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Chronic Fatigue Syndrome gets a new name: Systemic Exertion Intolerance Disease

Emerging Concepts in the Pathogenesis and Treatment of Polycystic Ovary Syndrome

Case Study: The Management of Hypothyroidism
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Welcome to the first ACNEM Journal for 2015.

In this issue we have Part 2 of Nicole Bijlsma’s excellent article addressing the health hazards in the built environment and their potential effect on childhood neuro-behavioural disorders, following Part 1 in the December 2014 ACNEM Journal.

Nirupama Deshpande has provided another great article, this time on Chronic Fatigue Syndrome. Nirupama discusses a recent report proposing a new name and diagnostic criteria for the disease.

We also present a discussion on Polycystic Ovary Syndrome (PCOS) from Obstetrician and Gynaecologist, Jim Parker, reprinted from ‘Current Reviews in Women’s Health’.

Our case study for the April Journal explores treatment of hypothyroidism in general practice and comes from Dr Kate Norris, a recent ACNEM Fellowship graduate.

Also in this edition, we bring you ‘In The News’ from ACNEM staff member, Kelly Stuart. Kelly presents and discusses some recent articles in the field of nutritional medicine. We are very fortunate to have such talented staff on hand to provide up-to-date commentary on what is making news and we welcome any contributions and commentary from our members.

And we are also indebted to the contributors who provide such high quality articles for ACNEM members. We hope you enjoy reading this issue of the ACNEM Journal.
Health hazards in the built environment and their relationship to childhood neurobehavioural disorders — Part 2

Nicole Bijlsma, BHScAc(HONS), ND, Dip.Bi建ing Biology

Fluoride

Fluoride’s controversial history began in 1945 when it was first introduced into Michigan’s drinking water supply. The link between fluoride and a decline in children’s intelligence however was first highlighted twenty years ago from several Chinese studies where the levels of fluoride in well water varied significantly amongst the rural communities depending upon their geological conditions. A recent systematic review of 27 studies provided support that ingesting fluoridated water in levels above 1 mg/L may reduce IQ in children by 7 points. This was also observed by Cheng & Lynn (2013) who noted a 6 point reduction in IQ in children ingesting fluoridated water. Consequently fluoride was flagged as a neurodevelopmental toxin linked to neurobehavioural disorders in children. This is contrary to a recent NZ study which found no differences in IQ resulting from fluoride, though the authors were employed by the Faculty of Dentistry at the University of Otago, Dunedin, New Zealand.

Fluoride is classified as a pharmaceutical drug because it is not an essential nutrient required by the body and it is added to the water to treat a disease (tooth decay). Consequently it was rejected in the drinking water supply of 97% of Western Europe because it was considered unethical to mass medicate an entire population without informed consent. Consequently most countries do not fluoridate their drinking water. Whilst most would agree that topical application of fluoride has been shown to protect against tooth decay, the ingestion of fluoridated water and its role in the prevention of tooth decay remains controversial. Part of this may lie with the fact that toothpaste contains pharmaceutical grade fluoride as opposed to the industrial grade fluoride used in drinking water which is often contaminated with arsenic, lead, radioactive particles and other impurities because it is a by-product from the phosphate fertiliser industry.

In contrast to a breast fed infant, a bottle fed infant can receive up to 200 times more fluoride which substantially increases their risk for dental fluorosis because breast milk contains very little fluoride (0.006 ppm). Subsequently in 2006, the American Dental Association recommended that parents do not prepare infant formula with fluoridated tap water. There are some in the scientific community who argue that there is no adequate margin of safety from known harmful effects associated with fluoride.

Wireless Technology

In the past two decades, millions of children in industrialised countries have been exposed to varying levels of radiofrequency radiation from wireless technologies and the deployment of wireless infrastructure both in the school and home environment. These levels are up to 10^20 magnitude above the original background radiation since the birth of the universe. Given the inherent difficulties and ethics involved in conducting research on children in addition to the ubiquitous and changing nature of wireless technologies (frequency, amplitude, pulse, intensity, polarity and information content), the challenge in identifying biomarkers and the difficulty in establishing a control group, few clinical studies have been conducted on radiofrequency exposure and neurobehavioural disorders such as autism spectrum conditions. A recent systematic review concluded that the pathophysiology underpinning autism spectrum conditions are remarkably similar to those found from exposure to radiofrequency electromagnetic energy including but not limited to brain oxidative stress and inflammation, DNA damage, stress proteins, immune abnormalities, calcium channel dysfunction, disturbed circadian rhythms, degraded cognition and compromised blood brain barrier and brain perfusion.

Children are uniquely susceptible to the radiofrequency electromagnetic energy (RF EME) used in wireless technologies because unlike adults their skulls are thinner, they absorb twice as much microwave radiation, they are physically smaller in size, they have a longer lifetime exposure and they undergo rapid cell...
As a result of the increased risk in glioma associated with mobile phone use, on the 31st May 2011 the International Agency for Research on Cancer classified radiofrequency electromagnetic fields used in wireless technologies as a Group 2B carcinogen, i.e. possibly carcinogenic to humans. France and Belgium have consequently banned advertising mobile phones to children and some governments including Germany, France and Israel are recommending replacing wireless technology in schools with hard wired options instead. On 3rd July 2014, Telstra sent out a text to all of its users on how to reduce one’s exposure to the radiation from their products. As the manufacturers of this technology are not required to prove safety, the burden of proof falls on governments and researchers to prove harm; something that may take decades to achieve, essentially exposing generations of children to a hazard that even the World Health Organisation has raised concerns about.

Synergism and Additive Effects

Whilst epidemiological data on the interaction between electromagnetic fields and other environmental agents are scant and inconclusive, the combined effect was first raised in 1974 by three Soviet researchers who observed that irradiation of tissue by pulsed radiofrequency sources cause cell membranes to become more permeable to chemical mutagens. More recently, a systematic review of the combined biological and health effects of electromagnetic fields (EMFs) and at least one other agent was conducted using factor analysis amongst other methods. This paper highlighted both the beneficial effects (accelerated fracture and wound healing, limb regeneration in amphibians, enhanced drug delivery and bacterial inactivation for prolonged food storage) and the adverse effects EMFs may have on biological systems when combined with other agents. Adverse health effects identified were the ability of EMFs to interfere with DNA repair mechanisms, enhance the effects of known carcinogenic or mutagenic agents, enhance oxidative damage and increase glioma incidence in workers who were also exposed to solvents, lead and herbicides amongst other effects. Whissel and Persinger (2007) observed that very weak magnetic fields strongly potentiated the effects of drugs through opiate, cholinergic, dopaminergic, serotonergic and nitric oxide pathways and that these synergistic effects were several times larger than those evoked by the drugs alone.

Central to this combined effect is the ability of radiofrequency electromagnetic energy (RF EME) to increase the permeability of the blood-brain barrier (BBB); an intricate hydrophobic barrier that protects the brain from large proteins and water soluble chemicals through strict control of selective diffusion. Some studies have reported no changes to the BBB permeability whilst others have consistently reported increased BBB permeability after exposure to RF EME. The mechanism by which this radiation induces neuronal injury and increases the permeability of the BBB is suspected to be due to its ability to broaden and fracture intercellular tight junctions. The impact of exposing children to wireless technologies that increase the permeability of the blood brain barrier and placental barrier to chemicals, heavy metals and microbes is essentially unknown and warrants investigation as it may provide important clues to the rapid rise in neurobehavioural disorders in children. A recent Korean study found that the ADHD risk associated with mobile phone use was primarily observed in children with higher blood lead levels suggesting that increased permeability to the blood brain barrier may be involved. Despite this, this technology continues to be marketed and deployed in schools and to consumers, such that today’s children are subjected to different frequencies that are thousands of times more levels of radiation compared to when their grandparents were young. Not surprisingly, Kostoff and Lau (2013) concluded that the combined effects of EME with other agents were primarily synergistic in nature and should be the focus of a much more detailed study. Similarly Verschaeye and Maes (1998) concluded “we believe that synergistic investigations deserve special attention... it may well be that a radiofrequency exposure alone is ineffective whereas this exposure might enhance the mutagenicity, carcinogenicity or teratogenicity of chemical or physical factors.” This ‘allostatic load’ may be central to understanding how various risk factors interact to cause ‘intermittent’ autism and the wide array of symptoms amongst sufferers. This poses an important question: could the electromagnetic fields typically found in the built environment potentiate the effects of neurodevelopmental toxins commonly found in a child’s home?
CHILDREN ARE NOT LITTLE ADULTS

Prior to the thalidomide tragedy, it was widely assumed that harmful chemicals could not cross the placenta and that animal (rodent) studies do not reflect what happens in humans. How things have changed! Extensive epidemiological evidence supports a causal relationship between prenatal and early childhood exposure to environmental toxins such as lead, DES and alcohol, with adverse health outcomes in children. An emerging concern is the impact of endocrine disrupting chemicals during critical windows of development (which rodent studies adequately predicted) that at very low levels (non-nomotonic dose response) may derail reproductive development.

