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Therapeutic effect of Quinoa Seeds (*Chenopodium Quinoa*) on obese, diabetic and obese diabetic male albino rats

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Abstract:

The current study was conducted to find out the potential therapeutic nutritional effects of quinoa seeds on ratsinflicted with obesity, diabetes and both. 50 adult white mice were used. The weight of the ratsaveraged (150 ± 5) grams. All rats werefed on the basic diet for a week, then were divided into 10 equal groups and one of them was left as a negative control group.Basal diet used also for control (+) groups. Otheras a obese, diabetic & obese / diabetic rats groups fed with quinoa seeds by proportions 5% and 7% . Obesity induced by feeding on 20% animal fat, while diabetes induced by alloxan injection 150 mg / kg of body weight into the peritoneum. The experiment lasted for 28 days. At the end of the experiment, the rats were weighed, sacrificed , and blood samples collected after 12 hours fasting. Then the serum was analyzed to estimate the level of glucose in the blood, The internal organs (liver, kidneys, spleen, lungs, and heart) were separated and weighed. Also, the body weight was estimated. Feed intake and feed efficiency ratio Were calculated. The results of this study have shown that eating quinoa seeds caused a reduction in the acquired weight, feed intake, feed efficiency ratio , and improved glucose,blood lipid levels, and kidney and liver functions. All the treatments showed improvements in the analysed parameters , and the best for the results recorded for quinoa for 7%. Therefore, quinoa may be used for obese and diabetic patients.

Key words: Quinoa seeds - alloxan - Obesity - Blood glucose .

Introduction:

Overweight/obesity is a growing public health problem throughout the world. Preventive measures to help individuals maintain energy balance and avoid excessive weight gain are critical, because of the cost and limited effectiveness of currently available treatments. Weight gain usually results from a combination of excessive caloric intake and inadequate physical inactivity. Effective population-level prevention requires consideration of the important role that physical, social, and economic forces play in shaping individual choices as school, workplace , and physical activity at the local level. There is an urgent need to identify and evaluate policy approaches that make the physical and social environment more conducive to a healthy lifestyle (**Eby and Conditz,2008).**

The prevalence of pediatric overweight and obesity, which is increasing worldwide, has tripled in the United States in the last 30 years, with the percentage of obese youth doubling in the last 20 years. Adolescent obesity is associated with both immediate and long-term health problems (e.g., hypertension, type2 diabetes and asthma) and psychosocial problems (e.g., social isolation, depression, disordered eating behaviors) such that adolescent obesity has implications for current and future generations health and longevity. Further, several comorbid symptoms, particularly binge eating and social problems, may exacerbate weight gain and fat mass over time and/or interfere with treatment and prevention outcomes. Individual, familial, peer, and neighborhood factors, in conjunction with the pervasive obesogenic environment, interact with one another to influence the emergence and maintenance of overweight. Prevention and treatment efforts that take a multilevel, socioecological approach to reducing overweight are clearly warranted to address such broad environmental risk factors. These programs require innovative approaches to teach adolescents the skills needed to overcome individual, interpersonal, and contextual risk factors and the skills to seek out positive sources of social support for healthy lifestyle behaviors in order to stem tracking of adolescent overweight, and related comorbid outcomes, into adulthood **(Sinton and Wilfley , 2011) .**

Quinoa *(Chenopodium quinoa Willd., Amaranthaceae)* is a grain-like, stress-tolerant food crop that has provided subsistence, nutrition, and medicine for Andean indigenous cultures for thousands of years.

Quinoa contains a high content of health-beneficial phytochemicals, including amino acids, fiber, polyunsaturated fatty acids, vitamins, minerals, saponins , phytosterols , phytoecdysteroids, phenolics , betalains, and glycine betaine . Over the past 2 decades, numerous food and nutraceutical products and processes have been developed from quinoa. Furthermore, 4 clinical studies have demonstrated that quinoa supplementation exerts significant, positive effects on metabolic, cardiovascular, and gastrointestinal health in humans.**Graf *et al.*, 2015**

The popularity of its seeds has increased in recent years due to the claims of health benefits and superfood qualities. Studies to date on the health benefits of quinoa have been restricted to animal models, and the results provide weak to moderate evidence to support improved plasma lipid profiles. The consumption of 50 g quinoa/d lowers serum TGs in overweight and obese participants and reduces the prevalence of MetS.**Diana *et al.*,2017**

Diabetes mellitus is a chronic disease that has affected humankind throught the world. The records of the ancient civilization of Egypt, Itndia, Japan, Greece and Rome describe the symptoms of the disease, and usually include recommendation for nutraceutical treatment. Diabetes is considered as a major healthy problem at ages in all world. It is a powerful and independent risk factor for cardiovascular disease, which remains the major cause of death in diabetic person. Diabetes mellitus is caused due to deficiency in production of insulin by the pancreas, or the ineffectiveness of the produced insulin. It is a global problem and number of those affected is increasing day by day**(Mukherjee *et al.*, 2006) .**

