JSIANE : Vol. 1, Issue 2, 2021 Page 01–04

Journal of Science Innovations and Nature of Earth

Journal homepage : www.jsiane.com

Studies of the effect of Carbofuran Inbosting kidney of the *Channa punctatus* (Bloch.)

Pooja Kumari[1],Himanshi Goswami[1], Shivendra P. Chauhan[1], Prem Sagar[1], Vishan Kumar*[2],Anand Pratap Singh[3] and Surendra Singh[1]

1. **Department of Zoology, School of Life Sciences, Dr. B. R. Ambedkar University, Agra**
2. **Department of Zoology, NREC College, Khurja, C. C. S. University, Meerut.**
3. **Department of Zoology, Agra College, Agra**

*Corresponding author's email: vishankumar1000cc@gmail.com

Abstract

Water contamination is a big issue on a global scale. It has been stated that it is the leading cause of death and disease on a global scale, responsible for the deaths of thousands of people every day. Numerous pesticides are also a cause of pollution in agriculture, as they are sprayed on crops to protect them against pests and insects. They have an effect on the soil's structure and fertility. The purpose of this study is to determine the toxicity of the pesticide carbofuran on the fish *Channa punctatus*. The investigation will be conducted in the kidney and will include histopathological and biochemical examinations. The criteria chosen include urea, uric acid, calcium, potassium, sodium, and creatinine, which can result in catastrophic consequences in many fish diseases. *Channa punctatus* is a common freshwater fish that was gathered from September to October. It is a robust fish that adapts well to aquarium conditions. Carbofuran is a white, crystalline, powdered insecticide that was used for this study. The elevated serum urea, uric acid, and creatinine concentrations in *Channa punctatus* at different time intervals of 24, 48, 72, and 96 hours in comparison to the control group reflect compromised kidney functions caused by carbofuran poisoning.

Agriculture, Carbofuran, *Channa punctatus*, Pollution of water, Pesticide

Introduction

Aquatic systems have gained prominence as a result of growing concern about the consequences of human population growth and activity on aquatic life and water quality. Water contamination is a big issue on a global scale. It has been stated that it is the leading cause of death and disease on a global scale, responsible for the deaths of thousands of people every day. Water is often considered polluted when it is contaminated by anthropogenic contaminants and either becomes unfit for human use, such as drinking water, or exhibits a dramatic decline in its ability to support its constituent biotic populations, such as fish. Numerous pesticides are also a source of water pollution in agriculture, as they are sprayed on crops to protect them from pests and insects. They have an effect on the soil's structure and fertility. These pesticides are making their way into bodies of water such as rivers, ponds, the ocean, and lakes via runoff. Pesticides are one of the most efficient weapons mankind has devised for defending agricultural products against pest attack. However, widespread pesticide usage poses a persistent threat to aquatic life by modifying the habitat's behaviour pattern, development rate, and reproductive capacity. Contaminants that enter the body of an organism via absorption or other means penetrate deeply into the tissues, affecting the organism's physiology, biochemistry, and metabolism. Numerous fertilisers and insecticides with varying chemical compositions have been used to accomplish this goal, all of which have direct or indirect deadly effects on a variety of animals (Singh et al. 2010). Population growth, industry, urbanisation, and agricultural activities have all contributed to water contamination, which has become a major worry for humanity (Edori et al. 2013a). Carbofuran is a pesticide marketed under the trade name Furadan by the FMC Corporation of Philadelphia. It is used to control insects in a wide variety of field crops, including potatoes, corn, and soybeans. It is absorbed by plants via their roots, and insecticidal concentrations are achieved throughout. Carbofuran is one of the most harmful pesticides used on field crops due to its high acute toxicity. According to Kumar and Pant (1984), histopathological investigations are beneficial for evaluating pesticide contamination potential because trace amounts of pesticides that do not cause animal mortality over a particular length of time are capable of causing significant original harm.

Materials and Methods

The test animal was Channa punctatus (Bloch.). It has an elongated body with a large head and a tapering tail. It is frequently seen in fresh water. It is a tough fish that adapts well to aquarium conditions. The fish were taken between September and October, when the ambient temperature ranged between 25 and 30 degrees Celsius. Adult live *Channa punctatus* (Bloch.) specimens ranging in size from 16-18 cm and weighing 40–70 g were acquired from a local market. They were kept in laboratory conditions for 15 days in a huge glass aquarium. Every alternate day, dechlorinated water is utilised and changed. Numerous physiochemical properties of test water were routinely reported, including temperature, pH, and hardness. Five aquariums were employed in the experiment; one was used as a control, while the remaining four were used to research pollution. Each tank includes ten fish that were exposed to sublethal doses of carbofuran over a period of time (24, 48, 72 and 96 hour). Sublethal concentrations were chosen based on the LC_{50}

