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# Nut Consumption and Risk of Hypertension in US Male Physicians

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## Abstract

**Background & Aims:** Hypertension is risk factor for cardiovascular disease and dietary factors may play an important role in its prevention. We sought to examine the association between nut consumption and incident hypertension.

**Methods:** Prospective cohort of 15,966 participants from the Physicians' Health Study I who were free of hypertension at baseline. Nut consumption was assessed using a simple abbreviated food questionnaire and hypertension was self-reported. We used Cox regression to estimate relative risks of hypertension according to nut consumption.

**Results:** During 237,585 person-years of follow up, 8,423 new cases of hypertension occurred. Compared to subjects who did not consume nuts, multivariable adjusted hazard ratios (95% CI) for hypertension were 0.97 (0.91-1.03), 0.98 (0.92-1.05), 0.96 (0.89-1.03), and 0.82 (0.71-0.94) for nut consumption of 1-2 times per month and 1, 2-6, and ≥ 7 times/week, respectively. In a secondary analysis stratified by body mass index, there was an inverse relation between nut intake and hypertension in lean subjects (p for trend 0.0019) but not in overweight or obese subjects (p for interaction 0.0037).

**Conclusion:** Our data suggest that nut consumption is associated with a lower risk of hypertension in US male physicians and that such relation may be influenced by adiposity.

### Keywords

Diet; hypertension; epidemiology; nuts; nutrition

## Introduction

Hypertension is the most common primary diagnosis in the US with about 35 million office visits[1] and is a major risk factor for stroke[2], myocardial infarction[3], heart failure[4], and chronic kidney disease[5]. The lifetime risk of developing hypertension is 90% (9 in 10) among adults between 55 and 65 years[6]. It has been recognized that modifiable lifestyle factors (low sodium intake[7], DASH diet[8], weight control[9], and exercise[10] among other factors) could play an

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important role in the prevention of hypertension. Consequently, the Seventh Report of the Joint National Committee on Prevention, detection, Evaluation, and Treatment of high blood pressure (JNC VII) recognized lifestyle management as an important factor in the prevention of hypertension[11]. Despite the availability of effective lifestyle factors and pharmacologic agents for the management of hypertension, only modest improvements have been achieved to date. Based on data from the National Health and Nutrition Examination Surveys (1976-2000), about 60% of people with hypertension are treated and only about a third of treated hypertensive subjects attain the treatment goals[12].

Nut consumption has been associated with a lower risk of type 2 diabetes and weight gain. Since obesity and abnormal glucose metabolism contribute to the development of hypertension, it is possible that nut consumption may lower the risk of hypertension. However, few studies have examined the effects of nuts on incident hypertension. Nuts are low in sodium and also contain a variety of nutrients including unsaturated fatty acids, minerals such as magnesium and potassium with possible beneficial effects on blood pressure[13,14]. In a food supplement trial, a Mediterranean diet enriched with nuts was associated with reduced blood pressure compared to a low-fat diet[15]. Because the difference in blood pressure between a diet with and without nuts was modest, it is important to estimate possible effects of nut consumption on blood pressure in other populations. The current study prospectively examined whether nut consumption is associated with a lower risk of incident hypertension. Since individuals who are overweight or obese may avoid nuts as a source of energy and fat, we also assessed whether overweight/obesity modified the association between nut consumption and hypertension.

## Materials and Methods

### Study population

We used data from the Physicians' Health Study (PHS) I – a completed randomized, double-blind, placebo-controlled trial designed to study low-dose aspirin and beta-carotene for the primary prevention of cardiovascular disease and cancer. Detailed description of the PHS I has been published previously[16]. Of the total 22,071 participants, we excluded 615 subjects because of missing data on nut consumption; 5,174 subjects with prevalent hypertension at the time of exposure assessment; and 316 people with missing covariates. Thus, a final sample of 15,966 participants was used for current analyses. Each participant gave written informed consent and the Institutional Review Board at Brigham and Women's Hospital approved the study protocol.

