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ENVIRONMENTAL CARCINOGENESIS IN HARYANA, INDIA: A CRITICAL COMPREHENSIVE REVIEW OF CANCER SURVEILLANCE, MULTIEXPOSURE PATHWAYS, AND PREVENTION PRIORITIES

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Abstract- **Haryana combines rapid industrialisation, intensive agriculture, severe air pollution, groundwater stress, and uneven occupational and environmental regulation, creating a plausible setting for cumulative carcinogenic exposure. This critical comprehensive review integrates cancer surveillance, Haryana-specific environmental studies, institutional monitoring, mechanistic evidence, and international epidemiology while distinguishing hazard detection from demonstrated local causation. ICMR-NCRP estimates indicate that annual incident cancer cases increased from 29,219 in 2020 to 32,513 in 2024. The peer-reviewed Haryana Cancer Atlas analysis included 36,736 confirmed cancers diagnosed in 2016-2017 from 126 participating centres, supplemented by eligible records from 28 hospital-based cancer registries, and identified substantial district and site-specific variation. The broader administrative atlas reported 42,699 records from 149 sources, illustrating why analytic and administrative totals should not be conflated. Rohtak had the highest male all-site age-adjusted incidence rate (143.9 per 100,000), whereas Gurugram had the highest female rate (108.2); high head-and-neck cancer rates among men and district variation in breast, lung, prostate, and cervical cancer require targeted surveillance but do not establish environmental causation. Groundwater monitoring identified nitrate, fluoride, arsenic, and uranium exceedances, while pesticide residues, industrial mixtures, ambient particulate matter, combustion emissions, and workplace exposures create additional prevention priorities. The strongest causal evidence relevant to Haryana concerns outdoor air pollution, benzene, inorganic arsenic, hexavalent chromium, cadmium, and selected occupational exposures; pesticide evidence remains compound- and outcome-specific, and uranium requires cautious interpretation because its drinking-water guideline primarily reflects chemical nephrotoxicity. The review proposes a transparent evidence hierarchy, an eco-oncology framework, district-cluster research priorities, and an implementation roadmap centred on sustained population-based cancer registration, historical exposure reconstruction, safe water, clean air, pesticide stewardship, and worker protection. Haryana offers a transferable model for environmental-cancer surveillance in rapidly industrialising, agriculturally intensive, data-constrained regions.**

Keywords- **air pollution; cancer surveillance; environmental carcinogens; groundwater; Haryana; occupational exposure; pesticides; uranium**

I. INTRODUCTION

Haryana is an important setting in which to examine environmental determinants of cancer because several development trajectories intersect within a relatively small and densely connected territory. The state includes highly urbanized districts within the National Capital Region, large industrial corridors, intensive irrigated agriculture, groundwater-dependent rural communities, thermal and transport emission sources, and rapidly expanding peri-urban settlements. These conditions create multiple opportunities for inhalation, ingestion, dermal, and occupational exposure to carcinogenic or potentially carcinogenic agents and reflect the broader contribution of pollution to preventable disease [1]. They also create substantial heterogeneity: the exposure profile of Gurugram or Faridabad is not equivalent to that of Hisar, Sirsa, or Mahendragarh, and a single state-level narrative can

conceal important differences in source, route, duration, and population vulnerability.

Cancer burden is also increasing. ICMR-NCRP model-based estimates reported by the Government of India rose from 29,219 incident cases in 2020 to 32,513 in 2024, an increase of approximately 11.3% over four years [2], [3]. These figures are planning estimates rather than direct enumeration of every new diagnosis. Globally and nationally, cancer burden is shaped by population growth, ageing, tobacco and alcohol use, infection, reproductive and metabolic factors, occupational conditions, and environmental exposures; accordingly, trends cannot be assigned to pollution without site-specific and confounder-adjusted analysis [4], [5], [6], [7].

The Development of an Atlas of Cancer in Haryana State was a major surveillance advance. The administrative report compiled 42,699 registrations from 149 sources during 2016-2017 [8]. The peer-reviewed analytic publication reported 36,736 confirmed cancers after data-quality procedures, using 126 consenting centres and eligible records from 28 hospital-based cancer registries across Haryana and neighbouring states [9]. It calculated district- and sex-specific age-adjusted incidence rates using the world standard population, applied de-duplication and coding checks, and explicitly concluded that Haryana required stable population-based cancer registration. The different totals reflect distinct stages and analytic datasets and should not be presented as interchangeable. This work builds on India's earlier cancer-atlas methodology [10].

The central challenge is therefore not whether environmental hazards exist-many are well documented-but how confidently they can be linked to cancer in a specific population. Hazard identification, exposure assessment, and causal attribution are distinct steps. A contaminant above a guideline value warrants risk management, yet does not by itself demonstrate that the contaminant caused a district's cancer pattern. Conversely, lack of local epidemiological proof should not be used to delay control of agents already established as human carcinogens. A high-quality review must preserve both principles.

This article provides a critical comprehensive synthesis of evidence relevant to environmental carcinogenesis in Haryana. Its contribution is not another catalogue of contaminants. Instead, it integrates cancer-registration evidence, compound-specific carcinogenic classifications, local exposure measurements, occupational pathways, spatial vulnerability, and governance capacity within a calibrated hazard-exposure-causation framework. The objectives are to: (i) characterise the state's cancer-surveillance context; (ii) critically evaluate major environmental and occupational exposure domains; (iii) identify where Haryana-specific evidence is strong, suggestive, or absent; (iv) distinguish district surveillance signals from causal cancer clusters; and (v) translate the evidence into testable research priorities and feasible prevention actions. Haryana is treated as a case study for other rapidly industrialising and agriculturally intensive low- and middle-income settings where environmental data and cancer surveillance remain institutionally separated [1], [11].

II. REVIEW SCOPE AND CRITICAL EVIDENCE-APPRAISAL APPROACH

This article was designed as a critical comprehensive review, not a systematic review or meta-analysis. Consistent with methodological guidance for critical and integrative literature reviews, the purpose was conceptual integration, evidence calibration, and identification of decision-relevant gaps rather than exhaustive retrieval or pooled effect estimation [12], [13]. The literature base was updated through 10 July 2026.

Source identification combined targeted searches of PubMed/PMC, Google Scholar discovery, journal and DOI landing pages, and official institutional repositories. Government and technical sources included ICMR-NCDIR/NCRP, the Ministry of Health and Family Welfare, Parliamentary question annexures, the Central Ground Water Board, WHO, IARC, and relevant pollution-control and public-health documents. Search concepts combined Haryana or North India with cancer incidence, cancer atlas, population-based cancer registry, PM2.5, air pollution, pesticide, agricultural worker, uranium, nitrate, arsenic, chromium, groundwater, occupational carcinogen, waste burning, indoor air pollution, and environmental justice.

