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Persistent organochlorines in the serum of the non-occupationally exposed New Zealand population

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Abstract

Concentrations of polychlorinated dibenzo-*p*-dioxins (PCDDs), polychlorinated dibenzofurans (PCDFs), polychlorinated biphenyls (PCBs), and organochlorine pesticides were measured in the serum of a sample of the New Zealand population aged 15 years and older. This was the first study to obtain representative measures of PCDDs, PCDFs and PCBs in the adult population of an entire country. Serum samples were obtained in 1996–1997. Potentially occupationally exposed individuals were excluded. Serum samples were pooled according to stratification criteria for area of residence, ethnicity, age, and sex. Of the 80 possible strata, sufficient serum for chemical analysis was available for 60, to which 1,834 individual samples contributed. For the PCDDs and PCDFs, most 2,3,7,8-chlorinated congeners were measured in all strata, with a mean toxic equivalents concentration across all strata of 12.8 ng TEQ kg^{-1} lipid. Seven PCB congeners were frequently measured, including the coplanar congeners #126 and #169, quantified in all strata. Of the pesticides and their metabolites, only β-HCH, dieldrin and *pp′*-DDE were consistently detected across strata. There was a general trend of increasing concentration with age. There were no consistent differences between the sexes, or between people of Maori (the indigenous people of New Zealand) and non-Maori ethnicity. Concentrations of PCDDs and PCDFs tended to increase in a North–South direction, possibly reflecting greater levels of industrialization and population concentration, and concentrations of the pesticide products were highest in the South, possibly reflecting historical use patterns. Results were consistent with a recent study of concentrations of these compounds in the milk of first-time mothers.

Keywords: Dioxins; New Zealand; Polychlorinated dibenzo-*p*-dioxins; Polychlorinated dibenzofurans; Polychlorinated biphenyls (PCBs); Organochlorine pesticides

1. Introduction

The polychlorinated dibenzo-*p*-dioxins (PCDDs), polychlorinated dibenzofurans (PCDFs), polychlorinated biphenyls (PCBs) and organochlorine pesticides, such as DDT and dieldrin, are prominent among the

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chemicals often referred to as persistent organic pollutants (POPs). These chemicals are widely dispersed in the environment, and are probably present in all human beings, particularly those in the industrial world. Concern about the toxicity and the chemical/physical properties of POPs has led to the development of an international treaty, the Stockholm Convention, which aims to protect human health and the environment by reducing exposure to these chemicals (Karlaganis et al., 2001).

Because of their persistence, resistance to degradation, and fat solubility, measurements of POPs in the lipid component of body tissues and fluids provide a good indication of cumulative exposure. Such exposures have been shown to vary widely across populations. However, most studies of body burdens have involved small geographic areas, selected subsets of populations, or occupationally exposed groups. To our knowledge, the present study was the first to obtain population-based estimates of the body burdens of the PCDDs, PCDFs and PCBs for the entire adult population of a country, although data have now been published for the United States (National Center for Environmental Health, 2003).

Many studies of organochlorines have involved human milk samples (Bates et al., 1994; LaKind et al., 2001). Such samples have advantages: they can be obtained non-invasively, milk contains a relatively high proportion of fat (in which the substances accumulate), they provide a good measure of infant exposure, and studies in different countries have often used similar protocols, permitting inter-country comparison of results (WHO, 1996). However, human milk samples suffer from the disadvantage that they can be collected only from a particular subset of any population. Blood serum, on the other hand, can be obtained from anyone, although its collection is invasive, and it contains a smaller lipid fraction than breast milk. For obtaining population-based estimates of POP exposures, the advantages of serum outweigh the disadvantages.

In 1995, the New Zealand Ministry for the Environment commenced a national Organochlorines Programme to characterize the extent of contamination of the New Zealand environment by PCDDs, PCDFs, PCBs, and organochlorine pesticides, and to establish risk-based environmental acceptance criteria for these substances. A series of detailed scientific investigations on population and environmental exposures to organochlorines, including surveys to determine the concentrations of organochlorine residues in food produce, human serum, and environmental media were undertaken. This paper reports key results from the serum study. Results of analyses in other media have been reported elsewhere (Buckland et al., 1998a,b,c, 1999; Scobie et al., 1998).

Scientific objectives of the serum study were to:

- Obtain estimates of baseline concentrations of PCDDs, PCDFs, PCBs and organochlorine pesticides in serum sampled from the non-occupationally exposed New Zealand population.
- Determine the relationships of organochlorine contaminant concentrations to age, ethnicity, sex and geographic region.

The present study was undertaken to determine the background concentrations of organochlorine substances in serum from a cross-sectional sample of adult New Zealanders. It was not intended to identify or characterize highly exposed or at-risk populations (such as occupational groups), or to assess exposures related to known emission point sources.

2. Materials and methods

2.1. Population sample selection

Collection of serum samples was carried out in conjunction with the National Nutrition Survey (NNS) (Quigley and Watts, 1997), which was itself linked to the New Zealand Health Survey (NZHS) (Ministry of Health, 1999). The target population of the NZHS was all New Zealanders aged 15 or older. To increase the number of Maori (the indigenous people of New Zealand) and Pacific Island people in the NZHS, additional households were selected from areas in which there was a high proportion of Maori or Pacific Island people. The sampling frame is described in detail elsewhere (Buckland et al., 2001).

2.2. Serum collection, processing and transportation

During the period December 1996–November 1997, NNS subjects were interviewed and up to three 10 ml vacutainers of blood were collected by trained phlebotomists. The first two vacutainers were used for the NNS and the third vacutainer of blood was allocated to the organochlorines study. The interview questionnaire contained questions about possible occupational exposures to organochlorine compounds.

