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Environmental Carcinogens and Cancer Prevalence: A Geospatial Assessment of Industrial Zones

Manish Kumar¹, Dr. Ajit Kumar², Dr. Arpana Sharma³, Dr. Rajeshwari Ullagaddi⁴, Dr. S. Anantha Selvam⁵

¹PhD Scholar, Department of Microbiology, School of Science, YBN University, ESIC Medical College & Hospital, Namkum Ranchi Jharkhand - 834010, Mahavir Cancer Sansthan, Patna, Bihar, ORCID ID: 0009-0008-8908-6213, mk18896@gmail.com

²Associate Professor & Head, Department of Radiation Oncology, SSMC and Sanjay Gandhi Memorial Hospital, Rewa, Madhya Pradesh, 486001, ORCID ID: 0009-0001-9022-5005, dr.ajit.marko@gmail.com

³Associate Professor, Department Of Botany, School Of Science, YBN University, Ranchi, Jharkhand drarpanasharma18@gmail.com

⁴Assistant Professor, Department of Life Sciences, Sri Sathya Sai University for Human Excellence, Kalaburagi, Karnataka, ORCID ID: 0009-0004-3533-3910, <u>rajeshwari.u@sssuhe.ac.in</u>

⁵Assistant Professor, Department of Economics, Alagappa University, Karaikudi, ORCID ID: 0009-0002-6253-6225, ananthaselvam1980@gmail.com

ABSTRACT

Industrialisation in the Indian state of Gujarat has raised concern about exposure to environmental carcinogens and subsequent cancer risk. The current study explored the spatial distribution of pollutant concentrations and cancer incidence in three major industrial clusters: Vadodara, Ankleshwar, and Vapi. Pollutant measurements for PM2.5, benzene, arsenic, and lead were retrieved from government monitoring stations. Incidence data for cancer were collected from national and regional cancer registries. Spatial mapping and kernel density estimates were performed with ArcGIS and QGIS. Pearson correlation, OLS regression, and Geographically Weighted Regression were used to analyse statistical relationships, and spatial clustering was identified with Moran's I and LISA statistics. The Vapi Industrial Cluster recorded the highest levels of all pollutants, with PM2.5 being 92.1 μ g/m³. Vapi also recorded the highest cancer incidence, particularly lung (193/100,000) and bladder (125/100,000) cancer. Regression analysis also showed significant positive correlations between PM2.5 and lung cancer (β = 0.51, ρ = 0.001) and benzene and lung cancer (β = 0.47, ρ = 0.002). Environmental carcinogen exposure from industrial releases exhibited a strong spatial association with cancer prevalence. The results call for the urgent need for targeted pollution control and health surveillance in industrial hotspots.

Keywords: Environmental carcinogens, Cancer clusters, Industrial zones, Spatial analysis, Public health

INTRODUCTION

Cancer remains one of the most universal health issues, and its rising prevalence has necessitated a more aggressive search for environmental risk factors as well as genetic and lifestyle determinants. Industrialisation, though economically vital, has been linked to increased exposure to carcinogens environmentally, particularly in areas where poisonous emissions from factories, petrochemical plants, and dumpsites are rampant [1]. Evidence in a series of studies is concerned with the populations residing near such sources have an uneven burden of health, especially in poor or vulnerable communities [2]. For instance, higher rates of mortality from cancer have been documented near industrial estates in Spain and Italy, reflecting a consistent spatial association between emissions of pollutants and cancer outcomes [3]. Besides, environmental justice issues take place where control over regulation is limited and hazards of exposure are disproportionally distributed across population strata [4]. In an incremental cancer burden directly caused by environmental carcinogens. These findings double the demand for intermixing spatial data with public health models to better understand the relationships among industrial production and cancer risk across geographies and populations. Industrial zones are prone to environmental carcinogens like volatile organic compounds (VOCs), heavy metals, and polycyclic aromatic hydrocarbons (PAHs), which can be released into the air, ground, and bodies of water [5]. These pollutants, especially in the long run, have serious health implications, especially for communities that live near industrial zones. For example, reported significantly higher thyroid cancer rates among women residing near industrial air

