GENOTOXIC EFFECT OF NICKEL CHLORIDE AND ZINC SULPHATE ON FISH HYPOPHTHALMICHTHYS MOLITRIX

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ABSTRACT

The present investigation is to assess the genotoxic potential of nickel chloride and zinc sulphate on gill cells of silver carp Hypophthalmichthys molitrix. Fishes were exposed in sublethal concentration of nickel chloride 5.7 mg/l and zinc sulphate 6.8 mg/l, and sampled at 10, 20 and 30 days. Nickel chloride and zinc sulphate treated fishes exhibited an apparent increase in the aberration frequency and a decrease in the mitotic index as compared to control. Acentric fragment, chromatid break, endoreduplication, chromatid gap, centromeric fusion, ploidy, sticky plate, dicentric chromosome, clumping and partial sticky plates were some of the abnormalities observed. The chromosomal aberrations in the treated fishes were significant compared to control.

Keywords: Hypophthalmichthys molitrix, nickel chloride, zinc sulphate, gill cells, chromosomal aberration

INTRODUCTION

As fish may act as ‘sentinel’ organisms for indicating aquatic pollution, several species have been successfully used as test materials for detecting genotoxic activity in the aquatic environment (Kligerman et al., 1975; Hooftman, 1981; Manna and Mukherjee 1989). Fish, particularly those living in rivers or confined waters, also run the risk of direct or indirect exposure to various chemical mutagens present in the run-offs along with various toxicants, pesticides, industrial wastes, etc. Guha and Khuda-Bukhsh (2003). Analysis of metaphase chromosomes in fish for the occurrence of chromosome aberrations and sister-chromatid (SCE) in order to detect as well as quantify the extent of genotoxicity or point mutation induced by an agent has proven to be useful in fish models (Kligerman 1982, Krishnaja and Rege 1982, Manna, 1984). Manna et al., (1985); Monoharan and Prabhakaran (1994) were reported the genotoxic nature of heavy metal pollutants in fish. The genotoxic effects of cadmium chloride (CdCl₂) and Azadirachtin (Aza) were assessed singly and conjointly in a
fish, *Oreochromis mossambicus*, with endpoints such as chromosome aberrations (Chandra and Khuda-Bukhsh, 2004). The present investigation study was designed to find the genotoxicity induced by nickel chloride and zinc sulphate in the fish *H. molitrix*.

**MATERIAL AND METHODS**

Healthy specimens of *H. molitrix* measuring 5.5 ± 0.5 cm length and 6.0 ± 1.0 g weight were collected from Poondy reservoir near Chennai and brought to the laboratory in well-aerated containers. The fish were acclimatized to the laboratory conditions for 15 to 20 days prior to experimentation. Acute toxicity assay were carried and 96 h LCso value found to be 57 mg/l for nickel chloride and 68 mg/l for zinc sulphate. For genotoxicity assay fishes were exposed to sublethal concentration of nickel chloride 5.7 mg/l and zinc sulphate 6.8 mg/l individually. Fishes are sampled after 10, 20 and 30 days of exposure. The fishes were sacrificed and the gill was dissected out and the slides were screened for metaphase plates under the light microscope and microphotographs were taken using Carl Zeiss Photomicroscope.

**RESULTS AND DISCUSSION**

*H. molitrix* has 48 (2n=48) chromosomes. The diploid chromosome number is in conformity with earlier report (Khuda and Chakrabarti 1996; Yao et al. 1994). The varied frequency of chromosomal aberrations due to exposure of *H. molitrix* to nickel chloride were found to be indistinct chromosome morphology, sticky plate, chromatid gap, nucleolar organizer region, centromic fusion, dicentric chromosome clumping and ring chromosome (Fig.1). Zinc sulphate treated chromosome were found to be ring chromosome condensed chromosome short arm, polyploidy, dicentric chromosome, sticky plate, nucleolar organizer region, endoreduplication and centromic fusion (Fig.2). The frequencies of chromosomal aberrations were found to be decrease with in increase in duration in both nickel chloride and zinc sulphate treated fishes (Table 1). According to Cavas and Ergene-Gozukara (2003), differences in the micronucleus frequencies with time seem to be related to cell kinetics and replacement. One other reason could be that the gill cell are directly and continuously exposed to contaminated water.