Evidence was prioritised in four tiers. Tier 1 comprised Haryana-specific cancer-registration or environmental measurements. Tier 2 comprised North Indian studies relevant to comparable exposure systems. Tier 3 comprised Indian national surveillance, burden, or regulatory evidence. Tier 4 comprised international epidemiological, toxicological, mechanistic, and IARC evidence used to interpret biological plausibility and causal strength. Peer-reviewed studies, authoritative monographs, and official monitoring reports were favoured over media summaries; media reports were not used as primary scientific evidence.

Each source was appraised for design, population, exposure measurement, outcome ascertainment, temporality, geographic relevance, confounder control, and interpretive limitations. Evidence was classified qualitatively as strong, moderate, limited, or insufficient. Strong evidence required established carcinogenicity and consistent epidemiological support; moderate evidence indicated plausible, reasonably consistent associations with important local data gaps; limited evidence indicated suggestive findings or weak exposure-outcome linkage; insufficient evidence indicated that local causal attribution could not be made.

No pooled effect estimate was calculated because the evidence spans incompatible designs, exposure metrics, time periods, and cancer outcomes. Five safeguards were applied: environmental exceedance was not treated as proof of cancer; ecological co-location was considered hypothesis-generating; historical exposure and cancer latency were emphasised; all-site cancer was not used as a substitute for biologically matched site-specific outcomes; and potential distortion from

tobacco, age, urbanisation, socioeconomic position, healthcare access, referral, residential mobility, and incomplete registration was explicitly considered. Table 1 documents the search and appraisal framework, and Table 2 summarises the Haryana-focused evidence base.

Table 1: Search, selection, and critical-appraisal framework for the comprehensive review

Component	Operational approach
Review purpose	Critical integration of cancer surveillance, environmental exposure, carcinogenic evidence, governance, and prevention; not an exhaustive systematic review.
Search coverage	PubMed/PMC; Google Scholar discovery; journal/DOI pages; ICMR-NCDIR/NCRP; Ministry of Health and Family Welfare; Parliamentary annexures; CGWB; WHO/IARC; relevant official repositories.
Temporal scope	Primarily 2010-2026, with older foundational sources retained for cancer registration, carcinogen classification, exposure science, and landmark epidemiology. Last update: 10 July 2026.
Core concepts	Haryana, cancer atlas, PBCR, PM2.5, pesticides, agricultural workers, groundwater, nitrate, arsenic, uranium, industrial metals, benzene, occupational carcinogens, waste burning, indoor air pollution, environmental justice.
Priority evidence	Haryana empirical data > comparable North Indian data > Indian national evidence > international causal and mechanistic evidence.
Appraisal domains	Study design, exposure measurement, outcome validity, temporality, latency, geographic relevance, confounding, bias, precision, and transferability.
Synthesis rule	Qualitative certainty categories (strong/moderate/limited/insufficient); no pooling because designs, metrics, periods, and outcomes were heterogeneous.
Causal safeguards	No equation of exceedance with disease; no causal interpretation of ecological overlap; site-specific outcome matching; explicit consideration of tobacco, age, mobility, referral, and diagnostic access.

The Haryana evidence base is uneven: cancer surveillance is stronger than exposure-linked epidemiology, groundwater measurements are more abundant than longitudinal health data, and occupational exposure remains under-characterised. The studies below are therefore interpreted according to what they directly measured rather than the importance of the hazard alone.

Table 2: Characteristics and critical appraisal of principal Haryana-focused evidence

Source	Design/period	Setting/sample	Direct contribution	Critical limitation
Chaturvedi et al. [9]	Statewide cancer atlas; 2016-2017	21 districts; 36,736 confirmed cancers; 126 centres plus eligible records from 28 HBCRs	District/sex/site AARs; cumulative risk; tobacco-related cancer proportions	Best peer-reviewed statewide spatial cancer description; not a PBCR and no individual environmental exposure linkage.
NCDIR [8]	Administrative cancer-atlas report	42,699 registrations from 149 sources	District cancer counts and AAR maps	Broader administrative total than the analytic paper; source coverage and referral can affect district comparisons.
CGWB [58]	National groundwater monitoring; 2023 samples	Haryana: 879 nitrate/fluoride and 857 arsenic/uranium samples	14.56% nitrate, 23.66% fluoride, 0.7% arsenic, and 18.7% uranium exceedance	Cross-sectional water-quality surveillance; does not provide lifetime individual dose or cancer outcomes.
Daulta et al. [14]	Cross-sectional groundwater survey	Eastern Haryana, including Panipat-Sonipat belt	Uranium distribution and ingestion-risk estimates	Useful spatial evidence; risk estimates depend on assumed intake and do not establish observed cancer incidence.
Duggal et al. [15]	Cross-	Western Haryana	Uranium	Substantial spatial variation; no

	sectional groundwater survey		concentrations and modelled radiological/chemical risk	individual health follow-up or cancer ascertainment.
Mehra et al. [16]	Groundwater risk assessment	Mahendragarh district	Natural uranium and ingestion-risk metrics	Localised sampling and model-based risk; limited temporal and clinical linkage.
Duggal et al. [17]	Groundwater risk assessment	Hisar district	Uranium concentration and ingestion-risk estimates	District-specific evidence; water-source history, renal biomarkers, and cancer outcomes were not longitudinally assessed.
Tanwer et al. [18]	Groundwater distribution and health-risk assessment	Panchkula district	Uranium distribution and chemo-radiological risk	Lower concentrations than several western/eastern studies; illustrates marked intrastate heterogeneity.
Malik et al. [19]	Haryana-focused review	Statewide literature	Sources, distribution, health risks, and remediation for inorganic groundwater contamination	Integrative but dependent on heterogeneous secondary studies; not cancer epidemiology.
Mishra et al. [20]	Environmental residue monitoring and risk assessment	Vegetables, soil, and water in Haryana	Pesticide residues and dietary/non-dietary risk indices	Demonstrates exposure plausibility; residue or risk-index exceedance is not a cancer endpoint.
Dutta and Chavalparit [38]	North Indian PM2.5 synthesis	Multiple North Indian states, including Haryana context	Exposure, health, environmental, and economic implications	Regional rather than individual Haryana cancer linkage; useful for exposure context.
Behlam et al. [21]	Case-control genetic association study	North Indian breast-cancer population	MTHFR and CYP1A1 variants	Suggests susceptibility research directions; replication, exposure measurement, and interaction testing are required.
Interpretive safeguard: This review distinguishes (1) presence of a hazardous agent, (2) population exposure, (3) biological plausibility, and (4) demonstrated contribution to cancer. Only the fourth supports local causal attribution.				

