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Response to Draft Dietary Guidelines Submitted to the Ministry of Health April 2014

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Preamble

Below is our response to the request for "limited stakeholder feedback" from the Ministry of Health for the guideline statements on healthy eating and physical activity. Whilst we were not considered initially as a group that should be considered for feedback, Ministry of Health officials later granted our request.

We understand that the Ministry of Health intends to publish a concise document based on revised key healthy eating and physical activity messages (guidelines statements) for adult New Zealanders. These statements will also be included in the Ministry's health education resources written for the public.

These statements are important because any increase in population levels of physical activity and healthy eating are likely to result in the prevention, and improve the treatment, of chronic diseases. These benefits will be realised at the population level, reducing the burden on the public health dollar, but more importantly confer a higher quality of life for New Zealanders. It is important that the scientific evidence is considered carefully before giving such advice and it is our view that advice over the last several decades in this arena has not had the confidence of such evidence. It is our contention that this has (partially) contributed harm to public health, especially the most vulnerable in the population. We believe this to be relevant particularly to the "demonisation" of dietary fat, the now refuted lipid hypothesis, and an emphasis on consuming large amounts of dietary carbohydrate.

As such, we have written a response to the new draft guidelines. These guidelines are more or less the same (with minor alterations) in principle to those of released during the last 40 years in New Zealand.

Our group has had a high media profile in our critique of these guidelines in recent times. We have been criticised for "cherry picking" evidence to suit our hypothesis, as well as ignoring the "totality" of evidence (see Appendix C). As such, we have tried to limit much of our scientific analysis given in this response to the same studies used to support the draft guidelines for healthy eating which promote a lower fat, higher carbohydrate diet based on the principle of energy balance. Notwithstanding, we do include additional studies but it is important to note that these studies themselves do not support the case for the current or new draft guidelines.

We are open to debate around any, or all, of the scientific points we make in this document. It is also our intention to eventually publish the review and response below for public scrutiny.

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Simplified (lay) Summary of response to draft dietary guidelines 2014

Advice on diet, as well as regulation of the food supply, has long been a function of good Government; for example, the Ministry of Food campaigns during the Second World War were designed to ensure that all citizens would be adequately fed despite wartime restrictions on the availability of many of the foods commonly eaten in peacetime. The addition of iodine to salt, the food handling regulations, the provision of nutritional information on packaged foods, are well-known examples of sound and effective Governmental foresight.

Dietary guidelines were originally intended to prevent the various deficiency diseases, to protect the population from food- and water-borne illnesses, to warn against the adulteration of the food supply with non-food items, and to discourage both food waste and the excessive use of specific items linked to disease (e.g. alcohol, sugar).

In the late 20th century some scientists and politicians came to believe that dietary guidelines could be used to alter the eating habits of the population in ways that would protect them from the effects of specific diseases. The food industry came to welcome these changes as they gave wide scope for the exploitation of cheap, often subsidised crops (sugar, wheat, corn and soy) in the manufacture of processed foods which could then be marketed as "healthy" for one reason or another. The result has been that the good intentions of those politicians have been undermined, and the mistakes in the reasoning of those scientists, working as they were from the less complete biological knowledge of an earlier era, have been magnified, while some of the original purposes of dietary guidelines have tended to be neglected by authorities or subverted by the food industry, which has itself become a surrogate authority, from which we now receive many of our popular ideas about healthy eating.

The result has been an epidemic of type 2 diabetes, obesity, and fatty liver disease, diseases clearly related to diet, and conditions which predispose people to further illnesses. While life expectancy has increased and the incidence of some diseases has dropped over the period in question, it is arguable that this has been due more to the significant decline in the rate of smoking, as well as (amongst other factors) the Clean Air act, controls on vehicle emissions, the reduction of lead in the environment, significant advances in medical treatment, OSH workplace safety regulations, and very large changes to New Zealand's ethnic (and hence genetic) makeup during the time in question, including many first-generation migrants who tend to retain their traditional eating patterns in a new country.

We have listed each of the proposed dietary guidelines below, along with our summary and comments. A more detailed scientific review follows this summary.

The new dietary guidelines (below) are essentially the same as the old dietary guidelines. Our detailed submission to the Ministry is intended to critique these guidelines in the light of the evolving scientific knowledge about the links between food and disease. A simplified summary of our discussion follows, followed by our own proposal for dietary guidelines based on real food and real meals.

Summary of our response to each proposed guideline

1. To be a healthy weight, balance your intake of food and drinks with your activity levels.

We think that this statement a truism, misleads, as it ignores the complex hormonal interaction between body, food and environment. The simplicity of this statement is being used by the food industry to justify a "balanced diet, as long as you are physically active" message. When such a statement is interpreted in food advertising and corporate marketing speak, this may mislead the public justifying the consumption of highly processed food.

Such a simplistic statement ignores the pivotal role dietary carbohydrate plays in regulating insulin and therefore fat burning and fat storage mechanisms in humans. This circular truism of the calories in and calories out explanation provides no information about the most effective ways to manage metabolic health.

Both people eating low fat (the guidelines diet) and low carbohydrate diets tend to reduce weight, when compared to people eating the standard Western diet.
However, people on low carbohydrate diets tend to have better weight control and better blood test results than people on low fat diets, even when the low fat dieters are told to eat fewer calories, and the low carbohydrate dieters are not. The low carbohydrate dieters are more likely to automatically limit their food intake to suit the amount of work they need to do.

