Position statement

Dietary fats and dietary sterols for cardiovascular health

This position statement provides recommendations to the general population and professionals on the consumption of dietary fats and dietary sterols to manage the lipid profile, maintain cardiovascular health (CVH) and reduce cardiovascular disease (CVD).

Our position on dietary fats and dietary sterols also determines our recommendations for product change within the food industry and recommended policy change from governments to improve the food supply.

Key findings

Summary of evidence. Dietary fats and dietary cholesterol for cardiovascular health[1] **and** ***A review of the relationship between dietary fat and cardiovascular disease***[2]

The 1999 findings are largely upheld, with advances in nutritional science leading to stronger evidence for the negative impact of trans fatty acids (tFA) on CVH.

The key findings in relation to fatty acid classes, individual fatty acids, total fat and dietary cholesterol are as follows.

Saturated fatty acids (SFA)

- SFA intake is associated with coronary heart disease (CHD).
- Increasing SFA intake results in an increase in total cholesterol (TC) and low-density lipoprotein cholesterol (LDL-C) compared to carbohydrate, polyunsaturated fatty acids (PUFA) and monounsaturated fatty acids (MUFA) (1999 evidence statement retained).
- Individual SFA have differential effects on the lipid profile.
- Lowering dietary SFA to < 7% of energy intake with restricted dietary cholesterol results in further LDL-C lowering than diets containing < 10% of energy intake from SFA.
- Replacing SFA with omega-6 PUFA (n-6 PUFA) to achieve a ratio of PUFA to SFA of greater than 1 will reduce the risk of CHD (1999 evidence statement retained).

Our position on dietary fats and dietary cholesterol was developed from a review of the latest scientific evidence[1] and incorporates recommendations from our previous papers: *Position statement. Fish, fish oils, n-3 polyunsaturated fatty acids and cardiovascular health*[3] and *Position statement on phytosterol/stanol enriched foods*.[4] This position statement updates and supersedes our 1999 position statement *A review of the relationship between dietary fat and cardiovascular disease*.[2]

tFA

- tFA intake is associated with an increased risk of myocardial infarction (MI).
- tFA intake is associated with increased CHD incidence and risk of CHD.
- The consumption of tFA increases LDL-C and decreases high-density lipoprotein cholesterol (HDL-C).
- Replacing tFA and SFA with MUFA or PUFA is an effective measure for improving blood lipid profiles.

n-6 PUFA

- n-6 PUFA intake lowers LDL-C.

Total fat

- There is no direct relationship between total fat intake and the incidence of CHD.

Dietary cholesterol

- Dietary cholesterol increases TC and LDL-C, but substantially less so than SFA and tFA (1999 evidence statement retained).

Specific foods

- Within a low SFA diet, individuals can consume up to six eggs per week without adversely affecting CVD outcomes.

Position statement. Fish, fish oils, n-3 polyunsaturated fatty acids and cardiovascular health[3]

The key findings in relation to fish, fish oils and polyunsaturated fatty acids are as follows.

- Fish and fish oil consumption is associated with a reduced risk of CHD, CHD mortality and stroke.
- In secondary prevention, 2 g per day alpha-linolenic acid (ALA) and ≥ 850 mg per day of omega-3 polyunsaturated fatty acids (n-3 PUFA) as fish oil reduces the risk of CHD.
- 1,000–4,000 mg per day of n-3 PUFA as fish oil decreases triglycerides (TG) by 25–30% and increases HDL-C by 1–3%.
- Australian fish, seafood and fish oil are generally very low in methylmercury, dioxins and other environmental contaminants.
- Consuming fish high in methylmercury may result in neurological damage, especially in unborn children.
- While caution should be taken in consuming fish high in methylmercury, the benefits of consuming fish and fish oil are numerous and should be encouraged in the general population.
- n-3 PUFA fortification of food and farmed fish are likely to play an increasing role in the Australian diet.

Position statement on phytosterol/stanol enriched foods[4]

The key findings in relation to phytosterol/stanol enriched foods are as follows.

