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Prenatal dichlorodiphenyldichloroethylene (DDE) exposure and child growth during the first year of life

Sheyla Garced^a, Luisa Torres-Sánchez^b, Mariano E Cebrián^c, Luz Claudio^a, and Lizbeth López-Carrillo^b

Sheyla Garced: sgarced@gmail.com; Luisa Torres-Sánchez: ltorress@insp.mx; Mariano E Cebrián: mcebrian@cinvestav.mx; Luz Claudio: luz.claudio@mssm.edu; Lizbeth López-Carrillo: lizbeth@insp.mx

^aDepartment of Preventive Medicine, Mount Sinai School of Medicine. International Training and Research in Environmental and Occupational Health Program. 17 E 102nd Street – CAM Building, 3 West, One Gustave L. Levy Place, Box 1057, New York, NY 10029

^bNational Institute of Public Health, Av. Universidad 655, Col Sta María Ahuacatitlán. CP 62100, Cuernavaca, Morelos, Mexico

^cDepartment of Toxicology, CINVESTAV, Av. Instituto Politécnico Nacional 2508, Col. San Pedro Zacatenco, C.P. 07360 México, D.F. Apartado postal: 14-740, 07000 México, D.F

Abstract

Background—Due to its long-term persistence in the environment and its ability to cross the placental barrier, prenatal p,p'-dichlorodiphenyldichloroethene (DDE) exposure continues to be a public health concern. This study aimed to evaluate the association between prenatal DDE exposure and child growth, at birth and during the first year of life.

Methods—253 pregnant women were recruited between January 2001 and June 2005 in a prospective cohort in Morelos, Mexico. Serum levels of DDE were measured during each trimester of pregnancy by gas chromatography with an electron capture detector. Using generalized mixed effects models, the association between DDE and child growth parameters (weight-for-age, length-for-age, weight-for-length, BMI-for-age, and head circumference-for-age Z-scores) from birth to 1 year of age was assessed. Maternal dietary intake was considered as covariable among others.

Results—DDE levels were 6.3 ± 2.8 ng/mL (first trimester), 6.6 ± 2.9 ng/mL (second trimester), and 7.6 ± 2.9 ng/mL (third trimester). After adjusting for potential confounder variables, no significant associations were observed with prenatal DDE exposure and each of the selected parameters.

Conclusions—Our results show no evidence of an association between prenatal DDE exposure and child growth during the first year of life.

Keywords

p,p'-dichlorodiphenyldichloroethene (DDE); child anthropometric measurements; organochlorines; Z-scores; Mexico

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Correspondence: Dr. Lizbeth López-Carrillo. National Institute of Public Health, Mexico. Av. Universidad 655. Col. Sta María Ahuacatitlán. CP 62100. Cuernavaca Morelos, México. Phone (777) 329-20-00 ext 2501. lizbeth@insp.mx.

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1. Introduction

Organochlorine pesticide dichlorodiphenyltrichloroethane (DDT) was widely used to control malaria before being banned during the 1970s (ATSDR, 2002). DDT is rapidly metabolized to its main metabolite p,p'-dichlorodiphenyldichloroethene (DDE), and because of its chemical stability and lipophilicity, it bioaccumulates in the food chain and persists in the environment years after application (ATSDR, 2002). In Mexico, DDT was used until 1999 and concentrations of DDE are still detectable in populations not occupationally exposed (Torres and López, 2007). DDT is considered an endocrine disruptor due to its antiandrogenic properties (Kelce et al., 1997). During pregnancy, DDT is able to cross the placental barrier (Dorea et al., 2001). Prenatal DDT exposure has been inconsistently associated with intrauterine growth retardation (IUGR), infant neurodevelopment and infant growth (Eskenazi et al., 2009). Hence body burdens of DDT continue to be a public health issue, especially in children.

