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This work is a continuation of the effort to determine environmental and genetic determinants of breast cancer. Since the last report data on serum organochlorine levels, e.g. serum DDE, HCB, total PCBs, and mirex, became available. These data were explored by first conducting a thorough descriptive analysis. Results from this cross-sectional research suggested that increased fruit and vegetable intake may be associated with higher levels of serum DDE, HCB, and total PCBs. No associations with fish intake were observed for any of the compounds under investigation. Weak associations were found for intake of individual meats and dairy products and serum levels of DDE, HCB, and total PCBs. Caffeine intake appeared to be inversely related to DDE, HCB, and total PCBs. We observed no strong inverse associations with any of these compounds and duration of lactation. Future analytic efforts will focus on the association between these organochlorine compounds and breast cancer risk.

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INTRODUCTION

Previous work in this postdoctoral research project has focused on genetic variability in hormone and carcinogen metabolism in relation to breast cancer risk. This work was conducted by Dr. Christine B. Ambrosone, who has recently left the Department of Social and Preventive Medicine to take a position at the National Center for Toxicology Research in Little Rock, Arkansas. Since July 1, 1996 this grant supports the work of Ms. Kirsten B. Moysich, which examines the relationship between serum levels of organochlorines and breast cancer risk in postmenopausal women.

Since the last report of this kind toxicological data on serum levels of DDE, a major metabolite of 2,2-bis(p-chlorophenyl)-1,1,1-trichloroethane (DDT), hexachlorobenzene (HCB), polychlorinated biphenyls (PCBs), and mirex became available. We initiated our examination of these data by first conducting a thorough descriptive analysis, before investigating the effect of these compounds on breast cancer risk in our population. Therefore, this report will focus on the cross-sectional component of this research. The rationale for this work is based on the observation that there is evidence that these compounds may affect long term health. In order to study their effects, an understanding of the factors associated with this exposure and of potential confounding to the relationship is essential. We examined here dietary, occupational, medical, and lifestyle correlates of serum organochlorine levels in a group of healthy postmenopausal women. In addition to the results from the descriptive analysis, a detailed plan for future analytic efforts will be outlined.

A. Background

(1) DDE: 2,2-bis(p-chlorophenyl)-1,1,1-trichloroethane (DDT) was one of the most widely used chemicals for controlling insect pests on agricultural crops and controlling insects that carry such diseases such as malaria and typhus. Humans are currently exposed to DDT primarily by eating foods that contain small amounts of these compounds. Even though DDT has not been used in the US since 1972, small amounts of DDT and DDE are still detectable in soil and may be transferred to crops grown on this soil. These compounds are resistant to breakdown and are readily absorbed by

sediments and soil, which act both as sinks and long-term sources of exposure (IARC, 1991). Furthermore, imported food stuffs may still be directly exposed to DDT. DDT and its breakdown products are also found in air, water, and soil samples. However, these levels are low, and exposure by these pathways is of little concern.

The physiochemical characteristics of DDT and its metabolites enable organisms to absorb them readily. Once absorbed, DDT compounds are stored most readily in adipose tissue. Stored amounts leave the body very slowly. Adipose tissue levels may remain relatively constant or even increase with continued exposure over time. DDT metabolites are excreted primarily through urine and breast milk. (ATSDR, 1989b).

(2) PCBs: Another group of organochlorines, polychlorinated biphenyls (PCBs) have been manufactured commercially since 1929 for a variety of applications, including use as dielectrics in transformers and capacitors and as cooling fluids in hydraulic systems. PCBs were also utilized in the formulation of lubricating and cutting oils, in pesticides and flame retardents, and as plasticizers in paints, copying paper, adhesives, sealants, and plastics. In addition to the suitability of PCBs for a number of commercial applications, their resistance to chemical and biological breakdown contributed to their widespread commercial use (Silberhorn et al., 1990). PCBs were manufactured commercially by the chlorination of biphenyl, with varying numbers of chlorine atoms introduced into the biphenyl molecule. Isomeric PCBs are those with the same molecular weight, e.g. the same number of halogen substitutions. There are 209 possible PCB congeners or homologues. The PCB congeners with the highest degree of bioaccumulation have five to seven chlorine atoms per molecule. These moderately chlorinated isomer groups (penta-, hexa-, and hepta- chlorobiphenyls) account for 112 of the 209 congeners. They were synthesized in high proportions in many commercial preparations and are likely to be prevalent in the environment. The more highly chlorinated congeners are generally less available to organisms both because they bind more tightly with soils and sediments and because they are present in lower quantities in the environment. Congeners with fewer chlorines are more readily metabolized and eliminated with less tendency to bioaccumulate (Mc Farland & Clarke, 1989).

Although commercial production of PCB mixtures was banned in the US in the late 1970's, there is still evidence of the presence of PCB residues in the general population. Presently, the primary source of new exposure to PCBs in the general population is the consumption of contaminated fish (ATSDR, 1989a).

(3) HCB and Mirex: Hexachlorobenzene (HCB) is a widespread chlorinated hydrocarbon originating from agricultural and industrial sources. It was formerly used as a fungicide, but currently, the major source of HCB is industrial emission as a side product related to the manufacture of organochlorinated products. It has a strong tendency to accumulate the food chain and lipid rich tissues of animals and humans (To-Figueras et al. 1995).

Mirex, an organochlorine pesticide, was extensively used in the southeastern US for the control of the red fire ant. Mirex was also used as a fire retardant coating. Mirex is a persistent compound and due to its pharmacodynamics, it possesses environmental mobility and can be incorporated into the food chain, is lipophilic, with storage and accumulation in the lipid portion of animal tissues. Mirex was banned in 1974 due to its adverse effects on the reproductive system, its carcinogenic effects in mice and rats, and its bioaccumulation and persistence in the environment (Waters et al., 1977).

B. Review of the literature

Previous studies describing organochlorine levels in humans are few in number and hampered by methodological inconsistencies. Most of these studies were designed to document organochlorine in either special exposure populations or in the general population of different geographic regions. Levels of these compounds were measured in adipose tissue, human breast milk , and blood.

(1) Population organochlorine levels: Among the studies focusing on special exposure populations, Dewailly et al. (1994) examined plasma levels of several PCB congeners among 10 highly exposed fishermen from the Gulf of St. Lawrence (Canada). Compared with nonexposed controls, the fishermen had significantly higher fat lipid plasma levels for all PCB congeners (e.g., 2457 vs. 73 ng/g for congener 153). Sonzogni et al. (1991) reported on congener specific PCB levels among 173

Wisconsin sport fish consumers, the mean level of congener 153 was 1.43 ng/g. Another U.S. study examined mean serum levels of total PCBs and DDT among 30 Michigan women and children living on farms with PCB contaminated silos. Mean total PCB levels were 6.8 ng/g for children and 9.6 ng/g for women; mean DDT levels were 2.0 ng/g and 5.1 ng/g, respectively (Schatz et al., 1994). Schechter and colleagues (1994) compared PCB congener levels of Yusho patients 21 years after the Yusho accident with those of occupationally exposed workers and those of unexposed controls. The mean whole blood levels for PCB congener 153 were 2.36 ng/g, 1.59 ng/g, and 0.65 ng/g for the patients, workers, and controls, respectively. In a Croatian study, occupationally exposed workers had more than twice the median serum level of total PCBs than environmentally exposed individuals (8 ng/g vs. 3 ng/g) (Krauthacker, 1993). Similarly, Kannan et al. (1994) found that whole blood PCB congener levels among occupationally exposed German capacitor workers were substantially higher than among people from the general population (e.g., congener 153 2.3 vs. 0.36 ng/g). Results from a finish study demonstrated that workers who were accidentally exposed to PCBs through a capacitor fire had higher serum PCB congener levels than capacitor manufacture workers and individuals who were environmentally exposed to these compounds (e.g., congener 153 1.94 ng/g, 1. ng/g, and 1.2 ng/g, respectively) (Luotamo et al., 1991). Among male Ontario residents who were occupationally exposed to organochlorines whole blood analyses indicated mean levels of 18 ng/g for total PCBs and 3.4 ng/g for t-DDT (Frank et al., 1988). Two additional investigations describe organochlorine levels in the general population of various geographical regions. Krauthacker (1991) studied young women from the Northern Adriatic area of the former Yugoslavia and found median serum measures of 2 ng/g for total PCBs, 6 ng/g for DDE, and 2 ng/g for HCB. In a similar study conducted in Norway serum levels of young women were 10 ng/g for total PCBs, 19 ng/g for DDE, and 1 ng/g for HCB (Skaare et al., 1988)

(2) Predictors of organochlorines: Few studies attempted to relate organochlorine levels to dietary, reproductive, or demographic variables. In a German study, Lommel and colleagues (1992) compared blood organochlorine levels with self-reported

questionnaire data from 135 Elbe River sports fishermen. Results indicated that there was a positive association between age and fish consumption for HCB and DDE levels. For both of these compounds there was a suggestion of a positive association with relative body weight (Broco Index=kg/cm-100). No associations were observed for occupation, health status, and other dietary variables such as dairy, meat, or egg intake. In a less direct approach, Frank et al. (1993) compared DDE and PCB residues in the general diet with blood levels of Ontario residents. Blood samples were obtained from individuals who suspected oral, dermal, or inhalation exposure of PCBs. As for foods, pork and milk contained the highest levels of both DDE (0.77 and 0.32 ng/g, respectively) and PCBs (1.1 and 0.60 ng/g, respectively). This observation was more pronounced when these compounds were measured in extractable fat of the foods. Relatively low levels were found in raw fruits and vegetables and wheat products. Mean residues of PCBs and DDE were measured in whole blood from residents of large and medium to small urban centers across the province of Ontario. PCB levels ranged from 6.4 ng/g in Mississauga to 13.0 ng/g in the Niagara region. DDE levels were lowest in London and Dundas (2.7 ng/g) and highest in Burlington (4.5 ng/g).

Kannan et al. (1992) attempted to monitor the extent of organochlorine contamination in a wide variety of foodstuffs in Vietnam and to explore its impact on human dietary exposure. The authors measured PCBs, DDT, and HCB in different foods and linked measures with daily dietary intake (g/person/day) in Vietnam. PCB and DDT levels were highest in animal fat (61 and 130 ng/g, respectively), and HCB levels were highest in butter (5 ng/g). Lowest levels for all compounds were observed in rice. Fish, shellfish, prawn, and crab were the primary route of DDT to humans, whereas, cereals and vegetables were the predominant sources of PCBs and HCB in this geographical region. In a similar study, Kashyap et al. (1994) monitored DDT levels in duplicate diet samples and correlated these with respective residue blood samples from healthy Indians. The total food consumed by these exclusively vegetarian participants per day was collected and categorized as fatty food, nonfatty food, water and beverages. Fatty foods had the highest DDE residue levels (vegetable 167.33 ng/g and milk 306 ng/g) when compared to nonfatty foods (2.65 ng/g), water (3.21 ng/g),

and other beverages (0.74 ng/g). The average total DDT consumed by an adult was 19.240 ng, and this intake was reflected in blood DDT levels (r=0.69). In a more recent study, average daily dietary exposure to DDE and other contaminants were estimated for approximately 120,000 U.S. adults (MacIntosh et al., 1996). Food frequency questionnaire data from the Nurses Health Study and the Health Professionals' Follow-up Study were matched to residue data for table-ready foods collected as part of the FDA Total Diet Study. Contaminants were measured in 234 food items. The estimated average daily intake of DDE was 1200 ng for both men and women. Consumption of beef and whole milk are estimated to be major contributors to dietary DDE exposure.

Kreiss (1985) reported that the mean serum levels of PCBs in US population groups without occupational exposure to these agents are usually between 4 and 8 ng/g . Individuals who consume fish from contaminated waters (e.g. Great Lakes) exhibit a four-fold increase in serum PCB levels when compared to individuals who do not consume fish. Furthermore, levels of some fish consumers have been found to be within the range of those of with industrial exposure by involvement in capacitor manufacture. Evans et al. (1994) found that serum DDE levels were significantly higher among individuals who consumed one pound of fish per week from contaminated river areas than among controls (11.27 vs. 6.34 ng/g). In a Swedish study blood levels of PCBs and DDE were compared among individuals with varying amounts of fish consumption from the Baltic sea (Asplund et al. 1994). Individuals who were classified as high fish consumers had the highest plasma DDE (14 ng/g) and individual PCB congener (e.g., congener 153 3.4 ng/g) levels, when compared to moderate (3.5 and 1.8 ng/g, respectively) and non-consumers (2.4 and 1.5 ng/g, respectively). The authors reported strong correlations between dietary fish intake and blood levels of these contaminants (r=0.58-0.87). Fiore et al. (1989) found mean serum PCB levels of 2.2 ng/g and mean DDE levels of 6.3 ng/g among 192 Wisconsin anglers. Weak correlations were observed between total number of fish meals and body burden. Fish consumption and lifestyle variables were related to serum PCB and DDT levels among Michigan sports fishermen and regional controls (Hovinga et al.,

1993). Serum PCB and DDT levels were significantly higher in the fishermen (19.0 and 15.6 ng/g, respectively) than in the controls (6.8 and 6.8 ng/g, respectively). When fishermen were analyzed separately, historical fish consumption and age emerged as the strongest predictors for serum PCB and DDT levels. No associations were observed for tobacco or alcohol consumption and body mass index.

