Health promotion when the 'vaccine' does not work

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Introduction

The control of an epidemic of vaccine-preventable disease involves a spectrum of public health functions. At one end lies the development, testing and production of the vaccine while at the other, programs are developed to ensure maximum coverage of the population. Front-line workers concentrating on delivering the vaccine depend on those at the manufacturing end to ensure the vaccine's safety and efficacy. The course of the epidemic is tracked and, if the vaccine were to fail, measures would be taken to develop an effective replacement. This is basic public health practice.

How is it, then, that we find ourselves in the midst of an epidemic where we continue to administer a 'vaccine' that does not work in the face of compelling evidence to that effect, where the epidemic continues to advance in spite of our best efforts to deliver the 'vaccine', where we accept the premise that the failure is related to delivery, not efficacy, where we ignore evidence that a different approach shows promise and where research into alternatives may actually have been discouraged? In the face of an epidemic of infectious disease this would be unthinkable, yet this is precisely what is happening where the inter-related epidemics of obesity, metabolic syndrome and type 2 diabetes are concerned and where our intervention, or 'vaccine', is the prescription of a low-fat diet and exercise.

This is an edited version of a keynote address given at the 16th Annual Health Promotion Conference held in Alice Springs on 23-26 April 2006.

The 'vaccine'

Excluding alcohol, our calories come from three macronutrients: fat, protein and carbohydrates. There is a limit to how much protein we can eat. A minimum amount is needed to prevent wasting but too much will cause malaise.[1] We are advised to get about 15% of our calories from protein, leaving fats and carbohydrates to provide most of our energy. Since the conventional wisdom, our 'vaccine', prescribes that fat comprise no more than 30% of calories, the recommended carbohydrate

Abstract

The epidemics of obesity, metabolic syndrome and type 2 diabetes have worsened over the past decades. During this time our preventive and therapeutic approach (the 'vaccine'), consisting of a low-fat diet and exercise, has remained fundamentally unchanged. A case is made that these conditions are inter-related and may be caused by a single underlying factor related to the carbohydrate content of diet. The validity of the present approach is challenged when those most knowledgeable in its application succumb to diseases it is meant to prevent. Others argue against the status quo that a low-carbohydrate diet may be more beneficial. A strong belief in the present approach discouraged research into low-carbohydrate diets until recently. Several studies have now demonstrated their benefits and are refuting old claims that they cause harm. Aboriginal people suffer more acutely from the epidemics in question and their dietary history suggests that a sudden increase in carbohydrates is to blame. Recent studies and a case history demonstrate that carbohydrate consumption can drive appetite and over-eating while carbohydrate restriction leads to weight loss and improvement in the markers for metabolic syndrome and type 2 diabetes. The growing evidence in support of low-carbohydrate diets will encounter resistance from economic interests threatened by changes in consumption patterns.

*Health Promotion Journal of Australia 2006;**17**:91-6*

So what?

In the face of mounting new evidence, our rigid adherence to a low-fat diet approach can no longer be justified. We must be prepared to offer more flexible dietary recommendations based on emerging evidence that carbohydrate restriction has a salutary effect on obesity, metabolic syndrome and type 2 diabetes.

intake becomes, by default, 55% of calories. And why not, as carbohydrates are viewed as benign foods, even in their refined configurations, as opposed to dietary fat, which, as we all know, clogs the arteries? These proportions are reflected in government food guides and the recommendations of the main disease-related institutions such as the diabetes associations, the heart and stroke foundations and the cancer agencies.[2-5] Although there are occasional updates, nothing has fundamentally changed in these recommendations over the past 30 years, during which time we have witnessed an extraordinary rise in obesity, metabolic syndrome and type 2 diabetes, conditions these recommendations are meant to prevent.[6,7]

Even as the rise in prevalence of these conditions has become obvious, we have stayed the course, secure in our knowledge that our 'vaccine' would be effective, if only we could do a better job of delivering it.

