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Postprandial Effect of Almond (*Prunus dulcis*) Nut on Glucose and Lipid Levels in Apparently Healthy Individuals

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Abstract: ***There is a complex and predominantly unfavorable effect of increased intake of highly processed carbohydrate and lipid profile which may have implications for metabolic syndrome, diabetes, and coronary heart disease. This study aims to determine the effect of almond consumption on postprandial glucose and lipid levels. Fifty (50) volunteers (35 males and 15 females) of varying weights, heights and age group were recruited, and were placed on 12 hours overnight fast. The volunteers were equally divided into two groups (the test and control group), 25 volunteers for each group. The test individuals were given an 830g meal of rice, together with 30 seeds of almond nut, with a 50cl of water while, the control individuals were given the same size of rice with a 50cl of water, but they were not given almond nut. The weight and height of each volunteer were measured, and blood samples (fasting and 2hr postprandial) were collected from each volunteer, analysis for glucose and lipid profile was done on the samples. The control's postprandial low density lipoprotein cholesterol (2.74 ± 1.05mmolL^{-1}) was significantly higher than the test's postprandial LDL-C (1.88 ± 0.87mmolL^{-1}), (p = 0.0036). The control's postprandial total cholesterol level (5.20 ± 1.18mmolL^{-1}) was found to be significantly higher than the test's postprandial TC level (4.19 ± 0.97mmolL^{-1}), (p=0.002). The postprandial glucose of the control (5.02 ± 0.61mmolL^{-1}) was found to be significantly greater than the postprandial glucose of the test (4.44 ± 0.31mmolL^{-1}), (p-value = 9.57E-0.5). No significant difference was observed between the postprandial triglyceride levels of the test and control (p = 0.1338), and the postprandial high density lipoprotein levels of the test and control (p = 0.7998). Almond nut consumption may induce postprandial reduction in human's blood glucose, total cholesterol, and Low density Lipoprotein Cholesterol levels, thereby preventing the complications of diabetes mellitus and cardiovascular diseases and so is recommended as an in-between meal snacks for patients suffering from these diseases.***

Keywords: Postprandial, lipidaemia, diabetes, almond and lipoproteins

1. Introduction

There is a complex and predominantly unfavorable effect of increased intake of highly processed carbohydrate on lipid profile which may have implications for metabolic syndrome, diabetes, and coronary heart disease [1]. Increased carbohydrate consumption and intake of food with a high Glycaemic Index produces higher postprandial glucose and insulin concentrations. Ultimately, this may decrease insulin sensitivity, raising fasting triglyceride concentrations and reducing HDL-C levels [1].

It is becoming increasingly clear that suboptimal blood glucose control results in adverse effects on large blood vessels, thereby accelerating atherosclerosis and cardiovascular disease, manifested as myocardial infarction, stroke, and peripheral vascular disease. Cardiovascular disease is accelerated by both type 1 and type- 2 –diabetes [2].

Observational studies suggest that nut consumption is inversely associated with the incidence of cardiovascular disease and cancer. In addition to being rich in several vitamins and minerals, unsaturated fatty acids, and fiber, nuts contain numerous phytochemicals that may contribute to promoting health and reducing the risk of chronic disease. While many of these bioactive constituents remain to be fully identified and characterized, broad classes include carotenoids, phenols, and phytosterols.

