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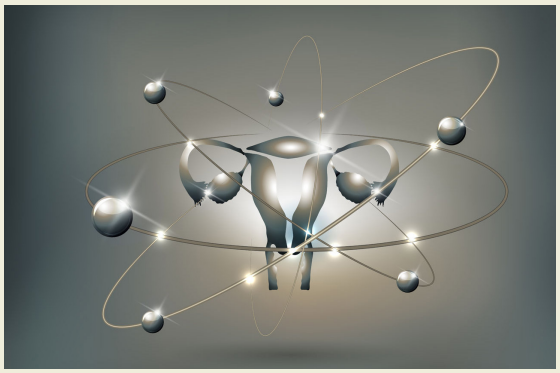

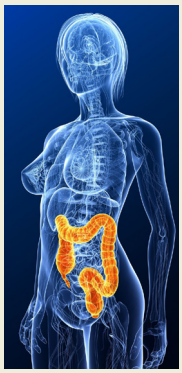

ABOUT ACNEM

The Australasian College of Nutritional and Environmental Medicine (ACNEM) Inc, is a not-for-profit medical college established in 1982, offering postgraduate training and education for doctors and other graduate healthcare professionals in Nutritional and Environmental Medicine.

WHAT IS NEM?

Nutritional and Environmental Medicine (NEM) is concerned with the interaction of nutritional and environmental factors with human biochemistry and physiology, and the resulting physiological and psychological symptoms and pathology. NEM is evidence-based, drawing on the latest biomedical and genetic science and research to develop new treatment approaches to illness and disease, for primary prevention and to promote optimal health and well-being.

Nutritional deficiencies, imbalances, or the presence of environmental toxins in the body can result in cellular dysfunction, illness or disease. Treatment is aimed at correcting underlying causes as well as providing symptomatic relief. This may involve removal of certain foods from the diet or toxins from the patient's environment, or prescription of supplements such as vitamins, minerals, trace elements and essential fatty acids where diet and lifestyle alone cannot rectify physiological imbalances.

ACNEM MEMBERSHIP

Ordinary Membership of the College is open to doctors and dentists. Associate Membership is available to graduate and practising affiliated healthcare professionals. Membership benefits include free access to the Natural Medicines Database, free online lectures selected from ACNEM Online Learning, the ACNEM Journal, a regular email newsletter, and discounts on education and training. Members of the public are also invited to become ACNEM Community members to support the work of the College.

ACNEM TRAINING

ACNEM training is regarded as unique in the world. The training programs are designed by the expert ACNEM Faculty for practitioners wanting to learn more effective ways of treating their patients. Content is strongly referenced and presented by some of Australia and New Zealand's leading clinicians. The emphasis is always on 'putting it into practice', with guidelines and practical tools to aid implementation. Training is held throughout the year at locations around Australia and New Zealand, and also by online learning.

ACNEM EDUCATION

ACNEM training optionally leads to Certification in NEM Parts 1 & 2 and Fellowship in NEM. The Education program is open to healthcare practitioners who meet Associate Membership requirements while the Fellowship is open to Ordinary Members (doctors and dentists). The ACNEM Primary Modules in NEM are the starting point for ACNEM Education and can be completed face-to-face, online or a combination of both.

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ACNEM is an accredited RACGP QI&CPD training provider for the 2014-2016 Triennium with 40 Category 1 points allocated to most training programs. ACRRM, RNZCGP and CPD/CME points from other professional organisations may also be available.

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From the Editor

Dr Jim Parker, BMed, BSc, DRANZCOG, FRANZCOG

Welcome to the June 2015 edition of the ACNEM Journal.

I would like to thank the ACNEM Board for inviting me to take up the position of Editor of the Journal. The quality of the research and publications in the previous Journals has been of the highest standard and reflects the professional nature of contemporary nutritional research. The presentation of the Journal reflects the hard work and dedication of the Executive Officer, Jimena Acevedo and her team.

I am an Obstetrician and Gynaecologist in private practice in Camden NSW and endeavor to integrate nutritional medicine into my specialist gynaecological practice. I am a Conjoint Senior Lecturer at the University of Western Sydney and am involved in medical student and registrar training.

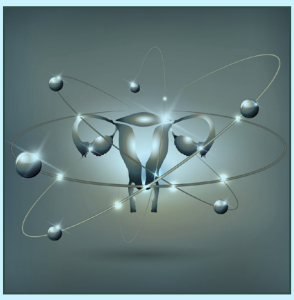
I have had a long-term interest in evidenced-based medicine and have published case series, retrospective and prospective studies, hypotheses, systematic reviews and clinical opinion. I plan to continue with this approach in the ACNEM Journal.

The recent ACNEM Nutrition in Medicine Conference in Melbourne was inspiring, educational and very successful. Not surprisingly, the Gastrointestinal stream focused on the microbiome and dysbiosis in the pathogenesis of many GIT diseases. The microbiome also featured in the Mental Health stream and many of the high quality plenary sessions. A more detailed review of the Conference is provided later in the Journal by Alison Coates and myself.

This issue of the Journal includes an original paper that follows on from the ideas developed in my paper 'Emerging concepts in the pathogenesis of polycystic ovary syndrome' that was published in the February ACNEM Journal. I hope this encourages other authors to contribute original research for publication in the Journal.

We have also included a review of the AHPRA/AMC 'Code of Conduct – Good Medical Practice', an interesting case report on inflammatory bowel disease, an article on cooking oils, a book review, a review of the best abstract from the ACNEM Conference and the 'In The News' segment. We plan to continue with this interesting diversity of commentaries.

I hope to build on the current co-operative team approach to produce a high quality Journal. I am passionate about promoting the rapid development of new ideas in NEM and helping to pass this on to the next generation of practitioners. I look forward to working with you all.



A new hypothesis for the mechanism of glyphosate induced intestinal permeability in the pathogenesis of polycystic ovary syndrome

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Abstract

Our understanding of the pathogenesis of polycystic ovary syndrome (PCOS) has been gradually evolving over many years. Studies suggest that there is a strong genetic component that is influenced by the gestational environment and subsequent lifestyle factors. The Dysbiosis of Gut Microbiota (DOGMA) theory has been proposed as a possible explanation of all the observed clinical and pathophysiological features of PCOS. This new paradigm suggests that poor quality diet results in disturbance of bowel bacterial flora causing mucosal damage and increased intestinal permeability. This initiates a cascade of events that results in the observed systemic features of PCOS. The human population is exposed to a large number of environmental toxins that result in a significant body burden of foreign chemicals. A number of these ubiquitous environmental chemicals have been identified as having a possible role in the pathogenesis of PCOS. The objective of the current study is to develop a new hypothesis for the possible role of the herbicide glyphosate in the existing pathogenic model.

Keywords: polycystic ovary syndrome, glyphosate, intestinal permeability, zonulin, indole

Main Text

Polycystic ovary syndrome affects 5-10% of reproductive age women and is the most common endocrine disorder in women[1]. Fertility problems are encountered by 15% of couples in Western countries and PCOS is the most common cause of female infertility[2]. PCOS is associated with significant clinical symptoms including menstrual disturbance, acne and hirsutism. The pathophysiological hallmarks of PCOS include chronic inflammation, hyperandrogenaemia and insulin resistance (IR)[3]. The pathophysiological and clinical manifestations are reversible following lifestyle modification using diet, exercise and weight loss[4-6]. PCOS is therefore considered to be a reversible metabolic condition rather than a disease with organ involvement[7]. If left untreated patients with PCOS may develop organ involvement from sequelae such as metabolic syndrome, diabetes, cardiovascular disease and endometrial cancer[3]. The personal and economic health burden of PCOS is therefore significant but has not been well studied[8,9].

Our current understanding of the pathogenesis of PCOS suggests that there is a strong genetic component that is affected by the gestational environment and subsequent lifestyle factors (Figure 1)[7]. Both twin and familial studies have demonstrated a genetic susceptibility for some women to develop PCOS[10,11]. The majority of patients with PCOS are obese and may have inherited the metabolic ability to efficiently store excess dietary caloric intake. In 1962 Neel proposed the "thrifty gene hypothesis" to explain why some humans have a genetic ability to efficiently store energy in times of famine[12]. This hypothesis suggests that we have inherited specific genes from our hunter-gatherer ancestors to regulate efficient intake and utilisation of fuel stores. Chakravarthy extended this hypothesis to link cycles of feast and famine to cycles of physical activity and rest[13]. This concept proposes that cycling of food stores, blood insulin, insulin sensitivity, and metabolic regulatory proteins, driven by cycles of feast/famine and physical activity/rest has moulded the selection of "thrifty" genes so that these metabolically advantaged individuals would have a survival benefit. Women with susceptible genomic variants exposed to a combination of continuous food abundance and physical inactivity would therefore be at increased risk of developing PCOS and diabetes.