A disease continuum

Dr Paul Zimmet, a prominent Australian diabetologist, suggests that type 2 diabetes, or glucose intolerance, is just the visible tip of an iceberg consisting of the conditions that constitute metabolic syndrome (MetS): hyperinsulinemia, dyslipidemia, hypertension, abdominal obesity and insulin resistance.[8] Since overweight and obesity are the usual precursors of MetS, one can argue that these conditions constitute an even larger iceberg under the iceberg described by Zimmet.[9]

A further case can be made for connecting the causality of these conditions. In a recent article in *Diabetic Care*, Pladevall et al. reported on their confirmatory factor analysis which determined not only that all the factors that constitute MetS belong together, but that there is a single underlying factor linking them.[10] Volek and Feinman, writing in *Nutrition and Metabolism*, reviewed the literature on low-carbohydrate diets and concluded that since all the factors that comprise MetS reverse towards normal when carbohydrate consumption is reduced, MetS could be defined by its response to dietary carbohydrate restriction.[11]

Returning to Zimmet's concept of an iceberg consisting of related conditions, it could be argued that type 2 diabetes and MetS and possibly obesity and overweight actually constitute a single syndrome of carbohydrate intolerance. If this is the case, our approach to the prevention and management of these conditions stands in sharp contrast to that of other dietary intolerances. Consider how gluten intolerance, lactose intolerance and phenolketonuria are managed. In all cases, the first line of treatment is an elimination diet. Not so with carbohydrate intolerance, however, where a so-called 'balanced' diet, 55% of which consists of the very foods that are not tolerated, is prescribed.[12] It was not always so. In the days before the discovery of insulin, the standard treatment for diabetes was a very low carbohydrate diet.[13]

Four cardiologists (Three Funerals and a Scolding)

In medicine, we observe a time-honoured post-mortem tradition of striving to learn from each passing, better to serve those who are still living. It is in this spirit that the recent deaths of some colleagues are examined.

Dr Frederick Cobb was a learned colleague at the pinnacle of knowledge in the prevention and management of cardiovascular disease. He was director of the Duke Program for Prevention and Treatment of Heart and Vascular Disease at the prestigious Duke University Medical Centre in the eastern United States. His approach to prevention conformed to the accepted standard of exercise plus a polypharmacy of statins, antihypertensives and oral hypoglycemics. He recently died of a heart attack while riding his exercise bicycle. He was 67.

Dr Lynn Alan Smaha, also a leader in this field, was president of the American Heart Association, a fervent disciple of the hypothesis that high lipids lead to heart disease, and a strong proponent of diet and exercise. He further believed that doctors needed to lead by example. Addressing an American Heart Association meeting in 1999, he said: "There are those among us who do not follow proper diets, who do not engage in physical activity, and if we do not do it ourselves, how then can we translate effective techniques to our patients?"[14] Dr. Smaha died recently, felled by a heart attack after his daily run. He was 63.

Dr Mike Eades, author of *Protein Power*, a popular low-carbohydrate diet book, offered this comment: "Dr Smaha, I'm sure, followed his own advice, kept his weight down, ate a low-fat diet, did aerobic exercise, and made sure his cholesterol remained in check — in short he did everything he recommended to others to avoid heart disease, yet he succumbed to a heart attack. If cholesterol were the cause of heart disease, if a low-fat diet was truly 'heart healthy', if aerobic exercise kept coronary arteries supple and plaque-free, then Dr Smaha would surely still be with us today. But he isn't. And I don't think that all the beliefs he had on the proper prevention of heart disease were valid."[15]

Dr Sylvan Lee Weinberg, another prominent cardiologist, past president of the American College of Cardiology, past president of the American College of Chest Physicians, editor of the *American Heart Hospital Journal* and director of medical education at the Dayton Heart Hospital in Dayton, Ohio, disagrees with his two deceased colleagues when it comes to diet. He is highly critical of the present dietary prescription. Weinberg wrote in the *Journal of the American College of Cardiology* in 2004: "A balanced appraisal of the diet-heart hypothesis must recognise the unintended and unanticipated role that the LF-HCarb [low-fat high-carbohydrate] diet may well have played in the current epidemic of obesity, abnormal lipid patterns, type II diabetes, and the metabolic syndrome.

