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ScienceDirect
Nutrition Research 26 (2006) 397–402
NUTRITION RESEARCH
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A pecan-enriched diet increases γ-tocopherol/cholesterol and decreases thiobarbituric acid reactive substances in plasma of adults

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Received 19 April 2006; revised 13 June 2006; accepted 28 June 2006

Abstract

Consumption of nuts is associated with a reduced risk of coronary heart disease, and dietary intervention studies incorporating pecans show improved lipid profiles. The unsaturated fats in pecans are protected against oxidation by the high concentrations of γ-tocopherol and polymeric flavanols. The aim of this study was to determine whether plasma concentrations of tocopherols and measures of antioxidant capacity and of oxidative stress are affected by incorporation of pecans in the diet. In a randomized, controlled, crossover feeding study, 24 subjects were assigned to 2 diets, each for 4 weeks: a control diet and a pecan-enriched (20% of energy) diet. Cholesterol-adjusted plasma γ-tocopherol increased by 10.1% ($P < .001$), α-tocopherol decreased by 4.6% ($P < .001$), and malondialdehyde concentrations measured as thiobarbituric acid reactive substances decreased by 7.4% ($P < .05$) on the pecan diet. No changes were observed for ferric-reducing ability of plasma or Trolox equivalent antioxidant capacity values. These data provide some evidence for potential protective effects of pecan consumption in healthy individuals.

Keywords: Pecans; Humans; α-Tocopherol; γ-Tocopherol; TBARS; FRAP; TEAC

1. Introduction

Consumption of nuts has been associated with a reduced incidence of cardiovascular mortality [1-4]. Controlled-feeding trials with pecans [5,6] and other nuts [7-10] have shown improved lipid profiles with a decrease in total and low-density lipoprotein (LDL) cholesterol and in triglycerides in healthy and hyperlipidemic individuals. These results are attributed to the fatty acid contribution of nuts, low in saturated fat and high in mono- and polyunsaturated fat. Further benefits of nut consumption may result from their content of tocopherols and polyphenolic substances. These substances are of interest because of their potent antioxidant capacity and possible protective effect on human health.

Pecans (*Carya illinoensis*), now a popular American tree nut, were an early staple of Native Americans who inhabited the Southern regions of the country. The main fatty acids in pecan nuts are oleic acid and linoleic acid, contributing 56.4% and 18.9% of the total lipid, respectively [11]. Although the favorable effects of pecan intake on the serum lipid profile have been shown, there has been very little investigation into the contribution of nuts to antioxidant protection. The most important natural antioxidants in plant fats are tocopherols, and pecans are a rich source of γ- and a poor source of α-tocopherol, containing 24.4 and 1.4 mg per 100 g of nut, respectively. Pecans also contain complex flavanoid substances, especially proanthocyanidins, or condensed tannins, which are monomers and polymers of the flavan-3-ols unit [12,13]. These substances are recognized for their effective inhibition of lipid oxidation in foods and possibly in biological systems.

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doi:10.1016/j.nutres.2006.06.022

Previously, we showed that a pecan-enriched diet improved serum lipids in human subjects [5]. The aim of this study was to evaluate the effect of consuming a pecan-rich diet on plasma α- and γ-tocopherol concentrations and on measures of antioxidant capacity and lipid peroxidation in healthy persons.

2. Methods and materials

2.1. Subjects

Twenty-four healthy volunteers (14 men and 10 women) were enrolled in this study. The subjects were between 25 and 55 years of age (mean ± SD, 38.1 ± 8.8) and their mean body mass index was 25.4 ± 5.0 kg/m^2 and did not change during the study. All study participants were in good health with no history of hypertension, heart disease, or other metabolic disease, and were not taking medications known to affect serum cholesterol. Subjects were within reference ranges on clinical laboratory tests, including measurement of triacylglycerol (1.23 ± 0.67 mmol/L), total cholesterol (5.04 ± 0.84 mmol/L), and LDL cholesterol (3.27 ± 0.65 mmol/L). All subjects attended detailed informational sessions and gave written informed consent to their participation in the study. The study was approved by the Institutional Review Board of Loma Linda University.

