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Pesticide Exposures during Pregnancy and Health Outcomes in Latin America and the Caribbean: A Scoping Review of Human Observational Studies

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Background: Latin America and the Caribbean (LAC) are regions with intense pesticide use. Numerous studies have demonstrated the adverse health effects associated with pesticide exposure. The embryonic and fetal periods are particularly susceptible to xenobiotics, with pesticides exhibiting potentially teratogenic effects.

Objectives: The objective was to review the scientific literature on outcomes associated with prenatal pesticide exposure, identifying challenges and gaps in this field.

Methods: We conducted a scoping review using terms related to pesticides, LAC, and pregnancy across six databases. The final search was conducted on 5 March 2024. The inclusion criteria for the studies were as follows: *a*) being human observational studies involving pregnant women of any age or gestational age duration, newborns from these pregnancies, or both; *b*) reporting any exposure to pesticide and any adverse outcome; *c*) having been conducted in any country of LAC; *d*) having been published between 1 January 2000 and 5 March 2024; *e*) having the full text available in English, Spanish, or Portuguese; and *f*) presenting original results. Nonoriginal research papers, such as qualitative studies, reviews, critical analyses, and opinion papers, were excluded. The included studies were categorized and presented based on the outcomes they evaluated.

Results: We included 80 studies conducted in 13 countries; the included studies encompassed obstetric outcomes, anthropometric parameters, congenital anomalies, neurodevelopment, respiratory infections, and childhood leukemia, as well as molecular effects. Organochlorines were the primary type of exposure investigated among the included studies. Many studies relied on indirect measures of pesticide exposure.

Discussion: Adverse outcomes associated with prenatal pesticide exposure have been observed in Latin American and Caribbean populations, consistent with the global literature. Significant knowledge gaps remain, especially regarding groups of pesticides other than organochlorines. Less than half of the countries in LAC have conducted any study on the potential effects of prenatal exposure. Ongoing research into the risks of prenatal exposure is imperative. It is essential to consider the region's unique characteristics, particularly when investigating the risks associated with pesticides authorized exclusively in this region. https://doi.org/10.1289/JHP1043

Introduction

Latin America and the Caribbean (LAC) are home to 660 million inhabitants and encompass a land surface of 2 billion hectares, of which 38% is dedicated to agriculture.[1,2] The countries within LAC are major exporters of soybeans, pork, corn, poultry, animal feed, sugar, coffee, fruits, and vegetables.[2] Brazil, Argentina, and Mexico are this region's largest agricultural and food exporters.[2] Substantial agricultural growth has been observed over the past five decades.[3] Most of this growth has resulted from productivity improvements rather than expanding the agricultural land under cultivation.[4] At the same time, there has been an increase in the production and use of pesticides over the last two decades.[4–6] A pesticide is defined as any substance, mixture of substances, or microorganisms intended for repelling, destroying, or controlling any pest.[7] This includes vectors of human or animal disease and unwanted species of plants or animals that cause harm or otherwise interfere with the production, processing, storage, transport, or marketing of food, agricultural commodities, wood, and wood products or animal feed.

Since the 1960s, the scientific community has closely examined the environmental impact of pesticides. Evidence has shown that pesticides can persist in soil and water, accumulate in invertebrate and vertebrate tissues, traverse food chains, and impact apex predators.[8] An extensive body of literature examines the link between pesticide exposure and detrimental effects on human health, including type 2 diabetes,[9] neurological disorders,[10] cancer,[11,12] respiratory diseases,[13] and allergies.[14] Given the teratogenic potential of xenobiotics for pregnant women, the impact of pesticides on this population has received considerable attention. Pesticide exposure during pregnancy has been associated with an increased risk of childhood leukemia.[15] In addition, potential links to clinical manifestations of autism spectrum disorder have been explored,[16] whereas findings regarding association with obesity remain inconclusive.[17] Furthermore, there is a lack of consistency among the studies evaluating the association between maternal pesticide exposure and obstetric and neonatal outcomes.[18,19] Moreover, a substantial number of studies assess the effects of prenatal pesticide exposure on congenital anomalies.[20–32]

A recent scoping review investigated the current state of research on the health effects of pesticide exposure in populations from LAC.[33] That review underscored the potential health impacts of pesticide exposure, gathering findings pertaining to genotoxicity, neurobehavioral effects, cancer risks, reproduction, placental outcomes and teratogenicity, birth and child outcomes, and other effects. Moreover, it delineated significant methodological limitations within the scientific output on this subject. To address these limitations and research gaps within LAC, the

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authors advocated for increased investment in research and development, collaborative initiatives within the region, and enhanced methodological rigor in epidemiological studies. The aforementioned review has provided a comprehensive depiction of the research landscape, showing substantial heterogeneity among the included studies concerning exposure and outcomes. Considering the susceptibility of embryo and fetus development to xenobiotic agents throughout gestation and the adverse effects that exposure can induce, we conducted a scoping review further focused on prenatal pesticide exposure and adverse outcomes. In addition, our review expands the search to six databases, including one dedicated to studies specific to the LAC region, and extends the time frame to encompass publications on the theme over more than the last two decades. Our aim is to identify the main challenges and research gaps in pregnancy-related pesticide exposure and outcomes within LAC and offer insights for future studies.

Methods

The present study was conducted in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses extension for Scoping Reviews (PRISMA-ScR).[34] A protocol was prepared prior to the identification step and this protocol has been registered within the Open Science Framework.[35]

The selection of terms for the search strategy was based on the Population, Concept, and Context (PCC) framework,[36] and this search strategy was expanded to include related terms according to the Medical Subject Headings (MeSH) terms. The search strategy protocol, including all selected keywords and index terms, was adapted for each database or information source (see "Search Terms," in the Supplemental Material). The development of the full search strategy protocol was facilitated by the assistance of a research librarian.

The following databases were searched to identify potentially relevant studies: Embase, PubMed, Scielo, Scopus, Virtual Health Library, and Web of Science. The first search was conducted on 14 November 2022. The final search was conducted on 5 March 2024. The inclusion criteria for the studies were as follows: *a*) being human observational studies involving pregnant women of any age or gestational age duration, newborns from these pregnancies, or both (population); *b*) reporting any exposure to pesticide and any adverse outcome (concept); *c*) having been conducted in any country of LAC (context), as the grouping of countries presented by the OECD-FAO document[2]; *d*) having been published between 1 January 2000, and 5 March 2024; *e*) having the full text available in English, Spanish, or Portuguese; and *f*) presenting original results. Nonoriginal research papers, such as qualitative studies, reviews, critical analyses, and opinion papers, were excluded. To capture the scientific literature concerning prenatal exposure to pesticides and its associated adverse outcomes, articles that did not gather data on exposure measured within the context of pregnancy were excluded. This decision was made because the target population of the study should consist of pregnant women, their newborns, or both. In addition, articles that did not directly assess adverse outcomes, such as those focusing on enzyme activity levels, were excluded.

All identified articles were collected and uploaded to Rayyan,[37] a web application that provides tools for conducting the initial screening of abstracts and titles during the article selection process. We used Rayyan's tool to identify duplicate articles and then manually reviewed each possible duplicate identified, excluding those articles that were duplicate records. Subsequently, we conducted a test of titles and abstracts screening 20 articles, followed by a discussion to ensure consistent understanding of relevance among the reviewers. Finally, we screened the titles and abstracts of all articles. Each article was screened by two reviewers. Relevant studies were imported into Mendeley.[38] The full text of each selected paper was then thoroughly assessed by the independent reviewers to further ensure compliance with the study's inclusion criteria. Finally, the reviewers recorded the reasons why the excluded papers did not meet inclusion criteria.

Data extraction from the selected studies was performed by two independent reviewers using a standardized spreadsheet that included specific details about each study's participants, concept, context, methods, and key findings relevant to the present study's review questions. Disagreements between the reviewers at any stage of the selection process or data extraction were resolved by an additional reviewer or discussions between reviewers.

The selected studies were organized into tables based on their outcomes and included descriptions of the author's name, year, exposure, outcomes, and key findings. In each table, studies were grouped by country of origin, with studies within each group presented chronologically by of year of publication. Finally, a narrative summary was performed.

Results

The search across the six databases yielded 1,765 records in the first search and an additional 165 records were returned in the final search, resulting in a total of 1,930 records. After removing duplicates, 1,021 articles were then screened by examining titles and abstracts (see the screening step in Figure 1). From this screening, 853 articles were excluded, and we kept 168 articles for further analysis. In the next step, the full-text assessment, 88 more articles were excluded, resulting in 80 human studies being included in this scoping review.

We identified studies from 13 distinct countries: Mexico ($n = 26$), Brazil ($n = 14$), Guadeloupe ($n = 13$), Argentina ($n = 6$), Costa Rica ($n = 7$), Ecuador ($n = 4$), Puerto Rico ($n = 2$), Colombia ($n = 2$), Chile ($n = 2$), Bolivia ($n = 1$), Paraguay ($n = 1$), Peru ($n = 1$), and Guatemala ($n = 1$) (Figure 2A). It is worth noting that we identified several articles originating from the same study population. Notably, the Costa Rican Childhood Leukemia Study (CRCLS) contributed 2 articles.[39,40] Similarly, The Infants' Environmental Health (ISA) study from Costa Rica gave rise to 5 articles.[41–45] The EcoSalud Project in Ecuador produced 2 articles,[46,47] and there was a pilot study and a subsequently another study conducted in the town of Tabacundo, Ecuador, resulting in 2 articles.[48,49] Among the articles from Brazil, 2 evaluated data from the Multicentric Study of Infant Leukemia.[50,51] In addition, we included 13 articles that originated from data analysis of the prospective mother–child cohort TIMOUN study conducted in Guadeloupe.[52–64] In the case of Mexico, several groups of articles originated from the same study: Seven articles came from the prospective perinatal cohort study in the State of Morelos,[65–71] 3 articles came from the Early Life Exposures in Mexico to Environmental Toxicants (ELEMENT) study,[72–74] and another 3 articles originated from the study conducted in Tapachula, State of Chiapas.[75–77] The predominant study designs among the articles included were cohort ($n = 37$, 46.2%), cross-sectional ($n = 18$, 22.5%), and case–control ($n = 17$, 21.2%) (Figure 2B). Most studies evaluated pesticide exposure directly using levels measured in biological samples, with maternal blood ($n = 19$, 23.7%), cord blood ($n = 13$, 16.2%) and maternal urine ($n = 10$, 12.5%) being the most common biological matrixes (Figure 2C). A notable portion of studies indirectly assessed pesticide exposure through questionnaires ($n = 27$, 33.8%), surveying the participants about proximity of residences to pesticide applications sites, occupational exposure, or domestic use. The articles were categorized into five groups based on their respective outcomes: *a*) adverse obstetric outcomes ($n = 20$, 25.0%), *b*) congenital anomalies ($n = 21$, 26.3%), *c*) anthropometric measures and sex ratio ($n = 18$,

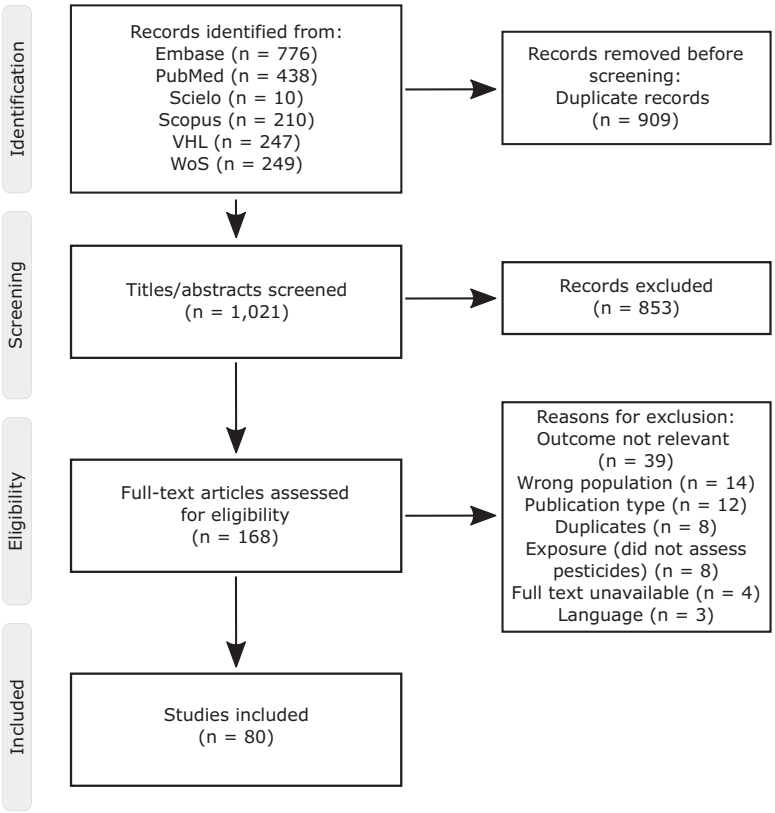

Figure 1. Flow diagram of the study selection process. Note: Embase, Excerpta Medica Database; Scielo, Scientific Electronic Library Online; VHL, Virtual Health Library; WoS, Web of Science.