Defense of the LF-HCarb diet, because it conforms to current traditional dietary recommendations, by appealing to the authority of its prestigious medical and institutional sponsors or by ignoring an increasingly critical medical literature, is no longer tenable. The categoric rejection of experience and an increasingly favourable medical literature, though still not conclusive, which suggests that the much-maligned LCarb-HP [low-carbohydrate high-protein] diet may have a favourable impact on obesity, lipid patterns, type II diabetes, and the metabolic syndrome, is also no longer tenable."[16] Dr. Weinberg is still with us.

Dr Robert C. Atkins was also a cardiologist. Early in his career he studied weight-loss approaches before choosing a low-carbohydrate diet to lose extra pounds he had gained during training. Intrigued by the effectiveness of this intervention, he convinced the executives of AT&T, a large American corporation, to use a low-carbohydrate diet to improve the health of senior management. Their success inspired him to expand his practice to include a low-carbohydrate diet regimen.[17] In 1972 he published his first book, *Dr Atkins Diet Revolution*, which became an enduring best-seller. In 1973, the *Journal of the American Medical Association* published a commentary that was highly critical of low-carbohydrate diets and of Dr Atkins' diet in particular.[18] In the same year, the US Senate struck the Senate Select Committee on Nutrition and Human Needs and Atkins was summoned to testify. By that time he had already successfully treated more than 10,000 patients using his low-carbohydrate approach. In his testimony, he noted the irony of the attack by the American Medical Association as it was from articles in its journal that he learned of the potential benefits of low-carbohydrate diets. He urged the committee to mandate that research be done on his diet, suggesting that it would be simple to test it against other dietary approaches. He expressed confidence in what would be the results. Unfortunately, the recommendations of the committee did not include any such mandate and, furthermore, sanctioned the one-size-fits-all approach that has been the basis of nutritional policy ever since.[19]

The vilification of Dr Atkins and his diet by the scientific establishment, exemplified by the *Journal of the American Medical Association* article, persisted to the extent that nobody seriously studied his dietary approach until almost 30 years had elapsed.

Dr Atkins slipped on ice while walking to his clinic in Manhattan one morning. He sustained a serious head injury, fell into a coma and died several days later at the age of 72. The campaign to discredit him became more venal immediately following his death when his autopsy results were obtained fraudulently and false information was circulated suggesting that he was obese and had coronary artery disease.

Fortunately, by this time a number of studies were examining his diet and several had already been published, all of which began to refute the dire warnings of harm associated with his diet, the roots of which can be traced back to the 1973 *Journal of the American Medical Association* article.

In 2000, Dr Eric Westman, an internist at Duke University, observed that some overweight patients achieved sustained weight loss using the Atkins diet. Like other clinicians, Westman was frustrated with the poor results delivered by the standard approach. Intrigued by the success of these patients, Westman decided to investigate. He put a cohort of obese subjects on the Atkins diet for six months. Remarkably, 80% of them were compliant, losing an average of 10% of their body weight without serious side-effects. In addition, they significantly improved their lipids, a finding contrary to the prevailing belief that lipid profiles would worsen.[20]

Other studies followed, the results of which remained consistent with Westman's initial findings. Notably, Dr William Yancy, working with Westman, conducted a randomised trial comparing Atkins dieters to controls who ate the American Heart Association (AHA) 'prudent' diet. The Atkins group had better compliance, lost more weight and demonstrated greater improvements in their lipids. This was remarkable considering that the AHA diet limited fat intake and restricted calories whereas the Atkins diet did neither.[21]

Dr Frederick Samaha, at the University of Pennsylvania, conducted a similar randomised trial where the Atkins diet outperformed the control diet. In this case, however, the data also demonstrated the improvement in lipids and insulin sensitivity among the Atkins dieters was greater than what could be attributed to weight loss alone.[22]