Despite the fact that children are more susceptible to environmental hazards, there is no legislation, national program, policy, agenda or organisation that specifically addresses children's environmental health in Australia. In contrast to adults, children have unique exposure pathways: in-utero (lead, mercury, PCBs, alcohol, PBDEs, alkyl phenols, DES, thalidomide, radiation) and breast feeding (susceptible to lipophilic chemicals including persistent organic pollutants, lead, mercury, nicotine, PBDEs and so on). Being at the end of the food chain, the body burden of chemicals in newborns is significantly higher kilogram for kilogram than most adults which is why the World Health Organisation use breast milk as a biomarker for the level of environmental contamination in the world.

According to Ginsberg (2002), children are more vulnerable to xenobiotics because they are in an anabolic state (they require more calories and water and are geared to absorbing nutrients very efficiently), phase I & phase II liver detoxification pathways are less efficient, the blood-brain barrier is not fully developed, and they have reduced renal elimination (reduced GFR). Their immune system is still developing which makes them uniquely susceptible to developmental immunotoxins such as chlordane, lead and DES. The growth and development of their respiratory system is not complete until 18-20 years of age which makes them more vulnerable to environmental tobacco smoke and mycotoxins.

Unlike adults, infants and children spend more time in fewer locations such as the bed, floor, high chair, and desk. This makes it even more important to investigate their exposure to hazards such as flame retardants which are an integral component of their bedding, low fire risk pyjamas and mattresses as well as to electromagnetic fields (the bed's proximity to fridge/oven and smart meter, wireless devices...), tap water (bathing and making infant formula) and associated use of personal care products as their level of exposure to these hazards may be greater. In contrast, school age children will be exposed to different environments depending on the school's proximity to major arterial routes, wireless technology and computers, pesticides and so on and so forth.

A child's respiratory rate is higher and their breathing zone is closer to the floor where dust and their associated contaminants (volatile organic compounds such as pesticides and flame retardants, house dust mites and microbes) are located. The Bhopal disaster in 1984 was a tragic example of children being more vulnerable as the dense gas cloud stayed closer to the ground. Pesticides and flame retardants are of particular concern as they are found in household dust. This may explain why polybrominated flame retardant levels in Australian children in the 0-4 years age group were twice as high as the 5-15 years age group and four times higher than the over 16 years age groups.

A US study conducted measured almost twice as many residues of metabolites of the pesticide chlorpyrifos in children aged between 6 and 11 than in adults. Another threat to children's health is the exposure to the mercury vapour from a broken compact fluorescent light bulb as mercury is denser than air; the level of exposure closer to the ground is likely to have a higher concentration.

A child may ingest up to eight times more dirt than an adult due to their exploratory behaviour. This hand to mouth activity may be a significant source of exposure for children 12 to 36 months of age as the house dust is often contaminated with numerous chemicals like PBDEs, lead and pesticides. Dust sampling can be a useful indicator of exposure to toxins in the built environment, particularly for young children who are in frequent contact with carpets.

Children undergo critical windows of development which makes them especially vulnerable to endocrine disrupting chemicals, with strong data sets showing that exposure to PCBs, lead and methylmercury early in life cause cognitive and behavioural deficits in humans. Longer life expectancies allow longer exposure to chemicals and wireless technologies and the development of diseases which have longer latency periods. Lastly, children do not recognise danger.

The Need For Further Research

Remarkably, the vast majority of hazards identified in this paper are found in the home and school environment where children spend up to 85% of their time. Despite this, few physicians in the medical or complementary therapy industries have an awareness of these hazards let alone why children are uniquely vulnerable to them, even though there has been a significant increase in data on this topic in recent times. There is definitive evidence that lead and mercury cause neurodevelopmental disorders, strong evidence that prenatal and postnatal exposure to pesticides and PBDEs may affect neurodevelopment and emerging evidence that radiofrequency electromagnetic energy used in wireless technologies may also be involved. Little is known about their interaction, synergistic or otherwise and many questions remain unanswered.

What is the body burden of neurodevelopmental toxins in Australian children?

Which neurodevelopmental toxicants are found in Australian homes and if so, at what levels?

Do these levels vary in healthy versus children diagnosed with neurobehavioural disorders such as autism?

Can radiofrequency electromagnetic energy used in wireless technologies potentiate the effects of neurodevelopmental toxins?

What strategies can be adopted to reduce one's exposure to these hazards?

Survey tools such as questionnaires that enable physicians and the public to adequately assess their exposure to hazards typically found in the built environment are woefully lacking. Technological advances however in the field of DNA sequencing (PCR), factor analysis and integrated environmental health impact assessments provide exciting opportunities to investigate the relationship between environmental hazards to childhood diseases.
Children live in a very different world compared to when their grandparents were young. The number of chemicals in their food, water and air has increased exponentially post WWII. In addition, wireless technologies have become an everyday part of their lives which, in conjunction with their increased body burden of chemicals may provide important clues to the pandemic of childhood neurodevelopmental disorders we are now seeing. Children’s health outcomes could be improved if the interactions between multiple hazards during critical windows of development were better understood. Reviewing the various hazards likely to be involved in neurodevelopment does not prove that these parallels imply causality, rather it emphasises the complex nature of neurobehavioural disorders in children and the need for research in this area. The challenge for researchers is to create studies and/or models that reflect real life scenarios and consider the synergistic and additive effects of multiple hazards such as chemicals, heavy metals, biotoxins and radiofrequencies typically found in a child’s environment. Integrated environmental health impact assessments, surveys and ongoing systematic reviews are needed as the issues concerned are complex and interwoven and are likely to have multiple causes, such that the interventions likely to arise from these outcomes may involve numerous organisations with far-reaching effects.

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Chronic Fatigue Syndrome gets a new name: Systemic Exertion Intolerance Disease

Nirupama Deshpande, PhD

**Abstract**

Chronic fatigue syndrome (CFS) is a serious debilitating disease that often profoundly affects the lives of patients. As many as 180,000 Australians are directly affected by CFS. An estimated 84 to 91 percent of people with CFS have not yet been diagnosed, meaning the true prevalence of ME/CFS is unknown. Thus, many people struggle with symptoms for years before receiving a diagnosis. Although many health care providers are aware of CFS, they may misunderstand the disease or lack knowledge about how to diagnose and treat it. Such gaps in understanding lead to delayed diagnoses and inappropriate management of patients’ symptoms. This probably stems from the fact that CFS remains an incompletely characterized illness, in part due to controversy regarding its definition, biological basis and diagnosis. Recently, a committee from the Institute of Medicine published a report1: Beyond Myalgic Encephalomyelitis/Chronic Fatigue Syndrome: Redefining an Illness, proposing new diagnostic clinical criteria and a new name—Systemic Exertion Intolerance Disease (SEID). This review briefly summarizes the findings of that report along with insights from latest research about the cause and cure.

**Background**

Chronic fatigue syndrome (CFS) is a complex disease involving profound dysregulation of the central nervous system2-4 and immune system5-9, dysfunction of cellular energy metabolism and ion transport10-12 and cardiovascular abnormalities13-15. CFS is also known by an alternate name: myalgic encephalomyelitis (ME)16, preferred by many experts as it underscores a physiological basis for the condition. The illness is now commonly referred to as ME/CFS. This disease is characterized by profound fatigue, cognitive dysfunction, sleep abnormalities, autonomic manifestations, pain, and other symptoms that are made worse by exertion of any sort. ME/CFS can severely impair patients’ ability to conduct their normal lives and can cause long-term disability. ME/CFS affects women more often than men. Most patients currently diagnosed with ME/CFS are Caucasian, but some studies suggest that CFS is more common in minority groups. The average age of onset is 33, although CFS has been reported in patients younger than age 10 and older than age 70. The cause of ME/CFS remains unknown, although in many cases, symptoms may have been triggered by an infection or other prodromal event, such as immunisation, anesthetics, physical trauma, exposure to environmental pollutants, chemicals and heavy metals, and rarely blood transfusions17. But fewer than one-third of medical school curricula and less than half of medical textbooks include information about ME/CFS. Furthermore, some health care providers have been skeptical about the serious physiological—rather than psychological—nature of the illness. Thus, diagnosing ME/CFS is a challenge, and patients often struggle with their illness for years before receiving a diagnosis, and an estimated 84 to 91 percent of patients affected by ME/CFS are not yet diagnosed. Once diagnosed, patients often complain of receiving hostility from their health care provider as well as being subjected to treatment strategies that exacerbate their symptoms.

