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LETTER

Evaluation of an organic diet intervention to reduce pesticide exposure: a repeated measures cross-over study among rural and urban communities in Selangor, Malaysia

Vivien How[1,*], **Maisarah Nasution Waras**[2], **Jamilatul Wahidah Jamal Rodzi**[2], **Nurul Afina Ahmad Zamani**[1] and **Hafizah Rukman**[1]

[1] Department of Environmental and Occupational Health, Faculty of Medicine and Health Sciences, Universiti Putra Malaysia, 43400 UPM Serdang, Selangor, Malaysia
[2] Department of Toxicology, Advanced Medical & Dental Institute, Universiti Sains Malaysia, 13200 Kepala Batas, Penang, Malaysia
[*] Author to whom any correspondence should be addressed.

E-mail: vivien@upm.edu.my

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Abstract

Introduction. Organophosphate (OP) pesticides are widely used in agriculture, resulting in dietary pesticide exposure through conventionally grown foods. While organic diets are often recommended to reduce pesticide burden, the effectiveness of such interventions and the influence of local environmental factors and metabolic differences remain underexplored in mixed urban–rural settings. This study investigated the impact of a short-term organic diet intervention on urinary OP metabolite concentrations among rural and urban adults and children in Selangor, Malaysia, and assessed the rebound effect after returning to a conventional diet. *Method*. Seventeen families (9 rural, 8 urban) participated in a 16-day cross-over intervention, consisting of a baseline conventional diet phase (4 days), an organic diet phase (7 days), and a post-intervention conventional diet phase (5 days). Daily first-morning void urine samples were analyzed for four OP metabolites (DMP, DEP, DMTP, DETP). Linear mixed-effects models assessed differences across dietary phases, and daily trends illustrated changes over time. *Result*. Urinary concentrations of DMP, DEP, and DMTP declined significantly during the organic diet phase compared to baseline ($p < 0.05$), with the largest reductions observed in children. DETP levels remained relatively stable across phases. Rebound effects were evident for DMP and DMTP when participants resumed a conventional diet, particularly among rural children, reflecting ongoing environmental exposure and age-related differences in metabolic clearance. Urban adults showed the most consistent reduction, while rural families exhibited greater variability due to overlapping dietary and non-dietary pathways. *Conclusion*. A short-term switch to an organic diet can significantly reduce urinary OP metabolite concentrations, but re-exposure occurs rapidly once conventional foods are reintroduced, especially among children in agricultural settings. These findings highlight the importance of sustained dietary changes, improved pesticide management, and policies that increase access to affordable organic produce. Future research should examine longer-term interventions and integrate health outcome monitoring to support evidence-based pesticide exposure reduction strategies.

1. Introduction

Pesticide residues in food are a widespread environmental and public health concern due to their potential long-term health effects. Organophosphate (OP) pesticides, commonly used in agriculture and household pest

control, have been associated with adverse health outcomes, including neurotoxicity, endocrine disruption, metabolic disorders, and reproductive toxicity [1]. Chronic exposure, low-level OP exposure is associated with oxidative stress, immune dysregulation, and developmental impacts, particularly among vulnerable populations such as children and pregnant women [2].

Although Maximum Residue Levels (MRLs) are established to protect consumer safety, concerns remain regarding cumulative and chronic exposure from multiple pesticide residues through diet [3]. Regulatory risk assessments often focus on single-pesticide exposures, while real-world exposure typically involves complex mixtures that may have additive or synergistic effects. Furthermore, epidemiological studies have associated dietary pesticide exposure to an increased risk of neurodegenerative and endocrine-related metabolic disorders [4], though conclusive evidence is still emerging.

Rural and urban communities exhibit distinct dietary and environmental exposure patterns that influence their cumulative health risks [5]. Rural households in agricultural regions may be exposed through direct contact with pesticides through ambient air, water sources, and occupational pathways. Farm workers and their families are at higher risk of exposure due to pesticide drift, direct handling of agrochemicals, and potential contamination of drinking water sources [6]. In contrast, urban residents are primarily exposed through dietary intake of conventionally grown produce and indoor pesticide use [7]. Socioeconomic status, food accessibility, and purchasing behaviour further contribute to differences in dietary pesticide exposure between rural and urban residents [8]. Lower-income households may have limited access to organic food and face higher exposure risks due to suboptimal housing conditions and increased reliance on processed foods containing imported pesticide-treated ingredients.

Past research has showed that switching to an organic diet can significantly reduce urinary pesticide metabolite concentrations within days [9], but most of this research comes from Western countries. Malaysia, particularly Selangor state, provides a unique context for examining rural-urban differences: it has both intensive agriculture and densely populated urban areas, allowing investigation of how dietary intervention may work across diverse exposure settings in a tropical, middle-income country.

This study evaluates whether a short-term organic diet can significantly reduce urinary OP pesticide metabolites among rural and urban families in Selangor, Malaysia. It also explores whether specific demographic groups, such as children or rural households are more vulnerable to pesticide cumulation and may benefit most from dietary changes. This finding aims to support evidence-based strategies for dietary exposure reduction, inform sustainable agricultural policy, and strengthen public health recommendations for pesticide risk mitigation.

2. Material and methods

2.1. Study population

This study recruited a convenience sample of families from rural and urban communities in Selangor, a state located on the west coast of Peninsular Malaysia. Selangor is Malaysia's most populous state, with both densely urbanized areas and rural agricultural regions, making it a representative setting for exploring differences in dietary pesticide exposure and the potential impact of organic diet interventions. A total of 17 families participated, comprising 9 rural and 8 urban families. Eligible families included at least one parent (mother or father) and a child aged between 3 and 6 years who was toilet-trained and regularly consumed conventionally grown (non-organic) food before the intervention.

Participants were approached through local kindergartens, and social support groups with the help of community health volunteers and local gatekeepers. A total of 23 eligible families were initially invited to participate, and 17 consented to enrol, resulting in a participation rate of approximately 74%. Families who declined cited time constraints and concerns about daily urine collection. All participating families completed the full 16-day diet protocol.

To minimize dietary variation due to cultural differences, only Malay families were selected. In this context, Malay refers to families from Malaysia's majority ethnic group, who typically share similar food preferences, preparation methods, and household dietary practices. This approach ensured that dietary habits, food preparation, and food sourcing remained as consistent as possible across both urban and rural groups. The inclusion of both urban and rural Malay families allows for an assessment of differential exposure risks based on environmental and dietary contexts, providing insights into potential health disparities and informing recommendations for organic food consumption strategies in Malaysia.

2.2. Data collection

Families participated in the study for 16 consecutive days. One week before the data collection commenced, researchers obtained informed consent, administered a baseline questionnaire to collect information on

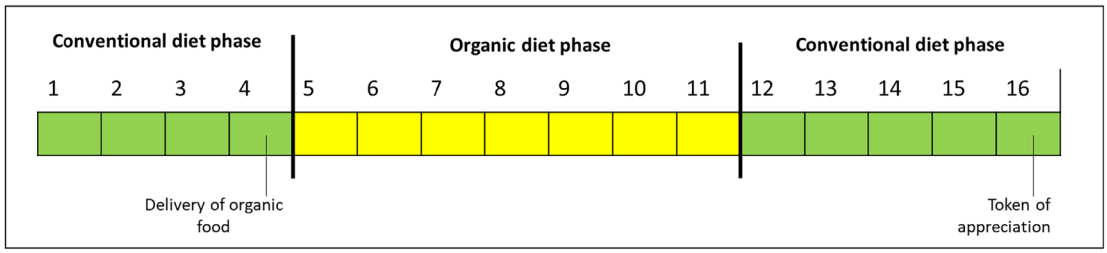

Figure 1. Study activities by day. Note: Participants followed a 16-day crossover diet protocol consisting of an initial Conventional Diet Phase (Days 1–4), an Organic Diet Phase (Days 5–11), and a second Conventional Diet Phase (Days 12–16). Families received their supply of organic foods on Day 4. Urine samples were collected daily throughout all phases (16 samples per participant). Daily in-person interviews were conducted with parents during each home visit to gather information on dietary adherence, possible non-organic food consumption, and household pesticide use. A final token of appreciation was given to families at the end of the study on Day 16.

household characteristics, provided materials for urine specimen collection, and trained parents on proper urine specimen collection techniques. Parents also submitted a grocery list of food items to be consumed supplied during the organic diet phase.

