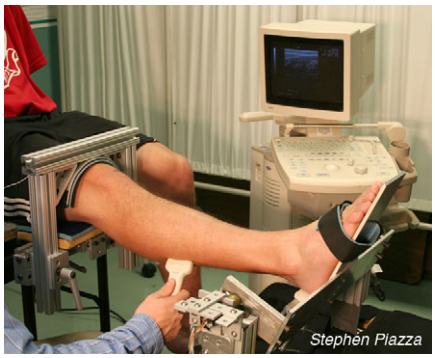
Inside JEB highlights the key developments in *The Journal of Experimental Biology*. Written by science journalists, the short reports give the inside view of the science in JEB.

Inside JEB

SHORT HEELS GIVE ELITE SPRINTERS THE EDGE



When 100 m sprinters launches themselves from the starting blocks, the race can be won or lost in the first few strides. Acceleration through the first few strides is the key to winning gold. So when Stephen Piazza was approached by an American football star, who sprints in his position of wide receiver, to find out how he could improve his technique and training regime, Piazza decided to focus on the athlete's ankles to try to discover what gives elite sprinters the edge over ordinary mortals (p. 3700).

The effectiveness of an accelerating sprinter's push off depends on the amount of leverage that the calf muscles have when pulling on the back of the heel to pull it up as it pushes the toes down, and off the ground. Piazza figured that the athlete's foot would have a large distance from the ankle to the back of the heel to produce a long 'heel lever' for the calf muscle to pull on when pushing the toes down. In this case, the calf muscle would have to contract and pull the heel up over a long distance, so Piazza measured how far the athlete's tendon moved (translated) while pulling the athlete's heel up to see how it compared with that of non-sprinters. Piazza says 'I thought it would be one of the largest (tendon translations]) we had ever measured'. But when he and his student, Sabrina Lee, measured the distance, they were surprised to find that it was much shorter than average. Was the football star the exception or the rule?

Piazza decided to compare the Achilles' tendon translation of elite athletes with that of non-sprinters. Working with sprinters and long jumpers from Lock Haven University, and local non-sprinters, Piazza and Lee used ultrasound imaging to measure the tendon's translation as the subjects pointed their toes. Amazingly, the distance was 25% shorter in the elite athletes than in the non-sprinters. Instead of benefiting from the mechanical advantage of having a long heel lever, the sprinters seemed to be at a mechanical disadvantage because their heel levers were much shorter.

Puzzled by this unexpected discovery, Piazza turned to the literature to find out how animal sprinters' ankles are constructed, and quickly realised that the human elite athletes were built inline with their animal counterparts, which also have short heel levers. So how does this mechanically disadvantageous arrangement give elite sprinters the edge over weekend joggers?

Piazza and Lee realised that a fundamental property of all muscles could be responsible for the sprinters' unexpectedly short Achilles' tendon translations. He explains that muscles that contract quickly cannot generate much force, giving runners with a long moment arm a weak push off despite their increased mechanical advantage. However, muscles that contract slowly produce much greater forces that overcome the mechanical disadvantage of a short heel lever, giving sprinters with a short heel lever a powerful push off.

Testing his theory with a mathematical model of a sprinter's body, it was clear that the extra force generated by the calf muscle as it pulled the short heel lever would provide sprinters with the additional acceleration required to get ahead in the first few strides. And when the duo compared other physical characteristics between the sprinters and non-athletes, they noticed that the sprinters' toes were almost 1 cm longer than those of the non-sprinters. Not only could the sprinter generate more force while accelerating, but their longer toes allowed them to remain in contact with the ground longer during each stride, giving them longer to push against the surface and out perform slower sprinters.

10.1242/jeb.039735

Lee, S. S. M. and Piazza, S. J. (2009). Built for speed: musculoskeletal structure and sprinting ability. *J. Exp. Biol.* **212**, 3700-3707.

GLOWING SHARKS USE HORMONE ON/OFF SWITCHES

Not much sunlight penetrates deep in the ocean, but this does not mean that the depths are completely dark. Julien Claes from the Catholic University of Louvain, Belgium, explains that many deep ocean creatures produce their own light. But while the light production mechanisms of bony fish are quite well understood, almost nothing was known about the way that luminescent elasmobranchs produce light. 'Luminescent sharks live in the deep sea and you need live animals to study luminescence, but it is difficult to keep

them alive at the surface,' explains Claes. However, when Claes came across a paper by a muscle physiologist, Harald Kryvi, describing how he had kept velvet belly lantern sharks alive in a Norwegian fjord, he realised that this could be the breakthrough. In winter, the surface temperature in Norwegian Fjords is the same as that at depth, so the fish survive the ascent. Claes and his supervisor, Jerome Mallefet, headed north to the Raunefjord to catch live velvet belly lantern sharks and test how they regulate their bioluminescence (p. 3684).

