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Effect of Fenvalerate Toxicity on Biochemical and Haematological Parameters and Ameliorative Effect of Quinoa Seeds in Swiss Albino Mice

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Abstract

Fenvalerate is a synthetic pyrethroid insecticide with moderate mammalian toxicity. It affects the central nervous system and is used for the control of flies and ticks in barns and stables. Exposure of this insecticide to humans occurs in the field. The present study was conducted to observe the toxicity of insecticide on biochemical and blood parameters after treatment with fenvalerate, a synthetic pyrethroid and find out the possible protective role of Quinoa seeds in Swiss albino mice. The mice in this study were divided into 5 groups, and 6 animals were taken into each group. Group A control mice received vehicle distilled water only. Group B and C were administered with fenvalerate at 12.5 and 25 mg/kg body weight respectively for 30 days. Group D was administered 12.5 mg/kg fenvalerate +25 mg/kg Quinoa seeds and Group E received 25 mg/kg fenvalerate +50 mg/kg Quinoa seeds. Fenvalerate treatment resulted in a significant increase in the levels of alkaline phosphatase, acid phosphatase, Serum Glutamate Pyruvate Transaminase (SGPT) and Serum Glutamate Oxaloacetate Transaminase (SGOT). Administration of Quinoa seeds along with fenvalerate resulted in a decrease in these biochemical values. A significant increase in the number of RBC, WBC and haemoglobin was observed in the fenvalerate-treated groups. The result suggests a possible protective role of Quinoa seeds on fenvalerate toxicity.

Keywords: Biochemical, Blood, Fenvalerate, Mice, Quinoa Seeds

1. Introduction

Insecticides are substances used to kill insects or pests on crops. Insecticides belong to four categories: organophosphates, organochlorines, carbamates and synthetic pyrethroids. Synthetic pyrethroids are synthetically derived from naturally occurring pyrethrin, which is taken from pyrethrum, the oleoresin extract of the dried *Chrysanthemum* flowers. The keto alcoholic esters of chrysanthemic and pyrethroid acids provide insecticidal properties to the pyrethrin. The derived acids show lipophilic properties and enter the insect body and paralyse their nervous systems[1]. Synthetic pyrethroids are of two types- Type I and Type II among which Type II shows more potency. Type I are the esters without an alpha-cyano group and include Allethrin, Bifenthrin, Resmethrin, and Permethrin whereas Type II are the esters with an alpha-cyano group and include Cypermethrin, Deltamethrin, Fenvalerate and Cyhalothrin. These pyrethroids cause prolonged opening of the voltage-gated sodium channels by inhibiting deactivation and stabilizing the open state[2] which causes the excitation of axonal membranes for a

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longer period. These derivatives are chemically designed to be highly potent with longer half-life which on accumulation can affect human health. Pyrethroids show toxicity in aquatic organisms including fish which results in damage to gills and behavioural changes with LC_{50} values are less than 1.0 parts per billion which are similar for insects like mosquitoes, and tsetse flies. Non-target aquatic organisms like lobsters, shrimps, nymphs and zooplanktons are also susceptible to these insecticides[3].

Fenvalerate is a synthetic pyrethroid insecticide. Fenvalerate (RS)-alpha-cyano-3-phenoxybenzyl (RS)-2-(4-chlorophenyl)-3-methylbutyrate insecticide is a mixture of four optical isomers that have different insecticidal activities. Esfenvalerate, which has a 2-S alpha (or SS) configuration, is the most insecticidal active isomer and 23% of this isomer is present in fenvalerate. It is commonly used to control insects in food, feed and cotton products and is also helpful for the control of flies and ticks in barns and stables. Pests and insects exposed to fenvalerate have shown rapid paralysing effects. In small animals, salivation, chewing, burrowing, choreoathetosis and seizures are observed[4]. It shows moderate mammalian toxicity and on contact causes irritation of the skin and eyes, harmful if swallowed. Tingling and pruritus with blotchy erythema on an exposed area that is worsened by sweating or touching in case of dermal exposure while lacrimation and transient conjunctivitis in case of ocular exposure. Increased dermal exposure may lead to systemic toxicity, which includes headache, dizziness, anorexia and hypersalivation. Severe poisoning resulting in Systemic toxicity is detected by impaired consciousness, muscle fasciculation, convolutions and rarely occurring non-cardiogenic pulmonary oedema[5]. Acute or short-term exposure to this in laboratory animals causes neurotoxicity in the central and peripheral nervous systems. Diminished action potential amplitude, and membrane depolarization, leading to total neural activity blockage are observed. It is a potent inhibitor of calcineurin[4], a calcium and calmodulin-dependent serine/threonine protein phosphatase which is essential for normal muscle and heart functioning, learning and memory, transcription and apoptosis. The plants are not affected by fenvalerate but it remains active for an extended period of time. In experimental animals, the oral LD_{50} in rats was greater than or equal to 451 mg/kg bw (vehicle dependent), whereas, in mice, it was greater than or equal to 100 mg/kg b.w. (vehicle dependent)[6]. Several effects of fenvalerate (100 mg/kg) were observed on hepatic and cerebral xenobiotic metabolizing enzymes in selenium and/or iodine-deficient rats[7]. Carcinogenicity at high doses was reported. In male mice, a slight increase in the incidence of liver-cell tumours and a low survival rate in females were seen[8]. A low total leukocyte count was reported in haemato biochemical assessment of broiler chicks which were exposed to fenvalerate[9] whereas an increased number of leukocytes in peripheral blood with a decrease in the number of erythrocytes as well as haemoglobin and haematocrit levels were reported as a result of acute poisoning in mice[10].

