Keeping track of the literature isn't easy, so Outside JEB is a monthly feature that reports the most exciting developments in experimental biology. Short articles that have been selected and written by a team of active research scientists highlight the papers that JEB readers can't afford to miss.

Outside JEB


## BOUNCING BITES

In the children's poem *The Ants at the Olympics* by Richard Digance, ant athletes get no respect. According to the poem:

*'At last year's Jungle Olympics,*
*the Ants were completely outclassed.*
*In fact, from an entry of sixty-two teams,*
*the Ants came their usual last…'*

The poor ants don't win any medals because they have to compete against the hippos in the shot put and the cheetahs in the sprints. Too bad there were no events such as 'fastest predatory strike' or 'highest acceleration of an appendage,' because trap-jaw ants would win gold every time.

In a recent paper in *Proceedings of the National Academy of Science*, Sheila Patek and colleagues report the results of a biomechanical study of jaw-closing in trap-jaw ants. They found that these ants use their jaws to carry out some truly remarkable and downright athletic feats.

Trap jaw ants are known for their absurdly large jaws that they use in fast predatory attacks. Patek and colleagues wanted to investigate the jaw-closing event using an ultra-fast video camera, because previous attempts with slower cameras were unable to resolve the entire event. The team was also interested in documenting other lesser-known uses of the jaw-snapping such as fending off intruders and escaping predators.

To do this, Patek and her coworkers carried out a series of experiments with a high speed video camera that could capture $5\times10^4$ frames per second. This allowed them to describe the details of jaw closing, and investigate whether the ants used their explosive jaws to propel themselves and/or intruders, and if so, how.

They found that the jaws close with an average velocity of 38.4 m $s^{-1}$, which gives trap-jaw ants not only the gold medal, but also the world record for the fastest movement generated by any organism. Falcons can drop out of the sky at far greater speeds, but they have gravity on their side. Trap-jaws pull off their feat using only stored internal energy.

Getting the mandibles up to 38.4 m $s^{-1}$ in just 100 ns requires incredible acceleration, in this case, $10^5$ ***g***, which is exceeded only by the discharge of cnidarian nematocysts. The researchers also calculated that closing jaws can exert 47 mN of force, or about 400 times their own body weight. While this kind of force is certainly adequate to incapacitate prey, it is also enough to launch an ant into the air if the force is directed against something massive.

The researchers found that the ants indeed launched themselves in the air when confronted with potential threats, and they did so with two distinct behaviours – one they used to repel intruders (called a 'bouncer defense'), and another that they used to escape predators (called an 'escape jump'). The bouncer defense was instigated by firing the jaws against an intruder, which had the effect of sending it and the ant off in opposite directions. The escape jump was elicited by directing the jaws down at the ground, sending the ant flying in the air, and presumably out of harm's way.

Watching the supplementary online videos for this paper is strangely entertaining. In slow motion, one gets to watch the ants launch themselves into the air, all the while spinning at the impressive speed of 63 rev $s^{-1}$. It's enough to make an Olympic gymnast or figure skater jealous – until you see the landing.

10.1242/jeb.02577

**Patek, S. N., Baio, J. E., Fisher, B. L. and Suarez, A. V.** (2006). Multifunctionality and mechanical origins: Ballistic jaw propulsion in trap-jaw ants. *Proc. Natl. Acad. Sci. USA* **103**, 12787-12792.

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NERVE SIGNALLING

## SWITCHING MAMMALIAN NEURONES ON AND OFF

Recording from neural circuits is the bread-and-butter of neuroscience. As well as simply recording passively from neurones *in vitro* or in the live animal, it is important to impose experimental constraints on the system; for example by electrically stimulating a particular neurone and following the responses of others in the circuit, or by applying drugs to the preparation. Such work is informative, but painstaking and prone to artefact.

Recently, transgenic technologies have provided exciting new technologies for neuronal monitoring and monitoring. My own lab was the first to produce animals that carry transgenic calcium reporters, allowing synaptic transmission to be monitored; and recently, *Drosophila* have been made carrying transgenic potassium channels that act to make neurones less excitable, so shutting them down. Elaine Tan's paper in *Neuron* adds a valuable new technique that allows mammalian neurones to be selectively and reversibly switched off. To do so, the team has drawn on insect endocrinology.

Previous work from the group had shown *in vitro* that mammalian neurones transfected with the gene for an insect allatostatin receptor would stop firing action potentials when allatostatin was added. Allatostatin is a peptide hormone that normally acts to inhibit the production of juvenile hormone, a key hormone that regulates the quality of insect moulting. Critically, it is sufficiently different from any vertebrate hormone that one would not anticipate cross-activation of any vertebrate receptor with the insect neuropeptide. Thus, only those cells in which the allatostatin receptor is expressed should respond to allatostatin. The nature of the response is also critical; it appears that in mammalian neurones, the allatostatin receptor activates a potassium channel that hyperpolarizes (makes more negative) the neuronal membrane. Under such circumstances, the neurone becomes most unlikely to fire when neighbouring cells stimulate it: it is effectively 'switched off'.

