THE CONTRIBUTION OF BREAST-FEEDING TO THE DEPURATION OF DDE

LA CONTRIBUTION DE L'ALLAITEMENT MATERNEL À LA DÉPURATION DU DDE

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ABSTRACT

The presence of persistent organic halogens (POHs) in maternal plasma and milk has been recognized since the 1970s. Yet, the presence of these compounds in human milk poses a still undetermined risk to the health of mother and child. The vertical transfer of POHs during either pregnancy or after birth during breast-feeding remains a concern. The paucity of data on maternal plasma contaminant levels before and after long periods of breast-feeding poses a serious problem for researchers attempting to model contaminant changes in maternal plasma and contaminant transfer via milk to the baby. This issue is of clinical concern since there is inadequate data to model infant exposure to POHs, leading to uninformed decisions about risks and benefits of breast-feeding. To address this issue we carried out a longitudinal analysis of the association between maternal plasma p,p'-DDE (1,1-dichloro-2,2-bis(p-chlorophenyl) ethylene) load and breast-feeding duration and parity. This pesticide residue was chosen because it is both an ubiquitous and abundant POH. The women recruited for the study were 34.4 $\pm$ 2.8 yr and had breast-fed an average of 2.1 children a total of 64.6 $\pm$ 32.6 weeks when they were sampled at the end of the study. A questionnaire was used to determine the mother's breast-feeding activity. We found that the women had very low levels of p,p'-DDE in their plasma and milk compared to other populations in the world and that the trend in Canada towards lower contaminant loads has been sustained in our study population. The regression of change in plasma p,p'-DDE with breast-feeding duration is significant ($p < 0.01$, $F = 15.80$, and $n=12$). We found that maternal plasma p,p'-DDE levels changed by 0.0057 μg/L for every week of breast-feeding. Thus, women breast-feeding their infants lost, on average, 30 μg of this contaminant per week. These results suggest that maternal depuration of p,p'-DDE via breast-feeding is a significant cause of maternal contaminant loss and contaminant accumulation in the baby.

KEY WORDS

persistent organic contaminants, persistent organic halogens, DDE, breast-feeding, pollution, human milk

THIS ARTICLE HAS BEEN PEER-REVIEWED

RÉSUMÉ

La présence d'halogènes organiques persistants (HOP) dans le plasma et le lait maternel est reconnue depuis les années 70. Toutefois, la présence de ces composés dans le lait maternel pose encore un risque indéterminé en ce qui a trait à la santé de la mère et de l'enfant. Le transfert vertical des HOP durant la grossesse ou après la naissance durant l'allaitement, demeure une source de préoccupations. L'indigence de données sur les niveaux de polluants présents dans le plasma maternel avant et après de longues périodes d'allaitement, pose un sérieux problème aux chercheurs qui essaient de déterminer les changements des polluants dans le plasma maternel et le transfert de polluants au bébé

par l'intermédiaire du lait maternel. Cette question est une source de préoccupation clinique puisque les données sont inadéquates pour déterminer l'exposition des enfants aux HOP. Ceci conduit à des prises de décisions qui ne sont pas éclairées à propos des risques et des avantages de l'allaitement. Pour aborder cette question, nous avons effectué une analyse longitudinale de la relation entre la teneur en p,p'-DDE (1,1 dichloro-2, 2-bis (p-chlorophénylique) éthylène) présent dans le plasma maternel et la durée de l'allaitement maternel et la parité. Ce résidu de pesticide fut choisi parce qu'il est un HOP à la fois omniprésent et abondant. Les femmes recrutées pour cette étude avaient 34.4 $\pm$ 2.8 ans et avaient allaité 2.1 enfants en moyenne pour une durée totale de 64.6 $\pm$ 32.6 semaines lorsque les échantillons furent prélevés à la fin de l'étude. Nous avons utilisé un questionnaire pour déterminer les comportements d'allaitement des mères. Nous avons déterminé que les niveaux de p,p'-DDE dans le plasma et dans le lait des femmes étaient très faibles comparativement aux autres populations à travers le monde et qu'au Canada, cette tendance à avoir des teneurs plus faibles en polluants fut maintenue dans la population de notre étude. La régression du changement de p,p'-DDE dans le plasma avec la durée de l'allaitement est significative ($p < 0.01$, $F = 15.80$, et $n=12$). Nous avons déterminé que les niveaux de p,p'-DDE dans le plasma maternel variaient de 0.0057 μg/L à chaque semaine d'allaitement. Donc, les femmes qui allaitaient leurs bébés, perdaient en moyenne 30 μg de ce polluant par semaine. Ces résultats laissent entendre que la dépuration maternelle de p,p'-DDE par l'intermédiaire de l'allaitement est une des causes significatives de la perte de polluants chez la mère et de l'accumulation de ceux-ci chez le bébé.

