

Study of the effect of Beta glycan supplemented diet on vascular functions in male rats

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Keywords

- Beta glycan
- Hypertension
- vascular reactivity
- oxidative stress
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Abstract

Background/ Aim: Several studies suggested that beta-glucan may have a cardioprotective role against developing high blood pressure (BP). The current study investigated the underlying mechanism of beta glycan as antihypertensive and its effect on vascular functions in males. **Materials & Methods:** A total of 20 adult male Wistar rats weighing 180-200 g were divided into 2 groups 10 per each. **Group I:** Control group were fed standard rat chow for 15 weeks. **Group II:** Beta glycan fed group in the form of oat cereals by gavage (305 mg per kg body weight per day) for a period of 15 weeks. Rats of both groups were subjected to blood pressure measurement at 0, 10, 15 weeks. On the day of sacrifice, blood samples were collected for measurement of plasma catalase, plasma MDA & lipid profile. Aortic tissue was excised to study vascular reactivity and measurement of aortic tissue nitrate. **Results:** Significant reduction in the systolic & diastolic blood pressure in beta glycan fed group. This was accompanied by significant increase in plasma catalase & aortic tissue nitrate while plasma MDA, TC, TAG, LDL & atherogenic index were significantly decreased. Significant decrease in the contractile response of aorta to phenylephrine with significant increase in the relaxing response to acetylcholine in the beta glycan fed ones. **In Conclusion:** Beta glycan is a beneficial antihypertensive vasorelaxing agent that is recommended to be added to the diet especially in males to balance the pro-atherogenic effect of testosterone. This cardioprotective role is achieved through decreasing the oxidative stress, triglycerides and total cholesterol level. In addition, the aortic tissue nitrate was significantly increased which is considered to be a powerful vasodilator agent.

INTRODUCTION

Hypertension is one of the main causes of death around the world and is recognised as a global health issue. There are multiple risk factors which contribute to an individual's risk of developing high blood pressure e.g. age, ethnicity, smoking, family history and gender (1).

Distinct gender differences in the incidence and severity of hypertension are well established where males have a higher incidence of hypertension compared to females of the same age. There is significant evidence that androgens, such as testosterone, play an important role in gender-associated differences in blood pressure regulation (2).

Increases in blood pressure are more evident in males especially over the age of 50, total testosterone is inversely related with systolic and diastolic blood pressure resulting in increased risk of death (3). However, some studies showed that low testosterone is associated with increased pulse wave velocity in older hypogonadal males which was partially reversed with testosterone supplementation (4).

Another line of evidence that testosterone may play an important role in higher blood pressure in males is castration studies in male rats because castration attenuated the development of hypertension in male rats. Moreover, Women with polycystic ovary syndrome which are characterized by elevated testosterone levels, experience hypertension (5)

Because male rats have higher blood pressures than do females, it is possible that female hormones may play a role in protecting females from developing higher blood pressures. Interestingly, the blood pressure does not increase during the transitional phase from perimenopause

to menopause, but rather the increase in blood pressure after menopause develop, suggesting that lack of female hormones may not be the only contributing factor for the elevated blood pressure.(6)

Men have a proatherogenic plasma lipid profile, including greater plasma triglyceride (TG) and total and low-density lipoprotein (LDL)-cholesterol concentrations, lower high-density lipoprotein (HDL)-cholesterol concentration, and smaller HDL particles, than women (7). On the other hand, castration of male rats decreases plasma TG concentration, and testosterone replacement reverses this effect (8).

However, few studies have evaluated the effect of testosterone on plasma lipid metabolism and concentrations, and the results are equivocal because many studies used synthetic androgen receptor agonists rather than testosterone per se, or used supraphysiological doses of testosterone or evaluated the effects of testosterone therapy in men who already have high baseline testosterone availability(9).

Oxidative stress plays a central role in the development of vascular aging in hypertension. Impairment in reactive oxygen species (ROS) regulation, especially superoxide appears to modulate this decrease in nitric oxide (NO) through uncoupling of NO synthase and result in the production of peroxynitrate which may produce deleterious effects as well (10).

NO plays an important role as a main modulator of vascular homeostasis and vascular health. It is recognized for its potential as arteriolar vasodilator (11).

