Evaluation of the Health Value of some Beverages Prepared from Vegetable and Fruit Wastes

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Abstract: This study aims to evaluate the health value of some beverages prepared from the wastes of fruits and vegetables. Artichoke leaves, pomegranate peel and orange peel were used to prepare beverages that were proved to be palatable and accepted by panel testing. An experiment was done on rats fed on high cholesterol diet without or containing 20% of the dry matter of each of these fruit or vegetable wastes alone or in combination for 6 weeks followed by 2 weeks on the water extract. The health value was evaluated through determination of parameters that express the oxidation state such as plasma molondialdehyde, antioxidant enzymes and nitric oxide, lipid pattern as total cholesterol (TC), LDL-C, HDL-C and triacylglycerols (TG). The liver and kidney functions were assessed by determination of the activities of AST, ALT, urea and creatinine. The results showed that the level of plasma malondialdehyde of rats fed on the high cholesterol diet significantly increased and returned back to near normal control value when the vegetable or the fruits wastes were added. The activities of each of the antioxidant enzymes superoxide dismutase (SOD), catalase and glutathione peroxidase were all decreased due to consumption of the high cholesterol diet. When vegetable or the fruit wastes were added, the activities were within the normal control range. Plasma nitric oxide level of control rats was 14.76 ± 0.64 µmol/L and changed to 18.99 ± 0.72 µmol/L for rats fed on high cholesterol diet. Addition of fruit or vegetable wastes did not correct this change. The value of TC of rats fed on the high cholesterol diet was remarkably increased from a value of 94.8 ± 4.03 to 262.78 ± 8.99 mg/dl. The LDL-C & TG were increased. The HDL-C was decreased. Addition of fruit or vegetable wastes powder to the diet lowered the increase in the plasma cholesterol. The values obtained were 141.35 ± 5.96, 159.26 ± 6.51, 150.53 ± 5.75, 169.79 ± 5.44 and 162.02 ± 6.53 mg/dl for rats given each of pomegranate peel, orange peel, pomegranate +orange peel, artichoke leaves, or artichoke leaves + orange peel, respectively. The change in parameters denoting the liver and kidney functions were corrected by addition of fruit and vegetable wastes to the diets. The conclusion is that the natural phytochemicals present in these fruit or vegetable wastes that have antioxidant properties succeeded to protect against oxidative free radicals and in turn prevent chronic diseases. It is recommended to use the beverages prepared from these wastes to make use of their health value and palatability.

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1. Introduction:

A considerable amount of food wastes are generated by the fruit-and-vegetable industries and handling. Forty five billion kg of fresh vegetables, fruits, milk, and grain products are lost to waste in the United States every year. In the United Kingdom, 20 million ton of food waste is produced annually (Maria, 2009). Fruits and vegetable processing in Ireland generates substantial quantities of waste/by-products (Environmental Protection Agency, 2003). In Egypt, estimates up to 30% of fruit and vegetable production are wasted (Industrial Union of Egypt, 2011). Studies concerning residual sources have been augmented considerably caused by a value adding recycling interest of the agro- and food industry, and also to increase information on the specific location of active compounds and their modification during processing (Alonso, et al., 2002; and Amro, et al., 2002). Several studies reported that wastes and by-products of fruits may be an abundant source of antioxidant polyphenols (Peschel et al., 2006 & Wijngaard et al., 2009). At the present time, fruit and vegetable wastes and by-products are disposed often at a cost to the manufacturer. Therefore, using these wastes in a suitable form as a source of polyphenols may be of considerable economic benefit to food processors. Fruit and vegetable wastes and by-products can range from pomace (leftovers after pressing), to cabbage cut-offs and whole fruits and vegetables.

Antioxidant polyphenols play an important role as health-protective factor, since they neutralize the hazardous effect of free radicals in the cell and in turn protect against chronic diseases. Most of those diseases genetically referred are linked to the aging process and have been associated with oxidative damage, a constant process that occurs in aerobic organisms. Reactive oxygen species or free radicals are produced in the mitochondria as normal by-products in

the phosphorylating chain of ATP (Harman, 1972) by enzymatic reactions, such as the NADPH oxidase in the phagocytes, to destroy invading microbes, or xanthine oxidase for uric acid formation (Halliwell and Gutteridge, 1989). In addition, exposure to environmental pollution such as cigarette smoke, and ultraviolet radiation cause the production of free radical (Dean et al. 1997). Free radicals are highly reactive; they react with other molecules, to return to the stable states, rendering this molecule another free radical (Halliwell and Gutteridge, 1989). Excessive production of free radicals leads to several consequences, it causes damage to lipids, proteins, carbohydrates, and DNA (Dean et al.,1997) cell membranes lose their ability to properly transport nutrients; lipoproteins are oxidized ; and damaged DNA has the potential to accumulate consecutive mutations, which can lead to carcinogenesis (Scalbert et al., 2005). This shows that antioxidants that are able to slow down the rate of the oxidative process are important for the homeostasis of normal body metabolism. Epidemiologic data show that the consumption of Fruits and Vegetables is insufficient both in adults and children, and their low intake is among the top 10 risk factors contributing to attributable mortality (Lock et al., 2005 & WHO, 2003).