Despite quinoa's composition and properties, scientific evidence supporting health claims such as weight loss, antidiabetic effects, and appetite suppression in in vivo models is limited and the evidence is restricted to a few animal studies. Among this body of evidence, it can be observed that a possible health benefit of quinoa consumption may be linked to a potential lipid-lowering effect **(Takao *et al.*, 2005) .** Quinoa has a complete protein, one of the only plant foods offering all nine of the essential amino acids the human body needs in a healthy balance. Quinoa has high amounts of potassium, which helps control blood pressure, and is rich in antioxidants that may help to manage type two diabetes. Though tiny, quinoa has been found to help you feel fuller,

longer. “Quinoa is a low-glycemic-index carbohydrate since it’s very rich in fiber and protein , “This means you’ll feel fuller longer after consuming it, which may help you eat less over time.”

Despite all these attributes, quinoa is not widely used due to the high cost of the imported grain and little knowledge of its benefits by most consumers. More studies are needed to increase knowledge about this "pseudo cereal", to demonstrate its functional and nutritional benefits and to study its ant nutritional effects, since it presents high commercial value and excellent nutritional quality.

Material and Methods:

Materials:

Plants: The tested seeds in this investigation were quinoa seeds *(Chenopodium quinoa)* , these quinoa seeds were purchased from local market in Menoufia, Egypt and milled to obtain powder from it .

Animal: Normal male albino rats (50) of Sprague Dawley Strain obtained from the laboratory Animal Colony, Ministry of Health and Population, Helwan, Cairo, Egypt.

Chemicals: Alloxan were obtained from EL- Gomhouria company, Cairo, Egypt.

Diets: Diet consists of casein, sucrose, corn oil, choline chloride, vitamin mixture, mineral mixture, cellulose, and corn starch were purchased from El-Gomhouria Company for Drugs and Medical Equipments, Cairo, Egypt.

Experimental design:

Fifty (50) rats fed on standard diet for 7 days for acclimatization. Then, rats were randomly distributed into 10 equal groups, of 5 rats each. Group 1 was fed on the basal diet and set as a negative control group (normal rats). The groups 2,3,4 will be feeding on high fat diet (20% animal lipid), groups 5,6,7 were injected with alloxan to obtain the hyperglycemic rats and groups 8,9,10 obese rats will be induced by feeding on high fat diet(20% animal lipid) and injected with alloxan. All groups were fed for 4 weeks according to the following groups:

Group (1): (Control " – ") rats (n=5)were fed basal diet only.

Group (2):(Control " + ") rats (n=5) were keptwith out any treatment and fed on basal diet after fed on high fat diet (20% animal lipid)and used as a positive control group.

Group (3): (Quinoa seeds 5%) rats (n=5) were fed on basal diet containing 5% quinoa seeds after being fed on high fat diet (20% animal lipid)

Group (4): (Quinoa seeds 7%) rats (n=5) were fed on basal diet containing 7% quinoa seeds after being fed on high fat diet (20% animal lipid)

Group (5):(Control "+" of hyperglycemic rats), rats (n=5) were injected with alloxan to obtain the hyperglycemic rats.

Group (6): (Quinoa seeds 5%) rats (n=5) were fed on basal diet containing 5% quinoa seeds after injected with alloxan

Group (7): (Quinoa seeds 7%) rats (n=5) were fed on basal diet containing 7% quinoa seeds after injected with alloxan

Group (8): (Control " + " Obese hyperglycemic rats) rats (n=5). Obese rats will be induced by being fed on high fat diet (20% animal lipid) supplemented in the basal diet and used as a positive control group and injected with alloxan to obtain the hyperglycemic rats.

Group (9): (Quinoa seeds 5%) rats (n=5) will be fed on basal diet containing 5% quinoa seeds afterhyperglycemic rats induced post feeding on high fat diet (20% animal lipid).

Group (10):(Quinoa seeds 7%) rats (n=5) will be fed on basal diet containing 7% quinoa seeds after hyperglycemic rats induced post feeding on high fat diet (20% animal lipid).

Biological Evaluation:

During the experimental period (28days), the consumed diet was daily recorded (feed intake), biological evaluation of the different diets was carried out by determination of body weight gain (BWG) and feed efficiency ratio (FER) according to **Chapman *et al.*, (1959).**

Blood Sampling:

At the end of the experiment, rats were fasted overnight and anesthetized with diethyl ether. Blood samples were collected in clean dry centrifuge tubes from hepatic portal vein; serum obtained by centrifugation was carefully aspirated, transferred into clean cuvette tubes and stored frozen at -20°C for analysis **(Malhotra, 2003).**

Serum samples analyzed for determination the following parameters:

