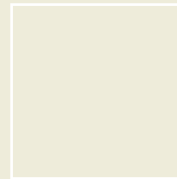


THE JOURNAL OF THE AUSTRALASIAN COLLEGE OF  
NUTRITIONAL AND ENVIRONMENTAL MEDICINE



**JUST BE NORMAL!  
(SURVIVING MEDICARE)**

**A NEW STRAIN OF 'SWINE'  
INFLUENZA TYPE A H1N1 OR  
A CHANGE IN SURVEILLANCE?**

**PUTTING STATINS IN PERSPECTIVE**

**CASE STUDIES – COELIAC DISEASE  
– ADRENAL FATIGUE**





## COLLEGE PROFILE

The Australasian College of Nutritional and Environmental Medicine (ACNEM) is a not-for-profit medical college established in 1982, offering postgraduate training for health professionals in nutritional and environmental medicine (NEM), representation and networking for members, and a popular referral service for members of the public looking for doctors with training and experience in NEM. ACNEM also holds free public lectures for health professionals and the general public at which experts speak on various aspects of NEM.

Full Membership of the college is open to registered medical doctors and dentists, while Associate Membership is available to other tertiary qualified healthcare professionals. Members of the public are also invited to become Friends of ACNEM. Members and Friends of ACNEM receive a regular email newsletter, access to resources on the ACNEM website, and the peer-reviewed ACNEM Journal, containing original scientific papers, articles, news and comment relevant to this area of medicine.

Nutritional Medicine is the study and application of the interactions of nutritional factors with human physiology. In particular, it is concerned with the normal biochemical pathways and the consequences of inadequate or inappropriate food intake. Nutritional Medicine is central to health optimisation and fundamental in the prevention and treatment of most conditions.

Environmental Medicine is concerned with those physiological and psychological symptoms and interactions that result from allergy or sensitivity to various inhalants and chemical substances in air, water and food.

Treatment with nutritional and environmental medicine may involve the removal of certain foods or chemicals from the patient's environment, the use of rotation diets and prescription of supplements, such as minerals, vitamins, trace elements and essential fatty acids, where diet alone cannot rectify physiological imbalances. Excesses or deficiencies of any nutrient or the presence of toxic chemicals or electromagnetic radiation may result in cellular dysfunction and illness, whereas the homeostasis promoted in NEM allows optimal self-healing by the body.



Dr Matt Shelton lecturing in the Primary Course

ACNEM training is regarded as unique in the world, with doctors attending regularly from overseas, especially Asia. The four-day foundation (Primary) course and the wide range of topical two-day courses (STPs) are designed for registered health professionals, predominantly medical doctors, who wish to learn more effective ways of treating their patients. Content is strongly referenced and presented by some of Australia's leading medical and clinical experts, with practical tools to aid integration into clinical practice.

ACNEM training leads to a strong sense of collegiality amongst delegates, with many members later enrolling in the ACNEM Fellowship program.

After 25 years of pioneering nutritional and environmental medicine into general

practice, ACNEM is looking forward to a future where 'integrative medicine' is just 'good medicine'.

ACNEM is a fully accredited RACGP QA&CPD training provider for the 2008-2010 Triennium, with 40 Category 1 points allocated to each training program. ACRRM points are also applicable.

## PRIMARY COURSE (4 DAYS)

The Primary course covers the key nutritional, environmental and biochemical factors in well-being. The course enables practitioners to immediately begin practising nutritional medicine, confidently and safely.

## STPS (2 DAYS)

An "STP" is ACNEM-speak for a Special Training Program, usually a two-day, highly practical, discussion-based examination of relevant topics. Prior attendance at the Primary Course is preferred but not essential. Some STPs offer Certification as an optional examination, such as for Chelation Therapy. A wide variety of STPs are offered at locations around Australia and New Zealand to provide continuing specialised education in NEM for our Primary Course graduates, members and other interested health professionals.

## FELLOWSHIP

The ACNEM Fellowship Program is open to medical doctors who are full members of the College and who have completed the Primary Course in the previous two years or are soon to do so.

The Fellowship Program ensures thorough training and practice in NEM, provides peer recognition of a high level of competence in NEM, creates opportunities for "specialty" recognition resulting in higher fees and increased credibility with patients.

For more information about ACNEM, please visit [www.acnem.org](http://www.acnem.org), email [mail@acnem.org](mailto:mail@acnem.org) or phone (03) 9597 0363.

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# FROM THE CEO

Stephen Penman, M App Sc (Health Sc), GC Tert Teach Learn



Dear Members and Friends,

It is with great pleasure that I write on behalf of the Board to invite you to the first ACNEM Fellowship and Graduate Reunion, to be held in Melbourne in March 2010.

You may be aware that the College has been through a time of transition in recent years, and this year has enjoyed a strong sense of renewal with a record number of training events in more locations, strong attendances and increasing memberships.

What better time to have a celebration of ACNEM, to acknowledge the proud history of the College and the growth of NEM over the years? We also want to celebrate some of the initiatives that are underway, such as the development of Online Learning, which we expect to launch in March along with a redesigned nested Certificate, Diploma and Fellowship program.

We've put together a once-in-a-lifetime program for 11-14 March. There is of course, the usual 4-day Primary Course, running Thursday-Sunday. You may like to take advantage of the half price offer to repeat the Primary to see how it has evolved over the years.

Running concurrently with the Primary course will be two, 2-day courses; The GUT (Thursday-Friday), featuring our international guest lecturer, Dr Jean Monro, also Prof Ian Brighthope, Prof Mel Sydney-Smith, Dr James Read and others. Dr Monro is an internationally recognised expert in environmental medicine and Medical Director of Breakspear Hospital in the UK.

The second course (Saturday-Sunday) will be Epigenetics/ Nutrigenomics, featuring Prof Michael Fenech (CSIRO), Dr Naomi Bishop, Prof Mel Sydney-Smith, Dr Russell Cooper, Dr Kerry Harris and others.

On the Friday night we will host a Cocktail Reception, featuring addresses by Prof Ian Brighthope (Past President of ACNEM), Dr Gary Deed (President of ACNEM) and Prof Kerry Phelps



(President of AIMA), followed by a Keynote address by Dr Jean Monro on Provocation Neutralisation. Partners and colleagues are invited. The cost is \$90 per person.

On Saturday night, there will be a Fellowship and Graduate Reunion Dinner, featuring three courses, drinks, live entertainment (also from some of our own!), speakers and other pleasant surprises. The Dinner costs \$150 per person and once again partners and colleagues are invited.

To give you some idea of the sense of occasion, we have invited ACNEM's 168 Life Members and Fellows, and another 1200 past graduates for whom we still have contact details. We have also invited many influential figures in the field, including politicians, academics and business people.

It promises to be a memorable event for ACNEM and an opportunity to share hospitality with old friends, familiar faces and new colleagues, whether you can attend for the whole four days, for two days, or just for the Friday or Saturday night social gatherings. All the details are on the website where you can also register online. You can also RSVP to [mail@acnem.org](mailto:mail@acnem.org) or +61 (0)3 9597 0363.

Finally, our full schedule of training events for 2010 is available on the website, including courses in Melbourne, Perth, Queenstown and Sydney. Those of you interested in Injectable Nutrients or Chelation Therapy training will be pleased to know that we will be holding this popular course in Melbourne in July.

2010 promises to be an exciting year for ACNEM. See you at the Reunion!

With warm regards and seasons greetings,

A handwritten signature in black ink, appearing to read 'Stephen Penman'.

Stephen Penman  
CEO



# JUST BE NORMAL! (SURVIVING MEDICARE)

Karel Hromek, BMed, BSc, FACNEM, FACRRM

Over the years that I have been involved in teaching with ACNEM, I have been concerned about the significant number of ACNEM practitioners who have come to the notice of the Professional Services Review (PSR) for referral concerning their medical practice. Many of ACNEM's senior practitioners have been investigated for possible breaches of the Medical Benefits Scheme (MBS), most frequently for excessive long consultations or excessive pathology testing. Many practitioners have felt that their non-orthodox approach was the underlying reason that they were being investigated.

A recent survey of Australian doctors indicates that 58% of GPs have been the subject of medico-legal action in their working life (Nash 2009), compared with 91% of obstetricians/gynaecologists, 86% of surgeons and 52% of pathologists. The most common medico-legal actions against GPs were complaints to a healthcare complaints body (28%) and compensation claims (21%). Of the GPs, 8% had faced a medical board inquiry and 5% had faced a coronial inquiry. The authors advise that medico-legal action is a "*likely part of medical practice*", and that psychological distress peaked in practitioners facing a current medico-legal matter<sup>6</sup>.

There is a problem with being investigated, in that it is usually very destructive to the practitioner. Doctors who are involved in a medico-legal matter are more likely to experience symptoms such as anxiety, insomnia, depression and social dysfunction than practitioners who had not been investigated<sup>6</sup>. This can go on

for years as the process takes a very long time.

