Antidiabetic and Hypocholesrolemic effect of Different Types of Vinegar in Rats

Sahar S.A. Soltan[1] and Manal M. E. M. Shehata[2]

[1] Department of Home Economics (Nutrition and Food Science), Faculty of Specific Education, Fayoum University, Fayoum, Egypt
[2]Department of Food Science, Faculty of Agriculture, Zagazig University, Zagazig, Egypt
ssamsoltan@hotmail.com

Abstract: Vinegar is a traditional remedy for aliments including diabetes. This study was conducted to investigate the effects of different types of vinegar (sugarcane, apple, grape, coconut, artificial and palm vinegar) on serum Biochemical and Histopathological of pancreas and stomach of diabetic rats for 6 weeks at 15% concentration. The results indicated that, all of vinegar caused significant decrease $P< 0.05$ in glucose, TC , LDL-c and significant increase in HDL cholesterol. Apple vinegar was the most effective to decrease glucose, TC and LDL-c followed by grape, sugarcane, coconut, artificial and palm vinegar. Apple vinegar contained the higher concentration of organic acid and phenolic compound compared to other vinegar. Apple vinegar and grape vinegar were the most effective to decrease liver and kidney function. Administrating 15% vinegar with diet for 6 weeks decrease the food intake and feed efficiency ratio compared to control group. Moreover, administration different types of vinegar showed that no histopathological change in stomach and has protected effect of pancreas from undesirable change in B cells. In conclusion, using the different types of vinegar with diet for 6 weeks have beneficial effects on diabetic rats and have hypocholesterolemic effect. The vinegar did not effect on stomach histopathological structure and have protective effect of pancreas from damage.

[Sahar S.A. Soltan and Manal M. E. M. Shehata. **Antidiabetic and Hypocholesrolemic effect of Different Types of Vinegar in Rats**. *Life Sci J* 2012;9(4):2141-2151] (ISSN: 1097-8135). http://www.lifesciencesite.com. 319

Keywords: Diabetes mellitus, Apple vinegar, Grape vinegar, Coconut vinegar, Organic acid in vinegar, Histopathology of pancreas and stomach.

1. Introduction

Diabetes mellitus has been defined as a chronic disease with persistently elevated blood glucose concentration (*Greenbaun and Harrison, 2008*). It is a major and growing public health problem throughout the world. Diabetes is the most common endocrine disorder and by the year 2010, it is estimated that more than 200 million people worldwide will have diabetes mellitus and 300 million will subsequently have the disease by 2025 (*Wild et al., 2004 and Hamden et al., 2011*. Diabetes is the sixth most important cause of disability burden in Egypt (NICHP, 2004). Over the last century changes in human behavior and lifestyle have resulted in a dramatic increase in the incidence of diabetes in the world (*Kaushik et al., 2010*). The burden of the disease is increasing both for the progressive aging of population and for the worsening of lifestyle (*Zimmet, 2000*). Dietary and lifestyle factors play an important role not only in the etiology but also in the management of diabetic patients. In addition to the drug treatment, simple and inexpensive diet strategies should aid in achieving and maintaining optimal control of diabetes and diabetic complication (*Xuemei et al., 2012*).

Vinegar is a liquid product from fermentation of carbohydrate. It has been made and used dating from around 300 BC and is an important element in Asian, European, Western and other traditional cuisines of the world. Vinegar has been used for various foods for preservation and often used for flavoring food and pickling. Moreover, diluted unpolished rice vinegar has been drunk as a health food in Japan and its antioxidant activity has been reported (*Nishidai et al., 2000; Shimoji et al., 2002*).

Many medicinal components that are good for health have been reported in natural vinegar, such as carbohydrates (*Johnston et al., 2004 and Leeman et al., 2005*), organic acid (acetic, formic, lactic, malic, citric, succinic and tartaric), alcohols and amino acids and peptides (*Cocchia et al., 2006; Fushimi et al., 2006*), vitamins, mineral salts, amino acids, polyphenolic compounds (e.g., Gallic acid, catechin, caffeic, ferulic acid)(*Morales et al., 2002; Natera et al., 2003*). Traditional vinegar is produced from regional foods according to well- established customs. The balsamic vinegar of Modena, Italy is made from the local white Trebbiano grapes. Traditional rice wine vinegar is produced in Asia, coconut and cane vinegar is common in India and Philippines and date vinegars are popular in the Middle East. Some scientific investigation clearly benefits of vinegar such as: antimicrobial properties (*Vijayakumar and Wolf-Hall.,2002; Sengun and Karapinar.,2005),*prevent inflammation and hypertension (*Murooka and Yamshita, 2008*), lower serum cholesterol (*Fushimi et al*., *2006*), treatment of ear infection (otitis external,

otitis media) (*Aminifarshidmehr, 1996; Jung et al., 2002*), treating mal fungus and warts (*Takano- Lee et al., 2004*), reduction in systolic blood pressure (*Kondo et al., 2000*), enhanced calcium absorption and retention (*Kishi et al., 1999*), decrease the glycemic index of carbohydrate food for people with and without diabetes (*Sugiyama et al., 2003; Johnston et al., 2004).* Antiglycemic effects of vinegar have been known for more than a century and have been demonstrated in animal as well as human studies (*Salbe et al., 2009*). So that the objective of this work was to investigate the antidiabetic effects and hypocholestrolemic effect of different types of vinegar (sugarcane, apple, grape, coconut, artificial and palm vinegar) in rats.

2. Materials and Methods

Materials

Fructose sugar was purchased from Sigma-Aldrich, St., and Louis, Mo, USA. Natural sugar vinegar (Sugarcane vinegar, 6% acetic acid), Natural apple vinegar (6 % acetic acid), Natural grape vinegar (6%), Coconut vinegar (6% acetic acid), artificial vinegar (6% acetic acid) and Palm vinegar (6% acetic acid) were purchased from local market Cairo, Egypt. Kits for blood analysis were purchased from Biodiagnostic 29 Tahreer St., Dokki, Giza, Egypt.

Methods

HPLC analysis of organic acids in different types of vinegar

Organic acids of different types of vinegar were determined by a HPLC according to the method by *Zbigniew et al., 1991*. 1ml of each sample was diluted by 10 ml water and take 35 µl for injection into HPLC Hewllet Packard (series 1050) equipped with auto sampling injector, solvent degasser, ultraviolet (UV) detector set at 210 nm and quaternary HP pump (series 1100). Packed column Hypesil BDS- C18, 4.0 x 250 mm was used to separate organic acid. The column temperature was maintained at 55°C, at flow rate 1ml/min. Organic acid standard from Sigma Co. were dissolved in a mobile phase (phosphoric acid) and injected into HPLC. Retention time and peak area were used to calculation of organic acids concentration by data analysis of Hewllet Packard software.

