The Effect of Green, Roasted and Decaffeinated Coffee on Serum Glucose, Insulin and Serum Lipid Profile in Diabetic Experimental Animals

Eman A.Sadeek[*1] , Hala, A. Abd El;-Rahman[2] and Waffa, Sh. Ali[3]

[1]Department of Biochemistry & Nutrition -Women's College –Ain –Shams University. [2] Food Tech. Res. Ins. Agric. Res. Center. [3]College of Home Economics, Helwan University. Cairo, Egypt
[*]dr_emansadeek@yahoo.com

Abstract: Aim of the work: Assessing the Effect of green, roasted and decaffeinated coffee on serum glucose, insulin and serum lipid profile in diabetic rat models. Methods: Design of the Study: Thirty female wistar rats weighing 124.5 ±5.41g (mean ±S.D) were divided into 5 groups. The first group served as a control and consumed a standard diet according to (AIN – 93). The other 4 groups were injected intraperitoneally with 105 mg / kg body weight of alloxan . One group was kept without further treatment and served as a positive diabetic control. Groups 3, 4, 5 consumed 5% green, roasted and decaffeinated coffee in drinking water, respectively. The feeding trial continued for four weeks. At the end of the experiments, the animals were sacrificed, blood samples were collected, and the liver, kidney, spleen and heart were separated, washed, dried and weighed. Laboratory investigations Consisted of serum glucose, insulin, calcium, phosphorus and complete lipid profile was determined to test the magnitude of antioxidant potential green, roasted and decaffeinated coffee. Results: The present study show a significant difference ($p < 0.05$) in body weight gain and food intake between all treatment groups , with non significant difference in water intake , relative weight of organs including liver , kidney , spleen and heart . the study also shows significant elevation ($p < 0.05$) in serum glucose and insulin in diabetic control group as compared to normal control group. This indicates uncontrolled hyperglycemia in alloxan diabetic rats. While consumption of green, roasted and decaffeinated coffee resulted in a decrease in serum glucose and insulin ($p < 0.05$) .There is a significant decrease ($p < 0.05$) in serum calcium and serum phosphorus in groups 3,4 and 5 fed green, roasted and decaffeinated coffee respectively indicating an association between coffee consumption and bone health. our results also shows that alloxan injection produced a significant increase($p < 0.05$) in serum total- cholesterol(TC); triacylglycerol (TAG); LDL-C ; VLDL-C and in LDL\ HDL ratio and TC \ HDL ratio however a significant decrease ($p < 0.05$) in serum HDL-C is observed ; In diabetic rats compared to normal control .green, roasted and decaffeinated coffee resulted in a significant decrease ($p < 0.05$) in triacylglycerol (TAG); LDL-C ; VLDL-C and in LDL\ HDL ratio and TC \ HDL ratio .on the other hand a significant increase ($p < 0.05$) in serum HDL-C is observed in green, roasted and decaffeinated coffee groups compared to diabetic rats compared to normal control with the highest value for green coffee .Non significant effect on serum total- cholesterol(TC) reported in this study . Conclusion: The observed improvement in glucose, insulin profile, triacylglycerol and HDL-C confirm the potent biological action of green, roasted and decaffeinated coffee and suggest that chlorogenic acid (a component in coffee) might have an antagonistic effect on glucose transport. Suggesting a novel function of coffee on lowering the risk factors of diabetes and delaying the progress of diabetes complications as well.
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1. Introduction:

Type 2 diabetes is a chronic disease associated with high rates of morbidity and premature mortality(Nathan , 1993)1 An alarming increase in the prevalence of type 2 diabetes is expected,(Wild et al ., 2004) and the need for preventive action is widely acknowledged. While increased physical activity and restriction of energy intake can substantially reduce the incidence of type 2 diabetes (Tuomilehto et al., 2001 Knowler et al., 2002) , insight into the role of other lifestyle factors may contribute to additional prevention strategies for type 2 diabetes.

