Inside JEB is a twice monthly feature, which highlights the key developments in the *Journal of Experimental Biology*. Written by science journalists, the short reports give the inside view of the science in JEB.

Inside JEB

A TRIBUTE TO PETER LUTZ

One defining feature of Peter Lutz's career was that he was fascinated by a wide range of animals and how they coped with environmental extremes, particularly hypoxia and anoxia. When he passed away in February 2005, his friends and colleagues wanted to celebrate his career and the diversity of his research interests. The four papers featured in this issue of *The Journal of Experimental Biology* touch on many aspects of how animals survive with little or no oxygen. They follow on from the major contributions that Peter Lutz made to the study of diving adaptations in sea turtles, and also to the understanding of the mechanisms of anoxic brain survival in the freshwater turtle.

It is because of the extraordinary ability of freshwater turtles to survive anoxia that they have been intensively studied as anoxic 'model organisms'. Moving from the turtle brain to the turtle heart, Johannes Overgaard, Hans Gesser and Tobias Wang review how freshwater turtles' hearts cope during anoxia and hypoxia (p. 1687). During the cold winter months, turtles settle down on pond beds to see through the winter as ice forms on the surface above. Under these anoxic conditions, few animals would survive, but many turtles emerge from their overwintering unscathed. How do they keep their cellular energy levels high enough so that cells don't die, but not run down their precious energy supplies? Turtles have an arsenal of coping mechanisms at their disposal, from matching their energy use with the low amount of ATP produced by anaerobic metabolism, to relying on breakdown of the molecule phosphocreatine to top up ATP levels and using their shells to buffer against the acidic conditions.

Moving from turtles to fish, Tony Farrell discusses another cardiac response to low oxygen: hypoxic bradycardia, where heart rate slows down dramatically when oxygen is scarce (p. 1715). But what are the benefits to the fish? Researchers suspect that bradycardia benefits the heart muscle because blood is held within the heart for longer, giving oxygen more time to diffuse into the muscle. An unusual property of fish heart muscle allows them to extensively increase cardiac stroke volume – the volume of blood shifted out of the ventricle per heart beat – while maintaining cardiac output, the total volume of blood pumped each minute. Not only will this send enough blood to the body tissues, but also stretch the walls of the heart, further favouring oxygen diffusion, and allowing the fish to draw out as much oxygen as possible from the ever dwindling supply in venous blood. Bradycardia severe enough to reduce the heart's output and demand for ATP could also be beneficial if the heart has to temporarily perform without any oxygen and rely on glycolysis, something that Peter Lutz's favourite animal model, the turtle, are experts at. Farrell also highlights the intriguing possibility that some fishes lost the bradycardia response when they evolved air breathing: the circulatory design of fish means that air breathing during hypoxia raises oxygen partial pressure in venous blood and hence oxygen availability to cardiac tissues.

Low oxygen levels do more than just trigger a cardiac response; when the going gets tough animals slow down their metabolism, which can also occur if the temperature plummets or if food and water are scarce. As Kenneth and Janet Storey explain, hypometabolism caused by hypoxia is easiest to study, and the freshwater turtle is the experimental organism of choice for many researchers (p. 1700). Animals need to ensure that their cells have enough ATP to survive so hypometabolism is highly regulated. They both minimize and reprioritize their ATP use to sustain necessary processes such as ion pumping and largely shut off optional ones like protein synthesis. At the same time other processes swing into action to protect the cell, including antioxidants, which mop up damaging reactive oxygen species, and chaperone proteins and protease inhibitors that protect the cell's macromolecules. This ensures that the molecules stay stable and cells remain viable, ready for action when oxygen levels rise once more.

Meanwhile, some colourful newcomers to the field of hypoxia research have been introduced by Göran Nilsson, Jean-Paul Hobbs and Sara Östlund-Nilsson (p. 1673). Tropical coral reefs are littered with hypoxic hotspots and the brightly coloured fishes that teem over the reefs have to be very adaptable to these conditions. At night, crevices in between corals become

hypoxic since photosynthesis has stopped, however fish rely on these crevices to hide from predators. Fishes may also become trapped in hypoxic shallow pools when the tide goes out at night, and some are able to breathe air by absorbing oxygen through their skin when the coral becomes exposed. Another challenge faced by the males of some mouth-brooders is maximising clutch size, while still being able to swim and keep water flowing over the gills. While researchers have only just begun to scratch the surface of how these fish adapt to hypoxia, studying one of the most diverse vertebrate communities in the world means that researchers 'can be certain that a wealth of respiratory adaptations remain to be discovered among coral-reef fishes', says Nilsson.

It is clear from the papers in this tribute that Peter Lutz's contributions to integrative physiology continue to inspire researchers studying animals in hypoxic environments. As Tony Farrell writes, 'he sought answers to the question why…and had an infectious enthusiasm for science'. Nilsson is sure that Lutz would have joined him and his colleagues in their study of tropical fishes, continuing to challenge himself and others to make exciting discoveries.

