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IN BRIEF

REGENERATION

Switching it on and off with enhancers

As mammalian tissues have limited regenerative capacity, understanding how regeneration processes are controlled has considerable clinical potential. Kang *et al.* investigated regulatory sequences associated with the activation of regenerative programmes. By studying regenerating tissues in zebrafish, they identified a 1.3 kb sequence (termed LEN) in the enhancer region of the leptin b gene (*lepb*), which, when placed upstream of reporter genes, could drive their robust expression selectively following injury. Using LEN and the minimal *lepb* promoter, the authors were able to induce ectopic expression of both pro- and anti-regenerative factors in the injured tissues and to modulate the regeneration process. Importantly, LEN could also drive reporter gene expression following injury in mice, opening exciting new avenues in the field of regenerative biology.

ORIGINAL ARTICLE Kang, J. *et al.* Modulation of tissue repair by regeneration enhancer elements. *Nature* http://dx.doi.org/10.1038/nature17644 (2016)

TRANSLATION

The features of pathologic RAN translation

Some repetitive sequences can undergo erroneous expansion during DNA replication, which may lead to neurodegeneration and other pathologies. Such expanded repeats can be aberrantly expressed in a process known as repeat-associated non-AUG (RAN) translation. Kearse *et al.* established a reporter assay to study the mechanism of RAN translation both *in vitro* and in cells. Focusing on CGG repeats, which are found in the *FMR1* gene and can cause fragile X mental retardation, they showed that translation of repeats initiated at non-AUG codons, at least in part, shares a mechanism with canonical translation, involving 5′ cap-dependent scanning by ribosomes. They further revealed that RAN translation is influenced by the repeat length, the reading frame and the surrounding sequence, generating diverse protein products with variable efficiency. Analysis of other pathology-associated repeats should provide further insights into the features and mechanism of RAN translation.

ORIGINAL ARTICLE Kearse, M. G. *et al.* CGG repeat-associated non-AUG translation utilizes a cap-dependent scanning mechanism of initiation to produce toxic proteins. *Mol. Cell* http://dx.doi.org/10.1016/j.molcel.2016.02.034 (2016)

CELL MIGRATION

Recycling active integrin for adhesion reassembly

Regulated assembly and disassembly of cell–matrix adhesions is critical for efficient cell migration. It has previously been shown that disassembly of these adhesions depends mostly on integrin endocytosis, but the fate of internalized integrins has remained elusive. Nader *et al.* now demonstrate that internalized integrins are recycled back to the membrane in a Src signalling-dependent manner, where they engage in the formation of new adhesions. This integrin pool is maintained in an active conformation by the activity of focal adhesion kinase (FAK), which acts through recruiting a canonical integrin activator, talin, to integrin-containing endosomes. Importantly, both FAK and Src signalling were necessary for efficient focal adhesion reassembly and cell migration, indicating the importance of the maintenance of activation of internalized integrin and its recycling for migratory processes.

ORIGINAL ARTICLE Nader, G. P. F., Ezratty, E. J. & Gundersen, G. G. FAK, talin and PIPKIγ regulate endocytosed integrin activation to polarize focal adhesion assembly. *Nat. Cell Biol.* http://dx.doi.org/10.1038/ncb3333 (2016)