Enhance the dauer phenotype of the biosynthetic pathway used by becoming adults. Now, Patel and colleagues have discovered a key enzyme in conditions, larvae enter the dormant diapausal dauer stage instead of becoming adults. This enzyme is involved in the intracellular cholesterol transporters that prevent dauer arrest.

Steroid hormone and insulin/IGF-1-like signalling can intersect to direct development. Discovery of HSD-1-mediated steroid signal alters the subcellular localization of the DAF-16/FOXO transcription factor, a component of the insulin signalling pathway. This important result reveals a novel way in which steroid hormone and insulin/IGF-1-like signalling can interact to direct development.

Worming into steroid and insulin signal intersection

In C. elegans larvae, steroid hormone signalling functions with insulin/IGF-1-like signalling to promote reproductive development and to prevent dauer arrest—in hostile conditions, larvae enter the dormant diapausal dauer stage instead of becoming adults. Now, Patel and colleagues have discovered a key enzyme in the biosynthetic pathway used by C. elegans to make steroid hormones—HSD-1, which is orthologous to vertebrate 3β-hydroxysteroid dehydrogenases (3β-HSDs; see p. 2239). They found HSD-1 by screening for mutations that enhance the dauer phenotype of ncr-1 mutants; NCR-1 and NCR-2 are intracellular cholesterol transporters that prevent dauer arrest. hsd-1; ncr-1 double mutants, they report, fail to inhibit dauer arrest; feeding these worms with certain steroid hormone precursors rescues this defect. They also show that reduction of the HSD-1-mediated steroid signal alters the subcellular localization of the DAF-16/FOXO transcription factor, a component of the insulin signalling pathway. This important result reveals a novel way in which steroid hormone and insulin/IGF-1-like signalling can interact to direct development.

Shh noses into craniofacial development...

Noses come in many shapes and sizes, and some morphologies are marked by characteristic nasal malformations. Facial morphogenesis depends on inductive interactions between cephalic neural crest cells (NCCs, which give rise to the nasal capsule and other head structures) and cephalic epithelia, but which molecules provide the instructive signals? Benouaiche and co-workers now report that sonic hedgehog (Shh) signalling from the foregut endoderm patterns the avian nasal capsule (see p. 2221). The surgical removal in ovo of the most rostral zone of the endoderm (EZ-I), they report, prevents the formation of mesethmoid cartilage (a ventral part of the nasal capsule that forms the upper beak), but this defect can be rescued by the implantation of Shh-loaded beads. Correspondingly, when the authors grafted an extra EZ-I into developing embryos, an ectopic mesethmoid formed, the development of which they inhibited by suppressing Shh signalling. These results support the notion that early endodermal regionalization drives normal facial morphogenesis and suggest that its disruption might result in craniofacial defects.

Wnt signal transduction via Src kinases in developing CNS

During nervous system development, axons are guided by many attractive and repulsive cues. For example, members of the RYK/Derailed family of inactive receptor tyrosine kinases guide axons in the Drosophila ventral nerve cord and in the mammalian brain by acting as Wnt receptors. On p. 2277, Wouda et al. reveal how these kinase-inactive RYKs might transduce Wnt signals by reporting that Wnt5-mediated signalling through Derailed in the Drosophila embryonic CNS involves the non-receptor Src family tyrosine kinases Src64B and Src42A. Src64B/Src42A double mutants, they show, have defects in the formation of the nerve fibre tracts that connect the two sides of the brain (commissures) similar to those seen in Wnt5 and derailed mutants. Derailed and Src64B, they report, form a complex, the formation and/or stability of which requires SRC64B activity. Furthermore, the mammalian orthologues of these proteins also form complexes together. Thus, Src family kinases might play novel roles in Wnt5/Derailed signalling during CNS development in flies and in mammals.

A tail of axial progenitors

The vertebrate tail bud is thought to contain multipotent progenitor cell populations that generate the embryo’s axial structures (the neural tube, notochord and paraxial mesoderm). Now, by grafting tissue from a new transgenic chick line in which all embryonic cells express GFP into unlabelled embryos, McGrew and colleagues identify three progenitor cell populations in the avian tail bud (see p. 2289). Cells from the embryonic chordoneural hinge, they report, contribute descendants to all of these axial structures, whereas cells from the dorsoposterior tail bud yield mesodermal tissue only. Both these populations are likely to be ‘long-term axial progenitors’ because they are retained in the tail bud after serial transplantation. By contrast, a ventral tail bud cell population, which also generates paraxial mesoderm, is not retained after serial transplantation. Finally, by showing that transplantation of tail bud progenitor cells into earlier embryos resets their Hox expression (which determines the anteroposterior identity of axial cells), the researchers challenge the idea that Hox identity is fixed during gastrulation.