Protective Effects of Quinoa on Hepatotoxicity in Male Rats Exposed to Chlorfenapyre

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Abstract: Chlorfenapyr, a member of a new class of chemicals called pyrroles, is widely used in agriculture as insecticide, fungicide, miticide and rodenticide. Quinoa (*Chenopodium Quinoa* Willd), ancient seeds, are classified as a whole grain cereal and is considered as a natural functional food because of the presence of bioactive phytochemicals, minerals, vitamins, fatty acids and the high amount of protein content. The consumption of quinoa effects in humans is not widely studied, so the present study was aimed to determine the effect of dietary supplementation with quinoa seeds (QS) to give more protection against hepatotoxicity induced by chlorfenapyre (Chf) exposure in rats. Twenty adult male rats were divided into 4 equal groups; group (I) as a control was fed on the basal diet only, group II (QS at 30% from the basal diet)), group III (Chf at 1/3 LD_{50}, was fed on basal diet only) and group IV (Chf at 1/3 LD_{50} plus QS at 30% from the basal diet). Chf was orally administered to rats and the experimental period was four weeks. Hematological, biochemical parameters and lipid profile as well as liver histopathological changes were determined. Results showed significant decrease ($p < 0.05$) in gained weight of body and significant elevation ($p < 0.05$) in the relative liver weight of groups III and IV, significant changes in some hematological parameters compared with the control group. Also, the liver function enzymes activity, total cholesterol, high density cholesterol (HDL), very low density lipoproteins (VLDL) and triglycerides in group III were significantly elevated ($p < 0.05$) compared with control group. On the other hand groups II and IV which supplemented with QS in their fed showed mitigation of the previous effects and these results confirmed with the histopathological examination of liver tissue.

These findings suggested that quinoa seeds may act as a moderate protective agent which can reduce most of the adverse effects of chlorfenapyre exposure in rats.

Keywords: Chlorfenapyre, Quinoa, Hepatotoxicity, Relative organ weight, Hematological, Histopathological.

1.Introduction

Agriculture is the main consumer of pesticides (about 85% of production around the world) to control different pests. In addition, we use pesticides in public health to manage vector-borne diseases (e.g., malaria) and undesired plants (e.g., grass and weeds) in ornamental landscaping, gardens, and parks. Also they are used in inhibiting the growth of bacteria, fungi, and algae or preventing insects, in electrical instruments, refrigerators, paper, carpets, paint, and food packaging stuff **(Gilden et al., 2010).** Accidently exposure to pesticides results in highly hazardous effects to human being and other non-target living organisms. Exposure to pesticides may be directly from occupational, agricultural, and household use, or transferred indirectly through food

Chlorfenapyr (Chf) is a broad-spectrum insecticide and acaricide, which used in agriculture to control leafminers, thrips, mites and many pests, and in public health against termites, cockroaches, ants, bedbugs, flies, spiders, centipedes and other different insect pests. Chf is the first commercial pesticide from a new class called halogenated pyrroles which produced by bacteria and it is moderately toxic to mammals. The persistent nature of Chf in soil and on vegetation makes it likely that birds and animals would continue to ingest the insecticide for many weeks beyond the last application **(Albers *et al*., 2006**). Chf is widely used in Egypt on grapes, citrus, tomato and green bean. Residues of Chf were detected in grapefruit, nectarine and granadilla samples imported from South Africa and Colombia; also it was one of pesticides which detected in exported European Union mandarins over the permissible limits **(Sdeek *et al*., 2016; Badawy *et al* 2020).**

Chf is a pro-insecticide, which means that it acquires insecticidal properties after metabolic activation and its mode of action rely on disrupts ATP production and causes cell death **(Albers *et al*., 2006**). The body get ride about 90% of the administered dose by 7 days. The main elimination route was through faeces, while the other route was urinary with a minor role **(Metruccio and Boobis, 2012).**

