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**Commentary**

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# Complementary DNA Trans-Membrane Conductance and Regulator Expression in the Human Lung

**Diaco Kochar***

*Department of Pharmacy, Baghdad University, Baghdad, Iraq*

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## DESCRIPTION

The human lung serves as the body's primary organ for gas exchange, delivering inspired air and venous blood into close vicinity so that oxygen may be delivered to red blood cells flowing through the alveolar walls. In return, the body releases carbon dioxide, a waste product of metabolism. Although fascinating from a biological perspective, this complexity presents a barrier for gene and cellular therapists and focused their study on using gene therapy to treat lung disease and stem/progenitor cell treatment to replace an injured lung. This requires the transfer and adequate expression of the normal human Cystic Fibrosis (CF) trans-membrane conductance regulator complementary DNA (cDNA) in more than 10% of airway epithelium dispersed among 223 dichotomously branching airways with a diameter ranging from 1 cm to 50 mm. Not only vectors be secure (with no insertion mutagenesis or significant adverse host reactions to the vector delivery system), but the expression of the cystic fibrosis trans-membrane gene must also be persistent and controlled in a manner that is comparable to that of the healthy airway epithelium.

These defences have evolved to remove viruses and foreign macromolecules from the lung, as well as to restrict the transfer of the cystic fibrosis trans membrane cDNA to the airway epithelium. Additionally, new vector delivery techniques have been developed, and improved experimental animals with CF-relevant traits have been created. As a result, a lot of progress has been made, and many studies are underway to advance the potential of gene therapy to treat CF. After all, the study of gene therapy has recently achieved outstanding success in the treatment of blindness, immunodeficiency, and haemophilia. If a patient has chronic lung failure, replacing a damaged lung with a normal lung can restore normal gas exchange. Identifying the stem/progenitor cells of the lung and creating methods to regenerate healthy lung tissue, but might accomplished by creating new lung tissue from stem/progenitor cells. If effective, this would be applicable not only to Cystic Fibrosis (CF), but also to the majority of chronic lung diseases, such as pulmonary hypertension, interstitial lung disorders (fibrotic lung diseases), and chronic obstructive pulmonary disease, emphysema and chronic bronchitis. Whereas regenerative lung studies are still in its adolescence compared to lung gene therapy, significant progress has been made. As thoroughly illustrated in the evaluation, there have been numerous new discoveries regarding the biology of lung regeneration, including *exvivo* lung bioengineering, bone marrow-derived mesenchyme stromal cells, and embryonic stem cells, induced potent stem cells, and lung-related endogenous progenitors and circulating endothelial progenitors.

The difficulties are great and current biology knowledge is insufficient. First, despite the fascinating advancements made over the past ten years, stem/progenitor cells remain mysterious "black boxes" for which science has just begun to scratch the surface of knowledge. Second, the lung is an extremely intricate organ with two distinct circulations, complicated alveolar structures, and airways. It is also a muscular organ, and carbon dioxide cannot be expelled without regular respiration. The pulmonary immune/inflammatory cell host defences, neurological input that regulates airway and vascular tone and controls respiration, local and circulating mediators, and cell-to-cell interaction that regulate lung cell biology are all superimposed on all of this. The regulation of the cells in the regenerated tissue is the final difficulty. These cells may malfunction after being transplanted into a person, which could lead to disorganised tissue as well as, in the most extreme case, malignant transformation.