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Household Environmental Pollutants and Cancer Risk in Adults: An Integrated Review from Environmental Health and Oncology Perspectives

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Abstract

Background: Household environments can expose adults to carcinogens such as radon, secondhand smoke, solid-fuel emissions, cooking fumes, VOCs, pesticides, and water contaminants. This review synthesized evidence on the association between household environmental exposures and cancer risk in adults, with emphasis on environmental health and oncology perspectives.

Materials and Methods: A structured narrative review of Persian- and English-language studies (2000–March 2026) was conducted across PubMed/MEDLINE, Scopus, Web of Science, CIVILICA, and Google Scholar. Eligible studies assessed adult household exposures to radon, tobacco smoke, solid fuels, indoor chemicals, pesticides, or water contaminants with cancer outcomes. Findings were thematically synthesized by pollutant, pathway, outcome, evidence consistency, limitations, and prevention potential.

Results: Residential radon and secondhand tobacco smoke showed the most consistent association with increased lung cancer risk. Evidence for household coal combustion was strong, while biomass-fuel smoke and cooking fumes demonstrated moderate, context-dependent associations influenced by fuel type, stove design, ventilation, and exposure duration. Although benzene and formaldehyde are recognized carcinogens, evidence for cancer risk at typical household levels remained limited. Associations between household pesticide exposure and selected hematological malignancies, and between nitrate or trihalomethane exposure in drinking water and gastrointestinal or bladder cancer, were heterogeneous and generally of low-to-moderate certainty. Key limitations included exposure misclassification, reliance on self-reported exposure, and confounding by active smoking, socioeconomic conditions, diet, occupational exposure, and outdoor air pollution.

Conclusion: Radon, secondhand smoke, and solid-fuel combustion are key modifiable household exposures linked to lung cancer. Priority prevention includes radon testing and remediation, smoke-free homes and vehicles, cleaner household energy, improved ventilation, safer chemical use, water-quality monitoring, and systematic environmental exposure assessment in primary and oncology care.

Key Words: Adults, Cancer, Household environmental exposure, Indoor air pollution, Oncology.

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1- INTRODUCTION

Cancer is one of the most significant public health challenges and a leading cause of death worldwide. The rising global cancer burden is associated with population growth and ageing, changing lifestyle patterns, urbanization, and continued exposure to environmental risk factors. Cancer comprises a heterogeneous group of diseases characterized by uncontrolled cell proliferation, invasion of adjacent tissues, and, in some cases, metastasis to distant organs. Consequently, prevention, early diagnosis, treatment, and palliative care constitute the core pillars of cancer control (1, 2).

Advances in diagnosis and treatment—including surgery, radiotherapy, chemotherapy, targeted therapies, and immunotherapy—have improved outcomes for many malignancies. However, a sustained reduction in the cancer burden is not achievable without serious attention to primary prevention. In this context, identifying and controlling modifiable risk factors, particularly environmental exposures, is of considerable importance (1, 2). The concept of the "exposome" further emphasizes the role of cumulative environmental exposures across the lifespan and their interactions with genetic and biological factors in the development of chronic diseases, including cancer (3).

The home is one of the most important human exposure environments, as individuals spend a substantial proportion of their time in residential settings. Poor indoor air quality, inadequate ventilation, secondhand tobacco smoke, combustion of wood, coal, and other solid fuels, use of inefficient heating appliances or stoves, certain building materials, and some consumer products can all increase exposure to harmful pollutants. Some of these sources introduce particulate matter, benzene, formaldehyde, carbon monoxide, nitrogen oxides, and polycyclic aromatic hydrocarbons into the home environment (4, 5). Radon is a colorless, radioactive gas that is naturally released from soil and rock and can accumulate in buildings when ventilation is inadequate. Radon decay products emit alpha particles that can cause genetic damage in lung tissue. A pooled analysis of data from 13 European case–control studies has shown that increased residential radon concentration is associated with an elevated risk of lung cancer. This association is particularly important in smokers and individuals with a history of tobacco use, as the effects of radon and tobacco smoke can act synergistically (6, 7).

Secondhand tobacco smoke and pollution from household fuel combustion are important and preventable exposures. The International Agency for Research on Cancer (IARC) has classified secondhand tobacco smoke and emissions from household coal combustion as Group 1 carcinogens (carcinogenic to humans). Emissions from biomass combustion, particularly wood, have been classified as probably carcinogenic to humans (Group 2A) (8, 9). Pooled analyses and systematic reviews have also supported an association between household air pollution, secondhand smoke exposure, and increased lung cancer risk (10, 11).

However, the mere presence of a pollutant in the home environment does not inevitably cause cancer. Concentration and duration of exposure, age and health status of the individual, building characteristics, ventilation levels, tobacco use, socioeconomic status, and co-exposure to multiple pollutants can all modify the ultimate risk. Furthermore, differences in exposure measurement methods, study designs, definitions of pollutant sources, and control of confounders make direct comparison of research findings difficult. Therefore, when interpreting evidence, it is essential to distinguish between classifying an agent as carcinogenic, demonstrating a

statistical association, and establishing a causal relationship (3, 8, 9). From an environmental health perspective, identifying pollutant sources, monitoring indoor air quality, improving ventilation, reducing the use of polluting fuels, controlling radon, and eliminating indoor tobacco smoking are important measures for reducing exposure (4, 5, 8, 12). From an oncology perspective, understanding environmental factors that influence cancer can strengthen primary prevention, educate patients and families, identify at-risk groups, and inform health policy. Thus, collaboration among environmental health specialists, epidemiologists, toxicologists, family physicians, and oncologists is essential to translate evidence into effective interventions.

1.1. Objective

The objective of this narrative review is to provide an integrated summary of existing evidence on the association between household environmental pollutants and cancer risk in adults, from the shared perspective of environmental health and oncology. This article focuses specifically on radon, secondhand tobacco smoke, pollution from household fuel combustion, and certain chemical pollutants in indoor environments, and examines the importance of primary prevention, reduction of modifiable exposures, and interdisciplinary collaboration to reduce the cancer burden.

2- MATERIALS AND METHODS

2-1. Study design

This study was conducted as a structured narrative review. The rationale for selecting this approach was to integrate and interpret existing evidence on sources of household environmental pollutants, exposure pathways, potential carcinogenic mechanisms, and their oncological outcomes in adults. Given the diversity of pollutants, differences in exposure assessment methods, variability in study designs, and heterogeneity of cancer types, a narrative synthesis was deemed appropriate for organizing, comparing, and interpreting the available evidence (13, 14).

Although this study was not a meta-analysis, the stages of literature search, source selection, data extraction, and evidence interpretation were conducted in a structured and transparent manner. In reporting the methodology, components of the SANRA scale—including stating the importance of the topic, defining objectives, describing the search strategy, adequacy of referencing, scientific reasoning, and appropriate presentation of outcome data—were addressed (15).

2-2. Search strategy and information sources

A structured search of the scientific literature was conducted in both English and Persian across the following databases: PubMed/MEDLINE, Scopus, Web of Science, Scientific Information Database (SID), Magiran, and Civilica. Google Scholar was also used to identify additional relevant sources, previous reviews, and studies that may not have been retrieved from the primary databases.

In addition to scientific databases, official websites and documents from the World Health Organization (WHO), the International Agency for Research on Cancer (IARC), and the United States Environmental Protection Agency (EPA) were reviewed. These sources were used to identify reference reports, specialised definitions, classifications of carcinogenic agents, disease burden estimates, and preventive recommendations (4, 16, 17).

The search timeframe was set from 1 January 2000 to December 2025, with the final search completed in March 2026. Sources published before 2000 were reviewed and included if they had fundamental or historical importance and

were directly relevant to the study objective. These sources included reference studies on radon, secondhand tobacco smoke, household air pollution, the exposome concept, and carcinogenicity assessments of pollutants.

