Health hazards in the built environment and their relationship to childhood neurobehavioural disorders – Part 1

Mild iodine deficiency and the need for iodine supplementation during pregnancy

Case Study: The effects of excess fructose in a patient with Autism Spectrum Disorder

Case Study: Sore Eyes, Sick Bones, and a Penchant for Potato Chips

Case Study: Dientamoeba Infection & Post-Infectious Dysbiosis
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From the Editor

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Dr Oscar Serrallach, MBChB, FRACGP

We’re pleased to bring you our final Journal issue for 2014.

In this issue we have Part 1 of Nicole Bijlsma’s excellent article addressing the health hazards in the built environment and their potential effect on childhood neuro-behavioural disorders. Look out for Part 2 in the March 2015 issue of the ACNEM Journal.

There’s also an interesting article by Nirupma Deshpande addressing mild iodine deficiency and the need for supplementation during pregnancy.

In this issue we also bring you three great Case Studies from some recent ACNEM Fellowship graduates; Dr Frank Golik, Dr Natalie Ryan and Dr Mark Robertson.

I’d like to take this opportunity thank all the contributors and on behalf of the ACNEM team I wish all our members a safe and happy holiday season.

We look forward to seeing you in 2015.
In the past 20 years, the incidence of childhood neurobehavioural disorders such as autism and ADHD have increased worldwide. In the 1970s, autism was estimated to affect up to 5 in 10,000 children, now it is 1 in 68 US children. Whilst the incidence of autism in Australia is lower (1:100) according to Autism Spectrum Australia, disturbingly it has increased by 79% between 2009 and 2012. Better diagnosis and reporting cannot account for this rapid rise, nor can genetics, as none of the genes discovered so far seem to be responsible for more than a small proportion of cases. Emerging evidence suggests the environment is likely to play a crucial role.

The dramatic increase in autism spectrum conditions has occurred coincidentally with the deployment of wireless technologies and shows remarkable similarities to the pathophysiology following exposure to radiofrequency electromagnetic fields. In addition, a growing number of industrial chemicals have been identified as neurodevelopmental toxicants. Since 2006, the number of chemicals known to damage the human brain that are not regulated to protect children’s health increased to 214. Research conducted by Lintas et al (2012) identified immune genes and not neurodevelopmental genes as the most consistent abnormality typically found in neurodevelopmental disorders. An additional finding that further supports the environment as a contributing factor is the ‘good and bad days’ observed by parents and the transient reversal of symptoms in some children during fever and short term antibiotic treatments, all of which question the premise that autism is a disease due to a ‘broken brain’.

**Developmental Neurotoxicants**

Since WWII, thousands of chemicals have been introduced into building materials and consumer products. Since 1970, the global sale of chemicals has increased by a factor of 25 from $171 billion to $4.1 trillion US dollars and this is expected to accelerate. It is estimated that 84,000 chemicals are used commercially, 38,000 of which listed for use in Australia. Consequently the body burden of chemicals is increasing with each generation. There is strong evidence that industrial chemicals are contributing to a global pandemic of neurodevelopmental disorders which affect millions of children worldwide, the implications of which have devastating consequences on families and the global economy. Despite this, neurodevelopmental toxicity data are missing for most industrial chemicals in widespread use, even when population wide exposures are documented. Since 2006, eleven industrial chemicals have been identified as developmental neurotoxicants: lead, methylmercury, polychlorinated biphenyls, arsenic, toluene, manganese, fluoride, chlorpyrifos, DDT, tetrachloroethylene and polybrominated diphenyl ethers with many more likely to be discovered. Some like lead and mercury have extensive documented histories of adverse health effects in children dating back to Roman times; others like pesticides, flame retardants and industrial solvents and lubricants have gained notoriety because they are ubiquitous throughout the environment, they bioaccumulate up the food chain and are biologically persistent having been found extensively in wildlife and in most of the general population. Three are listed as persistent organic pollutants in the Stockholm Convention and all of them are listed as potential endocrine disrupting chemicals on the TEDX List (Endocrine Disruption Exchange, 2014).

**Flame retardants**

Since the 1970s, flame retardants have been used in consumer products to reduce the likelihood that an item will ignite, inhibit the spread of a fire, and to provide occupants more time to escape from a fire. They have been incorporated into paints, children’s clothing (low fire danger pyjamas), foams used in upholstered furniture, carpet padding, pillows and mattresses, as well as in plastic housings for televisions, computers, telephone handsets, power point fronts, light switches and kitchen appliances. Consequently the levels of flame retardants are greater indoors than outdoors and higher in buildings that have recently been renovated, carpeted or are air conditioned. Brominated flame retardants (also referred to as polybrominated diphenyl ethers or PBDEs) are the most...
common flame retardants found in households, eighty of which are used commercially in Australia\textsuperscript{21}. PBDEs are a group of more than 200 distinct chemicals that are structurally similar to polychlorinated biphenyls\textsuperscript{22}. The three types used commercially are pentaBDE, octaBDE and decaBDE each of which contains a mixture of congeners of PBDEs. Globally, decaBDEs are the most widely used brominated flame retardants as they are incorporated into wiring insulation, television and computer casings\textsuperscript{23}. PBDEs have gained notoriety amongst the scientific community because their bioaccumulative and persistent nature, in addition to their endocrine disrupting effects, has adverse health outcomes in animals and humans\textsuperscript{24}. Consequently tetraBDE and pentaBDEs are listed as persistent organic pollutants in the Stockholm Convention\textsuperscript{25}. Prenatal and childhood exposures to PBDEs in both human and animal studies resulted in increased hyperactivity, lower IQ and significant deficits in learning and memory\textsuperscript{26-29}. The mechanism of action is likely to be via its thyroid disrupting effects\textsuperscript{30} although a smaller number of studies have also examined disruption of the oestrogen/androgen hormone system\textsuperscript{31}. That’s a concern in light of the fact that the levels of PBDEs measured in the breast milk and blood of Australians is twice as high as those found in Europeans\textsuperscript{32-33} although it is lower than that found in Californian children who have the highest levels as a result of their fire safety law\textsuperscript{34}. Since PBDEs are semivolatile, they are not chemically bound to the substrate material which is why they are commonly found in household dust which accounts for 80% of the total exposure to PBDEs in infants as compared to only 14% in adults\textsuperscript{35}. As a result of this, blood concentrations of PBDEs in children is considerably higher than those found in adults\textsuperscript{36}. Inhalation and ingestion of household dust is likely to be the most common route of exposure which makes young children particularly vulnerable\textsuperscript{37}. The European Union has consequently mandated the phase out of certain PBDEs. Whilst NICNAS has banned the import and manufacture of two brominated flame retardants - pentaBDE and octaPBDE, it has not restricted their use in imported products (where children are most likely to be exposed), relying instead on a decline of these chemicals from voluntary activity by industry and to the lack of commercial availability as a result of international regulatory action\textsuperscript{38}. At the same time, they insist that “there is no evidence of any adverse health effects in newborns, children or adults from exposure to PBDEs\textsuperscript{39}. In light of recent evidence associating prenatal and early childhood exposure to PBDEs with a decline in IQ and increased hyperactivity\textsuperscript{29,31}, assumptions of safety can no longer be taken for granted.

**Lead**

Lead has a long history of adverse health effects dating back to Roman times. Unlike other developmental neurotoxicants, it is the most common and best understood childhood disease of toxic environmental origin that accounts for 0.6% of the global burden of disease\textsuperscript{40}. The deleterious effects of blood lead levels above10 ug/dL on brain function are well documented and include lowered intelligence and behavioural problems\textsuperscript{40-42}. At lower levels of exposure that previously were considered safe, lead is now known to produce a spectrum of injury across multiple body systems\textsuperscript{43}. A growing number of studies have shown that levels lower than 10 ug/dL are associated with adverse health effects such as inattention\textsuperscript{44}, cognitive loss\textsuperscript{45-46}, Attention Deficit Hyperactivity Disorder\textsuperscript{47}, reduced IQ and increased antisocial behaviour\textsuperscript{48} and delays in sexual maturation in adolescent girls and boys\textsuperscript{49}. The mechanism of action involves demyelination of neurons, death of brain cells, and disruptive effects on the dopaminergic system\textsuperscript{49}. Many are questioning as to whether there are any safe blood lead levels\textsuperscript{50}. Consequently Germany has lowered its action level to 3.5 ug/dL, the US Centers for Disease Control to 5 ug/dL, and the NHMRC is currently reviewing Australia’s action level currently set at 10 ug/dL. Australian children are especially at risk as it is estimated that 3.5 million homes contain paint with 50% lead content which is just one of several sources of lead in the environment\textsuperscript{51}. An Australian national survey of lead in 1,575 children found an average of 5 ug/dl in 1 to 4 year olds (7% exceeded 10 ug/dl) although this was conducted before leaded petrol was phased out in 2002\textsuperscript{52-53}. Nonetheless, inhalation and ingestion of house dust containing leaded paint remains a common source of exposure in young children.

