structures with several poles in the middle result. Immunofluorescence microscopy reveals that on microtubules in normal spindles Eg5 seems to be more concentrated towards the pole. Moreover, in a motility assay, Eg5 functions as a plus-end motor, moving microtubules over glass with their minus ends leading at about 0.03 $\mu m\ s^{-1}$ (that is with the same polarity as kinesin, but ten times slower). Sawin *et al.* propose that the Eg5 motor protein could attach to a matrix near the spindle pole, generating the forces that could contribute to spindle pole organization and generate poleward tubulin flux (see Fig. 4*c* of ref. 3). Interestingly enough, the proposed attachment of a plus-end directed microtubule motor to a spindle matrix was predicted by Pickett-Heaps[7] from his cytological studies of mitosis, particularly in diatoms.

Before anaphase, Eg5 may help to maintain the organization of the assembled spindle by exerting pole-directed forces on antiparallel microtubules, as an opposing force pulls the poles together. Similarly, in the yeast *Saccharomyces cerevisiae*, Saunders and Hoyt have reported that the kinesin-like protein Kar3 may function as such a motor, pulling the spindle pole bodies together and thus counterbalancing the activity of yeast Eg5 analogues, encoded by the *KLP1* and *CIN8* genes (ref. 8; see also ref. 9).

Two of the other groups with papers in this issue[2,4] used similar approaches to show that kinesin-like proteins play important roles in mitosis. In both cases monoclonal antibodies that interfere with normal spindle functions after microinjection into cultured cells[10,11] were used to isolate cDNAs encoding polypeptides of the corresponding motor proteins. It turns out that both CENP-E (described by Yen *et al.*[2]) and MKLP1 (cloned by Nislow *et al.*[4]) are members of the kinesin superfamily with a characteristic motor domain (up to 500 amino acids long) attached to a coiled-coil α-helical extension — which, in the case of CENP-E, is enormous, almost four times longer than the stalk of conventional kinesin. Immunofluorescent microscopy showed that CENP-E is bound to kinetochores during prometaphase and metaphase, but at anaphase it translocates to associate with fibres of the spindle midzone. MKLP1, however, is never found on kinetochores but is restricted to the interzone of the anaphase spindle.

A remarkable feature of the work by Nislow *et al.*[4] was the use of a modification of an *in vitro* motility assay to demonstrate that recombinant MKLP1 drives antiparallel microtubule–microtubule sliding. Geometrically, this movement *in vitro* is equivalent to the microtubule–microtubule sliding that underlies anaphase spindle elongation[12], suggesting that MKLP1 may drive anaphase B. It is however possible that MKLP1 is involved earlier on in the formation of the metaphase spindle, because microinjection of the antibody CHO1 directed against MKLP1 leads to arrest of the cell cycle in prometaphase[11].

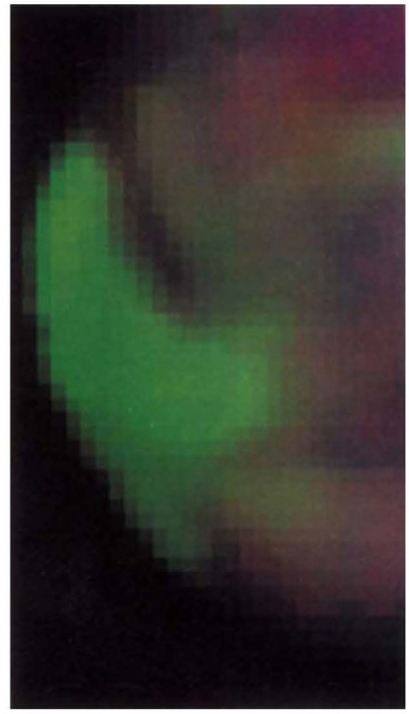
What regulates the association of these mitotic motors with spindle components? The motor protein CENP-E is degraded upon completion of mitosis and is not resynthesized until late in the next interphase; thus its presence in cells is restricted to M-phase, when it is needed. The predicted sequence of the MKLP1 polypeptide, on the other hand, contains a nuclear localization signal which seems to target the motor to the interphase nucleus, allowing contact with microtubules only during mitosis following breakdown of the nuclear envelope. Further details of regulation should also prove to be illuminating; what, for example, controls the striking translocation of CENP-E from kinetochores to fibres of the midzone during anaphase?

A comprehensive analysis of motor functions in the kinetochore will require the reconstruction of a functional complex from individual components. An important step in this direction has been taken by Hyman, Middleton and coworkers[1] who report that an isolated yeast protein complex that specifically binds to centromeric DNA[13] contains the motor activity. This activity moves latex beads with the attached centromeric DNA along microtubules towards their minus ends, at speeds close to the rate of anaphase chromosome movement in animal cells. The specificity of the isolation procedure was beautifully demonstrated by showing that the motor complex does not move the beads containing mutant centromeric DNA. The identity of the polypeptide(s) within this complex responsible for the motion is not yet known, but the combination of biochemistry and *in vitro* motility assays, and the powerful genetics of *S. cerevisiae*, promises rapid progress.

We can now see that motor proteins,

Venus night lights

THE night sky on Venus is not so dark. In common with other planets, Venus is bathed in a pale glow of infrared light resulting from photochemical reactions in its upper atmosphere. On page 516 of this issue, D. Allen and colleagues present high-resolution images of the nightside airglow over Venus, which they use to track atmospheric circulation. The connection is that the airglow is the product of a giant photochemical reaction vessel encompassing the venusian mesosphere. Carbon dioxide on the Sunwards side of the planet (97 per cent of the atmosphere is CO_2) is heated and photolysed to generate oxygen atoms. These are carried by high-altitude winds to the nightside, where the cooled air descends. With increasing pressure, three-body reactions between oxygen atoms and additional species allow molecular oxygen to form, but in an electronically excited state. It is at this point that the airglow starts, as the molecules radiate their excess energy (green in the image). On its low-altitude return trip, the oxygen reacts with carbon monoxide to regenerate CO_2. By monitoring the airglow when Venus's nightside faced towards us, Allen *et al.* found dramatic changes in the local wind conditions over periods as short as an hour — the beginnings of a meteorology for the planet's upper atmosphere. R.P.



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