Coffee is considered one of the most popular beverages consumed in the world due to its pleasant flavor and pharmacological properties(DÓREA and COSTA ., 2005). Prospective and epidemiologic studies of green and especially of roasted coffee consumption has been carried out to investigate its biological effects on lipids, blood pressure and

glycaemia(CORTI et al ., 2002 ; DAGLIA et al ., 2000 and ROBINSON et al. ,2004) . Scientific evidences have demonstrated that green and regular coffee beverages present high antioxidant properties in vivo and in vitro (KARAKAWA, 2004 and SOMOZA et al. ., 2003). Few recent studies have indicated that soluble extracts of green coffee were effective against the high blood pressure in mice(SUZUKI, A. et al. ,2000) and in human(KOZUM. et al. , 2005 and OCHIAI, . et al. 2004). It is possible that its antihypertensive action be related to vasoreactive factors produced and released from the vascular endothelium (OCHIAI, R. et al. 2004).

The roasting process causes a loss of water from the green bean and degradation of many of the compounds including the antioxidant polyphenols; however, there is very little difference in total antioxidants between the different roasts of a bean (Daglia et al., 2000).

There are three main methods of coffee preparation; boiled unfiltered coffee, filtered coffee, and decaffeinated coffee, the latter primarily consumed as instant coffee.

There are over a thousand compounds, many formed during the roasting process,which produce the unique taste and smell of coffee(Parliament et al ., 2005). However, from the point of view of concentration in coffee, prior detection of the parent compound or metabolites in the body, and physiological effects, there are essentially only three ingredients that are important; caffeine, the diterpene alcohols cafestol and kahweol, and chlorogenic acid and other polyphenols. In specialty coffees consumed outside the home the range is 18–80 mg/cup and decaffeinated coffees averaged 5 mg/cup(McCusker et al ., 2003). Coffee is an important source of caffeine; it provides 71% of the caffeine in the US diet (Frary et al., 2005). The diterpenoid alcohols are the oils in coffee and their concentration depends on the how the coffee is prepared. Filtered coffee has less than 0.1 mg/100 ml, i.e. essentially none, and unfiltered coffee can have between 0.2 and 18 mg/100 ml depending on the method.

High consumption of unfiltered types of coffee, such as French press and boiled coffee has been shown to increase low-density-lipoprotein-cholesterol concentrations. In addition, limiting caffeinated coffee intake during pregnancy seems a prudent choice. However, evidence has been accumulating that frequent consumption of coffee may reduce risk of type 2 diabetes and liver cancer(van Dam ., 2008).

Higher habitual coffee consumption was associated with higher insulin sensitivity (Arnlov et al ., 2004) and a lower risk for type 2 diabetes(van Dam et al ., 2002 ; Rosengren et al ., 2004 ; Salazar-Martinez et al ., 2004 ; Tuomilehto et al ., 2004 and Carlsson et al ., 2004) in diverse populations. In contrast, short-term metabolic studies showed that caffeine intake can acutely lower insulin sensitivity (Keijzers et al ., 2002 and Thong et al ., 2002) and increase glucose concentrations(Mougios et al ., 2003 and Lane et al ., 2004)

Tunnicliffe and Shearer, 2008 found that Coffee consumption may also mediate levels of gut peptides (glucose-dependent insulinotropic polypeptide and glucagon-like peptide-1), hormones intimately involved in the regulation of satiety and insulin secretion. Finally, coffee may have prebiotic-like properties, altering gut flora and ultimately digestion.

It has been reported that Coffee intake might have an antiatherogenic property by increasing of ATP-binding cassette transporter (ABC)G1 and scavenger receptor class B type I (SR-BI) expression and enhancing HDL-mediated cholesterol efflux from the macrophages via its plasma phenolic acids(Uto-Kondo et al ., 2010).

2. Materials and methods

Materials:

Chemicals:

All chemicals including alloxan were fine grade, chemicals purchased from local distributer (Sigma chemical) Cairo.Egypt.

Green, roasted and decaffeinated coffee where purchased from a local market ,Cairo, Egypt and was added to drinking water at a concentration of 5 g% after following preparation : 5g of green , roasted and decaffeinated coffee dissolved in 100 ml boiled water for 10 minutes .

The basal standard diet was prepared in accordance with AIN-93 formulation (Revees et al., 1993).

Composition of diet (g/100g)

Corn starch 62.07;casein 14 ;sucrose 10 ;cellulose 5 ;corn oil 4 ;salt mixture 3.5 ; vitamin mixture 1;L-cystine0.18; choline bitratrate 0.25 and tert.butylhydroxy quinine 0.008.

