

Overview of the epidemiologic studies on the health effects of ELF electric and magnetic fields (ELF-EMF) published in the fourth quarter of 2025.

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1. Reviews and meta-analyses

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2. Residential exposure

2.1 A Cohort Study on Alzheimer's Disease in Relation to Residential Magnetic Fields From Indoor Transformer Stations

Liimatainen, A., Roivainen, P., Juutilainen, J., Höytö, A., Naarala, J. (2025). Bioelectromagnetics, 46:e70031. <https://doi.org/10.1002/bem.70031>

Background and objective

Meta-analyses of epidemiological studies have suggested that Alzheimer's disease (AD) may be linked with exposure to extremely low frequency (ELF) magnetic fields (MF). This is the first study investigating the association of AD with exposure to residential ELF MFs from indoor transformer stations, using a study design that avoids shortcomings of previous studies.

Methods

All cohort members had lived in buildings with indoor transformer stations. MF exposure was assessed based on the location of their apartment in relation to the transformer room. AD patients were identified from Drug Purchase Register and Drug Reimbursement Register. Out of the 155,562 individuals, 5652 (111,357 person-years of follow-up) living in apartments next to transformer stations were considered as exposed, while 115,772 (2,289,526 person-years of follow-up) individuals living on higher floors of the same buildings were considered as referents. Associations between MF exposure and AD were examined using Cox proportional hazard models.

Results

The hazard ratio (HR) was 1.02 (95% confidence interval: 0.85–1.22), indicating that the risk of AD is not associated with residential ELF MFs present in apartments next to transformer stations. The duration of residence did not essentially change the HR. The risk of AD was slightly but not statistically significantly higher (HR 1.22, 95% confidence interval: 0.94–1.57) for those whose residence started before the age of 50 years.

Conclusion

The results did not support positive findings from previous studies that have reported a link between AD and occupational or residential MF exposure.

3. Occupational exposure

3.1 Occupational Exposure to Extremely Low-Frequency Magnetic Fields and Postmenopausal Breast Cancer Risk

Moayedi-Nia, S., Almadin, C., Labrèche, F., Goldberg, M.S., Richardson, L., Cardis, E., Ho, V. (2025). Journal of Occupational and Environmental Medicine, 68(2): e163.
<https://doi.org/10.1097/JOM.0000000000003564>

Background and objective

Breast cancer is the most commonly diagnosed cancer in the world and the most common cause of cancer mortality among women.¹ In 2020, an estimated 2.26 million new cases were diagnosed, and almost 685,000 deaths were attributed to breast cancer. The objective of this study is to estimate the association between occupational exposures to extremely low-frequency magnetic fields (ELF-MF) and postmenopausal breast cancer.

Methods

Lifetime job histories from a population-based case-control study (2008 to 2011) of histologically confirmed breast cancer in Montréal, Canada, were linked to a job-exposure matrix to assign geometric mean ELF-MF exposure/workday. Logistic regression estimated odds ratios and 95% confidence intervals for cumulative, average, maximum, and duration of maximum exposure to ELF-MFs (per interquartile range increase), adjusting for individual-level and ecological covariates.

Results

Data from 663 cases and 592 controls revealed no association between occupational ELF-MF exposure and postmenopausal breast cancer, though restricting exposures to 0 to 10 years before interview and to those during breast development, some positive associations were observed, particularly for ER+/PR+ tumors.

Conclusions

These findings suggest no association between occupational ELF-MF exposure and postmenopausal breast cancer risk.

4. Exposure Assessment

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5. Leukaemia Studies

5.1 Retrospective study on the association between paternal occupational exposure to agrochemicals and childhood leukemia in Michoacán de Ocampo, México

Jiménez-Alcántar, P., Gómez-García, A., López-Meza, J.E., Ochoa-Zarzosa, A., Espino-Barajas L.A., Morales-Manilla, L.M., Pérez-Rivera, E., Zúñiga-Quijano, L.Y., Gutiérrez-Castellanos, S. (2025). PeerJ, 13:e20219. <http://doi.org/10.7717/peerj.20219>

Background and objective

The constant use of agrochemicals in avocado plantations, because of their susceptibility to pests and diseases, continuously exposes those who work or reside near these orchards to health risks. The purpose of this study was to investigate the association between childhood leukemia cases in Michoacán, paternal occupational exposure to agrochemicals, and environmental exposure due to residential proximity.

Methods.

A retrospective observational cohort study was performed. The authors analyzed 430 cases of leukemia in children under 18 years of age diagnosed between 2010-2023. Logistic regression models were used to estimate odds ratios and 95% confidence intervals, adjusted for sociodemographic factors. Survival was analyzed using Kaplan-Meier curves.

Results

A total of 46.6% of the parents of children with leukemia in this study had jobs related to the use of agrochemicals (e.g., day laborers, peasants, farmers). Additionally, 65.4% of the leukemia cases occurred in municipalities producing avocado, the most important perennial crop in the state. Regarding the residential area analysis (mapping), many cases were found in contiguous zip codes and in areas densely occupied by avocado orchards. In addition, paternal occupations related to agrochemical use were associated with the avocado-growing zone, with an OR = 1.764 (95% CI [1.034–

3.009], $p = 0.0379$). Interestingly, survival associated with agrochemical-related occupations has a higher mean survival (139.3 months) than all other parental occupations ($p = 0.0148$).

