

GI News—March 2012



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Recently, an opinion piece in *Nature* called on governments to regulate sugar in a fashion akin to alcohol. Presented as a possible solution to the obesity epidemic, the jist of the arguments were that worldwide sugar consumption has increased, sugar is toxic and addictive and, therefore, regulating sugar like alcohol or tobacco (including taxation and limiting access to individuals below the age of 17), would reduce obesity and prevent the metabolic syndrome. In a thoughtful [commentary](#) on the piece, Dr Arya Sharma (professor of medicine and chairman in obesity research and management at the University of Alberta) highlights the complexity of the obesity issue and points out the relevant microeconomic supply-demand issues and practicalities (and potential absurdities) of enforcement. Check it out in Food for Thought.

Good eating, good health and good reading.

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Food for Thought

Why banning sugar will not solve obesity.

Guest contributor, Dr Arya M. Sharma, takes issue both with the recent proposal to tax and ban sugar as well as the rather simplistic causal linking of sugar to the obesity epidemic. Here is why.

My main criticism is that, as so often, the authors have chosen to focus on the 'what' (eating too much sugar) rather than on the far more complex issue of the 'why' (why is this happening?). The *Nature* article (<http://www.nature.com/nature/journal/v482/n7383/full/482027a.html>) is no doubt well intended, however I sincerely fear that these rather simplistic and superficial 'one-size-fits-all' solutions to the obesity epidemic based on principles of shame, blame, tax, and ban, merely distract us from having a value-driven and non-judgemental discussion about the true drivers of the societal (e.g. industrialization and centralization of food production), psychological (e.g. stress, lack of sleep, emotional deprivation) and biological (e.g. fetal imprinting, endocrine disrupters) changes that have led to this epidemic.

While there is no doubt that overconsumption of sugar (like consuming too much salt – not sodium, trans-fats, alcohol, or perhaps processed foods in general) may well promote ill health, these links may be far less robust or scientifically proven than the article suggests. More importantly, there is very little evidence from high-quality intervention studies (outside of the rather artificial setting of a clinical trial) that the proposed population measures (namely attempting to restrict sugar consumption by banning or taxing it) would have the desired effect on obesity or anything else – if there are such examples, the article certainly fails to mention them.

Obesity is a multifactorial complex condition driven by a myriad of socioeconomic, psychological, and biological factors – some of which do indeed make many of us prone to ‘overconsume’ salt, sugar, fats, and perhaps alcohol or illicit drugs. In the case of sugar, the article unfortunately fails to seriously delve into what exactly these socioeconomic, psychological, or biological drivers to consume more sugar may be (beyond simply suggesting that sugar is cheap, omnipresent and ‘addictive’). Unfortunately, by reducing the solution to the obesity epidemic to simply a matter of banning and taxing sugar, the article not only reinforces the widely held stereotype that obese people are obese simply because they eat too much (in this case sugar) but also that obese people, because of the damage they do to themselves and society, need to be punished and policed for the benefit of all.

But, even if sugar was indeed a major driver of obesity, calling for interventions primarily on the demand side (making sugar less accessible and more expensive) rather than the supply side (making sugar less attractive for farmers to produce) is problematic. Paradoxically, changing demand without changing supply, at least in the short term, may well have exactly the opposite effect– sugar becomes even cheaper, thus making it an even more attractive ingredient for food producers. Reductions in the price of raw materials will likely quickly neutralize any increased cost of taxation with the net effect on consumption being zero. If, in the long run, such interventions did actually reduce sugar consumption in countries where it is regulated, we would simply be diverting streams to countries where it is not (worldwide tobacco consumption is the perfect case study for this).

