Uploaded to VFC Website

July 2013

This Document has been provided to you courtesy of Veterans-For-Change!

Feel free to pass to any veteran who might be able to use this information!

For thousands more files like this and hundreds of links to useful information, and hundreds of "Frequently Asked Questions, please go to:

Veterans-For-Change

Veterans-For-Change is a 501(c)(3) Non-Profit Corporation
Tax ID #27-3820181

If Veteran's don't help Veteran's, who will?

We appreciate all donations to continue to provide information and services to Veterans and their families.

https://www.paypal.com/cgi-bin/webscr?cmd=_s-xclick&hosted_button_id=WGT2M5UTB9A78

Note: VFC is not liable for source information in this document, it is merely provided as a courtesy to our members.

Addendum to the DDT/DDD/DDE Toxicological Profile

Agency for Toxic Substances and Disease Registry
Division of Toxicology and Environmental Medicine
Atlanta, GA 30333

November 18, 2008

TABLE OF CONTENTS

LIST OF FIGURES

LIST OF TABLES

ADDENDUM for DDT/DDD/DDE
Supplement to the 2002 Toxicological Profile for DDT/DDD/DDE

Background Statement

This addendum for DDT/DDD/DDE supplements the Toxicological Profile for DDT, DDE, and DDD that was released in 2002.

Toxicological profiles are developed in response to the Superfund Amendments and Reauthorization Act (SARA) of 1986 which amended the Comprehensive Environmental Response, Compensation, and Liability Act of 1980 (CERCLA or Superfund). CERCLA mandates that the Administrator of ATSDR prepare toxicological profiles on substances on the Priority List and that the profiles be revised "no less often than once every three years". CERCLA further states that the Administrator will "establish and maintain inventory of literature, research, and studies on the health effects of toxic substances" [§ 9604, Section 9604 (i)(1)(B)].

The purpose of this addendum is to provide, to the public, other federal, state, and local agencies a non-peer reviewed supplement of the scientific data that was published in the open peer-reviewed literature since the release of the profile in 2002.

Chapter numbers in this addendum coincide with the toxicological profile for DDT (2002). This document should be used in conjunction with the profile. It does not replace it.

2. Relevance to Public Health

2.2 Summary of Health Effects

Reproductive/Developmental Effects. Several studies have been conducted to assess the relationship of DDT on male reproduction parameters. Overall, the available epidemiological evidence suggests that if such a relationship exists, it is found in populations exposed to high DDT concentrations. An association between high serum DDE concentrations and decreased male reproduction parameters has been found in studies conducted in areas where DDT was used for vector-control (Aneck-Hahn et al., 2007; Ayotte et al. 2001; De Jager et al. 2006).However, no association was found in studies conducted in populations with lower DDE/DDT serum

concentrations (Charlier and Foidart 2005; Hauser et al. 2003a, 2003b; Rignell-Hydbom et al. 2004, 2005; Spanò et al. 2005).

2.3 Minimal Risk Levels

No changes in MRLs from September 2002 Toxicological Profile

Acute-duration oral MRL = 0.0005 mg/kg/day

Intermediate-duration oral MRL = 0.0005 mg/kg/day

3. Health Effects

3.2.2.2 Systemic Effects

Rignell_Hydbom et al. (2007) analyzing serum-samples from Swedish women (n=544) found a significant association of p,p'-DDE (OR 1.3; 95%CI: 1.1- 1.6) with diabetes mellitus types.

3.2.2.3 Immunological and Lymphoreticular Effects

Cross-sectional studies conducted in German children reported a strong relationship between DDE and asthma, and increased total immunoglobulin E (IgE) (Karmaus et al., 2001; 2003). Beard et al (2003) reported an increase of asthma mortality and asthma prevalence in adults among an older cohort of DDT sprayers. In a longitudinal study among women attending antenatal care and their offspring in Menorca, Spain, Sunyer et al. (2005) investigated the association of prenatal exposure to DDE and asthma in children. Asthma was defined as wheezing at 4 years of age, persistent wheezing, or doctor-diagnosed asthma. Wheezing at 4 years of age increased with DDE concentration, particularly at the highest quartile (>1.90 ng/ml) with a RR of 2.36 (95%CI: 1.19, 4.69) compared to the lowest quartile (<0.57 ng/ml) after

adjusting for maternal asthma, maternal smoking, breastfeeding, education and other organochlorine levels. Specific IgE was not associated with DDE.

3.2.1.5 Reproductive Effects

Various reproductive and hormonal endpoints have been examined in both men and women, and although associations have been recorded, causal links have not been confirmed. Table 3.1 , which was adapted from Rogan and Chen (2005), summarizes the published studies of DDT and DDE effects by reproductive outcomes.

Spontaneous Abortion

In a matched case-control study among female Chinese textile workers (age 22-34 years), selected from a longitudinal study of reproductive effects, 15 women with spontaneous abortions (cases) were compared with 15 women with live births (controls) regarding serum DDT and DDE levels (Korrick et al. 2001). A significant higher serum level of p,p′-DDE (22 vs.12 ng/g) and o,p′-DDE (0.09 vs. 0.05 ng/g) was present in cases than in controls. After adjustment for age and body mass index, each ng/g serum increase in p,p′-DDE was associated with a 1.13 [95% confidence interval (CI): 1.02, 1.26) increased odds of spontaneous abortion.

In a study of 45 Japanese women with a history of three or more consecutive first-trimester spontaneous abortions, Sugiura-Ogasawara et al. (2003) did not observe an association between blood DDE (0.70 ±0.51 ppb) compared to 30 healthy women with no history of live birth and infertility (0.91±0.58 ppb) .

Salazar-Garcia et al. (2004) examined the association of occupational exposure to DDT and reproductive outcomes in 2,033 male workers (mean ± SD adipose tissue DDE =61±93 µg/g fat) in the anti-malaria campaign of Mexico. Workers provided information about 9,187 pregnancies. No significant association was found for spontaneous abortion or sex ratio. However, an increased risk of birth defects associated with high occupational exposure to DDT in this group of workers was found. Fifty-five children from these pregnancies were born with birth defects: the most common congenital malformations reported were in the nervous system (13 cases) and the osteomuscular system (12 cases). Other birth defects cases were as follows: eye and ear (7 cases,), cardiovascular (6 cases,), leporino lip (6 cases), chromosomal (9 cases), and other congenital malformations (9 cases).

Recently, Longnecker et al. (2005) investigated 1717 pregnant women enrolled in the U.S. Collaborative Perinatal Project (United States, 1959-1965) for the relationship between stored serum level of DDE and fetal losses in previous pregnancies. Odds of previous fetal loss were examined in relation to DDE level in logistic regression models. Women were grouped according to their DDE level : <15µg/L; 15-29 µg/L; 30-44 µg/L; 45-59 µg/L; and 60+ µg /L. When compared with women whose DDE level was <15 µg/L, the adjusted odds ratios of fetal loss per 60 µg/L increase was 1.4 (95% CI: 1.1, 1.6). The results were consistent with an adverse effect of DDE on fetal loss, but were inconclusive owing to the possibility that previous pregnancies ending in fetal loss decreased serum DDE levels less than did those carried to term.

Venners et al (2005) examined the association of preconception serum total DDT (sum of DDT isomers and metabolites) concentration and subsequent pregnancy losses in 388 newly married,

nonsmoking, female textile workers in China between 1996 and 1998. Daily urinary human chorionic gonadotropin was assayed to detect conception and early pregnancy losses, and pregnancies were followed to detect clinical spontaneous abortions. There were 128 (26%) early pregnancy losses in 500 conceptions and 36 (10%) spontaneous abortions in 372 clinical pregnancies. The subjects were grouped in tertiles by preconception serum total DDT concentration (group 1: 5.5-22.9 ng/g; group 2: 23.0-36.5 ng/g; group 3: 36.6-113.3 ng/g). Odds ratios were adjusted for maternal age, education, body mass index perceived life stress, tea drinking habit, occupational exposures to dust and noise, and exposure to passive smoking from husband. When compared to the referent group 1, group 2 had adjusted relative odds of early pregnancy losses of 1.23 (95% CI: 0.72, 2.10), and group 3 had adjusted odds of 2.12 (95% CI: 1.26, 3.57). The relative odds of early pregnancy losses associated with a 10 ng/g increase in serum total DDT were 1.17 (95% CI: 1.05, 1.29). Spontaneous abortions following clinical detection of pregnancy were not associated with serum total DDT.

Preterm Delivery

Torres-Arreola and colleagues (2003), in a case-control study of 233 women in maternity hospitals in Mexico City, found no association between serum DDE and preterm delivery. The median serum DDE level was 0.19 µg/g for the case group, and 0.15 µg/g for the control group.

Birth Weight

In a study of 912 infants in the USA, maternal serum DDE (median = 13µg/L) was not associated with birth weight (Rogan et al. 1986).

Bjerregaard et al. (2000) in a study of 178 newborn babies in Greenland found no association between maternal serum DDE (mean =5 µg/L) and birth weight.

In a case-control study among Indian women (Siddiqui et al. 2003), raised maternal serum DDE was associated with increase risk of uterine growth restriction. The mean serum DDE of the case group (n=30) and the control group (n = 24) were 9 µg/L and 6 µg/L, respectively.

In a study of 197 singleton infants from two Ukraine cities, Gladen et al. (2003) found no association between maternal milk DDE (2.5 µg/g fat) and birth weight.

Karmaus et al (2004) identified 168 offspring who were born after 1968 and had maternal exposure information from the Great Lakes Fish Eater Study in Michigan. Maternal PCB and DDE serum measurement closest to the date of delivery was used for the analysis. The maternal DDE and PCB serum concentrations were categorized as follows: $0 \leq 5$ µg /L, $5 \leq 15$ µg /L, $15 \leq 25$ µg /L, ≥ 25 µg /L. Estimated adjusted mean birth weight were controlled for gender, birth order, gestational age, date of delivery as well as maternal age, height, education, and smoking status. The authors found a reduced birth weight for the offspring of mothers who had a PCB concentration ≥ 25 µg /L (adjusted birth weight = 2,958 g, $p = 0.022$).The birth weight of offspring was increased in women with higher DDE concentrations when controlling for PCBs; however, this association was not statistically significant.

Weisskopf et al. (2005) investigated the role of DDE in relation to birth weight in 143 mothers consuming Great Lakes (USA) sport-caught fish. They found that the natural log of maternal serum DDE was associated with decreased birth weight.

Using data from the Child Health and Development Studies, a longitudinal study of 20,754 pregnancies among San Francisco Bay Area women from 1959 to 1967, Farhang et al (2005) examined the effects of maternal serum DDT and DDE concentrations on preterm birth, small-for-gestational-age birth, birth weight, and gestational age in 420 male subjects. Data were analyzed using multivariate logistic regression for preterm and small-for-gestational-age birth and linear regression for birth weight and gestational age. Median serum concentrations of DDE were 43 µg /L (interquartile range: 32-57; range: 7-153) and of DDT were 11 µg /L (interquartile range: 8-16; range: 3-72), several times higher than current US concentrations. The adjusted odds ratio for preterm birth was 1.28 (95% confidence interval (CI): 0.73, 2.23) for DDE and 0.94 (95% CI: 0.50, 1.78) for DDT. For small-for-gestational-age birth, the adjusted odds ratio was 0.75 (95% CI: 0.44, 1.26) for DDE and 0.69 (95% CI: 0.73, 1.27) for DDT; none of the study results achieved statistical significance. Jusko et al. (2006), using the same cohort, also did not find any relation of maternal serum DDT or DDE and impaired growth of the offspring at child birth as well as at five years of age.

High prenatal exposure to p,p'-DDE has been reported to decrease height in children (Ribas-Fitó et al. 2006). Using stored serum samples from pregnant women from the US Collaborative Perinatal Project (CPP), the authors compared the height of 1,712 children born between 1959 and 1966 with measured p,p'-DDE concentrations in their mother's serum samples from pregnancy. The highest prenatal concentrations of p,p'-DDE (≥ 60 microg/l), as compared with the lowest (<15 microg/l), were associated with decreased height at age 1 year [adjusted coefficient (SE) = -0.72 cm (0.37), n = 1,540], 4 years [-1.14 cm (0.56), n = 1,289], and 7 years

[-2.19 (0.46), n = 1,371]. Among subjects in lower categories of exposure no association was observed.

Fenster et al. (2006), in a birth cohort of 385 low-income Latinas living in the Salinas Valley, California, examined the effect of maternal serum DDT and DDE on gestation length, birth weight, and crown-heel length. No adverse association was found between DDT and DDE exposure and the outcomes investigated.

No associations were found between p,p'-DDE exposure during pregnancy and any measures of infant birth size, such as birth weight, crown-heel length and head circumference in a cohort of 722 infants born between 1993 and 1998 to mothers residing near a Superfund site in New Bedford, Massachusetts (Sagiv et al. 2007).

Time to pregnancy

Cohn et al (2003) compared p,p'-DDT and p,p'-DDE in preserved maternal serum samples, drawn 1-3 days after delivery between 1960 and 1963, with recorded time to pregnancy in 289 eldest daughters 28-31 years later. Maternal serum DDE (median =48 µg /L) was associated with raised probability of daughters' pregnancy, whereas maternal serum DDT (median = 13 µg /L) was associated with reduced probability of daughters' pregnancy.

Law et al (2005) investigated the association between DDE exposure and time to pregnancy by using serum levels measured in 390 pregnant women in the Collaborative Perinatal Project enrolled at 12 study centers in the United States from 1959 to 1965. The authors estimated adjusted fecundability odds ratios by using Cox proportional hazards modeling for discrete time

data. Compared with time to pregnancy for women in the lowest exposure category, DDE <14 µg /L, time to pregnancy increased for women in the highest exposure category (fecundability odds ratio for DDE ≥ 60 µg /L = 0.65, 95% CI: 0.32, 1.31), but did not reach statistical significance.

Cocco et al. (2005) examined the association of occupational exposure to DDT and reproductive outcomes in spouses of 105 male workers in Sardinia, Italy. They found an increased stillbirth and reduced male/female ratio among offspring and reduced fecundity ratio among DDT-exposed workers, and particularly among DDT applicators.

Duration of Lactation

Karmaus et al. (2005) using data from 176 pregnancies of 91 mothers in the USA who had maternal exposure information and gave birth between 1969 and 1995, extrapolated DDE concentrations. They found that the extrapolated DDE in non-smoking mothers was associated with reduced duration of lactation. However, the results should be interpreted with caution since the exposure data are not actually measured.

Conversely, in a population study in Chiapas, Mexico, Cupil-Icab et al. (2008) did not find support for the hypothesis that DDE exposure shortens length of lactation. The study was conducted in a cohort of 784 mother–son pairs in which maternal serum DDT and DDE levels at delivery had been previously determined. This population was exposed to DDT for almost 40 years: DDT was used for agriculture until 1991 and for malaria control until 1998.

Menstrual cycle

Windham et al (2005) investigated the relation between DDT/DDE and ovarian function in 50 women (age 18-40 years) born in Southeast Asia (49 born in Laos and one born in Vietnam) residing in the USA. Mean and SD serum concentrations for DDT was 1.77±3.84 ng/ml (range: 0.04-22.1) and for DDE was 20.8±22.9 ng/ml (range: 0.99-113.5). Analysis by quartile, adjusted for lipid levels, found a decrement in mean cycle length in the highest quartile group of DDE compared to the lowest quartile (referent group) of 4.3 days (95%CI: -8.3, -0.22). When the analysis was adjusted for age, parity and tubal ligation status, the resulting mean cycle decrement was attenuated to 0.74 days and did not achieve statistical significance (95%CI: -4.4, 2.9). Adjusted mean luteal phase length was shorter, approximately 1.5 days at the highest quartile of DDE (95%CI: -2.6, -0.20) and DDT (95% CI: -2.6,- 0.30). With each doubling of DDE concentration, the cycle length decreased 1.1 days (95% CI: -2.4, 0.23) and luteal phase decreased 0.6 (95%CI: -1.1, -0.2)

A decrease of menstrual cycle has also been reported by Ouyang et al (2005). In a cross sectional study conducted among 466 newly married, non-smoking, nulliparous female Chinese textile workers (age 20-34 years), the authors reported that relative to the lowest serum DDT quartile, those in the highest quartile had higher odds to experience a short cycle in the previous year: adjusted OR=2.78 (95% CI:0,99-1.36). Also, women in the highest serum DDT had a lower mean age at menarche (-1.11 years) compared to those in the lowest quartile. In a subset (n=287) of the same cohort, Perry et al. (2006) found that serum DDE was associated with a decrease of estrogen (p=0.03) and progesterone (p=0.03) in the periovulation and luteal phase of

the menstrual cycle, respectively. Chen et al. (2005), in their study of 47 Chinese women (age 20-24 years) did not find an association of DDE and DDT with length of the menstrual cycle.

