

# EFFECT OF GREEN TEA EXTRACT ON FOOD CONSUMPTION AND BODY WEIGHT IN EXPERIMENTALLY-INDUCED MYOCARDIAL INFARCTION IN ADULT MALE ALBINO RAT

By

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## ABSTRACT

**Background:** There is an increasing realization that herbs can influence the course of heart disease and its treatment. Green tea is an important medicinal plant having strong anti-oxidant properties. **Objective:** Evaluation of the effect of green tea extract on food consumption and body weight of isoprenaline (ISO)-induced myocardial infarction in adult male albino rat. **Material and Methods:** Seventy adult male albino rats of local strain were chosen as an animal model for this study weighed 110 -130 g (average weight was 120 g). Myocardial infarction was induced in experimental rats by injecting isoprenaline. Food consumption was measured daily. Body weight was measured at the beginning and at the end of experiment. Heart weight/body weight ratio was calculated at the end of the experiment (30 days). **Results:** During the experiment, changes in body weight and food consumption in different groups were more or less concomitant together. Increase in body weight was associated with increase in food consumption and vice versa. ISO showed an increase of food consumption which was accompanied by an increase of body weight. GTE showed reduction of food consumption which was accompanied by loss of body weight, while metoprolol produced an increase of food consumption accompanied by an increase of body weight. GTE and metoprolol in combination produced the same effect that produced by green tea extract. **Conclusion:** Green tea extract is effective in decreasing food consumption and body weight.

**Key words:** Green tea, myocardial infarction, food consumption, body weight, isoprenaline

## INTRODUCTION

Several varieties of green tea exist, which differ substantially due to growing conditions, horticulture, production processing, and time of harvest. Catechins are the main bioactive constituents of green tea leaves, and account for 25% to 35% of their dry weight. The main catechin group consists of eight polyphenolic flavonoid-type compounds, namely, catechin (C), Epicatechin (EC), Gallocatechin (GC), Epigallocatechin (EGC), Catechin gallate

(CG), Epicatechin gallate (EG), Gallocatechin gallate (GCG) and Epigallocatechin gallate (EGCG). Epigallocatechin gallate is the most abundant of the tea catechins and thought to be responsible for the majority of the biological activity of green tea extracts (Sutherland et al., 2013). Other bioactive molecules are amino acids like theanine, proteins, caffeine, vitamin C, carbohydrates, polysaccharides and lipids. In the cells of a tea leaf, catechins reside in the cell sap, while oxidative enzymes are located in the cell wall

(Chacko *et al.*, 2010). Epigallocatechin-3-gallate (EGCG) purified from green tea decreases diet-induced obesity by decreasing energy absorption and increasing fat oxidation (Klaus *et al.*, 2005). The increased and prolonged sympathetic stimulation of thermogenesis by the interaction between polyphenols and caffeine could be of value in assessing the management of obesity (Dulloo *et al.*, 2000).

The consumption of green tea and green tea extracts may help reduce body weight, mainly body fat, by increasing postprandial thermogenesis and fat oxidation (Boschmann and Thielecke, 2007).

The present work aimed to study the changes of food consumption and body weight by green tea extract on isoprenaline-induced myocardial infarction in adult male albino rat.

## MATERIAL AND METHODS

**Animals:** Seventy adult male albino rats of local strain were chosen as an animal model for this study, and were kept in suitable cages (20x32x20 cm for every four rats) at room temperature, with the natural light-dark cycle in the Physiology Department, Al-Azhar Faculty of Medicine. They weighed 110 -130 g (average weight was 120 g). They were fed on the standard food in addition to bread and green vegetables with free water supply. They were kept for 10 days for the adaptation to the new environments before the start of the experiment. The animals were divided into seven equal groups as follows:

(I)-Normal control group (II)-Intra-peritoneal saline-treated group received

0.5 ml normal saline / 100 g body weight intraperitoneally for two consecutive days (29th and 30th). (III)-Green tea extract-treated group received daily 10 mg / 100 g body weight orally for 30 days. (IV)-Isoprenaline-treated (MI) group received 11 mg / 100 g body weight intraperitoneally for two consecutive days (29th and 30th). (V)- Green tea extract + Isoprenaline-treated group received green tea extract 10 mg / 100 g body weight /day orally for 30 days in addition to i.p. injection of isoprenaline in a dose of 11 mg / 100 g body weight intraperitoneally for two consecutive days (29th and 30th). (VI)- Metoprolol + Isoprenaline-treated group (standard group) received Metoprolol (1 mg / 100 gm body weight /day orally) for 30 days in addition to ISO 11 mg /100 gm i.p. on the 29th and 30th days. (VII)- Green tea extract + Metoprolol + Isoprenaline-treated group treated group received green tea extract (10 mg / 100g body weight /day orally for 30 days), Metoprolol (1 mg / 100 g body weight /day orally for 30 days), and i.p. injection of isoprenaline in a dose of 11mg / 100 gm body weight for two consecutive days (29th and 30th ).