Serum glutamate oxaloacetate transaminase S.GOT or (AST) was determined as Unit/L according to**Yound (1975)**, S.GPT or (ALT) was determined as Unit/L according to **Yound (1975)**, serum alkaline phosphatase (ALP) was determined U/L according to **(IFCC, 1983)**,

total cholesterol was determined according to **Allain (1974),** enzymatic colorimetric determination of triglycerides was carried out according to **Fossati and Prencipe (1982),**determination of HDL was carried out according to the method of **Lopez (1977),** of LDL and VLDL carried out according to the method of **Friedwald and Levy (1972) ,**atherognic index (AI) was calculated as the VLDL + LDL cholesterol / HDL ratio according to the formula of**Nakabayashi *et al.*, (1995),** urea determination was according to the enzymatic method of **Malhotra (2003),** uric acid determination was according to the enzymatic colorimetric test of **Barham and Trinder (1972),**creatinine was measured using the modified kinetic method according to **Bartles *et al.*, (1972) and Larsen (1972)** the principle used of glucose determination was according to**Trinder (1959).**

Results and discussion:

A- Biological changes:

Results of body weight gain (BWG), feed intake (FI) and feed efficiency ratio (FER) of experimental rats are presented in table (1) .There were significant increases in BWG, FI and FER of obese rats compared to control (-) while there were significant decrease in BWG, FI and FER of all obese rats fed on 5% and 7% quinoa compared to control (+) . The best effect on BWG, FI, and FER showed by obese rats fed on 7%quinoa (group 4) when compared to control (+) group.

There were significant increases in BWG, FI and FER of diabetic rats compared to control (-) while there were significant decrease in BWG, FI and FER of all diabetic rats fed on 5% and 7% quinoa compared to control (+) .

The best effect on BWG, FI, and FER showed by diabetic rats fed on 7% quinoa (group 7) when compared to control (+) group.There were significant increases in BWG, FI and FER of obese diabetic rats compared to control (-) while there were significant decrease in BWG, FI and FER of all obese diabetic rats fed on 5% and 7% quinoa compared to control (+) .

The best effect on BWG, FI, and FER showed by obese diabetic rats fed on 7% quinoa (group 10) when compared to control (+) group. These results agree with **Abellán *et al.*, (2017)** who reported that processed quinoa intake during 28 days decreases BMI and HbA1c levels, maintains FPG levels, and increases the satiation and fullness (complete) degree in prediabetic patients. **Foucault *et al.*,**

(2014)demonstrated that a supplementation of a high-fat diet with a quinoa extract enriched in 20-hydroxyecdysone (QE) or pure 20-hydroxyecdysone (20E) could prevent the development of obese.

It could be noticed from table (1)that lowest BWG, regardless of significance , recorded for obese diabetic rats fed on quinoa diet 7%. This many indicate the value of quinoa for clinical nutrition . This was supported by finding that FI and FER values were lowest for obese diabetic group fed on quinoa diet 7% also. Nevertheless without quinoa diet control (+) group of obese diabetic rats was worst.

Table (1): Body weight gain (BWG), feed intake (FI) and feed efficiency ratio (FER) of normal, obese , diabetic and obese diabetic rats due to feeding on quinoa diet

Parameter / Groups		BWG(g/d) Mean ± SD	FI(g) Mean±SD	FER Mean± SD
control	**G1 (– ve)**	12.6^{h}±1	13^{g}±1.52	0.197^{b}±0.1
Obese	**G2 (+ Obese control)**	39.3^{b}± 2.2	17.66^{a}±2.08	0.488^{a}±0.048
	G3 Quinoa 5%	30.6^{c}± 0.8	16.5^{bc}± 2	0.321^{b}±0.1
	G4 Quinoa 7%	28^{d} ± 2	16.01^{cd}±1	0.30^{b}±0.1
Diabetic	**G5(+ Diabetic control)**	38.4^{b}± 0.8	17.33^{ab} ± 2.64	0.455^{a}±0.035
	G6 Quinoa 5%	27.3^{d} ± 0. 4	15.66^{cd} ± 1	0.294^{b} ± 0.011
	G7 Quinoa 7%	25.4^{e} ± 0.5	15.02^{de} ± 2	0.287^{b} ± 0.026
Obese Diabetic	**G8 (+ Obese Diabetic control)**	41.0^{a} ± 0.7	18^{a} ± 1.4	0.479^{a}± 0.016
	G9 (Quinoa 5%)	22.3^{f} ± 0.7	14.33^{ef} ± 1.6	0.247^{b} ± 0.15
	G10 (Quinoa 7%)	19.06^{g} ± 0.8	13.66^{fg} ± 2.2	0.208^{b} ± 0.13
	LSD	1.0464	0.9132	0.0861

Values with different column indicate significant differences Between the groups (P<0.05), and vice versa. LSD: least significant Differences (P<0.05).

As indicated by FER Results best group were that fed on 7% quinoa diet with no significant difference for obese, diabetic and obese diabetic rats.

B- Relative organs weight :

Data presented in table (2), show the relative organ weight (liver, heart, kidneys, spleen and lungs).There were significant decreases in heart, liver, kidney, lungs, spleen weight of obese rats compared to control (-) while there were significant decrease in heart, liver, kidney, lungs, spleen weight of all obese rats fed on 5% and 7% quinoa compared to control (+). The best effect on heart, liver, kidney, lungs, spleenweight showed by obese rats fed on 7% quinoa (group 4) when compared to control (+) group.