It is thus necessary to search for any source rich in phytochemical antioxidants that are bioavailable and therefore able to prevent or retard chronic diseases and prepare them in an acceptable, palatable and attractive form to the consumers. In a previous study (El-Shobaki et al., 2011), it was possible to produce a number of beverages prepared from vegetable and fruit wastes. These beverages proved to be palatable and accepted as evidenced from panel testing. They do not cause any health hazards and promote growth.

The aim of the present investigation is to assess the health value of these beverages on experimental animals regarding lipid pattern, antioxidant state, activities of antioxidant enzymes and nitric oxide.

2. Material and Methods

Three vegetable and fruit wastes were chosen for this study namely pomegranate peel, orange peel & artichoke leaves. The fruits and vegetables were purchased from the local market during their ripening season.

Preparation of vegetable and fruit wastes

Pomegranate peel, orange peel and artichoke leaves were taken, washed carefully with running water till being completely clean. They were then dried in an air circulating oven regulated at a temperature of 60 C^o until complete dryness. The dry matter was then ground in a mixer to fine powder and then kept in clean dry containers till used. The water extract was prepared by soaking the suitable amount of the powder in boiling water for one hour.

Preparation of Diets

The standard control diet was prepared according to Reeves et al., (1993) as shown in table (1). The high cholesterol diet was prepared by adding 2% pure cholesterol, 0.25% bile salts and 20% fat to the standard diet. The other diets given to the rats that contain the vegetable and fruit powder were prepared by adding the amount of powder to the diet on the expense of starch (table 2). The concentrations used were 20% pomegranate peel powder, 20% orange peel powder, 15% pomegranate peel powder + 5% orange peel powder, 20% artichoke leave powder, 15% artichoke leave powder + 5% orange peel powder.

Animal experiment

This experiment was done on male Sprague dawley rats of body weight ranging from 129 to 215 g. They were obtained from the Central Animal House, National Research Centre. Rats were divided into 7 groups each of 7 rats. Each rat was housed individually into separate cage in an air conditioned room regulated at a temperature of 25 C^0. Food and water were admitted to the rats ad-libitum. Group1 was given the control standard diet, group 2 was given the high cholesterol diet, group 3 was given the high cholesterol diet that contain the pomegranate peel powder (20%), group 4 was given the diet that contain the orange peel powder 20%, group 5 was given the diet that contain 15% pomegranate peel powder + 5% orange peel powder, group 6 was given 20% artichoke leave powder and group 7 was given 15% artichoke leave powder + 5% orange peel powder. The experiment lasted for 6 weeks on the diets containing the powder form of the fruit and vegetable wastes then another 2 weeks on each of the same diet but containing the water extract of the same amount of the powder. At the end of the 6 week period, a blood sample was taken from each rat in the different groups from the orbital vein under slight diethyl ether anesthesia for determination of the different biochemical parameters.

At the end of the experiment (8 weeks), Rats were fasted overnight; in the morning, blood was withdrawn from each rat by open heart puncture under slight diethyl ether anesthesia over heparin. Blood was centrifuged at 3500 rpm for 15 minutes and plasma was separated and stored at -20 C^o until analysis. RBCs were separated and washed by saline 3 times and stored at -70 C^o until analysis of antioxidant enzymes.

Biochemical parameters

The analyzed parameters were determined using kits from Stanbio Laboratories, Texas, USA, for determination of triacylglycerols, total cholesterol, high

density lipoprotein cholesterol, creatinine, uric acid and total proteins. Kits used for determination of low density lipoprotein cholesterol, urea, AST and ALT were obtained from Quimica Clinica Aplicada S. A., Spain. Kits used for determination of nitric oxide, malondialdehyde, superoxide dismutase (SOD), glutathione peroxidase (GPx) and catalase were obtained from Biodiagnostic Company, Egypt.