To ensure authenticity, all organic foods were purchased exclusively from certified organic grocery store chains registered with the Department of Agriculture Malaysia. The selected items were delivered directly to each family's home to minimize the risk of substitution with conventional products on the fourth day (figure 1). Researchers conducted daily in-person interviews with the guardian (mother or father) when collecting urine specimens. These interviews gathered information on household and workplace pesticide use and on each child's adherence to the diet protocols. Parents were asked whether the child ate any food outside the home, consumed any conventional foods during the organic diet phase, or had any leftover organic food during the second conventional diet phase. Compliance with the diet protocol relied entirely on these self-reported interviews; no additional objective verification (such as receipts or spot checks) was conducted.

2.3. Diet protocol

Participants followed a structured dietary protocol consisting of three consecutive phases: an initial conventional diet phase (C1) for 4 days, an organic diet phase for 7 days, and a second conventional diet phase (C2) for 5 days. This crossover design enables direct comparison of pesticide metabolite levels between dietary phases.

During the conventional diet phases, families consumed their usual, commercially available foods purchased from local markets or grocery stores without any restrictions on pesticide residue. These foods typically included rice, bread, noodles, fruits, vegetables, meat, fish, eggs, and processed snacks as commonly eaten by the family before the study.

For the organic diet phase, parents were instructed to select food items that the participating child and family typically consumed, but exclusively in certified organic form. This is to minimize potential confounding factors due to dietary changes. The provided organic foods included a variety of staple items such as organic rice, fruits, bread, cereals, vegetables, dairy products, eggs, juices, and snack foods. To facilitate adherence to the organic diet phase, sufficient quantities of organic food were supplied for the entire family. All organic food items were sourced from the same grocery store chain in both urban and rural communities to ensure consistency in product availability and quality. The supplier holds valid organic certification under Malaysia's national organic certification scheme (*Skim Pensijilan Organik Malaysia* (myOrganic)), regulated by the Department of Agriculture Malaysia.

Diet compliance was monitored through daily in-person interviews and parental self-reports. Families were asked to confirm whether only the provided organic foods were consumed during the organic phase. No additional objective checks (such as grocery receipts or spot inspections) were used, and no formal quantitative compliance score was calculated. Parents reported full adherence throughout the organic phase.

2.4. Urine specimen collection

Participants provided first-morning voids urine samples for 16 consecutive days, starting on the second day of the study (i.e. days 1–16, refer to figure 1). Urine specimens were collected from both guardians and parents and children using sterile urine collection jars. Parents recorded the collection time of each sample and stored the specimens in a portable refrigerator provided by the research team to maintain sample integrity. Researchers collected urine specimens daily and provided parents with new collection materials for the next sampling.

All urine samples were aliquoted and stored at –80°C to prevent degradation before being transported on dry ice to the Advanced Medical and Dental Institute, Universiti Sains Malaysia (USM), for laboratory analysis. A total of 544 first-morning void urine samples were collected from all participating families across both urban and rural communities. This systematic collection protocol ensured a high level of sample integrity and allowed for enabled a comprehensive assessment of pesticide metabolite variations across dietary intervention phases.

2.5. Laboratory analysis of urine specimens

Urinary organophosphate metabolites were analyzed using solid-phase extraction and derivatization, following a validated method by Prapamontol *et al* (2014) [10]. Briefly, 2.5 ml urine samples were extracted using a salt-assisted liquid–liquid extraction method with acetonitrile and diethyl ether, followed by acid hydrolysis, derivatization with pentafluorobenzyl bromide (PFBBr), and purification using anhydrous Na_2SO_4 and K_2CO_3. The final dried extract was reconstituted in toluene and analyzed using gas chromatography–mass spectrometry (GC–MS) (Model: 7820 A GC System (Agilent Technologies, CA, USA), equipped with a 5977 B network mass-selective detector (Agilent J and W Scientific, USA)).

Instrumental conditions included electron ionization (EI) at 70 eV, separation on a DB-5MS column (30 m × 0.25 mm × 0.25 μm), and detection using selected ion monitoring (SIM) mode at mass-to-charge ratios (m/z) specific for OP metabolites. The method detection limit (LOD) and limit of quantification (LOQ) were determined using a signal-to-noise ratio of 3:1 and 10:1, respectively. A group LOD and LOQ was applied for all metabolites: LOD = 0.0008 ppm and LOQ = 0.0010 ppm.

The four main urinary OP pesticide metabolites analyzed in this study were:

a) Dimethyl phosphate (DMP) (CAS No. 813-78-5)

b) Diethyl phosphate (DEP) (CAS No. 598-02-7)

c) Dimethyl thiophosphate (DMTP) (CAS No. 1112-38-5)

d) Diethyl thiophosphate (DETP) (CAS No. 2465-65-8)

Analytical standards for all OP metabolites and the internal standard (e.g., dibutyl phosphate (DBP), CAS No. 107-66-4) were purchased from Sigma-Aldrich (St. Louis, MO, USA) and were of certified analytical grade (⩾ 98% purity). Urinary OP metabolite concentrations were reported both as unadjusted values (nmol/l) and as creatinine-adjusted concentrations (nmol/g creatinine), calculated by dividing the urinary metabolite concentration by the measured urinary creatinine level for each sample.

Quality assurance and quality control (QA/QC) procedures included the use of internal standards (ISTDs), five-point calibration curves with coefficients of determination (R^2) > 0.995, procedural blanks, duplicate sample analyses, and matrix spikes. Mean recoveries for all OP metabolites ranged from 85% to 110%, and the relative standard deviation (RSD) for replicate measurements was maintained below 15%, confirming the method's accuracy and precision.

2.6. Statistical analysis

All statistical analyses were performed using IBM SPSS Statistics for Windows, Version [25.0] (IBM Corp., Armonk, NY, USA). Urinary organophosphate (OP) metabolite concentrations were expressed in micrograms per liter (μg/l). Values below the limit of detection (LOD) were substituted with LOD/2.

Descriptive statistics summarized household sociodemographic characteristics (table 1) and presented both creatinine-adjusted and unadjusted urinary OP metabolite concentrations across three diet phases for both urban and rural communities (table 2).

To assess the effect of the organic diet intervention across phases, a linear mixed-effects model was applied using the MIXED procedure in SPSS. Diet phase (Conventional Phase I, Organic Phase, Conventional Phase II) was specified as a fixed effect, and participant ID was included as a random effect to account for within-subject repeated measures. Estimated marginal means (EMMeans) with Bonferroni adjustment were used for post-hoc comparisons, specifically to evaluate potential rebound effects in Conventional Phase II. Model estimates comparing Conventional Phase I to the Organic Phase, including percent reductions, 95% confidence intervals (Cis), and p-values, are presented in tables 3 and 4 for urban and rural families, respectively.

