Fish kill caused by aluminium and iron contamination in a natural pond used for fish rearing: a case report

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ABSTRACT: Contamination of Pansky Pond, in March 2013, with 119 mg/l aluminium, and 87 mg/l iron by acidic (pH 3.17) inflow from a nearby quarry caused fish die-off, while exhibiting symptoms of suffocation. Transformation of soluble forms of aluminium and iron into insoluble forms occurred on fish gill where the content of aluminium and iron was 100-fold and 12-fold, respectively, that found in control fish in an unaffected pond. In addition to insoluble aluminium and iron, gills showed presence of iron bacteria. Histopathology was characterised by expression of reactive processes and regressive alterations resulting in gill tissue necrosis. Impairment of the excretory function of gills was reflected in significantly ($P < 0.01$) higher concentrations of ammonia in the blood plasma of exposed fish compared to the control. Damage to parenchymatous tissues (kidney, liver, spleen) of the exposed fish was manifested as dystrophic alterations, higher aluminium and iron content, and enhanced activity of transaminases in blood plasma compared to the control.

Keywords: aluminium; iron; water pH; iron bacteria; remobilisation; gill injury; plasma ammonium

Aluminium (Al) is the third most abundant element on the earth´s crust (Fernandez-Davila et al. 2012), occurring mostly as aluminium oxide and aluminium silicate (Scancar et al. 2004). Aluminium is commonly used in kitchen utensils, cans, food and drink packaging, dyes, baking powder and anti-acids, water purification processes (Fernandez-Davila 2012), cosmetics, medications, paper, and herbicides (Schmitz 2006; Callister 2007). The accidental release of aluminium-based coagulants from drinking water purification may represent a source of Al in water (Svobodova et al. 2008). Aluminium-containing preparations such as PAX, $Al_2(SO_4)_3$, and $NaAl(OH)_4$ are used for cyanobacteria elimination in reservoirs (Jancula et al. 2011). These formulations combine with phosphorus, an essential element for cyanobacteria, and the resulting compounds are deposited on the bottom of reservoirs (Wauer and Teien 2010). At pH less than 6, aluminium may also be leached from soil and sediments into the water, so-called remobilisation. Acidic rainwater and strongly acidic mine water are other sources of aluminium, since their low pH increases the migration of aluminium from soil and sediments (Cronan and Schofield 1979; Pitter 2009).

Studies have shown that Al can evoke oxidative stress through stimulation of reactive oxygen species (ROS) production in cells (Li et al. 2006; Sinha et al. 2007); lipid peroxidation (LPO) induction; disruption of activity of antioxidant enzymes including superoxide dismutase (SOD), catalase (CAT) and glutathione peroxidase (GPx); and facilitation of protein oxidation (Almroth et al. 2005; Parvez and Raisuddin 2005; Vlahogianni et al. 2007).

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In mammals, Al accumulation in the brain has been suggested to be involved in the development of neurodegenerative disorders, amyotrophic lateral sclerosis, and dialysis encephalopathy (Flora et al. 2003; Bondy 2010). Similar changes in brain have been found in rainbow trout exposed to Al (Exley et al. 1997).

In fishes, Al may be associated with gill damage due to its deposition and changes in osmoregulation, as well as with oxidative stress in lymphocytes (Galar-Martinez et al. 2010; Garcia-Medina et al. 2010). According to Stohs and Bagchi (1995) and Bondy and Cambell (2001) Al can cause direct damage to the mitochondrion and affect electron transport in the respiratory chain, increasing LPO and, subsequently, ROS production. Fernandez-Davila et al. (2012) described a time-dependent increase in SOD activity in grass carp after 48 h Al exposure. The most toxic form of Al to fish is inorganic Al, the content of which in water increases linearly with reduction of pH, as acidity facilitates release of aluminium from bottom sediments (Gensemer and Playle 1999). Garcia-Medina et al. (2011) have described time-dependent DNA damage in the lymphocytes of common carp after Al exposure even at low concentrations. In *Prochilodus lineatus*, 6 h and 24 h exposure to Al elicited increases in catalase and glutathione-S-transferase (GST) as well as DNA damage (Galindo et al. 2010).