Quinoa (*Chenopodium quinoa*) is a flowering plant belonging to the Amaranth family. It is a herbaceous annual plant grown as a crop primarily for its edible seeds. Quinoa is a source of food for the Andes region's native population. It is rich in proteins, dietary fibre, vitamin B, and dietary minerals and contains all nine essential amino acids and hence it is often referred to as a "superfood". It is gluten-free and because of its bitter taste (due to the presence of polyphenols and flavonoids), it is commercialized without a seed coat which has been postulated to have antioxidant properties. The hepato-protective role of Quinoa seeds against CCl_4-induced liver damage in Swiss albino male mice has been reported[11].

Several reports about fenvalerate toxicity have been reported earlier, but little study has been performed about the use of Quinoa seeds in reducing the toxicity and its ameliorative role. With the objective to determine the toxicity of fenvalerate on biochemical and blood parameters of Swiss albino mice and to assess the possible protective role of Quinoa seed on fenvalerate toxicity, this study was planned.

2. Materials and Methods

2.1 Chemicals

Market-grade Fenvalerate was procured from a pesticide vendor in the local market, Jaipur and was dissolved in distilled water to prepare different doses i.e. 12.5 mg/kg b.w. and 25 mg/kg b.w.[12]. Market grade Quinoa seeds were brought from a convenience store in Jaipur and were ground to form a fine powder which was dissolved in water to give concentrations of 25 mg/kg b.w. and 50 mg/kg b.w. All other reagents used were of analytical reagent grade.

2.2 Experimental Animals

Swiss albino mice with an average weight of 25-35 g were used in this experiment. The animals were kept in the IIS (Deemed to be University) animal house approved

by CPCSEA (Registration no.: 1689/PO/a/13/ CPCSEA). All animals were housed in plastic cages with wire tops. The cages were maintained in a room with a controlled temperature (25±2 °C), relative humidity (20±10 %) and light/dark cycle (12hr/12hr). Animals were given standard pellets and water *ad libitum*. The approval of the use of Swiss albino mice was taken from IAEC of IIS Deemed to be University (approval no. IISU/IAEC/2021/I/2). The doses were administered orally through an intubation tube.

2.3 Experimental Plan

Animals were randomly divided into 7 groups [Group-1: Control group (animals were given vehicle distilled water only), Group-2: Fenevelate treated (12.5 mg/kg b.w.), Group-3: Fenvalerate treated (25 mg/kg b.w.), Group-4: Fenvalerate (12.5 mg/kg b.w.) + Quinoa seeds (25 mg/kg b.w.), Group-5: Fenvalerate (12.5 mg/kg b.w.) + Quinoa seeds (50 mg/kg b.w.), Group-6: Fenvalerate (25 mg/kg b.w.) + Quinoa seeds (25 mg/kg b.w.), Group-7: Fenvalerate (25 mg/kg b.w.) + Quinoa seeds (50 mg/kg b.w.)] with 6 mice in each group. The treatment was given for 30 days to all the groups and on the 31st day, animals were weighed and sacrificed by cervical dislocation. Blood was collected from the heart for biochemical and blood parameters. For serum biochemical parameters, the blood was allowed to clot and then centrifuged at 3000 rpm and the serum was separated. For cholesterol testing, the liver tissue was collected.

2.4 Biochemical Parameters

The following biochemical parameters were performed (estimation from the serum) Aspartate Transaminase/ Serum Glutamate Oxaloacetate Transaminase (AST/ SGOT), Alanine transaminase/Serum Glutamate Pyruvate Transaminase (ALT/SGPT), Acid Phosphatase (ACP), Alkaline Phosphatase (ALP)[13] - all the four parameters were performed with a kit (Accurex Biomedical Mumbai, India). Cholesterol estimation was done by Zak's method.