Animals

In the present study 30 female rats of wistar strain weighing (124.50 ±5.41 g) obtained from Institute of Ophthalmology(Cairo, Egypt) were used in this study . The rats were maintained under standard laboratory conditions in an air conditioned room and housed in stainless steel cages one per cage at temperature 22±3 °C and relative humidity 30-70 %. The animal diet was given ad libitum . Animals were acclimatized for one week prior to experiment.

Thirty rats were divided into 6 groups each of 6 rats.

Group 1(G1): Served as normal control and received standard diet.

Group 2(G2): Diabetic control group. Green, roasted and decaffeinated coffee

Group 3(G3): Diabetic group which received 5 % green coffee in drinking water.

Group 4(G4): Diabetic group which received 5 % roasted coffee in drinking water.

Group 5(G5 Diabetic group which received 5 % decaffeinated coffee in drinking water.

The experiment lasted for 4 weeks.

Assays:

At the end of experimental period, all rats were fasted overnight and then anesthetized by ether and sacrificed. Blood was collected and allowed to clot; serum was separated by centrifugation at 3000 rpm for 15 minutes serum was then transferred into properly labeled sterile vials and stored at -20° C till the performance of Laboratory analysis.

liver , kidney and spleen and heart were excised, rinsed in chilled saline solution and then blotted on filter paper ,weighed separately to calculate the relative weight.

$$\text{The relative weight of organ} = \frac{\text{absolute weight of organ}}{\text{Final body weight of rat}} \times 100$$

Serum was used for determination of seum glucose according to Barham and Trinder , (1972) . Serum insulin was determined according to Vuppugalla et al., (2003). Serum total cholesterol was assayed by the method of Richmond , (1973) , serum triacylglycerol according to Fossati and Prencipe , (1982) , serum HDL by the method of Steele et al ., (1976) while serum LDL-cholesterol by the use of the equation of Friedewald et al ., (1972) .

Statistical analysis:

Statistical analysis: were performed using SPSS for Windows 10.0(SPSS Inc,Chicago.IL.USA). Data were expressed as mean ± S.D. One way analysis of variance (ANOVA) at ($p < 0.05$) was used to compare mean values of continuous variable in cases and control.

3. Results

The present study show a significant difference ($p < 0.05$) in body weight gain and food intake between all treatment groups, with non significant difference in water intake, relative weight of organs including liver , kidney , spleen and heart .these data suggesting that green, roasted and decaffeinated coffee did not influence the relative organ weight and caused the reduction in food intake and gain weight in diabetic rats as compared to normal control group.(Table 1).

Table 2 shows significant elevation ($p < 0.05$) in serum glucose and insulin in diabetic control group as compared to normal control group at the end of experiment. This indicates uncontrolled hyperglycemia in alloxan diabetic rats. While consumption of green, roasted and decaffeinated coffee resulted in a decrease in serum glucose and insulin ($p < 0.05$) with the lowest glucose value observed with green coffee and with decaffeinated coffee for insulin.

There is a significant decrease ($p < 0.05$) in serum calcium and serum phosphorus in groups 3,4 and 5 fed green and roasted coffee respectively indicating an association between caffeine consumption and bone health.(Table 3)

Table (4) shows that alloxan injection produced a significant increase($p < 0.05$) in serum total- cholesterol(TC);triacylglycerol(TAG); LDL-C ; VLDL-C and in LDL\ HDL ratio and TC \ HDL ratio however a significant decrease ($p < 0.05$) in serum HDL-C is observed ; In diabetic rats compared to normal control .

Green, roasted and decaffeinated coffee resulted in a significant decrease ($p < 0.05$) in triacylglycerol(TAG); LDL-C ; VLDL-C and in LDL\ HDL ratio and TC \ HDL ratio .on the other hand a significant increase ($p < 0.05$) in serum HDL-C is observed in green, roasted and decaffeinated coffee groups compared to diabetic rats with the highest value for green coffee .Non significant effect on serum total- cholesterol(TC) reported in this study .

4. Discussion:

In this study the observed decrease in body weight is in agreement with animal studies and the prospective epidemiologic studies on weight loss (Muroyama et al ., 2003 and van Dam et al., 2006) suggest that long-term caffeine and coffee consumption could decrease body weight in humans.

