Inside JEB, formerly known as 'In this issue', 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

DEATHLY SILENCE IMPROVES A BAT'S CHANCE

Once a bat has locked its beam of sound onto a moth victim, there's little hope for the fluttering insect. As the bat closes in, it switches its call pattern, triggering the victim to fly erratically in a last attempt to elude the predator as it homes in. And all the time, the moth is listening to the attacker's ultrasonic calls. James Fullard is fascinated by moths' simple ears, which have mostly evolved to defend the moth from a single predator, the bat. He explains that moths hear their bat infested world through simple ears. Some species have as few as one sound receptor to detect the incoming threat, but how does this simple auditory system respond during the closing seconds of a bat attack? Working with his colleagues Jeff Dawson and David Jacobs, Fullard began playing recordings of a closing bat's cries and recording how the moth's ear sound receptors responded. The team expected to see the nerve impulses produced by the receptors rocket as the bat recording closed in, but were astonished when the moths' gave up warning of the impending attack, and continued firing as if the bat had suddenly retreated (p. 281)!

How bats and moths interact acoustically has fascinated scientists since the late 1950s, when Kenneth Roeder began analysing moths' responses to bats in his back yard. He discovered that noctuid moths, such as the underwing moths, have two auditory cells, called A1 and A2, while notodontid moths lack the A2 receptor, so they pick up the whole attack with the A1 cell alone. Roeder had suggested that the moth's vain attempts to escape capture were triggered by signals from the A2 cell, but how would the notodontids evade attack when they lack the A2 receptor?

This wasn't clear, and it was impossible to simulate the sound of an incoming bat from a moth's perspective, until James Simmons made some unique bat recordings; he trained a bat to attack a microphone in the lab. Fullard and his team decided to replay the final two seconds of Simmon's remarkable recordings to noctuid and notodontid moth ears, and recorded their nerve signals to see what the moths really heard.

As expected, the A1 cell burst into action as the sounds closed in, firing with increasing intensity as the bat approached. The team kept waiting for the A2 cell to begin firing, but the cell didn't respond strongly during the insect's final moments, so the A1 receptor tracks the attack from start to finish. Even more surprisingly, within the moth's final fifth of a second of life, the A1 cell suddenly gave up firing rapidly, and returned to signalling intermittently, as if the attack had failed and the bat was some distance away!

Fullard explains that there are two possible reasons for the moth's sudden deafness. The most obvious explanation is that there's little point in registering the final assault, as the moth has no chance of escape once the bat is within striking range.

But as predation by bats is the major evolutionary pressure on moth ears, Fullard has an alternative theory. He suggests that the hunters could have selected for failure of the A1 receptor, so that the moths final moment of peace improves the bat's chances of success.

10.1242/jeb.00120

Fullard, J. H., Dawson, J. W. and Jacobs, D. S. (2003). Auditory encoding during the last moment of a moth's life. *J. Exp. Biol.* **206**, 281-294.

NEMATODES FREEZE-DRY TO SURVIVE

Even though 99% of the Antarctic land mass is smothered in ice, it is one of the driest places on earth. Few plants and animals have risen to the challenge of surviving at the continent's bitterly cold

temperatures, but even though the continent's temperature can fall forty degrees below freezing, some nooks and crannies manage to reach temperatures that would be pleasing even in temperate zones. However, surviving the daily freeze/thaw cycle adds another challenge to microscopic nematodes that must protect themselves from the extensive damage wreacked by ice crystals. David Wharton is fascinated by how animals survive in the planet's harshest environments, and recently became interested in how Antarctic nematodes survive freezing. Amazingly, *Panagrolaimus davidi* is one of the few creatures that can survive intracellular freezing, but when Wharton looked at the way that the nematode responds at temperatures close to zero, he found that rather than freezing solid, the nematodes survive by freeze-drying (p. 215).

Wharton describes how he found the nematodes in a coastal valley when he visited Antarctica in 1989. The nematodes were living on a patch of algae that were growing in a snow-melt stream. When he returned to his New Zealand lab, he realised that the nematodes were remarkably easy to keep in culture, flourishing at 25°C and producing another generation every week, as opposed to the year that it takes the worms to reproduce in their natural environment.

Fascinated by how the worms survived freezing, Wharton had watched ice crystals form in the worm's cells as he dropped the temperature, but he had also realised that some of the worms didn't freeze; they shrivelled up and dried out instead. Keen to know why some worms froze while others desiccated, Wharton began watching how the worms fared as ice formed around them at different temperatures.

Wharton suspended thousands of the nematodes in water, and sandwiched the drop between two coverslips so that he could film the worms as the water froze. After lowering the temperature, he initiated freezing by touching the edge of the droplet with an ice crystal, and watched as the ice slowly encased the nematodes. After 30 minutes Wharton recorded the fraction of frozen nematodes versus dehydrated worms, and found that even though the water was completely frozen at –1°C, all of the worms had dehydrated, while at –5°C, all of the worms froze, and only one third had survived when they thawed.