As pointed out above the comparability of organochlorine levels across studies is restricted, due to the following inconsistencies: first, studies differ in their use of either serum, plasma, or whole blood for toxicological analyses; second, studies vary greatly with regard to sample size, which affects the stability of the reported mean values; third, studies differ in their treatment of participants with nondetectable organochlorine levels (below the limit of detection (LOD)), fourth, not all studies report means for males and females separately; and finally, most studies conducted among women utilized pregnant women as participants. It cannot be ruled out, that the endocrine changes associated with pregnancy have not affected organochlorine levels. Furthermore, investigations on special exposure populations can only reflect the impact of a particular exposure on body burden and cannot be applied to the general population. Similarly, comparisons of organochlorine levels from populations derived from different geographic regions must be made with caution, in that organochlorine exposure is likely to vary among these regions, and there may be genetic differences in metabolism of these compounds across ethnic groups, that would affect body burden. In this study, frequency of occurrence and magnitude of organochlorine levels among healthy, postmenopausal women were determined.

As described above, the body of literature on correlates of serum organochlorine levels is very limited. To our knowledge, no previous investigation has attempted to relate biological markers of these compounds to a wide variety of potentially correlated variables. In the present study, dietary, demographic, occupational, and lifestyle variables were investigated with regard to their association with serum DDE, PCB, HCB, and mirex levels.

MATERIALS AND METHODS

A. Study population

The study sample in this descriptive analysis, a subset of the control group, consists 193 postmenopausal women ranging in age from 45 to 85. Women in the study were enrolled from 1986 to 1989 in Erie and Niagara counties in Western New York. Women older than 50 years of age who had ceased menstruation, as well as women younger than 50 years of age with natural menopause or nonfunctional ovaries (e.g., due to bilateral oophorectomy or irradiation) were considered postmenopausal.

1076 female residents of the two counties were randomly selected to participate as controls. Women older than 65 years of age were selected from Health Care Finance Administration rolls (41.7% of all control subjects), whereas women younger than 65 years of age were selected from New York State Department of Motor Vehicles rolls. After selection, participants were sent letters asking for participation and then called by an interviewer in order to make appointments for the interview. 494 (45.9 percent) of the 1076 randomly selected postmenopausal women agreed to participate. Among these women 322 (65%) agreed to provide a blood sample. Characteristics of postmenopausal women who gave blood and those who did not were compared. There were no differences with regard to variables related to demographics, dietary intake, medical history, or lifestyle. The only differences were that women who gave blood had slightly more pregnancies than those who did not give blood (3.5 vs. 2.9 pregnancies) and were less likely to smoke cigarettes (Ambrosone, 1994). Almost all samples were drawn from the women in a fasting state, and records were kept of whether the sample was fasting or not. Blood drawing was done by a commercial laboratory. Upon collection, blood specimens were transported on ice within three hours to our laboratory. Blood was processed immediately by a trained lab technician and placed in a freezer. Since collection, the samples have remained frozen at -70°C. A subset of 193 of the stored blood specimens was utilized for this research.

B. Interview

Participants were interviewed in-person by trained nurse-interviewers. The interview took approximately two hours to complete and included assessment of medical history, reproductive history, occupational history, exposure to exogenous hormones,

family history of cancer, and a food frequency questionnaire that assessed usual intake in the year two years prior to the interview. Intake assessment included frequency of intake, seasonal intake, and food preparation. Smoking and alcohol consumption histories, including age at which regular smoking and drinking began, duration, and quantities used were also assessed.

Organochlorine levels were compared to a lifestyle, dietary, reproductive, medical, and occupational variables. Lifestyle variables included age, years of education, body mass index (weight/height^2), weight, height, weekly hours of strenuous physical activity, defined as activity resulting into perspiration, and weekly hours of walking for exercise, pleasure, or transportation. Additional lifestyle variables were dietary fat consumption, water intake, and consumption of caffinated beverages, including coffee, soft drinks, chocolate drinks, etc.. Consumption of the latter is based on self-reported intake two years prior to the interview. Lifetime consumption of cigarettes (in packyears), and alcoholic beverages, including beer, wine, hard liquor, and total alcohol, was also examined in relation to organochlorine levels. Cigarette consumption was also assessed as a categorical variable, in which the sample is divided into never, former, and current smokers. The effect of place of residence was examined in categories of women who have urban, suburban, or rural places of residence.

Dietary variables were combined into general food groups, such as fruit, vegetables, grains, dairy, fish, poultry, beef, pork, bacon, total red meat, and total processed meat. The units of measurement of these variables were grams per months and were based on self-reported intake two years prior to the interview.

Several reproductive variables were compared to organochlorine levels. Months of lactation was computed for all participants, including the nulliparous women . A second months of lactation variable was computed for women with at least one livebirth. However, associations with organochlorines did not differ between these lactation variables, thus reported results were based on months of lactation among parous and nulliparous women. The effect of pregnancy on organochlorine levels was assessed though parity, as a continuous and categorical variable, and total weeks of pregnancy. Additional reproductive variables were age at menarche and menopause, as well as total months menstruated.

Finally, the effect of having been breastfed was examined in this analysis. However, only 72 percent of the women in this sample could provide information on this variable.

As for medical history, four dichotomous variables were examined. Specifically, women with or without prior history of births control pill use, hormone replacement therapy use, benign breast disease, and family history of breast cancer were compared with regard to serum organochlorine levels.

Four occupational categories were created for this analysis. These categories reflect self-reported occupation two years prior to the interviews and include professional occupations, sales and administrative occupations, service occupations, and labor occupations. The last category includes a variety of reported occupations, including farm workers, machine operator, labor inspector, and precision, craft, or repair worker.

C. Laboratory Analyses

Toxicological analyses of PCB congeners, DDE, HCB and mirex were performed by the Toxicology Research Center Analytical Laboratory at SUNYAB (TRC). The analytic procedures for PCB congeners, DDE and mirex and the QA/QC methods and data reporting have been adapted from the methods of Dr. Brian Bush of the New York State Department of Health (Bush 1982, Bush 1983, Hong 1992). Small batches of ten samples were send to the TRC. Vials for PCB analyses will include a blank, one duplicate, a spiked sample, and a quality control check or reference sample. Empty vials will first be tested to ensure that no materials from these vials will contaminate the sera, particularly important for the PCB analysis.

(1) Sample Extraction and Clean-up: The extraction and clean-up procedures for separation of non-planar and coplanar PCBs and gas chromatographic conditions for non-planar congeners are as follows. The method consists of adding alcohol to 5g of serum and extracting with hexane. The extract is concentrated by evaporation under nitrogen to a final volume of 2 mL and placed on the top of a 10 x 500 mm column containing 10g of 4% deactivated florisil topped by a layer of anhydrous sodium sulfate and eluted with 60ml of hexane. The hexane eluate is concentrated to 50 ml and injected onto a porous graphitic HPLC (Hypercarb) column for separation into non-coplanar and coplanar PCB fractions. The non-coplanar PCBs as well as DDE and mirex are collected in the first 4 ml of hexane

eluting the HPLC column in the forward direction. The coplanar PCB fraction consisting of 12 ml of hexane is collected in the reverse flow mode. These fractions are concentrated to 0.5 and 0.1 ml respectively, prior to analysis by capillary GC with electron capture detection (GC/EC).

(2) Chromatographic Determinations: Two GC-EC systems are used for the analyses, one for the non-planar congeners using a SPB-5 column and a second equipped with a HP-Ultra 2 column to separate and quantify the coplanar congeners. The latter also provides confirmation of the results of the first system. All data generated by the two chromatographs is transmitted to an IBM PC computer by the 2600 PE-Nelson system. The retention of individual congeners are determined from the analysis of mixtures of Aroclor and congener standards. A congener identification table was established from the relative retention time of each congener, to the internal standard retention time by the data system.

The detector responses are calibrated using congener standard mixtures of IUPAC #52, #6, #205, #101, #185, #44, #153, #138 and #180, as well as HCB, DDE, mirex, and the internal standards having IUPAC #30 and #204, for the non-planar congeners. The detector responses for the second GC/EC system used for the analyses of the coplanar PCBs, are calculated using the calibration mixture of all coplanar congeners: #77, #81, #105, #114, #118, #123, #126, #156, #167, #169 and #189, plus the internal standards #30 and #204. The congeners with retention times lower or equal to that of #110 are calibrated using #30 as the internal standard, those with retention times greater than #110, use #204 as the internal standard. Three concentrations of the congener calibration mixtures are used in the range of expected concentrations to obtain a calibration curve for each of these congeners. The response factors obtained with these mixtures are related to chlorine content and used for other congeners in the complex Aroclor mixtures. An Aroclor standard consisting of a 1:1:1:1 mixture of EPA repository AR-1016, -1221, -1254 and -1260 at 200 ng/ml each, plus 10 ng/ml each of p,p'-DDE and mirex, is used to calculate the relative retention times and response factors for the individual PCB congeners. These congeners were chosen because they are present at different concentrations in the Aroclor mixtures found in the Western New York region. At least five percent (5%) of the samples

will be run for confirmation by GC/EC on a second column or if needed by GC/MS analysis on a Kratos MS80 instrument. The PE/Nelson System calculates the response factors for each congener relative to the internal standard, generates the calibration curves and calculates the concentration of each PCB congener. A batch processor generates a summary table for each congener in a batch of ten samples and QC controls and transfers the information to a spreadsheet (Quattro Pro). The transfer of results is all done electronically without the need of entering values manually, thus eliminating common errors of data entry always present when entering a great volume of numbers. The results of the QC controls are logged in the appropriate QC control charts.

(3) QA/QC Methods and Data Reporting: The general procedures of the laboratory follow the QC/QA protocol of the NY Department of Health and EPA recommended practices. Namely, for each batch of ten samples the following QC samples are also analyzed: a blank, a spiked sample, a QC check and a duplicate sample. All these controls are associated with a specific batch of samples by a unique batch number. These procedures are followed for all analytes. A blank sample consists of an equal amount of a similar matrix sample containing a minimal amount, or no analyte, (i.e. sheep serum). A spiked sample is a randomly selected sample to which a solution containing a selected concentration of analytes is added. A QC check sample is a matrix blank (ex. sheep blood) to which a solution containing a selected concentration of the analyte(s) has been added. A duplicate sample is a second aliquot of a randomly chosen sample. QC charts are kept to track each QC control in computerized spreadsheets, where the data is categorized by date of the analyses, and batch number. These QC controls are grouped by sample matrix.

Control charts for QC check samples are built at the beginning of the project by measuring the mean and the standard deviation of the concentration of the analytes for ten samples. The control limits are set as the mean ± 3 S.D. Any QC check sample which falls outside these limits, and the batch associated with it, has to be re-analyzed. A warning limit (WL) is also plotted for the mean ± 2 S.D. as an indication of potential problems. The values for the QC blanks are tabulated, the acceptable range is equal or less than two times the limit of detection for the particular analyte. A table with the values for the spike samples, the corresponding non-spiked sample and the amount of each analyte added is

kept. The percent recovery is calculated for each spike sample and control charts similar to those for the QC check samples are kept.

The routine GC/EC analysis of PCBs are preceded by a series of QA/QC runs. The order of injection and runs are: hexane solution (to check the response of the instrument), the calibration standards, the QC check and the blank extract. The quantitation results are evaluated for compliance with quality control charts. If acceptable, the samples are run. The analyses then consists of a series of four specimen samples plus a calibration standard. This sequence is repeated until the total batch is analyzed. The samples are re-analyzed if the blank and/or the QC check controls are outside the control limits established for these analyses.

D. Statistical Analysis

Frequency of occurrence of serum organochlorine levels was determined by obtaining a ratio of the number of women with detectable levels to the total number of women in the study sample (n=193). Magnitude of occurrence among those with detectable levels was demonstrated by obtaining measures of central tendency (mean, median, mode) and the ranges of detected values. This was performed for DDE, HCB, mirex, total PCBs, as well as for 56 PCB congeners.

Correlates of organochlorine levels were investigated for DDE, HCB, mirex, total PCBs, and all PCB congeners for which the recovery rate was greater than 90 percent (congeners 180, 153, 138, 118, 203+196, and 194). The distribution of DDE, total PCBs, and all PCB congeners were skewed to the right, thus natural log transformations of these variables were performed prior to statistical analyses. HCB levels were normally distributed and were not transformed. Mirex was detected in less than one quarter of the sample, and this variable was dichotomized into groups of women with and without detectable levels. For continuous predictor variables Pearson and Spearman correlation coefficients were computed to evaluate the direction and strength of possible association with exposure variables. Partial correlation coefficient were obtained in order to account for the effect of age and serum lipids on organochlorine levels. For categorical variables mean values of these compounds were computed for all categories. Analysis of covariance (ANCOVA) was utilized to obtain adjusted means for the categories. Covariates in this analysis were

serum lipids and age. Multiple linear regression analysis was used to identify significant predictors of serum organochlorine levels. Model construction was initiated with a basic regression equation that included age, serum triglycerides, and serum cholesterol. Previously identified correlates of a particular dependent variable were included into the model, using the stepwise method. Inclusion into the final regression model was based upon statistical significance at the .10 level and contribution to the overall ability of the model to explain the observed variance.