2.2. Study design, dietary intervention, and study timeline

A detailed description of the study procedures has been published elsewhere [5]. Briefly, this was a randomized, single-blind, crossover, controlled-feeding trial. After an initial 2-week run-in phase, the subjects were randomly assigned to consume either a control diet or a pecan-enriched diet for 4 weeks. The groups then reversed diets and continued for another 4 weeks. Initially, subjects were assigned to a specific energy level based on height, weight, age, and physical activity. Thereafter, subjects were regularly weighed and energy levels were adjusted to maintain body weight. All meals were obtained from research staff. Sunday through Friday breakfast and dinner were consumed at the U.D. Register Nutrition Research dining facility under the supervision of a senior researcher. Subjects picked up lunch at breakfast and evening snack at dinner, and Saturday meals were frozen and/or packed in ice for home consumption. All foods were weighed to the nearest 0.5 g.

Nutrient composition of study diets is shown in Table 1. The control diet adhered to the recommendations of a heart-healthy diet [14]. The pecan diet used the same food items as the control diet except that 20% of energy was provided by pecans. Each food item of the control menu was proportionately scaled down by 20% (ie, total energy was reduced by 20%) to accommodate the pecans. Eight weekday menus and 2 weekend menus were used on a rotating basis. The nutrient content of the study diets was calculated by using the Nutrition Data System for Research software developed by the Nutrition Coordinating Center of the University of Minnesota version 5.0 (Minneapolis, Minn). The pecan diet was substantially higher in oleic acid, linoleic acid, and γ-tocopherol than the control diet.

Table 1
Nutrient content of the control and pecan supplemented diets provided to participants per 8400 kJ

	Control diet	Pecan diet
Energy (kJ)	8400	8400
Total fat		
(g/d)	62.4	91.6
(% Energy)	28.1	41.2
Saturated fat		
(g/d)	20.6	20.1
(% Energy)	9.27	9.05
Monounsaturated fat		
(g/d)	24.1	42.9
(% Energy)	10.85	19.31
Polyunsaturated fat		
(g/d)	13.1	22.9
(% Energy)	5.90	10.31
Linoleic acid		
(g/d)	11.6	21.2
(% Energy)	5.22	9.54
α-Linolenic acid		
(g/d)	1.3	1.6
(% Energy)	0.59	0.72
Protein		
(g/d)	65.9	58.0
(% Energy)	13.18	11.6
Carbohydrate (g)	306	253
Dietary fiber (g)	22.0	23.1
Dietary cholesterol (mg)	165	132
α-Tocopherol (mg)	6.76	6.22
γ-Tocopherol (mg)	13.7	25.1

Values were obtained by using the Nutrition Data System for Research (NDS-R) (Version 5.0, 2004, University of Minnesota Nutrition Coordinating Center, Minneapolis, MN). Averages were calculated from 8 weekday menus and 2 Saturday (take home) menus.

2.3. Sample collection and biochemical measurements

Blood samples were collected after the subjects had fasted overnight at the end of each of the 4-week experimental periods. Plasma and serum were prepared within 60 minutes of collection and were immediately portioned into aliquots and stored at −80°C.

Concentrations of α- and γ-tocopherol in plasma were measured by using normal-phase high-performance liquid chromatography (HPLC) with fluorometric detection (excitation, 292 nm; emission, 330 nm) as described by Kramer et al [15]. Plasma proteins were precipitated with ethanol and samples were extracted with hexane and injected onto a silica column (Supelcosil LC-Diol, 5 μm, 250 × 4.6 mm; Supelco Inc., Bellefonte, Penn) using hexane-isopropanol (99:1, vol/vol) as a mobile phase at a flow rate of 1 mL/min.

The thiobarbituric acid (TBA) assay is commonly used to measure malondialdehyde (MDA). Because of the lack of specificity of this assay in biological systems, results are expressed as thiobarbituric acid reactive substances (TBARS). Plasma TBARS were measured by using HPLC

Table 2
Plasma α- and γ-tocopherol and the ratio of α- and γ-tocopherol to cholesterol of subjects given the control and pecan diets

	Control diet		Pecan diet		Diet effect (pecan minus control)		P
	Mean	SEM	Mean	SEM	Mean	SEM	
α-Tocopherol (μmol/L)	24.68	2.07	21.41	2.07	−3.27	0.51	<.001
γ-Tocopherol (μmol/L)	3.82	0.26	3.84	0.26	0.02	0.10	.936
α-Tocopherol/total cholesterol (mmol/mol)	5.00	0.27	4.63	0.27	−0.37	0.09	<.001
γ-Tocopherol/total cholesterol (mmol/mol)	0.79	0.05	0.87	0.05	0.08	0.02	<.001

LSM and SEM after each diet and the estimated diet effect for outcome variables.