Although a spate of studies followed, demonstrating consistently superior results for the Atkins diet, critics cautioned that this approach could not be endorsed without longer-termed studies. Then, two 12-month studies were published. In both, the Atkins dieters achieved greater weight loss compared with AHA dieters at the six-month point but the difference lost statistical significance by one year (lipid improvements remained statistically significant). These results gave comfort to the detractors of low-carbohydrate diets who, ignoring the lipid improvements, could argue that the Atkins diet was no better than the AHA diet. A closer look at the data suggests otherwise. In one study, which randomised 63 obese subjects, the Atkins dieters lost 76% more weight than the controls,[23] while in the other, which followed 132 obese subjects, they lost 65% more.[24] It is possible that the loss of statistical power was more attributable to the small numbers, which were further diminished by attrition, than to the relative performance of the diets themselves.

People of the Nass

My work takes me into the remote parts of Canada's north west, where First Nations people still harvest and eat their traditional foods. On a recent trip to the Nass River Valley, home of the Nisga'a people, I attended the annual harvest of the oolichan, a smelt-like fish that spawns in the river in great numbers. The fish are netted and placed in log bins where they ferment for one to two weeks and then are simmered in large vats for several hours. When this concoction is cooled, fish oil rises to the surface, where it is collected, strained and stored for use as a staple food throughout the year. Other fat-rich traditional foods from the river include sea lion and five species of salmon. Protein and fat are also obtained from bear, mountain goat and moose. Berries and seasonal wild plants round out the diet. Missing is any significant source of sugar or starch. The traditional diet of the Nisga'a, like that of most Canadian indigenous peoples, was low in carbohydrates whereas their modern diet includes large amounts of introduced carbohydrate foods. Diabetes was unknown to the Nisga'a people in olden times. There is no word for diabetes in their language. Today the Nisga'a have high rates of obesity, MetS and type 2 diabetes, like indigenous people around the globe who eat a modern diet.[25] In one Nisga'a village, the local chief told me that of 77 elders, 43 have type 2 diabetes (personal communication, Chief Willard Martin). Across Canada, Aboriginal rates of type 2 diabetes are up to five times the general population rate.[26,27] In one study, MetS among Aboriginal people was 42% compared with a 25% rate in the general population.[28] Overweight and obesity rates are also significantly higher.[29]

Five important studies

In 1984, Dr Kerin O'Dea studied Australian Aboriginal men with diabetes who returned to a traditional way of life. Over a seven-week period their diabetes improved greatly.[30] Obviously, diet was not the only factor as there was also an increase in exercise. How important was each factor? Insight is gained by examining a similar study among Canadian Cree, where a diabetic cohort lived in the bush for three months while a control cohort stayed in town. Unlike their Aboriginal counterparts, the bush-living Cree did not achieve significant improvements. The authors attribute this disappointing result to the fact that the bush-living cohort continued to eat a diet of store-bought food.[31] When these studies are compared, it appears that diet was the major factor contributing to the improved outcomes of the Aboriginal men.

In 1999, Dr David Ludwig at Children's Hospital Boston examined the relationship between appetite and the glycemic value of food in obese teenage boys. Using a crossover design, the boys ate eucaloric meals of varying glycemic value. They had the same meal for breakfast and lunch followed by five hours of observation. After a high-glycemic meal the boys became hungry sooner, at 2.5 hours versus four hours for a low-glycemic meal. In the five hours following the high-glycemic meal they ate 81% more calories. In addition, following the high-glycemic meal, serum epinephrine levels began to rise at 2.5 hours, reaching 100% of baseline at the five-hour point. The researchers concluded that high-glycemic foods led to excessive calorie intake. It is interesting to consider what constituted the respective meals. The low-glycemic meal was a vegetable omelet and fruit. The high-glycemic meal was instant oatmeal, milk and sugar.[32] The food guides, in promoting a high-carbohydrate diet, suggest we eat whole grains.[2] Among these boys, the meal corresponding to that recommendation led to overeating.

