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TOXICOLOGICAL ANALYSIS OF CYPERMETHRIN: INDUCED AND FEEDING EFFECTS ON ALAT AND ASAT ENZYMATIC ACTIVITY IN GALLUS DOMESTICUS

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Abstract

This research examines the toxic effects of cypermethrin on the activity of enzymes Alanine Aminotransferase (ALAT) and Aspartate Aminotransferase (ASAT) in Gallus domesticus (chicks). Two methods of exposure were used: induced and feeding. Fifteen-day-old chicks treated with cypermethrin through intramuscular injection at doses of 20 µl/body weight (b.wt) and 40 µl/b.wt, as well as through feed at concentrations of 1 µl/20 mg and 1 µl/40 mg, administered weekly for a duration of up to 20 weeks. A control group was kept untreated. Enzyme activity was evaluated at intervals of 5 weeks. Marked changes in ALAT levels were recorded in the induced group. After 5 weeks, ALAT activity in chicks given 20 µl/b.wt cypermethrin (34.24 ± 0.845) showed a significant difference (P<0.05) compared to both the control group (219.99 ± 13.14) and the higher dose group (297.69 ± 30.55). By 20 weeks, ALAT activity in the 20 µl/b.wt group increased (254.62 ± 199.75) but differed significantly from the controls (324.56 ± 4.54). Likewise, exposure through feeding at 1 µl/20 mg resulted in significant ALAT changes at 5, 10, and 15 weeks when compared to the control group. ASAT levels exhibited a similar pattern. Induced exposure of both doses (20 µl/b.wt and 40 µl/b.wt) led to significant increases at all assessment points (5, 10, 15, and 20 weeks), suggested ongoing hepatic stress. Groups treated via feed (1 µl/20 mg and 1 µl/40 mg) also displayed significant ASAT changes throughout the experimental duration (P<0.05). These results imply that cypermethrin, regardless of the method of administration, has hepatotoxic effects and disrupts physiological development in chicks. The changes observed in ALAT and ASAT activities place these enzymes as reliable indicators of pesticide-induced liver dysfunction in poultry. This study emphasizes the potential dangers of cypermethrin exposure in poultry and reinforces the necessity for caution in its agricultural application.

INTRODUCTION

Pesticides, chemical fertilizers, and antibiotics are frequently and unnecessarily employed to enhance agricultural productivity to support population growth [1]. Pesticides are present in various environments (water, soil, and air); they have the potential to harm natural plants and animals, as well as non-target organisms such as aquatic life, livestock, and wildlife, especially birds, leading to a significant

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public health concern [2]. Unfortunately, many pesticides are ineffective as they may harm non-target organisms that are either neutral or beneficial to the ecosystem. Overall, only about 0.1 percent of pesticides actually reach their intended targets, while the majority cause damage to the environment [3]. Pesticides have also entered the food web, accumulating over time at higher trophic levels. Recent studies have linked pesticide exposure to various acute and chronic health issues in humans [4]. Pyrethroid toxins can be detected in contaminated fruits, vegetables, and other food items, as well as in residues from earlier crops [5], and are also components in a range of commercially available insecticides [6]. High levels of pyrethroid components are still present in the yields of several farming communities, even though crop processing reduces pyrethroid contamination through methods like evaporation, washing, and exposure to sunlight. Nonetheless, instances of pyrethroid pesticide concentrations exceeding the recommended safe limits are still detected in the environment, particularly in developing countries in Asia and Africa where both natural and more commonly synthetic pyrethroids have been extensively applied for agricultural protection for many years [7]. Contemporary conservation practices lead to increased pyrethroid exposure in the soil, environment, vulnerable individuals, and the food chain and storage facilities [8]. Persistent exposure to toxic substances can lead to damage to essential organs, which may cause organ dysfunction and increased toxicity in the affected organism over time, depending on the duration and concentration of the toxic agents [9]. Cypermethrin, a type of pyrethroid insecticide, is commonly utilized for pest control [10-12]. This synthetic pesticide is widely applied to manage various pests in residential, industrial, and agricultural settings [13]. Due to its rapid insecticidal action and relatively low toxicity in mammals and other land-based vertebrates, cypermethrin is extensively used in both agricultural and domestic environments in countries like India and Pakistan [14]. Mechanistically, Cypermethrin may lead to damage in voltage-sensitive sodium channels, allowing these channels to stay open longer than they should, and it can also inhibit ATPase enzymes that are critical for ion transport against concentration gradients. These molecular changes can contribute to the unique toxicity of cypermethrin at the larger scale within an organism [15]. Research has shown that cypermethrin induces oxidative stress in rodents by impacting both enzymatic and non-enzymatic antioxidant systems, as evidenced by elevated levels of malondialdehyde in tissues and decreased activity of superoxide dismutase, catalase, and glutathione peroxidase [16]. Owing to its lipophilic characteristics, it tends to accumulate in the nervous system and fatty tissues [17]. Due to the presence of fur, cypermethrin is not easily absorbed through the skin when applied to animals. While it does have a moderate absorption rate into the bloodstream, it is quickly metabolized in the liver into non-toxic byproducts that are excreted in urine, a process that is accelerated by the enzyme glucuronidase [18-20]. Birds can die from pesticide exposure through several means, including direct ingestion of pesticide granules and contaminated seeds or crops, direct contact with sprays, exposure to polluted water, or consuming infected prey and baits [21,22]. Chicken serves as a significant global food source and is also a critical animal model in toxicology and biomedical studies [23]. The presence of cypermethrin has been analyzed across various mammalian tissues and species, including the liver, kidneys, central and peripheral nervous tissues, and lipids [24,25]. This study aims to investigate the impact of cypermethrin on chicks of *Gallus domesticus*, as they are a vital dietary resource for humans and rank high in the pecking order, making them potential bio indicators of pesticide pollution.