I am not an expert in politics or the law, but I have assisted some practitioners as they have dealt with their PSR referral. I have also prepared and given lectures at ACNEM training on how *not* to become a target for referral. While others may be working to change the system, my advice is essentially designed to reduce the incidence of ACNEM doctors being referred to the PSR and harm minimisation. My advice is also very general and may not be the right approach for specific issues or the right approach at all!

## Why does the PSR investigate doctors?

The purpose of the PSR is to protect the integrity of the Medical Benefits Scheme and the Pharmaceutical Benefits Scheme by protecting the community against inappropriate practice and by preventing costs of services from inappropriate practice<sup>8</sup>. In the most recent yearly, "*Report to the Professions 2007-2008*", the PSR highlighted its concerns<sup>1</sup>:

- Inappropriate prescribing of analgesics and benzodiazepine drugs
- Antibiotic prescribing
- Excessive pathology ordering
- Poor clinical notes

The majority of cases that the PSR dealt with during 2007-2008 generally related to<sup>2</sup>:

- Inappropriate use of MBS attendance items

- Inappropriate use of diagnostic imaging or pathology
- Inappropriate use of MBS procedural items
- Inappropriate prescribing

Referral to the PSR can be for a specific issue, but frequently occurs because of a statistical aberrance from a survey of the practitioner's MBS claims for services. Doctors under referral are compared to all the other general practitioners in Australia, e.g. *during the review period, Dr A provided 13,087 MBS services, which placed him in the 98<sup>th</sup> percentile compared to all vocationally registered practitioners in Australia*<sup>3</sup>.

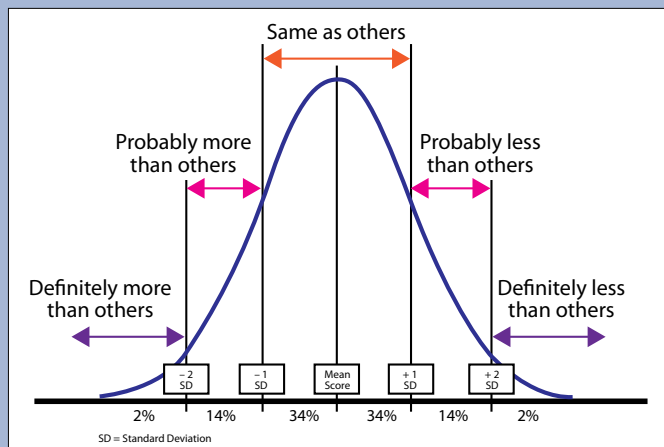
Once referred, a practitioner will be investigated by the PSR. This involves an interview and viewing of the clinical notes. Referral to the PSR does not always result in a finding of inappropriate practice. Following a detailed examination of clinical notes and meeting with the practitioner, the PSR has been able to dismiss about 15% of referrals<sup>4</sup>.

## How do doctors become identified?

The authorities use statistical indices to identify doctors who are practising medicine significantly differently to the majority of similar practitioners in Australia. They can use the item numbers that are claimed by practitioners to draw up a population curve. The two extremes are defined by taking two standard deviations from the mean and anything beyond that is seen to be not normal.

*continued next page*

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Bell shaped curves define normality and are in fact also called *normal curves*. Like any population, a bell shaped curve can be drawn representing doctors and their Medicare claim items. The authorities need only look at the two extremes of the curve to identify doctors who either do too little or too much of whichever item selected. The percentile ratings can be attributed to individual practitioners and those who appear too far from the norm are identified as potentially practising inappropriately which may result in investigation. In other words if you are *normal* you probably won't be investigated, hence the title of this article.

However, if abnormal practitioners are removed from the extremes of the curve by moving them towards the mean or by removing them from the population, other practitioners will now have to occupy the extremes. It is obvious that there will always be some practitioners at each extreme. If successful, over time this approach by the PSR should result in a taller and steeper sloped population curve resembling a tall rectangle.

### Why do NEM doctors attract attention?

NEM practitioners often deal with difficult chronic cases and frequently order a large number of investigations as nutritional medicine is strongly biochemically based. It does not take many repeated investigations to be in the 90<sup>th</sup> percentile or greater. This is particularly apparent for practitioners ordering hormonal

testing like thyroid function tests in the first instance without an abnormal TSH, or a battery of female hormones to adjust bio-identical hormone replacement.

**“BASICALLY, IF YOU WANT TO USE THE SYSTEM, YOU NEED TO STRICTLY FOLLOW THE RULES”**

The PSR is concerned about practitioners who have adopted the practice of ordering the same large bank of tests when investigating patients. For example, ordering B12 and iron studies before receiving the results of a full blood count results in many unnecessary investigations being charged to Medicare<sup>6</sup>. This may not be the best example as it is easy to demonstrate severe B12 or iron deficiency without any haematological parameter indication. However this example does illustrate the manner of approach to interpreting the MBS and PBS. Basically, if you want to use the system, you need to strictly follow the rules.

In its 2005-06 report, the PSR made specific mention of, “*unusual medical practice*”. The PSR reminds practitioners that, “*within the legislation encompassing both schemes there are strict criteria for benefit eligibility. Practitioners providing medicine that can be characterised as alternative or complementary need to be aware that for their services to be eligible for a benefit, they must still meet the prescribed criteria. The most important point is that the service must be clinically relevant. That is, the service must be generally accepted by the medical profession as being necessary for the appropriate treatment of the patient*”.

Then in 2007-08, “*Doctors are reminded that extensive pathology or diagnostic imaging is not a substitute for obtaining a detailed history and making a physical examination. Practitioners who order investigations without clinical relevance will be found to have practised inappropriately and will be required to repay the Medicare benefits for any unnecessary investigations*”.

**“BE EXTRAORDINARY TO YOUR PATIENTS,  
BUT TO MEDICARE, JUST BE NORMAL!”**

NEM practitioners are prone to take a lot of time with their patients and to order a lot of investigations. The clinical notes would therefore need to support the reasoning behind the orders for investigation and the time taken.

**Some suggestions for avoiding PSR review:**

- ♦ Read the Medical Benefits Schedule and the Pharmaceutical Benefits Schedule. You will see how easily some doctors may be inadvertently breaking some of the rules. Obviously you cannot read both large bureaucratic schedules, but have a look at the items that you commonly use.
- ♦ Remember, it's their system, not ours. The rules are the rules. To be fair, if one looks at the published prosecutions by the PSR over the last three reports, the vast majority of offending practitioners appear to be justly prosecuted for excessive services and inappropriate prescribing.
- ♦ Obtain informed consent for any unorthodox treatment or advice.
- ♦ Don't always order the same bank of tests – be patient specific.
- ♦ A good written record is essential to justify the service rendered or pathology initiated. Take extensive notes, paying attention to record the comprehensive examination required when claiming a long or prolonged consultation, and the prerequisites required before ordering pathology testing.

- ♦ Be aware of items that Medicare should not pay for and get your patient to pay privately. Examples include hormonal testing, thyroid function tests with a “normal” TSH or the nutritional status of your patient (unless indicated by your notes!) Don't lose your license to save your patient some money.
- ♦ Caution when you write scripts that your patient fits the strict criteria for prescribing. Write private or non-PBS scripts.
- ♦ Record patient's rejection of conventional treatment, e.g. “Declines statins”. “Seeks bio-identical hormones”, “Declines antibiotics.”
- ♦ Be extraordinary to your patients, but to Medicare, just be normal! Good Luck.

*Karel Hromek is a senior lecturer and Vice President of ACNEM and is an expert advisor for the NSW Health Care Complaints Commission.*

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# A NEW STRAIN OF 'SWINE' INFLUENZA TYPE A H1N1 OR A CHANGE IN SURVEILLANCE?

Judy Wilyman, BSc, MSc, Dip Ed – PhD Candidate Murdoch University

The Australian Government recently prioritised a vaccine for community use against a new strain of influenza. This preventative action is notable as there has been little evidence in the community that suggests this influenza strain is more virulent than other new strains which occur regularly. In fact, the World Health Organization (WHO) in 2009 stated that the majority of people who contract this disease experience the milder form of influenza and recover without requiring treatment<sup>1</sup>.

An examination of evidence provided by the Western Australian Health Department regarding deaths to swine influenza Type A H1N1 prompts us to ask if it is possible that a change in the surveillance of influenza in 2009 has resulted in the creation of hysteria over a new strain of influenza?

Influenza is a disease that is caused by many strains of virus. These viruses spread easily and new strains develop regularly<sup>2</sup>. A vaccine against influenza will only protect against one to three strains depending on the type of vaccine used<sup>3</sup>. For example, the current seasonal influenza vaccine protects against Type A (H1N1), Type A (H3N2) and Type B (3). Influenza Type A H1N1 is a strain that has been covered in influenza vaccines for many years.

The new strain of 'swine' flu is stated to be a recombination of genetic material from human Type A H1N1, a strain of bird flu and 2 strains of pig flu<sup>1</sup>. The WHO states, "there are no known instances of humans getting this strain of

influenza from pigs and other animals". It is also stated that this strain is not known to be endemic in pigs<sup>1</sup>. Yet this flu has been promoted to the public as 'swine flu' even though it is a strain that has never been found in pigs. The public has been misinformed about this strain of influenza. The term 'swine flu' creates anxiety and fear of a disease that has come from pigs when the official medical term for this new strain is 'Influenza Type A, H1N1, human strain'<sup>1</sup>.

