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

CHICKS ARE COOL ABOUT STAYING WARM

Most chicks spend their young lives being waited on by their parents; all they have to do is eat and grow. But precocial chicks are much more independent. Within hours of breaking out of their shells, the youngsters are foraging for themselves. Which is fine in temperate climes, where the chicks stay warm, but for shorebird chicks in the Canadian tundra, the cold conditions place a massive metabolic burden on their tiny bodies. Robert Ricklefs is fascinated by the precise metabolic balance that the birds strike as they stray from the nest's warmth, gathering food to fuel their fast growth rate. He needed to know how newborn chicks cope with the cold, so he began slowly cooling and warming the youngsters in the laboratory while he measured their metabolic rate, and found that the chicks seem to be saving energy by dropping their metabolic rate and letting the environment do the work as they warm up (p. 2883).

Ricklefs and Joe Williams headed north to the Canadian tundra to put the young birds to the metabolic test. But capturing the chicks was tricky. Once the youngsters have hatched, they scatter into the undergrowth, 'and become invisible' says Ricklefs. So instead of returning with chicks, the team gathered eggs from six species, incubating them in the lab until they hatched. Having made sure that the chicks were happy foraging in their protected enclosure, the team began measuring a dunlin chick's metabolic rate and body temperature, as the air temperature changed.

At first, the dunlin's metabolic rate rose slowly as the air slowly cooled around it, but even with the extra metabolic effort, the chick's body temperature dropped. After the air temperature had fallen close to freezing, Williams began gently warming the chamber, expecting the chick's metabolic rate to stay high as it's body temperature recovered. But instead, the dunlin's metabolic rate fell while the chamber began warming the chick. And when Williams tested other species' chicks, they all showed the same response. Ricklefs explains that 'the hysteresis was completely unexpected'.

But chicks rarely experience a gentle temperature drop in their natural environment; as soon as they leave the nest they are at the mercy of the elements, so the team tested how the chicks faired when the temperature plunged to 5°C. This time the birds' metabolic rate rocketed as the chicks reacted to the large temperature gradient at their warm skins. Ricklefs explains that the rapid metabolic increase must be regulated by peripheral thermosensors, detecting the sudden drop in temperature, rather than a single thermosensor situated in the brain.

Ricklefs' original motivation for following the chick's metabolism was to take their metabolic rate as an index for the youngster's muscular development. But now that he's found that the birds' metabolic rate is dependent on their body temperature, measuring a chick's metabolic rate in the field could give a deceptive impression of their maturity. After all, a cold chick that's recently returned from a foraging trip could have a misleadingly low metabolic rate; especially if it's just been snuggling up to mum.

10.1242/jeb.00506

Ricklefs, R. E. and Williams, J. B. (2003). Metabolic responses of shorebird chicks to cold stress: hysteresis of cooling and warming phases. *J. Exp. Biol.* **206**, 2883-2893.

MAPK SIGNALS SALVATION

Just a few moments without oxygen can do terrible damage to a mammal's heart. As the oxygen rushes back, toxic reactive oxygen species cause untold damage to cellular structures. But many amphibians often experience periods of oxygen deprivation, and suffer few ill effects. Isidoros Beis is intrigued by the mechanisms that protect amphibian hearts from these potentially fatal situations. Working with his team in Athens, Greece, he has focused on the cellular stress pathways that are activated by oxidative stress, and in this issue of the *J. Exp. Biol.*, he describes how the oxygen free radical,

hydrogen peroxide, triggers the protective p38-MAPK signalling cascade (p. 2759).

Beis explains that he chose to study *Rana ridibunda* hearts, because he knew that the frogs' hearts respond to certain types of oxidative stress very differently to mammalian hearts; they seem almost immune to oxidative damage after a period of anoxia! But reoxygenating a mammal's heart after a period of ischemia triggers one of the mitogen activated protein kinase (MAPK) signalling pathways; specifically the p38-MAPK pathway, often culminating in cell death and permanent damage to the tissue. When he tested the frog's heart, reoxygenation failed to activate the signalling cascade. Beis explains that shortly before a period of anoxia, the amphibian produces high levels of antioxidant enzymes, ready to mop up any free radicals produced as oxygen returns to the animal's tissues. So, the anoxia-tolerant animal doesn't need to resort to protection from cellular signalling pathways after a bout of anoxia.

But oxidative stress is caused by many factors, other than anoxia. Beis wondered whether these other forms of oxidative stress might activate one of the three major MAPK stress-signalling pathways.