The absorption of pesticides by fish leads to their movement up the food chain, resulting in detrimental health effects for humans when consumed. The drift of pesticides into aquatic environments results in chemical pollution, which has adverse (long-term) impacts. There is a connection between exposure to pesticides and synthetic chemicals in humans and various health issues, including cancer, obesity, endocrine disruption, and other ailments [26]. According to the projected outcomes, it can be inferred that if the test organisms have been influenced by cypermethrin and humans are ingesting them, then it could be having considerable effects on humans too.

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MATERIAL AND METHODS

1.1. **Experimental Organism:** The chicks of *Gallus domesticus*, which are part of the order *Galliformes* and the family *Phasianidae*, were utilized for experimental purposes. A total of 50 chicks were involved throughout the experimental period, lasting up to 20 weeks, all experimental subjects were consistently fed an 80-gram portion of Master Poultry Feed.

- Control group (untreated group).
- Treated group receiving 20 µl/b.wt. of cypermethrin administered in the thigh region.
- Treated group receiving 40 µl/b.wt. of cypermethrin administered in the thigh region.
- Treated group having 1 µl/20 mg of cypermethrin mixed into their feed.
- Treated group having 1 µl/40 mg of cypermethrin mixed into their feed.

1.2. **Preparation of Chemical:** 10 EC Cypermethrin was used, 2 ml of cypermethrin was added in 18 ml of distilled water to prepare 1% stock solution. To achieve the desired 0.5% concentration, additional 20 ml of distilled water was added.

1.3. **Method of Treatment:** The treatment involved both induced and feeding methods. For the induced method, doses of 20µl/b.wt and 40µl/bwt of cypermethrin were administered via syringe injections into the thigh area of the chicks. In the feeding method, cypermethrin was delivered in concentrations of 1µl/20mg and 1µl/40mg mixed into the feed at intervals of one week. The control group remained untreated for the entire duration of the 20-week experiment. After 5, 10, 15, and 20 weeks, the experimental subjects were dissected, and the internal organs (liver, kidney, and muscles) were extracted.

1.4. **Analysis of Enzymatic Activities:** The activity of Alanine aminotransferase (ALAT) and Aspartate aminotransferase (ASAT) in each organ (liver, kidney, and muscles) was determined using the Innoline Kit, available under Catalog No. 5.17531.0001 for ALAT and 5.17521.0001 for ASAT.

All samples underwent testing for both ALAT and ASAT levels.

1.5. **Procedure:**

- **Weighing**: Internal organs were removed, and a digital balance was used to carefully weigh 0.5 grams from each organ.

- **Crushing:** One milliliter of distilled water was added to 0.5 grams of tissue in a clean mortar and pestle. In Vilest, a uniform mixture was prepared and gathered.

- **Centrifuge:** A Labofuge 200 Heraeus centrifuge was used to centrifuge this combination for 20 minutes at 3500 rpm.

- **Supernatant:** After removing the centrifuge vilest, the supernatant was separated and the debris disposed of. All subsequent tests were conducted using this supernatant.

Statistical Analysis: The data were statistically evaluated with SPSS, and the significance threshold was set at $P<0.05$.

RESULTS AND DISCUSSION

The domestic chicken is an omnivorous animal known for its meat and eggs, which are both excellent sources of protein. As their organs are also consumed, the contamination of poultry can easily transfer to humans through contaminated food. To meet the needs of our growing population, crop production must be enhanced. Therefore, controlling various pests, especially insects that lead to losses in crop yields, is critically important. Prolonged exposure to Cypermethrin, even at levels significantly lower than the residual amounts present in food, can severely disrupt reproductive functions and general physiological processes [27]. The exposure to environmentally relevant levels of cypermethrin in grass carp resulted in kidney impairment, oxidative stress, endoplasmic reticulum stress, and cell death, suggesting a disturbance in the antioxidant defense mechanisms [28]. The intake of chicken is rising swiftly, which is also true for the application of various pesticides that can accumulate in humans through affected poultry consumption. The enzymes ALAT (alanine aminotransferase) and ASAT (aspartate aminotransferase) are generated in the cytoplasm of

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muscles and hepatocytes, respectively. They are used in clinical research to assess liver injury [29].

