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lung fibroblasts increases focal adhesion size and remodeling in the bronchial epithelium. During remodeling of the bronchial epithelium following allergen challenge, the number of focal adhesions (FAs) and the extent of FA remodeling increase within 10 days. In this study, we sought to determine whether interleukin-1beta (IL-1beta) and tumour necrosis factor-alpha (TNF-alpha) play a role in FA remodeling in vitro. Human bronchial fibroblasts were studied as a representative cell type of the bronchial epithelium, because bronchial fibroblasts constitute a major part of the extracellular matrix in the airway. We have previously shown that TNF-alpha increases FA area and reorganizes FAs in a variety of cell types; however, the contribution of other cytokines on FA remodeling is unknown. Here, we have demonstrated that IL-1beta and TNF-alpha activate a number of cell signaling pathways in fibroblasts, including phosphoinositide 3-kinase (PI3-K), mitogen-activated protein kinase (MAPK) and C-Jun N-terminal kinase (JNK). Importantly, we have demonstrated that FAs are enlarged, indicating that cell size plays a role in cell remodeling. Interestingly, we have demonstrated that the extent of FA remodeling correlates with changes in cell size. Taken together, we suggest that IL-1beta, TNF-alpha and FA remodeling may be part of a complex interplay of cell signaling pathways and cell-matrix interactions.) Relative positioning of a conventional window display with respect to the ground plane and/or the top surface of a merchandising shelf. André, et al. U.S

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