As a result of diet and lifestyle factors, cardiovascular disease (CVD) remains the leading cause of death in Australia. Excessive dietary saturated fat, elevating blood cholesterol levels, impacts the health of many. By including almonds in a healthy diet, blood health becomes improving CVD risk.

Studies have revealed a 2% decrease in total cholesterol with almonds, and a 5% reduction in low density lipoprotein cholesterol (LDL-C) leading to a 3% decrease in CVD. From clinical trials, where the diet has been manipulated to accommodate almonds (68-84g/day), there is up to a 10% reduction in CVD.

Mechanisms that may account for the reductions seen in cohort and clinical intervention studies are likely due to the unique food matrix, including favourable fatty acids, reduction in LDL-C, and increased alpha-linolenic acid.

Results from the Physicians’ Health Study revealed a 25% reduction in the risk of cardiovascular disease and decreased risk of sudden cardiac death in the Physicians’ Health Study.

Lower LDL cholesterol with Australian almonds as an important part of a healthy diet for health professionals.

**Key Findings**

As a result of diet and lifestyle factors, cardiovascular disease (CVD) remains the leading cause of death in Australia. Excessive dietary saturated fat, elevating blood cholesterol levels impact the health of many. By including almonds in a balanced diet, blood lipid levels reduce CVD risk.

Studies have revealed a linear dose response to almonds with a 1% reduction in low density lipoprotein cholesterol (LDL-C) resulting from each 7g serving of almonds, and with this, a 2% reduction in CVD. From clinical trials, where the diet has been manipulated to accommodate larger serves of almonds (68-84g/day), there is up to a 10% reduction in LDL-C.

Mechanisms that may account for the reductions seen in cohort and clinical intervention studies are likely due to the unique food matrix, including favourable fatty acids, with this, a 2% reduction in CVD. From clinical trials, where the diet has been manipulated to accommodate larger serves of almonds (68-84g/day), there is up to a 10% reduction in LDL-C.

For Health Professionals
In Australia, saturated fat intake is too high. As a consequence, elevated blood cholesterol and blood pressure are regular features of the Australian population. Lipid levels are common risk factors for CVD. LDL-C and the proportion of LDL-C specifically, is an important biochemical biomarker for heart disease and there is early clinical evidence demonstrating that almonds also assist with biomarkers for heart disease and there is early clinical evidence demonstrating that almonds also assist with lowering blood pressure.

In a review of the four large prospective epidemiological studies, the cholesterol reduction associated with almond consumption, has been primarily attributed to the replacement of saturated fat with MUFA. The addition of MUFA to the diet from almonds also reduces the LDL:HDL ratio. The polyunsaturated to saturated fat ratio is 4 but MUFA predominates, whilst almonds are considered high in fat, they have a favourable fatty acid profile. The amount of Vitamin E in almonds is higher than other nuts and may potentially contribute to reduced risk of atherosclerosis. The amount of Vitamin E in almonds is 28mg/100g, a recognised antioxidant with the potential to reduce the amount of LDL particles that are oxidised (28mg/100g), a recognised antioxidant with the potential to reduce the amount of LDL particles that are oxidised within the food matrix. The fatty acid and amino acid profiles, the Vitamin E antioxidant content, folic acid level, amino acids, mineral content, the polyphenolic compound, and the fibre content of almonds are all potential contributors to reduce CVD risk beyond what can be measured with cholesterol testing alone. The consumption of just one handful of almonds has been shown to lower LDL cholesterol.

In a study of 307 healthy adults, those consuming almonds were compared to those who did not consume almonds. There was a reduction in risk of CVD death by 23% for each 24g/day of almonds consumed compared to controls. There was a 37% reduction in CVD risk for those consuming nuts more than four times per week compared to those who never, or seldom, consume nuts. Overall, there was a reduction in risk of CVD death of 8.3% for each 30g serving of nuts consumed weekly. In clinical trials, larger serves of almonds have achieved up to a 10% reduction in LDL-C there is approximately a 2% reduction in risk of CVD per 1% reduction in LDL-C, whilst as little as 7g of almonds achieves a 1% reduction in LDL-C and is supported by others. Importantly, for every 1% reduction in LDL-C there is approximately a 2% reduction in risk of CVD.