Table (1) illustrates the mean activity level of Alanine Aminotransferase (ALAT) in control chicks (*Gallus domesticus*) was noted as 289.88 ± 16.12 in the liver, 264.34 ± 25.09 in the kidney, and 292.78 ± 30.93 in the muscle tissue. Conversely, chicks subjected to cypermethrin displayed a dose-dependent reduction in ALAT activity in all tissues. Chicks treated with 20 µl/b.wt of cypermethrin showed a significant decrease in enzyme activity: 172.42 ± 96.66 in the liver, 116.32 ± 66.40 in the kidney, and 132.55 ± 65.06 in the muscle. A higher dosage of 40 µl/b.wt led to even greater suppression, with ALAT levels at 145.93 ± 99.61 in the liver, 155.46 ± 90.41 in the kidney, and 85.11 ± 42.54 in the muscles. When represented as percentages relative to the control group (which is considered 100%), ALAT activity in the 20 µl/b.wt group fell to 59.45% in the liver, 44.01% in the kidney, and 45.29% in the muscles, whereas in the 40 µl/b.wt group, it declined to 50.32%, 58.84%, and 29.07% in the liver, kidney, and muscles, respectively. Likewise, when cypermethrin was given through feed at concentrations of 1 µl/20 mg and 1 µl/40 mg, a significant inhibition of ALAT activity was observed once more. The 1 µl/20 mg group reported mean ALAT levels of 172.42 ± 96.66 in the liver, 116.32 ± 66.40 in the kidney, and 132.55 ± 65.06 in the muscle. The 1 µl/40 mg group displayed even lower values: 145.93 ± 99.61 in the liver, 155.46 ± 90.41 in the kidney, and 85.11 ± 42.54 in the muscles. As percentages of the control, these figures corresponded to 40.55%, 55.99%, and 54.71% for the 1 µl/20 mg group, and 49.68%, 41.16%, and 70.93% for the 1 µl/40 mg group in liver, kidney, and muscle tissues, respectively.

Table 1: Activity of ALAT in the Organs (Liver, Kidney and Muscles) of *Gallus domesticus* (Chicks) Exposed to Cypermethrin.

Experimental Groups	Organs	Mean	Std. Error	Minimum	Maximum	% Inhibition /Activity
Control	Liver	289.88	16.12	245	315.7	100
	Kidney	264.34	25.09	214	327.1	100
	Muscles	292.78	30.93	200.5	330.9	100
Induced 20µl/b.wt	Liver	172.42	96.66	25.0	441.5	86.34
	Kidney	116.32*	66.40	28.8	312	29.62
	Muscles	132.55	65.06	15.4	279.4	78.03
Induced 40µl/b.wt	Liver	145.93*	99.61	8.7	440	19.62
	Kidney	155.46	90.41	24.7	422.2	102.95
	Muscles	85.11*	42.54	29.1	208.9	32.27
Treated with 1µl/20mg	Liver	172.42*	96.66	25.0	441.5	40.55
	Kidney	116.32*	66.40	28.8	312	55.99
	Muscles	132.55*	65.06	15.4	279.4	54.71
Treated with 1µl/40mg	Liver	145.93*	99.61	8.7	440	49.68
	Kidney	155.46*	90.41	24.7	422.2	41.16
	Muscles	85.11	42.54	29.1	208.9	70.93

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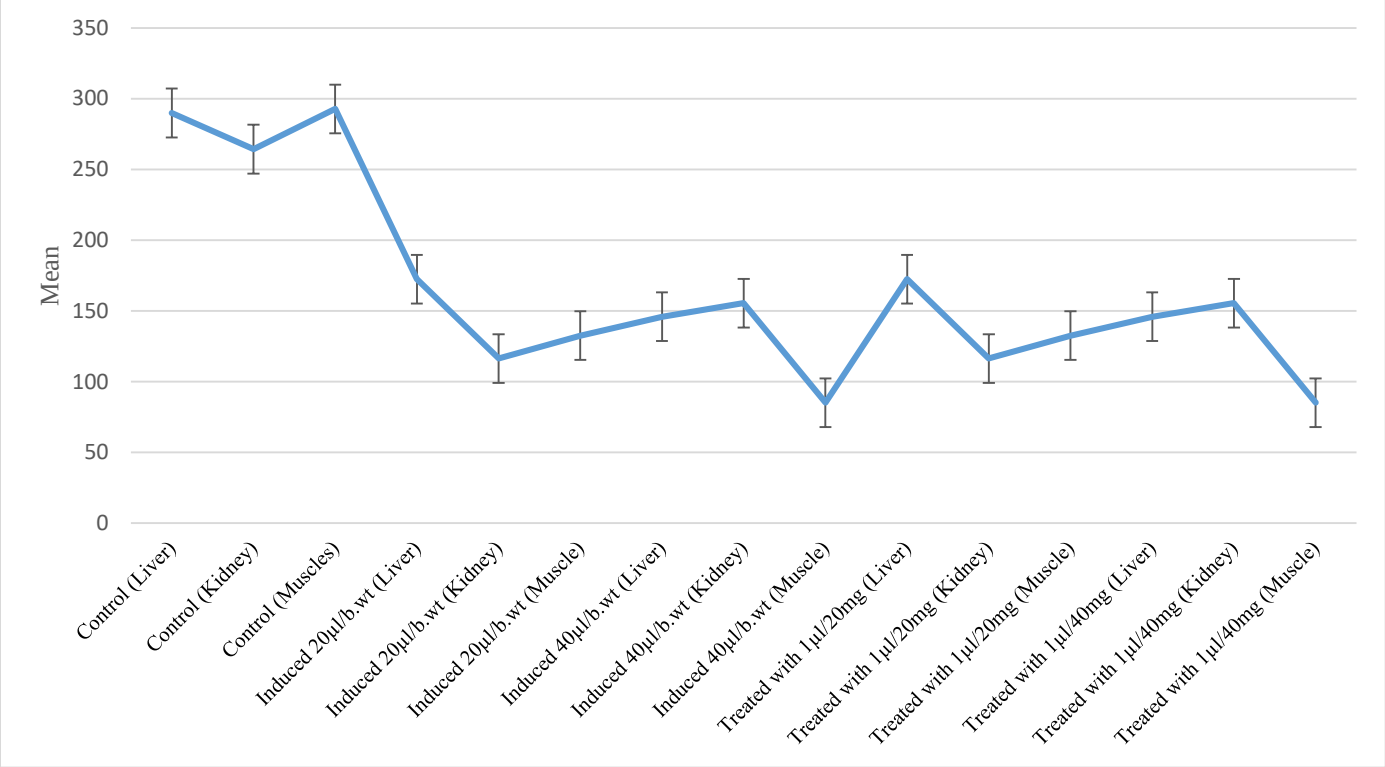