2.5 Blood Parameters

The blood parameters included RBC count, WBC count, haemoglobin percentage, haematocrit value, MCH (Mean Corpuscular Haemoglobin), MCHC (Mean Corpuscular Haemoglobin Concentration) and MCV (Mean Corpuscular Volume). All these parameters were performed using standard protocols.

2.6 Statistical Analysis

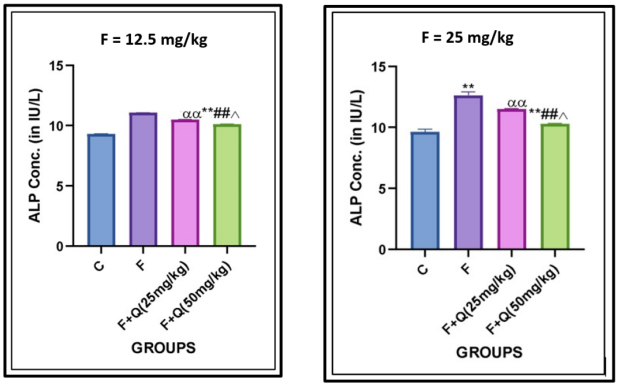
Data were analysed by one-way analysis of variance (ANOVA) using Prism 8 (GraphPad software). To determine whether there are any statistically significant differences ($p<0.05$) between the means of three or more independent (unrelated) groups, a one-way analysis of variance (ANOVA) is used.

3. Observations and Results

3.1 Effect on Biochemical Parameters

There was a highly significant increase in the values of Alkaline Phosphatase (ALP) levels in the fenvalerate 50 mg/kg/body weight treated groups as compared to control levels (Figure 1). The increase was more significant at higher doses of fenvalerate. Groups subjected to a combination of fenvalerate and quinoa showed a significant decrease in ALP levels in both groups. The group subjected to 50 mg/kg of quinoa showed a more diminished level of ALP when compared to the 25 mg/kg group.

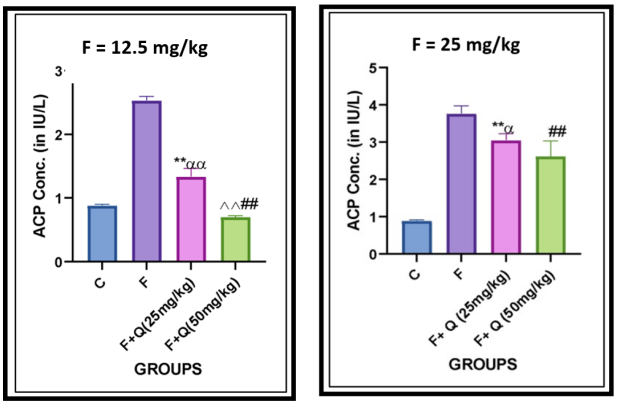
A non-significant increase was seen in the level of ACP Conc. in the fenvalerate-administered group. A significant decrease was observed in both groups of fenvalerate 12.5 mg/kg b.w. and 25 mg/kg b.w. administered with quinoa at 25 mg/kg. Groups administered with quinoa conc. of 50 mg/kg showed significant results when



C - Control, **F** - Fenvalerate, **Q** - Quinoa

Figure 1. ALP level in control and different groups on the administration of Fenvalerate, and combinations Fenvalerate + Quinoa (25 mg/kg), Fenvalerate + Quinoa (50 mg/kg).

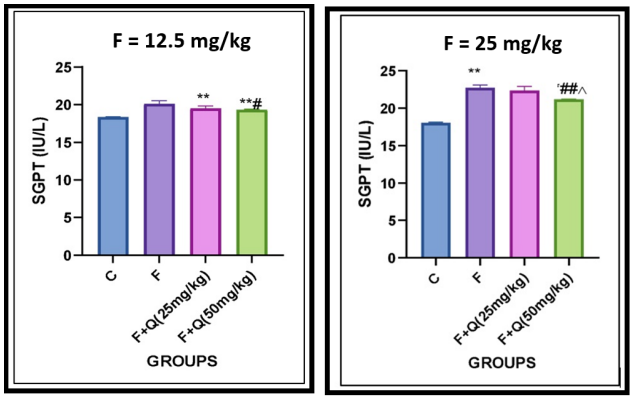
Data: Mean±SEM *P<0.05, **P<0.01 when compared within groups; #P<0.05 ##P<0.01 when compared in between F and F+Q 50 mg/kg, αP<0.05 ααP<0.01 when compared between F and F+Q 25 mg/kg, ^P<0.05 when compared between F+Q 25 mg/kg and F+Q 50 mg/kg (ANOVA)



C - Control, F - Fenvalerate, Q - Quinoa

Figure 2. ACP level in control and different groups on the administration of Fenvalerate, and combinations Fenvalerate + Quinoa (25 mg/kg), Fenvalerate + Quinoa (50 mg/kg).

