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Gene networks and toxicity pathways induced by acute cadmium exposure in adult largemouth bass (*Micropterus salmoides*)

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ABSTRACT

Cadmium is a heavy metal that can accumulate to toxic levels in the environment leading to detrimental effects in animals and humans including kidney, liver and lung injuries. Using a transcriptomics approach, genes and cellular pathways affected by a low dose of cadmium were investigated. Adult largemouth bass were intraperitoneally injected with 20 µg/kg of cadmium chloride (mean exposure level - 2.6 µg of cadmium per fish) and microarray analyses were conducted in the liver and testis 48 h after injection. Transcriptomic profiles identified in response to cadmium exposure were tissue-specific with the most differential expression changes found in the liver tissues, which also contained much higher levels of cadmium than the testis. Acute exposure to a low dose of cadmium induced oxidative stress response and oxidative damage pathways in the liver. The mRNA levels of antioxidants such as catalase increased and numerous transcripts related to DNA damage and DNA repair were significantly altered. Hepatic mRNA levels of metallothionein, a molecular marker of metal exposure, did not increase significantly after 48 h exposure. Carbohydrate metabolic pathways were also disrupted with hepatic transcripts such as UDP-glucose, pyrophosphorylase 2, and sorbitol dehydrogenase highly induced. Both tissues exhibited a disruption of steroid signalling pathways. In the testis, estrogen receptor beta and transcripts linked to cholesterol metabolism were suppressed. On the contrary, genes involved in cholesterol metabolism were highly increased in the liver including genes encoding for the rate limiting steroidogenic acute regulatory protein and the catalytic enzyme 7-dehydrocholesterol reductase. Integration of the transcriptomic data using functional enrichment analyses revealed a number of enriched gene networks associated with previously reported adverse outcomes of cadmium exposure such as liver toxicity and impaired reproduction.