MOTS CLÉS
Les polluants organiques persistants, les halogènes organiques persistants, le DDE, l'allaitement, la pollution, le lait maternel

CET ARTICLE FUT RÉVISÉ PAR SES PAIRS

Introduction

The benefits of breastfeeding for the infant are numerous and well explored. However, since the 1970's persistent organic halogens (POHs) have been measured in maternal plasma and milk. Exposure to these compounds is thought to occur via ingestion of contaminated food products or workplace vapours, and is associated with living close to contaminated sites.[1,2,3,4] The presence of these compounds in human milk poses a still unknown health risk to mother and child.

POHs are lipophilic environmental pollutants containing chlorine or bromine that accumulate in the body and resist catabolism. These pollutants can be divided into two groups: 1) organochlorine compounds (OCs), which includes certain pesticides (e.g., p, p'-diphenyldichlorotrichloroethane (p,p'-DDT)) and pesticide breakdown products (e.g., p,p'-dichlorodiphenyldichloroethene (p,p'-DDE), as well as polychlorinated biphenyls (PCBs); and 2) brominated molecules, such as polybrominated biphenyls (PBBs) and brominated diphenyl ethers (BDEs). In humans, the majority of these chemicals can be detected in serum, tissue, amniotic fluid, ovarian follicular fluid, milk, and seminal plasma.[5,6] Generally, levels of persistent organochlorine compounds (POHs) were directly related to age; inversely related to parity or duration of breastfeeding.[7,8,9,10] The rate of loss (depuration) of POHs due to breastfeeding is not known.

In southern Canadian populations (defined as south of the 60 north latitude), human milk contaminant loads for the majority of POHs have fallen rapidly since the initiation of studies in the early 1970's, even though levels of BDEs have increased steadily since that time (Ryan, personal communication).[11] In fact, historic decreases in milk contamination have been found all over the world, except for BDE levels which have been doubling in concentration every five years since 1972.[8,12,13,14]

Few studies have examined the change in maternal contaminant loads of POHs compounds over an extended period of time. Czaja's group measured contaminants levels in milk and found that levels did not change consistently in any of the 8 women examined.[15] This study failed to indicate whether or not levels of POHs, such as p,p'-DDE or PCBs, decreased in maternal serum over the lactation period. The preponderance of data collected to date is on the

level of contaminants present in populations; however, there is relatively little data on the depuration rates of POHs, metabolically or vertically, in lactating woman. This lack of data makes meaningful predictions of changes in POH burden during lactation difficult.

A further complication is the lack of studies which attempted to separate the effect of parity versus that of breastfeeding on alteration of maternal contaminant loads. Studies focusing on parity have failed to factor out the effect of breastfeeding, and conversely, breastfeeding studies have not separated out the effect of parity. LaKind points out that "the limitations of the existing data restrict our ability to predict infant body burdens of these chemicals from breast-feeding".[16]

