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Effect of Peanut Consumption on Nutritional Status Indices of HIV Infected Adults in Nyeri County, Kenya

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Abstract **Introduction and objectives;** Peanuts are a rich source of magnesium, folate, fibre, α-tocopherol, copper, arginine and resveratrol. These compounds have been shown to reduce the CVD risk in various ways. The purpose of this study was to investigate the effect of peanut supplementation on nutrition status of HIV-infected adults attending comprehensive care clinic in Nyeri Level- 5- Hospital. **Methodology**; The study design was a randomized cross-over trial. The eligible participants were randomly assigned to a two arm study. In treatment I, the participants consumed their regular diet supplemented with 80gms of peanuts; while in treatment II, the participants were counseled on healthy diet and supplemented it with 80gms of peanut. The participants then crossed over to respective treatments. Each treatment took 8 weeks, with a six weeks washout period between treatments. A paired T- test was used to compare subject differences in markers at baseline and at the end of each treatment. Multiple regression analysis was used to determine the effect of peanut supplementation on nutrition status. **Results**; Peanut supplementation significantly increased intake of total fat while carbohydrate intake decreased significantly ($p < 0.05$). There was no significant change in weight, BMI, waist circumference, hip circumference, body fat, body muscle, systolic and diastolic blood pressure and fasting blood glucose. There was a significant decrease ($p < 0.05$) in total cholesterol, triglycerides and Low density lipoprotein in both treatments while High density lipoprotein increased significantly ($p < 0.05$). **Conclusion**; Regular supplementation of a healthy diet with 80gms of peanut may improve the lipid profile without affecting the body weight status.

Keywords: *peanut consumption, weight, blood glucose, lipid profile and blood pressure, nutrition status indices*

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1. Introduction

Nuts are from different plant families and are classified as tree nuts (a one-seeded fruit with a hard shell) or peanuts (a member of the leguminous family). Peanuts are also called ground nuts because they develop in the soil. Despite their diversity tree nut varieties share common nutritional characteristics with peanut. Peanuts are nutrient dense foods and also contain a high fat content half of which is unsaturated, which includes monounsaturated fatty acids (oleic) and polyunsaturated fatty acids. [1] The fatty fraction of nuts also contains a sizable amount of plant sterols, with anti-oxidant [2] and cholesterol lowering properties [3]. They are also rich sources of other bioactive macronutrients that include protein and fibre. They contain high amounts of L-arginine which is the amino acid precursor of the endogenous vasodilator nitric acid [4].

Epidemiologic studies on prevention of diabetes, coronary heart disease and sudden deaths have found that unsaturated fatty acids contribute to the beneficial associations of frequent nut intake and decreases in other CVD risk factors. Nuts form a complex food matrix that are sources of other bioactive compounds which include fiber and protein; micronutrients, such as potassium, calcium, magnesium, and tocopherols; phytochemicals, such as phytosterols and phenolic compounds; arginine and resveratrol [1]. The beneficial effects of nuts on cardiovascular diseases can be explained by the composite and individual cardio protective nutrients in the nuts.

Nuts are cholesterol-free, but their fatty fraction contains sizeable amounts of chemically related non cholesterol sterols belonging to a heterogeneous group of compounds known as plant sterols or phytosterols [3]. They are non-nutritive components of all plants that play an important structural role in membranes, where they serve to stabilize phospholipids' bilayers just as cholesterol does in animal cell membranes [5]. Phytosterols interfere with cholesterol absorption and thus help lower blood cholesterol when present in sufficient amounts in the intestinal lumen.

2. Methodology

The purpose of this study was to investigate the effect of peanut supplementation on nutrition status in HIV-infected adults. The study design was a randomized cross-over clinical trial. The study duration was 22 weeks. A sample of 85 participants was randomly selected. The participants were then randomly assigned to two groups. Group 1 started with TI and crossed over to TII, while group 2 started with TII and crossed over to TI after the washout period.

