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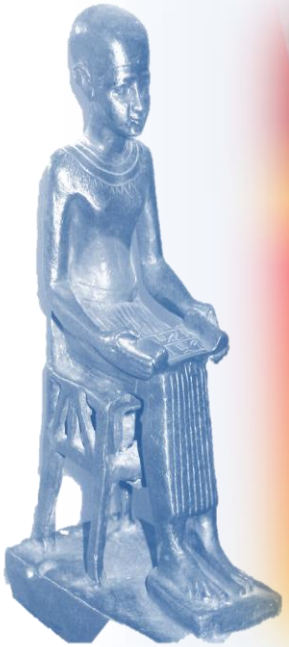

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Original Article

The Possible Protective Effects of Chamomile Extract and/or Green Tea Extract on Obese Male Albino Rats

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Abstract

Background: Obesity and metabolic syndrome can lead to serious medical problems. Both chamomile and green tea are herbal medicines that can provide alternative option for the obesity treatment and its related complications.

Aim of the study: This study aimed to investigate the effects of chamomile extract alone or green tea extract alone or in combination on obese male albino rats.

Materials and Methods: The study was carried out on 50 rats, 10 rats were allocated as normal control group. A total of 40 rats developed obesity by feeding rats high fat diet [HFD] for 10 weeks. Then the obese rats were categorized into 4 groups of equal numbers assigned as obese control group, chamomile treated, green tea treated and combined chamomile and green tea treated groups. Effects of monotherapy and combined therapy of chamomile and green tea on anthropometric measurements, glycemic state, lipid profile, oxidative stress markers and blood pressure were recorded.

Results: HFD led to significant increase in body weight, BMI, fasting glucose and insulin, lipid profile, blood pressure and malondialdehyde associated with significant reduction of HDL-C and catalase. Treatment with Chamomile and green tea led to marked decrease of body weight, BMI, fasting glucose and insulin, lipid profile, blood pressure and malondialdehyde associated with significant increase in HDL-C and catalase. Green tea was superior in body weight management, lipid profile improvement and its antioxidant power while chamomile was more dominant in improvement of glycemic markers and blood pressure.

Conclusion: Chamomile and green tea extracts are of the most useful traditional medicines that showed promising results that aid in treatment of the problems associated with obesity and metabolic disturbances [e.g., insulin resistance, dyslipidemia, hypertension and oxidative stress].

Keywords: Obesity; Matricaria chamomilla; Green Tea; Oxidative stress; High Fat Diet.

INTRODUCTION

Obesity is considered social and medical issue of the twenty first century. It is considered a type of a chronic, non-infectious, metabolic disorder. The accumulation of fat is associated with an oxidative stress and low grade inflammation. This aggravates the obesity-associated disturbances and provokes in obesity-related cardiac, and metabolic complications [1]. In obesity, the metabolic changes include significant increase of blood lipids, mainly cholesterol and low density lipoproteins and decreased high density lipoproteins. In addition, obesity is associated with hypertension and impaired glucose tolerance [2].

Matricaria chamomilla [chamomile] is a widely used medicinal plant. The flowers preparations have many therapeutic effects [e.g., antioxidant, anti-inflammatory, antispasmodic, anti-inflammatory and sedative effects]. These attributed to its content of essential oils [e.g., α-bisabolol, chamazulene, spiroethers, phenolic acids, flavonoids and coumarins] [3]. Polyphenols had anti-oxidant effects [e.g., in metabolic syndrome and obesity]. Thus, chamomile protects against oxidative stress associated with obesity [4]. In addition, chamomile inhibits digestion of carbohydrates and intestinal absorption of glucose [5]. Furthermore, it showed hypo-glycemic and hypo-cholestrolemic effects [6].

Tea is popularly consumed beverage worldwide. It is consumed in the forms of green, black or oolong tea. Green tea had anti-obesity effects, as evidenced by experimental and clinical studies. This was achieved by the reduction of body weight [mainly body fat], by through increased postprandial thermogenesis and oxidation of fat [7], reduce differentiation and proliferation of adipocytes, triglycerides, free fatty acids [FFA], cholesterol, glucose, insulin and leptin [8]. However, the protective effects in obesity is still under-investigated. Thus, this study was designed to investigate the effects of chamomile and green tea as a protective agent on obese male albino rats.

MATERIALS AND METHODS

The present work was completed on 50 adult male albino rats. Their weight 100-150 g per rat [average 130 g]. Rats were purchased from [Nile Pharmaceuticals Company, Egypt]. Then, rats were housed in suitable cages [50 × 30 × 20 cm; 5 rats in each cage] at room temperature with a natural light dark cycle. Rats were fed a balanced diet [bread and green vegetables] with free access to water. They kept in such conditions for two weeks to be acclimatized with environment prior to the start of the experiment. Then, 40 rats were fed a special high fat diet [HFD] regimen for 10 weeks for to induce obesity.

