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NUTS AND HEART HEALTH

This paper outlines the evidence for the Heart Foundation of New Zealand’s position on the relationship of nuts to heart health
# Table of Contents

List of Tables ........................................................................................................ ii  
List of Appendices .............................................................................................. iii  
List of Abbreviations .......................................................................................... iv  

Section 1: Introduction ....................................................................................... 1  
Section 2: Nutrient composition of nuts............................................................ 6  
Section 3: Nut consumption and lipid-mediated risk factors for CVD .............. 7  
Section 3.1 Epidemiological studies: Heart disease and mortality ................. 10  
Section 3.2 Intervention trials: Lipids, lipoproteins, and apolipoproteins ....... 10  
Section 3.2.1 Lipid profile .................................................................................. 10  
Section 3.2.2 Apolipoprotein profile ............................................................... 12  
Section 4: Nut consumption and blood pressure ............................................. 12  
Section 4.1 Epidemiological studies: Hypertension, stroke, and atrial fibrillation ............................................................................................................ 12  
Section 4.2 Intervention trials: Blood pressure .............................................. 13  
Section 5: Nut consumption and novel biomarkers of CVD ......................... 13  
Section 5.1 Epidemiological studies: Novel risk factors .................................. 13  
Section 5.2 Intervention trials: Novel risk factors .......................................... 14  
Section 5.2.1 The role of antioxidants in oxidative stress .............................. 14  
Section 5.2.2 Inflammation ............................................................................. 14  
Section 5.2.3 Endothelial function .................................................................. 15  
Section 6: Nut consumption and risk of diabetes .......................................... 15  
Section 6.1 Epidemiological studies: Type 2 diabetes ..................................... 15  
Section 6.2 Intervention trials: Insulin resistance and glycaemic control ....... 16  
Section 7: Nut consumption and body weight ............................................... 17  
Section 7.1 Epidemiological studies: Body weight .......................................... 17  
Section 7.2 Intervention trials: Body weight .................................................. 17  
Section 8: Mechanisms for beneficial effects ................................................ 18  
Section 8.1 Mechanisms for biochemical indices ......................................... 18  
Section 8.1.1 Fatty acid profile ...................................................................... 18  
Section 8.1.2 Lysine to arginine ratio .............................................................. 19  
Section 8.1.3 Dietary fibre ............................................................................ 19  
Section 8.1.4 Phytochemicals ....................................................................... 19  
Section 8.1.5 Antioxidants ......................................................................... 19  
Section 8.1.6 Micronutrients ....................................................................... 19  
Section 8.2: Mechanisms for less than predicted weight gain ....................... 20  
Section 8.2.1 Dietary compensation .............................................................. 20  
Section 8.2.2 Energy malabsorption .............................................................. 20  
Section 8.2.3 Metabolic rate ......................................................................... 20  
Section 9: Sensory factors that may affect nut consumption ....................... 21  
Section 10: Safety of nut consumption .......................................................... 21  
Section 11: Summary of evidence: Health benefits of nut consumption ......... 23  
Section 12: New Zealand context .................................................................... 25  
Section 13: Recommendations ...................................................................... 26  
Section 14: References .................................................................................... 28
LIST OF TABLES

Table 1. Level of evidence ................................................................. 3

Table 2. Evidence table for dietary intervention trials that have examined
the effects of chronic nut consumption on CVD risk factors or acceptance 4

Table 3. Average macronutrient composition for 100 g and one serving
(30g) of raw nuts .................................................................................. 8

Table 4. Average phytosterol and micronutrient composition for 100 g and
one serving (30 g) of raw nuts ................................................................ 9

Table 5. Summary of scientific evidence: effects of nut consumption on
diseases and risk factors .................................................................... 28
Appendix A. Epidemiological studies investigating the effects of nut consumption on risk of cardiovascular disease and mortality (4 cohorts)

Appendix B. Dietary intervention trials investigating the effects of nut consumption on blood lipids and lipoproteins ($n = 46$)

Appendix C. Dietary intervention trials investigating the effects of nut consumption on apolipoprotein A1 and B ($n = 19$)

Appendix D. Epidemiological studies investigating the effects of nut consumption on the risk of hypertension, stroke, and atrial fibrillation (2 cohorts)

Appendix E. Dietary intervention trials investigating the effects of nut consumption on diastolic and systolic blood pressure ($n = 20$)

Appendix F. Epidemiological studies investigating the effects of nut consumption on novel risk factors for CVD (5 populations)

Appendix G. Dietary intervention trials investigating the effects of nut consumption on antioxidant activity and biomarkers of antioxidant status ($n = 19$)

Appendix H. Dietary intervention trials investigating the effects of nut consumption on biomarkers of oxidative stress ($n = 16$)

Appendix I. Dietary intervention trials investigating the effects of nut consumption on biomarkers of inflammation ($n = 11$)

Appendix J. Dietary intervention trials investigating the effects of nut consumption on biomarkers of endothelial function ($n = 9$)

Appendix K. Epidemiological studies investigating the effects of nut consumption on type 2 diabetes (4 cohorts)

Appendix L. Dietary intervention trials investigating the effects of nut consumption on markers of glycaemic control ($n = 19$)

Appendix M. Cross-sectional studies examining the association between baseline nut consumption and BMI (7 populations)

Appendix N. Epidemiological studies investigating the effects of nut consumption on weight gain and obesity (3 cohorts)

Appendix O. Dietary intervention trials investigating the effects of nut consumption on body weight ($n = 43$)

Appendix P. Dietary intervention trials investigating the effects of nut consumption on body weight as a primary outcome ($n = 5$)

Appendix Q. Dietary intervention trials investigating the effects of nut consumption on acceptance ($n = 4$)
**List of Abbreviations**

<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>AHS</td>
<td>Adventist Health Study</td>
</tr>
<tr>
<td>ALA</td>
<td>Alpha linolenic acid</td>
</tr>
<tr>
<td>apo</td>
<td>Apolipoprotein</td>
</tr>
<tr>
<td>CHD</td>
<td>Coronary heart disease</td>
</tr>
<tr>
<td>CVD</td>
<td>Cardiovascular disease</td>
</tr>
<tr>
<td>EPIC</td>
<td>European Prospective Investigation into Cancer and Nutrition</td>
</tr>
<tr>
<td>FFQ</td>
<td>Food frequency questionnaire</td>
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<tr>
<td>HDL-C</td>
<td>High-density lipoprotein cholesterol</td>
</tr>
<tr>
<td>IHD</td>
<td>Ischaemic heart disease</td>
</tr>
<tr>
<td>IWHS</td>
<td>Iowa Women’s Health Study</td>
</tr>
<tr>
<td>LDL-C</td>
<td>Low-density lipoprotein cholesterol</td>
</tr>
<tr>
<td>MI</td>
<td>Myocardial infarction</td>
</tr>
<tr>
<td>MUFA</td>
<td>Monounsaturated fatty acids</td>
</tr>
<tr>
<td>NHANES</td>
<td>National Health and Nutrition Examination Survey</td>
</tr>
<tr>
<td>NHS</td>
<td>Nurses’ Health Study</td>
</tr>
<tr>
<td>NZ</td>
<td>New Zealand</td>
</tr>
<tr>
<td>PHS</td>
<td>Physicians’ Health Study</td>
</tr>
<tr>
<td>PREDIMED</td>
<td>Prevencion con Dieta Mediterranea</td>
</tr>
<tr>
<td>PUFA</td>
<td>Polyunsaturated fatty acids</td>
</tr>
<tr>
<td>RCT</td>
<td>Randomised controlled trial</td>
</tr>
<tr>
<td>RDI</td>
<td>Recommended dietary intake</td>
</tr>
<tr>
<td>RMR</td>
<td>Resting metabolic rate</td>
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<tr>
<td>SFA</td>
<td>Saturated fatty acids</td>
</tr>
<tr>
<td>SUN</td>
<td>Seguimiento Universidad de Navarra</td>
</tr>
<tr>
<td>TAG</td>
<td>Triacylglycerol</td>
</tr>
<tr>
<td>TC</td>
<td>Total cholesterol</td>
</tr>
<tr>
<td>US</td>
<td>United States</td>
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Section 1: Introduction

Cardiovascular disease (CVD) is the generic term that describes a group of disorders of the heart and blood vessels. It contributes to a third of all global deaths. It poses a large global economic burden, costing billions of dollars annually for healthcare, productivity losses, and informal care. The World Health Organisation has identified numerous risk factors for CVD, which include tobacco use, physical inactivity, harmful use of alcohol, unhealthy diet, obesity, abnormal blood lipids, high blood pressure, and diabetes. More recently, evidence suggests that novel risk factors such as oxidative stress, chronic inflammation, and endothelial dysfunction also play a key role in determining an individual’s cardiovascular risk.

Nuts are rich sources of cis-unsaturated fatty acids, vegetable protein, dietary fibre, phytochemicals, antioxidants, vitamins and minerals, which could act synergistically to reduce CVD risk. The primary mechanism is likely to be the significant reductions in total cholesterol (TC) and low-density lipoprotein cholesterol (LDL-C) concentrations. Improvements in novel risk factors namely oxidative stress, inflammation, and endothelial dysfunction may also in part explain the observed reduction in CVD risk.