The relationship between testosterone and redox status is still complex (12). Some studies stated that ROS generation was increased by

testosterone, while others found that a decrease in antioxidant activity was observed and reversed with testosterone supplementation (13)

New diet trends are suggested to antagonize the pro-hypertensive effects of AAS e.g. high-fiber diet and beta-glucan (14). Studies reported the potential of oats containing beta glycan against cardiovascular disease and that consumption of beta-glucan has been reported to reduce systolic BP and diastolic BP(15).

Maki et al., (2007) (16) stated that beta glycan is beneficial in terms of reducing BP in obese men with body mass index above a median of 31.5 kg/m² and that consumption of oats with beta-glucan (5.5 g per day) has been reported to reduce systolic BP by 7.5 mmHg and diastolic BP by 5.5 mmHg in patients diagnosed with mild or borderline hypertension.

The current study aims to investigate the effect of Beta glycan on the vascular reactivity and the possible underlying mechanism(s) of beta glycan supplemented diet on decreasing blood pressure in male rats

Site of Research: Physiology Department, Faculty of Medicine, Ain Shams University, Cairo, Egypt.

Materials and Methods:

Ethics Committee

All rats care according to the criteria outlined in the "Guide for the Care and Use of Laboratory Animals" prepared according to guidelines of animal use of the Ethical committee of Ain Shams University and according to National Institutes of Health guide for the care and use of laboratory animals (NIH Publications No 8023, revised 1978).

Experimental Protocol:

Animals:

Twenty adult male Wistar rats, initially weighing 180-200 grams(aging 8-10 weeks), were purchased from the Animal Farm (Helwan), Egypt, and were housed in Animal House of Ain Shams Medical Research Centre (5 rats / cage) with suitable ventilation, temperature of 22-25°C, 12 hours light dark cycle and free access to food (standard rat chow) and water *ad libitum*.

After one week of acclimatization, rats were randomly and equally divided into the following two groups:

Group I: Control group (10 rats): rats of this group were given diet consisting of bread, milk, green vegetables and tap water was provided *ad libitum* for 15 weeks.

Group II: Beta glycan supplemented diet group (10 rats): rats of this group are fed oat cereals that contains beta glycan (305 mg per kg body weight per day) by oral gavage for a period of 15 weeks (17). Oat cereals have been obtained from the local market Cairo-Egypt.

The oats were grinded enough into a fine powder using a blender, when a tablespoon of oats dissolves easily in hot water until reaching palatable drink(18)

. The drink was then given to rats by gavage

Experimental Procedures:

Blood pressure (BP) measurement was carried out in both groups of animals at 0 week (baseline) as well as 10 and 15 weeks of diet supplementation before sacrifice (17). Measurements of arterial blood pressure (ABP) using small animal tail blood pressure system (NIBP200A).

The animal heating chamber was switched on and the temperature was set to at 32°C to warm the animal tail, this is followed by putting the rat inside the restrainer (animal holder) suitable to its

size, with its tail outside allowing a limited movement. Within the restrainer, the rats were placed in a heating chamber for 30 minutes then removed.

The suitable IRsensor was connected to the tail of the rat inside the restrainer, fitting between the midpoint of the tail and tail end. Measurements were not started till the animal was relaxed and inactive.

Recording

1- After launching the BIOPAC software, the IRsensor was calibrated for the pressure (A1). By pressing the start button on the front panel of NIBP200A, the IRsensor pumped up the cuff automatically.

3- When the cuff pressure on A1 reached 30mmHg, the cuff pressure and tail pulse signals were generated, then the recording stopped automatically after 24 seconds.

4- Serial measurements were recorded and mean values were calculated.

Analysis

Calculation of systolic, diastolic and mean blood pressure

1- The A1 channel and the cursor I were selected, and then an area from the graphical display was selected, starting from the point of first pulse to the point of the maximum wave.

2- The maximum value (systolic pressure), minimum value (diastolic pressure) and the mean blood pressure (mean value) were reviewed from the label on the top of the A1 panel.

