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Dietary Intake of Nuts and Cardiovascular Prognosis

David Good, MD,[*] Carl J. Lavie, MD,[†] Hector O. Ventura, MD[†]

[*]*Montgomery Cardiovascular Associates, Montgomery, AL*
[†]*Ochsner Clinic Foundation, New Orleans, LA*

ABSTRACT

There is a general perception that fat intake increases cardiovascular (CV) risk. We review the literature regarding the consumption of a high fat food, nuts, and CV risk. We also review the effect of metabolic studies relating dietary intervention with nuts and the effect of this intervention on established CV risk factors, especially low-density lipoprotein (LDL) and high-density lipoprotein (HDL) cholesterol and weight. The consumption of nuts is associated with a marked 40%–50% decrease in CV risk in large population based studies. Nut consumption is also associated with clinically relevant reduction in LDL cholesterol (−9% to −16%) without adversely affecting HDL cholesterol or causing a significant amount of weight gain.

Dietary habits have long been thought to be related to cardiovascular (CV) disease prevalence and progression. Many large epidemiological studies have shown correlations between fat intake, especially saturated fat intake, and CV risk. The seven-country study was especially important in solidifying the role of saturated fat intake as a major risk factor for CV disease, while showing monounsaturated fat to be negatively correlated and other fats to be neutral.[1] After this initial work, low-fat diets were generally felt to be best for patients with coronary heart disease (CHD). One of the tenets of the American Heart Association step 1 and 2 diets was the limitation of total fat intake. Because of these early works and early dietary prescriptions, studies have cast doubt upon the wisdom of substantially limiting total fat intake. In light of this, as well as other issues, we review impact of a diet high in nuts, a food with very high fat content, and its effect upon CV disease. While studies of nuts have many of the same limitations and confounding variables that most epidemiological studies suffer from, the overall pattern is very suggestive that the consumption of nuts can decrease the incidence of CHD.

Address correspondence to:
Carl J. Lavie, MD, FACC, FACP, FCCP
Medical Director, Cardiac Rehabilitation
Director, Exercise Laboratories
Ochsner Clinic Foundation
1514 Jefferson Highway
New Orleans, LA 70121-2483
Tel: (504) 842-5874
Fax: (504) 842-5875
Email: clavie@ochsner.org

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EPIDEMIOLOGICAL STUDIES

The virtues and limitations of epidemiological studies are the subject of an enormous amount of literature and will not be reiterated here. Suffice it to say that a multitude of confounding factors (genetic, environmental, and cultural/lifestyle) can plague conclusions that are drawn from such studies. One confounder relevant to our discussion is that the consumption of nuts is inversely proportional to the consumption of animal products,[2] nut consumption being highest in vegetarian and vegan groups.

Sub-studies in both the Nurses' Health Study[3] and the Physicians' Health Study[4] noted that the intake of nuts was associated with a lowered risk of adverse CV events. In the Physicians' Health Study, consuming nuts more than two times a week was associated with a 48% reduction in the risk of sudden cardiac death (SCD), as well as a 30% risk reduction for total CHD death.[4] Rates of non-SCD and acute myocardial infarction (MI) were not different.

Some of the most striking evidence comes from the Adventist Health Study, which determined the dietary and lifestyle habits of members of the Seventh-Day Adventist church and correlated these to CV risk.[5] Seventh-Day Adventists have religious prohibitions against smoking and alcohol consumption and the reported rates of both were quite low in the samples. The risk reduction in this study was similar to that in the Physicians' Health Study, with those consuming nuts >5 times per week having a nearly 50% reduction in CV risk.[6] There was also a dose-response relationship between the amount of nuts consumed and the decrease in relative risk.

There are also two large trials with more complex dietary intervention. Nuts were included in the Dietary Approaches to Stop Hypertension (DASH) Diet plan in a category of ''nuts, seeds, and legumes'' as a part of the DASH diet.[7] The Portfolio diet had multiple interventions intended to lower serum low density

Table 1. Profiles of Various Nuts

Nut type	MUFA	PUFA	Sat	Fiber	Mg	K	ALA
Almonds	30.889	12.07	3.7	12.2	268	705	
Peanuts	24.4	15.6	6.8	8.5	168	7.5	
Walnuts	8.9	47.2	6.1	6.7	158	441	9.1
Hazelnuts	45.7	7.9	4.5	9.7	163	680	
Pistachios	23.3	13.45	5.4	10.3	121	1025	
Pecans	40.8	21.6	6.1	9.17	121	410	
Macadamia nuts	58.9	1.5	12.1	8.6	130	368	
Cashews	23.8	7.8	7.8	3.3	292	660	
Chestnuts	.6	.3	.16		84	447	

From USDA Web site: www.nal.usda.gov/fnic/foodcomp/search/.

