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We live in difficult times. So-called Fake News is becoming more common. Fact checkers have a hard time keeping up with the lies and flip-flops of politicians. One even gets the impression that deception has become the new norm in marketing and commercial public relations. Most individuals now face the hopeless task of sorting truth from deception.

On December 20 the respected journal, the *Annals of Internal Medicine,* published a scathing attack on global health advice concerning limiting the consumption of sugar and claimed that warnings to cut sugar cannot be trusted because they are based on weak and inconsistent evidence. Critics immediately pointed out in an accompanying editorial that the funding source for this study was the North American branch of the *International Life Sciences Institute* which is in fact a trade group representing The Coco-Cola Company, Dr Pepper Snapple Group, The Hershey company, Mars, Inc., Nestle USA and PepsiCo, among others, and that some of the authors also had serious conflicts of interest. One of the authors was a member of the scientific advisory board of one of the world's largest suppliers of high-fructose corn syrup. The *New York Times* quoted Marion Nestle, a professor of nutrition as saying "This comes right out of the tobacco industry playbook: "cast doubt on the science." The guidelines examined by the authors were published between 1995 and 2016 and critics pointed out that one would expect inconsistencies as scientific knowledge evolved, and in fact, the most recent guidelines from Public Health England, the World Health Organization and the US department of Agriculture showed remarkable consistency in their recommendations to limit sugar intake.

In a letter to the editor published in the same issue, four scientists summarized a study which examined the correlation between pro-industry results of experimental studies and meta-analyses and industry support of the research. Out of 26 industry-supported studies, 25 produced industry-favorable results whereas, only 1 in 34 of non-industry supported studies presented industry favorable results. Also, a professor of nutrition at the University of North Carolina at Chapel Hill, Dr. Barry Popkin, told the *Times* that he was stunned that the paper was even published at all because the authors "ignored hundreds of randomized controlled trials that have documented the harms of sugar. They ignored the real data, created false

scores, and somehow got through a peer reviews system that I cannot understand," he said. "It is quite astounding.

One can also wonder if the timing of the article was to coincide with the release of Gary Taubes' new book *The Case Against Sugar.* Taubes is one of the most highly respected medical and nutritional journalists in the world and the industry will not welcome his new book which became available on December 27. IHN readers may recall his monumental work *Good Calories, Bad Calories* which discussed the mythology associated with dietary fats and carbohydrates.

Wishing you and your family continuing good health,

William R. Ware, PhD, Editor

Highlights

CAUSES OF DIABETES AND IMPLICATIONS FOR PREVENTION

SUMMARY

Research over the past 6 years has resulted in a theory of how diabetes develops which is supported with a remarkably successful dietary intervention that reverses diabetes unless it is of long duration. This research provides guidance regarding how to prevent diabetes and also how to deal with prediabetes. Early detection of prediabetes is important and implies routine screening from an early age, ideally involving both fasting blood (plasma) glucose and glycated hemoglobin (HbA1c). That type 2 diabetes is a huge and worldwide problem is known to most readers of this newsletter. Undiagnosed diabetes is remarkably common. Thus the question—what do the causes of diabetes being postulated suggest for prevention. The simple answer is to not consume more calories than the minimum your body needs and annually screen for prediabetes and diabetes. Also, get a glucose meter and monitor fasting blood glucose.

In 2011 Professor Roy Taylor of Newcastle University in the UK and his research associates published the results of a small clinical study which demonstrated that an eight-week 600-700 calorie per day diet was able to reverse type 2 diabetes returning the subjects to near normal glucose metabolism.[1] It was important that the post-diet weight was maintained when more calories were of necessity introduced.[2] The intervention is called the Newcastle Diet. Taylor reports that for motivated individuals who do not regain weight, maintenance of normal glucose metabolism has been