The article is also rather cavalier about how exactly such measures would be implemented and enforced. As we well know from the hopelessly lost ‘war on drugs’, if people really want something (like sugar, assuming it is indeed as addictive as the authors suggest), they’ll find ways to get it. So making something ‘illegal’ is meaningless unless government is also prepared to enforce any such legislation. For a substance as omnipresent as sugar, this would require a rather expensive bureaucracy (I can already see food and drug inspectors raiding schools, recreation facilities, and grocery stores to ensure that no candy is sold to anyone below the legal age). I would imagine that the money required to effectively police and enforce any such new legislation would more than outweigh any potential revenues from the ‘sugar tax’ thereby snuffing any hope that such revenues could perhaps be used for other efforts to reduce obesity (like building bicycle lanes).

Finally, it is not clear to me why the authors would chose to simply focus their attention on sugar – it would have made as much sense to include all refined carbs, as it takes very little for our digestive systems to turn a slice of Wonder Bread or pizza into glucose. Will all refined carbs (and what exactly is the definition of ‘refined’ in this context? Do we include polished rice?) be next on the list of toxic substances that require a permit? And what about other natural sources of sugar – are we going to tax cane sugar, beets, honey, or perhaps even maple syrup? Let us also not forget that biologically there is little difference (if any) between

the ample sugar in fruit juice and the sugar I add to my cup of tea.

Check out Dr Sharma's website at www.drsharma.ca. You can also subscribe to his regular blog postings.

News Briefs

News Briefs includes four fructose studies published in the last year (three of them in recent weeks). We didn't intend to focus on fructose this issue, but with all the heated debate about sugars (and in particular fructose) and obesity at present, we felt made the decision to run them all, as we felt that *GI News* readers would be keen to see the latest science on a topical issue that often generates more heat than light. We are sure that you'll agree that it's vital to get the science right and the dogma off the table to have any chance of coming up with solutions (note the plural) to the obesity epidemic.

For weightloss, a moderate natural fructose diet superior to low-fructose diet.

In Food for Thought, Dr Arya Sharma reminds us that there is very little evidence from high-quality intervention studies that attempting to restrict sugar consumption would have the desired effect on obesity or anything else. In fact, there are very few high quality intervention studies at all regarding sugar and fructose consumption and the effects on health. This recent randomised, 6-week pilot study published in *Metabolism* (www.ncbi.nlm.nih.gov/pubmed/21621801) from Magdalena Madero and colleagues provides a useful addition to the literature. The researchers report that for weightloss, an energy-restricted, moderate natural fructose diet was superior to an energy restricted, low-fructose diet. The 131 obese participants were placed on low-fructose (less than 10 grams a day) diet or moderate natural fructose diet (50–70 grams a day with natural fruit supplements). The diets consisted of 55% carbs, 15% protein and 30% fat. All participants lost weight, but those in the moderate natural fructose group lost nearly 50% more (4.19 ± 0.30 kg) than participants in the low fructose group (2.83 ± 0.29 kg). The percentage of body fat dropped in both groups but the difference wasn't statistically significant. In terms of changes in the measured health parameters (blood glucose, blood pressure, blood fats, insulin resistance) there were no significant differences between the groups. Concluding, they write: 'for weightloss, a moderate natural fructose diet was superior to a low-fructose diet. Such a diet may offer greater benefits than other energy-restriction diets, as it does not entail the restriction of total carbohydrate intake and hence may be more sustainable.'

Commenting on this study, Dr Alan Barclay says that while it appears to be about fructose, when you read through it you see that the authors state that the reason why the moderate fructose (high fructose by Australian standards) diet caused greater weight loss than the low fructose diet was because it has a lower GI: 'patients in the very low fructose diet had a higher glycemic index and glycemic load'.

Is fructose being blamed unfairly for obesity epidemic? Or do we just eat and drink too many calories?

In a systematic review and meta-analysis of more than 40 published studies on whether the fructose molecule itself causes weight gain in *Annals of Internal Medicine*, (www.annals.org/content/156/4/291.abstract) researchers from St. Michael's Hospital conclude that: 'Fructose does not seem to cause weight gain when it is substituted for other carbohydrates in diets providing similar calories. Free fructose at high doses that provided

excess calories modestly increased body weight, an effect that may be due to the extra calories rather than the fructose.'