Contrasting evidence is available regarding the potential effect of DDT exposure on child growth. Some studies have found a negative association between DDE exposure and child anthropometric measurements. For example, a significant decrease in height (0.72 cm at 1 year, 1.14 cm at 4 years, and 2.19 cm at 7 years) was found in children prenatally exposed to high concentrations (≥ 60 ppb), specifically among African-American girls (Ribas-Fitó et al., 2006). Another study by Karmaus and colleagues also reported a significant decrease in height (1.8 cm) among girls, even though DDE concentrations were measured at 8 years of age (Karmaus et al., 2002). In contrast, an increase in height (6.3 cm) and weight-for-height (6.9 kg) has also been documented among exposed boys in a 5-year prospective cohort study (Gladen et al., 2000). A positive association has also been reported between BMI and prenatal DDE exposure which was enhanced among children of smoking mothers (Verhulst et al., 2009). Also an increased head circumference at 5 years of age was associated with prenatal DDE exposure at their highest percentile concentration (8.56 ppm) (Jusko et al., 2006). Other studies have not been able to detect an association between prenatal DDE and height (Gladen et al., 2004), BMI in adolescent males (Gladen et al., 2004) and birth weight or length (Verhulst et al., 2009; Ribas-Fitó et al., 2006; Jusko et al., 2006), including a recent study on height and BMI among Mexican male children during their first year of life (Cupul-Uicab et al., 2010) although they were born in an highly endemic malaria area where average prenatal exposure to DDT (676 ppb) and DDE (4,843 ppb) was high (Koepke et al., 2004).

Potential methodological considerations that may explain the inconsistent results include the lack of standardized anthropometric measurements like z-scores to assess child growth, which enables a more comprehensive interpretation and comparison with international child growth data (Jusko et al., 2006; Verhulst et al., 2009; Cupul-Uicab et al., 2010). Also, all previous studies have failed to take into account some potential key confounders such as maternal nutritional information during pregnancy (Cupul-Uicab et al., 2010; Verhulst et al., 2009; Jusko et al., 2006; Ribas-Fitó et al., 2006; Gladen et al., 2004; Karmaus et al., 2002).

The present study aims to evaluate the association between prenatal DDE exposure and standardized child growth parameters, at birth and during the first year of life, among a cohort of Mexican children. Additionally, we aimed to assess whether this relationship persists after adjusting for maternal nutritional variables.

2. Materials and Methods

2.1. Study Population

Between January 2001 and June 2005, 996 women of reproductive age that were living in the State of Morelos, Mexico participated in a prospective cohort study. At the time of enrollment, eligible criteria included: (1) being at a reproductive age; (2) having the intention of living in one of the selected counties during the upcoming 2 years; (3) not breastfeeding; (4) not using an anticonvulsive drug; (5) no history of renal, hepatic, digestive or thyroid pathology; and (6) not using a permanent contraceptive method. After the first 5 years of follow up, 442 children were born in the cohort. For the purpose of this analysis we selected 253 children from mothers older than 15 years old with no delivery complications. More detailed information on the cohort assembling methods has been published elsewhere (Torres-Sánchez et al., 2007).

The study was approved by the Institutional Review Board of the National Institute of Public Health in Cuernavaca, Mexico. Informed consent was obtained from study participants at baseline and at the onset of the postnatal follow-up.

2.2. Maternal Evaluation

Before and during pregnancy, maternal information on diet, tobacco use, pesticide exposure, reproductive and sociodemographic characteristics were obtained. Body mass index (BMI) at first trimester of pregnancy was calculated based on actual measurements of maternal weight and height.

Dietary information during the first trimester of pregnancy was obtained through a previously validated semiquantitative food frequency questionnaire (Galván-Portillo et al., 2007). The frequency of consumption of 95 items with predetermined portions was classified according to 10 response categories, going from 'never' up to 'six times per day'. Nutrient intake including folate, vitamin B₁₂, calcium, iron and zinc was estimated through a computer program developed by the University of Texas (Food Intake Analysis System [FIAS] 3.0) and used previously in other similar populations (Torres et al., 2006; López-Carrillo et al., 1999). More details on the methodology are available elsewhere (Río García et al., 2009).