Given the right-skewed distribution of urinary metabolite data, sensitivity analyses were conduced using natural log (In)-transformed concentrations. Both untransformed and log-transformed models yielded consistent patterns and statistical significance; therefore, untransformed values are reported in the main text for interpretability, while log-transformed results are included as Supplementary table 1 and 2.

Table 1. The sociodemographic data of the household demographic (N = 17).

Sociodemographic characteristics		n (%) Rural (n = 9)	n (%) Urban (n = 8)
Age (year) (parent)	Median (IQR)	36 (31–41)	36 (31–41)
Age (year) (children)	Median (IQR)	7 (4–11)	6 (4–8)
Gender (parent)	Male	3 (33.3)	2 (25.0)
	Female	6 (66.6)	6 (75.0)
Gender (Children)	Male	4 (44.4)	4 (50.0)
	Female	5 (55.5)	4 (50.0)
Educational level (parent)	High School level[a]	6 (66.6)	1 (12.5)
	Degree level[b]	3 (33.3)	7 (75.0)
	Master level[c]	—	1 (12.5)
Existing Health Problems (Children)	Asthma	2 (22.2)	5 (83.3)
Total Household income (RM)	<5,000	9 (100.0)	3 (37.5)
	5,000—9,999	—	2 (25.0)
	>10,000	—	3 (37.5)

[a] High School level: Completed secondary school education;
[b] Degree level: Completed tertiary education with a Bachelor's degree from an accredited university or college;
[c] Master level: Completed postgraduate education with a Master's degree.

Graphical plots illustrating daily variations and phase-wise trends in urinary OP metabolite concentrations (figures 2–5) were also generated to visualize group and individual responses across the intervention. Statistical significance was set up a $p < 0.05$.

3. Results

As shown in table 1, the median age of parents was 36 years in both rural and urban households. Children's median age was slightly higher in rural families (7 years) than in urban families (6 years). Most parents were female, and gender distribution among children was balanced across groups. Rural parents were mainly educated to a high school level, while urban parents were more likely to hold a university degree. Asthma was more common among urban children (83.3%) than rural children (22.2%). All rural households reported monthly incomes below RM 5,000, whereas urban households showed greater income diversity.

Table 2 presents the median urinary OP metabolite concentrations, reported as both unadjusted (nmol/l) and creatinine-adjusted (nmol/g creatinine) values, across the three diet phases among urban and rural adults and children. Overall, DMP, DEP, and DMTP levels declined during the organic diet phase compared to Conventional Phase I, then rose again in Conventional Phase II. For example, median unadjusted DMP levels ranged from 0.169 to 0.220 nmol/l, while DEP ranged from 0.299 to 0.40 nmol l^{-1}. DETP concentrations remained relatively stable across phases. Children consistently showed higher median metabolite concentrations than adults.

Table 3 shows the unadjusted median urinary OP metabolite concentrations and mixed-effects model estimates for urban adults and children across the three diet phases. Among adults, median DMP levels decreased from 0.223 nmol l^{-1} (IQR: 0.179–0.271) in Conventional Phase I to 0.169 nmol l^{-1} (IQR: 0.167–0.172) during the Organic Phase, with a percent reduction of 9.2% ($p < 0.001$). DEP and DMTP levels showed larger reductions of 36.2% and 53.5%, respectively, both statistically significant ($p < 0.001$). DETP showed no significant reduction.

In urban children, DMP concentrations declined by 56.1% from the initial conventional phase (median: 0.313 nmol l^{-1}) to the organic phase (median: 0.169 nmol l^{-1}), with the strongest model estimate ($p < 0.001$). DEP and DMTP levels in children also showed significant reductions of 27.0% and 48.9%, respectively (both $p < 0.001$). DETP concentrations changed only slightly (12.5% reduction; $p = 0.05$). Besides, EMMeans contrasts indicated that DMP and DMTP levels showed a clear rebound effect in children after returning to a

Table 2. Urinary analyte concentrations (adjusted and unadjusted) in each diet phase among children and adults from urban and rural families (N = 16).

Analyte	Study group [U = urban/ R = rural]	Conventional phase I median (IQR)		Organic phase median (IQR)		Conventional phase II Median (IQR)	
		Unadjusted nmol/l	Adjusted nmol/ g creatinine	Unadjusted nmol/l	Adjusted nmol/g creatinine	Unadjusted nmol/l	Adjusted nmol/g creatinine
DMP	U/Adult	0.223 (0.179, 0.271)	0.300 (0.220–0.380)	0.169 (0.167, 0.172)	0.22 (0.21–0.24)	0.206 (0.183, 0.272)	0.28 (0.24–0.36)
	U/Children	0.313 (0.186, 0.633)	0.450 (0.270–0.850)	0.169 (0.167, 0.171)	0.24 (0.21–0.26)	0.217 (0.181, 0.299)	0.32 (0.26–0.44)
	R/Adult	0.224 (0.179, 0.289)	0.310 (0.220–0.400)	0.204 (0.175, 0.217)	0.28 (0.23–0.30)	0.298 (0.223, 0.485)	0.40 (0.30–0.65)
	R/Children	0.272 (0.182, 0.328)	0.380 (0.250–0.480)	0.189 (0.172, 0.223)	0.26 (0.23–0.31)	0.248 (0.203, 0.351)	0.35 (0.28–0.52)
DEP	U/Adult	0.399 (0.259, 0.649)	0.520 (0.350–0.850)	0.299 (0.260, 0.437)	0.40 (0.35–0.60)	0.236 (0.296, 0.389)	0.32 (0.40–0.53)
	U/Children	0.466 (0.281, 0.565)	0.610 (0.380–0.750)	0.319 (0.258, 0.408)	0.43 (0.35–0.55)	0.303 (0.255, 0.581)	0.42 (0.35–0.80)
	R/Adult	0.388 (0.284, 0.625)	0.500 (0.370–0.800)	0.289 (0.191, 0.432)	0.38 (0.26–0.58)	0.270 (0.234 0.389)	0.36 (0.31–0.52)
	R/Children	0.329 (0.249, 1.275)	0.450 (0.330–1.800)	0.448 (0.334, 0.526)	0.56 (0.42–0.65)	0.352 (0.252, 0.525)	0.46 (0.33–0.70)
DMTP	U/Adult	0.098 (0.089, 0.355)	0.130 (0.100–0.480)	0.078 (0.076, 0.111)	0.10 (0.10–0.15)	0.0806 (0.078, 0.084)	0.10 (0.10–0.13)
	U/Children	0.093 (0.084, 0.223)	0.120 (0.090–0.300)	0.078 (0.076, 0.080)	0.10 (0.10–0.11)	0.0911 (0.079, 0.119)	0.12 (0.10–0.16)
	R/Adult	0.189 (0.149, 0.231)	0.240 (0.190–0.290)	0.107 (0.086, 0.126)	0.14 (0.11–0.17)	0.0997 (0.082, 0.110)	0.13 (0.10–0.15)
	R/Children	0.193 (0.159, 0.203)	0.250 (0.200–0.270)	0.150 (0.099, 0.168)	0.19 (0.13–0.21)	0.083 (0.082, 0.136)	0.11 (0.10–0.18)
DETP	U/Adult	0.257 (0.252, 0.262)	0.340 (0.300–0.370)	0.249 (0.249, 0.253)	0.32 (0.32–0.34)	0.253 (0.252, 0.256)	0.33 (0.32–0.35)
	U/Children	0.256 (0.254, 0.260)	0.340 (0.300–0.350)	0.2502 (0.249, 0.251)	0.32 (0.31–0.33)	0.255 (0.252, 0.257)	0.33 (0.32–0.35)
	R/Adult	0.268 (0.264, 0.829)	0.360 (0.320–1.200)	0.256 (0.254, 0.256)	0.33 (0.32–0.34)	0.267 (0.258, 0.274)	0.35 (0.33–0.37)
	R/Children	0.271 (0.264, 0.275)	0.370 (0.320–0.380)	0.256 (0.254, 0.259)	0.34 (0.33–0.35)	0.262 (0.255, 0.285)	0.34 (0.32–0.38)

Limit of Detection (LOD) for all analyte—0.0008 ppm; LOQ—0.0010 ppm.
Total Dimehtylphopshates (Total DM = DMP + DMTP); Total diethylphosphates (Total DE = DEP + DETP); Total diakylphosphates (Total DAP = Total DM + Total DE).

conventional diet (both $p < 0.001$), whereas adults showed smaller or non-significant rebounds for some metabolites.