Plasma triacylglycerol (TG) was determined according to Scheletter and Nussel (1975). Plasma total cholesterol was estimated as described by Allain et al., (1974). Plasma HDL-cholesterol was determined according to Warnick and Albers (1978). Plasma LDL-cholesterol was determined as described by Assmann et al., (1984). Plasma ALT and AST were estimated according to Bergmeyer (1985). Plasma total protein was determined according to Cannon et al., (1974). Plasma urea was estimated according to the method of Fawcett and Soctt (1960). Plasma creatinine was determined as described by Bartles et al., (1972). Plasma uric acid was assessed according to Fossati et al., (1980). Plasma malondialdehyde was determined according to the method of Ohkawa (1979). Plasma nitric oxide was estimated according to Montgomery & Dymock (1961). Antioxidant enzymes including; RBCs superoxide dismutase (SOD) was determined according to the method described by Nishikimi, et al., (1972), RBCs glutathione peroxidase was estimated according to the method of Paglia and Valentine (1967) and plasma catalase was estimated as described by Aebi (1984). The concentration of all of the previously mentioned biochemical parameters were measured by a colorimetric technique using a spectrophotometer (Shimadzu UV-2401 PC, Australia).

Data were represented as mean ± SE, and statistically analyzed by independent "T" test. A probability level of 0.05 or less ($P<0.05$) was taken as a significance level. Statistical analysis was performed using SPSS program, version "17".

3. Results

Malondialdehyde and nitric oxide

As shown in table (3) the level of plasma malondialdehyde of rats fed on the high cholesterol diet was significantly increased. This increase returned back to near normal control value when the vegetable or the fruit wastes were added to the diets.

The value for nitric oxide of control rats (table 3) was 14.76 ± 0.64 µmol/L. This value increased to 18.99 ± 0.72 µmol/L for rats fed on high cholesterol diet. Addition of pomegranate to the diet even caused an increase in this value to 22.0 ± 1.19 µmol/L, 18.99 ± 0.72 µmol/L in case of orange peel, 15.64 ± 0.87 µmol/L in case of pomegranate peel + orange peel,17.57 ± 1.22 µmol/L in case of artichoke leaves and 16.66 ± 0.78 µmol/L in case of artichoke leaves + orange leaves.

Antioxidant enzymes

In general, the activities of each of the antioxidant enzymes superoxide dismutase (SOD), Catalase and glutathione peroxidase were all decreased due to consumption of the high cholesterol diet (table 4). The values obtained were 453.0 ± 12.9 U/ml, 521.9 ± 17.69 U/L, 806.2 ± 43.3 mU/ml, respectively relative to values of 502.5 ± 13.0 U/ml, 739.3 ± 15.81 U/L, 11138.9 ± 41.65 mU/ml for control rats. When each of the vegetable or the fruit wastes was added to the diet, the activities of these enzymes were within the normal control range.

Lipid pattern

The lipid pattern of groups of rats used including total cholesterol, (LDL-C), (HDL-C) and (TG) are shown in table (5). As shown in the table, the TC of rats fed on the high cholesterol diet was remarkably increased from a value of 94.8 ± 4.03 to 262.78 ± 8.99 mg/dl. The LDL-C and TG of rats fed on the high cholesterol diet were also increased. The high density lipoprotein was decreased. The addition of any of the fruits or the vegetable waste powder to the diet of the rats lowered the increase in the plasma cholesterol that occurred due to consumption of the high cholesterol diet. The values obtained were 141.35 ± 5.96, 159.26 ± 6.51, 150.53 ± 5.75, 169.79 ± 5.44 and 162.02 ± 6.53 mg./dl for rats given each of pomegranate peel, orange peel, pomegranate + orange peel artichoke leaves, or artichoke leaves + orange peel, respectively. A similar lowering effect occurred in the level of LDL-C of rats fed on the diets containing the fruits and the vegetable powder. The obtained values ranged between 141.35 ± 5.96 & 69.79 ± 5.44 mg/dl. The drop in the level of HDL-C that occurred due to the high cholesterol diet was not observed when the vegetable or the fruits waste powder was added to the diets, the values obtained were even higher than the control value. The slight increase in the level of TG due to consumption of the high cholesterol diet was not observed in rats fed on the diets that contain the vegetables or the fruits wastes, the values were even lower.

Liver function

The liver function of rats in different groups was assessed by estimation of the activities of each the enzymes AST, ALT and total protein. The values are reported in table (6). The activities of the liver enzymes AST and ALT were elevated due to consumption of the high cholesterol diet. The total plasma protein was also increased. The values reported to the activities of the enzymes were 51.57 ± 2.26 U/ml for AST, 20.0 ± 1.09

U/ml for ALT and 7.4 1± 0.14 g./dl for plasma protein. These values were changed to 64.57 ± 1.71, 26.6 ± 1.41 and 8.32 ± 0.13 respectively. The increased activity of the AST due to high cholesterol consumption was not observed when any of the fruits or vegetables powder was added to the diet of the animals. The increased activity of the ALT persisted in most cases. The total plasma protein was within normal standard value.

