
**GEOSPATIAL ASSOCIATIONS BETWEEN
ENVIRONMENTAL CONTAMINATION AND CANCER
INCIDENCE IN UKRAINE**

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INTRODUCTION

Cancer is a leading cause of morbidity and mortality worldwide, with growing attention focused on its environmental determinants. In Ukraine cancer is a major public health challenge, ranking second among causes of mortality and accounting for nearly 10% of all deaths^{1,2}. While individual risk factors such as genetics and lifestyle remain critical, an increasing body of evidence highlights the spatial variability of cancer incidence and its potential association with environmental exposures. In this context, geographic disparities in cancer outcomes are not merely statistical artifacts but may reflect underlying differences in pollution levels, urbanization, access to healthcare, and socioeconomic structure. Understanding the spatial distribution of cancer and its correlation with environmental pressures is essential for effective public health interventions, especially in regions undergoing ecological, industrial, and geopolitical transitions.

1. Methodological Approaches to Investigating Regional Cancer Burden and Environmental Risk Factors

Ukraine presents a unique case for spatial epidemiological analysis of cancer. As a post-industrial nation with significant environmental burdens – including legacy pollution, widespread agricultural chemical use, and residual radioactive contamination – Ukraine exhibits marked regional disparities in oncological outcomes. These disparities are further compounded by

¹ Kornus O., Kornus A., Skyba O., Mazhak I., Budnik S. (2023). Forecasting the population mortality rate from cardiovascular diseases as a condition of the economic security of the state. *Georgian Med News*. 6(339):62–66.

² Kornus O., Kornus A., Skyba O., Shyshchuk V., Pshenychna L., Danylchenko O. (2022). Nosogeographic assessment of the adult population morbidity of the Sumy region (Ukraine). *J Geol Geogr Geocool* 31(1):89–98. DOI: <https://doi.org/10.15421/112209>

recent armed conflict, which has disrupted healthcare systems, population distribution, and environmental monitoring in affected areas. Despite this complexity, comprehensive spatial studies integrating cancer epidemiology and environmental data remain limited.

This study addresses this gap by applying a geospatial approach to analyze the relationship between cancer incidence and environmental risk factors across 26 administrative regions of Ukraine. Using regional-level data and spatial statistical methods, we explore whether specific environmental indicators – such as emissions of methane, sulfur dioxide, particulate matter, and ammonia – are associated with elevated cancer burdens. By combining epidemiological metrics with environmental pollution datasets and visualizing their geographic patterns, we aim to identify ecologically vulnerable regions and highlight potential environmental drivers of cancer disparities in Ukraine.

The aim of this study is to assess regional variations in cancer indicators in Ukraine and examine their correlation with selected environmental pollutants. By combining medical statistics and environmental monitoring data, this study contributes to understanding the geographical inequalities in cancer burden and the potential environmental drivers of these disparities. Geographical analysis of cancer incidence allows us to identify vulnerable regions, understand the causes of high incidence, and develop effective prevention strategies, which are key to improving the healthcare system and the quality of life of the population.

The analysis encompasses twenty-six administrative regions of Ukraine, covering both urban and rural populations, and reflects the most recent pre-war period for which complete datasets were available (2018–2022). Cancer incidence, prevalence, and mortality data were obtained from the National Cancer Registry of Ukraine, which provides annual regionally disaggregated statistics by sex, age cohort, and diagnostic category. Socio-demographic information, including the distribution of urban and rural populations, as well as population density metrics, was sourced from the State Statistics Service of Ukraine. Environmental variables were derived from several official datasets, including the regional Ecological Passports issued by the Ministry of Environmental Protection and Natural Resources, the annual statistical compendium Environment of Ukraine, and radiation safety data from the Uatom platform. All medical indicators were standardised per 100,000 inhabitants to ensure comparability across regions with divergent population structures.

The analytical framework integrated descriptive summaries with inferential statistical procedures. Descriptive measures were used to outline regional profiles of oncological burden, while Pearson's correlation coefficients were employed to evaluate the strength and direction of associations between cancer metrics and environmental parameters. The significance of correlation

coefficients was tested with a two-tailed t-test based on twenty-four degrees of freedom, corresponding to the number of regional observations. Correlations meeting or exceeding $r = 0.404$ were treated as statistically significant at the 95% confidence threshold ($p < 0.05$), whereas coefficients of $r \geq 0.340$ were considered marginally significant at the 90% level ($p < 0.10$), allowing the study to capture weaker yet potentially meaningful relationships.

The medical component of the dataset comprised fifty distinct oncological indicators. These included general measures of cancer incidence and mortality for the total, urban, and rural populations, as well as for male, female, working-age, and paediatric cohorts. Site-specific incidence indicators covered a broad spectrum of malignant neoplasms, including cancers of the trachea, bronchus, and lung; stomach; prostate; breast; uterine body and cervix; ovary; skin (melanoma and non-melanoma); lip, oral cavity, and pharynx; oesophagus; colon and rectum; liver and pancreas; larynx; kidney and bladder; thyroid; brain; and bone and joint cartilage. Haematological malignancies were represented through incidence data on Hodgkin lymphoma, non-Hodgkin lymphomas, multiple myeloma, and leukaemias. Prevalence indicators included the number of patients under active medical follow-up at the end of each year, both in aggregate and for specific cancer sites, including breast and uterine cancers, skin tumours, and childhood leukaemias and lymphomas. Mortality parameters captured total deaths from malignant neoplasms, as well as mortality in working-age and paediatric groups.

The environmental dataset comprised twenty-five indicators reflecting major categories of anthropogenic pressure. Air quality parameters included emissions from stationary sources of sulphur dioxide, nitrogen dioxide, carbon monoxide, non-methane volatile organic compounds, ammonia, methane, carbon dioxide, suspended particulate matter, cyanides, fluorine-, chlorine-, and bromine-containing compounds, freons, and persistent organic pollutants. Total emissions were analysed in absolute terms and normalised per unit area and per capita. Radioactive contamination was assessed using regional surface contamination by ^{137}Cs and ^{90}Sr across specified activity ranges. Waste-related indicators included per-capita generation of hazardous wastes (classes I–III) and low-hazard wastes (class IV). Water quality pressures were represented by the annual volume of polluted wastewater discharged within each region.

