

Eat Fat, Cut The Carbs and Avoid Snacking To Reverse Obesity and Type 2 Diabetes



In association with



Introduction

After 250,000 years of evolution, humans became a healthy, well-nourished species with a long life expectancy. In only 30 years, things have gone catastrophically wrong, with epidemics of obesity and type 2 diabetes, unchecked by current policies and interventions likely to cause reduced life-expectancy, and bankruptcy of healthcare systems. This report represents a potential shift in failed policies, and recommends alternatives which are likely to reverse some of the counterproductive effects of recent policy.

Solving the obesity epidemic and its associated adverse health consequences such as type 2 diabetes is today's most important public health challenge. Obesity directly costs the National Health Service (NHS) more than £6 billion per year. Directly and indirectly type 2 diabetes costs £20 billion and without effective action, this price is likely to double over the next 20 years. [1] In the United States the cost of diabetes has soared recently, reaching US \$245 billion in 2012.

The roots of obesity and type 2 diabetes are firmly embedded in the food environment. Legislative efforts to encourage less consumption of processed foods and sugary drinks will help significantly reduce the burden of diet related disease which now contributes to more disease and death globally than physical inactivity, smoking and alcohol combined. [2] The role of poor dietary advice has been ignored for too long. Specifically, the "low fat" and "lower cholesterol" message have had unintended disastrous health consequences.

The flawed science behind this message and subsequent change in dietary guidelines introduced for Americans in 1977 [3] followed by the UK public health dietary advice issued by the National Advisory Committee on Nutritional Education in 1983 [4] has resulted in increased consumption of low fat junk food, refined carbohydrates and polyunsaturated vegetable oils. In the United States between 1961-2011 90% of the increased calorie intake has come from carbohydrates and polyunsaturated vegetable oils. [5]

The conspicuous rise in obesity immediately following their introduction suggests that they are a root cause of the problem. Nutrition science was originally founded on human correlational studies, which are frequently flawed. But that science has also been corrupted by commercial influences. The undue influence of the food industry on official guideline bodies and politicians has posed a significant threat to public health.

Public health professor Kelly Brownell noted that it took 50 years from the first published scientific evidence linking smoking and lung cancer until effective regulation was introduced to curb tobacco consumption. Big tobacco companies adopted a strategy of denial, planting doubt, obfuscating, and even buying the

loyalty of scientists. The recent similarities with big food companies are nothing short of chilling. [6]

We understand members of the food industry have a fiduciary responsibility to produce profit for their shareholders. But the real scandal, at great expense to public health, is that academics, institutions and journals whose primary responsibility is to patients and scientific integrity have at times colluded with industry for financial gain.

The latest Eatwell guide from Public Health England is perhaps an example of commercial interests trumping independent scientific evidence. The majority of the group established by Public Health England to determine the healthy eating role model for the country represented the food and drink industry. [Should the food and drink federation have a say in the design of England's healthy eating advice, or should independent clinicians fulfil that role?](#)

It is also shocking to suggest that consuming 22 teaspoons of sugar daily can fall within recommended guidelines. Yet, this has been true for over a decade. [7] For optimum health there is NO dietary or biological requirement whatsoever for added sugar.

To wind back from these harms the National Obesity Forum and Public Health Collaboration therefore, based on the most up to date scientific and clinical evidence, recommend a complete overhaul of dietary advice and public health messaging. The most effective and important changes can be covered in the following ten points.

1. Eating Fat Does Not Make You Fat

Evidence from multiple randomised controlled trials have revealed that a higher fat, lower carbohydrate diet is superior to a low-fat diet for weight loss and cardiovascular disease risk reduction [8, 9] An exhaustive analysis of 53 randomised controlled trials involving 68,128 participants conducted by the Harvard School of Public Health concluded " when compared with dietary interventions of similar intensity, evidence from randomised controlled trials does not support low fat diets over other dietary interventions for long term weight loss. In weight loss trials, higher fat weight loss interventions led to significantly greater weight loss than low-fat interventions."

Furthermore the Women's Health Initiative was the largest randomised controlled diet trial ever performed. 48,835 post-menopausal women were randomised to either their usual diet or a low-fat, calorie reduced diet with increased exercise with the hypothesis that this would reduce cardiovascular disease. The mean follow-up period was 8.1 years. The intervention achieved an 8.2% energy decrease in total fat intake and a 2.9% energy decrease in saturated fat intake, but did not reduce risk of CHD or stroke.[10] While not specifically a weight loss trial, nevertheless, the reduction in dietary fat and total daily calories (361 calories/day reduction) also failed to produce any significant weight loss over the duration of the study. This rejected the notion that the low-fat diet is either beneficial for cardiovascular disease or weight loss.