These findings confirm that the organic diet phase significantly reduced urinary pesticide metabolites in urban families, particularly for DMP and DMTP, with children experiencing the largest percentage declines but also showing the strongest rebound effect.

Table 4 presents the unadjusted urinary OP metabolite concentrations and mixed-effects model estimates for rural adults and children across the three diet phases. Among adults, median DMP levels decreased by 16.1% from 0.224 nmol l^{-1} (IQR: 0.179–0.289) in Conventional Phase I to 0.204 nmol l^{-1} (IQR: 0.175–0.217) during the Organic Phase ($p < 0.001$). DEP and DMTP showed larger reductions of 31.7% and 42.2%, respectively (both $p < 0.001$). DETP levels declined by 52.6% ($p < 0.001$).

For rural children, DMP concentrations dropped by 22.7% from 0.272 nmol l^{-1} (IQR: 0.182–0.328) to 0.189 nmol l^{-1} (IQR: 0.172–0.223), with a significant model estimate ($p < 0.001$). DEP and DMTP levels decreased by 37.1% and 26.5%, respectively, both statistically significant ($p < 0.001$). DETP levels showed only a slight reduction of 5.9% ($p < 0.05$). On the other hand, EMMeans contrasts indicated that for rural adults, DMP and DEP showed significant rebound effects after returning to a conventional diet ($p < 0.05$). For children, only DMP showed a significant rebound ($p < 0.05$), while other metabolites remained largely unchanged after the organic phase.

Table 3. Urinary Analyte Concentrations (unadjusted, nmol/l)in Each Diet Phase among Children and Adults from Urban Families.

Analyte	Conventional phase I median (IQR)	Organic phase median (IQR)	Conventional phase II median (IQR)	% Reduction[a]	Mixed-effects model estimate (95% CI)	p-value	EMMeans (95% CI)	p-value
Adult (n = 8)								
DMP	0.223 (0.179, 0.271)	0.169 (0.167, 0.172)	0.206 (0.183, 0.272)	9.2%	–0.025 (–0.042, –0.008)	<0.001**	–0.043 (–0.122, 0.036)	<0.05*
DEP	0.399 (0.259, 0.649)	0.299 (0.260, 0.437)	0.236 (0.296, 0.389)	36.2%	–0.203 (–0.316, –0.090)	<0.001**	0.101 (–0.275, 0.477)	<0.05*
DMTP	0.098 (0.089, 0.355)	0.078 (0.076, 0.111)	0.0806 (0.078, 0.084)	53.5%	–0.099 (–0.134, –0.064)	<0.001**	–0.004 (–0.007, –0.001)	>0.05
DETP	0.257 (0.252, 0.262)	0.249 (0.249, 0.253)	0.253 (0.252, 0.256)	12.1%	–0.031 (–0.065, 0.003)	>0.05	0.028 (0.002—0.045)	<0.05*
Children (n = 8)								
DMP	0.313 (0.186, 0.633)	0.169 (0.167, 0.171)	0.217 (0.181, 0.299)	56.1%	–0.216 (–0.273, –0.159)	<0.001**	0.422 (–0.513, 0.357)	<0.001**
DEP	0.466 (0.281, 0.565)	0.319 (0.258, 0.408)	0.303 (0.255, 0.581)	27.0%	–0.129 (–0.194, –0.064)	<0.001**	0.036 (–0.101, 0.173)	<0.05*
DMTP	0.093 (0.084, 0.223)	0.078 (0.076, 0.080)	0.0911 (0.079, 0.119)	48.9%	–0.068 (–0.099, –0.037)	<0.001**	0.035 (–0.019, 0.089)	<0.05*
DETP	0.256 (0.254, 0.260)	0.2502 (0.249, 0.251)	0.255 (0.252, 0.257)	12.5%	–0.032 (–0.063, –0.001)	0.05*	0.030 (−0.056–0.015)	>0.05

1. Limit of Detection (LOD) for all analyte:0.0008 ppm; LOQ: 0.0010 ppm.

2. Total Dimehtylphopshates (Total DM = DMP + DMTP); Total diethylphosphates (Total DE = DEP + DETP); Total diakylphosphates (Total DAP = Total DM + Total DE).

3. [a]Percent reduction was calculated as: $\left(\frac{Conventional\ Phase\ I - Organic\ Phase}{Conventional\ Phase\ I}\right) \times 100$

4. Mixed-effects model estimate based on unadjusted metabolite concentration,indicates the mean change in urinary pesticide metabolite concentration from Conventional Phase I to the Organic Phase, adjusting for repeated measures within participants.

5. EMMeans contrasts show the estimated based on unadjusted metabolite concentration, indicates the mean change in urinary pesticide metabolite concentration from Organic Phase to Phase II to illustrate the rebound effect (i.e., whether levels increased after returning to a conventional diet.

6. Model: $Y_{ij} = \beta_o + \beta_1(Diet\ Phase) + u_i + \epsilon_{ij}$

Y_{ij}: *urine pesticide level for participant i at time j*

β_o : *Overall intercept*

β_1 : *Fixed effect of diet phase*

u_i: *random intercept for participant i*

ϵ_{ij}: *residual error*

7. **p-value is significant at 0.001 level; *p-value is significant at 0.05 level.

Table 4. Urinary analyte concentrations (nmol/l) in each diet phase among children and adults from rural families (N = 18).

Analyte	Conventional phase I median (IQR)	Organic phase median (IQR)	Conventional phase II median (IQR)	% Reduction[a]	Mixed-effects model estimate (95% CI)	p-value	EMMeans (95% CI)	p-value
Adult (n = 9)								
DMP	0.224 (0.179, 0.289)	0.204 (0.175, 0.217)	0.298 (0.223, 0.485)	16.1%	−0.038 (–0.117, 0.041)	<0.001**	0.149 (0.070, 0.228)	<0.05**
DEP	0.388 (0.284, 0.625)	0.289 (0.191, 0.432)	0.270 (0.234 0.389)	31.7%	–0.060 (–0.153, 0.033)	<0.001**	0.065 (–0.133, 0.263)	<0.05**
DMTP	0.189 (0.149, 0.231)	0.107 (0.086, 0.126)	0.0997 (0.082, 0.110)	42.2%	–0.139 (−0.337, 0.059)	<0.001**	–0.012 (–0.015, –0.009)	>0.05
DETP	0.268 (0.264, 0.829)	0.256 (0.254, 0.256)	0.267 (0.258, 0.274)	52.6%	−0.261 (−0.398, −0.124)	<0.001**	0.011 (0.017, 0.031)	>0.05
Children (n = 9)								
DMP	0.272 (0.182, 0.328)	0.189 (0.172, 0.223)	0.248 (0.203, 0.351)	22.7%	−0.081 (–0.084, –0.078)	<0.001**	0.079 (–0.014, 0.172)	<0.05**
DEP	0.329 (0.249, 1.275)	0.448 (0.334, 0.526)	0.352 (0.252, 0.525)	37.1%	–0.050 (–0.079, −0.021)	<0.001**	0.048 (–0.089, 0.185)	>0.05
DMTP	0.193 (0.159, 0.203)	0.150 (0.099, 0.168)	0.083 (0.082, 0.136)	26.5%	−0.284 (−0.237, 0.015)	<0.001**	0.024 (–0.006, 0.054)	>0.05
DETP	0.271 (0.264, 0.275)	0.256 (0.254, 0.259)	0.262 (0.255, 0.285)	5.9%	−0.016 (−0.009, −0.019)	<0.05*	0.014 (0.005, 0.025)	>0.05

