# Coronary artery calcification and dietary cholesterol intake in Korean men

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**Objective —** This study was performed to examine the relationship between dietary cholesterol intake and coronary artery calcification (CAC) score in healthy men.

**Methods —** Electron beam computed tomography (EBCT) was used to examine the CAC score in 135 Korean men aged 40-81 years who did not have clinical illness. Dietary cholesterol intake was assessed by a nutritionist using a semiquantitative food frequency method. Body mass index (BMI), serum lipid levels, cigarette use, alcohol intake, exercise, and a past history of cardiovascular disease were determined during interview and examination.

**Results —** The resultant median CAC score among those who experienced CAC was 22.5 (1-697) and average intakes of total fat and cholesterol were 22.4% (13.8-40.7) of total energy intake and 306.0 mg/day (84-1191). When the participants were classified into high (≥ 75 percentile) and low (< 75 percentile) CAC score groups, multiple logistic analysis showed that the cholesterol intake (per 10 mg/1000 kcal of energy) was significantly related to a high CAC score (OR 1.12: 95% CI 1.02 – 1.24), after adjustment for age, BMI, serum triglyceride level, past history of hypertension, past history of hyperlipidaemia, and energy intake. Also, when participants were classified into 2 groups (CAC score ≥ 100 vs. < 100), cholesterol intake was found to be significantly related to CAC score. However, fatty acid intakes were not significantly related to the CAC score.

**Conclusion —** These results suggest that in a population with a relatively low risk of coronary heart disease, higher cholesterol intake may increase the level of CAC. *(Acta Cardiol 2002; 57(1): 5-11)*

**Keywords:** *electron beam computed tomography – coronary artery calcification – cholesterol intake.*

## Introduction

The great majority of patients has symptomatic coronary artery disease (CAD), but there are many other adults with lower degrees of atherosclerosis affecting their coronary arteries, and therefore, there is a need to identify individuals at risk of premature cardiovascular disease, so that preventive measures may be instituted before CAD occurs. Currently, we are able to use non-invasive measures to evaluate the early process in asymptomatic adults and to examine the relationship with established cardiovascular risk factors. Coronary artery calcium detection by electron beam computed tomography (EBCT) is one of these noninvasive methods for early CAD detection[1,2].

EBCT is a highly sensitive, noninvasive, and relatively inexpensive technique for detecting coronary artery calcium and has been used with increasing frequency in the United States and other countries over the past 10 years. It is chiefly used to screen people with a high risk of developing coronary heart disease (CHD) and to diagnose obstructive coronary artery disease. EBCT has great potential as a method for further determining risk, particularly in elderly asymptomatic patients, and others at intermediate risk[3-6].

The prevalence and average score of coronary artery calcification (CAC) in the general population

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have already been reported in the West[3-6]. However, dietary risk factors related to CAC score were not studied in these reports. Recently, the distribution of CAC scores and risk factors related to CAC in healthy adults and non-insulin-dependent diabetes mellitus patients were reported in Korea[7,8] and reports about the importance of the dietary patterns of CHD patients have also appeared in Korea[9-12], but no research has been conducted to date on the determination of CAC-related dietary factors among the general population by EBCT.

In this study, we investigated the association between dietary intake and CAC scores in healthy Korean men.

## Methods

During the period November 1997 to September 1998, 135 male subjects without any known disease including CHD were selected from a University teaching hospital in Seoul, Korea. EBCT was performed with an Imatron C-150 ultrafast computed tomographic scanner (Imatron XP-150, S. San Francisco, CA). Forty contiguous slices of 3-mm thickness were obtained during a single breath-hold, starting at the lower edge of the carina. The scan time was 100 ms per slice, with synchronized ECG triggering at 80% of the R-R interval. CAC scores were calculated according to the method proposed by Agatston et al.[13]

A trained interviewer systematically questioned the following: sociodemographic characteristics (age, marital status, education, and income), cigarette smoking, alcohol consumption, physical exercise, and past disease history (stroke, hypertension, hyperlipidaemia, and diabetes mellitus). Leisure-time physical exercise was measured by asking the type and frequency of exercise performed weekly. Cigarette smoking history was assessed by questions about current or past use of cigarettes, and to be classified as an ex-smoker, a patient had to have reported quitting smoking at least 1 year prior to the time of interview. Questions about alcohol intake assessed the frequency of drinking per week and the amount of alcohol consumed.