To support the interpretation of statistical and spatial patterns, correlation outcomes were visualised using cartographic and graphical tools. Choropleth maps produced in GIS software illustrated the spatial distribution of cancer indicators and environmental pressures across regions. Complementary scatterplots and heatmaps were generated in Python (version 3.11) using the `seaborn` and `matplotlib` libraries within the Google Colaboratory environment.

These visualisations enabled the identification of pollutant groups exerting the strongest influence on spatial variation in oncological indicators and provided analytical support for the interpretation.

2. Spatial Epidemiology of Cancer in Ukraine: Environmental Determinants, Demographic Structure, and Urban–Rural Inequalities

Regional Differences in Primary Cancer Morbidity in Ukraine

As of January 1, 2023, the primary cancer incidence rate in Ukraine was 233.8 cases per 100,000 population. Regional differences in cancer incidence reflect a kind of “geographical mirror” of environmental challenges, demographic realities, and the consequences of war. A high level of cancer incidence has been recorded in 11 regions of Ukraine, with Cherkasy, Kirovohrad, Khmelnytskyi, Chernihiv, and Sumy regions standing out in particular. In Cherkasy, Kirovohrad, and Khmelnytskyi – located in the central part of the country – agricultural production has traditionally been one of the leading economic sectors. Intensive use of pesticides and other agrochemicals, combined with emissions from local industrial facilities, creates an environmental context that may be conducive to the development of malignant neoplasms. An additional contributing factor in Kirovohrad is elevated background radiation due to the presence of uranium deposits, which may significantly affect population health. In Chernihiv and Sumy regions, oil and natural gas extraction is ongoing and is associated with environmental contamination by carcinogenic substances such as polycyclic aromatic hydrocarbons (PAHs), volatile organic compounds (VOCs), and PM.

These pollutants are linked to an increased risk of lung and other respiratory cancers. Moreover, oil spills, drilling fluid leaks, and improper handling of industrial waste contribute to the contamination of soil and surface waters, facilitating the entry of toxic compounds into the food chain. This route of exposure is critically important given the potential for chronic carcinogenic effects. The release of heavy metals and chemical compounds during oil and gas extraction may damage cellular DNA, induce mutations, and initiate carcinogenesis. Workers in the extractive sector are also subject to prolonged exposure to toxic agents, thereby increasing their risk of cancer development. In addition to technogenic factors, socio-demographic characteristics also play a significant role.

Central regions of Ukraine are characterized by a higher proportion of elderly individuals, who belong to the age group most vulnerable to cancer. Further contributing factors include smoking, poor diet, sedentary lifestyle, and elevated stress levels – all of which may increase oncological risk. The interaction of these factors can lead to a cumulative effect, whereby simultaneous exposure to multiple toxic agents substantially elevates the probability of

malignant disease³⁴. Another important explanation for elevated cancer rates may lie in more efficient healthcare systems. Regions with well-developed medical infrastructure and active cancer screening programs tend to register more cases due to earlier and more frequent detection, thus increasing recorded incidence rates. Therefore, the spatial patterns observed are shaped not only by biophysical, ecological, and industrial factors, but also by social environments, demographic structures, and healthcare quality.

The combined influence of these elements creates conditions for cumulative cancer risk. The interaction of agrochemical exposure, atmospheric pollution, socio-economic factors, and the presence of effective diagnostic systems increases both the likelihood of developing cancer and the chances of its detection. As such, elevated cancer rates in these regions reflect a multifactorial interplay that requires interdisciplinary approaches to analysis, prevention, and public health response. At the same time, in 2022, the lowest number of newly diagnosed cancer cases was reported in the Zaporizhzhia, Kherson, Mykolaiv, Kharkiv, Donetsk, and Luhansk regions. The seemingly more favorable situation in these areas is likely attributable to the ongoing war, which has led to the destruction of healthcare infrastructure, large-scale population displacement, and widespread disruptions in medical services. These factors have significantly hampered cancer detection and registration efforts, making it extremely difficult to assess the true epidemiological burden in the affected territories.

Overall, these spatial patterns underscore the multifactorial nature of cancer risk in Ukraine, where environmental exposures, demographic aging, unequal access to healthcare, and socioeconomic disparities collectively shape regional cancer burdens. A nuanced understanding of these complex interactions is essential for guiding evidence-based policy decisions and designing targeted prevention strategies that reflect regional needs and vulnerabilities. The study of regional differences in the prevalence of malignant neoplasms among urban and rural populations is a pressing issue, as such studies allow for a more comprehensive assessment of the impact of socioeconomic, environmental, and demographic factors on cancer incidence. Spatial differentiation of indicators among different types of settlements makes it possible to identify not only the specifics of morbidity, but also the factors that contribute to its formation in certain population groups. The lowest rates of primary cancer incidence among urban residents were recorded in Mykolaiv region (166.2 cases per 100,000 population), Donetsk (141.9), Kharkiv (71.3), and Luhansk (34.7).

³ Cancer Topics (2025). International Agency for Research on Cancer. URL: <https://www.iarc.who.int/cancer-topics/>

⁴ EPA Efforts to Reduce Exposure to Carcinogens and Prevent Cancer (2025). URL: <https://www.epa.gov/environmental-topics/epa-efforts-reduce-exposure-carcinogens-and-prevent-cancer>

These figures should be interpreted with caution, as ongoing military conflict, population displacement, and restricted access to healthcare in these regions may have resulted in significant underreporting and gaps in medical surveillance systems. In contrast, the regions with the highest levels of primary cancer incidence in urban areas include Kirovohrad (430.7), Cherkasy (402.8), and Khmelnytskyi (380 per 100,000 population). Among rural populations, the highest rates were also found in Khmelnytskyi (356), Cherkasy (356), Kirovohrad (335.9), Chernihiv (322.3), and Sumy (316 per 100,000 population) regions. Notably, Kirovohrad, Cherkasy, and Khmelnytskyi regions consistently exhibited elevated incidence rates across both urban and rural populations, indicating a broader regional influence of environmental and socio-economic risk factors. In contrast, the high rural incidence in Chernihiv and Sumy may be partially explained by the aging demographic structure of these areas, where a large proportion of the population resides in villages with limited access to early cancer detection and healthcare services.

