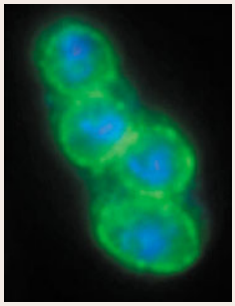
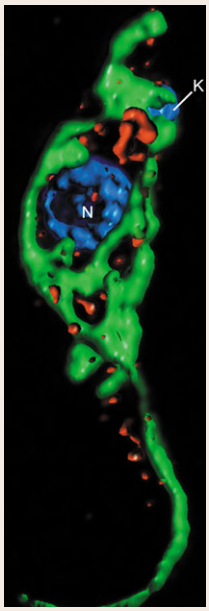
In this issue



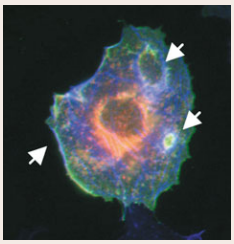
Yeast spores heed SINful message

During mitosis in fission yeast, a septum forms to separate the newly replicated copies of the genome. Formation of this structure, which is coordinated with the nuclear cycle, is triggered by the septation initiation network (SIN). When starved, fission yeast change to meiotic division to form spores. Here, a forespore membrane, which is organized by the spindle pole body, separates the four haploid nuclei rather than a septum. Viesturs Simanis and colleagues now report that the SIN is also required for forespore membrane formation and subsequent spore formation (see p. 2882). The authors show that meiosis is normal in SIN mutants but, although forespore formation is initiated, the spores do not encapsulate properly. In addition, they report that SIN components localize to the spindle pole body during meiosis as they do during mitosis; all the components necessary for SIN activation are not present until meiosis II, when the forespore membrane begins to form. Thus, the authors conclude, the important compartmentalization processes that occur during mitosis and meiosis are both regulated by SIN signalling.



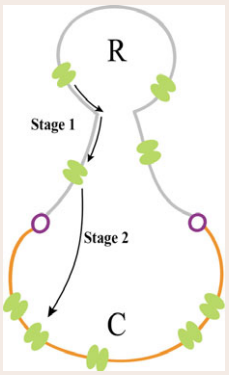
Dynamin-like protein rules and divides trypanosomes

Dynamins, a family of large modular GTPases, are required for endocytosis whereas dynamin-like proteins (DLPs) generally function in the division of mitochondria and other organelles. *Trypanosoma brucei*, however, has a single DLP (TbDLP) and no dynamins – so what is the role of the DLP in this parasite? On p. 2968, André Schneider and co-authors report that TbDLP is required for mitochondrial fission, endocytosis and, unexpectedly, the completion of cytokinesis. Previous work indicated that TbDLP is involved in mitochondrial fission but not endocytosis. Schneider and colleagues, however, demonstrate that ablation of TbDLP function by RNAi or expression of a dominant-negative mutant abolishes both these processes. Moreover, they show that TbDLP localizes to both the single mitochondrion of *T. brucei* and the flagellar pocket, the site where endocytosis occurs in this organism. The authors' final discovery that ablation of TbDLP induces a precise cell-cycle block leads them to suggest that mitochondrial fission is a key event that is monitored by the cytokinesis checkpoint in *T. brucei*.



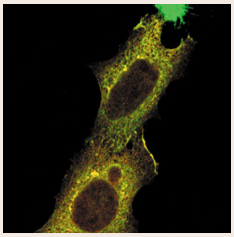
PDGF signalling: two forms of Src'asm

Src family kinases (SFKs) regulate both mitogenesis and morphological changes induced by platelet-derived growth factor (PDGF). How PDGF uses SFKs to transmit these two very different signals is largely unknown. Now, however, Christine Benistant and colleagues report that two distinct pools of SFKs control PDGF-induced responses in mouse fibroblasts (see p. 2921). The authors first show that caveolae (cholesterol-enriched membrane microdomains) are required for SFK-mediated mitogenic signalling and that caveolae-enriched subcellular membranes regulate the formation of PDGF-receptor–SFK complexes. Then, they identify a second pool of PDGF-activated SFKs, whose activity, unlike that of the caveolae-associated pool, is insensitive to cholesterol depletion. This pool of activated SFKs is required for induction of dorsal ruffles and is regulated by a pathway involving phospholipase Cγ, sphingosine 1-phosphate and the G protein G_i; none of the components of this pathway is involved in the mitogenic response to PDGF. Thus, the authors suggest, PDGF stimulates two spatially distinct pools of SFKs to control DNA synthesis and dorsal ruffle formation.