Laying long lines on the fjord bottom, Claes and Mallefet routinely caught 30–40 velvet belly sharks on their fishing trips before returning to Bergen where they kept the fish in tanks. According to Claes, the shark's light producing organs (photophores) are much tinier (<150 µm diameter) than bony fish's photophores (up to 1 cm diameter), and the shark can have as many as 2000 cm^{-2} distributed across their bellies and fins. Dissecting 0.55 cm diameter patches of skin from the fish's belly, Claes and Mallefet injected neurotransmitters, such as adrenaline and GABA, into the skin and measured the light produced with a luminometer, to test whether the photophores are controlled by the shark's nervous system, but were unable to stimulate the skin to glow.

Having ruled out neural control, Claes and Mallefet turned to three hormones that are known to regulate skin coloration in sharks: melatonin, prolactin and α-MSH. Injecting melatonin into a skin patch and shutting it inside the luminometer, Claes and Mallefet were amazed to see a perfect luminosity curve plotted on the computer screen. 'It was a fantastic moment,' says Claes, 'just amazing'. And when the duo repeated the experiment with prolactin, the skin glowed again. Both hormones stimulated the skin to glow, with melatonin producing a long weak glow, while prolactin generated a shorter brighter burst of light. And when the duo applied α-MSH to a piece of skin before stimulating it with melatonin, the skin would not glow. α-MSH inactivated the photophores. Claes and Mallefet had found the skin's on and off switches.

But why use slow acting hormones to activate bioluminescence when bony fish use fast acting nerves? Claes explains that sharks probably use bioluminescence for two reasons: camouflage against background light from the surface and communication. As shark melatonin levels depend on light levels in the environment, this could be a perfect mechanism allowing sharks to match their own luminosity with the background – and remain invisible to predators and prey hovering below – while prolactin activates the glowing photophores briefly and rapidly, probably for communication with members of their own species.

10.1242/jeb.039727

Claes, J. M. and Mallefet, J. (2009). Hormonal control of luminescence from lantern shark (*Etmopterus spinax*) photophores. *J. Exp. Biol.* **212**, 3684-3692.

LODGER BUGS' ANTIBIOTICS PROTECT HOOPOE'S FEATHERS

Feathers say a lot about a bird, so males and females invest a lot in protecting their plumage. According to Magdalena Ruiz-Rodríguez from the University of Granada, Spain, the bacteria *Bacillus licheniformis* is a major threat to a hoopoe's plumage, digesting the feather's keratin barbs with an enzyme. And nestlings are particularly at risk from feather damage, harbouring hordes of the bacteria in their filthy hole-nests as their feathers grow prior to fledging. However, another bacteria that is carried by hoopoes, *Enterococcus faecalis*, produces antibiotics against the feather-feasting bugs. Realising that *E. faecalis* hitch a lift in the hoopoe's uropygial gland, which produces secretions that the birds use while preening to protect their feathers, Ruiz-Rodríguez and her colleagues wondered whether the *E. faecalis*' antibiotics may protect the hoopoe's plumage from *B. licheniformis* damage (p. 3621).

Collecting breast feathers from male and female hoopoes, the team first sterilized the feathers then incubated them for a week in mixtures of the bacteria and one of the *E. faecalis* antibiotics to see whether *E. faecalis* and its antibiotic may protect the feathers from degradation. Scrutinizing the feathers with scanning electron microscopy, Ruiz-Rodríguez could see that the the feathers were completely ravaged by *B. licheniformis*, but in preparations where the *B. licheniformis* had been mixed with *E. faecalis*, or its antibiotic, the feathers were almost unharmed. And when the team searched for the tell tale bacterial plaques that signify a *B. licheniformis* infection, they only found the plaques on feathers incubated with *B. licheniformis*. Feathers incubated with *E. faecalis* and *B. licheniformis*, or the antibiotic and *B. licheniformis* were free of the feather degrading bacterial plaques.

