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AGROCHEMICAL EXPOSURE AND PEDIATRIC LEUKEMIA: A NARRATIVE REVIEW

Paparan Bahan Agrokimia dan Leukimia pada Anak: Sebuah Tinjauan Naratif

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Abstract

Pediatric leukemia is the most common childhood cancer worldwide, with acute lymphoblastic leukemia (ALL) and acute myeloid leukemia (AML) representing the majority of cases. Although genetic susceptibility contributes to disease development, it explains a minority of cases, emphasizing the potential role of environmental exposures in the pathogenesis. Among these, agrochemicals, including pesticides, herbicides, and insecticides, are extensively used in agricultural and residential settings and have been widely investigated of their biological plausibility for carcinogenic effects through mechanisms such as genotoxicity, oxidative stress, endocrine disruption, and epigenetic alteration. This narrative review synthesizes evidence on the association between agrochemical exposure and pediatric leukemia from systematic reviews, meta-analyses, and observational studies. Overall, the literature consistently reports modest but positive associations between pesticide exposure and childhood leukemia, particularly ALL. Stronger associations are observed for maternal prenatal, residential pesticide use, and early-life exposure. Evidence also indicates increased risk associated with paternal occupational exposure near conception, maternal occupational exposure during pregnancy, indoor insecticide use, and certain classes of insecticides and herbicides. Residential proximity to agricultural areas has been linked to higher incidence, potentially reflecting pesticide drift. Despite consistent findings, limitations remain, including heterogeneity in exposure assessment, reliance on self-reported data, and predominance of observational designs. Future studies should incorporate prospective designs, biomarker-based exposure measurement, and improved geospatial modeling to strengthen causal inference, clarify risk magnitude, and inform preventive strategies.

Keywords: agrochemicals, pesticides, pediatric leukemia

Abstrak

Leukemia pediatrik merupakan kanker anak paling umum di seluruh dunia, dengan leukemia limfoblastik akut (ALL) dan leukemia mieloid akut (AML) mewakili mayoritas kasus. Meskipun kerentanan genetik berkontribusi terhadap perkembangan penyakit, namun faktor tersebut hanya menjelaskan sebagian kecil kasus, sehingga menekankan potensi peran paparan lingkungan dalam patogenesisnya. Berbagai bahan agrokimia, seperti pestisida, herbisida, dan insektisida, telah banyak diteliti terkait kemungkinan efek karsinogeniknya melalui mekanisme seperti genotoksisitas, stres oksidatif, gangguan endokrin, dan perubahan epigenetik. Tinjauan naratif ini mensintesis bukti mengenai hubungan antara paparan bahan agrokimia dan leukemia pediatrik berdasarkan *systematic review*, meta-analisis, dan studi observasional. Secara umum, literatur secara konsisten melaporkan hubungan positif antara paparan pestisida dan leukemia pada anak, khususnya ALL. Hubungan yang lebih kuat ditemukan pada paparan yang terjadi pada maternal prenatal, penggunaan pestisida di lingkungan rumah, dan paparan pada awal kehidupan. Bukti juga menunjukkan peningkatan risiko yang berkaitan dengan paparan pekerjaan ayah menjelang konsepsi, paparan pekerjaan ibu selama kehamilan, penggunaan insektisida di dalam ruangan, serta jenis insektisida dan herbisida tertentu. Kedekatan tempat tinggal dengan area pertanian juga dikaitkan dengan peningkatan insidensi, yang kemungkinan mencerminkan penyebaran pestisida melalui udara. Meskipun temuan cukup konsisten, masih terdapat beberapa keterbatasan, termasuk heterogenitas dalam penilaian paparan, ketergantungan pada data laporan mandiri, dan dominasi desain studi observasional. Penelitian di masa mendatang perlu mengintegrasikan desain prospektif, pengukuran paparan berbasis biomarker, serta pemodelan geospasial yang lebih baik untuk memperkuat inferensi kausal, memperjelas besarnya risiko, dan mendukung strategi pencegahan yang efektif.

