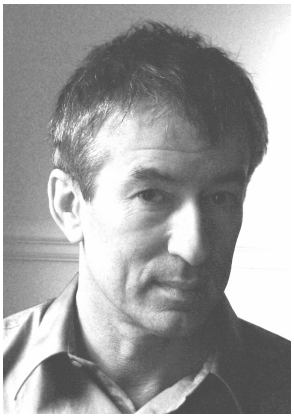

Dr Paul Clayton's Health Newsletter

Omega 3s good for bones as well as joints

The old story about fish oil being good for joints is proven; we know that if you take enough Omega 3s, inflammation and joint pain is reduced in rheumatoid arthritis (James et al '10) and, possibly, osteoarthritis; via a mechanism which prevents or reduces the destruction of cartilage in the affected joints (Wann et al '10).

Now there is evidence that fish oil also protects bone. Specifically, a daily dose of 300 ml of Omega 3 appears to prevent bone loss and indeed promote bone re-growth in post-menopausal women, when combined with aerobic, weight-bearing exercise (Tartibian et al '11).

This was an Iran/USA collaboration, and a fine example of how scientists can work together even while their idiot political masters bicker and strut. The researchers found that the fish oil - exercise combination reduced blood levels of the important inflammatory markers IL-6 and TNF-alpha by 40% and 80% respectively; and increased bone mineral density (BMD) by up to 15% in the lumbar vertebrae, and 19%.at the femoral neck.

"We become more depleted in cell-renewal vitamins D, K2, C, B6, and cell-breakdown inhibitors such as flavonoids and Omega3."

Fish oil alone cannot do these things, and neither can exercise. As the scientists themselves pointed out, *"The combination of PUFA supplementation with aerobic exercise provided benefits on inflammation and bone density over exercise alone or supplementation alone."*

I was initially very sceptical about these hugely positive results, as the increases in BMD were achieved within a mere 6 months. This is very short time as far as bone is concerned; it is a very slowly metabolising tissue, which is why most trials of this sort run for a minimum of 1 year, and preferably longer. But when I thought about the mechanisms of action of the combined fish oil and exercise, it began to seem rather more plausible.

BMD (roughly, the amount of calcified tissue in the bone), is a dynamic entity. It is continually being broken down and replaced, and the main reason why we tend to lose bone as we move inexorably towards old age is that we move into a metabolic and lifestyle pattern that favours breakdown, or catabolism.

We become progressively more depleted in anabolic factors such as vitamins D, K2, C, B6 and a handful of minerals, and we take less exercise. At the same time we become more depleted in factors which slow breakdown, such as the anti-inflammatory flavonoids and Omega 3s. This is crucial, as the breakdown of bone involves inflammatory processes.

The Iran/USA team was inadvertently targeting both sides of the equation. The fish oils were, via their anti-inflammatory actions, reducing bone breakdown. The exercise, by directing bone-forming cells to the affected areas of bone, was driving regeneration, and may also have up-regulated the body's own anti-oxidant and anti-inflammatory enzymes.

As the exercise involved walking and jogging three times a week, it was increasing appetite (you may have noticed that exercise does this), and therefore increasing intakes of a range of vitamins and minerals. Sadly, the scientists did not record dietary changes in their subjects, but these will have occurred and will also have contributed to the positive outcomes.

I shall wait to see if other groups are able to reproduce these findings, and would recommend to any scientists interested in developing this provocative work that they consider adding the other key anabolic inputs to the mix. I would add that the Iranian group have been studying (and publishing) the anti-inflammatory effects of fish oils in various exercise models for a few years (Tartibian et al '09, '10, '11b), and appear to know what they are doing!

Inside this issue:

New Year—New Format Newsletter

- Easier to read
- All references on the back page
- More up-to-date design
- Same pioneering and insightful approach
- Inimitable no-holds-barred style

Below the Salt, Above the Sugar

New salt-reducing flavouring "Salty-R"

New sweetener "PureFruit"

I wrote about the harm caused by excess sodium consumption as recently as the 2011 Summer newsletter, but there is some good news on the way. Horphag, a Swiss company better known for its longstanding interest in the pine bark extract pycnogenol, has launched a new salt-reducing food flavour.