Figure 1: Activity of ALAT in the Organs (Liver, Kidney and Muscles) of *Gallus domesticus* (Chicks)

Exposed to Cypermethrin.

Figure 1 indicates a consistent, dose-dependent decline in ALAT activity, especially in the liver and muscles, highlighting tissue-specific sensitivity to cypermethrin. The varying enzyme responses across organs, particularly the unexpected fluctuations in kidney activity might imply different toxicokinetics, possible organ stress, or compensatory responses. These findings highlight ALAT as a dependable biochemical marker for evaluating cypermethrin-induced hepatotoxicity, nephrotoxicity, and myotoxicity in poultry.

Table (2) reports the statistical analysis of Alanine Aminotransferase (ALAT) levels in *Gallus domesticus* demonstrated notable variations over time and with different doses between the control group and those treated with cypermethrin. In the control group, ALAT activity showed a consistent increase throughout the 20-week study, with levels rising from 219.99 ± 13.14 at 5 weeks to 324.56 ± 4.54 by the end of the 20 weeks. Conversely, chicks that received 20 µl/b.wt of cypermethrin initially displayed significantly reduced ALAT activity in the first weeks recording 34.24 ± 0.845 at 5 weeks and 30.46 ± 10.91 at 10 weeks followed by a slight increase at 15 weeks 67.32 ± 14.16 and a notable increase at 20 weeks 254.62 ± 199.75. The group administered 40 µl/b.wt cypermethrin experienced varying ALAT levels over the study period, with higher readings at 5 weeks 297.69 ± 30.55, a drastic drop at 10 weeks 118.03 ± 44.47, a rebound at 15 weeks 204.18 ± 86.80, and levels nearly equivalent to the control by week 20 to 318.35 ± 149.84. Statistically, at 5 weeks, the group receiving 20 µl/b.wt cypermethrin showed significant differences from both the control and the 40 µl/b.wt group ($P<0.05$), while the control and the 40 µl/b.wt groups did not present significant differences. Significant distinctions were noted between the two cypermethrin-treated groups at 10 and 15 weeks ($P<0.05$), although neither group significantly differed from the control. By week 20, all three groups exhibited significant variability among each other ($P<0.05$), highlighting the delayed yet distinct impacts of cypermethrin over time. In a related feeding study, chicks receiving 1 µl/20 mg of cypermethrin in their feed showed ALAT levels of 28.23 ± 1.704 at 5 weeks, a surge to 158.41 ± 36.59 at

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10 weeks, a reduction to 30.84 ± 7.712 at 15 weeks, and a significant increase to 344.25 ± 49.50 by the 20th week. Likewise, those treated with 1 µl/40 mg of cypermethrin displayed an initial ALAT level of 79.44 ± 7.750 at 5 weeks, which dropped to 20.95 ± 6.25 at 10 weeks, showed a small recovery to 57.91 ± 23.49 at 15 weeks, and then surged to 357.05 ± 74.23 by 20 weeks. Both feed-treated groups differed significantly from the control and each other at 5, 10, and 15 weeks ($P<0.05$). However, by week 20, there were no significant differences observed among any groups, indicating a possible compensatory or adaptive physiological reaction to extended exposure to cypermethrin. Cypermethrin administration resulted in the inhibition of Na, K, and Mg dependent ATPase activity in the livers of albino rats [30]. In contrast, succinate dehydrogenase and ATPase activities were found to be inhibited in the brain, kidney, and liver of *Labeo rohita*, also known as the Indian large carp [31]. Likewise, in the present study, the average ALAT activity in the livers of treated chicks showed a greater decline compared to the control chicks.