Quinoa seed (QS) is considered one of the best cereal protein sources, as its protein levels are higher than those present in wheat, rice, maize, barley, corn, rye, and sorghum. The interest in QS has risen due to its ability to adapt with environmental conditions; it is resistant for frost, drought and salinity; it grows on dry and arid soils and heights. The nutritional quality of QS is well determined; its content of protein ranges 13–17 g/100 g and considered as a gluten-free protein source; rich in starchy carbohydrate components; unsaturated fats; dietary fiber; phytochemicals ; micronutrients and have a higher nutritional value compared with other cereals **(Matiacevich *et al.* 2006)**. It is also has been used by the National

Aeronautics and Space Administration (NASA) because of its versatility in supplying the needs of humans during space missions **(Cooper, 2015).** This supports the use of QS which described as "one of the 21st century's grains" **(Li *et al.*, 2018)**. The revival and reintroduction of QS into the diet is linked to the epidemiological condition, which involves diseases that present risk factors that can be minimized with a balanced nutritious diet, in which QS has a main role, being considered as a "superfood" **(Tang *et al.*, 2015).** Although all these health benefits, consumption of quinoa is still limited due to many reasons, such as high import costs of the grain and lack of knowledge regarding its benefits among consumers. The main target of this research was to conducted effect of dietary supplementation with Quinoa seeds at concentration 30% of the meal to give more protection against the hazard effect which revealed as a result of Chlorfenapyre exposure in male rats.

2. Materials and Methods

2.1. Experimental Materials:

Chlorfenapyr (Corps Top 24% SC) was obtained from Agrimar Company for Commercial Agencies. Quinoa seeds was purchased from CEDAR, Packed by Groupe PHONICIA Inc. Montreal, Canada QC.H4S1T2.

2.2. Experimental design:

Twenty adult male albino rats, weighing 150 -160 g were obtained from Faculty of Medicine; Alexandria University. Rats were allowed to acclimatize for 14 days before the initiation of the experiment under laboratory conditions (12 h light / 12 h dark, 22– 26 °C., 40–70% humidity) in stainless steel cages and provided with commercial basal diet and water *ad libitum*. Animals were divided into equal 4 groups , and orally treated 5 doses /week over a period of 4 weeks as the following: Group I: Rats were served as control and given commercial basal diet. Group II: Rats were given QS at 30% from the basal diet Group III: Rats were given Chf 180 mg/kg bw which represent 1/3 LD_{50} (Oral LD_{50} was 544.3 mg/ kg bw; according to Department of Mammalian Toxicology, Pesticide Central Laboratory, Agriculture Research Center). Group IV: Rats were given Chf 180 mg/kg bw plus QS at 30% from the basal diet. Chf was orally administrated to animals by esophageal intubation and the body weights of all groups were recorded weekly. At the end of the experiment the animals were sacrificed and dissected, then the liver was removed, rinsed in saline solution (0.9% NaCl), dried on filter paper and individually weighted from all animals and measured the relative liver weight (liver weight: body weight).A specimen of the liver was fixed immediately in 10% buffered formalin for histological examination.

2.3. Blood sample collection:

The rats were fasted overnight at the end of the experimental period then, anaesthetized, sacrificed and blood samples were collected from the aorta. The blood was placed immediately into two tubes; the first tube containing EDTA for hematological analysis and the second tube without anticoagulant for serum preparation was allowed to stand at room temperature for 30 min. till clotted and centrifuged at 1,200 g using Sigma 3K30 bench centrifuge for 15 min to separate the serum; then stored at -20 ºC for the biochemical studies.

2.4. Hematological analysis:

Non-coagulated blood samples were analyzed for peripheral blood cell indicators; white blood cells (WBC's) count, red blood cells (RBC's) count, hemoglobin content (Hb), Hematocrite percentage (Hct) and platelet (PLT) count were estimated as described by **Dacie and Lewis (1991).** Calculation of erythrocyte indices including mean corpuscular volume (MCV), mean corpuscular haemoglobin (MCH) and mean concentration of corpuscular haemoglobin (MCHC) were recorded. Also the Leukocyte Formula (Lymphocytes, Neutrophils, Monocytes and Eosinophils percentages) were reported.

