Precancer markers identified in airway epithelium cells of healthy smokers

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Smoking may be associated with the development of molecular features of cancer in the large airway epithelium. In the small airway epithelium, molecular cancerization is associated with development of chronic obstructive pulmonary disease, according to recent data.

"We are striving to find the earliest molecular changes that are induced by environmental stressors — in this case, smoking," said Renat Shaykhiev, M.D., Ph.D., assistant professor of genetic medicine at Weill Cornell Medical College, who presented the findings at the AACR-IASLC Joint Conference on Molecular Origins of Lung Cancer: Biology, Therapy and Personalized Medicine, held Jan. 8-11, 2012. "Our goal is to understand the early pathogenesis of lung cancer and to develop strategies to prevent lung cancer in susceptible individuals."

Shaykhiev and colleagues analyzed the large and small airway epithelia of healthy nonsmokers, healthy smokers and smokers with chronic obstructive pulmonary disease (COPD), which is typically caused by long-term smoking, for expression of so-called "molecular cancerization" features (i.e., the genes upregulated in lung cancer compared with nonmalignant adjacent tissue).

Researchers found significantly more cancer-like gene expression changes in the large airway epithelia of smokers than in those of nonsmokers. When analyzing the small airway epithelium, though, they did not find significant differences between healthy smokers and nonsmokers, but they did find significant overall upregulation of

cancerization genes in smokers with COPD. Analysis of these genes in the large and small airway epithelia obtained from the same individuals revealed that molecular cancerization occurs more frequently in the large airway epithelium than in the small airway epithelium.

Shaykhiev and colleagues drew the following conclusions: Smoking is associated with acquisition of molecular cancerization features in the large airway epithelium prior to the development of disease, and the large airway epithelium is likely more susceptible to smoking-induced changes than the small airway epithelium, implying that it may be the primary site of molecular alterations leading to lung cancer in smokers.

These findings could potentially lead to the development of a diagnostic test that would look for these genetic changes in susceptible individuals, the researchers suggested.

"Ideally, we would use these genes to do very routine analysis to determine which smokers or even nonsmokers are at risk for development of lung cancer," said Shaykhiev.

Provided by American Association for Cancer Research

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