EVIDENCE-BASED
BEST PRACTICE
GUIDELINE

THE ASSESSMENT AND MANAGEMENT OF CARDIOVASCULAR RISK

Heart Foundation
The Heart of Our Nation

STROKE FOUNDATION
OF NEW ZEALAND INC

MINISTRY OF HEALTH
MANATŪ HAUORA

NEW ZEALAND GUIDELINES GROUP

DECEMBER 2003

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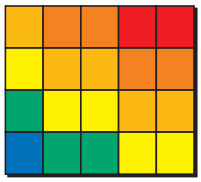
Best Practice Evidence-based Guideline

THE ASSESSMENT AND MANAGEMENT OF CARDIOVASCULAR RISK

Ruia taitea
Kia tū ko taikākā anake

(Strip away the sapwood
Gather only the heartwood)



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STATEMENT OF INTENT

Evidence-based best practice guidelines are produced to help health practitioners, patients and consumers make shared decisions about health care choices in specific clinical circumstances. If properly developed, communicated and implemented, guidelines can improve care. While they represent a statement of best practice based on the latest available evidence (at the time of publishing), they are not intended to replace the health practitioner's judgment in each individual case.

Care decisions should consider the following:

- the individual's clinical state, age and co-morbidities
- personal preferences and preferences of family/whānau
- current best practice based on the latest available research evidence.

The clinical expertise (including skills and experience) of the practitioner is the key to integrating these elements to achieve the best possible outcome for an individual.

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PURPOSE

The purpose of this guideline is to provide an evidence-based summary of effective practice in the assessment and management of cardiovascular risk. It seeks to assist informed decision-making by adults with and without known cardiovascular disease, their families, whānau and their health care providers, with the ultimate goal of improving the cardiovascular health of New Zealanders.

This guideline is intended for use principally by primary care practitioners involved in the clinical management of the major modifiable cardiovascular risk factors, including general practitioners, nurse practitioners, practice nurses and dietitians. It may also be valuable to other health practitioners involved in the care of people with cardiovascular disease or diabetes in the community, such as pharmacists, as well as to health practitioners working in the secondary and tertiary care sectors.

Further resources are being developed for people at risk of cardiovascular disease, diabetes and stroke and for their families and whanāu. It is hoped that the information in these guidelines will be used to inform people at risk and their advisers about the benefits of understanding and reducing their risk of disease, and that this will ultimately lead to improved health outcomes for all New Zealanders.

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ABOUT THE GUIDELINE

The previous New Zealand guidelines on the management of dyslipidaemia and mild hypertension were written in 1994 and 1996 respectively. Earlier New Zealand guidelines for the management of diabetes comprised a set of four guidelines on glycaemic control, retinal screening, microalbuminuria screening and foot screening, and were last published in 2000. These did not give guidance on the management of cardiovascular risk in people with diabetes. The current guideline integrates advice for the management of all cardiovascular risk factors in the prevention of coronary and cerebrovascular disease and has a separate section on the management of cardiovascular risk in people with diabetes. It has taken into account new evidence published since these previous guidelines.

This guideline covers the assessment and management of people with known cardiovascular disease or who are at risk of developing cardiovascular disease, including people with type 1 and type 2 diabetes. The cardiovascular diseases included in this guideline are angina, myocardial infarction, coronary death, ischaemic stroke, transient ischaemic attack and peripheral vascular disease.

Advice on the detailed management of diabetes or the other non-cardiovascular (microvascular) complications of diabetes is not included. Also excluded are the specific management of people with genetic lipid disorders, heart failure, acute coronary syndromes, sleep apnoea, and cardiovascular disease in children. Best practice in diabetes care, cardiac rehabilitation and acute stroke management have been covered in other evidence-based New Zealand guidelines (available at www.nzgg.org.nz).

Cardiovascular risk assessment should be considered as a screening process. The National Health Committee,[1] has identified important ethical considerations when undertaking screening. These are as applicable to screening undertaken opportunistically as they are to screening programmes.

There is no randomized control trial evidence to support universal screening by cardiovascular risk assessment in population groups. However, there is a body of evidence that supports identifying people at high risk of cardiovascular disease and managing them accordingly. The recommendations in these guidelines are based on this evidence. If practitioners are to offer screening opportunistically to people in the groups identified in the guidelines, they should also ensure that there is a process for auditing this practice to ensure that it is safe and effective, and for assessing outcomes. There are a range of tools available in primary care that are suitable for this. The process is made easier by the use of electronic patient management systems. Practitioners should be aware of the need to focus on population groups that have a high burden of cardiovascular disease. These groups should be specifically targeted to ensure that they are able to benefit from risk screening and subsequent management. Practice audit should include examining the extent to which these groups are participating.

GAPS IN CURRENT PRACTICE

The potential for improvement in the management of cardiovascular disease in New Zealand has been identified by the National Cardiovascular Advisory Group. Cardiovascular disease is a priority area of the *New Zealand Health Strategy*[2] and this document, along with *He Korowai Oranga: Māori Health Strategy*[3] set the strategic direction for the health sector, emphasising the importance of removing socioeconomic and ethnic inequalities in health. Māori have the poorest health status of any group in New Zealand and improving Māori health is therefore a priority for action to address health inequalities in New Zealand. The promotion of best practice through the development of this guideline is one of four activities identified as critical to improving cardiovascular health and reducing inequalities. The other components are:

- service development and integration
- improving consumer knowledge and education
- addressing workforce issues.

While there has been a reduction in age-standardised cardiovascular mortality across all socioeconomic groups in the last two decades, this decline has been greatest among those of higher socioeconomic position. As a result, socioeconomic inequalities in cardiovascular disease have widened, with cardiovascular disease increasingly associated with disadvantage.[4,5] Age-standardised death rates for coronary heart disease since 1960 have fallen at a slower rate for Māori than for non-Māori.[6]

A clear association between socioeconomic position and cardiovascular disease and all-cause mortality has been demonstrated overseas and in New Zealand.[5,7,8] However, the widening gap for Māori cannot be explained solely by occupational class.[9,10] The development of the NZDep small area score has permitted the measurement of socioeconomic position, that in association with other small area census variables allows the assessment of ethnicity by socioeconomic gradient. A number of models provide insight into the nature of ethnic health disparities. In New Zealand, the gap framework uses the NZDep to interpret the effects of socioeconomic deprivation on health outcomes.[11] It identifies three gaps:

1. *The distribution gap* – over half of Māori are in the three most deprived deciles in New Zealand. This means Māori are exposed to a higher incidence and prevalence of cardiovascular diseases and cardiovascular risk factors.
2. *The outcome gap* – across deprivation deciles, average Māori life expectancy at birth is significantly lower than that of non-Māori. Notably, Māori in the least deprived deciles have a lower life expectancy than non-Māori in the most deprived deciles.
3. *The gradient gap* – as deprivation increases, the relative difference in outcomes (eg, mortality) between Māori and non-Māori widens.[11,12]

This guideline focuses on the evidence base for treatment and lifestyle advice to be given equitably to all New Zealanders. The priority for the implementation phase of this project will be to ensure equitable access to services that can produce health gains for Māori. An implementation plan is presented in Chapter 16, *Implementation*, with provisional performance indicators included in Appendix A. Measurement of the current status of cardiovascular health and any progress towards meeting guideline goals are important elements to the overall strategy. An action plan for improving the cardiovascular health of Māori has been presented by the Māori Cardiovascular Advisory Group and will be published separately.

Risk Factor Profiles and Prescribing

Data on risk factor profiles and current clinical practice in New Zealand come from the National Nutrition Survey,[14] from primary care/IPA databases, and from prescribing data collected by PHARMAC.

In New Zealand, the 'Get Checked' programme collects data on risk factors for people with diabetes at an individual level. These data are collated by Local Diabetes Teams (LDTs) and reported to the Ministry of Health.

There are important differences in the prevalence of risk factors among different groups within New Zealand society. For example, Māori men and women smoke more than non-Māori men and women at all ages and at all levels of deprivation.[15]

Overseas data suggest that the size of the gap between best evidence and current practice is likely to be fairly large. In an Australian study of people with known coronary heart disease in 1999/2000, 54.0% (95% CI, 49 – 59%) of people with known coronary heart disease did not achieve a total cholesterol of less than 4.5 mmol/L, and 39.5% did not achieve blood pressure levels of less than 140/90 mm Hg.[16]

New Zealand studies have also shown sub-optimal management of lipids.[17] In New Zealand the prescribing of statins has improved since the cost of these agents has reduced and restrictions on access were lifted. PHARMAC data in April 2001 show that of an estimated 175,000 people eligible for statins under the old Special Authority criteria, less than 40% (67,000) were being dispensed statins.[18] The age-standardised dispensing rate of statins for those eligible in the lowest-ranked District Health Board (DHB) was 61% of that in the highest-ranked DHB.

While it is difficult to estimate with accuracy the size of the gap between evidence and practice, the judgment of how much effort will be required to close the gap and the likelihood that the proposed changes can be implemented has been made by the National Cardiovascular Advisory Group.

The DHB toolkits provide a summary of the government's strategy to address cardiovascular disease. They can be viewed at www.newhealth.govt.nz/toolkits/cardiovascular.htm

Implementation issues and the suggested information that can be collected to measure changes in practice and outcome are contained in Chapter 16, *Implementation*, and Appendix A.

GUIDELINE DEVELOPMENT PROCESS

The Guideline Development Team took a pragmatic approach to the development of this guideline. Several evidence-based guidelines were available internationally and all had been rigorously and systematically developed. A process for adapting overseas guidelines was agreed. The quality of the international guidelines was to be assessed, and relevant sections of these international guidelines reviewed. Where issues were identified that were not covered by previous guidelines, new searches were either commissioned from the New Zealand Health Technology Assessment (NZHTA), or were performed by the New Zealand Guidelines Group (NZGG).

The Guideline Development Team was convened by the NZGG as a partnership between the NZGG, the National Heart Foundation of New Zealand (NHF), the Stroke Foundation of New Zealand and the Ministry of Health. Members were nominated by a wide variety of stakeholder organisations. The Guideline Development Team first met in March 2002, and had five face-to-face meetings and several subgroup teleconferences. The chairperson of the team was responsible for chairing the guideline meetings and teleconferences. The project manager was responsible for the day-to-day organisational arrangements for the team, and for writing drafts of the guideline, with content as agreed initially by subgroups and then by the whole team.

Two members of the Guideline Development Team for the New Zealand Guideline for the Management of Diabetes (a diabetologist and a cardiologist) worked with the Guideline Development Team for the *Assessment and Management of Cardiovascular Risk* on issues of overlap, with each team reviewing the drafts produced by the other. Further information about the New Zealand Guideline, *Management of Type 2 Diabetes* is available at www.nzgg.org.nz

NZGG approached the Scottish Intercollegiate Guidelines Network (SIGN) for permission to adapt SIGN guidelines to New Zealand circumstances, including changes to recommendations where the guidelines development team considered these necessary. The specific guidelines that were adapted were the *Hypertension in Older People* (SIGN 49), *The Management of Diabetes* (SIGN 55), and *Secondary Prevention of Coronary Heart Disease following Myocardial Infarction* (SIGN 41). Permission was generously granted. The developers of the other international guidelines were also approached for permission to adapt their guidelines as necessary.

The steps in the development of this evidence-based adapted guideline are listed below:

1 The group met and agreed on the key issues or questions to be addressed by the guideline (see NZGG's website at www.nzgg.org.nz – click on *Supporting Materials* for this guideline).

2 NZHTA provided information packs containing the international guidelines to date.

3 The international guidelines were appraised for the quality of their methodologies, using the AGREE appraisal instrument.[19] All the guidelines listed in the evidence tables were assessed as being well developed, and suitable for adaptation to New Zealand circumstances. The topic areas common to the proposed cardiovascular guideline and other existing international lipid, blood pressure and diabetes guidelines were identified, reviewed and the recommendations compared (available at www.nzgg.org.nz).

4 The subgroups drafted chapters and an algorithm which further defined the clinical questions. Agreement was reached on which questions required a further literature search.

5 New literature searches were completed in three ways:

   i evidence tables were commissioned from NZHTA. Searches were undertaken, the studies were appraised using the Generic Appraisal Tool for Epidemiology (GATE) and the results were presented in evidence tables (see NZGG's website at www.nzgg.org.nz click on *Supporting Materials* for this guideline)

   ii searches by the project manager identified other papers and literature published since 2001 and these were appraised independently. The cut-off for new evidence for systematic searches was February 2003

   iii systematic reviews and meta-analyses published since 1999 were presented for review by the Guideline Development Team.

6 These reviews, summary evidence tables and tables returned by NZHTA were reviewed by the Guideline Development Team and a considered judgment process was used to agree levels of evidence and draft recommendations.

7 The statements and recommendations were drafted, reviewed and revised by sub-groups and then by the whole team. Resources and appendices were drafted in the same way. This process continued until the draft document was at an appropriate stage for peer review and consultation.

8 The dietary sections were authored separately using evidence prepared in tables and the draft reviewed by the Dietary Interventions Subgroup and others from the main Guideline Development Team.

9 Copies of the draft were sent out to key individuals and sector groups for comment and peer review.

10 These comments were approved by the team and incorporated into the final document and supporting resources.

11 The final guideline was sent to sector groups for endorsement.

GUIDELINE DEVELOPMENT TEAM

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We would like to thank the following for their contributions to the development of the chapter, *Cardiovascular Health of Māori:* **Dr Dale Bramley, Dr Sue Crengle, Dr Elana Curtis, Dr Matire Harwood, Deirdre Nehua** and **Dr Papaarangi Reid**.

We would like to thank the following for their comments on the chapter, *Cardiovascular Health of Pacific People*: **Dr Debbie Ryan, Dr Justine Mesui** and **Professor Sitaleki Finau**.

Recognition is given to the following for their contributions and support; **Dr Cathy Pikholz, Dr Kirsten Coppell, Anne Blumgart, Leonie Brunt, Anne Buckley, Carole Webb, Stephanie Dixon**, **Melanie McKissack, Naomi Brewer** and **Paula Bell** from the NZGG.

Special thanks to the staff of the NZHTA for their contributions and skill in searching and appraising the literature. Special thanks to **Dr Natasha Rafter**, Public Health Registrar, EPIQ, School of Population Health, University of Auckland and **Dr Victoria Andersen**, Department of General Practice and Primary Health Care, University of Auckland for their critical appraisals, and to **Pip Furness** (medical student) for the retrieval of papers.

Declaration of Potential Competing Interests

Professor Jim Mann has received financial support towards conference travel and research projects from the following pharmaceutical companies:

- Merck Sharp and Dohme
- Roche
- Novo Nordisk
- Novartis
- Bristol Myers Squibb/Mead Johnson.

He is a participant in the Navigator Study of Nateglinide in the prevention of type 2 diabetes but none of the other support has been for trials of drugs. He has not been a consultant to any pharmaceutical company nor received any personal remuneration from a pharmaceutical company.

Associate Professor Bruce Arroll is on the primary care committee for the future forum funded by AstraZeneca and has received financial support from the New Zealand office of Eli Lilly.

Professor Rod Jackson has received research and development funding from:

- Health Research Council of New Zealand
- National Heart Foundation of New Zealand
- ACC
- Alcohol Advisory Council of New Zealand
- Ministry of Health
- ProCare
- South Auckland Health.

Professor Jackson has been paid for teaching sessions, received fees or travel support from:

- PHARMAC
- ADIS Press
- Royal Australasian College of Surgeons
- Aviation Medical Society of Australia and New Zealand
- Civil Aviation Authority
- American Society of Hypertension
- World Heart Federation
- Merck Sharp and Dohme sponsored 'State of the Nation's Health Forum'.

Dr Stewart Mann has received research funding, travel support or has acted as a consultant for the New Zealand offices of the following pharmaceutical companies:

- Roche
- AstraZeneca
- GlaxoSmithKline.

Dr Diana North has acted as a consultant for the Roche pharmaceutical company.

Professor Russell Scott has received research funding, travel support or has acted as a consultant for the New Zealand and international offices of the following pharmaceutical companies:

- Merck Sharp and Dohme
- Roche
- GSK Diabetes
- Abbott
- Eli Lilly.

Professor Harvey White has received research funding, travel support or has acted as a consultant for the following pharmaceutical companies:

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- Aventis
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- Merck Sharp and Dohme
- AstraZeneca
- Wyeth Ayerst
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The remaining members of the Guideline Development Team did not report any competing interests.

CONSULTATION AND PEER REVIEW

The draft of this guideline was widely distributed to many organisations including consumer groups, primary health organisations, DHBs, service and provider organisations, expert reviewers, clinicians, and other health care practitioners for comment as part of the consultation and peer review process. Thanks and acknowledgement to Dr Peter Moodie, Dr Scott Metcalf, Tracey Barron, Sean Dougherty and Hew Norris from PHARMAC who have reviewed and commented on some drafts. At the time of peer review, the draft guideline was also available on the NZGG website.

Comment was received from many individuals, groups and organisations including consumers, health care practitioners and academics. The following individuals assessed the guideline using the AGREE tool:

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Comments were considered by a subgroup of the Guideline Development Team and the NZGG, and amendments made.

FUNDING

The guideline was funded by the Ministry of Health and developed by the Guideline Development Team under the stewardship of the NZGG, the National Heart Foundation of New Zealand, and the Stroke Foundation of New Zealand. The NZGG is an independent organisation and not involved in service provision.

EVIDENCE AND RECOMMENDATION GRADING SYSTEM

The levels of evidence, adapted from SIGN, and the NZGG grades of recommendation, are as follows:

NZGG LEVELS OF EVIDENCE	
1++	High quality meta-analyses, systematic reviews of RCTs, or RCTs with a very low risk of bias
1+	Well conducted meta-analyses, systematic reviews of RCTs, or RCTs with a low risk of bias
1-	Meta-analyses, systematic reviews of RCTs, or RCTs with a high risk of bias
2E	An economic evaluation that has used local data (in this case from New Zealand) with level 1 evidence on effectiveness of interventions from well conducted meta-analyses or RCTs
2++	High quality systematic reviews of case-control or cohort studies High quality case-control or cohort studies with a very low risk of confounding or bias and a high probability that the relationship is causal
2+	Well-conducted case-control or cohort studies with a low risk of confounding or bias and a moderate probability that the relationship is causal
2-	Case-control or cohort studies with a high risk of confounding or bias and a significant risk that the relationship is not causal
3	Non-analytic studies eg, case reports, case series
4	Expert opinion

The NZGG grading system is based on the SIGN grading system. More information can be found at www.sign.ac.uk

In this guideline the level of evidence is included alongside the references. This is formatted as reference (level of evidence), for example,[95](**2+**).

NZGG GRADES OF RECOMMENDATIONS	
The recommendation is supported by **good evidence**	**A**
The recommendation is supported by **fair evidence**	**B**
The recommendation is supported by **non-analytic studies or consistent expert opinion**	**C**
The evidence is insufficient, evidence is lacking, of poor quality or opinions conflicting, the balance of benefits and harms cannot be determined	**I**
Recommended practice based on the clinical experience of the Guideline Development Team	✓

Studies with a high risk of bias (**1-** or **2-**) were excluded from consideration in forming recommendations. Studies that were assessed using the GATE critical appraisal tool (further information regarding this tool at the EPIQ website, www.health.auckland.ac.nz/population-health/epidemiology-biostats/epiq/index.html) or studies that had been critically appraised by other guideline groups using other systems were scored for validity using the SIGN framework. Additional scores for applicability or for the precision of results were considered by the Guideline Development Team in forming the recommendations.

SUMMARY

KEY MESSAGES

- Assessment of absolute cardiovascular risk is the starting point for all discussions with people who have cardiovascular risk factors measured. Reduction in cardiovascular risk is the goal of treatment.
- Risk assessment for most asymptomatic men is recommended from the age of 45 (or from the age of 35 if they have risk factors). Risk assessment for most asymptomatic women is recommended from the age of 55 (or from the age of 45 if they have risk factors).
- Māori should be assessed for cardiovascular risk 10 years earlier than non-Māori. There is an urgent need to focus intervention programmes on Māori, who bear the greatest burden of cardiovascular disease in New Zealand. The 'outcome gap' between Māori and non-Māori is widening.
- A fasting lipid profile, fasting plasma glucose and two blood pressure measurements are recommended investigations for comprehensive risk assessment.
- People with known cardiovascular disease and those at high risk because of diabetes with renal disease, or some genetic lipid disorders, are clinically defined at very high risk.
- Cardiovascular mortality is high in people with impaired glucose tolerance (IGT) or diabetes and most will require intensive intervention. Particular attention is required for Māori who have a high rate of cardiovascular and renal complications from diabetes.
- Lifestyle change and drug intervention should be considered together. The intensity of intervention recommended depends on the level of cardiovascular risk:
  - a life free from cigarette smoke, eating a heart healthy diet and taking every opportunity to be physically active is recommended for people at less than 10% 5-year CV risk
  - lifestyle interventions for people at more than 10% 5-year CV risk are strongly recommended and this group should receive individualised advice using motivational interviewing techniques relating to smoking cessation if relevant, a cardioprotective diet and regular physical activity
  - cardiovascular risk should be reduced in people at greater than 15% 5-year CV risk by lifestyle interventions, aspirin, blood pressure lowering medication and lipid modifying therapy (statins). There should be a greater intensity of treatment for higher risk people (more than 20 – 30%)
  - after myocardial infarction, comprehensive programmes that promote lifestyle change for people are best delivered by a cardiac rehabilitation team. Most people with angina or after myocardial infarction will be taking at least four standard drugs, low-dose aspirin (75 – 150 mg), a beta-blocker, a statin and an ACE-inhibitor
  - virtually all ischaemic stroke and transient ischaemic attack survivors should be taking low dose aspirin, a combination of two blood pressure drugs and a statin.

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EFFECTIVE ASSESSMENT AND MANAGEMENT OF CARDIOVASCULAR RISK

The Burden of Cardiovascular Disease

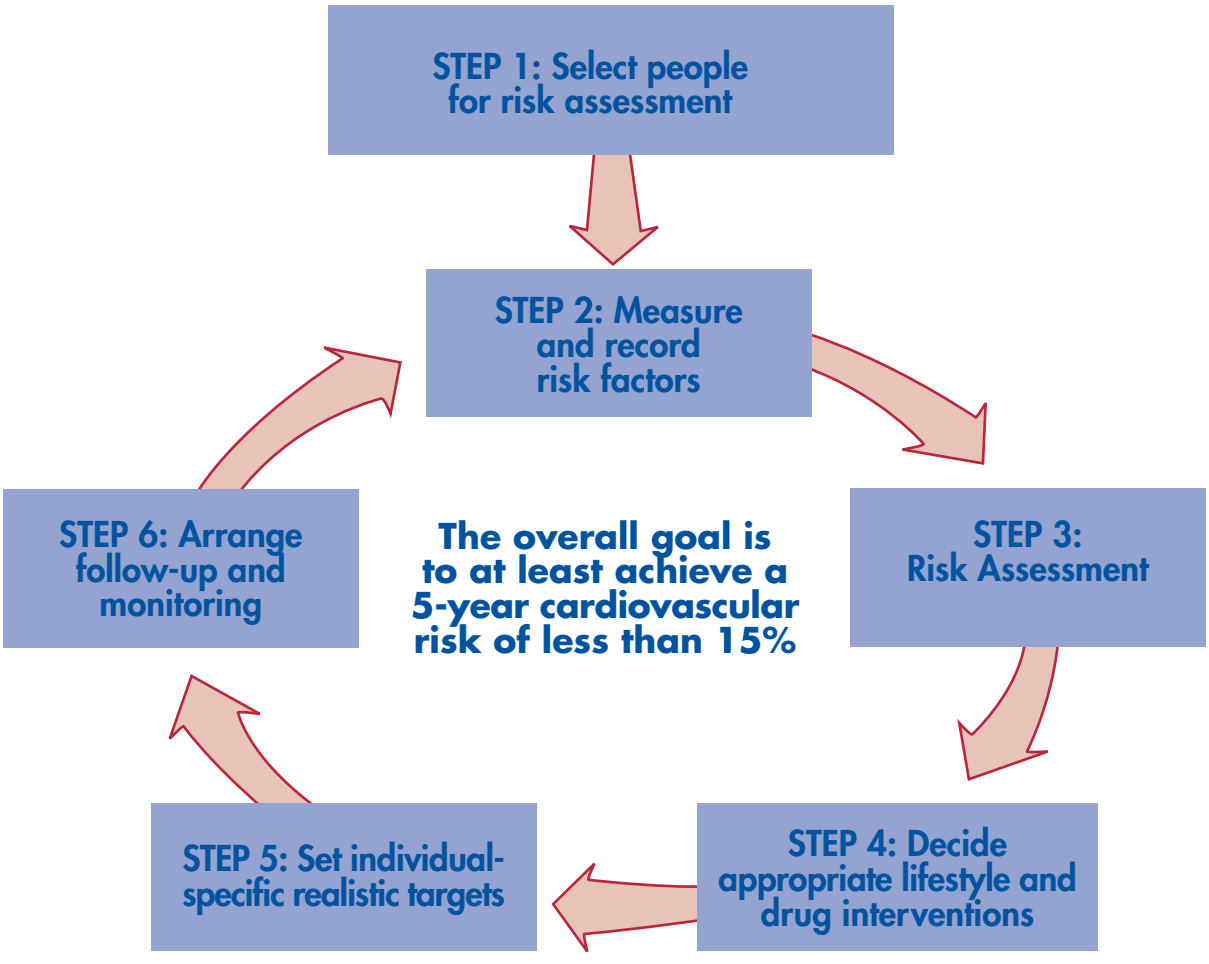
Cardiovascular disease is the leading cause of death in New Zealand, accounting for 40% of all deaths. While age-standardised mortality has halved over the past 30 years, the total number of deaths from cardiovascular disease has changed little because of the growing number of older people and at-risk individuals. The burden of cardiovascular disease falls disproportionately on Māori and also lower socioeconomic groups at a younger age. Cardiovascular disease can be reduced through lifestyle change and appropriate drug therapy.

Absolute Cardiovascular Risk

Treatment decisions are based on the likelihood an individual will have a cardiovascular event over a given period of time. This replaces decision-making based on individual risk factor levels. By knowing the risk level an individual and their practitioner can make decisions for prevention and treatment of cardiovascular disease, including lifestyle advice, diabetes care, the prescription of lipid-modifiying and blood pressure lowering medication and/or medication after myocardial infarction or ischaemic stroke.

The following steps outlined in Figure 1 explain the actions taken at each stage.

Figure 1: Steps in the assessment of cardiovascular risk



STEP 1: Select People for Risk Assessment

People with diabetes should have risk assessments from the time of diagnosis.

Table 1: Recommended age levels for initiating cardiovascular risk assessment

	Men	Women
Māori, Pacific peoples and people from the Indian subcontinent	Age 35 years	Age 45 years
People with known cardiovascular risk factors or at high risk of developing diabetes	Age 35 years	Age 45 years
Asymptomatic people, without known risk factors	Age 45 years	Age 55 years

STEP 2: Measure and Record Risk Factors

A comprehensive cardiovascular risk assessment includes measurement and recording of the following:

- age
- gender
- ethnicity
- smoking history
- a fasting lipid profile
- a fasting plasma glucose
- the average of two sitting blood pressures
- family history
- waist circumference
- body mass index.

People with diabetes will require additional tests:

- HbA1c
- albumin:creatinine ratio
- creatinine
- date of diagnosis.

The risk of myocardial infarction and ischaemic stroke increases before diagnostic levels of plasma glucose for diabetes are reached. People with IGT, impaired fasting glucose (IFG) or the metabolic syndrome need active intervention and careful follow-up.

STEP 3: Risk Assessment

5-year cardiovascular risk in the following groups is assumed clinically to be more than 20%:

- people who have had a previous cardiovascular event (angina, myocardial infarction, angioplasty, coronary artery bypass grafts, transient ischaemic attack, ischaemic stroke or peripheral vascular disease)
- people with some genetic lipid disorders (familial hypercholesterolaemia, familial defective ApoB and familial combined dyslipidaemia)
- people with diabetes and overt nephropathy (albumin:creatinine ratio ≥30 mg/mmol) or diabetes with other renal disease.

Cardiovascular risk in all other people can be calculated using the National Heart Foundation's cardiovascular risk tables (see Figure 2, *Assessing 5-year Cardiovascular Risk and Treatment Benefit*), or an electronic decision-support tool based on the Framingham risk equation for first cardiovascular events.

People with isolated elevated single risk factor levels will have at least greater than 15% CV risk over 5 years. They should have a risk assessment because, when all risk factors are taken into account, they may have a calculated 5-year CV risk higher than this.

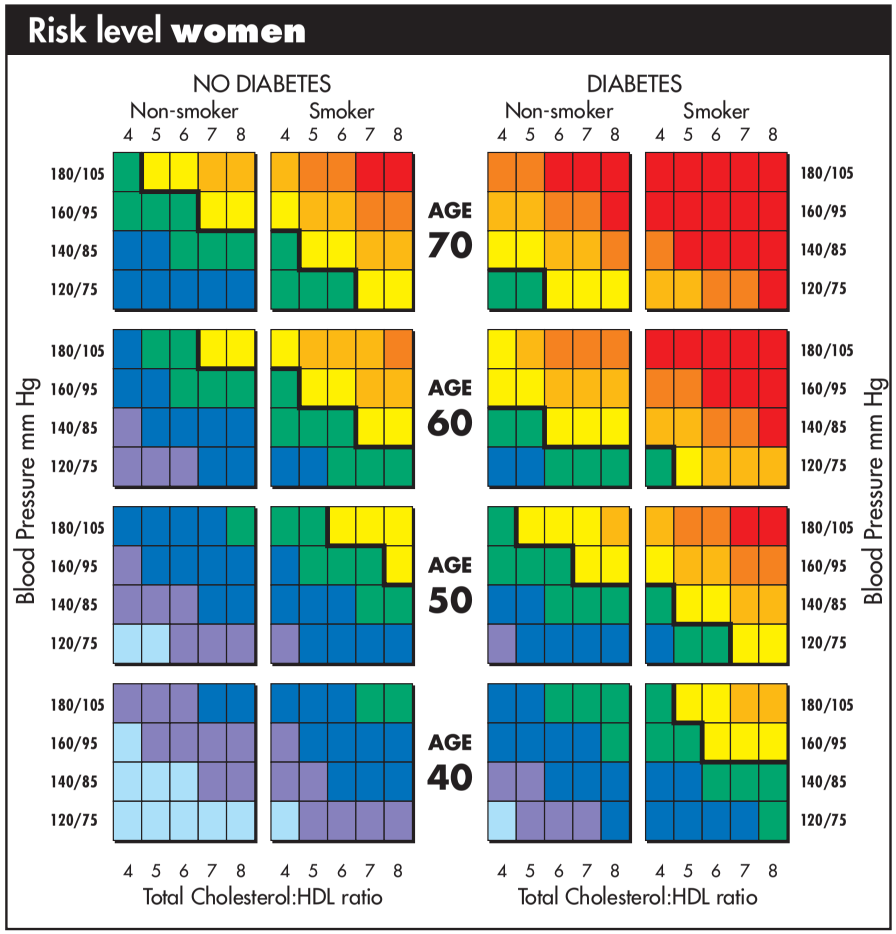
Isolated elevated single risk factor levels are defined as:

- TC greater than 8 mmol/L
- TC:HDL ratio greater than 8
- blood pressure consistently greater than 170/100 mm Hg.

Steps 4, 5 and 6: Interventions, Setting Treatment Targets and Follow-up

All treatment decisions should be based on an individual's 5-year absolute cardiovascular risk, and risk factor levels need interpretation in this context (see Figure 3 and Table 2).

ASSESSING CARDIOVASCULAR RISK AND TREATMENT BENEFIT



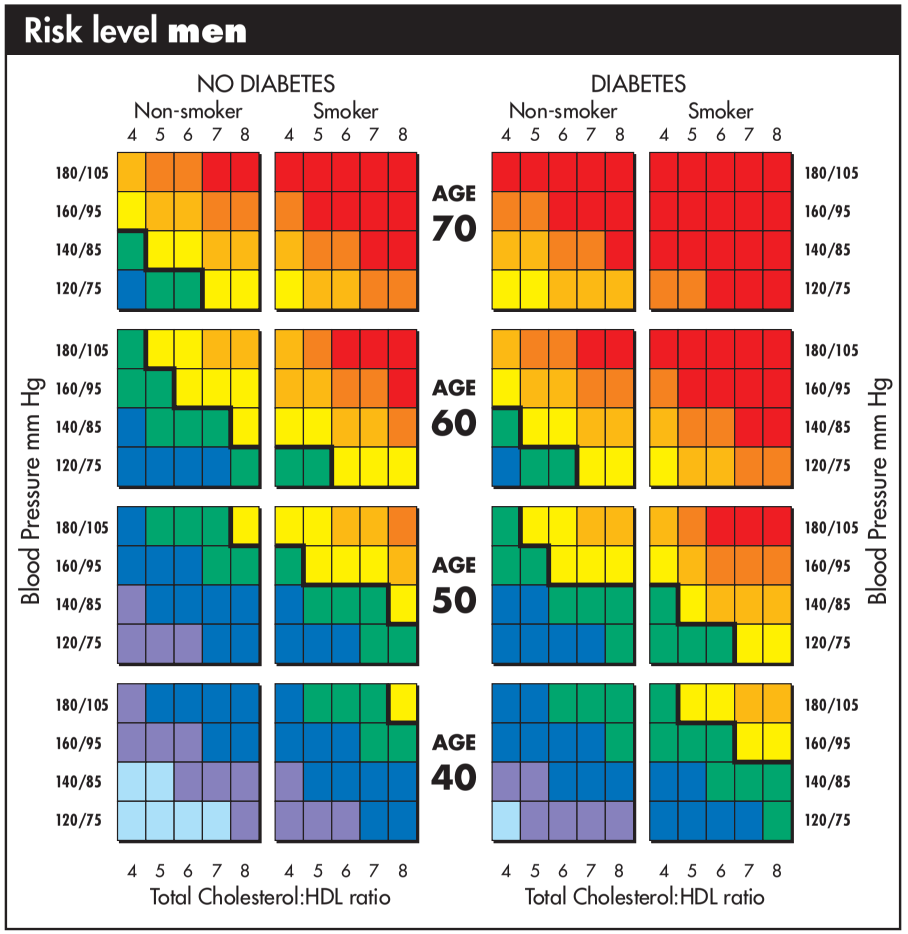


Figure 2: Assessing 5-year cardiovascular risk and treatment benefit

Risk Level
5 year CVD risk (non-fatal and fatal)

Very High	>30%	High	15–20%	Mild	5–10%
	25–30%	Moderate	10–15%		2.5–5%
	20–25%				<2.5%

How to use the Tables

- Identify the table relating to the person's sex, diabetic status, smoking history and age.
- Within the table choose the cell nearest to the person's age, blood pressure and TC:HDL ratio. When the systolic and diastolic values fall in different risk levels, the higher category applies.
- For example, the lower left cell contains all non-smokers without diabetes who are less than 45 years and have a TC:HDL ratio less than 4.5 and a blood pressure less than 130/80 mm Hg. People who fall exactly on a threshold between cells are placed in the cell indicating higher risk.

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MINISTRY OF HEALTH
MANATŪ HAUORA

NEW ZEALAND GUIDELINES GROUP

Notes for Figure 2

People at very high risk (>20% over 5 years) determined clinically

- People who have had a previous cardiovascular event (angina, myocardial infarction, angioplasty, coronary artery bypass grafts, transient ischaemic attack, ischaemic stroke or peripheral vascular disease).
- People with genetic lipid disorders (familial hypercholesterolaemia, familial defective ApoB and familial combined dyslipidaemia).
- People with diabetes and overt nephropathy (albumin:creatinine ratio >30 mg/mmol) or diabetes and other renal disease.

Where CV risk is determined using the Framingham risk equation and tables

The following groups should be moved up one risk category (5%), as their cardiovascular risk may be underestimated in the Framingham risk equation:

- people with a family history of premature coronary heart disease or ischaemic stroke in a first-degree male relative before the age of 55 years or a first-degree female relative before the age of 65 years
- Māori
- Pacific peoples or people from the Indian subcontinent
- people with both diabetes and microalbuminuria
- people who have had type 2 diabetes for more than 10 years or who have an HbA1c consistently greater than 8%
- people with the metabolic syndrome.

These adjustments should be made once only for people who have more than one criteria (the maximum adjustment is 5%).

Where risk factor levels are extreme

- If blood pressure is consistently greater than 170/100 mm Hg or total cholesterol greater than 8 mmol/L or TC:HDL ratio greater than 8 the person is classified at least at high risk (>15%) and should receive specific lifestyle advice and medication to lower their risk, irrespective of their calculated cardiovascular risk.
- For age greater than 75 years the 5-year cardiovascular risk is greater than 15% in nearly all individuals.

Risk level: 5-year CV risk (fatal and non-fatal)	**Benefits: NNT for 5 years to prevent one event** (CVD events prevented per 100 people treated for 5 years)		
	1 intervention (25% risk reduction)	**2 interventions (45% risk reduction)**	**3 interventions (55% risk reduction)**
30%	13 (7.5 per 100)	7 (14 per 100)	6 (16 per 100)
20%	20 (5 per 100)	11 (9 per 100)	9 (11 per 100)
15%	27 (4 per 100)	15 (7 per 100)	12 (8 per 100)
10%	40 (2.5 per 100)	22 (4.5 per 100)	18 (5.5 per 100)
5%	80 (1.25 per 100)	44 (2.25 per 100)	36 (3 per 100)

Based on the conservative estimate that each intervention: aspirin, blood pressure treatment (lowering systolic blood pressure by 10 mm Hg) or lipid modification (lowering LDL-C by 20%) reduces CV risk by about 25% over 5 years.

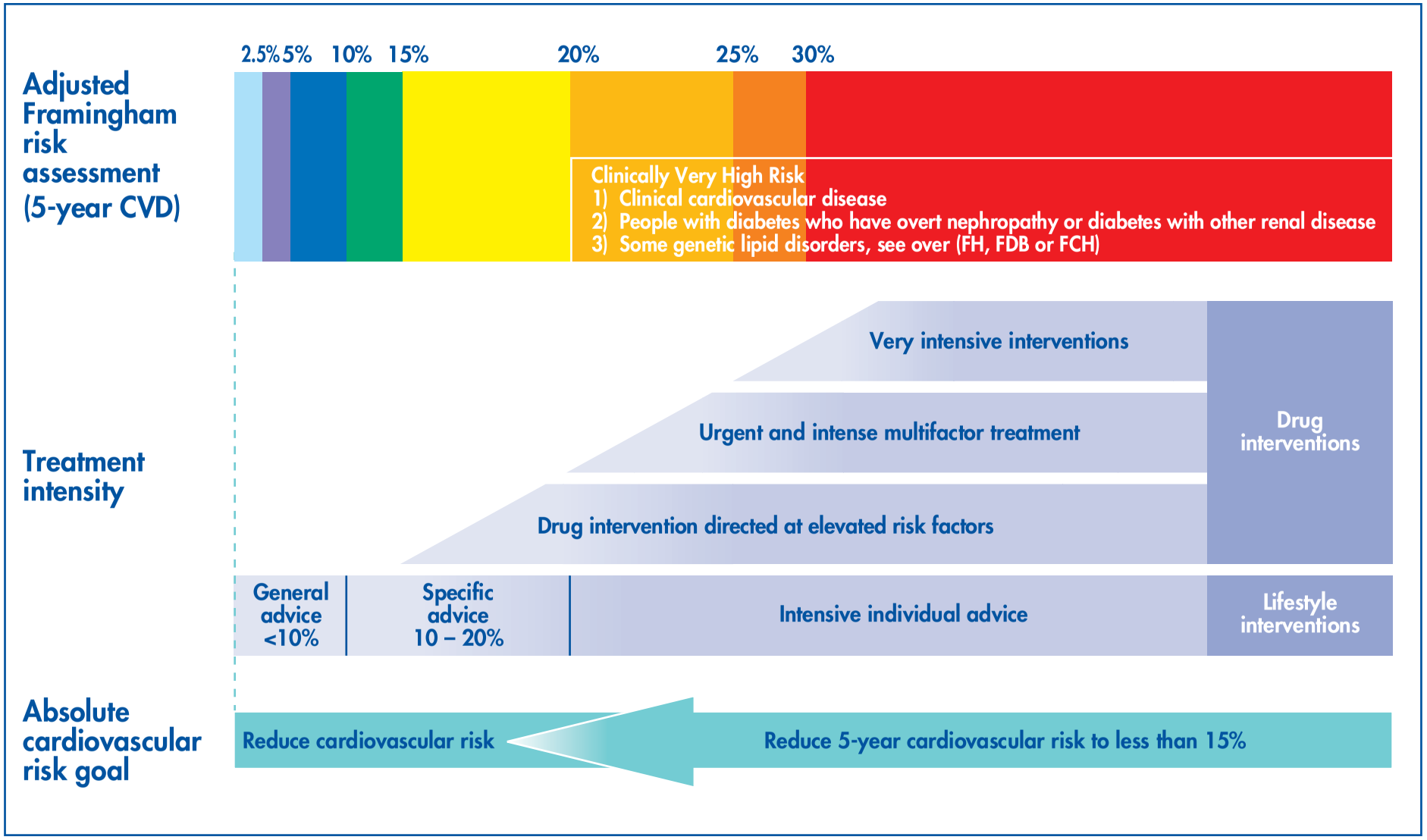


Figure 3: Treatment decisions based on 5-year cardiovascular risk

The higher the person's absolute risk of a cardiovascular event the more aggressively modifiable risk factors should be managed.

Table 2: Intervention according to cardiovascular risk assessment

Cardiovascular risk	Lifestyle	Drug therapy	Treatment goals	Follow-up
CVD risk clinically determined* more than 20%	Intensive lifestyle advice on a cardioprotective dietary pattern with a dietitian, physical activity and smoking cessation interventions. Lifestyle advice should be given simultaneously with drug treatment	Aspirin, if not contra-indicated, a beta blocker, statin and an ACE-inhibitor (after MI) or aspirin, statin and a new or increased dose of a blood pressure lowering agent (after stroke)	Efforts should be made to reach optimal risk factor levels	Cardiovascular risk assessments at least annually, risk factor monitoring every 3 to 6 months
CVD risk calculated more than 20%	Intensive lifestyle advice on a cardioprotective dietary pattern with a dietitian, physical activity and smoking cessation interventions. Lifestyle advice should be given simultaneously with drug treatment	Aspirin and drug treatment of all modifiable risk factors (blood pressure lowering, lipid modification and glycaemic control)	Risk factors treated to a level that will lower 5-year cardiovascular risk to less than 15% (by recalculating risk)	Cardiovascular risk assessments at least annually, risk factor monitoring every 3 to 6 months
15 to 20%	Specific individualised lifestyle advice on a cardioprotective dietary pattern, physical activity and smoking cessation. This lifestyle advice should be given by the primary health care team for 3 to 6 months prior to initiating drug treatment	Aspirin and drug treatment of all modifiable risk factors (blood pressure lowering, lipid modification and glycaemic control). Drug therapy indicated for people with extreme risk factor levels#	Risk factors treated to a level that will lower 5-year cardiovascular risk to less than 15% (by recalculating risk)	Cardiovascular risk assessments at least annually, risk factor monitoring every 3 to 6 months
10 to 15%	Specific individualised lifestyle advice on a cardioprotective dietary pattern, physical activity and smoking cessation. This lifestyle advice should be given by the primary health care team	Non-pharmacological approach to treating multiple risk factors	Lifestyle advice aimed at reducing cardiovascular risk	Further cardiovascular risk assessment in 5 years
less than 10%	General lifestyle advice on a cardioprotective dietary pattern, physical activity and smoking cessation	Non-pharmacological approach to treating multiple risk factors	Lifestyle advice aimed at reducing cardiovascular risk	Further cardiovascular risk assessment in 5 to 10 years

*People who have had a previous cardiovascular event (angina, myocardial infarction, angioplasty, coronary artery bypass grafts, transient ischaemic attacks, ischaemic stroke or peripheral vascular disease) OR people with certain genetic lipid disorders (FH, FDB ,FCH) OR people with diabetes and overt diabetic nephropathy OR people with diabetes and renal disease

People with isolated high risk factor levels either total cholesterol greater than 8 mmol/L or TC:HDL ratio greater than 8 or blood pressure greater than 170/100 mm Hg should have these risk factors treated regardless of their calculated cardiovascular risk.

BACKGROUND

Cardiovascular disease is the leading cause of death in New Zealand, accounting for 40% of all deaths. While age-standardised mortality has halved over the past 30 years, the total number of deaths from cardiovascular disease has changed little because of the growing number of older people and at-risk individuals.[6]

In 1998 New Zealand had an age-standardised mortality rate from coronary heart disease of 111 per 100,000 compared to 150 per 100,000 in Scotland; 107 per 100,000 in the USA; 94 per 100,000 in Australia; and 31 per 100,000 in Japan in 1997.[6] There were 23,569 admissions to hospital for coronary heart disease in New Zealand per annum in 1998/99.[20] In New Zealand, approximately 7000 people will have a stroke every year and fewer than half will be alive and independent one year after stroke. In 2001, there were an estimated 32,000 survivors of stroke in New Zealand.[21,22]

Coronary heart disease death rates for Māori aged less than 75 years are 2 to 3 times higher than those in non-Māori[6] and up to twice as high for Pacific peoples. Coronary heart disease occurs at a younger age in Māori with 53% of male coronary heart disease deaths and 33% of the female coronary heart disease deaths occurring in those under 65 years.[6] An Auckland study has shown that Māori and Pacific peoples have a higher relative risk of stroke than New Zealand Europeans and a higher risk of death within 28 days of stroke. The mean age for a stroke event was 55 years in Māori, 60 years in Pacific peoples and 73 years in New Zealand Europeans.[23]

In this guideline cardiovascular disease is defined as angina, myocardial infarction, ischaemic stroke, transient ischaemic attack and peripheral vascular disease.

Established risk factors for cardiovascular disease include:

- age
- sex
- personal history of cardiovascular disease
- smoking
- blood pressure
- lipids
- IGT or diabetes
- obesity
- physical inactivity
- atrial fibrillation
- a family history of coronary heart disease
- socioeconomic position.