The acute Al toxicity in the pH range 4.5 to 5.5 is believed to be based on interactions of cationic Al hydrolysis products with gill surface ligands (Neville and Campbell 1988; Playle and Wood 1989; Exley et al. 1991). Al accumulation, in addition to injury to the gill epithelium, causes apoptosis and necrosis of gill ion-transporting cells (Eeckhaoudt 1994), which is considered to be the main cause of ion-regulatory and osmoregulatory dysfunction (Witters et al. 1996). Fish death following Al exposure is also associated with an inflammatory response to Al-hydroxides with excessive mucous production and subsequent disruption of O_2 and CO_2 diffusion (Playle et al. 1989; Witters et al. 1991).

Iron is an essential element in many physiological processes. In biological systems, iron occurs in three oxidation states, II, III, and IV, and is largely bound to haemoglobin, transferrin, ferritin, and iron-containing enzymes, with free iron existing in trace amounts (Valko et al. 2005). Excessive uptake of iron, or disturbances in its regulation, causes cellular injury related to iron catalysis of ROS formation via the Fenton reaction (Orino et al. 2001). Iron has been shown to induce oxidative stress (Sevcikova et al. 2011). Li et al. (2009) observed lipid peroxidation (LPO) and alterations in antioxidant enzyme activity in embryonic and adult medaka *Oryzias latipes* exposed to nano-iron. After an iron-enriched diet, LPO was observed in the liver and heart of the African catfish *Clarias gariepinus* (Baker et al. 1997). High levels of LPO were found in erythrocytes of cichlid fish from a river containing high levels of iron (Ruas et al. 2008).

Iron is found in sediments in various forms. As with Al, it is released into water from sediments via remobilisation processes at pH less than 6 (Pitter 2009). Iron formulations are also used to eliminate cyanobacteria in eutrophic water (van Anholt et al. 2002; Velisek et al. 2014). The mechanism of action of these preparations is identical to Al, binding of phosphorus to form compounds that are deposited on bottom (Hayes et al. 1984).

Iron solubility in water depends on pH, oxidation-reducing potential, temperature, oxygen, and the presence of substances to which it will bind, such as OH^-, SO_4^{2-}, Cl^-, and humic substances. In oxygenated water, insoluble ferric (Fe^{3+}) iron dominates over the bivalent form, Fe^{2+}, which is toxic to aquatic animals (Davidson 1993). Fe^{2+} has the ability to bind to the alkaline gill surface and to oxidise to insoluble Fe^{3+}. These insoluble substances cover the gill fringes and induce epithelial damage and disruption of respiration (Teien et al. 2008).

Iron bacteria are found in water with high Fe content. Iron bacteria such as *Acidithiobacillus* spp. and *Leptospirillum* spp. derive energy and multiply by oxidizing dissolved ferrous iron, or the less frequently available manganese, to produce insoluble ferric oxide or manganese dioxide. Optimal conditions for growth of iron bacteria are low pH, low water temperature, and high iron concentration. Iron bacteria create colonies on gill surfaces and exacerbate gill damage. Iron is absorbed into the organism and subsequently causes tissue damage due to ROS production and lipoperoxidation (Baker et al. 1997; Lappivaara et al. 1999).

Case description

The Pansky and Klucenicky Ponds are small bodies of water (0.4 and 0.3 ha, respectively) located in the Central Bohemia Region of the Czech Republic (Figure 1). A quarry, located approximately 2 km

