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ON HYPERLIPIDEMIC RATS**

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Abstract:

The purpose of this study was to investigate the effects of quinoa seeds on hyperlipidemic rats. Eighteen male rats weighing (126 ± 10 g) were randomly divided into three groups (6 each). The first group served as a negative control group (-ve) fed on a basal diet, the second group fed on a basal diet with 1.5 % cholesterol for 21 days and served as an untreated hyperlipidemic group (+ve) and the third group was hyperlipidemic group fed on a basal diet with 10% quinoa seeds for 30 days. The experiment lasted for 60 days, during which food intake and rat's weight were recorded to get nutritious parameters. Blood samples were collected to assays some kidney and liver functions, lipid profile and Troponin T levels. Also, the histopathological examination changes in cardiac tissues and aorty of heart. The Study results showed that the untreated hyperlipidemic group (+ve) had a significant increase and imbalance in body weight gain and the levels of some kidney and liver functions, lipid profile and Troponin T when comparing to the negative control group (-ve). While, hyperlipidemic rats feed on quinoa seeds had a significant decrease and improvement in levels in body weight gain and the levels of some kidney and liver functions, lipid profile and Troponin T comparing to untreated hyperlipidemic group (+ve). Also, the histopathological examination showed positive effect in hyperlipidemic group treated with quinoa seeds. We recommend consuming quinoa seeds with diets because they play a significantly role in the treatment of hyperlipidemia.

Keywords: Quinoa seeds, *Chenopodium quinoa* Willd., Lipid profile, Hypercholesterolemia, Triglycerides, Troponin and Rats

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INTRODUCTION:

Hyperlipidemic one of the biggest risk factors for the occurrence and seriousness of coronary heart disease globally. Hyperlipidemic is a state that incorporates many genetic and acquired disorders that illustrate elevated lipid levels within the human body (**Stewart *et al.*, 2020** and **Abdel-Wahhab *et al.*, 2021**). The abnormal consumption of energy dense diets such as high-fat diets with decreased of physical activities is considered to be the leading cause of obesity in human being. Obesity is associated with several phenotypic and metabolic changes, including insulin resistance, low-grade inflammation, hyperglycemia, hyperleptinemia, hyperinsulinemia, hyperlipidemia, systemic inflammation and hepatic steatosis (**Apovian, 2016** and **Alkhudhayri *et al.*, 2021**).

Cholesterol level is influenced by a number of factors, including genetics, a diet heavy in saturated fat, and different metabolic disorders. The relationship between total cholesterol's risk factor levels and the risk factor disease, it was chosen over other potential blood lipid-related measures of risk, such as high density lipoprotein (HDL), low density lipoprotein (LDL) and very low density lipoprotein (VLDL) (**Lawes *et al.*, 2006**).

Quinoa (*Chenopodium quinoa* Willd.) is a highly nutritious grain that has a high proportion of other vital elements like lipids, Fiber, vitamins, and minerals. It is also one of the few plants that has all the amino acids required by humans (**Dakhili *et al.*, 2019** and **Navruz-Varli & Sanlier, 2016**). It also have a variety of bioactive substances that have been shown to improve the skin, immunological system, and neurological system, such as saponins, phenolics, phytosterols, and bioactive peptides (**Dakhili *et al.*, 2019**). Additionally, these substances may protect consumers from cancer, diabetes, and obesity. People worldwide are becoming more interested in foods high in nutrients as they become more health conscious, and over the past few decades, quinoa has seen a sharp increase in demand (**Schmidt *et al.*, 2023**). This study aimed to assess the potential impact of quinoa seeds on Hyperlipidemic rats.

MATERIALS AND METHODS:

A-Materials:

Cholesterol powder was purchased from El-Gomhouria Company (for trading Drugs, Chemicals, and Medical Appliances), Mansoura city branch, El-Dakahlia Governorate, Egypt. Seeds of Quinoa (*Chenopodium quinoa Willd.*) were obtained from Agriculture Research Center, El-Giza Governorate, Egypt. Eighteen healthy adult male albino rats (Sprague–Dawley strain) weighing (126 ± 10 g) were purchased from the Agricultural Research Center, El-Giza Governorate, Egypt. Rats were fed on basal diet and were given ad-libitum access to fresh and clean water. The basal diet was prepared according to **NRC (1995)** as the following: 20% Casein, 49.7% Corn starch, 10% Sugar, 3% Cellulose, 5% Corn oil, 10% mineral admixtures, 2% vitamin admixtures, 0.3% DL-methionine.