No risk factor should be judged in isolation. Individuals with multiple risk factors are at greatly increased risk of a cardiovascular event.[24,25](**2++**) These risk factors tend to cluster and act synergistically. When considering an intervention, the individual's absolute risk of a cardiovascular event should be determined.[26](**2++**)

2

CARDIOVASCULAR HEALTH OF MĀORI

KEY MESSAGES

- The burden of cardiovascular disease falls heavily on Māori.
- Current cardiovascular care in New Zealand demonstrates the inverse care law – those most in need receive the least care.
- Practitioner bias and discriminatory practice may influence access to and through cardiovascular care service.
- By applying evidence-based guidelines to clinical decision-making there is potential to improve the lives of Māori individuals, their wider whānau, hapū and iwi.
- Absolute cardiovascular risk determines treatment decisions and benefits. Within this guideline, cardiovascular risk assessment is recommended ten years earlier for Māori.
- Significant health and economic gains can be made for all New Zealanders when Māori cardiovascular health is improved.
- The Māori Cardiovascular Action Plan provides a guide for cardiovascular policy development and implementation by New Zealand health services.
- A population health approach is indicated.

INTRODUCTION

Māori die almost a decade earlier than non-Māori in New Zealand. Cardiovascular disease contributes significantly to Māori death and disease and premature Māori mortality and morbidity.[27]

This chapter addresses cardiovascular inequalities and encourages health practitioners to reflect on the evidence and take action on the findings within this guideline. Māori need to be moved from the fringe of New Zealand's health care system toward its centre; Māori cardiovascular health outcomes must be improved.

Burden of Disease

The burden of cardiovascular disease falls heavily on Māori. Since the late 1960s (when coronary heart disease rates peaked in New Zealand), age-standardised death rates for coronary heart disease have fallen for both Māori and non-Māori.[28] However, these reductions have been smaller for Māori compared with non-Māori.

As a result Māori disparities in cardiovascular disease have widened during the 1980s and 1990s.[29]

Cardiovascular death and disease occur prematurely for Māori. Age-specific coronary heart disease death rates are 2 to 3 times higher for Māori compared with non-Māori in those aged less than 75 years.[6] Approximately 1 in 2 Māori males and 1 in 3 Māori females with coronary heart disease die of the disease before the age of 65 years. This compares with 1 in 5 non-Māori male and 1 in 20 non-Māori female coronary heart disease deaths that occur before the age of 65 years.[6]

Mortality rates from cerebrovascular disease are 1.2 times higher for Māori than non-Māori[6] with the mean age for a stroke event occurring at 55 years for Māori, 60 years for Pacific peoples and 73 years for New Zealand Europeans.[23]

Inverse Care Law

Current cardiovascular care in New Zealand demonstrates the inverse care law – those most in need receive the least care. Given the higher prevalence of cardiovascular diseases among Māori men and women[6,30,31] and the ethnic difference in cardiovascular outcomes, one would expect to find a level of health service intervention that matched that need. However, Māori receive fewer cardiac interventions than required and expected.[32-35]

A recent study in New Zealand found that Māori men received well below half the age-standardised coronary artery bypass grafting (CABG) and percutaneous coronary angioplasty (PTCA) interventions that non-Māori men received.[36] Māori women received about 74% of CABG and 43% of PTCA interventions that non-Māori women received. Of note in this study, the Pacific experience was found to be either equal to that of Māori or intermediary between Māori and New Zealand Europeans. In another study that controlled for differences in age, sex and deprivation, Māori hospitalisation rates for CABG and PTCA interventions were found to be about half those of non-Māori.[35]

This clearly exemplifies the inverse care law which states that the availability of health care varies inversely to the need of the population served.[37] The fact that ethnic inequalities in cardiovascular disease exist and are widening[6,29]means that these inequalities can also be reduced and eliminated. Current government policy[2,3,38,39] and health legislation[40] note that it is no longer acceptable to tolerate these inequalities in New Zealand.

Practitioner Bias and Discriminatory Practice

Bias and discriminatory practice may influence access to and through cardiovascular care services. A recent literature review of both national and international studies found that even after controlling for differences in clinical factors, individual's preferences, socioeconomic position, and health system organisation, ethnic disparities in cardiovascular care persist.[41]

Bias among health practitioners may lead to differences in the selection of people for medical assessment and treatment. Some studies have suggested physician bias influences access to care, ranging from simple pharmacological therapy to heart transplantation.[42,43] Practitioner bias may manifest as direct interpersonal discrimination (such as inherent assumptions made about a person's information needs, ability to comply, or to make healthy choices based on their ethnicity) and through institutional racism (such as the creation of 'gate-keeper' barriers to health care services).[44] Such actions, both conscious and unconscious, may undermine our strongest health policy strategies.

A number of models provide insight into the nature of ethnic health disparities. In New Zealand, the *gap framework* uses the NZDep[45] to interpret the effects of socioeconomic deprivation on health outcomes.[11] It identifies three gaps:

1. *The distribution gap* – over half of Māori are in the three most deprived deciles in New Zealand. This means Māori are exposed to a higher incidence and prevalence of cardiovascular diseases and cardiovascular risk factors.[46]
2. *The outcome gap* – across deprivation deciles, average Māori life expectancy at birth is significantly lower than that of non-Māori. Notably, Māori in the least deprived deciles have a lower life expectancy than non-Māori in the most deprived deciles.
3. *The gradient gap* – as deprivation increases, the relative difference in outcomes (eg, mortality) between Māori and non-Māori widens.

The *cultural deficit model* attributes ethnic inequalities in health to genetic, biological or cultural/lifestyle factors.[47] This model assumes that ethnic health disparities are due to the failings and deficits of indigenous peoples and their cultures, rather than taking account of the role that structural institutions and discriminatory practices may play in their production. In New Zealand, health research is often consonant with the cultural deficit model. Māori health outcomes are typically framed as 'abnormal' compared to the 'normal' New Zealand population.[11]

Kreiger[48] in 2001 described two opposed theories that are used to explain ethnic inequalities in health: the ethnic expression of biology (ie, genes and biomedical causation) or the biological expression of racism (ie, social causation of disease distribution and population health). Of the two theories, social causation has received increasing interest and understanding.[49,50]

However, Jones postulates that the association between socioeconomic position, ethnicity and health is determined by racism.[46] She identifies three levels of racism: *institutionalised*, *personally-mediated* and *internalised*.

Institutional racism is defined as:

> differential access to goods, services and opportunities of society by race. It is structural, having been codified in our institutions of custom, practice, and law so there need not be an identifiable perpetrator. Indeed, institutionalised racism is often evident as inaction in the face of need. (p 300)

Personally-mediated racism is defined as:

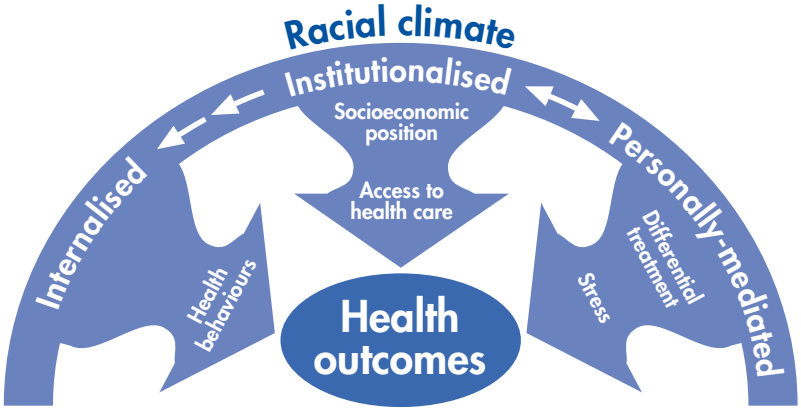
> prejudice and discrimination, where prejudice is differential assumptions about the abilities, motives, and intents of others by race, and discrimination is differential actions towards others by race. (p 300)

Lastly, internalised racism is defined as:

> acceptance by members of the stigmatised races of negative messages about their own abilities and intrinsic worth. (p 300)

Institutional racism is the most important because it manifests as ethnic differences in income, education, occupation, housing and access to health care. Jones presents a pictorial representation of the relationship between racism and health outcomes that can be applied to the New Zealand racial climate (see Figure 4).

Figure 4: Jones model on the impact of racism on health



Source: Jones 2001[46]

Using Evidence to Bridge the Gap

By applying evidence-based guidelines to clinical decision-making there is potential to improve the lives of Māori individuals, their wider whānau, hapū and iwi and to help bridge the gap between research and actual practice – to counter the inverse care law.

By applying risk assessment criteria to clinical decision-making, clinical practice can be re-oriented to meet the needs of Māori. This guideline supports a population health approach to cardiovascular risk assessment and management. This includes the need to focus interventions on population groups that bear the greatest burden of disease. The priority must be to adequately assess and treat those who are identified as being at high risk with multiple risk factors.

Evidence also supports a lifecourse approach to cardiovascular risk assessment and management. A lifecourse perspective considers how the accumulation and interaction of risk factors at all stages in life affect absolute cardiovascular risk - from the time a person is born with a set of familial and socioeconomic risk factors, to later life events such as health service interactions that further accumulate or mitigate risk. A stepwise approach to management based on absolute risk means that those at highest risk are targeted and treated more aggressively than those at lower risk, across the entire lifecourse.

Risk Assessment 10 Years Earlier for Māori

Absolute cardiovascular risk determines treatment decisions and benefits. Within this guideline, cardiovascular risk assessment is recommended ten years earlier for Māori. At any age, Māori have an increased prevalence of cardiovascular disease and cardiovascular risk factors compared with non-Māori. Thus, 5-year absolute cardiovascular risk is likely to be higher for Māori than non-Māori at the same age. For this reason, cardiovascular risk assessment is recommended in this guideline 10 years earlier for Māori (ie, from the age of 35 years for Māori men and 45 years for Māori women). Although this recommendation is derived from a needs-based approach, it is supported by indigenous rights and the Treaty of Waitangi, which are equally important drivers toward equity of cardiovascular health care and health outcomes for Māori.

Positive Impact for All New Zealanders

Significant health and economic gains can be made for all New Zealanders when Māori cardiovascular health is improved. If practitioners use their clinical expertise to implement these guidelines there is potential to significantly improve the lives of Māori individuals, their wider whānau, hapū and iwi. In addition, by targeting Māori cardiovascular inequalities we are likely to see benefits that extend beyond Māori to impact positively on all New Zealanders. These are:

- a fairer society where everyone has the opportunity for good health
- an inclusive society where everyone has a sense of belonging and feels that their contribution is valued
- improved health and well-being for the population as a whole not just for those groups who are currently experiencing relatively poor health
- a stronger economy because a healthier population can contribute to a richer social and economic life.[3]

Māori Cardiovascular Action Plan

The Māori Cardiovascular Action Plan provides a guide for cardiovascular policy development and implementation. The Māori Cardiovascular Advisory Group recommends that the implementation of these guidelines proceed first for Māori as priority. The group has written the Māori Cardiovascular Action Plan to help guide cardiovascular policy development and implementation by health services in New Zealand.

Within the plan several areas for health service action are proposed.

1. Policy – Treaty of Waitangi based policy and decision-making
2. Information systems – complete and consistent collection of ethnicity data; service provider funding
3. Access, delivery and standards development – cardiovascular health needs assessments; kaupapa Māori health services
4. Audit, evaluation and quality standards improvement – measurement of key performance indicators to monitor service responsiveness to Māori cardiovascular needs
5. Workforce and health service development
6. Research.

The Māori Cardiovascular Action Plan moves the focus away from individuals to interventions aimed at a service and health practitioner level. The action plan is to be published separately.

The challenge for health practitioners is to re-orient their current practice within the context of this guideline and the Māori Cardiovascular Action Plan. This may include the need to audit the implementation of best-practice systems in order to identify, and modify, differential service provision by ethnicity. A further challenge both nationally and for DHBs will be to evaluate and monitor their ongoing implementation.

Population Health

Any health service intervention that focuses on individuals and changing lifestyle behaviours has limitations. This guideline highlights the need for practitioners to be aware of both the presence of cardiovascular risk factors at the individual level, as well as the socioeconomic determinants of health that impact on populations. This includes an awareness of the evidence for practitioner bias and discriminatory practice.

Targeted lifestyle interventions aimed at risk factor reduction will partially address Māori disparities in cardiovascular outcomes. However, action must go beyond health promotion and traditional health sector approaches, to focus on the root causes of differential income, employment, deprivation, education and housing for Māori in New Zealand. Structural problems require structural solutions across many sectors: income, employment, housing, education and health. Action at the level of social structures is essential to eliminate inequalities in health.[51]

The health sector can set a precedent for this. Population health programmes, personal health services and disability support services can and should:

- include the Treaty of Waitangi in policy development so that Māori health gain is recognised as a priority in service planning and provision, including strategic goals and objectives
- commit to service-wide education and recognition of the Treaty of Waitangi, the wider socioeconomic determinants of health, and the elimination of ethnic inequalities in health
- commit to the complete and consistent collection of ethnicity data
- conduct cardiovascular health needs assessments for the populations they serve in order to identify levels of unmet need
- allocate resources appropriately to reflect unmet need, and match this with the government mandate to reduce socioeconomic and ethnic inequalities in health. This may require expenditure analyses (of current and required budgets) and dedicated resource and funding allocations
- audit and evaluate their service using key performance indicators that monitor responsiveness to Māori cardiovascular need and ensure continuous quality improvement
- develop a Māori workforce plan including affirmative action policies and proactive career planning (recruitment, advancement and retention)
- identify barriers in access to care, and expand Māori access to cardiovascular preventive and treatment services. This may include piloting new models of service delivery such as ambulatory care in marae or community-based clinics.

CONCLUSION

In conclusion, eliminating Māori inequalities in health is a high-level priority for our health services that will be supported by evidence-based practice. There is substantial scope to effectively and efficiently achieve health gains and remove Māori cardiovascular inequalities in New Zealand.

KEY - *Grades indicate the strength of the supporting evidence not the importance of the recommendations - see page xvii for details*

A Recommendation is supported by good evidence
B Recommendation is supported by fair evidence
C Recommendation is supported by non-analytic studies or consistent expert opinion
I No recommendation can be made because the evidence is insufficient
✓ Good Practice Point

CARDIOVASCULAR RISK ASSESSMENT

RECOMMENDATIONS: WHO SHOULD BE ASSESSED	
Cardiovascular risk assessments are recommended: • from the age of 45 years for asymptomatic men without other known risk factors • from the age of 55 years for asymptomatic women without other known risk factors.	C
Cardiovascular risk assessments are recommended 10 years earlier for Māori (from the age of 35 years for men and 45 years for women).	C
Cardiovascular risk assessments are recommended 10 years earlier for Pacific peoples and people from the Indian subcontinent (from the age of 35 years for men and 45 years for women).	C
Cardiovascular risk assessments are recommended annually from the time of diagnosis for people with diabetes.	C
Cardiovascular risk assessments are recommended: • from the age of 35 years for men with other known cardiovascular risk factors or at high risk of developing diabetes • from the age of 45 years for women with other known cardiovascular risk factors or at high risk of developing diabetes. These people will have one or more of the following risk factors: • family history of premature cardiovascular disease in a first-degree male relative (parent or sibling) under 55 years or female relative under 65 years • family history of diabetes in a first-degree relative (parent or sibling) • personal history of gestational diabetes • personal history of polycystic ovary syndrome • personal history of current or recent smoking • prior blood pressure of more than 160/95 mm Hg* • prior TC:HDL ratio of more than 7* • known IGT or IFG (see Table 22) • obesity (BMI ≥30*) or truncal obesity (waist circumference ≥100 cm* in men or ≥90 cm* in women).	C

*Where risk factor thresholds are given these should be interpreted as approximate guides to clinical practice only.

RECOMMENDATIONS: WHO SHOULD BE ASSESSED (CONTINUED)	
All those with cardiovascular disease should have comprehensive risk factor measurements to determine the best management approach.	C
Tracing the siblings and children of index cases known to have a genetic lipid disorder is the recommended method of identifying individuals with genetic lipid disorders.	C

RECOMMENDATION: WHO SHOULD DO THE ASSESSEMENT	
Risk assessments should be provided at the primary care level by health practitioners with appropriate training, infrastructure support, systems for follow-up and systems that improve quality.	C

RECOMMENDATIONS: FREQUENCY OF CARDIOVASCULAR RISK ASSESSMENT	
People with a 5-year cardiovascular risk under 5% should have a further cardiovascular risk assessment in 10 years.	C
People with a 5-year cardiovascular risk between 5 and 15% should have a further cardiovascular risk assessment in 5 years.	C
Annual cardiovascular risk assessments are recommended in people with: • a 5-year cardiovascular risk greater than 15% • diabetes • people receiving treatment with lipid-modifying or blood pressure lowering medication.	C
People with diabetes or receiving medication or intensive lifestyle advice may need individual risk factor measurements taken more frequently, eg, monitored 3 monthly until controlled, then every 6 months.	C

TARGETING CARDIOVASCULAR RISK ASSESSMENT

The purpose of screening is to detect increased cardiovascular risk in asymptomatic people and also in the sub-group who have genetic lipid disorders. There are no data currently quantifying the benefits or harms of population screening in asymptomatic people and none to suggest that an organised population screening programme is warranted. If practitioners are to offer screening to people in the groups identified in the guidelines, they should also ensure there is a process for auditing this practice to ensure that it is safe and effective, and for assessing outcomes. A number of tools are available for use in primary care that are suitable for this process which is made easier by the use of electronic patient management systems.

KEY - *Grades indicate the strength of the supporting evidence not the importance of the recommendations - see page xvii for details*

A Recommendation is supported by good evidence
B Recommendation is supported by fair evidence
C Recommendation is supported by non-analytic studies or consistent expert opinion
I No recommendation can be made because the evidence is insufficient
✓ Good Practice Point

Practitioners should be aware of the need to focus on population groups that have a high burden of cardiovascular disease. These groups should be specifically targeted to ensure that they are able to benefit from risk screening and subsequent management. Practice audit should include examining the extent to which these groups are participating in screening.

There is evidence that a programme of targeted screening of asymptomatic individuals (non-smoking men aged 45 years and over or non-smoking women aged 55 years and over: smoking men aged 35 years and over or smoking women aged 45 years and over), followed by appropriate treatment with statins is cost-effective compared with other community drug therapies that are funded in New Zealand (see Appendix B).[52](**2E**) Targeted screening for diabetes, followed by appropriate lipid-modifying treatment, as part of a cardiovascular risk assessment is likely to be even more cost-effective.[52](**2**)

The most cost-effective way to identify people with familial hypercholesterolaemia is family tracing of the siblings and children of index cases known to have a genetic lipid disorder.[53,54](**2**)

At any given age, there is an increased prevalence of cardiovascular disease in Māori. The decision to start risk assessments a decade earlier for Māori is thus based on demonstrated need and made in recognition of indigenous rights.

At any given age, there is also an increased prevalence of cardiovascular disease in Pacific peoples and those from the Indian subcontinent compared to other ethnic groups.[6,23] For this reason screening in these ethnic groups is recommended a decade earlier.

Anyone offered a cardiovascular risk assessment should be fully informed of the known likely individual benefits, harms and implications of screening.

ABSOLUTE CARDIOVASCULAR RISK

Absolute cardiovascular risk is the likelihood that a person will have a cardiovascular event over a given period of time.

In New Zealand, absolute cardiovascular risk is usually calculated from the National Heart Foundation's cardiovascular risk tables or electronic decision support tools based on the US Framingham Heart Study.[26] The Framingham Heart Study assessed risk factors in about 5200 males and females aged 30 to 74 years and monitored long-term cardiovascular outcomes. The Framingham risk equations are not applicable to people under the age of 35 years and may have limited applicability to Māori or Pacific peoples in New Zealand.

A first cardiovascular event is defined in the Framingham risk equation as:

- myocardial infarction
- new angina
- ischaemic stroke
- transient ischaemic attack
- peripheral vascular disease
- congestive heart failure
- cardiovascular-related death.

Framingham risk equations have been validated in different populations.[55](**2++**) The cardiovascular equation has been validated and is currently the best tool for estimating cardiovascular risk in New Zealand.[56,57]

The accuracy of cardiovascular risk assessments is limited by the precision of the Framingham risk equation which dichotomises risk factors, excludes some major risk factors (obesity, physical inactivity and a family history of cardiovascular disease) and has some measurement issues. However, it is the

best available tool for predicting cardiovascular risk and is more accurate for predicting cardiovascular events than using the levels of individual risk factors.[58,59](**3**)

Framingham risk equations may be used to calculate cardiovascular risk over different time periods (5 or 10 years) and may be used to calculate coronary heart disease risk and/or ischaemic stroke risk separately.[26](**2++**)

A *cardiovascular* disease risk of 15% over 5 years (30% over 10 years) is approximately equivalent to a *coronary* heart disease risk of 20% over 10 years.[60](**2++**)

Coronary heart disease risk is used by the National Cholesterol Education Programme (NCEP)[61] and UK guidelines.[62]

A risk assessment tool using data from the UKPDS study can be used to assess risk in people with type 2 diabetes.[63] This equation includes the additional parameters of HbA1c, duration of diabetes, and atrial fibrillation, and gives separate stroke and coronary heart disease risk calculations. It is available for downloading at www.dtu.ox.ac.uk

CARDIOVASCULAR RISK FACTORS

Standard Risk Factors

The standard cardiovascular risk factors included in the National Heart Foundation's cardiovascular risk tables are listed below.

A Personal History of Cardiovascular Disease

This is defined as previous myocardial infarction, angina, ischaemic stroke, transient ischaemic attack, peripheral vascular disease, or congestive heart failure.

The majority of myocardial infarctions occur in people who are known to have cardiovascular disease.

Age

Age is a major risk factor, with the prevalence of cardiovascular disease increasing with age. In New Zealand, 85% of all coronary heart disease deaths occur in those aged 65 years and over.[64]

Sex

The age-specific mortality rate from coronary heart disease is 2 to 5 times higher for middle-aged men than women.[64] In contrast, the death rates for cerebrovascular disease are similar for men and women of the same age.[6]

Smoking Status

The risk of coronary heart disease is 2 to 3 times higher in smokers. The relationship between the amount a person smokes and cardiovascular disease is continuous.[65] In New Zealand, adult cigarette smokers smoke an average of 12 cigarettes per day. One half of Māori adults smoke cigarettes (95% CI, 47.9 – 54.4%) and 21% of non-Māori adults (95% CI, 20.4 – 22.3%).[66]

Lipids

Prospective studies demonstrate a constant linear relationship between relative cardiovascular risk and total cholesterol (TC) in the range of about 4 to 8 mmol/L.[67,68] The higher the total cholesterol level the greater the cardiovascular risk. A weaker association has been observed with ischaemic stroke. People at high risk of a cardiovascular event with low to normal LDL-C levels may benefit from further reductions in LDL-C with lipid-modifying treatment.[69] A 1.0 mmol/L reduction in LDL-C leads to a 30 to 35% reduction in coronary heart disease and a 30 to 35% reduction in stroke.[70]

In 1997, the average total cholesterol in New Zealand was 5.7 mmol/L (LDL-C greater than 4.0 mmol/L) and 23% of the population had a total cholesterol greater than 6.5 mmol/L (LDL-C greater than 5.0 mmol/L). The average high density lipoprotein cholesterol (HDL-C) for men was 1.2 mmol/L and for women 1.5 mmol/L, but due to data limitation no ethnic-specific prevalences were calculated.[14]

HDL-C is an independent predictor of cardiovascular risk.[71,72] In the Framingham Heart Study, TC:HDL ratio was found to be a better predictor of the likelihood of cardiovascular events than either total cholesterol, LDL-C, HDL-C or triglycerides considered individually.[73] A low HDL-C increases cardiovascular risk, but a high HDL-C does not necessarily imply low risk. The cardiovascular risk of people with high LDL-C, and high HDL-C may be higher than predicted with the risk charts. HDL-C cannot be accurately measured in the laboratory if triglyceride levels are elevated (over 4.0 mmol/L). Therefore those with high triglyceride levels need a fasting blood test and follow-up. A triglyceride level above 1.7 mmol/L (fasting) is a predictor of cardiovascular risk.

Blood Pressure

Prospective studies demonstrate a constant linear relationship between relative cardiovascular risk and blood pressure levels in the range of about 155/70 to 170/100 mm Hg. A 10 mm Hg reduction in systolic blood pressure or a 5 mm Hg reduction in diastolic blood pressure between the ages of 40 and 69 years is associated with about a 40% reduction in the risk of stroke and a 30% reduction in the risk of death from ischaemic heart disease or other vascular causes.[74] **(2++)**

One in five New Zealanders over the age of 15 years have a blood pressure over 160/95 or are taking medication to lower blood pressure.[27] Data limitations in this study prevented the calculation of ethnic-specific prevalences.

Diabetes

There is a strong association between blood glucose and the risk of cardiovascular disease. Prospective studies suggest that there is a constant linear relationship between cardiovascular risk and HbA1c[75] and between cardiovascular risk and 2-hour blood glucose levels.[76] In people with diabetes, each 1% decrease in HbA1c is associated with a 7% reduction in myocardial infarction over 5 years.[77] The overall prevalence of diagnosed diabetes in New Zealand adults has been estimated to be 3 to 4%.[78] The prevalence of diagnosed diabetes among Māori (5 to 10%) and Pacific peoples (4 to 8%) is higher than among New Zealanders of European origin (3%) and Asian origin (4%).[79,80]

Other Well-established Determinants of Cardiovascular Risk

Atrial Fibrillation

In people with atrial fibrillation the risk of stroke increases 5-fold compared to those of a similar age without atrial fibrillation. The incidence of atrial fibrillation increases with age, and 23.5% of strokes in the age group 80 to 89 years are attributable to atrial fibrillation. People aged 75 years and over are particularly vulnerable to stroke when atrial fibrillation is present.[81] If selected people with atrial fibrillation are treated with warfarin, the risk of embolic stroke is reduced by about two-thirds. For further details see the New Zealand Guideline on the Assessment and Treatment of Atrial Fibrillation.

Obesity

People with a body mass index (BMI) over 30 have a 40-fold increased risk of developing diabetes and a 2 to 3-fold increase in risk of both coronary heart disease[82,83] and thromboembolic stroke.[84] Central (truncal) obesity, measured by waist circumference, is a better predictor of coronary heart disease risk, than general obesity determined by BMI.[82] Truncal obesity is associated with the insulin resistant state (the metabolic syndrome). One-sixth (17%) of New Zealand adults have a BMI greater than 30.[14]

Impaired Carbohydrate Metabolism

IGT is a significant risk factor for the development of diabetes[85] and cardiovascular disease.[76] Hyperinsulinaemia is associated with IGT, and these two risk factors are frequently associated with the raised blood pressure, obesity, raised triglycerides and low HDL-C in the metabolic syndrome.

Metabolic Syndrome

The metabolic syndrome or insulin resistant state (defined in Table 24) is a significant risk factor for cardiovascular disease. People with the metabolic syndrome may have a cardiovascular risk approaching that of people with diabetes.[86-88]

Nutrition and Dietary Patterns

Modern Western dietary patterns are energy-dense and characterised by higher intakes of red meat, processed meat, refined grains, sweets and dessert, deep-fried potato and high-fat dairy products. These dietary patterns predispose to weight gain, are associated with selected components of the metabolic syndrome, and higher rates of type 2 diabetes and coronary heart disease.[89-93]

Physical Inactivity

Regular physical activity is associated with reduced risk of cardiovascular disease morbidity and mortality.[94] This observation is consistent over a range of intensities and frequencies with the more intense and frequent activity conferring greater protection (a dose-related effect).[95,96] The protective effect of physical activity is greatest in individuals at higher risk of cardiovascular disease.[97]

More than one third of New Zealand adults are inactive (ie, they take part in less than 2.5 hours of physical activity per week). A sixth of adults are sedentary and perform no physical activity at all.[98]

Family History of Premature Cardiovascular Disease

This is defined as a history of clinically proven cardiovascular disease (angina, myocardial infarction, transient ischaemic attack, or ischaemic stroke) in a first-degree relative (parent, sibling) before the

age of 55 years in men and 65 years in women. In people with a family history of coronary disease, the risk of a coronary event is approximately doubled.[99] The risk of ischaemic stroke in men with a family history of stroke is slightly less than double that risk for those without a family history, RR 1.89 (95% CI, 1.23 – 2.91).[100]

Socioeconomic Position

There is approximately a 2-fold increase in the risk of cardiovascular death for those people living in the areas with the lowest as compared to the highest socioeconomic rating, as measured by the NZDep score.[7] Māori are over-represented in the most socioeconomically disadvantaged areas of New Zealand. However, even after adjusting for socioeconomic position, the risk for Māori remains higher than that for non-Māori.

Depression, Social Isolation and Social Support

New evidence suggests an independent causal association between depression, social isolation and lack of quality social support and the causes and prognosis of coronary heart disease.[101] The systematic review by the National Heart Foundation of Australia found no evidence for a causal association between chronic life events, work-related stressors (job control, demands and strain), Type A behaviour patterns, hostility, anxiety disorders or panic disorders and coronary heart disease.[101]

Emerging Risk Factors that May Have a Role in Predicting Cardiovascular Risk

There are a number of other risk factors associated with cardiovascular disease but the size of their independent predictive value is not quantified and there is limited data on the effectiveness of interventions. Assessment of emerging risk factors is only useful in special circumstances as their measurement may be unreliable and they are not included in the Framingham risk equation.

ApoB

Apolipoprotein B (ApoB) is the major protein associated with LDL-C, very low density lipoprotein (VLDL) and intermediate density lipoprotein (IDL). ApoB is a good predictor of risk and may be more widely used as assays become standardised.

LDL-C Particle Size

Small LDL-C particle size promotes atherogenicity. Triglyceride levels over 1.7 mmol/L are a useful surrogate measure for small dense LDL-C.

Urinary Albumin/Microalbuminuria

Microalbuminuria is a marker for generalised endothelial damage and urinary albumin excretion correlates with atherosclerotic arterial disease. Microalbuminuria may indicate vascular damage, especially in people with elevated blood pressure.[102] However, further data on the effectiveness of interventions in reducing albumin excretion and its impact on cardiovascular outcomes is required before routine measurement can be recommended. In people with diabetes or other glucose disorders, raised albumin excretion or microalbuminuria increases the likelihood of cardiovascular events.[103] Microalbuminuria often predates progression to overt renal nephropathy.

Hs-CRP

C-reactive protein (CRP) is a non-specific marker of inflammation. High sensitivity assay methods for CRP (Hs-CRP) allow accurate measurement of small increases in CRP and there is good evidence that high concentrations of HsCRP identify people at higher risk of cardiovascular disease.[104] Recent recommendations from the AHA/CDC indicate that the best discretionary use of Hs-CRP is in refining the risk profile of those who fall near a threshold for pharmacological treatment in primary prevention. Hs-CRP is not recommended for general screening or in those already at high risk or for monitoring treatment.[105]

Lp(a)

Lp(a) is a lipoprotein that consists of a low density lipoprotein particle covalently linked to the molecule apolipoprotein(a). The use of Lp(a) as a risk factor for cardiovascular disease remains controversial.

Hyperhomocysteinaemia

Homocysteine is an amino-acid produced during the metabolism of methionine. Higher levels are associated with an increased risk of cardiovascular disease, and this increase in risk with higher levels is graded. Dietary and supplemental sources of folate, along with vitamins B_{12} and B_6, have been shown to reduce homocysteine levels. It is unclear whether normalising homocysteine levels reduces cardiovascular events, although one trial in people after coronary angioplasty has shown that vitamin supplementation reduces restenosis rates.[106]

Prothrombotic Factors

Several prospective studies have shown an association between fibrinogen and coronary heart disease. Smoking is a major cause of high fibrinogen levels.

Calcium Scoring and Other Techniques

Electron Beam Computerised Tomography (EBCT) and helical Computerised Tomography (CT) are sensitive techniques for detecting coronary artery calcium, which is specific for the presence of atheroma. However, EBCT is not anatomically accurate for predicting the site of a future coronary artery occlusion. At present there are only a limited number of studies assessing the positive predictive value of calcium scoring in asymptomatic people, with a small number of hard clinical end-points.[107-109] The test has a high negative predictive value and a negative result indicates a minimal risk of future cardiovascular events. There is currently insufficient evidence to recommend that these investigations are part of a risk assessment or used in screening for cardiovascular disease.

MEASUREMENT OF RISK FACTORS

Lipids

A fasting lipid profile (total cholesterol, LDL-C, HDL-C, TC:HDL ratio and triglycerides) should be taken. A single TC:HDL ratio is used to calculate cardiovascular risk. Two lipid measurements should be taken prior to initiating drug treatment or intensive lifestyle treatment. If the total cholesterol level varies more than 0.8 to 1 mmol/L in the two samples, a third sample should be taken and the average of the three samples should be used as the baseline measure. A fasting sample is required for the measurement of triglycerides.

Blood Pressure

The average of two sitting blood pressure measurements is recommended at the initial risk assessment. This should be repeated on three separate occasions to obtain a baseline prior to the initiation of either intensive lifestyle modification or drug treatment.

Diabetes

Fasting plasma glucose is recommended for the initial risk assessment. The diagnosis of diabetes or IFG should be confirmed with either a second fasting glucose or a 75 g oral glucose tolerance test (OGTT) as indicated. An OGTT is recommended in people with a fasting glucose of 6.1 to 6.9 mmol/L. An OGTT is also recommended in people with a fasting glucose of 5.5 to 6 mmol/L who are not of European ethnicity or who have a family history of diabetes, a past history of gestational diabetes or the features of the metabolic syndrome (see Table 24). An HbA1c measurement and an early morning urine sample for the urinary albumin:creatinine ratio is recommended for everyone with diabetes to help determine their cardiovascular risk.

Obesity

Waist circumference (in cm) should be part of a cardiovascular risk assessment. BMI (weight in kg/height in m^2) should also be recorded.

Smoking Status

Current and past smoking habits should be recorded. A non-smoker is defined as someone who has never smoked or has not smoked for more than 12 months.

Atrial Fibrillation

The presence or absence of atrial fibrillation will be suspected after detecting an irregular pulse or irregular heart rhythm and an ECG should be performed to confirm atrial fibrillation.

HOW TO DETERMINE CARDIOVASCULAR RISK

Cardiovascular Risk Determined Clinically (previously NHF Group A)

The following groups defined by clinical history alone are considered at very high risk or potentially at very high risk (5-year cardiovascular risk greater than 20%):

- **People with a previous history of cardiovascular disease**

  People who have had a previous cardiovascular event (angina, myocardial infarction, angioplasty, coronary artery bypass grafts, transient ischaemic attack, ischaemic stroke or peripheral vascular disease)

- **People with specific genetic lipid disorders (FH, FDB and FCH)**

  People with the following genetic lipid disorders should be referred for specialist assessment, advice on management, and family tracing:

  - **Familial hypercholesterolaemia (FH)**

    People with FH usually have a family history of premature coronary heart disease compatible with autosomal dominant inheritance. Heterozygous FH has a prevalence in the general

population of at least 1 in 500. The relative risk for developing coronary heart disease in people with FH is approximately nine times greater than that of the general population. Over 50% of men with untreated FH have a fatal or non-fatal myocardial infarction by the age of 50 years. Children with an affected parent have a 50% chance of having FH.

Family tracing of the siblings and children of people with FH is recommended. People presenting with total cholesterol levels greater than 8 mmol/L plus a family history of premature coronary heart disease, or tendon xanthelasma should also be offered family tracing. They should be referred to a centre with expertise in management of lipid problems as mutation analysis can only be undertaken by specialist services. Mutation analysis is possible in about 40% of people with FH and if a mutation is detected this allows more precise family tracing/screening. If referral is not possible advice should be sought from an appropriate specialist.

- **Familial defective ApoB: (FDB)**

  People with elevated ApoB levels are generally identified, managed and referred as for people with FH.

- **Familial combined dyslipidaemia: (FCH)**

  This is characterised by a strong family history of cardiovascular disease and a combined dyslipidaemia high LDL-C, high triglycerides and usually a low HDL-C with small dense LDL-C particles.

- **People with diabetes who have overt diabetic nephropathy or diabetes with other renal disease**

  People with diabetes who have overt diabetic nephropathy (albumin:creatinine ratio greater than 30 mg/mmol) or diabetes with other renal disease have a very high risk of cardiovascular disease (5-year cardiovascular risk greater than 20%).

  Overt diabetic nephropathy is defined by a raised urinary albumin excretion greater than or equal to 300 mg/day (this is equivalent to an urinary albumin:creatinine ratio ≥30 mg/mmol, or urinary albumin concentration >200 mg/L). This represents a more severe and established form of diabetic nephropathy and is more predictive of total mortality, cardiovascular mortality, cardiovascular morbidity and end-stage renal failure than microalbuminuria. When there is overt diabetic nephropathy and blood pressure is over 140/90 mm Hg, the standardised mortality ratio (SMR) is increased 11 to 18-fold in people with type 1 diabetes and 2 to 8-fold in people with type 2 diabetes.[110,111] **(2++)**

Where Cardiovascular Risk is Determined from the Framingham Risk Equation

All other men and women in the appropriate age bands should have their absolute cardiovascular risk calculated using the National Heart Foundation's cardiovascular risk tables or an electronic decision support tool based on the Framingham risk equation for first cardiovascular events. Young people, under the age of 35 years, with a strong family history of cardiovascular disease may be difficult to risk stratify and will need specific and individual assessment.

A family history of premature coronary heart disease or ischaemic stroke is positive for people with a first degree male relative (father or brother) developing clinically proven cardiovascular disease (angina, myocardial infarction, transient ischaemic attack, or ischaemic stroke) before the age of 55 years or a first degree female relative (mother or sister) developing clinically proven cardiovascular disease (angina, myocardial infarction, transient ischaemic attack, or ischaemic stroke) before the age of 65 years.

Adjustments to Cardiovascular Risk Calculations

People in the following groups should be moved up one risk category (5%) as their cardiovascular risk may be underestimated in the Framingham risk equation:(**4**)

- people with a strong family history of proven coronary heart disease or ischaemic stroke
- Māori
- Pacific peoples
- people from the Indian subcontinent
- people with both diabetes and microalbuminuria
- people who have had type 2 diabetes for at least 10 years or who have an HbA1c consistently over 8% (where risk factor thresholds are given these should be interpreted as approximate guides to clincial practice only)
- people who meet the definition of the metabolic syndrome (see Table 24).

These adjustments should be made once only for people who have more than one criterion (the maximum adjustment is 5%).

Microalbuminuria is defined as a sustained urinary albumin excretion of between 30 and 300 mg/L per day. An adequate estimate of the daily albumin excretion is provided by the urinary albumin: creatinine ratio (ACR). Microalbuminuria is present if the ACR greater than or equal to 2.5 mg/mmol in men or greater than or equal to 3.5 mg/mmol in women. A urinary albumin concentration greater than or equal to 20 mg/L also indicates microalbuminuria. Microalbuminuria is an independent marker of cardiovascular risk in people with type 2 diabetes, and is associated with at least a doubling of cardiovascular risk.[103]

Where Risk Factor Levels are Extreme

People with elevated risk factor levels are assumed to have 5-year cardiovascular risk above 15%. They should have a risk assessment because, when all risk factors are taken into account, they may have a calculated 5-year CV risk higher than this. These people should receive specific lifestyle intervention and lipid-modifying or blood pressure-lowering treatment as appropriate.

Isolated elevated single risk factor levels are defined as:

- TC greater than 8 mmol/L
- TC:HDL ratio greater than 8
- blood pressure consistently greater than 170/100 mm Hg.

Cardiovascular Risk and Age

Assessment of the balance of risks and benefits in older people (over about 70 years) is more difficult than in younger people. Older people gain a similar relative benefit from cholesterol lowering but are more likely to benefit in absolute terms because of their much higher cardiovascular risk. Those people in good health with a reasonable life expectancy should be considered for treatment. Other older people with advanced chronological or physiological age or severe co-morbidities may not be suitable for intensive therapy. A clinical judgment should take into account the results of a risk assessment, the likely benefits and risks of treatment and the person's values.

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✓ Good Practice Point

TREATMENT DECISIONS

RECOMMENDATIONS: TREATMENT DECISIONS	
All treatment decisions should be based on an individual's 5-year absolute cardiovascular risk.	A
Everyone with risk factors should be involved in the decision-making process regarding their treatment.	C
The higher an individual's absolute risk of a cardiovascular event the more aggressive management should be.	C
Everyone with a history of a cardiovascular event and any risk factor above optimal levels should be considered for treatment to reduce their cardiovascular risk. Treatment should aim to lower the risk factors to optimal levels.	A
Everyone with isolated very high-risk factor levels, either a total cholesterol greater than 8 mmol/L or a TC:HDL ratio greater than 8 or blood pressure greater than 170/100 mm Hg should have drug treatment and specific lifestyle advice to lower risk factor levels.	C
Everyone with the specific genetic lipid disorders (familial hypercholesterolaemia, familial defective ApoB or familial combined dyslipidaemia) or diabetes with overt nephropathy should be considered for treatment to reduce their cardiovascular risk. Treatment should aim to lower the risk factors to optimal levels.	A
Everyone with cardiovascular disease, a 5-year cardiovascular risk of greater than 20%, genetic lipid disorders, diabetes or the metabolic syndrome should receive intensive lifestyle advice. Lifestyle changes that have been shown to benefit people with these risk profiles include: • dietary change (A) • smoking cessation (A) • physical activity (B).	A
Intensive dietary advice should be given in individual/group sessions with a dietition.	A
People with a 5-year cardiovascular risk greater than 20% should receive intensive lifestyle advice and drug treatment of all modifiable risk factors simultaneously.	C

RECOMMENDATIONS: TREATMENT DECISIONS (CONTINUED)	
People with a 5-year cardiovascular risk of between 15 and 20% are likely to need treatment of all modifiable risk factors. Specific lifestyle advice may be given for 3 to 6 months prior to drug treatment.	C
Among people with a 5-year cardiovascular risk greater than 15% the aim of treatment is to lower 5-year cardiovascular risk to less than 15%.	C
People with a 5-year cardiovascular risk between 10% and 20% should receive specific lifestyle advice on a healthy cardioprotective dietary pattern, physical activity and smoking cessation from their primary health care team. This advice should be followed for 3 to 6 months prior to considering drug treatment.	B
People with a 5-year cardiovascular risk of less than 15% should receive non-pharmacological approaches to treating multiple risk factors.	C
People with a 5-year cardiovascular risk of less than 10% should receive general lifestyle advice on a healthy cardioprotective dietary pattern, physical activity and smoking cessation.	B
The order in which to start interventions should take into account individual risk factor profiles, potential side effects, other concurrent illness, compliance, personal preference and cost. It is appropriate to treat multiple risk factors simultaneously.	✓

TREATMENT DECISIONS

All treatment decisions should be based on an individual's 5-year absolute cardiovascular risk. The interpretation of risk factor levels and approach to intervention according to level of cardiovascular risk is outlined below and detailed in Table 2. An individual's risk factor levels should always be interpreted within the context of their calculated cardiovascular risk.

People at Very High Risk (5-Year Cardiovascular Risk >20%) Determined Clinically with:

- **a previous history of cardiovascular disease**
- **the specific lipid disorders of a genetic basis (FH, FDB and FCH)**
- **diabetes and overt diabetic nephropathy or diabetes with other renal disease.**

People with risk greater than 20% should receive lifestyle advice and drug treatment simultaneously. The drug treatment will usually include aspirin, a statin and blood pressure lowering medication (a beta-blocker and an ACE-inhibitor after myocardial infarction). They should receive intensive lifestyle advice on a healthy cardioprotective dietary pattern, physical activity and smoking cessation, ideally in individual sessions with a dietitian, and lifestyle interventions should be continued indefinitely.

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Where Cardiovascular Risk is Determined from the Framingham Risk Equation

- **People with a 5-year cardiovascular risk greater than 20%**

  The goal for people with a risk greater than 20% is to reduce 5-year cardiovascular risk to less than 15%. This can be achieved more easily by the simultaneous reduction in several risk factors. These people should receive intensive lifestyle advice, aspirin and drug treatment to modify lipids, lower blood pressure and improve glycaemic control (for people with diabetes, IGT or IFG) simultaneously. They should receive intensive dietary advice ideally in individual sessions with a dietitian. Dietary intervention should be continued indefinitely.

- **People with a 5-year cardiovascular risk of 15 to 20%**

  The goal for people with a 15 to 20% risk is to reduce 5-year cardiovascular risk to less than 15%. This can be achieved more easily by the simultaneous reduction in several risk factors. These people should receive up to 6 months of specific lifestyle advice prior to being considered for treatment with aspirin, drug treatment to modify lipids, lower blood pressure and improve glycaemic control (for people with diabetes, IGT or IFG). This individualised lifestyle advice should be given by the primary health care team. Dietary intervention should be continued indefinitely.

- **People with extreme risk factor levels (5-year cardiovascular risk assumed to be >15%)**

  People with a total cholesterol greater than 8 mmol/L or TC:HDL ratio greater than 8 or with blood pressure greater than 170/100 mm Hg should have a cardiovascular risk assessment and receive specific lifestyle intervention, aspirin, lipid-modifying or blood pressure lowering treatment as appropriate.

- **People with a 5-year cardiovascular risk of 10 to 15%**

  Clinical judgment is required. In general, people with a 10 to 15% risk should be treated with specific individualised advice given by the primary health care team on lifestyle interventions including dietary advice on a cardioprotective dietary pattern, physical activity and smoking cessation advice.

- **People with a 5-year cardiovascular risk of less than 10%**

  People with a risk of less than 10% should receive general advice on lifestyle interventions including dietary, physical activity and smoking cessation advice.

  All individuals should be involved in the decision-making process regarding their treatment.

