

Prenatal Organophosphate Pesticide Exposure and Neurodevelopmental Outcomes at 6 Months among Children of Smallholder Horticultural Growers in Exposed and Comparison Districts of the Southern Highlands of Tanzania

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Abstract

Background

Pesticide exposure during pregnancy has been associated with adverse neurodevelopmental outcomes in children, yet limited data exist from resource-limited agricultural settings in sub-Saharan Africa. This study aimed to assess the association between maternal pesticide exposure and neurodevelopmental outcomes among children aged 6 months in Tanzania.

Methods

A comparative cross-sectional study was conducted among 337 mother–child pairs recruited from four districts in Tanzania: Kilolo District Council, Makambako Town Council (exposed districts), and Njombe Town Council and Iringa Municipal Council (comparison districts). Maternal pesticide exposure was assessed through Cholinesterase activity inhibition using Test-mate ChE Cholinesterase Test System (Model 400), with exposure levels categorized based on a cut-off value of 24.5 µg/L. Child neurodevelopment was evaluated using the Malawi Developmental Assessment Tool (MDAT). Data were collected using structured questionnaires. Univariate and multivariate logistic regression analyses were performed to identify factors associated with neurodevelopmental outcomes, with adjusted odds ratios (AOR) and 95% confidence intervals (CI) calculated.

Results

Among the 337 participants, 220 (65.3%) were from exposed districts and 117 (34.7%) from comparison districts. The majority of children demonstrated normal development across all domains. Pesticide use practices revealed heavy dependence on organophosphate. About, 27.6% of mothers had high pesticide exposure. In univariate analysis, maternal marital status was the only factor significantly associated with composite neurodevelopment; children of married mothers had 71% lower odds of delayed/mild neurodevelopment compared with those of single or divorced mothers (OR = 0.29, 95% CI: 0.11–0.80; $p = 0.017$). This association remained significant after adjustment for confounders (AOR = 0.25, 95% CI: 0.09–0.71; $p = 0.009$).

Conclusion

This study found widespread use of hazardous pesticides and high maternal pesticide exposure in horticultural communities. Children of mothers with moderate-to-high pesticide exposure had an increased risk of fine motor developmental delay, highlighting the potential impact of prenatal pesticide exposure on child neurodevelopment. These findings emphasize the need to strengthen pesticide safety practices, promote safer pest management strategies.

1.0 Introduction

Agricultural intensification in Tanzania has driven increased pesticide use, raising health concerns. Agriculture is central to food security and rural livelihoods (FAO, 2022; World Bank, 2021), and intensification has increased reliance on synthetic pesticides (Maldani, 2017). This growth in pesticide use has created new pathways of community exposure, particularly in agriculturally intensive regions.

The Southern Highlands of Tanzania is a site of especially intensive, self-reinforcing pesticide use. The "pesticide treadmill" phenomenon drives escalating chemical application as pest resistance develops (Carvalho, 2017), and the region is among Tanzania's most pesticide-intensive due to widespread tomato and vegetable farming (Kariathi, 2021). This makes the Southern Highlands a critical setting for examining pesticide-related health risks.

Pregnant women and fetuses are highly vulnerable to pesticide exposure. Prenatal neurodevelopment is sensitive to environmental disruption, and even low-level exposure during this period may affect developmental trajectories (Grandjean & Landrigan, 2014). Exposure occurs through occupational contact, environmental drift, household dust, and contaminated food or clothing (Kariathi, 2021). These overlapping pathways make maternal-fetal exposure an important but understudied concern.

Organophosphate (OP) and carbamate insecticides, widely used in smallholder horticulture, increase exposure risk via placental transfer. Their low cost drives frequent, poorly regulated use (Maldani, 2017); pregnant women absorb these compounds through inhalation, dermal contact, or ingestion, exposing the fetus during a critical developmental window (Mudyanselage et al., 2023). Economic reliance on these insecticides translates directly into fetal exposure risk.

OP and carbamate toxicity operates through both cholinergic and non-cholinergic mechanisms. These insecticides inhibit acetylcholinesterase (AChE) (Aroniadou-Anderjaska et al., 2023); emerging evidence also implicates oxidative stress, disrupted calcium signaling, impaired axonal transport, and neuroinflammation as non-cholinergic contributors to neurotoxicity (Naughton & Terry, 2018; Mudyanselage et al., 2023). Multiple toxic pathways support the biological plausibility of subtle neurodevelopmental harm even without acute poisoning.

Epidemiological evidence links prenatal OP exposure to poorer child neurodevelopment. In the CHAMACOS cohort, prenatal dialkyl phosphate metabolites were associated with lower mental development scores at 24 months (Eskenazi et al., 2007) and reduced full-scale IQ at age 7 (Bouchard et al., 2011); similar associations with neurodevelopment and cognition have been reported in other cohorts (Engel et al., 2007; Rauh et al., 2006). Despite a consistent direction of effect across cohorts, methodological heterogeneity limits causal certainty.

Methodological limitations, especially in LMIC settings, constrain confidence in existing findings. Reliance on single, non-specific urine biomarkers and inconsistent developmental tools weakens exposure-outcome links, particularly where biomonitoring resources are scarce (Kariathi, 2021). These gaps are most severe for sub-Saharan African smallholder communities and largely unexamined at early milestones like 6 months of age.

This study addresses that evidence gap by examines prenatal OP exposure and neurodevelopmental outcomes at 6 months among children of smallholder growers in the Southern Highlands of Tanzania, combining biomarker data with standardized developmental assessment. The findings aim to inform exposure-reduction strategies and policy to protect maternal and child health in vulnerable farming communities.

2.0 Method

2.1 Study Area and settings

The study was carried out in Iringa and Njombe regions, located in the Southern Highlands Zone of Tanzania, an area characterized by intensive smallholder and horticultural agriculture (including tomato, maize, tea and vegetable production) and by localized small-scale mining activity. Within Iringa region, participants were drawn from Iringa Municipal Council and Kilolo District Council; within Njombe region, participants were drawn from Makambako Town

Council and Njombe Town Council. Data were collected from reproductive and child health (RCH)/infant welfare clinics at three tiers of the public health system dispensaries, health centers and hospitals in each district, reflecting the routine platform through which infants attend for 6-month immunization and growth-monitoring services.

2.2 Study Design and Setting

This was a comparative cross-sectional study conducted in four districts of Tanzania: Kilolo District Council, Makambako Town Council (exposed districts), and Njombe Town Council and Iringa Municipal Council (comparison districts). The study areas were selected based on their agricultural practices, with Kilolo and Makambako representing areas with intensive horticultural farming and extensive pesticide use, while Njombe and Iringa served as comparison areas with relatively lower agricultural pesticide application. The study was conducted to assess the association between maternal pesticide exposure and neurodevelopmental outcomes in children aged 6 months.

2.2 Study Population

The study population comprised mother–child pairs residing in the selected districts. Mothers were included if they had been residents in the study area for at least one year prior to pregnancy, had a child aged 6 months at the time of recruitment, and provided informed consent. Mother–child pairs were excluded if the child had a diagnosed congenital anomaly, severe birth complications, or a known neurological disorder that could independently affect neurodevelopmental outcomes.

1.5 Sample Size Determination and Sampling Procedure

The sample size was calculated using the formula for comparative cross-sectional studies, assuming a 95% confidence level, 80% power, and an expected difference in neurodevelopmental delay of 15% between exposed and unexposed groups. A design effect of 1.5 was applied to account for the cluster sampling approach, and an additional 10% was added for non-response, yielding a minimum sample size of 330 mother–child pairs.