All numbers in grams/100 grams. MUFA=mono-unsaturated fats; PUFA=polyunsaturated fats; Sat=saturated fats; Mg=magnesium; K=potassium; ALA=alpha-linolenic acid.

lipoprotein (LDL) cholesterol. This diet included almonds as part of the dietary ''portfolio,'' as well as oats (soluble fibers), plant sterols, and soy products; it lowered LDL cholesterol, as well as C-reactive protein (CRP) levels, with comparable efficacy to taking 20 mg of lovastatin while on a low-fat diet.[8]

One practical concern is whether the type of nut is important (the relative content of nutrients and minerals is listed in Table 1). Although peanuts are technically legumes, they are included as they are essentially interchanged with nuts in the American diet. The following are some of the important differences. Peanuts have a larger percentage of protein (26%) than most nuts. Chestnuts, on the other hand, consist mostly of carbohydrates with very little fat content. Although most nuts have very little saturated fatty acid content, various nuts differ in this regard. For example, macadamia nuts have almost twice the saturated fatty acid content, while almonds have roughly half the average saturated fatty acid content.

Outside of peanuts and chestnuts, the main difference in nutrient content between nut types is the ratio of poly-unsaturated fats (PUFA, the predominant fat in walnuts) content and mono-unsaturated fats (MUFA) content (the predominant form in almonds). Included in polyunsaturated fatty acids is alpha-linolenic acid or ALA (N (18, 3), 9, 12, 15-octadecatrienoic acid), an omega-3 fatty acid that has somewhat similar metabolic effects of the marine omega-3 fatty acids, that is only present in significant amounts in walnuts.

Although there may be small differences, the consumption of MUFA and PUFA appear to have similar effects on total cholesterol/LDL cholesterol lowering and have no-to-minimal effect on high-density lipoprotein (HDL) cholesterol.[7] Multiple studies done in CHD patients have shown that increasing consumption of nuts with following careful dietary advice can lead to substantial reductions in LDL cholesterol by −9% to −16%.[9–12] In fact, pecans,[9] walnuts,[10,11] pistachios,[12] and peanuts[13] all lower levels of LDL cholesterol. One study has suggested a positive correlation between walnut consumption, HDL cholesterol and apolipoprotein A1 levels[14] and another suggested increasing HDL with pistachios.[12] These effects are probably not the most important, as the most consistent quantifiable results have been a decrease of LDL cholesterol with increasing consumption of nuts.[10,11,15,16]

Other than fatty acids, the major dietary components of nuts are vitamins, minerals, and fiber. Nuts are particularly high in magnesium, which is vital to many biological processes, including renal function and blood pressure regulation. Low intake of magnesium is correlated with hypertension, type II diabetes mellitus, and metabolic syndrome,[17] and serum magnesium levels are inversely correlated with blood pressure. However, two Cochrane systematic reviews determined that, in hypertensive patients, supplementation with magnesium or combinations of magnesium and potassium or calcium has not been adequately studied to draw definitive conclusions.[18,19] The effects of these minerals to prevent hypertension in the normotensive individuals, however, are intriguing, but are not definitively known. The specific effects of dietary supplementation with nuts on blood pressure have not been well studied. Some nuts (notably almonds) also have very high content of vitamin E. Dietary intake of vitamin E has been associated with decreased amount of CHD, although the results have been far from consistent.[20–22]

FATTY ACIDS AND CV DISEASE

The omega-3 and omega-6 fatty acids have been extensively studied epidemiologically as well as in basic science contexts.[23–26] (The ''omega'' system defines fatty acids from the position of the first double bond relative to the last [omega] carbon, leading to