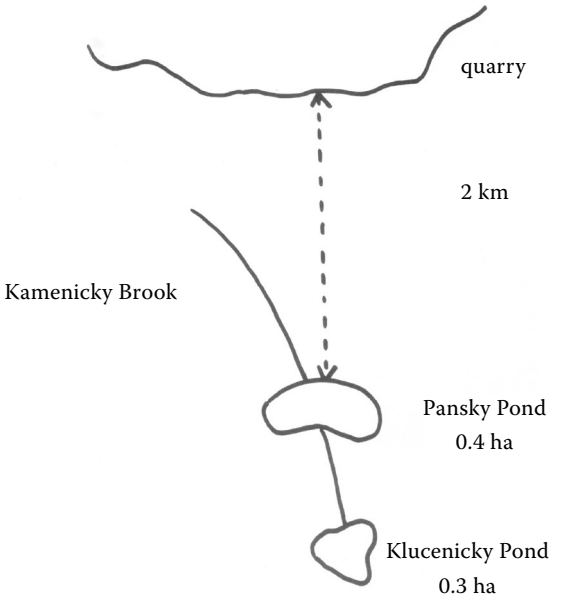

Figure 1. Map of affected locality

upstream of these ponds previously drained via a spillway onto adjacent grasslands. After construction of a drainage system, the quarry water was redirected to drain directly into Pansky Pond and from there downstream to Klucenicky Pond. Approximately 14 days after quarry outflow was redirected into Pansky Pond (20 March, 2013), changes in fish behaviour and fish die-off were observed. Fish kill continued, with the highest number of dead fish recorded on 24 March. At the time of the fish kill, water in both ponds was turbid and blue-green in colour, and surviving fish in both ponds were gasping. They remained close to the water surface and showed no escape response. Gills were coated with a brown deposit. The pH of the water in both ponds was 5.8. Oxygen saturation in Pansky Pond was 65% near the surface and the water temperature was 5.6 °C.

On 24 March, samples were taken from the quarry and Pansky Pond water, bottom sediment of both ponds, and fish.

Fish examination

Clinical signs. On 24 March, fish were dark-coloured. They stayed near the pond bank in normal orientation, exhibited rapid respiration, did not react to external stimuli, and did not show escape responses.

Post-mortem findings. Twelve common carp of body weight 1800–2350 g from Pansky pond were examined, along with three roach from the same pond of body weight 212–272 g. Nutritional status of the examined fish was good. In all specimens skin was dark with excessive mucus production. Four carp exhibited haemorrhage on the ventral surface, and swelling of the anus was observed in six fish. Gills of all species were swollen, haemorrhaging after mechanical injury, and coated with a brown deposit. In the body cavity, blood vessels of internal organs were dilated. The intestines were empty.

Parasitic examination. No indication of parasite infection was found in skin, gill, blood, or gut of any specimen.

Histological, histochemical, and bacteriological examination of gills. The most extensive anomalies were found in gills. Histopathology was characterised by expression of reactive processes and extensive regressive alterations resulting in gill tissue necrosis. Reactive processes were based on activation of large eosinophilic cells with granulated cytoplasm in large clusters in the respiratory epithelium, mainly at the base of gill filaments. Cellular inflammatory infiltration was visible in the gill arch epithelium. Regressive alterations affected the respiratory epithelium of filaments with differing degrees of dystrophic changes including exces-

Table 1. Analysis of blood plasma of common carp from Pansky Pond and from the control Novy Pond

Index	Pansky Pond (n = 6)	Novy Pond (n = 6)
	mean ± SD	
Weight (g)	2015 ± 235	2320 ± 381
N-ammonia** (μmol/l)	265 ± 23.3	183 ± 16.6
AST (IU/l)	29.0 ± 13.58	13.9 ± 2.23
ALT (IU/l)	3.43 ± 0.12	2.63 ± 0.59
CK** (IU/l)	4 483 ± 582.2	2 083 ± 424.6
BCHE (IU/l)	24.3 ± 1.33	30.7 ± 4.96

