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ORIGINAL PAPER

Persistent and Non-persistent Organic Pollutants in Human Breast Milk Samples, Association with Lifestyle and Socio-demographic Factors, and Their Genotoxic Effects on Mammary Epithelial Cells

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Abstract

Organic pollutants (OPs) have been associated with increasing rates of cancer, metabolic disorders, and obesity, and may accumulate in human breast milk, creating potential risks for breastfeeding infants. This study assessed the presence and concentrations of OPs in breast milk, including polychlorinated biphenyls (PCBs), organochlorine pesticides (OCPs), and selected non-POPs, and examined associations between pollutant levels and socio-demographic/lifestyle factors, as well as DNA damage in exfoliated mammary epithelial cells among lactating mothers living in Istanbul, Türkiye. In this hospital-based cross-sectional study, breast milk samples were collected and participants completed face-to-face questionnaires on sociodemographic and lifestyle determinants. Genotoxicity in exfoliated mammary epithelial cells was evaluated using the Comet assay. OPs in milk were quantified by gas chromatography and liquid chromatography-tandem mass spectrometry. PCBs were the predominant contaminants and PCB 153, PCB 101, and PCB 180 showed the highest concentrations. Among DDT (Dichlorodiphenyltrichloroethane) metabolites, p,p′-DDT was highest, while γ-HCH was the dominant HCH congener. Several non-POPs (propiconazole, pyriproxyfen, acetochlor, and propargite) were detected. Regular poultry consumption was associated with higher PCB 153 levels. Passive smoking correlated positively with total HCHs and γ-HCH, while active smoking was positively associated with PCB 153. Living in Istanbul for >5 years was linked to higher propiconazole but lower PCB 28, o, p′-DDE, and p, p′-DDE. Comet tail intensity and tail moment were moderately positively associated with PCB 202, o, p′-DDE, and propiconazole. Detectable OP residues were present in breast milk, underscoring potential exposure risks for mothers and infants and supporting ongoing monitoring in urban populations to inform public health strategies.

Keywords Polychlorinated biphenyls · Organochlorinated pesticides · Endocrine-disrupting chemicals · DNA damage · Lifestyle · Breast milk · GC · LC-MS/MS

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Introduction

Non-communicable chronic diseases are a significant contributor to global mortality and morbidity. Non-communicable diseases, including cancers and metabolic diseases such as diabetes and obesity, rank first all over the world, although the causes of mortality and morbidity vary according to socioeconomic level (WHO 2020). Nearly 80% of global non-communicable disease mortality occurs in middle- and low-income countries. Chronic diseases have multifactorial aetiologies that can be broadly classified as environmental, genetic, and individual lifestyle variables. There is an increasing number of environmental health studies on industrial pollutants and human exposure in the literature (Hromić-Jahjefendić et al. 2023).

Production of chemicals has increased dramatically worldwide during the past few decades. Out of the numerous compounds manufactured, it is predicted that around 1000 chemicals may have endocrine disruptor characteristics. Endocrine-disrupting chemicals (EDCs) have been suggested to play a role in the rising global prevalence of cancer, metabolic disorders, and obesity in recent decades (Yavuz et al. 2023; Yilmaz et al. 2020, 2025). These environmental pollutants have the potential to contribute to metabolic changes in individuals, leading to different pathophysiological mechanisms, including adipogenesis, lipogenesis, lipolysis, immunity, disruptions in the intestinal microbiota, and intestinal functioning, specifically when exposure occurs during critical developmental periods such as foetal, early life, and adolescence (Khalil et al. 2023). Among EDCs, POPs are of significant importance. POPs persist in the environment for prolonged duration due to their long half-lives and have considerable toxicity to human health. These compounds are lipophilic and often present in significant amounts, particularly in foods high in fat. Polychlorinated biphenyls (PCBs) and organochlorine pesticides (OCPs) were classified under "Dirty Dozen" at the Stockholm Convention in 2001 (Sandal et al. 2004). PCBs and OCPs bioaccumulate in adipose tissue upon entering an organism. The main route of human exposure to these contaminants occurs through consumption of contaminated food and polluted water, and, to a lesser extent, via inhalation and transdermal absorption. Furthermore, research has demonstrated that they can be transmitted to the developing foetus through placenta and to the infant through nursing (Yilmaz et al. 2020).

PCBs were first mass-produced in the 1930s. Due to their anti-flammable and insulating qualities, they found widespread application as hydraulic fluids, paints, and insulators in electrical components like capacitors and transformers (Carpenter 2006). Despite production bans in most countries in the late 1970 s, they continue to be a global environmental health concern due to their lipophilic nature and persistent properties (Carpenter 2006). OCPs were heavily utilized in agricultural and forestry sectors during the 20th century. They were also widely utilized in Turkish agriculture for a long time (Agus et al. 2022; Daglioglu et al. 2011). Although all OCPs were prohibited after 1985, except for endosulfan and toxaphene, they also resisted environmental degradation due to their persistent properties (Daglioglu et al. 2010). Groundwater, rivers, lakes, and oceans have all been contaminated by these substances. The presence of hexachlorobenzene (HCB) in air samples collected from the North Arctic Region suggests widespread contamination of the Earth's atmosphere with POPs (Ding et al. 2007). Türkiye is one of the countries with PCB- and OCP-contaminated areas (Çok et al. 2012; Erdoğrul et al. 2004 Göl et al. 2023; Ulutaş et al. 2015).

There is extensive research on the detrimental impacts of POPs on human health, such as neurological, reproductive, metabolic, immunological, hepatotoxic and developmental effects (Kutlu et al. 2007; Sandal et al. 2005; Yilmaz et al. 2020). Metabolic disorders (Lamat et al. 2022) such as diabetes mellitus, overweight and obesity, dyslipidaemia, insulin resistance, hepatic steatosis (Liu et al. 2024), change in foetal thyroid hormone levels (Gheidarloo et al. 2020), cardiovascular diseases (Donat-Vargas et al. 2020), hypertension (Park et al. 2016), and autism (Panesar et al. 2020) have been associated with POPs. In addition, several cancers, such as non-Hodgkin lymphoma (Gheidarloo et al. 2020) and liver cancer (Cocco et al. 2000) cases have been attributed to human exposure to these environmental pollutants. However, there is inconsistent evidence regarding breast cancer, and the mechanisms underlying the carcinogenic consequences of PCBs and pesticides remain poorly understood(Liu et al. 2023). It has been suggested that they might imitate or counteract the effects of the organism's natural endocrine hormones by activating oestrogen receptors or xenobiotic response elements on DNA (Cocco et al. 2000).

Non-POP synthetic pesticides (non-POPs), that do not meet Stockholm Convention persistence, bioaccumulation, and long-range environmental transport criteria, constitute a major portion of pesticide active ingredients, and are widely used in agriculture worldwide for crop protection, food preservation and vector control. Non-POP pesticides such as propiconazole have shorter environmental half-lives, reduced bioaccumulation potential, and faster biological destruction than DDTs or PCBs (Yildizdas et al. 2019). Many studies have reported that long-term occupational exposure to non-POP pesticides has adverse health effects. Exposure has been associated with signs of impaired nervous system function, cognitive impairment, changes in sex and thyroid hormone profiles, development of lung and breast cancer,

and development of leukaemia (Kiliç et al. 2005; Ozcan et al. 2004; Shekhar et al. 2024; Uslu et al. 2013; Yilmaz et al. 2006). Residues of non-POP pesticides have been found in human blood, hair and breast milk (Çelik et al. 2021; Yildizdas et al. 2019), so the foetus can be exposed to these pesticides through maternal blood and new-borns through breast milk. Some governments regulate and prohibit non-POPs due to their endocrine-disrupting, reproductive, and carcinogenic effects on human health. Since some of them such as propiconazole and epoxiconazole are banned in EU due to their endocrine disruptor effects, some of them such as pyriproxyfen, and acetochlor are still used in use in many of the countries, including Türkiye (Arena et al. 2017; Yildizdas et al. 2019). Further research is needed regarding their health impact and possible mechanisms, because they are used widely in agriculture and people are exposed to them over time (Gormez et al. 2025).

While the Stockholm Convention covers numerous POPs, the focus of this study is on well-known and historically widely used POPs, particularly polychlorinated biphenyls (PCBs) and organochlorine pesticides (OCPs). These compounds have a high potential for accumulation in human milk due to their highly lipophilic nature and are important in terms of their long-term effects on breast tissue (Ugalde-Resano et al. 2023). According to the International Agency for Research on Cancer (IARC), several compounds have recognized carcinogenic potential; PCBs and lindane (γ-HCH) are classified as Group 1 carcinogens (carcinogenic to humans), while dichlorodiphenyltrichloroethane (DDT) is classified as Group 2 A (probably carcinogenic to humans) (IARC 2018). Selected non-POP pesticides, such as propiconazole, which continue to be widely used in agriculture and may be associated with breast cancer, were also included in the study. We believe this selection process will help assess the potential impacts of both past pollution and current agricultural practices on maternal and infant health.