A multi-stage sampling approach was used: districts were treated as strata, and study participants were drawn proportionally from each stratum to reflect the relative population size of each council, after which systematic random sampling was applied to identify individual participants at the household or health-facility level. A multi-stage sampling approach was applied. First, Iringa and Njombe regions were purposively selected on the basis of their predominantly agricultural economies and documented use of agrochemicals. Second, within each region, districts and health facilities were selected to ensure representation across all three facility levels (dispensary, health centre and hospital). Third, the calculated sample size was allocated proportionally across facilities according to expected infant attendance volume. Finally, eligible mother–infant pairs attending each facility for the 6-month clinic visit during the data-collection period were consecutively/systematically recruited until the facility-specific sample quota was reached.

2.4 Data Collection Procedures

Structured questionnaires were administered by trained research assistants through face-to-face interviews with mothers. The questionnaire captured information on maternal socio-demographic characteristics (age, marital status, education level, occupation), obstetric history (parity, gestational age at delivery, mode of delivery, place of birth), maternal health status (BMI, anemia), and child characteristics (sex, age, Apgar score). Maternal BMI was calculated from measured weight and height and categorized according to WHO criteria: underweight ($< 18.5 \text{ kg/m}^2$), normal ($18.5\text{--}24.9 \text{ kg/m}^2$), and overweight ($\geq 25 \text{ kg/m}^2$). Maternal anemia status was determined by measuring hemoglobin levels during the study visit.

2.4.3 Neurodevelopmental Assessment

Child neurodevelopment was determined using the Malawi Developmental Assessment Tool (MDAT), a culturally validated and standardized tool designed for use in African settings (Gladstone et al. 2010). The MDAT evaluates performance across four developmental domains: gross motor, fine motor, language, and social–emotional functioning, and has been used in translated and used in Tanzania among the Swahili speakers with a success (Nyanza et al. 2021).

Neurodevelopment was assessed at 12 months of age (± 2 weeks), with age determined from health cards and recorded in months with decimal precision. Assessments were conducted in home or community settings using standardized, low-cost materials (e.g., pegs, bricks, soap), and children were tested starting at their exact age. Administration followed standard stopping rules, with testing in each domain discontinued after seven consecutive failed items or upon completion of all items. Neurodevelopment was measured using the Malawi Developmental Assessment Tool (MDAT), a validated and culturally appropriate instrument for low-resource settings in sub-Saharan Africa, comprising 136–138 items across four domains: gross motor (36 items), fine motor (36 items), language (36 items), and social (30 items).

Trained assessors, certified through a 1-week training program (mdat.org.uk) and demonstrating inter-rater reliability ($\kappa > 0.75$), administered the tool through direct observation; for fewer than 10% of items where children were uncooperative, parent report was used. Each item was scored as pass (1) or fail/"don't know" (0), and raw domain scores were calculated as the sum of passed items. Age-standardized developmental z-scores (DAZ) for total and domain-specific performance were computed using the MDAT Shiny application (v1.1) based on Malawi reference norms ($n = 1,426$ healthy children). Z-scores ≥ -1 SD were considered normal, between -1 and -2 SD as mild developmental delay, and < -2 SD as significant developmental delay. In addition, MDAT screening criteria were applied, whereby failure of two or more items that at least 90% of reference children pass (sensitivity 97%, specificity 82%) flagged potential delay; failure of more than two items in more than one domain was considered a "red flag" warranting further assessment or referral rather than a definitive diagnosis of impairment. The primary outcome was the fine motor DAZ score, selected as a proxy for cognitive development and due to its sensitivity to environmental exposures such as pesticides and nutrition.

2.5 Variables and Measurements

2.5.1 Independent Variables

2.4.2 Pesticide Exposure Assessment

Maternal pesticide exposure was assessed through two complementary approaches, First using a structured inventory which was administered to assess the types of pesticides used by farmers, frequency of application, mixing practices, and personal protective equipment use. Pesticides were classified according to the International Resistance Action Committee (IRAC), Fungicide Resistance Action Committee (FRAC), and Herbicide Resistance Action Committee (HRAC) classification systems. Chemical combinations frequently used by farmers were documented and analyzed.

Second using biological biomarkers. Exposure to organophosphate pesticides was assessed through biological monitoring using measurement of acetylcholinesterase (AChE) activity in blood samples collected from mothers. Acetylcholinesterase plays a critical role in the nervous system by breaking down acetylcholine at synaptic junctions. Organophosphate pesticides inhibit this enzyme, leading to accumulation of acetylcholine and disruption of normal

nerve transmission. Blood samples were analyzed using the EQM Test-Mate Cholinesterase system (Model 400, EQM Research, Inc., Cincinnati, OH, USA, which requires only 10 µL of blood obtained via fingerstick sampling. The assay can be completed in less than four minutes, enabling rapid evaluation of pesticide exposure levels. The assay was based on the Ellman method, wherein acetylthiocholine (AcTC) or butyrylthiocholine (BuTC) is hydrolyzed by AChE, respectively, producing thiocholine, which subsequently reacts with 5,5'-dithiobis-(2-nitrobenzoic acid) (DTNB) to form a yellow chromophore measured spectrophotometrically at 450 nm. The rate of color formation is directly proportional to cholinesterase activity.

The assay procedure was performed following manufacturer's protocols. Briefly, assay tubes containing buffer solution were blanked in the photometric analyzer. Blood samples (10 µL) were added to the tubes and vigorously shaken for 15 seconds to disperse the sample. Following a second blank reading, reconstituted reagent (prepared by dissolving lyophilized substrate in three drops of distilled water) was added to the assay tubes using transfer pipettes. The tubes were gently inverted for 5 seconds to mix, then incubated for approximately 80 seconds, followed by absorbance readings.

Results were automatically adjusted for reagent blank activity (approximately 15% correction), temperature-normalized to 25°C using the built-in temperature sensor, and hemoglobin-corrected for AChE (expressed as U/g hemoglobin). Hemoglobin concentration was determined from the same sample. The analyzer displayed two results for AChE mode (U/mL, % normal, hemoglobin, hemoglobin % normal, quotient in U/g, quotient % normal). Erythrocyte AChE activity expressed in units per gram of haemoglobin (U/g Hb). Results were classified into three exposure categories using standard kit reference cut-offs: normal (> 31.3 U/g Hb), inhibited/moderate exposure (24.5–31.3 U/g Hb), and severely inhibited/high exposure (< 24.5 U/g Hb). The photometric analyzer was factory-calibrated and required no additional calibration. Quality control was performed using either unexposed operator blood (preferred, due to intraindividual variability < 5% per week) or refrigerated EDTA-anticoagulated venipuncture blood (stable for at least one month). Controls were run on each day of testing.

3.6.3 Confounding Factors Assessment

Several confounding variables known to influence neurodevelopment were measured and controlled for during statistical analysis. These included maternal lifestyle factors, child nutritional status, hereditary factors, anemia, stimulation within the home environment, and prenatal care. Data on alcohol consumption, smoking, and drug use during pregnancy were collected using items adapted from the WHO verbal autopsy tool. Nutritional status was assessed using WHO Anthro software, while stimulation within the home environment was evaluated using the HOME inventory. Hemoglobin levels were measured to assess maternal anemia, and Apgar scores recorded at birth were used as baseline indicators of newborn health.

3.9 Nutritional and Health Status Assessment

Nutritional status was evaluated using WHO Child Growth Standards through WHO Anthro software. Anthropometric measurements including child weight, height, age, and sex were used to calculate Weight-for-Age (WAZ) and Height-for-Age (HAZ) z-scores. Child anemia status was determined using WHO hemoglobin cut-off values, and Apgar scores were used to assess newborn health status at birth.