HPLC analysis of phenolic compound in different types of vinegar

Phenolic compound in different types of vinegar were determined by HPLC according to the method of *Coupy et al., 1999.* 1ml of sample was diluted by 10 ml water and take 100µl for injection into HPLC Hewllet Packard (series 1050) equipped with auto sampling injector, solvent degasser, ultraviolet detector set at 280 nm and quaternary HP pump series 1100) . Packed column Hypesil BDS- C18, 4.0 x 250 mm was used to separation phenolic compound. The column temperature was maintained at 35 °C. Gradient separation was carried out with methanol and acetonitrile as a mobile phase at flow rate of 1 ml/min. phenolic acid standard from sigma Co. were dissolved in mobile phase and injected into HPLC. Retention time and peak area were used to calculation of phenolic compounds concentration by data analysis of Hewllet Packard software, Germany.

Animals and treatment

Normal forty eight male albino rats weighing 80-100 grams were used for the study. They obtained from animal house of El-Salam Farm, Giza, Egypt. The animal housed individually in stainless steal cages under controlled condition at constant temperature (22 °C) and lighting (12 h. light- dark cycle) and given free access to food and water at all times. The rats were divided randomly into eight groups, six rats each and were fed on the following diets for six weeks:

Group1: Rats were fed on standard diet as served as normal control (negative control group). Standard diet was prepared according to *Reeves et al., 1993*. It contained 14% casein, 5% cellulose, 3.5% mineral mixture, 1% vitamin mixture, 0.25% choline, 0.3% Dl-methionine, 5% oil and 65% starch.

Group2: Rats were fed fructose rich diet (66% fructose) as diabetic group (positive control group). Fructose diet was prepared according to *Yador et al., 2004; Veerapur et al., 2010.*

Group 3: Rats were fed on fructose rich diet +15% natural sugarcane vinegar

Group 4: Rats were fed on fructose rich diet + 15% natural apple vinegar

Group 5: Rats were fed on fructose rich diet + 15% natural grape vinegar

Group 6: Rats were fed on fructose rich diet + 15% natural coconut vinegar

Group 7: Rats were fed on fructose rich diet + 15% artificial vinegar

Group 8: Rats were fed on fructose rich diet + 15% Palm vinegar

Each rat has been weighted at the beginning and the end of experimental and food intake was daily recorded. At the end of experimental period (six weeks), rats were sacrificed after overnight fasting. The blood of each rat was collected in two tubes. The first tub was containing sodium fluoride to preserve glucose (to determination of glucose). The blood in the second tube was centrifuged at 3000 rpm for 20 minutes to obtain the serum, which is kept at -20 °C until analysis.

Chemical analysis

A- Serum glucose was determined according to *Trinder, 1969*

B- Lipid Profiles

Serum total cholesterol (TC), LDL-c, HDL-c and Triacylglycerol (TG) were measured by enzymatic method using commercial kits according to *Richmond, 1973*, *Burstein et al., 1970*, *Wieland and Seidel, 1983*, *Jacobs and Vandermark, 1960.*

C -Kidney function

Serum creatinine and serum Urea were determined according to Larson*, 1972; Patton and Crouch,* 1977

D- Liver enzyme

ALT and AST were determined by the method of *Reitman and Frankal, 1957***.**

E- Antioxidant enzyme in liver

Glutathione in liver was determined according to the method by *Beulter et al., 1963***.**

F- Hemoglobin concentration was performed using a UDI- HMI automatic hematology analyzer (France).

Histopathological assessment

At necropsy, stomach and pancreas were fixed in 10% buffered formalin until analysis. Tissue of stomach and pancreas were routinely processed for paraffin embedding and sections were prepared and stained with hematoxylin and eosin (using light microscopy). Histopathological assessment was performed on all tissues of control group and treatment

Statistical analysis

Analysis of the data was of preventative variable in the form mean ±SD by SPSS version 17.0 according to *Snedecoer and Cochran, 1967*.

3. Results and Discussion

Data in Table (1) revealed that all of samples vinegar was contained acetic acid and oxalic acid. Acetic acid and succinic acid was major organic acid in sugarcane, apple and grape vinegar. Oxalic, citric, formic, ascorbic, acetic, succinic and malic acid could be found in apple vinegar. These results are agreement with *Shahidi et al., 2008* indicated that acetic, citric, malic, lactic, succinic, tartaric and fumaric acid could be found in fruit vinegar including apple and grape. *Giumanin et al., 2001* found that apple vinegar contained succinic, malic, glutaric, lactic, citric and tartaric acid. Nevertheless, lactic, glutaric and tartaric acids could not be detected in our apple vinegar. Organic acid in fruit vinegar might source from original material and be generated during fermentation process (*Shahidi et al., 2008*). Artificial vinegar and palm vinegar contained acetic acid and oxalic acid only. Other organic acid could not be detected. Meanwhile, oxalic, formic, ascorbic and acetic acids could be found in coconut vinegar. Ascorbic acid could be found only in sugarcane, apple, grape and coconut.

Data in table (2) illustrated that the higher concentration of catechin was detectable in apple vinegar (13.24mg/100ml) followed by grape vinegar (9.21mg/100ml), coconut vinegar and sugarcane vinegar (0.43 and 0.21mg/100ml). Meanwhile, it is not detectable in artificial and palm vinegar. Pyrogallol was only identified and major compound in apple vinegar (37.05mg/100ml). Higher concentration of Salycillic was observed in artificial vinegar (13.25mg/100ml) followed by palm vinegar (8.50mg/100ml), grape vinegar (3.13mg/100ml), apple vinegar (1.52mg/100ml), and coconut vinegar (0.21mg/100ml). Different in phenolic compounds may be due to different source of fruit used to produce vinegar. Phenolic compounds have been shown to good markers of the quality and origin of vinegar (Galvez et al., 1995).

The serum blood glucose concentration elevated from 134.0±4.94 mg/dl of control group to 187.53±4.75 mg/dl of diabetic group rats (Fig 1). Vinegar reduced glucose concentration, rate of decrease was 28.59%, 30.48%, 29.15%, 28.45%, 25.38% and 26.46% of sugarcane, apple, grape, coconut, artificial and palm vinegars respectively. All of types vinegar showed that significant decrease of glucose compared to the diabetic group. Apple, grape and sugarcane vinegar were the most effective decrease of glucose. This could be due to possibility the active ingredient in vinegar (acetic acid and organic acid) to enhanced secretion of insulin from beta cell. The higher effective of apple, grape and sugarcane vinegar may be they contained more organic acid than other types of vinegar. It is not known how vinegar alters blood glucose concentration, but several mechanisms have been proposed. Acetic acid in vinegar may interfere with digestion of starch molecules there by reducing the amount of glucose absorbed into the blood stream after meal (*Ogawa et al., 2000*). Other suggest that vinegar slows the rate of gastric emptying and thus delays carbohydrate absorption and improves satiety (*O'Keefe et al., 2008*), and or acetic acid enhances uptake of glucose from the blood stream into tissues thereby keeping blood glucose concentration (*Fushimi et al., 2001*).Other investigation for human found that 10 grams with a meal was the most effective dose to lower blood glucose levels (*Johnston et al., 2010*). Also the consumption of apple cider vinegar slowed the rise of blood sugar after the high carbohydrate vinegar breakfast (*Johnston et al., 2004***)**.