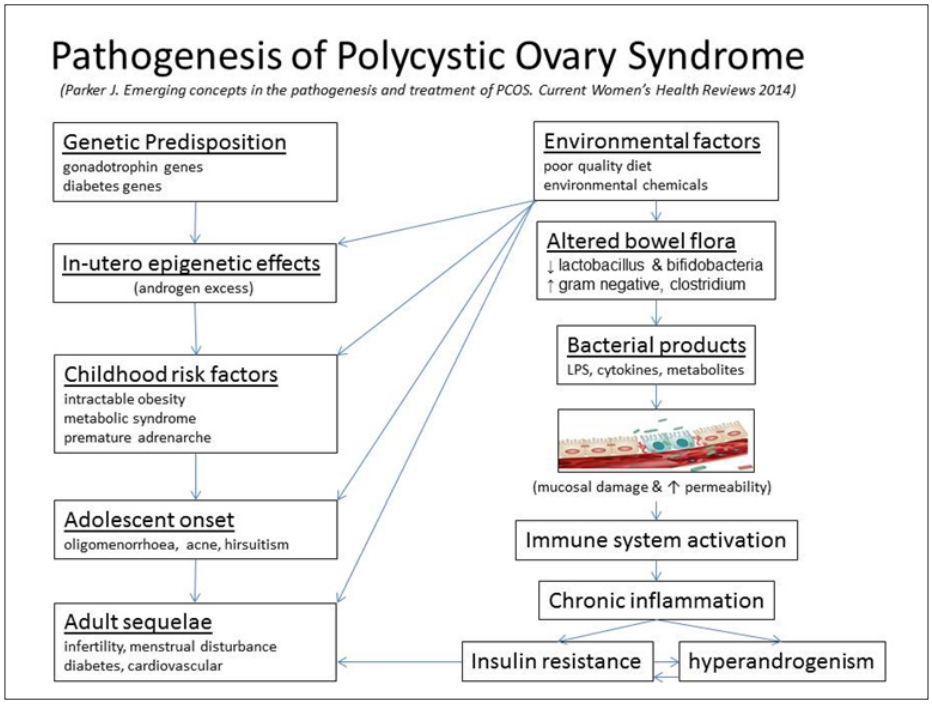

Figure 1. Pathogenesis of polycystic ovary syndrome (reprinted with permission of Bentham Science Publishers)

Recent advances in the understanding of the role of the gastrointestinal microbiome have resulted in the development of a number of new models to explain the pathogenesis of many chronic diseases[14]. The Dysbiosis of Gut Microbiota (DOGMA) theory has been proposed as an explanation to unify the observed clinical, biochemical and metabolic components of PCOS with our current understanding of the gut-related changes in the microbiome[15]. The DOGMA theory proposes that a diet high in saturated fat and refined sugars and low in fibre, favours the growth of gram negative bacteria in the gut and reduces the growth of beneficial bacteria such as Lactobaccilus and Bifidobacteria. This poor quality diet and other factors cause an increase in gut permeability facilitating the transfer of gram negative bacterial cell-wall derived lipopolysaccharide from the gut lumen into the circulation. Lipopolysaccaride is a known powerful immunostimulant that results in activation of the innate immune system once it enters the systemic circulation. This in turn activates macrophages resulting in chronic inflammation and insulin resistance. Hyperinsulinaemia drives the ovaries to produce androgens and halts the normal ovulatory process[15].

Before the DOGMA theory can gain more widespread acceptance, further "proof of concept" scientific studies are required. There is strong evidence that a poor quality diet, high in sugar and fat and low in fibre, can result in changes to the gut microbiome[15-18]. The majority of patients with PCOS are obese or have IR and both animal and human studies have shown that obesity and IR are associated with increased gut permeability[19-22]. A key function of the gastrointestinal tract epithelium is to regulate the trafficking of environmental antigens across the host mucosal barrier[23,24]. Intestinal tight junctions (TJ) are responsible for regulating paracellular trafficking of macromolecules and therefore contribute to a balance between tolerance and immune-response to non-self antigens[23]. A link between changes in the gut microbiome and increased epithelial cell permeability in patients with PCOS would further support the DOGMA hypothesis.

Recent studies have shown that the protein zonulin is able to reversibly regulate intestinal permeability by modulating intercellular TJ[23]. Small intestinal exposure to bacteria and gluten has been found to trigger zonulin release[25,26]. Small intestinal cells exposed to enteric bacteria secrete zonulin which transactivates membrane-bound epidermal growth factor receptor through proteinase activated receptor 2[24]. This zonulin signalling activates the intracellular zonulin pathway resulting in TJ protein phosphorylation and actin polymerisation. These changes cause displacement of proteins from the junctional complex, as demonstrated by freeze-fracture electron microscopy, resulting in disassembly of TJ[24]. Once the zonulin signalling is over, the TJ resume their baseline steady state. As a result of these and other studies, serum zonulin has become a biomarker of impaired gut barrier function for a number of diseases including autoimmune diseases, diseases of the nervous system, some cancers and metabolic diseases[23,24,27,28].

Further supporting evidence for the DOGMA hypothesis is provided by a number of studies in humans and enterocyte cell lines. A recent study found that serum zonulin is increased in women with PCOS and correlates with IR and severity of menstrual disorders[27]. This provides additional support for a role for alterations in gut permeability in the pathophysiology of PCOS. A study in Asian Indians found that circulating lipopolysaccharide and zonulin were both elevated in patients with type 2 diabetes[28]. Given the similarities in the pathophysiology of type 2 diabetes and PCOS, replication of this study in patients with PCOS would provide support for a link between lipopolysaccharide and zonulin-mediated increased intestinal permeability, as proposed in the DOGMA theory. Further supporting evidence would be provided by experimental studies directed at elucidating details of the signalling mechanism between enteric bacteria and gastrointestinal cells that secrete zonulin.

Interkingdom signalling is an established process in the gastrointestinal tract[29]. Human hormones have been shown to trigger responses in bacteria and bacterial signals have been found to stimulate intestinal epithelial cells[30]. The bacterial signalling molecule indole has been found to increase epithelial-cell TJ

resistance and attenuate indicators of inflammation in vitro[30]. Indole is produced in commensal E. coli from L-tryptophan[31] and is an extracellular signalling molecule that has been found to cause changes in the expression of multiple genes in intestinal epithelial cells[30]. This results in increased expression of anti-inflammatory genes, strengthened epithelial cell barrier properties, increased mucous production and decreased pathogen colonisation. Indole is therefore recognised as a beneficial signal by intestinal epithelial cells. It is conceivable that dietary components or ingested environmental chemicals may cause alterations of the gut microbiome that result in decreased indole secretion, epithelial cell damage, release of zonulin and increased paracellular trafficking of bacterial lipopolysaccharide into the submucosa, as proposed in the DOGMA theory.

A relatively unexplored component of the human diet that may have a significant impact on many of the pathophysiological mechanisms in PCOS is the role of environmental chemicals. Human exposure to environmental chemicals is ubiquitous and continuous on a daily basis[32-34]. Environmental chemicals have been found in the air, soil, water, packaging equipment, food and beverage packaging, common household items, carpet, children's toys, personal care products and commonly consumed food items[34-40]. This leads to daily human exposure via oral ingestion, inhalation and dermal contact. Biomonitoring data of multiple human populations suggest that 100% of the population is exposed to a variety of potentially toxic environmental chemicals. Chemical metabolites have been found in all human tissues and fluids examined. These include blood, urine, sweat, saliva, breast milk, ovarian follicular fluid, amniotic fluid and cord blood[7,38].

A range of environmental chemicals have been associated with PCOS[39-41]. These data are mainly from epidemiological studies of endocrine disrupting compounds such as bisphenol-A and phthalates comparing metabolite levels in patients with and without PCOS[39,40]. Glyphosate is suspected of having teratogenic effects[42,43,34] and the World Health Organisation recently classified glyphosate as "probably carcinogenic to humans"[44]. In the current review no studies were identified examining the effects of glyphosate in patients with PCOS. Given our current understanding of the pathogenesis of PCOS and the large body of scientific data on the toxic effects of glyphosate in the literature, it is possible to develop a number of hypotheses regarding the possible role of glyphosate in the pathogenesis of PCOS.

Glyphosate is the active ingredient in the herbicide Roundup[45]. The mode of action of glyphosate is through specific inhibition of 5-enolpyruvoylshikimate-3-phosphate synthase (EPSPS) which is an enzyme in the Shikomate pathway that governs the synthesis of aromatic amino acids found in plants, algae, bacteria and fungi[46]. This specificity to plants and microbes, along with its metabolic breakdown to aminomethylphosphonate and thereafter to carbondioxide, provided rationale for its development as a safe herbicide. Glyphosate occurs as a hydrophilic acidic salt which impairs its entry to lipid membrane bound cells[47]. The herbicidal activity of glyphosate is increased by adding surfactants and other adjuvants, and a number of these mixtures are marketed under the term 'Roundup'[48,49]. Subsequent studies of glyphosate toxicity have examined the effects of glyphosate alone or as a component of the Roundup mixture. The Roundup mixture has been found to be 1000 times more toxic than glyphosate alone[50].

Glyphosate has been found to have variable effects on different bacterial species. The EPSP synthase variants derived from some microbes, such as Agrobacterium tumefaciens species CP4, are not inhibited by glyphosate and are used to engineer glyphosate-tolerant genetically modified (GM) crops[51,52]. Some bacterial species are highly susceptible to the effects of glyphosate. Evidence for disruption of gut bacteria by glyphosate is available for poultry, cattle and pigs[53-55]. Shehata et al studied the effect of glyphosate on growth and viability of potential pathogens and beneficial members of poultry microbiota in vitro[53]. Most of the tested pathogenic bacteria were highly resistant to glyphosate and most of the beneficial bacteria, such as lactobacillus and bifidobacterium, were found to be moderate to highly susceptible. Bifidobacteria are considered beneficial microorganisms and thought to create conditions unfavourable to the growth of pathogens[56]. Bifidobacterium lactis was found to reduce epithelial permeability and improve the integrity of the TJ in human colon cells in vitro[57]. The effects of glyphosate on the human GIT microbiome has not been studied, but if glyphosate is found to have similar effects to those in other animals it would support the role of glyphosate in GIT dysbiosis and the pathogenesis of PCOS.