This can be explained in part that at a physiological level that consumption of fat induce fullness or satiation and that compared to the other macronutrients protein and carbohydrates, fat has the LEAST impact on blood glucose and insulin responses. Furthermore, insulin resistance is a precursor to type 2 diabetes and in men is the number one risk factor for heart attack. (see point 3)

We recommend that guidelines for weight loss for the UK should include an ad libitum low refined carbohydrate and a high healthy high fat diet (i.e non-processed foods or "real" foods) as an acceptable, effective and safe approach for preventing weight gain and aiding weight loss.

2. Saturated Fat Does Not Cause Heart Disease. Full fat dairy is likely protective.

The rush to condemn saturated fat as a nutrient of concern was premature. A new meta-analysis of the evidence available prior to the 1977 and 1983 US and UK change in dietary guidelines did not support the introduced dietary fat restrictions. Despite the lack of scientific consensus, entire nations were advised to limit total fat to 30% of calorie intake and saturated fat to 10% of calorie intake. As the authors stated: “dietary recommendations were introduced for 220 million US and 56 million UK citizens by 1983, in the absence of supporting evidence from randomised controlled trials.” [11]

This mistake was not rectified until 2014, more than three decades later, when a systematic review of 76 studies with over 600,000 participants from 18 countries concluded that: “Current evidence does not clearly support cardiovascular guidelines that encourage high consumption of polyunsaturated fatty acids and low consumption of total saturated fats.” [12] De Souza *et al* summarised the most recent verdict on saturated fat: “Saturated fat intake was not associated with all-cause mortality, CVD mortality, total CHD, ischemic stroke, or type 2 diabetes.” [13]

Current nutritional guidelines erroneously focus on total fat and saturated fat content, rather than specific food sources and the fatty acid subtypes. In particular, consumption of margaric acid, prevalent in full-fat dairy foods, significantly reduced the risk of cardiovascular disease. Thorning *et al* found that “Diets with cheese and meat as primary sources of Saturated Fatty Acids cause higher HDL cholesterol and apo A-1 and, therefore, appear to be less atherogenic than is a low-fat, high-carbohydrate diet.” [14] Full-fat dairy may also protect against obesity. A 2014 study concluded: “Participants in the highest tertile of whole-fat dairy intakes (milk, cheese, yogurt) had significantly lower odds for being obese.” [15]

Prospective cohort studies confirm that plasma saturated fatty acids from dairy sources such as cheese and yoghurt is inversely related to the incidence of type 2 diabetes. By contrast, the plasma saturated fat palmitic acid, linked to the consumption of starch, sugar and alcohol, is strongly associated with the development of type 2 diabetes. [16]

The most natural and nutritious foods available – meat, fish, eggs, nuts, seeds, olive, avocados – all contain saturated fat. These natural foods have formed part of the human diet since Paleolithic times and have been eaten ad libitum without adverse health consequences for millennia. The continued demonisation of omnipresent natural fat drives people away from highly nourishing, wholesome and health promoting foods.

3. Processed foods labelled 'low fat', 'lite', 'low cholesterol' or "proven to lower cholesterol" should be avoided.

No single piece of evidence exists that demonstrates reducing dietary saturated fat reduces cardiovascular events and death. Instead, improvements in hard cardiovascular outcomes are independent of cholesterol lowering. Dietary trials that provide abundant natural fats such as α -linoleic acid, polyphenols and Ω -3 fatty acids found in nuts, olive oil, oily fish and vegetables rapidly exert positive health effects. [17] The mechanism may include attenuating inflammation, atherosclerosis and thrombosis. Replacing saturated fat with omega 6 containing vegetable oils lowers cholesterol but does NOT improve cardiovascular mortality. [18] Worryingly, studies revealed a trend towards increased mortality.