Data in Table (3) revealed that there are no significant differences in initial body weight $P< 0.05$ of eight groups. While different types of vinegar administrated for 6 weeks demonstrated decreased in

body weight gain compared to the control group. Since the decrease in body weight gain was non significant between diabetic group and treatment vinegar groups. Acetic acid was considered to be the active ingredient in vinegar that effected reduction body fat and body weight gain (*Kondo et al., 2009*). These results are in agreement with (*Moon et al., 2010*) who reported that there were no significant difference in weight gain among mice groups intake different diet with persimmon – vinegar. Another investigator examined the effect daily vinegar ingestion on body weight of human, he found that the health adults ingested 2 tables spoons of apple cider vinegar (1 g acetic acid) twice daily for 4 week (*Johnston, 2006*) lead to body loss an average of 1.6 pound where the control subject gained 0.6 pound. Data in the same Table illustrated that there are significant decrease $P<0.05$ in food intake and feed efficiency ratio of all groups were administrated of vinegar compared to the control group. Meanwhile, no significant difference was observed between diabetic group and other groups were intake different types of vinegar. Decrease of food intake may be due to decrease appetite of food because a strong acidic taste and pungent smell of vinegar .These results are in line with the results by *Moon et al., 2010* reported that there were no significant different in feed consumption among all the vinegar administrated groups.

Table (4) illustrated that the effect of different types of vinegar on weight of organs. There are no significant change in weight of organs for all rats was administrated different types of vinegar.

Serum lipid profiles are shown in Table (5). Serum TC and LDL-c concentration significantly decreased $P< 0.05$ in all types of vinegar administrated groups. Apple, grape, sugarcane and coconut vinegar revealed reduction of TC and LDL-c more than artificial and palm vinegar. These results may be due to the apple, grape, sugarcane and coconut were contained ascorbic acid (20.05%, 10.23, 2.33 and 0.34 mg/100ml) respectively behind acetic acid. *McRac, 2008* reported that the supplementation with ascorbic acid lower serum low density lipoprotein and total cholesterol. These data may be due to the acetic acid (active component in vinegar) reduced serum cholesterol via the inhibition of hepatic lipogenesis and the promotion of fecal bile acid excretion. Acetic acid is converted to acetate in vitro, and acetate metabolism by tissues activates AMPK which play a key role in lipid homeostasis which may explain the lipid lowering effects of ingested acetic acid in animals (*Yamashita et al., 2007*). While HDL-c concentration showed significantly increase compared to the diabetic group, but there was no significant difference seen among the vinegar administrated groups. Improved in lipid profiles by vinegar were also observed in another study with rats. *Fushimi et al., 2006* reported that serum TC decreased when 0.3% (w/w) acetic acid was administrated for 19 days routine diet containing 1% cholesterol. *Moon et al., 2010* have reported similar finding that a persimmon- vinegar decease serum TC concentration in mice. *Shishehbor et al., 2008* reported that apple cider vinegar improved the serum lipid profile in normal and diabetic rats by decreasing serum LDL, TG and increasing serum HDL. TG in the same Table revealed that increase in diabetic group 60.10±5.91 compared to the control group 40.37±7.45, but there was no significant difference seen among the vinegar administrated groups. These results not on line with *Fushimi et al., 2006* who reported that the vinegar decrease TG.

Effect of administration of vinegar on liver function and kidney function are shown in Table (6). ALT and AST increased in diabetic group (48.30±2.67 and 49.07±1.43) compared to the control group (30.60±2.37 and 30.57±6.6) respectively. Administration of vinegar was decrease liver function when compared to the diabetic group. The best results of ALT and AST was observed in apple vinegar and grape vinegar (rate of decrease was (52.23%, 26.77% and 50.94% and 49.86%). Also, creatinine and urea increased in diabetic group (3.97±1.0 and 23.73±2.16) when compared to the control group (1.67±0.44 and 19.87±1.42) respectively. Vinegar administrations lead to significant decrease in creatinine and urea $P< 0.05$ of all treatment when compared to the diabetic group. The apple vinegar was the most efficiency in kidney function. This results may be due to apple vinegar have high levels from phenolic compounds specifically catechin and pyrogallol, which prevent kidney from destroyed induced by diabetic disease. *Pitchai and Manikkam, 2011* reported that the administration catechin lowered urea and creatinine in diabetic rat. Our results disagreement with *Kondo et al., 2009* who reported that there are no significant change in measurements of liver function (AST and ALT) or kidney function of two doses of apple vinegar. Data in the same Table showed that there are no significant differences between control, diabetic, sugarcane vinegar and apple vinegar groups in hemoglobin concentration. Meanwhile, grape vinegar group, coconut vinegar group, artificial vinegar group and palm vinegar group showed that significant decrease $P< 0.05$ in hemoglobin concentration compared to the diabetic group. Data in (Fig 2) showed that significant decrease of glutathione in diabetic group, coconut vinegar group, palm vinegar group (3.19±0.52 mM/L) ^{bc} , (3.0±0.72)^{c} and (3.20±0.10)^{bc} compared to the control group (4.95±0.65)^{a}. Decrease of glutathione in diabetic group may be due to increase in lipid oxidation in fructose induced diabetic rats (*Suwamaphat et al., 2010*). Decrease in glutathione of

coconut vinegar group and palm vinegar group may be due to increase in catabolism of fructose caused the reduction of total glutathione levels (*Oda et al., 1994 and Reddy et al., 2009*). Meanwhile, there are no significant difference between other vinegar groups and control group.

Histopathological Assessment

Pancreas

Microscopically, pancreas of rat from group 1 (control group) revealed no histopathological changes (Fig 3). Meanwhile, pancreas of rats from group 2 (diabetic group) showed atrophy of islets of langerhan's and hyperplasia of ß cells of islets of langerhan's (Fig 4). This result agreement with *Riccillo et al., 2012* and *Verma et al., 2012* reported that the type-2 diabetic induce markedly abnormal change in rat islets. Also, *Balamurugan and Ignacimuthu`, 2011* found that small atrophies islets cells in diabetic control, whereas rats from groups 3 to 7 showed no histopathological changes (Figs 5 - 9). Moreover some section from group 8 rats was fed on palm vinegar revealed slight hyperplasia of ß cells of islets of langerhan's (Fig 10). These results are on line with *Xuemei, et al., 2012* reported that vinegar improved pancreatic ß cell deficit in STZ- induced diabetic in rats.

Stomach

Microscopically, stomach of rats from group 1 (control group) and diabetic group (group2) revealed no histopathological changes (Figs 11 and 12). Meanwhile, stomach of the rats from group 3 fed on 15% sugarcane vinegar showed few sub mucosal inflammatory cells infiltration (Fig 13). However, stomach of rats from group 4, 5, 6, 7 and 8 revealed no histopathological changes (Figs 14 - 18). These data suggest the vinegar intake at 15% concentration did not effect on stomach tissues.