III. CANCER SURVEILLANCE AND THE PROBLEM OF ECOLOGICAL INTERPRETATION

The Haryana Cancer Atlas remains the most detailed statewide district comparison in the public domain. In the peer-reviewed analytic report, 36,736 confirmed cancers diagnosed in 2016-2017 were collated: 18,712 in 2016 and 18,024 in 2017. Data came from 126 participating centres in Haryana and neighbouring states and were supplemented with eligible records from 28 hospital-based cancer registries. Mandatory fields, ICD-O-3/ICD-10 coding, centre-level clinical oversight, electronic data capture, data-quality queries, and multi-identifier de-duplication strengthened validity [9]. The administrative atlas reported 42,699 registrations from 149 sources, a broader total that should be distinguished from the final peer-reviewed analytic dataset [8].

District variation in age-adjusted incidence was substantial. Among men, Rohtak had the highest all-site AAR (143.9 per 100,000), followed by Gurugram (124.8), Jhajjar (109.9), Hisar (105.3), Jind (104.7), and Faridabad (94.8). Among women, Gurugram had the highest all-site AAR (108.2), while Ambala also showed a comparatively high rate (85.7). Site-specific signals included male head-and-neck cancer AARs of 53.5 in Rohtak, 48.9 in Jhajjar, and 48.6 in Jind; lung cancer in men was highest in Rohtak (15.1); prostate cancer was highest in Gurugram (18.5); breast cancer was highest in Gurugram (34.5) and was 26.8 in Ambala; and cervical cancer was highest in Rohtak (11.8) [9]. Figure 1 presents these reported signals without implying environmental causation.

Environmental cancer research requires site-specific outcomes. All-site incidence combines malignancies with distinct causes, latency periods, sex distributions, and diagnostic pathways.

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The atlas reported particularly high proportions of tobacco-related cancers among men in Kaithal (58.6%) and among women in Kaithal (20.8%) and Hisar (20.6%), reinforcing the need to control tobacco before attributing head-and-neck or lung cancer patterns to ambient pollution [9], [22]. Breast, cervical, and prostate patterns may also reflect reproductive, hormonal, screening, and urbanisation-related factors. Exposure-cancer hypotheses should therefore be specified by agent, route, cancer site, sex, and latency.

The official incidence estimates for 2020-2024 are projections based on national registry methods, not direct enumeration of every new case. The atlas itself was designed to address surveillance gaps and explicitly recommended establishing PBCRs in suitable Haryana districts [9]. International experience shows that high-quality cancer atlases depend on stable population denominators, comparability, completeness, mortality linkage, and repeated periods of observation [23], [24], [25]. Haryana therefore needs continuous PBCR-quality registration before temporal trends or exposure-linked clusters can be interpreted confidently.

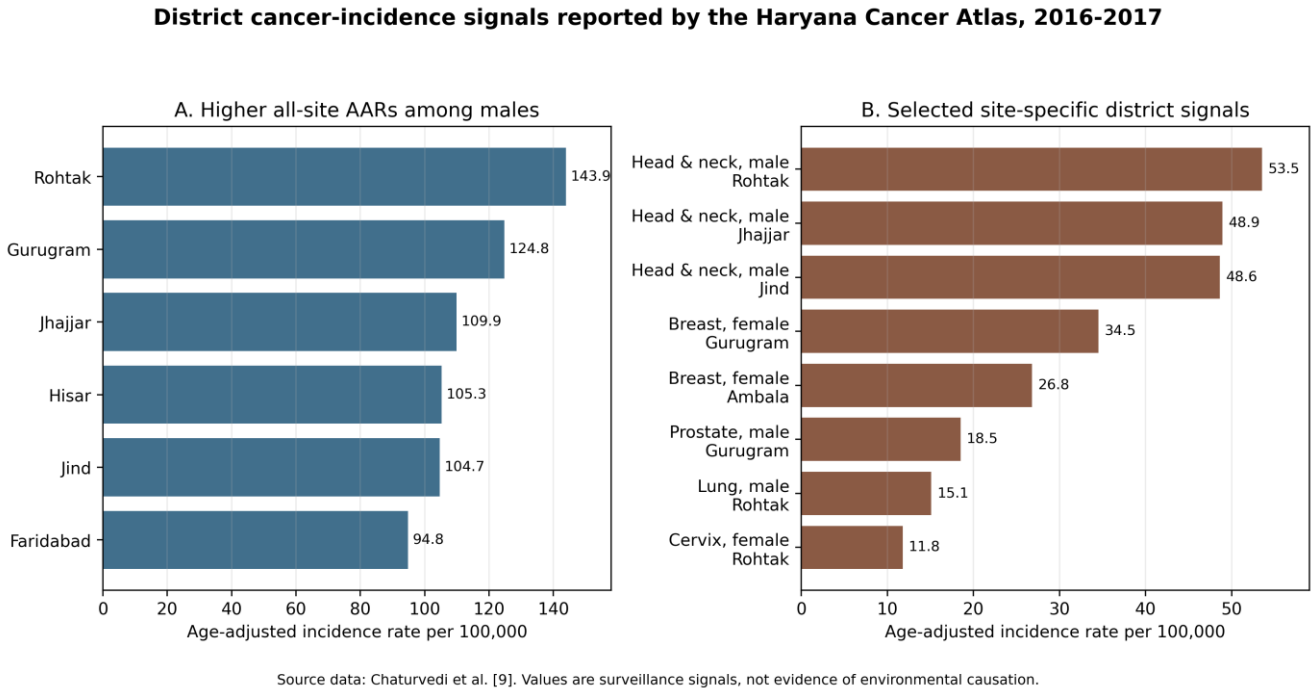

Figure 1: Selected district and site-specific cancer-incidence signals reported by the Haryana Cancer Atlas (2016-2017). AARs are surveillance measures and do not establish environmental causation. Data source: Chaturvedi et al. [9].

IV. AN ECO-ONCOLOGY FRAMEWORK FOR HARYANA

Environmental carcinogenesis is best understood as a chain rather than a list of pollutants. Emission or contamination sources enter air, water, soil, food, or workplace microenvironments; people are exposed through inhalation, ingestion, dermal contact, and, in some cases, prenatal pathways; biological processes such as oxidative stress, chronic inflammation, DNA damage, altered DNA repair, epigenetic dysregulation, and clonal promotion influence disease development. The final cancer pattern is modified by age, sex, tobacco and alcohol use, nutrition, occupation, genetics, residential mobility, exposure duration, latency, healthcare access, and registration completeness. Figure 2 presents this framework and explains why simple geographic overlap cannot establish causation. This life-course formulation is consistent with the exposome concept, which emphasises the totality, timing, interaction, and biological embedding of environmental exposures rather than isolated single-agent snapshots [26], [27].

