In vitro Study for Tannic and Gallic acids as antioxidant in Diabetes Mellitus

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Abstract

Antioxidants are a molecule capable of slowing or preventing the oxidation of other molecule by give up their own electrons to free radicals which start a chain of reactions that damages cells. In this study, we attempted to assessment the antioxidant activity of tannic and gallic acids *in vitro*. These acids extracted from tea specially green tea, pomegranates, persimmons, Most berries, such as cranberries, Citrus, Legumes and Chocolate. The study consist of 150 subject which divided into three groups according to type of diabetes mellitus, smoking and hypertension. Serum lipid peroxidation was promote by incubation with copper ions (copper sulphate) (1×10^{-4} M) at 37° C at 48 hr. the results of the incubation with copper ions showed that copper ions promoted lipid peroxidation which measured by malon di aldehyde (MDA) while tannic and gallic acids can decreased lipid peroxidation when they used alone or together with concentration (1×10^{-4} M) for each one.

Keywords: Diabetes Mellitus, Tannic acid, Gallic acid, Smoking, Hypertension, Lipid Peroxidation

Introduction

Diabetes mellitus (DM) is a disease in which the body does not produce enough, or properly respond to insulin this causes sugar accumulate in the blood, often leading to chronic hyperglycemia[1]. Hyperglycemia results in oxidative stress by generating free radicals and reactive oxygen species (ROS) and it is implicated in pathogenesis of DM. The adverse effects of smoking may results from oxidative damage to biologic substances. Such damage could result both from cigarette smoking and from the activation of phagocytic cells that generate ROS[2], hypertension is induced by oxidative stress, in particular lipid peroxidation (measured as levels of malondialdehyde (MDA). MDA is formed as an end product of lipid peroxidation. Highly reactive free radicals and oxygen species are present in biological systems from a wide variety of sources. These free radicals may oxidize nucleic acid, proteins, lipids or DNA and can initiate a variety of disease such as diabetes [3][4][5].

Antioxidant compounds like phenolic acids, polyphenols and flavonoids scavenge free radicals and thus inhibit the oxidative mechanisms that lead to degenerative diseases. Antioxidant effect of a plant is mainly due to phenolic compounds such as flavonoids, phenolic acids and tannins[6].Polyphenolic antioxidants are potent free radical terminators[7].They donate hydrogen to free radical and hence, break the reaction of lipid peroxidation at the initiation step[8]. Polyphenolic antioxidants are a type of antioxidant containing a polyphenolic substructure which numbering over 4000 distinct species[9]. The main source of polyphenol antioxidants are nutritional, since they are found in a wide array of phytonutrient-bearing foods . [10][11] [12].

In this study we choose the tannic and gallic acids as a polyphenolic antioxidant, which tannic acid is a naturally polyphenolic antioxidant which distributed in tea, nettle, wood, berries. Oak wood is very rich in tannic[13]. Tannic acid has structure as shown in figure(1) with formula($\mathbf{C_{34}H_{28}O_{21}}$) and molecular weight : 772.57 Dalton[14]. Tannic acid has many sub names such as: Gallotanic acid, digallic acid, allotannin, tannimum. Tannic acid is a polymer

of gallic acid molecules and glucose. The anti-oxidant of tannic acid is beneficial[15].There is many experimental evidence for the effects of tannic acid against cardiovascular disease, inflammation, diabetes and urinary tract infections[16] and gallic acid is a polyphenolic antioxidant compound which distributed in sumac, witch hazel, tea leaves, oak, and other plants[17].Gallic acid has structure as shown in figure(1) with formula($\mathbf{C_7H_6O_5}$) and molecular weight : 170.12 Dalton[18]. Gallic acid is found both free and as part of tannins. Gallic acid acts as an antioxidant and help to protect our cells against oxidative damage which was found to show cytotoxicity against cancer cells, without harming healthy cells[19].

