Effect of Quinoa Sprouts (*Chenopodium Quinoa*) on Inflammation and Oxidative Stress Markers of insulin resistance rats

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Abstract

One of the pseudo-cereal cereals that are high in both macronutrients and micronutrients is quinoa. This research records changes in the phenolic content and antioxidant bioactivity of quinoa seeds during germination. This study's objective was to assess the impacts of quinoa sprouts (QS) on dexamethasone- induced insulin resistance rats. Thirty-five Sprague-Dawley adult male rats were divided into five groups. Group (1) served as a negative control group and was given a basal diet, While the other rats (n= 28) were injected intraperitoneally with dexamethasone (Dexa) in a dose of (1 mg/kg b.wt) 3 times/ week for 6 weeks to induce insulin resistance. Group (2) was then maintained as insulin resistance rats (positive control) and the other three groups were given 10, 15, and 20% of QS, respectively. The findings revealed that the germination of quinoa improved the phenolic content and antioxidant activity of quinoa seeds and decreased anti-nutritional factors. Meanwhile, administration of QS significantly reversed the biochemical parameters induced by Dexa in rats, it caused significant changes in body weight gains, feed efficiency ratio, feed intake, and decreased serum liver, kidney function, and lipid profile. It also decreased fasting insulin, fasting glucose, glucose tolerance, and HOMA-IR. Furthermore, significantly increased in antioxidant enzymes (SOD, CAT, Gpx and TAC) while, a decrease in MDA, serum cytokines as interleukin-1 (IL-β1) and tumor necrotic factor-α (TNF-α) in insulin resistance rats. In conclusion, incorporating quinoa sprouts as functional food ingredients should be taken into consideration given their positive effects and possible health benefits in reducing inflammation and oxidative stress in insulin resistance rats.

Keywords: *Chenopodium Quinoa*, Insulin resistance, Glucose tolerance, Dexamethasone, Inflammation, Oxidative stress.

Introduction

A decrease in the reactions of insulin target cells is known as insulin resistance (IR) **(Creus *et al*., 2017)**. IR can be a risk factor for the emergence of dyslipidemia and cardiovascular illnesses, and is crucial in the prediction of type 2 diabetes. IR alters glucose and lipid metabolism, which is evidenced by reduced glycogen synthesis, increased lipolysis, and impaired glucose uptake and oxidation **(Akhtar *et al*., 2019)**.

Dexamethasone (Dexa) is a synthetic glucocorticoid widely. Despite being a potent immune suppressant and anti-inflammatory, there are a number of adverse effects that could occur from its use, including skeletal muscle atrophy hyperglycemia, dyslipidemia, glucose intolerance and insulin resistance **(Safaeian *et al*., 2018)**. Dexa increases the generation of free radicals, which in turn increases cellular damage brought on by oxidative stress. Numerous antioxidant-rich plants have been shown to reduce insulin resistance because oxidative stress is a key factor in the formation of the condition **(Aryaeian et al., 2017**and **Abou-Seif *et al*., 2019)**.

The pseudo cereal quinoa (*Chenopodium quinoa Willd*.) is referred to as "one of the cereals of the 21st century" **(Dakhili *et al*., 2019)**. Quinoa has a high nutritional value because it's a good source of high-quality protein, fiber, carbohydrates, vitamins, and minerals, and it also has therapeutic qualities that make it suitable for use as a functional food **(Pereira *et al*., 2019)**. In recent years, quinoa sprouts have gained enormous popularity because of their numerous health advantages. Germination can help to reduce antinutritional factors such as saponins, tannins, and phytic acids, which can reduce bioavailability due to forming insoluble complexes with minerals, such as zinc and iron **(Al-Qabba *et al*., 2020)**. Germination includes a sequence of activities that start with the dry, dormant seed absorbing water and end with the elongation of the embryo axis **(Benincasa *et al*., 2019)**. It has been thought that germination is a low-cost and efficient way to boost antioxidant capacity and increase the bioavailability of vital minerals and vitamins **(Maldonado-Alvarado *et al*., 2023)**. This study's objective was to assess the effect of germination on the chemical composition, vitamin and mineral content, antioxidants, and anti-nutritional factors of quinoa.

Additionally, a biological evaluation was done to monitor the effect of quinoa sprouts at (10, 15, and 20) % on inflammation and oxidative stress markers of insulin resistant rats.

Materials and Methods

Materials

Quinoa was obtained from the grains and herbs market in Cairo, Egypt.

Chemicals: Casein, cellulose, vitamins, minerals, and dexamethasone were supplied from Sigma Chemical Company in Cairo, Egypt.

Kits: for biochemical analysis were obtained from Gama Trade Company, Cairo, Egypt.

Rats: Thirty-five Sprague-Dawley adult male rats (weighing 195±10 g) were obtained from the animal house of the National Research Center, Giza, Egypt.

Methods

Quinoa seeds germination

Quinoa grains were cleansed of foreign objects and then soaked in distilled water at 25°C for 12 hours in a ratio of 1:5 grains to water (w/v) to remove saponin, which is responsible for the bitter taste **(Brady *et al*., 2007)**. For germination, the previously soaked grains were spread out separately on damp jute bags, then covered with muslin cloth and another wet jute bag. The grains were then watered every 12 hours until the germination time was complete (72 hr.).The sprouted grains were carefully picked along, washed, dried in an oven at 50°C for 24 hours, crushed, and kept in labeled polyethylene bags **(Padmashree *et al*., 2019)**.

A- Gross chemical composition

According to the method described in the **A.O.A.C (2016)**, the moisture, protein, fat, fiber, and ash contents of quinoa seeds and sprouts were determined. By using the differential, total carbohydrates were computed. According to **Chaney (2006)** description, gross energy was determined using the following equation: Gross energy = 4 × (Protein % + Carb. %) + 9 × (Fat %).

Determination of Total phenolic, flavonoid content, antioxidant, and anti-nutritional factors.

Folin-Ciocalteu colorimetric method was used to estimate total phenolic compounds (TPC) of raw grains and sprouts of quinoa by **Yawadio Nsimba *et al*., (2008)**. The total flavonoid content (TFC) of raw grains and sprouts of quinoa were determined according to **Mohdaly *et al*., (2012)**. Total antioxidant capacity was determined by the method of **Prieto *et al*., (1999)**. The contents of saponin, phytic acid, alkaloids, tannins, and oxalates were measured according to the methodology described by **Obadoni and Ochuko, (2001); Reason *et al*., (2015); Harborne (1973); Makkar *et al*., (1993),** and **Abaza *et al*., (1968)**, respectively.

Vitamins content

Vitamin B2 and C were measured by the method of **Pinheiro *et al*., (2021)** and **Sun-Ju *et al*., (2006)**, respectively.

Minerals content

According to the method outlined by **Tazrart *et al*., (2016)** an atomic absorption spectrometer was used to measure the amounts of magnesium (Mg), iron (Fe), calcium (Ca), and zinc (Zn).

Experimental design

For adaptation, 35 adult male Sprague-Dawley rats weighing (195±10) g were fed on a baseline diet for a week. According to **Reeves *et al*., (1993)**, the basal diet (AIN-93M) was created to provide rats with the suggested amounts of nutrients.

Ethical approval: The study received approval from the research ethics committee of the faculty of nursing at Port Said University, code number: NUR (4-12-2022) (20).

After one week, rats will be randomly divided into five groups as follow: Group (1) (n=7) received the baseline diet as a negative control group, while the other four groups (n=28) received intraperitoneally (i.p.) injections of Dexa at a dosage of 1 mg/kg/bw 3 times/week for 6 weeks to cause IR according to **Shittu *et al*., (2021)**. One of these groups served as positive control which was group (2) while, the other three groups were given (10, 15, and 20) % of QS, respectively. Rats underwent a glucose tolerance test at the

conclusion of the treatment. At the end of the experiment, rats were serially anaesthetized with diethyl ether after a 12-hr fast. Rats were euthanized and organs were dissected. The posterior vena cava was used to gather blood samples into dry, clean centrifuge tubes, which were then allowed to clot at room temperature before being centrifuged for 10 minutes at 3000 rpm to separate the serum. For biochemical examination, serum samples were frozen at -20°C.

