Food Chemistry 135 (2012) 1873–1893

Contents lists available at SciVerse ScienceDirect

Food Chemistry

journal homepage: www.elsevier.com/locate/foodchem

Human health risk of dietary intake of organochlorine pesticide residues in bovine meat and tissues from Veracruz, México

Violeta Pardío *, David Martínez, Argel Flores, Dora Romero, Víctor Suárez, Karla López, Roxana Uscanga

Faculty of Veterinary Medicine, Universidad Veracruzana, Veracruz, Mexico

ARTICLE INFO

Article history:
Received 1 April 2012
Received in revised form 10 June 2012
Accepted 24 June 2012
Available online 29 June 2012

Keywords:
Organochlorine pesticides
Bovine meat
Dietary intake
Health risk

ABSTRACT

Tissue distribution patterns of organochlorine pesticides in bovine carcasses varied significantly among seasons, geographic locations and tissues. The highest concentrations of Σ−DDT during the dry season were detected in lungs from Paso de Ovejas (2,834.90 μg/kg lipid) and, during the rainy season, Lindane and Σ−HCH in muscle and lung samples from Paso de Ovejas (995.80 and 1,690.10 μg/kg lipid). Estimated daily intakes of γ−HCH and Σ−DDT (3.35 and 1.22 μg/kg bw/day) through consumption of muscle tissues from Paso de Ovejas and Puente Nacional during the rainy season showed the highest contribution. During the rainy season the highest non-cancer Hazard Ratios estimated corresponded to γ−HCH (3.97) and Σ−DDT (4.39) detected in muscle samples from Puente Nacional. The highest Hazard Ratios of cancer risk to the 95th centile daily consumption through meat corresponded to p,p'-DDT from Alvarado (7.76E + 06) and from Paso de Ovejas for γ−HCH (1.50E + 05) during rainy season. The results indicate potential non- and carcinogenic risks to consumer health through meat consumption.

1. Introduction

The use of persistent organochlorine pesticides (OCPs) in tropical countries has particular implications with regard to environmental and human exposure and food safety because high temperatures in these regions allow the OCPs to volatilize and distribute uniformly in all parts of the environment (Manirakiza et al., 2002). In Mexico, DDT was the principal insecticide used in malaria vector control programmes (Casas, Torres, Bown, Rodríguez, & Arredondo, 1998; INE, 2002), routinely sprayed in the malaria-endemic regions of Veracruz following WHO guidelines until 2003 (SSA, 2006). Hexachlorocyclohexane (HCH) is a synthetic pesticide with one isomer γ-HCH, commonly referred to as Lindane, still used in México in veterinary pest control. These contaminants are deposited through either dry gaseous, dry particle-bound, or wet deposition to soil and plants, the first trophic level in the food terrestrial chain (Bolt & Degen, 2002), and due to their lipophilic properties they bioaccumulate and biomagnify through the food chain (Kalantzi et al., 2001; Vieira, Torres, & Malm, 2001). Animal exposure may rise from direct treatment with pesticides, inhalation of contaminated air, or through ingestion of contaminated forages, herbage and feedstuffs (Kalantzi et al., 2001; Semeena, Feichter, & Lammel, 2005; Willett, O'Donnell, Durst, & Kurz, 1993). Because of their high lipid solubility, OCPs are deposited in adipose tissue and are excreted in milk (Pardío, Waliszewski, Landín, & Bautista, 2003). In bovine organisms after resorption, OCPs enter the liver and are metabolised slowly before they are released into the circulatory system and either finally deposited in the fat or excreted and passed onto the calf or to the consumer in milk (Jandacek & Tso, 2001). According to MacLachlan (1996), a beef animal develops 100–150 kg of fat that accumulates the contaminant over its 36 months lifespan as meat animals have no major fat excretion pathway and may never attain steady state levels in its 1–3 years lifespan (Rosenbaum, Mckone, & Jolliet, 2009).

Because of their high thermodynamic stability and lipid solubility, OCPs bind to lipid components in animal tissues, becoming a major route of human exposure when consumed as food, contributing to more than 90% of the daily exposure to these compounds. Previous studies have documented their bioaccumulation in the human body, especially in adipose tissue and breast milk (Covaci, de Boer, Ryan, Voorspoels, & Schepens, 2002; Cruz, Lino, & Silveira, 2003; Pardío et al., 1998; Snedeker, 2001; Waliszewski, Pardío, Chantiri, Infanzón & Rivera, 1996a; Zumbado et al., 2005). This tendency to accumulate in body tissues, its long persistence and the acute health risks of OCPs, have raised concerns about possible human health impacts due to low, but chronic, exposure from dietary intake. Organochlorine pesticides have been considered as 'endocrine—disrupting chemicals' and carcinogenic substances (Amaral-Mendes, 2002; Kavlock, 1996; Lemaire, Terouanne, Mauvais, Michel, & Rahmani, 2004; Witorsch, 2002). DDE inhibits androgen binding to the androgen receptor, whereas DDT has potent estrogenic activity in

* Corresponding author. Address: Laboratory of Toxicology, Faculty of Veterinary Medicine, Universidad Veracruzana, Avenida Miguel Ángel de Quevedo s/n, Colonia Unidad Veracruzana, Veracruz 91710, Mexico. Tel.: +52 229 9342075x24125; fax: +52 229 9342075x24104.
E-mail addresses: vpardio@uv.mx, vpardio@yahoo.com.mx (V. Pardío).

0308-8146/$ - see front matter © 2012 Elsevier Ltd. All rights reserved.
http://dx.doi.org/10.1016/j.foodchem.2012.06.079

mammals (Kelce, Gray, & Wilson, 1998; Kelce et al., 1995; Sonnenschein & Soto, 1998). Recent epidemiological studies indicate that some of these compounds may influence the concentrations of thyroid hormones (Meeker, Altshul, & Hauser, 2007), and the possible association between exposure to DDT and various types of cancers in humans, including leukaemia, prostate, brain, and lymphopoietic cancers, non-Hodgkins lymphoma and multiple myeloma, have been studied extensively (Beard, 2006; Dreiher & Kordysh, 2006; IARC, 2008; Quintana et al., 2004). Cox, Niskar, Narayan, and Marcus (2007) found among Mexican Americans residing in the southerwestern United States with elevated serum glucose levels that self-reported diabetes was significantly associated with serum levels of β−HCH, p,p′-DDT, and p,p′-DDE, suggesting that higher serum levels of certain organochlorine pesticides may be associated with increased prevalence of diabetes. The impacts of prenatal exposure to DDT on children's neurodevelopment have also been investigated; Eskenazi et al. (2006) found an association with delays in neurodevelopment during early childhood.

These compounds also have the potential to compromise the health and productivity of domestic animals. Monitoring OCPs in animal tissues collected from different locations is important because it provides useful information concerning the extent of pollution trends in relation to local origin, it helps to elucidate the spatial variation in contamination patterns, and to assess the health risks associated with the consumption of contaminated food from animals (Jevsnik, Cerkvenik, & Doganoc, 2004). It is estimated that meat and meat products contribute to 15–20% of OCPs when compared the other foodstuffs. Meat and meat products are important foods and there are various reports on the residual levels of OCPs in these products over the world (Barkatina et al., 1999; Glynn et al., 2000; Hashemy-Tonkabony et al., 1981; Manirakiza et al., 2002; Osibanjo & Adeyeye, 1997; Schecter, Cramer, Boggess, Stanley, & Olson, 2001).

Meat consumption in México has increased considerably over the last few years as a consequence of diet diversification resulting from rising incomes and new styles of food consumption, such as 'fast food'. The *per-capita* consumption of beef meat in Mexico has been reported as 58.3 g/person/day (SAGARPA, 2008). Mexican annual production of beef meat in 2008 was 1,656.4 thousand tons, with Veracruz State the major producer. Initial data on OCPs residues in beef meat from Veracruz, México have been proceeding since 1996 and indicated relatively higher mean levels than those reported in other countries (Waliszewski, Pardío, & Waliszewski, 1996b). In spite to the relevance of the food chain accumulation and transfer to humans, and the anthropogenic activities leading to a release of these pesticides, no appraisals on episodes dealing with livestock production systems threatened by OCPS were found in the literature. Moreover, no study has been performed to evaluate the temporal trends of OCPs residues in bovines slaughtered for meat production and the possible differences in concentrations depending on the location of the livestock producer. Also, no attempt has been made to estimate the potential human health risks from exposure to OCPs posed by meat consumption. The aim of this study was to assess the spatial and seasonal changes of organochlorine pesticide residues in bovine meat, organs and tissues from the central tropical region of Veracruz, Mexico, and to determine the possible health hazard for populations consuming meat contaminated with these pesticide residues.

2. Materials and methods

2.1. Sampling

A total of 168 samples consisting of three samples of each tissue - muscle, liver, heart, lung, kidney, spinal marrow and adipose tissue, were collected at random from 24 carcasses of healthy male steers that were 12–16 months old; each group of 3 animals slaughtered at the slaughter house located in Vargas, Veracruz, México, were sampled during two sampling periods. The cattle were fed a balanced diet and tap water *ad libitum* as part of routine management practices used at each production center. Livestock originated from four extensive breeding stock on commercial bovine producing farms located in the south-west region from the central agrarian zone of Veracruz, Mexico showed in Fig. 1: Puente Nacional (PN) (19°20′ N, 95°08′ W and 100 m over sea level), Alvarado (ALV) (18°46′ N, 95°46′ W and 10 m over sea level), Paso de Ovejas (PO) (19°17′ N, 95°46′ W and 40 m over sea level), and Manlio Fabio Altamirano (MFA) (19°06′ N, 96°20′ W and 40 m over sea level). The climate in this region is characterised by a dry season (from November to April) and a rainy season (from May to October) (INEGI, 2008).

Three samples (500 g each) of each tissue -muscle, subcutaneous adipose tissue, spinal marrow, liver, heart, lung and kidney, were individually wrapped in pre-treated aluminium foil immediately after collection and packed separately in ziplock plastic bags and transported fresh in coolers to the Toxicology laboratory of the Veterinary Faculty. Muscle and tissue samples were analysed within 24 h or kept at −25 °C until extraction was carried out. Prior to the extraction, all glassware were properly washed with detergent (free of chlorinated hydrocarbons) and rinsed with distilled water. The water was removed with acetone and the acetone with petroleum ether and glassware then heated in an oven at 400° C for 4 h to avoid any contamination of pesticides (UNEP, 1988). All chemicals were analytical grade and obtained from Merck (Darmstadt, Germany), J.T. Baker and Sigma–Aldrich Company (St. Louis, MO, USA) and used without further purification.

2.2. Determination of organochlorine pesticide residual concentrations

Extraction was performed according to the method suggested by Murphy (1972) with modifications. Briefly, samples (250 g each) were minced and ground with thorough mixing after each grinding. Then, 1 g of adipose tissue and 10 g of muscle or other tissue (spinal marrow, liver, heart, lung and kidney) sample were taken for residue analysis. Each sample was ground with a sufficient amount of anhydrous sulfate and extracted with petroleum ether (120 mL) in a 50 × 1 cm i.d. chromatographic column. The extract was centrifugated for 15 min at 3000 rpm. The organic phase was obtained and the extraction procedure was repeated twice more. The three organic extracts were combined and then concentrated at low pressure in a vacuum concentrator to a volume of 1 mL. To this residue, 0.5 mL of concentrated sulfuric acid was added and the mixture centrifugated for 10 min at 3000 rpm. The acid residue was extracted twice more with the addition of 1 mL hexane. The three organic phases were collected and evaporated to dryness. The dry residue was re-dissolved in 1 mL of hexane. Clean-up was done using a glass column packed with 10 g of anhydrous sulfate and then extracted with hexane (15 mL) and 1% methanol in hexane (10 mL). After clean-up the final extracts were evaporated to dryness using a rotatory evaporator. The dried extract fortified with the internal standard, p,p′dichlorobenzophenone (Supelco Park Bellefonte, PA, USA), prepared in hexane (10 μg/mL), was dissolved in 2 mL *n*-hexane for gas chomatographic analysis. The concentration values were reported as μg/kg on a fat basis and non-detect data was assumed to be half the method limit of detection.

Tissue fat content was determined according to AOAC Official Method 991.36 (1998) modified. Briefly, 5 g of muscle or tissue sample were placed in a filter paper #42 (11 cm) and extracted with acetone (60 mL) for 6 h in a Soxhlet apparatus. The fat content was used to calculate dietary burden estimates and to express organochlorine pesticide concentrations on a fat basis.

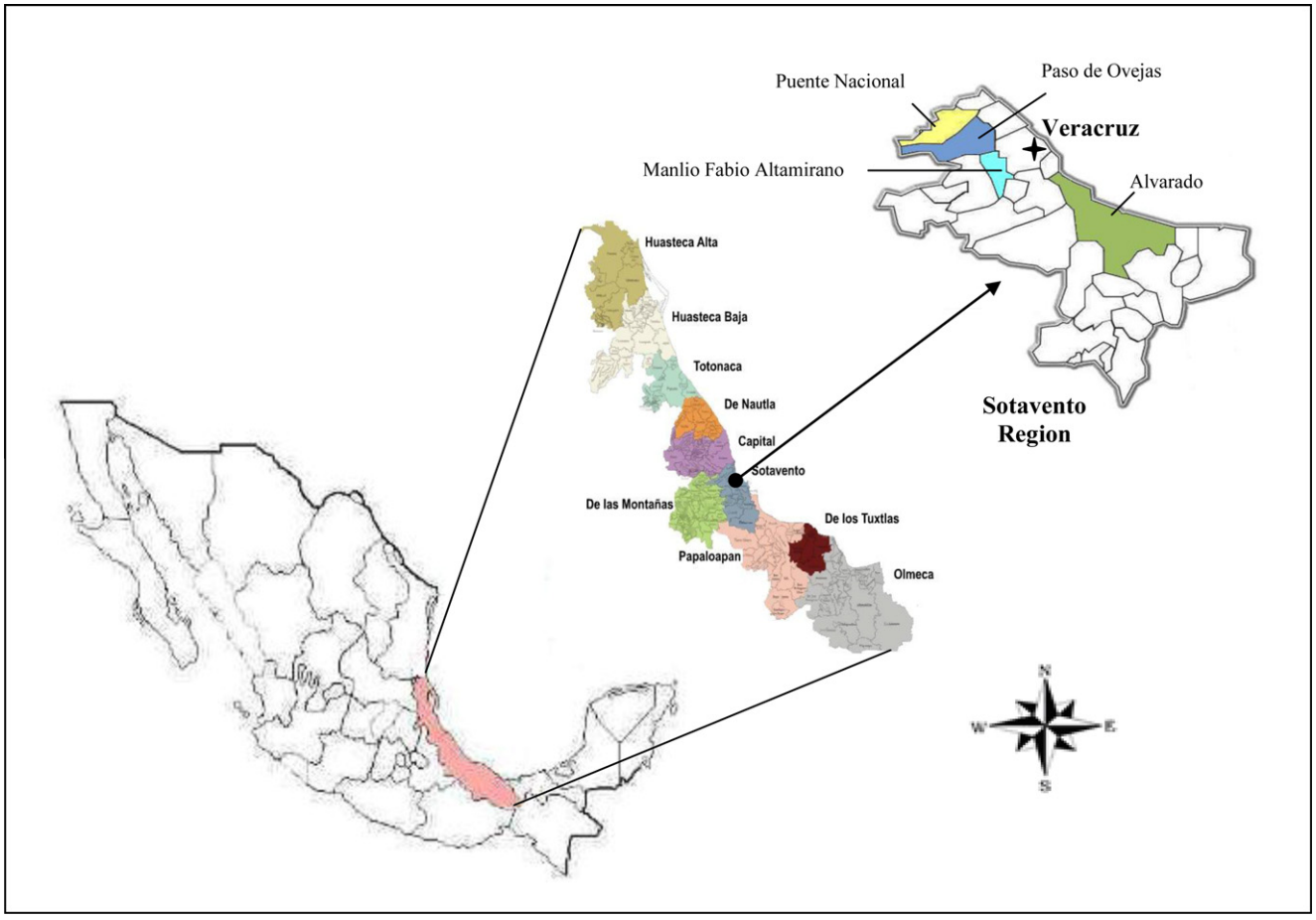


Fig. 1. Commercial bovine producers' locations in the central agrarian region of Veracruz, México.

2.3. Chromatographic analysis

A one-microliter aliquot was injected into GC-μECD model Hewlett Packard 6890-Plus, equipped with a ^{63}Ni μ−electron capture detector and HP3398A GC ChemStation software. GC operating parameters were as follows: a split/split-less programmed temperature injector operated in the split-less mode at a temperature 250 °C and a detector temperature 330 °C. For pesticide separation, a fused silica column HP 608 30 m × 0.53 mm ID, 0.5 mm film was used. The temperature column was programmed from 85 to 195 °C at 30 °C min^{-1}, 195 to 250 °C at 5 °C min^{-1}, and then held for 5 min. The carrier gas was ultrapure grade nitrogen at a constant flow rate of 2.4 mL min^{-1}.

2.4. Quality control and quality assurance

Detector linearity was determined by linear regression analysis of five-point calibration curves (response versus concentration) for each analyte. Linear regression equations were used to quantify analytes in samples. Calibration of the instrument was performed before the sample analysis using standards of pesticides obtained from ChemService (Chem Service, Inc., West Chester, PA, USA) and Supelco (Supelco Park, Bellefonte, PA, USA). Qualitative and quantitative analyses were performed by comparing the retention time and peak area of the sample, respectively, with those of the calibrated reference standards. A working standard mixture of OC pesticides was prepared by combining aliquots of each stock standard solution of 100 μg/mL and diluting up to a concentration of 1.0 μg/mL with hexane. All standard solutions were stored at 4° C in amber glass vials with Teflon-lined screw caps. The method of linear regression was applied for quantification using 5 concentration levels in linear area with 10, 25, 50, 75 and 100 μL of the standard mixture. Two sets of 5 samples each of muscle with low levels of OC pesticide contamination were spiked with 10, 25, 50, 75 and 100 μL of working standard mixture of OC pesticide. Each fortified sample was shaken vigorously for 2 h on an automatic shaker and was left to stand overnight at 4° C. Each sample was equilibrated to room temperature before the extraction procedure. These samples were extracted and analysed as previously described. Linear regression was applied to these data and method limits of detection (MLOD) and quantification (MLOQ) were calculated from the curve obtained from the recovery studies, according to Su (1998). Table 1 shows the calibration curve obtained for each pesticide, average recovery (%) and RSD (%) for each pesticide, and method limits of detection and quantification of pesticides studied in fortified muscle samples, reported as mg/kg on a muscle fat basis. Acceptable precision was considered to be an RSD of ⩽10%.

2.5. Health risk assessment

Two guidelines were used to assess the risk of OCPs in meat and tissues in the present study. Firstly, the dietary pesticide exposure was assessed estimating the acceptable daily intakes of the determined levels of OCPs in these samples following the recommended Guidelines for Predicting Dietary Intake of Pesticide Residues of the Food and Agricultural Organization/World Health Organization (FAO/WHO, 1997). The Estimated Daily Intake (EDI) of organochlorine pesticides was calculated from the following relationship:

Table 1
Correlation coefficient (r) of standard calibration curves, average recovery (%) and RSD (%), and limits of detection and quantitation for organochlorine pesticides studied in meat fat samples.