**Pesticides**

The adverse health effects arising from pesticide exposure gained worldwide attention when Rachel Carson documented the abnormal mating behaviour in bald eagles and the collapse of the eagle population that were exposed to high levels of DDT in her book Silent Spring\textsuperscript{54}. Organochlorine pesticides (OC) such as DDT, dieldrin, aldrin, heptachlor and chlordane were used extensively in Australia during the 1950s to mid-1970s, but were subsequently phased out by 1990 following serious adverse health effects in animal and human studies and because they persist in the environment. They were replaced with the organophosphate pesticides (OP) which are widely used in agriculture primarily because their half-life is significantly shorter. Approx. 5,000 tonnes of OP are used annually in Australia\textsuperscript{55}. Whilst data associated with acute and/or accidental poisonings of pesticides in children is readily available, little data is available on subclinical pesticide exposure despite the fact that it is so widespread\textsuperscript{56}. In 1998, Australian doctors at Townsville Hospital tested the meconium of 46 newborn babies and found a wide
range of hazardous chemicals including POPs and pesticides such as chlorpyrifos\textsuperscript{64}. Data which describes the full, or even partial, extent of human health effects from exposure to pesticides is difficult to source due to potential long latency periods for chronic illness, the difficulty in diagnosis, the non-specific nature of pesticide health effects and the lack of effective monitoring systems\textsuperscript{59,63,65}. Three prospective epidemiological birth cohort studies provide new evidence that prenatal exposure to OP pesticides can cause developmental neurotoxicity\textsuperscript{56-58}. More recently, the Childhood Autism Risks from Genetics and Environment (CHARGE) study identified that children with ASD were 60\% more likely to have organophosphates applied near their home, whilst children with developmental delays were 150\% more likely to have carbamate pesticides applied near the home during pregnancy\textsuperscript{59}. The mechanism of action is likely to be due to the fact that these pesticides are inhibitory neurotransmitters which are necessary in the development and maintenance of neuronal transmission. Prenatal exposure to OP pesticides in animal studies have demonstrated more severe neurodevelopmental effects for males than for females, suggesting endocrine disruption maybe involved\textsuperscript{60}. Other classes of pesticides including the carbamates and the synthetic pyrethroids have also been linked to neurodevelopmental deficits in children\textsuperscript{61-62}.

Children are more vulnerable to pesticides because they receive a larger dose per unit of body weight for a given exposure due to their smaller body size\textsuperscript{63}, their unique diet (pureed fruit, veges...), their breathing zone is closer to the floor, and the enzyme involved in detoxification - paraoxonase-1 (PON-1) is less active making them more vulnerable to OP toxicity\textsuperscript{64}. Children are exposed to pesticides through inhalation (household dust, spraying), ingestion (food, drinking water and accidental poisoning) and dermal absorption (lice and scabies treatment, insect repellants, lawn, household dust...). Levels of pesticides in carpet dust can be useful indicators of exposure in epidemiologic studies, particularly for young children who are in frequent contact with carpets\textsuperscript{65}. Pesticides may persist for long periods of time inside the home, where they are protected from degradation by sunlight, rain, temperature extremes, and microbial action\textsuperscript{66}. Carpets are repositories for pesticides\textsuperscript{67-68} as the fibres and underlying foam pad appear to act as long-term reservoirs that continuously transfer pesticides to carpet dust.

**Endocrine disrupting chemicals (EDCs)**

All of the neurodevelopmental toxicants highlighted by Grandjean and Landrigan (2014)\textsuperscript{59} are listed as potential endocrine disrupting chemicals (EDCs) on the TEDX List (Endocrine Disruption Exchange, 2014). EDCs pose an additional concern for the unborn foetus and children because unlike other chemicals, their impact on cognition and behaviour is likely to arise at low levels of exposure during critical windows of development\textsuperscript{1}. EDCs may well provide a vital clue as to why males are up to five times more likely to develop autism than females as highlighted by a recent spatial incidence study involving one third of the entire US population using insurance claims datasets. The authors concluded that the strongest predictors for autism were associated with the environment, as autism incidence was strongly linked to congenital malformations of the reproductive system in males which are not typically associated with genetic causes (an increase in autism incidence by 283\% for every per cent of increase in the incidence of malformations)\textsuperscript{69,70}. The incidence of genital malformations such as cryptorchidism and hypospadias has increased in recent times. The prevalence of hypospadias rose 2\% every year between 1980 and 2000 in Western Australia\textsuperscript{71} and similar trends were seen in South Australia\textsuperscript{72} but not in Victoria or NSW. These malformations typically arise during early embryonic development – specifically between weeks 9 to 12 which corresponds to the time when cell division and migration takes place in brain development\textsuperscript{72}. Coincidentally it is also the time when maternal exposure to xeno-oestrogens in animal models affects both the brain and genital development in male progeny\textsuperscript{73}. Xeno-oestrogens are found in a number of environmental toxins including (but not limited to) OP pesticides, polybrominated flame retardants and polychlorinated biphenyls. A study published in 2007, identified that babies born with cryptorchidism or hypospadias had a more than 2.5 fold increased risk of having detectable levels of DDT and its metabolite DDE, lindane and several other organochlorine pesticides in their blood\textsuperscript{74}. Some researchers have explained the gender bias seen in autism as a result of the fact that the female brain requires more extreme genetic alterations than does the male brain to produce symptoms of neurodevelopmental disorders (not related to the X chromosome)\textsuperscript{75}. Gender bias is present in several neurodevelopmental disorders including autism, intellectual disability, and attention deficit hyperactivity disorder.

References


Mild Iodine Deficiency and the Need for Iodine Supplementation During Pregnancy

Nirupama Deshpande, PhD

Abstract

Iodine deficiency is one of the most common nutrient deficiencies in the world with almost two billion people affected. This has profound effects on intellectual development with the most extreme being cretinism. In fact, iodine deficiency is considered the single most cause of preventable brain damage and mental retardation. Iodine requirement increase substantially during pregnancy and lactation making pregnant and lactating women and their babies high risk groups for iodine deficiency. There is now irrefutable evidence that iodine deficiency is widespread in Australia and New Zealand. As a result in 2009, the Australian Health ministry made it mandatory to use iodized salt in the bread-making process. Despite mandatory iodine fortification, poor knowledge remains amongst pregnant and lactating women in Australia about the role and sources of iodine in the diet. Therefore a health education campaign is much needed to encourage all pregnant women and breastfeeding mothers to take iodine supplements. Summarized here is a review on current literature about the role of iodine in pregnancy and the potential benefits of maternal supplementation with iodine during pregnancy.

Introduction

Iodine is a crucial micronutrient that humans need to produce thyroid hormones throughout life. In fact, the only known physiological function for iodine is in the synthesis of thyroid hormones. These hormones are especially needed to ensure normal development of the brain and nervous system during gestation and early life. And yet iodine deficiency affects 2 billion people worldwide and is the main global cause of preventable neurodevelopmental disorders. Inadequate iodine intake during pregnancy is of particular concern as depending on the severity of deficiency, it can result in miscarriages, stillbirths, cretinism, irreversible mental retardation, impaired psychomotor development and behavioral problems.

Every year around 40 million babies are born into this world with brain damage due to iodine deficiency. The fetus is totally dependent in early pregnancy on maternal thyroid hormones for normal brain development. During lactation, the mammary glands concentrate iodine within breast milk to nourish the newborn whose iodine requirement is approximately 7mg/kg of body weight. Thus it is very crucial that pregnant and lactating women consume enough iodine.

Physiology of Maternal Thyroid in Pregnancy

Pregnancy has a profound impact on the function and structure of the thyroid gland. Moderate thyroid enlargement and increased vascularity occurs as a result of pregnancy hormone-induced glandular hyperplasia. During pregnancy, thyroid stimulation starts in the first trimester due to increased iodine uptake and production of the human chorionic gonadotropin hormone (beta-HCG), which shares some structural homology with thyroid-stimulating hormone (TSH) and has a mild thyrotropic activity. Beta-HCG is at its greatest concentration during the first trimester, while serum TSH drops (Figure 1). Thus the scale for the normal range of TSH during pregnancy is compressed, compared to euthyroid (normal functioning thyroid) adults, with a lower limit of “normal” at both ends of the spectrum. The lower normal TSH in the first trimester is still poorly defined but recently, a TSH of 2.5mIU/L has been accepted as the upper limit of normal for TSH. There is also an estrogen-mediated 2-3 fold increase in circulating levels of thyroid-binding globulin (TBG). Following the rise is TBG levels, there is a sharp rise in the concentrations of total thyroxine (T4) and total triiodothyronine (T3). However changes in free T4 and T3 concentrations during pregnancy are controversial. Some authors have reported a decrease in free hormones, whereas others have reported no change or even an increase. Pregnant women in general have lower free-hormone concentrations at term than nonpregnant women. Furthermore, there is a relative decline in the availability of iodine, secondary to the increased renal clearance and overall losses to the fetus and placenta. Therefore to meet the demand of pregnancy for the thyroid hormone in the first trimester, a pregnant woman needs to increase iodine intake by at least 75 mg per day.
The physiology of fetal thyroid

The thyroid hormones are most important to fetal brain development during the first trimester of pregnancy. Still, significant fetal brain development continues considerably beyond the first trimester, making thyroid hormones important also later in gestation. The fetal thyroid gland begins concentrating iodine and synthesizing thyroid hormones after 12 weeks of gestation. Any requirement for the thyroid hormones before this time is solely supplied by the mother. Overt maternal thyroid failure during the first half of pregnancy has been associated with several pregnancy complications and intellectual impairment in the offspring. It is currently less clear whether milder forms of thyroid dysfunction have similar effects on pregnancy and infant outcomes and is discussed below.

Mild to moderate iodine deficiency during pregnancy

The adverse effects of iodine deficiency are collectively termed iodine deficiency disorders (IDD). The consequences of iodine deficiency depend on several factors such as the degree of deficiency, the age and duration of exposure with fetal development and early infancy being particular critical times.

