



Effects of a calcium supplement on serum lipoproteins, apolipoprotein B, and blood pressure in overweight men

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ABSTRACT

Background: Calcium supplements can improve hypertension, hyperlipidemia, and regulation of body weight in overweight persons.

Objectives: The aim of the study was to assess the effect of calcium supplementation on blood pressure and serum lipid profile.

Materials and Methods: 49 overweight men (age range = 34.4 ± 4.8 , BMI = 27.5 ± 1.7) were given a placebo or carbonate calcium (1,250 mg elemental calcium) daily for 8 weeks in a randomized, double-blind clinical trial. Serum lipids and blood pressure were measured before and after the trial. A 24-hour dietary recall questionnaire was completed for each person at baseline, in the 4th week, and at the end of the study.

Results: There were no significant differences in weight, serum triglycerides, and HDL-c between the experimental and control groups at the end of the study, but there were significant differences in the mean changes of total cholesterol (12.9 versus 1%, $P < .001$), LDL-c (8.5 versus 1.6%, $P = .003$), and apolipoprotein B (7.2 vs. 0.18%, $P = .044$) in the experimental versus control groups at the end of the study. Additionally, in the experimental group, mean changes of systolic blood pressure had reduced significantly compared to the placebo group (6.84 vs. 3.05%, $P = .046$).

Conclusions: The results suggest that calcium supplements may have a favorable effect on reducing cardiovascular risk in overweight men.

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► Implication for health policy/practice/research/medical education:

This article has implication for health policy, research and medical education because the mortality due to cardiovascular disease is increasing, on the other hand we have low calcium intake in our society and this article can be a good article for Health policy, research and medical education.

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1. Background

Overweight and obesity, and their adverse effects such as diabetes, are some of the most important dietary problems worldwide (1, 2). Despite very impressive recent advances in coronary risk factor identification and modification, heart attack remains the most common

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cause of death in developed and developing societies (3). Major risk or even causative factors for heart attack, as well as other cardiovascular diseases, are elevated blood pressure, lipid profile and obesity (4). It seems reasonable to concentrate more on the effects of dietary habits and supplements on modifying hyperlipidemia because treating the adverse effects of this problem such as insulin resistance and atherosclerosis are much more expensive, and yet the results are still unsatisfactory (5, 6). Thus, finding dietary supplements to reduce these risk factors has been one of the most important fields of medical research (7-9). It has been established over a century that some elements such as calcium and magnesium not only can interact directly with lipid absorption, but also can bind to bile acids and increase their excretion and lower serum cholesterol when they are consumed more than the intestine can absorb (10, 11). Other newer studies have also shown similar results (12). These hypolipidemic effects may be beneficial for individuals with coronary heart disease (CHD). For example in a study in Iowa, the mortality rate in women in the highest percentile of calcium intake was a third of that of other women (13). Another study also has reported a strong inverse relationship between calcium intake and CHD (14). Weight loss has been shown to be the most efficient way to reduce the risk of CHD (15); even small amounts of weight loss can modify blood pressure, lipid profiles, and glucose intolerance significantly. Some studies have shown that calcium can help people lose weight (16). Other studies have demonstrated that an increase in dietary calcium in American society could result in the loss of adipose mass (17). Another study showed that calcium supplements can explain a larger percentage of weight loss than diet (18, 19). Simply put, an increase in calcium intake has been reported to be beneficial for reducing weight and regulating the serum lipid profile in most of studies (20, 21). There are articles that deny such relationships for individuals in certain situations (22, 23), but both of these studies had limitations in sample size, general methodology, or the use of an insufficient calcium supplement dose to change serum lipoproteins. On the other hand, there are much less evidence about the relationship between calcium intake and the level of apolipoproteins; furthermore, no such studies have been conducted on the Iranian population.

2. Objectives

Thus, there is a real need to conduct precise, controlled studies of individuals with different diets in other cultures (especially in Iran, where the rates of overweight and obesity are increasing and CHD due to increased body fat has a high mortality rate). The aim of this study is to investigate the potential effects of calcium supplementation on serum lipoproteins, apolipoprotein B, and blood pressure in moderately obese Iranian men.

3. Materials and Methods

3.1. Subjects

In this parallel, double-blind, randomized clinical trial, 82 overweight but otherwise healthy male workers aged 25 to 35 years from the shipwrights company, Bandar-e-Anzali, Iran, were recruited.