Pesticide	(r)	Average recovery (%)	Precision RSD (%)[a]	LOD (μg/kg)[b]	LOQ (μg/kg)[c]
α-HCH	0.9996	94	0.44	0.00002	0.0001
β-HCH	0.9983	97	0.62	0.00024	0.0008
γ-HCH	0.9997	98	1.70	0.00031	0.0010
δ-HCH	0.9994	99	0.01	0.00009	0.0003
p,p′-DDE	0.9991	98	1.66	0.00036	0.0012
p,p′-DDT	0.9769	99	1.04	0.0004	0.0014
p,p′-DDD	0.9966	96	1.06	0.0003	0.0010
o,p′-DDT	0.9927	91	0.88	0.0003	0.0008
HCB	0.9975	92	0.35	0.00024	0.0008
Aldrin	0.9994	95	0.38	0.010	0.005
Dieldrin	0.9992	89	0.19	0.003	0.010
Endrin	0.9936	96	0.33	0.0051	0.017
Endrin Aldehide	0.9512	91	0.59	0.003	0.004
Endosulfan I	0.9983	92	0.74	0.00124	0.0041
Endosulfan II	0.9891	94	0.96	0.00417	0.0139
Endosulfan sulphate	0.9934	93	1.46	0.00156	0.005
Heptachlor	0.9994	96	0.47	0.00013	0.0004
Heptachlor epoxide	0.9992	99	1.39	0.00125	0.0042

[a] RSD = relative standard deviation.
[b] LOD = detection limit expressed as μg/kg fat basis.
[c] LOQ = quantitation limit expressed as μg/kg fat basis.

$$EDI = C_a \times F \times F_i$$

where Ca is the average residue level of an organochlorine pesticide in the edible portion of samples in μg/kg lipid base, F is the fat content (%) in the muscle and tissue samples, and Fi is the amount of food intake as meat consumed per person in g/kg bw (body weight) per day. The EDIs were reported in μg of pesticide/kg bw/day. The exposures were estimated on a per-capita basis of the consumption of meat in México, 58.3 g/person/day during 2008 (SAGARPA, 2008), the amount of regional per-capita daily consumption of cattle kidney and liver (0.2 and 0.3 g/day) according to the Global Environment Monitoring System/Food Contamination Monitoring and Assessment Programme (GEMS/Food Regional Diets) (WHO, 2003) and the average weight per person in Mexico, estimated at 60 kg, both reported by the National Institute of Public Health (INSP, 2007). The dietary intake estimates of pesticide residues (EDIs) were compared with the recommended guidelines of acceptable daily intake (ADI) formulated by FAO/WHO (2009). In predicting chronic exposure, average daily food consumption values are used in predicting exposure to pesticide residues and contaminants for assessing long-term risks to permit valid comparison with the ADI, which is based on intake over a lifetime (FAO/WHO, 2008a). Secondly, risks associated with cancer and non-cancer health effects due to exposure were considered separately. Potential risk of estimated exposures, based on the dietary intake of OCs from meat consumption, was evaluated by taking both chronic effects and carcinogenic effects into consideration, proposed by the US EPA (2009) and as described in other references (Dougherty et al., 2000; Jiang et al., 2005). The exposures of undetected congeners were calculated as one half of the respective LOD.

For non-cancer hazards, Hazard Ratios (HRs) were estimated by dividing the average daily exposure by the benchmark (BMC) for non-carcinogenic effects based on the US EPA Reference Dose (RfD) from US EPA Integrated Risk Information System (IRIS) for each contaminant, the benchmark concentration for noncarcinogenic effect:

$$Hazard\ ratio\ (HR) = \frac{Average\ daily\ exposure}{Benchamark\ concentration} \quad (1)$$

For each contaminant, the average daily exposure level for a population was calculated by:

$$Average\ daily\ exposure\ (\mu/kg\ b.w.hh) = Meat\ consumption\ (g/kg\ b.w.)\ X\ Contaminant\ concentration\ (\mu/g) \quad (2)$$

where b.w. = body weight

A benchmark concentration represents a daily concentration below which there is a high probability of no adverse health effect. A hazard ratio greater than unity indicates that the average exposure level exceeds the benchmark concentration and that there is a potential risk to human health (Dougherty et al., 2000).

$$Benchmark\ concentration = \frac{Risk \times Body\ weight}{Meat\ consumption \times Cancer\ Slope\ factor} \quad (3)$$

For lifetime cancer risk assessment, the average daily exposure was divided by the benchmark concentration for carcinogenic effect derived, where risk is the probability of lifetime cancer risk and the cancer slope factor is taken from US EPA Integrated Risk Information System (IRIS) for each contaminant so as to estimate chemical-specific risk for a reasonable maximum exposure, using the cancer risk of one in one million for a lifetime exposure.

The benchmark concentration for carcinogenic effects represents the exposure concentration at which lifetime cancer risk is one in one million for lifetime exposure. To assess the potential cancer risk, the HR was estimated at the 50th percentile exposure concentrations, and the other was based on the 95th percentile of the upper confidence of the mean exposure concentration (Jiang et al., 2005; US EPA, 2009). The average daily doses were calculated by assuming 100% absorption of organochlorine pesticides, as reference doses and cancer potencies generally do not account for bioavailability.

2.6. Statistical analysis

Data were subjected to MANOVA and ANOVA analysis to determine the differences in the organochlorine contents among the different locations, dry and rainy seasons and tissues analysed. Significant differences among means were analysed by Tukey's Test using the statistical software XLSTAT v.2011.2.05.

3. Results and discussion

3.1. Contamination patterns of organochlorine pesticide residues in bovine tissues from different areas and seasons

The organochlorine pesticides analysed HCH and DDT and its metabolites. They were detected at an incidence of contamination of 100%. In most cases, their residues were detected in 66–90% of all samples analysed. The fat content of tissue samples ranged between 2.4–3.4% (lung, kidney, and heart), 3.2–5.8% (liver and muscle), and 26.9–94.4% (spinal marrow and adipose tissue), similar to those reported by Covacci, Gheorghe and Schepens (2004).

3.1.1. Hexaclorociclohexane (HCH)

Concentrations of HCH isomers during dry and rainy seasons from the different locations are presented in Tables 2 and 3.

The detection frequency of the isomer α–HCH in bovine tissue samples during the dry season were 66.7% (PN), 81.0% (ALV), 83.3% (PO), and 81.0% (MFA), representing twice the frequency detected during the rainy season. α–HCH mean residue levels were detected in 100% of muscle and liver samples, 91.7% in heart and kidney samples, 83.3, 44.4, and 25.0% in lung, spinal marrow and adipose tissue, respectively, all during the dry season. Meanwhile, during the rainy season, this isomer was detected in 83.3, 66.7, and 58.3% of heart, adipose tissue and lung tissue samples, respectively, and 16.7, 11.2, and 11.1% in liver, kidney and spinal marrow tissue samples, but it was not detected in muscle samples (data not shown). As shown in Table 2 the mean residual concentrations of α–HCH in lung tissue samples from PN of 164.87 (dry season) and 145.45 μg/kg lipid (rainy season), were significantly higher ($P < 0.05$) compared to the levels in other tissues samples, and α–HCH mean levels in lung tissue samples from PO (307.08 μg/kg lipid) were higher ($P > 0.05$) during rainy season. Muscle tissue samples presented the lower ($P > 0.05$) α–HCH mean residue levels from PN, ALV, PO, and MFA (23.72, 13.11, 34.29, and 14.64 μg/kg lipid, respectively) during the dry season when compared to other tissues, and it was not detected (0.05 μg/kg lipid) during the rainy season. Adipose tissue concentrations from PN and ALV were also lower ($P > 0.05$) during the dry season (26.28 and 29.10 μg/kg lipid) and α–HCH was not detected (0.05 μg/kg lipid) in bovines from PO and MFA.

During the dry season the distribution pattern of this metabolite was in the order of lung > liver > kidney > heart > spinal marrow > adipose tissue > muscle, and during the rainy season the distribution changed to liver > lung > adipose tissue > heart > kidney > spinal marrow > muscle. The formation of α–HCH in mixed anaerobic bacterial cultures obtained from rumen has been mentioned by Haider and Jagnow (1975) who reported that dechlorination and volatile metabolites also occurred with bovine rumen fluid or in anaerobic culture solutions inoculated with rumen fluid. The lower accumulation of α–HCH in the spinal marrow during dry (42.01 μg/kg lipid) or rainy season (8.70 μg/kg lipid) could be due to its preferential accumulation in the white matter as this tissue contains white and gray matter, and to a different lipid composition as the main lipidic constituents of spinal marrow are cholesterol and phospholipid, while triglycerides constitute more than 90% of the total lipid content in fat (Jacobson, 1991).

The percentages of positive samples of β–HCH in bovine tissue samples during the rainy season (85.7% PN, 71.4% ALV, 94.4% PO, and 90.5% MFA) were several times higher than the percentages of isolation during the dry season (19.0% PN, 14.3% ALV, 100.0% PO, and 9.5% MFA). The distribution pattern for this metabolite was 41.7% of liver samples, 25.0 and 17% in kidney, lung, and adipose tissue samples during the dry season. As can be seen in Table 2, during the dry season the highest ($P < 0.05$) β–HCH mean level was detected in lung tissues from PN (59.10 μg/kg lipid), but it was not detected (0.0001 μg/kg lipid) in muscle, heart and spinal marrow samples from all locations, and in lung tissue samples from PO and MFA, kidney samples from MFA and adipose tissue samples from MFA and PN. However, this metabolite was detected

Table 2
Residues of HCH and metabolites (μg/kg lipid basis) in bovine tissues from different areas during dry season.

Pesticide	% of samples positive	Tissues						
		Muscle	Liver	Heart	Lung	Kidney	Spinal marrow	Adipose tissue
Fat content (%)		5.85	5.27	3.44	3.32	3.32	36.85	94.40
Puente Nacional								
α–HCH	66.7	23.72 ± 5.57^{a}	62.01 ± 37.97^{a,b}	43.00 ± 15.66^{a}	164.87 ± 102.57^{b}	93.29 ± 36.42^{a,b}	0.00001^{c}	26.28 ± 0.00^{a}
β–HCH	19.0	0.0001^{a}	6.98 ± 0.10^{b}	0.0001^{a}	59.10 ± 0.30^{c}	32.59 ± 0.10^{c}	0.0001^{a}	13.32 ± 0.20^{b}
δ–HCH	100.0	12.63 ± 5.65^{a}	34.75 ± 18.38^{a}	18.28 ± 17.67^{a}	55.18 ± 26.53^{a}	59.39 ± 20.15^{a}	149.57 ± 85.36^{a}	58.43 ± 66.77^{a}
γ–HCH	28.6	0.0002^{a}	0.0002^{a}	0.0002^{a}	81.34 ± 83.310^{b}	0.0002^{a}	97.40 ± 62.27^{b}	0.0002^{a}
Σ–HCH		36.30 ± 11.10^{a}	99.10 ± 42.00^{a}	61.30 ± 24.30^{a}	266.10 ± 257.20^{a}	132.40 ± 63.50^{a}	247.00 ± 147.20^{a}	71.60 ± 57.30^{a}
Alvarado								
α–HCH	81.0	13.11 ± 0.89^{a}	42.72 ± 23.35^{a}	52.00 ± 25.68^{a}	119.71 ± 31.17^{b}	38.83 ± 13.72^{a}	59.81 ± 0.00^{a}	29.10 ± 3.52^{a}
β–HCH	14.3	0.0001^{a}	14.62 ± 0.00^{b}	0.0001^{a}	31.04 ± 0.00^{b}	19.78 ± 0.00^{b}	0.0001^{a}	0.0001^{a}
δ–HCH	100.0	7.00 ± 0.97^{a}	29.89 ± 11.88^{a}	30.56 ± 32.67^{a}	45.57 ± 16.79^{a}	15.77 ± 8.51^{a}	27.92 ± 14.45^{a}	113.39 ± 154.91^{a}
γ–HCH	47.6	4.70 ± 2.00^{a}	0.0002^{a}	0.0005^{a}	52.89 ± 25.65^{b}	13.22 ± 0.55^{a}	23.39 ± 3.77^{a}	55.62 ± 62.66^{b}
Σ–HCH		21.70 ± 1.60^{a}	77.50 ± 23.10^{a}	82.60 ± 57.20^{a}	188.60 ± 78.60^{a}	65.60 ± 7.80^{a}	71.20 ± 30.40^{a}	169.90 ± 192.60^{a}
Paso de Ovejas								
α–HCH	83.3	34.29 ± 16.44^{a}	88.44 ± 61.66^{a}	63.74 ± 17.54^{a}	106.86 ± 28.90^{a}	53.94 ± 13.75^{a}	n.a.	0.00001^{b}
β–HCH	16.7	0.0001^{a}	34.80 ± 0.00^{b}	0.0001^{a}	0.00035^{a}	1.38 ± 0.00^{b}	n.a.	16.87 ± 0.00^{b}
δ–HCH	100.0	36.15 ± 35.85^{a}	38.18 ± 20.25^{a}	22.65 ± 8.57^{a}	88.87 ± 56.14^{a}	18.23 ± 4.97^{a}	n.a.	141.75 ± 100.89^{a}
γ–HCH	27.8	26.57 ± 0.50^{a}	0.0002^{b}	0.0005^{b}	34.81 ± 0.90^{a}	0.0002^{b}	n.a.	70.49 ± 51.39^{a}
Σ–HCH		79.30 ± 59.70^{a}	138.20 ± 101.70^{a}	86.40 ± 17.00^{a}	207.30 ± 43.00^{a}	72.60 ± 19.50^{a}	n.a.	217.90 ± 141.80^{a}
Manlio Fabio Altamirano								
α–HCH	81.0	14.64 ± 9.80^{a}	74.09 ± 61.10^{a}	39.73 ± 6.19^{a}	72.43 ± 15.32^{a}	40.48 ± 15.57^{a}	36.08 ± 17.48^{a}	0.00001^{b}
β–HCH	9.5	0.0001^{a}	34.19 ± 21.78^{b}	0.0001^{a}	0.0001^{a}	0.0001^{a}	0.0001^{a}	0.0001^{a}
δ–HCH	100.0	11.62 ± 4.04^{a}	49.73 ± 19.44^{a}	35.30 ± 20.10^{a}	59.32 ± 41.29^{a}	19.97 ± 11.12^{a}	37.88 ± 18.11^{a}	119.52 ± 134.71^{a}
γ–HCH	38.1	0.0002^{a}	0.0002^{a}	45.00 ± 2.00^{b}	22.87 ± 5.60^{b}	0.0002^{a}	31.33 ± 15.64^{b}	114.54 ± 154.83^{b}
Σ–HCH		26.30 ± 13.00^{a}	146.60 ± 67.80^{a}	76.80 ± 25.30^{a}	147.00 ± 44.30^{a}	60.40 ± 26.70^{a}	94.90 ± 56.30^{a}	234.10 ± 289.30^{a}

Means with different letters were statistically different at $P < 0.05$ among tissues.
Σ–HCH = α–HCH + β–HCH + γ–HCH; n.a. = not analysed.

Table 3
Residues of HCH and metabolites (μg/kg lipid basis) in bovine tissues from different areas during rainy season.

Pesticide	% of samples positive	Tissues						
		Muscle	Liver	Heart	Lung	Kidney	Spinal marrow	Adipose tissue
Fat content (%)		5.24	3.20	2.67	2.42	2.54	26.92	91.28
Puente Nacional								
α–HCH	42.9	0.00001[a]	0.00001[a]	28.28 ± 18.02[b]	145.45 ± 5.45[c]	26.09 ± 9.41[b]	0.00001[a]	59.17 ± 60.44[b]
β–HCH	85.7	174.90 ± 60.50[a]	75.30 ± 44.50[a]	136.40 ± 55.40[a]	294.90 ± 257.10[a]	60.80 ± 0.10[a]	41.20 ± 12.40[a]	129.60 ± 193.80[a]
δ–HCH	95.2	63.20 ± 24.30[a]	18.60 ± 5.10[a]	59.80 ± 30.10[a]	228.80 ± 248.20[a]	34.40 ± 10.90[a]	21.40 ± 8.40[a]	95.10 ± 132.50[a]
γ–HCH	76.2	76.00 ± 0.50[a]	139.40 ± 0.50[a]	160.30 ± 153.50[a]	141.50 ± 110.70[a]	39.60 ± 13.70[a]	13.00 ± 4.20[a]	54.50 ± 53.70[a]
Σ–HCH		263.40 ± 77.30[a]	140.40 ± 125.80[a]	355.40 ± 165.10[a]	663.90 ± 663.30[a]	111.70 ± 44.10[a]	75.70 ± 11.80[a]	320.20 ± 434.90[a]
Alvarado								
α–HCH	47.6	0.00001[a]	207.63 ± 0.30[c]	31.94 ± 12.56[b]	37.41 ± 10.16[b]	0.00001[a]	8.70 ± 0.65[b]	33.99 ± 9.28[b]
β–HCH	71.4	228.20 ± 0.40[a]	280.80 ± 213.30[a]	107.40 ± 61.70[a]	70.40 ± 39.40[a]	256.60 ± 60.40[a]	88.40 ± 106.50[a]	56.60 ± 59.50[a]
δ–HCH	100.0	62.90 ± 62.80[a]	168.90 ± 160.20[a]	28.00 ± 24.50[a]	51.00 ± 14.20[a]	127.10 ± 110.10[a]	71.70 ± 55.60[a]	46.40 ± 28.60[a]
γ–HCH	90.5	105.50 ± 61.90[a]	217.70 ± 0.20[a]	94.50 ± 29.50[a]	47.40 ± 29.40[a]	145.30 ± 68.30[a]	54.60 ± 42.50[a]	37.70 ± 28.60[a]
Σ–HCH		244.40 ± 131.60[a]	591.50 ± 606.90[a]	226.10 ± 120.70[a]	170.30 ± 59.30[a]	443.50 ± 331.70[a]	188.10 ± 181.70[a]	174.80 ± 124.10[a]
Paso de Ovejas								
α–HCH	44.4	0.00001[a]	158.28 ± 0.33[b]	39.31 ± 39.42[b]	307.08 ± 303.79[b]	0.00001[a]	n.a.	90.12 ± 91.39[b]
β–HCH	94.4	496.30 ± 502.30[a]	347.40 ± 245.60[a]	121.10 ± 144.10[a]	853.70 ± 1104.40[a]	107.30 ± 109.50[a]	n.a.	151.70 ± 235.20[a]
δ–HCH	100.0	273.80 ± 115.80[a]	152.20 ± 101.40[a]	83.20 ± 95.80[a]	499.40 ± 522.70[a]	89.30 ± 74.40[a]	n.a.	105.20 ± 147.60[a]
γ–HCH	75.8	995.80 ± 496.20[a]	0.0002[b]	75.90 ± 77.16[a]	416.90 ± 415.60[a]	85.00 ± 72.40[a]	n.a.	88.30 ± 128.50[a]
Σ–HCH		1434.00 ± 1230.10[a]	552.4 ± 377.20[b]	319.50 ± 356.40[b]	1690.10 ± 2115.60[a]	281.50 ± 253.20[b]	n.a.	405.20 ± 593.60[a]
Manlio Fabio Altamirano								
α–HCH	14.3	0.00001[a]	0.00001[a]	59.65 ± 50.75[b]	42.30 ± 0.65[b]	0.00001[a]	0.00001[a]	0.00001[a]
β–HCH	90.5	108.60 ± 72.80[a]	347.20 ± 441.30[a]	203.80 ± 246.70[a]	41.80 ± 14.40[a]	636.40 ± 852.15[a]	27.70 ± 0.40[a]	14.70 ± 4.40[a]
δ–HCH	100.0	36.60 ± 8.30[a]	168.40 ± 241.10[a]	53.10 ± 53.40[a]	27.70 ± 4.50[a]	295.60 ± 304.70[a]	26.70 ± 8.30[a]	15.30 ± 5.50[a]
γ–HCH	85.7	138.70 ± 0.250[a]	132.70 ± 15.60[a]	134.20 ± 134.80[a]	36.40 ± 8.60[a]	358.10 ± 395.80[a]	14.30 ± 9.20[a]	10.80 ± 4.00[a]
Σ–HCH		191.40 ± 60.10[a]	488.30 ± 535.70[a]	430.80 ± 483.50[a]	106.10 ± 23.10[a]	1290.10 ± 1552.00[a]	59.50 ± 23.30[a]	40.70 ± 13.80[a]

Means with different letters were statistically different at $P < 0.05$ among tissues.
Σ–HCH = α–HCH + β–HCH + γ–HCH; n.a. = not analysed.

in all tissues during the rainy season, mainly in adipose tissue (100.0%), liver and heart samples (92.0%), muscle samples (83.0%), and in lung, kidney and spinal marrow samples (75.0%). Table 3 shows that the highest mean levels ($P > 0.05$) during the rainy season were found in lung tissue samples from PO (853.70 μg/kg lipid) followed by kidney tissue samples from FMA (636.40 μg/kg lipid) and muscle and liver samples from PO (496.30 and 347.40 μg/kg lipid). Because β−HCH has a much lower vapour pressure and bio-concentration factor in fat as compared to the α−HCH, and since this isomer is known to be metabolised more slowly (Willett, Ulrich, & Hites, 1998) its high levels and frequency of detection in these tissues were expected. Thus, the accumulation of β–HCH in tissues might reflect enhanced affinity for lipids due to a higher log K_{ow}, as this isomer is known to be more lipophilic than the other isomers; furthermore, the α−isomers may isomerize to its β-isomer in living organisms (Jensen, 1983).