In studies using the nutrients present in nuts like almonds, counteract these effects. In studies using phytosterols and in particular, sitosterol – the most abundant plant sterol in almonds - interfere with cholesterol absorption and lower the proportion of LDL particles that are oxidised within the food matrix. The fatty acid and amino acid profiles, the Vitamin E antioxidant content, folic acid level, amino acids, mineral content, the polyphenolic compound, and the fibre content of almonds are all potential contributors to reduce CVD risk beyond what can be measured with cholesterol testing alone.
Almonds: Their Unique Composition

Understanding the mechanism of the key biomarkers: LDL cholesterol and blood pressure. Almonds have been shown to decrease LDL cholesterol and the proportion of LDL-C, specifically, in important biomarkers for heart disease and there is early clinical evidence demonstrating that almonds also assist with lowering blood pressure.

Almonds contain a significant amount of alpha tocopherol (28mg/100g), a recognised antioxidant with the potential to reduce the amount of LDL particles that are oxidised (28mg/100g), a recognised antioxidant with the potential to reduce the amount of LDL particles that are oxidised. Oxidised LDL may adhere to the endothelial wall and is responsible for the progression of atherosclerosis. The amount of Vitamin E in almonds is higher than other nuts and may potentially contribute to the reduced risk of atherosclerosis development. Studies demonstrating some similarities to omega-3 fatty acids in its ability to down regulate pro-inflammatory cytokines and reduce cellular adhesion particularly in endothelial dysfunction and atherosclerosis. Furthermore, studies consistently show that saturated fatty acids impair endothelial function and regulate pro-inflammatory cytokines and reduce cellular adhesion particularly in endothelial dysfunction and atherosclerosis. Furthermore, studies consistently show that saturated fatty acids impair endothelial function and regulate pro-inflammatory cytokines and reduce cellular adhesion particularly in endothelial dysfunction and atherosclerosis. Furthermore, studies consistently show that saturated fatty acids impair endothelial function and regulate pro-inflammatory cytokines and reduce cellular adhesion particularly in endothelial dysfunction and atherosclerosis. Furthermore, studies consistently show that saturated fatty acids impair endothelial function and regulate pro-inflammatory cytokines and reduce cellular adhesion particularly in endothelial dysfunction and atherosclerosis. Furthermore, studies consistently show that saturated fatty acids impair endothelial function and regulate pro-inflammatory cytokines and reduce cellular adhesion particularly in endothelial dysfunction and atherosclerosis. Furthermore, studies consistently show that saturated fatty acids impair endothelial function and regulate pro-inflammatory cytokines and reduce cellular adhesion particularly in endothelial dysfunction and atherosclerosis. Furthermore, studies consistently show that saturated fatty acids impair endothelial function and regulate pro-inflammatory cytokines and reduce cellular adhesion particularly in endothelial dysfunction and atherosclerosis. Furthermore, studies consistently show that saturated fatty acids impair endothelial function and regulate pro-inflammatory cytokines and reduce cellular adhesion particularly in endothelial dysfunction and atherosclerosis. Furthermore, studies consistently show that saturated fatty acids impair endothelial function and regulate pro-inflammatory cytokines and reduce cellular adhesion particularly in endothelial dysfunction and atherosclerosis. Furthermore, studies consistently show that saturated fatty acids impair endothelial function and regulate pro-inflammatory cytokines and reduce cellular adhesion particularly in endothelial dysfunction and atherosclerosis. Furthermore, studies consistently show that saturated fatty acids impair endothelial function and regulate pro-inflammatory cytokines and reduce cellular adhesion particularly in endothelial dysfunction and atherosclerosis. Furthermore, studies consistently show that saturated fatty acids impair endothelial function and regulate pro-inflammatory cytokines and reduce cellular adhesion particularly in endothelial dysfunction and atherosclerosis. Furthermore, studies consistently show that saturated