Full Length Research Paper

Toxic effect of potassium permanganate on Oreochromis niloticus based on hematological parameters and biomarkers of oxidative stress

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Potassium permanganate (KMnO₄) is an oxidizing agent used in aquaculture for many years. Despite its known efficacy for the treatment and prevention of fish diseases, information about their potential toxicity in non-target organisms is still very limited. The objective of this study was to evaluate the sublethal effects of potassium permanganate (1.0 and 4.0 mg L⁻¹) on Nile tilapia, Oreochromis niloticus, using hematological analysis and oxidative stress (reduced glutathione concentration, glutathione S-transferase activity, catalase and lipid peroxidation) as biomarkers. Fish exposed to a concentration at 1.0 mg L⁻¹ had no significant variations in blood parameters. At 4.0 mg L⁻¹, the changes in hematological parameters of the exposed animals indicated hemolysis as a result of the oxidizing action of potassium permanganate. Regarding the oxidative stress analyses, only the reduced glutathione presented significant increase in fish exposed to potassium permanganate, indicating adaptive and protection responses against the oxidative stress. This study demonstrated that concentrations of potassium permanganate (1.0 to 4.0 mg L⁻¹) usually recommended for the treatment of fish diseases, can be toxic to Nile tilapia (O. niloticus).

Key words: Potassium permanganate, Nile tilapia, hematology, oxidative stress.

INTRODUCTION

Potassium permanganate is an oxidizing agent used for many years in aquaculture for the treatment or prevention of diseases in fish, removal of parasites, disinfection of fish tanks and aquariums, reduction of products toxic to fish as rotenone and antimycin, and for the control of fungi and algae for managing problems when temporary reduction of oxygen in tanks occurs (Lay, 1971; Duncan, 1974). Despite its widespread use for the treatment of diseases in cultivated fish, this chemical compound is not approved by the U.S. Food and Drug Administration as a therapeutic agent in aquaculture (FDA, 2007).

Potassium permanganate is potentially toxic to humans and other organisms (Kegley et al., 2010), including several fish species (Marking and Bills, 1975; Cruz and Tamse, 1989; Bills et al., 1993; Straus, 2004; Silva et al., 2006) but is not included in regulatory categories. Therefore, there is no pattern or established limit to this compound and it may be used freely and without restriction in aquaculture. According to the FDA (2007), the inclusion of this compound in a regulatory class still depends on further studies. Several studies on the implementation and effectiveness of potassium permanganate in aquaculture are available in the literature, but information

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about its toxic potential in organs and tissues in fish is still scanty.

The impact of contaminants on the aquatic ecosystem can be measured through a variety of parameters, from low levels of biological organization (molecular and biochemical responses) to high organization levels (population and community response) (Au, 2004). In this context, the use of biomarkers at cellular and molecular levels is extremely important as a sensitive tool to measure the biological effects during the assessment of environmental quality (Sarkar et al., 2006), since they are more specific, sensitive, reproducible and easy to determine (Au, 2004).

The Nile tilapia, *O. niloticus* (Linnaeus, 1758) was selected as the test organism in this study for its great aquaculture and commercial value. Tilapia is the third most important cultured fish group in the world, after carps and salmonids. Their hardiness and adaptability to a wide range of culture systems has led to the commercialization of tilapia production in more than 100 countries. Nile tilapia represents approximately 84% of total global tilapia production. In 2008, Nile tilapia culture alone was ranked fifth among the most cultured species in the world, with a total aquaculture production of 2.3 million tonnes (FAO, 2009). In 2010, it is anticipated that global Nile tilapia production will reach nearly 2.5 million tonnes (FAO, 2010).

The objective of this study was to evaluate the sub-lethal effects of potassium permanganate for tilapia, *O. niloticus*, using the hematological analyses and oxidative stress as biomarkers.

