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## research highlights

### IN BRIEF

#### AGING

## A roadmap for NHP ovarian aging

Wang, S. et al. *Cell* **180**, 585–600 (2020)

Ovarian aging is associated with a decrease in follicle number and oocyte quality, resulting in a gradual decline in female fertility. The molecular mechanisms driving ovarian aging remain unclear, hampering the development of interventions to treat female infertility and age-associated ovarian diseases, such as ovarian cancer.

In a new study published in *Cell*, a team of investigators from China and USA performed a single-cell transcriptomic analysis of ovaries from young and old cynomolgus monkeys; comparison of gene expression profiles in the different cell types present in the ovaries of the nonhuman primates (NHP) revealed changes in antioxidant signaling in oocytes and granulosa cells with aging, which indicates that oxidative damage is a crucial factor in ovarian functional decline. **ALB**

<https://doi.org/10.1038/s41684-020-0515-3>

### REGENERATIVE MEDICINE

## Building human vessels in pigs

Das, S. et al. *Nat. Biotechnol.* <https://doi.org/10.1038/s41587-019-0373-y> (2020)

To address the shortage of donor organs for transplantation, researchers are exploring different approaches, including strategies to produce human organs in animals. Previous studies have described the successful generation of xenogeneic organs in rodents by injection of pluripotent stem cells (PSCs) into blastocysts lacking a gene essential for the development of the organ. However, the organs generated by blastocyst complementation still contain host endothelium and might be rejected by the recipient if used for transplantation.

In a new study published in *Nature Biotechnology*, investigators injected human PSCs into pig blastocysts lacking *ETV2*, a transcription factor critical for the development of the endothelial lineage. Analysis of the resulting embryos revealed that all endothelial cells were of human origin, indicating that *ETV2* null pigs might be used to generate exogenous organs with reduced immunogenicity. **ALB**

<https://doi.org/10.1038/s41684-020-0517-1>

### NEURODEGENERATIVE DISEASE

## Parkinson's disease: from gut to brain

Challis, C. et al. *Nat. Neurosci.* <https://doi.org/10.1038/s41593-020-0589-7> (2020)

Synucleinopathies are neurodegenerative diseases characterized by the abnormal accumulation of alpha-synuclein ( $\alpha$ -Syn) protein aggregates in neurons, nerve fibres or glial cells. In Parkinson's disease, motor symptom manifestations coincide with the appearance of  $\alpha$ -Syn pathology in midbrain dopaminergic neurons, but increasing evidence suggests that the pathology might originate earlier in the gastrointestinal tract before progressing to the brain.

By showing that the inoculation of  $\alpha$ -Syn fibrils in the duodenum wall of aged mice, but not younger mice, resulted in progression of  $\alpha$ -Syn histopathology to the midbrain and subsequent motor defects, a new study provides further evidence of peripheral synucleinopathy in early Parkinson's disease. **ALB**

<https://doi.org/10.1038/s41684-020-0516-2>

### IMAGING

## A closer look at the eye

Prahst, C. et al. *elife* **9**, e49779 (2020)

Eye diseases are becoming increasingly common in the aging population, but the mechanisms underlying these conditions are still not fully understood. The difficulty to image spherical eye tissues with standard imaging techniques might have hampered new discoveries.

In a new study, Katie Bentley and her team used light-sheet fluorescence microscopy (LSFM) to look at a well-studied model of eye disease in mice (oxygen-induced retinopathy). Contrary to confocal microscopy that requires the slicing and mounting of the retina, LSFM can be used to image whole-tissue samples and perform live imaging. LSFM revealed unknown features of the eye disease, including a “knotted” morphology to pathological vascular tufts, as well as abnormal cell motility and filopodia dynamics. LSFM could be used to inform studies on various eye diseases. **ALB**

<https://doi.org/10.1038/s41684-020-0518-0>