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Activity of Chamomile  
(*Matricaria chamomilla* L.)  
Powder on Diabetic Rats**

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## Antidiabetic and Antioxidative Activity of Chamomile (*Matricaria chamomilla* L.) Powder on Diabetic Rats

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### Abstract:

This work is aimed to investigate the effect of chamomile flower and roots on diabetic rats and determine the content of polyphenolic compounds and flavonoid of chamomile. Twenty four adult male albino rats of *Sprague Dawley* strain, weighting  $150 \pm 5$ g were used in this study, the rats were divided into two main groups. Group 1 (6 rats) fed on basal diet as a control (-ve) group. Group 2 was (18 rats) injected with aqueous solution of streptozotocin (55 mg/kg body weight) to induce diabetes by intravenous injection and fed on basal diet as a control (+ve) group. Groups 3 and 4 were administrated 10% and 20% chamomile for 28 days. The results obtained that, the main components of polyphenolic compounds and flavonoid of chamomile (luteolin O-acylhexoside, quercetin, ellagic acid, catechol and chlorogenic acid), respectively. Injected rats with streptozotocin induced significant decrease in feed intake, body weight gain%, HDL-C, acetylcholine esterase, catalase activity and glutathione activity and induced significant increase in (serum brain and glucose), total lipids, cholesterol, triglycerides, LDL-C, VLDL-C and lipid peroxidation, as compared to healthy rats group (non-injected rats). Diabetic rats treated with the levels of 10% and 20% chamomile show significant increasing at the mean value of feed intake, HDL-C, acetylcholine esterase, catalase activity and glutathione activity, on the other hand these treatments induced significant decrease in the other tested parameters, as compared to the (+ve) control. Chamomile improved the nutritional and biochemical status of diabetic rats, especially level at 20% chamomile.

**Key words:** chamomile, polyphenolic compounds, flavonoides, hyperglycaemia.

### Introduction:

Chamomile belongs to daisy family (*Asteraceae* or *Compositae*). It is widely represented by two known varieties *Matricaria Chamomilla*

known as wild chamomile and Roman chamomile considered as common specie. Both types of chamomile are used in traditional medicine and herbalism (**Hansen and Christensen, 2009; Singh et al., 2011 and Raal et al., 2012**). One of the oldest, widely used, native of the old world and well documented medicinal plant is chamomile (**Astin et al., 2000**). *Matricaria chamomilla* is the most popular source of chamomile; a herbal product used for a variety of medical purposes and has been used since ancient times (**Grieve, 1996; Petri and Lemberkovics, 1994**). Chamomile has a favorable effect to inhibit the histopathological changes of the pancreas in STZ-induced diabetes (**Cemek et al., 2008**). Chamomile's one of the main roles is as a multipurpose digestive aid. It can also be used to treat different gastrointestinal disturbances such as anorexia, flatulence, motion sickness, indigestion, nausea, vomiting and diarrhea. Chamomile is used as herbal better to stimulate the liver and also have tendency to heal ulcers (**Mann and Staba, 1986**). some studies recorded that chamomile has potential to lower blood sugar levels in hyperglycemia (**Kato et al., 2008; Raal et al., 2012 and McKay and Blumberg, 2006**).

The active constituents of chamomile are terpenoids: a-bisabolol, a-bisabolol oxide A and B, chamazulene, sesquiterpenes; flavonoids such as apigenin, luteolin, quercetin, umbelliferone, herniarin, esculetin; Spiroethers: en-yndicycloether, other constituents: anthemic acid, choline, tannin and polysaccharides (**Paya et al., 1992; Newall et al., 1996; Kaneko et al., 2007 and Singh et al., 2011**).

Diabetes is a chronic disease characterized by hyperglycemia and many macrovascular complications resulting from defects in insulin secretion (**Chandra et al., 2007**). The macrovascular complications of diabetes are associated with oxidative stress induced by hyperglycemia (**Evans et al., 2002**). There is possibility of hyperlipidemia and liver damage in the later stages of diabetes due to disorders in lipid metabolism and increased gluconeogenesis and ketogenesis (**Virdi et al., 2003**). The side effects of antidiabetic drugs have led to use several species of medicinal plants with hypoglycemic properties (**Li and Crawford, 2004**). The hypoglycemic properties of these plants are reported to be due to their higher contents of flavonoides and different bioactive compounds.

The present study examined the protective effects of chamomile in diabetes induced hyperglycaemia and metabolic disorders.

## Materials and Methods:

### Materials:

- **Chamomile** (*Matricaria chamomilla* L.): Flower and roots of chamomile were obtained from the local market of Egypt.
- **Streptozotocin (STZ)** was obtained from SIGMA Company for Pharmaceutical Industries Cairo Egypt.
- **Rats:** Twenty four adult male albino rats of *Sprague Dawley* strain, weighting  $150 \pm 5$ g were obtained from the Animal Colony, Helwan farm, Egypt.