During treatment I (TI) the participants were required to go on consuming their regular diet supplemented with 80g of peanuts daily for eight weeks. Their nutritional status was assessed at baseline and after eight weeks. During the second treatment (TII) the participants were counseled on a healthy diet at baseline and then after four weeks before they picked the next batch of peanut. Counseling covered aspects such as substituting the saturated fat with unsaturated fats, consumption of high fibre foods and inclusion of all the WHO recommended 10-12 food groups in their proper quantities... The participants were also provided 80g of peanuts daily for eight weeks. A six weeks washout period was allowed between treatments. Their nutritional status was assessed at baseline and again after eight weeks.

Dietary intake was assessed using a 24-hour recall that was conducted on three randomly selected days during baseline, treatment I and treatment II. This was to help measure nutrient intake. Anthropometric measurements were taken using different equipment. Body weight and body composition were measured using a bio electric impedance machine, Height was measured using a standiometer while waist and hip circumference were measured using an elastic tape.

Blood pressure and heart rate were measured with an automatic blood pressure monitor (Visomat® Comfort 20/40, Roche Diagnostics) during each visit.

Fasting blood samples was collected in the morning between 7.00 and 8.00 am. Capillary blood was collected and tested for fasting blood glucose while approximately 5 ml of venous blood was collected for fasting lipid profile and transferred to heparinized collecting tubes. Blood in the tubes was centrifuged at 3000g for 3 min. Separation of serum and plasma was done using an automatic pipette and transferred into specific labeled tubes in a rack ready for analysis. A drop of capillary blood specimen was obtained from a sterilized fingertip area using a lancing device. Lipid profile assays were routinely analyzed on Mindray BSseries auto analyzer (Mindray-Bio Medical GmbH, Hamburg, and Germany) using established techniques.

Diabetes risk classification [6] was adopted. Classification of overweight and obesity and waist hip ratio [7] was used. Executive Summary of the Third Report of the National Cholesterol Education Program (ATP III) (2001) [8] guidelines were used to classify risk for lipid profile. Student T-tests was used to assess whether there was significant difference on the lipid profile and BMI at baseline and end of each treatment. Multiple regression analysis was used to determine the effect of peanut supplementation and nutrition counseling on the nutrition status.

Ethical clearance was sought from Kenyatta University Ethical Review Committee (REF:KU.R/COMM/51/273), permission was sought from NACOSTI (REF: NCST/RCD/12A/013/4) and informed consent obtained from the study participants.

3. Results

Age, marital status, education level were not significantly different ($p>0.05$) between group 1 that started with treatment I, and group 2 that started the intervention with treatment II. This indicated that the social economic status did not have an effect on the impact of the two treatments offered

3.1. Dietary Intake

Table 1 shows the mean change in the dietary intake when peanut was added to regular diet (T I) and when counseling in healthy diet plus 80g of peanut were given daily for the duration of the treatment (T II). There was a statistically significant difference in fat intake ($F(2, 48) = 13.185$, $p < 0.05$) between baseline and the two treatments. The same was found for carbohydrate intake ($F(2, 48) = 11.664$, $p < 0.05$), polyunsaturated fatty acid intake ($F(2, 48) = 55.091$, $p < 0.05$), Vitamin E intake ($F(2, 48) = 37.614$, $p < 0.05$) and mono unsaturated fatty acids intake ($F(2, 48) = 34.328$, $p < 0.05$). Compared with baseline, energy intake from fat increased significantly during TI and TII (both $p< 0.001$), MUFA and PUFA increased significantly during TI and TII (all p< /0.001), while SFA remained unchanged. Non-fatty acid lipid materials such as sterols were not measured. There was a significant decrease of carbohydrate intake during TI and TII (both $p < /0.001$ and $p < 0.001$), respectively. Dietary intakes of vitamin E ($p < 0.001$) increased significantly from baseline in Treatment I as well as in treatment II (vitamin E p </0.001).