Glyphosate inhibition of the EPSPS enzyme in the Shikomate pathway results in decreased synthesis of the aromatic amino acid tryptophan[46]. Tryptophan is a precursor for the quorum-sensing molecule indole, that mediates intercellular signals in bacteria[58]. Indole is produced by a variety of gram-positive and gram-negative commensal bacteria that possess tryptophanase, a bacteria-specific enzyme that catabolizes tryptophan. A recent study indicated that indole enhances barrier functions of intestinal epithelial cells in-vitro by inducing the expression of several genes[30]. These genes included those responsible for TJs, adherens junctions, actin cytoskeleton and mucin production[30]. These transcriptional changes suggest that indole promotes intestinal epithelial cell barrier function and increases resistance to pathogen colonisation. An in-vivo study of colonic epithelium in germ-free mice showed that indole promoted the establishment of the intestinal epithelial barrier by inducing the expression of junctional complex molecules[59]. Microbial reconstitution experiments in commensal-depleted mice showed that bacterial indoles directly affect intestinal barrier properties in vivo through actions mediated by the pregnane X receptor[60]. Toll-like receptors have been identified on the luminal and basal surfaces of intestinal epithelial cells[61]. Studies of germ-free mice subsequently colonised with commensal bacteroides species have shown that certain toll-like receptors activate genes that regulate intestinal epithelial cell barrier function[61]. Indole-mediated pregnane X receptor activation has been implicated as a central regulator of toll-like receptor 4 mediated control of the intestinal epithelial barrier[60].

These findings suggest that indole, produced by commensal gut microbiota, plays an essential role in enhancing epithelial barrier function in the intestine. Glyphosate may affect intestinal epithelial barrier function by initiating a cascade of events that follows inhibition of EPSPS in beneficial gut bacteria. Glyphoste-mediated gut dysbiosis could therefore play a significant role in the pathogenesis of PCOS by causing decreased tryptophan synthesis resulting in reduced indole production and release from beneficial gut bacteria. Since indole has been shown to stimulate intestinal cell production of protective mucopolysaccaride[30], the loss of mucous may allow bacterial colonisation of the luminal side of the epithelial cells and activate the zonulin pathway as previously shown in several mammalian and human ex-vivo intestinal cell models[25]. The decrease in indole release from benefical bacteria would also lead to reduced transcription of genes regulating TJ barrier maintenance proteins resulting in increased intestinal permeability as previously described (Figure 2).

Our current understanding of the pathogenesis of PCOS is that poor quality diet that is high in fat and sugar and low in fibre, causes an imbalance of intestinal bacteria and increased

intestinal permeability. These changes initiate a cascade of events that results in the observed systemic features. The current paper advances the hypothesis that human ingestion of glyphosate in the diet could contribute to the imbalance of gut bacteria and subsequent increase in intestinal permeability. This hypothesis includes a detailed description of the possible biochemical and pathophysiological changes that may result from the known mechanisms of action of glyphosate from both in vitro and in vivo experimental studies. It is possible that dietary factors and environmental chemicals such as glyphosate act synergistically in the pathogenesis of PCOS. The current evidence supports the need for further experimental and clinical studies to test the hypothesis that glyphosate, ingested in the diet, may induce increased intestinal permeability and contribute to the pathogenesis of PCOS.

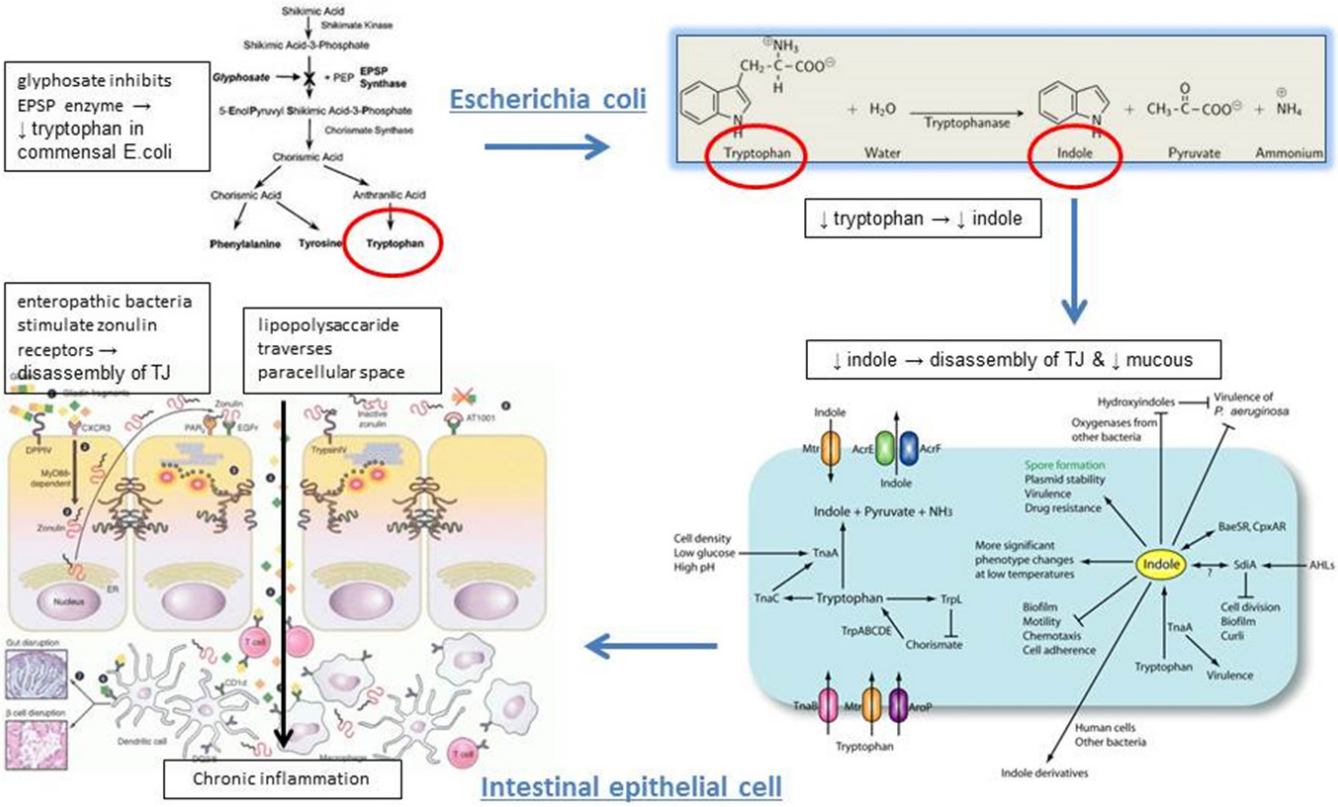

Figure 2. Pathogenic model for glyphosate induced loss of intestinal epithelial barrier integrity.

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Cooking with Extra Virgin Olive Oil

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Abstract

There are a variety of cooking oils used in Australia with a number of confusing messages available to both health professionals and consumers. It is integral that health professionals are aware of the major differences between common cooking oils including details on the smoke point of various oils and how they behave under conditions of thermal stress. The current paper is a review of available cooking oils and compares their stability when heated with respect to smoke point and oxidative stability. This review also compares the interaction between cooking oils and food during the cooking process.

Australian Extra Virgin Olive Oil (EVOO) was found to be a fresher product when compared with international varieties. Australian extra virgin olive oil has a reasonably high smoke point which is well above the standard cooking temperatures. The high smoke point of Australian EVOO is associated with the low free fatty acid content of the oil and increased stability. EVOO has a high level of oxidative stability which is attributed to the mono-unsaturated fat content and the high level of phenolic (antioxidant) compounds.

When heated, the phenolic compounds in extra virgin olive oil can reduce. However, there is evidence to show that a significant fraction of phenolic compounds in the oil transfer to the cooked food and this can help with the absorption of some nutrients found in certain vegetables. Australian EVOO has a higher level of natural antioxidants which also have other potential health benefits. In addition, EVOO is free from trans-fats which can be detrimental to cardiovascular health. Contrary to unsubstantiated popular myths, Australian EVOO is an excellent choice for cooking.

Introduction

Fats and oils are not only a source of calories, but they also serve many chemical, physical, and nutritional functions in the body. Some of the most important functions that fats and oils serve in food are:

Appearance and texture: Fats and oils can alter a food's appearance by creating a glossy or moist visual texture, making foods such as salads and vegetables appear more desirable.

Flavour: Fats and oils have the unique ability to absorb and preserve flavours, increasing the palatability of certain foods (such as vegetables and salads).

Nutrition: Fats and oils are the most calorie dense component of food and are also important for delivering fat soluble vitamins such as vitamins A, D, E and K.

Satiety: Fats and oils play an important role in achieving satiety after a meal.

Solubility: While fats and oils are not soluble in water, there are other chemical compounds that are only soluble in fats. Many of these fat-soluble compounds are responsible for the flavour in food and vitamin content.

Heat transfer: Fats and oils provide one of the most efficient modes of heat transfer during cooking[1].

When cooking oils are exposed to high temperatures a variety of chemical changes can occur, such as hydrolysis, oxidation and other thermal reactions[2,3]. Heating oils too high, or for too long, can result in oil decomposition and the production of byproducts, including free fatty acids (FFAs), alcohol, cyclic compounds, dimers and polymers[3]. The chemical reactions that take place are governed by factors such as; the type and quality of oil used in cooking, the properties of the food being cooked, temperature, time of exposure and the food/oil ratio[2].

The end result of the chemical changes that occur through heating oil are twofold, potentially affecting both the organoleptic properties of the oil and the nutritional value[4]. In addition, some of the products formed through oil decomposition may have adverse effects on human health[3].

The total deterioration of oil when frying can be measured by both sensory (i.e. changes in colour, smell and taste) and laboratory values (i.e. measuring FFAs and the formation of total polar compounds (TPC))[3]. Overall, there are two major properties of cooking oils which dictate the behavior of that oil, and subsequent safety when exposed to high cooking temperatures – smoke point and, most importantly, oxidative stability.