Keywords: bahan agrokimia, pestisida, leukemia pediatrik

INTRODUCTION

Pediatric leukemia is the most common childhood malignancy worldwide, with acute lymphoblastic leukemia (ALL) accounting for the majority of cases (Perdana *et al*., 2020). Although survival rates have improved substantially, the etiology of most cases remains unclear, and environmental exposures during critical developmental windows are increasingly investigated as potential contributors (Mancini *et al*., 2026). Among these, agrochemicals including pesticides, herbicides, and insecticides are of particular interest due to their widespread use in agriculture and domestic settings and their potential for early-life exposure (Mancini *et al*., 2026).

Experimental evidence suggests that several agrochemical compounds may influence leukemogenesis through mechanisms such as genotoxicity, oxidative stress, endocrine disruption, and interference with hematopoietic development. These biological pathways are especially relevant during prenatal and early postnatal periods when rapidly dividing cells are more vulnerable to environmental insults (Hernández & Menéndez, 2016). Emerging evidence also indicates that certain pesticides may induce epigenetic alterations, including changes in DNA methylation and gene expression, which could contribute to leukemogenic process initiated during fetal development (Hernández & Menéndez, 2016).

Epidemiological studies, including multiple systematic reviews and meta-analyses, have consistently reported modest but positive associations between pesticide exposure and childhood leukemia, particularly with prenatal and residential exposure (Karalexi *et al*., 2021; Wang *et al*., 2019). Stronger associations have been observed for insecticides and maternal exposure during pregnancy, although variability in exposure assessment and study design limits causal interpretation (Ferri *et al*., 2018). Several meta-analyses have further

 Volume 18 No. 3: 614-625
June 2026
https://doi.org/10.24843/bulvet.2026.v18.i03.p13

demonstrated that the highest risk estimates are generally observed for prenatal exposure to residential insecticides, supporting the hypothesis that in utero exposure represents a critical window of susceptibility for leukemia development (Turner *et al*., 2010; Van Maele-Fabry *et al*., 2011).

Despite these limitations, the consistency of findings across studies and the biological plausibility of proposed mechanisms support continued investigation of agrochemical exposure as a potentially modifiable risk factor for pediatric leukemia. Nevertheless, important knowledge gaps remain regarding the relative contribution of different exposure sources and the critical periods of susceptibility during early life. This narrative review aimed to synthesize current evidence on this association, with emphasis on exposure timing, exposure pathways, and the strength of epidemiological evidence.

RESEARCH METHODS

This narrative literature review aimed to synthesize evidence on the association between agrochemical exposure and pediatric leukemia. A structured search was conducted in PubMed and ScienceDirect to identify relevant studies published between 2010 and 2026, reflecting contemporary developments in epidemiological methods and exposure assessment. Full text articles were obtained through open-access sources and academic library subscriptions. Search terms included combinations of keywords related to agrochemicals (including pesticides, herbicides, and insecticides), exposure pathways (prenatal, residential, and occupational exposure), and pediatric leukemia.

Studies were included if they: (1) investigated the association between agrochemical exposure and leukemia in children or adolescents; (2) were published in English; (3) were available in full text; and (4) employed observational epidemiological designs, including cohort, case-control, or cross-sectional studies, as well as systematic reviews and meta-analyses. Studies were excluded if they: (1) focused exclusively on adult leukemia; (2) did not assess agrochemical exposure; (3) were conference abstracts, editorials, letters, or commentaries; or (4) lacked sufficient relevance to the review topic.

Reference lists of relevant articles from systematic reviews and meta-analyses were manually screened to identify further eligible articles. Priority was given to higher levels of evidence, including systematic reviews, meta-analyses, cohort studies, and case-control studies. Due to heterogeneity in study designs, exposure measurements, populations, and outcome definitions, the findings were synthesized narratively rather than quantitatively pooled.

RESULT AND DISCUSSION

Result

The reviewed studies consistently demonstrated positive associations between agrochemical exposure and pediatric leukemia, particularly ALL (Table 1). Meta-analyses and pooled analyses reported increased leukemia risk associated with residential pesticide exposure during preconception, pregnancy, and childhood. Stronger associations were frequently observed for prenatal exposure to insecticides and herbicides.

