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# Effects of Roasted Soy-Nut Supplementation on Lipid Profile of Iranian Postmenopausal Women

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**Abstract:** The present study was carried out to study the effect of roasted soy-nut consumption on the lipid profile of postmenopausal women. Randomized Controlled trial was conducted using 100 postmenopausal women aged 45-60 years. Participants in the treatment group were provided with 75g roasted soy-nut for daily intake for 2-months. The participants in the control group were asked not to change their usual dietary habits and lifestyles and all were instructed to avoid taking soybean and soybean products. The changes in blood lipid profile were measured beside some other outcome measures comprised of anthropometry, dietary variables and lifestyle questionnaires, measured at baseline and at the end of the study. Results revealed significant ($p<.005$) differences in $ApoA_I$ were observed between two groups at baseline. The t-test only showed significant ($p<.005$) difference in the mean total cholesterol (TC) between the treatment and control groups after two months intervention. Using the General Linear Model (GLM) for repeated measures, significant mean differences were observed for TC (-5.58%), low-density lipoprotein cholesterol (LDL-C) (-9.54%), non-high density lipoprotein cholesterol (HDL-C) (-7.34%), apolipoprotein $A_I$ (12.26%) and apolipoprotein B (-7.69%). No significant differences in triglyceride (TG), HDL-C and the ratio of ApoB:$ApoA_I$ were observed. The results from GLM were reconfirmed using Analysis of Covariance. In c*onclusion:* A short-term intake of roasted soy-nut improved the lipid profile of the postmenopausal subjects.

**Key words:** Soy-nut % Menopause % Lipids % Lipoproteins % Apolipoproteins

## INTRODUCTION

Both aging and menopause influence lipid metabolism, which consequently lead to higher risk of cardiovascular diseases (CVD) [1, 2]. Enhanced intestinal cholesterol absorption and decreased hepatic bile salt synthesis are consequences of aging per se [3, 4] Withdrawal of endogenous estrogens in postmenopausal women also decrease the hepatic expression of LDL receptors, increases abdominal lipoprotein lipase activity [5] and deteriorate the regulation of plasma triglyceride (TG) [6, 7]. Because estrogen exerts beneficial effects on circulating lipid and lipoprotein levels, it is believed that non-steroidal compounds such as isoflavones, which weakly binds estrogen receptors, may also have these effects [8, 9]. Soybean represents significant amount of isoflavonoids. For this reason some women have adopted soy products as a natural alternative to hormone therapies [10]. The replacement of animal protein in the diet with soybean protein has been shown to reduce the level of plasma lipids in humans [11]. The initial cholesterol level, gender and the intake of dietary cholesterol, the overall amount of soy protein consumed and the isoflavone content of soy protein are the factors which seem to influence the hypocholesterolemic effects of soy protein. Although the mechanism by which soy protein decrease the cholesterol is not fully known, it is believed that it might be due largely to the amino acid composition of soy protein, soy oil, soy lecithin, saponins, protein digestibility, protein phosphorylation and isoflavones/ phytoestrogens [12]. The clinical significance of ingesting soybean on lipid profile still remains controversial. Some

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studies have reported lipid-lowering effects of soybean but some showed no changes even with long-term duration of exposure to the soybean. It may be attributed to the chemical heterogeneity of the soy products used, exposure length and study design [13]. The evaluation of cocktail effects of entire valuable components in whole soy-nuts, which was neglected by most of the earlier studies, seems more accessible and uncomplicated for achieving clinical reasons. Some of the previous studies achieved the lipid lowering effects of soybean by replacement of animal protein with soy protein or by switching to soy oil, suggested that the reductions were perhaps a reply to low saturated fat and cholesterol intake [14]. This study was to investigate the effects of consuming roasted soy-nut on total cholesterol (TC), TG, low density lipoprotein cholesterol (LDL-C), high density lipoprotein cholesterol (HDL-C), non high density lipoprotein cholesterol (non-HDL-C), apolipoprotein $A_I$ ($ApoA_I$), apolipoprotein B (ApoB) and ApoB: $ApoA_I$ of postmenopausal women.

