A case of ceroid deposition in the kidney of feral rainbow trout, Oncorhynchus mykiss, in Alicura impoundment, Argentina

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NOTE

A case of ceroid deposition in the kidney of feral rainbow trout, *Oncorhynchus mykiss*, in Alicura impoundment, Argentina

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Abstract

A severe case of ceroid deposition was observed in the kidney of a small number of feral *Oncorhynchus mykiss* from Alicura impoundment, Argentinian Patagonia. The pigments were observed intracellularly in the kidney tubules and in the parenchyma cells, varying from a large number of small droplets to one big droplet almost filling the cell and pushing the cytoplasm to one side of the cell. An extensive study of farmed fish, as well as wild specimens from the same impoudment, did not show any ceroid deposition. Further, no abnormal mortality rates were observed in the farms. The mechanism that caused this condition in just a small number of fish is not known.

Ceroidosis is a nutritional fish disease which involves the deposition of ceroid, a brown-yellow, acid fast, lipid positive pigment, resistant to organic solvents (Smith, 1979) in the liver, kidney or spleen, which has been observed in several farmed fish species like *Salmo salar*, *Oncorhynchus mykiss*, *O. kisutch*, *Ictalurus punctatus*, *Pomacentrus pavo* and *Amphiprion ocellaris* (Smith, 1979; Roald *et al.*, 1981; Blazer and Wolke, 1983; Holliman and Southgate, 1986; Saraiva *et al.*, 1986; Atsuta *et al.*, 1991; Pearson and Chinabut, 1993; Weisman and Miller, 2006).

The ceroid deposition is more frequently observed in the liver, causing the so-called Lipoid Liver Degeneration (LLD), and it is related principally to the ingestion of food with excessive oxidation of lipids and low level of antioxidants, namely vitamins C and E (Smith, 1979). LLD can be prevented by adding the adequate levels of antioxidants to the food (Smith, 1979).

Sometimes this condition was related, alone or in combination with other factors, as a cause of mortality of farmed fish (Holliman and Southgate, 1986; Saraiva *et al.*, 1986; Pearson

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and Chinabut, 1993; Atsuta *et al.* 1999) with a significant economic impact. Therefore, LLD constitutes a factor which may lead to fish losses and the prevention of its occurrence is important to fish farmers (Smith, 1979; Hardy, 2001).

The present article describes a case of ceroid deposition in the kidney of feral *O. mykiss* from Alicura impoundment.

A total of 1200 *O. mykiss* were sampled over a two year period from Alicura impoundment, built in river Limay, Argentinian Patagonia, in order to verify the eventual occurrence of notifiable salmonid diseases according to the OIE. Samples were taken every season and consisted of 75 fish from netpens of eight farms (8-10 fish per farm) and of 75 free living fish caught by angling around netpens resulting in 600 fish from farms and 600 free living fish. The mean total length of the farmed fish was 34.9 ± 2.3 cm, and of fish caught around netpens was 29.7 ± 1.7 cm. Fish were anaesthetised with benzocaine.

All the specimens were necropsied and samples of liver, anterior and posterior kidney, brain, heart, integument and muscle were taken for histopathological and molecular studies. Histological sections were stained with Haematoxylin and Eosin, Mallory Trichromic stain, and, when appropriate, periodic acid-Schiff (PAS) and Ziehl-Nielsen stain. All the specimens were inspected by OIE standard methods for the presence of BKD (Bacterial Kidney Disease), VHS (Viral Haemorrhagic Septicaemia), IHN (Infectious Haematopoietic Necrosis), IPN (Infectious Pancreatic Necrosis), ISA (Infectious Salmon Anaemia) and *Piscirickettsia salmonis*.

Fish in all the netpens were fed with pelleted food of the same quality and preserved under the same storing conditions.

Twelve out of the 600 examined fish, caught around the netpens, showed a slightly hypertrophied kidney with zones of discolouration (Figure 1). Histological examination of the kidney showed an intracellular deposition of compact granular formations. They were present in the tubular cells or in the parenchyma cells. In the first case their size was larger, measuring about 8 – 14 μm in diameter, sometimes occupying most of the cell, and the cytoplasm was somewhat pushed to one side of the cell (Figure 2). Some cells contained just one big droplet, while others had several smaller ones. In the parenchyma cells the granules were much smaller, having a diameter of about 1 and 3 μm, and almost filled the cells (Figure 3), which were somewhat distorted. Abundant macrophages were observed, which were intensely red coloured due to the colour of the droplets in Mallory staining, sometimes forming compact amounts. Further, in all the cases the droplets stained with Ziehl-Nielsen stain and were PAS positive thus all staining properties being consistent with ceroid. Some specimens showed a moderate degree of splenomegaly, but ceroid deposition was never observed in the spleen or in the liver. Fish showing alterations were captured in all the seasons of both years.