**Methods:** Myocardial infarction was induced in experimental rats by injecting isoprenaline (ISO) 11 mg (dissolved in physiological saline) / 100 g body weight i.p. for 2 days (29th and 30th).

Green tea extract (GTE) tablets (Arab Co. for Pharmaceuticals & Medicinal Plants, Mepaco-Pharma, Egypt): Each tablet contains 200 mg GTE. The stock solution was prepared by dissolving 6 tablets in 60 ml distilled water with a concentration of 20 mg GTE per 1 ml distilled water. From that stock solution,

appropriate volume of GTE was given to rat in a dose of 100 mg / kg body weight /day orally by orogastric tube for 30 days. Stock still stable at room temperature for seven days (Ojo et al., 2006).

Food consumption was measured daily. Body weight was measured at the beginning and at the end of experiment.

Heart weight/body weight ratio: The rat was weighed. Removal of the heart was performed by dissecting the aortic root immediately above the aortic valve and the superior vena cava above the atrium. Adjacent mediastinal fat pads were removed from the excised heart carefully. The dry heart was weighed and recorded, then heart weight/ body weight ratio was calculated (Neha and Lubna, 2014).

**Statistical analysis:** The computer program SPSS version "17" was used for one way ANOVA (Analysis Of Variance) test, calculation of the descriptive statistics in studied groups (means  $\pm$  standard deviations), detection of any significant difference between different groups and between different samples, performing multiple comparisons between each group and another, and each sample and another by using the "Post Hoc LSD" multiple comparison test.  $P < 0.5$  was considered significant.

## RESULTS

### Changes in food consumption during the whole experiment (Figure 1):

In group I, the mean  $\pm$  SD was  $15.6 \pm 2.56$  g /day/rat. In group II, the mean  $\pm$  SD was  $16.4 \pm 2.7$  g/day/rat during the whole experiment with no significant difference between group I and group II. In group III, the mean  $\pm$  SD was  $11.98 \pm 3.4$  g/day/rat

with significant decrease as compared to group I. In group IV, the mean  $\pm$  SD was  $15.98 \pm 4.5$  g/day/rat during the whole experiment with insignificant difference between group IV and group II. In group V, the mean  $\pm$  SD was  $12.06 \pm 2.3$  g /day/rat with significant decrease when compared to group IV, but with no significant difference when compared to group III. In group VI, the mean  $\pm$  SD was  $15.3 \pm 2.2$  g/day/rat with significant increase when compared to group V. There was no significant difference between group IV and VI. In group VII, the mean  $\pm$  SD was  $11.6 \pm 3.1$  g/day/rat. In relation to group IV, group VII showed significant decrease in food consumption. In relation to group VI, group VII showed significant decrease in food consumption, but with no significant difference as compared to group V.

During the experiment, changes in body weight and food consumption in different groups were more or less concomitant together. Increase in body weight was associated with increase in food consumption and vice versa. ISO showed an increase of food consumption which was accompanied by an increase of body weight. GTE showed reduction of food consumption which was accompanied by loss of body weight, while metoprolol produced an increase of food consumption accompanied by an increase of body weight. GTE and metoprolol in combination produced the same effect that produced by green tea extract.

### Changes in body weight during the whole experiment (Figure 1):

In group I (control-received oral distilled water), the means  $\pm$  standard deviations of body weight (BW) were

116.42 ± 9.4 g at the beginning of the experiment, 128.4 ± 7.37 g at the end of the 2nd week and 136 ± 8.6 g at the end of the 4th week. Within this group, BW showed significant increase at the 2nd measurement and the 3rd measurement in relation to the 1st one. No significant difference was shown between the 2nd and the 3rd measurements.

In group II (control-received saline 0.9% I.P.), the means ± standard deviations were 115.71 ± 8.01 g at the beginning of the experiment, 124.7 ± 8.2 g at the end of the 2nd week, and 135.1 ± 6.4 g at the end of the 4th week. Within this group, BW showed significant increase at the 2nd measurement in relation to the 1st measurement, significant increase at the 3rd measurement in relation to the 1st one and also significant increase at the 3rd measurement in relation to the 2nd one. The differences between control group I and control group II were insignificant at the three intervals.

In group III (control-received green tea extract orally), the means ± standard deviations were 122.14 ± 6.36 g at the beginning of the experiment, 114.7 ± 12.59 g at the end of the 2<sup>nd</sup> week, and 109.14 ± 13.5 g at the end of the 4th week. Within this group, there was no significant difference in BW between the 1st measurement and the 2<sup>nd</sup> one and between the 2nd measurement and the 3<sup>rd</sup> measurement, but there was significant decrease at 3rd measurement as compared to the 1st one. The difference between control group I and group III was insignificant at the beginning of the experiment, but it significantly decreased in group III as compared to group I at the latter two measurements.