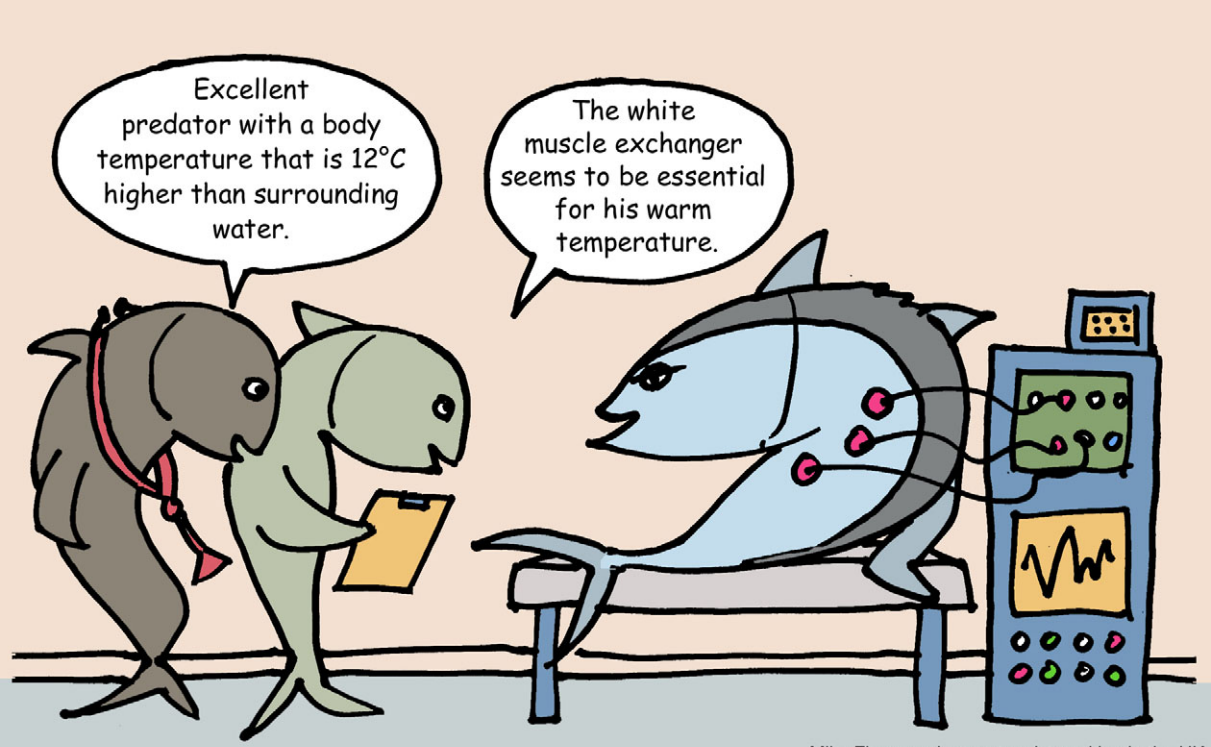
Finally, to confirm that *B. licheniformis* was digesting the feathers' protein, keratin, the team incubated the feathers with the bacteria for 2, 5 and 16 days, and measured the amount of protein that had been released from the feathers. *Enterococcus faecalis* slowed the damage done by *B. licheniformis*, while the antibiotic completely stopped *B. licheniformis* from digesting the feathers.

Ruiz-Rodríguez and her team suspect that the protective power of hoopoe's uropygial gland secretions is due in part to the gland's *E. faecalis* population and the antibiotics that the bacteria produce. And the team back up their suggestion with the observation that both nestlings and incubating females spread the antibacterial uropygial gland secretions through their feathers while sitting in their nests.

10.1242/jeb.039701

Ruiz-Rodríguez, M., Valdivia, E., Soler, J. J., Martín-Vivaldi, M., Martín-Platero, A. M. and Martínez-Bueno, M. (2009). Symbiotic bacteria living in the hoopoe's uropygial gland prevent feather degradation. *J. Exp. Biol.* **212**, 3621-3626.

WHITE MUSCLE KEEPS TUNA WARM



Mike Flanagan is a cartoonist working in the UK

Tuna are one of the top ocean predators. Few fish out run them as they scythe through the oceans. The key to the tuna's place at the pinnacle of the ocean community is their warm muscles. Retaining heat generated by their red muscle through a network of blood vessels, known as retes, which transfer heat from the blood as it leaves the warm internal muscles to cold blood returning from the animal's surface, tuna are able to maintain a body temperature that is 12°C higher than the surrounding water. Most attempts to understand how this super-predator maintains its high body temperature have focused on retention of the heat generated by the fish's red muscle. 'However, the white muscle fibre portions of the myotomes… account for approximately 45 to 55% of the total body mass,' says Hans Malte from the University of Aarhus. Could white muscle be contributing the fish's higher body temperature? Knowing that rete-like structures had been identified in tuna white muscle in the 1960s, Malte and his colleagues Jess Boye, Michael Musyl and Richard Brill decided to build a mathematical model of heat movements in bigeye tuna, to see whether white muscle counter current heat exchangers may contribute to the fish's phenomenal thermal efficiency (p. 3708).

Building models of a tuna fish in an environment that remained at a constant temperature and of another fish that made deep sea excursions to the chilly waters 400 m down, the team found that the only way that the tuna could maintained their body temperature was by recycling 96% of the heat from their white muscle, and 99% from their red muscle. And when they tested the effect of taking away the white muscle heat exchanger and leaving the fish with only its red muscle heat exchanger, the mathematical fish's temperature never reached the 32°C that had been recorded in the wild. The white muscle exchanger seemed to be essential for the fish's warm temperature.

Finally, the team ran a calculation that showed that the fish may actively regulate their body temperatures by adjusting the amount of blood that they send through heat exchanger tissues to warm and cool as they move through the water column.

'Our model shows that the presence of a functional rete in the blood supply to the white muscle is necessary to achieve realistic model outputs,' says Malte and he is keen to find out more about the fish's white muscle heat exchangers to understand how this top predator keeps warm.

10.1242/jeb.039719

Boye, J., Musyl, M., Brill, R. and Malte, H. (2009). Transectional heat transfer in thermoregulating bigeye tuna (*Thunnus obesus*) – a 2D heat flux model. *J. Exp. Biol.* **212**, 3708-3718.

Kathryn Knight
kathryn@biologists.com