Health professionals and the public must shift focus away from total and LDL cholesterol as a marker of cardiovascular health. Evidence clearly shows that a cholesterol profile characterised by high triglycerides and low HDL is more predictive of cardiac risk and a reliable surrogate marker of insulin resistance. Mathematical modelling reveals that preventing insulin resistance in young men would prevent 42% of myocardial infarctions, a larger reduction than correcting hypertension (36%), low high-density lipoprotein cholesterol (31%), body mass index (21%) or low-density lipoprotein cholesterol (16%). [19] The QRISK calculator, used by clinicians to estimate the 10-year risk of CVD, pointedly does NOT use LDL cholesterol but total cholesterol/HDL ratio instead.

The process by which cholesterol lowering foods 'work' raises serious concerns for coronary heart disease: *"However there is no evidence that plant sterols reduce the risk of CHD and much evidence that they are detrimental."* [20]

4. Limit starchy and refined carbohydrates to prevent and reverse Type 2 diabetes.

Type 2 diabetes is a disease of excessive insulin resistance, which manifests itself by elevated blood glucose levels. Patients demonstrate profound carbohydrate intolerance and public health messages should reflect that. Refined and starchy carbohydrates are well known to increase blood glucose levels, requirements for medications and weight gain. It is therefore very disturbing that both Diabetes UK and NHS websites prioritise the consumption of starchy carbohydrates for type 2 diabetics. Such diets will potentially lead to progressive worsening of disease with all its attendant complications, such as blindness, kidney failure, nerve damage, peripheral vascular disease, heart disease and stroke.

A recent comprehensive review concludes that dietary carbohydrate restriction is the “single most effective intervention for reducing all of the features of the metabolic syndrome” and should be the first approach in diabetes management. A ketogenic diet (one that comprises less than 10 per cent of calorie intake from carbohydrates) results in the greatest falls in HbA1C and reduction in the use of medications. These benefits accrue independently of weight loss. [21]

Royal College of General Practitioners clinical expert in diabetes Dr David Unwin, has very recently published profound results by simply recommending the LCHF diet to patients. He was recognized as the Innovator of the Year at the NHS Leadership Recognition Awards 2016. In addition to dramatically improving his patients health, Dr. Unwin has saved the NHS £45,000 per year for medications for diabetes compared to the average of his Clinical Commissioning Group . [22] If we were able to replicate these results in the 9,400 surgeries across the UK we could potentially save £423 million a year on drugs for diabetes alone. The downstream savings from reduced end organ damage (heart attacks, strokes, cancer etc.) are many orders of magnitude higher. This does not even factor in the significant savings in human suffering.

5. Optimum Sugar Consumption For Health is ZERO.

Added sugar has no nutritional value whatsoever. There are no biochemical reactions in the human body that require dietary fructose. No single study exists that demonstrates benefit associated with its consumption. [23] Excess sugar consumption is strongly associated with increasing risk of type 2 diabetes, hypertension, and cardiovascular disease, independent of its calories or its effects on body weight [24, 25, 26]. While we welcome the recent World Health Organisation maximum LIMIT recommendations, public health messaging should emphasize the fact that sugar plays NO role in a healthy diet.

Sugar should be relegated to the status of condiment or food additive, rather than food. It should return to its role as a decadent unnecessary thing to be consumed occasionally rather than a daily part of a healthy diet.

In addition we recommend that food labelling on added sugar should be recorded as number of teaspoons. This enables consumers to make more informed decisions when purchasing products in the supermarket.

6. Industrial Vegetable Oils Should Be Avoided.

For virtually all of the 2-4 million years of human history, pure vegetable oils have not been part of the diet. Linoleic acid (the omega-6 fat in these vegetable oils) is extremely susceptible to oxidation, making foods rancid, but also oxidizing in the body. [27, 28] In nature, linoleic acid exists in whole foods like seeds, nuts, fish, and eggs that also contain vitamins, minerals, antioxidants, to protect this highly susceptible polyunsaturated fat.

Recent human data suggest that we should remove these vegetable oils from our food supply for optimal health. A meta-analysis including almost 10,000 patients has confirmed that high intake of omega-6 oils (from vegetable oils/margarines) increases the risk of death and heart disease compared to saturated fat plus trans-fat. [29] Older data was confounded by the inclusion of omega-3 polyunsaturated fatty acid intake. This misrepresented the health benefits of omega-6. Other studies, such as the Anti-Coronary Club trial confirm that omega-6 PUFA increases death and coronary heart disease compared to animal fat. [30]

Importantly, cancer mortality increased with the consumption of omega-6 rich vegetable oils in the LA Veterans trial. [31] Numerous animal studies have found that omega-6 PUFA promotes the growth of experimentally induced cancers, whereas omega-3 inhibits the growth. Omega-6 rich vegetable oil (such as sunflower and corn oil), linked to the increased risk of death, coronary heart disease, and cancer in humans [32] as well as the growth of cancer in animal models [33] cannot be considered safe. Indeed it can barely be considered a food.

