Review of the evidence for the potential impact and feasibility of substituting saturated fat in the New Zealand diet

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Abstract

Objective: To estimate the potential impact on cardiovascular health of modifying dietary intake of saturated fat across the New Zealand population, and whether this would be appropriate and feasible.

Methods: First, a literature review of meta-analyses was conducted to estimate the magnitude of reduction in risk for cardiovascular events in response to a reduction in dietary saturated fat intake (with or without substitution with other macronutrients). Second, data from the New Zealand Adult Nutrition Survey 2008/09 were used to determine whether a change to the population's dietary fat intake would be warranted and feasible.

Results: Five relevant meta-analyses were identified. No significant association between saturated fat intake alone and cardiovascular disease was found. However, the incidence of cardiovascular disease events was less when dietary saturated fats were replaced with polyunsaturated fats, reducing the risk of cardiovascular events by about 10%. Compared with nutritional guidelines, New Zealanders' current saturated fat intake is excessive while polyunsaturated fat intake is inadequate; both would be corrected by a substitution of 5% of daily energy intake.

Conclusions: Replacing 5% of daily energy consumed as saturated fat with polyunsaturated fats would be expected to reduce cardiovascular events by about 10%.

Implications: In order to achieve the population-wide dietary fat modifications needed to improve cardiovascular health for New Zealanders, a public health strategy (e.g. fiscal, regulatory and/or educational interventions) must be implemented. Further work is needed to establish the cost-effectiveness of the various strategies.

***Key words*:** Dietary fats, unsaturated fats, saturated fats, polyunsaturated fats, cardiovascular disease

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Cardiovascular disease (CVD) is a leading cause of death in New Zealand, accounting for around one-third of all deaths in 2009.[1] More than 5% of the adult NZ population have been diagnosed with ischaemic heart disease (IHD) alone.[2] CVD is thus a high priority for preventative public health interventions.

The majority of IHD deaths in NZ are attributable to nutrition-related risk factors, with high blood cholesterol levels believed to have contributed to at least half of all CVD deaths in 1997.[3] The mean serum total cholesterol level was borderline high at 5.13 mmol/L in a study of nutrition in New Zealanders;[4] levels <4 mmol/L are ideal. More than 300,000 New Zealanders are receiving cholesterol-lowering treatments at a cost of more than NZ$40 million per year.[5]

These factors emphasise the need for a population-wide approach to reducing cholesterol levels in the NZ population. Cholesterol-lowering drugs, such as statins, are effective in lowering cholesterol levels and reducing cardiovascular risk.[6,7] However, while pharmaceutical treatment may be appropriate on a person-by-person basis, depending on the individual's level of risk, it would be undesirable (and unacceptable to many people) to recommend that all individuals above a certain age receive pharmaceuticals to shift the population's distribution of cardiovascular risk. Furthermore, greater benefits may be achieved from public health interventions to reduce the risk for CVD across the large number of people within the population who are at low risk of the disease, compared to interventions aimed at the lesser number of high-risk individuals.

Population-wide implementation of dietary changes related to fat intake could potentially address certain determinants of CVD incidence at the root of the problem, and are likely to be cost-effective. New Zealanders are currently consuming higher than recommended levels of saturated fat and lower than recommended levels of polyunsaturated fats.[4] This imbalance may be amenable to population-wide dietary interventions. Replacement of dietary saturated fats with polyunsaturated fats lowers total cholesterol and low-density lipoprotein (LDL) cholesterol levels.[8-11]

Regulatory actions to improve health behaviour are not uncommon. Higher prices may reduce consumption of foods high in saturated fat.[12] Certainly, the converse was shown in the recent Supermarket Healthy Options Project (SHOP) conducted in NZ, in which a 12.5% reduction in price of healthy foods led to a significant 11% increase in the purchase of healthy foods.[13]

In October 2011, Denmark was the first jurisdiction to introduce a tax on saturated fat to supplement existing pro-health taxes on soft drinks, chocolate and other sweets. This new saturated fat tax was applied to a wide range of foods which had a saturated fat content exceeding 2.3% (attracting a tax at the level of 16 Danish kroner (≈NZ$3) per