B-Methods:

Preparation of seeds powder:

300 g of quinoa seeds were carefully inspected to remove any impurities, and then soaked in distilled water (2 L) for 72 h. During soak, quinoa seeds were rinsed twice a day with 400 ml of distilled water. After soak, samples were washed carefully with distilled water and then dried at 60°C. Dried samples were ground to pass through a 60-mesh sieve using an analytical mill according to **Park & Morita (2005)**.

Experimental animal design:

Rats were kept under observation for 7 days for adaptation and were given ad-libitum access to fresh and clean water and fed on basal diet according to **NRC (1992)**. After adaptation period rats were subdivided randomly into three groups (6 each), one served as a negative control group (-ve) and two groups served as rat models of Hyperlipidemic which fed on a basal diet with 1.5 % cholesterol for 21 days. After the end of this period, the Hyperlipidemic rats were re-divided as the following: positive control group (+ve) which served as untreated Hyperlipidemic rats group and quinoa seeds group which fed on a basal diet with 10% quinoa seeds for 30 days. All the biological experimental procedures were applied in accordance

with internationally guidelines for the care and use of laboratory animals. Ethical guidelines were maintained during animal handling and permission was obtained from the Research Ethics Committee at the Faculty of Specific Education, Mansoura University, under animal protocol code No (R/11).

Chemical analysis of Quinoa seeds:

Total ash content was determined according to **Thiex et al., (2012)**, Fat was estimated according to **Lee et al., (2005)**, Fiber was determined according to **Thiex et al., (2012)**, Total carbohydrate was determined according to **Gul & Safdar (2009)**, Protein and moisture Content was determined according to **AOAC (1990)**. These analyses were conducted at the Agricultural Research Center, Mansoura city branch, El-Dakahlia Governorate, Egypt.

Phytochemical analysis of Quinoa seeds:

The phytochemical analysis of quinoa seeds was detected by standard method of **Savithramma et al., (2011)**, and was conducted at the Agricultural Research Center, Mansoura city branch, El-Dakahlia Governorate, Egypt.

Nutritional Parameters:

During experiment period food intake by rats was recorded every day, while weightiness was measured once a week to record the weight gained. When the experimental period over the following equations were used to determined Body weight gain and feed efficiency ratio (FER) according to **Chapman et al., (1959)**:

$$\text{Body weight gain} = \frac{\text{Final weight (g)} - \text{Initial weight (g)}}{\text{Initial weight (g)}}$$

$$\text{Body weight gain \%} = \frac{\text{Final weight (g)} - \text{Initial weight (g)}}{\text{Initial weight (g)}} \times 100$$

$$\text{Feed efficiency ratio (FER)} = \text{Body weight gain (g)} / \text{Food intake (g)}$$

Biological analyses:

All biological analyses were performed in a private analysis laboratory in Mansoura city, El-Dakahlia, Egypt. Rats were scarified under

ether an anesthesia at the end of the experiment's blood samples were taken from the inner canthus of the rats' eyes, after 12 hours of fasting. According to **Drury & Wallington (1980)**, blood samples were received into clean, dry centrifuge tubes, allowed to clot at room temperature, and then spun at 5000 rpm for 10 min to extract serum. The samples were kept in a deep freezer at -18°C until they were used for biochemical analyses, using kits obtained from a laboratory kits company in Mansoura city, Dakahlia, Egypt. Aspartate aminotransferase (AST) and alanine aminotransferase (ALT) were measured according to the method described by **Tietz (1999)**, Serum Albumin was evaluation utilization the method of **DOUMAS *et al.*, (1972)**, Total Cholesterol (TC) was determined according to the method of **Allain *et al.*, (1974)**, Triglycerides (TG) were determined according to the method of **Fossati & Prencipe (1982)**, High-Density Lipoprotein cholesterol (HDL): was gauged according to the method of **Lopes-Virella *et al.*, (1977)**, Low-Density Lipoprotein cholesterol (LDL): was measured using the following formula: $LDL = TC - (TG / 5) - HDL$. Very Low-Density Lipoprotein cholesterol (VLDL): was measured using the following formula: $VLDL = TG / 5$. Kidney function parameters were assessment using kits purchased from Diamond Biodiagnostic Company (Egypt), as the following: Serum Uric acid, Urea and Creatinine were determined according to the methods described by **Fossati *et al.*, (1980)**; **Rock *et al.*, (1987)** and **Young (2001)** respectively, Troponin level was determined using ELISA kits according to **Mair *et al.*, (1994)**.

