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Abstract

Several physiological functions in fish are shaped by environmental stimuli received during early life. In particular, early-life hypoxia has been reported to have long-lasting effects on fish metabolism, with potential consequences for fish life history traits. In the present study, we examine whether the synergistic stressors hypoxia (40% and 100% air saturation) and temperature (15° and 20°C), encountered during early life, could condition later metabolic response in European sea bass (*Dicentrarchus labrax*) juveniles. Growth rate and metabolic parameters related to carbohydrate and lipid metabolism in the liver were investigated at the juvenile stage under normoxic and chronic hypoxic conditions. Juvenile growth rates were significantly lower ($p< 1x10^{-6}$) under hypoxic conditions and were not improved by prior early-life exposure to hypoxia. Growth was 1.3 times higher ($p < 5x10^{-3}$) in juveniles reared at 15°C during the larval stage than those reared at 20°C, suggesting that compensatory growth had occurred. Early-life exposure to hypoxia induced higher ($p < 2x10^{-6}$) glycogen stores in juveniles even though there was no apparent regulation of their carbohydrate metabolism. In the liver of juveniles exposed to chronic hypoxia, lower glycogen content combined with stimulation of phosphoenolpyruvate carboxykinase gene expression and higher lactate concentration indicated a stimulation of the anaerobic glycolytic pathway. Furthermore, hypoxia only induced lower ($p< 1x10^{-3}$) lipid content in the liver of juveniles that had experienced 15°C at the larval stage. The present study provides evidence that environmental conditions experienced during early life shape the metabolic traits of *D. labrax* with potential consequences for juvenile physiological performance.

Key words: hypoxia, developmental plasticity, metabolism, European sea bass

Abbreviations: *Atgl,* adipose triglyceride lipase; CJ, control juvenile group; CL, control larval group; Cq, quantification cycle; *Dgat1,* diacylglycerol O-acyltransferase 1; DMSO, dimethyl sulfoxide; Dph, days post hatching; *Ef1α,* elongation factor-1 isoform alpha; *Glut2*, glucose transporter-2; GP,