RESULTS

A. Prevalence of organochlorines

Table 1 depicts the frequency of occurrence and descriptive characteristics of the organochlorines in this study sample of 193 postmenopausal western New York residents. The mean values reported are based on measures for participants with detectable levels. Nearly all participants had detectable levels of DDE (99%), total PCBs (100%), and HCB (98%), with mean values of 10.76 ng/g, 4.06 ng/g, and 0.43 ng/g, respectively. DDE levels varied greatly ranging from 0.24 to 76.22 ng/g. Less than one quarter of the sample had detectable levels of mirex with a mean of 0.15 ng/g.

Gas chromatography provided measures for 56 individual PCB congeners displayed in Tables 2 to 5. For one fifth of the congeners (n=11) detectable levels were found for more than 75 percent of the sample (Table 2). All participants had detectable levels for congeners 180, 153, and almost all for 138, 118, 203+196, and 194. These most frequently occurring congeners are predominantly members of the moderately to highly chlorinated isomer groups; specifically pentachlorobiphenyls (#118 & #105+132), hexachlorobiphenyls (#138 & #153), heptachlorobiphenyls (# 180, #177, #187, #188), and octachlorobiphenyls (#194, #200, #203+196). The highest mean levels (> 0.4 ng/g) were observed for congeners 153, 138, 180, and 118, whereas low levels (< 0.10 ng/g) were associated with congeners 200, 177, and 188.

PCB congeners for which more than 50 percent of the study sample had detectable levels are displayed in Table 3. This group of five congeners (9% of all

available congeners) consisted of three hepatchlorobiphenyls (#171+156, #176, #183), one octachlorobiphenyl (#195), and one nonachlorobiphenyl (#206). The mean values for individual congeners among participants were highest for congener 206 (0.16 ng/g) and lowest for congener 195 (0.07 ng/g).

Individual PCB congener for which the frequency of detection ranged between 49 and 25 percent are displayed in Table 4. These five congeners are members of the lower to moderately chlorinated isomer groups, with one di-, tri-, and tetrachlorinatedbiphenyl (#7+9, #31+28, and #47+48, respectively, and two hexachlorinatedbiphenyls (#141+179 & 128+167). All congeners in this group happen to be mixed peaks, for which it is not possible to determine the individual level of each congener within a peak. Mean levels vary greatly, ranging from as low as 0.04 for congeners 128+167 to 0.65 for congeners 47+48.

For 63 percent (e.g., 35) of the PCB congeners under investigation less than 25 percent of the participants had detectable levels (Table 5). Further, most of these congeners were detected in fewer than ten percent of the participants. The majority of these congeners were members of the lower chlorinated isomer groups, specifically dichlorobiphenyl (#6), trichlorobiphenyls (#19, #18, 15+17, #24+27, #16+32), and tetrachlorobiphenyls (#60, #55, #66+95, #70, #40, #59=42, #44, #49, #52, #45); however, other isomer groups were also represented, such as pentachlorobiphenyls (##87, #97, #99, #101), hexachlorobiphenyls (#129, #134, #149, #147, #135, #151+82, #136), heptachlorobibhenyls (##172, #174+181, #185), and octachlorobiphenyl (#205). Mean values for these congeners vary, ranging from 0.02 ng/g for congener 77+110 to 0.90 ng/g for congener 99. However, these estimates are based on particularly small numbers and should be viewed with caution.

Subsequent analyses were designed to identify potential correlates and predictors of organochlorines among healthy postmenopausal women. This investigation focused on serum DDE, total PCB's, HCB, mirex levels, and several individual PCB congeners, those for whom at least 90 percent of this study population had detectable levels. For the purpose of these analyses, participants who had values below the LOD for DDE, HCB, and some of the PCB congeners were assigned levels

of one half of the LOD for that organochlorine. For mirex, less than one quarter of the sample had detectable levels of these compound; therefore this outcome variable was transformed to a dichotomous variable, characterized by women with and without detectable mirex levels.

B. Demographic and lifestyle variables

Associations between several demographic and lifestyle variables and serum organochlorine levels are shown in Table 6. Measures of associations utilized here included Pearson and Spearman correlation coefficients, as well partial coefficients adjusted for age and serum lipids (e.g., cholesterol and triglycerides). Following are some of the pertinent Pearson correlations. Age was associated with serum DDE (r=0.16), total PCBs (r=0.26), HCB (r=0.30), as well as with PCB congeners 180 (r=0.26), 153 (r=0.17), 118 (r=0.20), 203+196 (r=0.27), and 194 (r=0.22). Education was not significantly associated with any of the exposure variables under investigation. DDE was weakly positively related to quetelet index (r=0.14). A somewhat stronger positive association with quetelet index was observed for congener 118 (r=0.20). Inverse associations with quetelet index were found for congeners 180 (r=-0.20) and congener 194 (r=-0.20). Weight in pounds was positively associated with congeners 203+196 (r=0.18), although this association disappeared after control for age and serum lipids. A positive association with weight and congener 118 was observed after adjustment for age and lipids (partial r=0.21). In contrast, weight in pounds was inversely associated with congeners 180 (r=-0.25) and 194; for the latter, though, the association became apparent only after age and serum lipids were controlled for (partial r=-0.21). Strenuous physical activity was inversely related to some of these compounds after adjustment for age and serum lipids; e.g. DDE (partial r=-0.19), total PCBs (partial r=-0.30), congener 118 (partial r=-0.26), and congener 153 (partial r=-0.28). A positive association with strenuous physical activity was observed for HCB (r=0.29), however this association disappeared after adjustment for age and lipids (partial r=0.05). Several compounds were positively related to hours of walking for recreation or transportation. These compounds include total PCBs (r=0.17), congener 180 (r=0.16), congener 153 (r=0.22), congener 138 (r=0.15), congener 118

(r=0.22), and congeners 203+196 (r=0.16). Total dietary fat intake was not related to serum organochlorine levels, with the exception of a weak inverse relationship between total fat and HCB (r=-0.15). Similarly, total water intake was not related to any of the compounds under investigation. Total Caffeine intake was inversely related to the following compounds: DDE (r=-0.20), total PCBs (r=-0.20), HCB (r=-0.16), congener 153 (r=-0.16), congener 118 (r=-0.24), and congener 203+196 (r=-0.16). The variable packyears of cigarettes smoked was only associated with congener 118 (r=-0.25). Total alcohol consumption, and wine, beer, or hard liquor consumption were not related to serum levels of these compounds. However, wine consumption appeared to be inversely related to congener 138 (r=-0.17).

C. Dietary Variables

Pearson, Spearman, and age and lipid adjusted correlation coefficients for the association between dietary variables and organochlorine levels are displayed in Table 7. Monthly intake of various food categories (in grams) was examined in relation to serum measures of these compounds (ng/g). Positive associations with fruit intake were observed for DDE (Pearson r=0.18) , total PCBs (Pearson r=0.14) and HCB (Pearson r=0.17). Serum DDE was weakly associated with vegetable (Pearson r=0.13) and dairy intake (Pearson r=0.12) . Spearman correlations revealed inverse relations between beef (r=-0.14), total red meat (r= -0.15), and total processed meat (r= -0.15) and HCB levels, although these relations were not reflected in Pearson and partial correlations. No associations with any food category were observed for serum mirex levels. As for individual PCB congeners, fruit intake was positively associated with congeners 153 (r=0.20) and 138 (r=0.16), and vegetable intake was related to congeners 180 (r=0.13), 153 (r=0.15), 138 (r=0.16), and 203+196 (r=0.13). Fish intake was positively related to congener 153 (r=0.14), and there was a weak inverse association for congener 194 and bacon intake (r=-0.15). No associations were found for any of the compounds under investigation with grain and poultry intake.

D. Reproductive Variables

Associations between serum organochlorine levels and reproductive characteristics are shown in Table 8. Contrary to expectation, decreasing

organochlorine levels in this study population did not emerge as a function of increasing duration of lactation. Parity, however, was inversely related to DDE (r=-0.17) and total PCBs (r=-0.15). In addition, total weeks pregnant was inversely associated with DDE (Pearson r=-0.16). Total months menstruated was inversely associated with mirex levels (r=-0.16), although this association was not present after adjustment for age and lipids (partial r=0.06). This variable was positively related with congener 118 (r=0.16) and HCB, however for the latter this association was more pronounced when the Spearman coefficient was considered (r=0.19), and was not present when age and lipids were adjusted for (partial r=0.06). Age at menarche and age at menopause were not related to these compounds. Weak associations were only observed for serum mirex levels, which was positively associated with age at menarche (Spearman r=0.14) and inversely associated with age at menopause (Spearman r=-0.15).

E. Categorical variables

Analysis of covariance (ANCOVA) was employed to obtain adjusted means for serum levels all organchlorines in different groups of categorical predictor variables. The following tables present crude means as well as means adjusted for serum lipids alone, for age alone, and for serum lipids and age. Differences in means were examined among dichotomous variables and variables with three or more categories. As for the latter, group means were compared to a specified reference group. In general, discussions concerning differences between means will be restricted to means adjusted for serum lipid and age.

Due to the lack of an observed inverse association between duration of lactation and serum organochlorine levels, lactation was treated as a categorical variable and mean organochlorine levels were compared among women who reported that they never breastfed an infant with those that reported that they ever breastfed (Table 9). No statistically significant differences were apparent for the two groups. Serum DDE levels tended to be higher among women who never breastfed compared to those who did (adjusted means 11.53 ng/g and 10.24 ng/g, respectively). No differences were observed for the remaining compounds; in fact total PCBs and

congeners 153 and 138 were elevated in women had breastfed in comparison to those who have not.

The relationship between parity as a categorical variable and serum organochlorine levels is presented in Table .10. Women with differing numbers of children were compared to nulliparous women. Age and lipid adjusted mean DDE levels of nulliparous women (adjusted mean=12.62 ng/g) did not differ significantly from those of women in any of the other parity groups. Nevertheless, these women had markedly higher levels than women with four children (adjusted mean=8.58 ng/g) and five or more children (adjusted mean=8.08 ng/g). The highest DDE levels were observed among women with one child (adjusted mean=13.10 ng/g). Similarly, for total PCBs, women with one child had significantly higher levels than nulliparous women (adjusted means 5.46 ng/g and 3.64 ng/g, respectively). The lowest serum PCB levels were observed among women with five or more children (adjusted mean=3.44 ng/g). Serum levels of HCB and mirex were similar across parity groups. Women with one child had significantly higher levels of congeners 180, 153, and 138 (adjusted means 0.63 ng/g, 1.22 ng/g, 0.98 ng/g, respectively) than nulliparous women (adjusted means 0.46 ng/g, 0.76 ng/g, 0.62 ng/g, respectively). Levels of women with five or more children were similar to those of nulliparous women. No differences with regard to serum levels of congeners 118, 203+196, and 194 were observed across the parity groups.

Crude and adjusted means of serum organochlorine levels among postmenopausal women who either were or were not breastfed as an infant are displayed in Table 11. There was no statistically significant difference for the two groups with regard to age and lipid adjusted mean DDE levels, although levels of those women who were breastfed were higher than those of women who were not (adjusted means 10.29 ng/g and 6.17 ng/g, respectively). It should be pointed out, however, that the mean of those who were not breastfed is based on a small number of women (n=15).There were no differences between the groups for any of the other organochlorines under investigation.

Mean organochlorine levels of women who reported prior use of birth control bills in comparison to women who reported no such use are presented in Table 12. Slight differences between the groups were apparent for crude DDE levels, but after adjustment for age and lipids, levels for women who used birth control pills did not differ from those who did not (adjusted means 9.63 ng/g and 10.19 ng/g, respectively). Similarly, for total PCBs and HCB; differences between the groups were observed when crude means were considered, but these differences decreased in size once age and lipids were controlled for. Women who never used birth control bills had adjusted mean levels of 4.05 ng/g for PCBs and 0.41 ng/g for HCB, whereas women who did use birth control pills had levels of 3.41 ng/g and 0.36 ng/g, respectively. Adjustment for age and lipids did not markedly change mean levels of mirex and the individual PCB congeners. Although no significant differences were observed for any of these compounds, the overall observation of higher levels among women with no prior use of birth control use prevailed.

In Table 13 is displayed serum organochlorine levels among women who did and did not use postmenopausal hormone replacement therapy (HRT). In general, there were no statistically significant differences between the age and serum lipid adjusted means of women who reported to have ever used HRT and those who never used HRT. Mean DDE levels tended to be higher among HRT user than among nonusers (adjusted means 11.61 ng/g and 10.39 ng/g, respectively), and lower levels for these groups were observed for total PCBs (adjusted means 3.88 ng/g and 4.15 ng/g, respectively) and congener 118 (adjusted means 0.38 ng/g and 0.44, respectively).

Crude and adjusted mean organochlorine levels among with and without previous benign breast disease are compared in Table 14. These two groups are very similar with regard to mean levels for any of the compounds under investigation.

Organochlorine levels of women with or without a family history of breast cancer are shown in Table 15. Although no statistically differences between the groups were observed for any of the compounds, it appeared that women with a family history of breast cancer had higher levels of DDE (adjusted mean=11.70 ng/g), mirex

(adjusted mean=0.07 ng/g), congener 153 (adjusted mean=0.92 ng/g), congener 138 (adjusted mean=0.82 ng/g), and congener 118 (adjusted mean=0.53 ng/g) than women with no such history (adjusted means 10.40 ng/g, 0.03 ng/g, 0.83 ng/g, 0.63 ng/g, and 0.40 ng/g, respectively).

