Dr Paul Clayton's Health Newsletter

Health and Fatness

We are getting fatter as a population, but we have not reached **peak fat**. If we let out our waistbands, and if our politicians cave in to the brazen attempt by the USA to drag our nutritional standards lower (via the Transatlantic Trade and Investment Partnership TTIP), our supermarkets will fill up with even more junk, and mobility scooters ferrying the kind of 400lb (28 stone; 180kg) victims you can see in any US supermarket.

This newsletter focuses on fat and the fat-soluble nutrients, which are, collectively, an important part of health and health prospects. But let us start by asking why being overweight is bad for us. Or is it?

The simple message from the health authorities that overweight = bad is, like all government propaganda, an over-simplification. Being overweight is NOT always bad for your health; it depends on what kinds of food you eat on the way to becoming fat, and where on the body you store that fat – think of apples and pears.

Android (male-pattern) fatness—BAD

It has long been thought that fat deposited in the abdomen is more hazardous than fat deposited around the hips and thighs. The latest view is slightly more complex, and singles out android rather than abdominal (around the waist) adipose tissue.

Android (male-pattern) fatness refers to fat deposited around the liver and heart, sites that are significantly above the waist. This is the fat that is being highlighted in triggering metabolic disorder, chronic inflammation and cardiovascular mortality (Aucouturier et al '09, Kang et al '11, Samsell et al '14, Fabbrini et al '09).

Gynoid (female-pattern) fatness—BETTER

In contrast, gynoid adiposity (fat deposited in the hips and thighs, a female pattern) is considered to be either neutral or potentially protective (Terry et al '91, Danforth 2000). This is in line with a recent review of global data that found heart disease to be behind most of the excess deaths documented in adult men over the age of 40 (Solé-Auró et al '15, Crimmins et al '15, Beltrán-Sánchez et al '15); and with new research indicating that TNFα, an inflammatory substance involved in both liver and heart damage, is associated with android and central rather than gynoid fat (Garaulet et al 2000, Winkler et al '99).

Fat colour is important too

The location of body fat is important, but so is its colour. Healthy diets contain a range of fat-soluble micronutrients including tocopherols and tocotrienols (there are 8 of these, and they collectively make up vitamin E); carotenoids and xanthophylls, vitamin D, various polyphenols such as those in olive oil, and the valuable omega 3 fatty acids from oily fish. These compounds accumulate in our fat depots, staining the fat in shades of brown and orange.

At the same time, they reduce chronic inflammation in the fat and prevent it from releasing the so-called adipocytokines, fatty substances that trigger inflammation in adjacent tissues such as the liver and heart. Healthy diets create coloured, non-inflammatory fat; the typical Standard American Diet (the SAD diet), full of empty calories but depleted or deficient in micronutrients, produces large amounts of white and therefore inflammatory fat.

As D and other micronutrients such as vitamins A, E, K and the carotenoids are lipophilic (ie. fat-soluble), they concentrate in adipose tissue. This is important, because adipose tissue is a not only a site for storing spare calories but also a highly active endocrine organ.

In normal weight individuals, adipose tissue is involved in metabolic homeostasis, helping to maintain a balanced state or equilibrium; soaking up spare calories when they are in excess, and releasing them when needed.

In obesity, however, it becomes a pathological organ, inflamed and causing chronic inflammation elsewhere in the body (Olefsky et al '10, Gregor et al '11).

continued on p2

Inside this issue:

The Paul Clayton Health Newsletter

- **Easy to read**
- **All references on the back page**
- **Up-to-date news and design**
- **Pioneering and insightful approach**
- **Inimitable no-holds-barred style**

continued from p1

The inflammatory process in fat tissue is modified and subdued by fat-soluble "lipophile" micronutrients such as vitamins A, E, K and D, carotenoids, polyphenols, xanthophylls, and omega 3.

This inflammatory process is modified and subdued by a diet containing adequate amounts of the lipophiles, all of which have now been shown to exert damping, anti-inflammatory effects inside adipose tissue (Landrier et al '12). Fat-soluble polyphenols such as the secoiridoids in olives appear to do exactly the same thing (Ibarra et al '11).