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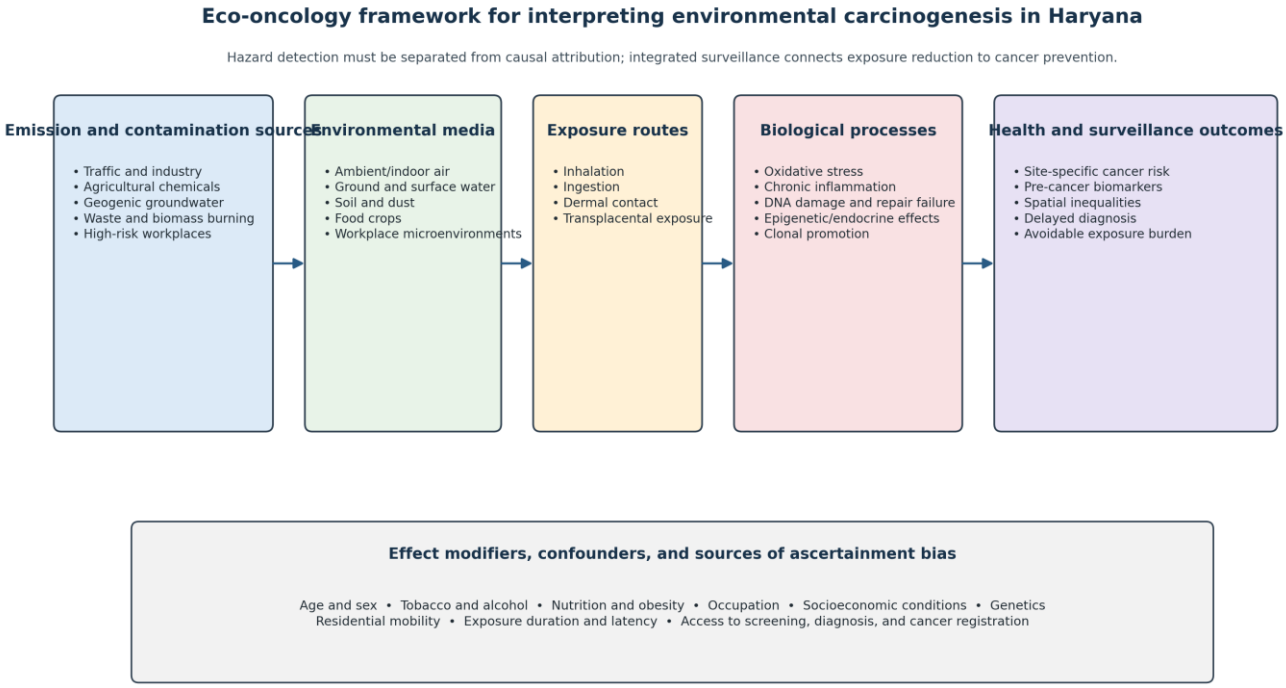

Figure 2: Eco-oncology framework for interpreting environmental carcinogenesis in Haryana.

V. MAJOR EXPOSURE DOMAINS

5.1 Ambient air pollution and combustion mixtures

Outdoor air pollution is the exposure domain with the strongest general causal foundation. IARC classifies outdoor air pollution and particulate matter in outdoor air as carcinogenic to humans (Group 1) [28], [29]. Long-term PM2.5 exposure has been associated consistently with lung-cancer incidence and mortality in cohort studies and meta-analyses, including among never-smokers [30], [31], [32], [33], [34], [35]. The mixture contains or carries combustion-derived PAHs, metals, elemental carbon, secondary inorganic aerosols, and reactive constituents; gaseous co-pollutants such as nitrogen dioxide and benzene can indicate traffic and industrial sources.

Haryana's exposure context is heterogeneous. Faridabad and Gurugram are influenced by dense traffic, construction, regional transport, and National Capital Region pollution; Panipat and Sonipat combine traffic with industrial and energy-related sources; agricultural districts experience seasonal biomass and crop-residue burning; and households using solid fuels may experience high indoor exposure. State-level disease-burden analyses and North Indian pollution studies demonstrate substantial health impacts, but they do not directly quantify cancer risk for individual Haryana residents [36], [37], [38]. India-wide analyses also show that air pollution imposes large mortality and economic burdens, strengthening the prevention rationale even before Haryana-specific cancer fractions can be estimated [39], [11].

The strongest biological evidence concerns the lung. Chronic exposure can induce oxidative stress and inflammatory signalling, while experimental and human translational evidence indicates that PM exposure may promote expansion of pre-existing mutant epithelial clones. Hill et al. [45], for example, linked particulate exposure to interleukin-1β-mediated promotion of EGFR-mutant lung cells, illustrating a tumour-promoting mechanism that does not require air pollution to initiate every mutation. Associations with breast, liver, gastrointestinal, brain, and childhood cancers are under active investigation, but are less consistent and more vulnerable to exposure misclassification and residual confounding [40], [41], [42], [43], [44].

For Haryana, the inference is therefore strong that air-pollution reduction is a cancer-prevention measure, especially for lung cancer, but limited regarding the fraction of observed district cancer burden attributable to PM2.5. A defensible local study would reconstruct multi-year residential exposure before diagnosis, distinguish ambient from occupational and household sources, and control for tobacco, age, socioeconomic position, and diagnostic access.

5.2 Pesticides and agricultural exposure

Agricultural exposure is a major concern because Haryana's high-input farming can generate repeated contact among applicators, family members, nearby residents, and consumers. Exposure may occur during mixing and spraying, through

drift, contaminated clothing and household dust, residues on food, or contamination of soil and water. A Haryana-specific study reported pesticide residues in vegetables, soil, and water and identified samples with potential dietary or non-dietary risk [20]. Such findings justify exposure reduction and surveillance, but a residue measurement is not equivalent to a cancer diagnosis or a quantified lifetime dose. The Agricultural Health Study and international agricultural cohorts demonstrate why product-specific exposure histories and prospective follow-up are more informative than treating "pesticides" as a uniform exposure category [46], [47], [48].

Cancer evidence is compound-specific. IARC evaluated diazinon, glyphosate, and malathion as probably carcinogenic to humans, while parathion and tetrachlorvinphos were classified as possibly carcinogenic; these conclusions should not be transferred automatically to chlorpyrifos, triazophos, or every pesticide detected in Haryana [49]. Epidemiological syntheses suggest associations between selected occupational or household pesticide exposures and non-Hodgkin lymphoma, childhood leukaemia, and colorectal cancer, but heterogeneity is substantial and exposure assessment is often based on job title, self-report, or broad chemical classes [50], [51], [52]. Meta-analytic evidence for non-Hodgkin lymphoma also varies substantially by active ingredient, study design, and exposure definition [53].