Data: Mean±SEM *P<0.05, **P<0.01 when compared within groups; #P<0.05 ##P<0.01 when compared in between F and F+Q 50 mg/kg, αP<0.05 ααP<0.01 when compared between F and F+Q 25 mg/kg, ^P<0.05 when compared between F+Q 25 mg/kg and F+Q 50 mg/kg (ANOVA)



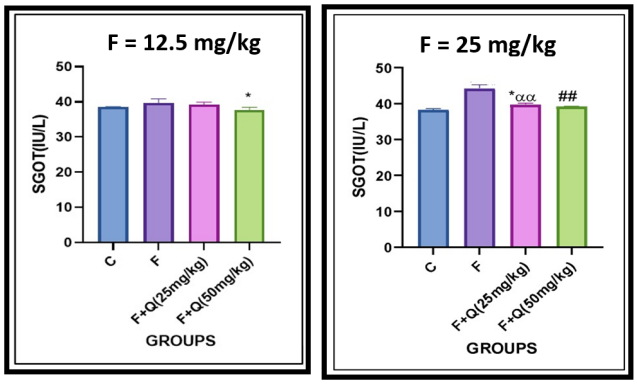
C - Control, **F** - Fenvalerate, **Q** - Quinoa

Figure 3. SGPT level in control and different groups on the administration of Fenvalerate, and combinations Fenvalerate + Quinoa (25 mg/kg), Fenvalerate + Quinoa (50 mg/kg).

Data: Mean±SEM *P<0.05, **P<0.01 when compared within groups; #P<0.05 ##P<0.01 when compared in between F and F+Q 50 mg/kg, αP<0.05 ααP<0.01 when compared between F and F+Q 25 mg/kg, ^P<0.05 when compared between F+Q 25 mg/kg and F+Q 50 mg/kg (ANOVA)

compared to groups administered with only fenvalerate and fenvalerate+quinoa (25 mg/kg b.w.) (Figure 2).

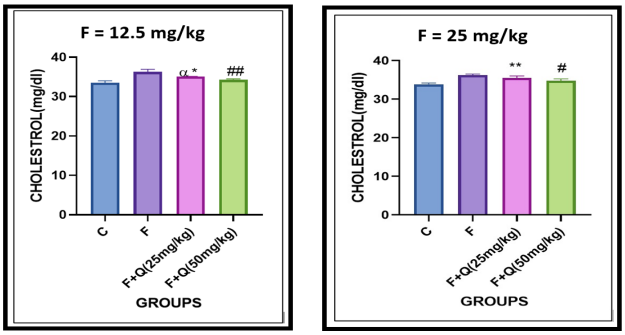
Serum Glutamic Oxaloacetic Transaminase (SGOT) and Serum Glutamic Pyruvic Transaminase (SGPT) values showed highly significant increases in the 25 mg/kg group, but levels were near normal in the 12.5 mg/kg group. Groups with the combination (Fenvalerate and Quinoa seeds) resulted in a slight decrease in levels of SGOT and SGPT (Figures 3 and 4).



C - Control, **F** - Fenvalerate, **Q** - Quinoa

Figure 4. SGOT level in control and different groups on the administration of Fenvalerate, and combinations Fenvalerate + Quinoa (25 mg/kg), Fenvalerate + Quinoa (50 mg/kg).

Data: Mean±SEM *P<0.05, **P<0.01 when compared within groups; #P<0.05 ##P<0.01 when compared in between F and F+Q 50 mg/kg, αP<0.05 ααP<0.01 when compared between F and F+Q 25 mg/kg, ^P<0.05 when compared between F+Q 25 mg/kg and F+Q 50 mg/kg (ANOVA)



C - Control, **F** - Fenvalerate, **Q** - Quinoa

Figure 5. Cholesterol level in control and different groups on the administration of Fenvalerate, and combinations Fenvalerate + Quinoa (25 mg/kg), Fenvalerate + Quinoa (50 mg/kg).

Data: Mean±SEM *P<0.05, **P<0.01 when compared within groups; #P<0.05 ##P<0.01 when compared in between F and F+Q 50 mg/kg, αP<0.05 ααP<0.01 when compared between F and F+Q 25 mg/kg, ^P<0.05 when compared between F+Q 25 mg/kg and F+Q 50 mg/kg (ANOVA)

Total cholesterol levels in the fenvalerate-treated group increased non-significantly. A significant decrease in total cholesterol level was observed in groups administered with F+Q (25 mg/kg) and F+Q (50 mg/kg) (Figure 5).

3.2 Effect on Blood Parameters

RBC Count: In both the 12.5 mg/kg b.w. and the 25 mg/kg b.w. groups, there was an increase in the number of RBCs in the fenvalerate-treated mice, the increase was significant in the 25 mg/kg b.w. group. The addition of quinoa along with fenvalerate brought the number of RBCs back to near normal. But when the quinoa dose was doubled, there was a significant increase in the number of RBCs again (Tables 1 and 3).