8. Limit of Detection (LOD) for all analyte:0.0008 ppm; LOQ: 0.0010 ppm.

1. Total Dimehtylphopshates (Total DM = DMP + DMTP); Total diethylphosphates (Total DE = DEP + DETP); Total diakylphosphates (Total DAP = Total DM + Total DE).

2. [a]Percent reduction was calculated as: $\left(\frac{Conventional\ Phase\ I - Organic\ Phase}{Conventional\ Phase\ I}\right) \times 100$.

3. Mixed-effects model estimate indicates the mean change in urinary pesticide metabolite concentration from Conventional Phase I to the Organic Phase, adjusting for repeated measures within participants.

4. EMMeans contrasts show the estimated indicates the mean change in urinary pesticide metabolite concentration from Organic Phase to Phase II to illustrate the rebound effect (i.e., whether levels increased after returning to a conventional diet.

5. Model: $Y_{ij} = \beta_o + \beta_1(Diet\ Phase) + u_i + \epsilon_{ij}$

Y_{ij}: *urine pesticide level for participant i at time j*

β_o : *Overall intercept*

β_1 : *Fixed effect of diet phase*

u_i: *random intercept for participant i*

ϵ_{ij}: *residual error*

6. **p-value is significant at 0.001 level; *p-value is significant at 0.05 level.

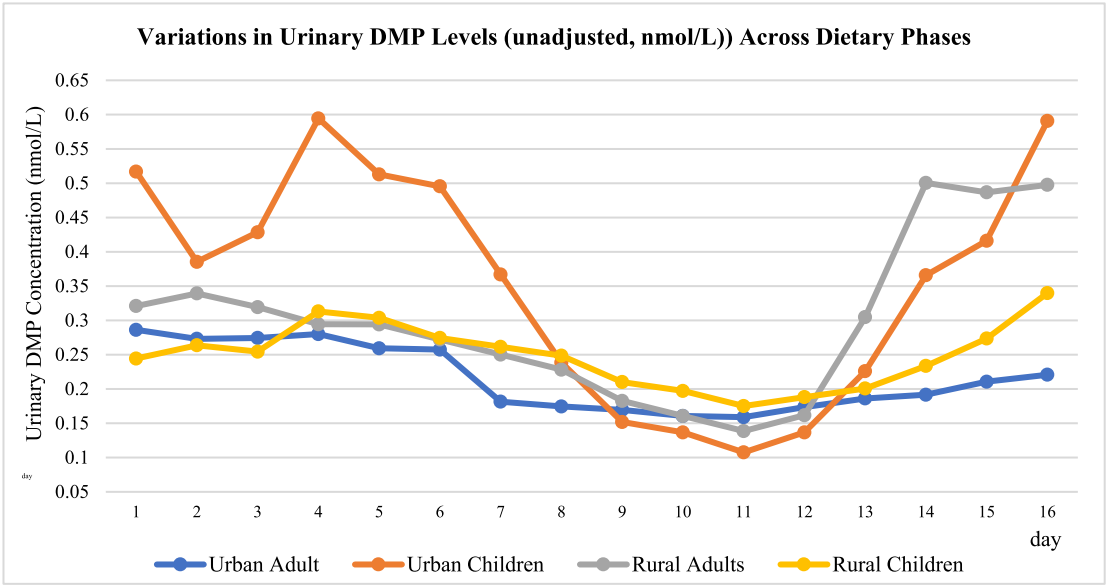

Figure 2. Variations in urinary DMP levels across dietary phases. Note: Daily variations in urinary DMP concentrations (nmol/l) for adults and children in urban and rural households across all dietary phases. Each point shows the daily mean value for each subgroup. Lines connect actual data points to illustrate the time trend. Days 1–4 = Conventional Phase I; Days 5–11 = Organic Phase; Days 12–16 = Conventional Phase II.

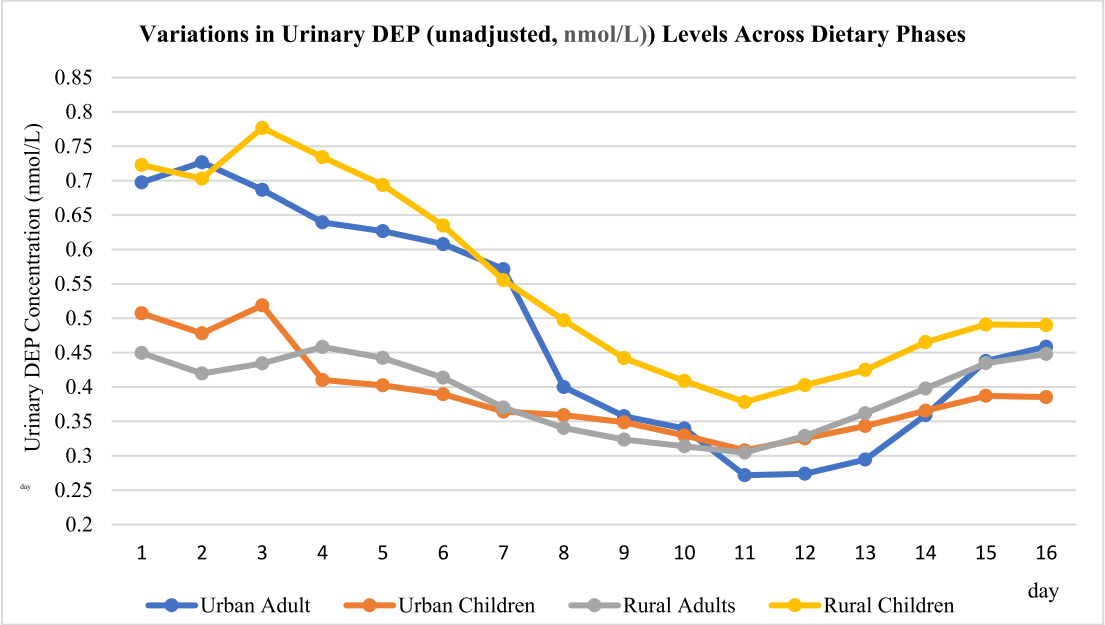

Figure 3. Variations in urinary DEP levels across dietary phases. Note: Daily variations in urinary DEP concentrations (nmol/l) for adults and children in urban and rural households across all dietary phases. Each point shows the daily mean value for each subgroup. Lines connect actual data points to illustrate the time trend. Days 1–4 = Conventional Phase I; Days 5–11 = Organic Phase; Days 12–16 = Conventional Phase II.