Regional Inequalities in Cancer Prevalence in Ukraine

In 2022, the overall prevalence of malignant neoplasms among the urban population of Ukraine was 2,972 cases per 100,000 population. However, this rate varied considerably across regions. The lowest prevalence levels were recorded in Dnipropetrovsk (2,891.9), Rivne (2,856.0), Ivano-Frankivsk (2,820.3), Transcarpathian (2,563.4), and Donetsk (1,377.9 per 100,000 population) regions. The prevalence of cancer is significantly higher among city residents, especially in Cherkasy (4,287.8 per 100,000 population), Sumy (4,185.8), and Mykolaiv (4,124.6) regions. These disparities are likely driven by a combination of factors, including environmental conditions, population structure, the accessibility and effectiveness of healthcare services, and the quality of cancer diagnostics and case registration.

Among rural populations, the national average cancer prevalence was 2,422.1 cases per 100,000 individuals. Fourteen regions exceeded this benchmark, with the highest rates reported in Kyiv (3,435.7), Cherkasy (3,174.7), Mykolaiv (3,108.5), Sumy (2,929.2), and Vinnytsia (2,871.9) regions. On the other end of the spectrum, the lowest rural cancer prevalence was observed in Rivne (1,972.4), Transcarpathian (1,746.4), and Donetsk (565.9) regions. Elevated prevalence in Kyiv, Cherkasy, Sumy, Vinnytsia, and Mykolaiv regions may be attributed to a combination of factors, including better access to medical diagnostics, a more developed healthcare infrastructure, ecological pressures from intensive agricultural activity (e.g., the use of fertilizers and pesticides), aging rural populations, and higher awareness of cancer risks among primary care providers.

In contrast, the low incidence rates observed in Rivne, Transcarpathian, and Donetsk regions are more likely to reflect limited access to medical services, a low

level of cancer detection, high labor migration (particularly in Transcarpathian), as well as incomplete statistical reporting due to ongoing hostilities and the loss of territorial control (as in the case of Donetsk). Additionally, ethnocultural characteristics and dietary traditions specific to certain western Ukrainian regions may have an indirect positive effect on population health.

Urban–Rural Correlation in Cancer Prevalence

To assess the relationship between cancer incidence rates in urban and rural areas, Pearson correlation coefficients were calculated for three variables: cancer prevalence in urban populations, in rural populations, and the difference between them, represented as a new variable “Difference”. The analysis revealed a very strong positive correlation ($r = 0.98$) between urban and rural cancer prevalence rates, indicating that regions with high rates among urban residents generally also exhibit elevated rates among rural populations.

Despite differences in absolute values, the close correlation suggests that the underlying regional risk factors— whether environmental, demographic, or related to healthcare access – are similar for both population groups. For instance, in Cherkasy and Kirovohrad regions, the prevalence among urban residents was 4,287.8 and 4,124.6 per 100,000, respectively, while rural rates were 3,174.7 and 3,108.5. Although the absolute differences exceed 1,000 cases, the proportional difference is moderate, reflecting a shared exposure to regional cancer determinants. The correlation between urban prevalence and the urban–rural difference was moderately strong ($r = 0.61$), suggesting that higher urban rates are often accompanied by a greater disparity between urban and rural groups. This may reflect the influence of urban-specific factors such as industrial emissions, lifestyle differences, or more advanced diagnostic infrastructure. In Mykolaiv and Odesa regions, for example, the urban prevalence was 3,872.3 and 3,796.2, respectively, while rural figures were 2,391.7 and 2,399.3 – yielding differences of over 1,400 cases per 100,000 population. These disparities likely point to additional urban environmental or socio-behavioral risks.

In contrast, the correlation between rural prevalence and the urban–rural difference was weaker ($r = 0.43$), indicating that variation in rural cancer rates had a smaller impact on the observed disparities. This may be due to the typically lower and less variable prevalence among rural populations, as well as barriers to healthcare access and underdiagnosis in rural settings. Analysis of regional cancer data in Ukraine revealed consistently higher prevalence rates in urban areas compared to rural counterparts. While absolute values varied across regions, the urban–rural pattern was evident in most territories. The most pronounced differences were observed in regions such as Mykolaiv and Odesa, where urban cancer prevalence exceeded rural levels by more than 1,400 cases per 100,000 population. Higher cancer prevalence in urban areas

can be attributed to several interrelated factors. Urban populations typically benefit from better access to medical services, including a greater density of healthcare facilities, specialized diagnostic infrastructure, and oncological centers, which enhances early detection and increases reported prevalence. Additionally, environmental conditions in industrialized cities – such as air, water, and soil pollution – may elevate actual cancer risk through prolonged exposure to carcinogenic substances.

Lifestyle-related factors more common in urban settings, including higher stress levels, physical inactivity, unbalanced diets, and greater use of tobacco and alcohol, may also contribute to the higher cancer burden. Conversely, the lower prevalence observed in rural areas may not necessarily reflect lower true incidence. Rather, it may result from limited access to modern diagnostics and specialized care, which contributes to underdetection and delayed diagnosis. Furthermore, demographic differences – particularly a younger age structure in some rural regions – may partially explain the lower observed prevalence. The strong correlation between urban and rural cancer prevalence ($r = 0.98$) underscores the influence of shared regional risk factors affecting both settlement types. However, the presence of substantial absolute differences in certain regions suggests an additional impact of urban-specific conditions. In particular, high urban prevalence not only increases the overall disease burden but also widens the urban–rural gap—likely due to a combination of more intense exposure to risk factors and more efficient diagnosis in cities. Taken together, these findings highlight the importance of interpreting cancer prevalence data in the context of both regional and settlement-type characteristics, including healthcare accessibility, diagnostic infrastructure, and environmental conditions. They also support the need for tailored cancer control strategies that address urban-specific exposures as well as persistent healthcare barriers in rural areas.

The demographic structure of the population, particularly the proportion of older residents, plays a critical role in shaping cancer risk. In addition, lifestyle-related factors such as smoking, unbalanced diet, low physical activity, and chronic stress may contribute to increased incidence in certain areas. In several regions, the interplay of multiple risk factors – agricultural chemical exposure, industrial emissions, and socio-economic vulnerability – creates a cumulative effect that may explain the elevated cancer burden.

Demographic and Gender-Specific Patterns in Cancer Prevalence.