Menstrual cycle irregularity slightly increased in a study of 2314 pregnant women in the USA, serum DDE (mean = 30 µg/L), whereas no association was found with cycle length (Cooper et al. 2005). Vasiliu et al (2004) reported, in a group of 151 women of a Michigan (USA) angler cohort, that an increase in the *in utero* DDE exposure of 15 µg/L reduced the age at menarche by 1 year. However, after adjustment by body size at menarche, the DDE association was no longer significant.

In a population-based case-control study, consisting of 1407 women (median serum DDE =3 µg/L), carried out in 1993-1996 in the USA, high levels of DDE were associated with early age at menopause (Cooper et. al. 2002). Serum levels of p,p'-DDT, p,p'-DDE, have been associated with a younger age at menopause in a sample of 219 menopausal women participating in the Hispanic Health and Nutrition Examination Survey in 1982-1984 (Akkina et al. 2004). Women with exposure levels in the highest exposure categories (serum p,p'-DDT≥ 6ppb) had an adjusted mean age at menopause on average 5.7 yr earlier than women with serum levels of DDT below the detection limit. Women with serum p,p'-DDE levels greater than 23.6 ppb (highest quintile) had an adjusted mean age at menopause 1.7 yr earlier than women with serum p,p'-DDE levels less than 5.5 ppb (lowest quintile). However, no consistent dose-response effect was apparent across low, medium, and high exposure categories.

Semen quality

Several studies have been conducted to assess the relation between DDT and male reproduction parameters. Overall, the available epidemiological evidence tends to suggest that if such relation exists, it is found in populations exposed at high DDT concentrations. In fact, whereas an association between higher serum DDE concentrations and decreased male reproduction parameters was found in studies conducted in malaria endemic–areas where until recently, DDT was sprayed, such associations were not found in studies conducted in populations where DDE/DDT DDT concentrations were lower.

Non-occupational DDT exposure was associated with impaired seminal parameters in a cross-sectional study of 311 healthy male subjects (age: mean and SD 23±5; range 18-40) from the Limpopo Province, South Africa; an endemic malaria area in which DDT is sprayed annually. The mean p,p'-DDT and p,p'-DDE concentrations were 90.23 µg/g lipid (±102.4) and 215.47 µg/g lipid (±210.6), respectively (Aneck-Hahn et al., 2007). The multivariate linear regression analyses indicated that mean Hamilton Thorne Computer Assisted Sperm Analysis (CASA) motility was statistical significantly lower with a higher *p,p*'-DDE concentration and the CASA parameter beat cross-frequency (BCF) was significantly higher with a higher *p,p*'-DDT concentration. There was also a statistically significant positive association between percent sperm with cytoplasmic droplets and *p,p*'-DDT concentration. The ejaculate volume (mean 1.9+/-1.33 mL) was lower than the normal range (≥2.0 mL) according to WHO, and decreased significantly with increased p,p'-DDE values.

Ayotte et al. (2001), in a small study comprising 24 young men (mean age = 21 years; range =16–28) from a DDT-sprayed area in Mexico, found that *p,p*-DDE concentration was inversely

correlated to both semen volume and sperm count. The mean concentration of *p,p* '-DDE in serum lipids was 77.9 µg/g (range = 17.0–177.2). Also, they found that *p,p* '-DDE concentration was positively correlated to serum sex hormone-binding globulin (SHBG) and negatively correlated to the bio-available/total testosterone ratio.

A cross-sectional study of 47 workers, near the Malaria Control Center in the Limpopo Province, South Africa, found an inconsistent association between DDT exposure (median p,p'-DDE= 52 µg/g serum lipid; median p,p'-DDT= 26.9 µg/g serum lipid; median total DDT= 83.3 µg/g serum lipid) and semen , sexual function and fertility outcome (Dalvie et al. 2004a, 2004b). In the multivariate analysis, sperm count was negatively associated with p'p'-DDT (p=0.04). In the bivariate analysis, sperm density was positively associated with p'p'-DDE, however this association disappeared in the multivariate analysis. An inconsistent association was also found with sex hormones such as estradiol and testosterone. No association with DDT and serum sex hormone-binding globulin (Dalvie et al. 2004c).

In a cross-sectional study, nonoccupational exposure to DDT associated with poorer semen parameters in men has been reported by De Jager et al. (2006). The authors investigated semen parameters in 116 men aged 27 years (SD = 8.2) living in malaria endemic-areas in Chiapas (Mexico), where DDT was sprayed until 2000. The mean p,p'-DDE concentration adjusted for total lipids was 45 µg/g (SD= 32). Crude regression analysis showed that several sperm motion parameters, including the percentage of motile sperm, decreased with higher p,p'-DDE concentrations (beta = -8.38; P = .05 for squared motility), and the percentage of sperm with morphological tail defects increased with higher plasma p,p'-DDE concentration (beta = 0.003; P

= .017). Insufficient sperm chromatin condensation was also observed in 46.6% of participants, and the most severe category of incomplete DNA condensation was also positively correlated with p,p'-DDE concentration (r =.223; P = .044). It is worthwhile to note that the mean DDE concentration reported in De Jager study is 100-fold higher than those studies that found no association of DDE and semen parameters.

In a case-control study conducted by Charlier and Foidart (2005) no association was found between male sperm parameters (sperm concentration, motility, and morphology) and serum p,p'-DDE (mean ± SD: cases = 1.05 ± 0.55 µg/g lipid; controls = 0.98 ± 0.53 µg/g lipid). The control group was composed by 73 young men considered as fertile, based on semen analysis, whereas the case group was composed by 82 men classified as subfertile or unfertile. In a subset, where the serum of the mother were obtained (23 mothers in the control group and 19 in the case group), a significantly higher p,p'-DDE serum was found in the mothers of the case group (mean ± SD= 2.45± 1.65 µg/g lipid) compared to the mothers of the control group(mean ± SD = 0.77 ± 0.45 µg/g lipid).

In a cross-sectional study of 149 Swedish fishermen, Tiido et al. (2005) found a moderate positive association between serum level of p,p'-DDE [median (range) =242 ng/g lipid (40.4-2251) and the proportion of Y-chromosome bearing sperms. However, no association was found when the study was extended to include other three different population cohorts (Tiido et al.,2006).

Spanò et al. (2005) found no significant association between sperm chromatin structure assay (SCSA)-derived parameters and p, p'-DDE serum concentrations in a cross-sectional study involving 707 adult males (193 Inuits from Greenland, 178 Swedish fishermen, 141 men from Warsaw, Poland, and 195 men from Kharkiv, Ukraine). The median concentration of p,p'-DDE in the entire group was 560 ng/g lipid (range = 6–13000).

Hauser et al (2003a) investigated 212 male partners of sub-fertile couples who presented to the Massachusetts General Hospital Andrology Laboratory. They reported no statistically significant associations between DNA integrity in sperm and p,p'-DDE. The median p,p'-DDE concentration was 220 ng/g lipid (range 72.5–7776). No significant association between p,p'-DDE and sperm motility was found in a separate report (Hauser et al 2003b).

No significant associations between p,p'-DDE and sperm motility was found in a study conducted in 195 Swedish fishermen, aged 24-65 years (Rignell-Hydbom et al. 2004). The subjects had a median p,p'-DDE serum level of 240 ng/g lipid (range 80-887 ng/g lipid). In a subset of 176 fishermen the authors assessed the sperm DNA/chromatin integrity. No association was found between DNA fragmentation index and p,p'-DDE (Rignell-Hydbom et al. 2005).

Several reports have specifically addressed serum hormone levels in relation to p,p'-DDE serum levels. In a US study of 137 men, North Carolina African-American farmers with median serum levels of p,p'-DDE of 1.2 µg/g lipid (range= 0.56-2.14µg/g lipid), a 23% decrease in total testosterone and free androgen index was reported among subjects in the top tenth percentile of serum levels of p,p'-DDE (Martin et al.2002). Similar results were seen among 24 young

Mexican men, with mean serum DDE levels of 77.9 µg/g lipid (Ayotte et al. 2001).On the other hand, no correlation was observed between levels of serum p,p'-DDE (median = 0.8 µg/g lipid) and levels of plasma hormones (such as follicle-stimulating hormone, luteinizing hormone, prolactin, plasma thyrotropin, free and total T3, free and total T4, and free testosterone) among 110 men who consumed varying amounts of fish from the Baltic Sea (Hagmar et al.2001).

Cocco et al. (2004) explored serum sex hormone levels in relation to past DDT exposure and current p,p'-DDE serum levels (median 0.4 µg/g lipid; range 0.16-1.05 µg/g lipid) among 107 men who participated as young adults in an anti-malarial campaign in the Italian region of Sardinia. Their results suggest that (1) the low current p,p'-DDE serum concentration does not affect serum hormone levels, and (2) past cumulative DDT exposure is not correlated with the current p,p'-DDE serum level, nor does it show persistent effects on serum hormone levels.

Gladen et al. (2004) studied the effect of prenatal DDT exposures to pubertal parameters was in 304 males born in Philadelphia (USA) in the early 1960s. The stored maternal serum samples was used to measure p,p -DDE, p,p -DDT and o,p -DDT The outcomes examined in the boys were height, ratio of sitting height to height, body mass index, triceps skinfold thickness, ratio of subscapular to the sum of triceps and subscapular skinfold thicknesses, skeletal age, serum testosterone, and serum dehydroepiandrosterone sulfate. No associations between prenatal exposure to any of the DDT compounds and any outcome measure were seen.

3.2.2.6 Developmental Effects

Table 3.1, summarizes the published studies of DDT and DDE effects and birth defects.

Two nested case-control study, one nested in a US birth cohort begun in 1959–1966 and using stored maternal serum (Longnecker et al. 2002), and the other nested in a longitudinal cohort of pregnancies that occurred between 1959 and 1967 (Bhatia et al. 2005) do not support an association of DDT or DDE and hypospadias or cryptorchidism. Even though Longnecker et al. (2002) found small, not statistically significant increases in cryptorchidism, hypospadias, and polythelia among boys with the highest maternal levels when compared with those with the lowest maternal levels. The lack of association between DDE and hypospadias (Flores-Leuvano et al. 2003) or cryptorchidism (Damgaard et al. 2006) has also been reported by other groups.

In utero exposure to DDE was not related to reduced androgen action as reflected by ano-genital distance or penile dimensions at birth in a cross-sectional study of 781 newly delivered male infants conducted in 2002-2003 in Chiapas, México (Longnecker et al. 2007). The range of maternal serum DDE levels in the population was large (0.8-398 microg/liter).

More recently, Brucker-Davis et al. (2008) reported a weak correlation between cryptorchid status at birth and DDE in milk ($p=0.05$), but no correlation with DDE levels in cord blood. The prospective case-control study conducted in France consisted of a total of 164 infant/mother pairs (78 cryptorchid and 86 controls). The median maternal milk DDE was 0.119 µg/g lipid for the case group and 0.080 µg/g lipid for the control group.

Several studies have investigated the neurodevelopmental toxicity of DDT and DDE. Rogan et al (1986) reported a dose-dependent relationship between DDE levels in breast milk and hyporeflexia in infants. The same group also reported no association between transplacental or

lactational DDE exposure and child performance on the Bayley Scales of Infant Development at up to 24 months of age (Gladen et al., 1988; Rogan and Gladen, 1991) on the McCarthy Scale at 3, 4, and 5 years of age, or in school at 8 to 10.5 years of age (Gladen and Rogan, 1991). Conversely, investigation of the Oswego cohort found no association of cord blood DDE levels with hyporeflexia (Stewart et al., 2000) or with performance on the Fagan test of infant intelligence (FTII) at 6 and 12 months of age (Darvill et al., 2000). More recently, Eskenazi et al. (2006) evaluated a birth cohort using two neurodevelopmental indices, based on the concentration of DDT in maternal blood collected before birth. That study involved 360 Mexican American children and found a decrease of ~2 points in the psychomotor developmental index score (PDI) with each 10-fold increase in maternal p,p'-DDT blood serum levels when the children reached 6 and 12 months of age (but not at 24 months) and maternal p,p'-DDE levels at 6 months of age only. They also reported no association with mental development at 6 months of age, but a 2 to 3-point decrease in Bayley Mental development index score for maternal p,p'-DDT and o,p'-DDT at 12 and 24 months of age.

Ribas-Fito et al (2003) reported that prenatal exposure to p,p'-DDE measured from cord blood was associated with a delay in mental and psychomotor development of children assessed at 13 months of age.

3.2.2.7 Cancer

Cancer is a chronic disease and can have a latency time of 15–20 years after initiation. Theoretically, DDT could contribute to breast cancer either by being a complete carcinogen, an initiator, or a promoter. Thus, for DDT to cause or contribute to cancer, exposure needs to occur

at a time substantially before the time of diagnosis of the cancer. A common methodological approach in many of the cancer studies reported below has been to assess DDT body burdens at or shortly before the time of breast cancer diagnosis. The drawback of trying to associate current residue levels determined at or near the time of diagnosis with the occurrence of cancer is that levels at diagnosis may be very different than those at the time when cancer began to develop, and this is particularly relevant for cancers that exhibit a long latency. Furthermore, the biological monitoring of DDT presents its own potential for epidemiological bias since serum levels can also be influenced by factors that relate directly to the outcome of interest, in particular, weight change. The weight loss experienced by cancer patients in advanced stages will mobilize the DDT/DDE stored in the adipose tissues and thus increase the serum levels. Therefore, comparison of these subjects with healthy individuals would suggest an association between high levels of DDT and breast cancer even though this association was caused by the disease, rather than being a cause of the disease. This potential bias may be overcome with the use of a nested case-control study design. A nested case-control study depends on the pre-existence of a cohort that has been followed over time. The cohort is assembled in such a way that information on exposure is collected on all subjects at baseline before the occurrence of the disease occurrence, such as blood sample taken and stored. When a case of the outcome of interest is identified, samples of the cohort who have not developed the outcome by that time are selected as controls. The advantage of the nested case-control design is that the most appropriate control group is chosen from members of the same cohort who have not developed the outcome at the time that they are chosen.

These and other issues inherent to epidemiological studies, such as the role of cofounders for example, makes it difficult to draw definite conclusions about exposure to DDT/DDE/DDD and cancer.

Breast cancer

Many epidemiological studies have investigated the association between breast cancer and levels of DDT and DDT-derived compounds in blood or adipose tissue from humans. Some studies have suggested a positive association: a nested case-control study (Wolff et al. 1993); and several case-control studies (Charlier et al. 2003, 2004; Dewailly et al. 1994; Falck et al. 1992; Güttes et al. 1998; Mathur et al. 2002; Romieu et al. 2000; Wasserman et al. 1976). However others studies do not support such an association: several nested case-control studies (Demers et al. 2000; Dorgan et al. 1999; Helzlsouer et al. 1999; Høyer et al. 1998; Hunter et al. 1997; Krieger et al. 1994; Raaschou-Nielsen et al. 2005; Ward et al. 2000; Wolff et al. 2000b); and several case-control studies (Aronson et al. 2000; Gatto et al. 2007; Ibarluzea et al 2004; Laden et al. 2001; Liljegren et al. 1998; Lopez-Carrillo et al. 1997; Mendonca et al. 1999; Moysich et al. 1998; Pavuk et al. 2003; Rubin et al. 2006; Schecter et al. 1997; Siddiqui et al. 2005; Unger et al. 1984; van't Veer et al. 1997; Wolff et al. 2000a; Zheng et al. 1999, 2000). Table 3-2 summarizes the epidemiological studies and their association between DDE levels and human breast cancer.

