Model of Organ Fibrosis

Mouse Model with a global deletion in the Spag17 gene

Novel ROSA^{mT/mG} Labeled Spag17 Knockout Mouse

Spag17 is a complex gene associated with cilia and microtubule cytoskeleton. Loss of Spag17 leads to developmental defects, primary cilia dyskinesia, accelerated aging, male infertility, and multi-organ fibrosis. Researchers at Virginia Commonwealth University (VCU) have developed a novel mouse model with a global deletion for the Spag17 gene using a CMV reporter and a ROSA^{mT/mG} linage tracing system.

The technology

The novel knockout mouse model that was created has a global deletion in the Spag17 gene. Cells deficient in Spag17 express an EGFP fluorophore for *in vivo* tracking. The generated mouse model presents a significant avenue to study primary cilia dyskinesia, development, skeletal dysplasia, primary cilia disfunction, multi-organ fibrosis and neuronal cells differentiation after.

Organ Tissue Fibrosis

WT KO WT KO WT KO WT KO

Figure 1. Multi-organ fibrosis is one of the phenotypes developed after conditional deletion of Spag17. Shown above are representative histological sections from skin, lung, kidney, liver, muscle and heart tissue stained by Masson's Trichrome stain as a fibrotic marker. Increased collagen deposition (blue) is observed in Spag17 knockout mice compared to wild-type controls.

Benefits

ROSA^{mT/mG} allows lineage tracking of Spag17 deficient cells

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Previously unidentified target gene and mouse model of fibrotic disease

Applications

- >> Evaluation and study of primary cilia dyskinesia
- Evaluation and study of skeletal dysplasia
- Evaluation and study of fibrotic disease
- >>> Identification of novel therapeutics for treatment of fibrotic disease
- Identification of novel pathways and mechanisms related to Spag17 deficiency

Patent status:

Patent pending: U.S. and foreign rights are available.

License status:

This technology is available for licensing to industry for further development and commercialization.

Category:

Biomedical

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