Conclusion

The epidemiologic evidence found in this study supports the association between paternal occupational exposure to agrochemicals and childhood leukemia.

5.2 A global systematic evaluation of the impact of air pollution on pediatric cancer development

Khoshakhlagh, A.H., Ghobakhloo, S., Ghantous, A., Carlsen, L. (2025). Ecotoxicology and Environmental Safety 305: e119231. <https://doi.org/10.1016/j.ecoenv.2025.119231>

Background and objective

Increasing evidence links childhood cancers to environmental pollutants, especially air pollutants. This systematic review examined the relationship between various environmental pollutants and the risk of childhood cancers during the prenatal and pediatric periods.

Methods

A systematic search of Scopus, Web of Science, PubMed, Embase, Medline, Epistemonikos, and Cochrane databases using standard keywords identified 41,858 studies, of which 89 were ultimately included in the final analysis.

Results

The trend in studies from 1984 to 2025 indicates a growing focus on specific pollutants, including PM2.5, volatile organic compounds (VOCs), and benzene. Some studies reported statistically significant odds ratios (ORs) linking exposure to VOCs with the development of leukemia and other childhood cancers. Traffic sources were identified as the primary contributors to pollution associated with pediatric cancers, followed by industrial and indoor sources. Although data on prenatal exposures were sometimes sparse and inconclusive, there is more substantial evidence linking childhood exposures, particularly in urban and industrial areas, to the development of childhood cancers; however, the strength of this association varies depending on the type of pollutant and cancer. Geographically, the majority of studies have been conducted in high-income countries, which limits the generalizability of the findings to low- and middle-income regions.

Conclusion

These findings underscore the need for targeted public health policies to mitigate pollutant emissions and safeguard children, particularly in urban and industrial areas. Future studies should prioritize

precise individual exposure assessments and robust methodologies to elucidate causal pathways and enhance prevention strategies.

5.3 Impact of the environment on childhood cancer clinical outcomes

Metayer, C. (2025). Current Problems in Pediatric and Adolescent Health Care, 55: e101801.
<https://doi.org/10.1016/j.cppeds.2025.101801>

Background and objective

Incidence rates of several childhood cancers have increased in the past decades in high- and middle-income countries, and epidemiologic studies worldwide have documented the important role of several environmental factors contributing to these trends, such as sources of air pollution and pesticides. What is less known is whether these chemicals also affect clinical outcomes in childhood cancer patients and survivors, via biological pathways involving DNA damage, epigenetic changes, oxidative stress, pro-inflammatory and immune responses which may operate across the cancer continuum from etiology, response to treatment, survival and survivorship. Despite overall improvement in cancer cure rates, children of color and underserved backgrounds still experience poorer prognosis than their white and affluent counterparts. While social determinants largely explain these observations, the independent effect of chemical exposures is not well-characterized.

Methods

Drawing from adult studies, the authors present emerging evidence from epidemiologic childhood cancer studies on the impact of pre- and post-natal exposures to ubiquitous chemicals such as tobacco smoke, traffic-and industry-related air pollution, and pesticides on the health of childhood cancer patients and survivors.

Results and conclusion

Data presented in this review provide the foundation to consider integrating environmental health into the treatment plan for childhood cancer patients and survivors, alongside other known therapeutic and modifiable prognostic factors.

5.4 Association between exposure to PM2.5 and black carbon and the risk of childhood leukemia in Tehran: A case-control study with critical exposure time windows

Norzaee, S., Kermani, M. et al. (2025). Environmental Research, 287: e123168.
<https://doi.org/10.1016/j.envres.2025.123168>

Background and objective

Limited research has explored the relationship between air pollutants and childhood leukemia during critical exposure periods, and no such research has been conducted in Tehran to date. This study assessed the association between exposure to fine particulate matter and black carbon and the risk of childhood leukemia, focusing on various postnatal exposure windows.

Methods

This case-control study involved 428 children aged 1–15 years diagnosed with leukemia in Tehran. Annual concentrations of PM2.5 and BC were estimated using the Gradient Boosting Machine (GBM) algorithm, integrating ground-based measurements with meteorological data and satellite-derived Aerosol Optical Depth (AOD). The relationship between pollutant exposure and the probability of developing leukemia was investigated using logistic regression models with 95 % confidence intervals. Three exposure periods were considered: the entire exposure period from birth to diagnosis, the year prior to the diagnosis, and the age range corresponding to when children were aged between two and four years. Additionally, a logistic regression model with cubic splines was used to allow for nonlinear associations.