When assessing the characteristics of EVOO, it is essential to differentiate between different quality EVOOs which are available in Australia. When compared with international varieties, Australian EVOOs are generally of a higher quality for consumers purchasing the product in Australia[5]. Locally produced Australian products are fresher and reach the consumers much more quickly than international variants[5]. As a result, Australian EVOO has a superior quality for Australian consumers[5].

The smoke point of an oil or fat is the temperature at which, under defined conditions, sufficient volatile compounds emerge from the oil and a bluish smoke becomes clearly visible. At this temperature, volatile compounds, such as water, FFAs, and short-chain degradation products of oxidation evaporate from the oil. The smoke point is the temperature at which the oil is decomposed and where possible toxicologically relevant compounds are formed. The smoke point of an oil generally increases as the FFA content decreases, and the degree of refining increases (see Figure 1)[6].

When cooking foods, they are exposed to a variety of temperatures:

- Pan frying (sauté) on stove top heat – 120°C
- Deep frying – 160 to 180°C
- Oven baking – below 200°C

Australian EVOO has a reasonably high smoke point ranging between 200°C and 215°C, which is well above the standard temperatures required for cooking. This is due to the low FFA content that results from fresh, healthy olive fruit, and care taken when handling and milling the fruit to minimise deterioration (deteriorated fruit delivers oils with higher FFA and thus lower smoke point)[7]. High quality EVOO is able to withstand high cooking temperatures due to the high stability of the oil, and subsequently high smoke point, making it a suitable oil for use for hot and cold cooking via conventional methods including pan frying (sauté), stir frying, deep frying, BBQ's and oven baking.

When heating oils the process of fat oxidation is accelerated. Fat oxidation is where the fat molecules interact with oxygen, leading to the potential formation of harmful compounds. Oxidative stability is the best predictor of the behaviour of oil during cooking[8,9,10] (see Figure 2). EVOO has a high level of oxidative stability and is not likely to undergo oxidation[4]. This is primarily attributed to two factors:

1. Phenolic compounds and α-tocopherol (antioxidants) which are naturally found in EVOO, improve the oil's resistance to oxidative deterioration[11]. These minor components of EVOO not only add to the health profile of the oil, but also improve heat stability, due to a reduction in oxidative processes in heated oil[3].
2. High ratio of mono-unsaturated (MUFA)/polyunsaturated (PUFA) fatty acids – the high level of oleic acid (a MUFA), and low levels of linoleic acid and linolenic acid (PUFAs) in EVOO contributes to the resultant high oxidative stability and resistance to the formation of harmful compounds when heated (such as polar compounds).

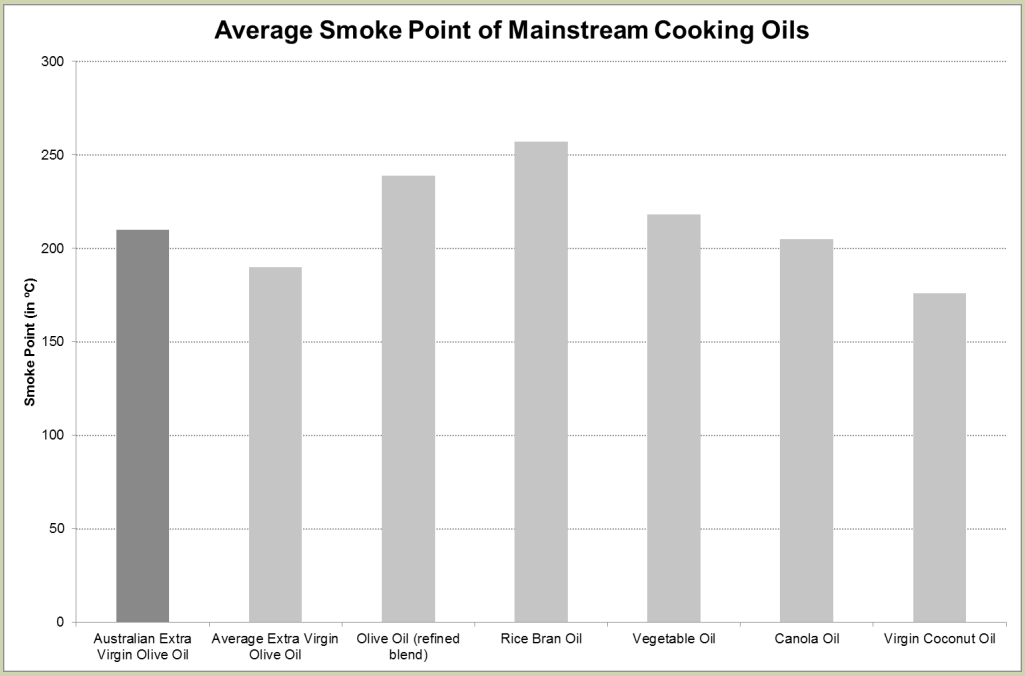

Figure 1 – Average smoke point of mainstream cooking oils based on analysis performed on standard supermarket products by ISO 17025 accredited laboratory

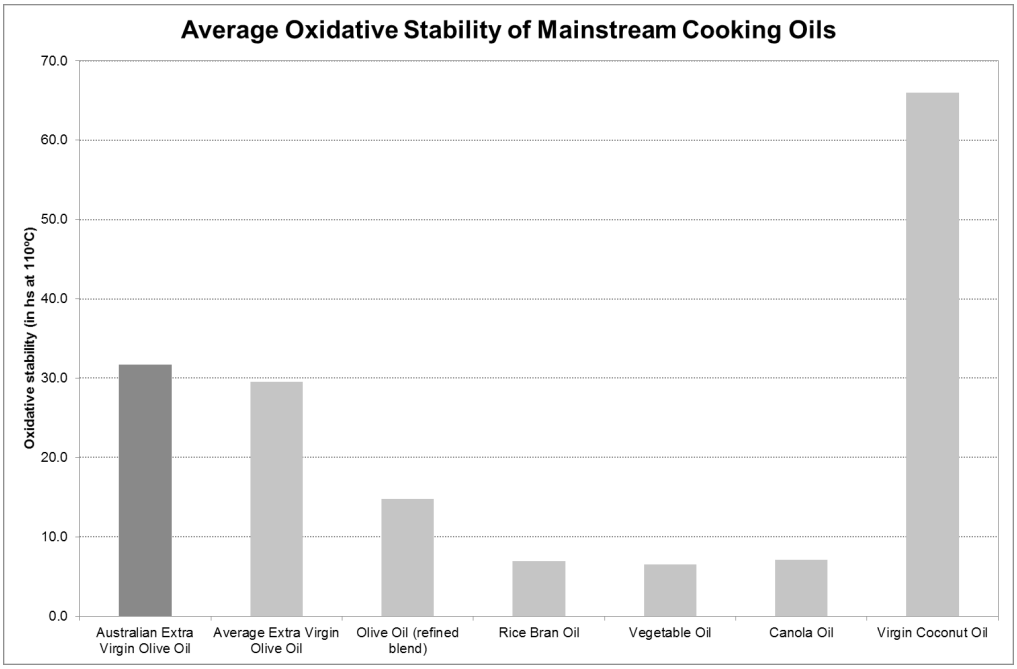

Figure 2 – Average oxidative stability of mainstream cooking oils based on analysis performed on standard supermarket products by ISO 17025 accredited laboratory

The combination of a high content of MUFA (and low PUFA content), together with the antioxidant components found in EVOO, make it highly resistant to oxidation, and therefore there is less chance that harmful substances will be formed upon the application of heat, through cooking. This also means that EVOO will last longer in storage[3,4,10,11].

Cooking with EVOO can improve the nutritional properties of the food. During any cooking process, when oil is the heating medium, two phases typically occur. The first phase is where water evaporates from the food being cooked, and this commences once the oil reaches 100ºC, and continues until most of the food moisture has evaporated (see Figure 3). The second phase is that during which the food absorbs and releases (in the case of fatty foods) fat. This second process determines the changes in fatty acid composition of the food which occurs during the cooking process (see Figure 3).

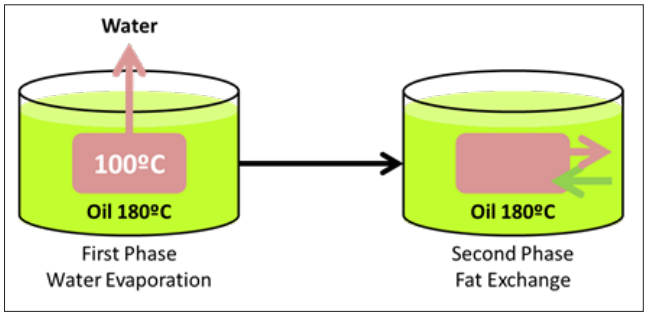

Figure 3 – Typical dynamics during deep frying fatty foods

It has been well documented that the fatty acid profile of the food after deep frying is closer to that of the oil used to fry the food than that of the raw food itself[12,13]. Furthermore, despite the impact that heating has in reducing the antioxidant content in EVOO, a significant amount of polyphenols, tocopherols, sterols and squalene still remain in EVOO after heating, and they are absorbed by the cooked food.

When fatty foods (i.e. meat) are cooked in EVOO there is an increase in the food's MUFA content and a reduction in the saturated fatty acid (SFA) and PUFA content. The resultant cooked food will also have a higher content of powerful bio-active substances such as polyphenols (i.e. hydroxytyrosol and oleuropein), tocopherols, squalene and phytosterols[12,13]. When vegetables are cooked in EVOO there are additional health benefits – carotenoids require dietary fat (i.e. EVOO) for absorption, and glucosinolates (found in broccoli, cauliflower, kale, cabbage, Brussels sprouts) are absorbed better with fat (i.e. EVOO). When food is cooked with EVOO it is healthier than if cooked with other oils that have a different fatty acid profile, or contain lower amounts natural bio-active substances[12,13,14,15].