Differences in means between groups of women with variable smoking status are presented in Table 16. Age and lipid adjusted means of former and current smokers were compared to never smokers. These groups had similar levels of DDE, total PCBs, HCB, mirex, and most of the PCB congeners. However, former and current smokers had significantly lower levels of congener 118 than women who never smoked (adjusted means 0.37 ng/g, 0.31 ng/g, and 0.49 ng/g).

Mean organochlorine levels among women with professional occupations, sale or administrative occupations, service occupations, and labor occupations are presented in Table 17. Differences in means were be examined among all groups. No significant difference between these occupational groups were observed for DDE and PCB levels. For DDE the highest levels were observed among women in labor occupations (adjusted means=12.12 ng/g) and lowest levels among women in sale and administrative occupations (adjusted mean=10.09 ng/g); for total PCBs professional women had the highest levels (adjusted mean=4.51 ng/g), whereas lowest levels were observed among women in sale and administrative occupations (adjusted mean=4.00 ng/g). Serum HCB levels for women in service and sale and administrative occupations were significantly lower than those of professional women (adjusted means 0.39 ng/g, 0.41 ng/g, and 0.48 ng/g, respectively). No significant differences were observed for mirex and any PCB congener, although highest levels for professional women were found for congeners 153, 138, and 194.

Data shown in Table 18 compares organochlorine levels with regard to current place of residence. Again, differences between means will be examined among all groups, specifically, urban, suburban, and rural residents. Suburban women had significantly lower DDE and mirex levels (adjusted means 9.39 ng/g and 0.02 ng/g, respectively) than urban women (adjusted means 12.51 ng/g and 0.05 ng/g, respectively). There was also a significant difference with regard to total PCBs, with

lower levels for suburban women (adjusted mean=3.96 ng/g) than for rural women (adjusted mean=4.43 ng/g). As for HCB, rural women had significantly higher levels than urban women (adjusted means 0.47 ng/g and 0.40 ng/g, respectively). No significant differences were observed for most of the PCB congeners. Congener 180 levels were significantly higher among suburban women (adjusted mean=0.52) than among urban women (adjusted mean=0.45 ng/g) and rural women had significantly higher levels of congener 138 (adjusted mean=0.75 ng/g) than urban women (adjusted mean=0.63 ng/g).

F. Multiple linear regression analysis

In subsequent analysis multiple regression analysis was utilized to identify significant correlates of serum DDE, total PCB, HCB, as well as PCB congeners 180, 153, 138, 118, 203+196, and 194. Model construction for each of these independent variables was initiated with a basic regression equation that included age, serum cholesterol, and serum triglycerides as independent variables. Additional independent variables were selected based upon biological relevance (e.g., duration of lactation and quetelet index) and upon observed associations with the outcome variables in previous analyses. Some of the organochlorines under investigation were weakly correlated with fruit and vegetable intake. For these variables associations with individual fruits and vegetables were examined. Individual fruits and vegetables associated with the organochlorines were included in the corresponding exploratory regression models. Inclusion into the final regression model was based on upon statistical significance at the .10 level and contribution to the overall ability of the model to explain the observed variance.

The multiple linear regression model for serum DDE is presented in Table 19. This model explained 28 percent of the observed variance in serum DDE levels. The major contributor to the explained variance was serum PCBs (partial r^2=0.12; β=0.19). Without the inclusion of total PCBs into the model, 16 percent of the variance is explained. Both parity (β=-0.09) and caffeine intake (β=-2.74) were statistically significant predictors and contributed four percent to the explained variables. Three percent of variance was explained by the dichotomous variables suburban residence

(β=-0.39) and having been breastfed as an infant (β=0.56). Finally, total fruit intake was a significant predictor of serum DDE levels, although the partial r^2 was small (partial r^2=0.02; β=3.15). Age and serum lipids did not contribute to the model and the independent variables remained significant when these potential confounders were included into the model. In addition, suspected predictors of serum DDE levels, such as duration of lactation and quetelet index, as well other variables previously found to be associated with serum DDE, including some individual fruits and vegetables, did not reach statistical significance, nor did they contribute to explain the observed variance.

The regression model for total PCBs (Table 20) accounted for 42 percent of the observed variance in serum PCB levels. The major contributors to the model were serum DDE (partial r^2=0.14; β=0.01) and serum HCB (partial r^2=0.10; β=0.91), together explaining 24 percent of the variance. In absence of the organochlorines in the model, 18 percent of the variance was explained. Age was an additional strong predictor of serum total PCB levels (partial r^2=0.06; β=0.01). Other independent variables that were included in the model were serum triglycerides (partial r^2=0.04; β=0.001), cherry intake (partial r^2=0.03; β=2.47), caffeine intake (partial r^2=0.02; β=-8.58), asparagus intake (partial r^2=0.02; β=3.02), and cucumber intake (partial r^2=0.01; β=4.26). Serum cholesterol did not contribute to the overall model and its inclusion did not alter the effect of the independent variables levels included in the model. Furthermore, other previously identified correlates of total PCB levels or duration of lactation and quetelet index were not significant predictors of total PCBs.

Results from the multiple linear regression analysis for serum HCB are shown in Table 21. The final model explained 43 percent of the observed variance, although the major contributor to the model was serum PCB (partial r^2=0.26; β=0.05). 17 percent of the variance was explained by the model that excluded total PCBs. Age was a strong predictor for HCB levels, explaining nine percent of the variance (β=0.01). Dietary predictors of HCB were intake of prunes or plums (partial r^2=0.05; β=1.4) and asparagus intake (partial r^2=0.03; β=1.47). These results did not change when serum

lipids were controlled for. Potential predictor variables such as serum DDE, duration of lactation, quetelet index, and other previously identified correlates did not contribute significantly to the model.

The regression model for serum levels of PCB congener 180 explained 48 percent of the observed variance (Table 22). Again, other organochlorines were the major contributors to the model. Serum HCB explained 13 percent (β=0.95) and serum DDE explained seven percent of the variance (β=0.001). The regression model that did not include these organochlorines explained 28 percent of the variance. Age (β=0.01) and lettuce intake (β=1.19) were strong predictors of serum levels of congener 180 with partial r^2 of 0.08 for both variables. Additional variables included in the final model were weight (partial r^2=0.04; β=-0.003), serum triglycerides (partial r^2=0.04; β=9.05), intake of prunes or plums (partial r^2=0.02; β=2.02), and having one child only (partial r^2=0.02; β=0.21). Inclusion of serum cholesterol did not affect these results. Again, hypothesized predictors of serum congener 180 levels, lactation and quetelet index, or other previously identified correlates did not reach statistical significance, nor did they contribute in explaining the observed variance.

Results from the multiple regression analysis for PCB congener 153 are presented in Table 23. The final model explained 41 percent of the observed variance, of which more than half is explained by serum DDE levels (partial r^2=0.20; β=0.02) and serum HCB (partial r^2=0.04; β=0.62). When these organochlorines were not included in the model, 17 percent of the variance was explained. Contributors to this model were serum triglycerides (partial r^2=0.05; β=0.001), dietary asparagus intake (partial r^2=0.04; β=2.71), having one child only (partial r^2=0.03; β=0.31), age (partial r^2=0.02; β=0.01), dietary lettuce intake (partial r^2=0.02; β=1.18), and dietary cherry intake (partial r^2=0.01; β=1.78). Further adjustment for serum cholesterol, did not affect these results. Duration of lactation, quetelet index, and other previously identified correlates of congener 153 did not reach statistical significance or contribute to the final model.

The multiple linear regression model for PCB congener 138 is displayed in Table 24. This model explains 29 percent of the observed variance, primarily contributed to serum HCB (partial r^2=0.12; β=1.55) and serum DDE (partial r^2=0.05; β=0.02), and 12 percent were explained when these organochlorines were not entered into the regression model. Other independent variables that contributed to the latter model included dietary lettuce intake (partial r^2=0.05; β=1.42) asparagus intake (partial r^2=0.02; β=4.41), wine consumption (partial r^2=0.02; β=-5.73), having a family history of breast cancer (partial r^2=0.02; β=0.32), and intake of prunes or plums (partial r^2=0.01; β=4.58). Adjustment of age and serum lipids did not alter these results, nor did these variables or other suspected predictors of serum levels of congener 138, such as lactation and quetelet index, contribute to the regression model.

The regression model for PCB congener 118 explained 31 percent of the observed variance (Table 25) when serum DDE (partial r^2=0.05; β=0.23) was included, and 26 percent when this compound was not entered into the model. In this model no major contributor to the model was identified, rather moderate contributors included never smoker status (partial r^2=0.05; β=0.26), age (partial r^2=0.04; β=0.01), weekly hours of walking (partial r^2=0.04; β=0.35), and quetelet index (partial r^2=0.04; β=0.03). Additional, somewhat weaker predictors included dietary asparagus intake (partial r^2=0.03; β=5.88), dietary cereal intake (partial r^2=0.02; β=5.50), suburban residence (partial r^2=0.02; β=-0.24), and serum cholesterol (partial r^2=0.02; β=0.003). Inclusion of serum triglycerides did not change these observations, however triglycerides or other suspected predictors of serum congener 118 levels or duration of lactation did not contribute to the explanation of the observed variance.

The regression model for PCB congeners 203+196 is presented in Table 26. This model explained 25 percent of the variance when serum DDE (partial r^2=0.03; β=0.01) and serum HCB (partial r^2=0.02; β=0.80) were allowed into the model, and 20 percent of the variance was explained by a model that excluded these compounds. Age was the strongest predictor (partial r^2=0.07; β=0.02) of PCB congeners 203+196. Additional variables included in the model were lettuce intake (partial r^2=0.04;

β=1.72), serum triglycerides (partial r^2=0.03; β=0.001), intake of prunes or plums (partial r^2=0.02; β=4.13), serum cholesterol (partial r^2=0.02; β=0.003), weekly hours of walking (partial r^2=0.02; β=0.22).Other correlates and lactation and quetelet index did not significantly contribute to this model.

Finally, the regression model for congener 194 explained 20 percent of the variance when serum HCB (partial r^2=0.07; β=1.11) was included in the model (Table 27). The model that did not include this organochlorine explained 13 percent of the variance and included age (partial r^2=0.04; β=0.02), quetelet index (partial r^2=0.04; β=-0.03), triglycerides (partial r^2=0.04; β=0.002), and intake of prunes or plums (partial r^2=0.01; β=3.61). Further adjustment for serum cholesterol did not affect these estimates and duration of lactation did not contribute to the model.

Table 1

Serum DDE, Total PCBs, HCB and Mirex: Descriptive Data for Postmenopausal Women - Western New York 1986-1991.

			Measures for participants with detectable levels (>LOD)			
(ng/g)	LOD[1]	%>LOD (n)	mean (SD)	median	min.	max.
DDE	.09	99% (192)	10.76 (10.63)	8.04	0.24	76.22
Total PCBs	na	100% (193)	4.06 (2.24)	3.62	0.94	19.04
HCB	0.12	98% (190)	0.43 (0.192)	0.39	0.14	1.35
Mirex	0.06	23% (44)	0.15 (0.16)	0.10	0.06	0.99

[1] LOD = limit of detection

Table 2

Serum Levels of PCB Congeners with Recovery Rate of Greater than 75 Percent: Descriptive Data for Postmenopausal Women - Western New York 1986-1991.

			Measures for participants with detectable levels (>LOD)			
Congener (ng/g)	LOD[1]	%>LOD (n)	mean (SD)	median	min.	max.
#180	0.02	100% (193)	0.49 (.25)	0.42	0.07	2.22
#153	0.09	100% (193)	0.83 (0.48)	0.76	0.10	4.77
#138	0.05	98% (189)	0.67 (0.44)	0.57	0.11	4.0
#118	0.09	97% (187)	0.44 (0.31)	0.34	0.09	2.48
#203+196	0.01	96% (186)	0.11 (0.06)	0.09	0.02	0.40
#194	0.03	96% (186)	0.14 (0.12)	0.11	0.03	0.99
#177	0.02	89% (172)	0.06 (0.08)	0.04	0.04	0.99
#200	0.00	88% (169)	0.03 (0.08)	0.02	0.01	0.99
#187	0.03	81% (156)	0.18 (0.17)	0.14	0.02	1.65
#188	0.03	81% (156)	0.08 (0.04)	0.07	0.03	0.30
#105+132	0.04	77% (148)	0.11 (0.07)	0.09	0.05	0.50

[1] LOD = limit of detection

Table 3

Serum Levels of PCB Congeners with Recovery Rate of Greater than 50 Percent: Descriptive Data for Postmenopausal Women - Western New York 1986-1991.