The demographic structure of the population, particularly the proportion of older individuals, plays a significant role in shaping cancer risk. Regions with an older age profile tend to exhibit higher prevalence rates, while a younger population structure may partly explain lower cancer burden in certain areas. Age remains one of the most influential risk factors for cancer due to the

accumulation of genetic mutations and prolonged exposure to environmental and lifestyle-related carcinogens over time. Gender-related differences in cancer prevalence are also well established and follow patterns observed in many other countries. Men tend to have higher overall cancer prevalence, which is often linked to both behavioral risk factors and occupational exposure. Among men, lung and esophageal cancers are more frequently diagnosed, reflecting these combined influences. In contrast, women are more often affected by hormone-dependent cancers, including reproductive system malignancies, which are shaped by endocrine, reproductive, and genetic factors. Childhood cancer presents a different epidemiological profile, with leukemia, lymphomas, and brain tumors being the most commonly diagnosed malignancies. As of January 1, 2023, the incidence rate of childhood cancer in Ukraine was 89.1 per 100,000 population. The highest incidence rates among children were observed in Dnipropetrovsk (100.4), Sumy (104.7), Poltava (108.6), Lviv (113.5), Khmelnytskyi (114.6), Cherkasy (145.9) regions and the city of Kyiv (104.5 per 100,000 people). This can be explained not only by environmental or medical and social factors, but also by the peculiarities of the organization of diagnostics.

3. Statistically Supported Associations Between Environmental Pollution and Cancer Incidence in Ukraine

To investigate potential associations between environmental quality indicators and cancer incidence, a total of 1,250 pairwise Pearson correlation coefficients were calculated. From this dataset, 69 coefficients with moderate to strong positive correlations ($r \geq 0.3$) were selected for further statistical validation. The significance of these correlations was assessed using t-statistics and corresponding p-values, based on regional data from 26 Ukrainian regions.

Out of the 69 correlations analyzed, 13 were statistically significant at the 90% confidence threshold ($p < 0.10$). These results indicate that a portion of the observed associations between environmental factors and cancer incidence can be considered statistically substantiated, which probably reflects the effects of chronic exposure to environmental carcinogens. The analysis suggests that certain types of cancer may be influenced by environmental exposures – particularly air and water pollution. Statistically significant correlations were found between cancer incidence and emissions of specific hazardous substances. Visualization of the correlation matrix enabled clear identification of the strongest associations that were statistically significant at the 95% confidence level ($p < 0.05$). Among the 23 significant correlations, several environmental variables appeared repeatedly, indicating the presence of potentially robust and consistent relationships.

Specifically, significant positive correlations were found between the total number of cancer patients and emissions of methane ($r = 0.582$), sulfur dioxide ($r = 0.466$), and PM ($r = 0.508$). These associations may indicate a possible synergistic effect of combustion byproducts, which are known markers of industrial pollution. Similar relationships were confirmed when analyzing urban populations separately: the correlation between cancer prevalence in cities and methane emissions reached $r = 0.598$, and for particulate matter, $r = 0.534$.

In rural areas, a statistically significant correlation was also identified between cancer incidence and nitrogen dioxide emissions ($r = 0.431$), which may be linked to local waste burning, agrochemical use, or transboundary transport of pollutants.

A notable association was observed between air pollution and the incidence of malignant neoplasms of the breast. This cancer type demonstrated statistically significant positive correlations with three major pollutants: methane emissions ($r = 0.527$), sulfur dioxide ($r = 0.479$), and total atmospheric emissions from stationary sources ($r = 0.457$). The multifactorial nature of these associations may suggest a complex endocrine-modulating effect of industrial pollutants, which could suppress immune surveillance or exert genotoxic effects on target tissues.

Similar patterns were found for skin cancers, which showed significant correlations with both methane emissions ($r = 0.509$) and overall air pollutant emissions ($r = 0.423$). These associations may be explained by the photoreactive properties of air pollutants that enhance ultraviolet-induced damage, as well as the direct harmful effects of NMVOCs accumulating in urban air.

Several statistically significant associations were also observed for lymphoproliferative malignancies. In particular, the incidence of Hodgkin lymphoma was positively correlated with emissions of PM ($r = 0.430$) and carbon monoxide ($r = 0.418$ – 0.452), suggesting a possible role of chronic airway inflammation or immunosuppression as intermediate mechanisms. Non-Hodgkin lymphomas also showed a correlation with methane emissions ($r = 0.427$), which, considering the properties of this compound, may reflect the impact of high environmental concentrations of organic degradation products in residential settings.

Notably, some of these associations involved recurring links with specific ecological indicators, such as total emissions of pollutants, concentrations of suspended particulate matter, sulfur dioxide, and methane. The repeated appearance of these variables suggests a certain degree of stability in the observed associations, particularly in regions characterized by intensive industrial activity or elevated anthropogenic pressure.

Methane emissions were the most frequently recurring factor, significantly correlated with six cancer indicators: the total number of registered cancer

patients, the number of cases among urban residents, and the incidence of breast cancer, skin cancer, uterine cancer, and non-Hodgkin lymphomas. This pattern may reflect the widespread release of methane from industrial and agricultural sources and its co-emission with other hazardous substances.

Sulfur dioxide emissions showed four significant correlations, including associations with the total cancer registry population, cancer incidence among urban residents, and the incidence of breast and uterine cancers. Similarly, the total volume of air pollutant emissions was significantly associated with four cancer types: breast, skin, brain, and uterine cancers. These indicators reflect general environmental load and may act as integrative markers of regional pollution pressure.

PM was associated with three indicators: total registered cancer cases, urban cancer prevalence, and Hodgkin lymphoma. NMVOCs were significantly associated with cancer mortality and Hodgkin lymphoma, highlighting the potential role of photochemical smog and organic pollutants in cancer outcomes.

Other environmental indicators, such as bromine compounds, carbon monoxide, nitrogen dioxide, and generation of class I–III hazardous waste, each demonstrated a statistically significant correlation with at least one cancer indicator. Although these associations were less frequent, their presence within the most statistically robust subset warrants further toxicological and epidemiological investigation.

4. Environmental Pollution and Its Multidimensional Associations with Cancer Incidence in Ukraine

The observed positive correlation between atmospheric ammonia emissions and the overall cancer incidence rate – particularly among men – is consistent with findings from previous studies indicating the potential toxicity of ammonia as an indicator of agricultural pollution^{5,6}. The correlation coefficients ($r \approx 0.343\text{--}0.346$) suggest that ammonia emissions may serve not only as a direct irritant or toxicant, but also as a proxy for broader environmental stressors associated with intensive livestock farming and fertilizer use. Ammonia released into the air from agricultural sources may reflect a composite burden of co-emitted pollutants, some of which possess immunosuppressive or mutagenic potential when chronically inhaled. Its recurring association with general cancer

⁵ Roney N., Lladós F., Little S.S., Knaebel D.B. (2004). Toxicological profile for ammonia, Agency for Toxic Substances and Disease Registry, 269 p.