**MATERIALS AND METHODS**

This study was carried out at the Laboratory of Pathology of Aquatic Organisms (Fisheries Institute, Sao Paulo State, Brazil) under a controlled environment. The fish used as test organisms were juveniles of tilapia, *O. niloticus*, purchased in commercial fish farming, with mean weight of 44.52 ± 11.07 g and mean length of 13.87 ± 1.14 cm. Fish were previously acclimated for 15 days in the same environmental conditions of testing containers, that is, they were randomly distributed into three 300 L tanks equipped with artificial aeration and biological filters previously matured, at the density of 70 fish per tank (7.6 g L⁻¹). The water quality variables were measured daily during the period of acclimation and they are appropriate for the specie in the initial conditions: temperature 22 ± 0.7°C; pH 7.7 ± 0.2; dissolved oxygen 6.9 ± 0.3 mg L⁻¹ and total ammonia between 0.33 and 0.40 mg NH₄ L⁻¹. Test solutions were set up by dissolving potassium permanganate (K₂MnO₄ Merck™ in dechlorinated tap water. Nominal test concentrations of potassium permanganate (1.0 and 4.0 mg L⁻¹) were used in the assays, and it was chosen based on minimum and maximum doses of this compound reported in the literature to treat diseases in fish (Francis-Floyd and Klinger, 1997). A control group with no addition of potassium permanganate was also carried out in the same conditions of testing containers.

Test solutions were partially replaced every three days at the proportion of 1/3 of total volume (renewal test system). Fish were fed with commercial ration 32% crude protein, and food residues were cleaned up at every operation of water replacement.

The assay was carried out for 30 days, with sampling of 10 individuals per group for the hematological analyses and biochemical after 7, 15 and 30 days of exposure time.

For hematological analyses, the animals were anesthetized with benzocaine (3%); blood was drawn by caudal puncture, with the help of a needle and syringe previously heparinized. The blood specimens were assayed for: number of erythrocytes (RBC), counted in a Neubauer chamber; hemocrit (Ht) by the microhematocrit technique; and hemoglobin level (Hb) by the cyanomethemoglobin method. After these procedures, the following RBC indices were calculated: mean corpuscular volume (MCV), mean corpuscular hemoglobin (MCH) and mean corpuscular hemoglobin concentration (MCHC). Smears were made using the same blood aliquots, and the slides were later stained with May-Grunwald-Giemsa, which were utilized for total leukocyte number (WBC) and different cell count for each type of leukocyte (lymphocytes, neutrophils, basophils, eosinophils and monocytes) and total thrombocytes.

The biochemical analyses were performed at the Laboratory of Cell Toxicology, in the Federal University of Paraná (UFPR), Curitiba, Parana State, Brazil. Fish were sacrificed by deep sedation with benzocaine. Portions of the liver were sampled and maintained at ~80°C freezer before performing analysis. Then, samples were homogenized in phosphate buffered saline (PBS, pH 7.4) and frozen. Further, aliquots were separated from samples to the determinations of total protein concentration, glutathione S-transferase (GST) and catalase (CAT) activities, as well as the concentrations of reduced glutathione (GSH) and lipid peroxidation (LPO). Total protein concentration was determined by the Bradford method (Bradford, 1976). The activity of glutathione S-transferase was monitored by the reaction of the substrate 1-chloro-2,4-dinitrobenzene with GSH, generating a thioether that absorbs light at 340 nm (Habig et al., 1974; Keen et al., 1976). The concentration of GSH was determined according to Sediak and Lindsay (1968) after protein precipitation with 10% of trichloroacetic acid, and the activity of CAT was determined according to Aebi (1984). Lipid peroxidation was determined by the FOX method (Ferrous Oxidation/ Xylenol Orange Method) according to Jiang et al. (1991, 1992).

In order to assure that the probable toxic effects observed were attributed to potassium permanganate, the following physical and chemical variables of water were measured at the beginning of the experiment and then at every 24 h: temperature (°C) and dissolved oxygen (mg L⁻¹ and % saturation) were measured using the oximeter YSI - Mod.55; pH values were obtained using potentiometer and the electrical conductivity (μScm⁻¹) using conductivimeter. The analysis of hardness was done by the titration method with EDTA, alkalinity (mg CaCO₃ L⁻¹) by titration. The following analyses were performed weekly: the hardness, alkalinity (mg CaCO₃ L⁻¹) and total ammonia (mg NH₄ L⁻¹) according to standardized methodology in APHA et al. (2005). Non-ionized ammonia (NH₃, mg L⁻¹) was calculated according to Bower and Bidwell (1978).