Blood Serum

Nested case-control studies

Rubin et al. (2006) conducted a retrospective case control study among Alaska native women. They analyzed banked serum collected between 1981 and 1987, from 63 case women who subsequently developed breast cancer and 63 age-matched control women who remained cancer-free. A higher geometric mean for p,p'-DDE levels was found among case women (8.67 ppb; 95% CI: 7.48, 10.04) compared to control women, where the geometric mean was 7.36 ppb (95% CI:6.53, 8.30). Using conditional logistic regression analysis to adjust for potential confounders (e.g., ethnicity, family history of breast cancer, parity), an odds ratio of 1.43 (0.46, 4.47) was found for the highest tertile of DDE exposure, but it was not statistically significant.

Cohn et al. (2007) performed a prospective, nested case-control study to investigate whether DDT exposure in young women during the period of peak DDT use predicts breast cancer. The analyzed banked serum was collected from young women enrolled in the Child Health and Development Studies, Oakland, California who provided blood samples 1-3 days after giving birth (mean age, 26 years) during 1957-1967. Cases (n = 129) developed breast cancer before the age of 50 years. Controls (n = 129) were matched to cases on birth year. Using conditional logistic regression analysis to adjust for potential confounders (e.g., ethnicity, family history of breast cancer, parity), high level of serum p,p'-DDT predicted a statistically significant 5-fold increased risk of breast cancer (OR= 4.2; 95%CI: 1.4-19.1) among women born after 1931. Exposure to p,p'-DDE was not associated with breast cancer risk.

Case-Control Studies

Mathur et al. (2002) found significant higher level of total serum DDT (the sum of DDE, DDD, and DDT) in 135 women diagnosed with breast cancer (3.623±0.497 mg/L) compared to 50 female normal controls (1.332 ±0.239 mg/L) from the Birla Cancer Institute, SMS Hospital, Jaipur, India.

Charlier et al. (2003) conducted a case-control study where blood levels of total DDT (the summary of all DDT and DDE isomers) were compared in 159 women with breast cancer and 250 presumably healthy controls. Breast cancer risk as estimated by crude ORs at 0.5 ppb threshold level of total DDT was significantly higher for breast cancer patients than for controls (OR= 5.36; 95% CI:1.83, 17.51). In the breast cancer group, estrogen receptor status was available for 102 women but was not correlated with the total DDT concentration (r = 0.02, p = 0.88).

Charlier et al. (2004) conducted a hospital case-control study among Caucasian women during the period June 2001–January 2002. Two hundred thirty-one women with breast cancer at the time of surgery and 290 age matched controls were randomly selected in a population of presumably healthy women consulting for routine systematic cervico-vaginal cytological screening. They were evaluated for p,p'-DDT, o,p'-DDE, and p,p'-DDE. Serum levels of organochlorines were corrected for lipid content. The mean level of p,p'-DDE was significantly higher ($p<0.0001$) in women with breast cancer than in control subjects (3.46 ±3.48 ppb (0.58±0.58 mg/g lipid) vs. 1.85 ±2.09 ppb (0.31±0.35 mg/g lipid)). When adjusting for potential confounders such as menopausal status, full-term pregnancies, lactation, use of HRT, and family

history of breast cancer, the association between the presence of p,p'-DDE and breast cancer remained highly significant ($p<0.0001$). Post-menopausal women were at higher risk of developing the disease, whereas the effects of parity and familial history of breast cancer were not significant. When expressing p,p'-DDE in terms of continuous variables, logistic regression analysis revealed that the odds ratios were equal to 1.24 (95% CI: 1.15-1.34).

In a small case-control study, Pavuk et al. (2003) investigated the association between 2,2'-*bis*(4-chlorophenyl)-1,1-dichloroethylene (DDE), 2,2-*bis*(4-chlorophenyl)-1,1,1-trichloroethane (DDT) and risk of breast cancer in an area of high environmental exposure in the Michalovce district of eastern Slovakia. Serum DDE and DDT were measured in 24 incident breast cancer cases diagnosed between May 1997 and May 1999 and in 88 population controls in 1998-1999. Levels of DDE (median: 5745 ng/g of lipid and 3226.5 ng/g of lipid in case and controls, respectively) and DDT (median: 155.1 ng/g of lipid and 107.6 ng/g of lipid in case and controls, respectively) were statistically significantly higher in cases (P=0.04 and 0.03, respectively) compared to controls. However, when the OR was adjusted for several confounders (age, age of menarche, education, alcohol consumption and smoking) there was no significant association of DDT and DDE with increased risk of breast cancer.

Siddiqui et al. (2005) conducted a hospital-based case–control study in India, among 50 women undergoing surgery for breast disease to examine the association between DDT exposure and breast cancer risk. Blood, tumor, and surrounding adipose tissue of the breast were collected from the subjects with benign (control) and malignant breast (study) lesions. Total DDT levels were higher, but not statistically significant, in the blood of the study group (25 cases) than in

those of the controls (25 cases). Total DDT was higher in the tumor and in the breast adipose tissues of the controls than in those of the study group, but they were not statistically different. This study with limited statistical power does not support a positive association between exposure to DDT and risk of breast cancer.

Gatto et al. (2007) did not find any association between serum DDE and breast cancer in a population-based case-control study of African-American women (355 breast cancer case patients, 327 controls).

Adipose Tissues

Ibarluzea et al. (2004) investigated the relation between breast or abdominal adipose tissue concentration of DDE and breast cancer in a hospital- based case-control study in 198 women undergoing surgery for breast cancer and 260 women undergoing non-cancer- related surgery as controls. Although the levels of the geometric mean (geometric SD) of DDE were higher in the cases than in the controls, 3.26.86 (2.78) ng/g of lipid vs. 307.34 (3.62), there were no statistically significant differences between cases and controls. The calculated ORs were not significant.

Recently, Raaschou-Nielsen et al. (2005) examined the association between organochlorines, among them DDE and DDT, in adipose tissues and the development of breast cancer in a nested case-control study of 409 postmenopausal women who developed breast cancer and 409 controls selected from the 29,875 women enrolled in the Danish Diet, Cancer, and Health cohort between 1993 and 1997. The results showed no higher risk of breast cancer among women with higher

levels of any pesticides; the RR associated with the upper quartile of DDE concentration was 0.7 [95% confidence interval (95% CI), 0.5-1.2] contrasting the lower quartile. A pattern of substantially lower risk of estrogen receptor-negative breast cancer in association with higher levels of most of the pesticides: the RR for the higher quartile of DDE was 0.1 (95% CI, 0.0-0.5). The interpretation of the inverse association for estrogen receptor-negative breast cancer is currently unclear.

A meta-analysis of 22 studies (López-Cervantes et al. 2004) calculated the summary OR using the random-effects model and found no evidence of an association between p,p'-DDE body burden levels and breast cancer risk (OR= 0.97; 95% CI: 0.87, 1.09). No significant overall heterogeneity in the OR was found (chi square=27.93; df =23; p=0.218). According to the type of study design the summary OR was 0.91 (95% CI: 0.74, 1.12) for nested case-control studies; 1.11 (95% CI: 0.89, 1.38) for retrospective population based case-control studies; and 0.93 (95%CI: 0.77, 1.12) for retrospective hospital based case-control studies.

Liver cancer

A nested case-control study among participants of the Nutritional Intervention Trials in Linxian, China, was conducted by McGlynn et al. (2006) to study the potential association of DDT and DDE with hepato-carcinogenesis. The case group was composed of 168 individuals who developed liver cancer during the trial, and the control group by 385 individuals, frequency-matched on age and sex who were alive and well at the end of the study. The geometric mean of DDT was 487 ng/g lipid in the case subjects and 490 ng/g lipid in the control subjects. The geometric mean of DDE was 2931 ng/g lipid in the case subjects and 2957 ng/g lipid in the

control subjects. Increased serum DDT concentration was associated with risk of developing liver cancer (OR for quintile 1 versus quintile 5 = 3.8, 95% CI = 1.7 -8.6, p(trend) = 0.002). In contrast, there was not a statistically significant association between liver cancer and serum DDE concentration.

Lymphoma

Cocco et al (2008) did not find evidence of an association between non-Hodgkin's lymphoma (NHL) risk and serum level of DDT and DDE in a study of 174 NHL cases and 203 controls from France, Germany and Spain.

Genital Cancers

Studies investigating an association of DDT with endometrial cancer (Weiderpass et al. 2000) and prostate and testicular cancer (Ritchie et al. 2003) have been inconclusive or do not support an association. Recently, risk of both seminomatous and nonseminomatous testicular germ cell tumors (TGCTs) has been reported to be associated with increase exposure to *p,p*'-DDE (McGlynn et al., 2008). The case-control study investigated prediagnostic serum samples from 754 case subjects and 928 control subjects enrolled in the US Servicemen's Testicular Tumor Environmental and Endocrine Determinants (STEED) for DDT exposure. Subjects in highest serum *p,p*'-DDE quartile (>0.390 µg/g lipid) had an OR (95% CI) of 1.71 (1.23-2.38) to have TGCTs compared to those in the first serum *p,p*'-DDE quartile (≤0.157 µg/g lipid.

Table 3-1 Summary of DDT and DDE effects on reproductive outcomes

Ref	Design and population	DDT or DDE concentrations	Effects
Spontaneous abortion			
Saxena et al. 1981 (in Tox profile)	10 cases and 25 controls in India	Mean DDE 164 µg/L (cases) and 13 µg/L serum (controls)	Raised DDE associated with increased risk of spontaneous abortion
Longnecker et al. 2005	1717 pregnancy women in the USA	Median DDE 25 µg/L serum	Highest DDE quartile was associated with increased fetal loss in previous pregnancies
Korrick et al. 2001	15 cases and 15 controls in China	Mean DDE 22 µg/L (cases) and 12 µg/L serum (controls)	Raised DDE associated with increased risk of spontaneous abortion
Leoni et al. 1989 (in Tox profile)	120 cases and 120 controls in Italy	Mean DDE 5·2 µg/L (cases) and 4·6 µg/L serum (controls)	No associations recorded
Gerhard et al.1998 (in Tox profile)	89 women with repeated miscarriages in Germany	Mean DDE 1·2 µg/L serum	14% of cases with DDE higher than range of previously investigated reference population
Sugiura-Ogasawara et al.	45 cases and 30 controls in Japan	Mean DDE 0·7 µg/L (cases) and 0·9 µg/L serum	No associations recorded

Table 3-1 Summary of DDT and DDE effects on reproductive outcomes

Ref	Design and population	DDT or DDE concentrations	Effects
2003		(controls)	
Salazar-Garcia et al. 2004	Paternal occupational DDT exposure (n=2003) during anti-malaria campaign in Mexico. Information about 9817 pregnancies	Paternal Exposure: Mean±SD adipose tissue DDE = 61±93µg/g fat.	No significant association for spontaneous abortion or sex ratio. High occupational exposure to DDE associate with an increased risk of birth defects
Venners et al. 2005	Prospective study of 388 newly married, non-smoking, Chinese textile workers between 1996-1998	Preconception serum total DDT: Median = 0.028 µg/g; Range = 0.006-0.113 µg/g.	The odds of early pregnancy losses were increased among those in the highest tertile compared to those in lowest tertile. Also there was a linear trend of increasing odds of early pregnancy losses with increasing serum total DDT concentration
Preterm delivery			
Torres-Arreola et al.2003	100 preterm cases and 133 full-term controls in Mexico	Median DDE 0.19 µg/g (cases) and 0.15 µg/g serum lipid (controls)	Raised maternal DDE was not associated with a statistical significant increased risk of preterm delivery

Table 3-1 Summary of DDT and DDE effects on reproductive outcomes

Ref	Design and population	DDT or DDE concentrations	Effects
Saxena et al. 1981 (in Tox profile)	15 preterm cases and 25 full-term controls in India	Mean DDE 58 µg/L (cases) and 13 µg/L serum (controls)	Cases had higher maternal DDE concentrations than did controls
Farhang et al. 2005	420 pregnant women in USA during 1959–1967	Maternal preconception (n=86)or postpartum (n=334) median p,p′-DDE 43 µg/L serum, p,p′-DDT 11 µg/L serum	Raised maternal DDE was not associated with a statistical significant increased risk of preterm delivery
Lognecker et al. 2001 (in Tox profile)	2613 pregnant women in the USA	Median DDE 25 µg/L serum	Raised maternal DDE associated with increased risk of preterm delivery
Berkowitz et al. 2001 (in Tox profile)	20 preterm cases and 20 full-term controls in the USA	Median DDE 1.3 µg/L (cases) and 1.4 µg/L serum (controls)	No association recorded
Birth weight			
Gladen et al. 2003	197 singleton infants in	Median DDE= 2.5 µg/g	No association between DDE and birth weight after

Table 3-1 Summary of DDT and DDE effects on reproductive outcomes

Ref	Design and population	DDT or DDE concentrations	Effects
	Ukraine	breastmilk fat	adjustment for potential confounders
Fenster et al. 2006	birth cohort of 385 low-income Latinas living in the Salinas Valley, California USA	Maternal serum p.p'-DDE: Median = 1.00 µg/g lipid; Range = 0.049-159.3 µg/g lipid	No association between maternal DDE and birth weight
Farhang et al. 2005	420 offspring of pregnant women from the Child Health and Development Studies cohort, USA during 1959–1967	Maternal preconception (n=86)or postpartum (n=334) median p,p′-DDE= 43 µg/L serum. Median p,p′-DDT =11 µg/L serum	No association between DDE and birth weight. No increased risk of small-for-gestational-age.
Lognecker et al. 2001 (in Tox profile)	2613 pregnant women in the USA	Median DDE 25 µg/L serum	Raised maternal DDE associated with increased risk of small-for-gestational-age
Rogan et al. 1986	912 infants in the USA	Maternal median DDE at birth 13 µg/L serum	Maternal DDE burden not associated with birth weight
Siddiqui et al. 2003	30 intrauterine growth restriction cases and 24	Mean DDE 9 µg/L (cases)	Raised maternal DDE associated with increased risk of

Table 3-1 Summary of DDT and DDE effects on reproductive outcomes

Ref	Design and population	DDT or DDE concentrations	Effects
	controls in India	and 6 µg/L serum (controls)	intrauterine growth restriction
Jusko et al. 2005	399 offspring of pregnant women from the Child Health and Development Studies cohort, USA during 1959–1967	Maternal serum p.p'-DDE: Mean±SD = 6.58±4.80µg/g lipid. Median = 5.88 µg/g lipid.	No association between maternal DDE and impaired growth of the offspring at child birth and at 5 years of age.
Bjerregaard et al. 2000	178 newborn babies in Greenland	Maternal mean DDE = 5 µg/L plasma	No association with birth weight
Karmaus et al. 2004	168 offspring of anglers in the Great Lakes Fish Eater Study cohort,USA	Maternal Exposure serum DDE: Range >5 µg -, ≥25 µg	No association between DDE and birth weight.
Weisskopf et al. 2005	119 frequent fish eaters and 24 infrequent fish eaters in the USA	Median DDE =2 µg/L (frequent eaters) and 1 µg/L ser um (infrequent eaters)	Natural log of maternal serum DDE inversely associated with birth weight
Sagiv et al. 2007	birth cohort of 722 women in USA	Maternal serum p.p'-DDE: Range from 0- 14.93 ng/g	No association between maternal DDE and birth weight, and other measures of infant birth size.