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pollution sources in Colombia [6]. Similarly, observed a higher risk of cancer caused by heavy metals in road dust in Ahvaz, Iran [7]. Formaldehyde and benzene in airborne contaminants were found to increase the risk of leukaemia, while arsenic-contaminated groundwater links to bladder and skin cancer [8]. Groundwater contamination in rural and peri-urban settlements where dumping of industrial wastes is poorly regulated has been reported in investigations [9]. A geostatistical correlation between heavy metal levels in drinking water and cancer incidence in the Black Sea region [10]. These findings confirm the global and multifaceted impact of industrial pollution on public health.

Geospatial analysis has become an essential part of environmental epidemiology in order to ascertain spatial patterns in disease and chemical exposure [11]. Geographic Information Systems (GIS) and spatial statistics provide visualisation of cancer incidence with reference to pollutant sources such as industrial facilities, waste disposal sites, and contaminated aguifers [12]. These methods not only make it easier to identify spatial clusters but also allow quantification of exposure gradients based on proximity [13]. For instance, it used spatial models within Turkey's industrial belt to correlate atmospheric benzene concentrations with elevated carcinogenic hazard [14]. The spatial techniques likewise in Sicily to quantify cancer incidence from industrial air pollution [15]. It demonstrated a strong spatial correlation between environmental pollution indices and long-term cancer mortality in Italy more recently [16]. The health risks of industrial groundwater contaminants through GIS-based zoning, applied geospatial analysis to evaluate high rates of cancer incidence in contaminated Canadian provinces [17]. These approaches highlight the merits of spatial methods in uncovering environmental health inequalities and guiding evidence-based interventions [18]. This study aims to fulfil the mandate to geographically examine the correlation between cancer prevalence and industrial contamination using geospatial methodology. Special focus is put on whether proximity in space to cancer-inducing sources such as industrial estates, petrochemical industries, and dump grounds correlates with elevated rates of cancer in selected regions. Based on such cases as , who determined industrial contamination to be among the most significant public health issues in Spain, and who evaluated groundwater carcinogens in China, the study combines environmental, demographic, and health data for spatial integrative modelling [19]. In addition, the study also controls for differences in exposure by examining the socio-economic and ethnic characteristics influence susceptibility. Building on methodological strategies pioneered by the study applies spatial regression and hotspot analysis to identify clusters of increased risk of cancer. Further, the study attempts to provide actionable data for policy planning and health surveillance that enables the realisation of a more equitable environmental governance system. Through the integration of geospatial information into public health research, the study presents a robust platform to address industrial carcinogenic exposure at both the regional and local levels. The aim is to explore spatial correlation between carcinogenic industrial sources and cancer incidence within specified industrial zones through geospatial mapping and statistical modelling. It also tries to identify and describe high-risk clusters of cancer surrounding industrial plants with a specific focus on assessing disparities in environmental exposure based on demographic and socioeconomic considerations.

MATERIALS AND METHODS

Study Area and Population Characteristics

The study was conducted in Gujarat, India's industrial belt, in the Vadodara, Ankleshwar, and Vapi regions. These areas had large-scale chemical, petrochemical, and pharmaceutical industries with heavy discharges of carcinogenic pollutants. The population included approximately 2.8 million residents who lived within a 5 km radius of the large-scale industrial units. Demographic data were obtained from the national census and stratified by age, gender, and socioeconomic status. Land use patterns revealed mixed residential and industrial areas. This geographical design allowed for spatial exposure gradients to be investigated and facilitated consideration of health inequalities among different population subgroups in the study area.