For data analyses we used the statistical program TOXSTAT 3.5 (West and Gulley, 1996). Data normality was tested by Shapiro-Wilk’s test and homoscedasticity of variances by the Bartlett’s test. Then, data was analyzed using the statistical test ANOVA. Alterations were considered significant when P < 0.05.

**RESULTS**

Survival rate of 100% was observed in experiments and all fish appeared healthy. Physical and chemical variables...
The animals exposed to a concentration at 4 mg L\(^{-1}\) KMnO\(_4\) had a significant reduction (P<0.05) in red blood cells on the seventh and 15\(^{th}\) day after collection, and a reduction of hematocrit on the 30\(^{th}\) day (Table 1). On the seventh day of collection, a significant increase of MCH (P<0.05) was detected in fish exposed to a concentration of 4 mg L\(^{-1}\) KMnO\(_4\). Table 1. Mean values and standard error of the mean of red blood series of Oreochromis niloticus exposed to potassium permanganate.

<table>
<thead>
<tr>
<th>Treatments</th>
<th>RBC (10(^6)µL(^{-1}))</th>
<th>Ht (%)</th>
<th>Hb (g dL(^{-1}))</th>
<th>MCH (pg)</th>
<th>MCV (fL)</th>
<th>MCHC (%)</th>
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<tbody>
<tr>
<td>7 days</td>
<td></td>
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</tr>
<tr>
<td>Control</td>
<td>2.03 ± 0.05(^{a})</td>
<td>30.10 ± 1.10(^{a})</td>
<td>6.43 ± 0.25(^{a})</td>
<td>32.01 ± 1.83(^{a})</td>
<td>149.92 ± 0.83(^{a})</td>
<td>21.39 ± 0.48(^{a})</td>
</tr>
<tr>
<td>1 mg L(^{-1})</td>
<td>2.11 ± 0.07(^{a})</td>
<td>27.20 ± 0.36(^{a})</td>
<td>5.92 ± 0.36(^{a})</td>
<td>28.47 ± 2.13(^{a})</td>
<td>130.29 ± 0.77(^{a})</td>
<td>21.86 ± 1.04(^{a})</td>
</tr>
<tr>
<td>4 mg L(^{-1})</td>
<td>1.52 ± 0.06(^{b})</td>
<td>26.85 ± 1.82(^{a})</td>
<td>6.41 ± 0.54(^{a})</td>
<td>42.19 ± 3.36(^{a})</td>
<td>177.78 ± 12.55(^{a})</td>
<td>23.60 ± 0.82(^{a})</td>
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<tr>
<td>15 days</td>
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<tr>
<td>Control</td>
<td>1.98 ± 0.06(^{a})</td>
<td>28.00 ± 1.08(^{a})</td>
<td>6.67 ± 0.36(^{a})</td>
<td>33.58 ± 1.39(^{a})</td>
<td>141.39 ± 0.83(^{a})</td>
<td>24.09 ± 1.42(^{a})</td>
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<tr>
<td>1 mg L(^{-1})</td>
<td>1.86 ± 0.06(^{ab})</td>
<td>29.80 ± 0.26(^{a})</td>
<td>5.92 ± 0.36(^{a})</td>
<td>39.49 ± 1.48(^{a})</td>
<td>161.15 ± 0.77(^{a})</td>
<td>24.69 ± 0.96(^{a})</td>
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<tr>
<td>4 mg L(^{-1})</td>
<td>1.71 ± 0.06(^{bc})</td>
<td>26.45 ± 0.78(^{a})</td>
<td>6.41 ± 0.54(^{a})</td>
<td>35.20 ± 2.44(^{a})</td>
<td>156.03 ± 12.55(^{a})</td>
<td>22.70 ± 1.45(^{a})</td>
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<tr>
<td>30 days</td>
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<tr>
<td>Control</td>
<td>1.79 ± 0.13(^{a})</td>
<td>28.25 ± 1.07(^{a})</td>
<td>6.58 ± 0.32(^{a})</td>
<td>37.58 ± 2.07(^{a})</td>
<td>162.08 ± 0.83(^{a})</td>
<td>23.32 ± 0.83(^{a})</td>
</tr>
<tr>
<td>1 mg L(^{-1})</td>
<td>1.48 ± 0.09(^{a})</td>
<td>26.50 ± 0.92(^{ab})</td>
<td>6.19 ± 0.37(^{a})</td>
<td>42.62 ± 2.48(^{a})</td>
<td>183.71 ± 0.99(^{a})</td>
<td>23.27 ± 0.95(^{a})</td>
</tr>
<tr>
<td>4 mg L(^{-1})</td>
<td>1.64 ± 0.11(^{bc})</td>
<td>24.15 ± 0.87(^{bc})</td>
<td>6.09 ± 0.18(^{a})</td>
<td>38.18 ± 2.03(^{a})</td>
<td>152.68 ± 11.56(^{a})</td>
<td>25.42 ± 0.92(^{a})</td>
</tr>
</tbody>
</table>