That the recommendation to eat low fat food and the food industry's use of slogans such as "99% fat free" has coincided with our obesity epidemic seems consistent with the science we have reviewed, and we propose that guidelines which allow people to freely choose the balance of fat and carbohydrate that suits, mainly from whole foods eaten in traditional meals, will have the best impact on energy balance.

2. Enjoy a variety of nutritious foods every day including:
a) plenty of different coloured vegetables and fruit

We agree with this statement.

2. Enjoy a variety of nutritious foods every day including:
b) a range of grains and cereals that are naturally high in fibre

We disagree that grains are necessary foods for health; there have been, and still are, many societies that maintain good health with no grains in the diet. While high consumption of refined grains (flour) may be a disease risk factor, in ways whole grains are not, there is no evidence that grain fibre specifically protects against disease, and some risk that bran decreases the absorption of minerals, such as iron. Following the dietary guidelines would see the majority of calories coming from grains, yet wholegrains are a poorly defined food; wholemeal pastas and some wholegrain breads, for example, may be more accurately described as energy dense processed foods.
Though we have no wish to discourage the consumption of fibrous vegetables, which are desirable foods for other reasons, we found only one study in which these were associated with a significantly decreased risk of a disease (colon cancer), and this effect was only

seen when dairy fat was included in the diet, increasing proportionally to the intake of full-fat dairy foods.

2. Enjoy a variety of nutritious foods every day including:
c) some low fat milk products and/or calcium-fortified milk alternative

The dietary guidelines are opposed to the consumption of dairy fat, yet the available scientific evidence shows that full-fat dairy products, and not low-fat milk, are protective against diabetes, cancer and cardiovascular disease, and that dairy fat does not contribute to obesity.

Dairy fat is a source of vitamins not easily found in other foods and, like all fats, increases the absorption of the vitamins and antioxidants found in green vegetables and in fruits like tomatoes and capsicums. We believe that the guidelines oppose dairy fat consumption, despite the evidence for health benefits, purely due to the outdated theory that saturated fat is harmful, a theory that we have not found to be supported by the research.

2. Enjoy a variety of nutritious foods every day including:
d) some legumes, nuts, seeds, fish, eggs, lean poultry or lean red meat.*
**Legumes include cooked dried beans (eg baked beans), split peas, lentils and chickpeas.*

We support most of these recommendations. However we feel that the evidence supports avoiding industrially processed meat products, and does not support avoiding fatty cuts of meat in favour of lean muscle. We also wish to point out that traditional Chinese cooking as practiced in New Zealand does not focus on muscle meat but makes good use of all parts of an animal, as our forefathers used to do. A diet that includes muscle meat only is wasteful and may be nutritionally inferior to a diet that also includes organs and other edible animal parts. We believe that cheese should be a food listed in this section.

3. Choose and prepare foods and drinks:
a) With minimal fat, especially saturated fat; if you choose to add fat use plant-based oils and spreads

We do not find that the evidence supports the specific avoidance of saturated fat. Differences in saturated fat intake seem to have no impact on the risk of cardiovascular disease. There may be a protective effect of the various polyunsaturated fats in the proper quantities, which will be supplied by including foods such as fish, nuts, seeds, meat, avocado and olive oil in the diet. Given the probable health benefits of dairy fat there is no reason to use artificial spreads or non-traditional refined oils. Rather than avoiding animal fats, it makes more sense to avoid the fats and oils used in processed and deep fried foods, which may contain potentially harmful contaminants such as *trans* fats and lipid peroxides.

The effect of various fats, starches, sugars and proteins on serum cholesterol, and the subsequent effect of serum cholesterol on any disease process is a complicated and largely undecided matter, and the idea that this question can be simplified by isolating the effect of saturated fat is no longer tenable, given that there seems to be no direct link between saturated fat intake and heart disease. Pacific Islanders still eating traditional

diets who get the majority of their daily calories from coconut saturated fats have higher cholesterol, yet the same low rate of cardiovascular disease, compared to neighbouring islanders eating traditional diets with less saturated fat.

3. Choose and prepare foods and drinks:
b) low in salt (sodium); if using salt, choose iodised salt

Restricting salt without making other diet and lifestyle changes only lowers blood pressure slightly, and very low sodium intakes may increase the risk of health problems. The average intake of sodium in New Zealand has been estimated at 3900mg per day (around 1&1/2 teaspoons of salt), a level well within the range indicated as having no effect on death or disease risk (between 2,645 and 4,945 mg) and so the recommendation to reduce sodium intake is in our opinion unnecessary. New Zealanders are at risk of iodine deficiency and we support the use of iodised table salt.

3. Choose and prepare foods and drinks:
c) with little or no added sugar

Sugar as a factor in disease has been neglected until relatively recently. Recent studies show that a high consumption of added sugars is strongly associated with heart disease, type 2 diabetes, tooth decay and obesity. The World Health Organisation recently revised its maximum recommended sugar intake downwards, to 30g per day. There is more than this amount in a single serving of many sweet drinks and confectionaries, and high levels in many processed foods. We support this recommendation and feel that advice to limit intake of processed foods is needed to lower sugar intake. Highly refined (high-GI) starches may be similar to sugars in their impact on health.

3. Choose and prepare foods and drinks:
d) Make water your first choice for drinks

We agree with this recommendation.

3. Choose and prepare foods and drinks:
e) Buy, prepare, cook and store food to ensure food safety

We agree with this recommendation.

