EFFECT OF NICKEL ON SOME BIOCHEMICAL PARAMETERS OF TISSUES LIKE LIVER, KIDNEY, BRAIN AND OVARY OF THE FRESH WATER FISH Oreochromis mossambicus

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Received: May 9, 2018
Accepted: June 5, 2018

ABSTRACT
Water pollution is recognized as a potential threat to aquatic organisms. Water resources are polluted due to discharge of effluents from various industries, agricultural run-off having insecticides, pesticides, heavy metals, fertilizers, chemicals, sewage and other domestic wastes. The present study was designed to analyse the toxic effect of nickel on the fish Oreochromis mossambicus. The protein, glucose and cholesterol content of tissues from liver, kidney, brain and ovary were analysed. The protein content of all the tissues are significantly decreased upto 121.44%, Glucose content increased significantly upto 26.40%, Cholesterol increased significantly upto 23.37% and Lipid decreased upto 34.48% significantly.

Keywords:

INTRODUCTION
Nickel (Ni) is an important contaminant present at elevated concentrations in aquatic ecosystem that is currently impacted by the many industrial uses and natural ways (Eisler1998; Muysen et al. 2004). Ni concentrations, which are typically below 10 μg/L in non contaminated water, may reach as high as several hundreds to 1000 μg/L in highly contaminated water (Eisler1998). Although Ni is considered to be an essential for a wide variety of animals species, its essentiality to aquatic animals is not fully established (Muysen et al. 2004). Several studies reported a Ni-related depression of immune system both in vertebrates and invertebrates (Eisler1998; Vijayavel et al. 2009). For example, the exposure of the mud crab Scylla serrata to Ni has been reported to modulate the hemocytic defense system (Vijayavel et al. 2009). Also, the fish immune responses seem to be a sensitive target for the suppressive effects of Ni, decreasing the number of lymphocytes (Zelikoff1994; Zelikoff et al. 1996). In addition, Ni has been well studied in mammals due to its toxic effects on the immune system (Zhang et al. 2008). In this study we have tested the toxic effect of nickel on the fish Oreochromis mossambicus.

MATERIALS
The fish Oreochromis mossambicus belongs to the family Cichlidae of the order perciformes. The locally known Tilapia is a plentiful available fish in Sambalpur area and highly consumed by the people as food rich in protein. It is laterally compressed, and has a deep body with long dorsal fins, the front part of which has spines. Adults reach approximately 35 centimeters in length and up to 1.13 kg. Size and coloration may vary in captive and naturalized populations due to environmental and breeding pressures. It lives up to 11 years.

It is a remarkably robust and fecund fish, readily adapting to available food sources and breeding under suboptimal conditions. It also tolerates brackish water and survives temperatures below 50 °F (10 °C) and above 100 °F (38 °C). So this fish can easily survive in laboratory condition.

EXPERIMENTAL SET UP
Specimen were disinfected in 0.01% KMnO₄ and acclimatized to laboratory conditions for 15 days in many large glass aquaria of 40 liters capacity. The water used in aquaria was chlorine free and changed every alternate day. They were fed daily with rice bran and mustard oil cake up to 24 hr prior to initiation of experiment but no food was given during experimental period to determine LC₅₀ dose.

The experimental fishes were divided into several batches of 10 each, irrespective of sex and a body weight of 30-35 gram and length 10 to 15 cm. Various biochemical changes in the tissues in liver, kidney, brain and ovary were observed. Four groups of fishes were treated with 10, 20, 30 and 40 ppm Nickel for 30 days and a fifth group of fishes were kept as control. At a sub lethal dose of 10 ppm Nickel for 60 days and alternations in the blood parameters were analysed at 15, 30, 45 and 60 days of exposure. The alternations were subjected to t’ test to ascertain, whether the changes were significant or not.
Tissue Sampling

Tissues were collected from the organs like liver, kidney, brain and ovary and analysed for the glucose, cholesterol, protein and lipid contents after various days of exposure to nickel. Protein contents of the tissues were done by the method of Lowry et al. (1951) using folins phenol reagent. Cholesterol estimation was done by the method of Zlatkis et al. (1953). Method suggested by Floch (1957) was used to estimate lipid content. Yemm and Willis (1954) method as used to estimate glucose content.