In group IV (Myocardial infarction group received isoprenaline I.P.), the means ± standard deviations were 114.14 ± 9.65 g at the beginning of the experiment, 121.3 ± 12.63 g at the end of the 2nd week and 133.3 ± 13.2 g at the end of the 4<sup>th</sup> week. Within this group, the results also showed significant increase at the 3rd measurement in relation to the 1<sup>st</sup> one, but the difference between the 1st measurements and 2<sup>nd</sup> one, and between the 2<sup>nd</sup> measurements and 3rd one were insignificant. The difference between control group II and myocardial infarction group IV was insignificant at the three intervals.

In group V (received GTE. orally + ISO I.P.), the means ± standard deviations were 119.42 ± 8.30 g at the beginning of the experiment, 112.6 ± 12.99 g at the end of the 2<sup>nd</sup> week, and 106.9 ± 11.1g at the end of the 4<sup>th</sup> week. Within this group, the results showed significant decrease at the 3<sup>rd</sup> measurement in relation to the 1<sup>st</sup> one, but the difference between the 1<sup>st</sup> measurements and the 2<sup>nd</sup> one, and, between the 2<sup>nd</sup> measurements and the 3<sup>rd</sup> one were insignificant. The difference between group III and group V were insignificant at the three intervals. The difference in group V and group IV were insignificant at the beginning and at the end of the 2<sup>nd</sup> week, but significantly decreased at the end of the 4<sup>th</sup> week.

In group VI (received metoprolol orally+ ISO I.P.), the means ± standard deviations were 117.57 ± 10.6 g at the beginning of the experiment, 132.1 ± 9.26 g at the end of the 2<sup>nd</sup> week, and 136.9 ± 6.06 g at the end of the 4<sup>th</sup> week. Within this group, BW showed significant increase at the 2<sup>nd</sup> measurement (at the end of 2<sup>nd</sup> week) in relation to the 1st measurement (at the beginning of the

experiment), and also significant increase at the 3<sup>rd</sup> measurement (at the end of the 4<sup>th</sup> week) in relation to the 1<sup>st</sup> one. No significant difference was shown between the 2<sup>nd</sup> and the 3<sup>rd</sup> measurements.

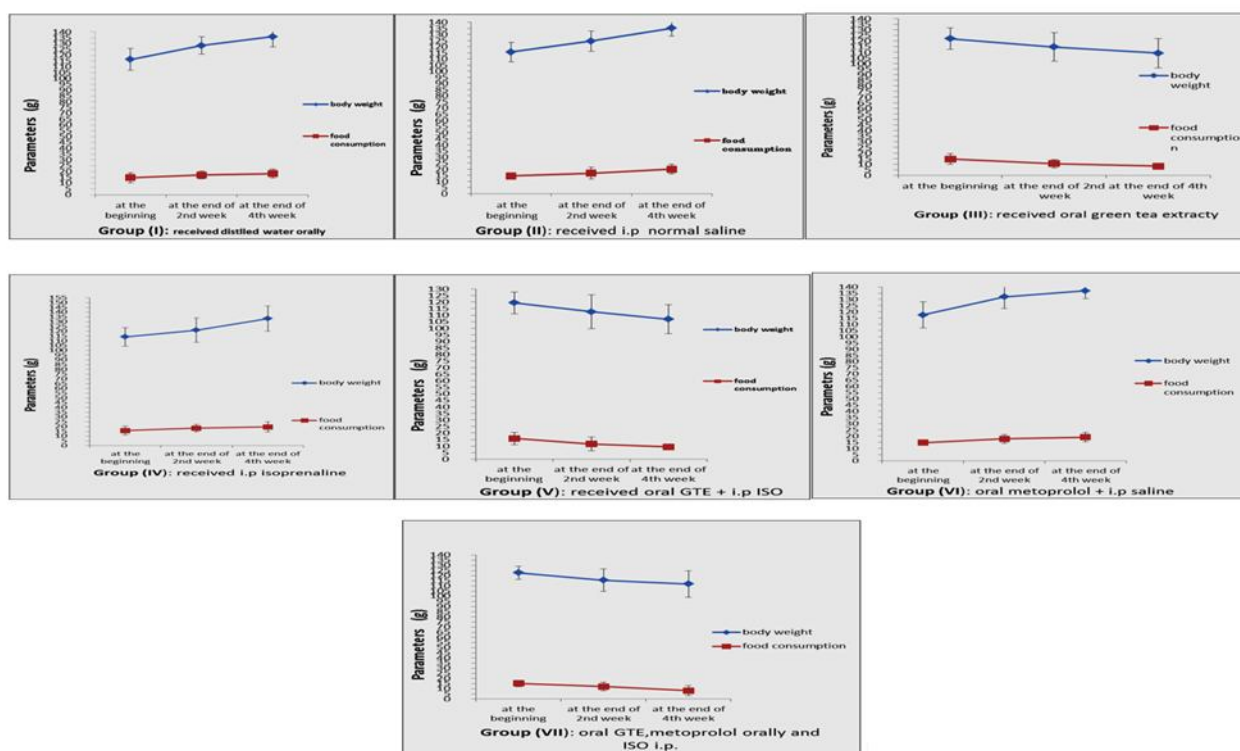
The difference between group IV and group VI were insignificant at any interval. The difference in group VI in relation to group V was insignificant at the beginning of experiment, but at the end of the 2<sup>nd</sup> and 4<sup>th</sup> week increased significantly.