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kilogram of saturated fat).[14] This tax was discontinued after a year, apparently due to such concerns as job losses during the economic crisis and the effect of cross-border shopping in Germany and Sweden[15] (a problem that island nations such as NZ and Australia would not face). If such a tax was applied in NZ, it would increase the cost of a 500 g pack of butter by about NZ$1 and would raise revenue that could be used to improve nutrition in other ways (e.g. providing healthy school lunches). Other countries are still moving in this direction. Hungary, for instance, has recently introduced taxes on foods with high fat, sugar and salt content.[16]

This paper considers whether a public health intervention (such as a saturated fat tax or other regulatory or educational strategies) to reduce dietary intake of saturated fat across the population might be appropriate in NZ to improve cardiovascular health. First, a literature review was conducted to estimate how effective a reduction in saturated fat intake would be in reducing risk for cardiovascular events, considering both reduction of saturated fat intake in isolation, and replacement of saturated fat with other macronutrients. Second, data on dietary patterns in NZ were used to determine whether pushing a change to the population's dietary fat intake would be warranted and feasible.

Methods

Literature search

A literature search was undertaken to estimate to what extent a reduction in intake of dietary saturated fats affects the relative risks (RRs) for CVD or IHD events in adults in developed countries.

With the aim of finding the best evidence rather than all evidence, the literature search was structured to identify high-quality systematic reviews relevant to the research question, rather than identifying individual studies. Given the large body of evidence, the search was limited to systematic reviews published since 2000. Medline, Embase, the Cochrane library, the DARE and HTA databases, and the New Zealand Ministry of Health publications webpage were searched. Keywords (mapped to subject headings where possible) were: saturated fats, OR; dietary fats, OR; dietary cholesterol, AND; cardiovascular disease (exploded where possible). In Medline, the limit "reviews (best balance of sensitivity and specificity)" was applied. Snowballing to identify additional reviews from reference lists of relevant papers was also carried out.

Papers were considered potentially relevant if they focused on interventions that aimed to reduce saturated fat intake, either in isolation or by replacement of saturated fats with polyunsaturated fats, monounsaturated fats or carbohydrates. Papers addressing the risk of IHD or CVD events according to saturated fat intake were also included as being of secondary interest. Papers that focused on supplementation with healthy oils (rather than a reduction in saturated fat intake as the primary intervention), multifactorial interventions or use of antihyperlipidaemic drugs were excluded.

To be included, reviews had to: describe a systematic process for searching for and selecting relevant articles; include general populations of adults (with or without previous cardiovascular events); include IHD and/or CVD events as outcomes; and have been published in 2000 or later. Reviews describing only effects on cardiovascular risk factors (rather than events) and those that focused on a specific high-risk population (e.g. diabetics) were excluded.

Of the 368 citations retrieved, 53 were considered potentially relevant based on initial title scanning (see Figure 1). After more detailed application of inclusion and exclusion criteria to these 53 papers, seven relevant citations were retained. Most papers that were excluded at this stage were editorials or descriptive reviews that did not meet our criteria for systematic review; for instance, 81% of Medline-identified articles were excluded for this reason. The other most common reasons for exclusion were that the review covered a range of interventions and had inadequate details on reduction/modification of saturated fat intake, or an updated version of the review had been published.

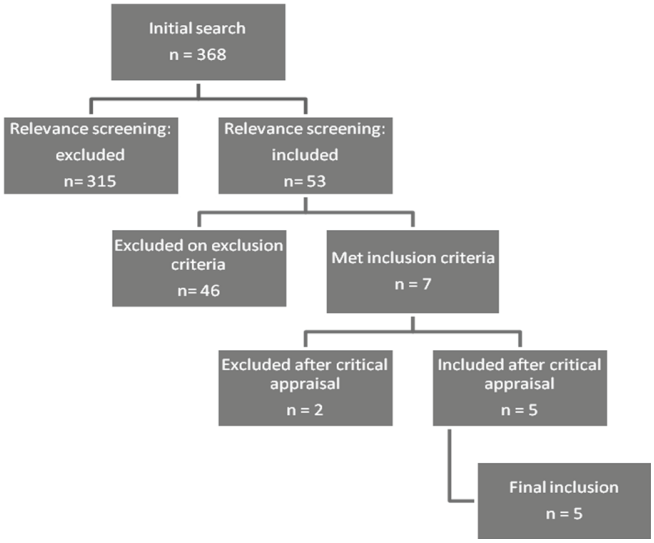
The seven reviews that met the inclusion criteria were critically appraised using the CASP tool[17]. Two studies were then excluded: one review did not describe use of adequate systematic methods[18] and the other had inadequate detail on the outcomes of interest.[19]