## MATERIALS AND METHODS

**Subjects:** A total of 100 postmenopausal women (45-60 years) from the city of Isfahan, Iran who met the selection criteria were enrolled in the study. Women were excluded if they had any significant chronic or degenerative disease, regular use of medication (e.g., lipid-lowering drugs, anti-diabetic medications, anti-hypertension drugs, or aspirin) known to interfere with the study endpoints, current or previous (in the preceding 6 mo) use of estrogen therapy, consumption of a strict vegetarian diet, soybean or soy-derived products, high fiber or low fat diet, vigorous physical activity and cigarette smoking. Institutional ethical approval was obtained and each participant provided informed consent prior to data collection.

**Method:** It was a 2-month randomized controlled trial (RCT). Participants were randomly assigned to the treatment and control groups. The treatment group was provided 75g roasted soy-nut for daily usage during the 2-month intervention. The control group was encouraged not to change their usual dietary habits and lifestyles and was instructed to avoid taking soybean and soybean products. The treatment group was provided with 14 packages of 75g of soy-nut for two weeks supply. Refills were provided the following two weeks and the compliance of soy-nut intakes were recorded during those visits. The nutrient composition of soy-nut was based on the estimation of Azadbakht *et al*. [15]. Thus, the women in the treatment group received 225 mg of isoflavonoids (including: 22.5 mg glycitein, 132.5 mg genistein and 100 mg diadzein) per day.

Assessment of nutrient intake was based on 3-day dietary record (at baseline and after the intervention) and food frequency questionnaires (FFQ) (only at baseline). Verbal instructions were given to help participants to complete the forms. The 3-day Food Record was analyzed using Nutritionist IV, which was designed for Iranian foods.

Weight was measured using a digital scale calibrated with 1 kg known weight and with the accuracy of 0.1 kg. A tape measure to the nearest 0.1 cm was used for height and body circumferences measurements. Blood pressure was measured after participants rested at least 5 minutes in a comfortable sitting position while they were not involved in active conversation. A total of 10 cc venous blood sample was drawn after 10-12 hours of fasting. All blood samples were analyzed in the central laboratory of Isfahan University of Medical Sciences Research Center, Department of Nutrition. All of the biochemical tests were assayed on BioSystems A15 Autoanalyzer (BioSystems S.A. Costa Brava 30, Barcelona, Spain). The concentration of both TC and TG were measured using BioSystems spectrophotometry assay kits. HDL-C was enzymatic spectrophotometrically measured using BioSystems kit. Both $ApoA_I$ and ApoB were assayed by turbidimetry method using BioSystems kits. The levels of LDL-C were calculated by Friedwald formula (TC - HDL-C - 0.20 × TG) for those with TG<400 mg/dl and in the case of TG>400 mg/dl using the following Friedwald formula: TC - HDL-C - 0.16 × TG [17].

**Statistics:** All data were analyzed using SPSS for Windows (Version 15) to obtain descriptive and inferential statistics. Independent t-test was used to compare the means of variables before and after intervention. GLM (repeated measures) was used to compare means of lipid profile before and after intervention. The independent t-test of mean differences also was applied. The percentage change for each variable was calculated by the formula $[(E - B)/B]$ X 100, while $E$ is the value after intervention and $B$ is the baseline value. The mean percentage change differences, which were derived by calculating the differences in percentage change for each variable in pair-wise group comparisons, were also calculated. The results from GLM were reconfirmed again using Analysis of Covariance (ANCOVA). The nonparametric Mann-Whitney test was

used for variables, which were not normally distributed or were ordinal or nominal measurements. P-value of less than 0.05 was considered significant.

## RESULTS

A total of 42 out of 50 women in treatment group and 41 out of 50 women in the control group completed the study. Stomachache, bloating, diarrhea and constipation were some of the reasons for dropout. Nutrient intakes of participants are shown in Table 1. There were significant (p<.005) differences in the mean intake of calorie and nutrient content between the two groups after the intervention. The anthropometric measurements, blood pressure and lipid profile are shown in Table 2. At baseline there were no significant differences in the mean values of socio demographic characteristics of the patients, anthropometric measurements, blood pressure and lipid profile except for $ApoA_I$. After the two months of intervention, only TC was significantly (p<.005) different between the treatment and control groups. Using independent t-test for mean differences (Table 3), a significant reduction in the percent change in TC in the treatment (13.3%) compared to the control (7.8%) groups was observed. Similar reductions were seen for LDL-C in the treatment group (17.1%) and in the control group (7.5%). Using the GLM for repeated measures, significant mean differences were observed for TC, LDL-C, $ApoA_I$, ApoB and non-HDL-C. Analysis of covariance also reconfirmed the outcomes obtained from previous analysis.