reported for up to 3 years in studies,[3] but the record is about 12 years.[4] The results from the Newcastle Diet study are totally contrary to the conventional wisdom but consistent with repeated observations of the elimination of diabetes through the weight loss produced by bariatric surgery. The paper appeared in the major diabetes journal *Diabetologia.* Taylor also presented this data and his views in more detail in the 2012 Banting Memorial Lecture which was published in *Diabetic Medicine.*[5] In 2013 Taylor published a lengthy article in *Diabetes Care* on his views of what causes diabetes and why it is reversible.[6] This article elaborated on his explanation of the reversibility of diabetes with short-term severe calorie restriction. This was followed by a bigger diet study which confirmed the initial study and also found that the dietary approach failed for some if the diabetes had existed for over more than 8-10years.[3,7] Taylor and his group have continued to publish on this subject, expanding the evidence for their model.[8,9] One advantage they have is also being experts in MRI imaging of organ fat since changes in liver and pancreatic fat are an essential feature of their theory. Furthermore, even in the original diet study they investigated the impact on the two phases of insulin excretion following a glucose challenge, and this measurement goes considerably beyond the simple blood tests uses to confirm or reject suspected diabetes. In what follows, we will briefly examine the lessons that can be learned from the work of Taylor and colleagues.

The path from normal glucose metabolism to type 2 diabetes in a simplified version is as follows. We obtain the energy required to exist and function mostly by "burning" carbohydrate-derived glucose, and as well fat, both dietary and when necessary derived from body stores by mechanisms that evolved to protect from periods of food shortage or famine. The Taylor hypothesis goes as follows.[6]

- First, mostly due to taking in more calories than needed, muscle insulin resistance develops, commonly accompanied by weight gain or obesity. Insulin is required for the cellular absorption of glucose as a necessary step prior to energy production. Insulin resistance involves decreased cellular response to insulin and the need for more circulating insulin to cope with circulating glucose. Thus increasing insulin levels are an important initial marker.

- When more calories are consumed than needed, the liver converts some carbohydrates into fat and stores some of it. Thus the liver builds up excess fat including triacylglycerol. At some point the liver starts exporting fats for use and storage elsewhere. One destination is the pancreas where triacylglycerol is toxic to the beta cells which produce insulin, and a decline in insulin production in response to circulating glucose commences. The accumulation of liver and pancreatic fat can be documented accurately by MRI scans. The decline in beta cell function aggravates the elevated blood sugar problem and eventually the individual is diagnosed with diabetes having gone through a prediabetic phase, generally ignored or never diagnosed.

- As the destruction of beta cells continues, diet and diabetes medications ultimately fail to control the elevated blood sugar and the individual becomes what is termed an insulin depended diabetic, the last stage in the evolution of the failure of beta cell function and normal glucose metabolism.

What is the evidence? The observations on reversal of type 2 diabetes with rapid weight loss alone with no diabetic medications and the necessity of subsequent weight maintenance confirm that if the primary influence of positive calorie balance is removed then the process of development of diabetes is reversible with the documented disappearance of accumulated liver and pancreatic fat and the restoration to normal of the initial insulin response to circulating glucose (called the first phase). Furthermore, it appears not to matter if the weight loss still leaves the individual overweight or even obese.[6] The necessity of large fast weight loss does not require an absolute target weight, just the weight loss process. The numerous studies from Taylor's group from 2008 to 2016 appear to be studiously ignored by mainstream medicine, especially in North America, even though published in major diabetes journals, and no drug is involved that can be promoted on TV and to doctors by drug reps.

The above sequence of events postulated to result in diabetes gives rise to the commonly held view that type 2 diabetes is a progressive disorder only modestly influenced by medications and unless death from an unrelated problem intervenes, the insulin dependent individual will generally experience progressive adverse effects including vision problems, circulation problems that can require amputation, cardiovascular disease, painful peripheral neuropathy now well described in TV ads on the US evening network news, and a shortened life expectancy. The hope and expectations generated by early control of blood sugar with medications or as it is said, the proper *management* of the disease, is now gone. Some diets and exercise programs have a modest impact, but regression to normal glucose metabolism is very rare. The progression is inexorable, diabetes is a lifelong condition, and at this point the diabetic must realize that they have an incurable condition according to the almost universally held view. Note we are discussing only type 2 diabetes.

