In never-smokers, air pollution may result in lung cancer.

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Abstract

According to late-breaking research [to be] presented at the ESMO Congress 2022 by scientists of the Francis Crick Institute and University College London, funded by Cancer Research UK, a new mechanism has been identified through which very small pollutant particles in the air may cause lung cancer in people who have never smoked, opening the door to new prevention strategies and the development of therapies. The particles, which are commonly present in fossil fuel smoke and vehicle exhaust, are linked to a higher risk of non-small cell lung cancer (NSCLC), which is responsible for approximately 250,000 lung cancer-related deaths worldwide each year.

Keywords: Lung Cancer, Air pollution, Gene Mutations.

Introduction

The same airborne particles that contribute to climate change by burning fossil fuels also have a significant and hitherto unrecognized influence on human health by inducing cancer in lung cells. Although air pollution poses a lesser risk of lung cancer than smoking does, we cannot control what we all breathe. These new findings demonstrate the significance of tackling climate health in order to improve human health as more people globally are exposed to hazardous levels of air pollution than to toxic compounds in cigarette smoking [1].

The new findings are supported by clinical and laboratory research on EGFR gene mutations, which are present in around 50% of lung cancer patients who have never smoked. In a study including over 500,000 persons who lived in England, South Korea, and Taiwan, exposure to rising levels of airborne particulate matter (PM) with a diameter of 2.5 micrometres (m) was associated with a higher risk of developing NSCLC with EGFR mutations [2].

Researchers from the Francis Crick Institute demonstrated in laboratory investigations that the same pollutant particles (PM2.5) accelerated the transition of airway cells with EGFR and KRAS mutations into a state resembling cancer stem cells. Additionally, they discovered that exposure to PM2.5 causes an influx of macrophages to release the inflammatory mediator interleukin-1, causing the proliferation of cells with EGFR mutations. They also discovered that blocking interleukin-1 prevented the development of lung cancer. These results were in line with data from a prior significant clinical trial that demonstrated a dose-dependent decrease in lung cancer incidence in patients receiving the anti-IL1 antibody canakinumab [3].

In a final set of tests, the Francis Crick team employed ultra-deep mutational profiling to identify EGFR and KRAS driver mutations in 18% and 33%, respectively, in small samples of normal lung tissue. We discovered that the EGFR and KRAS genes, which are frequently mutated in lung malignancies, are also present in healthy lung tissue and are probably brought on by ageing. According to our findings, these alterations weakenedly exacerbated cancer in lab models. But when lung cells with these mutations were exposed to air pollutants, we observed more cancers and these developed faster than when lung cells with these mutations were not exposed to pollutants, indicating that air pollution encourages the initiation of lung cancer in cells carrying driver gene mutations. Finding out why some lung cells with mutations develop cancer when exposed to pollution but not others is the next step [4].

This research raises the question of whether it may one day be able to use lung scans to detect precancerous lesions in the lungs and attempt to treat them with drugs like interleukin-1 inhibitors. Discussions are still quite theoretical because we don't yet know if highly sensitive EGFR profiling on blood or other samples will be able to identify non-smokers who are prone to lung cancer and may benefit from lung scanning [5].

Conclusion

The use of stratified analysis by sex and air quality conditions, as well as the study's high sample size, are its main advantages. The epidemiological risk factors for lung cancer were passive smoking exposure, cooking practices, medical history, and cancer in the family. The self-reporting of smoking status and other factors is a weakness of this study. As a result, never-smokers might have been misclassified, particularly those who were ex-smokers and had quit smoking less than 15 years prior. Lung cancer formation is a complicated process that incorporates a number of variables, including epidemiological, genetic, and gene-environment interactions.

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