

## Indicators of chronic metal stress in wild yellow perch from metal-contaminated environments

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### Abstract

Recent research examining the effects of metals on fish has focused almost exclusively on typical lab species, like rainbow trout (*Oncorhynchus mykiss*) or fathead minnows (*Pimephales promelas*) under tightly-controlled laboratory conditions. Although much valuable information has resulted from this work, particularly with regard to understanding mechanisms of toxic action and the development of predictive toxicological models, questions remain about ecological relevance. During a recent survey of twelve Sudbury-area lakes varying in their degree of metal contamination (from relatively pristine to highly metal contaminated), 21 fish species were identified (Rajotte et al., in prep.). Fathead minnows were found in three lakes and rainbow trout were not found in any of the lakes studied. However, yellow perch (*Perca flavescens*) were found in all twelve lakes, including those most metal contaminated. This is perhaps not surprising given that yellow perch are nearly ubiquitous in northern Canadian lakes, and several times more tolerant to metal exposure than more traditionally-studied fish species. Furthermore, lab-based studies do not adequately account for the inherent variability of natural systems. Research recently conducted by our lab has focused on industrial metal effects on wild yellow perch from lakes forming a metal contamination gradient, and evaluates this species as an environmental sentinel in industrially-contaminated aquatic systems.

### **Factors Affecting Tissue Metal Bioaccumulation**

A first line of evidence for assessing potential metal effects on fish health is to examine whether the metals of concern accumulate in target tissues. The mere presence of a given metal at high concentrations in water or sediment does not necessarily involve a direct toxicological risk to fish, especially in the absence of significant tissue metal bioaccumulation. Several biotic and abiotic factors are known to mediate metal bioaccumulation, and a comprehensive review would be beyond the scope of this paper. Most biotic factors that influence metal bioaccumulation are associated with the diet and include metal concentration in prey species and feeding rate. Water quality variables, including pH, alkalinity, and hardness, are dominant abiotic factors that influence metal uptake and subsequent bioaccumulation. Therefore, bioaccumulation

is to a large extent mediated by biotic factors that influence dietary metal uptake, and by abiotic factors that influence aqueous metal uptake. Temperature may also be an important abiotic factor influencing metal uptake because of its relationship with metabolic rate, which drives metal handling (i.e., excretion and detoxification) and feeding rates.

Studies that examine metal accumulation in wild yellow perch are mostly restricted to lakes in the vicinity of Rouyn-Noranda, Quebec, and of Sudbury, Ontario. Growing evidence from this research suggests that tissue metal concentrations largely reflect environmental contamination<sup>1-7</sup> especially for cadmium (Cd), a non-essential metal for which there is no known mechanism for physiological regulation. Copper (Cu) is often elevated in tissues of metal-contaminated yellow perch compared to fish from reference lakes. This metal is known to be an essential

nutrient in fish and other biota. However, during periods of low feeding activity (i.e., relatively low dietary metal uptake), Cu appears to be maintained within the range of 10-50  $\mu\text{g}\cdot\text{g dry wt}^{-1}$  in liver of fish from contaminated lakes, which is the range of hepatic Cu concentrations that is measured in fish from reference or intermediate lakes (Couture and Rajotte, unpublished results). That hepatic Cu concentrations are similar in fish from metal-contaminated and reference lakes during periods of low intake reflects Cu's essentiality and homeostatic regulation. Environmental nickel (Ni) contamination, like Cu, is often but not always reflected in yellow perch tissues<sup>1,7</sup>. Zinc (Zn) also behaves like Cu and Ni, exhibiting elevated tissue concentrations only during some periods of the year in the tissues of metal-polluted fish<sup>1-3</sup>. Like Cu, Zn is an essential metal that is under homeostatic control even though it is maintained at much higher concentrations than Cu. These data reveal that elevated environmental metal contamination is most likely to be reflected in fish tissues for non-essential metals like Cd, whereas essential metals like Cu, Ni, and Zn are under homeostatic control and, therefore, tissue concentrations only reflect environmental metal contamination in periods of high metal intake.

Liver metal concentrations of essential metals have been shown to vary seasonally in wild yellow perch. Previous studies in our lab have demonstrated that there are important seasonal variations in tissue metal concentrations in yellow perch from metal-contaminated environments, but not in reference lakes, and that differences between reference and polluted fish are not obvious throughout the year<sup>1,3</sup>. Therefore, it is critical that tissue metal concentrations are monitored seasonally in order to conclude whether or not fish from polluted environments express elevated tissue metal concentrations, with the

associated potential toxic risk. At present, we do not know whether there is a relationship between the metabolic costs of metal regulation or detoxification and fish condition. Therefore, there is no evidence to suggest that chronic metal exposure alone, when it is not associated with elevated tissue metal concentrations, can negatively affect wild fish condition. Studies that exploit natural metal contamination gradients, where fish are collected from several lakes ranging in their degree of metal contamination, including several reference lakes to provide a firm understanding of reference-level variability, are required to examine this relationship.

### **Influence of metal contamination on fish growth and condition**

A second line of evidence that should be examined in metal-contaminated fish is their condition, using simple age, weight, and length measurements from which growth rates and condition indicators can be calculated. These measurements can also provide estimates of recruitment and longevity if sampling is carried out in such a way as to ensure that the catch is representative of the size distribution of the target population and if sample size is sufficient.

