Model of Organ Fibrosis



Mouse Model for the deletion of the Spag17 gene after Cre recombination

Novel Spag17 Flox 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 the potential for the deletion of the Spag17 gene once crossed with a Cre mouse.

The technology

The novel flox mouse model that was created has the potential to generate a knockout for Spag17 once crossed with a Cre mouse. The generated mouse model presents a significant avenue to study primary cilia dyskinesia, development, skeletal dysplasia, primary cilia disfunction, aging, infertility, multi-organ fibrosis and neuronal cells differentiation after Cre recombination.

Organ Tissue Fibrosis

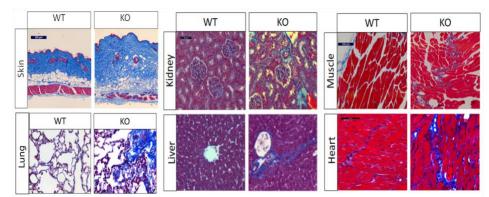


Figure 1. Multi-organ fibrosis is one of the phenotypes developed after conditional deletion of Spag17. Shown above are representative histological sections from skin, lung, kidney, liver, muscle and heart tissue stained by Masson's Trichrome stain as a fibrotic marker. Increased collagen deposition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

Benefits

Potential for the deletion of the gene by any Cre mouse model.

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 organ fibrosis

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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