There were significant decreases in heart, liver, kidney, lunge, spleen weight of diabetic rats compared to control (-) while there were significant decreases in heart, liver, kidney, lunge, spleen weight of all obese rats fed on 5% and 7% quinoa compared to control (+) . The best effect on heart, liver, kidney, lunge, spleenweight showed by diabetic rats fed on 7% quinoa (group 7) when compared to control (+) group.

There were significant decreases in heart, liver, kidney, lunge, spleen weight of obese diabetic rats compared to control (-) while there were significant decrease in heart, liver, kidney, lunge, spleen weight of all diabetes obesity rats fed on 5% and 7% quinoa compared to control (+) . The best effect on heart, liver, kidney, lunge, spleen weight showed by obese diabetic rats fed on 7% quinoa (group 10) when compared to control (+) group.

The lowest organ weight in table 2 recoded for obese diabetic rats fed on 7% quinoa diet, which was also found for other biological parameters (Table 1) , regardless of that obese diabetic rats control (+) revealed highest values compared to obese &diabetic rats control (+) . This indicate the potent clinical nutrition action of quinoa 7% diet, being not dependent on control (+) level of same treatment.

Table (2): Organs weights of normal, obese, diabetic and obese diabetic rats due to feeding on quinoa diet

Parameter / Groups		Heart (g) Mean ± SD	Liver (g) Mean ± SD	Kidney (g) Mean ± SD	Lungs (g) Mean ± SD	Spleen (g) Mean ± SD
Negative	**G1 (– ve)**	0.21 ^{c}± 0.1	3.2 ^{d}± 0.25	0.75 ^{a}± 0.08	0.26^{b}±0.16	0.14 ^{c}± 0.02
Obese	**G2 (+ Obese control)**	0.43^{ab}± 0.2	4.45 ^{a}± 0.13	1.08 ^{a}± 0.6	0.57 ^{a}±0.15	0.4 ^{a}± 0.1
	G3 Quinoa 5%	0.4 ^{ab}± 0.1	4.00 ^{b}± 0.36	1.02 ^{b}±0.6	0.54^{ab}±0.47	0.33^{b}±0.04
	G4 Quinoa 7%	0.39 ^{ab}± 0.21	3.68^{bc}± 0.2	1 ^{a} ± 0.5	0.51^{ab}± 0.13	0.29 ^{ab}± 0.09
Diabetic	**G5 (+ Diabetic control)**	0.41^{ab}± 0.2	4.40 ^{a} ± 0.49	1.11 ^{a}± 0.3	0.55^{ab}±0.18	0.39 ^{a}± 0.09
	G6 Quinoa 5%	0.34^{abc} ± 0. 2	3.90 ^{b} ± 0.35	0.95 ^{a} ± 0.3	0.50^{ab} ± 0.23	0.30 ^{ab}± 0.04
	G7 Quinoa 7%	0.33^{abc} ± 0.1	3.72^{bc} ± 0.59	0.88 ^{a} ± 0.2	0.48 ^{ab} ± 0.2	0.27 ^{ab}± 0.1
Obese Diabetic control (+)	**G8 (+ Obese Diabetic control)**	0.45 ^{a} ± 0.1	4.58 ^{a} ± 0.41	1.04 ^{a} ± 0.3	0.56^{ab}± 0.08	0.41 ^{a}± 0.2
	G9 (Quinoa 5%)	0.35 ^{abc} ± 0.25	3.52 ^{cd} ± 0.26	0.92 ^{a} ± 0.3	0.38^{ab} ± 0.15	0.29 ^{ab}± 0.1
	G10 (Quinoa 7%)	0.27 ^{bc} ± 0.1	3.41 ^{cd} ± 0.62	0.82 ^{b} ± 0.1	0.30 ^{b} ± 0.2	0.22 ^{ab}± 0.05
	LSD	0.1042	0.2802	0.3186	0.1806	0.0873

Values with different column indicate significant differences Between the groups (P<0.05), and vice versa. LSD: least significant Differences (P<0.05).

C- Biochemical data changes:

1-Lipids fraction of serum:

Data presented in table (3), show the effect on serum lipids fractions .There were significant increases in Total cholesterol , Triglycerides, LDL, VLDL, VLDL+LDL/ HDL (mg\dl) of obese rats compared to control (-) while there were significant increase in Total cholesterol , Triglycerides , LDL, VLDL, VLDL+LDL/ HDL (mg\dl) of all obese rats fed on 5% and 7% Quinoa compared to control (+) .The

best effect on Total cholesterol , Triglycerides , LDL, VLDL, VLDL+LDL/ HDL (mg\dl) showed by obese rats fed on 7% quinoagroup 4 when compared to control (+) group.

-There were significant decreases in HDL (mg\dl) of obese rats compared to control (-) while there were significant increase in HDL (mg\dl) of all obese rats fed on 5% and 7% quinoacompared to control (+) .The best effect on HDL (mg\dl) showed by obese rats fed on 7% quinoagroup 4 when compared to control (+) group.