There are an abundance of cooking oils on the market and it can often become challenging for patients to make an educated choice on using the most appropriate oil. As a result, it is essential that healthcare professionals understand the differences between the most commonly used cooking oils (see Table 1). It is evident that EVOO contains the highest amount of phenolic compounds when compared to other cooking oils, and it also contains a favourable fatty acid profile.

In addition to EVOO containing higher levels of phenolic compounds compared with any other type of cooking oil, it also contains no trans-fats due to its natural production method. Trans-fats have the potential to elevate HDL cholesterol levels, having a detrimental effect on cardiovascular health[16]. The consumption of trans-fats in the diet can increase the risk of atherosclerosis[16].

There has been significant publicity in the media around the health benefits of coconut oil. As can be seen in Table 1, virgin coconut oil has a lower smoke point when compared with EVOO. In addition, virgin coconut oil does not contain any valuable phenolic or antioxidant components. However, like EVOO, virgin coconut oil contains no trans-fats.

	Premium Australian EVOO	Average EVOO	Olive oil (refined blend)	Rice bran oil	Vegetable oil	Canola oil	Virgin coconut oil	Canola oil
MUFAs (%)	72–76	65–78	65–78	40–45	50–60	60–70	4-8	60–70
PUFAs (%)	9–13	9–15	9–15	30–35	30–35	25–30	<1	25–30
Squalene (ppm)	3500–4500	2500–5500	2000–4000	350–450	30–300	100–200	0.0	100–200
Phenolic compounds (ppm)	500–900	170–340	10–150	<1	<1	<5	<1	<5
Trans fats (%)	0.0	0.0	0.1–0.3	0.6–1.9	0.6–1.5	0.6–1.7	0.0	0.6–1.7
Smoke point (°C)	200–215	190	230	210	220	210	170	210

Table 1 – A comparison of the constituents of common cooking oils based on analysis performed on standard supermarket products by ISO 17025 accredited laboratory.

There is a common myth that cooking with EVOO reduces the nutrient value of foods. Applying heat to food in any form of cooking (frying, steaming baking, boiling etc.) will change the properties of the food. The added benefit of cooking with EVOO is that fats are required to absorb carotenoids and glucosinolates (phytonutrients) found in some foods[7]. By cooking foods that contain these elements (such as cabbage, broccoli, cauliflower and kale) in EVOO, a high percentage of the phytonutrients will be absorbed, and this is more nutritious that with steaming or boiling the vegetables. Furthermore, EVOO's oleic acid, sterols and squalene exhibit a high stability against oxidation. Although not as stable under heat, a large amount of polyphenols and tocopherols remain in EVOO after cooking. Therefore, despite the heating conditions during cooking, EVOO maintains most of its minor compounds and, therefore, most of its nutritional properties[12,13,14,15,16].

Another common myth is that heating EVOO converts the mono-unsaturated fats to tran- fats. It is important to understand that trans-fats form when any edible oil is subjected to an industrial process such as refining or hydrogenation (which is designed to turn liquid oil into an edible fat that is solid at room temperature (i.e. margarine))[18,19]. The vast majority of trans-fats in the average person's diet arise from fast food, inexpensive margarines, or more commonly commercially baked products[18,19] EVOO is naturally trans-fat free and cooking with EVOO will ensure that undesirable trans-fats are not added to the diet.

Contrary to popular myths, high quality EVOO is an excellent choice for cooking. High quality EVOO has a smoke point well above the standard temperatures required for cooking, and its resistance to oxidation is higher than most cooking oils due to the antioxidant and mono-unsaturated fat content. Therefore, it is ideal for both hot and cold cooking and there is less chance that harmful substances will be formed upon the application of heat through cooking. Furthermore, although the heating process will reduce the natural antioxidant content of EVOO there is scientific evidence to demonstrate that a substantial amount of antioxidants can still be found in the prepared meal[14,15].

Conflict of interest: The author is a consultant for Cobram Estate Extra Virgin Olive Oil.

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A Review of the Medical Board of Australia Code of Conduct for Good Medical Practice

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Background

The aim of this discussion is to consider and refresh your understand of the Good Medical Practice (the Code) as developed by the Medical Board of Australia. The Code outlines principles that characterise good medical practice and which makes explicit the standards of ethical and professional conduct expected of doctors by their professional peers and the community. The Code's application will vary according to individual circumstances, but the principles [as set out in the Code] should not be compromised[1].

Where professional conduct varies significantly from the Code, explanation and justification for decisions and actions will be required. Serious or repeated failure to meet the standards will have consequences for ongoing medical registration[1].

This Code adds to the 2006 Australian Medical Association Code of Ethics and is consistent with the Declaration of Geneva and the International Code of Medical Ethics, issued by the World Medical Association.

This Code is not a substitute for the provisions of legislation and case law and where a conflict arises between the Code and the law, the law takes precedence.

About the patient

A. Patient Centred Care

While doctors have their own personal beliefs and values, they are expected to base their practice on professional values such as:

i. a duty to make the care of patients their first concern;
ii. to practise medicine safely and effectively by minimising risk to patients and maximising their record keeping skills, reporting obligations and compliance with insurance requirements as well as being honest and transparent in financial dealings with patients;
iii. maintaining good health and wellbeing, as well as being aware of that of your colleagues;
iv. being ethical and trustworthy;
v. protecting their patient's confidentiality;
vi. understanding each patient is unique;
vii. working in partnership with patients;
viii. being culturally aware.

Patients trust their doctors because they believe that, in addition to being competent, they will not take advantage of them and will display qualities such as integrity, truthfulness, dependability and compassion.

B. How to provide the standard of care expected?

The Code states providing good patient care is a primary concern of clinical practice and entails patient assessment (history and physical examination etc) often resulting in the formulation and implementation of clinical management plans. The Code explicitly re-enforces patients' rights (autonomy) to make their own decisions.

The provision of care under the Code expects doctors to maintain a high level of medical competence and professional conduct involving working within limits of their own competency, ensuring adequate knowledge and skills are maintained. In so doing, the Code requires a balancing of benefit and harm in all clinical-management decisions where treatment options are based on the best available information. Patients are encouraged to take

an interest in, and responsibility for, the management of their health, whilst ensuring a doctor's personal views do not adversely affect the care of the patient.

The Code reinforces healthcare decision-making as a shared responsibility of the doctor and the patient, the ultimate decision of which is decided by the patient based upon best available information[1]. Priority to investigating and treating patients on the basis of clinical need and effectiveness of the proposed investigations or treatment is required.

As a requirement of registration, continuing professional development is mandatory and aims to ensure good medical practice expected by the public and peers is achieved. To ensure the maintenance of effective communication between patient and doctor, patients are encouraged to discuss alternative or complementary therapies and other health management approaches in use with the doctor. Patient must also be informed about all aspects of their healthcare management giving them adequate opportunity to question or refuse intervention and treatment including their potential benefit and harm and material risks associated.

Good record keeping must record these and related discussions, investigations and treatments[(1)].

C. Informed consent

The Code enshrines informed consent as the patient's right of voluntary decision-making about their medical care. The patient's decision must be made with knowledge and understanding of the benefits and risks involved. The type of information necessary for the doctors to provide patients is detailed in the National Health and Medical Research Council (NHMRC) guidelines[(2)]. Good medical practice [essentially] involves providing information to patients in a way that they can understand before their consent (or not) for the treatment is obtained.

D. Working in teams

To enhance patient care doctors are encouraged to have good relationships with medical colleagues, nurses and other health care professionals based upon mutual respect and clear communication which acknowledges and respects the contribution of all health care professionals involved in the care of the patient. With this approach applied, the Code anticipates improved patient care. However, working in a team does not alter a doctor's personal accountability for professional conduct and the care provided.

E. Children, young people, patients with additional needs and end of life care

Caring for children and young people brings additional responsibilities. A doctor's primary concern is for the interests and wellbeing of the child whose decision-making capacity and consent should be considered through meaningful communication and education. Doctors should recognise the parent's role through encouragement, promotion of the child's care and decisions-making role. Doctors must be mindful of children who may be at risk and notification to the appropriate authorities as required by law must be accomplished[(3)].

Some patients (including those with impaired decision-making capacity) have additional needs. These patients may be at greater risk than ordinary patients and their care may involve a range of people at different times. Particular attention to communication, the need for third party consent and increased advocacy will assist just access to health care is obtained. Aspects of care will incorporate consideration of relatives, carers, partners and others close to the patient.

Knowing patients have the right to refuse medical treatment or to request withdrawal of treatment already started, doctors can facilitate end of life planning with advance care plans. Where end of life issues arise patient management to prolong life (which is not a doctor's duty) may not benefit the patient but may involve provision of palliative care and symptom management consistent with the patient's values and wishes. Doctors have a duty to know when not to initiate and when to cease attempts at prolonging life, while ensuring patients receive appropriate relief from distress. Where possible all support must be provided to patients and families, for example, communicating bad news thoughtfully, explaining the circumstances of death unless otherwise requested not to by patients[(3)].

F. Ending patient/doctor relationships

To end the doctor/patient relationship, a doctor should ensure patients are adequately informed of any decision and in so doing, the doctor should facilitate arrangements for ongoing patient care including referral to another practitioner.

Where a practice closes or relocates, where possible, patients should be provided advance notice, arranging for ongoing medical care with another practitioner or the transferring of or appropriate management of patient records[(3)].

G. Emergencies

In case of emergencies doctors should, in addition to the patient's best care, consider a range of issues, such as, the doctor's own safety, skill set, whether other options are available, the impact on other patients under care and providing assistance until care is not required.