Our previous study has shown that the Comet Assay method revealed DNA strand breaks in exfoliated epithelial cells without affecting their cell viability and mitotic potential (Agus et al. 2022; Sandal et al. 2008). Accumulation of genotoxic damage in breast tissue caused by lipophilic toxic chemicals might initiate cancer development. It is generally believed that breastfeeding has protective effects against breast cancer. It has been suggested that removal of impaired mammary epithelial cells through breastfeeding during lactation could potentially serve as a protective factor against the development of breast cancer (Arcaro and Anderton 2008). Furthermore, it is crucial to understand the extent of organic contaminants, including both POPs and newer-generation non-POP pesticide compounds in Türkiye and analyse the concentrations of selected organic contaminants in breast milk to assess the potential adverse impacts of these environmental contaminants on newborns.

Therefore, undertaking epidemiological studies to monitor concentrations of pollutants in human samples plays a vital role in identifying the magnitude of the burden and establishing policies to mitigate the risk. Thus, this study aimed to investigate the biomonitoring of selected POP and non-POP concentrations in human breast milk, determine whether these compounds were associated with DNA damage in exfoliated mammary epithelial cells, and examine any relationship with socio-demographic and behavioural determinants in 62 breastfeeding mothers residing in Istanbul.

Materials and Methods

Study Population and Data Collection

The study protocol was approved by the Clinical Ethics Board of Yeditepe University in Istanbul, Türkiye. A total of 62 breast milk samples were collected from healthy lactating mothers at Kanuni Sultan Suleyman Research and Training Hospital and Yeditepe University Hospital. Participants were requested to complete a face-to-face structured questionnaire. The questionnaire used in this study was adapted and modified from a previous study conducted by the same research team to collect data regarding participants' age, number of children breastfed, age at first childbirth, occupational history, dietary habits, duration of residency in Istanbul, family history of breast and other cancers, and height and weight (Agus et al. 2022). The final version of the questionnaire used in this study is provided as Supplementary File 1. Milk samples (10 ml) were acquired through manual hand-pumping techniques and carefully collected in sterile glass bottles, ensuring proper preservation. Subsequently, they were stored at −20 °C to maintain their integrity. The breast milk samples were processed to examine DNA damage in mammary epithelial cells using Comet Assay at Yeditepe University Faculty of Medicine Physiology laboratories. Concentrations of PCBs, OCPs and non-POPs in breast milk samples were analysed at Çukurova University Medical School Forensic Medicine laboratory.

Comet Assay

The experimental protocol has previously been reported (Yilmaz et al. 2012). The Comet assay was performed on freshly isolated mammary epithelial cells within a few hours of sample collection. Briefly, breast milk samples (1 ml) were placed into test tubes. Following mixing with Phosphate-Buffered Saline (PBS) (1:1), the sample was centrifuged at 1500 rpm for 10 min. Then, the milk was

sedimented into three layers where the fat is in the upper part, the middle is serum, and the pellet includes mammary epithelial cells. The serum and fat layers were separated, and mammary epithelial cells were mixed with PBS (500 ul). Prior to embedding, cell viability was determined using 0.4% trypan blue exclusion test; only samples with viability above 85% were included in the Comet assay analysis. Before the experiment, the slides were covered with 0.65% High-Melting Agarose (HMA). Lysis solution and alkaline solution for electrophoresis were prepared.

After the mammary epithelial cells were suspended into PBS solution, it was mixed with 0.65% LMA. The cell suspension was spread on the slides. They were incubated at 4 °C for 10 min. Then the coverslip was removed, and the slides were incubated at 4 °C for 1 h in lysis solution. Following the lysis, the slides were placed on the electrophoresis tank and incubated within the electrophoresis solution for 20 min. Later, the electrophoresis was run at 300 mA for 25 min. At the end, the slides were incubated neutralizing buffer at 4 °C and for 5 min. The procedure was repeated at three times. The slides were exposed to ethidium bromide (20 µg/ml). They were visualized under fluorescent microscope at 200 X magnification. DNA damage assessment was performed by using a software (Comet Assay IV), and Tail Moment (TM) and Tail Intensity (TI) were the comet assay parameters recorded for analysis. The remaining milk samples (after cell isolation) were stored at −20 °C for subsequent chemical analyses of PCBs, OCPs, and non-POPs.

Extraction of Breast Milk Samples for POPs

All breast milk samples were stored at −20 °C until analysed for indicator PCBs, OCPs, and non-POPs. An amount of 7.5 mL of breast milk samples was homogenized by the addition of 7.5 mL of pure water at 1000 rpm for 1 min. At this stage, α-HCH for PCB and OCP analytes and dimethoate-d6 for non-POP analytes were added as internal standards (IS) prior to extraction. IS mix was added at a final concentration of 25 ng/mL. Acetonitrile (15 mL) with (1%) acetic acid was added to the homogenate and incubated at −20 °C for 2 h. After incubation, 2 gr of NaCl was added onto samples and centrifuged at 3000 rpm for 5 min. Liquid (upper) phase was removed and mixed with 7 gr of Na_2SO_4. Later, upper phase was vortexed with the addition of 0.3 gr of PSA and 0.9 gr of $MgSO_4$ and centrifuged again at 3000 rpm for 3 min. No additional C18 sorbent was used in the clean-up step. This approach was selected because PSA-based QuEChERS clean-up has previously been reported as effective for human milk analysis, whereas inclusion of C18 may reduce the recoveries of some lipophilic analytes in milk matrices (Jeong et al. 2012; Luzardo et al. 2013). Upper phase was transferred into glass tube and evaporated under nitrogen gas. The evaporated samples are dissolved in 1 mL of acetonitrile and then applied onto the devices for analysis.

Extraction recovery was evaluated by comparing the peak areas of breast milk samples spiked before extraction with those of corresponding samples spiked after extraction at the same concentration level, and recovery percentages were calculated as (pre-extraction spike area/post-extraction spike area) × 100. Using this approach, recoveries ranged from 67.4% to 109.2% for OCP and PCB analytes and from 64.5% to 124.7% for non-POP analytes. Detailed analyte-specific recovery data are provided in Supplementary Table 1 (Table S1).

Gas Chromatography

Gas Chromatography analysis was performed using an Agilent 6890 N model G.C. equipped with an ECD detector (UK). A silica capillary column (60 m X 0.32 mm DI.) HP-5 (Agilent, USA) was used for chromatographic separation of PCBs and OCPs. Working conditions are injector temperature 250 °C; detector temperature 300 °C; column temperature was 70–280 °C (Temperature program; 2 min at 70 °C; 25 °C/min., 70 to 150 °C; 30 °C/min., 150 to 200 °C; 80 °C/min., 200 to 280 °C; 10 min at 280 °C). Helium was used as the carrier gas. Standard pesticide solutions were prepared in methanol in order to determine the retention times of pesticides by GC. The excellent linearity was obtained in the concentration range that the correlation coefficients (r) were between 0.9970 and 0.9999. For the target PCB and OCP analytes, the Limit of Detection (LOD) values ranged from 0.30 to 7.51 ng/mL, whereas the Limit of Quantification (LOQ) values ranged from 1 to 25 ng/mL. Calibration was performed over the range of 1–500 ng/mL. Samples with concentrations exceeding the calibration range were diluted 1:50 and re-analysed.

Liquid Chromatography Tandem Mass Spectrometry (LC-MS/MS)

A total of 140 selected non-POPs were analysed in breast milk samples using Shimadzu 8040 model LC-MS/MS (Kyoto, Japan). Analytes were separated chromatographically on a Shim-pack column (150 × 2.0 mm ID) purchased from Shimadzu (Tokyo, Japan) at a flow rate of 0.4 mL/min at room temperature. No obvious retention time shift affecting analyte assignment was observed during routine batch analyses. The mobile phase consisted of 5 mM ammonium formate in water (A) and 5 mM ammonium formate in methanol (B). For the positive ionisation mode, the gradient program was as follow: 5% B from 0.0 to 0.5 min, increased linearly to 95% B at 12.50 min, held at 95% B until 17.00 min, and then returned to 5% B at 17.01 min for

re-equilibration. Total run time per sample was 120 min. All selected analytes were analysed in positive ionisation mode (ESI+). Detailed LC-MS/MS acquisition parameters for the target non-POP analytes are presented in Table S2. For the target non-POP analytes, the LOD values ranged from 0.02 to 0.25 ng/mL, whereas the LOQ values ranged from 0.07 to 0.84 ng/mL. Calibration was performed over the range of 1–100 ng/mL.