-There were significant increases in Total cholesterol , Triglycerides , LDL, VLDL, VLDL+LDL/ HDL (mg\dl) of diabetic rats compared to control (-) while there were significant decrease in Total cholesterol , Triglycerides , LDL, VLDL, VLDL+LDL/ HDL (mg\dl) of all diabetes rats fed on 5% and 7% quinoa compared to control (+) .The best effect on Total cholesterol , Triglycerides , LDL, VLDL, VLDL+LDL/ HDL (mg\dl) showed by diabetic rats fed on 7% quinoagroup 7 when compared to control (+) group. Diabetic rat seem to be of worse results than obese rats and the worst of allwas the group of obese diabetic rats .

-There were significant increases in Total cholesterol , Triglycerides , LDL, VLDL, VLDL+LDL/ HDL (mg\dl) of obesediabetic rats compared to control (-) while there were significant decrease in Total cholesterol , Triglycerides , LDL, VLDL, VLDL+LDL/ HDL (mg\dl) of all obese rats fed on 5% and 7% Quinoa compared to control (+) .The best effect on Total cholesterol , Triglycerides , LDL, VLDL, VLDL+LDL/ HDL (mg\dl) showed by obese diabetic rats fed on 7% quinoagroup 10 when compared to control (+) group.

- There were significant decreases in HDL (mg\dl) of obese diabetic rats compared to control (-) while there were significant increase in HDL (mg\dl)of all obese diabetic rats fed on 5% and 7% Quinoa compared to control (+) .The best effect on HDL (mg\dl) showed by obese diabetic rats fed on 7% quinoa(group 10) when compared to control (+) group.The consumption of 50 g quinoa/d lowers serum TGs in overweight and obese participants and reduces the prevalence of Mets as report of **Diana *et al.*,(2017).**

Table (3): Lipids fractions in serum of normal ,obese, diabetic , obese diabetic rats

Groups \ Parameter		TC (mg/dl) Mean ± SD	T G (mg/dl) Mean ± SD	VLDLc (mg/dl) Mean ± SD	HDLc (mg/dl) Mean ± SD	LDLc (mg/dl) Mean ± SD	AI Mean ± SD
Negative	**G1 (– ve)**	68^{h}±3.5	33^{j}±1.4	6.6^{f}± 0.5	57^{a}±1.5	4.4^{h}±1	0.19^{f}± 0.13
Obese	**G2 (+ Obese control)**	90^{c}±2.5	50^{d}±1.5	10^{d}± 0.7	40^{h}±1.2	40^{c}±2.3	1.25^{c}± 0.3
	G3 Quinoa 5%	75^{f}±3	40^{h}± 0.9	8^{e}±1	50^{d}±0.74	17^{f}±1.6	0.5^{efg}± 0.1
	G4 Quinoa 7%	71^{f}±2.08	36^{i}±2.4	7.2^{f}±1.2	54^{b} ±1	9.8^{g}±2.1	0.31^{fg}± 0.08
Diabetic	**G5 (+ Diabetic control)**	95^{g}±2.51	56^{c} ± 2.9	11.2^{c}± 0.9	36^{i}± 0.86	47.8^{b}±3.1	1.64^{b}± 0.2
	G6 Quinoa 5%	81^{d} ± 4.04	44^{f} ± 3	8.8^{e} ± 1.1	48^{e} ± 1.4	24.2^{e}±2.9	0.68^{def}± 0.09
	G7 Quinoa 7%	78.4^{e} ± 4.1	42^{g} ± 1.9	8.4^{e} ± 0.6	52^{c} ± 2	18^{f}± 0.5	0.50^{efg}± 0.07
Obese Diabetic	**G8 (+ Obese Diabetic control)**	105^{a} ± 6.2	66^{a} ± 2	13.2^{a} ± 0.4	32^{j}± 0.79	59.8^{a}± 1.2	2.3^{a}± 0.6
	G9 (Quinoa 5%)	91^{c} ± 4	60^{b} ± 2.6	12^{b} ± 2	46^{g} ± 0.9	33^{d}±1.8	0.98^{cd}± 0.09
	G10 (Quinoa 7%)	83^{d} ± 1	53^{d} ± 3.1	10.6^{cd} ± 0.8	47^{f} ± 1.3	25.4^{e}± 2	0.76^{de}± 0.15
	LSD	2.4433	1.2888	0.78733	- 0.6610	1.3988	0.2797

Values with different column indicate significant differences Between the groups (P<0.05), and vice versa. LSD: least significant Differences (P<0.05).

2-Kidney function:

The results illustrated in table (4) show the serum creatinine (mg/dl), urea (mg/dl) &uric acid (mg/dl) of experimental rats. It could be noticed There were significant increases in urea, uric acid and creatinine of obese rats compared to control (-) while there were significant decrease in urea, uric acid and creatinine of all obese rats fed on 5% and

7% quinoa compared to control (+) . The best effect on urea, uric acid and creatinine showed by obese rats fed on 7% quinoa (group 4) when compared to control (+) group.