Until recently, iodine deficiency was considered to be not much more than being responsible for endemic goiter. But now it is well established that severe iodine deficiency has major consequences on neurological and physical development. However, the adverse consequences of mild-to-moderate iodine deficiency are less certain. This is mainly because the degree of iodine deficiency that results in significant health effects are not easy to define. The effects of iodine deficiency can be subtle at an individual level and are often sub-clinical. In addition, studies are challenged by ethical constraints and the difficulty of controlling for confounding variables. There are a limited number of published studies that investigate the health consequences of mild iodine deficiency. Preliminary evidence by Bath et al and Hynes et al suggest that even mild iodine deficiency during pregnancy can have long term adverse impacts on fetal neurocognition that are not ameliorated by iodine sufficiency during childhood.

Hypothyroidism during pregnancy

The major causes of maternal hypothyroidism and fetal hypothyroxinaemia are iodine deficiency and autoimmune thyroid disease. Risk of severe hypothyroidism to pregnancy has been well documented, but only recently, mild or subclinical hypothyroidism has been reported to increase the risk of impaired neurodevelopment in the offspring. Subclinical hyperthyroidism is defined as raised TSH combined with a normal serum free thyroxine level.

Figure 1 Comparison of the changes of Beta-HCG vs. TSH during gestation, taken from Glinoer et al, 2007.
Table 1: Epidemiological criteria for assessing iodine nutrition in a population based median urinary iodine concentration in school-aged children

<table>
<thead>
<tr>
<th>Median UIC (ug/L)</th>
<th>Iodine Intake</th>
<th>Iodine Status</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;20</td>
<td>Insufficient</td>
<td>Severe iodine deficiency</td>
</tr>
<tr>
<td>20-49</td>
<td>Insufficient</td>
<td>Moderate iodine deficiency</td>
</tr>
<tr>
<td>50-99</td>
<td>Insufficient</td>
<td>Mild iodine deficiency</td>
</tr>
<tr>
<td>100-199</td>
<td>Adequate</td>
<td>Optimal</td>
</tr>
<tr>
<td>200-299</td>
<td>More than adequate</td>
<td>Risk of iodine induced hyperthyroidism</td>
</tr>
<tr>
<td>&gt; 300</td>
<td>Excessive</td>
<td>Risk of adverse health consequences</td>
</tr>
</tbody>
</table>

The normal range of TSH varies according to geographic region and ethnic background. In the absence of local normative data, the recommended upper limit of TSH in the first trimester of pregnancy is 2.5 mIU/L, and 3.0 mIU/L in the second and third trimester. The prevalence of undiagnosed subclinical hypothyroidism in pregnant women ranges from 3% to 15%. Subclinical maternal hypothyroidism may be associated with poor pregnancy outcomes such as placental abruption, gestational diabetes, gestational hypertension, preterm delivery, low birth weight infants and decreased IQ in the offsprings.

Symptoms of hypothyroidism can often be masked by the hypermetabolic state of pregnancy. During the early weeks of pregnancy, a fall in serum TSH and increase in free thyroxine is observed that may confuse the diagnosis of hypothyroidism. The reference range for serum TSH in nonpregnant women is 0.45-4.5mU/L, with more than 95% of individuals having a value below 2.5 mU/L. Moreover, during a normal pregnancy the thyroid gland increases in volume to about 10% in iodine deficient women, and up to 30% in iodine deficient women. The thyroid gland produces 50% more thyroxine that requires 50% more iodine. Thus it is imperative that the iodine intake must be increased by 50% daily to ensure adequate maternal thyroid hormone production. This will ensure optimal iodine nutrition for mother and fetus and prevent maternal and/or fetal hypothyroidism. There is also a need to increase and optimize thyroxine replacement therapy in thyroxine treated hypothyroid pregnant women before or soon as possible after conception.

Iodine deficiency in Australia

Australia was officially classified as mildly iodine deficient by the World Health Organisation (WHO) in 2007. In the 1990s, mild to moderate iodine deficiency re-emerged in Australia

<table>
<thead>
<tr>
<th>Publications</th>
<th>Subjects</th>
<th>Site</th>
<th>N=</th>
<th>% &lt;50mg/L</th>
<th>Median UIE mg/L</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gunton et al 1999</td>
<td>Pregnant Post-partum</td>
<td>RNSH Sydney</td>
<td>81</td>
<td>19.8</td>
<td>104</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>28</td>
<td>19.2</td>
<td>79</td>
</tr>
<tr>
<td>Li &amp; Eastman 1999</td>
<td>Pregnant women</td>
<td>Western Sydney</td>
<td>101</td>
<td>20.6</td>
<td>88</td>
</tr>
<tr>
<td>Hamrosi et al 2005</td>
<td>Caucasian Vietnam</td>
<td>Victoria</td>
<td>227</td>
<td>48.4</td>
<td>52</td>
</tr>
<tr>
<td></td>
<td>India Srilanka</td>
<td></td>
<td>263</td>
<td>38.4</td>
<td>58</td>
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<td></td>
<td></td>
<td></td>
<td>262</td>
<td>40.8</td>
<td>61</td>
</tr>
<tr>
<td>Travers &amp; Eastman 2006</td>
<td>Pregnant &gt;28 weeks</td>
<td>Central Coast NSW</td>
<td>815</td>
<td>17.0</td>
<td>85</td>
</tr>
</tbody>
</table>

Table 2: Iodine status of pregnant women in Australia (courtesy Prof. C Eastman)
This was primarily due to a decline in iodine content in milk and dairy products coupled with the use of non-iodized salt in households. Several measures were introduced by the Australian health ministry to address the issue of iodine deficiency. In 2009, mandatory iodine fortification of salt in the bread-making process was introduced. But it was recognized that mandatory fortification of bread did not meet the increased needs of pregnant and lactating women. Therefore, in 2010 the National Health and Medical Research Council released a public statement that recommended iodine supplementation (150mg/day) for pregnant and lactating women. Despite these efforts, iodine deficiency continues to be prevalent in many pregnant women in Australia. A study done on pregnant and lactating women in the regional area of New South Wales, Australia revealed poor knowledge about the detrimental outcomes of iodine deficiency during pregnancy and sources of iodine in the diet. Few women were aware that bread is required by law to be fortified with iodine. Many women incorrectly identified neural tube defects and weak bones and teeth to be related to iodine deficiency.

The simultaneous implementation of both folic acid and iodine fortification of bread in Australia has contributed to this consumer misunderstanding. The study revealed that 40% of pregnant women and 55% of lactating women were not receiving iodine supplementation. However, an important improvement has been that the use of supplements containing iodine had increased from 20% to 60% in pregnant women after mandatory fortification program was introduced. Unfortunately, following the introduction of mandatory iodine fortification, there has been no public or professional education initiatives directed towards increasing awareness about the importance of iodine during pregnancy and lactation. A public health policy is urgently needed in the face of clear documentation of iodine deficiency and strong evidence of the deleterious effect on neurodevelopment of children. Until measures are taken to ensure that iodine needs can be met by usual dietary sources, it is recommended that pregnant and breastfeeding women should insist that the prenatal vitamins they are prescribed contain iodine. The only exceptions are women who have pre-existing thyroid disease e.g Graves’ disease or Hashimoto’s disease.

<table>
<thead>
<tr>
<th>Population group</th>
<th>Recommended Nutrient Intake for Iodine (mg/d)</th>
<th>Excessive Iodine Intake (mg/d)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Adult men and women</td>
<td>150</td>
<td>&gt; 500</td>
</tr>
<tr>
<td>Pregnant women</td>
<td>250</td>
<td>&gt; 500</td>
</tr>
<tr>
<td>Lactating women</td>
<td>250</td>
<td>&gt; 500</td>
</tr>
<tr>
<td>Child &lt; 2 years</td>
<td>90</td>
<td>&gt; 180</td>
</tr>
</tbody>
</table>

*Table 3: Recommended daily Iodine intake WHO 2005*  

Iodine Supplementation

Urinary iodine excretion is an accurate indicator of dietary iodine intake as less than 90% of ingested iodine is excreted in the urine. Using data from published median urinary iodine excretion levels in pregnant Australia women (80 mg/l) and assuming an average daily urine output of 1.5 litres, the estimated daily iodine intake is approximately 130 mg. This is about half the recommended daily requirement. Thus iodine is an essential nutrient that can be primarily derived from the diet and from vitamin/mineral preparations. The WHO recommends 250 mg/d for pregnant and lactating women. Tolerable upper intake levels for iodine have been established to determine the highest level of daily nutrient intake that is likely to be tolerated biologically and to pose no risk of adverse health effects for almost all individuals in the general population. The upper intake levels are based on total intake of a nutrient from food, water, and supplements and apply to chronic daily use. The tolerable upper limit for daily iodine intake is 1100mg/d in all adults, including pregnant women. The WHO has stated that daily iodine intake >500mg may be excessive in pregnancy.

Medications and some supplements can be a source of excessive iodine intake for some individuals such as certain types of anti-asthmatic medications, expectorants, intravenous radiographic contrast agents and topical antispetics.

Conclusion

Although substantial progress has been made over the last five years, iodine deficiency remains a significant health problem. The ongoing monitoring of population iodine status remains crucially important and particular attention may need to be paid to monitoring the status of vulnerable populations, such as pregnant women and infants. There is also need for ongoing monitoring of iodized salt and other dietary iodine sources in order to prevent excess as well as insufficient iodine nutrition. Finally it will be essential to coordinate interventions designed to reduce population sodium intake with salt iodization programs in order to maintain adequate levels of iodine as salt intake declines.
References:


Case Study: The effects of excess fructose in a patient with Autism Spectrum Disorder infections

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Abstract

This case study will outline an overview of the metabolic effects of fructose in a child with ASD.

Numerous dietary approaches have been tried as therapeutic management tools in children with ASD. This paper aims to highlight some of the sequelae resulting from the excess use of fructose in the diet. This case concerns a nine year old male who significantly improved in focus, behaviour, engagement, confidence, and his expressive language as a result of dietary management and nutrient supplementation.