Shortly after blood collection, vacutainers were centrifuged and the separated serum stored in vials at −70 °C, pending transportation on dry ice to the analytical laboratory (Centers for Disease Control and Prevention, Atlanta, Georgia).

2.3. Serum sample pooling

From each participant, up to 10 ml of whole blood was collected and processed to provide serum for ana-

lysis. The optimal volume of serum required for analysis, to achieve the desired limits of detection for PCDDs, PCDFs and non-*ortho* PCBs, was approximately 50 ml. Therefore, to achieve adequate volumes for analysis, individual serum samples were pooled according to defined criteria. Only individuals who did not report evidence of occupational exposure to organochlorines were included in the pooled samples.

Pools were defined based on age (5 groups), ethnicity (Maori and non-Maori), sex and geographic region (four regions, running North to South). To ensure that no individual was overly influential on the pooled results, all serum samples included in any one pool were of equal volume (minimum of 2 ml). The stratification criteria provided a total of 80 possible strata for the study. To maximize the volume of serum available for analysis, and because individual serum samples of equal volume were used within any one stratum, not all collected samples could be used.

A minimum number of five eligible subjects were required for pooling in any stratum to reduce the possible impact on the pooled analytic results of individuals who had had unusually high exposures, and who had not been identified by the screening questions as potentially being occupationally exposed. When five eligible subjects were not available in a particular stratum, then no pooled serum sample was prepared and no organochlorine concentration data were obtained for that stratum. A minimum pooled serum volume of 25 ml was required for a stratum to be included in the study, although a volume of 50 ml was preferred, to optimize limits of detection.

2.4. Chemical analysis

The pooled serum samples were analyzed for the following organochlorine compounds:

PCDDs and PCDFs. All seventeen 2,3,7,8-chlorinated congeners were determined.

PCBs. Using the congener numbering system of Ballschmiter and Zell (1980), four non-*ortho* PCB congeners (#81, #77, #126, #169) and 38 *ortho* PCB congeners (PCB #18, #28, #52, #49, #44, #74, #66, #101, #99, #87, #110, #118, #105, #151, #149, #146, #153, #138, #158, #128, #167, #156, #157, #178, #187, #183, #177, #172, #180, #170, #189, #201, #196, #203, #195, #194, #206, #209) were determined.

Pesticides. Hexachlorocyclohexanes (β- and γ-HCH), hexachlorobenzene (HCB), aldrin, dieldrin, endrin, *op′*-DDT, *pp′*-DDT and mirex were determined, along with the pesticide byproducts, metabolites, and degradation products: heptachlor epoxide, *trans*-nonachlor, oxychlordane and *pp′*-DDE.

PCDDs, PCDFs and non-*ortho* PCBs were extracted from serum by C_{18} solid phase extraction (SPE) and purified using a multi-column cleanup of acid/neutral/basic silica, followed by acidic alumina, then carbon. *Ortho*-PCBs and organochlorine pesticides were extracted by C_{18} SPE and purified by Florisil.

Target analytes were quantified by ^{13}C isotope dilution using capillary gas chromatography high resolution mass spectrometry operating in the selected ion monitoring mode. Data are reported on a lipid weight basis, corrected for recovery of ^{13}C surrogate standards. Full methodological details are reported elsewhere (Buckland et al., 2001).

2.5. Presentation of data

PCDD, PCDF and PCB toxic equivalency (TEQ) data were calculated using the toxic equivalency factors (TEFs) proposed by the World Health Organization (WHO) in 1997 (Van den Berg et al., 1998).

For the PCDDs and PCDFs, almost all congeners were detected in almost every stratum. Consequently, there is little difference in the TEQ level determined, regardless of whether limit of detection (LOD) values are included or excluded from the TEQ calculation. However, a large number of PCB congeners were not quantified, and are reported as less than the analytical LOD. As a result, there are marked differences in the TEQ levels calculated when either including or excluding LOD values. Because of the impact of LOD values on the TEQ, the values obtained for "PCB TEQ (exc LODs)", which treat undetected PCB congeners as having zero concentration, are generally more informative than "PCB TEQ (inc l/2 LODs)", which assign non-quantified PCB congeners a concentration of half their LOD value.

In all tables, median and mean concentrations are shown only for those compounds that were detected in at least 66% of all strata, or 66% of strata grouped on the basis of age, ethnicity, or sex. The reason was that, if a contaminant was not frequently quantified, the estimated median or mean value might be non-representative of the true value for New Zealanders. In the calculation of medians and means, individual stratum concentrations were weighted in terms of the numbers of samples contributing to the pooled sample for each stratum.

3. Results

3.1. Sample numbers

The selected sample invited to participate in the NZHS consisted of 10 600 individuals. Of these, 7016 completed the NZHS, and of these, 5613 agreed to be contacted for participation in the NNS. 4644 were actually interviewed and 3376 provided a blood sample for the NNS. Of these people, 2925 supplied a third vacutainer

of blood for organochlorine analysis and responded to questions on occupational exposure. Of these vacutainers, 2788 contained at least 2 ml of serum, and 2497 (1487 females and 1010 males) were judged eligible for inclusion in the current study on the basis of there being no evidence of occupational exposure. Of the eligible samples, 1834 (1034 females and 800 males) were actually included in pooled samples. The other samples were not used because of the necessity to maximize the total serum volume available for analysis and because of the requirement for each individual to contribute an equal amount of serum to a pool.

