent otitis media (AOM) and glue ear (OME), asthma, eczema, gastro-enteritis and food allergies.

The previous incidence of respiratory-tract infections, childhood infectious diseases, tonsillitis and AOM peaked at age four-to-six years old, and AOM media was uncommon. Progressively, we see the incidence increasing and peak incidence shifting to ages six-to-12 months, so that by 2000, AOM was found in a large US study to occur in 48 per cent of children by six months of age, 79 per cent by 12 months and 91 per cent by 24 months.

Similarly, asthma has doubled over the past 20 years; allergies are increasing, with 54 per cent of children in the UK having atopic symptoms at some time; and atopic eczema now affecting about 18 per cent of children in their first two years of life, up from two per cent a few decades ago.

Food allergies have become a particular problem: peanut allergy has dramatically increased from a rare allergic phenomena to affecting one in 50 children and one in 400 adults, in a 2003 study. Similarly, other food allergies now affect nearly 30 per cent of children. Infants are also being affected



by food allergies, with 70 per cent of infants with colic improving when their breastfeeding mothers are placed on a strict allergy-free diet in one study.

In addition to infections and allergies, there is an apparent increase in GI dysfunction, beginning in infancy. Gastrointestinal reflux has increased in incidence in infants as well as in adults in recent years, and is now a major cause of illness and failure to thrive, particularly in neurologically impaired children. Reflux has also been associated with an increased incidence of asthma, respiratory infections, AOM and childhood chronic sinusitis.

The link between immunity and neurodevelopment

There is an apparent correlation between immunological disorders and infections and the neurodevelopmental disorders. A recent study of over 600 preschoolers with asthma found greater behavioural problems with asthmatic preschoolers than non-asthmatics, particularly if their parents had not had asthma. An Australian study of 12,000 children found

a significant association between asthma, allergies, ear disorders and other mental-health disorders in boys with a speech disorder. In girls, recurrent urinary-tract infections were associated. Recurrent AOM in infancy is correlated with a greater incidence of ADHD, increased distractibility later in life, later low-IQ scores, poor performance on tests of reading, spelling, maths, increased retention in grade, increased attention deficits, and increased behaviour problems at school. There is also a high correlation between the prevalence of AOM and autism, with earlier onset and increased incidence each correlated with a more severe form of autism. A 2005 study of 206 autistic children found a history of nearly 10 bouts per year of AOM with an average of 12 courses of antibiotics, usually Augmentin.

AOM is now the most commonly diagnosed illness in children, and is one of the most common reasons for antibiotic prescribing, with males under 15-years-old the highest antibiotic usage group.

Several studies have linked prenatal and early infancy antibiotics to later

allergy and asthma, showing a greater than twofold increase in the incidence of asthma and an increase in allergies in children who have received at least one antibiotic by the age of 12 months old. In comparison, children who have had fewer vaccines, fewer antibiotics and have diets containing live lactobacilli have a reduced incidence of atopy.

The role of antibiotics and the gut

Antibiotics are known to induce change in GI flora, and this change is thought to be the reason behind the Th2 shift, manifesting as increased asthma and allergy. The loss of protective flora, dysbiosis, clostridial and candidal overgrowth is well documented. Antibiotics can also induce increased intestinal permeability as a consequence of the dysbiosis, and intestinal permeability is a documented marker for food allergies. The mechanism is via the contact of viral, bacterial and food antigens with the gut wall, inducing inflammation and impairing enzymatic digestion, with resultant transport of macromolecules, bacteria and toxins



across the gut wall. These molecules then pass through the gut-associated lymphoid tissue (GALT) and portal circulation, resulting in an overriding of the normal tolerogenic signals and an allergenic stimulus that favours allergenic reactions. Increased intestinal permeability has been found in various auto-immune disorders, inflammatory bowel disease, post gastro-enteritis, eczema, ADHD and autism.