Biological Evaluation

Daily amounts of food consumed and/or wasted were recorded while total feed intake (FI) was calculated. Additionally, rats body weights (BW) were tracked weekly. According to **Champman *et al.*, (1959)**, body weight increase percentage (BWG%) was calculated using the following equation:

$$\mathbf{BWG}\% = \frac{\text{Final body weight} - \text{Initial body weight}}{\text{Initial body weight}} \times 100$$

$$\mathbf{FER} = \frac{\text{weight Gain (g)}}{\text{Feed intake (g)}}$$

Oral glucose tolerance test

After a 12-hr fast, glucose (2 g/kg) was administered to all of the rats in the groups. Rat tail vein blood samples were used to evaluate the oral glucose tolerance test (OGT) at 0, 15, 30, 60, 90, and 120 minutes after glucose administration **(Rohling *et al.*, 2019)**.

Biochemical analysis

Fasting blood glucose (FBG) was determined according to **Burrin and Price, (1985)**. According to **Chevenne *et al.*, (1998)** description the enzyme-linked immunosorbent assay (ELISA) method was used to measure insulin activity. Homeostasis of Insulin Resistance index (HOMA-IR) calculated by **Salgado *et al.*, (2010)** using the following equation:

HOMA‑IR= {[fasting insulin (μU/ml)] × [FBG (mmol/L)]} /22.5.

Aspartate aminotransferase (AST) and alanine aminotransferase (ALT) were measured to determine liver function according to **Bergmeyer *et al.*(1978)**, alkaline phosphatase (ALP) was measured using **Belfield and Goldberg (1971)**. Total protein was determined

by the method of **Weichse-lbauml, (1946)**. Urea, uric acid, and creatinine were measured to determine kidney function according to **Patton and Crouch, (1977) Fossati *et al*., (1980)**, and **Bonsens and Taussky (1984),** respectively. Triglycerides (TG), total cholesterol (TC), and cholesterol contents of high density lipoprotein (HDL) were measured in accordance with **Wahlefeld, (1974)**, **Fossati and Principe,(1982)** and **Albers *et al*.,(1983)**, respectively. Calculations of low density lipoprotein cholesterol (LDL) and very low-density lipoprotein cholesterol (VLDL) by the equation of **Fruchart, (1982)**. LDL-c = TC-[HDL-c + (TG/5)] VLDL-c = TG/5.

Serum-selected cytokines such as Interleukin-1 (IL-1β) and tumor necrotic factor-α (TNF-α) were determined according to **Kandir and Keskin, (2016)**. For assessing lipid peroxidation, the plasma level of Malondialdehyde (MDA) was determined according to **Draper and Hadley, (1990)**. The activity of superoxide dismutase (SOD) was assessed according to **Spitz and Oberley, (1989)**. Catalase (CAT) and Glutathione peroxidase (GPX) were measured methods by **Aebi, (1984), Pagalia and Valentine, (1967)** respectively. **Benzie and Strain, (1996)** method was used to determine the total antioxidant capacity (TAC).

Statistical analysis

The results were presented as mean ± Standard Error (SE). One-way ANOVA followed by post hoc multiple comparisons were used to statistically evaluate the data using the SPSS program (**Snedecor and Cochron, 1989)**.

Results and Discussion

Table (1) observed that the germination of quinoa showed an increase in protein content from 15.4 to 17.5% due to protein synthesis. In addition the increased level of fiber content from 3.98 to 5.91% as compared with quinoa seeds. When, a new plant is created as a result of germination, its cell wall contains a high percentage of insoluble fiber. Also, quinoa seeds and sprouts provided energy of (373.13 - 368.21 Kcal/100g), respectively. These outcomes were in line with **Maldonado-Alvarado *et al*., (2023)** found that germination increased fiber and protein content ranged between (16 and 19%). Similarly, **Ibrahem and Mohamed, (2021)**

observed germination of quinoa showed an increase in the protein and fiber content (18.9 and 5.23%) respectively. **Mohamed *et al*., (2022a,b)** proved that germinated quinoa had the best outcomes in terms of proteins, followed by non-germinated quinoa (19.1 and 14.9). Additionally, quinoa grains offered 375.8 Kcal/100g of energy. Furthermore, **Guardianelli *et al*., (2022)** noted that quinoa sprouts (QS) had less moisture compared to row quinoa (RQ), and after 48 h of germination, lipid values decreased. This effect was ascribed to a portion of the lipids being degraded to produce energy for the growth of the new plant. The ash content of the quinoa samples, according to **Pilco-Quesada *et al*., (2020)**, varied from 2.3 to 4.3%; however, there was a preliminary decrease in ash content during soaking, which might be accounted for by the mineral lixiviation occurring at this time.

Table (1): Chemical composition of quinoa seeds and sprouts

Components / Samples	Moisture %	Protein %	Fat %	Carb %	Ash %	Fiber %	Gross Energy kcal/100g
Quinoa Seeds	6	15.4	5.65	65.17	3.8	3.98	373.13
Quinoa Sprouts	4.5	17.5	4.29	64.9	2.9	5.91	368.21

The results presented in Table (2) showed that germination caused an increase in total phenolic, flavonoids compounds, and antioxidant capacity compared to the RQ from (197.75, 130.21 and 415.62 mg/100g) to (344.48, 168.26 and 1018.6 mg/100g), respectively. Consistent with **Mohamed *et al*., (2022a,b)** showed that TPC and total antioxidant capacity of raw and germinated quinoa seeds were recorded (212.5 , 362.7 mg GAE/100g) and (450.44 , 998.9 mg/100g ascorbic acid) respectively. A similar study by **Namrata and Haripriya, (2022)** noted that the germination of quinoa improved TPC, and the antioxidant activity increased by 7.5% compared to the raw sample due to biochemical, physical, and enzymatic activities. This aligns with the results of a previous study by **Le *et al*., (2021)** observed that 72h germination resulted in an increase of 101.2% in TPC. In addition TFC in quinoa sprouts value ranged from (100.38 to 304.10 mg QE/100g^{-1} DW). On the other hand, **Guardianelli *et***

***al*., (2022)** observed that the TFC content of quinoa increased significantly with germination time. Consistent with these reports **Ibrahem and Mohamed, (2021)** found that the germination significantly increased ($P \leq 0.05$) in TFC and total antioxidant capacity (123.65 to 161.7 mg/100g) and (410.64 to 1013.9 %) compared to the RQ. Hence, germination helps produce more bioactive substances like phenols and flavonoids **(Ruijuan *et al*., 2021)**.

Table (2): Total phenolic, flavonoid and antioxidant activity.

Components \ Samples	Quinoa Seeds	Quinoa Sprouts
Total Phenolic (mg/ GAE 100g)	197.75	344.48
Total Flavonoids (mg QE/100 g)	130.21	168.26
Antioxidant activity (mg ASE/100g)	415.62	1018.6

GAE: Gallic acid, QE: Quercetin, ASE: ascorbic acid equivalent

Table (3) illustrates germination process gradually decreased saponin, phytic acid, oxalate, tannins and alkaloids content at (1.3, 1.26, 0.13, 0.18 and 0.19% respectively) as a result of their leaching from quinoa grains during soaking and washing. These outcomes were in line with **Mohamed *et al*., (2022a,b)** showed that The germination phase is where anti-nutritional factors are found to be reduced the most. Similarly, **Namrata and Haripriya, (2022)** showed that the total saponin and tannin content decreased from (1.597 to 0.938 g SE/100 g) and (343 to 48.02 mg TA/100 g) respectively, during 48 h germination, which seems to be in accordance with our findings. **Maldonado-Alvarado *et al*., (2023)** indicated that Phytic acid levels in the white quinoa significantly decreased after one week of germination.