Table 1: Mean ±SD of Macronutrient Intakes at Baseline and after the Intervention

| | Baseline | | | | | After Intervention | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | Treatment | | Control | | | Treatment | | Control | | |
| Nutrient | Mean ±SD | Percentage | Mean ±SD | Percentage | P-value | Mean ±SD | Percentage | Mean ±SD | Percentage | P-value |
| Total Calorie (kcal) | 1838±204 | - | 1917 ±204 | - | 0.079 | 2206 ±179 | - | 1887 ±211 | - | 0.000* |
| CHO (g) | 320.51±44.26 | 69.63±3.91 | 316.88±45.57 | 65.99±5.22 | 0.714 | 346.33±42.22 | 62.65±3.92 | 314.10±45.29 | 66.40±4.17 | 0.001* |
| Protein (g) | 54.20±8.12 | 11.79±1.15 | 56.03±9.79 | 11.64±1.21 | 0.356 | 83.66±7.26 | 15.18±0.83 | 55.73±8.05 | 11.80±0.98 | 0.000* |
| Total Fat (g) | 37.68±8.62 | 18.56±4.18 | 45.88±10.37 | 21.65±5.04 | 0.000* | 58.42±8.99 | 23.95±4.04 | 45.48±8.10 | 21.85±4.17 | 0.000* |
| SFA (g) | 7.13±1.83 | 3.50±0.87 | 8.83±2.14 | 4.16±1.01 | 0.000* | 10.33±2.05 | 4.23±0.87 | 8.86±2.26 | 4.26±1.16 | 0.003* |
| MUFA (g) | 9.52±2.62 | 4.68±1.25 | 12.32±2.86 | 5.79±1.24 | 0.000* | 14.01±2.53 | 5.74±1.11 | 11.69±2.56 | 5.61±1.27 | 0.000* |
| PUFA (g) | 21.02±5.15 | 10.36±2.55 | 24.72±6.71 | 11.70±3.38 | 0.006* | 33.07±5.51 | 13.56±2.46 | 24.92±4.61 | 11.97±2.33 | 0.000* |
| Fiber (g) | 32.16±6.87 | - | 33.34±7.04 | - | 0.433 | 54.79±6.70 | - | 32.73±6.90 | - | 0.000* |

Note: CHO, carbohydrate; SFA, saturated fatty acid; MUFA, monounsaturated fatty acid; PUFA, polyunsaturated fatty acid

* P <0.05

Table 2: Mean ± SD for anthropometric measurements, blood pressure and lipid profile at baseline and after Intervention