Table 3-1 Summary of DDT and DDE effects on reproductive outcomes

Ref	Design and population	DDT or DDE concentrations	Effects
Time to pregnancy			
Cohn et al. 2003	289 women (age 28-31 years) born in the early 1960s in the USA	Maternal postpartum median p,p′-DDE 48 µg/L serum, p,p′-DDT 13 µg/L serum	DDE associated with raised probability of pregnancy, and DDT associated with reduced probability of pregnancy
Cocco et al. 2005	Spouses of 105 men occupational DDT-exposed during the 1946-1950 anti-malaria campaign in Sardinia, Italy	Work history	Slightly increased stillbirth rate; reduced male-to-female ratio among offspring and probability of pregnancy in DDT users
Law et al. 2005	390 pregnant women from 1959-1965 in USA.	Median serum p,p'-DDE:= 22.6 µg/L	Slightly, but not statistical significant, time to pregnancy increase in subjects in the highest DDE quartile compared to those on the lowest DDE quartile.
Duration of lactation			

Table 3-1 Summary of DDT and DDE effects on reproductive outcomes

Ref	Design and population	DDT or DDE concentrations	Effects
Gladen et al. 1995	229 postpartum women in Mexico	Median DDE 6 µg/g breastmilk fat	Raised DDE associated with reduced duration of lactation
Rogan et al. 1987	858 postpartum women in the USA	Median DDE 2 µg/g breastmilk fat	Raised DDE associated with reduced duration of lactation
Karmaus et al. 2005	176 pregnancies of 91 mothers in the USA who had maternal exposure information and gave birth between 1969 and 1995.	The extrapolated maternal DDE serum concentrations were categorized as follows: 0 to < 5 µg/L, 5 to < 10 µg/L, ≥ 10 µg/L.	Raised extrapolated DDE in non-smoking mothers associated with reduced duration of lactation.
Menstrual cycle			
Akkina et al. 2004	219 Hispanic women in the USA	Mean p,p′-DDE 36 µg/L serum	Highest DDT exposure levels associated with 5.7 years early age at menopause. High DDE concentration associated with early age (1.7 years) at menopause compared to the lowest DDE exposure levels.
Cooper et al. 2005	2314 pregnant women in the	Mean DDE 30 µg/L	Menstrual cycle irregularity slightly increased, no

Table 3-1 Summary of DDT and DDE effects on reproductive outcomes

Ref	Design and population	DDT or DDE concentrations	Effects
	USA		association with cycle length and bleeding duration
Windham et al. 2005	50 Laotian immigrants coming to the USA	Mean±SD serum DDT=1.77±3.84 µg/L; Mean±SD serum DDE =20.8±22.9 µg/L;	Highest quartile of DDE and DDT associated with reduced mean luteal phase length (by 1.5 days) and decreased progesterone during luteal phase
Vasiliu et al. 2004	151 offspring of anglers in the USA	Maternal DDE range 0–17 µg/L serum	An increase in the *in utero* DDE exposure of 15 µg/L reduces age at menarche by 1 year. When adjusted by body size at menarche, the DDE association was no longer significant.
Cooper et al. 2002	1407 women in a breast cancer case-control study in North Carolina, USA	Median DDE 3 µg/L plasma	High DDE associated with early age at menopause
Ouyang et al. 2005	446 newly married, non-smoking, nulliparous Chinese textile workers (age 20-34 years).	Mean±SD serum total DDT =0.032±0.0188 µg/g; Mean±SD serum p.p'-DDE = 0.03±0.017 µg/g;	Highest quartile serum total DDT associated with short cycle and reduced age at menarche by 1.1 year compared to lowest DDT quartile.

Table 3-1 Summary of DDT and DDE effects on reproductive outcomes

Ref	Design and population	DDT or DDE concentrations	Effects
Perry et al. 2006	287 newly married, non-smoking, nulliparous Chinese textile workers (Subset of the cohort of Ouyang et al. 2005)	Serum p,p'-DDE: Median = 0.027 µg/g; Range = 0.005-0.098 µg/g	Serum DDE associated with a decrease of estrogen and progesterone in the periovulation and luteal phase of the cycle, respectively.
Chen et al. 2005	47 Chinese women (age 20-24 years) divided by location: Urban (n=16); Suburban (n=18) and; Rural (n=13)	Serum p,p'-DDE Mean±SD : Urban 13.1±9.0 µg/L; Suburban 17.9±10.6 µg/L; Rural42.3±15.5 µg/L;	No association of p.p'-DDT and p.p'-DDE with length of menstrual cycle, durations of menses or heaviness of menstrual flow.
Toft et al. 2005	Cross-sectional multicenter study of 1049 women (454 Inuit from Greenland, 463 Swedish fishermen's wives, 203 Poland women, and 374 Ukraine women)	Mean serum p,p'-DDE: Swedish cohort =2.15 µg/g lipid; Greenland cohort = 0.44µg/g lipid; Poland cohort = 0.43 µg/g lipid; Ukraine cohort = 0.80 µg/g lipid	Within population, increased risk of longer cycles among Polish women exposed to high level of DDE. Conversely, high levels of DDE were protective against long menstrual cycles in the Greenland cohort.

Table 3-1 Summary of DDT and DDE effects on reproductive outcomes

Ref	Design and population	DDT or DDE concentrations	Effects
Semen quality			
Ayotte et al. 2001	24 young men (mean age = 21 years; range 16-28 years) from DDT-sprayed area in Mexico	Serum p,p'-DDE: mean = 78 µg/g lipid; range =17.0-177.2 µg/g lipid	DDE amounts: 1) inversely correlated with semen volume, sperm count, and testosterone concentration, and; 2) positively correlated with sex-hormone-binding globulin.
Dalvie et al. 2004a and Dalvie et al. 2004b	47 malaria workers in South Africa (mean age 45 years)	Mean p,p′-DDE 52 µg/g serum lipid	No consistent association with semen quality and sex hormones (estradiol, and testosterone) levels. No association with sex-hormone-binding globulin.
Martin et al. 2002	137 North Carolina Africa-American farmer men (mean age 62 years)	Serum p,p'-DDE: median =1.2 µg/g lipid; range = 0.56-2.14 µg/g lipid	Subjects in the top tenth percentile of DDE associated with decreased testosterone and free androgen index.
Hagmar et al. 2001	110 Baltic sea fish eaters (age range 23–79 years)	Serum p,p'-DDE: Median p0.8 µg/g lipid	No correlation was between levels of serum p,p'-DDE (median = 1.2 µg/g lipid) and levels of plasma hormones (such as follicle-stimulating hormone, luteinizing hormone, prolactin, plasma thyrotropin, free and total T3, free and total T4, and free testosterone)

Table 3-1 Summary of DDT and DDE effects on reproductive outcomes

Ref	Design and population	DDT or DDE concentrations	Effects
Cocco et al. 2004	107 previous malaria workers in Italy (mean age 78 years)	Serum p,p'-DDE: median =0,4 µg/g lipid; range 0.16-1.05µg/g lipid	No association with sex hormone levels (such as estradiol, testosterone, luteinizing hormone, follicle-stimulating hormone, and sex-hormone-binding globulin)
Hauser et al. 2003a and Hauser et al. 2003b	212 male partners of sub-fertile couples in the USA (mean age 37 years)	Serum p,p'-DDE: median =0.24 µg/g lipid; range =0.07-7.78 µg/g lipid	No association with sperm concentration, motility, morphology, and DNA damage
Rignell-Hydbom et al. 2004 and Rignell-Hydbom et al. 2005	195 Swedish fishermen (median age 51 years; range 24-65)	Serum p,p'-DDE: median =0.22 µg/g lipid; range =0.08-0.89 µg/g lipid	No association between sperm motility or sperm DNA fragmentation and DDE.
Charlier and Foidart 2005	Case-control study. Cases: n= 82 men classified as sub-fertile or unfertile. Controls: n=73 men classified as fertile	Serum p,p'-DDE mean ±SD: cases=1.05 ±0.55 µg/g lipid; controls=0.98 ±0.53 µg/g lipid	No association between sperm concentration, motility, and morphology and serum DDE.

Table 3-1 Summary of DDT and DDE effects on reproductive outcomes

Ref	Design and population	DDT or DDE concentrations	Effects
Spanò et al. 2005	Cross-sectional multicenter study of 707 adult men (193 Inuit from Greenland, 178 Swedish fishermen, 141 Poland men, and 195 Ukraine men)	Serum p,p'-DDE: median =0.56 µg/g lipid; range =0.006-13 µg/g lipid	No significant association between sperm chromatin structure assay (SCSA)-derived parameters and serum DDE.
Tiido et al. 2005 and Tiido et al. 2006	149 Swedish fishermen (mean age 47 years); 157 Greenland men (mean age 31 years); 121 Poland men (mean age 30 years); 120 Ukraine men (mean age 26 years).	Median serum p,p'-DDE: Swedish cohort =0.24 µg/g lipid; Greenland cohort = 0.59µg/g lipid; Poland cohort = 0.49 µg/g lipid; Ukraine cohort = 1.00 µg/g lipid	Serum levels of DDE were associated with slightly higher proportion of Y-chromosome bearing sperms in the Swedish cohort only.
Aneck-Hahn et al. 2007	311 male subjects in South Africa (mean age 23 years; range= 18-40 years)	Mean±SD serum p,p′-DDE = 215.47±210.6 µg/g serum lipid Mean±SD serum p,p′-DDT = 90.23±102.4 µg/g serum lipid	Mean CASA motility was statistical significantly lower with a higher *p,p*'-DDE concentration. CASA parameter beat cross-frequency (BCF) was significantly higher with a higher *p,p*'-DDT concentration. Significant positive association between percent sperm with cytoplasmic droplets and *p,p*'-DDT. The ejaculate volume decreased significantly with increased p,p'-

Table 3-1 Summary of DDT and DDE effects on reproductive outcomes

Ref	Design and population	DDT or DDE concentrations	Effects
			DDE values
De Jager et al. 2006	116 men (mean ± SD age = 27 ±8.2 years from DDT-sprayed area in Chiapas, Mexico	serum p,p′-DDE: mean ± SD = 45±32 µg/g lipid; median=41 µg/g lipid	Higher serum p,p'-DDE levels were associated with decreased sperm motility, increased sperm morphological tail. Incomplete sperm DNA condensation was, also, positively correlated with p,p'-DDE concentration.
Birth defects			
Longnecker et al. 2007	Cross-sectional study of 781 male newborns in Chiapas, Mexico during 2002-2003	Maternal serum DDE : Median (Range)= 2.7 (0.1-56.1) µg/g lipid	No association between DDE and anogenital distance or penile dimensions at birth.
Flores-Luèvano et al.,2003	Case –control study. 41 hypospadias, and 28 control babies in Mexico.	Maternal serum DDE, mean±SD: Cases=1.2±1.69µg/g lipid; Controls= 1.4±1.32µg/g lipid	No association between DDE and hypospadia.
Brucker-Davis et al. 2008	Prospective case-control study. A total of 164	Median (range) cord blood DDE : Cases = 0.2 (0.1-0.4)	Weak correlations between cryptorchid status at birth and DDE in milk (p=0.05). No correlation between

Table 3-1 Summary of DDT and DDE effects on reproductive outcomes

Ref	Design and population	DDT or DDE concentrations	Effects
	infant/mother pairs (78 cryptorchid and 86 controls). One hundred and fifty-one cord bloods (67 cryptorchid, 84 matched controls) and 125 colostrums (56 for cryptorchid and 69 for controls)	µg/L ; Controls = 0.2 (0.1-2.6) µg/L. Median (range) maternal milk DDE : Cases = 0.119 (0.024-2.165) µg/g lipid; Controls = 0.080 (0.003-1.031) µg/g lipid..	cryptorchid status at birth and DDE in cord blood.
Damgaard et al. 2006	Case–control study from a prospective, longitudinal birth cohort study in Finland and Denmark. 62 milk samples from mothers of cryptorchid boys and 68 from mothers of healthy boys.	Maternal breast milk DDE, Median (Range): Cases = 0.097 (0.022-0.428) µg/g lipid; Controls = 0.084 (0.019-0.377) µg/g lipid.	No association between DDE and cryptorchidism.
Bhatia et al.2005	75 cryptorchidism, 66 hypospadias and 283 control babies in the USA	Median DDE 43 µg/L (cryptorchidism and controls) and 41 µg/L (hypospadias)	No association between DDE and hypospadia or cryptorchidism.
Longnecker et al. 2002	219 cryptorchidism, 199 hypospadias, 167 polythelia,	Median DDE 24 µg/L (cryptorchidism,	Not statistically significant increases in for the investigated birth defects.

Table 3-1 Summary of DDT and DDE effects on reproductive outcomes

Ref	Design and population	DDT or DDE concentrations	Effects
	and 552 control babies in the USA	hypospadias, and controls) and 32 μg/L (polythelia)	
Salazar-Garcia et al. 2004	Paternal occupational DDT exposure (n=2003) during anti-malaria campaign in Mexico. Information about 9817 pregnancies	Paternal Exposure: Mean±SD adipose tissue DDE = 61±93μg/g fat.	High occupational exposure to DDE associate with an increased risk of birth defects

Rows are in order of decreasing serum DDE dose.

Table 3-2. Epidemiological studies on DDE and human breast cancer risk.

Author, year	Tissue	Cases/ controls	Study Populations	DDE concentration, mean(SD) Cases	Controls (units)	OR (95%CI)[a]
Nested Case-Control Studies						
Wolff (1993)	S	58/171	NY Women Health Study	11.8(9.1)	7.7(2.39) ng/mL	3.68 (1.01-13.50)
Krieger(1994)	S	150/150	Medical Care Program	43.3(25.9)	43.1(23.7) ppb	1.33 (0.68-2.2)
Hunter (1997)	S	236/236	Nurses' Health Study	6.01(4.56)[c]	6.97(5.99) ppb[c]	0.72 (0.37-1.40)
Høyer (1998)	S	240/477	Copenhagen City Heart Study	Not provided		0.88 (0.56-1.37)
Helzlsouer(1999)	S (1974 cohort)	235/235	CLUE I	1,699(929)[b]	1,920(1,409)ng/g[b]	0.73 (0.40-1.32)
	S (1989 cohort)	105/105	CLUE II	1,311(1,037)[b]	1,586(1,557)ng/g[b]	0.58 (0.29-1.17)
Dorgan (1999)	S	105/208	Breast Cancer Serum Bank	NA	(Median)16.3ng/mL	0.8 (0.4-1.5)
Wolff (2000)	S	110/213	NY Women Health Study	977(2.46)[b]	1,097(2.29)ng/g [b]	1.30 (0.51-3.35)
Raaschou-Nielsen (2005)	A	409/409	Danish Diet, Cancer, and Health cohort	639	686.3 µg/kg	0.7 (0.5-1.2)[d]
Rubin (2006)	S	63/63	Alaskan Native Women Serum Bank	8.67	7.36 ppb	1.43 (0.46-4.47)

Author, year	Tissue	Cases/ Controls	Type of Control	DDE concentration, mean(SD) Cases	Controls (units)	OR (95%CI)[a]
Cohn (2007)	S	129/129	CHDS, California, USA	5.1 µg/g p.p'-DDT=1.4 µg/g		0.9 (0.3-3.0) 5.52 (1.4-19.1)
Case-Control Studies						
Schecter(1997)	S	20/20	BBD	12.17(2.41)	16.67(4.14)ng/mL	1.14 (0.23-5.68)
Lopez-Carrillo(1997)	S	141/141	HC	562.5(676.2)[b]	505.5(567.2)ppb[b]	0.76 (0.41-1.42)
van't Veer(1997)	A	347/374	HC, CC	(median)1.35	1.51 µg/g	0.48 (0.25-0.95)
Güttes (1998)	A	45/20	BBD	805	496 µg/kg	p = 0.017
Liljegren (1998)	A	35/43	BBD	767	1,026 ng/g	0.4 (0.1-1.2)
Moysich(1998)	S	154/192	CC	11.47(10.49)[b]	10.77(10.64)ng/g[b]	1.34 (0.71-2.55)
Olaya-Contreras (1998)	S	153/153	HC	3.30(4.12)	2.50(3.60)ng/mL	1.95 (1.10-3.52)
Mendonca (1999)	S	151/306	CC	3.1	4.8 ng/mL	0.83 (0.40-1.6)
Dello Iacovo(1999)	S	170/190	CC	9.55(5.42)	8.98(5.17)ng/mL	1.24 (0.70-2.20)
Zheng (1999)	A	304/186	BBD	736.5	736.5ppb	0.9 (0.5-1.5)

Author, year	Tissue	Cases/ Controls	Type of Control	DDE concentration, mean(SD) Cases	Controls (units)	OR (95%CI)[a]
Zheng (2000)	S	475/502	BBD	460.1[b]	456.2ppb[b]	0.96 (0.67-1.36)
Romieu (2000)	S	120/126	CC	3.84(5.98)[b]	2.52(1.97)µg/g[b]	3.81 (1.14-12.80)
Mathur (2002)	S	135/55	HC	0.862(0.154)[e]	0.047(0.018) [e]	p < 0.05
Pavuk (2003)	S	24/88	CC	(median)5745[b]	3226.5 ng/g [b]	3.04 (0.65-14.3)
Charlier (2003)	S	159/250	HC	3.94(3.88)[f]	1.83(1.98)pbb [f]	5.36 (1.83-17.51)
Charlier (2004)	S	231/290	HC	3.46(3.48)[b]	1.85(2.09)pbb[b]	1.24 (1.15-1.34)
Ibarlueza (2004)	A	198/260	HC	326.86(2.78)[g]	307.34(3.62) ng/g [b,g]	1.22 (0.68-2.21)
Siddiqui (2005)	S	25/25	BBD	11.69(3.29)	20.66(8.24)pbb	p > 0.05
Gatto (2007)	S	355/327	CC	1.40 ± 1.54	1.25 ± 1.26 µg/g [b]	1.02 (0.61-1.72)

Abbreviations: BBD, benign breast disease controls; CC, community or population controls; CLUE I, Campaign against Cancer and Stroke(Washington County, MD USA); CLUE II: Campaign against Cancer and Heart Disease(Washington County, MD USA); HC, hospital controls (hospitalized for non-breast disease and non-cancer related conditions unless otherwise noted); PM, postmortem autopsy controls; SD, standard deviation; [a] = OR adjusted for potential confounding factors, comparison between highest and lowest levels of DDE (i.e., tertile, quartile, or quintile); [b] = Serum DDE adjusted for lipid concentration; [c] = Plasma DDE adjusted for cholesterol; [d] = Relative Risk; [e] = (Standard Error); [f] = Total DDT (sum of DDT, DDD and DDE); [g] = Geometric Mean (Geometric SD)

4.0 CHEMICAL AND PHYSICAL INFORMATION

NO DATA.