Objectives

The objective of this position statement is to provide an evidence-based summary of the effects of regular nut consumption on risk factors for cardiovascular disease.

Overview

This position statement focuses first on the nutrient composition of nuts. Secondly, an overview of major epidemiological studies and clinical trials, which examine the relationship between nut consumption and potentially reversible risk factors for CVD, namely blood lipids, blood pressure, oxidative stress, inflammation, and endothelial dysfunction, diabetes, and body weight. The potential mechanisms for the health benefits gained from nut consumption will be reviewed in Section 8. The sensory factors that may affect compliance to the guideline to consume nuts regularly will be discussed. A brief section on safety of nuts will be summarised. A summary of the strength of evidence regarding nut consumption in relation to CVD and risk factors will then be reported. Finally, nut consumption in the New Zealand (NZ) context and recommendations regarding nut consumption will be outlined.
Clinical trials (1-), epidemiological studies (2-), and cross-sectional studies (3) are deemed to contain a high risk of bias, therefore these studies were not considered when conclusions were made. The studies included in this review had to be an original article, written in English, and published in a scientific journal, between 1 January 1990 and 1 August 2012.

In total, 98 clinical trials that had investigated the effects of chronic nut consumption on at least one CVD risk factor or acceptance of nuts were identified (Table 2). However, only 62 of them met the inclusion criteria; 26 controlled feeding RCTs were given 1++; 36 RCTs conducted under free-living situations were given 1+; and finally, 36 clinical trials were given 1- and the main reasons were: single intervention design (n = 9); reported same risk factors in different publications (n = 2); or lack of randomisation (n = 12), control group (n = 9) or between-group analysis for primary outcome (n = 4). The characteristics and findings of 1++ and 1+ interventions can be found in the Appendices.
Table 2: Level of evidence

<table>
<thead>
<tr>
<th>Level of evidence</th>
<th>Description</th>
<th>Examples</th>
</tr>
</thead>
<tbody>
<tr>
<td>1+++</td>
<td>High quality randomised controlled trials with a very low risk of bias.</td>
<td>Randomised controlled trials conducted under controlled feeding conditions</td>
</tr>
<tr>
<td>1+</td>
<td>Well-conducted randomised controlled trials with a low risk of bias.</td>
<td>Randomised controlled trials conducted under free-living conditions</td>
</tr>
<tr>
<td>2+++</td>
<td>High quality cohort studies with a very low risk of confounding or bias and a high probability that the relationship is causal.</td>
<td>Cohort studies with multiple adjustments, multiple dietary assessment periods, dietary assessment method with documented validity, and follow-up ≥ 5 years. All or most of the criteria have been fulfilled.</td>
</tr>
<tr>
<td>2+</td>
<td>Well-conducted cohort studies with a low risk of confounding or bias and a moderate probability that the relationship is causal.</td>
<td>Cohort studies with multiple adjustments, multiple dietary assessment periods, dietary assessment method with documented validity, and follow-up ≥ 5 years. Some of the criteria have been fulfilled.</td>
</tr>
<tr>
<td>1-</td>
<td>Clinical trials with a high risk of bias. These studies were not considered when conclusions or recommendations were made.</td>
<td>Single intervention. Clinical trials without randomisation (i.e. sequential design), control group, or between-group analysis. Clinical trials that have reported the same risk factors in different publications.</td>
</tr>
<tr>
<td>2-</td>
<td>Cohort studies with a high risk of confounding or bias and a significant risk that the relationship is not causal. These studies were not considered when conclusions or recommendations were made.</td>
<td>Cohort studies with multiple adjustments, multiple dietary assessment periods, dietary assessment method with documented validity, and follow-up ≥ 5 years. Few or no criteria fulfilled.</td>
</tr>
<tr>
<td>3</td>
<td>Cross-sectional studies. These studies were not considered when conclusions or recommendations were made.</td>
<td>Both exposure and outcome were measured at the same point in time.</td>
</tr>
</tbody>
</table>

Adapted from New Zealand Guidelines Group\(^{(69)}\)
### Table 2. Evidence table for dietary intervention trials that have examined the effects of chronic nut consumption on CVD risk factors or acceptance of nuts

<table>
<thead>
<tr>
<th>Nuts</th>
<th>Evidence 1++</th>
<th>Evidence 1+</th>
<th>Evidence 1-</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Brazil nuts</strong> (n=2)</td>
<td>Davis et al. (2007) [38]</td>
<td>Tey et al. (2011) [39]</td>
<td>Jalali-Khanabadi et al. (2010) [40]</td>
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<tr>
<td></td>
<td>Schutte et al. (2006) [47]</td>
<td></td>
<td>Koegali et al. (2011) [48]</td>
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<tr>
<td><strong>Hazelnuts</strong> (n=8)</td>
<td>Tey et al. (2011) [49]</td>
<td>Alphan et al. (1997) [50]</td>
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<tr>
<td></td>
<td>Tey et al. (2012) [51]</td>
<td>Durak et al. (1999) [52]</td>
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<td></td>
<td></td>
<td>Mercanligil et al. (2007) [53]</td>
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<td></td>
<td></td>
<td>Yucesan et al. (2010) [54]</td>
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<td></td>
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<td>Tey et al. (2011) [55]</td>
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<td></td>
<td></td>
<td>Tey et al. (2011) [56]</td>
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<tr>
<td><strong>Macadamias</strong> (n=6)</td>
<td>Curb et al. (2000) [57]</td>
<td>Colquhoun et al. (1996) [58]</td>
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<td></td>
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<td>Hiroaka-Yamamoto et al. (2004) [61]</td>
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<td></td>
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<td>Garg et al. (2007) [62]</td>
</tr>
<tr>
<td><strong>Peanuts</strong> (n=8)</td>
<td>Kris-Etherton et al. (1999) [63]</td>
<td>Claesson et al. (2009) [64]</td>
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<td></td>
<td></td>
<td></td>
<td>Alper &amp; Mattes (2002) [68]</td>
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<td></td>
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<td></td>
<td>Alper &amp; Mattes (2003) [69]</td>
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<td></td>
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<td></td>
<td>McKiernan et al. (2010) [70]</td>
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<tr>
<td></td>
<td>Haddad et al. (2006) [73]</td>
<td>Eastman &amp; Clayshulte (2005) [74]</td>
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<tr>
<td><strong>Pistachios</strong> (n=11)</td>
<td>Gebauer et al. (2008) [75]</td>
<td>Kocygig et al. (2006) [76]</td>
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<td></td>
<td>Kay et al. (2010) [77]</td>
<td>Sheridan et al. (2007) [78]</td>
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<td></td>
<td>Baer et al. (2012) [79]</td>
<td>Li et al. (2010) [80]</td>
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<tr>
<td></td>
<td></td>
<td>Wang et al. (2012) [81]</td>
<td></td>
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<tr>
<td><strong>Walnuts</strong> (n=30)</td>
<td>Sabate et al. (1993) [82]</td>
<td>Chisholm et al. (1998) [83]</td>
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<td></td>
<td>Zambon et al. (2000) [84]</td>
<td>Ros et al. (2004) [85]</td>
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<td></td>
<td>Iwamoto et al. (2002) [86]</td>
<td>Tapsell et al. (2004) [87]</td>
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<tr>
<td></td>
<td>Zhao et al. (2004) [88]</td>
<td>Sabate et al. (2005) [89]</td>
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<td></td>
<td>Davis et al. (2007) [90]</td>
<td>Spaccarotella et al. (2008) [91]</td>
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<td></td>
<td>Mukuddem-Petersen et al. (2007) [92]</td>
<td>Tapsell et al. (2009) [93]</td>
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<tr>
<td></td>
<td>Perez-Martinez et al. (2007) [94]</td>
<td>Ma et al. (2010) [95]</td>
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<tr>
<td></td>
<td>Zhao et al. (2007) [96]</td>
<td>Torabian et al. (2010) [97]</td>
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<td>Rajaram et al. (2009) [98]</td>
<td>Wu et al. (2010) [99]</td>
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<td></td>
<td>West et al. (2010) [100]</td>
<td>Damasceno et al. (2011) [101]</td>
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<tr>
<td></td>
<td></td>
<td>Din et al. (2011) [102]</td>
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</tbody>
</table>

**Evidence ratings:**
- **Evidence 1++:** Strong evidence
- **Evidence 1+:** Moderate evidence
- **Evidence 1-:** Limited evidence
Mixed nuts
(n=4)

Chisholm et al. (2005)
Lopez-Uriarte et al. (2010)
Casas-Agustench et al. (2011)
Jenkins et al. (2011)

Total (n=98)

<table>
<thead>
<tr>
<th></th>
<th>Evidence 1++: n = 26</th>
<th>Evidence 1+: n = 36</th>
<th>Evidence 1-: n = 36</th>
</tr>
</thead>
</table>

1 Studies in italic font have compared the effects of two different types of nuts (n = 7).

2 Reasons for being assessed as 1-: a Single intervention; b No randomisation; c No control group; d Reported the same risk factors in different publications; e No between-group analysis for primary outcome.
Section 2: Nutrient composition of nuts

A tree nut is defined as a one-seeded dried fruit or an edible kernel surrounded by a hard shell \[\text{(116)}\]. These include almonds, Brazil nuts, cashews, hazelnuts, pecans, pine nuts, pistachios, macadamias, and walnuts. Most consumers also perceive peanuts as nuts, although they are botanically classed as a legume \[\text{(11)}\]. For this position statement, the term “nuts” includes peanuts and all tree nuts, except for chestnuts and coconuts. This is because peanuts have a very similar nutrient profile as all the other tree nuts whereas chestnuts and coconuts differ from tree nuts in several ways. Chestnuts are relatively high in carbohydrate (53.2 g per 100 g) and coconuts are high in saturated fatty acids (SFA) (29.7 g per 100 g). In addition, these two nuts contain high amounts of moisture (49 g per 100 g) and very little protein (< 7 g per 100 g). These attributes make their nutrient profiles different from the other aforementioned nuts \[\text{(11,117)}\].

