Airway and lung remodelling in chronic pulmonary obstructive disease: a role for muscarinic receptor antagonists?

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Lung tissue remodelling in chronic inflammatory lung diseases has long been regarded as a follow-up event to inflammation. Recent studies have indicated that, although airway and lung tissue remodelling is often independent of inflammation, it precedes or causes inflammation. None of the available therapies has a significant effect on airway and lung tissue remodelling in asthma, bronchiectasis, fibrosis and chronic obstructive pulmonary disease (COPD). The goal of stopping or reversing lung tissue remodelling is difficult, as the term summarizes the net effect of independent events, including (1) cell proliferation, (2) cell volume increase, (3) cell migration, (4) modified deposition and metabolism of specific extracellular matrix components, and (5) local action of infiltrated inflammatory cells. The extracellular matrix of the lung has a very high turnover, and thus small changes may accumulate to significant structural pathologies, which seem to be irreversible. The most important question is 'why are pathological changes of the lung structure irreversible and resistant to drugs?' Many drugs have the potential to reduce remodelling mechanisms in vitro but fail in clinical trials. New evidence suggests that muscarinic receptor inhibitors have the potential to improve lung function through modifying tissue remodelling. However, the role of muscarinic receptors in lung remodelling, especially their supportive role for other remodelling driving factors, needs to be further investigated. The focus of this review is the role of muscarinic receptors in lung tissue remodelling as it has been reported in the human lung.

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