The search strategy in English-language databases was designed using a combination of controlled vocabulary, including MeSH terms in PubMed, and free-text words in titles and abstracts. The strategy was based on three main domains:

- Household environmental pollutants and exposures;
- Cancer and oncological outcomes;
- Adult populations and human studies.

An example of the search strategy used in PubMed was as follows:

("Indoor Air Pollution"[Mesh] OR "indoor air pollution"[tiab] OR "household air pollution"[tiab] OR "household environmental pollutant*"[tiab] OR "residential air pollution"[tiab] OR "indoor exposure"[tiab] OR "biomass fuel"[tiab] OR "solid fuel"[tiab] OR "secondhand smoke"[tiab] OR "passive smoking"[tiab] OR "environmental tobacco smoke"[tiab] OR radon[tiab] OR formaldehyde[tiab] OR benzene[tiab] OR "volatile organic compound*"[tiab] OR pesticide*[tiab] OR "drinking water contaminant*"[tiab] OR trihalomethane*[tiab] OR nitrate[tiab])
AND
("Neoplasms"[Mesh] OR cancer*[tiab] OR neoplasm*[tiab] OR carcinoma*[tiab] OR malignan*[tiab] OR "lung cancer"[tiab] OR "bladder cancer"[tiab] OR leukemia[tiab])
AND
("Adult"[Mesh] OR adult*[tiab] OR "never-smoker*"[tiab] OR "non-smoker*"[tiab]).

In Persian-language databases, combinations of the following terms were searched using the logical operators AND and OR: "آلودگی هوای داخل ساختمان" (indoor air pollution), "آلودگی هوای خانگی" (household air pollution), "آلاینده‌های محیطی خانگی" (household environmental pollutants), "سوخت زیست‌توده" (biomass fuel), "سوخت جامد" (solid fuel), "رادون" (radon), "دود دست‌دوم دخانیات" (secondhand tobacco smoke), "ترکیبات آلی فرّار" (volatile organic compounds), "بنزن" (benzene), "فرمالدهید" (formaldehyde), "آفت‌کش‌های خانگی" (household pesticides), "آلودگی آب آشامیدنی" (drinking water contamination), "سرطان" (cancer), "سرطان ریه" (lung cancer), "بدخیمی" (malignancy), "انکولوژی" (oncology), and "بزرگسالان" (adults).

2-3. Inclusion criteria

Sources were included in the review if they met one or more of the following criteria:

- Human epidemiological studies, including cohort, case–control, and cross-sectional designs;
- Systematic reviews, meta-analyses, and relevant narrative reviews;
- Reports, guidelines, and official documents from reputable organisations, including WHO, IARC, and EPA;
- Studies published in Persian or English;
- Studies conducted in home environments, residential settings, household cooking spaces, or other residential environments;
- Studies that examined at least one of the following pollutants or exposures:
    - Residential radon;
    - Secondhand or environmental tobacco smoke;
    - Pollution from combustion of solid fuels and biomass;

    - Smoke and vapors from cooking;
    - Particulate matter and other combustion products in indoor environments;
    - Volatile organic compounds, benzene, and formaldehyde;
    - Polycyclic aromatic hydrocarbons;
    - Pesticides and insecticides used in household environments;
    - Drinking water pollutants associated with residential environments, including nitrates and trihalomethanes;
- Studies in which the study population consisted of adults aged 18 years and older, or where results for adults were reported separately;
- Studies that examined one of the following outcomes:
    - Cancer incidence;
    - Cancer-specific mortality;
    - Cancer type, stage, or prognosis;
    - Epidemiological indicators related to cancer risk;
    - Biomarkers related to carcinogenesis, provided that their association with household exposure and the carcinogenic pathway was interpretable.

Animal, laboratory, and cellular studies were not included in the primary analysis of human evidence. However, where directly relevant, they were used to explain potential carcinogenic mechanisms and to support biological interpretation of human findings (4, 16).

2-4. Exclusion criteria

The following were excluded from the review:

- Laboratory or animal studies that had no direct relevance to the interpretation of human evidence;
- Studies that examined exposure solely in occupational, industrial, or outdoor urban environments without a specific component of household exposure;
- Studies in which the study population consisted exclusively of children or adolescents;
- Studies that did not report cancer outcomes or cancer-related indicators;
- Case reports, letters to the editor, conference abstracts, news items, non-scientific materials, and sources lacking sufficient methodological information;
- Duplicate studies or reports based on the same dataset where findings had been presented in a more complete or recent article;
- Articles that did not provide sufficient information on the study population, type of exposure, exposure environment, or cancer outcome.

In studies that included a broad age range, the study was included only if data for adults could be extracted or interpreted separately (17, 18).

2-5. Study selection and screening

After transferring search results to reference management software, duplicates were identified and removed. Titles and abstracts of the remaining sources were then independently screened by two researchers. Sources that appeared relevant to the target population, exposure, and outcome based on title and abstract were selected for full-text assessment.

In the second stage, full texts of selected sources were evaluated against the inclusion and exclusion criteria. Reasons for excluding studies at the full-text assessment stage were recorded. Disagreements between the two researchers were resolved through discussion and consensus. Where disagreements persisted, the matter was referred to a third researcher with expertise in epidemiology for final decision-making. This process was undertaken to reduce selection bias and increase reliability in source selection (15, 19).

2-6. Data extraction and organization

For each included source, key data were extracted and recorded in a researcher-designed table, including: first author name and year of publication; country or region where the study was conducted; source type and study design; sample size and population characteristics; type of pollutant and its household source; exposure measurement method or indicator; duration and intensity of exposure (where reported); type of cancer or oncological outcome; effect estimates (odds ratio, relative risk, or hazard ratio) and 95% confidence intervals (where reported); main confounding factors (particularly tobacco use, socioeconomic status, occupational exposure, and outdoor air pollution); key study findings; methodological limitations; and conflicts of interest and funding sources (where reported).

To avoid inappropriate pooling of heterogeneous evidence, data were organized according to pollutant type, exposure source, cancer outcome, and study design. The main synthesis themes included: residential radon and lung cancer; secondhand tobacco smoke and lung cancer; combustion of solid fuels and biomass; chemical pollutants in indoor air; drinking water and pollutants associated with residential environments; potential carcinogenic mechanisms; and prevention and exposure reduction strategies (4, 8–10, 17, 18).

2-7. Evidence synthesis and interpretation

Findings were synthesized descriptively and thematically, first by pollutant type and then by cancer type and level of evidence. Where multiple studies examined the same exposure and outcome, results were compared in terms of direction of association, effect size, consistency of findings, presence of dose–response relationships, methodological quality, and extent of attention to confounding factors.

Due to substantial heterogeneity in the definition and measurement of exposures, study methods, and cancer outcomes, quantitative pooling of results and calculation of new effect estimates were not performed. Therefore, effect sizes and numerical estimates reported in this article were directly extracted from primary studies or reputable reviews and presented with due consideration of the context and limitations of each study (14, 18, 20).

In interpreting findings, distinctions were made between the following three concepts:

- Potential carcinogenicity: based on toxicological, mechanistic, and epidemiological evidence;
- Statistical association: observation of an association between an exposure and an outcome in human studies;
- Causal relationship: a stronger conclusion requiring concurrent consideration of consistency, dose–response relationship, temporality, biological coherence, and exclusion of alternative explanations.

The IARC classification was used to describe the level of evidence for

carcinogenicity of an agent, but this classification was not interpreted as a direct estimate of an individual's probability of developing cancer at a specific level of exposure (4, 16).

2-8. Quality assessment of sources

The quality of sources was assessed based on study type and methodological characteristics. Components considered included clarity of exposure definition, validity of measurement methods, adequacy of sample size, quality of cancer outcome definition, control of confounding factors, completeness of results reporting, and transparency in stating limitations.