Macronutrients and selected micronutrients in 100 g and one serving (30 g) of nuts obtained from food composition data are presented in Table 3 and Table 4, respectively. Nuts can be considered as one of the most nutritionally dense foods. Most nuts, although relatively high in fat, have a high content of cis-unsaturated fatty acids whereas the SFA content is relatively low (4 to 17%). Together, monounsaturated fatty acids (MUFA) and polyunsaturated fatty acids (PUFA) in nuts contribute around 70 to 87% of the energy from fat (Table 3). Almost one-half of the total fat content of most nuts such as almonds, cashew nuts, hazelnuts, macadamia nuts, peanuts, pecan nuts, and pistachio nuts is MUFA whereas walnuts contain mostly PUFA. Similar proportions of MUFA and PUFA are found in Brazil nuts and pine nuts \[\text{(14)}\]. In addition, nuts are high in protein and dietary fibre, where 100 g of nuts can provide up to 26 g of protein and 8 g of fibre \[\text{(118,119)}\].

Nuts are important sources of an array of phytochemicals including phenolics such as flavonoids, phenolic acids, and tannins, as well as lipids including carotenoids and phytosterols \[\text{(120,121)}\]. The phytosterols in tree nuts range from 72 to 214 mg per 100 g (Table 4). In addition, nuts especially almonds and hazelnuts have an exceptionally high content of α-tocopherol (5 to 8 mg per serving) whereas pecans and walnuts are high in γ-tocopherol (7 to 9 mg per serving) \[\text{(122-124)}\]. Nuts are low in sodium while rich in a wide range of essential micronutrients such as folate, calcium, magnesium, copper, and potassium, which are required in very small amounts yet perform a vital role in maintaining overall good health \[\text{(117,125,126)}\].

Nut consumption has been consistently reported to improve nutrient intakes and diet quality \[\text{(127-129)}\]. Clearly, a realistic daily quantity of nuts cannot provide sufficient amounts of arginine, dietary fibre, phytochemicals, vitamin E, and the essential micronutrients to exert individual cholesterol-lowering or anti-inflammatory effects \[\text{(11,12)}\]. Nevertheless, multiple small effects on top of the favourable fatty acid...
composition could be beneficial for both prevention and treatment of CVD (6,17,130,131).

SECTION 3: NUT CONSUMPTION AND LIPID-MEDIATED RISK FACTORS FOR CVD

Both epidemiological studies and clinical trials have shown that regular nut consumption plays an important protective role in the management of plasma lipids, CVD morbidity, and coronary heart disease (CHD) mortality (132,133). This section will firstly summarise the findings of four large epidemiological studies on frequent nut consumption and CVD risk (Section 3.1). Secondly, it will review clinical trials that have been conducted to examine the role of nuts in improving lipids and lipoprotein mediated risk factors for CVD such as blood lipids and lipoproteins (Section 3.2.1) as well as the apolipoprotein (apo) profile (Section 3.2.2).
Table 3: Average macronutrient composition for 100 g and one serving (30 g) of raw nuts

<table>
<thead>
<tr>
<th></th>
<th>Energy (kJ)</th>
<th>Total fat (g)</th>
<th>SFA (g)</th>
<th>MUFA (g)</th>
<th>PUFA (g)</th>
<th>Protein (g)</th>
<th>CHO (g)</th>
<th>Fibre (g)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>100 g</td>
<td>30 g</td>
<td>100 g</td>
<td>30 g</td>
<td>100 g</td>
<td>30 g</td>
<td>100 g</td>
<td>30 g</td>
</tr>
<tr>
<td>Almonds</td>
<td>2550</td>
<td>765</td>
<td>55.8</td>
<td>16.7</td>
<td>4.4</td>
<td>1.3</td>
<td>38.0</td>
<td>11.4</td>
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<tr>
<td>Brazil nuts</td>
<td>2830</td>
<td>849</td>
<td>68.2</td>
<td>20.5</td>
<td>17.4</td>
<td>5.2</td>
<td>22.4</td>
<td>6.7</td>
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<td>Cashews</td>
<td>2440</td>
<td>732</td>
<td>49.2</td>
<td>14.8</td>
<td>8.4</td>
<td>2.5</td>
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<td>9.3</td>
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<td>Hazelnuts</td>
<td>2620</td>
<td>786</td>
<td>59.8</td>
<td>17.9</td>
<td>5.7</td>
<td>1.7</td>
<td>42.4</td>
<td>12.7</td>
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<td>Macadamias</td>
<td>2990</td>
<td>897</td>
<td>73.7</td>
<td>22.1</td>
<td>11.0</td>
<td>3.3</td>
<td>58.2</td>
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<tr>
<td>Mixed nuts</td>
<td>2520</td>
<td>756</td>
<td>52.5</td>
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<td>Peanuts</td>
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<td>Pecan nuts</td>
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<td>67.6</td>
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<td>5.4</td>
<td>1.6</td>
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<tr>
<td>Pine nuts</td>
<td>2520</td>
<td>756</td>
<td>50.7</td>
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<td>7.8</td>
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<td>19.2</td>
<td>5.8</td>
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<tr>
<td>Pistachios</td>
<td>2610</td>
<td>783</td>
<td>54.4</td>
<td>16.3</td>
<td>6.9</td>
<td>2.1</td>
<td>36.8</td>
<td>11.0</td>
</tr>
<tr>
<td>Walnuts</td>
<td>2930</td>
<td>879</td>
<td>64.5</td>
<td>19.4</td>
<td>6.5</td>
<td>2.0</td>
<td>12.4</td>
<td>3.7</td>
</tr>
</tbody>
</table>

*Source: The Concise New Zealand Food Composition Tables, 8th Edition. Abbreviations used: CHO, carbohydrate; MUFA, monounsaturated fatty acids; PUFA, polyunsaturated fatty acids; SFA, saturated fatty acids.*
Table 4: Average phytosterol and micronutrient composition for 100 g and one serving (30 g) of raw nuts

<table>
<thead>
<tr>
<th></th>
<th>Phytosterol (mg)</th>
<th>Vitamin E (mg)</th>
<th>Folate (mcg)</th>
<th>Calcium (mg)</th>
<th>Magnesium (mg)</th>
<th>Copper (mg)</th>
<th>Potassium (mg)</th>
<th>Sodium (mg)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>100 g</td>
<td>30 g</td>
<td>100 g</td>
<td>30 g</td>
<td>100 g</td>
<td>30 g</td>
<td>100 g</td>
<td>30 g</td>
</tr>
<tr>
<td>Almonds</td>
<td>141</td>
<td>43</td>
<td>26.2</td>
<td>7.9</td>
<td>96</td>
<td>29</td>
<td>250</td>
<td>75</td>
</tr>
<tr>
<td>Brazil nuts</td>
<td>N/A</td>
<td>N/A</td>
<td>5.7</td>
<td>1.7</td>
<td>4</td>
<td>1</td>
<td>180</td>
<td>54</td>
</tr>
<tr>
<td>Cashews</td>
<td>N/A</td>
<td>N/A</td>
<td>0.9</td>
<td>0.3</td>
<td>67</td>
<td>20</td>
<td>34</td>
<td>10</td>
</tr>
<tr>
<td>Hazelnuts</td>
<td>96</td>
<td>29</td>
<td>15.0</td>
<td>4.5</td>
<td>116</td>
<td>35</td>
<td>179</td>
<td>54</td>
</tr>
<tr>
<td>Macadamias</td>
<td>116</td>
<td>35</td>
<td>0.5</td>
<td>0.2</td>
<td>16</td>
<td>5</td>
<td>70</td>
<td>21</td>
</tr>
<tr>
<td>Mixed nuts</td>
<td>N/A</td>
<td>N/A</td>
<td>N/A</td>
<td>N/A</td>
<td>N/A</td>
<td>N/A</td>
<td>N/A</td>
<td>N/A</td>
</tr>
<tr>
<td>Peanuts</td>
<td>N/A</td>
<td>N/A</td>
<td>N/A</td>
<td>N/A</td>
<td>N/A</td>
<td>110</td>
<td>33</td>
<td>61</td>
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<td>Pecan nuts</td>
<td>102</td>
<td>31</td>
<td>1.4</td>
<td>0.4</td>
<td>39</td>
<td>12</td>
<td>36</td>
<td>11</td>
</tr>
<tr>
<td>Pine nuts</td>
<td>141</td>
<td>42</td>
<td>9.3</td>
<td>2.8</td>
<td>54</td>
<td>16</td>
<td>26</td>
<td>8</td>
</tr>
<tr>
<td>Pistachios</td>
<td>214</td>
<td>64</td>
<td>2.3</td>
<td>0.7</td>
<td>58</td>
<td>17</td>
<td>135</td>
<td>41</td>
</tr>
<tr>
<td>Walnuts</td>
<td>72</td>
<td>22</td>
<td>0.7</td>
<td>0.2</td>
<td>83</td>
<td>25</td>
<td>129</td>
<td>39</td>
</tr>
</tbody>
</table>