Introduction

There have been a number of dietary approaches in the management of patients with Autism and Autism Spectrum Disorders (ASD). Despite controversy in mainstream Paediatrics and amongst Dieticians, quite a reasonable percentage of parents and a number of doctors and Paediatricians both here and internationally have explored the therapeutic tool of dietary intervention in the management of ASD. The fundamental shift in focus in the last fifteen years or so is to realise the importance of: the gut associated lymphoid tissue (GALT), the entire digestive processes, the G-I microbiome, the correct absorption of nutrients, increased intestinal permeability (metabolic endotoxaemia)\textsuperscript{11-12}, the influence of foods upon the immune system\textsuperscript{11-12}, brain, emotions and behaviour, and epigenetic expression\textsuperscript{13}. This evolving concept illustrates the so called Gut-Brain connection: particularly exemplified in ASD\textsuperscript{11-12,22}.

The most common starting point that is utilised in ASD dietary management is to be Gluten and Casein-free (GFCF)\textsuperscript{14-15}. A parent questionnaire performed by Autism Research Institute in California showed a 69\% rating of benefit with the elimination of these sustances\textsuperscript{16-17}. The next commonly avoided ingredients are: added sugars, colouring dyes, preservatives, salicylates, amines, glutamates, soy and maize. A number of ASD children have elements of ADHD as a co-morbidity. The use of a Failsafe diet\textsuperscript{18-20} in conjunction with the GFCF diet can have a calming effect. It appears that maize can exert a gluten-like effect; and soy products may exert a dairy-like effect.

The next dietary concept was the use of the Specific Carbohydrate diet (SCD): which was initially introduced for the management of Inflammatory Bowel Disease (IBD) by Elaine Gottshall\textsuperscript{21}. A percentage of children with ASD will benefit from this diet. It uses some fruit. An offshoot from the SCD is the Gut and Psychology Syndrome (GAPS) diet, which was developed by Dr Natasha Campbell-McBride\textsuperscript{22-23}. Later foods allowed in the GAPS diet include some fruits, carrots and fermented dairy. Another variant along these lines is the Paleolithic (‘Paleo’) diet\textsuperscript{24}. This presents in different formats; but it is basically sugar and grain-free. It also contains sweet fruits and reasonable amounts of coloured vegetables.

The FODMAP diet\textsuperscript{25} looks at the adverse effects of fermentable sugars (including fructose) upon the gastrointestinal tract.

The last two dietary considerations are the avoidance of the Solanaceae (‘Nightshade’)\textsuperscript{26} family of foods in a small percentage of these children and the Low Oxalate diet (LOD)\textsuperscript{27} in those patients who form considerable oxalate crystals. Time does not permit an extensive explanation for using any of the aforementioned diets. It is vitally important that growing children are eating macro and micro-nutrient-dense, ideally organic and pasture-fed, non-processed quality foods with essential protein content, plenty of (mostly green) vegetables, low fructose fruit and good oils (Coconut, EVP Olive, Monosaturates), adequate dietary fibre, and activated nuts and seeds (oxalate foods). Fermented vegetables, Kefir-fermented coconut water and milk, and bone broths (high oxalate) can all be frequently helpful in children with ASD.

I propose to look at the potential harm from consuming excess fructose in our diet especially in patients with an ASD diagnosis.
INITIAL PRESENTATION

A 7 year old boy with diagnoses (at 4 years of age) of Autism Spectrum Disorder (ASD), ADHD, and anxiety presented with his mother in July 2013. He is the eldest of three children. His two sisters have been diagnosed as being on the Autism Spectrum.

His father has Asperger’s syndrome; and his mother has Sjögren’s syndrome, Rheumatoid Arthritis and IBS. His mother fell pregnant very easily with him; her first pregnancy. His mother suffered from numerous migraines in his pregnancy for which she took a “lot of paracetamol” as well as propranolol. She had four amalgam fillings present in this pregnancy and her grandmother was terminally ill at the same time. At 39+ weeks, she went into spontaneous labour; she had Pethidine, nitrous oxide and an epidural. There was foetal distress before vaginal delivery: reasonable APGARS. The child was given vitamin K and hepatitis B vaccination at birth. He had low muscle tone, poor suckling/swallowing: which resulted in mastitis and multiple breast abscesses requiring drainage and Flucloxacinil for 8 weeks. His mother breast-fed for 10 weeks before introducing Karicare for 12 months. Solids were given to him at 5 months. His developmental milestones were advanced generally. There was no distinct regression but his mother described him as being a very difficult baby and toddler due to high anxiety mixed with obvious high intelligence. She also described him as being a friendly, personable and polite child.

At presentation he was eating a moderate amount of grain-based refined carbohydrates, fruits and carrots, lactose-free milk, meats and some lollies and chocolate. He craved meats and ice cream. His mother suspected that he reacted to FODMAP’s, Salicylate and Amine-containing foods. He was fully vaccinated. He experienced vomiting, diarrhoea and sleep disturbance after his 2 months vaccinations. He cried after his 18 month vaccinations. He was quite restless, verbalising or having ‘melt-downs’ if worried over something. He was quite hyperactive, unable to tolerate oral medications. His mother described him as a very difficult baby and toddler due to high anxiety mixed with obvious high intelligence. She also described him as being a friendly, personable and polite child.

His attitudes demonstrated marked rigidity and inflexibility. He was agitated by waiting and his major concern was his word-finding difficulty. When I asked him for some treatment goals, he stated that he had none and did not want to change.

After seeing Paediatricians, Speech therapist and Occupational therapist, he has been trialled on Amitriptyline and Fluoxetine: both giving him uricaria. Risperidone gave him nasty reactions and tremors and Escitalopram did nothing for him either way. Atomoxetine 25mg daily was helping partially with his anxiety initially as well as helping his focus and diminution of his stimming. His mother had found good responses in Speech and Occupational therapies, using the Gluten-free, low Salicylate and low FODMAP diets. They helped to bring some reduction in anxiety and hyperactivity.

His other presenting symptoms included stimming (severe and constant), head-banging, lack of any imaginative or pretend play, nail-biting, mood swings, impulsivity, poor coordination, central processing problems, sensitivity to crowds, poor memory, low body weight, poor focus and attention, cold intolerance, Tics (severe), sensitivity to bright light and certain sounds, bloating and foul flatus, fussiness with foods and textures, some OCD behaviours, toe- walking, and great difficulty tolerating oral medications. His father is an IT consultant who runs all the computers at home on Wi-Fi. Clinically, he was a pale, extremely thin, withdrawn and an almost lifeless boy who was mouth-breathing and disconnected. His height was on the 50th centile; but his weight (21kg) was on the 5th centile. Physical examination was otherwise unremarkable. There was no keratosis pilaris or white spots on his fingernails; and his second toe was shorter than his hallux. His pathology testing showed: Cholesterol 3.8 (L), HDL L.57, LDL 2.0; normal E/LFT’s; normal FBC except for elevated eosinophilia (1.25); IgE 1511 (H) with RAST positive to Dustmite (V) and Grass pollen (III); normal TFT’s and no TPO Abs: mild iron deficiency (ferritin 28); B12 311 (L), RBC Folate 839; TTG and Anti-Gliadin Abs neg; hs CRP <0.3; ANA, ENA, dsDNA and Cardiolipin Abs : all neg; HCY 3.9; Cortisol 173 (L), DHEAS 0.5; 25 OH-Vitamin D3: 32 (v.Low); Immunoglobulins (except IgE) in normal range; Helicobacter serology neg; ASOT 316 (H) and AntiDNase B 649 (H); WB Histamine 1.2; Plasma Zinc 17.7; Plasma Copper 15.3; Free Copper 4%; Urine Iodine 191; Feces: no ova, cysts or parasites seen; Faecal Calprotectin 25 (N); His urine showed Oxalates; Apolipoprotein E4/3 genotype. HPL (Hydroxyhaemopyrrolin-2-one): 16.5, corrected for SG: 11.4; MALDI-TOF MS of his faeces revealed E. coli were 96.5% of aerobes and Enterococcus faecium 3.5%; Bacteroides spp. were 88.6% of anaerobes and Clostridium spp. were 8.5%. DNA screening revealed a Heterozygous SNP on MTHFR (C667T), Homozygous SNPs on MTRR, and Heterozygous SNP on CBS.

MANAGEMENT

His mother agreed to gradually move his diet to a GAPS-style direction (minus sweet fruit and dairy), low FODMAP, low OXALATE and low SALICYLATE. Mum was going to make bone broths. He was fairly accepting of this shift. He was prescribed oil-distilled Cod Liver Oil: increasing to 8mls noce. In a few weeks he started on a combined supplement of Zinc picolinate 25mg/Pyrindoxyl 5phos 25mg/ Magnesium glycinate 50mg : 1 , increasing to, 3 mane. He also commenced Epsom salt (1/2 a cup) bath soaks: 3 nights per week for 20minutes.

In 4-5 weeks, he was much happier, making jokes, having improved eye contact, and showing more personality and affection. His appetite had significantly improved and his stools were formed and less offensive. In August I prescribed a fully compounded morning and evening supplement list.

By October 2013, he had gained 2 kg in weight. He was more robust, calmer and acting out pranks. Wheat exposure created teary and aggressive melt-downs. I started a very slow reduction in his dosage of Atomoxetine, without any problems. Acreryl L-carnitine 500mg bd and Ubiquinol 100mg noce were added in order to assist his mitochondrial function and his low muscle tone.