Statistical Analyses

The statistical analysis was conducted using the SPSS 26.0 software package (SPSS Inc., Chicago, IL, USA). Numbers and percentages represented the categorical variables, whereas the continuous variables were represented by their mean and standard error of the mean. The assessment of normality for the continuous variables was conducted utilizing the Kolmogorov-Smirnov test. The assumption of homoscedasticity was considered violated when the correlations between standardized predicted values and absolute predicted values obtained from linear regression analyses were statistically significant ($p<0.05$). Given that the assumptions of normality and homoscedasticity were violated in the dataset, wild bootstrapping with 2000 replications was utilized in the linear regression analyses to investigate the relationships between categorical data and the outcomes. The bootstrapped Spearman correlation tests were employed to assess the effect sizes and bias-corrected and accelerated (BCa) 95% confidence intervals (CI) of the correlations among continuous variables. Toxic Equivalency (TEQ) values were estimated using WHO 2005 toxic equivalency factors (TEFs). Since PCB 118 was the only dioxin-like PCB analysed in this study, a partial TEQ was calculated using a TEF of 0.00003 (Van den Berg et al. 2006).

Table 1 Socio-demographic and health characteristics of the participants in the study

	n (%)	Mean (± SD)	Min-Max
Age (years) (*n* = 62)	–	28.2 ± 6.6	17–42
> 5 years residency in Istanbul (*n* = 61)	46 (75.4)	–	–
Reproductive health			
Gravidity (*n* = 62)		2.5 ± 1.4	1–7
Age at first birth (*n* = 62)		23.1 ± 5.0	16–39
Nursed children number (*n* = 62)		2.1 ± 1.2	1–7
Health behaviours			
BMI (kg/m2) (*n* = 59)		26.7 ± 4.5	13.9–37.1
Normal	20 (33.9)	–	–
Overweight	26 (44.1)	–	–
Obese	13 (22.0)	–	–
Active smoker (*n* = 62)	7 (11.3)	–	–
Passive smoker (*n* = 61)	28 (45.2)	–	–
Infant characteristics			
Infant gender (*n* = 43)			
Female	18 (41.9)	–	–
Male	25 (58.1)	–	–
Birth weight (kg) (*n* = 42)		3.1 ± 0.7	1.3–4.4
Birth type (*n* = 55)			
Normal	15 (27.3)	–	–
C-section	40 (72.7)	–	–
General health			
Presence of Covid-19 in the last month (*n* = 62)	7 (11.3)	–	–
Oral contraceptive use (*n* = 62)	9 (14.5)	–	–
Prescribed medicine use (*n* = 59)	13 (22.0)	–	–
Supplement use (*n* = 56)	34 (60.7)	–	–
Family cancer history			
Breast cancer (*n* = 61)	6 (9.8)	–	–
Ovarian cancer (*n* = 61)	0 (0.0)	–	–
Other cancers (*n* = 61)	17 (27.9)	–	–

Results

Characteristics of the Study Population

A total of 62 women who were breastfeeding at the time of data collection were included in the analyses. The mean age of the mothers was 28.2 ± 6.6, with a minimum age of 17 and a maximum age of 42. 75.4% of the participants had been residing in Istanbul for over five years. The mean lifetime number of nursed children was 2.1 ± 1.2, whereas the highest was 7. The mean BMI level in the population was 26.7 ± 4.5 kg/m 2, and 66.1% were overweight and obese. The percentages of active and passive smoking were 11.3% and 45.2%, respectively. 9.8% of the mothers had breast cancer among their close relatives (Table 1).

Concentrations of POPs and Non-POPs in Breast Milk

Table 2 summarizes concentrations of all the indicator PCBs and OCPs in the breast milk samples. Among PCB congeners, PCB 153, PCB 101, and PCB 180 had the highest concentrations, with mean values of 1,983.3 ± 2,049.9, 274.4 ± 292.7, and 96.2 ± 156.2 ng/g lipid, respectively. γ-HCH had the highest concentration among HCH congeners. HCB was detected in the highest number of breast milk samples; however, it had the lowest concentration compared to other hexachlorocyclohexanes (HCHs). All DDT metabolites were detected in human breast milk samples, with p, p-DDT and o, p-DDT having the highest concentrations. propiconazole, a systemic fungicide used in agricultural activities, was detected in 16 breast milk samples with a mean level of 18.4 ± 24.3 ng/g lipid. Pyriproxyfen and

Table 2 Concentrations of persistent organic pollutants (POPs) and selected non-persistent pesticides (non-POPs) in breast milk samples (ng/g lipid).*

	n	Mean ± SD	Median	Min-Max
PCBs	57	1,937.1 ± 2,067.3	919.0	10.0–8162.8
PCB 28	21	17.2 ± 8.2	14.4	10.1–38.6
PCB 52	31	24.9 ± 27.0	17.6	5.6–130.3
PCB 101	34	274.4 ± 292.7	115.8	5.1–898.6
PCB 118	14	27.3 ± 41.9	9.5	6.1–158.7
PCB 138	22	32.5 ± 19.7	35.6	5.2–60.0
PCB 153	47	1,983.3 ± 2,049.9	1,481.8	17.6-8,041.8
PCB 180	55	96.2 ± 156.2	49.6	7.7–900.8
PCB 202	46	7.5 ± 7.8	5.3	1.0–49.2
HCB	39	1.4 ± 0.7	1.2	1.0–4.7
HCHs	35	125.0 ± 180.9	82.5	6.6–845.3
β-HCH	26	67.6 ± 41.4	54.4	10.6–152.0
δ-HCH	8	40.9 ± 60.0	9.5	8.3–161.4
γ-HCH	25	91.6 ± 207.3	19.5	6.6–782.8
DDTs	55	229.4 ± 329.5	144.5	5.1–1644.9
o, p-DDE	29	59.8 ± 37.5	50.5	11.7–144.7
p, p-DDE	27	48.5 ± 52.9	28.6	4.2–208.1
o, p-DDD	44	46.3 ± 136.2	12.3	5.2–710.4
p, p-DDD	42	31.1 ± 46.1	16.7	5.1–260.2
o, p-DDT	23	33.9 ± 28.6	29.0	4.2–124.5
p, p-DDT	39	139.8 ± 271.1	45.0	12.9-1,239.7
Non-POPs	28	17.2 ± 21.1	11.4	1.7–109.2
Propiconazole	16	18.4 ± 24.3	11.0	7.6–107.6
Epoxiconazole	1	22.6	22.6	-
Pirimiphos-methyl	1	3.34	3.4	-
Pyriproxyfen	5	4.4 ± 5.3	2.3	1.6–13.8
Propargite	3	31.4 ± 20.0	26.6	14.3–53.4
Pyridaben	1	2.1	2.1	-
Acetochlor	5	3.0 ± 0.7	2.9	2.0–3.8
Dimethomorph	2	14.1 ± 3.0	14.1	12.0–16.2

*Values are presented as mean ± standard deviation (SD), median, and range (min–max) in ng/g lipid. Sample sizes (n) vary across compounds due to concentrations below the limit of detection (LOD). Both mean and median are reported due to skewed distributions of POP concentrations

Acetochlor were detected in 5 breast milk samples; in addition, Propargite was detected in 3 samples, whereas the other non-POPs were detected only in one milk sample (Table 2). Based on the WHO 2005 TEF value, the estimated partial TEQ for PCB 118 was 0.82 pg TEQ/g lipid. However, this value does not reflect the total TEQ since other dioxin-like congeners were not included in the analysis.

DNA Damage in Mammary Epithelial Cells

Representative fluorescence microscopy images of the Comet assay obtained from mammary epithelial cells collected from healthy lactating mothers are shown in Fig. 1. These images were captured using the Comet Assay IV software system, which was also used for quantitative analysis of DNA damage. The mean values of tail intensity and tail moment were 20.4 ± 13.1 and 0.66 ± 0.77, respectively. These two parameters were strongly correlated with a Spearman's Rho coefficient of 0.88 (Table 3). The correlation between tail intensity and the number of nursed children was determined to be significantly negative [B = −8.10, 95% CI = −14.83 to −0.94]. In addition, the association between tail moment and active smoking was determined as significantly positive [B = 0.37, 95% CI = 0.10–0.61] (Fig. 2, Table S3).