Several case-control studies further identified elevated risks related to household pesticide use, indoor insecticides, and residential proximity to agricultural areas or pesticide-treated fields. Increased leukemia incidence was also reported among children with parental occupational exposure to pesticides, particularly in farming-related occupations. Biomarker-based studies supported these findings by demonstrating higher pesticide metabolite levels among children with leukemia.

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Pendidikan Tinggi, Riset, dan Teknologi No. 158/E/KPT/2021

Volume 18 No. 3: 614-625
June 2026
https://doi.org/10.24843/bulvet.2026.v18.i03.p13

Although a small number of studies reported non-significant associations, the overall evidence demonstrated a recurring pattern linking prenatal, residential, and occupational agrochemical exposure with increased risk of pediatric leukemia, especially ALL and, to a lesser extent, acute myeloid leukemia (AML).

Discussion

This narrative review highlights a consistent pattern of association between agrochemical exposure and pediatric leukemia, particularly ALL, across multiple epidemiological studies. Evidence from systematic reviews, meta-analyses, pooled analyses, and observational studies indicates that pesticide exposure during prenatal and early-life periods may contribute to elevated leukemia risk and leukemogenesis in children (Table 1) (Bailey *et al*., 2015; Karalexi *et al*., 2021; Turner *et al*., 2010; Van Maele-Fabry *et al*., 2011). Although the reported effect sizes varied between studies, the overall direction of association remained relatively consistent, especially for residential pesticide exposure, indoor insecticide use, and parental occupational exposure.

One of the most important findings across the literature is the critical role of prenatal exposure. Maternal exposure to pesticides during pregnancy was repeatedly associated with increased leukemia risk in offspring (Ferreira *et al*., 2013; Onyije *et al*., 2022; Turner *et al*., 2010; Wang *et al*., 2019). This observation is biologically plausible because fetal hematopoietic stem and progenitor cells undergo rapid proliferation and differentiation during embryonic development, making them highly susceptible to environmental toxicants. Several pesticides have been shown to induce oxidative stress, DNA damage, chromosomal translocations, and epigenetic dysregulation, which are mechanisms strongly implicated in leukemogenesis (Greaves, 2018). Experimental evidence further suggests that certain agrochemicals may inhibit topoisomerase II, an enzyme involved in DNA replication and repair, potentially contributing to chromosomal rearrangements frequently observed in childhood leukemia (Greaves, 2018; Karalexi *et al*., 2021).

Residential exposure emerged as another important pathway. Multiple studies reported increased leukemia risk associated with indoor insecticide use, household pesticide application, and living near agricultural areas (Bailey *et al*., 2015; Chen *et al*., 2015; Desai *et al*., 2025; Poynter *et al*., 2017; Ruth *et al*., 2023; Van Maele-Fabry *et al*., 2011, 2019). Children may experience chronic low-dose exposure through contaminated household dust, surfaces, inhalation, or dietary intake. Young children are particularly vulnerable because of their developing immune systems, higher metabolic rates, and frequent hand-to-mouth behaviors. Furthermore, studies examining residential proximity to crop fields and pesticide-treated land support concerns regarding pesticide drift and environmental contamination in surrounding communities (Cárceles-Álvarez *et al*., 2017; Ding *et al*., 2012; Malagoli *et al*., 2016; Mancini *et al*., 2026; Yilmaz *et al*., 2025).

Parental occupational exposure was also consistently associated with elevated leukemia risk. Studies involving agricultural workers, pesticide applicators, and farming populations demonstrated increased risk among offspring, particularly following paternal exposure around conception or maternal exposure during pregnancy (Coste *et al*., 2020; Gunier *et al*., 2017; Maryam *et al*., 2015; Poynter *et al*., 2017). Occupational exposure may indirectly affect children through contamination of clothing, equipment, or household environments. In addition, preconception exposure may contribute to germ cell damage and genetic alterations that increase susceptibility to leukemia in offspring.