The WHO states that influenza A (H1N1) 2009 is a new virus and one to which most people have no or little immunity<sup>1</sup>. In a study conducted by the Centres for Disease Control (CDC) in the US it was shown that individuals between the ages of 18-64 had antibodies present that reacted to the swine flu virus<sup>4</sup>. Whilst this doesn't indicate clinical protection it does suggest that some individuals may have immunity from previous exposure to H1N1<sup>4</sup>. There is no reason to assume that the population will have no immunity to this new strain as it may be immunologically similar to previous H1N1 viruses<sup>5</sup>.

H1N1 is a strain of influenza that has been covered for many years in the seasonal influenza vaccine. Therefore you would expect that the Health Department would have mortality data for seasonal H1N1 from previous years. This appears not to be the case. The Health Department has stated, "This data has not been collected in previous years, or for this year", even though Type A H1N1 has been one of the most virulent and

prevalent strains and regularly covered in the influenza vaccine<sup>3</sup>.

In 2009, the Health Department changed the surveillance of influenza in the community<sup>6</sup>. The Department of Health suggests the reason there is good data on the mortality associated with influenza H1N1 2009 is because of enhanced surveillance systems that were put in place specifically to monitor the pandemic<sup>6</sup>. Prior to 2009, influenza that was notified by General Practitioners (GPs) and laboratories was not systematically followed up or linked to hospitalisation/death data to determine outcomes<sup>6</sup>. In addition, post-mortem victims were not routinely tested for sub-types of influenza<sup>6</sup>. In previous years deaths were listed as 'influenza' and were not routinely sub-typed for the strain<sup>6</sup>. The Health Department also states, "Hospitals were less likely to routinely test admitted patients with respiratory viruses, including pneumonia, for influenza", so (in previous years) many cases remained undiagnosed or were assumed to be primary bacterial infections<sup>6</sup>.

This year most cases of influenza notified by labs or GPs were followed up to see if the cases were hospitalised or resulted in death. The health department was also systematically testing hospitalisations/deaths for H1N1. As a result, the Health Department reports that 90-95% of laboratory proven influenza cases are due to 'swine' H1N1<sup>6</sup>.

It is known that incidence figures for a disease can be inflated by monitoring a disease in a more systematic manner. A





more sensitive or systematic test will identify cases that would previously have gone unidentified. However, a greater incidence of a disease does not always indicate greater severity to the population<sup>7</sup>. This is the case with a disease such as influenza which has a high incidence in the community but epidemics are known to be mild for the majority of people<sup>8</sup>.

How can the public be sure that the number of deaths attributed to this new strain of 'swine' H1N1 is different to the number of deaths associated with seasonal H1N1 in previous years if this testing was not being done?

These changes in surveillance mean that even though influenza Type A H1N1 has been prevalent in previous years there is no data on the number of deaths associated with this strain in previous years because it hasn't been monitored. The Health Department also admits that it is unclear to what extent 'swine' H1N1 infection may have contributed to the deaths it is linked with this year because there are usually several infections present and in most cases, underlying medical conditions<sup>6</sup>. It is well known that disease diagnosis and cause of death is an inexact science and it is up to the medical practitioner to state the primary cause of death<sup>9</sup>. The Health Department has not produced statistics that show the overall death rate for influenza to be significantly worse this year than in previous years<sup>3</sup>. The Therapeutic Goods Association states, "The experience in Australia of the disease is mild in most cases<sup>10</sup>."

The evidence presented above illustrates how surveillance methods can enhance the perception of incidence of disease in the community. This leaves the cause of the increase in incidence open to interpretation. For this reason the government should be required to publicise any changes to surveillance practices whenever there is an increase in incidence reporting of a disease. This will ensure that the information the public receives can be interpreted in an open and transparent fashion that will lead to less fear and panic.

In addition, the government admits that 'swine flu' is a misnomer, thereby misinforming the public, but have stated that they, "Are unable to control how the media reports on the Influenza A (H1N1) virus to the community<sup>10</sup>." Why did the government

not correct this information in the media by stating it is not a swine flu and informing the public of its medical name? This is of concern when it is observed that fear is used to encourage the public to widely accept a medical intervention (vaccination) in healthy individuals.

It is extremely important that we have an accurate knowledge of the potential harm associated with the use of multiple vaccines in otherwise healthy individuals and until this science is complete we need to assess carefully how many vaccines are necessary. A change in surveillance has a significant impact on the perception of incidence of disease in the community and the Public as the main stakeholder in the use of vaccines, has a right and a need to understand the basis on which disease incidence is reported, without which we cannot make a proper assessment of the need for a vaccine.

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# PUTTING STATINS IN PERSPECTIVE

Associate Professor Peter Dingle, BEd, BSc, PhD

Millions of Australians are prescribed cholesterol-lowering drugs called statins, such as Pravachol, Zocor and Lipitor at a cost of more than one billion dollars to the healthcare system each year. This article maintains that despite the pro-statin hype, at best these drugs lower the real risk of heart attack or stroke by less than 1% and at the same time have serious side effects in up to 5% of the population of users. Unfortunately, far too many people take statins without considering the alternatives, and far too many prescriptions are dished out by GPs without fully understanding the literature.

The first indication that something may be wrong is that the majority of what we know about statins and their effects (beneficial or otherwise) comes directly from scientific trials which are largely funded and coordinated by the drug companies<sup>1</sup>, not from long-term, independent, evidence-based observations. As a result, most of the information relating to statins we have received is likely to be biased.

The cholesterol-lowering program of the past 30 years has in large part failed to stem the epidemic of cardiovascular disease. At the same time, the focus on cholesterol reduction alone has deflected interest in other therapeutic aspects of inflammation treatment that provide significantly greater benefit. It has been known now for many years that despite significant Low Density Lipoprotein-cholesterol (LDL-C) reduction, large numbers of subjects in the statin treatment groups continue to have heart attacks and strokes despite achieving significant LDL-C reduction. This myopic focus on LDL alone is not surprising given the vested interests of the pharmaceutical industry but it has distracted us from the real problem. Cholesterol is not the notorious substance that it is made out to be, it is just the messenger. Despite this, the statin drugs do provide a small benefit of reducing the risk of heart attack or stroke. One of these effects - unrelated to lipid lowering - is to stimulate nitric oxide (NO) in the arteries<sup>2</sup>, which has numerous positive effects on the arteries and blood vessels<sup>3</sup>. A number of foods such as almonds can also achieve this outcome. The only other scientifically proven action of statins is their

capability of lowering blood levels of C-reactive protein (CRP), a marker of inflammation in the body, and a major risk factor for heart disease. Raised CRP levels and raised cholesterol levels in the blood are both the symptoms of an underlying problem but like cholesterol, CRP is not the cause<sup>4</sup>.

Therefore, the reasons that some studies have found statins to bring about a small 'real' reduction in the risk of CVD may not be as a result of the reduction in the blood cholesterol level but rather their effect on nitric oxide and their action as an anti-inflammatory agent. However, there are many very cheap and natural ways to reduce inflammation and improve nitric oxide levels, most of which involve healthy food and lifestyle changes.

Statin therapy is extremely efficient in lowering cholesterol numbers but unfortunately not without adverse effects on the body<sup>5</sup>. To prevent a first heart attack, for every life that is saved (1% over 10 years of use), statins cause an equal number of adverse deaths due to accidents, infection, suicide and cancer, also 1% over 10 years of use<sup>6</sup>, and significantly greater levels of serious side effects and suffering.

Because statins interfere with major biochemical pathways they have serious side effects. Statins inhibit the production of many other vital substances as well as cholesterol. A recent review of the adverse effects of these drugs included more than 900 studies<sup>7</sup>. Statin drugs block Coenzyme Q10 (CoQ10), which is an essential enzyme involved in energy production and also acts as an essential fat soluble antioxidant<sup>8</sup>. CoQ10 plays a vital role protecting the heart and cardio vascular system<sup>9,10,11,12</sup> and is our natural defence against atherosclerosis development, the build up of plaque in the arteries that leads to cardiovascular disease. CoQ10 inhibits the oxidation of LDL cholesterol, inappropriate clotting of the blood and ultimately lowers blood pressure<sup>13,14</sup>.

Statin treatment may also lead to serious muscle toxicity<sup>15</sup>. At least 5% to 7% of statin users experience significant muscle problems<sup>16</sup>, more than 10% if higher doses are taken<sup>17</sup>, and as

many as a quarter of statin users who exercise may experience muscle fatigue, weakness, aches, and cramping due to statin therapy<sup>18</sup>. This defeats the purpose when those with elevated risk of heart attack or stroke find it hard to exercise. Statins have also been implicated as negatively impacting brain function<sup>19,20</sup>. Cholesterol is the most abundant organic molecule in the brain<sup>21</sup>. The housekeeping functions in the brain, synapse function<sup>22,23,24</sup> and serotonin, all rely on cholesterol produced in the brain because it is too large to pass through the blood-brain barrier<sup>25</sup>. The statin drugs can easily pass into the brain and directly interfere with the synthesis of cholesterol in the brain<sup>26</sup>. No wonder a major side effect of the statin drugs is their impact on memory and thinking. Amnesia is a known adverse effect of taking Lipitor. A study by the drug company Pfizer found two percent of people taking Lipitor have serious amnesia<sup>27</sup>. Ironically, the Amnesia was only recorded if it was remembered and reported by the study participants. Many people reported memory blanks and forgetfulness, but this was not considered as amnesia in the study<sup>27</sup>. Even so the two percent is at least 385 times more likely than the general population to have amnesia.