Table 2: Intervention according to cardiovascular risk assessment

Cardiovascular risk	Lifestyle	Drug therapy	Treatment goals	Follow-up
CVD risk clinically determined* more than 20%	Intensive lifestyle advice on a cardioprotective dietary pattern with a dietitian, physical activity and smoking cessation interventions. Lifestyle advice should be given simultaneously with drug treatment	Aspirin, if not contra-indicated, a beta blocker, statin and an ACE-inhibitor (after MI) or aspirin, statin and a new or increased dose of a blood pressure lowering agent (after stroke)	Efforts should be made to reach optimal risk factor levels	Cardiovascular risk assessments at least annually, risk factor monitoring every 3 to 6 months
CVD risk calculated more than 20%	Intensive lifestyle advice on a cardioprotective dietary pattern with a dietitian, physical activity and smoking cessation interventions. Lifestyle advice should be given simultaneously with drug treatment	Aspirin and drug treatment of all modifiable risk factors (blood pressure lowering, lipid modification and glycaemic control)	Risk factors treated to a level that will lower 5-year cardiovascular risk to less than 15% (by recalculating risk)	Cardiovascular risk assessments at least annually, risk factor monitoring every 3 to 6 months
15 to 20%	Specific individualised lifestyle advice on a cardioprotective dietary pattern, physical activity and smoking cessation. This lifestyle advice should be given by the primary health care team for 3 to 6 months prior to initiating drug treatment	Aspirin and drug treatment of all modifiable risk factors (blood pressure lowering, lipid modification and glycaemic control). Drug therapy indicated for people with extreme risk factor levels#	Risk factors treated to a level that will lower 5-year cardiovascular risk to less than 15% (by recalculating risk)	Cardiovascular risk assessments at least annually, risk factor monitoring every 3 to 6 months
10 to 15%	Specific individualised lifestyle advice on a cardioprotective dietary pattern, physical activity and smoking cessation. This lifestyle advice should be given by the primary health care team	Non-pharmacological approach to treating multiple risk factors	Lifestyle advice aimed at reducing cardiovascular risk	Further cardiovascular risk assessment in 5 years
less than 10%	General lifestyle advice on a cardioprotective dietary pattern, physical activity and smoking cessation	Non-pharmacological approach to treating multiple risk factors	Lifestyle advice aimed at reducing cardiovascular risk	Further cardiovascular risk assessment in 5 to 10 years

*People who have had a previous cardiovascular event (angina, myocardial infarction, angioplasty, coronary artery bypass grafts, transient ischaemic attacks, ischaemic stroke or peripheral vascular disease) OR people with certain genetic lipid disorders (FH, FDB ,FCH) OR people with diabetes and overt diabetic nephropathy OR people with diabetes and renal disease

People with isolated high risk factor levels either total cholesterol greater than 8 mmol/L or TC:HDL ratio greater than 8 or blood pressure greater than 170/100 mm Hg should have these risk factors treated regardless of their calculated cardiovascular risk.

INTERVENTION: CARDIOPROTECTIVE DIETARY PATTERNS

RECOMMENDATIONS: CARDIOPROTECTIVE DIETARY PATTERNS	
Dietary intervention is strongly recommended as an integral component of the management of cardiovascular risk.	A
Use behavioural and motivational strategies in education and counselling to achieve and sustain dietary change.	A
Everyone with cardiovascular disease, a 5-year cardiovascular risk of greater than 20%, genetic lipid disorders, diabetes or the metabolic syndrome should receive intensive lifestyle advice. Lifestyle changes that have been shown to benefit people with these risk profiles include: • dietary change (A) • smoking cessation (A) • physical activity (B).	A
Intensive dietary advice should be given in individual/group sessions with a dietition.	A
Everyone with a 5-year cardiovascular risk between 10 and 20% should receive specific lifestyle advice on a cardioprotective dietary pattern, physical activity and smoking cessation from their primary health care team. This advice should be followed for 3 to 6 months prior to considering drug treatment and continued for life.	A
People with a 5-year cardiovascular risk of less than 10% should receive general lifestyle advice on a cardioprotective dietary pattern, physical activity and smoking cessation.	✓
Everyone should be encouraged to adopt a cardioprotective dietary pattern that includes fruit and vegetables, whole grains, fish and/or dried peas and beans or soy products, oil, margarine spreads, nuts or seeds, very low-fat milk products, and optional small servings of lean meat or skinned poultry. This dietary pattern avoids regular consumption of foods prepared with meat or dairy fats.	A
A cardioprotective diet in people with type 2 diabetes who are overweight or obese should be tailored to promote weight loss.	B

RECOMMENDATIONS: CARDIOPROTECTIVE DIETARY PATTERNS	
Fish oil supplements, 1 g/day EPA and DHA combined, may be offered post myocardial infarction.	A
The use of antioxidant supplements is not recommended for the prevention or treatment of cardiovascular disease.	A
Individualise dietary counselling and other lifestyle changes to complement prescribed risk factor modifying pharmacological agents to assist the individual in reducing their absolute risk of cardiovascular disease.	✓

EVIDENCE STATEMENTS

- A range of dietary patterns have been shown to be cardioprotective.(**1+**)
- Cardioprotective dietary patterns are associated with lower cardiovascular morbidity and mortality in the general population at low or moderate cardiovascular risk and in people with cardiovascular disease.(**2+**,**1+**)
- Oily fish (300 g/week) and fish oil supplements (1 g/day EPA and DHA combined) have been shown to lower coronary heart disease and all-cause mortality in people with cardiovascular disease.(**1++**) Higher consumption of fish or long chain omega-3 fatty acids is associated with lower incidence of coronary heart disease and total mortality among women with type 2 diabetes.(**2+**)
- Currently there is insufficient evidence to make a recommendation for the use of antioxidant supplements for the treatment or prevention of cardiovascular disease(**1+**) or in the prevention and treatment of macrovascular and microvascular diabetes complications in type 2 diabetes.(**1+**)
- While it is accepted that low to moderate alcohol consumption can be an enjoyable positive aspect of social life, there are considerable health and social risks associated with heavy or binge drinking, particularly among young people.(**2+**) Those who abstain from drinking alcohol, or drink only infrequently, should not start drinking for health reasons.(**2+**)
- Voluntary and intentional weight loss is associated with reduced cardiovascular disease, diabetes-related and all-cause mortality in adults with type 2 diabetes.(**2+**)
- The composition of the diet will depend on the individual metabolic profile, body weight, distribution, and dietary preferences of the individual.(**4**)
- Long-term individually target-driven, intense multiple risk factor interventions and a strict low-fat diet, low in saturated faty acids, lower cardiovascular risk in adults with type 2 diabetes and microalbuminuria.(**1++**)
- Behavioural dietary counselling from a primary health care professional or qualified dietitian reduces risk in people with a 5-year cardiovascular risk greater than 10%.(**1+**)
- Effective interventions combine nutrition education with behaviourally-oriented counselling to help people acquire the skills, motivation, and support needed to alter their daily eating patterns and food preparation practices.(**1+**)
- The 5-A's provide a framework for behavioural counselling for dietary change.(**1+**)

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I No recommendation can be made because the evidence is insufficient
✓ Good Practice Point

CARDIOPROTECTIVE DIETARY PATTERNS

A range of dietary patterns have been associated with reduced cardiovascular morbidity and mortality.[112-115] Examples of cardioprotective dietary patterns include traditional Asian and Mediterranean patterns, vegetarian dietary patterns that comply with food and nutrition guidelines, and modified-Western dietary patterns.

Dietary patterns found to reduce heart attacks in individuals at very high risk are low in saturated fatty acids. The carbohydrate content is derived mainly from fruits, vegetables, dried peas and beans including soy, whole grains and other appropriately processed cereals. The dietary intake of refined sugar and white flour products is low, and any pre-prepared packaged foods have a high fibre content and are low in saturated fatty acids.[115]

These dietary patterns act via mechanisms that improve the lipid profile, lower blood pressure, reduce the risk of thrombosis, increase thrombolysis and decrease the inflammatory reponse. Cardioprotective dietary patterns with weight reduction where appropriate, improve insulin sensitivity and lower blood glucose in those with type 2 diabetes or the metabolic syndrome.

Food-based dietary statements promoted through the National Heart Foundation of New Zealand emphasise key features of the cardioprotective dietary pattern and can be used to inform and educate the individual on the components of a cardioprotective dietary pattern for the treatment and prevention of cardiovascular disease.

The National Heart Foundation of New Zealand Food-based Dietary Statements

1. Enjoy three meals each day, select from dishes that include plant foods and fish and avoid dairy fat, meat fat or deep fried foods.
2. Choose fruits and/or vegetables at every meal and most snacks.
3. Select whole grains, whole grain breads, or high-fibre breakfast cereals in place of white bread and low fibre varieties at most meals and snacks.
4. Include fish, or dried peas, beans and soy products, or a small serving of lean meat or skinned poultry, at 1 or 2 meals each day.
5. Choose low-fat milk, low-fat milk products, soy or legume products every day.
6. Use small amounts of oil, margarine, nuts or seeds.
7. Drink plenty of fluids each day, particularly water, and limit sugar-sweetened drinks and alcohol.
8. Use only small amounts of total fats and oils, sugar and salt when cooking and preparing meals, snacks or drinks. Choose ready-prepared foods low in these ingredients.
9. Mostly avoid or rarely include butter, deep-fried and fatty foods, and only occasionally choose sweet bakery products.

Table 3: Cardioprotective dietary patterns

Food Component	Daily Servings *(except as noted)*	Serving Size Examples	Notes
Vegetables and fruit			
Vegetables	At least 3 – 4 servings	½ cup cooked vegetables 1 cup raw green vegetable or salad 1 tomato or carrot	Include at every meal Choose coloured varieties daily, especially the green, orange and red vegetables. Also includes cauliflower, onions, mushrooms and turnips
Fruit	At least 3 – 4 servings	1 medium apple, pear, orange, nectarine, small banana ½ cup stewed, frozen or canned fruit in natural or 'lite' juice 2 – 3 small apricots or plums 10 – 15 grapes, cherries, strawberries 1 cup other berries 3 prunes, dates, figs 1 tbsp raisins, sultanas 6 – 8 halves of dried apricots 180 ml 100% fruit juice	No more than one serving of fruit juice per day
Breads, cereals, grains and starchy vegetables			
Breads, cereals and grains	At least 6 servings	1 medium slice of whole grain bread or ½ bread roll 30 g of other breads such as pita, naan, corn tortilla, wraps ½ cup bran cereal or ⅔ cup wheat cereal or ½ cup cooked porridge or ⅓ cup muesli or 3 crispbreads ½ cup cooked pasta or ⅓ cup cooked rice	Choose more or less depending on body weight and level of physical activity Include at every meal Choose a variety of grain products with at least half as whole grain products
Starchy vegetables		1 small potato ½ kumara ⅓ cup yams ½ cup corn ½ parsnip 1 small round of taro	These vegetables replace breads or grain products in a meal Limit for weight control and diabetes control

Food Component	Daily Servings *(except as noted)*	Serving Size Examples	Notes
Milk and milk products			
Low-fat or fat-free milk products	2 – 3 servings or replace with soy products	1 glass trim or low-fat milk (250 ml) 1 pottle low-fat yoghurt ⅓ cup cottage cheese ½ cup low-fat cottage cheese ¼ cup quark or ricotta 2 tbsp parmesan cheese or 3 tbsp grated cheddar cheese 2 cm cube cheddar cheese 3 cm cube standard camembert, brie, edam, feta, mozzarella	Use 0 to 0.5% fat milk and <1% fat yoghurt Hard cheese and semi-soft cheeses can be included up to 4 times weekly in very small amounts
Fish and seafood, dried peas and beans, soy products, skinned poultry, lean meat and eggs			
Choose 1 – 3 servings per day from this group of foods depending on body weight and level of physical activity.			
Fish, seafood	1 – 2 servings weekly	2 small, 1 large fillet of cooked fish ½ cup tuna 1 cup mussels ⅓ cup salmon or ½ can sardines	If eating fish, choose some oily fish species such as tuna, kahawai, trevally, kingfish, warehou, dory, salmon, sardines, eel, squid, mussels or oysters
Dried peas, beans, soy products (legumes)	4 – 5 servings weekly	1 cup cooked dried beans, chickpeas, lentils, dahl ½ cup tofu or tempeh 1 glass fortified soy milk (250 ml)	
Skinned chicken or very lean meats	Limit to 1 to 1½ servings	2 slices trimmed meat/chicken (100 – 120 g) ½ cup lean mince or casserole (125 g) 1 small lean steak (100 g) 1 small chicken breast (120 g) 2 small drumsticks or 1 leg, skinned	Use alternatives to meat several times a week.
Eggs	3 eggs weekly	1 egg	

Food Component	Daily Servings *(except as noted)*	Serving Size Examples	Notes
Oils, spreads, nuts, seeds and avocado			
Liquid oils, unsaturated margarines and spreads (including sterol-fortified spreads), or avocado	3 or more servings	1 tsp soft table margarine or oil 2 tsp light margarine (50 – 60% fat) 2 tsp mayonnaise or vinaigrette (50 – 60% fat) 3 tbsp reduced-fat mayonnaise or dressing (10% fat or less) 1 tbsp avocado	Choose more or less depending on body weight and level of physical activity Choose products made from sunflower, soya bean, olive, canola, linseed, safflower or nuts and seeds, other than coconut Percent fat describes fat per100 g on product label Include spreads with plant sterols if LDL-C is high
Nuts, seeds	Eat regularly up to 30 g/day	1 dsp nuts or pumpkin seeds 1 dsp peanut butter 1 tbsp sunflower or sesame seeds	For weight control 1 serving of nuts replaces other oils and spreads
Sweets and added sugar			
Confection-ery and added sugar	Up to 1 for weight control for those with high triglycerides/ diabetes as part of a meal or snack. Or up to 3 /day if lean and active normal triglycerides and no diabetes	1 tbsp sugar, jam, syrup or honey 2 tbsp all-fruit jam spreads 6 jet planes 180 ml fruit juice or softdrink Small pottle reduced-fat ice-cream or frozen yoghurt 2 fruit slice biscuits	Best incorporated as part of the meal or snack only if diabetes is well controlled Choose more or less depending on body weight and level of physical activity Artificial sweeteners may be used for additional sweetness as a replacement for sugar

Food Component	Daily Servings *(except as noted)*	Serving Size Examples	Notes
Salt and salty foods			
Minimise added Salt	Limit high salt seasonings to 1/day	1 tsp seasoning paste ⅙ stock cube ⅛ tsp stock powder ⅓ tsp gravy mix 1 tbsp liquid seasoning	Use minimal salt in cooking. Do not add salt to meals
Limit high salt foods	Limit these high salt foods to less than 4 servings/day	30 g lean ham/pastrami 1 tbsp pickles 1 tsp marmite/vegemite 1 tsp soy sauce 20 to 30 g cheese ½ cup canned/packet soup 50 g canned or smoked salmon/tuna 30 g other smoked fish/sardines	Choose breads and cereals with less than 450 mg/100 g sodium and spreads with less than 400 mg/100 g sodium Choose low or reduced-salt/sodium canned foods, soups, sauces seasonings, crispbreads, relishes and meals Check labels of cured, corned, pickled, smoked, marinated and canned foods
Drinks			
Alcoholic drinks	Limit to 3 or less drinks for men and 2 or less for women	1 (300 ml) glass ordinary strength beer 1 (60 ml) glass fortified wine (sherry, martini, port) 1 (30 ml) pub measure spirits (whisky, gin, vodka) 1 (100 ml) glass of table wine	
Non-alcoholic drinks	6 – 8 drinks /day	1 glass water (250 ml) 1 cup 'diet' soft drink (180 ml) 1 glass trim or low-fat milk (250 ml) 1 cup tea, coffee or cocoa 1 cup vegetable juices (180 ml)	Drink plenty of water every day Low-fat milk is a nutritious alternative to water. Limit the consumption of fruit juice, cordial and fizzy drinks because of their high sugar content.

Table 4: Including carbohydrate in the cardioprotective dietary pattern

Dietary Fibre (note 1)

Recommendations

- Hyperlipidaemia – 30 g or more per day
- Diabetes/Metabolic Syndrome – 40 g or more per day

Cardioprotective carbohydrate foods	Servings per day (note 2)	Average fibre per serving	Foods with higher than average fibre per serving
Vegetables (note 3)	4 – 5+	1.5 – 3 g	Green peas, larger servings of vegetables
Fruit	3 – 4+	2 – 3 g	Raspberries, blackberries, oranges
Whole grain breads or cereals (note 3)	4 – 6+	2 – 3 g	Heavy seed breads, bran cereals, oat/rice bran
Dried peas and beans	½ serving = ½ cup	4 – 6 g per ½ serving	Black beans, red kidney beans

Added Sugar (note 4)

Recommendations

- Hyperlipidaemia – Up to 3 servings (45 g) if lean and active
- 1 serving (15 g) for weight loss, diabetes, high triglycerides

Sugar containing foods	Non-fat sources of sugars = 1 serving	Low-fat sources of sugars = 1 – 2 servings
1 serving = tablespoon or 15 g of sugars	1 tablespoon honey, regular jam Brown/white sugar, syrup 2 tablespoons all-fruit/reduced sugar jams 3 – 4 hard lollies/7 – 10 gum lollies 180 ml juice/150 ml soft drink 120 ml energy drink/Ribena™/ blackcurrant/cranberry juice 1 iceblock/Popsicle™	'Low-fat' fruit bars 'Low-fat' cereal bars 2 Weight Watcher™ biscuits or round wine biscuits 2 Snack Right Fruit Biscuits™ 4 toffees, 3 sprats Weight Watcher™ muffin Reduced fat fruit yoghurt Reduced fat frozen yoghurt Reduced fat frozen dessert

GI (notes 1,5,6)

Recommendations

- Diabetes/Metabolic Syndrome – Most servings moderate or low GI and high-fibre and at least one low GI/high-fibre serving at each meal

	Low GI <55	Moderate GI 56 – 69	High GI >70
High-fibre choices >1.5 g fibre/ serving	All-Bran™, oat porridge, natural muesli, oat bran, rice bran, pearled barley, bulghur (cracked) wheat.	Oat bran, Weet-Bix™	Litebix™
		Instant porridge	Mini Wheats™ blackcurrant
		Vita-Brits™	Sultana Bran™
		Just Right™	Puffed wheat
	Breads with high content of whole grain, seeds AND 5+ g fibre/100 g bread, eg,whole grain, mixed grain, whole grain rye, oat bran, sunflower/ barley soy/linseed, pumpernickel 9-grain + breads, wholemeal pasta	Fruitful Lite™	Molenburg™
		Mini Wheats™ whole wheat	Grain breads with high white flour content
		Vita-wheat™ crispbread	Ryvita™ crispbread
		Other wholemeal bread	
	Apples, pears, oranges, grapefruit, berry and stone fruits, kiwi, mango, prunes, dried apricots, under-ripe banana (yellow skin)	Melons, pineapple, raisins, sultanas, very ripe bananas	Dates
			Most potatoes, parsnip, kumara, broad beans, mashed potato
	Baked beans, other dried peas/beans, sweet corn, yams, peas	Small canned potatoes, taro (1.3 fibre)	
Low fibre choices <1 g fibre/ serving	Special K™, Frosties™	Nutragrain™	Cornflakes™, Rice Bubbles™
	White wheat pasta, sushi Koshikari short grain rice, some varieties long grain rice (eg, Uncle Bens™)	Pita bread, hamburger bun	White bread, Fibre White™, bagels, wheatmeal bread, baguette, white buns
		Basmati/doongara rice, most long grain rice, rice pasta/noodles	Corn thins, rice cakes, rice crackers
	Fruit breads, grapes	Udon noodles	Jasmine Calrose rice, long cooked white rice, sticky/ glutinous rice
	Juices – apple, orange, grapefruit and vegetable	Couscous	
		Rice pudding	Golden Fruit™ biscuits, pikelets, scones, water crackers
	Snack Right Fruit Slice™	Plain popcorn	
	Milo™		

Notes

1. Some carbohydrate-containing foods are not included in the fibre and glycaemic index (GI) lists. Milk products, nuts and seeds contain low GI carbohydrate, but these foods are selected for their protein and fat content rather than their carbohydrate content. Snack foods, confectionery, biscuits, desserts, cakes and other high-fat or high sugar items have other nutritional reasons for their restriction, irrespective of their GI or fibre content.
2. One serving of carbohydrate-rich food on Table 3 (grain product, starchy vegetable, fruit or dried peas and beans) contains approximately 15 g of carbohydrate.
3. Vegetables rich in starch – potatoes, kumara, yams (1 – 2 g fibre), taro (0.6 g), parsnip, and corn (2 – 3 g) – yellow-white vegetables – are not included in the vegetable group. Grain products are also rich in starch and the number of servings depends on energy needs. More active, lean individuals may need 12 or more servings of grain and starch-rich vegetables per day.
4. 'Added' sugars do not refer to sugars naturally occurring in fruits. 'Added' sugars have been defined as 'free sugars' by the WHO/FAO. 'Free sugars' refers to all monosaccharides and disaccharides added to foods by the manufacturer, cook or consumer plus sugars naturally occurring in honey, syrups and fruit juices.[117]
5. GI measures how quickly the carbohydrate in a food raises blood sugar levels. A food that is digested quickly has a high GI, a food that breaks down slowly has a low GI. GI is relevant only to carbohydrate-rich foods, and not to carbohydrate-free foods such as meats, poultry and fish. GI lists are confined to food products tested under physiological conditions. Tested trade-marked foods are identified since similar products may not have the same GI.
6. Reference for GI values:[116]

Table 5: Changing the ratio of unsaturated:saturated fatty acids

Recommendation	Best Choices		Mostly Avoid
Rich sources of specific fatty acids	Predominantly polyunsaturated	Predominantly monounsaturated	Predominantly saturated +/or high content transunsaturated
Fats & Oils	Safflower oil* Wheatgerm oil* Soy oil*/# Sesame oil* Sunflower oil* Grapeseed oil* Linseed or flaxseed oil*/# Fish oil capsules#	Olive oil (Extra Virgin) Canola (Rapeseed) oil# Peanut oil Avocado oil Almond oil	Lard, Suet Dripping, beef fat Palm oil/palm kernel oil Coconut oil 'Hydrogenated' oils - baking margarine Hard white block fats, eg, Chefade™, Kremelta™
Spreads	Polyunsaturated spreads (18 g – PUFA/100 g) products with sunflower* or sunflower* + canola oils*/# Phytosterol enriched, Pro-activ™/Lite Pro-activ™*	Monounsaturated spreads (25 g – MUFA/100 g) products with canola#, olive or avocado oils Phytostanol enriched, Logicol™	Butter Lite butter Butter blends Semi-soft butter Shortenings Foods or spreads made with any of the fats in this column
Foods	Walnuts*/# Brazil nuts* Pine nuts* Pumpkin seeds* Sunflower seeds* Sesame seeds* Linseeds*/# Oily fish#/†	Peanuts Hazelnuts Almonds Cashews Pistachios Avocados Olives Pecans Macadamias Brazil nuts* Oily fish#/†	Coconut Coconut cream Cocoa butter (chocolate) Reduced or sour cream Cream or double cream cheese Visible white meat fat Paté Chicken skin, chicken fat

Notes

* Rich sources of omega-6 polyunsaturated fatty acids (PUFA). Products richer in omega-6 PUFA reduce LDL-C and triglyceride levels more than other oils and a P:S ratio of 1:1 or more reduces cardiovascular risk. Exclusive use of olive oil products does not ensure a good polyunsaturated fat intake.

\# Good sources of omega-3 fatty acids.

† Cooked or canned oily fish are spreadable and a useful way to increase omega-3 intakes.

The Dietary Approach

Behavioural dietary counselling is recommended for all people with a 5-year cardiovascular risk greater than 10%.[118](**1+**) However, there is insufficient evidence to recommend for or against routine behavioural counselling to promote a healthy diet in people with a 5-year cardiovascular risk less than 10% in primary care settings.[118]

Several brief dietary assessment questionnaires have been validated for use in the primary care setting. These instruments can identify dietary needs, guide interventions, and monitor changes in dietary patterns. These instruments are susceptible to bias from self-reporting of dietary intake. When used to evaluate the efficacy of counselling, efforts to verify self-reported information are recommended.[119](**1+**)

Effective interventions combine nutrition education with behaviourally-oriented counselling to help people acquire the skills, motivation, and support needed to alter their daily eating patterns and food preparation practices.[119](**1+**) Examples of behaviourally-oriented counselling interventions include teaching self-monitoring, training to overcome common barriers to selecting a healthy diet, helping people to set their own goals, providing guidance in shopping and food preparation, role playing, and arranging for intra-treatment social support. In general, these interventions can be described with reference to the 5-A behavioural counselling framework:[120](**1+**)

- **Assess** dietary practices and related risk factors
- **Advise** to change dietary practices
- **Agree** on individual diet-change goals
- **Assist** to change dietary practices or address motivational barriers
- **Arrange** regular follow-up and support or refer to more intensive nutritional counselling if needed.

Two approaches appear promising for adults in primary care settings:

1. Medium-intensity face-to-face dietary counselling (2 – 3 group or individual sessions) delivered by a specially-trained primary care doctor or practice nurse or a dietitian or qualified nutritionist.
2. Lower-intensity interventions that involve 5 minutes or less of primary care provider counselling supplemented by self-help materials, telephone counselling, or other interactive health communications.

DIETARY INTERVENTIONS THAT REDUCE MORBIDITY AND MORTALITY

Traditional dietary approaches to reducing cardiovascular disease have centred on the modification or reduction of dietary fat and cholesterol. Intervention trials of dietary fat with clinical endpoints have shown equivocal results with trials of at least 2 years follow-up providing some evidence for protection from cardiovascular events.[121](**1-**)

More recently dietary approaches have focused on the identification of dietary patterns that reduce risk for cardiovascular disease.[122](**2+**) This approach acknowledges the importance of reducing or modifying dietary fat intake but includes this within a whole-diet approach to cardiovascular risk reduction.[121,123,124] A range of dietary patterns exist that can lower modifiable risk factors and protect against cardiovascular disease and all-cause mortality.[112-115,125](**2+**,**1+**) Intervention trials replicating these dietary patterns post-myocardial infarction reduce cardiac death and non-fatal myocardial infarction (ARR=10.0%, NNT=10 over 3.75 years).[112](**1+**) Food-based dietary statements promoted through the National Heart Foundation of New Zealand are supported by a robust evidence base.[115,126]

Dietary supplements show equivocal results in reducing mortality from cardiovascular disease. At this time the evidence does not support the use of vitamin E (alpha-tocopherol) and vitamin C supplements using the doses that have been tested in low and high-risk groups.[127,128](**1+**) Beta-carotene may be harmful in some people, particularly heavy smokers.[129](**1+**)

EPA and DHA are long-chain omega-3 PUFA that are derived principally from fish and fish oils. Dietary supplements of EPA and DHA combined (1 g/day) have been shown to lower cardiovascular and all-cause mortality in people with cardiovascular disease.[123,130](**1++**) Meta-analysis of trials of omega-3 PUFA intake have reported that this reduces myocardial infarction (ARR=1.48%, NNT=68 over 1.67 years), sudden death (ARR=0.4%, NNT=111 over 1.67 years) and all-cause mortality (ARR=1.8%, NNT=56 over 1.67 years).[123](**1++**)

It remains to be determined if EPA and DHA supplements are more beneficial than eating fish. Poor regulation of the dietary supplements industry in New Zealand means the quality and consistency of supplements may vary. Dietary advice to eat at least 300 g/week of oily fish can reduce coronary heart disease death (ARR=4.0%, NNT=27 over 2 years) and all-cause mortality in people post-myocardial infarction (ARR=2.0%, NNT=50 over 2 years).[125](**1+**) Fish and seafood also provide valuable nutrition (animal protein low in saturated fatty acids, calcium from soft bone and fat soluble vitamins) in addition to omega-3 PUFA. These foods are frequently consumed among Māori and Pacific people and may enhance compliance with other dietary advice.[125](**4**)

Heavy drinking of alcohol is associated with an increased risk of cardiovascular disease, and there are social risks associated with heavy or binge drinking, particularly among young people. A low to moderate intake of alcohol is associated with protection from coronary heart disease, and can be an enjoyable, positive aspect of social life.[126,131-134](**2+**)

The importance of the absolute risk of cardiovascular disease must be stressed when advice regarding alcohol consumption is given. For those at low absolute risk, that is, men below the age of 45 years, and women below the age of 55 years, there is no advantage in consuming alcohol.[126,135] Conditions in which alcohol should not be used at all include: pregnancy; cirrhosis of the liver; uncontrolled hypertension; congestive heart failure; previous haemorrhagic stroke; pancreatitis; and those at high risk of breast cancer.

Table 6: Safe drinking guidelines

Drinking Occasion	Men	Women
If drinking every day, drink no more than:	3 standard drinks	2 standard drinks
On any one drinking occasion, drink no more than:	6 standard drinks	4 standard drinks
In any one week, drink no more than:	21 standard drinks	14 standard drinks

Source: Alcohol Liquor Advisory Council, 2003.[136]

Interventions that Reduce Cardiovascular Mortality and Morbidity in People with Diabetes

Intensive lifestyle advice and treatment for individual risk factors reduces cardiovascular morbidity and mortality in people with type 2 diabetes.[149-153](**1+**) In high-risk individuals with diabetes and microalbuminuria, a pharmacological and lifestyle intervention, intensified in order to meet strict treatment targets, has been shown to reduce the rates of progression to microvascular complications and cardiovascular events by 50% compared with a conventional risk-factor treatment and standard treatment goals. This included a strict low-fat diet, low in saturated fatty acids.[137](**1++**)

Moderate weight reduction has been associated with an improvement in the co-morbidities associated with type 2 diabetes. In the American Cancer Society's *Cancer Prevention Study*, voluntary weight losses of 9 to 27 kg were associated with a 25% reduction in total mortality in participants with type 2 diabetes.[138](**2+**)

Large-scale trials of vitamin E (400 IU) supplementation[139-141](**1+**) or a vitamin E, vitamin C and beta-carotene supplement,[128](**1+**) found no effects on cardiovascular outcomes in individuals with diabetes. Vitamin E supplementation has shown no effects on nephropathy, need for retinal laser therapy or diabetes control.[139,142](**1+**)

In women with diabetes, fish consumption 2 to 4 times per week or 5 times per week has been associated with 36% and 64% reduced risk of coronary heart disease respectively. A trend for an inverse association between long-chain omega-3 PUFA consumption and incidence of coronary heart disease and total mortality was also observed.[143](**2+**) In men with type 2 diabetes a higher dietary ratio of polyunsturated fat to saturated fatty acids (P:S ratio >0.28) is associated with lower rates of coronary heart disease death.[144](**2+**)

Prospective cohort studies indicate that mild to moderate alcohol consumption is associated with 30 to 80% reduced risk of coronary heart disease among adults with type 2 diabetes. The lowest adjusted risk was associated with average daily alcohol consumption of 5 to 15 g in women and 14 to 28 g (or more) in men, compared with no alcohol consumption.[145-148](**2+**) These levels support a recommendation that safe intakes for people with type 2 diabetes are the same or slightly lower than intakes recommended for people without diabetes.

Risk reduction begins with 2 g of alcohol per day in type 2 diabetes.[146] An assessment of absolute risk of cardiovascular disease in terms of the influence of alcohol on each independent risk factor is a priority when providing advice concerning alcohol intake. Due to the low numbers of study participants consuming the highest levels of alcohol intake, intake levels associated with risk in type 2 diabetes remain undetermined. Moderate alcohol intakes in people with diabetes may not be of benefit in reducing blood pressure or microalbuminuria and there is no evidence that quantifies the risk of alcohol consumption in people with both coronary heart disease and type 2 diabetes.

Intensive lifestyle advice and treatment for individual risk factors reduces cardiovascular morbidity and mortality in people with diabetes.[149-153](**1+**) One randomized controlled trial shows long-term intense multiple risk factor interventions in people with type 2 diabetes and microalbuminuria compared with standard treatment lowers cardiovascular risk by 50%.[137](**1++**) Recent studies show that dietary modification and enhanced physical activity can delay or prevent the transition from IGT to type 2 diabetes. Metformin also had a significant though less strong effect in preventing the progression from IGT to type 2 diabetes.[154,155](**1++**)

DIETARY AND DRUG THERAPY

EVIDENCE STATEMENTS

- Randomised controlled trials have demonstrated that a cardioprotective diet improves lipid profiles.(**1+**)
- Dietary treatment is additive to drug therapy and can reduce cholesterol by an additional 5 to 15%. Advice on dietary patterns is integral to reducing cardiovascular risk.(**1+**)
- A small degree of weight loss (5 –10%) in people who are overweight or obese with elevated blood pressure and at low cardiovascular risk, can result in a step-down of certain blood pressure medications.(**1+**)

Dietary treatment includes the promotion of cardioprotective dietary patterns that offer more than simple LDL-C reduction. These dietary patterns act via mechanisms that improve the lipid profile, lower blood pressure, and reduce the risk of clotting. These patterns, with weight reduction where appropriate, improve insulin sensitivity and lower blood glucose in those with elevated glucose levels.

Dietary intervention is strongly recommended as an integral component in the management of cardiovascular risk. Attempts to lower risk without investigating dietary habits and weight history may result in the need to use larger doses of medications or combinations of medications due to inappropriate food choices and sedentary behaviours. This contributes to the increased risk of side effects and adverse drug interactions.[156,157]

Dietary therapy has an independent and additive effect on reducing cardiovascular risk. Cardioprotective dietary patterns, with and without plant sterol-fortified spreads, can lower LDL-C a further 5 to 15% in people treated with statins.[158-167](**1+**) This is at least equivalent to doubling the dose of the statin.[61] Cardioprotective dietary patterns can also reduce the daily dosage of certain blood pressure medications.[168](**1+**) A small degree of weight loss (5 to 10%) in people who are overweight or obese with elevated blood pressure and at low cardiovascular risk, can result in a step-down of certain blood pressure medications.[169,170](**1+**)

POPULATION HEALTH APPROACH

It is recognised that two principal strategies are needed to achieve improvements in the food environment. Firstly, a strategy aimed at finding and modifying the dietary habits of people at high-risk of cardiovascular disease and secondly, one aimed at the whole population. If successful, they both reduce the cardiovascular risk of the current generation of adults. In addition, a population strategy has the potential to reduce the risk of subsequent generations. Regardless, the scope for individual and population change within the primary health care setting is substantial. The population approach is outlined in a recently published national framework designed to lower risk in the whole population by integrating nutrition, physical activity and obesity.[171] *The Assessment and Management of Cardiovascular Risk* guideline seeks to define the evidence for personal health advice.

KEY - *Grades indicate the strength of the supporting evidence not the importance of the recommendations - see page xvii for details*

A Recommendation is supported by good evidence
B Recommendation is supported by fair evidence
C Recommendation is supported by non-analytic studies or consistent expert opinion
I No recommendation can be made because the evidence is insufficient
✓ Good Practice Point

INTERVENTION: PHYSICAL ACTIVITY

RECOMMENDATIONS: PHYSICAL ACTIVITY	
Everyone should aim to do a minimum of 30 minutes of moderate-intensity physical activity (3 to 6 METs) on most days of the week.	B
For people with time constraints this physical activity may be accumulated in bouts of 8 to 10 minutes.	B
People who are already doing 30 minutes of moderate-intensity physical activity per day should be encouraged to do physical activity of higher intensity or for longer to increase the beneficial effect by further improving their cardiorespiratory fitness.	B
Physical activity is an integral part of the lifestyle advice for people with increased cardiovascular risk.	B
Individuals with a history of cardiovascular disease should consult their doctor before they undertake vigorous physical activity. Vigorous activity is generally not encouraged in people with impaired left ventricular function, severe coronary artery disease, recent myocardial infarction, significant ventricular arrhythmias or stenotic valve disease.	B
Physical activity for people with coronary heart disease should begin at a low intensity and gradually increase over several weeks.	C

ASSESSMENT OF PHYSICAL ACTIVITY

An assessment of physical activity should include the following aspects:

- duration
- frequency
- intensity
- type.

The metabolic equivalent (MET) scale is a convenient tool for quantifying the relative energy expenditure and thus intensity of typical leisure time activities and activities of daily living. One MET is the typical resting energy expenditure equivalent to oxygen consumption (3.5 ml O_2/kg/min).[172] An individual exercising at 2 METs is consuming oxygen at twice the resting rate.

'Exercise' is a subset of physical activity and is more formal and exertional in nature. It is planned, structured and repetitive bodily movement performed to improve or maintain one or more components of physical fitness. Exercise is often performed to achieve objectives such as improved fitness, performance and health, and can provide a means of social interaction. The preferred term 'physical activity' is used in this guideline to encompass all grades of activity.

'Moderate physical activity' is defined as activities with energy expenditure of 3 to 6 METs. People who perform activities of this intensity for 30 minutes per day will meet the recommendations for cardiovascular benefit.

'Vigorous physical activity' is defined as activities with energy expenditures of greater than 7 METs.[172] It is suggested that people who are already doing moderate-intensity physical activity on most days of the week begin to include these types of activity into the daily routine. Increasing cardiorespiratory fitness has additional benefits.

Cardiorespiratory fitness is defined as the ability of the circulation and respiration to supply oxygen during sustained physical activity.

*Table 7: Metabolic equivalents (METs) for selected activities**

For Leisure Activities		METS (Min)	METS (Max)
Aerobics		6	9
Cycling	8 km per hour	2	3
	16 km per hour	5	6
	21 km per hour	8	9
Dancing	Ballroom	4	5
Gardening	Mowing lawn (pushing)	3	6
	Weeding/cultivating	4	5
Music	Playing an instrument	2.5	4
Running	General light jogging	6	8
	Training 10 km per hour	9	11
Skipping	<80 per minute	8	10
	120 – 140 per minute	11	11
Swimming	Breast stroke	8	9
	Freestyle	9	10
Tennis		4	9
Walking	1 – 3 km per hour	1	3
	3 – 6 km per hour	3	6

For Tasks of Daily Living	METS (Min)	METS (Max)
Bed making	2	6
Carrying heavy groceries	5	7
Cleaning windows	3	4
Cooking	2	3
Dressing	2	3
Driving a car	1	2
Eating	1	2
General housework	3	4
Grocery shopping	2	4
Loading/unloading washing machine	4	5
Lying awake	1	2
Mowing by hand	5	7
Painting/decorating	4	5
Sexual intercourse	3	5
Showering	3	4
Vacuuming	3	3.5
Walking up stairs	4	7
Washing a car	6	7
Washing dishes	2	3
Watching television	1	2

*Adapted from Ainsworth, 2002.

One MET equals oxygen consumption at rest which is about 3.5 ml O_2/kg/min. An individual exercising at 2 METs is consuming oxygen at twice the resting rate.

BENEFITS OF PHYSICAL ACTIVITY INTERVENTIONS

Regular physical activity is associated with reduced risk of cardiovascular disease morbidity and mortality. Sedentary occupations compared with active occupations have almost a doubled risk of coronary heart disease (RR 1.9, 95% CI, 1.6 – 2.2).[94] This observation is consistent over a range of intensities and frequencies with the more intense and frequent activity conferring greater protection (a dose-related effect).[95,96](**2++**) The protective effect of physical activity is greatest in individuals at higher risk of cardiovascular disease.[97](**1++**) Limited evidence from small randomized controlled trials show that the improvements in cardiorespiratory fitness, lipid profiles and blood pressure from exercise regimes comprised of several short sessions per day are as effective as those comprising longer continuous sessions.[173](**1+**)

Physical activity has been shown to result in favourable changes in blood lipid profiles. The most consistent finding is an increase in HDL-C seen in both men and women. More vigorous activity has been shown to decrease LDL-C.[174-176](**1+**) Moderate or vigorous-intensity physical activity has been shown to reduce systolic and diastolic blood pressure. Meta-analyses have shown a reduction in both systolic and diastolic blood pressure of about 5/3 mm Hg with moderate physical activity.[177-180] The effect is more pronounced in individuals with higher baseline blood pressure levels.[177,178,180](**1++**) Long-term (longer than 4 years) regular physical activity is associated with a reduced risk of developing type 2 diabetes.[181-183](**2+**) This protective effect of physical activity is greatest in people at high risk of developing diabetes and those with a family history of diabetes.[184](**2++**) In people with IGT, lifestyle interventions that include physical activity reduce the risk of developing type 2 diabetes.[154,155](**1++**)

Weight loss can be increased when physical activity is combined with a cardioprotective dietary pattern and reduced energy intake.[185](**1+**) In those who are inactive, an increase in physical activity can promote weight loss, by reducing total body fat, and can assist in weight maintenance.[186,187](**1++**) In general, diet is more effective for weight loss than physical activity. A meta-analysis investigating the effect of diet alone, exercise alone, or diet and exercise together in reducing weight has shown that diet and exercise together, and diet alone, are significantly more effective in reducing weight than exercise alone.[185]Although diet is more effective for producing weight loss, evidence suggests that physical activity may be the single best predictor of weight loss maintenance.[186,187]

RISKS OF PHYSICAL ACTIVITY

There is a small, transient increase in risk of myocardial infarction or sudden death with vigorous activity in people with coronary heart disease who do not undertake regular physical activity.[188](**2++**) The risk of myocardial infarction was found to be approximately six times higher during vigorous physical activity compared to the risk at rest.[188] The level of risk with vigorous activity depends on the individual's baseline level of physical activity. Assessing risk prior to starting a physical activity programme and by beginning with activity of a low intensity and steadily increasing duration and intensity over a couple of weeks, reduces this risk.

The transient increase in risk with vigorous activity is more than compensated for by the reduction in overall cardiovascular risk in those who are physically active most of the time.[189](**2++**)

Several questionnaires and checklists are available for the assessment of people considering becoming more physically active. The *PAR-Q and You* questionnaire developed by the Canadian Society for Exercise Physiology and the *PARmed-X* checklist for use by physicians are useful, freely available tools at www.csep.ca/forms.asp

PHYSICAL ACTIVITY INTERVENTIONS

Physical activity is an integral part of the lifestyle advice for people with increased cardiovascular risk. Everyone should aim to do a minimum of 30 minutes of moderate intensity physical activity (3 – 6 METs) on most days of the week. For people with time constraints this physical activity may be accumulated in bouts of 8 to 10 minutes. People who are already doing 30 minutes of moderate intensity physical activity per day should be encouraged to do physical activity of higher intensity or for longer to maximise the benefit of increasing their cardiorespiratory fitness. One meta-analysis suggests that being unfit warrants consideration as a separate risk factor for cardiovascular disease, distinct from inactivity.[190]

Snack-tivity

Recent evidence from the *Harvard Alumni Study*[95] has shown that the accumulation of shorter sessions of physical activity is associated with the same reduction in coronary risk as longer sessions, as long as the total amount of energy expended is the same. A few small clinical trials show similar improvements in cardiovascular fitness with short bouts of physical activity.[180] There is evidence suggesting that multiple short bouts of exercise may improve adherence to exercise programmes, as it is easier to integrate them into people's busy lifestyles.

'Green Prescription'

'Green Prescriptions' are an initiative of Sport and Recreation New Zealand (SPARC). A green prescription is a clinician's or practice nurse's written advice on physical activity, with optional individual follow-up available through regional sports' trusts. This intervention has been shown to improve self-rated quality of life, mean energy expenditure (total and leisure time) and increase the proportion of people undertaking at least 2.5 hours per week of leisure-time physical activity. There was a non-significant reduction in blood pressure (1 – 2 mm Hg over 12 months), and no reduction in coronary events over 4 years.[191]

More details on 'Green Prescriptions' are available at www.pushplay.org.nz

KEY - *Grades indicate the strength of the supporting evidence not the importance of the recommendations - see page xvii for details*

A Recommendation is supported by good evidence
B Recommendation is supported by fair evidence
C Recommendation is supported by non-analytic studies or consistent expert opinion
I No recommendation can be made because the evidence is insufficient
✓ Good Practice Point

INTERVENTION: WEIGHT MANAGEMENT

RECOMMENDATIONS: WEIGHT MANAGEMENT	
Measure body mass index (BMI) and waist circumference as part of a comprehensive cardiovascular risk assessment.	B
The immediate priorities in weight management are to prevent weight gain, to achieve and sustain moderate weight loss (5 – 10%) where appropriate and to increase physical fitness.	B
Encourage people with a 5-year cardiovascular risk above 15% or with diabetes and a BMI greater than 25 (especially anyone who has a BMI >30), to commence graduated lifestyle change aimed at weight reduction.	B
For significant weight loss, recommend a reduction in energy intake and an increase in physical activity.	A
Discourage the use of weight loss programmes that promote the exclusion of food groups from the cardioprotective dietary pattern or that increase saturated fatty acid intake.	C
Consider referral to weight management health care practitioners for motivational counselling or specific energy balance assessment and advice when general lifestyle advice does not achieve a sustained weight loss.	✓
Appropriate equipment is required to assess the cardiovascular risk in people who are overweight or obese.	✓
Review the indication for use of drugs that cause weight gain. Offer weight management support to people requiring drugs that cause weight gain.	✓
Only initiate pharmacological interventions as an adjunct to a comprehensive weight management programme that includes diet and physical activity and uses motivational and behavioural methods.	✓
Surgery may be considered for people with a BMI greater than 40. Decisions should take into account both the absolute cardiovascular risk and other health risks and co-morbidities.	✓

ASSESSMENT OF CARDIOVASCULAR RISK IN PEOPLE WHO ARE OVERWEIGHT OR OBESE

Use of BMI and Waist Circumference

BMI greater than or equal to 30 and waist circumferences greater than or equal to 90 cm in females or greater than or equal to 100 cm in males are all associated with increased risk of hypertension, dyslipidaemia, diabetes, coronary heart disease mortality and thromboembolic stroke.[82-84]

BMI and waist circumference are reliable measurements of obesity and abdominal obesity.[192-195] **(2++)**

Special equipment required:

- firm height meter attached to a large platform scale that measures beyond 130 kg
- sturdy step stool and examination table
- a range of large adult and thigh blood pressure cuffs should be readily available. A large adult cuff (16 cm wide) should be chosen for people with mild to moderate obesity (or arm circumference 35 – 44 cm) while a thigh cuff (20 cm wide) will need to be used for people whose arm circumference is greater than 45 cm (see Table 17).[196]
- a 2-metre cloth or paper tape should be available for measurement of waist circumference.