These results demonstrate that the organic diet phase significantly reduced urinary OP metabolite concentrations in rural families, with the strongest declines seen for DMTP and DETP in adults and for DEP and DMTP in children.

Figures 2–5 show daily variations in unadjusted urinary DMP, DMTP, and DETP concentrations for adults and children in urban and rural households across all diet phases.

For DMP (figure 2), all groups showed a clear decline during the organic diet phase (Days 5–11) compared to the initial conventional phase (Days 1–4), with concentrations rebounding during the second conventional phase (Days 12–16). Rural children showed the highest DMP levels and the most pronounced rebound. A

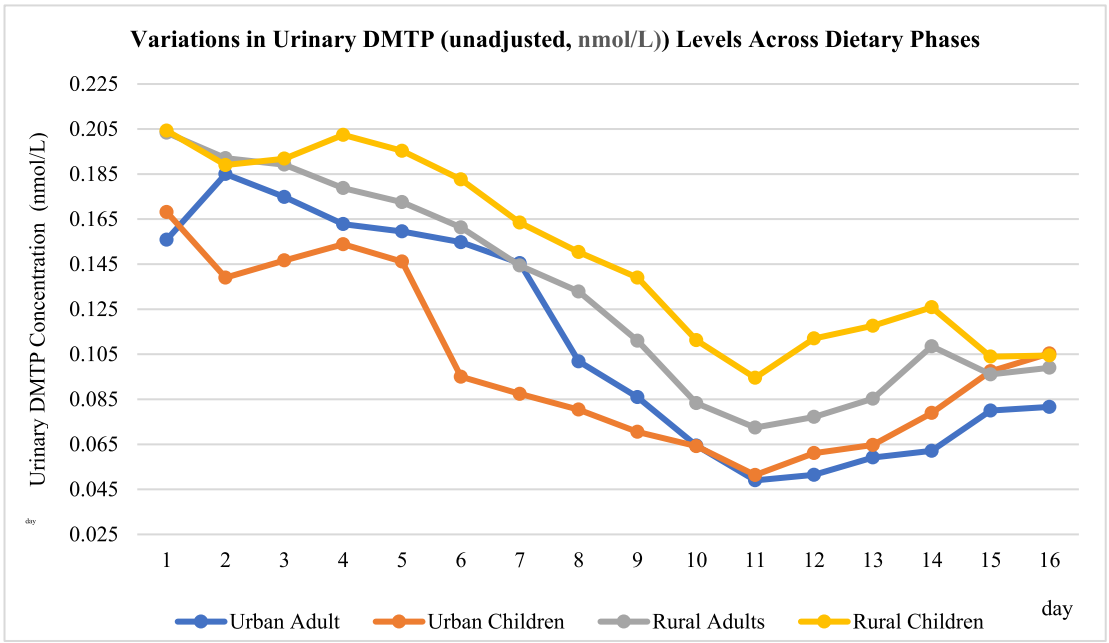

Figure 4. Variations in urinary DMTP levels across dietary phases. Note: Daily variations in urinary DMTP concentrations (nmol/l) for adults and children in urban and rural households across all dietary phases. Each point shows the daily mean value for each subgroup. Lines connect actual data points to illustrate the time trend. Days 1–4 = Conventional Phase I; Days 5–11 = Organic Phase; Days 12–16 = Conventional Phase II.

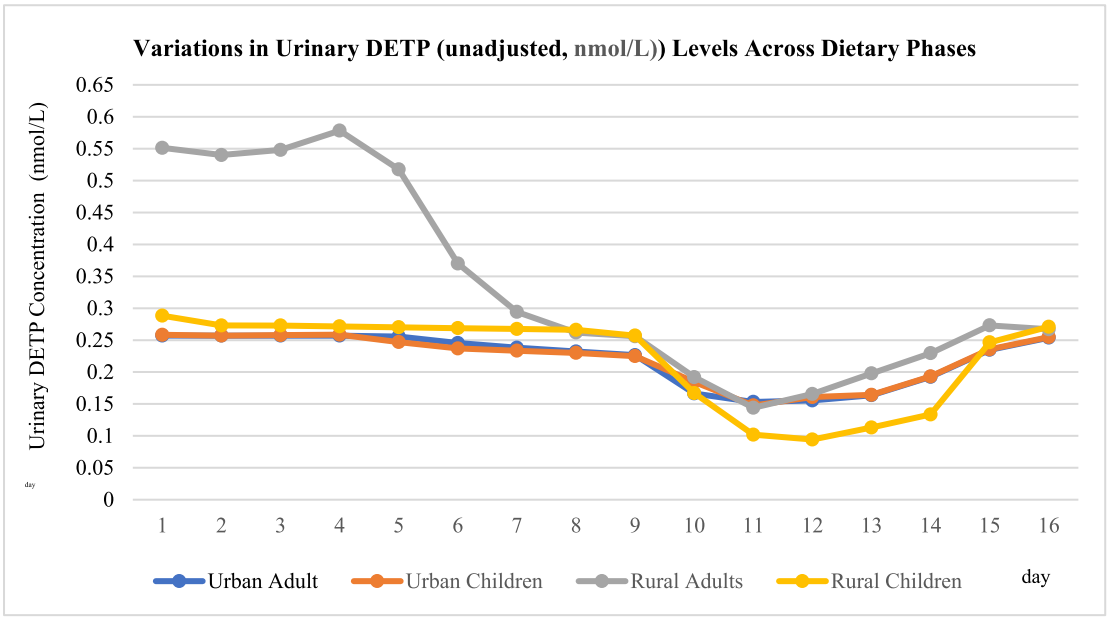

Figure 5. Variations in urinary DETP levels across dietary phases. Note: Daily variations in urinary DETP concentrations (nmol/l) for adults and children in urban and rural households across all dietary phases. Each point shows the daily mean value for each subgroup. Lines connect actual data points to illustrate the time trend. Days 1–4 = Conventional Phase I; Days 5–11 = Organic Phase; Days 12–16 = Conventional Phase II.

similar pattern was observed for DEP (figure 3), where concentrations steadily decreased during the organic phase and rose again afterward. Rural children maintained the highest DEP concentrations throughout.

For DMTP (figure 4), a similar pattern was observed: levels decreased steadily during the organic phase across all groups, with rural children again showing higher concentrations than adults. Rebound effects were more modest for DMTP but still visible after returning to a conventional diet. In contrast, DETP levels (figure 5) remained relatively stable for most groups throughout the study period, with only slight declines during the organic phase and minor rebounds thereafter.

Overall, these daily trends support the mixed-effects model findings, indicating that the organic diet phase effectively reduced urinary concentrations of DMP, DEP, and DMTP, with partial reversals following re-exposure to a conventional diet.

4. Discussion

This repeated measures cross-over study demonstrates that a short-term organic diet can significantly reduce urinary concentrations of key OP pesticide metabolites in rural and urban households in Selangor, Malaysia. The most pronounced declines were observed for DMP, DEP, and DMTP, while DETP showed smaller or more stable changes. These findings align with earlier studies [11, 12] but add new evidence from a tropical middle-income setting where pesticide use remains widespread in both agricultural and household contexts.

Daily trends confirmed that metabolite levels dropped during the organic phase and rebounded when participants returned to their conventional diet, highlighting the temporary nature of diet-based exposure reduction if not sustained. Children consistently showed higher baseline concentrations and larger fluctuations than adults, emphasizing their increased vulnerability to dietary pesticide exposure.