As well as their anti-inflammatory effects, this interesting and important group of micro- and phyto-nutrients influences gene expression, and through this generates many beneficial effects on their target tissues. Most if not all of these lipophiles are capable of killing and/or switching off cancer cells, and many of them seem to be able to inhibit the growth of adipose tissue (Ibarra et al '11, Landrier et al '12).

Now we come back to the terrible, depleted modern diet, stuffed with empty calories and depleted or deficient in the lipophilic micro-nutrients (and all the other micronutrients). This dangerous combination is creating too many people with too much adipose tissue; and not enough of the damping lipophiles (Kimmons et al '06, Garcia et al '09).

As a result, their fat is in the fire, and it is inflaming the rest of their bodies. This is serious, as chronic inflammation is now seen as THE cause of degenerative disease.

You can actually see this during surgery. When well-fed vegetarians are cut open, their fatty tissues are coloured from yellow to orange, which is the hallmark of healthy fat. The fat inside junk food enthusiasts, which is typically pearl to pale yellow, is toxic and pro-inflammatory.

In short, all fat is not created equal. This explains why some folk can carry excess weight and remain healthy, while others suffer all manner of health problems.

Traditional fishing in the Arctic

The Inuit diet contained other actives—not just Omega 3—from the algae consumed by the fish they ate.

Fish oil alone doesn't work

Taking fish oil on its own as a magic-bullet single supplement just doesn't work.

Clinical trials have been generating terrible results lately. These include a clutch of major studies which looked at the cardio-protective effects of fish oil (JELLIS, GISSI-HF, ALPHA -OMEGA, OMEGA, SU.FO.OM, ORIGIN, CART), all of which failed to show any benefits. More recently AREDS-2, which examined the ability of fish oil to protect the brain from ageing, also failed (Chew et al '15). Specifically, AREDS-2 showed that one gram per day of EPA plus DHA omega-3s did not slow normal age-related cognitive decline in 4,000 subjects over the age of 70.

This trial was set up to confirm or refute preliminary data indicating that a diet rich in oily fish was good for the brain (Cederholm et al '13), but as the intervention failed to show any positive effect at all, the story that fish oil alone protected the brain now looks like a busted flush. The idea that fish oil on its own can protect against the development and progression of Alzheimer's disease has been similarly demolished (Wu et al '15).

It is all very reminiscent of the ideas people used to have about beta carotene being protective against lung cancer; preliminary findings showed that a diet rich in beta carotene was cancer protective, but when this idea was put into a formal trial it became clear that beta carotene supplements INCREASED the risk of cancer (the huge and expensive ATBC and CARET studies both showed negative effects).

When a strong initial association between diet and health is contradicted by the results of a prospective, randomized and placebo-controlled clinical trial, this generally means only one thing; we tested the wrong dietary ingredient. So it is with fish oil ….

Go back to the Inuit diet

When you go back to the start of the story – the Inuit diet – you can see right away that they ate a diet containing many more actives than are found in any fish oil capsule.

One of those actives is one of the rare polyphenols that is soluble in oil and fat, and which is produced by the same cold water algae that produce the omega 3s that are consumed by krill, fish, marine mammals and eventually the Inuit. This polyphenol is a treasure, as it is not only the best antioxidant for fish oils yet discovered (ie Wang '09), it is also a powerful anti -inflammatory compound in its own right (Dutot et al '12, Yang et al '14).

– combine with polyphenols

Perhaps even more relevant is the fact that whereas the commonly used antioxidant vitamin E not only fails to prevent the oxidation of ingested omega 3 fatty acids even when used at high doses (Allard et al '97), but can even increase the oxidation of fish oils (Tirosh et al '15), the oil-soluble polyphenols provide the anti-oxidant protection that nature originally 'designed' them for (Clayton & Ladi '15).

The lesson is clear. The pharmaceutical mind-set of 20th century medics and industrialists has led us all, once again, down a long blind alley costing too much money and too many lives. Diet is complex, and any attempts to reduce it to a single nutrient are inevitably doomed to failure.

