

## Articles of Significant Interest Selected from This Issue by the Editors

### Spleen Transcriptome and Pathology in Tristetraprolin Deficiency

Members of the tristetraprolin (TTP) family of tandem zinc finger proteins influence mRNA levels by binding to AU-rich elements in the 3' untranslated regions of specific mRNAs and promoting their deadenylation and decay. Patial et al. (p. 1395–1411) identified many changes in steady-state transcript levels in TTP-deficient mice that also lacked tumor necrosis factor receptors to prevent the inflammatory phenotype that normally accompanies TTP deficiency. Although a large proportion of the splenic transcriptome was affected by the absence of TTP, only a small fraction exhibited mRNA/pre-mRNA ratios that suggested increased posttranscriptional mRNA stability.

### Tumor Necrosis Factor Alpha Upregulates Splicing Independently of Transcription and in Cooperation with Spt5

Tumor necrosis factor alpha (TNF- $\alpha$ ) is a proinflammatory cytokine known to induce transcription of many genes, primarily through activation of NF- $\kappa$ B. This induction relies in part on Spt5, the major subunit of the transcription elongation factor DSIF. Diamant et al. (p. 1342–1353) now show that TNF- $\alpha$  stimulation also enhances splicing in an unexpectedly large number of genes without inducing transcription. Accordingly, this enhancement is independent of NF- $\kappa$ B activation. The splicing effect is mediated by Spt5, and affected genes become enriched with the Spt5-RNA polymerase II (Pol II) complex following TNF- $\alpha$  treatment. This study identifies a novel activity of TNF- $\alpha$  and uncovers Pol II-Spt5 as a competent coordinator of cotranscriptional splicing.