2.5. Biochemical studies:

Sera samples were used to estimate aspartate aminotransferase (AST) and alanine aminotransferase (ALT) activities according to **Retiman and Frankel, 1957**. Also, total cholesterol, high density lipoprotein (HDL) and triglycerides levels were determined according to **Trinder, 1969; Grove, 1979** and **Fassati and Prencipe, 1982** respectively. Low density lipoprotein (LDL) and very low density lipoprotein (VLDL) were calculated by using the method of **Friedewald *et al.*, 1972.**

2.6. Histopathological studies:

Dehydrated the fixed liver specimens by the standard procedures, embedded in paraffin, sections were cut to about 5 µm thick, stained with haematoxylin and eosin (H and E) stains and then examined using light microscope **(Drury, 1980).**

2.7. Statistical analysis:

Data were analyzed using one-way analysis of variance (ANOVA) followed by the Student–Newman–Keuls test to estimate significance between many experimental groups, and were expressed as mean ± standard error (SE). The statistical significance threshold was set at $p<0.05$.

3. Results and Discussion

3.1. Gained body weight and relative liver weight:

Changes in body, organ and relative organ weights are determined phenomena in toxicological

studies for assessing toxicity. The liver, which is the site of xenobiotic metabolism, increased in size was due to the increased functional load **(Heikal *et al.*, 2011).** In the present study, the animals which exposed to chlorfenapyre (groupIII) showed a significant decrease ($p < 0.05$) of gained body weight around (82 gm) while in control (group I) it was around (102 gm), this may be due to decreases in feed consumption which explained after the dissection of the treated animals where the high degree of inflammation in the small and large intestine was noticed. On the other hand, animals of group (II) represented a significant decrease ($p < 0.05$) in the gained body weight and that may be the effect of quinoa as a healthy grain in the obesity cases treatment to decrease the weight. Also the relative liver weights (relative to body weight) of group (III) animals were significantly increased ($p < 0.05$) by about (149%) in comparing to control animals (group I). When Chf was combined with QS the relative liver weights reduced that effect (122.2%) as shown in table (1). The above obtained results in agree with another studies which showed statistically significant reduction in feed consumption and consequently, decrease in body weight; while the absolute and relative liver weights were increased across the Chf treated groups **(Metruccio and Boobis, 2012; Sleem, *et al.*, 2019).**

Table (1) Changes in the Relative Liver Weights and gained body weight of Male Rats after 28 days of Treatment with Chlorfenapyre (180 mg/kg bw), Quinoa seed(30% of basal diet) and Their Combination.

Animal Group	Relative liver weight (%)	% Change [a]	Gained body weight (gm)
Control(I)	3.63 ± 0.08^{c}	-	101.7 ± 1.2^{a}
QS (II)	3.5 ± 0.21^{c}	-3.5	95.0 ± 1.1^{b}
Chf (III)	5.4 ± 0.33^{a}	148.8	82.3 ± 1.4^{d}
Chf+QS (IV)	4.4 ± 0.1^{b}	122.2	90.3 ± 0.8^{c}

Values are expressed as means (Five rats) ± standard error (SE).
Values in column with different letters are significantly different at ($p < 0.05$).
[a] Percentage of decrease (-) or increase in liver weights due to the Chf treatment relative to control = liver Weight of treatment / liver Weight of control.

