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- Thyroid hormones speed cellular aging

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- TopBP1/Dpb11 binds DNA anaphase bridges to prevent genome instability

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- Long noncoding RNA-mediated intrachromosomal interactions promote imprinting at the *Kcnq1* locus

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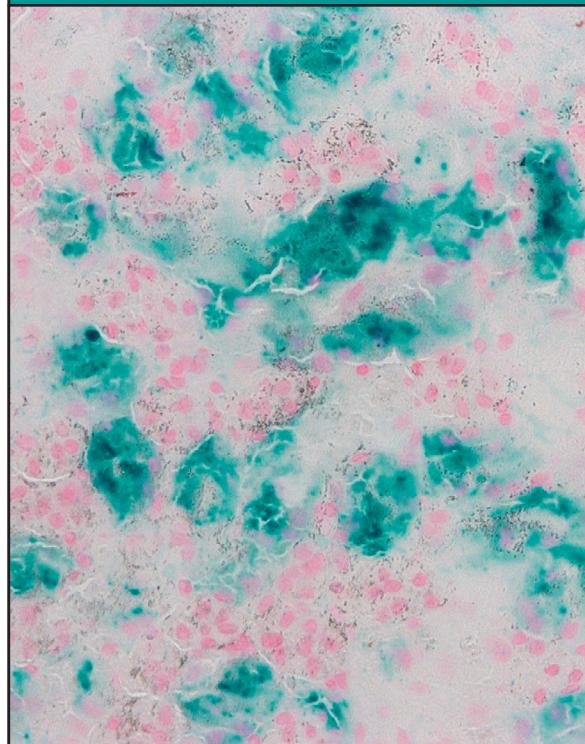
- Presynaptic CK2 promotes synapse organization and stability by targeting Ankyrin2

Victoria Bulat, Melanie Rast, and Jan Pielage

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- Cytoplasmic protein methylation is essential for neural crest migration

Katie L. Vermillion, Kevin A. Lidberg, and Laura S. Gammill



On the cover

β-Galactosidase activity (blue) indicates senescent cells in the liver of hyperthyroid mice. Zambrano et al. describe how thyroid hormone and its receptor cause increased mitochondrial respiration and reactive oxygen species production, leading to DNA damage and premature cell senescence.

Image © 2014 Zambrano et al.

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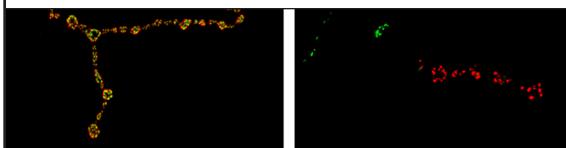
Dbl3 drives Cdc42 signaling at the apical margin to regulate junction position and apical differentiation

Ceniz Zihni, Peter M.G. Munro, Ahmed Elbediwy, Nicholas H. Keep, Stephen J. Terry, John Harris, Maria S. Balda, and Karl Matter

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The thyroid hormone receptor β induces DNA damage and premature senescence

Alberto Zambrano, Verónica García-Carpizo, María Esther Gallardo, Raquel Villamuera, María Ana Gómez-Ferreira, Angel Pascual, Nicolas Buisine, Laurent M. Sachs, Rafael Garesse, and Ana Aranda



Bulat et al. reveal that casein kinase 2 promotes synapse organization and stability by phosphorylating the adaptor molecule Anykrin2. In a neuromuscular junction from a control fly (left), the presynaptic active zone marker Bruchpilot (green) is closely apposed to the postsynaptic glutamate receptors (red). But this organization is lost in flies whose neurons lack the regulatory β subunit of casein kinase 2 (right).
Image © 2014 Bulat et al.
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