Inclusion criteria were a serum cholesterol level of less than 240 mg/dl, a serum triglyceride level of less than 400 mg/dl, and a body mass index (BMI) of more than 25 kg/m². Any subject with diabetes or any kind of liver, thyroid, or kidney disorder was excluded from the study. None of the individuals were taking medications such as calcium supplements, nonsteroidal anti-inflammatories, antihypertensive medication, lipid-lowering drugs, diuretics, or any other drug that affects the lipid metabolism for at least 6 months. After the initial screening, 54 subjects who met all of our criteria were included in this study. All individuals remained on their normal diets (without a weight-reduction diet) and with no changes in lifestyle. All participants were informed about the process of the trial and that they were free to leave the study whenever they wanted. Forty-nine subjects (25 experimental and 24 controls) successfully completed the study.

3.2. Calcium supplement

The calcium supplement and the placebo were provided by Amin pharmaceutical company, Tehran, Iran as 625-mg capsules. The placebo contained dextrose, and the supplement contained 250 mg of elemental calcium in the form of calcium carbonate because of its higher bioavailability, low cost, and availability compared to other calcium supplements.

3.3. Study design

The participants were divided randomly (by random-number tables) into case and placebo groups. In the beginning, all subjects received an identification number. The capsules were distributed weekly, and subjects were prescribed 5 capsules daily (one with each meal, one between breakfast and lunch, and one at night time). The subjects had to pick up their capsules each week from the local clinic. No other changes were made to their lifestyles or diets. This protocol ended after 8 weeks.

3.4. Data gathering and Information

Weight and height were taken by an accurate scale (Seca, Germany) and a standard measuring tape, respectively, and then BMI was calculated. Waist circumference was measured midway between the lowest rib and the iliac crest with no garments included in the measurement. Hip circumference was measured in undergarments at the place of the largest circumference around the buttocks (24). Blood pressure was checked with a mercury

monometer (Richter, Germany) at the beginning and the end of the study, twice at each time point in 30-minute intervals, and the mean of the four values was recorded as the patient's blood pressure. Physical activity was assessed by the International Physical Activity Questionnaire (25). A questionnaire was designed and used to obtain demographic information such as age, marital status, education level, smoking history, and other medical history. At the beginning and the end of the fourth and eighth weeks, a 24-hour dietary recall was used (26). Dietary calcium, lipid and protein consumption, and total energy intake were analyzed with Nutrition III software (26). All laboratory tests were conducted at a single Medical Diagnostic Laboratory in the beginning and the end of the study. Two 10-cc blood samples were obtained by two technicians and a physician after 12 to 14 hours of fasting and then centrifuged by 1,000 round/min to separate the plasma. Total cholesterol levels, HDL-c, LDL-c, triglycerides, apolipoprotein B, and calcium were measured as follows: total cholesterol (TC) was assayed using enzymatic CHOD-PAP, and laboratory kits were provided by Pars Azmoon Inc., Tehran, Iran. The base test was run with cholesterol oxidase and peroxidase enzyme and their reaction with aminoantipyrin, a color reagent, and then calculating the concentration by the means of the color intensity. HDL-c was assayed with the same method. At first, very low density lipoprotein (VLDL) and low-density lipoprotein (LDL) were excluded from HDL-c with antihuman-B lipoprotein.

Triglyceride (TG) was also assayed by the same method. All subjects had a TG level of less than 400 mg/dl. To measure the LDL-c correctly, the Friedwald equation was used; specifically, apolipoprotein B (apo-B) was measured by immunoturbidometric assays (Pars Azmoon Inc., Tehran, Iran) based on changes of the solution to filter light before and after the reaction between antigen and goat antihuman apo-B antibody, which is specific for apo-B. Calcium was measured by a colorimetric

method using the Cresolphthalein complex and special kits (Pars Azmoon Inc., Tehran, Iran). The measurements were taken in duplicates (Hitachi 912 automatic analyzer, Japan). The intra-assay CV for these assays (n.10) were 1.1, 0.9, 0.95, 0.95, and 1.3% for TC, HDL-c, LDL-c, TG, apo-B, and calcium, respectively, and the interassay CVs (n.10) were 1.3, 1.3, 1.4, 1.4, 1.2, and 1.6%, respectively.