A wealth of data exists demonstrating an array of developmental difficulties associated with fetal POH exposure. It has been shown that PCB exposure significantly decreased children's performance on several developmental tests. [17, 18, 19] The literature on the developmental effects of POHs has recently been reviewed by Schantz. [20] A variety of epidemiological reports show long term adverse reproductive health outcomes associated with organochlorine contaminants including increased time to pregnancy (TTP), decreased fertility, increased spontaneous abortion, and disrupted menstrual cycles.[21, 22, 23, 24, 25, 26, 27] Studies on mice exposed to modest levels of PCBs support human data: showing decrements in levels of mouse fertility.[28] Alterations in human endocrine function have also been reported, as well as changes in sex ratio of offspring.[29, 30, 31] Adding to the confusion, however, are studies which refute the role of PCBs in fertility and sex ratio.[32, 33] The available literature neither supports nor refutes the hypothesis that human reproductive health is significantly altered by typical loads of POHs.[34, 35, 36] In summary, the role of POHs as toxicants remains equivocal, particularly when the study of effects is confined to either human populations or experimental animals with modest contaminant exposure.

Vertical transfer of POHs either to the fetus via the placenta, or to the infant during breastfeeding remains a concern. And the paucity of longitudinal data on maternal blood contaminant levels poses a serious problem for researchers attempting to model contaminant changes in maternal plasma and contaminant transfer via milk to the baby. This issue is of clinical concern since there is inadequate data to model infant exposure to POHs, leading to uninformed decisions about risks and benefits of breastfeeding. In an attempt to address this issue, the present study presents longitudinal data on the association between maternal human milk p,p'-DDE load and breastfeeding duration and parity in a population of women with low contaminant loads.

Methods

The women who participated in this study were selected from a group of 65 women recruited in 1996-1997 study investigating the relationship between contaminant load and time to pregnancy (TTP). The recruitment criteria for this cohort included women who resided in Hamilton, Ontario, were less than 35 years of age, non-smokers, and who were, for the first time, in the third-trimester of their pregnancy. All subjects were recruited through the obstetrical service of St. Joseph's Hospital in Hamilton, Ontario and all women were delivered at this site.

Approval for this study was obtained from the Research Ethics Committee of St. Joseph's Hospital. All of the test subjects were informed of their TTP results by mail following the study. Those who could not be contacted by mail were reached through their family physicians. Over 90% of the original participants were notified of the study to look at contaminant levels in human milk. Of this number, 15 were recruited into the new study by the completion of sampling in November, 2001. All of the women had blood samples taken, but milk samples were only available from 12-- the remaining women either did not attempt to breast-feed or had ceased breastfeeding before samples could be taken. Although greater numbers of recruits were hoped for, the successful enrollment of 15 women represents the largest longitudinal follow-up study of this kind to date, a fact that reflects the difficulties inherent in this type of study.

At commencement of the study, subjects were provided with a double-breast pump and collection containers to store the milk samples. All equipment was thoroughly washed and rinsed with distilled water and alcohol prior to use. To be sensitive to the needs

and challenges of these new mothers, all test subjects were encouraged to arrange their appointments with the research nurse according to their needs. Emotional support was essential to enhance collection and compliance.

Maternal blood samples were processed by the clinical chemistry laboratory at St. Joseph's Hospital, Hamilton, Ontario. A questionnaire was used to determine the mother's time to pregnancy, diet, and breastfeeding activity. Questionnaires were completed during the initial home visit or left for the woman to complete at her convenience. They were then returned by mail within a few weeks of collection. Participants indicated a high degree of support for the study, and were compensated with a $50 gift certificate for a local pharmacy. Parking expenses at the hospital were also covered when necessary.

Chemical analysis of plasma and milk was conducted for p, p'-DDE, p, p'-DDT, endosulphan, hexachlorobenzene (HCB), â-hexachlorocyclohexane (benzene hexachloride, hexachlorohexane, â-BHC, BHC), Mirex, trans-nonachlor (t-nonachlor), oxychlordane, and the PCB congeners: 5+8, 28, 52, 66+95, 74+94, 99, 101, 105, 118,138,146+188, 153, 156+171, 170, 172, 177, 180, 183, 194, 196+203, 201, 202+157 and 206. Plasma cotinine was also measured. The analytical work was carried out under the supervision of Jean-Philippe Weber from the Quebec National Institute of Public Health, Quebec City, PQ. In this paper the results for p,p-DDE will be considered since it showed up in 100% of the samples and is implicated as an endocrine disrupting substance.