In another examination of the relationship between diet and appetite, Dr Guenther Boden, at Temple University in Pennsylvania, observed overweight type 2 diabetic women. To establish their baseline diet the participants ate ad lib for seven days. Over the following 14 days, they continued to eat ad lib except they had no access to foods containing starch or sugar. On this very low-carbohydrate diet, the women lost weight and their diabetes improved significantly, and, although they had no calorie restriction, they spontaneously dropped their caloric intake by a third. Notably, the lost calories attributable to the missing carbohydrates were not replaced by any significant increase in fat or protein. While fat and protein were now proportionately larger because of the absence of carbohydrates, they did not increase significantly in absolute value.[33] This may be important for two reasons. People with MetS and diabetes may have kidney damage, in which case an increase in protein would need to be undertaken carefully. This is cited as a reason to avoid low-carbohydrate diets. Boden's study demonstrates that a low-carbohydrate diet need not result in a significant increase in protein. Second, these results speak to the affordability of low-carbohydrate diets, something of importance in the lower socio-economic strata where the problems of obesity, MetS and type 2 diabetes are worse. The women in this study would arguably have saved money on their low-carbohydrate diet.

Another criticism of low-carbohydrate diets suggests the improvements in diabetes are attributable to weight loss, not the method by which the reduction was achieved. Doctors Mary Gannon and Frank Nuttall, from the Veterans Administration in Minneapolis, have shown that reducing carbohydrates can deliver significant improvements even with no loss of weight. Their subjects ate a diet where the carbohydrate content was reduced to 20% of calories. After five weeks, significant improvements in fasting glucose, HgA1c and insulin sensitivity were observed in the absence of weight loss.[34] The salutary results were due to the reduction in carbohydrates.

Taken together, these studies suggest that significant improvements in MetS and type 2 diabetes can be achieved simply by reducing the carbohydrate content of the diet without the need for exercise or even weight loss, and that consumption of carbohydrates, especially those of high glycemic value, drives appetite and excessive intake.

Jimmy's Story

What happens when a First Nation person with diabetes and MetS returns to a traditional diet? Jimmy Wilson is a 48-year-old Kwakiutl man from a coastal area near the Nass River Valley. A type 2 diabetic, he had been on insulin for 17 years. He had high cholesterol and was medicated for hypertension. His fasting glucose was significantly above normal despite frequent testing and four insulin injections per day. His blood pressure also remained high despite his use of an ACE inhibitor. Jimmy started a low-carbohydrate diet and within two weeks had lost 17 lbs, normalised his fasting glucose and discontinued his insulin. After a month, with a weight loss of 31 lbs, he had normalised his blood pressure and discarded his antihypertensive. Two years later his weight loss has leveled off at 50 lbs; he is still overweight, but has maintained normal fasting glucose, HgA1c, blood pressure and cholesterol without medications. Exercise was not a part of his regimen, although he reports being more active now as he feels more fit and energetic. For 17 years, he failed to achieve blood sugar control on a prescription of exercise, high-carbohydrate diet and multiple medications. Going against the advice of his care-givers and simply returning to his traditional way of eating, he has eradicated the signs of diabetes and MetS without medication (personal communication, Jimmy Wilson).

Conclusion

The shedding of an entrenched system of beliefs, even when the evidence is compelling, is not easily done. Although cracks are appearing in the wall of nutritional science dogma, there are reactionary forces working to maintain the status quo. Consider the economic interests threatened by a big dietary shift or a population-wide drop in caloric intake. Large corporations profit from an array of expensive drugs, the need for which might diminish greatly. The power these interests wield can influence the media, distort government policy-making and affect the behaviour of educational and research institutions. Although we are familiar with the disinformation campaigns and other odious tactics used by the tobacco industry when its business was threatened, we find it hard to fathom corporations that produce food and medicine might use similar tactics when they, too, are threatened by change. As more low-carbohydrate research is published, the weight of scientific evidence will eventually topple the monolithic belief system in favour of a more flexible range of dietary options. In the meantime, those of us who toil on the front lines can do our part by asking, "How long are we going to continue delivering a vaccine that clearly doesn't work?"

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