2.6 Data Quality Assurance

Several measures were implemented to ensure data quality. Research assistants underwent intensive training for one week on questionnaire administration, pesticide exposure assessment protocols, and neurodevelopmental assessment using the MDAT. Inter-rater reliability was assessed during training, with a kappa statistic of ≥ 0.80 achieved for neurodevelopmental assessments. The structured questionnaire was pre-tested in a pilot study involving

30 mother–child pairs from a nearby district not included in the main study, and modifications were made to improve clarity and comprehension.

3.7.4 Pesticide Use Pattern Analysis

Pesticide use data were analyzed to identify patterns in pesticide application and chemical class distribution. Trade names reported by farmers were verified using pesticide databases and product labels. Each pesticide product was converted to its corresponding active ingredient and classified according to international pesticide classification systems including IRAC, FRAC, and HRAC. Descriptive analyses were conducted to determine the frequency of pesticide active ingredients and identify common pesticide mixtures used by farmers. Resistance risk levels were categorized based on frequency of pesticide class use.

2.7 Data Analysis

Data analysis was performed using STATA version 17.0 .Data were collected using a structured questionnaire capturing socio-demographic characteristics alongside the laboratory-confirmed exposure classification, and were entered and cleaned in Microsoft Excel before analysis in excel. Descriptive statistics (frequencies and percentages) were used to summarise the distribution of exposure categories overall and by district. Cross-tabulation was performed to generate row percentages (distribution of exposure categories within each district) and column percentages (contribution of each district to the overall exposure category).The distribution of maternal pesticide residue levels across districts was visualized using box plots. Chi-square tests of association were used to compare categorical variables between the exposed and comparison districts, with $p < 0.05$ considered statistically significant.

Univariate logistic regression analysis was performed to examine the association between each independent variable and the neurodevelopmental outcomes (gross motor, fine motor, language, social, and composite neurodevelopment). Odds ratios (OR) with 95% confidence intervals (CI) were calculated to estimate the strength of associations. Variables with $p < 0.20$ in the univariate analysis, along with clinically important variables (including maternal pesticide exposure), were considered for inclusion in the multivariate logistic regression model for composite neurodevelopment.

Multivariate logistic regression analysis was performed to adjust for potential confounders, with maternal marital status, maternal BMI, maternal anemia, and maternal pesticide exposure entered into the model. Adjusted odds ratios (AOR) with 95% CI were calculated, and $p < 0.05$ was considered statistically significant. Model fit was assessed using the Hosmer-Lemeshow goodness-of-fit test. All analyses were conducted with appropriate weighting to account for the sampling design.

2.8 Ethical Considerations

Ethical approval

was obtained from the Joint Catholic University of Health and Allied Sciences and Bugando Medical Centre Research Ethics and Review Committee (*Certificate# CREC/866/2024*). Written informed consent was obtained from all participants in the Antenatal Care (ANC) unit. Confidentiality and anonymity were rigorously maintained, and participants were informed of their right to withdraw from the study at any point without any negative consequences. Clinical trial number, not applicable. The study was adhered to the ethical principles of respect for persons, beneficence, and justice, as well as the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) guidelines for reporting observational studies.

2.9 Limitations

This study has several limitations such as the use of a single exposure measurement time point also fails to capture chronic exposure patterns across pregnancy, while the developmental assessment conducted at one time point may not reflect the full trajectory of neurodevelopment. Statistical concerns include small subgroup sizes, which produced wide confidence intervals indicating considerable instability, and the lack of correction for multiple comparisons across four developmental domains and numerous variables, raising the possibility that some significant findings reflect chance. The developmental assessment tool may also have variable sensitivity across domains, potentially explaining why associations appeared in some areas but not others. An inconsistency in sample sizes across tables should be verified, and the generalisability of findings is limited to the two horticultural districts studied, with uncertain applicability to other agricultural zones or urban populations. Despite these limitations, this study provides valuable information on the relationship between maternal pesticide exposure and child neurodevelopment in a resource-limited agricultural setting. Future research should consider longitudinal designs to assess exposure dynamics over time and incorporate detailed exposure pathway assessments to identify the most critical intervention points.

3.0 Results

3.1 Social demographic characteristics

A total of 337 mother–child pairs were enrolled in the study, including 220 (65.3%) from the exposed district (Kilolo and Makambako) and 117 (34.7%) from the comparison district (Njombe town and Iringa Municipal). Overall, 58.8% of the children were female, and most mothers had attained primary education (52.2%). Most deliveries occurred in health facilities (78.9%), 65.9% of mothers were engaged in horticultural activities. More than half of the mothers (51.3%) were overweight, 34.1% and most participants had normal maternal pesticide residue levels (61.7%), although 27.6% had high exposure, with slightly higher prevalence in the exposed district. Table 1 below.

Table 1
Social demographic characteristics (N = 337)

	District		
	Comparison District	Exposed District	Total, 337 (100.0)
Gender child			
Female	65 (55.6)	133 (60.5)	198 (58.8)
Male	52 (44.4)	87 (39.5)	139 (41.2)
Marital Status			
Single/Divorced	3 (2.6)	18 (8.2)	21 (6.2)
Married	114 (97.4)	202 (91.8)	316 (93.8)
Education			
No Education/Illiterate	7 (6.0)	26 (11.8)	33 (9.8)
Primary School	57 (48.7)	119 (54.1)	176 (52.2)
High/Secondary School	36 (30.8)	61 (27.7)	97 (28.8)
Tertiary Education	17 (14.5)	14 (6.4)	31 (9.2)
Birthplace			
Health Facility	94 (80.3)	172 (78.2)	266 (78.9)
Home	23 (19.7)	48 (21.8)	71 (21.1)
Occupation			
Non-Horticultural	61 (52.1)	54 (24.5)	115 (34.1)
Horticultural	56 (47.9)	166 (75.5)	222 (65.9)
Medication prior pregnancy?			
No	44 (37.6)	73 (33.2)	117 (34.7)
Yes	73 (62.4)	147 (66.8)	220 (65.3)
Method of delivering			
Assisted Vaginal Delivery	13 (11.1)	30 (13.6)	43 (12.8)
C-Section	40 (34.2)	56 (25.5)	96 (28.5)
Natural/Unassisted	64 (54.7)	134 (60.9)	198 (58.8)
Delivery terms			
Full gestation	101 (86.3)	197 (89.5)	298 (88.4)
Not full gestation	16 (13.7)	23 (10.5)	39 (11.6)
Maternal BMI			
Normal BMI	23 (19.7)	26 (11.8)	49 (14.5)

	District		
	Comparison District	Exposed District	Total, 337 (100.0)
Underweight	41 (35.0)	74 (33.6)	115 (34.1)
Over weight	53 (45.3)	120 (54.5)	173 (51.3)
Anemia (mHgb)			
Normal	91 (77.8)	155 (70.5)	246 (73.0)
Anemic	26 (22.2)	65 (29.5)	91 (27.0)
Maternal_pesticides_residual			
Normal	75 (64.1)	133 (60.5)	208 (61.7)
Moderate Exposure	11 (9.4)	25 (11.4)	36 (10.7)
High Exposure	31 (26.5)	62 (28.2)	93 (27.6)
Mining			
No	111 (94.9)	211 (95.9)	322 (95.3)
Yes	6 (5.1)	9 (4.1)	15 (4.7)
FEFO-before conception			
No	1 (0.9)	1 (0.5)	2 (0.6)
Yes	116 (99.1)	219 (99.5)	335 (99.4)
Smoke			
No	116 (99.1)	219 (99.5)	335 (99.4)
Yes	1 (0.9)	1 (0.5)	2 (0.6)
Alcohol			
No	101 (86.3)	180 (81.8)	281 (83.4)
Yes	16 (13.7)	40 (18.2)	56 (16.6)
Eating soil			
No	84 (71.8)	181 (82.3)	265 (78.6)
Yes	33 (28.2)	39 (17.7)	72 (21.4)
Is this child your first Pregnancy?			
No	78 (66.7)	118 (53.6)	196 (58.2)
Yes	39 (33.3)	102 (46.4)	141 (41.8)
Health Facility Level			
Dispensary	27 (23.1)	96 (43.6)	123 (36.5)
Health Centre	72 (61.5)	65 (29.5)	137 (40.7)
Hospital	18 (15.4)	59 (26.8)	77 (22.8)