In January 2014, he was continuing to make steady progress. He has two friends: one being neurotypical. He told me that he was proud of being a ‘geek’. He loves dragons, dinosaurs and Harry Potter. He is now 24.5kg, having good formed stools and he is sleeping well.

I received an email from his mother in February 2014 saying how thrilled she was to have her son interested in Superheros and watching a Batman movie without
interruption as well as having full interactions and being calm, happy, engaged and completely present.

In May 2014, I added 5-methyltetrahydrofolate 400mcg mane and S/C injections of Methylcobalamin (64.5mcg/kg) every third night initially for 6 weeks, then nightly. Again his mother sent an email describing her “new” son who was now playful, chatty, warmly affectionate, energetic, more self-aware, and a “total joy to watch.”

At a consultation in June 2014, he was happier, making new friends at school but still sensitive to bullying and his unsupportive support teacher. His stools were Type 4 dark brown; and he was sleeping well. He was compliant with the food choices given to him.

He can become “silly” with sweet fruit such as apples (fructose 8.4g/100g) and banana (9g/100g). These fruits are no longer eaten. The silliness is often characteristic of a yeast overgrowth.

**DISCUSSION**

This case illustrates numerous issues in treating these children. My focus in this paper is to highlight the significant impact of various food choices upon the ASD and ADHD conditions. Simply put, foods can have beneficial or detrimental effects. The actions of fructose in these conditions has had limited exposure in the medical literature. The dangers associated with High Fructose Corn Syrup (HFCS) have been noted, especially with its mercury content. Consumption of HFCS and inorganic mercury may both modulate paraoxonase-1 (PON1) gene expression. Consumption of HFCS may lead to mineral imbalances, including Zinc, Calcium and Phosphorus loss and Copper gain.

There are connections between fructose and the potential overgrowth of gastrointestinal Streptococci, Candida spp. and parasites. Therefore, fructose can increase the symptoms of PANDAS (Paediatric Autoimmune Neuropsychiatric Disorders Associated with Streptococcal infections). PANDAS syndrome can give symptoms of OCD, marked, and often irrational anxiety and phobia. It can also be associated with Tic disorder and Tourette’s syndrome. Fructose can help create more oxalate formation and excretion in the urine and faeces. Symptoms associated with oxalate crystallisation are dysuria, sandy stools, sleep disturbance, gritty eyes, arthralgia and myalgia. The use of calcium citrate in the afternoon can be very helpful in precipitating the crystals. Typically, the gut bacteria called Oxalobacter formigenes is meant to break down oxalates. It is commonly depressed by the use of antibiotics. Thus, I find that there is a close triangular link between fructose, PANDAS and oxalate issues.

Fructose can exacerbate insulin resistance, metabolic syndrome, inflammation, obesity, metabolic acidosis and raise urate levels as well as being a major contributor in the development of non-alcoholic fatty liver. See Figure 1.
The obvious concern that may be raised is the question of this child having adequate micronutrients in a somewhat restricted diet. Firstly, it is vitally important to provide substantial macronutrients of first quality pasture-fed, if not organic, protein and organic vegetables (mostly of the green variety) as carbohydrate and fibre, and a moderate amount of healthy oils (coconut, olive, avocado etc).

The most common micronutrients that are found to be low in these children are: Iodine, Iron, Magnesium, Calcium, Zinc, Methylcobalamin, Selenium, Chromium and Molybdenum. It is essential that these nutrients are adequately supplemented. This generally results in improvements. Dairy products are not essential for the delivery of calcium. I always ask the question: “where do cows get calcium from?”. Grains are not essential for fibre or B group vitamins. Vegetables, avocado, nuts and seeds will give fibre and healthy populations of G-I E. coli will manufacture B group vitamins. Otherwise one can supplement with the B group vitamins. I monitor vitamin and mineral levels throughout treatment. I also monitor blood lipid levels. These, in nearly all cases, improve on this dietary program. Muscle development and general health improves as well. The treated children generally look extremely healthy.

**CONCLUSIONS**

The consequences of excess fructose in the diet of children on the Autism Spectrum can have significant effects upon their behaviour, mental processing and general wellbeing.

**EXPLANATIONS OF ABBREVIATIONS IN FIGURE 1: DANGER THEORY OF INFLAMMATION**

**PRRs:** Pattern recognition receptors.

**NLRs:** Nucleotide-binding oligomerization domain leucine rich repeat-containing receptors. They are a family of cytosolic pattern recognition receptors that activate immune system inflammation (IL-1β, IL-18 and NF-kB) via inflammasomes.

**PAMPs:** Pathogen associated molecular patterns: from overwhelming infections (viral, bacterial, fungal).

**DAMPs:** Danger associated molecular patterns: from significant trauma or persistent tissue stressors (environmental, mental/emotional, endogenous).

Activation of certain NLRs under conditions of cellular stress or injury leads to assembly of unique intracellular protein complexes termed: INFLAMMASOMES (eg. NLRP3).

**NLRP3 inflammasome** instigates obesity-induced inflammation and insulin resistance: especially in the presence of extracellular acidosis.

**ALARMS:** include PAMPs and DAMPs.

**ECM:** Extracellular matrix
Abstract
A child presents with eye pathology. Underlying is a major eating disorder connected to a genetic abnormality. Serious bone pathology develops and a connection to vitamin A supplementation is investigated.

Introduction
At the National Institute of Integrative Medicine (NIIM) one-day symposium on 25 October 2014, mention was made of vitamin A’s ability to cause significant bone loss. Dr John Murtagh, on the same day, quoted Louis Pasteur when he said, “the greatest derangement of the mind is to believe in something because one wishes it were true”. This case involves therapy with vitamin A and the later development of osteoporosis. The evidence is assessed for vitamin A’s contribution.

Case Presentation
EJ—female, presented at age 9½ with severe bilateral eye inflammation.

Dietary history: 5 small bags of salted potato chips every day exclusively for over 4 years.

The child was ‘funny looking’ with a high arched palate and dental deformities, small stature and obvious intellectual disability. She was attending the special school 2 days a week and the primary school for a full day a week. She had limited speech but a good memory, fluttered her eyelids, laughed a lot and displayed personality. She could walk and run, displaying no impairment of gross motor function. She reportedly slept well once asleep and woke up alert.

Parents and 3 elder siblings were ‘normal’. There was a lifelong history of eating problems. EJ was induced for polyhydraminos at 36½ weeks gestation. Nasogastric tube feeding was commenced in her first week due to a ‘poor suck’ and was continued until 18 months of age with infant formula and additives and a weekly change of tube. She was then fed from a spoon: water and 10 tins of certain baby food daily; custards with some ingestion of chicken, noodles and vegetables. There was some vitamin supplementation from the dietician. From the age of 5, EJ refused all baby food except chocolate custard. She was admitted to Royal Children’s Hospital (RCH) mother and baby unit on recommendation from Infant Welfare. There were no eating breakthroughs except that she developed a liking for potato chips. She was discharged as a potato chip eater and potato chips became her staple diet for the next 4 years. There was another admission to the RCH for an intensive feeding program with input from a Speech Therapist. This resulted in no new dietary achievements except the occasional acceptance of malten milk and Anzac biscuits, rice bubbles, honey and honey joys. Her only supplement was B12 which had been low on investigation. Just prior to consulting me, she had been started on sodium valproate for generalised seizures.

EJ hated anything, except potato chips, in her mouth. She refused teeth cleaning.

EJ had seen an ophthalmologist for her severe eye inflammation. Her eyes remained red and dry despite topical lubricants and anti-inflammatories. Surgery was under consideration to retain eye lubrication.

EJ’s eyes responded dramatically to empirical vitamin A (A+E as a miscel with liquid zinc), returning to normal.

From that time until now, 12 years later, EJ has been my patient. Early on, I saw EJ weekly with a new miniscule food challenge. She delighted in visiting me to show off her toy horse collection. She had 2 further hospital admissions, including 6 weeks in the Austin Hospital Child Unit with her family, with ongoing mental health support.

Eventually, mainly due to family perseverance, EJ consumed little pieces of minced meat, egg white, a narrow range of soft vegetables, grated apple, cheese, fried rice, popcorn and vegemite. EJ’s staple diet still remained packet chips every day for the next 11 years.

She devoured 3 bags daily with enthusiasm and delight. EJ had one at breakfast, one after lunch and one at supper. She continued to only drink water by the dessertspoon.
Supplements were challenging to introduce. Acceptance was only to liquids in small amounts and did not seem to be related to taste.

Looking at her supplement scripts over the 12 years in my care, in addition to the liquid A + E and zinc, there has been vitamin D3 liquid drops, an essential fatty acid mix, cod liver oil, amino acid complete blend, trace elements solution, selenium drops, magnesiuum concentrated liquid, iron and folic acid liquid, dissolved calcium phosphate, liquid B vitamins, CsQ10, biogreen powder, sodium ascorbate, and a kids multivitamin mixed from powder.

Blood tests were and are acceptable if very seldom. Measurements have been done for Vitamin A, D3, selenium, magnesium, zinc, copper, iron studies, B12, B6, calcium, fatty acid profile, parathyroid, thyroid function, and growth hormone (IGF-1), cortisol and antibodies for coeliac. Vitamin A was a recurring deficiency if supplements were not kept up. There were documented deficiencies in iron, B12, D3, selenium, magnesium, zinc, calcium (with variable PTH), and growth hormone.

EJ remained small: height under 130cm, weight under 30 kg. The potato chips and supplements did not make her fat.

There was no progression to puberty.

Then 3 years ago, tragedy struck.

At the age of 18, EJ sustained spontaneous crush fractures of T10, 11, 12 and then T9 and L1.