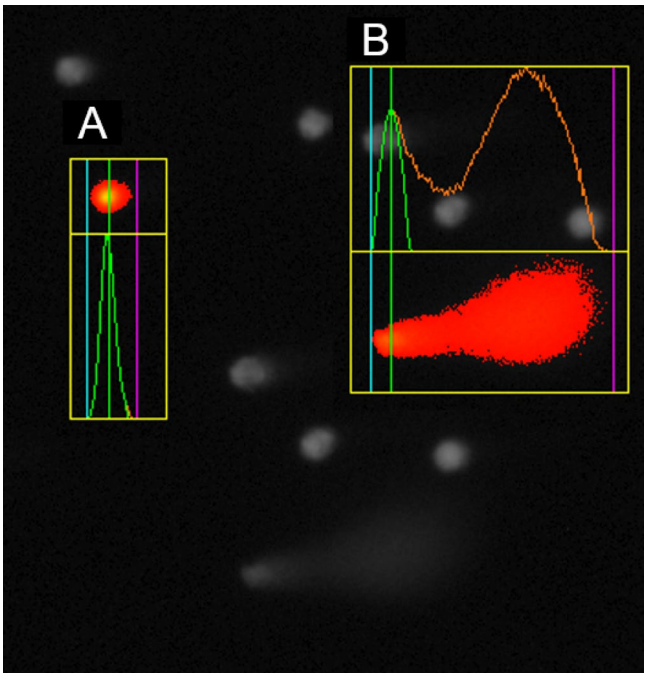


Fig. 1 Representative fluorescence microscopy images obtained from the Comet Assay IV Imaging System for the Comet assay performed on mammary epithelial cells collected from healthy lactating mothers. **A** An undamaged cell (no DNA damage, tail intensity/moment). **B** A cell with high DNA damage (high tail intensity/moment), exhibiting a prominent tail indicative of DNA strand breaks. Magnification: 40×

Table 3 DNA damage parameters measured by comet assay in breast milk samples and their correlation.*

	n	Mean (± SD)	Median	Min-Max
Tail intensity (%)	52	20.4 ± 13.1	18.6	3.9–75.9
Tail moment (arbitrary units)	52	0.7 ± 0.8	0.5	0.04–4.43
		Spearman's rho		BCa 95% CI
Tail intensity – Tail moment		0.88		0.79–0.94

*Values are presented as mean ± SD, median, and range. Spearman's correlation coefficient (ρ) and bias-corrected and accelerated (BCa) 95% confidence intervals are shown

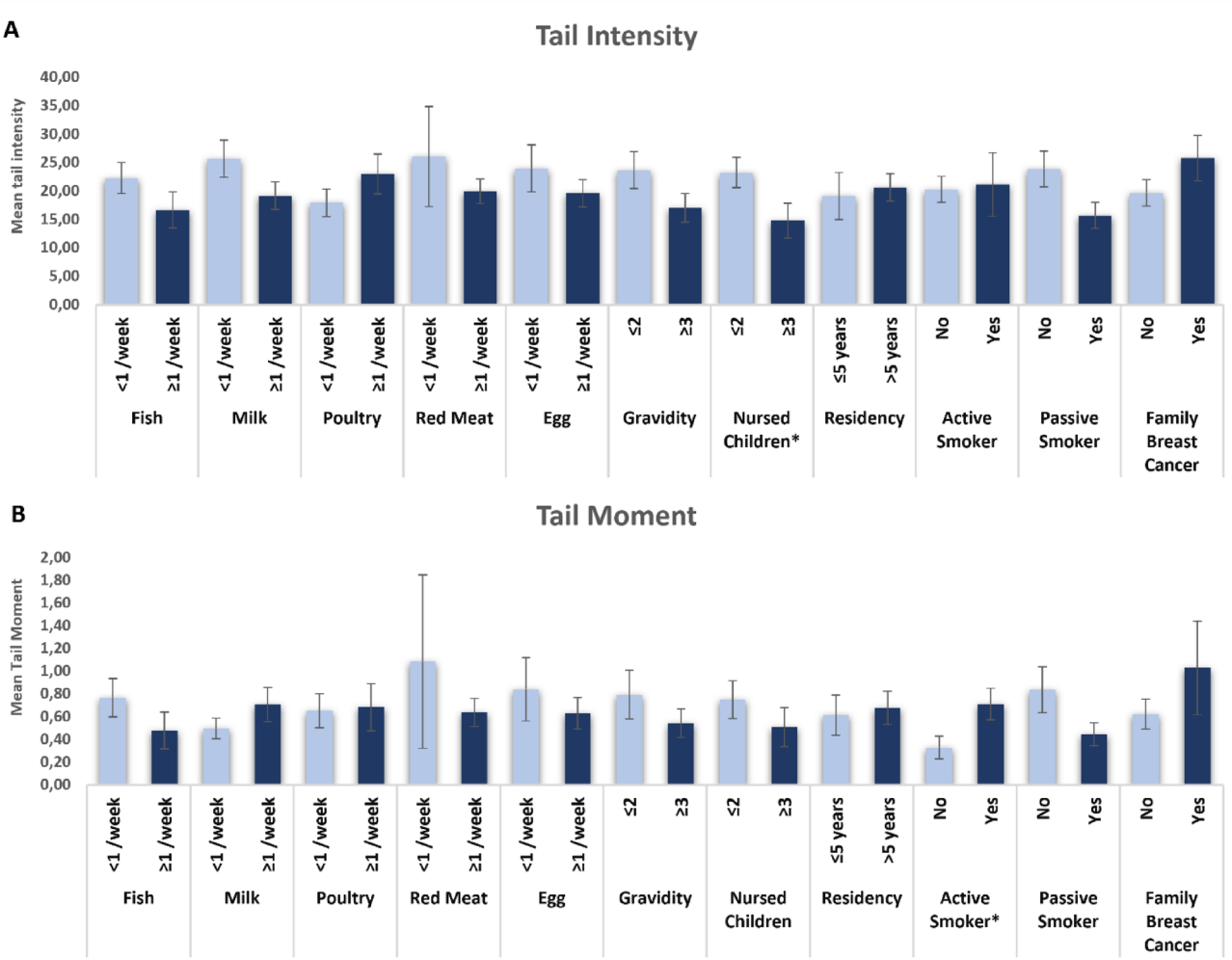


Fig. 2 Associations between selected dietary, lifestyle, and reproductive factors and DNA damage indicators measured by the comet assay. Panels show **A** tail intensity and **B** tail moment. Bars represent mean values with standard errors (SE). Statistical significance was determined based on bootstrapped linear regression models, and significant associations ($p<0.05$) are indicated with an asterisk (*). Regression coefficients are presented in Table S3

Correlations Between Pollutant Levels and Maternal and Infant Characteristics

Table 4 presents the correlations between the concentration of POPs and several factors including the mother's age, age at first birth, infant birth weight, maternal BMI, and DNA damage. The age of the mother showed a positive correlation with PCB 180, DDTs, and p, p-DDD. Rho coefficients along with their 95% confidence intervals were reported as follows: 0.57 (0.21–0.79), 0.43 (0.10–0.67), and 0.51 (0.13–0.77), respectively. The age at first birth showed a positive correlation with PCB 118 and p, p-DDD, while it exhibited a negative correlation with o, p-DDE. The Rho coefficients and their 95% confidence intervals were 0.55 (0.00–0.88), 0.59 (0.21–0.83), and −0.65 (−0.88- −0.12), respectively. The birth weight of the infant showed a negative correlation with HCB and propiconazole, with Rho coefficients and 95% confidence intervals of −0.41 (−0.71 to −0.01) and −0.75 (−1.00 to −0.09), respectively. The primary dependent variables of the study, tail intensity and tail moment, exhibited a positive moderate correlation with PCB 202, with Rho co-efficient and 95% confidence intervals of 0.58 (0.32–0.73) for tail intensity and 0.62 (0.42–0.74) for tail moment. Similarly, correlations with o, p-DDE were noted, with Rho coefficients of 0.53 (0.11–0.92) for tail intensity and 0.54 (0.04–0.86) for tail moment. For propiconazole, the Rho coefficients were 0.57 (0.13–0.81) for tail intensity, and 0.64 (0.18–0.96) for tail moment. Conversely, a negative correlation was observed with γ-HCH, where the Rho values for TI and TM were −0.73 (−0.96 to −0.28) and −0.67 (−0.92 to −0.18), respectively. The mother's BMI did not demonstrate any notable impact on chemical exposure (Table 4).