Obviously the work of Taylor and colleagues contradicts this conventional wisdom. An intervention over the early years (up to 8-10) of diabetes yielding in the majority of patients complete remission at the end of eight weeks is accomplished by a short term highly calorie restricted diet. The diabetes is no longer present. The same happens to many after they have undergone bariatric surgery which also produces a large weight loss similar to the Newcastle Diet.[10]

Because being overweight or obese is commonly cited as a risk factor for type 2 diabetes, one might assume that normal weight individuals are immune. Unfortunately this is not the case and in fact normal weight persons not only are not exempt but weight loss even if the starting weight is normal produces the above described reversal. There is considerable data to substantiate this conclusion. Taylor and Holman hypothesize that rather than weight or body mass index, the extent of liver and pancreatic fat accumulation is the critical factor but modulated by individual

susceptibility to the local effects of this lipid excess.[11] This seems to underscore the importance for screening for prediabetes or diabetes independent of weight in order to achieve timely intervention. Prediabetics can cure themselves with the Newcastle diet, perhaps even in fewer weeks. Progress can be monitored with the glucose meter diabetics use and a good picture can be obtained by measuring fasting, pre-meal and 2 hours post-meal values along with daily weight and even a food diary.

What does all this mean in terms of preventing diabetes? First the question to ask is, do I have prediabetes or type 2 diabetes? Routine physical exams should provide the answer and no excuse should be accepted for not including the required simple blood tests. If prediabetes is present this implies the early recognition of consuming more calories than needed to supply the daily needs, independent of whether one is lean or has normal weight or is obese and suggests the importance of maintaining normal weight and annual blood tests. The most noticeable warning sign would be gradual weight gain. This could be followed by prediabetes which is manifest by mildly elevated fasting blood glucose (dependent almost solely on glucose production in the liver, generally overnight) and/or a slightly elevated HbA1c, a blood component which gives a 3-month average of blood glucose. Either indicates the need for intervention to reverse what is occurring.

In this context, a study[5] that followed the development in diabetes, and found that over the first 12 years the fasting glucose slowly and linearly increased from 5.5 to 6.0 mmol/L and then over two years shot up to 7.5 mmol/L, i.e. a long prediabetic period but continuous slow progression of fasting glucose which appears to be an early warning sign and is easy to monitor, even at home. It is natural to view the change from 5.5 to 6.0 as not very concerning, but this appears to be untrue.

There will be many individuals who will be overweight but show no signs of prediabetes or diabetes.[12] While trying to lose weight is still indicated on general principles, regular monitoring for signs of prediabetes is still essential. It turns out that a high percentage of diabetics are in fact obese, but a much smaller number of obese are diabetic. As discussed above, normal weight is no guarantee of low or negligible risk. Obviously there is a significant variability in susceptibility and probably there is a genetic component.[11] In addition, a substantial number of diabetics are undiagnosed and this also applies to prediabetes.

It is not surprising in view of the above discussion that non-alcoholic fatty liver disease (NAFLD) also carries a considerably enhanced risk of diabetes and thus elevated liver enzymes should also raise a red flag and their measurement should be part of routine physical exams. If NAFLD is present or developing, this seems to call for frequent screening for prediabetes or diabetes.

The Newcastle diet was described and discussed in the October 2014 issue of *International Health News*. While it involves a liquid diet for the 600 calories and supplementation with vegetables for the other 100 calories, the liquid diet is not special, just convenient, and in fact necessary in trials because of the need of a standardized

diet for all. The liquid diet was not carbohydrate restricted, having about 46% energy from carbohydrates. While apparently not as yet fully investigated, Taylor claims that it makes little difference what is eaten and patient satisfaction helps adherence to the program.[4] The diet must rapidly eliminate excess energy intake, and force the decrease of liver fat, and stop the export of triacylglycerol. The excellent studies from Taylors's group make it clear that this is exactly what happens with this diet. Slow weight loss would probably allow the body to compensate without correcting the fundamental liver and pancreatic problems. What is perhaps most significant if not sensational is that pancreatic beta cell first response to glucose is restored, frequently significantly in the first week, unless the damage is too extensive (greater than about 8-10 years of diabetes). It is interesting that muscle insulin resistance does not change materially either during the onset of diabetes or the reversal.[6] As of 2016, the longest reversal has lasted almost 12 years.[4]