Table 2: Level (Mean ± S.E) of ALAT in the *Gallus domesticus* (Chicks) Exposed to Cypermethrin after Different Time Intervals.

Experimental Groups	After 5 Weeks	After 10 Weeks	After 15 Weeks	After 20 Weeks
Control	219.99 ± 13.14	279.33 ± 23.49	305.45 ± 12.96	324.56 ± 4.54
Induced 20µl/b.wt	34.24 ± 0.845	30.46 ± 10.91	67.32 ± 14.16	254.62 ± 199.75
Induced 40µl/b.wt	297.69 ± 30.55	118.03 ± 44.47	204.18 ± 86.80	318.35 ± 149.84
Treated with 1µl/20mg	28.23 ± 1.704	158.41 ± 36.59	30.84 ± 7.712	344.25 ± 49.50
Treated with 1µl/40mg	79.44 ± 7.750	20.95 ± 6.25	57.91 ± 23.49	357.05 ± 74.23

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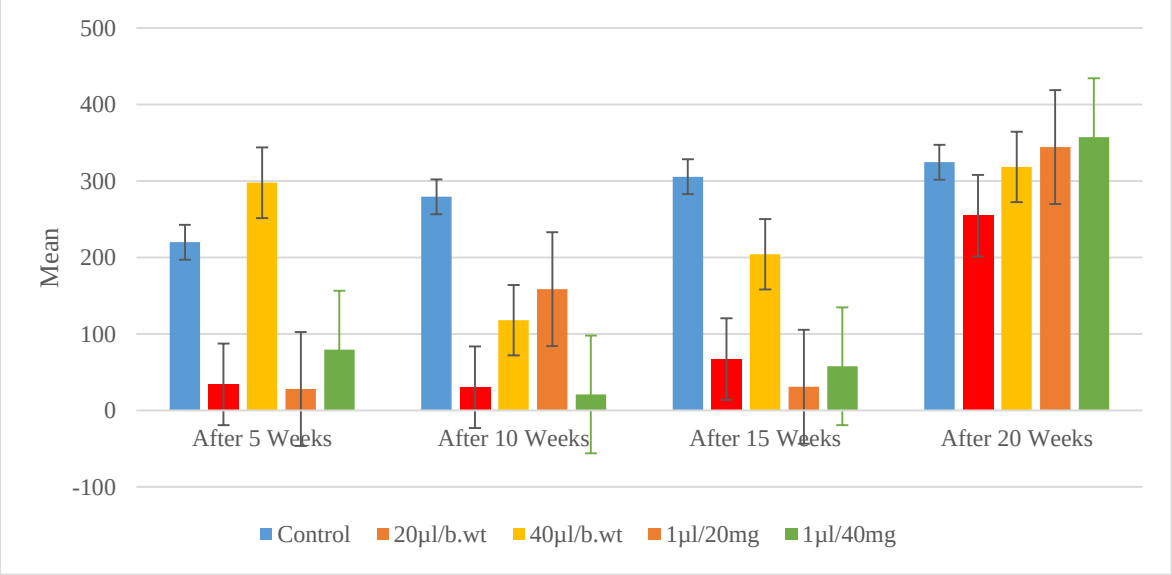

Figure 2: Level (Mean ± S.E) of ALAT in the *Gallus domesticus* (Chicks) Exposed to Cypermethrin after Different Time Intervals.

Figure 2 suggests that ALAT activity in *Gallus domesticus* is substantially influenced by cypermethrin in a complex manner affected by both dosage and duration of exposure. The ALAT is present in the liver, its diagnostic significance in poultry is minimal. Occasionally, animals with significant liver damage might show normal ALAT activity, suggesting low enzyme activity in liver cells. Furthermore, in most avian species, the typical activity of ALAT is below the detection limits of many commercial analyzers [32]. Similar findings were noted in the present study.

Table (3) illustrates the mean activity of aspartate aminotransferase (ASAT) in the liver, kidney, and muscle tissues of *Gallus domesticus* was measured to investigate the biochemical impact of cypermethrin exposure. In the control group, ASAT activity remained consistently low and stable across all tissues, with average values of 67.00 ± 43.02 for the liver, 69.22 ± 44.11 for the kidney, and 66.85 ± 42.92 for the muscles. Conversely, chicks exposed to cypermethrin showed significant increases in ASAT activity, indicating considerable tissue stress. In the group treated with 20 µl/b.wt of cypermethrin, ASAT levels surged to 190.02 ± 63.11 in the liver, 260.15 ± 85.59 in the kidney, and 196.27 ± 80.52 in the muscles. The group receiving 40 µl/b.wt displayed even higher levels, with liver, kidney, and muscle ASAT activities recorded at 198.63 ± 59.93, 196.27 ± 64.45, and 268.74 ± 123.99, respectively. When expressed as percentages compared to the control group (deemed 100%), the 20 µl/b.wt treatment caused increases of 283.58% in the liver, 375.93% in the kidney, and 293.49% in the muscles, while the 40 µl/b.wt group revealed increases of 296.41%, 283.52%, and 401.94% in those respective tissues. A related experiment involving feed-based exposure exhibited similar patterns. Chicks that consumed 1 µl/20 mg of cypermethrin showed ASAT levels of 140.71 ± 60.72 in the liver, 177.22 ± 71.76 in the kidney, and 196.49 ± 72.96 in the muscles, reflecting increases of 210%, 256.06%, and 293.79%, respectively. Those given 1 µl/40 mg cypermethrin had ASAT activities of 147.90 ± 79.60 in the liver, 169.07 ± 72.45 in the kidney, and 139.87 ± 74.68 in the muscles corresponding to 220.74%, 244.21%, and 209.27% above control values.