Table (3): Effect of germinating on anti-nutritional factors in quinoa.

anti-nutritional \ Samples	Saponin %	Phytic acid %	Oxalate %	Tannins %	Alkaloids %
Quinoa Seeds	2.8	1.36	0.21	0.49	0.74
Quinoa Sprouts	1.3	1.26	0.13	0.18	0.19

The data in Table (4) showed that the germination process gradually increased the vitamins B_2 and C content from (0.060 and 195 to 0.305 and 258 mg/100g) respectively. These outcomes are consistent with **He *et al*., (2022)** and **Pinheiro *et al*., (2021)** discovered that as the germination period was extended, the contents of VC and VB_2 increased. **Le *et al*., (2021)** found the amount of VB_2 ranged from (0.059 to 0.076 mg/100 g DW) in quinoa seeds. **Darwish *et al*., (2020)** found that quinoa germinating for 72 h, increase the vitamin C content by 32.17%, with values hitting 260.5 mg/100g DW. The outcomes demonstrate that increasing the nutritional value of quinoa seeds through germination is a viable strategy.

Table (4): Effect of germinating on the content of quinoa seeds from vitamins.

Vitamins / Samples	Quinoa Seeds	Quinoa Sprouts
	mg/100g	
Vitamin B2	0.060	0.305
Vitamin C	195	258

Table (5) observed that the germination process gradually increased (Zn, Fe, Mg, and Ca) content at (2.4, 14.9, 138.7and 89.8 mg/100g respectively). These findings support **Maldonado-Alvarado *et al*., (2023)** reported that germination caused a substantial increase in the calcium content of quinoa by 17%, zinc content by 10%, and iron concentration increased significantly ($p \leq 0.05$) with germination until the seventh day, In comparison to the values of the initial raw material. Similarly, **Guardianelli *et al*., (2022)** showed that the greatest calcium content was found in germinated white quinoa, followed by iron and zinc. Similarly, **Ibrahem and Mohamed, (2021)** noted that the germination of quinoa improved calcium concentration increased by 20%. According to **Kajla *et al*., (2017)**, changes in these micronutrients may result from the hydrolysis of complex organic compounds that release minerals during germination and may also as enzymatic cofactors, causing them to differ depending on the stage. Furthermore, phytate hydrolysis during germination could be a simple way to improve the abundance of minerals for vegetarian diets **(Maldonado-Alvarado *et al*., 2023**).

Table (5): Effect of germinating on the content of quinoa of minerals.

Minerals / Samples	Zn	Fe	Mg	Ca
	mg/100g			
Quinoa Seeds	1.9	10.9	127.9	78.9
Quinoa Sprouts	2.4	14.9	138.7	89.8

Results from Table (6) show that dexamethasone significantly reduced body weight while significantly increasing ($p \leq 0.5$) liver weight of insulin resistance rats compared to normal control. This is consistent with **Poualeu Kamani *et al*., (2022)** and **Shittu *et al*., (2021)** who reported that dexa administration caused rat's body weight to significantly reduce while increasing the relative liver weight by 40% when compared to the healthy control group. In contrast, quinoa sprouts (QS)-treated at (10, 15, and 20) % caused a significant increase in body weight and feed intake but caused a significantly reduce ($p \leq 0.05$) in liver weight of rats compared to dexa group. Which, this liver hypertrophy induced by dexa was reduced by quinoa sprouts. No significant differences in levels of IBW and FBW between the groups treated with QS.

Table (6): Effect of Quinoa sprouts consumption on body weight, feed intake and relative liver weight of insulin resistant rats

Parameters / Groups	IBW (g)	FBW (g)	BWG %	FI (g/ day)	FER	Relative liver weight
Normal control	198.81±1.73^{a}	220.02±1.60^{a}	10.67±2.81^{a}	17.20	0.029±0.03^{a}	2.51±0.06^{d}
Dexa	205.30±1.64^{a}	179.24±1.44^{c}	-12.69±1.83^{d}	13.38	-0.046±0.02^{d}	5.33±0.12^{a}
Quinoa sprouts (10%)	201.24±1.13^{a}	192.50±2.62^{b}	- 4.34±1.65^{c}	15.52	-0.013±0.04^{c}	4.09±0.08^{b}
Quinoa sprouts (15%)	202.23±1.24^{a}	198.51±2.41^{b}	-1.84±1.09^{bc}	15.64	-0.006±0.03^{bc}	3.54±0.04^{c}
Quinoa sprouts (20%)	203.90±0.60^{a}	201.22±3.54^{b}	-1.31±0.59^{b}	15.93	-0.004±0.03^{b}	2.80±0.09^{d}

Initial body weight (IBW), Final body weight (FBW), Body weight gain% (BWG %), feed intake (FI) and feed efficiency ratio (FER). Results are expressed as mean ± SE.

Values in each column which have different letters are significantly different at ($P \leq 0.05$).

Our results are corroborating those previously released reports by **Mohamed *et al*., (2022a,b)** revealed that the basic diet supplemented with quinoa that had been germinated had the best results for reducing weight. **Ibrahem and Mohamed, (2021)** showed that germinating of quinoa seeds resulted in the greatest reductions in FBW, BWG%, and FER. Similarly, **Wahba *et al*., (2019)** observed that quinoa seed powder demonstrated improvement in each of BWG, FI, and FER compared to the (+ve) group at different levels (10 to 40%). Additionally, these results are consistent with **Lopes *et al*., (2019)** noted that in rats given diets supplemented with quinoa, the sprouting processes potentiate the ability to reduce FI due to the chemical changes promoted by processing quinoa.

Table (7) showed subcutaneous dexa successfully produced IR in normal rats, as evidenced by hyperglycemia, hyperinsulinemia, impaired oral glucose tolerance, and elevated HOMA-IR This is consistent with **Mahmoud *et al*., (2022)** demonstrated that dexa cause IR in rats at various dose levels and for various lengths of time. **Alkot *et al*., (2022)** observed the introduction of dexa caused a substantial increase in FBS, insulin level, and HOMA-IR when compared to the normal control group. Also, **Alona *et al*., (2021)** showed that dexa caused rats to acquire a condition known as IR and glucose intolerance, which occurs before the onset of diabetes and obesity.

Table (7): Effect of Quinoa sprouts consumption on glycemic indices of insulin resistance rats.

Parameters / Groups	Fasting Insulin (FI) (µIU/ml)	Fasting Blood Glucose (FBG) (mmol/L)	Homeostasis Insulin Resistance (HOMA- IR)
Normal control	8.95 ± 1.29^{c}	4.82 ± 0.19^{d}	1.92 ± 0.05^{d}
Dexa	14.96 ± 2.38^{a}	10.54 ± 0.35^{a}	7.01 ± 0.04^{a}
Quinoa sprouts (10%)	10.27 ± 1.26^{b}	7.17 ± 0.14^{b}	3.27 ± 0.01^{b}
Quinoa sprouts (15%)	9.93 ± 0.42^{b}	6.37 ± 0.28^{c}	2.81 ± 0.01^{c}
Quinoa sprouts (20%)	9.24 ± 0.61^{bc}	6.20 ± 0.27^{c}	2.55 ± 0.01^{cd}

Results are expressed as mean ± SE.
Values in each column which have different letters are significantly different at ($P \leq 0.05$).