During the dry season the distribution pattern of this metabolite was in the order of lung > liver > kidney > adipose tissue > heart > muscle > spinal marrow, and, during the rainy season the distribution changed to lung > kidney > muscle > liver > heart > adipose tissue > spinal marrow, a distribution pattern similar to the distribution in rats reported by Srinivasan and Radhakrishnamurty (1983): fat > kidney > lung > liver > muscle > heart > spleen > brain > blood. Nevertheless in ALV and MFA, the isomer showed a different pattern of residue distribution to various tissues. Distribution of β−HCH mean levels in ALV tissue samples was liver > kidney > muscle > heart > spinal marrow > lung > adipose tissue and in MFA kidney > liver > heart > muscle > lung > spinal marrow > adipose tissue. This distribution pattern was similar to the contribution of β−HCH in pork tissues reported by Covaci et al. (2004): liver > heart > brain > kidney > muscle > spinal marrow. As can be seen, the tissue distribution of β−HCH was not very consistent between animals of the production units studied, suggesting that the distribution phase was incomplete and that β−HCH was still associated mainly with circulating blood lipids rather than as an equilibrium with depot fat. As explained earlier, beef animals have no major fat excretion pathway and may never attain steady state levels in their 1–3 years lifespan (MacLachlan, 1996; Rosenbaum et al., 2009). This variation in distribution patterns was probably influenced by the level of contamination and distribution of the metabolite in the diet, and the exposure to contaminated air, as the higher levels of β−HCH were detected during the rainy season. This is consistent with a study by Srinivasan and Radhakrishnamurty (1983) indicating that β−HCH is accumulated in a dose and time dependent manner, producing a more marked elevation of tissue residue levels in rats. The stability of β−HCH, its tendency to accumulate in human and animal tissues over time, the rapid bio-concentration in man (approximately 525), and its slower elimination than other HCH isomers (Sang, Petrovic, & Cuddeford, 1999) represent a significant risk with respect to its chronic toxicity. This metabolite is the most toxic followed, by α-, γ-, and δ-HCH, probably due to its longer biological half-life in the body of seven to eight years (ATSDR, 2005).

The isomer δ−HCH was detected in 100% of all tissue samples analysed during both dry and rainy seasons. According to Table 2, the highest mean concentration ($P > 0.05$) of this metabolite was detected during the rainy season in lung tissue samples (499.40 μg/kg lipid) and muscle samples (273.80 μg/kg lipid) from PO, in kidney samples (295.60 μg/kg lipid) from MFA, and in lung tissue samples (228.80 μg/kg lipid) from PN. In contrast, mean residue levels of δ−HCH during the dry season were detected mainly in spinal marrow samples from PN (149.57 μg/kg lipid), and adipose tissue samples from PO, MFA and ALV (141.75, 119.52, and 113.39 μg/kg lipid, respectively).

During the dry season the distribution pattern of this metabolite was in the order of adipose tissue > spinal marrow > lung > heart > liver > kidney > heart > muscle, and, during the rainy season, the distribution changed to lung > kidney > liver > muscle > adipose tissue > heart > spinal marrow. The high frequency of detection of δ−HCH (100%) during both seasons and in all production units could be due to the intake of soil, feed and grass contaminated with this isomer or to the isomerization of γ−HCH. The isomerization of γ−HCH to δ−HCH in aquatic sediments has been reported by Newland, Chester, and Lee (1969) and it is generally observed that γ−HCH is more easily biodegraded than the δ−HCH isomer, which was found to be more stable due to its equatorial chlorine atoms (Jagnow, Haider, & Ellwardt, 1977).

The detection frequency of Lindane (γ−HCH) during the rainy season (76.2% PN, 90.5% ALV, 75.8% PO, and 85.7% MFA) was double the percentages observed during the dry season. As shown in Table 2, during the dry season, Lindane mean residue levels in spinal marrow (97.40 μg/kg lipid) and lung tissue samples (81.34 μg/kg lipid) from PN were significantly higher ($P < 0.05$) as this pesticide was not detected in other tissue samples from this location. High mean concentrations ($P < 0.05$) of Lindane were also found in adipose tissue samples (114.54 μg/kg lipid) from MFA. As can be seen in Table 3, the highest mean residue levels ($P > 0.05$) were detected in muscle and lung samples from PO (995.80, 416.90 μg/kg lipid) followed by kidney samples in MFA (358.10 μg/kg lipid), liver samples from ALV (217.70 μg/kg lipid), and heart tissue samples from PN (160.30 μg/kg lipid), while the lowest concentrations were found in spinal marrow from PN (13.00 μg/kg lipid) and adipose tissue from MFA (10.80 μg/kg lipid).

During the rainy season, Lindane was detected in 100% of the heart, lung, kidney and spinal marrow samples, 92.0% of adipose tissue, and 58.0 and 33.0% of muscle and liver samples, and it was not detected in liver samples during the dry season. The distribution pattern of this metabolite during the dry season was in the order of adipose tissue > lung > spinal marrow > heart > muscle > kidney > liver, and during the rainy season the distribution changed to muscle > lung > kidney > liver > heart > adipose tissue > spinal marrow. A similar distribution was noted by Covaci et al. (2004) who found that γ−HCH was accumulated at a higher concentration in lung and brain pork tissues, and by Siddiqui, Nigam, Kaul, and Seth (1996) who reported that the overall distribution of γ−HCH was greatest in lung, kidney, brain, liver, and heart tissues of dukray strain rats of 20 weeks of age. As explained previously, the spinal marrow may contain lower concentrations of Lindane during the rainy season due to higher lipid contents (36.85 and 26.92%) and to a different lipid composition from that of fat, as in muscle this pesticide is bound to triglycerides that constitute more than 90% of the total lipid content of the tissue.

The difference of distribution patterns of γ−HCH in tissues between seasons and locations could be attributed to a combination of husbandry practices, effect of season and variation in kinetics due to physical–chemical properties of the isomer. Different metabolic activities of each tissue for every isomer could be related to lipid mobilization, leading to elevated levels of the isomer among target tissues. Potential heterogeneous partitioning of contaminant within tissues is of particular concern because the analytical sample is often a very small proportion of the whole organ. Furthermore, possible non-homogeneity in the concentrations of pollutants may be due to remobilization of the lipids and contaminants (Tilbury, Stein, Meador, Krone, & Chan, 1997). Adipose tissue storage is non-permanent because the normally slow release of HCH and its metabolites into the circulating blood stream is increased during periods of accelerated lipid mobilization which can potentially lead to elevated level of pollutants in the remaining lipid of the tissue and in other tissues (Wania, 1999). van Loon et al. (2005) studies in lipid metabolism have led to an alternative mechanism, suggesting that elevated non-esterified fatty acids (NEFA) delivery and/or impaired FA oxidation result in intra-myocellular accumulation of

triacylglycerol and FA metabolites (such as fatty acyl-CoA, ceramides and diacylglycerol), reducing residence time of the lipid deposits as fat sources are mobilised to contribute to the energy demands. The lipid metabolization from adipose tissue, caused by increased animal energy expenditure on exposure to high temperatures characteristic of tropical areas during rainy seasons, may led to the mobilization of organochlorine pesticides carried in lipoproteins and taken up by the liver. These may be subjected to metabolism by the liver before entering the systemic circulation and becoming available to muscle tissues as γ−HCH is bounded to triglycerides that represent more than 90% of the total lipid content of this tissue. Uptake by other tissues and organs also occurs, in some cases resulting from induction of and affinity for enzyme systems such as aryldehydrogenase, and in other cases from the lipid content of the tissue. In the case of liver, HCH and metabolites were almost always detected in elevated concentrations, which is consistent with organ functionality.

Ratios of α-HCH/γ-HCH below unity are used as indicators of recent γ-HCH inputs into the environment (Krauthacker, Romanic, & Reiner, 2001). It is generally considered that a high ratio of α/γ (>4) is an indication of recent use of technical HCH (Kalantzi et al., 2001). During the dry season, mean levels of α−HCH in lung tissue samples from the four locations were higher ($P < 0.05$) than those found in other tissue samples. This is probably a reflection of the high volatility of this isomer. As the Henry's law constant for α−HCH is twice that of γ−HCH, it is more readily removed from air by rain and deposited in soils and then α−HCH partitions to the air, which is reflected in the higher concentrations detected in lung samples. Multivariate analysis (data not shown) indicated statistical differences between tissue α/γ ratios of dry and rainy seasons: α/γ ratios for liver (133,632.0), kidney (44.3), heart (12.4), muscle (8.2) and lung (2.63) tissue samples of the dry season were significantly higher ($P < 0.05$) than α/γ ratios for liver (0.6), kidney (0.03), heart (0.3), muscle (0.00001), and lung (0.93) tissues during the rainy season, signifying higher mean residue levels of γ−HCH in tissues samples during this season. However, adipose tissue α/γ HCH ratio for the rainy season (0.9) was higher ($P < 0.05$) than for dry season ratio (0.1). Moreover, α/γ ratio for liver tissue samples from PO (176,888.0), FMA (148,179.0), PN (124,023.0), and ALV (85,436.0) corresponding to the dry season were significantly higher ($P < 0.05$) than rainy season α/γ ratios for liver tissue samples from PO (105,518.0), MFA (0.0003), PN (0.00005), and ALV (1.0). The seasonal fluctuation of the α/γ ratio is evidence for temperature-dependent photo-isomerization, as was suggested by Oehme, Haugen, and Schlabach (1996). However, other factors such as the different rates of atmospheric volatilization and deposition of the isomers, the volatilization flux of γ−HCH which is controlled by temperature over land and by winds may all be influenced by precipitation patterns (Semeena, Feichter, & Lammel, 2005). The seasonal use of Lindane or technical-grade HCH could also explain the fluctuations in the α/γ ratio (Oehme, Haugen, & Schlabach, 1996). Our results indicated that the ratio α-/γ-HCH varied from 176,888.0 (liver-dry season) to 1.0 (liver-rainy season) that reflects, respectively, an old and recent animal exposure to Lindane due to an intensive and extensive pesticide application during the rainy season, as low ratios, particularly below one, indicate recent input. According to Lorber et al. (1994) the farming system can potentially affect exposure because pesticide deposited/sorbed from the atmosphere onto the pasture surface can be consumed by cattle from soil. The daily intake of soil by beef cattle is 180 kg/year from grazing fresh grass/silage or hay (Fries & Faustenbach, 1990), and/or feeding on harvested forage. Soil ingestion also affects contaminant uptake by cattle and other ruminants but this can occur in animals kept outdoors on pasture and in those fed on grass silage indoors. Foliar uptake seems to contribute more significantly towards plant intake of most organic contaminants; temperature, rainfall and wind are the main environmental conditions influencing the content of OCPs in plants (Sharpe & Livesey, 2005). This is particularly important for grassland systems where subsequent bioaccumulation into grazing livestock becomes a consideration. In our study, cattle from PO and MFA production units were raised mainly on grass grazing pasture, supplemented with a grain-based mixture of cereals, poultry blood meal, animal fat and a commerical premix containing minerals and vitamins. Meanwhile, bovines from PN and ALV were kept inside feeding facilities and fed in feedlots with chopped dried grasses, premixed with dry barley hay fibre, cereals, poultry meal, and a commercial premix containing minerals and vitamins. Thus, higher contamination levels in bovines produced by grazing management practices are probably due to higher source of contamination of grazing areas influenced by season climate, strong exposures of soil to pesticides in the past, and feeding diets formulated with heavily contaminated feed ingredients. In this context, technical-grade HCH has been used in Veracruz, México as an acaricide to control external parasites infesting cattle to treat against ticks; these management practices have resulted in significantly higher ($P < 0.05$) mean levels of α−HCH, γ−HCH, and β−HCH (74.70, 135.23, 347.60 μg/kg lipid, respectively) in grass Star of Africa (*Cynodon plectostachyus*) in this agrarian SW region of Veracruz (Manríquez-Mendoza, 2004).

In reference to the Σ−HCH mean levels, showed in Table 2, during the dry season, among Σ−HCHs, α−HCH contributed more compared to other isomers, followed by δ−HCH; the highest significant ($P < 0.05$) mean concentrations were detected during the rainy season in lung and muscle tissue samples from PO (1,690.10 and 1,434.00 μg/kg lipid), and the contribution of β−HCH was higher in all tissues, followed by γ−HCH and δ−HCH, as shown in Table 3.

The pattern of tissue contamination detected in this study was different to the contamination levels found in previous studies (Waliszewski et al., 1996b). In this 1996 study, lung tissue mean levels of α−HCH (105.00 μg/kg lipid), β−HCH (232.00 μg/kg lipid) and γ−HCH (163.00 μg/kg lipid) were significantly different ($P < 0.05$) to other tissues concentrations, and the mean residues detected in muscle were β−HCH (310.00 μg/kg lipid) and γ−HCH (91.00 μg/kg lipid). No δ−HCH residues were detected above its detection limit. In the present study, during the rainy season the mean levels of α−HCH (133.06 μg/kg lipid), β−HCH (315.2.00 μg/kg lipid), and γ−HCH (160.55 μg/kg lipid) observed in lung tissue samples, and the mean residues of β−HCH (252.00 μg/kg lipid), γ−HCH (329.00 μg/kg lipid), and δ−HCH (109.13 μg/kg lipid) detected in muscle tissue samples were higher than in the previous study, indicating an increased time trend probably due to a continuous usage of technical HCH and Lindane in the region. The above observations suggest a concern about the consequences of the HCHs bioaccumulation in bovine meat and tissues consumed as food in México. When compared to other studies the mean levels detected in our study are considerable higher than those reported in other parts of the world where HCH has been used in animal husbandry. Osibanjo and Adeyeye (1997) reported mean concentration levels of Lindane in heart (35.0 μg/kg lipid), liver (30.0 μg/kg lipid), kidney (25.0 μg/kg lipid), and muscle (14.0 μg/kg lipid) of cows, collected from slaughter houses in south western Nigeria. Glynn et al. (2000) reported a median concentration of α−HCH of 0.7 μg/kg lipid from bulls 36 old or younger in subcutaneous adipose tissue samples in big slaughter houses in Sweden. Manirakiza et al. (2002) detected α−, β−, and γ−HCH (0.6, 0.2, and 1.0 μg/kg fat) and mean concentration levels of Σ−HCHs (1.0–4.6 μg/kg fat) in cattle fat samples collected from local butchers and abattoirs in the Greater Banjul area. Covaci et al. (2004) analysed tissue samples (abdominal fat, liver, lung, brain, spinal marrow, muscle, heart, kidney, spleen) obtained from domestic pork (*Sus scrofa*, n¼ 4) from 4 different farms located in Romania. β−HCH was the most

persistent HCH isomer in all tissues, accounting for 40–97% of the sum of HCHs. For all animals, the highest concentrations of β−HCH (303.5 μg/kg lipid) and HCHs were found in liver, the highest residue levels of γ−HCH (23.4, 14.6 μg/kg lipid) in lung and muscle, and α−HCH (8.8, 6.3 μg/kg lipid) was detected in lung and fat, but the lowest α− and γ−HCH concentrations were measured in brain (1.1, 0.6 μg/kg lipid) and spinal marrow (0.8, 0.4 μg/kg lipid), respectively, where the highest lipid concentration was found (24.0, 29.6% lipid). Darko and Acquaah (2007) found that the average concentrations of lindane in fat from the Kumasi Abattoir (4.04 μg/kg) and Buoho Abattoirs (1.79 μg/kg) were higher than those in lean meat (2.07 and 0.60 μg/kg). Sallam, Mohammed, and Morshedy (2008) determined the organochlorine pesticide residues in a total of 270 meat samples, comprising the muscle, liver, and kidney collected from 90 carcasses (30 each of native breeds of camel, cattle and sheep) slaughtered in Sharkia Province, Egypt. Lindane levels in muscle (0.58 ng/g ww, 16.20 μg/kg lipid) were significantly lower ($P < 0.01$) than its levels in liver (5.13 ng/g ww, 119.03 μg/kg lipid) or kidney (4.57 ng/g ww, 88.40 μg/kg lipid), while no significant difference ($P > 0.05$) was detected between the levels in liver and kidneys. Ahmad, Salem, and Estaitieh (2010) analysed meat samples (beef and lamb), produced and imported into Jordan from Australia, China and India, and detected α−HCH, β−HCH and Lindane (53, 28 and 208 μg/kg fat basis). All meat samples that exceeded the MRL for Lindane were imported from India. The difference among these countries and Mexico could be related to the wide and intensive use of HCH in Mexico, as approximately 261 ton/year of Lindane were imported and sprayed during 1990–2000 (Walker, 2009). Lindane was introduced for agricultural purposes, for insect pest and parasite control in livestock mainly in the tropical regions and its use still continues in the country. Its low price increased its availability, which in addition to management negligence involving the use of inadequate pesticide concentrations and frequent applications, lead to the widespread presence of their residues in different types of foods and in the environment at levels which represented an acute problem.