4. If you drink alcohol, keep your intake low. Don't drink if you are pregnant or planning to become pregnant.

We agree with this recommendation.

Summary and our proposed guidelines

Overall, we submit that the proposed dietary guidelines, which currently detail a diet eaten mainly by European, university educated professionals and the worried well, should instead be flexible enough to reflect the variety of healthy and nutritious dietary patterns possible in a multicultural society.

As well, we have developed a set of dietary guidelines which we believe are consistent with the evidence and practiced by the population would result in net public health benefit. We call these the "real" food guidelines based on the emphasis on whole unprocessed food, a set of terms absent from the current draft guidelines. Our guidelines draw some inspiration from the recent draft Brazilian dietary guidelines, which can be read in summary at http://civileats.com/2014/03/12/brazils-new-dietary-guidelines-cook-and-eat-whole-foods-be-wary-of-ads/

The real food guidelines
Real food for real people, based on real evidence

1) Enjoy nutritious foods everyday including plenty of fresh vegetables and fruit.

2) Buy and prepare food from whole unprocessed sources of dairy, nuts, seeds, eggs, meat, fish and poultry.

3) Keep sugar, added sugars, and processed foods to a minimum in all foods and drinks.

4) If you drink alcohol, keep your intake low. Don't drink if you are pregnant or planning to become pregnant.

5) Prepare, cook, and eat minimally processed traditional foods with family, friends, and your community.

6) Discretionary calories (energy foods) should:
a) Favour minimally refined grains and legumes, properly prepared, over refined or processed versions, and boiled or baked potatoes, kumara or taro over deep fried or processed potato fries and chips.
b) Favour traditional oils, fats and spreads over refined and processed versions.

Key points in relation to the draft eating guideline statements

The draft statements are:

1) To be a healthy weight, balance your intake of food and drinks with your activity levels.

2) Enjoy a variety of nutritious foods every day including:
   a) plenty of different coloured vegetables and fruit
   b) a range of grains and cereals that are naturally high in fibre
   c) some low fat milk products and/or calcium-fortified milk alternatives
   d) some legumes*, nuts, seeds, fish, eggs, lean poultry or lean red meat.
   *Legumes include cooked dried beans (e.g. baked beans), split peas, lentils and chickpeas.

3) Choose and prepare foods and drinks:
   a) with minimal fat, especially saturated fat; if you choose to add fat use plant-based oils and spreads
   b) low in salt (sodium); if using salt, choose iodised salt
   c) with little or no added sugar.

4) Make water your first choice for drinks.

5) Buy, prepare, cook and store food to ensure food safety.

6) If you drink alcohol, keep your intake low. Don't drink if you are pregnant or planning to become pregnant.

Overall response to the draft guideline statements

Regarding the proposed statements, we agree that whole unprocessed food, including vegetables and fruits, is part of a healthy and nutrient dense diet. We would like to see more emphasis on unprocessed "whole" foods in both purchasing and preparation. This is conspicuously absent. We agree with the statements regarding limits on sugar, the first choice of water as a drink, and limiting alcohol consumption.

We object to the majority of content in Guidelines 1, 2 and 3 above as the statement is either scientifically misleading or not consistent with the current body of scientific knowledge. Further, we want to provide scientific feedback on certain lines of evidence which we think do not provide evidence for improved public health, and at worst, may cause harm.

As well, we have developed a set of dietary guidelines (Appendix A) which we believe are consistent with the evidence and, if practiced by the population, would result in net public health benefit. We call these the "real" food guidelines based on an emphasis on whole unprocessed food, a set of terms absent from the current draft.

These arguments are accompanied by a short supplementary comment (Appendix B): "The lipid hypothesis; an obsolescent explanation for a dubious association."

Specific responses to each of the draft guideline statements:

1. To be a healthy weight, balance your intake of food and drinks with your activity levels.

One reason for endorsing a low-fat high-carbohydrate diet is based on the greater energy density of fats compared to carbohydrates. The Atwater factor for carbohydrate is 4 calories per gram compared to 9 calories per gram for fats. Thus avoiding fat equates to fewer calories per mouthful and therefore a greater likelihood of weight gain. Willet and Liebel (2002) concluded that "*within the United States, a substantial decline in the percentage of energy from fat during the last two decades has corresponded with a massive increase in the prevalence of obesity. Diets high in fat do not appear to be the primary cause of the high prevalence of excess body fat in our society, and reductions in fat will not be a solution* (p.1)."

If one objective of the Dietary Guidelines is to improve weight control or metabolic health (including a reduction in CVD risk factors), the comparisons of low-fat and high-fat diets that measure these outcomes are valuable.

It is a common finding that low-carbohydrate diets with unlimited calories are as effective for weight control as calorie-restricted low-fat diets (Nordmann et al., 2006). This is perhaps due to a spontaneous reduction in energy intake due to the greater satiating effect of fats and protein, compared to carbohydrate. This work is further supported by findings of Shai and colleagues (2008) who investigated weight loss and cardiometabolic markers in people on either an energy restricted low-fat regime, an energy restricted Mediterranean regime, or an unlimited energy low-carbohydrate regime. After 24 months, the low-carbohydrate regime had produced the greatest overall weight loss compared to the other two regimes, with more favourable changes to cardiometabolic markers including HDL cholesterol, triglycerides, and ratio of total cholesterol to HDL cholesterol.