The local paediatricians and specialist gastroenterologist and endocrinologists have since become actively involved. Full hormonal studies were carried out and bone, brain, thyroid and pelvic imaging. Diagnoses: failure to progress to puberty and osteoporosis, and in the last year only, hyperthyroid (T3).

Genetic testing, earlier declined, was finally arranged after the fractures. EJ was given a diagnosis of Cardiofaciocutaneous (CFC) syndrome.

A peg tube has only been inserted in the last 12 months. EJ currently receives night feeds of a nutritional formula (Nutrison Energy) running in while she sleeps at 65 mls/hour for 9 hours-prescribed and monitored by Dietetics at RCH for 80% of her nutritional requirements.

There was a terrible reaction to biphosphonates-non specific neuralgia and myalgia lasting for 3-4 months.

EJ has been commenced on oestradiol valerate 2mg nightly, with a recent addition of medroxyprogesterone, by the endocrinologist. The tonic clonic seizures have worsened. Migraines have started occurring regularly. She has been commenced on carbimazole for hyperthyroid.

Current supplements: Vitamin A and E, D3, magnesium, calcium phosphate, and complete amino acid blend, fish oil, evening primrose oil and sodium ascorbate.

Recent pathology results: Serum oestradiol is 163, renal function and calcium phosphate are normal, vitamin D3 is 82, T3 is high: 6.3. Vitamin A, after 11 years of supplementation and recurrently being in the sub reference range, is now therapeutic at 1.09 umol/l.

**Discussion**

Cardiofaciocutaneous (CFC) syndrome, usually from a BRAF gene mutation, is very rare, with an estimated 200-300 persons worldwide. The BRAF gene codes for certain signalling proteins influencing cell growth, differentiation, movement and apoptosis. Polyhydramnios is common during gestation. Cardiac defects are usual (not present for EJ) and distinctive facial features: relatively large head, widely spaced down-slanting eyes, sparse eye brows, small chin, curly sparse scalp hair. Failure to grow, dry scaly skin, oral aversion, feeding difficulties, intellectual impairment (EJ’s IQ= 54) and seizures are in reported cases. Visual problems are common5.

Vitamin A is an essential component of rhodopsin, a protein that absorbs light in the retina and supports the normal differentiation and functioning of eye epithelium. Night blindness is an early symptom of Vitamin A deficiency. Xerophthalmia (greek for dry eyes), which was EJ’s presentation, is a later sign. The conjunctival membranes and cornea become dry, thickened and inflamed with clumps of keratin debris. There is reduced tear formation. It can progress to ulceration of the eye and total blindness and is the most common cause of childhood blindness worldwide5-8.

Vitamin A aids in the utilisation of dietary protein and is necessary for growth and cell differentiation, immunity and steroid production4. It appears to have an anti-inflammatory effect and to be protective against free radical damage, environmental toxins, and some cancers. It is necessary for some of the actions of vitamin D, and has modulating effects on vitamins E and K5-9.

Had the Vitamin A therapy contributed to EJ’s osteoporosis?

There are studies linking Vitamin A ingestion, even in non-excessive amounts, with osteoporosis.

A review of 20 clinical trials from a MEDLINE search in 2004, admitted limitations to the studies, but consistently linked higher intake of vitamin A and serum retinol (and not B carotene), beyond only double the recommended daily allowance, with higher risk of fracture10.

The Nurses’ Health Study published in 2002 showed a significant increase in hip fracture among postmenopausal women, not taking hormone replacement, above a modest intake of both supplemental and dietary vitamin A (> 1,700 iu/day), even when adjusted for other factors11.
A 1998 Swedish study showed intakes > 5000 iu/day of vitamin A was associated with a 10 percent decrease in bone mineral density (BMD) at the hip and a doubling of hip fractures\(^1\). The Rancho Bernardo study in 2002, showed different results: a U shaped curve between vitamin A intake and BMD. Best density at intake 2000-2800 iu daily. Low intake, identified from food sources, had a dose dependent increase in BMD. High intake, from supplementation, had a dose dependent decrease in BMD\(^2\).

Similar U curves were found in studies in 2004\(^3\) and 2006\(^4\). Analysis of data from the Iowa Women's health study (>41,000 post menopausal) found no association with dietary A intake but an increase of hip fractures with supplemented A. The study has been criticised for only using a one-week food frequency questionnaire\(^5\).

Conversely in a UK 2005 study by Barker and others, there was a 15 per cent decrease in osteoporotic fractures in women over 75 years in the highest quintile of vitamin A from cod liver oil or a multivitamin\(^6\).

Ballew and others in 2001 failed to find any relationship\(^7\) but have been criticised for their data analysis\(^8\). New light is shed however when Vitamin A is looked at concurrently with Vitamin D.

Myhre and others did a retrospective study of vitamin A toxicity with information about vitamin D status. Vitamin D radically increased the dose of vitamin A needed to cause toxicity by an enormous 2,300 iu/kg. For a 75kg person this equates to an extra 175,000 iu/day\(^9\). A 2004 animal study showed no negative effects of vitamin A up to very high doses on bone mineralisation provided sufficient vitamin D was present\(^10\). Potential bone toxicity of A was switched off with D less than a recommended intake of 2000 iu daily\(^11\).

Re-evaluating the Nurses' Health Study, the average consumption of vitamin A, in the top quintile of vitamin A consumption, was only 1000 iu. In the 1998 Swedish study it was only 400 iu. Vitamin D, in the lowest quintile of both these groups, was only 200 iu, one tenth of the level to switch off A's bone toxicity\(^12\).

Cod liver oil was used regularly to treat rickets and eye conditions from the mid 1800's\(^13\). Cod liver oil was used up to very high doses on bone mineralisation provided sufficient vitamin D was present\(^14\). New light is shed however when Vitamin A is looked at concurrently with Vitamin D. Vitamin A was initially given to treat urgent eye disease, Xerophthalmia, related to vitamin A deficiency. The Vitamin D deficiency was only replaced, with other nutrients, once the eye disease had reversed. Despite ongoing vitamin A and vitamin D replacement, EJ had a blood test showing a severe vitamin D deficiency (23.1 nmol/l, normal range 60-160) 3 years into my care and then again at 10 years (23 nmol/l). Blood tests for vitamin A, from the same blood samples, were also deficient (0.49 and 0.27 umol/l, normal range 0.9-3.00). The eye disease did not relapse despite the ongoing deficient vitamin A measurements. The supplementation of vitamin A may have been too low to have significantly contributed to the osteoporosis even in the presence of low D. The vitamin D was low for an uncertain duration. Low D, with years of malnutrition of protein, vitamins and minerals with delayed growth and puberty, in this unique case, pales the significance of vitamin A as a contributor to the debilitating osteoporosis. For a precautionary approach to osteoporosis, where vitamin A is deemed necessary, is would be prudent, on the basis of this research, to ensure adequate D is present from the outset of A supplementation and all other potential causes of devastating bone loss addressed as comprehensively as possible, as early as possible.

Every person's metabolism is unique. The current enteral tube feeding is meant to provide 80 percent of EJ's recommended daily allowance. It is likely to be inadequate to prevent further nutritional deficiencies. EJ is now more resistant to tasting and consuming whole foods. How can a man made formula adequately supplemented when embarking on vitamin A replacement. Vitamin A was initially given to treat urgent eye disease, Xerophthalmia, related to vitamin A deficiency. The Vitamin D deficiency was only replaced, with other nutrients, once the eye disease had reversed. Despite ongoing vitamin A and vitamin D replacement, EJ had a blood test showing a severe vitamin D deficiency (23.1 nmol/l, normal range 60-160) 3 years into my care and then again at 10 years (23 nmol/l). Blood tests for vitamin A, from the same blood samples, were also deficient (0.49 and 0.27 umol/l, normal range 0.9-3.00). The eye disease did not relapse despite the ongoing deficient vitamin A measurements. The supplementation of vitamin A may have been too low to have significantly contributed to the osteoporosis even in the presence of low D. The vitamin D was low for an uncertain duration. Low D, with years of malnutrition of protein, vitamins and minerals with delayed growth and puberty, in this unique case, pales the significance of vitamin A as a contributor to the debilitating osteoporosis. For a precautionary approach to osteoporosis, where vitamin A is deemed necessary, is would be prudent, on the basis of this research, to ensure adequate D is present from the outset of A supplementation and all other potential causes of devastating bone loss addressed as comprehensively as possible, as early as possible.

**Conclusion**

The literature supports that adequate vitamin D offsets any detrimental effect of vitamin A in relation to bones. Ideally, adequate levels of vitamin D need to be confirmed, and if low, appropriately supplemented when embarking on vitamin A replacement. It is likely general malnutrition, pubertal failure and vitamin D deficiency have been the main contributors to EJ's catastrophic osteoporotic fractures.
connective tissue weakness. It needs to be a team approach to continually monitor and individualise her treatment.

EJ remains slight, height 131cm, weight 32 kg with a curved spine and low lying ribs. Her running days are over at age 21 years. She only walks now due to back pain. Intermittently she uses a manual wheelchair. She swims once a week and has a hydrotherapy exercise program. She goes to adult day care and TAFE twice weekly. At other times she is fully cared for by her mother. She is loved and lovable and still adores horses and devours her potato chips.