Table 4 Spearman's correlation coefficients and BCa 95% confidence intervals for associations between pollutant concentrations and maternal/infant characteristics and DNA damage parameters

A)	Spearman's rho (BCa 95%CI)								
	PCBs	PCB-28	PCB-52	PCB-101	PCB-118	PCB-138	PCB-153	PCB-180	PCB-202
Age	0.13 (−0.17-0.43)	−0.10 (−0.78-0.77)	0.04 (−0.47-0.53)	0.34 (−0.10-0.69)	0.13 (−0.58-0.71)	−0.35 (−0.71-0.23)	−0.01 (−0.36-0.37)	**0.57** (0.21–0.79)**	−0.05 (−0.48-0.39)
Age at 1 st birth	−0.06 (−0.43-0.33)	−0.58 (−0.99-0.21)	−0.23 (−0.68-0.26)	−0.15 (−0.51-0.25)	**0.55* (0.00–0.88)**	−0.07 (−0.77-0.70)	0.01 (−0.35-0.37)	0.24 (−0.16-0.58)	−0.24 (−0.62-0.18)
Birth weight	−0.02 (−0.41-0.36)	0.08 (−0.74-0.79)	0.24 (−0.33-0.66)	0.14 (−0.35-0.57)	0.27 (−0.47-0.82)	−0.50 (−0.94-0.16)	0.04 (−0.35-0.43)	0.05 (−0.34-0.42)	−0.20 (−0.54-0.18)
BMI	−0.10 (−0.40-0.19	−0.20 (−0.65-0.38)	−0.13 (−0.42-0.21)	−0.21 (−0.50-0.11)	−0.11 (−0.65-0.55)	−0.09 (−0.53-0.39)	0.04 (−0.25-0.34)	−0.09 (−0.35-0.20)	−0.03 (−0.037-0.28)
Tail intensity	0.11 (−0.24-0.45)	0.20 (−0.46-0.76)	0.29 (−0.23-0.72)	0.09 (−0.39-0.52)	−0.15 (−0.95-0.94)	0.50 (−0.36-0.91)	0.05 (−0.33-0.46)	0.10 (−0.28-0.43)	**0.58** (0.32–0.73)**
Tail moment	0.19 (−0.15-0.51)	0.07 (−0.60-0.68)	0.44 (0.08–0.69)	0.06 (−0.44-0.52)	−0.05 (−0.94-1.00)	0.44 (−0.58-1.00)	0.14 (−0.23-0.53)	0.05 (−0.33-0.42)	**0.62** (0.42–0.74)**

B)	DDTs	o, *p*-DDE	*p*, *p*-DDE	o, *p*-DDD	*p*, *p*-DDD	o, *p*-DDT	*p*, *p*-DDT
Age	**0.43* (0.10–0.67)**	−0.42 (−0.76-0.15)	0.00 (−0.50-0.55)	0.23 (−0.27-0.62)	**0.51* (0.13–0.77)**	0.14 (−0.46-0.65)	0.25 (−0.21-0.67)
Age at 1 st birth	−0.02 (−0.38-0.37)	**−0.65** (−0.88- −0.12)**	0.00 (−0.45-0.45)	−0.03 (−0.47-0.39)	**0.59** (0.21–0.83)**	0.08 (−0.53-0.54)	0.06 (−0.40-0.52)
Birth weight	−0.11 (−0.44-0.23)	−0.02 (−0.65-0.57)	0.06 (−0.46-0.50)	−0.34 (−0.69-0.14)	0.16 (−0.27-0.53)	−0.36 (−0.86-0.32)	−0.02 (−0.40-0.34)
BMI	−0.11 (−0.39-0.14)	−0.08 (−0.48-0.30)	−0.15 (−0.57-0.27)	0.09 (−0.25-0.44)	0.14 (−0.21-0.44)	−0.22 (−0.65-0.24)	0.23 (−0.09-0.57)
Tail intensity	0.05 (−0.31-0.38)	**0.53* (0.11–0.92)**	−0.16 (−0.67-0.44)	0.08 (−0.27-0.44)	0.13 (−0.36-0.57)	−0.13 (−0.63-0.42)	0.12 (−0.38-0.61)
Tail moment	0.06 (−0.25-0.39)	**0.54* (0.04–0.86)**	−0.19 (−0.69-0.39)	0.11 (−0.27-0.49)	0.06 (−0.39-0.51)	−0.15 (−0.62-0.41)	0.25 (−0.22-0.65)

C)	HCHs	HCB	β-HCH	δ-HCH	γ-HCH	Non-POPs	Propiconazole
Age	0.27 (−0.24-0.73)	−0.10 (−0.53-0.38)	0.09 (−0.51-0.67)	0.00 (−0.67-1.00)	0.00 (−0.55-0.56)	0.11 (−0.51-0.70)	−0.71 (−1.00-0.06)
Age at 1 st birth	0.03 (−0.45-0.51)	−0.10 (−0.59-0.40)	−0.21 (−0.70-0.43)	−0.50 (−1.00-0.88)	0.00 (−0.74-0.67)	0.04 (−0.58-0.59)	−0.49 (−0.96-0.17)
Birth weight	0.11 (−0.50-0.63)	**−0.41 (−0.71- −0.01)**	0.26 (−0.38-0.73)	−0.12 (−0.71-0.56)	−0.12 (−0.61-0.42)	0.17 (−0.49-0.72)	**−0.75 (−1.00- −0.09)**
BMI	0.09 (−0.28-0.44)	0.00 (−0.31-0.28)	0.18 (−0.26-0.57)	−0.69 (−1.00-0.44)	−0.05 (−0.50-0.43)	0.25 (−0.17-0.60)	−0.19 (−0.69-0.43)
Tail intensity	−0.33 (−0.81-0.25)	0.11 (−0.44-0.63)	0.35 (−0.12-0.70)	−0.22 (−1.0-0.87)	**−0.73** (−0.96- −0.28)**	0.21 (−0.34-0.64)	**0.57 (0.13–0.81)**
Tail moment	−0.22 (−0.74-0.45)	0.07 (−0.49-0.61)	0.36 (−0.11-0.68)	−0.18 (−1.0-0.77)	**−0.67** (−0.92–0.18)**	0.12 (−0.47-0.66)	**0.64 (0.18–0.96)**

Values are Spearman's rank correlation coefficients (ρ) with bias-corrected and accelerated (BCa) 95% confidence intervals. Significant correlations are indicated in bold (*$p<0.05$, **$p<0.01$)

Associations Between Pollutant Levels and Lifestyle and Socio-demographic Factors

The relationships between HCB, total HCHs, DDTs, and PCBs with the categorical variables evaluated in the study are illustrated in Fig. 3 and Table S3. There was a significant correlation between PCBs and active smoking [B = 1598.8 (867.4–2382.6)], while HCHs showed a significant correlation with passive smoking [B = 119.8 (8.5–241.3)]. The analysis revealed a positive correlation of HCB and DDTs with red meat consumption, with coefficients of B = 0.38 (0.2–0.6) for HCB and B = 173.78 (65.1–282.0) for DDTs. Additionally, an analysis of the relationships between the congeners of these pollutants and the variables revealed that the level of PCB 153 was significantly elevated in individuals who consumed poultry more than once

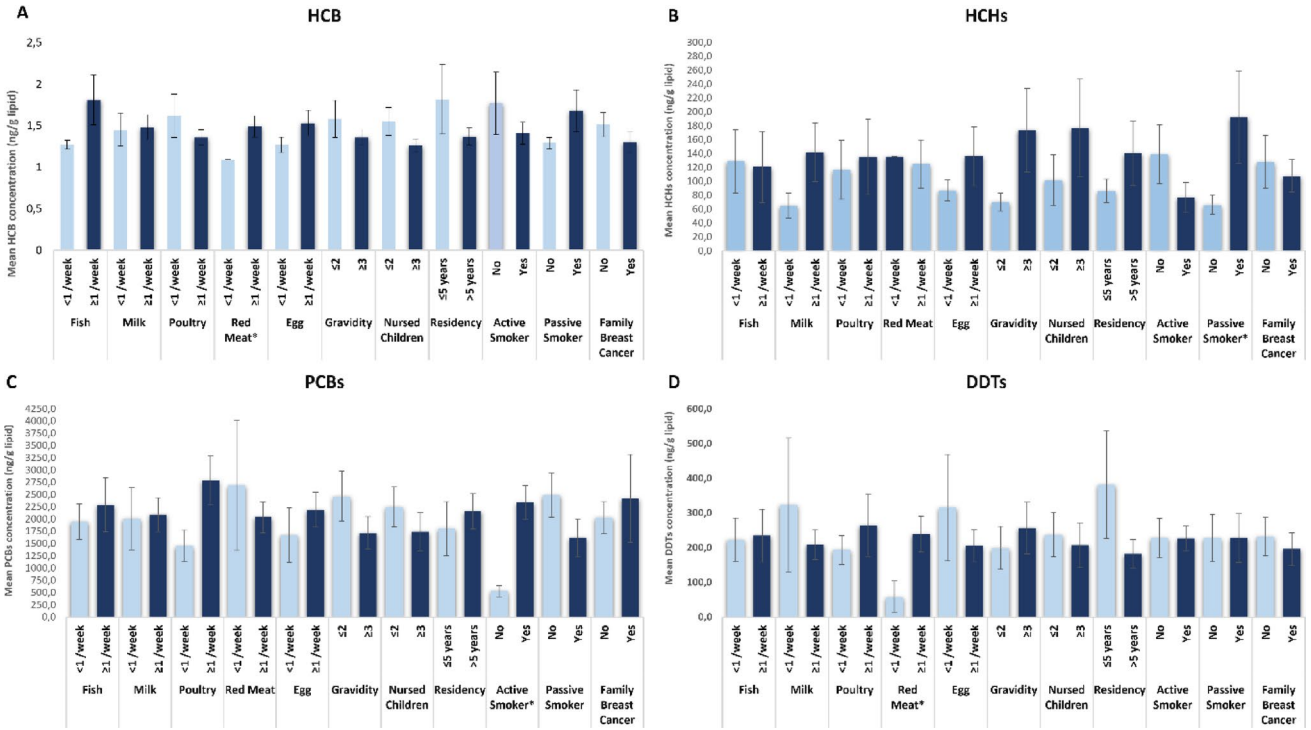


Fig. 3 Associations between selected dietary, lifestyle, and reproductive factors and concentrations of organochlorine compounds in breast milk. Panels show **A** HCB, **B** HCHs, **C** PCBs, and **D** DDTs (ng/g lipid). Bars represent mean concentrations with standard errors (SE). Statistical significance was determined based on bootstrapped linear regression models, and significant associations ($p < 0.05$) are indicated with an asterisk (*). Regression coefficients are presented in Table S3