The concentrations detected in muscle, liver, kidney and spinal marrow tissue samples - habitually eaten in México, were compared with the recommended tolerance limits set by current Mexican and International Regulations. During the rainy season the concentrations of α−HCH in bovine liver from ALV and lung tissues from PO (207.63 and 307.08 μg/kg lipid) were 1.0 and 1.5 times, respectively, above the maximum residue limit (MRL) of 200.0 μg/kg fat, according European Union (2009). The mean levels of β−HCH during the rainy season exceeded the MRL 100.00 μg/kg fat recommended by FAO/WHO (2008b) and the European Union (2009) in the following tissue samples: bovine muscle residues from PN, ALV, PO, and MFA were 1.8, 2.3, 5.0, and 1.1 times, respectively; liver tissues levels from ALV, PO, and MFA were 2.8, 3.5, and 3.5 times, respectively, and kidney tissues concentrations from ALV, PO, and MFA were 2.6, 1.1, and 6.4 times, respectively. In reference to Lindane, the MRL of 20.0 μg/kg fat according European Union (2009) was exceeded during the dry season in meat samples from PO 1.0 times, in spinal marrow samples from PN, ALV, and MFA 4.9, 1.2, and 1.6 times, respectively. During the rainy season this MRL was exceeded 3.8 times by the mean levels found in muscle samples from PN, 2.0 and 4.3 times by residues in kidney tissues from PN and PO, respectively, and 2.7 times in spinal marrow samples from ALV. Lindane mean levels detected during the rainy season above the MRL 100.0 μg/kg fat recommended by FAO/WHO (2008b) were observed in meat samples from PO, MFA and ALV by 10.0, 1.4 and 1.1 times, respectively; in liver tissues from ALV, PN and MFA by 2.2, 1.4 and 1.3 times, respectively. Lindane concentrations in kidney tissues above this MRL were found in MFA and ALV by 3.6 and 1.5 times, respectively. This has to be viewed with serious concern, as Lindane is carcinogenic in nature (IARC, 2008), and recently this pesticide has been implemented in the list of SC on POPs (Conference of the Parties to the Stockholm Convention, 2009). The MRL for Lindane (7,000.0 μg/kg fat according to Mexican Regulation NOM-004-ZOO-1994 SAGARPA (2001) was not exceeded by any tissue sample analysed in this study. The maximum residue limit of Σ−HCH in meat 1000.0 μg/kg lipid recommended by FAO/WHO (2008b) was exceeded 1.4 times in muscle from PO and 1.3 times in kidney from MFA during the rainy season. These levels of contamination detected in bovine tissues are broadly consistent with the concentrations that we have detected in cow's milk. We have reported that mean levels of β-HCH and γ-HCH in cow's milk samples collected from the same region were 3.5 and 13.0 times above the FAO/WHO MRLs for these pesticides (Pardío et al., 2003). These higher concentrations may be a result of the intensive use of technical-grade HCH in livestock husbandry in the region and represent a threat to public health according to current toxicological research findings.

3.1.2. DDT and metabolites

Residual concentrations of DDT and metabolites during the dry and rainy seasons from the different locations are presented in Tables 4 and 5.

The percentage of positive samples of p,p′-DDE in bovine tissue samples during the dry season were 100.0% (PN), 95.2% (ALV), 100.0% (PO), and 90.5% (MFA), representing about two times the frequency detected during the rainy season. Mean residue levels of p,p′-DDE were detected in 100.0% of muscle, liver, heart, lung, kidney, 89.0% in spinal marrow samples, and 83.0% in adipose tissue during the dry season. During the rainy season this metabolite was detected in 75.0% of muscle, 50.0% in liver and heart, 42.0 and 44.0% in lung and spinal marrow, and 33.0% in kidney and adipose tissue samples. As shown in Table 4, during the dry season significantly higher mean residue levels of this metabolite were detected in kidney tissues from PN (232.23 μg/kg lipid) and in lung tissues from PO and MFA (401.73, 294.72 μg/kg lipid, respectively). During the rainy season (Table 5) mean levels of p,p′-DDE were significantly higher ($P < 0.05$) in muscle and lung tissues samples from PO (218.30, 265.80 μg/kg lipid, respectively) and in liver tissues from MFA (107.10 μg/kg lipid). The distribution pattern of this metabolite during the dry season showed affinity for storage in lung, decreasing in the order of lung > kidney > liver > heart > adipose tissue > spinal marrow > muscle, and during the rainy season the distribution changed to lung > liver > muscle > spinal marrow > kidney > heart > adipose tissue. This distribution pattern is different to the distribution in female pork tissues reported by Covaci et al. (2004): fat > muscle > liver > lung > kidney > heart > brain. These high levels of p,p'-DDE detected in lung tissues could be related to the elevated atmospheric concentrations of ΣDDT that have been reported in air (*p,p′*-DDT + *o,p′*-DDT + *p,p′*-DDE + *o,p′*-DDE + *p,p′*-DDD + *o,p′*-DDD) of 1166.7 pg m^{-3} in Veracruz, Mexico (Wong et al., 2009).

The frequency of detection of p,p′-DDD in bovine tissue samples during the rainy season (57.1% PN, 61.9% ALV, 55.6% PO, 66.7% MFA) was similar to the percentages isolated during the dry season, except for PO which was 88.9% isolated during the dry season compared to 55.6% for the rainy season. The contribution pattern of this metabolite during the dry season was 83.0% in muscle and lung samples, 78.0, 67.0, 58.0, 50.0, 25.0% in spinal marrow, heart, adipose tissue, kidney, and liver tissues, respectively; during the rainy season it was distributed 83.0% in muscle and liver, 78.0% in spinal marrow, 58% in heart and lungs, and 33% in kidney and adipose tissues. As can be seen in Table 4, during the dry season the highest p,p'-DDD residue levels, significantly higher at $P < 0.05$, were detected in heart and lung from PO (404.30, and 1,103.50 μg/kg lipid), and in lung from MFA (533.10 μg/kg lipid,

Table 4
Residues of DDT and metabolites (μg/g lipid basis) in bovine tissues from different areas during dry season.

Pesticide	% of samples positive	Tissues						
		Muscle	Liver	Heart	Lung	Kidney	Spinal marrow	Adipose tissue
Puente Nacional								
p,p′-DDE	100.0	43.80 ± 32.96[a]	89.50 ± 76.48[a]	79.53 ± 62.54[a]	84.58 ± 34.49[a]	232.23 ± 12.45[b]	137.08 ± 71.31[a]	58.93 ± 50.72[a]
p,p′-DDD	47.6	49.00 ± 62.10[a]	0.0002[b]	14.50 ± 4.09[a]	93.00 ± 2.00[a]	0.0002[b]	385.20 ± 89.40[c]	29.00 ± 9.40[a]
o,p′-DDT	47.6	85.60 ± 18.60[a]	0.0002[b]	25.40 ± 1.30	151.60 ± 19.10[a]	0.0002[b]	673.30 ± 31.10[c]	50.70 ± 16.40[a]
p,p′-DDT	28.6	0.002[a]	0.002[a]	32.80 ± 2.23[b]	68.70 ± 33.50[b]	68.90 ± 8.07[b]	626.10 ± 45.38[c]	0.002[a]
Σ−DDT		133.50 ± 76.40[a]	89.50 ± 76.50[a]	103.70 ± 49.80[a]	316.30 ± 44.50[a]	255.20 ± 81.80[a]	1404.30 ± 82.90[b]	112.00 ± 29.10[a]
DDE/DDT		*	*	2.625	1.771	3.362	0.368	*
Alvarado								
p,p′-DDE	95.2	25.97 ± 6.02[a]	82.64 ± 58.66[a]	85.39 ± 69.15[a]	109.67 ± 82.56[a]	130.22 ± 61.13[a]	33.17 ± 8.91[a]	33.54 ± 23.67[a]
p,p′-DDD	52.4	9.30 ± 3.80[a]	9.10 ± 1.15[a]	153.50 ± 52.90[b]	112.10 ± 25.40[b]	161.70 ± 16.80[b]	41.00 ± 39.30[a]	84.30 ± 10.33[a]
o,p′-DDT	42.9	16.30 ± 6.60[a]	0.0002[b]	457.20 ± 3.62[c]	225.30 ± 3.00[b]	282.70 ± 2.89[b]	71.70 ± 68.80[a]	147.30 ± 0.00[a]
p,p′-DDT	38.1	42.30 ± 1.13[a]	8.20 ± 5.21[a]	0.002[a]	0.002[a]	315.60 ± 5.44[b]	50.10 ± 4.50[a]	385.30 ± 12.70[b]
Σ−DDT		57.20 ± 28.80[a]	88.40 ± 53.80[a]	340.10 ± 70.10[b]	334.60 ± 49.60[b]	383.60 ± 86.30b	158.40 ± 96.30[a]	356.40 ± 60.42[b]
DDE/DDT		0.619	2.269	*	*	0.681	0.675	0.680
Paso de Ovejas								
p,p′-DDE	100.0	69.57 ± 55.59[a]	163.28 ± 14.57[a]	196.11 ± 50.75[a]	401.73 ± 12.19[b]	127.67 ± 22.48[a]	n.a.	129.02 ± 93.470[a]
p,p′-DDD	88.9	47.60 ± 35.80[a]	60.30 ± 3.44[a]	404.30 ± 22.60[b]	1103.50 ± 48.40[b]	138.80 ± 23.30[a]	n.a.	151.30 ± 80.70[a]
o,p′-DDT	83.3	83.20 ± 62.50[a]	105.40 ± 0.00[a]	706.70 ± 14.20[b]	1994.50 ± 43.30[b]	242.70 ± 40.70[a]	n.a.	264.50 ± 15.90[a]
p,p′-DDT	0.0	0.002[a]	0.002[a]	0.002[a]	0.002[a]	0.002[a]	n.a.	0.002[a]
Σ−DDT		200.40 ± 45.00[a]	218.50 ± 98.00[a]	1307.10 ± 83.50[b]	2834.90 ± 42.10[b]	509.10 ± 57.20[a]	n.a.	544.90 ± 54.60[a]
DDE/DDT		*	*	*	*	*	n.a.	*
Manlio Fabio Altamirano								
p,p′-DDE	90.5	25.74 ± 11.21[a]	211.84 ± 73.38[a,b]	102.99 ± 73.30[a,b]	294.72 ± 32.79[b]	80.24 ± 32.91[a]	17.65 ± 3.22[a]	129.49 ± 72.70[a,b]
p,p′-DDD	66.7	20.60 ± 7.70[a]	23.20 ± 7.74[a]	363.50 ± 57.30[b]	533.10 ± 59.40[b]	62.20 ± 32.50[a]	42.30 ± 9.40[a]	344.00 ± 24.02[b]
o,p′-DDT	61.9	36.10 ± 13.50[a]	40.50 ± 2.42[a]	635.30 ± 24.50[b]	931.90 ± 53.50[b]	68.60 ± 3.00[a]	74.00 ± 16.40[a]	601.30 ± 12.46[b]
p,p′-DDT	38.1	65.90 ± 4.03[a]	0.002[a]	59.90 ± 1.20[a]	225.40 ± 19.07[c]	104.90 ± 41.50[a]	0.002[a]	333.00 ± 64.30[c]
Σ−DDT		104.40 ± 62.80[a]	233.00 ± 52.70[a]	808.80 ± 41.50[b]	1834.80 ± 94.30[c]	214.50 ± 16.40[a]	133.90 ± 22.60[a]	935.10 ± 35.40[b]
DDE/DDT		0.630	*	1.819	3.110	0.744	*	0.896

Means with different letters were statistically different at $P < 0.05$ among tissues.
Σ–DDT = p,p′-DDT + p,p′-DDE + p,p′-DDD + o,p′-DDT; n.a. = not analysed; *value not calculated as metabolite was detected below LQD (not detected).

Table 5
Residues of DDT and metabolites (μg/g lipid basis) in bovine tissues from different areas during rainy season.

Pesticide	% of samples positive	Tissues						
		Muscle	Liver	Heart	Lung	Kidney	Spinal marrow	Adipose tissue
Puente Nacional								
p,p′-DDE	28.6	54.70 ± 7.80[a]	19.20 ± 4.78[a]	6.70 ± 1.36[a]	0.0002[b]	25.30 ± 1.46[a]	91.40 ± 26.00[a]	0.0002[b]
p,p′-DDD	57.1	147.33 ± 26.08[a]	87.84 ± 7.60[a]	103.69 ± 8.51[a]	71.78 ± 5.40[a]	45.88 ± 2.60[a]	205.10 ± 18.79[c]	0.0002[b]
o,p′-DDT	57.1	257.50 ± 45.60[a]	153.50 ± 13.30[a]	181.30 ± 5.46[a]	125.50 ± 4.60[a]	80.20 ± 8.03[a]	358.50 ± 32.80[b]	0.0002[c]
p,p′-DDT	38.1	105.00 ± 26.80[a]	127.10 ± 18.57[a]	161.90 ± 18.13[a]	27.60 ± 2.85[a]	0.0005[b]	181.10 ± 14.69[a]	3.10 ± 0.70[a]
Σ−DDT		493.10 ± 97.10[b]	290.20 ± 15.20[a]	226.80 ± 31.20[a]	224.90 ± 31.02[a]	75.70 ± 71.30[a]	684.90 ± 67.10[b]	3.10 ± 0.70[a]
DDE/DDT		0.548	0.644	0.453	0.000004	*	0.501	0.441
Alvarado								
p,p′-DDE	61.9	56.20 ± 74.40[a]	228.60 ± 10.28[a]	16.30 ± 6.30[a]	15.20 ± 9.20[a]	50.80 ± 25.80[a]	76.40 ± 3.93[a]	6.20 ± 0.13[a]
p,p′-DDD	61.9	112.47 ± 83.90[b]	357.34 ± 68.74[c]	21.05 ± 4.50[b]	35.45 ± 26.09[b]	308.82 ± 8.05[c]	449.85 ± 33.93[c]	174.53 ± 7.00[a]
o,p′-DDT	61.9	196.60 ± 46.70[a]	624.60 ± 95.00[c]	36.80 ± 7.90[b]	62.00 ± 45.60[b]	539.80 ± 28.15[c]	786.30 ± 46.80[c]	305.10 ± 10.28[a]
p,p′-DDT	52.4	277.50 ± 40.50[a]	310.10 ± 56.50[a]	8.00 ± 0.48[b]	14.50 ± 0.89[b]	5.00 ± 0.64[b]	527.50 ± 51.45[c]	4.00 ± 1.90[b]
Σ−DDT		539.80 ± 51.00[b]	1264.90 ± 602.70[c]	76.90 ± 12.00[a]	79.90 ± 63.20[a]	318.40 ± 19.20[a]	1840.10 ± 0.00[c]	164.60 ± 72.80[a]
DDE/DDT		0.284	0.740	2.556	1.141	10.580	0.157	1.282
Paso de Ovejas								
p,p′-DDE	38.9	218.30 ± 81.40[a]	98.90 ± 9.37[b]	34.50 ± 3.10[b]	265.80 ± 47.60[a]	0.0002[c]	n.a.	35.20 ± 5.14[b]
p,p′-DDD	55.6	480.86 ± 49.75[c]	168.90 ± 21.07[a]	148.24 ± 13.65[a]	43.69 ± 14.70[b]	282.29 ± 58.96[a]	n.a.	273.28 ± 14.11[a]
o,p′-DDT	55.6	840.60 ± 46.60[c]	295.20 ± 21.60[a]	259.10 ± 26.87[a]	76.40 ± 25.14[b]	493.40 ± 37.60[c]	n.a.	477.70 ± 69.40[c]
p,p′-DDT	33.3	142.90 ± 19.10[a]	0.002[b]	123.10 ± 28.40[a]	1970.30 ± 81.37[c]	0.002[b]	n.a.	209.30 ± 24.20[a]
Σ−DDT		1121.70 ± 74.60[a]	497.10 ± 37.20[b]	565.00 ± 33.60[b]	1310.90 ± 65.90[a]	775.70 ± 12.50[b]	n.a.	651.90 ± 40.10[b]
DDE/DDT		1.537	*	0.286	0.286	*	n.a.	0.272
Manlio Fabio Altamirano								
p,p′-DDE	57.1	54.70 ± 27.90[a]	107.10 ± 12.60[b]	18.00 ± 1.24[a]	49.40 ± 4.95[a]	43.40 ± 4.63[a]	31.10 ± 3.20[a]	13.90 ± 3.80[a]
p,p′-DDD	66.7	107.20 ± 35.51[a]	160.79 ± 21.76[a]	103.35 ± 15.31[a]	39.54 ± 3.44[b]	462.80 ± 34.67[c]	150.60 ± 80.16[a]	64.68 ± 5.70[b]
o,p′-DDT	61.9	187.40 ± 63.80[a]	281.10 ± 36.00[a]	180.70 ± 21.60[a]	69.10 ± 6.00[b]	809.00 ± 39.60[c]	343.70 ± 21.70[a]	113.11 ± 34.57[a]
p,p′-DDT	47.6	95.90 ± 43.80[a]	223.90 ± 12.80[c]	20.20 ± 6.80[a]	0.002[b]	0.002[b]	118.80 ± 4.90[a]	127.30 ± 10.40[a]
Σ−DDT		445.10 ± 61.90[a]	403.60 ± 316.90[a]	202.10 ± 65.20[a]	125.10 ± 19.10[b]	1315.20 ± 36.10[c]	429.70 ± 13.00[a]	195.80 ± 95.20[b]
DDE/DDT		0.692	0.668	0.915	*	*	0.356	0.271

Means with different letters were statistically different at $P < 0.05$ among tissues.
Σ-DDT = p,p′-DDT + p,p′-DDE + p,p′-DDD + o,p′-DDT; n.a. = not analysed; *value not calculated as metabolite was detected below LQD (not detected).

respectively). According to the data in Table 5, during the rainy season significantly higher ($P < 0.05$) residual concentrations of p,p′-DDD were detected in spinal marrow tissues from ALV (449.85 μg/kg lipid), in liver tissues from PN (357.34 μg/kg lipid), in muscle tissues from PO (480.86 μg/kg lipid), and in kidney tissue samples from MFA (462.80 μg/kg lipid). Fries, Marrow, and Gordon (1969) demonstrated that rumen microorganisms are very effective in converting o,p′-DDT and p,p′-DDT to the corresponding p,p′-DDD isomer. Tebourbi, Driss, Sakly, and Ben Rhouma (2006) observed that p,p′-DDD accumulation was higher than that of p,p′-DDE in liver, explained by a rapid metabolism of p,p′-DDT to p,p′-DDD by the hepatic tissue, since this organ plays a crucial role in the detoxification process and constitutes the main centre for degradation of xenobiotics. This active role in detoxification may explain the liver tissue ratio DDD/DDT of 1.3 during the rainy and 3.8 during the dry season. Wisniewski, Moody, Hammock, and Shull (1987) reported that cattle livers had the lowest microsomal and cytosolic activities and goat livers had the highest, revealing differences in xenobiotic metabolism among cattle, goats and sheep.

During the dry season the distribution pattern of this metabolite showed the affinity for storage in lung, decreasing in the order of lung > heart > spinal marrow > adipose tissue > kidney > muscle > liver, and during the rainy season the distribution change to kidney > spinal marrow > liver > adipose tissue > muscle > heart > lung. These distribution patterns were different to the distribution of DDTs in female pork tissues reported by Covaci et al. (2004): liver > muscle > adipose tissue > kidney > lung > heart > spinal marrow.