Thus, five systematic reviews with meta-analyses were included in the analysis.[9,20-23] The analysis by Jakobsen et al.[21] was a pooled analysis of individual-level data, but such data could be obtained for only 11 of the 14 studies considered to be relevant. Thus, this analysis is not strictly systematic. Nevertheless, it has been described as robust and of higher quality than earlier meta-analyses,[9,20] and therefore it was included.

New Zealand Dietary Analysis

Data for the dietary analysis were primarily from the 2008/09 New Zealand Adult Nutrition Survey, which collected information from a national sample of 4,721 New Zealanders 15 years and over.[4] Data collected included self-reported food and nutrient intake (24-hour diet recall) and information on dietary habits and eating patterns. Anthropometric measures were also taken, along with blood and urine samples.

Figure 1: Flow diagram of literature search.



Results

The five meta-analyses of the effect of reducing (or modifying) dietary saturated fat intake on cardiovascular events were published between 2009 and 2012, but included studies dating back to the 1960s.[9,20-23] They only included studies from developed countries, with the majority being from the US and Europe. All had adult participants. Outcomes from cohort studies and randomised controlled trials (RCTs) were analysed separately. Both primary and secondary prevention studies were included in most analyses.

Reduction of saturated fat intake

No significant association between saturated fat intake alone and CVD was found. This was addressed by two meta-analyses of cohort studies, neither of which found an increased risk of IHD or CVD events or death when highest dietary saturated fat intake was compared to lowest intake.[20,22] Furthermore, two meta-analyses of RCTs found that interventions that reduced dietary saturated fat intake in isolation did not significantly reduce the incidence of IHD/CVD events or death.[20,23]

Modification of dietary fat intake

Four meta-analyses addressed outcomes of replacing dietary saturated fatty acids (SFAs) with polyunsaturated fatty acids (PUFAs), monounsaturated fats (MUFAs) or carbohydrates.

The meta-analyses of Skeaff et al.[20] and Mozaffarian et al.[9] included the same eight RCTs of SFA/PUFA replacement; Skeaff et al. included one additional small RCT. The meta-analysis by Skeaff et al. was inconsistent with regards to the endpoints extracted from the individual studies, whereas the meta-analysis by Mozaffarian et al. included only 'hard' IHD endpoints: nonfatal myocardial infarction (MI); IHD death; and/or sudden death. Thus, the results from only the Mozaffarian et al. meta-analysis are presented in detail for SFA/PUFA replacement.

The Hooper et al.[23] review was a Cochrane review. However, the interventions were more widely defined than the other meta-analyses (e.g. cointervention with oil supplementation, fruit and vegetables etc. was permitted), and thus analyses showed greater heterogeneity than the other meta-analyses, and results may be less reliable. Likely due to the broader criteria, the Hooper et al.[23] review identified 15 RCTs of fat modification, compared with eight and nine in the Mozaffarian et al.[9] and Skeaff et al.[20] reviews, respectively.