In 31 'isocaloric' trials they reviewed, participants ate a similar number of calories, but one group ate pure fructose and the other ate non-fructose carbohydrates. The fructose group did not gain weight. In 10 'hypercaloric' trials, one group consumed their usual diet and the other added excess calories in the form of pure fructose to their usual diet or a control diet. Those who consumed the extra calories as fructose did gain weight. However, all that could mean is that one calorie is simply the same as another, and when we consume too many calories we gain weight, said the lead author, Dr John Sievenpiper.

'Fructose may not be to blame for obesity,' he said. 'It may just be calories from any food source. Overconsumption is the issue.' Participants in the studies examined by Dr. Sievenpiper ate fructose in the form of free crystalline fructose, which was either baked into food or sprinkled on cereals or beverages. The studies did not look at high fructose corn syrup, which has been singled out as the main culprit for weight gain as it is only 55 per cent fructose, along with water and glucose. Dr. Sievenpiper said the majority of studies they examined were small, of short-duration and of poor quality, so there is a real need for larger, longer and better quality studies.

Prolonged fructose intake not linked to rise in blood pressure

Eating fructose over an extended period of time does not lead to an increase in blood pressure, according to a new study published in *Hypertension* (<http://hyper.ahajournals.org/content/early/2012/02/13/HYPERTENSIONAHA.111.182311>). It found that despite previous research showing blood pressure rose in humans immediately after they consumed fructose, there is no evidence fructose increases blood pressure when it has been eaten for more than seven days. In fact, researchers led by Drs David Jenkins and John Sievenpiper observed a significant decrease in diastolic blood pressure – the measure of blood pressure when the heart is relaxed between contractions– in people who had eaten fructose for an extended period of time. 'A lot of health concerns have been raised about fructose being a dietary risk factor for hypertension, which can lead to stroke, cardiovascular disease, renal disease and death,' said Vanessa Ha, lead author of the paper. 'However, we wanted to determine whether fructose itself raised blood pressure, or if the apparent harm attributed to fructose was simply because people are eating too many calories. For example, we know that people are consuming more soft drinks than ever, but is it the fructose, the extra calories, or possible other factors that are adding to their illnesses?'

The study looked at the effect of all sources of fructose, including natural and crystalline. In the systematic review and meta-analysis, Ha and colleagues pooled the results of 13 controlled feeding trials which investigated the effects of fructose on blood pressure in people who had ingested fructose for more than seven days. The 352 participants included in their analysis ate an average of 78.5g of fructose every day for about four weeks. Ninety five percent of all people in the US consume less than 87 g per day (<http://jn.nutrition.org/content/139/6/1228S/T2.expansion.html>).

Fructose and BGLs

First of all, despite what this story says, we are not advocating that you add pure fructose to your meals instead of your usual sweetener – many factors need to be taken into consideration when making that decision and we recommend you discuss the issue with your

health professional first. The point of studies like this is to get the science right and the dogma off the table. In this small meta-analysis of 6 controlled feeding studies published in the *British Journal of Nutrition* (www.ncbi.nlm.nih.gov/pubmed/22354959?dopt=Abstract), Dr John Sievenpiper and researchers from St Michael's Hospital found that contrary to concerns that fructose may have adverse metabolic effects, there is evidence that a small 'dose' (around 10g or 2 teaspoons) of fructose can actually lower the glycemic response to a high GI meal without adverse effects on fasting insulin or body weight. In their conclusion, they call for larger and longer trials to confirm these results.

Is 'fat' the sixth taste?