WBC Count: At 12.5 mg/kg b.w., there was a significant increase in the number of WBCs in 12.5 mg/kg b.w. fenvalerate-treated mice and a decrease in the number of WBCs at 25 mg/kg b.w. In both groups, in addition to quinoa along with fenvalerate and a double dose of quinoa, there was an insignificant increase in the number of WBCs (Tables 1 and 3).

Haemoglobin level (Hb) and Haematocrit (Hct) value showed a highly significant increase in 12.5 mg/kg b.w. fenvalerate-treated mice, whereas non-significant increase levels were seen in 25 mg/kg b.w. fenvalerate treated group. The addition of quinoa brings Hb and Hct values back to near normal levels (Tables 1 and 3).

MCH level: In both 12.5 mg/kg b.w. and 25 mg/kg b.w., there was a non-significant decrease in MCH level in fenvalerate-treated mice. The addition of quinoa with fenvalerate resulted in a non-significant increase in the level of MCH that reached a significant rate (Tables 2 and 4).

MCHC: In both 12.5 mg/kg b.w. and 25 mg/kg b.w., there was a highly significant decrease in the level of the MCHC value at 25 mg/kg b.w. in the fenvalerate-treated group as compared to the control. Also, there was a decrease in MCHC values when Quinoa was administered at 25 mg/kg b.w. and 50 mg/kg b.w. (Tables 2 and 4).

MCV: At 12.5 mg/kg b.w., there was a minor increase in the MCV value in fenvalerate-treated mice and a decrease at 25 mg/kg b.w. treated. The MCV value increases on treatment with quinoa (Tables 2 and 4).

Table 1. RBC, WBC, Haemoglobin and Haematocrit values in 12.5 mg/kg b.w. dose of fenvalerate on various groups

Groups	Number of RBCs (in million)	Number of WBCs (per cubic mm)	Haemoglobin (g/dl)	Haematocrit Value (%)
Group-1 (Control)	17.51 ± 0.31	3778 ± 375	13.3 ± 0.2	24.5 ± 0.9
Group-2 (Fenvalerate 12.5 mg/kg b.w.)	22.34 ± 1.21	5750 ± 485*	15.6 ± 0.3**	35.5 ± 3.2
Group-4 (Fenvalerate 12.5 mg/kg b.w. + Quinoa 25 mg/kg)	18.67 ± 3.92	5583 ± 187	14.4 ± 0.2	34.4 ± 3.2
Group-5 (Fenvalerate 12.5 mg/kg b.w. + Quinoa 50 mg/kg)	19.54 ± 3.29	4566 ± 552	15.8 ± 0.3**	37.6 ± 2.2*

Data: Mean±SEM *P<0.05, **P<0.01 when compared within groups; #P<0.05 when compared in between groups (ANOVA)

Table 2. MCH, MCHC and MCV values in 12.5 mg/kg b.w. dose of fenvalerate on various groups

Groups	MCH Value (in pg)	MCHC (%)	MCV Value (Fl)
Group-1 (Control)	8.03 ± 0.1	54.6 ± 1.7	14.5 ± 0.7
Group-2 (Fenvalerate 12.5 mg/kg b.w.)	6.9 ± 0.2	45 ± 5.5	15.6 ± 1.3
Group-4 (Fenvalerate 12.5 mg/kg b.w. + Quinoa 25 mg/kg)	8.56 ± 2.0	42.3 ± 3.5	19.7 ± 3.1
Group-5 (Fenvalerate 12.5 mg/kg b.w. + Quinoa 50 mg/kg)	9.14 ± 2.2	41.3 ± 1.4	21.75 ± 6.2

Data: Mean±SEM *P<0.05, **P<0.01 when compared within groups; #P<0.05 when compared in between groups (ANOVA)

Table 3. RBC, WBC, Haemoglobin and Haematocrit values in 25 mg/kg b.w. of fenvalerate dose on various groups

Groups	Number of RBCs (in million)	Number of WBCs (per cubic mm)	Haemoglobin (g/dl)	Haematocrit Value (%)
Group-1 (Control)	17.5 ± 0.3	3778 ± 375	8.3 ± 0.2	24.4 ± 0.9
Group-3 (Fenvalerate 25 mg/kg b.w.)	25.01 ± 0.2**	2833 ± 260	11 ± 0.1	33 ± 0.5**
Group-6 (Fenvalerate 25 mg/kg b.w. + Quinoa 25 mg/kg)	21.39 ± 1.8	3500 ± 251	10.3 ± 0.6	31 ± 2.0*
Group-7 (Fenvalerate 25 mg/kg b.w. + Quinoa 50 mg/kg)	24.51 ± 1.2*	3250 ± 304	9.9 ± 0.4	29.6 ± 1.4

