Max-Planck-Innovation

Technology Offer

Novel cyclic peptide for the treatment of lung and liver fibrosis

Ref.-No.: 0105-5855-IKF

This technology offer describes a novel cyclic peptide that targets collagen-binding integrins to block fibroblast activation and extracellular matrix accumulation, showing strong anti-fibrotic effects in lung and liver disease models.

Background

Fibrosis is characterized by excessive extracellular matrix (ECM) deposition that disrupts tissue architecture and ultimately leads to organ failure. In industrialized countries, fibrotic diseases are estimated to contribute to roughly 45% of all deaths, highlighting a major unmet medical need. Despite extensive research, current anti-fibrotic therapies remain limited in both efficacy and indication range, with no approved drugs for liver fibrosis and only partially effective options for lung fibrosis such as idiopathic pulmonary fibrosis (IPF) or acute respiratory distress syndrome (ARDS). Recent work has underscored the central role of collagen-binding integrins in driving fibroblast activation and ECM accumulation, suggesting that targeted integrin modulation can attenuate fibrosis in preclinical models.

Technology

Scientists at the Max Planck Institute of Biochemistry have developed a novel cyclic peptide that selectively targets collagen-binding integrins on fibroblasts, thereby modulating pro-fibrotic signaling and ECM production. The peptide is optimized for stability and integrin binding, and can be formulated for systemic administration (e. g., subcutaneous depot), enabling efficient *in vivo* exposure.

Advantages

- Directly interferes with collagen-integrin signaling to prevent and reverse fibroblast activation.
- Demonstrates potent anti-fibrotic efficacy *in vitro* at low-nanomolar concentrations (down to ~20 nM).
- Reduces collagen accumulation in established mouse models of lung and liver fibrosis with favorable preliminary safety (no overt liver, kidney, or hematologic toxicity).
- Downstream mechanism of action suggests a broad therapeutic window and combinability with existing standard-of-care treatments.

Applications include treatment of liver fibrosis of various etiologies and lung fibrosis (e.g., IPF, ARDS-associated fibrosis and other interstitial lung diseases), as well as potential use in other fibro-proliferative conditions.

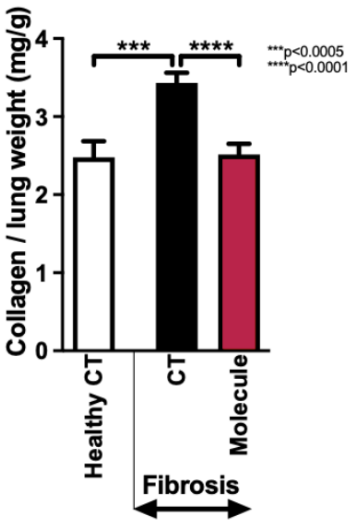

Figure 1 Treatment suppresses lung fibrosis. Treatment ("molecule") diminished collagen accumulation in the lung compared to fibrotic mice receiving 0.9% NaCl. Hamelmann et al. 2025, BioRxiv

Max-Planck-Innovation GmbH
Arnulfstr. 58
80335 München
www.max-planck-innovation.de

Patent Information

The international patent application WO2021018923A1 was filed in 2020.

Publication

The data is publicly available as preprint on bioRxiv: Hamelmann et al. 2025, Modulation of collagen-binding integrins affects fibroblast activation and inhibits fibrosis,
doi: https://doi.org/10.1101/2025.05.14.653428

Opportunity

We are open to research partnerships and license agreements to accelerate the integration of our antifibrotic compound into the clinical practice.

Contact

Dr. Ingrid Kapser-Fischer
Patent- & License Manager
Nutritionist, M.Sc.
Email:
kapser-fischer@max-planck-innovation.de

Max-Planck-Innovation GmbH
Arnulfstr. 58
80335 München
www.max-planck-innovation.de