Kidney function

The values of plasma urea, uric acid and creatinine were determined and used as indicators to kidney function (table 7). The values reported to these parameters were 14.49 ± 0.76 mg /dl for urea, 5.06 ± 0.28 mg/dl for uric acid and 0.73 ± 0.01 mg/dl for creatinine. These values were all raised to 18.80 ± 1.02, 17.9 ± 0.42 and 0.91 ± 0.03 when the rats consumed the high cholesterol diet for 6 weeks. When the vegetables or the fruits waste powder was added to the high cholesterol diet, the level of plasma urea was found to be lower than before addition but did not reach the normal value. Plasma uric acid was also lower but not normalized. Plasma creatinine of all groups taken the diet with the added fruits and vegetables waste powder were within normal range.

Biochemical parameters at the end of the experiment (8 weeks)

To test whether the water extract of these fruit or vegetable wastes could exert similar effect like the dry matter, the latter was replaced by the water extract. At week 6 the dry matter of vegetable and fruit wastes were replaced by the water extract and lasted till the end of the eighth week. Another blood sample was obtained from the animals in different groups and analyzed for the same parameters. The data obtained are shown in tables (8, 9, 10, 11 & 12). A general pattern was observed that most of the trends that occurred in the first treatment still persist in the second.

4. Discussion

It is evidenced from the results that consumption of high cholesterol diet caused considerable health complications. Several investigators reported the health hazards due to consumption of high cholesterol food. It has been shown that hypercholesterolemia increases oxidative stress and leads to lipid peroxidation (Cox et al., 1996). Oxidative stress is involved in the pathogenesis of a number of diseases, and feeding a high cholesterol diet increases tissue oxidative stress in various organs (Balkan et al., 2002 & Homma et al., 2004). It was suggested that excessive tissue oxidative damage induced by high dietary cholesterol could potentiate pro-inflammatory cytokine production by fibroblasts stimulated with bacterial pathogens (Tomofuji et al., 2006). Oxidative stress is involved in the pathogenesis of a number of diseases, including periodontitis (Wei et al., 2004). It is clear that, the high cholesterol diet used in this experiment could induce many of the health hazards reported by different investigators. This shows how important it is to find a way or a mean to avoid these health complications. The beverages produced in this study which was prepared from the vegetable or fruit wastes can be that mean. These beverages proved to contain considerable number and quantities of the polyphenolic antioxidants (El-Shobaki et al., 2011) which are believed to participate in prevention of these health hazards.

The results from this study show that hypercholesterolemia increased oxidative stress evidenced by the increase in the level of plasma malondialdehyde in rats fed on the high cholesterol diet and the remarkable decrease in the activities of the antioxidant enzymes namely SOD Catalase and GPx. Several investigators showed that high-cholesterol diet induced reactive oxygen species (ROS) overproduction which could in turn initiate lipid peroxidation (Martinet et al., 2001,Balkan et al., 2004 & Montilla et al., 2004). This may explain the increase in the malndialdehyde and the decrease in the antioxidant enzymes reported in rats fed on the high cholesterol diet.

Rats fed on the high cholesterol diet showed a high plasma nitric oxide level. Devrim et al., (2008) reported that cholesterol feeding causes an increase in xanthine oxide activity and a decrease in nitric oxide synthase activity in the erythrocytes from rats. Nitric oxide synthase catalyzes the reaction by which L-arginine is converted to L-citrulline, and nitric oxide is produced. This means a reduction in nitric oxide level of rats fed on the high cholesterol diet which is contrary to our finding. However, it has been reported that in spite of the hypercholesterolemia, rats are resistant to atherosclerosis a condition associated with low level of plasma nitric oxide (Balkan et al., 2004). Thus it is assumed that a different mechanism exists in rats compared to rabbits, and this can be the reason behind the resistance of rats to atherosclerotic action.

Addition of the dry matter of vegetable or fruit wastes to the diet caused a return to near normal values of the parameters malondialdehyde, nitric oxide and the activities of the antioxidant enzymes. Phenolic substances present in these vegetable and fruit wastes are believed to be behind this effect. They have been implicated in increasing the antioxidative systems, acting as enzyme modulators and metal chelators (Butera et al., 2002 & Edenharder & Grunhage, 2003). These agents inhibit peroxidation reactions and significantly reduce the oxidative stress (Fuhrman et al., 2001 & Pari & Saravanan, 2002). Moreover, a high consumption of phenolic compounds has already been found to decrease serum cholesterol and triacylglycerol

concentrations in rat (Afaf et al., 2000). The beverages used in this study could realize a similar effect. The high plasma total cholesterol, LDL-C and triacylglycerol that occurred due to consumption of the high cholesterol diet were corrected when rats received the diets that contain the vegetable and fruit wastes. As reported before, these agents inhibit peroxidation reactions and significantly reduce the oxidative stress. There is an increasing amount of evidence that a shift in scavenging activity and redox status is one of the mechanisms that might be activated in hypercholesterolemia and might hinder health complications. This is supported from our finding that the deterioration in liver and kidney functions that occurred due to hypercholesterolemia were corrected when the fruit and vegetable wastes or their extracts were included in the diet.