Despite the consistency of epidemiological findings, several methodological limitations should be considered. Most available evidence is derived from retrospective case-control studies,

Terakreditasi Nasional Sinta 4, berdasarkan Keputusan Direktur Jenderal
Pendidikan Tinggi, Riset, dan Teknologi No. 158/E/KPT/2021

Volume 18 No. 3: 614-625
June 2026
https://doi.org/10.24843/bulvet.2026.v18.i03.p13

which are susceptible to recall bias and selection bias. Many studies relied on self-reported pesticide exposure rather than objective biomarker measurements, increasing the possibility of exposure misclassification. Additionally, pesticide exposure is often categorized broadly despite considerable heterogeneity in chemical composition, toxicity, and mechanism of action among individual compounds (Karalexi *et al*., 2021; Van Maele-Fabry *et al*., 2011). This limitation makes it difficult to identify which specific pesticides are most strongly associated with leukemia risk

Another important challenge is the substantial heterogeneity across studies in exposure assessment methods, study populations, timing of exposure, and leukemia subtype classification. Differences in agricultural practices, regulatory policies, and environmental conditions between countries may also contribute to variability in reported associations. Furthermore, potential confounding factors such as socioeconomic status, parental smoking, air pollution, dietary exposures, and genetic susceptibility were not consistently controlled across studies. Consequently, while the evidence strongly suggests an association, definitive causal relationships cannot yet be fully established.

Nevertheless, the convergence of epidemiological evidence and biological plausibility strengthens concern that agrochemical exposure may contribute to pediatric leukemogenesis. The repeated observation of elevated risk across diverse geographic regions and study designs suggests that pesticide exposure represents a potentially important and modifiable environmental risk factor for childhood leukemia (Bailey *et al*., 2015; Karalexi *et al*., 2021; Turner *et al*., 2010).

CONCLUSIONS AND SUGGESTIONS

Conclusions

Current evidence supports a potential association between agrochemical exposure and increased risk of pediatric leukemia, particularly ALL. Prenatal exposure, residential pesticide use, and parental occupational exposure were the most consistently identified risk factors.

Suggestions

Future studies should prioritize prospective cohort designs, biomarker-based exposure assessment, and evaluation of specific pesticide compounds to improve causal inference. Strengthening exposure characterization and integrating genetic and environmental data may help identify vulnerable populations and support preventive public health strategies aimed at reducing pesticide exposure during critical developmental periods.

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 Volume 18 No. 3: 614-625
June 2026
https://doi.org/10.24843/bulvet.2026.v18.i03.p13

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Volume 18 No. 3: 614-625
June 2026
https://doi.org/10.24843/bulvet.2026.v18.i03.p13

Table

Table 1. Epidemiological evidence linking agrochemical exposure to pediatric leukemia

Study Type	Exposure type	Exposure Window	Leukemia type	Geographic Location	Risk Estimate (OR/HR)	Significant Findings	References
Case-control study	Proximity to crop areas (Barley/Viticulture)	Year preceding inclusion	ALL	Mainland France	OR: 1.05 (1.00–1.10) per 3% density increase	Barley and viticulture densities within 1000m were positively associated with ALL risk.	Mancini *et al*. (2026)
Survival/Cohort study	Residential rodenticides	Prenatal	ALL Survival	California, USA	HR: 1.91 (95% CI: 1.15–3.16)	Exposure to rodenticides during pregnancy was significantly associated with reduced 5-year survival (increased mortality risk).	Desai *et al*. (2025)
Case-control study	Termite insecticides (Household)	Prenatal	ALL	USA	aOR: 4.21 (95% CI: 2.00–8.88)	Exposure to termite insecticides during pregnancy was linked to the highest risk for childhood ALL.	Ruth *et al*. (2023)
Meta-analysis synthesis	General pesticides	Preconception and Prenatal	ALL	International (High-income countries)	RR varies (Strong level of evidence)	Convincing evidence of an association between general pesticide exposure during pregnancy and childhood ALL.	Onyije *et al*. (2022)
Meta-analysis	Occupational and environmental pesticide exposure	Prenatal and occupational exposure	ALL		2.51 95%CI: 1.39–4.55	Occupational exposure consistently associated with elevated risk	Karalexi *et al*. (2021)
Census-based	Parental occupational exposure	Not in source	Non-CNS solid tumors	Switzerland	Father HR: 1.84 (1.31–2.58);	Significant increased risk for non-CNS solid tumors, specifically bone tumors and soft tissue sarcomas.	Coste *et al*. (2020)