5.0 PRODUCTION, IMPORT/EXPORT, USE, DISPOSAL

NO DATA.

6.0 POTENTIAL FOR HUMAN EXPOSURE

6.5 General Population and Occupational Exposure

The human Acceptable Daily Intake (ADI) level for ΣDDT is 20 µg/kg body weight (µg/kg/bw) established by the FAO/WHO in 1984 (Coulston,1985). Based on this level, as well as more recent studies, the Provisional Tolerable Daily Intake (PTDI) for ΣDDT was set at 10 µg/kg/bw (FAO, 2005) in 2000 by the JMPR. More recently, the Maximum Residue Limit (MRL) set by Codex Alimentarius for extraneously-derived ΣDDT in milk was set at 20 µg/kg wm (FAO, 2005). Serum levels of p,p'-DDE, p,p'-DDT and o,p'-DDT were measured in a subsample of the United States National Health and Nutrition Examination Survey (NHANES) participants aged 12 years and older. The results from the continuous NHANES 1999-2000 and NHANES 2001-2002 were reported in the Third National Report on Human Exposure to Environmental Chemicals (CDC, 2005). In the NHANES 2001-2002 subsample the geometric mean serum level of p,p'-DDE of 1.81 ng/g (whole weight) was similar to the measurement reported in a previous study (geometric mean serum p,p'-DDE = 1.6 µg/l) conducted in adult participants of the Germany Environmental Survey 1998 (Becker et al., 2002). Figure 6.1 shows the geometric mean (GM) and the standard error of the GM for serum levels of p,p'-DDE (lipid adjusted) by gender, age group and race/ethnicity in subsample of NHANES participants aged 12 years and older conducted during the years 1999-2000, 2001-2002, and 2003-2004. The data for the NHANES 2003-2004 subsamples

were released in the public domain in April 2008 and can be accessed at the CDC website (CDC, NCHS, NHANES 2003-2004. **Lab 28 Organochlorine Pesticides [Data, Docs])**

In NHANES 2003-2004 the geometric mean levels of p,p'-DDE (lipid adjusted) was lower than the previous NHANES 2001-2002 subsamples. The decline which was found in both gender, age group and race/ethnicity, was more accentuated in the Mexican American group, where the serum level of p,p'-DDE dropped from 652 ng/g of lipid to 444 ng/g of lipid. This remarkable decline may be attributed to the Mexico's decision to phase out and discontinue the use of DDT for malaria vector control, replacing it with synthetic pyrethroids, as part of the North American Agreement on Environmental Cooperation, a side accord to the North American Free Trade Agreement (NAFTA) signed in 1994. By 2000, Mexico was able to achieve an early elimination of DDT use and production (Chanon et al. 2003).

Levels of DDE and DDT in human milk, blood, and tissue appear in Table 6-1.

Figure 6-1 Geometric Mean and Standard Error p.p'-DDE (lipid adjusted) for the U.S. population aged 12 years and older, National Health and Nutrition Examination Survey, 1999-2004

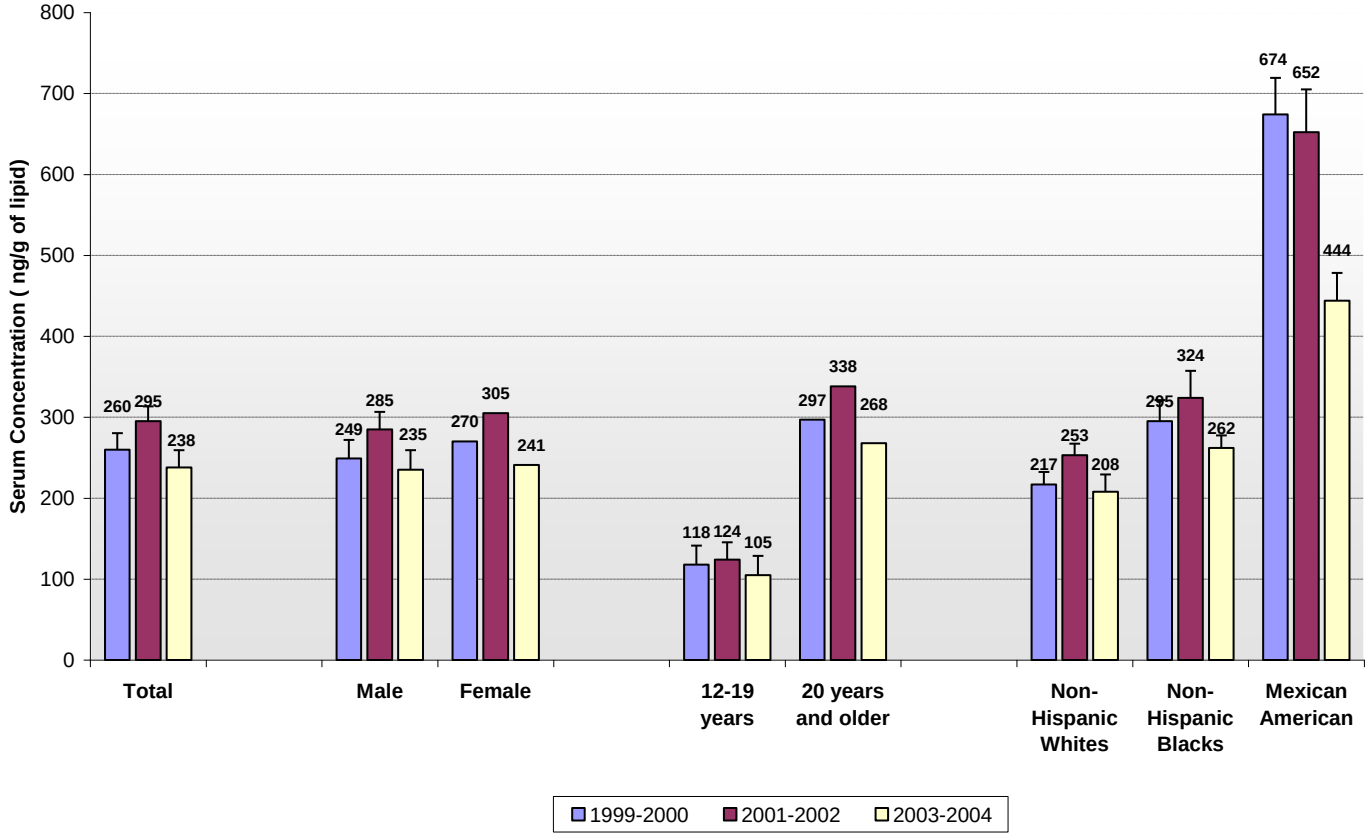

Table 6-1. Levels of DDT Compounds in Human Milk, Blood, and Tissues — Recent Studies (2002- May 2008)
(continuation of Table 6-4, Toxicological Profile for DDT/DDE/DDD, 2002)

DDT compound	Population	Tissue	Mean[a] ± Standard Deviation concentration	Units as reported	Reference
Milk					
p,*p*'-DDT	Mothers in Saudi Arabia	Breast Milk	0.183 ± 1.16	µg/g lipid	al-Saleh I et al. 2003
p,*p*'-DDT	Mothers (n=54) in United Kingdom, 2001-2003	Breast Milk	Median = 6.2; Range= 1.1 -760	ng/g lipid	Kalantzi et al. 2004
p,*p*'-DDT	Mothers in Hanoi (n=42) and Hochiminh (n=44), northeastern China, 2000-2001	Breast Milk	Hanoi: Mean= 170; (Range = 34-6900) Hochiminh: Mean=265; (Range = 100-1000)	ng/g lipid	Minh et al. 2004
p,*p*'-DDT	Mothers (n=36) in Taiwan	Breast Milk	23 ± 14	ng/g lipid	Chao et al. 2006
p,*p*'-DDT	Mothers (Primiparae: n=14; Secundiparae: n=13)in Poland	Breast Milk	Primiparae; 76.6 ± 24.8 Secundiparae; 81.0 ± 36.2	ng/g lipid	Szyrwińska and Lulek 2007
p,*p*'-DDE	Mothers in Saudi Arabia	Breast Milk	0.62 ± 1.53	µg/g lipid	al-Saleh I et al. 2003
p,*p*'-DDE	Mothers (n=54) in United Kingdom, 2001-2003	Breast Milk	Median = 150; Range= 22-1600	ng/g lipid	Kalantzi et al. 2004
p,*p*'-DDE	Mothers in Hanoi (n=42) and Hochiminh (n=44),	Breast Milk	Hanoi: Mean= 1900; (Range = 420-6300) Hochiminh: Mean=2000; (Range = 340-	ng/g lipid	Minh et al. 2004

	Northeastern China, 2000-2001		16000)		
p,p'-DDE	Primiparae mothers in Dalian (n=20) and Shenyang (n=20), northeastern China, 2002	Breast Milk	Dalian: 2000 ± 1200 Shenyang: 830 ± 730	ng/g lipid	Kunisue et al. 2004b
p,p'-DDE	Birth cohort including 92 mother-infant pairs in Spain., 1997- 1999	Colostrum (µg/g) Mature milk (µg/g)	Median= 1.03; IQ range= 0.49–1.90 Median= 0.80; IQ range= 0.45–1.23	µg/g µg/g	Ribas-Fitó et al. 2005
p,p'-DDE	Mothers (n=36) in Taiwan	Breast Milk	310 ± 270	ng/g lipid	Chao et al. 2006
p,p'-DDE	Mothers in the town of Jozini, Mkuze and Kwaliweni , South Africa	Breast Milk	Jozini Primiparae (*n* = 33): 165.05 ±224.66 Jozini Multiparae (*n* = 52) : 63.82±49.54 Mkuzi Primiparae (*n* = 4): 38.79±46.38 Mkuzi Multiparae (*n* = 22): 28.45±28.96 Kwaliweni Primiparae (*n* = 16):17.69±22.87 Kwaliweni Multiparae (*n* = 25): 17.51±12.65	µg/L	Bouwman et al. 2006
p,p'-DDE	Mothers (Primiparae: n=14; Secundiparae: n=13) in Poland	Breast Milk	Primiparae; 1051.6 ± 464 Secundiparae; 1040 ± 819	ng/g lipid	Szyrwińska and Lulek 2007
p,p'-DDE	Mothers in Denmark (n=43) and Finland (n=43)	Breast Milk	Denmark = 137.01 Finland = 77.26	ng/g lipid	Shen et al. 2007
p,p'-DDE	Mothers in Tunisia	Breast Milk (n=78)	2.421	µg/g lipid	Ennaceur et al. 2007
p,p'-DDE	Mothers in China	Breast Milk (n=50)	GM=10.35; 95%CI: 5.30-20.24	ng/g lipid	Wang et al. 2008

p,p'-DDE	Mothers in Brasil	Breast Milk (n=69)	GM=343.4	ng/g lipid	Azeredo et al. 2008
Σ *p,p*'-DDT	Hmong mothers (n=25) in Northern Thailand	Breast Milk	392.9 ± 441.3	ng/mL milk	Stuetz et al. 2001
Σ *p,p*'-DDT	Mothers (n=103) from eight villages in Siphofaneni area in Swaziland, 1996-1997	Breast Milk	Village with the highest measurement: Mphaphati (n=14) = 3.22 ± 3.75 Village with the lowest measurement: Siphofaneni (n=14)= 0.36 ± 0.43	mg/kg whole milk	Okonkwo and Kampira. 2002
Σ *p,p*'-DDT	Mothers in Saudi Arabia	Breast Milk	0.856 ± 2.64	µg/g lipid	al-Saleh I et al. 2003
Σ *p,p*'-DDT	Mothers in Urban (n=16) and Suburban (n=20) area of Phnom Penh city, Cambodia	Breast Milk	Urban Area: Mean= 1800; (Range = 310-11000) Suburban Area: Mean= 1200; (Range = 300-3800)	ng/g lipid	Kunisue et al. 2004a
Σ *p,p*'-DDT	Mothers (Primiparae: n=14; Secundiparae: n=13)in Poland	Breast Milk	Primiparae; 1195 ± 475 Secundiparae; 1126± 836	ng/g lipid	Szyrwińska and Lulek 2007
Σ *p,p*'-DDT	Mothers in Brasil	Breast Milk (n=69)	GM=492.8	ng/g lipid	Azeredo et al. 2008
Blood					
p,p'-DDT	Women participant in the Child Health And Development Study, San Francisco Bay Area, California, 1963-1967.	Serum (n=399)	1931 ± 1247 (Median= 1611)	ng/g lipid	James et al. 2002
p,p'-DDT	Cross-sectional study among pregnant women hospital patients in Rio de	Plasma (n =16)	0.982± 2.17	ng/g	Sarcinelli et al.2003

	Jainero, Brasil 1997-1998.				
p,p'-DDT	Cross-sectional study among men from Chiapas Mexico.	Plasma (n =144)	67.4	µg/L	Barraza-Villarreal et al. 2004
p,p'-DDE	Pregnant women in Tapachula, Chiapas, Mexico in 1998	Serum (n =52)	Median= 676; range: 56-23169	ng/g lipid	Koepke et al. 2004
p,p'-DDT	Children residents of high (n=30) and low (n=30) exposure communities, from Chiapas, Mexico	Serum	HEC= 15.9±8.2 LEC= 1.9±3.6	µg/L	Herrera-Portugal et al. 2005
p,p'-DDT	Children cohort study, Minorca, Spain.	Umbilical Cord Blood (n=410)	0.18 ± 0.27	µg/L	Carrizo et al. 2006
p,p'-DDT	Children cohort study, Minorca, Spain.	Serum (4 years old) (n=285)	0.073 ± 0.1 2	µg/L	Carrizo et al. 2006
p,p'-DDT	Cross-sectional study among mother-infants pair (n=39)in Thailand, 2003-2004	Serum	195± 217	ng/g lipid	Asawasinsopon et al. 2006
		Umbilical Cord Blood (n=37)	77.1 ± 112		
p,p'-DDT	Cross-sectional study in the Michalovce district (contaminated area), and in the Svidnik and Stropkov districts (background area), in Slovakia (n=2047)	Serum	Contaminated area: Median= 72.9 Background area: Median =33.2	ng/g lipid	Petrik et al. 2006
p,p'-DDT	Pregnant women in prenatal care, Sweden.	Serum (n=321-323)	5 (range: 2-124)	ng/g lipid	Glynn et al. 2007

p,p'-DDE	Alaska Native women, 1980-1987.	Serum (n=131)	9.10	µg/L	Rubin et al. 2001
p,p'-DDE	German Environmental Survey 1998 (GerES III)	Serum	West Germany: GM=1.33b (95%CI:1.28 -1.38) East Germany: GM = 3.37 (95%CI: 3.11-3.64)	µg/L	Becker et al. 2002
p,p'-DDE	Women participant in the Child Health And Development Study, San Francisco Bay Area, California, 1963-1967.	Serum (n=399)	6854 ± 4804 (Median= 5878)	ng/g lipid	James et al. 2002
p,p'-DDE	Cross-sectional study among pregnant women hospital patients in Rio de Jainero, Brasil 1997-1998	Plasma (n= 62) Cord Blood (n = 6)	1.77± 1.53 0.757 ± 0.288	ng/g	Sarcinelli et al.2003
p,p'-DDE	Cross-sectional study among men from Chiapas Mexico.	Plasma (n =144)	203.5	µg/L	Barraza-Villarreal et al. 2004
p,p'-DDE	Pregnant women in Tapachula, Chiapas, Mexico in 1998	Serum (n =52)	Median= 4,843; range: 113-41964	ng/g lipid	Koepke et al. 2004
p,p'-DDE	Ten breast-fed infants and 10 bottle-fed infants.	Serum	Breast-fed: 1.05 (range:0.76-3.49) Bottle-fed: 0.18 (range: 0.07-0.54) 14.3 (unexposed)	µg/L	Lackmann et al. 2004