Researchers in Australia and the US have found that along with sweet, sour, salty, bitter and umami there seems to be a sixth taste – 'fatty'. Deakin University researchers reported in the *British Journal of Nutrition* (<http://journals.cambridge.org/action/displayAbstract?fromPage=online&aid=7816724>) that our tongues can detect a sixth taste – fat. They also found that people with a high sensitivity to the taste of fat tended to eat less fatty foods and were less likely to be overweight. They suggest that training our taste buds' sensitivity to fat is a potential way to reduce overweight and obesity. Deakin's Dr Russell Keast explains that: 'people insensitive to fat taste tend to consume more energy because their body does not tell them to stop eating. What is measured in the mouth reflects the body's response to fats,' he said. 'Those who are insensitive to fat taste do not get the fullness signals. So, when consuming a fatty meal, a healthy weight person would start to feel full and stop eating and the gap between meals would also be extended. However, those who are insensitive to fat taste do not feel full and therefore keep eating and the gap between meals is also reduced. In our study we also found that the people with less sensitivity to fat taste ate significantly greater quantities of butter, meat and dairy. This finding leads us to believe that specific foods groups and perhaps different types of fats are associated with decreased sensitivity to fat in foods.'

According to researchers at Washington University School of Medicine writing in the *Journal of Lipid Research* (<http://www.ncbi.nlm.nih.gov/pubmed/22210925>), genes probably do play a part. They found that people who made more CD36 protein could easily detect the presence of fat – in fact, those who made the most were eight times more sensitive to the presence of fat than those who made about 50 per cent less of the protein. 'The ultimate goal is to understand how our perception of fat in food might influence what foods we eat and the quantities of fat that we consume,' says Prof Nada A. Abumrad. 'What we will need to determine in the future is whether our ability to detect fat in foods influences our fat intake, which clearly would have an impact on obesity.'

[Get the Scoop with Emma Stirling](#)

The scoop on dietary AGEs.

Have you heard about dietary AGEs? No we're not talking about your real age compared with a predicted health age, but a fascinating and emerging area of sophisticated nutrition science. AGE's stand for Advanced Glycation End Products and researchers are becoming more interested in the role they play in triggering inflammation and damaging our health as we age. Let's explore ...

AGE gracefully? Advanced Glycation End Products are formed in many different foods,

even natural foods like fruit and vegetables, like the browning of a cut apple or toasting bread. However, the levels are much, much higher in processed foods and our modern way of eating. AGEs are commonly formed in foods that have been heated to extreme temperatures like the commercial frying or manufacture of foods, but also in other processes like long term storage and refrigeration. Research suggests that the level of AGEs in our food supply may accelerate physiological aging and lead to premature disease development such as type 2 diabetes, cardiovascular and renal (kidney) disease. So it's no surprise that scientists are on the path to discover a whole lot more.

Act your AGE It's impossible to completely eliminate AGEs from your diet, but there are many steps you can take to lower your intake. In fact a recent study by Baker IDI Heart and Diabetes Institute in Australia has provided evidence that dietary intervention using diets low in AGE content may improve inflammatory profiles, insulin sensitivity plus risk and complications of type 2 diabetes in overweight people. This has built on previous research, including a paper in the *Journal of the American Dietetic Association* (www.ncbi.nlm.nih.gov/pubmed/15281050) stating that avoiding fast, processed and fried foods and returning to traditional methods of cooking such as boiling and stewing, can reduce the AGE content of the diet by up to 50%.

According to Associate Professor Josephine Forbes, formerly at Baker IDI and now at the Mater Medical Research Institute in Brisbane, 'Glycation is a major problem in diabetes and these molecules have the capacity to do major damage to the organs of a diabetic person over several decades. Essentially, it speeds up the ageing process, and can lead to a 'caramelisation' of organs such as the kidney.'

Lower your AGE So it would seem that the old mantra 'fresh is best' shines through once again, and we now have new evidence for the superiority of eating seasonal, just picked produce as well. We asked Professor Forbes for her top tips on lowering dietary AGEs.

- Cut right down on processed foods and eat more whole foods close to their natural source
- Follow the seasons and eat like a locavore, avoiding food that has been in long storage
- Look for handmade, artisan food products like breads and wholegrain breakfast cereals
- Favour cooking techniques like steaming and poaching, over grilling and frying
- Add plenty of aromatic fresh herbs and acidic juices (such as lemon and lime) to your meals.

