

NIH Study Links Particulate Air Pollution to Increased Mutations in Lung Cancers Among Nonsmokers

Whole-genome sequencing study found air pollution to cause more cancer-related changes than secondhand smoke.

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Scientists at the National Institutes of Health (NIH) and their colleagues at the University of California, San Diego, have found that fine-particulate air pollution, which includes pollution from vehicles and industry, was strongly associated with increased genomic changes in lung cancer tumors among people who have never smoked.

By assembling the largest-ever whole-genome analysis of lung cancer in individuals who have never smoked, researchers were able to link air pollution exposure to increased cancer-driving and cancer-promoting genetic mutations. This could potentially lead to more prevention strategies for never-smokers.

Researchers analyzed lung tumors from 871 never-smoker patients across 28 geographic locations worldwide as part of the [Sherlock-Lung study](#). They found associations between air pollution exposure and changes in the TP53 gene, and other genetic mutational signatures previously associated with tobacco smoking. They also observed a relationship between air pollution and shorter telomeres, which are sections of DNA found at the end of chromosomes. Telomeres shorten naturally with age and shorter telomeres are related to cells inability to continue to replicate. However, scientists found fine particulate air pollution was linked to premature shortening of telomeres.

Prior genomic studies of lung cancer have focused on tobacco smokers, leaving a significant gap in our understanding of how lung cancer develops in people who have never used tobacco. By beginning to uncover the mechanisms through which tissues acquire cancer-causing or cancer-promoting mutations following environmental exposures, this study helps scientists better understand the primary drivers of lung cancer in this population—which represents up to 25% of all lung cancer cases globally.

Interestingly, the researchers found that while exposure to secondhand smoke was associated with slightly higher mutation burdens and shorter telomeres, compared to tumors in patients who

were not exposed, it did not lead to an increase in cancer-driving mutations or mutational signatures. This suggests that secondhand smoke may have a lower overall ability to cause genetic mutations, known as mutagenicity, compared to air pollution.

This work was led by researchers at NIH's National Cancer Institute and the University of California, San Diego, and [published in Nature](#) on July 2, 2025.

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