with fluorescence detection as described by Fukunaga and Yoshida [16]. The reaction was carried out by mixing an aliquot of plasma with 0.2% (wt/vol) thiobarbituric acid in 0.1 mol/L sodium acetate buffer (pH 3.5). After heating at 95°C for 60 minutes, the reaction solution was centrifuged and an aliquot of the supernatant was injected into the HPLC column without neutralization or extraction. The TBA-MDA adduct was separated on a reversed-phase TSK gel ODS 80Tm column (Tosoh Biosep, Montgomeryville, Penn) and quantified by a Shimadzu RF-535 (Moorpark, Calif) fluorescence detector with excitation at 515 nm and emission at 553 nm. The mobile phase was a mixture of acetonitrile-water (7:3, vol/vol) under isocratic conditions at ambient temperature.

The ferric-reducing ability of plasma (FRAP) assay measures the combined reductive or antioxidant potential of electron-donating antioxidant in plasma. It uses a timed, redox-linked ferric to ferrous iron reduction in a 2,4,6-tripyridyl-s-triazine colorimetric reaction. The assay was performed as described by Benzie and Strain [17] using a Beckman DU spectrophotometer at 37°C and a 5-minute reaction time. Solutions of known concentrations of Fe(II), ascorbic acid, and Trolox were used as standards.

Trolox equivalent antioxidant capacity (TEAC) was measured in plasma by using the method of Miller et al [18] as modified by Re et al [19]. This improved technique for the generation of the 2,2′-azinobis(3-ethylbenzothiazoline 6-sulfonate) radical cation ($ABTS^{\bullet+}$) involves the direct production of the blue-green chromophore through the reaction between ABTS and potassium persulfate. Plasma samples were deproteinized by adding an equal volume of 10% trichloroacetic acid. After centrifugation, the supernatant was added to the ($ABTS^{\bullet+}$) radical solution. The decrease in radical concentration was monitored spectrophotometrically at 30°C for 6 minutes and related to the decrease obtained with Trolox. Results are expressed as TEAC.

2.4. Statistical analyses

Changes in outcome variables in response to dietary treatment were estimated by using mixed linear models that included fixed terms for diet and period and a random term for subjects. The model for TBARS used the log-transformed data to correct for nonnormality of the random subject effects. Analyses were performed by using the SAS software version 8.0 (SAS, Inc., Cary, NC). Data are expressed as least squares means (LSM) ± SEM.

3. Results

3.1. Effect of the pecan diet on α- and γ-tocopherol

In this study, a portion of pecans equivalent to 20% of energy was incorporated in the diet. To adjust energy intake, all other foods were reduced proportionately. Consequently, the mean intake of natural-source α-tocopherol decreased slightly and that of γ-tocopherol increased substantially on the pecan diet. As shown in Table 2, there was a statistically significant decrease in serum α-tocopherol and serum α-tocopherol normalized to serum total cholesterol, and a significant increase in serum γ-tocopherol normalized to serum cholesterol on the pecan diet.

3.2. Effect of the pecan diet on antioxidant markers

There was no significant difference between the 2 diets in the antioxidant capacity as measured by the FRAP or TEAC assay. Plasma TBARS measured by HPLC was significantly lower on the pecan diet (Table 3).

Table 3
Plasma markers of lipid oxidation and antioxidant status of subjects given the control and pecan diets

	Control diet, mean (95% CI)	Pecan diet, mean (95% CI)	Diet effect (pecan minus control), mean (95% CI)	P
MDA[a] (mmol/L)	0.5798 (0.4881-0.6884)	0.5369 (0.4517-0.6377)	−0.048 (−0.074-−0.009)	<.014
FRAP (mmol/L)	0.831 (0.767-0.894)	0.839 (0.776-0.902)	0.008 (−0.029-0.045)	.661
TEAC (mmol/L)	1.784 (1.669-1.900)	1.791 (1.675-1.907)	0.007 (−0.025-0.038)	.672

LSM (and 95% confidence interval [CI]) after each diet and the estimated diet effect for outcome variables.
[a] Measured as TBARS.

4. Discussion

Despite the favorable effects of diets high in unsaturated fat on lipid profiles, concern exists that such diets could increase lipid peroxidation, thereby negating some of the cardioprotective effects [20,21]. The fat of nuts, although highly unsaturated, is rich in tocopherols, and tocopherols, once absorbed, may potentially inhibit in vivo oxidative modification of lipoproteins [22]. Pecans contain little α- but substantial amounts of γ-tocopherol, and the introduction of pecans at 20% of energy produced a diet containing less α- and more γ-tocopherol than the control diet. Therefore, the reduction in absolute and lipid-adjusted plasma concentrations of α-tocopherol on the pecan diet may be explained by the diet's lower α-tocopherol content.