1 Source: The Concise New Zealand Food Composition Tables, 8th Edition (34) unless otherwise indicated. Abbreviations used: N/A, not available; T, trace value.
3 Phytosterol is calculated as the total amount of stigmasterol, campesterol and beta-sitosterol.
4 Vitamin E refers to the α-tocopherol content.
**SECTION 3.1 EPIDEMIOLOGICAL STUDIES: HEART DISEASE AND MORTALITY**

Four large epidemiological studies conducted in the United States (US) have examined the associations between nut consumption and CHD risk. These studies include the Adventist Health Study (AHS) \(^{136-138}\), the Iowa Women’s Health Study (IWHS) \(^{139-142}\), the Nurses’ Health Study (NHS) \(^{143,144}\), and the Physicians’ Health Study (PHS) \(^{145,146}\), as shown in Appendix A. A food frequency questionnaire (FFQ) was used to assess nut consumption; with categories ranging from ‘never consume’ to ‘consume more than five times per week’, where one time is equivalent to one serving (1 ounce = 28.35 grams). The NHS had been given a 2++ as nut consumption was measured biennially and the cumulative mean consumption of nuts during follow-up was used. The other cohort studies (2+) only measured baseline nut consumption.

Results from these four large US epidemiological studies are remarkably consistent. An inverse, dose-response relationship was found between nut intakes and several clinical outcomes, such as CHD, sudden cardiac death, and all-cause mortality in different population groups after six to 22 years of follow-up period \(^{11,133}\). A pooled analysis of these four cohorts published up until the year 2006 showed that participants who had the highest nut intake had a 34% (95% CI: 23%, 44%) reduction in risk of CHD incidence and 35% (95%: 11%, 53%) reduction in risk of total CHD \(^{131}\). A similar beneficial effect was reported in the European Prospective Investigation into Cancer and Nutrition (EPIC) study among 399,633 Europeans, where participants with the highest nut intake had a 29% (95% CI: 2%, 49%) reduction in the risk of CHD mortality compared to those who had the lowest nut intake \(^{147}\). For an increase of one serving (~30g) of nut consumption weekly, there was an average 8.3% reduction in the risk of CHD death \(^{133}\). Similar reductions were seen for the risks of sudden cardiac death and all-cause mortality \(^{148}\).

Over all, the results suggest that the cardioprotective effect of nut consumption in relation to CHD are independent of recognised coronary risk factors, such as participants’ age (from young adults to the oldest-old population), sex (men or women), race (non Hispanic white or Black American), hypertension, smoking status, relative weight and physical activity level, alcohol use, and other nutritional characteristics \(^{130,148-151}\).

**SECTION 3.2 INTERVENTION TRIALS: LIPIDS, LIPOPROTEINS, AND APOLIPOPROTEINS**

**SECTION 3.2.1 LIPID PROFILE**

Forty-six out of 79 clinical trials which have investigated the effects of nut consumption on blood lipids and lipoproteins met the criteria outlined in Section 1 (Appendix B). In line with the findings from epidemiological studies, four RCTs using almonds \(^{20,27}\), pistachios \(^{78}\), and mixed nuts \(^{115}\) have shown that TC and LDL-C concentrations were lowered in a dose-dependent manner. The reductions in TC and LDL-C were most pronounced...
in participants who consumed high-dose nut-enriched diets, followed by low-dose nut-enriched diets. These studies also reported that the incorporation of nuts into a Step I diet, Step II diet or a diet recommended by the National Cholesterol Education Program Adult Treatment Panel III and the American Diabetes Association produced further improvements in lipid profiles, compared with the recommended control diets without nuts.

Previous studies have suggested that the cell wall of intact nuts may limit the release of lipids and other nutrients available for digestion, and thus different forms of nuts may have different biological consequences in health outcomes. Five RCTs have compared the effects of different forms of nuts on blood lipids and lipoproteins. Two studies suggested the lipid-lowering effects of all processed almonds (dry roasted whole almonds, almond oil, roasted salted almonds, roasted almond butter, raw almonds) are comparable, which can be translated into a similar reduction in CVD risk. Similarly, a well-controlled feeding trial has shown that compared with the average American diet, peanuts/peanut butter and peanut oil diets significantly reduced TC, LDL-C, and triacylglycerol (TAG) concentrations. Two recent studies reported no significant differences in blood lipids between different forms of peanuts (raw, roasted unsalted, roasted salted, honey roasted, and peanut butter) and hazelnuts (ground, sliced, and whole). However, compared with baseline, all forms of nuts significantly lowered TC and LDL-C whereas high-density lipoprotein cholesterol (HDL-C) increased significantly at the end of the study in participants who had elevated plasma lipids. The results of all five studies above demonstrate that the ingestion of different forms of nuts reduce TC and LDL-C concentrations significantly, accompanied by a favourable change in HDL-C and TAG concentrations, when compared with a baseline diet and typical American diet in both free living and well-controlled settings.

A recent pooled analysis of 25 intervention trials investigated the effects of consumption of different types of nuts on blood lipids in 583 participants. Participants with high baseline LDL-C (> 3.37 mmol/L) and lower BMI (< 25 kg/m²) showed a greater improvement in blood lipids with nut consumption and this effect was independent of the nut type consumed. The pooled analysis estimated an average reduction of 0.28 mmol/L (5.1 %) in TC and 0.26 mmol/L (7.4 %) in LDL-C, while no significant effects were found on HDL-C and TAG with consumption of 67 g of nuts daily. Consistent with this finding, a meta-analysis of five RCTs with almonds and a meta-analysis of 13 feeding studies with walnuts estimated mean reductions of TC (0.18 to 0.27 mmol/L) and LDL-C (0.15 to 0.24 mmol/L) following a nut-enriched diet (30 to 108 g/d), without adversely affecting HDL-C and TAG compared to a control diet or participants’ baseline diet.

Although the clinical trials presented in Appendix B differ in the degree of dietary control, participants’ characteristics, sample size, duration of the intervention, as well as the amount or type of nuts consumed, collectively
the trials have convincingly found that the inclusion of a variety of nuts for periods of three to twelve weeks results in significant reductions in TC and LDL-C and have minor effects, if any, on HDL-C and TAG in the study participants. The cholesterol-lowering effect of nut consumption was more pronounced in participants with elevated blood lipids and lower BMI.

**Section 3.2.2 Apolipoprotein Profile**

Nineteen of 31 studies investigating the effects of nuts on apolipoproteins concentrations met the inclusion criteria (Appendix C). The majority of studies have reported that inclusion of nuts in the diet does not affect apo A1 concentration. Two studies reported a significant increase in apo A1 after consuming a diet supplemented with pistachio nuts (82) and pecans (74) for four weeks. In contrast, one trial found a small but significant decrease in apo A1 concentrations after consuming a nut-enriched diet when compared with an average American diet possibly due to the 6% reduction observed in HDL-C (91).

Fifteen out of 19 trials reported significant reductions in apo B100 concentrations when the participants consumed a nut-enriched diet compared to an average American diet (66,51, 90, 96), the Mediterranean diet (89), Step I diet (20, 74, 78), Step II diet (25), and the control diet (27, 68, 82, 92, 94, 115). The beneficial effects of nuts would seem to be apparent over a wide range of intakes, ranging from 25 to 126 g of nuts per day.

Overall, in spite of the diversity in participant characteristics, comparison diets, and study design, the trials consistently demonstrated reductions in apo B100 while apo A1 remained largely unchanged with nut consumption.

**Section 4: Nut Consumption and Blood Pressure**

Elevated blood pressure is one of the major modifiable risk factors of CVD and accounts for 7.6 million premature deaths worldwide (156, 157). Compliance with advice to consume nuts has been found to inversely correlate with systolic ($P = 0.017$) and diastolic ($P = 0.041$) blood pressure (158). This section will firstly review the epidemiological studies that have examined the association between nut consumption and the risks of hypertension, stroke, and atrial fibrillation (Section 4.1). Secondly, it will focus on the clinical trials that have investigated the effects of nut consumption on blood pressure, as one of their outcome measurements (Section 4.2).

**Section 4.1 Epidemiological Studies: Hypertension, Stroke, and Atrial Fibrillation**

A cross-sectional study consisting of 13,292 adults who took part in the 1999-2004 National Health and Nutrition Examination Survey (NHANES) found that nut consumers had significantly lower systolic blood pressure ($P < 0.01$) and prevalence of hypertension ($P < 0.05$) than non-nut consumers (129, 159). It is possible that the reduction in blood pressure with frequent nut
consumption may result in a lower risk of hypertension, stroke, and atrial fibrillation\(^{160}\), which is a significant risk factor for stroke \(^{161}\).