Results

The analysis revealed that a 5.09 $\mu\text{g}/\text{m}^3$ increase in PM2.5 concentration (interquartile range (IQR)) was associated with an increased odds ratio for childhood leukemia of 1.10 (95 % CI: 1.05–1.15). Similarly, for each 1 $\mu\text{g}/\text{m}^3$ increase in BC, the OR was 1.14 (95 % CI: 0.99–2.20). The findings showed that with each increase of IQR in the PM2.5 concentration in the year before diagnosis, the OR was 1.12 (95 % CI: 1.01–1.11) for childhood leukemia, while for BC, the OR was 1.16 (95 % CI: 1.00–2.15). Cubic spline analysis suggested a potentially non-linear association between PM2.5 and BC concentrations and the risk of childhood leukemia, with an increasing trend observed at higher exposure levels.

Conclusion

These results demonstrate a significant relationship between the incidence of leukemia in children and residential exposures to either PM2.5 or BC.

5.5 Prenatal and postnatal exposure to traffic-related air pollution (TRAP) and childhood cancer: Systematic review and meta-analysis

Oh, J., Shah, S. et al. (2025). Environmental Research, 292: e123646.

<https://doi.org/10.1016/j.envres.2025.123646>

Background and objective

Previous studies have suggested a possible link between traffic-related air pollution (TRAP) and the risk of childhood cancers, though findings remain inconsistent. This systematic review and meta-analysis aimed at evaluating the association between TRAP exposure and childhood cancer risk,

focusing on prenatal and postnatal exposure to fine particulate matter (PM2.5), nitrogen dioxide (NO₂), and benzene.

Methods

Peer-reviewed manuscripts were identified through PubMed (n = 1,116), Web of Science (n = 578), and Cochrane Library (n = 163) databases through June 21, 2024. Five reviewers independently screened titles, abstracts, and full texts for eligibility. A meta-analysis was conducted, with subgroup analyses based on exposure period (prenatal vs. postnatal) and age group. Results are presented as odds ratios (ORs) and 95 % confidence intervals (CIs) per 10 µg/m³ increase in PM2.5 and NO₂ exposure, and per 1 µg/m³ increase in benzene exposure.

Results

Of 1,632 studies screened after duplicate removal, 25 met the inclusion criteria. The findings indicated an elevated risk of acute lymphoblastic leukemia (ALL) in children exposed to PM2.5 (OR: 1.29, 95 % CI: 1.01–1.63; 5 studies; I² = 72.1 %), and increased risks of all childhood cancers (OR: 1.12, 95 % CI: 1.02–1.22; 4 studies; I² = 0.0 %) and acute myeloid leukemia (AML, OR: 1.22, 95 % CI: 1.02–1.46; 4 studies; I² = 0.0 %) associated with benzene exposure. PM2.5 exposure was also associated with a higher risk of retinoblastoma (OR: 1.68, 95 % CI: 1.16–2.43; 3 studies; I² = 0.0 %). Subgroup analyses revealed a stronger association between postnatal TRAP exposure (PM2.5 and NO₂) and leukemia risk compared to prenatal exposure.

Conclusion

This study provides evidence of a link between TRAP exposure and increased childhood cancer risk, particularly during the postnatal period. Further research is needed to confirm these findings.

5.6 The Determining of Pesticide Residue Levels in Children Diagnosed with Acute Leukemia in Cukurova Region, Turkiye

Yilmaz, S., Daglioglu, N., et al. (2025). Asian Pacific Journal of Cancer Prevention, 26 (10), 3699-3704. <https://doi.org/10.31557/APJCP.2025.26.10.3699>

Background and objective

Acute lymphoblastic leukemia (ALL) is the most common childhood malignancy. in children. Children can be exposed to pesticides during pregnancy or by their parent carried out pesticides to home. The objective of this study was to study pesticide residues in both acute leukemia patients and healthy children.

Methods

Twenty nine patients with acute leukemia [22 boys (76%) and 7 girls (24%)] and 33 healthy children [(19 boys (57%), 14 girls (43%)] were included in the study. We analyzed eight PCB (PCB 28, PCB 52, PCB 101, PCB 118, PCB 138, PCB 153, PCB 180 and PCB 202), HCB, three HCH isomers, 4,4'-DDT, 4,4'-DDD and 4,4'-DDE were investigated in bone marrow of newly diagnosed acute leukemia patients and peripheral blood samples of healthy children.

Results

The most detectable OCPs were β -HCH, δ -HCH and PCB28, triflumizole and thiometon were commonly found as OPPs. The significant difference was found statistically between ALL cases and control group according to the residues of PCB28 ($74,06 \pm 85,29$), β -HCH ($72,06 \pm 89,07$) and δ -HCH ($23,35 \pm 42,93$) ($p=0.001$). Additionally, OPPs consisted triflumizole ($7,23 \pm 9,17$), thiometon ($3,37 \pm 4,30$) and halfenprox ($1,90 \pm 3,22$) showed distinctive difference statistically ($p=0.001$).

Conclusion

Pesticide residues were estimated significantly in bone marrow of leukemia patients should be considered an important for public health with the decreasing or preventing of pesticide exposure which have a role in leukemia ethiology.

6. REFERENCES

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