Table 1. Estimated mean daily energy and nutrient intakes from three random-day 24-h recall

	Baseline	T1	T2
Energy (kcal/day)	1937.10±309.98 [a]	2056.02±224.12[a]	2091.99±307.47[a]
Fat (%energy)	21.82±6.22[a]	32.05±7.64[b]	32.76±6.91 [b]
SFA	14.61±9.33[a]	19.39±4.51 [a]	19.39±5.72 [a]
MUFA	16.33±7.95[a]	32.53±6.17 [b]	33.19±5.84 [b]
PUFA	8.89±3.61[a]	17.86±2.66 [b]	19.16±2.96 [b]
Cholesterol (mg)	118.91±157.18[a]	118.12±211.06[a]	103.34±206.23 [a]
Protein(% energy)	12.23±2.56[a]	13.88±3.19[a]	13.35±2.47 [a]
Carbohydrate (% energy)	66.00±7.77[a]	54.23±9.71 [b]	53.64±7.58 [b]
Vitamin E	3.25±2.70[a]	8.87±2.13 [b]	8.80±1.48 [b]
Folate (mg/day)	313.89±188.11[a]	387.40±229.22 [a]	395.12±230.52 [a]
Magnesium (mg/day)	489.68±102.89[a]	592.53±142.34 [a]	618.22±248.03 [a]
Carotene	456.32±1103.60[a]	2149.58±4765.67 [a]	1892.89±4173.92 [a]
Dietaryfibre (g/day)	24.61±8.76[a]	29.57±10.78 [a]	31.87±9.11 [a]

All these changes can be attributed to inclusion of peanuts in the diet. However folate and magnesium did not change significantly in both treatments from baseline. There was no significant difference between the dietary intake in treatment I and treatment I.

Values presented as the mean±/standard deviation; n=17. Means with different superscript letters are statistically significant at (P<0.05). PUFA-poly unsaturated fatty acid, MUFA- mono unsaturated fatty acids, SFA - saturated fatty acid.

3.2. Nutritional Status Markers

The two groups were not significantly different at baseline for all the nutrition status markers.

Table 2 shows change in nutritional status markers for Treatment 1 and Treatment II.

Weight, body fat, waist circumference and fasting blood glucose increased slightly in treatment I but decreased in treatment II. There was no statistically significant change in weight, BMI, waist circumference, hip circumference, body fat, body muscle, systolic and diastolic blood pressure and fasting blood glucose after consumption of peanut with regular diet and consumption of peanut combined with nutritional counseling on healthy diet. There was no statistically significant difference in nutritional status between the two treatments.

Values presented as the mean±/standard deviation; n=85. Means with same superscript letters are not statistically significant at (P< 0.05). BMI- body mass index, BF- body fat, LBM- lean body muscle, W.C- waist circumference, H.C-hip circumference, SBP- systolic blood pressure, DBP-diastolic blood pressure, FBG- fasting blood glucose

Regression analysis did not establish relationship between the changes in weight, BMI, waist and fasting blood glucose in treatment I and treatment II and the changes in energy, fat and carbohydrate intake as predicted.

3.3. Mean Change in Serum Lipid Profile

There was a 3.07% decrease in total cholesterol in treatment I while the decrease in treatment II was 5.39% (Figure 1). The decrease was significant in both treatment I and II (p<0.001). The mean change between the two treatments was also significant (p < 0.001).

There was also a decrease in triglycerides in treatment 1 of 12.81% while in treatment 2 it was 17.01% (Figure 1). The mean change between treatments was however not significant (p=0.121). There was a significant but slight increase in HDL-C in treatment I and II at 7.38% and 5.1% respectively (Figure 1). The mean change between the two treatments was also significant (p = 0.012).

