When the choice is between eating and mating, the Caenorhabditis elegans male chooses eating, according to the experiments of Gruninger et al. The authors started by looking at the unc-103 gene, which is necessary for appropriate timing of sex-related behaviors. unc-103 encodes an ERG (ether-a-go-go-related gene)-like voltage-gated potassium channel. Using mutations in unc-103 that caused males to protract spicule muscles in the absence of mating cues, the authors discovered a functional link between pharyngeal neurons and muscles involved in feeding and the mating-specific spicule muscles. The mutant phenotype was suppressed by an allele of the worm tropomyosin gene, lev-11, as well as by food deprivation. The authors suggest that pharyngeal neurons monitor tropomyosin-mediated pharyngeal contractions as a measure of nutrient availability. In the presence of normal pharyngeal muscle activity, pharyngeal neuronal activity is attenuated. However, if pharyngeal muscular pumping is abnormal, the neurons upregulate pharyngeal muscle activity and downregulate the spicule muscles. It seems to have worked for the worm.

This week, Lomniczi et al. examined the neuronal–glial signaling network that controls the onset of female puberty, as marked by an increase in hypothalamic luteinizing hormone release hormone (LHRH). The authors report that TACE, the tumor necrosis factor-α converting enzyme, is required for the TGFα–erbB1 signaling in hypothalamic astrocytes. The work focused on the median eminence. This region lacks neuronal cell bodies; rather, it contains astrocytes and modified ependymoglial cells called tanyocytes. In explants of the median eminence, stimulation of glutamate receptors on astrocytes led to LHRH release that was blocked by TACE inhibition. The signaling cascade involved glutamate receptor-mediated calcium influx, protein kinase C-dependent increases in TACE activity, release of TGFα from astrocytes, and activation of erbB1 receptors. Inhibition of TACE activity in the median eminence in vivo delayed the age of first ovulation in female rats, consistent with a role for this signaling cascade in the timing of puberty.

Mutations in the nerve terminal protein α-synuclein can cause autosomal dominant familial Parkinson’s disease (PD). This week, Martin et al. expressed the two human mutations, A53T and A30P, in mice to look for neuronal degeneration and cell death. Expression of the mutant protein was driven by the mouse prion protein promoter. The A53T mice developed axonal swelling as well as somatic changes in the brainstem and spinal cord. Inclusion bodies, which had characteristics of the Lewy bodies observed in PD, were present in cortical neurons as well as spinal motor neurons. The number of motor neurons was reduced in A53T mice and, to a lesser extent, in A30P mice. There was also evidence of mitochondrial damage and apoptotic cell death. Given the susceptibility of motor neurons to injury and death in the A53T mice, this mouse may be useful in studies of motor neuron disease as well as Parkinson’s disease.