Mechanistically, some pesticides can induce oxidative stress, genotoxicity, endocrine disruption, immune alteration, or epigenetic change, while organophosphates also inhibit cholinesterase [54]. Biomonitoring studies of exposed workers have reported DNA damage and biochemical abnormalities, but biomarkers of effect are not cancer-specific and cannot be interpreted as proof of future malignancy [55], [56], [57].

The highest-value Haryana research would be a prospective agricultural-worker cohort or a carefully designed case-control study with crop-specific task histories, product names, application frequency, protective practices, residential proximity, pesticide metabolites, and relevant cancer outcomes. In the meantime, prevention does not need to wait: licensing and sales tracking, integrated pest management, substitution of highly hazardous products, applicator training, re-entry intervals, and affordable protective equipment are justified by acute and chronic toxicity even where cancer attribution remains uncertain.

5.3 Groundwater, nitrate, uranium, arsenic, and industrial metals

Groundwater contamination is among the clearest environmental-management priorities in Haryana, but the carcinogenic significance differs sharply by contaminant. The Central Ground Water Board's 2024 national report, based on 2023 sampling, analysed 879 Haryana samples for nitrate and fluoride and 857 for arsenic and uranium. It reported nitrate above 45 mg/L in 14.56% of samples and across 21 districts, fluoride above 1.5 mg/L in 23.66% of samples and 17 districts, arsenic above 10 µg/L in 0.7% of samples and five districts, and uranium above 30 µg/L in 18.7% of samples and 16 districts [58]. These data indicate a geographically widespread multi-contaminant problem, but they do not provide individual exposure duration or cancer outcomes.

Inorganic arsenic is an established human carcinogen associated with skin, lung, and bladder cancer, and is also linked to cardiovascular and other chronic disease [59]. Hexavalent chromium and cadmium are established carcinogenic hazards in relevant exposure circumstances, while lead has important systemic toxicity but is not interpreted identically. Industrial and geogenic sources must therefore be chemically speciated wherever possible. A total "heavy metal" value is insufficient for cancer-risk interpretation.

Uranium requires particular caution. Multiple studies have reported elevated uranium concentrations in parts of Hisar, western Haryana, Mahendragarh, Panipat, Sonipat, and Panchkula, with spatial variation linked to hydrogeology, groundwater chemistry, fertilizer use, and other possible anthropogenic influences [60], [14], [15], [16], [17], [18], [19]. The WHO provisional guideline of 30 µg/L is based predominantly on chemical toxicity to the kidney rather than radiological cancer risk [61]. Therefore, describing every uranium exceedance as a direct cause of cancer is scientifically inaccurate. Exceedance nevertheless warrants source control, alternative water supply, treatment, and health surveillance because chronic nephrotoxicity and uncertainty at high exposure remain important. Human studies of chronic uranium ingestion have more consistently focused on renal tubular and glomerular effects than on cancer, which supports a kidney-toxicity-centred interpretation of drinking-water guidance [62], [63].

Nitrate contamination is strongly linked to agricultural nitrogen inputs, sanitation, and hydrogeological conditions. Ingested nitrate can contribute to endogenous formation of N-nitroso compounds under conditions influenced by diet, antioxidants, infection, and microbial processes. IARC has evaluated ingested nitrate or nitrite under conditions that result in endogenous nitrosation as probably carcinogenic to humans, while epidemiological reviews and a nationwide Danish cohort have reported associations with colorectal and other cancers at varying exposure ranges [64], [65], [66]. Haryana-specific cancer attribution is not yet available and requires long-term household water-source histories. Fluoride exceedance is an important cause of dental and skeletal fluorosis and should be managed urgently, but fluoride is not treated here as an established environmental carcinogen.

For industrially affected water systems, monitoring should include arsenic species, chromium(VI), cadmium, nickel, lead, volatile organic compounds, and source-specific organic pollutants rather than relying only on broad water-quality indicators. The Ghaggar and other receiving systems require coordinated upstream-downstream monitoring, effluent source apportionment, and testing of drinking-water and food-chain pathways. Cancer-focused research should prioritise

contaminants with established site-specific carcinogenicity and verified chronic human exposure.

5.4 Industrial emissions and occupational carcinogens

Haryana's industrial economy includes petrochemical, textile, dyeing, metal-processing, rubber, engineering, and waste-related activities. Workers may encounter benzene, aromatic amines, solvents, welding fumes, silica, hexavalent chromium, nickel compounds, formaldehyde, and combustion products. Several of these agents are established or probable human carcinogens in specific exposure settings. Benzene, for example, is a Group 1 carcinogen with strong evidence for haematological malignancy [67]. IARC evaluations identify several of these agents or exposure circumstances as established human carcinogens, including benzene and relevant exposures to welding fumes, certain metals, silica, and aromatic amines [68], [67], [69].

Occupational risk is often concentrated in contract, informal, and small-enterprise work where exposure records, ventilation, substitution, personal protective equipment, and medical surveillance are weakest. Community exposure can also occur through fugitive emissions, contaminated dust, effluent, and take-home contamination on clothing. Yet the manuscript evidence base contains little Haryana-specific occupational cancer epidemiology. Assertions of increased bladder cancer, leukaemia, or lung cancer in particular worker groups should therefore be framed as biologically plausible priorities for surveillance rather than documented local excesses.

A state occupational carcinogen programme should establish industry-specific exposure inventories, job-exposure matrices, periodic industrial hygiene measurements, and worker health records that remain available across employment changes. Biomonitoring may be useful for selected agents, but it should complement-not replace-engineering controls. The hierarchy of controls remains essential: elimination or substitution, enclosure and ventilation, administrative controls, and only then personal protective equipment.

5.5 Open waste burning, informal recycling, and mixed combustion exposure

Open burning of municipal, plastic, electronic, agricultural, and biomedical waste generates a poorly characterized mixture of fine particles, black carbon, polycyclic aromatic hydrocarbons, dioxins and furans, acid gases, and metals. The exact composition depends on the waste stream and combustion temperature. India's waste-management literature identifies open burning and under-reporting as major barriers to sustainable control [70]. Global emission inventories show that uncontrolled domestic-waste burning releases particulate matter, carbon monoxide, PAHs, dioxin-related compounds, and other hazardous air pollutants; modelling studies indicate a material chronic PM2.5 mortality burden, although cancer-specific attribution remains uncertain [71], [72].

Cancer relevance arises because the mixture can include agents with established carcinogenicity, but direct epidemiological evidence for residents near Haryana burn sites is not currently sufficient to quantify excess cancer risk. Informal waste pickers and recyclers may face higher and more continuous exposure through smoke, dust, dermal contact, and contaminated food or water. Children may have additional vulnerability due to hand-to-mouth behaviour and longer future latency.