3.5. Statistical analyses

All data were gathered in a checklist and then transferred into SPSS version 15 for further analysis. First, the normal distribution of the quantitative variables was verified by Kolmogorov Smirnov tests. Statistical tests such as paired and student's t tests, Chi-squares, and one-way ANOVAs were used as appropriate. Correlations were assessed by Pearson correlation coefficients. The results were reported as means \pm SDs, and *P* values of less than 0.05 were considered to be statistically significant.

4. Results

This study began with 27 individuals in each group but ended with 49 (25 in the experimental group and 24 in the control group) because four participants used low-calorie diets and we had to exclude them from the study. The mean age was 34.42 ± 4.85 years. The anthropometric data are shown in Table 1. No significant differences were found between the two groups.

BMI measuring was done in the beginning and the end of the 8th week of the study for both groups. There was no significant difference between these two groups, as shown in Table 2. All BMIs were over 25, and 21% of all individuals had abdominal obesity (waist circumference of more than 95 cm). BMI and waist circumference were significantly, positively correlated ($P < .001$, $r = 0.538$). Daily dietary intakes at the beginning and end of the 4th and 8th weeks are illustrated in Table 3. There were no significant differences in the energy consumption and nu-

Table 1. Age, weight, height, and waist circumference data in two groups

Group	Control, n=24	Calcium, n=25	Total, n=49
Age, y	33.8 \pm 4.8	35.1 \pm 4.8	34.3 \pm 4.8
Weight, kg	85.6 \pm 8.0	81.4 \pm 7.7	84.3 \pm 8.6
Height, cm	175.8 \pm 6.4	172.6 \pm 6.0	174.1 \pm 6.6
Waist circumference, cm	93.5 \pm 5.5	94 \pm 4.4	93.7 \pm 5.0
BMI, kg/m ²	27.9 \pm 1.8	27.5 \pm 1.6	27.5 \pm 1.7

Table 2. Changes of BMI at beginning and end of the study

Group	Control, n=24	Calcium, n=25	<i>P</i> value
Beginning	27.9 \pm 1.8 ^b	27.5 \pm 1.6	NS ^a
End	28.1 \pm 0.5	27.6 \pm 2.7	NS
Changes (%)	0.3 \pm 1.2 (1.07)	0.02 \pm 1.17 (0.01)	NS
<i>P</i> value	NS	NS	-

^a NS: Not significant

^b Mean \pm SD

Table 3. Total energy and nutrient intake at different interval during the study

Nutrients and energy	Calcium group			Control group		
	Beginning	4th week	8th week	Beginning	4th week	8th week
Energy, kcal/day	3181 ± 516	3266 ± 478	3149 ± 443	3117 ± 418	3263 ± 571	3102 ± 421
Carbohydrates, g/day	485 ± 280	480 ± 252	480 ± 230	471 ± 271	478 ± 310	478 ± 310
Protein, g/day	102 ± 30	106 ± 22	99 ± 30	99 ± 18	110 ± 95	95 ± 29
Lipid, g/day	90 ± 26	98 ± 29	92 ± 25	99 ± 29	93 ± 16	89 ± 18
Cholesterol, mg/day	362 ± 93	386 ± 71	331 ± 85	355 ± 58	380 ± 119	326 ± 78
Calcium, mg/day	739 ± 262	857 ± 281	756 ± 213	786 ± 219	873 ± 354	787 ± 321
SFA, g/day	40.1 ± 9.0	40.5 ± 5.4	40.2 ± 5.4	36.7 ± 8.5	39.0 ± 4.2	37.5 ± 4.4
MUFA, g/day	34.1 ± 6.8	38.9 ± 10.5	35.8 ± 8.6	33 ± 3.3	37.1 ± 4.3	34.2 ± 8.2
PUFA, g/day	17.7 ± 8.6	19.6 ± 8.3	19.1 ± 6.7	18.2 ± 7.0	19.0 ± 9.4	19.8 ± 5.8
Magnesium, mg/day	364 ± 109	38 ± 130	355 ± 98	383 ± 148	394 ± 119	405 ± 140
Iron, mg/day	17.8 ± 4.0	17.9 ± 2.6	17.6 ± 3.4	18.3 ± 5.0	18.2 ± 3.1	18.1 ± 6.4
Phosphor, mg/day	1539 ± 465	1608 ± 648	1561 ± 479	1600 ± 515	1613 ± 582	1619 ± 413

trients throughout the study, not only within each group but also between the two groups.

