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IN BRIEF

RNA INTERFERENCE

Genome-wide resources of endoribonuclease-prepared short interfering RNAs for specific loss-of-function studies.

Kittler, R. *et al. Nature Methods* 11 Mar 2007 (doi:10.1038/nmeth1025)

Although short interfering (si)RNA-mediated gene knockdown has become a standard technique for loss-of-function studies, genome-wide siRNA studies are expensive. Kittler *et al.* previously reported an alternative and cheaper method for generating siRNAs using endoribonuclease; however, these endoribonuclease-prepared siRNAs (esiRNAs) were hampered by low knockdown efficiency and high off-target effects. Now, the group has generated an algorithm that predicts the optimal sequence in a coding region of a protein against which to generate esiRNAs (available free online). In comparative experiments against chemically synthesized siRNAs, the optimized esiRNAs achieved a similar level of mRNA knockdown with fewer off-target gene-silencing effects.

EPIGENETICS

Dynamics of replication-independent histone turnover in budding yeast.

Dion, M. F. *et al. Science* **315**, 1405–1408 (2007)

Histone replacement marks the boundaries of cis-regulatory domains.

Mito, Y. *et al. Science* **315**,1408–1411 (2007)

Two papers report that the turnover of H3 histone proteins is highly variable across the genome of both *Saccharomyces cerevisiae* and *Drosophila melanogaster*. In both species, the highest turnover was found to occur at defined promoter regions and at chromatin boundary elements — specialized regions that divide functionally independent domains of genetic activity. The groups propose that high histone-protein turnover at promoter regions might serve to transiently allow access to transcription-factor-binding sites, whereas rapid turnover at chromatin boundaries might help to maintain functionally independent genetic regions.

CYTOSKELETON

Mechanism of actin network attachment to moving membranes: barbed end capture by N-WASP WH2 domains.

Co, C. *et al. Cell* **128**, 901–913 (2007)

Assembly of actin networks at membrane surfaces requires Wiskott–Aldrich syndrome protein (WASP) to deliver actin monomers to the actin-polymerizing ARP2/3 complex at barbed (growing) ends of actin filaments. Growing actin filaments can exert forces against the membrane, generating cell protrusions or driving vesicle transport. However, how actin filaments maintain attachments to a membrane while allowing actin monomers to be added to the barbed end is an unresolved question. Co *et al.* show an unanticipated interaction between the WASP-homology-2 (WH2) domains of WASP and the barbed ends of actin filaments after the polymerization event, and this interaction was essential in keeping actin filaments hooked onto the membrane. The authors propose that the affinity of WH2 for barbed ends is low, allowing rounds of WH2 detachment and attachment, and therefore permitting the incorporation of new actin monomers to the growing filament.