Height and weight were measured and body mass index (BMI) was calculated by dividing weight (kg) by height (m) squared. Serum total cholesterol and triglyceride were determined by the enzymatic method (Hitachi 736-40, Japan), and HDL cholesterol was measured by the precipitation method with dextran sulfate magnesium chloride.

Dietary data were obtained by a nutritionist using a semiquantitative food frequency questionnaire (FFQ). We developed our own FFQ which contains 93 food items which are generally consumed in Korea on a daily basis, and which contain substantial amounts of total fat, fatty acids, and cholesterol. Each subject was asked to report the usual frequency of consumption and the usual portion size during the previous year. Consumption frequency was measured on a 9-grade scale: never, once a month, 2 times per month, once per week, 3 times per week, 5 times per week, once a day, 2 times per day, 3 times per day. The questionnaire also included a specific question about the type of fat used for cooking. This information was also used in the recipes of the mixed dishes. During the interview, food models and reference utensils were shown to subjects to help them estimate portion size. The participants who had substantially changed their dietary pattern during the preceding year were excluded from the analysis.

Nutrient intake based on this information was calculated using Korean food composition tables, while fatty acids and cholesterol were calculated according to other published data[14-16]. To calculate the daily nutrient intake, the nutrient content of each food item was multiplied by the frequency of its daily consumption and all items summed. Calculated dietary information included the individual's daily intake of energy, total fat, protein and carbohydrates (CHO) as well as saturated fatty acid (SFA), monounsaturated fatty acid (MUFA), polyunsaturated fatty acid (PUFA) and cholesterol. The dietary method adopted was validated by a pilot study, which was carried out on 78 subjects (31 men, 47 women) who visited the hospital for health screening. FFQ was compared with the 3-day dietary record. The results showed that the method provided a reasonable measure of cholesterol, total and specific types of fat. The unadjusted correlations between FFQ and diet record were 0.44 for total fat, 0.48 for SFA and 0.36 for cholesterol.

The CAC score was categorized into high (≥ 75 percentile) and low (< 75 percentile) CAC score groups, and age, BMI, lifestyle variables, blood lipid profiles and nutrient intakes of high (CAC Score ≥ 13) and low CAC score (CAC Score < 13) group were compared using two-sample t-tests or $\chi^2$-tests. The association between cholesterol intake and the risk of higher CAC score was determined by multiple logistic regression analysis. In a previous publication[17], it has been reported that the subtypes of fat (PUFA, SFA) are related to CAD or CHD, Therefore, we compared 2 multivariate analyses that examined the relation between cholesterol intake and the risk of a high CAC score. Model 1 controlled for cholesterol and energy intake, but not for fat subtype, and model 2 controlled for the variables included in model 1 and for SFA and PUFA. Data analysis was performed using statistical analysis system (SAS) software (version 8.1, SAS Institute Inc, Cary, NC).

## Results

The number of participants with a CAC score greater than 0 and more than 100 was 56 (41.5%), 12 (8.9%), respectively, and the median CAC score among

those who experienced CAC was 22.5 (range: 1-697) (Figure 1).

Table 1 shows the crude nutrient intake and nutrient intake as a percentage of total energy. The average daily energy intake was 2,117 kcal and average cholesterol intake was 306 mg. When macronutrient intake was expressed as a percent of energy intake, the total fat intake was 22.4% and the energy composition of carbohydrates, protein and fat was 59.8%, 15.5% and 22.4%, respectively (Table 1). The average daily intakes of PUFA, MUFA, and SFA were 11.4 g, 19.2 g, and 16.5 g, respectively, and the ratio of polyunsaturated fatty acids/saturated fatty acids (P/S) was 0.7. PUFA, MUFA and SFA accounted for 4.7%, 7.9%, and 6.8% of energy intake, respectively. In terms of PUFA, n-6 fatty acid and n-3 fatty acid were 9.7 g and 1.6 g per day, respectively, and the ratio of n-6/n-3 fatty acid was 6.2.

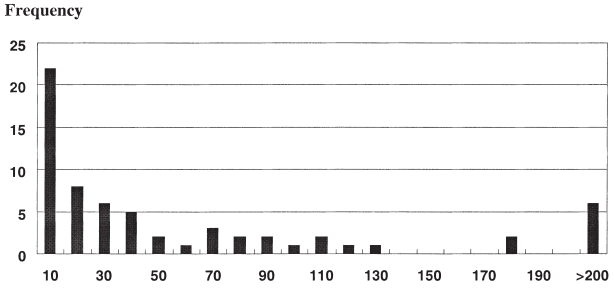


Fig. 1. – Frequency distribution of the coronary artery calcification (CAC) score.