The decrease in LDL-C was 5.56% in treatment I and 4.32% in treatment II (Figure 1). The change was significant at (p<0.001) in both treatments. The mean change between the two treatments was not significant (p =0.242). The mean reduction in total cholesterol was found to be higher for participants who had TC >5.1 than those who had TC less than 5.1mmol/L. The mean reduction in triglycerides was found to be higher for participants who had TAG >2.25 than for those with TAG less than 2.25. The mean reduction in LDL-C was found to be higher for participants who had LDL-C > 4.2mmol/L than for those with less than 4.2mmol/L. Mean increase in HDL-C was higher in participants who had normal levels of HDL-C (1.03-1.55) and it was least in participants who had high HDL-C (> 1.55). Total cholesterol and HDL-C were statistically different (p = 0.05) between the two treatments indicating the effect of nutritional counseling.

Table 2. Change in nutritional status markers for Treatment 1 and Treatment II

	Treatment I		Treatment II	
	Baseline	Week 8	Baseline	Week 8
Weight (kg)	72.12±13.47[a]	72.32±13.73[a]	72.27±13.46[a]	72.15±13.60[a]
BMI (kg/m^2)	27.39±5.20[a]	27.45±5.31[a]	27.4585±5.22[a]	27.39±5.30[a]
BF (%)	30.37±8.42[a]	33.96±20.85[a]	32.62±13.15[a]	31.12±9.12[a]
LBM (%)	32.26±4.22[a]	31.92±4.28[a]	35.35±29.27[a]	31.96±4.23[a]
WC(cm)	91.78±10.86[a]	92.45±10.58[a]	92.37±11.17[a]	91.80±10.69[a]
HC(cm)	102.31±10.42[a]	101.85±9.76[a]	101.79±11.24[a]	100.50±10.62[a]
SBP(mmHg)	135.11±18.36[a]	134.57±16.40[a]	134.41±17.64[a]	134.09±20.25[a]
DBP(mmHg)	81.71±9.83[a]	81.5882±10.22[a]	81.48±12.46[a]	80.82±10.68[a]
FBG(mg/dl)	81.68±28.99[a]	86.36±32.84[a]	88.32±24.36[a]	88.69±35.84[a]

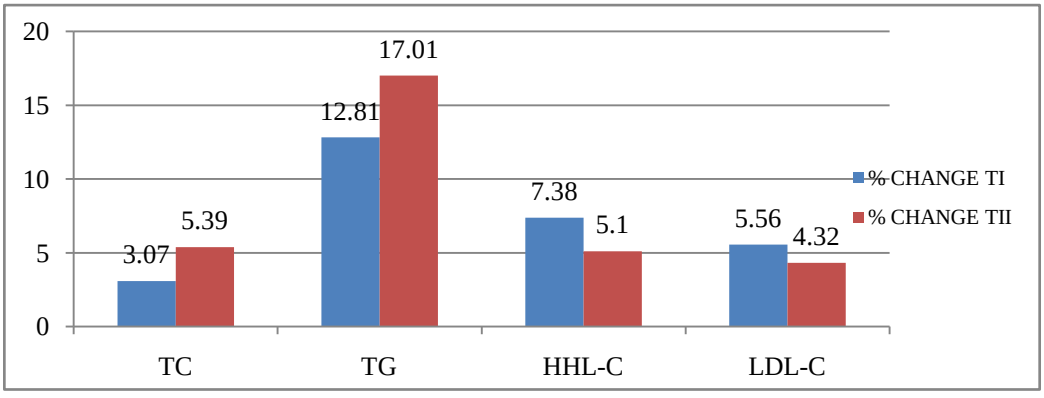

Figure 1. Percentage change in lipid profile in the two treatments

Table 3. Mean change in serum lipid profile

	Treatment I			Treatment II			D1 &D2
	Baseline	End	P value (t-test)	Baseline	end	P value (t-test)	P value (t-test)
TC (mmol/L)	5.17±1.18	5.01±1.07	0.001	5.17±1.13	4.89±1.08	0.001	0.001
TG (mmol/L)	1.88±.85	1.64±.83	0.001	1.89±.90	1.57±.94	0.001	0.121
HDL- C (mmol/L)	1.40±.41	1.51±.42	0.001	1.42±.42	1.49±.42	0.001	0.012
LDL-C (mmol/L)	3.31±1.01	3.12±.92	0.001	3.25±1.00	3.11±.99	0001	0.242

Values presented as the mean±/standard deviation; n=85. Means are statistically significantly different at (P< 0.05). TC-total cholesterol, TG-triglycerides, HDL-C-high density lipoprotein cholesterol, LDL-C-Low density lipoprotein cholesterol, D1-delta change in treatment I, D2- delta change in treatment II.