Those familiar with the ongoing diet debate between the "a calorie is a calorie" vs. the view that sugar, excessive refined carbohydrates, junk food etc. are the villains in diabetes and other chronic diseases will realize that the above does not resolve this issue but suggests that for diabetes the essential dietary aspect is excess calorie intake. This is because the central feature of the Taylor hypothesis involves excess calories being converted into liver fat and the exporting of excess liver triacylglycerol to the pancreas where the key damage occurs. There appears to be no reason why one cannot simply take their normal diet and cut back on all items to achieve 700 calories. However, it is important to get enough vitamins and minerals. As long as the diet, which is more or less a starvation diet, results in all the food normally metabolized for energy being used for only that along with a large deficit. Even a diet of just ice cream, celery and a multi-vitamin-mineral pill probably would work but it is unlikely that this will ever be scientifically tested but perhaps is a great idea for diabetic children, but no studies on children or adolescents appear to have been published using the Newcastle approach. See the October 2014 IHN for more details concerning the diet, the suggested restrictions and precautions. The diet should be supervised by a health care professional. Stopping diabetic drugs should be orchestrated by a physician. There are special issues for pregnant women.

An almost universal observation is that in small, modern populations who live in isolation and eat as did their ancestors over centuries, diabetes is very rare. The diets varied dramatically but these people had no access to refined sugar or starch or processed and junk foods. If so-called modern civilization moves in on them (generally government orchestrated) or some members migrate to where modern civilization governs how they eat, the prevalence of diabetes increases rapidly to that of the new environment. The problem in understanding why this happens is sorting out the prevalence of excess calories and the presence of a dramatic change in diet which generally involves the introduction of much more sugar and refined starch. These are of course not independent. For a comprehensive and well documented discussion, read Gary Taubes' new book *The Case Against Sugar.* It is not a simple issue.

TREATING EXCESS STOMACH ACID AND RISK OF CHRONIC KIDNEY DISEASE

SUMMARY

This is a perfect example of a drug intervention that alters a basic aspect of human physiology, stomach acidity. Another involves the statins which inhibit a pathway which then has repercussions for a large number of vital functions and pathways downstream. The cavalier attitude toward such interventions is a manifestation of a lack of appreciation of the magnitude of the complexities of human biochemistry and physiology. It should be a general principle in medicine that one does not mess around with major physiology and biochemistry that has evolved over eons without considering carefully what is really being done and reflecting on how little we really know about the complex interrelations present. As regards stomach acid, it appears that one should significantly reduce it only temporarily and for very compelling reasons.

A study just published has examined the question of an association between kidney disease and the use of the anti-acid class of drugs called proton pump inhibitors (PPIs). The study was prompted by the high prevalence of chronic kidney disease in the US (approximately 14% of adults) and the fact that the increasing prevalence cannot be explained by trends in known risk factors.[13] Over 10,000 subjects were followed for about 20 years. The authors provided estimated absolute risks of chronic kidney disease over 10 years. For those taking PPIs, the risk was 11.8% whereas for those who did not use them, 8.5%, giving a 3.3% absolute risk increase. The comparison was only between users and non-users and thus there was no duration of use or dose results provided. Ten years does not mean ten years of use.

Chronic kidney disease is a serious matter and can lead to dialysis which has its own set of serious side effects, and eventually to the need for kidney transplant. Anyone on dialysis will tell you that it has a significant impact on quality of life, especially if the hospital is remote.

Indications for the use of PPIs include so-called heartburn and stomach acid related disorders such as gastroesophageal reflux disease (GERD) which can involve serious damage to the esophagus which in turn is a risk factor for Barrett's esophagus which is a risk factor for cancer. Damage includes narrowing of the esophagus or inducing ulcers in the esophagus. In addition PPIs are indicated for stomach ulcer prevention and treatment. Most treatments are short term, but the availability of this class of drug without prescription has resulted in widespread use which at least in some cases involves taking the drug over the long term for problems outside the above indications. In the paper describing the research cited above, the authors comment that in 2013 more than 15 million Americans used prescription PIPs and cite a study suggesting that up to 70% of these prescriptions are without a justifying indication and that 30% of PIP users could discontinue use without developing symptoms. They call for reduction of unnecessary use. In the case of unnecessary use, any side effect is has no balancing benefit.

On the risk side of the balance sheet, not only do we have as discussed an increased risk of chronic kidney disease, but according to US FDA drug safety communications:

- Risk of *Clostridium difficile* associated diarrhea.
- Risk of low magnesium levels, a serious problem frequently overlooked and not routinely checked with blood tests.
- Possible increased risk of fractures of the hip, wrist, and spine.