Then rats were divided into five equal groups [each contain 10 rats]. Group I as a control group and include rates fed on basal diet throughout the experiment period [18 weeks]. The second group [Group II] included rats exposed to high fat diet [HFD] for induction of obesity [10 weeks] then were fed on basal diet for 8 weeks after that. Group III [chamomile treated group] that included rats exposed to HFD for induction of obesity [10 weeks] then fed on basal diet with chamomile aqueous extract at a dose of 100 ml/kg for 8 weeks after that. The fourth group [green tea treated group] included rats exposed to HFD for induction of obesity [10 weeks] then fed on basal diet with green tea aqueous extract at a dose of 1 ml /100 gm for 8 weeks after that. The final group [Group V] [combined chamomile and green tea group] and included rats exposed to HFD for induction of obesity [10 weeks] then were fed on basal diet with combined chamomile and green tea aqueous extracts for 8 weeks after that.

Herbs: Chamomile and green tea were obtained from local market. All materials were milled to soft powder by electric grinder.

Diets: [normal or high fat] were purchased from Al-Gomhoria Company, Cairo, Egypt. HFD was preserved at 4°C until used. The experimental and high fat diets [g/kg diet] were prepared according to the formula of **Noeman *et al.*** [9].

The chamomile extract preparation was done as described in **Jabri *et al.*** [10] and chamomile decoction extract [CDE] was orally administered to rats at a dose of 100 ml/ kg for each of them before meals. In addition, the green tea extract was prepared according to **Bakr and Header** [11] and administered to rats at a dose of 1 ml /100 gm for each of them by oral gavage before meals

For all groups the following parameters were measured: 1] aanthropometric measures [Body weight [g], Body weight gain, Naso-anal length [cm] and BMI [gm/ cm^2]; 2] m**easurement of blood pressure, 3] measurement of Food intake. Then, blood samples were collected for the measurement of [b**lood glucose, HbA1c [Glycated haemoglobin], Insulin level and lipid profile]. Then HOMA-IR [insulin resistance was calculated. The lipid profile included total cholesterol [TC], Low density lipoprotein [LDL], high density lipoprotein [HDL], triglyceride3s [TG], very low density lipoprotein. Then, atherogenic index of plasma was determined. Finally, markers of antioxidant indicators were estimated, mainly catalase [CAT] and malondialdehyde [MDA].

Calculation of Body mass index: The diethyl ether was used to keep rats in a state of light Anaesthesia. Then, rats were weighted and the naso-anal length was measured for each rat on a calibrated platform. Measures were ued to calculate the body mass index [BMI = body weight / $length^2$ [gm/cm^2] [12].

Body weight gain [BWG] and food intake [FI]: During the experimental period [18 weeks] the net food intake was daily recorded. Rats were weighted at the beginning of the study, after induction of obesity and at the end of the study then the BWG was calculated. BWG percentage was calculated according to **Champman *et al.*** [13] using the following equation: BWG % = [final body weight – initial body weight] / initial body weight×100.

Measurement of blood pressure: Blood pressure [systolic, diastolic and mean arterial pressure] was measured by the tail-cuff method after induction of obesity and at the end of the study for all groups. This was achieved using a specialized volume pressure recording [VPR] sensor. This measures the changes in blood volume changes over the animal's tail. Rats were restrained in specific holders. Thein,

their tails were artificially heated to maintain normal blood pressure [14]. Systolic and diastolic pressures were recorded and averaged over at least three consecutive readings per rat [15].

Collection of Blood Samples: After calculation of the BMI, the animal was anesthetized with ether [Diethyl ether: [Merk]]. This was performed using the anesthetic box [filled with anesthetic gas]. The rat was placed in this box, while anesthetic gas was maintained by periodical application of liquid ether to a cotton wool on the base of the box. At the stage of surgical anesthesia [Judged by loss of withdrawal reflex], the rat was removed and positioned on a table. Blood was collected [3 ml of blood for each] using heparinized capillary tube from the retro-orbital plexus by insertion of the tube into the medial canthus medial to eye globe [16]. To obtain serum, the blood samples were collected into a dry clean graduated glass centrifuge tube. It was rapidly set to centrifuge at 5000 r.p.m. for 10 minutes. The supernatant serum was sucked out into Eppendorf tubes and stored frozen at -20ºC [17].

The determination of blood glucose levels and glycated hemoglobin was determined according to method described by **Nathan *et al.*** [18], while serum insulin was measured by direct immune-enzymatic method using bio-diagnostic laboratory kit[19]. The HOMA-IR value was calculated using the formula: [HOMA-IR = fasting glucose [mg/dl] × fasting insulin [µ IU/ml] / 405] [20].