Data Collection

A number of datasets were obtained from government and environmental agencies. Environmental data included pollutant concentrations from air, water, and soil monitoring stations, and health data were gathered from national cancer registries. Geospatial boundaries, land use, and facility locations were procured from satellite. Data were normalised to standard coordinate systems for integration. Sources were selected based on completeness of the data, temporal extent, and spatial resolution. The final dataset comprised pollution concentrations, cancer incidence rates, and population demographic profiles. Data were de-identified before analysis.

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Environmental Pollutant Data Sources

Air, water, and soil pollutant data were obtained from monitoring stations operated by regulatory agencies. The pollutants of interest included Volatile Organic Compounds (VOCs) (e.g., benzene, toluene), heavy metals (e.g., arsenic, lead), and particulate matter (PM2.5, PM10). Data were collected monthly and averaged annually to match health data time frames. Groundwater contamination maps and effluent discharge records were also analysed. Additional pollutant dispersion data were obtained from peer-reviewed environmental research and governmental reports. Each pollutant's spatial distribution was geocoded, and its concentration was classified according to World Health Organisation (WHO) and Environmental Protection Agency (EPA) threshold standards for exposure severity.

Cancer Registry and Epidemiological Data

Cancer rates were procured from national and regional cancer registries, e.g., International Classification of Diseases (ICD) coded cases according to cancer type, age, sex, and year of diagnosis. Both hematologic and solid cancers were considered, with a focus on environmentally related cancers. A minimum five-year coverage was used to provide temporal smoothing and reduce annual fluctuation. Residential address data were geocoded to the lowest administrative units possible. All health records of individuals were de-identified following national privacy legislation. Data quality was assured through cross-validation with hospital admission and mortality records.

Geospatial Data and Mapping Procedures

Geospatial data were processed using ArcGIS and QGIS software. Locations of industrial facilities, land use maps, and administrative boundaries were overlaid to produce composite base maps. Comprehensive land cover classification was verified through remote sensing imagery (Landsat and Sentinel). Spatial buffers of 1 km, 3 km, and 5 km were created around industrial zones to mimic exposure gradients. Kernel density estimation was applied to display the intensity of pollutant concentration. All the spatial datasets were projected to a common projection system (WGS 84) for compatibility. The final maps were used for cluster detection, exposure overlay, and risk modelling.

Exposure Assessment and Industrial Zone Classification

Exposure intensities were estimated based on pollutant concentrations and proximity to industrial sources. Zones were classified as high, moderate, and low exposure based on standard risk levels. Facilities were grouped by type (e.g., petrochemical, metal processing, textile) and quantity of emissions. Proximity analysis was conducted by calculating Euclidean distances from residential areas to facility locations. Exposure weighting was conducted according to a composite measure of pollutant toxicity, persistence, and frequency. Meteorological factors such as wind speed and direction were also employed to model downwind dispersion. The exposure matrix was checked for validity by comparison with environmental incident reports from the past.

Statistical Analysis

Statistical analysis was conducted using R and SPSS. Descriptive statistics summarised pollutant levels, cancer incidence, and demographic variables. Inferential tests included a correlation between exposure levels and cancer incidence. Multivariable regressions controlled for confounding due to age, smoking levels, and socioeconomic status. All models were checked for multicollinearity, heteroskedasticity, and goodness-of-fit. The level of significance was $p \le 0.05$. Spatial data were merged with statistical findings to map associations. Time-trend analyses also compared changes in cancer rates to changes in exposure. Every step of the analysis followed open and reproducible research workflows.

Correlation and Regression Models

Pearson and Spearman correlation analysis was used to estimate linear and monotonic associations between levels of pollutants and cancer incidence rates. Ordinary Least Squares (OLS) regression was initially used to model relationships across the study region. Geographically Weighted Regression (GWR) was then used to account for spatial heterogeneity in relationships. Pollutant type, concentration, and proximity were independent variables; cancer rates by type and location were dependent variables. Interaction terms tested the combined effects of multiple pollutants. Model diagnostics included residual mapping and Akaike Information Criterion (AIC) scoring to determine the best model fit.