**statistical significance $P < 0.01$

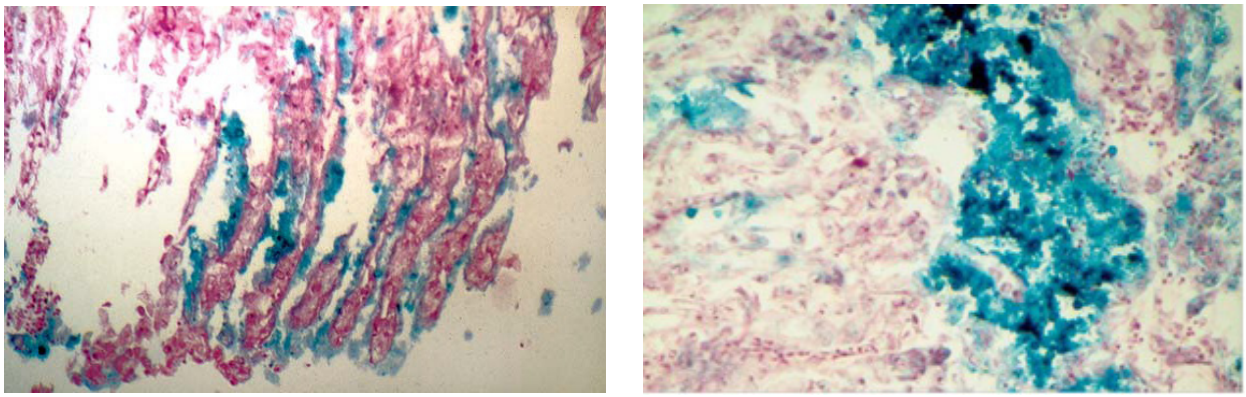

Figure 2. Evidence of iron in fish gill according to Pearl's method and obivous epithelonecrosis. **A** – scattered iron deposits; original magnification, 100×; **B** – deposits of iron on gills, original magnification, 400× (photo L. Groch)

sive vacuolisation, swelling, and disorganisation of cells of the lamellar epithelium and filling the space between filaments with detached and degenerated epithelia. These regressive processes resulted in necrosis of gill tissue that progressed from the distal regions of the lamellae to their base. Spaces between gill filaments and lamellae were filled with eosinophilic necrotic tissue, mucus, and deposits of ochre-brown pigment. Histochemical analysis for iron in this pigment was positive (Figure 2).

Gill sections stained with Giemsa showed fine rod-like bacteria in the necrotic masses, organised into clusters or concentrated as isolated formations around conglomerates of iron pigment grains. Sections tested positive for the presence of iron bacteria, and a high presence of iron bacteria was shown microscopically and in culture.

Examination of other organs showed dystrophic changes to the epithelium of proximal kidney tubules and of liver parenchyma, with excessive pycnosis of hepatocyte nuclei. Other organs did not exhibit pathological abnormalities.

Common carp blood plasma. Results of analysis of blood plasma of common carp are given in Table l. Control fish were captured from Novy Pond in Vodnany, where no fish kills or atypical fish behaviour were observed during winter and spring. Water quality parameters in this pond at the time of fish capture were temperature 6.2 °C, pH 7.1, and oxygen saturation 80%.

Results of analysis of blood plasma of the carp from Pansky Pond and the controls from Novy Pond showed comparable levels of aminotransferase (ALT) and butyrylcholinesterase (BCHE). The concentration of N-ammonia and creatine kinase (CK) in the blood plasma of common carp from the Pansky Pond were significantly higher ($P < 0.01$), while the activity of aminotransferase (AST) in carp from the Pansky Pond was higher than in controls, but the difference was not significant.

Chemical and toxicological analysis of fish tissues

Assessment of iron and aluminium content in tissue of common carp was conducted by atomic absorption spectrometry (AAS) after preceding mineralisation. Results of analysis of individual tissue samples of six common carp from Pansky Pond and of six carp from Novy Pond are shown in Tables 2 and 3.

A high level of iron was found in the gills of common carp from Pansky Pond, with mean iron content 12-fold that observed in control carp ($P < 0.01$). Higher levels of iron in exposed common carp were also found in spleen and kidney (5-fold and 4-fold, respectively) when compared to the control specimens (Table 2).

Mean aluminium content in the gills of common carp from Pansky Pond was nearly 100-fold that found in control common carp specimens ($P < 0.01$). Higher aluminium levels in exposed carp were also observed in kidney, liver, muscle and spleen (10-fold, 3-fold, 1.7-fold and 1.6-fold, respectively) when compared to the control specimens (Table 2).