To date, only the PHS and the Seguimiento Universidad de Navarra (SUN) cohorts have examined the association between frequent nut consumption and hypertension \(^{162,163}\), stroke \(^{164}\), and atrial fibrillation \(^{160}\) (Appendix D). The current limited epidemiologic evidence suggests a protective effect of nut consumption on the risk of hypertension, especially those with BMI < 25 kg/m\(^2\) \(^{162}\), and no effect on stroke \(^{164}\) and atrial fibrillation \(^{160}\). It is important to point out that the forms (i.e. salted, spiced, roasted, grilled, fried, raw) and types of nuts consumed, which may influence the risk of hypertension, stroke, and arterial fibrillation, were not examined in either cohort \(^{165}\).

**Section 4.2 Intervention trials: Blood pressure**

Twenty of 27 studies which have investigated the effects of nut consumption on systolic and diastolic blood pressure met the inclusion criteria (Appendix E). Most of these small-sized clinical trials performed to date have found mixed results with either a beneficial effect \((n = 5)\) or no effect \((n = 14)\) of nut consumption on blood pressure. Five studies showed that blood pressure was significantly lower following a nut-enriched diet, when compared with baseline \(^{104,114}\), an average American diet \(^{95}\), a complex-carbohydrate low calorie diet \(^{28}\), and a low SFA and cholesterol control diet \(^{115}\). Only one study reported a significantly higher systolic and diastolic blood pressure after eight-weeks of consuming an ad libitum diet enriched with 56 g of walnuts, compared with an ad libitum diet without nuts in 24 type 2 diabetic patients \(^{102}\). It is not clear whether there is a difference in the response to nut consumption on blood pressure between persons with diabetes and healthy or hypercholesterolaemic participants.

When taken together, there is no apparent effect of nut consumption on blood pressure among normotensive individuals. Future studies incorporating nuts into the usual diet and utilising a larger sample size are required to show whether there is a true blood pressure lowering effect with nut consumption in different population groups.

**Section 5: Nut consumption and novel biomarkers of CVD**

Recent research suggests that the inclusion of nuts into the diet may also lower CVD risk through other mechanisms such as decreasing oxidative stress, reducing inflammation, and improving endothelial dysfunction \(^{6,12,131,132,165,166}\).

**Section 5.1 Epidemiological studies: Novel risk factors**

The characteristics and findings of cross-sectional studies that have examined the association between nut consumption and novel risk factors in different population groups are shown in Appendix F \(^{129,159,167-170}\). When the results are taken together, the epidemiological data suggests that nut consumption especially in the context of a healthy dietary pattern appears.
to be inversely related to lipid peroxidation, inflammatory markers, cell adhesion molecules, and mortality from inflammatory disease.

Section 5.2 Intervention trials: Novel risk factors

Section 5.2.1 The role of antioxidants in oxidative stress

Oxidative damage plays a key role in the development of atherosclerosis, cancer, and other chronic diseases. The predominant fatty acids found in most nuts are MUFA, which is not an oxidation substrate. On the other hand, walnuts are rich in PUFA, which is the most vulnerable to LDL-oxidation due to the double bonds in PUFA. Studies have suggested that the higher amounts of antioxidants and phytochemicals in walnuts may counteract the pro-oxidant effect of PUFA and prevent oxidation from occurring.

Nineteen of 24 clinical trials that have examined the effect of nut consumption on antioxidant activity and biomarkers of antioxidant status met the inclusion criteria. The overall evidence seems to suggest that a nut-enriched diet improves antioxidant activity and biomarkers of antioxidant status. However, whether these potential increases can be translated into a reduction in oxidative-stress related disease is unknown.

Sixteen of 24 clinical trials that have examined the effects of chronic nut consumption on oxidative stress have met the inclusion criteria. Collectively most of the results suggest diets high in MUFA-rich nuts such as almonds, peanuts, pecans, and pistachios improved biomarkers of oxidative stress. On the other hand, the consumption of PUFA-rich walnuts had no apparent effect on these biomarkers. No definitive conclusions can be drawn based on the available evidence due to differences in methods, biomarkers assessed, and the relatively small number of studies using the same biomarker.

Section 5.2.2 Inflammation

Chronic inflammation plays an important role in the development of CVD and other chronic diseases such as cancer and diabetes. Several reviews have reported inverse associations between healthy dietary components such as nuts and concentrations of inflammatory markers.

Eleven of 16 studies that examined the effect of nut consumption on inflammatory markers as one of their endpoints met the inclusion criteria for this review. The dose of nuts used in these studies ranged from 27 to 115 g/d over four to twelve weeks. Overall, the current evidence suggests that nut consumption together with a modification of the background diet (e.g. Mediterranean diet or a diet low in SFA) may be necessary to elicit an anti-inflammatory effect. These studies did
not identify and isolate nut-specific effects. Currently, there is a gap in knowledge regarding the independent effect of nut consumption on inflammatory markers. Thus, it would be of interest for future research to investigate the true anti-inflammatory effect of nut consumption without controlling or changing the background diet.

Section 5.2.3 Endothelial function

Several CVD risk factors, such as obesity, diabetes, and high levels of LDL-C can impair the ability to maintain proper vascular tone and lead to endothelial dysfunction (110). Endothelial function is a critical early event in the pathogenesis of atherosclerosis, which is characterised by the reduced bioavailability of nitric oxide (endogenous vasodilator) and the increased expression of cellular adhesion molecules (3). Endothelial dysfunction is reversible and could potentially be improved by a diet that is high in unsaturated fatty acids and arginine (183).

To date, nine (three almonds, five walnuts, and one mixed nuts) out of twelve studies that have examined the effect of nut consumption on endothelial function met the inclusion criteria for this review (Appendix J). The dose of nuts used in these studies ranged from 27 to 75 g/d over four to twelve weeks. Overall the evidence seems to indicate that chronic walnut consumption favourably affects endothelial function (91,92,95,97,102). Furthermore, incorporating PUFA-rich walnuts or MUFA-rich almonds into a Step I diet (24), a Mediterranean diet (97) or a high carbohydrate diet enriched with omega-3 fatty acids (92) have shown to improve endothelial function. It is important to point out that the majority of these studies were conducted in a tightly controlled feeding situation (24,91,92,95) and included modification of the background diet as well as advice to consume nuts. This makes it difficult to evaluate the effects of nuts per se. There is a need for further investigations using the simple inclusion of nuts into the habitual diet to provide conclusive answers on the independent effect of nut consumption on endothelial function.

Section 6: Nut consumption and risk of diabetes

Diabetes mellitus is a group of conditions characterised by raised blood glucose levels and is a major risk factor for CVD (184,185). According to the 2008/09 NZ Adult Nutrition Survey, the total prevalence of diabetes in NZ was 7.1% in the population aged ≥ 15 years (186). This section will review the epidemiological studies and clinical trials that have investigated the role of nuts in the prevention of diabetes.

Section 6.1 Epidemiological studies: Type 2 diabetes

A cross-sectional study consisting of 13,292 adults who took part in the 1999-2004 NHANES found that tree nut consumers had a significantly lower prevalence of several risk factors for metabolic syndrome such as hypertension, low HDL-C, overweight/obesity, abdominal obesity, and elevated fasting blood glucose than non-tree nut consumers (129,159).
To date, only four epidemiological studies have examined the effects of nut consumption and risk of type 2 diabetes (Appendix K). Two studies have reported an inverse relationship between nut consumption and type 2 diabetes in women\(^{(187,188)}\). The strengths of these two cohorts include repeated dietary measurements and the use of validated FFQ. On the other hand, a positive association between frequent nut consumption and the risk of diabetes was observed in the IWHS\(^{(189)}\) whereas no association was found in the PHS\(^{(190)}\). The contradictory results observed in these four cohorts could be due to the differences in subjects’ characteristics such as sex and age, sample size, definition of nuts, frequency of dietary assessments, questionnaire used to confirm diabetes diagnoses, and level of adjustments in the statistical analyses.

Given the limited epidemiologic studies in this area, further prospective studies of longer duration using multiple validated dietary assessments, standardised diagnostic for cases are required to determine the potential role of nut consumption on type 2 diabetes.

**Section 6.2 Intervention trials: Insulin resistance and glycaemic control**

Nineteen of 29 clinical trials that have investigated the effects of chronic nut consumption on glycaemic control met the inclusion criteria (Appendix L). The dose of nuts ranged from 27 to 100 g/d. Only one study showed significantly lower blood glucose after consuming a diet enriched with almonds\(^{(25)}\) when compared with the control Step II diet. Similarly, four out of 14 studies showed significantly lower blood insulin with a diet high in almonds\(^{(25,33)}\), walnuts\(^{(101)}\), mixed nuts\(^{(114)}\). Only one study showed a significant increase in blood glucose after consuming a diet high in cashew nuts\(^{(50)}\) and another study showed a significant increase in blood insulin after the consumption of a walnut-enriched diet\(^{(102)}\). The reason for this is unclear but could be due to the differences in doses and types of nuts, study design, subjects’ characteristics, and diet composition.