22.5%), *d*) neurodevelopment and other outcomes throughout childhood ($n = 29$, 36.3%), and *e*) molecular effects ($n = 6$, 7.5%) (Figure 2D). We provide a summary of the findings from these articles in the subsequent sections, organized based on these outcome categories.

Obstetric Outcomes

Twenty studies explored the association between prenatal pesticide exposure and obstetric outcomes (Table 1). These studies include three sets of outcomes: premature birth, intrauterine growth restriction, and spontaneous abortion.

Premature birth. Thirteen studies examined the impact of pesticide exposure on preterm birth and gestational length. Among these, 6 did not yield statistically significant findings.[44,79,80,82,88,93] The study by Wendel de Joode et al.[44] found no association between 3,5,6-trichloro-2-pyridinol (TCPY), 3-(2,2-dichlorovinyl)-2,2-dimethylcyclopropanecarboxylic acid (DCCA), 3-phenoxybenzoic acid (3-PBA), 2,4-dichlorophenoxyacetic acid (2,4-D), ethylenethiourea (ETU), hydroxy pyrimethanil (OHP), and length of gestation, even when stratifying levels in the first and second half of gestation. In the study by Rodriguez et al.,[80] a similar average gestational age at birth was observed when comparing pregnant women from urban and rural regions. However, differences in the concentration levels of chlorpyrifos were observed between the two groups. In the study by Souza et al.,[78] the average gestational age at birth in an Argentinian region of intense use of pesticides decreased by 2 wk during the spraying period. An ecological study with data from the Brazilian Live Birth Information System (SINASC) showed that regions with higher levels of per capita pesticide consumption presented a higher prevalence of births before 22 wk of gestation ($p < 0.001$).[83] A similar approach showed that the prevalence ratio (PR) of prematurity was associated with per capita fungicide [PR = 1.23; 95% confidence interval (CI): 1.19, 1.28], acaricide (PR = 1.08; 95% CI: 1.05, 1.12), herbicide (PR = 1.24; 95% CI: 1.20, 1.28), and insecticide (PR = 1.25; 95% CI: 1.21, 1.30) use.[84] Other studies have examined the effects of specific pesticides on premature birth. In the TIMOUN Mother–Child Cohort Study conducted in Guadeloupe, a 1-$\log_{10}$ increase in chlordecone concentration was significantly associated with a reduction in the length of gestation (−0.27 wk; 95% CI: −0.50, −0.03) and risk of preterm birth [hazard ratio (HR) = 1.6%; 95% CI: 1.1, 2.3].[52] Arrebola et al.[81] found a negative association between 1,1-dichloro-2,2-bis(*p*-chlorophenyl)ethylene (*p,p′*-DDE) but not its metabolite 2,2-(2-chlorophenyl-4′-chlorophenyl)-1,1-dichloroethene (*o,p′*-DDE) and gestation length ($\beta = -0.004$; 95% CI: −0.008, −0.001). Based on urine samples collected at ∼26 wk of gestation, Silver et al.[94] reported that prenatal exposure to glyphosate (OR = 1.45; 95% CI: 1.00, 2.10) or aminomethylphosphonic acid (AMPA) (OR = 1.88; 95% CI: 1.35, 2.62) were associated with spontaneous preterm birth.

Intrauterine growth restriction. Evaluating data from 26 Brazilian states, Siqueira et al.[82] showed an association between pesticide use and risk of low birth weight ($\beta = 0.26$, p 0.045). Using national Brazilian data, Mendonça Guimarães et al.[84] observed higher PRs of low birth weight in the higher tertiles of fungicide (PR = 1.14; 95% CI: 1.10, 1.17), acaricide (PR = 1.07; 95% CI: 1.03, 1.10), herbicide (PR = 1.15; 95% CI: 1.12, 1.19), and insecticide (PR = 1.14; 95% CI: 1.11, 1.18) consumption. However, Cremonese et al.[83] reported null findings in a similar study that was limited to only three southern Brazilian states. Levario-Carrillo et al.,[89] using data from Mexico, reported a significant association between prenatal exposure to pesticides and the presence of intrauterine growth restriction (IUGR) (OR = 2.3;

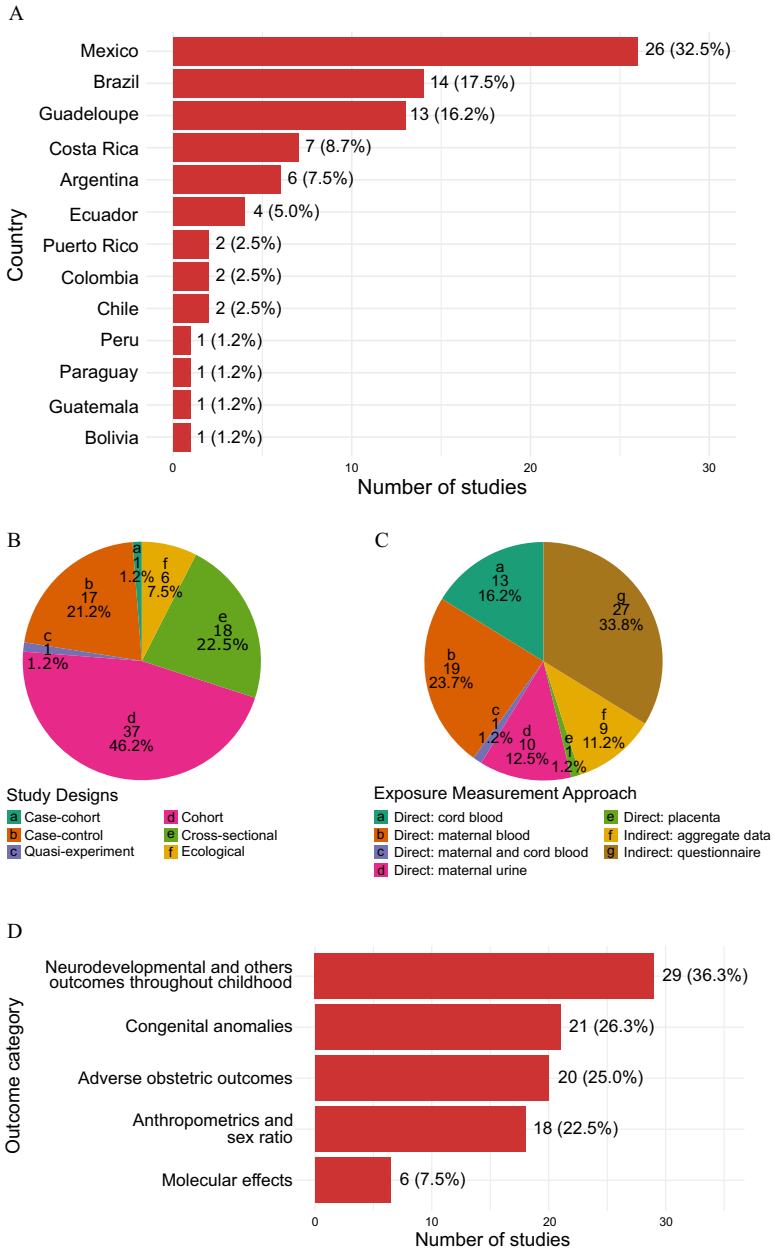

Figure 2. Characteristics of human observational studies assessing prenatal exposure to pesticides and adverse outcomes. Number of studies by (A) LAC country, (B) study design, (C) approaches employed to measure exposure, and (D) number of studies in each outcome category. Note that a study may be classified into multiple outcome categories.

95% CI: 1.0, 5.3). In Mexico, the study by Acosta-Maldonado et al.,[91] which analyzed placentas, identified an association between pesticide exposure and morphometric parameters of placental maturity, which, in turn, is linked to birth weight. Cecchi et al.[79] studied the outcomes of pregnant women in Argentina based on their residential proximity to fruit croplands with intense pesticide applications. The study did not identify any urban/rural differences in the rates of IUGR or newborns classified as small for gestational age (SGA). In another study, prenatal exposure to *p,p′*-DDE was not associated with higher risk for SGA.[93]

Spontaneous abortion. We identified seven studies evaluating exposure to pesticides and spontaneous abortion. DDT exposure was not related to spontaneous abortion among workers of the malaria control program in Mexico.[90] A rate of spontaneous abortion 8.5 times higher (95% CI: 6.72, 10.65) than average was observed in an agricultural area in Chile than at the national level; besides that, there were statistically higher rates of agricultural workers among women with spontaneous abortion than among all women from the same agricultural area.[86] Braga et al.[85] observed higher rates of fetal death in regions with high agricultural production compared with regions with low agricultural production in the state of São Paulo, Brazil. In Ecuador, Handal and Harlow reported a significant association between maternal employment in the flower industry within the past 6 y and an increased likelihood of reporting a history of spontaneous abortion (OR = 2.6; 95% CI: 1.03, 6.73).[47] Conversely, in the study from Blanco-Muñoz et al.[92] in Mexico, pregnant women who have worked as a floriculturist during the period of 3 months before pregnancy to 5 months into gestation did not have an increased risk of miscarriage compared with pregnant women who did not work as a floriculturist during the same period.

Table 1. Latin American and Caribbean studies on prenatal pesticide exposure and adverse obstetric outcomes published between 2000 and 2024 ($n = 20$).