Of the possible 80 strata, 20 had insufficient eligible subjects or pooled serum volume for inclusion in the study, leaving a total of 60 eligible strata for which organochlorine concentration data were obtained. All of the ineligible strata were for Maori people, and particularly in the older age groups.

The full serum concentration dataset is available at: http://www.mfe.govt.nz/issues/waste/ocreports.htm.

3.2. PCDDs and PCDFs

Minimum, maximum, weighted median and weighted mean, and age group-specific concentrations for individual 2,3,7,8-PCDD and PCDF congeners, and the sums of 2,3,7,8-PCDD/PCDF congeners and PCDD/PCDF TEQs across all strata are reported in Table 1. Reference to PCDD and PCDF TEQ levels in this section refers specifically to TEQs including half LOD values. However, as previously mentioned, the inclusion of LOD values has minimal effect on the TEQ level calculated for these contaminants, and comments would apply equally to TEQ levels calculated after excluding congeners with LOD values.

The congeners detected at the highest concentrations were 1,2,3,4,6,7,8-HpCDD and OCDD. 2,3,7,8-TCDD was detected in almost all strata, at a maximum concentration of 7.0 ng kg^{-1} lipid and 1,2,3,7,8-PeCDD was detected in all strata at a maximum concentration of 9.3 ng kg^{-1} lipid. The three 2,3,7,8-HxCDD congeners were also detected in more than 90% of strata, and in a concentration profile dominated by 1,2,3,6,7,8-HxCDD, which is typical of the profile normally observed in human tissue (Päpke, 1998).

2,3,7,8-TCDF was detected in less than 50% of strata, at a maximum concentration of 0.7 ng kg^{-1} lipid. In contrast, 2,3,4,7,8-PeCDF, which was more frequently detected and present at higher levels than 1,2,3,7,8-PeCDF, was quantified in all strata to a maximum concentration of 8.3 ng kg^{-1} lipid. Three of the four HxCDF congeners were detected in all or almost all of the strata, while 1,2,3,7,8,9-HxCDF was not quantified in any strata. Again, the profile of the 2,3,7,8-HxCDF congeners is typical of that observed in human tissue (Päpke, 1998). OCDF was not quantified in any strata.

The age group-specific data show a trend toward higher concentrations in the older age groups, from 6.69 ng TEQ kg^{-1} lipid for the 15–24 age group to 20.7 ng TEQ kg^{-1} lipid for the 65+ age group. The mean PCDD and PCDF TEQ level across all strata was 12.8 ng TEQ kg^{-1} lipid.

The variation in mean PCDD and PCDF TEQ levels for the four possible combinations of ethnicity and sex in our data, across age groups, is shown in Fig. 1. Again, the trend of increasing PCDD and PCDF concentrations with increasing age is evident. The data also show:

1. There is little difference in the mean TEQ levels between males and females and Maori and non-Maori within the three younger age groups.
2. The mean TEQ concentration in Maori females in the 50-64 year age group is markedly higher than for Maori females in any of the other age groups. However, this result is based on the analysis of a single pooled sample comprised of only five individual serum samples.
3. In the 65+ age group, non-Maori females had appreciably higher TEQ levels than non-Maori males.

No comparison of PCDD and PCDF concentrations between Maori and non-Maori could be made for the 65+ year age group, as there were insufficient serum samples collected from Maori aged 65+ for measurements to be obtained.

Although the indvidual 2,3,7,8-substituted congener profiles for PCDDs and PCDFs are not shown here, they showed no consistent differences in exposure patterns between Maori and non-Maori, or between males and females.

3.3. Polychlorinated biphenyls

Minimum, maximum, weighted median, weighted mean, and age group-specific concentrations for selected PCB congeners, and sums of PCB congeners and PCB TEQ across all strata are reported in Table 2. This table presents data only for those congeners that were frequently detected, since median and mean results were calculated only if a congener was detected in at least 66% of strata.

Of the four non-*ortho* PCBs, congeners #126 and #169 were detected in all strata, PCB #81 was detected in only three strata (at a maximum concentration of 0.0029 μg kg^{-1} lipid), and PCB #77 was not detected in any strata. Limits of detection for PCB #81 were in the range 0.002–0.02 μg kg^{-1} lipid and for PCB #77 in the range 0.03 to 0.2 μg kg^{-1} lipid.

Of the *ortho*-substituted congeners, those consistently detected in most strata and at the highest concentrations were PCBs #153, #138+#158, and #180. Congeners #74, #118, #187, #170 and #194 were detected