Called **Salty-R** (they missed a trick – surely Salty-C would have been better), it was a wow at the Supply Side West trade show in Las Vegas and will be appearing in a range of processed foods as early as mid-summer this year..

Extracted from a natural (vegetable) source, this compound acts on taste receptors on the tongue and amplifies the taste of salt; thus enabling the actual amount of sodium in foods to be reduced by as much as 30%; and when combined with potassium chloride, up to 50 percent.

Salty-R is a major threat to the sodium chloride industry, so expect dirty tricks. Look out for an anti-Salty-R campaign directed and funded by the usual suspects, and fronted by the usual scientifically illiterate health activists.

Look out, too, for **Monk's Fruit Extract**, the latest (and by my taste buds the best) sugar substitute to hit the market. Tate and Lyle acquired the European rights for this, and have branded it as **PureFruit** sweetener.

At zero calories, 200 times the sweetness of sugar and at least 300 years of use in the Far East, it should be a smash.

But here too, it will probably not be long before the same luddites who railed serially against saccharin, cyclamate, acesulfame-K, aspartame and sucralose, steam into view!

"We need to reduce our salt and sugar consumption, so don't be put off by dirty tricks from sodium chloride and sugar producers—these safe, good-tasting, naturally sourced substitutes are to be welcomed."

New Body, Anybody?

Some folk make resolutions at New Year, and although I am too old and too cynical to indulge in such things, I am always happy to help those who do. An old friend has decided that he wants a new body, with less fat and more muscle, and he asked me whether nutrition might help him to achieve this.

Here is what I suggested …

1. Take more **exercise** in a form that you enjoy, and can therefore more easily maintain.
2. Take a **capsaicin** or **fucoxanthin** supplement. These compounds are thermogens, they raise the body's thermostat so that you burn more calories (Kawabata et al '06). This tends to increase the appetite, so they are best combined with a natural appetite suppressant such as Slimaluma (Kuriyan et al '07, Kamalakkanan et al '11), an extract of the food plant *Caralluma fimbriata*.
3. Use the fenugreek extract **Testofen**, which raises levels of free testosterone and enhances muscle building (Wilborn et al '10). [This was covered in a previous newsletter].
4. Add a protein product enriched with the **amino acid l-leucine**.

This last point derives from a recent US Army study which found that l-leucine supplements taken during endurance exercise increased muscle protein synthesis by as much as 33% (Pasiakos et al '11).

This is not resistance exercise, where lifting heavy weights is used specifically to build muscle; but the type of exercise that many more people do, such as running, jogging, dancing or cycling. These exercise activities do not generally build muscle bulk; rather, they can lead to loss of muscle volume.

Leucine supplements appear to prevent potential muscle loss by enhancing muscle protein synthesis in the recovery phase after exercise. For anyone interested, whey protein products offer better leucine payloads than soy, milk or egg-based products.

Take more exercise in a form that you enjoy.

Auto-Immune Diseases—Clues and Therapies

The autoimmune diseases are a riddle, wrapped in a mystery, inside an enigma. Worryingly, rheumatoid arthritis, Graves' Disease, Type 1 diabetes, multiple sclerosis and other less common forms such as Hashimoto's thyroiditis, are all increasing at the rate of a couple of percent a year (Miller '05) .

Women are disproportionately affected, being three times more likely to acquire an autoimmune disease than are men, although when a man acquires an autoimmune disease it is likely to be more severe (Klein '00) . Unfortunately for patients of both sexes, current treatments are both ineffective and toxic.

We know that certain pathogens, when they infect individuals who carry certain risk-denoting genes, can increase the risk of an auto-immune disease developing (de Abreu et al '11); and that the female hormones enhance this risk (Regner & Lambert '02, Fairweather & Rose '02, Fairweather & Rose '04).

There is persuasive evidence that extreme stress may act as a trigger, even in those who are not genetically susceptible (Sonino et al '93), but nobody knows why all the autoimmune diseases are increasing. Recent findings, however, have contributed to a new theory of autoimmunity which fits at least some of the facts, and may form the basis for new preventative regimes or treatments; which may include sunbathing.

