dr paul clayton's Health Newsletter

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The sugar curse
Beta carotene
Green Tea
Coffee

Honey, I'm diabetic! The sugar curse

I have recently written about **high-fructose corn syrup** (**HFCS**), the sweetener used in many soft drinks and confectionery items, and showed how strikingly similar it is to table sugar and honey. High-fructose syrup HFCS55 – the form used to sweeten drinks – consists of 55% fructose and 45% glucose. Honey is almost identical, and sucrose (table sugar) is 50% fructose and 50% glucose; so the differences between HFCS, **honey** and **table sugar** are negligible. Fruits and therefore fruit juices such as apple and pear are even 'worse' than HFCS, containing 66% fructose and 33% glucose.

So why are we so alarmed about fructose, and high-fructose corn syrup? Well, people are getting fatter and more diabetic (particularly in the USA, although we are not far behind), and rather then blame ourselves for eating too much and taking too little exercise, increasing numbers of folks are looking for a villain. HFCS is, at least on the surface, typecast for the role.

In America, consumption of HFCS increased over a thousand-fold between 1970 and 1990, far exceeding the changes in intake of any other food or food group. The increase in HFCS consumption means that a surprisingly large number of people are now getting a surprisingly large percentage of their daily calories from soft drinks and foods sweetened with HFCS. The most conservative estimates indicate that Americans are, on average, getting 3 to 5% of all their calories from HFCS, and the top 20% of soft drink consumers are getting around 10% of their calories from this source (Bray et al '04).

The increase in HFCS consumption between 1970 and 1990 ran almost in parallel with an equally remarkable increase in the incidence of overweight, obesity and diabetes, and it was this coincidence that started the anti-HFCS bandwagon. Admittedly, there is some scientific evidence which appears to support this; when fructose is metabolised in the liver, for example, it promotes fat synthesis.

Now a new study claims to have found a genetic 'missing link' which explains how fructose does this, and how high-fructose diets may increase the risk of diabetes (Nagai et al '09). In this experiment, high doses of pure fructose were given to 'PGC-1b knock-out' mice. These are mice specifically bred without the gene PGC-1b, a gene which is very influential in determining the rate of fat synthesis in the liver. In these mice the impact of high dose fructose on fat synthesis in the liver was blocked; and they did not develop the metabolic signs of diabetes.

The scientists concluded that the PGC-1b gene was the link between high fructose intake, and the huge increases that have occurred in overweight, obesity and diabetes.

"Both **metabolic syndrome** and **diabetes** have reached epidemic proportions worldwide with the global adoption of the westernized diet along with increased consumption of fructose," they said, "stemming from the wide and increasing use of high-fructose corn syrup sweeteners."

They added, however, "The combination of glucose, which will stimulate insulin secretion more potently than fructose, along with fructose, which is metabolized very rapidly and differently than glucose, might promote more lipogenesis than either one alone. We are currently examining this hypothesis."

And here, even if they didn't realise it, was the key. Diets containing excessive calories and excessive amounts of HFCS, or sugar, or honey, will all lead to the same metabolic end-point; especially in people who do not take enough exercise.

For those who still cling to the myth of the evils of HFCS, let me just end with the outcome of the annual American Society for Nutrition Public Information Committee symposium for 2007, charmingly titled "High Fructose Corn Syrup (HFCS): Everything You Wanted to Know, But Were Afraid to Ask".

At this well-attended conference, speakers from academia and industry came together to provide up-to-date information on this food ingredient. The scientists concluded that HFCS is very similar to sucrose, being about 55% fructose and 45% glucose, and thus, not surprisingly, few if any metabolic differences were found between HFCS and sucrose (Fulgoni V '08).

Having said that, HFCS does contribute to added sugars and calories, and those concerned with managing their weight should be concerned about calories from all sources. Switch to drinks sweetened with **aspartame**, **sucralose**, or **stevia**.

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A smoking carrot ...

Many studies show that in people who eat diets rich in fruits and vegetables, the risk of cancers including lung cancer is reduced – even among smokers.

For a while, scientists thought that it was the **beta-carotene** in fruits and vegetables that conferred protection, because beta-carotene is quite effective at killing cancer cells in vitro. Then came CARET and ATBC, two high profile clinical trials specifically set up to measure how effective beta carotene supplements might be in protecting smokers against cancer. The results were a major blow to those who try to use nutrients like drugs, ie as single entities and at high doses. They showed that when smokers took supplements of beta-carotene, the risk of lung cancer was increased.