### Nut consumption

Information on nut consumption was self-reported using a simple abbreviated semi-quantitative food frequency questionnaire at 12 months post-randomization (1983-1985). Participants were asked the following: “Please indicate how often, on average, you have eaten each of the following foods during the past year”. “Nuts (small packet or 1 oz.)” Possible response categories included “rarely/never”, “1-3/month”, “1/week”, “2-4/week”, “5-6/week”, “daily”, and “2+/day”. Due to limited number of cases in some of the categories, we preserved the first 3 categories and combined adjacent pairs of categories to obtain stable estimates as previously published[17]. While the food frequency questionnaire was not validated in the Physicians' Health Study, it has been validated in several cohorts[18,19]. We did not include information on peanut butter in the exposure as peanuts are legumes.

### Ascertainment of hypertension in the PHS

Detailed description of hypertension ascertainment in the PHS has been published[20]. The diagnosis of hypertension was made based on self-reported treatment for hypertension and reported blood pressure values. For current analyses, hypertension was defined as systolic

blood pressure of at least 140 mm Hg or diastolic blood pressure of at least 90 mm Hg or treatment for hypertension. Accuracy of self-reported blood pressure/hypertension in the general population[21] or among physicians or female health professional[22] has been described elsewhere.

### Other variables

Demographic data were collected at baseline. Information on comorbidity (i.e. diabetes mellitus) has been collected through annual follow-up questionnaires. Data on selected foods such as fruits and vegetables, breakfast cereal with brand; physical activity; smoking; alcohol consumption; liver intake; low fat and whole milk; and history of hypercholesterolemia or treatment for hypercholesterolemia were obtained through self-reports at baseline. During the 12 month questionnaire, information on selected foods including fish, red meat, ice cream, cheese; chicken or turkey was collected. We assigned the mid-point of the response category as outlined for nuts above to each response category and sum up subcategories within each food group (i.e. low fat milk, yogurt, and cheese for dairy; dark meat fish, tuna, and other fish for fish; and beef, pork, lamb, and hotdogs for red meat). For "2+/day" category, we assigned a value of 14 times per week.

### Statistical analyses

We classified each subject into one of the following categories of nut consumption: none, 1-3 per month, 1 per week, 2-6 per week, and 7+ per week. We computed person-time of follow up from exposure assessment (12 months post-randomization) until the first occurrence of a) hypertension, b) death, or c) censoring date – date of receipt of last follow-up questionnaire. For baseline characteristics, p value for linear trend was obtained by fitting logistic regression for categorical variables and linear regression for continuous variables. We used Cox proportional hazard models to compute multivariable adjusted hazard ratios with corresponding 95% confidence intervals using subjects in the lowest category of nut consumption as the reference group. We assessed confounding by established risk factors for hypertension. The initial model only adjusted for age; a **final** model controlled for age (<45, 45-49, 50-54, 55-59, 60-64, 65-69, 70-74, 75+ y), body mass index (<25, 25-29, 30 kg/m$^2$), smoking (never, former, current smoker), alcohol consumption (<1, 1-4, 5-7, 8+ drinks/week), breakfast cereal (0, ≤ 1, 2-6, and 7+/week) and type; red meat (quintiles); fish (quintiles); dairy (quintiles); exercise (0, ≤ 1, 2-4, 5+/week), fruit and vegetable intake (<3, 3-4, 5-6, 7-13, 14+ servings per week), multivitamin use, treatment assignment, and history of diabetes and hypercholesterolemia or treatment for hypercholesterolemia. Assumptions for proportional hazard models were tested (by including main effects and product terms of covariates and logarithmic transformed time factor) and were met (all p values >0.05). To examine whether overweight/obesity modifies the association between nut intake and hypertension, we used BMI of 25 kg/m$^2$ as cut point to separate lean from overweigh/obese subjects. We then conducted stratified analyses by overweight status (BMI < 25 or ≥ 25 kg/m$^2$) and tested statistical interaction using a product term of nut consumption and BMI variable in a hierarchical model. We also conducted sensitivity analyses excluding subjects with prevalent diabetes mellitus (n=334) or those with follow-up time of less than 2 years (n=1495). In addition, we conducted stratified analysis by smoking (current smokers yes/no). All analyses were completed using SAS, version 9.1 (SAS Institute, NC). Significance level was set at 0.05.