In a study to see the effects on patients of raising the Lipitor levels from 10 to 80mg, those taking 80mg had increased liver problems, i.e. raised liver enzymes, six times higher than those given 10mg of Lipitor. Even though the total deaths due to CVD in the 80mg group was less (126) than the 10mg group (155), the total deaths due to other causes was higher in the 80mg (158) than the 10mg (127) group. There was no difference in the overall mortality rate.

If they have major side effects, and far too many for me to describe here, what are the benefits? While there is a lot of hype about the benefits of statins, there are almost as many studies showing no benefits at all. This may be brought about by the misuse of statistics. Various independent studies in prestigious peer reviewed scientific journals have shown that statin use in primary prevention, where there is no previous history of a heart attack or stroke, has minimal or no value in reducing mortality<sup>29,30</sup>. To quote one of the papers, "Primary prevention with statins provides only small and clinically hardly relevant improvement of cardiovascular morbidity/mortality<sup>31</sup>." Another review found, "Current clinical evidence does not demonstrate that titrating lipid therapy to achieve proposed low LDL cholesterol levels is beneficial or safe<sup>32</sup>."

As readers of the scientific literature, we should not get confused between statistical significance and clinical significance. Statistically significant means that the outcome was likely (95% chance) a result of the treatment whether it was 100% effective or less than 0.1% effective. That is if you treat 1000 people to save one life (0.1%) it may be statistically significant but it is not necessarily clinically significant. Clinical significance is generally 20 to 30% or more. Tests of significance should always be accompanied by effect size statistics, which approximate the size and thus the practical importance of the difference. The studies on statins usually only report statistical significance, mostly 1% or less, and none have so far found any clinical significance.

*continued next page*



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The studies on statins also usually report relative risk not absolute or real risk. The reported relative risk reduction can be highly misleading<sup>33,34,35,36,37</sup> if not deceptive. An example of relative risk is where you have 4 people in a study who die in the placebo group (no drug) compared to 3 people who die in the drug treatment group - that is 4 were also supposed to die in the drug treatment group but only 3 did - then there is a 25% relative risk reduction. However, if to get the effect of saving one life you had to treat 1000 people, the real risk reduction would be 0.1%.

A well known study on statin use, the JUPITER Study, reported 68 heart attacks in the placebo group compared to 31 heart attacks in the drug treatment group, a 58% relative risk reduction, and 64 strokes in the placebo group compared to 33 strokes in the treatment group, a relative risk reduction of 48%<sup>38</sup>. Sounds good doesn't it? However, the drug treatment group had 8901 participants in it. In real terms the heart attack risk went from a low 0.76% to 0.35% and the risk of stroke went from 0.72% to 0.37%. Effectively if you treated 300 people with these drugs you might save one life. Under the best possible scenario the real risk reduction in this study was well under one half of one percent. By comparison, the real risk reduction from regularly

consuming a handful of raw mixed nuts is around 30% and a relative risk reduction of more than 600%.

Even treating people with high cholesterol and other risk factors, the real risk reduction comes in below 1%<sup>39,40,41,42</sup>. The Heart Protection Study in the United Kingdom, with over 20,000 participants aged 40 to 80 years with high risk of cardiovascular disease (CVD), produced a 25% relative risk reduction over five years<sup>43</sup>. There were 10,269 people on statins and 10,267 people on placebo. The real percentage improvement was actually 1.7% over 5 years. This means that over the five year study of people at high risk with previous cerebrovascular disease, peripheral artery disease, renal impairment or diabetes, they saved 25 people per year. These were seriously ill people and they still only got a benefit of 1.7% over 5 years. They also forgot to mention that around 30,000 people were dropped from the study, and NOT counted in the percentage of people with side effects.

More recently, a meta-analysis of 10 randomised clinical trials of about 70,000 people with risk factors for cardiovascular disease but no history of existing disease, had a relative risk reduction of 12% for total mortality, 30% for coronary event and 19% for a cerebrovascular event<sup>44</sup>. However, the real risk reduction was 0.6%,

1.3% and 0.4% respectively. The actual number of people needed to treat to save one life was 167.

It is not just the scientists jumping up and down over the mis-quoting of statistics and resulting potential misuse of drugs. Health economists are also questioning the reason for so much statin treatment. In an economic review of statin use, the authors reported that it is not cost effective to treat low risk people<sup>45</sup>. A recent study in the UK found statins in primary prevention cost £27,828 per life-years gained (LYG), reaching £69,373 per LYG in men aged 35 to 44<sup>34</sup>. That is to add one year to a persons' life they need to spend £69,373 (around A\$125,000) per year, and reported that, "Amounts of NHS funding are being spent on relatively less cost-effective interventions, such as statins for primary prevention". Perhaps you might say that every life is worth that. Unfortunately, it is a big economic price to pay and one we cannot afford. On the positive side, nutrition and lifestyle changes can bring about much greater real benefits and at much lower costs, but not when people think drugs are the only solution.

Most medical research is financed by the pharmaceutical companies; the same companies who conduct the research and pay the researchers, also decide what







is or is not to be published. It has been shown that pharmaceutical companies routinely fail to publish negative studies. Yet this same 'scientific evidence' informs the medical profession and public health policy.

Many doctors feel constrained to prescribe statins because they are now expected to be prescribed, both by the medical profession and the public, despite scientific evidence to the contrary and that there may be other means of tackling the problem. In this author's view, the pharmaceutical companies have made a complete farce of the medical system, rendering the medical profession the retail arm of the pharmaceutical industry, and making it difficult or impossible to convince people otherwise. Cholesterol is not the killer. It is not even a risk factor, it is a symptom.

If this does not convince you that we have a problem with these drugs, I have hundreds of other scientific studies that add weight to my argument. It is time for a public debate on this. Please visit [www.drddingle.com](http://www.drddingle.com), print a copy of this article, send it to your local GP and distribute it as widely as possible.

#### About the author

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#### Acknowledgments

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# CASE STUDY OF Ms BM – COELIAC DISEASE

Jim Bartley, MBChB, FRACS, FFPMANZCA

## PRESENTING HISTORY

Ms BM was a 29-year-old European lady who presented with a 4-year history of left cheek pain. The pain was continual, rated 6/10 in severity and could be associated with nausea and vomiting. The pain was said to have started following an episode of infective sinusitis. She mentioned that she might have been iron deficient at the time. She had had extensive dental work done looking for a dental cause of the pain. Three dentists and three otolaryngologists had been unable to find a cause for the pain. She had no neck pain, shoulder pain or low back pain. There were no irritable bowel symptoms or period problems. There was no past history of anxiety or depression. The pain was poorly controlled with conventional anti-inflammatory drugs and Amitriptyline. She had had normal ultrasound and MRI scans of the area. Previous blood tests had shown an iron deficiency, but no anaemia. She was married with two children. She had tertiary and post-graduate qualifications.

## EXAMINATION

Height 165 cm, weight 42kg, BMI 16. Tender left masseter muscle; palpation of muscle replicates pain. No other significant physical findings. Trigger point in left masseter muscle.

## INITIAL MANAGEMENT

1. Injection of the left masseter muscle with local anaesthetic. This completely relieved the pain for 6 days.
2. She was advised regarding heat and self massage techniques to the masseter muscle.
3. Investigations were initially performed firstly to exclude metabolic causes for the trigger point and secondly looking for causes of the repeated iron deficiency.

## RESULTS OF INITIAL INVESTIGATIONS

Iron 12µg/L, ferritin 42µg/L, vitamin B12 528pmol/L, zinc 11 µmol/L and random urinary iodide 50µmol/L, tissue transglutaminase (IgG) 2 and tissue transglutaminase (IgA) 2, Vitamin D 69 nmol/L, MCV 86fl and MCH 27pg.

## INITIAL MANAGEMENT UPON RECEIPT OF INVESTIGATIONS

She was advised to take vitamin D, zinc and iodine. A gluten-free, dairy-free diet was also discussed but the patient was extremely reluctant to trial this.

A blood test 3 months later showed an iron level of 3µmol/L. Because of the long history of intermittent iron deficiency and her low BMI, she was sent to a gastroenterologist to exclude Coeliac Disease (CD) as a cause of her persisting iron deficiency. The duodenal biopsies (six) were positive for CD:

“Villi are of a variable height, some quite short. The lamina propria contains increased chronic inflammatory cells. The surface epithelium is heavily infiltrated by lymphocytes. No parasites are identified. Appearances are consistent with coeliac disease.”