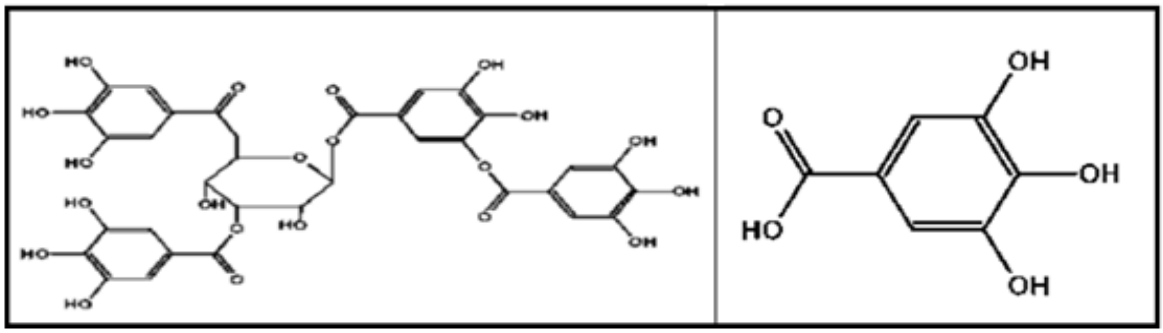

Figure (1): Tannic acid Gallic acid

Tannic acid and Gallic acid may have the potential to become the lead compound in the development of new types of antidiabetic pharmaceuticals that are able to reduce blood glucose levels[20]. These compounds appear to aid in diabetes control and in reducing the complications associated with this disease[21].

Tannic and Gallic acids were tested on pancreatic cells, which produce the hormone insulin in the presence of glucose (sugar). Small blood vessels, called

capillaries, are damaged in diabetes as a result of elevated blood sugar levels[22].

It is important to study the effect of tannic and gallic acids as a polyphenolic antioxidant on oxidation promoted by copper ions *in vitro* in this search.

Materials and Methods:-

1-Design of study

This study conducted at AL-Hussein Education Hospital and The Special Center of The Endocrine Glands and Diabetes in Nassriyah from January 25 ,2011 to July 15 ,2011 .

There were one hundred-fifty (150) subjects, patient with Diabetes Mellitus (DM), aged (18-60) years were included in this study and classified into ten groups according to type of disease, smoking and hypertension as illustrated in the following tables below:-

Table (1):- Data of type of disease groups

Group	Name of group	N	Age (Y)
T1DM	Type 1 diabetes mellitus	50	18-60
T2DM	Type 2 diabetes mellitus	50	18-60

N: number of subjects

Y: years

Table (2):- Data of smoking groups

Group	Name of group	N	Age (Y)
T1DMS	Type 1 diabetes mellitus smokers	50	18-60
T1DMO	Type 1 diabetes mellitus nonsmokers	50	18-60
T2DMS	Type 2 diabetes mellitus smokers	50	18-60
T2DMO	Type 2 diabetes mellitus nonsmokers	50	18-60

T1DMO = T1DM = Type 1 diabetes mellitus

T2DMO = T2DM = Type 2 diabetes mellitus

Table (3):- Data of hypertension groups

Group	Name of group	N	Age (Y)
T1DMP	Type 1 diabetic-hypertension patients	50	18-60
T1DMR	Type 1 diabetic-non hypertension patients	50	18-60
T2DMP	Type 2 diabetic-hypertension patients	50	18-60
T2DMR	Type 2 diabetic-non hypertension patients	50	18-60

T1DMR = T1DM = Type 1 diabetes mellitus

T2DMR = T2DM = Type 2 diabetes mellitus

2- *Sample Collection*

One hundred-fifty subjects with fasting blood sugar (11±0.22 mmol/L) for type 1 diabetes mellitus and (16±1.7 mmol/L) for type 2 diabetes mellitus for all patients subjects. About (4 mL) of blood were withdrawn by venipuncture from each subjects and transferred into disposable tube and prepared to the next treatments.