H. Advertising

Medical service advertising, whilst useful in providing patient information, must conform to relevant consumer protection legislation, the advertising provisions in the National Law and Advertising Guidelines. In summary, all advertised information

must be factual, verifiable and based on justifiable claims with no promises of guarantee cures, exploiting patients' vulnerability or fears, or raising unrealistic expectations[3].

I. Complaints

Patients who are dissatisfied have a right to complain about their care. Doctors should:

ix. acknowledge the issue and work with the patient to resolve it by providing prompt, open, constructive responses, explanations and, if appropriate an apology;

x. where appropriate, refer the patient to another practitioner;

xi. comply with relevant complaints legislation.

About public health

The Code clearly outlines a doctor's public policy duty in four distinct but related aspects. Firstly, a duty is owed to each patient by way of a healthcare entitlement with a corresponding duty on doctors to ensure that that right is delivered in a consistent, judicious effective, efficient and fair manner to ensure services provided are both necessary and likely to benefit the patient so that access to the appropriate level of care is optimised for all patients. The GM Code next engenders a transparent and equitable allocation of health care resources since patient management can affect other patients' access to similar health care resources.

The Code goes beyond the immediacy of the doctor-patient relationship by outlining the significant disparities in the health status of different groups within the Australian community. It links good medical practice with a doctor's expertise and influence on the local scene to protect and advance the health and wellbeing of individual patients, communities and populations.

The Code finally details a doctor's responsibility to promote community health through disease prevention and control by applying the principles of public health, including health education, health promotion, disease prevention and control and screening, as well as participating in efforts to promote the health of the community.

Teaching, supervising, assessment and research

As part of good medical practice, activities which provide and contribute to the support, assessment, feedback and supervision for colleagues, doctors in training and students, are good for patient care because accurate, constructive and justifiable information should be provided. All communications should be given in a respectful, patient manner.

Research in Australia is governed by The National Statement on Ethical Conduct in Human Research (2007) [National Statement (2007)] which is a series of guidelines subject to rolling reviews made in accordance with the National Health and Medical Research Council Act 1992. All doctors considering entering research or conducting research whilst in practice should review, consider and comply with the National Statement[4].

Conclusion

The Good Medical Practice Code clearly and plainly articulates the standard to which a doctor must practice. As explained, deviation from these principles and those practiced by peers may result in the ultimate sanction of deregistration. The Code provides all doctors with sound principals against which good medical care will be measured.

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Integrative management of inflammatory bowel disease: a case report

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Abstract

Inflammatory bowel disease (IBD) is a chronic relapsing disease of the gastrointestinal tract with extra-intestinal disease present in up to 40% of patients. Several interacting aetiological factors are highlighted including genetic variability in intestinal barrier function, the role of intestinal microflora, food allergy and the potential for infective agents to exacerbate ulcerative colitis (UC). This case study discusses a patient with UC who was advised to have a colectomy due to persistent symptomatic disease whilst on maximal immunosuppressive therapy. He has demonstrated a positive response to dietary eliminations, probiotics and treatment for a suspected intestinal parasite resulting in significant reduction in symptoms and medication highlighting the therapeutic potential of an integrative response to IBD.

Key words: Inflammatory bowel disease; ulcerative colitis; diet; probiotics; Strongyloides; integrative medicine

Introduction

Inflammatory bowel disease (IBD) is a group of relapsing inflammatory disorders of the gastrointestinal tract that primarily comprises ulcerative colitis (UC) and Crohn's disease (CD). IBD is acknowledged to have a complex multi-factorial aetiology that involves an interplay of factors associated with the immune system, the environment, enteric commensal microbiota and host genotype[1,2]. Dysfunction in intestinal epithelial barrier function appears to play a role in both UC and CD. UC is associated with a mutation of the Toll-like receptor (TLR)-4 gene resulting in impaired lipopolysaccharide signaling and induction of inflammatory responses[3]. A number of other polymorphisms have been identified involving various genetic loci[2] all involving either immune reactivity or early response to bacteria with individuals genetically predisposed to IBD likely to have dysregulated responses to microbial challenges mediated by the innate immune system[2-4].

There is a potential role for probiotics in the treatment of IBD, especially in UC where beneficial bacteria have been shown to have a protective action on the intestinal epithelium[3,5]. Diet also has the potential to influence IBD through a number of mechanisms including food allergy and intolerance exacerbating intestinal inflammation and symptoms but also by altering the composition of the microbiota[6]. In addition, there have been a number of reports documenting cases of infection with the helminth, Strongyloides stercolaris mimicking ulcerative colitis with potentially lethal outcomes[7-9].

Case Presentation

A 38 year old human relations consultant who was an existing patient at our practice presented in August 2013. His medical history included a diagnosis of colitis based on colonoscopy April 2006 following several years of intermittent diarrhea and PR bleeding. He was initially treated with Mesalazine. His diagnosis of ulcerative colitis was confirmed in 2012 after a repeat colonoscopy reported a pancolitis following a flare up. He was found to be quantiFERON positive and was treated for TB prior to commencing infliximab infusions. His history included living and working as a volunteer in India in remote locations, where TB is endemic, from 1999-2004. Whilst in India he also had confirmed infections with Hepatitis A and Malaria. On return from India he spent 8 weeks working in a remote Aboriginal community in the Gascoyne, WA.

At the time of his presentation in 2013 he had been hosptialised with an exacerbation of ulcerative colitis. His medications included Mercaptopurine Mesalazine and Infliximab infusions. He was also taking a short course of oral Prednisalone. He reported up to 15 bloody bowel actions a day and his treating gastroenterologist recommended a total colectomy. He had been vegetarian for a number of years but admitted that his diet was not a healthy vegetarian diet and included high dairy, sugar

and processed foods. His chiropractor had performed a stool analysis test that included a DNA microbial analysis, digestive and inflammatory markers. The test was positive for Strongyloides DNA however faeces microscopy and culture and subsequent serological antibody testing were negative.

On examination he was afebrile and his abdomen was distended with increased bowel sounds and no masses or focal tenderness. Investigations showed normal vitamin D 34 (75-250), haemoglobin 128 (130-180), ferritin 62 (30-500), transferrin saturation 0.10 (0.20-0.50) and iron 8 (9-30). Strongyloides serology was negative and faecal cultures were negative for parasites. Liver function tests, coeliac serology, B12, RBC folate, plasma zinc, TSH and CRP were all normal. Bloodspot IgG/IgA vegetarian, herb and spice panels (US Biotek) showed high reactivity to dairy, sugar, coffee, and moderate to gluten grains, almond, spirulina, valerian and licorice. These results need to be interpreted in the context of immunosuppression. GI Effects Stool Analysis (Genova Diagnostics) showed adequate beneficial flora, Strongyloides DNA positive, elevated lactoferrin and low elastase.

He was commenced on a diet low in processed food with elimination of dairy, sugar, reactive grains and other reactive foods as indicated by testing. He received nutritional supplementation with D3Forte drops 3000IU(3 drops) twice daily, glutamine and intestinal nutritional support complex one teaspoon twice daily, digestive enzymes three times daily with meals and phosphatidyl choline 420mg twice daily.

He was also prescribed a number of supplements by a naturopath and these included Ultraflora Immune, a probiotic (l.acidophilis, b. lactis, l.rhamnosus), plant omega EPA/DHA and vegan vitamin B12. He self-administered Men's Ultivite 1 daily. He was advised to continue the current medications. After discussion with his gastroenterologist he was treated for possible Strongyloides infection with a limited course of Ivermectin.

By November 2013 he reported feeling well with reduced stool frequency, 2-3 bowel actions per day and less bleeding. A repeat stool specimen was sent in December 2013 however the test method used by the laboratory had changed and they were no longer using DNA analysis. Microscopy and culture were negative for parasites including Strongyloides. Over the following 12 months he continued to improve and started reducing his oral medication and ceased medication June 2014. There was a brief relapse after a period of dietary lapse which responded to a brief course of oral prednisolone. He continued to receive Infliximab infusions. By Februrary 2015 he reported a formed stool twice a day with occasional blood.

Discussion

Current conventional treatment for inflammatory bowel disease is based on medical and surgical interventions. These treatments are expensive, associated with potential severe adverse effects, either as a direct effect of the drug, or from the immunosuppression that most of them rely on for their action, which often fail to completely control the disease process[1].

This case highlights the importance of and integrative approach to inflammatory bowel disease as there are likely to be multiple contributing factors specific to each individual. Each patient will require a unique treatment approach which may involve medication, diet, nutritional therapies and lifestyle changes. It also highlights the limitations of treatment based on evidence from single intervention trials which cannot adequately assess the outcomes of multiple interventions required for effective individual treatment. Whilst there is a growing database of good quality evidence for holistic approaches there are still limitations in funding for non-drug treatments and in trial design for some of these interventions. Lack of evidence is not necessarily associated with lack of patient benefit[10].

Hou et al review a number of specific diets including carbohydrate restriction, low FODMAP and Paleo diet in the treatment of IBD[6]. They concluded that, whilst existing data does not support the recommendation of any specific diet, anecdotally, patients report positive response to some of these diets. They also note that patients demonstrate the ability to identify specific foods that exacerbate their symptoms and that these foods are unique to different individuals. Conventional allergists test mainly for IgE reaction to foods with either skin prick testing or blood RAST. The gold standard for an adverse food reaction is via elimination and later provocation. This can be a very time consuming process. Delayed hypersensitivity reaction to foods is mediated by IgG and IgA antibiodies and can present with various systemic symptoms and signs which may be difficult to distinguish from other chronic diseases and conditions. These reactions are caused by antigen-antibody complex deposition in different tissues[11].