The total cholesterol level was assayed by quantitative enzymatic colorimetric method using bio-diagnostic laboratory kit as described by **Allain *et al.*** [21]. Serum TG level was assayed by quantitative enzymatic colorimetric method using bio-diagnostic kit as described by **Fossati and Prencipe**[22]. HDL-C level was assayed by quantitative enzymatic colorimetric determination using bio-diagnostic laboratory kit [23].

An estimation of LDL-Cholesterol is calculated from direct measurements of total cholesterol, triglycerides and HDL-Cholesterol, LDL-Cholesterol can be calculated using the **Friedewald** formula as following: VLDL-C = Triglycerides / 5. Or LDL-C = Total cholesterol – VLDL-C – HDL-C [24]. Atherogenic index of plasma [AIP] was measured as log10 [TGs / HDL-C] [25].

Determination of malondialdehyde [MDA]: Erdelmeier *et al.* [26] used the enzymatic method for a spectro-photometric assay of malondialdehyde using Bioxytech MDA-586™ kit.

Determination of catalase: Sinha [27] used quantitative colorimetric determination of catalase activity by using the Biodiagnostic kit.

Statistical Analysis of Data: Data management and statistical analysis were performed by the Statistical Package for Social Sciences [SPSS] vs. 21. Numerical data were summarized as means and standard deviations. Kolmogorov-Smirnov test of normality was done to assess normality of continues variables before starting the analysis. Differences among the five groups were analyzed with one-way analysis of variance [ANOVA] and post hoc Tukey test. Repeated measure ANOVA was done to assess the effect of time [before and after treatment]. P-values [P>0.05] were considered insignificant but [P<0.05] were considered significant

RESULTS

The results of the current work showed that, obese group [Group II] had significantly increase of weight, length, BMI, weight gain after induction of obesity and weight gain at the end, when compared to control groups. The administration of chamomile and green tea [individually] was associated with significant reduction of anthropometric measures when compared to obese group. However, the values still high than the values in the control group. The co-administration of chamomile and green tea was associated with better effect. However, it did not reach the basal values [as in the control group] [Table 1].

Obesity was associated with significant increase of fasting blood glucose, glycated hemoglobin, fasting insulin and insulin resistance, when compared to control group. The administration of chamomile and green tea [co-administration or individual administration] was associated with significant reduction of FBG, HbA1c, insulin and insulin resistance than obese group. However, values still higher than the control group [Table 2].

Induced obesity was associated with significant increase of TC, TG, LDL, vLDL, atherogenic index and significant reduction of HDL, when compared to control group. The individual or co-administration of chamomile or green tea was associated with significant improvement of lipid profile. However, it did not reach values as in the control group [Table 3].

Induced obesity was associated with oxidative stress, which was ameliorated by individual or co-administration of green tea or chamomile. In this study, this effect was evidenced by significant increase of MDA, and significant decrease of catalase by the induced obesity. Then, the values reversed toward healthy controls by the administration of green tea and/or chamomile [Table 4].

Blood pressure [systolic, diastolic and mean arterial pressure] was significantly increased with obesity. This effects did not be reversed after induction. However, at the end of experiment, there was significant reduction of blood pressure with green tea and/or chamomile administration. However, it did not return to values as in control group [Table 5].

Food intake before treatment was significantly increase in obese groups with or without green tea and/or chamomile than the control group before treatment. However, the food intake did not differ between obese groups. After treatment the food intake increased in obese groups than the control group. However, green tea was associated with significant reduction of food intake than chamomile group [Table 6].

Table [1]: Weight, length, BMI and BWG of the studied groups at the end of the experiment.

	Length [cm]	Weight [gm]	BMI [gm/cm²]	Weight gain after induction [g, %]	Weight gain at the end [g, %]
Group 1[n=10]	20.7±0.24	268.5±13.44	0.62±0.02	82.2±4.2 [43%]	158.8±1.8 [59%]
Group 2 [n=10]	22.2±0.26#	393±10.6#	0.79±0.007#	212.7±10.5 [63%]#	268.4±10.2 [68%]#
Group 3 [n=10]	21±0.28#*	339±13.1#*	0.75±0.01#*	215±6.2 [64%]#	220.9±11.2 [65%]#*
Group 4 [n=10]	21±0.16#*	326.5±5.8#*	0.73±0.009#*	210.6±5.3 [63%]#	205±11.8 [62%]#*$
Group 5 [n=10]	20.9±0.24*	317.5±7.8#*$	0.73±0.01#*$	208.2±7.5 [62%]#	190.8±4.9 [59%]#*$@

Data are presented as means ± SD [n=10] and were tested by one-way ANOVA test followed by post hoc Tukey test and significant change was reported at p< 0.05. SD: standard deviation. #: Significance versus group 1[Control group]; *: Significance versus group 2 [Obese group]. $: Significance versus group 3[Chamomile treated group]. @: Significance versus group 4 [Green tea treated group].