Table (1): HPLC analysis of organic acids in different types of vinegar

Organic acids	Types of vinegar					
	Sugarcane mg/100ml	Apple mg/100ml	Grape mg/100ml	Coconut mg/100ml	Artificial g/100ml	Palm mg/100ml
Oxalic	47.65	12.47	23.82	2.40	1.13	5.41
Citric	54.97	95.70	19.70	-	-	-
Formic	-	96.85	-	10.80	-	-
Acetic	6380.32	6499.33	7336.27	8816.95	7210.37	7807.99
Ascorbic	20.05	10.23	2.33	0.34	-	-
Succinic	133.94	202.77	133.53	-	-	-
Malic	-	5.58	2.35	-	-	-

Table (2): HPLC analysis of phenolic compounds in different types of vinegar

Phenolic compounds	Types of vinegar					
	Sugarcane mg/100ml	Apple mg/100ml	Grape mg/100ml	Coconut mg/100ml	Artificial mg/100ml	Palm mg/100ml
Gallic	0.03	-	-	0.03	-	0.02
Catechin	0.21	13.24	9.21	0.43	-	-
Ferulic	0.01	-	-	0.01	0.03	0.02
Benzoic	0.36	-	-	0.36	-	-
Pyrogallol	-	37.05	-	-	-	-
Protocatechuic	-	1.48	-	-	-	-
Catechol	-	1.08	-	-	-	-
Vanillic	-	0.52	0.73	-	-	-
P-Coumaric	-	0.24	-	-	-	-
Salycilic	-	1.52	3.13	0.21	13.25	8.50
Chlorogenic	-	-	2.16	-	-	-
Caffeic	-	-	0.70	0.01	-	0.01
Caffien	-	-	0.50	-	-	-
Coumarin	-	-	-	-	0.46	0.29

Table (3): Effect of different types of vinegar on body weight, food intake and feed efficiency

Groups	IBW* (gm) Mean ±SD	FBW° (gm) Mean ±SD	BWG‡ (gm) Mean ±SD	Food intake daily Means ±SD	Feed efficiency ratio (FER) Means ±SD
1- Control	84.15±3.11[a]	141.65±16.3[a]	57.5±17.4[a]	8.47±0.67[a]	6.79±1.09[a]
2-Datbetic	85.17±1.91[a]	99.13±4.7[b]	13.97±5.12[b]	5.56±1.3[bc]	2.64±2.09[b]
3-Sugarcane vinegar	83.53 ±1.61[a]	102.67±7.05[b]	19.13±6.50[b]	4.73±0.66[c]	4.13±1.55[b]
4- Apple vinegar	83.0±3.12[a]	104.03±11.30[b]	21.03±11.37[b]	5.98±0.61[b]	3.62±2.06[b]
5- Grape vinegar	86.07±3.6[a]	104.22±5.97[b]	18.20±6.67[b]	5.27±0.32[bc]	3.89±0.97[b]
6- coconut vinegar	85.86±4.03[a]	104.57±9.03[b]	18.70±9.90[b]	5.82±0.21[bc]	3.24±1.81[b]
7- Artificial vinegar	84.63±1.73[a]	101.10±16.47[b]	18.77±13.57[b]	5.95±1.2[b]	3.23±2.20[b]
8- Palm vinegar	84.0±2.12[a]	97.97±3.19[b]	13.97±5.12[b]	6.23±1.2[b]	2.25±0.81[b]

Initial body weight* Final body weight°, Body weight gain‡

Table (4): Effect of different types of vinegar on weight of organs

Group	Weight of Heart g/kg	Weight of Liver g/kg	Weight of Kidney g/kg	Weight of Spleen g/kg
1- Control	0.366 ± 5.16[a]	4.80± 0.32[a]	0.767±5.16[a.]	0.267±0.02[a]
2-Diabetic	0.366 ±5.16[a]	4.53 ±0.65[a]	0.733±5.16[a]	0.233±0.02[a]
3- Sugarcane vinegar	0.400 ± 0.0[a]	4.30±0.98[a]	0.733±1.37[a]	0.267±0.02[a]
4- Apple vinegar	0.367± 0.15[a]	5.10± 0.62[a]	0.767±5.16[a]	0.233±0.02[a]
5- Grape vinegar	0.367±0.10 [a]	4.30±1.03[a]	0.700±0.0[a]	0.267±0.02[a]
6- Coconut vinegar	0.400±8.9[a]	4.53±0.41[a]	0.800±8.9[a]	0.267±0.02[a]
7- Artificial vinegar	0.400 ±8.9[a]	4.833±0.45[a]	0.762±4.60[a]	0.233±0.02[a]
8- Palm vinegar	0.400 ±0.0[a]	4.47±0.22[a]	0.767±5.16[a]	0.300±0.02[a]

Table (5): Effect of different types of vinegar on lipid profile

Group	Total Cholesterol mg/dl	Total Triacylglycerol mg/dl	HDL Cholesterol mg/dl	LDL Cholesterol mg/dl
1- Control	200.27 ± 0.95[e]	40.37± 7.45[b]	44.83±6.91[a.]	147.36±8.23[d]
2-Diabetic	260.93 ±1.37[a]	60.10 ±5.91[a]	35.0±3.34[b]	213.91±2.04[a]
3- Sugarcane vinegar	233.57 ± 12.5[cd]	55.07±11.09[a]	44.83±6.20[a]	177.72±14.8[bc]
4- Apple vinegar	220.4 ± 23.5[d]	52.73± 3.13[a]	46.67±10.6[a]	163.19±12.7[c]
5- Grape vinegar	225.23±9.48[d]	53.83±9.7[a]	46.37±4.60[a]	167.86±20.9[c]
6- Coconut vinegar	242.87±10.8[bc]	58.97±5.38[a]	44.03±4.19[a]	189.82±12.6[b]
7- Artificial vinegar	244.47±9.86[bc]	55.03±9.4[a]	40.53±4.2[ab]	191.57±12.9[b]
8- Palm vinegar	253.83±9.48[ab]	53.07±6.59[a]	34.80±5.41[a]	207.22±6.11[a]

Table (6): Effect of different types of vinegar on liver and kidney functions and Hemoglobin

Group	ALT (µl/dl) Mean± SD	AST µl/dl) Mean± SD	Creatinine mg/dl) Mean± SD	Urea mg/dl) Mean± SD	Hemoglobin mg/dl Mean± SD
1- Control	30.60±2.37[c]	30.57±6.6[c]	1.67±0.44[e]	19.87±1.42[bcd]	13.03±0.37[abc]
2-Diabetic	48.30±2.67[a]	49.07±1.43[a]	3.97±0.10[a]	23.73±2.16 [a]	13.57±0.68[a]
3- Sugarcane vinegar	37.20±4.43[b]	33.53±4.72[bc]	2.65±0.41[cd]	19.57±0.77[cd]	13.70±1.08[a]
4- Apple vinegar	23.07±2.04[c]	24.07±4.83[d]	2.90±0.02[bc]	17.93±1.65[e]	13.40±0.70[ab]
5- Grape vinegar	35.37±5.48[bc]	24.60±6.60[d]	2.97±0.10[bc]	18.97±1.25[de]	11.37±0.67[c]
6- Coconut vinegar	37.40±3.03[b]	33.43±3.58[bc]	3.83±0.49[a]	20.73±0.52[bc]	12.13±1.15[bcd]
7- Artificial vinegar	39.50±6.35[b]	38.87±5.23[b]	3.17±0.42[b]	20.30±0.72[bcd]	11.90±1.09 [cd]
8- Palm vinegar	38.13±3.10[b]	23.17±5.09[d]	2.30±0.47[d]	21.47± 1.28[b]	12.47±1.76[abcd]