10.1242/jeb.02786

Overgaard, J., Gesser, H. and Wang, T. (2007). Tribute to P. L. Lutz: cardiac performance and cardiovascular regulation during anoxia/hypoxia in freshwater turtles. *J. Exp. Biol.* **210**, 1687-1699.
Farrell, A. P. (2007). Tribute to P. L. Lutz: a message from the heart – why hypoxic bradycardia in fishes? *J. Exp. Biol.* **210**, 1715-1725.
Storey, K. B. and Storey, J. M. (2007). Tribute to P. L. Lutz: putting life on 'pause' – molecular regulation of hypometabolism. *J. Exp. Biol.* **210**, 1700-1714.
Nilsson, G. E., Hobbs, J.-P. A. and Östlund-Nilsson, S. (2007). Tribute to P. L. Lutz: respiratory ecophysiology of coral-reef teleosts. *J. Exp. Biol.* **210**, 1673-1686.

A NOSE FOR SURVIVAL

Life can sometimes be pretty dangerous for larval California newts (*Taricha torosa*). If the adults' favourite diet of worms is scarce, they cannibalise their larvae for an alternative snack. Adult newts in turn avoid being munched by secreting the paralysing poison tetrodotoxin (TTX) from their skin as a defence against predators. Richard Zimmer from the University of California, Los Angeles, was surprised to see that

when larvae caught a whiff of TTX they would flee to the nearest shelter, suggesting that TTX was also acting as a warning signal. By delving into the newts' complex chemical world, Zimmer and his colleague Ryan Ferrer found that interactions between different chemicals affect the behaviour of both larval and adult newts, in very different ways.

The team were alerted to the fact that the story might be more complicated when they saw research by Jacob Kerby and Lee Kats showing that when adults are dining on their favourite worms, larvae stay put. Worm body fluid contains a lot of the amino acid arginine, which is also a feeding cue in aquatic systems. Could arginine be suppressing the larval newts' response to cannibalistic adults? Similarities in the chemical structure between arginine and TTX led Zimmer and Ferrer to suspect that these two chemicals were interacting and somehow affecting larval behaviour.

Following their hunch, the team collected eggs from the field and hatched them back in the lab to find out how the larvae would react to TTX and arginine (p. 1768). They placed the larvae in specially designed flow tanks, and targeted a stream of TTX solution towards them, finding that TTX on its own caused the larvae to swim for shelter, as they expected. When they blocked their noses with inert silicon gel, the larvae didn't flee, showing that they were 'smelling' TTX in the water. Next they mixed arginine with TTX, to simulate adults gorging themselves on worms, and found that the larvae didn't try to escape to a refuge, showing that arginine cancelled out the escape response caused by TTX. Because of the similarity in structure between TTX and arginine, the team suspect that these two chemicals are probably competing for olfactory binding sites in the nose: arginine binds instead of TTX, meaning that the larvae effectively can't smell TTX.

But how robust was the larvae's response? The team chemically manipulated the structure of the different groups on the arginine molecule, and found that only changes to the guanidinium group – the structure arginine shares with TTX – prevented arginine from blocking the larvae's response to TTX, suggesting that this group is key to how the newts' noses detect TTX and arginine.

Next, Ferrer and Zimmer trekked to the field site in Malibu, California, carrying all their equipment in backpacks, to test how the adults responded to arginine (p. 1776). To introduce the chemicals into the stream water from a distance without disturbing the newts, the team devised a system involving 3 m long transparent hollow rods connected to pumps. 'The animals can be fairly skittish, so we had to remove all visual cues,' says Ferrer. Testing the response of adults to amino acids released from damaged worms, including others like alanine and glycine, they found that the newts responded most strongly to arginine