| | Baseline | | | | After Intervention | | | |
|---|---|---|---|---|---|---|---|---|
| Parameters | Treatment Mean ± SD | Control Mean ± SD | T-test | P-value | Treatment Mean ± SD | Control Mean ± SD | T-test | P-value |
| Height (cm) | 157.47±5.760 | 157.24±6.280 | 0.175 | 0.861 | - | - | - | - |
| Weight (kg) | 65.71±7.570 | 68.17±7.380 | -1.495 | 0.139 | 65.16±7.230 | 67.43±7.380 | -1.416 | 0.161 |
| BMI (kg/m2) | 26.51±2.900 | 27.57±2.460 | -1.780 | 0.079 | 26.29±2.700 | 27.27±2.430 | -1.708 | 0.091 |
| WC (cm) | 98.42±6.550 | 98.87±6.270 | -0.319 | 0.750 | 96.64±7.190 | 98.43±6.330 | -1.206 | 0.231 |
| HC (cm) | 104.57±5.280 | 104.82±5.810 | -0.211 | 0.833 | 102.90±4.650 | 103.65±5.510 | -0.647 | 0.503 |
| WHR | 0.94±0.050 | 0.94±0.040 | -0.202 | 0.840 | 0.93±0.050 | 0.95±0.040 | -1.079 | 0.284 |
| SBP (mm Hg) | 123.57±16.68 | 124.26±15.59 | -0.196 | 0.845 | 125.71±19.01 | 120.97±13.14 | 1.318 | 0.191 |
| DBP (mm Hg) | 81.90±9.170 | 79.39±10.62 | 1.155 | 0.251 | 76.42±10.55 | 76.97±11.74 | -0.223 | 0.824 |
| TC | 209.90±32.91 | 212.17±28.87 | -0.333 | 0.740 | 181.90±28.18 | 195.70±34.21 | -2.008 | 0.048* |
| TG | 148.52±58.19 | 153.48±62.03 | -0.376 | 0.708 | 140.76±68.74 | 141.85±68.22 | -0.073 | 0.942 |
| LDL-C | 129.98±27.46 | 128.23±28.76 | 0.284 | 0.777 | 107.80±24.23 | 118.59±33.70 | -1.677 | 0.097 |
| HDL-C | 50.21±10.07 | 53.23±10.39 | -1.347 | 0.182 | 45.94±9.530 | 48.74±8.860 | -1.384 | 0.170 |
| Apo AI | 132.52±18.95 | 141.59±14.50 | -2.443 | 0.017* | 147.80±16.22 | 140.56±18.55 | 1.896 | 0.062 |
| Apo B | 148.66±27.61 | 158.41±34.56 | -1.421 | 0.159 | 144.73±22.62 | 142.04±30.27 | 0.459 | 0.647 |
| Non-HDL-C | 159.69±31.90 | 158.93±29.94 | 0.112 | 0.911 | 135.95±27.41 | 146.96±36.36 | -1.559 | 0.123 |
| Apo B /Apo AI | 1.17±0.450 | 1.13±0.280 | 0.463 | 0.645 | 0.99±0.200 | 1.02±0.250 | -0.636 | 0.526 |

Note: SD, standard deviation; BMI, body mass index; WC, waist circumferences, HC, hip circumference; WHR, waist hip ratio; SBP, systolic blood pressure; DBP, diastolic blood pressure; TC, total cholesterol; TG, triglycerides; LDL-C, low density lipoprotein cholesterol; HDL-C, high density lipoprotein cholesterol; Apo AI, apolipoprotein AI; Apo B; apolipoprotein B

* p<0.05

Table 3: Mean ± SE and Mean d ± SE d of Changes in Lipid Profile after the Intervention

| | Mean reduction± SE (Within groups) | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| Lipid Profile (mg/dl) | Treatment group | Percent Changes | Control group | Percent Changes | Mean d ± SE d (Between groups) | Total percent changes | T-test | P-value |
| TC pre-post | 28.00±3.71 | 13.34 | 16.46±3.47 | 7.76 | 11.53±5.080 | 5.58 | 2.269 | 0.026* |
| TG pre-post | 7.76±8.05 | 5.22 | 11.63±7.79 | 7.58 | -3.87±11.20 | -2.36 | -0.345 | 0.731 |
| LDL-C pre-post | 22.18±2.94 | 17.06 | 9.64±3.17 | 7.52 | 12.53±4.320 | 9.54 | 2.900 | 0.005* |
| HDL-C pre-post | 4.26±0.89 | 8.50 | 4.49±0.88 | 8.44 | -0.22±1.250 | 0.06 | -0.182 | 0.856 |
| Apo AI pre-post | -15.28±2.14 | -11.53 | 1.02±3.11 | 0.73 | -16.31±3.760 | -12.26 | -4.335 | 0.000* |
| Apo B pre-post | 3.92±2.60 | 2.64 | 16.36±3.56 | 10.33 | -12.43±4.390 | -7.69 | -2.818 | 0.006* |
| Non-HDL-C pre-post | 23.73±3.19 | 14.87 | 11.97±3.11 | 7.53 | 11.76±4.460 | 7.34 | 2.636 | 0.010* |
| Apo B/Apo AI pre-post | 0.17±0.06 | 15.38 | 0.10±0.02 | 9.73 | 0.06±0.060 | 5.65 | 1.072 | 0.287 |

Note: SE= Standard Error of Changes in each group; Meand= Mean of differences; SEd =Standard Error of Differences; TC, total cholesterol; TG, triglycerides; LDL-C, low density lipoprotein cholesterol; HDL-C, high density lipoprotein cholesterol; Apo AI, apolipoprotein AI; Apo B; apolipoprotein B (Apo B)
* $p<0.05$