### Methods:

**HPLC analysis of polyphenols and flavonoids:** HPLC analysis was selected for detection according to (Merfort *et al.*, 1997).

**Animals and treatment:** Adult male albino rats were selected for the study. They had the same age (2 months) and weight ( $150 \pm 5$ g). The animals were housed in acrylic cages in standard conditions of temperature prior to the experiment for 1 week in order to adapt to the laboratory condition, fed with commercial diet and water ad libitum, obtained from the Experimental Animal House of Helwan, Egypt. The animals were kept under observation for five days before experiment and fed on standard diet according to Reeves *et al.* (1993).

### Induction of Diabetes :

Diabetes was induced by intravenous injection of a single doses of streptozotocin (55 mg/kg body weight) to induce diabetes according to Peschke *et al.* (2000). Blood was extracted from the tail vein for glucose analysis and rats with fasting glucose ranging from 210-220 mg/dl, showing clear signs of polyuria, polyphagia and polydipsia were considered diabetic and were analyzed 48 hours after streptozotocin treatment. Animals with fasting blood glucose less than 150 mg/dl were rejected. The rats were divided into the following groups of 6 animals each:

**Group I:** Fed on standard diet only (-ve) control.

**Group II:** Diabetic rats fed on standard diet only (the control group) (+ve) control.

**Group III:** Diabetic rats fed on standard diet containing 10% chamomile as treated group.

**Group IIII:** Diabetic rats fed on standard diet containing 20% chamomile as treated group.

During the experimental period (28 days), the diets consumed and body weights were recorded every week. At the end of the experiment, the rats were fasted overnight, then the rats were anaesthetized and sacrificed, and blood samples were collected from the aorta. The blood samples were centrifuged and serum was separated to estimate some biochemical parameters, i.e. Serum glucose levels were measured according to **Burrin and Price (1985)**. Enzymatic colorimetric determination of triglycerides was carried out according to **Fossati and Prencipel (1982)**. Total lipids, total cholesterol, HDL, VLDL and LDL were determined by colorimetric method according to (**Allian et al., 1974; Fnedewaid, 1972; Gordon and Amer, 1977 and Fnedewaid, 1972**), respectively. Serum in Lipid peroxidation was determined by measuring malondialdehyde (MDA) according to **Ohkawa et al. (1979)**. Catalase (CAT) activity was estimated by **Aebi (1983)**. Glutathione (GSH) level was determined according to previously reported methods **Tietze (1969)**. Acetylcholine esterase (AChE) activity was determined colorimetrically according to **Hestrin (1949)**. Isoenzymatic specturm of AChE was resolved by polyacrylamide gel electrophoresis, as develop by **Davis (1964) and Ornstein, (1964)**.

#### **Statistical analysis:**

The obtained data were statistically analyzed using computerized SPSS. Effects of different treatments were analyzed by one way ANOVA (Analysis of variance) test using Duncan's multiple range test and  $p < 0.05$  was used to indicate significance between different groups (**Snedecor and Cochran, 1967**).

#### **Results and Discussion:**

Table (1) illustrates that the chamomile flower and roots contained high considerable amount of polyphenolic compounds and flavonoid with dominant components consisting of (luteolin O-acylhexoside, querctin,

ellagic acid, catechol and chlorogenic acid) were (2801.99, 1765.01, 1582.81, 1104.49 and 937.48). In similar studies the chemical composition of chamomile flowers that luteolin O-acylhexoside was found to be the predominant constituent (**Hansel et al., 1992**).

The root of chamomile contains only traces Despite the fact that some therapeutically active compounds such as chamazulene and  $\alpha$ -bisabolol are missing, other substances such as E- $\beta$ -farnesene,  $\alpha$ -farnesene, chamomilla ester, en-in-dicycloethers, chamomillol,  $\beta$ -caryophyllene and caryophyllene-epoxide can be detected. The other active substances in the flowers of chamomile are flavonoids, coumarins and polysaccharides (**Della Loggia et al., 1986 and Carle et al., 1987**).

The effect of chamomile on feed intake (g/day rat) and body weight gain% of diabetic rats presented in Table (2). The mean value of feed intake in the (+ve) control decreased significantly as compared to the (-ve) control. Treating diabetic groups with diet containing levels of 10% and 20% chamomile showed significant increase in feed intake, as compared to the (+ve) control. The mean value of feed intake in diabetic groups treated with chamomile decreased significantly, as compared to the (-ve) control group.