- There were significant decreases in urea, uric acid and creatinine of diabetic rats compared to control (-) while there were significant decrease in urea, uric acid and creatinine of all diabetic rats fed on 5% and 7% quinoa compared to control (+) . The best effect on urea, uric acid and creatinine showed by diabetes rats fed on 7% quinoa (group 7) when compared to control (+) group.
- There were significant increases in urea, uric acid and creatinine of obesediabetic rats compared to control (-) while there were significant decrease in urea, uric acid and creatinine of all obese diabetic rats fed on 5% and 7% quinoa compared to control (+) . The best effect on urea, uric acid and creatinine showed by obese diabetic rats fed on 7% quinoa (group 10) when compared to control (+) group.

Table (4): Kidney function ((creatinine (mg/dl), Urea (mg/dl)&uric acid (mg/dl)) in serum of normal, obese , diabetic and obese diabetic rats due to feeding on quinoa diet

Parameter / Groups		Urea (mg/dl) Mean ± SD	Uric acid (mg/dl) Mean ± SD	Creatinine (mg/dl) Mean ± SD
Negative	**G1 (– ve)**	24 ^{i} ± 1.8	1.7 ^{e} ± 0.2	0.59 ^{i} ± 0.081
Obese	**G2 (+ Obese control)**	44.5^{bc} ± 2.5	3.9 ^{b} ± 0.3	0.99 ^{a} ± 0.068
	G3 Quinoa 5%	35 ^{e} ± 3	2.9 ^{cd} ± 0.5	0.87 ^{d} ±0.031
	G4 Quinoa 7%	27 ^{h} ± 3.4	2 ^{de} ± 0.5	0.76 ^{f} ± 0.015
Diabetic	**G5 (+ Diabetic control)**	46 ^{b} ± 1	4.5 ^{ab} ± 0.1	0.91 ^{c} ± 0.041
	G6 Quinoa 5%	38 ^{d} ± 2.1	3.6 ^{bc} ± 0.8	0.81 ^{e} ± 0.047
	G7 Quinoa 7%	32 ^{f} ± 4.1	2.6 ^{de} ± 1.7	0.72 ^{g} ± 0.021
Obese Diabetic	**G8 (+ Obese Diabetic control)**	50 ^{a} ± 2	5.2 ^{a} ± 0.6	0.95 ^{b} ± 0.024
	G9 (Quinoa 5%)	43 ^{c} ± 1.4	4.2 ^{b} ± 0.7	0.78^{ef} ± 0.029
	G10 (Quinoa 7%)	30 ^{g} ± 1.5	2.3 ^{de} ± 0.9	0.68 ^{h} ± 0.053
	LSD	1.6666	0.7800	0.0366

Values with different column indicate significant differences Between the groups (P<0.05), and vice versa. LSD: least significant Differences (P<0.05).

Never the less difference as calculated for the 3parameters were not markedconsidering diabetic and obese diabetic rats.

3- Liver enzymesactivities:

Data presented in table (5), show the effect on AST, ALT and ALP.There were significant increases in (AST or Got) in these

parameters of obese rats compared to control (-) while there were significant decreases in all obese rats fed on 5% and 7% quinoa compared to control (+) . The best effect showed by obese rats fed on 7% quinoa (group 4) when compared to control (+) group.

- There were significant increases in AST , ALTand ALP of diabetic rats compared to control (-) while there were significant decreasesin all diabetic rats fed on 5% and 7% quinoa compared to control (+) . The best effectshowed by diabetic rats fed on 7% quinoa (group 7) when compared to control (+) group.
- There were significant increases in AST, ALT and ALP of obese diabetic rats compared to control (-) while there were significant decrease in all obese diabetic rats fed on 5% and 7% quinoa compared to control (+) . The best effectshowed by obese rats fed on 7% quinoa (group 4) when compared to control (+) group. Similar results obtained by **Sneha *et al.*,(2017)**who concluded that the Quinoa seeds*(Chenopodium quinoa)*showed hepato protective effects against CC14- induced damage in Swiss albino male mice.

Table (5): Serum activity of AST, ALT and ALP ratioof normal, obese , diabetic and obese diabetic rats due to feeding on quinoa diet

Parameter / Groups		AST (U/L) Mean ± SD	ALT (U/L) Mean ± SD	ALP(U/L) Mean ± SD
Negative	**G1 (– ve)**	35 ^{j}± 1.4	26 ^{j}± 2	135 ^{i}± 0.7
Obese	**G2 (+ Obese control)**	75 ^{f}± 1.9	60 ^{a}± 2.5	173 ^{c}± 3
	G3 Quinoa 5%	70 ^{g}± .5	45 ^{f}± 1.5	168 ^{d}±1
	G4 Quinoa 7%	41 ^{l}± 2.2	29 ^{l}± 1.4	158 ^{f}± 2.5
Diabetic	**G5 (+ Diabetic control)**	95 ^{c}± 3	54 ^{c} ± 1.6	179 ^{b}± 3
	G6 Quinoa 5%	88 ^{d} ± 2	52 ^{d} ± 2.1	163 ^{e} ± 1.4
	G7 Quinoa 7%	61 ^{h} ± 1.2	38 ^{h} ± 2.7	152 ^{g} ± 5
Obese Diabetic	**G8 (+ Obese Diabetic control)**	107 ^{a} ± 2.5	57 ^{b} ± 0.5	186 ^{a} ± 4
	G9 (Quinoa 5%)	102 ^{b} ± 3.3	47 ^{e} ± 1.4	162 ^{e} ± 1
	G10 (Quinoa 7%)	85 ^{e} ± 1	40 ^{g} ± 2.8	146 ^{n} ± 2
	LSD	1.5278	1.2190	2.4261