The frequency of detection of o,p′-DDT in bovine tissue samples during the rainy season (57.1% PN, 61.9% ALV, 55.6% PO, 61.9% MFA) was similar to the percentages of isolation during the dry season, except for PO which was 83.3% isolated during the dry season compared to 55.6% for the rainy season. The frequency of detection of this metabolite during the dry season was 83.0% in muscle samples, 78.0 and 75.0% in spinal marrow and lung tissues, 58.0% in adipose tissue, 42.0 and 17.0% in kidney and liver tissues; during the rainy season this metabolite showed a similar pattern. It was detected in 83.0% in muscle and liver tissue samples, 67.0% in spinal marrow, 58.0% in heart and lung tissues, and 33.0% in kidney and adipose tissues. As it can be observed from Table 4, during the dry season significantly higher ($P < 0.05$) residual concentrations of o,p′-DDT were observed in spinal marrow from PN (673.30 μg/kg lipid), in lung and heart tissues from PO (1,994.50, 706.70 μg/kg lipid, respectively), and in lung from MFA (931.90 μg/kg lipid). As shown in Table 5, during the rainy season significantly higher ($P < 0.05$) mean levels of o,p′-DDT were found in spinal marrow from PN and ALV (358.50, 786.30 μg/kg lipid), in liver tissue from ALV (624.60 μg/kg lipid), in muscle tissues from PO (840.60 μg/kg lipid), and in kidney tissues from MFA (809.00 μg/kg lipid).

During the dry season, the distribution pattern of this metabolite showed an affinity for storage in lung, decreasing in the order of lung > heart > spinal marrow > adipose tissue > kidney > liver > muscle; meanwhile, during the rainy season, the distribution changed to kidney > spinal marrow > liver > adipose tissue > muscle > heart > lung. These concentrations detected in lung tissues represent possible current regional and seasonal background values of DDTs levels monitored in air within the central agrarian region of Veracruz by Wong et al. (2009) who observed fresher DDT residues at Veracruz, México and also o,p′-DDT. This agrees with the former heavy use of DDT in this endemic malarious area of the country.

In reference to p,p′-DDT, the relative contribution of this isomer in bovine samples during the rainy season (38.1% PN, 52.4% ALV, 33.3% PO, 47.6% MFA) was 1.3 times the contribution during the dry season, except for PO where this isomer was not detected in any tissue sample from PO. The frequency of detection during the dry season p,p′-DDT showed a lower pattern, being detected in 44.0% of spinal marrow samples, 33.0% of lung, kidney, and adipose tissues, 25.0, 17.0, and 8.0% of heart, muscle and liver tissue samples. During the rainy season the frequency of detection of this metabolite was 83.0% in muscle, 75.0% in adipose tissue, 42.0 and 41.0% in lung and liver tissues, 33.0% in heart and spinal marrow, and 8.0% in kidney tissue samples. During the dry season (Table 4) significantly higher ($P < 0.05$) mean concentrations of p,p′-DDT were detected in spinal marrow from PN (626.10 μg/kg lipid), in adipose tissue samples from ALV (385.30 μg/kg lipid), in lung and adipose tissues from MFA (225.40 and 333.00 μg/kg lipid), and it was not detected in any tissue sample from PO. During the rainy season (Table 5), significantly higher ($P < 0.05$) residual concentrations of p,p′-DDT were observed in spinal marrow from ALV (527.50 μg/kg lipid), and in lung tissues from PO (1,970.30 μg/kg lipid).

The distribution pattern of p,p′-DDT during the rainy season was greatest in lung > spinal marrow > liver > muscle > adipose tissue > heart > kidney. As explained before, the high concentrations detected in lung tissues represent possible current regional and seasonal background values of DDTs in the air as DDT was intensively used in this agrarian area. Meanwhile, during the dry season this distribution changed to adipose tissue > spinal marrow > kidney > lung > muscle > heart > liver. High concentrations observed in adipose tissue during the dry season were also reported by Covaci et al. (2004) in female pork tissues in a distribution pattern of adipose tissue > muscle > spinal marrow > heart > liver > kidney > lung.

In reference to the Σ−DDT(Table 4), during the dry season significantly higher ($P < 0.05$) mean residues were detected in spinal marrow from PN (1,404.30 μg/kg lipid), in heart and lung tissues from PO (1,307.10 and 2,834.90 μg/kg lipid), and in heart, lung, and adipose tissues from MFA (808.80, 1,834.80, 935.10 μg/kg lipid, respectively). As shown in Table 5, during the rainy season significantly higher ($P < 0.05$) mean residues of Σ−DDT were found in muscle and spinal marrow tissues from PN (493.10 and 684.90 μg/kg lipid), in liver and spinal marrow from ALV (1,264.90 and 1,840.10 μg/kg lipid), in muscle and lung tissues from PO (1,121.70 and 1,310.90 μg/kg lipid), and in kidney tissues from MFA (1,315.20 μg/kg lipid).

Multivariate analysis of p,p′-DDE/p,p′-DDT ratio (data not shown) indicated that dry ratio (1.19) was significantly higher ($P < 0.05$) than rainy ratio (0.547). The highest ratio ($P < 0.05$) was found in lung tissue samples (1.34); during the dry season, the p,p′-DDE/p,p′-DDT ratios found in ALV and PN (1.36 and 1.30) were significantly higher ($P < 0.05$) than other location ratios, and the ratio in liver during the dry season (5.50) was also significantly higher ($P < 0.05$). As presented in Tables 4 and 5, several tissue samples from different locations showed p,p′-DDE/p,p′-DDT > 1.0, indicating that these tissues accumulated p,p′-DDE from previous exposure through contaminated air, water, soil or feed, or p,p′-DDT has been metabolised to p,p′-DDE (Krauthacker et al., 2001). Furthermore, p,p′-DDT and o,p′-DDT seem equally effective in elevating the levels of the four gluconeogenic enzymes; pyruvate carboxylase, phosphoenolpyruvate carboxykinase, fructose-1, 6-diphosphatase and glucose-6-phosphatase located in kidney cortex (Kacew, Singhal, Hrdina, & Ling, 1972), metabolising p,p′-DDT to p,p′-DDE and p,p′-DDD.

Comparing the results of this study with previous data reported in 1996 (Waliszewski et al., 1996b) for bovine tissues, a decrease in p,p′-DDT was observed in kidney from 1,965.00 to 76.79 μg/kg lipid (25-fold), muscle from 1,571.00 to 143.67 μg/kg lipid (11-fold), lung, liver, and heart tissues (8-fold) from 2,713.00, 955.00, and 506.00 to 349.11, 123.61, and 66.53 μg/kg lipid, respectively, and adipose tissue 467.00 to 173.16 μg/kg lipid (2.7-fold). A slight decrease in p,p′-DDE was also observed in heart and muscle from

94.0 and 75.0 to 83.35 and 62.83 μg/kg lipid (1.1-fold), respectively; nevertheless these metabolite residues increased in lung and adipose tissues from 92.0 and 35.0 to 193.14 and 68.51 μg/kg lipid (2-fold), in liver and kidney tissues from 88.0 and 84.0 to 128.32 and 117.59 μg/kg lipid (1.4-fold). The o,p′-DDT concentrations increased 2.5-fold in muscle tissues from 78.0 to 198.534 μg/kg lipid and in lung tissues from 215.0 to 504.464 μg/kg lipid, and 60-fold in liver tissues from 5.0 to 303.94 μg/kg lipid. When the mean residue levels of p,p′-DDT from 1996 were compared with p,p′-DDT from 2008 tissue concentrations, a decreasing time trend in p,p′-DDT residual concentrations has occurred after a period of 10 years. Such a decline may be related to the fact that the use of DDT in México was restricted for malaria control programmes of the Ministry of Health, according to the Stockholm Convention on Persistent Organic Pollutants signed in 2001 (CICOPLAFEST, 2004; Nuttall, 2009). However, relatively high mean levels of DDT and metabolites were detected in bovine tissues from several producer locations, probably due to the heavy loads of DDT during its past legal use that have resulted in a higher contamination of grazing areas for bovines and the contamination of the food producing animal chain.

When compared to other studies, the mean levels detected in our study are similar; nevertheless, in some bovine tissues from PO and MFA DDT and metabolite levels are considerably higher than those reported in other parts of the world where DDT has been used in sanitary programs. Osibanjo and Adeyeye (1997) reported mean concentration levels of p,p′-DDE and Σ−DDT in heart (91.0, 122.0 μg/kg lipid), liver (88.0, 164.0 μg/kg lipid), kidney (106.0, 148.0 μg/kg lipid), and muscle (33.0, 57.0 μg/kg lipid) of cows collected from slaughter houses in south western Nigeria. Σ−DDT values were highest in the liver; the high residue contents in this organ may be related with its detoxifying function. These authors explained that their results reflected an accumulation through food and other indirect sources, or from historical use. Manirakiza et al. (2002) reported mean residues of p,p′-DDE (34.8 μg/kg fat), p,p′-DDD (0.4 μg/kg fat), o,p′-DDT (0.1 μg/kg fat), p,p′-DDT (3.9 μg/kg fat), and Σ−DDT (39.5 μg/kg fat) cattle fat samples collected from local butchers and abattoirs in the Greater Banjul area. Darko and Acquaah (2007) analysing beef samples collected from the Kumasi Abattoir and Kumasi Meat Packaging Company at Buoho in Ghana, found higher residues of p,p′-DDE and p,p′-DDT in fat samples (118.45, 545.24 μg/kg) than in beef samples (42.93, 18.83 μg/kg), showing that DDT residues, like the other organochlorines, concentrate more in fat than in lean meat. Alcover-Vidal, Castellanos-Ruelas, Herrera-Chalé, Chel-Guerrero, and Betancur Ancona (2007) analysed muscle, kidney and liver samples obtained from non-castrated bovine males of Zebú and F1 (zebu x european) breeds produced in the state of Yucatán, México, collected in either the Faculty of Veterinary Medicine and Animal Husbandry Processing Plant, or the Mérida City Municipal Abattoir. These authors reported no findings of DDTs, which is surprising because Mexico, as with China and India, are DDT producers and have sprayed the pesticide extensively in mosquito control programs. Sallam et al. (2008), when determining OCP residues in cattle slaughtered in Sharkia Province, Egypt, found that the mean values of the residual concentrations of DDTs in muscle samples were 17.9 ng/g ww or 500.0 μg/kg lipid, in liver 57.2 ng/g ww or 1,327.15 μg/kg lipid, and in kidneys 36.3 ng/g ww or 702.13 μg/kg lipid.Liver samples generally showed the highest ($P < 0.01$) contamination followed by kidneys, while muscle samples exhibited the lowest residual concentrations. Glynn et al. (2009) observed that the concentrations of p,p′-DDE in bovine adipose tissue samples declined on average 6% per year from 1991–2004 in Sweden from 3.3 to 2.4 μg/kg lipid. The p,p′-DDE contamination of adipose tissues was higher in southern Sweden than in the northern part of the country, suggesting that the environmental loads of DDT affect the level of contamination in the meat. These low residues found in Sweden were attributed to the efforts of developed countries to reduce organochlorine contamination of animal feed and of the environment and that these have had a positive impact on food producing animals. In most developing countries such as Mexico, cattle are frequently raised on grass and/or crop residues, although supplementation with concentrates is increasing. Thus, the differences in DDT levels detected could also be related to the farming system because pesticides deposited from the atmosphere onto the pasture surface can be consumed by cattle fed with grass, hay or silage. Animals kept outdoors on pasture can ingest soil affecting contaminant uptake as well (Sharpe & Livesey, 2005). The higher contamination levels detected in bovines produced by grazing management practices are probably due to higher source of contamination of grazing areas with high pesticide exposures in the past, and feeding diets formulated with heavily contaminated feed ingredients. In México, young cattle are usually raised on forage or a pasture based diet, both of which have low energy densities. This is followed by a fattening period in which a high energy grain based diet is fed before slaughter. In addition to the complexities added by the feeding system to estimates of growth and intake, the concentration of environmental contaminants is usually considered to be much greater in forages than in grains. As mentioned, our studies in grass pasture (*C. plectostachyus*) indicated high contamination levels of DDT in this agrarian zone. Ahmad et al. (2010) detected p,p′-DDE in 27% of meat samples analysed and the residual concentration of this metabolite was 38 μg/kg fat. The p,p′-DDT was detected in 5.9% of meat, with a mean concentration of 64 μg/kg fat. The p,p′-DDD was found to be present in 2.2% of meat samples with a concentration range varying between 10 and 150 μg/kg fat.

The concentrations detected in muscle, liver, kidney, and spinal marrow tissue samples - consumed habitually as meat in México, were compared with the recommended tolerance limits set by current Mexican and International Regulations. The mean concentrations of Σ−DDT in muscle tissue samples from PO during the rainy season (1,121.70 μg/kg lipid), spinal marrow samples from ALV (1,840.10 μg/kg lipid) during the rainy season and PN (1,404.30 μg/kg lipid) during the dry season, kidney and liver samples from MFA (1,315.20 μg/kg lipid) and ALV (1,264.90 μg/kg lipid) of the rainy season, were 1.12, 1.84, 1.40, 1.31, and 1.26 times above the maximum limit 1,000.0 μg/kg fat according to the European Union (2009). The MRL of 5000.00 μg/kg fat recommended by FAO/WHO (2008b) and Mexican Regulation (NOM-004-ZOO-1994 SAGARPA, 2001) was not exceeded by any tissue sample analysed in this study.

3.1.3. Principal component analysis

To evaluate further the spatial and temporal differences between organochlorine pesticide concentrations, principal component analysis (PCA) was used. PCA was performed using XLSTAT v.2011.2.05 software. The seasons and the sampling sites were the active variables. As it can be seen in Fig. 2A, the first axis represents 71.50% of the explained variance level, and the second one represents 20.81%. Relationships between tissues and seasons and between HCH concentrations explained 92.31% of the total variance. The mean levels of α−HCH were positively correlated to lung and liver tissues during the rainy season and to lung during the dry season, and the greatest levels of γ−HCH correlated to muscle tissues during the rainy season compared to other tissues during the dry and rainy seasons. The separation of α−HCH and lung of the rainy season is of significance as it is fairly persistent, volatile and a ubiquitous OCP. The high levels of α−HCH in lung tissue samples could arise, in part, from its characteristic to be typically predominant in ambient air and because the animal production units are located close to coastal waters. During long-range

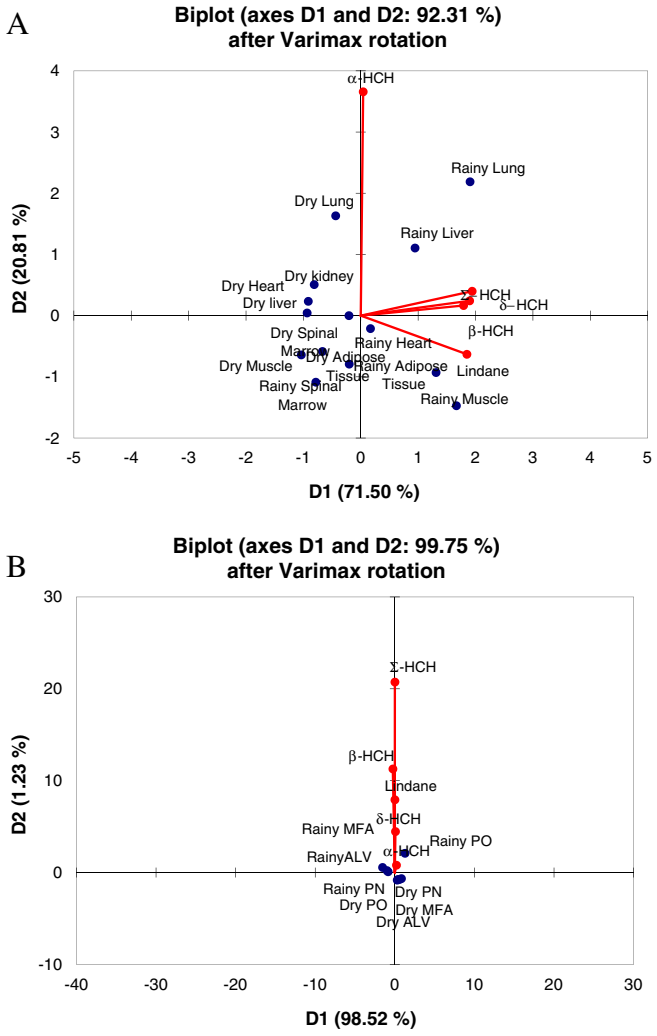


Fig. 2. PCA projections of scores and loadings for the first two principal components for the analysis of HCH and metabolites. Score plot indicates (A) tissues and seasons, and (B) sampling sites and seasons of the loading plot variables.

transport in air over oceans, γ–HCH is more likely to be removed either through direct partitioning into water or through washout in rain, leaving proportionately more α–HCH in the air (Walker, Vallero, and Lewis, 1999). There was also a clear separation of mean levels according to sampling seasons (Fig. 2B) that explained 99.75% of the total variance. The highest residue levels of all HCHs detected in PO during the rainy season was distinguished from the other locations during dry and rainy seasons that cluster in the opposite side of the chart. According to multivariate analysis (data not shown), mean concentration levels of the following HCH and metabolites during the rainy season were significantly higher ($P < 0.05$) than during the dry season: α–HCH (76.61, 56.52 μg/kg lipid), β–HCH (201.64, 24.91 μg/kg lipid), δ–HCH (108.15, 49.54 μg/kg lipid), γ–HCH (136.05, 57.38 μg/kg lipid), and Σ–HCH (417.00, 117.73 μg/kg lipid). During the rainy season, the highest mean residue levels ($P < 0.05$) of α–HCH, β–HCH, δ–HCH, γ–HCH, and Σ–HCH were found in PO and no significant differences ($P > 0.05$) between seasons were detected in other production unit locations. Among tissues analysed, the highest mean concentrations ($P < 0.05$) of α–HCH, β–HCH, δ–HCH, and Σ–HCH were detected in lung tissues and γ–HCH in muscle tissues. During the rainy season, the highest mean concentrations ($P < 0.05$) of α–HCH were detected in liver and lung tissues (182.96, 146.02 μg/kg lipid), β–HCH in lung and kidney samples (315.21, 311.66 μg/kg lipid), δ–HCH in lung tissues (201.73 μg/kg lipid), γ–HCH in muscle samples (360.40 μg/kg lipid), and Σ–HCH in lung samples (657.60 μg/kg lipid). There was evidence that the highest mean levels ($P < 0.05$) were found in lung samples from PO for α–HCH, β–HCH, δ–HCH, and Σ–HCH (186.95, 853.69, 294.12, and 948.73 μg/kg lipid), and γ–HCH in muscle samples (672.70 μg/kg lipid).These levels are significantly higher ($P < 0.05$) than other mean residues in other tissue samples of animal production unit locations.