Author, year	Country	Exposure	Outcome	Study design (n)	Key findings
Souza et al., 2006[78]	Argentina	OP exposure estimated by maternal blood cholinesterase levels	Gestational length	Cohort (342)	Gestational age decreased by 2 wk during the fumigation period
Cecchi et al., 2021[79]	Argentina	Maternal residential proximity to fruit croplands with intense pesticide applications	IUGR, preterm premature rupture of membranes, preterm birth (delivery at <37 wk gestation), fetal death, threat of premature labor, miscarriage, threatened miscarriage (a pregnancy-related bloody vaginal discharge or frank bleeding during the first half of pregnancy without cervical dilation), and SGA (weight-for-gestational age percentile <10 based on the INTERGROWTH-21st standards)	Cohort (776)	The rural population had a significantly higher rate of threatened miscarriage compared with the urban population (0.3% vs. 3%, $p < 0.05$). The percentage of intrauterine growth retardation, preterm premature rupture of membranes, intrauterine fetal death, fetal death, threat of premature labor, and miscarriage were similar in both groups
Rodriguez et al., 2023[80]	Argentina	OCPs, chlorpyrifos, trifluralin, and chlorothalonil levels measured from placental samples collected immediately after delivery	Gestational age at birth	Cross-sectional (85)	The gestational age was similar between urban and rural groups, despite the rural group showing higher levels of chlorpyrifos and total OCPs + chlorpyrifos
Arrebola et al., 2016[81]	Bolivia	o,p'-DDT and p,p'-DDE levels quantified in serum from cord blood samples collected after delivery	Length of gestation	Cohort (200)	p,p'-DDE was negatively associated with length of gestation ($\beta = -0.004$; 95% CI: −0.008, −0.001) and o,p'-DDT did not reach a significant association[a]
Siqueira et al., 2010[82]	Brazil	Pesticide use (kg/hectare/y)	Prematurity (live births with pregnancy duration <37 wk) and low birth weight (live births with weight <2,500 g)	Ecological (3,154,233)	An association between pesticide use and low birth weight ($\beta = 0.26$; 95% CI: 0.006, 0.52). No association was found between pesticide use and prematurity[b]
Cremonese et al., 2012[83]	Brazil	Per capita pesticide consumption	Premature birth (gestational age <22 wk), 1- and 5-min Apgar score, and low birth weight	Ecological (2,268,704)	In the upper quartile of pesticide consumption, higher PRs were observed for premature birth ($p < 0.001$) and low first ($p < 0.001$) and 5-min ($p < 0.001$) Apgar score. No significant findings were observed regarding low birth weight
Mendonça Guimarães et al., 2014[84]	Brazil	Per capita pesticides consumption	Premature births (live births <37 wk) and low birth weight (live births <2,500 g)	Ecological (NA)	Associations between premature births and fungicide (PR = 1.23; 95% CI: 1.19, 1.28), acaricide (PR = 1.08; 95% CI: 1.05, 1.12), herbicide (PR = 1.24; 95% CI: 1.20, 1.28), and insecticide (PR = 1.25; 95% CI: 1.21, 1.30); associations between low birth weight and fungicide (PR = 1.14; 95% CI: 1.10, 1.17), acaricide (PR = 1.07; 95% CI: 1.03, 1.10), herbicide (PR = 1.15; 95% CI: 1.12, 1.19), and insecticide (PR = 1.14; 95% CI: 1.11, 1.18)
Braga et al., 2023[85]	Brazil	Estimates of the amount of pesticides used within selected regions	Rates of fetal death before 22 wk and rates of fetal deaths weighing <500 g	Ecological (NA)	Higher ratios of fetal death rates at <500 g weight and higher ratios of fetal death rates before 22 wk in regions with high agricultural production compared with the region with the lowest agricultural production
Contreras-Levicoy et al., 2005[86]	Chile	Living in agricultural area	Spontaneous abortion	Cross-sectional (975)	Spontaneous abortion rates of 8.5 (95% CI: 6.72, 10.65) times higher in the agricultural area than the national level. More agricultural workers among women with spontaneous abortion than among those from the same agricultural area
Camacho and Mejía, 2017[87]	Colombia	Aerial spraying of glyphosate	Miscarriages	Quasi-experiment (687,735)	Aerial spraying significantly increases miscarriage rates ($\beta = 0.118$, $p < 0.01$)

Table 1. (*Continued.*)

Author, year	Country	Exposure	Outcome	Study design (*n*)	Key findings
Wendel de Joode et al., 2024[44]	Costa Rica	Pesticide metabolites (ETU, TCPY, 2,4-D, 3-PBA, DCCA, and OHP) measured from maternal urine samples collected during the first half (<20 wk) and second half (≥20 wk) of gestation	Length of gestation	Cohort (386)	No statistically significant findings[c]
Handal and Harlow, 2009[47]	Ecuador	Work in the flower industry	Spontaneous abortions	Cross-sectional (153)	Women in the flower industry had 2.6 times higher odds of reporting a history of spontaneous abortion compared with nonindustry workers (OR = 2.6; 95% CI: 1.03, 6.73)[d]
Kadhel et al., 2014[52]	Guadeloupe	Chlordecone levels quantified from maternal blood samples collected in the third trimester	Length of gestation and preterm birth (birth before 37 completed wk of gestation)	Cohort (818)	Chlordecone levels were associated with a shorter length of gestation (β = −0.27 wk; 95% CI: −0.50, −0.03), as well as with preterm birth (HR = 1.6; 95% CI: 1.1, 2.3)[e]
Torres-Arreola et al., 2003[88]	Mexico	Serum levels of *p,p′*-DDE, β-HCH and HCB from blood samples collected within 24 h after delivery	Preterm birth (<37 wk of gestation)	Case–cohort (233)	No statistically significant findings
Levario-Carrillo et al., 2004[89]	Mexico	Environmental exposure to cholinesterase inhibitory pesticides during the gestational period	IUGR (newborns with weight-for-gestational age <10th percentile)	Case–control (371)	Association between pesticide exposure and the presence of IUGR (aOR = 2.3; 95% CI: 1.0, 5.3)[f]
Salazar-García et al., 2004[90]	Mexico	DDT occupational exposure during pregnancy	Spontaneous abortion	Cross-sectional (9,187)	No statistically significant findings for spontaneous abortion
Acosta-Maldonado et al., 2009[91]	Mexico	Residing in agricultural communities, proximity to pesticide application zones, or cohabitation with agricultural worker during pregnancy	PMI and gestational age	Cross-sectional (54)	Exposure was associated with higher PMI. Significant correlation between PMI and birth weight (rho = 0.54, $p < 0.01$) and between PMI and gestational age (rho = 0.44, $p < 0.01$) from week 21 to 38
Blanco-Muñoz et al., 2013[92]	Mexico	Floricultural work between 3 months before the beginning of pregnancy and the first 5 months of pregnancy	Miscarriage (pregnancies ending at a gestational age of <20 wk)	Cross-sectional (612)	Floricultural work during the periconceptional period and the first 5 months of pregnancy did not increase the risk of miscarriage (OR = 0.59; 95% CI: 0.26, 1.31)
Torres-Sánchez et al., 2023[93]	Mexico	*p,p′*-DDE levels measured from maternal blood samples collected before 18 wk of pregnancy	Gestational age at birth, preterm birth (birth <37 complete wk), and SGA (weight-for-gestational age percentile <10 based on the INTERGROWTH-21st standards)	Cohort (170)	No statistically significant findings[g]
Silver, et al., 2021[94]	Puerto Rico	Glyphosate and AMPA concentrations in maternal urine samples collected during the second and third trimesters	Preterm birth (delivery before 37 completed wk of gestation)	Case–control (247)	Preterm birth was significantly associated with glyphosate (aOR = 1.45; 95% CI: 1.00, 2.10) and AMPA (aOR = 1.88; 95% CI: 1.35, 2.62) levels from urine samples collected at ∼26 wk of gestation, whereas associations with levels at ∼18 wk of gestation were null or inconsistent[h]

Note: 2,4-D, 2,4-dichlorophenoxyacetic acid; 3-PBA, 3-phenoxybenzoic acid; AMPA, aminomethylphosphonic acid; aOR, adjusted odds ratio; β-HCH, beta-hexachlorocyclohexane; BMI, body mass index; CI, confidence interval; DCCA, 3-(2,2-dichlorovinyl)-2,2-dimethylcyclopropanecarboxylic acid; DDE, dichlorodiphenyldichloroethylene; DDT, dichlorodiphenyltrichloroethane; ETU, ethylenethiourea; HCB, hexachlorobenzene; HR, hazard ratio; IUGR, intrauterine growth restriction; NA, data not available; OCPs, organochlorine pesticides; OHP, hydroxy pyrimethanil; OP, organophosphates; OR, odds ratio; PMI, placental maturity index; PR, prevalence ratio; SGA, small for gestational age; TCPY, 3,5,6-trichloro-2-pyridinol.

[a]Adjusted for gestational weight gain, maternal age, parity, smoking habit, log (newborn weight), and maternal BMI.

[b]Adjusted for proportion of pregnant women with a low number of prenatal care visits.

[c]Adjusted for newborn's sex, maternal age, parity, smoking during pregnancy, prepregnancy BMI, and country of birth.

[d]Adjusted for maternal age.

[e]Adjusted for maternal age, place of birth, enrollment site, marital status, educational level, BMI, high blood pressure during pregnancy, and total plasma lipid level (g/L).

[f]Adjusted for fat-free mass, placental weight, and anti-cytomegalovirus antibodies.

[g]Adjusted for newborn sex, gestational age at birth, number of previous pregnancies, tobacco consumption history, and BMI.

[h]Adjusted for maternal age, education, prepregnancy BMI, and smoking.

Another study found a significantly higher risk of threatened miscarriage, defined as a pregnancy-related bloody vaginal discharge or frank bleeding during the first half of pregnancy without cervical dilation, among Argentinian women living in close proximity to areas with intensive pesticide application compared with those in urban areas (3% vs. 0.3%, $p < 0.05$).[79] However, that study found no significant differences in terms of miscarriages or fetal death. In a quasi-experimental study performed with data from Colombia, it was shown that exposure to aerial sprayed glyphosate in the months preceding miscarriage had a strong and statistically significant association with the reported miscarriage ($\beta = 0.118$, $p < 0.01$).[87]

Congenital Anomalies

A total of 21 studies examining the potential association between prenatal pesticide exposure and congenital anomalies were included in the present review (Table 2). Among these studies, 11 had the primary objective of evaluating congenital anomalies in general, whereas the remaining 10 focused on specific groups of anomalies.

In the ecological study from Mendonça Guimarães et al.[84] using Brazilian data, the total pesticides and herbicides per capita consumption were correlated with the prevalence of congenital anomalies at birth. Moreover, when comparing the highest tertile of per capita pesticide consumption with the lowest tertile, higher PRs of congenital anomalies were observed in the highest tertile for fungicides (PR = 1.33; 95% CI: 1.28, 1.38), acaricides (PR = 1.25; 95% CI: 1.20, 1.30), insecticides (PR = 1.38; 95% CI: 1.33, 1.43), and herbicides (PR = 1.38; 95% CI: 1.33, 1.43). In another Brazilian study context, Siqueira et al.[82] found that the ratio of pesticide use by cultivated area was not associated with the frequency of total congenital abnormalities at birth. In the ecological study by Braga et al.,[85] an increase in the incidence of congenital anomalies among live births was observed over time. However, a pattern of higher incidence of congenital anomalies in regions with higher pesticide use, compared with regions with lower use, was not identified. On the other hand, a study by Tipiana et al.[110] in Peru found that women who reported going to sprayed fields (OR = 3.82; 95% CI: 1.92, 7.60), living near sprayed fields (OR = 3.07; 95% CI: 1.59, 5.92), and having a partner who works in sprayed fields (OR = 2.40; 95% CI: 1.21, 4.78) showed an increased risk of having a newborn with congenital anomalies compared with women who did not report these exposures. Similarly, Peruvian women who had direct exposure to pesticides during pregnancy presented a significantly increased risk of having a newborn with congenital anomalies compared with those who did not have such exposure (OR = 4.51; 95% CI: 1.77, 11.46).[109] Another study reported a higher proportion of parental exposure to pesticides and a higher proportion of maternal residence around fumigated crops among newborns with congenital anomalies than in newborns from the control group.[101] Salazar-García et al.[90] showed an association between DDE levels from fathers and the risk of congenital anomalies (OR = 3.76; 95% CI: 1.24, 11.44 for the highest quartile). One study conducted in Brazil investigated parental residence and occupation, whereas another study in Guadeloupe assessed maternal and cord blood chlordecone levels in relation to congenital anomalies, but neither study found an association.[56,96] Similarly, a case–control study from Argentina identified no difference in quantified pesticide levels from blood samples between mothers of newborns with congenital anomalies and mothers of control newborns.[95]

Dutra and Ferreira used data from a Brazilian state to compare the prevalence of different groups of congenital anomalies between a region with low pesticide use and another region with high pesticide use.[99] They found that the prevalence of cleft lip and palate (OR = 1.64; 95% CI: 1.27, 2.12), circulatory system anomalies (OR = 2.79; 95% CI: 1.87, 4.16), genitourinary system anomalies (OR = 1.62; 95% CI: 1.28, 2.06), and digestive system anomalies (OR = 1.56; 95% CI: 1.15, 2.13) were higher in the region with high pesticide use.

