Stress and heartache: Heart morphology deviation and energy resource exhaustion induced by chronic stress impaired the swimming performance of Atlantic salmon

Opinion April Grace[1], Alvéstegui Montalvo Debora Maria[2], Shiels Holly[3], Aerts Johan[4] and De Boeck Gudrun[5]

[1] ECOSPHERE, University of Antwerp
E-mail: aprilgrace.opinion@uantwerpen.be
[2] Ghent University, Coupure Links 653, 9000 Gent, Belgium
[3] University of Manchester, 46 Grafton Street, Manchester, United Kingdom
[4] Flanders Research Institute for Agriculture, Fisheries and Food, Belgium, Ankerstraat 1, 8400, Ostend, Belgium
[5] University of Antwerp, Groenenborgerlaan 171, 2020, Antwerp, Belgium

Salmonids have experienced considerable mortality rates in both aquaculture and natural environments. In Atlantic salmon (*Salmo salar*) aquaculture alone, concerns for animal welfare have heightened given the increasing frequency and severity of salmon mass mortality events with approximately 865 million deceased individuals recorded globally from 2012-2022. Although the exact causes are not fully understood, cardiac abnormalities have been identified as a major contributor to these losses. The development of these cardiac pathologies has been associated with stress, primarily based on studies involving exogenous exposure to the stress hormone cortisol. Since then, the link between cardiac abnormalities and endogenous cortisol has been implied but not established experimentally. Here, we exposed *S. salar* to unpredictable chronic stress for 29 days to induce a long-term endogenous stress response and compared their cardiac morphology and performance with undisturbed (control) fish. The cardiac parameters were then correlated with systemic stress indicators including plasma cortisol, glucose and lactate. Results showed that stressed fish developed bigger, rounder and less symmetrical hearts, which corresponded with the reduced cardiac performance indicated by the lower critical swimming speed (U_{crit}). The morphological remodeling of the heart in stressed fish appears to be linked to the sustained increase in cardiac workload, as evidenced by the chronic stress-induced elevation in routine heart rate. Although stress exposure did not affect the basal glucose and lactate levels, the magnitude of post-U_{crit} elevation of these parameters and the hepatosomatic index were lower in stressed fish indicating exhaustion of energy reserves that could further contribute to the observed U_{crit} impairment. Unexpectedly, the plasma cortisol levels were comparable between treatments, and could not help explain the observed cardiac remodelling. Overall, we show that long-term stress leads to the depletion of energy resources and cardiac impairment through mechanisms independent of plasma cortisol.

Keywords
Chronic Stress; Cardiac Performance; Heart Morphology; Glucocorticoids; Energy Metabolites