Values with different column indicate significant differences Between the groups (P<0.05), and vice versa. LSD: least significant Differences (P<0.05).

4-Glucose (GLU) :

Data presented in table (5), show the effect on glucose . There were significant increases in glucose of obese rats compared to control (-) while there were significant decreases in glucose of all obese rats fed on

5% and 7% quinoa compared to control (+) . The best effect on glucose showed by obese rats fed on 7% quinoa (group 4) when compared to control (+) group.

-There were significant increases in glucose of diabetic rats compared to control (-) while there were significant decrease in glucose of all diabetic rats fed on 5% and 7% quinoa compared to control (+) . The best effect on glucose showed by diabetic rats fed on 7% quinoa (group 7) when compared to control (+) group.

- There were significant increases in glucose of obese diabetic rats compared to control (-) while there were significant decrease in glucose of all obese diabetic rats fed on 5% and 7% quinoa compared to control (+) . The best effect on glucose showed by obese diabetic rats fed on 7% quinoa (group 10) when compared to control (+) group.Similar results obtained by **Liangkui *et al.*,(2018)** showing that daily consumption of quinoa in this-term intervention appears to modify glucose response.

Table (6): Glucose level of normal, obese, diabetic and obese diabetic rats due to feeding on quinoa diet

Groups \ Parameter		Glucose Mean ± SD
Negative	**G1 (– ve)**	45^{j}±1
Obese	**G2 (+ Obese control)**	102^{c}±1
	G3 Quinoa 5%	59^{g}±2
	G4 Quinoa 7%	54^{h}±1
Diabetic	**G5 (+ Diabetic control)**	180^{b}± 3
	G6 Quinoa 5%	69^{e} ± 1
	G7 Quinoa 7%	64^{f} ± 2
Obese Diabetic	**G8 (+ Obese Diabetic control)**	189^{a} ± 1
	G9 (Quinoa 5%)	76^{d} ± 1
	G10 (Quinoa 7%)	50^{i} ± 2
	LSD	1.2129

Values with different column indicate significant differences Between the groups (P<0.05), and vice versa. LSD: least significant Differences (P<0.05).

It is clear that quinoa seed diets improved the biological & biochemical parameters of obese and obese diabetic rats (Tables 1-6).

Reference

Allain, C.C. (1974): Cholesterol enzymatic colorimetric method. J. of Clin. Chem., 20 : 470.

Abellán, R.; Barnuevo, E.; García, S,; Contreras, F.; Aldeguer, G.; Soto M.; Guillén, G.; Luque, R.; Quinde, R.; Martínez, G, and López, R. (2017) : Effect of quinoa *(Chenopodium quinoa)*consumption as a coadjuvant in nutritional intervention in prediabetic subjects . Nutr Hosp. 34(5):1163-1169.

Barham, D.andTrinder, P. (1972): Determination of uric acid. Analyst, 97: 142.

Bartles, J.; Bohmer, M. and Heirli, C.(1972): Determination of creatinine. Clin. Chem.Acta, 37:193.

Chapman, D.G.; Castilla, R. and Champbell, J.A. (1959): Evaluation of protein in food. I.A. Method for the determination of protein efficiency ratio.Can. J. Biochemistry. Physiology, 37: 679-686.

Diana N.; Jessica R.; Audrey T. and Markandeya J (2017): Quinoa seed lowers serum triglycerides in overweight and obese subjects: A dose-response randomized controlled clinical trial. Curr. Dev.Nutr. 2017 Sep; 1(9): e001321. doi: 10.3945/cdn.117.001321

Eby, J.G. and Colditz, G.A. (2008) : Obesity / Overweight : Prevention and weight management.

Friedwald, W.T. and Levy, R.I. (1972) : Estimation of the concentration of low density lipoprotein separated by three different methods. Clinchem., 18 : 499- 50.

Fossati, P. and Prencipe, L. (1982): Triglyceride enzymatic colorimetric method. J. of Clin. Chem., (28): 2077.

Foucault, A.; Even, P.; Lafont, R.; Dioh, W.; Veillet, S .; Tomé, D.;Huneau, J.F2;Hermier, D2, Quignard,B. A. and Physiol, B. (2014): Quinoa extract enriched in 20-hydroxyecdysone affects energy homeostasis and intestinal fat absorption in mice fed a high-fat diet.128:226-31. doi: 10.1016/j.physbeh.