7. Stop Counting Calories ***(Calorie focused thinking has damaged public health.)***

A calorie is only a calorie if it is incinerated, and the heat given off measured; indeed that is the definition. However, calories from different foods have entirely different metabolic effects on the human body rendering that definition useless. Therefore, the effect on our health differs substantially depending upon where that calorie is derived from. For example, equal calorie portions of sugar, alcohol, meat or olive oil have widely differing effects on hormonal systems such as insulin, and satiety signals such as cholecystokinin or peptide YY. It is highly irrelevant how many calories a portion of food on a plate contains. What matters is how our body responds to the ingestion and absorption of those calories, and how they are metabolised, depending upon the specific food in question.

Current caloric reduction strategies for weight loss are highly ineffective. Data from the UK Clinical Practice Research Datalink from 2004-2014 estimates the probability of attaining a normal weight at 1 in 167 [34]. This equals a failure rate greater than 99%. This is easily confirmed by the experiences of virtually all people attempting a diet, the majority of which are calorie-based methods.

It is often assumed that excessive caloric intake is the root cause of obesity, but this is untrue. A calorie of food energy can have different metabolic fates depending upon the hormonal stimulation. For example, that same calorie of food may be used to generate body heat or stored as body fat. Obesity is a disease of energy partitioning, not one of total energy intake. The primary driver of this partitioning is the hormone insulin.

Calorie-focused thinking is inherently biased against high-fat foods, many of which may be protective against obesity and related diseases, and supportive of starchy and sugary replacements, which are particularly detrimental for those with insulin resistance.

Shifting focus away from calories and emphasising a dietary pattern that focuses on food quality rather than quantity will help to rapidly reduce obesity, related diseases and cardiovascular risk. [35] Rapid weight loss and regain from fad dieting is detrimental to health. Such 'weight cycling' contributes to hypertension, insulin resistance and dyslipidaemia resulting in increased mortality risk and worse cardiovascular outcomes. [36] The look AHEAD (Action for Health in Diabetes) trial found no cardiovascular benefits with a low calorie diet combined with increased physical activity in type 2 diabetic patients. Despite significant weight loss even up to the maximum follow-up of 13.5 years, no health benefits could be found. [37]

Public health should work primarily to support the consumption of whole foods that help protect against obesity-promoting energy imbalance and metabolic dysfunction rather than continue to promote calorie-directed messages that may create and blame victims and possibly exacerbate epidemics of obesity and related diseases.

8. *You cannot outrun a bad diet.*

It is widely accepted amongst the public and media that consuming more calories than we burn is the cause of the obesity epidemic, and thus the solution is to do more exercise. This is not correct. Obesity is a hormonal disorder leading to abnormal energy partitioning, which cannot be solely fixed by increasing exercise.

For many years, food and drink companies have pushed the physical activity message to exonerate themselves of their own role. Indeed, companies promoting processed and highly refined foods linked to the obesity epidemic have sponsored major sporting events e.g. the Olympics. Regular physical activity does indeed have a multitude of beneficial health effects but weight loss is not one of them. There has been little change in our levels of physical activity in the past three decades, whilst levels of obesity has increased. [38] One of the world's pre-eminent sports scientists, Professor Timothy Noakes says "the benefits of exercise are unbelievable but if you have to exercise to keep your weight down, your diet is wrong."

The association of ultra-processed food and soft drinks with sport is troubling. Celebrity endorsements give the wrong message, particularly to children. With the future of the nation at risk, we cannot afford to wait. We must dissociate physical activity from obesity. You won't be OK if you eat junk food even if you play sports. [39]

9. Snacking will make you fat (Grandma was right!)

There have been two major changes in our dietary habits since the 1970s, prior to the onset of the obesity epidemic. The change to a high carbohydrate, low fat diet has been well documented and has played an important role in causing obesity. The other change, the increase in meal frequency plays an equal if not larger role and has been largely ignored. In the 1970s, the average number of eating opportunities was three – breakfast, lunch and dinner. Fast-forward to 2005 and that number has almost doubled. [40] Now we eat breakfast, snack, lunch, snack, dinner and snack. And each of these are more often refined carbohydrate containing than not.