In interpreting observational studies, the potential for selection bias, recall bias, exposure misclassification, residual confounding, and the impact of concurrent exposures was considered. Additionally, results from studies with very high exposure levels were not generalized to typical living conditions without explicitly stating relevant limitations.

The level of confidence in evidence was expressed descriptively based on convergence of results, reproducibility of findings across different populations, presence of dose–response relationships, biological coherence, and overall study quality. In this review, SANRA was used to assess the quality of narrative review reporting and was not used as a substitute for risk of bias assessment in primary studies (15).

2-9. Ethical considerations

This study was a literature review based on published data and involved no direct intervention on humans or animals, biological sampling, access to individual records, or use of identifiable data. Therefore, obtaining informed consent from participants was not applicable. Furthermore, in accordance with the regulations of the implementing institution and the requirements of the ethics committee and target journal, obtaining a specific ethics code for this study was deemed unnecessary.

Throughout all stages, principles of scientific integrity were upheld, including accurate referencing of primary sources, distinguishing findings from primary studies from review results, balanced reporting of concordant and discordant findings, avoidance of selective omission of evidence, and prevention of plagiarism. Limitations arising from study heterogeneity, differences in exposure assessment methods, potential publication bias, and language search restrictions were also considered in the final interpretation of results. To mitigate these limitations, searches were conducted across multiple Persian and English databases, sources were independently reviewed by two researchers, a third researcher was consulted in cases of disagreement, hand-searching of references and citation tracking were performed, inclusion and exclusion criteria were documented, data were extracted in a structured manner, and evidence was interpreted conservatively. These actions were consistent with existing recommendations for writing transparent and rigorous narrative reviews (19, 20).

2-10. Methodological limitations

Given the narrative nature of the study, there is a possibility that some relevant sources were omitted or that researcher judgement influenced the selection and interpretation of evidence. Additionally, due to differences in pollutant definitions, exposure assessment methods, study populations, and cancer outcomes, direct comparison of results and quantitative conclusions were not feasible. To reduce these limitations, the search strategy, selection criteria, screening process, data extraction method, and basis for evidence interpretation were reported transparently.

3- RESULTS

This section presents the findings of the narrative synthesis of evidence on household environmental exposures and cancer outcomes. The residential environment may be a source of chronic, cumulative, and often imperceptible exposure to a wide range of pollutants, some of which have established carcinogenic hazards under occupational, experimental, or high-exposure conditions. However, the relevance of these hazards to household settings depends on exposure concentration, frequency and duration, ventilation, building characteristics, co-exposures, and individual biological susceptibility (1–27).

In terms of the direction and certainty of evidence, the reviewed pollutants were distributed across three broad spectra: relatively strong and consistent epidemiological evidence for radon and secondhand tobacco smoke in relation to lung cancer (1–6); moderate to relatively strong but context-dependent evidence for household combustion of solid fuels, benzene, and formaldehyde (7–12, 16, 25); and more heterogeneous evidence for pesticides, nitrates, and disinfection by-products in drinking water, including trihalomethanes (10–15, 23, 26, 27). Evidence concerning cooking fumes, particularly those generated during high-temperature frying in poorly ventilated kitchens, was suggestive but more susceptible to co-exposure confounding (7–9).

The findings are organized into eight themes, including the general characteristics of the evidence; major household pollutants such as radon, combustion products, volatile organic compounds, pesticides, and drinking-water contaminants; the synergistic role of building conditions; and preventive strategies. An overview of pollutants, sources and exposure pathways, cancer outcomes, and interpretive considerations is presented in **Table 1**. **Table 2** summarizes the main cancer outcomes, plausible biological mechanisms, major confounding or effect-modifying factors, and the narrative assessment of the direction, consistency, and certainty of evidence for each exposure. Proposed preventive interventions are presented in **Table 3**.

Given the non-meta-analytic nature of this review, the tables support narrative synthesis only and do not provide pooled quantitative estimates (15). The certainty categories in Table 2 represent a narrative, non-GRADE (Grading of Recommendations Assessment, Development and Evaluation) appraisal and should not be interpreted as formal certainty ratings. Reporting an association does not establish causality but indicates a potential increase in risk under specified exposure conditions (1–27).

3-1. General characteristics of the evidence and distribution of household exposures

Synthesis of the reviewed epidemiological and toxicological evidence indicated that household environmental pollutants may be associated with selected malignancies through multiple biological and physicochemical pathways. The main exposure domains identified were radon gas, secondhand tobacco smoke, combustion products from coal and biomass fuels, cooking fumes, benzene, formaldehyde and other volatile organic compounds, household pesticides, nitrates, and drinking-water disinfection by-products, particularly trihalomethanes.

Most available studies focused on adult cancer outcomes, especially lung cancer and selected hematological malignancies. However, evidence related to household pesticide exposure also included childhood malignancies, particularly leukemias and lymphomas. The strength, certainty, and reliability of evidence varied according to

pollutant type, concentration, exposure duration, route of entry, ventilation conditions, building characteristics, and methodological quality of the underlying studies.

A major pattern identified across studies was the distinction between the intrinsic carcinogenic hazard of a substance and the magnitude of risk under typical household exposure conditions. Classification of a chemical or exposure as carcinogenic based on occupational, experimental, or animal evidence does not necessarily imply an equivalent level of risk in residential environments. Estimating household cancer risk requires consideration of actual concentrations, cumulative duration and frequency of exposure, air-exchange rates, building features, concurrent exposures, and individual susceptibility (1–27).

3-2. Radon gas and naturally occurring radiation from the ground

Radon was identified as one of the most important radioactive pollutants in indoor environments. It is generated through the natural decay of uranium in soil and bedrock and may enter buildings through basements, cracks in floors and walls, gaps around pipes, drainage routes, service ducts, and, in some circumstances, building materials. Radon accumulation is more likely in poorly ventilated dwellings, enclosed basements, and buildings with low air-exchange rates (1–3).

Long-term exposure to radon and its short-lived decay products was consistently associated with an increased risk of lung cancer. Radon progeny can attach to airborne particles and, after inhalation, deposit in the respiratory tract. Alpha-particle radiation emitted at these sites may induce DNA double-strand breaks, genomic instability, mutations, and other carcinogenic alterations (1–3).

Evidence from miner studies, residential case–control studies, and pooled analyses supports a dose–response relationship between cumulative radon exposure and lung cancer risk. This association appears stronger in regions with substantial radon infiltration from the ground and in dwellings with insufficient ventilation (1–3). Radon is regarded as one of the leading preventable causes of lung cancer after active tobacco smoking; however, absolute risk depends on radon concentration, duration of residence, cumulative exposure, and smoking status.

The interaction between radon exposure and tobacco use is particularly important. Smokers exposed to radon have a substantially higher lung cancer risk than non-smokers, and the effects may be additive or interactive. Accordingly, radon testing in high-risk areas, remediation of buildings with elevated concentrations, and strengthening smoking-cessation programs are key preventive strategies (**Table 3**).

3-3. Combustion products: secondhand smoke, solid fuels, and cooking fumes

3-3-1. Secondhand tobacco smoke

Passive exposure to environmental tobacco smoke in homes and vehicles was recognized as one of the most important preventable carcinogenic exposures. Secondhand smoke contains fine particles, nicotine, benzene, formaldehyde, carbon monoxide, polycyclic aromatic hydrocarbons, and numerous other toxic and carcinogenic compounds (4–6).

Continuous inhalation of tobacco smoke in enclosed domestic environments was associated with an increased risk of lung cancer among non-smokers. The direction of association was relatively consistent across studies and was more pronounced among individuals exposed for prolonged periods at home or in vehicles (4–6). In addition to secondhand smoke, tobacco-related compounds may deposit on indoor surfaces, carpets, curtains, clothing, and household dust, creating potential

exposure to thirdhand smoke. Although concentrations in residential settings may be lower than in certain occupational environments, repeated and long-term exposure in homes where tobacco is used daily can result in a substantial cumulative exposure burden. Opening windows, using ventilation, or isolating smoking to a particular room may reduce concentrations of some pollutants but cannot eliminate exposure. Therefore, a complete ban on tobacco use in homes and vehicles is the most effective preventive strategy.

