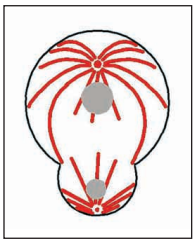
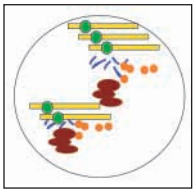


Asymmetric cell division

Asymmetric division allows cells in a developing organism to generate progeny that have distinct fates. The daughter cells can inherit different cell fate determinants and/or be of different sizes - both of which can determine the developmental pathway that is subsequently followed. Asymmetric cell division is brought about by cytoskeletal rearrangements that direct the positioning of the mitotic spindle and the cleavage plane. In a Commentary on p. 2257, Julia Kaltschmidt and Andrea Brand discuss studies that are revealing how this is controlled. Several different mechanisms appear to exist. Asymmetric division of the *C. elegans* zygote, for instance, is due to repositioning of the mitotic spindle by polarity factors such as PAR-2 and PAR-3, which generate asymmetry in the net forces on spindle poles by regulating microtubule dynamics. In *Drosophila* neuroblasts, by contrast, the spindle is asymmetric rather than simply repositioned, and this asymmetry is induced by a G protein signalling pathway involving the protein Inscuteable and its partner, Pins.

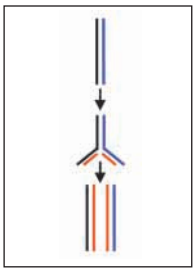


Nucleolar assembly

Like the nucleus itself, the nucleolus - the cell's ribosome factory - is dismantled in preparation for cell division. Its reassembly begins in telophase, and the compartment is fully formed by early G1 phase. What drives this process, and how is it regulated? In a Commentary on p. 2265, Danièle Hernandez-Verdun and co-workers review work that has shed light on the mechanics of nucleolar assembly and its coordination with the cell cycle. Studies some years ago suggested that assembly is driven by activation of the Pol I transcriptional machinery, the rRNA-processing machinery being recruited by the pre-ribosomal RNAs (pre-rRNAs) transcribed. More recent experiments, however, indicate that things are more complex. For example, inhibition of Pol I during the assembly process has revealed that partly processed rRNAs remain during mitosis and recruit processing components such as fibrillarin to forming nucleoli independently of Pol I transcription. Furthermore, whereas Pol I transcription is regulated by the mitotic kinase Cdc2 (CDK1), recruitment of the processing machinery seems to depend on another, unidentified, CDK.

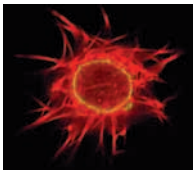
Novel translational control mechanism for BiP

BiP is a chaperone that prevents misfolding of proteins in the ER. The protein is also a homeostatic sensor in the unfolded-protein response (UPR), a signalling pathway activated by ER stress. Its levels must therefore be tightly controlled to avoid inappropriate UPR activation. Speculating that post-transcriptional mechanisms regulate BiP, Ingrid Haas and co-workers have used a clever trick to discriminate regulation of BiP mRNA and protein levels. They introduced a mouse *BiP* gene under the control of a tetracyclin-sensitive promoter into human cells and compared the levels of human and mouse BiP protein and mRNA under different conditions (see p. 2443). They show that, after removal of tetracyclin, the artificially elevated levels of BiP mRNA do not increase the level of BiP protein in unstressed cells. Under ER stress, by contrast, BiP protein levels do rise, which the authors demonstrate is due to increased translational efficiency. Since the effects are independent of the 5´ and 3´ UTRs of BiP mRNA, Haas and co-workers conclude that a novel translational feedback mechanism is involved.



DNA template segregation in stem cells

The genomes of small-intestinal stem cells appear to be extremely well protected: despite dividing thousands of times, they do not lose their proliferative potential and rarely acquire oncogenic mutations. Why are they so resistant to replication-induced errors? One hypothesis is that, when each stem cell divides, template DNA strands are segregated into the stem cell daughter whereas newly synthesized DNA enters the transit cell daughter - replication-induced errors would thus be passed on to cells destined to differentiate rather than to those that propagate indefinitely. This hypothesis has proven extremely difficult to test, but Chris Potten and co-workers have now performed the definitive experiment by sequentially labelling stem cell DNA with tritiated thymidine (^{3}H) and bromodeoxyuridine (see p. 2381). They demonstrate that ^{3}H-labelled small-intestinal stem cells can be labelled with bromodeoxyuridine but that, whereas the ^{3}H is retained in stem cells, the bromodeoxyuridine is lost after a second round of replication. This indicates that stem cells can indeed segregate template (^{3}H-labelled) and newly synthesized (bromodeoxyuridine-labelled) DNA strands. How they do so is anybody's guess.



Microtubule nucleation in plants

Microtubule nucleation in yeast and animal cells is initiated by γ-tubulin ring complexes (γ-TuRCs) associated with centrosomes or spindle pole bodies. Microtubule nucleation in plants is less well understood: the microtubule arrays differ significantly from those in animals and lack centrosome-like microtubule-organizing centres (MTOCs). Moreover, γ-tubulin is distributed along plant microtubules rather than at one end, which is perplexing given its role in nucleation in animals and yeast. Anne-Catherine Schmit and co-workers now provide evidence that plants do in fact use γ-TuRC-like structures to nucleate microtubules (see p. 2423). They have cloned and characterized rice and *Arabidopsis* orthologues of the γ-TuRC component SPC98, demonstrating that higher plants contain γ-TuRC components other than γ-tubulin. Significantly, SPC98 does not colocalize with γ-tubulin along the length of plant microtubules but does colocalize with it at the nuclear surface and cell cortex - locations for putative plant MTOCs. These findings suggest that γ-TuRCs containing SPC98 and γ-tubulin function in microtubule nucleation at plant MTOCs but that γ-tubulin decorating the length of microtubules has an additional, γ-TuRC-independent function.

Sticky Wicket - postdoctoral training

Graduate training usually includes an initial assessment early on and a written thesis and oral defence at the end. The only milestones that characterize postdoctoral training, however, are publications and fellowship/job applications. Caveman believes that this does not constitute a good training programme. On p. 2253, he argues that more effort should be made to assess post-docs periodically so that they have an idea of how far they have progressed.

In the next issue of JCS