It is worth mentioning that the results obtained for the biochemical parameters analyzed for rats that were given diets containing the water extract of the vegetable or fruits waste have more or less the same trend as those given the diets containing dry wastes for six weeks. This shows that the water extract contain most of the phytocompounds with antioxidant power that could keep on the protective effect of these fruit and vegetable wastes.

In conclusion, this study shows that high cholesterol consumption leads to several health complications caused mainly by oxidative stress affecting the lipid profile, liver and kidney functions. It is possible to prevent these complications by regular consumption of beverages prepared from wastes of some fruits and vegetables that are rich in phytochemicals with antioxidative power. It is recommended to use these beverages to make use of their health value.

Table (1): Composition of control diet (g/100g diet).

Ingredients	Amount
Skim milk*	42.9
Sucrose	5
Cellulose	4
Corn oil	8
Salt mixture (AIN-93)*	3.5
Vitamin mixture (AIN-93)*	1
Choline choloride	0.06
Corn starch	35.5

* Protein content of skim milk was estimated as 31%.
* Control diet was prepared according to Reeves et al., 1993.
* Salt and vitamin mixtures were prepared according to Reeves et al., 1993.

Table (2): Composition of diets given to groups from 2 to 7 (g/100g diet).

Group / Ingredients	Gr 2	Gr 3	Gr 4	Gr 5	Gr 6	Gr 7
Skim milk	42.9	42.9	42.9	42.9	42.9	42.9
Fat	20	20	20	20	20	20
Cholesterol	2	2	2	2	2	2
Bile salts	0.25	0.25	0.25	0.25	0.25	0.25
Sucrose	5	5	5	5	5	5
Salt mixture (AIN-93)*	3.5	3.5	3.5	3.5	3.5	3.5
Vitamin mixture (AIN-93)*	1	1	1	1	1	1
Choline choloride	0.06	0.06	0.06	0.06	0.06	0.06
Corn starch	25.29	5.29	5.29	5.29	5.29	5.29
Pomgraneet peel powder	0	20	0	15	0	0
Orange peel powder	0	0	20	5	0	5
Artichoke leave powder	0	0	0	0	20	15

Table (3): Plasma malondialdehyde & nitric oxide concentrations of different groups of treatment (dry matter) compared to the normal control and to the hypercholesterolemic rats.

Group \ Test	Malondialdehyde nmol/ml	Nitric oxide µmol/L
Control		
mean ± SE	1.98 ± 0.07	14.76 ± 0.64
Hypercholesterolemia		
mean ± SE	2.59 ± 0.11	18.99 ± 0.72
Pa<	0.001	0.01
Hyper.+ Pome. peel		
mean ± SE	1.94 ± 0.09	22.00 ± 1.19
Pa<	N. S.	0.001
Pb<	0.001	0.05
Hyper+Orange peel		
mean ± SE	2.26 ±0.07	18.44 ± 1.58
Pa<	0.02	N. S.
Pb<	0.05	N. S.
Hyper+Pome. peel+Orang peel		
mean ± SE	1.89 ± 0.09	15.64 ± 0.87
Pa<	N. S.	N. S.
Pb<	0.001	0.02
Hyper+Artich. leaves		
mean ± SE	2.04 ± 0.04	17.57 ± 1.22
Pa<	N. S.	N. S.
Pb<	0.01	N. S.
Hyper+Artich. leaves+Orang peel		
mean ± SE	1.99 ± 0.05	16.66 ± 0.78
Pa<	N. S.	N. S.
Pb<	0.001	0.05

Table (4): RBCs SOD, glutathione peroxidase (GPx) and plasma catalase concentrations of rats in different treated (dry matter) groups compared to the normal control and to the hypercholesterolemic rats.

Group \ Test	SOD (U/ml)	Catalase (U/L)	GPx (mU/ml)
Control			
mean ± SE	502.5 ± 13.0	739.3 ± 15.81	1138.9 ± 41.65
Hypercholesterolemia			
mean ± SE	453.0 ± 12.9	521.9 ± 17.69	806.2 ± 43.4
Pa<	0.02	0.001	0.001
Hyper.+ Pome. peel			
mean ± SE	494.2 ± 11.3	726.3 ± 25.4	1060.8 ± 23.8
Pa<	N. S.	N. S.	N. S.
Pb<	0.02	0.001	0.001
Hyper+Orange peel			
mean ± SE	511.2 ± 14.0	706.7 ± 24.1	1031.8 ± 18.07
Pa<	N. S.	N. S.	N. S.
Pb<	0.02	0.001	0.001
Hyper+Pome. peel+Orang peel			
mean ± SE	501.7 ± 8.5	703.7 ± 19.4	1109.9 ± 22.4
Pa<	N. S.	N. S.	N. S.
Pb<	0.02	0.001	0.001
Hyper+Artich. leaves			
mean ± SE	523.9 ± 9.4	719.3 ± 22.0	1084.7 ± 21.4
Pa<	N. S.	N. S.	N. S.
Pb<	0.01	0.001	0.001
Hyper+Artich. leaves+Orang peel			
mean ± SE	541.8 ± 13.8	696.8 ± 24.3	1072.4 ± 12.5
Pa<	N. S.	N. S.	N. S.
Pb<	0.001	0.01	0.001

