INSIDE JEB

Potassium leak short circuits trout heart at high temperatures

Rainbow trout (*Oncorhynchus mykiss*), swimming in the River Vrelo in Perucac, Serbia. Photo credit: Liquid Art CC BY-SA (https://creativecommons.org/licenses/by-sa/4.0).

Some fish could be facing the fight of their lives as temperatures rise. Jaakko Haverinen and Matti Vornanen from the University of Eastern Finland explain that there may come a point when their hearts simply can't take the heat any longer and give out. But what brings about this fatal heart failure at high temperatures? Scientists have known for almost a century that the heart ventricle fails before the atrium at high temperatures, but it wasn't clear whether the pacemaker that drives the beating atrium fails, causing the heart to beat dangerously slowly, or if the ventricle becomes less sensitive to the electrical currents that keep the heart ticking over. To discover which option sends trout hearts into collapse when the mercury rises, Haverinen and Vornanen began recording ECGs as they gradually increased the fish's temperature from a comfortable 12°C up to 27°C.

At normal temperatures (12°C), the bursts of electrical current in the two heart chambers (the atrium and the ventricle) that maintain the heart's rhythm were perfectly synchronised and remained so even as the temperature increased. But when the temperature hit 25°C, things began to go wrong. The coordination between the two heart chambers disintegrated: the atrium beat rate rocketed to 188 beats min^{-1} at 27°C while the ventricle failed to keep pace as its beat rate dipped to 111 beats min^{-1}. 'The heart has two separate beating rates at critically high temperatures, one for the atrium and another for the ventricle', say Haverinen and Vornanen. They realised that the ventricle was becoming less sensitive to the electrical currents that drive the heartbeat.

Next, the duo focused on the currents flowing into and out of heart muscle cells and tracked the failure back to ion channels that control the entry and exit of the sodium and potassium ions carrying the electrical currents that drive contractions. Placing minute pipettes filled with either potassium or sodium ions on the surface of muscle cells, they were able to record potassium ions leaking from the muscle at 25°C, making it impossible for sufficient sodium to flood into the cell to trigger a contraction.

The researchers conclude that the ventricle is the weak point that fails at high temperatures when potassium leaks from the muscle cells, preventing it from contracting. They add, 'the sequence of events from the level of ion channels to the cardiac function *in vivo* provides a mechanistic explanation for the depression of cardiac output in fish at critically high temperature'.

10.1242/jeb.230169

Haverinen, J. and Vornanen, M. (2020). Reduced ventricular excitability causes atrioventricular block and depression of heart rate in fish at critically high temperatures. *J. Exp. Biol.* **223**, jeb225227. doi:10.1242/jeb.225227

Kathryn Knight
kathryn.knight@biologists.com