After exposing frog hearts to short pulses of hydrogen peroxide, Beis' team began searching for evidence of stress signalling pathways that had been activated. Both ERK and JNK, two of the MAPKstress signalling pathways, were activated by short exposures to hydrogen peroxide, but the p38-MAPK pathway was activated to a much greater extent, yet didn't seem to trigger tissue damage. Knowing that one form of p38-MAPK stimulated a pathway that protects cells from stress, they decided to follow the signalling cascade to find out which proteins it activated.

By probing tissue extracts with antibodies that recognise proteins downstream of the p38-MAPK signal, the team realised that the pathway ultimately activated a heat shock protein; HSP27. Many heat shock proteins mediate a variety of cellular responses to different environmental stresses by protecting proteins from degradation, but whether HSP27 acts as a chaperone to protect the heart, is not obvious.

Having identified the cascade that is triggered by hydrogen peroxide oxidative damage, Beis is keen to find the antioxidant enzymes that the cascade in turn activates, to protect the animals from oxidative stress.

10.1242/jeb.00508

Gaitanaki, C., Konstantina, S., Chrysa, S. and Beis, I. (2003). Oxidative stress stimulates multiple MAPK signalling pathways and the phosphorylation of the small HSP27 in the perfused amphibian heart. *J. Exp. Biol.* **206**, 2759-2769.

SENSING THE PATTER OF TINY FEET

Watch an insect scuttling along; its legs are almost a blur as they drum against the ground. Each leg's lightning fast movements are controlled by mechanosensors, which detect each footfall, and trigger the neural circuits that control the next footstep. But Reinhold Hustert explains that there is a problem; when ever scientists had calculated the time that it would take for a mechanosensor's nerve signal to travel from the foot to the central nervous system, it was simply too long. Hustert was puzzled by this apparent paradox; he knew that he would have to find a mechanoreceptor (p. 2715) that could send the neural message fast enough to keep the insect scampering.

Insects are covered in thousands of microscopic sensory receptors for detecting the world around them. The receptors range from tiny hair-like structures that fire when something brushes past them, to campaniform sensilla, which are microscopic dome structures that detect when the insect's cuticle deforms as it moves. Hustert realised that the mechanoreceptor responsible for triggering the insect's rapid reflexes must satisfy two conditions. First it must be close to the insect's body, to cut down the distance that the nerve signals travel before reaching the central nervous system (CNS). And secondly, the receptor must have a high conductivity to transmit the signal swiftly. Hustert knew that campaniform sensilla have high conductivity rates, but would he find them close enough to the insect's body?

Markus Höltje began testing locusts' reflexes. He gently probed various sensory organs on the insect's legs, and recording the motoneuron's response in one of the leg's depressor muscles. He also measured the length of time that it took nerve signals to reach the CNS, and found that the signals from most of the leg's receptors took almost 10 ms to travel to the CNS; far too slow. But when he recorded the signal transduction time from the campaniform sensilla near to the body on the leg's trochanter, it was almost 10 times faster, delivering a nerve spike to the CNS in 1 ms. And the superfast signal triggered motoneurons in the locust's depressor muscle.