How TRPL channels see the light

Signalling proteins are often segregated into specialized intracellular domains to ensure efficient signal transduction. In *Drosophila* photoreceptors, transient receptor potential-like (TRPL) channels are localized to the rhabdomere (a microvillus-rich part of the apical membrane that houses most of the light signalling components) and translocate to the basolateral membrane during long-term light adaptation. On p. 2935, Susan Tsunoda and co-authors reveal that this translocation involves two steps. Within 5 minutes of light exposure, TRPL channels begin to move into the stalk membrane by a phospholipase-C-dependent process. This is part of the apical membrane; so the channels may diffuse laterally to their new position, possibly after release from an anchoring mechanism involving visual arrestin 2, which holds TRPL in the rhabdomere. In the second step, which takes >6 hours, the channels move to the basolateral membrane. This step depends on the other light-activated channel, TRP, and an eye-specific protein kinase C. Because adherens junctions separate the basolateral and stalk membranes, the authors suggest it may involve transport of the channels in vesicles.



Myotubularins pick more partners

Myotubularins (MTMs) specifically hydrolyse phosphatidylinositol 3-phosphate (PtdIns3*P*) and phosphatidylinositol (3,5)-bisphosphate [PtdIns(3,5)P_2], two lipid regulators of endosomal trafficking. Heteromeric interactions between MTMs are thought to be important but are poorly characterized. Michael Clague and colleagues have therefore performed a systematic analysis of possible MTM interactions (see p. 2953). They confirm previously identified interactions and identify two new ones: MTMR8 interacts with MTMR9; and MTMR3 interacts with MTMR4. The second of these provides the first example of a heteromeric interaction between enzymatically active MTMs – there are 14 MTMs but only eight are catalytically active. Although much attention has focused on possible endosomal functions for MTMs, the authors show that only MTMR4 localizes to endosomal structures. Furthermore, although all the catalytically active MTMs that they test hydrolyse the endosomal pool of PtdIns3*P* when overexpressed, most MTM constructs have little effect on EGF receptor trafficking. A catalytically inactive form of MTMR4, however, severely impedes EGF receptor downregulation. These results suggest that MTMR4 is involved in endosomal function and provide a general framework for further investigation of this family of lipid phosphatases.

Development in press

Neural map making revisited

To transmit unbroken images, retinal axons must terminate on their target brain region in the correct relative positions to form a retinotopic map. In a paper published in *Development*, David Willshaw presents a new computationally generated model for retinotopic map formation, using data from mouse EphA receptor knockin and knockout experiments. Neural map formation is thought to involve two steps: an activity-independent step, which uses position-specific molecular labels to establish a crude map of where retinal axons should migrate, and an activity-dependent mechanism, which refines the map. From his analysis of experimental data, Willshaw concludes that the guiding principle behind retinotopic mapping is that axons carrying similar amounts of Eph receptor terminate near each other on their target and activity-based mechanisms only function later in development. He shows that the 30-year-old marker induction model (in which fixed retinal labels induce labels on tectal cells) can simulate EphA receptor knockin and knockout experiments. Finally, he proposes a refined model – the 'retinal induction model' – in which the retinal and tectal labels are Ephs and ephrin ligands, respectively.

Willshaw, D. (2006). Analysis of mouse EphA knockins and knockouts suggests that retinal axons programme target cells to form ordered retinotopic maps. *Development* **133**, 2705-2717.