Eating continuously from the moment we arise to the moment we go to sleep does not allow our body to digest and use some of the foods that we eat. The entire day becomes an opportunity to store food energy without a chance to burn it. Eating six times a day does not result in weight loss (41), but tends to increase overall consumption of food.

Snacks tend to be highly insulinogenic (or fattening) since we demand the convenience and shelf stability of refined carbohydrates. It is simple to eat some crackers as a snack, but not a small piece of grilled salmon.

We must balance the time that we spend eating, and the time we spent NOT eating, or fasting. The very word 'break fast', which denotes the meal that breaks our fast implicitly acknowledges that we must spend some part of the day fasting, even if it is only 12-14 hours in the evening and night while we are mostly sleeping. Reducing the frequency of our meals will pay enormous dividends in weight loss efforts. Eliminating snacking (especially after dinner) and adding back periods of fasting are simple ideas practiced widely before the obesity epidemic. This is not likely coincidental.

10. Evidence Based Nutrition Should Be Incorporated In to Education Curricula For All Healthcare Professionals.

Given the immediate health threat posed by diet related disease we believe that it is imperative that education curricula for undergraduate, postgraduate and continuing medical education incorporate up to date evidence based nutrition. A global survey carried out by investment bank Credit Suisse worryingly revealed a substantial level of misinformation that exists amongst doctors with 92% believing that fat consumption could lead to cardiovascular issues, 87% suggesting obesity as a consequence with nutritionists coming up with broadly similar conclusions. Incorrectly 54% of doctors and 40% of nutritionists thought that eating cholesterol rich foods raises blood cholesterol. Most “shocking” in the report was that 83% of doctors thought butter was worse than margarine and 66% believed vegetable oils are beneficial to health. [42]

This is not only about preventing disease, but involves nutritional interventions that address and eliminate the root causes of chronic disease as opposed to a limited model of treating symptoms and risk factors with pharmacotherapy. The fact that prescription medications are now the third most common cause of death globally after heart disease and cancer should be a wake up call that the future of healthcare will require a strategy that incorporates evidence based lifestyle changes to treat illness in addition or often as an alternative to medical treatments which come with side effects. We support the Academy of Medical Royal Colleges and the BMJ who jointly provide a blueprint to reduce the harms of “too much medicine” as part of the Choosing Wisely campaign. One of their recommendations encourages educating the public to ask medical professionals whether there are “simpler or safer options” to taking medications. [43]

Healthy Eating Guidelines & Weight Loss Advice For The United Kingdom

As well as helping write this document the Public Health Collaboration (PHC) have also written a more extensive report on healthy eating guidelines and weight loss advice for the United Kingdom. This report can be downloaded from the PHC website at www.PHCuk.org/healthy-eating-guidelines-weight-loss-advice-for-the-uk/ where you can also find out more information.