Table 1
Concentrations of PCDDs and PCDFs in the serum of adult New Zealanders

Congener	TEF	No. of positives	Concentration, ng kg^{-1} lipid weight basis[a]								
			Minimum	Maximum	Weighted median	Weighted mean[b]	Mean				
							15–24 years	25–34 years	35–49 years	50–64 years	65+ years
2,3,7,8-TCDD	1	58	<1	7.0	2.0	2.3	1.0	1.4	2.0	3.1	4.6
1,2,3,7,8-PeCDD	1	60	2.0	9.3	4.9	4.7	2.6	3.4	4.7	5.8	7.2
1,2,3,4,7,8-HxCDD	0.1	56	<2	5.9	2.7	2.8	1.3	1.9	3.0	3.5	4.4
1,2,3,6,7,8-HxCDD	0.1	58	<0.7	39.6	20.2	20.8	8.3	15.2	21.7	26.3	33.6
1,2,3,7,8,9-HxCDD	0.1	59	<2	8.3	3.9	4.2	2.6	3.4	4.1	4.9	6.1
1,2,3,4,6,7,8-HpCDD	0.01	58	<30	85.9	37.4	38.4	21.7	32.3	38.6	47.3	53.3
OCDD	0.0001	60	143	961	366	361	227	320	385	411	455
2,3,7,8-TCDF	0.1	23	<0.2	0.7	nc	nc	nc	nc	nc	nc	0.3
1,2,3,7,8-PeCDF	0.05	12	<0.2	0.7	nc	nc	nc	nc	nc	nc	nc
2,3,4,7,8-PeCDF	0.5	60	1.8	8.3	3.8	4.0	2.3	2.9	4.0	5.0	6.1
1,2,3,4,7,8-HxCDF	0.1	60	0.90	4.3	2.1	2.2	1.3	1.6	2.1	2.7	3.3
1,2,3,6,7,8-HxCDF	0.1	60	1.1	4.6	2.5	2.6	1.5	2.0	2.5	3.2	3.7
2,3,4,6,7,8-HxCDF	0.1	56	<0.9	1.4	0.8	0.8	0.5	0.7	0.8	1.0	0.9
1,2,3,7,8,9-HxCDF	0.1	0	<0.2	<2	nc	nc	nc	nc	nc	nc	nc
1,2,3,4,6,7,8-HpCDF	0.01	30	<5	21.7	nc	nc	nc	nc	5.9	4.8	nc
1,2,3,4,7,8,9-HpCDF	0.01	29	<0.3	1.2	nc	nc	nc	nc	0.3	nc	nc
OCDF	0.0001	0	<1	<200	nc	nc	nc	nc	nc	nc	nc
Sum of 2,3,7,8-PCDD/Fs (inc 1/2 LODs)[c]			178	1170	465	459	287	399	487	526	589
Sum of 2,3,7,8-PCDD/Fs (exc LODs)[c]			174	1070	440	447	270	387	474	518	582
PCDD/F TEQ (inc 1/2 LODs)[c]			5.05	26.7	12.4	12.8	6.69	9.27	12.6	16.1	20.7
PCDD/F TEQ (exc LODs)[c]			4.98	26.7	12.4	12.7	6.54	9.18	12.6	16.1	20.7

Abbreviations: nc, not calculated (median or mean reported only if a congener was detected on more than 66% of occasions across all strata); inc 1/2 LODs, half the limits of detection were included in the sum of 2,3,7,8-PCDD/Fs and the TEQ calculation; exc LODs, values below LOD were excluded from the sum of 2,3,7,8-PCDD/Fs and the TEQ calculation.

[a] For any congener, calculation of the median and mean includes half LOD values.

[b] Weighted mean across all strata; $n = 60$, except $n = 59$ for 1,2,3,7,8-PeCDF and $n = 58$ for 1,2,3,4,7,8-HxCDD.

[c] Sum of 2,3,7,8-PCDD/Fs and TEQ data are taken or calculated from the individual strata analyzed, and not from the summarized congener data reported in the table.

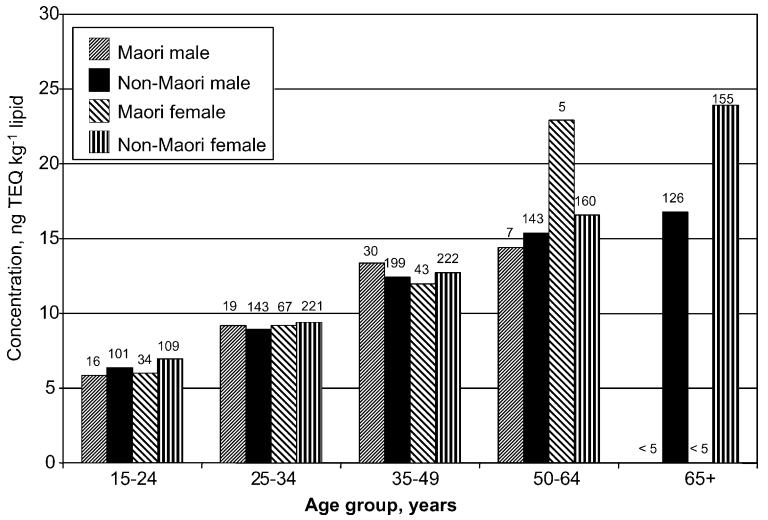

Fig. 1. Mean PCDD and PCDF TEQ in serum, by age, ethnicity and sex. (The number at the top of each column represents the number of individual serum samples that contributed to the mean concentration. If insufficient serum samples (less than 5) were collected from an ethnicity-sex group for analytical measurements to be made, a "<5" notation is shown where the column would otherwise have been.)

primarily in the older age groups, and at comparatively lower concentrations.

In addition to the PCBs reported in Table 2, a few other *ortho*-substituted congeners were detected in a limited number of strata, namely PCB #146 (detected in 8 strata), #156 (11 strata), #183 (2 strata), #177 (3 strata), #201 (8 strata) and #196 + #203 (8 strata). Typical limits of detection for *ortho*-substituted congeners ranged between 4 and 20 $\mu g\,kg^{-1}$ lipid.

As with PCDDs and PCDFs (Table 1), there is a general trend of increasing PCB concentration with age.

For ease of comparison with the PCDD and PCDF TEQ data reported in Table 1, PCB TEQ levels in Table 2 are expressed as $ng\,kg^{-1}$ lipid, rather than $\mu g\,kg^{-1}$ lipid. At the bottom of the table, the PCDD and PCDF TEQs (from Table 1) are summed with PCB TEQs to give overall TEQ levels. When undetected congeners are excluded, the PCBs contribute approximately 15–30% of the total TEQ determined. This comparison of TEQ levels excluding LODs is justified because the estimation of PCB TEQs was markedly affected by inclusion of assumed levels for non-detected congeners in the calculation.