One of the worst pieces of health advice our governments have given us (and there have been plenty) was to minimise our exposure to sunlight. This was due to erroneous ideas about sunlight and skin cancer—for example, outdoor workers have **less** melanoma than office workers (Rivers '04, Levell et al '09); and it has contributed to widespread D depletion and even deficiency; which doctors initially experience as an increasing incidence of osteomalacia and rickets (Moore '02, Robinson et al '06, Irish Health '06, NetDoctor '11). But there are other forms of D-related Disease. Over the last decade or so it has become apparent that a low D status also increases the risk of autoimmune disorders.

Although the majority of cases of low D status are directly due to inadequate exposure to sunlight, there is a smaller group of people who suffer from low D even when they do sunbathe because they have genetic polymorphisms (variants) which make them less sensitive to circulating D; and these too are more prone to autoimmune diseases (Xhou et al '09). This latter group has presumably increased in line with overall population increase, but the numbers of folk with healthy genes who have learned to shun the sun have increased hugely.

The association with low D status is particularly strong with multiple sclerosis (de Abreu et al '11); but there is an association with Type 1 diabetes and rheumatoid arthritis, although viruses could also be involved here (Jankosky et al '11).

Recent work is beginning to show how low D status might contribute. The vitamin (it's more accurately a hormone) has an essential role in coordinating and regulating the functions of both the innate and the adaptive immune systems (Bock et al '11, Fabri et al '11). For example, a good D status has been shown to thus increase resistance to TB (Fabri et al '11); conversely a poor D status has been shown to lead to numerous immunological issues (Haroon & Fitzgerald '11). More specifically relating to autoimmune disease, low D status degrades the performance of invariant natural killer (iNKT) cells, which is critical as these cells suppress autoimmune problems (Cantorna et al '11).

This mechanism appears to explain how low D predisposes to autoimmune illness, and it has encouraged clinicians to treat autoimmunity with high doses of D, or D analogues; but so far they have had only mixed success (Fletcher et al '11). Moreover, the positive clinical responses to D may be due to immunological improvements, but they could also be caused by other, non-specific effects of D, as it is an anti-inflammatory agent (Chapkin et al '09) [see box R].

Evidence is beginning to accumulate that to achieve maximal protection against autoimmune illness, it may be best to ensure good D status in the womb, and the first few years of life. D is very important in modifying and supporting the developing immune system (Cantorna et al '11, de Abreu et al '11), and if sufficient D is not present during the formative years it may not be as effective when used later in life, as the crucial iNKT cells may never fully recover (Cantorna et al '11).

It is not yet possible to be conclusive, but it looks as if the best way to reduce the incidence of autoimmune disease is to encourage women of child-bearing age to sunbathe; or to supplement with D3, as they already do with folic acid. [D2, which is currently being re-evaluated, would not be recommended (Bjelakovic et al '11) .]

Auto-Immune Diseases are increasing

- **Multiple Sclerosis**
- **Type 1 Diabetes**
- **Rheumatoid Arthritis**
- **Graves' Disease**

linked to Vit D depletion?

Spend more time in the sun.

"I have used the powerfully anti-inflammatory combination of Omega 3s, vitamin D and mixed flavonoids to treat a number of auto-immune conditions, with a range of outcomes which included cases of total remission of symptoms."

The Dr Paul Clayton Health Newsletter describes developments in the new field of pharmaco-nutrition, where nature and science are combined to offer non-drug solutions to degenerative disease.

The newsletters are intended to increase knowledge and awareness of health issues and are for information only. No health claims for specific products are made or intended and the information should not be used as a substitute for medical advice.

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Vitamin E not Just an Anti-Oxidant

Many compounds act as antioxidants in the body. There are the well-known antioxidant vitamins and carotenoids, the slightly less well-known xanthophylls, flavonoids and isoflavones; and hundreds of other, more 'obscure' compounds such as urea, bilirubin, estrogen, albumin … the list is a long one, and the keen observer will have noticed that all of these have important functions in the body which are nothing to do with oxidation.

The **flavonoids** are powerful anti-inflammatory agents, the **carotenoids** and **xanthophylls** are re-differentiating agents (ie. they protect against cancer), **urea** is a waste product produced in the body when proteins are broken down, **bilirubin** is a breakdown product of haemoglobin, **(o)estrogen** is a sex hormone and **albumin** is a key protein in the blood.