⁶ Wyer K.E., Kelleghan D.B., Blanes-Vidal V., Schaubberger G., Curran T.P. (2022). Ammonia emissions from agriculture and their contribution to fine particulate matter: A review of implications for human health. *J Environ Manage* 323:116285.

morbidity across the dataset supports the hypothesis of a cumulative effect or interaction with other environmental carcinogens.

In addition to ammonia, a statistically significant correlation was observed between male cancer incidence and area contaminated with ^{90}Sr at 5.55–111 kBq/m² ($r = 0.351$). This reinforces existing evidence regarding the long-term health impacts of residual radionuclide contamination from the Chernobyl disaster^{7,8}. ^{90}Sr is a beta-emitting isotope that accumulates in bone tissue and has been identified as a risk factor for malignancies, particularly those affecting the hematopoietic and musculoskeletal systems^{9,10}. Given its long physical and biological half-life, ^{90}Sr may exert delayed carcinogenic effects – especially in male populations, who may have been more occupationally exposed in historically irradiated territories.

Among the specific cancer types demonstrating significant positive correlations with PM emissions, pharyngeal cancer ($r = 0.351$) and pancreatic cancer ($r = 0.374$) warrant particular attention. These associations are consistent with the hypothesis of systemic toxicity associated with aerosol pollution^{11,12}. In the case of pharyngeal cancer, the respiratory tract represents the primary site of exposure to inhaled particulates. For pancreatic cancer, the observed association may reflect an indirect pathway, whereby fine and ultrafine particles (e.g., PM_{2.5}, PM₁₀) are capable of translocating from the alveoli into the bloodstream, acting as vectors for adsorbed carcinogens such as PAHs and heavy metals. These particles can reach distant organs, including the pancreas, which is known to be highly sensitive to both endogenous and exogenous toxic insults¹³.

⁷ Repin V., Novak N., Perevoznikov O., Tsygankov N. (2000). Retrospective estimation of strontium-90 intake dynamics and doses for the population living in the territories affected by the Chernobyl accident. *Radiat Prot Dosimetry* 88(3):207–221.

⁸ Christodouleas J.P., Forrest R.D., Ainsley C.G., Tochner Z., Hahn S.M., Glatstein E. (2011). Short-term and long-term health risks of nuclear-power-plant accidents. *N Engl J Med* 364(24):2334–2341.

⁹ Sources and Effects of Ionizing Radiation (2000). Report to the General Assembly, United Nations. United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR), New York, Volume I: Sources, 657 p.

¹⁰ Glasco A.D., Snyder L.A., Paunesku T., Howard S.C., Hooper D.A., Golden A.P., Woloschak G.E. (2024). Revisiting the historic Strontium-90 ingestion Beagle study conducted at the University of California Davis: Opportunity in archival materials. *Radiat Res* 202(2):289–308.

¹¹ Peleman J., Ruan M., Dey T., Chiang S., Dilger A., Mitchell B., Jung Y.S., Ramanathan M., Mady L., Yu S., Cramer J., Lee S.E. (2024). Air pollution exposure and head and neck cancer incidence. *Sci Rep* 14:26998.

¹² Bhavsar N.A., Jowers K., Yang L.Z., Guha S., Lin X., Peskoe S., McManus H., McElroy L., Bravo M., Reiter J.P., Whitsel E., Timmins C. (2025). The association between long-term PM_{2.5} exposure and risk for pancreatic cancer: an application of social informatics. *Am J Epidemiol* 194(3):730–737.

¹³ Loomis D., Grosse Y., Lauby-Secretan B., El Ghissassi F., Bouvard V., Benbrahim-Tallaa L., Guha N., Baan R., Mattedo H., Straif K. (2013). The carcinogenicity of outdoor air pollution. *Lancet Oncol* 14(13):1262–12.

A distinct group of correlations was identified between NMVOCs and cancers of the bladder ($r = 0.395$), breast ($r = 0.341$), and corpus uteri ($r = 0.369$). These associations are biologically plausible, given that numerous NMVOCs – such as benzene, toluene, and formaldehyde – are classified as carcinogenic and have been shown to affect both reproductive and urinary tract tissues^{14,15}. Due to their lipophilic properties, some of these compounds tend to accumulate in adipose tissue or penetrate the pelvic organs via systemic circulation. Their impact may occur through direct inhalation exposure, or via metabolic activation followed by DNA damage. These findings are supported by toxicological and epidemiological evidence indicating the carcinogenic potential of benzene, formaldehyde, and acrolein in relation to breast and bladder cancers^{16,17}. The likely sources of such compounds include industrial emissions and motor vehicle exhaust, further implicating urban air pollution as a shared risk factor across multiple cancer types.

In this study, significant correlations were also observed between freon emissions and both skin cancer incidence ($r = 0.378$) and childhood mortality from malignant neoplasms ($r = 0.384$). Although freons themselves are not classified as direct carcinogens and exhibit relatively low toxicity, this association may represent an indirect effect mediated through their well-documented role in ozone layer depletion¹⁸. Increased ultraviolet (UV) radiation due to ozone thinning may elevate skin cancer risks, especially among children, whose developing tissues are more vulnerable to environmental insults¹⁹. These findings, while somewhat unexpected, warrant attention as a potential ecological signal linking anthropogenic emissions and radiation-mediated carcinogenesis.

Among the environmental indicators significantly associated with cancer outcomes at the $p < 0.10$ level, ammonia emissions appeared most frequently – showing correlations with overall cancer incidence, male cancer incidence, and the registered patient population living with cancer. This repetition suggests

¹⁴ Shala N.K., Stenehjem J.S., Babigumira R., Liu F.-C., Berge L., Silverman D.T., Friesen M.C., Rothman N., Lan Q., Hosgood H.D., Samuelsen S.O., Bråtveit M., Kirkeleit J., Andreassen B.K., Veierød M.B., Grimsrud T.K. (2023). Exposure to benzene and other hydrocarbons and risk of bladder cancer among male offshore petroleum workers. *Br J Cancer* 129:838–851.