## Results

Table 1 presents the baseline characteristics of 15,966 study participants according to nut consumption. The mean age was 52.3±8.9 years (range 40.7 to 87.1). Consumption of nuts was associated with a higher proportion of current drinkers, multivitamin use, fish, dairy, red meat, and breakfast cereal consumption. During the follow up, 8,423 new cases of hypertension

occurred. Compared to subjects who did not consume nuts, multivariable adjusted hazard ratios (95% CI) for hypertension were 0.97 (0.91-1.03), 0.98 (0.92-1.05), 0.96 (0.89-1.03), and 0.82 (0.71-0.94) for nut consumption of 1-2 times per month, 1 time/week, 2-6 times/week, and 7 + times/week, respectively (Table 2).

When stratified by the BMI, we observed an inverse association between nut consumption and hypertension among lean (BMI < 25 kg/m$^2$) but not overweight/obese subjects (Table 3). For lean subjects, multivariable adjusted hazard ratios (95% CI) were 1.0 (reference), 0.93 (0.86-1.01), 0.94 (0.86-1.03), 0.87 (0.79-0.96), and 0.77 (0.64-0.93) from the lowest to the highest category of nut consumption, respectively (p for trend 0.0019, Table 3). We did not observe an inverse association between nuts and incident hypertension for BMI ≥ 25 kg/m$^2$ (p for trend 0.39, Table 3). There was evidence for an interaction between BMI and nut consumption on the risk of hypertension (p for interaction 0.0037). Stratification by smoking did not alter the findings and there was no interaction between smoking status and nut intake on hypertension risk (p for interaction 0.16). Exclusion of subjects with prevalent diabetes or those with follow-up time below 2 years did not alter these findings (data not shown).

## Discussion

In this prospective cohort, we observed a lower incidence of hypertension with nut consumption among US male physicians. In addition, this association was mainly observed among lean subjects (BMI < 25 kg/m$^2$) and not in overweight or obese individuals. These findings are consistent with preliminary reports from the PREDIMED trial, which showed that compared with a low-fat diet, Mediterranean diets supplemented with nuts and olive oil were associated with 7.1 and 5.9 mm Hg reduction in systolic blood pressure after 3 months of intervention, respectively, among ~257 adult men and women per intervention group[15]. In that study, corresponding effects for diastolic blood pressure were 2.6 and 1.6 mm Hg for Mediterranean diet with nuts and olive oil[15], respectively. Because the Mediterranean diet with nuts was compared to either Mediterranean diet with olive oil or low-fat diet, it is difficult to estimate the individual contribution of nuts on blood pressure reduction in that trial.

In addition to direct effects of nuts on blood pressure, previous studies have reported beneficial effects of nut consumption on other major risk factors for hypertension. Data from the SUN Study[23] found that subjects who consumed nuts 2+ times per week had a 31% (OR: 0.69; 95% CI: 0.53-0.90) lower risk of weight gain (defined as adding 5+ kg of body weight during the follow up) than those who never ate nuts. Similarly, individuals who never ate nuts gained on average 424 g (95% CI: 102-746) more than frequent nut eaters after a median follow up of 28 months[23]. In addition, nut consumption has been associated with a lower risk of type 2 diabetes among female nurses in a dose-response fashion[24].

Though nut consumption has been shown to reduce the risk of cardiovascular disease and improve lipid profiles, concerns that intake of nuts (rich in fat) may lead to obesity remain in the general population. However, such concerns have not been substantiated by epidemiological data. For example, in the SUN study[23], the relative risk of becoming overweight/obese among subjects consuming nuts at least 2 times per week was 0.73 (0.48-1.11) compared with people who never ate nuts after adjustment for age, sex, baseline BMI, exercise, smoking, television viewing, and energy intake. Furthermore, there was no evidence for a statistically significant weight gain with Mediterranean diet supplemented with nuts compared with baseline weight[15]. What physiologic mechanisms could help establish a causal relation between nuts and hypertension?