Policy should focus on preventing combustion rather than measuring smoke after the event. Source segregation, reliable collection, material-recovery facilities, safe management of e-waste and biomedical waste, enforcement against burning, and formal inclusion of waste workers in occupational-health systems are core interventions. Sentinel exposure studies could measure personal PM2.5, urinary PAH metabolites, blood metals, and respiratory or genotoxicity markers, but cancer outcomes require long-term follow-up.

5.6 Indoor air pollution and household exposure

Household exposure can arise from solid-fuel cooking and heating, kerosene, environmental tobacco smoke, mosquito coils, incense, and high-temperature cooking in poorly ventilated spaces. Women, children, older adults, and people spending more time indoors may receive disproportionate doses. Evidence from prospective research has linked frequent indoor wood-burning to lung-cancer risk, although stove design, ventilation, fuel type, and smoking remain important modifiers [73]. IARC has classified household combustion of coal as carcinogenic to humans, while epidemiological syntheses and contemporary cohort evidence support concern regarding lung cancer from long-term household combustion exposure [74], [75], [76], [73].

For Haryana, the present evidence does not support assigning a specific fraction of cancer to household fuel use. Nevertheless, clean cooking energy, kitchen ventilation, smoke-free homes, and safer combustion practices provide immediate respiratory and cardiovascular benefits and are consistent with cancer prevention. Future surveys should avoid a simple binary classification of "biomass use" and instead capture fuel stacking, stove type, ventilation, time-activity, and historical changes in fuel use.

Table 3: Evidence appraisal for major environmental exposure domains relevant to cancer in Haryana

Exposure domain	General carcinogenic evidence	Haryana-specific evidence	Inference for Haryana	Priority evidence gap
Outdoor air pollution/PM2.5	Strong causal evidence for lung	High exposure plausibility in	Moderate for local contribution; strong	Long-term geocoded exposure, smoking control,

	cancer; suggestive evidence for several other sites.	NCR, industrial corridors, and seasonal burning regions; limited individual-level cancer linkage.	basis for prevention.	site-specific incidence, latency.
Pesticides	Compound-specific; strongest signals for selected lympho-haematological and other cancers.	Haryana residue and occupational exposure evidence exists; no robust prospective cancer cohort.	Limited to moderate, depending on compound and outcome.	Product-specific histories, biomarkers, task-based exposure, confounder control.
Arsenic/Cr(VI)/cadmium	Established human carcinogens in relevant routes and doses.	Localized water/industrial exposure plausible; chemical speciation and chronic-dose data incomplete.	Moderate where exposure is verified; local cancer attribution untested.	Speciation, source apportionment, individual water histories, site-specific cancers.
Uranium in groundwater	WHO guideline primarily reflects chemical nephrotoxicity; environmental-level cancer evidence is limited.	Frequent exceedance in monitoring and multiple district studies.	High water-quality concern; low certainty for local cancer causation.	Longitudinal exposure, renal outcomes, isotope/radiological characterization, cautious cancer analysis.
Nitrate in groundwater	Biologically plausible via endogenous N-nitroso formation; epidemiology is context-dependent.	Widespread exceedance; duration and dietary modifiers not measured.	Limited for Haryana cancer attribution.	Historical nitrate, diet, vitamin intake, microbiome/medical factors, GI cancer outcomes.
Industrial/occupational agents	Strong for specific agents such as benzene, aromatic amines, silica, and Cr(VI).	High-risk sectors present; exposure and cancer registries are poorly linked.	Moderate plausibility, limited local quantification.	Job-exposure matrices, industrial hygiene, worker cohorts, employment histories.
Open waste burning	Mixture contains several carcinogenic agents; direct community cancer evidence is limited.	Burning is common, but site-specific exposure measurements are sparse.	Limited for cancer; strong justification for emission control.	Personal monitoring, source characterization, worker follow-up.
Indoor combustion	Evidence strongest for selected solid-fuel/wood-smoke scenarios and lung cancer.	Household fuel patterns are changing and under-characterized.	Limited local inference.	Fuel stacking, ventilation, time-activity, smoking and socioeconomic adjustment.

VI. SPATIAL VULNERABILITY WITHOUT CAUSAL OVERSTATEMENT

The spatial convergence of environmental hazards and cancer incidence is important, but it should be described as a research-priority pattern rather than a proven cluster. Chaturvedi et al. [9] identified a higher male all-site AAR belt across Rohtak, Gurugram, Jhajjar, Hisar, Jind, and Faridabad, while female patterns included Gurugram and Ambala. The same study documented substantial site-specific variation and high tobacco-related cancer proportions in selected semi-urban districts. These results justify etiological studies, but they do not demonstrate that industrial, agricultural, air, or water pollution caused the observed rates.

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Urban and industrial districts such as Gurugram, Faridabad, Panipat, and Sonipat warrant air-pollution and occupational studies. Gurugram and Faridabad may simultaneously exhibit ascertainment advantages because of hospital density, specialist access, private diagnostics, and referral. Rohtak and Jhajjar require site-specific analyses that control tobacco and separate local residence from referral to the major Rohtak medical centre. Hisar, Sirsa, and Fatehabad require integrated agricultural-task and lifetime water-source assessment. Kaithal requires explicit tobacco adjustment given the atlas finding that 58.6% of male cancers were tobacco-related. The appropriate output is therefore a prioritisation matrix, not a causal risk map.

Gene-environment research is potentially informative but currently preliminary. A North Indian case-control study reported associations involving MTHFR and CYP1A1 variants in breast cancer [21], but such findings require replication, careful population stratification, and measured environmental exposure. Genetic susceptibility should not be used to shift responsibility from controllable pollution to individuals, nor should it justify population genetic screening before analytical validity, clinical utility, consent, and equity are established.