Shimod et al., (2006) showed that consumption of green coffee bean extract (GCBE) for 14 days caused a suppressive effect on weight gain and visceral fat accumulation in mice. GCBE contains 10% caffeine and 27% chlorogenic acid as the principal constituents, and these constituents showed a tendency to suppress body weight gain and visceral fat accumulation. Thus, these constituents are suggested to be partially involved in the suppressive effect of GCBE on body weight gain and visceral fat accumulation. Caffeine is known to be a lipolytic compound. On the other hand, the effect of

Table (1): Effect of green, roasted and decaffeinated coffee on weight gain , food intake and water intake/day and relative weights of different organs (liver , kidney& spleen and heart) In diabetic rats (Mean ± S.D.).

Groups / Parameters	Group (1) Normal control	Group (2) Diabetic	Group (3) Diabetic + Green coffee	Group (4) Diabetic + Roasted coffee	Group (5) Diabetic + Decaffeinated coffee
Weight gain(g)	45.00 ± 4.98	a 35.83 ± 2.99	a 36.00 ± 4.98	a 36.17 ± 3.60	a 36.50 ± 2.81
Food intake(g\day)	18.10 ± 0.85	17.82 ±0.96	a,b 15.75 ± 0.62	a,b 15.67 ± 1.18	a,b 15.05 ± 0.80
Water intake (ml\day)	14.25 ± 1.44	14.67 ± 1.13	14.42 ± 1.06	14.83 ± 1.37	14.42 ± 1.66
Relative weight of liver (g%)	2.62 ± 0.33	2.63 ± 0.19	2.61 ± 0.11	2.43 ± 0.29	2.58 ± 0.25
Relative weight of kidney (g%)	0.57 ± 0.08	0.53 ± 0.11	0.59 ± 0.09	0.57 ± 0.09	0.64 ± 0.05
Relative weight of spleen (g%)	0.16 ± 0.03	0.15 ± 0.04	0.17 ± 0.03	0.16 ± 0.02	0.16 ± 0.03
Relative weight of herat (g%)	0.24 ± 0.03	0.25 ± 0.04	0.25 ± 0.05	0.26 ± 0.03	0.24 ± 0.05

Significant difference (P < 0.05): (a) compared to group 1, (b): to group 2, (c): to group 3, (d): to group 4, (e): to group 5.

Table (2) :): Effect of green, roasted and decaffeinated coffee on serum glucose and insulin In diabetic rats (Mean ± S.D.).

Groups / Parameters	**Group (1) Normal control**	**Group (2) Diabetic**	**Group (3) Diabetic + Green coffee**	**Group (4) Diabetic + Roasted coffee**	**Group (5) Diabetic + Decaffeinated coffee**
Glucose (mg\dl)	96.40 ± 0.42	a 183.03 ± 2.18	b 97.77 ± 1.06	a,b,c,e 101.40 ± 0.72	a,b,d 98.58 ± 1.35
Insulin (µ/ml)	35.67 ± 0.43	a 42.18 ± 1.71	b 36.33 ± 0.64	a,b 37.37 ± 0.84	b 36.13 ± 1.18

Significant difference (P < 0.05): (a) compared to group 1, (b): to group 2, (c): to group 3, (d): to group 4, (e): to group 5.

Table (3) :): Effect of green, roasted and decaffeinated coffee on serum calcium and phosphurus In diabetic rats (Mean ± S.D.).

Groups / Parameters	**Group (1) Normal control**	**Group (2) Diabetic**	**Group (3) Diabetic + Green coffee**	**Group (4) Diabetic + Roasted coffee**	**Group (5) Diabetic + Decaffeinated coffee**
Calcium (mg\dl)	7.75 ± 0.23	7.48 ± 0.29	a,b,e 6.53 ± 0.22	a,b,e 6.57 ± 0.18	c,d 7.43 ± 0.46
phosphorus (mg\dl)	3.53 ± 0.15	3.62 ± 0.22	a,b 2.75 ± 0.15	a,b 2.74 ± 0.13	c,d 3.29 ± 0.47

Significant difference (P < 0.05): (a) compared to group 1, (b): to group 2, (c): to group 3, (d): to group 4, (e): to group5.

Table (4): Effect of green, roasted and decaffeinated coffee on serum total- cholesterol(TC); triacylglycerol(TAG); LDL-C; HDL-C;VLDL-C and on LDL\HDL ratio and TC \HDL ratio In diabetic rats (Mean ± S.D.).