**A new name for CFS**

For years, patients, clinicians, and researchers have debated changing the name of ME/CFS. Reaching consensus on a name for this illness is particularly challenging in part because its etiology and pathology remain unknown. Although a variety of names have been proposed for this illness, the most commonly used today are ‘chronic fatigue syndrome,’ myalgic encephalomyelitis, and the umbrella term ‘ME/CFS.’ But there are many researchers who maintain that ME and CFS are two different illnesses and oppose simply changing the name of CFS to ME22. According to the recent published report1, the term myalgic encephalomyelitis does not accurately describe major features of the disease as myalgia (muscle pain) is not a core symptom of the disease and there is lack of evidence for encephalomyelitis (brain inflammation) in ME/CFS patients. And the term chronic fatigue syndrome can result in trivialization and stigmatization for patients afflicted with this illness causing people not to take the disorder seriously23. The Institute of Medicine committee panel recommended

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that the illness be renamed: systemic exertion intolerance disease (SEID)\(^1\). According to the report\(^1\), the term SEID accurately describes the core symptom of the illness: a sustained depletion of energy after minimal physical, emotional or cognitive exertion, called post-exertional malaise. The report also stresses on the fact that ME/CFS (SEID) is a medical—not a psychiatric or psychological—illness.

**NEW DIAGNOSTIC CRITERIA FOR CFS**

Developing diagnostic criteria is easiest when medical tests can be used such as with HIV or hepatitis C, but much harder when an illness must be defined by its symptoms. Experts generally agree that the disease has a physical basis, but they have struggled for decades to characterise its symptoms. The most commonly used has been the CDC (Center for Disease Control) definition\(^24\), but many researchers and clinicians complain that those criteria identify many patients who more likely are suffering from depression and other conditions that can cause prolonged fatigue.

Researchers and clinicians have developed at least 20 different diagnostic criteria or case definitions over the years for the condition\(^24\). The new report\(^1\) lists the major symptoms of ME/CFS (SEID) and recommends a diagnostic process. This new diagnostic criteria include six months of profound, unexplained fatigue and post-exertional malaise, as well as a third key symptom: unrefreshing sleep. Patients must also exhibit cognitive problems or orthostatic intolerance (See Box 1). Physicians should diagnose approach but not meet the criteria for ME/CFS (SEID) include, for example, protracted recovery from EBV mononucleosis or gradual emergence of a different chronic illness, such as multiple sclerosis, colon cancer, or a primary sleep disorder. Comorbidities such as fibromyalgia and irritable bowel syndrome are common in ME/CFS (SEID) patients. These comorbidities should be diagnosed and treated when caring for patients. The presence of other illnesses should not preclude patients from receiving a diagnosis of ME/CFS (SEID) except in the unlikely event that all symptoms can be accounted for by these other illnesses.

**Box 1**

Diagnosis requires that the patient have the following three symptoms:

1. A substantial reduction or impairment in the ability to engage in pre-illness levels of occupational, educational, social, or personal activities, that persists for more than 6 months and is accompanied by fatigue, which is often profound, is of new or definite onset (not lifelong), is not the result of ongoing excessive exertion, and is not substantially alleviated by rest, and
2. Post-exertional malaise,* and
3. Unrefreshing sleep*

At least one of the two following manifestations is also required:

1. Cognitive impairment* or
2. Orthostatic intolerance

*Frequency and severity of symptoms should be assessed. The diagnosis of ME/CFS (SEID) should be questioned if patients do not have these symptoms at least half of the time with moderate, substantial, or severe intensity.

ME/CFS (SEID) if diagnostic criteria are met following an appropriate history, physical examination, and medical workup, including appropriate specialty referrals. Patients who do not meet the criteria for ME/CFS (SEID) should continue to be diagnosed by other criteria as their symptoms and evaluations dictate. These patients should also receive appropriate care. Conditions that may

**CURRENT RESEARCH**

Most patients develop ME/CFS (SEID) after an acute infection (e.g. flu-like illnesses, upper respiratory infections, glandular fever) but it may also be triggered by events such as toxic exposure (e.g. pesticides, heavy metals and environmental pollutants), physical trauma (e.g. major surgery or a serious accident), immunisation and anesthetics. Studies using molecular and physiologic methods suggest that the nervous and immune system dysfunction (in particular an abnormal response to infection) and mitochondrial malfunction play major parts in what is a complex disease process involving multiple systems of the body. But despite substantial efforts by researchers to better understand ME/CFS (SEID), no known cause or effective treatment has been identified. However, many unique physiological signatures have been determined. Abnormal cytokine patterns are thought to drive sickness behaviors and contribute to hypersensitivity to pain\(^5\), \(^6\). Most patients also have limited natural killer cell numbers and function. ME/CFS (SEID) patients have limited cardiopulmonary capacity and blood flow irregularities and therefore poor cerebral profusion, which could explain the intolerance to exercise and depressive symptoms\(^27\). Some research studies have suggested that neuro-inflammation to be the cause of ME/CFS\(^27\). Advanced imaging studies in ME/CFS found widespread neuro-inflammation in several key brain regions including the cingulate cortex, hippocampus, amygdala, thalamus, midbrain, and pons. Inflammation in these areas was 45%-199% times higher in ME/CFS patients than in healthy controls. The degree of neuro-inflammation directly correlated with symptom severity\(^28\). Further, inflammatory marker Leptin was found to correlate with the degree of neuro-inflammation in a small cohort of patients\(^29\). Lastly, a recent study showed reduced Brain-Derived Neurotrophic Factor (BDNF) in ME/CFS patients that was far greater than in multiple sclerosis patients suggesting an obstacle in neurogenesis and plasticity\(^30\).
Numerous studies have demonstrated mitochondrial insufficiency in ME/CFS patients perhaps due to a vicious cycle of oxidative stress\(^\text{31,32}\). Inflammatory cytokines and other immune cells propagate reactive oxygen species that damage cellular membranes, myelin, and mitochondria. As a consequence, studies have noted severely depleted CoQ10 in patients suggesting mitochondrial dysfunction\(^\text{31,32}\). Many studies have linked the cause of ME/CFS to viral infections\(^\text{33-39}\). Proposed candidates have included Epstein-Barr virus, human herpesvirus 6, enteroviruses, Borrelia disease virus, Borrelia burgdorferi, Coxiella burnetii, Candida albicans, Mycoplasma pneumoniae, and retroviruses\(^\text{33-39}\). A few years back, the xenotropic murine leukemia virus-related virus (XMRV)\(^\text{40}\) and polytropic murine leukemia virus (pMLV)-related gene sequences\(^\text{41}\) were found to be present in the blood of ME/CFS patients and was enthusiastically received as evidence of a tractable cause for ME/CFS. But a recent study showed no evidence of either XMRV or pMLV infection\(^\text{42}\). Most if not all people with ME/CFS have a gut disturbance such as irritable bowel syndrome, gastric reflux, constipation, diarrhea, and/or bloating. Research suggests altered intestinal microbiota may be linked to the pathogenesis of the illness\(^\text{43}\).

**Conclusion**

Beyond Myalgic Encephalomyelitis/Chronic Fatigue Syndrome: Redefining an Illness\(^\text{1}\) is a valuable resource to promote the prompt diagnosis of patients with this complex, multisystem, and often-devastating disorder. The committee believes systemic exertion intolerance disease appropriately captures the complexity and severity of the illness. Therefore, it recommends that patients, who meet the proposed diagnostic criteria, whether or not they have already been diagnosed with ME/CFS, should henceforth be diagnosed with SEID. Further they point out that the new diagnostic criteria will not improve outcomes for ME/CFS patients unless health care providers use them. Therefore, the committee recommends a broad nationwide dissemination, as the use of these criteria is essential to improve understanding of the disease, enhance public understanding and provide a firm foundation for future improvements in diagnosis and treatment.

**Treatment**

At this stage there is no universally successful treatment or cure for ME/CFS. The major focus is on managing the illness. The widely recommended approach to the treatment of ME/CFS is for a flexible, individualised illness management plan to be drawn up in relation to the person’s particular symptoms, total illness burden, illness history and other personal circumstances\(^\text{1}\). The need for the treating practitioner to recognise the biological pathophysiology of the illness, to respect the illness experience of the individual and for the person with ME/CFS to have autonomy concerning the complexity and pacing of activities has been emphasized\(^\text{16,44}\).