## SUBSEQUENT MANAGEMENT

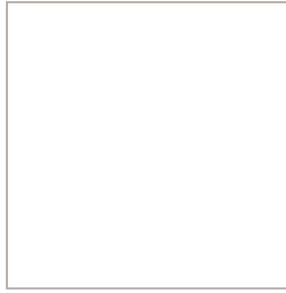
When placed on a gluten-free diet her pain resolved almost completely and she was able to stop all pain medication. She put on 2kg in weight. Despite my advice to also go dairy free she was reluctant to and did not do this.

## DISCUSSION

This case illustrates the difficulties in diagnosing CD. Classic textbook symptoms include abdominal bloating, vomiting, diarrhoea, weight loss, and in children, stunted growth. However abnormal bowel symptoms, particularly in adults and also in this case, may be limited or even absent. With this patient, the clue was the repeated low iron levels. There was very little on blood testing alone that caused one's suspicions to be raised. There were minimal other clues to suggest other nutritional deficiencies. MCV and MCH were in the lower half of the normal range. While she was zinc deficient, zinc deficiency is common in the New Zealand population. New Zealand soils are frequently deficient in both zinc and selenium. Blood testing for zinc status is also notoriously unreliable. While this lady had a low BMI in keeping with CD, this is not necessarily a reliable clinical sign<sup>1</sup>.

## INVESTIGATIONS

Antibody testing for transglutaminase IgA antibodies has a reported sensitivity (diagnosis of disease) of greater than 90% and a specificity (exclusion of disease) of greater than 95%. HLA testing is more useful in excluding CD. Duodenal biopsy remains the gold standard due to the false-negative rate associated with serology alone. This was the situation with this patient. Multiple samples (ideally 6) need to be taken from the second part of the duodenum and beyond on endoscopy. Not all areas may be equally affected; if biopsies are taken from healthy bowel tissue, the result will be a false negative<sup>2</sup>.



## **PATHOPHYSIOLOGY**

CD is a complex multifactorial condition in that while genetic factors are important, other factors are necessary for the disease to manifest itself. The vast majority of coeliac patients express the HLA-DQ2 or HLA-DQ8 genes, which are part of the MHC class II antigen presenting receptor system distinguishing cells between self and non-self in the immune system. However in the West, 25-30% of the population express HLA DQ2 and are exposed to gluten, but only a minority develop CD. Around 5% of patients with CD do not have the HLA-DQ2 gene<sup>3</sup>. The HLA-DQ molecules bind to and present gluten derived peptides to T lymphocytes, initiating the autoimmune process. Most coeliac patients have a two-gene HLA-DQ2 haplotype referred to as DQ2.5 haplotype. While most coeliac patients inherit only one copy of this DQ2.5 haplotype, some inherit it from both parents. This latter group is especially at risk for CD, as well as being more susceptible to severe complications<sup>4</sup>. The frequency of the HLA-DQ genes varies geographically. DQ2.5 has a high frequency in peoples of North and Western Europe; Ireland particularly has a high frequency<sup>5</sup>. While genetic predisposition is a factor, the amount of gluten in the diet may also predispose people to CD. In the Swedish CD epidemic an increase in gluten content in the food resulted in a tripling of CD incidence<sup>6</sup>. However a diet high in gluten is also recognised to cause intestinal damage, regardless of whether a person has CD or not<sup>7</sup>.

Gliadin in wheat belongs to a group of storage proteins called prolamins, rich in proline (*prol-*) and glutamine (*-amin*). Hordein in barley and secalin in rye are also prolamins. One particular region in a-gliadin stimulates enterocytes in the gut allowing the leakage of larger molecules between the cells. These gliadin peptides then stimulate both the innate and the adaptive (T-helper cell mediated) immune

response. The innate immune response to gliadin results in inflammation.

The enzyme tissue transglutaminase (tTG) generates gluten peptides that bind with high affinity to HLA-DQ2 or HLA DQ8. Antibodies to the enzyme tTG are present in most coeliac patients. Gluten peptides are modified by tTG in two ways, deamination or transamination. In deamination, a glutamate residue is formed. In transamination, cross-linking of a glutamine residue from the gliadin peptide to a lysine residue of tTG occurs. Cross-linking may occur either within or outside the active site of the enzyme. This results in the formation of new epitopes, which are believed to then trigger the primary immune response by which the autoantibodies against tTG develop<sup>8,9</sup>. The inflammatory process, mediated by T cells, leads to disruption of the structure and function of the small bowel's mucosal lining causing malabsorption.

## **MUSCLES AND COELIAC DISEASE**

Myopathy in the context of gluten sensitivity exists, but has not been well characterized. The myopathy normally presents as a proximal muscle weakness. Endomysium-specific antibodies (antibodies that bind with high affinity to cell-surface tTG) are thought to be involved, but these are also the most sensitive markers for the presence of enteropathy, which were not present in this patient. A spectrum of abnormalities has been described on muscle histology in gluten sensitivity-associated myopathy. Muscle inflammation is a common feature. IgA deposits against tTG have been demonstrated in extra intestinal target sites such as muscle in a patient with coeliac disease and myopathy and cerebellar tissue in a patient with gluten ataxia. Myopathy may be a manifestation of gluten sensitivity and may have an immune mediated pathogenesis<sup>10</sup>.

## **CONCLUSION**

This case report documents an unusual presentation of CD, where a high index of clinical suspicion was necessary to make the diagnosis. The reasons for the isolated masseter muscle pain are difficult to explain, but a potential etiological mechanism is described.

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# CASE STUDY OF Ms KL – ADRENAL FATIGUE

Paul Wheatley, MBBS, DRACOG, FRACGP, FACNEM

I was asked to see Ms KL, a 46 year old woman with fatigue, by a colleague who had been caring for her the previous year and a half. The patient had been diagnosed as having Chronic Fatigue Syndrome five years prior. To a lesser degree, she was troubled by pain in her muscles, diagnosed as fibromyalgia, as well as insomnia. She reported low energy, particularly in the morning. She had bloating and flatulence and some pain in the right iliac fossa and had identified dairy and gluten as triggers. Stool analysis for dysbiosis done previously showed reduced lactobacilli but no ova, cysts or parasites. She had taken probiotics and nystatin anticandida treatment without any obvious improvement.

Her past history included asthma and rosacea. She was treated for helicobacter pylori in 1990. Her serum DHEAS and serum morning cortisol, assessed one and a half years previously, had both been within laboratory reference range, 5.4 (RR 1.0 -11.6) and 539 (RR 138-690) respectively.

Family history included osteoporosis and osteoarthritis on the paternal side and a sibling with irritable bowel syndrome. Death of a family member and of a good friend in the previous 10 years had distressed her greatly.

On examination she was a slim woman with mild abdominal tenderness and painful trigger points in keeping with her diagnosis of fibromyalgia.

Salivary cortisol and melatonin tests showed a low morning cortisol level (6 with a target of 25) (Table 1). The highest level should occur at 6am whereas both her 12pm and 6pm levels were higher than at 6am. The 6pm level should also be significantly lower than earlier levels but it was similar to the level at 12pm suggesting the normal diurnal rhythm in cortisol levels had been disturbed.

Table 1. Results of salivary cortisol (nmol/l) and melatonin (pg/ml) testing throughout one day and night in chronological order. Reference range is supplied by Analytical Reference Laboratory.

Time of day (hrs)	Hormone	Result	Reference range	Target
0600	cortisol	6	5.3-25	25
1200	cortisol	9	NA	15
1800	cortisol	8	1.2-12.3	5-10
2200	cortisol	2	NA	<5
2400	melatonin	18	10-40	35
0600	melatonin	37	<3	1

NA indicates no range specified.

In addition her melatonin remained elevated at 6am when it should have been much lower than it was on the preceding evening at 12 midnight. This may be due to inadequate suppression of melatonin production by cortisol. Poor liver detoxification facilitating clearance of melatonin may also play a role.

These results were from sampling on a single day (n=1). Replicate sampling over a greater number of days would enable a higher level of certainty. However, a single low cortisol result is likely to be more reliable than a single raised result, which could have been affected by stress or excess coffee or sugar on that particular day.

A diagnosis of adrenal fatigue was made based on the combination of her symptoms and low early morning cortisol of 6 (RR 5.3-25 with target of 25) (Table 1). This may have developed as a consequence of the stress related to the two deaths that preceded the development of her fatigue. Her serum morning cortisol taken a year and a half previously, in the upper normal range, makes Addisons disease unlikely and it was considered that a Short Synacthen Test was not warranted.





She was commenced on a tablet formulation containing Withania 12g daily (extract equivalent to dried root), Liquorice 12g equivalent daily, Siberian ginseng 9g equivalent daily, Korean ginseng 0.9g equivalent daily and tyrosine 1.5g daily, as well as Gingko biloba 4g (equivalent to dried leaf) daily, vitamin C 1g bd, vitamin B complex with an additional 550mg of Pantothenic acid and 100mg of B6, Mg 200mg bd and Vitamin E 500mg daily. It should be noted that Korean ginseng (*Panax ginseng*) has the possibility in women of exerting effects similar to excess DHEA with an increase in facial hair and acne. This preparation was used for its convenience with observation for any early signs of this effect.