fatty acids impair endothelial function and regulate pro-inflammatory cytokines and reduce cellular adhesion particularly in endothelial dysfunction and atherosclerosis. Furthermore, studies consistently show that saturated fatty acids impair endothelial function and regulate pro-inflammatory cytokines and reduce cellular adhesion particularly in endothelial dysfunction and atherosclerosis. Furthermore, studies consistently show that saturated fatty acids impair endothelial function and regulate pro-inflammatory cytokines and reduce cellular adhesion particularly in endothelial dysfunction and atherosclerosis. Furthermore, studies consistently show that saturated fatty acids impair endothelial function and regulate pro-inflammatory cytokines and reduce cellular adhesion particularly in endothelial dysfunction and atherosclerosis. Furthermore, studies consistently show that saturated fatty acids impair endothelial function and regulate pro-inflammatory cytokines and reduce cellular adhesion particularly in endothelial dysfunction and atherosclerosis. Furthermore, studies consistently show that saturated fatty acids impair endothelial function and regulate pro-inflammatory cytokines and reduce cellular adhesion particularly in endothelial dysfunction and atherosclerosis. Furthermore, studies consistently show that saturated fatty acids impair endothelial function and regulate pro-inflammatory cytokines and reduce cellular adhesion particularly in endothelial dysfunction and atherosclerosis. Furthermore, studies consistently show that saturated fatty acids impair endothelial function and regulate pro-inflammatory cytokines and reduce cellular adhesion particularly in endothelial dysfunction and atherosclerosis. Furthermore, studies consistently show that saturated fatty acids impair endothelial function and regulate pro-inflammatory cytokines and reduce cellular adhesion particularly in endothelial dysfunction and atherosclerosis. Furthermore, studies consistently show that saturated fatty acids impair endothelial function and regulate pro-inflammatory cytokines and reduce cellular adhesion particularly in endothelial dysfunction and atherosclerosis. Furthermore, studies consistently show that saturated fatty acids impair endothelial function and regulate pro-inflammatory cytokines and reduce cellular adhesion particularly in endothelial dysfunction and atherosclerosis. Furthermore, studies consistently show that saturated fatty acids impair endothelial function and regulate pro-inflammatory cytokines and reduce cellular adhesion particularly in endothelial dysfunction and atherosclerosis. Furthermore, studies consistently show that saturated fatty acids impair endothelial function and regulate pro-inflammatory cytokines and reduce cellular adhesion particularly in endothelial dysfunction and atherosclerosis. Furthermore, studies consistently show that saturated fatty acids impair endothelial function and regulate pro-inflammatory cytokines and reduce cellular adhesion particularly in endothelial dysfunction and atherosclerosis. Furthermore, studies consistently show that saturated fatty acids impair endothelial function and regulate pro-inflammatory cytokines and reduce cellular adhesion particularly in endothelial dysfunction and atherosclerosis. Furthermore, studies consistently show that saturated fatty acids impair endothelial function and regulate pro-inflammatory cytokines and reduce cellular adhesion particularly in endothelial dysfunction and atherosclerosis. Furthermore, studies consistently show that saturated fatty acids impair endothelial function and regulate pro-inflammatory cytokines and reduce cellular adhesion particularly in endothelial dysfunction and atherosclerosis. Furthermore, studies consistently show that saturated fatty acids impair endothelial function and regulate pro-inflammatory cytokines and reduce cellular adhesion particularly in endothelial dysfunction and atherosclerosis. Furthermore, studies consistently show that saturated fatty acids impair endothelial function and regulate pro-inflammatory cytokines and reduce cellular adhesion particularly in endothelial dysfunction and atherosclerosis. Furthermore, studies consistently show that saturated fatty acids impair endothelial function and regulate pro-inflammatory cytokines and reduce cellular adhesion particularly in endothelial dysfunction and atherosclerosis. Furthermore, studies consistently