Alternatively, integrative and functional medicine offers some evidenced-based interventions that can be applied to manage severe symptoms of ME/CFS [SEID]. An anti-inflammatory diet could provide modest relief of pain or fatigue in many patients\(^\text{45}\). A nutrient-dense diet also provides abundant cofactors to promote energy production at the cellular level. Gut healing dietary strategies can also be utilised to ensure intestinal integrity and immune function. Many patients eliminate known gut irritants such as gluten, dairy, alcohol, and refined sugars\(^\text{45}\). Experimental evidence suggests that administration of probiotic bacteria may attenuate the underlying pathology of ME/CFS, namely systemic inflammation and oxidative stress\(^\text{46}\). Probiotic bacteria have also been demonstrated to influence HPA-axis function and mood in humans, which may be of particular relevance to ME/CFS sufferers\(^\text{47}\). Some studies have suggested the need for mitochondrial nutrients along with basic vitamin and mineral cofactors\(^\text{47}\). One study showed significant improvement with just 2 months of supplementation with a multivitamin/multimineral\(^\text{48}\). Immune boosting nutrients are commonly utilized to support a healthy immune response, prevent viral reactivation, and improve NK cell function. These include such nutrients as transfer factors, medicinal mushrooms, and curcumin\(^\text{49}\). Anti-inflammatory nutrients are also indicated including omega 3 oils, vitamin D, and antioxidants like resveratrol and EGCG.
Emerging Concepts in the Pathogenesis and Treatment of Polycystic Ovary Syndrome

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Abstract

Polycystic ovary syndrome (PCOS) is a multifactorial disorder that becomes apparent during adolescence with a variety of hormonal and metabolic symptoms. Patients with PCOS can present with ovulatory dysfunction, polycystic ovaries, androgen excess, metabolic abnormalities or a combination of some or all of these problems. The cause of PCOS is unknown but studies suggest a strong genetic component that is affected by the gestational environment and lifestyle factors. Recent advances in our understanding of genetics, diet-induced inflammation, gut microbiome, epigenetics and molecular toxicology suggest that there may be multiple mechanisms that could contribute to the variety of clinical presentations observed in PCOS. Prepubertal metabolic dysfunction may be one of the first phenotypic traits observed in adolescent girls likely to develop PCOS. In the future it may be possible to identify girls at risk of developing PCOS and implement preventative measures prior to the onset of clinical signs and symptoms. PCOS can be effectively treated with a combination of lifestyle approaches including diet and exercise. There is emerging evidence that a high quality low glycaemic load (GL) diet may have an important role in improving anthropomorphic and metabolic outcomes in women with PCOS.

Keywords: Diet, epigenetics, microbiome, molecular toxicology, polycystic ovary syndrome.

Introduction

Patients with PCOS commonly present with menstrual irregularities such as amenorrhoea, oligomenorrhoea and abnormal bleeding and/or hyperandrogenic symptoms such as hirsuitism and acne. Although there is debate about the diagnostic requirements for PCOS the Rotterdam criteria are the currently accepted standard. Patients are diagnosed with PCOS if they have two or three criteria including oligomenorrhoea, biochemical evidence of androgen excess or ultrasound features of PCOS. PCOS affects 5-10% of reproductive age women and is the most common endocrine disorder in women. PCOS is associated with reproductive problems and is the most common cause of infertility. Women with PCOS have an increased risk of metabolic problems such as insulin resistance, diabetes, metabolic syndrome and cardiovascular disease.

The objective of the study has been to review the evidence relating to environmental and genetic factors that may be involved in the pathogenesis of PCOS. The study consisted of searching the literature for articles pertaining to genetic, epigenetic, nutritional and environmental factors related to the pathogenesis of PCOS. The role of genetics, chronic inflammation and the gut microbiome are discussed. The study presents an overview of the literature related to molecular toxicology and the possible relationship between environmental toxins and the development of PCOS. Specific examples are discussed to highlight the possible pathogenic mechanisms that may be involved in the development of PCOS. This was a review of studies involving human, animal, toxicology and industrial nanocatalyst exposure. The discussion is intended to provide a conceptual overview of the new field of molecular toxicology with respect to the pathogenesis of PCOS and is not an exhaustive review of the effects of all possible environmental toxins. A review of dietary treatment for women diagnosed with PCOS was also performed.

PCOS becomes symptomatic during adolescence and a number of possible childhood risk factors have been identified. These include premature adrenarche and pubarche, atypical sexual precocity, intractable obesity, metabolic syndrome, pseudoCushing syndrome and pseudo-acromegaly. Premature pubarche, the appearance of sexual hair before the age of 8 years, has traditionally been considered an extreme variation of normal.
In 1993 Ibanez et al performed a study of 35 postmenarchal girls with a history of premature pubarche and found that many girls with premature adrenarche went on to develop PCOS during adolescence. This type of longitudinal study is difficult to perform and as yet has not been replicated in other studies. Insulin-resistant prepubertal and peripubertal obesity may also be a risk factor for the development of PCOS. Insulin-lowering treatment with metformin has been shown to improve insulin-resistance, obesity and androgen levels in girls with premature pubarche. No studies were identified in this review that employed a dietary intervention strategy to alter the outcome of girls at risk of developing PCOS. Incidental discovery of a polycystic ovary and persistent physiological anovulation may also be risk factors for the development of PCOS. Most girls with anovulation for more than 2 years after menarche will have menstrual irregularities and are at increased risk of developing PCOS. If future research confirms these findings it may be possible to implement lifestyle changes in girls found to be at risk of developing PCOS to help reduce the impact of the clinical and metabolic disturbances that are usually seen.

Currently, PCOS is considered a polygenic trait that might result from the interaction between the environment and susceptible genomic variants. Both twin and familial studies have demonstrated a large genetic influence in the pathogenesis of PCOS. It has been proposed that the female fetus might be programmed to develop PCOS in adult life due to exposure to an excess of androgen in fetal life because of genetic or environmental factors. It is possible that environmental influences may therefore have a transgenerational effect and be inherited in the offspring of women with PCOS. A recent study found that the neonates of women with PCOS had increased oxidative stress markers that were strongly associated with their mother’s oxidative stress status. A study of the parents of adolescents with PCOS found an increased incidence of obesity and metabolic syndrome in the fathers and concluded that familial factors may be fundamental to the pathogenesis of PCOS.

Studies have shown disparities in the clinical manifestations of PCOS in populations from different ethnic backgrounds. Chinese patients with PCOS more often present with amenorrhea and hyperandrogenism and patients of Northern European ancestry have a higher incidence of hirsuitism. A cross-ethnic meta-analysis of genetic variants for PCOS found a common genetic risk profile in patients from Northern European and Chinese ancestry. Recent genome-wide studies have identified 11 genetic loci that are associated with PCOS. They occur in regions that contain genes important for gonadotrophin action and type 2 diabetes. It is possible that nutritional and environmental substances cause epigenetic changes in some of these genes resulting in the observed metabolic and endocrine features of PCOS. Hypersecretion of androgens from the ovary may be the primary event that leads to the development of PCOS by favouring excess leutenising hormone secretion and insulin resistance. Alternatively, there is evidence that insulin resistance may be the initiator, followed by hyperandrogenism. Future genome-wide association studies are expected to help determine the relationship between causal gene variants and the observed phenotypic and functional differences. Knowledge of the genetic and familial underpinnings of PCOS may also have significant health implications for the children of women with PCOS. There is a need for future studies to examine early lifestyle intervention measures in children at high risk of developing PCOS, prior to the onset of puberty and the development of clinical features of PCOS.

It appears likely that there are a number of patho-physiological mechanisms that may result in the observed clinical and laboratory features of PCOS. Obesity may play a role in the pathogenesis of PCOS as approximately 50% of women with PCOS are overweight or obese. Insulin resistance is commonly found in women with PCOS affecting 50-70% of women with the disorder. A large group of women with PCOS are lean and genetic polymorphisms have been identified in lean women with PCOS that may protect them from the development of insulin resistance and obesity. Lean women with PCOS have been found to have increased serum levels of advanced glycation and oxidation end products that may incite chronic inflammation.

Chronic low-grade inflammation has been implicated as a contributor to the pathogenesis of PCOS. Markers of oxidative stress and inflammation have also been found to be highly correlated with circulating androgens in women with PCOS. A dietary trigger such as glucose has been found to induce an inflammatory response and oxidative stress in women with PCOS. These inflammatory mediators may directly stimulate the polycystic ovary to produce androgens. Insulin resistance in PCOS is a post-receptor defect in insulin signalling that may be mediated by the proinflammatory cytokine tumour necrosis factor alpha. In reviewing the evidence on the role of chronic inflammation in the pathogenesis of PCOS, Gonzalez has hypothesised that diet triggered inflammation may underpin both the insulin resistance and ovarian dysfunction observed in most patients. These studies provide evidence that dietary or environmental influences may promote the development of PCOS in genetically susceptible women.
The Dysbiosis of Gut Microbiota (DOGMA) theory of PCOS has been proposed as an explanation for all three components of the syndrome, including menstrual irregularity, hyperandrogenism and the development of multiple small ovarian cysts. This microbiological hypothesis is a novel paradigm that suggests that poor diet results in disturbances of bowel bacterial flora causing mucosal damage and increased gut permeability. This allows lipopolysaccharide from gram negative colonic bacteria into the systemic circulation resulting in activation of the immune system. Immune cells release cytokines that interfere with intracellular insulin receptor function which drives up serum insulin levels. Increased serum insulin has a direct effect on the ovaries resulting in increased androgen production and interference with normal follicle development. Dietary interventions focused on improvement in diet quality may therefore be expected to result in improved metabolic and hormonal parameters.