We discussed her need for salt and possible problems with reactive hypoglycaemia. She was advised to purchase a copy of James Wilson's book "Adrenal Fatigue the 21<sup>st</sup> century stress syndrome", which provides both a useful explanation and additional treatment suggestions for patients. We looked at her ongoing stress and what could be done to address that. She was encouraged to engage in a mindfulness meditation program. She was advised to open her curtains to ensure light in the morning and to help re-establish her body clock.

She presented three months later reporting that she had less fatigue.

Avoidance of large amounts of fruit as part of her management of hypoglycaemia had also led to an improvement in her bowel symptoms.

Her insomnia was still a problem. Her raised melatonin in the morning suggests a shift in the melatonin curve to the right as has been observed in patients with chronic illness. As well as possible interference with getting to sleep this is likely to be an additional contributor to her morning fatigue. Future management may involve support for melatonin production through the use of tryptophan and possibly a trial of melatonin in the hope of resetting her body clock. Her symptoms of abdominal bloating, flatulence and pain may be related to food sensitivities and future management could also involve the use of an exclusions diet.

Cortisol (or hydrocortisone) to provide physiological levels, and pregnenolone could play a role in her management if required, with risks weighed against advantages.

The family history of osteoporosis should be considered and bone density monitoring would be mandatory. Adrenal support is the preferred treatment option and has been helpful to this point.

This case suggests that a morning serum cortisol test, which in this case had indicated normal cortisol levels in the

higher part of the reference range when tested one and a half years previously, may not be a reliable way of assessing dynamic adrenal function. Serum is considered to be a more reliable estimate of DHEAs production and since her blood levels were adequate when tested, salivary levels were not ordered.

Adrenal fatigue is likely to be the primary event causing her fatigue, possible food sensitivities, as well as acting as a trigger for her fibromyalgia. It is hoped that ongoing management for adrenal fatigue will allow for continuing improvement in her symptoms.

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# MINDD FOUNDATION

Leslie Embersits, BA, MA

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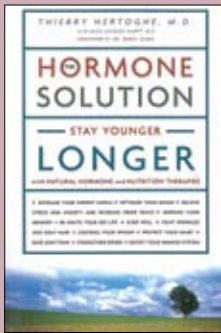
## BOOK REVIEWS



### **'ADRENAL FATIGUE – THE 21ST CENTURY STRESS SYNDROME' BY JAMES L WILSON, ND, DC, PHD**

Reviewed by Dr Sandeep Gupta, MBBS (UQ), FACNEM

This is one of the few comprehensive books on this extremely important but unrecognised condition. The book, although written in layperson's language, covers the signs and symptoms of adrenal fatigue, its causes, progression, prognosis and management in an engaging and entertaining manner. The illustrations in the book are particularly engaging, and aptly depict the caricature of the person suffering from this disorder. The book also covers pertinent topics in diagnosis such as the difficulty in using blood tests to diagnose adrenal fatigue, the use of salivary hormone and synacthen stimulation testing, and use of examination screens such as postural blood pressure measurement. Importantly it places lifestyle and diet at the core of managing the condition, with herbal, nutritional and hormone replacement medicine as supplementary measures. Appropriately so. And despite the casual 'down-to-earth' presentation of the book, it has an extensive list of references for each chapter for those who wish to delve deeper into the medical literature and confirm that this disorder is in fact as scientifically based as any other, despite the paucity of medical awareness of the condition. Highly recommended for practitioners and patients alike.



### **THE HORMONE SOLUTION - STAY YOUNGER LONGER BY THIERRY HERTOGE, MD**

Reviewed by Shirley Schurmann, RN, BAppSci, Dip NurseEd, MEdStudies, Grad Cert Nutr & Enviro Med

Hormones are crucial to every single function of our bodies, however according to the author, in the environment in which we live and as we age we have less than optimal levels of hormones thus less than optimal health.

The programs in this book address issues such as fatigue, forgetfulness, insomnia, anxiety and depression with a combination of nutrition and hormone balancing.

The book gives an overview of the major hormones and how they work, along with signs and symptoms of deficiencies and imbalances as well the laboratory tests required to assess hormone status. Importantly, the author claims that without balanced nutrition and the right diet, the body cannot make all the hormones it needs in the proportions required. If hormonal supplementation is still necessary, bio-identical hormones can recreate the state of health and wellbeing once enjoyed, he says.

The middle section of the book consists of a series of self tests that identify signs and symptoms of deficiency of each of the most important hormone's impact on issues such as energy, sex, sleep, skin and hair, weight control, stress, mood, circulation, joints, bones and immune system. Detecting and decoding deficiencies in each of these areas are addressed with plans on how to eat for healing, nutrients required and hormonal

supplementation necessary. The author reiterates that any perceived deficiencies will need to be confirmed with lab tests before treatment is commenced.

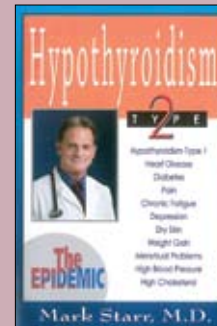
The last section of The Hormone Solution considers diet the most natural way to balance hormones. It claims there is no one diet plan that can optimise hormone levels for every person. The author gives an outline of the best way to eat to support hormones thereby addressing each person's unique situation and applying eating plans to specific hormonal deficiencies and to specific health conditions.

If all else fails, the last chapter reviews hormonal treatment recommendations as well as nutritional information that can be used to balance hormones or improve the performance of hormone supplements already being used to achieve optimal health outcomes.

The author addresses popular misconceptions and 'bad press' given to the mainly synthetic large dose hormone therapy used in conventional medicine. This book has a sound evidence-base with 59 pages of references, numerous case studies and the experience of a third-generation endocrinologist with long standing interest and experience. It is a valuable resource for medical professionals to better understand chronic disease from a hormonal perspective.

From a patient viewpoint, understanding of the early signs of hormonal inefficiencies prior to ensuing chronic disease manifestation is important. For those with existing illness the book may provide insights to hormonal imbalances that underpin their condition.

The book is a valuable resource for patients to work with their doctor to attain optimal health.



### **'HYPOTHYROIDISM TYPE 2' BY MARK STARR, M.D**

Reviewed by Dr Sandeep Gupta, MBBS(UQ), FACNEM

Undiagnosed or undertreated hypothyroidism is a major epidemic in Australia currently. This book specifically deals with "type 2" hypothyroidism (as Starr refers to it), that is thyroid disease in which the levels of thyroid hormones in the blood are normal however the intracellular utilisation of thyroid hormones are low. Also known as peripheral thyroid resistance, this form of thyroid hormone deficiency is well described in the literature, however is often missed by the standard "TSH" blood test. The book, although designed for the lay public, is at a level at which practitioners new to this form of thyroid disorder, will find benefit. The majority of the text is devoted to the symptoms and different organ manifestations of type 2 hyperthyroidism, which while slightly cumbersome is certainly exhaustive. The sections on treatment and diagnosis, although brief, cover major points including basal temperature testing and the use of dessicated thyroid. Overall, this book, although lacking in conciseness, is a good introduction to the subject for patients and practitioners alike.

# IN THE NEWS

Shirley Schurmann, RN, BAppSci, Dip NurseEd, MEdStudies, Grad Cert Nutr & Enviro Med

## SELENIUM COULD CUT CANCER RISK

A recent Australian study has shown that selenium could dramatically reduce the incidence of bowel cancer. The study last year by Prof Graeme Young, head of the Flinders Centre for Cancer Prevention and Control at Flinders University, showed that mice fed extra selenium had a 60% reduced incidence of bowel cancer. A follow up study of 23 healthy people aged over 50 who had extra selenium added to their daily milk, indicated a likely similar protective effect. Findings were presented at the Australian Gastroenterology Week conference in Adelaide.

Ref: <http://www.smb.com.au/lifestyle/wellbeing/mineral-could-cut-cancer-risk-20091023-hc3c.html>

Readers may remember an earlier trial of selenium supplementation for skin cancer prevention, in which significant reductions in total cancer mortality and total cancer incidence in the treatment group caused the trial to be stopped early.

Conducted by Clark et al, of the Nutritional Prevention of Cancer Study Group at the University of Arizona in 1996, this RCT looked at 1312 patients with a history of basal or squamous cell carcinoma to determine the effects of oral supplementation with 200mcg selenium per day or placebo. No selenium toxicity occurred.

Selenium did not significantly affect the incidence of basal cell or squamous cell carcinoma of the skin. However analysis of secondary end points revealed that patients treated with selenium compared with controls, had a non significant reduction in all cause mortality, and significant reductions in total cancer mortality (50%), total cancer incidence, and incidence of lung colorectal and prostate cancer.

Results suggested that supplemental selenium may reduce the incidence of and mortality from carcinomas of several sites. The patients were all from areas with low amounts of selenium in the soil.

The researchers reported that the effects of selenium on skin cancer prevention still required confirmation, however this significant research could be seen as a starting point.

We expect the selenium story to continue unfolding in future years.