Almonds are good sources of anti-oxidant nutrients. Almonds contain proteins and certain minerals such as calcium and magnesium. They are a rich source of vitamin E, dietary fiber, B-vitamins, essential minerals mono-unsaturated fats and phytosterols which have cholesterol lowering properties. Almonds are a useful food remedy for anaemia. They are beneficial in the treatment of constipation and various skin diseases like eczema, pimples. Almonds are also useful in treating gastro-enteritis, kidney pains, diabetes, head lice, facial neuralgia and gastric ulcers[3]. They are rich in nutrients such as monounsaturated fats, magnesium, protein and vitamin E as well as fiber and phytochemicals [4]. The antilipidemic agent (phytosterol) in almonds helps to lower low density lipoprotein, LDL level and elevate blood levels of high density lipoproteins, HDL[5]. Almonds have also been found to treat kidney stones, gallstones, constipation, stimulate respiration, improve digestion, and helps the body's overall health and well-being. The most interesting medical impact is that almond seed contains a form of Vitamin B which has a positive effect on the treatment of cancer. Studies have showed that animals received almond oil prior to the administration of CCl4, demonstrated that almond oil has potent hepatoprotective effects; and could be developed as a functional food for the therapy and prevention of liver damage.[6]. Almond consumption also reduces 24-hour insulin secretion [7].

Almond seed has the potential for improving the lipid profile in diabetic rat model [8]. Almonds decrease post-prandial glycaemia and oxidative damage in healthy individuals [9]. The flower and seed extracts, at a dose of 500mg/kg, also showed significant reduction ($p < 0.001$) in the blood glucose levels of the diabetic mice on the 15th day of the study [10].

A study by [11] suggests that almonds possess a memory enhancing activity in view of its facilitatory effect on the retention of special memory in scopolamine induced amnesia. The study by[12] showed that almond consumption at 75 g/d for 4 weeks improved time trial distance and the elements related to endurance performance more than did isocaloric cookie consumption in trained Chinese cyclists and triathletes during winter season training when compared to those at the beginning of the training season. This study tends to ascertain whether consumption of almond nuts after meal could have a beneficial postprandial effect on blood glucose and lipids.

2. Materials and Methods

2.1 Experimental Design and Protocol

Fifty (50) volunteers (35 males and 15 females) of varying weights, heights and age group were recruited, and were placed on 12 hours overnight fast. The volunteers were equally divided into two groups (the test and control group), 25 volunteer for each group. The weight and height of each volunteer was measured, and then a fasting (12 hour overnight fast) blood sample was collected from each volunteer.

The test individuals were given an 830g meal of rice together with 30 seeds of almond nut, with a 50cl of water. The control individuals were given the same size of rice like the one given to the test individuals with a 50cl of water, but they were not given almond nut. After two hours of consumption of the meal by both the test and control individuals, a two hours postprandial blood sample was collected from both the test and control individuals, the samples were transferred into an appropriate sample bottle, Glucose estimation was done immediately, while the rest of the sample were separated, and the plasma stored on a refrigerator, the estimation of lipid profile was carried out later.

2.2 Analysis

The estimation of glucose (both fasting and 2hrs post-prandial) was done immediately after sample collection using the glucose oxidase method. Blood samples for Lipid profile (both fasting and 2 hours postprandial) were collected into a Lithium heparin bottle, the samples were separated and the plasma Lipid profile determined.

3. Results

The results of the analysis carried out using student T- test are expressed as Mean ± Standard Deviation.

3.1 Comparison of Blood Glucose Level

Table 4.1: Comparison of Test and Control, FBS and Postprandial Glucose using T-test

Parameters (mmol/L)	*FBS*	*2Hr. P. P. Glucose*	*P-Value*	*Remark*
Test (mean ± S.D)	4.72 ± 0.30	4.44 ± 0.31	0.00214	S
Control (Mean ± S.D)	4.61 ± 0.67	5.02 ± 0.61	0.0265	S
P- Value	0.43797	9.57 E - 0.5		
Remark	NS	S		

There is no significant difference between the fasting blood sugar of the test (4.72 ± 0.30) and that of the control (4.61 ± 0.67 mmol/L), p = 0.43797) while the test's FBS (4.72 ± 0.30 mmol/L) is greater than the test's postprandial glucose (4.44 ± 0.31 mmol/L, p= 0.00214); the control's postprandial glucose (5.02 ± 0.61 mmol/L) is significantly greater than its fasting glucose (4.61 ± 0.67 mmol/L, p = 0.0265), while the postprandial glucose of the control (5.02 ± 0.61 $mmolL^{-1}$) is significantly greater than that of the test (4.44 ± 0.31 $mmolL^{-1}$, p = 9.57 E - 0.5).