To measure waist circumference ask the person to hold the end of the tape against their iliac crest and to turn around. The person should be relaxed. Measurement is done against the skin. The tape should be snug but not tight and parallel to the floor. Record the circumference midway between the lower rib margin and the iliac crest to the nearest 1 cm.

Table 8: Classification of overweight in adults according to BMI

Classification	BMI (kg/m^2)	Risk of co-morbidities
Underweight	<18.5	Low (but risk of other clinical problems increased)
Normal range	18.5 – 24.9	Average
Overweight	≥25	
Pre-obese	25 – 29.9	Increased
Obese class I	30.0 – 34.9	Moderate
Obese class II	35.0 – 39.9	Severe
Obese class III	≥40	Very severe

In recent years, different ranges of BMI cut-off points for overweight and obesity have been proposed, in particular for the Asia-Pacific region. However, sparse data exist at present to make definitive recommendations. Where risk factor thresholds are given these should be interpreted as approximate guides to clinical practice only.

BENEFITS OF WEIGHT LOSS INTERVENTION

Obesity is an independent predictor of coronary heart disease, congestive heart failure, and cardiovascular morbidity and mortality. Prospective studies show that a BMI of greater than 30 and/or a waist circumference greater than 90 cm in females or greater than 100 cm in males are associated with increased blood pressure, total cholesterol, an increased risk of diabetes, coronary heart disease mortality and thromboembolic stroke. [82-84] **(2++)**

Risk of co-morbidities and total mortality increases in individuals with a BMI greater than 25.[197] More than 50% of the adult population of New Zealand is in this category. The risks of type 2 diabetes, coronary heart disease, congestive heart failure, hyperlipidaemia and hypertension rise continuously with increasing body weight starting from a BMI between 20 and 25.[117,198](**2++**)

For people with a BMI greater than or equal to 30, mortality rates from all causes, but especially cardiovascular disease, are increased by 50 to 100% above that of people with a BMI of 20 to 25.[198] More than 14% of men and 19% of women in New Zealand are obese (BMI >30), and up to half of some population groups are classified as obese.[199]

There are no large-scale clinical trials that have assessed the effects of intentional weight loss on cardiovascular morbidity or mortality. However, intentional weight loss is associated with lower mortality.[138,200](**2+**)

Weight loss improves lipid levels, blood pressure levels, glycaemic parameters, insulin sensitivity, and co-morbidities associated with type 2 diabetes, delays the transition from IGT to type 2 diabetes and can reduce the need for medications. An increase in physical activity improves weight loss and promotes the maintenance of weight loss. Modest weight loss leads to improvements in LDL-C, blood pressure, insulin resistance and left-ventricular mass.[201-206](**1++**)[207](**2++**)

Benefits commence at levels of a 5 to 10% loss of initial body weight.[198,208] A 5 kg weight loss is recommended as an initial goal in people who are overweight or obese (BMI >25) and who have co-morbidities.

The benefits of lifestyle interventions are limited by the difficulties in maintaining weight loss. Within 3 to 5 years of achieving targets during an intensive, individually-tailored weight loss programme, 13 to 38% of people have regained the weight lost.[155,209] By 5 years after highly restrictive standardised diet regimens or behavioural therapy alone, without continuous follow-up, more than 90% of participants may have regained their weight.[210,211] However, after a mean 5-year follow-up period, 10 to 27% of participants in a variety of long-term weight loss trials have maintained effective weight losses of 9 to 11 kg (mean 15% success rate).[212](**2++**)

Since weight gain promotes multiple risk factors of heart and blood vessel disease, and regain of weight is common after weight loss, the prevention of weight gain is recommended for all people with cardiovascular risk factors.(**2++**) The risk reduction package includes assessment and ongoing monitoring of weight change with age, advice to avoid weight gain, and recommended action if weight increases.

Recent clinical trials show that modest and sustainable weight losses of 5 to 10% of basal body weight are achievable.[207] Modest weight loss of less than 10% of body weight has been shown to improve insulin sensitivity and glucose tolerance.[208] Recent studies show that dietary modification and enhanced physical activity with weight loss can delay or prevent the transition from IGT to type 2 diabetes.[154,155] With weight loss, people with type 2 diabetes who are obese may be able to withdraw from insulin therapy completely without any deterioration in glycaemic control if their pancreatic response is adequate.[213] Intensive insulin treatment may lead to a 12 month weight gain of about 3 kg.[214]

WEIGHT MANAGEMENT INTERVENTIONS

Prevention

The predisposition to gain weight is a chronic disorder and requires ongoing treatment. Body weight is determined by energy balance – kilojoules consumed relative to kilojoules expended. Many people who are obese or overweight expend less than 30% of their daily energy output in physical movement

from leisure and daily living activities combined[197] and may not achieve weight reduction until energy intake is restricted to approximate their basal metabolic requirements.

There is convincing evidence that regular physical activity and higher intakes of dietary fibre are protective against unhealthy weight gain, and that sedentary lifestyles and high intakes of energy-dense micronutrient-poor foods promote weight gain and obesity.[117](**2++**) Evidence suggests a probable link between high intakes of sugar-sweetened drinks and heavy marketing of energy dense foods in the increasing prevalence of obesity. Foods with a lower GI are possibly protective, while large portion sizes, eating a high proportion of foods prepared outside the home and restrictive eating patterns possibly promote obesity.[117]

An increase in the kilojoule density of foods offered for ad libitum consumption (more kilojoules per bite) leads to a positive energy balance and weight gain in normal weight individuals.[215-220] Dietary fat and energy intake are highly correlated,[221] and fat contributes most kilojoules per gram of food. Change in dietary fat intake correlates with change in energy intake and change in body weight.[222,223](**2++**)

The main determinants of a high kilojoule food are fat and, to a lesser extent, added sugars and dense flour products (greasy or dry foods). The main characteristic of low kilojoule foods is their high water content and high dietary fibre content (low starch or wet foods, ie, vegetables and fruit. Short-term reduction in the energy density of foods offered for ad libitum consumption reduces energy intake.[220,224-228]

Increasing dietary fibre by 14 g/day increases weight loss by 1.9 kg over 2 months.[229] Restriction of total fat intake prevents short-term (<6 months) weight gain and leads to modest weight loss in adults with a BMI of 22 to 29.[230](**1+**) A 10% reduction in fat energy corresponds with a reduction in energy intake of about 1 megajoule and a loss of 3 to 4 kg in body weight in groups of normal and overweight persons.[222,223,231](**2++**)

Intervention

Interventions that prevent weight gain, and treatments for weight loss, are multimodal. A variety of programmes achieve results. A combination of advice on diet and exercise, supported by behaviour therapy, is more effective in achieving weight loss than either diet or exercise alone.[185,198,233-240](**1+**)

Motivational and behavioural approaches to dietary change promote sustained compliance and are based on experiential learning that addresses overcoming barriers to adherence while reinforcing newly adopted behavioural changes.[155,198,239,241-243] Key features associated with sustained lifestyle change include choices among therapies (diet and exercise training), greater intensity at engagement, patient-centred goal setting, more frequent contact with a health care practitioner, family support, continued intervention and follow-up (ie, no discharge).(**1+**)

Initiating Weight Loss

The 5-A behavioural framework (see Chapter 5, *Intervention: Cardioprotective Dietary Patterns*) is an appropriate framework for health care practitioner guided weight management. Essential tools include baseline assessment of dietary intake, use of self-monitoring and dietary information (see Chapter 5, *Intervention: Cardioprotective Dietary Patterns*) targeted to the level of readiness to change.

Table 9: Points to consider when discussing weight loss and diet

1	Individualise the method of reducing kilojoule intake
2	Ensure nutritional adequacy
3	Ensure cardiovascular protection
4	Consider the metabolic profile and goals (including glycaemic, LDL-C, HDL-C, triglyceride levels, insulin resistance and blood pressure)
5	Include physical activity when possible

*Table 10: A dietary strategy for weight loss and cardioprotection**

1	Reduce foods rich in fats and oils, particularly saturated fat rich foods and deep-fried products
2	Increase coloured vegetable and fruit intakes
3	Increase food sources of dietary fibre
4	Reduce white flour products and partially replace with whole grain products
5	Reduce foods and drinks rich in added sugars (bakery and confectionery items)
6	Include dried peas, beans and fish

*See also Chapter 5, *Intervention: Cardioprotective Dietary Patterns*

This dietary plan offers the full benefit of essential macro- and micronutrients, adequate fibre, dietary antioxidants and other bio-active phytochemicals while promoting a lower energy intake. Recommended changes can be tailored to individual risk, stage of change, lifestyle and cultural or social considerations.

This conservative qualitative approach emphasises sustainable changes and aims to achieve a gradual and moderate weight loss through a slowly progressive reduction in habitual energy intake. Together with an increasingly structured exercise programme it is likely to favour the development of regular lifestyle habits and belief systems compatible with weight management.(**4**) Behavioural motivation and self-management techniques with individualised goal setting, enhance these behavioural changes.[239,241-244]

Simple qualitative approaches will be sufficient to initiate weight loss. However, progression to energy-controlled approaches individualised to body size and physical activity may be required. Qualitative approaches are additive. For example, in overweight and obese people, reducing dietary fat may reduce body weight by 4.4 kg[223] and increasing dietary fibre may reduce weight by 2.4 kg.[229](**1+**) The addition of a high-fibre instruction to people consuming a low-fibre diet may triple weight loss.[245]

Specialised dietary advice by a dietitian and/or referral to a psychologist or lifestyle change specialist should be considered before drug therapies, very low energy diets or surgery, since these are therapies generally used for restricting energy intake.

Dietary Interventions that Reduce Weight

Dietary intervention is pivotal to the achievement of weight loss, while exercise and behavioural programmes or drug therapies are adjunctive therapies and alone do not achieve outcomes equivalent to those that include a dietary component.[185,198,233,235] The potential energy deficit from restricting kilojoule intake far exceeds that from adding physical activity.

Interventions for weight loss are generally evaluated over months rather than years. Since the majority of people's lives are spent in the weight stable or weight gain conditions,[246] altering the composition of short-lived diets for rapid weight loss is unlikely to confer long-term benefits despite small short-term advantages. Short term trials (less than 6 months) need to be evaluated in terms of sustainable longer-term benefits.

A number of dietary interventions have been trialled for 6 months, 12 months, or longer. A variety of programmes achieve 5 to 15 kg weight losses over one year, with the majority of weight lost in the first 6 months.[198,212,247] An appropriate initial rate of weight loss is related to the degree of excess body weight, the urgency for intervention and individual readiness and capability for change.(**4**)

A scientific review of more than 150 popular and published weight reduction diets and a separate meta-analysis demonstrate that kilojoule balance is the key determinant of weight loss regardless of macronutrient composition, and that all low-kilojoule diets result in loss of body weight and body fat.[248,249](**1++**) A low to moderate fat, balanced-nutrient reduction diet is nutritionally adequate, whereas high-fat, very low carbohydrate diets are nutritionally inadequate and require supplementation.[249,250](**3**)

Weight loss programmes that specifically reduce saturated fatty acids achieve greater improvements in risk factors.[251,252](**1+**) When energy intake is strictly controlled between comparison diets in the medium-term (12 weeks or more), weight loss is the same regardless of protein content, glycaemic index or percentage dietary fat.[252-260](**1+**)

The true benefits of specific energy-controlled dietary regimens in the treatment of excess body weight are not realised unless management is continuous, as for other treatments. Dietary recommendations to reduce saturated fatty acids and total fat intake, increase dietary fibre, reduce high glycaemic index foods, reduce added sugars and moderately increase protein intake (up to 28% energy), foster dietary changes that sustain improvements in risk factors and aid the maintenance of weight loss, with qualitative or ad libitum education styles.[253,261]

A variety of dietary compositions, teaching or behavioural techniques can promote longer-term, progressive and sustained changes in energy balance when tailored to individual needs. Simple healthy eating advice is rarely enough to achieve significant weight loss in people with obesity, since a substantial reduction in daily energy intake is required.(**2+**)

Ad Libitum Diets

Ad libitum styles of dietary education attempt to restrict food sources of one macronutrient so that dietary intake is sufficiently restricted to reduce energy intake from usual levels. These simplified methods of instruction are novel and may appear less restrictive to the person since they offer lists of 'eat these' and 'don't eat these' promoting an idea of 'eat all you like of...'. They do not comprehensively address all facets of the cardioprotective dietary pattern.

Ad libitum fat restriction diets promote weight loss if they reduce kilojoule intake.[198,262] Restricting dietary fat to 20 to 35 g per day is no more effective for losing weight than restricting energy intake to 4 to 6 megajoule.[262](**1++**)

In people who are overweight or obese, restriction of dietary fat (ad libitum carbohydrate and protein) reduces energy intake. However, weight loss is increased from 5 to 8 kg, to 9 to 12 kg (50 – 100%) over 12 to 20 weeks when energy intake is also specifically restricted (20 – 30% fat).[263-265](**1+**) An ad libitum protein diet, low in fat has achieved greater weight loss than an ad libitum carbohydrate diet, low in fat, in healthy overweight individuals (protein 25% of energy).[266](**1+**)

Weight is not associated with dietary carbohydrate content in individuals with or without diabetes.[248](**1++**) Carbohydrate restriction leads to weight loss if it reduces kilojoule intake. A popular

very low carbohydrate (ad libitum fat and protein) diet has achieved 1.5 to 4 kg greater weight losses than a kilojoule controlled diet after 6 months,[267-270] but differences were no longer present at 12 months.[269,271] High attrition rates, poor adherence and wide standard deviations were reported in these studies and some report extreme differences in weight loss between individuals, reflecting wide variance in dietary compliance.[268](**1+**) Absolute weight losses in people completing the programme are no greater than those achieved in energy-controlled trials (4 – 10 kg).[185,198,233] These diets do not encourage changes towards the cardioprotective dietary pattern during the weight-stable condition.

Moderate Energy Deficit Diets

Educational and motivational interviewing techniques that promote a moderate energy reduction yield slower weight loss than more specific energy restriction regimens.[198,233,243,272,273](**1+**) These programmes promote behavioural change, self-efficacy training, and the potential to work through short-term relapses without demoralising weight regain and may yet prove to be sustained in the long-term, providing a slow, regular weight loss is promoted.(**4**)

Individualised 2.1 to 3 MJ energy deficit dietary programmes are based on prescribing a fixed energy deficit calculated from the estimated energy expenditure. Another method is to base the deficit on reported usual intake, although this is less accurate due to patient under-reporting of intake. These methods minimise the challenge to appetite mechanisms since they aim to achieve average weight losses of around 2 kg monthly.[198]

The energy expenditure deficit method was used in the placebo and treatment arms of trials studying the use of orlistat and sibutramine. In trials of one year duration, weight losses were 1.27 to 6.4 kg with placebo (the moderate energy deficit diet and recommendation to exercise) and 3.09 to 10.3 kg with orlistat (weighted mean difference between treatments 2.7 kg).[274](**1++**)

The lifestyle coaches in the *Diabetes Prevention Program* were mainly dietitians. The intensive lifestyle arm (reported energy deficit 1.9 MJ) was compared to the metformin treatment arm (energy deficit 1.24 MJ). Weight losses were 5.6 kg and 2.1 kg respectively.(**1+**) Reduction of total fat intake was the first step for participants in the intensive intervention arm (mean BMI 34). Several weeks later, lifestyle coaches calculated individual energy deficit diets (-2.2 – -2.4 MJ depending on body weight) and tailored instruction to suit each participant.[155,275]

Low Energy Diets

Low energy diets (4.4 – 6.7 MJ) promote moderate weight losses of 3 to 13 kg in long-term studies.[198,212,234,235,249,276](**1++**) Average weight reductions of around 8% of weight in 3 to 12 months are associated with reductions in waist circumference.[198](**1+**) Behaviour modification programmes using low energy diets and exercise achieve 9 kg losses in 20 to 26 weeks. [244]

Meal replacement products in the form of drinks or cereal-based bars are designed to replace 1 or 2 meals daily to achieve a defined low or moderate energy intake (3.4 – 6.8 MJ/day). Compared with conventional reduced kilojoule diets, partial meal replacement plans achieve 2.5 kg greater weight loss over 3 month and 12 month periods.[277](**1++**) One year weight losses were 6.97 kg versus 4.35 kg for partial meal replacement diets and conventional reduced energy diets respectively.

Very Low Energy Diets

Very low energy diets or formula diets providing less than 3.4 megajoules per day with intensive behavioural and dietetic support may be appropriate in selected individuals with a BMI greater than 30.[198] Indications for use include the need to experience weight loss in those unsuccessful with motivation-dependent methods.(**4**) These very restrictive, very low energy diets are commercially-prepared formulas that replace all or most of food intake for several weeks or months. Very low energy

diets provide less than basal energy requirements (<3 – 4 MJ/day) and induce up to twice the weight loss compared to weight loss medications or moderate energy restriction.[198,276,278,279](**1++**)

Very low energy diets can result in weight loss of 13 to 23 kg over 6 to 12 months, compared to a loss of 9 to 13 kg with low energy diets.[198,279](**1++**) However long-term results with very low energy diets are no more consistent than more moderate treatments, and regain is usual after therapy.[198,212,230,276,279](**1+**)

Despite high attrition rates in trials, 25 to 35% of people completing the weight loss phase may sustain a weight loss (>10% of initial body weight) after 2 to 7 years, particularly if there is follow-up, intensive behavioural support and an exercise programme during the maintenance period.[198,212,230,276,279](**2+**)

Maintenance of Weight Loss

Methods for the maintenance of weight loss, are less well studied than methods for weight loss. Available evidence suggests that factors associated with successful maintenance include low-fat and low kilojoule intakes, and continued physical activity.[186,187,276,280-283]

Many of these strategies were incorporated in large trials (>500 people) of intensive lifestyle interventions involving weight reduction and active maintenance. After 2 to 3 years, 38 to 44% of people with IGT or hypertension had maintained a weight loss of at least 4.5 to 5 kg,[236,284] or 7% of their baseline weight.[155](**1++**) A subset analysis of subjects with IGT, who maintained a mean weight loss of 4.9 kg after 4 years, also maintained their baseline insulin secretory capacity.[285] More than half of subjects withdrawn from antihypertensive agents during lifestyle treatment, remained off medications 4 years after termination of the maintenance programme.[169] In these programmes, eight to 18 intervention sessions in the first year and 1 to 3 monthly one-on-one contacts during the maintenance phase aimed to re-engage less successful participants and to increasingly customise the intervention programme to individual needs. They all included optional group sessions. In a similar trial, weight had been regained by 36 months but most of the education was carried out in group sessions until after 18 months when individual sessions were offered.[209]

Evidence suggests that long-term maintenance of a substantial weight loss is promoted by progressively increasing exercise from 3.3 to 5 hours, and up to 7 to 10 hours of moderate-intensity physical activity per week (equivalent to 6 – 10 MJ additional energy expenditure).[237,286-288] In a cross-sectional study, people successful at long-term weight loss and weight maintenance (an average of 30 kg maintained for 5.1 years) reported continued consumption of low-kilojoule (5 – 7 MJ), and low-fat diets (24% of energy from fat)[281] and high levels of physical activity (11 MJ per week).[280]

There is evidence that early achievement of personal targets may be an important factor in the maintenance of weight loss. A greater initial weight loss may improve and predict long-term weight maintenance in programmes with follow-up for some individuals who are obese, greater than 5% of body weight at 3 months, greater than 10% at 6 months.[276,283,288-290](**2+**) Most individuals achieve their maximum weight loss within the first 20 to 24 weeks of a lifestyle intervention.[247](**1+**) Other interventions associated with weight loss maintenance include multifaceted programmes:

- especially group support with low energy diets
- behaviour modification with very low energy diets
- active individualised follow-up treatments[212]
- continued professional contact
- continued skills training
- social support, and monitoring[291]
- consciousness of behaviours
- use of social support

- problem confrontation
- personally developed self-help strategies[187]
- walking training after very low energy diets[292]
- problem-solving therapy[293]
- focus on weight[155,294]
- number of intervention sessions[291]
- use of an ad libitum low-fat diet with contacts 2 to 3 times monthly[295]
- pharmacotherapy after the active weight loss phase.[202,296,297]

Table 11: Checklist for evaluating a cardioprotective weight loss programme

Does the programme include • large servings of vegetables, especially coloured varieties • several servings of fruit each day • enough whole grains, whole grain cereals, whole grain breads to satisfy appetite • legumes (dried beans) and fish • small servings of oils, soft margarine spreads, nuts or seeds • low-fat milk, low-fat milk products, or soy products • optional small servings of lean meat and skinned poultry?
Does the programme limit • high kilojoule foods (>20 kJ/g – check labels) • fats, deep-fried foods, pastries • fatty meats, full-fat milk products • alcoholic beverages • sources of added sugar – drinks, sweets, desserts • concentrated starch-rich foods/sweet bakery products?
Further tips for reducing kilojoule intake • make main meals with low-fat, high-fibre foods from the 5 main food groups • choose animal foods with the lowest fat content • choose breads, cereals and grains for their fibre content • choose few biscuits, cakes, crackers, other fancy baked goods, muesli and confectionery bars, or snacks; check their kilojoule content as well as their fat content.

Physical Activity

Physical activity (also see Chaper 6, *Intervention: Physical Activity*) enhances dietary-induced weight loss and the maintenance of weight loss in both short-term [198,222,237] and long-term studies.[186,187,276,287,298](**2++**) Although diet is more effective for producing weight loss, evidence suggests that physical activity may be a major predictor of weight loss maintenance.

Physical activity improves insulin sensitivity reduces blood pressure, lowers triglyceride levels, levels of intra-abdominal fat, and all characteristics of the metabolic syndrome.[299]

Physical activity is recommended as part of a comprehensive weight loss and weight maintenance programme because it modestly contributes to weight loss in overweight and obese adults, decreases

abdominal fat, increases cardiorespiratory fitness and aids the maintenance of weight loss.[198] Cardiorespiratory fitness appears to be protective against cardiovascular disease even in those who are overweight or obese.[300-302] Many people do not achieve their goal weight but they can improve their health by becoming more physically fit.

Pharmacotherapy

Pharmacotherapy can be considered for people with a BMI greater than 30 or people with BMI greater than 25 and at a 5-year cardiovascular risk of greater than 20%. Pharmacotherapy alone is not as effective as pharmacotherapy given in conjunction with a comprehensive weight-management programme, including behaviour modification, diet education, and exercise counselling.[198,278,303-305]

Three main classes of drugs are available but not subsidised in New Zealand. Anorexiants (phentermine and diethylpropion), orlistat and sibutramine. It is beyond the scope of this guideline to discuss the pharmacological management of obesity in detail.

The addition of orlistat or sibutramine to a diet and exercise programme has been shown to increase weight loss by 2.7 and 4.3 kg respectively (2.9 and 4.6% of initial body weight, attrition rates 33% and 43% respectively) in studies of one-year duration.[274](**1+**)

Common weight-inducing drug groups are:

- antidepressant medications (tricyclic antidepressants, selective serotonin re-uptake inhibitors)
- lithium
- antipsychotic medications (phenothiazines, butyropenones, atypical agents)
- anti-epileptic agents (valproic acid and carbamazepine)
- steroid hormones (corticosteroid derivatives, megestrol acetate, oestrogen)
- antidiabetic medications (insulin, sulfonylureas and glitazones).

Surgery

Surgery to aid weight reduction (bariatric surgery) may be considered when all other measures have failed. Key aims of this type of surgery are to achieve weight reduction and maintain any loss through restriction of intake and/or malabsorption of food. Bariatric surgery is not commonly undertaken in New Zealand. A review of the literature and recommendations on an approach to the treatment of morbid obesity is available from the National Institute of Clinical Excellence (NICE) in England at www.nice.org.uk/pdf/Fullguidance-PDF-morbid.pdf

INTERVENTION: SMOKING CESSATION

RECOMMENDATIONS: SMOKING CESSATION	
All smokers should be encouraged to stop smoking. Smoking cessation has major and immediate health benefits for smokers of all ages.	A
The recording of current and past smoking habits is recommended as part of a comprehensive cardiovascular risk assessment.	✓
Nicotine replacement therapy (NRT) is recommended as first-line pharmacotherapy for smoking cessation in New Zealand. Buproprion or nortriptyline hydrochloride are alternatives and recommended as second-line agents.	A
Use NRT cautiously (after discussion with a specialist) in the immediate post-myocardial infarction period (4 weeks) and in those with serious arrhythmias, or severe or worsening angina.	C
Nortriptyline hydrochloride is contraindicated during the acute recovery period after myocardial infarction.	C

Tobacco smoking is estimated to kill between 4300 and 4700 New Zealanders per year.[66] Nearly half of these deaths occur in middle age (35 to 69 years).[306]

All smokers should be encouraged to stop smoking. It is beneficial to stop smoking at any age. Smoking cessation has major and immediate health benefits for smokers of all ages. Former smokers have fewer days of illness, fewer health complaints, and view themselves as healthier.[79] Within one day of quitting the risk of having a myocardial infarction is reduced. The excess risk of heart disease is reduced by half after one year's abstinence. The risk of a coronary event reduces to the level of a never smoker within 5 years. In those with existing heart disease, cessation reduces the risk of recurrent infarction or mortality by half.

NRT decreases withdrawal symptoms and improves cessation outcomes.[307](**1++**) NRT is currently available as patches and gum in New Zealand for purchase over-the-counter at pharmacies and, through a subsidised scheme, from the Quitline and authorised providers. NRT is also available as a nicotine nasal spray (prescription medicine) and a nicotine inhaler (pharmacy only).

This chapter is adapted from the National Health Committee (NHC) Guidelines on smoking cessation. These are available from: www.nhc.govt.nz/publications/Smoking%20Cessation.pdf

Two medications, buproprion (not subsidised in New Zealand) and nortriptyline hydrochloride (subsidised) have been shown to be effective for smoking cessation in randomized controlled trials. The evidence is sufficient to endorse their use in clinical practice.

People with cardiovascular disease should start NRT at a lower dose. It is more dangerous for those with coronary heart disease to continue smoking than to use NRT. The dose may be increased if withdrawal symptoms occur. People should be followed up closely. Nortriptyline hydrochloride is contraindicated during the acute recovery period after myocardial infarction. Bupropion is a suitable treatment, if appropriate, for people with cardiovascular disease

There is good evidence for the effectiveness of:

- brief advice from health practitioners[308]
- all forms of NRT available in New Zealand for people smoking more than 10 to 15 cigarettes per day[307]
- self-help materials alone
- follow-up telephone calls which improve effectiveness
- organised group programmes, which are better than self-help materials but no better than intensive health professional advice
- counselling and self-help materials for pregnant women who smoke
- specific counselling for men at risk of ischaemic heart disease
- some interventions provided in specialist smoking cessation clinics
- the antidepressant drugs bupropion and nortriptyline hydrochloride as second-line pharmacotherapy.[309]

KEY - *Grades indicate the strength of the supporting evidence not the importance of the recommendations - see page xvii for details*

A Recommendation is supported by good evidence
B Recommendation is supported by fair evidence
C Recommendation is supported by non-analytic studies or consistent expert opinion
I No recommendation can be made because the evidence is insufficient
✓ Good Practice Point

INTERVENTION: LIPID MODIFICATION

RECOMMENDATIONS: MANAGEMENT OF LIPID ABNORMALITIES	
The higher the calculated cardiovascular risk, the more aggressive the management of modifiable risk factors, including lipids, should be.	C
People presenting after an acute cardiac event (myocardial infarction or unstable angina) should start treatment with a statin simultaneously with intensive lifestyle advice. Treatment should aim to lower LDL-C to less than 2.5 mmol/L.* This should be given in association with other appropriate medication such as aspirin, a beta-blocker and an ACE-inhibitor.	A
Lipids should ideally be measured at the time of the acute event. Since the metabolic disturbance continues for 10 to 12 weeks after a myocardial infarction, further measurements should be deferred for three months.	C
People presenting after an acute cardiac event with hypertriglyceridaemia and a low HDL-C should be considered for a fibrate or combination therapy.	C
In people with venous CABG, treatment should aim to lower the total cholesterol to less than 3.5 mmol/L and LDL-C to less than 2.0 mmol/L.*	A
Most people presenting after an ischaemic stroke or transient ischaemic attack should start treatment with a statin.	B
Everyone with a total cholesterol greater than 8 mmol/L or TC:HDL ratio greater than 8* should have drug treatment and specific lifestyle advice to lower risk factor levels.	C
Within the range of total cholesterol 4 to 8 mmol/L, all decisions to treat should be based on the individual's cardiovascular risk.	B
People with low HDL-C and elevated triglycerides with a 5-year cardiovascular risk greater than 15% should be treated with intensive lifestyle interventions and are likely to need treatment with a fibrate or combination drug therapy.	B

*Where risk factor thresholds are given these should be interpreted as approximate guides to clinical practice only.

RECOMMENDATIONS: MANAGEMENT OF LIPID ABNORMALITIES (CONTINUED)	
A cardioprotective dietary pattern is strongly recommended as an integral component of lipid management.	A
Dietary advice should be tailored to the individual's risk factor and lipid profile.	B
Among people with a 5-year cardiovascular risk greater than 15% the aim of treatment is to lower 5-year cardiovascular risk to less than 15%.	B
LDL-C should be used as the primary indicator of optimum lipid management and should be used to monitor lipid-modifying treatment.	C

LIPIDS AND CARDIOVASCULAR RISK

Lipids

A fasting lipid profile (total cholesterol, LDL-C, HDL-C, TC:HDL ratio and triglycerides) should be taken. A single TC:HDL ratio is used to calculate cardiovascular risk. Two lipid measurements should be taken prior to initiating drug treatment or intensive lifestyle treatment. If the total cholesterol level varies more than 0.8 to 1 mmol in the two samples, a third sample should be taken and the average of the three samples should be used as the baseline measure. A fasting sample is required for the measurement of triglycerides.

Secondary Causes of Lipid Abnormalities

The secondary causes of lipid abnormalities include diabetes, obesity, insulin resistance, liver disorders, thyroid disorders, some haematological disorders and renal disease. A rise in triglycerides is seen in people with diabetes, people who are obese, or who have excessive alcohol consumption. Any identifiable cause should be treated prior to initiating lipid-lowering treatment. A rise in cholesterol is normal in pregnancy and a cholesterol level should not be measured at this time.

Genetic Causes of Lipid Abnormalities

People with FH, FDB and FCH (see below) are considered at very high risk or potentially at very high risk (5-year cardiovascular >20%) see Chapter 3, *Cardiovascular Risk Assessment*. People with the other genetic causes referred to below may have a 5-year cardiovascular risk of less than 20%. Both groups should be referred for specialist assessment, advice on management, and family tracing:

- **5-year cardiovascular risk greater than 20%**
  - **Familial hypercholesterolaemia (FH)**
  - **Familial defective ApoB (FDB)**
  - **Familial combined dyslipidaemia (FCH)**

KEY - *Grades indicate the strength of the supporting evidence not the importance of the recommendations - see page xvii for details*
A Recommendation is supported by good evidence
B Recommendation is supported by fair evidence
C Recommendation is supported by non-analytic studies or consistent expert opinion
I No recommendation can be made because the evidence is insufficient
✓ Good Practice Point

- **5-year cardiovascular risk less than 20%**
  - **Low HDL-C syndromes**

    Low HDL-C confers a high risk for cardiovascular events. The causes of low HDL-C are multiple and these subjects are refractory to most drug interventions.
  - **High LP(a)**

    The genetic cause of high LP(a) is unknown. High values are refractory to most drug interventions.
  - **Isolated high triglycerides (>8 mmol/L)**

    The management of people with isolated high triglycerides should be discussed with the appropriate specialist.
  - **Broad beta disease**

    If the TC:triglyceride ratio approaches one, with both lipid fractions elevated, then further investigation is needed.

Benefits of Lipid Modification

Prospective studies demonstrate a constant linear relationship between relative cardiovascular risk and total cholesterol in the range of about 4 to 8 mmol/L. Within this range, treatment results in similar relative benefits regardless of baseline cholesterol levels.[67,68](**2++**)

DIETARY INTERVENTIONS THAT MODIFY LIPIDS

RECOMMENDATIONS: DIETARY INTERVENTIONS FOR LIPID MODIFICATION	
Dietary intervention is strongly recommended as an integral component of the management of blood cholesterol and lipids.	A
Advise a cardioprotective dietary pattern rich in plant foods including fruits, vegetables, dried peas and beans including soy, whole grains and other appropriately processed cereals, suitable plant oils, nuts and seeds. This eating pattern may include plant sterol or stanol-fortified spreads and regular use of oily fish.	A
Assist the individual in identifying and choosing foods which are low in saturated fatty acids, transunsaturated fat and dietary cholesterol.	A
People who are overweight or obese should be offered appropriate weight loss interventions.	A
Identify excessive alcohol intake and advise reduction or substitution with non-alcoholic beverages.	A
Individualise nutritional counselling and other lifestyle changes to complement prescribed risk factor modifying pharmacological agents to reduce absolute cardiovascular risk.	✓

EVIDENCE STATEMENTS

- There is a dose response relationship between saturated fatty acids and LDL-C. Diets high in saturated fatty acids raise LDL-C. Reducing saturated fatty acids lowers LDL-C.(**1+**)
- Transunsaturated fats raise LDL-C and lower HDL-C.(**1+**)
- Higher levels of dietary cholesterol raise LDL-C.(**1+**)
- Dairy fats and meat fats are a source of saturated fatty acids and raise LDL-C.(**1+**)
- Omega-6 polyunsaturated fats lower LDL-C when added to the diet.(**1+**)
- Monounsaturated fats lower LDL-C when substituted for saturated fatty acids in the diet.(**1+**)
- Cardioprotective dietary patterns featuring multiple food and nutrient components lower LDL-C.(**1+**)
- Modest weight loss may raise HDL-C and lower triglyceride.(**1+**)
- The addition of plant sterol and stanol-fortified spreads to a cardioprotective dietary pattern further lowers LDL-C.(**1+**)
- Nuts and seeds, excluding coconuts, are a source of unsaturated fatty acids, and if eaten in conjunction with a lower saturated fatty acid diet can lower LDL-C.(**1+**)
- Viscous fibre (eg, from oats, dried peas and beans and psyllium) 2 to 10 g/day lowers LDL-C.(**1+**)
- Soy protein, 25 g/day (eg, from soy milk, soy flour or tofu) lowers LDL-C, especially when it replaces animal food products.(**1+**)
- Oily fish or fish oil supplements (EPA and DHA, 4 g/day) may be useful in people with treatment resistant triglycerides.(**1+**)
- Alcohol raises HDL-C and triglyceride.(**1+**)
- The consumption of boiled or plunger coffee raises LDL-C and should be limited or the coffee filtered.(**1+**)
- The macronutrient composition of the diet for the management of blood cholesterol and lipids will rely on the individual metabolic profile, body weight and shape, and dietary preferences.(**4**)

Cardioprotective Dietary Patterns

The macronutrient composition of the diet for the management of blood lipids has previously been defined by the AHA and NCEP *Step I* and *Step II* diets. These diets were designed to progressively reduce saturated fatty acids and dietary cholesterol and to promote weight loss if indicated. These dietary options could be expected to lower LDL-C by 12% and 16% respectively.[222](**1+**) Additional dietary instruction to include more fruits, vegetables and cereal fibre can achieve greater reductions in LDL-C.[115] More recently these diets have been superseded by AHA dietary guidelines that place increased emphasis on foods and an overall dietary pattern, rather than nutrients.[310] These guidelines are generally consistent with The National Heart Foundation's food-based dietary statements. The composition of the dietary pattern will often rely on the individual metabolic profile, body weight and shape, and dietary preferences.(**4**)

Energy Balance

Total energy intake should be sufficient to achieve and maintain a healthy body weight and waist circumference. If excessive weight and/or abdominal fat is present, total energy intake should be lowered and physical activity increased to encourage weight loss. Modest weight loss will improve blood lipids. For every 1% decrease in body weight, triglyceride decreases by 0.011 mmol/L and HDL-C increases by 0.011 mmol/L.[222](**1+**)

Dietary Fat and Cholesterol

The major dietary lipids known to raise LDL-C are saturated fatty acids and to a lesser extent dietary cholesterol.[311](**1+**) Transunsaturated fats, formed through hydrogenation (hardening) of vegetable oils, raise LDL-C and lower HDL-C.[311](**1+**) Adding omega-6 PUFA will lower LDL-C independently of the saturated fatty acids content of the diet.[312](**1+**) Monounsaturated fats (*cis*-oleic acid) lower LDL-C but only when substituted for saturated fatty acids in the diet.[312] A 1% increase in total energy from saturated fatty acids would result in a 0.05 mmol/L increase in LDL-C; a 1% increase in PUFA would result in a 0.01 mmol/L decrease in LDL-C. A 1 mg/day change in dietary cholesterol would produce a 0.001 mmol/L change in blood cholesterol.[313]

Butter, Margarines and Spreads

After coconut, butter contains the highest total content of saturated fatty acids within common New Zealand foods. The National Heart Foundation of New Zealand recommends replacing butter with margarines and spreads.[314] There is public concern that margarines and spreads may contain transunsaturated fats. Changes to manufacturing techniques now allow people to choose from two distinct groups of margarines and spreads – those with 1% or less transunsaturated fat and those with between 12 to 18% transunsaturated fat.[315] A meta-analysis reports that replacing butter with low transunsaturated margarine in the diet reduces LDL-C.[316](**1+**) Even spreads with moderate amounts of transunsaturated fat are still better than butter in reducing LDL-C.[317,318](**1+**)

Functional foods claim to have added benefit over and above their usual nutrients. There is currently insufficient evidence for a cardioprotective effect of functional foods other than plant-sterol and stanol-fortified spreads. Daily intake of plant sterols or stanols (0.8 – 3 g/day) is additive to conventional dietary therapy and can be expected to lower LDL-C by 9 to 20%.[319](**1+**) In order to minimise any decrease in plasma carotenoids associated with the consumption of plant-sterol or stanol-fortified spreads, the diet should include a daily intake of yellow and orange vegetables and fruits.[320](**1+**)

Meat and Meat Products

Meat fat contains saturated fatty acids which increase LDL-C.[321](**1+**) Meat fat should be limited in those seeking to lower cardiovascular risk. Most processed meats or meat products, such as sausage, saveloy, luncheon meat, paté and salami, are high in saturated fatty acids. Lean meat will not adversely affect LDL-C when the background diet is low in saturated fatty acids.[322-326](**1+**) Traditional practice recommends 100 to 150 g and 150 to 200 g cooked lean meat per day for women and men respectively, depending on their body size.(**4**)

Nuts and Seeds

Nuts and seeds are generally rich sources of monosaturated fatty acid (MUFA) and PUFA, particularly omega-6 PUFA. They are also sources of plant proteins, viscous fibres, various micronutrients, phytosterols and many different phytochemicals, some of which have antioxidant properties.[327] Polyunsaturated and MUFA-rich nuts lower LDL-C.[328-341](**1+**) The evidence reviewed is from studies of almonds, walnuts, pecans, pistachios, macadamias, and peanuts. There is no research available on

other nuts or on sunflower, pumpkin or sesame seeds. The fat composition of their oils would indicate favourable effects on LDL-C.

Coconuts differ from other nuts in that 91% of the fat is saturated fatty acids. All nuts are high in fat and should replace saturated fatty acids in the diet rather than as an additional food. A suitable quantity would be 30 g/day, this is to prevent an increase in total fat which may lead to weight gain.**(4)**

Fish and Fish Oils

Eating oily fish frequently, or consuming EPA and DHA supplements, has been reported to lower triglycerides and raise HDL-C.[342] A dose-response relationship exists between EPA and DHA and triglyceride lowering. Four grams of EPA and DHA per day can lower triglyceride 25 to 30%, with accompanying increases in LDL-C of 5 to 10% and in HDL-C of 1 to 3%.[342] The ingestion of this amount of EPA and DHA is only possible through supplementation. The triglyceride-lowering effect appears to be most pronounced in those with severely elevated triglycerides and is specific to marine not plant sources of omega-3 PUFAs.[343,344] The lipid response to EPA and DHA supplements are comparable in people with or without diabetes.[345]**(1+)** People taking more than 3 g/day of these fatty acids from supplements should do so only under a doctor's care. Potential side effects should be kept in mind and supplements screened for peroxide value and flavour. Rancid oils within supplements will lead to gastric upset and very high intakes could cause excessive bleeding in some people.

Table 12: Risks for side effects from ingestion of EPA and DHA supplements

Omega-3 Dose	Gastro-intestinal upset	Clinical bleeding	Fishy after taste	Worsening glycaemia	Rise in LDL-C
<1 g/day	Very low	Very low	Low	Very low	Very low
1 – 3 g/day	Moderate	Very low	Moderate	Low	Moderate
>3 g/day	Moderate	Low	Likely	Moderate	Likely

Source: Kris-Etherton et al.[130]

Dietary Carbohydrate

Gradual substitution of total and saturated fatty acids energy with carbohydrate energy is recommended for LDL-C reduction but may lower HDL-C and raise triglycerides when the type of carbohydrate is not adequately defined.[312] This adverse lipid reaction may be avoided by promoting carbohydrate food choices that provide moderate amounts of non-starch polysaccharide, such as vegetables, fruits, dried peas and beans, whole grains and other appropriately processed cereals.[346]**(4)** When combined with a low saturated fatty acids' diet, non-starch polysaccharide in the form of viscous fibre (2 to 10 g/day) lowers LDL-C 5 to 10% and the effect of viscous fibre may be greatest in those at higher cardiovascular risk.[347]**(1+)** There is insufficient evidence on the efficacy of glycaemic index for the dietary management of blood cholesterol and lipids.**(I)**

Dietary Protein

Iso-energetic exchange of added sugars and other refined carbohydrate containing foods, with low-fat protein sources may improve the lipid profile.**(4)**. Plant and marine-based protein sources including nuts, dried peas and beans including soy, whole grains and fish have broader cardioprotective benefits than eggs, meat or poultry food choices.

Soy and Soy Products

Soy proteins are legumes and lower LDL-C in those people at higher cardiovascular risk.[348](**1+**) Soy proteins are relatively easy to incorporate into the traditional New Zealand diet, with several food products commonly available. Three to 4 glasses a day of soy milk would provide approximately 25 g of soy protein per day, a level that has been shown to lower LDL-C.[348](**1+**) Other food sources include tofu, soy flour, isolated soy protein, and meat analogue. There have been reports of allergies to soy products, including anaphylactic reactions, though these are rare. There is no evidence that adverse effects are common or important in adults or children consuming soy protein.

Alcohol

Moderate to excessive alcohol consumption can raise HDL-C and triglyceride in the blood.[134](**2+**) Alcohol intake should not exceed 3 standards drinks for men and 2 standard drinks for women a day (see Table 6).

Other Dietary Factors

Garlic

The active component of garlic is allicin, which is released by mechanical disruption of garlic cloves. There is a wide variation of allicin concentration in cloves and in industrial preparations (up to 20-fold in commercially available preparations) which complicates the comparison of studies.[349] A number of studies on the lipid-lowering effects of garlic have methodological flaws. When combined in meta-analysis, there appears to be a positive effect of garlic supplements, but these results have probably overstated the effects of garlic.[350,351](**1+**) At this stage, the evidence does not appear strong enough to recommend taking garlic supplements for cardioprotection, though garlic appears to cause little harm when eaten as a food within a varied diet.

Coffee

The consumption of boiled or plunger coffee raises LDL-C.[352](**1+**) This is a dose-response relationship. Two lipid-containing substances, cafestol and kahweol, have been implicated as the components in coffee responsible for its cholesterol-raising effects. Filtering may lower as much as 80% of the cafestol and kahweol in coffee and mitigates the increase in LDL-C.[353]

DRUG INTERVENTIONS THAT MODIFY LIPIDS

Benefits of Lipid-modifying Drugs

Randomized controlled trials with statin medications have demonstrated reductions in cardiovascular disease (coronary heart disease and ischaemic stroke) morbidity, mortality and total mortality. With the dosage regimens used in these clinical trials, reductions in risk of 30 to 50% for both coronary heart disease and ischaemic stroke have been observed.[69,354-357](**1++**) There is no evidence that reducing LDL-C to below 1.7 mmol/L confers additional benefit.(**3**)

Early treatment of lipids in people with acute coronary syndromes has been shown to improve long-term compliance.[358](**1+**).

Treatment of people with a 5-year cardiovascular risk greater than 15% with intensive lifestyle advice and statin therapy is cost-effective compared to other community drug therapies that are funded in New Zealand.[52](**2E**)

Lipid-modifying Drug Therapy

Priority for drug treatment is given to those at higher absolute risk, because treatment in this group gives greater benefit and is more cost-effective.

Statin Treatment

Statins should be the first-line treatment if the main abnormality is elevated total cholesterol or LDL-C. It is recognised that there are other benefits of statin therapy, pleiotropic effects, mediated through anti-inflammatory or antithrombotic factors and through mechanisms that improve endothelial function. Statins, except atorvastatin, should be taken in the evening when their effect is greater.

Large clinical trials have shown that statins (simvastatin and pravastatin) reduce cardiovascular events, cardiovascular disease mortality and total mortality.[69,355,357] High-dose atorvastatin (80 mg/day) and simvastatin (80 mg/day) have been reported to reduce LDL-C by 50%. A recent large study of 19,342 people on treatment for elevated blood pressure (without known coronary heart disease) at moderate cardiovascular risk has shown that 10 mg of atorvastatin can reduce the combined endpoint (non-fatal myocardial infarction, silent myocardial infarction and fatal coronary heart disease) by 36% (OR 0.64, 95% CI, 0.50 – 0.83, NNT=91 over 3.3 years). Fatal and non-fatal strokes were reduced by 27% (OR 0.73, 95% CI, 0.56 – 0.96, NNT=143 over 3.3 years).[360]

As with all medication there is a dose-response curve. Doubling a statin dose gives a further 6 to 10% reduction of LDL-C.[361] There is debate whether people should be started on clinical trial dosages (eg, simvastatin 40 mg) or titrated up from a lower dose.

Starting Doses

In people at a 5-year cardiovascular risk of between 15 and 20%, a dose equivalent to simvastatin 20 mg should be tried first and this dose can be titrated up if needed to meet the absolute risk target. This strategy may help to minimise side effects and maximise compliance.