Table [2]: Blood glucose, HbA1c, insulin level and HOMA-IR of the studied groups during the experiment.

Animal groups	FBG [mg/dl]	HbA1c	Insulin [µIU/ml]	Insulin resistance [HOMA-IR]
Group 1[n=10]	75.9±11.8	4.43±0.57	3.09±0.09	0.57±0.09
Group 2 [n=10]	163.8±3.8#	7.14±0.41#	4.48±0.19#	1.79±0.07#
Group 3 [n=10]	140.2±3.4#*	6.28±0.31#*	4.01±0.19#*	1.37±0.07#*
Group 4 [n=10]	134.8±4.6#*	6.52±0.24#	3.74±0.19#*$	1.24±0.06#*$
Group 5 [n=10]	129.1±2.7#*$	5.95±0.28#*$	3.47±0.22#*$@	1.1±0.07#*$@

Data are presented as means ± SD [n=10] and were tested by one-way ANOVA test followed by post hoc Tukey test and significant change was reported at p< 0.05. SD: standard deviation. #: Significance versus group 1[Control group]. *: Significance versus group 2 [Obese group]. $: Significance versus group 3[Chamomile treated group]. @: Significance versus group 4 [Green tea treated group].

Table [3]: Lipid profile of the studied groups during the experiment.

	Total cholesterol [mg/dl]	Triglycerides [mg/dl]	HDL [mg/dl]	LDL [mg/dl]	VLDL [mg/dl]	Atherogenic index [AIP]
Group 1	91±2.3	64.4±5.3	49±2.7	36.5±18.9	12.9±1.1	0.1±0.05
Group 2	136.7±6#	144.7±3.7#	33.2±2.2#	71.1±10.4#	28.9±0.7#	0.63±0.03#
Group 3	111.7±6.9#*	96.8±5.4#*	40.2±1.4#*	52.2±6.3#*	19.4±1.1#*	0.38±0.02#*
Group 4	99.5±2.5#*$	112.6±4.2#*$	41±0.8#*	33.9±2.8*$	24.3±0.8#*$	0.47±0.02#*$
Group 5	95.5±1.7*$	82.3±4#*$@	41.7±0.9#*	43±2.4*	16.5±0.8#*$@	0.29±0.02#*$@

Data are presented as means ± SD [n=10] and were tested by one-way ANOVA test followed by post hoc Tukey test and significant change was reported at p< 0.05. SD: standard deviation. #: Significance versus group 1[Control group]. *: Significance versus group 2 [Obese group]. $: Significance versus group 3[Chamomile treated group]. @: Significance versus group 4 [Green tea treated group].

Table [4]: MDA and catalase of the studied groups at the end of the experiment.

Animal groups	MDA [nmol/ml]	Catalase [IU/L]
Group 1	7.3±0.3	53.4±2.9
Group 2	13.6±0.45#	41.5±2.6#
Group 3	9.9±0.31#*	47.7±1.8#*
Group 4	8.9±0.3#*$	47±1.8#*
Group 5	8.4±0.32#*$@	49.7±2.1#*

Data are presented as means ± SD [n=10] and were tested by one-way ANOVA test followed by post hoc Tukey test and significant change was reported at p< 0.05. SD: standard deviation. P: probability. #: Significance versus group 1[Control group]. *: Significance versus group 2 [Obese group]. $: Significance versus group 3[Chamomile treated group]. @: Significance versus group 4 [Green tea treated group].

Table 5: Blood pressure changes of the studied groups during the experiment.

Animal groups	SBP after induction	DBP after induction	MAP After induction	SBP at end	DBP at end	MAP at end
Group 1	113.4±8.9	79.1±4.6	89.9±5.4	117.2±7.9	81.6±5.1	92.7±5.6
Group 2	145.7±5.7#	112.8±4.9#	123.9±4.9#	150.3±4.2#	115±4.1#	126.8±3.8#
Group 3	145.2±5.3#	112.3±5.6#	123.3±6#	130.4±3.1#*	95±2.7#*	106.9±2.7#*
Group 4	145.1±2.2#	112.3±4#	123±3.3#	135.2±3.6#*	102.6±2.9#*$	113.5±2.7#*$
Group 5	145.2±3.7#	112.6±5.5#	123.5±4.9#	128.3±2.8#*@	93.6±2.8#*@	105.3±2.7#*@

Data are presented as means ± SD [n=10] and were tested by one-way ANOVA test followed by post hoc Tukey test and significant change was reported at p< 0.05. SD: standard deviation. #: Significance versus group 1[Control group]. *: Significance versus group 2 [Obese group]. $: Significance versus group 3[Chamomile treated group]. @: Significance versus group 4 [Green tea treated group]

Table [6]: Food intake of the studied groups during the experiment.