RESULT

During the present course of investigation, there is significant reduction in the protein content of all the tissues like liver, brain, kidney and ovary of O. mossambicus on exposure to acute and chronic sub lethal doses of Nickel. Protein content of liver reduced significantly to 98.49%, kidney upto 97.25%, brain upto 62.50% and ovary upto 121.44% (Table 1, 2, 3, 4).

Due to exposure of nickel the glucose level of different tissues significantly increased with the increased concentration of nickel. Glucose content of liver increased upto 26.40%, kidney upto 21.33%, brain upto 22.55% and ovary upto 22.54%. Sobha et al (2007) also obtained similar increase in the glucose content in Catla catla when exposed to cadmium. Shastry and Sunita (1982, 1983) have reported alternations in the glucose levels in the tissues of Channa punctatus induced by cadmium, chromium and other heavy metals.

The cholesterol level of all the tissues showed significant increase when the fishes were treated with acute and chronic doses of nickel like liver cholesterol increase upto 23.37%, kidney upto 16.82%, brain upto 22.44% and ovary upto 20.54%. Hota (1999) also observed an increase in the cholesterol level of the liver of fishes treated with lead and arsenic respectively.

The presence of nickel showed significant decrease in lipid level in all the tissues. When treated with acute and chronic doses of nickel for 30 days the lipid content in liver decreased upto 28.38%, in kidney lipid decreased upto 28.44%, Brain upto 32.42% and ovary upto 34.48%.

Table 1: Different biochemical parameters of liver tissue for 30 days under the impact of nickel

<table>
<thead>
<tr>
<th>Concentration of nickel in ppm</th>
<th>Average protein content</th>
<th>Average Glucose content</th>
<th>Average cholesterol content</th>
<th>Average lipid content</th>
</tr>
</thead>
<tbody>
<tr>
<td>control</td>
<td>120</td>
<td>100</td>
<td>150</td>
<td>200</td>
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<tr>
<td>10 ppm</td>
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<td>90</td>
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<td>20 ppm</td>
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<td>30 ppm</td>
<td>90</td>
<td>70</td>
<td>120</td>
<td>170</td>
</tr>
<tr>
<td>40 ppm</td>
<td>80</td>
<td>60</td>
<td>110</td>
<td>160</td>
</tr>
</tbody>
</table>

Table 2: Different biochemical parameters of kidney for 30 days under the impact of nickel

<table>
<thead>
<tr>
<th>Concentration of nickel in ppm</th>
<th>Average protein content</th>
<th>Average Glucose content</th>
<th>Average cholesterol content</th>
<th>Average lipid content</th>
</tr>
</thead>
<tbody>
<tr>
<td>control</td>
<td>120</td>
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<tr>
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DISCUSSION

During the present investigation, it was observed that there was significant reduction in the protein content in the tissues of all the organs like liver, kidney, brain and ovary of the specimen fish *O. mossambicus* on exposure to acute and chronic sub lethal doses of nickel. Carbohydrates are the immediate source of energy in animals as well as fishes and proteins are spare energy source during prolonged stress conditions. Hence the steady and significant decrease in protein content noted during the period of present investigation after prolonged exposure to copper may be due to intensive proteolysis and glycolysis respectively. Radhakrishnaiah et.al. (1991) also observed increased amino acid and protease activity after chronic exposure to heavy metal zinc. Lynch et. al. (1969) suggested that reduced protein synthesis might be another cause of protein depletion.

In contrast to the protein and glucose level of all the tissues of the specimen *O. mossambicus*, the cholesterol level of all the tissues showed an increase, when the fishes were treated with acute and chronic doses of nickel. Similar increase in the cholesterol level was also noted by Sen et.al.(1992) when treated *Channa punctatus* with zinc. Karai et.al (1982) observed cholesterol increase in lead refinery factory workers. This decline in the cholesterol level may be due to structural damage of liver hepatocytes, which corroborates the observations of Skidmore (1970).

With the treatment of nickel the lipid level of all the fishes showed a significant decrease. The decrease in lipid content may be due to inhibition of lipid synthesis or increased utilization of stored lipids as a source of energy to conduct normal metabolic functions according to Shashi et. al (1989) when exposed to the fluoride in rabbits.

CONCLUSION

The present study concludes that the cause of death of fishes is not only due to gill damaged by heavy metals but also damages caused to vital organs like liver, brain, kidney and ovary. It is expected that
some physiological parameters may be also responsible for the damaged in these organs of the fish *O. mossambicus*.

**REFERENCE**