Nuts are low in sodium and also contain a variety of nutrients including mono- and polyunsaturated fatty acids, minerals such as magnesium and potassium, fiber, antioxidants,

and vitamins with beneficial influence on blood pressure[13,14]. Sodium reduction has been shown to result in lower blood pressure[8]. Dietary fiber influences satiety and energy intake and could improve blood pressure through weigh reduction. A meta-analysis of 25 studies reported blood pressure lowering effects of dietary fiber, especially among hypertensive subjects[25]. Magnesium intake has been shown to lower blood pressure[26] by acting as a calcium channel blocker and causing vasodilation[27]. Magnesium stimulates prostacyclin production and nitric oxide synthesis and modulates endothelium-dependent and independent vasodilation[28]. However, results from clinical trials have been heterogeneous and inconsistent[29]. Other minerals contained in nuts could favorably influence blood pressure[14]. In the Multi-Ethnic Study of Atherosclerosis[30], nut and seed consumption was associated with lower levels of C-reactive protein, interleukin-6, and fibrinogen. This suggests that nuts may inhibit inflammation and prevent progression of atherosclerosis, an important factor in the development of hypertension. It is however unlikely that these possible physiologic mechanisms could have been differential in lean vs. overweight/obese subjects to explain the observed interaction between adiposity and nuts on the risk of hypertension in the present study. A plausible explanation is that overweight/obese subjects avoided nuts as energy-dense foods in order to manage their weight. Alternatively, overweight/obese subjects may have been more likely to underreport their nut consumption, thereby diluting any beneficial effect of nut consumption on blood pressure.

Our study has some limitations. Participants were male physicians who may have different behaviors or lifestyle habits than the general population, thereby limiting the generalizability of our findings. Nut consumption was assessed only once (12 months post-randomization) and since subjects may have changed their dietary habits, we were not able to account for such changes in our analyses. Since we used a simple questionnaire to assess nut consumption, we were unable to adjust for total energy intake and other nutrients consumed by study subjects. In addition, we did not have data on types of nuts consumed; their preparation including salted, spiced, roasted, or raw nuts to examine the influence of types of nuts or preparation method on the risk of hypertension. It is possible that some of the subjects were misclassified due to inaccurate recall on nut intake. However, since information on nuts was collected before the occurrence of hypertension, it is more likely that such misclassification was non-differential and led to an underestimation of the true relation between nuts and hypertension. Since study participants were physicians, it is less likely that consumption of salted nuts was important in this population given the positive association between sodium intake and hypertension. Lastly, in the absence of random allocation of nuts, we can not exclude unmeasured or residual confounding (i.e. over- or underreporting of nut consumption) as a possible explanation for observed findings. Nevertheless, the large sample size, the longer duration of follow up, and the complete ascertainment of outcome via standardized follow-up questionnaires are strengths of the present study.

In conclusion, our data suggest that frequent consumption of nuts is associated with a lower risk of incident hypertension in US male physicians, and such relation appears to be limited to lean individuals. Further examination of the relation between nuts and incident hypertension in the general population and differential effects of types of nuts consumed is warranted.

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**Table 1**

Baseline characteristics of 15,966 US male physicians according to nut consumption

| | 0 (N=3,109) | 1-3 times /month (N=5,778) | 1 time /week (N=3,809) | 2-6 times /week (N=2,823) | ≥ 7 times /week (N=447) | P for linear trend |
|---|---|---|---|---|---|---|
| Age (y) | 53.8±9.2 | 53.0±8.7 | 53.1± 8.8 | 53.3±8.8 | 54.7±9.2 | 0.67 |
| Body mass index (kg/m$^2$) | 24.5±2.8 | 24.5±2.6 | 24.6±2.6 | 24.4±2.5 | 24.0±2.4 | 0.015 |
| Fruits & vegetables (serving/d) | 1.1±0.7 | 1.1±0.7 | 1.1±0.7 | 1.2±0.7 | 1.3±0.8 | <0.0001 |
| Dairy (servings/week) | 3.4±2.9 | 3.7±2.7 | 3.9±2.7 | 4.5±3.0 | 5.2±4.0 | <0.0001 |
| Fish (servings/week) | 1.9±1.8 | 2.0±1.7 | 2.1±1.7 | 2.1±1.8 | 2.4±2.6 | <0.0001 |
| Red meat (servings/week) | 5.2±3.3 | 5.6±3.3 | 5.8±3.3 | 5.7±3.3 | 5.6±3.8 | <0.0001 |
| Current smoking (%) | 12.5 | 10.5 | 9.8 | 10.1 | 11.4 | 0.98 |
| Current drinking (%) | 69.8 | 73.9 | 75.8 | 75.6 | 74.5 | <0.0001 |
| Current use of multivitamin (%) | 18.8 | 17.6 | 18.8 | 20.2 | 24.5 | 0.003 |
| Hypercholesterolemia (%)* | 10.4 | 9.7 | 10.8 | 10.6 | 10.8 | 0.34 |
| Assigned to aspirin (%) | 50.9 | 49.4 | 49.2 | 50.4 | 51.5 | 0.94 |
| Diabetes mellitus (%) | 2.1 | 1.9 | 2.0 | 2.3 | 3.4 | 0.18 |
| Current exercise (%) | 83.2 | 87.8 | 89.2 | 89.4 | 87.7 | <0.0001 |
| Breakfast cereal intake (%) | 64.9 | 72.0 | 75.8 | 76.1 | 75.8 | <0.0001 |

Data are presented as mean ± standard deviation or percentages.