	District		
	Comparison District	Exposed District	Total, 337 (100.0)
Apgar Score			
Normal	60 (51.3)	181 (82.3)	241 (71.5)
Abnormal	57 (48.7)	39 (17.7)	96 (28.5)

3.2 Types of pesticides use

Analysis of pesticide active ingredients according to the International Resistance Action Committee (IRAC), Fungicide Resistance Action Committee (FRAC), and Herbicide Resistance Action Committee (HRAC) classification systems revealed a predominance of a few major chemical groups in farmer pesticide use practices. The majority of insecticides belonged to IRAC Groups 1B, 3A, 4A, and 28, indicating heavy dependence on organophosphate, pyrethroid, neonicotinoid, and diamide insecticides, respectively. Organophosphate insecticides, particularly chlorpyrifos and profenofos (IRAC Group 1B), were among the most frequently reported compounds and were commonly applied either alone or in combination with other insecticides. Pyrethroid insecticides such as lambda-cyhalothrin and cypermethrin (IRAC Group 3A) were also widely used across the surveyed farms. The repeated use of organophosphates, pyrethroids, neonicotinoids, diamides, and multi-site fungicides may contribute to increased selection pressure for pesticide resistance, while also raising concerns regarding environmental contamination and impacts on non-target organisms. Table 2 below.

Table 2
showing pesticides classification

Active Ingredient / Product	Chemical Class	Classification System	Mode of Action Group
Imidacloprid	Neonicotinoid insecticide	IRAC	Group 4A – Nicotinic acetylcholine receptor (nAChR) modulators
Chlorpyrifos	Organophosphate insecticide	IRAC	Group 1B – Acetylcholinesterase inhibitors
Profenofos	Organophosphate insecticide	IRAC	Group 1B – Acetylcholinesterase inhibitors
Lambda-cyhalothrin	Pyrethroid insecticide	IRAC	Group 3A – Sodium channel modulators
Cypermethrin	Pyrethroid insecticide	IRAC	Group 3A – Sodium channel modulators
Flubendiamide	Diamide insecticide	IRAC	Group 28 – Ryanodine receptor modulators
Spirotetramat	Tetramic acid derivative insecticide	IRAC	Group 23 – Lipid biosynthesis inhibitors
Emamectin benzoate	Avermectin insecticide	IRAC	Group 6 – Glutamate-gated chloride channel activators
Abamectin	Avermectin insecticide	IRAC	Group 6 – Glutamate-gated chloride channel activators
Carbofuran	Carbamate insecticide	IRAC	Group 1A – Acetylcholinesterase inhibitors
Mancozeb	Dithiocarbamate fungicide	FRAC	Group M03 – Multi-site activity
Metalaxyl	Phenylamide fungicide	FRAC	Group 4 – RNA polymerase I inhibitors
Chlorothalonil	Chloronitrile fungicide	FRAC	Group M05 – Multi-site activity
Carbendazim	Benzimidazole fungicide	FRAC	Group 1 – β -tubulin assembly inhibitors
Copper hydroxide	Copper compound fungicide	FRAC	Group M01 – Multi-site contact activity
Glyphosate (Roundup)	Glycine herbicide	HRAC	Group 9 – EPSP synthase inhibitors

Most of the farmers use a cocktail mixture of pesticides, thus in most of the areas a mixture containing organophosphate was most prominent as compared to others. The most frequently reported combination was neonicotinoid insecticide + organophosphate insecticide, accounting for 34 records (24.3%). This was followed by dithiocarbamate fungicide + organophosphate insecticide + pyrethroid insecticide with 20 records (14.3%). Thus, results indicate a high dependence on mixed pesticide applications involving insecticides and fungicides from multiple chemical classes. Figure 1.

3.2: Maternal Pesticides exposure levels

Overall, moderate exposure was the most common category, accounting for 208 participants (61.7%), followed by high exposure with 93 participants (27.6%), while only 36 participants (10.7%) were classified as having normal exposure. At the district level, Makambako Town contributed the largest proportion of the study population (115

participants; 34.1%) and also accounted for the highest share of participants with high exposure (36 participants; 31.3% of the district population, representing 38.7% of all highly exposed participants). Kilolo DC had the second-largest sample (105 participants; 31.2%). Within this district, 26 participants (24.8%) were classified as having high exposure, 62 (59.0%) had moderate exposure, and 17 (16.2%) had normal exposure. Table 3 below.

Table 3
Maternal Pesticide Residue Distribution by District

District	High Exposure n (Row%; Col%)	Moderate Exposure n (Row%; Col%)	Normal n (Row%; Col%)	Total n (%)
Iringa MC	15 (26.3; 16.1)	38 (66.7; 18.3)	4 (7.0; 11.1)	57 (16.9)
Kilolo DC	26 (24.8; 28.0)	62 (59.0; 29.8)	17 (16.2; 47.2)	105 (31.2)
Makambako Town	36 (31.3; 38.7)	71 (61.7; 34.1)	8 (7.0; 22.2)	115 (34.1)
Njombe Town	16 (26.7; 17.2)	37 (61.7; 17.8)	7 (11.7; 19.4)	60 (17.8)
Total	93 (27.6; 100.0)	208 (61.7; 100.0)	36 (10.7; 100.0)	337 (100.0)

Row % = proportion within district; Col % = proportion within exposure category; χ^2 test of association, $p < 0.05$ considered statistically significant.

Overall, maternal pesticide residual levels were above the cut-off value of 24.5 across all four districts, indicating that the majority of mothers had high pesticide residual levels. Kilolo DC recorded the highest median residual level (27.0), Makambako Town exhibited the greatest variability in residual levels, while isolated low outliers were observed in Iringa MC and Kilolo DC, and high outliers in Kilolo DC and Njombe Town. Figure 2

3.3: Neurodevelopment outcomes

Majority of children demonstrated normal development across all neurodevelopmental domains. Gross motor development was normal in 95.5% of children, with 4.5% showing delayed or mild developmental delay. Similarly, 94.7% had normal fine motor development, while 5.3% exhibited delayed or mild development. Language development showed the highest proportion of normal outcomes, with 97.0% of children classified as having normal development and 3.0% experiencing delayed or mild development. Social development was also predominantly normal (97.3%), with only 2.7% of children showing developmental delay. Overall, 88.4% of children had normal composite neurodevelopment, whereas 11.6% were classified as having delayed or mild neurodevelopment, Table 4.