Ref: Clark L et al: JAMA. 1996 Dec 25;276(24):1957-63.

## FEELING BLUE? TRY A HIGH CARB, LOW FAT DIET

An Australian study published in The Archives of Internal Medicine that compared a low carb diet high in fat and protein with a low fat diet that was rich in carbs, found that the high carb diet was more beneficial to the dieter's mood.

Scientists from the CSIRO, SA University, and Flinders University conducted a randomised clinical trial involving 106 overweight and obese participants with an average age

of 50 years. Of these, 55 were randomly assigned to follow a low carbohydrate, high fat, high protein diet and 51 to a high carbohydrate, low fat diet for one year. Changes in body weight, mood and wellbeing, and cognitive functioning were assessed periodically during and following the one year intervention.

After one year, the overall average weight loss was 13.7 k with no difference between the two groups. Both groups initially (after the first eight weeks) experienced an improvement in mood. However most measurements of mood revealed a lasting improvement only in those following the high carb low fat diet, while those on the high fat, low carb diet returned to their initial baseline levels.

The mechanism for the observed effect on mood still remains largely unknown, explained Grant D Brinkworth, research scientist at the CSIRO. The mood difference could reflect how difficult it is to comply with a low carb diet in Australia where the typical diet consists of about 50% carbohydrate.

Altered mood has been shown to influence interpersonal behaviour and therefore the consumption of a very low carb diet may have psychosocial consequences for interpersonal behaviour and relationships. One of the factors that may pose risk for poor long term weight maintenance may be eating in response to negative emotions and stress.

Sustained weight loss and wellbeing may be better addressed by a balanced diet such as the Zone or the Paleolithic diet. These have the macronutrient balance and phytonutrients our bodies need, rather than the nutritionally empty calories of pasta and bread.

Ref: <http://www.smb.com.au/lifestyle/wellbeing/lowcarb-diets-can-put-you-in-the-grumpy-zone-20091110-i7lc.html>

## AUSTRALIANS RELAXED ABOUT H1N1 PANDEMIC

Two thirds of respondents in a national study of adult Australians indicated a willingness to receive pandemic (H1N1) vaccine despite 78% of respondents regarding the current influenza pandemic as only causing mild disease, and only 25% thought they were at increased risk of infection.

This intended vaccination uptake would achieve the coverage required to ensure herd immunity to pandemic influenza according to this survey published in the Medical Journal of Australia.

Australians also expressed a spirit of generosity with 96% of respondents supporting donation of surplus vaccine to neighboring developing countries. There is a theoretical excess of almost seven million of Australia's 21 million doses.

With 16% of respondents reporting being undecided about vaccination, results indicate a need to provide accessible information on vaccine safety.

NB: Updated treatment guidelines for H1N1 influenza from the World Health Organisation urge clinicians to administer antiviral



medications as soon as possible to patients in at risk groups with flu like symptoms, patients with pneumonia and those with uncomplicated influenza-like illness that worsens or fails to improve within 72 hours.

Ref: [http://www.mja.com.au/public/issues/192\\_01\\_040110/eas11124\\_fm.html](http://www.mja.com.au/public/issues/192_01_040110/eas11124_fm.html)

### **A MEDICAL ICE CREAM TO LICK SIDE EFFECTS OF CHEMOTHERAPY**

A new medical ice cream developed by Fonterra and the University of Auckland has shown promise for combating some of the unpleasant side effects of chemotherapy.

Clinical trials have started in NZ to assess the effectiveness of the ice cream known as "ReCharge" against chemotherapy induced diarrhea and anaemia, but the dessert with a difference could also reduce weight loss and damage to the immune system during chemotherapy.

200 patients from Oncology centres in seven NZ cities are taking part in the trial. Volunteers in the trial will undergo a daily regime that includes eating a 100g tub of strawberry ice cream that contains two bio active ingredients combined to address the unpleasant effects of chemotherapy.

Earlier trials in the laboratory found that weight loss and damage to the gut lining were significantly reduced by the active ingredients in ReCharge. There was also marked improvement in the immune system and blood markers, said Associate Professor Geoff Krissanson of LactoPharma, who is leading the project in conjunction with the Foundation for Research Science and Technology.

Ref: [http://www.uniservices.co.nz/uploadedfiles/uniservices/MediaRelease\\_MedicalIcecream.pdf](http://www.uniservices.co.nz/uploadedfiles/uniservices/MediaRelease_MedicalIcecream.pdf)

### **LIFESTYLE-BASED DIABETES PREVENTION HELPS FOR AT LEAST 10 YEARS**

A number of prospective studies have shown that behavioural and lifestyle interventions can provide lasting protection against new type 2 diabetes in people at increased risk. A recent study conducted by the Diabetes Prevention Program (DPP) research group published in the Lancet saw protective effects of both lifestyle interventions and treatment with Metformin lasting as long as 10 years. Their findings come from an unblinded extended follow up of most patients from the original DPP published in 2002 that compared an intensive lifestyle intervention program with Metformin or placebo in 3234 non-diabetic patients with elevated fasting glucose and impaired glucose tolerance.

The rate of new diabetes fell by 58% with intensive lifestyle intervention, or 31% with Metformin only compared to placebo over a mean of 2.8 years. Over the subsequent mean follow up of 5.7 years, the original lifestyle intervention subjects maintained their low onset diabetes rate during the extended phase.

In a combined analysis of the two studies the new diabetes rate fell 34% in the lifestyle intervention group and by 18% in the Metformin group over 10 years compared with the placebo group.

The clear message is that lifestyle intervention is very effective, both in the short and long term, as participants were able to maintain it and some of their original 7 kg average weight loss over a 10 year period. We now know that an intensive lifestyle intervention is effective over 10 years and remains the best bet for prevention of diabetes, said Dr Anop Misra in an accompanying editorial. Further, during the extended phase, the Metformin group did as well as the intensive lifestyle intervention group which might possibly be due to the addition of

lifestyle interventions to the drug group, he speculates.

Results of trials that have tested lifestyle modification such as this study and a Finnish diabetes prevention study have been remarkably similar. Although this is a step in the right direction, it is not sufficient. Work remains to be done because after 10 years, half the people in the program had still developed diabetes.

Ref: <http://www.theheart.org/article/1017341.do>

### **CAM THERAPIES AND COMMUNICATION RECOMMENDATIONS FOR CLINICIANS**

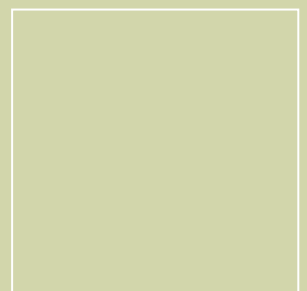
Guidelines for discussing CAM practice implications have been developed at the Nursing and Support Care Research at the Peter McCallum Cancer Centre and results broadcast on the Health Report ABC Radio National on 26 October.

The aim was to develop evidence-based guidelines to assist Oncology health professionals to have respectful, balanced and useful discussions with patients about CAM. No randomised controlled trials specifically addressing the methods or benefits of discussing CAM were identified.

Evidence-based guidelines are presented as a sequence of recommended steps. Given the concerns surrounding CAM use by conventional medical practitioners it is critical to encourage informed decision making about CAM and ultimately improve outcomes for patients, according to Dr Schofield.

CAM practitioners can assist informed decision-making regarding treatment options by empowering patients to work with oncology health practitioners.

Ref: Schofield et al, *Patient Educ Couns.* 2009 Sep 25.  
<http://www.ncbi.nlm.nih.gov/pubmed/19783116>



# YOUR COLLEGE

## STEPPING OUT IN 2009

ACNEM has been busier than usual this year with attendance at a number of outside events of like-minded organisations. In the most recent quarter, AIMA (the Australasian Integrative Medicine Association) held their annual Holistic Health Conference at the Novotel in St Kilda in October, where ACNEM once again had an exhibitor table that was visited by many delegates. Interest in ACNEM was high, especially following the talk by Prof Ian Brichtope, and we saw a number of delegates at our Auckland event in November who attended as a result of first attending the AIMA Conference.

The ASMI (Australian Self Medication Industry) Association Conference was held in Sydney in November and was attended by ACNEM CEO, Stephen Penman. ASMI is the association representing the interests of the consumer self-care products industry companies, many of whom of course, are well known to ACNEM. A host of key speakers including the Hon Nicola Roxon, MP, Minister for Health and Ageing and the Hon Mark Butler MP, Parliamentary Secretary for Health, and a large number of delegates made this an important area for future development of relationship.

The GPCE (General Practice Conference and Expo) ran its annual Melbourne event that same weekend and once again ACNEM and AIMA displayed their 'team spirit' and shared a display stand. The draw to win an ACNEM Primary course proved very popular again...the winner will be contacted shortly.

The annual Gawler Foundation conference was the third event that weekend and was a huge success with over 400 delegates in attendance. There was a lot of interest generated in ACNEM's literature and future course dates and we look forward to catching up with those practitioners at future ACNEM events.