3-3-2. Household solid fuels

Combustion of biomass fuels, including firewood, agricultural residues, and animal dung, as well as household coal use for cooking and heating, releases substantial quantities of fine particulate matter, black carbon, polycyclic aromatic hydrocarbons, carbon monoxide, aldehydes, and volatile organic compounds into indoor environments (7–9).

Available evidence suggested that frequent and long-term use of solid fuels is associated with an increased risk of lung cancer. Within this exposure category, evidence of carcinogenic hazard was stronger and more consistent for household coal combustion, whereas evidence for biomass fuels was more context-dependent. Indoor emissions from household coal combustion have been classified as carcinogenic to humans, while indoor emissions from household biomass combustion, primarily wood, have been classified as probably carcinogenic to humans. These classifications describe carcinogenic hazard and do not indicate an identical level of risk at all household exposure concentrations (7–9).

Some studies have also reported associations between household solid-fuel exposure and cancers of the upper aerodigestive or digestive tract. However, these findings were generally less consistent than those for lung cancer and were influenced by fuel type, stove design, combustion efficiency, ventilation, cooking practices, and duration of exposure (7–9).

The highest exposure levels were reported in dwellings using traditional stoves without effective flues or with insufficient air exchange. Accordingly, replacing solid fuels with cleaner energy sources, improving stove efficiency, ensuring appropriate flue design and maintenance, and increasing ventilation are important exposure-reduction measures.

3-3-3. Cooking fumes and high-temperature frying

Cooking fumes generated during frying, grilling, stir-frying, and high-temperature heating of oils may contain fine particulate matter, aldehydes, polycyclic aromatic hydrocarbons, heterocyclic amines, and other volatile compounds. Studies conducted predominantly among women and non-smokers have reported associations between frequent exposure to poorly ventilated cooking fumes and increased lung cancer risk in selected populations (7–9).

Nevertheless, interpretation of these findings remains challenging. Cooking-fume exposure frequently co-occurs with exposure to solid-fuel combustion, inadequate kitchen ventilation, ambient air pollution, and secondhand tobacco smoke. Therefore, the independent contribution of cooking fumes, particularly emissions from overheated oils, cannot always be reliably distinguished.

The available evidence should consequently be considered suggestive to moderate and context-dependent rather than as proof that all cooking fumes cause lung cancer. The use of externally vented range hoods, adequate kitchen ventilation, and avoidance of excessive oil heating are pragmatic exposure-reduction strategies (7–9).

3-4. Volatile organic compounds and building-related chemical pollutants

3-4-1. Benzene

Benzene is an important volatile aromatic hydrocarbon in indoor air. Household sources include tobacco smoke, paints, solvents, adhesives, thinners, petroleum products, fuel vapors, and some consumer products. Benzene concentrations may also increase when petrol vapors enter from attached garages or when fuel and solvents are stored improperly within or near residential spaces (10–12).

Following metabolism, benzene is converted into reactive metabolites that can induce oxidative stress, chromosomal damage, disruption of hematopoietic stem-cell function, and bone-marrow toxicity. Epidemiological evidence, particularly from occupational settings, has established an association between benzene exposure and hematological malignancies, especially acute myeloid leukemia (10–12).

Benzene is a recognized human carcinogenic hazard; however, the certainty of evidence concerning cancer risk under typical household exposure conditions is more limited. Although benzene concentrations in homes are generally lower than in industrial settings, continuous and repeated exposure from multiple domestic sources may be relevant to environmental health. Direct extrapolation from high-dose occupational evidence to household conditions should nevertheless be cautious. Source identification, direct measurement of indoor concentrations, exposure duration, and assessment of concurrent occupational exposure are important for interpretation (10–12).

3-4-2. Formaldehyde

Formaldehyde may be released from medium-density fiberboard, pressed wood products, resins, insulation foams, parquet adhesives, furniture, paints, disinfectants, and selected cosmetic or household products. Emissions tend to be higher in newly constructed or recently renovated buildings and may increase with higher temperature and relative humidity (4, 16, 25). The synthesized evidence suggested that inhalation exposure to formaldehyde may be associated with chronic respiratory mucosal irritation and an increased risk of selected malignancies, particularly nasopharyngeal cancer under relevant exposure conditions. Plausible mechanisms include oxidative stress, DNA–protein cross-link formation, genetic damage, and impairment of DNA repair processes (4, 16, 25).

The IRIS toxicological assessment has evaluated human, animal, and mechanistic evidence related to formaldehyde toxicity and carcinogenicity (25). However, the relevance of household exposure depends strongly on indoor concentration, duration of contact, temperature, humidity, emission rate of materials, and ventilation. Therefore, direct generalization of evidence from high occupational concentrations to typical residential levels requires methodological caution.

3-4-3. Other volatile organic compounds

Other volatile organic compounds (VOCs) constitute a chemically heterogeneous group. Their toxicity, persistence, biological mechanisms, and carcinogenic potential differ substantially. Consequently, evidence concerning one compound, such as benzene or formaldehyde, cannot be automatically extrapolated to all VOCs.

Interpretation should focus on the specific compound, source, concentration, duration of exposure, route of entry, ventilation, and cancer outcome. At present, the available evidence does not support assigning one specific cancer outcome or one overall certainty level to all household VOCs (10–12).

3-5. Pesticides and household poisons

The use of insecticides, household poisons, herbicides, and pest-control products in homes or in areas adjacent to agricultural activity is a potential source of chemical exposure. These substances may enter the body through inhalation, dermal contact, inadvertent ingestion, contact with contaminated surfaces, and transfer into homes on shoes and clothing (10–12, 26).

Children may experience relatively higher exposure doses because of closer contact with floors and dust, hand-to-mouth behaviors, lower body weight, and developmental susceptibility. The reviewed evidence suggested that direct or indirect household exposure to certain pesticide classes may be associated with childhood leukemias, lymphomas, and selected solid tumors. However, these findings varied substantially according to active ingredient, exposure window, study design, and exposure-assessment method (10–12, 26).

Pesticides comprise a highly heterogeneous group of compounds that differ in chemical structure, mechanism of action, persistence, route of exposure, and bioaccumulation potential. Heterogeneity in active ingredients, reliance on parental self-reporting, variation in use patterns, and difficulties in measuring dose have resulted in low-to-moderate certainty of evidence. Thus, these findings should not be interpreted as evidence that all pesticides, all household-use patterns, or all exposure levels cause cancer.

Nonetheless, avoidance of unnecessary preventive spraying, implementation of integrated pest-management approaches, adherence to safe-use instructions, adequate ventilation during and after application, and secure storage away from children are reasonable precautionary measures (10–12, 26).

3-6. Household drinking-water pollutants

3-6-1. Nitrates

The evidence review indicated a possible association between elevated nitrate concentrations in drinking-water sources and selected gastrointestinal malignancies, particularly stomach and colorectal cancer (13–15). Biologically, nitrate may be converted to nitrite and, under certain conditions, contribute to the endogenous formation of N-nitroso compounds in the presence of amines or amides. Some N-nitroso compounds have demonstrated carcinogenic properties in laboratory studies. However, their formation within the body depends on numerous factors, including diet, gastric acidity, microbiome composition, vitamin C intake, other antioxidants, and consumption of processed meat (13–15).

Findings from human studies were heterogeneous. Variability in water-source quality, exposure assessment, dietary nitrate intake, processed-food consumption, nutritional status, genetic factors, and climatic conditions may explain discrepancies. Nitrates should therefore be considered a potential public-health concern requiring continued monitoring, rather than a definitive and independent cause of gastrointestinal or other cancers across all household exposure settings.