ELISA IgG testing has been demonstrated to be elevated in IBD patients compared with controls[12]. Detection of IgG antibodies and elimination of identified foods has also been shown to be beneficial in IBS[13,14]. IgG food antibody testing may be a useful tool to identify foods exacerbating inflammation in IBD. Testing for IgA antibodies has the potential to detect a further 30-40% of food allergy that tested negative for IgG and IgE antibodies (US Biotek laboratory manual). IgA antibodies are produced by intestinal mucosal cells as first line of defense in the gastrointestinal tract and may also be significant in intestinal inflammation in response to certain foods.

Although the exact aetiology is not clear, the generally accepted hypothesis suggests that IBD arises from loss of oral tolerance to commensal microbiota resulting in chronic intestinal inflammation in genetically predisposed hosts. The use of probiotics enables manipulation of intestinal microbiota composition, the immune system and host barrier function[1].

Probiotics are "live microorganisms that, when administered in adequate amounts, confer a health benefit on the host"[15]. Probiotics may be beneficial in IBD by several mechanisms. They affect the composition of the microbial ecosystem by competition for nutrients and adhesion sites, by the production of antimicrobial substances. They affect the host immune system by interaction with gut-associated immune cells, contribute to the production of short chain fatty acids, induce mucin secretion and enhance tight-junction expression and function[1].

A number of studies have found different bacterial numbers in active IBD compared with inactive and also different composition and less diversity comparing inflamed with non-inflamed mucosal samples within patients[1]. Meijer and Dieleman conclude that the rational to use probiotics is supported by research demonstrating the involvement of microbiota and their influence on host responses in both rodent and human IBD models[5]. Probiotics have been found to be beneficial for ulcerative colitis with evidence for the use of E. coli Nissle 1917 in maintenance of remission in UC and for VSL#3, a high potency multi-strain probiotic, in maintenance of remission in pouchitis and induction of remission in UC. Probiotics have not been shown to have a benefit for treatment of CD.

An interesting feature of this case is the possibility of an intestinal parasite,

Strongyloides stercoralis, as either the causative agent or a significant trigger in a susceptible individual. There are a number of documented cases in the literature of Strongyloides mimicking ulcerative colitis[9,16,17]. Diagnosis is often problematic with stool examination negative in 50-70% of patients. It should be considered as a differential diagnosis in any patient with features of colitis who has travelled to an endemic area as disseminated strongyloidiasis is a complication of immunosuppressive treatment which is the mainstay of treatment for IBD. This patient had limited antimicrobial treatment to cover the possibility of infection with this parasite.

Conclusion

This case illustrates the importance of an integrative approach to patients with IBD. There are likely to be multiple individual factors including genetic, immunological, dietary and possibly infective contributing to intestinal inflammation and all need to be considered and addressed to achieve the best outcome for the patient.

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The 5th Science of Nutrition in Medicine and Healthcare Conference

Commentary

A/Prof Alison Coates and Dr Jim Parker

The 2015 Science of Nutrition in Medicine and Healthcare Conference in Melbourne in May was declared a great success by all who attended. A total of 408 delegates attended representing a wide range of professional interests. The opening session set the scene for the high quality plenary sessions, concurrent workshops and scientific abstract presentations that followed.

Award-winning journalist Jayne Azzopardi did an outstanding job as Master of Ceremonies and Facilitator of the Conference. The ACNEM President A/Prof Eugen Molodysky gave a brief introductory presentation to remind the audience of the importance of Nutritional and Environmental Medicine in the primary prevention of many of the current chronic diseases confronting the health system. His message regarding the importance of implementing preventative health care in general practice to help reduce health care expenditure was well received by the Federal Health Minister, who was in attendance to open the conference.

The Hon Sussan Ley MP took the comments on-board and expressed her enthusiasm for learning more about the role of Nutritional and Environmental Medicine and how to implement preventative strategies into primary care. The Minister clearly showed her knowledge of the many facets of her portfolio and told some interesting personal stories of her own life experiences. Her presentation was followed by entertaining Barrister Charles Waterstreet (alias Rake) who gave a humorous, and unfortunately honest, account of his previous high risk diet, alcohol, drug, smoking and sexual behaviour. Fortunately, he was guided to improved health by the ACNEM President and provided numerous personal examples of where primary and secondary interventions could lead to improved health outcomes.

The Gastrointestinal, Mental Health and Heart, Diabetes and Weight concurrent sessions included a wide variety of topics and speakers. The diversity of topics successfully met the wide range of interests of practitioners from all disciplines who attended the Conference. The successful juxtaposition of complementary, mainstream and scientific researchers was a significant achievement of the conference organisers and demonstrates the narrowing gap between different health-care providers. The Conference clearly showed the emerging importance of the role of the gastrointestinal microbiome which featured in almost half of the presentations.

The Scientific Abstract sessions went smoothly with excellent presentations across a wide variety of topics in both oral and poster formats. Presenters were a mixture of health professionals, academics and postgraduate students. Prizes were awarded to postgraduate students for the best oral presentation (awarded to Ms Emma Beckett for her presentation on 'Bitter taste phenotype influences total energy intake but not composition') and the best poster presentation (awarded to Mrs Charlotte Martin for her presentation on 'Dietary vitamin B12 intake and cognitive decline in an Australian population').

The Gala Conference Dinner was well attended and included a delicious buffet of nutritious foods. The dinner speaker was acclaimed actor and director Damon Gameau, who spoke about the development of his documentary "That Sugar Film". This was followed by a great band who had the crowd on their feet to increase their energy expenditure.

The Conference organising committee, particularly Dr Braham Rabinov and Jimena Acevedo, are to be congratulated on putting together such a high quality and interesting Scientific Meeting. Planning is already underway for The 6th Science of Nutrition in Medicine and Healthcare Conference in Sydney, 30 April – 1 May 2016.

L-R, A/Prof Eugen Molodysky; Hon Sussan Ley, MP; Charles Waterstreet; Jayne Azzopardi

Best Abstract Presenter

Emma Beckett, BBiomed(Hons), GDipClinEpi, MScMgt

I was fortunate enough to present some of my PhD work at the ACNEM Nutrition in Medicine Conference held in Melbourne in May 2015. I am researching the relationship between taste, diet and disease risk. The primary aim of the study was to determine if bitter taste phenotype (how sensitive a person is to bitter flavours) influenced total energy intake or intake of specific macronutrients. Bitter tasting status is often regarded as a marker for taste acuity in general. My research question is do people who have higher sensitivity to bitter taste eat more or less food?

We can test a participant's bitter taste phenotype easily with a series of bitter taste solutions (containing a bitter chemical called 6-n-propylthiouraciil). 'Tasters' are averse to the taste of this solution, while 'non-tasters' are unaffected. In our cohort of 252 people, 25% were 'non-tasters', with similar numbers of males and females in each group. Total energy and macronutrient intake are estimated using food frequency questionnaires, which document which foods people eat and how often. All analyses were adjusted for age and gender.

We found that 'non-tasters' consumed more total energy, approximately 800 kilojoules more per day. However, the relative contribution of macronutrients (fat, carbohydrates, protein) and fibre and alcohol did not vary between groups. Interestingly, if non-drinkers were excluded (as people may choose to avoid alcohol for many reasons other than taste) non-tasters consumed much more alcohol. Eating more kilojoules and drinking more alcohol may mean that non-tasters are at greater risk of diseases linked to poor diet.

The research was conducted in a cohort of patients having a colonoscopy and had a secondary aim of seeing if taster status influenced risk of occurrence of adenomatous colorectal polyps, the precursor to colorectal cancer. Colorectal cancer is a disease with well-established dietary risk factors. No difference in risk was seen between 'tasters' and 'non-tasters', however, this may be because the study was underpowered to detect an outcome, as only 24% of participants had polyps. A larger cohort and consideration of additional confounding factors in future studies may be required to demonstrate a relationship.

As the winner of the Best Abstract Presenter prize, I was given the opportunity to repeat my presentation in the final plenary session of the Conference. As I am still in the very early stages of my career this was the largest audience I had ever presented to. I was initially very nervous, but soon settled into it. The audience appeared enthusiastic and supportive and I am very grateful for the experience. I thoroughly enjoyed all the scientific abstracts presented and hope to see more people in the session next year.

I gratefully acknowledge the contribution of my supervisors, A/Prof Mark Lucock, A/Prof Martin Veysey, Dr Zoe Yates and Dr Konsta Duesing, as well as my co-authors Lyndell Boyd and Xiaowei Ng. My PhD is funded by the CSIRO (CSIRO OCE scholarship) and my attendance at the conference was funded by an ACNEM Conference scholarship.

Best Poster Presenter

Charlotte Martin won Best Poster Presenter at the recent Science of Nutrition in Medicine Conference for her poster titled 'Dietary Vitamin B12 Intake And Cognitive Decline In An Australian Population'

Charlotte is a PhD candidate at the University of Newcastle.

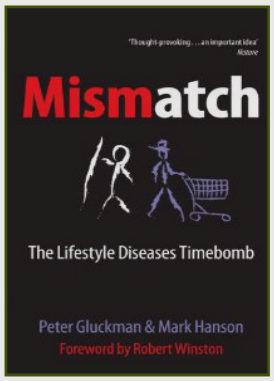
Prof John Funder

Prof Mimi Tang

Damon Gameau

Book Review

Mismatch - The Lifestyle Diseases Timebomb
by Peter Gluckman and Mark Hanson



This very interesting book by two eminent authors working in New Zealand and England should appeal to many ACNEM members. It seeks to explain how our genetic make-up is finding it difficult to adapt to the rapid changes in diet, lifestyle and environment. The authors hypothesise that disease arises as a result of an individual being "... faced with environmental conditions that generate a challenge which goes beyond his or her ability to adapt fully." ie., people are susceptible to disease because they are out of their comfort zone, for example, in times of stress. This perspective is radically different from the classical view of disease.