Mean concentrations of PCBs in the four combinations of ethnicity and sex, by age group, are presented in Figs. 2 and 3. For Fig. 2, results are shown as PCB TEQ and include half LOD values for non-detected congeners, for direct comparability with Fig. 1. For Fig. 3, concentrations are expressed as the sum of PCB congeners, for which measurements lower than the LOD were set at zero. The increase in PCB concentration with age is more apparent when data are expressed as the sum of congeners (Fig. 3) than as PCB TEQ. A clear increasing concentration trend with age is also evident for congener #126. This congener was consistently the major contributor to TEQ levels, and was quantified in all strata. A suggestion of higher PCB congener levels in males than in females is evident in Fig. 3. As was observed with PCDDs and PCDFs (Fig. 1), the PCB concentration in Maori females in the 50–64 age group appears unusually elevated. Again, this result is based on a single pooled sample made up of a minimal number (five) of serum samples.

For the individual congeners, other than age-trends, there were no clear and consistent patterns, except that congener-specific concentrations in males (Maori and non-Maori) tended to be similar, as were concentrations in females.

3.4. Organochlorine pesticides

Minimum, maximum, weighted median and weighted mean and age group-specific concentrations for organochlorine pesticides, and their degradation products, across all strata are presented in Tables 3 and 4. For the calculation of means, concentrations at or below the LOD were set at zero. Median and mean results were calculated only if a pesticide was detected in at least 66% of all strata.

The most frequently detected pesticides were *pp*′-DDE (detected in all 60 strata), dieldrin (57 strata), β-HCH (40 strata) and *pp*′-DOT (18 strata). All of the other pesticides were either not detected, or detected in less than six strata.

Table 2
Concentrations of PCBs in the serum of adult New Zealanders

Congener	TEF	No. of positives	Concentration, $\mu g\,kg^{-1}$ lipid weight basis[a]								
			Minimum	Maximum	Weighted median	Weighted mean[b]	Mean				
							15–24 years	25–34 years	35–49 years	50–64 years	65+ years
PCB #126	0.1	60	0.011	0.065	0.023	0.030	0.014	0.019	0.023	0.035	0.045
PCB #169	0.01	60	0.0076	0.038	0.021	0.020	0.010	0.015	0.023	0.027	0.030
PCB #74		20	<4	10.9	nc	nc	nc	nc	nc	4.7	7.5
PCB #118	0.0001	20	<4	10.0	nc	nc	nc	nc	nc	4.9	7.0
PCB #153		60	6.1	49.9	24.6	23.7	9.1	16.1	25.2	32.6	36.6
PCB #138 + #158		56	<6	36.2	15.6	15.3	5.6	10.6	15.9	20.9	24.2
PCB #187		30	<4	15.3	nc	nc	nc	nc	5.2	7.2	8.5
PCB #180		57	<7	44.6	20.0	20.3	7.3	13.5	22.2	28.1	30.9
PCB #170		42	<5	18.6	8.6	8.4	nc	nc	9.3	11.6	12.7
PCB #194		22	<6	9.2	nc	nc	nc	nc	nc	5.1	5.9
Sum of PCBs (inc 1/2 LODs)[c]			127	286	192	201	170	172	210	219	237
Sum of PCBs (exc LODs)[c]			6.12	208	81.9	79.0	20.8	44.9	81.8	118	139
PCB concentration, ng TEQ kg^{-1} lipid weight basis											
PCB TEQ (inc 1/2 LODs)[d]			4.29	11.9	6.42	6.86	5.90	5.80	6.50	7.60	9.20
PCB TEQ (exc LODs)[d]			1.21	10.2	2.46	3.33	1.50	2.00	2.70	4.80	6.50
Total TEQ concentration for PCBs, PCDDs and PCDFs (from Table 1), ng TEQ kg^{-1} lipid weight basis											
PCDD/F TEQ + PCB TEQ (inc 1/2 LODs)[e]			9.71	38.5	18.7	19.7	12.6	15.1	19.1	23.7	29.9
PCDD/F TEQ + PCB TEQ (exc LODs)[e]			6.19	36.9	14.8	16.1	8.04	11.2	15.3	20.9	27.2

Abbreviations: nc, not calculated (median or mean reported only if a congener was detected on more than 66% of occasions across all strata); inc 1/2 LODs, half the limits of detection were included in the sum of 2,3,7,8-PCDD/Fs and the TEQ calculation; exc LODs, values below limits of detection were excluded from the sum of 2,3,7,8-PCDD/Fs and the TEQ calculation.

[a] For any individual congener, calculation of the median and mean includes half LOD values.

[b] $n = 60$.

[c] Sum of PCBs is the sum of all 42 congeners analyzed.

[d] PCB TEQ is the TEQ calculated for the 10 dioxin-like PCB congeners analyzed.

[e] For minimum, maximum and median, these data are derived from the individual strata, not from a summation of the PCDD/F TEQ (Table 1) and PCB TEQ (this table).

