

ACUTE EXPOSURE OF JUVENILE COBIA *Rachycentron canadum* TO NITRATE INDUCES GILL, ESOPHAGEAL, AND BRAIN DAMAGE

Ricardo V. Rodrigues*, Michael H. Schwarz, Brendan C. Delbos, Eduardo P. Carvalho, Luis A. Romano and Luís A. Sampaio

Universidade Federal do Rio Grande – FURG
Laboratório de Piscicultura Estuarina e Marinha
CP 474, Rio Grande, RS, 96201-900, Brazil
vr.ricardo@gmail.com

Cobia *Rachycentron canadum* is a fast growing fish with world-wide potential for aquaculture, with nascent production globally in recirculating aquaculture systems (RAS). Nitrate is the end process of ammonia nitrification and tends to accumulate in RAS. The objective of this study was to evaluate the acute toxicity and the histopathological effects of nitrate to juvenile cobia.

Juvenile cobia (6.87 ± 0.36 g; 11.8 ± 0.19 cm) were acutely exposed to nitrate (500–3000 ppm $\text{NO}_3\text{-N}$) plus a control for 96 h. Each nitrate concentration was carried out in triplicate. At the end of the nitrate exposure, surviving juvenile cobia were euthanized and fixed in formalin for histopathological evaluation. Median lethal concentrations (LC_{50}) of nitrate to juvenile cobia were estimated at 12, 24, 48, 72, and 96 h of exposure. Toxicity of nitrate increased significantly over time, the LC_{50} was reduced from 2,661 to 1829 mg/L $\text{NO}_3\text{-N}$ from 12 to 96 h (Table 1). Fish exposed to different nitrate concentrations displayed histopathological alterations in the gills, esophagus and brain. Gills revealed epithelial hyperplasia with completely lamellar fusion, telangectasy, and lamellar shortening induced by necrosis (Fig. 1). The esophagus presented hyperplasia of epithelium and mucus cells. The brain showed dead neurons, along with edema and necrosis (Fig. 2). The results of the present study indicate that despite survival at high nitrate concentrations, juvenile cobia presented serious histopathologies. Therefore, more studies are necessary to determine precisely safe and sublethal effects for chronic nitrate levels on juvenile cobia *R. canadum*.

Financial support: CNPq/MCT/MPA and VT-VSAREC

Table 1 – Median lethal concentrations (LC_{50}) of nitrate-N to juvenile cobia *Rachycentron canadum*. Data in brackets represent the 95% confidence interval of the LC_{50} .

Time (h)	$\text{NO}_3\text{-N}$ (mg/L)
12	2,661 (2,561 – 2,764) ^a
24	2,407 (2,236 – 2,592) ^{ab}
48	2,071 (1,929 – 2,222) ^{bc}
72	2,028 (1,888 – 2,179) ^c
96	1,829 (1,758 – 1,903) ^c

Values followed by different letters represent significantly difference ($P < 0.05$) after one-way ANOVA followed by the Test of Tukey.

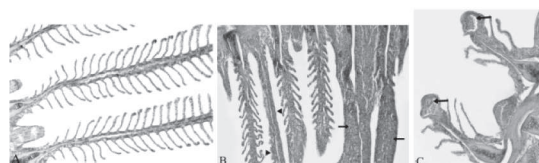


Figure 1. Photomicrography of gills of cobia *Rachycentron canadum* juvenile stained with haematoxylin-eosin. (A) Control treatment showing normal structure of gills (200 \times); (B) juvenile exposed to 1500 mg/L $\text{NO}_3\text{-N}$ for 96 h, showing hyperplasia in the secondary lamella with complete lamellar fusion (arrow), and lamellar shortening (arrow head; 200 \times); (C) juvenile exposed to 1500 mg/L $\text{NO}_3\text{-N}$ for 96 h, showing telangectasy of lamella (arrow; 400 \times);

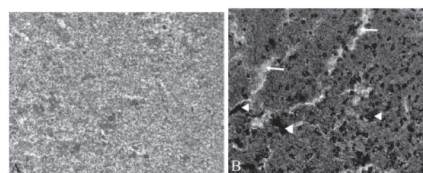


Figure 2. Photomicrography of brain of juvenile cobia *Rachycentron canadum* stained with silver staining technique. (A) Control treatment showing normal structure of brain (400 \times); (B) juvenile exposed to 1500 mg/L $\text{NO}_3\text{-N}$ for 96 h, showing dead neurons with edema (arrow head), and necrotic area (arrow; 400 \times).