			Measures for participants with detectable levels (>LOD)			
Congener (ng/g)	LOD[1]	%>LOD (n)	mean (SD)	median	min.	max.
#171+156	0.05	69% (133)	0.14 (0.10)	0.12	0.05	0.88
#176	0.01	63% (122)	0.11 (0.17)	0.06	0.01	1.12
#206	0.08	63% (121)	0.16 (0.08)	0.13	0.08	0.51
#183	0.03	62% (120)	0.09 (0.15)	0.05	0.03	0.99
#195	0.02	57% (110)	0.07 (0.10)	0.05	0.02	0.99

[1] LOD = limit of detection

Table 4

Serum Levels of PCB Congeners with Recovery Rate of Greater than 25 Percent: Descriptive Data for Postmenopausal Women - Western New York 1986-1991

			Measures for participants with detectable levels (>LOD)			
Congener (ng/g)	LOD[1]	%>LOD (n)	mean (sd)	median	min.	max.
#31+28	0.21	38% (73)	0.34 (0.13)	0.28	0.21	0.80
#7+9	0.02	38% (74)	0.14 (0.10)	0.13	0.02	0.66
#141+179	0.03	38% (74)	0.05 (0.03)	0.04	0.03	0.17
#47+48	0.03	37% (72)	0.65 (0.30)	0.06	0.03	0.14
#128+167	0.01	31% (60)	0.04 (0.03)	0.04	0.01	0.18

[1] LOD = limit of detection

Table 5

Serum Levels of PCB Congeners with Recovery Rate of Less than 25 Percent: Descriptive Data for Postmenopausal Women - Western New York 1986-1991

			Measures for participants with detectable levels (>LOD)			
Congener (ng/g)	LOD[1]	%>LOD (n)	mean (SD)	median	min.	max.
#147	0.01	22% (42)	0.03 (0.02)	0.02	0.01	0.16
#66+95	0.19	20% (38)	0.25 (0.06)	0.24	0.19	0.49
#22	0.11	16% (32)	0.16 (0.05)	0.16	0.11	0.29
#101	0.15	16% (30)	0.20 (0.06)	0.18	0.15	0.39
#87	0.03	15% (28)	0.06 (0.02)	0.05	0.04	0.12
#134	0.02	15% (29)	0.04 (0.03)	0.03	0.02	0.15
#129	0.09	14% (27)	0.16 (0.09)	0.12	0.09	0.47
#205	0.01	13% (25)	0.07 (0.12 0	0.02	0.01	0.41
#77+110	0.00	12% (23)	0.02 (0.01)	0.01	0.01	0.04
#174+181	0.02	12% (23)	0.06 (0.03)	0.06	0.03	0.13
#172	0.06	11% (21)	0.14 (0.10)	0.10	0.06	0.42
#6	0.11	11% (21)	0.18 (0.04)	0.18	0.12	0.26
#185	0.02	9% (18)	0.07 (0.06)	0.05	0.02	0.30
#151+82	0.05	9% (18)	0.08 (0.05)	0.06	0.05	0.24
#99	0.63	9% (17)	0.90 (0.14)	0.87	0.64	1.1
#135	0.03	8% (16)	0.05 (0.02)	0.05	0.03	0.09
#60	0.07	8% (15)	0.11 (0.05)	0.09	0.07	0.21
#18	0.48	8% (15)	0.52 (0.03)	0.51	0.48	0.57
#25+50	0.32	7% (13)	0.45 (0.26)	0.35	0.11	1.14
#149	0.08	7% (13)	0.12 (0.05)	0.10	0.08	0.25
#55	0.03	7% (14)	0.04 (0.01)	0.04	0.03	0.06

#45	0.08	7% (13)	0.10 (0.02)	0.09	0.08	0.14
#44	0.16	5% (10)	0.21 (0.05)	0.19	0.17	0.30
#15+17	0.22	5% (10)	0.35 (0.26)	0.24	0.23	1.06
#97	0.05	4% (7)	0.07 (0.01)	0.07	0.06	0.09
#70	0.11	4% (7)	0.14 (0.05)	0.12	0.10	0.23
#40	0.09	4% (7)	0.21 (0.14)	0.15	0.09	0.45
#59+42	0.14	4% (7)	0.17 (0.02)	0.17	0.14	0.21
#16+32	0.28	4% (8)	0.31 (0.02)	0.30	0.28	0.35
#136	0.08	2% (4)	0.11 (0.01)	0.11	0.09	0.12
#49	0.70	2% (4)	0.89 (0.14)	0.89	0.73	1.03
#52	0.24	2% (3)	0.29 (0.04)	0.31	0.25	0.32
#33	0.45	1% (2)	0.36 (0.23)	0.36	0.20	0.52
#19	0.23	1% (2)	0.378 (0.05)	0.38	0.34	0.41
#24+27	0.10	0% (0)	na	na	na	na

[1] LOD = limit of detection

Table 6

Associations between serum organochlorine levels and demographic and lifestyle characteristics among 193 postmenopausal women - Western New York 1986-1991

	DDE[1]			PCB[1]			Mirex[2]			HCB		
	Crude	Adj.[3]	Spear.	Crude	Adj.[3]	Spear.	Crude	Adj.[3]	Spear.	Crude	Adj.[3]	Spear.
Age	.16*	na	.20**	.26**	na	.22**	-.07	na	-.06	.30**	na	.37**
Education	-.09	-.06	-.16*	-.03	-.06	-.03	.03	-.02	.02	-.06	.06	-.07
Quetelet Index[4]	.14*	.11	.20**	.02	-.03	.06	.01	-.01	.01	.10	.07	.16*
Weight (pounds)	.09	.09	.14*	-.03	-.03	-.01	.05	.01	.09	.06	.09	.12
Height (inches)	-.06	-.04	-.06	-.03	.01	-.07	.13	.10	.13	-.02	.02	.01
Physical Activity (hrs)	-.04	-.19*	.01	.04	-.30*	.06	.07	.04	-.03	.29**	.05	.17*
Walking (hrs)	.13	.12	.09	.17*	.15*	.14*	.12	.10	.12	.06	.05	.07
Fat[5]	-.06	-.05	-.15*	-.03	.02	-.10	-.05	-.11	-.01	-.15*	-.11	-.25**
Water[5]	-.03	-.01	-.01	-.01	.05	.01	-.04	-.07	-.01	-.12	-.05	-.13
Caffeine[5]	-.20**	-.18*	-.16*	-.20**	-.13	-.12	.02	.01	.10	-.16*	-.10	-.18*
Packyears[6]	-.03	-.03	-.04	-.04	-.02	-.05	.07	.06	.07	-.11	-.09	-.09
Total Alcohol[6]	-.09	-.07	-.05	.05	.11	.03	-.01	-.07	.02	-.13	-.08	-.08
Wine[6]	-.07	-.09	-.13	-.02	.04	-.07	.01	.03	.09	-.08	-.06	-.07
Beer[6]	-.01	.06	.08	.12	.14	.11	-.05	-.03	-.10	-.07	-.05	-.06
Liquor[6]	-.11	-.07	-.11	.01	.06	-.06	.01	.05	.03	-.08	-.07	-.01

[1] natural log
[2] dichotomized
[3] adjusted for age and serum lipids
[4] weight/height^2
[5] measures reflect intake of two years prior to interview
[6] measures reflect lifetime consumption
* $p<.05$
** $p<.01$

Table 6 (cont.)

	PCB Congener 180[1]			PCB Congener 153[1]			PCB Congener 138[1]		
	Crude	Adj.[2]	Spear.	Crude	Adj.[2]	Spear.	Crude	Adj.[2]	Spear.
Age	.26**	na	.24**	.17*	na	.16*	.10	na	.10
Education	-.09	.01	-.06	-.05	.01	-.04	-.09	-.06	-.04
Quetelet Index[4]	-.20**	-.28**	-.15*	-.01	-.06	.01	.06	.05	.06
Weight (pounds)	-.25**	-.28**	-.21**	-.06	-.08	-.03	.04	.05	.02
Height (inches)	-.10	-.06	-.13	-.08	-.04	-.09	-.01	.01	-.08
Physical Activity (hrs)	.02	-.09	-.04	-.07	-.28*	-.08	-.01	-.19	-.09
Walking (hrs)	.16*	.13	.09	.22**	.16*	.14*	.15*	.15*	.19**
Fat[5]	-.05	-.02	-.12	-.01	.02	-.07	-.04	-.03	-.10
Water[5]	-.02	.04	-.02	.03	.07	.04	-.01	.01	.03
Caffeine[5]	-.08	-.02	-.12	-.16*	-.12	-.10	-.12	-.09	-.08
Packyears[6]	-.01	.02	-.05	-.06	-.05	-.09	-.13	-.14*	-.15*
Total Alcohol[6]	.04	.10	.08	.03	.08	.06	-.09	-.07	.05
Wine[6]	-.01	.08	.03	.04	.09	.05	-.17*	-.20**	.03
Beer[6]	.01	.06	.08	.02	.07	.09	.06	.08	.07
Liquor[6]	.07	.12	.06	-.01	.03	-.01	-.01	.01	-.01

[1] natural log
[2] dichotomized
[3] adjusted for age and serum lipids
[4] weight/height^2
[5] measures reflect intake of two years prior to interview
[6] measures reflect lifetime consumption
* $p<.05$
** $p<.01$

Table 6 (cont.)

	PCB Congener 118[1]			PCB Congener 203+196[1]			PCB Congener 194[1]		
	Crude	Adj.[2]	Spear.	Crude	Adj.[2]	Spear.	Crude	Adj.[2]	Spear.
Age	.20**	na	.13	.27**	na	.32**	.22**	na	.22**
Education	-.06	-.01	-.02	-.06	.05	-.06	.01	.09	.02
Quetelet Index[4]	.20**	.19**	.19**	-.06	-.13	-.04	-.20**	.26**	-.19**
Weight (pounds)	.04	.21**	.16*	.18**	-.08	-.09	-.07	-.21**	-.19**
Height (inches)	-.01	.07	.01	.04	.06	-.08	.01	.02	-.03
Physical Activity (hrs)	-.06	-.26*	.02	-.01	-.09	-.02	.09	-.03	.01
Walking (hrs)	.22**	.20**	.22**	.16*	.15*	.14*	.10	.09	.12
Fat[5]	-.06	-.01	-.09	-.11	-.08	-.15*	-.02	.01	-.09
Water[5]	-.09	-.04	-.04	-.12	-.06	-.13	-.05	-.01	-.08
Caffeine[5]	-.24**	-.19**	-.09	-.16*	-.12	-.14*	-.05	.01	-.10
Packyears[6]	-.25**	-.25**	-.27**	-.11	-.10	-.12	.04	.05	.04
Total Alcohol[6]	.07	.11	.04	.01	.06	.01	.02	.06	-.03
Wine[6]	.03	.06	-.01	-.02	.03	-.05	-.03	.03	-.10
Beer[6]	.11	.15	.13	.04	.06	.03	.02	-.01	.04
Liquor[6]	-.01	.02	-.04	.01	.06	-.05	.07	.09	-.01

[1] natural log
[2] dichotomized
[3] adjusted for age and serum lipids
[4] weight/height^2
[5] measures reflect intake of two years prior to interview
[6] measures reflect lifetime consumption
* $p<.05$
** $p<.01$

Table 7

Associations between serum organochlorine levels and several food categories among 193 postmenopausal women - Western New York 1986-1991.

	DDE[1]			PCB[1]			Mirex[2]			HCB		
Dietary Intake grams/months[4]	Crude	Adj.[3]	Spear.	Crude	Adj.[3]	Spear.	Crude	Adj.[3]	Spear.	Crude	Adj.[3]	Spear.
Fruit	.18*	.13	.17*	.14	.06	.07	-.05	-.05	-.01	.17*	.11	.09
Vegetables	.13	.14	.10	.11	.13	.10	-.10	-.13	-.05	.13	.03	-.08
Grains	.02	.02	.06	.02	.02	.07	.02	.02	.07	.04	-.04	-.04
Dairy	.12	.13	.05	.06	.06	.08	-.05	-.05	-.01	.03	.08	.12
Fish	.04	.05	.01	.08	.11	.07	-.04	-.05	-.01	.04	.02	-.07
Poultry	-.03	-.01	-.09	.01	.06	-.01	-.01	-.04	.04	.04	-.01	-.11
Beef	-.02	-.01	-.06	-.03	.01	.01	.02	-.02	.04	-.01	-.11	-.14*
Pork	.01	.01	-.06	-.08	-.05	-.08	-.02	-.05	-.04	-.12	-.12	.12
Bacon	.12	.14	.11	-.06	-.03	-.03	.02	-.01	-.01	-.08	-.11	-.10
Red Meat	-.01	-.01	-.06	-.05	-.01	-.03	.02	-.03	-.02	-.05	-.12	-.15*
Processed Meat	.09	.10	-.06	-.05	-.03	-.01	-.03	-.05	.01	-.07	-.09	-.15*

[1] natural log
[2] dichotomized
[3] adjusted for age and serum lipids
[4] measures reflect intake of two years prior to interview
* $p<.05$
** $p<.01$

Table 7 (cont.)