Considering more specific congenital anomalies, maternal occupation in agriculture from 3 months before to 1 month after the last menstrual period was identified as a risk factor for anencephaly (OR = 4.57; 95% CI: 1.05, 19.96).[105] Another study demonstrated that the use of pesticides in home gardens (OR = 5.66; 95% CI: 1.44, 22.20) and the use of pesticides in farms close to home (OR = 2.56; 95% CI: 1.21, 5.43) were associated with cleft lip and palate.[100] In addition, pesticide exposure during the first 3 months of pregnancy was associated with an increased risk of nonsyndromic cleft lip, cleft palate, or both (OR = 2.2; 95% CI: 1.1, 4.7).[107] In a cohort of orofacial cleft cases from a pediatric plastic surgery program in Guatemala, 49.1% of the mothers reported maternal pesticide or herbicide exposure during pregnancy.[102] A study administered questionnaires to mothers of newborns with central nervous system (CNS) anomalies.[108] It was found that 40.2% reported exposure to pesticides during pregnancy, whereas 39.8% reported exposure to herbicides. Some studies investigated associations between pesticide exposure and male external genital malformations.[84,97,103,104,106] Maternal levels of DDT and DDE were not associated with the risk of hypospadias.[103] Organochlorine pesticide levels from maternal blood samples were associated with cryptorchidism.[104,106]

The mortality caused by congenital anomalies was also assessed. Siqueira et al.[82] found an association between pesticide use and proportional mortality of congenital anomalies ($\beta = 1.52$, $p = 0.004$) and infant death rate ($\beta = 0.17$, $p = 0.039$) in infants <1 year of age. Cremonese et al.[98] investigated the mortality rate caused by anomalies in the CNS and cardiovascular system (CVS) in infants <1 y of age. They observed a trend of increased relative risk with an increase in pesticide use ($p < 0.05$) and higher relative risk for CNS (RR = 1.18; 95% CI: 1.01, 1.37) and CVS (RR = 1.15; 95% CI: 1.03, 1.29) in the highest quintile compared with the lowest quintile of exposure. The results were statistically significant in rural areas but not in urban areas.

Anthropometric Measures and Sex Ratio

We identified 16 studies assessing the association between prenatal pesticide exposure and anthropometric parameters in newborns and children (Table 3). In addition, 2 studies investigated the proportion of male births in populations with high pesticide exposure.

An ecologic study performed with data from the Brazilian state of Paraná did not find any correlation between the amount of pesticide sales and the proportion of male newborns.[112] There was no difference in the male-to-female sex ratio of children born from workers on the malaria control program in Mexico.[90]

Longnecker et al.[75] investigated the effect of *in utero* exposure to DDE on penile and anogenital dimensions in newborn males. The penis length, width, and anogenital distance did not differ significantly across categories of serum DDE levels. Interestingly, Torres-Sánchez et al.[67] observed a significant reduction in the anogenital distance index among boys ($\beta = -0.02$; 95% CI: −0.03, −0.003), but not among girls, associated with a 2-fold increase in maternal DDE serum levels during the first trimester of pregnancy. In another study, DDT was not associated with anogenital distance, but polychlorobiphenyls (PCBs) 28 ($\beta = -0.005$; 95% CI: −0.008, −0.001), PCB 74 ($\beta = -0.003$; 95% CI: −0.006, −0.0007), and PCB 170 ($\beta = -0.005$; 95% CI: −0.008, −0.001) were associated with anogenital distance reduction in boys.[114]

Studies where the exposure variable was defined as the third trimester of pregnancy coinciding with the period of pesticide spraying,[111] maternal residential proximity to fruit croplands,[79]

Table 2. Latin American and Caribbean studies on prenatal pesticide exposure and congenital anomalies published between 2000 and 2024 ($n = 21$).

Author, year	Country	Exposure	Outcome	Study design (n)	Key findings
Colussi et al., 2024[95]	Argentina	Organochlorine, organophosphate, and pyrethroid pesticides quantified from maternal blood samples collected after delivery	Congenital anomalies	Case–control (290)	There were no significant differences in the concentrations of the pesticides assessed between cases and controls
Siqueira et al., 2010[82]	Brazil	Pesticide use (kg/hectare/y)	Congenital abnormality at birth and infant death caused by congenital abnormality	Ecological (3,154,233)	Pesticide use was positively associated with proportional mortality by congenital abnormality in <1-y-old infants ($\beta = 1.52$, $p = 0.004$), as well as infant death rate by congenital abnormality ($\beta = 0.17$, $p = 0.039$). Associations with congenital abnormalities rates were null[a]
Gonçalves e Silva et al., 2011[96]	Brazil	Occupational and environmental exposure to pesticides	Congenital defects	Case–control (126)	No statistically significant findings
Gaspari et al., 2012[97]	Brazil	Occupational activities and lifestyles with potential exposure to EDCs before and during pregnancy	Male genital malformations	Cross-sectional (2,710)	High rate of occupational EDC exposure from mothers (>66%) and fathers (>55%) of newborns with male genital malformations. High fetal exposure to EDCs was reported (91% cases)
Cremonese et al., 2014[98]	Brazil	Per capita pesticide consumption	Infant mortality caused from CNS and CVS congenital malformations	Ecological (5,241)	Correlations between per capita pesticide consumption and mortality rates due to CNS and CVS defects in rural microregions ($p < 0.001$)
Mendonça Guimarães et al., 2014[84]	Brazil	Per capita consumption of pesticides	Birth defects and cryptorchidism	Ecological (NA)	Higher PRs of congenital anomalies in the highest tertile of exposure to fungicides (PR = 1.33; 95% CI: 1.25, 1.38), acaricides (PR = 1.25; 95% CI: 1.20, 1.30), insecticides (PR = 1.38; 95% CI: 1.33, 143), and herbicides (PR = 1.38; 95% CI: 1.33, 143) compared with the lowest tertile
Dutra and Ferreira, 2017[99]	Brazil	Regions with high and low pesticide use	Groups of congenital malformations	Cross-sectional (NA)	Region with higher exposure showed higher rates of cleft lip and palate (OR = 1.64; 95% CI: 1.27, 2.12), circulatory (OR = 2.79; 95% CI: 1.87, 4,16), genitourinary (OR = 1.62; 95% CI: 1.28, 2.06), and digestive (OR = 1.56; 95% CI: 1.15, 2.13) system anomalies
Silvestre et al., 2022[100]	Brazil	Parents' environmental and occupational exposures	Cleft lip and palate	Case–control (375)	The use of pesticides in home gardens (OR = 5.66; 95% CI: 1.44, 22.20) and the use of pesticides in farms close to home (OR = 2.56; 95% CI: 1.21, 5.43) were associated with cleft lip and palate
Braga et al., 2023[85]	Brazil	Estimates of the amount of pesticides used within selected regions	Prevalence of newborns with congenital anomalies per live birth	Ecological (NA)	An upward trend in the prevalence of congenital anomalies. Higher prevalence rates of congenital anomalies were not observed in regions with higher exposure
Rojas et al., 2000[101]	Chile	Occupational or environmental exposure to pesticides by parents during pregnancy	Congenital malformations	Case–control (858)	Higher frequency of agricultural labor or rural residency in the case group ($X2 = 19.52$, $p < 0.00001$). Association between rural residency and congenital malformations (OR = 2.16; 95% CI: 1.55, 3.00)
Rouget et al., 2020[56]	Guadeloupe	Chlordecone concentrations in maternal blood samples collected at third-trimester and cord blood samples collected after delivery	Congenital anomalies	Cohort (1,068)	No statistically significant findings[b]
Card et al., 2023[102]	Guatemala[c]	Pesticide and herbicide exposure during pregnancy through home use	Orofacial clefts	Cohort (55)	Pesticide or herbicide use during pregnancy was reported by 49.1% of the mothers
Flores-Luévano et al., 2003[103]	Mexico	Maternal serum DDT/DDE levels from maternal blood samples collected <18 y after delivery	Hypospadias	Case–control (69)	Maternal levels of DDT and DDE showed no association with hypospadias

Table 2. (*Continued.*)

Author, year	Country	Exposure	Outcome	Study design (*n*)	Key findings
Salazar-García et al., 2004[90]	Mexico	DDT occupational exposure during pregnancy	Congenital malformations	Cross-sectional (9,187)	Parents with estimated high exposure to DDT exhibited an elevated risk of having a child with congenital malformations (quartile 3: OR = 4.15; 95% CI: 1.38, 12.46; quartile 4 OR = 3.76; 95% CI: 1.24, 11.44)[d]
Waliszewski et al., 2005[104]	Mexico	Organochlorine pesticides in maternal blood serum lipids from samples collected postpartum	Undescended testis	Case–control (60)	The relative risk was higher for the case group compared with the control group for HCB (RR = 1.16; 95% CI: 1.07, 1.26), β-HCH (RR = 0.73; 95% CI: 0.66, 0.79), and total DDT (RR = 1.12; 95% CI: 1.09, 1.15)
Lacasaña et al., 2006[105]	Mexico	Parental occupational exposure to agricultural work	Anencephaly	Case–control (302)	Mothers involved in agricultural work during the periconceptional period had a higher risk of having children with anencephaly (OR = 4.57; 95% CI: 1.05, 19.96)[e]
Montes et al., 2010[106]	Mexico	Persistent organochlorine pesticides (HCB, β-HCH, *p,p′*-DDE, *o,p′*-DDT, *p,p′*-DDT) quantified from newborns' blood samples collected after delivery	Undescended testicles (cryptorchidism)	Case–control (82)	Association between serum levels of HCB (OR = 1.17; 95% CI: 1.08, 1.27) and total DDT (OR = 1.14; 95% CI: 1.09, 1.19) and cryptorchidism
Ibarra-Lopez et al., 2013[107]	Mexico	Environmental and occupational exposure to pesticides during the first trimester	Nonsyndromic cleft lip and palate	Case–control (204)	Association between pesticide exposure and nonsyndromic cleft lip and palate (aOR = 2.2; 95% CI: 1.1, 4.7)[f]
Enríquez-Vera et al., 2023[108]	Mexico	Types of pesticides reported by mothers	Congenital malformations of the CNS	Cross-Sectional (113)	During pregnancy, 40.2% of mothers reported exposure to pesticides, whereas 39.8% reported exposure to herbicides
Ojeda and Leite, 2018[109]	Paraguay	Occupational and environmental exposure to agrochemicals during pregnancy	Congenital malformations	Case–control (132)	Association between direct maternal exposure to agrochemicals and congenital malformations (OR = 4.51; 95% CI: 1.77, 11.46)
Tipiana et al., 2015[110]	Peru	Any contact with pesticides during pregnancy	Congenital malformations	Case–control (171)	Having exposure to pesticides (OR = 2.85; 95% CI: 1.46, 5.54), going to sprayed fields (OR = 3.82; 95% CI: 1.92, 7.60), living near sprayed fields (OR = 3.07; 95% CI: 1.59, 5.92), and having a partner working in sprayed fields (OR = 2.40; 95% CI: 1.21, 4.78) during pregnancy were associated with a higher risk of having a child with a congenital anomaly

Note: aOR, adjusted odds ratio; β-HCH, beta-hexachlorocyclohexane; CI, confidence interval; CNS, central nervous system; CVS, cardiovascular system; DDE, dichlorodiphenyldichloroethylene; DDT, dichlorodiphenyltrichloroethane; EDCs, endocrine-disrupting chemicals; HCB, hexachlorobenzene; NA, data not available; OR, odds ratio; PR, prevalence ratio; RR, relative risk; X2, result for the chi-squared test.

[a]Adjusted for proportion of pregnant women with a low number of prenatal care visits.

[b]Adjusted for maternal place of birth, maternal education, parity, gestational age, maternal age, and total plasma lipids.

[c]This study compared patients born in Guatemala with those from the United States.

[d]Adjusted for maternal age and application of malathion.

[e]Adjusted for age of the mother, socioeconomic level (family income and education of the mother), adverse reproductive history with previous children, folate intake of the mother, and energy of the mother.

[f]Adjusted for age at conception, years of schooling, region of origin of the mother, folic acid intake, and tobacco exposure.

Table 3. Latin American and Caribbean studies on prenatal pesticide exposure and anthropometrics or sex ratio published between 2000 and 2024 ($n = 18$).