Table 3: Activity of ASAT in the Organs (Liver, Kidney and Muscles) of *Gallus domesticus* (Chicks) Exposed to Cypermethrin.

Experimental Groups	Organs	Mean	Std. Error	Minimum	Maximum	% Inhibition/ Activity

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Control	Liver	67.00	43.02	2.9	188.6	100
	Kidney	69.22	44.11	5.2	198.8	100
	Muscles	66.85	42.92	8.8	193.4	100
Induced 20µl/b.wt	Liver	190.02*	63.11	11.4	308.2	283.58
	Kidney	260.15*	85.59	35.8	402.7	375.93
	Muscles	196.27*	80.52	50.1	379.5	293.49
Induced 40µl/b.wt	Liver	198.63*	59.93	48.3	341.6	296.41
	Kidney	196.27*	64.45	54.7	350.1	283.52
	Muscles	268.74*	123.99	45.7	550	401.94
Treated with 1µl/20mg	Liver	140.71*	60.72	56.8	317.2	210
	Kidney	177.22*	71.76	29.7	374.2	256.06
	Muscles	196.49*	72.96	24.4	373.4	293.79
Treated with 1µl/40mg	Liver	147.90*	79.60	25	376.9	220.74
	Kidney	169.07*	72.45	29.7	372.8	244.21
	Muscles	139.87*	74.68	25.3	356.8	209.27

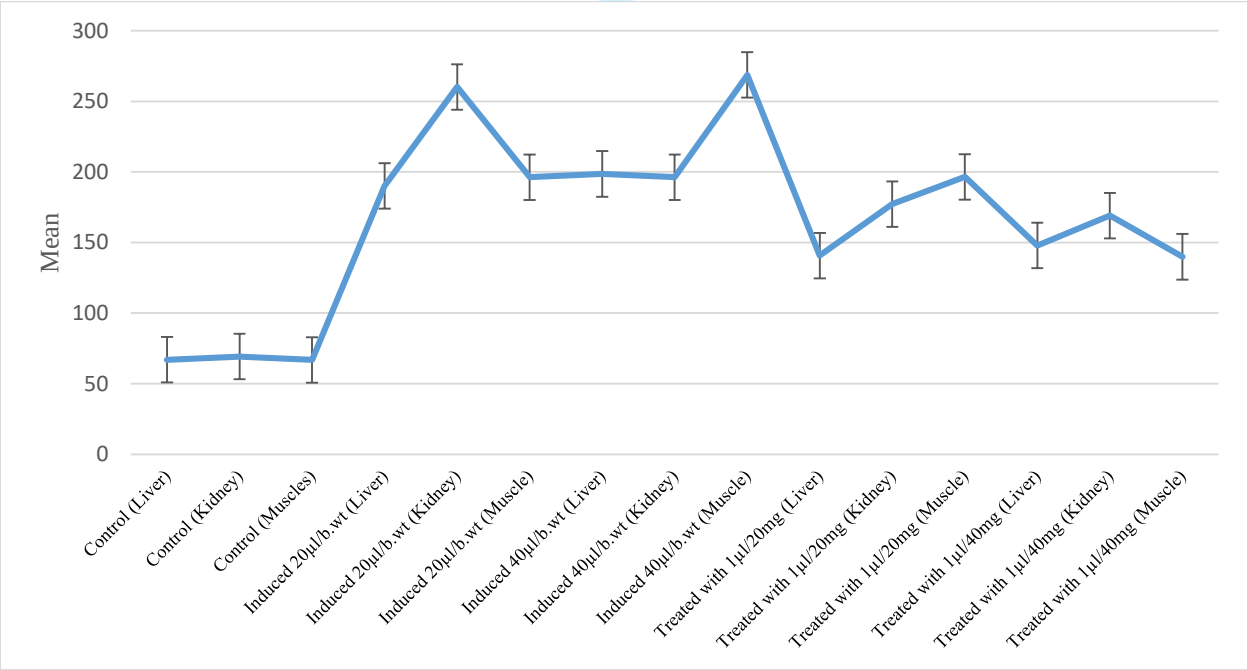

Figure 3: Activity of ASAT in the Organs (Liver, Kidney and Muscles) of *Gallus domesticus* (Chicks)

Exposed to Cypermethrin.