4.1. Urban and rural exposure and the impact of an organic diet

The results demonstrate that both urban and rural households experienced significant reductions in urinary DMP, DEP, and DMTP levels during the organic diet phase, confirming that dietary intake remains a dominant pathway for OP pesticide exposure in Malaysian communities. However, baseline exposure levels differed substantially between groups, with rural participants, particularly children, showing consistently higher initial concentrations. This pattern is likely due to a combination of direct and indirect environmental exposures that amplify dietary intake.

In rural settings, widespread pesticide use for commercial and subsistence farming increases the risk of multiple exposure routes. Beyond residues on food, families may be exposed to pesticide through ambient drift during spraying, deposition on household surfaces, and runoff into local water sources used for drinking or irrigation. Previous studies in agricultural communities have shown that OP pesticide residues can be detected in household dust, children's play areas, and drinking water, contributing to continuous low-level exposure even when dietary intake is reduced [13–15]. Occupational pathways also play a role; parents engaged in farming may inadvertently bring pesticide residues into the home via clothing and equipment, a well-documented 'take-home' pathway that raises household contamination levels.

In addition, the intervention's observed effectiveness with marked by drops in urinary DMP, DEP, and DMTP levels during organic diet phase. This trend reinforces that food choice can rapidly modulate internal pesticide burden, even in populations with ongoing non-dietary exposure. These findings align with prior intervention studies indicating that switching to certified organic produce significantly reduces dietary pesticide residues within a few days [11, 12]. The daily metabolite trends in this study further validate the short half-lives of OP metabolites, with levels dropping during the organic phase and rising again as soon as conventional foods are reintroduced.

The sharper rebound effect among children, especially rural children, highlights both physiological and behavioral vulnerability. Children have higher food consumption relative to body weight, and their developing metabolic pathways may be less efficient at detoxifying OP compounds, extending the biological half-life of metabolites [16]. Moreover, children are more likely to consume fresh local produce such as home-grown fruits and vegetables that may not be certified organic, potentially undermining strict dietary substitution during the intervention. Household food storage practices, informal pesticide applications around homes and gardens, and limited awareness of organic food standards in rural communities may also contribute to residual exposure and faster reaccumulation of pesticide burden.

This urban–rural difference underscores the complexity of pesticide exposure pathways in mixed land-use regions like Selangor, where intensive agriculture and urban settlements coexist. It also highlights that while dietary interventions can yield immediate measurable benefits, their long-term effectiveness in rural populations depends on addressing overlapping environmental and occupational exposure sources, as well as improving access to affordable, trustworthy organic food options.

4.2. Variability in pesticide metabolites and food-specific exposure

The distinct reduction and rebound patterns observed for DMP, DEP, DMTP, and DETP highlight how dietary choices interact with metabolite-specific characteristics, food sourcing, and environmental persistence. The marked declines in DMP and DMTP during the organic diet phase suggest that these metabolites are strongly linked to foods that commonly carry dimethyl OP pesticide residues, primarily fresh fruits, vegetables, and

cereals [17]. These foods often receive pre- and post-harvest pesticide applications to maintain yield and shelf life, especially in tropical climates like Malaysia's, where pest pressure is high year-round.

The sharp DMP rebound seen in urban and rural children after returning to conventional foods indicates how quickly exposure can resume when organic substitutions are not sustained. This is consistent with evidence showing that imported or locally grown conventional produce often carries multiple residues, particularly on items with thin skins or high-water content such as leafy greens, tomatoes, berries, and apples [11, 18]. Children may be more exposed because their diets are richer in fruit and vegetable servings per kilogram of body weight compared to adults, and because processed or ready-to-eat items can also contain pesticide residues from multiple supply chains.

DMTP showed significant reductions across all groups during the organic phase and a visible rebound, especially in urban children. This metabolite reflects exposure to thiophosphate forms of OP pesticides, which are widely used in vegetable farming for pest control in Malaysia. The steeper rebound among urban children may reflect greater reliance on market-bought fresh produce, often conventionally farmed or imported with limited residue monitoring at point of sale. By contrast, rural households may supplement store-bought produce with homegrown vegetables, which can vary in pesticide use depending on family practices and awareness.

DEP displayed a more gradual decline and a moderate rebound, suggesting partial success of the organic phase in lowering exposure. DEP is commonly derived from diethyl-based OP pesticides, which are widely used on staple grains like rice and wheat [19]. In Malaysia, local organic rice options remain limited and more expensive, so families may have continued consuming conventional grains during the organic phase, dampening the full impact of the dietary switch. This finding aligns with past intervention studies showing that pesticide residues can persist when staple grains or imported dry goods are not replaced with certified organic alternatives [11, 17].

DETP levels remained relatively stable across phases, especially among urban groups. This pattern implies that DETP exposure may be less influenced by acute dietary intake and more by environmental or household sources. DETP has been detected in house dust, soil, and stored foods, and can persist due to residual contamination from past pesticide applications or informal household pest control measures [17, 19]. These stable levels suggest that even a strictly managed short-term dietary shift may not fully interrupt low-level chronic exposure to certain metabolites.

Together, these metabolite-specific findings highlight that while switching to organic foods can rapidly reduce exposure to some pesticide residues, the magnitude of benefit depends on the types of food replaced, local agricultural practices, supply chain quality, and continued household behaviors. The variability and partial rebound, especially among children, reinforce that food-specific interventions must be supported by broader improvements in food system transparency, local organic production capacity, and household-level pesticide awareness to sustain risk reduction.

4.3. Environmental exposure and metabolite-specific differences

The observed differences in rebound patterns and baseline levels between urban and rural households underscore the importance of environmental and non-dietary exposure pathways in shaping overall pesticide body burden. While dietary intake remains the dominant route for OP pesticide exposure, rural participants' consistently higher baseline metabolite concentrations indicate additional pathways including direct exposure from nearby agricultural activities, contaminated drinking water, and pesticide drift.

In rural areas of Malaysia, pesticides are frequently applied by smallholder farmers who may use handheld sprayers without adequate buffer zones between farmland and residences. This increases the risk of airborne drift settling on household surfaces and contaminating soil and water sources [13–15]. Rainfall and irrigation runoff can further transport residues into community wells and local streams, sustaining exposure long after active spraying ends. Studies in comparable tropical agricultural communities have shown that even residents who do not apply pesticides themselves can have measurable pesticide residues in household dust, soil near living areas, and indoor air.

Household practices may also amplify this pathway. Farming families often store agrochemicals within or near living spaces, sometimes without proper containment, increasing the risk of accidental spills and chronic low-level contamination. The 'take-home' pathway in which workers unintentionally bring residues home on skin, hair, clothing, and tools is well documented in occupational health research and can elevate children's indoor exposure, especially where separate changing or washing facilities are lacking.

Urban households, by contrast, are less directly exposed to agricultural drift but may face unique non-dietary pesticide sources. Common household pest control products, such as aerosol sprays, insecticides, and mosquito repellents, frequently contain OP or pyrethroid compounds. Routine indoor application of such products, particularly in densely populated residential areas, can lead to pesticide residues accumulating in

household dust and on surfaces where children play and eat [20]. Children's typical hand-to-mouth behavior and floor-level activity mean they are disproportionately exposed to residues that adults are less likely to ingest.

These overlapping exposure pathways may help explain why certain metabolites, especially DETP, showed stable concentrations across phases despite the dietary switch. DETP's persistence suggests that it may reflect chronic low-level exposure from household environments or older residues in soil or stored food, which an organic diet alone cannot fully address. This finding emphasizes that while food substitution can reduce acute pesticide intake, comprehensive exposure reduction strategies must also address household pesticide use practices, safe agrochemical storage, local spraying regulations, and community-level awareness.