Participants were classified into high (≥ 75 percentile) and low (< 75 percentile) CAC score groups. Table 2 shows the distributions of age, BMI, and blood lipid profiles according to the CAC score group. Age was significantly higher in the higher CAC score group, and the serum triglyceride level of this group was also higher, but this was of marginal significance. BMI, serum total cholesterol, HDL and LDL levels were not significantly different in the two groups.

Table 3 shows the distributions of lifestyle variables, namely, cigarette smoking, alcohol consumption, physical exercise and past history of disease (hypertension, hyperlipidaemia) according to CAC score group. The proportions of subjects with a past history of hypertension or a past history of hyperlipidaemia were significantly higher in the high CAC group than in the low CAC group. No significant differences were found in

Table 1. – *Mean intakes and mean percentage of energy from specific nutrients*

| | Crude intakes (n = 135) Mean ± SD | Intakes as % of energy (n = 135) Mean ± SD |
|---|---|---|
| Energy (kcal) | 2116.9 ± 479.2 | – |
| Carbohydrates (g) | 313.1 ± 59.8 | 59.8 ± 6.3 |
| Protein (g) | 82.9 ± 25.3 | 15.5 ± 2.1 |
| Total fat (g) | 54.0 ± 21.8 | 22.4 ± 4.8 |
| PUFA (g) | 11.4 ± 4.9 | 4.7 ± 1.2 |
| n-6 fatty acid (g) | 9.7 ± 4.2 | 4.0 ± 1.1 |
| n-3 fatty acid (g) | 1.6 ± 0.8 | 0.7 ± 0.2 |
| MUFA (g) | 19.2 ± 9.8 | 7.9 ± 2.5 |
| SFA (g) | 16.5 ± 8.2 | 6.8 ± 2.2 |
| P/S | 0.7 ± 0.2 | – |
| n-6/n-3 | 6.2 ± 1.7 | – |
| Cholesterol (mg) | 306.0 ± 163.9 | – |

SD = standard deviation; PUFA = polyunsaturated fatty acid; MUFA = monounsaturated fatty acid; SFA = saturated fatty acid; P/S = polyunsaturated fatty acid/saturated fatty acid.

Table 2. – *Age, body mass index, and serum lipid profiles according to CAC score*

| | CAC score ≥ 13 (n = 34) Mean ± SD | CAC score < 13 (n = 101) Mean ± SD |
|---|---|---|
| Age (years) | 59.9 ± 6.3 | 54.2 ± 6.8*** |
| Body mass index (kg/m$^2$) | 24.4 ± 2.5 | 23.7 ± 2.3 |
| Serum log triglyceride (mmol/l) | 0.59 ± 0.50 | 0.36 ± 0.49* |
| Serum cholesterol (mmol/l) | 5.6 ± 0.7 | 5.3 ± 0.8 |
| Serum HDL (mmol/l) | 1.2 ± 0.3 | 1.2 ± 0.3 |
| Serum LDL (mmol/l) | 3.4 ± 0.7 | 3.4 ± 0.7 |

*p < 0.1, **p < 0.05, ***p < 0.01.
CAC = coronary artery calcification; SD = standard deviation; HDL = high-density lipoprotein; LDL = low-density lipoprotein.

Table 3. – *General characteristics according to CAC score*

| | | CAC ≥ 13 (n = 34) No (%) | CAC < 13 (n = 101) No (%) | p value |
|---|---|---|---|---|
| Education | High school | 6(17.7) | 23(22.8) | 0.529 |
| | University | 28(82.4) | 78(77.2) | |
| Income | < $3,000 | 9(26.5) | 38(37.6) | 0.377 |
| | $3,000-4,000 | 6(17.7) | 20(19.8) | |
| | ≥ $4,000 | 19(55.9) | 43(42.6) | |
| Past history of hypertension | Yes | 20(58.8) | 29(28.7) | 0.002 |
| | No | 14(41.2) | 72(71.3) | |
| Past history of hyperlipidaemia | Yes | 9(26.5) | 12(11.9) | 0.042 |
| | No | 25(73.5) | 89(88.1) | |
| Smoking | Current smoker | 9(27.3) | 32(31.7) | 0.125 |
| | Ex-smoker | 18(54.6) | 36(35.6) | |
| | Never smoker | 6(18.2) | 33(32.7) | |
| Drinking | Current drinker | 28(84.9) | 79(78.2) | 0.619 |
| | Ex-drinker | 3( 9.1) | 10( 9.9) | |
| | Never drinker | 2( 6.1) | 12(11.9) | |
| Exercise | Regular | 22(66.7) | 58(57.4) | 0.347 |
| | Irregular | 11(33.3) | 43(42.6) | |

CAC = coronary artery calcification.

