

IN BRIEF

 RNA LOCALIZATION**Promoters of mRNA fate**

Cells respond to stress by transcribing survival genes. Zid and O'shea found that, in response to glucose starvation in budding yeast, the translation of a subset of transcriptionally activated mRNAs was increased, and that of another subset was decreased. Transcripts of the first subset were diffusely localized in the cytoplasm, whereas those of the second subset were localized to stress granules, which could explain their decreased translation. When promoter sequences of both gene subsets were fused to a reporter gene, it recapitulated the localization and translation of the endogenous mRNAs. Specifically, promoter sequences responsive to heat shock factor 1 (HSF1) conferred mRNA diffused localization and increased translation. Thus, promoters could influence gene expression by mechanisms other than transcriptional control, perhaps through mediating the loading onto mRNAs of proteins that direct their localization.

ORIGINAL RESEARCH PAPER Zid, B. M. & O'shea E. K. Promoter sequences direct cytoplasmic localization and translation of mRNAs during starvation in yeast. *Nature* <http://dx.doi.org/10.1038/nature13578> (2014)

 CELL MIGRATION**The forces that close wounds**

Epithelial tissues repair wounds through the collective movement of epithelial cells into the damaged area. This involves extension of the leading edge of cells, which exerts traction on the substrate to propel the cells forward, and the contraction of a supracellular actomyosin ring around the wound. Brugués *et al.* quantified the forces that cultured cells exert on substrates after laser-induced wounding. Consistent with forward movement, cells adjacent to the wound generated radial traction forces pointing away from the wound. Inward-pointing forces and tangential forces (parallel to the wound margin), which result from the transmission of contractile forces from the actomyosin ring to the substrate through focal adhesions, were also observed. Tangential forces could only be explained by the actomyosin ring being a heterogeneous structure, with some regions sustaining greater tension than others. Thus, alternate patterns of substrate compression and stretching drag the cell sheet into the wound area.

ORIGINAL RESEARCH PAPER Brugués, A. *et al.* Forces driving epithelial wound healing. *Nature Phys.* <http://dx.doi.org/10.1038/nphys3040> (2014)

 STEM CELLS**A role for nuclear p120 catenin in differentiation**

p120 catenin interacts with E-cadherin and functions at adherens junctions; it can also modulate gene expression in the nucleus. Lee *et al.* reveal that nuclear p120 catenin binds to REST and Co-REST, components of a transcriptional repressor complex. Depleting mouse embryonic stem (ES) cells of p120 catenin enhanced REST–DNA binding and decreased REST target gene expression; overexpressing p120 catenin had the opposite effect. REST–Co-REST is thought to inhibit neural stem cell differentiation. Overexpression of p120 catenin in mouse ES cells accelerated their differentiation and depletion of p120 catenin correlated with a decrease in the expression of REST–CoREST gene targets that are involved in neural development. Thus, p120 catenin may inhibit REST–Co-REST to promote neural differentiation. Further data suggest a model in which E-cadherin might regulate the effects of p120 catenin on REST–Co-REST by regulating the level of nuclear p120 catenin.

ORIGINAL RESEARCH PAPER Lee, M. *et al.* P120-catenin regulates REST/CoREST, and modulates mouse embryonic stem cell differentiation. *J. Cell Sci.* <http://dx.doi.org/10.1242/jcs.151944> (2014)