VIROLOGY

Oh dear, SARS-CoV-2 in deer mice

Fagre, A. et al. *Plos Pathog* **17**, e1009585 (2021)

Laboratory mice of the *mus musculus* variety aren't naturally susceptible to SARS-CoV-2 infection, which relies on ACE2 as its point of entry into hosts. Other members of the rodent family, however, can be infected with the virus; these species include Syrian golden hamsters and, per new research from Colorado State University, deer mice.

Peromyscus maniculatus, which can be found throughout North America, have 17 of the 20 ACE2 residues considered critical for SARS-CoV-2 binding, suggesting they *might* be susceptible to infection. To confirm, the team experimentally challenged animals of both sexes. They found viral particles in the lungs and brains of the mice as well as elevated expression of genes involved with immune responses. Infected deer mice could also transmit the virus to naïve conspecifics. The results suggest deer mice could serve as an additional rodent model of SARS-CoV-2 infection, while also raising concerns about a novel reservoir should the virus spill back into wild populations. *EPN*

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REGENERATION

Failure to regrow

Lin, T., et al. *Dev Cell (2021)* https://doi.org/10.1016/j.devcel.2021.04.016

When it comes to regenerating limbs, results may vary. Among adult amphibians, axolotl salamanders can achieve complete limb recovery while *Xenopus* frogs only manage a partially regenerated 'spike.' Both make it to the same regenerative structure – a limb bud referred to as a blastema – but the frogs don't properly complete the program.

A new study combines cell lineage tracing in transgenic subjects of both species with single-cell sequencing and transplantation-based functional assays to compare the cellular and molecular differences behind the axolotls' regenerative abilities and *Xenopus*' lack thereof. Dedifferentiation appears to have something to do with it. When the axolotls reach the blastema stage, several intrinsic factors kick into gear that let fibroblasts, cells that make up connective tissue, turn into the missing bone, cartilage, tendons, and ligaments. In the frogs, those fibroblasts seem unable to be reprogrammed. *EPN*

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VISION

Seeing the violet light

Jiang, X. et al *PNAS* (2021) https://doi.org/10.1073/pnas.2018840118

Myopia is a growing problem in school-age children around the world. Beyond nearsightedness, the accompanying shape changes in the eye can contribute to a number of other visual issues, including cataracts, retinal detachment, glaucoma, and blindness. The cause of myopia is unclear, but outdoor activity appears to have a protective effect; some research suggests this might be related to the distinct wavelengths found in sunlight compared to those emitted by artificial lighting.

A new study in mice homes in on violet light, the shortest wavelength of visible light that is abundant outside but rare indoors. Exposing a mouse model of lens-induced myopia to violet light helped prevent myopia progression, but this depended on time of day – dusk exposure provided the protection – and on retinal expression of the neuropsin OPN5. *EPN*

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IMMUNOLOGY

Innate immunity in mutator mice

Lei, Y. et al. *Sci Adv* **22**, eabe7548 (2021)

Mutator mice have been used to study myriad elements of mitochondrial biology and disease. These mice, which carry mutations that negatively affect mitochondrial DNA, experience premature aging as well as a number of pathologies that human patients with mitochondrial diseases often contend with, including cardiomyopathy, anemia, and hearing loss. Mitochondrial disease patients often find themselves susceptible to infections as well, a consideration that's had little research in mouse models. A recent study in polymerase gamma (POLG) mutator mouse models of mitochondrial DNA instability explored how the genetic deficits influence innate immune responses.

The researchers involved found that the POLG mutator mice exhibit a hyperinflammatory response following lipopolysaccharide challenge, which models endotoxin shock. The mice increased type 1 interferon signaling, which in turn suppressed activation of nuclear factor erythroid 2-related factor 2. These changes increased oxidative stress, cytokine response, and metabolic dysfunction. *EPN*

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