Table 4. – *Mean intakes and mean percentage of energy from specific nutrients according to CAC score*

| | CAC ≥ 13 (n = 34) Mean ± SD | CAC < 13 (n = 101) Mean ± SD |
|---|---|---|
| Energy (kcal) | 1993.1 ± 462.8 | 2158.5 ± 479.6* |
| Carbohydrates (% of energy) | 60.6 ± 5.7 | 59.6 ± 6.5 |
| Protein (% of energy) | 15.7 ± 1.6 | 15.5 ± 2.2 |
| Fat (% of energy) | 21.4 ± 4.8 | 22.7 ± 4.8 |
| PUFA (% of energy) | 4.7 ± 1.0 | 4.7 ± 1.3 |
| MUFA (% of energy) | 7.7 ± 2.3 | 8.0 ± 2.6 |
| SFA (% of energy) | 6.3 ± 1.8 | 7.0 ± 2.3 |
| P/S | 0.8 ± 0.2 | 0.7 ± 0.2 |
| Cholesterol (mg/1000 kcal) | 147.3 ± 54.0 | 137.6 ± 52.7 |

*p < 0.1.
SD = standard deviation; PUFA = polyunsaturated fatty acid; MUFA = monounsaturated fatty acid; SFA = saturated fatty acid; P/S = polyunsaturated fatty acid/saturated fatty acid; CAC = coronary artery calcification.

Table 5. – *Odds ratio and 95% confidence interval of CAC score for cholesterol intake (n = 135)*

| | Model 1 | Model 2 |
|---|---|---|
| Age (years) | 1.18(1.08-1.29) | 1.17(1.07-1.28) |
| BMI (kg/m$^2$) | 1.28(1.02-1.62) | 1.28(1.01-1.62) |
| Past history of hypertension | 1.37(0.50-3.73) | 1.42(0.51-3.98) |
| Past history of hyperlipidaemia | 2.31(0.68-7.84) | 2.44(0.67-8.84) |
| Serum log triglyceride (mmol/l) | 3.31(1.21-9.05) | 3.09(1.08-8.88) |
| Energy (Kcal) | 1.00(1.00-1.00) | 1.00(1.00-1.00) |
| PUFA (% of energy) | | 1.03(0.62-1.73) |
| SFA (% of energy) | | 0.80(0.57-1.12) |
| Cholesterol (10 mg/1000 kcal) | 1.12(1.02-1.24) | 1.02(1.003-1.03) |

CAC = coronary artery calcification; BMI = body mass index; PUFA = polyunsaturated fatty acid; SFA = saturated fatty acid.

smoking status, alcohol consumption, physical exercise, education and income level between the two groups.

Table 4 shows energy, total fat, PUFA, SFA and cholesterol intakes according to the CAC score group. The energy intake of the low CAC score group was higher than that of the high CAC score group. Total fat, PUFA, and SFA intakes were not significantly different. The intake of cholesterol was higher in the high CAC score group, but this was not significant.

Multivariate analysis results are shown in Table 5. Taking other risk factors (age, BMI, past history of hypertension, past history of hyperlipidaemia, serum triglyceride and energy intake) into consideration (in model 1), the odds ratio (OR) of cholesterol intake with CAC score was 1.12 (95% CI 1.02-1.24). After additionally controlling for subtypes of fat (SFA and PUFA), the association between the intake of cholesterol and the CAC score was found to be significantly positive (in model 2). Also, when the participants were classified into 2 groups (CAC score ≥ 100 vs < 100), the cholesterol intake was found to be significantly related to the CAC score after adjustment for non-dietary variables and energy intake (OR 1.17: 95% CI 1.04 – 1.33) (data not shown). When PUFA and SFA were additionally controlled, the above association was retained (OR 1.02: 95% CI 1.003 – 1.03). The intakes of SFA or PUFA and the CAC score were not significantly associated.

## Discussion

In this study, 58.5% of the participants had a CAC score of zero, and only 8.9% had a score of more than 100. Detrano et al.[6] studied 501 symptomatic patients, of which 1.8% died and 1.2% had non-fatal MI during a mean follow-up period of 31 months. A threshold of 100 or greater in the CAC score was proven to be highly predictive for identifying those patients likely to experience cardiac events during follow-up. The participants of this study showed a lower rate of CAC progress than those in studies conducted in the United States[18,19]. Choe et al.[7] also reported that the prevalence of CAC or the average CAC score of healthy middle-aged men and women in Korea was lower than that of the United States by 10-20 years in all age groups of both sexes.

