

PHARMACOTHERAPY

**Cystic fibrosis in ferrets**

Sun, X. et al. *Sci. Transl. Med.* **11**, eaau7531 (2019)

Although evidence suggests that cystic fibrosis (CF) — a multiorgan disease caused by mutations in the gene encoding cystic fibrosis transmembrane conductance regulator (CFTR) — starts in utero, the long-term effects of in utero abnormalities on the pathology of CF remain unclear. VX-770 (ivacaftor) is a CFTR potentiator that rescues channel gating in dysfunctional CFTR proteins from patients carrying a *CFTR*<sup>G551D</sup> mutation. The results of a new study showing that in utero administration of VX-770 in *CFTR*<sup>G551D</sup> ferrets protects the animals from developmental abnormalities and neonatal mortality associated with CF suggest that early interventions could improve the treatment of CF. The study also offers a new model to understand the role of CFTR in CF pathophysiology and inform future therapies. ALB

<https://doi.org/10.1038/s41684-019-0322-x>

STROKE

**Cognitive impairment after stroke**

Cuartero, M.I. et al. *J. Clin. Invest.* **129**, 1536–1550 (2019)

Although cognitive impairment is frequent in patients after a stroke, the mechanisms underlying post-stroke cognitive deficits remain uncertain. Animal models of the condition are lacking, which delays the understanding of its pathophysiology and the identification of therapies. A new study using mice subjected to middle cerebral artery occlusion (MCAO mice) to induce cortical ischemia suggests that the hippocampus largely contributes to long-term memory deficits after stroke. By showing that hippocampal neurogenesis after stroke positively correlates with memory impairment, that stroke-induced newborn neurons promote an aberrant hippocampal circuitry remodelling, and that inhibition of stroke-induced hippocampal neurogenesis mitigates hippocampus-dependent memory deficits, the study supports the targeting of hippocampal neurogenesis as a potential approach for the treatment of cognitive impairment after stroke. ALB

<https://doi.org/10.1038/s41684-019-0323-9>

BIOLOGICAL TECHNIQUES

**Exosomes for DMD**

Rogers, R.G. et al. *JCI Insight* **4**, e125754

Duchenne muscular dystrophy (DMD) is a genetic disease characterized by progressive muscle degeneration and weakness. DMD is caused by mutations in the *DMD* locus that alter the production of dystrophin, a structural protein connecting the internal cytoskeleton to the extracellular matrix in muscles. No cure exists for DMD and the use of anti-inflammatory steroids has been associated with severe side effects such as respiratory dysfunction. A new study reports that intravenous injection of cardiosphere-derived cells (CDCs), cardiac stromal cells with anti-inflammatory, antifibrotic and cardiomyogenic properties, improves the dystrophic phenotype of *mdx* mice, a mouse model of DMD. The study also shows that CDCs improve the function and regenerative capacity of muscle by secreting exosomes, thereby identifying CDCs-derived exosomes as new therapeutic candidates for DMD. ALB

<https://doi.org/10.1038/s41684-019-0324-8>

GUT MICROBIOTA

**Gut feeling**

Kraimi, N. et al. *J. Exp. Biol.* jeb.202879 (2019)

Several studies have started to link alterations in the gut microbiota (GM)-brain axis to gastrointestinal and neurological diseases. A study now suggests that the gut-brain axis also influences emotional reactivity. Germ-free chicks from a Japanese quail line (*Coturnix japonica*) selected for a high emotional reactivity (E+) that had been colonized with GM from a line with low emotional reactivity (E-) showed reduced emotional behaviour compared with germ-free chicks from E+ line colonized with GM from the E+ line. Results were reversed when behaviour was tested at a later stage, most likely owing to the resilience of GM to recover its equilibrium present in the E+ host. This study shows that GM transfer can influence emotional reactivity in Japanese quails and supports the existence of a GM-brain axis in birds. ALB

<https://doi.org/10.1038/s41684-019-0325-7>

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