In the Sudbury area, as in Rouyn-Noranda, chronic metal exposure is related to lower growth rates and condition indicators in wild yellow perch<sup>1-7</sup>. Over the last five years, we have also consistently observed a lower longevity in yellow perch from metal-contaminated lakes (unpublished results). Longevity in fish from the most polluted lakes (Whitson, Hannah and Kelly) seldom exceeded 4 years and only reached 5 years once in Whitson Lake and 7 once in Kelly Lake (unpublished results). In contrast, fish up to 11 years old were recorded in a range of clean lakes whether or not yellow perch co-

existed with other species. There are several possibilities to explain this lower longevity in yellow perch inhabiting metal-polluted lakes, that can be divided in two categories: direct toxic effects on metabolic processes, and indirect effects that reduce food availability through effects on the food web. It is possible that yellow perch from polluted lakes may have access to limited dietary resources, which inhibits normal ontogenetic diet shifting, as suggested by Kövecses and co-workers<sup>8</sup>. Young yellow perch are planktivorous, but when they reach a certain size they normally switch to a benthivorous diet, and later to piscivory. There could be direct toxic effects of sediment contaminants on benthic invertebrates, and their decreased abundance could indirectly decrease the longevity of yellow perch that would find it more difficult to make the switch to benthivory. In addition, such benthic invertebrates could contain elevated levels of metals that may cause direct toxic effects in fish or increased costs for excretion and detoxification. Likely, the cause of decreased longevity in polluted perch is a combination of several of these factors. The absence of smaller fish species does not appear to be an impediment for growth in yellow perch, since they can be cannibalistic and grow to a large size even in a lake where they are the only species to occur (Birch Lake<sup>7</sup>).

The same factors affecting longevity probably influence the condition of yellow perch, as reflected in growth rates and condition indicators. Growth rate was estimated in a number of studies using length-at-age relationships and consistently indicated that fish from the most polluted lakes demonstrated reduced growth relative to fish from reference lakes<sup>1,2</sup>. Similarly, condition factors derived from length and weight measurements (Fulton's Condition Factor (FCF) and relative condition factor (Kn)) have also often indicated lower condition in

polluted fish relative to reference fish<sup>1-6</sup>. However, in both clean and polluted lakes, condition factors have shown important seasonal variations that could confound the interpretation of ecotoxicological data if they are ignored<sup>1,3</sup>. Furthermore, while tissue metal concentrations do not vary seasonally in fish from clean lakes, their variations in polluted fish do not necessarily correspond with seasonal variations in condition. Because water quality parameters do not vary substantially by season<sup>3</sup>, our data suggest that diet plays a major role in tissue metal concentrations, and that the seasonal variations in feeding rate induce the variations in tissue metal concentrations. Overall, condition factors (FCF or Kn) and scaling coefficients have shown good potential as condition indicators for metal-polluted yellow perch when combined with tissue metal concentrations, if seasonality is taken into account.

### **Influence of metal contamination on metabolic capacities of fish tissue**

A third line of evidence for assessing fish health in metal contaminated systems involves the measurement of metabolic capacities in relation to tissue and environmental metal concentration. Metabolic impairment, when combined with decreased condition in fish from metal-contaminated lakes, provides convincing evidence for sublethal effects of chronic metal exposure. Our data demonstrate that yellow perch aerobic capacities are negatively related to environmental metal contamination.

Critical swimming speed (Ucrit) provides a reproducible measure of a fish's aerobic performance<sup>10,11</sup> even though yellow perch rarely swim at their Ucrit in nature. Recent studies in our lab have shown that Ucrit is negatively associated with environmental metal contamination in yellow perch<sup>2</sup>.

Moreover, there is a direct positive relationship between oxygen consumption both at rest and after exhaustive exercise and hepatic Cu and Cd concentrations, respectively<sup>9</sup>. These studies support our hypothesis that increased metal contamination reduces aerobic capacities, leading to a reduction in aerobic swim performance.

Several studies on Sudbury-area yellow perch have reported negative relationships between aerobic capacities in white muscle, as determined by citrate synthase (CS) activities, and environmental metal contamination<sup>2,3,9</sup>. Citrate synthase is a regulatory enzyme involved in tissue ATP production via the Krebs's Cycle. Yellow perch from metal contaminated lakes examined in the summer months have lower CS activities than in spring or fall, which correlates with higher metal concentrations in stomach contents and liver<sup>3,9</sup>. In white muscle, the activity of enzymes involved in lipid breakdown<sup>2</sup> and electron transport (unpublished results) are also affected by chronic exposure to metals in the field. Interestingly, although liver is a main target for metal accumulation, the impairment of aerobic capacities mostly occurs in the white muscle<sup>2,3</sup>. In contrast to aerobic capacities, tissue anaerobic capacities, determined from lactate dehydrogenase (LDH) activities, are not impaired by metal contamination<sup>2,3,6,9</sup>. Some studies indeed suggest that an impairment of aerobic capacities may be to some extent compensated by increased anaerobic capacities in the white muscle of metal-contaminated wild yellow perch<sup>2,9</sup>. Overall, these studies support the hypothesis of a toxicological consequence of chronic, sublethal metal exposure that affects aerobic metabolism in wild yellow perch. Current research in our laboratory is investigating the extent of impairment in each of the major metabolic pathways in white muscle that could be affected by metal contamination. We are also trying to determine the extent to

which mitochondria are targeted by metals and thus the source of the reductions in aerobic capacities, as has been shown in many laboratory studies<sup>12-15</sup>.

## Conclusions

Yellow perch are abundant in both clean and metal-contaminated lakes around industrial regions of northern Canada, and are a good choice for assessing the ecotoxicological consequences of industrial metal pollution. Recent investigations in Sudbury and Rouyn-Noranda have begun probing how this species responds to elevated metals in its environment in terms of metal bioaccumulation, fish condition, and metabolic capacities. By combining these elements in ecotoxicological investigations and exploiting metal contamination gradients, an ecologically-relevant picture begins to emerge that demonstrates the multi-faceted consequences of chronic metal exposure in wild yellow perch populations. This new knowledge will ultimately be utilized to improve ecological risk assessments.

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