**Winter Warmth**

Man has bucked the trend when it comes to over wintering. As the nights draw in and the temperatures drop, we retreat to our homes, and turn the heating up. But other mammals don’t have that choice. For them it’s adapt or die. For small animals that have a large surface-to-volume ratio and cool rapidly, the problem is even more urgent. Stuart Egginton assumed that animals which pass the winter in hibernation use different approaches to preparing for the cold than animals that keep active to stay warm. He tricked hamsters and rats into preparing for winter and looked at the way they modified their muscles with the unexpected result that rats put on muscle, while the hamster’s muscles wasted (p. 829).

Small rodents have two choices; either to cool down and hibernate like hamsters, or to burn more calories to keep warm like rats. Active animals generate much of their heat in their skeletal muscle, while dormant hamsters have to maintain their muscles during their inactivity, ready for when they reawaken.

If you want to know how an animal really prepares for the winter, you’ve got to slow them down gradually. Just putting an animal into a fridge is nothing like the real onset of winter, it’s ‘cold’ shock. Egginton and his graduate student Durmus Deveci set about designing ‘rodent biospheres’ where they could control the temperature and day length to convince the animals to prepare for winter. Over a month, they cooled the animals from long warm late summer days down to a day that lasted just 1 hour at 5°C. Once the animals had spent another month in their artificial winter conditions, they compared the winter animals with their summer cousins. Deveci had a surprise when he looked at the rats; their muscles were built up, while everyone else who’d ever looked at cold rats had watched the animal’s muscles wither.

The rats need bigger muscles to generate the heat to keep them warm. Deveci saw that the muscle fibres had expanded. Bigger muscles also need more fuel and generate more waste, so the rats also have to plumb-in more blood vessels to supply the bigger muscles. But the increased blood flow doesn’t increase the muscle’s capillary density, because the muscle had expanded at the same rate as the increase in the number of capillaries.

Hamsters, on the other hand, have an entirely different set of problems. As they prepare for their long winter nap, they cool down and their blood thickens, which makes it harder to pump through tiny capillaries. At the same time, the animal’s muscle fibres shrivel because it is immobile. The hamsters don’t put more blood vessels into their muscles to overcome the problem of pumping thicker blood, they concentrate the same number of capillaries into a smaller muscle to increase the muscle’s capillary density and maintain the essential fuel supply to the resting muscle.

Egginton’s rodent biosphere has turned out to be the key to this truly comparative study of the animal’s approaches to over wintering. Both animal’s successful survival hinges on a good blood supply, but they use entirely different body building strategies to get them through to spring.

**Scorpion’s Sting Stops Sodium**

Scorpions sting over 250,000 people every year in Mexico alone. Most victims are lucky enough to survive if they get a dose of anti-venom in time, but several hundred still die every year because they can’t reach medical assistance. Scorpion venom is a cocktail of hundreds of individual toxins, which destroy the victim’s nervous system by targeting specific molecules in the nerve cells. Lourival Possani, working in Cuernavaca has discovered a completely new type of scorpion toxin that wrecks havoc by plugging the nerve’s essential sodium channels (p. 869).

Electrical signals flash along nerve cells when ion channels allow ions to flood into the cell, and then out again, as the nerves electric charge changes and the electrical signal moves along the cell. The ion channels select one of four ion types as the nerve cell fires. Possani is particularly interested in the scorpion sting’s toxic ingredients and their fatal biochemistry. But each scorpion produces a vanishingly small amount of venom, so Possani collects stings from over 20,000 scorpions before he has enough of the lethal cocktail to separate the individual toxic components. Although a single scorpion’s sting will be deadly for many different species, each species will be targeted by a unique set of toxins from the mixture, and those same poisonous peptides may be completely inert in another species. Separating the individual components on the bases of the molecule’s size and charge, Possani is able to test which animal the toxin attacks, before finding out how it delivers its fatal blow.