Animal groups	Food intake before treatment [gm/day]	Food intake after treatment [gm/day]
Group 1	15.9±0.36	16.2±0.38
Group 2	19.6±0.49#	19.4±0.28#
Group 3	19.5±0.34#	20.6±1#
Group 4	19.5±0.38#	16.9±1.64*$
Group 5	19.5±0.26#	17.8±0.75$

Data are presented as means ± SD [n=10] and were tested by one-way ANOVA test followed by post hoc Tukey test and significant change was reported at p< 0.05. SD: standard deviation. #: Significance versus group 1[Control group]. *: Significance versus group 2 [Obese group]. $: Significance versus group 3[Chamomile treated group].

DISCUSSION

In the current work, the protective effects of chamomile and green tea were investigated on induced-obesity in male rats. Results revealed that HFD feeding for 10 weeks lead to a significant increase in body weight, body weight gain, length, BMI, and food intake. The effects which partially reversed by green tea and/or chamomile. These results are consistent with previous literature. For example, **Subramaniam *et al.*** [28] reported that HFD led to positive fat balance and fat accumulation in adipose tissue, leading to increased body weight, body weight gain [BWG], and body mass index [BMI]. **Lacerda Leocádio *et al.*** [29] **and De Moura *et al.*** [30] further supported these findings, showing that HFD feeding for 4 weeks led to increased food intake, body weight gains and abdominal fat accumulation. **Ben Salem *et al.*** [31] also demonstrated that body weight was significantly increased in HFD groups than the control groups, which was associated with increased food intake. Eating HFD promotes the development of positive energy balance, with subsequent increase of the deposited visceral fat and truncal obesity. Moreover, **Elhassaneen *et al.*** [32] reported that feeding rats on HFD for 4 weeks was associated with a significant increase in BWG and food intake than the normal group.

Our results are also consistent with **El-Kholie and Abd El-Hamed** [33] who demonstrated that chamomile extract at different doses [2.5% and 5%] significantly decreased body weight gain when compared to the HFD group. Similarly, **Barcin-Güzeldere *et al.*** [34] reported significant weight loss in the chamomile group compared to the HFD group. This may be attributed to chamomile contents of luteolin and other flavonoids which induce adipocyte browning and increase thermogenesis [35].

Regarding green tea effects, our results are comparable to that of **Bakr and Header** [11] who demonstrated that green tea extract at doses of 10% and 20% led to a reduction of body weight gain and food intake. Also, **Ohishi *et al.*** [36] reported significant weight reduction in green tea-treated groups compared to obese groups in various animal models. Also, **Bagheri *et al.*** [37] observed significant reduction in body weight, BMI, waist-to-hip ratio, and body fat percentage [BFP] in green tea-treated overweight middle-aged men.

Green tea promotes changes in eating habits by reducing lipid and protein absorption through the inhibition of digestive enzymes, leading to increased fecal weight and lipid content [38]. Green tea catechins suppress the gene expression and formation of proteins included in adipogenesis and lipogenesis while stimulating those involved in fatty acid mobilization [39]. The primary effect of green tea on body composition is mainly attributed to catechins, which inhibit adipocyte differentiation and proliferation, reduce fat absorption, inhibit Catechol-O-methyl-transferase [COMT], and increase fat utilization, energy expenditure, and thermogenesis [37].

As regards the lipid profile, our results showed that rats fed by a HFD for 10 weeks resulted in a significant increase in TC, TGs, VLDL, AIP, and LDL-C, along with a significant decrease in HDL-C levels. These findings are consistent with **Ramalho *et al.*** [40] who reported significantly higher serum levels of TC, TG, and LDL, and lower HDL levels in HFD-fed rats compared to their control. Our results are further supported by **Yang *et al.*** [41] who reported that feeding rats on HFD for 4 weeks led to significant increase of TC, TGs, and LDL-C, and marked decreased of HDL-C levels, potentially due to changes in gut microbiota composition with relative abundance of pro-inflammatory and pathogenic bacteria.

Our result showed that administration of chamomile extract in obese rats for 8 weeks led to a significant decrease in TC, TGs, VLDL, LDL-C, and AIP, and a significant increase in HDL-C compared to the HFD group. These findings are in line with **Rafraf *et al.*** [42] who reported that chamomile tea significantly decreased TC and TG levels in patients with type 2 diabetes mellitus. **El-Kholie and Abd El-Hamed** [33] also reported significantly lower levels of TC, TGs, LDL-C, VLDL, and AIP, and higher HDL-C levels in chamomile-treated rats compared to obese control. The anti-hyperlipidemic activity of chamomile may be attributed to its high concentration of essential oils and chlorogenic acid, which modulate peroxisome proliferator-activated receptors [PPARs] [3].