The average total fat intake of the participants was 22.4% (as a % of total energy intake), and was in the range (20-25%) recommended by the Korean Nutrition Society[15]. This level of total fat intake was a little higher than the previously determined mean total fat intake of Koreans (19% of energy intake)[20], but was much lower than that quoted by Western countries (34-45% of energy intake)[21,22]. In terms of fatty acid intakes, the SFA intake was 6.8% of the energy intake, which is a half of that reported in Western countries (11-16% SFA of energy intake). Also, the average intake of cholesterol was 306 mg, and 43.7% consumed more than 300 mg/day (the recommended level of the Korean Nutrition Society[15] and USA National Cholesterol Education Program[23]).

In this study, cholesterol intake was found to be positively related to the CAC score (OR 1.12, 95% CI 1.02-1.24). After additionally adjusting for SFA and PUFA, the association between cholesterol intake and the CAC score proved to be consistent. In addition, when the participants were classified into 2 groups about a CAC score of 100 (CAC score ≥ 100 vs. < 100), which is sometimes used as a standard for high risk[2], the cholesterol intake was found to be significantly associated with the CAC score. The Leiden Intervention Study[24] provided the first direct evidence that dietary modifications (P/S ratio: 2, cholesterol intake < 100 mg/day) can influence the natural course of coronary artery atherosclerosis. Subsequent angiographic trials including the Life Style Heart Trial[25], and the St. Thomas' Atherosclerosis Regression Study[26], showed that lifestyle modifications including dietary changes (chiefly total fat and cholesterol intake reduction), smoking cessation, weight control, and physical activities can reduce the progression of coronary artery atherosclerosis. In particular, Markus et al.[27] reported that reducing dietary cholesterol intake by 100 mg/day on average, would reduce the annual rate of carotid wall intima-media thickness (IMT) progression by 0.028 mm/year. In Korea, no report has been issued concerning CAC-related dietary factors among symptomatic and asymptomatic subjects, but the following reports about dietary factors, which are related to CHD, have been presented. Patients with an angiographically documented narrowing of more than 50% of the coronary artery experienced significant regression of coronary atherosclerosis when they followed a strict lifestyle modification programme (less than 15% fat of energy intake and less than 100 mg/day of cholesterol)[11]. Choi et al.[12] reported that well-planned nutrition counselling could reduce the risk of cardiovascular disease by improving dietary behaviour, lipid profiles, and antioxidant status in CVD patients. In addition, it has also been reported that the degree of narrowing of coronary vessels or the number of coronary vessels with narrowing is associated with higher cholesterol intake, and is not associated with fatty acid intakes[9,10].

Nevertheless, it has been reported that fatty acid intake is associated with CAC. The St. Thomas' Atherosclerosis Regression Study[28] and the ARIC study[29] reported that coronary atherosclerosis is positively related with total fat (especially SFA) and the Keys' score, and negatively associated with PUFA. The Cholesterol Lowering Atherosclerosis Study[30] showed that increases in lauric acid, oleic acid and linoleic acid intakes, are associated with the development of new lesions in coronary arteries. However, in our study, the

subtype of fat (SFA, PUFA, and MUFA) and the individual fatty acids were not found to be related to the CAC score.

If dietary risk factors are excluded, age and gender are the most important risk factors of CAC[5,31,32]. Several investigators have investigated the relationship between risk factors and CAC. Elevated serum cholesterol has shown a consistent association with CAC. Obesity, elevated blood pressure, diminished HDL cholesterol, cigarette smoking, diabetes, and elevated triglyceride have also been proven to be associated with CAC[5,31,33]. In the univariate analysis of our study, age, serum triglyceride level, past history of hypertension, and past history of hyperlipidaemia showed an association with the CAC score. However, multiple logistic analysis revealed that only age, BMI and serum triglyceride level were significantly related to the CAC score.

Although participants in the present study did not have any clinical illness and the absolute intakes and ratios of most of the nutrients fell into the "normal" category, the dietary cholesterol intake and the degree of CAC were significantly associated. This result suggests that the importance of dietary intake (especially cholesterol) should be emphasized in a population with a relatively low risk of CHD in order to reduce the incidence of coronary atherosclerosis.

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