There is a clear relation between GI dysfunction and behaviour, the so-called 'gut-brain axis' as Dr Andrew Wakefield expressed in the *Journal of Pediatric Gastroenterology and Nutrition*: 'gut-brain interactions may be central to abnormal neural development and the subsequent expression of aberrant behaviours'. Numerous studies have confirmed significant GI dysfunction in children with autism and, anecdotally, also in ADHD, with colonic dysbiosis commonly noted. In autism — the more severe end of the neurodevelopmental behavioural disorder spectrum — inflammation of the entire GI tract has been noted, along with impairment of disaccharidase enzyme function. Intestinal permeability, as previously noted, is also increased.

Given these GI disorders, it is no surprise that food reactions are noted in ADHD. In a subset of children with ADHD, food allergies and or sensitivities are significant, and EEG changes have been noted to occur immediately following the ingestion of a previously sensitising food. Low reactive foods have been found clinically beneficial in a subset of children, particularly if there is a strong atopic history, gut symptoms or measured increased intestinal permeability.

Reactions to food additives, first documented and popularised by Ben Feingold in the 1950s, have been confirmed: in one study, 50 per cent of children reacted to calcium propionate

with irritability and inattention. In a recent UK study of 2000 preschool children, it was noted that the 'observed effect of food additives and colorings on hyperactivity in this community sample is substantial, at least for parent ratings', despite the dose for additives being 'on the low side', the effect also being seen in children with neither allergies nor hyperactivity. As the researchers commented: 'We showed there was an effect on perfectly normal children. If that is confirmed by further research, then there is a public health issue'. In a further review of 23 controlled studies, 17 found evidence that some behaviour significantly worsens after children consume artificial colors or some foods, notably milk or wheat: 'Limited research with such tools as electro-encephalogram (EEG) indicates that certain foods trigger physiological changes in sensitive individuals'.

Eating too modern a diet

Perhaps the issue is not so much whether diet has an effect on children with ADHD, as the modern western diet itself. In comparison to how man has eaten for millennia, the modern highly processed diet has little fruit and vegetables and high glycaemic load, with predominantly high-quantity, low-quality refined cereal-based carbohydrates, higher omega-6 to omega-3 essential fatty acid (EFA) ratio, decreased micronutrient intake, increased acidity, increased sodium and decreased fibre.

Modern dairy is almost exclusively Friesian cattle or similar (A1-type casein) processed by modern methods of pasteurisation and homogenisation, in comparison to primitive and traditional dairy from goats, sheep or, later, Jersey cattle or similar (A2-type casein), which was soured by various processes.

Overconsumption of food (more calories, not necessarily better nutrition) is another feature of our modern diet:

in children aged 10 to 15 years between 1985 to 1995, there was a 15 per cent increase in the caloric intake in boys and 12 per cent in girls; the main increase being in refined carbohydrates from cereal-based foods, confectionary, non-alcoholic beverages and sugar products.

It would appear that children in the ADHD/autism neurodevelopmental spectrum have an increased sensitivity to particular food components of the modern diet. As previously mentioned, one of the most common reactive foods in children with ADHD is dairy. A high intake of cow's milk has been correlated with behavioural disorders and aggression in children, and a reduced consumption of dairy resulted in a significant reduction in the incidence of antisocial behaviour in these children. Interestingly, young males, notably adolescent males, have the highest average intake of dairy as milk. Dairy intolerance is also associated with sleep difficulties in infancy, GI dysfunction and constipation, and recurrent serous otitis media, nasal congestion and sinusitis, all common symptoms in ADHD.

Similarly, another western-society staple, wheat, has been found to be reactive in some ADHD children, and is being increasingly noted to produce adverse effects in a variety of other conditions, including IBS and autism. The peptide in wheat (gluten) is particularly antigenic and has a well-documented association with neurologic disease in some individuals, including schizophrenia, epilepsy with intracerebral calcifications and cryptogenic neurologic disease.

The importance of omega-3

Since the Industrial Revolution there has been a significant increase in the omega-6:omega-3 EFA ratio (now 20:1) and trans-fatty acids in the diet. Omega-3 EFA are essential for the growth and functional development of



the brain in infants, and maintenance of normal brain function in adults. As the turnover of DHA in the brain is rapid, the conversion of the shorter-chain precursor α -linolenic acid to EPA and DHA (functional forms of omega-3 EFA) is inefficient, and omega-6 EFA compete with omega-3 EFA for the same enzyme, DHA deficiencies are common in general, and have been found to be common in ADHD and aggressive hostility. Numerous studies have confirmed the benefits of supplementation with, for example, fewer learning problems, less hyperactivity, overall improved academic achievement and ability in mathematics. In dyspraxias, academic and cognitive gains (reading, spelling, reduction of ADHD symptoms) have been achieved with omega-3 supplementation.

