Research Paper: Moving Towards a New Species of *C. briggsae*?, pp. 189–95

Speciation occurs when two similar species are no longer able to interbreed and create fertile offspring. This diversity is created by an accumulation of genetic barriers over time. In this research paper, Baird and Stonesifer describe one particular genetic barrier between temperate strains and the tropical strain of *Caenorhabditis briggsae*, AF16. The researchers find that crosses between both strains results in a delayed development phenotype stemming from dysgenic interactions between maternal—and zygotic—effect loci.

Commentaries: Fighting Pathogens Using G-proteins, pp. 196–201

Protection from invading microorganisms mean that an animal has to mount a response on many different levels—from an immune response to altering its behavior. The coordination of these behaviors in mammals is a complex process and therefore difficult to study. Anderson and McMullan tackle this by using *C. elegans* as their model. The team addresses their recent findings on G-protein signaling pathways in neurons and epithelial cells that are able to coordinate these behaviors, thus protecting the nematode from pathogenic attack.

Octopamine: A Worm’s Norepinephrine, pp. 202–6

In mammals, noradrenergic signaling plays a role in the perception of pain. Increasing our understanding of this mechanism is important as it could lead to some needed, novel analgesics. *C. elegans* do not synthesize norepinephrine but do produce an analog, octopamine. In this article, Mills et al. discuss their recent research on octopamine and its similarities to noradrenergic signaling in mammals, specifically in the case of nociception.

The Great Pathogenic Escape, pp. 207–11

In order for a pathogen to spread and infect, it must first be able to release itself successfully from inside the host cell. Cell culture studies are often used to study mechanisms allowing for their release, however, Szumowski, Estes and Troemel use *C. elegans* instead as this see-through worm is easily visualized and has intact intestinal epithelial cells—a shortcoming of in vitro systems. The group discusses their findings on the mechanism that *Nematocida parisii* uses for a successful exit from host cells and how this has implications for other common pathogenic exit strategies (Fig. 1).

Silencing by Removal, pp. 212–5

Prone to gene amplifications, infections and transposon activation, nematodes are able to suppress unwanted gene activity with RNA interference and transgene-mediated cosuppression. How these two processes work is generally well understood, however, there appears to be another way in which these processes take their effect. Adamo and La Volpe review their recent findings that aside from their expected mechanisms of gene silencing, both RNA interference and transgene-mediated cosuppression also increase germ-line apoptosis—yet another effective way to eliminate infected cells, protecting the genome across multiple generations.

To Be or Not to Be, pp. 216–20

Autophagy has two seemingly conflicting roles: as a protective mechanism and to cause cell death. Such ambiguousness
leaves us with an unclear understanding of what the role autophagy plays in death vs. survival. In this commentary, Michelet and Legouis use a *C. elegans* Endosomal Sorting Complex Required for Transport (ESCRT) mutant to address this issue. These mutants have a faulty vesicular degradative pathway and therefore accumulate cellular defects that are normally taken care of by this endosomal pathway. The authors describe findings that autophagy is induced in these mutants in a desperate attempt to correct the defects, but are unable to ultimately rescue them. The study provides novel insights on interactions between endosomal and autophagic pathways.

**The Molting Timer, pp. 221–30**

The timing and coordination of specific developmental events in animals has not yet been resolved. However, studying the molting cycle of *C. elegans* should be able to provide some keen insights. Monsalve and Frand expand on their recent findings on LIN-42, a protein related to a circadian clock protein, and its role as part of a molting timer. This timer may offer a solution to the question of how rhythmic and developmental processes are integrated (Fig. 2).