At the end of the 15 weeks experimental period, all rats were subjected to the following measurements:

On the day of sacrifice, overnight fasted rats were anaesthetized with intraperitoneal injection of Pentobarbitone (40-60 mg/kg body weight)

with booster doses as needed. The dose was given according to the guidelines rodent anesthesia analgesia formulary-UBC animal care

When the stage of surgical anaesthesia (judged by loss of withdrawal reflexes) had been reached , the animal was placed on its back and fixed on the operating table the following was done:

1- Abdominal incision was done and blood samples were collected from abdominal aorta into a heparinized tube.

Blood of heparinized tubes was centrifuged, and the separated plasma was used for:

- (i) **Estimation of plasma catalase** This was carried out using an enzymatic colourimetric technique according to *Aebi (19)*, using kits supplied by Bio-Diagnostic Co
- (ii) **Estimation of plasma levels of malondialdehyde (MDA):** This was carried out using an enzymatic colourimetric technique according to *Satoh (20)* as thiobarbituric acid reactive substrate. Using kits supplied by Bio-Diagnostic Co.
- (iii) **Triglycerides measurement:** This was carried out using enzymatic calorimetric method according to *Fassati and Prencipe (21)*. The kits were supplied by Biodiagnostic Co
- (iv) **Cholesterol measurement:** Plasma cholesterol was estimated by enzymatic calorimetric technique using kits supplied by Biodiagnostic Co. as described by *Richmond (22)*.
- (v) **HDL measurement:** This was carried out using enzymatic calorimetric method

according to *Burstein (23)*. The kits were supplied by Biodiagnostic Co.

(vi) **LDL measurement:** From the previous data (total cholesterol, triglycerides and HDL), LDL was calculated using the following formula *Friedewald (24)* Total cholesterol - (Triglycerides + HDL) 5.

(vii) **Atherogenic index (AI):** From the previous data (total cholesterol and HDL), AI was calculated using the following formula *Grundy et al., (25)*: Total cholesterol.

HDL

(2) Study of aortic ring reactivity

a- **Preparation of isolated aortic rings according to *Rahimian et al., (26)*:**

Chemicals: the following chemicals were purchased from El-Gomhorya Co: Sodium chloride, potassium chloride, calcium chloride, disodium ethylene diamine tetra- acetic acid (EDTA), sodium bicarbonate and glucose.

Phenylephrine and acetylcholine were purchased from Sigma Co.

The thoracic aorta was dissected out, excised and rapidly placed at room temperature in modified Krebs's solution containing (in Mm / L): NaCl 119, NaHCO₃ 24.9, KCl 4.7, Na₂ EDTA 0.023, MgSO₄ 7 H₂O 1.17, KH₂PO₄ 1.18, CaCl₂ 1.6 and glucose 11.1 mM.

On each day of the experiment, NaCl was freshly prepared at a concentration of 9 g / L. Freshly prepared NaHCO₃ (12.93 g / L) was gassed for 30 minutes with carbogen (95% O₂ : 5% CO₂).

Finally, all these solutions were used to prepare the working modified Krebs's solution by adding the following volumes as indicated below:

Substance	Stock (g/L)	Volume (mL)
NaCl	9	1000
KCl	11.5	40
CaCl ₂ (anhydrous)	11.2	19.2
MgSO ₄ .7H ₂ O	38.2	10
KH ₂ PO ₄	21.4	10
Na ₂ EDTA	40.7	0.3
NaHCO ₃	12.93	210

Glucose was dissolved in the mixture at a final concentration of 2 g / L. After addition of freshly prepared sodium bicarbonate solution (12.92 g / L) gassing with carbogen O₂: CO₂ (95% : 5%) was continued for the whole mixture till the end of the experiment.

The aorta was cleaned from adhering fat and connective tissue then cut into rings. Rings of aorta were suspended separately in an organ bath containing warmed modified Krebs's solution continually bubbled with 95% O₂ : 5% CO₂.

Rings were suspended horizontally between 2 stainless steel hook, one of them was fixed in the bath and the other connected to isometric force displacement transducer (UGO Basile, Comerio- Italy) for recording isometric contractions.