Our results showed that administration of green tea extract in obese rats for 8 weeks resulted in significantly lower levels of TC, TGs, LDL-C, VLDL, and atherogenic index associated with significantly higher levels of HDL-C when compared to control obese rats and chamomile groups. These results are consistent with **Bakr and Header** [11] who observed that green tea extract at 10% and 20% doses significantly decreased TC, LDL, VLDL, and TG levels, and increased HDL levels in obese rats.

Catechins decrease values of plasma TC, cholesterol ester, and HDL-c levels, and lower the atherogenic index, indicating a hypo-cholesterolemic effect. Catechins also prevent invasion of the vascular smooth muscles by inhibiting melatonin receptor 1-matrix metallopeptidase [MT1-MMP]. This ability might contribute to the protective effect of green tea against atherosclerosis and cancer. The hypolipidemic effects of green tea are explained by high flavonoid content, especially catechins, with its potent antioxidant properties [37]. Green tea improves lipid profiles by decreasing the micellar solubility and intestinal absorption of cholesterol, and lowering concentration of hepatic cholesterol [43].

In contrast to our results, **Huang *et al.*** [44] found no significant differences in BMI, fasting blood sugar, TC, TG, HDL, adiponectin, and ghrelin levels between green tea extract and placebo groups after 6 weeks of treatment, although leptin levels increased and LDL-C levels decreased. The short study duration might not have allowed sufficient time for significant changes.

As regards glycemic status, the results of the present study showed that feeding rats with HFD for 10 weeks led to significantly higher levels of serum glucose, insulin, HOMA-IR, and HbA1c. These results are consistent with **De Moura *et al.*** [30] who found significantly higher blood glucose and insulin concentrations, as well as HOMA index values, in groups consuming an obesogenic diet compared to controls. Similarly, **Andonova *et al.*** [45] reported a significant increase in insulin, glucose, and HOMA-IR in rats administered HFD for 4 weeks. Glycemic parameters changes are due to inability of insulin

metabolism to adapt to the damage induced by the chronic excess of calories pf the HFD, which led to gradual and progressive deterioration of the insulin activity, leading to IR and development of T2DM [46]. In addition, the HFD stimulates the accumulation of ectopic fat in the pancreas. This markedly exerts a stresses of beta cells and disrupting the production of insulin. This fat accumulation outside the adipose tissue, [e.g., in the liver and other organs] can lead to IR and hyperglycaemia, since saturated fatty acids interfere with the insulin receptors activity and glucose transporters [35].

Our results showed that administration of chamomile extract in obese rats for 8 weeks led to significant decreases in blood glucose, serum insulin, HOMA-IR, and HbA1c compared to the obese group. These findings align with **Rafraf *et al.*** [42] who reported that after 2 weeks CDE-treated mice exhibit reduced fasting blood glucose, reduced fasting plasma insulin levels and decreased HFD-induced insulin resistance. CDE-treated mice revealed increased glucose clearance of a standard glucose load [2 g/kg]. **Zemestani *et al.*** [6] reported that chamomile consumption could exert favorable effects on serum blood glucose, serum insulin, HOMA-IR and HbA1C in diabetic patients. Similarly, **Jabri *et al.*** [47] demonstrated that CDE had a significant protective effect against HFD-induced obesity and oxidative stress because of its antioxidant properties, by inhibiting intestinal glucose absorption via the SGLT1 modulation or negatively regulating the intracellular mediators [e.g., calcium, H2O2 and free iron]. Indeed, the SGLT1 is the major intestinal glucose transporter so, its modulation leads to adjustment of sugars and control of food intake. Moreover, **Barcin-Güzeldere *et al.*** [34] recorded the lowest blood glucose levels in chamomile-treated obese rats. Chamomile can ameliorate the metabolic overload of high glucose or fructose diets by different mechanisms. For example, it can attenuate their absorption by inhibiting intestinal carbohydrate-digestive enzymes and hexose transporters, inhibition of key enzymes of gluconeogenesis and glycogenolysis as well as induction of glucose use. Moreover, the molecular antidiabetic mechanism of Chamomile extracts interplays with liver PPARs. Activation of PPARγ improves the sensitivity of insulin, while activation of PPARα reduces plasma cholesterols and IR [3].

Our results showed that green tea administration for obese rats for 8 weeks led to significant decreases in blood sugar, serum insulin, and HOMA-IR than in the control group, with non-significant reduction of HbA1c. These results agree with **Sundaram *et al.*** [48] who found that daily oral green tea extract for 30 days is associated with a significant decrease of plasma glucose in diabetic rats.

