

FGF-18 Treatment for Diseases Involving Cartilage Degeneration

TECHNICAL FIELD

Therapeutic: FGF-18 (2001-0911)

BACKGROUND

Cartilage is an essential structural component in many articular and non-articular areas of the body. Diseases involving cartilage degeneration are common and are associated with significant pain and morbidity and, depending on location, may be life threatening.

Currently, there are no therapies or treatments directed specifically at inhibiting cartilage degeneration or, more importantly, promoting cartilage regeneration and reconstruction. Therefore, there exists a need to identify and develop an effective therapy to treat diseases associated with cartilage degeneration.



TECHNOLOGY

Using various animal models and research tools, our investigator has reported marked expansion of tracheal-bronchial cartilage tissue in conducting airways of fetal mice expressing the transgene encoding mouse FGF-18 cDNA. In addition, ectopic expression of cartilage in the lung periphery is observed at sites not normally containing cartilage. *In situ* hybridization studies demonstrated that mouse FGF-18 mRNA was selectively expressed in stromal cells surrounding tracheal cartilage rings and in larynx at the sites of cartilage formation.

These findings support the concept that FGF-18 uniquely induces cartilage programming during development and specification of tissue and, thus, along with its receptors and signaling pathways, represents part of a molecular pathway that could be used to induce new cartilage formation or expand cartilage growth in various sites in the body including tracheal-bronchial rings, larynx, joints, bones and other sites where deposition of cartilage and bone could be therapeutic or beneficial.

APPLICATIONS

1. Therapeutic target for diseases associated with cartilage degeneration
2. Research tool

ADVANTAGES

- Direct target
- Potential to develop new therapeutic market

INVESTIGATOR

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STATUS

Patent applications pending.

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FGF-18 Treatment for Diseases Involving Cartilage Degeneration

THE INVENTOR

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BACKGROUND

Jeffrey A. Whitsett, MD, is chief of the Section of Neonatology, Perinatal and Pulmonary Biology at Cincinnati Children's Hospital Medical Center.

Dr. Whitsett received his medical degree from Columbia University, in New York, and has been a faculty member since 1977. He is internationally known for his research in pulmonary medicine, as well as for his clinical expertise in neonatology.



Dr. Whitsett has made a series of groundbreaking contributions in pulmonary medicine. His major pioneering work has been on surfactant proteins A, B, C, and D, cloning their genes, and clarifying their roles in lung development.

Throughout his career, Dr. Whitsett has had the remarkable ability to move from molecular biology, to animal models, to diagnosis and therapy of human disease. He played a critical role in making surfactant protein replacement a routine tool for treating immature lungs and respiratory distress syndrome in premature infants. His laboratory has contributed to the identification of a number of genes critical for lung formation and function. Mutations in genes regulating surfactant homeostasis were shown to cause acute and chronic lung disease in infants and adults.

Dr. Whitsett is a member of the Institute of Medicine, National Academy of Sciences and is the recipient of the Mead Johnson Award, a National Institutes of Health (NIH) Merit Award, the first Julius Comroe Lectureship in Pulmonary Research from FASEB, the William Cooper Procter Award from Cincinnati Children's, the Amberson Lecture Award of the American Thoracic Society, and the prestigious Daniel Drake Medal for scientific contributions from the University of Cincinnati College of Medicine.

Dr. Whitsett is the author of over 400 papers in both the basic science and clinical literature.