Given that the Dietary Guidelines do not establish energy limits, it might be thought that recommendations around food choices that have a greater potential to promote the spontaneous regulation of energy balance (i.e. a low-carbohydrate, high-fat diet) should have been made if the goal is to reduce the incidence of obesity and diabetes. Guidelines that instead focus on the greater nutritional value of a diet rich in whole, unprocessed foods, minimally refined ingredients, and on the greater social and health benefits of traditional meals compared to pre-packaged and convenience food could naturally result in this outcome. This would be along similar lines to the recently proposed Brazilian Dietary Guidelines. For an English summary of these guidelines, please refer to http://civileats.com/2014/03/12/brazils-new-dietary-guidelines-cook-and-eat-whole-foods-be-wary-of-ads/.

2a) Enjoy a variety of nutritious foods every day including plenty of different coloured vegetables and fruit

We agree with this statement.

2b) Enjoy a variety of nutritious foods every day including a range of grains and cereals that are naturally high in fibre

There is contradictory evidence for the health benefits of grain fibre including concerns that cereal fibre in particular contributes to malnutrition. For decades, fibre has been promoted

as an essential component of a healthy diet. The supposed benefits of a fibre-rich diet have been mandated via several agencies including the government, health professionals and the food industry; however, much of this supposed benefit is not supported by evidence. The "health halo" that surrounds plant fibre is likely because fibrous growing plants may be rich in both soluble and insoluble fibre, resistant starch, antioxidants, vitamins, water, electrolytes, omega 3 and 6 essential fatty acids, and phytochemicals. Because of the belief that fruit and vegetable consumption protects against colorectal cancers by virtue of its fibre content, there has been a series of lab-based trials of grain-based fibre and psyllium fibre in the secondary prevention of colorectal cancers. The protective effects of fibre in these trials, that is, where fibre is added to an otherwise adequate diet has failed to be demonstrated. The majority of studies supporting the benefits of dietary fibre on health outcomes such as obesity, heart disease and cancer, and that have informed our current guidelines, have been epidemiological by design, yielding (confounded) weak associations as evidence, rather than isolating fibre as being a causative factor (Bingham et al., 2003; Key, Allen, Spencer, & Travis, 2002)

When looking at the proposed benefits of fibre in the risk against colorectal cancer, several studies indicate that it simply does not have the beneficial effects that current guidelines suggest. One of these studies is a 16-year prospective cohort study conducted by Fuchs et al., (1999) who reported no evidence that dietary fibre on the whole reduces the risk of colorectal cancer. In this study fibre derived from fruit was associated with an appreciable reduction in risk (multivariate relative risk, 0.86; 95% confidence interval, 0.67-1.10), but the overall trend was not statistically significant (*P*=0.16). In contrast, a greater consumption of vegetable fibre was associated with a significant increase in the risk of colorectal cancer (multivariate relative risk, 1.35; 95 % confidence interval, 1.05-1.72; *P*=0.004). Furthermore, Park et al., (2005) conducted a pooled analysis of 13 prospective cohort studies which included 725 628 individuals (both male and female), and reported no association between dietary fibre and colorectal cancer once other dietary risk factors were accounted for.

In the argument for reduced recurrence of colorectal adenoma, several intervention studies have indicated that fibre has little to no protective effect. Jacobs et al., (2002) reported that neither fibre intake from a wheat bran supplement, nor total fibre intake, reduced the reoccurrence of colorectal adenoma in a clinical trial where participants were randomised to a cereal fibre supplement of either 13.5 g or 2.0 g/day. On the contrary, Bonithon-Kopp et al. (2000) found that psyllium supplementation increased recurrence of colorectal adenoma (odds ratio: 1.67 (95% confidence interval: 1.01-2.76, *P*=0.042)). In a Cochrane meta-analysis which included five randomised controlled trials (RCTs) using psyllium, bran, and a combination of mixed grain and vegetable-based fibre, Asano and McCleod (2002) found no evidence to indicate that increased dietary fibre intake will reduce the incidence or recurrence of adenomatous polyps within a two-to-four year period.

The cardiovascular outcomes of the DART trial are also supportive of the lack of benefit from fibre. This trial investigated the effects of advice to increase fibre intake from cereals in the secondary prevention of myocardial infarction. Burr et al. (1989) reported that subjects given advice on increasing dietary fibre had a slightly higher mortality than those who weren't; however, this was not a significant finding.

An argument can be made for grain-based fibre being detrimental for health from a

micronutrient bioavailability standpoint. Grain-based husks contain the anti-nutrient phytic acid, which has an effect on reducing the mineral availability of zinc, magnesium, iron and calcium in the body (Torre, Rodriguez, & Saura-Calixto, 1991). The issue of anti-nutrients is a well-established cause of malnutrition in the developing world where diets are high in unrefined cereals and low in animal products. In Western populations, the same effect is seen. Bach Kristensen et al., (2005) reported that the consumption of the recommended daily intake of fibre from fibre-rich wheat bread after four months resulted in an impairment of iron status in women with initially sufficient iron stores. Furthermore, reduction of the phytic acid concentration in the bread was not sufficient to maintain iron status in this group. Similarly, Knudsen et al., (1996) found (in a small, short-term experiment) that intestinal and urinary losses of the minerals zinc, copper and magnesium exceeded their fractional absorption in participants consuming a fibre-rich diet, thereby resulting in negative balances of these nutrients.