¹⁵ Saeedi M., Malekmohammadi B., Tajalli S. (2024). Interaction of benzene, toluene, ethylbenzene, and xylene with human's body: Insights into characteristics, sources and health risks. *J Hazard Mater Adv* 16:100459.

¹⁶ Chemical Agents and Related Occupations. (2012). IARC Monographs on the Evaluation of Carcinogenic Risks to Humans, Volume 100F. International Agency for Research on Cancer. 624 p.

¹⁷ Xiong Y., Du K., Huang Y. (2024). One-third of global population at cancer risk due to elevated volatile organic compounds levels. *NPJ Clim Atmos Sci* 7:54.

¹⁸ Freon: Refrigeration characteristics and environmental impact (2024). URL: <https://www.thermal-engineering.org/freon-refrigeration-characteristics-environmental-impact>

¹⁹ Madronich S., McKenzie R.L., Björn L.O., Caldwell M.M. (1998). Changes in biologically active ultraviolet radiation reaching the Earth's surface. *J Photochem Photobiol B* 46(1-3):5-19.

a potential systemic role for ammonia as a marker of cumulative environmental burden in regions with intensified agricultural or industrial activity. Its consistent appearance across different population subgroups strengthens the hypothesis²⁰ that ammonia functions not only as a pollutant but also as a proxy for broader complex exposures in vulnerable areas.

Further evidence of cancer sensitivity to atmospheric pollution is provided by the statistically significant correlation between bone and joint neoplasms and per capita emissions of air pollutants ($r = 0.366$). This association is particularly notable given the relative rarity of bone cancers. Since per capita emissions reflect the intensity of environmental burden relative to population size, the observed correlation may suggest the importance of cumulative exposure – possibly to heavy metals or radioactive isotopes – among populations residing long-term in environmentally stressed regions.

While some environmental indicators have already demonstrated statistically significant correlations with certain cancer types at the 90% confidence level ($p < 0.10$), further analysis reveals that many of these same pollutants also show stronger associations – now at the 95% confidence level ($p < 0.05$) – but with different cancer indicators. This recurrence of environmental variables across confidence levels reinforces their potential importance and suggests broader or more complex relationships between specific pollutants and cancer outcomes. Therefore, the following section revisits several key environmental factors, not to repeat prior findings, but to highlight their statistically stronger and newly emerging associations with distinct oncological indicators.

A statistically significant correlation was observed between childhood lymphoma incidence and atmospheric emissions of bromine compounds ($r = 0.468$), which are recognized environmental toxicants with potential mutagenic and neurotoxic properties. This association may be linked to the widespread use of brominated organic compounds – particularly flame retardants – in the production of textiles, electronics, and plastics. Under certain conditions, these substances can be released into the ambient environment, where they exhibit persistence and a high potential for bioaccumulation. Brominated compounds, including polybrominated diphenyl ethers (PBDEs), are well-documented for their endocrine-disrupting activity and immunomodulatory effects, both of which are relevant to lymphoid tissue homeostasis. Evidence suggests that these compounds may contribute to lymphoproliferative processes and genomic instability.

²⁰ Chen G., Wang C., Huang S., Yang S., Su Q., Wang Y., Dai W. (2025). Novel roles of ammonia in physiology and cancer. *J Mol Cell Biol.* mjad007.

For example, Fonnum et al.²¹, Dingemans et al.²², and Huang et al.²³ have reported mutagenic and immunotoxic effects of various PBDE congeners. Furthermore, Helleday et al.²⁴ demonstrated that 3,4-dibromodiphenyl ether (DBDE) induces intragenic recombination in V79 Chinese hamster cells, indicating a direct genotoxic mechanism. In a related study, Eriksson et al.²⁵ investigated the effects of 2,2',4,4',5-pentabromodiphenyl ether in neonatal mice and found persistent neurobehavioral impairments, highlighting the heightened sensitivity of the developing nervous system to brominated exposures. Given the biological vulnerability of children and the prolonged latency period characteristic of many cancers, these findings collectively support the hypothesis that bromine-based compounds may act as environmental triggers in pediatric oncopathology. The presence of a statistically significant correlation in the current dataset reinforces the need for further toxicological and epidemiological studies focused on brominated emissions and their impact on childhood cancer risk.

Although this association was detected in a single correlation within the sample, its strength and toxicological plausibility highlight the need for continued monitoring of brominated emissions, particularly in regions where elevated childhood cancer incidence is observed. A similarly noteworthy finding was the correlation between hazardous waste generation (classes I–III) and the prevalence of skin cancer ($r = 0.415$). This relationship may reflect the role of domestic or occupational chemical exposure, inadequate industrial waste management, or direct contact with toxic substances through contaminated air, water, or surfaces in environmentally burdened area.

Equally noteworthy are the associations observed between emissions of methane, sulfur dioxide, and the total volume of air pollutants, and the recorded prevalence of malignant neoplasms of the uterus ($r = 0.625, 0.428, \text{ and } 0.446$, respectively). The relatively high strength of these correlations supports the hypothesis that air pollutants may contribute to the development of hormone-sensitive reproductive cancers. Potential mechanisms include disruption of

²¹ Fonnum F., Mariussen E. (2009). Mechanisms involved in the neurotoxic effects of environmental toxicants such as polychlorinated biphenyls and brominated flame retardants. *J Neurochem* 111:1327–1347

²² Dingemans M.M.L., van den Berg M., Westerink R.H.S. (2011). Neurotoxicity of brominated flame retardants: (In)direct effects of parent and hydroxylated polybrominated diphenyl ethers on the (developing) nervous system. *Environ Health Perspect* 119(7):900–907. <https://doi.org/10.1289/ehp.1003035>

²³ Huang H., Liu C., Liang C., Fang J. (2022). Comparative effects of brominated flame retardants BDE-209, TBBPA, and HBCD on neurotoxicity in mice. *Chem Res Toxicol* 35(10):1882–1893

²⁴ Helleday T., Lundin C., Nilsson R. (1999). Brominated flame retardant induces intrachromosomal recombination in mammalian cells. *Mutat Res Genet Toxicol Environ Mutagen* 439(1):137–147

²⁵ Eriksson P., Viberg H., Jakobsson E., Örn U., Fredriksson A. (2002). A brominated flame retardant, 2,2',4,4',5-pentabromodiphenyl ether: Uptake, retention, and induction of neurobehavioral alterations in mice during a critical phase of neonatal brain development. *Toxicol Sci* 67(1):98–103.

endocrine homeostasis or indirect effects on hepatic metabolism, which plays a central role in sex hormone regulation.