- A daily intake of 2–3 g phytosterols have been shown to lower blood cholesterol levels by up to 10% depending on the age and individual metabolism of the person.
- Consuming more than 3 g per day phytosterols provides no additional benefits.

Recommendations

The Heart Foundation makes the following recommendations with respect to dietary fats and dietary sterols to improve the CVH of all Australians.

These recommendations are based on the evidence presented in *Summary of evidence. Dietary fats and dietary cholesterol for cardiovascular health*.[1] These recommendations update and supersede those developed in the 1999 position statement *A review of the relationship between dietary fat and cardiovascular disease*.[2] They also incorporate recommendations from two other Heart Foundation position papers: *Position statement. Fish, fish oils, n-3 polyunsaturated fatty acids and cardiovascular health*[3] and *Position statement on phytosterol/stanol enriched foods*.[4]

Note:
A grade of recommendation (GOR) is assigned only to recommendations derived from evidence statements with a National Health and Medical Research Council (NHMRC) level of evidence.

(2009d)—Recommendations developed from *Summary of evidence. Dietary fats and dietary cholesterol for cardiovascular health* (2009).[1]

(1999d)—Recommendations retained from *A review of the relationship between dietary fat and cardiovascular disease* (1999).[2]

(2008f)—Recommendations extracted from *Position statement. Fish, fish oils, n-3 polyunsaturated fatty acids and cardiovascular health* (2008).[3] For more details visit www.heartfoundation.org.au.

(2007p)—Recommendations extracted from *Position statement on phytosterol/stanol enriched foods* (2007).[4] For more details visit www.heartfoundation.org.au.

All Australians

1. Reduce their intake of SFA to < 7% of total energy intake (GOR A; 2009d) and tFA to < 1% of total energy intake.
2. Replace SFA with MUFA and PUFA as a strategy for reducing the intake of SFA (1999d).
3. Consume 500 mg per day of combined docosahexaenoic acid (DHA) and eicosapentaenoic acid (EPA) through a combination of the following: 2–3 serves (150 g per serve) of oily fish per week, fish oil capsules or liquid and food and drinks enriched with marine n-3 PUFA (2008f). In addition, women who are pregnant, women planning pregnancy and children should follow the advice from Food Standards Australia and New Zealand on mercury in fish (2008f).
4. Consume at least 2 g per day ALA by including foods, such as canola- or soybean-based oils and margarine spreads, seeds (especially linseeds), nuts (particularly walnuts), legumes (including soybeans), eggs and green leafy vegetables (GOR A; 2008f).
5. Consume 8–10% of total energy intake as n-6 PUFA (1999d).
6. May consume up to six eggs per week within a cardioprotective eating pattern (reduced SFA) without increasing their risk of CVD (GOR B; 2009d).

Health professionals

Health professionals should advise adult Australians to follow the above recommendations for all Australians with the following differences.

People with documented CHD

Consume **1,000 mg per day** of combined DHA and EPA through a combination of the following: 2–3 serves (150 g per serve) of oily fish per week, fish oil capsules or liquid, and food and drinks enriched with marine n-3 PUFA (2008f).

Those with elevated TG

Take fish oil capsules or liquid and marine n-3 PUFA enriched foods and drink as first-line therapy by starting with a dose of 1,200 mg per day of DHA and EPA, and if appropriate, increase the dose to 4,000 mg per day of DHA and EPA. Doctors should check their patient's response every 3–4 weeks when the dose is changed until target TG levels are reached (2008f).

Those with high absolute risk of CVD, elevated LDL-C, familial hypercholesterolaemia or type 2 diabetes

1. Consume 2–3 g of phytosterols per day from margarine, breakfast cereal, reduced fat yoghurt or reduced fat milk enriched with phytosterols (approximately 2–3 serves per day of these enriched foods) (GOR A; 2007p).* People using phytosterols should also choose at least one daily serve of fruit or vegetable high in beta-carotene (GOR A; 2007p).
2. Include phytosterol enriched foods in addition to statin therapy (GOR A; 2007p).
3. Continue compliance with cholesterol-lowering medication (2007p).