Most people at very high risk (5-year cardiovascular risk >20%) without clinical cardiovascular disease can be started on the lower dose of simvastatin 20 mg according to their starting LDL-C (see Table 14). Some will need a higher initial dose. The individual dose required to achieve a 5-year cardiovascular risk of less than 15% will vary.

Two strategies for starting statins are possible for people with known cardiovascular disease or at very high risk, defined clinically. Either a fixed dose of 40 mg simvastatin can be used as a starting dose, or a dose titration strategy similar to that advocated for people at lower risk can be used. In the *Heart Protection Study*[69] a fixed dose of simvastatin 40 mg was used and a relative risk reduction for cardiovascular events of about 25% was achieved. In the *4S Study*[357] 37% of the intervention group were titrated from 20 mg per day of simvastatin to 40 mg per day, to reach a target total cholesterol 3 to 5.2 mmol/L. A relative risk reduction for cardiovascular events of about 35% was achieved.

Serious side effects of statin treatment are very rare (<1:100,000) and include myopathy and hepatotoxicity. Muscle soreness is common in both treated and placebo arms of clinical trials. However, people who experience muscle soreness, tenderness or pain with a rise in creatine kinase greater than 10 times the upper limit of normal, should stop statin medication. If moderate rises (3 – 10 times the upper limit of normal) are observed with symptoms, creatine kinase levels should be monitored weekly and specialist advice sought. A reduction in statin dose or a temporary discontinuation may

be appropriate. If muscle pain occurs in the absence of a creatine kinase rise, a reduction in statin dose or temporary discontinuation may also be appropriate. The incidence of myopathy increases in those with renal impairment and with the combination treatments (statin and fibrate, or statin and nicotinic acid).[362](**3**)

Fibrate Treatment

Dyslipidaemic people with low HDL-C and elevated triglycerides may benefit from intensive lifestyle interventions, fibrates and/or combination therapies.

One randomized controlled trial with gemfibrozil has shown a reduction in the combined endpoint of coronary heart disease events and ischaemic stroke of 24% (RR 0.76, 95% CI, 0.64 to 0.89, ARR 5.6%, NNT=18 over 5.1 years).[363](**1++**) A trial of bezafibrate treatment in people with coronary artery disease showed increases in HDL-C and a non-significant trend towards a reduction in fatal and non-fatal coronary events.[364]

Serious side effects of fibrate treatment are rare, but include myopathy. Minor dyspepsia and erectile dysfunction are more common. Caution is advised in prescribing fibrates for people with hepatic or renal dysfunction or pre-existing gallstone disease. Myopathy is more likely to occur when renal function is impaired (GFR <60 ml/min/1.73m^2) or if statins are given in combination therapy with fibrates.[365](**3**)

Other Lipid-modifying Drugs

Ezetimibe (not subsidised in New Zealand), niacin/nicotinic acid and/or cholestyramine resin may prove useful additions in some people where goal reductions in risk have not been achieved.

Randomized trials show that across multiple ezetimibe/statin trials the addition of ezetimibe to a statin produces about a 21% further reduction in LDL-C.[366,367]

There is one randomized controlled trial that has shown niacin (nicotinic acid) reduces non-fatal recurrent myocardial infarction but not total mortality. In this trial a reduction in mortality was observed after a mean follow-up of 15 years in the niacin-treated group.[368](**1+**)

In people without clinical cardiovascular disease, cholestyramine resin (24 g/day) reduces the incidence of fatal and non-fatal myocardial infarction but not total mortality.[369](**1+**)

Choice of Lipid-modifying Therapy

The lipid profile will determine the drug treatment selected to lower lipid levels.

Predominant Hypercholesterolaemia

Statins should be used to lower LDL-C when the triglyceride level is normal or only slightly elevated. Ezetimibe 10 mg per day will generally give about a 15% additional LDL-C reduction when combined with a statin.[367]

Low HDL-C on Statin Therapy

When HDL-C is below 0.9 mmol/L and the person with diabetes is on statin therapy, those treatments that augment LDL-C cholesterol and triglyceride lowering actions and raise HDL-C cholesterol should be considered. Such people may need specialist review and those with very low HDL-C levels (<0.7 mmol/L) should be referred.

Predominant Hypertriglyceridaemia and Low HDL-C

Fibrates may need to be used either as monotherapy or in combination with statins in people with predominant hypertriglyceridaemia and low HDL-C with normal to slightly elevated LDL-C levels (<3 mmol/L). If treatment with a fibrate is not tolerated or if additional triglyceride-lowering effect is required, fish oil can be used with close monitoring of glycaemic control. Acipimox is often useful in those not responding to fibrate agents. Niacin can be used, but will often require specialist review. Statins are not usually useful when triglycerides are markedly elevated (>5.0 mmol/L).

Combined Dyslipidaemia

Treatment with a statin and a fibrate should be considered in people with moderate to marked elevation of both LDL-C and triglycerides. Because of the increased risk of myositis with combinations (particularly those that include gemfibrozil), special care should be taken to fully inform and monitor people on combination treatments.

Table 13: The effect of various drug classes and plant sterols on lipid profiles

		Cholesterol	LDL Cholesterol	Triglycerides	HDL	IDL
Statins*	simvastatin atorvastatin 20 – 40 mg/ day	⇓⇓⇓⇓	⇓⇓⇓⇓	↓→	→↑	↓ Variable
Fibrates	gemfibrozil 1200 mg/day bezafibrate 600 mg/ day or bezafibrate retard 400 mg/day	↓	↓→	⇓⇓	↑	⇓⇓
	ezetimibe 10 mg/day	⇓⇓	⇓⇓	↓	↓→	
Niacin	>1.5 g/day	⇓⇓⇓	⇓⇓	⇓⇓	↑	⇓⇓
Acipimox	750 – 1000 mg/day	↓	↓	↓	↑	?
Plant sterols	20 g/day of product	↓	↓	↓	↑	?

KEY	0	<5%	5 – 10%	10 – 20%	20 – 30%	>30%
% change	→	↓→	↓	⇓⇓	⇓⇓⇓	⇓⇓⇓⇓

*Statins on a mg dose basis: rosuvastatin > atorvastatin > simvastatin > pravastatin > fluvastatin.

Table 14: Doses of various statins required to reach a target

Average daily doses (in mg/day) of various statins needed to achieve a LDL-C target less than 2.6 mmol/L (100 mg/day) in at least 50% of subjects with various baseline total cholesterol and LDL-C levels (and normal range HDL-C and triglycerides).

	TC mmol/L	LDL-C mmol/L	% change needed	Fluvastatin (mg/day)	Pravastatin (mg/day)	Simvastatin (mg/day)	Atorvastatin (mg/day)	Rosuvastatin (mg/day)
Lipid profiles of subjects	4.5	3	15	20	10	5	2.5	
	5	3.5	25	40	20	10	5	1
	5.5	4	35	80	40	20	10	2.5
	6	4.5	45	Combination therapy needed	80	40	20	5
	6.5	5	50			80	40	10
	7.5	6	60				80	20
Doubling the statin dose gives a further 6 – 10% reduction of LDL								

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Note: At the time of publication ezetimibe, rosuvastatin and pravastatin are not subsidised in New Zealand and fluvastatin is not available.

THE OVERALL GOAL OF INTERVENTION

The goal for everyone is to reduce 5-year cardiovascular risk to less than 15%. The intensity of treatment to achieve this goal should be related to the pre-treatment cardiovascular risk. The higher the level of risk, the greater the effort made to achieve the optimal lipid levels. An individual's risk factor levels should always be interpreted within the context of their calculated cardiovascular risk.

Examples of Specific Targets

People at very high risk (5-year cardiovascular risks 20%) determined clinically with:

- **a previous history of cardiovascular disease**
- **the specific lipid disorders of a genetic basis (FH, FDB or FCH)**
- **diabetes and overt diabetic nephropathy or diabetes with other renal disease.**

People with a risk greater than 20% should start intensive lifestyle advice concurrently with drug therapy. For people with coronary artery disease all efforts should be made to reach optimal lipid levels (see Table 15). In people with venous CABG, treatment should aim to lower the total cholesterol to less than 3.5 mmol/L and LDL-C to less than 2.0 mmol/L.[370] After stroke or transient ischaemic attack treatment with a statin is recommended irrespective of lipid levels.

People with a calculated 5-year cardiovascular risk greater than 15%

The goal for people with a risk greater than 15% is to reduce 5-year cardiovascular risk to less than 15%. There is no normal or ideal lipid level. When lipid levels are chosen as targets they should be individualised to each person according to their level of cardiovascular risk.

Total cholesterol greater than 8 mmol/L or TC:HDL ratio greater than 8

People with total cholesterol greater than 8 mmol/L or TC:HDL ratio greater than 8 should have a cardiovascular risk assessment and receive specific lifestyle intervention and lipid-modifying treatment. It may be impossible to reach optimal levels in some people (eg, people with genetic lipid disorders). People with genetic lipid disorders (TC >8 mmol/L plus a family history of premature coronary heart disease or xanthelasma) should be referred for specialist assessment, advice on management and family tracing.

People Aged 75 Years and Over

People aged 75 years and over should be treated in the same way as younger people.

Table 15: Optimal lipid levels

Lipid Fraction	Value
Total Cholesterol	<4 mmol/L
LDL Cholesterol	<2.5 mmol/L
HDL Cholesterol	>1 mmol/L
TC:HDL Ratio	<4.5
Triglycerides	<1.7 mmol/L

There is no normal or ideal lipid level. When lipid levels are chosen as targets they should be individualised to each patient and the calculated risk.

LDL-C should be used as the primary indicator of optimum lipid management and used to monitor lipid-lowering treatment. HDL-C is a secondary indicator of optimum lipid management.

It may be impossible to reach these optimal levels in some people and for these people the simultaneous improvement of several risk factors represents a better approach than the aggressive pursuit of further small reductions of LDL-C. An individual's risk factor levels should always be interpreted within the context of their calculated cardiovascular risk.

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✓ Good Practice Point

MONITORING AND DURATION OF TREATMENT

RECOMMENDATIONS: TREATMENT MONITORING AND DURATION	
For people prescribed intensive lifestyle therapy or lipid-lowering medication, lifelong treatment is recommended.	✓
Lipid monitoring is recommended for those on lipid-lowering drug treatment every 3 months until levels are controlled, then every 6 months.	C
A baseline transaminase level (ALT) should be taken prior to initiating statin medication. A baseline ALT and creatinine should also be taken prior to initiating fibrate medication. A second ALT should be taken at the time of the first follow-up, and thereafter if indicated clinically.	✓
Creatine kinase should be requested for people who have definite unexplained muscle symptoms.	✓
Lipids should be measured when people present acutely at the time of a myocardial infarction. Further measurements should be deferred until 3 months.	C

If ALT rises up to 3 times normal it is usually possible to continue with a statin medication. If ALT rises more than 3 times normal, then increased monitoring is required. Discussion with a specialist should be considered.

If muscle pain occurs in the absence of a creatine kinase rise a reduction in statin dose or temporary discontinuation may be appropriate. If moderate rises (3 – 10 times the upper limit of normal) of creatine kinase are observed with symptoms, creatine kinase levels should be monitored weekly and specialist advice sought. A reduction in statin dose or a temporary discontinuation may be appropriate. Statin therapy should be stopped if creatine kinase rises to more than 10 times the upper limit of normal. Monitoring advice is based on the ACC/AHA/NHLBI clinical advisory on the use and safety of statins.[362] **(3)**

Lipids should ideally be measured when people present at the time of an acute myocardial infarction then further measurements should be deferred until 3 months. LDL-C and HDL-C fall in the first 9 days after a myocardial infarction, but triglycerides remain unchanged.[371] **(2+)**

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INTERVENTION: BLOOD PRESSURE LOWERING

RECOMMENDATIONS: MANAGEMENT OF BLOOD PRESSURE	
The higher the calculated cardiovascular risk, the more aggressive the management of modifiable risk factors, including blood pressure, should be.	C
People presenting after an acute myocardial infarction should be considered for a beta-blocker and ACE-inhibitor regardless of blood pressure level, concurrently with intensive lifestyle advice. This should be given in association with other appropriate medication, such as aspirin and a statin.	A
People presenting after an acute ischaemic stroke or transient ischaemic attack should start blood pressure lowering medication unless the person has symptomatic hypotension. This medication should be given in addition to other appropriate medication such as aspirin, a statin, or warfarin, if indicated. Treatment should start concurrently with intensive lifestyle advice. It is usually advisable to wait 7 to 14 days before starting blood pressure lowering medication.	A
Everyone with blood pressure consistently greater than 170/100 mm Hg should have drug treatment and specific lifestyle advice to lower risk factor levels.	C
Within the blood pressure range 115/70 to 170/100 mm Hg, all decisions to treat should be based on the individual's cardiovascular risk.	B
A cardioprotective dietary pattern is strongly recommended as an integral component of blood pressure management.	A
Dietary advice should include the limitation of both alcohol (see Table 6) and sodium consumption.	B
Among people with a 5-year cardiovascular risk greater than 15% the aim of treatment is to lower 5-year cardiovascular risk to less than 15%.	B
A low-dose thiazide diuretic is the drug of first choice in those without contraindications.	A

RECOMMENDATIONS: MANAGEMENT OF BLOOD PRESSURE (CONTINUED)	
Intensive blood pressure management is required (with early consideration of an ACE-inhibitor) in all people with diabetes due to the increased risk of renal complications.	A
More than one drug is frequently required to lower blood pressure to optimum levels.	B
Aggressive blood pressure control is indicated in people with diabetes and overt nephropathy, or diabetes and microalbuminuria or diabetes and other renal disease.	A
People with diabetes and overt nephropathy or diabetes and confirmed microalbuminuria should be started on an ACE-inhibitor or A2 receptor-blocker (if there are no contraindications) irrespective of blood pressure levels.	A
Most of the treatment benefit is achieved by reaching the following blood pressure levels: • 140/85 mm Hg* in people without clinical cardiovascular disease • 130/80 mm Hg* in people with diabetes or cardiovascular disease.	A
A blood pressure lower than 130/80 mm Hg is preferable for people with diabetes and overt nephropathy or diabetes with other renal disease.	✓

BLOOD PRESSURE AND CARDIOVASCULAR RISK

Blood Pressure

The average of two sitting blood pressure measurements is recommended at the initial risk assessment. This should be repeated on three separate occasions to obtain a baseline prior to the initiation of either intensive lifestyle modification or drug treatment.

Secondary Causes of Raised Blood Pressure

Secondary causes of raised blood pressure include high alcohol intake, sleep apnoea, oestrogen and glucocorticoid administration, anti-inflammatory agents, cyclosporin, liquorice intake and use of sympathomimetics. Rarer causes that require further investigation are renal disease, coarctation of the aorta, renal artery stenosis, phaeochromocytoma, Cushing's syndrome and Conn's syndrome.

*Where risk factor thresholds are given these should be interpreted as approximate guides to clinical practice only.

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Table 16: Recommended method of blood pressure measurement

1	Use a device with validated accuracy that is properly maintained and calibrated
2	Measure sitting blood pressure routinely. Measure sitting and standing blood pressure in the elderly or people with diabetes
3	Remove tight clothing, support arm with blood pressure cuff at heart level, and ensure the hand is relaxed
4	Use cuff of appropriate size for arm circumference (see Table 17)
5	Inflate the cuff until the radial pulse is no longer palpable
6	Lower mercury slowly, by not greater than 2 mm Hg per second
7	Read blood pressure to the nearest 2 mm Hg
8	Measure diastolic blood pressure as disappearance of sounds (phase 5)
9	Two measurements at a single visit are sufficient for calculating cardiovascular risk
10	At least two measurements should be made at each of three visits to determine blood pressure thresholds if considering treatment – some of these can be recorded at nurse consultations using this measurement technique
11	Possible indications for 'home' or ambulatory blood pressure monitoring include the diagnosis of 'white coat hypertension', suspected hypotension, excessive blood pressure variability and resistance to drug therapy
12	Home-based measurement may be lower than office measurement and therefore treatment decisions should be based predominantly on office measurement

Adapted from *Guidelines for the Management of Hypertension: report of the third working party of the British Hypertension Society*.[372]

Table 17: Acceptable blood pressure cuff dimensions for arms of different sizes

Cuff	Arm circumference range at midpoint (cm)	Bladder Width (cm)	Bladder Length (cm)
Newborn	<6	3	6
Infant	6-15*	5	15
Child	16-21*	8	21
Small adult	22-26	10	24
Adult	27-34	13	30
Large adult	35-44	16	38
Adult thigh	45-52	20	42

Source: Perloff et al.[196]

*To approximate the bladder width:arm circumference ratio of 0.40 more closely in infants and children, additional cuffs are recommended.

Table 18: Recommended investigations prior to treatment of raised blood pressure

Investigation	Rationale
Full blood count*	An elevated mean cell volume may indicate alcohol excess
Serum creatinine*	A normal creatinine level does not exclude renal disease. An elevated level may require further investigation. Baseline values are valuable for future reference
Potassium and sodium*	Hyperkalaemia may be due to renal failure or drug causes, haemolysis in transit is a common cause Hypokalaemia may be due to diuretic therapy or very rarely renovascular disease. The possibility of primary aldosteronism and Cushing's syndrome should be borne in mind
Gamma glutamyl transpeptidase (γGT)*	An elevation suggest the possibility of alcohol abuse
Serum urate*	May be useful as a baseline before initiating diuretic therapy or when starting aspirin
Mid-stream urine sample	Proteinuria, microscopic haematuria and casts may indicate renal disease
Thyroid stimulating hormone (TSH)	Raised blood pressure can rarely be associated with either hyper- or hypo-thyroidism
Serum calcium profile	Should be measured to exclude hyperparathyroidism if this is clinically suspected. This is rarely associated with hypertension in some patients

Adapted from the SIGN Guideline *Hypertension in Older People: a national clinical guideline.*[373]

Benefits of Blood Pressure Lowering Intervention

Prospective studies demonstrate a constant liner relationship between relative cardiovacsular risk and blood pressure levels of about 115/70 to 170/100 mm Hg. Within this range, treatment results in similar relative benefits regardless of the baseline blood pressure.[74]**(2++)** People at greater cardiovascular risk derive the most absolute benefit from treatment.

*Recommended as routine.

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DIETARY INTERVENTIONS THAT REDUCE BLOOD PRESSURE

RECOMMENDATIONS: DIETARY INTERVENTIONS FOR BLOOD PRESSURE LOWERING	
Dietary intervention is strongly recommended as an integral component of the management of elevated blood pressure.	A
Advise an eating plan low in total fat, saturated fatty acids, and dietary cholesterol, and rich in fruits, vegetables, and low-fat dairy products.	A
People who are overweight or obese should be offered appropriate weight loss interventions.	A
Identify excessive alcohol intake and advise reduction or substitution with non-alcoholic beverages.	A
Assist the individual to reduce sodium intake to no more than 2 g per day (6 g sodium chloride).	A
Individualise nutritional counselling and other lifestyle changes to complement prescribed risk factor modifying pharmacological agents to reduce cardiovascular risk.	✓

EVIDENCE STATEMENTS

- Cardioprotective dietary patterns featuring multiple food and nutrient components lower blood pressure.(**1+**)
- A 5 to 10% body-weight loss in people who are overweight or obese lowers blood pressure.(**1+**)
- Everyone should be counselled to avoid drinking alcohol to excess.(**1+**)
- Sodium reduction lowers blood pressure and improves the response to blood pressure lowering medications.(**1+**)

Cardioprotective Dietary Patterns

Lifestyle factors, such as dietary patterns, contribute to the continued high prevalence of elevated blood pressure.[374] Equally, a range of dietary patterns have been shown to lower blood pressure.[113,375-377](**1+**) A dietary pattern low in total fat, saturated fatty acids, and dietary cholesterol, and rich in fruits, vegetables, and low-fat dairy products can produce blood pressure reductions exceeding 11/5 mm Hg in people at higher cardiovascular risk.[375](**1+**) Weight loss, the restriction of dietary sodium, and regular intake of oily fish may enhance these effects.[378,379] The macronutrient composition of the diet for the management of elevated blood pressure will rely on the individual metabolic profile, body weight and shape, and dietary preferences.(**4**)

Energy Balance

The prevalence of high blood pressure in a population increases with higher BMI.[380] A meta-analysis of 18 trials has shown that weight loss accomplished by dietary interventions can lower blood pressure

even if 'desirable' body weight is not achieved.[381] [**1-**] In people with elevated blood pressure, a body weight loss in the range of 5 to 10% is associated with an average blood pressure reduction of 3/3 mm Hg. The pooled data from this meta-analysis did not reach statistical significance. However, considering the totality of evidence, it would be prudent to recommend weight reduction for the management of elevated blood pressure (consistent with the recommendations in other published clinical guidelines).[198,382]

Alcohol

Assessment of alcohol intake should be an important part of routine medical assessments. In people who drink excessive quantities of alcohol, reduction in alcohol intake will lower blood pressure.[383] (**1+**) Appropriate national guidelines have been established by the Alcohol Liquor Advisory Council (see Table 6).

Minerals

Sodium

Dietary sodium or salt (sodium chloride) intake is positively associated with level of blood pressure, and prevalence of elevated blood pressure, within, and across populations.[384,385] Six meta-analyses and one systematic review report that a reduction in sodium intake lowers blood pressure.[386-392] (**1++**) There is support for a dose-response relationship, suggesting the lower the sodium intake the greater the effect on blood pressure. A 100 mmol/day reduction in sodium (2 g/day sodium or 6 g/day sodium chloride) predicts a fall in blood pressure of 7/4 mm Hg in people with elevated blood pressure and 4/2 mm Hg in people with lower blood-pressure levels.[389] Lower sodium diets also seem to allow people with elevated blood pressure to reduce medication.[387] Up to 85% of dietary sodium may come in hidden form in processed and manufactured foods, while only 15% is potentially accounted for when added in cooking or from the salt shaker at the table. It is recognised that assistance from food manufacturers will be required if people are to achieve reduced sodium intakes in their diets. If salt is used in the home, it should be iodised salt.

Calcium

Dietary calcium levels below national recommended intakes may increase risk of developing high blood pressure.[393] Health care practitioners should encourage people to regularly consume low saturated fatty acids food sources of calcium, principally from milk and milk products. Some people will have increased dietary requirements (older women 1000 mg/day, pregnant women 1300 mg/day, and lactating women 1400 mg/day). A meta-analysis of six trials examined the use of calcium supplements (median 1076 mg/day) to improve calcium intake and lower blood pressure in high-risk individuals. This reported a modest reduction in blood pressure of 4/1.5 mm Hg.[394] (**1-**) The data are limited to a few studies of short duration and do not adequately support a rationale for use of calcium supplements to lower blood pressure.

Potassium

A meta-analysis of 33 trials examined the use of potassium supplements (median 2925 mg/day) to improve potassium intake and lower blood pressure in high-risk individuals. This reported a modest reduction in blood pressure, 4.4/2.5 mm Hg.[395] (**1+**) A number of studies have methodological flaws and few consider baseline dietary intake. As such, efforts are best directed toward ensuring people are meeting the recommended dietary intake for potassium (approximately 1950 to 5460 mg/day) from food sources.

Other Dietary Factors

Dietary Fat

While the quality of dietary fat is important for the management of cardiovascular risk, there is insufficient evidence to define a specific role in reducing blood pressure. Epidemiological studies are equivocal with respect to dietary fat.[396] Meta-analysis of controlled trials examining omega-3 PUFA in the form of EPA and DHA supplements suggest a modest blood pressure lowering effect.[397](**1+**) High intakes of EPA and DHA (median 3.7 g/day) may lower blood pressure in older people, 2.5/2.3 mm Hg, and those at higher cardiovascular risk, 4.0/2.7 mm Hg. However, many small trials with large reductions in blood pressure may overstate the effect of these supplements. It remains to be determined if EPA and DHA supplements are more beneficial than the whole fish from which they are derived (see Chapter 5, *Intervention: Cardioprotective Dietary Patterns* for discussion on quality of fish oil supplements).

Vegetables and Fruits

There are surprisingly few well-controlled dietary trials specifically examining the role of vegetables and fruits in lowering blood pressure. Where compliance has been adequately achieved, a daily increase of 1.4 servings (112 g/day), from 3.4 (272 g/day) servings per day up to 4.8 (384 g/day) servings per day, has lowered blood pressure 4.0/1.5 mm Hg.[398](**1+**) These findings are corroborated by the vegetable and fruit intervention arm of the DASH diet, where these foods reduced blood pressure 2.8/1.1 mm Hg.[375](**1+**) Non-starch polysaccharide and mineral content may explain why a diet rich in vegetables and fruits can reduce blood pressure.

Caffeine

Cessation of caffeine consumption or a change to decaffeinated coffee in habitual caffeine consumers may lower ambulatory blood-pressure levels among low-risk individuals.[399-404](**1+**) The evidence is less clear for those at higher cardiovascular risk and a recommendation cannot be made until further studies are conducted within this population group.(**I**)

Liquorice

Regular consumption of liquorice is a well-known cause of hypermineralocorticoidism. This is characterised by high blood pressure, sodium retention and hypokalaemia. The active component of liquorice responsible for this action is glycyrrhizic acid. There is a wide range of glycyrrhizic acid concentrations in liquorice which complicates the comparison of studies. A dose-response relationship between liquorice and blood pressure has been reported. In healthy people 100 g/day can cause a moderate and reversible (after withdrawal of the liquorice) rise in systolic blood pressure (6.5 mm Hg).[405]

DRUG INTERVENTIONS THAT LOWER BLOOD PRESSURE

Benefits of Blood Pressure Lowering Medication

Randomized controlled trials have shown that lowering blood pressure reduces the risk of both cardiovascular and total mortality, without adverse effect on quality of life. These trials show a similar relative reduction in coronary heart disease risk of 15 to 25% and reduction in ischaemic stroke risk of 30 to 40%.[140,406-410](**1++**) However, there have been no randomized controlled trials of blood pressure

lowering in asymptomatic people without cardiovascular disease or diabetes and with blood pressure less than 150/90 mm Hg at entry.

Randomized controlled trials show a benefit in treating people with cardiovascular disease or diabetes irrespective of baseline blood pressure.[140,407,411](**1++**)

Choice of Blood Pressure Lowering Medication

All the medications that lower blood pressure have similar efficacy and all have the potential for side effects. The use of low-dose combination therapies can maximise effectiveness and help minimise side effects. The choice of medication depends on the medical history and individual contraindications for particular medications (see Table 19).

Randomized controlled trials have shown that thiazide diuretics are at least as effective as other agents in reducing cardiovascular risk.[412](**1++**) A network meta-analysis of 42 clinical trials has shown that thiazides are effective in reducing a wide variety of clinical endpoints and confirmed these drugs as appropriate first-line treatment in the majority of people at increased cardiovascular risk.[413](**1++**)

There are limited data showing that angiotensin 2 receptor-blockers (A2 receptor-blockers) are as effective as other antihypertensive agents.[408](**1++**) However, A2 receptor-blockers should usually be tried in those who are ACE-inhibitor intolerant before alpha-blockers. A2 receptor-blockers may be better than beta-blockers in high-risk people with left ventricular hypertrophy.

Alpha-blockers are less effective in reducing blood pressure than other agents and are associated with a greater incidence of heart failure when compared to thiazide diuretics.[414](**1+**) Therefore, alpha-blockers, are recommended as second-line treatment after other blood pressure lowering medications have been tried. Caution is required when prescribing alpha-blockers in people aged 75 years and over because of the tendency of these drugs to cause postural hypotension.

After Myocardial Infarction

Randomized controlled trials have shown that treating people after a coronary event with a beta-blocker (propranolol, timolol and metoprolol) reduces total mortality, cardiovascular mortality and morbidity.[415](**1++**) Treating those with left ventricular dysfunction after myocardial infarction with an ACE-inhibitor also reduces mortality and recurrent infarction.[416](**1++**)

After Stroke or Transient Ischaemic Attack

One randomized controlled trial has shown that a combination of ACE-inhibitor and thiazide diuretic reduces recurrent stroke and other major vascular events in people after ischaemic stroke or transient ischaemic attack. This trial excluded people within 14 days of stroke onset.[407](**1++**) There is currently insufficient evidence to determine whether the benefit is specific to the combination of drugs used in this trial or if other blood pressure lowering drugs are equally effective. A 12/5 mm Hg reduction in blood presssure was achieved in the combination therapy group. There are likely to be further reductions in risk with further reduction in blood pressure down to the population optimal level of 115/70 mm Hg. Individualising treatment targets for people after a stroke should take into account the number and dose of medications prescribed as well as co-morbidities and general frailty.

People with Diabetes

In people with diabetes and with uncomplicated raised blood pressure, ACE-inhibitors, thiazide diuretics, beta-blockers and calcium channel blockers are all effective in lowering blood pressure and reducing cardiovascular risk. The majority of people with diabetes will require more than one antihypertensive agent in order to achieve acceptable cardiovascular risk reduction.

ACE-inhibitors should be considered as first-line therapy in people with microalbuminuria, diabetes and overt diabetic nephropathy (albumin:creatinine ratio ≥30 mg/mmol) or diabetes with other renal disease, irrespective of the blood pressure level. A2 receptor-blockers exert a nephroprotective effect and are indicated if ACE-inhibitors are not tolerated or contraindicated. A2 receptor-blockers can be considered for combination therapy with an ACE-inhibitor to achieve blood pressure targets. People with diabetes who have had a cardiovascular event should be considered for both a beta-blocker and an ACE-inhibitor. ACE-inhibitors should be considered early in all people with diabetes (and especially for Māori and Pacific people who have a high incidence of end-stage renal failure) as they reduce cardiovascular risk in those at high risk and are also nephroprotective for those with microalbuminuria or overt diabetic nephropathy.

People aged 75 years and over

People aged 75 years and over have a higher cardiovascular risk because of their age and therefore have a greater potential to benefit from treatment. They appear to tolerate antihypertensive treatments as well as younger age groups and in general the range of blood pressure treatments available are equally effective. A low-dose thiazide diuretic is generally the first drug of choice[417-419] but monitoring for electrolyte disturbance is recommended. Beta-blockers and ACE-inhibitors can be used in this group of people. Long-acting dihydropyridine calcium channel blockers are a suitable alternative in people aged 75 years and over with isolated systolic hypertension when thiazides are contraindicated or poorly tolerated.[420]

People aged 75 years and over with isolated raised systolic blood pressure (systolic blood pressure greater than 160 mm Hg with a diastolic blood pressure less than 90 mm Hg) are at increased risk of ischaemic stroke and should be managed vigourously.[417](**1++**)

In Pregnancy

The choice of drugs is restricted to those which are safe for the foetus. Methyldopa and hydralazine have been traditionally used but are commonly associated with side effects. Labetalol, some beta-blockers (notably propranolol, atenolol and oxprenolol which have reasonable evidence of safety) and nifedipine have been used successfully in pregnancy. Thiazide diuretics, ACE-inhibitors and A2 receptor-blockers should be avoided.

Table 19: Indications and contraindications for the use of various drug classes to lower blood pressure

Condition	Drug Therapy		
	Compelling evidence for use	**May have favourable effects**	**May have unfavourable effects**
Angina		Beta-blockers Calcium channel blockers	
Aortic dissection	Beta-blockers		
Atrial tachycardia and fibrillation		Beta-blockers Calcium channel blockers (rate limiting)	
Asthma and CORD			Beta-blockers (contraindicated)
Depression			Beta-blockers
Diabetes mellitus			Thiazide diuretics (high dose)
Type 2		Low- dose diuretics ACE-inhibitors A2 receptor-blockers Beta-blockers Calcium channel blockers	
People with diabetes and microalbuminuria	ACE-inhibitors A2 receptor-blockers	Calcium channel blockers (rate limiting)	Potassium-sparing diuretics
Dyslipidaemia		Alpha-blockers	Diuretics (high dose)
Essential tremour		Beta-blockers (non-cardioselective)	
Gout and hyperuricaemia		A2 receptor-blockers	Diuretics
Heart block Secondary or tertiary			Beta-blockers (contraindicated) Rate limiting Calcium channel blockers (contraindicated)
Heart failure	ACE-inhibitors Diuretics Beta-blockers A2 receptor-blockers		Calcium channel blockers (except amlodipine, felodipine)
Isolated systolic hypertension	Diuretics Calcium channel blockers (long-acting dihydropyridine)		
Liver disease			Labetalol
Myocardial infarction	Beta-blockers ACE-inhibitors		
Osteoporosis		Thiazides	
Peripheral vascular disease			Beta-blockers*
Pregnancy			ACE-inhibitors (contraindicated) A2 receptor-blockers (contraindicated)
Prostatism		Alpha-blockers	
Renal insufficiency#		ACE-inhibitors A2 receptor-blockers	Potassium-sparing diuretics
Renovascular disease			ACE-inhibitors A2 receptor-blockers

*Beta-blockers should not be used in people with critical ischaemia (rest pain or impending gangrene).
#Caution in people with renovascular hypertension and creatinine greater than 150 mmol/L.

Adapted from SIGN: Hypertension in the Older Person.[373]

THE OVERALL GOAL OF INTERVENTION

The goal for everyone is to reduce 5-year cardiovascular risk to less than 15%.

The intensity of treatment to achieve this goal should be related to pre-treatment cardiovascular risk. This goal may be achieved more easily by the simultaneous reduction in several risk factors. The higher the level of risk, the greater should be the effort made to achieve lower blood pressure levels. An individual's risk factor levels should always be interpreted within the context of their calculated cardiovascular risk.

Examples of Specific Targets

People with very high risk (5-year cardiovascular risk >20%) determined clinically with:

- **a previous history of cardiovascular disease**
- **the specific lipid disorders of a genetic basis (FH, FDB or FCH)**
- **diabetes and overt diabetic nephropathy or diabetes with other renal disease.**

People with clinically determined risk greater than 20% should start intensive lifestyle advice concurrently with drug therapy. All efforts should be made to reach a systolic blood pressure less than 130 mm Hg and a diastolic blood pressure less than 80 mm Hg. In people with diabetes and overt diabetic nephropathy (albumin:creatinine ratio greater than 30 mg/mmol) or diabetes with other renal disease all efforts should be made to reach a lower blood pressure. For people after a transient ischaemic attack or stroke systolic blood pressure should be reduced irrespective of starting levels by at least 10 mm Hg.

People with a calculated 5-year cardiovascular risk greater than 15%

The goal for people with risk greater than 15% is to reduce 5-year cardiovascular risk to less than 15%. In this group, blood pressure lowering treatment should aim to achieve a systolic blood pressure less than 140 mm Hg and diastolic blood pressure less than 85 mm Hg.

People with blood pressure consistently over 170/100 mm Hg

People with blood pressure consistently over 170/100 mm Hg should have their cardiovascular risk calculated and receive specific lifestyle advice and blood pressure lowering treatment.

Table 20: Target blood pressure levels

	Systolic Blood Pressure	Diastolic Blood Pressure
People without clinical cardiovascular disease	<140 mm Hg*	<85 mm Hg*
People with diabetes or cardiovascular disease	<130 mm Hg*	<80 mm Hg*
People with diabetes and overt nephropathy, diabetes and microalbuminuria or diabetes with other renal disease	Aggressive blood pressure control is recommended	

*Where risk factor thresholds are given these should be interpreted as approximate guides to clinical practice only

MONITORING AND DURATION OF TREATMENT

RECOMMENDATIONS: MONITORING AND DURATION OF TREATMENT	
Lifelong treatment is advised for people prescribed medication or intensive lifestyle advice.	✓
Side effects of blood pressure lowering treatment are uncommon, but the possibility of drug-specific unwanted effects should be discussed prior to treatment.	✓
For those on drug treatment, blood pressure monitoring is recommended every 3 months until the blood pressure is controlled, then every 6 months. Ongoing monitoring of creatinine and electrolytes is advisable for people with high initial values, persistent elevated blood pressure or in those taking diuretics, ACE-inhibitors or A2 receptor-blockers.	✓
People with diabetes who are receiving medication should have their blood pressure monitored every 3 months until adequate control is achieved, then every 6 months.	✓

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INTERVENTION: ANTIPLATELET THERAPY

RECOMMENDATIONS: ANTIPLATELET THERAPY FOR PEOPLE WITHOUT CLINICAL CARDIOVASCULAR DISEASE	
Everyone with a 5-year cardiovascular risk greater than 15%, should be started on low-dose aspirin (75 – 150 mg/day) if there are no contraindications.	A
Aspirin is contraindicated in people with aspirin allergies or intolerance, active peptic ulceration, uncontrolled blood pressure and in people with other major bleeding risks.	A

ASPIRIN

Population-based observational studies have found that regular use of aspirin (at a dose of <300 mg/day) is associated with around a 2-fold increased risk of upper gastrointestinal bleeding (or perforation).[421](**2++**) The decision to use aspirin should be based on a balance of the risks and benefits for each person taking into account their absolute risk of an event.

The cardiovascular benefits of low-dose aspirin outweigh the harm in people with a 5-year cardiovascular risk greater than 15%.[422,423](**1++**) However, the risk of a significant bleed or major haemorrhage with aspirin outweighs the benefit for people with a 5-year cardiovascular risk of less than 15%.[422](**1++**)

Low-dose aspirin (75 – 150 mg/day) is as effective as higher daily doses and may be associated with less bleeding.[424](**1++**)

OTHER ANTIPLATELET AGENTS

When compared with aspirin in patients with pre-existing cardiovascular disease, clopidogrel reduced ischaemic stroke, myocardial infarction, or vascular death by a further 8.7% (95% CI, 0.3 to 16.5%, p=0.034). People treated with clopidogrel had an annual 5.32% risk of these events compared with 5.83% of people treated with aspirin.[425]

Recommendations on the management of atrial fibrillation and the use of warfarin to prevent stroke in people with atrial fibrillation are contained in a separate guideline. This guidelines will be available at www.nzgg.org.nz

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INTERVENTION: COMPLEMENTARY AND ALTERNATIVE THERAPIES

RECOMMENDATIONS: COMPLEMENTARY AND ALTERNATIVE THERAPIES	
Clinicians should enquire about the use of alternative and complementary medicines when assessing cardiovascular risk or prescribing medication.	✓
There is insufficient evidence to recommend the following complementary and alternative therapies for the treatment or prevention of cardiovascular disease: • herbal medicines, botanicals[426] • garlic[427-429]/ginkgo biloba/rosemary/horse-chestnut seeds/ xin bao • acupuncture[426] • chelation[430] • oriental medicine • aromatherapy • homeopathy • hypnosis • meditation • yoga/tai chi • intercessory prayer[431] • Strauss heart drops	I
Feverfew, garlic, ginkgo biloba, ginger, and ginseng may alter bleeding time and should not be used concomitantly with warfarin.[432]	C
St John's Wort reduces serum digoxin levels and can enhance the metabolism of warfarin.[433]	C
Herbs (eg, karela and ginseng) may affect blood glucose levels and should not be used in people with diabetes mellitus.[432]	C

Complementary and alternative therapies are used widely by people and practitioners in New Zealand and overseas.[434-439](3)

There is currently no randomized controlled trial evidence that herbal, complementary or alternative therapies reduce cardiovascular mortality or morbidity.

There is randomized controlled trial evidence that vitamin supplementation with antioxidant vitamins (beta-carotene, vitamin C and vitamin E) do not reduce cardiovascular risk.[128,440](1++)

A meta-analysis of antioxidant vitamins has shown that beta-carotene led to a small but significant increase in all-cause mortality (7·4 vs 7·0%, OR 1·07(95% CI, 1·02 to 1·11) p=0·003) and a slight increase in cardiovascular death (3·4 vs 3·1%, OR 1·1(95% CI,1·03 to 1·17) p=0·003).[441]

Some herbal medicines have the potential for toxic effects[432](**1+**) and some interact with medication eg, warfarin.[432,442,443](**3**)

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MANAGEMENT OF PEOPLE WITH DIABETES, HYPERGLYCAEMIC STATES OR THE METABOLIC SYNDROME

RECOMMENDATIONS: REDUCING CARDIOVASCULAR RISK	
The higher the calculated cardiovascular risk, the more aggressive the management of modifiable risk factors, including diabetes, should be.	C
Everyone with diabetes should be offered risk factor treatment to lower their 5-year cardiovascular risk to less than 15%. Where possible treatment should aim to achieve optimal levels: LDL-C less than 2.5 mmol/L, blood pressure less than 130/80 mm Hg, HbA1c less than 7%.	C
Everyone with diabetes or the metabolic syndrome should receive intensive lifestyle advice. Lifestyle changes that have been shown to benefit people with these risk profiles include: • dietary change (A) • smoking cessation (A) • physical activity (B).	A
Intensive dietary advice should be given in individual/group sessions with a dietition.	A
A cardioprotective dietary pattern is strongly recommended as an integral component of diabetes management.	A
The optimal level of HbA1c is as close to physiological levels as possible, preferably less than 7% for most people.	B
Due to the increased risk of renal complications, intensive blood pressure management is required (with early consideration of an ACE-inhibitor) in all people with diabetes.	A
More than one drug is frequently required to lower blood pressure to optimum levels.	B
Aggressive blood pressure control is indicated in people with diabetes and overt nephropathy, diabetes and confirmed microalbuminuria or diabetes with other renal disease.	A

RECOMMENDATIONS: REDUCING CARDIOVASCULAR RISK (CONTINUED)	
People with diabetes and overt nephropathy or diabetes with confirmed microalbuminuria should be started on an ACE-inhibitor or A2 receptor-blocker (if there are no contraindications) irrespective of blood pressure levels.	A
Most of the treatment benefit is achieved by reaching the following blood pressure levels: • 140/85 mm Hg in people without clinical cardiovascular disease • 130/80 mm Hg in people with diabetes or cardiovascular disease.	A
A blood pressure lower than 130/80 mm Hg is preferable for people with diabetes and overt nephropathy or other renal disease.	✓

DIAGNOSTIC CRITERIA FOR TYPE 2 DIABETES, IGT AND IFG

RECOMMENDATIONS: DIAGNOSTIC CRITERIA FOR TYPE 2 DIABETES, IGT AND IFG	
Two fasting venous plasma glucose results greater than or equal to 7 mmol/L on two different days are diagnostic of diabetes and do not require an OGTT.	C
A random venous plasma glucose result of greater than 11 mmol/L on two different days is diagnostic of diabetes.	C
A fasting venous plasma glucose of 6.1 to 6.9 mmol/L indicates impaired fasting glycaemia and an OGTT is recommended to look for diabetes or IGT.	C
Some people with a fasting venous plasma glucose of 5.5 to 6.0 mmol/L show diabetes or IGT with an OGTT.	C
An OGTT is recommended in people with a fasting glucose of 5.5 to 6.0 mmol/L who are not of European ethnicity or who have a family history of diabetes, a past history of gestational diabetes or the other features of the metabolic syndrome.	C
A fasting venous plasma glucose result of less than 5.5 mmol/L is normal.	C
HbA1c should not be used for the diagnosis of diabetes.	✓

The WHO has defined values for the diagnosis of different categories of hyperglycaemia, diabetes mellitus, IGT, and IFG. Several clinical phases, both asymptomatic and symptomatic, form a continuum during the natural history of the disease.

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Table 21: Diagnosis of diabetes and other hyperglycaemic states

	Blood test	Result Venous plasma glucose concentration (mmol/L)
Diabetes mellitus	Fasting OR 2-hour post glucose load OR both	≥7 ≥11.1
IGT	Fasting (if measured) AND 2-hour post glucose load	<7.0 ≥7.8 and <11.1
IFG	Fasting AND (if measured) 2-hour post glucose load	≥6.1 and <7.0 <7.8

For epidemiological or population screening purposes, the fasting or 2-hour value after 75 g oral glucose may be used alone. For clinical purposes, the diagnosis of diabetes should always be confirmed by repeating the test on another day unless there is unequivocal hyperglycaemia with acute metabolic decompensation or obvious symptoms (thirst or polyuria).

Table 22: Action following the fasting venous plasma glucose

Result	Action	Reason
7.0 mmol/L or more	Repeat a fasting plasma glucose	Two results above this level, on separate occasions, are diagnostic of diabetes and do not require an OGTT
6.1 to 6.9 mmol/L	Request an OGTT	This level is diagnostic of IFG. Diabetes or IGT have not been excluded
5.5 to 6.0 mmol/L	Request an OGTT in high risk groups*	At this level, the result may be normal, but some patients will show diabetes or IGT in an OGTT
5.4 mmol/L or less	Retest in 5 years	This result is normal

A fasting venous plasma glucose is recommended for a comprehensive cardiovascular risk assessment. Where the non-fasting test has been performed, a fasting test is recommended if the non-fasting result is greater than 5.5 mmol/L, especially when the sample was taken more than 2 hours after a meal.

HbA1c is not a reliable test for either diagnosis or screening for diabetes. Although the reproducibility of the HbA1c test within individual laboratories often meets the minimum requirement for monitoring, standardisation between laboratories remains a problem. The HbA1c test should only be used for monitoring glycaemic control.

Point-of-care capillary testing is not recommended as part of a comprehensive cardiovascular risk assessment or as a formal screening procedure.

*Within the age ranges and context of a cardiovascular risk assessment - defined as people who are not of European ethnicity or who have a first degree relative with diabetes, or a past history of gestational diabetes or features of the metabolic syndrome

DIAGNOSTIC CRITERIA FOR THE METABOLIC SYNDROME

There is no ideal definition for the metabolic syndrome (the insulin resistance syndrome). The National Cholesterol Education Programme (NCEP) definition shown in Table 23 is a useful clinical definition despite its limitation of using categorical variables for risk factors which form part of a continuum. The WHO criteria have suggested that abnormal glucose metabolism (IGT or IFG) is an essential component of the condition.

The prevalence of the metabolic syndrome among adults aged over 20 years in the United States is 24%.[446] The prevalence of the metabolic syndrome in New Zealand is unknown, but with increasing truncal obesity, awareness of this condition in clinical practice is important.

People from the Indian subcontinent are more likely to demonstrate features of the metabolic syndrome at lower waist circumferences than those of European descent.**(4)**

There are currently no data available on the prevalence of the metabolic syndrome in Māori, Pacific peoples or people from the Indian subcontinent.

Three or more of the five risk factors in Table 23 are required for a diagnosis of metabolic syndrome. Where risk factor thresholds are given these should be interpreted as approximate guides to clinical practice only.