All subjects were male and married. Only 2.5% of the subjects had an academic degree. Fifty-five percent of the individuals did not exercise regularly. No significant difference was found for physical activity between the two groups. The amount of physical activity had significant,

negative correlations with BMI ($P = .045$, $r = 0.288$) and waist circumference ($P = .002$, $r = 0.423$). A comparison of plasma lipoprotein and apo-B between the experimental and control groups at the beginning and the end of the study is shown in *Table 4*. There was a significant decrease in TC at the end of study compared to beginning values in the experimental group ($P < .0001$). Similarly, a sig-

Table 4. Comparison of lipid profile and other serum parameters between and within groups in the beginning and the end of the study

Parameters	Groups	Beginning	End	Changes (Mean ± SD (%))	P^b
Total cholesterol, mg/dl	Ca ^a	218.4 ± 42.1	190.0 ± 33.6	-28.3 ± 16.7 (-12.9)	< 0.001
	Control	211.9 ± 39.3	209.9 ± 44.9	-2.1 ± 17.5 (-1)	NS
	p^c	NS	NS	<0.001	
Triglyceride, mg/dl	Ca	238.7 ± 81.2	218.3 ± 80.9	-20.3 ± 81.4 (-8.5)	NS
	Control	217.8 ± 59.5	231.9 ± 75.5	13.4 ± 47.6 (6.4)	NS
	p^c	NS	NS	NS	
LDL-c, mg/dl	Ca	125.8 ± 35.9	102.9 ± 27±5	-22.9 ± 24.1 (-8.5)	< 0.001
	Control	128.6 ± 37.2	124.4 ± 42.1	-4.2 ± 18.1 (6.4)	NS
	p^c	NS	0.04	0.003	
HDL-c, mg/dl	Ca	46.4 ± 7.1	40.7 ± 5.7	-5.6 ± 5.5 (-12)	< 0.001
	Control	45.2 ± 6.3	39.5 ± 6.7	-6.5 ± 5.0 (-12.4)	< 0.001
	p^c	NS	NS	NS	
Apo-B, mg/dl	Ca	116.0 ± 20.5	108.0 ± 18.1	-8 ± 8.7 (-7.2)	<0.001
	Control	111.4 ± 17.1	111.2 ± 19.5	-0.02 ± 15.9 (-0.18)	NS
	p^c	NS	NS	0.044	
Serum Calcium, mg/dl	Ca	9.1 ± 0.06	9.8 ± 0.3	0.6 ± 0.4 (7.10)	< 0.001
	Control	9.2 ± 0.1	9.5 ± 0.5	0.3 ± 0.4 (3.56)	< 0.001
	p^c	NS	NS	0.02	
TC/HDL-c	Ca	4.7 ± 1.2	4.7 ± 1	-0.05 ± 0.7 (-1.04)	NS
	Control	4.9 ± 1.9	5.4 ± 1.5	0.5 ± 1.02 (11.6)	NS
	p^c	NS	0.006	0.018	
LDL-c/HDL-c	Ca	2.7 ± 0.8	2.5 ± 0.6	-0.2 ± 0.6 (-7.6)	< 0.001
	Control	2.9 ± 0.9	3.2 ± 1.2	0.2 ± 0.7 (8.4)	< 0.001
	p^c	NS	0.02	0.02	
LDL-c/Apo-B	Ca	1.08 ± 0.3	0.95 ± 0.2	-0.13 ± 0.18	0.02
	Control	1.15 ± 0.29	1.10 ± 0.3	-0.04 ± 0.2	NS
	p^c	NS	NS	NS	