## DISCUSSION

The current study showed that the consumption of 75g/day of roasted soy-nut for two months was associated with a significant reduction in TC level (13.3%) compared to the control group (7.8%). Similarly, LDL-C reduced by 17.1% in the treatment and 7.5% in the control groups. Identical results were reported by Chen *et al*. [18]. A recent study by Azadbakht *et al*. [15] also showed that soy-nut diet resulted higher declines in TC and LDL-C concentrations compared to soy protein diet. This study achieved upward trends in the mean TG and HDL-C, respectively by 5.22 and 8.50% in the treatment group and by 7.58 and 8.44% in the control group. None of the t-test, GLM and ANCOVA showed significant increase in the mean TG and HDL-C between the treatment and control groups. Consistently the review by Zhan and Ho [19] reported significant reductions in TC (3.77%), LDL-C (5.25%) and TG (7.27%) through soy isoflavone intervention but an increase in HDL-C by 3.03%. Improvements in HDL-C have been only observed in studies with duration longer than 12 weeks [19]. Hence, unchanged level of HDL-C achieved in this study might be due to the shorter period of intervention.

The findings also showed an increase in $ApoA_I$ by 11.53% for the treatment group although two other recent studies showed no significant differences [15, 20]. This significant increase in $ApoA_I$ did not correspond with HDL-C changes. In contrast Matthan et al. [21] showed similar patterns of significant increase in $ApoA_I$ and HDL-C levels however Apo $A_I$ is more crucial in biochemical pathways that provides HDL-C its antiatherogenic effect [22]. The study showed higher reduction in ApoB in the control group (10.33%) compared to the treatment group (2.64%). Therefore an overall increase by 7.69% in ApoB was observed after the intervention that was unparallel with the downward trend in LDL-C. On the contrary, Azadbakht *et al.* [15] illustrated a similar reduction pattern in ApoB and LDL-C for both soy-protein and soy-nut groups. These results confirmed with other studies as well [18, 23] however, soymilk intake showed no significant difference in ApoB but a modest decrease (4%) in LDL-C [21]. Recent studies have suggested that ApoB is more atherogenic than LDL-C [24, 25]. Because there is one ApoB molecule per lipoprotein particle, ApoB level provides a good measure of total number of VLDL-C, IDL-C and LDL-C particles thus the concentration of proatherogenic particles [26, 22].

Non-HDL-C is also established to predict CVD risk [27]. The outcomes of this study showed unparallel changes in non-HDL-C (a reduction by 7.34%) and ApoB. Similar findings for non-HDL-C were also observed in other clinical trials [18, 23]. The inverse trend in changes of ApoB and non-HDL-C seems to be conflicting to some extent [26, 28]. Since plasma concentration of atherogenic lipoproteins may be more critical in development of atherosclerosis than the amount of cholesterol that the lipoproteins carry into the arterial wall, ApoB is more strongly correlated to CVD incidence than non-HDL-C and LDL-C [28]. However, the superiority of LDL-C, ApoB and Non-HDL-C as risk factors in predicting CHD events is still unsettled [29, 30]. The outcomes of the present study revealed that the overall reduction in the ratio of Apo B to Apo $A_I$ was almost 5.65%, which close to the cut-off point proposed by Walldius *et al*. [31] (0.8 for women). While the a higher ratio is associated with a higher risk of CVD, the increment of $ApoA_I$ level along with decreased in the ratio of ApoB to $ApoA_I$ after soy-nut intervention supported the beneficial effects of soy-nuts in CVD prevention.

The beneficial effects of soy protein replacement with animal protein in the diets has been proposed to be probably a respond to the upshot of low dietary saturated fat and cholesterol intake [18] nonetheless, the present study investigated the inclusion of extra 75 g soy-nut into the usual diets without replacing the animal proteins. In contrast to the animal proteins comprising higher amount of indispensable amino acids such as lysine contributing hypercholesterolemic effects, soy protein encompasses superior amount of dispensable amino acids such as arginine, glycine and alanine that tend to lower cholesterol concentrations [32].

Two meta-analyses concluded that the isoflavone content of soy may be responsible for its lipid-lowering effect [19, 33] hence alcohol-extracted soy protein (removed phytoestrogens) was less effective at lowering plasma cholesterol levels relative to non-alcohol-washed soy protein diets [34]. Conversely no estrogenic activity of soy isoflavone on the lipid profile was observed in other studies [8, 13].