Analysis of water

Physical and chemical examination of water. Water in both samples (centre and outlet of Pansky Pond) showed decreased pH, increased $BNC_{8.3}$, and

Table 2. Iron and aluminium content in tissue of common carp from Pansky Pond and the control Novy Pond

	Iron content				Aluminium content			
	Pansky Pond (n = 6)		Novy Pond (n = 6)		Pansky Pond (n = 6)		Novy Pond (n = 6)	
	Fe mean ± SD (mg/kg)							
	fresh tissue	dry matter	fresh tissue	dry matter	fresh tissue	dry matter	fresh tissue	dry matter
Muscle	4.1 ± 1.55	20.8 ± 7.17	8.5 ± 4.53	41.2 ± 22.18	4.6 ± 1.70	23.5 ± 8.55	2.7 ± 1.50	13.3 ± 7.85
Gill**	669 ± 250.4	3 829 ± 575.2	58 ± 13.7	328 ± 80.2	1 078 ± 218	7 232 ± 1 468	14 ± 4.9	78 ± 30.2
Liver[1]	36.5	190.5	39.4	155.0	11.6	62.1	5.5	21.5
Kidney[1]	32.1	190.0	13.9	47.6	21.3	126.0	2.9	9.8
Spleen[1]	213.0	1 184.0	44.7	252.0	9.8	54.3	6.2	34.7

**statistical significance $P < 0.01$

[1]analysis of pooled sample of six specimens

increased concentration of ammonia, nitrites, iron, and aluminium compared to typical values found in ponds in this period of the year (Table 3).

Analysis of water in the quarry revealed aluminium levels of 119 mg/l, iron levels of 87 mg/l and pH of 3.17.

Biological assay of water toxicity. The assay was performed using the cladocerans *Daphnia magna* with both water samples (centre and outlet of Pansky Pond). Ten *Daphnia* were added to each sample and the test was performed in duplicate. No mortality or immobilisation was observed during a 48 h exposure. A biological assay was conducted in Pansky Pond water to which the brown substance obtained by wiping the gills of common carp was added. Total mortality of *Daphnia magna* was seen within 24 h.

Analysis of bottom sediment

Samples of bottom sediment were taken from five sites in Pansky Pond and from five sites in Klucenicky Pond. Immediately after sampling, the superficial layer (2 cm) and bottom layer (15 cm) were separated. From these samples, four mixed samples were prepared (e.g. superficial layer and bottom layer from Pansky Pond and superficial layer and bottom layer from Klucenicky Pond).

Content of aluminium and iron in the sediment of ponds was comparable, and similar values for the elements were seen in the upper and bottom sediment layers. These values were typical of concentrations occurring in pond sediments (Svobodova et al. 1996) (Table 4).

Table 3. Chemical composition of water from Pansky Pond

Sampling site	At outlet	In pond centre
pH	5.80	5.90
$ANC_{4.5}$ (mmol/l)	0.50	0.40
$BNC_{8.3}$ (mmol/l)	0.20	0.15
BOD_5 (mg/l O_2)	1.10	0.50
COD_{Mn} (mg/l O_2)	3.20	4.70
N-(NH_4^++NH_3) (mg/l)	0.61	0.56
N-NO_2^- (mg/l)	0.146	0.161
Fe^{2+} (mg/l)	0.80	0.65
Al (mg/l)	0.56	–

DISCUSSION AND CONCLUSIONS

The quarry waters that caused fish poisoning in Pansky and Klucenicky Ponds exhibited extremely low pH values and high concentrations of iron and aluminium and negatively affected the quality of pond water. pH values of water is less than or equal six were reported to initiate remobilisation processes, during which soluble forms of iron and aluminium were released from bottom sediment into the water (Pitter 2009).

Iron is an essential compound for physiological processes in animals and humans; however, at greater than optimal concentrations it is injurious to living organisms (Davies 1991; Misra and Mani 1992).

The iron-containing compounds in water can release free cations (Fe^{2+}) that react with other anions. In the presence of oxygen, Fe^{2+} can react with the hydroxyl radical (OH^-) of water to precipitate ferric hydroxide (Debnath et al. 2012). At neutral to slightly acidic pH, iron oxidises with the aid of dissolved oxygen to the ferric form (Heath 1995). In water, the dissolved inorganic $Fe(OH)_2$ is the most frequently occurring form of iron (Dave 1984). Although ferric iron does not present a hazard to fish, some soluble