The design of the three meta-analyses of interest are shown in Table 1. All of the meta-analyses used random effects models, although Hooper et al. included a fixed effects model in a sensitivity analysis.[23] Only the Jakobsen et al. analysis used individual-level data.[21]

The analysis by Jakobsen et al. modelled a 5% reduction in energy intake from SFAs and a concomitant 5% increase in energy intake from PUFAs (i.e. isocaloric replacement).[21] Their model also adjusted for age, body mass index, physical activity, history of hypertension, education level, age, smoking, alcohol intake, fibre and cholesterol intake. In Mozaffarian et al.,[9] results were reported according to the increase in PUFA intake (as a percentage of daily energy) being a replacement for SFA intake; the replacement was presumably isocaloric although this was not explicitly stated. In the Hooper et al. analysis, a modified diet was defined as including ≥30% daily energy from total fats and higher levels of MUFAs or PUFAs than a 'usual' diet. Thus, the extent (if any) of reduction in SFA intake was unclear, and findings from this report may be less useful for informing isocaloric SFA/PUFA replacement.

SFA/PUFA replacement

The results of the meta-analyses for SFA/PUFA replacement are shown in Table 1. There is a reasonable degree of consistency in the results from the meta-analyses, all of which found that the incidence of CVD events was reduced when dietary saturated fats were replaced with polyunsaturated fats.

The Mozaffarian et al.[9] meta-analysis of RCTs found that the overall risk of IHD events was reduced by 19% (RR 0.81: 95% CI 0.70, 0.95). This was the result of SFA/PUFA replacement equivalent to 9.9% of daily energy. Thus, the reduction in IHD events per 5% of daily energy SFA/PUFA replacement was 10% (RR 0.90; 95% CI 0.83-0.97).[9] A similar effect was reported from the Jakobsen et al. meta-analysis of cohort studies, in which there was a 13% reduction in IHD events per 5% of daily energy SFA/PUFA replacement (RR 0.87: 95% CI 0.77, 0.97). An 18% reduction in CVD events was reported by Hooper et al. for fat modification; this was marginal in terms of statistical significance (RR 0.82: 95% CI 0.66-1.02), but effects were significant amongst studies of longer duration (two years or more).[23]

The effects on IHD or CVD mortality are less clear. Jakobsen et al. reported a 26% reduction in IHD death per 5% daily energy SFA/PUFA replacement (0.74: 95% CI 0.61, 0.89).[21] A significant reduction in IHD death of 20% (RR 0.80: 95% CI 0.65, 0.98) was also reported by Mozaffarian et al, but this equates to a 10% reduction per 5% daily energy SFA/PUFA replacement − less than half that reported by Jakobsen et al.[9] Furthermore, the Skeaff et al. analysis of largely similar data did not find the effect of fat modification on IHD death to be significant (RR 0.84:95% CI 0.62, 1.12).[20] No significant reduction in CVD death was found by Hooper et al.[23]

Primary versus secondary prevention was not found to be a significant determinant of the risk reduction in Mozaffarian et al.,[9] and effects did not differ by CVD risk level in Hooper et al.[23] The meta-analyses of cohort studies by Siri-Tarino et al.[22] and Jakobsen et al.[21] reported that there was no effect modification by sex or age. However, it should be noted that there were relatively constrained age bands included in the studies, meaning that conclusions about younger and older age groups (75+ years) cannot be drawn. For instance, the analysis by Jakobsen et al. excluded patients younger than 35, and the included studies had median ages of 47 to 61 (80% central range 39 to 76 years).

The duration of the SFA/PUFA modification diet does appear to be important, and any CVD reduction benefit may not be maintained if the dietary modification is stopped. Study duration was an independent determinant of the magnitude of risk reduction

in Mozaffarian et al.[9]; the effect on IHD events was only significant in trials greater than the median of ≥4.25 years' duration (RR 0.73 [95%CI 0.61-0.87] vs. 0.91 [95%CI 0.76-1.10]).[9] Similarly, fat modification significantly reduced CVD events only when the duration of the intervention was greater than two years in Hooper et al.[23] One of the individual RCTs included in the meta-analyses (the Finnish Mental Hospital Study[24,25]) provides useful information on duration of effect because of its crossover design involving two hospitals. One hospital provided the modified fat diet – while the other provided a normal diet – for six years, and then the diets were swapped. After completion of the first six-year modified SFA/PUFA diet stage and return to the normal diet in the second 6-year phase of the study, not only did reductions in serum cholesterol levels rapidly reverse but the protective effect against IHD also reversed.[24,25]