As we acknowledge its important role in providing fuel for various gut microbes and by extension short chain fatty acids for the host

It is not our intention to *discourage* the consumption of dietary fibre, particularly natural sources of soluble fibre, as we acknowledge its important role in providing fuel for various gut microbes and by extension, short chain fatty acids for the host, amongst other possible (as yet unsubstantiated) benefits (Hugenholtz, Mullaney, Kleerebezem, Smidt, & Rosendale, 2013). It is rather that we highlight firstly that the evidence for its protection against disease is not as convincing as the current guidelines portray. Secondly, that both soluble and insoluble fibre can be sourced from fruit and vegetables alone without the need to include a recommendation for the excessive consumption of wholegrain products. There are several other critical reasons addressed in this document as to why the consumption of excessive amounts of processed carbohydrate-based foods (which include wholegrain products) should be avoided. This, in conjunction with the fact that these foods provide anti-nutrients and poor overall nutrient-density compared with natural soluble fibre containing foods (fruit and vegetables) suggests that their continued specific endorsement at the quantities that currently exist may be more harmful than beneficial to public health.

Lastly, there are traditional cultures that enjoyed good health without eating any grains whatsoever; Maori, Pasifika, and all hunter-gatherer societies studied by anthropologists (Prior, Davidson, Salmond, & Czochanska, 1981). In recent times, members of these groups have enjoyed improved health when grains are removed from the diet and replaced by traditional foods. Traditional Chinese cooking, which makes good use of the whole animal, rather than “lean meat and poultry”, does not seem compatible with the proposed guidelines.

2c) Enjoy a variety of nutritious foods every day including some low-fat milk products and/or calcium-fortified milk alternatives

Whilst we agree that dairy foods are nutritious, we feel this guideline is in direct conflict with the scientific evidence that consumption of full-fat dairy products has been uniquely identified as a beneficial modifier of disease risk. Conjugated linoleic acid (CLA) and trans-palmitoleic acid, specifically present in dairy fat, have also been found to be inversely associated with diabetes incidence (Castro-Webb, Ruiz-Narváez, & Campos, 2012; Mozaffarian et al., 2013). Holmberg and colleagues (2009) found a strong protective effect against coronary heart disease when high fat dairy was consumed in combination with a daily intake of fruit and vegetables. This was not seen in those choosing to consume low-

fat dairy products. A protective effect was also found by Larsson and colleagues (2006) who found that women consuming more than four serves of high-fat dairy foods per day had a 41% reduced likelihood of colorectal cancer compared to those consuming less than one serve daily. Contrary to their hypothesis, Berkey and colleagues (2005) discovered that lower fat varieties of milk products (and not dairy fat) were associated with weight gain in their investigation of dairy consumption in close to 13,000 children. Finally a recent review of the literature by Kratz and colleagues (2013) concluded the recommendations for consuming low-fat dairy foods was in contrast to the observational evidence of a reduced cardiometabolic risk within typical dietary patterns.

On this basis, therefore, we submit that there is no scientific reason to believe that dairy fat of the high quality available in New Zealand presents such a risk to health that it should be removed from the diet or replaced with processed fats, as the Dietary Guidelines recommend. We also draw the Committee's attention to the reported associations between health benefits and dairy fat related to the most saturated of all animal fats in the diet.

2d) Enjoy a variety of nutritious foods every day including some legumes, nuts, seeds, fish, eggs, lean poultry or lean red meat*

We agree with the majority of this statement, although, as detailed below, we submit that there is no evidence (or negative evidence) for efficacy and long-term safety of low-fat diets.

3a) Choose and prepare foods and drinks with minimal fat, especially saturated fat; if you choose to add fat use plant based oils and spreads.

We draw attention to the paucity of statistical evidence linking either reduced fat or modified fat (including saturated fat) with disease end-points. For example, a Cochrane review (Hooper et al., 2011) of randomised studies of the effect of modified or reduced fat interventions on total and cardiovascular disease (CVD) mortality showed no overall effect of the diets on either outcome (total mortality: relative risk 0.98, 95% CI: 0.93-1.04; and for CVD mortality: relative risk: 0.94, 95% CI: 0.85-1.04). A small relative reduction in cardiovascular events was noted (pooled RR: 0.86, 95% CI: 0.77-0.96). These effects are relatively modest, and for the most objective outcomes (overall and CVD mortality), no association was reported. The inconsistency of the effect for the three similar outcomes is also noted. We conclude that this study does not provide strong evidence for population guidelines to reduce either saturated fat intake or total fat to reduce the incidence of CVD end-points. Other meta-analyses, similarly, find little statistical evidence linking modified saturated fat intake with CVD mortality (Mente, de Koning, Shannon, & Anand, 2009; Siri-Tarino, Sun, Hu, & Krauss, 2010). We believe a causal effect of this dietary intervention is unlikely if there is little statistical evidence of association between the exposure and disease.