Table 4
Distribution of Neurodevelopmental Outcomes Among Children in Comparison and Exposed Districts

Neurodevelopmental Domain	Outcome	Comparison District	Exposed District	Overall (N = 337)
Gross motor	Normal Development	112 (95.7)	210 (95.5)	322 (95.5)
	Delayed/Mild Development	5 (4.3)	10 (4.5)	15 (4.5)
Fine motor	Normal Development	112 (95.7)	207 (94.1)	319 (94.7)
	Delayed/Mild Development	5 (4.3)	13 (5.9)	18 (5.3)
Language	Normal Development	111 (94.9)	216 (98.2)	327 (97.0)
	Delayed/Mild Development	6 (5.1)	4 (1.8)	10 (3.0)
Social	Normal Development	114 (97.4)	214 (97.3)	328 (97.3)
	Delayed/Mild Development	3 (2.6)	6 (2.7)	9 (2.7)
Composite neurodevelopment	Normal Development	105 (89.7)	193 (87.7)	298 (88.4)
	Delayed/Mild Development	12 (10.3)	27 (12.3)	39 (11.6)

3.4 Domain-specific Neurodevelopment determinants in the comparison district

In the comparison district, child sex was the only variable significantly associated with a developmental outcome: male children had almost four times higher odds of fine motor delay than female children (OR = 3.72, 95% CI [1.11–12.49], $p = .033$). Male sex was also associated with elevated, though non-significant, odds of gross motor delay (OR = 1.56, 95% CI [0.44–5.56]) and language delay (OR = 1.54, 95% CI [0.21–11.15]). A borderline protective association was observed between underweight maternal BMI and gross motor delay (OR = 0.11, 95% CI [0.01–1.06], $p = .056$), although this did not reach conventional significance, Table 5.

Table 5

Univariate logistic regression analysis of factors associated with developmental delays in Gross Motor, Fine Motor, Language, and Social domains among children Comparison, District.

Variable	Category	Gross Motor OR [95% CI]	P-value	Fine Motor OR [95% CI]	P-value	Language OR [95% CI]	P-value	Social OR [95% CI]	P-value
Maternal age [years]	Continuous	1.06 [0.97–1.17]	0.201	0.93 [0.83–1.04]	0.202	0.95 [0.85–1.07]	0.406	0.94 [0.81–1.10]	0.470
Child sex	Female	1		1		1		1	
	Male	1.56 [0.44–5.56]	0.492	3.72 [1.11–12.49]	0.033	1.54 [0.21–11.15]	0.668	0.76 [0.14–4.23]	0.753
Maternal education	Literate	1		1		1		1	
	Illiterate	2.13 [0.44–10.30]	0.346	1.17 [0.35–3.95]	0.795	1.56 [0.16–15.29]	0.701	1.04 [0.19–5.79]	0.968
Place of birth	Health facility	1		1		1		1	
	Home	0.89 [0.18–4.34]	0.887	0.28 [0.04–2.24]	0.232	3.70 [0.51–26.95]	0.197	0.71 [0.08–6.23]	0.758
Maternal occupation	Other occupations	1		1		—	—	1	
	Horticultural	3.04 [0.38–24.55]	0.297	1.09 [0.29–4.11]	0.899	—	—	0.64 [0.11–3.61]	0.615
Medication during pregnancy	No	1		1		1		—	—
	Yes	0.86 [0.22–3.42]	0.827	1.28 [0.40–4.05]	0.677	2.04 [0.28–14.80]	0.480	—	—
Delivery mode	Caesarean section	1		1		1		1	
	Natural/Unassisted	0.63 [0.18–2.24]	0.473	0.73 [0.24–2.27]	0.592	1.95 [0.20–19.02]	0.567	3.29 [0.38–28.69]	0.280
Delivery term	Full term	—	—	1		—	—	1	

Variable	Category	Gross Motor OR [95% CI]	P-value	Fine Motor OR [95% CI]	P-value	Language OR [95% CI]	P-value	Social OR [95% CI]	P-value
	Preterm [Not full term]	—	—	1.61 [0.33–7.76]	0.553	—	—	1.75 [0.19–15.63]	0.618
BMI category	Normal weight	1		1		—	—	1	
	Underweight	0.11 [0.01–1.06]	0.056	0.87 [0.16–4.78]	0.872	—	—	0.33 [0.04–2.50]	0.285
	Overweight	0.40 [0.09–1.73]	0.222	0.63 [0.12–3.32]	0.587	—	—	0.20 [0.03–1.52]	0.120
Maternal residual exposure	Low exposure	1		1		1		1	
	Moderate exposure	1.02 [0.28–3.72]	0.976	1.33 [0.43–4.11]	0.616	0.50 [0.05–4.92]	0.556	0.76 [0.14–4.23]	0.753

3.2 Domain-specific determinants in the exposure district

In the exposure district, increasing maternal age was associated with significantly lower odds of both gross motor delay (OR = 0.79, 95% CI [0.41–0.93], $p = .046$) and language delay (OR = 0.75, 95% CI [0.47–0.98], $p = .033$), indicating a protective effect of advancing maternal age in this district. The clearest risk factor identified in the entire analysis was maternal residual pesticide exposure: children of mothers classified as having moderate-to-high residual exposure had almost eight-fold higher odds of fine motor delay compared with those in the low-exposure group (OR = 7.79, 95% CI [1.84–72.15], $p = .041$). Table 6.

Table 6

Univariate logistic regression analysis of factors associated with developmental delays in Gross Motor, Fine Motor, Language, and Social domains among children exposure, District.

Variable	Category	Gross Motor OR [95% CI]	P- value	Fine Motor OR [95% CI]	P- value	Language OR [95% CI]	P- value	Social OR [95% CI]	P- value
Maternal age [years]	Continuous	0.79 [0.41–0.93]	0.046	0.92 [0.66–1.12]	0.413	0.75 [0.47–0.98]	0.033	1 [0.80–1.25]	0.984
Child sex	Female	1		1		1		1	
	Male	1.93 [0.31–12]	0.481	0.83 [0.13–5.14]	0.838	2.63 [0.46–14.93]	0.277	2.56 [0.23–29.04]	0.448
Maternal education	Literate	1		1		1		—	—
	Illiterate	3.47 [0.38–32.0]	0.273	1.25 [0.20–7.80]	0.808	4.41 [0.50–38.95]	0.182	—	—
Place of birth	Health facility	1		—	—	1		—	—
	Home	1.02 [0.11–9.61]	0.984	—	—	0.81 [0.09–7.28]	0.850	—	—
Occupation	Other occupations	—	—	1		—	—	1	
	Horticultural	—	—	0.72 [0.12–4.45]	0.720	—	—	0.54 [0.05–6.08]	0.615
Medication during pregnancy	No	1		—	—	1		1	
	Yes	1.11 [0.18–6.92]	0.910	—	—	1.71 [0.33–8.86]	0.524	0.83 [0.07–9.38]	0.877
Delivery mode	Caesarean section	1		1		1		1	
	Natural/Unassisted	1.25 [0.20–7.80]	0.808	0.54 [0.09–3.34]	0.506	1.70 [0.30–9.67]	0.550	1.68 [0.15–19.03]	0.676
Delivery term	Full term	1		1		1		1	
	Preterm [Not full term]	1.62	0.677	4.67	0.107	1.28	0.827	3.30	0.342

Variable	Category	Gross Motor OR [95% CI]	P- value	Fine Motor OR [95% CI]	P- value	Language OR [95% CI]	P- value	Social OR [95% CI]	P- value
		[0.17– 15.46]		[0.72– 30.42]		[0.14– 11.73]		[0.28– 38.67]	
BMI category	Normal weight	—	—	1		1		—	—
	Underweight	—	—	0.17 [0.02– 1.71]	0.131	2.38 [0.25– 22.65]	0.451	—	—
	Overweight	—	—	0.13 [0.01– 1.31]	0.083	0.42 [0.03– 7.07]	0.549	—	—
Maternal residual exposure	Low exposure	1		1		1		1	
	Moderate/High exposure	1.20 [1.19– 7.48]	0.845	7.79 [1.84– 72.15]	0.041	0.89 [0.76– 5.06]	0.893	0.89 [0.78– 10.12]	0.925

3.3 Composite univariate comparison between exposed and comparison districts

When developmental outcomes were combined into a composite measure and compared between the exposure district and comparison district, none of the maternal or child variables reached statistical significance in either stratum (all $p > .05$). The largest, though non-significant, effect size in the exposure district was for maternal occupation, with horticultural work associated with lower odds of composite delay relative to other occupations (OR = 0.33, 95% CI [0.08–1.28], $p = .108$), Table 7.

