



## A zebrafish model for liver regeneration

Understanding how damaged livers regenerate is key to developing new treatments for liver disease. Curado et al. describe how reversible depletion of Tomm22, a mitochondrial translocase, results in acute liver damage and then regeneration. Tomm22 mutant fish will be a useful resource as a model for liver regeneration; as a rapid screening tool for drug and gene discovery, and for studying mitochondrial hepatopathies. *K.W.*

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## Polycystin toxicity in *C. elegans*

Patients with autosomal dominant polycystic kidney disease (ADPKD) develop fluid-filled renal cysts that can precipitate kidney failure. *PKD1* and *PKD2*, which are compromised in familial ADPKD, encode polycystin proteins that combine to form a cation channel in the sensory cilia of the renal epithelium. Miller and Portman show that polycystin homologues in *C. elegans* interact with a novel transmembrane protein CWP-5. CWP-5 mutation disrupts the formation of sensory cilia and may prevent toxic polycystin forms from reaching cilia. *K.K.*

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## Fatty liver disease in ricefish

Nonalcoholic steatohepatitis (NASH) causes liver degeneration and fibrosis, which can lead to cirrhosis and/or carcinoma. Matsumoto et al. show that medaka ricefish fed a high-fat diet exhibit physiological changes reminiscent of human patients. The n-3 polyunsaturated fatty acids in fish help to prevent the disease. The NASH-like symptoms in fish were ameliorated with EPA, a drug used to treat the human condition, supporting the relevance of this model for studying NASH. *K.K.*

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## Mouse susceptibility to depression

Susceptibility to depression and a poor response to antidepressants are associated with the presence of a certain allele (S) encoding the serotonin transporter. Bartolomucci et al. show that mice heterozygous for this allele experience more dramatic behavioral changes than normal animals when faced with stress. Serotonin turnover is decreased in these mice, suggesting that a change in serotonin metabolism may underlie a predisposition to depression in individuals with the S form of the serotonin receptor. *K.K.*

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## Functional conservation of FXR genes

The FXR gene family consists of *FMRI*, *FXR1* and *FXR2*. Loss of *FMRI* function causes fragile X syndrome in humans but the contribution of related genes to disease is largely unknown. Coffee, Jr et al. show that *FMRI* function in *Drosophila* neurons is distinct from other gene family members, as neither *FXR1* nor *FXR2* rescue *FMRI* deficiency in neurons. However, expression of any of the three human proteins reverses deficiency effects on spermatogenesis. Redundancies in FXR gene functions are tissue specific in the fly. *K.K.*

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## Fracture repair in *Ccr2* knockout mice

Bone healing and regeneration following fracture is a complex process, and an influx of inflammatory cells is known to be required for successful healing to occur. Xing and co-authors show that, in mice lacking the *CCR2* chemokine receptor, which is required for monocyte trafficking and osteoclast function, fracture repair is significantly delayed. A better understanding of how inflammation regulates bone repair may lead to the development of new methods to optimize healing of traumatic skeletal injuries. *K.W.*

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## Tissue regeneration

Regenerating damaged or diseased tissue requires specific activation of a variety of specific precursors with a coordinated set of instructions. Lee et al. demonstrate that the regeneration of an amputated zebrafish fin causes Ras activation in a subset of precursor cells. Cells expand and migrate to form a new striped fin, similar to the original. Ras stimulates necessary melanocyte expansion and regeneration, and activated Ras leads to hyperpigmentation. Ras is a central regulator of pigmentation and pattern formation in the regenerating fin. *K.K.*

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