3- *Blood Treatment with Sulphate copper, tannic and Gallic acids:-*

Each (2.5 mL) of blood samples were treated with several solutions which can be illustrated below:-

	Blood	Buffer	CuSO4	Tannic acid	Gallic acid
Test Tube 1	0.5 mL	0.5 mL	------	------	------
Test Tube 2	0.5 mL	0.5 mL	0.01 mL	------	------
Test Tube 3	0.5 mL	0.5 mL	0.01 mL	0.01 mL	------
Test Tube 4	0.5 mL	0.5 mL	0.01 mL	--------	0.01 mL
Test Tube 5	0.5 mL	0.5 mL	0.01 mL	0.01 mL	0.01 mL

Buffer = phosphate buffer pH= 7.5, (Randox Laboratories, England)

$CuSO_4$ = (1×10^{-4} M , BDH, England)

Tannic acid = (1×10^{-4} M , BDH, England)

Gallic acid = (1×10^{-4} M , BDH, England)

The treated samples then were incubated at 37° C for two days (48 hour). Serum malondialdehyde was measured after centrifugation of samples.

4- Determination of serum malondialdehyde (MDA):-

Determination of serum malondialdehyde level which consider as a lipid peroxidation marker were preformed according to the method of Fong *et al.* 1973[23]. In this method MDA reacts with thiobarbituric acid (TBA). In shaking water bath for 90 min at 60° C to developed a colored complex $MDA(TBA)_2$ which measured at 532 nm after cooling and centrifugation for 10 min at 600 g.

5- Statistical Analysis

Statistical analysis was carried out by One way ANOVA-test was used to compare parameters in different groups. P-values ($p \leq 0.05$) were considered statistically significant. The results were expressed as mean ± standard deviations (mean±SD) by using SPSS version 10.0.

Results and Discussion:-

Free radicals and reactive oxygen attack the normal cells to damage them and lead to formed in the body[24]. Lipid peroxidation is one of several processes which is initiated by free radicals activities on lipids of cell membranes. Polyphenolic antioxidants are potent free radicals and hence, break the reaction of lipid peroxidation at the initiation step[25], therefore, in this study malondialdehyde (MDA) (an end product of lipid peroxidation) to evaluate free radicals generation and catalyzed lipid peroxidation by used copper sulphate and estimate the role of phenolic compounds such as tannic and gallic acids as antioxidants.

According to type of diabetes mellitus table(4) shows a significant ($p \leq 0.05$) increase in serum MDA levels in (B+C) T1DM in comparison with (B, B+C+T, B+C+G, B+C+T+G) T1DM and a significant increase ($P \leq 0.05$) increase in serum MDA levels in (B+C)T2DM in comparison with (B, B+C+T, B+C+G, B+C+T+G)T2DM but a significant decrease in serum MDA concentrations in (B, B+C, B+C+T, B+C+G, B+C+T+G) T1DM in comparison with (B, B+C, B+C+T, B+C+G, B+C+T+G) T1DM

Table(4):- Serum MDA levels of (B, B+C, B+C+T, B+C+G, B+C+T+G) T1DM and T2DM

GROUP	N		MDA concentration (nmol/L)* mean± SD	
	T1DM	T2DM	T1DM	T2DM
B	10	10	64.61 ± 0.63[a]	66.78 ± 0.22[a]
B+C	10	10	84.74± 0.10[b]	89.14± 0.56[b]
B+C+T	10	10	65.22 ± 0.27[c]	68.33 ± 0.13[c]
B+C+G	10	10	68.35 ± 0.08[d]	70.63 ± 0.23[d]
B+C+T+G	10	10	64.92 ± 0.59[e]	66.99 ± 0.06[e]

* Each value represents mean ± SD values with non identical superscript (a , b or c ...etc.) were considered significantly differences ($P \leq 0.05$).