Proposed interventions include monitoring drinking-water sources, identifying areas with nitrate contamination, reducing agricultural fertilizer runoff, protecting wells from sewage contamination, and providing information on validated treatment methods. Any treatment recommendation should be appropriate for the contaminant and should preserve microbiological safety.

3-6-2. Trihalomethanes and other disinfection by-products

Trihalomethanes and other disinfection by-products are formed when disinfectants, particularly chlorine, react with naturally

occurring organic matter in water. Chloroform, bromodichloromethane, dibromochloromethane, and bromoform are among the most common trihalomethanes (23, 27).

Household exposure can occur through drinking water, cooking, inhalation of vapors during bathing or showering, and dermal absorption. Some epidemiological studies have reported an association between long-term exposure to higher concentrations of trihalomethanes and other disinfection by-products and increased bladder cancer risk. This association has been reported more frequently among individuals with prolonged exposure and higher estimated cumulative doses (23, 27).

The meta-analysis by Xie and colleagues reported evidence of an increased bladder cancer risk associated with trihalomethanes and other disinfection by-products. However, differences in exposure-assessment methods, temporal variation in concentrations, regional differences in water composition, concurrent exposure to multiple by-products, and variation in drinking and bathing behaviors limit interpretation (27).

Available evidence indicates a relevant public-health concern but remains insufficient to attribute a definitive cancer risk to all trihalomethanes or all household exposure levels. Evidence for colorectal or rectal cancer remains limited and inconclusive (23, 27).

Reducing or weakening drinking-water disinfection is not an appropriate solution, because it may increase the risk of waterborne infectious diseases. More appropriate approaches include optimization of water-treatment processes, reduction of precursor organic matter, use of pre-treatment technologies, and control of disinfection by-product formation while maintaining microbiological safety.

3-7. Synergy of building factors, indoor air quality, and preventive strategies

In addition to individual pollutants, physical and behavioral features of the residential environment influence cumulative exposure. Inadequate mechanical ventilation, high-emission building materials, inefficient or damaged flues, attached garages, elevated indoor temperature and humidity, improper storage of chemicals, and unsafe household practices can increase pollutant concentrations (16–24).

Household exposure often results from mixtures of pollutants rather than a single agent. For example, a home that uses solid fuel and has poor ventilation while tobacco is also used indoors may expose residents simultaneously to particulate matter, carbon monoxide, polycyclic aromatic hydrocarbons, benzene, aldehydes, and tobacco-related compounds. Such co-exposures may complicate estimation of the independent effect of individual pollutants and contribute to heterogeneity across studies (16–24).

Preventive measures should therefore not focus on a single pollutant. A multi-level approach should include source elimination or reduction, improved ventilation, use of low-emission materials, appropriate design and maintenance of flues, transition to clean energy, complete tobacco-free home and vehicle policies, safe pesticide practices, and periodic monitoring of indoor air and drinking-water quality.

3-8. Summary of results

Overall, this integrative narrative review showed that the home environment is not necessarily a safe or neutral setting with regard to exposure to carcinogenic agents. Rather, it may be a setting for chronic, imperceptible, and cumulative contact with multiple pollutants.

Among the exposures examined, radon and secondhand tobacco smoke demonstrated the strongest and most consistent epidemiological evidence for increased lung cancer risk. Evidence for household coal and other solid-fuel combustion was also notable, although the magnitude and certainty of association varied according to fuel type, combustion efficiency, stove and flue design, ventilation, and duration of exposure. Evidence concerning cooking fumes, particularly under conditions of high-temperature oil heating and inadequate ventilation, was more limited and vulnerable to co-exposure confounding (1–9).

Benzene and formaldehyde are important because of their established carcinogenic hazards and mechanistic evidence. However, estimating their cancer risk in residential settings depends on concentration, duration of contact, ventilation, source proximity, and concurrent exposures. For benzene, carcinogenicity is well established, but direct evidence concerning typical household exposure is more limited. Similarly, evidence concerning formaldehyde supports carcinogenic hazard under relevant exposure conditions but does not permit uniform risk estimation for all homes (10–12, 16, 25).

By contrast, evidence concerning pesticides, nitrates, and trihalomethanes was more heterogeneous and context-dependent. These findings should be interpreted cautiously because of exposure measurement error, substance-specific variability, confounding, temporal changes in exposure, and population heterogeneity. In particular, evidence for colorectal or rectal cancer in relation to trihalomethanes remains limited and inconclusive (13–15, 23, 26, 27).

These findings support a multi-level approach to risk reduction, ranging from radon monitoring and remediation, tobacco control, transition to cleaner fuels, and low-emission building materials to safe pesticide use, water-quality monitoring, optimization of disinfection processes, and enhancement of household environmental-health literacy. Proposed interventions are summarized in **Table 3**.

Table-1: Synthesis of evidence on household environmental exposures and cancer outcomes.

Pollutant or exposure	Main household sources or exposure pathways	Cancer outcomes reported	Summary of evidence and direction of association	Main limitations and interpretive considerations	References
Radon gas	Entry from soil, bedrock, structural cracks, basements, plumbing routes, service ducts, and poorly ventilated spaces	Lung cancer	Relatively strong and consistent epidemiological evidence; a cumulative exposure–response relationship has been reported	Actual radon concentration, cumulative exposure, residential duration, ventilation, building characteristics, and tobacco use	(1–3)
Secondhand tobacco smoke	Smoking cigarettes, waterpipes, or other tobacco products in homes and vehicles; inhalation and	Lung cancer, especially among non-smokers	Relatively strong and consistent epidemiological evidence of increased lung cancer risk among non-smokers; thirdhand smoke	Cumulative exposure assessment, workplace exposure, recall bias, active smoking misclassification, and ventilation	(4–6)

Pollutant or exposure	Main household sources or exposure pathways	Cancer outcomes reported	Summary of evidence and direction of association	Main limitations and interpretive considerations	References
	deposition on indoor surfaces		may contribute to persistent exposure		
Household coal combustion	Coal use for cooking or heating	Mainly lung cancer	Strong carcinogenic hazard; household cancer risk depends on coal type, combustion conditions, stove design, and ventilation	Coal type, stove design, flue efficiency, ventilation, exposure duration, and co-exposure to tobacco smoke	(7–9)
Biomass fuels	Firewood, agricultural residues, animal dung, and traditional stoves	Mainly lung cancer; possible upper aerodigestive or digestive cancers	Moderate and context-dependent evidence; higher risk has been reported with long-term exposure and poor ventilation	Fuel heterogeneity, exposure measurement, stove characteristics, poverty, tobacco-related confounding, and duration of exposure	(7–9)
Cooking fumes and high-temperature frying	Heated oils, frying, grilling, stir-frying, and inadequate kitchen ventilation	Lung cancer, particularly among women and non-smokers in some populations	Suggestive to moderate and context-dependent evidence; positive associations have been reported in selected populations	Difficulty separating cooking fumes from solid-fuel use, ambient air pollution, secondhand smoke, and socioeconomic factors	(7–9)
Fine particles and combustion products	Stoves, heaters, fireplaces, and heating appliances without effective ventilation	Mainly lung cancer	Biological and epidemiological evidence supports a potential carcinogenic role, but this represents a mixed exposure rather than a single pollutant	Difficulty separating independent effects of $PM_{2.5}$ from PAHs, gases, aldehydes, and other combustion pollutants	(7–9)
Benzene	Tobacco smoke, fuel vapours, paints, solvents, adhesives, petroleum products, consumer products, and attached garages	Leukaemia, particularly acute myeloid leukaemia	Benzene is a recognised human carcinogenic hazard; direct evidence for typical household exposure is more limited	Predominance of occupational evidence, concurrent sources, concentration, ventilation, exposure duration, and occupational co-exposure	(10–12)
Formaldehyde	Pressed wood, medium-density fibreboard, adhesives,	Nasopharyngeal cancer and selected respiratory	Evidence for carcinogenic hazard exists; household risk depends on	Pollutant mixtures, lack of longitudinal personal exposure monitoring, building	(4, 16, 25)