Regression analysis did not establish relationship between the changes in lipid profile in treatment I and treatment II and the change in poly unsaturated fatty acid, monounsaturated fatty acid and saturated fatty acid intake as predicted.

4. Discussion

Average consumption of energy at baseline was below the RDAs for both males and females (3139 ±365 Kcals and 2479 ±312Kcals respectively. The results are similar to [9] in a study in HIV infected adults in Mweiga, Nyeri County.

This study was an interventional cross over study. Eighty grams (80g) of peanut was given to be taken together with regular diet in treatment I. The same amount was given in treatment II but the participants were counseled on healthy diet. Energy intake did not change significantly when the peanuts were supplemented to regular diet and healthy diet. These findings agree with [10] Alper & Mattes, 2003 who found similar results in a 30-week crossover intervention where subjects were provided 500 (±136) kcal as peanuts during an eight-week free feeding (FF) diet. Mckiernan, et al, 2010 [11] also did not report significant changes in energy intake in their 4 week randomized clinical trial.

There were no significant differences between peanut supplementation on healthy diet versus peanut supplementation with regular diet at baseline with respect to nutritional status markers. There was also no significant time period effect respect to change in nutritional status markers following the intervention. This study did not report significant difference in weight, BMI, waist and hip circumference and fasting blood glucose during the two treatments with peanut. This was not expected given that 80g of peanut was expected to contribute an extra approximately 500Kcal/day. A large cohort of women followed for 16 years found a slight decrease in the body mass index (BMI) even as the consumption of nut increased. After adjustment for potential confounders, their average weight gain across nut consumption categories was not significantly different [12].

Other studies have found no net weight gain when nuts are consumed as a replacement food. The same has been reported even when nuts have been added to diet, even though the intake of total energy was substantially [13,14].

No weight gain was reported when 48 g of walnuts was added to the diet for six weeks despite increase in energy intake by 1661 kJ/day [15]. Since the nut intervention duration is relatively short in most of these trials, the long-term effect of nut intake may not be indicated. However an isolated intervention study showed a negative effect on body weight. A slight but significant increase in body weight (0.9 kg for men, 0.3 kg for women) was observed when normal weight participants were given 100g of almonds to add to their usual diet for a period of four weeks [16].

There are several explanations why nut intake might protect against weight gain. Nuts are rich in fiber and are energy-dense, high fat foods, with a high content of unsaturated fatty acids. Evidence suggests that monounsaturated and polyunsaturated fatty acids are more readily oxidized [17] and have a greater thermogenic effect [18] than saturated fatty acids, which can lead to less fat accumulation. Nuts are also good sources of plant proteins, which may enhance satiety and suppress subsequent hunger [19]. A high content of dietary fiber, from both vegetables and nuts, is believed to increase satiety and reduce feelings of hunger [20]. Furthermore, fat malabsorption has been reported after nut intake and attributed to the fat being contained within walled cellular structures that are incompletely digested in the gut, [21] an effect that can be compounded by incomplete mastication [22]. Finally, other mechanisms of protection against adiposity may depend on many other bioactive compounds that are present in nuts [3].