Methane deserves particular attention in this analysis. Although it is not traditionally classified as a direct carcinogen, methane is widely recognized as a marker of organic pollution, typically associated with landfills, agro-industrial facilities, and thermal power plants. The high frequency of statistically significant associations involving methane emissions suggests the need for further investigation into its potential role in carcinogenesis.

Methane demonstrated the strongest observed correlation in our dataset: a remarkably high coefficient was found between methane emissions and the prevalence of uterine cancer ($r = 0.625$), as well as strong associations with the total number of cancer patients in urban populations ($r = 0.598$), the overall cancer registry ($r = 0.582$), and specific cancer types including breast cancer ($r = 0.527$), skin cancer ($r = 0.509$), and non-Hodgkin's lymphomas ($r = 0.427$). This pattern strengthens the hypothesis that methane – or more likely, co-occurring volatile compounds – may contribute to the development of hormone-sensitive malignancies.

The recurrence of these associations suggests a systemic effect of methane-related emissions across multiple cancer types, possibly linked to complex toxic interactions or by-products of combustion processes. Although methane itself is relatively inert, its emissions often coincide with broader industrial pollutant mixtures, including PAHs, nitrogen and sulfur oxides, and various volatile organic compounds. Atmospheric photochemical reactions involving these substances can lead to the formation of secondary pollutants such as peroxyacyl nitrates (PANs), which have been shown to exert mutagenic and immunosuppressive effects^{26, 27, 28, 29}.

Taken together, the strength and consistency of methane-related correlations, particularly with hormonally mediated cancers, raise the possibility of an indirect hormonal or immunological pathway of action. These findings support the need to reconsider methane not solely as a climate-relevant gas, but also as a proxy for evaluating broader environmental health risks in complex exposure settings.

²⁶ Heddle J.A., Shepson P.B., Gingerich J.D., So K.W. (1993). Mutagenicity of peroxyacetyl nitrate (PAN) in vivo: Tests for somatic mutations and chromosomal aberrations. *Environ Mol Mutagen* 21:58–66.

²⁷ Kligerman A.D., Mottus K., Erexson G.L. (1995). Cytogenetic analyses of the in vitro and in vivo responses of murine cells to peroxyacetyl nitrate (PAN). *Mutat Res Genet Toxicol* 341(3):199–206.

²⁸ Vyskocil A., Viau C., Lamy S. (1998). Peroxyacetyl nitrate: review of toxicity. *Hum Exp Toxicol* 17(4):212–220.

²⁹ Thomas G.B., Fenters J.D., Ehrlich R., Gardner D.E. (1981). Effects of exposure to peroxyacetyl nitrate on susceptibility to acute and chronic bacterial infection. *J Toxicol Environ Health* 8(4):559–574.

Similarly, sulfur dioxide (SO₂) was found to be associated with multiple cancer-related indicators, including the number of women diagnosed with malignant neoplasms of the breast (r = 0.479) and uterus (r = 0.428), cancer incidence among urban populations (r = 0.413), and the overall number of cancer patients (r = 0.466). This pollutant, primarily emitted by thermal power plants, chemical industries, and metallurgy, is a byproduct of coal combustion and other industrial processes. While SO₂ is well documented as a contributor to respiratory disease^{30, 31, 32, 33}, our findings suggest a broader role in hormone-sensitive cancers, potentially via indirect pathways such as endocrine disruption.

Although SO₂ itself is not classified as a human carcinogen, it may increase the toxicity of other airborne pollutants through mucosal irritation and impairment of epithelial barrier function. In particular, its presence can amplify the health impact of co-emitted PAHs and industrial solvents, which may act as vehicles for toxic compounds, facilitate their penetration, or exert independent cytotoxic effects. These interactions create a permissive environment for carcinogenic agents to reach sensitive tissues and initiate oncogenic processes. Taken together, the associations observed in this study support the hypothesis that SO₂, beyond its known respiratory effects, may play a compounding role in environmental carcinogenic exposure, especially in urban-industrial settings.

Emissions of NMVOCs showed consistent and noteworthy associations with both Hodgkin's lymphoma (r = 0.452) and overall cancer-related mortality (r = 0.427). NMVOCs comprise a diverse group of chemical substances commonly found in petrochemical products, solvents, paints, and industrial coatings. Many of these compounds can undergo photochemical transformations in the presence of UV radiation, producing secondary pollutants such as formaldehyde and acetaldehyde – both of which are known to have carcinogenic properties.

Several NMVOCs possess documented cytotoxic and genotoxic effects, and even low-level exposures have been shown to interfere with cellular regulatory

³⁰ Sulfur Dioxide. (2023). American Lung Association. URL: <https://www.lung.org/clean-air/outdoors/what-makes-air-unhealthy/sulfur-dioxide>

³¹ Coal explained/ (2024). U.S. Energy Information Administration. Available from: <https://www.eia.gov/energyexplained/coal/>

³² Tomić-Spirić V., Kovačević G., Marinković J., Janković J., Ćirković A., Đerić A.M., Relić N., Janković S. (2021). Sulfur dioxide and exacerbation of allergic respiratory diseases: A time-stratified case-crossover study. *J Res Med Sci* 26:109.

³³ Cao D., Zheng D., Qian Z., Shen H., Liu Y., Liu Q., Sun J., Zhang S., Jiao G., Yang X., Vaughn M.G., Wang C., Zhang X., Zhang H. (2022). Ambient sulfur dioxide and hospital expenditures and length of hospital stay for respiratory diseases: A multicity study in China. *Ecotoxicol Environ Saf* 229:113082.

mechanisms, potentially inducing mutations and altering the cell cycle^{34, 35, 36}. Given the inclusion of various PAHs among NMVOCs – many of which are classified as probable or known carcinogens – the observed correlation with hematologic malignancies is biologically plausible and consistent with prior toxicological evidence.