In group VII (received Metoprolol + green tea extract orally +ISO I.P), the means ± standard deviations were 122.85 ± 6.36 g at the beginning of the experiment, 115.8 ±10.90 g at the end of the 2<sup>nd</sup> week and 112 ± 12.8 g at the end of the 4<sup>th</sup> week. Within this group, the result showed significant decrease at the 3<sup>rd</sup> measurement (at the end of the 4<sup>th</sup> week) in relation to the 1<sup>st</sup> one, but the differences between the 1<sup>st</sup> measurements

and 2<sup>nd</sup> one and between the 2<sup>nd</sup> measurements and 3<sup>rd</sup> one were insignificant. The differences in group VII in relation to group IV were insignificant at the beginning and at the end of the 2<sup>nd</sup> week, but at the end of the 4<sup>th</sup> week decreased significantly. The differences between group V and group VII were insignificant at the three intervals. The difference in group VII in relation to group VI was insignificant at the beginning of experiment, but at the end of the 2<sup>nd</sup> and 4<sup>th</sup> week decreased significantly.

So, ISO showed insignificant change in body weight. Green tea extract (GTE) decreased the body weight at the end of experiment, while metoprolol showed insignificant change in body weight. GTE and metoprolol in combination decreased body weight due to the effect of green tea.

Figure (1) Relation between food consumption and body weight in different groups (mean±SD).



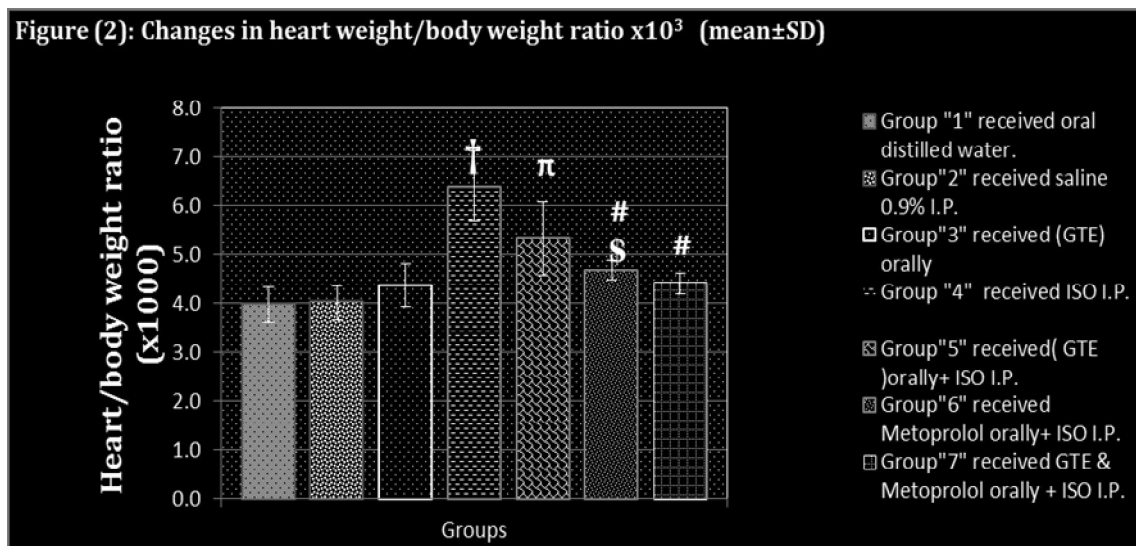
### Heart weight/ body weight ratio (Figure 2):

In group I, the mean  $\pm$  SD of heart weight and heart weight /body weight ratio were  $0.55 \pm 0.076$  and  $3.99 \pm 0.36$  g respectively. In group II, the mean  $\pm$  SD of heart weight and heart weight / body weight ratio were  $0.54 \pm 0.057$  and  $4.02 \pm 0.34$  g respectively, with no significant difference between group I and group II. In group III, the mean  $\pm$  SD of heart weight and heart weight / body weight ratio were  $0.48 \pm 0.077$  and  $4.4 \pm 0.44$  g respectively with insignificant change when compared to group I. In group IV, the mean  $\pm$  SD of heart weight and heart weight / body weight ratio were  $0.85 \pm 0.14$  and  $6.4 \pm 0.72$  g respectively with significant increase when compared to group II. In group V, the mean  $\pm$  SD of heart weight and heart weight / body weight ratio were  $0.57 \pm 0.10$  and  $5.3 \pm 0.75$  g respectively. In relation to group III, the results showed significant increase in the ratio and insignificant change in the heart weight. In relation to group IV, the result showed significant decrease in the

heart weight and the ratio. In group VI, the mean  $\pm$  SD of heart weight and heart weight / body weight ratio were  $0.62 \pm 0.07$  and  $4.7 \pm 0.20$  g respectively. As compared to group IV, the differences in the heart weight and the ratio significantly decreased. In relation to group V, the result showed significant decrease in the ratio and insignificant change in the heart weight. In group VII, the mean  $\pm$  SD of heart weight and heart weight / body weight ratio were  $0.49 \pm 0.04$  and  $4.4 \pm 0.22$  g respectively. As compared to group IV, it was noticed that the difference in the heart weight and the ratio significantly decreased. In relation to group V, the results showed significant decrease in the ratio and insignificant change in the heart weight. As compared to group VI, it showed significant decrease in the heart weight without significant change in the ratio.