* 2103 subjects had missing data on hypercholesterolemia.

**Table 2**

Hazard ratios (95%CI) for hypertension according to nut consumption

| Nut intake | Cases/person-years | Hazard ratios (95% CI) | | |
|---|---|---|---|---|
| | | Age-adjusted | Model 1* | Model 2† |
| None | 1676/4432 | 1.0 | 1.0 | 1.0 |
| 1-3/month | 3036/86878 | 0.95 (0.89-1.01) | 0.95 (0.90-1.01) | 0.97 (0.91-1.03) |
| 1/week | 2025/56635 | 0.96 (0.90-1.03) | 0.96 (0.90-1.03) | 0.98 (0.92-1.05) |
| 2-6/week | 1477/42612 | 0.92 (0.86-0.99) | 0.93 (0.87-1.00) | 0.96 (0.89-1.03) |
| ≥ 7/week | 209/6828 | 0.78 (0.67-0.90) | 0.79 (0.69-0.92) | 0.82 (0.71-0.94) |
| P for trend | | 0.003 | 0.0096 | 0.014 |

*Adjusted for age (<45, 45-49, 50-54, 55-59, 60-64, 65-69, 70-74, 75+ y), body mass index (<25, 25-29, 30 $kg/m^2$), smoking (never, former, current smoker), alcohol consumption (<1, 1-4, 5-7, 8+ drinks/week), and history of diabetes.

†Additional control for exercise (0, ≤ 1, 2-4, 5+/week), fruit and vegetable intake (<3, 3-4, 5-6, 7-13, 14+ servings/week), breakfast cereal (0, ≤ 1, 2-6, and 7+/week) and type (whole grain vs. refined); red meat (quintiles); fish (quintiles); dairy (quintiles); multivitamin use, treatment assignment, and history of hypercholesterolemia/treatment for hypercholesterolemia.

**Table 3**

Hazard ratios (95% CI) for hypertension according to nut consumption and overweight/obesity

| Nut intake | BMI < 25 kg/m$^2$ | | BMI ≥ 25 kg/m$^2$ | |
|---|---|---|---|---|
| | Cases/person-years | Hazard ratio (95% CI)* | Cases/person-years | Hazard ratio (95% CI)* |
| None | 938/28312 | 1.0 | 738/16320 | 1.0 |
| 1-3/month | 1670/55758 | 0.93 (0.86-1.01) | 1366/31120 | 1.02 (0.93-1.11) |
| 1/week | 1098/36181 | 0.94 (0.86-1.03) | 927/20454 | 1.04 (0.94-1.15) |
| 2-6/week | 836/28545 | 0.87 (0.79-0.96) | 641/14067 | 1.07 (0.96-1.20) |
| ≥ 7/week | 132/4925 | 0.77 (0.64-0.93) | 77/1904 | 0.90 (0.71-1.14) |
| P for trend | | 0.0019 | | 0.39 |
| P for interaction | | 0.0037 | | |

*Adjusted for age (<45, 45-49, 50-54, 55-59, 60-64, 65-69, 70-74, 75+ y), body mass index (<25, 25-29, 30 kg/m$^2$), smoking (never, former, current smoker), alcohol consumption (<1, 1-4, 5-7, 8+ drinks/week), exercise (0, ≤ 1, 2-4, 5+/week), fruit and vegetable intake (<3, 3-4, 5-6, 7-13, 14+ servings/week), breakfast cereal (0, ≤ 1, 2-6, and 7+/week) and type, red meat (quintiles), fish (quintiles), dairy (quintiles); multivitamin use, treatment assignment, and history of diabetes and hypercholesterolemia/treatment for hypercholesterolemia.