Emma Stirling is an Accredited Practising Dietitian and health writer with over ten years experience writing for major publications. She is editor of The Scoop on Nutrition (www.scoopnutrition.com) – a blog by expert dietitians. Check it out for hot news bites and a healthy serve of what's in flavour.

[In the GI News Kitchen](#)

American dietitian and author of *Good Carbs, Bad Carbs*, **Johanna Burani**, shares favourite recipes with a low or moderate GI from her Italian kitchen. For more information, check out Johanna's website at www.eatgoodcarbs.com. The photographs are by Sergio Burani. His food, travel and wine photography website is www.photosbysergio.com.

Salmon and chicory open-face grilled sandwiches

Italians have a long-standing love affair with Mediterranean bitter greens. In fact, they have been consuming chicory and its cousin, escarole, as a popular vegetable choice since the 14th century. These greens are more commonly used raw, as in salads, but they are absolutely delicious when braised as in this recipe. I use Alaskan wild smoked sockeye salmon for this recipe. Makes 4 servings.

1 garlic head
3 tsp olive oil, separated
½ tsp salt
450g/1lb. fresh chicory
250g/8oz sliced smoked salmon
4 slices whole wheat sourdough bread (30g/1oz slices)

Preheat the oven to 200°C (400°F).

Bake the head of garlic – remove the outer layers of garlic skin, taking care to keep cloves intact. Using a sharp knife, cut off top 5mm/1/4in from the garlic head and discard. Drizzle 1 teaspoon oil over the garlic head, wrap it in aluminum and seal, creating a steam tent. Bake for 30 minutes. Cloves will be soft to the touch. Remove from oven, unwrap and cool.

Remove outermost layers of chicory and discard. Separate remaining leaves and wash thoroughly. Gently drain, allowing some of the water to remain on the leaves. Chop coarsely and place in a large sauté pan. Cover and braise over medium heat. Remove chicory from heat. Add in remaining oil and the salt. Mix well and set aside.

Grill (or toast) bread on both sides. While still warm, spread one or more cloves of baked garlic on one side of each slice of bread.

Place 60g/2oz salmon on each slice of bread. Pile about ½ cup of cooked chicory on top. Serve warm.

Per serving

Energy: 850kJ/203cals; Protein 19g; Fat 6g (includes less than 1g saturated fat and 10mg cholesterol); Available carbohydrate 21g; Fibre 4g

Cut back on the food bills and enjoy fresh-tasting, easily prepared, seasonal, satisfying and delicious low or moderate GI meals that don't compromise on quality and flavour one little bit with this **Money Saving Meals** recipe making the most of quinoa. For more recipes check out the Money Saving Meals website at www.moneysavingmeals.com.au

Chicken and pumpkin soup with quinoa

When a warm and comforting soup is all that's needed, Anneka Manning's recipe from *The Low GI Family Cookbook* is the perfect meal in a bowl. Gluten-free, quick cooking quinoa is an excellent source of low GI carbs, fibre and complete protein, as well as being rich in B vitamins and minerals. Serves 6

1 small brown onion, finely chopped
1 leek (pale section only), cleaned, halved lengthwise, thinly sliced
2 medium carrots, scrubbed, diced
1 celery stick, diced
2 garlic cloves, crushed
2 tsp ground cumin
2½ cups water

4 cups salt-reduced chicken stock
500g/1lb 2oz chicken breast or tenderloin fillets, trimmed
¾ cup quinoa, rinsed
400g/14oz pumpkin, peeled, deseeded, cut into 1cm (½in) pieces
freshly ground black pepper, to taste
2–3 tbsp chopped flat-leaf (Italian) parsley

Combine the onion, leek, carrots, celery, garlic, cumin and 1/2 cup water in a medium saucepan. Cover and cook over medium heat, stirring occasionally, for 10 minutes or until the onion is tender.

Add the remaining 2 cups water, the chicken stock and the chicken. Bring to a simmer over medium heat. Reduce heat to low and cook, very gently, for a further 2–5 minutes or until the chicken is just tender. Remove the chicken from the soup with a slotted spoon and set aside.

