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Title: Promotion of lung cancer progression by COPD-like chronic inflammation in a mouse model

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Body: Background: Chronic inflammation has been suggested as a central role in pathogenesis of lung cancer in COPD patients, the knowledge is still in its infancy. We investigated whether the COPD-like airway inflammation can promote the progression of lung cancer using a COPD mouse model, to demonstrate the role of inflammation in pathogenesis of lung cancer combined with COPD. Methods: Adult mouse model of COPD was induced by intratracheal instillation of LPS and exposure to cigarette smoke. To validate this model, we tested the mouse lung function, examined the Lung histology, detected some inflammatory markers in plasma, counted the white cells in BAFL. Then, EGFP-transfected lewis cells were introduced into the lung of COPD mouse and normal mouse by directly puncture through the skin intercostal space. All the mice were scanned by animal fluorescence imaging instrument to observe the progression of lung cancer. Results: All the detecting of cigarette smoke and LPS-induced mouse showed that they had features of COPD and chronic inflammation. Compared with the normal mouse, lung cancer in cigarette smoke and LPS-induced mouse developed more quickly and metastasized much earlier. Mouse of COPD combined lung cancer have shorter survival time than those of lung cancer without COPD. IL-6, was significantly higher in the serum of cancer combined with COPD-like mouse model than the COPD-like mouse model and normal mouse. Conclusions: We have successfully developed a mouse model with COPD-like airway inflammation. The COPD-like inflammation can promote the progression and metastasis of lung cancer in vivo, and IL-6 may play an important role in this process.