So, ISO showed elevation of heart weight/body weight ratio. GTE and/or metoprolol reduced the ratio. However, the effect of combination was better than the effect of GTE alone.

Figure (2): Changes in heart weight/body weight ratio  $\times 10^3$  (mean $\pm$ SD)



## DISCUSSION

The present study showed valuable reduction in food consumption and body weight in the groups given green tea extract (GTE) as compared to other groups. The result showed reduction of body weight and food consumption in (GTE) group as compared to control group, (GTE + ISO) group as compared to ISO group or (ISO + metoprolol) group and (GTE + metoprolol + ISO) group as compared to ISO group or (ISO + metoprolol) group. The result was in agreement with **Sayama et al. (2000)** who observed that the mice given green tea in their diets had a significant suppression of food intake, body weight gain, and fat tissue accumulation.

Green tea demonstrated the ability to suppress appetite. The major appetite suppressant factor lies behind its effect on norepinephrine and dopamine. These catecholamines activate the sympathetic nervous system; one of the known effects includes a reduction in the desire for food. Epigallocatechin gallate (EGCG), one of the most active compounds in green tea, mediates this effect by inhibiting the breakdown of an enzyme that would normally break down the catecholamine (**Kao et al., 2000**).

Other mechanism by the body to manage appetite involves the production of cholecystokinin. This hormone, released in the intestines after the consumption of a meal, tells the brain that the body has received adequate amounts of food. The release of this hormone immediately reduces appetite. The

consumption of green tea increases the release of CCK (**Liao et al., 2001**). **Chantre and Lairon (2002)** observed that administration of GTE to humans has been reported to decrease body weight and body fat. Also, **Mandel et al. (2008)** reported that the long term consumption of green tea and its extract have been associated with weight loss mainly through a thermogenic mechanism. The main active ingredients in GTE, i.e. catechins epigallocatechin gallate (EGCG, epigallocatechin (EGC), epicatechin gallate (EG), and epicatechin (EC) are responsible for many of the beneficial effects of green tea. Catechins make up 30% of green tea leaves by weight and are, therefore, a concentrated source of EGCG. Catechins influence metabolism in several ways, i.e. inhibiting intestinal lipases, decreasing fat absorption, increasing fat excretion, increasing uncoupling proteins, increasing thermogenesis, decreasing lipogenic enzymes and suppressing appetite (**Klaus et al., 2005**). Green tea prevents lipid absorption and stimulates energy consumption (**Dulloo et al., 2000**). Tea catechins can also provide modest shifts in metabolism that may improve weight loss and maintenance (**Hursel et al., 2011**).

Green tea extract has been shown to suppress the elevation of blood glucose during food intake and reduce the body weight. These mechanisms may be related to certain pathways such as modulations of energy balance, endocrine systems, food intake, lipid and carbohydrate metabolism, and redox status (**Yang et al., 2001**). Green tea extract have some

impact on increasing energy expenditure, perhaps through its catechin content. The mechanism for increasing energy expenditure is by inhibiting catecholamine-O-methyl transferase (COMT), an enzyme that degrades norepinephrine. The inhibition of COMT by catechins allows norepinephrine to exert a prolonged influence on thermogenesis and fat metabolism. Both of these metabolic processes are controlled by the sympathetic nervous system via norepinephrine. Most of the rise in metabolic rate is from increased fat oxidation, which would have the greatest impact upon decreasing body fat stores (**Kao et al., 2000**).

The present results showed valuable elevation in heart weight (HW) and heart weight / body weight ratio in ISO treated rats, as compared to control group. Our results were in harmony with those of **Upanlawar & Balaraman (2010)** and **Neha & Lubna (2014)** who reported significant increase in heart weight and heart/ body weight ratio in ISO injected rat which indicate cardiac hypertrophy. **Gayathri et al. (2002)**, **Nirmala & Puvanakrishnan (2004)** and **Chauhan & Naik (2005)** stated that rat injected with ISO S.C showed significant increase in heart weight accompanied by decrease in body weight. **Li et al. (2012)** observed a significant increase in the heart weight and the ratio of heart weight (HW) to body weight (BW) in ISO-induced rats. The increase in HW/BW ratio by ISO injection may be due to that ISO augmented the proliferation of non-contractile protein collagen in heart muscle (**Kumar and Sharma, 2007**). Relatively, short time period of administration of repeated low doses of

isoproterenol induce cardiac hypertrophy (**Kralova et al., 2008**).