Add the quinoa to the soup. Bring back to a simmer over high heat then reduce heat to medium and simmer for 10 minutes. Add the pumpkin and simmer for a further 5 minutes or until the pumpkin and quinoa are tender.

Meanwhile, use 2 forks or your fingers to shred the chicken. Add the chicken to the soup and heat through. Taste, season with pepper and serve immediately sprinkled with the parsley.

Per serving

Energy: 1060kJ/250cals; Protein 27g; Fat 6.5g (includes 1.8g saturated fat); Available carbohydrate 21g; Fibre 4g

My Meatless Mondays.

Cauliflower and celeriac soup

This wonderfully comforting soup has a delicious, unusual flavour that will keep your fellow diners guessing. Serves 4 to 6

1 tbsp olive oil
1 onion, roughly chopped
250g/8oz celeriac, peeled and roughly chopped
450g/1lb cauliflower, roughly chopped
440g/15oz can cannellini beans, rinsed and drained
4 cups chicken stock
Flaky sea salt and freshly ground black pepper to taste
Extra virgin olive oil

Heat the oil in a large heavy-bottomed saucepan over medium-high heat and cook the onion for 2 to 3 minutes. Add the celeriac, cauliflower, and beans and cook for another 3–4 minutes, stirring frequently. Pour in the stock. Increase the heat and bring to a boil, then reduce the heat and simmer, covered, for 15–20 minutes or until the vegetables are cooked through.

Allow to cool slightly, then puree with an immersion blender or in a blender. Season with salt and pepper. Ladle the soup into bowls and finish with a drizzle of extra virgin olive oil.

Per serve (based on 4 serves)

226 calories; 10 g protein; 36 g carb; 6 g fat; 0 g saturated fat; 5 mg cholesterol; 10 g fiber; 591 mg sodium

From *High Protein, Low GI, Bold Flavor: Recipes to Boost Health and Promote Weight Loss*, copyright © Fiona Carns, 2012; reprinted by permission of the publisher, The Experiment. Available wherever books are sold.

Busting Food Myths with Nicole Senior

Cravings tell us what our body needs

I'm pregnant and have a real and persistent hankering for sour foods: I'm putting lemon juice on everything, have a new respect for balsamic vinegar and love the taste of anything that makes my mouth pucker. I think I'm experiencing cravings. It is common for women to experience both food aversions and food cravings during pregnancy but research is sketchy as to why. While we joke about women craving strange combinations like pickles and ice cream, more typical cravings are for sweet, salty or sour foods (so I tick box number 3). Although hormonal upheaval is blamed, there's no good science to support the idea that women crave food they need more of. Weirdly, some women crave inedible things like dirt, clay and even cigarette butts (eew): this is called pica. But cravings are also recognised outside of pregnancy.

Some people — especially women—indulge their (non-pregnant) cravings because their 'body knows best'. However, the most common food craving in women is chocolate – a food the body doesn't need at all (although it is delightful). Unsurprisingly, cravings for broccoli, eggs and green leafy vegetables are rare. In fact, cravings are more common in women than in men and the facts point to the mind and not the body calling the shots.

Sweet cravings can fluctuate throughout a woman's menstrual cycle, indicating hormones play a role. But I've never seen a premenstrual woman crave a bowl of oats that would best meet her need for slow-release carbs. For some people feeling low, food cravings may be an attempt to prop up serotonin levels – which is akin to self-medicating with food. However, it is non-physiological factors that guide them toward sweets and cakes because they would achieve a good serotonin boost with the carbohydrate in a glass of milk or a slice of grainy toast (and it would last longer due to their lower GI).

We're hard-wired to eat a variety of foods to ensure our nutrient needs are met. In one way, wanting something different to eat is the body's way of getting what it needs. But cravings for cookies, ice cream and chocolate all point to a desire for pleasure rather than nutrients. Food cravings are most often for 'naughty' foods and prohibition simply leads to greater desire. If food cravings are problematic, psychological help is needed. In one study, visually picturing something that is non-food related was able to reduce the grip of food cravings.