Data: Mean±SEM *P<0.05, **P<0.01 when compared within groups; #P<0.05 when compared in between groups (ANOVA)

Table 4. MCH, MCHC and MCV values in 25 mg/kg b.w. dose of fenvalerate on various groups

Groups	MCH Value (in pg)	MCHC (%)	MCV Value (fL)
Group-1 (Control)	8.03 ± 0.1	54.6 ± 1.7	14.5 ± 0.7
Group-3 (Fenvalerate 25 mg/kg b.w.)	7.26 ± 0.2	32.3 ± 0.6**	13.1 ± 0.1
Group-6 (Fenvalerate 25 mg/kg b.w. + Quinoa 25 mg/kg)	8.83 ± 0.8	32.3 ± 0.6**	14.5 ± 0.3
Group-7 (Fenvalerate 25 mg/kg b.w. + Quinoa 50 mg/kg)	7.66 ± 0.3	32.3 ± 0.6**	12.1 ± 0.9

Data: Mean±SEM *P<0.05, **P<0.01 when compared within groups; #P<0.05 when compared in between groups (ANOVA)

4. Discussion

The study shows the intensity of effects due to exposure to fenvalerate, a synthetic pyrethroid on biochemical and blood parameters of Swiss albino mice.

Fenvalerate, an insecticide which is widely used in agricultural practices almost everywhere in the world and is effective against a wide variety of pests which are resistant to organochlorine, organophosphorus and carbamate has some oxidative and degenerative effects on biochemical parameters after prolonged exposure.

This study was chosen to contribute to the limited number of published studies on this synthetic pyrethroid, fenvalerate and its possible amelioration effects by Quinoa seeds.

Alkaline phosphatase (ALP) levels in serum are indicative of liver health, this enzyme is responsible for the hydrolysis of phosphate monoesters in the body and is chiefly secreted by the liver. Higher levels of ALP are correlated with damage to liver cells[14]. The levels of ALP in our study were significantly elevated in both lower (12.5 mg/kg b.w.) and higher (25 mg/kg b.w.) concentrations when compared to control groups. Acid phosphatase is an enzyme which acts to liberate phosphate under acidic conditions in the liver, bones, prostate and spleen. Increased levels of ACP indicate possible osteosclerosis, liver injury, and prostate cancer. The levels of ACP were significantly increased in our study in both the fenvalerate-treated groups. SGPT amounts are lower in kidney and skeletal muscles; hence, a rise in the levels of alanine transferase is specific to liver damage[15,16]. AST or SGOT is present in cytosol as well as in mitochondria. Hepatic acinus has a high concentration of AST, and injury in this zone is responsible for alterations in serum AST levels[16,17]. Levels of cholesterol are indicative of liver health, and increased levels are seen during liver sepsis or injury.

Post-exposure analysis of the serum from both fenvalerate-treated groups i.e. 12.5 mg/kg b.w. and 25 mg/kg b.w. reveals that there was a significant increase in the levels of Alkaline Phosphatase (ALP), Acid Phosphatase (ACP), Serum Glutamate Pyruvate Transaminase (SGPT), Serum Glutamate Oxaloacetate Transaminase (SGOT) and cholesterol when compared to control levels. The study correlates with previous studies with fenvalerate which reported the toxicology and carcinology in B6C3F1 mice, showing increased glutamate oxaloacetate transaminase levels[18]. It is also responsible for oxidative stress and increased levels of ALT and AST[19,20]. A non-significant increase was observed in total cholesterol in the group treated with fenvalerate whereas, in an earlier

study, a significant increase in the total cholesterol levels was observed[21].

The study reported a slight but significant decrease in the elevated levels of the above-mentioned parameters by co-administration of Quinoa seeds. The group which was administered the double dose of quinoa shows lower levels than the group with a single dose of quinoa. This correlates with the study done earlier revealing that vitamin C administration to fenvalerate treated mice reduced the oxidative stress caused by this synthetic pyrethroid[19].

A vital role in assessing the toxicity of any exogenous compound in the system is the evaluation of haematological indices. The blood constitutes the greatest percentage of the total body fluid and it is the commonest route of transport of food, drug and any foreign substance. A clinicopathological condition arises when a compound in excess or lower than the normal level of a compound is accumulated in the blood. The toxic effect of a compound is seen as an excess or deficit of one or more serum markers; an enzyme, a hormone or any endogenous compound. In assessing hepatotoxicity, an increase or decrease in blood variables indicates excess or suppression of blood cell production respectively or an imbalance between blood cell production and destruction[22].