*Individuals with sitosterolaemia should restrict their intake of phytosterols. In general, children (other than those with familial hypercholesterolaemia) and lactating or pregnant women do not need phytosterol enriched foods.

Governments

1. Commit to collecting Australian population dietary intake data through a national nutrition survey conducted every five years.
2. Introduce mandatory evidence-based labelling for the inclusion of tFA on the nutrition information panel (NIP)† so that consumers can clearly identify the fat content of foods when shopping in the supermarket. Currently, insufficient evidence exists to warrant differentiation between industrially produced and naturally occurring tFA on the label.
3. Introduce mandatory evidence-based nutrition information labelling for foods eaten outside the home. With alarming statistics on the increasing number of meals eaten out, the need for such labelling is clear.
4. Support well-funded and evaluated social marketing campaigns to raise awareness about dietary fats.
5. Set targets for the food supply—from paddock to plate—to increase the proportion of foods that are grown, formulated and reformulated, eliminate the use of industrially produced tFA and reduce the use of both tFA and SFA.
6. Recommend that Australians consume fish and fish oil.
7. Include fish oil capsules and liquid in the Pharmaceutical Benefits Scheme to assist health professionals in prescribing them to those who have CHD.
8. Support professional development for doctors, which includes advice on:
   - fish and fish oil consumption for those with and at risk of CHD
   - reducing the intake of SFA and tFA.
9. Support sustainable fishing practices and healthy, sustainable marine ecosystems.
10. Monitor levels of methylmercury and dioxins in Australian fish.

The food industry

The Heart Foundation encourages the food industry to make the following changes to the food supply to improve the CVH of the Australian population.

1. Reformulate foods to reduce SFA and tFA by replacing with *cis*-MUFA or *cis*-PUFA.
2. Commit to the development of ingredients with lower SFA and tFA profiles by:
   - replacing partially hydrogenated or hydrogenated oils with oils high in *cis*-MUFA or *cis*-PUFA
   - using ingredients and products with negligible tFA, but not at the expense of significantly increasing SFA
   - adapting new technologies to produce products with negligible tFA.
3. Develop a variety of foods that are enriched with marine n-3 PUFA.
4. Promote fish and seafood that come from sustainable sources.
5. Retain marine oils where possible during the processing of fish and seafood.
6. Work with the Heart Foundation Tick to achieve high nutritional standards in the retail and food service areas.
7. Introduce an evidence-based nutrition information panel for all foods eaten outside the home.

† The minimum information required on the NIP is regulated by Food Standards Australia New Zealand in the Food Standards Code. The Heart Foundation supports evidence-based labelling where it relates to allergens and health claims.

Rationale

CVD refers to all diseases and conditions of the heart and blood vessels. The main types include coronary (or *ischaemic*) heart disease, stroke, heart failure, peripheral vascular disease, rheumatic heart disease and congenital heart diseases. CHD is the most prevalent and serious form of CVD. CHD is manifested as angina, myocardial infarction and sudden death.

There are several accepted modifiable risk factors for CVD, including hypertension, elevated serum cholesterol, obesity, poor nutrition, physical inactivity, tobacco smoking, alcohol consumption and excessive stress. Other risk factors include age, gender, raised TG, raised homocysteine and lipoprotein a (Lp(a)).

***Summary of evidence. Dietary fats and dietary cholesterol for cardiovascular health*[1] and *A review of the relationship between dietary fat and cardiovascular disease*[2]**

To ensure that the Heart Foundation recommendations were based on the best available evidence, a summary of evidence sought to collect and analyse the highest quality international studies, reviews and reports relating to dietary fats, dietary cholesterol, CVH and CVD. The findings and the levels of evidence are summarised in the table on page 7.