Table (5): Plasma Cholesterol & Triacylglycerol concentrations of rats in different treated (dry matter) groups compared to normal rats and to the hypercholesterolemic ones .

Test / Group	TC (mg/dl)	TG (mg/dl)	LDL-C (mg/dl)	HDL-C (mg/dl)
Control mean ± SE	94.8 0± 0.64	52.0 ± 1.84	35.52 ± 3.11	32.58 ± 2.42
Hypercholesterolemia mean ± SE Pa<	262.78 ± 8.99 0.001	56.44 ± 1.05 0.05	170.71 ± 9.86 0.05	12.37 ± 1.04 0.001
Hyper.+ Pome. peel mean ± SE Pa< Pb<	141.35 ± 5.96 0.001 0.001	48.40 ± 1.27 N. S. 0.001	68.61 ± 5.62 0.001 0.001	41.83 ± 2.64 0.05 0.001
Hyper+Orange peel mean ± SE Pa< Pb<	159.26 ± 6.51 0.001 0.001	44.57 ± 2.28 0.05 0.01	79.75 ± 7.53 0.001 0.001	45.69 ± 3.01 0.01 0.001
Hyper+Pome. peel+Orang peel mean ± SE Pa< Pb<	150.53 ± 5.75 0.001 0.001	32.69 ± 1.99 0.001 0.001	60.73 ± 5.21 0.001 0.001	44.46 ± 3.16 0.02 0.001
Hyper+Artich. leaves mean ± SE Pa< Pb<	169.79 ± 5.44 0.001 0.001	36.60 ± 1.78 0.001 0.001	53.08 ± 4.56 0.01 0.001	50.39 ± 3.37 0.01 0.001
Hyper+Artich. leaves+Orang peel mean ± SE Pa< Pb<	162.02 ± 6.53 0.001 0.001	29.81 ± 2.6 0.001 0.001	44.3 ± 3.99 0.05 0.001	62.91 ± 4.12 0.001 0.001

Table (6): Plasma AST, ALT & total protein of rats in different treated (dry matter) groups compared to the normal control and to the hypercholesterolemic rats .

Test / Group	AST (U/ml)	ALT (U/ml)	Total protein (g/dl)
Control mean ± SE	51.57 ± 2.26	20.0 ± 1.09	7.41 ± 0.14
Hypercholesterolemia mean ± SE Pa<	64.57 ± 1.71 0.001	26.6 ± 1.41 0.01	8.32 ± 0.13 0.01
Hyper.+ Pome. peel mean ± SE Pa< Pb<	57.1 ± 1.89 N. S. 0.02	23.6 ± 1.21 0.05 N. S.	7.46 ± 0.24 N.S. 0.02
Hyper+Orange peel mean ± SE Pa< Pb<	50.0 ± 1.70 N. S. 0.001	27.0 ± 2028 0.02 N. S.	7.58 ± 0.26 N. S. 0.05
Hyper+Pome. peel+Orang peel mean ± SE Pa< Pb<	45.4 ± 1.66 0.05 0.001	25.1 ± 2.23 N. S. N. S.	7.37 ± 0.25 N. S. 0.01
Hyper+Artich. leaves mean ± SE Pa< Pb<	48.57 ± 1.88 N. S. 0.001	27.0 ± 1.01 0.01 N. S.	7.45 ± 0.15 N. S. 0.01
Hyper+Artich. leaves+Orang peel mean ± SE Pa< Pb<	45.71 ± 1.88 N. S. 0.001	26.9 ± 1.39 0.01 N. S.	7.63 ± 0.16 N. S. 0.01

Table (7): Plasma urea, uric acid & creatinine concentrations of different treated (dry matter) groups compared to the normal control and to the hypercholesterolemic rats.