PM also emerges as a significant factor due to its consistent positive associations with multiple cancer indicators. Notably, PM emissions were correlated with overall cancer incidence ($r = 0.508$), urban cancer prevalence ($r = 0.534$), and the incidence of Hodgkin's lymphoma ($r = 0.430$). Given that PM can act as a carrier for carcinogenic substances such as arsenic, cadmium, lead, and benzo[a]pyrene^{37, 38, 39}, its contribution to cancer risk is biologically plausible. Inhalation of PM has been shown to induce chronic airway inflammation, oxidative stress, and DNA damage – mechanisms central to environmentally induced carcinogenesis, especially under conditions of long-term, low-dose exposure.

The correlation observed with total pollutant emissions – used here as a generalized indicator of air quality – further supports the multifactorial nature of carcinogenesis. Elevated correlation coefficients with cumulative air pollution levels were found for several cancer types, including breast cancer ($r = 0.457$), skin cancer ($r = 0.423$), uterine cancer ($r = 0.446$), and brain tumors ($r = 0.420$). These findings point to the complex composition of the carcinogenic burden, particularly in industrialized regions, and suggest a systemic influence of mixed pollutants. Such exposures may include oxides, heavy metals, and volatile organic compounds, which can enter the human body through inhalation, dermal absorption, or the food chain and accumulate in specific target tissues.

Additional environmental indicators, such as the per capita generation of hazardous waste (classes I–III), also demonstrated noteworthy correlations.

³⁴ Kreja L., Seidel H-J. (2002). Evaluation of the genotoxic potential of some microbial volatile organic compounds (MVOC) with the comet assay, the micronucleus assay and the HPRT gene mutation assay. *Mutat Res Genet Toxicol Environ Mutagen* 513(1–2):143–150.

³⁵ Kreja L., Seidel H-J. (2002). On the cytotoxicity of some microbial volatile organic compounds as studied in the human lung cell line. *Chemosphere* 49(1):105–110.

³⁶ Seo Y., Kim S. (2024). Volatile organic compounds and cancer risk assessment in an intensive care unit. *Int J Biometeorol* 68:1731–1739.

³⁷ Sakunkoo P., Thonglua T., Sangkham S., Jirapornkul C., Limmongkon Y., Rayubkul J., Thongtip S., Maneenin N., Daduang S., Tessiri T., Pimonsree S. (2022). Human health risk assessment of PM_{2.5}-bound heavy metal of anthropogenic sources in the Khon Kaen Province of Northeast Thailand. *Heliyon* 8(6):e09572.

³⁸ Sui X., Cao Y., Wang W., Xie Z., Wang Y., Liu H. (2022). Pollution characteristics and health risk assessment of PM_{2.5}-bound arsenic: A 7-year observation in the urban area of Jinan, China. *Environ Geochem Health* 44:2679–2692.

³⁹ Fang D., Li X., Meng L., Li X., Zhang M., Yu Y. (2023). Pollution characteristics and human health risks of PM_{2.5}-bound heavy metals: A 3-year observation in Suzhou, China. *Toxics* 11(4):278.

For instance, the observed association with skin cancer prevalence ($r = 0.415$) may reflect localized environmental contamination, chronic dermal exposure to carcinogens, or indirect exposure through contaminated soil and water entering the food chain. These mechanisms are particularly relevant in the context of dermatological oncology, which encompasses both external (e.g., chemical contact) and internal (e.g., metabolic activation) risk pathways.

Collectively, these findings reinforce the hypothesis that cancer incidence patterns in Ukraine are shaped by a complex interplay of environmental exposures. They also underscore the value of using integrated pollution indicators and waste generation metrics as proxies for assessing environmental health risks in spatial epidemiological research.

Overall, the results of the analysis indicate a significant contribution of a number of atmospheric pollutants, especially methane, PM, sulfur dioxide, and volatile organic compounds, to the spread of various forms of cancer in Ukraine. The heat maps not only made it easy to visualize these connections, but also to identify substances associated with several types of neoplasms, which could subsequently form the basis for prioritizing preventive and regulatory measures.

CONCLUSIONS

Cancer remains a major public health challenge in Ukraine, ranking second among causes of mortality and accounting for nearly 10% of all deaths in 2022. Although official statistics suggest a decline in incidence, the true burden is likely underestimated due to territorial losses, disrupted access to healthcare, and incomplete reporting from conflict-affected regions. The analysis revealed persistent regional inequalities shaped by environmental exposure, healthcare accessibility, and demographic structure. Industrial–agricultural regions such as Dnipropetrovsk, Poltava, and Cherkasy exhibited elevated incidence and prevalence, whereas rural and mountainous regions showed lower reported rates, likely influenced by diagnostic gaps rather than genuinely reduced risk. Pediatric cancer incidence appears particularly vulnerable to underreporting in areas affected by conflict.

Correlation analysis demonstrated statistically significant associations between cancer indicators and environmental pollutants, including methane, sulfur dioxide, particulate matter, and hazardous waste generation. Using regional-level data from 26 administrative units, the study examined 1,250 pairwise correlations between 50 cancer indicators and 25 environmental variables; 69 showed moderate to strong associations, and 23 were statistically significant at the 95% confidence level. Methane emerged as the most recurrently associated pollutant, correlating with overall cancer prevalence,

urban incidence, and multiple hormonally mediated or environmentally sensitive cancers. Sulfur dioxide, particulate matter, ammonia emissions, and ⁹⁰Sr-contaminated areas were also linked to increased incidence of specific cancers. Geospatial and heatmap visualisation highlighted recurrent pollutant–cancer patterns, underscoring the multifactorial and regionally mediated nature of environmental carcinogenesis in Ukraine.

SUMMARY

This study provides quantitative evidence that environmental pollution contributes to regional variability in cancer incidence across Ukraine. Although the strength of individual associations is mostly moderate, their statistical reliability, recurrence across multiple cancer types, and biological plausibility point to meaningful environmental influences on cancer burden. Methane, sulfur dioxide, particulate matter, NMVOCs, and hazardous waste generation appear to serve as sentinel indicators of complex environmental exposures and merit priority attention in environmental health policy. These findings should be interpreted as early-warning signals rather than direct causal proof, yet they underline the need for strengthened environmental monitoring, improved diagnostic capacity, and the integration of spatial epidemiological tools into cancer prevention strategies. Targeted interventions in high-burden regions, particularly those characterised by intensive industrial or agricultural activity, will be essential for mitigating environmentally linked cancer risks and reducing geographic disparities in disease burden across Ukraine.

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