The mean value of BWG % of the (+ve) control (diabetic group) showed significant decrease as compared to the (-ve) control ( $13.40 \pm 1.34$  and  $20.60 \pm 0.89$ ) respectively. All treated groups showed significant increase in BWG%, as compared to the (+ve) control. The data in this Table showed non-significant differences in BWG% between diabetic group fed on basal diet containing levels of 10% and 20% chamomile. Chamomile administration has also controlled the reduction in weight in diabetic rats as compared to diabetic control and the results were not very much different from standard. Weight reduction is normally observed in diabetes as insulin plays significant role in lipid metabolism. Insulin insufficiency leads to hypercholesterolaemia because of lipolysis in adipose tissues and protein breakdown resulting in muscle wasting and therefore weight loss (**Agardh et al., 1999; Vasudevan and Sreekumari, 2007 and Mohamed-Amine et al., 2017**). Thus that reduction in weight was controlled probably by controlling the level of insulin.

The results in Table (3) showed the effect of diet containing levels of 10% and 20% chamomile on brain glucose level ( $\mu\text{M/g}$ ) and serum glucose ( $\text{mg/dl}$ ) of diabetic rats. Injected rats with streptozotocin induced increase in brain and serum glucose, as compared to non-injected rats ( $2.60 \pm 0.05 \mu\text{M/g}$  and  $179.60 \pm 4.93 \text{ mg/dl}$ ) vs. ( $0.92 \pm 0.03 \mu\text{M/g}$  and  $95.00 \pm 4.58 \text{ mg/dl}$ ), respectively.

Feeding diabetic rats on basal diet containing levels of 10% or 20% chamomile led to significant decrease in brain and serum glucose, as compared to the (+ve) control. On the other hand, these treatments showed significant increase in this parameter, as compared to the (-ve) control group. Treating diabetic group with diet containing level of 20% chamomile decreased the mean value of brain and serum glucose significantly as compared to the group which treated with diet containing level of 10% chamomile. Brain and serum glucose decreased in diabetic group which treated with 20% chamomile by about 43.814% and 46.362%, than that of the (+ve) control.

In this respect, (**Khan *et al.*, 2014 and Mohamed-Amine *et al.*, 2017**) indicate that chamomile tea have a glucose lowering effect in diabetic rats so its daily consumption can be potentially useful in hyperglycemia. Glycosides, alkaloid and flavonoids are important metabolites responsible for hypoglycemic effect in various plants (**Loew and Kaszkin, 2002**). Chamomile contains high levels of polyphenolic compounds such as coumarins (herniarin, umbelliferone and esculetin) and flavanoids (apigenin, leuteolin and quercetin) (**Paya *et al.*, 1992, Kaneko *et al.*, 2007 and Kato *et al.*, 2008**). **Elbessoumy and Mahmoud (2013)** found that, a possible mechanism of chamomile tea may be improve the insulin secretion either from existing beta cells or its liberation from bounded form.

The effect of diets containing levels of 10% and 20% chamomile on total lipid, cholesterol, triglycerides, high density lipoprotein, low and very low density lipoprotein-cholesterol of diabetic rats were presented in Table (4). Injected rats with streptozotocin increased all lipid profile parameters, except high density lipoprotein-cholesterol, as compared to the (-ve) control group. Treating diabetic groups with levels of 10% and 20% chamomile decreased total lipid, cholesterol, triglycerides, low and

very low density lipoprotein-cholesterol, while high density lipoprotein-cholesterol increased, as compared to the (+ve) control.

Feeding diabetic rats on diet containing 20% chamomile improved all lipid profile, than that of diabetic rats fed on diet containing 10% chamomile. Total lipid, total cholesterol, triglycerides, LDL-C and VLDL-C decreased in level 20% chamomile group by about 34.5%, 38.1%, 20.2%, 67% and 20.2%, than that of the (+ve) control(diabetic group), respectively. On the other hand HDL-c increased by about in this treatment by about 9.003%.

The hyperlipoproteinemia need long term with drugs which lower the lipid level. Prolonged treatment with these drugs can cause serious side effect (**Kovacik and Backor 2007**). In this study serum total lipid, cholesterol (TC), triacylglycerol (TG), LDL-C and VLDL-C are significantly increased in diabetic group compared to the control, while HDL-C is decreased. After treatment with chamomile levels, these parameters improved, as compared to the (+ve) control. The increase in serum levels of cholesterol and TG may be due to the uninhibited actions of lipolytic hormones on the fat depots or the increase in the metabolism of free fatty acids from the peripheral fat depots (**Goodman and Gilman, 1985 and Pari and Latha, 2002**).

**Estakhr and Javdan (2011)** reported that the cause of depletion of glucose, TC and TG in hyperglycemia condition after using *Matricaria recutita* extract is the inhibition of the enzyme glycogen phosphorylase catalyzes glycogenolysis. This action inhibits glucagon which according to feedback inhibition favours the production of insulin (**Liu et al., 2007**).