There is an emerging body of evidence implicating environmental toxins in the pathogenesis of many chronic diseases. The body burden of xenobiotic chemicals has been described as the cumulative load of toxic substances in the human body. It has been estimated that there is human exposure to over 80,000 environmental chemicals with less than 5 percent subject to scientific study. These include industrial chemicals, pesticides, heavy metals and radioactive compounds. These are in the form of endotoxins or exotoxins. The dysbiosis theory of the pathogenesis of PCOS proposes an endotoxic model. The recent emergence of the field of molecular toxicology proposes an exotoxic model for the development of cellular dysfunction and chronic disease. This new paradigm proposes that nanogram levels of toxic environmental chemicals may disrupt cellular functions and subsequently lead to the development of chronic disease. The core assumption of regulatory toxicology is that experiments using high doses will reveal the potential effects of low doses. Numerous studies have shown that endocrine disrupting chemicals mimic hormones that act in the part-per-trillion concentrations and exert their effect via biologically relevant nonmonotonic dose response curves. The usual assumptions of conventional toxicology assessment may therefore not apply to environmental toxin exposure.

Endocrine disruptors (ED) have been defined as substances foreign to the body that have deleterious effects on the individuals or their descendants, due to changes in endocrine function. Many different ED are present in the environment (air, water and land) and food (of plant and animal origin). Xenosterogens are a group of ED that imitate the effect of oestrogen at extremely low concentrations. Environmental exposure to industrial xenosterogens, including bisphenol A from plastics, and other ED have been shown to be present in utero, cord blood, breast milk, children and adults. A number of studies have identified an association between ED and PCOS. It is possible that ED are involved in the pathogenesis or may exacerbate the clinical course of PCOS. Cumulative environmental insults may affect susceptible women who develop the clinical phenotype of PCOS. Further population-based studies are needed to investigate a causal role for ED in the development of PCOS.

Although there are a large number of epidemiological studies that show a clear association between many environmental substances and human disease there are relatively few studies that have examined the molecular mechanisms that may cause these effects. Most studies of xenosterogens have focussed on changes in gene expression resulting from interactions of contaminants with the estrogen receptor located within the cell nucleus. Wozniak et al showed that several xenosterogens alter intracellular signalling pathways that are initiated on the cell membrane surface. There are extensive data from nutritional carcinogenesis research examining the detailed molecular physiology of nutritional carcinogenic factors. Carcinogenic processes are known to involve a multi-step process of initiation, promotion and progression. Food related mutagens work through genotoxic and non-genotoxic pathways. Genotoxic pathways work on the level of DNA causing DNA damage. Non-genotoxic pathways affect the cell through tumour promoters such as inflammation, immunosuppression, oxidative stress and free radical production.

In addition, there are considerable data from chemical engineering research that may predict the likely mechanisms of action of how environmental toxins contribute to human disease. It is evident that there is increasing human health-related risks from exposure to industrial nanocatalysts. Many studies have evaluated the toxicity of industrial nanocatalysts in cell and animal models. A variety of molecular mechanisms have been identified for various nanocatalysts to reveal common toxicity-inducing pathways. These occur through a variety of effects on the immune system, at a membrane receptor level, via intracellular signalling pathways or by generation of reactive oxygen species. Substances such as heavy metals, solvents, pesticides, phthalates, polyaromatic hydrocarbons and any of the other environmental chemicals presently known, may have insidious detrimental cellular effects. These could conceivably exert their adverse cellular effects in ways similar to those described for nanoparticles. It would be prudent to apply the precautionary principle and minimise exposure to known environmental toxins in patients with PCOS until further scientific evaluation is performed.

A range of treatment approaches are usually recommended to ameliorate metabolic dysfunction, hirsutism and anovulatory infertility in women with PCOS. These include lifestyle modification, medical and surgical alternatives. The aim of treatment is to reduce future metabolic complications such as diabetes and cardiovascular disease, decrease clinical and biochemical hyperandrogenism, improve reproductive function and reduce adverse psychological outcomes. Obese women with PCOS and those with metabolic syndrome appear to benefit from metformin treatment in terms of higher HDL cholesterol, lower diastolic blood pressure and lower BMI. Lifestyle interventions, including diet and exercise, have been shown to be more effective than medical management in women with PCOS who are overweight. Ovulation induction can be achieved with medical or surgical treatments. Medical options include metformin, clomiphene citrate, aromatase inhibitors and gonadotrophin. Laparoscopic ovarian drilling is considered a second-line treatment option for women who have clomiphene citrate resistant anovulation. There is general consensus that lifestyle management options including diet, exercise and/or behavioural interventions should be recommended to all women with PCOS.
While lifestyle management is recommended as first-line treatment for PCOS, a recent systematic review concluded that there is limited literature on the optimal dietary composition that should be recommended. This review identified 5 studies that compared the effect of different diet compositions on anthropomorphic, reproductive, metabolic and psychological outcomes in PCOS. They were unable to perform a meta-analysis due to the heterogeneity of the studies, but concluded that a low glycaemic index diet was associated with improved menstrual regularity, metabolic parameters and quality of life. Weight loss improved the presentation of PCOS regardless of dietary composition. Poor quality diet has been found to be associated with obesity in patients with PCOS. Rodriguez et al performed a cross-sectional study of 100 women with PCOS to assess diet quality using a healthy index eating score. They found that the high rates of obesity in their study population were related to poor quality diet. Women with PCOS have been found to have a dietary pattern characterised by a greater amount of specific foods with a high glycaemic index. In particular, they reported that women with PCOS consumed larger quantities of refined cola beverages, white bread, fried and cooked potatoes, white rice, pasta and fast foods compared to matched controls.

High glycaemic load diets have been linked to the development of diabetes in women and in overweight PCOS patients. A high GL diet is a biologically plausible cause of PCOS as it causes a rapid and prolonged increase in blood sugar and insulin levels and development of insulin resistance. Hyperinsulinaemia then drives increased ovarian androgen production and halts normal ovulatory processes. The DOGMA theory of PCOS proposes that a diet high in saturated fat and refined sugars causes imbalances in gut microflora that result in increased mucosal permeability. This increased permeability allows gut bacterial endotoxins to enter the circulation and initiate a series of immunological and metabolic changes. These changes cause impaired insulin receptor function and insulin resistance, resulting in the observed clinical and laboratory features of PCOS. Hyperglycaemia has also been found to incite an inflammatory response in women with PCOS. Proinflammatory cytokines are known to promote insulin resistance and hyperandrogenism and may be key contributors to the pathogenesis of PCOS. The available evidence therefore supports the contention that a poor quality diet (high in fat and sugar and low in fibre) is likely to contribute to the pathogenesis of PCOS.

Several studies have shown a beneficial effect of a low glycaemic index (GI) diet in the management of women with PCOS. A Cochrane review was performed to assess the effectiveness of lifestyle treatment in improving reproductive, anthropometric, metabolic and quality of life factors in PCOS. They identified 6 randomized controlled trials that compared lifestyle treatment to minimal or no treatment in women with PCOS. They concluded that lifestyle intervention incorporating diet, exercise and behavioural interventions improved body composition, hyperandrogenism and insulin resistance in women with PCOS. The available evidence therefore supports the assumption that the quality, rather than the quantity, of the food consumed appears to play a role in shaping the anthropomorphic and metabolic outcomes in women with PCOS. This is in line with findings from the general population that obesity is positively associated with poor diet quality.

There is a need for future studies to evaluate diets with high nutrient quality rather than diets based on caloric content or macronutrient ratios. High nutrient quality diets contain organic vegetables, salad and fruits, nuts and seeds, pasture-raised lean meat and chicken and wild-caught fish. A high nutrient dense diet would therefore be low in saturated animal fat, low GI, low sugar, high fibre, high in essential fatty acids and high in antioxidant foods. This type of diet would minimise the environmental chemical load normally consumed in the diet such as pesticides, herbicides and growth-enhancing hormones. A high nutrient value diet would therefore exclude fast food that is high in saturated fat and hydrogenated oils, packaged food that is high in sugar, fat and salt and high GI food. These studies would need to include lean women with PCOS as there do not appear to be any specific studies that have evaluated lifestyle intervention in lean women with PCOS.