Overall in Figure 3, these findings indicate a distinct dose-dependent rise in ASAT activity, particularly in the kidney and muscle tissues, suggesting that cypermethrin causes substantial biochemical stress and potential cellular injury. The results reinforce the effectiveness of ASAT as a responsive biomarker for evaluating pesticide-related toxicity in poultry.

Table (4) shows a detailed statistical analysis of Aspartate Aminotransferase (ASAT) activity in *Gallus domesticus* over a span of 20 weeks showed notable, dose- and time-related variations between the control group and those exposed to cypermethrin. The control group displayed a steady increase in ASAT levels as they aged, rising from 5.65 ± 1.72 at 5 weeks to 193.58 ± 2.93 at 20 weeks. In comparison, chicks

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treated with 20 µl/b.wt of cypermethrin exhibited significantly higher ASAT levels, starting at 39.86 ± 17.75 at 5 weeks, reaching a peak of 330.28 ± 51.67 by 15 weeks, and then showing a slight decline to 222.12 ± 101.90 at 20 weeks. Likewise, the 40 µl/b.wt group maintained elevated ASAT activity throughout, with measurements of 122.99 ± 61.65, 126.23 ± 44.18, 320.19 ± 58.88, and 315.44 ± 143.57 at 5, 10, 15, and 20 weeks respectively. Statistically significant differences ($P<0.05$) were found between both treatment groups and the control group at each time point, especially at 5 and 10 weeks, indicating early liver stress due to cypermethrin exposure. By 15 weeks, while both treated groups continued to show significant differences from the control, there was no significant difference between them, indicating a trend towards similar ASAT activity after continuous exposure. However, by 20 weeks, all groups, including the two treatment groups, displayed significant differences from each other, with the highest ASAT activity recorded in the 40 µl/b.wt group. Supporting results from a feed-based exposure study corroborated these observations, with chicks treated with 1 µl/20 mg and 1 µl/40 mg cypermethrin exhibiting significantly higher ASAT levels at all intervals when compared to controls, particularly at 15 weeks, when both groups reached their highest enzyme activity but did not differ significantly from each other. By 20 weeks, the treated groups again showed significant divergence, highlighting the cumulative, dose-dependent toxicity of cypermethrin. High ASAT activity has been observed in the liver, heart, and muscles of various poultry species. Elevated levels of this enzyme are often associated with liver or muscle damage. The measurement of this enzyme offers better insights when evaluated alongside other basic tests; for instance, assessing creatine kinase levels can help determine if muscle damage is causing increased ASAT levels [33]. The findings of the current investigation were supportive of the aforementioned author. The liver and kidneys play a crucial role in processing and eliminating toxic substances like pesticides [34].

Table 4: Level (Mean ± S.E) of ASAT in the *Gallus domesticus* (chicks) Exposed to Cypermethrin after Different Time Intervals

Experimental Groups	After 5 Weeks	After 10 Weeks	After 15 Weeks	After 20 Weeks
Control	5.65 ± 1.718	17.27 ± 4.20	54.26 ± 6.535	193.58 ± 2.93
Induced 20µl/b.wt	39.86 ± 17.75	269.66 ± 26.98	330.28 ± 51.67	222.12 ± 101.90
Induced 40µl/b.wt	122.99 ± 61.65	126.23 ± 44.18	320.19 ± 58.88	315.44 ± 143.57
Treated with 1µl/20mg	36.95 ± 17.34	144.26 ± 17.32	354.92 ± 32.68	149.76 ± 84.12
Treated with 1µl/40mg	37.25 ± 17.02	75.08 ± 58.30	368.79 ± 10.61	128 ± 10.18

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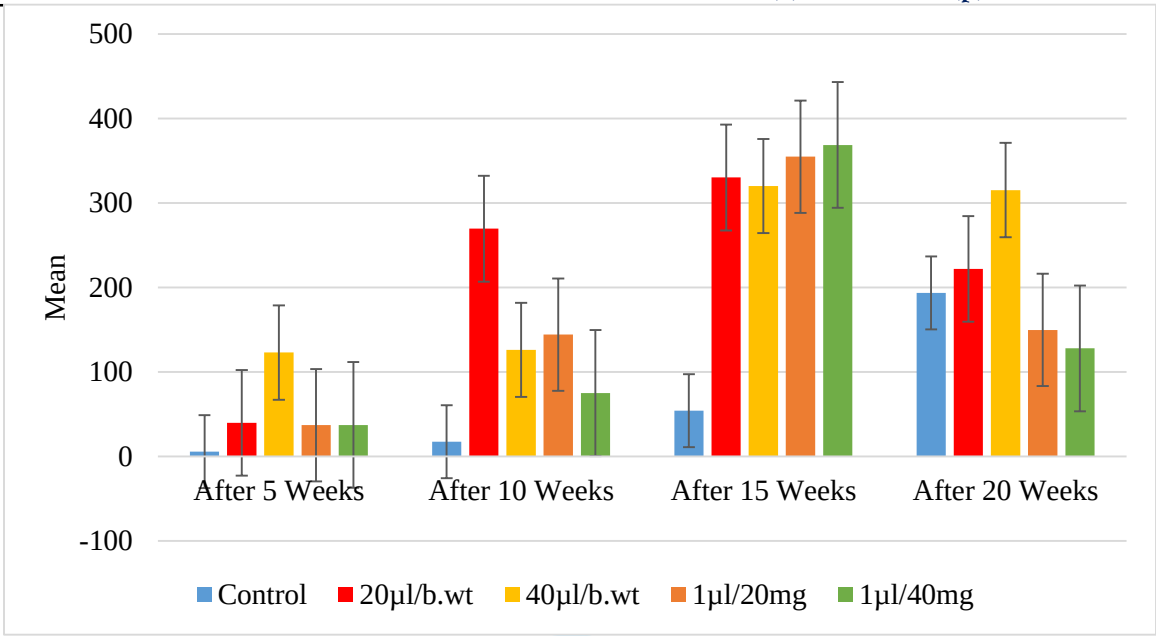