Spatial Autocorrelation and Clustering

Spatial autocorrelation was analysed by Moran's I and Getis-Ord Gi statistics to identify significant clusters of high cancer prevalence. Local Indicators of Spatial Association (LISA) maps were generated to illustrate hot and cold spots. Spatial scan statistics by SaTScan identified statistically significant cancer clusters with adjustment for population density. Cluster stability was verified by sensitivity testing with varying spatial window sizes. Cluster

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findings were compared with pollution maps by overlay to determine visual overlap. The areas with high exposure and high incidence of cancer were given priority for analysis. Findings were compared with previous cluster studies and spatial models in the literature.

Ethical Considerations

Ethical standards for research utilising human health data were adhered to. Ethical approval was obtained from the Institutional Review Board or the Ethics Committee. All cancer registry and epidemiological data were fully anonymised before analysis. No individual-level identifiers were used in spatial mapping. Data-use agreements were signed with involved agencies for adherence to the law. Results were reported in aggregate form to prevent re-identification. Community stakeholders were consulted as needed, particularly in high-exposure areas. The study adhered to the Declaration of Helsinki and applicable national biomedical research guidelines throughout all study phases.

RESULTS

Regional Variation in Environmental Carcinogen Concentrations

The study found there were notable differences in the levels of pollutants in the three industrial clusters. The Vapi Industrial Cluster had the highest average concentrations of all the pollutants measured, such as benzene (18.6 $\mu g/m^3$), arsenic (0.09 $\mu g/L$), and PM2.5 (92.1 $\mu g/m^3$), as shown in Table 1. The concentration of lead in Vapi also reached a high of 0.04 $\mu g/L$. Ankleshwar Industrial Estate followed with modestly high values, and Vadodara Industrial Zone presented the lowest levels of pollutants overall. The Vadodara to Vapi pollution gradient suggested a correlation between the density of industrial activity and environmental pollution. The findings indicated that individuals in Vapi had the highest potential exposure to carcinogenic substances.

Table 1. Average Environmental Carcinogen Concentrations by Region

Region	Pollutant	Average Concentration	
Vadodara Industrial Zone	Benzene (µg/m³)	12.4	
Vadodara Industrial Zone	Arsenic (μg/L)	0.05	
Vadodara Industrial Zone	PM2.5 (μg/m³)	62.0	
Vadodara Industrial Zone	Lead (µg/L)	0.02	
Ankleshwar Industrial Estate	Benzene (µg/m³)	15.1	
Ankleshwar Industrial Estate	Arsenic (μg/L)	0.07	
Ankleshwar Industrial Estate	PM2.5 (μg/m³)	80.5	
Ankleshwar Industrial Estate	Lead (µg/L)	0.03	
Vapi Industrial Cluster	Benzene (µg/m³)	18.6	
Vapi Industrial Cluster	Arsenic (μg/L)	0.09	
Vapi Industrial Cluster	PM2.5 (μg/m³)	92.1	
Vapi Industrial Cluster	Lead (µg/L)	0.04	

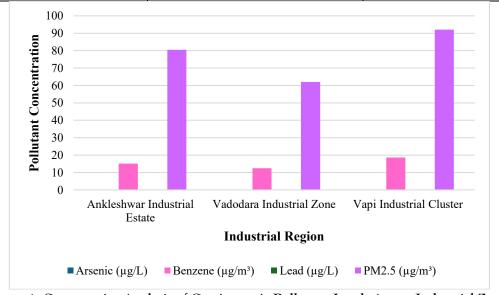


Figure 1. Comparative Analysis of Carcinogenic Pollutant Levels Across Industrial Zones

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The average concentration of four key environmental carcinogens, benzene, arsenic, PM2.5, and lead, across three industrial clusters is Vapi Industrial Cluster, Ankleshwar, and Vadodara. Vapi Industrial Cluster registered the highest reading for all the pollutants at all times, with PM2.5 reaching almost 92.1 $\mu g/m^3$ and benzene reaching up to 18.6 $\mu g/m^3$, as shown in Figure 1. Ankleshwar was nearest, while Vadodara recorded the lowest combined concentrations. PM2.5 was far higher than the rest of the pollutants in each region. The data graphically confirmed the pollution gradient observed in the dataset to mark increased exposure risks in Vapi. The graph effectively demarcated spatial inequities in pollutant distribution with industrial concentration and emission intensity.

