ORIGINAL ARTICLE

Lipid Lowering Effect of Almonds *(Prunus Dulcis)* in Healthy Adults

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ABSTRACT

Background: Almonds (*Prunus dulcis*) are low in saturated fats and cholesterol and high in unsaturated fatty acids. Almonds also contain high concentrations of other nutrients like vitamin E, plantsterols, phytochemicals and dietary fibers. Almonds are associated with a reduced risk of cardiovascular disorders (CVD) by having a potentially beneficial impact of on serum lipids due to their nutrient composition.
Aim: To investigate the effect of regular almond consumption on the serum lipid profile of normolipidemic adults.
Methods: In this non-randomized prospective study, 19 normolipidemic adults (10 males, 9 females) with an age range from 21 to 60 years consumed 50 grams of raw almonds for 30 days. Fasting blood samples were collected from each participant at baseline and on the 31st dayfor lipid profile analysis.
Results: Marked decreases in serum total cholesterol level(p-value= 0.000) and serum low-density lipoprotein (LDL) level (p-value= 0.047) were observed from baseline values following almond treatment for a month. An increase in high density lipoprotein (HDL) levelwas also seen but it was not statistically significant (p-value=0.081).
Conclusion: Regular intake of almondscan help maintain a normallipid profile in healthy adults and reduce the risk of CVD. Almond consumption should be encouraged in the local healthy population for improved metabolic and cardiovascular health outcomes.
Keywords: Monounsaturated fats, almonds, humans, lipids

INTRODUCTION

Almonds, the edible seeds of the Almond tree (Prunus dulcis, also referred to as Prunus amygdalus), have been a part of human diet from times immemorial. The nutrient components of almonds primarily include amino acids and proteins, lipids, vitamins, dietary fibers and minerals[1]. Lipids form a major component of the nutritional source of almonds. Lipids constitute almost 50% of the total weight of almonds. They are mainly present as unsaturated fatty acids, predominantly oleic acid and linoleic acid. Oleic acid is about 70% of the lipid component of almonds while linoleic acid is about 20%. Dietary fibers on an average constitute 10% -13% of the total weight of almonds. The major forms of dietary fibers in almonds include cellulose, mucilage, pectin and xyloglucans[2].

The major health benefits of almonds are related to its beneficial impact on lipid profile[3]. Around 90% of the lipids are in the unsaturated form which are cardioprotective by decreasing low-density lipoprotein (LDL) cholesterol and mildly increasing high density lipoprotein (HDL) cholesterol[4,5]. Almonds also contain other cardio-protective components including fibers and plant phytosterols which inhibit endogenous cholesterol biosynthesis as well as reduce absorption of exogenous cholesterol[6]. Another mechanism of the heart-friendly effects of almond intake is the decrease in of oxidative stress. Vitamin E, a fat-soluble vitamin having potent antioxidant function, is present in almonds mainly as α-tocopherol[7]. Almond intake is known to enhancecellular antioxidant defense by increasing the levels of glutathione, catalase and superoxide dismutase in hepatocytes[8,9]. The skin of almonds is rich in antioxidants[10] like polyphenols and flavanols such as catechins[11]. Although some of these positive health effects of almonds are well established, yet the data supporting these beneficial outcomes stem almost entirely from studies conducted in populations whose metabolic norms are quite different from the sub-continental population. There is a need to generate data from local population to completely understand and utilize the potentially beneficial impact of almond consumption. The present study explored the effect of regular almond intake on the lipid profile of normolipidemic individuals in the local population.

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METHODS

This prospective non-randomized study was carried out at Institute of Molecular Biology and Biochemistry (IMBB), University of Lahore (UOL) in collaboration with Jinnah Hospital Lahore and Central Park Medical College between April to September 2016. The study was approved by Ethical Committee. A total of 19 healthy subjects fulfilling the eligibility criteria for the study were selected.Written informed consent was obtained from each participant prior to enrollment. Individuals suffering from chronic illnesses including diabetes mellitus, hypertension, renal disease, cardiovascular disease or known hypersensitivity reactions were excluded from the study.Subjects underwent baseline fasting lipid profile testing and only those subjects were included whose lipid profiles were within the following ranges; serum total cholesterol less then 200mg/dl or equal to it, serum LDL less than 130mg/dl or equal to it, serum fasting triglycerides less then 150mg/dl or equal to it and serum HDL more than 40mg/dl or equal to it. A daily dose 50 mg ofAmerican almonds was given to each subjectfor 30 days consecutively. A post-treatment fasting serum lipid profile test was done on the 31st day.