Histopathological examination:

The examination was conducted in the Department of Pathology, Faculty of Medicine, Mansoura University. Heart was removed and washed in saline solution, weighted and kept in formalin solution (10%, v/v) according to methods described by **Drury & Wallington (1980)**. Cardiac tissues and aorty of heart samples from the sacrificed rats' autopsies were taken. Samples were fixed in 10% formalin saline solution for 10 hours, according to the procedure of **Banchroft *et al.*, (1996)**.

Statistical analysis:

Statistical analysis was reported as mean and standard deviation (mean ± S.D.) and statistically analyzed using univariate analysis of variance (ANOVA). Means between groups were compared using the least significant difference (LSD) and Duncan^a statistical tests according to **Abu-Bader (2011)**.

RESULTS AND DISCUSSION:

Chemical composition of Quinoa seeds:

Chemical composition (Protein, carbohydrate, fat, fiber, Moisture and ash content) of quinoa seeds were demonstrated in Table 1 Our results revealed that quinoa seeds content of Protein, carbohydrate, fat, fiber, Moisture and ash were 14.97%, 65.78%, 6.09%, 8.55%, 10.67% and 2.5%, respectively. Our results are in accordance with **Bhargava et al., (2006)** who reported that the fat level of quinoa was 6.31 %. Also, **Alvarez-Jubete et al., (2010)** stated that quinoa typically has higher levels of crude protein, ash, and crude fat than conventional cereals like wheat. According to **Nascimento et al., (2014)** the values of protein, ash, fiber and fat in quinoa seeds were 12.1, 2.01, 10.4 and 6.3 % respectively. Also, **Demir & Kilinc (2017)** discovered that adding quinoa flour enhanced the amount of ash, crude protein, and crude fat. While, **Johnson & Wallace (2019)** said that quinoa seeds has greater dietary fiber content (8–13%) than maize and rice.

Table 1: Chemical composition of Quinoa seeds:

Chemical composition (%)	Quinoa seeds
Protein	14.97 ±0.36
Carbohydrate	65.78 ±3.02
Fat	6.09 ±0.89
Fiber	8.55 ±0.47
Moisture	10.67 ±2.4
Ash	2.5 ±0.09

Results were shown as estimates (mean±SD)

Phytochemical analysis of Quinoa seeds:

The data in table 2 showed the phytochemical analysis of quinoa seeds. The data revealed the presence of flavonoids, Tannis, saponins and steroides. According to **Gawlik-Dziki *et al.*, (2013)**; **Pathan & Siddiqui (2022)** and **Alamri *et al.*, (2023)** quinoa seeds consist of enlarging quantities of bioactive components, which provides health advantages beyond their nutritional value. Polyphenols, carotenoids, tocols, phytoecdysteroids, phytosterols, Saponins, peptides, tannins and phytic acid are examples of significant bioactive chemicals components in quinoa. The chemopreventive properties of quinoa seeds may help lower oxidative stress, prevent heart disease and other illnesses because the seeds have antioxidant, anti-inflammatory, and anticarcinogenic effects. While **Lin *et al.*, (2019)** reported that the phytochemicals found in quinoa are many and varied, and they have potential to mitigate the metabolic problems associated with diabetes and obesity.