The compositional pattern of HCH and metabolites during the dry season showed higher levels of α–HCH in muscle, liver, heart, lung, kidney, and spinal marrow decreasing in the order α–HCH > δ–HCH > β–HCH > γ–HCH. This contrasts with the adipose tissue composition of δ–HCH > γ–HCH > β–HCH > α–HCH. Meanwhile, during the rainy season higher levels of β–HCH, decreasing in the order of β–HCH > δ–HCH > α–HCH > γ–HCH, were detected in lung, kidney, spinal marrow, and adipose tissue. These findings indicate that significant different contamination levels exist among locations, tissues, and seasons indicating that evaporation and deposition of HCHs vary with season in tropical areas. Additionally, different degradation rates of HCH isomers may contribute to the variable accumulation of isomers. Moreover, the HCH accumulation differed from one organ to another, probably related to the lipid composition, diet and environmental exposure. In the present study, tissue distribution patterns of HCHs were significantly different between dry and rainy seasons and

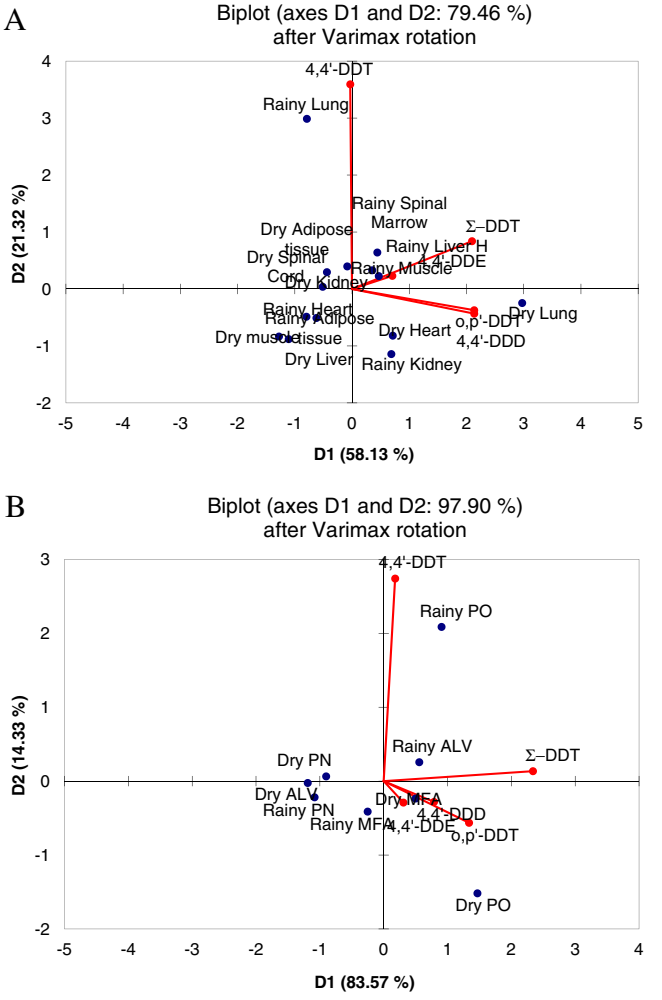


Fig. 3. PCA projections of scores and loadings for the first two principal components for the analysis of DDT and metabolites. Score plot indicates (A) tissues and seasons, and (B) sampling sites and seasons of the loading plot variables.

different geographic locations of production units. According to the geographic distribution (Fig. 1), significantly higher ($P < 0.05$) mean concentrations of α−, β−, and δ−HCH were detected in lung, and liver, and γ−HCH in muscle tissue samples from PO during the rainy season compared to mean concentrations of other production units during the dry season, suggesting that the on-going usage of Lindane and/or technical HCH in this location and livestock management, the influences of regional climate and the physico-chemical properties of this pesticide have significant effects on pesticide accumulation by beef animals.

The PCA analysis also indicated that relationships between tissues and seasons and DDT concentrations explained 79.46% of the total variance (Fig. 3A), indicating that tissue and season samplings represent the 58.13% of the explained variance level. There was also a clear separation of mean levels according to sampling seasons (Fig. 3B) that explained 97.90% of the total variance, indicating that location and season samplings represent the 83.57% of the explained variance level (Fig. 3B). The mean levels of p,p′-DDT were positively correlated to lung tissues and PO during the rainy season, and the levels of 4,4′-DDD and o,p′-DDT correlated to lung and heart tissues during the dry season. Meanwhile, 4,4′-DDE, 4,4′-DDD, o,p′-DDT, and Σ−DDT concentrations correlated to PO during the dry season. The separation of 4,4′-DDT and lung of the rainy season from PO indicated that 4,4′-DDT levels were, in general, higher at this location and still measurable, and reflect the local use in the past and the recent inputs related to transport through the atmosphere and deposition through rain. In accordance with the multivariate analysis (data not shown), the 4,4′-DDE, 4,4′-DDD, o,p′-DDT, and Σ−DDT mean concentration levels during the dry season (119.85, 214.91, 364.96, 518.60 μg/kg lipid) were significantly higher ($P < 0.05$) than levels during the rainy season (72.15, 166.99, 295.85, 476.25 μg/kg lipid). However, p,p′-DDT mean concentration levels during the rainy season (193.81 μg/kg lipid) were significantly higher ($P < 0.05$) than dry season mean levels (159.44 μg/kg lipid). The highest mean residue levels ($P < 0.05$) of 4,4′-DDE, 4,4′-DDD, o,p′-DDT, 4,4′-DDT, and Σ−DDT were found in lung tissue samples (193.14, 330.89, 504.47, 349.11, and 924.94 μg/kg lipid), and during the dry season the highest mean residue levels ($P < 0.05$) of 4,4′-DDE, 4,4′-DDD, o,p′-DDT, and Σ−DDT were detected in PO (181.23, 349.80, 532.37, and 935.81 μg/kg lipid). Meanwhile, 4,4′-DDT mean levels were found to be significantly higher ($P < 0.05$) in PO during the rainy season (466.23 μg/kg lipid). No significant differences ($P > 0.05$) between seasons were detected in other production unit locations.

The compositional pattern of DDT metabolites during the dry season showed higher levels of o,p′-DDT in lung and spinal marrow tissues decreasing in the order o,p′-DDT > 4,4′-DDT > 4,4′-DDD > 4,4′-DDE, in kidney and muscle tissues in the order o,p′-DDT > 4,4′-DDT > 4,4′-DDE > 4,4′-DDD; meanwhile the composition in liver, heart and adipose tissues varied. During the rainy season, higher levels of o,p′-DDT, decreasing in the order of o,p′-DDT > 4,4′-DDT > 4,4′-DDD > 4,4′-DDE, were detected in liver, heart and spinal marrow tissues, and in kidney with the a composition of: o,p′-DDT > 4,4′-DDD > 4,4′-DDE > 4,4′-DDT. The compositional patterns of adipose and muscle tissues were observed in the decreasing order of o,p′-DDT > 4,4′-DDD > 4,4′-DDT > 4,4′-DDE, and lung tissue composition was different from other tissues: 4,4′-DDT > 4,4′-DDE > o,p′-DDT > 4,4′-DDD.

The high concentrations of DDT and metabolites detected in lung tissues may be related to environmental exposure throughout inhalation of contaminated air. DDT and other chlorinated pesticides contaminate soil during application. The residues remain in the soil for 10–15 years after application, representing a continuing source of atmospheric contamination through abiotic processes such as volatilization, often without substantial alteration of the chemical structure (Bidleman & Leone, 2004; Nagabe and Bidleman, 2006). Dissipation of DDT and other chlorinated pesticides from soil is faster in tropical conditions due to increased volatilization and degradation (Wandiga, 2001). Moreover, the volatilization flux of DDT is controlled by temperature over land and by winds over the ocean (Semeena et al., 2005). The high p,p′-DDT concentrations detected in lung tissues during the rainy season in PO (1,970.30 μg/kg lipid) may be a reflection of environmental conditions triggering the volatilization accumulated DDT residues in soil during the dry season. The heavy precipitations during autumn (hurricane season) reduce DDT atmospheric residence time through deposition to soil and plants that will result in higher levels in soil/plant and a continued increase in the ambient air levels in the next early summer. Grazing livestock exposure can occur to DDT residues of soil and ambient air through ingestion of contaminated vegetation, feed, and soil adhering to vegetation, and water. The rate of transfer is a function of factors such as livestock species, climate, grazing habits, etc. In the central agrarian area of Veracruz, bovines are feed with fresh pasture, grass products (hay, grass silage), corn silage and grain concentrates. Average soil ingestion has been estimated as 6–8% when pasture is the only feed source. Our studies in grass pasture (*C. plectostachyus*) collected from Alvarado farms have showed higher ($P < 0.05$) residual concentrations of p,p′-DDT (1,774.0 μg/kg lipid) during the rainy season than dry season levels (395.6 μg/kg lipid) (Hernández-Lara, 2003), indicating the deposition of DDT on pasture due to high temperatures during the rainy season in this agrarian zone. Wan, Kuo, and Pasternak (2005) found that OCP residues were generally highest in crop soils by 50%, as the DT50 rates of these pesticides vary in soils from 4 to 30 year, depending on climatic and other environmental conditions.

These results indicate that the widespread dispersion of DDT in tropical zones of Mexico where intensive applications during past legal uses have resulted in high concentrations of DDT and metabolites of concern to public health. High mean levels of DDT and metabolites were also observed in adipose tissue during the rainy season (Table 5). Retention in adipose tissue may also influence the distribution to other tissues as this tissue is the principal depot for the accumulation of many lipophilic xenobiotics. During accelerated lipid mobilization, the adipose stores are depleted. In times of increased physiological demand, free fatty acids leave the adipose tissue and are transported to muscle, liver and other sites of oxidative degradation. In our study, the mean concentration ratios for DDT and metabolites in paired tissue ratio liver/adipose tissue were 1.9 (p,p′-DDE), 1.0 (p,p′-DDD), and 1.1 (o,p′-DDT), indicating a mobilization of metabolites from adipose tissue to the liver. Mussalo-Rauhamaa (1991) observed that the mean concentration ratios for p,p′-DDE in paired liver/adipose samples were ~2–2.5 times those in humans. Covaci et al. (2004) reported that the order of magnitude for the level of DDTs in pork liver samples was 3 times that of the muscle. As explained before, bovines largely depend on NEFAS for energy (Baumgard et al., 2007). According to Jandacek et al. (2005), an increase in plasma NEFAS and a decrease in circulating lipoproteins suggest the importance of free fatty acids in carrying hexachlorobenzene at the water-triacylglycerol interface during lipolysis. The normally slow release of DDT and its metabolites mobilized from adipose tissue is increased, being mobilized from adipose tissue incorporated into chylomicrons and transported in the blood to the liver and peripheral tissues. As explained, lipid metabolization from adipose tissue, caused by increased energy expenditure due to, e.g., exposure to high temperatures characteristic of tropical areas during rainy seasons, may led to the mobilization of OCPs carried in lipoproteins and taken up by the liver. These may be subjected to metabolism by the liver before they enter the systemic circulation and become available to muscle tissues as OCPs are bound to triglycerides that represent more than 90% of the total lipid content of this tissue. Müllerová and Kopecký (2007) have reported that hydrophobic environmental

contaminants, such as organochlorine pesticides, stored in white adipose tissue (WAT) may modulate the activity of key transcription factors engaged in control of differentiation, metabolism and the secretory function of adipocytes, and consequently influence cellular energy metabolism and fat deposition, affecting the physiological role of WAT. Moreover, according to Dugald, MacLachlana, and Rajumati (2009) body composition also impacts on tissue residues. This author used physiologically-based pharmacokinetic (PBPK) modeling to explore differences between classes of food-producing animals and predicted residues in fat lower in animals with higher body fat and that the half-lives for elimination of residues from the fat compartment increase with increasing body fat.

The distribution pattern of HCHs and DDTs in tissues during the dry season differed significantly from that during the rainy season, indicating that the physicochemical properties of the pesticide, the lipid composition of the tissue, decisive for the tissue-specific pattern of the pesticide distribution, are also influenced by geographical distribution due to use patterns of the pesticide in the past and by seasonal changes and the environmental burden, as DDT has been re-entered in the context of malaria prevention in tropical countries. The more polar metabolites of organochlorine compounds may undergo a different pattern of distribution due to fluctuations in feed and environmental contamination during seasons. To our knowledge, this is the first study in México to report associations between variations in environmental pollutants disposition in bovine tissues produced under tropical conditions in different geospatial locations. These results can provide an indication of the bioaccumulation of organochlorine pesticides in food producing animals and, therefore, the potential exposure to humans through food consumption.

3.2. Human health risk assessment

In recent years the evidence for increased residues of chemical compounds in food as a consequence of environmental pollution has driven the need to screen for the potential public health significance of concentrations and exposures. Risk assessment for persistent compounds, such as organochlorine pesticides, plays an important role concerning consumer health, as well as animal health, economic, and feasibility aspects. In this study, the potential health risks were assessed based on the dietary intake of OCs from meat consumption.

3.2.1. Dietary intake of OCs from bovine muscle and tissue consumption

The estimated dietary intakes of OCPs through bovine muscle and tissue consumption from rainy and dry seasons are summarized in Table 6.

Among tissues analysed, consumer exposure to pesticide residues through consumption of muscle tissues showed the highest contributions to dietary exposure. Dietary intakes from the rainy season were higher than exposures during the dry season. Estimated dietary intakes through consumption of muscle tissues during the rainy season showed the highest contributions for γ−HCH from PO and Σ−DDT from PN (3.35and 1.22 μg/kg bw/day), that would be 0.7 and 0.12 times the recommended ADIs (FAO/WHO, 2009) shown in Table 7. When γ−HCH and Σ−DDT mean residues in muscle tissues reported in 1996 (91.00 and 2,545.00 μg/kg lipid) (Waliszewski et al., 1996b) were compared, a different trend was noted 10 years later. The EDI of γ−HCH through muscle tissue increased from 1.06 μg/kg bw/day to 3.35 μg/kg bw/day, representing a 3.2 times increase, meanwhile Σ−DDT estimated intake from 1996 (29.6 μg/kg bw/day) decreased to 1.22 μg/kg bw/day which means a considerable decline (95.8%) with respect to the past estimated intakes. The increase of γ−HCH intake indicates the continuing use of this pesticide, as tick control with Lindane still prevails in México, and the decrease in Σ−DDT intake may be related to the fact that the use of DDT was restricted in México for malaria control programs until 2003. As previously explained, although several studies have determined the levels of OCPs in

Table 6
Estimated Daily Intakes (EDI) (μg/kg bw/d), Noncancer, and Cancer Hazard Ratios (HR) for daily consumption of contaminants in bovine muscle from rainy and dry season.

Pesticide	EDI (μg/kg bw/d)		Noncancer HR		Cancer HR 50th centile		Cancer HR 95th centile	
	Rainy	Dry	Rainy	Dry	Rainy	Dry	Rainy	Dry
Puente Nacional								
α−HCH	–	–	–	–	2.40E-02	6.70E + 02	2.40E-02	3.61E + 03
β−HCH	–	–	–	–	4.80E + 03	2.83E-03	8.92E + 03	9.60E-03
γ−HCH	8.83E-01	1.71E-06	3.97E + 00	7.70E-06	2.09E + 03	4.04E-03	4.17E + 03	1.37E-02
p,p′-DDE	–	–	–	–	3.78E + 05	8.91E + 04	3.78E + 05	8.69E + 05
p,p′-DDT	–	–	–	–	7.26E + 05	1.02E + 00	1.15E + 06	3.46E + 00
Σ−DDT	1.22E + 00	1.71E-06	4.39E + 00	6.16E-06	-	-	-	-
Alvarado								
α−HCH	–	-	–	–	5.62E-03	8.82E + 02	2.40E-02	1.47E + 03
β−HCH	–	-	–	–	1.47E + 03	6.73E-03	9.05E + 03	9.60E-03
γ−HCH	2.87E-01	4.07E-06	1.29E + 00	1.83E-05	6.78E + 02	9.61E-03	7.12E + 03	2.74E + 02
p,p′-DDE	–	–	–	–	9.09E + 04	1.26E + 05	1.67E + 06	2.83E + 05
p,p′-DDT	–	–	–	–	4.49E + 05	2.05E + 05	7.76E + 06	2.92E + 05
Σ−DDT	7.55E-01	3.44E-01	2.72E + 00	1.24E + 00	–	–	–	–
Paso de Ovejas								
α−HCH	–	–	–	–	6.95E-03	8.92E + 02	2.40E-02	7.21E + 03
β−HCH	–	–	–	–	3.94E + 03	2.60E-03	4.79E + 04	9.60E-03
γ−HCH	11.57E + 00	8.36E-02	1.51E + 01	3.76E-01	7.90E + 03	1.98E + 02	1.50E + 05	1.48E + 03
p,p′-DDE	–	–	–	–	4.36E + 05	1.30E + 05	2.78E + 06	1.44E + 06
p,p′-DDT	–	–	–	–	2.86E + 05	9.36E-01	2.34E + 06	3.46E + 00
Σ−DDT	4.80E-01	13.04E + 00	1.73E + 00	5.67E-06	–	–	–	–
Manlio Fabio Altamirano								
α−HCH	–	–	–	–	5.50E-03	9.71E + 02	2.40E-02	3.75E + 03
β−HCH	–	–	–	–	6.83E + 02	6.63E-03	7.89E + 03	9.60E-03
γ−HCH	3.69E-01	4.01E-06	1.66E + 00	1.81E-05	8.72E + 02	9.48E-03	7.68E + 03	1.37E-02
p,p′-DDE	–	–	–	–	8.66E + 04	1.23E + 05	8.58E + 05	3.70E + 05
p,p′-DDT	–	–	–	–	1.52E + 05	3.15E + 05	1.41E + 06	4.56E + 05
Σ−DDT	2.55E-01	5.29E-01	9.19E-01	1.90E + 00	–	–	–	–

(–) = Not Assessed under the IRIS program.

Table 7
Reference values for persistent organochlorine pesticides and benchmarks concentrations for daily consumption of bovine muscle and organs.

Pesticides	Acceptable daily intake (μg/kg/day)[a]	Oral RfD (μg/kg day)[b]	Cancer slope factor (μg/kg/day)[b]	WOE 86 Guidelines*	Cancer Benchmark concentration (μg/kg/day)[b]	Cancer Benchmark concentration for Meat (μg/kg day)	Cancer Benchmark concentration for Liver (μg/kg day)	Cancer Benchmark concentration for Kidney and Spinal Marrow (μg/kg day)
α−HCH	–	NA	6.3	B2	-	0.0001634	0.0317460	0.0476190
β−HCH	–	NA	1.8	C	-	0.0005718	0.1111111	0.1666667
Lindane	5.0	0.3	1.8	NA	0.00077	0.0005718	0.1111111	0.1666667
p,p′-DDE	–	NA	340	B2	-	0.0000030	0.0005882	0.0008824
p,p′-DDD	–	NA	240	B2	-	0.0000043	0.0008333	0.0012500
p,p′-DDT	–	0.5	340	B2	0.003	0.0000030	0.0005882	0.0008824
Σ−DDT	10.0[c]	–	–	–	–	–	–	–

NA = Not Assessed under the IRIS program.
B2 = Probable human carcinogen – based on sufficient evidence of carcinogenicity in animals.
C = Possible human carcinogen.
D = Not classifiable as to human carcinogenicity.
*WOE = weight of evidence of carcinogenicity described by categories from Group A for known human carcinogens through Group E for agents with evidence of noncarcinogenicity under US EPA's Risk Assessment Guidelines (1986) available in http://www.epa.gov.
[a] FAO/WHO, 2009.
[b] US EPA IRIS, 2006.
[c] Σ−DDT = p,p'-DDE + p,p'-DDD + p,p'-DDT.

meat, information concerning dietary intake from this commodity is rather scarce. Darnerud et al. (2006) in a Swedish Market basket study estimated an intake of 0.0828 μg/kg bw/d for Σ−DDT and 0.00949 μg/kg bw/d for Σ−HCH from meat products (beef, pork, lamb, poultry, cured/processed meats) from different cities in Sweden. According to their findings, the Σ−DDT was an important contributor to the total intake of organohalogen contaminants for Swedish consumers through fish, meat, dairy products and fats/oils. Moreover, the levels of Σ−DDTs in meat products showed a geographical trend from Swedish-produced bovines and pigs, with higher levels in samples from southern Sweden probably due to a higher degree of environmental contamination. Recently, Perelló, Gómez-Catalán, Castell, Llobet, and Domingo (2012) estimated the dietary intake of HCB by the population of Catalonia, Spain, and reported that the highest contribution corresponded to meat and meat products (26.4%) with a HCB intake of 9.97 ng/day.