T1DM= Type 1 Diabetes Mellitus

T2DM= Type2 Diabetes Mellitus

B=buffer

B+C=buffer+ $CuSO_4$

B+C+T=buffer+ $CuSO_4$+tannic

B+C+G=buffer+ $CuSO_4$+gallic

B+C+T+G=buffer+ $CuSO_4$+tannic+gallic

Araya M, et al (2002)[26]found that copper ions act as promoter of lipid peroxidation this finding matched with our results. It can be shown that the concentrations of serum MDA has been decreased in case using tannic and gallic acids alone or together. Many reporter[27][28][29] provide that tannic and gallic acids are a free radical scavenger against toxic effects of active oxygen

which decrease lipid peroxidation and MDA concentrations which similar to our finding.

Tables (5) and (6) ,respectively, show the studied group according to smoking. Table(5) showed a significant increase ($p \leq 0.05$) in serum MDA in group (B+C)T1DMS in comparison with (B, B+C+T, B+C+G, B+C+T+G) T1DMS and a significant increase ($p \leq 0.05$) increase in serum MDA levels in (B+C) T1DMO in comparison with (B, B+C+T, B+C+G, B+C+T+G)T1DMO. Also a significant decrease($p \leq 0.05$)(B,B+C,B+C+T,B+C+G,B+C+T+G)T1DMO in comparison with (B, B+C, B+C+T, B+C+G, B+C+T+G) T1DMS.

Table(5):- Serum MDA levels of (B, B+C, B+C+T, B+C+G, B+C+T+G) T1DMS and T1DMO

GROUP	**N**		**MDA concentration (nmol/L)* mean± SD**	
	T1DMS	**T1DMO**	**T1DMS**	**T1DMO**
B	10	10	79.32 ± 0.11^{a}	64.61 ± 0.63^{a}
B+C	10	10	97.67 ± 0.31^{b}	84.74 ± 0.10^{b}
B+C+T	10	10	83.18 ± 0.58^{c}	65.22 ± 0.27^{c}
B+C+G	10	10	87.39 ± 0.29^{d}	68.35 ± 0.08^{d}
B+C+T+G	10	10	80.02 ± 0.43^{e}	64.92 ± 0.59^{e}

* Each value represents mean ± SD values with non identical superscript (a , b or c ...etc.) were considered significantly differences ($P \leq 0.05$).

T1DMS= Type 1 Diabetes Mellitus smokers

T1DMO= Type 1 Diabetes Mellitus non smokers= T1DM

B=buffer

B+C=buffer+ $CuSO_4$

B+C+T=buffer+ $CuSO_4$+tannic

B+C+G=buffer+ $CuSO_4$+gallic

B+C+T+G=buffer+ $CuSO_4$+tannic+gallic

Table(6):- Serum MDA levels of (B, B+C, B+C+T, B+C+G, B+C+T+G) T2DMS and T2DMO

GROUP	**N**		**MDA concentration (nmol/L)* mean± SD**	
	T2DMS	**T2DMO**	**T2DMS**	**T2DMO**
B	10	10	89.26 ± 0.48^{a}	66.78 ± 0.22^{a}
B+C	10	10	112.50 ± 0.19^{b}	89.14 ± 0.56^{b}
B+C+T	10	10	97.74 ± 0.65^{c}	68.33 ± 0.13^{c}
B+C+G	10	10	95.19 ± 0.12^{d}	70.63 ± 0.23^{d}
B+C+T+G	10	10	90.35 ± 0.73^{e}	66.99 ± 0.06^{e}

* Each value represents mean ± SD values with non identical superscript (a , b or c …etc.) were considered significantly differences ($P \leq 0.05$).