3.2 Comparison of Total Cholesterol Level

Table 4.2: Comparison of Test and Control, Fasting TC and Postprandial TC Using T-test

Parameters (mmol/L)	*Fasting TC*	*2Hr. P. Prandial TC*	*P-Value (2-tail)*	*Remark*
Test (mean ± S.D)	4.94 ± 1.23	4.19 ± 0.97	0.0211	S
Control (Mean ± S.D)	4.48 ± 1.23	5.20 ± 1.18	0.0326	S
P- Value (2-tail)	0.1726	0.002		
Remark	NS	S		

The fasting total cholesterol level of the test is 4.94 ± 1.23$mmolL^{-1}$; that of the control is 4.48 ± 1.23$mmolL^{-1}$. While the postprandial TC level of test is 4.19 ± 0.97$mmolL^{-1}$ and that of control is 5.18 ± 1.18$mmolL^{-1}$. There is no significant difference between the test and control fasting TC level (P = 0.1726), but the control's postprandial TC level is significantly greater than the test's postprandial TC level (p = 0.002), while the test's fasting TC is significantly greater than the test's postprandial TC level (P = 0.0211); and the control's postprandial TC is significantly greater than its fasting TC (p = 0.0326).

3.3 Comparison of Triglyceride Level

Table 4.3: Comparison of Test and Control, Fasting Tg and Post Prandial Tg using T-test

Parameters (mmol/L)	*Fasting Tg*	*2Hr. P. Prandial Tg*	*P-Value (2-tail)*	*Remark*
Test (mean ± S.D)	0.83 ± 0.26	0.92 ± 0.40	0.3568	NS
Control (Mean ± S.D)	0.90 ± 0.39	1.08 ± 0.37	0.0956	NS
P- Value (2- tail)	0.4436	0.1338		
Remark	NS	NS		

There is no significant difference between the test's fasting Triglyceride level (0.83 ± 0.26mmolL^{-1}) and control's fasting Triglyceride level (0.90 ± 0.39 mmolL^{-1},p = 0.4436), and no significant difference between the test's postprandial Triglyceride level (0.92 ± 0.41mmolL^{-1}) and control's postprandial Tg (1.08 ± 0.37mmolL^{-1},p = 0.1338). There are also no significant differences between the control's fasting and postprandial Tg (p = 0.0956), and the test's fasting and postprandial Tg (p = 0.3568).

3.4 Comparison of High Density Lipoprotein Cholesterol Level

Table 4.4: Comparison of test and control, fasting HDL-C and post prandial HDL-C

Parameters (mmol/L)	*Fasting HDL-C*	*Post Prandial HDL-C*	*P-Value (2-tail)*	*Remark*
Test (mean ± S.D)	1.90 ± 0.50	1.91 ± 0.38	0.9898	NS
Control (Mean ± S.D)	1.93 ± 0.36	1.94 ± 0.43	0.9972	NS
P- Value (2- tail)	0.8051	0.7998		
Remark	NS	NS		

There is no significant difference between the test's fasting high density lipoprotein cholesterol (1.90 ± 0.50mmolL^{-1}) and its post prandial HDL-C (1.91 ± 0.38mmolL^{-1}, p=0.9898) and no significant difference between the controls fasting HDL-C (1.93 ± 0.36mmolL^{-1}) and its post prandial HDL-C level (1.94 ± 0.43mmolL^{-1}), (P = 0.9972). There are also no significant difference between the test and control postprandial HDL-C, (p = 0.7998) and no significant difference between the control's fasting HDL-C and the test's fasting HDL-C (p = 0.8051).