Graf, B.; Rojas, S.; Rojo, L.; Delatorre, H.; Baldeón, M. and Raskin, I. (2015): Innovations in Health Value and Functional Food Development of Quinoa *(Chenopodium quinoa Willd)*. ComprRev Food Sci Food Saf. 14(4):431-445. Epub 2015 Apr 10.

IFCC (1983) : Methods for the Measurement of Catalytic Concentration of Enzymes, Part 5: IFCC, methods for alkaline phosphatase. J.Clin. Chem.Clin. Biochem., 21 : 731-748.

Liangkui, L.; Georg, L.; Wendym B.; Anthony, W.; Ben, M. and Chris, S. (2018) : Effects of Quinoa *(Chenopodium quinoa Willd.)* Consumption on Markers of CVD Risk. Nutrients.10(6): 777.

Lopez, M.F. (1977): HDL- cholesterol colorimetric method. J. of Clin. Chem., 230: 882

Malhotra, V.K.(2003): Practical Biochemistry for student .Fourth edition, Jaypee Brothers Medical Publishers (P) LTD, NEW Delhi.

Mukherjee, P.K.; Maiti, K.; Mukherjee, K. and Houghton, P.J. (2006): Leads from Indian medicinal plants with hypoglycemic potentials . J. of Ethnopharmacol., 106(1): 1-28.

Nakabayashi, A.; Kitagawa, Y.; Suwa, Y.; Akimoto, K. et al., (1995): Tocopherol enhances the hypocholeserolemic action of sesames in rats. Internat. J. Vit. Nutr. Res., 65 (3): 162-168.

Sinton, M.M. and Wlfley, D.E. (2011) : Overweight / Obesity.http:// dx

Sneha, S.; Lata, S. and Pradeep, B. (2017) : Hepatoprotective effect of *Chenopodium quinoa* seed against CCL4-induced liver toxicity in Swiss albino male mice . Article (PDF Available) in Asian Journal of Pharmaceutical and Clinical Research, 10(11):273 ·2019 Feb; 11(2): 395.

Takao, T.; Nakamichi, W.; Kana, Y.; Syoko, I.; Saori, S.; Yukari, T.; Kenichi, N. and Yotaro, K. (2005) : Hypocholesterolemic effect of protein isolated from quinoa *(Chenopodium quinoa Willd)* seeds. Food SciTechnol Res 2005;11:161–7.

Trinder, p.(1959): Determination of phenazoneUsing 4-amino phenazone. J.Clin.Path., 2:246.

Yound, D.S. (1975): Determination ofGOT. Chem., 22 (5) : 1-21.

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التاثير العلاجى لبذور الكينوا على ذكور الفئران البيضاء المصابة بالسمنة و المصابه بالسكر و المصابة بالسمنة و السكر معا

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المستخلص العربي:

تم اجراء الدراسة الحالية لمعرفة التأثيرات التغذوية العلاجية المحتملة لبذور الكينوا على الفئران المصابه بالسمنه و السكر و الاتنين معا. تم استخدام 50 فار ابيض بالغ يتراوح وزن الفار (150± 5) جرام ، تم تغذيتها على الوجبة الأساسية لمدة أسبوع ثم تم تقسيمهم الى 10 مجاميع متساوية و تركت احداهما كمجموعة ضابطة سالبة و كذلك استخدام الغذاء الأساسى للمجموعات الضابطة الموجبة .الفئران المصابه بالسمنة و السكرى و السمنة / سكرى تمت تغذيتها على بذور الكينوا بنسبه 5%، 7% و تمت الأصابة بمرض السمنه عن طريق تغذية الفئران على20 % دهن حيوانى .كما تم الاصابة فئران سمينة و غير سمينة بالسكرى عن طريق الحقن بواسطة الالوكسان 150 ملجم / كجم من وزن الجسم داخل الغشاء البريتونى .

استمرت التجربة لمدة 28 يوم وفى نهاية التجربة تم وزن الفئران ثم تجميع عينات الدم بعد صيام 12 ساعة و فصل السيرم لتقدير مستوى الجلوكوز في الدم ،ثم تم فصل الأعضاء الداخلية (الكبد والكلى والطحال والرئتين والقلب)ووزنها وأيضا تم تقدير وزن الجسم المكتسب، والمأخوذ من الغذاء ومؤشر الاستفادة من الغذاء. وقد أظهرت نتائج هذه الدراسة أن بذور الكينوا قد تسببت فى تقليل فى الوزن المكتسب ، الماخوذ الغذائى ، نسبة كفاءة التغذية و قد تحسن مستوى الجلوكوز و دهون الدم ، و قد نتج عنه أيضا تحسن وظائف الكلى و الكبد . و قد اظهرت كل المعاملات تحسن فى العوامل المدروسة و كانت افضل مجموعة هى كينوا 7% وعلى ذلك فانه قد يمكن استخدام الكينوا كغذاء لمريض السمنه و السكرى.

الكلمات الافتتاحية : بذور الكينوا – الالوكسان – السمنة – جلوكوز الدم.