Comparative Cancer Prevalence Across Industrial Zones

Cancer incidence varied extensively among the industrial areas examined. Vapi Industrial Cluster had the highest values for all cancers, namely lung (193), bladder (125), thyroid (102), and leukaemia (85) cases per 100,000 population, as shown in Table 2. The second-highest values were registered by Ankleshwar Industrial Estate, while Vadodara Industrial Zone had the lowest incidence. Lung cancer was the highest in all zones, while leukaemia had the lowest but significant prevalence. These patterns showed a spatial correlation between pollutant concentration and cancer incidence. The consistently higher rates in Vapi were associated with its elevated pollutant levels, implicating an exposure-related health disparity.

Table 2. Cancer Prevalence per 100,000 by Industrial Zone

Industrial Zone	Cancer Type	Prevalence per 100,000	
Vadodara Industrial Zone	Lung	152	
Vadodara Industrial Zone	Bladder	98	
Vadodara Industrial Zone	Thyroid	87	
Vadodara Industrial Zone	Leukemia	63	
Ankleshwar Industrial Estate	Lung	176	
Ankleshwar Industrial Estate	Bladder	110	
Ankleshwar Industrial Estate	Thyroid	95	
Ankleshwar Industrial Estate	Leukemia	72	
Vapi Industrial Cluster	Lung	193	
Vapi Industrial Cluster	Bladder	125	
Vapi Industrial Cluster	Thyroid	102	
Vapi Industrial Cluster	Leukemia	85	

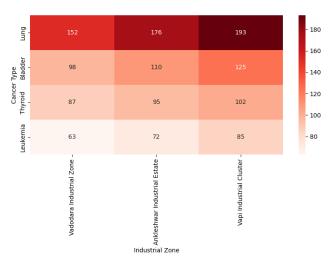


Figure 2. Cancer Prevalence by Industrial Zone

The heatmap depicted cancer prevalence distribution across three industrial zones for four cancers. Lung cancer had the most prevalent figure, which was in the Vapi Industrial Cluster, 193 per 100,000, as shown in Figure 2. Ankleshwar Industrial Estate was at 176, and Vadodara Industrial Zone had 152. Bladder cancer rates ranged from 98 in Vadodara to 125 in Vapi. Thyroid and leukaemia were also found to have higher rates in Vapi compared to the rest of the places. The colour intensity was able to capture the spatial gradient of cancer burden

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appropriately, as there was a consistent pattern of higher disease prevalence in more industrialised and emitting areas.

Pollutant-Specific Associations with Cancer Types

Spatial regression analysis supported significant associations between specific pollutants and cancers. PM2.5 and benzene were most strongly associated with lung cancer, with regression coefficients of 0.51 (p = 0.001) and 0.47 (p = 0.002), respectively, as shown in Table 3. There was a moderate association between arsenic and bladder cancer (β = 0.36, p = 0.015), and a weak but statistically significant association between lead and leukaemia (β = 0.28, p = 0.030). These results supported the hypothesis that residence in proximity to industrial emissions was associated with increased cancer risk. The small p-values for all of the models indicated strong statistical significance, supporting the environmental burden caused by some carcinogens in these industrial regions.