Specifically, the Beta-Carotene And Retinol Efficacy Trial (CARET) in the United States and the Alpha-Tocopherol, Beta-Carotene Cancer Prevention (ATBC) trial in Finland, reported that beta-carotene, alone or in combination with vitamin E or retinyl palmitate, could increase the risk of lung cancer in smokers by 36 and 16 per cent, respectively, when compared to a placebo group.

More recently, a large population-based study at the University of North Carolina has produced similar findings. Jessie Satia's team found that long-term supplementation

with beta-carotene and the related compounds lutein and vitamin A increased a smoker's risk of lung cancer (Satia et al '09).

Furthermore, the risk of developing lung cancer increased with increasing length of time of supplementation, a pattern of dose and response that supports the main results.

It must be said that the Satia results have been criticised by some. Professor Hans Konrad Biesalski of the Institute for Biological Chemistry and Nutrition at the University of Hohenheim, has attacked the statistics and in particular the data collection (Biesalski '09).

"For this study participants were asked to give details from memory of food supplements which they had taken in some cases 10 years ago," said Biesalski. "It is hardly conceivable that the subjects were able to remember accurately enough in which sequence, how frequently and in what composition they had taken products containing micronutrients in the previous four or ten years. The validity of the questionnaires used and above all the conclusions drawn from them are therefore questionable."

Although I have some sympathy with the good Professor Biesalski's arguments, I must respectfully disagree with him. I find that the evidence against beta-carotene, vitamin A and lutein, in smokers, is persuasive and coherent.

For example, detailed biochemical studies have shown that when highly oxidative tobacco smoke enters the lungs, it oxidises and damages beta-carotene and related compounds and renders them reactive and dangerous (Liu et al '03). Dose too is important; low (ie dietary) doses of beta-carotene have anti-cancer effects, while high (pharmaceutical) doses have pro-cancer effects (Liu et al '04a). Liu's research group, based at the Human Nutrition Research Center on Aging at Tufts University, Boston, has also demonstrated that the impact of beta-carotene depends on the presence or absence of other antioxidant micronutrients such as vitamins E and C (Liu et al '04b).

These studies go a long way to fleshing out the beta-carotene story.

People eating a good diet consume low (physiological) amounts of beta-carotene combined with many other chemo-protective phytonutients, so dietary beta-carotene will be predominantly cancer-protective.

Many smokers do not eat well, and as a result are more likely to be depleted in most of the anti-cancer phytonutrients. In addition their vitamin C levels are generally reduced because of oxidant tobacco smoke; and so here, the high doses of beta-carotene in supplements are likely to be oxidised, and exert predominantly pro-cancer effects.

My final take is that if you are reckless enough to be a smoker, do not compound that recklessness by taking the wrong supplements. Eat a very healthy diet and top up with a properly designed nutrient support programme. NutriShield, with its wide spectrum nutritional content, qualifies.

For the virtuous non-smokers, such a programme is equally suitable, and will contribute to lowering the risk of cancer, heart disease, macular degeneration and many other diseases.

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A downside to lone folate

Several years ago the Aspirin/Folate Polyp Prevention Study (AFPP) was initiated. This was a placebo-controlled randomised intervention study, designed to investigate the role of aspirin and high dose folic acid on colon polyps and cancer in men and women at high risk of the disease. Very recently, an analysis of the data found that in men, supplementing with 1 mg of folate per day for ten years was linked to a trebling of the incidence of prostate cancer (Figueiredo et al '09). Should we be worried?

Commenting on the study, the editors of the journal in which this paper was published wrote: "Given the small number of prostate cancers in this study, the estimates of prostate cancer risk in the placebo and folic acid groups should be interpreted with caution."

We should be cautious, because folate is a complex thing. Folate deficiency results in damage to DNA that may lead to cancer, and several studies have associated diets low in folate with increased risk of breast, pancreatic, and colon cancer (ie Rohan et al 2000). These pieces of evidence suggest that supplementing with folate should reduce cancer; but in the AFPP study there was an apparent increase in prostate cancer, and increased intakes of folate have been linked to a possible increased risk of bowel and breast cancer also (Mason et al '07).

One possible explanation for this apparent paradox is that folate is a requirement for all dividing cells – which is why folate antagonists such as methotrexate are used as anti-cancer drugs. If cancer cells are already present, an excess of folate in the system might well enable them to grow more rapidly than they otherwise would. This is a worrying prospect for those taking large doses of folate over long periods of time. (I do not include in this category women trying to become pregnant, and the well-documented ability of short-term folate to reduce the risk of neural tube defects.)