***Position statement. Fish, fish oils, n-3 polyunsaturated fatty acids and cardiovascular health*[3]**

Fish, fish oils and n-3 PUFA have been associated with a reduced risk of CVD.[3,5]

New findings published in Australia and internationally regarding the benefits and cautions of consuming n-3 PUFA were consolidated from the extensive literature on fish, fish oils and n-3 PUFA, and explore the international recommendations regarding their CVH benefits. The findings from that review and the levels of evidence are summarised in the table on page 8.

***Position statement on phytosterol/stanol enriched foods*[4]**

Studies indicate that incorporating phytosterol/stanols into the diet may be an effective method of lowering total and LDL-C levels. The Heart Foundation reviewed the literature around phytosterol/stanols and updated the 2003 position statement[6] to examine the cholesterol-lowering effect of phytosterol/stanol enriched foods. The findings and the levels of evidence are summarised in the table on page 9.

Summary of evidence

NHMRC levels of evidence were attributed to the recommendations where appropriate, and the associated studies were listed. Where a level of evidence was not attributed, the evidence was obtained from documents and reports of recognised national and international authorities, if the paper had not been subject to peer review. Where there was insufficient, inconclusive or conflicting evidence, the level of evidence was considered not applicable (n/a). Little/moderate/good were the levels of evidence used in our previous position statement on dietary fat (1999).[2]

Evidence on dietary fats and dietary cholesterol[1,2]	Level of evidence
SFA	
SFA intake is associated with CHD[7]	III–2
Replacing SFA with *cis*-unsaturated fatty acids has a greater positive influence on CHD risk than replacing SFA with carbohydrates (CHO)[8,9]	I
Replacing SFA with n-6 PUFA to achieve a ratio of PUFA to SFA of greater than 1 will reduce the risk of CHD (1999 evidence statement retained)[2]	Good
Replacing SFA with MUFA lowers TC and LDL-C, although not to the same extent as PUFA (1999 evidence statement retained)[2]	Good
Increasing SFA intake results in an increase in TC and LDL-C compared to CHO, PUFA and MUFA (1999 evidence statement retained)[2]	Good
Myristic, palmitic acids and lauric acids are associated with elevated LDL-C[9,10]	I
Stearic acid has a negligible effect on LDL-C[9,10]	I
Lauric, myristic and palmitic acids are associated with an increase in HDL-C compared to CHO. Lauric acid increase of HDL-C is greater than other individual SFA[9,10]	I
Lowering dietary SFA to < 7% of energy intake with restricted dietary cholesterol results in further LDL-C lowering than diets containing < 10% of energy intake from SFA[11]	II
There is little evidence that an increase in the consumption of SFA increases the incidence of stroke (1999 evidence statement retained)[2]	Little
There is little evidence that an increase in the consumption of SFA affects the susceptibility to thrombosis and arrhythmia or blood pressure (1999 evidence statement retained)[2]	Little
tFA	
tFA intake increases LDL-C, decreases HDL-C, increases Lp(a) and increases the fasting TG concentration[7,12-14]	II
The use of fatty acids in tissue as a marker of tFA has yielded conflicting results[7]	n/a
tFA intake is associated with increased CHD incidence and the risk of CHD[7,8,13,15,16]	III–2
tFA intake is associated with an increased risk of non-fatal and fatal MI[7,16,17]	III–2
tFA intake has little effect on haemostatic factors, susceptibility of LDL-C to oxidation, or blood pressure, but may have an adverse effect on postprandial lipids and endothelial function[13,18]	II
Ruminant tFA have a similar effect on LDL-C and HDL-C as industrially produced tFA [17,19-21]	II
Replacing partially hydrogenated oils with oils high in *cis*-unsaturated fatty acids improves the lipid profile[9,22,23]	I
PUFA	
n-6 PUFA intake lowers LDL-C[24]	II
It is unclear whether lowering the n6:n3 ratio is beneficial for CVH[25-28]	n/a
The CVH benefits of n-3 PUFA are not influenced by background n-6 PUFA[29,30]	III–2
There is little evidence that an increase in the consumption of n-6 PUFA affects the susceptibility to thrombosis and arrhythmia or blood pressure (1999 evidence statement retained)[2]	Little