High sucrose intake also characterises our modern western diet. Sucrose intake has been demonstrated to acutely improve performance, however, the adverse effect of sucrose on neurobehavioural parameters may be seen more in habitual sucrose intake than acute intake, where there is a significant negative correlation between habitual sucrose intake and continuous performance test scores. Diets high in the proportion of refined carbohydrate have been significantly negatively correlated with intelligence and school-achievement scores.

Conclusion

A recent drug-effectiveness review performed by the Oregon State University in 2005 found that there was little evidence that the drugs used to treat ADD were either effective or safe. Commenting on this, Dr Jon Juriedini, Head of the Psychological Medicine Department at the Adelaide Women's and Children's Hospital stated that 'I think if there is not very good evidence for diet and there is not very good evidence for drugs, then I'd choose diet,

because at least we do less harm'.

The issue is not so much whether diet has an impact on ADHD, as what have we done in our modern diet that is impacting on health in general. The children with ADHD autism and the like can be regarded as the modern equivalent of the miner's canaries, the modern diet being the poison they are warning us against. The solution is not to restrict this food or that food, but to restore the totality of the diet to what humans have eaten for millennia.

There is a need to restore the diets of these children, and society in general, as far as possible to the traditional

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pre-Industrial Revolution model with no processing of foods; predominant protein base (plant and animal); little complex carbohydrate with no refined sugar, natural sugars from sources such as honey, dates and figs; increased fish, nuts and seeds; natural fats, high fruit and vegetable intake, soured dairy products particularly goats and sheep's milk products and grains other than wheat, prepared by traditional processes of soaking and fermenting. We are what we eat, and we are eating contrary to our genetic inheritance and reaping the consequences of so doing.

References

Available with the CD/DVD of this presentation from Northern Beaches Care Centre, Sydney (telephone 02 99799444, fax 02 99799016)

The psychologist: Dr Jacques Duff

Jacques Duff, BA(Psych), GradDipAppSc(Psych), MAPS, MAAAPB, MECNS, MISNR, AMACNEM, is completing a PhD in Clinical Neuroscience at the Brain Sciences Institute, Swinburne University of Technology

Attention Deficit Hyperactivity Disorder (ADHD) is a relatively common behavioural disorder that substantially interferes with a child's ability to function normally at home and in school. It is estimated that about 5–10 per cent of school children may be affected by ADHD¹⁻³, which is characterised by a mixture of difficulties in a number of areas. These include concentration, distractibility, forgetfulness, hyperactivity, fidgetiness, and general ability to sustain mental effort.^{3,4} ADHD has become a major public-health concern worldwide; an Australian national survey finding in 2000 that 11 per cent of Australian children and adolescents would meet the diagnostic criteria for ADHD, with 23 per cent having one of the childhood mental disorders surveyed.⁴

Despite an extensive body of research from various disciplines, there has been little consensus on the interrelationships between nutritional and metabolic anomalies, brain morphology, neurochemistry, neurophysiology and behavioural manifestations of the disorder. There is no single aetiology and no single laboratory test for ADHD. The diversity of the proposed causal factors and the range of core and associated behaviours suggests that ADHD may be a catch-all acronym for a range of underlying disorders with a wide range of behavioural manifestations.⁵



The neurological picture

Neuropsychological studies of children with ADHD have found consistent deficits in executive function, which are thought to be associated with frontal lobe deficits, motor inhibition and deficits in subcortical and parietal brain regions. Findings have been largely interpreted to suggest that covert orienting to cues mediated by the posterior attention system may be normal, but that overt control of attention mediated by the anterior attentional system may be deficient in children with ADHD.⁶⁻⁸