So cravings appear to be 'in the mind' and not the body, and we need to use our mind to steer ourselves towards healthier choices if cravings for rich pickings are out of control. If you're feeling low, forego chocolate as daily medicine and try regular meals including low GI carbs and protein to balance serotonin levels and regulate mood. And pregnant ladies, eat what you crave (within reason) and enjoy as it's only temporary!

Nicole Senior is an Accredited Practising Dietitian and Nutritionist. This is an extract from Nicole's new book called *Food Myths* (New Holland) available from bookshops and online at www.greatideas.net.au/advanced_search_result.php?keywords=myths&osCsid=ab748a02c59db77632f3be06f4226804. You can contact Nicole at nicole@nicolesenior.com.au

[GI Symbol News with Dr Alan Barclay](#)

Why refined starch is the hidden ingredient – and why it matters for your health.

With all the heated debate about sugars and in particular fructose, it's easy to forget that sugars only make up around half of the carbohydrate in foods that we eat. Everyone seems to have forgotten about the other form of carbohydrate that we all eat: starches (formerly known as complex carbohydrates). Here I am talking about refined starches like cornflour, not starch as it is found in traditional, nutrient-rich starchy foods like root vegetables, legumes, cracked wheat, brown rice, pearl barley, quinoa and hearty porridge oats.

It's easy to understand why refined starches are invisible:

- They are not listed in the Nutrition Information/Nutrition Facts panel on foods, and
- The names for added refined starches are often unpronounceable like acetylated distarch phosphate, or food additive code number 1414 if you prefer.

Why does it matter? Well, you may be surprised to learn that refined starches contain essentially the same amount of calories (kJ), total carbohydrate and fibre as refined sugars, and without fortification, are just as devoid of vitamins and minerals. They also have a high GI. In a nutshell, refined starches are as detrimental to our health as refined sugar. So how come we never hear about them? Here's one possible reason. When I did a PubMed search to find all the scientific studies investigating the health effects of refined starches on human health, I found a grand total of 20 papers. Starches are truly the dark continent of nutrition ... If you want to know how much starch is in a food, it's easy to work out. Here's how:

- If you live in Australia, NZ or the UK simply subtract the amount of sugars from the total carbohydrates list on the nutrition info panel.
- If you live in the US, subtract both the amount of sugars and dietary fiber from the amount of carbohydrates.

And you can find all of the food additive codes for refined starch in the table below.

Starch	
Code	Name
(1400)	Dextrin roasted starch
(1401)	Acid-treated starch
(1402)	Alkaline-treated starch
(1403)	Bleached starch
(1404)	Oxidized starch
(1405)	Starches, enzyme-treated
(1410)	Monostarch phosphate
(1412)	Distarch phosphate
(1413)	Phosphated distarch phosphate
(1414)	Acetylated distarch phosphate
(1420)	Starch acetate
(1422)	Acetylated distarch adipate
(1440)	Hydroxypropyl starch
(1442)	Hydroxypropyl distarch phosphate
(1443)	Hydroxypropyl distarch glycerol
(1450)	Starch sodium octenyl succinate
(1451)	Acetylated oxidized starch

But if you think this is all sounding too hard we would have to say that we agree. In fact, it has that taint of nutritionism that we at *GI News* dislike so much. We would argue that rather than obsessively avoiding either refined starches or sugars it is much easier to look at the total amount of available carbohydrate in foods (in North America available carbohydrate equals total carbohydrates minus dietary fiber) and the foods GI. Swap high GI carbs with healthy low GI alternatives – it's as simple as that!

And of course, we recommend that as much as possible, you eat and enjoy real foods made with wholesome ingredients and pass on the packaged foods with ingredient lists as long as your arm. We like Michael Pollan's Food Rules 6 & 7: 'avoid food products that contain more than five ingredients' and 'avoid food products containing ingredients that as third-grader cannot pronounce.' Buon appetito!