Those who support the need to reduce saturated fat often discount negative summary studies and focus instead on analyses that report positive associations observed between fat-modifying diets and CHD end points. One study (Jakobsen et al., 2009), for example, which pooled a limited selection of cohort data for which individual level data was available, showed a positive association between replacing saturated fat with polyunsaturated fat in reducing total coronary disease events (pooled hazard ratio 0.69; 95% CI: 0.59, 0.81) and coronary disease mortality (pooled hazard ratio 0.57; 95% CI: 0.42 to 0.77). The findings were, however, inconsistent in that replacing monounsaturated fat with saturated, resulted in no association with coronary death, with a similar null-result

reported for replacing carbohydrate with saturated fat. If we limit our discussion to this single study, the findings raise the question of whether saturated fat is truly the causal exposure because, if saturated fat is the principal cause of cardiovascular disease, the nature of the replacing nutrient (carbohydrate, monounsaturated or polyunsaturated fat) should have little effect on disease risk. The results, to us, may be interpreted as polyunsaturated fat protecting individuals from developing disease. This paper describes researchers investigating exposure subgroups, rather than reporting overall pooled results consistent with the original hypothesis (that saturated fat causes coronary disease). This suggests post-hoc analysis and enthusiasm on the part of researchers to "prove a hypothesis" in the face of generally unsupportive statistical evidence. We are unclear as to why this one positive association is so widely reported when the overall picture from a range of systematic reviewers shows little support for such a statistical association.

The objection to the statistical approach used by Jackobsen et al. (beyond the weak associations produced) is twofold; firstly, interpreted this way, the result is a hypothesis, not a finding. No one in the cohort studies actually changed their diet, so we do not really know what the effect of any change would be. The RCTs conducted in the past to test this hypothesis did not produce conclusive results, but were suggestive of benefit from omega 3 fatty acids only (Ramsden et al., 2013).

Secondly, as stated by Mozaffarian et al.(2010), in a meta-analysis of fatty acid substitution RCTs, the substitution method *"cannot distinguish between potentially distinct benefits of increasing PUFA versus decreasing SFA.*" This method, whether used in RCT design or cohort sub-group analysis, establishes an unnecessary contest between PUFAs, which are essential nutrients and likely to be protective through mechanisms including effects on blood clotting, inflammation, and endothelial function, and SFAs which are inessential sources of dietary energy. It ignores the possibility that similarly high intakes of SFA, monounsaturated fatty acids (MUFA) and mixed PUFAs together (proportions in which these nutrients are likely to have appeared in the diet during human evolutionary history) may be harmless or beneficial.

The meta-analysis by Skeaff and Miller (2009) found that higher intakes of total fat and saturated fat were not significantly associated with CHD in cohort studies, but that various substitutions of PUFA for SFA had beneficial associations in RCTs. However, the strength of results from RCT meta-analysis is highly dependent on selection criteria; inclusion of the Finnish Hospital study (Turpeinen, Pekkarinen, Miettinen, Elosuo, & Paavilainen, 1979), with its unusual "revolving door" methodology and confounding drug use, increases the likelihood of findings favourable to PUFA substitution, while adding the newly recovered data from the Sydney Diet Heart Study has the opposite effect (Ramsden, et al., 2013).

Furthermore, it is possible to interpret the "substitution" data in more ways than one. Jakobsen and colleagues (2009) predicted, from cohort studies analysed in this way, that substituting PUFA for SFA would reduce the risk of CHD deaths. However, substituting carbohydrate for SFA would increase the risk of CHD deaths, especially in women, and substituting MUFA for SFA would increase the risk of coronary events, but not deaths. A logical inference from this finding is that saturated fat should not replace PUFA in the diet, but should instead replace both carbohydrate and MUFA. If this proposal seems outrageous, it is no more so than interpreting the data in favour of reducing saturated fat.

It seems a serious failing, from a public health perspective, that findings regarding all-

cause mortality, or other specific causes of mortality apart from CHD, are neglected in most of the fat substitution analyses under discussion. The null finding by Hooper et al. (2011) for total mortality, in a paper which concludes in favour of substituting PUFA for SFA, deserves comment, because the overall mortality statistic is a weighted average of the effect of an intervention on a disease specific endpoint, and the effect of an intervention on other diseases. Total mortality is also the endpoint least prone to error and manipulation, so that the association between intervention and overall mortality is likely to be less biased than for disease-specific mortality endpoints. If an intervention reduces cardiovascular disease (CVD) death, and statistical power is adequate, then it should also reduce overall mortality, if the intervention has a neutral effect on death from other causes.

The method that Jakobsen et al. (2009) use is based on the *a priori* assumption that dietary SFA is harmful, and that the proportion of energy intake from SFA needs to be reduced. It thus supplies a circular argument that reinforces its own starting bias. There is also an unfortunate tendency to combine omega-3 long-chain PUFA in with other PUFAs to which it is not in fact equivalent, and which can abrogate the benefits of omega-3 long-chain fatty acids experimentally (Madsen & Kristiansen, 2012).

The evidence against saturated fat, such as it is, can be interpreted in more than one way, and, we submit, the parsimonious hypothesis should be, that "essential fatty acids are essential".

3b) Choose and prepare foods and drinks: low in salt (sodium); if using salt, choose iodised salt

The dietary recommendation to ‘choose and prepare foods that are low in salt (sodium)’ is based on the use of blood pressure as a surrogate for cardiovascular health. Indeed adequate intakes (AI) and the tolerable Upper Limit (UL) for sodium as set by the Institutes of Medicine of the United States National Academies (2005) and endorsed by the Ministry of Health (2006) recommendations are based upon this correlation. Reducing salt intake reduces blood pressure by between 1 and 3.5% (Graudal, Hubeck-Graudal, & Jurgens, 2011), however the evidence linking salt (sodium) reduction with improved mortality and morbidity is lacking and is insufficient to translate to public health recommendations. The evidence suggests that:

1) Reducing salt intake has no effect on population morbidity or mortality prevalence;
2) Low salt intakes are negatively associated with health outcomes in some population groups;
3) Population health guidelines that are not underpinned by evidence may serve to confuse end users further, thus reducing compliance with (legitimate and scientifically robust) guidelines; and
4) Reducing salt intakes further may negatively affect iodine status.