Table 7
Composite univariate comparison between exposed and comparison districts and the covariate.

Characteristics	Category	Exposed Districts OR [95% CI]	P-value	Comparison Districts OR [95% CI]	P-value
Maternal age [years]	Continuous	0.91 [0.79–1.03]	0.143	0.99 [0.93–1.06]	0.872
Child sex	Female	1		1	
	Male	1.28 [0.39–4.24]	0.683	1.49 [0.67–3.35]	0.331
Maternal education	Illiterate	1		1	
	Literate	2.72 [0.70–10.64]	0.149	1.55 [0.63–3.86]	0.342
Place of birth	Health facility	1		1	
	Home	0.34 [0.04–2.80]	0.318	1.03 [0.39–2.71]	0.957
Maternal occupation	Other occupations	1		1	
	Horticultural	0.33 [0.08–1.28]	0.108	1.16 [0.44–3.04]	0.765
Medication during pregnancy	No	1		1	
	Yes	0.81 [0.23–2.87]	0.747	1.01 [0.43–2.37]	0.986
Delivery mode	Caesarean section	1		1	
	Natural/Unassisted	0.56 [0.17–1.87]	0.343	0.92 [0.41–2.10]	0.851
Delivery term	Full term	1		1	
	Preterm	1.30 [0.26–6.56]	0.751	1.08 [0.30–3.91]	0.905
BMI category	Normal weight	1		1	
	Underweight	1.14 [0.26–5.08]	0.861	0.58 [0.18–1.93]	0.375
	Overweight	0.40 [0.07–2.15]	0.286	0.51 [0.16–1.58]	0.244
Maternal residual exposure	Low exposure	1		1	
	Moderate exposure	1.31 [0.39–4.43]	0.661	1.26 [0.56–2.84]	0.579

3.4 Multivariate composite comparison

After adjusting for all covariates simultaneously, maternal age remained an independent, statistically significant predictor of composite developmental delay in the exposure district (aOR = 0.87, 95% CI [0.64–0.98], $p = .044$),

confirming the protective effect observed in the domain-specific univariate model. Maternal illiteracy showed a large but borderline non-significant association with increased odds of delay in the exposure district (aOR = 4.27, 95% CI [0.88–20.65], $p = .071$). Table 8.

Table 8
Multivariate composite comparison exposed and comparison districts and the covariate.

Characteristics	Category	Exposed Districts aOR [95% CI]	P-value	Comparison Districts aOR [95% CI]	P-value
Maternal age [years]	Continuous	0.87 [0.64–0.98]	0.044	1.00 [0.93–1.09]	0.942
Child sex	Female	1		1	
	Male	1.68 [0.40–7.01]	0.475	1.59 [0.69–3.68]	0.277
Maternal education	Literate	1		1	
	Illiterate	4.27 [0.88–20.65]	0.071	1.67 [0.64–4.33]	0.291
Place of birth	Health facility	1		1	
	Home	0.24 [0.02–2.60]	0.242	1.05 [0.37–2.95]	0.923
Maternal occupation	Other occupations	1		1	
	Horticultural	0.24 [0.04–1.31]	0.100	1.11 [0.40–3.05]	0.838
Medication during pregnancy	No	1		1	
	Yes	1.08 [0.25–4.67]	0.917	0.90 [0.36–2.22]	0.811
Delivery mode	Caesarean section	1		1	
	Natural/Unassisted	0.38 [0.09–1.51]	0.169	0.90 [0.39–2.08]	0.808
Delivery term	Full term	1		1	
	Preterm [Not full term]	0.59 [0.09–3.71]	0.570	1.11 [0.30–4.15]	0.875
BMI category	Normal weight	1		1	
	Underweight	0.68 [0.11–4.31]	0.682	0.52 [0.15–1.79]	0.297
	Overweight	0.23 [0.03–1.72]	0.151	0.44 [0.13–1.47]	0.181
Maternal residual exposure	Low exposure	1		1	
	Moderate exposure	1.26 [0.30–5.24]	0.752	1.35 [0.58–3.14]	0.479

4.0 Discussion

The findings point to widespread, intensive pesticide use among farmers, with heavy reliance on organophosphates and pyrethroids a pattern consistent with reports from across sub-Saharan Africa (Abong'o et al., 2022; Lekei et al., 2020). The frequent use of organophosphates, particularly chlorpyrifos and profenofos, is especially concerning: these compounds inhibit acetylcholinesterase and carry significant acute and chronic health risks (Bjørning-Poulsen et al., 2008). A notable finding was the common practice of tank-mixing pesticides, with neonicotinoid–organophosphate

combinations appearing in 24.3% of records. Farmers often mix products to improve pest control and cut costs (Ngowi et al., 2016), but this practice can accelerate resistance (IRAC, 2021) and heighten toxicological risk through additive or synergistic effects combined organophosphate and pyrethroid exposure, for instance, has been shown to produce greater neurotoxicity than either compound alone (Abou-Donia et al., 2004).

Maternal pesticide residue analysis revealed a high exposure burden overall, with most participants classified as moderately or highly exposed. This is a significant public health concern, since pregnant women and young children are particularly susceptible to the neurotoxic and endocrine-disrupting effects of pesticides (Roberts et al., 2019). The pattern fits the agrarian character of Iringa and Njombe, where intensive cultivation of horticultural crops, maize, tea, and tobacco typically involves frequent and often poorly regulated agrochemical application (Chilipweli et al., 2021). Plausible exposure pathways include direct occupational contact during farming and spraying, residues in locally produced food and water, and environmental contamination of soil and surface water near commercial farms (Chilipweli et al 2025).

Makambako Town recorded the highest share of highly exposed mothers 31.3% within the district, likely reflecting its role as a major agricultural and commercial hub along a key transport corridor, where farming intensity and agrochemical turnover are concentrated (Mdee et al., 2020). Kilolo DC presents a more mixed picture: despite a substantial share of moderate exposure, it had a comparatively larger proportion of mothers in the normal category. This contrast may reflect differences in farming intensity, crop type, or Kilolo's more dispersed, highland settlement pattern relative to Makambako's concentrated agricultural and commercial activity. Even so, median residue levels were broadly similar across districts and all exceeded the safety threshold of 24.5 evidence that pesticide exposure is a regional problem rather than a localized one.

The elevated maternal exposure documented here is particularly concerning given the links between prenatal and early-life pesticide exposure and impaired neurodevelopment, including reduced cognitive function, attention deficits, and behavioral disorders (Rauh et al., 2012; Bouchard et al., 2011). Organophosphate insecticides readily cross the placental barrier and have been detected in fetal tissue, with maternal exposure associated with reduced birth weight, smaller head circumference, and shorter gestational age (Whyatt et al., 2003; Wolff et al., 2007). The widespread use of pesticide mixtures compounds this risk further, since cumulative or synergistic effects are not well captured by single-chemical risk assessments (Schantz et al., 2003).