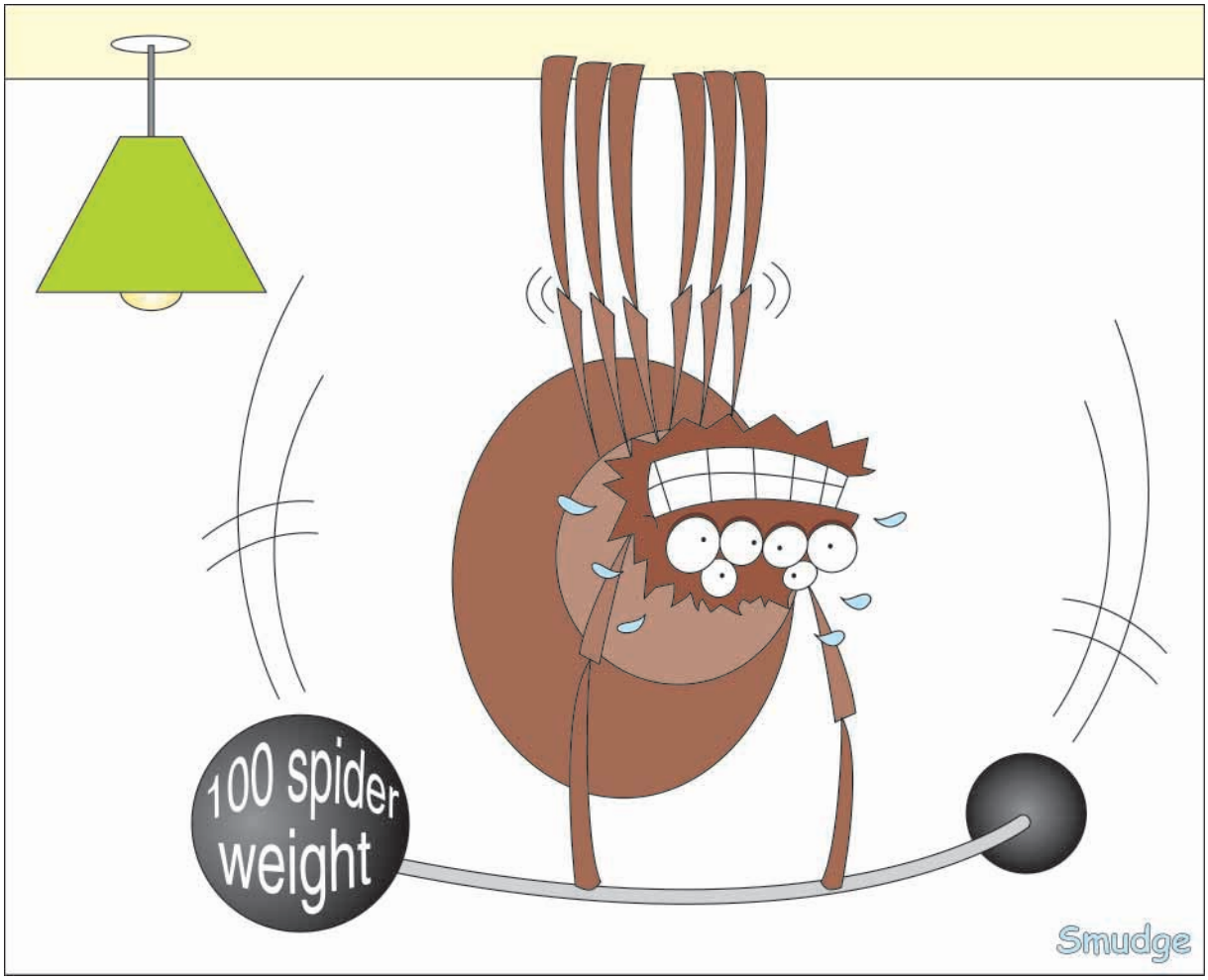
But how does a mechanoreceptor that is so far up the locust's leg detect the footfall? Hustert realised that as the insect's tarsus touches the ground, a tiny shock wave travels up the cuticle of the insect's leg, but at much faster speeds than any nerve signal travels. Knowing that sound travels through wood at 3500 m s^{-1}, Hustert estimates that the foot's impact generates a pressure wave that travels up the leg in 1 ms, which certainly makes the reflex fast enough. But can the receptor detect the footfall's tiny pressure wave? Höltje tested the delicate sensilla's threshold by gently touching the insect's tarsus, and realised that an impact that was fraction of the locust's body weight was enough to trigger the sensor.

Hustert adds that the campaniform sensilla's extreme sensitivity probably makes them extremely versatile sensory receptors. As well as triggering the insect's running reflex, he suspects that they also use the sensilla to detect gravity, helping the insect's to coordinate their movements, no matter what their orientation, as they roam through rough terrain.

10.1242/jeb.00507

Höltje, M. and Hustert, R. (2003). Rapid mechano-sensory pathways code leg impact and elicit very rapid reflexes in locusts. *J. Exp. Biol.* **206**, 2715-2724.

SPIDERS GET ATTACHED



Neil Smith is a cartoonist living in London.

Scampering up rough surfaces is fairly straightforward if you can get a grip, but staying attached to smooth surfaces is much trickier. Some creatures ooze a sticky goo from pads on their feet to adhere themselves to walls and ceilings. Other animals have opted for a drier approach; they have hundreds of thousands of microscopic hair-like structures that bond them to vertical surfaces. Antonia Kesel wondered how these tiny hair-like setules attach an animal to a smooth surface, so she began probing jumping spider's feet to find the origin of their super-adhesion (p. 2733).

Working with her team, she measured the setule's size by electron microscopy, and found that each settule is flattened at one end, to produce a tiny pad. As each jumping spider has well over half a million tiny hairs on its feet, the area soon adds up, but even, so each arachnid only has a microscopic footprint. So how do the miniscule pads attach the spider to a wall? The group probed individual setules with an atomic force microscope, and measured the force necessary to pull the probe away from a setule; almost 40 nN, which adds up to enough sticking power to support an animal that weighs over 2 g. Kesel's spiders only weigh a tiny fraction of that, so the setule's adhesive force anchors them very securely to any surface they choose. Which then poses another question; how arthropods ever pry their feet free, once attached?

10.1242/jeb.00505

Kesel, A. B., Martin, A. and Seidl, T. (2003). Adhesion measurements on the attachment devices of the jumping spider *Evarcha arcuata*. *J. Exp. Biol.* **206**, 2733-2738.

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