	PCB congener 180[1]			PCB congener 153[1]			PCB congener 138[1]		
Dietary Intake grams/months[4]	Crude	Adj.[2]	Spear.	Crude	Adj.[2]	Spear.	Crude	Adj.[2]	Spear.
Fruit	.11	.04	.07	.20**	.15*	.13	.16*	.13	.13
Vegetables	.13	.16*	.17*	.15*	.16*	.15*	.16*	.16*	.17
Grains	.04	.05	.06	-.01	-.01	.02	.03	-.04	.01
Dairy	.03	.02	.05	.09	.08	.08	-.07	-.08	.05
Fish	.04	.07	..04	.14*	.15*	.13	.08	.09	.10
Poultry	.04	.08	.03	.03	.06	.01	.01	.03	-.01
Beef	-.01	.02	.02	.04	.07	.07	.02	.03	.04
Pork	-.12	-.10	-.10	-.03	-.02	-.06	-.01	-.01	-.09
Bacon	-.08	-.04	-.06	.01	.03	.01	-.06	-.05	-.02
Red Meat	-.05	-.02	-.03	.02	.05	.03	.01	.03	.01
Processed Meat	-.07	-.05	-.04	.01	.01	.04	.01	.02	.04

[1] natural log
[2] dichotomized
[3] adjusted for age and serum lipids
[4] measures reflect intake of two years prior to interview
* p<.05
** p<.01

Table 7 (cont.)

	PCB congener 118[1]			PCB congeners 203+196[1]			PCB congener 194[1]		
Dietary Intake grams/months[4]	Crude	Adj.[2]	Spear.	Crude	Adj.[2]	Spear.	Crude	Adj.[2]	Spear.
Fruit	.12	.07	.08	.09	.02	.09	.05	-.02	.02
Vegetables	.01	.04	.01	.13	.16*	.14*	.03	.05	.07
Grains	-.09	-.09	.05	.05	.08	.07	.09	.10	.08
Dairy	-.01	-.01	.01	-.05	-.05	-.01	-.05	-.06	-.01
Fish	.07	.08	.05	.02	.06	.01	.01	.03	.01
Poultry	-.01	.03	-.06	.03	.07	.01	-.01	.01	-.03
Beef	-.01	.05	-.01	.03	.09	.04	-.10	-.09	-.08
Pork	-.01	.02	-.04	-.02	-.01	-.01	-.06	-.05	-.12
Bacon	.01	.03	.02	-.07	-.06	-.08	-.15*	-.13	-.09
Red Meat	-.01	.05	-.02	.02	.05	.01	-.11	-.09	-.11
Processed Meat	-.02	.01	.05	-.05	-.04	-.05	-.06	-.05	-.03

[1] natural log
[2] dichotomized
[3] adjusted for age and serum lipids
[4] measures reflect intake of two years prior to interview
* $p<.05$
** $p<.01$

Table 8

Associations between serum organochlorine levels and reproductive characteristics among 193 postmenopausal women - Western New York 1986-1991.

	DDE[1]			PCB[1]			Mirex[2]			HCB		
	Crude	Adj.[3]	Spear.	Crude	Adj.[3]	Spear.	Crude	Adj.[3]	Spear.	Crude	Adj.[3]	Spear.
Month of Lactation	-.05	-.08	.01	-.01	-.07	.06	-.01	.03	-.03	.07	-.01	.15*
# Live Births	-.17*	-.16*	-.16*	-.15*	-.12	-.12	-.01	.01	-.02	-.02	.01	-.10
Weeks Pregnant	-.16*	-.15*	-.16*	-.08	-.11	-.12	-.02	.01	-.03	-.03	.01	-.10
Months Menstruated	.09	.05	.10	.17*	.12	.13	-.16*	.06	-.15*	.13	.06	.19**
Breastfed	.12	.10	.09	.05	.04	.04	-.02	-.01	-.01	.06	.04	.05
Age at Menarche	.05	.04	.06	-.02	-.01	.02	.12	.13	.14*	-.11	-.14*	-.06
Age at Menopause	.05	.01	.04	.11	.06	.07	-.13	-.11	-.15*	.08	.02	.15*

[1] natural log
[2] dichotomized
[3] adjusted for age and serum lipids
* $p<.05$
** $p<.01$

Table 8 (cont.)

	PCB Congener 180[1]			PCB Congener 153[1]			PCB Congener 138[1]		
	Crude	Adj.[2]	Spear.	Crude	Adj.[2]	Spear.	Crude	Adj.[2]	Spear.
Month of Lactation	.02	-.04	.10	-.01	-.05	.05	.03	.01	.08
# Live Births	-.11	-.09	-.12	-.12	-.10	-.09	-.08	-.08	-.05
Weeks Pregnant	-.12	-.09	-.13	-.11	-.10	-.09	-.07	-.07	-.04
Months Menstruated	.11	.07	.13	.13	.10	.12	.10	.08	.09
Breastfed	.08	.07	.05	.05	.04	.03	.03	.02	.03
Age at Menarche	.04	.04	.03	.05	.06	.07	.06	.07	.06
Age at Menopause	.09	.05	.08	.10	.07	.08	.09	.07	.08

[1] natural log
[2] adjusted for age and serum lipids
* p<.05
** p<.01

Table 8 (cont.)

	PCB Congener 118[1]			PCB Congener 203+196[1]			PCB Congener 194[1]		
	Crude	Adj.[2]	Spear.	Crude	Adj.[2]	Spear.	Crude	Adj.[2]	Spear.
Month of Lactation	-.03	-.08	-.04	.01	-.05	.02	-.04	-.08	-.01
# Live Births	-.08	-.06	-.09	-.09	-.05	-.16*	-.06	-.02	-.09
Weeks Pregnant	-.08	-.06	-.09	.09	-.05	-.17*	-.06	-.02	-.10
Months Menstruated	.16*	.12	.14	.11	.07	.10	.10	.06	.13
Breastfed	-.01	-.04	-.01	.14*	.13	.11	.06	.04	.07
Age at Menrache	.01	.01	.01	.01	.01	.02	.08	.08	.04
Age at Menopause	.11	.07	.08	.09	.06	.04	.11	.08	.08

[1] natural log
[2] adjusted for age and serum lipids
* p<.05
** p<.01

Table 9

Crude and adjusted means of serum organochorine levels among postmenopausal women with and without a history of lactation

	History of Lactation							
	Nonlactated (n=166)				Lactated (n=26)			
	crude mean	adj. mean[1]	adj. mean[2]	adj. mean[3]	crude mean	adj. mean[1]	adj. mean[2]	adj. mean[3]
DDE	11.27	11.10	11.59	11.53	10.51	10.68	10.05	10.24
total PCBs	3.87	3.82	3.96	3.92	4.29	4.34	4.16	4.24
HCB	0.40	0.40	0.41	0.40	0.44	0.44	0.43	0.43
mirex	0.04	0.04	0.04	0.04	0.04	0.04	0.04	0.04
PCB180	0.46	0.46	0.47	0.47	0.51	0.52	0.50	0.51
PCB153	0.79	0.78	0.80	0.79	0.89	0.90	0.87	0.89
PCB138	0.61	0.60	0.62	0.61	0.70	0.71	0.69	0.70
PCB118	0.41	0.40	0.42	0.42	0.44	0.44	0.42	0.43
PCB203+196	0.10	0.10	0.10	0.10	0.11	0.11	0.10	0.11
PCB194	0.13	0.13	0.13	0.13	0.13	0.13	0.12	0.13

[1] adjusted for serum lipids
[2] adjusted for age
[3] adjusted for age and serum lipids

Table 10

Crude and adjusted means of serum organochorine levels among postmenopausal by parity

	Number of Livebirths															
	None (n=25)				one (n=14)				two (n=43)				three (n=41)			
	crude mean	adj. mean[1]	adj. mean[2]	adj. mean[3]	crude mean	adj. mean[1]	adj. mean[2]	adj. mean[3]	crude mean	adj. mean[1]	adj. mean[2]	adj. mean[3]	crude mean	adj. mean[1]	adj. mean[2]	adj. mean[3]
DDE	12.40	12.35	12.63	12.62	14.15	14.12	12.81	13.10	12.89	12.47	12.48	12.34	11.98	11.69	12.42	12.13
total PCBs	3.54	3.57	3.67	3.64	5.71	5.69	5.41	5.46	4.57	4.39	4.46	4.36	4.25	4.19	4.32	4.29
HCB	0.40	0.40	0.42	0.41	0.51	0.51	0.47	0.47	0.42	0.41	0.41	0.41	0.44	0.43	0.45	0.45
mirex	0.01	0.01	0.01	0.01	0.05	0.05	0.05	0.05	0.04	0.04	0.04	0.04	0.02	0.02	0.02	0.02
PCB180	0.45	0.46	0.46	0.46	0.66	0.66	0.62	0.63	0.52	0.50	0.51	0.49	0.52	0.51	0.52	0.52
PCB153	0.75	0.75	0.75	0.76	1.26	1.26	1.22	1.22	0.09	0.86	0.89	0.86	0.89	0.87	0.90	0.89
PCB138	0.61	0.62	0.61	0.62	1.01	1.01	0.98	0.98	0.68	0.65	0.67	0.65	0.70	0.69	0.71	0.70
PCB118	0.39	0.40	0.41	0.40	0.60	0.60	0.56	0.57	0.45	0.43	0.44	0.43	0.43	0.42	0.45	0.43
PCB203+196	0.10	0.10	0.11	0.10	0.14	0.14	0.12	0.13	0.11	0.11	0.11	0.10	0.11	0.11	0.11	0.11
PCB194	0.12	0.12	0.12	0.12	0.14	0.14	0.14	0.14	0.16	0.16	0.16	0.16	0.11	0.11	0.11	0.11

[1] adjusted for serum lipids
[2] adjusted for age
[3] adjusted for age and serum lipids

Table 10 (cont.)

Number of Livebirths (cont.)								
	four (n=35)				five+ (n=34)			
	crude mean	adj. mean[1]	adj. mean[2]	adj. mean[3]	crude mean	adj. mean[1]	adj. mean[2]	adj. mean[3]
DDE	7.02	8.57	8.05	8.58	7.52	7.66	8.12	8.08
total PCBs	3.89	4.08	3.92	4.08	3.31	3.35	3.40	3.44
HCB	0.41	0.42	0.41	0.42	0.38	0.39	0.40	0.40
mirex	0.05	0.05	0.05	0.05	0.04	0.04	0.04	0.04
PCB180	0.46	0.49	0.46	0.49	0.42	0.43	0.43	0.44
PCB153	0.75	0.79	0.75	0.79	0.73	0.74	0.75	0.76
PCB138	0.56	0.59	0.56	0.59	0.59	0.59	0.60	0.60
PCB118	0.40	0.42	0.40	0.42	0.36	0.36	0.37	0.37
PCB203+196	0.10	0.10	0.10	0.10	0.09	0.09	0.09	0.09
PCB194	0.11	0.12	0.11	0.12	0.13	0.13	0.13	0.13

[1] adjusted for serum lipids
[2] adjusted for age
[3] adjusted for age and serum lipids

Table 11

Crude and adjusted means of serum organochorine levels among postmenopausal women who have or have not been breastfed as an infant

Breastfed as Infant								
	No (n=15)				Yes (n=124)			
	crude mean	adj. mean[1]	adj. mean[2]	adj. mean[3]	crude mean	adj. mean[1]	adj. mean[2]	adj. mean[3]
DDE	5.91	6.00	6.14	6.17	10.55	10.46	10.25	10.29
total PCBs	3.97	3.95	4.07	4.05	3.98	4.00	3.87	3.91
HCB	0.41	0.41	0.42	0.42	0.41	0.41	0.40	0.40
mirex	0.06	0.06	0.06	0.05	0.03	0.04	0.03	0.04
PCB180	0.43	0.43	0.44	0.44	0.48	0.48	0.46	0.47
PCB153	0.77	0.76	0.78	0.78	0.81	0.82	0.80	0.80
PCB138	0.61	0.61	0.62	0.62	0.63	0.64	0.62	0.62
PCB118	0.40	0.41	0.41	0.41	0.43	0.43	0.42	0.42
PCB203+196	0.08	0.08	0.09	0.09	0.10	0.10	0.10	0.10
PCB194	0.17	0.16	0.17	0.17	0.12	0.12	0.12	0.17

[1] adjusted for serum lipids
[2] adjusted for age
[3] adjusted for age and serum lipids

Table 12

Crude and adjusted means of serum organochorine levels among postmenopausal women with and without prior use of birth control pills

Birth Control Pill Use								
	No (n=166)				Yes (n=26)			
	crude mean	adj. mean[1]	adj. mean[2]	adj. mean[3]	crude mean	adj. mean[1]	adj. mean[2]	adj. mean[3]
DDE	11.18	11.03	10.01	10.19	8.64	8.79	9.59	9.63
total PCBs	4.25	4.24	3.97	4.05	3.21	3.22	3.42	3.41
HCB	0.43	0.43	0.40	0.41	0.34	0.34	0.36	0.36
mirex	0.03	0.03	0.03	0.03	0.06	0.05	0.05	0.05
PCB180	0.50	0.50	0.47	0.48	0.42	0.42	0.45	0.45
PCB153	0.86	0.86	0.81	0.82	0.75	0.75	0.79	0.78
PCB138	0.68	0.68	0.64	0.65	0.56	0.56	0.59	0.58
PCB118	0.43	0.43	0.40	0.41	0.36	0.37	0.39	0.40
PCB203+196	0.11	0.11	0.10	0.10	0.08	0.08	0.09	0.09
PCB194	0.14	0.14	0.13	0.13	0.10	0.10	0.10	0.10

[1] adjusted for serum lipids
[2] adjusted for age
[3] adjusted for age and serum lipids