T2DMS= Type 2 Diabetes Mellitus smokers

T2DMO= Type 2 Diabetes Mellitus non smokers =T2DM

B=buffer

B+C=buffer+ $CuSO_4$

B+C+T=buffer+ $CuSO_4$+tannic

B+C+G=buffer+ $CuSO_4$+gallic

B+C+T+G=buffer+ $CuSO_4$+tannic+gallic

While table (6) show a significant (p≤0.05) decrease in the concentration of serum MDA in groups (B, B+C+T, B+C+G, B+C+T+G) T2DMS in comparison with (B+C)T2DMS and a significant increase (p≤0.05) in serum MDA levels in (B+C) T2DMO in comparison with (B, B+C+T, B+C+G, B+C+T+G)T2DMO.

Also a significant decrease (p≤0.05)(B,B+C,B+C+T,B+C+G,B+C+T+G) T2DMO in comparison with (B, B+C, B+C+T, B+C+G, B+C+T+G) T2DMS.

It has been reported[30][31][32] that cigarette smoking consider a major risk factor for development of hyperglycemia which causes free radical production and oxidative stress which result increase lipid peroxidation.

Yagi.k ,et al (2002)[33] found that increase lipid peroxidation in smokers supports of hypothesis that smoking increase free radical-mediated oxidative damage of lipid, where this result is similar to our results.

Our previous analysis showed the smoking-diabetic patients have the highest concentration of MDA. Cigarette smoking has already copper ions[34] and the incubation of samples with addition of copper ions (copper sulphate) as a promoter of lipid peroxidation which result this increase in MDA levels[35].

In this research we focused on the antioxidant activity of tannic and gallic acids these acids showed a strong and a substantial activity as an antioxidants by their donation of electron to reactive oxygen species (ROS) which initiated lipid peroxidation and this active belong to tannic and gallic acids may be potentially useful for their structures[36].

According to hypertension- diabetic groups table(7) shows a significant (p≤0.05) increase in serum MDA levels in (B+C) T1DMH in comparison with (B, B+C+T, B+C+G, B+C+T+G) T1DMH and a significant increase (P≤0.05) increase in serum MDA levels in (B+C)T1DMR in comparison with (B, B+C+T, B+C+G, B+C+T+G)T1DMR but a significant decrease in serum MDA concentrations in (B, B+C, B+C+T, B+C+G, B+C+T+G)T1DMR in comparison with (B, B+C, B+C+T, B+C+G, B+C+T+G) T1DMH.

Table(7):- Serum MDA levels of (B, B+C, B+C+T, B+C+G, B+C+T+G) T1DMH and T1DMR

GROUP	N		MDA concentration (nmol/L)* mean± SD	
	T1DMH	T1DMR	T1DMH	T1DMR
B	10	10	71.15 ± 0.95[a]	64.61 ± 0.63[a]
B+C	10	10	92.33± 0.21[b]	84.74± 0.10[b]
B+C+T	10	10	77.24 ± 0.62[c]	65.22 ± 0.27[c]
B+C+G	10	10	80.21 ± 0.81[d]	68.35 ± 0.08[d]
B+C+T+G	10	10	74.64 ± 0.56s[e]	64.92 ± 0.59[e]

* Each value represents mean ± SD values with non identical superscript (a , b or c ...etc.) were considered significantly differences (P ≤ 0.05).

T1DMH= Type 1 Diabetes Mellitus with hypertension

T1DMR= Type 1 Diabetes Mellitus non hypertension

B=buffer

B+C=buffer+ $CuSO_4$

B+C+T=buffer+ $CuSO_4$+tannic

B+C+G=buffer+ $CuSO_4$+gallic

B+C+T+G=buffer+ $CuSO_4$+tannic+gallic

Also a significant (p≤0.05) decrease in the concentration of serum MDA in groups (B, B+C+T, B+C+G, B+C+T+G) T2DMH in comparison with (B+C)T2DMH and a significant increase (p≤0.05) in serum MDA levels in (B+C) T2DMR in comparison with (B, B+C+T, B+C+G, B+C+T+G)T2DMR

and a significant increase (p≤0.05) (B, B+C, B+C+T, B+C+G, B+C+T+G) T2DMH in comparison with (B, B+C, B+C+T, B+C+G, B+C+T+G) T2DMR. As illustrated in table (8).