The magnitude of benefit may be greater when the diet is adhered to sufficiently to produce a significant reduction in serum cholesterol levels.[20] However, Mozaffarian et al. did not find any significant difference in outcomes between institutional studies where food was provided and adherence would be expected to be high, and those performed in free-living situations where participants were responsible for their own diets. The RR for CHD events was 0.76 (95%CI 0.55-1.04) for the four institutional studies where meals were provided (n=10,721) vs. 0.84 (95%CI 0.72-0.98) for the four free-living studies that generally provided only dietary advice (n=2893).[9]

Other dietary modification

Replacing saturated fat with carbohydrates did not reduce the risk of coronary events in the meta-analysis by Jakobsen et al.[21]

When other fats were adjusted for, such that the analyses represented replacement of carbohydrates rather than saturated fat, Skeaff et al.[20] found that a 5% increase in MUFA intake did not have a significant effect on IHD events or death, while a 5% increase in PUFA intake protected against IHD events (RR 0.84; 95%CI 0.70-1.00; $p<0.049$) but not IHD death.

Application to New Zealand Dietary Patterns

Dietary intake of fat is recommended to be no lower than 15−20% and no higher than 30−35% of daily energy, with less than 10% of daily energy being from saturated and/or trans fats.[10,26] Currently, New Zealanders are consuming, on average, almost 34% of daily energy as fat including 13% of energy as saturated fat,[4] i.e. at least 3% higher in absolute terms than the maximum recommended level of saturated fat.[10,26] Notably, these figures are from self-reported data that may under-report energy intake,[27] so saturated fat intake may be even higher than reported. Furthermore, many New Zealanders are

Table 1: Summary of meta-analyses of studies of the effect on CVD/IHD events of replacing dietary saturated fat (SFAs) with polyunsaturated fats (PUFAs).

	Mozaffarian et al[9] (2010)	Jakobsen et al[21] (2009)	Hooper et al[23] (2012)
Methods			
Population	Primary or secondary prevention	Primary prevention	Any level of cardiovascular risk
Intervention	Increased PUFA intake (total and/or omega-6) replacing SFA vs usual or control diet. Excluded studies of omega-3 PUFAs.	5% energy intake of SFA replaced by isocaloric PUFAs or carbohydrates. PUFAs could include omega-3 and -6.	Modified diet with increased PUFA or MUFA, with total fat intake maintained ≥30% of daily energy.[a] Omega-3 allowed if part of fat modification, but not if given alone.
Outcome measures	'Hard' IHD events (MI, IHD death, sudden death)	Nonfatal MI, IHD death, sudden death	All fatal or nonfatal CVD events[b]
Study type	RCTs >1 year duration	Cohort	RCTs > 6 month duration
No. studies	8	11[c]	15
No. subjects	13,614	344,696	13,004
Follow-up	Median 4.25 years	4-10 years	≤2 year and ≥2 year subgrouping
Results (Relative risk with 95% confidence interval, versus control diet)			
RR for events	IHD: 0.81 (0.70-0.95)*		CVD: 0.82 (0.66-1.02)
RR for death	IHD: 0.80 (0.65-0.98)*		CVD: 0.92 (0.73-1.15)
RR for events per 5% energy SFA/ PUFA replacement	IHD: 0.90 (0.83-0.97)*	IHD: 0.87 (0.77-0.97)*	
RR for death per 5% energy SFA/ PUFA replacement		IHD: 0.74 (0.61-0.89)*	
Heterogeneity	Heterogeneity moderate	No significant heterogeneity	Heterogeneity moderate-to-important

a Interventions were allowed to include supplementation and other co-interventions such as increased fruit and vegetables.

b MI, angina, stroke, heart failure, peripheral vascular events, atrial fibrillation, unplanned bypass or angioplasty

c Individual-level data from the 11 studies were pooled.