Serum cholesterol level was determined after hydrolysis and oxidation with cholesterol-esterase and cholesterol-oxidase enzymes[11]. The indicator quinone-imine formed from the resulting hydrogen peroxide and 4-aminophenazone in the presence of phenol and peroxidase. Serum triglycerides level were measured after enzymatic hydrolysis with lipases. The indicator

quinonimine was formed from hydrogen peroxide, 4-amino-antipyrine and 4- chloro-phenol under the catalytic influence of peroxidase. For determination of both serum cholesterol and triglycerides, three cuvettes were labeled as sample, standard and blank. 10µl of sample and standard were pipetted into sample and standard cuvettes. 1000µl of reagent was put into each of the three cuvettes, mixed and incubated for five minutes at 37ºC. The absorbances of the sample and standard were measured against the reagent blank within 60 minutes using specific parameters (wavelength Hg548nm**,** optical path 1cm, temperature 37ºC).

For measurement of HDL, LDL and very low-density lipoprotein (VLDL) were precipitated with phosphotungstate and magnesium ions. 0.2ml of the supernatant fluid and 0.5ml of the reagent were pipetted into centrifuge tubes, mixed and incubated for 10 minutes at room temperature, The sample was then centrifuged for 10 minutes at 4000rpm and the resultant supernatant contained the HDL fraction(12). Supernatant was carefully collected and determination of HDL performed employing the same parameters as described for cholesterol and triglycerides.Serum LDL was calculated by using the formula:

LDL =Total cholesterol –HDL-(TG)/5

(TG)/5 was used as an estimate of VLDL. All values were expressed in mg/dl.

Statistical analysis: The collected data were analyzed using SPSS version 23. Descriptive data were presented as percentages and frequencies. Mean $\pm$ SD was calculated for quantitative variables. Paired sample T-test was performed to observe group mean differences. P-values < 0.05 were considered significant.

RESULTS

The mean age of the study subjects was with a range of 21-60 years. 9/19 of the subjects were males (42.85%) and 12/19 were females (57.14%).

Table 1 and Figure 1 show the serum levels of the studies lipid profile parameters before and after treatment. Mean serum cholesterol level before treatment was 171.263 mg/dl with a range of 142-195 mg/dl. Mean serum cholesterol after treatment was 155.474 mg/dl with a range of 130-193 mg/dl. Serum cholesterol was significantly decreased after treatment (p=0.000) (Table 1, Figure 1). Mean serum LDL level before treatment was 100.0mg/dl with a range of 37-150 mg/dl-Mean serum LDL after treatment was 91.158 mg/dl with a range of 55-140 mg/dl. Serum LDL was significantly decreased after treatment (p=0.047) (Table 1, Figure 1). Mean serum HDL level before treatment was 39.368mg/dl with a range of 32-47 mg/dl. Mean serum HDL after treatment was 42.789 mg/dl with a range of 26-68 mg/dl. The increase in mean serum HDL was not statistically significant (p=0.081) (Table 1, Figure 1). Mean serum triglycerides level before treatment was 150.632 mg/dl with a range of 57-334 mg/dl. Mean serum triglycerides after treatment was 144.158 mg/dl with a range of 76-300mg/dl. Serum triglycerides was significantly decreased after treatment (p =0.196) (Table 1, Figure 1). Mean serum VLDL level before treatment was 28.263 mg/dl with a range of 16-53mg/dl. Mean serum VLDL after treatment was 26.684 mg/dl with a range of 11-49mg/dl. The decrease in mean serum VLDL after treatment was not statistically significant (p=0.272) (Table 1, Figure 1).

