Collagen VI (COL6), an extracellular matrix protein (ECM), is important in maintaining the integrity of the tissue. Our recent findings have demonstrated that excess COL6 is present in the lungs of comorbid patients with emphysema and adenocarcinoma. COL6 localized in the pulmonary interstitium is likely to interact with endothelial, epithelial and infiltrated pulmonary macrophages. The hypothesis is that excessive COL6 activates macrophages to promote inflammation, which may exacerbate pulmonary diseases. To test our hypothesis, bone marrow derived macrophages or macrophage cell lines were stimulated with soluble COL6 followed by analysis of activation markers and pro inflammatory cytokines. And results showed an increase in the number of CD86 positive cells and the levels of IL-12 and IFNγ production following stimulation. Taken together, our data have provided a link between increased amounts of COL6 and subsequent immune responses, which may play a role in the pathogenesis of pulmonary inflammatory diseases.

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