3.2. Hematological studies:

Detrmination of hematological parameters can be used as indicators of toxicity and have a broad potential application in environmental and occupational monitoring. Therefore, the variations of these parameters can be used for prediction and diagnosis of pesticide toxicity **(Haider and Rauf 2014).** WBCs have a major role in the immune system, because of their main defensive function and they reacted immediately to the change in medium as a result of toxicant. The data recorded a significant increase ($p < 0.05$) in the WBC count in the group treated with Chf (group III), An increase in white blood cells is known as leukocytosis which may be due to the activation of defense mechanism in treated animals and this may be attributed to the pesticide caused enteritis, allergic reactions and inflammation which observed after the dissection at the end of the experiment **(Kalender *et al.*, 2006),** while consuming QS overcame these toxic effects (group IV). Significant increase ($p < 0.05$) in the platelet concentration was observed in animals of (group III and IV), elevated platelet concentration exhibited thrombocytosis which may be congenital or due to abnormal platelets production **(Riaz and Yousafzai 2017**).On the other hand there were non-significant changes in values of Hb, Hct as well as RBC among the rats which treated with Chf (groups III and IV) as compared to control animals (table 2), similar results were Table (3) illustrates the immunotoxic potential indices and revealed significant changes ($p < 0.05$) in the Leukocyte formula (Lymphocytes, Neutrophils, Monocytes, Eosinophils and Basophils) due to Chf treatment. In comparing with control there was significant increase in the monocytes percentage, known as monocytosis , and that may due to inflammation caused by Chf exposure(group III).There were significant reduction in lymphocyte, basophil and eosinophil percentages in group(III) animals compared with control (group I).The low level of basophil may be due to a severe allergic reaction and the abnormally low eosinophil percentage can be the result of intoxication from Chf or excessive production of cortisol. Cortisol is a hormone naturally produced by the body. Low eosinophil and lymphocyte percentages in the general population are associated with increased short-term incidence of heart failure and coronary death **Shah *et al.*, (2016).** Erythrocytes indices (MCV, MCH and MCHC levels) were showed significantly increase ($p < 0.05$) in MCH and MCHC in respect to the control values as shown in table (4) and that can be explained as the conditions where hemoglobin is present outside of red blood cells due to red blood cell destruction or fragility. Also this recorded by **Metruccio and Boobis, 2012.**

Increase in MCHC could be due to the increased activity of bone marrow and deficiency of some hemopoietic factors(**Riaz and Yousafzai 2017)**.

These results agreed with **Eman and Basem (2008); Jasper *et al*., (2012)** who confirmed that Chf induce a significant increase in the reactive oxygen species production and RBCs membrane is very fragile and susceptible to oxidative stress that cause destruction and hemolysis of RBCs in the vascular system, and explained the observed increase in the WBC count which related to the inflammatory response to the oxidative damage by Chf.

Table (2) Peripheral blood cell indicators of male rats orally administrated to Chlorfenapyre (180 mg/kg bw) Quinoa seeds (30% of basal diet) and their combination for 28 days.

Animal Group	Parameters				
	WBC (10^3 / µl)	RBCs (10^6 / µl)	Hb (g/dl)	Hct (%)	PLT (10^3/ µl)
Control(I)	7.97 ± 0.35^a	7.79 ± 0.13^a	14.38 ± 0.32^a	45.70 ± 0.76^a	759.2 ± 19.77^a
QS (II)	7.37 ± 0.67^a	7.93 ± 0.19^a	14.52 ± 0.36^a	46.70 ± 0.94^a	785.6 ± 12.51^a
Chf (III)	14.02 ± 0.80^c	7.49 ± 0.05^a	14.52 ± 0.2^a	45.40 ± 0.35^a	780.2 ± 31.61^a
Chf+QS (IV)	10.92 ± 0.85^b	7.49 ±0.08^a	14.06 ± 0.19^a	44.50 ± 0.76^a	954.4 ± 36.54^b

Values are expressed as means (Five rats) ± standard error (SE).
Values in column with different letters are significantly different at ($p \leq 0.05$).

3.3. Biochemical studies:

3.3.1. Liver function enzymes:

Liver is the major site of toxicants metabolism and it is considered as the most preferable organ for chlorfenapyr with bile retention and metabolic disruption, therefore decrease its ability to absorb nutrients **(Albers, 2006).** The results in table (5) showed significant elevation ($p < 0.05$) in ALS and AST activities by 126% and 136% respectively in (group III) rats in comparison with control (group I) rats. The elevation of these liver enzymes may be due to the drastic condition caused by the Chf and/or severe hepatocellular damage as confirmed by the histopathological alteration. These results were in agreement with previously recorded by **Metruccio and Boobis, (2012).** When animals in (group IV) supplemented with quinoa, level of hepatic enzymes were significantly reduced and also the animals in (group II) showed better results in hepatic enzymes activities compared with control animals (group I) which illustrate the effect of quinoa on the liver and that results were supported by **Eman and Basem (2008); Saxena *et al.* (2017).**

Table (3) Immunotoxic potential of male rats administered to Chlorfenapyre (180 mg/kg bw), Quinoa seeds (30% of basal diet) and Their Combination for 28 days: The Leukocyte Formula

