Tankyrase inhibition for therapy of lung fibrosis

Researchers at Oslo University Hospital (OUH) have developed OM-247, a first-in-class small-molecule drug candidate targeting tankyrase (TNKS) inhibition. This novel therapeutic approach addresses idiopathic pulmonary fibrosis (IPF) by modulating the WNT/YAP signaling pathways, which are central drivers of fibrosis.

Business Opportunity

OM-247 shows potent anti-fibrotic activity in vitro, ex vivo (human precision-cut lung slices), and in validated rodent models. The compound exhibits favorable pharmacological and drug-like properties, positioning it as a promising candidate for clinical development. Currently, there are no TNKS inhibitors in clinical trials for lung fibrosis—an area of high unmet medical need. IPF is a progressive and fatal form of lung fibrosis, with a median survival of only three years after diagnosis. Moreover, patients recovering from severe viral lung infections, including COVID-19, are at increased risk of developing fibrosis, highlighting the urgent need for more effective therapies.

Technology Description

- OM-247 demonstrates favorable ADME properties and oral/inhalation PK profiles.
- Supported by a robust preclinical proof-of-concept dataset, including efficacy in vitro, ex vivo (human lung tissue), and in vivo in mouse and rat models.
- Current IPF therapies (Pirfenidone and Nintedanib) offer only modest efficacy and are associated with significant adverse effects.
- Aberrant WNT/β-catenin and YAP signaling are central to fibrosis progression.
- OM-247 is a highly potent and selective TNKS1/2 inhibitor, directly modulating these pathways.
- No TNKS/WNT/YAP inhibitors are currently in clinical development for fibrotic diseases—providing a clear first-mover advantage.

Category

Therapies/Small Molecules

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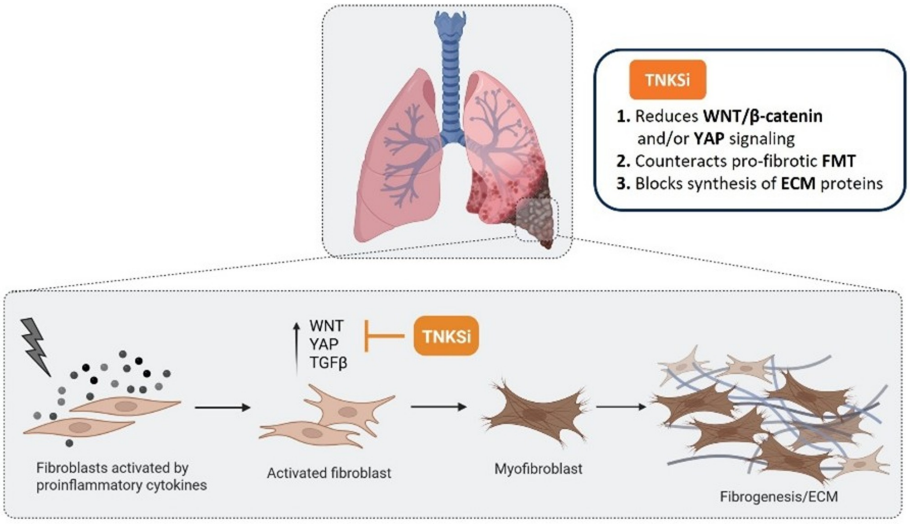

Fig. 1: Model for TNKS inhibitor anti-fibrotic mechanism of action.

Intellectual Property

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