Table 2: phytochemical analysis of Quinoa seeds:

Bioactive compound	Quinoa seeds
Flavonoid	+++
Tannins	+++
Alkaloids	++
Steroids	+
Saponin	+++

+++ Strong intensity reaction, ++ Medium intensity, + Weak intensity reaction, + trace, – Non detected

Nutritive effects in negative control group (-ve) and Hyperlipidemic rat’s groups which feed on the basal diet and Quinoa seeds:

The data in table 3 showed the initial weight, final weight, body weight gain, body weight gain percent, food intake and Feed efficiency ratio (FER) from the negative control group (-ve) and Hyperlipidemic rat’s groups which feed on the basal diet and Quinoa seeds. At the end of experiment final weight of rats group feeding on basal diet was 212.5 (g). While the final body weight of Hyperlipidemic rats (+ve) were 241.25 (g). Also, final weight of rats feeding on Quinoa seeds was 225.75 (g).

Hypercholesterolemia caused a significant increase in body weight gain, food intake and FER in positive control group (+ve) comparing to the negative control group (-ve). On the other hand, Hyperlipidemic rats feed on Quinoa seeds cause a significant decrease in body weight gain, food intake and FER in positive control (+ve) comparing to the negative control group (-ve). According to **Simnadis *et al.*, (2015)** and **Ali (2019)** the functional ingredients responsible for the effect on weight gain are thought to be the phenolic compounds, saponins, 20HE, and protein in quinoa. While, **Alghamdi (2018)** stated that Quinoa has been shown in both in vivo and human studies to have variable impacts on weight gain. Research has shown that quinoa eating significantly reduces body weight gain (BWG) in rats on a high-cholesterol diet.

Table 3. Nutritive effects in negative control group (-ve) and Hyperlipidemic rat's groups which feed on the basal diet and Quinoa seeds:

Parameter		Initial Weight (g)	Final Weight (g)	weight gain (%)	Body Weight daily	Food intake (g)	FER
Untreated	Negative Control (-ve)	127 ^a ±7.72	215 ^{bc} ±5.2	69.86 ^b ±10.84	1.47 ^c ±0.06	52.37 ^a ±5.3	0.16 ^b ±0.08
	Positive Control (+ve)	125 ^a ±2.5	241 ^a ±5.56	42.8 ^c ±4.1	1.91 ^a ±0.05	42.8 ^b ±7.5	0.25 ^a ±0.03
treated	Quinoa seeds	125 ^a ±9.27	226 ^b ±8.1	81 ^a ±14.95	1.74 ^b ±0.08	46.29 ^{ab} ±4.2	0.21 ^{ab} ±0.02

Results were shown as estimates (mean±SD) in each column having different combinations of superscripts (a, b, c, d...).

Biological analyzes:

1- Lipids profile [total cholesterol (TC), total triglycerides (TG), high density lipoprotein (HDL), low density lipoprotein (LDL) and very low density lipoprotein (VLDL)] in negative control group (-ve) and Hyperlipidemic rat's groups which feed on the basal diet and Quinoa seeds:

Data in Table 4 showed a significant increase in TC, TG, LDL and VLDL and decrease in HDL in positive control group (+ve) compared to the

negative control group (-ve). The Hyperlipidemic rat groups fed on quinoa seeds recorded a significant decrease in TC, TG, LDL and VLDL (68.19, 111.83, 11.27 and 22.37 mg/dL, respectively) and increase in HDL (28.88 mg/dL) compared to the negative control group (-ve). Our results are in accordance with **TAKAO *et al.*, (2005)** who found that the possible mechanism for quinoa effect may be based on bile acid activity. It was demonstrated that quinoa proteins have a greater ability to bind bile acids, which influences the absorption of fats. **Graf *et al.*, (2015)** stated that the hypocholesterolemic effect of Quinoa due to its content of saponins and the fiber. Also, **Navarro-Perez *et al.*, (2017)** study showed that the consumption of 50 g quinoa reduced serum Triglycerides which might help reduce the risk of cardiovascular disease.