SOYBEANS
a good dietary source of folate

How could a folate-rich diet be protective, while a folate supplement was not? If folates are consumed in a plant-rich diet, they are co-ingested with a number of other plant-derived compounds which have a range of anti-cancer properties, and the overall effect of such a diet is indeed cancer-protective. If folate is added as a simple supplement to a typical modern diet, however, which is depleted in many of the cancer-protective phyto-nutrients, then its potential cancer-promoting effects could come to the fore.

In my view this is yet another example of the potential problems that may arise when using nutrients as if they were drugs. Folate should never be used as a single agent, but only as one element in a comprehensive micro- and phyto-nutrient support programme, such as NutriShield, in which anabolic and chemo-protective compounds are combined, as they would be in a healthy diet.

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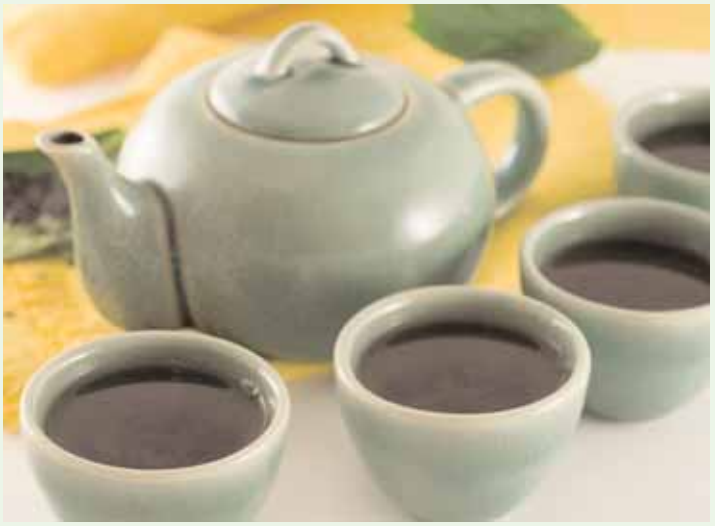
Green tea, green teeth

Gum disease affects around 30% of the population, and becomes more prevalent as we age. Oral hygiene plays a role, and nutritional factors are important too.

According to a new study from Japan's Kyushu University, drinking green tea may offer protection against gum disease (Kushiyama et al '09). The team of scientists recruited 940 men aged between 49 and 59, and analysed if green tea consumption had any effect on the incidence of gum disease, as measured using periodontal pocket depth (PD), clinical attachment loss (CAL) of gum tissue, and bleeding on probing (BOP) of the gum tissue. Men who regularly drank green tea had superior periodontal health than those who drank less green tea, and the effect was dose-related. For every one cup of green tea consumed per day, there was a 0.023-mm decrease in the mean PD, a 0.028-mm decrease in the mean CAL, and a 0.63 per cent decrease in BOP.



However, there is more to gum health than green tea, which may be good news to those who don't really like the stuff. It was almost certainly the flavonoids in the tea that were responsible for the tea's effects, and of course there are flavonoids in many other foods and beverages. For example, flavonoids in berry fruits such as the cranberry (Weiss et al '04, Yamanaka et al '04) reduce bacterial formation of plaque around the teeth by inhibiting the bacterial enzymes called glucosyltransferases which build biofilm and plaque.

Even more exciting are the edible seaweeds. The sulphated polysaccharides contained in some marine algae are highly effective in preventing plaque formation by interfering with glucan deposition (Saeki '94, Saeki et al '96). This approach has recently been developed as a nutritional supplement, standardised to its fucoidan content and sold to dentists and vets as 'PlaqueOff'. It is surprisingly effective at reducing and removing plaque, and it is probable that this mode of action will also protect against infection at other vulnerable sites where biofilm is critically involved, such as heart valves and prostheses (Wiklund '08).

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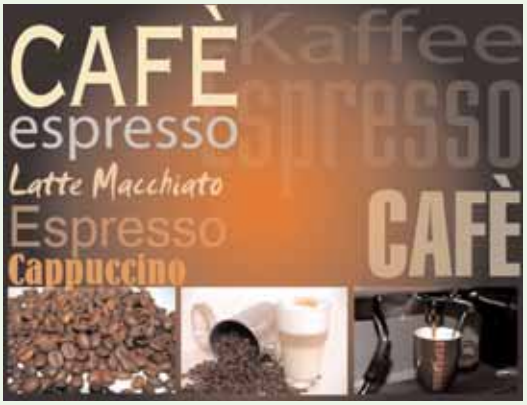
Coffee – bad science but good for you

Moderate consumption of instant coffee may boost the numbers of certain bacteria in the gut with reputed health benefits, says a study from the Nestle Research Center in Lausanne (Jaquet et al '09). Well, that's unexpected – and what a coincidence that the news should come from Nestle, a major producer of instant coffee.