show that saturated fatty acids impair endothelial function and regulate pro-inflammatory cytokines and reduce cellular adhesion particularly in endothelial dysfunction and atherosclerosis. Furthermore, studies consistently show that saturated fatty acids impair endothelial function and regulate pro-inflammatory cytokines and reduce cellular adhesion particularly in endothelial dysfunction and atherosclerosis. Furthermore, studies consistently show that saturated fatty acids impair endothelial function and regulate pro-inflammatory cytokines and reduce cellular adhesion particularly in endothelial dysfunction and atherosclerosis. Furthermore, studies consistently show that saturated fatty acids impair endothelial function and regulate pro-inflammatory cytokines and reduce cellular adhesion particularly in endothelial dysfunction and atherosclerosis. Furthermore, studies consistently show that saturated fatty acids impair endothelial function and regulate pro-inflammatory cytokines and reduce cellular adhesion particularly in endothelial dysfunction and atherosclerosis. Furthermore, studies consistently show that saturated fatty acids impair endothelial function and regulate pro-inflammatory cytokines and reduce cellular adhesion particularly in endothelial dysfunction and atherosclerosis. Furthermore, studies consistently show that saturated fatty acids impair endothelial function and regulate pro-inflammatory cytokines and reduce cellular adhesion particularly in endothelial dysfunction and atherosclerosis. Furthermore, studies consistently show that saturated fatty acids impair endothelial function and regulate pro-inflammatory cytokines and reduce cellular adhesion particularly in endothelial dysfunction and atherosclerosis. Furthermore, studies consistently show that saturated fatty acids impair endothelial function and regulate pro-inflammatory cytokines and reduce cellular adhesion particularly in endothelial dysfunction and atherosclerosis. Furthermore, studies consistently show that saturated fatty acids impair endothelial function and regulate pro-inflammatory cytokines and reduce cellular adhesion particularly in endothelial dysfunction and atherosclerosis. Furthermore, studies consistently show that saturated fatty acids impair endothelial function and regulate pro-inflammatory cytokines and reduce cellular adhesion particularly in endothelial dysfunction and atherosclerosis. Furthermore, studies consistently show that saturated fatty acids impair endothelial function and regulate pro-inflammatory cytokines and reduce cellular adhesion particularly in endothelial dysfunction and atherosclerosis. Furthermore, studies consistently show that saturated fatty acids impair endothelial function and regulate pro-inflammatory cytokines and reduce cellular adhesion particularly in endothelial dysfunction and atherosclerosis. Furthermore, studies consistently show that saturated fatty acids impair endothelial function and regulate pro-inflammatory cytokines and reduce cellular adhesion particularly in endothelial dysfunction and atherosclerosis. Furthermore, studies consistently show that saturated fatty acids impair endothelial function and regulate pro-inflammatory cytokines and reduce cellular adhesion particularly in endothelial dysfunction and atherosclerosis. Furthermore, studies consistently show that saturated fatty acids impair endothelial function and regulate pro-inflammatory cytokines and reduce cellular adhesion particularly in endothelial dysfunction and atherosclerosis. Furthermore, studies consistently show that saturated fatty acids impair endothelial function and regulate pro-inflammatory cytokines and reduce cellular adhesion particularly in endothelial dysfunction and atherosclerosis. Furthermore, studies consistently show that saturated fatty acids impair endothelial function and regulate pro-inflammatory cytokines and reduce cellular adhesion particularly in endothelial dysfunction and atherosclerosis. Furthermore, studies consistently show that saturated fatty acids impair endothelial function and regulate pro-inflammatory cytokines and reduce cellular adhesion particularly in endothelial dysfunction and atherosclerosis. Furthermore, studies consistently show that saturated fatty acids impair endothelial function and regulate pro-inflammatory cytokines and reduce cellular adhesion particularly in endothelial dysfunction and atherosclerosis. Furthermore, studies