However, studying nerve cells in culture introduces artefacts aplenty. Could this technology be replicated *in vivo*? Callaway's group put their gene into a modified adenovirus – a cold-like virus that has become popular for its ability to introduce transgenes into a wide variety of mammalian cells. They coupled the gene with a synapsin (neuronal-specific) promoter, ensuring that, although the adenovirus might infect many cell types, it would only make the allatostatin receptor in neurones. The team also incorporated green fluorescent protein into the virus, so that transfected cells would be fluorescently marked.

In essence, the technique worked exactly as predicted. Working first in rat cortex, then in ferret and monkey, the team were able to show that perfusion with allatostatin shut down neuronal signalling, and wash-out of the allatostatin allowed it to start again.

Although these results provide a valuable tool for non-model organisms, they also reveal some of the advantages of working in a suitable model. For example, it is very hard to introduce a transgene into just a few specific cells with adenovirus. However, the team showed that this was indeed possible in transgenic mouse, allowing them to express the allatostatin receptor in specific populations of spinal neurones and selectively inhibit neuronal firing.

Why is this important? The technology has the potential to allow a more quantitative, 'systems biology' approach to the study of neuronal circuits, although it will require refinement if it is to be useful beyond mouse. Additionally, it allows *in vivo* manipulation of neuronal circuits, so protecting the experimenter from the artefacts of cell culture. It is thus an exemplar of the new 'integrative physiology'.

10.1242/jeb.02576

**Tan, E. M., Yamaguchi, Y., Horwitz, Gosgnach, G. D. S., Lein, E. S., Goulding, M., Albright, T. D. and Callaway, E. M.** (2006). Selective and quickly reversible inactivation of mammalian neurons in vivo using the *Drosophila* allatostatin receptor. *Neuron* **51**, 157-170.

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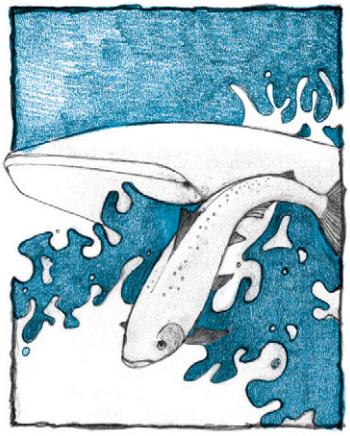
GAS TRANSFER



## BUSTED BENEFIT OF BRADYCARDIA?

When many fish experience environmental hypoxia (a drop in oxygen availability) or hypercarbia (an increase in environmental carbon dioxide levels) their heart rate decreases and their systemic vasculature constricts resulting in an increase in arterial blood pressure. Much previous theoretical and empirical data has lead to the suggestion that these cardiovascular adjustments should enhance gas transfer across the gills, enhancing $O_2$ uptake and $CO_2$ excretion. However, direct measurements of the consequences of a reduced heart rate, known as bradycardia, and increased blood pressure, known as hypertension, on branchial gas transfer in fish gills are extremely sparse, and the few studies that exist have so far yielded conflicting results as to the effects of bradycardia and hypertension on gas exchange. Steven Perry and Patrick Desforges of the Department of Biology at the University of Ottawa decided to rectify this situation by quantifying exactly how reduced heart rate and raised blood pressure affect branchial gas transfer during hypoxia and hypercarbia in the rainbow trout (*Oncorhynchus mykiss*).

The team first monitored heart rate, cardiac output (the amount of blood pumped by the heart), arterial blood pressure and arterial blood $O_2$ and $CO_2$ tensions in untreated rainbow trout exposed to either 40 min of hypoxia or 30 min of hypercarbia. Next, they tested the effects of bradycardia and hypertension on the fishes' physiology during hypoxia or hypercarbia by treating groups of fish either with atropine (to abolish bradycardia) or prazosin (to eliminate hypertension), before exposing the fish to hypoxia or hypercapnia and measuring their cardiovascular and blood gas responses. The expectation was that differences in blood gas tensions between the treated and

untreated fish would indicate if and how bradycardia and hypertension affects branchial gas transfer.