In addition, there is a fundamental issue. Humans evolved to have high stomach acidity (low pH) which has been the case for eons. The acid is hydrochloric acid excreted into the stomach. The high acidity is required for the converting of protein into its constituent amino acids which are then metabolized, and is also required for the absorption of some nutrients. The high-acid environment is also a shield against undesirable bacteria which are prevented from colonizing in the stomach or bowel. This suggests that the reasons must be compelling for changing this fundamental aspect of our gastrointestinal system. To make such a profound change as reducing the acidity to near neutral over a long period of time would thus be expected to have deleterious consequences, and yet this is done by millions of individuals, in many cases simply because they find occasional indigestion with acid reflux unpleasant. In fact, in some cases, this type of indigestion is resolved by *increasing stomach acid, not decreasing it.* Read Dr. Jonathan Wright's book *Why Stomach Acid is Good for You: Natural Relief from Heartburn, indigestion, Reflux and GERD.*

The pharmaceutical industry tends to minimize the importance of PPI side effects and there are not enough industry independent studies to have a clear picture of the problem. Thus one frequently sees the argument that there is insufficient data to justify concern for some proposed side effect. However, absence of evidence of risk does not imply that there is no risk. Furthermore, side effects experienced by long-term users who are obtaining the PPIs over-the-counter are a vast unknown, and yet these may represent a majority of long-term users.

NEWS BRIEFS

ADOLESCENT BARIATRIC SURGERY REVERSES TYPE 2 DIABETES

In a multicenter study just reported in the *New England Journal of Medicine* and also presented at the Obesity Society annual meeting in Los Angeles, the health status three years after bariatric surgery was examined in a group of 242 adolescents.[14] Most underwent the Roux-en-Y gastric bypass which reroutes what is eaten to mostly bypass the stomach but 28% were treated with the sleeve gastrectomy which restricts flow into the stomach. The average age was 17 years and the mean weight was 327 pounds i.e. morbid obesity that was probably intractable.

Three years after the surgery, the mean weight had decreased by 27% with the bypass and sleeve approaches yielding almost the same weight reductions. Remission of abnormal kidney function occurred in 89%, remission of elevated blood pressure

occurred in 74% and remission in dyslipidemia (low HDL, high triglycerides) was observed in 66%. These favorable changes occurred without a return anything close to a normal weight for an adolescent.

That is not all. The results for diabetes were nothing short of sensational. Before surgery, 29 (13%) had type 2 diabetes. At 3 years, 19 of 20 (95%) participants who had data available for evaluation were cured of their diabetes with HbA1c at 5.3% and fasting blood glucose at 88 mg/dL and presumably no diabetes medications. Prediabetes was found at baseline in 19 participants for whom end of study data was available, and 76% no longer had prediabetes after surgery.

However, surgical intervention is not without its post-surgical complications. In this study, 8% had major complications within 30 days and 13% underwent additional abdominal surgery within the subsequent 3 years.

The authors conclude that the study supports the benefits of this type of surgery and indicates significant durability of multiple benefits, at least over 3 years for severely obese adolescents. For these subjects, there does not appear to be an alternative therapeutic approach with these results which subjects are likely to undertake with significant adherence to the protocol.

While major drastic surgery may seem like overkill for obesity, several decades of experience with obesity clearly indicate that it presents an almost intractable problem using only the conventional approach of diet and exercise and weight loss drugs have a significant downside. Individuals eligible for bariatric surgery may want to first consider the 8-week Newcastle diet which will also yield significant weight loss and unless the diabetes is of long duration, also cure it. There are no adverse side effects whereas for bariatric surgery this is not necessarily the case, but the bypass surgery is presumably difficult to reverse. For severely morbidly obese, the weight loss from an 8-week 600-700 calorie diet might not produce sufficient weight loss to be helpful.