Group \ Test	S. Urea (mg/dl)	S. Uric acid (mg/dl)	S. Creatinine (mg/dl)
Control			
mean ± SE	14.49 ± 0.79	5.06 ± 0.28	0.73 ± 0.03
Hypercholesterolemia			
mean ± SE	18.80 ± 1.02	17.9 ± 0.42	0.91 ± 0.03
Pa<	0.01	0.001	0.01
Hyper.+ Pome. peel			
mean ± SE	16.84 ± 1.49	13.5 ± 0.36	0.71 ± 0.02
Pa<	N. S.	0.001	N. S.
Pb<	N. S.	0.02	0.001
Hyper+Orange peel			
mean ± SE	16.96 ± 0.81	12.66 ± 0.4	0.77 ± 0.03
Pa<	0.05	0.001	N. S.
Pb<	N. S.	0.01	0.01
Hyper+Pome. peel+Orang peel			
mean ± SE	14.46 ± 0.76	8.24 ± 0.36	0.73 ± 0.03
Pa<	N. S.	0.001	N. S.
Pb<	0.01	0.001	0.01
Hyper+Artich. leaves			
mean ± SE	16.69 ± 0.83	5.64 ± 0.24	0.69 ± 0.04
Pa<	N. S,	N. S.	N. S.
Pb<	N. S.	0.001	0.001
Hyper+Artich. leaves+Orang peel			
mean ± SE	15.50 ± 0.88	5.20 ± 0.30	0.71 ± 0.04
Pa<	N. S.	N. S.	N. S.
Pb<	0.05	0.001	0.01

Table (8): Plasma malondialdehyde & nitric oxide concentrations of different treated (water extract) groups compared to the normal control and to the hypercholesterolemic rats.

Group \ Test	Malondialdehyde (nmol/ml)	Nitric oxide (µmol/L)
Control		
mean ± SE	2.12 ± 0.12	14.07 ± 0.56
Hypercholesterolemia		
mean ± SE	3.11 ± 0.18	17.21 ± 0.79
Pa<	0.001	0.01
Hyper.+ Pome. peel		
mean ± SE	2.22 ± 0.08	15.09 ± 0.48
Pa<	N. S.	N. S.
Pb<	0.001	0.05
Hyper+Orange peel		
mean ± SE	1.82 ± 0.05	12.5 ± 0.64
Pa<	0.02	N. S.
Pb<	0.001	0.001
Hyper+Pome. peel+Orang peel		
mean ± SE	1.98 ± 0.03	14.79 ± 0.61
Pa<	N. S.	N. S.
Pb<	0.001	0.05
Hyper+Artich. leaves		
mean ± SE	1.66 ± 0.09	15.73 ± 0.62
Pa<	0.02	N. S.
Pb<	0.001	N. S.
Hyper+Artich. leaves+Orang peel		
mean ± SE	2.09 ± 0.07	14.69 ± 0.48
Pa<	N. S.	N. S.
Pb<	0.001	0.02

Table (9): RBCs SOD, plasma catalase & RBCs glutathione peroxidase (GPx) concentrations of different treated (water extract) groups compared to the normal control and to the hypercholesterolemic rats.

Group \ Test	SOD (U/ml)	Catalase (U/L)	GPx (mU/ml)
Control			
mean ± SE	539.9 ± 16.9	705.3 ± 14.9	1232.0 ± 39.9
Hypercholesterolemia			
mean ± SE	458.80 ± 12.3	554.2 ± 18.0	803.8 ± 33.6
Pa<	0.01	0.001	0.001
Hyper.+ Pome. peel			
mean ± SE	513.8 ± 5.18	672.2 ± 24.2	1145.4 ± 36.9
Pa<	N. S.	N. S.	N. S.
Pb<	0.01	0.01	0.001
Hyper+Orange peel			
mean ± SE	515.8 ± 7.2	616.7 ± 19.0	1113.2 ± 48.0
Pa<	N. S.	0.01	N. S.
Pb<	0.01	0.05	0.001
Hyper+Pome. peel+Orang peel			
mean ± SE	518.4 ± 7.7	638.1 ± 15.9	1128.1 ± 40.6
Pa<	N. S.	0.02	N. S.
Pb<	0.01	0.01	0.001
Hyper+Artich. leaves			
mean ± SE	535.5 ± 9.7	609.6 ± 15.6	1180.0 ± 27.1
Pa<	N. S.	0.01	N. S.
Pb<	0.01	0.05	0.001
Hyper+Artich. leaves+Orang peel			
mean ± SE	525.9 ± 11.9	605.1 ± 17.4	1142.8 ± 31.1
Pa<	N. S.	0.01	N. S.
Pb<	0.001	N. S.	0.001

Table (10): Plasma Cholesterol & Triacylglycerol concentrations of different treated (water extract) groups compared to the normal control and to the hypercholesterolemic rats .