Beneficial effects of green tea may be attributed mainly for catechins. Catechins had effects on glucose control by different mechanisms. First, catechins decrease the intestinal absorption of carbohydrate by inhibiting intestinal sucrose, alpha-amylase, and alpha-glucosidase. Second, catechins inhibit the hepatic gluconeogenesis by regulation of the gluconeogenic genes expression and phosphorylation of protein-tyrosine in the mouse liver. Third, catechins could improve insulin sensitivity and glucose metabolism and prevent the T2DM development. Green tea can significantly reduce blood sugar levels by increasing hepatic glycogens through reactivation of glycogen synthase system and reducing the activity of liver glucose-6-phosphatase, which is primarily responsible for releasing glucose molecules in the blood. Its ability to decrease blood glucose was comparable to that of the oral hypoglycemic drug [metformin] [49]. Green tea polyphenols also increase insulin sensitivity by enhancing glucose absorption by adipocytes improves their insulin biding ability and improving insulin secretion. Green tea can also increase the circulating levels of adiponectin. Adiponectin exerts protective anti-inflammatory effects and can positively moderate the endocrine system through enhancement of insulin sensitivity in obese rats as well as in humans. In addition, adiponectin enhances the catabolism of fatty acids and actively controls blood sugar. It also induces oxidation of fatty acids in skeletal muscles, and subsequently decreases the accumulation of triglycerides [50].

In contrast to our results **Stote *et al.*** [51] who declared that the short-term consumption of flavanols [for example from cocoa or green tea] do not improve the metabolism of glucose in obese adults at risk for IR. However, the consumption of cocoa and green tea improved certain oxidative stress indicators, inflammation biomarkers and hemostasis. This intervention of 5 days was relatively short; as different results are possible with longer exposure.

Combination of chamomile and green tea extracts for 8 weeks in obese rats led to the most significant decrease of blood glucose, serum insulin level, HOMA-IR and HbA1c when compared to obese group, chamomile treated group and green tea treated group. **Villa-Rodriguez *et al.*** [5] showed that chamomile and green teas are possible tools to control the absorption and metabolism of sugars with marked effects against the high sugar bolus stress. Chamomile and green teas inhibit the digestive enzymes [α-amylase and maltase] related to the release of intestinal sugar, and effectively inhibit the fructose or glucose transport through GLUT2 inhibition, and also inhibited GLUT5 thus reducing the glucose absorption and utilization.

As regards lipid peroxidation and oxidative stress markers, the result of the present study showed that feeding rats with HFD for 10 weeks led to a significant increase in MDA levels and decrease in serum catalase levels. The link between HFD and oxidative stress has long been recognized. Long-term feeding of a high-saturated fat diet induces OS by significantly attenuating the hepatic enzyme antioxidant system and increasing lipid peroxidation products in the liver and plasma. OS plays a crucial role in the pathogenesis of metabolic disorders, leading to insulin resistance, type-2 DM and obesity [52]. MDA is a representative product of lipid peroxidation. The over-generation of ROS results in the consumption of antioxidant substances, leading to a decrease in glutathione [GSH] content, CAT and superoxide dismutase [SOD] activity [53]. Our results are consistent with **Noeman *et al.*** [9] who found that HFD increased MDA levels and decreased catalase levels. Also, **Ben Salem *et al.*** [31] reported that high-fat diets generate an oxidative stress, indicated by a significant decrease in SOD, GSH, and glutathione peroxidase [GPx] activities and a statistically significant increase in MDA levels in heart tissue. The high blood glucose led to increased glucose autoxidation and non-enzymatic glycosylation of proteins and nucleic acids reactive carbonyl species. These processes lead to aggravation of damage exerted by oxidative stress. Increased levels of protein, DNA oxidation, and lipid peroxidation products are observed in the blood and fatty tissues of obese subjects and mice [3].

Our results showed that administration of chamomile extract in obese rats for 8 weeks led to a significant reduction in the serum MDA levels and a significant increase in serum catalase levels compared to obese control rats. Our results are in line with **Sebai *et al.*** [54] who revealed that chamomile tea significantly reduced the castor oil-induced increase of hydrogen peroxide in a dose dependent manner; this effect may be attributed to the phenolic compounds in chamomile. These molecules [e.g., quercetin and cafeic acid], are the main sources of antioxidant ability of chamomile by scavenging free radicals as hydroxyl radical [OH]; the major cause of lipid peroxidation. Also, our results agree with **Zemestani *et al.*** [6] who noticed decreased serum MDA level and increased total antioxidant capacity, SOD, glutathione peroxidase, and catalase activities in a significant manner in patients with T2DM. Similarly, **Jabri *et al.*** [47] found that feeding rats with HFD induced an increase in IL-1β and IL-6, oxidative stress marked by increased peroxidation of lipids, inhibited activity of antioxidant enzymes [e.g., SOD, CAT and GPx], and depletion of non-enzymatic antioxidants in the brain and other tissues. These changes were ameliorated by administration of chamomile extract. The antioxidant effects of chamomile are due to phenolic compounds and essential oils, which moderate signaling pathways responsible for

energy metabolism, inflammation, stress responses, and adipogenesis. Chamomile improves lipid oxidation by decreasing LDL-C and TG and increasing HDL-C through its phytoestrogen compounds [3]. Chamomile can ameliorate oxidative stress and improve antioxidant defense system by its antioxidants agents such as apigenin, luteolin, quercetin, volatile oils, polysaccharides and total flavonoids of Chamomile [55].