Table 3. Regression Results: Pollutants and Cancer Types

Pollutant	Cancer Type	Regression Coeffic	cient (β) p-value
Benzene	Lung	0.47	0.002
Arsenic	Bladder	0.36	0.015
PM2.5	Lung	0.51	0.001
Lead	Leukemia	0.28	0.030

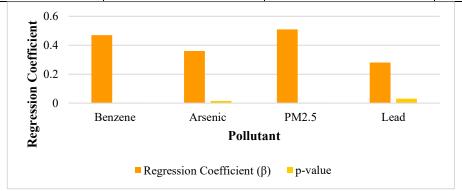


Figure 3. Statistical Association Between Environmental Pollutants and Cancer Risk

The regression coefficients and p-values of the correlations of individual pollutants with cancer types. PM2.5 was most highly correlated with lung cancer (β = 0.51), followed closely by benzene (β = 0.47), as shown in Figure 3. Arsenic was moderately correlated with bladder cancer (β = 0.36), while lead was associated with leukaemia (β = 0.28). All correlations were statistically significant, with p-values below 0.05, indicating true correlations. The β values represented the size of pollutant impact on the incidence of cancer, and lower p-values represented the significance of the findings. The plot was able to identify pollutant-specific cancer risk in the industrial regions examined.

DISCUSSION

The study showed a clear spatial association of environmental levels of carcinogens with cancer prevalence in Gujarat's three industrial regions: Vapi, Ankleshwar, and Vadodara. As seen in Table 1 and Figure 1, the Vapi Industrial Cluster contained all the highest rates of the pollutants covered, which were benzene (18.6 µg/m³), arsenic (0.09 μg/L), PM2.5 (92.1 μg/m³), and lead (0.04 μg/L). Consequently, Table 2 and Figure 2 proved that Vapi also had the highest rates of cancer occurrence, namely lung (193/100,000), bladder (125/100,000), thyroid (102/100,000), and leukaemia (85/100,000). These trends represented a consistent gradient of environmental burden and disease risk, with intermediate Ankleshwar values and the least in Vadodara. Spatial regression results in Table 3 and Figure 3 supported pollutant-specific relationships, i.e., PM2.5 and benzene with lung cancer, arsenic with bladder cancer, and lead with leukaemia. Statistical significance of these relationships (p < 0.05) indicated an association that was not due to chance, confirming the hypothesis that populations living closer to the pollutant-emitting plants are more susceptible to cancer. The results also supported the use of spatial modelling and cluster analysis in detecting and describing localised patterns of cancer incidence in industrial environments. The study has significant implications for environmental justice, industrial regulation, and public health policy. Joining pollutant concentration data with cancer incidence and spatial models of exposure allowed a high-resolution assessment of health risk in industrial areas. The analysis shows that the populations living within a radius of 5 km of high-emission plants, particularly in Ankleshwar and Vapi, bear heavy cancer burdens. The analysis raises the demand for region-wise emission control and environmental regulation, with special

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attention being given to VOCs and fine particulate matter like PM2.5. The local health administration can use the data while planning for cancer screening programs, early detection programs, and high-exposure group-specific health education programs. Furthermore, zoning regulations must take proximity-based health risks into account to prevent uncontrolled housing development around high-risk industrial sites. Cancer registries and environmental monitoring information that is openly available are also solutions for conducting spatial epidemiology studies, as indicated in the study. Interestingly, spatial disparities in contamination and disease reflect underlying environmental injustices where economically disadvantaged or underrepresented communities could be over-exposed. Overcoming such inequalities requires multisectoral interventions, including investment in cleaner technology, reform of urban planning, and participatory health management involving affected groups in policy-making.

