controls pattern formation in the newly formed tissues? According to Cebrià and Newmark, during anterior regeneration in the planarian Schmidtea mediterranea, the answer may involve the nervous system (see p. 833). The proper rewiring of the nervous system is a crucial event in regeneration, so the researchers began their study by identifying a planarian ortholog of the axon guidance receptor roundabout (Smed-roboA). Unexpectedly, RNAi knockdown of Smed-roboA led to the development of an extra pharynx (the worm’s feeding organ) and to ectopic head structures during anterior regeneration. The researchers report that the regenerating brain in these animals did not re-establish proper connections with the ventral nerve cords and that this defect preceded the development of ectopic structures. They therefore propose that, as in annelids and amphibians, the nervous system may be an important source of the signals needed for proper morphogenesis during planarian regeneration.

New interactions WAVE plants along

In plants and animals, the shape of many cells is controlled by the actin-nucleating ARP2-ARP3 complex, which itself is regulated by a multimeric complex that includes SCAR/WAVE (suppressor of cAMP receptor/Wiskott-Aldrich syndrome protein-family verproline-homologous protein). Now, on p. 967, Martin Hülskamp and colleagues provide new details about cell-shape control in plants. In Arabidopsis, they report, SCAR proteins seem to be direct effectors of small Rac-like GTPases called ROPs (Rho proteins of plants); by contrast, in animal cells, SCAR activity is indirectly regulated by RAC 1. The researchers show that AtSCAR2 (one of five Arabidopsis SCAR homologues) activates the ARP2-ARP3 complex in vitro and that plants mutant for AtSCAR2 have a similar phenotype to those mutant for ARP2 or ARP3. They then used yeast two-hybrid analysis and bimolecular fluorescence complementation to construct a protein-interaction network between the ROPs, the SCAR/WAVE complex and the ARP2-ARP3 complex. This network confirms many of the protein interactions previously identified in animals, but, note the researchers, it also reveals several new interactions.

APC doubles up to regulate female meiosis

Meiosis, the specialised two-stage cell division that produces eggs and sperm, involves a significant reorganisation of the canonical cell-cycle machinery. Swan and Schüpbach have been studying this important developmental change to this machinery and now report that two anaphase-promoting complexes – APC\textsuperscript{cy} and APC\textsuperscript{Cort} – cooperate during female meiosis in Drosophila (see p. 891). During mitosis, the E3 ubiquitin ligase APC and the adaptor protein Fzy target cyclins for destruction during anaphase. By examining various single and double mutants, the researchers reveal that Cort, a diverged Fzy homologue expressed in the female germline of Drosophila, functions with Fzy to drive anaphase in meiosis I and II. Both adaptors, they show, control global cyclin destruction but also the local destruction of spindle-associated cyclin B during meiosis II. The researchers suggest, therefore, that during female meiosis in Drosophila, the germline-specific APC\textsuperscript{Cort} cooperates with the more general APC\textsuperscript{cy} to target cyclins for destruction and allow progression through the two meiotic divisions.

Notch: an angiogenesis off switch

Notch signalling through the ligand Delta-like-4 (Dll4) is essential for normal vascular development, but which aspect of endothelial cell behaviour does this signalling pathway control? Leslie et al. now show for the first time that, in zebrafish embryos,Dll4-Notch signalling tells endothelial cells to stop migrating and proliferating (behaviours that form new sprouts on existing vessels) once a vascular circuit has been completed (see p. 839). The researchers report that, although blood vessel formation starts normally in embryos in which Dll4 production has been blocked with a morpholino antisense oligonucleotide, the embryos develop a network of aberrant interconnected branches unless vascular endothelial growth factor (VEGF) signalling is also blocked. Ectopic activation of Notch, by contrast, prevents endothelial sprouts forming. The researchers conclude that Notch signalling acts as an angiogenic ‘off’ switch in endothelial cells exposed to VEGF. Thus, given the recent demonstration that Dll4 blockade decreases tumour growth in mice by promoting non-productive angiogenesis, targeting Dll4 could provide a new way to treat cancer.