On the other hand, QS-treated following dexa administration reversed the IR that had been seen in the rats that had only received dexa, as demonstrated by a decrease in FI, FBG, and HOMA-IR in QS-treated groups as compared to dexa. Obviously, the effectiveness of the studied germinated quinoa is due to the presence of various biologically active substances. Furthermore, no apparent difference was obvious in the serum glycemic indices between the QS -treated rats of both doses (15 and 20 %). The fact that there was no change in FI and HOMA-IR levels of both groups given quinoa at (15 and 20%), is proof that quinoa is safe when compared to the healthy group. This is consistent with **Erfidan *et al*., (2022)** determined glucose, insulin levels, and HOMA-IR increased due to experimental insulin resistance while, it decreased with quinoa for the treatment group (IQ) and quinoa + IR for the prophylaxis group (QI), and the levels were discovered to approach the values of the control group. Due to the linear connection between HOMA-IR and insulin resistance, a higher HOMA-IR level denotes IR while a lower HOMA-IR level (compared to the control) denotes insulin sensitivity. Therefore, HOMA-IR index was determined that characterizes the degree of insulin resistance.

Similarly, **Al-Anazi, *et al.,*(2022)** observed that sprouted quinoa administration significantly reduced serum insulin and glucose levels. **Obaroakpo *et al*., (2020)** observed that sprouted quinoa yoghurt (QY) exhibited the highest hypoglycemic lowering effects when compared with non-germinated QY, and found that germination affected the glucose in T2DM mice. Also, **Lopes *et al.*, (2019)** reported that germination enhances quinoa's capacity to decrease blood glucose while increasing glucose tolerance in rats due to its ability to enhance the bioactive compounds.

Results of the oral glucose tolerance test are shown in Figure (1), all treatment groups showed an early rise in blood glucose levels at 30 min compared to the healthy control group. Furthermore, 120 min after reaching glucose tolerance, dexa groups experienced a greater rise in blood sugar levels than the healthy control group. These findings were on the same line with **(Erfidan *et al*., 2022** and **Poualeu Kamani *et al*., 2022)**. Contrarily, at the same time, a return to baseline blood glucose values was observed in all QS-treated groups. The postprandial hyperglycemia in QS-treated rats was

substantially reduced from the 60th to the 120th min, which was the best effect at a dose of 20%. Moreover, Dexa-induced impairment in glucose tolerance was completely and substantially reversed by QS. QS may have therefore reversed the body weight reduction brought on by IR. These results agreed with **Al-Okbi *et al*., (2021)** showed Quinoa has a high potential to improve glucose tolerance. **Obaroakpo *et al*., (2020)** reported that the germinated quinoa yoghurt groups had blood glucose levels 5.54% lower than the diabetic control 120 minutes after oral glucose administration. Similarly, **An *et al*., (2021)** observed that quinoa treatment could significantly reduce the level of blood glucose, and improve glucose tolerance of obese mice, the quinoa treatment substantially lowered the blood glucose levels 30, 60, 90, and 120 min before and after glucose administration in comparison to the control group. These findings suggest that quinoa may be useful in the management of diabetes and insulin resistance, including both prevention and therapy.

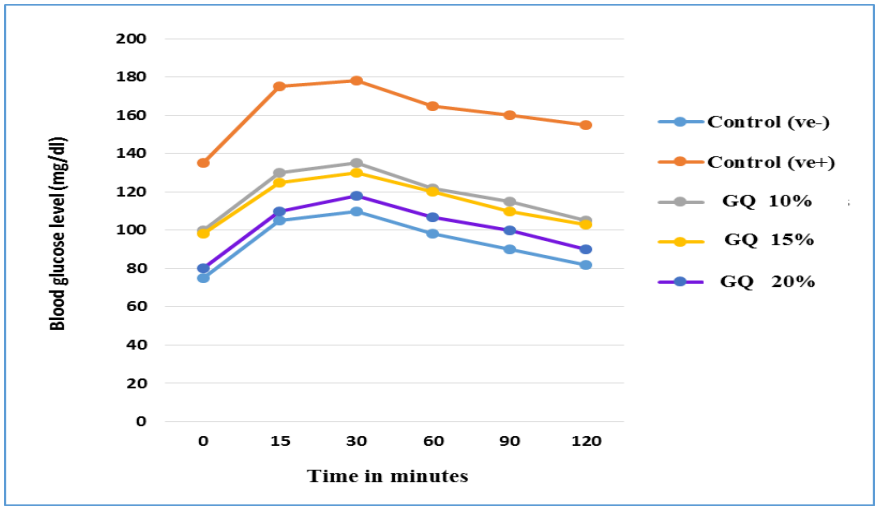

Figure (1): Effect of Quinoa sprouts on glucose tolerance test in insulin resistance rats

Table (8) described that dexa significantly increased ($p \leq 0.5$) in AST, ALT, and ALP, while decreasing the total protein of insulin resistance rats compared to normal control. This is consistent with **Poualeu Kamani *et al*., (2022)** noted that dexa significantly altered

biochemical parameters by increasing ALT, AST, and by decreasing total proteins. Similar results were reported by **Alkot *et al*., (2022)** revealed that dexa treatment significantly increased liver function. In contrast, quinoa sprouts (QS)-treated at (10, 15, and 20) % caused a significant decrease in AST, ALT, and ALP, while increasing total protein as compared to dexa group, approaching the level in the healthy group. Moreover, no significant difference ($p \leq 0.05$) in the T. protein among all groups quinoa- treated. IR- rats treated with QS at a dose of 20 % showed the greatest increase in liver function The outcomes corroborated the study of **Erfidan *et al*., (2022)** and **Ibrahem and Mohamed, (2021)** observed that quinoa administration significantly reduced liver function compared to the control groups. Similarly, **Obaroakpo *et al*., (2020)** observed that sprouted quinoa yoghurt (QY) significantly ($p \leq 0.05$) reduction of ALT and AST activities than non-germinated QY. **Al-Qabba *et al*., (2020)** observed that QS improved liver enzymes in CCl4-induced rats. These markers' decline, due to quinoa containing ascorbic acid, polysaccharides, and phenolic components, which are known as hepatoprotective factors **(Ng and Wang, 2021)**.

Table (8): Effect of Quinoa sprouts consumption on liver functions in rats of insulin resistance

Parameters / Groups	AST	ALT	ALP	T. protein
	(µ/L)			(g/dl)
Normal control	43.60±2.25^{e}	50.90±1.37^{d}	62.40±1.45^{e}	10.53±0.24^{a}
Dexa	120.54±1.27^{a}	150.60±0.96^{a}	120.80±2.08^{a}	5.32±0.10^{d}
Quinoa sprouts (10%)	88.85±1.22^{b}	120.80±0.79^{b}	100.94±1.47^{b}	6.98±0.19^{c}
Quinoa sprouts (15%)	65.92±1.08^{c}	98.30±2.61^{c}	83.43±1.38^{c}	7.64±0.27^{bc}
Quinoa sprouts (20%)	48.44±1.96^{d}	52.75±1.58^{d}	70.51±1.82^{d}	8.68±0.07^{b}

AST: Aspartate transaminase; ALT: Alanine transaminase; ALP: Alkaline phosphatase
Results are expressed as mean ± SE.
Values in each column which have different letters are significantly different at ($P \leq 0.05$).

Table (9) discovered that dexa significantly increased ($p \leq 0.5$) in urea, uric acid, and creatinine in insulin resistance rats in comparison to the normal control group. While the effect of quinoa

sprouts (QS)-treated at (10, 15, and 20) % caused a significant reduction in kidney function as compared to dexa group. Furthermore, no significant changes ($p \leq 0.05$) between the rats treated with quinoa at doses of 15% and 20% in uric acid. IR- rats treated with QS at a dose of 20 % showed the greatest increase in kidney function. This is consistent with **Erfidan *et al*., (2022)** dexa treatment significantly increase kidney function, while the best decrease was observed in the QI and IQ groups. Similarly, **Ali, (2019)** found that quinoa seeds at dose (10 and 5%) improved kidney and liver function in rats. **El-Kewawy and Morsy, (2019)** observed diet-fortified QS can improve serum urea, creatinine, and uric acid levels. Improvements in kidney function may be a consequence of the presence in QS of important compounds like flavonoids and phenolic compounds that lower uric acid levels and protect the kidney.