Epidemiological studies and clinical trials have demonstrated benefits of nuts and peanut consumption on CAD risk and associated risk factors. [23,24] These findings agree with a study by [25] who reported a 7.2% decrease in total cholesterol and 20% decrease in triglycerides when 500kcal/day peanut was incorporated with daily diet for eight weeks. A recent study by [11] reported significant reductions in total cholesterol, LDL-C and TAG concentrations were observed when hyperlipidemic individuals consumed 56 g of whole raw, roasted unsalted, roasted salted or honey roasted peanuts, or ground peanut butter daily for 4 weeks. However HDL-C concentrations increased significantly from baseline. Lokko et al (2007) [25] also reported significant decrease in total cholesterol (7.2%) and triacylglycerol (20.0%) after subjects were provided 2,092 kJ/day (500 kcal/day) peanuts to incorporate into their daily diet for 8 weeks at any time and in any form they chose. However, individually, high-density lipoprotein-cholesterol and low-density lipoprotein-cholesterol levels did not change significantly. Recently the findings of a pooled analysis of 1,284 observations contributed by 583 unique participants from 25 clinical studies performed with different nuts, including peanuts, and conducted in seven different countries have been reported. [26] The results show a dose-response cholesterol lowering effect and indicate that, for an average daily intake of 67 g of nuts

(roughly equivalent to 20% of energy), the mean estimated reductions of total cholesterol and LDL-cholesterol were 11 mg/dL (5%) and 10 mg/dL (7%), respectively. Nuts had no significant effect on HDL-cholesterol or triglycerides, except in participants with serum triglycerides >150 mg/dL, in whom a significant 10.2 mg/dL reduction was observed

In this study the mean decrease in serum total cholesterol, Low density lipoprotein and triglyceride was higher but not significantly higher in participants with high levels compared to those with normal serum levels. This is in contrast to a peanut intervention study that reported a 12% reduction in total cholesterol and a 10% reduction in LDL-C in normocholesterolaemic individuals consuming whole peanuts and peanut butter for 24days. [27] However reductions among normocholesterolaemic individuals were greater in those with the highest concentrations at baseline [27].

The study did not find a significant relationship between the changes in dietary PUFA, MUFA, dietary fat and fibre (individually or together) and the changes in the lipid profile after linear regression analysis. This is because there are other component in nuts such as fibre and phytosterols together with unsaturated fatty acid profile that are likely to contribute to the favourable effects nuts have on the plasma lipid [28,29]. The decrease in the triglycerides in this study may be due to the reduction in carbohydrate intake when the peanuts were added to the diet. Triglyceride concentration decreases with reduction in carbohydrate intake [30] and thus, the decreases in carbohydrate intake reported may have had an independent effect on lipid concentrations. Another decrease in triglyceride by 1.0 mmol/l may result in 14-37% decrease in overall CVD risk [31].

The fatty acid composition of nuts is suspected to play a role in modifying insulin resistance, and therefore, the risk for type-2 diabetes. Specific types of fatty acids have been found to be better predictors of the risk of type-2 diabetes than total dietary fat intake [32]. Studies have shown that a higher intake of n-3 PUFA is linked with lower risk of type-2 diabetes, while on the other hand, glycemic control is adversely affected by a higher intake of saturated and trans fatty acid hence increasing the risk of type-2 diabetes [32,33].

Compared to other common foods, nuts have an optimal nutritional density with respect to healthy minerals, such as calcium, magnesium, and potassium. Like that of most vegetables, the sodium content of raw or roasted but otherwise unprocessed nuts is very low, ranging from undetectable in hazelnuts to 18 mg/100 g in peanuts [3]. A high intake of calcium, magnesium and potassium, together with a low sodium intake, is associated with protection against bone demineralization, arterial hypertension, insulin resistance, and overall cardiovascular risk [34].

5. Conclusion

Consumption of peanut with regular diet or with counseling on healthy diet improves the lipid profile in people living with HIV and therefore reduces the 10 year risk of developing CHD. Peanut supplementation had no significant effect on biomarkers for weight gain.

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Conflict of Interest

The authors do not have any conflict of interest.

References

[1] Kris-Etherton PM, Yu-Poth S, Sabaté J, Ratcliffe HE, Zhao G, Etherton TD. Nuts and their bioactive constituents: effects on serum lipids and other factors that affect disease risk. *The American journal of clinical nutrition*. 1999 Sep 1; 70(3): 504s-11s.

