Converging on extension

Growth factors such as fibroblast-growth factor (FGF) or transforming-growth factor-β act during vertebrate embryogenesis to control cell growth, differentiation and morphogenesis. Cell movements and rearrangements continually occur during the complex reorganization of the developing embryo — for example convergent extension in a *Xenopus laevis* embryo leads to polarization of the mesoderm and intercalation of the mesodermal cells to elongate the embryo along the anterior–posterior axis. The FGF pathway is probably involved in this process, together with other pathways such as Wnt signalling, as blocking FGF signalling leads to a truncated anterior–posterior axis in the developing embryo.

In another model organism, *Drosophila melanogaster*, Sprouty, a membrane-anchored general inhibitor of receptor tyrosine kinases, inhibits the FGF-signalling pathway. This is an interesting connection as FGF signalling also acts to increase Sprouty expression. Nutt and colleagues have now cloned a *Xenopus* Sprouty homologue, *Xsprouty2* (*Xspry2*) (*Genes Dev.*, **15,** 1152–1166; 2001) which is expressed in embryos in a similar pattern to FGF. From overexpressing an Xsprouty2 construct, it appears that Xsprouty2 is also involved in axis formation during development, as overexpression of Xsprouty2 in *Xenopus* embryos leads to a shortened anterior–posterior axis (see picture). In particular, overexpression of Xsprouty2 inhibits convergent extension. Nutt and co-workers found that overexpression of Xsprouty2 inhibits the release of calcium from intracellular stores in a way that depends on the FGF receptor.

The role of calcium in convergent extension is poorly understood, but Wallingford *et al.* (*Curr. Biol.*, **11,** 652–661; 2001) have recently shown that these morphogenetic changes are accompanied by marked alterations in calcium dynamics. As cells undergo convergent extension in *Xenopus* explants, there are waves of calcium mobilization from internal stores. These waves lead to contraction of the embryo and are essential for development, as depletion of the calcium stores inhibits convergent extension.

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Both papers offer insight into how cell intercalation and movement might be involved in development. Calcium waves could be a common mechanism by which organisms control large cell movements. Although we do not yet fully understand how these movements are regulated, the cloning of *Xsprouty2* and the link between this and calcium release indicates that thoughts on the process are extending as well as converging.

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structure at the anastral-spindle poles. Microtubule organization provides no evidence for a distinct structure at the poles[8,13], but Msps and D-TACC co-localize in discrete centrosome-like bodies at the ends of these spindles. These structures do not appear to organize or nucleate microtubules, but may stabilize the spindle poles.

Consistent with a stabilizing function for Msps and D-TACC, *msps*- and *d-tacc*-mutant oocytes often contain tripolar meiotic spindles. Furthermore, *d-tacc* mutations block Msps localization to the centrosome-like structures at the tips of the anastral-spindle poles. Mutations in *msps* have a less severe effect on D-TACC localization, suggesting that D-TACC can associate with the spindle poles in the absence of Msps. In *msps* mutants, however, the presence of D-TACC is not sufficient to promote completely normal bipolar-spindle formation. The D-TACC–Msps complex thus seems to be critical for efficient spindle-pole formation or stability. The variable spindle defects observed in *d-tacc* and *msps* mutants are highly reminiscent of the cytological variability observed in the absence of Ncd, and indeed, Ncd is required to recruit Msps to the anastral-spindle poles[3]. Ncd broadly localizes to the anastral-spindle microtubules, and does not associate with the polar structure containing Msps and D-TACC. These observations suggest a model in which Ncd transports Msps to the poles, where Msps associates with D-TACC to form a fully functional stabilizing structure (Fig. 1). As D-TACC associates with spindle poles in *ncd* and *msps* mutants, this protein may provide the partially redundant spindle-stabilizing function inferred from analysis of *ncd*-null mutations.

Does this motor–MAP complex provide a similar function during astral-spindle assembly? Null mutations in *ncd* are viable and do not lead to severe mitotic defects, so this component seems to be specific to the anastral-spindle assembly pathway or may provide a redundant function during mitosis. Strong alleles of *msps* are lethal, however, and lead to severe mitotic defects, including multipolar and completely disorganized spindles, in somatic cells[10]. Although lethal alleles of *d-tacc* have not been identified, none of the existing alleles are functionally null; complete loss of *d-tacc* function could prove to disrupt mitosis in somatic cells. Consistent with this speculation, existing strong alleles of *d-tacc* allow development to the adult stage, but the resulting homozygous females produce eggs that do not hatch and that show mitotic defects that include spindles with broad poles and a reduced number of astral microtubules. Furthermore, Lee *et al.* showed that D-TACC and Msps form a complex in mitotic cells, and that D-TACC is required to recruit normal amounts of Msps to conventional centrosomes at the poles of astral spindles[4]. These authors also found that the mammalian homologues of D-TACC and Msps (TACC3 and ch-Tog) co-localize to the centrosome and co-immunoprecipitate. D-TACC and Msps thus seem to form an evolutionarily conserved complex with important functions during astral- and anastral-spindle assembly.

The precise role of the D-TACC–Msps complex during mitosis remains obscure. Many of the mitotic spindles in *d-tacc* mutant embryos have reduced astral microtubules and seem shorter than wild-type controls, and overexpression of D-TACC leads to an increase in both astral microtubules and Msps localization to the spindle. These observations indicate that the D-TACC–Msps complex stabilizes microtubules during mitosis[4]. This hypothesis is consistent with *in vitro* studies, indicating that the *Xenopus* homologue of