Clarkson and Anthony [35] suggested that alcohol extraction might remove the responsible active agent otherwise the isoflavones might become inactivated during the process of purification. Previous studies indicated that persons vary greatly in their ability to synthesize active form of isoflavones called equol, making it difficult to determine the ideal dose [33, 36]. Although Amani *et al*. [37] proposed that the hypocholesterolemic effect of isoflavones is not in a dose-response manner and isoflavones activity is closely related to soy protein. The intake of isoflavonoids and soy protein jointly has been accompanied by reduction in LDL-C levels and improvements in lipid profile [38-40]. The mechanism by which soy protein may bring about the effects of isoflavonoids on lipids is not known, but it may facilitate the transport of isoflavonoids in blood or their entry into the target organs, such as liver or muscle cells [8]. However several studies suggested that other non-protein components present in soy may be partially responsible for the hypocholesterolemic effects [12].

The current study did not include any specific dietary limitation and only the inclusion of soy-nut in the usual diets of the participants was assessed. The bioavailability of functional ingredients in soy meals may be also influenced by various diets. For example soybean has been shown to have hypolipidemic effects when high fat diets were consumed. Therefore a controlled diet for participants in study could reveal more apparent soy potential clinical effects. Although the prolonged periods of food interventions is associated with “diet fatigue” and lead to less attention paid to the diet, but the longer duration of the soy nut intervention may reveal more clear outcomes. Alternatively the possibility of different study design such as crossover study should be considered. The limitation of this study might have affected the outcomes, hence more practical and applicable studies may improve the knowledge about soybean and its products especially those with more natural structure.

## CONCLUSION

The findings indicated that supplementation of 75g roasted soy-nut into the diet without changes in dietary intake for a short-term improved the lipid profile of postmenopausal women. Therefore, moderate daily intake of soy may be a safe and practical modality to improve CVD risk and provides cardio-protective benefits.

## REFERENCES

1. Kolovou, G.D. and H.G. Bilianou, 2008. Influence of Aging and Menopause on Lipids and Lipoproteins in Women. Angiol., 59: 54S-57S.
2. Thomas, A.W., O. Frank and J.N. Robert, 2007. Soy protein concentrate lowers serum high-density lipoprotein cholesterol concentrations compared with casein in ovariectomized rats fed a low-fat, cholesterol-free diet. Nutrition Res., 27: 417-422.
3. Wang, D.Q., 2002. Aging per se is an independent risk factor for cholesterol gallstone formation in gallstone susceptible mice. J. Lipid Res., 43: 1950-1959.
4. Valdivieso, V., R. Palma, R. Wunkhaus, C. Antezana, C. Severin and A. Contreras, 1978. Effect of aging on biliary lipid composition and bile acid metabolism in normal Chilean women. Gastroenterol., 74: 871-874.
5. Tchernof, A., A. Desmeules, C. Richard *et al*., 2004. Ovarian hormone status and abdominal visceral adipose tissue metabolism. Journal of Clinical Endocrinology and Metabolism, 89: 3425-3430.
6. Kolovou, G.D., K.K. Anagnostopoulou, D.S. Damaskos *et al*., 2007. Gender influence on postprandial lipemia in heterozygotes for familial hypercholesterolemia. Annals of Clinical and Laboratory Sci., 37: 335-342.
7. Kolovou, G.D., K.K. Anagnostopoulou, K.D. Salpea *et al*., 2006. Postprandial lipemia in postmenopausal women with high fasting high-density lipoprotein cholesterol. The American J. the Medical Sci., 331: 10-16.