Fig. 1. Comparison of mean serum lipid profile before and after treatment with almonds

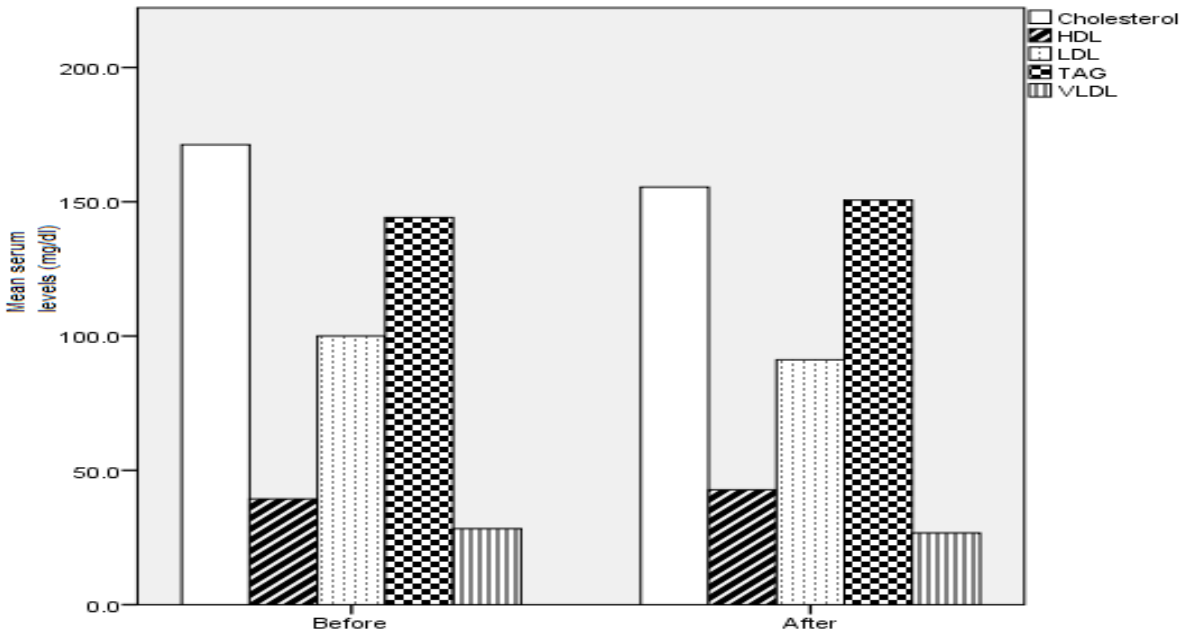

Table 1. Difference in serum lipid profile before and after treatment with almonds

Parameters	Mean Serum Level ± SEM (n=19)		Mean Difference	Trend	p-value
	Before Treatment	After Treatment			
Cholesterol	171.26 ±3.2	155.47 ±4.3	15.78	↓	0.000
LDL	100.00±5.6	91.15 ±4.8	8.8	↓	0.047
HDL	39.36±0.92	42.78 ±1.7	3.4	↑	0.081
Triglycerides	150.63 ±63	144.15 ±51	6.4	↓	0.196
VLDL	28.26 ±14.5	26.68 ±11.89	1.5	↓	0.272

*Difference is significant at $p < 0.05$

DISCUSSION

Almonds have garnered a lot of scientific interest over the years due to their potential health benefits particularly on lipid metabolism. The results of the present study in healthy normolipidemic individuals substantiate the favorable effects of almonds in maintaining a healthy lipid profile. The findings from the current work are consistent with many previous studies which have demonstrated decreases in LDL cholesterol, post-prandial blood glucose, insulin demand and body weight[13]. Almonds, being a rich source of monounsaturated fat, fibers and vitamins,have also been shown to improve the HDL cholesterol levels[14].

Past studies in dyslipidemic individuals with low serum HDL have shown almond intake to cause an increase in HDL levels from baselineespecially in South Asian population[15,16]. Several clinical studies in patients suffering from cardiovascular disease (CVD) have shown that although LDL cholesterol can be adequately lowered with treatment, HDL levels remain low in and are a major factor responsible for the poor health progression observed in these patients despite pharmacologic management based on current guidelines[17,18]. Our findings of the HDL-boosting properties of almonds suggest the possibility of their use in the management of CVDfor improving metabolic and clinical outcomes. Furthermore, almonds have also been shown to offer cardioprotective benefits via several other mechanisms including improvement in renal function, elevation of serum Vitamin E and mitigation of oxidative stress[19,20,21,22].

CONCLUSION

Owing to their unique distribution of biochemical ingredients such as unsaturated fats, flavonoids, vitamin E and dietary fibers, regular daily intake of almonds helps maintain the lipid profile within normal range in healthy individuals.Carefully designed trials in the future may open up way for incorporation of almonds as a key therapeutic component in the management of dyslipidemia and CVD.

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