The book covers all aspects of human life from pregnancy to old age. It explains how developmental plasticity enabled humans to evolve to adapt to different environments on the journey out of Africa. However, the speed of the industrial and IT revolutions is taking its toll on modern man.

Many interesting examples are given to support the authors' arguments of mismatch between genes and diet, genes and environment relating to such conditions as metabolic syndrome, heart disease, and diabetes. There are numerous references to original papers and an extensive index. It is a wide ranging book which should appeal to the lay reader as well as those working in the health sector. My one criticism is that the paperback version published in 2008 is set in fairly small type, just another example of mismatch between my visual acuity and the printed word, or maybe it is just that the batteries in my eyes are running down?

Reviewed by Dennis Crowley, BSc(Hons).

This book is published by Oxford University Press.

ISBN: 9780199228386

In The News

Kelly Stuart, BHSc (Nutritional Medicine)

Dietary fat stance reversed: new guidelines

The Committee that advises the US Government on national dietary guidelines (Dietary Guidelines Advisory Committee (DGAC), 15 June 2015) has removed restrictions on total fat consumption from its latest report and does not list it as a nutrient of concern[1]. The report concluded that reducing total fat and replacing saturated fat with carbohydrates does not minimise the risk of cardiovascular disease[2].

Based on this report, a Viewpoint article published by Journal of American Medical Association (JAMA) urges the US Government to promote the consumption of healthful fats and to remove limits on total fat consumption in their 2015 Dietary Guidelines[2]. The authors of the paper state that there is ample evidence which confirm that diets higher in healthful fats (replacing carbohydrate or protein), and exceeding the current 35% fat limit, reduce the risk of cardiovascular disease.

If these recommendations become part of the 2015 Dietary Guidelines for Americans, nearly four decades of nutrition policy will be reversed. When the recommendations for limiting total fat intake were introduced in 1980, the primary rationale was to lower cardiovascular risk by decreasing low-density lipoprotein cholesterol blood concentrations[2]. As nutrition science has advanced we now have evidence of the beneficial effects certain fats have on high-density lipoprotein cholesterol and that the consumption of a diet high in processed carbohydrates is a risk factor for metabolic dysfunction, obesity and cardiovascular disease[2]. The DGAC report did not recommend limiting total fat for obesity prevention; the focus was on promoting diet patterns that include more vegetables and fruit, seafood, legumes, wholegrains and dairy products, and include less meats, refined grains and sweetened drinks.

Middle aged women engaging in high risk drinking

Research by the University of Western Sydney shows that more than 500,000 middle aged women in Australia engage in high risk drinking. The lead researcher, Dr Janice Withnall, says that the number of women who are dependent on alcohol has risen from five percent in 1990 to 18% today[3]. Anxiety and feelings of being unable to cope with the demands of parenting, work and partners are listed as reasons why so many women are using alcohol as a first resort for managing emotional distress.

Dr Withnall comments that it is somewhat of a shock to most people to realise that more than two drinks a day for women is classified as high risk drinking and that women become dependent four times more quickly than men, becoming reliant on alcohol after three years as opposed to twelve for men[3]. Women drinkers are at higher risk than men as they have lower levels of the metabolising enzyme alcohol dehydrogenase which helps detoxify alcohol. Liver disease commences earlier for women than men and alcohol is also a significant risk factor for many types of cancer[4].

These alarming figures mirror those from a recent report in the UK from the Organisation for Economic Co-operation and Development, which states that women aged 45-64 are now drinking the most – and that women with a higher education are twice as likely to become alcohol dependent[5].

Smart Phone Alcohol tracker App

A new smartphone app alerts the user when he/she has exceeded recommended daily or weekly units of alcohol[6]. The developers of this app, The Alcohol Tracker, have published a commentary in the BMJ Innovations noting that their app is unique from other such apps. Individuals are asked to log absolute number of drinks and the app converts this into units of alcohol, rather than relying on blood alcohol readings as do most other current apps, and which have been shown to be inaccurate[7].

There will be immediate notification if users have exceeded the limits for the day or for the week. The notifications are pre-programmed based on the recommended number of units for men and women in accordance to the NICE (UK) and the CANMAT (Canada) guidelines. The Alcohol Tracker also includes links to alcohol helplines, has built in psychological therapies, such as a behavioural goals toolkit and a questionnaire that users can take to determine if their drinking patterns and behaviour put them at risk[7].

Semi-starvation diets to treat obesity

Severe diets don't necessarily lead to binge eating, as has been a common perception, and could be used to treat obesity, according to new research from the University of Sydney's Charles Perkins

Centre and the University of Western Sydney8.

The research involved a systematic review of fifteen clinical trials that measured binge eating before and after semi-starvation diets in people with obesity, and people who did and did not have problems with binge eating before the diet. The studies under review used nutritionally balanced shakes to replace meals and snacks. Diets that deliver less than 3300 kilojoules per day are termed very low energy diets, restricting energy intake to between 30 and 50 percent of total recommended intake[8].

The lead author of the study, Professor Amada Salis, says that most studies showed that for people who were obese and had problem binge eating behaviour before the extreme diet, their binge eating declined during and after their diet. "When clinically supervised, low or very low energy diets are an important treatment option for obesity. The results from this study show that these diets are not necessarily a trigger for binge eating, although eating behaviour should be monitored during their use," Associate Professor Salis said.

It was noted that although 'semi-starvation' diets sound as if they would be difficult, most people on the studies found them easy to adhere to, with many people saying this was because all their food choices were taken away. Sixty per cent of people who undertake the extreme weight loss diets maintained their weight loss after 12 months[8].

Associate Professor Salis emphasises that severe energy restriction diets are only recommended for people with a body mass index in the obese range (30 and over), or those with a body mass index of 27 or more who have risk factors for cardiovascular disease. She also warns that the long-term consequence of extreme diets are unknown as all studies, with the exception of one, followed participants for less than eighteen months.

Extreme diets must be undertaken with medical supervision and are often used as a last resort to avoid bariatric surgery or for individuals at risk of Type 2 diabetes and heart disease.

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The Nutritional Medicine Management & Detoxification of Lipophilic Heavy Metals

Dr Bradley McEwen PhD

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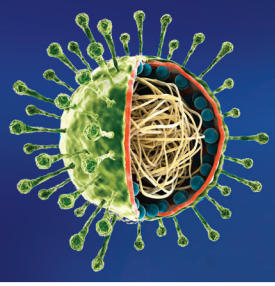
The Relationship Between Exposure to Epstein Barr Virus & Hair Tissue Mineral Analysis

A focus on EBV, the liver and copper.

Gary Moller



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DR BRADLEY MCEWEN PHD

Dr Bradley McEwen PhD is a naturopath, nutritionist, herbalist, lecturer and researcher with over 16 years clinical experience. He has lectured in nutritional medicine and the health sciences for 11 years and presents seminars and at conferences both nationally and internationally. He has numerous original research and review articles published in peer-reviewed journals. He has a passion for teaching and research.

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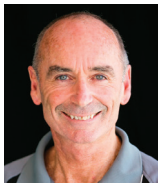
- Implications of EBV/glandular fever for exercise and competition, including coping strategies, especially as it relates to young people, such as competitive swimmers

GARY MOLLER



Like most natural health practitioners today, Gary is a "Health Sleuth", helping people understand and resolve complex and chronic health issues. His principal "CSI" tool is the InterClinical Laboratories HTMA, which Gary regularly incorporates into his clinical practice. Gary is deeply engrossed in preparations to enter a doctoral programme in nutrition to research using the HTMA to help uncover EBV's subtle and lasting consequences for health.

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THE 2016 INTERNATIONAL CONGRESS ON

NATURALMEDICINE

THE MOST SIGNIFICANT ANNUAL EVENT HELD IN THE NATURAL MEDICINE INDUSTRY

Saturday 11th - Monday 13th June | Sofitel Brisbane Central

INTERNATIONAL CONGRESS ON NATURAL MEDICINE 2016

Metagenics
Genetic Potential Through Nutrition

CONGRESS IS ALWAYS A SELL OUT - DON'T MISS YOUR SEAT IN BRISBANE

Your Invitation

The Metagenics International Congress on Natural Medicine has earned the privilege of being recognised as the most significant educational event in our industry. We are pleased to invite you to join us in Brisbane in 2016. This gives us the chance to come together as an industry with like-minded people for an enthusiastic and insightful long-weekend.

The Event – Neurology and Psychiatry

Metagenics International Congress on Natural Medicine for 2016 will address one of the most important areas of health care – neurology and psychiatry. Although Descartes philosophy split the mind from the body in the 1600s, we will be reuniting matter and function and exploring the impact of neural physiology on mood, learning and cognition. Functional psychiatry has taken substantial steps forward over the last few years with increasing understanding of the role of oxidative stress, inflammation, mitochondrial function and the blood brain barrier on conditions once thought to be purely psychological. We will also explore the role of the milieu that the brain finds itself in on neurological function, including nutrition, gut function, systemic infection and toxicity.

Don't miss the most significant event on the Natural Medicine calendar in 2016.

Registration Includes:

- Full Congress manual
- Congress gift pack
- Morning tea and refreshments upon arrival
- Healthy and delicious full buffet lunch
- Afternoon tea
- Coffee, tea and purified water
- A light dinner on Saturday night

Early Bird price of **$695.00** incl. GST
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(Same price applies in New Zealand).

Early Bird ends **Friday 29th January 2016**.

For full details of Congress go to **metagenics.com.au**

Call **1800 777 648** to book today.