consistently show that saturated fatty acids impair endothelial function and regulate pro-inflammatory cytokines and reduce cellular adhesion particularly in endothelial dysfunction and atherosclerosis. Furthermore, studies consistently show that saturated fatty acids impair endothelial function and regulate pro-inflammatory cytokines and reduce cellular adhesion particularly in endothelial dysfunction and atherosclerosis. Furthermore, studies consistently show that saturated fatty acids impair endothelial function and regulate pro-inflammatory cytokines and reduce cellular adhesion particularly in endothelial dysfunction and atherosclerosis. Furthermore, studies consistently show that saturated fatty acids impair endothelial function and regulate pro-inflammatory cytokines and reduce cellular adhesion particularly in endothelial dysfunction and atherosclerosis. Furthermore, studies consistently show that saturated fatty acids impair endothelial function and regulate pro-inflammatory cytokines and reduce cellular adhesion particularly in endothelial dysfunction and atherosclerosis. Furthermore, studies consistently show that saturated fatty acids impair endothelial function and regulate pro-inflammatory cytokines and reduce cellular adhesion particularly in endothelial dysfunction and atherosclerosis. Furthermore, studies consistently show that saturated fatty acids impair endothelial function and regulate pro-inflammatory cytokines and reduce cellular adhesion particularly in endothelial dysfunction and atherosclerosis. Furthermore, studies consistently show that saturated fatty acids impair endothelial function and regulate pro-inflammatory cytokines and reduce cellular adhesion particularly in endothelial dysfunction and atherosclerosis. Furthermore, studies consistently show that saturated fatty acids impair endothelial function and regulate pro-inflammatory cytokines and reduce cellular adhesion particularly in endothelial dysfunction and atherosclerosis. Furthermore, studies consistently show that saturated fatty acids impair endothelial function and regulate pro-inflammatory cytokines and reduce cellular adhesion particularly in endothelial dysfunction and atherosclerosis. Furthermore, studies consistently show that saturated fatty acids impair endothelial function and regulate pro-inflammatory cytokines and reduce cellular adhesion particularly in endothelial dysfunction and atherosclerosis. Furthermore, studies consistently show that saturated fatty acids impair endothelial function and regulate pro-inflammatory cytokines and reduce cellular adhesion particularly in endothelial dysfunction and atherosclerosis. Furthermore, studies consistently show that saturated fatty acids impair endothelial function and regulate pro-inflammatory cytokines and reduce cellular adhesion particularly in endothelial dysfunction and atherosclerosis. Furthermore, studies consistently show that saturated fatty acids impair endothelial function and regulate pro-inflammatory cytokines and reduce cellular adhesion particularly in endothelial dysfunction and atherosclerosis. Furthermore, studies consistently show that saturated fatty acids impair endothelial function and regulate pro-inflammatory cytokines and reduce cellular adhesion particularly in endothelial dysfunction and atherosclerosis. Furthermore, studies consistently show that satu
In Australia, saturated fat intake is too high. As a consequence, elevated blood pressure is common. Atherosclerosis, a significant public health problem in Australia, has many risk factors, including poor diet and unhealthy lifestyle patterns. Much evidence suggests that the atherosclerotic process is initiated by the oxidation of LDL cholesterol. Oxidised LDL may adhere to the endothelial wall and is responsible for the progression of atherosclerosis. The amount of Vitamin E in almonds is higher than other nuts and may potentially contribute to reduced risk of atherosclerosis. Furthermore, studies consistently show that saturated fatty acids impair endothelial function and atherosclerosis. Furthermore, studies consistently show that saturated fatty acids impair endothelial function and atherosclerosis. Studies have demonstrated that the administration of the nutrients present in nuts like almonds, counteract these effects. In studies using the dose and the dose of almonds suggested here. For further information go to www.australianalmonds.com.au