CVD = cardiovascular disease; IHD = ischaemic heart disease; MI = myocardial infarction; MUFA = monounsaturated fatty acids; PUFA = polyunsaturated fatty acids; RCT = randomised controlled trial; RR = relative risk; SFA = saturated fats

** = statistically significant effect.*

not consuming enough polyunsaturated fat, with an average intake of 5% of daily energy,[4] while recommended levels are 6-11%.[10,26]

The results from the meta-analyses in the previous section suggest that substituting 5% of daily energy from saturated fats with polyunsaturated fats across the NZ population would reduce the incidence of IHD events by about 10%.[9,21] The dietary data confirm that such a change would be desirable, and would place New Zealanders' fat intake within recommended levels for both saturated fats and PUFAs.

Such a dietary change would also appear to be feasible for most people when spread across a day's food intake. In 2008/09, the usual median daily intake in NZ was 10,380 kJ for males and 7,448 kJ for females across the adult population.[4] Thus, 5% of daily energy is 520 kJ for men and 370 kJ for women. One gram of dietary fat provides 37.7 kJ.[4] Therefore, a substitution of 5% of daily energy intake amounts to replacing 14 g of saturated fat with PUFA daily for men, and 10 g daily for women.

Such a change could be achieved relatively easily within NZ diets. Some examples of possible food substitutions to achieve a 15 g decrease in saturated fat and a concomitant increase in PUFA intake are shown in Table 2. For instance, two tablespoons of butter could be replaced by two tablespoons of a high-PUFA oil such as grapeseed or safflower oil, or 65 g of cheddar cheese could be replaced by 30 to 60 g of high-PUFA nuts and seeds such as walnuts or sunflower seeds. Use of margarine would reduce saturated fat intake compared to butter, but the amount needed to deliver 15 g of PUFAs would still contain a significant amount of saturated fat (10-15 g), and thus high-PUFA oils would be preferred to achieve optimal substitution. Replacement of fatty red meat with oily fish would reduce saturated fat and provide some increase in PUFA intake, although additional sources of PUFA might also be needed.

Discussion

In considering whether a public health intervention to reduce dietary intake of saturated fat would be appropriate, a fundamental issue is whether saturated fat is an appropriate target. This is not as straightforward as may have previously been assumed, given that reduction of saturated fat in isolation does not appear to reduce risk of cardiovascular disease. The key appears to be what the saturated fat is replaced with. While fat modification has been studied for over 50 years, results of individual trials of fat modification have often been contradictory, at least partly due to insufficient power to detect relatively small but potentially important effects (e.g. 10% change in risk). It is the more recent meta-analyses that allow a clearer picture of the effects of fat modification to be seen.

There is consistent evidence from the meta-analyses presented here to show that replacing saturated fats with polyunsaturated fats is beneficial in preventing CVD events. Polyunsaturated fats not only improve the lipid profile, but also have anti-inflammatory effects.[28] We estimate that a replacement of 5% of daily energy intake of saturated fats with polyunsaturated fats across the population would reduce the incidence of IHD events by about 10%. There are inadequate data to draw conclusions regarding whether replacing saturated fats with monounsaturated fats is beneficial in preventing IHD (noting that olive oil may be an exception because of its wide-ranging beneficial cardiovascular effects).[29] Replacement of saturated fat with carbohydrates has not been shown to be beneficial to date, but further investigation of the effects of replacement of saturated fats with complex carbohydrates with a low glycaemic index is required.

Importantly, nutritional data show that New Zealanders are currently consuming too much saturated fat and not enough polyunsaturated fat, such that a SFA/PUFA replacement of 5% of daily energy intake would be both feasible and beneficial. The changes that would be required to dietary habits would seem to be attainable for most people. Although New Zealanders have a relatively high meat intake and this may be difficult to modify to a large extent, less than 15% of total daily saturated fat intake comes from unprocessed meat.[4] The top sources of saturated fats in NZ are butter and margarine, milk, bread-based dishes, cheese, and cakes and muffins.[4] Most of these are readily amenable to substitution with lower saturated fat and, in most cases, higher PUFA products or ways of cooking.