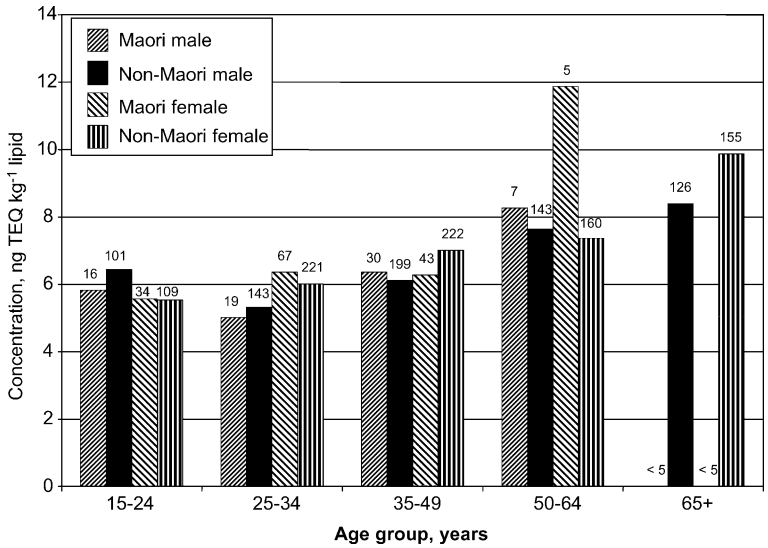

Fig. 2. Mean PCB TEQ in serum, by age, ethnicity and sex. (For an explanation of the number at the top of each column, see Fig. 1.)

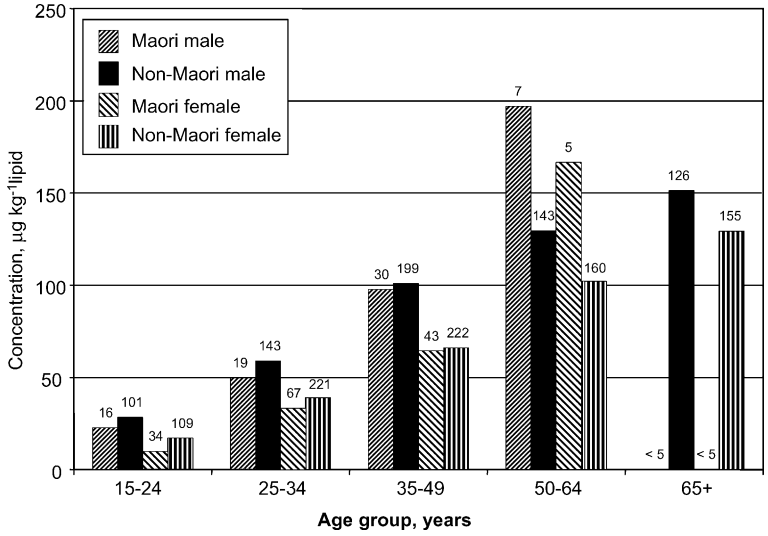

Fig. 3. Mean sum of PCB congeners in serum, by age, ethnicity and sex. (For an explanation of the number at the top of each column, see Fig. 1.)

In the remainder of this paper, the analysis of pesticide concentration data was limited to β-HCH, dieldrin, and *pp′*-DDE, which were the only pesticides detected in at least 66% of strata. For dieldrin and *pp′*-DDE there is a tendency for increasing concentration with age, but no such age-related increase was observed with β-HCH. However, any such trend may have been obscured because this pesticide was infrequently detected in the two youngest age groups. When the data were analyzed by geographic region (data not shown), an-age related trend was apparent.

Mean concentrations of β-HCH, dieldrin, and *pp′*-DDE stratified by ethnicity and sex are reported in Table 5. Mean concentrations are based only on those age groups and the two regions (Lower North Island and the South Island) for which there were data available for each of the four Maori/non-Maori and male/female combinations. Across age strata, there were no consistent differences by ethnicity and sex.

4. Discussion

This study represents the first ever cross-sectional survey of concentrations of PCDDs, PCDFs, PCBs and organochlorine pesticides in a representative sample of

Table 3
Concentrations of organochlorine pesticides in the serum of adult New Zealanders

Pesticide	Number of positives	Concentration, $\mu g\,kg^{-1}$ s lipid weight basis[a]			
		Minimum	Maximum	Weighted median	Weighted mean ($n = 60$)[b]
β-HCH	40	<7	73.1	10.7	19.7
γ-HCH	1	<5	91.1	nc	nc
HCB	4	<20	53.6	nc	nc
Aldrin	0	<3	<8	nc	nc
Dieldrin	57	<8	28.4	11.5	14.2
Endrin	0	<5	<9	nc	nc
Heptachlor epoxide	0	<3	<8	nc	nc
Oxychlordane	0	<3	<8	nc	nc
t-Nonachlor	3	<5	8.4	nc	nc
pp′-DDE	60	413	2780	919	1080
op′-DDT	0	<10	<20	nc	nc
pp′-DDT	18	<20	49.2	nc	nc
Mirex	0	<3	<10	nc	nc

[a] For any individual pesticide, calculation of the median includes LOD values and calculation of the mean excludes values below the LOD.
[b] $n = 59$ for β-HCH, γ-HCH, aldrin, heptachlor epoxide, oxychlordane, *t*-nonachlor and *op*′-DDT; $n = 55$ for endrin.

Table 4
Weighted mean concentrations of organochlorine pesticides in serum, by age

Pesticide	Concentration, $\mu g\,kg^{-1}$ hold weight basis[a]				
	15–24 years	25–34 years	35–49 years	50–64 years	65+ years
β-HCH	nc	nc	24.9	18.2	24.0
Dieldrin	10.8	11.5	12.0	16.1	22.4
pp′-DDE	646	771	1060	1310	1780

Abbreviation: nc, not calculated (mean value reported only if a pesticide was detected in more than 66% of strata for that age group).
[a] For any individual pesticide, calculation of the mean excludes values below the LOD.