Figure 4: Level (Mean ± S.E) of ASAT in the *Gallus domesticus* (Chicks) Exposed to Cypermethrin after Different time Intervals.

Figure 4 confirms ASAT as an effective biomarker for assessing systemic biochemical stress in poultry resulting from prolonged pesticide exposure. The harmful effects of Cypermethrin and Malathion on *Channa punctate*, sublethal exposure led to elevated white blood cell counts, decreased red blood cell levels and hemoglobin, genetic damage, and severe tissue damage in the liver, kidneys, gills, and muscles [35]. These results emphasize that in addition to causing death, these pesticides result in considerable hematological, genetic, and organ-specific harm, highlighting their ecological dangers. In the present study, the livers of chicks (*Gallus domesticus*) subjected to 40µl/b.wt of cypermethrin and those given 1µl/20mg of cypermethrin mixed with their feed displayed a slightly dull appearance and irregular shape after 20 weeks of experimentation, unlike the control group. The levels of cypermethrin accumulation in the liver, peritoneal fat, breast muscle, and leg muscle of the chickens were generally low, seldom surpassing the detection limit. These findings align with previous studies where cypermethrin-treated mammals, chickens, and laying hens showed residue levels in their organs and tissues that were below detectable limits [36-39]. However, in this study, there was a slight increase in the organ levels of treated chicks when compared to the control group. Various other investigations on pesticides have confirmed a notable increase in ALAT and ASAT levels in mice that were administered 100 mg of deltamethrin per kilogram of body weight [40]. Furthermore, the goats treated with 6 and 12 mg/kg of LBW cypermethrin exhibited elevated ASAT and ALAT levels [41]. This increase may be attributed to the heightened secretory function of the liver [42]. The release of tissue enzymes into the hepatic plasma can also be explained by alterations in the permeability of hepatocyte membranes [43]. A significant increase in the weight of the liver and kidneys at varying doses of cypermethrin, this weight gain may be attributed to the enzyme-inducing effect of cypermethrin accumulating in these organs for detoxification [44]. Comparable results were observed in rats and guinea pigs treated with cypermethrin [45-46], however, rats exhibited opposite effects after cypermethrin exposure [47]. While the present study aligns with the findings of [48], the % activity of ALAT and ASAT was found to be elevated in the organs (liver, kidney, and muscles) of chicks (*Gallus domesticus*) following cypermethrin treatment, with control organs' activity set at 100%. The groups exposed to cypermethrin experienced a significant reduction in serum levels of ALAT, ASAT, creatinine, and urea. Thus, in Japanese quail that were poisoned

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with cypermethrin, the notable increases in plasma ASAT activity could be seen as the most reliable indicators of liver damage [48]. The levels of ASAT, ALAT, and creatinine in quails exposed to cypermethrin in this study showed a significant rise, while the urea concentration remained unchanged. In this investigation, the ALAT and ASAT levels in all treated groups displayed a notable correlation when compared to the control group. The alterations in the activity of specific serum enzymes, indicating increased ASAT activity and decreased ALP activity in both male and female birds [49]. Although these results imply that cypermethrin may adversely affect liver function, even at high doses, these alterations are unlikely to result in significant biological consequences. The exposure to pesticides negatively impacts livestock, soil microorganisms, flora, humans, and pollinators, leading to worldwide health hazards and ecological harm [50].

CONCLUSION

The environmental effects of a pesticide depend on both its level of toxicity and how it spreads in the environment. Various factors affect its distribution and harmfulness, involving multiple phenomena and species. Biological monitoring studies indicate that human exposure to pesticides is quite common. Various health problems can arise from exposure to cypermethrin in occupational or environmental settings, along with loss of biodiversity and other ecological disruptions caused by the careless and frequent use of cypermethrin. This investigation into enzymatic functions across various organs (including the liver, kidneys, and muscles) demonstrated that administration of cypermethrin led to its accumulation in the internal organs of treated chicks, where it changed the enzymatic activity levels. They stress the importance of decreasing pesticide usage and implementing alternative methods for pest control to minimize environmental pollution. It is recommended that cypermethrin pesticide be utilized in restricted amounts at safer sites. The agricultural sector should advocate for and use an alternative type of pesticide.

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