p,p'-DDE	Women (n=200) hospital patients in Spain.	Serum	8.11 ± 12.76	µg/L	Botella et al. 2004
p,p'-DDE	Children residents of high (n=30) and low (n=30) exposure communities, from Chiapas, Mexico	Serum	HEC 58.2±29.2 LEC 9.2±5.7	µg/L	Herrera-Portugal et al. 2005
p,p'-DDE	Birth cohort including 92 mother-infant pairs in Spain., 1997- 1999	Cord serum Child's serum of 13 months	Median= 0.85; IQ range= 0.50–1.68 Median= 2.86; IQ range= 0. 0.19–6.62	ng/mL ng/mL	Ribas-Fitó et al. 2005
p,p'-DDE	Prospective cohort of singleton birth, Faroe Islands.	Umbilical Cord Blood	1.76 ± 1.525 (Median=1.320) 110 ± 211 (Median=71.3)	ng/L ng/g lipid	Barr et al. 2006
p,p'-DDE	Prospective cohort of singleton birth, Faroe Islands.	Serum (14 years old)	4.04 ± 4.93 (Median=2.460) 762 ± 467 (Median=467)	ng/L ng/g lipid	Barr et al. 2006
p,p'-DDE	Children cohort study, Minorca, Spain.	Umbilical Cord Blood (n=410)	1.6 ± 2.0	µg/L	Carrizo et al. 2006
p,p'-DDE	Children cohort study, Minorca, Spain.	Serum (4 years old) (n=285)	1.6 ± 3.2	µg/L	Carrizo et al. 2006
p,p'-DDE	Cross-sectional study among mother-infants pair (n=39)in Thailand, 2003-2004	Serum Umbilical Cord Blood	1792± 1759 1028 ± 866	ng/g lipid	Asawasinsopon et al. 2006
p,p'-DDE	Cross-sectional study in the Michalovce district (contaminated area), and in the Svidnik and Stropkov	Serum	Contaminated area: Median= 2521 Background area: Median =1368	ng/g lipid	Petrik et al. 2006

	districts (background area), in Slovakia (n=2047)				
p,p'-DDE	Mothers (n=50) and Children (n=50) in China	Serum	Mothers: GM=6.16; 95%CI: 4.88-7.77 Children: GM= 7.54; 95%CI: 6..02-9.44	µg/L	Wang et al. 2008
p,p'-DDE	Postpartum women (n=50) China	Umbilical Cord Blood	GM=1.64; 95%CI: 1.48-1.82	µg/L	Wang et al. 2008
Σ*p,p'*-DDT	Women participant in the Child Health And Development Study, San Francisco Bay Area, California, 1963-1967.	Serum (n=399)	9052 ± 5595 (Median= 7951)	ng/g lipid	James et al. 2002
Σ *p,p'*-DDT	Women (n=200) hospital patients in Spain.	Serum	12.10 ± 39.43	µg/L	Botella et al. 2004
Adipose and other tissue					
p,p'-DDT	Pregnant women in Philippines (n=449)	Hair	At Mid-gestation (n=2); Median=0.98 At Birth (n=23); Median = 1.22	µg/g	Oestra et al. 2006
p,p'-DDT	Infants in Philippines (n=638).	Meconium (n=4)	Median= 1.75 (interquartile range: 1.08-3.313)	µg/L	Oestra et al. 2008
p,p'-DDE	Women hospital patients , Mexico.	Adipose, breast	Coast residents (n-56)= 883 (95% CI:592.4- 1472.8) Central Mexico residents (n=149)=399.22 (95% CI: 311.03-512.42)	ng/g lipid	Galván-Portillo et al. 2002
p,p'-DDE	Women (n=200) hospital patients in Spain.	Adipose	508.83 ± 410.54	ng/g lipid	Botella et al. 2004
p,p'-DDE	Mothers in Denmark (n=43)	Placenta	Denmark = 47.15	ng/g lipid	Shen et al. 2007

	and Finland (n=43)		Finland = 21.23		
p,p'-DDE	Postpartum women (n=50) China	Ventral Fat	GM=208.27; 95%CI: 7.22-1133.33	ng/g lipid	Wang et al. 2008
Σ *p,p*'-DDT	Women (n=200) hospital patients in Spain.	Adipose	543.25 ± 432.51	ng/g lipid	Botella et al. 2004

[a]Arithmetic mean concentrations are reported unless otherwise specified.
GM= geometric mean concentrations.

6.6 Exposures of Children

Children in malaria endemic countries where DDT spraying continues to be used for infection control are likely at risk of higher levels of exposure due to DDT in breast milk. According to Jaga and Dharmani (2003), people in African countries are highly exposed to DDT and have higher levels of these chemicals.

Elevated levels of DDT and DDE in breast milk (Table 6.1) have been reported among women in Thailand (Stuetz et al. 2001), Swaziland (Okonkwo and Kampira. 2002), Saudi Arabia. (al-Saleh I et al. 2003), China (Minh et al. 2004, Kunisue et al. 2004b,), South Africa (Bouwman et al. 2006), and Brasil (Azeredo et al. 2008). Infants of lactating mothers investigated in these studies had estimated total DDT that exceeded the 20 micrograms/kg-day of body weight, the WHO/UNEP Acceptable Daily Intakes for a 5-Kg infant. While DDT has been widely banned for agricultural use, it remains an important malarial control agent. In their study conducted in three South African towns, Bouwman et al. (2006) found that only in one town the human Acceptable Daily Intake (ADI) was exceeded by 1.9 times. At the maximum level of total DDT found, the ADI was exceeded by 12.3 times. Despite the strong concern about the exposure experienced by the infants to these levels of ΣDDT, they caution that the threat of disease and death posed by malaria far outweighs the possible or real negative effects of DDT intake at these levels.

7.0 ANALYTICAL METHODS

NO DATA

8.0 REGULATIONS AND ADVISORIES

NO DATA

9.0 REFERENCES

Akkina J, Reif J, Keefe T, Bachand A. Age at natural menopause and exposure to organochlorine pesticides in Hispanic women. J Toxicol Environ Health A. 2004 Sep 24;67(18):1407-22

al-Saleh I, Shinwari N, Basile P, el-Doush I, al-Zahrani M, al-Shanshoury M, el-Din Mohammed G.
DDT and its metabolites in breast milk from two regions in Saudi Arabia.
J Occup Environ Med. 2003 Apr;45(4):410-27.

Aneck-Hahn NH, Schulenburg GW, Bornman MS, Farias P, de Jager C.
Impaired semen quality associated with environmental DDT exposure in young men living in a malaria area in the Limpopo Province, South Africa.
J Androl. 2007 May-Jun;28(3):423-34. Epub 2006 Dec 27.

Aronson KJ, Miller AB, Woolcott CG, Sterns EE, McCready DR, Lickley LA, Fish EB, Hiraki GY, Holloway C, Ross T, Hanna WM, SenGupta SK, Weber JP.Breast adipose tissue concentrations of polychlorinated biphenyls and other organochlorines and breast cancer risk. Cancer Epidemiol Biomarkers Prev. 2000 Jan;9(1):55-63

Asawasinsopon R, Prapamontol T, Prakobvitayakit O, Vaneesorn Y, Mangklabruks A, Hock B.The association between organochlorine and thyroid hormone levels in cord serum: a study from northern Thailand. Environ Int. 2006 May;32(4):554-9

Ayotte P, Giroux S, Dewailly E, Hernández Avila M, Farias P, Danis R, Villanueva Díaz C. DDT spraying for malaria control and reproductive function in Mexican men.
Epidemiology. 2001 May;12(3):366-7.

Azeredo A, Torres JP, de Freitas Fonseca M, Britto JL, Bastos WR, Azevedo E Silva CE, Cavalcanti G, Meire RO, Sarcinelli PN, Claudio L, Markowitz S, Malm O.
DDT and its metabolites in breast milk from the Madeira River basin in the Amazon, Brazil.
Chemosphere. 2008 Aug;73(1 Suppl):S246-51.

Barr DB, Weihe P, Davis MD, Needham LL, Grandjean P.
Serum polychlorinated biphenyl and organochlorine insecticide concentrations in a Faroese birth cohort.
Chemosphere. 2006 Feb;62(7):1167-82

Barraza-Villarreal A, Farías P, Díaz Sánchez V, Bailey JL, De Jager T, Ayotte P, Hernández-Avila M, Dewailly E. Nonoccupational determinants of plasma DDT and p,p'-DDE in men from Chiapas, Mexico.
Arch Environ Health. 2004 Jan;59(1):42-9.

Beard J, Sladden T, Morgan G, Berry G, Brooks L, McMichael A. Health impacts of pesticide exposure in a cohort of outdoor workers. Environ Health Perspect. 2003 May;111(5):724-30.

Becker K, Conrad A, Kirsch N, Kolossa-Gehring M, Schulz C, Seiwert M, Seifert B.
German Environmental Survey (GerES): human biomonitoring as a tool to identify exposure pathways. Int J Hyg Environ Health. 2007 May;210(3-4):267-9.

Berkowitz GS, Lapinski RH, Wolff MS. The role of DDE and polychlorinated biphenyl levels in preterm birth. Arch Environ Contam Toxicol. 1996 Jan;30(1):139-41.

Bhatia R, Shiau R, Petreas M, Weintraub JM, Farhang L, Eskenazi B. Organochlorine pesticides and male genital anomalies in the child health and development studies. Environ Health Perspect. 2005 Feb;113(2):220-4.

Bjerregaard P, Hansen JC. Organochlorines and heavy metals in pregnant women from the Disko Bay area in Greenland. Sci Total Environ. 2000 Jan 17;245(1-3):195-202.

Botella B, Crespo J, Rivas A, Cerrillo I, Olea-Serrano MF, Olea N. Exposure of women to organochlorine pesticides in Southern Spain. Environ Res. 2004 Sep;96(1):34-40.

Bouwman H, Sereda B, Meinhardt HM. Simultaneous presence of DDT and pyrethroid residues in human breast milk from a malaria endemic area in South Africa.
Environ Pollut. 2006 Dec;144(3):902-17.

Brucker-Davis F, Wagner-Mahler K, Delattre I, Ducot B, Ferrari P, Bongain A, Kurzenne JY, Mas JC, Fénichel P; Cryptorchidism Study Group from Nice Area. Cryptorchidism at birth in Nice area (France) is associated with higher prenatal exposure to PCBs and DDE, as assessed by colostrum concentrations. Hum Reprod. 2008 Aug;23(8):1708-18.

Carrizo D, Grimalt JO, Ribas-Fito N, Sunyer J, Torrent M. Physical-chemical and maternal determinants of the accumulation of organochlorine compounds in four-year-old children. Environ Sci Technol. 2006 Mar 1;40(5):1420-6.

CDC. Centers for Disease Control and Prevention. Third National Report on Human Exposure to Environmental Chemicals. Atlanta (GA): CDC, 2005.

CDC. Center for Disease Control and Prevention. National Center for Health Statistic. NHANES 2003-2004. Lab 28 Organochlorine Pesticides.

Chao HR, Wang SL, Lin TC, Chung XH.
Levels of organochlorine pesticides in human milk from central Taiwan.
Chemosphere. 2006 Mar;62(11):1774-85. Epub 2005 Sep 12.

Chanon KE, Méndez-Galván JF, Galindo-Jaramillo JM, Olguín-Bernal H, Borja-Aburto VH.
Cooperative actions to achieve malaria control without the use of DDT.
Int J Hyg Environ Health. 2003 Aug;206(4-5):387-94.

Charlier CJ, Foidart JM. Comparative study of dichlorodiphenyldichloroethylene in blood and semen of two young male populations: lack of relationship to infertility, but evidence of high exposure of the mothers.

Reprod Toxicol. 2005 Jul-Aug;20(2):215-20. Epub 2005 Apr 19.

Charlier C, Foidart JM, Pitance F, Herman P, Gaspard U, Meurisse M, Plomteux G.
Environmental dichlorodiphenyltrichlorethane or hexachlorobenzene exposure and breast cancer: is there a risk? Clin Chem Lab Med. 2004 Feb;42(2):222-7.

Charlier C, Albert A, Herman P, Hamoir E, Gaspard U, Meurisse M, Plomteux G.
Breast cancer and serum organochlorine residues. Occup Environ Med. 2003 May;60(5):348-51.

Chen A, Zhang J, Zhou L, Gao ES, Chen L, Rogan WJ, Wolff MS. DDT serum concentration and menstruation among young Chinese women. Environ Res. 2005 Nov;99(3):397-402.

Cocco P, Brennan P, Ibba A, de Sanjosé Llongueras S, Maynadié M, Nieters A, Becker N, Ennas MG, Tocco MG, Boffetta P. Plasma polychlorobiphenyl and organochlorine pesticide level and risk of major lymphoma subtypes.
Occup Environ Med. 2008 Feb;65(2):132-40.

Cocco P, Fadda D, Ibba A, Melis M, Tocco MG, Atzeri S, Avataneo G, Meloni M, Monni F, Flore C.
Reproductive outcomes in DDT applicators. Environ Res. 2005 May;98(1):120-6.

Cocco P, Loviselli A, Fadda D, Ibba A, Melis M, Oppo A, Serra S, Taberlet A, Tocco MG, Flore C.
Serum sex hormones in men occupationally exposed to dichloro-diphenyl-trichloro ethane (DDT) as young adults.
J Endocrinol. 2004 Sep;182(3):391-7.

Cohn BA, Cirillo PM, Wolff MS, Schwingl PJ, Cohen RD, Sholtz RI, Ferrara A, Christianson RE, van den Berg BJ, Siiteri PK. DDT and DDE exposure in mothers and time to pregnancy in daughters.
Lancet. 2003 Jun 28;361(9376):2205-6. Erratum in: Lancet. 2003 Nov 1;362(9394):1504.

Cohn BA, Wolff MS, Cirillo PM, Sholtz RI. DDT and breast cancer in young women: new data on the significance of age at exposure. Environ Health Perspect. 2007 Oct;115(10):1406-14.

Cooper GS, Klebanoff MA, Promislow J, Brock JW, Longnecker MP. Polychlorinated biphenyls and menstrual cycle characteristics. Epidemiology. 2005 Mar;16(2):191-200.

Cooper GS, Savitz DA, Millikan R, Chiu Kit T. Organochlorine exposure and age at natural menopause. Epidemiology. 2002 Nov;13(6):729-33.

Coulston F. Reconsideration of the dilemma of DDT for the establishment of an acceptable daily intake. Regul Toxicol Pharmacol. 1985 Dec;5(4):332-83.

Cupul-Uicab LA, Gladen BC, Hernández-Avila M, Weber JP, Longnecker MP.
DDE, a degradation product of DDT, and duration of lactation in a highly exposed area of Mexico. Environ Health Perspect. 2008 Feb;116(2):179-83.

Dalvie MA, Myers JE, Thompson ML, Robins TG, Dyer S, Riebow J, Molekwa J, Jeebhay M, Millar R, Kruger P. 2004a The long-term effects of DDT exposure on semen, fertility, and sexual function of malaria vector-control workers in Limpopo Province, South Africa. Environ Res. 2004 Sep;96(1):1-8.

Dalvie MA, Myers JE, Thompson ML, Robins TG, Omar S, Riebow J. 2004b

Exploration of different methods for measuring DDT exposure among malaria vector-control workers in Limpopo Province, South Africa.
Environ Res. 2004 Sep;96(1):20-7.

Dalvie MA, Myers JE, Lou Thompson M, Dyer S, Robins TG, Omar S, Riebow J, Molekwa J, Kruger P, Millar R. 2004c. The hormonal effects of long-term DDT exposure on malaria vector-control workers in Limpopo Province, South Africa.
Environ Res. 2004 Sep;96(1):9-19.