Table 4: Lipids profile [total cholesterol (TC), total triglycerides (TG), high density lipoprotein (HDL), low density lipoprotein (LDL) and very low density lipoprotein (VLDL)] in negative control group (-ve) and Hyperlipidemic rat's groups which feed on the basal diet and Quinoa seeds:

Parameter		TC	TG	HDL	LDL	VLDL
Rats Group		(mg/dL)	(mg/dL)	(mg/dL)	(mg/dL)	(mg/dL)
Untreated	Negative Control (-ve)	50.53 ^c ±0.49	51.26 ^c ±0.83	31.63 ^a ±0.62	8.45 ^c ±0.57	10.25 ^c ±0.17
	Positive Control (+ve)	156.98 ^a ±6.31	346.34 ^a ±2.59	20.73 ^c ±0.26	65.19 ^a ±0.09	69.27 ^a ±0.52
treated	Quinoa seeds	76.31 ^b ±0.54	191.41 ^b ±21.88	25.78 ^b ±0.19	34.53 ^b ±0.7	36.11 ^b ±1.95

Results were shown as estimates (mean±SD) in each column having different combinations of superscripts (a, b, c, d...).

2- Kidney function parameters: Urea, Creatinine and Uric acid levels in negative control group (-ve) and Hyperlipidemic rat's groups which feed on the basal diet and Quinoa seeds:

Data in Table 5 showed a significant increase in serum Urea, Creatinine and Uric acid levels (7.45, 0.89 and 7.5 mg/dL, receptively) in

positive control group (+ve) compared to the negative control group (-ve). While, the group feeding on quinoa seeds the levels of Urea, Creatinine and Uric acid were lowered to 3.21, 0.3 and 3.44 mg/dL respectively. According to **Halaby *et al.*, (2017)** the high cholesterol diet induced hypercholesterolemia rats had the higher values of serum urea, creatinin and uric acid. Also, our results are in agreement with **Alamri *et al.*, (2023)** and **An *et al.*, (2021)** who found that feeding on quinoa seeds could significantly reduce the levels of urea, Creatinine and Uric acid in the serum and showed good kidney protection in Hyperlipidemic rat.

Table 5: Kidney function parameters: Urea, Creatinine and Uric acid levels in negative control group (-ve) and Hyperlipidemic rat's groups which feed on the basal diet and Quinoa seeds:

Parameter		Urea	Creatinin	Uric acid
		(mg/dL)	(mg/dL)	(mg/dL)
Rats Group				
Untreat	Negative Control (-ve)	2.55 ^c ± 0.08	0.33 ^b ± 0.02	3.23 ^a ± 0.05
	Positive Control (+ve)	7.45 ^a ± 0.13	0.89 ^a ± 0.05	7.5 ^a ± 0.35
treat	Quinoa seeds	3.19 ^b ± 0.13	0.29 ^c ± 0.01	3.37 ^b ± 0.27

Results were shown as estimates (mean±SD) in each column having different combinations of superscripts (a, b, c, d...).

3- Troponin T levels in negative control group (-ve) and Hyperlipidemic rat's groups which feed on the basal diet and Quinoa seeds:

Data in Table 6 showed the lowest troponin T levels at (9.73 pg/ml) in negative control group (-ve), followed by Hyperlipidemic rats group feed on quinoa seeds at (14.60 pg/ml). While, positive control group (+ve) had the highest troponin T level (30.26 pg/ml) when compared with the negative control group (-ve). According to **Feng *et al.*, (2001)**; **Thygesen *et al.*, (2010)**; **White (2011)** and **Twerenbold *et al.*, (2012)** the cardiac troponins enzymes are structural proteins found only in the human's heart. Detection of troponin in peripheral blood indicates cardiomyocyte damage. Whether

cardiomyocyte damage indicated by troponin necessarily means cardiomyocyte death remains an unresolved question. As there are no sufficiently sensitive in vivo methods to measure cardiomyocyte integrity despite troponin release, we propose that any damage detected by troponin release should be considered irreversible. This hypothesis is supported by autopsy studies showing significant cardiomyocyte loss throughout life.

Table 6: Troponin T levels in negative control group (-ve) and Hyperlipidemic rat's groups which feed on the basal diet and Quinoa seeds:

Parameter		Troponin T (pg/ml)
Rats Group		
Untreated	Negative Control (-ve)	9.73 ^c ± 0.63
	Positive Control (+ve)	30.26 ^a ± 0.84
treated	Quinoa seeds	14.60 ^b ± 0.67