Limitations

A criticism of this review may be that data were extracted from meta-analyses rather than individual studies. However, good quality meta-analyses can be considered to be the highest level of evidence.[30] We focused on the two most rigorous meta-analyses identified. The Mozaffarian et al. meta-analysis was of RCTs of over one year's duration, including 13,614 participants, and including only 'hard' IHD endpoints.[9] Nevertheless, there were still some limitations with this meta-analysis. All but two studies were initiated prior to 1970, and in four of the trials participants were fully or partly institutionalised with little access to food other than the study-provided meals. However, the results from Mozaffarian et al. are strongly supported by the Jakobsen et al. meta-analysis of 11 cohort studies that pooled individual-level data for almost 350,000 participants.[21] The latter meta-analysis has been described by the Food and Agriculture Organization of the United Nations as providing "convincing evidence" that replacing saturated fats with polyunsaturated fats reduces the risk of IHD.[10,20] Furthermore, our estimate derived from these meta-analyses (i.e. 5% of daily energy SFA/PUFA replacement would reduce the incidence of IHD events by 10%) is supported by another expert panel that concluded that in Western countries a 1% replacement of energy from SFAs with PUFAs reduces the incidence of IHD by 2-3%.[31]

The nutritional data in this review are from the 2008/09 NZ Adult Nutrition Survey, which is the largest of its kind in NZ and repeats an earlier survey in 1997.[4] While this survey has good validation, the diet data are self-reported, and thus likely to underestimate intake. This suggests that saturated fat intake is even higher than reported, and thus a possibly even higher priority for intervention.

Table 2. Examples of food replacement to achieve a reduction in saturated fats (SFAs) and increase in polyunsaturated fats (PUFAs); serving sizes correspond to the amount needed to reduce SFA by 15g and/or increase PUFA by 15g.[a]

	Fat content (%)		Serving			Substitution
	SFA	PUFAs	Size (g)	SFA (g)	PUFAs (g)	
Examples of foods high in SFA with suggested substitutions						
Butter	53	2.7	30	15	0.8	High-PUFA oils
Cheese (cheddar)	23	1.3	65	15	0.9	High-PUFA nuts and seeds
Cream	25	1.3	60	15	0.8	Low-fat unsweetened yoghurt plus high-PUFA oils, nuts or other foods
Coconut cream	17	0.4	90	15	0.4	Low-fat unsweetened yoghurt plus high-PUFA oils or other foods
Palm oil	49	9.3	30	15	2.8	High-PUFA oils
Corned beef (canned)	5	0.2	280	15	0.6	Canned tuna or other high-PUFA fish
Beef silverside[b]	10	1.0	150	15	1.5	High-PUFA fish and/or lean meats cooked with high-PUFA oils
Lamb (composite cuts)[b]	13	0.7	115	15	0.8	
Pork bacon[b]	14	2.5	110	15	2.7	
Sausages (assorted meats)	10	0.6	145	15	0.9	
Potato crisps	16	3.6	91	15	3.3	High-PUFA nuts and seeds
Examples of foods higher in PUFAs to substitute for high SFA foods						
Oils						
Grapeseed oil	10	70	20	2	15	Butter, margarine
Safflower oil	11	64	25	3	15	
Sesame oil	14	42	35	5	15	
Sunflower	7	41	35	3	15	
Soybean oil	11	38	40	4	15	
Canola oil	7	26	60	4	15	
Rice bran oil	20	28	55	11	15	Due to SFA content, other high-PUFA oils preferred
Olive oil	17	12	130	21	15	High in MUFAs
Margarine						
Margarine high in PUFA	16.3	24.1	60	10	15	Due to SFA content, high-PUFA oils preferred
Margarine high in MUFA (e.g. canola and olive oil based)	16.1	16.5	90	15	15	
Nuts and seeds						
Walnuts	7	43	35	2	15	Cheese and other high-fat snacks (e.g. potato crisps, biscuits)
Sunflower seeds	5	33	45	2	15	
Pine nuts	8	22	70	5	15	
Sesame seeds	7	22	70	5	15	
Tahini (sesame seed paste)	8	28	55	4	15	Peanut butter (13% PUFA), dressings and dips
Omega-3 rich fish						
Tuna (canned in oil)	1.4	5.6	270	4	15	Non-lean red meats
Salmon (red, canned)	2	2.1	714	14	15	
Fresh salmon (New Zealand[c])	4.9	5.2	290	14	15	

a Nutrient contents of food were obtained from New Zealand Food Composition Tables, 9th Edition 2012 (New Zealand Food Composition Database; http://www.foodcomposition.co.nz) and/or the USDA Nutrient Data Laboratory foods database (http://fnic.nal.usda.gov/food-composition/usda-nutrient-data-laboratory). Serving sizes are rounded to the nearest 5g.

b Based on untrimmed meat with lean and fat included.

c www.regalsalmon.co.nz/nutrition. Note PUFA content in fresh salmon is variable and can be as low as 1–2% in some species.