Damgaard IN, Skakkebaek NE, Toppari J, Virtanen HE, Shen H, Schramm KW, Petersen JH, Jensen TK, Main KM; Nordic Cryptorchidism Study Group. Persistent pesticides in human breast milk and cryptorchidism. Environ Health Perspect. 2006 Jul;114(7):1133-8.

Darvill T, Lonky E, Reihman J, Stewart P, Pagano J. Prenatal exposure to PCBs and infant performance on the fagan test of infant intelligence. Neurotoxicology. 2000 Dec;21(6):1029-38.

De Jager C, Farias P, Barraza-Villarreal A, Avila MH, Ayotte P, Dewailly E, Dombrowski C, Rousseau F, Sanchez VD, Bailey JL.
Reduced seminal parameters associated with environmental DDT exposure and p,p'-DDE concentrations in men in Chiapas, Mexico: a cross-sectional study.
J Androl. 2006 Jan-Feb;27(1):16-27.

Dello Iacovo R, Celentano E, Strollo AM, Iazzetta G, Capasso I, Randazzo G.
Organochlorines and breast cancer. A study on Neapolitan women.
Adv Exp Med Biol. 1999;472:57-66. No abstract available.

Demers A, Ayotte P, Brisson J, Dodin S, Robert J, Dewailly E. Risk and aggressiveness of breast cancer in relation to plasma organochlorine concentrations. Cancer Epidemiol Biomarkers Prev. 2000 Feb;9(2):161-6.

Dewailly E, Dodin S, Verreault R, Ayotte P, Sauvé L, Morin J, Brisson J.
High organochlorine body burden in women with estrogen receptor-positive breast cancer. J Natl Cancer Inst. 1994 Feb 2;86(3):232-4. No abstract available.

Dorgan JF, Brock JW, Rothman N, Needham LL, Miller R, Stephenson HE Jr, Schussler N, Taylor PR.
Serum organochlorine pesticides and PCBs and breast cancer risk: results from a prospective analysis (USA). Cancer Causes Control. 1999 Feb;10(1):1-11

Ennaceur S, Gandoura N, Driss MR.
Organochlorine pesticide residues in human milk of mothers living in northern Tunisia.
Bull Environ Contam Toxicol. 2007 May;78(5):325-9.

Eskenazi B, Marks AR, Bradman A, Fenster L, Johnson C, Barr DB, Jewell NP. In utero exposure to dichlorodiphenyltrichloroethane (DDT) and dichlorodiphenyldichloroethylene (DDE) and neurodevelopment among young Mexican American children. Pediatrics. 2006 Jul;118(1):233-41.

FAO, 2005. http://www.codexalimentarius.net/web/index_en.jsp

Falck F Jr, Ricci A Jr, Wolff MS, Godbold J, Deckers P. Pesticides and polychlorinated biphenyl residues in human breast lipids and their relation to breast cancer. Arch Environ Health. 1992 Mar-Apr;47(2):143-6.

Farhang L, Weintraub JM, Petreas M, Eskenazi B, Bhatia R. Association of DDT and DDE with birth weight and length of gestation in the Child Health and Development Studies, 1959-1967. Am J Epidemiol. 2005 Oct 15;162(8):717-25. Epub 2005 Aug 24.

Fenster L, Eskenazi B, Anderson M, Bradman A, Harley K, Hernandez H, Hubbard A, Barr DB. Association of in utero organochlorine pesticide exposure and fetal growth and length of gestation in an agricultural population.
Environ Health Perspect. 2006 Apr;114(4):597-602.

Flores-Luévano S, Farías P, Hernández M, Romano-Riquer P, Weber JP, Dewailly E, Cuevas-Alpuche J, Romieu I. [DDT/DDE concentrations and risk of hypospadias. Pilot case-control study] Salud Publica Mex. 2003 Nov-Dec;45(6):431-8. Spanish.

Galván-Portillo M, Jiménez-Gutiérrez C, Torres-Sánchez L, López-Carrillo L.
Food consumption and adipose tissue DDT levels in Mexican women.
Cad Saude Publica. 2002 Mar-Apr;18(2):447-52.

Gatto NM, Longnecker MP, Press MF, Sullivan-Halley J, McKean-Cowdin R, Bernstein L. Serum organochlorines and breast cancer: a case-control study among African-American women. Cancer Causes Control. 2007 Feb;18(1):29-39.

Gerhard I, Daniel V, Link S, Monga B, Runnebaum B.
Chlorinated hydrocarbons in women with repeated miscarriages.
Environ Health Perspect. 1998 Oct;106(10):675-81.

Gladen BC, Klebanoff MA, Hediger ML, Katz SH, Barr DB, Davis MD, Longnecker MP. Prenatal DDT exposure in relation to anthropometric and pubertal measures in adolescent males. Environ Health Perspect. 2004 Dec;112(17):1761-7

Gladen BC, Rogan WJ, Hardy P, Thullen J, Tingelstad J, Tully M. Development after exposure to polychlorinated biphenyls and dichlorodiphenyl dichloroethene transplacentally and through human milk. J Pediatr. 1988 Dec;113(6):991-5.

Gladen BC, Rogan WJ. Effects of perinatal polychlorinated biphenyls and dichlorodiphenyl dichloroethene on later development.
J Pediatr. 1991 Jul;119(1 (Pt 1)):58-63.

Gladen BC, Shkiryak-Nyzhnyk ZA, Chyslovska N, Zadorozhnaja TD, Little RE. Persistent organochlorine compounds and birth weight. Ann Epidemiol. 2003 Mar;13(3):151-7.

Glynn A, Aune M, Darnerud PO, Cnattingius S, Bjerselius R, Becker W, Lignell S.
Determinants of serum concentrations of organochlorine compounds in Swedish pregnant women: a cross-sectional study. Environ Health. 2007 Feb 1;6:2.

Güttes S, Failing K, Neumann K, Kleinstein J, Georgii S, Brunn H. Chlororganic pesticides and polychlorinated biphenyls in breast tissue of women with benign and malignant breast disease. Arch Environ Contam Toxicol. 1998 Jul;35(1):140-7.

Hagmar L, Björk J, Sjödin A, Bergman A, Erfurth EM. Plasma levels of persistent organohalogens and hormone levels in adult male humans.
Arch Environ Health. 2001 Mar-Apr;56(2):138-43.

Hauser R, Singh NP, Chen Z, Pothier L, Altshul L. 2003a Lack of an association between environmental exposure to polychlorinated biphenyls and p,p'-DDE and DNA damage in human sperm measured using the neutral comet assay.
Hum Reprod. 2003 Dec;18(12):2525-33.

Hauser R, Chen Z, Pothier L, Ryan L, Altshul L. 2003b The relationship between human semen parameters and environmental exposure to polychlorinated biphenyls and p,p'-DDE. Environ Health Perspect. 2003 Sep;111(12):1505-11.

Helzlsouer KJ, Alberg AJ, Huang HY, Hoffman SC, Strickland PT, Brock JW, Burse VW, Needham LL, Bell DA, Lavigne JA, Yager JD, Comstock GW. Serum concentrations of organochlorine compounds and the subsequent development of breast cancer. Cancer Epidemiol Biomarkers Prev. 1999 Jun;8(6):525-32.

Herrera-Portugal C, Ochoa H, Franco-Sánchez G, Yáñez L, Díaz-Barriga F. Environmental pathways of exposure to DDT for children living in a malarious area of Chiapas, Mexico.
Environ Res. 2005 Oct;99(2):158-63.

Høyer AP, Grandjean P, Jørgensen T, Brock JW, Hartvig HB. Organochlorine exposure and risk of breast cancer. Lancet. 1998 Dec 5;352(9143):1816-20.

Hunter DJ, Hankinson SE, Laden F, Colditz GA, Manson JE, Willett WC, Speizer FE, Wolff MS.
Plasma organochlorine levels and the risk of breast cancer. N Engl J Med. 1997 Oct 30;337(18):1253-8.

Ibarluzea Jm J, Fernández MF, Santa-Marina L, Olea-Serrano MF, Rivas AM, Aurrekoetxea JJ, Expósito J, Lorenzo M, Torné P, Villalobos M, Pedraza V, Sasco AJ, Olea N. Breast cancer risk and the combined effect of environmental estrogens. Cancer Causes Control. 2004 Aug;15(6):591-600.

Jaga K, Dharmani C.
Global surveillance of DDT and DDE levels in human tissues.
Int J Occup Med Environ Health. 2003;16(1):7-20. Review. Corrected and republished in: Int J Occup Med Environ Health. 2006;19(1):83.

James RA, Hertz-Picciotto I, Willman E, Keller JA, Charles MJ.
Determinants of serum polychlorinated biphenyls and organochlorine pesticides measured in women from the child health and development study cohort, 1963-1967.
Environ Health Perspect. 2002 Jul;110(7):617-24.

Jusko TA, Koepsell TD, Baker RJ, Greenfield TA, Willman EJ, Charles MJ, Teplin SW, Checkoway H, Hertz-Picciotto I. Maternal DDT exposures in relation to fetal and 5-year growth. Epidemiology. 2006 Nov;17(6):692-700)

Kalantzi OI, Martin FL, Thomas GO, Alcock RE, Tang HR, Drury SC, Carmichael PL, Nicholson JK, Jones KC. Different levels of polybrominated diphenyl ethers (PBDEs) and chlorinated compounds in breast milk from two U.K. Regions.
Environ Health Perspect. 2004 Jul;112(10):1085-91.

Karmaus W, Davis S, Chen Q, Kuehr J, Kruse H.
Atopic manifestations, breast-feeding protection and the adverse effect of DDE.
Paediatr Perinat Epidemiol. 2003 Apr;17(2):212-20.

Karmaus W, Davis S, Fussman C, Brooks K.
Maternal concentration of dichlorodiphenyl dichloroethylene (DDE) and initiation and duration of breast feeding. Paediatr Perinat Epidemiol. 2005 Sep;19(5):388-98.

Karmaus W, Kuehr J, Kruse H.
Infections and atopic disorders in childhood and organochlorine exposure.
Arch Environ Health. 2001 Nov-Dec;56(6):485-92

Karmaus W, Zhu X. Maternal concentration of polychlorinated biphenyls and dichlorodiphenyl dichlorethylene and birth weight in Michigan fish eaters: a cohort study.
Environ Health. 2004 Jan 28;3(1):1.

Koepke R, Warner M, Petreas M, Cabria A, Danis R, Hernandez-Avila M, Eskenazi B.
Serum DDT and DDE levels in pregnant women of Chiapas, Mexico.
Arch Environ Health. 2004 Nov;59(11):559-65.

Korrick SA, Chen C, Damokosh AI, Ni J, Liu X, Cho SI, Altshul L, Ryan L, Xu X. Association of DDT with spontaneous abortion: a case-control study. Ann Epidemiol. 2001 Oct;11(7):491-6.

Krieger N, Wolff MS, Hiatt RA, Rivera M, Vogelman J, Orentreich N. Breast cancer and serum organochlorines: a prospective study among white, black, and Asian women. J Natl Cancer Inst. 1994 Apr 20;86(8):589-99.

Kunisue T, Someya M, Kayama F, Jin Y, Tanabe S.
Persistent organochlorines in human breast milk collected from primiparae in Dalian and Shenyang, China.
Environ Pollut. 2004 Oct;131(3):381-92.

Lackmann GM, Schaller KH, Angerer J.
Organochlorine compounds in breast-fed vs. bottle-fed infants: preliminary results at six weeks of age.
Sci Total Environ. 2004 Aug 15;329(1-3):289-93.

Laden F, Collman G, Iwamoto K, Alberg AJ, Berkowitz GS, Freudenheim JL, Hankinson SE, Helzlsouer KJ, Holford TR, Huang HY, Moysich KB, Tessari JD, Wolff MS, Zheng T, Hunter DJ. 1,1-Dichloro-2,2-bis(p-chlorophenyl)ethylene and polychlorinated biphenyls and breast cancer: combined analysis of five U.S. studies. J Natl Cancer Inst. 2001 May 16;93(10):768-76.

Law DC, Klebanoff MA, Brock JW, Dunson DB, Longnecker MP. Maternal serum levels of polychlorinated biphenyls and 1,1-dichloro-2,2-bis(p-chlorophenyl)ethylene (DDE) and time to pregnancy. Am J Epidemiol. 2005 Sep 15;162(6):523-32. Epub 2005 Aug 10.

Leoni V, Fabiani L, Marinelli G, Puccetti G, Tarsitani GF, De Carolis A, Vescia N, Morini A, Aleandri V, Pozzi V, et al. PCB and other organochlorine compounds in blood of women with or without miscarriage: a hypothesis of correlation.
Ecotoxicol Environ Saf. 1989 Feb;17(1):1-11.

Liljegren G, Hardell L, Lindström G, Dahl P, Magnuson A.
Case-control study on breast cancer and adipose tissue concentrations of congener specific polychlorinated biphenyls, DDE and hexachlorobenzene.
Eur J Cancer Prev. 1998 Apr;7(2):135-40.

Longnecker MP, Gladen BC, Cupul-Uicab LA, Romano-Riquer SP, Weber JP, Chapin RE, Hernández-Avila M. In utero exposure to the antiandrogen 1,1-dichloro-2,2-bis(p-chlorophenyl)ethylene (DDE) in relation to anogenital distance in male newborns from Chiapas, México. Am J Epidemiol. 2007 May 1;165(9):1015-22.

Longnecker MP, Klebanoff MA, Brock JW, Zhou H, Gray KA, Needham LL, Wilcox AJ. Maternal serum level of 1,1-dichloro-2,2-bis(p-chlorophenyl)ethylene and risk of cryptorchidism, hypospadias, and polythelia among male offspring. Am J Epidemiol. 2002 Feb 15;155(4):313-22.

Longnecker MP, Klebanoff MA, Dunson DB, Guo X, Chen Z, Zhou H, Brock JW.
Maternal serum level of the DDT metabolite DDE in relation to fetal loss in previous pregnancies.
Environ Res. 2005 Feb;97(2):127-33.

Longnecker MP, Klebanoff MA, Zhou H, Brock JW.
Association between maternal serum concentration of the DDT metabolite DDE and preterm and small-for-gestational-age babies at birth.
Lancet. 2001 Jul 14;358(9276):110-4.

López-Carrillo L, Blair A, López-Cervantes M, Cebrián M, Rueda C, Reyes R, Mohar A, Bravo J.
Dichlorodiphenyltrichloroethane serum levels and breast cancer risk: a case-control study from Mexico.
Cancer Res. 1997 Sep 1;57(17):3728-32.

López-Cervantes M, Torres-Sánchez L, Tobías A, López-Carrillo L. Dichlorodiphenyldichloroethane burden and breast cancer risk: a meta-analysis of the epidemiologic evidence. Environ Health Perspect. 2004 Feb;112(2):207-14.

Martin SA Jr, Harlow SD, Sowers MF, Longnecker MP, Garabrant D, Shore DL, Sandler DP. DDT metabolite and androgens in African-American farmers.
Epidemiology. 2002 Jul;13(4):454-8.

Mathur V, Bhatnagar P, Sharma RG, Acharya V, Sexana R. Breast cancer incidence and exposure to pesticides among women originating from Jaipur. Environ Int. 2002 Nov;28(5):331-6.

McGlynn KA, Abnet CC, Zhang M, Sun XD, Fan JH, O'Brien TR, Wei WQ, Ortiz-Conde BA, Dawsey SM, Weber JP, Taylor PR, Katki H, Mark SD, Qiao YL.

Serum concentrations of 1,1,1-trichloro-2,2-bis(p-chlorophenyl)ethane (DDT) and 1,1-dichloro-2,2-bis(p-chlorophenyl)ethylene (DDE) and risk of primary liver cancer.
J Natl Cancer Inst. 2006 Jul 19;98(14):1005-10.

McGlynn KA, Quraishi SM, Graubard BI, Weber JP, Rubertone MV, Erickson RL.
Persistent organochlorine pesticides and risk of testicular germ cell tumors.
J Natl Cancer Inst. 2008 May 7;100(9):663-71.

Mendonça GA, Eluf-Neto J, Andrada-Serpa MJ, Carmo PA, Barreto HH, Inomata ON, Kussumi TA.
Organochlorines and breast cancer: a case-control study in Brazil. Int J Cancer. 1999 Nov 26;83(5):596-600.