3.2.2. Risk assessment

The OCPs investigated in the present study have carcinogenic and non-carcinogenic effects (ATSDR, 2005). As the ADI formulated by WHO did not take into consideration other factors such as dietary habits and processing effects, the potential non-carcinogenic risk to human health was estimated by multiplying the average daily exposure by the exposure frequency of 3 times a week for meat consumption and once a month for liver, kidney and spinal marrow (Flores-Huerta, Acosta-Cázares, Rendón-Macías, Klünder-Klünder, & Gutiérrez-Trujillo, 2006). In addition, a processing factor was applied of 55% and 40% for Lindane and p,p'-DDT reported by Sallam et al. (2008) as a cooking loss due to boiling, since lipids and lipophilic compounds are partially removed during cooking and processing. Furthermore, such types of meat are always cooked before consumption in México. The hazard ratios (HR) for non-cancer and cancer risk are presented in Table 6. The HRs of potential carcinogenic risk to human health were estimated by multiplying the average daily exposure by the same exposure frequency and processing factors employed in non-cancer risk assessment, and assuming a body weight of 60 kg.

According to Table 6 the HRs for non-cancer risk of several OCPs present in muscle samples to the general population in México were higher than the target value of 1.0, indicating that the average exposure levels to these pesticides exceeds the Reference Dose for non-cancer health effects and would have a potential health impact. During the rainy season the highest non-cancer HRs estimated corresponded to γ−HCH (3.97) detected in muscle samples from PN, and Σ−DDT (4.39) detected in muscle samples from PN. During the dry season the non-cancer HR for γ−HCH (0.38) was lower than one, suggesting that dietary exposure to this pesticide due to the consumption of meat would have no adverse effects. Nevertheless, non-cancer HR for Σ−DDT (1.90) detected in muscle samples from MFA was higher than one, indicating a non-carcinogenic risk for consumers. The non-cancer HRs for OCPs detected in liver, kidney and spinal marrow samples were below 1.0 due to a lower daily exposure and consumption frequency, indicating that daily intakes never exceeded the exposure limit recommendations.

Cancer benchmarks of exposure to OCPs for daily consumption of bovine muscle and organs to the general population in México are summarized in Table 7.

Comparing the cancer benchmark concentrations (BMC) derived from this study with those derived by US EPA, several BMC for the Mexican population were higher than US EPA reference values. In the case of Lindane BMCs for liver and kidney/spinal marrow samples were 144 and 217 times the reference value. These differences could be due the higher liver and kidney/spinal marrow consumption rates in México. When exposure levels were compared with BMCs to estimate HRs, the cancer risks associated with bovine muscle and organ consumption based on the 50th and 95th centile concentrations were greater than one, indicating a potential health risk. As shown in Table 6, the highest HRs of cancer risk to the 50th centile daily consumption through meat corresponded to p,p'-DDT from PN (7.26E + 05) and p,p′-DDT from ALV (4.49E + 05) during the rainy season, and during the dry season to p,p′-DDT from MFA and ALV (3.15E + 05, 2.05E + 05). In particular, the HRs of cancer risk to the 50th centile daily consumption through meat from PO for β− and γ−HCH (3.94E + 03, 7.90E + 03) during the rainy season were 1–40-fold higher than HRs estimated for their corresponding exposures during the dry season. In reference to the HRs of cancer risk to the 95th centile daily consumption through meat shown in Table 6, the highest corresponded to p,p′-DDT from ALV (7.76E + 06) and p,p′-DDE from PO (2.78E + 06) during the dry season. The HRs of cancer risk to the 95th centile daily consumption through meat from PO for γ−HCH (1.50E + 05) during the rainy season were 1–45-fold higher than HRs estimated for exposures during the dry season, except for α−HCH (7.21E + 03) from PO during the dry season. As shown in Fig. 4, the highest HRs of cancer risk to the 95th centile daily consumption of p,p′-DDT corresponded to spinal marrow and liver from ALV (5.4 and 5.0, respectively) during the rainy season and

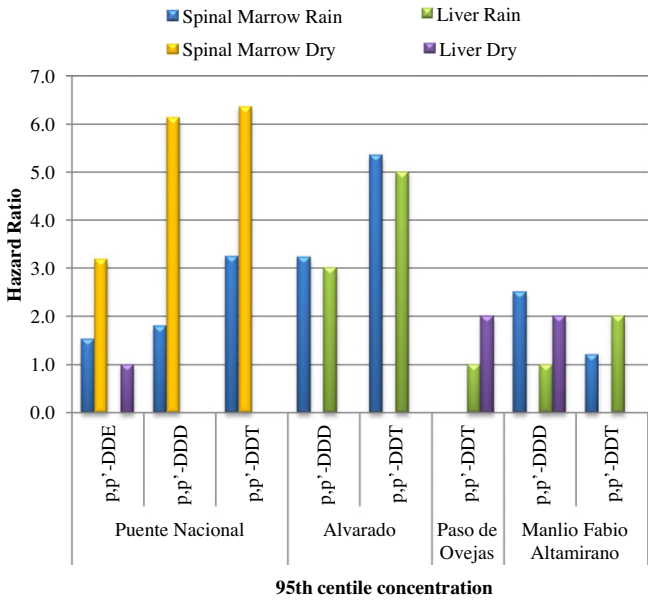


Fig. 4. Hazard ratios of potential cancer risk for OCPs by daily spinal marrow consumption of 50th centile concentration and by daily liver and spinal marrow consumption of 95th centile concentration during dry and rainy seasons.

to spinal marrow from PN during the dry season (6.4). In reference to p,p′-DDD, the highest HRs of cancer risk to the 95th centile daily consumption were observed during the dry season through spinal marrow from PN (6.4), suggesting that the average daily exposure to these pesticides due to meat, spinal marrow and liver consumption would have a potential health impact of lifetime cancer risk greater than one in one million. However, consumption of liver and spinal marrow by the average population is less frequent when compared with meat consumption.

From these findings, there is evidence that a higher risk from exposure will result for heavy meat consumers, particularly in sectors of the population whose major source of protein is from meat and meat products, as in the northern part of the country. Moreover, these results lead to different implications for different geographical regions and seasons, resulting in substantial differences in health risks for meat and organ consumption from bovines produced during the rainy season. Furthermore, to improve risk management in the field of food safety, appropriate management and control of toxic substances in animal production are crucial. However, the economic consequences to commercial beef producers can be significant because residue concentrations are near to or higher than the guidelines in national and international regulations. Therefore, a systematic monitoring program should be established to provide consistent regulatory decisions.Efforts and strategies to enforce the policies of restriction of use and release of these pesticides into the environment must become more intense.

Prolonged consumption of these toxic pesticides through meat and other tissues poses serious implications to the health of man and animals. As previously explained, studies have shown a significant association between the levels of these pesticides in fat and increased cancer incidence and endocrine disruption. Moreover, as lifetime human exposure to OCPs occurs mainly through the diet, more research is needed on the long-term potential effects of chronic exposure to these persistent pesticides at low levels. In particular, neurodevelopment and diabetes effects need to be studied because although the use of these compounds has been banned or restricted in México, residues of OCPs have been detected in human breast milk (Pardío et al., 1998). In addition, livestock producers are responsible for ensuring that their animals do not have unacceptable levels of pesticide residues. For meat produced within a country, this can be achieved by observing relevant regulatory withholding periods for crops and animals using data derived from monitoring programs.

According to this evidence, a better characterization of food consumption is needed to reduce the uncertainty associated with the consumption data. This could include the generation of a dietary database that records the quantity consumed and the dietary habits of consumers by country regions. Then, with accurate data from market basket database, coupled with that generated from epidemiological studies, a more accurate health risk assessment of OCPs exposure would be conducted for different types of foods and contaminant concentrations, providing a realistic risk assessment to humans.

4. Conclusions

Beef animals from the central agrarian area of Veracruz, México investigated showed a positive relationship between the highest mean levels of α−HCH, β−HCH, δ−HCH, Σ−HCH to lung, α−HCH and β−HCH to liver and kidney, and γ−HCH to muscle tissues during the rainy season, to the PO location and rainy season. δ−HCH was found in 100% of the samples analysed. Mean residues of DDT metabolites were higher during the dry season, except for p,p′-DDT that showed higher levels during the rainy season. 4,4′-DDE, 4,4′-DDD, o,p′-DDT, and Σ−DDT were found in lung tissues during the dry season from PO, the exception being 4,4′-DDT whose highest mean levels were found in PO during the rainy season. Results indicated that significant differences in contamination residues were related to locations, tissues and seasons, suggesting that levels and distribution of DDT and HCHs vary with season in these tropical areas, that the on-going usage of Lindane and/or technical HCH and the past use of DDT in this area in malaria control programs, livestock management, the influences of regional climate, and physico-chemical properties all have significant effects on pesticide accumulation by beef animals.

The current estimated intakes are higher than the recommended ADIs. Estimated dietary intakes through consumption of muscle tissues from PO during the rainy season showed the highest contributions for γ−HCH and Σ−DDT, at 0.7 and 0.12 times the recommended ADIs. Comparing the estimated daily intakes of HCH and DDT through muscle consumption from 1996 and the results of the present study, there is evidence that the γ−HCH estimated intakes increased 3 times and and Σ−DDT intake decreased 95.8%. These findings show the consequence of the ongoing usage of large amounts of Lindane and/or technical HCH and the impact of the ban on DDT in malaria control programs. Comparing these estimated dietary intakes of Σ−DDT and HCH through consumption of meat, as published in recent studies, dietary exposures to these OCPs estimated in the present study are higher. Taking into account international guidelines, the current dietary intake of meat by the Mexican population poses health risks. As a consequence, consumers of meat produced in this agrarian zone of Veracruz are exposed to dietary levels of OCPs of at least 10 to 55-fold higher than the exposure levels in developed countries.

Considering the increase in meat consumption and the potential risk associated with estimated exposures of both 50th and 95th centile HRs that were greater than unity, appropriate management strategies should become a priority in the near future due to the accumulation of OCPs in the food chain and the consequent population exposure through feed and food consumption. The results from this study indicate that the organochlorine pesticides DDTs and Lindane are of particular concern, because of their longer use in the country.

Even though this investigation did not consider differences in risk associated with socioeconomic, age and sex factors, the assessment indicates a high cancer risk due to the contamination levels of these pesticides in meat, liver and spinal marrow of bovines produced in different geographical locations and seasons. This study represents an important step towards a more comprehensive understanding of the human health risks associated with OCPs exposure via meat consumption in México and the region.

Acknowledgments

This work was supported by research grants from PROMEP 103.5/03/477 UVER-F-11. We thank MVZ. María Esther Muñoz Pérez who made the arrangements for collecting the samples.

References

Agency for Toxic Substances and Disease Registry (ATSDR). (2005). Toxicological Profiles of Toxic Substances. US Department of Health and Human Services, Public Health Service, Atlanta, GA. Available at <http:/www.atsdr.cdc.gov>.

Ahmad, R., Salem, N. M., & Estaitieh, H. (2010). Occurrence of organochlorine pesticide residues in eggs, chicken and meat in Jordan. *Chemosphere, 78*, 667–671.

Alcover-Vidal, V. M., Castellanos-Ruelas, A. F., Herrera-Chalé, F., Chel-Guerrero, L., & Betancur Ancona, A. (2007). Detection of heavy metals and DDT in the muscles and organs of cattle in Yucatán, México. *Técnica Pecuaria México, 45*(2), 237–247.

Amaral-Mendes, J. J. (2002). The endocrine disrupters: A major medical challenge. *Food and Chemical Toxicology, 40*, 781–788.

AOAC International. (1998).In: P. Cunnif (Ed.), Official Methods of Analisis of AOAC Internacional (16th ed.) (pp. 3–4). Gaithersburg, USA.

Barkatina, E. N., Pertsovsky, A. L., Murokh, V. I., Kolomiets, N. D., Shulyakovskaya, O. V., Venger, O. N., et al. (1999). Organochlorine pesticide residues in basic food products and diets in the Republic of Belarus. *Bulletin of Environmental Contamination and Toxicology, 63*, 235–242.

Baumgard, L. H., Wheelock, J. B., O'Brien, M., Shwartz, G., Zimbelman, R.B., Sanders, S.R., VanBaale, M.J., Collier, R.J., Rhoads, M.L., and Rhoads, R.P. (2007). The differential effects of heat stress vs. underfeeding on production and post-absorptive nutrient partitioning. In 22nd Annual Southwest Nutrition and Management Conference Tempe, AZ, pp. 116–124.

Beard, J. (2006). DDT and human health. *Science of the Total Environment, 355*(1–3), 78–89.

Bidleman, T. F., & Leone, A. D. (2004). Soil-air exchange of organochlorine pesticides in the Southern United States. *Environmental Pollution, 128*(1–2), 49–57.

Bolt, H. M., & Degen, G. H. (2002). Comparative assessment of endocrine modulators with estrogenic activity: II. Persistent organochlorine pollutants. *Archives of Toxicology, 76*, 187–193.

Casas, M., Torres, J. L., Bown, D. N., Rodríguez, M. H., & Arredondo, J. I. (1998). Selective and conventional house-spraying of DDT and Bendiocarb against *Anopheles pseudopuntipennis* in southern Mexico. *Journal of American Mosquito Control Association, 14*, 410–420.

Comisión Intersecretarial para el Control del Proceso y Uso de Plaguicidas, Fertilizantes y Sustancias Tóxicas (CICOPLAFEST). Catálogo de Plaguicidas. (2004). Available at <http:/www.cofepris.gob.mx>.

Conference of the Parties to the Stockholm Convention (COP-4) Fourth Meeting. (2009). A Reporting Service for Environment and Development Negotiations, Earth Negotiations Bulletin. International Institute for Environment and Development Negotiations, vol. 15 (169). <http://www.iisda.ca/chemical/pops/cop4/<.

Covaci, A., de Boer, J., Ryan, J. J., Voorspoels, S., & Schepens, P. (2002). Distribution of organobrominated and organochlorinated contaminants in Belgian human adipose tissue. *Environmental Research, 88*(3), 210–218.

Covaci, A., Gheorghe, A., & Schepens, P. (2004). Distribution of organochlorine pesticides, polychlorinated biphenyls and α−HCH enantiomers in pork tissues. *Chemosphere, 56*, 757–766.

Cox, S., Niskar, A., Narayan, V., & Marcus, M. (2007). Prevalence of self-reported diabetes and exposure to organochlorine pesticides among Mexican Americans: Hispanic health and nutrition examination survey, 1982–1984. *Environmental Health Perspectives, 115*(12), 1747–1752.

Cruz, S., Lino, C., & Silveira, M. I. (2003). Evaluation of organochlorine pesticide residues in human serum from an urban and two rural populations in Portugal. *Science of the Total Environment, 317*, 23–35.

Darko, G., & Acquaah, S. (2007). Levels of organochlorine pesticides residues in meat. *International Journal of Environmetal Science and Technology, 4*(4), 521–524.

Darnerud, P. O., Atuma, S., Aune, M., Bjerselius, R., Glynn, A., Petersson Grawe, K., et al. (2006). Dietary intake estimations of organohalogen contaminants (dioxins, PCB, PBDE and chlorinated pesticides, e.g. DDT) based on Swedish market basket data. *Food and Chemical Toxicology, 44*, 1597–1606.

Dougherty, Ch. P., Henricks Holtz, Reinert, J. C., Panyacosit, L., Axelrad, D. A., & Woodruff, T. J. (2000). Dietary exposures to food contaminants across the United States. *Environmental Research Section A, 84*, 170–185.

Dreiher, J., & Kordysh, E. (2006). Non-Hodgkin Lymphoma and pesticide exposure: 25 years of research. *Acta Haematologica, 116*(3), 153–164.

Dugald, J., MacLachlana, M., & Rajumati, B. (2009). Transfer of lipid-soluble pesticides from contaminated feed to livestock, and residue management. *Animal Feed Science and Technology, 149*, 307–321.

European Union Pesticide Data Base. (2009). Commision of the European Communities Legislation. Available at <http://ec.europa.eu/sanco_pesticides/public/index.cfm.

Eskenazi, B., Marks, A. R., Bradman, A., Fenster, L., Johnson, C., Barr, D. B., et al. (2006). In utero exposure to dichlorodiphenyltrichloroethane (DDT) and dichlorodiphenyldichloroethylene (DDE) and neurodevelopment among young Mexican American children. *Pediatrics, 118*(1), 233–241.

Flores-Huerta, S., Acosta-Cázares, B., Rendón-Macías, M. E., Klünder-Klünder, M., & Gutiérrez-Trujillo, G. (2006). Consumo de alimentos saludables o con riesgo para la salud. *Revista Médica del Instituto Mexicano del Seguro Social, 44*(Suppl. 1), S63–S78.

Food and Agriculture Organization and World Health Organization (FAO/WHO). (1997). Guidelines for Predicting Dietary Intake of Pesticide Residues (revised). Global Environment Monitoring System – Food Contamination Monitoring and Assessment Programme (GEMS/Food) in collaboration with the Codex Committee on Pesticide Residues WHO/FSF/FOS/97.7. Rome, Italy. Available at <http://who.int.

Food and Agriculture Organization and World Health Organization (FAO/WHO). (2008a). Dietary exposure assessment of chemicals in food. Report of a Joint FAO/WHO consultation, 2005.

Food and Agriculture Organization and World Health Organization (FAO/WHO). (2008b). CODEX Alimentarius, Pesticide residues in food. Available at <http://www.codexalimentarius.net.

Food and Agriculture Organization and World Health Organization (FAO/WHO). (2009). Pesticide residues in food. Joint FAO/WHO Meeting on Pesticide Residues. Report of the Joint Meeting of the FAO Panel of Experts on Pesticide Residues in Food and the Environment and the WHO Core Assessment Group on Pesticide Residues. Rome:World Health Organization, Food and Agriculture Organization of the United Nations.

Fries, G. R., Marrow, G. S., & Gordon, C. H. (1969). Metabolism of o, p′-DDT and p, p′-DDT by rumen microorganism. *Journal of Agricultural and Food Chemistry, 17*, 860–862.

Fries, G. F., & Faustenbach, D. J. (1990). Evaluation of potential transmission of of 2, 3, 7, 8-tetrachlorodibenzo-p-dioxin-contaminated incinerator emissions to humans via foods. *Journal of Toxicology and Environmental Health, 29*, 1–43.