**Busatto et al. (1999)** stated that the cause of cardiac hypertrophy induced by isoproterenol by the increased local production of angiotensin II in the heart may make a significant contribution to the rapid development of cardiac hypertrophy in the presence of sympathetic hyperactivity. **Nirmala & Puvanakrishnan (2004)** and **Patel et al. (2010)** reported that the cardiac hypertrophy in rats after ISO injection may be due to ventricular stiffness, increased water content and extensive necrosis of cardiac muscle followed by invasion of the damaged tissues by inflammatory cells. **Choudhary et al. (2006)** revealed that increased generation of reactive oxygen species and oxidative stress is also implicated in the progression of cardiac hypertrophy and heart failure.

The present study showed that the prior administration of GTE to ISO- treated rats reduced the elevation of heart weight and heart weight/ body weight ratio which produced by ISO. Also, heart weight reduced, but the ratio was still higher when compared to GTE treated rats. GTE blocks the development of cardiac hypertrophy in an animal model of chronic renal failure. The administration of GTE results in attenuation of left ventricular hypertrophy, hypertension, and preserved cardiac Na-K-ATPase activity in rats subjected to remnant kidney surgery and prevents increases in reactive oxygen species (ROS) production (**Li et al., 2013**).

Prevention of cardiac remodeling due to increased pressure overload is important to reduce morbidity and



mortality. Green tea extract prevents the development of left ventricular concentric hypertrophy by pressure overload. Cardiac hypertrophy can be induced by suprarenal transverse abdominal aortic constriction (AC) in rats. Experiment showed that after 3 weeks of AC surgery, heart to body weight ratio increased in the AC group by 34% compared to the sham group, while green tea extract administration suppressed the load-induced increase in heart weight by 69%. This suggests that increased left ventricular systolic dimensions and deteriorated systolic function were relieved by GTE (**Hao et al., 2007**). Green tea both prevents and reverses the cardiovascular remodeling and metabolic changes seen in high carbohydrate-fed rats. High carbohydrate diet-fed rats show glucose intolerance, hypertension and mild left ventricular hypertrophy. Administration of green tea to high carbohydrate diet-fed rats prevents and reverses glucose intolerance, increases systolic blood pressure, left ventricular wet weight, interstitial collagen and passive diastolic stiffness (**Rickman et al., 2010**).

The present study showed that the prior administration of metoprolol to ISO-treated rats reduced the elevation of HW and HW/BW ratio which were produced by ISO comparing to GTE group. The ratio decreased as a result of the effect of green tea on body weight without change in heart weight. The present study was in agreement with **Hendawy et al. (2012)** who observed that metoprolol significantly decreases the ventricular weight/body weight ratio. **Mustonen et al. (2010)** proved that metoprolol attenuates the elevated relative ventricular weight in a rat model of hypertensive

cardiac hypertrophy. **Maćzewski and Maćzewski (2008)** reported that metoprolol and ivabradine similarly improve LV function, although metoprolol prevents left ventricular dilation and hypertrophy in the post-infarction rat heart.

The attenuation of HW/BW after administration of metoprolol might be attributed to the reduction of collagen content in muscles due to the inhibitory effect of metoprolol on the concentration of hydroxyproline which plays an important role in collagen synthesis (**Wei et al., 2000**). Metoprolol directly inhibits the hypertrophic mediators including the adrenergic (**Bristow, 1997**), renin-angiotensin-aldosterone (**Blumenfeld et al., 1999**), and endothelin systems (**Krum et al., 1996**) as well as various inflammatory cytokines (**Prabhu et al., 2000**). In addition, **Chan et al. (2011)** found that chronic metoprolol treatment decreases the increased thoracic aortic wall thickness without affecting endothelium-dependent relaxation in the spontaneously hypertensive rats.

Prior administration of both GTE and metoprolol in combination to ISO rats reduced the elevated HW and HW/BW ratio produced by ISO. This indicated that they reduce the stimulus for hypertrophy. Comparing with group which pretreated with GTE alone, the ratio only decreased. Also, on comparing to group which pretreated with metoprolol alone, the heart weight only reduced. Thus, GTE or metoprolol has nearly the same ability to decrease the hypertrophy.

## REFERENCES

1. **Blumenfeld, J.D., Sealey, J. E., Mann, S. J., Bragat, A., Marion, R., Pecker, M. S. and**