3.5 Comparison of Low Density Lipoprotein Cholesterol Level

Table 4.5: Comparison of test and control, fasting LDL-C and postprandial LDL-C

Parameters (mmol/L)	*Fasting LDL-C*	*Postprandial LDL-C*	*P-Value (2-tail)*	*Remark*
Test (mean ± S.D)	2.67 ± 1.03	1.88 ± 0.87	0.0064	S
Control (Mean ± S.D)	2.18 ± 1.05	2.74 ± 1.05	0.0693	NS
P- Value (2-tail)	0.1064	0.0036		
Remark	NS	S		

The test's Fasting Low density lipoprotein cholesterol (2.67 ± 1.03mmolL^{-1}) is greater than the test's postprandial LDL-C (1.88 ± 0.94mmolL^{-1}, p = 0.0064), while there is no significant difference between the control's fasting LDL-C (2.18 ± 1.05mmolL^{-1}) and control's postprandial LDL-C (2.74 ± 1.05mmolL^{-1}, p = 0.0693). There is also no significant difference between the test's fasting LDL-C and control's fasting LDL-C (p = 0.1064), while the control's postprandial LDL-C is significantly greater than the test's postprandial LDL-C (p = 0.0036).

4. Discussion

There was a significant postprandial reduction in the glucose level of the test samples after consumption of the almond nuts. In comparison with the control, though the test's and control's FBS has no significant difference, the control postprandial glucose was noticed to be greater than that of the test, more so. These results confirmed that consumption of almond nut reduces blood glucose in humans; almond has a hypoglycaemic capacity on humans. This fact has also been proved in previous researches by [4, 10].

It was also discovered that almond induces a significant decrease in the total cholesterol level of the test individual, and also induces a reduction in the level of low density lipoprotein cholesterol in the test individuals. Almond seed has the potential of improving the lipid profile in diabetic rat model [8]. This is due to the fact that almond are poor in saturated fatty acids and rich in unsaturated fatty acids, and because of the antilipidaemic agent known as phytosterols which is contained in almond, (phytosterols inhibit the absorption of dietary cholesterol and lower serum cholesterol as well as antagonize selected inflammatory pathways). These facts have also been proved in previous researches by[13,14,15 and 5].

From the data and results obtained, almond consumption did not show any significant effect on the Triglyceride and high density lipoprotein cholesterol levels of the individuals; this is because almond consumption does not

cause an immediate alteration on serum triglyceride and HDL-C levels.

5. Conclusion

In conclusion, there is a complex and predominantly unfavorable effect of increased intake of highly processed carbohydrate on lipid profile, which may have implications for metabolic syndrome, diabetes, and coronary heart disease[1] (Ma, *et al.,* 2006). It is becoming increasingly clear that suboptimal blood glucose control results in adverse effects on large blood vessels, thereby accelerating atherosclerosis and cardiovascular disease, manifested as myocardial infarction, stroke, and peripheral vascular disease [2].

Almond consumption induces a post prandial reduction in human's blood glucose, total cholesterol, and Low density Lipoprotein Cholesterol levels, and so is helpful in the treatment of diabetes, cardiovascular diseases, atherosclerosis, hyperlipidaemia and coronary heart disease.

Based on the facts provided, almond nut consumption is recommended as an in-between meal snacks for patients suffering from diabetes, obesity, cardiovascular disease, atherosclerosis, and coronary heart disease. Due to the high potassium, phosphorus content, moderate magnesium content and the low sodium content of almond, more research on the effect of almond on electrolytes are recommended. From findings published by USDA National Nutrient Database for Standard Reference, it was shown that almond has 0µg of Vitamin K (phylloquinone). Vitamin K is required by the liver for the production of prothrombin (factor II) and factors VII, IX and X, which are all essential for the clotting of blood [16]. So therefore more research on the effect of almond on coagulation and blood clotting is recommended.

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