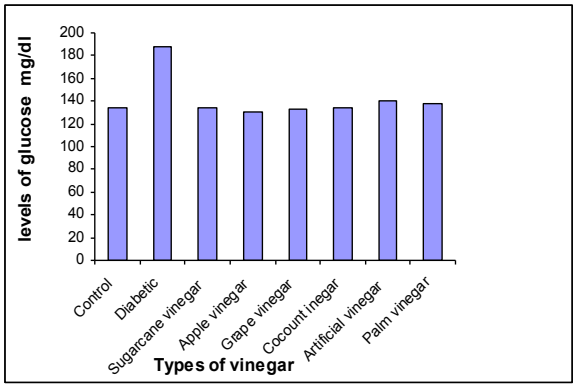

Fig (1): Effect of different types of vinegar on glucose level

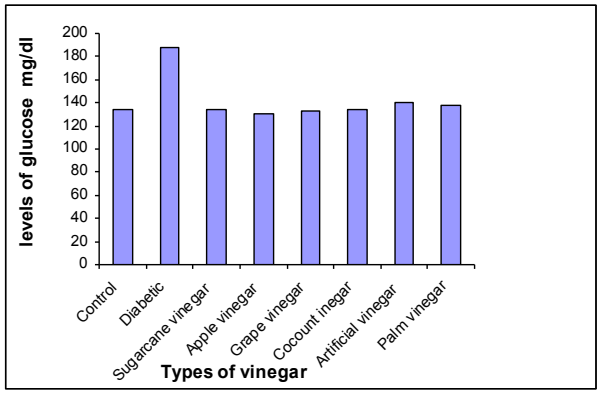

Fig (2): Effect different types of vinegar on glutathione levels

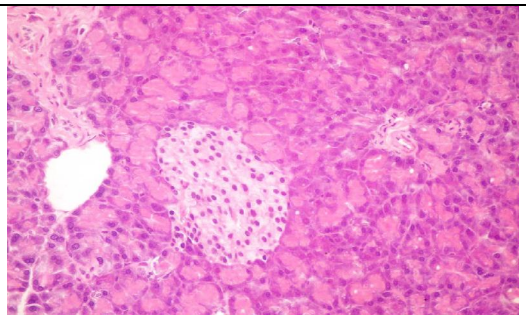

Fig (3): pancreas of control group (1) showing no histopathological Change (X-400)

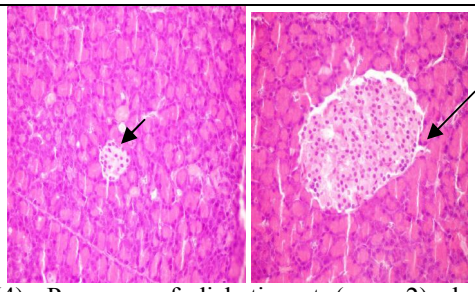

Fig (4): Pancreas of diabetic rat (group2) showing atrophy of Islets and hyperplasia of B cells of langerhans (X-400)

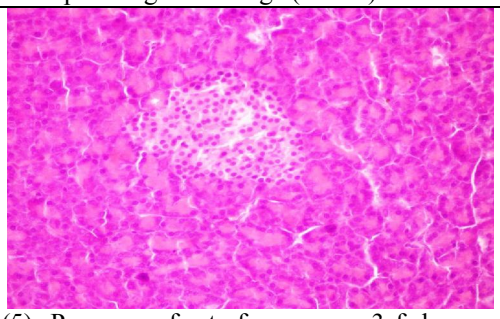

Fig (5): Pancreas of rats from group 3 fed sugarcane vinegar showing no Histopathological changes (X-400)

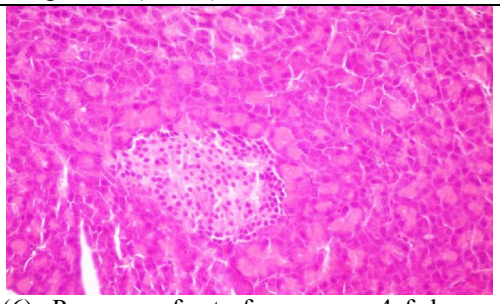

Fig (6): Pancreas of rats from group 4 fed on apple vinegar showing no Histopathological change (X-400)

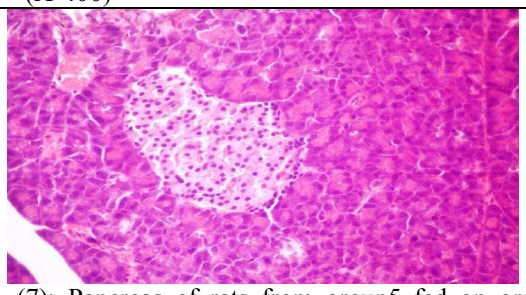

Fig (7): Pancreas of rats from group5 fed on grape vinegar showing no Histopathological changes (X-400)

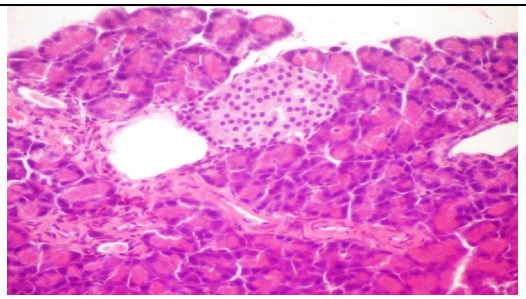

Fig (8): Pancreas of rats from group 6 fed on coconut vinegar showing no Histopathological change (X-400)

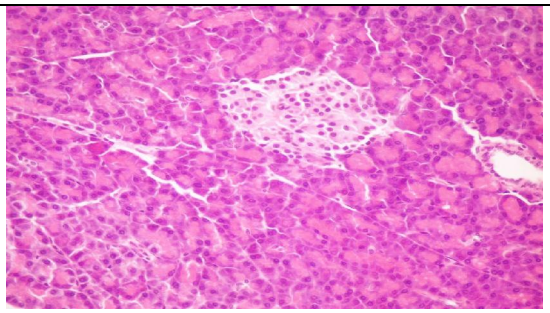

Fig (9): Pancreas of rats from group 7 fed On artificial vinegar showing no Histopatological changes (X-400)

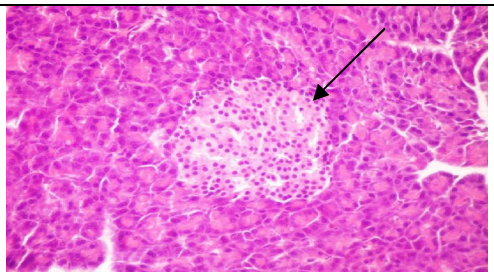

Fig (10): Pancreas pf rats from group 8 fed on palm vinegar showing slight Hyperplasia of B cells of islets of langerhan's (X-400)