Author, year	Country	Exposure	Outcome	Study design (n)	Key findings
Souza et al., 2006[78]	Argentina	OP exposure estimated by maternal blood cholinesterase levels	HC	Cohort (342)	Increase in HC in newborns of the women who lived in rural areas ($p \leq 0.05$) and who sprayed OP at home ($p \leq 0.05$)
Silvia et al., 2020[111]	Argentina	Third-trimester pregnancy coinciding with the period of pesticides spraying	Birth weight, length at birth, HC and the HC/weight ratio	Cross-sectional (53)	No statistically significant findings[a]
Cecchi et al., 2021[79]	Argentina	Maternal residential proximity to fruit croplands with intense pesticide applications	Newborn parameters	Cohort (776)	Higher ponderal index ($p < 0.05$), lower length at birth ($p < 0.01$), and HC <5th percentile ($p < 0.05$) in the rural group compared with the urban group[b]
Rodriguez et al., 2023[80]	Argentina	OCPs, chlorpyrifos, trifluralin, and chlorothalonil levels measured from placental samples collected immediately after delivery	Weight, length, and HC	Cross-sectional (85)	No statistically significant findings
Arrebola et al., 2016[81]	Bolivia	*o,p′*-DDT and *p,p′*-DDE levels quantified in serum from cord blood samples collected after delivery	Birth weight, HC, height, ponderal index	Cohort (200)	*p,p′*-DDE was associated with birth weight ($\beta = -0.014$; 95% CI: −0.028, −0.0010)[c]
Gibson and Koifman, 2008[112]	Brazil	Volume of pesticide sales	Proportion of male births	Ecological (NA)	No statistically significant findings
Villar et al., 2022[113]	Brazil[d]	Metabolite signatures untargeted from maternal blood samples collected around the second trimester and from umbilical cord venous blood samples collected at delivery	Fetal abdominal growth, postnatal growth, adiposity, and neurodevelopment	Cohort (3,206)	Maternal chlorothalonil was positively associated with an early accelerating growth phenotype and negatively associated with a faltering growth phenotype[e]
Wendel de Joode et al., 2024[44]	Costa Rica	Pesticide metabolites (ETU, TCPY, 2,4-D, 3-PBA, DCCA, and OHP) measured from maternal urine samples collected during the first half (<20 wk) and second half (≥20 wk) of gestation	Birth size: birth weight (g), body length (cm), and HC (cm)	Cohort (386)	Higher prenatal TCPY was associated with lower birth weight (−129.6; 95% CI: −255.8, −3.5 g) and smaller HC (−0.61; 95% CI: −1.05, −0.17 cm). 2,4-D was associated with lower birth weight (β per 10-fold-increase $= -125.1$; 95% CI: −228.8, −21.5), smaller HC ($\beta = -0.41$; 95% CI: −0.78, −0.03), and shorter body length ($\beta = -0.58$; 95% CI: −1.09, −0.07). ETU showed a nonlinearly association with HC during the second half of pregnancy[f]
Costet et al., 2015[54]	Guadeloupe	Chlordecone from cord blood sample collected at birth	Growth of children up to of 18 months of age	Cohort (222)	Association of chlordecone exposure and higher BMI in boys at 3 months of age ($\beta = 0.9$; 95% CI: 0.0, 1.8) and in girls at 8 months of age ($\beta = 0.7$; 95% CI: 0.0, 1.5)[g]
Hervé et al., 2016[53]	Guadeloupe	Chlordecone concentrations in cord blood collected immediately after delivery	Birth weight	Cohort (593)	No statistically significant findings[h]
Costet et al., 2022[60]	Guadeloupe	Chlordecone levels from cord blood sample collected after delivery	Adiposity in 7-y-old children	Cohort (575)	Boys ($\beta = 0.99$; 95% CI: 0.38, 1.61) and girls ($\beta = 0.48$; 95% CI: 0.03, 0.92) had higher adiposity scores in in the third quartile relative to the first quartile[i]
Salazar-García et al., 2004[90]	Mexico	DDT occupational exposure during pregnancy	Sex ratio	Cross-sectional (9,187)	No statistically significant findings

Table 3. (*Continued.*)

Author, year	Country	Exposure	Outcome	Study design (*n*)	Key findings
Longnecker et al., 2007[75]	Mexico	Maternal serum DDE and DDT levels from samples collected during the postpartum period	Anogenital and penile dimensions in newborn males	Cross-sectional (781)	No statistically significant findings[j]
Torres-Sánchez et al., 2008[67]	Mexico	DDE concentrations from maternal blood samples collected before pregnancy and during each trimester	Anogenital distance in male and female infants	Cross-sectional (71)	Reduction of anal position index among boys ($\beta = -0.02$; 95% CI: −0.03, −0.003, $p = 0.02$), but not among girls, only during the first trimester of pregnancy[k]
Cupul-Uicab et al., 2010[77]	Mexico	DDE and DDT levels measured from maternal serum collected within a day of delivery	Longitudinal measurements of height and BMI up to 40 months of age	Cohort (788)	No statistically significant findings[l]
Garced et al., 2012[70]	Mexico	DDE and DDT levels from maternal blood samples collected before pregnancy and during each trimester	Child growth parameters, at birth and during the first year	Cohort (253)	No statistically significant findings[m]
Loreto-Gómez et al., 2018[114]	Mexico	Persistent organic pollutants from maternal blood samples collected in the third trimester	Anogenital distance	Cohort (156)	Association between PCB 28 ($\beta = -0.005$; 95% CI: −0.008, −0.001), PCB 74 ($\beta = -0.003$; 95% CI: −0.006, −0.0007), and PCB 170 ($\beta = -0.005$; 95% CI: −0.008, −0.001) levels and anogenital distance index among boys[n]
Torres-Sánchez et al., 2023[93]	Mexico	*p,p′*-DDE levels measured from maternal blood samples collected before 18 wk of pregnancy	Birth weight and birth length	Cohort (170)	No statistically significant findings[o]

Note: 2,4-D, 2,4-dichlorophenoxyacetic acid; 3-PBA, 3-phenoxybenzoic acid; BMI, body mass index; CI, confidence interval; DCCA, 3-(2,2-dichlorovinyl)-2,2-dimethylcyclopropanecarboxylic acid; DDE, dichlorodiphenyldichloroethylene; DDT, dichlorodiphenyltrichloroethane; ETU, ethylenethiourea; HC, head circumference; NA, data not available; OCPs, organochlorine pesticides; OHP, hydroxy pyrimethanil; OP, organophosphates; PCB, polychlorinated biphenyl; TCPY, 3,5,6-trichloro-2-pyridinol.

[a]Adjusted for sex, gestational age, and parity.

[b]Adjusted for sex and gestational age.

[c]Adjusted for gestational weight change, maternal age, parity, smoking habit, gestational age, and maternal BMI.

[d]This study adopted a multinational design conducted across Brazil, Kenya, Pakistan, South Africa, Thailand, and the UK.

[e]Adjusted for maternal age and fetal sex.

[f]Adjusted for newborn's sex, maternal age, parity, smoking during pregnancy, prepregnancy BMI, and country of birth.

[g]Adjusted for duration of gestation, maternal place of birth, maternal age, parity, maternal prepregnancy height and weight, maternal weight gain during pregnancy, education level, marital status, smoking and alcohol consumption during pregnancy, and cord total lipid concentrations.

[h]Adjusted for gestational age, sex of the newborn, maternal height, maternal BMI, enrollment site, place of birth, family situation, education level, parity, gestational hypertension, gestational diabetes.

[i]Adjusted for age at measurement, maternal place of birth, maternal BMI before pregnancy, maternal level of education, duration of breastfeeding, time spent exercising per week, time spent watching TV or playing videogames per week, obesogenic dietary habits at 7 years of age, and cord blood total lipids (g/L) and concentrations of PCB153 and *p,p′*-DDE.

[j]Adjusted for birth weight, gestational age, urban vs. rural, hospital, and research nurse.

[k]Adjusted for birth weight and age at evaluation.

[l]Adjusted for smoking, hospital, rural residence, gestational age at delivery, maternal education, and maternal height.

[m]Adjusted for age at evaluation, maternal age, height, BMI, parity, tobacco, breastfeeding, iron, zinc, and caloric intake during the first trimester of pregnancy.

[n]Adjusted for the boy's age, age squared, and height.

[o]Adjusted for newborn sex, gestational age at birth, number of previous pregnancies, tobacco consumption history, and BMI.

and *p,p′*-DDE[93] did not find an association with birth weight. Conversely, in one study, *p,p′*-DDE showed a statistically significant positive linear relationship with birth weight (β = 0.012; 95% CI: 0.003, 0.021), whereas *o,p′*-DDT was negatively associated with this outcome (β = −0.014; 95% CI: −0.028, −0.0010).[81] In the cohort from Guadeloupe, chlordecone in cord blood was not associated with birth weight. However, a U-shaped association between cord blood chlordecone concentration and birth weight was observed, particularly among pregnant women who had excessive gestational weight gain.[53] In a subsequent time-delayed study on the same cohort, chlordecone in cord blood was associated with a higher body mass index (BMI) in boys at 3 months of age and in girls at 8 and 18 months of age.[54] At 7 years of age, placement in the third quartile of prenatal exposure was associated with significantly increased adiposity, more markedly in boys.[60] DDE levels in the maternal serum taken at childbirth were not associated with differences in BMI or height in measurements taken from newborns during their first 38 months.[76] Similarly, prenatal DDE exposure was not associated with child growth parameters at birth and at 1, 3, 6, and 12 months of age.[70]

A multinational study, which included data from Brazil, assessed prenatal exposure based on untargeted metabolic signature in maternal and umbilical samples.[113] Exposure to hydroxychlorothalonil, a metabolite of the fungicide chlorothalonil, was identified in both types of samples. Levels of the metabolite in maternal blood showed associations with two extreme abdominal circumference fetal phenotypes: *a*) a positive association between exposure to chlorothalonil and early accelerating growth, and *b*) a negative association between exposure to the fungicide and faltering growth. In a Costa Rican cohort study aiming to analyze the association between prenatal exposure to pesticides and birth size parameters, TCPY and 2,4-D were associated with lower birth weight and smaller head circumference.[44] Furthermore, 2,4-D was associated with shorter body length, and ETU presented a nonlinear association with head circumference. In another study, no significant associations were found between levels of *p,p′*-DDE, chlorpyrifos, and organochlorine pesticides and neonatal anthropometric parameters.[80]

In a cohort study, pregnant women who lived in a rural area and reported spraying pesticides at home had newborns with a greater head circumference than their counterparts ($p < 0.05$ for both cases).[78] Another similar study did not find a difference in newborns' head circumference between mothers from rural and urban regions. However, a higher proportion of newborns with head circumference below the fifth percentile of the standard curve, after correction for sex and gestational age, was observed in the rural group ($p < 0.05$).[79] Other studies that included head circumference as an outcome did not find significant results.[81,111]

Neurodevelopmental and Other Outcomes throughout Childhood

We identified 29 publications on childhood outcomes and their association with prenatal pesticide exposure (Table 4). Among these, 22 studies investigated cognitive, sensory, or neurological development, 4 examined childhood cancers, and 3 focused on respiratory diseases in infancy.

Neurodevelopment. Five studies on neurodevelopment originated from the same Mexican prospective cohort, which recruited 1,585 women of reproductive age from 2001–2005 in four municipalities in the State of Morelos.[65,66,68,69,71] In this region, DDT was used to eliminate the malaria mosquito until 1998. Pregnant women had DDT and DDE measured in blood during the three trimesters of pregnancy and their children were evaluated up to 60 months of age. The number of children evaluated for neurobehavioral outcomes across the five studies varied from 167 to 270. The authors found a negative association between DDE maternal levels during the first trimester of pregnancy and the Psychomotor Development Index of the Bayley Scale II version throughout the first year of life (β = −0.52; 95% CI: −0.96, −0.075).[66] In addition, assessments conducted between 3.5 and 5 years of age revealed negative associations between maternal DDE levels from the third trimester and general cognitive index (β = −2.01; 95% CI: −3.64, −0.38), as well as the memory (β = −1.26; 95% CI: −2.20, −0.32) component of the McCarthy Scale.[65] However, other investigations conducted during different periods of the neurodevelopment yielded null results.[68,69,71] Three other studies from Mexico also originated from another cohort, which recruited pregnant women and their offspring from Mexico City between 1997 and 2001.[72–74] Prenatal pesticide exposure was assessed by quantifying 3-PBA and TCPY levels in maternal urine samples collected during the third trimester of pregnancy. The sample size ranged from 137 to 187 mother–child pairs across the three studies. The outcomes assessed included neurodevelopment, sleep disorders, and attention deficit hyperactivity disorder (ADHD)–related behavior. No significant results were found, except for an association between prenatal TCPY levels and sleep duration (β = 36.2; 95% CI: 5.2, 67.3).[74]

Seven studies came from a birth cohort from Guadeloupe archipelago.[55,57–59,61,62,64] This cohort, named the TIMOUN Birth Cohort, included 1,068 pregnant women during their second or third trimester recruited between 2004 and 2007 in Guadeloupe. The number of children enrolled in the six studies varied from 111 to 410. They focused on prenatal, perinatal, and childhood exposure to chlordecone, a highly persistent organochlorine insecticide that was intensively used in banana fields. Concentrations of chlordecone and other environmental contaminants were measured in the umbilical cord blood from newborns in the cohort, and subsequently from blood samples provided by these children at various ages. The investigators reported negative associations with fine motor functions, visual recognition memory, novelty preference, and visual processing speed of pre- and postnatal exposures in 7- and 18-month-old children.[57,58,61] In assessments of children at ∼7 years of age, associations were observed between cord chlordecone levels and adverse effects on visual and neuromotor function, with differing effects noted between girls and boys concerning cognitive function and behavior problems.[55,59,64] In addition, one study found no sex-typed toy preference in relation to prenatal exposure to chlordecone.[62]

Two studies analyzed data from the community-based prospective birth cohort of the Costa Rica Caribbean, Infants' Environmental Health (ISA) study. In one study, the authors investigated the association between prenatal exposure to excess manganese (found in fungicides) and neurodevelopment at 1 year of age.[41] It was observed that among boys, higher manganese levels were associated with lower social-emotional scores (β per 10-fold increase = −4.6; 95% CI: −8.5, −0.8). In another study, the authors examined 2,4-D, pyrethroids, pyrimethanil, and chlorpyrifos metabolites.[45] An association was found between prenatal 2,4-D concentration and language (β per 10-fold increase = −2.0; 95% CI: −3.5, −0.5) and motor (β per 10-fold increase = −2.2; 95% CI: −4.2, −0.1) abilities composite scores for all children.