The twelve affected specimens were the only feral fish captured, i.e. fish that had escaped from the farm and lived free in the environment, and no other feral specimens were captured. This conclusion was based on the morphology of the specimens which

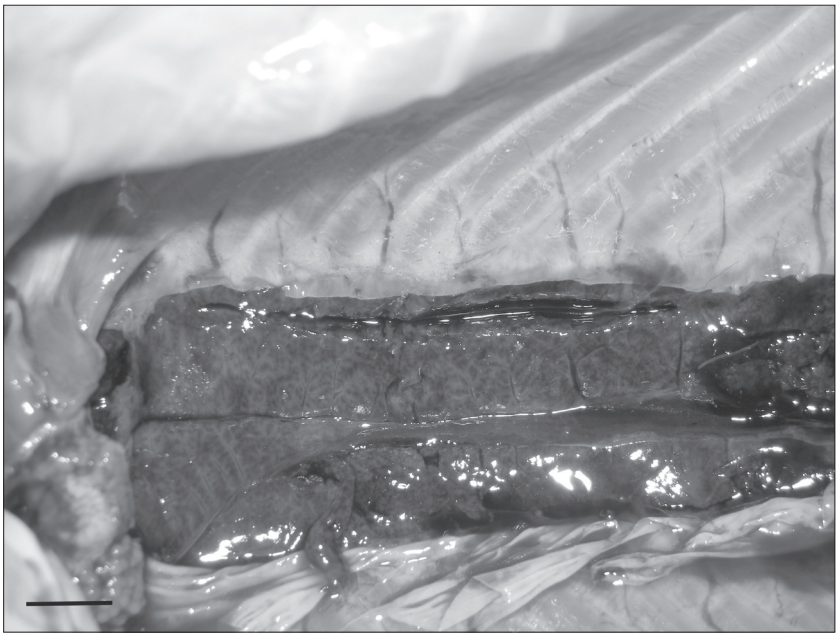

Figure 1. Anterior part of the kidney of affected fish. The organ is hypertrophied and presents small zones of discolouration. Bar = 1 cm.

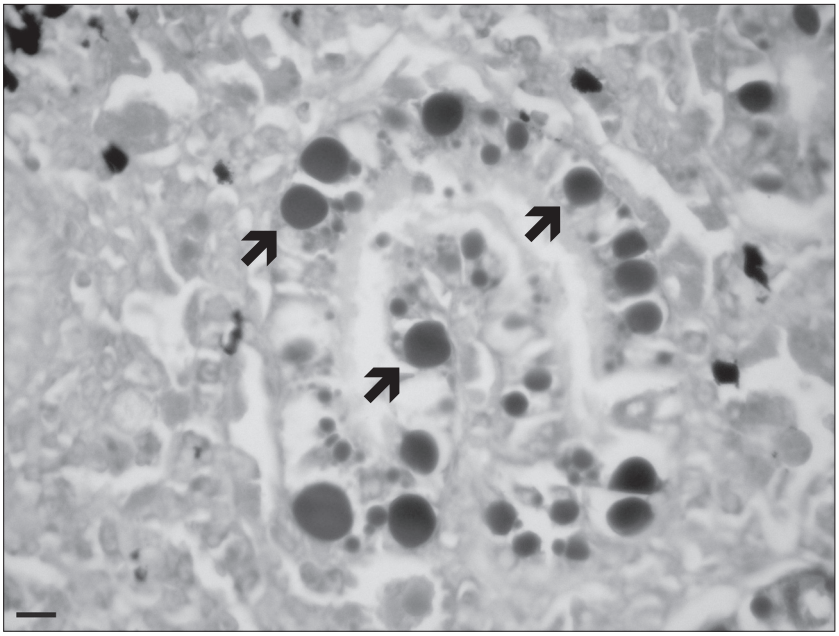

Figure 2. Ceroid deposition within the cells of kidney tubules. Note some very large inclusions occupying most of the cells and pushing the cytoplasm to one side of the cell (arrows). Ziel-Nielsen, Bar = 20 μm.

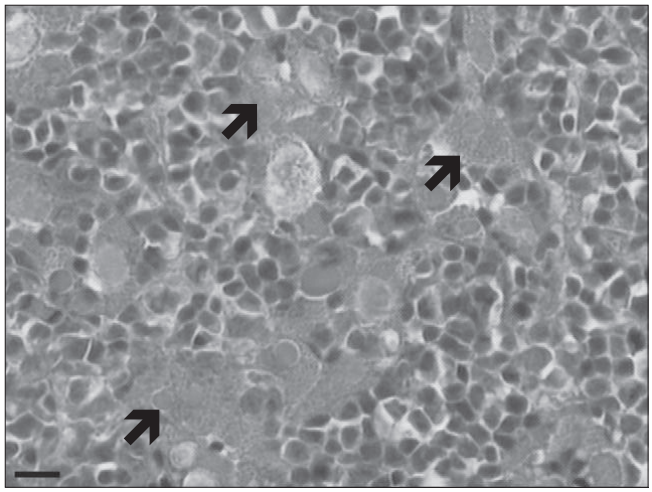

Figure 3. Ceroid deposition in cells of the kidney parenchyma. The cells have a large amount of very small ceroid droplets almost filling up the cell (arrows). Masson's Trichrome, Bar = 20 μm.

showed small lesions in the integument and fins, as well as some degree of retraction of the operculum, which are characteristics of farmed specimens which escaped from the net cages. All twelve specimens were females whose total length varied between 34.0 cm and 36.3 cm, and weighed 670 g to 800 g.