Rings of aorta were suspended horizontally between 2 stainless steel hook, one of them was fixed in the organ bath containing modified Krebs's solution and the other connected to isometric force displacement transducer (UGO Basile, Comerio- Italy) for recording isometric contractions in response to Kcl, PE, Ach. Recording was performed on a 4 channel oscillograph (UGO Basile Quartet 9400 four

channel recorder). The weight of the aortic rings was measured in 5 –Digit Metler balance (AE163). Rings were frozen at -80°C for subsequent estimation of tissue nitrate level

b- Recording and measurement of tension developed by aortic rings

Aortic rings were equilibrated for 60 min. under resting tension of 1 gm to allow development of a stable basal tone. In the last 30 min. of equilibration, washing of rings with 80 mM KCl (El-Gomhorya Co.) was repeated three times every 10 min (26)

Then rings were allowed to contract in response to 20 mM KCl until reproducible evoked contractile response was obtained. After recording the contractile response, rings were washed with modified Kreb's solution. After 15 min. later, the contractile response of aortic rings to 1 μM PE (Sigma) was recorded. After the plateau of contraction was reached, the relaxant response to 10 μM ACh (Sigma) was obtained on top of PE contraction.

Recording was performed on a 4 channel oscillograph (UGO Basile Quartet 9400 four channel recorder). The speed of recording was 2.5 mm /min and the sensitivity was 3.5. The resultant curve and its upstroke represent the contraction and the down stroke represents the relaxation.

The weight of the aortic rings was measured in 5 –Digit Metler balance (AE163). Rings were frozen at -80°C for subsequent estimation of tissue nitrate level.

Calculation of the results

The amplitude of the recorded contraction and relaxation was measured in millimetres and the equivalent force in grams was obtained from calibration curve. The calibration curve was performed at sensitivity 3.5 by adding different

weights ranging from 77.2 to 728.8 mg to the threads of the transducer and the amplitude of the response was recorded and the calibration curve was constructed. In this study, 5 millimetres were equivalent to 77.2 mg.

PE induced contraction was calculated as absolute value as well as percentage of KCl induced tension. Also, PE induced contraction was corrected for the weight of aortic ring. The relaxant response to ACh was determined as absolute values and as percentage of the KCl induced tension. Also, ACh induced relaxation was corrected for the weight of aortic ring.

(2) Determination of nitrate level in aortic tissue

The dissected pieces of thoracic aorta which was not subjected to vascular reactivity study were homogenized and the supernatant of aortic tissue homogenate was subjected to nitrate level assay.

On the day of assay, aortic tissues were allowed to thaw, cut into rings about 1-2 mm, weighed and homogenized in homogenization buffer (pH 7.2), for each 0.1 mg aortic tissue 1 ml buffer was added.

The supernatant of aortic tissue homogenate was subjected to nitrate level assay according to *Bories and Bories* (27) and modified by *Kassim* (28) method

Statistical Analysis

All results in this study were expressed as mean \pm SEM. Statistical Package for the Social Sciences (SPSS, Inc., Chicago, IL, USA) program, version 20.0 was used to compare significance between each two groups. Student's t test for unpaired data for intergroupal differences was used. Differences were considered significant when P is ≤ 0.05 .

Results:**(I) Arterial blood Pressure (Table “1” & Figure “1”)**

Systolic blood pressure (SBP), Diastolic blood pressure (DBP) and Mean arterial blood pressure (MAP) showed significant decrease in the Beta glycan group compared to the control group.

(II) Aortic ring responses to potassium chloride (KCl), phenylephrine (PE) and acetylcholine (ACh) (Table “2” Figures “3&4”)

Aortic ring showed significant decrease in response to KCl in the Beta glycan group compared to the control group. Significant decrease in the aortic ring response to phenylephrine (PE) was observed in the Beta glycan ones compared to control rats. Non Significant decrease in PE/KCl & in the response of aortic ring to ACh in the Beta glycan group compared to the control group while significant increase was observed in the ACh/ KCl & ACh/

PE percentage in the Beta glycan rats when compared to their controls.

(III) Biochemical Measurements (Tables “4 &5”, Figures “4-8”)

Rats in Beta glycan group showed significant increase in the plasma catalase and aortic tissue nitrate when compared to the control rats. On the contrary, malondialdehyde level was significantly decreased in the Beta glycan rats compared to their controls.