Reducing sodium intake has no effect on mortality nor morbidity

A 2011 meta-analysis of RCTs of at least 6 months did not find evidence for reduced mortality or CVD mortality, and concluded that there was no evidence available to support dietary advice to reduce salt intake. In addition, they noted an *increase* in all-cause mortality in those with heart failure who were advised to reduce their intake (Taylor, Ashton, Moxham, Hooper, & Ebrahim, 2011).

Although not supporting that low sodium intakes were positively correlated with morbidity or mortality in general, the Institute of Medicine of the National Academies *Sodium Intake in Populations: Assessment of Evidence* (2013) noted that the evidence suggests that outcomes for those with congestive heart failure are worsened by reductions in sodium. This group suggested a risk of adverse health outcomes associated with sodium intake levels in ranges approximating 1,500 to 2,300 mg per day in other disease-specific population subgroups, specifically those with diabetes, chronic kidney disease (CKD), or pre-existing CVD(Medicine, 2013)(Medicine, 2013)(Medicine, 2013)(Medicine, 2013), and noted no significant correlation between improved health outcomes and reductions in dietary sodium.

Current sodium intakes are safe

Mortality and morbidity are increased at both high and low levels of sodium intake (Alderman & Cohen, 2012; Graudal, Jürgens, Baslund, & Alderman, 2014), suggesting a 'U-curve' of morbidity related to extremes of intake (consistent with normal outcome curves for deficiencies/toxicities of other nutrients). The range within which no discernible health effects are seen lies between 2,645 and 4,945 mg (Graudal, et al., 2014) or as high as 6000mg (Alderman & Cohen, 2012).

The average intake of sodium in New Zealand has been estimated at 3900mg per day (McLean, Williams, Mann, & Parnell, 2012), a level well within the range indicated as having no effect (positive or negative) on mortality and morbidity and so the recommendation to reduce sodium intake is in our opinion unnecessary.

Further reductions in salt intake may increase iodine deficiency

We recognise the important role that iodised salt has played on reducing iodine deficiency and goitre in New Zealand. Dietary exposure to iodine has steadily decreased since 1982 (B. M. Thomson, Vannoort, & Haslemore, 2008). Thomson (2004) in a review of selenium and iodine status in New Zealand, found that iodine levels have been falling since the 1980's, and this is correlated with clinical measures of thyroid status, and that public health interventions to reduce salt intake may further reduce iodine status.

3c) Choose and prepare foods: with little or no added sugar.

We support this recommendation. There are data supporting the reduction of sugar in the diet including the research from the Nurses' Health Study analysed by Liu and colleagues (2000) which showed that women who consumed diets with a high glycaemic load (increased blood glucose excursions associated with intake of sweets or highly processed starches and sweets) had an increased CHD risk, with those in the highest quintile having a >2-fold risk during 10 years of follow-up.

This evidence is further corroborated by the recent study by Yang and colleagues (2014). These researchers found a 2.75-fold increased risk of CHD in persons consuming 25% or more of calories as added sugar, compared to those consuming 10% or less. The strength of this association at extreme intakes gives confidence that smaller effects seen at lower intakes are also likely to be valid, and that the recommendation to avoid added sugar is sound.

However, the evidence to support a high carbohydrate, low fat diet in the prevention and treatment of chronic disease is limited. While some evidence does exist, favourable

outcomes are evident only when these diets are compared to control group(s) consuming the standard industrial food diet, and often with a multicomponent intervention (e.g., parallel stress reduction and exercise components). When head-to-head comparisons are made to the types of eating patterns referenced above the carbohydrate-restricted diets confer better outcomes in the short and medium term.

Firstly, there is a strong argument that carbohydrates *per se* are not an essential nutrient (Westman, 2002). It is also likely that carbohydrate in the proportions recommended by the Dietary Guidelines resembles a dietary composition unlike that which most ancient humans evolved and subsisted on (mostly free of chronic disease, with high median life expectancies (Gurven & Kaplan, 2007)). In fact, anthropological evidence shows these diets to be relatively low in carbohydrate and high in fat depending on geographical location. However, few would deny that fruits and vegetables, especially non-starchy vegetables are a vital source of essential vitamins, minerals and other micronutrients. But the contention whether high carbohydrate regimes should be recommended remains, especially when the risks associated with hyperinsulinaemia are considered. In summary, when the body becomes resistant to insulin's actions, more insulin is secreted to compensate and restore glucose homeostasis (Weir & Bonner-Weir, 2004). This resultant hyperinsulinaemia is considered a key contributor to several different pathophysiologies including inflammation, increased production of reactive oxidative species (ROS), insulin-like growth factor-1 (IGF-1) and triglycerides, and decreased nitric oxide production (Ceriello & Motz, 2004; Giovannucci et al., 2010; Matafome, Santos-Silva, Sena, & Seiça, 2013). Furthermore, hyperinsulinaemia itself contributes to insulin resistance, thereby exacerbating via a positive feedback loop. Combined, these factors damage DNA, and can cause fatty liver disease, Alzheimer's disease, type 2 diabetes, cancer, and endothelial dysfunction resulting in (but not limited to), renal, hepatic and all forms of cardio- and cerebro-vascular disease (Bourdel-Marchasson, Lapre, Laksir, & Puget, 2010; Craft, 2009; Stout, 1990).

