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Nutritional status as the key modulator of antioxidant responses induced by salinity stress and high ammonia in European sea bass

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We evaluated the individual and combined effects of salinity challenge, ammonia toxicity and nutritional status on oxidative stress and antioxidant status in European sea bass (*Dicentrarchus labrax*). Fish were progressively acclimated to normal seawater (32 ppt), to brackish water (20 ppt and 10 ppt) and to hypo-saline water (2.5 ppt), and were then exposed to high environmental ammonia (HEA, 20 mg/L) for 12 h, 48 h, 84 h and 180 h. Fish were either fed or fasted. Results show that in response to decreasing salinities, oxidative stress indices such as hydrogen peroxide (H_2O_2) and malondialdehyde (MDA) content increased in the hepatic tissue of fasted fish only. HEA exposure at normal (32 ppt) and at reduced salinities (20 ppt and 10 ppt) increased ammonia accumulation in both feeding treatments which were associated with an increment of H_2O_2 and MDA content. Unlike fasted fish, H_2O_2 and MDA levels in fed fish were restored to control levels with a concomitant increase in superoxide dismutase (SOD), catalase (CAT), components of glutathione redox cycle, ascorbate peroxidase activity and reduced ascorbate content. On the contrary, fasted fish could not activate many of these anti-oxidative sentinels. These findings exemplify that single factors and a combination of HEA with reduced seawater salinities (upto 10 ppt) were insufficient to cause oxidative damage in fed fish due to the highly competent anti-oxidant system compared to fasted fish. Overall, this research suggests that feed deprivation can make fish more vulnerable to oxidative damage during ammonia threat especially at reduced seawater salinities.

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