Table 5
Weighted mean concentrations of organochlorine pesticides in serum, by ethnicity and sex

Pesticide	Concentration, $\mu g\,kg^{-1}$ lipid weight basis[a]			
	Female		Male	
	Maori	Non-Maori	Maori	Non-Maori
β-HCH	nc	9.4	nc	21.0
Dieldrin	14.6	10.9	13.5	11.7
pp′-DDE	920	782	1110	1080

Abbreviation: nc, not calculated (mean value reported only if a pesticide was detected in more than 66% of strata for that ethnicity-sex group).
[a] Weighted mean calculated using data only from the lower North Island and South Island regions and for the 15–24, 25–34 and 35–49 age groups. Calculation of the mean excludes values below the LOD.

the non-occupationally exposed adult New Zealand population. It is believed to be, at least for the PCDDs, PCDFs and PCBs, the first truly national survey of its kind ever undertaken in any country. However, recently data on organochlorine concentrations have been reported from samples collected in the National Health and Nutrition Examination survey (NHANES) during 1999–2000, across the United States (National Center for Environmental Health, 2003), This study has provided data against which future serum surveys can be compared, for inter-country comparisons, monitoring of temporal trends at a national or regional level, or for the assessment of exposed or potentially exposed subpopulations.

Overall, PCDD, PCDF and PCB congeners were consistently detected across all ages, geographic regions,

sexes and ethnic groups. Three pesticides (including degradation products) were also consistently detected: β-HCH, dieldrin and *pp′*-DDE. Of these, *pp′*-DDE was present in highest concentrations, being quantified at concentrations typically 50–100 times the levels found for other pesticides.

No *consistent* differences in contaminant concentrations between Maori and non-Maori or between males and females were found. Some variation in organochlorine concentrations between males and females for the oldest age group (65+ years) was observed, and the sum of PCB congeners tended to be marginally higher in males than in females for all age groups. There were some regional differences in organochlorine concentrations (data not shown). In particular, PCDD, PCDF and PCB concentrations appeared to be marginally higher in the two most northerly regions relative to the two more southerly regions. These differences may reflect the higher population density and level of industrialization in the north of New Zealand. *pp′*-DDE concentrations increased markedly in a north to south direction. Such a trend has previously been found in a study of organochlorines in breast milk (Bates, 1994) and is likely to reflect historical use patterns of the pesticide DDT.

A consistent trend found throughout the results of this study was a tendency for serum concentrations to progressively increase with age. In theoretical terms, one would expect that, given constant exposure over a long period, levels would plateau and stay fairly constant from the point at which the rate of excretion equalled the rate of absorption (i.e., once a steady state had been reached). There is uncertainty about the half-lives in humans of many of the chemicals studied. However, one chemical that has been thoroughly investigated and for which a number of estimates of the half-life exist, is 2,3,7,8-TCDD. Half-life estimates for this chemical range from several years to several decades, with a mean value of around 7.8 years (Geyer et al., 2002). Assuming a half-life of 7.8 years, and given constant exposure to 2,3,7,8-TCDD, it can be calculated that the body burden (as reflected in serum concentrations) would plateau at about age 30–40 years, or after 4–5 half-lives (Sim and McNeil, 1992). Such a plateauing has not been observed for TCDD in the current study (Table 1), nor for most of the other compounds measured (Tables 1–4).

There are at least three possible explanations for the absence of plateau concentrations. Any or all of these three mechanisms may be involved. First, earlier exposures to all the chemicals investigated are likely to have been higher than in more recent time periods. A number of measures to reduce population exposures to organochlorines have been implemented over the last 30 years in New Zealand. For example, the persistent organochlorine pesticides, including DDT and dieldrin, have not been used in agriculture since the 1970s. The use of PCBs has been prohibited since 1995, and measures to reduce emissions of PCDDs and PCDFs have been progressively implemented. Comparison of New Zealand breast milk studies conducted a decade apart supports such an explanation (Bates et al., 2002). Studies in other countries have also shown that population exposures to organochlorines have fallen in recent decades (Päpke, 1998; Wittsiepe et al., 2000). Despite this, it is not clear that the differences in exposures over time would have been sufficient alone to explain the observed results from this study.

A second possible explanation is that there may be variation in half-life by age, such that excretion slows down as people get older. What is known about half-lives of organochlorines in humans, particularly for 2,3,7,8-TCDD, is for the most part based on studies that have investigated relatively highly exposed populations of workers (primarily male) and younger people. The assumption has generally been made that results obtained for such groups are generalizable across the entire population, but this may not be so. Van der Molen et al. (1998) have produced evidence that the half-life of 2,3,7,8-TCDD increases with age, and Kreuzer et al. (1997) have concluded that half-life increases from about 5 months in infants to approximately 10 years between 40 and 60 years of age. If the mechanisms in the human body for the elimination of persistent organochlorine chemicals are common across the range of these chemicals, then this might have contributed to the observed results.

Thirdly, it is possible that half-life is concentration-dependent, such that lower body burdens are excreted more slowly than higher body burdens, In other words, the relatively low body burdens found in members of the general population may have particularly long half-lives, such that even in older people four or five half-lives have not yet been achieved. Most studies of half-lives have been conducted in populations with high exposures compared to background levels (Michalek et al., 2002). In a review of the literature, Geyer et al. (2002) conclude that there is evidence that lower body burdens of dioxin compounds are associated with longer half-lives.

Although no general differences between the sexes or between Maori and non-Maori were noted, there was some suggestion that women in the highest age group tended to have higher serum concentrations than men in the same age group. This was particularly evident for PCDDs and PCDFs, as TEQ (Fig. 1), some PCBs, and β-HCH, dieldrin and *pp′*-DDE (data not shown). This cannot be explained by a difference in the average age of men and women in that group (which might have been expected, given the longer life expectancy of women compared with men), as both sexes in that age range had an identical mean age of 72.9 years. The reason for the comparatively higher concentration in non-Maori females aged 65+ may be because half-lives

increase with body fat content, and females possess on average more body fat than males of the same age (Geyer et al., 2002).