"Our results show that the consumption of instant coffee produced an increase in the metabolic activity and/or numbers of Bifidobacterium spp, a bacterial group of reputed beneficial effects," said the researchers, "although the health benefits or the biological relevance associated with these findings have still to be assessed."



I should point out that this was a small study, involving only 16 volunteers, who were fed on a diet containing no probiotics, and no whole grains (in an attempt to reduce intakes of prebiotic fibre). And that the effects were relatively small. And that there is no plausible mechanism which would explain the reputed increase in bifidobacteria, other than the presence in instant coffee of very small amounts of soluble fibre. And that the only meaningful way to increase the numbers of possibly health-promoting bacteria in the gut is to consume significant amounts of prebiotic fibre such as inulin or resistant starch, with or without probiotic foods such as live yoghurts.

It seems to me that this is an example of bad science, where commercial scientists grasp at straws to try to demonstrate a spurious health benefit for their sponsor's product.

And it is so unnecessary, because – as readers of these newsletters will know – there are already so many health benefits linked to **coffee** and to its delightful combination of **flavonoids** and **caffeine**; including a reduced risk of Parkinsonism (Powers et al '08, but see also Simon et al '08), stroke (Lopez-Garcia et al '09), and colon and liver cancer (Higdon & Frei '06). This last is particularly interesting as caffeine has been found to restore the immune system's ability to target and kill cancer cells (Mandal & Poddar '08).

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Gout – a thoroughly modern disease

People often think of gout, a joint disease which causes extreme pain and swelling, as an 18th century disease. They might associate it with George Hepplewhite's gouty chairs and foot-stools, or with James Gillray's wonderful cartoons.

But gout is alive and kicking today, it has doubled in the USA since 1985 and is increasing rapidly in the UK also. Most common in men aged 40 and older, it is caused by excess uric acid in the blood leading to uric acid crystals collecting around the joints, where they trigger fierce inflammation – as was identified by Philippus Theophrastus Aureolus Bombastus von Hohenheim (aka Paracelsus) five centuries ago. But is it really a disease of excess, as Gillray famously portrayed it? Or is it, more specifically, caused by dietary imbalance?

Conventional dietary recommendations for gout have focused on the restriction of purines, dietary compounds that are broken down into uric acid. Purines are found in high levels in meat and meat products, especially liver and kidney, and in beans. Alcohol has long been suspected as a possible trigger factor, but new research shows that it may not be alcohol itself but the other compounds in alcoholic drinks that are important; wine does not cause problems but beer, which contains high levels of purines, certainly does (Choi et al '04). (This is good news for aficionados of cider which is similar to wine in that it contains no purines.)

Non-alcoholic beverages are also implicated. One recent study found that high consumption of soft drinks increased the risk of gout (Choi & Curhan '08). The scientists reckoned that it was the fructose (in HFCS) in the soft drinks that was doing this; fructose-rich fruits and fruit juices carried the same risk, but diet soft drinks did not. This is not a reason to switch to table sugar which, you'll remember, is effectively identical to HFCS – but it is a persuasive reason to cut down on both HFCS and sugar.

In general, therefore, meats and sugars are likely to increase risk, and these foods have increased significantly in our diets since 1950 – which would be one factor contributing to the increase in gout. But there is more to this story, because we have also reduced our intake of protective foods. We eat less (proportionately and in absolute terms) fruits and vegetables than we used to do 50 years ago, and certainly far less than we ate 150 years ago, and these foods contain valuable protective factors such as the flavonoids and vitamin C.

A substantial prospective 12 year study carried out in Vancouver, involving the extravagantly mustachio'd but scientifically rigorous Walter Willett, found that drinking coffee was associated with a reduced risk of developing gout (Choi et al '07). Another study, derived from the same Health Professionals database, discovered that higher doses of vitamin C were associated with lower uric acid levels (Gao et al '08); and a significantly reduced risk of gout (Choi et al '09). In this trial, carried out at Boston University School of Medicine, men with vitamin C intakes of at least 1,500 milligrams per day had 45% less gout – which is a highly significant degree of protection.

So for those men – and the few women – plagued with gout, there is an alternative to drugs. Switch from **meat** and **seafood** to **plant foods** (except for beans), and from **beer** to **wine**, and take a well-designed nutritional support programme which contains the flavonoids, vitamin C and, preferably, wide-spectrum nutrition.

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