There are so many anti-oxidants, and so many of these (if not all) have specific and critically important non-anti-oxidant functions, that we really have to re-think the whole concept of what an antioxidant is. For example, vitamin E has anti-oxidant properties, but until now we had no idea what its essential role in the body was; the essential role that makes it a vitamin. Now, at last, vitamin E's specific functionality is beginning to emerge.

Three scientists at the University of Georgia in the USA (Howard et al '11) have found that vitamin E has the ability to help repair tears in cell membranes; an essential function indeed, as progressive tears would otherwise lead to progressive cellular malfunction and death. Their work supports the use of vitamin E in diabetes, and the inclusion of vitamin E in any and all pharmaco-nutritional support programmes.

NB. There are **8 forms** of vitamin E, all of which appear to have slightly different functions (Nowak et al '11, Shin-Kang et al '11).

Opt for programmes which contain all 8 forms of natural E (ie. in mixed tocopherols and tocotrienols), rather than synthetic E.

REFERENCES

p1 Omega 3s

James M, Proudman S, Cleland L. Fish oil and rheumatoid arthritis: past, present and future. Proc Nutr Soc. 2010 Aug;69(3):316-23.

Wann AK, Mistry J, Blain EJ, Michael-Titus AT, Knight MM. Eicosapentaenoic acid and docosahexaenoic acid reduce interleukin-1β-mediated cartilage degradation. Arthritis Res Ther. 2010;12(6):R207.

Tartibian B, Maleki BH, Kanaley J, Sadeghi K. Long-term aerobic exercise and omega-3 supplementation modulate osteoporosis through inflammatory mechanisms in post-menopausal women: a randomized, repeated measures study. *Nutrition & Metabolism* 2011, 8:71 doi:10.1186/1743-7075-8-71

Tartibian B, Maleki BH, Abbasi A. The effects of ingestion of omega-3 fatty acids on perceived pain and external symptoms of delayed onset muscle soreness ... Clin J Sport Med. 2009 Mar;19(2):115-9.

Tartibian B, Maleki BH, Abbasi A. The effects of omega-3 supplementation on pulmonary function of young wrestlers during intensive training. J Sci Med Sport. 2010 Mar;13(2):281-6.

Tartibian B, Maleki BH, Abbasi A. Omega-3 fatty acids supplementation attenuates inflammatory markers after eccentric exercise in untrained men. Clin J Sport Med. 2011 Mar;21(2):131-7.

p2 New Body

Kamalakkannan S, Rajendran R, Venkatesh RV, Clayton P, Akbarsha MA. Antiobesogenic and Antiatherosclerotic Properties of Caralluma fimbriata Extract. J Nutr Metab. 2010;2010:285301.

Kawabata F, Inoue N, Yazawa S, Kawada T, Inoue K, Fushiki T. Effects of CH-19 Sweet, a Non-Pungent Cultivar of Red Pepper, in Decreasing the Body Weight and Suppressing Body Fat Accumulation by ... Biosci Biotechnol Biochem 70 (12):2824-2835, 2006

Kuriyan R, Raj T, Srinivas SK, Vaz M, Rajendran R, Kurpad AV. Effect of Caralluma fimbriata extract on appetite, food intake and anthropometry in adult Indian men and women. Appetite. 2007 May;48(3):338-44.

Pasiakos SM, McClung HL, McClung JP,.Margolis L M, Andersen NE, Cloutier GJ, Pikosky MA, Rood JC, Fielding RA, Young AJ. Leucine-enriched essential amino acid supplementation during moderate steady state exercise enhances ... Am J Clin Nutr. *2011.* 94: 809-818

Wilborn C, Taylor L, Poole C, Foster C, Willoughby D, Kreider R. Effects of a purported aromatase and 5α-reductase inhibitor on hormone profiles in college-age ... Int J Sport Nutr Exerc Metab. 2010 Dec;20(6):457-65.

p3 Auto-Immune

Bjelakovic G, Gluud LL, Nikolova D, et al. Vitamin D supplementation for prevention of mortality in adults. Vitamin D3 but not D2 reduces mortality – meta-analysis July 2011. Cochrane Database Syst Rev. 2011 Jul 6;(7):CD007470.