Table (9): Effect of Quinoa sprouts consumption on kidney function in rats of insulin resistance

Parameters / Groups	Urea	Uric acid	Creatinine
	(mg/dl)		
Normal control	38.74 ± 1.11^{e}	3.44 ± 0.08^{bc}	0.35 ± 0.02^{e}
Dexa	73.84 ± 1.72^{a}	4.22 ± 0.21^{a}	0.96 ± 0.04^{a}
Quinoa sprouts (10%)	63.07 ± 1.75^{b}	3.53 ± 0.09^{b}	0.75 ± 0.05^{b}
Quinoa sprouts (15%)	49.69 ± 1.93^{c}	3.28 ± 0.06^{cd}	0.66 ± 0.03^{c}
Quinoa sprouts (20%)	43.81 ± 1.67^{d}	3.20 ± 0.13^{d}	0.59 ± 0.05^{d}

Results are expressed as mean ± SE.
Values in each column which have different letters are significantly different at ($P \leq 0.05$).

Table (10) discovered that administration of dexa at a dose of 1 mg/kg caused dyslipidemia; it also induced a significant increase ($p \leq 0.05$) of TC, TG, LDL, and VLDL, but a decrease in HDL comparison to negative group. This is consistent with **Poualeu Kamani *et al*., (2022)** noted that dexa significantly altered biochemical parameters to raise TG levels. Numerous studies have shown that triglyceride is a useful indicator of insulin resistance **(Lee *et al*., 2021** and **Song *et al*., 2021)**. In contrast, the effect of Quinoa sprouts of insulin resistance rats caused a significant

decrease in lipid profile, but an increase in HDL comparing to dexa group. These results approve of the findings of **Ibrahem and Mohamed, (2021)** found that obese anemic rats treated with germinated quinoa had significantly reduced in TC, TG, VLDL, and LDL while increasing levels of HDL. **Al-Okbi *et al*., (2021)** and **Al-Qabba *et al*., (2020)** observed that quinoa sprouts improved the lipid profile due to the rich contents of phenols and antioxidants. Similarly, **Obaroakpo *et al*., (2020)** observed that germination had a significant (p 0.05) impact on lipid homeostasis in T2DM mice.

Table (10): Effect of Quinoa sprouts consumption on lipid profile in rats of insulin resistance

Parameters / Groups	TC	TG	HDL-c	VLDL-c	LDL-c
	(mg/dl)				
Normal control	130.50±2.38^{e}	110.84±1.40^{d}	64.72±1.21^{a}	22.17±0.28^{d}	43.61±4.29^{e}
Dexa	206.40±1.54^{a}	164.36±2.03^{a}	38.63±0.87^{c}	32.87±0.40^{a}	134.9±2.75^{a}
Quinoa sprouts (10%)	182.80±1.89^{b}	143.26±1.95^{b}	50.35±2.33^{b}	28.65±0.39^{b}	103.8±3.01^{b}
Quinoa sprouts (15%)	171.34±4.90^{c}	137.24±0.76^{c}	51.00±2.00^{b}	27.45±0.15^{c}	92.89±7.78^{c}
Quinoa sprouts (20%)	151.54±2.46^{d}	135.95±1.74^{c}	51.09±0.49^{b}	27.19±0.34^{c}	73.26±3.87^{d}

Total cholesterol (TC), Triglycerides (TG), High density lipoprotein -cholesterol (HDL), low density lipoprotein-cholesterol (LDL-C) and very low density lipoprotein-cholesterol (VLDL-C). Results are expressed as mean ± SE.
Values in each column which have different letters are significantly different at (P≤0.05).

Table (11) depicts that treatment by dexa caused a significantly increased (p≤0.5) in inflammatory cytokines including (IL-β1and TNF-α) compared to the normal control group. While, all quinoa sprouts groups at doses (10, 15, and 20%) remarkably reversed the (IL-β1 and TNF-α) level compared to the dexa group. The bioactive QS compounds may effectively conduct anti-inflammatory and anti-oxidative effects **(Pellegrini *et al*., 2019)**. The present results have been confirmed by **Erfidan *et al*., (2022)** observed that the increased production of cytokines like (IL-1β and TNF-α) caused

by the stimulation of inflammatory pathways by Dexa results in impaired insulin, which lowers glucose uptake and creates IR , while quinoa has been shown to significantly reduce (IL-1β and TNF-α) in IR- rats. Similarly, **Obaroakpo *et al*., (2020)** observed that Sprouted QY caused the level of pro-inflammatory cytokines were significant reduced when compared with the diabetic control group. **Al-Qabba *et al*., (2020)** observed that QS improved liver inflammation.

Table (11): Effects of Quinoa sprouts consumption on inflammation markers in rats of insulin resistance

Parameters / Groups	IL-1β (pg/ml)	TNF-α (pg/ml)
Normal control	6.65 ± 1.12^{d}	8.18 ± 0.60^{d}
Dexa	13.52 ± 0.02^{a}	18.28 ± 2.87^{a}
Quinoa sprouts (10%)	11.42 ± 0.87^{b}	15.01 ± 0.75^{b}
Quinoa sprouts (15%)	11.27 ± 0.19^{bc}	9.82 ± 0.26^{c}
Quinoa sprouts (20%)	10.75 ± 0.92^{c}	9.48 ± 0.67^{c}

Interleukin-1 (IL-1β) and Tumor necrotic factor-α (TNF-α)
Results are expressed as mean ± SE.
Values in each column which have different letters are significantly different at ($P\leq0.05$).

Table (12) depicts that treatment by dexa caused a significantly increased in MDA, whereas the antioxidant enzymes (SOD, CAT, Gpx and TAC) were significantly decreased compared to the normal group. However, quinoa sprouts significantly lowered ($p\leq0.5$) the oxidative stress to rats of insulin resistance by reducing MDA and increasing SOD, CAT, Gpx and TAC levels, due to their antioxidant properties. The present results have been confirmed by **Erfidan *et al*., (2022)** noted that quinoa improved biochemical markers antioxidant in experimental glucocorticoid-induced IR. **An *et al*., (2021)** observed that quinoa could increase oxidative stress but decrease the MDA of obese mice. Similarly, **El-Kewawy and Morsy, (2019)** reported that QS exhibited a significantly higher level of SOD, GPXs, GST, and CAT, but a significantly lower level of MDA compared to hyperuricemic control. **Al-Okbi *et al*., (2021)** and **Al-Qabba *et al*., (2020)** observed that QS extracts significantly reduced MDA compared to CCl4-rats. These findings were on the same line with **Obaroakpo *et al*., (2020)** observed that sprouted QY

had a greater capacity to regulate oxidative stress. **Wahba *et al*., (2019)** and **Ali, (2019)** found that quinoa seed powder improved antioxidant parameters such as (TAC and SOD) in female rats.

Table (12): Effect of Quinoa sprouts consumption on oxidative enzymes in rats of insulin resistance

Parameters / Groups	MDA (µmol/dl)	SOD (µ/dl)	CAT (µ/L)	Gpx µ/mg	TAC (mmol/L)
Normal control	11.67±1.90^{e}	92.34±1.37^{a}	130.13±0.30^{a}	65.44 ± 1.04^{a}	4.25±0.12^{a}
Dexa	34.56±2.04^{a}	54.43±1.22^{d}	68.79±1.23^{e}	27.14 ± 1.11^{d}	2.13±0.05^{c}
Quinoa sprouts (10%)	22.70±1.02^{b}	76.71±0.92^{c}	93.80±0.86^{d}	45.34 ± 1.06^{c}	3.80±0.07^{b}
Quinoa sprouts (15%)	18.90±0.88^{c}	72.92±1.31^{c}	103.39±1.02^{c}	53.21 ± 1.54^{b}	3.98±0.09^{ab}
Quinoa sprouts (20%)	13.75±1.30^{d}	85.63±1.10^{b}	121.02±1.08^{b}	67.73 ± 1.12^{a}	4.10±0.10^{a}

Results are expressed as mean ± SE.
Values in each column which have different letters are significantly different at ($P \leq 0.05$).
Malondialdehyde (MDA), Superoxide dismutase (SOD); CAT: catalase; GPx: Glutathione Peroxidase and Total antioxidant capacity (TAC).