Table 13

Crude and adjusted means of serum organochorine levels among postmenopausal women with and without prior use of hormone replacement therapy

Hormone Replacement Therapy Use								
	No (n=142)				Yes (n=50)			
	crude mean	adj. mean[1]	adj. mean[2]	adj. mean[3]	crude mean	adj. mean[1]	adj. mean[2]	adj. mean[3]
DDE	10.67	10.75	10.25	10.39	11.33	11.25	11.60	11.61
total PCBs	4.20	4.24	4.08	4.15	3.81	3.79	3.91	3.88
HCB	0.43	0.43	0.42	0.42	0.39	0.39	0.40	0.40
mirex	0.04	0.04	0.04	0.04	0.03	0.03	0.03	0.03
PCB180	0.50	0.50	0.48	0.49	0.48	0.47	0.49	0.48
PCB153	0.86	0.87	0.84	0.85	0.81	0.81	0.83	0.82
PCB138	0.66	0.67	0.64	0.65	0.66	0.66	0.67	0.67
PCB118	0.44	0.45	0.43	0.44	0.37	0.37	0.39	0.38
PCB203+196	0.10	0.10	0.10	0.10	0.11	0.11	0.11	0.11
PCB194	0.13	0.13	0.13	0.13	0.12	0.12	0.13	0.13

[1] adjusted for serum lipids
[2] adjusted for age
[3] adjusted for age and serum lipids

Table 14

Crude and adjusted means of serum organochorine levels among postmenopausal women with and without previous benign breast disease

	Previous Benign Breast Disease							
	No (n=154)				Yes (n=38)			
	crude mean	adj. mean[1]	adj. mean[2]	adj. mean[3]	crude mean	adj. mean[1]	adj. mean[2]	adj. mean[3]
DDE	10.95	10.67	10.68	10.49	10.10	10.37	10.20	10.55
total PCBs	4.15	4.15	4.07	4.10	3.95	3.95	3.96	4.00
HCB	0.43	0.43	0.42	0.42	0.38	0.38	0.38	0.38
mirex	0.04	0.04	0.04	0.04	0.03	0.02	0.03	0.02
PCB180	0.49	0.49	0.48	0.48	0.50	0.50	0.50	0.50
PCB153	0.85	0.85	0.83	0.84	0.85	0.85	0.85	0.86
PCB138	0.66	0.66	0.65	0.65	0.67	0.67	0.67	0.68
PCB118	0.42	0.42	0.42	0.41	0.43	0.43	0.43	0.44
PCB203+196	0.11	0.10	0.10	0.10	0.10	0.10	0.10	0.10
PCB194	0.13	0.13	0.13	0.13	0.14	0.14	0.14	0.14

[1] adjusted for serum lipids
[2] adjusted for age
[3] adjusted for age and serum lipids

Table 15

Crude and adjusted means of serum organochorine levels among postmenopausal women with and without a family history of breast cancer

Family History of Breast Cancer								
	No (n=174)				Yes (n=18)			
	crude mean	adj. mean[1]	adj. mean[2]	adj. mean[3]	crude mean	adj. mean[1]	adj. mean[2]	adj. mean[3]
DDE	10.79	10.61	10.38	10.40	11.31	11.47	11.27	11.70
total PCBs	4.11	4.09	4.00	4.02	4.05	4.08	4.03	4.14
HCB	0.42	0.42	0.41	0.41	0.39	0.40	0.40	0.40
mirex	0.03	0.03	0.03	0.03	0.07	0.07	0.07	0.07
PCB180	0.50	0.50	0.48	0.48	0.46	0.47	0.47	0.48
PCB153	0.84	0.84	0.82	0.83	0.90	0.91	0.89	0.92
PCB138	0.64	0.64	0.63	0.63	0.81	0.81	0.80	0.82
PCB118	0.41	0.41	0.40	0.40	0.52	0.53	0.51	0.53
PCB203+196	0.11	0.11	0.10	0.10	0.08	0.08	0.08	0.08
PCB194	0.13	0.13	0.13	0.13	0.12	0.12	0.12	0.12

[1] adjusted for serum lipids
[2] adjusted for age
[3] adjusted for age and serum lipids

Table 16

Crude and adjusted means of serum organochorine levels among postmenopausal by smoking status

	Smoking Status											
	Never (n=98)				Former (n=49)				Current (n=45)			
	crude mean	adj. mean[1]	adj. mean[2]	adj. mean[3]	crude mean	adj. mean[1]	adj. mean[2]	adj. mean[3]	crude mean	adj. mean[1]	adj. mean[2]	adj. mean[3]
DDE	11.03	10.40	10.74	10.21	10.43	10.81	10.34	10.77	10.83	11.08	11.06	11.31
total PCBs	4.16	4.09	4.09	4.04	4.25	4.30	4.22	4.29	3.83	3.84	3.85	3.90
HCB	0.43	0.42	0.42	0.42	0.42	0.42	0.42	0.42	0.40	0.40	0.40	0.41
mirex	0.03	0.03	0.03	0.03	0.05	0.05	0.05	0.05	0.05	0.04	0.04	0.04
PCB180	0.49	0.48	0.48	0.47	0.49	0.50	0.48	0.50	0.50	0.50	0.50	0.51
PCB153	0.87	0.85	0.85	0.85	0.86	0.88	0.86	0.87	0.78	0.78	0.78	0.79
PCB138	0.69	0.68	0.68	0.68	0.68	0.68	0.67	0.68	0.57	0.57	0.58	0.58
PCB118	0.50	0.49	0.50	0.49	0.37	0.38	0.37	0.37	0.30	0.30	0.31	0.31
PCB203+196	0.11	0.10	0.11	0.10	0.10	0.10	0.10	0.10	0.10	0.10	0.10	0.10
PCB194	0.13	0.13	0.12	0.12	0.13	0.13	0.13	0.13	0.14	0.14	0.14	0.14

[1] adjusted for serum lipids
[2] adjusted for age
[3] adjusted for age and serum lipids

Table 17

Crude and adjusted means of serum organochorine levels among postmenopausal by occupation

	Occupation															
	professional (n=33)				sales,administration (n=83)				service occupation (n=29)				labor (n=47)			
	crude mean	adj. mean[1]	adj. mean[2]	adj. mean[3]	crude mean	adj. mean[1]	adj. mean[2]	adj. mean[3]	crude mean	adj. mean[1]	adj. mean[2]	adj. mean[3]	crude mean	adj. mean[1]	adj. mean[2]	adj. mean[3]
DDE	10.80	10.84	11.60	11.67	9.66	9.73	10.13	10.09	11.02	11.55	10.77	11.17	12.90	12.37	12.38	12.12
total PCBs	4.25	4.28	4.46	4.51	3.88	3.90	4.01	4.00	4.16	4.30	4.04	4.20	4.39	4.24	4.23	4.17
HCB	0.49	0.45	0.48	0.48	0.40	0.40	0.41	0.41	0.39	0.40	0.38	0.39	0.45	0.45	0.44	0.44
mirex	0.03	0.03	0.03	0.03	0.04	0.04	0.03	0.03	0.05	0.05	0.04	0.05	0.03	0.03	0.03	0.03
PCB180	0.50	0.50	0.52	0.53	0.49	0.49	0.50	0.50	0.50	0.52	0.49	0.51	0.50	0.50	0.48	0.47
PCB153	0.92	0.93	0.95	0.97	0.80	0.81	0.83	0.83	0.88	0.91	0.85	0.89	0.92	0.93	0.94	0.81
PCB138	0.73	0.74	0.74	0.77	0.63	0.63	0.65	0.64	0.66	0.68	0.65	0.67	0.73	0.73	0.65	0.63
PCB118	0.42	0.42	0.45	0.45	0.41	0.41	0.42	0.42	0.42	0.43	0.41	0.42	0.42	0.42	0.45	0.44
PCB203+196	0.10	0.10	0.11	0.11	0.10	0.10	0.10	0.10	0.11	0.12	0.11	0.12	0.10	0.10	0.10	0.10
PCB194	0.15	0.15	0.15	0.15	0.12	0.12	0.13	0.13	0.14	0.14	0.13	0.14	0.15	0.15	0.12	0.12

[1] adjusted for serum lipids
[2] adjusted for age
[3] adjusted for age and serum lipids

Table 18

Crude and adjusted means of serum organochorine levels among postmenopausal by place of residence

	Place of Residence											
	Urban (n=60)				Suburban (n=102)				Rural (n=30)			
	crude mean	adj. mean[1]	adj. mean[2]	adj. mean[3]	crude mean	adj. mean[1]	adj. mean[2]	adj. mean[3]	crude mean	adj. mean[1]	adj. mean[2]	adj. mean[3]
DDE	12.76	12.72	12.45	12.51	9.41	9.34	9.52	9.39	11.76	11.87	11.50	12.02
total PCBs	4.29	4.24	4.21	4.18	3.94	3.95	3.94	3.96	4.34	4.38	4.26	4.43
HCB	0.40	0.40	0.40	0.40	0.42	0.42	0.42	0.42	0.46	0.46	0.46	0.47
mirex	0.05	0.05	0.05	0.05	0.02	0.02	0.02	0.02	0.04	0.04	0.04	0.04
PCB180	0.46	0.46	0.46	0.45	0.51	0.52	0.51	0.52	0.47	0.48	0.47	0.48
PCB153	0.86	0.85	0.84	0.84	0.83	0.83	0.83	0.83	0.89	0.90	0.88	0.91
PCB138	0.65	0.64	0.64	0.63	0.64	0.65	0.64	0.65	0.74	0.75	0.73	0.75
PCB118	0.46	0.46	0.45	0.45	0.38	0.38	0.39	0.39	0.48	0.49	0.47	0.49
PCB203+196	0.11	0.10	0.10	0.10	0.10	0.10	0.10	0.10	0.11	0.11	0.11	0.11
PCB194	0.13	0.13	0.13	0.13	0.13	0.13	0.13	0.13	0.13	0.14	0.13	0.14

[1] adjusted for serum lipids
[2] adjusted for age
[3] adjusted for age and serum lipids

Table 19

Multiple Linear Regression Model for DDE				
Variable	Coefficient	SE	Partial r^2	p value
Intercept	1.005	0.333		
# Livebirths	-0.085	0.037	0.04	.02
Caffeine Intake (mg)	-2.741	9.134	0.04	.002
Suburban Residence (yes=1)	-0.387	0.155	0.03	.01
Breastfed as Infant (yes=1)	0.557	0.252	0.03	.03
Fruit Intake (g)	3.154	1.499	0.02	.04
R^2 =0.16				
Total PCBs (mg/g)	0.185	0.039	0.12	.0001
R^2 = 0.28				

Table 20

Multiple Linear Regression Model for Total PCBs				
Variable	Coefficient	SE	Partial r^2	p value
Intercept	0.323	0.244		
Age	0.013	0.004	0.06	.003
Cherry Intake (g)	2.471	1.009	0.03	.02
Serum Triglycerides	0.001	4.355	0.04	.006
Caffeine Intake (mg)	-8.582	4.403	0.02	.05
Cucumber Intake (g)	4.262	2.510	0.01	.08
Asparagus Intake (g)	3.017	1.362	0.02	.03
R^2 =0.18				
DDE (mg/g)	0.013	0.003	0.14	.0001
HCB (mg/g)	0.908	0.168	0.10	.0001
R^2 = 0.42				

Table 21

Multiple Linear Regression Model for HCB				
Variable	Coefficient	SE	Partial r^2	p value
Intercept				
Age	0.007	0.002	0.09	.0001
Prunes/Plums Intake (g)	1.400	5.109	0.05	.008
Asparagus Intake (g)	1.472	4.768	0.03	.002
R^2 =0.17				
Total PCBs (mg/g)	0.046	0.005	0.26	.0001
R^2 = 0.43				

Table 22

Multiple Linear Regression Model for PCB Congener 180				
Variable	Coefficient	SE	Partial r^2	p value
Intercept	-1.250	0.233		
Lettuce Intake (g)	1.186	3.703	0.08	.002
Age	0.009	0.004	0.08	.01
Weight (pounds)	-0.003	7.857	0.04	.0001
Serum Triglycerides	9.050	3.562	.04	.01
Prunes/Plums Intake (g)	2.019	1.060	0.02	.06
One Child (yes=1)	0.209	0.090	0.02	.04
R^2 =0.28				
DDE (ng/g)	0.001	0.003	0.07	.0001
HCB (ng/g)	0.948	0.139	0.13	.0001
R^2 = 0.48				

Table 23

Multiple Linear Regression Model for PCB Congener 153				
Variable	Coefficient	SE	Partial r^2	p value
Intercept	-0.970	0.252		
Serum Tryglycerides	0.001	4.459	0.05	.008
Asparagus Intake (g)	2.709	1.394	0.04	.05
Lettuce Intake (g)	1.184	4.741	0.02	.01
Age	0.007	0.005	0.02	.10
Cherry Intake	1.776	1.019	0.01	.08
One Child (yes=1)	0.308	0.130	0.03	.02
R^2 =0.17				
DDE (ng/g)	0.021	0.003	0.20	.0001
HCB (ng/g)	0.617	0.170	0.04	.0004
R^2 = 0.41				