Eight studies have investigated the effects of nut consumption on HbA1c, which is a long-term marker of glycaemic control, in different population groups including adults with prediabetes, type 2 diabetes, and metabolic syndrome\(^{(19,33,34,98,101,102,104,115)}\). Two studies showed significant improvement after 12-weeks consumption of almonds\(^{(34)}\) or mixed nuts\(^{(115)}\) when compared with the control diet. It is important to note that the study conducted by Jenkins et al.\(^{(115)}\) was the only study that was powered to detect a significant difference in HbA1c between the nut and control groups. Tapsell et al.\(^{(101)}\) reported a significant decrease in HbA1c after consuming a walnut-enriched diet for 12 months, however this was not significantly different from the control diet. It should be noted that these four studies were conducted in people with type 2 diabetes and the baseline HbA1c of the nut groups range from 7.1 to 8.3%\(^{(34,101,115)}\). The lack of beneficial effect in the other five studies could be due to the fact that these studies were underpowered, the relatively short duration of the
intervention (< 16 weeks), and the participants in most of these studies had low baseline HbA1c values (< 7%).

Overall the effect of chronic nut consumption on long-term glycaemic control is inconclusive. Future studies with appropriate sample size calculation and longer duration are warranted to understand the potential role of nuts in the prevention and management of diabetes.

**SECTION 7: NUT CONSUMPTION AND BODY WEIGHT**

Obesity is a major public health concern both in NZ and internationally \(^{(191)}\). The 2008/09 NZ Adult Nutrition Survey reported the prevalence of overweight and obesity was 37.0% and 27.8% respectively \(^{(186)}\). Nuts are high-fat, energy-dense foods and in theory, frequent consumption of nuts could potentially contribute to a high energy intake and promote weight gain, which may offset the cardioprotective effects of nuts \(^{(192)}\). This section will focus on the epidemiological and clinical studies that have investigated the effect of regular nut consumption on body weight.

**SECTION 7.1 EPIDEMIOLOGICAL STUDIES: BODY WEIGHT**

Several studies have examined the relationship between baseline frequency of nut consumption and BMI cross-sectionally \((Appendix M)\). These studies have consistently demonstrated an inverse or no association between frequent nut consumption and BMI. Nut consumers tend to be leaner or less obese than those who do not regularly consume nuts \((137, 141, 143, 144, 160, 162, 167, 187, 190)\). A recent NHANES study found that nut and/or tree nut consumers had significantly lower body weight, BMI, and waist circumference, as well as a significant reduction in the prevalence of abdominal obesity \((P < 0.01)\) than non-nut/tree nut consumers \((199)\). A similar finding was reported in the Prevencion con Dieta Mediterranea (PREDIMED) study, in which each daily serving (30 g) of nuts was associated with a decrease in BMI by 0.78 kg/m\(^2\) and waist circumference by 2.1 cm. The reductions were similar in each gender \((193)\). In addition, the NHS and the SUN cohort studies showed that frequent nut consumption was inversely associated with less weight gain \((194-196)\) and the risks of becoming overweight or obese \((194, 196)\) \((Appendix N)\).

**SECTION 7.2 INTERVENTION TRIALS: BODY WEIGHT**

Thirty-seven of 65 intervention trials that have investigated the effects of nuts on body weight as one of the outcomes met the inclusion criteria for this review \((Appendix O)\). Although the study design and participant characteristics differ, overall these studies provide substantial evidence that short-term consumption (three to sixteen weeks) of moderate to large amounts of nuts (20 to 126 g) does not result in significant changes in body weight. Only one study has observed a small increase in body weight (0.9 kg in men, 0.3 kg in women), when a large amount of nuts (100 g) was added to a habitual diet for four weeks without adjusting for additional energy intake \((19)\). On the other hand, two studies reported that compared with baseline, there was a small but significant reduction in body weight
(0.9 kg to 2.2 kg) following a diet rich in walnuts \(^{(104)}\) or mixed nuts \(^{(114)}\). To date, only two studies have investigated the effects of long-term (> 6 months) consumption of nuts on CVD risk factors as well as body weight change \(^{(98,101)}\). Both studies reported weight loss ranging from 1.3 to 3.3 kg following a diet high in walnuts (30 g/d) \(^{(98,101)}\) for six to twelve months when compared with baseline. It should be noted that these studies were not designed to assess body weight as a primary outcome, and in many instances the investigators provided additional dietary advice or adjusted energy intake to prevent weight gain, which makes it difficult to draw any firm conclusions.

Furthermore, six RCTs have looked at the role of nuts in the context of weight loss diets (Appendix O). These studies have shown that incorporating nuts into diets for 4 weeks to 18 months resulted in higher compliance and satiety level \(^{(197,198)}\), greater or similar reductions in body weight \(^{(28,36,198)}\) and CVD risk markers such as blood lipid profile and blood pressure \(^{(28,83,197,199)}\), compared to a nut-free, low fat or high carbohydrate diet.

To date, only five studies involving the regular consumption of nuts have included body weight as a primary outcome; two on almonds \(^{(31,39)}\), one on hazelnuts \(^{(52)}\), one on peanuts \(^{(71)}\) and one on walnuts \(^{(99)}\) (Appendix P). The findings from these five studies provide evidence that adding nuts to habitual diets for eight to twenty-four weeks resulted in either no weight change or less weight gain that predicted from the additional calories provided by the nuts. Nevertheless, one should be mindful that nuts are energy-dense and should be consumed in place of other less healthful foods as part of a heart-healthy diet.

**SECTION 8: MECHANISMS FOR BENEFICIAL EFFECTS**

Nuts are rich sources of nutrients that have been shown to lower CVD risk. This section will firstly review the potential mechanisms for improvements in biochemical indices. Secondly, the mechanisms for the observation of less than predicted weight gain when nuts are consumed regularly will be discussed.

**SECTION 8.1 MECHANISMS FOR BIOCHEMICAL INDICES**

**SECTION 8.1.1 FATTY ACID PROFILE**

Nuts are rich sources of cis-unsaturated fatty acids \(^{(14)}\). The consumption of these fats is protective against CHD by lowering TC and LDL-C while maintaining HDL-C \(^{(200,201)}\). Specifically the intake of omega-6 PUFA is inversely associated with TAG and atherosclerotic plaque \(^{(202,203)}\), whereas the intake of omega-3 PUFA, mainly alpha linolenic acid (ALA) in walnuts, lowers TAG and has anti-thrombogenic properties \(^{(204,205)}\). Cis-unsaturated fatty acids are associated with reductions in coagulation factors, inflammation, and endothelial activation, which ultimately reduces the risk of CHD \(^{(166,172,180,206)}\). Also, the intake of MUFA may also reduce LDL
oxidative susceptibility. In addition, the high amounts of unsaturated fatty acids in nuts may improve beta cell efficiency by increasing the secretion of glucagon-like peptide-1, which plays an important role in the regulation of postprandial glucose clearance and insulin sensitivity \(^{(207)}\).

**Section 8.1.2 Lysine to Arginine Ratio**

Nuts are a rich source of protein with a very high content of L-arginine and a low lysine content, giving them a favourable lysine:arginine ratio (0.19 to 0.50) \(^{(118,208)}\). This ratio may improve endothelial function \(^{(183,209,210)}\). In addition, nuts with a relatively high arginine content may exert anti-inflammatory \(^{(211)}\) and hypocholesterolaemic \(^{(118,212)}\) effects.

**Section 8.1.3 Dietary Fibre**

Dietary fibre intake is inversely associated with serum cholesterol concentrations, blood pressure, and obesity \(^{(213)}\). In addition, dietary fibre may reduce the glucose and insulin response to the diet \(^{(188,207)}\). Thus, frequent nut consumption could be an effective way to achieve adequate dietary fibre intake and maintain general well being \(^{(119)}\).

**Section 8.1.4 Phytochemicals**

Nuts contain a wide range of phytochemicals including phytosterols and polyphenols such as flavonoids, proanthocyanidins, and phenolic acids \(^{(121)}\). Dietary polyphenols have antioxidant activity that may reduce oxidative stress and inflammation \(^{(113,121)}\). In addition, phytosterols in nuts with a high fat content could be an effective way to reduce dietary cholesterol absorption and hence, lower TC, and LDL-C concentrations \(^{(212)}\).

**Section 8.1.5 Antioxidants**

A network of bioactive antioxidants in nuts may work synergistically to protect against oxidative stress, inflammation, and cell adhesion molecule expression \(^{(120,140,174,183)}\). Furthermore, antioxidants may improve insulin resistance by improving beta-cell response to glucose and insulin action \(^{(207)}\). It is important to point out that most of the antioxidants are located in the pellicle or outer soft shell of the nuts, and removing the skin could result in the loss of greater than 50% of the antioxidants present in nuts \(^{(131,175)}\). Thus, nuts should be consumed with the skin on, in order to maximise their cardioprotective effect \(^{(214,215)}\).

**Section 8.1.6 Micronutrients**

Unprocessed raw nuts are low in sodium while high in folate, calcium, magnesium, copper, and potassium. Each serving of nuts (30 g) provides up to approximately 9% recommended dietary intake (RDI) of folate, 1 to 8% of calcium, 9 to 35% of magnesium, 18 to 58% copper, and 3 to 12% potassium \(^{(210)}\). Together, these components have been shown to have a substantial impact on lowering morbidity and mortality from chronic diseases such as CVD, diabetes, cancer, hypertension, osteoporosis and other illnesses \(^{(126,217-220)}\).
Section 8.2: Mechanisms for Less than Predicted Weight Gain

There are three potential mechanisms that may explain the lower than theoretically predicted weight gain observed with nut consumption. These include the displacement of foods from the habitual diet, energy malabsorption as a result of excretion of fat in the stools, and increased resting metabolic rate (RMR) or diet-induced thermogenesis (192,221-225).