Animal Group	Parameters				
	Lymphocytes (%)	Monocytes (%)	Neutrophils (%)	Eosinophils (%)	Basophils (%)
Control(I)	77.8 ±1.52^b	8.8 ± 0.43^b	10.0 ± 0.52^a	1.68 ± 0.21^b	4.83 ± 0.27 ^{ab}
QS (II)	74.4 ±1.97^{ab}	8.5 ± 0.29^b	10.2 ± 0.61^a	3.68 ± 0.24^d	4.03 ± 0.36 ^{b}
Chf (III)	73.4 ±1.39^{ab}	10.5 ± 0.51^a	10.9 ±0.38^a	0.84 ± 0.09^a	3.00 ± 0.23 ^{c}
Chf+QS (IV)	69.5 ± 1.35^a	8.1±0.17^b	16.0 ± 0.45^b	2.34 ± 0.24^c	5.40 ± 0.15 ^{a}

Values are expressed as means (Five rats) ± standard error (SE)
Values in column with different letters are significantly different at ($p \leq 0.05$).

Table (4) Erythrocytes indices of male rats orally administrated to Chlorfenapyre (180 mg/kg bw), Quinoa seeds (30% of basal diet) and Their Combination for 28 days.

Animal Group	Parameters		
	MCV (fL)	MCH (Pg)	MCHC (g/dL)
Control(I)	58.73 ± 0.33^a	18.863 ± 0.06^{bc}	31.16 ± 0.12^b
QS (II)	58.86 ± 0.21^a	18.46 ± 0.02^c	30.66 ± 0.18^b
Chf (III)	60.16 ± 0.45^a	19.43 ± 0.26^a	32.3 ±0.24^a
Chf+QS (IV)	58.96 ± 0.37^a	19.00 ±0.15^b	31.36 ± 0.18^b

Values are expressed as means (Five rats) ± standard error (SE)
Values in column with different letters are significantly different at ($p \leq 0.05$)

3.3.2. Lipid profile:

Hypercholesterolemia is a main factor in the predictive equations for cardiovascular disease; it may be due to impairment of liver or changes in liver chemistry and morphology or because of decrease in cholesterol catabolism which in turn increase the serum cholesterol level. In combination, quinoa grains components may have a high significant impact on blood lipids and cardiovascular disease, which appeared to be complications of hypercholesterolemia **Halaby *et al*., (2017).**

The present data showed significant increase ($p < 0.05$) in total cholesterol (24%), LDL (70%), triglycerides (53%) and VLDL (53%), while the HDL cholesterol value was significantly decreased ($p < 0.05$) by 46% in rats treated with Chf (group III) compared with control as shown in table (5). On the other hand the rats in group II which consumed QS showed effectively reduction in total cholesterol (9%), LDL (26%), triglycerides (37%), and VLDL (38 %) when compared to the control group, while the HDL value was. The combination effect of QS in the diet (group IV) showed less hazard effect of Chf on the lipid profile parameters, which proposed that the protein present within the quinoa reduced the reabsorption of bile acids and decreased cholesterol synthesis in the liver (**Paśko *et al* 2010*;* Halaby *et al*., 2017; Hafez, 2018).** Similarly, other studies investigated chlorfenapyr toxicity in rats and mice at different doses and described that, the increase in serum lipid profile may be linked to the catecholamines stimulation, which results in lipolysis and produce more fatty acids, or could be as a result of blockage in liver bile ducts leading to decreasing or reduction of its secretion to the duodenum subsequently resulting in cholestasis **(Eman and Basem 2008; Metruccio and Boobis, 2012).**

Table (5) Liver function activities and Lipid profile of male rats orally administrated to Chlorfenapyre (180 mg/kg bw), Quinoa seeds (30% of basal diet) and Their Combination for 28 days.