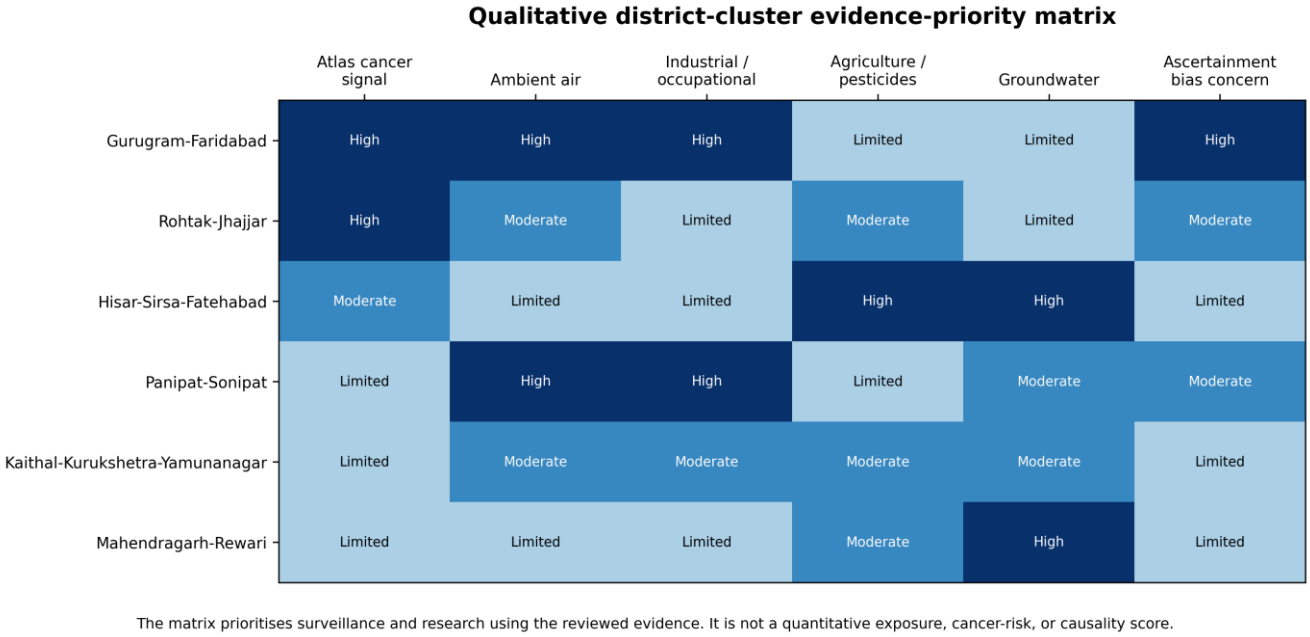

Figure 3: Qualitative district-cluster evidence-priority matrix derived from the reviewed cancer-surveillance, environmental, and ascertainment evidence. The matrix guides study design and is not a quantitative risk or causality score.

Table 4: Hypothesis-generating spatial priorities (not causal classifications)

Priority setting	Current signal	Relevant exposure profile	Research need
Gurugram-Faridabad	Higher reported cancer incidence in the atlas; dense diagnostic and referral infrastructure.	Traffic, regional PM2.5, construction, industrial and occupational mixtures.	Historical air-exposure reconstruction and separation of exposure from ascertainment effects.
Rohtak-Jhajjar	High male age-adjusted incidence reported in the atlas.	Peri-urban traffic, seasonal burning, mixed urban-rural sources.	Site-specific cancer registry linkage, residential history, and smoking-adjusted spatial analysis.
Hisar-Sirsa-Fatehabad	Hisar included in higher-incidence male belt; agricultural and water-quality concerns.	Pesticide tasks, nitrate, uranium, dust, and agricultural combustion.	Agricultural-worker cohort and lifetime drinking-water exposure assessment.
Panipat-Sonipat	Strong industrial and transport exposure plausibility; cancer evidence less complete.	Refinery/textile/industrial emissions, traffic, groundwater contaminants.	Worker and community exposure studies linked to site-specific cancer registration.
Mahendragarh and	No basis for broad cancer	Uranium, salinity, fluoride,	Safe-water intervention evaluation

western groundwater-stressed areas	attribution from current data.	nitrate, and hydrogeological vulnerability.	and long-term renal and cancer surveillance.
Ambala and northern districts	Female incidence pattern and preliminary gene-environment research.	Mixed urban, agricultural, and transport exposures.	Replication with measured exposures and cancer subtype information.

VII. GOVERNANCE AND SURVEILLANCE GAPS

The principal institutional weakness is fragmentation. Health systems record diagnoses; pollution-control agencies monitor ambient or industrial parameters; groundwater agencies sample water; agricultural departments regulate inputs; labour departments oversee workplaces; and municipalities manage waste. These systems rarely share identifiers, geographies, time scales, or public dashboards. As a result, Haryana can identify hazards and cancer cases but cannot reliably connect exposure histories to disease. Because many carcinogenic exposures are preventable, surveillance should be designed to trigger source reduction rather than only describe disease [77].

Surveillance continuity is equally important. The Cancer Atlas demonstrated the feasibility of multi-centre notification, but etiological research requires a permanent registry architecture with completeness audits, mortality linkage, tumour-site coding, stage, geocoded usual residence, migration, and occupational fields. Cancer notification without stable follow-up and denominator quality cannot substitute for a PBCR-quality system.

Environmental monitoring is often designed for regulatory compliance rather than exposure science. A station or groundwater sample may characterize a location at one time, while cancer reflects cumulative exposure over decades. Monitoring networks should retain raw data, methods, detection limits, quality assurance, and source metadata. Historical satellite products, land-use regression, industrial inventories, crop and pesticide records, and water-source histories can help reconstruct past exposure.

Regulatory action is also weakened when exceedance is treated as a binary event. Risk management should consider concentration, duration, population served, contaminant toxicity, feasibility of alternative supply, and vulnerable groups. Public disclosure must be accompanied by a remediation plan; otherwise dashboards can document risk without reducing it.

VIII. AN EVIDENCE-TO-ACTION POLICY ROADMAP

The policy objective should be to reduce established exposures immediately while building the data required to test uncertain cancer associations. Table 5 organizes actions by responsible institutions, time horizon, and measurable indicators.

Table 5: Policy roadmap for prevention and integrated surveillance

Priority action	**Lead institutions**	**Time horizon**	**Core indicators**
Create a Haryana Cancer-Environment Surveillance Platform	Health Department, NCDIR/ICMR, HSPCB, CGWB, Agriculture and Labour Departments	0-2 years	Data-sharing protocol; common geocodes; annual site-specific cancer and exposure report.
Sustain PBCR-quality cancer registration	Health Department and NCDIR/ICMR	0-3 years	Coverage and completeness metrics; mortality linkage; stage, occupation, and residence history fields.
Prioritize safe water in high-exceedance blocks	Public Health Engineering, CGWB, Panchayats, Jal Jeevan Mission	Immediate and ongoing	Population served by compliant water; repeat-confirmation testing; treatment performance; source-switching completion.
Strengthen air-pollution prevention	HSPCB, urban local bodies, transport and industry agencies	0-5 years	Annual population-weighted PM2.5; source-specific emission reductions; rural-industrial monitor coverage.
Establish occupational carcinogen surveillance	Labour Department, ESIC, industry associations, HSPCB	1-4 years	Job-exposure matrices; exposure measurements; worker enrolment; engineering-control compliance.
Implement pesticide stewardship	Agriculture Department, CIBRC-linked enforcement,	0-4 years	Digital sales records; training coverage; PPE access; highly hazardous pesticide substitution; residue trends.

	extension services		
Eliminate open waste burning and formalize waste work	Urban local bodies, HSPCB, Health and Social Justice Departments	Immediate and ongoing	Verified burning events; segregated collection; material-recovery capacity; registered workers with health coverage.
Require environmental health assessment for major projects	Environment Department and HSPCB	1-3 years	Health-impact component in approvals; baseline exposure data; post-clearance health and emission audit.