Because the study was intended to determine general population background concentrations and chemical analyses were carried out on pooled samples, an effort was made to identify and exclude anyone who was likely to have had occupational exposure to any of the chemicals being investigated. This was to minimize the possibility that inclusion of serum samples from 20 such people in a pooled serum sample could have produced a result that was not truly representative for that particular stratum. A minimum number of five serum samples in a pooled sample was also required to further attempt to minimise the impact of outliers. Despite these efforts, some of the results obtained suggest that the exclusion procedures may not have succeeded in entirely eliminating unusually highly exposed people from the study population. For example, in the pooled sample representing Maori women aged 50–64, the results for PCDDs and PCDFs (Fig. 1) and PCBs (Fig. 2) are all markedly elevated above what might be expected, based on comparison with the other strata. The pooled sample that generated these results contained serum from only five individuals. It is most likely that the elevated levels are the result of high concentrations, which were non-representative of New Zealand baseline concentrations, in the serum sample from a single woman. However, whether these higher concentrations are associated with historical exposure to PCBs is unclear. PCBs were primarily contaminated with PCDFs rather than PCDDs, and the PCDF concentrations in the pooled sample were not unduly elevated. The increase in PCDD and PCDF TEQ was mainly influenced by comparatively higher concentrations of two PCDD congeners, 2,3,7,8-TCDD and 1,2,3,7,8-PeCDD.

The data from this cross-sectional study tell us little about trends in organochlorine exposures in New Zealanders, as no previous study has been carried out using a comparable population-based sample. However, two small studies conducted for other purposes suggest that there may have been some reductions in exposures. The mean PCDD and PCDF concentration in serum collected in 1988 from nine New Zealand men (average age 53 years, range 45–62 years), used as a control group in another study, was 24.5 ng TEQ kg^{-1} lipid (Smith et al., 1992). By comparison, the weighted mean PCDD and PCDF concentration for males aged 50–64 years in the current study was 15.4 ng TEQ kg^{-1} lipid. A separate study, carried out in 1992/3, investigated the levels of PCDDs and PCDFs in a group of 28 non-occupationally exposed people, aged from 20 to 60 years, in the Wellington (New Zealand) region (Hannah et al., 1994). The mean PCDD and PCDF level from that study was 13.9 ng TEQ kg^{-1} lipid, comparable to 12.8 ng TEQ kg^{-1} lipid, the mean concentration determined in the current study. This suggests relatively little exposure reduction over the preceding 5 years.

The suggestion of a reduction in exposures to organochlorines over time is supported by comparison of two New Zealand breast milk studies conducted a decade apart (Bates et al., 2002). One finding of these studies was that concentrations of most compounds had decreased by about two-thirds over the period between the studies. The samples for the second breast milk study were collected during the period October 1998 to May 1999, which is similar to the sampling dates for the current serum study (December 1996 to November 1997). Appendix A shows a comparison of corresponding levels found in the two studies. There is good agreement between the results of the studies.

It is also important to put the results found in the New Zealand serum study into international context. We propose to separately publish a detailed comparison of our results with other data from non-exposed populations around the world.

In conclusion, this paper presents the first ever nationally representative study of organochlorines in an adult population. The data provide a baseline for investigation of trends and for international comparisons. The study also provides a model of how similar studies might be carried out in New Zealand in the future, and in other countries.

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Appendix A. Comparison of results of human milk and serum studies

A recent New Zealand study of organochlorine concentrations in human milk collected milk samples from a total of 54 first-time mothers from two urban and two rural centers (Bates et al., 2002). The average age of the mothers, across all samples, was 26.8 years. As this mean age is at the lower end of the 25–34 age group used in this serum study, and because contaminant concentrations have been shown to increase markedly with age, the mean serum concentrations across the 15–24 and 25–34 age groups were considered to be the most equivalent for purposes of comparison. Comparable data from the two studies are shown in Table 6.

Table 6
Comparative concentrations of organochlorines in serum and breast milk from New Zealanders

Organochlorine	Serum study[a]	Breast milk study[b]
Mean concentration, ng TEQ kg^{-1} lipid weight basis		
PCDD/F TEQ (inc 1/2 LODs)	8.6	6.3
PCDD/F TEQ (exc LODs)	8.5	6.2
PCB TEQ (inc 1/2 LODs)	5.9	4.2
PCB TEQ (exc 1/2 LODs)	2.0	3.1
Mean concentration, μg kg^{-1} lipid weight basis		
Sum of PCBs (inc 1/2 LODs)[c]	164	46.1
Sum of PCBs (exc LODs)[c]	30.2	45.3
β-HCH	12.6	16.3
Dieldrin	11.4	15.4
pp′-DDE	661	626

Abbreviations: inc 1/2 LODs, half the limits of detection were included in the sum of PCBs and the TEQ calculation; exc LOD, limits of detection were excluded from the sum of PCBs and the TEQ calculation.

[a] Weighted mean across all regions, for the 15–24 and 25–34 age groups.

[b] Concentration across all four study centers ($n = 53$) (Bates et al., 2002).

[c] Sum of 42 congeners for the serum study and sum of 34 congeners for the breast milk study.

With the exception of the results for the sum of PCB congeners including half LOD values, there is good agreement overall between the concentrations of organochlorines determined in the two studies. The exception, in the serum study, is markedly elevated by the inclusion of estimates for non-detected congeners.

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