To gain the original Inuit's relative freedom from degenerative disease, fish oil alone is not enough.

But if you combine it with polyphenols and/or virgin olive oil containing oil-soluble polyphenols (thus combining the best of the Inuit and the Mediterranean diets), chronic inflammation is effectively stopped in its tracks.

I have personally seen this in over 400 cases, and the combination will shortly face the ultimate test of a randomized and prospective clinical trial of its own.

Role of Vitamin E

Of course vitamin E is still needed. It does not appear to be as effective as the lipophilic polyphenols in protecting fish oil as it is consumed, absorbed and then ferried through the blood stream to the tissues where it will be built into cell membranes. It does, however, play a critical role in protecting the fatty acids once they have been built into those cell membranes, and it is involved in the repair of damaged cell membranes (Howard et al '11, Labazi et al '15).

This means that as more PUFAs (poly-unsaturated fatty acids) are consumed and built into cell membranes, it becomes important to increase the intake of vitamin E; otherwise the symptoms of vitamin E deficiency may begin to emerge. This is a real concern because up to 90% of consumers are consuming insufficient levels of vitamin E (McBurney et al '15). It becomes an even greater concern for consumers taking poorly formulated fish oil capsules that may deliver some omega 3s, but not enough vitamin E (and none of the critical polyphenols).

One of the reasons vitamin E depletion has become so common is poorly designed research (in my view) that suggested that vitamin E might increase the risk of prostate cancer (SELECT). At around the same time, other work indicated that fish oil might also increase the risk of this cancer (Brasky et al '14). While the Brasky paper has been thoroughly refuted, due to its gross sampling error, the lingering impact of the SELECT trial undoubtedly hit vitamin E sales. Now, however, new research is starting to debunk that idea too.

Confusion between vitamins E and K

At a recent symposium in Stuttgart's University of Hohenheim, a fascinating new theory was aired which focused on the confusion between vitamins E and K, which are so similar that they can be confused when taking measurements of biological tissues. It was suggested that high doses of vitamin E can lead to reductions in vitamin K levels, and it might have been the changes in vitamin K that caused the increased risk in cancer. As K2 intakes are already low, this suggests that fish oil, vitamin E and vitamin K should always be taken together – with selenium, of course, as this trace element works in tandem with vitamin E.

So here is another argument against treating nutrients as if they were drugs; an argument against monotherapy—a pharmaceutical idea—and for integrated nutritional interventions.

If you take fish oil, how much vitamin E should you wash it down with?

The first serious attempt to work out this equation has recently been performed by a team from DSM, one of the biggest fish oil producers, working with the well-respected Professor Philip Calder. According to their calculations, the daily requirement for vitamin E for anyone taking a typical dose of PUFAs ranged from 12 to 20 mg/day (Raederstorff et al '15).

Nutrition is extremely complicated, and sadly, the supplement business is still selling badly designed and possibly counter-productive products.

The pharmaceutical idea of monotherapy is not appropriate for nutrients—fish oil, vitamin E, vitamin K and selenium should be taken together in an integrated intervention.

It's important to take 12 - 20 mg of Vitamin E alongside a typical daily dose of PUFAs (fish oil).

The Dr Paul Clayton Health Newsletter describes developments in the 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.

Check out more free health information at

drpaulclayton.com
healthdefence.com

Videos too on Dr Clayton's YouTube channel

youtube.com/user/DrPaulClayton

Published in association with Uni-Vite Healthcare Ltd, producer of NutriShield, ImmunoShield and JointShield.

Vitamin K and calcium

Vitamin K, and especially vitamin K2, has an important role in maintaining healthy calcium levels in various tissues. Specifically, it is one of the keys to keeping calcium and magnesium in the bones, where it is wanted, and preventing these minerals from building up in the soft tissues where they are definitely not wanted.

Vascular calcification is a major problem in diabetes, and drives both vascular and renal disease. It is particularly prevalent when the picture is complicated by chronic kidney disease (CKD), which is relatively common in diabetics.