a week compared to those who consumed it less frequently [B = 1513.2, SE = 624.1, 95%CI = 138.1–3207.3]. There was a positive correlation between γ-HCH and passive smoking [B = 143.6, SE = 71.0, 95% CI = 15.2–293.0]; additionally, active smoking status showed a positive relationship with PCB153 [B = 2122.5, SE = 321.0, 95%CI = 1509.4–2730.2]. Individuals with a family history of breast cancer showed elevated levels of PCB 138 compared to those without such a history [B = 18.9, SE = 7.8, 95%CI = 4. 7–32.5]. Individuals who had lived in Istanbul for over five years showed elevated levels of propiconazole [B = 11.9, SE = 6.3, 95%CI = 0.01–23.2] and γ-HCH [B = 118.6, SE = 65.5, 95% CI = 1.4–236.3]. Conversely, PCB28 [B = −8.6, SE = 4.2, 95% CI = −16.3- −3.2], o,p-DDE [B = −41.4, SE = 11.9, 95%CI = −62.6- −24.0], and p, p-DDE [B = −69.5, SE = 27.1, 95%CI = −114.2- −38.0] levels were higher among mothers who had lived in Istanbul for less than five years compared to long-term residents. The mothers with fewer than three pregnancies exhibited lower levels of PCB28 [B = 6.6, SE = 3.3, 95% CI = 0.5–12.3], while displaying higher levels of PCB153 [B = −1030.3, SE = 604.6, 95%CI = −2064.4- −138.8]. The number of nursed children showed a negative correlation with PCB101 [B = −27.6, SE = 14.1, 95%CI = −51.5- −8.8], and p, p-DDD [B = −22.4, SE = 10.4, 95%CI = −42.0- −5.6] (Fig. 4, Table S4).

Discussion

The present study has shown that PCBs, OCPs, and selected non-POPs were detected in human breast milk samples collected from healthy breastfeeding mothers residing in Istanbul. PCBs appeared as the primary pollutants as they were at higher concentrations than OCPs. Specifically, PCBs 153, 101, and 180 exhibited the most prominent presence among the various PCB congeners analysed. Our findings have also demonstrated that p, p-DDT exhibited the highest concentration among the multiple compounds of DDTs, while γ-HCH was the highest detected pollutant among the HCH congeners found in the human breast milk. In addition, propiconazole, pyriproxyfen, acetochlor, and propargite were also present in the breast milk samples. These findings emphasize the ongoing environmental exposure to POPs. Several statistically significant correlations were observed: higher levels of DDTs were positively correlated with maternal age and red meat consumption; elevated levels of PCB 153 were correlated with active smoking and frequent poultry consumption; and higher levels of γ-HCH were correlated with passive smoking. PCB 202, o, p-DDE, and propiconazole levels showed positive correlations with DNA damage markers, including tail intensity and tail moment. In contrast, an unexpected inverse association was observed

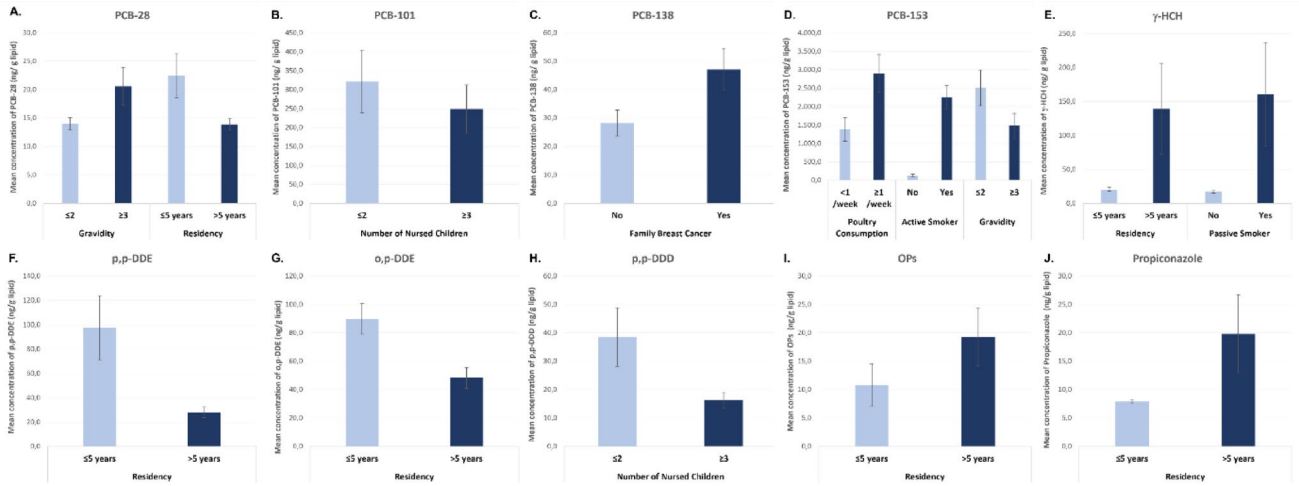


Fig. 4 Associations between selected lifestyle, dietary, and reproductive factors and concentrations of organochlorine compounds in breast milk. Panels show results for **A** PCB-28, **B** PCB-101, **C** PCB-138, **D** PCB-153, **E** γ-HCH, **F** p, p′-DDE, **G** o, p′-DDE, **H** p, p′-DDD, **I** OPs, and **J** propiconazole (ng/g lipid). Bars represent mean concentrations with standard errors (SE). Only statistically significant associations ($p < 0.05$) are presented. Statistical significance was determined using bootstrapped linear regression models. Regression coefficients are presented in Table S4

between γ-HCH and DNA damage parameters, despite its well-known toxic and carcinogenic properties. Additionally, the accumulation of different pollutants in the body was significantly influenced by factors such as the number of births, the duration of breastfeeding, and the number of years of residence in Istanbul.

It is generally accepted that the primary route of human exposure to PCBs is through consumption of contaminated food, and to a lesser extent, through both outdoor and indoor inhalation and dermal contact (Carpenter 2006; Yilmaz et al. 2020). Food consumption, particularly fish consumption, has been extensively investigated for evaluating transmission of PCBs to humans (Marushka et al. 2021; Simpson et al. 2023). However, several studies have also examined exposure through beef, dairy products, chicken, and eggs (Montano et al. 2022). In a study conducted in the US, all 203 PCB congeners in various food items were examined, where animal food other than fish consumption played an essential role in PCB dietary exposure. It was revealed that PCB concentrations in animal food clearly declined over the past two decades (Saktrakulkla et al. 2020). Our study has shown that concentrations of PCB 153 were associated with chicken consumption; additionally, HCB and DDTs levels were associated with red meat consumption. Unlike other studies, no notable association was observed between PCB levels and consumption of fish, milk, and eggs in the present study. In various studies, PCB levels differ in red meat and poultry. PCB levels were higher in poultry than in red meat in a study conducted in Germany (Weber et al. 2018), whereas HCB levels were higher in red meat than in poultry in China (Chen et al. 2024), very similar to our study. On the contrary, a study conducted in the US suggested that although poultry has detectable PCB levels, they are often lower than those present in fish and red meat (Saktrakulkla et al. 2020). It was concluded that several factors such as longer life span, more fat tissue in cattle, differences in metabolism, and differences in environmental exposure may lead to different levels of PCBs and DDTs in red meat and poultry. These results demonstrate the importance of source-specific contamination pathways and regional variations in POP exposure. Origin of PCBs and OCPs from different places could explain why the dietary sources that contain them are different. PCBs, which are synthetic industrial chemicals, mostly get into the food chain through contaminated feed, industrial waste, and air pollution that settles in agricultural areas. However, chemicals like DDT and HCB were widely employed in farming and vector control, which contaminated fields and soil for a long time and influenced grazing animals in ecosystems. Animals' foraging habits have a big effect on the types of food and environmental components they consume, which in turn determines how they are exposed to POPs (Chen et al. 2024; Saktrakulkla et al. 2020).