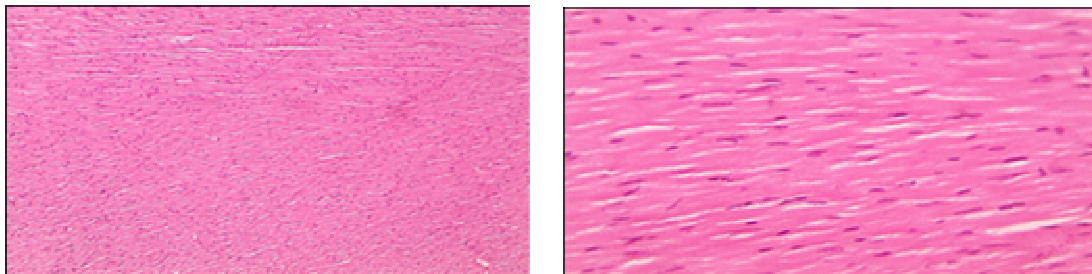
Results were shown as estimates (mean±SD) in the column having different combinations of superscripts (a, b, c, d...).

Histopathological analysis of specimens:

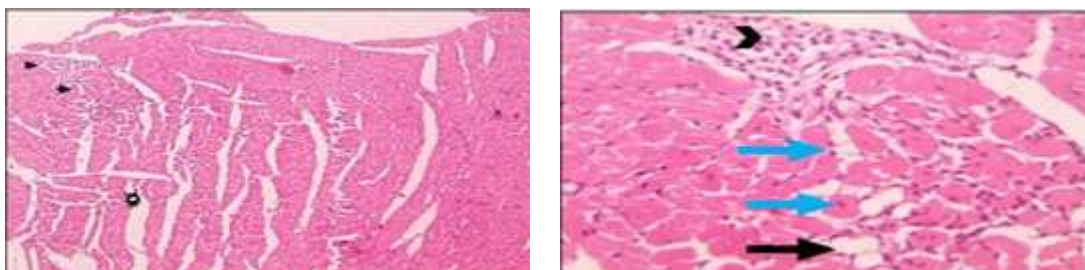
1. Cardiac tissues:

Microscopic pictures of cardiac tissues stained with haematoxylin and eosin (H&E) showing no histopathological changes including distinct myofibrils arrangement and no congestion in vessels in negative control group (-ve). Cardiac tissues from positive control group (+ve) showing disarray of myofibrils, marked tissue damage consisting of inflammatory cell infiltration (arrowheads), congestion (red arrow), widened interstitial space (*), vacuolization (black arrow), apoptosis (blue arrow) of myofibrils. Cardiac tissues from Hyperlipidemic rats group fed with Quinoa seeds showing markedly decreased interstitial space (*). Low magnification X:100 bar 100 and high magnification X:400 bar 50. **Halaby *et al.*, (2017) and Alghamdi (2018)** studies found that diet with 40% or 45% quinoa seeds had

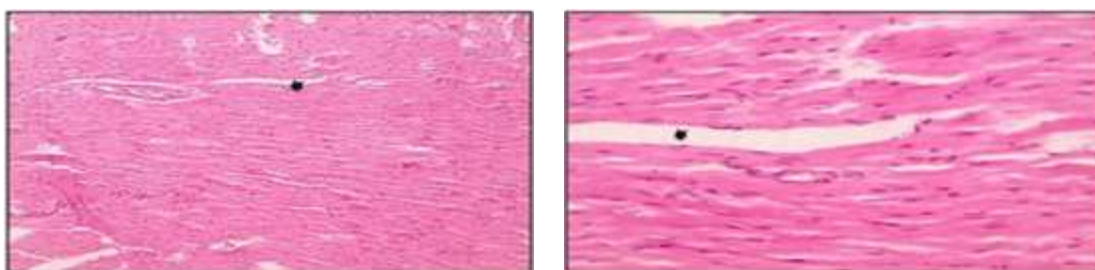
reduced the adverse effect of hypercholesterolemia and showed no histopathological changes in rat's cardiac tissues.



Pic (1): Cardiac tissues from the negative control group (-ve) showing no histopathological changes (H&E-stained, X: 400 bar 50).