As regards lipid profile, plasma triglycerides (mg/dl), plasma total cholesterol (mg/dl) and low density lipoproteins (mg/dl) levels showed significant decrease in the Beta glycan rats than control rats .This was accompanied by non significant decrease in the plasma high density lipoproteins in the Beta glycan group while Atherogenic index was significantly decreased in the Beta glycan rats than the control rats.

Table-1: Mean± SEM of systolic blood pressure (SBP, mmHg), diastolic blood pressure (DBP, mmHg) and mean blood pressure (MABP, mmHg) in the different studied groups.

Control n=10	SBP (mmHg)	DBP (mmHg)	MABP (mmHg)
Mean	102.7	80.4	90.3
SEM	±1.6	±1.05	±0.96
Beta glycan group n=10			
Mean	95.5	75.1	84.5
SEM	±1.8	±1.16	±1.37
P	≤0.01	≤0.01	≤0.01

Values are mean ± SEM of 10 rats in each group. P: Significance from the control group, at least $P \leq 0.05$

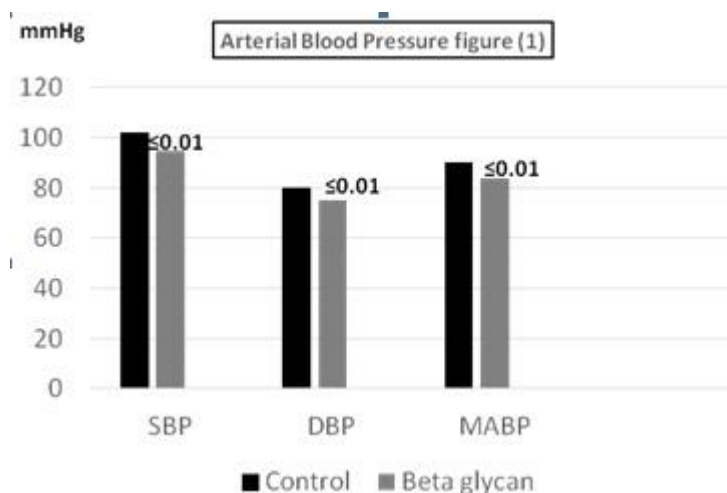


Table (2): Mean± SEM of aortic ring responses to potassium chloride (KCl), phenylephrine (PE) and acetylcholine (ACh) in the different studied groups.

Control (n=10)	KCl/ Aortic wt (g/mg)	PE/ Aortic wt (g/mg)	PE/ KCl (%)	ACh/ Aortic wt (g/mg)	ACh/ KCl (%)	ACh/ PE (%)
Mean	0.06	0.19	337.4	0.03	71	19.8
±SEM	±0.009	±0.009	±32.8	±0.003	±12.1	±2.35
Beta glycan group (n=10)						
Mean	0.02	0.06	325	0.02	136	50.2
±SEM	±0.003	±0.007	±58.4	±0.004	±21.8	±11.9
P	≤0.01	<0.001	NS	NS	<0.05	<0.05

Values are mean ± SEM of 10 rats in each group. P: Significance from the control group, at least P≤0.05

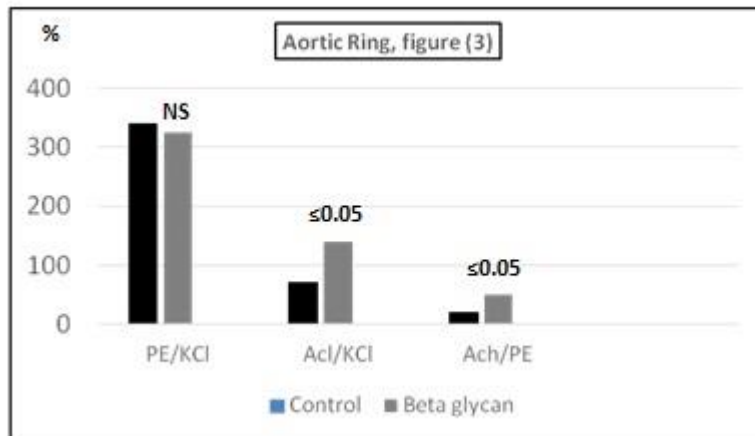
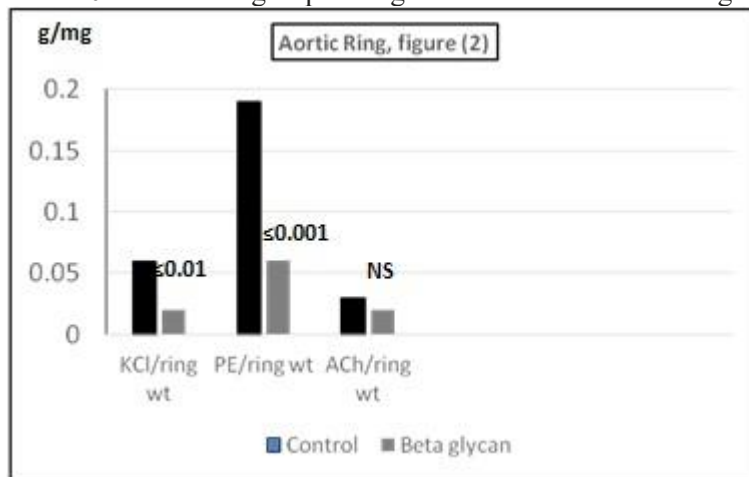


