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## research highlights

### IN BRIEF

#### IMMUNOLOGY

### Prepping for paternal pregnancy

Roth, O. et al. *PNAS* **117**, 9431-9439 (2020)

Pregnancy puts a particular strain on the body, as the gestating animal's immune system must come to tolerate the foreign DNA in its developing offspring. Much work has been done in mammalian mothers, but pregnancy has independently evolved in over 150 vertebrate lineages. These include seahorses and pipefish, in which many fathers take a turn at being pregnant.

New research looks at how these dads do it, finding evidence in the genomes of 12 different syngnathid species that paternal pregnancy is facilitated by a loss of major histocompatibility complex II. This collection of genes is an important component of the vertebrate adaptive immune system, and its loss to support pregnancy is despite the microbe-filled environment in which seahorses and pipefish live. The results suggest the vertebrate immune system may be more flexible than previously thought. *EPN*

<https://doi.org/10.1038/s41684-020-0562-9>

#### GENETICS

### Strain choice for SARM1

Uccellini, M.B. et al. *Cell Rep.* **31**, 107498 (2020)

Through studies of knockout mice, the protein sterile alpha and TIR motif containing 1 (SARM1) has been ascribed roles in mediating axon degeneration and in the susceptibility of the nervous system to various viral infections, including West Nile, La Crosse encephalitis, and vesicular stomatitis. The mice used for much of that work were generated by knocking out *Sarm1* in 129 embryonic stem cells and crossing those onto a C57BL/6 background. Those strain choices, however, may have been a bit confounding.

Using CRISPR, researchers at the Icahn School of Medicine at Mt. Sinai recently created SARM1-deficient mice on a pure black 6 background. The gene's potential role in axon degeneration and West Nile Virus susceptibility held true, but many other immune-related phenotypes observed in the classic knockout animals were absent. Background beware. *EPN*

<https://doi.org/10.1038/s41684-020-0564-7>

#### DEVELOPMENT

### A window to view mouse embryos

Huang, Q. et al. *Science* **368**, 181-186 (2020)

Direct, real-time observations of whole mouse embryos have long been obscured by the dam's body. To overcome this limitation, researchers at Duke University and MIT recently developed a new approach that provides a window—literally—into intrauterine development.

The team designed a small, 10 mm wide and 1.5 mm deep window with glass coverslip that can be surgically implanted into the mother's uterus at embryonic day 9.5, without lasting negative effects on the developing pups. The embryos can be visualized at cellular resolution, and, with coverslip removed, manipulated. In their paper, the researchers used the window to observe several fluorescently labelled neurons over time, deliver genes via adeno-associated virus vectors, perform *in utero* electroporation, and produce blastocyst chimeras. *EPN*

<https://doi.org/10.1038/s41684-020-0563-8>

#### NEUROSCIENCE

### A gene for juvenile sleep

Dilley, L.C. et al. *eLife* (2020) <https://doi.org/10.7554/eLife.52676>

The younger the animal, the longer it sleeps. That youngsters need more shut eye than their elders is likely related to the heavy demands of their developing nervous system—disrupted sleep in children can lead to neurocognitive problems as they become adults. The molecular underpinnings for such observations, however, remain unknown.

Day-old *Drosophila* also sleep more than more mature 4–5 day old fruit flies, and the insects lend themselves to high-throughput molecular screens. Using an RNAi screen, researchers at the Perelman School of Medicine at the University of Pennsylvania identify a gene that appears to be required for sleep maturation in the flies: *Pdm3*, a transcription factor involved in the wiring of dopaminergic neurites, which promote wakefulness, to areas of the brain that promote sleep. Young flies in which *pdm3* is knocked down don't sleep as much as they should. *EPN*

<https://doi.org/10.1038/s41684-020-0565-6>