Three studies came from Ecuador, assessing parental occupation in the agricultural sector as the exposure.[46,48,49] Prenatal maternal pesticide exposure was associated with deficits in the motor tests, visuospatial performance, and visual memory in one study,[46] and with lower fine motor skills and poorer visual acuity in another study.[49] In the study by Grandjean et al.,[48] prenatal

Table 4. Latin American and Caribbean studies on prenatal pesticide exposure and neurodevelopmental and other outcomes throughout childhood published between 2000 and 2024 ($n = 29$).

Author, year	Country	Exposure	Outcome	Study design (n)	Key findings
Neurodevelopment					
Benavides-Piracón et al., 2022[115]	Colombia	Environmental pesticide exposure during pregnancy	School-age children's cognitive ability	Cross-sectional (232)	No statistically significant findings[a]
Mora et al., 2018[41]	Costa Rica	ETU from maternal urine samples, Mn from maternal hair and blood samples collected across second and third trimesters	Neurodevelopmental outcomes in 1-y-old infants	Cohort (355)	Association between hair Mn levels and lower social-emotional scores among boys ($\beta = -4.6$; 95% CI: −8.5, −0.8)[b]
Conejo-Bolaños et al., 2024[45]	Costa Rica	OH-PYR, 5-OH-TBZ, TCPY, DCCA, 3-PBA, and 2,4-D measured from maternal urine samples during the second and third trimesters	Children's neurodevelopmental status at 1 year of age	Cohort (355)	Prenatal 2,4-D concentrations were associated with lower language (β per 10-fold increase $= -2.0$; 95% CI: −3.5, −0.5) and motor (β per 10-fold increase $= -2.2$, 95% CI: −4.2, −0.1) abilities composite scores across all children[c]
Grandjean et al., 2006[48]	Ecuador	Report of pesticide exposure during pregnancy	Blood pressure, neurological function, and neurobehavioral development	Cross-sectional (72)	Association between prenatal pesticide exposure and higher blood pressure ($\beta = 4.57$, SE = 1.89, $p = 0.018$), and lower Stanford–Binet copying score ($\beta = -1.01$, SE = 0.37, $p = 0.009$)[d]
Handal et al., 2008[46]	Ecuador	Maternal occupation in the flower industry	Neurobehavioral development of infants and toddlers 3–23 months of age	Cross-sectional (121)	Children whose mothers worked in the flower industry during pregnancy scored lower in fine motor skills ($\beta = -8.04$; 95% CI: −12.98, −3.10) and had a higher likelihood of having poor visual acuity (aOR = 4.7; 95% CI: 1.1, 20)[e]
Harari et al., 2010[49]	Ecuador	Paternal and maternal occupational pesticide exposure during pregnancy	Neurobehavior of schoolchildren 6–8 months of age	Cross-sectional (84)	Prenatal maternal exposure was associated with motor speed ($\beta = -7.1$; 95% CI: −12.5, −1.6), motor coordination (OR = 5.32; 95% CI: 1.03, 27.62), visuospatial performance ($\beta = 0.5$; 95% CI: 0.2, 1.0), and visual memory (OR = 6.62; 95% CI: 1.02, 42.93)[f]
Dallaire et al., 2012[57]	Guadeloupe	Chlordecone concentrations from cord blood samples collected at birth	Cognitive, visual, and motor development of 7-month-old infants	Cohort (153)	Association with reduced novelty preference ($\beta = -0.19$; 95% CI: −0.35, –0.03) and low scores on the fine motor scale (OR = 1.26; 95% CI: 1.09, 1.47)[g]
Boucher et al., 2013[58]	Guadeloupe	Chlordecone concentrations from cord blood samples collected at birth	Development in 18-month-old infants	Cohort (141)	Association with poorer fine motor scores ($\beta = -0.24$, $p = 0.03$)[h]
Cordier et al., 2015[61]	Guadeloupe	Chlordecone concentrations from cord blood samples collected at birth	Neurodevelopment at 18 months of age	Cohort (111)	The association with fine motor score remain significant among boys ($\beta = -10.1$; 95% CI: −19.4, −0.8)[i]
Cordier et al., 2020[62]	Guadeloupe	Chlordecone concentrations from cord blood samples collected at birth	Sex-typed play behavior of 7-y-old children	Cohort (116)	No statistically significant findings
Saint-Amour et al., 2020[55]	Guadeloupe	Chlordecone concentrations from cord blood samples collected at birth	Visual functions in school-age children ($\sim$7 years of age)	Cohort (285)	Association with lower contrast sensitivity ($\beta = -0.068$; 95% CI: −0.133, −0.002)[j]
Desrochers-Couture et al., 2022[59]	Guadeloupe	Chlordecone concentrations from cord blood samples collected at birth	Visuospatial processing and neuromotor function in 7-y-old children	Cohort (410)	Association with a regular frequency pattern of subtle hand tremors in dominant ($\beta = 0.02$; 95% CI: 0.002, 0.03) and nondominant hands ($\beta = 0.02$; 95% CI: 0.01, 0.04)[k]
Oulhote et al., 2023[64]	Guadeloupe	Chlordecone concentrations from cord blood samples collected at delivery	Cognitive function and behavior problems in 7-y-old children	Cohort (391)	Association with higher processing speed scores in boys ($\beta = 0.84$; 95% CI: 0.08, 1.60) and higher internalizing problems scores in girls (aMR = 1.03; 95% CI: 1.00, 1.06)[l]
Torres-Sánchez et al., 2007[66]	Mexico	Levels of p,p'-DDE in maternal serum samples collected during each trimester	Psychomotor and mental development during the first year	Cohort (244)	Levels of p,p'-DDE from the first trimester were associated with a reduction in the Psychomotor Development Index ($\beta = -0.52$; 95% CI: −0.96, −0.075)[m]
Torres-Sánchez et al., 2009[69]	Mexico	Levels of p,p'-DDE and p,p'-DDT in maternal serum samples collected during each trimester	Psychomotor and mental development until 30 months of age	Cohort (270)	No statistically significant findings

Table 4. (*Continued.*)

Author, year	Country	Exposure	Outcome	Study design (*n*)	Key findings
Bahena-Medina et al., 2011[68]	Mexico	Levels of DDE and *p,p′*-DDT in maternal serum samples collected during each trimester	Neonatal neurodevelopment at 1 month of age	Cohort (265)	No statistically significant findings
Torres-Sánchez et al., 2013[65]	Mexico	Levels of DDE and DDT in maternal serum samples collected during each trimester	Neurodevelopment at 42–60 months of age	Cohort (203)	Association between third-trimester DDE levels and the general cognitive index (β = −2.01; 95% CI: −3.64, −0.38) and the memory (β = −1.26; 95% CI: −2.20, −0.32) components of the McCarthy scale[n]
Fortenberry et al., 2014[72]	Mexico	Urinary TCPY levels from maternal third-trimester samples	Child neurodevelopment, specifically ADHD-related characteristics	Cohort (187)	No statistically significant findings
Osorio-Valencia et al., 2015[71]	Mexico	*p,p′*-DDE levels in maternal serum samples collected during the first, second, and third trimesters	Development of lateralization and spatial orientation in children 5 years of age	Cohort (167)	No statistically significant findings
Watkins et al., 2016[73]	Mexico	3-PBA concentrations in maternal urine samples collected during the third trimester	Child neurodevelopment at 24 and 36 months of age	Cohort (187)	No statistically significant findings
Zamora et al., 2022[74]	Mexico	Concentrations of 3-PBA and TCPY in maternal urinary samples collected during the third trimester	Sleep duration, timing, and fragmentation during adolescence	Cohort (137)	Higher tertile of TCPY levels was associated with longer sleep duration (β = 36.2; 95% CI: 5.2, 67.3)[o]
Jenkins et al., 2024[116]	Puerto Rico	Levels of glyphosate and its derivate AMPA quantified from maternal urine samples collected across the second and the third trimester of pregnancy	Children's neurodevelopmental status at 6, 12, and 24 months of age	Cohort (143)	At 12 months of age, associations were found between AMPA concentration and communication score (% change = 5.32; 95% CI: 9.04, 1.61), as well as the receptive (% change = 13.20; 95% CI: 21.97, 4.43) communication subdomain. At 24 months of age, there was negative association between AMPA concentration and communication score (% change = 7.00; 95% CI: 11.75, 2.26) and related subdomain scaled scores receptive (% change = 14.03; 95% CI: 24.82, 3.25) and expressive (% change = 14.94; 95% CI: 26.78, 3.11). Negative association between AMPA concentration and cognitive score (% change = 4.02; 95% CI: 6.72, 1.32) and associated subdomains attention and memory (% change = 9.54; 95% CI: 17.21, 1.86) and perception and concepts (% change = 8.10; 95% CI: 14.32, 1.87)
Leukemia					
Ferreira et al., 2012[50]	Brazil	Exposure to chemicals (including pesticides) during pregnancy	Childhood leukemia	Case–control (833)	Association between childhood leukemia development and maternal chemical exposure during pregnancy (OR = 1.35; 95 CI: 1.16, 2.58)
Ferreira et al., 2013[51]	Brazil	Pesticide exposure during the 3 months before pregnancy (periconceptual period) or throughout each trimester of pregnancy	Childhood leukemia	Case–control (675)	Pesticide use at any time during pregnancy was associated with ALL (aOR = 2.10; 95% CI: 1.14, 3.86) and AML (aOR = 5.01; 95% CI: 1.97, 12.7) in children 0–11 months of age, and with ALL (aOR = 1.88; 95% CI: 1.05, 5.23) in children 12–23 months of age[p]
Monge et al., 2007[39]	Costa Rica	Occupational, environmental, and home pesticide exposures of both parents	Childhood leukemia	Case–control (879)	Maternal pesticide exposure during the first trimester or second trimester was associated with childhood leukemia (aOR = 22.0; 95% CI: 2.8, 171.5 and aOR = 4.5; 95% CI: 1.4, 14.7, respectively). Paternal pesticide exposure during the second trimester also showed an association (aOR = 1.5; 95% CI: 1.0, 2.3)[q]

Table 4. (*Continued.*)

Author, year	Country	Exposure	Outcome	Study design (*n*)	Key findings
Hyland et al., 2018[40]	Costa Rica	Parental occupational and residential exposure to pesticides	Childhood ALL	Case–control (828)	Reported pregnancy exposure to insecticides was associated with ALL among boys (OR = 1.75; 95% CI: 1.13, 2.73)[r]
Respiratory infections					
Mora et al., 2020[42]	Costa Rica	Concentrations of ETU, TCPY, 3-PBA, 2,4-D, DCCA, OH-PYR, and 5-OH-TBZ in maternal urine samples collected during each trimester of pregnancy	Respiratory health in the first year of life	Cohort (355)	Association between high ETU concentrations and wheezing (OR = 0.50; 95% CI: 0.26, 0.96)
Islam et al., 2022[43]	Costa Rica	Chlorpyrifos, pyrethroids, mancozeb, pyrimethanil, thiabendazole and 2, 4-D measured from maternal urine samples collected during pregnancy	Respiratory and allergic outcomes among 5-y-old children	Cohort (303)	No statistically significant findings
Cupul-Uicab et al., 2014[77]	Mexico	*p,p′*-DDE and *p,p′*-DDT levels from maternal serum samples collected within a day of delivery	Recurrent lower respiratory tract infections during childhood among boys	Cohort (747)	No statistically significant findings

Note: % change, percentage change; 2,4-D, 2,4-dichlorophenoxyacetic acid; 3-PBA, 3-phenoxybenzoic acid; 5-OH-TBZ, hydroxythiabendazole; ADHD, attention deficit hyperactivity disorder; ALL, acute lymphoid leukemia; AML, acute myeloid leukemia; AMPA, aminomethylphosphonic acid; aMR, adjusted mean ratio; aOR, adjusted odds ratio; BMI, body mass index; CI, confidence interval; DCCA, 3-(2,2-dichlorovinyl)-2,2-dimethylcyclopropanecarboxylic acid; DDE, dichlorodiphenyldichloroethylene; DDT, dichlorodiphenyltrichloroethane; ETU, ethylene thiourea; HOME, Home Observation for Measurement of the Environment; IQ, intelligence quotient; Mn, manganese; OH-PYR, hydroxypyrimethanil; OR, odds ratio; PCB, polychlorinated biphenyl; SE, standard error; TCPY, 3,5,6-trichloro-2-pyridinol.

