Modulation of Myofibroblast Apoptosis for Treatment of Lung Fibrosis. (Hadasit)

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Raphael Breuer, Hadassah Ein Kerem, Department of Medicine, Pulmonology
Shulamit Richter-Dayan , Hadassah Ein Kerem

Investigators: Prof. Raphael Breuer and Dr. Shulamit Wallach-Dayan

Market Need

Regenerative capacity in vital organs is limited by fibrosis propensity. Idiopathic pulmonary fibrosis (IPF), a progressive lung disease linked with aging, is a classic example. IPF, with a 3–5-year survival, imposes a heavy economic burden on the medical system. Current treatment is palliative. The market for IPF was valued at $1,616 million in 2016, and is estimated to reach $3,569 million by 2023, growing at a CAGR of 11.9% from 2017 to 2023, due to rise in geriatric population and cigarette smoking. A critical factor in the market growth is that the cause of the disease is unknown, which makes the development of a therapeutic highly challenging, and poses an opportunity for identifying new mechanisms that underlie the disease pathogenesis.

Innovation

Our novel discovery that fibroblasts escape immune surveillance as a mechanism of tissue fibrosis (Wallach-Dayan, PNAS 2007), which was adopted by opinion leaders in the field of kidney fibrosis (Struz, Nephrol Dial Transplant (2008; 23: 2477–2479) and that small-molecules can regulate escape from immune surveillance, can pave the way for fibrosis resolution by offering a novel therapeutic approach for irremediable lung fibrosis by reducing myofibroblast resistance to apoptosis via manipulation of a certain target with small molecules.

Findings

- Target is essential for myofibroblast resistance to apoptosis and escape from immune surveillance.
- Identification of a mechanism of action.
- In vitro: small molecules downregulated human lung myofibroblast target levels and sensitized myofibroblasts induced apoptosis.
- In vivo: small molecule reduces target enzymatic activity and fibrosis (bleomycin-induced lung fibrosis).

Indications/Applications:

The small molecules and/or their derivatives and be used as a new class of proprietary pharmaceuticals for tissue fibrosis, such as lung fibrosis, and potentially in other organs. Lung fibrosis drugs will be delivered to the trachea, possibly via inhalation, possibly as a chronic treatment.

Competitive Advantage

The small molecules pose a novel mechanism of action for fibrosis in IPF and potentially other fibrotic diseases that may be treated based on the same mechanism. Because of the major unmet clinical need for patients suffering from these diseases and the limited selection of treatment, the opportunity for a novel class of drugs is highly attractive in the field of fibrosis.

Contact for more information:

Tal Almog ✉, 054-3187538

Hadasit Medical Research Services & Development Ltd
Mother & Child Pavilion, Hadassah Ein Kerem, Jerusalem , 91120 Israel
Phone: +972-2-6778757, Fax: +972-2-6437712, E-mail: skimhi@hadassah.org.il