Minh NH, Someya M, Minh TB, Kunisue T, Iwata H, Watanabe M, Tanabe S, Viet PH, Tuyen BC.
Persistent organochlorine residues in human breast milk from Hanoi and Hochiminh City, Vietnam: contamination, accumulation kinetics and risk assessment for infants. Environ Pollut. 2004 Jun;129(3):431-41.

Moysich KB, Ambrosone CB, Vena JE, Shields PG, Mendola P, Kostyniak P, Greizerstein H, Graham S, Marshall JR, Schisterman EF, Freudenheim JL.
Environmental organochlorine exposure and postmenopausal breast cancer risk.
Cancer Epidemiol Biomarkers Prev. 1998 Mar;7(3):181-8.

Okonkwo JO, Kampira L.
Organochlorine pesticide residues in mother's milk in Swaziland, 1996-1997.
Bull Environ Contam Toxicol. 2002 May;68(5):740-6.

Olaya-Contreras P, Rodríguez-Villamil J, Posso-Valencia HJ, Cortez JE.
Organochlorine exposure and breast cancer risk in Colombian women.
Cad Saude Publica. 1998;14 Suppl 3:125-32.

Ostrea EM Jr, Villanueva-Uy E, Bielawski DM, Posecion NC Jr, Corrion ML, Jin Y, Janisse JJ, Ager JW. Maternal hair--an appropriate matrix for detecting maternal exposure to pesticides during pregnancy. Environ Res. 2006 Jul;101(3):312-22

Ouyang F, Perry MJ, Venners SA, Chen C, Wang B, Yang F, Fang Z, Zang T, Wang L, Xu X, Wang X.
Serum DDT, age at menarche, and abnormal menstrual cycle length.
Occup Environ Med. 2005 Dec;62(12):878-84.

Pavuk M, Cerhan JR, Lynch CF, Kocan A, Petrik J, Chovancova J. Case-control study of PCBs, other organochlorines and breast cancer in Eastern Slovakia. J Expo Anal Environ Epidemiol. 2003 Jul;13(4):267-75.

Perry MJ, Ouyang F, Korrick SA, Venners SA, Chen C, Xu X, Lasley BL, Wang X.
A prospective study of serum DDT and progesterone and estrogen levels across the menstrual cycle in nulliparous women of reproductive age. Am J Epidemiol. 2006 Dec 1;164(11):1056-64. Epub 2006 Oct 5.

Petrik J, Drobna B, Pavuk M, Jursa S, Wimmerova S, Chovancova J. Serum PCBs and organochlorine pesticides in Slovakia: age, gender, and residence as determinants of organochlorine concentrations. Chemosphere. 2006 Oct;65(3):410-8.

Raaschou-Nielsen O, Pavuk M, Leblanc A, Dumas P, Philippe Weber J, Olsen A, Tjønneland A, Overvad K, Olsen JH. Adipose organochlorine concentrations and risk of breast cancer among postmenopausal Danish women. Cancer Epidemiol Biomarkers Prev. 2005 Jan;14(1):67-74.

Ribas-Fitó N, Cardo E, Sala M, Eulàlia de Muga M, Mazón C, Verdú A, Kogevinas M, Grimalt JO, Sunyer J. Breastfeeding, exposure to organochlorine compounds, and neurodevelopment in infants. Pediatrics. 2003 May;111(5 Pt 1):e580-5.

Ribas-Fitó N, Gladen BC, Brock JW, Klebanoff MA, Longnecker MP.
Prenatal exposure to 1,1-dichloro-2,2-bis (p-chlorophenyl)ethylene (p,p'-DDE) in relation to child growth. Int J Epidemiol. 2006 Aug;35(4):853-8..

Ribas-Fitó N, Grimalt JO, Marco E, Sala M, Mazón C, Sunyer J.
Breastfeeding and concentrations of HCB and p,p'-DDE at the age of 1 year.
Environ Res. 2005 May;98(1):8-13.

Rignell-Hydbom A, Rylander L, Giwercman A, Jönsson BA, Nilsson-Ehle P, Hagmar L. Exposure to CB-153 and p,p'-DDE and male reproductive function.
Hum Reprod. 2004 Sep;19(9):2066-75. Epub 2004 Jul 29.

Rignell-Hydbom A, Rylander L, Giwercman A, Jönsson BA, Lindh C, Eleuteri P, Rescia M, Leter G, Cordelli E, Spano M, Hagmar L. Exposure to PCBs and p,p'-DDE and human sperm chromatin integrity. Environ Health Perspect. 2005 Feb;113(2):175-9.
PMID: 15687046 [PubMed - indexed for MEDLINE]

Rignell-Hydbom A, Rylander L, Hagmar L.
Exposure to persistent organochlorine pollutants and type 2 diabetes mellitus.
Hum Exp Toxicol. 2007 May;26(5):447-52.

Ritchie JM, Vial SL, Fuortes LJ, Guo H, Reedy VE, Smith EM. Organochlorines and risk of prostate cancer. J Occup Environ Med. 2003 Jul;45(7):692-702.

Rogan WJ, Chen A.
Health risks and benefits of bis(4-chlorophenyl)-1,1,1-trichloroethane (DDT).
Lancet. 2005 Aug 27-Sep 2;366(9487):763-73.

Rogan WJ, Gladen BC, McKinney JD, Carreras N, Hardy P, Thullen J, Tingelstad J, Tully M.
Polychlorinated biphenyls (PCBs) and dichlorodiphenyl dichloroethene (DDE) in human milk: effects of maternal factors and previous lactation. Am J Public Health. 1986 Feb;76(2):172-7.

Rogan WJ, Gladen BC. PCBs, DDE, and child development at 18 and 24 months.
Ann Epidemiol. 1991 Aug;1(5):407-13.

Rogan WJ, Gladen BC, McKinney JD, Carreras N, Hardy P, Thullen J, Tinglestad J, Tully M. Neonatal effects of transplacental exposure to PCBs and DDE. J Pediatr. 1986 Aug;109(2):335-41

Romieu I, Hernandez-Avila M, Lazcano-Ponce E, Weber JP, Dewailly E. Breast cancer, lactation history, and serum organochlorines. Am J Epidemiol. 2000 Aug 15;152(4):363-70.

Rubin CH, Lanier A, Kieszak S, Brock JW, Koller KR, Strosnider H, Needham L, Zahm S, Harpster A. Breast cancer among Alaska Native women potentially exposed to environmental organochlorine chemicals. Int J Circumpolar Health. 2006 Feb;65(1):18-27.

Rubin CH, Lanier A, Socha M, Brock JW, Kieszak S, Zahm S.
Exposure to persistent organochlorines among Alaska Native women.
Int J Circumpolar Health. 2001 Apr;60(2):157-69.

Sagiv SK, Tolbert PE, Altshul LM, Korrick SA. Organochlorine exposures during pregnancy and infant size at birth. Epidemiology. 2007 Jan;18(1):120-9).

Salazar-García F, Gallardo-Díaz E, Cerón-Mireles P, Loomis D, Borja-Aburto VH.
Reproductive effects of occupational DDT exposure among male malaria control workers. Environ Health Perspect. 2004 Apr;112(5):542-7.

Sarcinelli PN, Pereira AC, Mesquita SA, Oliveira-Silva JJ, Meyer A, Menezes MA, Alves SR, Mattos RC, Moreira JC, Wolff M. Dietary and reproductive determinants of plasma organochlorine levels in pregnant women in Rio de Janeiro. Environ Res. 2003 Mar;91(3):143-50.

Saxena MC, Siddiqui MK, Seth TD, Krishna Murti CR, Bhargava AK, Kutty D.
Organochlorine pesticides in specimens from women undergoing spontaneous abortion, premature of full-term delivery.
J Anal Toxicol. 1981 Jan-Feb;5(1):6-9.

Schecter A, Toniolo P, Dai LC, Thuy LT, Wolff MS. Blood levels of DDT and breast cancer risk among women living in the north of Vietnam. Arch Environ Contam Toxicol. 1997 Nov;33(4):453-6.

Shen H, Main KM, Virtanen HE, Damggard IN, Haavisto AM, Kaleva M, Boisen KA, Schmidt IM, Chellakooty M, Skakkebaek NE, Toppari J, Schramm KW.
From mother to child: investigation of prenatal and postnatal exposure to persistent bioaccumulating toxicants using breast milk and placenta biomonitoring.
Chemosphere. 2007 Apr;67(9):S256-62.

Siddiqui MK, Anand M, Mehrotra PK, Sarangi R, Mathur N. Biomonitoring of organochlorines in women with benign and malignant breast disease. Environ Res. 2005 Jun;98(2):250-7.

Siddiqui MK, Srivastava S, Srivastava SP, Mehrotra PK, Mathur N, Tandon I. Persistent chlorinated pesticides and intra-uterine foetal growth retardation: a possible association. Int Arch Occup Environ Health. 2003 Feb;76(1):75-80. Epub 2002 Oct 17.

Spanò M, Toft G, Hagmar L, Eleuteri P, Rescia M, Rignell-Hydbom A, Tyrkiel E, Zvyezday V, Bonde JP; INUENDO.
Exposure to PCB and p, p'-DDE in European and Inuit populations: impact on human sperm chromatin integrity. Hum Reprod. 2005 Dec;20(12):3488-99. Epub 2005 Oct 13

Stewart P, Reihman J, Lonky E, Darvill T, Pagano J. Prenatal PCB exposure and neonatal behavioral assessment scale (NBAS) performance. Neurotoxicol Teratol. 2000 Jan-Feb;22(1):21-9.

Stuetz W, Prapamontol T, Erhardt JG, Classen HG.

Organochlorine pesticide residues in human milk of a Hmong hill tribe living in Northern Thailand. Sci Total Environ. 2001 Jun 12;273(1-3):53-60.

Sugiura-Ogasawara M, Ozaki Y, Sonta S, Makino T, Suzumori K. PCBs, hexachlorobenzene and DDE are not associated with recurrent miscarriage.
Am J Reprod Immunol. 2003 Dec;50(6):485-9.

Sunyer J, Torrent M, Muñoz-Ortiz L, Ribas-Fitó N, Carrizo D, Grimalt J, Antó JM, Cullinan P.
Prenatal dichlorodiphenyldichloroethylene (DDE) and asthma in children.
Environ Health Perspect. 2005 Dec;113(12):1787-90.

Szyrwińska K, Lulek J.
Exposure to specific polychlorinated biphenyls and some chlorinated pesticides via breast milk in Poland. Chemosphere. 2007 Jan;66(10):1895-903. Epub 2006 Oct 4.

Tiido T, Rignell-Hydbom A, Jönsson B, Giwercman YL, Rylander L, Hagmar L, Giwercman A.
Exposure to persistent organochlorine pollutants associates with human sperm Y:X chromosome ratio. Hum Reprod. 2005 Jul;20(7):1903-9. Epub 2005 Apr 28.

Tiido T, Rignell-Hydbom A, Jönsson BA, Giwercman YL, Pedersen HS, Wojtyniak B, Ludwicki JK, Lesovoy V, Zvyezday V, Spano M, Manicardi GC, Bizzaro D, Bonefeld-Jørgensen EC, Toft G, Bonde JP, Rylander L, Hagmar L, Giwercman A; INUENDO.
Impact of PCB and p,p'-DDE contaminants on human sperm Y:X chromosome ratio: studies in three European populations and the Inuit population in Greenland.
Environ Health Perspect. 2006 May;114(5):718-24.

Toft G, Axmon A, Lindh CH, Giwercman A, Bonde JP.
Menstrual cycle characteristics in European and Inuit women exposed to persistent organochlorine pollutants. Hum Reprod. 2008 Jan;23(1):193-200. Epub 2007 Nov 7.

Torres-Arreola L, Berkowitz G, Torres-Sánchez L, López-Cervantes M, Cebrián ME, Uribe M, López-Carrillo L. Preterm birth in relation to maternal organochlorine serum levels. Ann Epidemiol. 2003 Mar;13(3):158-62.

Unger M, Kiaer H, Blichert-Toft M, Olsen J, Clausen J. Organochlorine compounds in human breast fat from deceased with and without breast cancer and in a biopsy material from newly diagnosed patients undergoing breast surgery. Environ Res. 1984 Jun;34(1):24-8

van't Veer P, Lobbezoo IE, Martín-Moreno JM, Guallar E, Gómez-Aracena J, Kardinaal AF, Kohlmeier L, Martin BC, Strain JJ, Thamm M, van Zoonen P, Baumann BA, Huttunen JK, Kok FJ. DDT (dicophane) and postmenopausal breast cancer in Europe: case-control study. BMJ. 1997 Jul 12;315(7100):81-5.

Vasiliu O, Muttineni J, Karmaus W. In utero exposure to organochlorines and age at menarche. Hum Reprod. 2004 Jul;19(7):1506-12. Epub 2004 May 6.

Venners SA, Korrick S, Xu X, Chen C, Guang W, Huang A, Altshul L, Perry M, Fu L, Wang X.
Preconception serum DDT and pregnancy loss: a prospective study using a biomarker of pregnancy. Am J Epidemiol. 2005 Oct 15;162(8):709-16. Epub 2005 Aug 24.

Wang YR, Zhang M, Wang Q, Yang DY, Li CL, Liu J, Li JG, Li H, Yang XY.
Exposure of mother-child and postpartum woman-infant pairs to DDT and its metabolites in Tianjin, China.
Sci Total Environ. 2008 Jun 15;396(1):34-41.

Ward EM, Schulte P, Grajewski B, Andersen A, Patterson DG Jr, Turner W, Jellum E, Deddens JA, Friedland J, Roeleveld N, Waters M, Butler MA, DiPietro E, Needham LL.
Serum organochlorine levels and breast cancer: a nested case-control study of Norwegian women. Cancer Epidemiol Biomarkers Prev. 2000 Dec;9(12):1357-67

Wassermann M, Nogueira DP, Tomatis L, Mirra AP, Shibata H, Arie G, Cucos S, Wassermann D.
Organochlorine compounds in neoplastic and adjacent apparently normal breast tissue. Bull Environ Contam Toxicol. 1976 Apr;15(4):478-84.

Weiderpass E, Adami HO, Baron JA, Wicklund-Glynn A, Aune M, Atuma S, Persson I.
Organochlorines and endometrial cancer risk. Cancer Epidemiol Biomarkers Prev. 2000 May;9(5):487-93.

Weisskopf MG, Anderson HA, Hanrahan LP, Kanarek MS, Falk CM, Steenport DM, Draheim LA; Great Lakes Consortium. Maternal exposure to Great Lakes sport-caught fish and dichlorodiphenyl dichloroethylene, but not polychlorinated biphenyls, is associated with reduced birth weight. Environ Res. 2005 Feb;97(2):149-62.

Windham GC, Lee D, Mitchell P, Anderson M, Petreas M, Lasley B. Exposure to organochlorine compounds and effects on ovarian function. Epidemiology. 2005 Mar;16(2):182-90.

Wolff MS, Toniolo PG, Lee EW, Rivera M, Dubin N. Blood levels of organochlorine residues and risk of breast cancer. J Natl Cancer Inst. 1993 Apr 21;85(8):648-52.

Wolff MS, Berkowitz GS, Brower S, Senie R, Bleiweiss IJ, Tartter P, Pace B, Roy N, Wallenstein S, Weston A. Organochlorine exposures and breast cancer risk in New York City women. Environ Res. 2000 a Oct;84(2):151-61.

Wolff MS, Zeleniuch-Jacquotte A, Dubin N, Toniolo P. Risk of breast cancer and organochlorine exposure. Cancer Epidemiol Biomarkers Prev. 2000 b Mar;9(3):271-7.

Zheng T, Holford TR, Mayne ST, Ward B, Carter D, Owens PH, Dubrow R, Zahm SH, Boyle P, Archibeque S, Tessari J. DDE and DDT in breast adipose tissue and risk of female breast cancer. Am J Epidemiol. 1999 Sep 1;150(5):453-8.

Zheng T, Holford TR, Mayne ST, Tessari J, Ward B, Carter D, Owens PH, Boyle P, Dubrow R, Archibeque-Engle S, Dawood O, Zahm SH. Risk of female breast cancer associated with serum polychlorinated biphenyls and 1,1-dichloro-2,2'-bis(p-chlorophenyl)ethylene. Cancer Epidemiol Biomarkers Prev. 2000 Feb;9(2):167-74.