PCOS is a heterogenous disorder with a variety of clinical presentations and phenotypes. Not surprisingly, there are a number of theories on the pathogenesis of this complex disorder. These theories involve a possible role for the gut microbiome, chronic inflammation, epigenetics and molecular toxicity. All involve an inter-relationship between dietary or environmental triggers and genetic factors. The available evidence supports a strong link between poor quality diet, high in fat and sugar and low in fibre, and PCOS. Dietary advice should be provided to all women diagnosed with PCOS and current evidence supports a whole food, low glycaemic load diet, high in fibre and low in refined and processed foods. The future focus of dietary advice is likely to shift more towards diet quality and high nutrient dense food rather than on calories and macronutrients. The emerging field of molecular toxicology raises the possibility that a number of possible environmental toxins may cause cellular dysfunction and chronic disease. Many environmental toxins are endocrine disruptors and a number of studies have identified an association between endocrine disruptors and PCOS. It would be prudent to advise patients with PCOS to minimise their exposure to environmental chemicals until further evidence is obtained. In the future it may also be possible to identify genetically susceptible prepubertal girls and implement lifestyle preventative strategies prior to the onset of puberty and the development of clinical features of PCOS.

CONFLICT OF INTEREST
The author(s) confirm that this article content has no conflict of interest.

LIST OF ABBREVIATIONS
PCOS = Polycystic Ovary Syndrome
GL = Glycaemic load
GI: Glycaemic index
ED = Endocrine disruptors
DNA = Deoxynucleic acid
HDL = High-density lipoprotein
BMI = Body mass index
DOGMA= Dysbiosis of Gut Microbiota


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ABSTRACT

This case history discusses the nutritional and environmental factors involved in the development and treatment of Hypothyroidism in General Practice. Specific targeted dietary and lifestyle advice, along with crucial nutrient replacement, helped the optimisation of thyroid function in this case.

INTRODUCTION

Hypothyroidism is a common endocrine disorder in the Australian population, mainly affecting females. A 20 year follow up study in England found the annual incidence of primary hypothyroidism to be 3.5 per 1000 in women, although the actual number may be much higher due to the insidious presentation of symptoms. Iodine deficiency remains the commonest cause of hypothyroidism worldwide, whereas autoimmune thyroiditis is the most common aetiology in Western Countries.

CASE PRESENTATION

Ms S.Q. was a 32 year old administration officer who presented with worsening fatigue, constipation, hair thinning, cold intolerance and difficulty losing weight. These had been troubling her for several months and slowly worsening. She could not remember any obvious trigger when her symptoms started to develop.

Other symptoms included occasional abdominal bloating, decreased concentration, occasional myalgia and heavy periods. She also described feeling more low in mood recently.

Routine bloods had been performed by her regular GP with a TSH reading of 3.8, high B12 and folate, with normal iron studies. The only supplement she was taking was a B complex formula, with no other prescribed medication.

Past medical history: Ms S.Q. was born by forceps delivery and breastfed for 4 months. She had suffered recurrent bouts of tonsillitis in early years with resulting tonsillectomy. Appendicectomy was performed a few years later. Combined oral contraceptive was used from age 16 to 25. She developed some mild psoriasis in her late teens.

Diet history: Breakfast most days consisted of a green smoothie with a soy flat white. Lunch was usually a salad, sandwich or sushi, and she had meat with 3 vegetable servings most nights, pasta once per week and eating out at least one night per week. Most of her groceries were not organic and she consumed minimal seafood.

Social history: Ms S.Q. lived alone in an apartment in the city centre, working 40 hours per week. She was non-smoker and consumed 3-4 units of alcohol 2 nights per week.

Physical activity: Exercise included a 20 minute walk to and from work, with Pilates twice per week. She denied any current stressors with a good friendship circle and no financial worries.

Family history: Her mother also had thyroid problems and vitiligo.

Relevant examination findings included BMI 25.9, small goitre with no palpable nodules, dry skin, sparse eyebrows, cold hands and some mild scalp psoriasis. Objectively she was euthyemic with spontaneous speech.

Results: TSH 3.9, free T4 10.6, free T3 3.1, rT3 565, antithyroid peroxidase antibody 232, vitamin D 64, plasma zinc 8.9, spot urinary iodine 79, MTHFR compound heterozygous.

A working diagnosis of Hashimoto’s Hypothyroidism was made with specific nutrient and methylation deficiencies. Treatment options were discussed and it was decided to try diet, nutrient and lifestyle options before adding thyroid hormone replacement.

Treatment decisions: Ms S.Q. was counselled re dietary measures, which included a gluten free trial, reduction of goitrogenic foods...
with increased consumption of iodine and selenium rich foods. Supplementation with zinc picolinate 60mg was commenced, a multistrain probiotic, and an activated B complex. Stress management was discussed and encouraged, along with sensible sun exposure.

She was reviewed 2 months later with reduction in TSH to 3.1, increase in T4 (12), T3 (3.8) with symptomatic improvement. Her psoriasis had also resolved. She had been strict with her gluten free and other dietary suggestions.

**DISCUSSION**

Every cell in the body has receptors for thyroid hormone. These hormones are responsible for the most basic aspects of our body’s function, impacting all major systems. Thyroid hormone directly acts on the brain, the GI tract, the cardiovascular system, bone metabolism, red blood cell metabolism, gall bladder and liver function, steroid hormone production, glucose metabolism, lipid and cholesterol metabolism, protein metabolism and body temperature regulation.

The two major causes of thyroid disorders are iodine and/or selenium deficiency and autoimmune disease. Iodine is a crucial nutrient for thyroid function. It forms the backbone of thyroid hormone, and deficiency of iodine can cause both hypothyroidism and goitre. Australian soils are moderately depleted in iodine and can put the population at risk of iodine deficiency and the associated problems. Selenium is required to convert T4 into T3, with selenium deficiency exacerbating conditions caused by inadequate iodine intake. The most common autoimmune cause of thyroid problems is called Hashimoto’s disease. Some studies suggest that up to 90 percent of people with hypothyroidism have Hashimoto’s disease.

Iodine deficiency remains a significant problem around the world, both in industrialised and developing countries. Iodine is present in iodised salt, commercial dairy products certain breads and in seafood/sea vegetables.

Goitrogenic foods have been associated with thyroid problems. These include soy, millet, sweet potatoes, and cruciferous vegetables like cabbage, broccoli, brussel sprouts, cauliflower, bok choy, kale and collard greens. Cooking can reduce the levels of goitrogens in food. At relatively low concentrations, goitrogens decrease the uptake of iodine by the thyroid gland. Supplementing with iodine can often offset this effect, unless the exposure is in large amounts.

Selenium plays critical roles in thyroid hormone metabolism, DNA synthesis, reproduction and protection against oxidative damage and infection. Unfortunately, Australian soils are naturally depleted in this essential trace nutrient.

Maintaining adequate vitamin D levels is crucial for proper thyroid function. Vitamin D plays an important role in balancing and regulating the immune system, which is especially important for those with autoimmune thyroid disorders like Hashimoto’s. Vitamin D deficiency has been associated with autoimmune thyroid disease, and vitamin D supplementation has been shown to benefit autoimmune thyroid conditions. It may be possible to meet vitamin D needs through sun exposure and diet alone.

Low zinc status reduces the conversion of T4 to T3, and studies have shown that zinc supplementation can improve thyroid function.

Glutathione is the master antioxidant in the body. It protects against oxidative damage, and helps to balance and regulate the immune system. Glutathione has been shown to be low in patients with Hashimoto’s and improving glutathione status helps these patients. MTHFR genetic polymorphisms are associated with lowered glutathione levels and increased susceptibility to autoimmune diseases.

There’s a strong connection between intestinal health and thyroid function. Seventy to eighty percent of the immune cells in the body reside in the gut, and intestinal bacteria assist in the conversion of T4 to T3. Inflammation in the gut can increase cortisol levels, and high cortisol also reduces the conversion of T4 to T3. Low stomach acid, small intestine bacterial overgrowth and chronic parasitic, fungal or bacterial infections in the gut all contribute to increased intestinal permeability and systemic inflammation, both of which in turn can trigger or exacerbate autoimmune thyroid disease.

The importance of stress management cannot be overemphasised. Chronic stress impairs thyroid function in several ways:

- It depresses the function of the hypothalamus and the pituitary gland. These glands are responsible for telling the thyroid gland to produce thyroid hormone, so anything that affects them will also affect thyroid function.
- It reduces the conversion of T4 to T3.
- It promotes autoimmunity by weakening immune barriers. The gastrointestinal tract, lungs and blood-brain barrier are the primary immune barriers in the body. Stress weakens these barriers, weakens the immune system in general, and exacerbates autoimmune disease.
- It causes thyroid hormone resistance. In order for thyroid hormone to have a physiological effect, it must first activate receptors on cells. Stress, via its tendency to provoke inflammation, has been shown to reduce thyroid receptor site sensitivity.
- It causes hormone imbalances. Stress can raise cortisol levels, and prolonged elevations of cortisol decrease the amount of free, active thyroid hormone that is available to act on cells and tissues.
Conclusion

This case shows the possible aetiological factors contributing to hypothyroidism and the importance of addressing the underlying cause, rather than just prescribing thyroid replacement hormone. Understanding the biochemical processes affecting the functioning of the thyroid hormones and using appropriate diet, lifestyle and nutrients can have the ability to improve functioning of the thyroid and improve patients’ symptoms.