[a]Adjusted for child's sex, maternal educational attainment, and low height for age.

[b]Adjusted for maternal education, parity, gestational age at birth, child age, HOME score, and location of neurodevelopmental assessment.

[c]Adjusted for maternal education, parity, sex, gestational age at birth, child age, HOME score, location of assessment at 1-year visit, Mn in hair, Mn in blood, and ETU.

[d]Adjusted for age, sex, and body weight.

[e]Adjusted for anemia, stimulation at home, housing construction.

[f]Adjusted for the child's sex, age, BMI, number of daily meals, stunting, hematocrit, school grade, having repeated one grade, maternal education level, family living in a traditional house, drinking-water supply, and paternal education and employment.

[g]Adjusted for prepregnancy maternal employment status, cord plasma lipid concentrations, infant vitamin supplementation, postnatal exposure to maternal tobacco smoke, number of adults living with the infant, and child examiner and for plasma lipid concentrations, age of infant at testing, birthplace of mother, and domestic violence.

[h]Adjusted for sex, maternal education, and child age.

[i]Adjusted for maternal educational level and child age.

[j]Adjusted for child sex and age, maternal IQ, maternal education, maternal alcohol intake during pregnancy, and plasma lipids.

[k]Adjusted for child sex and age, as well as maternal Raven score and prenatal PCB-153 levels.

[l]Adjusted for child's age and sex, maternal age, parity, Raven score, education, marital status, monthly household income, and alcohol and smoking during pregnancy.

[m]Adjusted for birth weight, age at evaluation, breastfeeding, and HOME score at 6 months of age.

[n]Adjusted for child's age at examination, mother's education, marital status when child was 4 years of age, and HOME score, sex of child, and attendance at a child care center.

[o]Adjusted for maternal characteristics: education, parity, marital status, specific gravity, and adolescent characteristics: sex and age.

[p]Adjusted for use of oral contraceptives during pregnancy, maternal age and education, child's birth weight and skin color.

[q]Adjusted for residence (urban or rural).

[r]Adjusted year of birth and socioeconomic status.

pesticide exposure was associated with higher blood pressure and lower visuospatial parameter scores.

In a cross-sectional study conducted in Bogotá, prenatal exposure to pesticides was not associated with cognitive abilities in children.[115] In a prospective cohort study from Puerto Rico, the relationship between prenatal exposure to glyphosate and children's neurobehavioral development at 6, 12, and 24 months of age was assessed.[116] No statistically significant associations were found between glyphosate or AMPA concentrations and the neurobehavioral scores at 6 months of age. However, at 12 months of age, associations were observed between AMPA concentration and the communication score, whereas at 24 months of age, associations included motor, communication, and cognitive domain scores.

Childhood leukemia. Four articles investigated the association between prenatal pesticide exposure and childhood leukemia. In a case–control study, an association was found between maternal exposure to chemicals (including pesticides) during pregnancy and childhood leukemia (OR = 1.36; 95% CI: 1.16, 1.59).[50] In another article, children born to women who had at least one exposure to pesticides during pregnancy had a greater likelihood of developing acute lymphoid leukemia (OR = 2.10; 95% CI: 1.14, 3.86) and acute myeloid leukemia (OR = 5.01; 95% CI: 1.97, 12.70) in children 0–11 months of age, and acute lymphoid leukemia (OR = 1.88; 95% CI: 1.05, 5.23) in children 12–23 months of age.[51] In a different investigation, maternal exposures to any pesticides at any time (OR = 2.2; 95% CI: 1.0, 4.8), during the year before conception (OR = 2.4; 95% CI: 1.0, 5.9), and during the first (OR = 22.0; 95% CI: 2.8, 171.5) and second (OR = 4.5; 95% CI: 1.4, 14.7) trimesters were associated with an increased risk of having a child with leukemia.[39] In addition, an association was found for paternal exposures to any pesticides during the second trimester (OR = 2.2; 95% CI: 1.0, 4.8). In the study by Hyland et al.,[40] maternal insecticide use during pregnancy, but not herbicide use, was associated with increased odds of acute lymphoid leukemia among boys (OR = 1.75; 95% CI: 1.13, 2.73). This association appeared to be dose dependent ($p_{trend} = 0.03$).

Respiratory infections. Three studies explored the association between prenatal pesticide exposure and respiratory infections in early life. Cupul-Uicab et al.[77] did not find an association between prenatal exposure to *p,p′*-DDE or *p,p′*-DDT and lower respiratory tract infections (LRTI) in infants up to 2 years of age. In the cohort study by Mora et al.,[42] fungicides and organophosphate (OP) insecticides were not statistically associated with LRTI, but high levels of ETU (a metabolite of mancozeb) in the second half of pregnancy were associated with reduced odds of wheezing during the first year of life (OR = 0.50; 95% CI: 0.26, 0.96). Subsequent investigation on the same cohort at 5 years of age also did not reveal an association between metabolites of pesticides and LRTI.[43]

Molecular Effects

We found six observational human studies examining the molecular effects of prenatal pesticide exposure (Table 5). A study from Argentina aimed to determine the presence of environmental xenobiotics in the maternal blood of mothers of children with or without congenital anomalies.[95] In addition, the authors investigated the correlation between the xenobiotics present in maternal blood and genotoxicity parameters using the comet assay and the cytome assay. In the comet assay, β-HCH was positively correlated with the damage index. Furthermore, in the cytome assay, positive correlations between genotoxicity parameters were observed with levels of hexachlorobenzene (HCB), δ-HCH, lindane, exo-heptachlor epoxide, and methyl chlorpyrifos. In another study conducted in Argentina, placental parameters, besides target enzymes, erythrocyte osmotic fragility and DNA damage from umbilical blood cells were assessed.[117] The study aimed to investigate whether newborns of mothers living in a rural area with intense use of OPs had alterations in placental or biomarkers of oxidative stress. The researchers observed that the placental weight and placental index were higher in the group exposed to pesticides compared with the control group. In addition, higher erythrocyte osmotic fragility, higher DNA damage index, and lower superoxide dismutase (SOD) activity were observed in the rural group from the spray season compared with the rural group from the nonspray season. Acetylcholinesterase and SOD activities were found to be inversely correlated with the DNA damage index. In a Brazilian study comparing chromosomal damage in peripheral blood lymphocytes and oral epithelial cells between pregnant women from rural and urban areas, no statistical difference was found in the results of the micronucleus test.[118]

Three studies have investigated the effects of pesticide exposure on thyroid hormone levels. In a study from the TIMOUN cohort, umbilical cord chlordecone levels were associated with increased thyroid-stimulating hormone (TSH) levels at 3 months of age in boys.[61] In another study conducted with the same cohort, the association between *in utero* exposure to chlordecone and levels of thyroid, metabolic, and sex-steroid hormones at 7 years of age was investigated.[63] Higher levels of chlordecone were associated with higher levels of TSH in girls, as well with the hormones dehydroepiandrosterone (DHEA), total testosterone (TT) and dihydrotestosterone (DHT) in both sexes. In a birth cohort study from Bolivia, newborn TSH levels were not significantly associated with *p,p′*-DDE or *o,p′*-DDT quantified in serum from cord blood.[81]

Discussion

Latin America constitutes a geographical region with pronounced reliance on agricultural activities as a source of income.[2,119] The global rise in agricultural productivity over the past decades has co-occurred with an increase in the production and consumption of formulated pesticides.[3–5,120–122] A large amount of pesticides are formulated and commercialized by enterprises situated outside of LAC.[123–125] However, due to rigorous application of regulations within their countries of production, the use of some of these compounds is exclusively permissible in middle- and low-income nations, and not in the countries were they are produced.[126,127] Previous literature reviews have considered the accumulating evidence regarding health risks associated with environmental contamination resulting from pesticide use.[33,128] Compared with the review by Zúñiga-Venegas et al.,[33] our review exclusively focused on prenatal pesticide exposure and broadened the search to include six databases, as well as extending the publication date range to a larger and more recent time frame. Of the total 80 studies included in our review, 44 were not included on the previous review. The present scoping review identified a substantial body of literature from LAC concerning the potential risks associated with pesticide exposure during pregnancy. The findings underscore the importance of allocating resources from the generated agricultural wealth toward the investigation and mitigation of the repercussions linked to pesticide use in LAC agriculture.

Some studies included in the present review indicated an association between prenatal pesticide exposure and preterm birth. In animal studies, it was observed that exposure of pregnant rabbits to DDT increased the incidence of prematurity.[129] Given that DDT and its metabolites possess the capability to interact with progesterone receptors in an antagonistic manner,[130] exposure to DDT during pregnancy could potentially impact the duration of gestation. When human endometrial endothelial cells were

Table 5. Latin American and Caribbean studies on prenatal pesticide exposure and molecular adverse effects published between 2000 and 2024 ($n = 6$).

Author, year	Country	Exposure	Outcome/biological sample	Study design (n)	Key findings
Quintana et al., 2017[117]	Argentina	Living in an area with intensive pesticide application	Red blood cell osmotic fragility and cell viability	Cross-sectional (151)	Higher erythrocyte osmotic fragility and a higher DNA damage index were observed in the rural group from the spray season compared with the rural group from the nonspray season
Colussi et al., 2024[95]	Argentina	OCPs, OPs, and PYR quantified from maternal blood samples collected after delivery	Genotoxicity assessed using the comet assay and the cytome assay	Case–control (290)	β-HCH was positively correlated with the damage index comet assay (rho = 0.354, $p = 0.003$). HCB, δ-HCH, lindane, exo-heptachlor epoxide, and methyl chlorpyrifos were positively correlated with genotoxicity parameters
Arrebola et al., 2016[81]	Bolivia	*o,p′*-DDT and *p,p′*-DDE levels quantified in serum from cord blood samples collected after delivery	Newborn TSH levels	Cohort (200)	No statistically significant findings
Silva et al., 2019[118]	Brazil	Environmental and occupational exposure to agrochemicals	Chromosomal damage in peripheral blood lymphocytes and from oral epithelium samples	Cross-sectional (23)	No statistically significant findings
Cordier et al., 2015[61]	Guadeloupe	Chlordecone concentrations from cord blood samples collected at birth	TSH levels at 3 months of age	Cohort (111)	Boys from the higher exposure group showed 61.3% (95% CI: 2.0, 155.0) change in TSH levels[a]
Ayhan et al., 2021[63]	Guadeloupe	Organochlorine levels measured from umbilical cord blood samples collected at birth	Levels of TSH, FT3, FT4, IGF-1, adiponectin, leptin, DHEA, TT, and E2 hormones in children at 7 years of age	Cohort (438)	The third quartile of *in utero* chlordecone exposure relative to the lowest quartile was associated with higher TSH (β = 0.22; 95% CI: 0.01, 0.44) levels in girls and higher DHEA (β = 0.54; 95% CI: 0.08, 1.01, for boys; β = 0.36; 95% CI: 0.02, 0.71, for girls), TT (aOR = 3.22; 95% CI: 1.08, 9.6, for boys; aOR = 3.28; 95% CI: 1.32, 8.17, for girls), and DHT (aOR = 3.70; 95% CI: 1.29, 10.6, for boys; aOR = 3.20; 95% CI: 1.01, 10.2, for girls) levels[b]

Note: aOR, adjusted odds ratio; β-HCH, beta-hexaclorocyclohexane; BMI, body mass index; CI, confidence interval; δ-HCH, delta-hexachlorocyclohexane; DDE, dichlorodiphenyldichloroethylene; DDT, dichlorodiphenyltrichloroethane; DHEA, dehydroepiandrosterone; DHT, dihydrotestosterone; E2, estradiol; FT3, tri-iodothyronine; FT4, thyroxine; HCB, hexachlorobenzene; IGF-1, insulin growth-factor 1; OCPs, organochlorine pesticides; OPs, organophosphates; PCB, polychlorinated biphenyl; PYR, pyrethroids; TSH, thyroid-stimulating hormone; TT, total testosterone.

