OSMOTIC AND IONIC REGULATION IN JUVENILE FISH

EXPOSED TO CYANOBACTERIAL TOXINS

F B Eddy
Department of Biological Sciences
University of Dundee, Dundee, DD1 4HN, Scotland, UK.
f.b.eddy@dundee.ac.uk

J H Best and G A Codd
Department of Biological Sciences
University of Dundee, Dundee, DD1 4HN, Scotland, UK

EXTENDED ABSTRACT ONLY – DO NOT CITE

Juvenile salmonids inhabit a hypoosmotic environment and continuously lose salts by diffusion and gain water by osmosis. Even in recently hatched yolk sac fry (or alevins), achievement of hydromineral balance is by absorption of inorganic ions from the medium, possibly via mitochondrion rich cells on the body surface while excess water is excreted via the kidney. Alevins and fry drink freshwater, and although drinking has no obvious physiological function, it represents a route for water uptake, as well as for uptake of substances from the environment. Toxic substances in the water may enter directly via the body surface if they are sufficiently lipid-soluble, e.g. organic substances and unionised ammonia. Some toxins may inhibit ionic absorption processes in ion absorbing cells on the body surface, e.g. zinc, cadmium and the ammonium ion. Drinking represents another means of toxin presentation, with absorption via the gut mucosa, though little is known of this route, or its significance.

Cyanobacteria (blue green algae) produce a variety of toxins in fresh water, including neurotoxins and endotoxins and one group of cyclic peptides, the microcystins which are hepatotoxins, has been extensively studied in mammals. Following ingestion, symptoms include vomiting and diarrhoea followed by damage to liver parenchyma cells, which may be fatal. There have been fewer studies in fish and long term-exposure of brown trout to microcystin resulted in reduced growth rate and liver damage (Bury *et al* 1995). Microcystins are not sufficiently lipid soluble to enter via the body surface, but may enter via drinking and transport mechanisms in the gut (Bury *et al* 1997). Fish-kills sometimes occur during blooms of cyanobacteria though it is difficult to attribute mortality to a single cause. Mortality could result from deterioration of water quality often characterised by lowered oxygen levels, increased ammonia and pH levels, and toxins which may be released during senescence of the bloom.

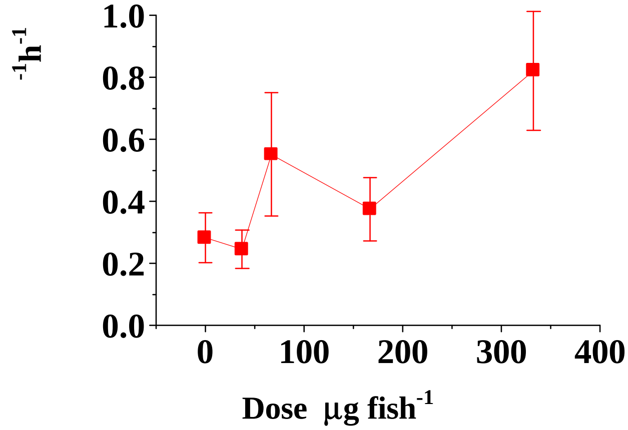

Fig 1. Effects of *Escherichia coli* LPS on drinking rate in juvenile rainbow trout

A variety of other substances are present during blooms and there is recent interest in lipopolysaccharides (LPS) produced by the cyanobacteria and bacteria present in blooms. Our studies aimed to investigate the effects of LPS on the osmoregulatory physiology of juvenile salmonids, with particular regard to the role of drinking. Exposure of juvenile rainbow trout to LPS of *E.coli* origin by injection, up to 300µg per fish, resulted in significantly increased drinking although the effects of crude extracts of *Anabaena* bloom produced a less marked effect. It is considered that LPS may contribute significantly to fish kills. They may enter via the body surface on account of their lipid-solubility and then, possibly via a cytokine system, stimulate production of nitric oxide (NO) through stimulation of inducible nitric oxide synthase (iNOS). Increased levels of NO result in vasodilation, with subsequent stimulation of the renin-angiotensin system (RAS) and production of angiotensin II (AII), which stimulated drinking in both fresh water and marine fish (Fuentes and Eddy 1997). Thus cyanobacterial blooms may increase the drinking rate of fish so allowing increased uptake of toxin via the gut which could be a contributory factor in mortalities. Physiological and environmental aspects of these events are explored.

References

Bury, N.R., F.B. Eddy and G.A. Codd 1995. The effects of the cyanobacterium *Microcystis aeruginosa* and the cyanobacterial toxin microcystin-LR and ammonia on growth rate and ionic regulation of brown trout Journal of Fish Biology, 46:1042-1054.

Bury, N.R., J.C. McGeer, F.B. Eddy and G.A. Codd 1997. Liver damage in brown trout *Salmo trutta* L., and rainbow trout *Oncorhynchus mykiss* (Walbaum), following administration of the cyanobacterial hepatotoxin microcystin-LR via the dorsal aorta. Journal of Fish Diseases 20: 209-215.

Fuentes,J. and F.B. Eddy 1997. Effect of manipulation of the renin-angiotensin system in control of drinking in juvenile Atlantic salmon (*Salmo salar* L) in fresh water and after transfer to sea water Journal of Comparative Physiology. 167: 438-443.