The Rotterdam group set up by Kees Vermeer and now led by Leon Schurgers, has done much of the research on K2. They have just published a new paper in which they showed that high dose K2 was particularly effective, in a pre-clinical model of CKD, at preventing calcification in the aorta and myocardium (Schurgers et al '15).

These are the two tissues most often involved in this kind of calcification, and as the researchers were also able to show how K2 was exerting its beneficial effect, the case for using high dose K2 routinely in diabetics and patients with chronic kidney disease (and osteoporosis) now looks very strong.

But not on its own!

Vitamin D and limb strength

Vitamin D supplementation significantly increases upper and lower limb strength, a review of seven trials has found.

Previous research on vitamin D has focused on supplementation and muscle strength in frail, deficient and elderly women. This latest review – compiled by researchers at Queen Mary University of London – was the first systematic summary of the impact on muscle strength in young, healthy individuals and included data from 310 adults with an average age of between 21.5 and 31.5 years.

The seven trials, which included 67% females, lasted from four weeks to six months while dosages ranged from 4,000 international units (IU) per day to 60,000 IU per week.

"This review has found that vitamin D3 supplementation improves upper and lower limb muscle strength in a healthy, adult, athletic and non-athletic population between the ages of 18 and 40," the authors wrote in the Journal of Science and Medicine in Sport.

The two studies that reported the most significant results employed total dosages between 60,000 and 14,000 IU of vitamin D per week for six to four months.

That said, further research is still needed to clarify its effect on muscle power, endurance and maximum strength.

REFERENCES

P1-2 Health and Fatness

Aucouturier J, Meyer M, Thivel D, Taillardat M, Duché P. **Effect of Android to Gynoid Fat Ratio on Insulin Resistance in Obese Youth**. Arch Pediatr Adolesc Med. 2009 Sep;163(9):826-31.

Beltrán-Sánchez H, Finch CE, Crimmins EM. **Twentieth century surge of excess adult male mortality**. Proc Natl Acad Sci U S A. 2015 Jul 6. pii: 201421942.

Crimmins EM. **Physiological Differences Across Populations ...** J Popul Ageing. 2015 Mar;8(1-2):51-69.

Danforth E Jr. **Failure of adipocyte differentiation causes type II diabetes mellitus?** Nat Genet 2000;26 (1) 13

Fabbrini E et al. (2009) **Intrahepatic fat, not visceral fat, is linked with metabolic complications of obesity**. Proc Natl Acad Sci U S A 106: 15430–5.

Garaulet M et al. **Anthropometric, computed tomography and fat cell data in an obese population** Eur J Endocrinol. 2000 Nov;143(5):657-66.

Garcia OP, Long KZ, Rosado JL. **Impact of micronutrient deficiencies on obesity**. Nutr. Rev. 2009;67:559–572.

Gregor MF, Hotamisligil GS. **Inflammatory mechanisms in obesity**. Annu Rev Immunol. 2011; 29():415-45. (Review)

Ibarra A, Bai N, He K, Bily A, Cases J, Roller M, Sang S. **Fraxinus excelsior seed extract limits weight gains and hyperglycemia in high-fat diet-induced obese mice.** Phytomedicine. 2011 Apr 15;18(6):479-85.

Kang SM, Yoon JW, et al. **Android Fat Depot Is More Closely Associated with Metabolic Syndrome than Abdominal Visceral Fat in Elderly People**. PLoS One. 2011;6(11):e27694.

Kimmons J.E., Blanck H.M., Tohill B.C., Zhang J., Khan L.K. **Associations between body mass index and the prevalence of low micronutrient levels among US adults**. MedGenMed.2006;8:59.

Landrier J-F, Marcotorchino J, Tourniaire F. **Lipophilic Micronutrients and Adipose Tissue Biology**. Nutrients 2012 Nov; 4(11): 1622–1649.

Olefsky JM, Glass CK. **Macrophages, inflammation, and insulin resistance.** Annu Rev Physiol. 2010; 72():219-46.