\(^{a,b}\) Different letters indicate significant different (P < 0.05) mean values between treatments. RBC, Number of erythrocytes; Ht: hematocrit; Hb, hemoglobin; MCH, mean corpuscular hemoglobin; MCV, mean corpuscular volume; MCHC, mean corpuscular hemoglobin concentration.

DISCUSSION

The reduced values of hematimetric values found in this present study suggest the induction of anemia caused by the toxicity of potassium permanganate. The anemia may be due to the inhibition of erythropoiesis and hematopoiesis and to an increase in the rate of erythrocyte destruction in hemopoietic organs, resulting in a reduced RBC (Adhikari et al., 2004). Other xenobiotics also induce changes that give evidence of decreased hematopoiesis followed by anemia induction in fish, for example, changes in erythrocyte profile induced by sublethal exposure to cypermethrin and carbofuran in Labeo rohita (Adhikari et al., 2004), diazinon in Cyprinus carpio (Banaee et al., 2008), mercuric chloride in Oreochromis niloticus (Isikawa et al., 2007), deltamethrin in Cyprinus carpio (Svoboda et al., 2003) and chromium in Labeo rohita (Vutukuru, 2005). The toxicity of potassium permanganate (Figure 1A). No significant change occurred in the activity of GST, CAT and LPO levels in fish exposed to potassium permanganate (Figure 1B to D).
Table 2. Mean values and standard error of the mean of leukocytes and thrombocytes of *O. niloticus* exposed to potassium permanganate.