value. Blood samples were allowed to coagulate for approximately one hour before being centrifuged at 2000 rpm for 15 minutes. The supernatant serum was separated using a tiny rubber bulb pipette. The serum was analysed for urea, uric acid, and creatinine. The serum urea concentration was determined using the GLDH-Urase method, as reported by Young (1990). The serum creatinine concentration was determined using the alkaline picrate method established by Toro and Ackermann (1975). The present study examined the kidney toxicity of carbofuran insecticide in control and exposed Channapunctatus fish (Bloch.). The kidney function and ionic parameters were determined 24 hours, 48 hours, 72 hours, and 96 hours following carbofuran pesticide exposure. The mean, standard deviation (S.D.), standard error of the mean (S.Em.), and test of significance, the student's "t" test, were calculated using the data obtained (stat pac version 3.0).

Result and Discussion

For 24, 48, 72, and 96 hours, serum urea concentrations in Channa punctatus were 30.330.67 mg/dl in the control group. And For 24 hours, Channa punctatus serum urea concentration was 35.800.25 mg/dl, 39.990.12 mg/dl for 48 hours, 45.500.33 mg/dl for 72 hours, and 49.880.30 mg/dl for 96 hours, respectively. The serum urea level increases in response to treatment. After 24 and 48 hours of repeated carbofuran administration, the increase in serum urea was not significant, but was substantial after 72 hours and very highly significant after 96 hours (Table 1).

Within the Control Group For 24, 48, 72, and 96 hours, Channa punctatus had a serum uric acid concentration of 20.500.20 mg/dl. For 24 hours, Channa punctatus had a serum uric acid concentration of 23.600.27 mg/dl, 28.330.27 mg/dl for 48 hours, 33.100.19 mg/dl for 72 hours, and 39.500.22 mg/dl for 96 hours, respectively. The serum uric acid level increases in response to treatment. After 24 and 48 hours of repeated carbofuran administration, the increase in serum uric acid was not significant, but was substantial after 72 hours and very highly significant after 96 hours (Table 2).

Containment Group For 24, 48, 72, and 96 hours, the blood creatinine level in Channa punctatus was 1.020.04 mg/dl. For 24 hours, the serum creatinine concentration in channa punctatus was 1.180.04 mg/dl, 1.2530.06 mg/dl for 48 hours, 1.310.05 mg/dl for 72 hours, and 1.390.03 mg/dl for 96 hours, respectively. The serum creatinine level increases in response to treatment. After 24 and 48 hours of serial carbofuran administration, the increase in serum creatinine was not significant, but was significant after 72 hours and very highly significant after 96 hours. (Table-3).

Table 1. Serum urea (mg/dl) in *Channa punctatus* after carbofuran treatment

S.No.	Duration	No. of fishes	Control	Treatment
			Mean±S.Em.	Mean±S.Em.
1.	24hrs	5	30.33±0.67	35.80±0.25*
2.	48hrs	5	30.33±0.67	39.99±0.12**
3.	72hrs	5	30.33±0.67	45.50±0.33**
4.	96hrs	5	30.33±0.67	49.88±0.30***

* Non-significant, **Significant, *** highly significant, **** Very highly significant

Table 2. Serum uric acid (mg/dl) in *Channa punctatus* after carbofuran treatment

S.No.	Duration	No. of fishes	Control	Treatment
			Mean±S.Em.	Mean±S.Em.
1.	24hrs	5	20.50±0.20	23.60±0.18*
2.	48hrs	5	20.50±0.20	28.33±0.27**
3.	72hrs	5	20.50±0.20	33.10±0.19****
4.	96hrs	5	20.50±0.20	39.50±0.22****

* Non-significant, **Significant, *** highly significant, **** Very highly significant

Table 3. Serum creatinine (mg/dl) in *Channa punctatus* after carbofuran treatment

S.No.	Duration	No. of fishes	Control	Treatment
			Mean±S.Em.	Mean±S.Em.
1.	24hrs	5	1.02±0.04	1.18±0.04*
2.	48hrs	5	1.02±0.04	1.25±0.06*
3.	72hrs	5	1.02±0.04	1.31±0.05**
4.	96hrs	5	1.02±0.04	1.39±0.03***

* Non-significant, **Significant, *** highly significant, **** Very highly significant