STATINS AND INCIDENCE OF ALZHEIMER'S DISEASE

It is generally accepted in mainstream medicine that no effective treatment exists for Alzheimer's disease. On December 12 the British newspaper *The Daily Telegraph* carried an article informing readers that statins may reduce the risk of Alzheimer's disease. The relative risk reductions cited were between 12% and 15%. I read this while on a flight home from Europe and was eager to see the actual scientific report which had just been published online.[15] It appears that this was not really newsworthy at all. Even though 400,000 were followed for a mean of about 7 years, the number of users needed to treat (NNT) to prevent one case was for high and low statin use 200 and 285 respectively over 7 years. Calculated from adjusted data, I find the NNT ranged for white men and women from 500 to 250 and only 3 out of five of the statins yielded statistical significance. These NNT are big numbers. Think of an auditorium filled with 200-300 people listening to a talk on reducing risk of Alzheimer's and they all decide to take statins. They return in 7 years and only one raises his or her hand when asked how many benefited. The authors concluded that statin treatment with the right statin "may

provide a relatively inexpensive means to lessen the burden of Alzheimer's disease." Even qualifying with the word "may" does not reveal the obvious lack of effectiveness by the standards of many clinicians if absolute rather than relative benefits are considered, i.e. the NNT. But the reader of the newspaper article probably gets a somewhat deceptive impression and might even conclude that they are lucky they are on statins which also, they believe, reduce their risk of cardiovascular event by anywhere from 20% to 35%. As readers of IHN know, this is another highly deceptive impression.

LOW CALORIE SWEETENERS – AT LEAST ONE OF THESE CAN BE VIEWED AS TERRIBLY TOXIC

An article published in the *Journal of the Academy of Nutrition and Dietetics* used recent population data to examine the question of the consumption of low-calorie sweeteners among children and adults in the US.[16] The researchers found that 25% of children and 41% of adults reported consuming LCSs, mostly in beverages. These numbers represent a 200% increase in LCSs for children and a 54% increase for adults from 1999 to 2012. The higher consumption was mostly from food and beverage sources and the paper presents an impressive list of commonly consumed products.

While this move to lower sugar consumption is noteworthy, the big question is the percentage of aspartame (trade name NutraSweet) involved. The study did not address this question and one is led to suspect that the significant place aspartame occupies in the LCS world is underappreciated. As discussed in IHN, aspartame breaks down upon ingestion to produce one molecule of methyl alcohol for each molecule of the sweetener. Methyl alcohol is also called methyl hydrate, methanol or wood alcohol and is sold in large quantities in paint departments and stores and of course marked as poisonous. While humans are able to excrete methanol as it circulates freely throughout the body including the brain, some is converted by an enzyme located in a number of critical areas and organs with the resultant production of formaldehyde which reacts rapidly and can cause extensive cellular, tissue and organ damage. The damage can be subtle, develop very slowly and thus go unnoticed even for years, and has been implicated in the development of a number of chronic diseases but this possibility is being ignored and studying this issue is not a good way to advance one's academic career. The toxicity can be minimized by consuming ordinary (drinking) alcohol and getting a patient drunk is a common emergency approach to methanol poisoning, since the enzyme is almost totally occupied with dealing with large amounts of ethanol and the methanol has time to be excreted. Methanol is classed as a poison, but mostly due to the conversion to formaldehyde. In fact it can be argued that a glass of an alcoholic beverage with each meal that includes aspartame could be justified. Rum and coke comes to mind! We also have the curious situation where there is concern of formaldehyde outgassing, for example, from new flooring materials and being inhaled, but a much more serious threat exists in processed foods and beverages but is ignored—a taboo subject.

Since the above facts regarding aspartame, methanol and formaldehyde are not widely known in spite of the efforts of scientists such as Woodrow C. Monte, PhD (see *While Science Sleeps, a Sweetener Kills)*; the food and beverage industry appears to have

little to worry about. After all, only a small percentage of the population even understands what an enzyme is nor can they appreciate the chemistry, for example in the liver or blood vessels which creates a fearsome toxin from a sweetener one can even purchase by the pound in the grocery store. However, aspartame is only one of a number of LCSs in current use. Critics might suggest that industry sponsored safety tests on the non-aspartame sweeteners possibly leave something to be desired. Finally, this same enzyme is involved in converting the alcohol many readers routinely drink (ethanol, ethyl alcohol) into what appears to be a relatively harmless metabolite. It is not the enzyme that is a bad actor; it is what one "feeds" it which is called a substrate. See the Research review in IHN, JUN 12 updated DEC12 concerning the dangers of aspartame.

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