Groups / Parameters	**Group (1) Normal control**	**Group (2) Diabetic**	**Group (3) Diabetic + Green coffee**	**Group (4) Diabetic + Roasted coffee**	**Group (5) Diabetic + Decaffeinated coffee**
(TC ,mg/dl)	91.75 ± 0.73	a 132.65 ± 0.70	a 131.78 ± 1.15	a 132.50 ± 0.75	a 131.83 ± 0.98

(TAG,mg/dl)	79.30 ± 0.89	a 161.95 ± 1.59	a,b 146.75 ± 1.36	a,b 145.77 ± 1.84	a,b 146.53 ± 1.79
(HDL-C ,mg/dl)	28.95 ± 0.69	a 19.37 ± 0.79	a,b 27.13 ± 0.63	a,b,c 26.13 ± 0.89	a,b 26.75 ± 0.58
(LDL-C,mg/dl)	46.94 ± 1.17	a 80.89 ± 1.21	a,b 75.30 ± 1.14	a,b,c 77.21 ± 0.95	a,b 75.78 ± 1.54
(VLDL-C, mg/dl)	15.86 ± 0.17	a 32.39 ± 0.32	a,b 29.35 ± 0.27	a,b 29.15 ± 0.37	a,b 29.31 ± 0.36
LDLl HDL ratio	1.62 ± 0.07	a 4.18 ± 0.22	a,b 2.78 ± 0.082	a,b,c 2.96 ± 0.13	a,b 2.83 ± 0.11
TC l HDL ratio	3.17 ± 0.09	a 6.86 ± 0.28	a,b 4.86 ± 0.097	a,b,c 5.07 ± 0.16	a,b 4.93 ± 0.13

Significant difference ($P < 0.05$): (a) compared to group 1, (b): to group 2, (c): to group 3, (d): to group 4, (e): to group 5.

chlorogenic acid on body weight gain has not yet been established.

Elevated serum glucose and insulin in diabetic control group as compared to normal control group confirm uncontrolled hyperglycemia, whereas green, roasted and decaffeinated coffee decreased serum glucose and insulin ($p < 0.05$) with the lowest glucose value observed with green coffee and with decaffeinated coffee for insulin.

These results are with the line of van Dam., (2008) who found that frequent consumption of coffee may reduce risk of type 2 diabetes and liver cancer.

Several plausible mechanisms for a beneficial effect of coffee on glucose metabolism exist. Coffee has been shown to be a major contributor to the total in vitro antioxidant capacity of the diet (Pulido et al ., 2003) which may be relevant as oxidative stress can contribute to the development of type 2 diabetes. Coffee is the major source of the phenol chlorogenic acid.(Clifford 2000) Intake of chlorogenic acid has been shown to reduce glucose concentrations in rats(Andrade-Cetto and Wiedenfeld ., 2001 and Rodriguez de Sotillo and Hadley 2002 and intake of quinides, degradation products of chlorogenic acids, increased insulin sensitivity in rats.(Shearer et al ., 2003) Chlorogenic acid contributes to the antioxidant effects of coffee, (Clifford 2000) may reduce hepatic glucose output through inhibition of glucose-6-phosphatase, (Arion et al ., 1997)and may improve tissue mineral distribution through its action as a metal chelator. (Rodriguez de Sotillo and Hadley 2002). In addition, chlorogenic acid acts as a competitive inhibitor of glucose absorption in the intestine. (Clifford 2000)Indeed, decaffeinated coffee seemed to delay intestinal absorption of glucose and increased glucagon-like peptide-1 concentrations in an intervention study in humans.(Johnston et al ., 2003) Glucagon-like peptide-1 is well known for its beneficial effects on glucose-induced insulin secretion and insulin action.(Drucker 1998) This effect may explain the observation that higher coffee consumption was associated with lower postload, rather than fasting, glucose concentrations.(Yamaji et al ., 2004 and)

Caffeine ingestion can acutely reduce glucose storage, but beneficial effects of caffeine on lipid oxidation and uncoupling protein-3 expression have also been suggested.(Yoshioka et al ., 2004) In US studies, decaffeinated coffee consumption was inversely associated with risk of type 2 diabetes.(Salazar-Martinez et al., 2004) In addition, in a Japanese study, the inverse association with hyperglycemia was stronger for coffee than for caffeine.(Isogawa et al ., 2003) These observations suggest that coffee components other than caffeine may have beneficial effects on risk of type 2 diabetes. Coffee also contains substantial amounts of magnesium, which has been linked to better insulin sensitivity and insulin secretion.(de Valk 1999) However, adjustment for magnesium intake did not explain the association between coffee consumption and risk of type 2 diabetes(Salazar-Martinez et al., 2004)

As the beneficial effects of coffee consumption exist for both decaffeinated and caffeinated coffee, a component of coffee other than caffeine must be responsible. Tunnicliffe and Shearer2008 reported that, being plant-derived; coffee contains many beneficial compounds found in fruits and vegetables, including antioxidants. In fact, coffee is the largest source of dietary antioxidants in industrialized nations. When green coffee is roasted at high temperatures, Maillard reactions create a number of unique compounds. Roasting causes a portion of the antioxidant, chlorogenic acid, to be transformed into quinides.