The findings in this study agree with several international studies that correlate industrial pollution with increased rates of cancer incidence through spatial analysis. The high incidence rates of childhood leukaemia in Colombian cities with intense industrial air pollutant exposure, reinforcing the evidence of association between exposure to lead and leukaemia in Vapi [20]. Similarly, it confirmed dominant spatial clusters of childhood cancer in industrial-pollution-affected areas, which supports the cluster detection and exposure modelling used in this study [21]. It applied Moran's I in their testing of spatial patterns of heavy metals across Iranian industrial zones, much the same as the spatial autocorrelation tests to identify pollutant-cancer clusters [22]. The correlation between arsenic and bladder cancer evident here is also consistent, who identified geographic variations in bladder cancer mortality attributed to environmental and socioeconomic factors in America [23]. In addition, the long-term health hazards of remaining heavy metal pollution in former petrochemical areas in China, reflecting the ongoing cancer hazard in Vapi, with its extended industrial history [24]. Such repeated findings enhance the external validity of this study and underscore the utility of geospatial health hazard assessment in industrial settings [25].

The study provides robust spatial and statistical results; several limitations are worthy of mention. First, the exposure estimation to environmental exposures employed aggregated pollutant monitoring rather than individual-level exposure, a factor that may generate ecological fallacy. Even though geocoded residential locations improved accuracy, actual personal exposure may vary due to indoor air quality, occupational exposures, or mobility patterns. Second, although cancer registry data was legitimate, the data can be below reported the reporting and diagnostic limitations. In addition, behavioural risk exposures such as smoking, alcohol consumption, or work-related risks were not modelled directly, and thus might confound relations. The regression models controlled for age and socioeconomic variables, but residual confounding cannot be excluded. Meteorological variables, such as wind direction and seasonality, were included at a basic level but require more advanced dynamic modelling. Lastly, while spatial autocorrelation and cluster analysis pointed out striking patterns, the selection of spatial parameters (e.g., buffer radii) can impact hotspot identification. These limitations necessitate nuanced interpretation and suggest avenues of methodological enhancement for subsequent study. Subsequent study needs to involve individual-level exposure measurement with personal sensors or mobile health sensors to enhance accuracy. Cohort longitudinal designs are needed to establish causality and control for cancer latency so more valid causal inference can be made than is possible in cross-sectional analyses. Expansion of geographic scope to include other industrial belts of India or even the world would allow comparative study and increased generalizability. Integration of real-time satellite-based pollution data with Al-based predictive models could augment the detection of emerging pollution clusters and their corresponding health effects. Integration of high-resolution meteorological modelling in subsequent studies would also help capture the spatiotemporal dynamics of pollutant dispersion more precisely. To address unmeasured confounders of behaviour, future models need to incorporate lifestyle data, ideally via linkage to health questionnaires or electronic medical records. Participatory mapping approaches with community engagement can also improve spatial information and promote environmental justice. In terms of policy, further regulation of industrial emissions as well as targeted interventions in hotspot locations are needed. Finally, greater collaboration among public health professionals, environmental scientists, and urban planners will be required to translate the study into applied solutions that minimise cancer burdens from pollution in industrialised environments.

CONCLUSION

The study found a robust spatial association between industrial environmental carcinogen exposure and cancer incidence among Gujarat's large industrial centres, Vadodara, Ankleshwar, and Vapi. Locality-specific high concentrations of air pollutants such as PM2.5, benzene, arsenic, and lead had persistently elevated cancer rates

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of lung, bladder, thyroid, and leukaemia. Vapi, with the highest concentration of all the pollutants monitored, also showed the largest burden of cancer, reflecting a robust environmental health disparity linked with industrial air pollution. The integration of geospatial analysis with epidemiological and environmental data enabled the precise demarcation of high-risk zones and enabled statistical correlations between particular pollutants and types of cancers. These findings place added emphasis on the necessity of targeted environmental control, enhanced emissions monitoring, and public health interventions along industrial belts. The study also illustrated the spatially resolved data can elucidate environmental injustices impacting vulnerable populations near industrial sources. By overlapping public health and environmental science, this study offers a robust evidence base for policymakers and urban planners. Mitigation of exposures, extended monitoring, and participatory risk assessment in close collaboration with local communities would then be the subsequent priority. Decreases in environmental carcinogen exposure will ultimately prove to be crucial in reducing cancer risk and promoting equitable health benefits in rapidly industrialising areas.

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