Structural neuroimaging studies have found inconsistent or conflicting results between different research groups in the morphology of frontal sub-cortical areas in ADHD, possibly due to cohort effects or differences in neuroimaging equipment and methodologies.⁹⁻¹¹ Despite controversial structural neuroimaging findings, there is a general consensus that ADHD may be associated with dysfunction of fronto-striatal networks and that the relevant regulatory circuits include the prefrontal cortex and the basal ganglia, which are modulated by dopaminergic innervation from the midbrain.¹²

Functional neuroimaging, such as PET^{13,14} and SPECT¹⁵⁻¹⁹ and fMRI studies²⁰⁻²², have been used to investigate the differences in function of cortical and specific areas of the brain associated with various cognitive tasks between ADHD and normal subjects. However, the findings have been frequently inconclusive and sometimes contradictory. On the other hand, functional electrophysiology can evaluate changes in brain activation with high temporal resolution, although the techniques used are limited to investigating cortical areas. Since the temporal resolution is in the order of milliseconds, small transient changes in brain electrical activity evoked by a stimulus can be resolved and studied.

Reduced amplitude of the parietal-evoked response potential (ERP) — the P300 component — when ADHD subjects attend to target stimuli, is the most consistent finding from ERP studies. This has been interpreted as suggesting that the brains of children with ADHD may be less reactive than normal to stimuli under task conditions. It may also reflect diminished deployment of attentional capacity and deficits in the allocation of attentional

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resources in later stages of stimulus processing.

Quantitative EEG findings of increased slow-wave (delta and theta) brain electrical activity and increased alpha and decreased fast-wave (beta) activity has been interpreted as suggesting cortical underarousal and reduced information processing.²³⁻²⁷ However, there are also studies showing increased beta activity in 13–20 per cent of children with ADHD compared with normal controls, which suggests increased mental activity and hyper-arousal.^{24,28,29} Chabot concluded that their data suggested both hypo-arousal or hyper-arousal amongst ADHD subjects, and that patterns of the quantitative EEG of children with ADHD suggested a deviation from normal development, more so than a maturational lag.²⁴

While these studies highlight the neurophysiological differences between children with ADHD and normal controls, they do not elucidate the

aetiology of ADHD. Although ADHD and associated neurodevelopmental disorders are heterogeneous, there may be common denominators.

Interplay of genes and nutrition

Although there is a genetic component in the aetiology of ADHD, mostly related to dopamine-receptor genes³⁰⁻³³, genetics alone cannot explain the recent increases in ADHD incidence. Several studies have highlighted the possible nutrigenomic factors responsible for ADHD and neurodevelopmental disorders. According to the NIH and WHO, the omega-6: omega-3 consumption ratio is estimated to be 20–40 times higher in the modern diet than in our evolutionary hunter-gatherer diet.³⁴ In particular, there is evidence of docosahexaenoic acid (DHA) deficiencies in the red cells of children with ADHD^{35,36}, dyslexia and autism.^{37,38} DHA makes up 30 per cent of the dry volume of brain cells³⁹⁻⁴¹, controls the migration of immature neurons to the cortical plate in corticogenesis, and is concentrated in synapses where it modulates the synthesis transport and release of neurotransmitters.⁴² There is also evidence of zinc⁴³⁻⁴⁶ and magnesium^{47,48} deficiencies in ADHD, and a rationale for the integrative management of these disorders has been proposed.^{35,49,50} There is evidence that fish-oil, zinc⁵¹⁻⁵⁴ and magnesium^{55,56} supplementation benefits children with ADHD and dyslexia.^{50,57-59}

Introducing neurotherapy

However, while improving diet and nutrient supplementation may improve symptoms of ADHD, the best-researched treatment for ADHD to date involves retraining the abnormal brainwave patterns with neurotherapy. Neurotherapy is operant conditioning of the EEG and has consistently been shown in over 120 studies to significantly improve or normalise ADHD symptoms



in over 85 per cent of children with ADHD and learning difficulties. There are now several well-designed effectiveness studies directly comparing neurotherapy to Ritalin that have found neurotherapy to be as effective, if not superior, to Ritalin.⁶⁰⁻⁶⁶ The treatment effects are permanent, and with no adverse effects after an average treatment requiring 40 sessions over 3-4 months.

- Neurotherapy will be discussed in a subsequent issue of the *JCM*

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