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Pendidikan Tinggi, Riset, dan Teknologi No. 158/E/KPT/2021

Volume 18 No. 3: 614-625
June 2026
https://doi.org/10.24843/bulvet.2026.v18.i03.p13

cohort study					Mother HR: 1.79 (1.13–2.84)		
Case-control study	Pesticides	Prenatal	ALL	Henan Province, China	Adjusted OR: 1.48 (95% CI: 1.67–2.28)	Maternal prenatal exposure significantly increased ALL risk; effects more pronounced in children < 5 years.	Wang *et al*. (2019)
Systematic review and meta-analysis	Residential insecticides	Prenatal	AML	Global (USA, Canada, Europe, Brazil, Asia, etc.)	OR: 1.90 (95% CI: 1.35–2.67)	The highest increased risks in this meta-analysis were observed for AML after prenatal indoor insecticide exposure.	Van Maele-Fabry *et al*. (2019)
Hospital-based case-control study	Maternal insecticides/rodenticides and field proximity (<100m)	Prenatal	Childhood Leukemia	Apulia region, Southern Italy	Proximity OR: 3.21 (95% CI: 1.37–7.53)	Increased risk linked to maternal prenatal use and living < 100m from treated fields.	Ferri *et al*. (2018)
Case-control study	Paternal occupational exposure	Perinatal	ALL	California, USA	General OR: 1.7 (95% CI: 1.2, 2.5);	Increased risk with paternal occupational exposure, particularly for nut crops.	Gunier *et al*. (2017)
Case-control study	Occupational / Residential Pesticides	Long-term (Adult exposure)	AML	Minnesota, USA	Not significantly associated	Pesticides were not significantly associated with AML or MDS in this specific adult/adolescent population.	Poynter *et al*. (2017)
Spatial cluster and case-series analysis	Community field fumigation	Prenatal	Acute Lymphoblastic Leukemia	Cieza, Murcia (Spain)	Relative Risk: 7.38	Detected a significant spatial cluster using pregnancy addresses; 62.5% of cases in cluster reported pesticide exposure.	Cárceles-Álvarez *et al*. (2017)

Volume 18 No. 3: 614-625
June 2026
https://doi.org/10.24843/bulvet.2026.v18.i03.p13

Population-based case-control study	Arable crop proximity (2,4-D, Glyphosate)	Time of diagnosis	Childhood Leukemia	Northern Italy	OR: 2.04 (95% CI: 0.50–8.35); Age < 5 OR: 5.76 (95% CI: 0.78–42.70)	Increased risk among children residing very close to arable crops, specifically those < 5 years old.	Malagoli *et al*. (2016)
Pooled analysis of case-control studies	Home pesticide exposure	Preconception, Prenatal Postnatal	ALL	International (North America, Europe, Australasia)	Preconception OR: 1.39 (95% CI: 1.25, 1.55); Pregnancy OR: 1.43 (95% CI: 1.32, 1.54); Postnatal OR: 1.36 (95% CI: 1.23, 1.51)	Statistically significant increased risk of ALL associated with pesticide exposure during all three windows (preconception, prenatal, and postnatal).	Bailey *et al*., 2015
Meta-analysis	Indoor residential insecticides and herbicides	Postnatal	Childhood Leukemia	Global (USA, Canada, Germany, etc.)	Leukemia OR: 1.47 (95% CI: 1.26–1.72); Herbicides OR: 1.26 (95% CI: 1.10–1.44)	Significant association with indoor insecticides and herbicides; no significant association found for outdoor insecticides.	Chen *et al*., 2015
Case-control study	Paternal occupational exposure	Lifetime / History of exposure	Acute Leukemia	Fars Iran	Farmers OR: 14.7 (95% CI: 5.6, 38.4)	Significant increased risk for farmers and their children	Maryam *et al*., 2015
Case-control study	Pyrethroid pesticides (DAP metabolites)	Postnatal (Birth to diagnosis)	ALL	Shanghai, China	OR: 9.1 (95% CI: 4.1–20.5)	Significant association between high urinary levels of pyrethroid metabolites and ALL.	Ding *et al*., 2012