These findings align with a growing body of evidence from agrarian settings across sub-Saharan Africa, where maternal and community-level pesticide exposure has reached levels of public health concern — particularly where agrochemical regulation is limited, protective practices among farmers are inadequate, and public awareness of handling and residue risks remains low (Chilipweli et al., 2021; Kapeleka et al 2024). Maternal exposure is especially concerning given the potential for placental transfer and excretion through breast milk, both of which raise the risk of low birth weight, preterm birth, and early-life neurodevelopmental effects in offspring, alongside broader endocrine-disrupting effects in mothers themselves (Benavides-Piracon et al., 2022; Hall et al., 2025; Sagiv et al., 2021)

This study examined maternal and child correlates of early developmental delay across gross motor, fine motor, language, and social domains in districts differentiated by horticultural pesticide use intensity. The most striking finding an almost eight-fold increase in the odds of fine motor delay among children of mothers with moderate-to-high residual pesticide exposure in the exposure district is consistent with a substantial body of evidence linking prenatal and early-life pesticide exposure to impaired child neurodevelopment. In Tanzania specifically, Chilipweli et al. (2021) reported that maternal pesticide exposure among smallholder tomato farmers in the Southern Agricultural Growth Corridor was associated with poorer child developmental outcomes, and more recently Mwakalasya et al. (2025) found that self-reported maternal pesticide spraying during pregnancy was associated with lower social-

emotional and executive-function scores among children aged 4–6 years in horticulture-intensive regions of Tanzania. The present finding of a strong fine motor association in the exposure district extends this local evidence to the motor domain and adds weight to the broader argument that horticultural pesticide use in Tanzania carries a measurable developmental cost for children.

The pattern observed here also mirrors the wider international literature on organophosphate and other non-persistent pesticides. Engel et al. (2011) reported that prenatal organophosphate exposure was associated with decrements in perceptual reasoning in early childhood, and Bouchard et al. (2011), following the CHAMACOS birth cohort, found that children in the highest quintile of prenatal organophosphate metabolite concentrations scored, on average, seven IQ points lower than those in the lowest quintile at age seven. A systematic review by González-Alzaga et al. (2014) concluded that prenatal organophosphate exposure is consistently linked to poorer motor and cognitive function in infancy and childhood, while Hertz-Picciotto et al. (2018) summarised evidence that fetal organophosphate exposure elevates the risk of cognitive, behavioural, and social developmental deficits, terming the cumulative effect a subclinical but population-level neurodevelopmental burden. The fine motor delay observed among children of moderately-to-highly exposed mothers in this study's exposure district is therefore biologically plausible and consistent with this body of work, even though the present study relied on categorical residual exposure classification rather than biomarker-based dose measurement.

The sex-specific vulnerability observed in the comparison district, where male children had substantially higher odds of fine motor delay than female children, is also consistent with the broader developmental literature, which repeatedly documents greater male susceptibility to early neurodevelopmental disruption. Notably, in the Tanzanian pesticide-exposure cohort described by Mwakalasya et al. (2025), sensitivity analyses showed that the negative associations between maternal pesticide spraying and social-emotional and executive-function scores were driven primarily by boys, mirroring the sex-differential pattern found in the present data, even though in this study the male vulnerability emerged in the comparison rather than the exposure district. This raises the possibility that male children carry an underlying biological susceptibility to motor delay that is not exclusively pesticide-mediated, but which may be unmasked or compounded by a range of environmental and perinatal exposures.

The protective association between increasing maternal age and both gross motor and language delay in the exposure district, which persisted after multivariate adjustment (aOR = 0.87), is a notable and somewhat counterintuitive finding, since advanced maternal age is more commonly discussed in the literature as a risk factor for adverse perinatal outcomes. A plausible explanation in this setting is that older mothers in horticultural communities have accumulated greater caregiving experience, household resources, or occupational seniority that reduces their direct hands-on exposure to pesticide spraying and application, thereby indirectly protecting child development. Alternatively, older mothers may adopt more consistent stimulation and care practices, a pathway not captured by the variables measured in this study. This finding should, however, be interpreted cautiously given the wide confidence interval and the possibility of residual confounding by unmeasured socioeconomic factors.

In contrast to the domain-specific findings, the composite univariate and multivariate comparisons between the exposure and comparison districts did not show an independent effect of district-level exposure status itself, and most maternal characteristics education, place of birth, occupation, medication use, delivery mode, delivery term, and BMI were not significantly associated with delay in either stratum. This null composite finding should not be read as evidence against a pesticide–development relationship; rather, it likely reflects the modest sample sizes within each exposure stratum, the wide confidence intervals surrounding most estimates, and the dilution of domain-specific effects (such as the fine motor association) when outcomes are aggregated into a single composite measure. Similar inconsistencies between aggregate and domain-specific outcomes have been noted elsewhere in the pesticide-

neurodevelopment literature, where associations are often domain- and sex-specific rather than uniform across all developmental areas.

Several limitations should be considered when interpreting these findings. First, the cross-sectional design precludes causal inference and raises the possibility of reverse causality or unmeasured confounding. Second, maternal residual pesticide exposure was determined through categorical classification rather than biological measurement (e.g., urinary metabolites), which may have introduced non-differential misclassification and biased associations toward the null in some models. Third, several subgroup estimates, particularly for fine motor delay in the exposure district, were based on small cell sizes, as reflected in the very wide confidence intervals (e.g., 1.84–72.15), and should be replicated in larger samples before firm conclusions are drawn. Finally, potential confounders such as household income, dietary micronutrient status, and home stimulation environment were not captured in the available variables and may partly explain both the maternal age and pesticide exposure associations observed.

5.0 Recommendations

Based on these findings, the following recommendations are proposed, Strengthen routine early childhood developmental screening within maternal and child health clinics serving horticultural and pesticide-exposure districts, with particular attention to fine motor assessment in male children and in children of mothers with moderate-to-high residual pesticide exposure.

Improve occupational safety for pregnant and lactating women engaged in horticultural work, including provision of personal protective equipment, training on safe pesticide handling and storage, and, where feasible, reassignment of pregnant workers away from direct spraying activities, consistent with the policy reforms recommended by Hertz-Picciotto et al. (2018). Promote integrated pest management and reduced reliance on highly hazardous pesticides in horticultural production areas, alongside community-level education on safe re-entry intervals and protective practices for household members.

Target younger mothers in exposure districts with reinforced antenatal counselling and parenting/stimulation support, given the observed protective association of maternal age, while further research clarifies the mechanism behind this relationship. Conduct larger, longitudinal studies with biomarker-based exposure assessment to validate the fine motor and language associations identified here and to disentangle the timing and dose of exposure across pregnancy.

6.0 Conclusion

This study demonstrates widespread use of hazardous pesticides and frequent pesticide mixing among farmers, resulting in elevated maternal pesticide exposure across the study area. Most mothers had moderate to high pesticide residue levels, highlighting a significant public health concern, particularly for pregnant women and young children. Children of mothers with moderate-to-high pesticide exposure, especially in horticultural areas, were at substantially higher risk of fine motor developmental delay, supporting evidence that prenatal pesticide exposure adversely affects early neurodevelopment. Although the cross-sectional design and exposure assessment methods limit causal inference, the findings underscore the need for strengthened pesticide safety practices, promotion of integrated pest management, and interventions to reduce maternal pesticide exposure in agricultural communities. Further longitudinal studies using biomarker-based exposure assessment are recommended to confirm these associations and guide public health policy.