Almonds: Their Unique Composition

Almonds are a rich source of polyunsaturated fat, which is essential for maintaining heart health. They are also a good source of monounsaturated and polyunsaturated fats, which can help to reduce the risk of heart disease. Almonds are a good source of vitamins, minerals, and antioxidants, which can help to reduce the risk of cancer and other chronic diseases. They are also a good source of dietary fibre, which can help to maintain a healthy weight and reduce the risk of heart disease.

Phytosterols

Almonds contain a significant amount of alpha-tocopherol (28mg/100g), a recognised antioxidant with the potential to reduce the amount of LDL particles that are oxidised. Almonds also have a high content of phytosterols, which can help to reduce the amount of cholesterol that is absorbed from the diet. Almonds also contain a significant amount of  alpha tocopherol (28mg/100g), a recognised antioxidant with the potential to reduce the amount of LDL particles that are oxidised. Almonds also have a high content of phytosterols, which can help to reduce the amount of cholesterol that is absorbed from the diet.

Vitamins

A, B1, B2, B3, B5, B6, B7, B9, C, D, E, K, and K2

Minerals

Calcium, magnesium, phosphorus, potassium, sodium, iron, zinc, copper, manganese, silicon, selenium, and iodine

Other bioactives

Protein, complex carbohydrates, dietary fibre, and other bioactives

Fat

While almonds are considered high in fat, they have a beneficial fatty acid profile. They contain high levels of monounsaturated and polyunsaturated fats, which can help to reduce the risk of heart disease. Almonds are also a good source of cholesterol. This is important to consider when evaluating the nutritional profile of almonds, as a high level of cholesterol can increase the risk of heart disease.

Almonds are an excellent source of healthy fats, which can help to reduce the risk of heart disease. They are also a good source of protein, which can help to maintain a healthy weight and reduce the risk of heart disease. Almonds are also a good source of cholesterol. This is important to consider when evaluating the nutritional profile of almonds, as a high level of cholesterol can increase the risk of heart disease.

Almonds are an excellent source of healthy fats, which can help to reduce the risk of heart disease. They are also a good source of protein, which can help to maintain a healthy weight and reduce the risk of heart disease.

Almonds are an excellent source of healthy fats, which can help to reduce the risk of heart disease. They are also a good source of protein, which can help to maintain a healthy weight and reduce the risk of heart disease.
As a result of diet and lifestyle factors, cardiovascular disease (CVD) remains the leading cause of death in Australia. Excessive dietary saturated fat, elevating blood cholesterol and triglycerides, impacts the health of many. By including almonds in a balanced diet, blood health improves, reducing CVD risk.

Studies have revealed a linear dose response to almonds with a 1% reduction in low density lipoprotein cholesterol (LDL-C) resulting from each 7g serve of almonds and with this, a 2% reduction in CVD. From clinical trials, where the diet has been manipulated to accommodate larger serves of almonds (68-84g/day), there is up to a 10% reduction in LDL-C.

Mechanisms that may account for the reductions seen in cohort and clinical intervention studies are likely due to the unique food matrix, including favourable fatty acids, with this, a 2% reduction in CVD. From clinical trials, where the diet has been manipulated to accommodate larger serves of almonds (68-84g/day), there is up to a 10% reduction in LDL-C.

Studies have revealed a linear dose response to almonds with a 1% reduction in low density lipoprotein cholesterol (LDL-C) resulting from each 7g serve of almonds and with this, a 2% reduction in CVD. From clinical trials, where the diet has been manipulated to accommodate larger serves of almonds (68-84g/day), there is up to a 10% reduction in LDL-C.

Mechanisms that may account for the reductions seen in cohort and clinical intervention studies are likely due to the unique food matrix, including favourable fatty acids, with this, a 2% reduction in CVD. From clinical trials, where the diet has been manipulated to accommodate larger serves of almonds (68-84g/day), there is up to a 10% reduction in LDL-C.

Studies have revealed a linear dose response to almonds with a 1% reduction in low density lipoprotein cholesterol (LDL-C) resulting from each 7g serve of almonds and with this, a 2% reduction in CVD. From clinical trials, where the diet has been manipulated to accommodate larger serves of almonds (68-84g/day), there is up to a 10% reduction in LDL-C.

Mechanisms that may account for the reductions seen in cohort and clinical intervention studies are likely due to the unique food matrix, including favourable fatty acids, with this, a 2% reduction in CVD. From clinical trials, where the diet has been manipulated to accommodate larger serves of almonds (68-84g/day), there is up to a 10% reduction in LDL-C.