Conclusion

The germination process improves on the phenolic content in quinoa seeds, enhanced the antioxidant potential, reduction in saponin and tannin content. Furthermore, quinoa sprouts improved inflammation and reduced the harmful impacts of induced oxidative stress in insulin resistance rats. Therefore the current study recommends the usage of dried quinoa sprouts in the formulation of functional foods in patients suffering from insulin resistance, abnormal glucose tolerance and overweight. Given the dearth of studies on the positive effects of quinoa on insulin resistance, it was determined that it would be suitable to assess the findings of this study as a crucial first step for future studies.

References

A.O.A.C. (2016). Official Methods of Analysis AOAC International (20th ed.; G. Latimer, ed.n) .

Abaza R.H.; Blake J. and Fisher E.J. (1968). Oxalate determination in: analytical problems encountered with certain plant species. Journal of AOAC: 51(4):963.

Abou-Seif HS, Hozayen WG and Hashem KS. (2019). Thymus vulgaris extract modulates dexamethasone induced liver injury and restores the hepatic antioxidant redox system. Beni-Suef University J Basic Applied Sci.;8[1]:1- 9.

Aebi, H. (1984). Catalase in vitro. Methods Enzymol., 105:121–126.

Akhtar, D. H., Iqbal, U., Vazquez-Montesino, L. M., Dennis, B. B and Ahmed, A. (2019). Pathogenesis of insulin resistance and atherogenic dyslipidemia in nonalcoholic fatty liver disease. Journal of Clinical and Translational Hepatology, 7(4), 362–370.

Albers, N.; Benderson, V. and Warnick, G. (1983). Enzymatic determination of high density lipoprotein cholesterol, Selected Methods, Clin. Chem,10 :91-99.

Al-Anazi, M.S.; El-Zahar, K.M.; and Rabie, N.A.-H.(2022). Nutritional and Therapeutic Properties of Fermented Camel Milk Fortified with Red *Chenopodium quinoa* Flour on Hypercholesterolemia Rats. *Molecules* , *27*, 7695.

Al-Qabba, M. M., El-Mowafy, M. A., Althwab, S. A., Alfheeaid, H. A., Aljutaily, T and Barakat, H. (2020). Phenolic profile, antioxidant activity, and ameliorating efficacy of chenopodium quinoa sprouts against CCl_4-induced oxidative stress in rats. *Nutrients*, *12*(10), 2904.

Al-Okbi SY, Hamed TE, Elewa TE, Ramadan AA, El-Karamany MF, and Bakry BA.(2021). Role of polar extracts from two quinoa varieties in prevention of steatohepatitis and cardiovascular diseases and improving glucose tolerance in rats. J Herbmed Pharmacol.; 10(1):93-101

Ali, O. (2019). Nutritional Value of Germinated Quinoa Seeds and Their Protective Effects on Rats Health Injected by Nicotine. *Egyptian Journal of Food Science*, *47*(2), 227-241.

Alkot AMF, Naeem SA, El Sayed SSMM, Algendy AMM, Hablas MGA.(2022). Study of the Protective Effect of Garlic Water Extract on Dexamethasone-Induced Hepatotoxicity and Insulin Resistance in Adult Male Albino Rats. IJMA; 4 [8]: 2606-2618.

Alona, S., Svitlana, M., and Sofia, N. (2021). Influence of some herbal mixtures on insulin resistance and glucose tolerance in rats. *PharmacologyOnLine*, *1*, 356-364.

An, T., Liu, JX., Yang, Xy., Lv, BH., Wu, YX., and Jiang,G J., (2021). Supplementation of quinoa regulates glycolipid metabolism and endoplasmic reticulum stress in the high-fat diet-induced female obese mice. *Nutr Metab (Lond)* **18**, 95 (2021).

Aryaeian N, Sedehi SK and Arablou T. (2017). Polyphenols and their effects on diabetes management: a review. Med J Islam Repub Iran.31:134.

Belfield, A. and D. Goldberg. (1971). Revised assay for serum phenyl phosphatase activity using 4- amino anti-pyrine enzyme. 12: 561-573.

Benincasa, P., Falcinelli, B., Lutts, S., Stagnari, F and Galieni, A. (2019). Sprouted grains: A comprehensive review. *Nutrients*, *11*(2), 1–29.

Benzie IF, and Strain JJ.(1996). The ferric reducing ability of plasma (FRAP) as a measure of antioxidant power: The FRAP assay. Anal Biochem;239:70‑6.

Bergmeyer, H.U., Scheibe, P., and Wahlefeld, A.W. (1978). Optimization of methods for aspartate aminotransferase and alanine aminotransferase. *Clinical chemistry*, *24*(1), 58-73.

Bonsens K E and Taussky D H (1984). Determination of serum creatinine .J ChemInv,27: 648-660.

Brady, K., Ho, Ch-T., Rosen, R.T., Sang, S and Karwe, M.V. (2007). Effects of processing on the nutraceutical profile of quinoa. Food Chemistry, 100, 1209-1216.

Burrin, JM and Price, CP. (1985). Measurement of blood glucose. Ann Clin Biochem. 22 (Pt 4):327-42.

Champman, D.; Castilla, R. and Compel, J. (1959). Evaluation of protein in foods. I-A: method for determination of protein efficiency ratio, Can. J. Biochem Physiol., 37: 679 – 686.

Chaney, S.G. (2006). Principles of Nutrition I: Macronutrients. In: Devlin, T.M. (Ed.) *Textbook of Biochemistry, with Clinical Correlation*, 6th ed. John Wiley and sons, New York, pp. 1071-1090.

Chevenne, D., Letailleur, A., Trivin, F., and Porquet, D., (1998). Effect of hemolysis on the concentration of insulin in serum determined by RIA and IRMA. Clin. Chem. 44,354–356.

Creus, A., Benmelej, A., Villafane, N and Lombardo, Y. B. (2017). Dietary Salba (*Salvia hispanica* L) improves the altered metabolic fate of glucose and reduces increased collagen deposition in the heart of insulin-resistant rats. Prostaglandins, Leukotrienes and Essential Fatty Acids, 121, 30–39.

Dakhili, S., Abdolalizadeh, L., Hosseini, S. M., Shojaee-Aliabadi, S and Mirmoghtadaie, L. (2019). Quinoa protein: Composition, structure and functional properties [Review]. Food Chemistry, 299, 125161.

Darwish, A.M.; Al-Jumayi, H.A.;and Elhendy, H.A. (2020). Effect of germination on the nutritional profile of quinoa (*Cheopodium quinoa* Willd.) seeds and its anti-anemic potential in Sprague Dawley male albino rats. *Cereal Chem.*, *98*, 315–327.

Draper, H and Hadley, M. (1990). Malondialdehyde determination as index of lipid per-oxidation. Methods Enzymol, 186: 421-431.

El-Kewawy HE and Morsy GM (2019). Beneficial role of quinoa and Nigella sativa seeds as antihyperuricemia in rats. *Bulletin of the National Nutrition Institute of the Arab Republic of Egypt*, *53*(1), 62-84.