Table 23: A definition of the metabolic syndrome

Risk Factor	Sex	Defining level
Abdominal Obesity	Men Women	≥100 cm waist circumference* ≥90 cm waist circumference*
Triglycerides		≥1.7 mmol/L
HDL Cholesterol	Men Women	<1.0 mmol/L <1.3 mmol/L
Blood Pressure		SBP ≥130 or DBP ≥85
Fasting Glucose		≥6.1 mmol/L

Adapted from the National Cholesterol Education Programme: ATPIII.[61]

*More details on how to measure waist circumference are found in Chapter 7, *Intervention: Weight Mangement.*

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DIABETES AND CARDIOVASCULAR RISK

RECOMMENDATIONS: CARDIOVASCULAR RISK ASSESSMENT IN PEOPLE WITH DIABETES OR AT HIGH RISK OF DIABETES	
All people with diabetes should be offered annual comprehensive cardiovascular risk assessments from the time of diagnosis.	C
Māori, Pacific peoples and people from the Indian subcontinent should be offered cardiovascular risk assessment from 35 years for men and 45 years for women.	C
Men and women at higher risk of diabetes should be offered cardiovascular risk assessment from 35 years for men and 45 years for women. These people are those with one or more of the following risk factors: • a family history of diabetes in a first-degree relative (parent or sibling) • a personal history of gestational diabetes • a personal history of polycystic ovary syndrome • known IGT or IFG (see Table 21) • obesity (BMI ≥30*) or truncal obesity (waist circumference ≥100 cm* in men or ≥90 cm* in women).	C
People with diabetes and overt diabetic nephropathy (albumin:creatinine ratio greater than 30 mg/mmol) or diabetes with other renal disease are classified clinically at very high risk (5-year cardiovascular risk ≥20%) without a cardiovascular risk calculation.	B
All other people with diabetes should have their 5-year cardiovascular risk calculated using the risk tables.	B
Certain people with diabetes or the metabolic syndrome are at increased cardiovascular risk and should be moved up one risk category as part of the cardiovascular risk assessment. These include: • people with diabetes and microalbuminuria • people with type 2 diabetes 10 years after diagnosis • people with HbA1c results consistently above 8% • people who meet the definition of the metabolic syndrome.	C
Everyone with diabetes should have uric acid levels as well as renal and liver-function tests performed at the time of a cardiovascular risk assessment.	C

Cardiovascular Risk in People with Hyperglycaemia

IGT is more strongly associated with the risk of cardiovascular disease than IFG.[447] For this reason an OGTT is recommended if a fasting plasma glucose is elevated (≥5.5 mmol/L). Hyperinsulinaemia has also been shown to predict coronary heart disease in healthy middle-aged men.[448] At present these hyperglycaemic states are not included in the cardiovascular risk assessment.

Cardiovascular Risk in People with The Metabolic Syndrome

The term 'metabolic syndrome' is used synonymously with the 'insulin resistance syndrome'.The cluster of clinical features that define the metabolic syndrome identifies individuals at increased risk of cardiovascular disease despite only moderate elevations in individual risk factors.**(2+)** People with

* Where risk factor thresholds are given these should be interpreted as approximate guides to clincial practice only.

the metabolic syndrome are at increased risk of ischaemic heart disease, raised blood pressure, sub-fertility, diabetes, gout, heart failure and obesity.(**2+**)

Cardiovascular Risk in People with Diabetes

Everyone with diabetes is classified at higher cardiovascular risk. Prospective studies show the morbidity and mortality from cardiovascular disease is 2 to 5 times higher in people with diabetes.[75,449-452](**2++**) Approximately two-thirds of people with type 2 diabetes die from cardiovascular disease.[452,453](**2++**) The case-fatality rate among those who have a cardiovascular event is higher in people with diabetes than in those without diabetes.[454](**2++**)

Cardiovascular risk is associated with the duration of diabetes, glycaemic control, diabetic renal disease and the presence of other cardiovascular risk factors. The association between HbA1c level and cardiovascular disease is continuous.[455] Each 1% reduction in HbA1c is associated with a 21% (95% CI, 15 – 27%) reduction in the risk of diabetes-related death and a 14% reduction in the risk of myocardial infarction over 10 years.[77]

People with diabetes and renal complications are at greater cardiovascular risk. Cardiovascular mortality is increased between 2 and 4-fold for people with microalbuminuria[103] and between 5 and 8-fold for people with proteinuria, compared to people without raised albumin levels.[110,111](**2++**) Approximately one third of people with type 2 diabetes and microalbuminuria will die within 5 years.[456](**2++**) When overt diabetic nephropathy is present and blood pressure is over 140/90 mm Hg, the mortality is increased between 11 and 18-fold in people with type 1 diabetes and 2 and 8-fold in people with type 2 diabetes.[110,111](**2++**)

Microalbuminuria

Microalbuminuria is defined as a sustained urinary albumin excretion of 30 to 300 mg/day. An adequate estimate of the daily albumin excretion is provided by the urinary albumin:creatinine ratio (ACR). Microalbuminuria is present if ACR is greater than or equal to 2.5 mg/mmol creatinine in men or greater than or equal to 3.5 mg/mmol in women. A urinary albumin concentration greater than or equal to 20 mg/L also indicates microalbuminuria.

Diabetes and Renal Disease

Measurements of urinary albumin loss and serum creatinine are the best screening tests for diabetic renal disease. Estimates of glomerular filtration rate (GFR) are the best indices of overall kidney function and can be readily estimated from prediction equations. Moderate renal impairment is suggested and referral indicated for people with a serum creatinine greater than or equal to 0.15 mmol/L or a calculated GFR less than 60 ml/min/1.73m^2. More details are described in the New Zealand Guideline, *The Management of Type 2 Diabetes* available at www.nzgg.org.nz

Overt Diabetic Nephropathy

Overt diabetic nephropathy is defined by a raised urinary albumin excretion greater than or equal to 300 mg/day (this is equivalent to an albumin:creatinine ratio ≥30 mg/mmol, or urinary albumin concentration >200mg/L) and represents a more severe and established form of diabetic nephropathy.

Adjustments to Cardiovascular Risk

A more refined assessment of cardiovascular risk for people with diabetes has been modelled using data from the UKPDS study which includes adjustments for HbA1c and duration of diabetes.[63] An electronic calculator is available for this from the Diabetes Trial Unit in Oxford at www.dtu.ox.ac.uk An approximation to this calculated risk can be made on the National Heart Foundation's cardiovascular risk tables by adjusting upwards one category (5%) those with an HbA1c consistently over 8% or duration of diabetes greater than 10 years.[63][**2++**]

Benefits of Intervention in People with Diabetes, Hyperglycaemic States or The Metabolic Syndrome

Lifestyle Interventions

People with diabetes, IGT, IFG or the metabolic syndrome are at higher cardiovascular risk than people without these conditions. There is now evidence that intensive lifestyle interventions (diet and physical activity) can reduce the risk of developing diabetes in those with IGT.[154,155,444](**1++**)

Type 2 diabetes can be seen as the end-product of years of metabolic stress accompanying a state of insulin resistance. The acceleration of atherogenesis begins with the onset of hyperglycaemia before the onset of diabetes.[445] Early detection of hyperglycaemia and/or insulin resistance can prevent the onset of cardiovascular disease.

Lifestyle factors contribute to the development and progression of type 2 diabetes and the metabolic syndrome, and the associated high cardiovascular risk. The primary lifestyle objectives for prevention and management are to limit the rate of loss of beta-cell function and to improve insulin sensitivity, lipid levels, blood pressure, postprandial hyperglycaemia and HbA1c. The cornerstones of lifestyle management are a cardioprotective diet, physical activity and weight management.

Cardioprotective Dietary Patterns

People with diabetes who have high triglycerides and low HDL-C as part of the metabolic syndrome should be considered for lifestyle treatments known to improve this lipid profile. These include a cardioprotective dietary pattern, increased physical activity and fish oil supplements.

The dietary ideal includes a dietary composition that will reduce insulin resistance, limit the rate of loss of beta-cell function and improve insulin sensitivity, dyslipidaemia, blood pressure and postprandial hyperglycaemia.

Weight Management

Modest weight reductions of 5 to 10% of initial body weight, are associated with significant improvements in lipid abnormalities, blood pressure levels, insulin resistance, glycaemic control and HbA1c.[208] A 5 kg weight loss is recommended as an initial goal in people who are overweight or obese (BMI >25). Modest weight loss can be achieved and sustained.[208,457]

Physical Activity

Physical activity is a key component of weight reduction and important for weight maintenance. Moderate physical activity increases maximal oxygen uptake and cardiovascular fitness.[458]

Physical training improves risk factors associated with the metabolic syndrome. Aerobic physical activity, 40 to 65% of VO_2max for 20 to 45 minutes per session, 3 to 4 times weekly, is associated with increased insulin sensitivity and lowers triglyceride levels and clotting factors (plasminogen activator

inhibitor and fibrinolytic activity). The addition of exercise to a weight loss programmes increases loss of intra-abdominal fat.[299](**1+**) Physical fitness is associated with lower blood pressure.[299](**2+**)

Exercise training reduces HbA1c and increases cardiorespiratory fitness in adults with type 2 diabetes. Physical activity with an intensity of 5.5 METs for 40 minutes or more per week is associated with protection from the development of type 2 diabetes.

DIETARY INTERVENTIONS

Dietary Interventions for People with IGT and IFG

RECOMMENDATIONS: DIETARY INTERVENTIONS FOR PEOPLE WITH HYPERGLYCAEMIC STATES	
People who are at risk of type 2 diabetes should avoid weight gain. Offer weight loss advice to people who are overweight or obese.	**A**
Everyone with IGT or IFG should receive intensive dietary advice. Intensive dietary advice should ideally be given in individual/group sessions with a dietitian. Physical activity should also be encouraged.	**A**
Encourage adults at risk of type 2 diabetes to adopt a cardioprotective dietary pattern, reduce saturated fatty acids and increase dietary fibre.	**B**

EVIDENCE STATEMENTS

- Weight gain in adult life is associated with increased risk of type 2 diabetes.(**2++**)
- Intentional weight loss is associated with reduced incidence of type 2 diabetes, IGT and insulin resistance.(**2++**)
- Intensified dietary advice in adults with IGT is effective in normalising glucose tolerance.(**1++**)
- Weight loss can normalise glucose tolerance.(**1++**)
- Individually tailored dietary modification (low-fat, low saturated fatty acids, high-fibre) together with increased physical activity and weight loss can delay the transition from IGT to type 2 diabetes, when compared with conventional care.(**1++**)
- The same interventions are likely to improve IFG.(**4**)
- Lower-fat, higher-fibre dietary interventions and weight loss programmes, especially in conjunction with increased physical activity (aerobic training), improve insulin sensitivity in adults at risk of type 2 diabetes.(**1+**)

Cardioprotective Dietary Patterns

The primary target groups for nutritional interventions that delay the onset of type 2 diabetes are people with IGT, IFG, the metabolic syndrome, a history of weight gain, a family history of diabetes or previous gestational diabetes. These approaches are particularly important for Māori, Pacific

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peoples and people from the Indian subcontinent who have a higher risk of developing diabetes. Current approaches may still underestimate cardiovascular risk in people who have insulin resistance and there is consensus that more intensive interventions are especially indicated for Māori with a high waist circumference.

Prospective cohort studies have demonstrated an association between a number of dietary factors and reduced risk of developing type 2 diabetes. These dietary factors are part of a cardioprotective dietary pattern.

In one study, a 'prudent' dietary pattern has been associated with reduced risk of diabetes, while a 'Western' dietary parttern has been associated with increased risk. A combination of a 'Western' dietary pattern and a BMI greater than 30 was associated with an 11-fold greater risk of developing diabetes compared to a 'prudent' dietary pattern and a BMI greater than 25.[91] **(2+)**

Together, a cluster of lifestyle factors have been associated with minimal risk of developing diabetes over a period of 16 years in women (a risk of <12% of the risk of all other women) including those identified with a family history of diabetes. This lifestyle was defined as a BMI less than 25, a diet high in cereal fibre and polyunsaturated fatty acids and low in transunsaturated fat and glycaemic load, and 30 minutes of moderate to vigorous physical activity daily. Having the optimal dietary score was associated with a 62% reduced risk of diabetes in participants with a BMI less than 25, and 51% reduced risk in those with a BMI greater than 30, compared with lowest dietary score in each group.[459] **(2+)** In this study, BMI was independently associated with incidence of diabetes; having a BMI greater than 30 increased the risk of diabetes 38.8 times compared with a BMI less than 23.

Weight Gain and Weight Loss

A BMI greater than 25 is the most important, modifiable risk factor for the development of type 2 diabetes.

Weight gain is associated with increased risk of diabetes in the general population.[459-465] Relative to people who are overweight and do not gain weight, a gain of one kg of weight per year for 10 years in adults who are overweight is associated with a 49% increased risk of developing diabetes in the next 10 years.[464]

Weight loss is associated with a substantially reduced risk of diabetes in the general population.[464-467] **(2+)** A kilogram of weight lost annually over 10 years in people who were overweight has been associated with a 33% lower risk of developing diabetes over the next 10 years.[464,466,468] Incidence of type 2 diabetes is reported as 33% lower in adults who have a sustained weight loss of 3.7 to 6.8 kg, and by 52% in those losing greater than 6.8 kg, but weight regain restores the usual risk.[467]

Primary prevention studies that target weight loss in subjects with IGT have achieved reductions of 3 to 5.5 kg in the first 1 to 2 years with some regain thereafter. These studies used intensive lifestyle interventions in people with IGT versus usual dietary and physical activity interventions, and have demonstrated greater weight losses and more subjects with a normal glucose tolerance test post-intervention.[154,155,183,469-471] **(1+)**

Weight loss improves measures of insulin resistance or insulin sensitivity in individuals who are overweight or obese.[470,472-476] Prevention of weight gain is therefore integral to the management of IGT and IFG. For such adults an attempt to lose weight is the first-line dietary management goal in order to delay progression to type 2 diabetes.

Intensity of Intervention

Intensive diet and physical activity weight loss programmes that involved personalised goal-setting, individually-tailored lifestyle advice and self-monitoring in adults with IGT have shown a 58% relative reduction in progression to type 2 diabetes over about 3 years, compared with more conventional treatments.[154,155](**1+**) These results have been repeated to a lesser, but also significant, degree in more adherent subjects or more intensive programmes.[183,469,477,478,533] In one study there was a strong inverse correlation between progression to diabetes and the degree that intervention goals were achieved.[154] Improvements were maintained in programmes that included 8 to 18 intervention sessions in the first year, then 2- to 3-monthly contacts during maintenance.[154,155,183](**1+**)

An intensive intervention has been shown to improve insulin sensitivity in adults with insulin resistance compared with a more moderate intervention.[479](**1+**) The intervention was dietary [low-fat (27% of energy), moderately high carbohydrate (51% of energy), contained 30 g of fibre daily (7 g CHO/g fibre)], increased physical activity (aerobic training for 20 minutes five times per week to 80 to 90% of age-predicted maximum heart rate) and weight loss.

Dietary Fat

Insulin sensitivity improves when saturated fatty acids are replaced with carbohydrate[480] or unsaturated fatty acids when total fat contributes less than 37% of energy intake in healthy individuals,[335,480-483] or in subjects with elevated triglyceride levels.[484](**1+**) Higher total fat intake is associated with lower insulin sensitivity among people with obesity.[485]

Intake of dietary fat, in particular saturated fatty acids, animal fat or P:S ratio, has been associated with the development of diabetes,[486-492] hyperinsulinaemia in the general population,[493-495] and with the predisposition to progress from IGT to type 2 diabetes.[486] Vegetable oils, PUFA and oily fish have been inversely associated with incidence of type 2 diabetes.[143,490-492](**2+**)

Dietary Carbohydrate

Higher intakes of whole grain products (6 – 10 servings per day) or dietary fibre (30 g/day) compared with lower intakes, or a lower versus higher dietary glycaemic index, improve insulin sensitivity in people with hyperinsulinaemia, insulin resistance or coronary heart disease risk factors or IGT respectively.[479,496-498](**1+**)

High carbohydrate (60% of energy), high-fibre (40 – 80 g/day, 5 – 8 g CHO/g fibre) diets improve insulin sensitivity, lipid levels and glycaemic parameters, compared with high-fat diets in healthy subjects,[482,484,499] and in individuals with IGT, especially if they exercise.[500](**1+**)

Dietary fibre, especially cereal fibre (7 – 10 g/day), whole grains (3 servings per day) and fruit and vegetables (more than 5 servings per day) are each inversely associated with risk of type 2 diabetes in the general population.[501-508](**2++**) Whole grain or fibre intakes are inversely associated with insulin levels in people without diabetes, particularly in those who are over-weight.[495,496,509-511](**1+**,**2+**)

Incidence of diabetes in the general population is associated with a high dietary glycaemic index or glycaemic load (total CHO intake times the glycaemic index of each contributing food) when carbohydrate intake is high and cereal fibre intake is low, but not when cereal fibre intake is high.[512,513](**2+**) Incidence is not associated with absolute carbohydrate intakes[487,488,495,501,505,514-516] or

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with sucrose intakes.[488,501,514,517] **(2+)** Population carbohydrate intakes are not associated with HbA1c, plasma glucose, or serum insulin concentrations, but are inversely associated with the risk of elevated serum C-peptide.[513]

Dietary Interventions that Improve Glycaemic, Insulin and Lipid Profiles in People with Type 2 Diabetes or The Metabolic Syndrome

RECOMMENDATIONS: DIETARY INTERVENTIONS FOR PEOPLE WITH TYPE 2 DIABETES OR THE METABOLIC SYNDROME	
Recommend a reduction in energy intake with weight loss as the primary objective for people with diabetes or the metabolic syndrome who are overweight or obese.	A
Everyone with type 2 diabetes or the metabolic syndrome should receive intensive dietary advice. Intensive dietary advice should be given in individual/group sessions with a dietitian. Physical activity should also be encouraged.	A
Encourage people with type 2 diabetes or the metabolic syndrome to gradually adopt a cardioprotective dietary pattern. Advise a reduction in the intake of foods rich in saturated fatty acids or added sugars, and white flour bakery products. Encourage a progressive replacement of these foods with vegetables, fruit, whole grain, high-fibre products, and dried peas and beans (legumes). Recommend an increase in the consumption of fish and include a source of polyunsaturated fat (see Table 5).	A
Interventions that are known to reduce risk factors in people without diabetes are also recommended for people with diabetes. Assess salt and alcohol consumption and provide guidance for limited use. Consider adding plant sterols/stanols to the diet.	A
For the optimal improvement of all risk factors, especially body weight and glycaemic control, employ intensive dietary interventions that include continuous education, behaviour modification, goal setting and intensive monitoring.	A
Identify and recommend qualitative dietary changes based on the habitual dietary pattern, and then progress to quantitative advice to promote the development of a structured eating plan.	C
Specific dietary advice for people with diabetes and the metabolic syndrome includes advice about the saturated fatty acid content of foods and the quality of carbohydrate choices to encourage a high-fibre intake of more than 40 g daily (see Table 4).	A
To control postprandial hyperglycaemia the following advice is recommended: • include high-fibre foods with a low to moderate glycaemic index at each meal • distribute carbohydrate foods evenly throughout the day • avoid a large volume of carbohydrate-rich foods at any one meal.	A
All people with diabetes should be referred to a dietitian.	✓

EVIDENCE STATEMENTS

- Intensified dietary intervention improves the dietary intake, parameters of risk and enhances the treatment of individual risk factors with reduction of microvascular complications.(**1+**)
- Weight loss can normalise or improve HbA1c, improve insulin sensitivity and reduce the need for hypoglycaemic medications.(**1++**)
- Reduced energy intake and weight loss improve fasting blood glucose, postprandial blood glucose, HbA1c, fasting insulin levels, insulin resistance, insulin sensitivity and proteinuria in people who are obese. Regaining weight leads to a relapse in these improvements.(**1++**)
- Reduced energy intake and weight loss also lower levels of triglyceride, IDL-C, VLDL-C, small dense LDL-C, total cholesterol, LDL-C, blood pressure, and increase levels of HDL-C.(**1++**)
- Individualised, behaviourally-based, structured weight loss programmes that include components of a cardioprotective dietary pattern and physical activity lead to moderate weight loss with improvements of metabolic control in individuals who are overweight or moderately obese.(**1+**)
- Reduced energy diets produce further improvements in all risk factors if they are also low in saturated fatty acids, high in dietary fibre and low to moderate in GI.(**1+**)
- There is no evidence that very low carbohydrate weight loss diets improve glycaemic or lipid indices more than equivalently energy-restrictive diets of higher carbohydrate content mostly derived from fruits, vegetables, whole grains, dried peas and beans, and skimmed milk products.
- Very low carbohydrate weight loss diets rich in saturated fatty acids and protein may be contraindicated for the treatment of insulin resistance, microalbuminuria and elevated LDL-C and have not been tested for long-term effectiveness of weight maintenance.(**4**)
- In individuals with morbid obesity, very low energy (formula) diets, with intensive behavioural and dietetic support, achieve large weight losses in the short-term diet phase. Weight tends to be regained.(**1+**)
- Postprandial hyperglycaemia is decreased by changing the type of carbohydrate (GI), reducing the total carbohydrate in the meal, or by dividing carbohydrate throughout the day into 3 meals and 2 to 3 snacks.(**1++**)
- Dietary modifications that reduce GI also lower HbA1c.(**1+**)
- Viscous fibre and dried peas and beans (legumes), when replacing other sources of carbohydrate, lower postprandial blood glucose levels and fasting blood glucose, LDL-C and triglyceride levels.(**1+**)
- Very high-fibre (>40 g/day), high carbohydrate, dietary patterns improve glycaemic control and reduce total LDL-C and triglyceride levels.(**1+**) They also improve insulin sensitivity.(**1+**)
- Artificial sweeteners can facilitate a reduction in dietary sucrose (and energy) without deleterious effects on other aspects of diabetes control. Some products may have laxative effects.(**1+**)
- Intakes up to 45 g per day (3 tablespoons) of added sugars (see glossary) in energy-controlled diets do not lead to the deterioration of glycaemic, insulin or lipid indices when substituted for equivalent carbohydrate as starch from high GI or low fibre sources.(**1+**)
- A range of dietary intakes of total fat (25 – 35% of energy) and carbohydrate (45 – 60% of energy) improve glycaemic and lipid profiles when the sources of fat comprise at least 66% unsaturated fatty acids and the sources of carbohydrate comprise mainly high-fibre food sources (>40 g of fibre per day ≤7 g CHO/g fibre) or sources that are low to moderate GI (GI <70).(**1+**)

- Very high carbohydrate intakes (>60% of energy) also low in fibre or high GI increase postprandial glycaemia, triglyceride and insulin levels, and may reduce HDL-C levels.(**1++**)
- High-fat intakes (>35% of energy) reduce insulin sensitivity and may increase insulin resistance.(**1+**)
- All dietary interventions shown to reduce blood pressure and improve lipid profiles in people without diabetes also improve these risk factors in people with diabetes.(**1++**)
- Omega-3 PUFA up to 3 g per day as fish or fish oil supplements reduce triglyceride levels without adversely affecting glycaemic control.(**1++**)
- The safe alcohol intakes for adults without diabetes (see Table 6) do not in general compromise the treatment of type 2 diabetes. Alcohol intake may affect body weight, increase triglyceride levels and blood pressure.(**2++**)
- There is insufficient evidence to draw conclusions that supplements of magnesium, vitamin E, alpha-lipoic acid, chromium or a number of herbal or plant remedies improve glycaemic control or diabetes complications in adults with type 2 diabetes.(**1++**)

Intensity of Intervention

Intensive and individualised dietary counselling with the specific aim to reduce intakes of saturated fatty acids (<7% of energy), total fat (<35% of energy) and added sugars, and to increase intakes of carbohydrate (42 – 50% of energy), fruit and vegetables, prescribed in conjunction with specific target-driven pharmacotherapy, reduces HbA1c, cholesterol, triglyceride and blood pressure levels, and reduce microvascular complications of diabetes and cardiovascular events, compared with usual care and general dietary advice.[137,152,518,519](**1++,2++**)

In medium-term studies, more intensive dietary interventions improve parameters of risk, or dietary intakes, in individuals with type 2 diabetes, compared with general dietary and exercise advice.[477,520-532](**1+**)

These trials included one or more of:

- more prescriptive dietary regimens
- monitoring of body weight and food intake
- more specific energy restriction
- provision of meals or intermittent use of a defined formula diet
- greater frequency of contact
- inclusion of exercise
- behavioural counselling.

At follow-up (1 – 5 years after discharge to usual care), the improved parameters of risk had regressed towards baseline values.[477,520,528-530,532]

Dietitians provided the dietary intervention,[137,152,477,520-532] or trained the nutritional instructors [521,523,524,527] in intensive intervention studies. Intensive dietary advice provided by dietitians in the outpatient setting is independently effective for the management of weight loss and/or diabetes control in adults with type 2 diabetes.

Cardioprotective Dietary Patterns

The cardioprotective dietary pattern, in its strictest form will also reduce cardiovascular risk in individuals with disorders of glucose regulation. Dietary interventions for the treatment of LDL-C, blood pressure, triglyceride and HDL-C levels discussed in the previous sections apply in the treatment of type 2 diabetes and the metabolic syndrome.

Dietary patterns characterised by an increase in fruit, vegetable, grain and legume or fish intakes, and a low saturated fat intake, together with other lifestyle factors, improve HbA1c and other risk factors associated with type 2 diabetes and the metabolic syndrome. These patterns were modified from typical Western patterns, or based on traditional Mediterranean, Mexican or vegetarian-style patterns.[154,155,470,479,522,524-527,578-580,584,586,589](1+)[582](2++)

Energy Balance

Weight management is the first-line dietary goal in the management of type 2 diabetes and the metabolic syndrome. A recommendation to prevent weight gain, to attempt weight loss and/or reduce waist circumference in individuals who are overweight or obese is integral to slowing the progression of cardiovascular disease in these people.

Weight-reducing regimens can normalise glucose tolerance,[534,535] or reduce or eliminate the need for hypoglycaemic therapy.[529,536-538](1+) Weight loss by any means improves insulin sensitivity in adults with type 2 diabetes or the metabolic syndrome.[270,472,475,529,539-544](1+)

Dietary intervention is pivotal to the achievement of weight loss, while exercise and behavioural programmes or drug therapies are adjunctive therapies and alone do not achieve outcomes equivalent to those that include a dietary intervention component.[233,474,477] Diet-alone weight loss strategies leading to mean weight losses of 9 kg with various dietary programmes are associated with a mean reduction of 2.7% in absolute HbA1c in subjects with type 2 diabetes.[233](1++)

The UKPDS study highlighted that early intensive dietary intervention and weight loss after diagnosis could delay the progression of type 2 diabetes. In 16% of newly diagnosed individuals, treatment goals could be achieved with dietary intervention alone.[545](2+)

Dietary intervention is very effective in promoting an initial weight loss and simultaneously improves the entire cluster of risk factors associated with type 2 diabetes and the metabolic syndrome, in those who are overweight or obese.[233,272](1++) These improvements have a dose-dependent relationship with the percentage of weight lost, begin after only modest weight losses of 5 to 10% of initial body weight,[208,545,546] and improve further with greater weight loss.[545,547](1+)

Energy restriction, independent of weight loss, improves glycaemic control within days of initiation and decreases fasting plasma glucose, free fatty acids and triglyceride levels, hepatic glucose production and increases insulin sensitivity and insulin secretion.[472,529,548,549](1+) Energy restriction also benefits those with BMI less than 25, especially if waist circumference is high.[550]

Weight loss due to energy restriction decreases fasting blood glucose, 2-hour postprandial blood glucose, HbA1c, systolic and diastolic blood pressure, serum triglyceride, total cholesterol, and usually LDL-C concentrations, with increases in HDL-C concentration and insulin sensitivity.[233,272,545,546,548,551,552](1+)

A variety of moderately energy-restricted dietary programmes with a variety of dietary compositions achieve mean 4 to 10 kg of weight loss over 6 to 12 months in participants with type 2 diabetes or the metabolic syndrome.[233,272,472,477,521,523,524,528,529,538,553-558](1++) Very low energy diets may have a role in the management of morbid obesity associated with type 2 diabetes.[278,529,472](1+) These restrictive regimens do not accomodate a continuum of change, and the weight maintenance phase requires continuous dietary education and intensive monitoring.

Meta-analysis has shown that weight loss is associated with restriction of energy intake and not with reduced carbohydrate content in individuals with or without diabetes.[248](**1++**) Popular ad libitum very low carbohydrate (high-fat and high protein) diets achieve no greater weight loss than reported in well-controlled trials of reduced energy intakes in people with diabetes (about 4 – 10 kg).[267-270](**1+**)

The comparison high carbohydrate diets in these studies were low in dietary fibre or high GI. How such high protein, high-fat, low carbohydrate diets influence the depletion of beta-cell function, hyperuricalcidaemia, microalbuminuria, bone loss or renal stones in people with type 2 diabetes is unknown.[250] These potential risks, together with the LDL-C elevating effects of high saturated fat intakes, suggest that such diets are contraindicated in the management of diabetes and the metabolic syndrome.[250]

In energy-controlled trials of low saturated fatty acids weight-reducing diets, an increase of protein to 27% of energy or a lowering of dietary GI, does not achieve greater short-term weight losses than a comparison diet in type 2 diabetes or the metabolic syndrome, but components of these programmes may enhance adherence to energy restriction.[254,255,559-562](**1+**)

Dietary Carbohydrate

Dietary carbohydrate is the primary dietary determinant of postprandial glucose and insulin responses. The amount, type (mostly glucose or fructose after digestion) and rate of digestion of dietary carbohydrate all influence postprandial indices and the overall glycaemic and lipid parameters associated with glucose intolerance.

Glucose-yielding carbohydrate (mostly sugar or starch) is found in a wide variety of foods of variable digestibility and nutrient value, in large part determined by their content of dietary fibre (non-starch polysaccharides and resistant starch), micronutrients, bioactive substances, and preparation and processing methods. Dietary fibre and processing methods are markers for the protective qualities of carbohydrate-yielding foods in glucose-intolerant states.

Glycaemic Index

Postprandial hyperglycaemia is decreased by reducing the total carbohydrate or the GI of the meal.[563](**1++**) The larger the carbohydrate portion of the meal, the larger the glucose and insulin response.[564](**1+**) Food sources of slowly absorbed carbohydrate produce maximum postprandial glucose concentrations that are more closely matched with the delayed first-phase insulin response to meals associated with type 2 diabetes, than rapidly absorbed sources of carbohydrate.[478]

The impact of reducing postprandial blood glucose elevations on overall glycaemic and lipid indices is greatest when a given amount of dietary carbohydrate is derived mostly from foods with the original structure intact (less processed, not finely milled or reconstituted after milling), rich in dietary fibre and/or processed so that the available sugars or starches are slowly absorbed (low to moderate GI).[116,565](**1+**) The GI predicts the postprandial glycaemic peak and 2-hour blood glucose response more effectively than does the refined sugar content.[116] Components of dietary fibre collectively explain up to 50% of the glycaemic response.[566] Among the regular food sources of carbohydrate, dried peas and beans (legumes), including soy beans, produce the lowest postprandial blood glucose response for a given quantity of carbohydrate.[116](**1++**)

Incorporating lower GI foods into the dietary pattern to lower the overall dietary glycaemic index consistently lowers glycosylated protein levels (HbA1c or fructosamine) by 2 to 16% compared with baseline diabetic diets, even over short periods of time, and frequently lowers LDL-C and triglyceride levels in type 2 diabetes.[497,553,561,563,567-576](**1+**) These effects are enhanced when the low GI diet is also higher in dietary fibre.[571] Compared with high GI diets, low GI diets reduce HbA1c by 6.1%.

Provision of a list of the glycaemic index of certain foods offers little towards the practical implementation of a dietary pattern that promotes overall health risk reduction, if more comprehensive education is not also provided. Commercially available books of lists of food glycaemic index and glycaemic load only list glycaemic index, an arbitrary serving size, the total carbohydrate of this serving and its glycaemic load (GL)[577] without listing saturated fatty acids, fibre, added sugar, salt or kilojoule levels of foods. Such lists overlook the impact of random selection of low GI foods for their energy density or overall nutritional value, and impact on lipids, blood pressure and other risk factors. High-fibre, carbohydrate-rich foods need to be emphasised as higher priority selections.

Dietary Fibre

Dietary interventions that provide a high percentage of carbohydrate as intact foods with their naturally occurring complement of dietary fibre (>40 g of dietary fibre per day or <7 g of carbohydrate/g of dietary fibre), essential micronutrients and bioactive compounds, are recommended for the improvement of glycaemic indices, lipid parameters and weight control. Compared with high carbohydrate low fibre diets, they improve glycaemic indices, total and LDL-C, and triglyceride levels.[571,575,578-585](**1+**) They also improve insulin sensitivity.[497,586](**1+**) Compared with diets that are low in carbohydrate, low in fibre and high in unsaturated fatty acids, they improve glycaemic parameters, total cholesterol and LDL-C levels while triglycerides remain unchanged.[575,578,580,587,588](**1+**) The results are most significant when the dietary interventions include a large proportion of low glycaemic index foods, such as dried peas and beans (legumes) or viscous non-starch polysaccharide (soluble fibre).[580,584,585,589]

When high carbohydrate diets (>55% of energy) of average fibre content (24 – 35 g/day) are compared with lower carbohydrate diets rich in monounsaturated fatty acids but with equivalent fibre, both diets lower LDL-C but the diets rich in monunsaturated fatty acids may lower triglyceride and VLDL-C levels more effectively in the short-term but without any difference in HbA1c.[590-597](**1++**) These differences are not seen when the high carbohydrate diet is also lower GI.[335,563,570,575,598]

Viscous non-starch polysaccharides (soluble fibre), studied as a supplement or as a component of food (pectin, Beta-glucan from oats, psyllium, guar gum), lower postprandial blood glucose excursions in people with type 2 diabetes.[116,599](**1++**) When included in dietary patterns these fibre supplements aid overall glycaemic control and produce LDL-C lowering, with variable effects on triglyceride levels,[466,600-611] similar to that achieved in adults with normal glucose tolerance (2 – 10 g/day lowers LDL-C by 5 – 10%).[347](**1+**)

Meal Frequency

Both size and type of the carbohydrate load at a given meal determines the postprandial glucose and insulin responses in single meal studies.[564] Various food factors are shown to slow the rate of meal glucose absorption and to blunt the insulin response. Single-day profiles of small frequent meals reduce all-day insulin secretion and mean blood glucose levels compared with 2 to 3 meals per day.[612,613] A longer-term study found no advantage of consuming nine small 1 to 2-hourly meals/snacks per day or 3 meals and a small snack per day.[612]

Consistency in the amount and source of carbohydrate intake from day-to-day improves glycaemia in individuals with type 1 diabetes treated with twice-daily insulin.[614] Behavioural aspects of frequent meal consumption have more relevance for overall glycaemic control. No consistent relationship has been reported between meal frequency and weight control under isoenergetic conditions in people without diabetes,[615] or with weight change in the general population,[616] or in 24-hour energy expenditure measured by whole-body calorimetry and doubly-labelled water.[615]

Sweeteners

Sugar alcohols and sugar-replacement compounds such as xylitol and sorbitol have a negligible GI in the quantities normally consumed[116] and contribute fibre-free carbohydrate energy to the diet (50 – 75% the energy yield of sucrose). Excessive use of some products may produce an osmotic laxative effect. Saccharin, aspartame, cyclamate and sucralose are non-nutritive sweeteners 180 to 600 times sweeter than sucrose with negligible energy value and no effect on postprandial blood glucose concentrations.[617] Various products and extracts of the 'dietary supplement' Stevia are 30 to 300 times sweeter than sugar. Granular forms of these highly-intense sweetening agents are produced by adding dextrose and/or maltodextrin to non-nutritive sweeteners in a ratio that yields negligble energy per standard dose.

Short-chain fructo-oligosaccharide, a new sweetening agent, is incompletely absorbed, yields less energy than other sugars but has not been shown to improve glycaemic or lipid parameters compared with other sugars.[618,619]

Added Sugars

Sucrose has an intermediate GI and raises glucose and insulin levels no more than equivalent carbohydrate as cooked starch of high GI in individuals with type 2 diabetes.[116,620-623](**1++**) Sucrose contributes concentrated, fibre-free energy to the diet and lacks nutritive value other than to improve the palatability of whole grain and fruit products. Intakes up to 45 g/day (3 tablespoons) in energy-controlled diets do not compromise glycaemic, insulin or lipid indices when substituted for equivalent carbohydrate as starch from high GI sources.[576,621,624-631](**1+**)

Ad libitum diets designed with a high proportion of simple sugars inhibit weight loss compared with higher-starch diets in people who have the metabolic syndrome[632] or are healthy or post-obese or have two coronary heart disease risk factors.[576,633]

The majority of people with type 2 diabetes and the metabolic syndrome require a reduced energy intake and a variety of protective foods that have a rich content of dietary fibre. A general recommendation to restrict sucrose or added sugar intake to 5% of energy within solid meals as flavouring for recommended foods is appropriate. This is equivalent to 15 to 30 g per day (1 – 2 tablespoons). Active individuals with a BMI less than 30 and type 2 diabetes or the metabolic syndrome can maintain target glycaemic and lipid parameters when consuming added sugars up to 10% of energy in energy-controlled diets, providing other protective components of diet are not compromised.

Dietary Fat and Cholesterol

Dietary restriction of saturated fatty acids and cholesterol reduces total cholesterol and LDL-C, and fish oil reduces triglyceride levels in adults with type 2 diabetes to a similar degree as in glucose-tolerant individuals.[585,634-639](**1++**) Partial replacement of saturated fatty acids with high-fibre carbohydrate-rich foods improves glycaemic and lipid indices in type 2 diabetes.[589,640] The effects of low fibre, high glycaemic index sources of carbohydrate on the TC:HDL ratio, important in the metabolic syndrome, are unclear.[311](**1++**)

An increase in the total fat intake (to 40% of energy) by increasing monounsaturated fatty acids, in order to reduce the dietary content of refined carbohydrate-containing foods, may improve glycaemic indices and triglyceride levels in type 2 diabetes in the short term.[641] They do not reduce insulin sensitivity unless diabetes is poorly controlled.[592,593,642] However, too much of the diet is consumed by low-nutrient energy and a recommendation to increase consumption of oils in uncontrolled free-living conditions may lead to an increase in dietary energy density (see carbohydrate and weight management sections).

A low polyunsaturated to saturated fatty acid (P:S) ratio is associated with coronary heart disease events in people with type 2 diabetes[144] and a higher dietary P:S ratio improves insulin sensitivity.[636] Saturated fatty acids reduce insulin sensitivity and restriction of saturated fatty acids reduces triglyceride levels. [643]

Plant Sterols and Stanols

Plant sterol and stanol-fortified esters (3 g/day) further lower LDL-C, IDL-C, and VLDL-C in adults with type 2 diabetes, over and above the effects of diet and of statin therapy.[644,645](**1+**)

Fish Oil

Fish oil supplementation in type 2 diabetes (3 – 18 g/day) lowers triglycerides (-0.56 mmol/L), raises LDL cholesterol (0.24 mmol/L), and has no statistically significant effect on HbA1c or fasting blood glucose. In trials that included hypertriglyceridemic subjects the triglyceride-lowering effect and the elevation in LDL cholesterol were most marked. Lower-dose levels (3 g/day) did not raise LDL cholesterol.[638,639](**1++**) After a dose of 4 g of fish oil per day, LDL particles from people with type 2 diabetes become less resistant to oxidation.[646] However, the effects of fish oil supplementation on cardiovascular endpoints in type 2 diabetes are as yet unreported.

Alcohol

Moderate alcohol intake consumed with a meal has little effect on postprandial blood glucose levels in people with type 2 diabetes.[647] Acute alcohol intake reduces peripheral insulin sensitivity.[648] Paradoxically, chronic light to moderate alcohol consumption is associated with enhanced insulin sensitivity, reduced fasting or postprandial insulin levels and decreased measures of insulin resistance in people with normal glucose tolerance,[649] except in one trial in women who were overweight.[650] In the IRAS study all levels of alcohol consumption, when adjusted for insulin sensitivity, were associated with reduced risk of carotid artery atherosclerosis in people with normal glucose tolerance, but the risk was J-shaped (lowest risk <0.5 g/day) in those with IGT. In those with diabetes, all levels of alcohol intake were associated with increased risk, compared with never drinking alcohol.[651]

A low-risk level of alcohol intake has not been determined for the metabolic syndrome. After assessment of intake, the risk-factor profile will determine the most appropriate recommended intake. Alcoholic beverages provide energy and potential effects on sugar intake, triglyceride levels and blood pressure.[647]

Dietary Supplements

A systematic review found there was insufficient evidence to draw definitive conclusions about the effectiveness of any herbs or dietary supplements to improve glycaemic control in diabetes.[652](**1+**) A meta-analysis of dietary chromium supplements found no effect of chromium on glucose or insulin concentrations in people without diabetes, but the evidence for people with diabetes was inconclusive.[653](**1++**)

Supplements of folic acid reduce homocysteine levels in people with type 2 diabetes, but the effects on disease endpoints remain undetermined.[654,655]

DRUG INTERVENTIONS FOR PEOPLE WITH DIABETES

People with diabetes who have had a cardiovascular event should start appropriate secondary-prevention treatment, aspirin, a beta-blocker and ACE-inhibitor after myocardial infarction, and ACE-inhibitor/thiazide combination after stroke, irrespective of baseline blood pressure or lipid levels.

People with diabetes and microalbuminuria, overt diabetic nephropathy or other renal disease should be treated with an ACE-inhibitor or an A2 receptor-blocker, aspirin and lipid-modifying treatment irrespective of baseline levels.

People with the metabolic syndrome who have high triglycerides and low HDL-C should be considered for treatments known to improve this lipid profile (ie, improved diet, increased physical activity, fish oils and drug treatment including fibrates).

People aged 75 years and over with diabetes should be treated in the same way as younger people.

Benefits of Blood Pressure-Lowering Medication

Raised blood pressure is 1.5 to 2 times more prevalent in people with diabetes. Randomized controlled trial evidence shows that lowering blood pressure in people with diabetes lowers cardiovascular risk.[150,409,656](**1++**) Each 10 mm Hg reduction in systolic blood pressure is associated with a 15% (95% CI; 12 – 18%) reduction in risk of cardiovascular death over 10 years.[149] Aggressive blood pressure control is indicated in everyone with diabetes especially those with diabetic renal disease, to achieve the reduction of both cardiovascular disease outcomes and renal complications.

The HOPE study[140](**1++**) has shown that treatment with an ACE-inhibitor decreases the risk of cardiovascular disease complications in people with type 2 diabetes. These benefits are similar in people with normal and raised blood pressure, and cannot be explained by blood pressure reduction alone. ACE-inhibitors are more effective than other agents in reducing urinary albumin loss. The benefit of ACE-inhibitor therapy on glomerular filtration rate is independent of blood pressure change.[411,657](**1++**)

Beta-blockers are widely used in people with type 2 diabetes and have been shown to have a cardioprotective benefit in people with diabetes and cardiovascular disease.[658,659](**2++**) A small number of people with type 1 diabetes and hypoglycaemic episodes or autonomic neuropathy cannot tolerate beta-blockers.(**1+**)

Choice of Therapy to Lower Blood Pressure

In people with diabetes and with uncomplicated raised blood pressure, ACE-inhibitors, thiazide diuretics, beta-blockers and calcium channel blockers are all effective in lowering blood pressure and reducing cardiovascular risk. The majority of people with diabetes will require more than one antihypertensive agent in order to achieve acceptable cardiovascular risk reduction.

ACE-inhibitors should be considered as first-line therapy in people with diabetes and microalbuminuria, overt diabetic nephropathy (albumin:creatinine ratio ≥30 mg/mmol) or other renal disease, irrespective of the blood pressure level. A2 receptor-blockers exert a nephroprotective effect and are indicated if ACE-inhibitors are not tolerated or contraindicated. A2 receptor-blockers can be considered for combination therapy with an ACE-inhibitor to achieve blood pressure targets. People with diabetes who have had a cardiovascular event should be considered for both a beta-blocker and an ACE-inhibitor. ACE-inhibitors should be considered early in people with diabetes (and especially for Māori and Pacific people who have a high incidence of end-stage renal failure) as they reduce cardiovascular risk in those at high risk and are also nephroprotective for those with microalbuminuria or overt diabetic nephropathy.

Benefits of Lipid-modifying Medication

Lipid abnormalities are common in people with type 2 diabetes. The most common type of abnormality in type 2 diabetes is a combination of elevated triglycerides, reduced HDL and small dense LDL-C. This small dense LDL-C composition is more atherogenic. Randomized controlled trial evidence shows that intensive lipid management in people with diabetes lowers cardiovascular risk.[69,355,660] **(1++)**

Choice of Lipid-modifying Therapy

The lipid profile in people with diabetes will determine the drug treatment selected to lower lipid levels.

Predominant Hypercholesterolaemia

Statins should be used to lower LDL-C when the triglyceride level is normal or only slightly elevated. If optimum levels are not achieved in people with diabetes with a calculated 5-year risk greater than 15%, low dose nicotinic acid (<1.5 g/day) or acipimox may be added. Such people may need specialist review.

Predominant Hypertriglyceridaemia and Low HDL-C

Fibrates should be used in people with predominant hypertriglyceridaemia and low HDL-C with normal to slightly elevated LDL-C levels (<3 mmol/L). If treatment with a fibrate is not tolerated or if additional triglyceride-lowering effect is required, fish oil can be used with close monitoring of glycaemic control. Acipimox is often useful in those not responding to fibrate agents. Nicotinic acid can be used, but will often require specialist review.

Combined Dyslipidaemia

Treatment with a statin and a fibrate should be considered in people with moderate to marked elevation of both LDL-C and triglycerides. Due to the increased risk of myositis with combinations (particularly those that include gemfibrozil), special care should be taken to fully inform and monitor people on combination therapy.