In agreement with our results, **Sharma et al. (2010)** reported that altered lipid and lipoprotein profile, i.e. increase in TC, TG and LDL-c with fall in HDL-c was reversed towards normal level after oral administration of *Matricaria recutita* in STZ-induced diabetic rats (**Kodama, 2007 and Koski, 2008**).

Data in Table (5) illustrated the effect of diet containing levels of 10% and 20% chamomile on lipid peroxidation (LPO), acetylcholine esterase (AChE), catalase activity (CAT), and glutathione activity (GSH) in the suerm of diabetic rats. The results in this table showed that, injected rats with streptozotocin induced significant increase in LPO,



while acetylcholine esterase (AChE), catalase activity (CAT) and glutathione activity (GSH) decreased significantly of the serum, as compared to non-injected rats. Treating diabetic rats with levels of 10% and 20% chamomile decreased the mean value of LPO significantly and increased CAT, AChE and GSH significantly in the serum of diabetic rats, as compared to the (+ve) control.

Feeding diabetic group on diet containing 20% level of chamomile led to significant decrease in LPO and increased GSH in the serum of diabetic rats, as compared to the group which fed on diet containing 10% chamomile, on the other hand, non-significant changes in the mean values of CAT and AChE was observed between these groups. The best results in these parameters recorded for the group which treated with 20% chamomile.

Chamomile contains several classes of biologically active compounds including several polyphenols (**Granzera et al., 2006 and McKay and Blumberg, 2006**). Some phenolic compounds have the capacity to quench lipid peroxidation products, prevent DNA oxidative damage, and scavenge reactive oxygen species (ROS) such as superoxide, hydrogen peroxide and hydroxyl radicals. An alcoholic extracts of German chamomile inhibited acetylcholine- and histamine-induced spasms (**Novakova et al., 2010 and Petronilho et al., 2012**).

### **Conclusion:**

This study shows that chamomile intake is effective in up-regulating the antioxidant defense mechanism by attenuating LPO and PO. Enhancement in the cholinergic system may be due to its neuroprotective effect in streptozotocin-induced diabetes. The high amount of polyphenolic compounds and flavonoid with dominant components consisting of (luteolin O-acylhexoside , quercetin, ellagic acid, catechol and chlorogenic acid) are likely to be responsible for the higher antioxidant activity of the chamomile. Also, chamomile improved the nutritional and biochemical status of diabetic rats, especially fed on 20% level of chamomile.

**Table (1): Polyphenolic compounds and flavonoid (ppm) of chamomile flower and roots.**

Phenolic compounds	(ppm)
Chlorogenic acid	937.48
Gallic acid	111.98
Catechol	1104.49
Vanillic	93.17
Ellagic acid	1582.81
Protocatechuic	462.54
Alpha-coumaric	67.11
Benzoic	50.49
Caffeine	509.51
luteolin O-acylhexoside	2801.99
Rosmarinic acid	31.23
Protocatechuic	362.14
Caffeic acid	847.65
E-vanillic	233.16
Quercetin	1765.01

**Table (2): Effect of chamomile levels on feed intake and body weight gain % (BWG %) of diabetic rat groups.**

Parameters Groups	Feed intake (g/ day)	BWG %
(-ve) control	17.20± 0.83 <sup>a</sup>	20.60± 0.89 <sup>a</sup>
(+ve) control	11.80± 1.09 <sup>c</sup>	13.40± 1.34 <sup>c</sup>
10% chamomile	15.80± 1.67 <sup>b</sup>	16.00± 1.91 <sup>b</sup>
20% chamomile	16.40± 1.67 <sup>b</sup>	16.40± 1.67 <sup>b</sup>

Values with the same letters by column indicate no significant different ( $p < 0.05$ ) and vice versa.

**Table (3): Effect of chamomile levels on glucose levels of serum and brain of diabetic rat groups**

Parameters Groups	Brain glucose μM /g	Serum glucose mg/dl
(-ve) control	0.92 ± 0.03 <sup>d</sup>	95.00± 4.58 <sup>c</sup>
(+ve) control	2.60 ± 0.05 <sup>a</sup>	179.60± 4.93 <sup>a</sup>
10% chamomile	1.51 ± 0.02 <sup>b</sup>	106.00± 3.16 <sup>b</sup>
20% chamomile	1.22 ± 0.04 <sup>c</sup>	97.80± 0.83 <sup>c</sup>

Values with the same letters by column indicate no significant different (p<0.05) and vice versa.