2.3. Child Evaluation

Information about weight and length at birth were measurements available in hospital discharge records, while gestational age, and maternal and/or child complications data were obtained face to face from mothers. Because not all children had information on their child's head circumference at birth, this information was not included in the analysis. During all visits (1, 3, 6 and 12 months of age), a trained research technician collected data on the child's anthropometric measurements, including recumbent length (cm), weight (kg), head circumference (cm), and breastfeeding.

2.3. DDT and DDE Determinations

Blood samples (7 mL) were obtained during the baseline interview and/or at each trimester visit. After centrifugation, the serum obtained was stored at -70°C in glass vials (prewashed with pesticide hexane grade) covered with a Teflon cap, until analyzed.

We determined the levels of DDE and p,p'-DDT in serum by means of gas chromatography with an electron capture detector (model 3400; Varian, Inc., Palo Alto, CA, USA), following the protocol recommended by the U.S. Environmental Protection Agency (1980). Concentrations of DDE and p,p'-DDT were reported in wet basis as nanograms per milliliter

(parts per billion). The detection limit was 0.05 ng/mL and 0.0045 ng/mL for DDE and p,p'-DDT, respectively. All (100%) of the serum samples were positive for DDE, whereas 11–22% (depending on the trimester of pregnancy) were positive for p,p'-DDT.

For internal quality control, each of the serum samples was fortified with aldrin and the average recovery was $98.15 \pm 8.8\%$. For every 10 study samples, one sample of bovine serum with known quantities of β -hexachlorocyclohexane (β -HCH), aldrin, hexachlorobenzene (HCB), DDE, and 1,1-dichloro-2,2-bis(pchlorophenyl) ethane (p,p'-DDD) was analyzed, with recovery of 100.8, 100.01, 100.91, 103.4, 104.1%, respectively. Additionally, one randomly selected sample was analyzed in duplicate in each batch with a coefficient of variation of 4.37% and 0.45% for DDE and p,p'-DDT, respectively. The results of the external quality control comparing our laboratory (CINVESTAV) and M. Wolff's laboratory in the Division of Environmental and Occupational Medicine at Mount Sinai Medical School showed a coefficient of Bland-Altman correlation between 10 split serum samples for DDE of 0.98.

2.4. Statistical Analysis

Z-scores were calculated for all anthropometric measurements (weight-for-age, length-for-age, weight-for-length, BMI-for-age and head circumference-for-age) and were standardized using the 2006 World Health Organization (WHO) Standards (WHO, 2006). A Z-score is the distance between the patient's value and the reference population's mean or median value; if the distribution is normal the mean and median values are equal. Positive Z- values are above the 50 percentile and negative Z-values are under the 50 percentile.

To classify an adequate consumption of essential nutrients for Mexican pregnant women during the study period, local cut-off points were taken as reference (Ávila et al., 2002). We considered values under the following levels as a deficient consumption for the following nutrients: 400 μ g/day for folate, 2.0 μ g/day for vitamin B₁₂, 30 mg/day for iron, 15 mg/day for zinc, and 1,200 mg/day for calcium.

To evaluate the association between DDE exposure and child growth parameters, simple and multivariate lineal regression models were calculated using generalized mixed effects models. Separate models were generated for each dependent variable (weight-for-age, length-for-age, weight-for-length, BMI-for-age and head circumference-for-age). Independent variables with fixed effects were: DDE levels at each trimester of pregnancy (on a logarithmic scale), maternal age (years), height (cm), education (years), paid occupation (yes/no), parity (none/1–2), and body mass index during first trimester of pregnancy (kg/m^2). Dietary intake during the first trimester of pregnancy was also assessed for folate (μ g/day), vitamin B₁₂ (μ g/day), calcium (mg/day), iron (mg/day) and zinc (mg/day). In addition, the following variables were considered as covariables: caloric intake of mothers during their first trimester of pregnancy (kcal/day), smoking status (non-smoker, not a passive smoker, passive smoker before and during first trimester of pregnancy, and smoker before and during first trimester of pregnancy), type of birth (vaginal/cesarean), sex of child (female/male), breastfeeding (yes/no) and age at child's evaluation. Random slopes of age at evaluation and age-squared were included in all models to account for accelerated growth as well as the nonlinear changes in group performance with age, and subject ID as random intercept. To facilitate interpretation, the regression coefficients were multiplied by 0.69 and the changes in child growth were estimated for each double increase of DDE.