Table (4): Mean± SEM of of plasma catalase (U/L), malondialdehyde (ng/ml) and aortic tissue nitrate levels (µmol/g) in the different studied groups.

	Catalase (U/L)	Malondialdehyde (ng/ml)	Aortic nitrate (µmol/g)
Control n=10			
Mean	121.5	20.2	0.08
SEM	±79	±2.29	±0.01
Beta glycan group n=10			
Mean	678.5	3.67	1.76
SEM	±25	±0.52	±0.32
P	<0.001	<0.001	<0.001

Values are mean ± SEM of 10 rats in each group. P: Significance from the control group, at least P≤0.05

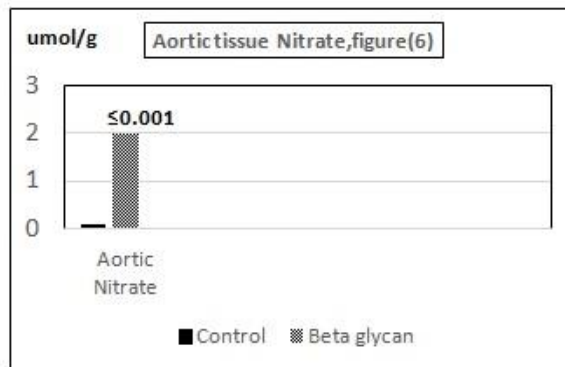
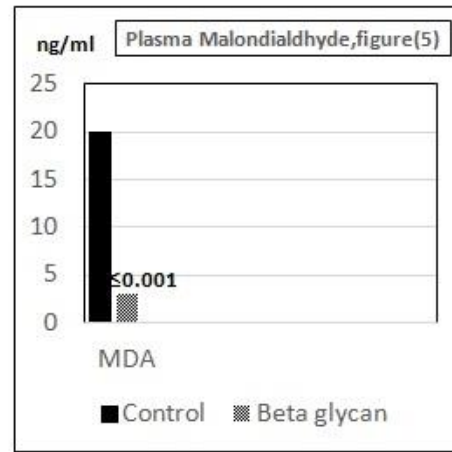
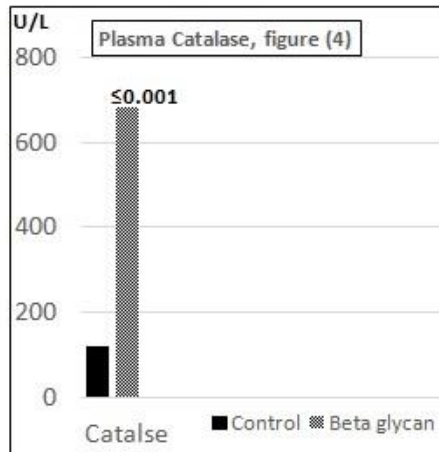
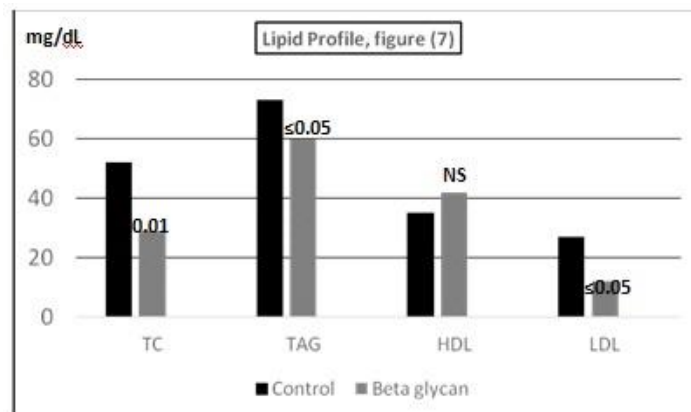
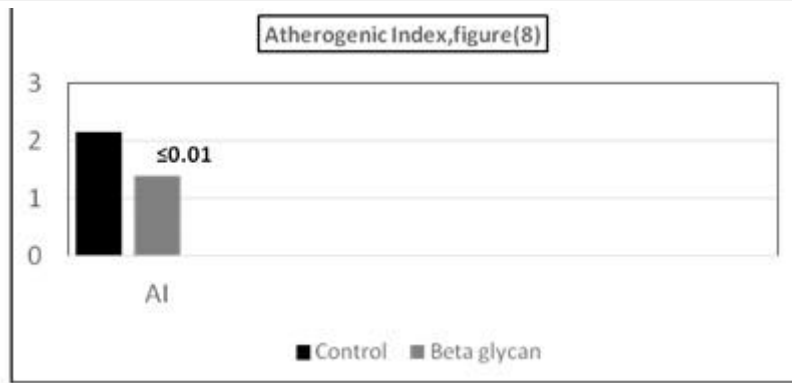