Our study shows that after exposure to fenvalerate for one month, a significant increase in the number of erythrocytes and treatment with quinoa doses reverses the effects of erythropoiesis in male Swiss Albino Mice which correlate with the previous studies showing stimulation of erythropoiesis and synthesis of haemoglobin in male mice and in both males and females, a general increase in the number of leukocytes in fenvalerate treated mice[23].

There was a remarkable increase in the number of leukocytes in all fenvalerate-treated mice showing relevance to the studies in which the pyrethroid exposure caused an increase in the number of leukocytes in peripheral blood and males, mobilization of haematopoietic system[10].

A study on fish suggested the changes in the blood indices were due to a defence reaction against toxicity of cypermethrin through the stimulation of erythropoiesis or may be due to the disturbances that occurred in both metabolic and hematopoietic activities of fish exposed to below-safe concentrations of cypermethrin[24].

The alteration observed may be due to physiological dysfunctioning of the haematopoietic tissue or may be due to the effects on bone marrow. The effect of fenvalerate is more pronounced than deltamethrin and cypermethrin in prolonged exposure[23].

Conclusively, the results of this research suggest that the current unrestricted use of synthetic pyrethroids is not appropriate for the sake of nature and its inhabitants and may lead to potential health hazards with prolonged use. The ameliorative effect of Quinoa seeds on fenvalerate toxicity is also evident from the study.

5. Conclusion

Fenvalerate treatment in both groups was 12.5 mg/kg b.w. and 25 mg/kg b.w. led to an increase in the values of ALP, ACP, SGOT and SGPT. The values decreased when quinoa seeds were given, showing ameliorative features due to the presence of certain compounds such as quercetin in quinoa which have antioxidant properties. The variations observed in haematological parameters in treated groups and values coming back to normal range after quinoa seed administration suggest the protective role of the Quinoa seeds on fenvalerate toxicity.

6. Acknowledgement

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7. References

1. Reigert JR, Roberts JR. Recognition and management of pesticide poisonings. 6th ed. Washington, DC: Office of Pesticide Programs United States Environmental Protection Agency. 2013; p. 38.
2. Silver KS, Du Y, Nomura Y, Oliveira EE, Salgado VL, Zhorov BS, Dong K. Voltage-Gated Sodium Channels as Insecticide Targets. Adv In Insect Phys. 2014; 46:389-433. https://doi.org/10.1016/B978-0-12-417010-0.00005-7 PMID: 29928068 PMCID: PMC6005695
3. Sayeed I, Parvez S, Pandey S, Bin-Hafeez B, Haque R, Raisuddin S. Oxidative stress biomarkers of exposure to deltamethrin in freshwater fish, *Channa punctatus* Bloch. Ecotoxicology and Environmental Safety. 2003; 56(2):295-301. https://doi.org/10.1016/S0147-6513(03)00009-5
4. Locey BJ, Reeves J. Fenvalerate. Encyclopedia of Toxicology 2nd ed. U.S.A. Academic Press. 2005; p. 328-330. https://doi.org/10.1016/B0-12-369400-0/00412-9
5. Cage SA, Bradberry SM, Meacham S, Vale JA. Fenvalerate. Inchem Report. National Poisons Information Service. Birmingham,United Kingdoms. 1998.