The GI Symbol, making healthy low GI choices easy choices



For more information about the GI Symbol Program

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GI Update

Prof Jennie Brand-Miller answers your questions

‘What’s wrong with today’s diet?’

We didn’t always eat the way we do today. One of the most important ways in which our diet differs from that of our ancestors is the speed of carbohydrate digestion and the resulting effect on our blood glucose and hence every cell in our bodies.

Our hunter-gatherer ancestors would have eaten plenty of animal foods and large amounts of micronutrient-rich plant foods (leaves, berries, nuts, tubers), which would have been gathered every day, ensuring that their overall diet was both naturally low GI and low GL. Their carbohydrate intakes were lower than ours, because the main plant foods they had available were fruits and vegetables rather than cereals like wheat.

Beginning about 10,000 years ago, when we became farmers growing crops rather than hunter-gatherers, our diet changed in many ways. Starch entered the human diet, in a big way, for the first time. Large quantities of harvested cereal grains tipped our diets from being more animal to more plant based. Those plants were what we would now call wholegrain cereals (wheat, rye, barley, oats, corn and rice). The cultivation of legumes (beans), starchy roots and tubers and fruits and berries also contributed to the higher carbohydrate intake of our farmer ancestors.

But food preparation was simple back then: grinding food between stones and cooking it over the heat of an open fire. The result was that although we were eating a higher carbohydrate diet, the carbs were digested and absorbed slowly and the effects on our BGLs minimal. This diet was ideal for hard working farmers because it provided slow-release energy that helped to delay hunger pangs and provided fuel for working muscles long after a meal had been eaten. It was also easy on the insulin-producing cells in the pancreas. As far as we can tell, diabetes was rare.

Over time however, we developed the technology to grind flours more and more finely, and to separate bran completely from white flour. Finally, with the advent of high-speed roller mills in the 19th century, it was possible to produce white flour so fine that it resembled talcum powder in appearance and texture. These fine white flours were – and are – highly prized because they make soft bread and delicious, airy cakes and pastries.

As incomes grew, the foods commonly eaten by our ancestors – slow-release barley, oats and legumes – were cast aside; consumption of fatty meat increased. The composition of the

average diet changed again. We began to eat more saturated fat, less fibre and lots more easily digested, highly refined carbohydrates. Then something we didn't expect happened, too – blood glucose rises after a meal became higher and more prolonged, stimulating the pancreas to produce more insulin. As a result of these developments, we not only experienced higher blood glucose spikes after a meal, but we also experienced greater insulin secretion. We now know that excessively high glucose and insulin levels are among the key factors responsible for diabetes, heart disease and hypertension. And because insulin also influences the way we metabolise foods, it ultimately determines fat storage around the body.

Low GI organic snack bars from Canada

Brother and sister team, Nima and Salma Fotovat, created Taste of Nature to make healthy fruit and nut bars they could be proud of – ‘Nothing artificial, no fillers and no chemicals with names as hard to pronounce as they are to digest’. They are also low GI, certified kosher, certified vegan and dairy and gluten free. From the Toronto home base, they are now sold in 30 countries around the world. Here's the current range with their GI values and carb content (rounded) per bar:

- Argentina Peanut Plains – GI21; 12g carbs
- Brazilian Nut Fiesta – GI39; 19g carbs
- California Almond Valley – GI40; 19g carbs
- Canadian Maple Forest – GI36; 19g carbs
- Caribbean Ginger Island – GI49; 22g carbs
- Himalayan Goji Summit – GI38; 18g carbs
- Mediterranean Pistachio Passion – GI39; 18g carbs
- Niagara Apple Country – GI47; 26g carbs
- Nova Scotia Blueberry Fields – GI44; 19g carbs
- Quebec Cranberry Carnival – GI48; 18g carbs
- Persian Pomegranate Garden – GI51; 20g carbs

For more information, check out their website at www.tasteofnature.ca.