Table 24

Multiple Linear Regression Model for PCB Congener 138				
Variable	Coefficient	SE	Partial r^2	p value
Intercept	-1.188	0.402		
Lettuce Intake (g)	1.422	7.324	0.05	.05
Asparagus Intake (g)	4.409	2.118	0.02	.04
Wine Intake (g)	-5.728	2.518	0.02	.02
Prunes/Plums Intake (g)	4.577	2.072	0.01	.03
Family History of Breast Cancer (yes=1)	0.324	0.178	0.02	0.06
R^2 =0.12				
DDE (ng/g)	0.016	0.005	0.05	.0007
HCB (ng/g)	1.554	0.275	0.12	.0001
R^2 = 0.29				

Table 25

Multiple Linear Regression Model for PCB Congener 118				
Variable	Coefficient	SE	Partial r^2	p value
Intercept	-3.773	0.549		
Age	0.007	0.007	0.04	.08
Never Smoker (yes=1)	0.256	0.94	0.05	.007
Walking (hrs/wk)	0.349	0.097	0.04	.0004
Quetelet Index	0.027	0.009	0.04	.003
Asparagus Intake (g)	5.878	1.928	0.03	.003
Non-Bran Dry Cereal Intake (g)	5.501	1.817	0.02	.003
Suburban Residence (yes=1)	-0.235	0.093	0.02	.01
Serum Cholesterol	0.003	0.001	0.02	.009
R^2 =0.26				
DDE (ng/g)	0.226	0.254	0.05	.0005
R^2 = 0.31				

Table 26

Multiple Linear Regression Model for PCB Congeners 203+196				
Variable	Coefficient	SE	Partial r^2	p value
Intercept	-5.117	0.532		
Age	0.022	0.007	0.07	.0008
Lettuce Intake (g)	1.719	7.129	0.04	.05
Serum Triglycerides	0.001	6.926	0.03	.04
Punes/Plums Intake (g)	4.125	2.063	0.02	.05
Serum Cholesterol	0.003	0.001	0.02	.03
Walking (hrs/wk)	0.224	0.108	0.02	.04
R^2 =0.20				
DDE (ng/g)	0.011	0.005	0.03	.04
HCB (ng/g)	0.800	0.291	0.02	.007
R^2 = 0.25				

Table 27

Multiple Linear Regression Model for PCB Congener 194				
Variable	Coefficient	SE	Partial r^2	p value
Intercept	-2.678	0.413		
Age	0.016	0.006	0.04	.008
Quetelet Index	-0.031	0.009	0.04	.0004
Triglycerides	0.002	6.258	0.04	.004
Prunes/Plums Intake (g)	3.612	1.860	0.01	.05
R^2 =0.13				
HCB (ng/g)	1.109	0.263	0.07	.0001
R^2 = 0.20				

DISCUSSION

The sample in the present study is unique in the investigation of frequency of occurrence and magnitude of occurrence of serum organochlorine levels. As indicated above, the majority of previous studies on this subject matter focused on special exposure populations and rarely included women. This study, however, examined organochlorine body burden in healthy, postmenopausal women from the general population in Erie and Niagara counties in western New York State, which hampers comparability with results from other investigation, due to differences in the study groups. Therefore, results on magnitude of occurrence of these compounds will only be discussed in relation to those studies which study populations most closely resemble that of the present study.

The mean level of serum DDE levels in this study was 10.76 ng/g, which lies well within the range of values of levels reported in previous investigations. Krauthacker (1991) reported median DDE levels of 6 ng/g among young women from the general population in the former Yugoslavia, and young women from Norway had DDE levels of 19 ng/g (Skaare, 1988). Two case control studies on organochlorines and breast cancer provided measurements of these compounds in the controls. In a study conducted in New York City, Wolff (1993) reported mean levels of 7.7 ng/g in the control group of healthy women. Krieger (1994) found DDE levels of 43.1 ng/g among healthy Californian control subjects. Thus, DDE levels detected in this study lie somewhere between those levels detected in previous investigations.

With regard to total PCBs, mean levels of 4.06 ng/g were detected in this sample. In a study conducted in the former Yugoslavia median levels of 3 ng/g were found among male and female members of the general population (Krauthacker, 1993). Among Norwegian young women mean levels of 10 ng/g were observed. . Wolff (1993) reported a total PCB mean of 6.7 ng/g for her control group, and the control group in the Californian study demonstrated a mean of 4.8 ng/g for total PCBs. Again, PCB levels among the sample in the present study are well within the range of the levels reported previously.

Comparable results for HCB levels was only provided by Krauthacker's study (1991). Among these young women, she found median HCB levels of 2 ng/g, which was higher than the mean HCB of 9.43 ng/g observed in the present study. No comparable

results were found for mirex or any of the individual PCB congeners. In interpreting these comparisons, it should be kept in mind, that, although the study populations these selected studies are the most comparable with the sample in this study, several differences remain. Specifically, this sample consists of postmenopausal women, and the average age is well above that of the other studies. In addition, most of the study to which these results were compared to were conducted in different geographic areas, where the environmental exposure to organochlorines may differ.

In this research it was attempted to identify correlates of organochlorine levels to a variety of exposure variables, including lifestyle, dietary, reproductive and occupational variables. Briefly, serum DDE was positively associated with age, body mass index, dietary fruit intake, having been breastfed as an infant, a positive family history of breast cancer, no prior history of birth control pill use, and labor occupations. Inverse associations for this compound were observed for strenuous physical activity, caffeine intake, parity, and suburban residence. Serum levels of total PCBs were positively related to age, serum triglycerides, walking for recreation and transportation, fruit intake in general and cherry intake in particular, intake of cucumbers and asparagus, no prior history of birth control pill use, and professional occupation; and negatively related to caffeine intake, physical activity, and suburban residence. HCB was found to be positively associated with age, fruit intake, in particular intake of prunes or plums, asparagus intake, as well as with no prior history of birth control bill use, professional occupation, and rural residence. An inverse association was found for HCB and caffeine intake. No significant correlates of serum mirex levels were identified in this study population. Less than one quarter of the study population had detectable levels of this compound and those levels were generally low. Environmental exposure to mirex is restricted to the southern United States, where it is used in the agricultural control of fire ants. Exposure to this substance in the western New York area could occur however through the consumption of Great Lake fish. Mirex was not associated with fish consumption in general, and no data on Great Lake fish consumption was available for this analysis.

Results from the congener specific analysis revealed positive associations between congener 180 and age, triglycerides, vegetable intake in general and intake of lettuce in

particular, intake of prunes or plums, walking, having one child only, and suburban residence, whereas negative associations were found for body mass index and weight. Congener 153 was found to be positively associated with age, serum triglycerides, physical activity, walking, total fruit, cherry, total vegetable, asparagus, lettuce and fish intake, as well as with a positive family history of breast cancer. An inverse association was observed for total caffeine intake. Results from the analysis of congener 138 revealed positive associations with age, walking, positive family history of breast cancer, rural residence, and consumption of fruits and vegetables in general and intake of lettuce, asparagus, and prunes or plums in particular, as well as a negative association with wine consumption. Congener 118 was positively related to age, serum cholesterol, quetelet index, weight, walking, never smoker status, asparagus intake, and a positive family history of breast cancer. Negative associations were observed for this congener and suburban residence, strenuous physical activity, caffeine intake, and packyears of cigarette smoking. Positive associations were found for congeners 203+196 and age, serum triglycerides, serum cholesterol, walking, and intake of prunes or plums and lettuce. An inverse association was observed for congeners 203+196 and caffeine intake. Finally, congener 194 was positively related to age, serum triglycerides, and intake of prunes or plums, and inversely related to quetelet index.

In general, age is a consistent correlate of serum organochlorines in this study population. Many of these compounds are positively associated with fruits and vegetables, among which specific produce such as lettuce, asparagus, cucumbers, cherries, and prunes or plums appear to be most frequently associated with total PCBs and several PCB congeners. Walking was positively associated with many of these compounds, whereas more strenuous physical activity was negatively associated with some of these compounds. A relatively consistent finding is that of an inverse association with caffeine intake and some of these organochlorines.

Again, it is difficult to relate the present research to previously conducted studies that attempted to identify predictors of organochlorines, due to the vast differences in study populations and exposure acertainment. Several studies found that increased fish consumption was related to higher serum levels of DDE, HCB, total PCBs, and individual PCB congeners (Lommel et al., 1992; Kreiss, 1985; Evans et al., 1994; Apslund et al.,

1994, Fiore et al, 1989; Hovinga et al, 1993). In these data we found no association between fish intake and serum organochlorine levels. However, studies that did find such an association, investigated organochlorines levels among sport fishermen, professional fishermen, or individuals who rely on consumption of contaminated fish for their diet. As for the fish consumption variables in this research, it is not possible to determine whether the fish consumed by the participants was caught in contaminated areas. In the interview all participants were asked to report usual consumption of fresh, frozen, or canned fish, shrimp, and other shellfish.

Previous studies that directly examined organochlorines in foods, found highest levels in fatty foods (Frank et al., 1993; Kannon et al., 1992; Kashyap et al., 1994, MacIntosh et al., 1996). In these data, increased intake of fatty foods, or total dietary fat intake was not associated with increased organochlorine levels. In contrast, dietary intake of fruits and vegetables were correlated with serum levels of these compounds. This difference may be due to the fact that in two of the studies were conducted in developing countries (Kannon et al., 1992; Kashyap et al., 1994), where patterns of exposure are likely to be different. Also, measures in food stuff reflects current levels of organochlorines, whereas serum levels of postmenopausal women reflect lifetime exposure. As discussed previously, usage of these substances has changed dramatically in the past decades, thus the positive association with fruits and vegetables in this study population may be the result of consumption of organochlorine treated produce in the past. Elevated levels in fatty foods to date may reflect the movement from directly affected foods (e.g., produce through direct exposure or contaminated soil) to indirectly affected foods (e.g., contaminated dairy and meats through exposed farm animals). Furthermore, in the only study to date that related serum organochlorine levels to self reported dietary no association between dairy products and meats was observed (Lommel et al., 1992).

There are a number of studies that examined organochlorine levels with regard to occupation (e.g., Krauthacker, 1993; Kannan et al., 1994; Luotamo et al., 1991), however, in these studies the sample consisted of occupationally exposed workers (e.g., capacitor workers) and comparison of results from these studies to those of the present study is not appropriate. Lommel et al. (1992) found no differences in serum organochlorine levels with

regard to occupation among sport fishermen, and in these data only a weak effect with labor occupation and increased DDE levels was found. This is not surprising, since the high organochlorine exposure occupations, such as capacitor workers or pest control workers are male dominated to date, and were so in the past when occupational exposure to these compounds was wide spread.

Lactation is considered the most important route of excretion of these compounds for women (Jensen, 1983). In these data, however, there was no trend of decreasing organochlorine levels with increasing duration of lactation. There was also no difference in mean organochlorine levels among women who never breastfed and women who ever breastfed their infants. This lack of a strong inverse association may be due greater exposure to organochlorines after these women breastfed their last child. These findings are also consistent with data from the Nurses Health Study, where the correlations between duration and lactation and serum levels of DDE and total PCBs were weak in magnitude (r=-0.10) (Hunter, personal communication).

OUTLINE FOR FUTURE ANALYSES

A. Organochlorines and breast cancer

Currently, the main effort of the work supported by this grant focuses on examining the association between serum organochlorine levels and breast cancer risk. Specifically, breast cancer cases will be compared to controls with regard to levels of DDE, HCB, total PCBs, PCB congener groups, and mirex. This association will be investigated in the entire sample as well as among women with and without a history of lactation. The rationale behind the latter analyses relates to differences in elimination of these compounds between these groups. These analyses are ongoing, but results are too preliminary to be included in this report.

As mentioned above, serum levels of groups of individual PCB congeners will be examined in relation to breast cancer risk. A meaningful approach in grouping these compounds is also currently being developed. Grouping approaches under consideration include: a) grouping with regard to biochemical characteristics (degree of chlorination of

the biphenyl molecule); b) grouping with regard to toxicological characteristics (P-450 enzyme induction activity); and c) grouping based on statistical properties (factor analysis).

Before the end of this year, we expect that data on estrogen receptor of the breast cancer patients status will become available. With these data, it will be possible to examine whether the organochlorine exposure has differing effects on estrogen receptor positive and estrogen receptor negative breast cancer.

B. Examination of gene-environment interactions

Upon completion of the analyses examining the effect of organochlorines on breast cancer risk in the entire sample, we will investigate whether rare polymorphisms in genes involved in carcinogen metabolism and detoxification will act as effect modifiers of the associations of these compounds with risk.

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APPENDIX

List of publications based on work supported by this grant:

Ambrosone CB, Freudenheim JL, Marshall JR, Graham S, Vena JE, Brasure JR, Michalek AM, Laughlin R, Nemoto T, Shields PG. The Association of polymorphic N-acetyltransferase (NAT2) with breast cancer risk. In: Bradlow HL, Osborne MP, Veronesi U (eds). Cancer Prevention:from the laboratory to the clinic - implications of genetic, molecular, and preventive research. Annals of the New York Academy of Sciences, Vol. 768:250-54. New York, 1995.

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Recd 1/11/2000

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US ARMY MEDICAL RESEARCH AND MATERIEL COMMAND
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REPLY TO
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PHYLIS M. RINEHART
Deputy Chief of Staff for
Information Management

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