**Table (4): Effect of chamomile levels on serum lipid profile of diabetic rat groups**

Parameters Groups	Total lipids g/l	TC mg/dl	TG mg/dl	LDL-C mg/dl	HDL-C mg/dl	VLDL-C mg/dl
(-ve) control	2.67± 0.33 <sup>c</sup>	96.73± 0.83 <sup>c</sup>	147.18± 1.58 <sup>c</sup>	20.19± 0.46 <sup>d</sup>	47.90± 1.14 <sup>a</sup>	29.43± 0.31 <sup>c</sup>
(+ve) control	4.25± 0.52 <sup>a</sup>	168.26± 10.96 <sup>a</sup>	216.00± 11.40 <sup>a</sup>	87.63± 12.26 <sup>a</sup>	37.43± 3.53 <sup>c</sup>	43.20± 2.28 <sup>a</sup>
10% chamomile	2.95± 0.19 <sup>b</sup>	108.12± 7.54 <sup>b</sup>	173.95± 3.55 <sup>b</sup>	28.89± 6.78 <sup>b</sup>	40.80± 2.49 <sup>b</sup>	34.76± 0.69 <sup>b</sup>
20% chamomile	2.78± 0.18 <sup>b</sup>	104.13± 5.36 <sup>b</sup>	172.20± 4.38 <sup>b</sup>	25.51± 8.69 <sup>c</sup>	47.85± 1.95 <sup>a</sup>	34.44± 0.87 <sup>b</sup>

Values with the same letters by column indicate no significant different (p<0.05) and vice versa

**Table (5): Effect of chamomile levels on serum lipid peroxidation (LPO), catalase activity (CAT), and acetylcholine esterase (AChE) and glutathione activity (GSH) in the serum of diabetic rat groups.**

Parameters Groups	LPO (nmol)	AChE (nmol)	CAT (nmol)	GSH (mg/g)
(-ve) control	180.4±	5.89±	70.13±	16.4±
	8.24 <sup>d</sup>	0.4 <sup>a</sup>	5.22 <sup>a</sup>	1.4 <sup>a</sup>
(+ve) control	333.5±	3.94±	21.25±	9.2 ±
	11.72 <sup>a</sup>	0.4 <sup>c</sup>	3.47 <sup>c</sup>	0.6 <sup>d</sup>
10% chamomile	301.67±	5.46±	68.33±	11.2 ±
	9.34 <sup>b</sup>	0.7 <sup>b</sup>	6.35 <sup>b</sup>	0.2 <sup>c</sup>
20% chamomile	291.35±	4.65±	63.14±	13.2±
	8.12 <sup>c</sup>	0.6 <sup>b</sup>	7.16 <sup>b</sup>	0.2 <sup>b</sup>

Values with the same letters indicate no significant different ( $p < 0.05$ ) and vice versa

**References:**

1. **Aebi, H.E. (1983):** In: Methods in Enzymatic Analysis, New York, Academic press, 273-302.
2. **Agardh, C.D.; Bjorgell, P. and Nilson, E.P. (1999):** The effect of tolbutamide in lipo-proteins and lipoprotein lipase and hormone sensitive lipase. Diabetes Res. Clin. Pract., 46: 99-108.
3. **Allian, C.C.; Poon L.S.; Chan C.S. and Richmond W. (1974):** Enzymatic determination of total serum cholesterol. Clin. Chem., 20: 470.
4. **Astin, J.A.; Pelletier, K.R.; Marie, A. and Haskell, W.L. (2000):** Complementary and alternative medicine use among elderly persons: One-year analysis of Blue Shield medicare supplement. J. Gerontol. A Biol. Sci. Med. Sci., 55(1): 4-9.
5. **Burrin, J.M. and Price, C.P. (1985):** Measurement of blood glucose. Ann Clin Biochem., 22 (Pt 4):327-42.
6. **Carle, R.; Fleischhauer, J. and Fehr, D. (1987):** Qualitätsbeurteilung von Kamillenolen. Deutscher Apotheker Zeitung 127:2451-2457.
7. **Cemek, M.; Kaga, S.; Simsek, N.; Buyukokuroglu, E.M. and Konuk, M. (2008):** Ant hyperglycemic and antioxidative potential of Matricaria chamomilla L. in Streptozotocin-induced diabetic rats. J Nat Med. 10: 1-10.
8. **Chandra, A.; Mahdi, A.A.; Ahmad, S. and Singh, R.K. (2007):** Indian herbs result in hypoglycemic responses in Streptozotocin-induced diabetic rats. Nutr. Res. 27(3): 161-168.
9. **Davis, B.J. (1964):** Disc Electrophoresis II. Methods and application to human serum proteins. Ann N Y Acad Sci 1964; 121:404-427.
10. **Della Loggia, R.; Tubaro, A.; Dri, P.; Zilli, C. and Del Negro, P. (1986):** The role of flavonoids in the anti-inflammatory activity of *Chamomilla recutita*. In: Plant flavonoids in biology and medicine, AR Liss, New York, S 481-484.
11. **Elbessoumy, A.A. and Mahmoud, E.A. (2013):** Biochemical and clinic opathological studies on some antihyperglycemic herbal