Table 4. Chemical analysis of bottom sediment

Analysis	Pansky Pond		Klucenicky Pond	
	upper layer	bottom layer	upper layer	bottom layer
Dry matter (%)	33.3	46.5	35.6	48.7
Al in dry matter (mg/kg)	79 024	78 685	80 465	78 842
Fe in dry matter (mg/kg)	36 392	33 530	39 659	38 446

iron forms are highly toxic (Sykora et al. 1972). At certain pH levels, ferric hydroxide can greatly affect populations of benthic organisms and of fish in streams (Koryak et al. 1972). Impaired feeding of fry and juvenile stages, with subsequent prolonged stress and reduced growth was observed at concentrations of iron is greater than 1.0 mg/l (Smith et al. 1973). In the short-term partial life cycle test, Debnath et al. (2012) recorded reduced feeding rate, behavioural changes and growth reduction of catla *Catla catla*, rohu *Labeo rohita*, and mrigal *Chirrhinus mrigala* larvae after ferrous sulphate exposure. These consequences might be due to the accumulation of iron in the body of larvae, especially in gills, thus altering respiration and osmoregulation (Debnath et al. 2012).

The negative effect of aluminium on fish has been described (Lydersen et al. 1990; Gensemer and Playle 1999; McCartney et al. 2003; Monette and McCormick 2008). Aluminium toxicity depends on water pH, and on the concentration of calcium and organic substances (Freda 1991; Wauer and Teien 2010). Solubility of aluminium increases linearly with decreasing pH. Lloyd (1992) reported the most toxic forms of aluminium to occur at pH ranging from 5.2 to 5.8.

Teien et al. (2008) reported that soluble compounds of iron and aluminium bind to alkaline-reacting gill where they oxidise to insoluble compounds. These compounds cover gill fringes, damaging gill epithelium. This phenomenon was observed in fish from Pansky Pond, the gills of which showed high concentrations of aluminium and iron, along with iron bacteria. Histopathology of gills was characterised by reactive processes and regressive alterations producing tissue necrosis.

Lukowicz (1976) reported that iron bacteria take part in oxidation of iron and form a coating on gills, limiting fish respiratory capacity. Iron compound deposits on gills and damage to gills have been described (Larson and Olsen 1950; Kinne and Rosenthal 1967; Brenner et al. 1976; Dalzell and MacFarlane 1999). Other authors (McDonald 1983; Wood 1989; Peuranen et al. 1994) have reported iron deposits only on the surface of gills, without gill epithelium injury, especially at low pH values.

The gill has three main functions: respiration, osmoregulation, and elimination of ammonium. Gill damage leads to a disruption of oxygen and carbon dioxide exchange, hypoxia, hypercapnia, and plasmatic acidosis (Playle and Wood 1989; Exley et al. 1991). Aluminium causes disruption of ion-regulation and osmotic regulation (Andren and Rydin 2012). The observed behaviour of fish in Pansky Pond was consistent with such a scenario. Fish stayed in littoral areas, lost the escape reflex, and exhibited gasping. Due to gill damage, ammonia excretion was impaired, reflected in significantly increased ammonia concentrations in blood plasma.

Iron and aluminium was also found to accumulate in the parenchymatous organs (liver, kidney, and spleen). Strbac et al. (2013) monitored the content of Al and Fe in the liver of common carp and other fish species from several sites in the Tisza River. They found the content of aluminium in the liver of common carp to be 5.26–45.56 µg/g wet weight and that of iron to be 76–193.66 µg/g wet weight depending on locality. These findings supported the hypothesis of Rincon-Leon et al. (1993) and Yousafzai et al. (2010) that omnivore and benthivore fishes accumulate greater amounts of metals than carnivorous fishes. Azmat et al. (2012) showed increased amounts of Al in the gills, kidney, and liver of fish after aluminium poisoning.

Histopathological changes were found in the liver and kidney. Injury to parenchymatous tissue, especially liver, was reflected in increased plasma enzyme activity (ALT, AST). An increase in the activity of CK in blood plasma was also found, as a result of muscle injury and the high stress to which the fish were exposed, including hypoxia, plasmatic acidosis, increased muscle activity with gasping, and impaired excretory function of gills (Playle and Wood 1989; Exley et al. 1991).

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