Section 8.2.1 Dietary Compensation

The dietary components in nuts, such as the high fibre and protein contents, could elicit a satiety effect and suppress appetite, resulting in a reduction in the intake of calories from other foods, i.e. dietary compensation (226). A recent review has estimated that dietary compensation accounts for 65 to 75% of the additional energy provided by nuts and seems to be the major contributor that offsets the extra energy provided by the inclusion of nuts into the diet (227).

Section 8.2.2 Energy Malabsorption

Some research has suggested that the lipids found in nuts may not be highly bioaccessible, meaning that a high proportion of these fats are excreted in the faeces when nuts are incorporated into the diet and therefore not available for energy metabolism. The mastication of nuts damaged only the first layer of cells and resulted in the incomplete release of lipids available for digestion and absorption (152,228). Studies have demonstrated a positive relationship between nut consumption and faecal fat excretion (114,229). Given that a large proportion of energy from nuts is made up of the lipid fraction, any loss of fat and energy in the stool could protect those consuming a diet rich in nuts from gaining weight (223). This theory is supported by two recent studies, which showed that the measured energy content of one serving of nuts (28 g) was 9 to 32% less than the value estimated by using Atwater factors (80,230). The incomplete energy absorption could potentially further offset around 5 to 15% of the energy contributed by nuts (227).

Section 8.2.3 Metabolic Rate

Nuts are rich in protein with a high unsaturated-to-saturated fatty acid ratio, which may increase RMR and diet-induced thermogenesis (231). Previous work has suggested that increased energy expenditure could be another possible mechanism for less weight gain than predicted and may account for approximately 10% of the energy provided by nuts (227). Two studies reported a significant increase in metabolic rate following a diet rich in peanuts for 2 to 19 weeks (68,71). Similarly, Coelho et al. (232) reported a significant 5% increase in RMR in obese individuals who consumed milk shakes that contained peanut oil for eight weeks. In contrast, daily supplementation of almonds (31,39), walnuts (101,233), and mixed nuts (including almonds, hazelnuts, and walnuts) (114) ranging from four days to twelve months failed to show any changes in RMR, resting energy
expenditure, or respiratory quotient. It is unclear whether the higher RMR in peanuts can be generalised to other types of nuts.

**Section 9: Sensory factors that may affect nut consumption**

In NZ as in many other countries, there is a recommendation to consume one serving (30 g to 42.5 g) of nuts per day \(^{234,235}\). As discussed in the previous sections, epidemiological studies and clinical trials have consistently demonstrated the importance of incorporating this amount of nuts into the diet for beneficial effects on cardiovascular risk factors \(^{130-133}\). However, to achieve optimal health benefits, nuts must be consumed regularly and in sufficient quantity. Thus, it is important to determine whether the current guideline regarding the regular consumption of raw, unsalted nuts is an acceptable and sustainable behaviour long-term.

Four studies, two using hazelnuts \(^{53,59}\) and two using peanuts \(^{71,73}\), have investigated the effects of repeated consumption of nuts on acceptance, with exposure periods ranging from four to nineteen weeks (**Appendix Q**). Raw nuts, which are relatively bland and neutral in flavour with moderately high initial liking, resulted in the maintenance for liking across exposure period \(^{53,59}\). Similarly, the addition of salt to nuts does not appear to influence liking over time \(^{71,73}\). When taken together, all four studies that have investigated the effects of regular nut consumption on acceptance suggest that nuts could be a food that is resistant to monotony as a product category in general and the recommendation to consume 30 to 42.5g nuts on a regular basis is an achievable and sustainable public health target.

Tey et al. \(^{59}\) also showed that the ‘desire to consume’ and ‘overall liking’ for ground hazelnuts were statistically significantly lower than both sliced and whole hazelnuts, whereas these measurements of the latter two did not differ statistically significantly \(^{59}\). Whole and sliced nuts were more acceptable than ground nuts, therefore recommendations should favour these forms. However, bear in mind that there are subgroups within the general population such as elderly, or people with dentures or a partial plate, who may find nuts with larger particle sizes difficult to consume. Given that the acceptance for all forms (ground, sliced or whole) of hazelnuts was high, it is advisable to recommend the inclusion of any form of hazelnuts based on the individual’s personal preference. This will provide more choices for consumers, thus enhancing compliance with the recommendation to consume nuts daily as part of a heart healthy diet.

**Section 10: Safety of nut consumption**

Despite the well-documented health benefits of nut consumption, it is important to consider two main consumer concerns regarding nut consumption, namely allergens and the potential presence of aflatoxins in nuts \(^{236}\).
Nuts are one of the most common food allergens identified (237). Food allergy occurs when an individual is exposed to a novel food and the body reacts by creating allergic antibodies (IgE) specific to the food consumed. Most nuts contain the same allergens and thus, individuals who are allergic to one nut type may have a high level of coallergy (cross-reactivity) to other closely related nut species (237,238). Studies in the UK and US have shown that around 1% of the general population suffer allergies to nuts (239). Similar prevalence (1 to 2%) has been reported in NZ (240). Approximately 20% of nut-allergic children outgrow their allergy, 20% develop more oral intolerance or sensitivity to nuts, and the remaining 60% maintains the same severity to their nut allergies (241).

Nut allergy can be diagnosed by skin-prick tests or radioallergosorbent test to detect nut-specific serum IgE antibodies, or by an oral challenge. Double-blind placebo-controlled food challenge is the gold standard for establishing a cause-effect relationship between the specific food and the clinical symptoms. Such challenges should only be conducted under the supervision of a physician or a medical specialist in a hospital setting (239,242). Once an individual has been diagnosed with nut allergy, preventive measures should be taken. In absence of proven medical treatment for food allergies, nut-allergic individuals should avoid all nuts, read all food labels carefully, and enquire about the ingredients of meals not prepared by themselves (237,238). In addition, the food industry should minimise cross-contamination, have allergen control programs and proper food labelling in order to prevent nut-allergic individuals from ingesting nuts inadvertently (237,239). Information on recognising and treating an allergic symptom should be provided to the individuals and his/her families (242).

Mycotoxins are toxic substances produced by Fusarium moulds including fumonisins B₁, B₂, and B₃ (243). Mycotoxins have been found to contaminate foods such as nuts, seeds, and grains (244). Aflatoxins are the major mycotoxin of concern because they are procarcinogens (245). However, studies have shown that the natural antioxidants and phytochemicals found in nuts can inhibit aflatoxin production (236). Currently, there is very little information available on the level of aflatoxin contamination in crops grown in NZ. Nevertheless, a recent survey on worldwide mycotoxin contamination shows that the presence and contamination of aflatoxins is very low (6%) in Oceania in comparison with other parts of the world (ranging from 13 to 88%) (246). In NZ, the maximum limit for total aflatoxins in tree nuts and peanuts is set at 0.015 mg/kg (247). The provisional maximum tolerable daily intake for fumonisins B₁, B₂, and B₃ together is 0.002 mg/kg of body weight per day (243). In addition, all peanut and pistachio nut products imported into NZ must meet the import clearance requirements from Ministry of Agriculture and Forestry Biosecurity NZ (248). As a result of the strict safety regulations in NZ, it appears that there should not be any major safety concerns regarding aflatoxin contamination in nuts purchased in NZ. Furthermore, given that peanuts and tree nuts are rarely consumed in a large quantity and they are not major components of
New Zealanders’ diet, it is likely that exposure to aflatoxins would be low and hence the detrimental effects on health would be minimal.

**SECTION 11: SUMMARY OF EVIDENCE: HEALTH BENEFITS OF NUT CONSUMPTION**

Table 5 shows a summary of evidence regarding the effects of nut consumption on chronic disease and risk factors related to these diseases. Frequent nut consumption appears to play a protective role in prevention of CVD, CHD, myocardial infarction (MI), and sudden death. Clinical studies suggest the primary mechanism is largely mediated through reductions in TC, LDL-C, and apo B concentrations. The cholesterol-lowering effects with nut consumption appears to be more pronounced in participants with elevated cholesterol concentrations, BMI < 25 kg/m², and those who are insulin-sensitive. The evidence for blood pressure, stroke, hypertension, atrial fibrillation, and diabetes is limited and warrants further investigations. Although studies suggest improvements in antioxidant status, oxidative stress, markers of inflammation and endothelial dysfunction could be another potential mechanism for the cardioprotective effect of nut consumption, the evidence is less conclusive as the improvements may not necessarily be attributed to nut consumption per se, since background diets were also modified. Both epidemiological and clinical studies have reported that despite the high energy and fat content of nuts, regular nut consumption does not appear to negatively affect body weight. It is important to highlight that no studies to date have demonstrated a detrimental effect of nut consumption on health. Nuts can therefore be incorporated as part of a heart-healthy diet.
Table 5. Summary of scientific evidence: effects of nut consumption on diseases and risk factors