Research and potential policy implications

A number of questions remain with regards to the best public health policy to achieve a 5% substitution of saturated fats with polyunsaturated fats. Any policy should ideally create a permanent change to New Zealanders' diet patterns. The benefit of dietary fat modification in terms of reduction in IHD risk may not be fully achieved in timeframes less than 2-4 years,[9,23] and the protective effects may be lost if the dietary change is not maintained.[24,25]

Although there is some evidence for the long-term effectiveness of nutrition education,[32] education alone is unlikely to be sufficient. For instance, in the New Zealand SHOP trial, education tailored to the individual's food purchasing patterns did not significantly increase purchasing of healthier foods, whereas a reduction in price did.[13]

A review by the World Health Organization in 2006 concluded that there was evidence, albeit mainly indirect, to suggest that policy-related economic tools such as taxes could reduce consumption of foods that are high in saturated fat.[12] Other US researchers have argued that a 'fat tax' would produce only a small change in fat consumption, and is regressive.[33] However, the effectiveness of a saturated fat tax would depend on the amount of that tax, to what extent it leads to price changes (tax pass-through) and finally the price elasticity of demand – that is, the extent to which consumers would reduce the purchase of products high in saturated fat in response to an increase in price. To be effective, the policy must also cause an increase in polyunsaturated fat consumption. This would, at least in part, depend on cross-price elasticity effects – that is, to what extent consumers would buy high-PUFA products instead of high saturated fat products if the price of the latter goes up.

The issue of the regressive nature of a saturated fat tax (i.e. that it will affect low-income people disproportionally more because they spend a greater proportion of their income on food) can be addressed in two ways. First, high-PUFA products need to be available at a price equal to or less than currently purchased high-saturated fat foods. Second, the health benefits should be seen as potentially 'progressive', since those on lower incomes are at great risk of IHD. For instance, the age-standardised rate of death from IHD is 1.6 times higher in Māori than non-Māori.[1]

Prior to considering a Danish-style saturated fat tax intervention, further research is needed on quantifying the likely benefits for NZ (e.g. in terms of health-adjusted life years gained and impact on health inequalities), the costs, and also the cost-effectiveness. Informed comparisons could then be made with alternative fiscal interventions such as taxes on other hazardous food components (e.g. sugar and salt) or on categories of unhealthy foods which are high in salt, sugar, and/or saturated fat.[34] Even a tax on methane from ruminant livestock[35] (which would lead to higher meat and dairy product prices) is an alternative intervention that would probably help prevent CVD and also contribute to the country's international commitments to address climate change. Similarly, a policy package could include low-income New Zealanders having subsidised access to vegetable oils high in PUFAs (e.g. via targeted smart cards for specific food products) or the funding of healthy school meals by central government. More broadly, comparisons could be made with other regulatory interventions (e.g. regulations to set maximum saturated fat levels permitted in foods), with mass media campaigns to improve diets, and with improved nutrition labelling of food (as per Australian modelling work).[34] Indeed, we plan to progress some of these comparisons in future health economic modelling work.[36]

Conclusions

Our review of the highest quality systematic reviews suggests that replacing 5% of daily energy consumed as saturated fat with polyunsaturated fats would reduce IHD events by about 10%, and that such a dietary change would appear to be both desirable and feasible for the NZ population. Public health interventions (fiscal, regulatory and/or educational) to create a sustained reduction in New Zealanders' saturated fat intake, and an increased polyunsaturated fat intake, may be an appropriate way to achieve this, if these are found to be cost-effective compared to other health sector interventions.

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