Pollutant or exposure	Main household sources or exposure pathways	Cancer outcomes reported	Summary of evidence and direction of association	Main limitations and interpretive considerations	References
	paints, furniture, insulation foams, disinfectants, and selected household products	malignancies under relevant exposure conditions	concentration, source strength, duration, temperature, humidity, and ventilation	age, temperature, humidity, and ventilation	
Other volatile organic compounds	Solvents, paints, adhesives, cleaning products, fragrances, fuels, furnishings, and consumer products	Compound-specific; no single cancer outcome can be assigned to all VOCs	Heterogeneous and compound-specific evidence; the evidence for one VOC cannot be generalised to all VOCs	Chemical identity, concentration, mixed exposures, ventilation, exposure duration, and limited longitudinal monitoring	(10–12)
Household pesticides and insecticides	Aerosols, insecticides, pest-control products, contaminated surfaces, agricultural drift, and take-home exposure on shoes or clothing	Childhood leukaemias, lymphomas, and selected solid tumours; possible adult haematological malignancies	Low-to-moderate certainty; evidence is limited and heterogeneous	Differences in active ingredients, frequency of use, exposure timing, agricultural or occupational exposure, recall bias, and self-reported exposure	(10–12, 26)
Trihalomethanes and other disinfection by-products	Drinking water, cooking, bathing, showering, inhalation of vapours, and dermal absorption	Bladder cancer; evidence for colorectal or rectal cancer remains limited and inconclusive	Possible association with bladder cancer, particularly after long-term higher-level exposure	Temporal variation in concentrations, other disinfection by-products, behavioural factors, water composition, and multiple exposure pathways	(23, 27)
Nitrates in drinking water	Water contaminated by agricultural runoff, nitrogen fertilisers, septic systems, or wastewater	Stomach and colorectal cancer in some studies	Possible associations have been reported, but evidence is heterogeneous and context-dependent	Diet, processed-meat intake, antioxidant intake, water source, microbiome, exposure misclassification, and other nitrate sources	(13–15)

Abbreviations: VOCs, volatile organic compounds; PAHs, polycyclic aromatic hydrocarbons; $PM_{2.5}$, particulate matter with an aerodynamic diameter less than or equal to 2.5 μm; DBPs, disinfection by-products; MDF, medium-density fiberboard.

Note: IARC classifications describe carcinogenic hazard and do not quantify the cancer risk associated with every household exposure level. The evidence assessments in this table refer specifically to the directness and consistency of evidence concerning household exposure.

Table-2: Cancer outcomes, plausible mechanisms, and narrative assessment of certainty of evidence.

Pollutant or exposure group	Main cancer outcomes	Plausible biological mechanisms	Important confounding or effect-modifying factors	Narrative assessment of direction, consistency, and certainty of evidence	References
Radon	Lung cancer	Alpha radiation, DNA double-strand breaks, mutations, and genomic instability	Active smoking, radon concentration, cumulative exposure, residential duration, and ventilation	Relatively high certainty; strong and consistent positive epidemiological association	(1–3)
Secondhand tobacco smoke	Lung cancer	Oxidative stress, chronic inflammation, DNA adduct formation, and mutagenesis	Active smoking, workplace exposure, duration of exposure, household smoking patterns, and ventilation	Relatively high certainty; relatively strong and consistent positive association among non-smokers	(4–6)
Household coal combustion	Primarily lung cancer; less consistent findings for other malignancies	Oxidative stress, inflammation, $PM_{2.5}$ exposure, PAHs, DNA damage, and impaired DNA repair	Coal type, stove and flue design, ventilation, socioeconomic status, tobacco use, and ambient pollution	High certainty for carcinogenic hazard; household risk remains strongly dependent on exposure conditions	(7–9)
Household biomass combustion	Primarily lung cancer; possible upper aerodigestive or digestive malignancies	Particulate matter, PAHs, aldehydes, oxidative stress, inflammation, and genotoxicity	Fuel type, stove and chimney, ventilation, duration of exposure, cooking role, tobacco smoking, and socioeconomic status	Moderate certainty; predominantly positive but heterogeneous and context-dependent association	(7–9)
Cooking fumes and high-temperature frying	Lung cancer	Fine particles, aldehydes, PAHs, heterocyclic amines, oxidative stress, and inflammation	Cooking style, fuel type, kitchen ventilation, tobacco smoke, and outdoor air pollution	Low-to-moderate certainty; suggestive positive association with substantial co-exposure confounding	(7–9)
Benzene	Leukaemia, especially acute myeloid leukaemia	Reactive metabolites, oxidative stress, bone-marrow toxicity, chromosomal damage, and haematopoietic stem-cell dysfunction	Occupational exposure, tobacco smoke, solvents, fuel storage, concentration, and duration of contact	High certainty for carcinogenic hazard but limited certainty for cancer risk under typical household exposure conditions	(10–12)

Pollutant or exposure group	Main cancer outcomes	Plausible biological mechanisms	Important confounding or effect-modifying factors	Narrative assessment of direction, consistency, and certainty of evidence	References
Formaldehyde	Nasopharyngeal and selected respiratory malignancies	DNA–protein cross-links, oxidative stress, local mucosal irritation, genetic damage, and impaired DNA repair	Indoor concentration, air-exchange rate, temperature, humidity, source strength, pollutant mixtures, and occupational exposure	Moderate certainty for carcinogenic hazard; limited-to-moderate certainty for household relevance, which is highly concentration-dependent	(4, 16, 25)
Other VOCs	Compound-specific	Compound-specific mechanisms, potentially including oxidative stress and genotoxicity	Chemical identity, concentration, co-exposures, ventilation, and duration	Low or insufficient certainty for a group-level cancer conclusion	(10–12)
Household pesticides	Childhood leukaemias, lymphomas, and selected solid tumours; possible adult haematological malignancies	Potential genotoxicity, oxidative stress, immune dysregulation, endocrine disruption, and epigenetic alterations	Pesticide class, active ingredient, exposure timing, frequency, dermal contact, agricultural exposure, and recall bias	Low-to-moderate certainty; heterogeneous evidence and no basis for generalisation to all pesticides	(10–12, 26)
Trihalomethanes and other DBPs	Bladder cancer; evidence for colorectal or rectal cancer is limited and inconclusive	Reactive metabolites, oxidative stress, cytotoxicity, and possible compensatory cell proliferation	Total water intake, bathing and showering behaviours, dermal and inhalational exposure, smoking, water composition, temporal variation, and co-exposure to other DBPs	Low-to-moderate certainty; possible positive association with bladder cancer at higher long-term exposure, but heterogeneous findings across settings	(23, 27)
Nitrates	Stomach and colorectal cancer	Endogenous formation of N-nitroso compounds, oxidative damage, and nitrosation reactions	Diet, processed meat, vitamin C and antioxidant intake, gastric acidity, microbiome, water quality, and other nitrate sources	Low-to-moderate certainty; highly variable and context-dependent evidence	(13–15)

Note: Certainty categories represent a narrative, non-GRADE assessment and should not be interpreted as formal certainty ratings. Biological plausibility and mechanistic evidence do not, by themselves, establish causality in humans.

Table-3: Proposed interventions to reduce household carcinogenic exposures.