References

1. Hex N, Bartlett C, Wright D, et al. Estimating the current and future costs of Type 1 and Type 2 diabetes in the United Kingdom, including direct health costs and indirect societal and productivity costs. *Diabetic Medicine* 2012;29:855–62.
2. Newton JN, Briggs AD, Murray CJ, Dicker D, Foreman KJ, Wang H, et al. Changes in health in England, with analysis by English regions and areas of deprivation, 1990–2013: a systematic analysis for the Global Burden of Disease Study 2013. *Lancet*. 2015. doi:10.1016/S0140-6736(15)00195-6.
3. Select Committee on Nutrition and Human Needs. *Dietary goals for the United States*. First ed. Washington: U.S. Govt. Print. Off., February 1977.
4. National Advisory Committee on Nutritional Education (NACNE). A discussion paper on proposals for nutritional guidelines for health education in Britain, 1983.
5. Credit Suisse. *Fat: The New Health Paradigm*: Research Institute, 2015:76.
6. Brownell KD, Warner KE. *The perils of ignoring history: big tobacco played dirty and millions died. How similar is big food?* *Milbank Q* 2009;87: 259–94.
7. Malhotra, A. (2013) The dietary advice on added sugar needs emergency surgery. *BMJ*, 346, f3199.
8. Sackner-Bernstein J, Kanter D, Kaul S. Dietary Intervention for Overweight and Obese Adults: Comparison of Low-Carbohydrate and Low-Fat Diets. A Meta-Analysis. *PLoS One* 2015;10(10):e0139817 doi: 10.1371/journal.pone.0139817[published Online First: Epub Date]].
9. Bazzano LA, Hu T, Reynolds K, et al. Effects of Low-Carbohydrate and Low-Fat Diets: A Randomized Trial Effects of Low-Carbohydrate and Low-Fat Diets. *Ann. Intern. Med.* 2014;161(5):309-18 doi: 10.7326/M14-0180[published Online First: Epub Date]].
10. Howard BV, Van Horn L, Hsia J, et al. Low-fat dietary pattern and risk of cardiovascular disease: the Women's Health Initiative Randomized Controlled Dietary Modification Trial. *JAMA* 2006;295(6):655-66 doi: 10.1001/jama.295.6.655[published Online First: Epub Date]].
11. Harcombe Z, Baker JS, Cooper SM, et al. Evidence from randomised controlled trials did not support the introduction of dietary fat guidelines in 1977 and 1983: a systematic review and meta-analysis. *Open Heart* 2015;2(1) doi: 10.1136/openhrt-2014-000196[published Online First: Epub Date]].
12. Chowdhury R, Warnakula S, Kunutsor S, et al. Association of Dietary, Circulating, and Supplement Fatty Acids With Coronary Risk: A Systematic Review and Meta-analysis. *Ann. Intern. Med.* 2014;160(6):398-406 doi: 10.7326/M13-1788[published Online First: Epub Date]].

13. de Souza RJ, Mente A, Maroleanu A, et al. Intake of saturated and trans unsaturated fatty acids and risk of all cause mortality, cardiovascular disease, and type 2 diabetes: systematic review and meta-analysis of observational studies. *BMJ* 2015;351 doi: 10.1136/bmj.h3978[published Online First: Epub Date]].
14. Thorning TK, Raziani F, Bendtsen NT, Astrup A, Tholstrup T, Raben A. Diets with high-fat cheese, high-fat meat, or carbohydrate on cardiovascular risk markers in overweight postmenopausal women: a randomized crossover trial. *The American journal of clinical nutrition* 2015 doi: 10.3945/ajcn.115.109116[published Online First: Epub Date]].
15. Crichton GE, Alkerwi Aa. Whole-fat dairy food intake is inversely associated with obesity prevalence: findings from the Observation of Cardiovascular Risk Factors in Luxembourg study. *Nutrition Research* 2014;34(11):936-43 doi: 10.1016/j.nutres.2014.07.014[published Online First: Epub Date]].
16. Mozaffarian D. Saturated fatty acids and type 2 diabetes: more evidence to re-invent dietary guidelines. *Lancet Diabetes Endocrinol* 2014;2:770-2
17. Chakrabarti S, Freedman JE. *Review: nutraceuticals as antithrombotic agents. Cardiovasc Ther* 2010;28:227-35.
18. Veerman J Lennert. Dietary fats: a new look at old data challenges established wisdom *BMJ* 2016; 353 :i1512
19. Eddy D, Schlessinger L, Kahn R, Peskin B, Schiebinger R. Relationship of insulin resistance and related metabolic variables to coronary artery disease: a mathematical analysis. *Diabetes Care*. 2009;32(2):361-6.
20. Harcombe Z, Baker J. Plant Sterols lower cholesterol, but increase risk for Coronary Heart Disease. *Online J. Biol. Sci.* 2014;14(3):167-69 doi: 10.3844/ojbssp.2014.167.169[published Online First: Epub Date]].
21. Feinman RD, Pogozelski WK, Astrup A, et al. *Dietary carbohydrate restriction as the first approach in diabetes management: critical review and evidence base. Nutrition* 2015;31:1-13.
22. Unwin D, Unwin J. Low carbohydrate diet to achieve weight loss and improve HbA1c in type 2 diabetes and pre-diabetes: experience from one general practice. *Practical Diabetes* 2014;31(2):76-79 doi: 10.1002/pdi.1835[published Online First: Epub Date]].
23. Credit Suisse - Sugar Consumption at a crossroads
24. Lustig RH: Sickeningly sweet: does sugar cause type 2 diabetes? YES. *Can J Diab* (in press).
25. Isocaloric fructose restriction and metabolic improvement in children with obesity and metabolic syndrome. Lustig RH, Mulligan K, Noworolski SM, Tai VW, Wen MJ, Erkin-Cakmak A, Gugliucci A, Schwarz JM. *Obesity* (Silver Spring). 2016 Feb;24(2):453-60. doi: 10.1002/oby.21371. Epub 2015 Oct 26.