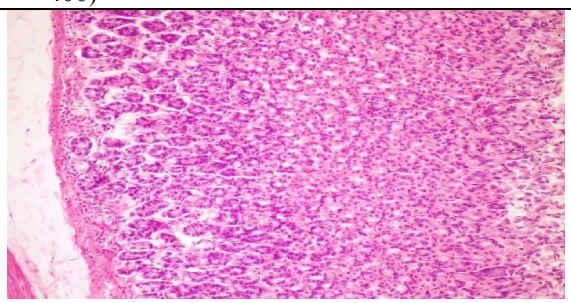

Fig (11): Stomach of control group (1) showing no histopathological Change (X-400)

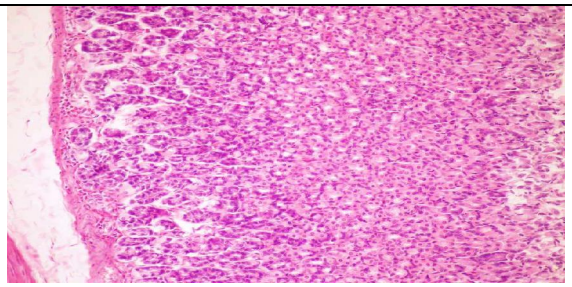

Fig (12) Stomach of diabetic rat (group2) showing no histopathological Change (X-400)

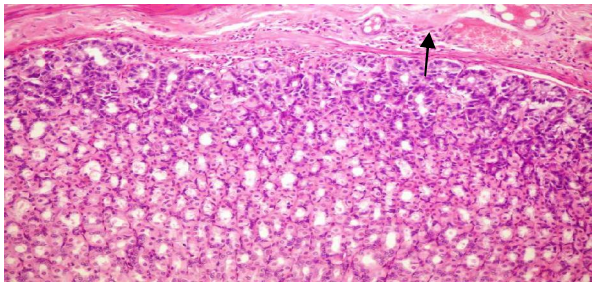

Fig (13): Stomach of rats from group 3 fed on sugarcane vinegar showing few sub mucosal cells infiltration (X-400)

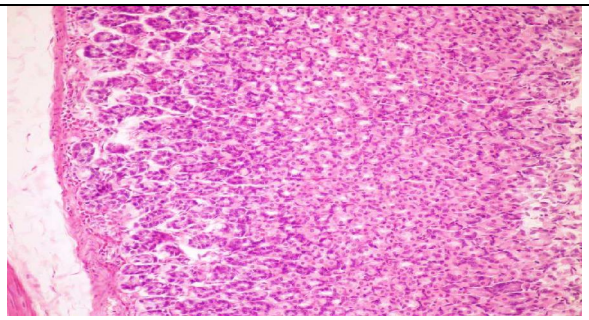

Fig (14): Stomach of rats from group 4 fed on apple vinegar showing no Histopathological changes (X-400

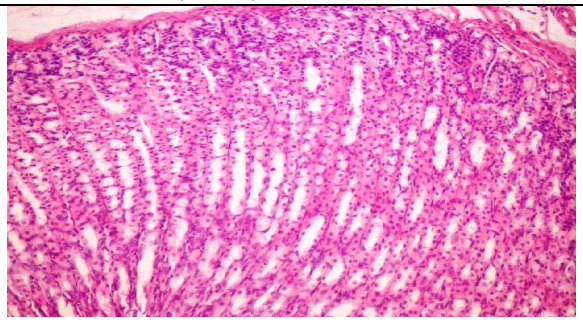

Fig (15): Stomach of rats from group5 fed on grape vinegar showing no histopathological changes (X-400)

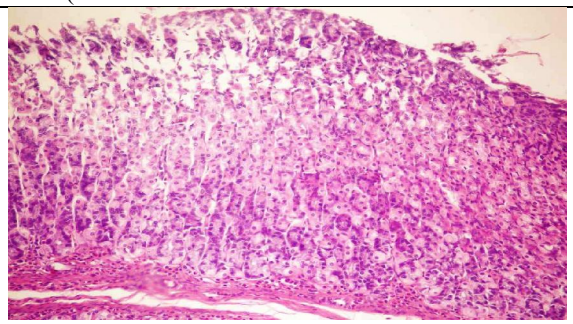

Fig (16): Stomach of rats from group 6 fed on coconut vinegar showing no histopathological change (X-400)

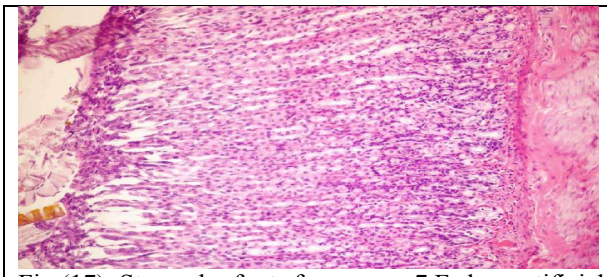

Fig (17): Stomach of rats from group7 Fed on artificial vinegar showing no hiatopathological change (X-400)

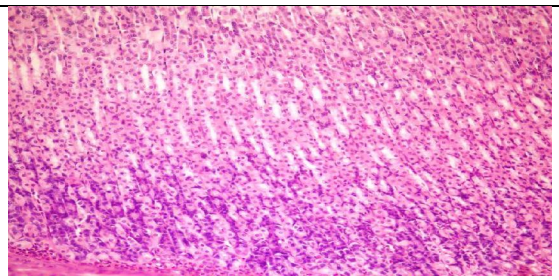

Fig (18): Stomach of rats from group 8 fed on palm vinegar showing no histopathological changes (X-400)

Conclusion

Vinegar has potential benefits of diabetic rats thought decrease glucose concentration and cholesterol. Apple vinegar and grape vinegar were the more effective to decrease total cholesterol and LDL-c than the other types of vinegar. Moreover, they were caused increase of HDL-c more than the other types of vinegar. Apple vinegar and grape vinegar decreased AST, ALT, urea and creatinine more than the sugarcane, coconut, artificial and palm vinegar. They contained more organic acid and phenolic compound than the other vinegar. Apple vinegar contained the highest concentration of catechin. Pyrogallol, protocatechuic, catechol, p-coumaric was only detectable in apple vinegar. Apple and sugarcane vinegar has no effect on hemoglobin concentration. Glutathione was decrease in diabetic group, coconut vinegar group and palm vinegar group. Apple vinegar followed grape, sugarcane, coconut, artificial and apple vinegar consider antidiabetic and hypocholestrolemic effect in diabetic rat. Different type of vinegar has protective effect of pancreas and did not effect on stomach with 15% concentration for 6 weeks. So that using vinegar has a beneficial effect of diabetic disease in rats.

Acknowledgement

We wish to acknowledge Dr. Kawkab Abd-Elaziz Ahmed professor of Histology in Faculty of Veterinary Medicine, Cairo University for her expertise and assistance in clarifying and explaining the changes in tissue slices of organs.

