The role of Myeloid-derived suppressor cells (MDSCs) in the pathogenesis of bleomycin-induced pulmonary fibrosis.

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Idiopathic pulmonary fibrosis (IPF) is a devastating disease characterized by the histopathological pattern of usual interstitial pneumonia and is associated with a high mortality rate. Although the mechanisms underlying IPF pathoetiologies remain poorly understood, M2 macrophages are believed to play a critical role in pulmonary fibrosis. Several papers have shown that myeloid-derived suppressor cells (MDSCs), which are anti-inflammatory cells as well as M2 macrophages, are increased in the peripheral bloods from IPF patients. In our previous study, we also found that MDSC in bone marrows and lungs from bleomycin (BLM)-treated mice were increased. In the present study, therefore, we examined the role of MDSC in the pathogenesis of pulmonary fibrosis. Administration of anti-Gr-1, a MDSC inhibitor, to BLM-treated mice decreased fibrosis area in the lungs. In contrast, intravenous administration of MDSC enhanced BLM-induced lung fibrosis and expression of α-SMA. These results strongly suggested that MDSCs act as malignant factor promoting the pulmonary fibrosis, as well as M2 macrophage. Now we are investigating the feature of MDSC, using in vitro co-culture system of MDSCs and fibroblasts.