Decreased serum insulin in this study is in agreement with The decreased insulin secretion reported by Tianying et al., (2005) is consistent with

the increased insulin sensitivity observed by Arnlov et al.,(2004). In contrast, Arnlov et al., 2004 did not observe a decrease in insulin secretion as assessed by early insulin response under glucose stimulation. However, C-peptide has a longer half-life than insulin and thus may better represent insulin secretion than insulin levels do (Chen et al., 1999).The independent association between decaffeinated coffee and C-peptide indicates active ingredients other than caffeine. Antioxidants may improve insulin sensitivity Bruce et al., 2003 (in type 2 diabetes and decrease insulin levels in rats (Thirunavukkarasu., 2004).

Tianying et al., (2005) concluded caffeinated and decaffeinated coffee consumption might prove to be an effective strategy for reducing insulin resistance, especially in overweight women.

Oka, 2007 demonstrated that the prophylactic effects of coffee on diabetes involve pleiotropy of plural components in accordance to the degree of the roasting. A new concept of nutritional blended coffee may be important to optimize the prophylactic effects of coffee on lowering the risk factors of diabetes and delaying the progress of diabetes complications as well.

On the other hand Contrary to our study Kempf et al., 2010 demonstrated that coffee consumption led to an increase in coffee-derived compounds, mainly serum caffeine, chlorogenic acid, and caffeic acid metabolites. , Whereas no changes were seen for markers of glucose metabolism in an oral-glucose-tolerance test.

On the other hand Robinson et al ., 2004 found evidence of a non significant caffeine-induced increase in insulin secretion in men with type 2 diabetes, and Petrie et al ., 2004 found no increase in such insulin secretion in obese men.

In this study the significant decrease in serum calcium and serum phosphorus in groups 3, 4 and 5 fed green, roasted coffee respectively is in agreement with the finding of Barrett-Connor et al., (1994) who reported that caffeinated coffee intake equivalent to two cups per day is associated with decreased bone density in older women who do not drink milk on a daily basis.

Also are in agreement with those of Rapuri, et al ., (2001) who reported that Intakes of caffeine in amounts >300 mg/d (≈514 g, or 18 oz, brewed coffee) accelerate bone loss at the spine in elderly postmenopausal women. They found a significant negative correlation between caffeine intake and calcium intake and suggested that high caffeine consumption per se has a negative effect on bone mineral density (BMD), which may be further accentuated by low calcium intakes. However, they could not gain insight into the mechanism of how caffeine exerts its negative effect because we found no significant changes in any of the biochemical indexes measured.

The decrease in serum calcium may be due to the effect of coffee consumption which caused an increase in endogenous fecal calcium and urinary calcium excretion.

our results on the other hand disagree with those of Sakamoto et al ., (2001) reported that strongly indicates that coffee does not stimulate bone loss in rats. They clarify the relationship between coffee consumption and bone metabolism using male Wistar rats. assigned to three treatment groups including a control-diet group , a 0.62% coffee-diet group, and a 1.36% coffee-diet group. They indicated no significant differences in body weight change, serum and urinary biochemical markers of bone metabolism, and bone histomorphometry were found between the coffee-diet groups and the control-diet group, except that urinary phosphorus excretion after 140 days of both coffee diets was significantly increased compared with controls ($p < 0.05$). In addition, the coffee diets were not associated with differences in tumor necrosis factor-α and interleukin-6, which have been implicated in the pathogenesis of bone loss together with interleukin-1β.

Green, roasted and decaffeinated coffee resulted in a significant decrease ($p < 0.05$) in triacylglycerol (TAG); LDL-C; VLDL-C and in LDL\ HDL ratio and TC \ HDL ratio. on the other hand a significant increase ($p < 0.05$) in serum HDL-C is observed in green, roasted and decaffeinated coffee groups compared to diabetic rats compared to normal control with the highest value for green coffee ,with non significant effect on serum total- cholesterol(TC) reported in this study .