The observed reduction in cholesterol-adjusted α-tocopherol may also be due to the vitamin's being transported in the plasma mainly by LDL and a change in lipoprotein levels impacts the vitamin's concentration [23]. As previously reported from this same study, the pecan-enriched diet resulted in significant decreases in plasma LDL [5]. Our results agree with those observed by Ros et al [24], who reported a decrease in α-tocopherol concentration in LDL when subjects were placed on a walnut-rich diet. Similar to pecans, walnuts contain mostly γ- and practically no α-tocopherol (20.8 and 0.7 mg per 100 g, respectively), and their introduction into a Mediterranean diet at 32% energy replaced α-tocopherol–containing dietary fats. Almonds, on the other hand, are rich in α-tocopherol (25.9 mg per 100 g). In a tightly controlled metabolic intervention study, a dose-response effect was observed between almond consumption and plasma lipid–adjusted α-tocopherol concentrations [25].

The pecan-enriched diet produced a significant increase in cholesterol-adjusted plasma γ-tocopherol. Both α- and γ-tocopherol are effective inhibitors of lipid oxidation in foods and biological systems. However, recent studies suggest that γ-tocopherol is an overlooked nutrient that may have unique functions in detoxifying nitrogen dioxide and reactive nitrogen species [26,27]. An investigation of the effect of α- and γ-tocopherol supplementation on platelet aggregation and thrombosis in rats found that γ-tocopherol leads to a greater decrease in platelet aggregation and delay of arterial thrombogenesis than does α-tocopherol supplementation [28,29]. In addition, there is some evidence that γ-tocopherol may be protective against cardiovascular disease, as plasma γ-tocopherol concentrations were inversely associated with increased morbidity and mortality due to cardiovascular disease in population studies [30,31].

The present study is, to our knowledge, the first controlled dietary intervention to test alterations in plasma α- and γ-tocopherol in response to eating pecans. The effects are modest compared with those reported by Cooney et al [32], who showed a marked increase in plasma γ-tocopherol in response to consumption of small quantities of sesame seed. It is postulated that the sesame effect is mediated by the activity of the sesame lignan sesamin.

The pecan diet did not influence fasting total antioxidant capacity measured by the FRAP or the TEAC assay. Plasma antioxidant capacity responds immediately, within 1 to 2 hours, after the consumption of antioxidant-rich foods such as tea [33,34], wine [35], or chocolate [36]. When determined by antioxidant capacity assays, extracts of pecans [37] and other nuts [38] compare favorably with berries, fruits, vegetables, or tea in phenolic content and antioxidant potential. However, consistent with our results, many [39-41] but not all [42,43] dietary intervention studies that tested enrichment of the diet with selected fruits and/or vegetables did not find significant difference in fasting plasma antioxidant capacity after several weeks of supplementation. One explanation for this discrepancy is the possible rapid metabolism and elimination of plant constituents with antioxidant activity. Currently, little is known about the homeostatic modulation of antioxidant capacity of body fluids and how these are influenced by the habitual diet [44]. The decrease in plasma TBARS is important because it indicates that tocopherols and polyphenols in pecans may be effective in inhibiting in vivo lipid peroxidation and degradation. The results are consistent with those of Actis-Goretta et al [45], who reported an 11% decrease in plasma TBARS concentrations in healthy individuals after 30 days of supplementation with a low-dose mixture of lipid-soluble antioxidants. Alternatively, plasma TBARS are correlated to the amount of linoleic acid in lipoprotein lipids [21], and the observed reduction may simply be a consequence of reduced plasma lipids achieved on the pecan diet [5]. The results should be viewed with caution because TBARS may not be a reliable indicator of oxidative stress and there is need for better measures of antioxidant protection.

In summary, using a controlled 8-week crossover intervention, we showed that the addition of pecans to a healthy diet can lead to improved antioxidant defenses by increasing cholesterol-adjusted γ-tocopherol and decreasing TBARS. Although highly unsaturated, a pecan-rich diet does not increase the degree of lipid peroxidation in the body and pecans serve as a valuable source of γ-tocopherol in the diet. These findings support the protective effects of nuts in reducing the risk of disease from previous epidemiologic studies.

Acknowledgment

This study was supported in part by a research grant from the National Pecan Shellers Association, Atlanta, Ga.

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