Group \ Test	TC (mg/dl)	TG (mg/dl)	LDL-C (mg/dl)	HDL-C (mg/dl)
Control				
mean ± SE	72.61 ± 5.9	49.23 ± 1.53	30.32 ± 3.05	32.49 ± 2.67
Hypercholesterolemia				
mean ± SE	255.64 ± 9.98	56.79 ± 1.35	130.11 ± 7.79	12.81 ± 1.23
Pa<	0.001	0.01	0.001	0.02
Hyper.+ Pome. peel				
mean ± SE	119.72 ± 7.57	42.86 ± 1.49	60.15 ± 5.11	41.52 ± 2.51
Pa<	0.001	0.02	0.001	0.05
Pb<	0.001	0.001	0.001	0.001
Hyper+Orange peel				
mean ± SE	130.26 ± 7.85	42.71 ± 1.36	66.72 ± 5.20	45.9 ± 2.99
Pa<	0.001	0.01	0.001	0.01
Pb<	0.001	0.001	0.001	0.001
Hyper+Pome. peel+Orang peel				
mean ± SE	134.83 ± 6.99	43.21 ± 1.51	52.57 ± 4.37	44.06 ± 3.07
Pa<	0.001	0.02	0.01	0.02
Pb<	0.001	0.001	0.001	0.001
Hyper+Artich. leaves				
mean ± SE	114.31 ± 6.24	34.4 ± 2.56	42.21 ± 3.97	49.85 ± 4.01
Pa<	0.001	0.001	0.05	0.01
Pb<	0.001	0.001	0.001	0.001
Hyper+Artich. leaves+Orang peel				
mean ± SE	163.48 ± 8.32	44.0 ± 1.73	41.16 ± 3.95	60.41 ± 4.53
Pa<	0.001	0.05	0.05	0.001
Pb<	0.001	0.001	0.001	0.001

Table (11): Plasma AST, ALT & total protein of different treated (water extract) groups compared to the normal rats and to the hypercholesterolemic rats.

Group \ Test	AST (U/ml)	ALT (U/ml)	Total protein (g/dl)
Control			
mean ± SE	58.29 ± 2.09	18.00 ± 1.41	7.10 ± 0.15
Hypercholesterolemia			
mean ± SE	79.71 ± 2.14	27.14 ± 1.37	8.16 ± 0.18
Pa<	0.001	0.001	0.001
Hyper.+ Pome. peel			
mean ± SE	62.29 ± 1.87	22.86 ± 1.94	7.25 ± 0.27
Pa<	N. S.	N. S.	N. S.
Pb<	0.001	N. S.	0.02
Hyper+Orange peel			
mean ± SE	56.14 ± 2.17	23.29 ± 1.29	7.36 ± 0.30
Pa<	N. S.	0.02	N.S.
Pb<	0.001	N. S.	0.05
Hyper+Pome. peel+Orang peel			
mean ± SE	50.57 ± 1.89	23.00 ± 0.9	7.52 ± 0.22
Pa<	0.02	0.02	N. S.
Pb<	0.001	0.05	0.05
Hyper+Artich. leaves			
mean ± SE	53.00 ± 2.22	26.71 ± 0.85	7.30 ± 0.18
Pa<	N. S.	0.001	N. S.
Pb<	0.001	N. S.	0.01
Hyper+Artich. leaves+Orang peel			
mean ± SE	49.00 ± 2.26	27.4 ± 2.01	7.29 ± 0.16
Pa<	0.02	0.01	N. S.
Pb<	0.001	N. S.	0.01

Table (12): Plasma urea, uric acid & creatinine concentrations of different treated (water extract) groups compared to the normal control and to the hypercholesterolemic rats.

Group \ Test	S. Urea (mg/dl)	S. Uric acid (mg/dl)	S. Creatinine (mg/dl)
Control			
mean ± SE	13.2 ± 0.76	6.09 ± 0.55	0.63 ± 0.02
Hypercholesterolemia			
mean ± SE	18.9 ± 0.96	19.73 ± 1.46	0.96 ± 0.04
Pa<	0.001	0.001	0.001
Hyper.+ Pome. peel			
mean ± SE	17.20 ± 1.19	15.10 ± 0.74	0.73 ± 0.03
Pa<	0.02	0.001	0.02
Pb<	N. S.	0.02	0.001
Hyper+Orange peel			
mean ± SE	14.69 ± 1.14	12.00 ± 0.78	0.76 ± 0.02
Pa<	N. S.	0.001	0.001
Pb<	0.01	0.001	0.001
Hyper+Pome. peel+Orang peel			
mean ± SE	14.39 ± 0.69	9.57 ± 0.72	0.72 ± 0.02
Pa<	N. S.	0.01	0.01
Pb<	0.01	0.001	0.001
Hyper+Artich. leaves			
mean ± SE	14.43 ± 0.83	6.89 ± 0.44	0.69 ± 0.02
Pa<	N. S.	N. S.	0.05
Pb<	0.01	0.001	0.001
Hyper+Artich. leaves+Orang peel			
mean ± SE	15.80 ± 0.99	6.04 ± 0.60	0.65 ± 0.02
Pa<	N. S.	N. S.	N. S.
Pb<	0.02	0.001	0.001

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