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# Collagen Deposition in Lung Parenchyma Driven by Depletion of LYVE-1+ Macrophages Protects Emphysema and Loss of Airway Function

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**Abstract :** Collagen is essential for maintaining lung structure and function, and its remodeling has been associated with respiratory diseases, including chronic obstructive pulmonary disease (COPD). However, the cellular mechanisms driving collagen remodeling and the functional implications of this process in the pathophysiology of pulmonary diseases remain poorly understood. Using a mouse model of Lyve-1 expressing macrophage depletion, we found that the absence of this subpopulation of tissue-resident macrophage led to the preferential deposition of type I collagen fibers around the alveoli and bronchi in the steady state. Further analysis by polarized light microscopy revealed that the collagen fibers accumulating in the lungs depleted of Lyve-1+ macrophages were thicker and crosslinked. A decrease in MMP-9 gene expression and proteolytic activity, together with an increase in Col1a1, Timp-3 and Lox gene expression, accompanied the collagen alterations. Next, we investigated the effect of the collagen remodeling on the pathophysiology of COPD and airway function in mouse lacking Lyve-1+ macrophage exposed chronically to cigarette smoke (CS), a well-established animal model of COPD. We showed that the deposition of collagen protected mouse against the destruction of alveoli (emphysema) and bronchi thickening after CS exposure and prevented loss of airway function. Thus, we demonstrate that interstitial Lyve-1+ macrophages regulate the composition, amount, and architecture of the collagen network in the lungs and that such collagen remodeling functionally impacts the development of COPD. This study further supports the potential of targeting collagen as a promising approach to treating respiratory diseases.

**Keywords :** lung, extracellular matrix, chronic obstructive pulmonary disease, matrix metalloproteinases, collagen
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