6. Anonymous. Fenvalerate. JMPR Report. Rome, Italy: World Health Organisation. 2012; p. 153.
7. Caglayan A, Kocer-Gumusel B, Erkekoglu P, Hincal F. The effects of fenvalerate on hepatic and cerebral xenobiotic metabolizing enzymes in selenium and/or iodine deficient rats. Iran J Basic Med Sci. 2016; 19(10):1040-48. PMID: 27872699 PMCID: PMC5110651
8. Cabral JBR, Galendo D. Carcinogenicity study of the pesticide fenvalerate in mice. Cancer Letters. 1990; 49(1):13-8. https://doi.org/10.1016/0304-3835(90)90133-I
9. Garg U, Pal AK, Jha GJ, Jadhao SB. Haemato-biochemical and immuno-pathophysiological effects of chronic toxicity with synthetic pyrethroid, organophosphate and chlorinated pesticides in broiler chicks. International Immunopharmacology. 2004; 4(13):1709-22. https://doi.org/10.1016/j.intimp.2004.08.002
10. Haratyam- Maj A. Hematological alterations after pyrethroids poisoning in mice. Annals of agricultural and environmental medicine. 2002; 9(2):199-206. Available from: http://www.aaem.pl/Hematological-alternations-after-pyrethroids-poisoning-in-mice-,72793,0,2.html
11. Saxena S, Shahani L, Bhatnagar P. Hepatoprotective effect of *Chenopodium quinoa* seed against CCL_4-induced liver toxicity in *Swiss albino* male mice. Asian J Pharm Clin Res. 2017; 10(11):273-6. https://doi.org/10.22159/ajpcr.2017.v10i11.20918
12. Luty S, Latuszyn`ska J, Obuchowska-Przebirowska D, Tokarska M, Haratyam- Maj A. Subacute toxicity of orally applied alpha-cypermethrin in Swiss mice. Ann Agric Environ Med. 2000; 7:33-41. https://doi.org/10.1111/an.2000.41.7.33
13. Karmen A, Wroblewski F, Ladue J.S. Transaminase activity in human blood. The Journal of Clinical Investigation. 1955; 34(1):126–31. https://dm5migu4zj3pb.cloudfront.net/manuscripts/103000/103055/JCI55103055.pdf https://doi.org/10.1172/JCI103055
14. Sharma U, Pal D, Prasad R. Alkaline Phosphatase: An Overview. Ind J Clin Biochem. 2014; 29(3):269-78. https://doi.org/10.1007/s12291-013-0408-y
15. Wróblewski F. The clinical significance of alterations in transaminase activities of serum and other body fluids. Advances in clinical chemistry, Elsevier. 1958; 1:313-351. https://doi.org/10.1016/S0065-2423(08)60362-5 PMID: 13571034
16. Giannini EG, Testa R, Savarino V. Liver enzyme alteration: A guide for clinicians. Cmaj. 2005; 172(3):367-79. https://doi.org/10.1503/cmaj.1040752
17. Rej R. Aminotransferases in disease. Clin Lab Med. 1989; 9(4):667-87. https://doi.org/10.1016/S0272-2712(18)30598-5 PMID: 2686908
18. Parker CM, McCullough CB, Gellatly JB, Johnston CD. Toxicologic and carcinogenic evaluation of fenvalerate in the B6C3F1 mouse. Fundamental and Applied Toxicology. 1983; 3(2):114-20. https://doi.org/10.1016/S0272-0590(83)80066-9
19. Hussein HK, Elnaggar MH, Al-Dailamy JM. Protective role of Vitamin C against hepatorenal toxicity of fenvalerate in male rats. Glo Adv Res J Environ Sci Toxicol. 2012; 1(4):060-5. https://www.researchgate.net/profile/Moustafa-Elnaggar/publication/303683848_Hussein_HK_ELNAGGAR_MHR_and_Al-Dailamy_JM_2012_Protective_role_of_Vitamin_C_against_hepatorenal_toxicity_of_fenvalerate_in_male_rats_Glob_Adv_Res_J_Environ_Sci_Toxicol_14_60-65/links/5cc6e698a6fdcc1d49b7c417/Hussein-HK-ELNAGGAR-MHR-and-Al-Dailamy-JM-2012-Protective-role-of-Vitamin-C-against-hepatorenal-toxicity-of-fenvalerate-in-male-rats-Glob-Adv-Res-J-Environ-Sci-Toxicol-14-60-65.pdf
20. Dar SA, Kaur R. Hematobiochemical evaluation of dermal subacute cypermethrin toxicity in buffalo calves. Toxicology International. 2014; 21(3):283-7. https://doi.org/10.4103/0971-6580.155364 PMID: 25948967 PMCID: PMC4413411
21. Ravikumar Y, Madhuri D, Lakshman M, Reddy AG, Kalakumar B. Study of cadmium and chlorpyrifos induced changes in protein and lipid profile in rats. Toxicology International. 2020; 27(1&2):54-7. https://doi.org/10.18311/ti/2020/v27i1&2/25279
22. Olson H, Betton G, Robinson D, Thomas K, Monro A, Kolaja G, Lilly P, Sanders J, Sipes G, Bracken W, Dorato M. B., and Heller, A. Concordance of the toxicity of pharmaceuticals in humans and in animals. Regul Toxicol Pharmacol. 2000; 32(1):56-67. https://doi.org/10.1006/rtph.2000.1399 PMID: 11029269
23. Tos-Luty S, Haratym-Maj A, Latuszynska J, Obuchowska-Przebirowska D, Tokarska-Rodak M. Oral toxicity of deltamethrin and fenvalerate in Swiss mice. Ann Agric Environ Med. 2001; 8(2):245-54 https://agro.icm.edu.pl/agro/element/bwmeta1.element.agro-article-56243671-30dd-4299-add2-edabf8f214e2
24. Masud S, Singh IJ. Effect of Cypermethrin on some hematological parameters and prediction of their recovery in a freshwater Teleost, *Cyprinus carpio.* Afr J Environ Sci Technol 2013; 7(9):852-6. https://doi.org/10.5897/AJEST11.376
25. Sharma P, Singh R, Jan M. Dose-dependent effect of deltamethrin in testis, liver, and kidney of Wistar rats. Toxicology International. 2014; 21(2):131-9. https://doi.org/10.4103/0971-6580.139789 PMID: 25253921. PMCID: PMC4170553