Low HDL-C on Statin Therapy

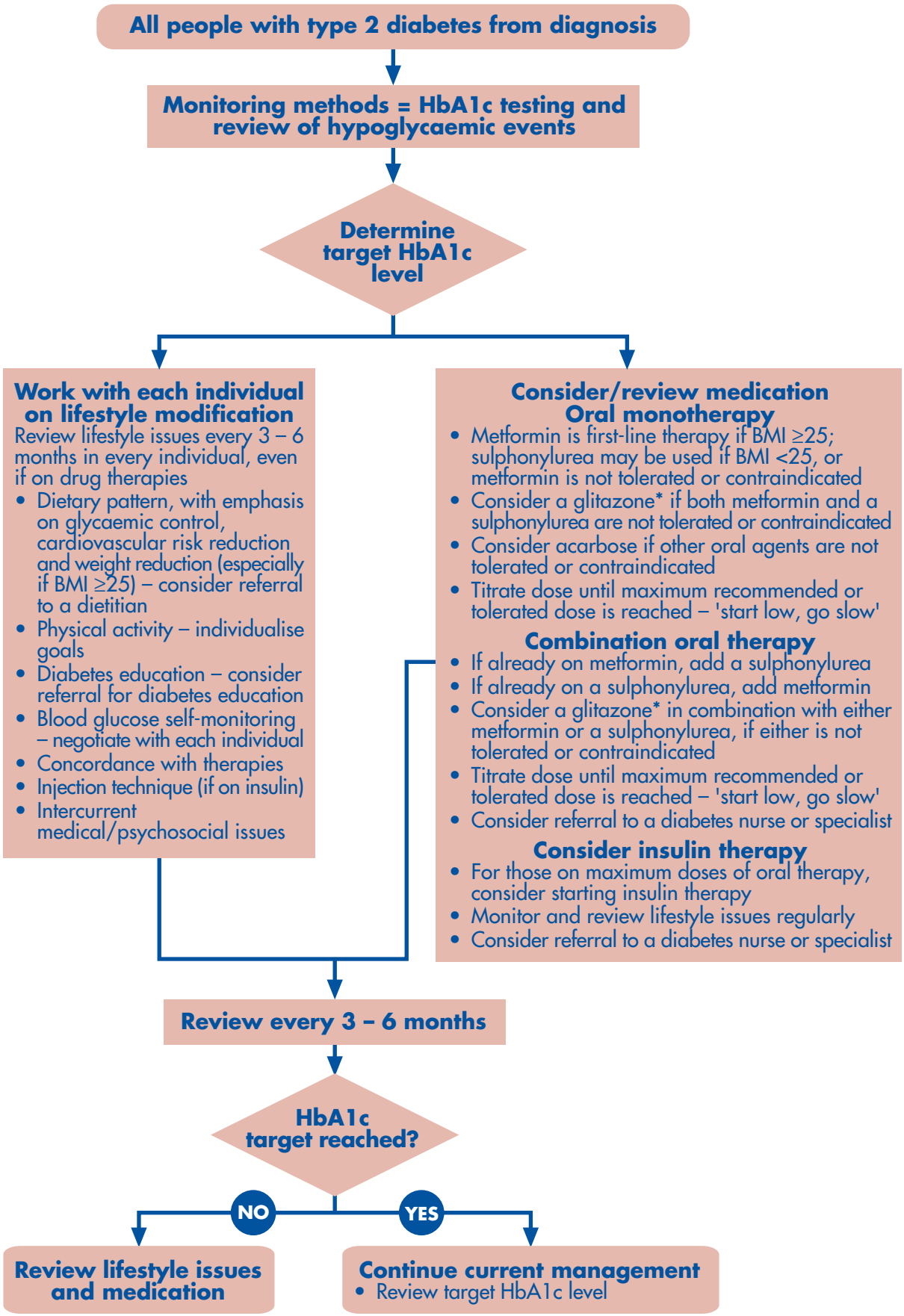
When HDL-C is below 0.9 mmol/L and the person with diabetes is on statin therapy, low-dose nicotinic acid therapy can be used to augment the LDL-C and triglyceride-lowering actions and raise HDL-C cholesterol. Such people may need specialist review and those with very low HDL-C levels (<0.7 mmol/L) should be referred.

Benefits of Glycaemic Control

Improved glycaemic control is associated with reduced cardiovascular morbidity and mortality. Prospective studies have shown that a 1% reduction in HbA1c is associated with a 14% reduction in myocardial infarction, 16% reduction in heart failure and 12% reduction in ischaemic stroke.[77] **(2++)**

In people with diabetes, the case fatality following myocardial infarction is double that of the non-diabetic population. People with diabetes more often present with a painless or silent myocardial infarction, which leads to a delayed admission to hospital. There is one randomized controlled trial that supports intensive insulin treatment during the acute management of myocardial infarction which shows a reduction in mortality at 1 year.[661] **(1++)**

Figure 5: Stepwise approach to glycaemic control in type 2 diabetes



For further details see the New Zealand Guideline, *Management of Type 2 Diabetes* (www.nzgg.org.nz).

*At the time of publishing, only rosiglitazone is available in New Zealand and it is not subsidised.

Choice of Therapy to Improve Glycaemic Control

A stepwise approach to increasing the dose of medication or changing the type of medication is recommended (see Figure 5). Metformin is an appropriate choice for those who are overweight (BMI ≥25).

THE OVERALL GOAL OF INTERVENTIONS IN PEOPLE WITH DIABETES

The overall goal of treatment is to reduce cardiovascular risk. The intensity of treatment to achieve this goal should be related to pre-treatment cardiovascular risk. The higher the level of risk, the greater the effort made to achieve the optimal risk factor levels. An individual's risk factor levels should always be interpreted within the context of their calculated cardiovascular risk.

All the major risk factors including smoking, blood pressure, lipids and glycaemic control require special attention in people with diabetes.

Specific Targets of Treatment

Good glycaemic control is a key goal of management and delays the onset and progression of microvascular complications. An optimal HbA1c level is as close to a physiological level as possible (aim for <7%). Postprandial and fasting glucose can also be used to monitor glycaemic control.

People at very high risk (5-year cardiovascular risk >20%) determined clinically with:

- **a previous history of cardiovascular disease**
- **the specific lipid disorders of a genetic basis (FH, FDB or FCH)**
- **diabetes and overt diabetic nephropathy or diabetes with other renal disease.**

Everyone classified clinically at very high risk should begin drug treatment concurrently with intensive lifestyle advice and all efforts should be made to reach optimal lipid levels.

An optimal HbA1c level is as close to a physiological level as possible, so aim to reduce HbA1c to less than 7%. In people with diabetes who have had a cardiovascular event, the goal of blood pressure treatment is 130/80mm Hg. More aggressive blood pressure treatment is recommended for people with diabetes and overt diabetic nephropathy (albumin:creatinine ratio ≥30 mg/mmol) or diabetes with other renal disease and blood pressure less than 120/75 mm Hg. Lipid management should aim to lower LDL-C to less than 2.5 mmol/L.

People with diabetes with a calculated 5-year risk greater than 15%

Everyone with diabetes should be offered risk factor treatment to lower their 5-year cardiovascular risk to less than 15%. Where possible, treatment should aim to achieve the optimal levels in Table 24. These levels have been set lower in people with diabetes to protect renal function. All people with diabetes and a 5-year cardiovascular risk greater then 15% should be offered low-dose aspirin.

People with diabetes and total cholesterol greater than 8 mmol/L or TC:HDL ratio greater than 8 or blood pressure greater than 170/100 mm Hg

People with diabetes and isolated elevated single risk factor levels are assumed to have a greater than 15% CV risk over 5 years, but should have a risk assessment because, when all risk factors are

taken into account, they may have a calculated 5-year CV risk higher than this. These people should receive intensive lifestyle intervention and lipid-modifying or blood pressure-lowering treatment as appropriate.

Table 24: Optimal risk factor levels in people with diabetes

Lipid Fraction	Value
Total Cholesterol	<4 mmol/L
LDL Cholesterol	<2.5 mmol/L
HDL Cholesterol	>1 mmol/L
TC:HDL ratio	<4.5
Triglycerides	<1.7 mmol/L

Blood Pressure	Systolic Blood Pressure	Diastolic Blood Pressure
People with diabetes or cardiovascular disease	<130 mm Hg	<80 mm Hg
People with diabetes and overt nephropathy, microalbuminuria or diabetes with other renal disease	Aggressive blood pressure control is recommended	
HbA1c	HbA1c as close to physiological levels as possible (aim for <7%)	

MONITORING AND DURATION OF TREATMENT

RECOMMENDATIONS: TREATMENT MONITORING AND DURATION	
Lifelong treatment is advised for people with diabetes.	✓
People with diabetes receiving medication should have their lipids and blood pressure, glycaemic control, diet and activity level monitored every 3 months until adequate control is achieved, then every 6 months.	✓
Referral to a specialist for an opinion or specialist management should be considered for people with type 2 diabetes if: • serum creatinine is greater than or equal to 0.15 mmol/L • calculated GFR is less than 60 ml/min/1.73m^2 • there is a rapid increase in level of microalbuminuria or proteinuria • there is a difficulty in achieving blood pressure targets • in situations where non-diabetic renal disease may be present, or may co-exist with diabetic renal disease: - there is absence of diabetic retinopathy in a person with renal disease - there are urinary abnormalities such as haematuria or casts (once infection has been excluded as the cause).	✓

KEY - *Grades indicate the strength of the supporting evidence not the importance of the recommendations - see page xvii for details*

A Recommendation is supported by good evidence
B Recommendation is supported by fair evidence
C Recommendation is supported by non-analytic studies or consistent expert opinion
I No recommendation can be made because the evidence is insufficient
✓ Good Practice Point

MEDICATION FOR CARDIOVASCULAR DISEASE

This section considers the medication to be used long-term for cardiovascular disease (angina, myocardial infarction, transient ischaemic attack or ischaemic stroke). The acute management of myocardial infarction and stroke in hospital has been excluded as this is covered in the New Zealand acute coronary syndromes guideline and the *Life After Stroke: New Zealand Guideline for Management of Stroke*. Rehabilitation after myocardial infarction is addressed in the New Zealand Cardiac Rehabilitation Guideline, see www.nzgg.org.nz

ASPIRIN

RECOMMENDATIONS: ASPIRIN USE AFTER MYOCARDIAL INFARCTION AND STROKE	
AFTER MYOCARDIAL INFARCTION	
Aspirin 75 to 150 mg/day should be given routinely and continued for life. These doses are at least as effective as higher doses.	A
AFTER STROKE	
Aspirin 75 to 150 mg/day should be given routinely after ischaemic stroke or transient ischaemic attack, unless there is an indication for anticoagulation with warfarin. These doses are at least as effective as higher doses.	A
CT scan should be obtained prior to aspirin therapy to exclude intracranial haemorrhage.	C

EVIDENCE STATEMENTS

- Aspirin is the cheapest and most effective single antiplatelet agent for reducing serious vascular events in people following stroke or myocardial infarction.(**1++**)
- The benefits of low-dose aspirin outweigh the harms in people with a history of cardiovascular disease. In these people, a meta-analysis has shown that low-dose aspirin reduces the risk of a cardiovascular event by 25%.[424](**1++**)

The Antithrombotic Trialist Collaboration systematic review and meta-analysis, has found that prolonged antiplatelet treatment reduces the risk of serious vascular

events in people at high risk of a cardiovascular event, myocardial infarction or stroke.[424] It showed that after myocardial infarction prolonged use of aspirin prevents 36/1000 serious vascular events (non-fatal myocardial infarction, non-fatal stroke or vascular death) over 2 years. It also showed that aspirin 75 to 150 mg per day is as effective as higher doses and reduced the risk of bleeding, but found insufficient evidence that doses below 75 mg per day are effective. No alternative antiplatelet regimen was shown to be superior to aspirin for long-term secondary prevention of cardiovascular events but clopidogrel was at least as effective and as safe as aspirin.

The most serious side effect of antiplatelet treatment is the risk of haemorrhage, particularly intracranial haemorrhage. Antiplatelet treatment is also associated with an increased risk of haemorrhage from other sites, mainly from the gastrointestinal tract. The absolute excess risk of an extracranial haemorrhage is about 1 to 2/1000 people treated per year post myocardial infarction. Most of the extracranial haemorrhages are non-fatal.

Two large trials of aspirin[662,663] started in the first 48-hours of stroke onset have shown that aspirin doses of 160 to 300 mg per day are effective in reducing the risk of further events. An analysis of the combined data from the 40,000 people in these trials shows that there was a highly significant reduction of 7/1000 in recurrent ischemic stroke (320 (1.6%) aspirin versus 457 (2.3%) control, p<0.000001, 2-sided test) and a less clearly significant reduction of 4/1000 in death without further stroke (5.0% versus 5.4%, p=0.05, 2-sided test). Against these benefits, there was an increase of 2/1000 in haemorrhagic stroke or haemorrhagic transformation of the original infarction (1.0% versus 0.8%, p=0.07, 2-sided test) and no apparent effect on further stroke of unknown cause (0.9% versus 0.9%). Post-stroke the risk of intracranial haemorrhage from aspirin given at the time of the acute event, before CT scan is low. Among the 9,000 people (22%) randomized without a prior CT scan, aspirin appeared to be of net benefit with no unusual excess of hemorrhagic stroke; moreover, even among the 800 (2%) who had inadvertently been randomized after a haemorrhagic stroke, there was no evidence of net hazard (further stroke or death, 63 aspirin versus 67 control).

It is not possible to reliably distinguish ischaemic and haemorrhagic stroke on clinical grounds. Good practice should therefore include a CT scan before aspirin wherever possible. However aspirin may be given after acute stroke prior to a CT scan when urgent CT scan is not readily available.[664] Aspirin can be stopped if a later CT scan, done within 48-hours, shows a haemorrhagic stroke. The appropriate use of aspirin and imaging after transient ischaemic attack is covered in *Life After Stroke: The New Zealand Guideline for Management of Stroke*, see www.nzgg.org.nz

CLOPIDOGREL

RECOMMENDATIONS: CLOPIDOGREL USE AFTER MYOCARDIAL INFARCTION AND STROKE	
AFTER MYOCARDIAL INFARCTION	
Clopidogrel (75 mg/day) is an effective alternative to aspirin for people with contraindications to aspirin or those who are intolerant of aspirin.	**A**
AFTER STROKE	
Clopidogrel (75 mg/day) can be used as a safe and effective alternative to aspirin after stroke.	**A**

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EVIDENCE STATEMENT

- Clopidogrel is at least as clinically effective and as safe as aspirin but may not be as cost-effective.(**1++**)

Note: Clopidogrel is substantially more expensive than aspirin and not funded in New Zealand at the time of publication. It is contraindicated for people with hepatic impairment or active pathological bleeding and carries a manufacturers precaution against its use in people with a predisposition to gastrointestinal bleeding.

The rates of serious side effects, such as intracranial or extracranial haemorrhages are similar for clopidogrel and aspirin.[424] The CAPRIE trial (clopidogrel versus aspirin in people at risk of ischaemic events) compared clopidogrel with aspirin in reducing the risk of vascular events in 19,185 people with clinical manifestations of atherosclerosis. Participants were randomized to receive daily oral clopidogrel (75 mg) or aspirin (325 mg). Treatment periods ranged from 1 to 3 years. The primary outcome measurement was an aggregate of myocardial infarction, ischaemic stroke and vascular death. When compared directly with aspirin clopidogrel reduced cardiovascular events by a further 8.7% (RR 0.913, 95% CI, 0.84 – 0.97, ARR 0.86% over about 2 years). Clinically, aspirin would be expected to prevent 19 major clinical events per 1000 treated for a year compared to 24 prevented with clopidogrel in a similar population to this trial. Gastrointestinal hemorrhages occurred in 1.99% of people treated with clopidogrel and 2.66% of those treated with aspirin ($p<0.002$).[425]

Clopidogrel inhibits platelet aggregation and when added to aspirin has been shown to reduce the composite endpoint of death, myocardial infarction and stroke, by 27% if given as a loading dose before and for a year after elective percutaneous coronary interventions (PCI), (RR 0.73, 95% CI, 0.66 – 0.96%, ARR 3% over 1 year). Risk of major bleeding in this trial at 1 year increased but not significantly (8.8% with clopidogrel versus 6.7% with placebo, $p=0.07$).[665] The addition of clopidogrel to aspirin following an acute coronary syndrome without ST-segment elevation has been shown to reduce combined cardiovascular events, non-fatal myocardial infarction, stroke or cardiovascular death, by 20% (RR 0.80, 95% CI, 0.72 – 0.90, ARR 2.1% over 9 months) compared to aspirin alone. However, the risk of a major gastrointestinal or intracranial bleed is increased in people treated with clopidogrel. Clinically for every 1000 treated for 9 months, 28 major events would be prevented, with 3 more major bleeds and 3 more transfusions.[666](**1++**)

The combination of clopidogrel with aspirin for people who have a transient ischaemic attack or ischaemic stroke while treated with aspirin has not been adequately studied.[667]

The systematic review of thienopyridine derivatives (ticlopidine and clopidogrel) versus aspirin found that the thienopyridines produced significantly less gastrointestinal haemorrhage and upper gastrointestinal side effects than aspirin. However, the risk of skin rashes and diarrhoea was increased with the thienopyridine derivatives. Ticlopidine (but not clopidogrel) increased the risk of neutropenia[667] and was associated with thrombocytopenia and thrombocytopenic purpura.[421,668] There is no evidence that these haematological side effects occur with clopidogrel.[669,670] Two randomized controlled trials of about 1700 people undergoing coronary artery stenting showed clopidogrel plus aspirin was safer and better tolerated than ticlopidine plus aspirin.

Clopidogrel is substantially more expensive than aspirin and is not currently funded for secondary prevention of cardiovascular events in New Zealand. The cost of clopidogrel in 2003 was approximately $NZ250 per month on a private prescription from a community pharmacy. Rational prescribing requires careful patient selection.

DIPYRIDAMOLE

RECOMMENDATIONS: DIPYRIDAMOLE USE AFTER STROKE	
AFTER STROKE	
There is insufficient evidence to recommend dipyridamole as a first-line treatment for the secondary prevention of vascular events, either as monotherapy or in combination with aspirin.	**I**
Combination treatment with modified-release dipyridamole and aspirin can be used for prevention of non-fatal stroke for people at high risk of cerebral ischaemic events, including those who have symptomatic cerebral ischaemia while treated with aspirin alone.	**B**
Monotherapy with modified-release dipyridamole is recommended for prevention of non-fatal stroke if aspirin is contraindicated and clopidogrel is unavailable.	**B**

EVIDENCE STATEMENT

- The routine addition of dipyridamole to aspirin reduces the risk of non-fatal stroke(**1-**) but does not further reduce the total number of vascular events compared with aspirin monotherapy.(**1+**).

Note: Special authority is required for funding of dipyridamole in New Zealand. Criteria include ischaemic symptoms while treated with aspirin or intolerance to aspirin.

There have been a number of trials of dipyridamole alone with inconsistent results in terms of combined cardiovascular outcomes. There have been no trials directly comparing dipyridamole with clopidogrel after stroke.

Despite a lack of high quality evidence, clinical practice suggests that combination treatment with modified release dipyridamole and aspirin can be used for people who have symptomatic cerebral ischaemia (transient ischaemic attack or ischaemic stroke) while treated with aspirin alone, and that dipyridamole monotherapy can be used if aspirin is contraindicated and clopidogrel is unavailable.

One systematic review has shown that adding dipyridamole to aspirin non-significantly reduced vascular events (not just stroke) in high-risk participants (OR 0.94, 95% CI, 0.83 – 1.06).[424] Overall, the review found that combination treatment reduced non-fatal strokes (OR 0.86, 95% CI, 0.77 – 0.98). However, most of the data in this review comes from one large trial comparing aspirin (50 mg/day), dipyridamole (400 mg/day) and combination therapy in about 6000 people with prior ischaemic stroke or transient ischaemic attack. In this trial, combination therapy produced a significant reduction in recurrent non-fatal strokes.[671] This finding has not been confirmed in other trials. These conflicting results may be because of the lower aspirin dose in the larger trial, or the antihypertensive effect of dipyridamole.

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WARFARIN

RECOMMENDATIONS: WARFARIN USE AFTER MYOCARDIAL INFARCTION AND STROKE	
AFTER MYOCARDIAL INFARCTION	
Warfarin should be prescribed for high-risk survivors of myocardial infarction including those with: • atrial fibrillation or paroxysmal atrial fibrillation • a large left ventricular aneurysm • thrombus demonstrated in the left ventricle at the infarction site by echocardiography • systemic embolism.	A
Warfarin should be considered for people who cannot be given antiplatelet agents after myocardial infarction.	A
AFTER STROKE	
Warfarin should not be prescribed for people with transient ischaemic attack or minor strokes unless cardiac embolism is suspected.	A
Warfarin should be considered for people after stroke associated with atrial fibrillation unless contraindicated.	A
Warfarin should be considered for people after ischaemic stroke associated with mitral valve disease, prosthetic heart valves, or within 30 days of myocardial infarction.	C
Warfarin should ideally be started in hospital. For minor stroke, it can be started after the first 48-hours or later if haemorrhage has been excluded by brain imaging. Delay for 7 to 14 days may be preferable for people after a major stroke.	C
AFTER MYOCARDIAL INFARCTION OR STROKE	
The target INR should be 2.5 (range 2 – 3), for most people prescribed warfarin after myocardial infarction or after ischaemic stroke associated with atrial fibrillation or mitral valve disease.	A

EVIDENCE STATEMENTS

- In people with atrial fibrillation, warfarin is superior to aspirin in preventing stroke.(**1++**)
- The benefits of warfarin therapy after myocardial infarction outweigh the harm if therapy is reserved for those at high risk of thromboembolism.(**1++**)

One randomized controlled trial of 2206 people over 7 years has shown that aspirin works as well as warfarin in preventing recurrent strokes in most people after stroke. In people with atrial fibrillation the evidence shows that warfarin is better than aspirin. Older people with atrial fibrillation are at very high risk of stroke.[672]

One randomized controlled trial of 1316 people with a previous transient ischaemic attack or non-disabling stroke compared aspirin with oral anticoagulant (target international normalised ratio 3.0 to 4.5) for about 14 months. The trial was stopped early because of an excess number of poor outcomes (almost exclusively related to cerebral haemorrhage) with anticoagulation versus aspirin (12.4% with anticoagulant v 5.4% with control; (ARI 7%, 95% CI, 4 to 10%, NNH 14, 95% CI, 10 – 25).[673]

One systematic review comparing oral anticoagulants versus placebo has found no significant difference in the risk of stroke recurrence after presumed ischaemic stroke in people in normal sinus rhythm. Risk of fatal intracranial and extracranial haemorrhage was increased.[674]

The timing of anticoagulation initiation after stroke is important, but optimal timing is uncertain. If warfarin is initiated too soon it may cause haemorrhagic transformation or bleeding. If it is initiated too late, the risk of a further event is greater. By 48-hours most people with minor stroke will have stabilised and could start warfarin. There are advantages in starting treatment in hospital rather than waiting for an arbitrary 14 days as is common practice. The absolute risk of early recurrent stroke in those with atrial fibrillation is however small.

If warfarin is used post myocardial infarction there is an increased risk of serious bleeding and three studies suggest that the balance of benefits versus harms favours only using warfarin for those at high risk of embolism.[675-677] Many medications, over-the-counter preparations and vitamin supplements (vitamin K and vitamin C) interact with warfarin, or alter the reponse to warfarin. People should be advised to check with their health professional before taking anything new while on warfarin. The amount of vitamin K in the diet will alter the effect of warfarin. Special consideration should be given to foods high in vitamin K such as alfalfa, spinach, broccoli, lettuce, soya beans, cabbage, beef liver and wheat bran.

Primary prevention of stroke in people with atrial fibrillation and without previous stroke will be covered by the New Zealand atrial fibrillation guideline.

BETA-BLOCKERS

RECOMMENDATIONS: BETA-BLOCKER USE AFTER MYOCARDIAL INFARCTION	
AFTER MYOCARDIAL INFARCTION	
Beta-blockers should be considered for everyone following myocardial infarction unless there are contraindications.	A
Beta-blockers are also recommended in those with left ventricular dysfunction and heart failure.	A
The initial dose of beta-blockers should be low and the dose should be titrated upwards slowly.	✓
Everyone should receive an explanation of the benefits and risks of treatment.	✓
Beta-blockers given at night may reduce the risks of postural hypotension and alleviate symptoms of tiredness and lethargy.	✓
Before discontinuing beta-blockers because of side effects a lower dose or alternative beta-blocker should be tried.	✓
If full doses of a beta-blocker and ACE-inhibitor are not tolerated moderate doses of both are preferable to a high dose of a single agent.	✓

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EVIDENCE STATEMENTS

- Beta-blocker treatment reduces all-cause mortality, coronary mortality, recurrent non-fatal myocardial infarction, and sudden death after myocardial infarction.(**1++**)
- Beta-blocker treatment (carvedilol) improves all-cause mortality in people with left ventricular dysfunction or heart failure after myocardial infarction.(**1++**)

There is strong evidence from systematic reviews that beta-blockers reduce the risk of all-cause mortality, coronary mortality, recurrent non-fatal myocardial infarction, and sudden death after myocardial infarction.[415,678,679] The reduction in mortality is seen for both men and women. Those at high risk of mortality after a myocardial infarction benefit most. These include people aged 50 years and over; have had previous myocardial infarction, angina pectoris, hypertension, or are on treatment with digoxin; have transient mechanical or electrical failure or had a higher heart rate at study entry.

An early review showed that long-term use of beta-blockers after myocardial infarction reduces total mortality (RR about 0.80, NNT=48), sudden death (RR about 0.70, NNT=63), and non-fatal re-infarction (RR about 0.75, NNT=56).[678] Benefits persist for as long as treatment is continued. This finding has been confirmed in later reviews. One showed improved survival in people given long-term beta-blockers (OR 0.81, 95% CI, 0.75 – 0.87).[679] Another showed a similar reduction in mortality with long-term beta-blockers (OR 0.77, 95% CI, 0.69 – 0.85, NNT=44).[415] Trial length varied in these reviews with an average of 2 to 3 years.

In these reviews about 25% suffered beta-blocker side effects, though there is also a high placebo rate of side effects. Lipid soluble beta-blockers are more likely to cause insomnia or nightmares. The rate of side effects between beta-blockers with and without cardio-selectivity or between those with different membrane stabilising properties, is no different. Some concerns remain about the lack of efficacy of beta-blockers with intrinsic sympathomimetic activity in the long-term use after myocardial infarction.[678]

Although side effects are common, the benefits of beta-blockers post myocardial infarction are significant. There is a lack of evidence on the effectiveness of low-dose combination therapies of beta-blockers and ACE-inhibitors. Diabetes, controlled heart failure and minor peripheral vascular disease are no longer seen as absolute contraindications to beta-blocker therapy. The use of beta-blockers is contraindicated in people with reversible airways obstruction (asthma and some COPD), decompensated heart failure or heart block/bradyarrythmia.

The good practice points in the recommendations may help improve compliance and combat the high rate of discontinuation of beta-blockers due to side effects. Postural hypotension often limits the doses used in combination therapy.

Beta-blockers may be used, following a myocardial infarction, in people at high risk who have left ventricular dysfunction or controlled heart failure.[680] One trial of 1959 people with left ventricular dysfunction (ejection fraction <40%) after myocardial infarction, showed that the beta-blocker, carvedilol, added to conventional treatment including ACE-inhibitors, reduced all-cause mortality by 23% (RR 0.77, 95% CI, 0.60 – 0.98 ARR 3%, NNT=33 over 1.3 years). Only three beta-blockers have been investigated in treatment of heart failure, carvedilol, bisoprolol and metoprolol (more details are available in the New Zealand Heart Failure guideline).[6]

Although there is little evidence to inform the decision on whether to start a beta-blocker or ACE-inhibitor first, in patients with left ventricular dysfunction or heart failure an ACE-inhibitor is a primary treatment consideration. In general, the aim should be to establish combination treatment without undue delay following myocardial infarction.

ACE-INHIBITORS

RECOMMENDATIONS: ACE-INHIBITOR USE AFTER MYOCARDIAL INFARCTION AND STROKE	
AFTER MYOCARDIAL INFARCTION	
An ACE-inhibitor should be prescribed for everyone after myocardial infarction, regardless of left ventricular function. Treatment should be started early and continued long-term especially in those with anterior infarction, left ventricular dysfunction or heart failure. Long-term ACE-inhibitor therapy should be prescribed for all people with coronary heart disease.	A
AFTER STROKE	
Blood pressure lowering medication or increased doses of current agents should be started for people presenting after an acute ischaemic stroke or transient ischaemic attack unless they have symptomatic hypotension. An ACE-inhibitor in conjunction with a thiazide diuretic is an appropriate combination.	A
Blood pressure targets after a stroke should take into account the number and dose of medications prescribed as well as comorbidities and general frailty.	✓
It is advisable to wait 7 to 14 days after an acute stroke to start blood pressure lowering medication.	✓
AFTER MYOCARDIAL INFARCTION OR STROKE	
In general low-dose combination therapies are good choices. Periodic monitoring of electrolytes and renal function is recommended.	✓

EVIDENCE STATEMENTS

- In people who have had a myocardial infarction and have left ventricular dysfunction or heart failure, ACE-inhibitors reduce mortality, hospitalisation for congestive heart failure, and recurrent non-fatal myocardial infarction.(**1++**)
- A combination of ACE-inhibitor and thiazide diuretic reduces recurrent stroke and other major vascular events in people after ischaemic stroke or transient ischaemic attack.(**1++**)

Note: The benefit of ACE-inhibitor combinations used after stroke may not necessarily apply to all ACE- inhibitors/thiazide combinations. It is reasonable in practice to use any currently funded ACE-inhibitor/thiazide combination.

Note: When ACE-inhibitors are used to treat blood pressure the beneficial effect applies to all ACE-inhibitors (a class effect). A class effect may also apply to ACE-inhibitors when used after myocardial infarction and in heart failure. For people at high cardiovascular risk a benefit has been demonstrated with ramipril[140] and perindopril[683] but no other ACE-inhibitor. It is reasonable in practice to use any currently funded ACE-inhibitor. Ramipril is not funded in New Zealand at the time of publication.

One systematic review compared the ACE-inhibitors (captopril, ramipril and trandolapril) in placebo controlled trials where ACE-inhibitors were started early 3 to 16 days after myocardial infarction and continued for 15 to 42 months. In 5966 people following a recent myocardial infarction and

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with clinical manifestations of congestive heart failure or moderate left ventricular dysfunction (ejection fraction >35 – 40%) ACE-inhibitors significantly reduced mortality (OR 0.74, 95% CI, 0.66 – 0.83, NNT=17 over 2 years to prevent 1 death). ACE-inhibitors also reduced hospitalisation for congestive heart failure (OR 0.73, 95% CI, 0.63 – 0.85, NNT=28) and recurrent non-fatal myocardial infarction (OR 0.80, 95% CI, 0.69 – 0.94, NNT 43).[681]

One large trial of 9297 people at high risk of cardiovascular events (over 55 years of age, with previous cardiovascular disease or diabetes and one other risk factor) but without impaired ventricular function or evidence of congestive heart failure has shown that the ACE-inhibitor ramipril (10 mg/day) reduced the composite primary outcome of cardiovascular death, stroke, or myocardial infarction (RR 0.78, 95% CI, 0.70 – 0.86, NNT=26 over 4.7 years). Death from any caused was reduced by 16% (RR 0.84, 95% CI, 0.75 – 0.9,5 ARR 1.8%, NNT=56). Ramipril compared to placebo reduced the composite outcome in all subgroups examined, including women and men; people aged over and under 65 years; those with and without a history of coronary artery disease; hypertension, diabetes, peripheral vascular disease, cerebrovascular disease, and those with and without microalbuminuria at study entry.[140]

There are several other ongoing large randomized controlled trials evaluating ACE-inhibitors in people with coronary heart disease without clinical manifestations of heart failure and with no or mild impairment in left ventricular systolic function. These include one trial of trandolapril in 8000 people with coronary artery disease, and one trial of perindopril in 10,500 people with stable coronary artery disease.[682] A recent study of perindopril among 13,655 people with stable coronary artery disease without heart failure or substantial hypertension confirmed the benefit of ACE-inhibitors in preventing cardiovascular mortality, myocardial infarction or cardiac arrest in a broad population of post myocardial infarction patients.(RR 0.80, 95% CI, 0.71 – 0.91, NNT=50 over 4 years).[683] ACE-inhibitors are now recommended for everyone with coronary artery disease (without contraindications to ACE-inhibitors) regardless of left ventricular function.

One randomized clinical trial in people after stroke or transient ischaemic attack showed that the combination of perindopril and indapamide reduced stroke by about 27% (RR 0.73, 95% CI, 0.64 – 0.84, NNT=27 over 3.9 years). This trial excluded people within 14 days of stroke onset and this benefit was seen irrespective of the initial blood pressure. This result is consistent with the hypothesis that the reduction in stroke is mainly due to the greater lowering of blood pressure with the combination therapy. There is currently insufficient evidence to determine whether the reduction in stroke is specific to the combination of drugs used in this trial or if other blood pressure lowering drugs are equally effective. A 12/5 mm Hg reduction in systolic blood pressure was achieved in the combination therapy group.[407] The optimal time to start blood pressure lowering medication after stroke is uncertain. It should be noted that side effect data show that hyponatraemia with indapamide is more common than with other thiazides.

LIPID-MODIFYING AGENTS

RECOMMENDATIONS: USE OF LIPID-MODIFYING AGENTS AFTER MYOCARDIAL INFARCTION AND STROKE	
AFTER MYOCARDIAL INFARCTION	
A statin equivalent to simvastatin 40 mg/day should be prescribed to everyone after myocardial infarction. Statin therapy should preferably be started in hospital.	**A**
AFTER STROKE	
Treatment with a statin is recommended for most people following ischaemic stroke or transient ischaemic attack. Statin therapy should preferably be started in hospital.	**B**

EVIDENCE STATEMENTS

- Lowering total cholesterol, LDL-C after myocardial infarction substantially reduces overall mortality, cardiovascular mortality, and the risk of non-fatal cardiovascular events.(**1++**)
- Statins used post stroke reduce the risk of major cardiovascular events.(**1+**)
- Dyslipidaemic people with low HDL and elevated triglycerides benefit from intensive lifestyle interventions, fibrates and or combination therapies.

Note: Statins have different safety profiles and potency.

Lowering total cholesterol and LDL-C after myocardial infarction substantially reduces overall mortality, cardiovascular mortality, and the risk of non-fatal cardiovascular events. The absolute benefit increases as baseline risk increases. Prospective studies suggest there is a continuous constant gradient of risk, with no treatment threshold between total cholesterol levels of about 4 to 8 mmol/L. Within this range, treatment gives similar relative benefits regardless of baseline cholesterol levels. There is no evidence that reducing LDL-C to below 1.7 mmol/L confers additional benefit. In addition to its lipid-lowering effect statin therapy also has other beneficial effects including an anti-inflammatory effect and a positive effect on endothelial function.

All people post myocardial infarction should start intensive lifestyle advice concurrently with drug treatment. All efforts should be made to reach optimal lipid levels (see Chapter 9, *Intervention: Lipid Modification*). In people following CABG, treatment should aim to lower the total cholesterol less than 3.5 mmol/L and LDL-C less than 2 mmol/L.[370]

Early treatment of lipids in people with acute coronary syndromes has been shown to improve long-term survival[684] and helps with compliance.

After stroke or transient ischaemic attack, treatment with a statin reduces the risk of recurrent major vascular events.[69] A study also showed that treatment with simvastatin 40 mg per day is safe. The beneficial effect of lipid-modifying treatment was observed in a wide range of people at high-risk, including those with no history of ischaemic heart disease and those with low or normal cholesterol values. One thousand eight hundred and twenty people with prior stroke and no history of ischaemic

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heart disease were included in this trial; the reduction in major vascular events (including stroke) was similar to other subgroups. No conclusion can be reached regarding recurrent stroke rate by itself. Other major trials of statin therapy after ischaemic stroke are ongoing.

Some people with low HDL-C, high triglycerides, and clinical features of a metabolic syndrome may be better treated with other therapies for correction of the lipid disorder and require further assessment.

There is debate as to whether people without cardiovascular disease should be started on clinical trial dosages (eg, simvastatin 40 mg/day) or started on a low dose and titrated upwards. People with known cardiovascular disease will usually require doses of simvastatin in the range 20 to 40 mg per day to reach optimum lipid levels.[69,357]

One randomized controlled trial with gemfibrozil has shown a reduction in coronary heart disease events and ischaemic stroke.[363](**1++**) A trial of bezafibrate treatment in people with coronary artery disease showed increases in HDL-C and a non-significant trend towards a reduction in fatal and non-fatal coronary events.[364]

ANTIARRHYTHMIC AGENTS

RECOMMENDATION: USE OF ANTIARRHYTHMIC AGENTS AFTER MYOCARDIAL INFARCTION

AFTER MYOCARDIAL INFARCTION

Antiarrhythmic therapy, apart from beta-blockers, is not recommended for routine use after myocardial infarction. **A**

EVIDENCE STATEMENT

- The routine use of class one antiarrhythmic drugs administered after myocardial infarction is associated with increased mortality.(**1++**)

The most commonly used antiarrhythmic agents in New Zealand are amiodarone, sotalol and flecanide. Specialist advice is recommended before the initiation of these therapies.

Approximately 4 to 18% of people post acute myocardial infarction, suffer potentially fatal ventricular fibrillation in the first 24 to 48-hours. Three-quarters of the sudden deaths occurring in the first year post myocardial infarction are caused by either ventricular tachycardia or fibrillation. As a result of these observations, a variety of trials have been undertaken with the prophylactic use of antiarrhythmic drugs post myocardial infarction. A meta-analysis of 138 trials included 18 long-term trials of class 1 agents after myocardial infarction and has shown a 21% increase in mortality with this antiarrythmic treatment (OR 1.21, 95% CI, 1.01 – 1.44).[679]

HORMONE REPLACEMENT THERAPY

RECOMMENDATION: HORMONE REPLACEMENT THERAPY AFTER MYOCARDIAL INFARCTION AND STROKE

AFTER STROKE OR MYOCARDIAL INFARCTION

Combined Hormone Replacement Therapy should not be used for the prevention of coronary heart disease/stroke or after a cardiovascular event.	**A**

Hormone Replacement Therapy (HRT) has no role in primary or secondary prevention of cardiovascular disease in women. The NZGG hormone replacement therapy guideline has recently been updated to take account of data from the *Heart and Estrogen/Progestin Replacement Study Follow-up* (HERS II)[685,686] and the Women's Health Initiative studies.[687] Combined HRT is effective for the control of troublesome menopausal symptoms of hot flushes and night sweats. However, even short-term use is associated with an increased risk of venous thromboembolism, stroke and coronary heart disease. HRT should only be used where menopausal symptoms are troublesome and women are fully informed of the risks.

CALCIUM CHANNEL BLOCKERS

RECOMMENDATION: CALCIUM CHANNEL BLOCKER USE AFTER MYOCARDIAL INFARCTION AND STROKE

AFTER MYOCARDIAL INFARCTION

Rate-limiting non-dihydropyridine calcium channel blockers may be considered for people with normal ventricular function where beta-blockers are contraindicated and treatment is required for concurrent angina or hypertension.	**A**

EVIDENCE STATEMENT

- Calcium channel blockers do not reduce mortality following myocardial infarction.(**1++**)

Calcium channel blockers are not a homogenous pharmacological group but can be broadly divided into two, the rate-limiting non-dihydropyridine calcium channel blockers (verapamil or diltiazem) and the dihydropyridine calcium channel blockers (nifedipine, felodipine and amlodipine). In people with impaired ventricular function the rate-limiting calcium channel blockers and those with negative inotropic effects may be harmful. Clinical trials of verapamil[688] and diltiazem[689] in people with normal left ventricular function have not shown a reduction in mortality or re-infarction. There is no conclusive evidence relating to the effect on mortality and re-infarction of other calcium channel-blocking drugs, eg, nifedipine, amlodipine, following myocardial infarction. Short acting dihydropyridines may be harmful.

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After myocardial infarction and stroke addition of calcium channel blockers is effective in reducing blood pressure, particularly systolic blood pressure, controlling angina and in treating some supra-ventricular tachycardias.

NITRATES

RECOMMENDATION: NITRATE USE AFTER MYOCARDIAL INFARCTION	
AFTER MYOCARDIAL INFARCTION	
Nitrates can be used after myocardial infarction for controlling syptoms of angina and heart failure, but are not indicated for reducing the risk of further events.	**A**

EVIDENCE STATEMENTS

- No reduction in mortality has been shown after 4 to 6 weeks' use of nitrates post myocardial infarction.[690,691] **(1+)**
- Nitrates are effective in controlling the symptoms of angina and occasionally heart failure but have no proven benefit for long-term use post myocardial infarction.**(4)**

WHEN TO START THERAPY AFTER MYOCARDIAL INFARCTION OR STROKE

RECOMMENDATIONS: WHEN TO START THERAPY AFTER MYOCARDIAL INFARCTION AND STROKE	
AFTER MYOCARDIAL INFARCTION	
Most therapies will have been started in hospital. Some people, on review in primary care, will require initiation or dose adjustment.	✓
AFTER STROKE	
Aspirin should be started as soon as possible after ischaemic stroke. Warfarin and statins should be started in hospital. Blood pressure lowering therapy with ACE-inhibitor and thiazide treatment should be started after 7 to 14 days.	✓

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15

CARDIOVASCULAR HEALTH OF PACIFIC PEOPLE

KEY POINTS

- The burden of cardiovascular disease falls heavily on Pacific people.
- Current cardiovascular care provision for Pacific people in New Zealand demonstrates the inverse care law — that those most in need receive the least care.

RECOMMENDATIONS: CARDIOVASCULAR HEALTH OF PACIFIC PEOPLE	
Consider that difficult socioeconomic circumstances present significant barriers to Pacific people's access to quality health care.	✓
Be cognisant of the diversity and heterogeneity of the Pacific population – made up of different Pacific ethnic groups each with a distinct language and culture.	✓
Be aware that the English language may be a barrier. Pacific ethnic-specific translators should be made available within services that provide for Pacific people.	✓
Absolute cardiovascular risk determines treatment decisions and benefits. Within this guideline, cardiovascular risk assessment is recommended 10 years earlier for Pacific people.	✓
Recognise the importance of the family to Pacific people. Involve family members in the management of the person's illness and suggested dietary and lifestyle changes.	✓
Invest in the development of Pacific ethnic-specific cardiovascular workforce and resources.	✓
Collect quality Pacific health information (including Pacific ethnic-specific data).	✓
Support and partner with Pacific providers and Pacific communities to promote cardiovascular health for Pacific people.	✓

INTRODUCTION

Pacific people die almost a decade earlier than Europeans in New Zealand. Cardiovascular disease contributes significantly to premature deaths and morbidity among Pacific people.[27] Improving the cardiovascular health outcomes for Pacific people is an urgent priority.

BURDEN OF DISEASE

The burden of cardiovascular disease falls heavily on Pacific people. Cardiovascular disease is the leading cause of death for Pacific people, accounting for 41% of all deaths in 1997, thus it has a large impact on health care service delivery.

The reductions in age-standardised mortality from coronary heart disease in the New Zealand population, since the late 1960s, have been smaller for Pacific people than for the European population.[28] Therefore, the disparities in cardiovascular disease prevalence for Pacific people compared to Europeans have widened during the 1980s and 1990s.[29]

Mortality rates for cerebrovascular disease are higher in Pacific people (115/100,000) than in Māori (82/100,000) and Europeans/others (64/100,000).[6,27] The average age of stroke for Pacific people is 60 years, compared to 55 years for Māori and 73 years for Europeans. The chances of being dependent at 12 months after a stroke are three times higher among Pacific people than among Europeans.[23]

The mortality rate from coronary heart disease in Pacific people is 199 per 100,000 compared to a rate of 158 per 100,000 for Europeans.

Pacific people have the highest hospitalisation rates for rheumatic fever with 19 per 100,000; twice that of Māori and 10 times that of Europeans. The hospitalisation rates for rheumatic heart disease were 22 per 100,000 for Pacific people; three times higher than for Europeans.[27]

The hospital discharge rate for heart failure, 26.3 per 10,000 for Pacific people, is more than twice the rate for Europeans.[27]

Cardiovascular Care Provision

The current cardiovascular care provision for Pacific people in New Zealand demonstrates the inverse care law which states that the availability of health care varies in an inversely proportional manner to the need of the population served, so that those most in need receive the least care.[37] The level of health service intervention for Pacific people with cardiovascular disease does not reflect or match the degree of health need, as expected from the burden of cardiovascular disease morbidity and mortality data.[27,39]

Despite Pacific people having higher rates of coronary artery disease morbidity and mortality, their revascularisation rates were lower than those of Europeans. A recent New Zealand study showed that Pacific people received significantly lower rates of CABG and PTCA interventions when clinically indicated. Pacific women received 73% of CABG and 21% of PTCA interventions that European women received. Pacific men received 64% of CABG and 25% of PTCA interventions that European men received.[36]

For the 1997–2002 period, the Health and Independence Report 2002[703] found that the disparities in the standardised discharge rates for angioplasty between Pacific people and Europeans had not improved. A slight improvement in the standardised discharge rates for CABG in Pacific people for the 2001/02 period was found. However, since Pacific people are at higher overall risk of death and

morbidity from cardiovascular disease, it is likely that significant barriers still exist for many Pacific people accessing appropriate cardiovascular care and interventions.

Differential treatment and discrimatory practice by practitioners may influence the access of Pacific people to high quality cardiovascular services. Service provision at primary, secondary and tertiary care services indicate inadequate access and provision for Pacific people.

The above findings for cardiovascular care[36,41,704] are consistent with other New Zealand studies investigating access to asthma and diabetes care among Pacific people. These studies have suggested that health practitioner bias influences access to high quality care.[42,705,706]

Health service barriers and the attitude of some health care workers adversely affect Pacific people's ability to adequately access health care services. They may also undermine good policy strategies to reduce health inequalities and improve health outcomes for Pacific people.