The histological examination of farmed fish (more or less of the same length and weight) sampled from all the farms, showed no ceroid deposition in the kidney or liver, and there was no abnormal mortality in the farms. All the specimens were free from the previously listed diseases.

All the feral *O. mykiss* studied in the present report showed severe kidney damage due to the deposition of ceroid pigment. Some morphological features indicated that these fish were feral fish which had previously escaped from netpens at Alicura impoundment. Obviously they continued to feed in the proximity of the cages profiting from the pellets provided to the farmed fish passing through the nets.

The interpretation of the present data is not easy. First it is difficult to explain why the fish did not develop LLD. Rancid food causes primarily ceroid deposition in the liver which is usually the main affected organ (Ashley, 1972; Snieszko, 1972; Penrith *et al.*, 1994; Tacon, 1996; Gatlin, 2008). Therefore, our findings are unusual and we do not have an explanation for such a heavy ceroid deposition only in the kidney. On the other hand, it is also difficult to understand why only the feral fish developed the condition. It would be expected that most of the fish at the farm would be affected, thus explaining the condition observed in feral fish, but this was not the case as revealed by the histological observations – not one farmed specimen showed ceroid deposition in the kidney or in the liver. Therefore, it has to be concluded that the fish food was in good condition and not oxidized. Also there was no abnormal mortality in the farmed fish, whose

condition was apparently good. It is known that heavy mortality rates are not uncommonly related to LLD ((Holliman and Southgate, 1986; Saraiva *et al.*, 1986; Atsuta *et al.*, 1991; Pearson and Chinabut, 1993).

The fact that wild fish did not present ceroid deposition is not surprising. Even in the case of ingestion of a certain amount of artificial food, their diet is likely to be more balanced by the ingestion of natural food.

In conclusion, we do not know how to explain this interesting case of ceroid deposition in the kidney of the fish under the circumstances described above. However, we considered that it would be interesting to communicate these observations to the scientific community.

References

Ashley LM (1972). Nutritional pathology. In "Fish Nutrition" (J Halver, Ed.), pp. 439-537. Academic Press, New York.

Atsuta S, Yakikurose H, Sakai M and Kobayashi M (1991). The causative agents of mortalities on pen.reared coho salmon, *Oncorhynchus kisutch*, in Japan. *Asian Fisheries Science* **4**, 115-118.

Blazer VS and Wolke RA (1983). Ceroid deposition, retinal degeneration and renal calcium oxalate crystals in cultured clownfish, *Amphiprion ocellaris*. *Journal of Fish Diseases* **6**, 365-376.

Gatlin DM III (2008). Non-infectious diseases: nutritional factors. In "Fish Diseases" , Vol. 2 (JC Eiras, H Segner, T Wahli and BG Kapoor, Eds), pp. 1201-1224. Science Publishers, Enfield (NH), USA.

Hardy RW (2001). The nutritional pathology of teleosts. In "Fish Pathology" (RJ Roberts, Ed) 3rd edn, pp 347-366. W.B. Saunders, London, Edinburgh, New York, Philadelphia, St. Louis, Sydney, Toronto.

Holliman A. and Southgate P **(**1986). Serious losses in rainbow trout (*Salmo gairdneri*) associated with hepatocellular ceroidosis. *Veterinary Record* **119**, 179.

Pearson M and Chinabut S (1993). Jaundice disease in the hybrid catfish. Animal Health Research Institute Newsletter, Bangkok, nº. 2.

Penrith ML, Bastianello SS and Penrith MJ **(**1994). Hepatic lipoidosis and fatty infiltration of organs in a captive African stonefish, *Synanceja verrucosa* Bloch and Schneider. *Journal of Fish Diseases* **17,** 171-176.

Roald SO, Armstrong D and Landsverk T (1981). Histochemical, fluorescent and electron microscopical appearance of hepatocellular ceroidosis in the Atlantic salmon *Salmo salar* L. *Journal of Fish Diseases* **4**, 1-14.

Saraiva A, Eiras JC and Bucke D (1986). Lipoid Liver Degeneration and pseudobranch pathology in rainbow trout, *Salmo gairdneri* Richardson, in Portugal. *Bulletin of the European Association of Fish Pathologists* **6**, 115-118.

Smith CE (1979). The prevention of liver lipoid degeneration (ceroidosis) and microcytic anaemia in rainbow trout *Salmo gairdneri* Richardson fed rancid diets: a preliminary report. *Journal of Fish Diseases* **2,** 429-437.

Snieszko SF (1972). Nutritional fish diseases. In "Fish Nutrition" (J Halver, Ed.) pp 404-437. Academic Press, New York.

Tacon AGC **(**1996). Lipid nutritional pathology in farmed fish. *Archives of Animal Nutrition* **49**, 33-39.

Weisman JL and Miller DJ (2006). Lipoid liver disease and steatitis in a captive sapphire damsel *Pomacentrus pavo*. *Acta Ichthyologica et Piscatoria* **36**, 99-104.