References


Obama Administration releases national action plan to combat antibiotic-resistant bacteria

The US Centres for Disease Control and Prevention (CDC) released a report in 2013 warning that, globally, we are quickly heading into the post-antibiotic era. To address the problem, on March 27, 2015, the US Government released a national action plan to combat antibiotic-resistant (AR) bacteria, along with the call to double funding to fight AR bacteria.

The emergence of drug resistance in bacteria is undermining our ability to treat bacterial infections and perform a range of modern medical procedures, including chemotherapy, surgery, dialysis, and organ transplantation. In the absence of antibiotics, even minor surgery and routine operations could become high-risk procedures. Many common infections will cease to have a cure. The WHO notes that this is an issue that pertains to even the most common of microbes - "it's not just about hospital superbugs".

Without effective antibiotics, tuberculosis becomes incurable. First there was TB, then multi-drug-resistant TB (MDR-TB) and now there is XDR-TB (extremely drug resistant TB). Resistant strains of gonorrhoea are also on the rise. The CDC estimates that drug-resistant bacteria cause 23,000 deaths and 2 million illnesses each year in the United States. Antibiotic resistance also threatens animal health, agriculture, and the economy.

Antibiotic resistance is a global problem that requires global solutions. The newly released Action Plan announced by the Obama administration is organised around five goals for collaborative action by the U.S. Government, in partnership with foreign governments, individuals, and organizations aiming to strengthen healthcare, public health, veterinary medicine, agriculture, food safety, and research and manufacturing.

The CDC estimates that up to half of all human antibiotic use is inappropriate or unnecessary. The Action Plan includes activities to foster improvements in the appropriate use of antibiotics by improving prescribing practices across all healthcare settings, preventing the spread of drug-resistant threats in healthcare facilities and communities, and continuing to eliminate the use of medically-important antibiotics for growth promotion in animals. Specific and measurable outcomes have been set to be achieved by 2020. These include:

- Elevation of antibiotic resistance as an international priority for global health and security.
- Enhanced capacity to identify antimicrobial resistant pathogens in more than 15 partner countries.
- Establishment of a common U.S.-European Union (EU) system for sharing and analyzing bacterial resistance patterns for priority pathogens.
- Development of a global database to collect harmonized quantitative data on the use of antibacterial agents in animals.

The report states that improved detection and control of antibiotic resistance in human and animal pathogens will be achieved through a ‘One-Health’ approach to disease surveillance that integrates data from multiple monitoring networks. This approach will significantly increase the currently very limited data and will provide information in a timely manner necessary to track resistant bacteria in diverse settings.

Liver Cancer

The World Cancer Research Fund International published the latest report in March 2015 for their Continuous Update Project (CUP) – an ongoing program that analyses global research on how diet, nutrition, physical activity and weight affect cancer risk and survival. The report is the most rigorous, systematic global analysis of the scientific research currently available on diet, weight, physical activity and liver cancer, and which of these factors increase or decrease the risk of developing the disease.

The CUP report analysed 34 studies on research about how lifestyle factors affect the risk for liver cancer. The studies included around 8.2 million people and 24,500 liver cancer cases. An independent panel of international experts reviewed the results.

Key Findings

- There is strong evidence that being overweight or obese increases the risk of liver cancer.
- There is strong evidence that consuming approximately 3 or more alcoholic drinks a day is a cause of liver cancer.
- There is strong evidence that consuming foods contaminated by aflatoxins (toxins produced by fungi) increases the risk of liver cancer. (Aflatoxins are produced by inadequate storage of food, and are generally an issue...
related to foods from warmer, developing regions of the world. Foods that may be affected by aflatoxins include cereals, spices, peanuts, pistachios, Brazil nuts, chillies, black pepper, dried fruit and figs).

- There is strong evidence that drinking coffee is linked to a decreased risk of liver cancer.4

The findings on being overweight or obese, and for coffee in this report are new. The findings for alcoholic drinks and aflatoxins remain strong and unchanged from the 2007 Second Expert Report5.

In the press release CUP panel member, Dr Stephen Hursting states, “We’re looking at a tsunami of obesity-related cancer coming. People really need to be aware of this issue, and we need more research on weight loss strategies and understanding the mechanisms so that we can break this connection.” He emphasised that, “the evidence on obesity and cancer is only getting stronger”3.

The report explains that body fat increases insulin-like growth factors and oestrogens, a process that stimulates inflammatory responses and creates a pro-carcinogenic environment. Body fat also increases the risk for type 2 diabetes, a known risk factor for liver cancer, and for non-alcoholic steatohepatitis, which can progress to cirrhosis and increase the risk for liver cancer4.

World Health Organization statistics state that liver cancer is the second most common cause of death from cancer worldwide, and rates of the disease are increasing5. Because liver cancer is usually diagnosed at an advanced stage, it is associated with a poor 5-year survival rate. “We now have a little more precision on the alcohol-liver cancer link,” Dr Hursting also said in the press release. “Above three drinks a day seems to dramatically impact the tumorigenic process and increase risk.”

Finally, the report found ‘limited’ though ‘consistent’ evidence that physical activity and fish consumption could decrease the risk for liver cancer, though further research is needed.

Physical activity may reduce risk of liver cancer through its beneficial effect on insulin sensitivity and body fat, the report explains, as well as decreasing chronic inflammation and oxidative stress. The omega 3 fatty acids found in fish and fish oils are hypothesised to inhibit carcinogenesis by inhibiting eicosanoid production4.

“Lifestyle factors are important contributors to the development of liver cancer, and even moderate changes in diet, alcohol consumption, and exercise can prevent it,” CUP panel member Hillel Tobias, MD, PhD, said in the press release6.

**EXERCISE AT ANY LEVEL MAY BENEFIT OVERWEIGHT PATIENTS WITH NONALCOHOLIC FATTY LIVER DISEASE**

Non-alcoholic fatty liver disease (NAFLD) - an accumulation of fat in liver cells that is not caused by alcohol - is the most common cause of chronic liver disease in the western world6. It most commonly occurs among overweight or obese individuals and those who have diabetes, high cholesterol or high triglycerides. A new study published April 2015, in the Journal of Hepatology, suggests exercise, regardless of its volume or intensity, may have significant benefits for overweight and obese individuals with NAFLD6.

While the condition causes no symptoms or complications for the majority of people, others may experience liver inflammation, known as nonalcoholic steatohepatitis (NASH). This may lead to liver scarring (cirrhosis) and, eventually, liver cancer or liver failure6.

Using a randomised, placebo-controlled clinical trial design, a team of investigators from the University of Sydney, enrolled 48 overweight or obese individuals with sedentary lifestyles, meaning they rarely engaged in physical activity. The participants were randomly assigned to one of four exercise groups for 8 weeks: a low-to-moderate intensity, high-volume aerobic exercise (LO:HI) group, a high-intensity, low-volume aerobic exercise (HI:LO) group, a low-to-moderate intensity, low-volume aerobic exercise (LO:LO); and a placebo group.

During the 8-week period, change in participants’ liver fat was assessed by magnetic resonance spectroscopy (MRS). Reduction in liver and visceral fat was seen with all exercise volumes and intensities6.

The researchers found that participants in the placebo group saw their liver fat increase by around 14%. Participants in the three exercise groups, however, showed improvement in liver fat with a reduction of about about 18 – 29%, with the greatest liver and visceral fat reduction found in participants in the HI:LO and LO:HI groups6.

Commenting on their findings, Dr. Johnson says:

“The results from our study show that all exercise doses, irrespective of volume or intensity, were efficacious in reducing liver fat and visceral fat by an amount that was clinically significant, in previously inactive, overweight or obese adults compared with placebo. These changes were observed without clinically significant weight loss.” In an editorial linked to the study, Dr. Roohit Loomba, of the University of California-San Diego, and Dr. Helena Cortez-Pinto, of the Hospital de Santa Maria in Lisbon, Portugal, say that although there is evidence to support that aerobic exercise - even in the absence of weight loss - may reduce liver fat in patients with NAFLD, “there is no data to support that exercise alone without weight loss can improve or reverse NASH.”

“The individual and joint effect of dose and intensity of exercise and their association with improvement in liver fat and other histologic features that are associated with NASH is a key research priority,” they add6.
References


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On the eve of our Conference we are delighted to announce that proceedings will be opened by Federal Minister for Health, the Honourable Sussan Ley MP.