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CASE STUDY: Dientamoeba Infection & Post-Infectious Dysbiosis

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Abstract

This case study reports on a 12 year old child who presented with failure to thrive over a one-year period; weight loss, anorexia, intermittent cramping abdominal pain after a gastrointestinal illness. After a normal panendoscopy, she was due to be admitted to hospital and be force-fed. Dientamoeba fragilis was subsequently found on stool testing. She was noted to have a dysbiosis on Comprehensive Digestive Stool Analysis. Once the Dientamoeba was eradicated and the dysbiosis treated, the patient started to regain her weight and lost her previous symptoms. At no time in the intervening year did she comment on having any diarrhoea (commonly associated with Dientamoeba), bloating or wind (typical IBS symptoms). It is unknown whether eradicating the Dientamoeba produced the improvement in the patient, whether treating the dysbiosis caused the symptoms to abate, or whether a combination of both brought about her cure.

Introduction

Dientamoeba fragilis infection has been associated with IBS in the literature but usually with the typical IBS symptoms of diarrhoea, flatulence, explosive stools & bloating. Preiss et al noted that abdominal pain was the most common complaint in children with chronic infections. Spencer et al noted that symptoms were diminished or eliminated after treatment and hence D. fragilis should be considered pathogenic in children with GI symptoms.

Dysbiosis, an alteration in the bowel flora and its activities, is thought to contribute to IBS. Lyra et al noted an infectious trigger and the identification of changes in bacterial composition playing a role in causation of IBS symptoms.

This case study examines whether Dientamoeba or dysbiosis or a combination of both caused a severe gastrointestinal problem and subsequent general malaise in an otherwise healthy twelve year old child.

Case Presentation

KC, a 12 year old, presented with her mother, with a history of having suffered a gastrointestinal illness with nausea, vomiting and diarrhoea, approximately one year previously. This had lasted approximately three days. Three months later, she had fainted at school then suffered another gastrointestinal type illness.

Since the initial illness she had failed to put on weight and had become anorexic. She felt sated very easily. Whilst having been always thin (and her family being of a lean predisposition), KC had started to lose significant weight during this time (3-4kg). She felt tired and unwell, had poor concentration at school and couldn’t achieve the sporting level that she had been able to achieve previously.

KC had attended her LMO and been referred to a local paediatrician. Tests done at that time showed a cortisol of 488, TSH 3.79, free T4 14.19, ferritin 74, CRP-HS 0.8. She had had a panendoscopy which showed slight stomach inflammation, and biopsy for coeliac disease was negative. She was due to see a further paediatric endocrinologist later.

Up to the time of her illness, KC was a normal child. She had been breastfed for 6 months. She had had glandular fever when she was 5 years and helicobacter when 7 years of age. This had been treated with the usual antibiotics. She had been on a gluten free, dairy free diet since then. Her mother had worked on a vegetable farm, including the using of sprays. Her siblings were all healthy & thriving. All children had been vaccinated fully.

On examination KC weighed 26kg (<3rd percentile) and her height was 146cm (10th percentile). All her body systems were normal.

I sent her for blood and stool tests. I was concerned about post-infectious dysbiosis (but she seemed too unwell for just this), bowel parasites, subclinical hypothyroidism and the effects of chemical spraying from the farm.
The results of the tests were: histamine 0.9 (High), homocysteine 6.3 NR 4-8), free T4 15.6; free T3 5.5; serum copper 16 (<17.5), ceruloplasmin 0.27, blood lead <0.01; plasma zinc 8.9 (14-20), thyroid ab's normal, FBC normal; Vitamin B12 568, RBC folate 2436 (High), Vitamin D 91, EUCr normal, LFT's – AST 37 (10-35) & ALT 35 (5-30), BSL 4.2, ferritin 63, iron studies normal, lipids normal, CRP <0.4; pyrroles 9.4, Histopath – Dientamoeba fragilis, faecal fats negative. A CDSA (Genova) showed generally low probiotics, high LCFAs (fat malabsorption) and high gut perductive SCFAs (protein malabsorption).

Meanwhile KC had seen the paediatric endocrinologist whom her mother had found out was associated with an Eating Disorders Clinic. She wanted to admit KC and force-feed her by nasogastric tube, feeling that her history was one of a psychological problem. This doctor also ended up calling in the Department Of Community Services to interrogate the family on suspected child abuse. I wrote a letter to this doctor detailing my findings.

KC was started on Furazolidone, Nitazoxanide and Secnidazole, according to the Centre For Digestive Diseases protocol? for 10 days. During this treatment, she became more tired, had increased abdominal cramps and joint aches. It was felt she had suffered a mild Herxheimer reaction and this was discussed with her mother. One month after the treatment, a further Histopath stool culture was taken and it showed no evidence of Dientamoeba. KC was then started on SB Floracraft (saccharomyces boulardii -Biocuticals), Ultrabiotic 45 (a probiotic containing bifidobacteria, lactobacilli & strep thermophilus -Biocuticals), Intestamine (Glutamine, slippery elm, aloe vera, pectin - Biocuticals), Zinc picolinate 25mg od (Now Foods) and digestive enzymes.

KC was already feeling slightly improved but was still tired and had a poor appetite. Her mother was still worried by the DOCS view of a psychological problem. I felt she had suffered a mild Herxheimer reaction and this was discussed with her mother. One month after the treatment, a further Histopath stool culture was taken and it showed no evidence of Dientamoeba. KC was then started on SB Floracraft (saccharomyces boulardii -Biocuticals), Ultrabiotic 45 (a probiotic containing bifidobacteria, lactobacilli & strep thermophilus -Biocuticals), Intestamine (Glutamine, slippery elm, aloe vera, pectin - Biocuticals), Ultrabiotic 45 (a probiotic containing bifidobacteria, lactobacilli & strep thermophilus -Biocuticals), Intestamine (Glutamine, slippery elm, aloe vera, pectin - Biocuticals), Zinc picolinate 25mg od (Now Foods) and digestive enzymes.

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By another 2 months post treatment, KC’s weight had increased to 26.6kg. Because she was suffering some anxiety post illness, I tried her on Inositol. Vitamin C & Magnesium was also added, for antioxidant and anti-inflammatory effects. This was later changed to Multicare For Kids (NAC, glutamine, glutathione, Vit D, iron - Metagenics). By another 2 weeks her weight had increased to 27.1kg and her appetite had improved. Because of some early satiety persisting, her digestive enzymes were changed to another brand.

Ma KC felt quite tired over the next 3 months. Her weight had increased to 29kg. I sent her for an adrenocortex stress profile and it showed adrenal insufficiency (total daily cortisol 37.8). Her Zinc was again slightly low at 12.5 so her dose was increased. An intestinal permeability test showed gut hyperpermeability; a CDSA test showed continued low levels of probiotics so her probiotics, Intestamine & Multicare for Kids were all continued. Adaptogenic herbs were added to the regime. I discussed the use of fermented foods and referred her to a nutritionist for further dietary advice.

By 12 months post treatment, KC’s height was 154cm and weight was 31.1kg. She is now in high school, doing sports and eating well (mother has her on a very healthy diet!). She remains on her various supplements at present.

**DISCUSSION**

This case highlights how a chronic post-infectious bowel problem can markedly interfere with a young girl’s life, and how some medical specialties are quick to consider a psychological cause, when they can’t find what they consider to be the usual causes. It also raises the question as to whether Dientamoeba fragilis could cause such severe symptoms and the associated dysbiosis, or whether the dysbiosis, which may have already been present, was playing a larger role in the symptomatology.

Dientamoeba fragilis is a single celled intestinal protozoan, initially thought to be a harmless commensal, but recently linked to chronic diarrhoea, abdominal pain, nausea, anorexia and excessive flatulence. Indeed, it mimics many of the symptoms of diarrhoea predominant in IBS and can present as a gastro type illness. However, in children, abdominal pain has been found to be a more common symptom, especially in chronic cases. Peripheral blood eosinophilia has been seen in a third of children with dientamoebiasis. The patient in this study had only ever had diarrhoea during the initial gastroenteritis and one other bout later, mild intermittent cramping (with no pain for long periods in between), and no eosinophilia on blood testing. Yet she had been quite unwell. But, once treated, the abdominal and general symptoms started to abate.

The dysbiosis seen on testing can develop in up to 30% of patients with previous gastroenteritis. It can cause immune activation and low-grade inflammation, which can last 8 years or more. However, in this case, was it a consequence of the Dientamoeba infection, and did it amplify the chronicity of the symptoms, or merely a background change that was not responsible for the weight loss etc.? Interestingly, whilst the patient improved after the D. fragilis was eradicated, she seemed to improve even more (with quicker weight gain etc.), when the dysbiosis was also treated.

**CONCLUSION**

Certainly this case emphasises the pathogenic potential of D. fragilis in children, and as such all laboratories should routinely test for this organism in any child with ongoing GIT symptoms. Further studies could investigate the association between Dientamoeba fragilis & dysbiosis, and whether one influences the other in determining symptom severity in those infected by this parasite.
References


Conquer your PCOS Naturally
by Dr Rebecca Harwin

Dr Harwin is a Doctor of Chiropractic Medicine, and also holds two other undergraduate Science degrees. Within her book she has covered everything I would like to explain to my patients but do not have the time. It will be a useful resource for me to suggest to patients to read to further their understanding of the life-style changes they can make to manage the complex imbalances that underpin Polycystic Ovarian Syndrome. It will then allow me more time to focus on the associated medical aspects.

The introductory chapters explain the underlying hormonal and biochemical imbalances in an easy to read format for patients. It is well researched and I was particularly pleased to be reminded of the history of the evolution of the understanding and management of this imbalance.

Within the book she demonstrates how the Polycystic Ovarian Syndrome is not a disease of the ovary per se but a complex inter-relationship of hormonal and biochemical processes that interact to sustain the disorder, and lifestyle and management options that patients can develop themselves.

Within the testing chapter she makes reference to oestrogen and progesterone ratios but it was not clear that this ratio was based on saliva testing, which may be confusing for those readers who have been tested on blood.