He also tested how the nematodes reacted as he cooled the forming ice at different rates and found that when the worms iced up quickly, they couldn't escape freezing, but if he cooled the forming ice slowly, again the worms dehydrated.

But how do the worms escape freezing at some temperatures and cooling rates and not others? Wharton explains that as the water surrounding the nematode freezes, the external vapour pressure falls below the vapour pressure of the liquid water still inside the worm's cells. If the worm is cooling slowly, or it is only just below freezing, the water is gently pulled out of the worm. But if the temperature drops too quickly before the dehydrating pressure gradient is established the worm cannot escape, and it freezes.

Wharton explains that some earthworms also protect their eggs from subzero temperatures by dehydration, but the Antarctic nematodes have capitalised on both approaches to thrive at a pedestrian rate in the coldest place on earth.

10.1242/jeb.00118

Wharton, D. A., Goodall, G. and Marshall, C. J. (2003). Freezing survival and cryoprotective dehydration as cold tolerance mechanisms in the Antarctic nematode *Panagrolaimus davidi*. *J. Exp. Biol.* **206**, 215-221.

COORDINATED GULPING

When *C. elegans* dine on bacteria, they Hoover up their meal quickly, and move on until they happen upon another bacterial colony. The nematodes gulp down their food by rhythmically contracting their muscular pharynx. Each stage of the contraction and relaxation cycle must be tightly coordinated as they gear up pumping from a few gulps per minute to five per second during a feast, otherwise the worm's pharynx blocks and the worm starves, even in the midst of plenty! The neurotransmitter serotonin also increases the pharynx's pumping frequency, but how it did this puzzled Timothy Niacaris and Leon Avery. Over the years, Avery's lab has built up a detailed understanding of the pump's physiology, identifying two neurons, MC and M3, that initiate the muscle's depolarisation and repolarisation, generating an action potential, which in turn initiates a contraction/relaxation cycle. But how serotonin functions to speed up pumping wasn't known. As shorter action potentials translate into faster pumping rates, Niacaris decided to measure how the muscle's action potential changed in response to serotonin. Working with nematodes where one or both of the nerves had been disconnected, Niacaris measured the muscle's action potential length in response to serotonin, and realised that the neurochemical coordinates faster pumping by altering the timing of the muscle's depolarisation and repolarisation (p. 223).

Niacaris worked with three groups of mutated worms to see if he could find which aspects of the muscles control system serotonin was regulating. The first group had lost components of the M3 circuit and retained the MC circuit that drives depolarisation of the muscle and triggers the muscle to contract. The second group of worms had lost the MC circuit, but retained the M3 circuit that drives the muscle's repolarisation at the end of the action potential, and allows the muscle to relax. Both neurons had been disconnected in the third group. Niacaris measured how serotonin affected the duration of the pharynx muscle's action potential in each group of worms.