Table (5): Mean± SEM of plasma triglycerides (TG, mg/dL), plasma total cholesterol (TC, mg/dL), plasma high density lipoproteins (HDL, mg/dL), low density lipoproteins (LDL, mg/dL) and atherogenic index (AI) in the different studied groups

	TAG (mg/dL)	TC (mg/dL)	HDL (mg/dL)	LDL (mg/dL)	AI
Control n=10					
Mean	52.3	73.2	35.5	27.2	2.15
±SEM	±4.27	±4.48	±2.42	±5.34	±2.19
Beta glycan group n=10					
Mean	29.2	60.5	42.4	12.3	1.41
SEM	±4	±3.23	±2.8	±1.78	±0.06
P	<0.01	<0.05	NS	<0.05	<0.01

Values are mean ± SEM of 10 rats in each group. P: Significance from the control group, at least P≤0.05





Discussion:

The present study investigated the effects of Beta glycan rich diet on the arterial blood pressure, aortic ring vascular reactivity to phenylephrine and acetylcholine in male rats. In addition, the plasma redox state, lipid profile and aortic tissue nitrate were determined.

Beta glycan rich diet resulted in significant decrease in the systolic, diastolic blood pressure and mean arterial blood pressure. Significant decrease in the aortic ring response to the pressor agent phenylephrine with significant increase in the relaxing response to Ach evidenced by increase in the ACh/ PE percentage. Biochemical analysis revealed that Beta glycan fed group experienced significant increase in the plasma catalase level while malondialdehyde level was significantly decreased. This was accompanied by significant decrease in the plasma triglycerides, plasma total cholesterol, low density lipoproteins & Atherogenic index. Furthermore, Aortic tissue nitrate was significantly increased. These findings highlight the cardioprotective beneficial role of Beta glycan in reducing the risk of developing high blood pressure and hyperlipidemia, also it can help treat it if already the condition is present.

Endogenous androgens may have deleterious effects on the cardiovascular system since young men have higher blood pressure and greater risk

of developing myocardial infarction than matched-age women (29).

Despite the advancement in treatment for hypertension over the years, new evidence-based hypertension management strategies are needed to tackle the current challenges associated with routine therapies (30). Studies suggest that a diet containing soluble fiber-rich whole oats containing Beta glycan can significantly reduce the need for antihypertensive medication and improve BP control. Considering the lipid improvements increased consumption of whole oats may significantly reduce cardiovascular disease risk (31).

An important finding of the current study is that fifteen weeks oat cereals containing beta-glycan supplementation resulted in a decrease of systolic, diastolic and mean arterial blood pressure (BP).