The models were constructed by backward elimination, and variables that modified by 10% the β coefficients remained in their respective models. To evaluate model fitness, we evaluated residuals normality. All analyses were made using STATA 10.1 (StataCorp., College Station, TX, USA).

3. Results

Table 1 describes selected characteristics of 253 women and children who comprised the total study sample. Almost 9% smoked during pregnancy and another 19% were exposed to secondhand smoking during pregnancy. More than half of the participants were under the recommended intake values for all the studied nutrients, except for vitamin B₁₂ (21.3%). Most children were breastfed for more than 12 weeks (87%), 56% were born by cesarean and 57% were male. DDE mean levels were higher than p,p'-DDT (in lipid as well as in wet bases) and they did not vary significantly during pregnancy (Table 2). On average, all standardized child growth z-scores were within one standard deviation (Table 3). Still, there were children under the recommended guidelines for child growth, especially on weight-for-length measures (23.5%).

No significant crude associations were found between prenatal DDE exposure and child growth z-scores. DDE geometric means significantly varied between mothers with and without iron and zinc deficiencies. Iron and zinc modified by more than 10% the DDE beta coefficient of weight-for-length, while iron alone modified in the same way the beta for BMI-for-length (data not shown), thus, those nutrients were included in the corresponding multivariate final models. After adjusting for potential confounder variables in each model separately, no significant associations were observed (Table 4). Assessment of interaction caused by child's sex and breastfeeding, respectively, resulted not statistically significant.

4. Discussion

Our results show no evidence of an association between prenatal DDE exposure with any of the child anthropometric measurements during the first year of life. The present findings are similar to those reported by other cohort studies for some of the child growth anthropometric measurements reported here (Cupul-Uicab et al., 2010; Verhulst et al., 2009; Jusko et al., 2006). A longitudinal study among boys in Chiapas, Mexico found no association between prenatal DDE levels and height or BMI (Cupul-Uicab et al., 2010). Verhulst and colleagues conducted a 3-year prospective cohort study in which they found no association between intrauterine exposure to DDE and birth length or weight (Verhulst et al., 2009). Another prospective study also documented the lack of association between DDE exposure and birth weight using standardized anthropometric measurements, albeit their higher DDE concentrations (Jusko et al., 2006).

While our results are consistent with previous findings, others have illustrated divergent results. Two studies have suggested an association between DDE and decreased height throughout childhood (Ribas-Fitó et al., 2006; Karmaus et al., 2002). Differences in sample composition may explain the disparity in results, like race stratification (Ribas-Fitó et al., 2006) and exposure assessment (Karmaus et al., 2002). Another study documented an association with increased BMI modified by maternal smoking at 1 and 3 years of age (Verhulst et al., 2009). Even though our study shared almost the same percentage of smoking mothers (8.7% vs 9.6%, respectively), after adjusting for passive and active smoking during the analyses, no associations were observed with child BMI. Another cohort study showed an apparent head circumference increase at 5 years (Jusko et al., 2006), however this differs from our findings which were made during the first year of life.

Our results should be considered in light of some study limitations. No information on paternal height and child head circumference at birth was available. In comparison with other studies, our sample was small and this could have been a possible source of error due to lack of statistical power. Our DDE concentration levels were lower than those reported in other studies (Verhulst et al., 2009; Jusko et al., 2006; Ribas-Fitó et al., 2006; Gladen et al.,

2004; Karmaus et al., 2002). No information of DDE levels on maternal milk was available for this study, limiting our ability to draw conclusions about potential exposures from this source.