Nickel chloride treated fishes exhibited higher chromosomal aberrations compared to zinc sulphate treated fishes. Chromosomal aberrations were analysed using t-test, and values are significant at p 0.05 level compared to the control. the present finding closely agrees with the findings of Manna *et al.* (1985) and Manoharan and Prabhakaran (1994) A1 Sabti, (1985a,b). Most of the toxic chemicals that produce genotoxic effects have been known to form reactive oxygen species as well as
TABLE 1: Frequency of chromosomal aberrations induced by nickel and zinc in the gill cells of *Hypophthalmichthys molitrix*.

<table>
<thead>
<tr>
<th>Treatment (days)</th>
<th>Metaphase plate studied</th>
<th>AF</th>
<th>CB</th>
<th>ED</th>
<th>CG</th>
<th>CF</th>
<th>PL</th>
<th>SP</th>
<th>DC</th>
<th>PS</th>
<th>Total No. of Aberrations</th>
<th>t-Value</th>
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<tbody>
<tr>
<td>Nickel 10</td>
<td>100</td>
<td>11</td>
<td>13</td>
<td>4</td>
<td>5</td>
<td>3</td>
<td>4</td>
<td>4</td>
<td>2</td>
<td>1</td>
<td>47</td>
<td>1.98</td>
</tr>
<tr>
<td>Nickel 20</td>
<td>100</td>
<td>9</td>
<td>8</td>
<td>3</td>
<td>4</td>
<td>2</td>
<td>4</td>
<td>5</td>
<td>2</td>
<td>-</td>
<td>37</td>
<td>1.96</td>
</tr>
<tr>
<td>Nickel 30</td>
<td>100</td>
<td>7</td>
<td>6</td>
<td>2</td>
<td>3</td>
<td>1</td>
<td>3</td>
<td>4</td>
<td>1</td>
<td>2</td>
<td>29</td>
<td>1.92</td>
</tr>
<tr>
<td>Zinc 10</td>
<td>100</td>
<td>10</td>
<td>11</td>
<td>4</td>
<td>5</td>
<td>3</td>
<td>4</td>
<td>3</td>
<td>2</td>
<td>1</td>
<td>43</td>
<td>1.97</td>
</tr>
<tr>
<td>Zinc 20</td>
<td>100</td>
<td>7</td>
<td>8</td>
<td>3</td>
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<td>4</td>
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<td>1</td>
<td>25</td>
<td>1.90</td>
</tr>
<tr>
<td>Control</td>
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<td>-</td>
<td>1</td>
<td>-</td>
<td>1</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>6</td>
<td></td>
</tr>
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</table>

Af - Acentric Fragment; CB - Chromatid Break; ED - Endo Reduplication; CG - Chromatid gap; CF - Centromeric Fusion; PL - Ploidy; SP - Sticky Plate; DC - Dicentric Chromosome; PS - Partial Sticky Plate.

*Values are significant at p 0.05% confidence limit.*
Fig. 1: Chromosomal aberrations induced by nickel in Hypophthalmichthys molitrix

A) Control chromosome
B) Indistinct chromosome morphology
C) Sticky plate
D) Chromatid Gap
E) Nucleolar Organizer Region
F) Centromic fusion
G) Dicentric Chromosome
H) Clumping
I) Ring chromosome
electrophilic free-radical metabolites that interact with DNA to cause disruptive changes (Chandra and Khuda-Bukhsh, 2004). Ni can bind to DNA and proteins in cells in vitro and to chromatin in vivo. Such binding to macromolecules could be correlated to the ability of Ni compounds to interfere with DNA synthesis and to induce slight increases in chromosome alterations, as well as its mutagenic action (Morita et al., 1991; Sarkar, 1995). It has also been suggested that Ni-induced abnormal DNA repair may be a mechanism for carcinogenesis (Au et al., 1994; Hartmann and Hartwig, 1998). In
conclusion, the present study reveals that the among the heavy metals nickel chloride and zinc sulphate, nickel chloride induces higher chromosomal aberration than zinc Sulphate in molitrix.

REFERENCES