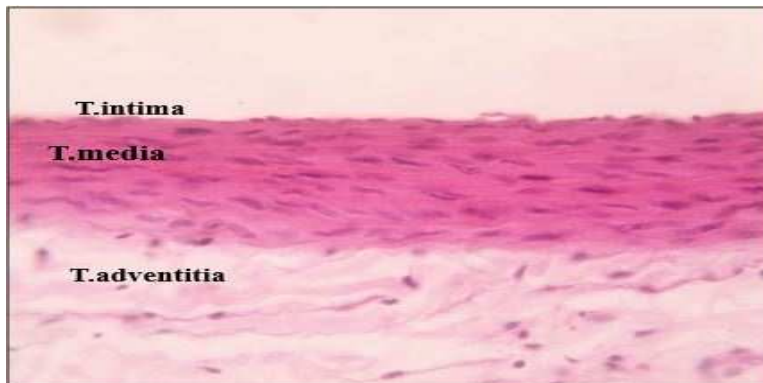
Pic (2): Cardiac tissues from the positive control group (+ve) showing disarray of myofibrils, marked tissue damage consisting of inflammatory cell infiltration (arrowheads), congestion (red arrow), widened interstitial space (*), vacuolization (black arrow), apoptosis (blue arrow) of myofibrils (H&E-stained, X: 400 bar 50).



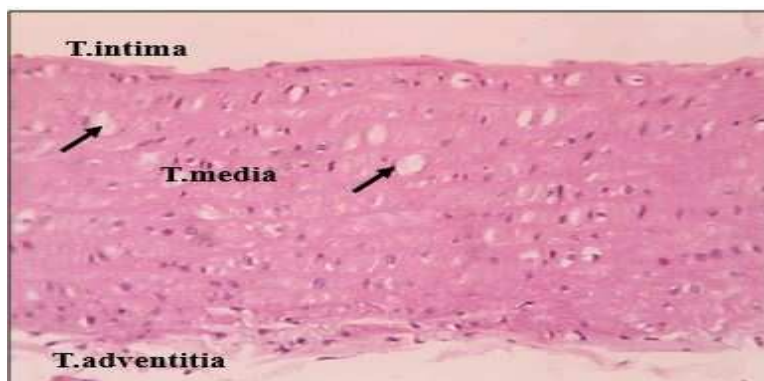
Pic (3): Cardiac tissues from Hyperlipidemic rats group fed with Quinoa seeds showing markedly decreased interstitial space (*) (H&E-stained, X: 400 bar 50).

2. Aortic sections:

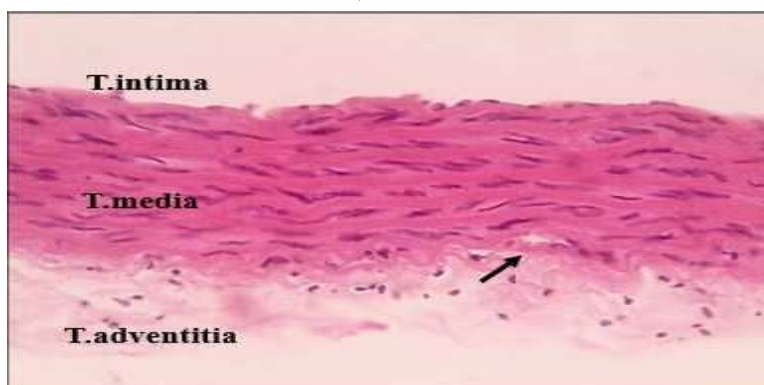
Microscopic pictures of aortic sections stained with haematoxylin and eosin (H&E) showing normal and regular arranged layers consisting of T. intima, T. media and T.adventitia in negative control group (-ve). Aortic sections from positive control group (+ve) showing increased thickness of T. media due to prominent vacuolation (arrows) and irregularity and disarrangement of smooth muscle fibers in T. media. Aortic sections from Hyperlipidemic rats fed with Quinoa seeds showing decreased thickness of whole aortic wall, regular layers, with very mild damage of smooth muscle fibers (arrow) in T. media. X:100 bar 100 and high magnification X:400 bar 50. According to **Zălar *et al.*, (2022)** the aortic histopathological section, we notice that the high-fat diet induced characteristic changes revealing aorta, media and intima thickening due to fat infiltration in liver and aortic sections which lead to smooth muscle cells, elastic fibers migration and a disorganized structure of the nucleus.