This was associated by combined decreasing contractile response to PE and the increasing relaxing response to ACh in the form of increase ACh/PE ratio which was accompanied by significant increase in the aortic tissue nitrate denoting the vasorelaxing action of beta glycan and its possible role as vasodilator.

This was consistent with *Evans et al.*, (32) who reported that in healthy adult men a high-fiber diet containing beta-glycan resulted in a lowering of BP compared to a low fiber diet containing no beta-glucan. In addition, *Tabesh et al.*, (33) found

that oat cereals consumption for 4 weeks significantly increases the serum NO level and brachial artery diameters (vasodilatation).

Lee et al., (34) attributed this protective role to the ability of Beta glycan in inhibiting angiotensin-converting enzyme & upregulating corin gene expression. The major function of corin is to cleave pro-Atrial natriuretic peptide to Atrial natriuretic peptide, which has natriuretic activity, thereby lowering blood pressure. In addition, increased production of ANP in the liver of mice treated orally with β -glucans was demonstrated and that the urinary excretion of sodium was increased in mice treated with β -glycans presumably through the production of ANP which also affects blood vessels by promoting vasodilation.

β -glucans enhances nitric oxide production by the macrophage as well as increasing the expression of mRNA for iNOS (35). Moreover, β -glycan increased the excretion of vascular endothelial growth factor (VEGF), which induces endothelium-dependent vasodilation (36).

These findings are supported by the significant decrease in the plasma catalase activity and the significant increase in the plasma malondialdehyde level that greatly affected the nitric oxide level evidenced by its significant decrease in rats fed diet containing no beta-glucan

In 2008, *Varizi* (37) reported that oxidative stress is an important factor involved in the pathogenesis of hypertension and that it results in vascular dysfunction through its deleterious effects.

The decrease in the markers of oxidative stress points at the beneficial effects of beta glycan as defense against oxidative damage. Experimental data suggest that β -glucans also function as an effective free-radical scavenger and macrophages

selectively phagocytize and sequester glucan and a specific receptor on macrophage that binds to the beta glycan molecule has been identified(38)..

This highlights the antihypertensive and cardioprotective effects of beta-glucan limiting oxidative stress. Studies have also reported that the cardioprotective effects of bioactives (e.g. beta glycan) are due to their antioxidant and or anti-inflammatory activities (39).

The significant decrease in the plasma TAG, TC, LDL & AI which was accompanied by non significant increase in the plasma HDL highlights the anti-atherogenic effect of beta glycan rich diet which lowers incidence of development of atherosclerosis and its role in positively modulating the risk factor for dyslipidemia and reduction of heart diseases risk through decreasing plasma cholesterol level & its deposition in the wall of the blood vessels (39)

This result is consistent with *Wang et al.*, 2015 (40) who demonstrated the hypocholesterolemic effects of Beta glycans and its ability in reducing atherogenic indices through reducing the intestinal absorption of cholesterol and bile acids by binding to glycans and shifting the liver from cholesterol syntheses to bile acid production.

The binding of cholesterol with beta-glycan and the resulting elimination of these molecules in the feces is helps reduce blood cholesterol (41). The amelioration of blood lipid profile may be due to decreased absorption of bile acids that cause a removal of steroids from the body by fecal excretion resulting in increased catabolism of cholesterol, an increase in the secretion of bile acids, a decrease in lipoprotein cholesterol secretion, and a reduction in the total body pool of cholesterol (42)

From the current study, it can be concluded that adding appropriate amounts of oat cereals containing beta glycan to the diet especially for males is of extreme importance in preventing cardiovascular diseases as it decreases the blood pressure, decreases the oxidative stress & improves vascular relaxation through increasing the release of nitric oxide, in addition, β -glycan succeeded in lowering TC, TAG, LDL-C & atherogenic index

It is of great value to balance the atherogenic & hyperlipidemic effects of male androgens & to reverse the state of oxidative stress that can be induced by exogenous androgen supplementation. However, data for the effects of beta glycan on the regulation of cardiovascular diseases are still unclear and further studies are needed to corroborate its potential as a cardioprotective compound and greater understanding of the mechanisms of action of β -glycans. This could lead to targeting a more precise clinical objective as the mechanism of action would be established..

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