Another route of PCB exposure is indoor and outdoor inhalation. Thermal activities include garbage incineration, steel production, and coal and wood combustion in residential and commercial properties; power generation, transformers and electrical capacitors are among the primary outdoor PCB emitters (Montano et al. 2022). In addition, PCBs can travel great distances via deposition and resuspension (Choi et al. 2008). They are commonly detected in high concentrations in urban areas and in proximity to industrial sites (Hombrecher et al. 2021). The findings of a study conducted at an industrial site in Türkiye showed

presence of PCBs in both the soil and ambient air, which were influenced by seasonal temperature changes (Bozlaker et al. 2008). Furthermore, indoor air can be examined for various PCB congeners, as they are commonly found in oil additives used in paints and building materials like window caulking, ceiling tiles, and floor tiles (Anh et al. 2021). PCBs are frequently encountered as commercial mixtures known as Aroclors, often present in older buildings (Carpenter 2006). According to a recent study, the levels of total indoor PCBs assessed in three schools exceeded the recommended limits set by the State for children of all ages. Additionally, these levels were beyond the limits recommended by the US EPA, specifically for children under the age of 3 (Hua et al. 2023). In the present study, PCB congeners were identified as the primary pollutants. In a review of global surveys conducted by the WHO and UNEP, the global levels of PCBs in human milk were generally highest in East and West Europe. Levels of PCBs in human milk remained significantly above toxicologically safe thresholds, which was 7 ng/g lipid, posing potential risks, especially from prenatal exposure (Van den Berg et al. 2017a). In a review conducted in China, concentrations of PCBs were higher in coastal and industrialized regions. Despite the declining trend over time due to regulatory measures, average concentration of non-dioxin-like PCBs congeners (PCB 28, 52, 101, 138, 153, and 180) was 9.63 ng/g lipid (Hu et al. 2021). In our study, PCB levels were considerably higher than those discovered in studies conducted in different regions worldwide (Fernandez et al. 2008; Hu et al. 2021), including Türkiye (Agus et al. 2022; Akgöl and Kanat Pektaş 2023). The breastfeeding mothers were enrolled in the present study from a public hospital. It is possible that these individuals may be exposed to elevated levels of pollutants in the buildings and neighbourhoods where they reside. It has been reported that various human populations may have varying levels of exposure to PCBs due to differences in health behaviours and living environment (Montano et al. 2022).

It has been reported that PCB 153 is the most commonly detected congener in human blood and tissues (Webster et al. 2013). PCBs 118, 138, 153 and 180 have been identified as the most commonly detected congeners in urine and blood serum samples in breast cancer patients worldwide (He et al. 2017). Thus, the presence of PCB compounds in breast milk samples in our study is of importance. Indeed, our analysis revealed an association between the PCB 138 levels and the family history of breast cancer in the lactating mothers in the present study. A study examined the relationship between PCB concentrations in breast adipose tissue and the development of breast cancer discovered that PCB-153 and PCB-138 were the most common congeners, with PCB-153 positively correlated with the development of breast cancer (He et al. 2017). According to a meta-analysis, PCB-99, PCB-183, and PCB-187 are among the non-dioxin-like PCB congeners that have been connected to an increased risk of breast cancer (Leng et al. 2016). In another study with similar results, non-dioxin-like PCBs (PCB-74, 99, 194, and 201) were linked to breast cancer (Parada et al. 2021). PCB congeners and their concentrations in relation to breast cancer have a complex relationship that varies depending on the congener. According to different research findings, these congeners present varying levels of toxicity and related risks. The suggested non-linear relationship between PCB levels and breast cancer risk raises the possibility that different congeners may have different effects and that different exposure levels may have different implications for the development of breast cancer. Different PCB congeners display unique behaviors that are impacted by several variables related to their chemical characteristics, biological interactions, and environmental dynamics. Significant variations exist between dioxin-like congeners and non-dioxin-like PCBs in how they interact with receptors, which may influence their biological effects and carcinogenic potential (Leng et al. 2016; Parada et al. 2021). In addition to PCBs, organochlorine pesticides and certain non-POP pesticides, such as propiconazole, may contribute to breast cancer risk through endocrine disruption, estrogen receptor interaction, and oxidative stress pathways (Gomez et al. 2023; Macedo et al. 2023). OCPs such as DDT and its metabolites (particularly p, p'-DDE) are well-known endocrine disruptors with weak estrogenic/anti-androgenic properties that can activate or antagonize estrogen receptors (Adami et al. 1995). Due to these properties, they are thought to play a role in the development of hormone-sensitive breast cancer. However, epidemiological studies have struggled to find a consistent association between OCP exposure and breast cancer risk, which may be attributed to factors such as timing of exposure (e.g., critical developmental windows), individual genetic susceptibility, and complex interactions among different OCP mixtures (Ritte et al. 2013). The positive correlation observed in our study between o, p'-DDE and genotoxicity parameters suggests a hypothesis that this metabolite may contribute to breast carcinogenesis through DNA damage.

Propiconazole, a triazole fungicide, has the potential to inhibit CYP450 enzymes involved in steroidogenesis in mammals with a particular effect on aromatase (Sanderson et al. 2002; Skolness et al. 2013). Inhibition of aromatase may alter the hormonal balance in breast tissue by reducing the conversion of androgens to estrogens. Although there are limited human studies directly linking propiconazole to breast cancer, animal studies and in vitro models suggest that this compound may increase proliferation or induce genotoxic damage in mammary epithelial cells through its endocrine-disrupting effects and the induction of oxidative

stress (Authority et al. 2017). The positive correlation found in our study between propiconazole levels and DNA damage (tail intensity and tail moment) in mammary epithelial cells supports this mechanism and raises the possibility that propiconazole exposure may contribute to genotoxic damage in breast tissue, thereby potentially increasing cancer risk. These mechanisms may also help explain the DNA damage findings observed in our comet assay analysis, providing a plausible link between pollutant exposure and breast carcinogenesis. This finding underscores the need for more comprehensive epidemiological and mechanistic studies on the long-term health effects of propiconazole and other non-POP pesticides.

The technical HCH, which is primarily synthesized through chemical reaction of chlorine gas and benzene in industrial manufacturing, is typically a mixture of multiple stereoisomers consisting of four main compounds (Al-Obaidi et al. 2022; Gotelli et al. 2020). Among those isomers, γ-HCH, also known as lindane, is the most potent component of technical HCH for pest control. The γ-HCH compounds in agricultural soils have half-lives of 20–50 days, whereas α-HCH compounds have half-lives of 20 weeks (Gotelli et al. 2020). The isomers δ-HCH and β-HCH exhibit greater resistance to environmental degradation compared to α-HCH and γ-HCH (Gotelli et al. 2020). Therefore, the presence of δ-HCH and β-HCH residues can serve as reliable indicators of the past utilization of HCH in agricultural areas (Zhang et al. 2024). Previous studies conducted in Türkiye assessed the levels of HCHs, specifically in the lipid fraction of human breast milk, at different time intervals. The HCH profile of human breast milk exhibited geographical variations, with ß-HCH consistently being the most prevalent among other HCHs across all populations examined (Çok et al. 2012; Erdoğrul et al. 2004). In the cross-sectional population in the present study, HCH levels were notably higher compared to previous studies, except for the one conducted in Adiyaman and Kahramanmaraş, the two south-eastern regions known for their extensive agricultural practices. Furthermore, our analysis revealed that γ-HCH had the highest concentration in breast milk. The use of pure lindane rather than technical lindane might be indicated by this profiling pattern of HCH isomer in our study. Compared to other isomers, the high potency and short half-life of γ-HCH suggests that its presence in specific rural farming areas may be due to unlawful usage or insufficient monitoring (Chandra Yadav et al. 2020).

In the present study, there was a positive correlation of passive smoking with HCHs and γ-HCH concentrations, as well as an association of active smoking with PCBs and PCB153. Pesticide residues in tobacco products are a well-documented consequence of using pesticides for control (Quadroni and Bettinetti 2019). The association between smoking and certain pesticides can be attributed to the availability of the remaining stock of tobacco following the prohibition all around the world, as well as the continued use of pesticides in areas where tobacco is cultivated (O'reilly and Yarto 2013).

The persistence of DDTs and their metabolites in the environment can extend up to 150 years in water and 30 years in soil. DDT and its metabolites, DDE and DDD, were discharged into the environment during their manufacturing and extensive application as an insecticide for agriculture and malaria prevention. DDT was banned in several countries after 1972. However, since the WHO agreed on its use in indoor spraying, a highly effective method for controlling disease vectors, it is currently utilized in countries where malaria poses a significant health concern (IARC 2016). In the WHO's global survey, elevated levels of DDTs were mainly found in tropical regions where DDT is used for malaria control. According to WHO's safety threshold concentration for DDTs in breast milk, which was 2300 ng/g lipid, DDTs levels were within or below safety thresholds in most regions, posing less risk compared to other contaminants (Van den Berg et al. 2017a).

