PKC Zeta Inhibitor Promotes Lung Injury Repair

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Technology description

Tech Description
Researchers at the University of Iowa have developed a potential treatment for patients suffering from ALI, including its most severe form, ARDS. This candidate treatment is the administration of PKC-zeta inhibitors, which block this key early signaling mediator that results in the cessation of proper lung function during periods of severe inflammation. The inhibitor construct that has been studied thus far is the peptide-based inhibitor with the following sequences: Ser-Ile-Tyr-Arg-Arg-Gly-Ala-Arg-Arg-Trp-Arg-Lys-LeuOH. Studies in mouse lung injury models have demonstrated that treatment with this PKC-zeta inhibitor preserve the alveolar epithelial barrier, partially preserve lung compliance, and promote lung repair by promoting lung progenitor cells. Additional studies have shown that this treatment may also be effective for chronic lung disease conditions, such as interstitial lung disease / pulmonary fibrosis.
Background Information
Acute lung injury (ALI) is condition that is characterized by acute severe hypoxia in patients without signs of left atrial hypertension. ALI is used to describe a continuum of clinical and radiographic changes in hypoxic patients, with acute respiratory distress syndrome (ARDS) representing the most severe end of the continuum. ALI is the result of an inflammatory response in the lung and is characterized by hypercapnia, diffuse chest infiltration and a substantial reduction in pulmonary compliance in addition to the severe hypoxia. The conventional treatment for ALI is lung protective ventilation, however, even with this treatment overall mortality rates remain 35-40% for patients suffering from this condition.

Advantages

NOVEL TREATMENT PATHWAY AND AGENT. The inhibition of PKC-delta signaling plays a key role in promoting multiple pathways for recovery in patients with acute pulmonary distress.
PROMOTES REPAIR OF DAMAGED LUNG TISSUE. Studies have demonstrated that this treatment stimulates the proliferation of tissue types that are damaged and poorly functioning during ALI / ARDS.

Institution

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