Intervention priority	Target pollutant or exposure	Proposed intervention	Level of implementation	Potential implication for cancer prevention	References
High	Radon	Radon testing in high-risk areas; sealing entry routes; basement or substructure ventilation where appropriate; structural remediation, including sub-slab depressurisation where appropriate	Household, building sector, and environmental-health policy	Reduces exposure to an established cause of lung cancer	(1–3)
High	Secondhand tobacco smoke	Complete prohibition of tobacco use in homes and vehicles; provision of evidence-based tobacco-cessation support	Individual, family, health system, and community	Reduces exposure to multiple carcinogens and lung cancer risk among non-smokers	(4–6)
High	Coal and biomass fuels	Progressive replacement with cleaner fuels or technologies, such as electricity, natural gas, or LPG where feasible; use of efficient stoves and adequately designed and maintained flues	Household, energy policy, and housing sector	Reduces $PM_{2.5}$, black carbon, PAHs, carbon monoxide, and combustion-related lung cancer exposure	(7–9)
Moderate	Cooking fumes	Use of externally vented range hoods; cross-ventilation; avoidance of overheated oils and prolonged high-temperature frying	Household and architectural-design levels	Reduces exposure to cooking-related particles, aldehydes, and volatile compounds	(7–9)
Moderate	Formaldehyde and selected VOCs	Selection of low-emission materials and products; adequate ventilation after renovation, painting, or solvent use; control of indoor temperature and humidity	Household, construction industry, and building-regulation levels	Reduces cumulative inhalational exposure to potentially harmful chemical pollutants	(4, 10–12, 16, 25)
Moderate	Benzene and fuel vapours	Avoidance of indoor storage of fuels and solvents; prevention of vapour migration from attached garages; appropriate garage ventilation and safe maintenance of fuel containers	Household, building, and regulatory levels	Reduces exposure to a recognised haematotoxic and carcinogenic hazard	(10–12)
Moderate	Household	Integrated pest management; avoidance	Household and health-education	Reduces avoidable inhalational,	(10–12,

Intervention priority	Target pollutant or exposure	Proposed intervention	Level of implementation	Potential implication for cancer prevention	References
	pesticides	of unnecessary routine spraying; strict adherence to product safety instructions; adequate ventilation during and after application; safe storage away from children	levels	dermal, and ingestion-related exposure, particularly among children	26)
Moderate	Nitrates in drinking water	Routine water-quality monitoring; control of agricultural runoff and sewage contamination; use of validated treatment methods in high-risk areas	Water-supply system, environmental authorities, agricultural sector, and households	Reduces long-term exposure to elevated nitrate concentrations	(13–15)
Moderate	Trihalomethanes and other disinfection by-products	Reduction of organic precursors before chlorination; optimisation of treatment processes; appropriate pre-treatment; monitoring of disinfection by-products while maintaining effective disinfection	Water-supply system and environmental-health authorities	May reduce long-term exposure to disinfection by-products while preserving protection against waterborne infections	(23, 27)
Cross-cutting	All household exposures	Incorporation of residential and environmental exposure history into clinical assessment, especially for lung cancer in non-smokers; improvement of household environmental-health literacy	Primary care, oncology services, public health, and households	Supports identification of modifiable risk factors and more targeted preventive counselling	(1–27)

Note: Intervention priority reflects a qualitative combination of strength of evidence, preventability, potential exposure burden, feasibility, and public-health relevance. It does not represent a quantitative estimate of cancer reduction or a formal cost-effectiveness ranking. Measures intended to reduce chemical exposure should not compromise ventilation, heating safety, or the microbiological safety of drinking water.

4- DISCUSSION

4-1. Interpretation of the Main Findings

This integrative narrative review indicates that the home environment may represent a persistent setting for chronic and cumulative exposure to carcinogenic and potentially carcinogenic agents. Residential cancer risk depends on pollutant hazard, concentration, exposure duration, ventilation, building characteristics, source strength, exposure pathway, co-exposures, and individual susceptibility, consistent with the exposome framework (3, 12, 16). A central implication is distinguishing carcinogenic hazard from real-world residential risk; classification based on occupational, toxicological, experimental, or mechanistic evidence does not necessarily indicate that

typical household exposure produces the same magnitude of risk, especially given indoor pollutant mixtures (4, 5, 16, 18, 25, 28, 29).

The pollutants examined can be classified into three evidence groups. Radon and secondhand tobacco smoke showed the strongest and most consistent evidence, particularly for lung cancer. Household solid-fuel combustion, benzene, and formaldehyde showed moderate or exposure-condition-dependent evidence. Evidence for pesticides, nitrates, and trihalomethanes was more heterogeneous and context-dependent, reflecting synthesized evidence strength rather than formal IARC hazard classifications (16).

4-2. Radon and Secondhand Tobacco Smoke

Radon showed the strongest and most consistent evidence for an association with lung cancer. Findings from miner studies, the BEIR VI report, the WHO handbook on indoor radon, and the pooled analysis of 13 European residential case–control studies support an exposure–response relationship between residential radon exposure and lung cancer risk (6, 7, 21). Radon enters buildings mainly through soil and bedrock, cracks in floors and foundations, construction joints, plumbing pathways, and building materials, accumulating in basements, enclosed spaces, and low air-exchange environments (4, 21). Radon decay products may attach to airborne particles and deposit in the respiratory tract; alpha-particle emissions can induce complex DNA damage, double-strand breaks, chromosomal abnormalities, and genomic instability (6, 7, 21). Simultaneous radon exposure and tobacco smoking may interact biologically and statistically (6, 7, 21). An umbrella review identified radon as a robust environmental risk factor for lung cancer (30). Priority measures include residential radon testing, identification of high-risk buildings, sealing entry routes, improving subfloor ventilation, remediation, and post-intervention monitoring (21).

Evidence regarding secondhand tobacco smoke was strong and biologically plausible. The pooled analysis of the International Lung Cancer Consortium demonstrated an association between secondhand smoke exposure and lung cancer by histological type (11), supported by the US Surgeon General's report (22). Secondhand smoke contains fine particulate matter, benzene, formaldehyde, carbon monoxide, polycyclic aromatic hydrocarbons, and other toxic substances (11, 22). WHO states there is no safe level of secondhand smoke exposure; completely smoke-free homes and vehicles are required (17, 31). Ventilation or partial separation cannot eliminate exposure; complete prohibition is more effective. Thirdhand smoke may provide additional exposure through contaminated surfaces, clothing, carpets, and dust, but direct evidence linking it with cancer remains substantially weaker and should be described as a potential public-health concern.

4-3. Solid Fuels and Household Air Pollution

Household solid-fuel combustion represents an important exposure domain. IARC evaluations and WHO reports identify household use of polluting fuels as a major source of indoor air pollution and relevant carcinogenic exposure (8, 9, 17, 32). Lee and colleagues reported associations between household air pollution and adverse health outcomes, including lung cancer (10). Incomplete combustion of biomass, coal, charcoal, agricultural residues, and animal dung produces fine particulate matter, carbon monoxide, polycyclic aromatic hydrocarbons, benzene, aldehydes, and black carbon (8–10, 17), contributing to oxidative stress, chronic inflammation, epithelial injury, and DNA damage (10,

12, 17, 18, 33). A recent review emphasized that combustion-derived particulate matter and air pollutants may influence cancer through inflammatory, oxidative, and genotoxic pathways (33).

Exposure magnitude depends on fuel type, stove efficiency, chimney design, ventilation, room size, cooking duration, and time near cooking areas. Indoor residential exposures from cooking, smoking, heating, renovation, household products, and insecticides contribute to pollutant mixtures, modified by ventilation and air-exchange rates (28). Replacing solid fuels with cleaner energy may reduce lung cancer, respiratory disease, cardiovascular disease, and premature mortality risks (10, 12, 17, 32). Clean-energy interventions must consider affordability, energy access, fuel reliability, and cultural acceptability.

4-4. Benzene and Formaldehyde

Benzene and formaldehyde are important due to established carcinogenic hazards and plausible mechanisms. Benzene enters indoor air from tobacco smoke, combustion, fuel vapors, attached garages, paints, solvents, adhesives, and consumer products (4, 5, 18, 28). Metabolites induce oxidative stress, chromosomal damage, hematopoietic stem-cell impairment, and bone-marrow toxicity; occupational evidence supports associations with acute myeloid leukemia and hematological malignancies (4, 16).