8. Nikander, E., A. Titinen, K. Laitinen, M. Tikkanen and O. Ylikorkala, 2004. Effects of Isolated Isoflavonoids on Lipids, Lipoproteins, Insulin Sensitivity and Ghrelin in Postmenopausal Women. J. Clinical Endocrinology and Metabolism, 89: 3567-3572.
9. Williamson-Hughes, P.S., B.D. Flickinger, M.J. Messina and M.W. Empie, 2006. Isofalvone supplements containing predominantly genistein reduce hot flash symptoms: a critical review of published studies. Menopause, 13: 831-9.
10. Morito, K., T. Hirose, J. Kinjo, R. Tsuchihashi, T. Aomori, T. Nagao, H. Okabe, T. Nohara and Y. Masamune, 2001. Interaction of phytoestrogens with estrogen receptors " and $. Biological and Pharmaceutical Bulletin, 24: 351-356.
11. Torres, N., I. Torre-Villalvazo and A.R. Tovar, 2006. Regulation of lipid metabolism by soy protein and its implication in diseases mediated by lipid disorders. The J. Nutritional Biochemistry, 17: 365-373.
12. Wilson, T.A., R.J. Nicolosia, T. Kotylaa and B. Fleckingerb, 2007. Soy protein without isoflavones reduces aortic total and cholesterol ester concentrations greater than soy protein with isoflavones compared with casein in hypercholesterolemic hamsters. Nutrition Res., 27: 498-504.
13. Nahas, E.A.P., J. Nahas-Neto, F.L. Orsatti, E.P. Carvalho, M.L.C.S. Oliveira and R. Dias, 2007. Efficacy and safety of a soy isoflavone extract in postmenopausal women: A randomized, double-blind and placebo-controlled study. Maturitas, 58: 249-258.
14. Dewell, A., P.L.W. Hollenbeck and C.B. Hollenbeck, 2006. Clinical review: a critical evaluation of the role of soy protein and isoflavones supplementation in the control of plasma cholesterol concentrations. J. Clinical Endocrinol. and Metabolism, 91: 772-780.
15. Azadbakht, L., M. Kimiagar, Y. Mehrabi, A. Esmaillzadeh, M. Padyab, F.B. Hu and W.C. Willett, 2007. Soy inclusion in the diet improves features of the metabolic syndrome: a randomized crossover study in postmenopausal women. The American J. Clinical Nutrition, 85: 735-741.
16. Reaburn, J.A., M. Krondl and D. Lau, 1979. Social determinants in food selection. J. American Dietetic Association, 74: 637-641.
17. Friedwald, W.T., R.I. Levy and D.S. Fredrickson, 1972. Estimation of the concentration of low-density lipoprotein cholesterol in plasma, without use of the preparative ultrac. Clinical Chemistry, 18: 499-502.
18. Chen, S.T., J.R. Chen, C.S. Yang, S.J. Peng and S.H. Ferng, 2006. Effect of soy protein on serum lipid profile and lipoprotein concentrations in patients undergoing hypercholesterolaemic entrifuge. Clinical Chemistry, 18: 499-502.
19. Zhan, S. and S.C. Ho, 2005. Meta-analysis of the effects of soy protein containinz isoflavones on the lipid profile. The American J. Clinical Nutrition, 81: 397- 408.
20. Garrido, A., P.M. De La Maza, S. Hirsch and L. Valladares, 2006. Soy isoflavones affect platelet thromboxane A2 receptor density but not plasma lipids in menopausal women. Maturitas, 54: 270-276.
21. Matthan, N.R., S.M. Jalbert, L.M. Ausman, J.T. Kuvin, R.H. Karas and A.L. Lichtenstein, 2007. Effect of soy protein from differently processed products on cardiovascular disease risk factors and vascular endothelial function in hypercholesterolemic subjects. The American J. Clinical Nutrition, 85: 960-966.
22. Van Der Steeg, W.A., S.M. Boekholdt, E.A. Stein, K. El-Harchaoui, E.S.G. Stroes, M.S. Sandhu *et al*., 2007. Role of the Apolipoprotein B-100 Apolipoprotein A-I Ratio in Cardiovascular Risk Assessment: A Case-Control Analysis in EPIC-Norfolk. Annals of Internal Medicine, 146: 640-648.
23. Teixeira, S.R., S.M. Potter, R. Weigel, S. Hannum, J.W. Erdman and C.M. Hasler, 2000. Effects of feeding 4 levels of soy protein for 3 and 6wk on blood lipids and apolipoproteins in moderately hypercholesterolemic men. The American J. Clinical Nutrition, 71: 1077-1084.
24. Sacks, F.M., 2006. The apolipoprotein story. Atherosclerosis Supplements, 7: 23- 27.
25. Zambon, A., B.G. Brown, S.S. Deeb and J.D. Brunzell, 2006. Genetics of apolipoprotein B and apolipoprotein AI and premature coronary artery disease. J. Internal Medicine, 259: 473-480.
26. Sniderman, A.D., C.D. Furberg, A. Keech, J.E. Roeters van Lennep, J. Frohlich, I. Jungner and G. Walldius, 2003. Apolipoproteins versus lipids as indices of coronary risk and as targets for statin treatment. Lancet, 361: 777-780.
27. Ridker, P.M., N. Rifai, N.R. Cook, G. Bradwin and J.E. Buring, 2005. Non-HDL cholesterol, apolipoproteins A-I and B100, standard lipid measures, lipid ratios and CRP as risk factors for cardiovascular disease in women. The J. American Medical Association, 294: 326-333.