Fernandes de Abreu DA, Landel V, Féron F. Seasonal, gestational and postnatal influences on multiple sclerosis: the beneficial role of D supplementation... J Neurol Sci. 2011 Dec 15;311(1-2):64-8.

Bock G, Prietl B, Mader JK, Höller E, Wolf M, Pilz S, Graninger WB, Obermayer-Pietsch BM, Pieber TR. The effect of vit D supplementation on peripheral regulatory T cells and β cell function in healthy humans. Diabetes Metab Res Rev. 2011 Nov;27(8):942-5

Cantorna MT, Zhao J, Yang L. Vitamin D, invariant natural killer T-cells and experimental autoimmune disease. Proc Nutr Soc. 2011 Oct 14:1-5. [Epub ahead of print]

Fabri M, Stenger S, Shin DM, et al. Vitamin D is required for IFN-gamma-mediated antimicrobial activity of human macrophages. Sci Transl Med. 2011 Oct 12;3(104):104ra102.

Fairweather D, Rose NR. Type I diabetes: virus infection or autoimmune disease? Nat Immun. 2002;3:338–40.

Fairweather D, Rose NR. Women and autoimmune diseases. Emerg Infect Dis. 2004 Nov;10(11):2005-11. Review.

Fletcher JM, Basdeo SA, Allen AC, Dunne PJ. Therapeutic Use of Vitamin D and its Analogues in Autoimmunity. Recent Pat Inflamm Allergy Drug Discov. 2011 Nov 28. [Epub ahead of print]

Irish Health. http://www.irishhealth.com/article.html?id=9693

Haroon M, Fitzgerald O. Vitamin D and its emerging role in immunopathology Clin Rheumatol. 2011 Oct 22. [Epub ahead of print]

Jankosky C, Deussing E, Gibson RL, Haverkos HW. Viruses and vitamin D in the etiology of type 1 diabetes mellitus and multiple sclerosis. Virus Res. 2011 Nov 20. [Epub ahead of print]

Klein SL. The effects of hormones on sex differences in infection: from genes to behavior. Neurosci Biobehav Rev. 2000;24:627–38.

Levell NJ, Beattie CC, Shuster S, Greenberg DC. Melanoma epidemic: a midsummer night's dream? Br J Dermatol. 2009 Sep;161(3):630-4.

Miller, Fred. Director, Environmental Autoimmunity Group, NIH. Personal Communication.

Moore J. Rickets due to vitamin D deficiency; Why is it re-emerging in the UK? Community Practitioner 2002, 75:4:144-146

NetDoctor 11. http://www.netdoctor.co.uk/infections/the-return-of-health-problems-from-the-past.htm

Regner M, Lambert P-H. Autoimmunity through infection or immunization? Nat Immun.2001;2:185–8.

Rivers JK. Is there more than one road to melanoma? Lancet. 2004 Feb 28;363(9410):728-30. Review.

Robinson PD, Högler W, Craig ME, et al. The re-emerging burden of rickets: a decade of experience from Sydney. Arch Dis Child. 2006 Jul;91(7):564-8.

Sonino N, Girelli ME, Boscaro M, Fallo F, Busnardo B, Fava GA. Life events in the pathogenesis of Graves' disease. A controlled study. Acta Endocrinol (Copenh). 1993 Apr;128(4):293-6.

Zhou H, Xu C, Gu M. Vitamin D receptor (VDR) gene polymorphisms and Graves' disease: a meta-analysis. Clin Endocrinol (Oxf). 2009 Jun;70(6):938-45.

p4 Vitamin E

Howard AC, McNeil AK, McNeil PL. Promotion of plasma membrane repair by vitamin E. Nat Commun. 2011 Dec 20;2:597.

Nowak G, Bakajsova D, Hayes C, Hauer-Jensen M, Compadre CM. γ-Tocotrienol Protects Against Mitochondrial Dysfunction and Renal Cell Death. J Pharmacol Exp Ther. 2011 Oct 31. [Epub ahead of print]

Shin-Kang S, Ramsauer VP, Lightner J, Chakraborty K, Stone W, et al. Tocotrienols inhibit AKT and ERK activation and suppress pancreatic cancer cell proliferation by suppressing the ErbB2 pathway. Free Radic Biol Med. 2011 Sep 15;51(6):1164-74.