Mini-Review: Degrading Proteins for Healthy Muscle, pp. 141–50

A delicate balance of both protein synthesis and protein degradation governs the maintenance of healthy muscle. The regulation of this balance is not well understood, but studying \textit{C. elegans} has shed some light on this process. In this mini-review, Lehmann et al. discuss genes and mechanisms that underlie the control of protein degradation in the muscle of \textit{C. elegans}. The authors then place these findings in other contexts, such as during development and in disease models.

Commentaries: Only Males Need TRY-5 for Sperm Activation, pp. 151–4

Fertilizing an egg can be a long, grueling process for a sole sperm cell and success often relies on accessory factors that are secreted along side the sperm. Interestingly, while seminal fluid plays a noteworthy part in the fertility of many animals, it is not known whether it holds a similar importance in \textit{C. elegans}. In this commentary, Smith and Stanfield review their recent findings on a serine protease, TRY-5. This protein is necessary for sperm activation in males, and yet does not affect male fertility. This is most likely due to other activating factors found in a receptive hermaphrodite. The authors also present other possible roles for seminal fluid in \textit{C. elegans} and illustrate the use of TRY-5 as marker to study other aspects of mating behavior.

Neurodegeneration via a Novel Form of Ciliopathy, pp. 155–9

In mammals, genetic defects of cilia create a host of grave symptoms as they prevent non-dividing cells from performing vital sensory and motile functions. Ciliary defects may arise from post-translational modifications of microtubules, modifications in which their functions are poorly understood. One known post-translational modification enzyme, CCP1, causes neurodegeneration in mice when mutated. In this commentary, O’Hagan and Barr look at the CCP1 homolog in \textit{C. elegans} and discuss similarities they found between mice and worm (Fig. 1).

Leaky Channels May Aid in Anesthetic Effects, pp. 164–9

For the majority of those that have gone under the knife, anesthetics are a normal and very welcome addition. Interestingly, in the case of volatile anesthetics, we are only too aware of how well they can work and what little wiggle room there is between too much and too little, and yet we still do not know how they exert their effects. In this commentary, Singaram, Morgan and Sedensky use optogenetics in \textit{C. elegans} to shed some light on this mystery. The authors find that volatile anesthetics may be taking their effects through sodium and potassium membrane leak channels.

VAB-23 Commits and Executes Cell Fate, pp. 170–5

Creating a new organ, such as the vulva in \textit{C. elegans}, requires that cells must first commit to a specific fate and then execute this fate. The mechanisms linking these two processes are, however, not well understood in developmental biology. In this commentary, Pellegrino and Hajnal review their findings on a recently identified zinc-finger transcription factor, VAB-23, and how it is able to link cell fate specification and execution in a tightly linked process. The authors also discuss VAB-23’s spatio-temporal role in organogenesis (Fig. 3).

Endocytosis and Trafficking Initiates Necrosis, pp. 176–81

Although cell death is necessary for normal development and for the maintenance of homeostasis, inappropriate cell death...
can lead to many pathological conditions, often severe. While apoptosis is relatively well understood, the molecular basis of necrosis is not. In this commentary, Troulinaki and Tavernarakis review their recent findings resulting from the genetic dissection of neurodegeneration models of *C. elegans*. The authors find a connection between proteins involved in endocytosis and intracellular trafficking, with necrosis. In fact, they find that along with the initiation of necrosis, there is a concordant rise of early and recycling endosomes. The authors further discuss implications that endocytosis and intracellular trafficking may have on necrosis.

**Is the Grass Greener Over There?, pp. 182–6**

It can be a difficult decision for an animal to leave its current patch of food on the off chance that something is better around the corner. What are the molecular mechanisms and neural circuits that prompt an animal to stay or go? In this commentary, Busch and Olofsson aim to address this question using *C. elegans* as their model organism. The authors also focus on the influence that metabolism, oxygen and carbon dioxide have on worm food abandoning behavior (Fig. 4).