Glynn, A., Wernroth, L., Atuma, S., Linder, C., Aune, M., Nilsson, I., et al. (2000). PCB and chlorinated pesticide concentrations in swine and bovine adipose tissue in Sweden 1991–1997: Spatial and temporal trends. *Science of the Total Environment, 246*, 195–206.

Glynn, A., Aune, M., Nilsson, I., Darnerud, P., Halldin, E., Bignert, A., et al. (2009). Declining levels of PCB, HCB and p, p′-DDE in adipose tissue from food producing bovines and swine in Sweden 1991–2004. *Chemosphere, 74*, 1457–1462.

Haider, K., & Jagnow, G. (1975). Abbau von 14C-, 3H-, und 36Cl-markiertem γ−Hexachlorcyclohexan durch anaerobe Bodenmikroorganismen. *Archives of Microbiology, 104*, 113–121.

Hashemy-Tonkabony, S. E., Afshar, A., Ghazisaidi, K., Assadi-Langaroodi, F., Messchi, M., & Ahmadi, Z. (1981). Chlorinated pesticide residues in meat and fat from animals raised in Iran. *Journal of American Oil Chemists' Society, 58*(2), 89–91.

Hernández-Lara, G. (2003). Monitoreo ambiental de DDT, HCH y sus metabolitos a través de pasto y leche bajo condiciones de trópico húmedo. Bachelor's Degree Thesis. Facultad de Ingeniería Química. Universidad Veracruzana.

Instituto Nacional de Ecología (INE). (2002). Secretaría de Medio Ambiente y Recursos Naturales. Available at <http://www.ine.gob.mx.

Instituto Nacional de Estadística, Geografía e Informática (INEGI). (2008). Available at <http://www.inegi.gob.mx.

Instituto Nacional de Salud Pública (INSP). (2007). Resultados de Nutrición de la Encuesta Nacional de Salud y Nutrición 2006. Secretaría de Salud. Available at <http://www.insp.mx/ensanut/.

International Agency for Research on Cancer (IARC). (2008). Overall Evaluations of Carcinogenicity to Humans: Update 2008. Available at <http://monographs.iarc.fr/ENG/Classification/crthallcas. php>.

Jacobson, M. (1991). *Developmental neurobiology*. New York: Plenum Press, pp. 95–142.

Jagnow, G., Haider, K., & Ellwardt, P. C. (1977). Anaerobic dechlorination and degradation of Hexachlorocyclohexane isomers by anaerobic and facultative anaerobic bacteria. *Archives of Microbiology, 115*, 285–292.

Jandacek, R. J., & Tso, P. (2001). Factors affecting the storage and excretion of toxic lipophilic xenobiotics. *Lipids, 36*(12), 1289–1305.

Jandacek, R. J., Anderson, N., Liu, M., Zheng, S., Yang, Q., & Tso, P. (2005). Effects of yo-yo diet, caloric restriction, and olestra on tissue distribution of hexachlorobenzene. *American Journal of Physiology and Gastrointestinal Liver Physiology, 288*, G292–G299.

Jensen, A. A. (1983). Chemical contaminants in human milk. *Residue Reviews, 89*, 2–128.

Jevsnik, M., Cerkvenik, V., & Doganoc, D. (2004). Evidence of organochlorine pesticide and polychlorinated biphenyl residues in Slovenian poultry tissues from 1997 to 1999. *Journal of Food Protection, 67*(10), 2326–2331.

Jiang, Q. T., Lee, T. K. M., Chen, K., Wong, H. L., Zheng, J. S., Giesy, J. P., et al. (2005). Human health risk assessment of organochlorines associated with fish consumption in a coastal city in China. *Environmental Pollution, 136*, 155–165.

Kacew, S., Singhal, R., Hrdina, P., & Ling, M. G. (1972). Changes in kidney cortes gluconeogenic enzymes induces by 1,1,1-trichloro-2,2-bis-(p-chlorophenyl) ethane (DDT). *Journal of Pharmacology and Experimental Therapeutics, 181*(2), 234–243.

Kalantzi, O. I., Alcock, R. E., Johnston, P. A., Santillo, D., Stringer, R. L., Thomas, G. O., et al. (2001). The global distribution of PCBs and organchlorine pesticides in butter. *Environmental Science and Technology, 35*, 1013–1018.

Kavlock, R. J. (1996). Research needs for the risk assessment of health and environmental effects of the US EPA-sponsored workshop. *Environmental Health Perspectives, 104*, 714–740.

Kelce, W. R., Stone, C. R., Laws, S. C., Gray, L. E., Kemppainen, J. A., & Wilson, E. M. (1995). Persistent DDT metabolite p, p′-DDE is a potent androgen receptor antagonist. *Nature, 375*, 581–585.

Kelce, W. R., Gray, L. E., & Wilson, E. M. (1998). Antiandrogens as environmental disruptors. *Reproduction and Fertility Development, 10*, 105–111.

Krauthacker, B., Romanic, S. H., & Reiner, E. (2001). Polychlorinated Biphenyls and organochlorine pesticides in vegetation samples collected in Croatia. *Bulletin of Environmental Contamination and Toxicology, 66*(3), 334–341.

Lemaire, G., Terouanne, B., Mauvais, P., Michel, S., & Rahmani, R. (2004). Effect of organochlorine pesticides on human androgen receptor activation in vitro. *Toxicology and Applied Pharmacology, 196*, 235–246.

Lorber, M., Cleverly, D., Schaum, J., Phillips, L., Schweer, G., & Leighton, T. (1994). Development and validation of an air-to-beef food chain model for dioxin-like compounds. *Science of the Total Environment, 156*, 39–65.

Manirakiza, P., Akimbamijo, O., Covaci, A., Adediran, S. A., Cisse, I., Fallc, S. T., et al. (2002). Persistent chlorinated pesticides in fish and cattle fat and their implications for human serum concentrations from the Sene-Gambian region. *Journal of Environmental Monitoring, 4*, 609–617.

Manríquez-Mendoza, L. Y. (2004). Biotransferencia de Hexaclorociclohexano y sus metabolitos α−, β−, γ− y δ− en el aire, pasto y su bioacumulación en sangre y leche de bovinos en trópico húmedo. Master Thesis. Facultad de Medicina Veterinaria y Zootecnia. Universidad Veracruzana.

MacLachlan, M. (1996). Bioaccumulation of hydrophobic chemicals in agricultural food chains. *Environmental Science and Technology, 30*, 252–259.

Meeker, J. D., Altshul, L., & Hauser, R. (2007). Serum PCBs, p, p′-DDE and HCB predict thyroid hormone levels in men. *Environmental Research, 104*(2), 296–304.

Müllerová, D., & Kopecký, J. (2007). White adipose tissue: Storage and effector site for environmental pollutants. *Physiological Research, 56*, 375–381.

Murphy, P. G. (1972). Sulfuric acid cleanyp of animal tissues for analysis of acid-stable chlorinated hydrocarbon residues. *Journal of the AOAC, 55*(6), 1360–1362.

Mussalo-Rauhamaa, H. (1991). Partitioning and levels of neutral organochlorine compounds in human serum, blood cells, and adipose and liver tissue. *Science of the Total Environment, 103*, 159–175.

Nagabe, B., & Bidleman, T. F. (2006). DDT Concentrations in Soils of Brazzaville, Congo. *Bulletin of Environmental Contamination and Toxicology, 76*, 697–704.

Newland, L. W., Chester, G., & Lee, G. B. (1969). Degradation of γ−BHC in simulated lake impoundments as affected by aeration. *Journal of Water Pollution, 41*, R174–183.

Nuttall, N. (2009). UNEP Spokesperson/Head of Media. United Nations Environment Programme. Available at <www.unep.org>.

Oehme, M., Haugen, J.-E., & Schlabach, M. (1996). Seasonal changes and relations between levels of organochlorines in Artic Ambient air. First results of an all-year-round monitoring program at Ny-Alesun, Svalbard, Norway. *Environmental Science and Technology, 30*, 2294–2304.

Osibanjo, O., & Adeyeye, A. (1997). Organochlorine pesticide residues in foodstuffs of animal origin in Nigeria. *Bulletin of Environmental Contamination and Toxicology, 58*, 206–212.

Pardío, V. T., Waliszewski, S. M., Aguirre, A. A., Coronel, H., Burelo, G. V., Infanzón, R. M., et al. (1998). DDT and its metabolites in human milk collected in Veracruz City and suburban areas (Mexico). *Bulletin of Environmental Contamination and Toxicology, 60*, 852–857.

Pardío, V. T., Waliszewski, K. N., Landín, L., & Bautista, R. G. (2003). Organochlorine pesticide residues in cow's milk from a tropical region of Mexico. *Food Additives and Contaminants, 20*(3), 259–269.

Perelló, G., Gómez-Catalán, J., Castell, V., Llobet, J. M., & Domingo, J. L. (2012). Estimation of the daily intake of hexachlorobenzene from food consumption by the population of Catalonia, Spain: Health risks. *Food Control, 23*, 198–202.

Quintana, P., Delfino, R., Korrick, S., Ziogas, A., Kutz, F., Jones, E., et al. (2004). Adipose tissue levels of organochlorine pesticides and polychlorinated biphenyls and risk of Non-Hodgkin's Lymphoma. *Environmental Health Perspectives, 112*(8), 854–861.

Rosenbaum, R., Mckone, T., & Jolliet, O. (2009). CKow: A dynamic model for chemical transfer to meat and milk. *Environmental Science and Technology, 43*, 8191–8198.

Sallam, K. I., Mohammed, A. E., & Morshedy, A. (2008). Organochlorine pesticide residues in camel, cattle and sheep carcasses slaughtered in Sharkia Province, Egypt. *Food Chemistry, 108*, 154–164.

Sang, S., Petrovic, S., and Cuddeford, V. (1999). Lindane: A review of toxicity and environmental fate. Wildlife Toxicology Program. Toronto: World Wildlife Fund.

Schecter, A., Cramer, P., Boggess, K., Stanley, J., & Olson, J. R. (2001). Levels of Dioxins, dibenzofurans, PCB and DDE congeners in polled food samples collected in 1995 at supermarkets across the United States. *Chemosphere, 34*, 1437–1447.

Secretaría de Agricultura, Ganadería, Desarrollo Rural, Pesca y Alimentación (SAGARPA). (2001). Modificación a la Norma Oficial Mexicana NOM-004-ZOO-1994, grasa hígado, músculo y riñón en aves, bovinos, caprinos, cérvidos, equinos, ovinos y porcinos. Residuos Tóxicos. Límites Máximos Permisibles y procedimientos de muestreo. Available at <www.sagarpa.gob.mx>.

Secretaría de Agricultura, Ganadería, Desarrollo Rural, Pesca y Alimentación (SAGARPA). (2008). Sistema de Información de Agricultura y Pesca. Available at <www.siap.sagarpa.gob.mx>.

Secretaría de Salubridad y Asistencia (SSA). (2006). Dirección General de Medicina Preventiva. México.

Semeena, V. S., Feichter, J., & Lammel, G. (2005). Impact of the regional climate and substance properties on the fate and atmospheric long-range transport of persistent organic pollutants – examples of DDT and γ−HCH. *Atmospheric Chemistry and Physics, 5*, 12569–12615.

Sharpe, R. T., & Livesey, C. T. (2005). Surveillance of suspect animal toxicoses with potential food safety implications in England and Wales between 1990 and 2002. *Veterinary Record, 157*, 465–469.

Siddiqui, M. K., Nigam, U., Kaul, P. P., & Seth, T. D. (1996). Bioaccumulation of HCH isomers in different tissues of young and old rats: A comparison. *Bulletin of Environmental Contamination and Toxicology, 56*, 896–902.

Snedeker, S. M. (2001). Pesticides and breast cancer risk: a review of DDT, DDE, and dieldrin. *Environmental Health Perspectives, 109*(Suppl 1), 35–47.

Sonnenschein, C., & Soto, A. M. (1998). An updated review of environmental estrogen and androgen mimics and antagonists. *Journal of Steroid Biochemistry and Molecular Biology, 65*, 143–150.

Srinivasan, K., & Radhakrishnamurty, R. (1983). Studies on the distribution of β− and γ−isomers of hexachlorocyclohexane in rat tissues. *Journal of Environmental Science and Health B, 18*, 401–418.

Su, G. C. C. (1998). A comparison of statistical and empirical detection limits. *Journal of AOAC International, 81*, 105–110.

Tebourbi, O., Driss, M., Sakly, M., & Ben Rhouma, K. (2006). Metabolism of DDT in different tissues of young rats. *Journal of Environmental Science and Health Part B, 41*, 167–176.

Tilbury, K. L., Stein, J. E., Meador, J. P., Krone, C. A., & Chan, S. L. (1997). Chemical contaminants in harbor porpoise (*Phocoena phocoena*) from the North Atlantic coast: tissue concentrations and intra- and inter-organ distribution. *Chemosphere, 34*, 2159–2181.

United Nations Environment Programme (UNEP). (1988). Determination of DDTs and PCBs by capillary gas chromatography and electron capture detection. Reference Methods for Marine Pollution Studies No. 40.

United States Environmental Protection Agency (US EPA). (2009). Available at <www.epa.gov>.

van Loon, L. J. C., Manders, R. J. F., Koopman, R., Kaastra, B., Stegen, J. H. C. H., Gijsen, A. P., et al. (2005). Inhibition of adipose tissue lipolysis increases intramuscular lipid use in type 2 diabetic patients. *Diabetologia, 48*, 2097–2107.

Vieira, E. D. R., Torres, J. P. M., & Malm, O. (2001). DDT Environmental persistence from its use in a vector control program: A case study. *Environmental Research Section A, 86*, 174–182.

Waliszewski, S. M., Pardío, V. T., Chantiri, J. N., Infanzón, R., & Rivera, J. (1996a). Organochlorine pesticide residues in adipose tissue of Mexicans. *Science of the Total Environment, 181*(2), 125–131.

Waliszewski, S. M., Pardío, V. T., & Waliszewski, K. N. (1996b). Organochlorine pesticide levels in bovine meat. *Fresenius Environmental Bulletin, 5*, 369–373.

Walker, K. (2009). Cost-comparison of DDT and alternative insecticides for malaria control. *Veterinary Medicine and Entomology, 14*, 345–354.

Walker, K., Vallero, D., & Lewis, R. (1999). Factors influencing the distribution of Lindane and other Hexachlorocyclohexanes in the environment. *Environmental Science and Technology, 33*(4), 4373–4378.

Wan, M. T., Kuo, J., & Pasternak, J. (2005). Residues of Endosulfan and other selected organochlorine pesticides in farm areas of the lower Fraser Valley, British Columbia, Canada. *Journal of Environmental Quality, 34*, 1186–1193.

Wania, F. (1999). On the origin of elevated levels of persistent chemicals in the environment. *Environmental Science Pollution, 6*, 11–19.

Wandiga, S. O. (2001). Use and distribution of organochlorine pesticides. The future in Africa. *Pure and Applied Chemistry, 73*(7), 1147–1155.

Willett, L., O'Donnell, A. F., Durst, H., & Kurz, M. M. (1993). Mechanisms of movement of organochlorine pesticides from soils to cows via forages. *Journal of Dairy Science, 76*, 1635–1644.

Willett, K., Ulrich, E., & Hites, R. (1998). Differencial toxicity and environmental fates of Hexachlorocyclohexane isomers. *Environmental Science and Technology, 32*, 2197–2207.

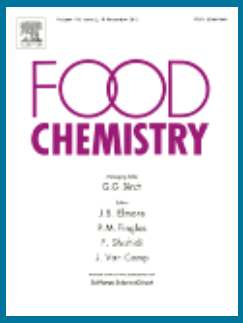
Wisniewski, J. A., Moody, D. E., Hammock, B. D., & Shull, L. R. (1987). Enzyme activities in cattle, goats and sheep. Interlobular distribution of hepatic xenobiotic-metabolizing. *Journal of Animal Science, 64*, 210–215.

Witorsch, R. J. (2002). Low-dose in utero effects of xenoestrogens in mice and their relevance to humans: an analytical review of the literature. *Food and Chemical Toxicology, 40*, 905–913.

Wong, F., Alegria, H. A., Bidleman, T. F., Alvarado, V., Angeles, F., Avila, A., et al. (2009). Passive air sampling of organochlorine pesticides in Mexico. *Environmental Science and Technology, 43*(3), 704–710.

World Health Organization (WHO). (2003). Food Safety Issues GEMS/Food Regional Diets. Regional per capita consumption of raw and semi-processed agricultural commodities prepared by the Global Environment Monitoring System/Food Contamination Monitoring and Assessment Programme (GEMS/Food). Food Safety Department WHO Geneva, Switzerland.

Zumbado, M., Goethals, M., Álvarez León, E., Luzardo, O., Cabrera, F., Serra, MajemL, et al. (2005). Inadvertent exposure to organochlorine pesticides DDT and derivatives in people from the Canary Islands (Spain). *Science of the Total Environment, 339*, 49–62.



Food Chemistry

Food Chemistry publishes original research papers dealing with the chemistry and biochemistry of foods and raw materials covering the entire food chain from `farm to fork.'

Topics include:

– Chemistry...

View full aims and scope

Managing Editor: G.G. Birch

View full editorial board

- Guide for Authors
- Submit Your Paper
- Track Your Paper
- Order Journal
- View Articles

Impact Factor: 3.655

5-Year Impact Factor: 4.268

Imprint: ELSEVIER

ISSN: 0308-8146

Journal News

Food Chemistry Accepted in PubMed

View All

Recent Articles

ScienceDirect

Development of a novel micro-assay for evaluation of peroxyl radical scavenger capacity: Application to carotenoids and structure–activity relationship

Eliseu Rodrigues | Lilian R.B. Mariutti | ...

Crude caffeine reduces memory impairment and amyloid β1–42 levels in an Alzheimer's mouse model

Yi-Fang Chu | Wen-Han Chang | ...

Effects of pH and ionic strength of NaCl on the stability of diacetyl and (−)-α-pinene in oil-in-water emulsions formed with food-grade emulsifiers

Grażyna Bortnowska

View All

Most Cited Articles

Scopus

Antioxidant activity and phenolic compounds in 32 selected herbs

Wojdyło, A. | Oszmiański, J. | ...

Call for Papers

Special issue of Food Research International on Tea – from bushes to mugs: composition, stability and health aspects

View All

Most Downloaded Articles

ScienceDirect

1. DPPH antioxidant assay revisited

Om P. Sharma | Tej K. Bhat

2. A critical review of methods for characterisation of polyphenolic compounds in fruits and vegetables

Ioana Ignat | Irina Volf | ...

3. Stevia rebaudiana Bertoni, source of a high-potency natural sweetener: A comprehensive review on the biochemical, nutritional and functional aspects

Roberto Lemus-Mondaca | Antonio Vega-Gálvez | ...

View All

Special Issues

Citrus flavonoids: Molecular structure, biological activity and nutritional properties: A review

Tripoli, E. | Guardia, M.L. | ...

A review: Current analytical methods for the determination of biogenic amines in foods

Önal, A.

View All

Advances in Potato Chemistry, Nutrition and Technology

Volume 133, Issue 4 (2012)

Order Now

6th International Conference on Water in Food

Volume 132, Issue 4 (2012)

Order Now

5th Conference on Water in Food

Volume 122, Issue 2 (2010)

Order Now

View All