IN BRIEF

RNA METABOLISM

Methyladenosine promotes translation

N^6-methyladenosine (m^6A) is the most common eukaryotic internal mRNA modification. Wang *et al.* show that the m^6A reader YTHDF1 promotes the translation of methylated mRNAs in human cells. Transcriptome-wide analyses indicated that YTHDF1 recognizes m^6A on many mRNAs. Ribosome profiling revealed a significant decrease in the translation efficiency of YTHDF1 targets following YTHDF1 depletion or the reduction of m^6A levels, and tethering YTHDF1 to a reporter transcript enhanced its translation. YTHDF1-dependent translation could be promoted by delivering mRNAs to the translation machinery and by enhancing translation initiation. At mRNAs targeted by both YTHDF1 and YTHDF2 (which decreases mRNA stability), YTHDF1 bound earlier than YTHDF2, suggesting that they function together to dynamically control gene expression.

ORIGINAL RESEARCH PAPER Wang, X. *et al.* N^6-methyladenosine modulates messenger RNA translation efficiency. *Cell* **161**, 1388–1399 (2015)

SPLICING

Unmasking exitrons

Kalyna and colleagues characterized protein-coding exonic sequences that can undergo alternative splicing, which they termed exonic introns (exitrons; previously termed cryptic introns). They defined >1,000 exitrons in >3% of *Arabidopsis thaliana* coding genes, including in many genes annotated as intron-less, and found that they possess sequence characteristics different from those of exons and introns. Expression analysis of ten genes with exitrons that preserve the reading frame upon splicing revealed tissue-, stress- and development-dependent exitron splicing, which was affected by the expression of several alternative splicing factors. Exitron abundance and characteristics are conserved between the *A. thaliana* and human genomes; such characteristics include tissue-dependent alternative splicing and enrichment in protein disordered regions, short linear motifs, and phosphorylation and ubiquitylation sites. The majority of exitrons seem to have originated from exons, but some are probably derivatives of intron degeneration.

ORIGINAL RESEARCH PAPER Marquez, Y. *et al.* Unmasking alternative splicing inside protein-coding exons defines exitrons and their role in proteome plasticity. *Genome Res.* http://dx.doi.org/10.1101/gr.186585.114 (2015)

CHROMOSOMES

Finding the right size

Cell and nucleus size vary dramatically between developmental stages, cell types and species. During metazoan early development, embryos do not increase in volume, and cell divisions result in smaller cells. Mitotic spindle length, centrosome size and nuclear size are known to scale with cell size. A study in *Caenorhabditis elegans* now reveals that cell size and nuclear size regulate chromosome length independently. High-resolution imaging of intact embryos showed that cell size reduction beyond a certain threshold was accompanied by chromosome shortening. Uncoupling nuclear and cell sizes by disrupting the RAN·GTP gradient across the nuclear membrane showed that chromosomes can become shorter in response to reduced nuclear size independently of cell size. The exact mechanisms that regulate chromosome compaction to attain specific lengths remain to be elucidated.

ORIGINAL RESEARCH PAPER Ladouceur, A.-M., Dorn, J. F. & Maddox, P. S. Mitotic chromosome length scales in response to both cell and nuclear size. *J. Cell Biol.* **209**, 645–652 (2015)