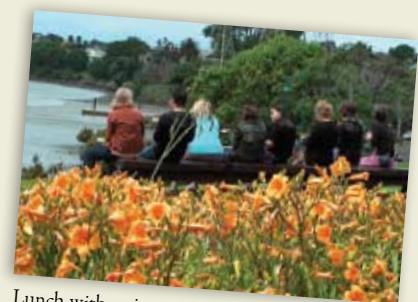
## ACNEM TRAINING AND AGM IN AUCKLAND

ACNEM's fifth and final training event for 2009 was held at the Waipuna Hotel in Auckland in November, re-establishing our presence in New Zealand. NZ practitioners demonstrated a strong interest in ACNEM training with exceptional attendance, exceeding the numbers we usually get to a Primary course in Australia! ACNEM was also pleased to host a free evening lecture for healthcare professionals in conjunction with our AIMA NZ colleagues, once again with record attendance. Dr Richard Coleman presented a fascinating lecture based on a practice-wide audit, "Vitamin D – the most cost effective medical intervention", discussing its clinical use for prevention and treatment of disease, including precautions in therapy. Thank you to everyone who participated to make this the most successful event of the year.

The ACNEM Annual General Meeting (AGM) was also held during the Auckland event and we thanked and said goodbye to Dr Sinclair Bode and Dr Karen Thompson who resigned from the Board during the year. Dr Emmanuel Varipatis and Dr Debbie Fewtrell have since been welcomed as new Board Members. Soon after, Dr Sinclair Bode was acknowledged by the Board by the granting of Honorary Life Membership for exceptional service to the College, making Dr Bode only ACNEM's 24th Life Member. There are so many people who have given exceptional service to ACNEM over the years, but it is especially gratifying once in a while to see someone so deserving formally recognised in this way.



Dr Richard Coleman presents a free seminar on Vitamin D



Lunch with a view



Panel Discussion



Dr Tane Taylor gives a traditional Maori welcome

## ACNEM BOARD AND FACULTY

ACNEM Board and Education Faculty members volunteer their time, expertise and energy to the College, in some cases, giving up nearly four weeks a year to lecture at ACNEM training events. We owe them a debt of thanks. To give some sense of the depth of knowledge and experience behind the ACNEM training you attend, the Board and Faculty members are listed here.

### The ACNEM Board for 2010 are:

Dr Gary Deed (President)

Dr Karel Hromek (Vice President)

Dr Tony Bartone (Treasurer)

Dr Jenny McKern (Secretary)

Dr Greg Emerson Dr Debbie Fewtrell

Dr Braham Rabinov Dr James Read

Dr Denis Rebic Dr Greg Schwarz

Dr Matt Shelton Dr Emmanuel Varipatis

### The Education Faculty are:

Dr Denis Rebic (Chair), MBBS, ABAAM, Dip ACNEM, Cert NEM  
Ann-Mary Hromek (Education Co-ordinator), RN, ND, MATMS

Dr Peter Baratosy, MBBS, PhD, Dip Acu, Dip Clin Hyp, FACNEM

Dr Gary Deed, MBBS, DipHerbMed, FACNEM

Dr Greg Emerson, MBChB, DipObs, DipDHM, FACEM, FACNEM

Dr Mervyn Garrett, MBBS, MACA, Specialist Allergist, FACNEM

Dr Kerry Harris, BMBS(Hons), FRACGP, BSc(Hons)

Dr Karel Hromek, BMed, BSc, FACNEM, FACRRM

Dr Carole Hungerford, BA, MBBS, FACNEM

Dr Richard Moore, BMed, BAppSc (Osteo), BSc (Hons), G.Dip (Acu)

Dr James Read, MBBS, Dip RANZCOG, FRACGP

Dr Matthew Shelton, MBChB, MRCGP, DRCOG, FCAAnaes DA

The ACNEM Board and Faculty are working hard to ensure that ACNEM increases its relevance and reach to the medical profession, to further the cause and uptake of NEM in Australia and New Zealand. We welcome your suggestions as to how we can better serve you.

## CHANGES AT AIMA

Following the AIMA AGM on 9 December, ACNEM would like to officially welcome Prof Kerryn Phelps (past President of the Australian Medical Association), as the new President of AIMA, and to thank Prof Avni Sali, the outgoing President, for his strong leadership of AIMA over the last two years, and his pioneering work for Integrative Medicine over many more years. We also welcome Dr Katherine Sevar as the new AIMA Vice President.

Regular readers of the ACNEM Journal will know that ACNEM works closely with AIMA on a range of issues important to our members, and right now it's very much a case of 'watch this space' as the benefits of this relationship to our members are expected to develop in coming years.

# FOOD FOR THOUGHT

*There is no medicine like hope, no incentive so great,  
no tonic so powerful as expectation of something tomorrow.*  
-Orison S. Marden

*We must not allow the clock and the calendar to blind us  
to the fact that each moment of life is a miracle and mystery.*  
-H. G. Wells

*I have not failed, I've just found 10,000 ways that won't work.*  
-Thomas Edison

*How far you go in life depends on you being tender with the  
young, compassionate with the aged, sympathetic with the striving  
and tolerant of the weak and the strong. Because someday in life  
you will have been all of these.*  
- George Washington Carver

*You don't get to choose how you're going to die. Or when. You can  
only decide how you're going to live. Now.*  
- Joan Baez

*Twenty years from now you will be more disappointed by the  
things you didn't do than by the ones you did do. So throw off the  
bowlines. Sail away from the safe harbor. Catch the trade winds in  
your sails. Explore. Dream. Discover.*  
- Mark Twain



# ACNEM TRAINING

**...INTEGRATING NUTRITIONAL & ENVIRONMENTAL MEDICINE  
INTO CLINICAL PRACTICE**



## ACNEM TRAINING IS...

### **Evidence and practice-based:**

- Practical and evidence-based, allowing you to start integrating nutritional therapeutics into your practice, immediately and safely. The training provides basic principles (often new to practitioners) and a framework to make sense of the plethora of information on non-orthodox treatments. It also leads to better identification of the underlying causes of disease, and to improved patient outcomes.
- Strongly referenced to the major medical literature, interactive and enjoyable with case studies and discussion; it includes the role of lifestyle, nutritional and environmental factors in health and disease. Comprehensive ongoing training, support, networking and a Fellowship Program are offered.

### **Time and cost-effective:**

Primary Course: 4 days, Thurs-Sun, 30 contact hours.  
2 day course, Thurs-Fri or Sat-Sun, 15 contact hours.

2010

**MARCH 11-14 - MELBOURNE**



Sebel Hotel, Albert Park

1. **Primary Course in NEM. ACNEM's foundation training in Nutritional and Environmental Medicine**  
(4 days, 11-14 March)
2. **The GUT - Nutritional Influences on Gastrointestinal Health and Disease including gut dysbiosis and permeability**  
(2 days, 11-12 March)
3. **Epigenetics & Nutrigenomics - Nature or Nurture?**  
The molecular basis of environmental and genetic interactions  
(2 days, 13-14 March)

### **Special events at the March training:**

**Cocktail Reception and Guest Speakers**

Friday 12 March 7pm-11pm

**Fellowship & Graduate Reunion Dinner**

Saturday 13 March 7pm-11pm

**To register your interest in any of these training programs,  
please visit [www.acnem.org](http://www.acnem.org), email [mail@acnem.org](mailto:mail@acnem.org) or phone (03) 9597 0363.**

**MAY 13-16 - FREMANTLE**

1. **Primary Course in NEM. ACNEM's foundation training in Nutritional and Environmental Medicine**  
(4 days, 13-16 May)
2. **Environmental Influences on Health and Disease including Toxicology and Multiple Chemical Sensitivity**  
(2 days, 13-14 May)
3. **Neurodegenerative Diseases and Cognitive Health, with focus on Alzheimers, Schizophrenia and Autism Spectrum Disorders** (2 days, 15-16 May)

**JULY 8-11 - MELBOURNE**

1. **Injectable Nutrients, including the safe and effective use of Vitamin C, B, Magnesium, Glutathione and Alpha-Lipoic Acid** (2 days, 8-9 July)
2. **Chelation Therapy, including indications, safety, protocols of treatment and chelating agents such as EDTA, DMSA, DMPS, GSH & IVC** (2 days, 10-11 July)

**Optional Certification in Chelation Therapy**  
(written and oral exam)

**AUGUST 12-15 - QUEENSTOWN, NZ**

1. **Primary Course in NEM. ACNEM's foundation training in Nutritional and Environmental Medicine**  
(4 days, 12-15 August)
2. **Cancer, "Every Third Person". What the experts say on biomedical influences on prevention and survival**  
(2 days, 12-13 August)
3. **Brain Health, from Autism and ADHD to Alzheimers - a biomedical approach** (2 days, 14-15 August)

**NOVEMBER 18-21 - SYDNEY**

1. **Primary Course in NEM. ACNEM's foundation training in Nutritional and Environmental Medicine**  
(4 days, 18-21 November)
2. **Scratches, Itches and Wheezes. Allergy, autoimmune, skin prick testing and dermatological conditions**  
(2 days, 18-19 November)
3. **Tired or Wired? ACNEM's popular training in Thyroid and Adrenal conditions**  
(2 days, 20-21 November)