Additional regression models were generated with lipid-based DDE (ng/g) to evaluate differences in significance, but none were found (data not shown). Still, our study shares strong features. There are few possibilities that DDE measurement errors could be differential as a function of the children's anthropometric measurements and the maternal DDE levels, because the researchers who evaluated the child anthropometric measurements did not know the maternal levels to DDE and the high interlaboratory concordance coefficient of DDE serum values suggests valid estimates. Adjusting for nutritional variables unmask potential confounding that these variables could generate and this strengthens our results. Both hypertension (7.5%) and alcohol use (5%) prevalence were low in the study population. They did not produce changes in the betas of interest.

5. Conclusion

This study presents no evidence of an association between prenatal DDE exposure and child growth during the first year of life. Because these children were evidently exposed to DDE during a critical window in their lives, there is a strong need to evaluate other health-related outcomes later in their lives.

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Highlights

- This study evaluated prenatal DDE exposure and child growth among Mexican children.
- Maternal serum DDE levels at each trimesters of pregnancy were used.
- Child growth was evaluated using standardized anthropometric measurements (z-scores).
- No significant associations were observed with prenatal DDE exposure and each of the growth parameters.
- Results suggest no evidence of an association between prenatal DDE exposure and child growth during the first year of life.

Table 1

Maternal and child characteristics of study population.

Characteristics	Overall (n = 253)	
	n	%
Maternal		
Age (yrs, mean ± SD)	21.7 ± 3.9	
Height (cm, mean ± SD)	155.9 ± 6.6	
Education (yrs, mean ± SD)	10.6 ± 3.1	
Paid occupation	117	46.3
Parity (None)	208	82.2
BMI (mean ± SD) ^a	23.2 ± 3.8	
Passive smoking		
Not a passive smoker	92	36.4
Before pregnancy	114	45.1
During 1 st trimester	47	18.6
Active smoking		
Non-smoker	136	53.8
Before pregnancy	95	37.6
During pregnancy	22	8.7
Dietary intake ^a		
Folate (< 400 µg/day)	158	62.9
Vit B ₁₂ (< 2.0 µg/day)	49	19.5
Calcium (< 1,200 mg/day)	158	62.9
Iron (< 30 mg/day)	225	89.64
Zinc (< 15 mg/day)	238	94.82
Energy (kcal/d) ^b	2,819.9 (1,536.5; 4,690.5)	
Child		
Sex (Male)	143	56.5
Cesarean birth	142	56.1
Breastfed		
None	20	7.9
≤ 12 weeks	13	5.1
> 12 weeks	220	87.0

^aDuring first trimester of pregnancy^bMean (P5–P95)

Table 2

DDE and p,p'-DDT maternal serum levels during pregnancy.

		Wet bases (ng/mL)	Lipid bases (ng/g)
Organochlorine compounds	n	GM $\pm$ GSD ^a	GM $\pm$ GSD ^a
p,p'-DDE			
1 st trimester	220	6.3 $\pm$ 2.8	1105.1 $\pm$ 2.7
2 nd trimester	181	6.6 $\pm$ 2.9	842.6 $\pm$ 2.9
3 rd trimester	190	7.6 $\pm$ 2.9	710.6 $\pm$ 3.0
p,p'-DDT			
1 st trimester	220	0.00697 $\pm$ 2.9	0.03 $\pm$ 9.5
2 nd trimester	180	0.00581 $\pm$ 2.1	0.02 $\pm$ 6.6
3 rd trimester	198	0.00591 $\pm$ 2.2	0.03 $\pm$ 8.5

^aGM $\pm$ GSD: Geometric mean and standard deviation

Table 3

Child growth measurements from birth to 12 months of age.