^a Calcium group

^b Paired t-test

^c Student t-test

nificant difference was observed in the mean changes of TC between the two groups ($P < .0001$). There was an 8% reduction of TG in the experimental group and a 6% rise in the control subjects, but significant differences were not observed within or between the groups. We found a significant reduction in LDL-c in the experimental group (18%) after the intervention compared to the baseline values ($P < .001$) and also between the mean changes of LDL-c for the control group ($P < .001$ and $P = .003$, respectively). HDL-c was significantly different at the end of study compared to the initial values in the placebo ($P < .001$) and experimental ($P < .001$) groups. Apo-B also had a significant difference in the case group at the end of the study compared to initial values ($P < .001$) and also between the mean changes of the two groups ($P = .044$) at the end of study. The mean change of the TC/HDL-c and the LDL-c/HDL-c ratio showed a significant difference between the two groups ($P = .018$ and $P = .026$, respectively). There were significant differences in LDL-c/HDL-c and TC/HDL-c between the two groups at the end of study ($P = .018$ and $P = 0.006$, respectively). LDL-c/apo-B (which is the indirect indicator of LDL particle size and an important risk factor of CHD) decreased significantly from the beginning to the end of the study in the experimental group ($P = .02$). Systolic and diastolic blood pressure and their changes are shown in Table 5. Both systolic and diastolic blood pressure decreased significantly in the experimental group ($P < .001$) but not in the placebo group. In addition, the mean changes of systolic blood pressure were significantly different between the two groups ($P = .046$). At the beginning of the study, no correlation was found between calcium intake and serum calcium and all other measured parameters. At the end of the study, only the changes in serum calcium and LDL-c had moderate, positive correlations ($r = 0.334$, $P = .035$). Other indices did not correlate significantly.

5. Discussion

Our data suggest that 1,250 mg/day of supplemental calcium may reduce total cholesterol, LDL-c, apo-B, TC/HDL-c, and LDL-c/HDL-c in overweight men. In this study, there was a significant decrease in TC at the end of the trial between the two groups, which was consistent with the findings from Denke (27), Paydas *et al* (28), Yocowitz *et al.* (11) and Fleischman *et al.* (29). The significant, 6%

decrease in TC in the Denke *et al.* study was due to high calcium intake (27). Bell *et al.* reported no significant effects on TC, which was in part due to low doses of the calcium supplement (400 mg/day), the high mean age of the participants, and not matching the subjects in the calcium and placebo groups by sex (30). Shakhkhalili reported a nonsignificant, 9% decrease in TC, but a low calcium dose (980 mg/day), short duration of the study (2 weeks), and an inadequate number of participants ($N = 10$) might have influenced the results (31). Other studies reported greater decreases in serum TC after intervention when the participant had higher TC before the trial (32, 33). The biological plausibility for a cholesterol lowering effect is that calcium is known to bind to bile acids to form insoluble soaps and thus presumably prevent cholesterol entering into the enterohepatic circulation (27, 31, 34). Experimental animal evidence strongly supports this theory. One study found that a calcium supplements that lowered cholesterol levels in rats, rabbits, and goats but not in pigs were associated with an increase in the excretion of fecal bile acids in most but not all studies, and was more pronounced when the diet contained a higher proportion of saturated fats (35). Increased fecal loss of fat, especially saturated fatty acids (SFAs), is important because SFAs will increase serum cholesterol levels when absorbed. This effect of saturated fats has been known for decades, but its mechanisms are not yet fully understood; most likely, SFAs inhibit the receptor-mediated uptake of LDL into hepatocytes, thereby decreasing the clearance of LDL particles from circulation. Consequently, decreased absorption of saturated fat would lead to decreases in serum TC. On the other hand, LDL uptake by LDL receptors requires the acid-dependent mechanism on which the receptors themselves are" LDL itself is dependent to normal intake of calcium (21). Calcium and other divalent cations such as magnesium may explain this effect (21). In our study, there was no significant difference in serum TG before and after the intervention in the calcium group or at the end of study between the two groups. Other studies have shown similar results. Ried *et al.* reported the same result in postmenopausal women who took calcium supplements for 1 year (1,000 mg/day; (36). Vaskonen *et al.* showed no significant change in serum TG in obese rats that were fed a high-fat diet with calcium supplements, but he reported a significant decrease in TG with for rats that were fed

Table 5. Comparison of systolic and diastolic blood pressure between and within groups in the beginning and the end of the study

Parameters	Group	Beginning	End	Changes (%)	P value ^b
Systolic blood pressure	Ca ^a	116.8 ± 11.9	108.9 ± 9.7	-8.0 ± 7.0 (-6.84)	<0.001
	Control	121.1 ± 12.8	117.4 ± 9.9	-3.7 ± 7.8 (-3.05)	NS ^d
	p ^c	NS	0.031	0.046	-
Diastolic blood pressure	Ca	79.5 ± 5.5	75.2 ± 5.3	-4.3 ± 3.4 (-5.40)	<0.001
	Control	80.9 ± 6.6	78.8 ± 8.2	-2.1 ± 6.1 (-2.60)	NS
	p ^c	NS	NS	NS	-