28. Pischon, T., C.J. Girman, F.M. Sacks, N.R. Rifai, M.J. Stampfer and E.B. Rimm, 2005. Non-High-Density Lipoprotein Cholesterol and Apolipoprotein B in the Prediction of Coronary Heart Disease in Men. Circulation, 112: 3375-3383.
29. Denke, M.A., 2005. Weighing in before the fight: low-density lipoprotein cholesterol and non-high-density lipoprotein cholesterol versus apolipoprotein B as the best predictor for coronary heart. Circulation, 112: 3368-3370.
30. Sniderman, A.D., 2005. Apolipoprotein B versus non-high-density lipoprotein cholesterol: and the winner is. Circulation, 112: 3366-3367.
31. Walldius, G. and I. Jungner, 2004. Apolipoprotein B and apolipoprotein A-I: risk indicators of coronary heart disease and targets for lipid-modifying therapy. J. Internal Medicine, 255: 188-205.
32. Radcliffe, J.D. and D.M. Czajka-Narins, 2000. Use of arginine to reduce the severity of retinoid-induced hypertriglyceridemia. Nutrition and Cancer, 36: 200-206.
33. Zhuo, X.G., M.K. Melby and S. Watanabe, 2004. Soy isoflavones intake lowers serum LDL cholesterol: A metaanalysis of 8 randomized controlled trials in humans. Nutrition J., 134: 2395-2400.
34. Wilsona, T.A., R.J. Nicolosia, T. Kotylaa and B. Fleckingerb, 2007. Soy protein without isoflavones reduces aortic total and cholesterol ester concentrations greater than soy protein with isoflavones compared with casein in hypercholesterolemic hamsters. Nutrition Res., 27: 498-504.
35. Clarkson, T.B. and M.S. Anthony, 1998. Phytoestrogens and coronary heart disease. Baillière's Clinical Endocrinology and Metabolism, 12: 589-604.
36. Rowland, I.R., H. Wisemanm, T.A. Sanders, H. Adlercreutzm and E.A. Bowey, 2000. Interindividual variation in metabolism of soy isoflavones and lignans: influence of habitual diet on equol production by the gut microflora. Nutrition and Cancer, 36: 27-32.
37. Amani, R., J. Baghdadchi and A. Zand-Moghaddam, 2005. Effects of Soy Protein Isoflavones on Serum Lipids, Lipoprotein Profile and Serum Glucose of Hypercholesterolemic Rabbits. International J. Endocrinology and Metabolism, 2: 87-92.
38. Dalais, F.S., P.R. Ebeling, D. Kotsopoulos, B.P. McGrath and H.J. Teede, 2003. The effects of soy protein containing isoflavones on lipids and indices of bone resorption in postmenopausal women. J. Clinical Endocrinology and Metabolism, 58: 704-709.
39. Jenkins, D.J.A., C.W.C. Kendall, C.J.C. Jackson, P.W. Connelly, T. Parker, D. Faulkner, E. Vidgen, S.C. Cunnane, L.A. Leiter and R.J. Josse, 2002. Effects of high- and low-isoflavone soyfoods on blood lipids, oxidized LDL, homocysteine andblood pressure in hyperlipidemic men and women. The American J. Clinical Nutrition, 76: 365-72.
40. Nahas, E.P., J. Nahas-Neto, L. De Luca, P. Traiman, A. Pontes and I. Dalben, 2004. Benefits of soy germ isoflavones in postmenopausal women with contraindication for conventional hormone replacement therapy. Maturitas, 48: 372-380.