Our results showed that administration of green tea extract in obese rats for 8 weeks led to a significant decrease in MDA levels and a significant increase in catalase activity when compared to obese group. These results are consistent with **Bogdanski *et al.*** [56] who reported a significant increase in the enzymes involved in oxidation/reduction or detoxification reactions, including catalase and glutathione after EGCG administration in animals treated with HFD. The antioxidant action of catechins is due to the direct inactivation of reactive oxygen and nitrogen species, chelation of transition metals, regeneration of antioxidants like β-carotene or α-tocopherol, and inhibition of pro-oxidant enzymes. Green tea catechins increase plasma antioxidant capacity via reducing lipid peroxidation product markers, oxidative stress markers in erythrocytes, and the level of oxidative DNA damage markers [57].

The combination of chamomile and green tea extracts for 8 weeks in obese rats led to a significant decrease in MDA levels compared to obese, chamomile, and green tea groups, as well as a significant increase in catalase levels compared to obese and chamomile groups. **Chatterjee *et al.*** [58] demonstrated better anti-inflammatory and antioxidant activity of zinc oxide nanoparticles mediated by chamomile and green tea combination, suggesting potential therapeutic applications.

As regards blood pressure, results of the present study showed that feeding rats on HFD for 10 weeks led to significant increase of SBP, DBP and MAP when compared to control group. These findings are consistent with **Duansak *et al.*** [59] who reported marked increased SBP and DBP in HFD mice compared to control mice on a normal diet. Obesity is often accompanied by a state of low-grade chronic inflammation [vascular and systemic] that can cause endothelial dysfunction. Fat accumulation led to activation of NF-κB, which established a pro-inflammatory and pro-thrombotic state promoting the alteration of vascular function predisposing to the development of hypertension [59].

In contrast to our results **Marques *et al.*** [60] reported that after 15 weeks, HFD was not sufficient to elevate SBP significantly in Sprague Dawley rats or Wister rats. The low salt content of the HFD used in this study [0.3%] in contrast with those used by other authors [0.8, 2 and 4%] may explain its lack of efficiency in increasing SBP of both rats.

Our results showed that administration of chamomile extract in obese rats for 8 weeks led to significant decrease of SBP, DBP and MAP when compared to obese group. These results are compatible with **Pourshaikhian *et al.*** [61] who stated that chamomile significantly lowers SBP, DBP and heart rate [HR] in acute coronary syndrome patients compared to those of the placebo group. Chamomile tea contains anthocyanin compounds and other antioxidants that may help blood vessels to resist the damage that can lead to their narrowing [62]. Our results showed that administration of green tea extract in obese rats for 8 weeks led to significant decrease in SBP, DBP and MAP when compared to obese group. **Szulińska *et al.*** [57] recorded that supplementation of green tea led to statistically significant lower values of SBP and DBP in NaCl induced hypertension in animals associated with beneficial effects on inflammatory and antioxidant status. In addition, our results also coincide with **Meena and Jayakumar** [63] who reported that green tea significantly reduced systolic and diastolic BP in hypertensive patients with longer tea intake duration [≥3 months] resulting in greater reductions.

In light of our results, it could be concluded that chamomile and green tea extracts are of the most useful traditional medicines that showed promising results for treatment of the problems associated with obesity and metabolic syndrome such as insulin resistance by decreasing glucose absorption and enhancing insulin signaling pathway. They also improve body weight, body weight gains and dyslipidemia associated with obesity by decreasing lipid absorption and increasing fecal excretion of fat. Oxidative stress is an inflammatory state associated with obesity and improved by chamomile and green tea extracts as they prevented the production of free radicals, neutralized and scavenged free radicals produced in the body. They also improved lipid metabolism leading to decrease of ROS production. All these effects of chamomile and green tea could decrease the rate of obesity mortality and morbidity.

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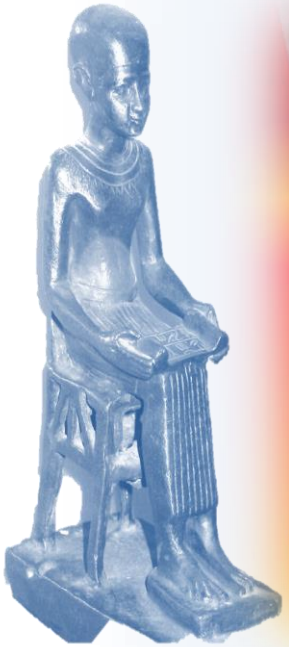

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