Polycomb group (PcG) and trithorax group (trxG) proteins control the transcription of Hox and other target genes during development by binding to their respective response elements, which cluster in regulatory regions called maintenance elements (MEs). But do PcG and trxG proteins act synergistically at MEs? Not in most cases, claim Petruk et al., who have examined how trxG and PcG proteins associate with the ME of the bxd regulatory region of an Ultrabithorax (Ubx) transgene in individual Drosophila salivary gland cells in vivo (see p. 2383). Multiple trxG and PcG proteins, they report, act through the same or at juxtaposed sequences in the bxd ME. However, trxG or PcG proteins, but not both, associate with the ME of an activated or repressed Ubx transgene, respectively. Only the PcG protein Asx and the trxG protein Ash1 require Trithorax to bind to their targets. These results provide new insights into how PcG and trxG proteins might regulate transcription during development and during pathogenic processes such as cancer.

Myosin IIB: a force for morphogenesis

Two tissue movements – convergence and extension – are essential for axial morphogenesis in vertebrate and invertebrate embryos. But what generates the tensile forces that drive the intercalation of cells that underlies these two movements? On p. 2435, Skoglund and colleagues report that in Xenopus laevis embryos, convergence and extension at gastrulation require a myosin IIB-dependent cortical actin network. Using morpholino knockdown, they show that myosin IIB (a cytoskeletal myosin that crosslinks actin filaments and acts as a molecular motor) is needed during gastrulation to maintain a stereotypical cortical actin cytoskeleton. This network is polarized relative to the embryonic axis, the researchers report, and cyclically lengthens and shortens during gastrulation. Depletion of myosin IIB also results in the loss of the polarized protrusive activity usually seen in intercalating cells, the loss of cell-cell and cell-matrix adhesion, and failure of blastopore closure. Together, these findings reveal how a molecular-scale motor protein can generate the tensile forces that drive tissue-scale embryonic morphogenesis.

IN JOURNAL OF CELL SCIENCE
Actin makes a move with annexin A2

The dynamic remodelling of the actin cytoskeleton is crucial for cell adhesion and motility, and can be triggered by stimuli that activate the insulin receptor (IR) and other receptor tyrosine kinases. IR activation promotes cell motility by disrupting cell-substrate contacts, but many steps in this signalling cascade are unknown. In Journal of Cell Science, Konietzko et al. now identify a key stage in the pathway – the tyrosine phosphorylation of the phospholipid- and actin-binding protein annexin A2. In kidney cells that overexpress the human IR, they show that annexin A2 is tyrosine phosphorylated in response to insulin and that annexin A2 and the IR co-immunoprecipitate, indicating that the IR phosphorylates annexin A2 directly. Rho/ROCK signalling, the authors show, mediates insulin-induced morphological changes, and knocking down annexin A2 inhibits insulin-triggered Rho activation and actin rearrangements. From their findings, the authors propose that annexin A2 tyrosine phosphorylation links IR activation to Rho/ROCK-mediated actin rearrangement and cell adhesion.