[2] Vivancos M, Moreno JJ. β-Sitosterol modulates antioxidant enzyme response in RAW 264.7 macrophages. *Free Radical Biology and Medicine*. 2005 Jul 1; 39(1): 91-7.

[3] Segura R, Javierre C, Lizarraga MA, Ros E. Other relevant components of nuts: phytosterols, folate and minerals. *British Journal of Nutrition*. 2006 Nov; 96(S2): S36-44.

[4] Huynh NN, Chin Dusting J. Amino acids, arginase and nitric oxide in vascular health. *Clinical and experimental pharmacology and physiology*. 2006 Jan; 33(1-2): 1-8.

[5] Hartmann MA. Plant sterols and the membrane environment. *Trends in plant science*. 1998 May 1; 3(5): 170-5.

[6] MELLITUS D. Diagnosis and Classification of Diabetes Mellitus. *DIABETES CARE*. 2008 Jan 1; 31: S55

[7] Consultation WE. Waist circumference and waist-hip ratio. Report of a WHO Expert Consultation. Geneva: *World Health Organization*. 2008 Dec; 2008: 8-11.

[8] Executive Summary of the Third Report of the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, And Treatment of High Blood Cholesterol In Adults (Adult Treatment Panel III). JAMA 2001; 285: 2486-2497

[9] Zipporah NW, Elizabeth K, Nicholas G, Dorcus M. Contribution of Amaranth Grain (A. Cruentus) on dietary intake and nutritional status of adults living with HIV in Mweiga, Nyeri, Kenya. *J Basic Appl Sci Res*. 2014; 4(4).

[10] Alper CM, Mattes RD. Peanut consumption improves indices of cardiovascular disease risk in healthy adults. *Journal of the American College of Nutrition*. 2003 Apr 1; 22(2): 133-41.

[11] McKiernan F, Lokko P, Kuevi A, Sales RL, Costa NM, Bressan J, Alfenas RC, Mattes RD. Effects of peanut processing on body weight and fasting plasma lipids. *British journal of nutrition*. 2010 Aug; 104(3): 418-26.

[12] Jiang R, Manson JE, Stampfer MJ, Liu S, Willett WC, Hu FB. Nut and peanut butter consumption and risk of type 2 diabetes in women. *Jama*. 2002 Nov 27; 288(20): 2554-60.

[13] Morgan WA, Clayshulte BJ. Pecans lower low density lipoprotein cholesterol in people with normal lipid levels. *Journal of the American Dietetic Association*. 2000 Mar 1; 100(3): 312-8.

[14] Morgan J, Horton K, Reese D, Carey C, Walker K, Capuzzi D. Effects of walnut consumption as part of a low-fat, low-cholesterol diet on serum cardiovascular risk factors. *International journal for vitamin and nutrition research*. 2002 Oct 1; 72(5): 341-7.

[15] Almario RU, Vonghavaravat V, Wong R, Kasim-Karakas SE. Effects of walnut consumption on plasma fatty acids and lipoproteins in combined hyperlipidemia. *The American journal of clinical nutrition*. 2001 Jul 1; 74(1): 72-9.

[16] Lovejoy JC, Most MM, Lefevre M, Greenway FL, Rood JC. Effect of diets enriched in almonds on insulin action and serum lipids in adults with normal glucose tolerance or type 2 diabetes. *The American journal of clinical nutrition*. 2002 Nov 1; 76(5): 1000-6.

[17] Piers LS, Walker KZ, Stoney RM, Soares MJ, O'dea K. The influence of the type of dietary fat on postprandial fat oxidation rates: monounsaturated (olive oil) vs saturated fat (cream). *International journal of obesity*. 2002 Jun; 26(6): 814-21.

[18] Casas-Agustench P, López-Uriarte P, Bulló M, Ros E, Gómez-Flores A, Salas-Salvadó J. Acute effects of three high-fat meals with different fat saturations on energy expenditure, substrate oxidation and satiety. *Clinical Nutrition*. 2009 Feb 1; 28(1): 39-45.

