The effect of waterborne iron on the carp (*Cyprinus carpio* L.) liver: a comparison of two iron salts

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Iron is a metal of vital importance for many organisms as it plays an essential role in numerous metabolic processes. It is an integral part of various proteins such as haemoglobin, myoglobin, cytochromes as well as of numerous enzymes such as catalases, peroxidases, dehydrogenases (succinate dehydrogenase, mitochondrial NADH dehydrogenase) and enzymes involved in DNA synthesis. Although essential, iron can be toxic when present in excess as it catalyses the generation of wide range of free radicals, including the hydroxyl radical, which can react with a large number of cell components (DNA, proteins, lipids, sugars) and can cause serious cellular damage [1].

The aim of this study was to monitor iron accumulation in carp liver tissue during long-term exposure to maximum allowed waterborne iron concentration (1 mg/L) in the form of iron-gluconate (FeG) and iron-dextran (FeD). Histological changes in liver and the activity of lactate dehydrogenase (LDH), a marker for cell/tissue damage, were also examined [2, 3]. Carp were held for 70 days in aquaria with constant iron concentration (1 mg/L). Liver tissue samples of control and exposed animals were taken every day during the first four days of the treatment and in intervals of five or seven days until the final 70th day of the treatment. Histochemical modified Perl´s reaction was used to detect iron in liver tissue [3]. Iron depositions were observed by light microscope and analyzed with image analyser (LUCIA G 4.81).

In carp liver of the control group, Perl´s reaction showed no iron deposits (Fig. 2). Iron started to accumulate on the 28th day of the treatment with both FeG and FeD and deposits were located in the sinusoid endothelium as mildly diffused reactions. Much less was found in the cytoplasm of hepatocytes in the form of grain and diffused reaction. After the 35th day of the treatment, iron was also accumulated in macrophages. Continued treatment with FeG and FeD increased the quantity of iron depositions in liver tissue (Fig. 1, 2). The results indicate that long-term exposure of carp to low iron concentrations causes significant Fe accumulation ($p<0, 05$) in carp liver. Both iron salts increased cytoplasmic LDH activity (Fig. 3). These results indicate that the maximum allowed waterborne iron concentration during the prolonged treatment is sufficiently high to induce histopathological changes.

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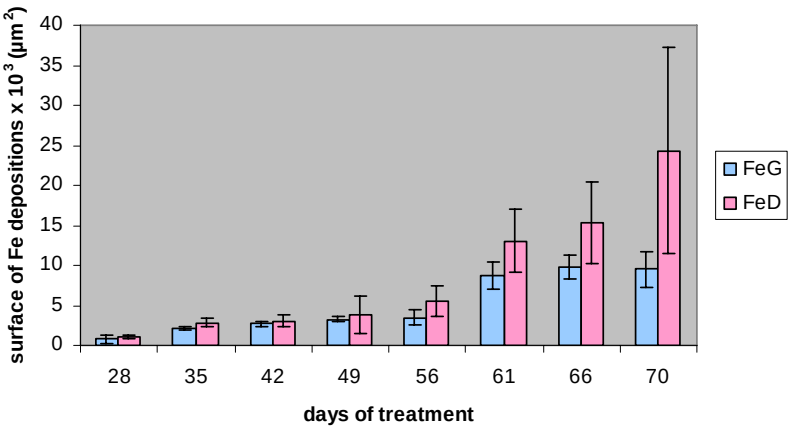

Figure 1. Average values of Perl's positive surfaces in liver tissue during long-term exposure of carp to FeG and FeD. Four samples per day were analysed. All values were significantly different from the controls ($p<0.05$; analysis of variance). Bars represent mean values ± S.E.

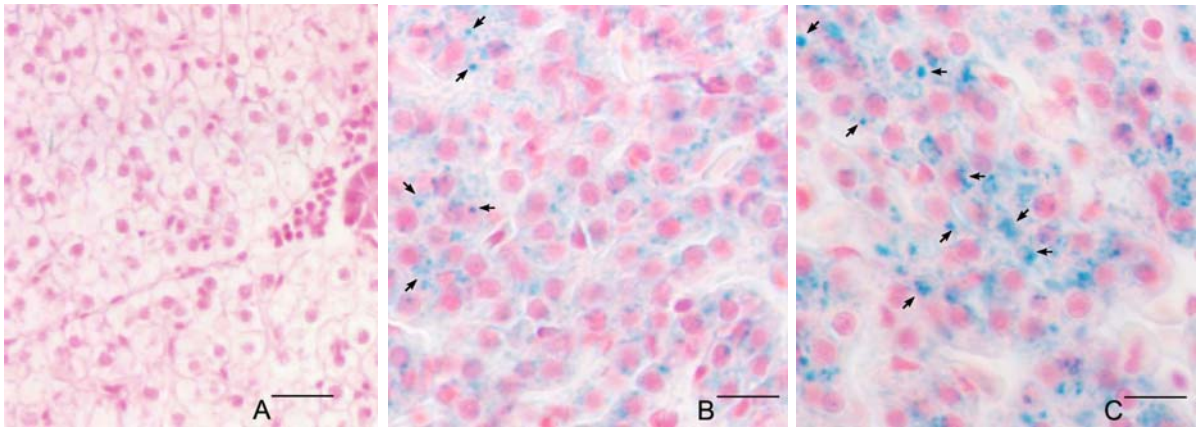

Figure 2. Perls-stained sections of carp liver. In control group, Perl's reaction showed no iron deposits (A). Heavy iron deposits in liver cells (arrowhead) on the 70th day of the treatment with FeG (B) and FeD (C). Bar = 25µm

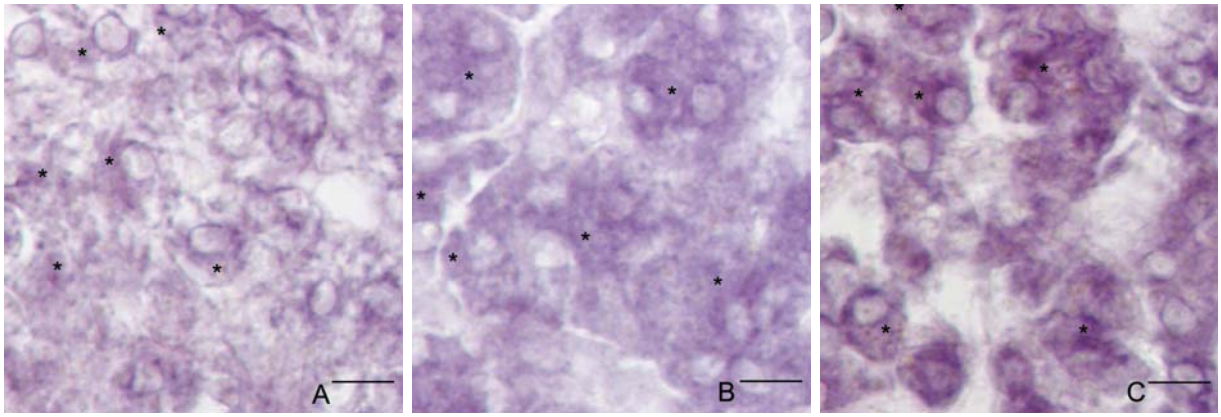

Figure 3. LDH activity in carp liver (*). Increased diffused LDH activity on the 70th day of the treatment with FeG (B) and FeD (C) can be seen in comparison to the control sample (A). Bar = 25µm

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