Evidence on dietary fats and dietary cholesterol[1,2] (cont.)	Level of evidence
MUFA	
There is little evidence that MUFA has an independent effect on coronary endpoints (1999 evidence statement retained)[2]	Little
There is little evidence that an increase in the consumption of MUFA affects the susceptibility to thrombosis and arrhythmia or blood pressure (1999 evidence statement retained)[2]	Little
Total fat	
There is no direct relationship between total fat intake and the incidence of CHD[8]	n/a
Dietary cholesterol	
There is inconclusive evidence supporting a relationship between dietary cholesterol and CVD outcomes[24]	n/a
There is little evidence of a relationship between serum cholesterol and stroke[31]	III–2
Dietary cholesterol increases TC and LDL-C, but substantially less so than SFA and tFA (1999 evidence statement retained)[2]	Moderate
HDL-C and TC:HDL-C	
Higher HDL-C may reduce the risk of CHD[31]	III–2
TC:HDL-C is an informative predictor of CHD mortality[32]	III–2
Specific foods	
Consuming up to six eggs per week in a diet low in SFA is not associated with adverse CVD outcomes. Egg consumption > 6 per week may increase CHD risk in people with diabetes.[33-40]	III–2
Consuming nuts improves the lipid profile[41,42]	I
Dietary modifications	
Dietary changes longer than two years can reduce cardiovascular events and total mortality among high-risk patients[43]	I

Evidence on fish, fish oils and n-3 PUFA[3]	Level of evidence
Individuals with a higher intake of fish have a lower risk of CHD mortality, total CHD and total stroke[44-46]	III–2
Consuming fish at least once a week is associated with a lower risk of total stroke and CHD mortality in the general population and in post-MI patients[44-48]	III–2
In secondary prevention, a diet with 2 g per day ALA decreases the risk of CHD[49-51]	II
In secondary prevention, ≥ 850 mg per day marine n-3 PUFA supplementation reduces the risk of CHD mortality and ≥ 1,800 mg per day reduces major coronary events[52-54]	II
In secondary prevention, there is conflicting evidence about the effects of marine n-3 PUFA supplementation on the risk of sudden death in patients[52,55-59]	II
Marine n-3 PUFA supplementation of 1,000–4,000 mg per day decreases serum TG levels by 25–30% and increases HDL-C levels by 1–3%. A dose relationship exists between the intake of marine n-3 PUFA and decreased serum TG levels[60-62]	I
Marine n-3 PUFA has an additive effect to statin therapy in decreasing serum TG levels and increasing HDL cholesterol[63-69]	II

Evidence on fish, fish oils and n-3 PUFA[3] (cont.)	Level of evidence
Consuming fish with high levels of methylmercury may result in long-term neurological damage. Gestational exposure to methylmercury may result in neurodevelopmental deficits[70]	III–3
The consumption of oily fish twice a week promotes cardiovascular health without excessive exposure to mercury[71,72]	III–1
There is inconclusive evidence to support a relationship between mercury exposure and the incidence of CVD[73]	n/a
Fish oil capsules available in Australia have no or close to no methylmercury content[74]	IV
Fish oil capsules in Australia contain very low levels of dioxins (polychlorinated biphenyl)[75]	IV