The dry weight (dw) lipid percentage of plasma and milk was determined by Dr. Michael Arts from the National Water Research Institute, Burlington, ON. We also had these values corroborated by the Quebec National Institute of Public Health. We observed no significant difference between values determined at the two sites. Normalization of the lipid content of milk is important since the lipid percent of milk varies widely.[37]

Statistics

All data was analyzed using SPSS 10. All analyses were conducted on lipid corrected and uncorrected POHs.

Results and Discussion

The 12 women in the current study were 34.4 ± 2.8 years of age, and had breast-fed an average of 2.1 children over a total of 64.6 ± 32.6 weeks. The average age of the study population at the beginning of sampling was 29.0, with an age range of 9 years. One of the benefits of measuring contaminants in breastfeeding women is that the ranges in ages are generally small, because reproduction and breastfeeding are concentrated from the mid-twenties to the mid-thirties, minimizing heterogeneity due to age.

Although gas chromatography (GC) has improved markedly since the 1970s, both in terms of detector sensitivity and quality control, methodological differences remain a source of error when comparing data between studies. Data for this study were generated in one laboratory using identical methods (GC with electron capture detector), providing us with a high degree of methodological consistency. It should be noted that comparisons between our results and those obtained using different extraction and analytical methods can be problematical.

Body mass and body mass index were measured for this study but the values were not incorporated into the analysis. However, the effect of weight loss and body mass index on circulating POH load has been documented by others.[38, 39] Unfortunately these variables cannot be fully compensated for even if the degree of weight loss is known. Furthermore, weight loss due to breastfeeding has not been shown to be associated with significant changes in contaminant body burden.[40] We found plasma p,p'-DDE levels to be 0.86 ± 0.30 µg/l, a value nearly three times lower than one observed by Greizerstein et al. (1999), 1.87 ±1.21 ìg/L (Mean ± Standard Deviation, Table 2), who examined DDE levels in a population from New York State. Similar differences are seen for other POHs.[41] For example, HCB levels in Greizerstein's study were 30.27± 18.12, while hexachlorobenzene (HCB) in our study was 9.99 ±3.04.[41] The lipid adjusted value for p,p'-DDE in our study was 50 ± 25 ìg/kg of lipid compared to a value of 316 ±171 ìg/kg measured by the Greizerstein group.[41] POH levels found in our group at the commencement of the study before any breastfeeding were lower than those reported in the

Anderson study or by Huisman in the Netherlands; or in any other study accessed in the literature.[42,43]

The data in Table 2 also shows the effect of parity on plasma contaminant loads. We compared the contaminant levels in woman following the birth of a first child, but prior to breastfeeding (t=1), to the levels found after the birth of a second, or in two cases a third baby, at a time 5-6 years after the birth of the first child: the time of this second sampling is designated t=2. p,p'-DDE levels at t=1 are low prior to breastfeeding, but became even lower at t = 2. It is interesting to note that in a few cases the contaminant loads of individuals increased (Table 3). One would expect this in the non-lactating population because of the positive relationship between age and POH load. The age-contaminant relationship is thought to be driven by the increased time of exposure to persistent contaminants. This is only part of the story, however, since exposure to organochlorines in food has decreased since approximately 1972.[44] It is likely that lower POHs loads in younger people are also due to the fact that food has become less contaminated and not just that that younger people have consumed less food.

In our study there was no relationship between contaminant load and age at t = 1 or t = 2. Furthermore, among the 65 women in the initial cohort there was no relationship between age and contaminant load.[45] At t=2 additional factors, including differences in breastfeeding duration and parity, make it less likely that a simple age-contaminant load relationship will be apparent.

Regression analysis was performed between maternal plasma p,p'-DDE levels post-breastfeeding and the duration of breastfeeding. And in our study the relationship between these two variables was not significant (n=12, F=0.15 p=0.71). This result was not effected by correcting for lipid content, as the lipid corrected p,p'-DDE at t=2 was also not significantly related to breastfeeding duration.