[19] Garcia-Lorda P, Rangil IM, Salas-Salvado J. Nut consumption, body weight and insulin resistance. *European Journal of Clinical Nutrition*. 2003 Sep; 57(1): S8-11.

[20] Salas-Salvadó J, Bulló M, Pérez-Heras A, Ros E. Dietary fibre, nuts and cardiovascular diseases. British *Journal of Nutrition*. 2006 Nov; 96(S2): S45-51.

[21] Ellis PR, Kendall CW, Ren Y, Parker C, Pacy JF, Waldron KW, Jenkins DJ. Role of cell walls in the bioaccessibility of lipids in almond seeds. *The American journal of clinical nutrition*. 2004 Sep 1; 80(3): 604-13.

[22] Cassady BA, Hollis JH, Fulford AD, Considine RV, Mattes RD. Mastication of almonds: effects of lipid bioaccessibility, appetite, and hormone response. *The American journal of clinical nutrition*. 2009 Mar 1; 89(3): 794-800.

[23] Ros, E. (2009). Nuts and novel biomarkers of cardiovascular disease. *The American journal of clinical nutrition*, 89(5), 1649S-1656S.

[24] Djoussé L, Rudich T, Gaziano JM. Nut consumption and risk of hypertension in US male physicians. *Clinical Nutrition*. 2009 Feb 1; 28(1): 10-4.

[25] Lokko P, Lartey A, Armar-Klemesu M, Mattes RD. Regular peanut consumption improves plasma lipid levels in healthy Ghanaians. *International journal of food sciences and nutrition*. 2007 Jan 1; 58(3): 190-200.

[26] Sabaté J, Oda K, Ros E. Nut consumption and blood lipid levels: a pooled analysis of 25 intervention trials. *Archives of internal medicine*. 2010 May 10; 170(9): 821-7.

[27] Kris-Etherton PM, Yu-Poth S, Sabaté J, Ratcliffe HE, Zhao G, Etherton TD. Nuts and their bioactive constituents: effects on serum lipids and other factors that affect disease risk. *The American journal of clinical nutrition*. 1999 Sep 1; 70(3): 504s-11s.

[28] Kris-Etherton, P.M., Hu, F.B., Ros E., et al. (2008). The role of tree nuts and peanuts in the prevention of coronary heart disease: multiple potential mechanisms. *Journal of Nutrition* 138, S1746-S1751.

[29] Griel, A. E., & Kris-Etherton, P. M. (2006). Tree nuts and the lipid profile: a review of clinical studies. *British Journal of Nutrition*, 96(S2), S68-S78.

[30] Appel LJ, Sacks FM, Carey VJ, Obarzanek E, Swain JF, Miller ER, Conlin PR, Erlinger TP, Rosner BA, Laranjo NM, Charleston J. Effects of protein, monounsaturated fat, and carbohydrate intake on blood pressure and serum lipids: results of the OmniHeart randomized trial. *Jama*. 2005 Nov 16; 294(19): 2455-64.

[31] Cullen P. Evidence that triglycerides are an independent coronary heart disease risk factor. *The American journal of cardiology*. 2000 Nov 1; 86(9): 943-9.

[32] Hu FB, Van Dam RM, Liu S. Diet and risk of type II diabetes: the role of types of fat and carbohydrate. *Diabetologia*. 2001 Jul 1; 44(7): 805-17.

[33] Vessby B, Uusitupa M, Hermansen K, Riccardi G, Rivellese AA, Tapsell LC, Nälsén C, Berglund L, Louheranta A, Rasmussen BM, Calvert GD. Substituting dietary saturated for monounsaturated fat impairs insulin sensitivity in healthy men and women: The KANWU Study. *Diabetologia*. 2001 Mar 1; 44(3): 312-9.

[34] Cordain L, Eaton SB, Sebastian A, Mann N, Lindeberg S, Watkins BA, O'Keefe JH, Brand-Miller J. Origins and evolution of the Western diet: health implications for the 21st century. *The American journal of clinical nutrition*. 2005 Feb 1; 81(2): 341-54.