Formaldehyde is emitted from medium-density fiberboard, particleboard, resins, adhesives, furniture, insulation, and household products, with higher emissions in new buildings and after installing new materials, increasing with temperature and humidity (4, 25). The 2024 EPA review evaluated human, animal, and mechanistic evidence for inhaled formaldehyde toxicity and carcinogenicity (25). Direct evidence for cancer risk from typical residential benzene and formaldehyde concentrations is more limited than occupational evidence; occupational data should not be extrapolated without considering indoor concentrations, exposure duration, ventilation, and source characteristics. These substances may contribute to cancer risk when exposure is elevated, prolonged, or from multiple sources, but mere detection does not establish increased risk. Preventive measures include source elimination, low-emission materials, adequate ventilation after painting or renovation, preventing fuel-vapor intrusion, and controlling temperature and humidity (4, 5, 25, 28).

4-5. Pesticides, Nitrates, and Trihalomethanes

Pesticide evidence was more heterogeneous. Pesticides represent diverse active substances with different structures, mechanisms, half-lives, and exposure pathways. Residential exposure occurs through direct use, contaminated surfaces, dust, clothing transfer, or agricultural drift. Some studies, particularly involving children, reported associations with leukemia, lymphoma, and solid tumors (26). However, Navarrete-Meneses and colleagues focused mainly on childhood cancer; findings should not be generalized to adults (26). Self-reported exposure, lack of biomonitoring, broad categories, recall inaccuracy, and inadequate occupational control reduce certainty. The statement “pesticides cause cancer” is not precise; interpretation requires active ingredient, dose, duration, pathway, and outcome specification. Integrated pest management, avoiding unnecessary spraying, safe storage, ventilation, and reducing contaminated surfaces are reasonable measures.

For nitrates, findings suggest possible associations with gastrointestinal cancers, particularly gastric and colorectal, although results are inconsistent (24). Nitrate conversion to nitrite may form N-nitroso compounds with amines or amides;

endogenous nitrosation depends on diet, gastric acidity, microbiome, vitamin C, and antioxidants (24). Nitrate risk should be interpreted with dietary patterns and other sources.

Trihalomethanes form when disinfectants react with organic matter in water. Household exposure occurs through drinking, cooking, bathing, inhalation, and dermal contact (23, 27, 34, 35). Meta-analyses by Villanueva and Xie support associations between long-term disinfection by-product exposure and bladder cancer (23, 27). Recent reviews suggest higher or prolonged exposure may increase cancer risk, particularly bladder cancer, limited by chemical composition, exposure assessment, treatment systems, and routes (34, 35). Reducing disinfection is inappropriate; preferred approaches control organic precursors, optimize treatment, apply pretreatment, and monitor microbiological safety and by-products.

4-6. Co-exposures and Common Mechanisms

Pollutants share mechanisms including DNA damage, oxidative stress, inflammation, impaired DNA repair, epigenetic alterations, chromosomal damage, and disrupted proliferation. Alpha particles from radon induce DNA damage and genomic instability (6, 7, 21). Fine particles and combustion products contribute to oxidative stress, inflammation, and epithelial injury (8–10, 18, 33). Benzene metabolites cause chromosomal damage and impaired hematopoiesis (4, 16); formaldehyde contributes through DNA–protein cross-links and genotoxicity (25). Nitrate and nitrite form N-nitroso compounds (24).

In homes, these mechanisms may operate simultaneously. Households using solid fuels, allowing indoor smoking, containing high-emission materials, and having inadequate ventilation represent mixture exposures. Co-exposures may influence individual pollutant effects and explain study differences. Current evidence is insufficient to quantify interactions; referring to possible additive or interactive effects is more appropriate than claiming synergistic toxicity.

4-7. Public Health Implications

Findings support multi-level household cancer prevention (10, 12, 17, 29, 32, 33). Household priorities include smoke-free homes and vehicles, cleaner fuels, improved ventilation, low-emission products, safe pesticide and solvent storage, and monitored drinking-water sources. Building-level priorities include radon testing, remediation, chimney maintenance, moisture control, ventilation, and low-emission materials (4, 5, 21, 25). Energy-efficiency renovations should assess ventilation to prevent radon and pollutant accumulation.

Policy priorities include equitable clean energy access, improved building standards, water contamination monitoring, chemical product regulation, national radon programs, and optimized disinfection (10, 12, 17, 21, 32). These measures are important for low-income households, rural communities, older buildings, and populations with limited access. Environmental justice should be considered; low-income and rural populations may experience higher exposure and fewer risk-reduction opportunities. Clinically, brief questions about household fuel, indoor smoking, ventilation, pesticide use, basement residence, attached garages, and water sources could support exposure-reduction counselling.

4-8. Strengths and Limitations

Strengths include an interdisciplinary approach, consideration of multiple pollutants, distinction between hazard and risk, and a structured narrative synthesis (13–15, 19, 20). Building characteristics,

ventilation, co-exposures, and social determinants were considered.

Limitations include the narrative (non-meta-analytic) design; conclusions represent evidence synthesis rather than pooled risk estimates (13–15, 19, 20). Studies were heterogeneous in exposure definitions, measurements, follow-up, outcomes, populations, and confounding adjustment. Exposure misclassification, recall bias, publication bias, absent direct measurements, and surrogate indicators limit certainty. Smoking, diet, socioeconomic status, location, occupation, outdoor pollution, housing quality, disease, and genetics may confound associations. Dietary patterns and nitrosation are important for nitrate interpretation; active ingredients and pathways complicate pesticide studies. Trihalomethane temporal and geographical variation and multiple routes remain uncertain. Some pesticide evidence derived from children or mixed-age populations (26); generalization to adults requires caution.

4-9. Recommendations for Future Research

Future research should adopt prospective, longitudinal, mixture-oriented, exposome-based designs (3). Priorities include repeated measurements, personal monitoring, biomonitoring, indoor sensors, source characterization, and standardized methods for radon, trihalomethanes, nitrates, pesticides, and VOCs. Studies should investigate radon–tobacco interactions, remediation effectiveness, clean-energy transitions, and retrofit effects on ventilation and accumulation. Cost-effectiveness and environmental-justice analyses are needed (12, 17, 29, 32, 33). Intervention studies should examine feasibility, affordability, equity, and effectiveness, comparing fuel replacement, improved stoves, chimneys, ventilation, and combined interventions.

5- CONCLUSION

This narrative review indicates that the home environment can be an important source of chronic, cumulative exposure to carcinogenic pollutants. The evidence is strongest and most consistent for residential radon and secondhand tobacco smoke as preventable causes of lung cancer, supported by dose–response relationships and biologically plausible mechanisms. Evidence also supports an association between household solid-fuel combustion and lung cancer, although the magnitude of risk varies according to fuel type, combustion conditions, ventilation, duration of exposure, and co-exposures.

By contrast, evidence concerning benzene, formaldehyde, pesticides, nitrates, and trihalomethanes remains heterogeneous and is highly dependent on exposure-assessment methods, building characteristics, environmental conditions, and concurrent exposures. A crucial distinction is therefore needed between intrinsic carcinogenicity and actual household risk: classification of a substance as carcinogenic on the basis of occupational or experimental evidence does not necessarily indicate an equivalent risk at typical residential exposure levels.

Effective prevention requires coordinated, multilevel strategies, including residential radon testing and remediation, smoke-free home policies, transition to cleaner household fuels, adequate ventilation, use of low-emission building materials, prudent pesticide use, and optimized water-treatment practices. Interdisciplinary collaboration among public-health professionals, clinicians, environmental scientists, and policymakers is essential to translate the available evidence into practical interventions and reduce the cancer burden attributable to household environmental exposures.

6- CONFLICT OF INTEREST: None.

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