26. Added sugar intake and cardiovascular diseases mortality among US adults. Yang Q, Zhang Z, Gregg EW, Flanders WD, Merritt R, Hu FB. *JAMA Intern Med.* 2014 Apr;174(4):516-24. doi: 10.1001/jamainternmed.2013.13563.
27. *Free Radic Biol Med.* 2001 Dec 1;31(11):1388-95. The stomach as a bioreactor: dietary lipid peroxidation in the gastric fluid and the effects of plant-derived antioxidants. Kanner J, Lapidot T.
28. *Curr Atheroscler Rep.* 2009 Nov;11(6):403-10. Impact of circulating esterified eicosanoids and other oxylipins on endothelial function. Shearer GC, Newman JW.
29. *BMJ.* 2013 Feb 4;346:e8707. doi: 10.1136/bmj.e8707. Use of dietary linoleic acid for secondary prevention of coronary heart disease and death: evaluation of recovered data from the Sydney Diet Heart Study and updated meta-analysis. Ramsden CE1, Zamora D, Leelarthaepin B, Majchrzak-Hong SF, Faurot KR, Suchindran CM, Ringel A, Davis JM, Hibbeln JR.
30. Christakis G, Rinzler SH, Archer M *et al.* Effect of the anti-coronary club program on coronary heart disease. Risk-factor status. *JAMA* 1966;198:597-604
31. Pearce ML, Dayton S. Incidence of cancer in men on a diet high in polyunsaturated fat. *Lancet* 1971;1:464-7.
32. *Am J Epidemiol.* 1998 Feb 15;147(4):342-52. Adipose tissue omega-3 and omega-6 fatty acid content and breast cancer in the EURAMIC study. European Community Multicenter Study on Antioxidants, Myocardial Infarction, and Breast Cancer. Simonsen N1, van't Veer P, Strain JJ, Martin-Moreno JM, Huttunen JK, Navajas JF, Martin BC, Thamm M, Kardinaal AF, Kok FJ, Kohlmeier L.
33. *Cancer Res.* 1988 Dec 1;48(23):6642-7. Effect of different levels of omega-3 and omega-6 fatty acids on azoxymethane-induced colon carcinogenesis in F344 rats. Reddy BS, Sugie S.
34. Fildes A *et al.* Probability of an Obese Person Attaining Normal Body Weight: Cohort Study Using Electronic Health Records. *Am J Public Health.* 2015;105:e54-e59
35. Malhotra A, DiNicolantonio JJ, Capewell S. It is time to stop counting calories, and time instead to promote dietary changes that substantially and rapidly reduce cardiovascular morbidity and mortality. *Open Heart* 2015;2(1) doi: 10.1136/openhrt-2015-000273[published Online First: Epub Date].
36. Mann T, Tomiyama AJ, Westling E, *et al.* *Medicare's search for effective obesity treatments: diets are not the answer.* *Am Psychol* 2007;62:220-33.
37. Wing RR, Bolin P, Brancati FL, *et al.* *Look AHEAD Research Group.* *Cardiovascular effects of intensive lifestyle intervention in type 2 diabetes.* *NEngl J Med* 2013;369:145-54.
38. Luke A, Cooper RS. *Physical activity does not influence obesity risk: time to clarify the public health message.* *Int J Epidemiol* 2013;42:1831-6.

39. It is time to bust the myth of physical inactivity and obesity: you cannot outrun a bad diet . A Malhotra, T Noakes, S Phinney *Br J Sports Med* *bjsports-2015-094911* Published Online First: 22 April 2015 doi:10.1136/bjsports-2015-094911
40. Popkin BM. Does hunger and satiety drive eating anymore? *Am J Clin Nutr* 2010;91:1342–7
41. Cameron JD. 6 meals per day does not result in greater weight loss. *Br J Nutr.* 2010 Apr;103(8):1098-101
42. Fat; The New Health Paradigm - Credit Suisse 2015
43. Malhotra A, Maughan D, Ansell J, Lehman R, Henderson A, Gray M et al. Choosing Wisely in the UK: the Academy of Medical Royal Colleges' initiative to reduce the harms of too much medicine *BMJ* 2015; 350 :h2308