Animal Group	Parameters						
	AST (U/L)	ALT (U/L)	Total Cholesterol (mg/dl)	HDL (mg/dl)	LDL (mg/dl)	Triglycerides (mg/dl)	VLDL (mg/dl)
Control(I)	163.3± 3.3^{b}	76.0 ±3.1^{b}	113.1± 1.5^{b}	75.4± 0.8^{a}	23.1± 0.8^{b}	72.7 ± 0.4^{b}	14.6± 0.1^{b}
QS (II)	133.3± 3.3^{c}	56.6± 1.6^{c}	103.2± 0.1^{b}	77.4 ±1.7^{a}	17.1± 1.8^{b}	45.6 ± 0.8^{c}	9.1 ± 0.2^{c}
Chf (III)	206.6± 12^{a}	103.3± 8.8^{a}	148.4± 3.7^{a}	40.6± 0.7^{c}	76.8 ±4.6^{a}	154.7 ±4.5^{a}	30.9 ± 0.9^{a}
Chf+QS (IV)	128.3 ±1.6^{c}	50.0± 0.1^{c}	120.2 ±3.4^{b}	68.5 ±2.0^{b}	30.3±3.1^{b}	80.5 ±1.6^{b}	13.7 ± 0.3^{b}

Values are expressed as means (Five rats) ± standard error (SE)
Values in column with different letters are significantly different at ($p \leq 0.05$).

4.3. Histopathological examination

The liver is composed from hepatic lobules consisting of radially organized strands of hepatocytes that extend from the central vein to lobular periphery. The blood sinusoids, which are lined with the endothelial cells and Kupffer cells, separate hepatocytes strands from each other. Histological alterations provide a quick means for detecting the effects of pesticides in different animal tissues and organs. **(Khaldoun-Oularbi *et al,* 2013).** Figure 1(A & B) showed histological structure of liver in control (group I) and QS (group II) treated rats with normal architecture and pathological free hepatic central vein (CV). While animals in group (III) the liver examination showed many changes (Fig.1C) such as focal hepatic degeneration (HD), vacuolar degeneration (VD), Congestion in central vein and sinusoidal dilatation (S) were dilated with increase kupffer cells (KC); also inflammation within the portal triad (Fig.1D) was noticed. The inflammatory cells were aggregated in portal tracts and present as differential foci in the liver parenchyma act as a defense mechanism due to irritation of toxic material and for the same reason the kupffer cells were activated **(Kolios *et al.,* 2006).** While consuming QS in the diet showed preservation of liver tissue nearly to its normal architecture which confirmed the protective role of QS against Chf induced liver damage (Fig.1E).

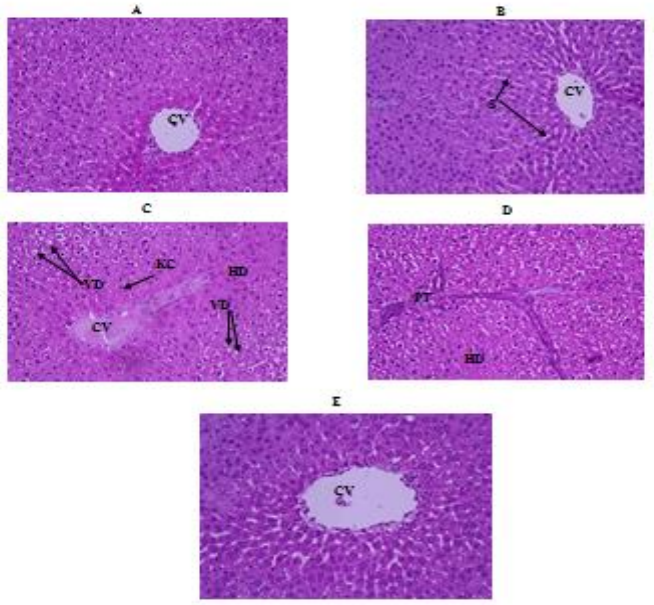

Fig.1(A): Liver ection of control showed central vein (CV) with normal architecture (H&E stain X400).
(B): Liver section of group II treated rats showed pathological free hepatic central vein with normal architecture (H&E stain x 400).
(C): Liver section of rat treated with 180 mg Chf / Kg bw showed architecture destruction and vacuolar degeneration (VD), Congestion in central vein and sinusoidal dilatation with increase kupffer cells (arrows) (H&E stain x 400).
(D): Liver section of rat treated with 180 mg Chf / Kg bw showed) focal hepatic degeneration (HD), inflammation within the portal triad (Pt) (H&E stain x 400).
(E): Liver section of rat treated with 180 mg Chf / Kg bw and 30%QS of basal diet histopathological changes denoting recovery (H&E stain x 400).