Urea, uric acid, and creatinine are all tests for kidney function. Urea is classified as a nitrogenous non-protein waste (NPW). It is the major product of protein metabolism and is eliminated by the kidneys; its content is proportional to protein absorption. Serum creatinine, on the other hand, is an NPN waste product formed during the breakdown of creatine and phosphocreatine. It is less diet-dependent and is therefore an excellent indication of renal function. Under toxic stress, fish avoid water intake by slowing opercular motility and increasing gulping frequency. Serum urea, uric acid, and creatinine concentrations were raised in Channa punctatus at different time periods of 24, 48, 72, and 96 hours, respectively, indicating compromised kidney function due to carbofuran toxicity. Renal failure associated with severe renal insufficiency or excessive protein breakdown as a result of toxic stress may be responsible for blood urea and creatinine elevations. Because creatinine excretion is almost entirely dependent on the process of glomerular filtration, a significant increase in serum creatinine levels may be caused by impaired glomerular function, tubular damage in the kidney, or a lack of oxygen on the glomerular filtration rate, all of which result in pathological changes in the kidneys. The present investigation confirmed Gautam et al. (2014)'s observation that an increased serum urea level suggests a deficient glomerular filtration rate (GFR) of the kidney following the harmful effect of Nuvan (organophosphate) on Clarias batrachus. Deka and Mahanta (2015) observed that catfish Heteropneustes fossilis (Bloch.) grown in water treated with the organophosphate insecticide dichlorvus create significantly more urea than ammonia via the ornithine cycle. and Channa punctatus's serum uric acid and creatinine concentrations increase. Abdelmeguid et al. (2002) demonstrated a substantial rise in serum creatinine concentrations in fish taken in significantly and moderately polluted areas compared to fish farm areas. Similarly, Priya et al. (2012) found that serum creatinine levels increased marginally in the experimental group compared to control mice, and that the rise was associated with renal failure. Attia and El-Badawi (2015) likewise observed a substantial increase in creatinine concentrations as the duration of dithiopyr exposure increased.

To corroborate the present findings, Balasubramanian and Kumar (2013) and Kumar et al. (2017) revealed that rats exposed to sodium arsenite had higher urea and creatinine levels due to nephrotoxicity. Similarly, Bhanu and Deepak (2015) discovered that carbofuran dramatically increased urea and creatinine levels in the blood of Cyprinuscarpio. Shawky and Fatma (2015) showed an increase in plasma urea and creatinine levels in Tilapia spp. captured in a polluted Lake Qarun habitat. The quantity and severity of tissue damage caused by a given substance as a toxicant is determined by the compound's toxic potential in organisms' tissues (Tilak et al. 2001). Chemical damage susceptibility varies significantly amongst tissues and cells within the same animal. It is even more pronounced in certain animal groupings. However, the location of the chemical's primary effect may be dictated by its mechanism of action. Each poison's mechanism of action and pattern of tissue vulnerability have been clearly established, as well as the hazardous dose of each chemical at which a fairly standard characteristic pattern of tissue damage occurs. Carbofuran exposure resulted in significant abnormalities in the kidney, which began with tubular organisation disturbance. Following that, tubular epithelial cell degeneration and lympocytic infiltration were observed. With vacuolation, blood clotting in certain sinusoids, and glomerular degeneration, the majority of these clinical alterations remained. When the bodily burden of carbofuran grows, new proteins such as metallothionein are generated in the liver and kidney (Ooi and Law, 1989). Carbofuran has the greatest effect on membrane organelles such as mitochondria, endoplasmic reticulum, and nuclear envelope, causing disarray, rearrangement, and dysfunction. Thus, carbofuran is more likely to damage the proximal tubules, which contain more mitochondria, than the distal tubules. Generally speaking, the collecting ducts are more resistant to carbofuran exposure. The damage to collecting ducts is only visible in fish exposed to greater carbofuran concentrations. The presence of atrophic or pyknotic nuclei in the kidney of fish rises as the time course progresses. Nuclear mutations occur in fish in a manner similar to that observed in other animals (Copius Peereboom and Copius-Peereboom-Stegeman, 1981). It has been claimed that prior to a trophy and necrosis of cells in other animals, nuclear and nuclealar alterations are induced. At first, the alteration may have been part of a defuse mechanism, preventing the activation of synthetic or other activities in the cell, such as metallothionein production. However, prolonged treatment with carbofuran results in the condensation of nuclear material, resulting in the formation of darkly pigmented pyknotic nuclei. Similarly to the current findings, Mehjbeen

et al. (2017) demonstrated multiple biomarker responses (serum biochemistry, oxidative stress, genotoxicity, and Jerome et al. (2018) investigated metal uptake, peroxidation, and histopathological alterations in various tissues of Callinectesamnicola exposed to industrial effluent and reported kidney damage, while Jerome et al. (2018) investigated metal uptake, peroxidation, and histopathological alterations in various tissues of Callinectesamnicola exposed to industrial effluent and reported kidney damage.

Conclusion

In the present study, it was observed that the toxicity of carbofuran pesticide dependent on time duration and concentration. A highly significant increase has been observed in urea, uric acid and creatinine in Channa punctatus at 24 to 96 hours after carbofuran toxicity. The above mentioned results indicate that carbofuran is highly toxic to aquatic fauna.

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