Corresponding author
Sahar S.A. Soltan
Department of Home Economics (Nutrition and Food Science), Faculty of Specific Education, Fayoum University, Fayoum, Egypt
ssamsoltan@hotmail.com

References

1. Aminifarshidmehr, N. (1996): The management of chronic supportive otitis media with acid media solution. The American Journal of Otology. 17: 24-25.
2. Balamurugan, R. and Ignacimuthu, S. (2011): Antidiabetic and hypolipidemic effect of methanol extract of Lippia no diflora L. in streptozotocin induced diabetic rats. Asian Pacific Journal of Tropical Biomedicine. 1: S30-S35.
3. Beulter, E., Duran, O. and Kelly, M. B. (1963): Determination of reduced glutathione. Journal of Laboratory and Clinical Medicine. 61: 882- 891.
4. Burstein, M., Scholinck, H. R. and Haarfin, R. (1970): Rapid method for the isolation of lipoprotein from human serum by precipitation with polyanion. Journal of Lipid Research.11: 583- 595.
5. Cocchia, M., Durantea, C., Grandia, M., Lambertinic, P., Manzinib, D., Manzinib, D. and Marchetti. A. (2006): Simultaneous determination of sugars and organic acids in aged vinegar and chemometric data analysis. Atlanta. 69: 1166- 1175.
6. Coupy, P., Hugues, M., Biovin, P. and Amiot, M.J. (1999): Antioxidant composition and activity of barley (Hordeum Vulgar) and malt extracts and of isolated phenolic compounds. Journal of the Science Food and Agriculture. 79: 1625- 1634
7. Fushimi, T., Suruga, K., Oshima, Y., Fukiharu, M., Tsukamoto, Y. and Goda, T. (2006): Dietary acetic reduced serum cholesterol and triacylglycerols in rats feed a cholesterol rich diet. British Journal of Nutrition. 95 (5): 916- 924.
8. Fushimi, T., Tayama, K., Fukaya, M., Kitakoshi, K., Nakai, N., Tsukamoto, Y. and Sato, Y. (2001): Acetic acid feeding enhances glycogen repletion in liver and skeletal muscle of rats. Journal of Nutrition. 131: 1973- 1979.
9. Galvez, M. C., Barroso, C. G. And Perez-Bustamante, J. A. (1995): Influence of the origin of Win vinegar in their low molecular weight phenolic content. Acta Horticulture. 338: 269- 272.
10. Giumanin, A.G., Verardo, G., Martine, D. D. And Toniutti, N. (2001): Improved method for the analysis of organic acids and new derivatization of alcohols in complex natural aqueous matrixes: application to wine and apple vinegar. Journal Agriculture of Food and Chemistry. 49: 2875- 2882.
11. Greenbaum, C. J. and Harrison, L. C. (2008): Diabetes: Translating research into practice. London: Informal Healthcare, P 1-2.

12. Hamden, K., Keskes, H., Belhaf, S., Mnafgui, K., Feki, A. and Allouch. N. (2011): Inhibitory potential of omega-3 fatty and fenugreek essential oil on key enzymes of carbohydrate- digestion and hypertension in diabetes rats. Lipids in Health and Disease. 10: 226- 231.
13. Jacobs, N. and Vandermark, P. J. (1960): Determination of serum triacylglycerol. Archives of Biochemistry Biophysics. 88: 250- 261.
14. Johnston, C. S., Kim, C. M. and Buller, A. J. (2004): Vinegar improves insulin sensitivity to a high carbohydrate meal in subjects with insulin resistance or type-2 diabetes mellitus. Diabetes Care. 27: 281- 282.
15. Johnston, C. S. (2006): Strategies for health weight loss: from vitamin C to the glycemic response. Journal of the American College Nutrition. 25: 158- 165.
16. Johnston, C. S., Steplewska, I. , Long, C. A. , Harris, L. N. and Ryals, R. H. (2010): Examination of the antiglycemic properties of vinegar in health adults. Annals of Nutrition & Metabolism, 56: 74- 79.
17. Jung, H. H., Shosd, C. K., Lim, H. H. and Chae, S. W. (2002): Vinegar treatment in the management of granular meningitis. J Laryngol Otol. 116: 176- 180.
18. Kaushik. G., Satya, S., Khandelwal. R. k. and Naik, S. N. (2010): Commonly consumed Indian plant food materials in the management of diabetes mellitus. Diabetes and Metabolic Syndrome: Clinical Research and Reviews. 4: 21- 40.
19. Kishi, M., Fukaya, M., Tsukamoto, Y., Nagasaw, T., Takenhana. K. and Nishizawa, N. (1999): Enhancing effect of dietary vinegar on the intestinal absorption of calcium in ovariectomized rats. . Bioscience, Biotechnology and Biochemistry. 63: 905- 910.
20. Kondo, S., Tayama, K., Tsukamoto, Y., Ikeda, K. and Yamori. Y. (2000): Antihypertensive effects of acetic acid and vinegar on spontaneously hypertensive rats. Bioscience, Biotechnology and Biochemistry. 65: 2690- 2694.
21. Kondo, T., Kishi, M., Fushimi, T, UgaJin and Kaga, T. (2009): Vinegar intake reduces body weight, body fat mass , and serum triglyceride levels in obese Japanese subjects. Bioscience, Biotechnology and Biochemistry 73 (8): 1837- 1843.
22. Larson, K. (1972): Creatinine assay by a reaction – kinetic principle. Clinical Chemical Acta. 41: 209- 217.
23. Leeman, M., Ostman, E. and Bjorck, I. (2005): Vinegar dressing and cold storage of potatoes lower postprandial glycemic and insulinaemic responses in healthy subjects. European Journal of Clinical Nutrition. 59: 1266- 1271.
24. McRac. M. P. (2008): Vitamin C supplementation lowers serum low density lipoprotein cholesterol and triglycerides: a meta- analysis of 13 randomized controlled trials. Journal Chiropractic Medicine. 7 (2): 48- 58.
25. Morales, M. L., Tesfay, W., Garcia-Barilla, M. C., Cases. J. A. and Tronco, A. M. (2002): Evaluation of the aroma profile of sherry wine vinegar during an experimental aging lagging in wood. Journal Agriculture and Food Chemistry. 50: 3173- 3178.
26. Moon, Y. J., Choi, D. S., Oh, S. H., Song, Y. S. and Cha, Y. S. (2010): Effects of persimmon- vinegar on lipid and carnitine profiles in Mice. Food Science and Biotechnology. 19 (2): 343 – 348.
27. Murooka, Y. and Yamshita, M. (2008): Traditional healthful fermented products of Japan. Journal Industrial Microbiology Biotechnology, 35: 791- 798.
28. Natera, R., Castro, R., Garcia- Moreno, M. V., Hernandez, M. J. And Garcia- Barroso, C. (2003): Chemometric studies of vinegars from different raw materials and processes of production. Journal of Agricultural and Food Chemistry. 51: 3345 – 3351.
29. (NICHP) National Center of Health and Population (2004): The Burden of Disease and Injury in Egypt (Mortality and Morbidity).
30. Nishidai. S., Nakamura, Y., Torikai, K., Yamamoto, M., Ishihhara, N. and Mori, H. (2000): Kurusu, a traditional vinegar produced from unpolished rice, suppresses lipid peroxidation vitro and in mouse skin. Bioscience, Biotechnology and Biochemistry. 64: 1909- 1914.
31. Oda, A., Bannai, C., Yamoka, T., Kotari, T., Masushima, T. And Yamashita. K. (1994): Inactivation of Cu, Zn-superoxide dismutase by in vitro glycosylation in erythrocytes of diabetic patients. Horm Metab Res. 26: 1-4.
32. Ogawa, N., Satsu, H., Watanobe, H., Fukay, M., Tsukamoto, Y. Miyamoto, Y. and Shimizu, M. (2000): Acetic acid suppresses the increase in disaccharides activity that occurs during culture of caco-2- cells. Journal Nutrition. 130: 507- 513.
33. O'Keefe, J. H., Gheewaha, N. M., Joan, M. S. and O'Keefe, D. (2008): Dietary strategies for improving post- prandial glucose, lipids, inflammation, and cardiovascular health. Journal of the American College of Cardiology. 51 (3): 249 – 255.
34. Patton, C. J. and Crouch, S. R. (1977): Spectrophotometric and kinetics investigation of the Berthelot reaction for the determination of ammonia. . Analytical Chemistry. 49 (3): 464- 469.
35. Pitchai, D. and Manikkam, R. (2011): Hypolipidemic, hepato- protective and renal damage recovering effects of catechin isolated from methanolic extract of cassia fistula stem bark on streptozotocin – induced diabetic Westar rats: a biochemical and morphological analysis. Medicinal Chemistry Research. 20: 1554- 8120.
36. Reddy, S. S., Ramatholisamma, P., Karuna, R. and Saralakumari, D. (2009): Preventive effect of Tinospora Cordifalia against high- fructose diet – induce insulin resistance and oxidative stress in mal Wister rats. Food Chemical Toxicology.
37. Reeves, R. G.; Nielsen, F. H. and Fahey, G. C. (1993): AIN-93- Purified diets for laboratory rodents. Journal of Nutrition. 123 (1):139.