- Sotelo, J. (1999):**  $\beta$ -adrenergic receptor blockade as a therapeutic approach for suppressing the renin-angiotensin-aldosterone system in normotensive and hypertensive subjects. *Am J Hypertens.*, 12(5):451-459.
- 2. Boschmann, M. and Thielecke, F. (2007):** The effects of epigallocatechin-3-gallate on thermogenesis and fat oxidation in obese men: a pilot study. *J Am Coll Nutr.*, 26(4):389S-395S.
- 3. Bristow, M. R. (1997):** Mechanism of action of beta-blocking agents in heart failure. *Am J Cardiol.*, 80(11): 26-40.
- 4. Busatto, V., Cunha, V., Cicilini, M.A. and Mill, J.G. (1999):** Differential effects of isoproterenol on the activity of angiotensin-converting enzyme in the rat heart and aorta. *Brazilian Journal of Medical and Biological Research*, 32: 355-360.
- 5. Chacko, S., Priya, T., Kuttan, R. and Nishigaki, I. (2010):** Beneficial effects of green tea. *Chinese Medicine*, 5:13-18.
- 6. Chan, D. and Ng, L.L. (2010):** Biomarkers in acute myocardial infarction. *BMC Med.*, 8(1):34-38.
- 7. Chan, V., Fenning, A., Hoeyb, A. and Lindsay, B. (2011):** Chronic  $\beta$ -adrenoceptor antagonist treatment controls cardiovascular remodeling in heart failure in the ageing spontaneously hypertensive rat. *J Cardiovas Pharmacol.*, 58(4):424-431.
- 8. Chantre, P. and Lairon, D. (2002):** Recent findings of green tea extract AR25 (Exolise) and its activity for the treatment of obesity. *Phytomedicine*, 9: 3-8.
- 9. Chauhan, G. M. and Naik, S. R. (2005):** Cardioprotective activity of A.V. Circulo in isoproterenol-induced myocardial necrosis. *Journal Herbal of Pharmacotherapy*, 5 (4): 51-55.
- 10. Choudhary, R., Mishra, K. P. and Subramanyam, C. (2006):** Prevention of Isoprenaline induced cardiac hypertrophy by eugenol, an antioxidant. *Indian J. Clin. Biochem.*, 21:107-113.
- 11. Dulloo, A.G., Seydoux, J., Girardier, L., Chantre, P. and Vandermader, J. (2000):** Green tea and thermogenesis: interactions between catechin-polyphenols, caffeine and sympathetic activity. *Int. J. Obes. Relat. Metab. Disord.*, 24(2):252-258.
- 12. Gayathri, C., Arulselven, N., Manohar, B.M. and Puvankrishnan, M. (2002):** Antioxidant potential of a novel tetrapeptide derivative in isoproterenol-induced myocardial necrosis in rats. *International Journal of Experimental and Clinical Pharmacology*, 65: 103-108.
- 13. Hao, J., Kim, C.H., Ha, T.S. and Ahn, H.Y. (2007):** Epigallocatechin-3-gallate prevents cardiac hypertrophy induced by pressure overload in rats. *Journal of Veterinary Science*, 8:121-129.
- 14. Hendawy, N., Helmy, A., Salah, H., Karouk, G., Badawy, M. and Sallam, H. (2012):** Differential effects of ivabradine and metoprolol on cardiovascular remodeling and myocardial infarction induced by isoprenaline in chronic N-nitro-L-arginine Methyl Ester (L-NAME) treated rats. *Egyptian Journal of Basic and Clinical Pharmacology*, 2 (1): 26-40.
- 15. Hursel, R., Viechtbauer, W., Dulloo, A.G. (2011):** The effects of catechin rich teas and caffeine on energy expenditure and fat oxidation: a meta-analysis. *Obes Rev.*, 12(7): 573-581.
- 16. Kao, Y. H., Hiipakka, R. A. and Liao, S. (2000):** Modulation of endocrine systems and food intake by green tea epigallocatechin gallate. *Endocrinology*, 141:980-987.
- 17. Klaus, S., Pultz, S., Thone-Reineke, C. and Wolfram, S. (2005):** Epigallocatechin gallate attenuates diet-induced obesity in mice by decreasing energy absorption and increasing fat oxidation. *Int. J. Obes.*, 29(6):615-623.
- 18. Kralova, E., Mokran, T., Murin, J. and Stancovicova, T. (2008):** Electrocardiography in two models of isoproterenol-induced left ventricular remodeling. *physiol. Res.*, 57 (2): S83-S89.
- 19. Krum, H., Gu, A., Wilshire-Clement, M., Sackner-Bernstein, J., Goldsmith, R., Medina, N., Yushak, M., Miller, M. and Packer, M. (1996):** Changes in plasma endothelin-1 levels reflect clinical response to