[a]Adjusted for hemolysis status, ssample storage duration, level of education, ln PCB-153 levels, ln *p,p′*-DDE levels, and total lipids.

[b]Adjusted for mothers' age at delivery, smoking during pregnancy, alcohol during pregnancy, mothers' BMI in early pregnancy, child *z*-score BMI, geographical origin, and breastfeeding depending on the outcome.

exposed to DDT, there was a decrease in the proportion of viable cells and an increase in the proportion of necrotic cells.[131] Furthermore, administrating DDT to rabbit embryos prior to the implantation stage resulted in intrauterine growth restriction,[132] and carbamate pre- and postimplantation in pregnant rats reduced fetal weight.[133] Exposure to a mixture of pesticides can lead to a decrease in birth weight, even at dose levels considerably lower than the no observed adverse effect levels of a single pesticide within the mixture.[134]

A possible mechanism involved in adverse obstetric outcomes is the functional impairment of the placenta due to oxidative stress.[117,135] Oxygen is a fundamental component for cellular metabolism, facilitating various biochemical reactions, and it is particularly crucial during the initial stages of development, where it influences key processes, such as cell differentiation, tissue growth, and organ formation.[136] Pesticides increase oxidative stress and induce the formation of oxidized lipids.[137,138] Exposure to OP pesticides leads to oxidative stress owing to decreased glutathione and increased reactive oxygen species.[139] Moreover, organochlorine pesticide residues found in the placental tissue of individuals with preterm delivery were associated with oxidative stress.[140] Further studies are needed to accurately understand the molecular mechanisms involved in oxidative stress triggered by pesticide exposure.

Other pathophysiological mechanisms should be taken into consideration. Some pesticides act as androgen-receptor antagonists, inhibitors of steroidogenic enzymes, or both, thus impairing endocrine signal pathways.[141–147] As a consequence, exposure during critical periods of development can trigger alterations in signal pathways in the tissues of the male reproductive system, causing anomalies of the external genitalia.[148–150] Anogenital distance has also been an androgen-dependent developmental biomarker applied to assess the effects of endocrine-disrupting pesticides.[151] Although outlying measurements of birth weight, birth length, and head circumference are not directly associated with a specific dysfunction or disease, these parameters are conventionally used to evaluate and

predict intrauterine development and possible alterations in growth.[152] In addition, anogenital distance is a well-established marker of androgen exposure in humans.[153] In the present review, we identified a set of studies evaluating anthropometrics parameters, including anogenital distance, penile dimensions, and the proportion of male newborns. Nonetheless, a subset of the investigations failed to incorporate standardized anthropometric measurements, such as *z*-scores for weight and length stratified by both sex and gestational age, which would facilitate a more robust interstudy comparison.

Among the studies assessing congenital anomalies, particular attention was given to male genital anomalies such as hypospadias and cryptorchidism. With the exception of one study that focused on hypospadias and had a small number of participants, the overall results indicate an association between prenatal exposure to pesticides and anomalies of the genitourinary system. Other congenital anomalies, such as anomalies in the circulatory and digestive systems, and cleft lip and palate, as well as infant mortality caused by congenital anomalies in the CNS and CVS, were also associated with prenatal exposure to pesticides. Some previous works have estimated the association by using exposure to organochlorines as a measure, which, as mentioned above, have an androgenic antagonist action.[144] However, other studies relied on indirect measures of exposure, such as parental occupation and place of residence, or employed ecological approaches. In these cases, findings are more susceptible to confounding factors and should be interpreted with caution.

Epidemiological studies have demonstrated an association between pesticide exposure and respiratory outcomes among farmworkers.[154–156] Among the studies included in the present review, we observed limited evidence concerning the association between prenatal exposure and respiratory outcomes in children.

We observed consistency among the results from studies in which the outcomes were childhood leukemia, which corresponds to the results of other studies conducted outside of LAC countries.[15,157] Despite the fact that the majority of childhood leukemia cases do not have a clear etiology,[158] it has been proposed that certain chemicals, including pesticides, exhibit toxic mechanisms that act on early hematopoietic stem and progenitor cells. These mechanisms involve topoisomerase II inhibition properties and the excessive generation of free radicals, which can lead to DNA single- and double-strand breaks.[159,160] The relationship between leukemia and pesticide exposure is still a relatively underexplored field in LAC. Given the morbidity and mortality associated with this disease, future studies aimed at understanding this relationship could potentially contribute to improving the childhood health indexes across countries from LAC.

We found a substantial body of literature evaluating the relationship between prenatal pesticide exposure and neurodevelopment. Notably, three Latin American cohorts were assessed for neurodevelopment parameters across different time points of childhood. Among the results of these studies, a positive association was reported between pesticide exposure and deficits in fine motor skills. In a similar way, tremors were reported among rat pups exposed to chlordecone,[161] as well as among human adults experiencing acute exposure.[162] According to the data available, authors have highlighted that chlordecone-induced tremors may involve multiple neurotransmitter systems.[58] Another possible explanation for these findings is the short-term visual memory measured by the novelty preference. Difficulty with recent memory was reported among the symptoms from workers intoxicated with chlorinated pesticides.[162] Memory impairment was also observed in preweaning rats exposed to chlordecone.[163,164] Specific to the findings on contrast sensitivity, researchers have proposed that contrast sensitivity may be mediated by dopaminergic-related mechanisms.[55]

Considering the aims and the inclusion criteria for this review, we did not include experimental studies. However, findings from studies examining the molecular effects of pesticide exposure on pregnant women, including genotoxicity tests, were presented.[95,117,118] In addition, other studies investigated TSH levels as potential mediators of adverse outcomes.[61,81] Although not the focus of this review, we underscore the significance of exploring the potential hazards associated with pesticide exposure during pregnancy through experimental studies, particularly those addressing placental health and development. Enhanced comprehension of how pesticides influence the physiology of pregnant women and the development of embryo and fetus, as elucidated by molecular mechanisms in *in vitro* studies and animal models, holds considerable value for interpreting findings from observational human studies.

Taking into account the body of studies presented, we would like to highlight some challenges we observed among these studies. The first challenge is related to the complexity of directly assessing pesticide exposure. Occupational exposure, proximity to cultivation areas, self-reported pesticide application, and rural/urban residence were among the indirect measures used as exposure. A limited number of studies (55.0%) have directly assessed exposure biomarkers in umbilical cord, maternal blood, or urine samples. A second challenge for researchers on this topic is the variations in the timing of sample collection (e.g., trimester of pregnancy) across these studies could potentially yield disparate outcomes, which may account for divergences observed among the different studies' results. The third challenge is that some outcomes are rarely observed, and prospective research on larger sample populations may be necessary to draw more meaningful results.

We have identified gaps in the literature that should be explored in future research. Considering that new pesticides are released for application each year, we emphasize the need to examine the effects resulting from prenatal exposure to pesticides other than organochlorines. Particularly, attention should be given to the effects of pesticides banned in high-income countries but widely used in Latin America. It is worth mentioning that the present review identified studies from 13 of the 45 LAC countries, denoting a lack of research on prenatal pesticide exposure and its adverse outcomes in certain LAC countries (Anguilla, Antigua and Barbuda, Aruba, Bahamas, Barbados, Belize, British Virgin Islands, Cayman Islands, Cuba, Dominica, Dominican Republic, El Salvador, Falkland Islands (Malvinas), French Guiana, Grenada, Guyana, Haiti, Honduras, Jamaica, Martinique, Montserrat, Nicaragua, Panama, Saint Kitts and Nevis, Saint Lucia, Saint Vincent and the Grenadines, Suriname, Trinidad and Tobago, Turks and Caicos Islands, US Virgin Islands, Uruguay, and Venezuela). Local efforts to investigate the effects of prenatal pesticide exposure can lead to more effective policies. In addition, another aspect deserving attention is the impact of prenatal pesticide exposure on socially vulnerable groups. Many pregnant women exposed to pesticides in LAC hail from rural communities, indigenous populations, riverine communities, or labor under poorly regulated and protected conditions.[165] The compound effect of prenatal pesticide exposure and social vulnerability can yield even more pronounced consequences that should be studied. Furthermore, no studies covering multiple LAC countries were identified. Given the shared characteristics and unique aspects related to pesticides regularization, collaborative studies between countries can also yield new insights to enhance the health of the LAC population. Finally, there is a dearth of prospective epidemiological studies that use a high-quality design and statistical approach. In this context, we advocate for the advancement of research endeavors that directly measure exposure levels through biomarkers in maternal blood or urine samples, among other

methods. In addition, it is imperative to evaluate exposure during each trimester and its association with adverse outcomes. Prospective studies offer a viable approach to accomplishing these practices, facilitating more precise and wide collection of data, and consequently yielding more robust findings. Given the unique characteristics of LAC, gaining a deeper understanding of the effects of environmental factors on maternal and child health is an area that demands robust scientific investment. This can significantly contribute to formulating more effective public policies for this population.

Our study has some noteworthy aspects. It synthesizes the scientific output from observational human studies concerning prenatal exposure to pesticides and adverse outcomes in LAC, a region characterized by lax regulatory frameworks and consequent high pesticide consumption. Our methodology enabled us to identify and assess studies not only in English but also in Portuguese and Spanish, the primary languages of the region. This approach ensured the inclusion of articles from both international and local journals. By focusing on prenatal exposure, our study consistently captured scientific output on teratology, a field of research essential for informing regulatory decisions concerning products with potential human health hazards. Our study also has some limitations. We did not systematically evaluate the quality of the studies or summarize their results quantitatively, because this was not the purpose of the scoping review. Our search strategy was defined based on three main terms: pesticides, pregnancy, and Latin America. We did not include the names of specific pesticides, which may have limited the articles recovered in the search. However, we included MeSH terms to expand the search strategy to incorporate related terms into the searches. Furthermore, our study was restricted to observational studies involving human participants. This decision was made to assess the real-life impact of pesticide use across LAC. However, it inevitably excluded numerous experimental studies conducted within the region on this topic. We could search for and read articles only in Portuguese, Spanish, and English. However, there may be scientific output in other languages within the region that was not included in this review.

In summary, we identified studies conducted in LAC that evaluated prenatal exposure to pesticides and this exposure's potential associations with adverse obstetric and neonatal outcomes, congenital anomalies, anthropometric parameters, neurodevelopment, childhood leukemia, and respiratory infections. In addition, we identified a limited number of studies assessing molecular effects. Given that a significant portion of the LAC populations are exposed to pesticides and considering the diverse range of adverse effects reported in the scientific literature due to pesticide exposure, continued research into the risks of prenatal exposure is essential. There are substantial gaps in the existing knowledge, particularly concerning groups of pesticides beyond organochlorines. It is crucial to consider the unique characteristics of the LAC region, especially regarding investigating risks associated with pesticides that are banned in some countries but permitted in LAC.

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