Abbreviations

AChE	Acetylcholinesterase
BMI	Body Mass Index
BuTC	Butyrylthiocholine
CHAMACOS	Center for the Health Assessment of Mothers and Children of Salinas
ChE	Cholinesterase
CREC	Catholic University of Health and Allied Sciences and Bugando Medical Centre Research and Ethics Review Committee
CUHAS	Catholic University of Health and Allied Sciences
DTNB	5,5'-Dithiobis-(2-nitrobenzoic acid)
EMRs	Electronic Medical Records
EQM	Environmental Quality Management
FAO	Food and Agriculture Organization of the United Nations
FRAC	Fungicide Resistance Action Committee
Hb	Hemoglobin
HAZ	Height-for-Age Z-score
HOME	Home Observation for Measurement of the Environment
HRAC	Herbicide Resistance Action Committee
IRAC	Insecticide Resistance Action Committee
IQ	Intelligence Quotient
LMICs	Low- and Middle-Income Countries
MDAT	Malawi Developmental Assessment Tool
OP	Organophosphate

PPE
Personal Protective Equipment
RCH
Reproductive and Child Health
U/g Hb
Units per Gram of Hemoglobin
WAZ
Weight-for-Age Z-score
WHO
World Health Organization

Declarations

Written informed consent to participate was obtained from all adult participants before enrolment and data collection. For participants younger than 16 years of age, written informed consent was obtained from their parent(s) or legal guardian(s) prior to participation. Participant confidentiality and anonymity were strictly maintained throughout the study, and participation was entirely voluntary. Participants were informed of the study objectives, procedures, potential risks and benefits, their right to decline participation or withdraw from the study at any time without penalty or loss of entitled services, and the measures taken to protect the confidentiality of their information.

Ethical approval :

Ethical approval for this study was obtained from the Catholic University of Health and Allied Sciences/Bugando Medical Centre Research Ethics and Review Committee (CUHAS/BMC CREC), which serves as the Institutional Review Board (IRB) for the Catholic University of Health and Allied Sciences and Bugando Medical Centre (Approval No. CREC/866/2024). All study procedures involving human participants were conducted in accordance with the ethical principles of the Declaration of Helsinki and all applicable national and institutional guidelines and regulations.

Consent to publication :

Not applicable. This manuscript does not contain any individual person's identifiable data, images, or other information requiring consent for publication

Data and Material Availability Statement

Data from this study are available upon request .Questionnaire was developed by the author (Supplementary Material 1)

Competing Interest:

The authors declare no conflicts of interest.

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Author Contribution

The initial topic of this commentary was developed by PMC and refined by AVN, who also led manuscript development, with detailed guidance and iterative feedback from AVN, KM and ECN. All authors reviewed the manuscript and approved it for publication. The study selection and field work was done by and is the primary corresponding author. All authors have reviewed, read and agreed to the published version of the manuscript

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Figures

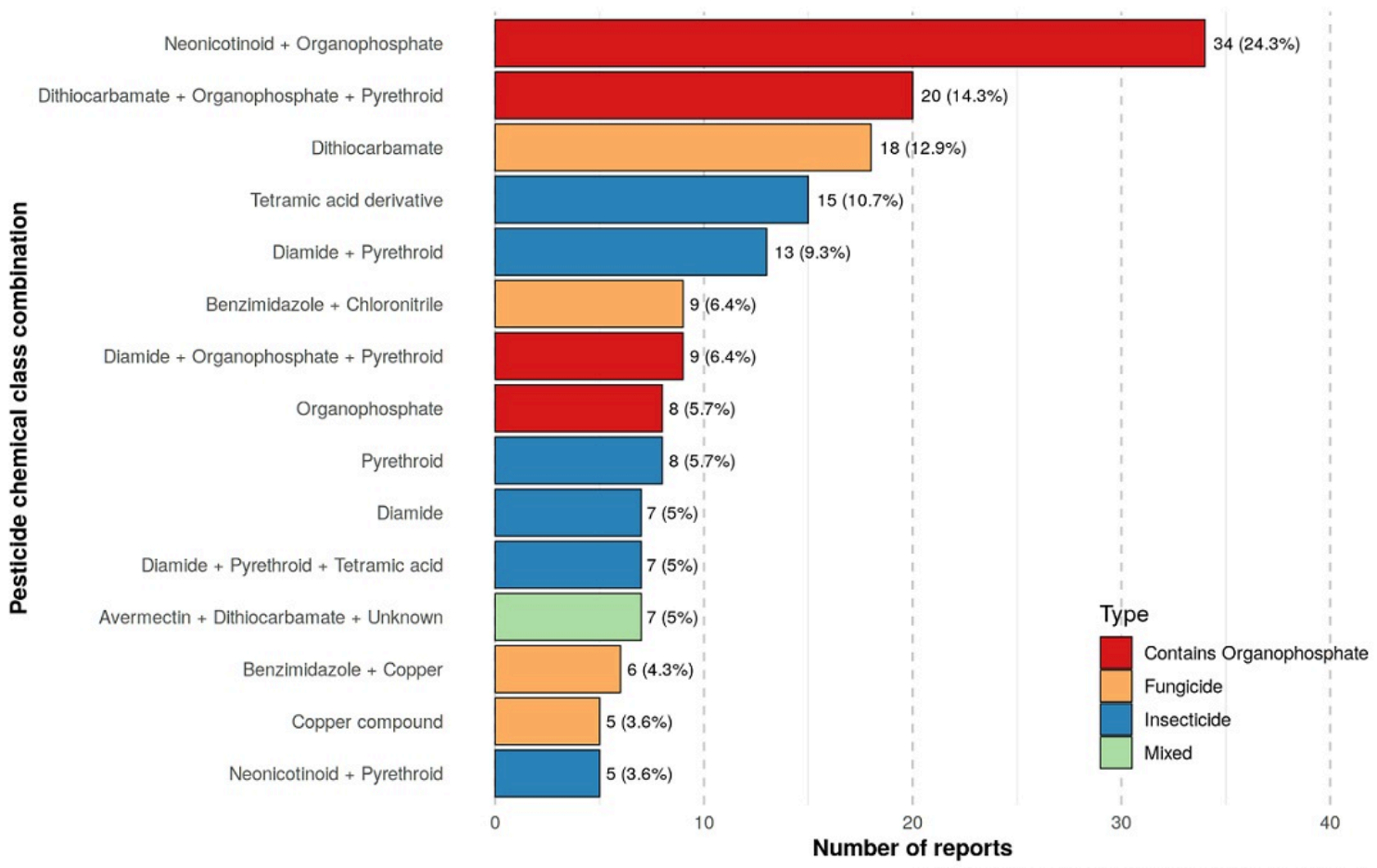


Figure 1

Most frequency reported chemical combinations (N=140)

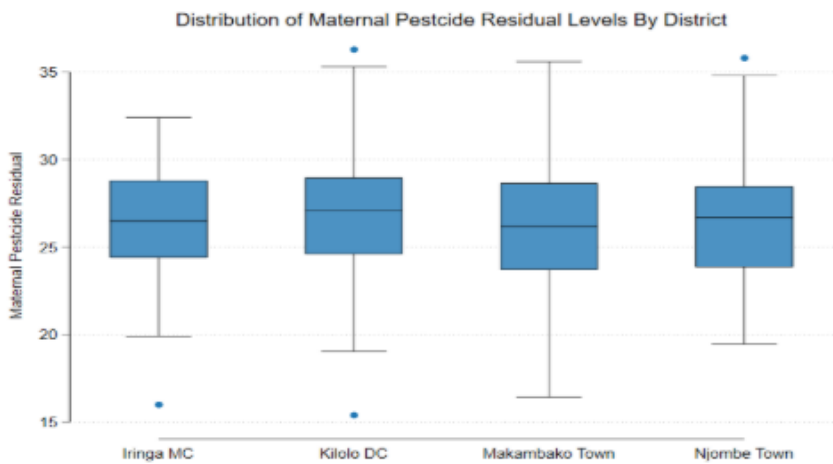


Figure 2

Presents the distribution of maternal pesticide residual levels across the four study districts: Iringa Municipal Council (MC), Kilolo District Council (DC), Makambako Town, and Njombe Town

Supplementary Files

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- [MaternalChildQuestionnaire6Months.pdf](#)