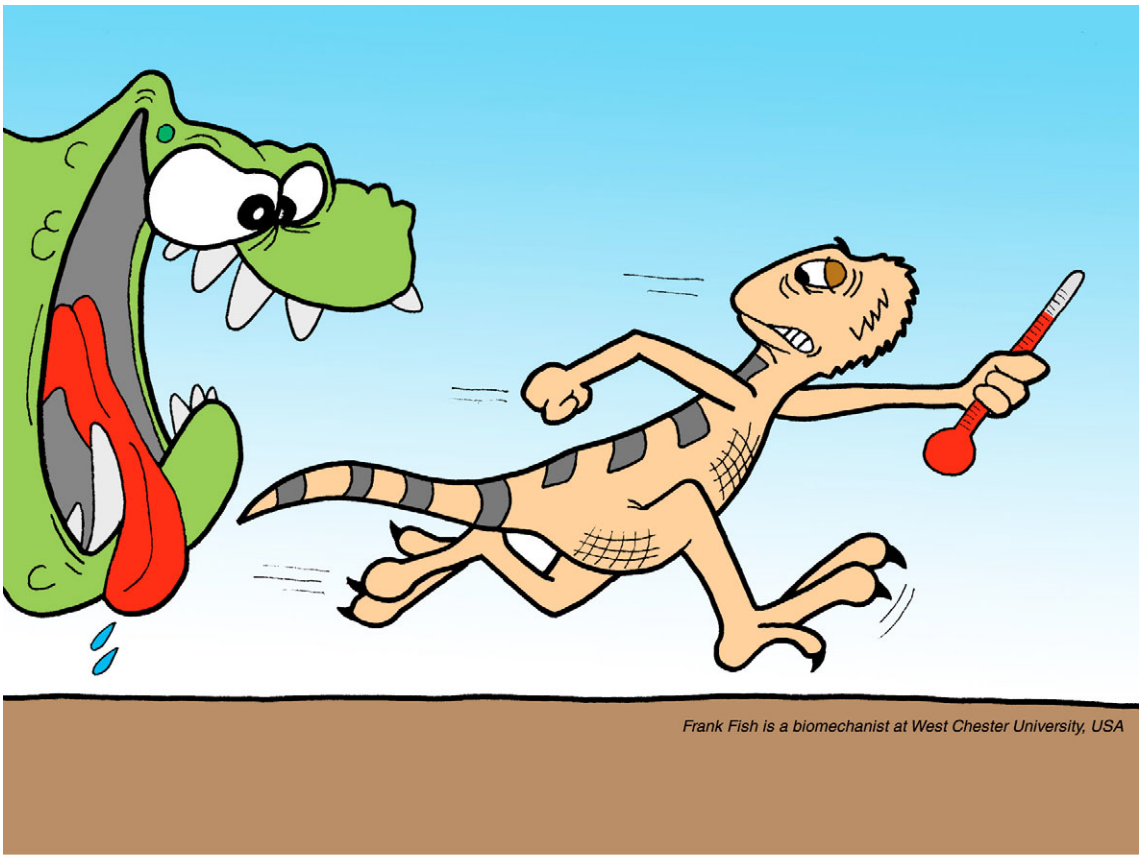
by swimming towards the source of the smell, and by raising their snouts into the odour plume or burying themselves into the stream bed. Newts with blocked up noses didn't respond, and they also couldn't detect arginine if the molecule had been modified in any way, showing that the adult newts had a much more specific response to arginine and were probably 'smelling' it in a different way.

'The big differences in behaviour caused by arginine between larvae and adults suggest that there is a change [in the nose] happening at metamorphosis' says Ferrer. So depending on whether you are little or big, arginine could be a signal that dinner is served, or that you don't have to swim for cover…yet.

10.1242/jeb.02787

Ferrer, R. P. and Zimmer, R. K. (2007). The scent of danger: arginine as an olfactory cue of reduced predation risk. *J. Exp. Biol.* **210**, 1768-1775.
Ferrer, R. P. and Zimmer, R. K. (2007). Chemosensory reception, behavioral expression, and ecological interactions at multiple trophic levels. *J. Exp. Biol.* **210**, 1776-1785.

BITE OR SPRINT?



Luckily, Trapelus had the temperature and knew it would be better to run than fight.

If a predator is looming on the horizon, agamid lizards, *Trapelus pallida*, prefer to run off in the opposite direction. But sometimes it is too cold to run, so as a last resort the lizards will stand their ground, behave aggressively, and bite. Scientists suspected that there might be a physiological reason for this shift in behaviour: the ability of a muscle to generate force, which is important during biting, is less temperature dependent than power output and contraction speed, which are important during running. This implies that lizards could defend themselves by biting at all temperatures, relying on running at higher temperatures only.

To test if physiological differences in lizards' leg and jaw muscles would explain the shift in behaviour, Anthony Herrel and his colleagues measured lizards sprinting down a 2 m track at different temperatures, finding that as temperature increased, the lizards ran faster (p. 1762). When they measured the lizards' biting force at a range of temperatures, they found that they could bite with a similar force at all temperatures, showing that force was less affected by temperature than muscle power and contraction speed.

To find out how temperature affected the largest muscle in the leg, and another in the jaw, they measured both muscles' contraction properties at different temperatures. In both muscles, maximum force production was not affected that much by temperature, while power and contraction speed improved with warmer temperatures. However, the jaw muscle was better at producing maximal force at all temperatures than the leg muscle. This suggests that the properties of the muscles may limit how the lizards can behave at different temperatures, and explain why they are more likely to run when it's warm and bite when it's cold.

10.1242/jeb.02788

Herrel, A., James, R. S. and Van Damme, R. (2007). Fight *versus* flight: physiological basis for temperature-dependent behavioral shifts in lizards. *J. Exp. Biol.* **210**, 1762-1767.

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