Table(7):- Serum MDA levels of (B, B+C, B+C+T, B+C+G, B+C+T+G) T2DMH and T2DMR

GROUP	N		MDA concentration (nmol/L)* mean± SD	
	T2DMH	T2DMR	T2DMH	T2DMR
B	10	10	79.51 ± 0.32[a]	66.78 ± 0.22[a]
B+C	10	10	108.47± 0.49[b]	89.14± 0.56[b]
B+C+T	10	10	89.88 ± 0.67[c]	68.33 ± 0.13[c]
B+C+G	10	10	90.14 ± 0.54[d]	70.63 ± 0.23[d]
B+C+T+G	10	10	85.20 ± 0.24[e]	66.99 ± 0.06[e]

* Each value represents mean ± SD values with non identical superscript (a , b or c …etc.) were considered significantly differences (P ≤ 0.05).

T2DMH= Type 2 Diabetes Mellitus with hypertension

T2DMR= Type 2 Diabetes Mellitus non hypertension=T2DM

B=buffer

B+C=buffer+ $CuSO_4$

B+C+T=buffer+ $CuSO_4$+tannic

B+C+G=buffer+ $CuSO_4$+gallic

B+C+T+G=buffer+ $CuSO_4$+tannic+gallic

A preponderance of studies in the past indicates that hypertension induced by oxidative stress which caused by reactive oxygen species and consequently lipid peroxidation increase, also many researches[37][38][39] proved that hypertension seems to be linked to increase some trace elements such as copper, in other words, hypertension induced the increase of lipid peroxidation and copper ions which can be promoted *in vitro* by incubation with copper ions (copper sulphate).

Observable, the results in tables (6)(7) that tannic and gallic acids have a fundamental role as antioxidant and they are capable of minimizing lipid peroxidation marker MDA[40]. Furthermore, the high inhibition of these two acids for copper mediated lipid peroxidation due to their structure which may be the essential factor for their antioxidant property[41][42][43].

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دراسة مختبريه لحامضي التانيك والكاليك كمضادات أكسدة في داء السكري

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الخلاصة

مضادات الأكسدة هي جزيئات قادرة على إبطاء أو منع أكسدة جزيئه أخرى من خلال منحها لإلكترونها إلى الجذور الحرة والتي تبدأ بسلسلة من التفاعلات التي تحطم الخلايا.حاولنا في هذه الدراسة تقييم الفعالية المضادة للأكسدة لحامضي التانيك والكاليك. استخلصت هذه الحوامض من الشاي خاصة الشاي الاخضر ،الرمان ، ثمر البرسيمون ،التوت على الاغلب التوت البري ،الليمون، البقوليات والكاكاو. تضمنت هذه الدراسة 150 شخصاً قسموا إلى ثلاث مجاميع بالاعتماد على نوع داء السكري ، التدخين و ارتفاع ضغط الدم .حيث تم تعزيز الأكسدة الفوقية للدهون بحضن مصل المرضى مع ايونات النحاس (كبريتات النحاس 1×10^{-4}مولاري) لمدة 48 ساعة عند 37° م . أظهرت نتائج حضن المصل مع أيونات النحاس بأن ايونات النحاس سوف يزيد من الأكسدة الفوقية للدهون المقاسة من خلال مستويات المالون ثنائي الالديهايد بينما حامضي التانيك والكاليك يستطيعان خفض الأكسدة الفوقية للدهون (MDA) عند استخدامهما لوحدهما أو كليهما وبتركيز (1×10^{-4}مولاري) لكل منهما .

الكلمات المفتاحية :- داء السكري، حامض التانيك، حامض الكاليك، التدخين، ارتفاع ضغط الدم، الأكسدة الفوقية للدهون