Toxin Cn11 makes up less than two percent of a scorpion’s sting, but 15 µg is enough to kill an adult crayfish. Comparing the peptide’s sequence with other scorpion toxins, Possani realised that he was dealing with a toxin that interfered with the nerve’s sodium ion channels. But how was the toxin disrupting with channels behaviour? The only way to find out was by looking at a poisoned nerve’s dying signals.

Possani’s team turned to electrophysiology and recorded the electrical signal from entire crayfish nerve cells as they washed the neurons with Cn11. Because they measured the voltage of an entire cell, Possani’s team was looking at the sum of each sodium channel’s contribution to the nerve’s electrical signal. If the toxin was jamming the channel closed, small amounts of toxin would only shut a few channels, so the current would fall a little. But as they increased the amount of toxin, more channels would shut, dropping the current further, until he administered enough toxin to close every channel and completely shut the current off. Possani’s recordings proved that the he had unexpectedly found the first sodium channel blocker in scorpion toxin.

Possani is very excited. Cn11 is the first example of an entirely new class of toxin. A few other creatures have also developed sodium channel blockers, but they have very different structures and ways of blocking the ion channel. Although many technical challenges remain, Possani is optimistic that this toxic peptide could become a useful probe that might help us unravel the complicated structure of its victim; the sodium ion channel.

**Still Wired**

Mel Robertson got interested in how temperature affects an insect’s nervous system one hot day almost 20 years ago. The lab’s air conditioning had broken down, and as the temperature rose, the experiments worked better than they ever had at room temperature! He realised that this made perfect sense, because cold-blooded insects keep flying, even at temperatures that no human nervous system could survive. Since then, Robertson has concentrated on finding out what protects the insect’s wiring against naturally rising temperatures. He has now added anoxia to the list of protective shocks that prime the insect’s nervous system to survive rocketing temperatures (p. 815).

All life depends on the ATP that is generated by aerobic respiration to drive its key processes, and the nervous system is no exception. But at times of physical stress, locusts conserve their...
nervous system’s valuable ATP stores by reducing the nervous system’s potassium currents, saving the huge amounts of energy required to pump potassium ions back into the cell after the nerve has fired. Robertson wondered whether depriving the insect of oxygen would reset the nervous system’s protection mechanisms to conserve ATP so that it could survive even higher temperatures than it could have before with-holding oxygen.

After two hours in a nitrogen environment, he raised the temperature to 53°C to see how well they survived the baking temperatures. After half an hour, six of the original 15 insects survived, while all of the insects that were heat shocked without preparation died! The insect’s nervous system was also able to stand even higher temperatures than before; the locust’s nerve cells were able to generate an action potential during a heat shock that destroyed unprepared neurons. Robertson realised that the insect reset the nervous system’s potassium channels so that it continued to send nerve signals at very high temperatures. But it didn’t work the other way around. An insect that had been heat shocked first was just as vulnerable to anoxia as before.

But would insect’s muscles survive as well? It’s no use having a nervous system that survives boiling temperatures if the signals it sends fall on deaf muscles. When Robertson looked at the insect’s muscle function under different stresses, he discovered that the heat pre-stress, which protected the insect’s nervous system, disrupted its muscular function! The insect could still fly, but the damaged muscle uses more energy.

Although some stresses were able to improve some component’s performance, an improvement in the nervous system’s function wasn’t always matched by an improvement in other essential flight systems. So it’s not enough to look at an individual component in isolation, biology is complex, and an animal’s ability to survive a variety of stresses can only be assessed by looking at the entire animal’s response. Even if the insect can tell its wings to flap, its damaged muscles might not be able to lift it from the ground.

Robertson has reason to believe that the locust’s flight control system could have parallels with the human respiratory system that may help us to understand situations when our breathing fails, such as hyperthermia. Robertson hopes that learning how a simple motor control system failure keeps an insect grounded could help us to understand how the human respiratory system collapses as temperatures rise.

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Still Nippy!