	Age at evaluation (mean $\pm$ SD)					
	0	1	3	6	12	
Weight (kg)	3.2 $\pm$ 0.42	4.5 $\pm$ 0.6	6.2 $\pm$ 0.7	7.6 $\pm$ 0.9	9.3 $\pm$ 1.3	
Weight-for-age (<i>z-score</i>)	-0.18 $\pm$ 0.88	0.19 $\pm$ 0.95	0.009 $\pm$ 0.96	-0.10 $\pm$ 0.97	-0.09 $\pm$ 1.13	
% of Children ≥ -2 ; ≤ 2 SD	95.1	95.2	97.0	96.5	92.3	
Weight-for-length (<i>z-score</i>)	-0.81 $\pm$ 1.51	-0.88 $\pm$ 1.34	-0.57 $\pm$ 1.41	-0.34 $\pm$ 1.27	0.09 $\pm$ 1.40	
% of Children ≥ -2 ; ≤ 2 SD	76.2	80.0	80.5	88.0	84.1	
Length (cm)	50.4 $\pm$ 2.3	50.4 $\pm$ 2.3	56.0 $\pm$ 2.5	62.1 $\pm$ 2.9	74.3 $\pm$ 3.5	
Length-for-age (<i>z-score</i>)	0.41 $\pm$ 1.19	0.94 $\pm$ 1.27	0.70 $\pm$ 1.37	0.42 $\pm$ 1.32	-0.31 $\pm$ 1.42	
% of Children ≥ -2 ; ≤ 2 SD	87.2	78.6	83.1	84.1	86.5	
BMI (kg/cm ²)	12.8 $\pm$ 1.6	14.3 $\pm$ 1.4	16.0 $\pm$ 1.8	16.6 $\pm$ 1.8	16.9 $\pm$ 2.1	
BMI-for-age (<i>z-score</i>)	-0.59 $\pm$ 1.29	-0.38 $\pm$ 1.05	-0.51 $\pm$ 1.27	-0.45 $\pm$ 1.27	0.11 $\pm$ 1.49	
% of Children ≥ -2 ; ≤ 2 SD	79.8	93.0	86.0	86.7	82.1	
HC (cm)	-	37.6 $\pm$ 1.4	40.3 $\pm$ 1.4	43.0 $\pm$ 1.4	45.5 $\pm$ 1.4	
HC-for-age (<i>z-score</i>)	-	0.54 $\pm$ 1.19	0.22 $\pm$ 1.11	0.09 $\pm$ 1.04	-0.03 $\pm$ 1.07	
% of Children ≥ -2 ; ≤ 2 SD-	-	88.4	90.7	92.9	94.2	

Table 4

Mixed-effects models to assess prenatal DDE exposure on standardized child growth measurements throughout the first year of life.

Child Growth Measurements	β^a	95% CI	p-value
Weight-for-age ^b			
1 st trimester	0.02	−0.05 to 0.10	0.53
2 nd trimester	0.01	−0.06 to 0.09	0.71
3 rd trimester	0.02	−0.06 to 0.09	0.64
Length-for-age ^c			
1 st trimester	0.04	−0.05 to 0.12	0.39
2 nd trimester	0.01	−0.08 to 0.09	0.85
3 rd trimester	0.08	−0.004 to 0.16	0.06
Weight-for-length ^d			
1 st trimester	0.002	−0.08 to 0.08	0.94
2 nd trimester	0.01	−0.08 to 0.1	0.83
3 rd trimester	−0.01	−0.10 to 0.07	0.74
BMI-for-age ^e			
1 st trimester	0.02	−0.05 to 0.1	0.56
2 nd trimester	0.005	−0.08 to 0.09	0.91
3 rd trimester	−0.02	−0.10 to 0.06	0.64
HC-for-age ^f			
1 st trimester	−0.05	−0.14 to 0.04	0.27
2 nd trimester	−0.02	−0.11 to 0.07	0.64
3 rd trimester	0.05	−0.04 to 0.14	0.27

^a Change by doubling increase of DDE levels

^b Model adjusted for age at evaluation, maternal age, height, BMI, iron and caloric intake during first trimester of pregnancy.

^c Model adjusted for age at evaluation, maternal age, height and parity.

^d Model adjusted for age at evaluation, maternal age, height, education, BMI, tobacco, breastfeeding, iron, zinc and caloric intake during the first trimester of pregnancy.

^e Model adjusted for age at evaluation, BMI, tobacco, iron and caloric intake during first trimester of pregnancy.

^f Model adjusted for age at evaluation and maternal height.