Table 2. Potential Benefits of Omega-3 Fatty Acids

- Improvement in autonomic tone (even at low doses)
- Anti-arrhythmic effects (even at low doses)
- Decreased platelet aggregators
- Vasodilation
- Redirection in blood pressure
- Anti-inflammatory effects
- Plaque stabilization
- Reduction in triglycerides

chemically diverse compounds being included in the same category. Note: This is not the system used by international union of pure and applied chemistry). The marine-derived omega 3 fatty acids were brought to the forefront due to the decreased incidence of CHD in fish eating populations, especially the Greenland natives subsisting on very high-fat diets, and the potential benefits are listed in Table 2. Eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA) are two significant marine-derived omega 3 fats. Fish intake and supplementation with DHA/EPA have been studied, and, in general, have been shown to be beneficial with regards to total mortality and SCD.[27] Most of these effects have been demonstrated in the context of acute and chronic CHD syndromes. There has also been considerable discussion of anti-arrhythmic effects, especially reductions in SCD.[23–25] The anti-arrhythmic effects are plausible based on population studies of SCD, but in the high-risk patient population with implantable cardiac defibrillators, the results have been discordant.[23–25,28]

The most common plant-derived omega-3 fatty acid is ALA, which is a PUFA with 18 carbons (N)[2,14] and is very abundant in some seed-derived oils such as flaxseed, but among nuts is only found in appreciable amounts in walnuts. ALA is converted to other omega-3 acids endogenously, and walnuts have been shown in one study to raise the levels of both ALA and EPA at very low levels of intake (four walnuts/day).[29] As marine-derived omega-3 fatty acids are not a large part of most Mediterranean diets, ALAs derived from walnuts, frogs, and snails are felt by some to be the active ingredient in the Mediterranean diet.[30,31]

One study has suggested that ALA content in serum (a proxy for dietary intake) is a marker of stroke risk.[32] In this study, serum saturated fatty acid levels were associated with an increase in stroke risk, whereas serum ALA level was associated with a significant decrease in risk. Other studies have suggested a favorable effect on inflammatory markers in hyperlipidemic patients[33,34] although they are attenuated by background diet as well as genetic factors.[35] Most trials of ALA supplementation are limited to trials with questionable methodology or with very low index event rates limiting the conclusions that may be drawn. The debate over the effect of ALA is only partially relevant in this context, as walnuts are the only nut with large quantities of ALA.

Maintaining an ideal dietary ratio of omega-6 to omega-3 may be important, but a detailed discussion of this is beyond the scope of this review. Omega-6 fatty acids provide precursors for pro-inflammatory arachidonic acid, providing the theoretical impetus to maintain a higher quantity of omega-6. Eating higher amounts of nuts would worsen this ratio, as most contain very large amount of omega-6 fatty acids. This ratio is of practical benefit in that this ratio needs to be lower to obtain a high amount of omega-3 while taking a reasonable amount of total fats in the diet to maintain body weight. In addition, an important paper and position statement recently from the American Heart Association supports the benefits of omega-6 as well as omega-3.[36]

Dietary studies, however, have enormous deficits to overcome. Also, when a particular association of a dietary component with a disease state has been well-established, the causal agent is not always clear. Thus, various conundrums have arisen, such as the so-called ''French paradox,'' that is not really a paradox, but rather a failure of the present model to adequately explain the risk associated with the French diet. These complexities make diet especially difficult to treat in a reductionist manner, and to some extent complex foods such as nuts may not at present have a very good mechanistic explanation of their effects on CHD risk. In light of this, we feel that theoretical considerations such as ''worsening of the omega-6:omega-3 ratio'' should be secondary to the actual observed marked reduction in CHD risk that has been noted.

WEIGHT REGULATION

One concern of increasing dietary intake of certain foods such as nuts (considering that one gram of fat is equal to 9 calories) is potentially increasing caloric consumption in a population with a very high and increasing prevalence of overweight and obesity.[37–40] The results are conflicting, but, in general, with whole nut supplements, there is no significant weight gain.[16] In fact, in free-living individuals eating a Mediterranean diet, high levels of nut consumption are correlated with a lower incidence of obesity and weight gain,[16,41] which may be due to increased satiety associated with nut consumption. Peanut oil supplementation, however, in obese patients has been reported to lead to a large amount of weight gain.[42]

CONCLUSIONS

In the general population, consuming nuts frequently appears to be effective for CV risk reduction.

The protective effect may be due to a reduction in LDL cholesterol, although the magnitude of risk reduction in epidemiologic studies appears to be out of proportion to the degree of LDL cholesterol lowering. Although theoretically nut consumption could increase caloric intake resulting in weight gain, this has not been associated with an increased risk of overweight or obesity. At the present time, for most individuals, increased nut consumption appears to ''outweigh'' the risks.

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