The lack of a relationship observed in our group between the duration of breastfeeding and plasma POH levels should not be surprising. Studies that demonstrate a relationship between maternal blood/milk contaminant concentration and breastfeeding history and/or parity have large sample numbers, often more than 100.[9, 46] However, these studies cannot be used to calculate the rate of maternal depuration via breastfeeding since they have no data on contaminant loads before commencement of breastfeeding.

Our study was designed to see if the change in contaminant load was related to both breastfeeding duration and parity. Correlative studies looking at total body burden are obscured by the fact that contaminant load is determined by a number of factors, such as age and diet, which are not related to either breastfeeding or parity. In the present study we were particularly interested in calculating the extent to which depuration results from these two variables. However, to calculate change one must first have knowledge of the initial body burden.

Regression analysis of the change in maternal p,p'-DDE levels did correlate significantly with the duration of breastfeeding ($p < 0.01$, $F=15.80$, and $n=12$). The linear regression provided a slope coefficient $\pm$ SE of 0.0057 ± 0.001, with an intercept $\pm$ SE of 0.11 ± 0.11, and an $r^2 = 0.61$ (Figure 1). The units for the slope are μg/L p,p'-DDE per week breastfeeding; therefore, maternal p,p'-DDE changes by 0.0057μg/L for every week of breastfeeding. Assuming a plasma volume of 5 L, the contaminant lost by the mother is approximately 30 μg per week of breastfeeding. The assumption that plasma volume was 5 L at both sampling times is a conservative estimate of that variable. It is probable that the plasma volume at t=1 was approximately 20% greater than at t=2 since at t=1 the women were pregnant and hemodilution had occurred. If the initial plasma volume is greater than 5 L then the depuration rate is greater than reported here since the contaminants were diluted at t=1 and the actual depuration rate is higher. The initial plasma level of p,p'-DDE in the women was about 0.86 μg/L; therefore, approximately 0.67% of total p,p'-DDE is lost per week of breastfeeding.

Correcting the contaminant load for the lipid content of milk or plasma did not appear to alter the relationship in a significant fashion. The purpose of lipid correction is to adjust for alterations in lipid content which results in changes in the levels of lipophilic contaminants. The change in the lipid-corrected p,p'-DDE level with weeks of breastfeeding was also significant ($p<0.01$, $F=14.2$, $n=12$). The

regression has a slope of slope coefficient ± SE of -0.52 ± 0.138, an intercept ± SE of -2.13 ± 10.4 and an r^2=0.59 (Figure 2).

The mean value of the milk contaminant load of p,p'-DDE was 3.18 ± SD of 2.26 μg/L (range 0.66-8.40 μg/L). Other authors have shown that the amount of p,p'-DDE in human milk is related to maternal plasma or plasma contaminant loads. In our study, there was not a significant relationship (F= 1.70, p=0.23), though our sample numbers are relatively low (n=10). This discrepancy with the literature may also be due to the relatively low levels of contaminant in both plasma and milk which made plasma contaminant analysis somewhat less accurate and the lack of variability in contaminant data between subjects.

Other authors have established that maternal plasma or serum contaminant load is related to breastfeeding duration or some proxy measure of breastfeeding behaviour as well as parity.[9, 46] The contribution of parity, distinct from that of breastfeeding duration, has not been studied in great detail and is very difficult to examine separately unless data sets are large and detailed breastfeeding histories are known. We considered parity as a categorical, non-continuous variable because we had only parities of two or three. Using this approach, we were able to use breastfeeding as a predictor; and the contaminant load as a response variable. In this analysis of covariance, once variance due to breastfeeding was taken into consideration, we were unable to detect any caused by parity alone. This, however, is not a very robust test for the effect of parity since our sample size is small and parity was only two or three.

