Nanomaterials

Jagged edge

It's highly debatable whether these sharp-toothed ribbons will ever be put to use as blades for nanoscale saws. But the wonder is that they form at all. Made from semiconducting cadmium selenide (CdSe), they have one edge that is atomically smooth whereas the other traces out a sawtooth shape reminiscent of a graph of random noise.

These structures, just a few micrometres wide, are produced by evaporating CdSe powder in a furnace and using a flow of nitrogen to carry the vapour to a cooler region strewn with gold nanoparticles

(C. Ma *et al.* *J. Am. Chem. Soc.* doi:10.1021/ja0395644; 2003). The gold particles act as catalysts for the regrowth of CdSe crystals as ribbon-shaped structures.

Ma *et al.* think that the asymmetry of the ribbon edges — one smooth, the other jagged — is caused by their differing chemical composition. The orientation of crystal planes in the ribbons means that their edges terminate in a layer of either cadmium or selenium. The former is more chemically reactive: in zinc oxide (which has the same crystal structure), a zinc-capped surface can act as a self-catalyst for

further growth, resulting in comb-like structures (Z. L. Wang *et al.* *Phys. Rev. Lett.* **91**, 185502; 2003). A similar process here at cadmium-

terminated edges produces lateral growth with a rough profile, which resolves itself into the sawtooth profile.

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to separate efficiently. So the authors screened for genes that were lethal only when expressed with Rec8 in mitotic fission yeast. They isolated one such gene, which is usually expressed only in meiosis, and named its encoded protein Sgo1, short for 'Sugoshin' — 'guardian spirit' in Japanese.

Katajima *et al.* found that expressing Sgo1 with Rec8 in mitotic cells allowed Rec8 to persist at mitotic centromeres. Moreover, Sgo1 associates with centromeres in early meiosis and remains there until anaphase I, as expected (Fig. 2). Deleting the gene results in normal chromosome segregation in meiosis I but random segregation of sister chromatids in meiosis II, because of premature loss of cohesion at anaphase I (because all Rec8 is now cleaved). The authors further show that Sgo1 and Rec8 associate in the same complex. These data are consistent with a role for Sgo1 in protecting centromeric Rec8 from separate at the onset of anaphase I.

How is Sgo1 recruited to centromeres? Here, too, Kitajima *et al.* suggest an answer. During both meiosis and mitosis, a 'checkpoint' monitors the attachment of centromeres to the spindle — the apparatus that physically separates chromosomes. This checkpoint also monitors the physical tension between sister centromeres. By doing so it ensures that sister chromatids make the correct attachments and achieve the required arrangement on the spindle⁹. Previous analyses of fission yeast¹⁰ had shown that one checkpoint component, Bub1, is required to maintain Rec8 at centromeres after anaphase I.

Kitajima *et al.* show that Bub1 is, in fact, required to recruit Sgo1 to centromeres, suggesting a connection between the protection of Rec8 and the spindle checkpoint. One

possibility is that the lack of tension between sister centromeres in meiosis I — resulting from their enforced attachment to the same spindle pole — activates Bub1, which then recruits Sgo1. Such a signal would have to differ from that normally associated with a lack of inter-centromere tension; otherwise, division would stop⁹.

Broadening their findings, Kitajima *et al.* found that fission yeast contains a protein related to Sgo1, which they call Sgo2. This protein is located at centromeres in mitotic cells, and again the association depends on Bub1. Its function is unclear, although deleting it results in viable cells with a higher rate of chromosome loss. In addition, there is a single Sgo protein, Sgo1, in budding yeast, which seems to behave similarly to fission yeast Sgo1 and Sgo2. The authors also identified proteins with regions of reasonable similarity in multicellular organisms. One of these proteins is the fruitfly Mei-S332 — so it seems that this protein's closely guarded secret is that it protects meiotic cohesion after all.

Our understanding of the role of Sgo1 in meiotic chromosome segregation has been confirmed and extended by three additional studies in both fission¹¹ and budding^{12,13} yeast. Further insight has been provided into the role of fission yeast Sgo2 that localizes with meiotic centromeres¹¹. It seems to be required in meiosis I to orient the unified sister-centromeres so that they only engage microtubules protruding from the same spindle pole. Thus, cells with Sgo2 deleted exhibit a primary defect in the segregation of chromosomes during meiosis I (ref. 11). The combined meiotic phenotype of cells lacking both Sgo1 and Sgo2 is essentially identical to that described for cells with no Bub1 (ref. 10). This is consistent with Bub1 being required to recruit these important

determiners of chromosome behaviour to meiotic centromeres.

Returning to the prevalence of chromosome-segregation defects in ageing human eggs: could one contributing factor be a premature loss of the protective influence of Sgo1 during meiosis I, resulting in aberrant chromosome segregation in meiosis II? Sgo1 itself is usually degraded during anaphase I (ref. 3), but perhaps its levels also decline stochastically with maternal age in humans. This would, however, account only for the proportion of aberrations that are caused by defective meiosis II — and these are few compared with those occurring in meiosis I (refs 1, 2). Nonetheless, the discovery of the Sgo proteins should prompt further investigations that might provide insight into defects caused by aberrant numbers of human chromosomes.

Robin Allshire is at The Wellcome Trust Centre for Cell Biology, University of Edinburgh, Edinburgh EH9 3JR, UK.
e-mail: robin.allshire@ed.ac.uk

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