- preparations in streptozotocin induced diabetic rats. Sch. J. App. Med. Sci., 1 (2): 48-55.
12. **Estakhr, J. and Javdan, N. (2011):** Antidiabetic Properties of Matricaria Recutita Extract in Streptozotocin-Induced Diabetic Rats. Pharmacologyonline, 3: 617-621.
13. **Evans, J.L.; Goldfine, I.D.; Maddux, B.A. and Grodsky, G.M. (2002):** Oxidative Stress and Stress-Activated Signaling Pathways: A Unifying Hypothesis of Type 2 Diabetes. Endocr. Rev. 23(5): 599-622.
14. **Fnedewaid, W.T. (1972):** Determination of HDL. Clin. Chem.; 8:499.
15. **Fossati, P. and Prencipel, L. (1982):** Determination of triglycerides, Bicon Diagnostics, made in Germany. Clinical Chemistry; 28: 2077-2078.
16. **Goodman, L.S. and Gilman, A. (1985):** The pharmacological basis of therapeutics, 7th Edition. Mac Millan, New York; 1490-510.
17. **Gordon, T. and Amer, M. (1977):** Determination of HDL. J. Med., 62: 707.
18. **Granzera, M.; Schneider, P. and Stuppner, H. (2006):** Inhibitory effects of the essential oil of chamomile (*Matricaria recutita*) and its major constituents on human cytochrome P450 enzymes. Life Sciences, 78: 856-861.
19. **Grieve, M. (1996):** A modern herbal. Dover Publications Inc. 1982. The Merck Index. 12th ed., Merck Research Labs. Division of Merck and Co. Whitehouse Station, New Jersey, 4434.
20. **Hansel, R.; Keller, K.; Rimpler, H. and Schneider, G. (1992):** (Hrsg.) Hagers Handbuch 4. Chamomilla. Springer-Verlag, Berlin, Heidelberg, New York, Paris, Tokyo, Hong Kong, Barcelona, Budapest, p. 817.
21. **Hansen, H.V. and Christensen, K.I. (2009):** The common chamomile and the scentless may weed revisited. Taxon., 58: 261-264.

22. **Hestrin, S. (1949):** The reaction of Acetylcholine and others Carboxylic acid derivative with hydroxylamine and its analytical application. J Biol Chem 1949; 180: 249-261.
23. **Kaneko, T.; Tahara, S. and Takabayashi, F. (2007):** Inhibitory effect of natural coumarin compounds, esculetin and esculin, on oxidative DNA damage and formation of aberrant crypt foci and tumors induced by 1,2- dimethylhydrazine in rat colons. Biol. Pharm. Bull., 30: 2052 -2057.
24. **Kato, A.; Minoshima, Y.; Yamamoto, J.; Adachi, I.; Watson, A.A. and Nash, R.J. (2008):** Protective effects of dietary chamomile tea on diabetic complications. J. Agric. Food Chem., 56 (17): 8206-8211.
25. **Khan, S.S.; Najam, R.; Anser, H.; Riaz, B. and Alam, N. (2014):** Chamomile tea: Herbal hypoglycemic alternative for conventional medicine. Pak. J. Pharm. Sci., 27 (5):1509-1514.
26. **Kodama, S. (2007):** Effect of aerobic exercise training on serum levels of high-density lipoprotein cholesterol. Archives of Internal Medicine., 167:999.
27. **Koski, R.R. (2008):** Omega-3-acid ethyl esters (Lavas) for severe hypertriglyceridemia. Pharmacy and Therapeutics.; 33:271.
28. **Kovacik, J. and Backor, M. (2007):** Changes of phenolic metabolism and oxidative status in nitrogen deficient Matricaria chamomilla plants. Plant Soil 2007;297:255-65.
29. **Kumar, G.; L. Karthik and B. Rao (2010):** Antimicrobial activity of latex of *Calotropis gigantean* against pathogenic microorganisms- an *in vitro* study. *Pharmacologyonline*, 3: 155-163.
30. **Li, M.K. and Crawford, J.M. (2004):** The Pathology of Cholestasis, Seminars in Liver Disease, 24 (1): 21–42.
31. **Liu, J.; Hongbin, S.; Weigang, D.; Dongyan, M. and Zhang, L. (2007):** Maslinic acid reduces blood glucose in KK-Ay mice. Biol. Pharm. Bull, 30: 2075-78.
32. **Loew, S. and Kaszkin, M. (2002):** Approaching the problem of bioequivalence of herbal medicinal products. *Phytother. Res.*, 16: 705-711.