IX. RESEARCH AGENDA FOR CAUSAL INFERENCE

• Build a stable, population-based, site-specific cancer registry and preserve exact residential geocodes under secure governance.
• Reconstruct historical exposure windows of at least 10-20 years using monitoring archives, satellite products, industrial inventories, pesticide-use records, water-source histories, and residential mobility.
• Conduct multicentre case-control studies for biologically matched outcomes: lung cancer with PM2.5 and occupational combustion; leukaemia with benzene; bladder/skin/lung cancers with arsenic; bladder cancer with aromatic amines; and selected lympho-haematological cancers with specific pesticides.
• Establish an agricultural-worker cohort with repeated exposure measurement, product-specific records, protective practices, biomarkers, and long-term linkage to cancer outcomes.
• Develop job-exposure matrices for Haryana's textile, refinery, metal, welding, rubber, pesticide, e-waste, and waste-management sectors.
• Use spatial Bayesian models only after aligning exposure periods, cancer sites, age/sex denominators, tobacco prevalence, deprivation, urbanization, healthcare access, and spatial autocorrelation.
• Evaluate natural experiments and interventions-for example, source switching for contaminated water, industrial emission controls, clean-fuel adoption, or waste-burning elimination-to determine whether measured exposure and intermediate biomarkers decline.
• Embed community participation, risk communication, privacy, and environmental-justice analysis so that data collection leads to remediation rather than stigma.

A key methodological principle is outcome-exposure matching. Broad indices of "environmental degradation" should not be regressed against all-site cancer without a causal model. Each study should pre-specify the agent, route, latency, plausible cancer site, confounders, and expected direction of effect. Negative findings should be published, and uncertainty should be quantified rather than hidden behind hotspot maps.

X. DISCUSSION

This review finds a mismatch between the strength of evidence that hazardous exposures exist in Haryana and the strength of evidence that those exposures explain observed cancer patterns. The first proposition is well supported by air-quality evidence, groundwater monitoring, residue studies, occupational plausibility, and established carcinogen classifications. The second remains incompletely tested because the state lacks continuous population-based incidence series linked to historical individual exposure. The Cancer Atlas provides a strong surveillance foundation, but its principal peer-reviewed contribution was to demonstrate district and site-specific heterogeneity and the feasibility of coordinated electronic registration-not to prove environmental causation [9].

The most defensible cancer-prevention priority is ambient and occupational air pollution. Outdoor air pollution is an established human carcinogen, Haryana includes several high-exposure settings, and emission reduction has broad benefits beyond cancer. Arsenic, benzene, hexavalent chromium, cadmium, silica, and selected aromatic amines also warrant strong precaution where exposure is verified. Pesticide policy should be chemical-specific and should avoid describing an entire pesticide class as carcinogenic. Uranium should be treated as a serious drinking-water contaminant while acknowledging that the principal WHO guideline rationale is nephrotoxicity, not proven cancer risk at typical environmental exposure.

The review also clarifies the interpretation of district patterns. Rohtak's high male all-site AAR and high head-and-neck and lung rates require attention, but the high proportion of tobacco-related cancers and the presence of a major referral centre are competing explanations. Gurugram's high all-site, female breast, and male prostate rates may reflect urban exposures, lifestyle transition, age structure, screening, and diagnostic access. Agricultural and groundwater-intensive western districts are high priorities for exposure research, but the available studies mostly measure environmental concentrations rather than cancer. A credible next generation of research must align historical exposure windows, biologically matched cancer sites, residential mobility, tobacco, occupation, and healthcare access.

Environmental justice is central. Rural households dependent on a contaminated borewell, contract workers in poorly monitored industries, informal waste workers, and communities near combustion or effluent sources may have the least capacity to avoid exposure and the weakest access to early diagnosis. Prevention should not depend on individuals purchasing private filtration, air purifiers, or protective equipment. The state has a responsibility to control sources,

provide safe public infrastructure, and disclose risks in a form that enables action. Children and other biologically or socially vulnerable groups may experience higher dose per body mass, longer future latency, and fewer resources for avoidance or treatment [78].

The proposed surveillance platform is feasible because the relevant institutions and data streams already exist. The innovation lies in linking them through common geographies, time stamps, quality standards, and governance. Such a platform could support rapid remediation of known hazards, formal evaluation of interventions, and increasingly rigorous causal studies. It would also provide a model for other Indian states facing similar combinations of industrialization, intensive agriculture, groundwater stress, and incomplete cancer surveillance. This approach is internationally transferable to other low- and middle-income regions where industrialisation, intensive farming, informal work, groundwater dependence, and fragmented data systems coexist. Its value lies in preventing both under-reaction to established hazards and over-interpretation of ecological coincidence.

XI. STRENGTHS AND LIMITATIONS OF THE REVIEW

This review's principal strengths are its explicit separation of hazard, exposure, and causal attribution; reconciliation of the administrative and peer-reviewed Haryana Cancer Atlas datasets; integration of cancer registration with compound-specific carcinogenic evidence; and transparent appraisal of Haryana-focused studies. It also links surveillance to feasible prevention and research actions through an eco-oncology and exposome-informed framework [26], [27].

The review is comprehensive and critically structured but not systematic; it does not claim exhaustive retrieval, duplicate screening, or formal risk-of-bias scoring. Publication bias, inaccessible local reports, heterogeneous sampling, inconsistent analytical quality, and reliance on secondary monitoring data remain limitations. The district-priority matrix is qualitative and should not be interpreted as a risk score. Cancer-atlas rates may be influenced by referral, diagnostic access, incomplete registration, changing denominators, and short observation periods. Environmental studies often use contemporary cross-sectional measurements that cannot reconstruct exposure during the long latency of cancer. Most importantly, the review cannot estimate population-attributable cancer fractions for any Haryana exposure.

XII. CONCLUSION

Haryana has a credible environmental cancer-prevention problem, but not yet a complete environmental cancer-causation dataset. The Haryana Cancer Atlas established substantial district and site-specific variation, including high male all-site and head-and-neck rates in Rohtak and high female all-site and breast-cancer rates in Gurugram, while also demonstrating the feasibility and limitations of coordinated atlas-based surveillance. Environmental monitoring confirms preventable exposure concerns involving ambient and occupational air pollution, pesticide handling, groundwater contaminants, industrial mixtures, and uncontrolled combustion. Immediate prevention is justified for established hazards even where the fraction of cancer attributable to those hazards is unknown. The scientific priority is continuous PBCR-quality registration linked securely to historical, geocoded, and route-specific exposure data. Such integration would allow Haryana to progress from ecological concern to testable causal inference and would provide a transferable model for environmental-cancer surveillance in rapidly changing, data-constrained regions.

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