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The inflammation associated with lung cancer is mediated by sphingosine-1-phosphate

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Abstract

Background. Epidemiological evidence indicate that almost 40% of COPD patients develop lung cancer; whereas cigarette smoke (CS), the leading cause and the most common risk factor for both COPD and lung cancer, is at the basis of almost 90% of lung cancer establishment.

Aims and objectives. In our previous study we demonstrated AIM2 inflammasome drives the release of pro-inflammatory and pro-fibrotic factors involved in the exacerbation stage of COPD and that it could play a pro-carcinogenic role in lung cancer, in that its activation in tumor-associated plasmacytoid dendritic cells (pDCs) is associated to lung tumor cell proliferation.

Methods. We took advantage of a mouse model of COPD induced by CS inhalation. We compared the murine data to human lung adenocarcinoma-derived samples stratified according to the smoking and COPD status.

Results. We demonstrated that the exposure to first-hand smoking leads to emphysematous changes typical of human COPD, associated to bronchial tone impairment, collagen deposition and IL-1-like cytokine release. We found that AIM2 was involved in smoking-induced lung inflammation and COPD-like features in that its expression was higher in lung recruited DCs and macrophages of smoking mice compared to mice exposed to filtered air (control group). On the other hand, we found that AIM2 expression was higher in the cancerous tissues of lung cancer patients than non-cancerous (normal) tissues, independently of COPD and smoking status. Interestingly, higher expression of AIM2 in non-cancerous tissues of smoker lung cancer patients with COPD was associated to a higher hazard ratio of lower survival rate than patients who presented lower levels of AIM2.

Conclusions. Our data imply that AIM2 inflammasome plays a role at the crosstalk between smoke-induced COPD and lung cancer, affecting patients' survival.

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Biography

Dr. Michela Terlizzi, PhD, is actually a Research Scientist at the Department of Pharmacy of the University of Salerno (Italy). Dr. Terlizzi's research activity over the years has involved the following lines of research providing an important contribution to the respiratory system physiology field: Study of molecular and cellular pathways involved in the alteration of physiological respiratory function following exposure to carcinogens, cigarette smoke and environmental pollutants both in in vivo models and in human samples; Identification of new inflammatory pathways involved in diseases of the respiratory system.

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