<table>
<thead>
<tr>
<th>Treatments</th>
<th>Lc (10^4 µL⁻¹)</th>
<th>Lf (10^4 µL⁻¹)</th>
<th>Nt (10^4 µL⁻¹)</th>
<th>Mn (10^4 µL⁻¹)</th>
<th>Tr (10^4 µL⁻¹)</th>
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<tr>
<td>7 days</td>
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<tr>
<td>Control</td>
<td>4.45 ± 0.66&lt;sup&gt;a&lt;/sup&gt;</td>
<td>4.27 ± 0.64&lt;sup&gt;a&lt;/sup&gt;</td>
<td>0.07 ± 0.01&lt;sup&gt;a&lt;/sup&gt;</td>
<td>0.08 ± 0.03&lt;sup&gt;a&lt;/sup&gt;</td>
<td>0.44 ± 0.14&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>1 mg L⁻¹</td>
<td>4.86 ± 0.67&lt;sup&gt;a&lt;/sup&gt;</td>
<td>4.65 ± 0.69&lt;sup&gt;a&lt;/sup&gt;</td>
<td>0.12 ± 0.05&lt;sup&gt;a&lt;/sup&gt;</td>
<td>0.07 ± 0.02&lt;sup&gt;a&lt;/sup&gt;</td>
<td>1.68 ± 0.30&lt;sup&gt;b&lt;/sup&gt;</td>
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<tr>
<td>4 mg L⁻¹</td>
<td>2.78 ± 0.38&lt;sup&gt;b&lt;/sup&gt;</td>
<td>2.65 ± 0.39&lt;sup&gt;b&lt;/sup&gt;</td>
<td>0.08 ± 0.03&lt;sup&gt;a&lt;/sup&gt;</td>
<td>0.04 ± 0.01&lt;sup&gt;a&lt;/sup&gt;</td>
<td>1.22 ± 0.38&lt;sup&gt;a&lt;/sup&gt;</td>
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<tr>
<td>15 days</td>
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<tr>
<td>Control</td>
<td>4.24 ± 0.45&lt;sup&gt;a&lt;/sup&gt;</td>
<td>4.13 ± 0.45&lt;sup&gt;a&lt;/sup&gt;</td>
<td>0.05 ± 0.01&lt;sup&gt;a&lt;/sup&gt;</td>
<td>0.06 ± 0.01&lt;sup&gt;a&lt;/sup&gt;</td>
<td>0.65 ± 0.19&lt;sup&gt;a&lt;/sup&gt;</td>
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<tr>
<td>1 mg L⁻¹</td>
<td>2.84 ± 0.44&lt;sup&gt;a&lt;/sup&gt;</td>
<td>2.75 ± 0.43&lt;sup&gt;a&lt;/sup&gt;</td>
<td>0.05 ± 0.02&lt;sup&gt;a&lt;/sup&gt;</td>
<td>0.03 ± 0.01&lt;sup&gt;a&lt;/sup&gt;</td>
<td>1.05 ± 0.25&lt;sup&gt;a&lt;/sup&gt;</td>
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<tr>
<td>4 mg L⁻¹</td>
<td>2.84 ± 0.51&lt;sup&gt;a&lt;/sup&gt;</td>
<td>2.77 ± 0.51&lt;sup&gt;a&lt;/sup&gt;</td>
<td>0.03 ± 0.01&lt;sup&gt;a&lt;/sup&gt;</td>
<td>0.05 ± 0.01&lt;sup&gt;a&lt;/sup&gt;</td>
<td>0.94 ± 0.23&lt;sup&gt;a&lt;/sup&gt;</td>
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<td>30 days</td>
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<tr>
<td>Control</td>
<td>3.70 ± 0.22&lt;sup&gt;a&lt;/sup&gt;</td>
<td>3.53 ± 0.20&lt;sup&gt;a&lt;/sup&gt;</td>
<td>0.07 ± 0.02&lt;sup&gt;a&lt;/sup&gt;</td>
<td>0.07 ± 0.01&lt;sup&gt;a&lt;/sup&gt;</td>
<td>0.49 ± 0.14&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>1 mg L⁻¹</td>
<td>2.00 ± 0.25&lt;sup&gt;b&lt;/sup&gt;</td>
<td>1.92 ± 0.25&lt;sup&gt;b&lt;/sup&gt;</td>
<td>0.03 ± 0.01&lt;sup&gt;a&lt;/sup&gt;</td>
<td>0.04 ± 0.01&lt;sup&gt;a&lt;/sup&gt;</td>
<td>0.76 ± 0.22&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>4 mg L⁻¹</td>
<td>2.06 ± 0.31&lt;sup&gt;b&lt;/sup&gt;</td>
<td>1.99 ± 0.31&lt;sup&gt;b&lt;/sup&gt;</td>
<td>0.02 ± 0.01&lt;sup&gt;a&lt;/sup&gt;</td>
<td>0.04 ± 0.01&lt;sup&gt;a&lt;/sup&gt;</td>
<td>0.51 ± 0.12&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
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</table>

<sup>ab</sup> Different letters indicate significant different (*P* < 0.05) mean values between treatments. Lc, Leukocytes; Lf, lymphocytes; Nt, neutrophils; Mn, monocytes; Tr, thrombocytes.