Carbohydrate restriction is supported by sufficient evidence to be considered as a therapeutic option for the primary or adjunctive treatment of fatty liver disease (Tendler et al., 2007); type 1 diabetes (Nielsen, Gando, Joensson, & Paulsson, 2012); type 2 diabetes (Yancy, Foy, Chalecki, Vernon, & Westman, 2005); cancer (Fine et al., 2012); and cognitive impairment (Krikorian et al., 2012).

Evidence suggests that carbohydrate restriction is likely superior to unrestricted carbohydrate, low-fat diets for improving cardiometabolic markers (Ebbeling et al., 2012; McAuley et al., 2006; Shai, et al., 2008). It should be noted that LDL-cholesterol may incur a transient increase with carbohydrate restriction. This may now be of little consequence as undifferentiated LDL, with respect to particle size or count, is no longer considered to have the greatest impact on cardiac risk; especially when compared to triglycerides, HDL-cholesterol and especially the triglyceride: HDL ratio (Sikaris, 2014).

An objection regarding low carbohydrate diets is that they are not known to be safe in the long term. We believe this logic to be misleading and fallacious on the two grounds. The first is that there is good evidence that humans have existed (chronic) disease free, with reasonable median life expectancies for most of human history on such diets (Eaton, Cordain, & Lindeberg, 2002). Humans in traditional lifestyles eating this way do well. Second, there is no reason to believe from a biological and mechanistic point of view that these types of diets provide any long term health risk given the medium term

improvements in metabolic markers and lack of nutrient deficiency.

To steer the public away from diets known to confer good metabolic health and towards diets known to produce, on average, inferior results is something the National Dietary Guidelines should not do. We contend that the current guidelines support a diet that will produce chronic hyperinsulinaemia in those who are insulin resistant. Thus, those most at risk of metabolic disease are most likely to be adversely affected by high carbohydrate diets, even of the low glycemic index type described by the dietary guidelines. More work does need to be done to fully elucidate this contention.

4. Make water your first choice for drinks.

We agree with this recommendation.

5. Buy, prepare, cook and store food to ensure food safety.

Whilst our team has no expertise in this area, we agree with the consumer Food Safety guidelines published by the Ministry of Primary Industries. As such, we regard the food safety recommendations in this guideline redundant.

6. If you drink alcohol, keep your intake low. Don't drink if you are pregnant or planning to become pregnant.

We agree with this recommendation.

Summary

Overall, we submit that the proposed dietary guidelines, which currently detail a diet eaten mainly by European, university educated professionals and the worried well, should instead be flexible enough to reflect the variety of healthy and nutritious dietary patterns possible in a multicultural society.

Appendix A: An alternative set of dietary guideline for New Zealanders

The real food guidelines
Real food for real people, based on real evidence

1. Enjoy nutritious foods everyday including plenty of fresh vegetables and fruit.
2. Buy and prepare food from whole unprocessed sources of dairy, nuts, seeds, eggs, meat, fish and poultry
3. Keep sugar, added sugars, and processed foods to a minimum in all foods and drinks.
4. If you drink alcohol, keep your intake low. Don't drink if you are pregnant or planning to become pregnant.
5. Prepare, cook, and eat minimally processed traditional foods with family, friends, and your community
6. Discretionary calories (energy foods) should:
    a) Favour minimally refined grains and legumes, properly prepared, over refined or processed versions, and boiled or baked potatoes, kumara or taro over deep fried or processed potato fries and chips.
    b) Favour traditional oils, fats and spreads over refined and processed versions.

Appendix B: The lipid hypothesis; an obsolete explanation for a dubious association

Despite the negative statistical evidence of association between saturated fat intake and cardiovascular disease, the link is often supported with reference to the link between saturated fat intake and low density lipoprotein (LDL) cholesterol concentration. It is believed that LDL cholesterol is the dominant mechanism by which dietary fats exert their purported harms and benefits, even when randomised and observational studies show no overall association between saturated fat intake and disease risk.

Thus, substituting MUFA for SFA either lowers, or has no effect on, LDL, but Jakobsen et al. (2009) report that this substitution will increase CHD risk. SFA elevates LDL, at least in the short term, but has no association with CHD risk (except when SFA intake is creatively reconceptualised by factoring in a corresponding reduction in the intake of PUFA, especially omega 3 long chain fatty acids. In RCTs, substituting PUFA for SFA has lowered LDL while increasing CVD death rates (e.g. Sydney Diet Heart Study), or lowered CVD mortality while leaving LDL unchanged (Lyon Diet Heart Study).

In the words of the well-known medical statistician, Austin Bradford-Hill, "What is biologically plausible depends upon the biological knowledge of the day." The theory developed in the mid-20th century, that saturated fat causes heart disease because it elevates LDL cholesterol (the lipid hypothesis), is not consistent with today's epidemiological evidence, and is also outdated in terms of current knowledge of cholesterol transport. LDL can now be distinguished by (amongst other things) particle size and number, and LDL particle size and number correspond more closely to CHD risk than total LDL or total cholesterol, and are influenced by both carbohydrate and fat intake, e.g. as reported by Siri and Krauss (2005).

Appendix C: Letter of criticism to Professor Schofield's (and team) view on current dietary guidelines

NB: Removed as requested from author.

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