A great amount of work had been put into the detail within the academic referencing, which unfortunately could be over-shadowed by the constant references to marketing of her monthly newsletters, bonus reports and references to her Facebook and Twitter pages. Without these self-references this book could have stood alone in discussing management options for patients under the guidance of their health care professional.

It would also be something I would have to preface before suggesting the book to my patients.

Likewise the monitoring of women for endometrial lining thickness was not addressed, and would need to be attended by the medical professional. The chapter on associated health risks may have been better placed earlier in the book, to emphasise the sequelae if the syndrome were not managed.

It was pleasing to see mention of the slim woman who can develop this syndrome, and discussion about maintaining ideal weight rather than just weight loss. There were also many options for self-management of stress, and to assist with movement and exercise. This also gave me more of an understanding of how chiropractors manage this area.

Overall I believe this could be a useful book for patients to read, to expand their knowledge and self-management of lifestyle changes, after medical assessment, and whilst working with their health care professional.

Reviewed by Dr Jennie McKern, MBBS, FACOHM, FACRRM, FACNEM

Published by Publishing Queen
Melbourne Training Event – November 2014

ACNEM's recent 4-day training event held at the Rydges Melbourne was a great success, with around 100 delegates attending over the weekend. We had a large number of new attendees completing the Primary Modules in NEM as well as many familiar faces in the Thyroid & Adrenal and A-Z of NEM modules.

The networking dinner was also well attended with around 50 delegates, staff and lecturers enjoying the networking opportunities and enjoying a lovely dinner at the Long Room restaurant.

The Annual General Meeting was held on Friday 21 November where the new Board was elected. For more information about the ACNEM Board please visit the ‘About Us’ page on the ACNEM website.

5th Science of Nutrition in Medicine Conference

The 5th Science of Nutrition in Medicine Conference will be held from 2-3 May 2015 at the Hilton on the Park Melbourne.

Conference themes include:
- Mental Health
- Heart, Diabetes & Weight
- Gastrointestinal Health
- Scientific Abstracts

The first conference early bird discount ends on 30 December so book now to get the best rate.

For up to date speaker and conference details please see the conference website: http://www.nutritionmedicine.org.au

ACNEM training in 2015/2016

The ACNEM Training Calendar has been confirmed, with the following courses to be presented over the next 18 months:

14-15 March, 2015 - Adelaide
- Primary Modules in Nutritional and Environmental Medicine
- Metabolic Conditions, Diabetes and cardiovascular Disease
- Allergy, Autoimmune & Dermatological Conditions

25-26 July, 2015 - Coolangatta
- Primary Module 1 in Nutritional and Environmental Medicine
- Cancer

19-22 November, 2015 – Sydney
- Primary Modules in Nutritional and Environmental Medicine
- Mental Health
- A-Z of NEM

12-14 March 2016 – Auckland
- Primary Module 1 in Nutritional and Environmental Medicine
- Children’s Health

Please visit the website for more info and to book online.
**New ACNEM Fellows**

Congratulations to Dr Frank Golik, Dr Natalie Ryan and Dr Mark Robertson for completing their ACNEM Fellowships recently at the Melbourne Training Event.

**Honorary Fellowship and ACNEM Awards**

At the recent November AGM, Louise Tijs was awarded an Honorary ACNEM Fellowship for her ongoing support of ACNEM as legal advisor.

Dr Vicki Kotsirilos was awarded an ACNEM Award for her ongoing support and collaboration with the College.

Dr Braham Rabinov, a long standing member of the Board and ACNEM Life Member also received an ACNEM Award for his enormous contribution to the College and the field of Nutritional and Environmental Medicine.
ACNEM EDUCATION

ACNEM Education is made up of learning modules, assessment tasks, practice logs, case studies and NEM Credit points. Each learning module represents approximately 15 hours of lectures.

Learning modules are available online and at face-to-face training events.

ACNEM PRIMARY MODULES

The ACNEM Primary modules are made up of two core modules. They provide foundation training in NEM, covering the key nutritional, environmental and biochemical factors in health and wellbeing, and treatment approaches to many of the conditions seen in primary care.

The course is pitched at a post-graduate medical level, providing the nutritional biochemistry not taught at medical school or during registrar training. With practical tools to aid integration into daily practice, the Primary Modules enables practitioners to begin practising Nutritional and Environmental Medicine confidently and safely.

Topics covered in Primary Modules 1 & 2 include:

- Introduction to NEM
- Gastrointestinal conditions
- Cardiovascular disease
- Allergies and food sensitivities
- Dietary history & the Low Stress Diet
- Cancer
- Mental health
- Chronic fatigue syndrome
- Vitamin C & antioxidants
- Migraines & other headaches
- Adrenal & thyroid conditions
- Women’s health & menopause
- Men’s health
- Childhood conditions
- Heavy metal toxicity
- The sensitive patient
- Arthritis, inflammation and pain
- Metabolic syndrome
- A-Z of hormones
- Contraindications & interactions
- Case histories
- Interactive panel discussions

LEARNING MODULES AVAILABLE

Online Modules
- Primary Module 1
- Primary Module 2
- Children’s Health
- Women’s Health
- Gastrointestinal Conditions
- Thyroid & Adrenal Conditions
- Epigenetics & Nutrigenomics
- Metabolic Conditions, Diabetes and CVD
- Mental Health
- Allergy, Autoimmune & Dermatological Conditions
  (available 2015)

ACNEM FACE-TO-FACE TRAINING

March 2015 Adelaide
- Primary Course in NEM – Modules 1&2 (14-15 Mar)
- Metabolic Conditions, Diabetes & Cardiovascular Disease (14 Mar)
- Allergy, Autoimmune & Dermatological Conditions (15 Mar)

July, 2015 - Coolangatta
- Primary Modules 1 in Nutritional and Environmental Medicine
  (25-26 July)
- Cancer (25-26 July)

November, 2015 - Sydney
- Primary Modules 1 in Nutritional and Environmental Medicine
  (25-26 Nov)
- Mental health (19-20 Nov)
- A-Z of NEM (21-22 Nov)

March, 2016 - Auckland
- Primary Modules 1 in Nutritional and Environmental Medicine
  (12-13 Mar)
- Children’s Health (12-13 Mar)
The 5th Science of Nutrition in Medicine and Healthcare Conference
Saturday 2 May – Sunday 3 May 2015 Hilton on the Park Melbourne

The Science of Nutrition in Medicine and Healthcare Conference is a highly anticipated event in the medical calendar, showcasing the latest scientific and evidence-based clinical applications of nutrition in medicine and healthcare.

International and local speakers will address the conference themes of Mental Health; Heart, Diabetes & Weight; and Gastrointestinal Health, with an overarching nutrigenomics theme throughout. Speakers for the 2015 Science of Nutrition in Medicine Conference include:

Prof John Funder MBBS, MD, MRACP, FRACP, PhD
Professor of Medicine, Monash University; Professorial Fellow, Centre for Neuroscience, University of Melbourne; Senior Fellow, Prince Henry’s Institute of Medical Research; Senior Fellow, Monash Institute of Medical Research; Professor, Institute of Molecular Biosciences, University of Queensland

Prof John Dixon MBBS, PhD, FRACGP, FRCP
Head of Clinical Obesity Research, Baker IDI Heart and Diabetes Institute, Melbourne; Head, Weight Assessment and Management Clinic, Baker IDI Heart & Diabetes Institute; Adjunct Professor, School of Primary Health Care, Monash University, Melbourne

Prof Joseph Proietto MBBS, FRACP, PhD
The Sir Edward Dunlop Medical Research Foundation, Professor of Medicine, and Head of the Metabolic Disorders Research Group in the Department of Medicine, Austin Health; The University of Melbourne Head of the Weight Control Clinic; Physician at the Endocrinology Unit at Austin Health

A/Prof Barbara Meyer BSc(Hons), RNutr, PhD
Associate Professor in the School of Medicine and Director of the Lipid Research Centre at the University of Wollongong, NSW.

Prof Jerome Sarris PhD, MHSc, Dip Nutrition
Senior Research Fellow at The University of Melbourne; Founding Vice Chair of The International Network of Integrative Mental Health (INIMH), and an executive committee member of the International Society for Nutritional Psychiatry Research.

Prof Ross Grant BEd, MAappSc, PhD
Biochemical Pharmacologist and Clinical Associate Professor at the Sydney Adventist Hospital Clinical School, University of Sydney and Head of the Australasian Research Institute.

Dr Paul Froomes BMedSci, MBBS, FRACP, MD
Visiting gastroenterologist at the Austin Repat Medical Centre and the Northern Hospital

Prof Mimi Tang MBBS, PhD, FRACP, FAAAAI
Director, Department of Allergy and Immunology, Royal Children’s Hospital; Group Leader, Allergy and Immune Disorders Research, Murdoch Childrens Research Institute, Department of Paediatrics, University of Melbourne

Prof Peter Gibson MD, FRACP
Professor and Director of Gastroenterology at The Alfred Hospital and Monash University

Prof John Dixon MBBS, PhD, FRACGP, FRCP
Head of Clinical Obesity Research, Baker IDI Heart and Diabetes Institute, Melbourne; Head, Weight Assessment and Management Clinic, Baker IDI Heart & Diabetes Institute; Adjunct Professor, School of Primary Health Care, Monash University, Melbourne

Prof Peter Clifton MBBS, BMedSci, FRACP, PhD
NHMRC Principal Research Scientist and Professor of Nutrition at the University of South Australia

Please refer to the conference website for up to date speaker and program information.

Abstracts and Scholarships
Submissions are invited for oral and/or poster presentations for the Scientific Abstracts stream of the conference. See the conference website for more details.

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