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"Association of particulate matter 2.5 with superficial spreading melanoma incidence in the United States"

Abstract

Particulate matter 2.5 (PM 2.5), or particulate matter less than or equal to 2.5 micrometers in diameter, is a key index of air pollution. Molecular studies have shown that the constituents of PM 2.5—chiefly dioxin and polycyclic aromatic hydrocarbons—mediate tyrosinase expression via the aryl hydrocarbon receptor, thereby promoting melanogenesis, and potentially, malignant melanoma. However, population-based studies assessing the impact of PM 2.5 levels on melanoma incidence in the United States are lacking.

We designed an ecological study using data from the National Cancer Institute's Surveillance, Epidemiology, and End Results (SEER) program and the Centers for Disease Control (CDC) and Prevention. Patients with superficial spreading melanoma (SSM) were identified from SEER-22 (2000-2020) using the International Classification of Diseases for Oncology (ICD-O-3) code 8744 and topographic codes C44.0-44.9 (all skin sites). Only patients with histologically confirmed SSMs were included. Information on PM 2.5 was collected from the CDC and linked to county level data from SEER. US counties were sorted into quartiles of PM 2.5 exposure such that each county had an equal number of individuals at risk. Incidence rate ratios (IRRs) capturing the relationship between PM 2.5 quartile and SSM incidence were computed using Poisson models adjusted for age, race, sex, household income, rural-urban living, and UV index; logarithm of county-level population data was applied as an offset in Poisson models.

We observed SSM incidence to be positively correlated with PM 2.5 (IRR 1.15, 95% CI 1.14-1.15, P < 0.001).

These findings suggest a statistically significant and positive association between ambient PM 2.5 exposure and the incidence of superficial spreading melanoma (SSM) at the population level in the United States. The observed 15% increase in melanoma incidence across PM 2.5 quartiles, independent of established risk factors such as age, sex, race, UV exposure, income, and rurality, supports a potential role for air pollution in melanoma pathogenesis. These results align with molecular studies indicating that PM 2.5 components, such as polycyclic aromatic hydrocarbons and dioxins, may promote melanogenesis through activation of the aryl hydrocarbon receptor pathway. While the ecological design limits causal inference, this analysis highlights a novel environmental risk factor for melanoma that warrants further investigation in longitudinal and mechanistic studies. Public health efforts aimed at air quality improvement may thus have implications beyond cardiopulmonary disease, potentially extending to melanoma prevention.