Conclusion

The present study demonstrated that sublethal oral administration of Chlorfenapyre for 28 days caused cytotoxic changes in the body and liver weights, hematological parameters lipid profile and hepatic biomarkers which reflect hazardous effects at various levels to non-target organisms. Consuming Quinoa seeds at 30% of the basal diet retarded the Chf induced hepatotoxicity by its protective effect because of the great structure and components which the QS contain. So that QS can be used as a dietary system to improve the overall health to overcome the effect of toxicants.

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التأثيرات الوقائية للكينوا على السمية الكبدية في ذكور الجرذان المعرضة للكلورفينابير

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الملخص العربى

يستخدم الكلورفينبير، وهو عضو في فئة جديدة من المواد الكيميائية تسمى pyrroles ، على نطاق واسع في الزراعة كمبيدات حشرية ومبيدات للفطريات ومبيدات للقراد والقوارض. الكينوا (Chenopodium Quinoa Willd) ، بذور قديمة ، تصنف على أنها حبوب حبوب كاملة وتعتبر كغذاء وظيفي طبيعي بسبب وجود المواد الكيميائية النباتية النشطة بيولوجيا والمعادن والفيتامينات والأحماض الدهنية وكمية عالية من البروتين. لم يتم دراسة تأثيرات استهلاك الكينوا على البشر على نطاق واسع ، لذلك هدفت الدراسة الحالية إلى تحديد تأثير ادخال ببذور الكينوا (QS) فى النظام الغذائى لإعطاء مزيد من الحماية ضد السمية الكبدية التي يسببها التعرض للكلورفينبير (Chf) في الفئران. تم تقسيم عشرين ذكور جرذ بالغ إلى 4 مجموعات متساوية. تم تغذية المجموعة الاولى(I) تمثل الكنترول على النظام الغذائي الأساسي فقط ، المجموعة الثانية(II) تم فيها ادخال(QS) بنسبة 30٪ من النظام الغذائي الأساسي ، المجموعة الثالثة(III) وفيها تمت معاملة الحيوانات بـ (Chf) عند 1/3 قيمة الLD_{50} و إطعامها على النظام الغذائي الأساسي فقط والمجموعة الرابعة (IV) وفيها تمت المعاملة بـ (Chf) بمعدل 1/3 من قيمة الـ LD50 بالإضافة إلى QS بنسبة 30٪ من النظام الغذائي الأساسي. تم إعطاء Chf عن طريق الفم للجرذان وكانت الفترة التجريبية أربعة أسابيع. تم تحديد العوامل الدموية والكيميائية الحيوية و صورة الدهون الكاملة وكذلك التغيرات النسيجية التشريحية للكبد. أظهرت النتائج انخفاضًا معنويًا ($p < 0.05$) في وزن الجسم المكتسب وارتفاع معنوي ($p < 0.05$) في الوزن النسبي للكبد للمجموعتين الثالثة والرابعة ، وتغيرات معنوية في بعض معاملات الدم مقارنة مع مجموعة الكنترول. كما ارتفع نشاط إنزيمات وظائف الكبد ، والكولسترول الكلي ، والكوليسترول عالي الكثافة (HDL) ، والبروتينات الدهنية منخفضة الكثافة (VLDL) والدهون الثلاثية بشكل ملحوظ ($p < 0.05$) مقارنة مع مجموعة الكنترول. من ناحية أخرى ، أظهرت المجموعتان الثانية والرابعة المكملة بـ QS في تغذيتهما التخفيف من الآثار السابقة وتأكدت هذه النتائج من خلال الفحص التشريحى لأنسجة الكبد.

تشير هذه النتائج إلى أن بذور الكينوا قد تعمل كعامل وقائي معتدل يمكن أن يقلل من معظم الآثار الضارةالناجمة عن التعرض للكلورفينابيرا في الفئران.