Erfidan, S., Dede, S., Usta, A., Yüksek, V., and Çetin, S. (2022). The effect of quinoa (*Chenopodium quinoa*) on apoptotic, autophagic, antioxidant and inflammation markers in glucocorticoid-induced insulin resistance in rats. *Molecular biology reports*, *49*(7), 6509–6516

Fossati P; Prencipe L and Berti G (1980). Use of 3, 5 dichloro-2-hydroxybenzene sulfonic acid /4-aminophenazon chromogenic system in direct enzymatic assay of uric acid in serum and urine. Clin. Chem., 26: 227-231.

Fossati, P. and Praneipe, L. (1982). Enzymatic colorimetric method to determination triglycerides. Clin Chem, 28: 2077–80.

Fruchart, GG. (1982). LDL-cholesterol determination after separation of low-density lipoprotein. Rev Fr Des Laboratories, 103(7):117.

Guardianelli, L.M.; Salinas, M.V.; Brites, C.; and Puppo, M.C. (2022). Germination of White and Red Quinoa Seeds: Improvement of Nutritional and Functional Quality of Flours. *Foods*, *11*, 3272.

Harborne, J.B. (**1973).** Phytochemical methods. Chapman and Hall, London. p 113.

He, Y. N., Song, S. H., Li, C., Zhang, X. D., and Liu, H. J. (2022). Effect of germination on the main chemical compounds and 5 methylte-trahydrofolate metabolism of different quinoa varieties. *Food Research International*, 159, 111601.

Ibrahem, E., and Mohamed, G. (2021). Effect of Different preparatory processes on Improving the Nutritional Value of Quinoa (Chenopodium quinoa, Willd) and Studying its Effect on Obese Anemic Rats. *Egyptian Journal of Nutrition and Health*, *16* (2), 71-94.

Kajla, P.; Sharma, A.; and Sood, D.R. (2017). Effect of germination on proximate principles, minerals and anti-nutrients of flaxseeds. *Asian J. Dairy Food Res.*, *36*, 52–57.

Kandir S. and Keskin E. (2016). Serum IL-1β, IL-6, IL-10 and TNF-α Levels in Thyroidectomized Rats, Kafkas Üniversitesi Veteriner Fakültesi Dergisi, 22 : 297-300.

Le, L., Gong, X., An, Q., Xiang, D., Zou, L., Peng, L., and Wan, Y. (2021). Quinoa sprouts as potential vegetable source: Nutrient composition and functional contents of different quinoa sprout varieties. Food Chemistry, 357, Article 129752.

Lee, J., Kim, B., Kim, W., Ahn, C., Choi, H. Y., Kim, J. G., Kim, J., Shin, H., Kang, J. G., and Moon, S. (2021). Lipid indices as simple and clinically useful surrogate markers for insulin resistance in the U.S. population. *Scientific reports*, *11*(1), 2366.

Lopes, C. O., Barcelos, M. F. P., Vieira, C. N. G., de Abreu, W. C., Ferreira, E. B., Pereira, R. C., and de Angelis-Pereira, M. C. (2019). Effects of sprouted and fermented quinoa (*Chenopodium quinoa*) on glycemic index of diet and biochemical parameters of blood of Wistar rats fed high carbohydrate diet. *Journal of food science and technology*, *56*(1), 40–48.

Makkar, H.P.; Blummel, M.; Borowy, N.K and Becker, K. (1993). Gravimetric determination of tannins and their correlation with chemical and protein precipitating method. Journal of the Science of Food and Agriculture. 61(2): 161 165.

Maldonado-Alvarado, P., Pavón-Vargas, D. J., Abarca-Robles, J., Valencia-Chamorro, S., and Haros, C. M. (2023). Effect of Germination on the Nutritional Properties, Phytic Acid Content, and Phytase Activity of Quinoa (Chenopodium quinoa Willd).*Foods*, *12*(2), 389.

Mohamed, A. S., Elgazar, A. F., and Ibrahem, E. S. (2022a). Effects of diet supplemented with raw, germinated and cooked quinoa crop on obese rats. *Journal of Pharmaceutical Negative Results*, 4559-4567.

Mohamed, A. S., Elgazar, A. F., and Ibrahem, E. S. (2022b). Studying the effect of different methods of quinoa preparation on obese rats. *Egyptian Journal of Applied Science*, 37(7), 43-57.

Mahmoud, M.F.; Ali, N.; Mostafa, I.; Hasan, R.A.; Sobeh, M. (2022). Coriander Oil Reverses Dexamethasone-Induced Insulin Resistance in Rats. *Antioxidants* **2022**, *11*, 441.

Mohdaly, A.A.; Hassanien, M.F.R.; Mahmoud, A.; Sarhan, M.A.; and Smetanska, I. (2012). Phenolics Extracted from Potato, Sugar Beet, and Sesame Processing By-Products. Int. J. Food Prop. 16, 1148–1168.

Namrata, S., and Haripriya, A. (2022). Effect of germination on selected phytochemicals and antioxidant activity of quinoa (*Chenopodium quinoa*). *Indian Journal of Traditional Knowledge (IJTK)*, *21*(4), 876-882.

Ng, C.Y. and Wang, M. (2021). The functional ingredients of quinoa (*Chenopodium quinoa*) and physiological effects of consuming quinoa: A review. *Food Frontiers*.

Obadoni B.O. and Ochuko P.O. (2001). Phytochemical studies and comparative efficacy of the crude extracts of some homeostatic plants in Edo and Delta States of Nigeria. Global Journal of Pure and Applied Science. 8(2): 203-208.

Obaroakpo, J. U., Nan, W., Hao, L., Liu, L., Zhang, S., Lu, J., Pang, X., and Lv, J. (2020). The hyperglycemic regulatory effect of sprouted quinoa yoghurt in high-fat-diet and streptozotocin-induced type 2 diabetic mice via glucose and lipid homeostasis. *Food & Function, 11*, 8354– 8368.

Padmashree, A.; Negi, N.; Handu, S.; Khan, M.A.; Semwal, A.D.; Sharma, G.K. (2019). Effect of germination on nutritional, antinutritional and rheological characteristics of quinoa (*Chenopodium quinoa*). *Def. Life Sci. J., 4*, 55–60.

Pagalia DE and Valentine WN. (1967). Studies on the quantitative and qualitative characterization of erythrocyte glutathione peroxidase. J Lab Clin Med; 70 (1) :158–69.

Patton CJ and Crouch SR (1977). Enzymatic colorimetric method to determination urea in serum. Anal. Chem., 49: 464.

Pellegrini, C., Fornai, M., Antonioli, L., Blandizzi, C., and Calderone, V. (2019). Phytochemicals as novel therapeutic strategies for NLRP3 inflammasome - related neurological, metabolic, and inflammatory diseases. *International Journal of Molecular Sciences*, *20*(12), 2876.

Pereira, E., Encina-Zelada, C., Barros, L., Gonzales-Barron, U., Cadavez, V and Ferreira, I. (2019). Chemical and nutritional characterization of *Chenopodium quinoa Willd* (quinoa) grains: A good alternative to nutritious food. *Food Chemistry*, *280*, 110–114.

Pilco-Quesada, S.; Tian, Y.; Yang, B.; Repo-Carrasco-Valencia, R.; and Suomela, J.P. (2020). Effects of germination and kilning on the phenolic compounds and nutritional properties of quinoa (*Chenopodium quinoa*) and kiwicha (*Amaranthus caudatus*). *J. Cereal Sci.* , *94*, 102996.

Pinheiro, S. S., Anunciação, P. C., de Morais Cardoso, L., Della Lucia, C. M., de Carvalho, C. W. P., Queiroz, V. A., and Sant'Ana, H. M. (2021). Stability of B vitamins, vitamin E, xanthophylls and flavonoids during germination and maceration of sorghum (Sorghum bicolor L.). *Food Chemistry*, *345*, 128775.