Our results are in agreement with those of Kempf et al., 2010 who reported that coffee consumption led to an increase in coffee-derived compounds, mainly serum caffeine, chlorogenic acid, and caffeic acid metabolites. Significant changes were also observed for serum concentrations of interleukin-18, 8-isoprostane, and adiponectin (8 compared with 0 cups coffee/d). Serum concentrations of total cholesterol, HDL cholesterol, and apolipoprotein A-I increased significantly by r, whereas the ratios of LDL to HDL cholesterol and of apolipoprotein B to apolipoprotein A-I decreased significantly by 8% and 9%, respectively (8 compared with 0 cups coffee/d), this indicate that coffee consumption appears to have beneficial effects on subclinical inflammation and HDL cholesterol.

In accordance to our study Shimod et al.,(2006) reported that serum and hepatic TG levels were lowered with intravenous administration of chlorogenic acid in Zucker fa/fa rats. However, the TG level in the adipose tissue was not lowered. Therefore, chlorogenic acid is suspected to be effective on hepatic TG, and not adipose TG. Chlorogenic acid is also a dietary polyphenolic compound with antioxidative activity. Thus, it is suggested that caffeine, chlorogenic acid and other polyphenolic compounds in GCBE act synergistically to suppress body weight gain and visceral fat accumulation in mice.

Uto-Kondo et al. (2010) hypothesized that coffee may enhance reverse cholesterol transport (RCT) as the antiatherogenic properties of high-density lipoprotein (HDL). Caffeic acid and ferulic acid, the major phenolic acids of coffee, enhanced cholesterol efflux from THP-1 macrophages mediated by HDL, but not apoA-I. Furthermore, .they concluded that Coffee intake might have an antiatherogenic property by increasing of ATP-binding cassette transporter (ABC)G1 and scavenger receptor class B type I (SR-BI) expression and enhancing HDL-mediated cholesterol efflux from the macrophages via its plasma phenolic acids.

Lee ., 2009 demonstrated that coffee may guard against Alzheimer's disease and other forms of dementia and somehow soften the blow of a heart attack.

Ozercana et al. (2006) found that lipid peroxidation products that increased in the plasma and liver tissue of the CCl4 group decreased by (instant coffee) IC administration. There was an increase in the measured antioxidant parameters, which were total antioxidant capacity (TAOC), sulphydryl (SH) and ceruloplasmin levels. They concluded that IC had a protective role in acute liver injury induced by CCl4, but did not affect steatosis.Lopez-Garcia et al., (2006) reported that there is no evidence that coffee consumption increases the risk of CHD.

Our results on the other hand disagree with the fnding of Rodrigues and Klein. (2006) who found that Caffeine is the most widely consumed psychostimulant drug in the world that mostly is consumed in the form of coffee. They examined the effects of caffeine intake, both alone and via coffee consumption, on key blood markers of CVD risk: lipoproteins (cholesterol, triglycerides), fibrinogen (a biomarker of blood clotting) and C-reactive protein (CRP; a biomarker of inflammation). They indicated a strong relationship between boiled, unfiltered coffee consumption and elevated cholesterol levels. Also disagree with those of Ricketts et al. (1993) who suggest that caffeine consumption is associated with increased serum cholesterol and/or low density lipoprotein cholesterol. They confirmed that when consumption of caffeine reaches 200 mg or more total cholesterol significantly increased in males. Low density lipoprotein cholesterol concentrations were somewhat increased in males who consumed 200 mg or more. In women, triglyceride levels significantly increased when dietary caffeine intake was 200 mg or higher. Dietary caffeine intake may be a factor to consider when evaluating serum lipid levels.

5. Conclusion

The observed improvement in glucose, insulin profile, triacylglycerol and HDL-C confirm the potent biological action of green, roasted and decaffeinated coffee and suggest that chlorogenic acid (a component in coffee) might have an antagonistic effect on glucose transport. Suggesting a novel function of coffee on lowering the risk factors of diabetes and delaying the progress of diabetes complications as well.

Corresponding author
Eman A.Sadeek
Department of Biochemistry & Nutrition -Women's College –Ain –Shams University, Cairo, Egypt
*dr_emansadeek@yahoo.com

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