Evidence on phytosterol/stanol enriched foods[4]	Level of evidence
Phytosterols lower LDL-C in normocholesterolaemic, hypercholesterolaemic and diabetic individuals[76]	I
For people with an increased risk of CVD, consuming phytosterol/stanol enriched foods provides an additional option for risk reduction through lowering the level of cholesterol[77,78]	II
A daily intake of approximately 2 g phytosterol/stanol from enriched margarine reduces LDL-C levels by approximately 10%, but has little effect on HDL-C or TG[79,80]	I
A daily intake of approximately 2.5 g phytosterols from enriched low fat breakfast cereal, yoghurt, milk or bread reduces LDL-C levels by approximately 5–15%[81-83]	II
Consuming phytosterol/stanol enriched foods at levels higher than 2–3 g per day provides no additional benefits to lowering LDL-C[84,85]	I
Daily consumption frequency does not influence the cholesterol-lowering efficacy of phytosterol/stanols[79,86,87]	II
Phytosterol/stanol enriched foods have an additive effect in lowering LDL-C when combined with statins[88-94]	II
There are no reported adverse effects from the daily consumption of phytosterol/stanol enriched foods, although long-term safety information is not available[84,95-104]	II
When consuming phytosterol/stanol enriched foods, blood carotenoids are reduced. An additional serving of high-carotenoid fruit and vegetables is effective in maintaining blood carotenoid concentrations[80,100,102,105-108]	II
Individuals with the rare inherited metabolic disease, homozygous sitosterolaemia, have high blood phytosterol levels and premature atherosclerosis. A restricted intake of phytosterols is recommended for these individuals[109]	II

Australian recommendations

In 2006, the NHMRC[26] reviewed the evidence and released their revised Nutrient Reference Values, which included recommendations for dietary fats and dietary cholesterol for all Australians by life stage and gender to reduce chronic disease outcomes.

To reduce chronic disease risk, the NHMRC recommended that:

- total fat intake be 20–35% of energy
- SFA and tFA together be no more than 10% of energy
- the range for linoleic acid (n-6 PUFA) equates to 4–10% of dietary energy
- the range for ALA (n-3) equates to 0.4–1% of dietary energy
- the suggested dietary target for combined DHA, EPA and docosapentaenoic acid (DPA) is 610 mg per day for men and 430 mg per day for women.

An upper limit for children, adolescents and adults was set at 3,000 mg per day for combined DHA, EPA and DPA.

Adequate intake values were set as follows:

- ALA: 1.3 g per day for men and 0.8 g per day for women
- combined DHA, EPA and DPA: 160 mg per day for men and 90 mg per day for women.

For children, adolescents and adults, no estimated average requirement, recommended dietary intake or adequate intake was set for total fat, as the type of fat was considered more important for physiological and health outcomes.

No recommendation for dietary cholesterol was included because the NHMRC position states that a reduction in SFA will bring with it lower cholesterol intakes as the two usually occur in the same foods. In addition, the effect on LDL-C from dietary cholesterol was considered less consistent than that of SFA.

Rating of the evidence for recommendations

Evidence is graded according to the NHMRC.[110]

Level of evidence	Study design
I	Evidence obtained from a systematic review of all relevant randomised controlled trials
II	Evidence obtained from at least one properly designed randomised controlled trial
III–1	Evidence obtained from well-designed pseudo-randomised controlled trials (alternate allocation or some other method)
III–2	Evidence obtained from comparative studies with concurrent controls and non-randomised allocation, cohort studies, case-control studies or interrupted time series with a control group
III–3	Evidence obtained from comparative studies with historical control, two or more single-arm studies or interrupted time series without a parallel control group
IV	Evidence obtained from case series, either post-test or pre-test and post-test

GOR	Description
A	Rich body of high quality randomised controlled trial evidence
B	Limited body of randomised controlled trial data or high quality non-randomised controlled trial data
C	Limited evidence
D	No evidence available—panel consensus judgement

For heart health information
1300 36 27 87
www.heartfoundation.org.au

Visit www.heartfoundation.org.au/Professional_Information/Lifestyle_Risk/Nutrition for:

- the full reference list for this position statement
- Q&As about dietary fats and dietary sterols
- *Summary of evidence. Dietary fats and dietary cholesterol for cardiovascular health*
- *Review of the evidence. Fish, fish oil, n-3 polyunsaturated fatty acids and cardiovascular health*
- *Position statement on phytosterol/stanol enriched foods.*

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