Poualeu-Kamani, S., Kamgaing-Waguia, J., Miaffo, D., Nchouwet, M., Demeni Kadji, C., Wego Kamgaing, M. T., Douho Djimeli, R., Mzoyem Ngnitedem, J., Kamanyi, A., and Wansi Ngnokam, S. (2022). Efficacy of *Emilia coccinea* aqueous extract on inhibition of α-amylase enzyme activity and insulin resistance in dexamethasone treated-rats. *Metabolism open*, *15*, 100193.

Prieto, P.; Pineda, M. and Aguilar, M. (1999). Spectrophotometric quantitation of antioxidant capacity through the formation of a phosphor molybdenum complex: specific application to the determination of vitamin E. Analytical biochemistry, 269(2), pp.337–41.

Reeves, P. G., Nielsen, F. H., and Fahey, G. C., Jr (1993). AIN-93 purified diets for laboratory rodents: final report of the American Institute of Nutrition ad hoc writing committee on the reformulation of the AIN-76A rodent diet. The Journal of nutrition, 123(11), 1939–1951.

Reason, D.; Watts, M.; Devez, A. (2015). Quantification of Phytic Acid in Grains. British Geological Survey. Available online: https://nora.nerc.ac.uk/id/eprint/512947/ (accessed on 10 September 2022).

Rohling, M., Martin, T., Wonnemann, M., Kragl, M., Klein, H. H., Heinemann, L., and Kempf, K. (2019). Determination of postprandial glycemic responses by continuous glucose monitoring in a real-world setting. *Nutrients*, *11*(10), 2305.

Ruijuan G.; Xiaowen C.; Guotao B.; Xiang L.; Yan D.; Xuebo L.; Lijun S. and Yutang W. (2021). Effects of household cooking methods on changes of tissue structure, phenolic antioxidant capacity and active component bio accessibility of quinoa, Food Chemistry,350: 129138.

Safaeian L, Zolfaghari B, and Ghazvini M. (2018). The effects of hydroalcolic extract of *Allium elburzense Wendelbo* bulb on dexamethasone –induced dyslipidemia hyperglycemia and oxidative stress in rats. Res Pharm Sci;13(1):22–9.

Salgado, A.L., Carvalho, L.d., Oliveira, A.C., Santos, V.N., Vieira, J.G., and Parise, E.R. (2010). Insulin resistance index (HOMA-IR) in the differentiation of patients with non-alcoholic fatty liver disease and healthy individuals. Arquivos de gastroenterologia, 47(2), 165–169.

Shittu, S. T., Lasisi, T. J., Shittu, S. A., Adeyemi, A., Adeoye, T. J., and Alada, A. A. (2021). *Ocimum gratissimum* enhances insulin sensitivity in male Wistar rats with dexamethasone-induced insulin resistance. *Journal of Diabetes & Metabolic Disorders*, *20*, 1257-1267.

Snedecor, G.W and Cochron, W.G. (1989). Statistical methods. 8th edi, USA, Lowa. State Univ. Press, Ames, Lowa.

Song, K., Park, G., Lee, H. S., Choi, Y., Oh, J. S., Choi, H. S., and Chae, H. W. (2021). Prediction of insulin resistance by modified triglyceride glucose indices in youth. *Life*, *11*(4), 286.

Spitz, D.R and Oberley, L.W. (1989). An assay for superoxide dismutase activity in mammalian tissue homogenates. Anal Biochem; 179:8- 18.

Sun-Ju, K., Chiami, K., Tatsuro, S., Katsuichi, S., Naoto, H., Shigenobu, T., and Abdelgawad, H. (2006). Effect of Natural Light Periods on Rutin, Free Amino Acid and Vitamin C Contents in the Sprouts of Common (Fagopyrum esculentum Moench) and Tartary (F. tataricum Gaertn.) Buckwheats. *Food Science and Technology Research*, *12*(3), 199-205.

Tazrart, K.; Lamacchia, C.; Zaidi, F.; and Haros, M. (2016). Nutrient composition and in vitro digestibility of fresh pasta enriched with Vicia faba.J. Food Compos. Anal.,47, 8–15.

Wahba, H.M., Mahmoud, M.H. and El-Mehiry, H.F. (2019). Effect of quinoa seeds against cisplatin toxicity in female rats. *Journal of Advanced Pharmacy Education and Research*, *9*(3), 47.

Wahlefeld, AW. (1974). Enzymatic Determination of triglycerides. Methods of enzymatic analysis. HU. Bergmeyer, Ed. Academic Press, New York, 5:1831-1835.

Weichse - lbaum, TF (1946). An accurate and rapid method for the determination of protein in small amount of blood serum and plasma .Am .J.Clin Path, (16):40.

Yawadio Nsimba, R.; Kikuzaki, H.; and Konishi, Y. (2008). Antioxidant activity of various extracts and fractions of Chenopodium quinoa and Amaranthus spp. seeds. Food Chem. 106, 760–766.

تأثير براعم الكينوا على مؤشرات الالتهاب والإجهاد التأكسدي في الفئران المقاومة للأنسولين

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الكينوا هي واحدة من الحبوب الزائفة التي تحتوي على نسبة عالية من المغذيات الكبيرة والمغذيات الدقيقة. يسجل هذا البحث التغيرات في المحتوى الفينولي والنشاط الحيوي المضاد للأكسدة لبذور الكينوا أثناء انبات بذور الكينوا. كان الهدف من هذه الدراسة هو تقييم آثار براعم الكينوا على مقاومة الأنسولين التي سببتها مادة الديكساميثازون في الفئران. حيث تم تقسيم خمسة وثلاثين من ذكور الفئران البالغة من نوع Sprague-Dawley إلى خمس مجموعات. المجموعة (١) التي تم تغذيتها على النظام الغذائي الأساسي كمجموعة ضابطة سالبة، بينما تم حقن مجموعات الفئران الأخرى (ن = ٢٨) يومياً تحت الجلد (الصفاق) بمادة ديكساميثازون بجرعة ١ مجم / كجم / ٣ مرات اسبوعيا لمدة ٦ اسابيع لإحداث مقاومة للأنسولين. ثم تم الحفاظ على المجموعة (٢) من الفئران المقاومة للأنسولين (كمجموعة ضابطة موجبة) وأعطيت المجموعات الثلاث الأخرى ١٠ و ١٥ و ٢٠٪ من براعم الكينوا. أظهرت النتائج أن إنبات الكينوا يحسن المحتوى الفينولي والنشاط المضاد للأكسدة لبذورالكينوا وتقلل من العوامل المضادة للتغذية . وفي الوقت نفسه، أدى استخدام براعم الكينوا إلى عكس المقاييس الكيميائية والحيوية التي يسببها ديكساميثازون في الفئران بشكل كبير، مما تسبب في تغيرات كبيرة في زيادة وزن الجسم ، ومعدل الاستفادة من الغذاء، والمأخوذ من الغذاء، وانخفاض وظائف الكبد والكلى ومستوى الدهون في الدم. كما أنه يقلل من الأنسولين والجلوكوز في حالة الصيام ومدى تحمل الجلوكوز ومقاومة الانسولين . علاوة على ذلك ، زادت بشكل ملحوظ الإنزيمات المضادة للأكسدة (SOD ، CAT، Gpx ، TAC) بينما انخفض مستوى MDA، والسيتوكين في الدم مثل (IL-β1 و TNF-α) في الفئران المقاومة للأنسولين. وفي الختام، يجب ان يؤخذ فى الاعتبار دمج براعم الكينوا كمكونات غذائية وظيفية نظرا لآثارها الإيجابية وفوائدها الصحية فى تقليل مؤشرات الالتهاب والإجهاد التأكسدى للفئران المقاومة للأنسولين .

الكلمات المفتاحية : الكينوا- مقاومة الانسولين – مدى تحمل الجلوكوز الديكساميثازون - الالتهاب - الاجهاد التأكسدي .