33. **Mann, C. and Staba, E. (1986):** The chemistry, pharmacology and commercial formulations of chamomile. In Herbs, spices and medicinal plants. Recent advances in Botany, Horticulture and Pharmacology; L.E. Craker and J.E. Simon Eds; Food products Press, 1: 235-280.
34. **McKay, D.L. and Blumberg, J.B. (2006):** A review of the bioactivity and potential healthbenefits of chamomile tea (*Matricaria recutita* L.). Phytother. Res. 20, 519–530.
35. **Merfort, I., Wary, V.S.M., and Willuhan, G. (1997):** Flavonol triglycerides from seeds of *Nigella Sativa*. Phetochem. 46(2):359-363.
36. **Mohamed-Amine, Jabri Mohsen Sakly , Lamjed Marzouki and Hichem Sebai, (2017):** Chamomile (*Matricaria recutita* L.) decoction extract inhibits in vitro intestinal glucose absorption and attenuates high fat diet-induced lipotoxicity and oxidative stress. Biomedicine and Pharmacotherapy. 87: 153–159
37. **Nayeemunnisa Ahmed, (2009):** Streptozotocin diabetes-induced oxidative stress and impairment of oxidative defense system in rat brain: neuroprotective effects of *cichorium intybus* . Department of Pharmacology, Al-Ameen College of Pharmacy, India.. Int J Diabetes & Metabolism 17:105-109.
38. **Newall, C.A.; Anderson, L.A. and Phillipson, J.D. (1996):** Herbal medicines: A guide for health-care professionals. London: Pharmaceutical Press: pp.296.
39. **Novakova, L.; Vildova, A.; Mateus, J.P.; Gonçalves, T. and Solich, P. (2010):** Development and application of UHPLC-MS/MS method for the determination of phenolic compounds in chamomile flowers and chamomile tea extracts. Talanta, 82, 1271-1280.
40. **Ohkawa, H; Ohishi, N. and Yagi, K. (1979):** Assay for lipid peroxides in animal tissues by thiobarbituric acid reaction. Anal Bio.; 95: 351-358.
41. **Ornstein, L. (1964):** Disc electrophoresis I. Background and theory. Ann N Y Acad Sci 1964; 121:321-349.



42. **Pari, L. and Latha, M. (2002):** Effect of Cassia auriculata flowers on blood sugar levels, serum and tissue lipids in Streptozotocin diabetic rats. Singapore Med J., 43(12): 612-621.
43. **Paya, M.; Halliwell, B. and Hoult, J.R. (1992):** Interactions of a series of coumarins with reactive oxygen species. Scavenging of superoxide, hypochlorous acid and hydroxyl radicals. Biochem. Pharmacol., 44: 205-214.
44. **Peschke, E., H. Ebel, H.J. Bromme and D. Peschke, (2000):** Classical and new diabetogens: Comparison of their effects on isolated rat pancreatic islets in vitro. Cell Mol. Life Sci., 57(1): 158-164.
45. **Petri, G. and Lemberkovics, E. (1994):** Gyogynovenyek es drogjaik muszeres vizsgalatanak lehetosegei. Acta. Pharmaceutica Hungarica, 64: 87-93.
46. **Petronilho, S.; Maraschin, M.; Coimbra, M.A. and Rocha, S.M. (2012).** In vitro and in vivo studies of natural products: A challenge for their valuation. The case study of chamomile (*Matricaria recutita* L.). Industrial Crops and Products, 40, 1-12.
47. **Raal, A.; Orav, A.; Puessa, T.; Valner, C.; Malmiste, B. and Arak, E. (2012):** Content of essential oil terpenoids and polyphenols in commercial chamomile (*Chamomilla recutita* L. Rauschert) teas from different countries. Food Chem. 131, 632–638.
48. **Reeves, P.G.; F.H. Nielsen and G.C. Fahmy (1993):** AIN-93 purified diets for laboratory rodents: final report of the American Institute of Nutrition ad hoc writing committee on the reformulation of the AIN-76A rodent diet. *J. Nutr.*;123(11):1939-1951.
49. **Schilcher, H. (1973):** Neuere Erkenntnisse bei der Quantitätsbeurteilung von Kamillenblüten bzw. Kamillenöl. Planta Med. 23:132-144.
50. **Schilcher, H. (1987).** Die Kamille. Handbuch, Wissenschaftliche Verlagsgesellschaft mbh Stuttgart, p. 57.
51. **Sharma, S.B.; Tanwar, R.S.; Rini, A.C.; Singh, U.R.; Gupta, S. and Shukla, S.K. (2010):** Protective effect of Morus rubra L. leaf