^a Calcium group

^b from paired t-Test

^c from student t-Test

^dNS: Not significant

a low-fat diet (20% of total calorie intake; (37). Similarly, Torres reported a significant decrease in TG with 1,200 to 1,300 mg of calcium and an energy-reduced diet (9). Flatt's study showed a similar, significant decrease with the same regimen (38). Indeed, in our study participants consumed a regular-fat diet (about 28% of total calorie intake), so it is reasonable to expect that there would be no significant decrease in serum TG. LDL-c decreased significantly (by 18%) by the end of the study compared to the control group. Paydas reported a 28% decrease of LDL-c in hemodialytic patients who consumed 3 g of calcium per day for 10 months (28). It seems that higher doses of calcium and a longer time period are the reasons for the higher reduction. Other studies have shown 15% (31), 11% (27), 6% (36), and 4.4% (36) decreases in LDL-c. The lack of a significant difference in Bostick's study was probably due to sporadic adenoma, patient disease, not matching for sex and age, and a nonfasting state (39). At the end of our study, there was no significant difference in HDL-c between the two groups, but the LDL-c/HDL-c ratio, which is a well-established risk factor for CVD, had significantly decreased in the calcium group compared to the placebo group. Ried reported a higher decrease in this ratio compared to our study, perhaps because of the longer period of their trial (36). In our study, calcium supplements led to significant decrease, about 7%, in apo-B. Denkeh also reported a 7% decrease in apo-B hypercholesterolemic men after 2,100 mg/day of calcium consumption over 10 days (27). Due to the fact that the RDA of calcium is about 1,000 mg/day (40), it seems that 2,100 mg/day may be not a suitable dose for intervention or recommendation. Other studies have shown no difference before and after calcium supplementation, perhaps due to low doses of the calcium supplements used. A reasonable explanation of this decrease is that higher calcium intake may lead to more conversion of apo-B100 mRNA to apoB-48 mRNA and finally a decrease of apo-B100, which translates to a decrease of LDL particles (30). Apo-B is the best predictor of high cardiovascular risk and subclinical extracoronary and coronary atherosclerosis (41). After the trial, systolic blood pressure in our case group had decreased significantly, and its changes showed significant differences between groups ($P = .031$). Still, diastolic blood pressure changes did not have the same result ($P = .078$). There are several studies in favor of reducing the effects of calcium supplementation on blood pressure (42). A Meta-analysis on 23 observational studies estimated that a 100-mg increase in calcium intake can lower systolic by about 0.39 mmHg and diastolic blood pressure by 0.35 mmHg (43). One possible explanation for this is that high calcium intake can increase sodium urinary excretion, increase the activity of calcium ATPase in erythrocytes' membranes, and result in lower intracellular calcium and dilation of vessels (44). More recent studies have also showed that increased dilatation of vessels (because of calcium-mediated potassium channels opening), heightened sensitivity to nitric oxide, and lower production of superoxide

and vessel constrictor prostaglandins can be a part of the cause of the blood-pressure-lowering effect of calcium (9, 23, 45, 46). Other studies have shown that the negative feedback on and decreased levels of PTH and vitamin D in serum are other possible contributors to this effect because these hormones can increase intracellular calcium and make the soft muscle cells contract and constrict the vessels, which finally result in higher blood pressure (47). In our study, the number of hypertensive individuals was limited; probably because our subjects were younger and more active than samples in many other studies. Furthermore, because of genetic variety, an individual's blood pressure can respond to obesity in different ways. We matched subjects on gender and close similarity in nutrition intake between groups, which may make our results more authentic. However, our study was limited in that we did not have access to ultracentrifuge to assay subtypes of lipoproteins (e.g. HDL2 and HDL3) directly, and we were unable to measure apoA-I or body fat percentage. For further study we recommend that trials be done on larger sample sizes for longer durations in other age groups with different physiological conditions. Other studies, such as the effects of calcium on the liver or vessel wall fat composites or lipids, should demonstrate insightful results. In conclusion, it seems that 1,250 mg/day of a calcium supplement may lower serum apo-B, lipoproteins, and systolic blood pressure and may have beneficial effects on risk factors of cardiovascular diseases.

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Conflict of interest

The authors reports no conflicts of interest.

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