4.4. Toxicokinetic difference between adults and children

A consistent pattern throughout this study was that children had higher median urinary OP metabolite concentrations and showed greater fluctuations across dietary phases compared to adults. This difference aligns with established toxicokinetic principles and highlights children's unique physiological vulnerability to pesticide exposure.

First, children consume more food per kilogram of body weight than adults, especially fresh fruits, vegetables, and snack foods that can carry surface pesticide residues [21]. This higher intake means that even low residue levels translate into proportionally greater pesticide burdens for young children. In agricultural communities, this effect may be amplified by children's greater consumption of locally grown produce, which can be less stringently regulated than certified market supply chains.

Second, children's metabolic systems are still developing, with immature liver enzyme pathways and limited detoxification capacity. Studies have shown that the biological half-lives of OP pesticide metabolites like DMP and DMTP can range from 6 to 12 h in healthy adults [9, 21], but may be longer in young children due to slower enzymatic conversion and renal excretion [22]. This means that repeated low-level exposures can accumulate more readily in children than in adults, raising the overall internal dose.

The strong rebound in metabolite concentrations among children after returning to a conventional diet, particularly for DMP and DMTP supports this mechanism. Children's higher baseline levels and faster return to pre-intervention concentrations indicate that their bodies can quickly re-accumulate pesticide residues when exposed through routine meals [23]. This rapid rebound also reflects the short half-lives of these metabolites: once new residues are ingested, measurable metabolites reappear in urine within a day or two.

Behavioral factors further heighten children's risk. Hand-to-mouth behavior, floor play, and poor hand hygiene can increase ingestion of residues from contaminated surfaces, household dust, or produce not washed properly. In rural settings, children may also accompany adults during farm work or play in treated fields, increasing incidental contact with pesticide residues.

Together, these toxicokinetic and behavioral differences demonstrate that while dietary interventions can temporarily reduce pesticide burden in children, a single short-term switch to organic foods is unlikely to provide sustained protection. Effective risk reduction must combine safer household pesticide practices, community education, and improved access to affordable organic or low-residue food options — especially for households with young children in farming regions.

4.5. Integration with global evidence on organic diet interventions

The findings of this study are consistent with a growing body of international evidence demonstrating that organic dietary interventions effectively reduce pesticide exposure among children and adults. In fact, randomized and longitudinal studies across diverse populations have reported significant reductions in urinary pesticide biomarkers following a shift to organic diets.

For instance, a cluster-randomized crossover trial conducted among primary school children in Cyprus [24] found that an organic diet led to substantial decreases in urinary concentrations of organophosphate metabolites and oxidative stress markers. A related study [25] further reported that dietary minimization of glyphosate, and pyrethroids was associated with reduced oxidative stress among children. Similarly, another study [26] detected widespread urinary metabolites of organophosphates and pyrethroids in an Italian pediatric cohort, underscoring the pervasive dietary pesticide exposure even in high-income European contexts.

In line with our findings, these studies particularly noted reductions in DMP and DMTP during the organic diet phase, highlighting their sensitivity to dietary changes. Additionally, a longitudinal study [27] among rural schoolchildren in Chile and found persistent levels of pyrethroid and herbicide metabolites, suggesting that environmental exposure sources such as pesticide drift or contaminated dust may sustain body burden despite dietary improvements. This observation aligns with our findings in rural Malaysian children, who exhibited higher baseline metabolite concentrations and more modest reductions, likely due to ambient agricultural exposures beyond dietary intake alone.

Collectively, these global studies reinforce the conclusion that diet is a modifiable and influential pathway of pesticide exposure. However, the magnitude of benefit varies depending on demographic, dietary, and environmental contexts. Our study contributes to this literature by providing evidence from a tropical, middle-income country setting, and supports the need for integrated risk mitigation strategies that combine dietary interventions with environmental exposure controls and policy support.

4.6. Study limitations and future directions

Although this study provides important evidence on how an organic diet can reduce pesticide exposure, several limitations should be acknowledged. First, the relatively small sample size limits the generalizability of the findings to other regions or populations with different dietary habits, socioeconomic backgrounds, or agricultural contexts. Second, while this study focused on urinary OP metabolites, potential non-dietary sources such as household pesticide use, environmental drift, and contaminated water were not directly measured, which may have contributed to residual exposure, especially among rural families.

Third, dietary compliance relied on daily parental self-reports during specimen collection visits. While families were asked to confirm whether only the supplied organic foods were consumed, and whether any outside food was eaten, no objective checks such as grocery receipts, independent food diaries, or chemical markers were used to verify compliance. This raises the possibility of underreporting or recall bias, although daily face-to-face visits likely helped reduce reporting errors.

Forth, despite employing a crossover design, families were not randomized into counterbalanced sequences (e.g. starting with organic versus conventional diets). All participants followed the same dietary sequence, which may introduce period effects or temporal confounding that randomization could have mitigated. This should be addressed in future crossover studies using randomized intervention sequences to enhance internal validity.

Finally, this study assessed only short-term changes in pesticide body burden and did not measure possible health outcomes associated with reduced exposure. Future research should examine longer intervention periods to evaluate sustained exposure reductions, link biomonitoring with clinical or developmental health endpoints, and test integrated strategies that combine dietary change with safer household pesticide practices. Further studies could also assess policy and market interventions to expand access to affordable certified organic produce and develop practical approaches for lowering pesticide residues in conventionally grown foods.

5. Conclusion

This study demonstrates that switching to an organic diet, even over a short period, can significantly reduce urinary concentrations of organophosphate (OP) pesticide metabolites in both rural and urban households in Malaysia. The reductions were most pronounced for DMP, DEP, and DMTP, while DETP remained relatively stable, highlighting that the effectiveness of dietary interventions is metabolite-specific and shaped by food sources, pesticide use patterns, and environmental persistence.

While urban adults showed clear and consistent benefits, rural participants, particularly children exhibited higher baseline exposures and stronger rebound effects when conventional foods were reintroduced. This underscores how local agricultural practices, environmental pathways such as pesticide drift and take-home exposure, and children's unique dietary behaviors and toxicokinetics interact with dietary measures.

The marked rebound in pesticide metabolites after resuming a conventional diet illustrates that short-term interventions alone may not be sufficient for sustained risk reduction, especially for children who are more physiologically vulnerable. Comprehensive strategies that combine longer-term organic food adoption with improved pesticide regulation, accessible certified organic supply chains, and household education are needed to achieve lasting exposure reduction.

In conclusion, this study supports the role of organic diets as an effective tool for lowering pesticide body burden but emphasizes that dietary change must be part of a broader, integrated approach. Future research should examine longer-term impacts, link biomonitoring with health outcomes, and inform policies that expand safe, affordable food choices for both rural and urban populations.

Ethics statement

This study was conducted in accordance with ethical guidelines and regulations governing research involving human subjects, including the principles outlined in the Declaration of Helsinki. The ethical procedures for this study, including the recruitment of participants, data collection, and handling of sensitive information, were thoroughly reviewed and approved by the Ethics Committee for Research Involving Human Subjects. Ethical clearance was granted under JKEUPM-2022-438, ensuring compliance with national and institutional ethical standards.

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Conflict of interest

The authors have no conflict of interest to declare.

Data availability statement

The data cannot be made publicly available upon publication due to legal restrictions preventing unrestricted public distribution. The data that support the findings of this study are available upon reasonable request from the authors.

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