Studies have revealed a linear dose response to almonds with a 1% reduction in low density lipoprotein cholesterol (LDL-C) resulting from each 7g serve of almonds and with this, a 2% reduction in CVD. From clinical trials, where the diet has been manipulated to accommodate larger serves of almonds (68-84g/day), there is up to a 10% reduction in LDL-C.

Mechanisms that may account for the reductions seen in cohort and clinical intervention studies are likely due to the unique food matrix, including favourable fatty acids, with this, a 2% reduction in CVD. From clinical trials, where the diet has been manipulated to accommodate larger serves of almonds (68-84g/day), there is up to a 10% reduction in LDL-C.

Studies have revealed a linear dose response to almonds with a 1% reduction in low density lipoprotein cholesterol (LDL-C) resulting from each 7g serve of almonds and with this, a 2% reduction in CVD. From clinical trials, where the diet has been manipulated to accommodate larger serves of almonds (68-84g/day), there is up to a 10% reduction in LDL-C.

Mechanisms that may account for the reductions seen in cohort and clinical intervention studies are likely due to the unique food matrix, including favourable fatty acids, with this, a 2% reduction in CVD. From clinical trials, where the diet has been manipulated to accommodate larger serves of almonds (68-84g/day), there is up to a 10% reduction in LDL-C.

Studies have revealed a linear dose response to almonds with a 1% reduction in low density lipoprotein cholesterol (LDL-C) resulting from each 7g serve of almonds and with this, a 2% reduction in CVD. From clinical trials, where the diet has been manipulated to accommodate larger serves of almonds (68-84g/day), there is up to a 10% reduction in LDL-C.

Mechanisms that may account for the reductions seen in cohort and clinical intervention studies are likely due to the unique food matrix, including favourable fatty acids, with this, a 2% reduction in CVD. From clinical trials, where the diet has been manipulated to accommodate larger serves of almonds (68-84g/day), there is up to a 10% reduction in LDL-C.

Studies have revealed a linear dose response to almonds with a 1% reduction in low density lipoprotein cholesterol (LDL-C) resulting from each 7g serve of almonds and with this, a 2% reduction in CVD. From clinical trials, where the diet has been manipulated to accommodate larger serves of almonds (68-84g/day), there is up to a 10% reduction in LDL-C.

Mechanisms that may account for the reductions seen in cohort and clinical intervention studies are likely due to the unique food matrix, including favourable fatty acids, with this, a 2% reduction in CVD. From clinical trials, where the diet has been manipulated to accommodate larger serves of almonds (68-84g/day), there is up to a 10% reduction in LDL-C.

Studies have revealed a linear dose response to almonds with a 1% reduction in low density lipoprotein cholesterol (LDL-C) resulting from each 7g serve of almonds and with this, a 2% reduction in CVD. From clinical trials, where the diet has been manipulated to accommodate larger serves of almonds (68-84g/day), there is up to a 10% reduction in LDL-C.

Mechanisms that may account for the reductions seen in cohort and clinical intervention studies are likely due to the unique food matrix, including favourable fatty acids, with this, a 2% reduction in CVD. From clinical trials, where the diet has been manipulated to accommodate larger serves of almonds (68-84g/day), there is up to a 10% reduction in LDL-C.

Studies have revealed a linear dose response to almonds with a 1% reduction in low density lipoprotein cholesterol (LDL-C) resulting from each 7g serve of almonds and with this, a 2% reduction in CVD. From clinical trials, where the diet has been manipulated to accommodate larger serves of almonds (68-84g/day), there is up to a 10% reduction in LDL-C.

Mechanisms that may account for the reductions seen in cohort and clinical intervention studies are likely due to the unique food matrix, including favourable fatty acids, with this, a 2% reduction in CVD. From clinical trials, where the diet has been manipulated to accommodate larger serves of almonds (68-84g/day), there is up to a 10% reduction in LDL-C.