The reputation of this event has been growing since its inception and we are privileged to have the opportunity to hear passionate clinicians and research scientists present the latest breakthroughs and research in nutritional science. Special thanks go to our loyal sponsors who have supported the work of our College and our mission for many years, and we are so pleased to welcome a number of new sponsors this year.

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That Sugar Film – Damon Gameau Speaker at Gala Dinner

Many of you will be aware of the recently released ‘That Sugar Film’ in which actor and filmmaker Damon Gameau exists on ‘healthy’ low fat food with a high sugar content for 60 days and develops, in this startlingly short time frame, the beginnings of fatty liver disease. We are excited to have Damon Gameau as guest speaker at our Gala Dinner.

Primary Modules in NEM

Please note that since we introduced the ‘half price’ offer for repeating the Primary Modules, hundreds of previous graduates have taken the opportunity to update their knowledge.

Practice Nurses working with a doctor practising NEM may be eligible to register for the Primary Modules at half price. Please contact mail@acnem.org for further details.

Fellowship

Congratulations to Dr Anjana Arunachalam and Dr Kate Norris who were both awarded their Fellowships during the Adelaide training weekend in March. Well done Anjana and Kate!

Adelaide Training

Held over the weekend of the 14-15 March at the Stamford Plaza, ACNEM presented the first two days of the Primary Course plus special training programs in Allergy, Autoimmune & Dermatological Conditions and Metabolic Conditions, Diabetes & Cardiovascular Disease.

Our Allergy module saw our first live cross during a training event when guest lecturer, Dr Ken Yeoh, presented from Singapore. We owe a big thank you to our dedicated Technical Manager, Max Wang, and Dr Yeoh’s son, Paul, who went to much effort to ensure this event ran seamlessly.

The online Allergy module will be ready to launch midyear on our Online learning site. Topics include allergy testing and low dose immunotherapy, the link to ear, nose and throat diseases, the link in dermatological conditions and dietary strategies. This course was received particularly enthusiastically by delegates and will prove to be extremely valuable in guiding practitioners in this confusing, complicated area.

Networking Dinner

Delegates and lecturers enjoyed a wonderful night of networking, with a delicious meal prepared from organic produce at Tara Restaurant. Tara is owned by ACNEM member and lecturer Mona Kaur. It was the final weekend of the Fringe Festival in Adelaide, and delegates and lecturers finished off the evening by enjoying a spectacular light show on the walk home from dinner.

ACNEM now on Facebook and Twitter

ACNEM now has a Twitter feed and a Facebook page.

Please visit our pages and follow us for up to date information, news, research and notice of upcoming events in the world of nutritional and environmental medicine.

You can also join in on the twitter conversation for the 2015 NIM conference by including #NIM2015 in your tweets.
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 foundational training in NEM, covering the key nutritional, environmental and biochemical factors in health and well-being, 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

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

November, 2015 - Sydney
- Primary Modules in Nutritional and Environmental Medicine
  (19-22 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)
6TH INTERNATIONAL CONFERENCE ON

The Science of Nutrition in Medicine and Healthcare

30 April - 1 May 2016
Sydney, Australia

Themes
- Allergy & Autoimmune
- Child & Adolescent Health
- Nutrition & the Environment
- Scientific (abstracts) stream

Who should attend
- Medical Practitioners
  - Specialists/Physicians
  - General Practitioners
  - Registrars
- Healthcare professionals
  - Nurses
  - Psychologists
  - Naturopaths
  - Nutritionists
  - Dieticians
- Scientists & Researchers
- Public health professionals

Registrations open July 2015
www.nutritionmedicine.org.au
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Understanding Metabolic Typing
Are You Treating the Tortoise or the Hare?

PART TWO: Monday 4th May, 2015 7:00pm – AEDT

FAST VS SLOW – IMPLICATIONS FOR CLINIC Janine Castle

What you will learn:
• What to look for in metabolic typing
• How do clients present as fast or slow types
• What symptoms characterize fast and slow metabolisms
• How to balance a metabolic type with supplementation
• Complimenting HTMA with pathology testing for metabolic types
• New case studies of fast and slow metabolisers

PART ONE: NOW Available on demand

THE SCIENCE OF METABOLIC TYPING Zac Bobrov

Don’t miss out on Part One! Our popular webinar, Part One: The Science of Metabolic Typing that was held in March is available on demand. If you missed out on this informative webinar, make sure to visit our website at http://www.interclinical.com.au/events.php to register for the on demand recording.

Are toxic metals such as lead and mercury affecting your patient’s health?
Is your patient suffering from an unidentified zinc/copper imbalance?
Which vitamin or mineral supplements are suitable for your individual patient’s needs?

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Reliable Clinical Data

InterClinical Laboratories provides reliable clinical data gathered from our fully accredited laboratory facilities on over 36 nutrient and toxic materials and 27 significant mineral ratios, documented in one of three formats including graphic, interpretive and comparative reports.

Advanced Interpretive Results

InterClinical Laboratories offer comprehensive, personalised reports highlighting the patient’s current mineral status with optional dietary and supplement recommendations. Practitioners are welcome to book a complimentary technical call to discuss any questions regarding their patient’s results.

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Cardiovascular disease (CVD) is the ultimate manifestation of the detrimental effects of a Western ‘diet of plenty’ and a sedentary lifestyle. Truly a modern epidemic, it shows no signs of abating despite the multitude of drugs and surgical options which have become broadly available. It has been said that you can’t medicate your way out of a problem you ate your way into, and this is why there is a tremendous opportunity for Practitioners of natural medicine to revolutionise the treatment of CVD. This seminar highlights the fundamental changes that have occurred in our understanding of the pathology and management of CVD, and the key role that you will play in the treatment of these patients in the future.

**Cardiovascular Disease: The Critical Gaps in Current Management and the Central Role of the Natural Medicine Practitioner.**

**JUNE - JULY 2015**

Cardiovascular disease (CVD) is the ultimate manifestation of the detrimental effects of a Western ‘diet of plenty’ and a sedentary lifestyle. Truly a modern epidemic, it shows no signs of abating despite the multitude of drugs and surgical options which have become broadly available. It has been said that you can’t medicate your way out of a problem you ate your way into, and this is why there is a tremendous opportunity for Practitioners of natural medicine to revolutionise the treatment of CVD. This seminar highlights the fundamental changes that have occurred in our understanding of the pathology and management of CVD, and the key role that you will play in the treatment of these patients in the future.

**Locations**

**QLD**
- Caloundra: Thursday 25 June
- Noosa: Friday 26 June
- Hervey Bay: Saturday 27 June
- Rockhampton: Monday 29 June
- Toowoomba: Tuesday 30 June
- Mackay: Wednesday 1 July
- Townsville: Friday 3 July
- Cairns: Saturday 4 July
- Gold Coast: Friday 10 July
- Brisbane: Sunday 12 July

**NSW & ACT**
- Ballina: Saturday 27 June
- Coffs Harbour: Sunday 28 June
- Port Macquarie: Tuesday 30 June
- Kingscliff: Thursday 2 July
- Baulkham Hills: Sunday 5 July
- Manly: Sunday 5 July
- Newcastle: Monday 6 July
- Forresters Beach: Wednesday 8 July
- Wollongong: Monday 13 July
- Sydney: Saturday 19 July
- Albury: Friday 24 July

**VIC**
- St Kilda: Saturday 18 July
- Glen Waverley: Monday 20 July
- Geelong: Tuesday 21 July
- Mornington: Wednesday 22 July
- Albury: Friday 24 July
- Melbourne: Sunday 26 July

**TAS**
- Hobart: Thursday 2 July

**SA**
- Barossa: Friday 17 July
- Adelaide: Saturday 18 July

**WA**
- Perth: Sunday 12 July
- Bunbury: Monday 13 July
- Albany: Tuesday 14 July

**NT**
- Darwin: Tuesday 14 July

**Speakers**
- Paul Mannion
- Laurence Katsaras
- Andrew Thurgood
- Nicola Reid
- Nathan Rose

**All Seminar Times**
- Registration: 2:30 to 3:00 pm
- Session 1: 3:00 to 4:30 pm
- Break: 4:30 to 5:00 pm
- Session 2: 5:00 to 6:30 pm
- Dinner: 6:30 to 7:30 pm

This seminar is recognised for Continuing Education and Development Points/Formal Learning hours with various associations. Please enquire with your individual association for more details.

**YOUR INVESTMENT**
- Account holders and students: $55.00 incl. GST
- Non-account holders: $110.00 incl. GST

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A very informative and well-presented seminar. My clients will definitely benefit from the knowledge I've gained.

HEIDI – NAPIER

HEIDI – NAPIER
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**MET4153 - 04/15**

**Cardiovascular Disease:**  
**The Critical Gaps in Current Management**  
**and the Central Role of the Natural Medicine Practitioner.**

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- **Session 2 5:00 to 6:30 pm**
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- **Paul Mannion**
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