Surprisingly, in contrast with current beliefs, the team found that bradycardia did not enhance branchial gas transfer efficiency in the rainbow trout during hypoxia or hypercarbia. No differences in blood gas $O_2$ or $CO_2$ tensions existed between untreated trout, which exhibited bradycardia, and atropine treated fish, which did not exhibit a decrease in heart rate. The team argues that this lack of difference in blood gas tensions indicates that reducing heart rate during hypoxia or hypercarbia does not have any beneficial effects on gas exchange.

Similarly, the team found no beneficial effect of hypertension on blood gas tensions during exposure to increased environmental $CO_2$. There were no differences in the blood gas levels between untreated fish and hypertension-inhibited fish. Perry and Desforges argue that this suggests that increased blood pressure does not increase gas transfer during hypercarbia.

The team did find that gas transfer across the gills was impaired in hypertension-inhibited fish during hypoxic exposure. Arterial blood $O_2$ tension was lower, and $CO_2$ tension was higher in fish treated with prazosin compared to untreated fish. However, Perry and Desforges suspect that this reduction was not caused by inhibition of the fish's hypertensive response. Rather, they suggest that it was probably caused by prazosin impairing the normal ventilatory responses to hypoxia, resulting in reduced gas transfer across the gills.

Thus, in contrast to current models of gas transfer in fishes, it appears that neither bradycardia nor hypertension enhances branchial gas transfer in rainbow trout during hypoxia or hypercarbia. It remains to be determined whether this finding can be extended to other fish species.

10.1242/jeb.02578

**Perry, S. F. and Desforges, P. R.** (2006). Does bradycardia or hypertension enhance gas transfer in rainbow trout (*Oncorhynchus mykiss*)? *Comp. Biochem. Physiol.* **144A**, 163-172.

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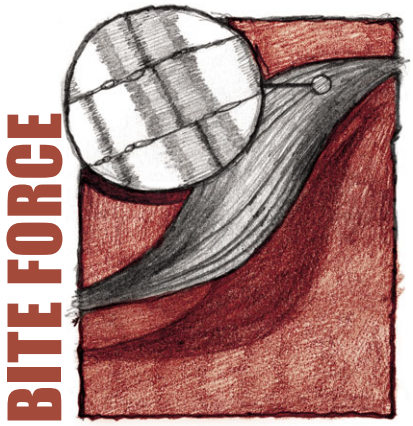


## A NEW TWIST ON UNDERGROUND EATING

Many of the principles behind jaw design are intuitive. For instance, you can form a good impression of a bird's diet from the size and shape of its beak, and everyone knows to keep their fingers away from an angry parrot. However, caecilian head design presents a puzzle: their jaw structure is consistent with high force development, suggesting specialisation for the consumption of hard food items such as seeds or snails, but they are predators that survive on a soft diet of earthworms and chewy subterranean arthropods. As caecilians don't appear to need such impressive jaws for crunching their food, how do these burrowing legless amphibians use this forceful bite?

John Measey and Anthony Herrel tackled this question by studying feeding caecilians with conventional and X-ray video, and direct measurements of bite forces. They confirm that the amphibian's bite forces can indeed be high, reaching 1.4–1.6 times the amphibian's body weight. The team explain that the caecilian's bite is powered with muscles situated *behind* rather than *around* the head, so that the muscles are positioned to produce the maximum bite force while maintaining a streamlined head that is suitable for burrowing through narrow channels.

However, it is the video observations that are the most fascinating and compelling aspect of this study: on biting its prey, a caecilian vigorously spins or corkscrews along its long axis. When applied to an earthworm, this has the effect of ripping it to pieces, and earthworm fragments recovered from caecilian guts look like pieces of twisted rope. Just like a crocodile's 'death roll', the caecilian spin can reduce prey items to swallowable pieces using relatively simple jaws, and without grasping limbs. Also, the amphibian's maximum gape size does not limit the size of prey they can tackle, allowing caecilians to consume relatively large victims without leaving a large bump in the gut that would certainly hinder burrowing.

So these observations begin to make some sense: caecilians can grip large, slippery earthworms using a forceful bite, and then twist them into mouth-sized pieces. However, Measey and Herrel also noticed that caecilians continue spinning even when their meal is almost over, or the prey is small enough to eat whole. Are the caecilians being 'stupid', continuing spinning when the work of dismembering their meal is complete; are they constrained to a stereotyped feeding behaviour? Or are the caecilians 'sizing-up' as well as 'chopping' with their bite-twist behaviour? Given that it is dark underground, Herrel and Measey suggest that it could be difficult for caecilians to gauge their victim's size without giving them a good twist. So the answer is probably a bit of both.

10.1242/jeb.02575

**Measey, G. J. and Herrel, A.** (2006). Rotational feeding in caecilians: putting a spin on the evolution of cranial design. *Biol. Lett.* doi: 10.1098/rsbl.2006.0516

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