- extract on diet induced atherosclerosis in diabetic rats. Indian J. Bioch. Biophys. 47: 26-31
52. **Singh, O.; Khanam, Z.; Misra, N. and Srivastava, M.K. (2011):** Chamomile (*Matricariachamomilla* L.): an overview. Pharmacogn. Rev. 5, 82–95.
53. **Snedecor, G.W. and Cochran, W.G. (1967):** Statistical Methods. 7<sup>th</sup> Ed., The Iowa State University Press., Ames, Iowa, U.S.A.
54. **Tietze, F. (1969):** Enzymatic methods for quantitative determination of nanogram amount of total and oxidized glutathione. Application to mammalian blood and other tissues. Anal Biochem 27: 502-522.
55. **Vasudevan, D.M. and Sreekumari, S. (2007):** Text book of biochemistry for medical student. Japee brothers medical publishers Ltd, New Delhi, pp.151-160.
56. **Virdi, J.; Sivakami, S.; Shahani, S.; Suthar, A.C.; Banavalikar, M.M. and Biyani, M.K. (2003):** Ant hyperglycemic effects of three extracts from Momordica charantia. J. Ethnopharmacol. 88(1): 107-111.
57. **Wang, M.; Avula, B.; Wang, Y.H.; Zhao, J.; Avonto, C.; Parcher, J.F. and Khan, I.A. (2014):** An integrated approach utilising chemometrics and GC/MS for classification of chamomile flowers, essential oils and commercial products. Food Chemistry, 152: 391-398.

## النشاط الخافض للسكر والمضاد للأكسدة للبانونج علي الفئران

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### الملخص العربي:

يهدف هذا البحث الي دراسة تأثير مسحوق عشبة البانونج علي الفئران المصابة بالسكر بالاضافة الي تقدير محتوى وتركيب المركبات الفعالة من بوليفينوليك وفلافونويد في جذور وازهار البانونج. في هذه الدراسة تم استخدام ٣٦ فأر من ذكور سلالة سبراجو داولي اوزانها  $150 \pm 5$  جم تم تقسيمها إلي مجموعتين رئيسيتين، المجموعة الاولي مكونة من ٦ فئران وتم تغذيتها علي الوجبة الاساسية فقط وهي تمثل المجموعة الضابطة والمجموعة الثانية مكونة من ٢٧ فأر تم حقنها بمحلول مائي من مادة بمادة استرنتوزوتوسين (٥٥ ملجم/كجم من وزن الجسم) لاصابتهم بمرض السكر وهذه المجموعة تم تقسيمها الي ثلاث مجموعات فرعية كل منها مكون من ٦ فئران، المجموعة الفرعية الاولي المصافة بالسكر تم تغذيتها علي الوجبة القياسية فقط وهي تمثل مجموعة الكنترول الموجب، المجموعة الفرعية الثانية تغذت علي الوجبة القياسية واطافة نسبة ١٠% من مسحوق البانونج. المجموعة الفرعية الثالثة تغذت علي الوجبة القياسية واطافة نسبة ٢٠% من مسحوق البانونج. وأشارت النتائج الي أن المركبات الفعالة من بوليفينوليك وفلافونويد في جذور وازهار البانونج كانت (luteolin O-acylhexoside , querctin, ellagic acid, catechol and chlorogenic acid) علي التوالي، حقن الفئران بمادة الاسترنتوزوتوسين أدت الي خفض معنوي في كل من كمية المتناول من الغذاء والزيادة في وزن الجسم والكوليسترول المرتفع الكثافة والاسيتيل كولين استريز ونشاط انزيمي الكتاليز والجلوتاثيون، في حين أدت الي ارتفاع معنوي لكل من مستوي سكر الدم في السيرم والمخ والليبيدات الكلية والكوليستيرول الكلي والجليسيريدات الثلاثية والكوليستيرول منخفض الكثافة وكذلك اكسدة الليبيدات بالمقارنة بمجموعة الفئران السليمة (الكنترول السالب)، تغذية الفئران المصابة بالسكر الوجبة الاساسية مضاف اليها نسبة ١٠% و ٢٠% من البانونج البودر أدى إلى زيادة معنوي لكل من كمية المتناول من الغذاء والكوليستيرول المرتفع الكثافة والاسيتيل كولين

استريز ونشاط انزيمي الكتاليز والجلوتاثيون، على الجانب الآخر أدت إلى انخفاض معنوي لباقي القياسات مقارنة بالمجموعة المصابة بالسكر (مجموعة الكنترول الموجب). وتوصى الدراسة إلى أن البايونج أدى إلى تحسين الحالة التغذوية والقياسات الحيوية للفئران المصابة بالسكر وبخاصة بتناول مستوى المرتفع من مسحوق البايونج بنسبة ٢٠%.