Figure 1. Concentration of GSH (A); activity of GST (B); activity of CAT (C); Lipid peroxidation (D), in liver of *O. niloticus*, exposed to potassium permanganate. Data are reported as mean ± standard error (*n*=10). *: Significant difference from control group (*P* < 0.05).
of MCV was higher than other groups, but without significant difference this indicates that the same amount of hemoglobin has occupied a larger volume in the cell. These results suggested the presence of a great amount of old and large erythrocytes, probably owing the prolonged mechanism or failure to replace these cells (Oliveira-Ribeiro et al., 2006), inducing a macrocytic anemia. Changes in the differential count of leukocytes provide an evidence for a decrease in non-specific immunity in fish after exposure to toxic substances (Svoboda et al., 2003; Oliveira-Ribeiro et al., 2006; El-Sayed et al., 2007). Thus, the leukopenia observed in fish exposed to potassium permanganate is primarily assigned to the reduction in the number of lymphocytes probably due to a reduced production and rapid destruction, leading to an increased rate of removal of these cells. Svoboda et al. (2001) and Banaee et al. (2008) observed a decrease in the percentage of lymphocytes and monocytes in Cyprinus carpio after exposure to diazinon, indicating a decrease in immunity, in response to stress caused by the toxic substance, corroborating the results of this present study.

The increased number of thrombocytes registered in stressed fish after being exposed to pollutants is related to damage to organs and tissues (Mazon et al., 2002; Gabriel et al., 2007). According to Francis-Floyd and Klinger (1997), the potassium permanganate is a compound capable of reacting indiscriminately with any organic material in the water, including fish tissues like skin and gills. Since it is a strong oxidizing agent, the high values of thrombocytes found in fish of both treatments (1.0 and 4.0 mg L\(^{-1}\) KMnO\(_4\)) can be associated with injuries in tissues such as skin and gills, once these organs are directly exposed to this chemical in the water.

According to Zhang et al. (2004), under severe stress, suppression in the levels of GSH may occur, due to the reduction capacity of synthesis, regeneration or oxidation of GSH. Moreover, the increase in GSH observed in this study reflects the stimulation of the detoxification metabolism, suggesting an adaptive response and a protection response of this biomolecule against a rapid oxidative stress, induced by contaminants (Pandey et al., 2003; Zhang et al., 2004; Farombi et al., 2007).

As observed in this study, Ventura-Lima et al. (2009) found no significant change in the levels of GST in Danio rerio exposed to arsenic, indicating that the tested concentrations were not deleterious enough to interfere in the activity of this enzyme. Sanchez et al. (2005) reported that the exposure to copper promoted antioxidant response in fish, revealed by the rapid increase of SOD (superoxide dismutase), catalase and GPx (glutathione peroxidase), but significant change was not observed for GST.

However, as observed for the GST, CAT and LPO levels did not vary significantly (Figures 1B to D). This may be associated with the increased levels of GSH observed, to balance the oxidative stress and as adaptive response to avoid the increase of reactive oxygen species (ROS) and oxidative damage. Similar results were reported by Ventura-Lima et al. (2009), where the absence of changes in the activity of GST, CAT and LPO levels in fish exposed to arsenic was due to the effectiveness of the increased levels of GSH for preventing additional oxidative disturbances. Thus, the results of this present study confirm that GSH is often the first defense against oxidative stress.

**Conclusion**

Information on the physiological changes in fish by acute and subchronic exposure to potassium permanganate is limited. Study performed by França et al. (2011) in O. niloticus, Kori-Siakpere (2008) in Clarias gariepinus and Silva et al. (2006) in Colossoma macropomum, revealed that the compost has high toxicity to fish in a short term exposure.

The results obtained in this present study have demonstrated that the prolonged use of concentrations considered safe by literature for prophylaxis and treatment of diseases in fish farming (1.0 and 4.0 mg L\(^{-1}\) KMnO\(_4\)) may cause considerable changes to health of exposed fish, evidencing the toxic potential of potassium permanganate in non-target organisms. Such alterations, especially in hematological variables, have pointed out that potassium permanganate induced the destruction of red blood cells in a short period of exposure to the chemical, indicating the direct action of its toxicity. Therefore, the use of different biomarkers become important as they can reflect more accurately the toxicity of contaminant substances studied and their effects impacting the aquatic ecosystem.

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