The lack of data may well have obscured any effect of parity; even so, breastfeeding is likely the major source of POH to babies and depuration of the mother. The rationale for this is that the amount of lipid and therefore contaminant in a newborn is relatively small compared to the amount of lipid transferred by breastfeeding. The lipid in a newborn would be on the order of 700 grams, for a 3.5 kg baby, assuming a body fat of about 20 %.[48] Because the fat in the newborn is derived from the maternal fat stores, in the same fashion as the fat in milk, then it is likely that contaminant transfer to the fetus through maternal lipid is similar to the transfer of contaminant in milk. If the transfer of lipid is approximately equal to the transfer of POHs, then breastfeeding would transfer an amount of contaminant in the first 28 days of breastfeeding equal to all the contaminant present when the baby was born, assuming a consumption of 500 ml/day and a milk fat of 3.5 %.[37, 48] A child breastfeeding exclusively at 6 months, and consuming approximately 800 ml/day of milk, would consume a similar amount of contaminant in roughly 18 days.[48] Because the amount of contaminant lost by the mother during birth is so small compared to that lost during breastfeeding, studies that purport to measure a "parity only" effect on maternal load of POHs must also be sensitive enough to distinguish between a few weeks of breastfeeding, which seems unlikely.

A further difficulty with studies that simply looked at parity is that they must also take into account age; because older women tend to have greater parity, not just longer breastfeeding duration. It is my opinion that there is not much variance in the POH data left to be explained once age and breastfeeding effects are taken into account.

A serious problem from a public health standpoint is that studies that show that breastfeeding results in decreases in maternal contaminant load and increases in the levels of contaminants in infants tend to give the impression that all breastfeeding will lead to the same amount of maternal decontamination and infant contamination. For example, Angulo et al. 1999 modeled contaminant intake by the infants as breastfeeding duration times the average of 100 contaminant loads measured in colostrum.[49] The first problem with this approach is that the average loss is not a useful number for decision making for individuals because contaminant concentration is governed by maternal load. The second problem is that contaminant loss due to contaminant movement from maternal plasma to milk is not zero order process (a constant number), rather it is a first order process, one which is dependent on the initial load of the individual; therefore, predictions made using a zero-order model could either under or over estimate the amount of contaminant transferred. In summary, it would be wrong to make a decision regarding breastfeeding without knowing what the maternal load is prior to breastfeeding and the true loss rate of POHs.

TABLE 1:
Summary statistics for maternal plasma organic contaminants loads at the birth of the first child. LC = lipid corrected (adjusted). Log transformations were accomplished after adding 1 to the original values to avoid negative scores.

Maternal Plasma Organic Contaminants	Mean	Standard Deviation	Maximum	Minimum	N
DDE (ug/L)	.56	.22	.97	.18	12
Log of DDE (ug/L)	.19	.06	.29	.07	12
LC DDE (ug/kg lipid)	52.13	27.84	122.01	15.93	12
Log of LC DDE (ug/kg lipid)	1.67	.23	2.09	1.23	12
DDT (ug/L)	.03	.02	.07	.02	12
Log of DDT (ug/L)	.01	.01	.03	.01	12
LC DDT (ug/kg lipid)	3.10	2.34	8.81	1.04	12
Log LC DDT (ug/kg lipid)	.56	.22	.99	.31	12
DDT + DDE (ug/L)	.60	.23	1.04	.23	12
Log of DDT + DDE (ug/L)	.20	.06	.31	.09	12
LC DDT + DDE (ug/kg lipid)	55.23	29.66	130.82	20.35	12
Log of LC DDT + DDE (ug/kg lipid)	1.70	.22	2.12	1.33	12

TABLE 2:
Summary statistics for maternal plasma organic contaminants loads 5-6 years after initial measurements. The women have increased parity by at least one and breastfed there children various lengths of time.
DDE= p,p'-dichlorodiphenyldichloroethane,
LC = lipid corrected (adjusted). Log transformations were accomplished after adding 1 to the original values to avoid negative scores.