<table>
<thead>
<tr>
<th>Diseases/Risk factors</th>
<th>No. of cohorts/clinical trials</th>
<th>Evidence (Effect)</th>
<th>Effects more pronounced in these conditions</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Cohorts</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CHD</td>
<td>4</td>
<td>++ (↓)</td>
<td></td>
</tr>
<tr>
<td>Non-fatal MI</td>
<td>3</td>
<td>+ (↓)</td>
<td></td>
</tr>
<tr>
<td>Heart failure</td>
<td>1</td>
<td>+/− (&quot;*)</td>
<td></td>
</tr>
<tr>
<td>Hypertension</td>
<td>2</td>
<td>+/− (&quot;*)</td>
<td></td>
</tr>
<tr>
<td>Stroke</td>
<td>1</td>
<td>+/− (&quot;*)</td>
<td></td>
</tr>
<tr>
<td>Atrial fibrillation</td>
<td>1</td>
<td>+/− (&quot;*)</td>
<td></td>
</tr>
<tr>
<td>Inflammatory markers</td>
<td>1</td>
<td>+/− (&quot;*)</td>
<td></td>
</tr>
<tr>
<td>Diabetes</td>
<td>4</td>
<td>+/− (&quot;*)</td>
<td></td>
</tr>
<tr>
<td>Weight gain</td>
<td>2</td>
<td>+ (↓)</td>
<td></td>
</tr>
<tr>
<td><strong>Clinical trials</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total cholesterol</td>
<td>43</td>
<td>++ (↓)</td>
<td>Participants with elevated cholesterol, BMI &lt; 25 kg/m², insulin-sensitive</td>
</tr>
<tr>
<td>LDL cholesterol</td>
<td>44</td>
<td>++ (↓)</td>
<td>Participants with elevated cholesterol, BMI &lt; 25 kg/m², insulin-sensitive</td>
</tr>
<tr>
<td>HDL cholesterol</td>
<td>41</td>
<td>+/− (&quot;*)</td>
<td></td>
</tr>
<tr>
<td>Triacylglycerol</td>
<td>43</td>
<td>+/− (&quot;*)</td>
<td></td>
</tr>
<tr>
<td>Apolipoprotein A1</td>
<td>19</td>
<td>+/− (&quot;*)</td>
<td></td>
</tr>
<tr>
<td>Apolipoprotein B100</td>
<td>19</td>
<td>++ (↓)</td>
<td></td>
</tr>
<tr>
<td>Systolic BP</td>
<td>19</td>
<td>+ (↓)</td>
<td>Participants with hypertension</td>
</tr>
<tr>
<td>Diastolic BP</td>
<td>19</td>
<td>+ (↓)</td>
<td>Participants with hypertension</td>
</tr>
<tr>
<td>Antioxidants</td>
<td>19</td>
<td>++ (?)</td>
<td></td>
</tr>
<tr>
<td>Oxidative stress</td>
<td>16</td>
<td>+ (↓)</td>
<td>Especially MUFA-rich nuts</td>
</tr>
<tr>
<td>Hs-CRP</td>
<td>9</td>
<td>+ (↓)</td>
<td>Especially if it includes background diet modification</td>
</tr>
<tr>
<td>Interleukin-6</td>
<td>4</td>
<td>+/− (&quot;*)</td>
<td>Especially PUFA-rich walnuts</td>
</tr>
<tr>
<td>Other inflammatory markers</td>
<td>4</td>
<td>+/− (&quot;*)</td>
<td>Especially PUFA-rich walnuts</td>
</tr>
<tr>
<td>FMD</td>
<td>3</td>
<td>+ (↓)</td>
<td>Especially PUFA-rich walnuts</td>
</tr>
<tr>
<td>ICAM-1</td>
<td>6</td>
<td>+ (↓)</td>
<td>Especially PUFA-rich walnuts</td>
</tr>
<tr>
<td>VCAM-1</td>
<td>6</td>
<td>+ (↓)</td>
<td>Especially PUFA-rich walnuts</td>
</tr>
<tr>
<td>Glycaemic control</td>
<td>19</td>
<td>+/− (&quot;*)</td>
<td></td>
</tr>
<tr>
<td>Body weight</td>
<td>37</td>
<td>++ (↓)</td>
<td></td>
</tr>
</tbody>
</table>

*Adapted from Ros [11]. Abbreviations used: CHD, coronary heart disease; BP, blood pressure; ICAM-1, inter-cellular adhesion molecule-1; FMD, flow-mediated dilation; Hs-CRP, high-sensitivity C-reactive protein; MI, myocardial infarction; PUFA, polyunsaturated fatty acids; VCAM-1, vascular adhesion molecule-1. +/− Equivocal evidence; + Limited evidence from a few studies; ++ Evidence from several studies.

No change; ↓ Decrease; ↑ Increase
SECTION 12: NEW ZEALAND CONTEXT

Forty percent of all deaths in NZ are attributed to cardiovascular disease \(^{(248)}\). It has been estimated that the annual direct and indirect costs for CVD in NZ are between \$561 million and \$721 million \(^{(250)}\). In NZ, it has been estimated that the combined effects of four nutrition-related risk factors namely elevated blood cholesterol concentrations, high blood pressure, high BMI, and inadequate fruit and vegetable intake accounted for approximately 87% of all ischaemic heart disease (IHD) deaths and 70% of all stroke deaths \(^{(251)}\). Thus, modest changes in one or more of these well-established determinants of cardiovascular risk can be an effective way to prevent and manage CVD \(^{(1)}\).

Most of the studies were conducted in populations comprised of individuals of European descent. Studies are lacking regarding the health effects of nut consumption in other populations e.g. Māori.

Two studies conducted in NZ have examined the National Heart Foundation recommendation to consume a serve of nuts per day \(^{(53,59)}\). These studies showed that consuming nuts (up to 42 g/d) on a regular basis is an acceptable and sustainable behaviour. In terms of affordability, 30 g of nuts (excluding pine nuts) costs less than \$1.50 per day, with almonds, cashews, and peanuts being the most affordable options (< \$0.60). This cost is similar to a serve of fruit, which is another healthy snack. In addition, this cost is comparable to less healthy snack foods, e.g. chocolate, cookies, crisps, and muesli bars. Consumers can choose from a wide range of nuts given that the cholesterol-lowering effects are similar across all forms (e.g. ground, sliced, whole) and varieties (e.g. almonds, hazelnuts). Thus, nuts can be an affordable and healthful inclusion in the NZ diet on a regular basis for many families.
SECTION 13: RECOMMENDATIONS

For the General Population

Nuts provide concentrated source of unsaturated fat, fibre, vitamin E, and a number of other nutrients often in short supply in the modern diet. Frequent nut consumption could be an effective way to achieve adequate essential nutrient intakes and maintain general well being. As different nuts have varying amounts of micronutrients, eating a selection is ideal.

To gain the most health benefit from nuts, it is important not to add salt, sugar or other fats, so unprocessed nuts are best. Raw nuts should be consumed with the skin on, as most of the antioxidants and phytochemicals are located in the pellicle or outer soft shell of the nut. Nuts can also be dry roasted by putting them in a shallow dish in the oven at a low to medium heat (around 100°C), stirring them occasionally until they have the desired crunch. Store nuts in a cool, dry, dark place.

The cost of nuts is often mentioned as a barrier to consumption. However, depending on the types and the sources, 30 g of nuts (excluding pine nuts) should cost less than $1.50. Even smaller amounts eaten regularly are better than none at all.

Nuts are the ultimate fast food, convenient, and easy to carry around. If you feel really hungry during morning or afternoon tea time, nuts can tide you over nicely until your lunch or evening meal.

Regular consumption of 30 g of raw nuts (a small handful) is recommended to improve diet quality and to reduce several risk factors associated with heart disease. This effect is more pronounced when nuts are consumed in place of unhealthy foods, which are highly processed with excessive amounts of salt, sugar, saturated fat, and trans fat (a fat often found in processed food).

Eating a serve of nuts each day should not adversely affect body weight in the general population. This is especially apparent when nuts replace other foods, instead of having them as add-ons to one’s usual diet.

Limited evidence suggests that the lipid-lowering effect of nut butter is comparable to whole nuts (eg. peanut butter). Thus, regular consumption of one serve of plain nut butter (2 tablespoons) may improve cardiovascular risk factors. Plain nut butter can be used to replace other spreads. Some nut butters contain added salt, sugar, and oil. Choose varieties low in added salt and sugar.

For those with or at high risk of heart disease

Individuals with high blood cholesterol concentrations should consume 30 g of nuts per day as a means of reducing blood cholesterol.

Individuals at high risk of heart disease may benefit from consuming raw nuts in place of less healthy snacks. This could further improve other risk
factors of heart disease including inflammatory markers and endothelial function.

Inclusion of one serve of nuts or plain nut butter per day should not adversely affect body weight in high risk populations.

For Health Professionals

Recent research has shown the general public will increase their nut consumption when recommended to do so by a health professional. Thus, health professionals should recommend the intake of 30 g of nuts per day as part of a heart healthy diet.

The perception by the general public that nuts are “fattening” is incorrect. Inclusion of a serve of nuts (30 g) in a person’s regular diet should not adversely affect body weight. This is especially apparent when nuts replace other foods. Plain nut butter, instead of other spreads, can be consumed as part of a healthy diet without compromising body weight.
Section 14: References


120. Vinson JA & Cai Y (2012) Nuts, especially walnuts, have both antioxidant quantity and efficacy and exhibit significant potential health benefits. *Food funct* 3(2): 134-140.