- beta-blockade in chronic heart failure. *Am Heart J.*, 131(2):337-341.
20. **Kumar R. and Sharma S. (2007):** Isoproterenol hydrochloride augment collagen proliferation and ATPase activity in mice soleus and EDL muscles. *Acta physiologica Hungarica.*, 94(3): 223-230.
  21. **Li, H., Xie, Y. H., Yang, Q., Wang, S.W. and Zhang, B. L (2012):** Cardio protective effect of paeonol and danshensu combination on isoproterenol-induced myocardial injury in rats. *Plos one*, 7(11): 48872-48878.
  22. **Li, N., Zhao, Y. and Liang, Y. (2013):** Cardio protective effects of tea and its catechins. *Health*, 5 (4A): 23-30.
  23. **Liao, S., Kao, Y. H., Dang, M.T., Song, C., Fukuchi, J., Kokontis, J. M. and Hiipakka, R. A. (2001):** Molecular basis for medicinal actions of androgens and green tea epigallocatechin gallate. *Drug Discovery and Traditional Chinese Medicine*, 89- 96.
  24. **Maczewski, M. and Maczewski, U. (2008):** Effect of metoprolol and ivabradine on left ventricular remodeling and Ca<sup>2+</sup> handling in the post-infarction rat heart. *Cardiovas Res.*, 79: 42-51.
  25. **Mustonen, E., Leskinen, H., Aro, J., Luodonpaa, M., Vuolteenaho, O., Ruskoaho, H. and Rysa, J. (2010):** Metoprolol treatment lowers thrombospondin-4 expression in rats with myocardial infarction and left ventricular hypertrophy. *Bas Clin Pharmacol Toxicol.*, 107: 709-717.
  26. **Neha, K. and Lubna, A. (2014):** Evaluation of cardio protective effect of *tinospora cordifolia* against isoprenaline induced myocardial infarction in rats. *Int. J. Curr. Microbiol. App. Sci.*, 3(3): 543-555.
  27. **Nirmala, C. and Puvanakrishnan, R. (2004):** Protective effect of curcumin against isoproterenol induced myocardial infarction in rats. *Biological and Cellular Biochemistry*, 159 (2): 85-90.
  28. **Ojo, O. O., Kabutu, F. R., Bello, M. and Babayo, U. (2006):** Inhibition of paracetamol-induced oxidative stress in rats by extracts of lemongrass (*Cymbropogon citratus*) and green tea (*Camellia sinensis*) in rats. *African J. Biotechnol.*, 5 (12): 1227-1232.
  29. **Patel, V., Upananlawar, A., Zalawadia, R. and Balaraman, R. (2010):** Cardioprotective effect of melatonin against isoproterenol induced myocardial infarction in rats: A biochemical, electrocardiographic and histo-architectural evaluation. *Eur J Pharmacol.*, 644:160-168.
  30. **Pesce, A. (1984):** Lactate dehydrogenase. *Kaplan A. Clin Chem.*, 1124:117, 438.
  31. **Rickman, C., Iyer, A., Chan, V. and Brown, L. (2010)** Green tea attenuates cardiovascular remodeling and meta- bolic symptoms in high carbohydrate-fed rats. *Current Pharmaceutical Biotechnology*, 11: 881-886.
  32. **Sayama, K., Lin, S., Zheng, G. and Oguni, I. (2000):** Effects of green tea on growth, food utilization, and lipid metabolism in mice. *In Vivo, J. Nutr.*, 14:481-484.
  33. **Sutherland, A., Rosanna, M.A. and Rahman, I. (2013):** Mechanisms of action of green tea catechins, with a focus on ischemia-induced neurodegeneration. *Journal of Nutritional Biochemistry*, 17: 291-306.
  34. **Upananlawar, A. and Balaraman, R. (2010):** Preventive effects of *lagenaria sinceraria* (molina) fruit juice in isoproterenol induced myocardial infarction. *International Journal Pharmacology*, 6: 645-651.
  35. **Wei, S., Chow, L.T. and Sanderson, J.E. (2000):** Effect of carvedilol in comparison with metoprolol on myocardial collagen post infarction. *Journal of American College of Cardiology*, 36(1): 276- 281.
  36. **Yang, M.H., Wang, C.H. and Chen, H.L. (2001):** Green, oolong and black tea extracts modulate lipid metabolism in hyperlipidemia rats fed high-sucrose diet. *J Nutr Biochem*, 12:14-20.

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قسم الفسيولوجيا الطبية – كلية طب الأزهر

**خلفية البحث:** هناك زيادة فى إدراك دور الأعشاب وتأثيرها على أمراض القلب. والشاى الأخضر كنبات طبي هام له تأثير قوى كمضاد للأكسدة.

**الهدف من البحث:** تقييم تأثير خلاصة الشاى الأخضر على إستهلاك الطعام ووزن الجسم فى حالات الإحتشاء القلبي فى ذكور الفئران البيضاء البالغة.

**مواد وطرق البحث:** إستخدم فى هذا البحث سبعون فأرا أبيضاً بالغاً من فصيلة محلية تتراوح أوزانها بين 110 – 130 جم (متوسط الوزن 120 جم). وقد تم إحداث الإحتشاء القلبي بالحقن بالأيزوبرينالين. وتم قياس الإستهلاك اليومى للطعام، ووزن الفئران فى نهاية فترة التجربة (ثلاثين يوماً). كما تم حساب نسبة وزن القلب إلى وزن الجسم فى تلك الفئران.

**النتائج:** أحدث الأيزوبرينالين والمتيوبرولول زيادة فى إستهلاك الطعام بنسبة متوازية مع زيادة وزن الجسم، بينما أحدثت خلاصة الشاى الأخضر نقصاً فى إستهلاك الطعام وكذلك وزن الجسم.

**الإستنتاج:** خلاصة الشاى الأخضر ذات فعالية فى نقص إستهلاك الطعام وفى وزن الجسم بنسب متساوية.