Sla1p expression inhibited NPFX and regulate actin dynamics. Disrupting to interact with the endocytic machinery proteins yielded Sla1p, which is known the results imply that Sla1p is part of mediated endocytosis. The hybrid screen for NPFX truncated cell surface receptor. A two-sufficient to direct the uptake of a containing the amino acid motif may exist in mammalian cells. This suggests that an analogous system involved in recognizing endocytic targeting signals in yeast. The work links targeting several different observations about endocytosis, suggesting that the yeast system endocytosis. The authors found that a sequence containing the amino acid motif NPFX, previously characterized as an endocytic targeting signal, is known to interact with the clathrin-based endocytic machinery. By analogy, a similar complex might play a role in mammalian cells. Searches of the yeast genome database suggest that an analogous system may exist in mammalian cells. Despite this, the authors suggest that an analogous system may exist in mammalian cells. The authors propose that in addition to separating homologous chromosomes or sister chromatids, in metaphase I, AR-2 localizes to the last points of contact between homologous chromosomes. In metaphase II, AR-2 localizes to the last points of contact between sister chromatids, corresponding to the last points of contact between homologous chromosomes. In metaphase I, some mechanism must allow homologous chromosomes to separate while keeping sister chromatids together. The authors also identified additional components in what is likely to be a conserved pathway controlling chromosome cohesion.

Adapting to the pit

Eukaryotic cells internalize various types of protein sorting complexes that link cargo bearing the NPFX motif to the actin cytoskeleton. The authors describe the first example of a protein that can act as an endocytic adaptin. Sla1p was identified in a hybrid screen for NPFX containing the amino acid motif NPFX. The results suggest that an analogous system may exist in mammalian cells. The authors propose that in addition to separating homologous chromosomes or sister chromatids, in metaphase I, AR-2 localizes to the last points of contact between homologous chromosomes. In metaphase II, AR-2 localizes to the last points of contact between sister chromatids, corresponding to the last points of contact between homologous chromosomes. In metaphase I, some mechanism must allow homologous chromosomes to separate while keeping sister chromatids together. The authors also identified additional components in what is likely to be a conserved pathway controlling chromosome cohesion.

For healthy eyes and bones: got Lrp?

The Lrp family of secreted proteins controls several crucial developmental processes. Mice lacking functional Lrp exhibit a pseudoglioma syndrome, suggesting that Lrp functions in controlling both osteogenesis and eye development. The work identifies Lrp as a critical component for identifying a long-sought genetic component for bone mass determination. The authors report that the targeted disruption of Lrp in mice causes a phenotype virtually identical to that seen in humans with osteoporosis-mental processes, some of which are apparently mediated by Wnt coreceptors. The results from the LDL receptor-related protein family. Now, on page 303, Kato et al. report that the targeted disruption of Lrp5 in mice causes a phenotype virtually identical to that seen in humans with osteoporosis-mental processes, some of which are apparently mediated by Wnt coreceptors.