38. Reitman, S. and Frankal. S. (1957): A calorimetric method for determination of serum AST. American Journal Clinical Pathology. 18: 26.
39. Riccillo, F. L., Bracanonat, M. I., Martinez, S. and Ronderos, J. R. (2012): Progressive rat modal of type-2 diabetic. Tissue and Cell. 44: 101 – 110.
40. Richmond, W. (1973): Preparation and properties of a cholesterol oxidase from Nacardia SP. enzymatic assay of total cholesterol in serum. Clinical Chemistry. 19 (12):1350- 1356.
41. Salbe, A. D., Johnston, C, S., Buyukbese, A. M., Tsitouros. P. D. and Harman, S. M. (2009): Vinegar lacks antiglycemic action on enterol carbohydrate absorption in human subject. Nutrition Research. 29: 846- 849.
42. Sengun, I. Y. and Karapinar. M. (2005): Effectiveness of Salmonella Typhimurium on rocket (Eruca Sativa Miller) and spring onion (Alliums Cape L.). International Journal of Food Microbiology. 98: 319 – 323.
43. Shahidi, F., McDonald, J. Chandrasekhar, A. and Zhong, Y. (2008): Phytochemical of foods, beverages and fruit vinegars: chemistry and health effects. Asia Pacific Journal Clinical Nutrition. 17: 380-382.
44. Shimoji, Y., Tamura, Y., Nakamura, Y., Nanda, K., Nishidai, S. And Nishikawa, Y. (2002): Isolation and identification of DPPH radical scavenging compounds in Kurusu (Japanese unpolished rice vinegar). Journal of Agricultural and Food Chemistry. 50: 6501- 6503.
45. Shishehbor, F.; Mansoori, A.; Sarkaki, A. R.; Jalali, M. T. and Latifi. M . (2008): Apple cider vinegar attenuates lipid profile in normal and diabetic rats. Pakistan Journal of Science, 11 (23): 2634- 2638.
46. Snedecoer, G. and Cochran, W. G. (1967): Statistical Methods, 6th ed Lowe State University Press. Amers, JOWA, U.S.A.
47. Sugiyama, M., Tang, A. C. , Wakaki, Y. And Koyama, W. (2003): Glycemic index of single and mixed meal foods among common Japanese foods with white rice as a reference food. European Journal Clinical Nutrition, 57: 743- 752.
48. Suwamaphat, W., Meeprom. A., Yibchok- Anun, S., Adisakwattana, S. (2010): Preventive effect of grape seed extract against high- fructose diet induced insulin resistance and oxidative stress in rats. Food and Chemical Toxicology. 48: 1853- 1857.
49. Takano- Lee, M., Adman, J. D., Mullens, B. A. and Clark, J. M. (2004): Home remedies to control head louse, pediculus human us capitis (Anoplura: pediculidae). Journal for Specialists in Pediatric Nursing. 19: 393- 398.
50. Trinder, P. (1969): Determination of glucose in blood. Annals Clinical Chemistry.6: 24.
51. Veerapur, V. P., Prabhakar, K. R., Thippeswamy. B. S., Bansal, P., Srinivasan, K. K. And Unnikrishnan, M. K. (2010): Antidiabetic effect of *Dodonaea viscosa* (L) Lacq. Aerial qeriul parts in high fructose – fed insulin resistance rats, A mechanism based study. Indian Journal of Experimental Biology. 48: 800- 810.
52. Verma, N., Amresh, G., Sahu, P. K., Mishra, N., Singh, A. P. and Ch-V, R. (2012): Antihyperglycemic activity, antihyperlipidemic activity, hematological effects and histopathological analysis of *Sapindu mukorossi* Gaerten fruit in streptozotocin induce diabetic rats. Asia Pacific Journal of Tropical Medicine. 518- 522.
53. Vijayakumar, C. and Wolf- Hall, C. E. (2002): Evaluation of household sanitizers for reducing levels of *Escherichia coli* on iceberg lettuce. Journal Food Protection. 65: 1646 – 1650
54. Wieland, H. and Seidel. D. (1983): A Simple specific method for precipitation of low density lipoprotein. Journal of Lipid Research. 24 (7): 904- 909.
55. Wild, S., Roglic, G., Green, A., Sigree, R. and King, H. (2004): Global prevalence of diabetes. Diabetes Care. 27: 1047 – 1053.
56. Xuemei, G., Hai- Lu., Jing, G., Juliana. C. N. and Peter, C. Y. T. (2012): White rice vinegar improves pancreatic beta- cell function and fatty liver in streptozotocin induced diabetic rats. Acta Diabetol. 49: 185 – 191.
57. Yador, S. P., Vats, A., Ammin, A. and Grover, J. K. (2004): Brassica Juncea (Rai) significantly prevented the development of insulin resistance in rats fed fructose enriched diet. Journal of Ethropharmacology. 93 (1): 113- 116
58. Yamashita, H. , Fujisaw, K., Ito, E., Idei, S., Kawaguchi, N., Kimoto, M., Hiemori, M. And Tsuji, H. (2007): Improvement of obesity and glucose tolerance by acetate in Type-2 diabetic otsuka Long- Evans Tokushiwa Fatty (OLETF) rats. Bioscience Biotechnology and Biochemistry. 17: 1236- 1243.
59. Zbigniew, J. W., Bogumit, T. and Marck, S. L. (1991): Chromatographic determination of citric acid monitoring the mould process. Journal of Chromatography A. 558 (1): 302- 305.
60. Zimmet, P. (2000): Globalization, coca – colonization and chronic disease epidemic: can the doomsday scenario be averted? Journal International Med. 247: 301 – 210.

10/10/2012