Pic (4): Aortic sections from the negative control group (-ve) showing normal and regular arranged layers consisting of T. intima, T. media and T.adventitia changes (H&E-stained, X: 400 bar 50).



Pic (5): Aortic sections from the positive control group (+ve) showing increased thickness of T. media due to prominent vacuolation (arrows) and irregularity and disarrangement of smooth muscle fibers in T. media (H&E-stained, X: 400 bar 50).



Pic (6): Aortic sections from Hyperlipidemic rats group fed with Quinoa seeds showing decreased thickness of whole aortic wall, regular layers, with very mild damage of smooth muscle fibers (arrow) in T. media (H&E-stained, X: 400 bar 50).

CONCLUSION:

This study concluded that hyperlipidemic rats fed quinoa seeds in their diet had a therapeutic effect in reducing the harmful effects of hyperlipidemia in the affected rats, and thus it is recommended to highlighting the importance of consuming quinoa seeds with diets as they play an important role in the treatment of hyperlipidemia.

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تأثير بذور الكينوا (*Chenopodium quinoa Willd.*)

على الفئران المصابة بارتفاع مستوى دهون الدم

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الملخص العربي:

صممت الدراسة الحالية لدراسة تأثير بذور الكينوا على الفئران المصابة بارتفاع مستوى دهون الدم. أجريت الدراسة على ثمانية عشر فأراً ذكراً متوسط وزنها (126 ± 10 جرام)، حيث تم تقسيمها بشكل عشوائي إلى ثلاث مجموعات (6 لكل منها). المجموعة الأولى الكنترول السالبة (-ve) والتي تغذت على الوجبة القياسية، والمجموعة الثانية المصابة بارتفاع مستوى دهون الدم والغير معالجة (+ve) والتي تغذت على نظام غذائي أساسي يحتوي على 1.5% كولسترول لمدة 21 يوماً، بينما المجموعة الثالثة مصابة بارتفاع مستوى دهون الدم وتغذت على نظام غذائي أساسي يحتوي على 1.0% من بذور الكينوا لمدة 30 يوماً. وقد استمرت التجربة لمدة 60 يوماً، تم خلالها تسجيل كمية الطعام المتناولة يومياً ووزن الفئران أسبوعياً، وفي نهاية التجربة تم جمع عينات الدم من الفئران لفحص المستويات التالية: بعض وظائف الكلى والكبد ومستويات دهون الدم ومستويات تروبونين T. كذلك، تم فحص التغيرات النسيجية في أنسجة القلب والشريان الأورطي. وقد أظهرت نتائج الدراسة أن المجموعة الضابطة الموجبة الغير معالجة (+ve) قد شهدت زيادة كبيرة في وزن الجسم وخلل كبير في مستويات بعض وظائف الكلى والكبد، ومستويات دهون الدم، وتروبونين T عند مقارنتها بالمجموعة الضابطة السالبة (-ve). بينما أظهرت مجموعة الفئران المصابة بارتفاع دهون الدم التي تتغذى على بذور الكينوا انخفاضاً في وزن الجسم وتحسناً كبيراً في مستويات بعض وظائف الكلى والكبد، ومستويات دهون الدم، وتروبونين T عند مقارنتها بالمجموعة الضابطة الموجبة الغير معالجة (+ve). كما أظهر الفحص النسيجي لأنسجة القلب والشريان الأورطي تأثيراً إيجابياً في المجموعة المصابة بارتفاع مستوى دهون الدم التي تتغذى على بذور الكينوا. استنتجت هذه الدراسة أن الفئران المصابة بارتفاع مستوى دهون الدم والتي تغذت على بذور الكينوا في نظامها الغذائي كان لها تأثير علاجي في تقليل التأثيرات الضارة بارتفاع مستوى دهون الدم لدى الفئران المصابة، ومن هنا يوصى بتسليط الضوء على أهمية تناول بذور الكينوا مع الوجبات الغذائية حيث تلعب دوراً هاماً في علاج ارتفاع مستوى دهون الدم.

الكلمات المفتاحية: بذور الكينوا - *Chenopodium quinoa Willd.* - ملف الدهون - ارتفاع

الكوليسترول في الدم - الدهون الثلاثية - تروبونين - فئران

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