In environmental studies, the presence of DDTs and their by-products was assessed in surface water, particularly in regions where DDTs are employed for malaria control near bodies of water (Amdany et al. 2014). The exposure of rivers, seas, and wells to DDTs through direct transfer and dry/wet atmospheric deposition was also evident (Choi et al. 2008). The surface water in Konya Basin, Türkiye was found to have varying concentrations of DDTs, where the primary metabolite observed was p, p-DDE (Aydin et al. 2013). DDT and its metabolites have been investigated in various soil types, household dust, and predominantly in fatty foods worldwide (Sharma et al. 2014).

In a study conducted in China, average concentration of p, p-DDE was 975 ng/g lipid, which was the dominant DDT metabolite. Total concentration of DDTs was 1105 ng/g lipid (Hu et al. 2021). There are few studies examining accumulation of DDT residues in breast milk in various locations in Türkiye. Despite collecting the samples from different years and locations, DDT levels were found to be similar in those studies. Concentration of p, p-DDE was substantially higher in comparison to other DDTs (Agus et al. 2022; Çok et al. 2012; Erdoğrul et al. 2004; Sharma et al. 2014). In the present study, all DDT metabolites were detected in human breast milk samples, and p, p-DDT had the highest concentration with a mean concentration of 139.8 ng/g lipid. In contrast, in the other studies, p,p-DDT levels were 10.54 ± 12.32, 58.50 ± 5.13, and 3.33 ± 2.05 ng/g lipid in Mersin, Adıyaman and Istanbul, respectively, at different periods.

Mothers residing in Istanbul for less than five years exhibited elevated levels of o, p′-DDE, p,p′-DDE, and PCB28, whereas those with prolonged residency demonstrated increased levels of propiconazole. The elevated concentrations of DDE and PCB28 may result from their lipophilic nature, allowing them to persist in the environment and accumulate in adipose tissue, indicating prior exposure, particularly among individuals from rural or agricultural regions where pesticides were historically utilised. Although DDT has been banned in Türkiye for decades, its persistence allows it to remain in the environment and accumulate in human tissues over time. In contrast, propiconazole is a non-persistent fungicide with lower bioaccumulation potential, and its presence among long-term residents may be due to more recent or ongoing exposure sources (Arena et al. 2017). All these results suggest that current residence may not fully explain the effects of cumulative exposure over time to both persistent and non-persistent pollutants.

In addition, bioaccumulation of DDTs significantly varied with age in our analysis. Multiple studies have demonstrated a consistent pattern of higher levels of POPs in human tissues as individuals age (Achour et al. 2023). There are several explanations regarding the association between POPs exposure and age. First, as individuals age, more prolonged exposure to these pollutants leads to higher accumulation in fatty tissues resulting in higher concentrations in their bodies. Secondly, aging affects the metabolism rate of individuals which may decrease the elimination of chemicals from the body (Lee et al. 2024).

It has been reported that higher concentrations of PCBs are linked to increased BMI across all age groups. Exposure to DDTs during pregnancy was found to be correlated with BMI during early childhood (Murro et al. 2022). In a recent study, BMI was an effect modifier in the association between dietary intake and PCB levels in serum because of the high accumulation of PCB compounds in adipose tissue (Lan et al. 2021). However, no significant association was determined between BMI and levels of organic pollutants examined in the present study.

Several studies have examined the association between POPs exposure and foetal growth, specifically birth weight. In our study, birth weight was negatively associated with HCB and propiconazole levels. In studies exploring the association between prenatal exposure to HCB and infant birth weight, HCB was found to be associated with decreased birth weight. Possible mechanisms include endocrine disruption, impaired placental function, triggered oxidative stress and inflammatory responses that could negatively impact foetal growth(Eggesbø et al. 2009). According to European Food Safety Authority's report, there is not enough epidemiological data about the effects of propiconazole on foetal growth, however, animal studies have shown that exposure during pregnancy led to early deliveries in rabbits, decreased litter size, fewer viable pups, and ossification abnormalities (Arena et al. 2017). These reproductive toxicity concerns suggest potential adverse effects on birth outcomes, including birth weight. In the report, it was mentioned that to better understand the relationship between propiconazole exposure, its reproductive toxicity, and possible mechanisms further studies are necessary (Arena et al. 2017).

There were mixed findings regarding the effects of the gravidity and number of nursed children on the accumulation of PCBs and DDTs in breast milk in our study. Mothers with fewer pregnancies exhibited lower levels of PCB28, while displaying higher levels of PCB153. Additionally, the number of nursed children showed negative correlations with PCB101 and p, p-DDD. Research shows that gravidity and the number of children breastfed may affect the levels of POP concentrations in breast milk in complex and inconsistent ways. Higher parity and longer breastfeeding duration are generally associated with lower concentrations of lipophilic POPs, such as PCBs and DDTs, as a result of toxin elimination through lactation. For example, Spanish mothers who had more children and breastfed for longer had less PCB and DDE levels in their breast milk (Ribas-Fitó et al. 2003), while a Swedish cohort study indicated that higher levels of PCB-153 and p, p'-DDE in blood were linked to having fewer prior births (Glynn et al. 2003). However, after adjustment for confounding factors such as mother's age, BMI, and cholesterol, some studies reported weaker or no significant associations (Cupul-Uicab et al. 2008). These inconsistencies may be explained by differences in lifetime exposure history, length of breastfeeding, individual differences in metabolism, and detoxification mechanisms (Santos et al. 2022).

Comet assay is used as a genotoxicity screening method to evaluate DNA damage in mammary cells exfoliated in breast milk (Arcaro and Anderton 2008; Daglioglu et al. 2011). DNA damage in leukocytes collected from placental samples has been associated with high concentrations of PCBs, and OCPs (Želježić et al. 2018). A recent study conducted in a population residing near recycling sites has shown that high levels of PCBs were measured among e-waste recycling workers, and a correlation between exposure to PCB 153 and the occurrence of DNA damage was demonstrated (Li et al. 2023). Our research showed that DNA damage markers were positively related to PCB 202, o, p-DDE, and propiconazole. PCB 202, a highly chlorinated non-dioxin PCB, is known for its strong lipophilic nature, its stability in the environment, and its long-lasting presence in the body, similar to other highly chlorinated PCBs. PCB 202 has the capacity to activate the ryanodine receptor (RyR). As a result it disrupts intracellular calcium equilibrium, and can potentially initiate the production of

reactive oxygen species (ROS) and mitochondrial dysfunction, which can lead to DNA damage (Holland et al. 2016). The observed associations between o, p′-DDE and propiconazole may also be attributed to similar oxidative and endocrine-disrupting mechanisms (Calaf et al. 2020).

In contrast, since γ-HCH (lindane) is a well-known toxicant classified as a Group 1 carcinogen, an unexpected inverse relationship was observed between the comet assay parameters and the level of γ-HCH. Several potential explanations could clarify this result. Firstly, this inverse correlation might be due to the limited sample size and the variability in pollutant levels. Secondly, given the complexity of mixture effects in environmental exposure, the observed association could have been affected by confounding variables, including concurrent exposure to other pollutants.

In conclusion, the present study demonstrates the presence of PCBs, OCPs, and selected non-POP pesticides in the breast milk of mothers residing in Istanbul. Our findings suggest that dietary factors such as red meat and chicken consumption contributed to the accumulation in the breast milk samples. Since POPs bioaccumulate in adipose tissue due to their lipophilic nature, the time interval between early exposure and development of the disease might be extended, and their hazardous effects on human health might emerge in later ages, specifically when the exposure occurs in sensitive periods such as perinatal life.

A significant implication of our research relates to the "breastfeeding paradox," in which breast milk, despite being the most suitable nutritional source for infants, could potentially facilitate exposure to environmental pollutants. While our findings indicate the existence of potentially harmful compounds, it is essential to emphasize that the established benefits of breastfeeding—providing immune protection, ideal nutrition, and a reduced risk of chronic diseases—generally outweigh the potential risks associated with chemical exposure (Van den Berg et al. 2017b). Nevertheless, these observations highlight the need for risk-reduction strategies. Practical measures could include limiting the intake of high-fat animal products, minimizing exposure to tobacco smoke, and advocating for public health initiatives designed to mitigate environmental pollution. Moreover, increasing understanding among mothers and healthcare workers about preventable exposure routes may help mitigate pollutant exposure while maintaining the benefits of nursing. These findings underscore the importance of including environmental health considerations in strategies for maternal and child health.

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Data Availability The datasets generated and/or analysed during the current study are available from the corresponding author on reasonable request.

Declarations

Conflict of interest The authors declare no conflicts of interest.

Ethical Approval and Consent to Participate The study protocol was approved by the Clinical Ethics Board of Yeditepe University in Istanbul, Türkiye. All procedures performed in studies involving human participants were in accordance with the 1964 Helsinki Declaration and its later amendments or comparable ethical standards (https://www.wma.net/policies-post/wma-declaration-of-helsinki/). Oral and written informed consent were obtained from all participants prior to data collection.

Consent for Publication Not applicable. No identifying images or other personal or clinical details of participants that compromise anonymity are included in this manuscript.

Trial Registration Not applicable.

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