Image courtesy of Andre Seale, University of Hawaii.

SENSORY SYSTEMS

Dolphins turn down the volume

Like bats — and submarines — dolphins use echolocation to investigate their environment. Unlike bats and submarines, though, how dolphins use this specialized sensory system is not well understood. A study of dolphins in the wild now reveals that they use a unique gain control system.

One problem faced by any echolocating organism is loss of the signal over distance. An echo from a distant prey animal will be much fainter than an echo from the same animal when it is very close. Bats overcome this problem using a system of auditory gain control — a specialized muscle in their middle ear contracts when they emit an echolocation call, which reduces the sensitivity of the auditory system and prevents the animal from being deafened by the call. Then, as the time from the call increases, the muscle relaxes and the ear gradually becomes more sensitive. The system is calibrated so that the amplitude of the echo signal remains constant regardless of how close the target is, within a certain range. Sonar systems engineered by humans use a similar principle.

Au and Benoit-Bird find that echolocating dolphins also use gain control to maintain a consistent signal — but rather than adjusting their receivers, they vary the strength of their calls with the distance to the target. The net effect is the same — the calls are precisely calibrated so that the returning signal has the same strength regardless of the range to the target.

So dolphins and bats, faced with the same problem in their unusual sensory system, have evolved two very different mechanisms that produce the same outcome. Bats use a neural mechanism that couples muscle contraction with the echolocation call; however, it seems that the dolphin's gain control is a natural result of their system of sound production. As they get closer to a target and the echoes are returned more rapidly, dolphins produce echolocation clicks more quickly; and because of the way the clicks are produced (which relies on pressurization of the nasal system) the amplitude of the clicks will naturally fall as the repetition rate increases. What at first seems like a physiological limitation has been turned into an efficient gain control system.

Rachel Jones

References and links

ORIGINAL RESEARCH PAPER Au, W. W. L. & Benoit-Bird, K. J. Automatic gain control in the echolocation system of dolphins. *Nature* **423**, 861–863 (2003)

IN BRIEF

PROCESS OUTGROWTH

Filopodial calcium transients regulate growth cone motility and guidance through local activation of calpain.

Robles, E. *et al*. *Neuron* **38**, 597–609 (2003)

Calcium transients in filopodia affect growth cone motility, but their downstream effectors are unknown. Here, the authors show that calpain is one of these effectors; it promotes a decrease in tyrosine dephosphorylation, the net effect of which is filopodial stabilization, reduced outgrowth and repulsive turning.

NEUROPHYSIOLOGY

Action potentials in basal and oblique dendrites of rat neocortical pyramidal neurons.

Antic, S. D. *J. Physiol. (Lond.)* 9 May 2003 (doi:10.1113/jphysiol.2002.033746)

The apical dendrites of pyramidal cells markedly modulate backpropagating action potentials, owing to the existence of active conductances. By using voltage-sensitive dyes, the author found this not to be the case for basal and oblique dendrites, which behaved as an integral part of the axosomatic compartment.

SENSORY SYSTEMS

NompC TRP channel required for vertebrate sensory hair cell mechanotransduction

Sidi, S. *et al*. *Science.* 12 June 2003 (doi:10.1126/science.1084370))

The mechanosensitive channel that mediates the excitation of hair cells in the inner ear in response to sound is unknown. Here, the authors identify the zebrafish homologue of the *Drosophila* protein NompC as the missing channel. Reducing NompC expression in zebrafish embryos led to a reduction of electrical responses in hair cells, deafness and loss of balance.

ION CHANNELS

Agonist unbinding from receptor dictates the nature of deactivation kinetics of G protein-gated K^+ channels.

Benians, A. *et al*. *Proc. Natl Acad. Sci. USA* **100**, 6239–6244 (2003)

G protein-gated K^+ channels are activated as a result of agoinst binding to G-protein-coupled receptors (GPCRs), and their deactivation depends on the hydrolysis of GTP by Gα after agonist removal. Here, the authors identify a new deactivation mechanism; for some agonists, the rate of unbinding from the GPCR is the key step in the termination of channel activity.

NEUROGENETICS

Neurons but not glial cells show reciprocal imprinting of sense and antisense transcripts of *Ube3a*.

Yamasaki, K. *et al*. *Hum. Mol. Genet.* **12**, 837–847 (2003)

The authors report that the mouse *Ube3a* gene, which codes for a ubiquitin ligase, is transcribed from both alleles in glia, but only from the maternal allele in neurons. This is the first evidence that genomic imprinting is cell-type specific in the brain.