Volume 18 No. 3: 614-625
June 2026
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Systematic review and meta-analysis	Residential / Household pesticides	Pregnancy and Postnatal	Childhood Leukemia and unspecified	Global (USA, Canada, Europe, Brazil, Asia, etc.)	mRR: 1.74 (95% CI: 1.37–2.21); SOR: 1.57 (95% CI: 1.27–1.95)	Statistically significant associations observed. Strongest risks for prenatal indoor exposure and for AML among children aged $\leq$ 2 years.	Van Maele-Fabry *et al.*, 2011
Systematic review and meta-analysis	Residential insecticides, herbicides, and unspecified pesticides	Pregnancy (Prenatal)	Childhood Leukemia	International (USA, Canada, Mexico, Japan, France, Brazil, Germany)	Insecticides OR: 2.05 (95% CI: 1.80–2.32); Herbicides OR: 1.61 (95% CI: 1.20–2.16); Unspecified OR: 1.54 (95% CI: 1.13–2.11)	Strong significant association observed for insecticides, herbicides, and unspecified pesticides during pregnancy.	Turner *et al.*, 2010

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Figure

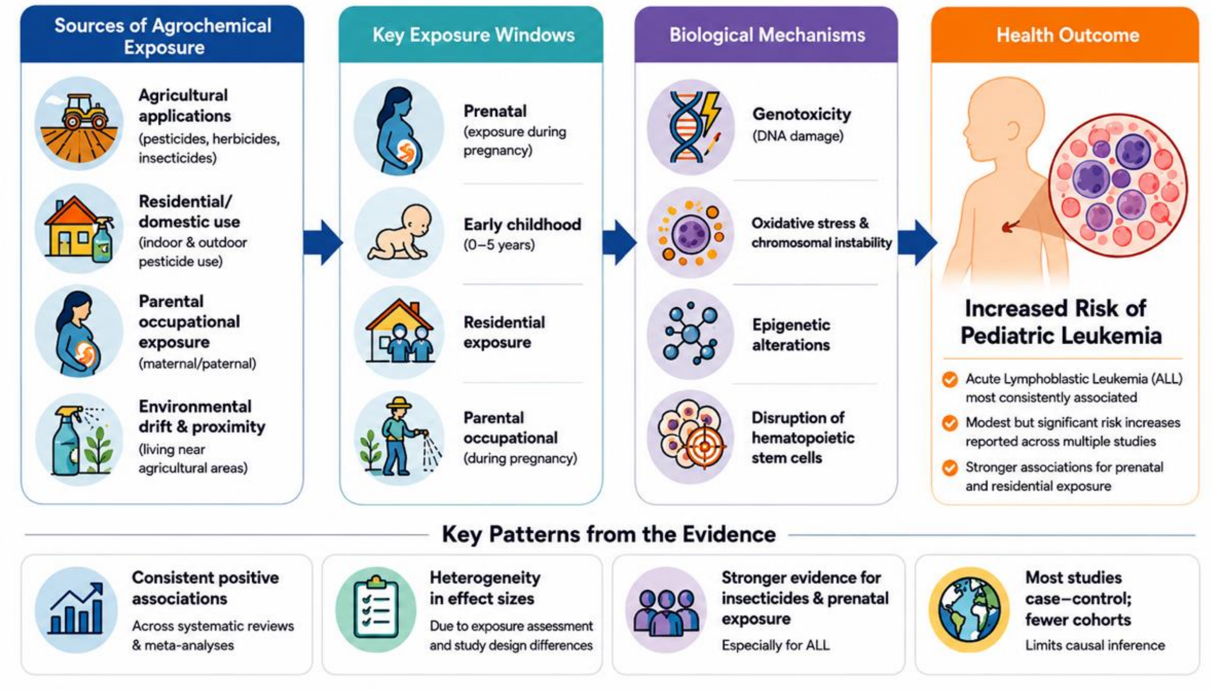

Figure 1**.** Proposed mechanisms linking agrochemical exposure to pediatric leukemia