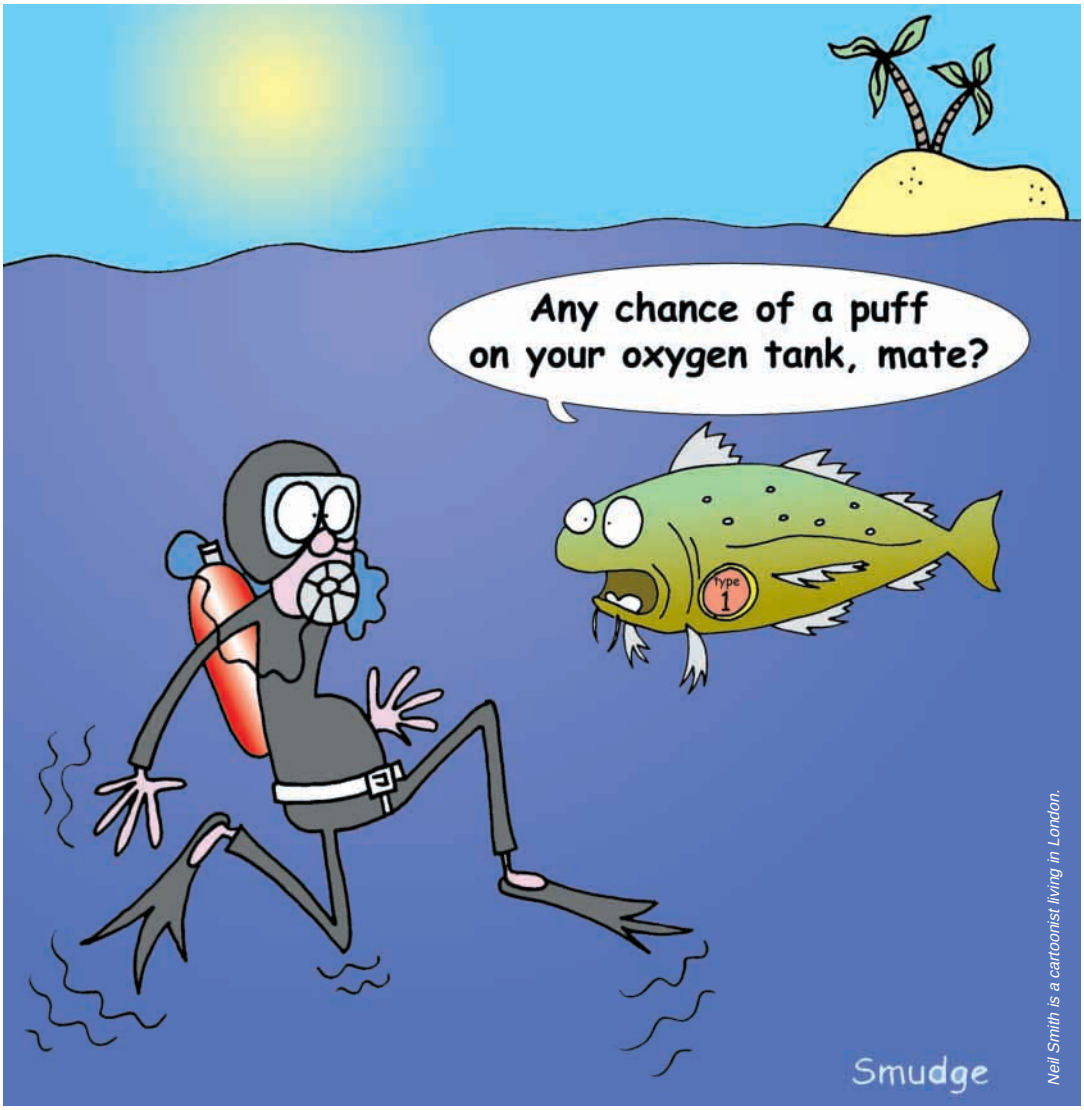
When he tested worms that retained use of one neural circuit, serotonin shortened the action potential, proving that both neurons were involved in faster pumping. But Niacaris needed to rule out the possibility that serotonin also affected any other neural circuits that could also alter the action potential length, so he tested the worms where both neurons had been disconnected. If serotonin affected the action potential through other neural circuits, then the neurotransmitter could still shorten each action potential, even though MC and M3 were disconnected. But these worms' action potentials didn't change, so serotonin only affects two neural circuits to control the roundworm's rhythmic feeding response.

Niacaris explains that it's relatively easy to understand how stimulating the M3 circuit with serotonin cuts the action potential length by swiftly repolarising the muscle, but it's less clear how altering the initial depolarisation also shortens the action potential. He adds that understanding how the tiny worms feed efficiently could help clinicians understand arrhythmia in the human heart. He explains that many of the key protein components that regulate the roundworm's pharynx have homologues in human heart muscle. So understanding how serotonin helps the round worm's pharynx to keep time could eventually help clinicians treat heart attacks by targeting the timing circuit to reset the heart's contraction clock.

10.1242/jeb.00119

Niacaris, T. and Avery, L. (2003). Serotonin regulates repolarization of the *C. elegans* pharyngeal muscle. *J. Exp. Biol.* **206**, 223-231.

COD COULD FACE A BREATHLESS FUTURE



As fish migrate over huge distances in the oceans, the various habitats that they experience at different stages of their lives can disrupt many physiological processes, so some species have evolved subtly altered haemoglobins, optimised to carry oxygen in different environments. Stocks of North Atlantic cod can be divided into haemoglobin type I, haemoglobin type II and a hybrid of the two subtypes, depending on the sequence of their haemoglobin peptide chain. Back in the 1960s, K. Sick found that type II fish predominantly occupied the cold waters of the North Atlantic, while the type I fish were found predominantly in the southern North Sea. Knowing that many environmental factors affect a fish's choice of temperature Maria Petersen and J. Steffensen wondered how fish with the two blood types would respond to hypoxia (p. 359). The team selected young cod with both blood types, and allowed them to swim in a shuttle box, where the fish could control the water temperature until they found their preferred temperature. When the fish settled, the team found that fish with type I haemoglobin preferred waters that were almost 8° warmer then the type II fish. Then they reduced the oxygen content of the water and waited to see which temperature the hypoxic fish preferred. When the oxygen levels were low, the type II fish chose water at the same temperature as they had when oxygen was abundant, but the type I fish preferred cooler waters when the oxygen level fell. Petersen and Steffensen explain out that moving to cooler waters would help the type I fish conserve oxygen in hypoxic conditions. But this could also face cod with a serious problem. If global temperatures continue to rise, then type I fish would flourish in the warmer Atlantic waters, but if oxygen levels fell too, the fish would come under threat, unable to retreat to more comfortable climes.

10.1242/jeb.00122

Petersen, M. F. and Steffensen, J. F. (2003). Preferred temperature of juvenile Atlantic cod *Gadus morhua* with different haemoglobin genotypes at normoxia and moderate hypoxia. *J. Exp. Biol.* **206**, 359-364.

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