POPULATION HEALTH APPROACH

The use of evidence-based guidelines in the clinical decision-making process has the potential to improve health outcomes for Pacific people and to reduce health inequalities.

This guideline supports a population health approach to cardiovascular risk assessment and management. A lifecourse approach to cardiovascular risk assessment and management is also recommended. A stepwise approach to management based on absolute risk means that effective interventions need to be tailored to the population groups who bear the greatest burden of cardiovascular disease. Population groups who have the highest risk need to be targeted and managed more aggressively than those at lower risk, across their entire lifecourse.

Interventions that focus on individuals and emphasise changing lifestyle behaviours alone have limited potential to improve health outcomes for Pacific people. In addition to screening for individual cardiovascular risk factors, practitioners need to be cognisant of the broader socioeconomic determinants of health that significantly impact on Pacific people's health and well-being.

Actions to improve health outcomes for Pacific people require a multi-pronged approach and intersectoral collaborations to address socioeconomic disadvantages and differentials in income, employment, education, and housing which form the structural root causes of ill-health among Pacific people.

Effective partnership with Pacific communities is urgently needed to improve service provision for Pacific people. Pacific providers, health workers, church and community leaders play a key role in negotiating a safe, trusting and effective partnership between mainstream health services and Pacific communities.

Population health programmes, personal health services and disability support services for Pacific people should:

- target the diverse Pacific population, accommodating the needs of both New Zealand-born Pacific people, recent migrants and older Pacific Island born migrants
- be community-based and delivered within the context of Pacific people and their families. Services need to be developed in partnership with Pacific communities
- have a holistic approach to health and be cognisant of the broader determinants of health
- develop quality Pacific ethnic-specific resources and translated into Pacific languages
- employ and train Pacific ethnic-specific health workers to be part of service planning and development, delivery and evaluation

- train non-Pacific practitioners to be aware and be sensitive to the realities and barriers to health care access that Pacific people face
- collect quality Pacific information (including Pacific ethnic-specific data) routinely, consistently and completely
- audit and evaluate their services using key performance indicators that monitor responsiveness to Pacific cardiovascular need and ensure continuing quality improvement.

CONCLUSION

Improving health outcomes and reducing health inequalities for Pacific people is an important and urgent priority. Applying evidence-based practice to the clinical decision-making process has the potential to improve health outcomes and reduce the inequalities in cardiovascular health for Pacific people.

IMPLEMENTATION

PRIORITY GROUPS

The Guideline Development Team strongly supports an approach to implementation that will address past inequities in service provision and the disparities in cardiovascular outcomes that exist between ethnic groups in New Zealand. A population health approach that uses intersectoral action to address socioeconomic, ethnic, sex and geographic inequalities is proposed.[39] This guideline has a personal health perspective which recognises that the attitudes, skills and resources available to individual providers of care can also reduce inequalities and potentially improve the health of all New Zealanders. Practitioners have an important role in identifying disadvantage among individuals and groups, identifying barriers to access for those who are disadvantaged, recording ethnicity accurately and being sensitive to cultural requirements.

There is a danger that any organised programme of risk assessment will preferentially serve those with the least need or lowest risk (the inverse care law[37]). This risk is greatest with an opportunistic screening strategy and there is evidence that screening programmes achieve lower coverage of Māori despite the increased risk.[692] The New Zealand criteria to assess screening programmes specify in criterion six that 'any screening programme reaches those who need it most, which may require specific initiatives to reach particular population groups'.[1] This guideline offers advice at an individual level to providers of health care and recommends that implementation proceeds first for the priority groups, Māori, Pacific peoples, and in the geographical areas of New Zealand with the highest rates of cardiovascular disease.

Māori and Implementation

A key message of this guideline is that cardiovascular risk assessment and management should be prioritised to high-risk groups. The guideline recommends that Māori are assessed for cardiovascular risk 10 years earlier than non-Māori (ie, from the age of 35 years for Māori men and 45 years for Māori women). There is an urgent need to focus intervention programmes on Māori, who bear the greatest burden of cardiovascular disease in New Zealand. Promoting change at three levels: policy, systems and practitioner/patient levels is required.

Implementation of the Māori Cardiovascular Action Plan

The Māori Cardiovascular Group has identified several categories of action with the overall aim of improving Māori cardiovascular health and removing inequalities in cardiovascular disease outcomes between Māori and non-Māori in New Zealand. The actions proposed are:

1. policy: Treaty of Waitangi-based policy and decision-making
2. information systems: complete and consistent collection of ethnicity data and service provider funding
3. access, delivery and standards development: cardiovascular health needs assessment and kaupapa Māori health services
4. audit, evaluation and quality standards improvement: measurement of key performance indicators to monitor service responsiveness to Māori cardiovascular need.

Implementation of the Guideline

For this guideline to be successfully implemented for Māori, the implementation process must:

- prioritise Māori cardiovascular health gain
- address the Treaty of Waitangi and indigenous rights
- promote equity in the provision and use of health services
- consult Māori stakeholders and communities
- use Māori health models
- audit and evaluate the implementation process.

If cardiovascular health services and programmes are to be accessible and effective for Māori, planning and priority setting will need to be undertaken.

Targeting Interventions for Māori

The following set of questions can assist those setting up cardiovascular programmes to target interventions for Māori.

1 What cardiovascular health problem is the programme trying to address?
2 What inequality exists in this health area?
   - What is the size/nature of the inequality?
   - How did the inequality occur? (What are the mechanisms by which this inequality is created, maintained, made worse?)
   - What are the determinants of the inequality?
3 Who is most advantaged by current services, and how?
4 How will the programme address the Treaty of Waitangi and indigenous rights?
5 What is the most appropriate and effective design for this programme?
   - What will the policies, objectives, strategies and priorities be?
   - What level of Māori consultation/participation/involvement will there be?
   - What services do Māori patients and whānau want?
   - Where will the service(s) be located?
   - What training and education do staff need for this programme?
   - What patient resources and educational materials will be used?
   - What might the unintended consequences of the programme be?
   - How will these be mitigated?
6 What are the resource implications?
7 What type of evaluation will be undertaken?
   - What inequality outcomes will be evaluated?

Based on Bro Taf Authority. 2000. Planning for Positive Impact: Health Inequality Impact Assessment Tool available at www.bro-taf-ha.wales.nhs.uk/pages/policies_raceequality.pdf

Pacific Peoples

The Pacific Cardiovascular Working Party has drafted a Cardiovascular Health Action Plan that has stated a goal to reduce the incidence and impact of cardiovascular disease in Pacific peoples in New Zealand. This is published separately and includes the following objectives:

- to reduce the incidence and impact of cardiovascular disease for Pacific peoples within each District Health Board (DHB) and to provide tools to enable this process
- to improve access for Pacific peoples to mainstream cardiovascular disease services, Primary Health Care Organisations (PHOs) and other Pacific providers
- to influence the development of the Pacific cardiovascular disease workforce
- to encourage and support healthy lifestyles for Pacific peoples
- to influence other Government agencies that have policies that affect and impact on Pacific peoples cardiovascular health.

Important actions suggested include ensuring that PHOs and other health promotion providers develop and implement culturally competent models for Pacific peoples that encourage and support healthy lifestyles which include nutrition, physical activity and smokefree advice. Health promotion activities and materials should recognise the unique cultural differences of Pacific peoples.

IMPLEMENTATION PLAN

The Guideline Development Team recommends that the following multifaceted strategies are adopted to disseminate the guideline and encourage its implementation through New Zealand. There is evidence that information transfer and learning through social influence and management support can be effective in implementing guidelines and innovations in general practice, as can reminders and feedback.[693] Information exchange is probably always required, but additional interventions are usually needed to achieve real change in practice routines. Cardiovascular risk assessments can be made using simple paper-based or electronic risk-prediction tools and many of these are now available, predicting different outcomes over different time periods using different risk factor combinations. There is increasing evidence that paper-based or electronic tools are usable in clinical practice[694,695] and lead to improved risk management.[696,697] Important features of electronic decision support are the ease of use and the integration of tools into the electronic medical record so that they are only a mouse click away. The relevance and accuracy of messages and the flexibility to respond to other factors influencing decision-making have been identified as key factors in implementing electronic decision support tools in primary care.[698]

Specific Distribution Strategies

Endorsement

Endorsement for the guideline was sought from stakeholder organisations with an interest in promoting the key messages. At the start of the process a number of organisations nominated team members to be part of the Guideline Development Team and endorsement from these organisations was sought after they had reviewed an advanced copy of the final guideline.

Quick Reference Guide

A quick reference guide including the key messages from the guideline is to be prepared and distributed to all primary care practitioners and included in key primary care magazines and publications.

Publication in Full

This is to be available electronically at the NZGG website (www.nzgg.org.nz) at no charge and will be circulated to DHBs, Independent Practitioner Organisations (IPAs), PHOs, Local Diabetes Teams (LDTs) and pharmacy facilitators. The NZGG website will also provide downloadable supporting information, quizzes, video clips and evidence tables for people seeking more detail.

Dissemination

Further dissemination is planned to reach all primary care practitioners. The following groups will also receive a summary document and the quick reference guide – national regulatory bodies, Medical and Nursing Colleges, IPAs, PHOs, DHBs and LDTs. Other provider organisations, dietitians, pharmacists, support groups, consumer and interest groups, commercial organisations and drug companies.

There is a need for a co-ordinated marketing campaign to advise people of the benefits of a risk assessment, and to develop an awareness of ways they can reduce their risk through lifestyle modification. Agencies including the Ministry of Health, PHARMAC, the Ministry of Sport and Recreation should co-ordinate these programmes to proactively promote lifestyle change.

Events, Presentations and Training

National and Local Events

The launch of the guideline will attract media interest and signal the start of the implementation phase. The launch will coincide with the launch of the New Zealand diabetes and stroke guidelines. Further opportunities for presentation at local postgraduate education meetings will help health care practitioners become familiar with the guideline.

Local Feedback Adaptation

This will be encouraged through presentations by members of the Guideline Development Team at local general practitioners' peer review meetings, and as part of PHO project planning initiatives. A clinical focus will be maintained through case studies and scenario-based teaching strategies.

Education Initiatives

The guideline and supplementary resources will be freely available for use in the education and training of pharmacy facilitators, general practitioners, nurse practitioners, nurses and pharmacists. Best Practice Advocacy Centre (BPAC) plans to run a series of case studies based on the guidelines. Other opportunities for web-based learning and the provision of CME points for courses completed will be investigated.

Champions

Local champions should be identified by DHBs and resources provided (powerpoint presentations, full guidelines, pre-prepared case studies etc) to support them in developing local, comprehensive and high quality cardiovascular risk assessment services. Expert members of the guideline team will be invited to prepare articles and publications for local and international journals and publications.

Audit

DHBs should audit the referral patterns, medication prescribing, outcomes and process indicators from risk-assessment programmes by PHOs. Audit is seen as a key quality improvement activity to promote a change in practice and uptake of the guideline.

Practical Tools, Electronic Decision Support

The National Heart Foundation's cardiovascular risk tables are already widely distributed in primary care. Further copies will be printed to accompany the guideline and will be included in consumer resources.

Electronic decision support tools are available in New Zealand and a process for incorporating the additional or changed messages from this guideline will be developed.

QUALITY IMPROVEMENT

Clinical Practice Improvement

Clinical practice improvement (CPI) has been defined as an evidence-based process for improving clinical practice that uses such tools as clinical pathways, outcome and performance indicators, clinical measurement and review in a continuous quality improvement cycle supported by appropriate information systems.[699] The assessment and management of cardiovascular risk is an activity that is ideally suited to such an approach. The adaptation of this guideline to suit the particular needs of local communities and providers of health care is encouraged and there is scope for the development of innovative programmes and approaches within the context of the current recommendations.

Quality improvement programmes have been shown to improve the practice of recording cardiovascular risk factors and result in earlier detection of people with a high risk of developing the disease. In one such study the risk factor levels in high risk people receiving treatment decreased over the duration of the study.[700]

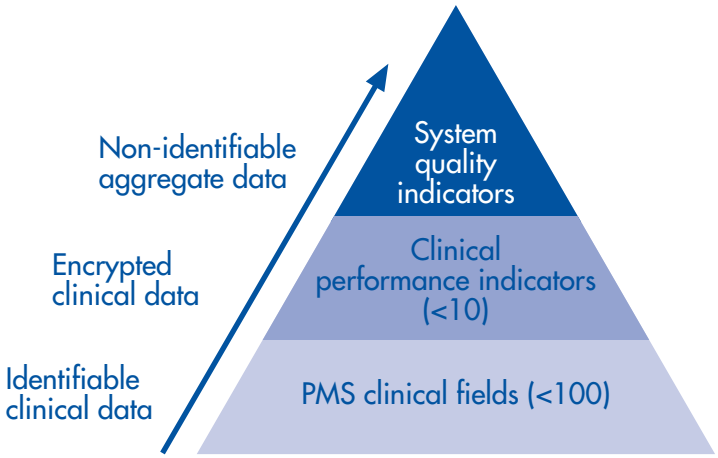
Indicators

The Guideline Development Team developed a strategy for selecting and evaluating indicators. An initial set of indicators were chosen including all the indicators in the Māori Cardiovascular Action Plan, current indicators and new indicators suggested by the recommendations in this guideline. A framework for evaluating these was developed based on the Victorian Acute Health Clinical Indicator Project framework[701] which specifies nine areas of evaluation and categories within each area for potential indicator development. To this has been added the domain of 'equity'. These areas are:

1. preventive care
2. access to care
3. clinical care
4. appropriateness of care
5. safety of care
6. effectiveness of care
7. continuity of care
8. satisfaction with care
9. organisational management
10. equity.

By considering the proposed indicators within these domains a comprehensive set of performance indicators was developed to address the needs of quality improvement at an institutional or local level while concurrently serving as reliable and valid measures for external accountability. A scoring system/review tool assessing each indicator was applied to the indicator set. **The final set of indicators based on Figure 6 is contained in Appendix A.**

Figure 6: A framework for performance indicators



The indicators proposed were selected for inclusion according to a rating scale which scored aspects of:

- importance
- scientific soundness
- data availability.

It is suggested that these indicators listed in Appendix A are regularly reviewed.

Clinical Pathway

The desktop reference (published separately) is designed for use by a multidisciplinary primary care team. The data collected by the practice team, using the Practice Management System (PMS) would be summarised by the practice or PHO into information for quality-improvement activities, peer review and clinical audit.

APPENDICES

A Performance indicators

B *Cardiovascular risk screening and lipid-lowering treatment in their economic context.* Executive summary of a report commissioned by the New Zealand Guidelines Group

C Additional resources and supporting documents

PERFORMANCE INDICATORS

DESCRIPTION OF PERFORMANCE INDICATORS

System Quality Indicators

CVD1: Primary care organisations have systems in place to identify and manage both people at increased cardiovascular risk and those with known cardiovascular disease, including systems to improve quality, monitor care and arrange for follow-up and recall.

CVD2: Hospitals and primary care organisations have systems in place to accurately record the self-identified ethnicity of all people seen or enrolled.

CVD3: Hospitals have clinical pathways in place to cover the management from admission to discharge of the acute and chronic cardiovascular conditions: acute coronary syndromes (unstable angina, acute myocardial infarction), heart failure, CABG, PTCA, transient ischaemic attack and stroke.

CVD4: These pathways and systems are monitored to ensure that people receive appropriate lifestyle advice, medication, referral to phase 2 cardiac rehabilitation and that information is sent to other providers for ongoing care co-ordination.

Clinical Performance Indicators

CVD5: The proportion of Māori men aged 35 and above and Māori women aged 45 and above who have had their 5-year absolute CV risk recorded in the last 5 years.

CVD6: The proportion of Pacific men or men from the Indian subcontinent aged 35 and above and Pacific women or women from the Indian subcontinent aged 45 and above who have had their 5-year absolute CV risk recorded in the last five years.

CVD7: The proportion of men identifying as European or 'other' aged 45 and above and women identifying as European or 'other' aged 55 and above who have had their 5-year absolute CV risk recorded in the last five years.

CVD8: The proportion of men or women with diabetes who have had their 5-year absolute CV risk recorded in the last five years (reported by age, sex and ethnicity).

CVD9: The proportion of people who have a 5-year absolute CV risk of 15% and above who

- have received intensive lifestyle advice
- are prescribed aspirin
- are prescribed a statin
- are prescribed blood pressure lowering medication

(reported by age, sex and ethnicity).

CVD10: The proportion of people with cardiovascular disease who

- have received intensive lifestyle advice
- are prescribed aspirin
- are prescribed a beta-blocker

- are prescribed a statin
- are prescribed an ACE-inhibitor

(reported by age, sex and ethnicity).

CVD11: The proportion of people with IGT who have received lifestyle advice from a dietitian and have been referred to an intensive physical activity programme (reported by age, sex and ethnicity).

CVD12: The proportion of people who smoke who have completed a smoking cessation programme (reported by age, sex and ethnicity).

CVD13: The proportion of people with genetic lipid disorders who have received family tracing follow-up (reported by age, sex and ethnicity).

CVD14: The proportion of people discharged following an acute coronary syndrome (unstable angina, acute myocardial infarction) heart failure, CABG, PTCA who:

- have received intensive lifestyle advice
- are prescribed aspirin
- are prescribed a beta-blocker
- are prescribed a statin
- are prescribed an ACE-inhibitor
- are referred to phase 2 cardiac rehabilitation
- have had relevant clinical information sent to their nominated GP

(reported by age, sex and ethnicity)

CVD15: The proportion of people discharged following a transient ischaemic attack or stroke who:

- have received intensive lifestyle advice
- are prescribed aspirin
- are prescribed a statin
- have had relevant clinical information sent to their nominated GP

(reported by age, sex and ethnicity).

CVD16: The proportion of smokers who have been discharged following unstable angina, acute myocardial infarction, heart failure, CABG, PTCA, transient ischaemic attack and stroke who have been referred to a smoking cessation programme (reported by age, sex and ethnicity).

CVD17: The proportion of people who have been discharged from hospital services following unstable angina, acute myocardial infarction, heart failure, CABG and PTCA who have completed a phase 2 cardiac rehabilitation programme (reported by age, sex and ethnicity).

CVD18: The proportion of people who have been discharged from a phase 2 cardiac rehabilitation programme following unstable angina, acute myocardial infarction, heart failure, CABG and PTCA who have been referred to a phase 3 cardiac rehabilitation programme (reported by age, sex and ethnicity).

CARDIOVASCULAR RISK SCREENING AND LIPID-LOWERING TREATMENT IN THEIR ECONOMIC CONTEXT

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This is the Executive Summary of a report commissioned by the New Zealand Guidelines Group. The full report[52] is available at www.nzgg.org.nz

EXECUTIVE SUMMARY

This report sets decision-making about cardiovascular risk screening and clinical management into an economic context. It focuses on risk screening and lipid-lowering therapy for individuals without known cardiovascular disease, because a risk screening programme is recommended in these cardiovascular guidelines and lipid-lowering statin therapy has recently been made available for primary prevention of cardiovascular disease in New Zealand. We assess the economic impact for New Zealand of a 'screen and treat' strategy that is recommended by the NZGG Guideline Development Team for the assessment and management of cardiovascular risk, guided partly by these economic analyses. Therapy is maintained for 5 years and benefits are evaluated over the lifetime of the treated cohort. The comparator is clinical practice prior to April 2002, when access to statins was largely restricted to persons with cardiovascular disease.

The guideline recommends comprehensive global cardiovascular risk screening of men aged 45 years or over and women aged 55 years or over without known cardiovascular disease; known smokers and Māori, Pacific peoples and people from the Indian subcontinent are to be screened 10 years earlier. People with diabetes are to be screened at diagnosis. We report the benefits and costs of a 5-year screening programme coupled with 5 years of lipid-lowering therapy for all eligible men and women. We assume that equal numbers of individuals are identified each year over the 5-year period following introduction of the guideline. We also assume that the results of previous lipid tests (if any) will need to be confirmed under the new guideline.

The analysis takes a health care perspective and includes costs to the Ministry of Health as the major third party funder plus private expenditure on health. It excludes non-medical costs such as patient time and transportation and indirect costs including loss of income and/or productivity.

Cardiovascular risk is estimated using a Framingham Heart Study risk equation that predicts the risk of 'any incident cardiovascular event' including myocardial infarction, stroke, angina, transient ischaemic attack, peripheral vascular disease or congestive heart failure. This equation, which is the basis of the previous and the revised guidelines, predicts first cardiovascular admissions and deaths reasonably accurately at a population level. The analysis of receiver operator characteristics of the Framingham Heart Study risk equation indicates that at clinically useful treatment thresholds, its specificity is over 90% but its sensitivity is poor. This highlights the need to complement targeted therapy with population risk reduction strategies.

A risk screening and treatment programme is assumed to be implemented gradually over 5 years and we assume that equal numbers of individuals are screened over years 1 to 5. We also assume that risk screening requires 5 minutes of general practitioner (GP) time and/or practice nurse time plus a full lipid profile in duplicate and one blood glucose test. Individuals who are identified by the screening programme as having 5-year cardiovascular risk greater than 15% are treated for 5 years with simvastatin* at a starting dose of 20 mg per day titrated to 40 mg per day for one-third of these individuals. Individuals with 'extreme' lipid profiles defined as TC:HDL greater than 8 mmol and/or total cholesterol greater than 8 mmol/L are offered atorvastatin 40 mg per day, regardless of their estimated risk. Drug acquisition costs comprise contracted prices# plus pharmacy mark-up and dispensing fees.

Liver function (alanine amino-transferase) and creatine kinase tests are utilised before and after initiation of statin therapy. After therapy is instigated, full lipid profiles are obtained quarterly in year 1, then biannually for 4 more years. We assume that monitoring of drug therapy entails 20 minutes of GP time in year 1 then 10 minutes per annum in years 2 to 4.

Over 5 years, this strategy would screen 570,000 men and 350,000 women at a (discounted) 5-year cost of $25.8m. This includes $15.3m in laboratory tests plus $10.5m in GP consultation fees, representing a case load equivalent of 12 additional full-time GPs.

Therapy to prevent or delay cardiovascular events can be multifactorial (ie, lifestyle measures, blood pressure lowering and lipid modifying drugs can be utilised concurrently). We assume that lifestyle modification such as smoking cessation and weight reduction therapy has been attempted and that blood pressure lowering therapy is utilised for high risk individuals with TC:HDL less than 4.5 but with elevated blood pressure. In the base case analysis, individuals are offered lipid-lowering therapy if their 5-year risk is at least 15% and TC:HDL greater than or equal to 4.5 or they have 'extreme' lipid values (total serum cholesterol or cholesterol ratio (TC:HDL) greater than 8.0).

Prevention of cardiovascular events with lipid-lowering therapy avoids hospital admissions and community costs of ongoing medical treatment including quarterly GP consultations and medications such as aspirin, beta-blockers and ACE-inhibitors. The cost of 5 years of hospital admissions with a primary cardiovascular diagnosis following a non-fatal first admission over a 5-year period were obtained from routine statistics, based on WEIS5A, medical and surgical plus assessment, treatment and rehabilitation costs. Case fatality at 30 days, 1 year and 5 years beyond a first cardiovascular admission or death was also obtained from routine statistics. The costs of medication for secondary prevention, excluding simvastatin, were obtained from the Pharmaceutical Schedule and the cost of simvastatin was obtained from PHARMAC. Life expectancy was obtained from period life tables and utility (a single index measure of quality of life) for specified cardiovascular endpoints was obtained from the published literature. All costs and benefits were discounted to present value at 5% per annum.

The summary table shows the 5-year costs, lifetime health benefits and cost utility of the proposed 'screen and treat' strategy compared with clinical practice prior to April 2002. For comparison, it also shows the average and incremental benefits, costs and cost utility of broadening the treatment target to include individuals with risk in the range 10 to 15%.

*Nothing in this document is intended to imply that simvastatin and atorvastatin are the only appropriate lipid modifying agents, however these agents are fully reimbursed by PHARMAC and widely prescribed. Fibrates were not evaluated because they are not widely utilised.
#Supplier rebates on simvastatin are excluded

Summary Table

Benefits, costs and cost-effectiveness of a 'screen and treat' strategy at 15% and 10% treatment thresholds (age 35 – 84, threshold lipid ratio 4.5, discount rate 5% per annum, 2003 dollars) from a health care perspective.

Numbers	Men	Women	Total	NNS (events)
Screened	573,758	347,281	921,039	
Treated (>15% risk)	106,937	48,106	155,043	137
Treated (>10% risk)	179,038	101,004	280,043	106
Treated (10 – 15% risk)	72,101	52,898	124,999	

Benefits	Events averted	Deaths delayed	LYG	QALYs gained	NNT (events)
>15% risk	6716	1885	17,205	21,317	23
>10% risk	8716	2312	21,969	28,107	32
10 – 15% risk	2000	427	4763	6789	

5-year Costs ($m)	Pharms	Labs	GMS	Co-payments	Hospital cost offset	Net cost
>15% risk	$63	$15	$4	$29	-$40	$70
>10% risk	$108	$26	$6	$55	-$53	$142
10 – 15% risk	$45	$12	$2	$25	-$13	$72

Cost-effectiveness	Cost per event averted	Cost per premature death averted	Cost per life year gained	Cost per QALY
>15% risk	$10,459	$37,269	$4083	$3295
>10% risk	$16,263	$61,303	$6452	$5043
10 – 15% risk	$35,753	$167,276	$15,011	$10,532

Abbreviations: **Co-payments** = patient payments; **GMS** = General Medical Services benefit; **Labs** = laboratory tests; **LYG** = life years gained over a lifetime; **NNS** = number of persons screened and treated to prevent one event in 5 years; **NNT** = number of persons treated to prevent one event in 5 years; **Pharms** = pharmaceuticals; **QALY** = quality adjusted life year.

Compared with no systematic screening and lipid-lowering programme, the recommended 'screen and treat' strategy (at a treatment threshold of 15% absolute risk) would prevent 6716 incident cardiovascular events and 1885 premature deaths. Over the lifetime of the cohort this provides 17,205 life years and 21,317 QALYs. The net 5-year cost of the strategy is $70m including pharmaceuticals, GP consultations and laboratory tests. Hospital costs avoided over 5 years (including incident and recurrent medical and surgical cardiovascular admissions, AT&R and rural admissions) offset about two-thirds of the net cost of pharmaceuticals.

The cost-effectiveness ratios of $4083 per life year gained and $3295 per QALY gained are well within the range of those that have been accepted in reimbursing novel drug therapies in New Zealand. Enlarging the pool of individuals who are eligible for statin treatment by reducing the treatment threshold from 15 to 10% absolute risk increases the QALY benefits by about one-third but doubles the total cost of the strategy.

These analyses assume that adherence to therapy is 80% of that achieved in the *Heart Protection Study*, in which 85% of individuals took at least 80% of the tablets over a 5-year period. In practice, adherence may be even lower, with proportionally lower costs and benefits and slightly higher cost effectiveness ratios. Targetting Māori people at a threshold age 10 years lower than non Māori is likely to be at least as cost effective as targetting non Māori people, but insufficient information is available to estimate cost effectiveness ratios. Even less information is available for peoples from the Pacific or Asia

The cost-effectiveness ratio is very sensitive to the screening age threshold but only moderately sensitive to the treatment thresholds (absolute risk 15% or 10% and lipid ratio TC:HDL 4.5 – 6.5), the 5-year case fatality following an incident cardiovascular event, hospital costs, adherence to treatment and long-term health utilities following an event. The incremental cost per QALY remains less than $NZ10,000 over a wide range of these values, indicating that a 'screen and treat' strategy is very cost-effective compared to no such strategy. Further work is required to analyse the effects of lifestyle modifications, antiplatelet and blood pressure lowering therapy in conjunction with lipid-lowering therapy in primary prevention of cardiovascular disease.

In conclusion, global screening for cardiovascular risk, coupled with lipid-lowering therapy at a 5-year risk threshold of either 15% or 10% and a lipid ratio TC:HDL greater than 4.5, is as cost-effective as recently funded novel drug therapies in New Zealand and highly cost-effective by international criteria. However, the analysis raises questions about resourcing of statins and laboratory tests and also about GP case loads, especially if the current risk threshold of 10% is maintained. It may also raise equity issues because of differential willingness to accept co-payments on both statins and GP consultations.

ADDITIONAL RESOURCES AND SUPPORTING DOCUMENTS

A list of additional resources and supporting documents available for downloading at www.nzgg.org.nz

- Questions Addressed in the Guideline Development Process
- Guideline Comparisons
- Evidence Tables
- Economic Analysis of Cardiovascular Risk Screening and Lipid Lowering Treatment
- Consumer Resources

ABBREVIATIONS

A2 receptor-blockers	Angiotensin II receptor blockers
ABPM	Ambulatory Blood Pressure Monitoring
ACC	American College of Cardiology
ACE-inhibitor	Angiotensin Converting Enzyme Inhibitor
ACR	Albumin:Creatinine Ratio
ADA	American Diabetes Association
AER	Albumin Excretion Rate
AFPHM	Australasian Faculty of Public Health Medicine
AGREE	Appraisal of Guidelines for Research and Evaluation
AHA	American Heart Association
ALLHAT	Antihypertensive and Lipid-lowering Treatment to Prevent Heart Attack Trial
ALT	Alanine Transaminase
ARR	Absolute Risk Reduction
AST	Aspartate Aminotransferase
BMI	Body Mass Index
BP	Blood Pressure
CABG	Coronary Artery Bypass Grafting
CARE	Cholesterol And Recurrent Events Study
CDC	Centre For Disease Control (America)
CHD	Coronary Heart Disease
CHF	Congestive Heart Failure
CHO	Carbohydrate
CI	Confidence Interval
CK	Creatine Kinase
CRP	C-Reactive Protein
CT	Computed Tomography Scan
CVD	Cardiovascular Disease
DHB	District Health Board
DPP	Diabetes Prevention Program

EBCT	Electron Beam Computerised Tomography
FAO	Food and Agriculture Organisation of the United Nations
FCH	Familial Combined Dyslipidaemia
FDPS	Finish Diabetes Prevention Study
FH	Familial Hypercholesterolaemia
FIELD	Fenofibrate Intervention and Event Lowering in Diabetes Study
FPG	Fasting Plasma Glucose
GATE	Generic Assessment Tool for Epidemiology
GFR	Glomerular Filtration Rate
GGT (γGT)	Gamma Glutamyl Transpeptidase
GI	Glycaemic Index
GP	General Practitioner
HbA1c	Glycosylated (glycated) Haemoglobin
HDL-C	High Density Lipoprotein Cholesterol
HHS	Hospital and Health Services
HOPE	Heart Outcomes Protection Evaluation Study
HPS	Heart Protection Study
HR	Hazard Ratio
Hs-CRP	Highly Sensitive C-reactive Protein
IDL	Intermediate Density Lipoprotein
IGT	Impaired Glucose Tolerance
IFG	Impaired Fasting Glucose
IPA	Independent Practitioner Organisation
ISH	Isolated Systolic Hypertension
LDL-C	Low Density Lipoprotein Cholesterol
LDT	Local Diabetes Team
LIFE	Losartan Intervention for Endpoint Reduction in Hypertension Study
LIPID	Long-term Intervention with Pravastatin in Ischaemic Disease study
LP(a)	Lipoprotein (a)
LV	Left Ventricle
MET	Metabolic Equivalent
MI	Myocardial Infarction
MUFA	Monounsaturated Fatty Acids
NCEP	National Cholesterol Education Program
NHANES	National Health And Nutrition Examination Survey
NHF	National Heart Foundation of New Zealand
NHLBI	National Heart, Lung and Blood Institute

NHMRC	National Health and Medical Research Council (of Australia)
NNT	Number Needed to Treat
NPDR	Non-proliferative Diabetic Retinopathy
NZDEP	New Zealand Small Area Deprivation score
NZGG	New Zealand Guidelines Group
NZHTA	New Zealand Health Technology Assessment
NZSSD	New Zealand Society for the Study of Diabetes
OGTT	Oral Glucose Tolerance Test
OHA	Oral Hypoglycaemic Agents
OR	Odds Ratio
PHO	Primary Health Organisation
PMS	Practice Management System
PTCA	Percutaneous Coronary Angioplasty
PUFA	Polyunsaturated Fatty Acids
PVD	Peripheral Vascular Disease
QALY	Quality Adjusted Life Year
RCGP	Royal College of General Practitioners (UK)
RNZCGP	Royal New Zealand College of General Practitioners
RCT	Randomized Controlled Trial
RR	Relative Risk
SBGM	Self Blood Glucose Monitoring
SFA	Saturated Fatty Acids
SIGN	Scottish Intercollegiate Guidelines Network
SMR	Standardised Mortality Ratio
SPARC	Sport and Recreation New Zealand
TC	Total Cholesterol
TG	Triglyceride
TIA	Transient Ischaemic Attack
UAC	Urinary albumin concentration
UKPDS	United Kingdom Prospective Diabetes Study
VHA	Veterans Health Administration
VLDL	Very Low Density Lipoprotein
VO_2max	Maximum Ventilatory Oxygen Capacity
WHO	World Health Organization
WHR	Waist/ Hip Ratio

GLOSSARY

Abdominal obesity: Accumulation of fat around the abdomen. This form of obesity is most associated with adverse health outcomes.

Absolute risk reduction (ARR): Difference in the absolute risk (rates of adverse events) between intervention and control populations in a research study.

Added sugars: These are the sugars added to foods in production/processing. 'Added' sugars do not refer to sugars naturally occurring in fruits. 'Added' sugars have been defined as 'free sugars' by the WHO/FAO. 'Free sugars' refers to all monosaccharides and disaccharides added to foods by the manufacturer, cook or consumer plus sugars naturally occurring in honey, syrups and fruit juices'.[117]

Adult: A person aged over 18 years.

Angina: Heavienss or tightness in the chest which may spread to the arms, neck, jaw, face or back due to the inadequate blood supply to meet the demands of the heart muscle commonly during effort or emotion and which is eased by rest or use of nitrates.

Antiplatelet agent/drug: Act against or destroy blood platelets. Blood platelets help blood clotting.

Arrhthymia: Abnormal heart rhythm which may be permanent, intermittent or transient.

Atherosclerosis: The condition in which plaques containing cholesterol and other materials form in the inner linings of large and medium-sized arteries leading to localised thickening.

Beta-blocker: A drug which antagonises the effects of the sympathetic stimulation, thereby producing a slower heart rate, lower blood pressure and reduced heart-muscle contraction leading to lessened oxygen demands of the heart muscle and hence decreasing angina pectoris.

Blood Pressure: Pressure exerted on the walls of blood vessels and especially arteries. It is usually measured on the radial artery in the arm using a sphymomanometer and reported as the systolic blood pressure over the diastolic blood pressure eg, 120/80 mm Hg or the systolic blood pressure alone eg, 120 mm Hg.

Systolic Blood Pressure: Maximum blood pressure following contraction of the left ventricle of the heart.

Diastolic Blood Pressure: Minimum blood pressure during filling of the heart with blood.

Body mass index (BMI): An indicator of body fatness. It is calculated from the formula: weight/height squared, where weight is in kilograms and height is in metres.

Calcium: A mineral that is essential for building strong bones and teeth. The most common dietary source is milk and milk products.

Cardiorespiratory fitness: The ability of the circulatory and respiratory systems to supply oxygen during sustained physical activity.

Cardiovascular disease: An all encompassing term used to describe all diseases and conditions involving the heart and blood vessels.

Child: A person aged between 3 and 14 years of age.

Cholesterol: A fat-like steroid found in animal fats, oils, bile, brain tissue, milk, egg yolk, nerve myelin, liver, kidneys and adrenals. Mostly synthesised in the liver, it is important in the synthesis of steroid hormones and bile acids.

Complementary and alternative treatment: A heterogeneous set of practices which are offered as an alternative or an addition to conventional medicine for the preservation of health and the diagnosis and treatment of health related problems.

Congestive heart failure: Heart failure in which the heart is unable to maintain adequate circulation of the blood or to pump out the blood returned to it.

Contraindication: Any factor in a person's condition that makes it unwise to pursue a certain line of treatment.

Coronary heart disease: Heart disease resulting from the atherosclerotic narrowing of a coronary artery/arteries.

District Health Boards (DHBs): Organisations established to protect, promote and improve the health and independence of geographically defined populations. Each DHB will fund, provide (or ensure) the provisions of health and disability services for its population.

Diabetes Type 1: Previously known as IDDM – insulin-dependent diabetes mellitus. Caused by the destruction of insulin-producing cells, resulting in insulin deficiency.

Diabetes Type 2: Previously known as NIDDM – non-insulin-dependent diabetes mellitus. Of unknown cause but associated with a combination of insulin resistance and a relative insulin deficit. The major risk factors for type 2 diabetes are obesity, increasing age, physical inactivity, and nutritional factors such as a high intake of saturated fatty acids. Type 2 diabetes makes up about 85 to 90% of all diabetes in developed countries.

Dietary pattern: This comprises many different foods contributing to a specific combination of interacting nutrients that are consumed together in varying proportions.

Dietary supplements: Food supplements in the form of tablets/liquids or powders that may be consumed in addition to the diet to supplement intakes of vitamins, minerals, herbs or other substances.

Exercise: Exercise is a subset of physical activity and is more formal and exertional in nature. It is planned, structured and repetitive bodily movement done to improve or maintain one or more components of physical fitness. Specific objectives: improving fitness, performance and health, and providing a means of social interaction.

Fitness: see Cardiorespiratory fitness.

Folate/folic acid: A vitamin of the B group essential for cell division, growth and red blood cell formation. The term 'folate' is a generic name for folic acid (pteroyl monoglutamic acid) and related polyglutamate compounds which exhibit the biological activity of folic acid.

Fortified foods: Foods that have had nutrients added, usually vitamins and minerals, during manufacture. The types and amounts of these are specified in food legislation.

Functional foods: Foods similar in appearance to conventional foods but which have been modified to have benefits beyond the provision of simple nutrient requirements.

Glycaemic index (GI): The incremental area under the blood glucose response curve of a 50g carbohydrate portion of a test food, expressed as a percent of the response to the same amount of carbohydrate from a standard food (either white bread or glucose), taken by the same subject.

Hapū: The social and political unit made up of several whānau sharing common descent.

HDL cholesterol (HDL-C): High-density lipoprotein cholesterol. Also known as the good cholesterol, as it has a protective effect against heart disease.

Hillary Commission: The body responsible for sport and recreation in New Zealand; disestablished and reconstituted as Sport and Recreation NZ (SPARC) in 2002.

Inactive: In the context of physical activity, refers to two categories of inactivity. These are: no activity (sedentary) over 7 days; or some activity (relatively inactive) but less than the recommended 30 minutes of moderate-intensity physical activity on most, if not all, days of the week, as specified in the New Zealand Physical Activity Guidelines.

Infants: Children aged less than 12 months.

Ischaemic stroke: Stroke caused by obstruction (as by a blood clot) of a blood vessel of the brain.

Iwi: A social and political unit made up of several hapū sharing common descent; Māori tribe or nation.

Kaumātua: Wise and experienced older members of the whānau, usually aged over 55 years.

LDL cholesterol (LDL-C): Low-density cholesterol. Also known as the bad cholesterol, as a high level in the blood can promote the formation of plaque in the walls of arteries.

Lipids: General term embracing all fats, oils, and waxy substances that are insoluble in water. In medical terms blood lipids refer to triglycerides and cholesterol.

Medication: A substance administered by mouth, applied to the body or introduced into the body for the purpose of treatment.

Meta-analysis: Results from several studies, identified in a systematic review, are combined and summarised quantitatively.

Metabolic equivalent (MET):. Used as an index of the intensity of activities. 1 MET is the resting energy expenditure equivalent to oxygen consumption (3.5ml O_2 per Kg per min).[172] An individual exercising at 2 METs is consuming oxygen at twice the resting rate.

Moderate activity: Moderate-intensity activity is activity that will cause a slight, but noticeable increase in breathing and heart rate. This is equivalent to brisk walking.[172] For adults, moderate activity is activity requiring 3 to 6 times more energy than at rest (3 – 6 METS).

Micronutrients: The essential nutrients, which include vitamins and minerals and are usually required in small quantities.

Morbidity: A diseased state or symptom.

Myocardial infarction: Damage to heart muscle that results typically from the partial or complete blocking of a coronary artery.

Myopathy: A disorder of muscle tissue or muscles.

Number Needed to Treat (NNT): When the treatment reduces the risk of specified adverse outcomes of a condition. NNT is the number of participants with a particular condition who must receive a treatment for a prescribed period in order to prevent the occurrence of the adverse outcomes. This number is the inverse of the absolute risk reduction.

Non-recreational physical activity: Includes active commuting (physical activity as a form of transport), and incidental activity (such as climbing stairs at work, household domestic activity such as washing windows and the car).

Nutrients: Food components essential to support human life.

NZDep: This is an index of deprivation based on the residential address of the individual. The index is based on eight dimensions of deprivation derived from the census: income, access to a car, living space, home ownership, employment, qualifications, support, and access to a telephone. The ten-point scale ranges from 1 (individuals living in the least deprived areas) to 10 (individuals living in the most deprived areas).

People from the Indian subcontinent: People with the self-identified ethnicity codes 43 and 44 according to the New Zealand standard classification of ethnicity.[702]

Peripheral vascular disease: Is clinically defined as a disease of the peripheral blood vessels characterized by narrowing and hardening of the arteries that supply the legs and feet, with resulting decrease in blood flow. For cardiovascular risk assessment, a history of intermittent claudication and reduced foot pulses on examination or radiological evidence of peripheral vascular disease will put the person at high cardiovascular risk.

Personal health services: Services offered on an individual basis. Includes most treatment services, and face to face visits to General Practitioners and other health practitioners.

Physical activity: The entire spectrum of 'bodily movements' that each person can undertake in daily life, ranging from normal active living conditions to 'intentional' moderate physical activities, to structured and repetitive physical exercises, to physical fitness and training sessions, and collective sport activities, especially leisure and recreational sports. It can be analysed in terms of duration, frequency, intensity, type and context.

Prevalence: The number of instances of a given disease or other condition in a population at a designated time. Prevalence includes both new (incident) and existing cases of disease.

Primary health care: Usually the health services of first point of contact, based around key health practitioners or providers such as General Practitioners. Generally community-based, but can include hospitals and other health services. Can also refer to essential health care made available universally to individuals and families in the community, by means acceptable to them.

Public health services: Services offered on a population basis. These include all programmes, interventions, policies and activities that improve and protect the health of individuals and the community. Public health services intervene at the population or group level, as distinct from individual personal health services.

Push Play: A Sport and Recreation New Zealand national social marketing campaign encouraging New Zealanders to do at least 30 minutes of moderate-intensity physical activity on most, if not all, days of the week. Supported regionally and locally by regional sports' trusts and community partners.

Recommended dietary intakes (RDIs): Recommended levels of nutrient intake based on basal, average or low-risk requirements.

Risk factor: An aspect of personal behaviour or lifestyle, an environmental exposure, or an inherited characteristic that is associated with an increased risk of a person developing a disease.

Saturated fat / fatty acids: Fatty acids with no double bonds. Many saturated fats / fatty acids tend to raise levels of blood cholesterol. They are common in animal fats, coconut oil and palm oil.

Secondary health care: Specialist care that is typically provided in a hospital setting.

Sedentary: No physical activity in the past 7 days.

Serum C peptide: The principal use of C-peptide is in the evaluation of hypoglycemia. People with insulin-secreting neoplasms have high levels of both C-peptide and endogenous insulin; in contrast, people with factitious hypoglycemia will have low C-peptide levels in the presence of elevated (exogenous) serum insulin. C-peptide is also useful in evaluating residual beta-cell function in insulin-dependent diabetics, many of whom have antibodies that interfere with insulin assays. Glucagon-stimulated C-peptide concentration has been shown to be a good discriminator between insulin-requiring and noninsulin-requiring diabetic people. The diagnosis of islet cell tumor is supported by elevation of C-peptide when plasma glucose is less than or equal to 40 mg/dL.

Sport and Recreation NZ (SPARC): The organisational body responsible for sport and recreation in New Zealand.

Stroke: Sudden decrease or loss of consciousness, sensation, and movement caused by rupture or obstruction (as by a blood clot) of a blood vessel of the brain.

Takeaways: Foods that are purchased in a ready-to-eat form. They tend to be high in fat and salt. Examples include fish and chips, hamburgers, fried chicken and chips, pizzas, and Chinese takeaways.

Tertiary health care: Very specialised care, often only provided in a small number of locations.

Te reo: Language (usually used for Māori language).

Transient ischaemic attack: A brief episode of reduced blood flow to the brain. Temporary blurring of vision, slurring of speech, numbness, and muscle weakness are common features.

Treaty of Waitangi: New Zealand's founding document, which establishes the relationship between the Crown and Māori as tangata whenua, and requires both the Crown and Māori to act reasonably towards each other and with utmost good faith.

Treatment: Treatment includes reference to lifestyle interventions and drug therapy.

Toddlers: Infants aged from 1 to 2 years of age.

Triglycerides: A form of fat in food and the body consisting of glycerol plus three fatty acids. A high level of blood triglycerides is a risk factor for heart disease and stroke and is usually raised by being overweight and by excess alcohol.

Vigorous activity: The New Zealand Physical Activity Guidelines define vigorous activity as activity that makes people breathe hard or 'puff'. For adults, it is activity requiring seven times as much energy as at rest, or greater; equivalent to jogging (7 METS).[172]

Weight management: Includes the approaches outlined below.

1. Primary prevention of weight gain in adults, particularly in those with a family history of obesity-related disorders or with early risk factors of a developing metabolic syndrome.
2. Weight loss programmes for the overweight.
3. Weight maintenance: see definition below.
4. Management of interacting risk factors: smoking, physical inactivity, inappropriate diets, eg, in relation to sodium intake and hypertension, saturated fatty acids and ischaemic heart disease, and a low fruit and vegetable consumption (ie, less than 5 portions per day).
5. Control of weight gain in growing children who are identified as overweight.

Weight maintenance: Secondary prevention of weight gain after completing a weight loss programme. This is considered to be long-term, ie, greater than 2-year maintenance of body weight achieved following a period of weight loss (usually a 3-month weight loss programme).

Whānau: Relationships that have blood links to a common ancestor; extended family.

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