Maternal Plasma Organic Contaminants	Mean	Standard Deviation	Maximum	Minimum	N
DDE (ug/L)	.56	.22	.97	.18	12
Log of DDE (ug/L)	.19	.06	.29	.07	12
LC DDE (ug/kg lipid)	52.13	27.84	122.01	15.93	12
Log of LC DDE (ug/kg lipid)	1.67	.23	2.09	1.23	12
DDT (ug/L)	.03	.02	.07	.02	12
Log of DDT (ug/L)	.01	.01	.03	.01	12
LC DDT (ug/kg lipid)	3.10	2.34	8.81	1.04	12
Log LC DDT (ug/kg lipid)	.56	.22	.99	.31	12
DDT + DDE (ug/L)	.60	.23	1.04	.23	12
Log of DDT + DDE (ug/L)	.20	.06	.31	.09	12
LC DDT + DDE (ug/kg lipid)	55.23	29.66	130.82	20.35	12
Log of LC DDT + DDE (ug/kg lipid)	1.70	.22	2.12	1.33	12

TABLE 3:
Summary statistics for the change in maternal plasma organic contaminants loads after an increase in parity of at least one and variable periods of breastfeeding.
DDT = p,p'-dichlorodiphenyltrichloroethane,
DDE= p,p'-dichlorodiphenyldichloroethane,
LC = lipid corrected (adjusted).

Maternal Plasma Organic Contaminants	Mean	Standard Deviation	Maximum	Minimum	N
Change in DDE load (ug/L)	-.28	.25	.01	-.80	12
Change in LC DDE load (ug/kg lipid)	-37.34	23.25	-.78	-78.26	12
Change in the sum of DDE and DDT (ug/L)	-.29	.27	.02	-.85	12
Change in the sum of LC DDE and DDT load (ug/kg lipid)	-38.51	24.61	.12	-82.53	12
Change in DDE load (%)	-30.48	20.71	.90	-57.11	12
Change in LC DDE load (%)	-40.80	21.50	-1.46	-74.57	12
Change in the sum of DDE and DDT (%)	-29.21	20.70	2.63	-57.45	12
Change in the sum of LC DDE and DDT (%)	-39.82	21.34	.23	-74.21	12

FIGURE 1:
The relationship between the change in maternal plasma p,p'-DDE concentration and breastfeeding duration in weeks. The regression line has error bars at the 95% confidence interval.

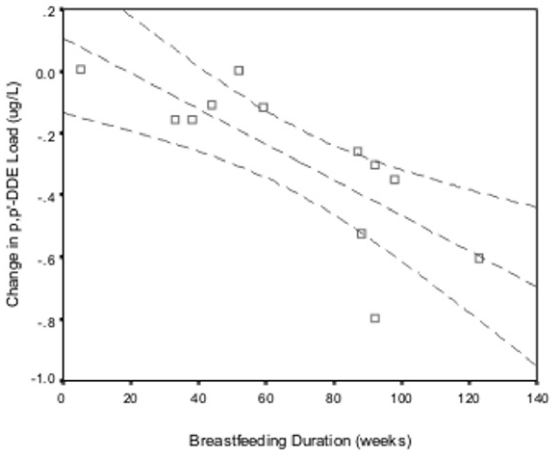
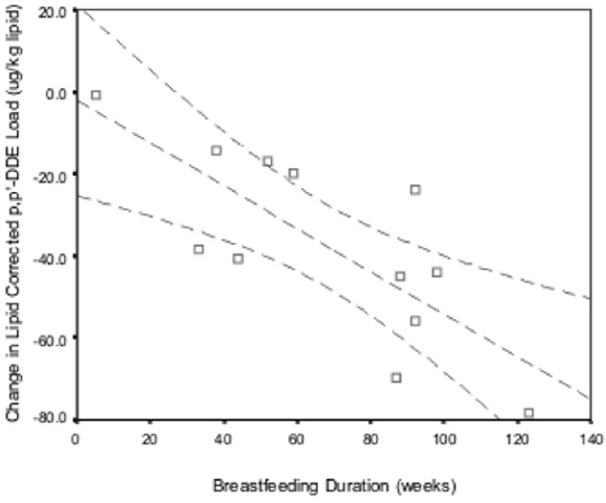


FIGURE 2:
The relationship between the change in maternal plasma p,p'-DDE concentration adjusted for lipid content of the plasma and breastfeeding duration in weeks. The regression line has error bars at the 95% confidence interval.



FOOTNOTES

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