Samsell L, Regier M, Walton C, Cottrell L. **Importance of Android/Gynoid Fat Ratio in Predicting … Disease Risk ...** J Obes. 2014;2014:846578.

Solé-Auró A, Michaud PC, Hurd M, Crimmins E. **Disease incidence and mortality among older Americans and Europeans**. Demography. 2015 Apr;52(2):593-611.

Terry RB, Stefanick ML, Haskell WL, Wood PD. **Contributions of regional adipose tissue depots to plasma lipoprotein concentrations ...effects of thigh fat**. Metabolism 1991;40 (7) 733- 740

Winkler G, Lakatos P, et al. **Elevated serum TNF-alpha level as a link between endothelial dysfunction and insulin resistance in normotensive obese patients**. Diabet Med. 1999 Mar;16(3):207-11.

P2-3 Fish oil

Allard JP, Kurian R, et al. **Lipid peroxidation during n-3 fatty acid and vitamin E supplementation in humans**. Lipids. 1997 May;32(5):535-41.

Cederholm T, Salem N Jr, Palmblad J. **ω-3 fatty acids in the prevention of cognitive decline in humans**. Adv Nutr. 2013 Nov 6;4(6):672-6.

Chew EY, Clemons TE, et al; Age-Related Eye Disease Study 2 (AREDS2) Research Group. **Effect of Omega-3 Fatty Acids … on Cognitive Function**: JAMA. 2015 Aug 25;314(8):791-801.

Clayton P, Ladi S. **From Alga to Omega: have we reached peak fish oil?** JRSM September '15, in press.

Dutot M, Fagon R, et al. **Antioxidant, anti-inflammatory, and anti-senescence activities of a …extract from brown seaweed Ascophyllum nodosum.** Appl Biochem Biotechnol. 2012 Aug;167(8):2234-40.

Tirosh O, Shpaizer A, Kanner J. **Lipid Peroxidation in a Stomach Medium Is Affected by Dietary Oils (Olive/Fish) and Antioxidants** ... J Agric Food Chem. 2015 Aug 12;63(31):7016-23.

Wang T 2009. Industry Research Thesis, Open University, Iceland. ISBN 978-9979-9928-3-7 http://skemman.is/stream/get/1946/4139/11867/1/Final_fixed.pdf

Wu S, Ding Y, Wu F, Li R, Hou J, Mao P. **Omega-3 fatty acids intake and risks of dementia and Alzheimer's disease: a meta-analysis**. Neurosci Biobehav Rev. 2015 Jan;48:1-9.

Yang EJ, Moon JY, et al. **Jeju seaweeds suppress lipopolysaccharide-stimulated proinflammatory response in RAW 264.7 murine macrophages**. Asian Pac J Trop Biomed. 2014 Jul;4(7):529-37.

P3 Vitamin E

Brasky TM, et al. **Plasma phospholipid fatty acids and prostate cancer risk in the SELECT trial**. J Natl Cancer Inst. 2013 Aug 7;105(15):1132-41.

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

Labazi M, McNeil AK, et al. **Antioxidant requirement for plasma membrane repair in skeletal muscle.** Free Radic Biol Med. 2015 Jul;84:246-53.

McBurney MI, et al. **Suboptimal Serum α-Tocopherol Concentrations … NHANES 2003-2006**. PLoS One Vol 10, Number 8:e0135510. doi: 10.1371/journal.pone.0135510

Raederstorff D, Wyss A, et al. **Vitamin E function and requirements in relation to PUFA.** British Journal of Nutrition, Published online ahead of print, doi: 10.1017/S000711451500272X

P4 Vitamin K, Vitamin D

Schurgers L, et al. **High-Dose Menaquinone-7 Supplementation Reduces Cardiovascular Calcification ...** Nutrients. 2015 Aug 18;7(8):6991-7011

Tomlinson PB, Joseph C, Angioi M. … **Vitamin D supplementation on upper and lower body muscle strength. .. Review with meta-analysis** J Sci Med Sport 18, Iss 5, 575–580, doi:10.1016/j.jsams.2014.07.022