Concentrations of persistent organic pollutants in the milk of New Zealand women

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SUMMARY

Background. This survey is the third of three consecutive breast milk surveys, conducted in 1988, 1998 and 2008, aiming to measure individual breast milk levels of persistent organic pollutants (POPs) in New Zealand first time mothers aged 20 to 30. The survey was designed to provide time trend data for dioxins/furans, polychlorinated biphenyls (PCBs) and organochlorine pesticides (OCPs) and their metabolites, and provide baseline data for brominated flame retardants (BFRs). The study followed the guidelines of the fourth World Health Organization (WHO) Coordinated Survey of human milk for persistent organic pollutants.

Methods. Four study areas were included in order to have representation of urban areas as well as rural areas of both the New Zealand North and South Island. A total of 39 women aged 20-30 years each provided approximately 200 ml of hand expressed breast milk and completed a questionnaire: 17 women from Wellington, 10 from the Wairarapa, 9 from Christchurch and 3 from North Canterbury. The individual breast milk samples were analysed for 7 dioxins, 10 furans, 45 PCBs, 23 OCPs and metabolites, 36 brominated diphenyl ethers (BDEs) and 4 additional BFRs, including hexabrominated biphenyl (BB153), by high-resolution gas chromatography/high-resolution mass spectrometry (HRGC/HRMS). Levels were expressed in picogram per gram milk lipid (pg/g lipid).

Results. Comparison of the 2008 levels with the previous surveys indicated that the mean dioxin and furan levels have further declined by 40% over the 10 year period between 1998 and 2008 to a mean toxic equivalence (TEQ) of 3.54 pg/g lipid. Over this period, the levels of dioxin-like PCBs have declined by 54% to a mean PCB TEQ of 1.29 pg/g lipid. For all the OCPs and metabolites for which time trend data could be provided (*alpha*-HCH (hexachlorocyclohexane), *beta*-HCH, *gamma*-HCH, hexachlorobenzene, dieldrin, heptachlor-epoxide, *gamma*-chlordane, *o,p'*-DDT, *p,p'*-DDT, *p,p'*-DDE), the levels of the 2008 survey were below the levels of the 1998 survey, with the percentage decline ranging between 34% and 90%. The total TEQ for both the dioxins/furans as well as the dioxin-like PCBs, were higher ($p_{t\text{-}test}<0.05$) in the rural areas compared to the urban areas. This was also the case for dieldrin and *p,p,*-DDE. In addition, age was associated with the measured levels of dioxins/furans, PCBs and OCPs, with higher age being associated with higher levels. Other population characteristics such as residency in the North or South Island, (ex-)smoking and body mass index (BMI) were not associated with the levels of dioxins/furans, PCBs or OCPs. Of the 36 BDEs determined, the highest levels were measured for BDE47 (2542 pg/g), followed by BDE153 (717 pg/g), BDE100 (536 pg/g) and BDE99 (532 pg/g). BDE levels were not associated with age or North/South Island residency, but for some BDEs the levels were higher for the urban areas compared to the rural areas (BDE184, BDE196, BDE197, BDE201, BDE203 and BDE207), and for other BDEs the levels were higher in women with a lower body mass index (BMI) compared to those with a BMI above 25 (BDE28&33, BDE49, BDE66).

Conclusions. Over the past decade the background levels of the three classes of POPs (dioxins/furans, PCBs, and OCPs) in breast-feeding women aged 20-30 years have continued to substantially decline. It is likely that there have been similar declines in the New Zealand population. This survey reconfirms that the New Zealand levels of these three classes of POPs in breast milk are low by international standards. The baseline data for BFRs show that the BDEs that are most abundantly present in the New Zealand breast milk samples are similar to those reported for other countries, and that the levels are comparable to or higher than those measured in Europe, while being substantially lower than those reported for the United States and Australia. Subsequent surveys need to determine how levels of BFRs and other emerging POPs change over time in the New Zealand population.

1. INTRODUCTION

Persistent Organic Pollutants (POPs) are defined as organic chemicals that possess the following combination of characteristics:

- they are environmentally persistent (remain intact for many years);
- they become widely distributed throughout the environment as a result of natural processes involving soil, water and, most notably, air;
- they bio-accumulate (accumulate in the fatty tissue of living organisms including humans, and are found at higher concentrations at higher levels in the food chain);
- they are toxic to both humans and wildlife.

Examples of POPs include organochlorine pesticides such as DDT; industrial chemicals such as polychlorinated biphenyls (PCBs); and unintended by-products such as polychlorinated dibenzo-p-dioxins (PCDDs or dioxins) and polychlorinated dibenzofurans (PCDFs or furans). Several brominated and fluorinated compounds are now also considered POPs. Background information on POPs is included in **Appendix 1**.

Due to human activities, POPs have been released into the environment for many years, particularly since the 1950s. As a result, POPs are now widely distributed over large regions, including those where POPs have never been used. This extensive contamination of the environment and living organisms includes many foodstuffs and has resulted in the sustained exposure of many species, including humans, for periods of time that span generations, resulting in both acute and chronic toxic effects.

Specific effects of POPs can include cancer, damage to the central and peripheral nervous systems, reproductive disorders, and disruption of the immune system. Some POPs are also considered to be endocrine disrupters, which, by altering the hormonal system, can damage the reproductive and immune systems of exposed individuals as well as their offspring and have developmental effects.

Given their ubiquitous distribution, it has been recognized that no one governing body acting alone can protect its citizens or its environment from POPs. In response, a global treaty to protect human health and the environment from POPs (the Stockholm Convention) was put into place. The Stockholm Convention on Persistent Organic Pollutants was adopted in 2001 and entered into force in 2004, requiring parties to take measures to eliminate or reduce the release of POPs into the environment. New Zealand signed the Convention in 2001 and ratified it in 2004.

Article 11 of the Stockholm Convention (Research, development and monitoring), specifies that the ratifying parties shall, within their capabilities, encourage and/or undertake appropriate research on the presence, levels and trends in humans and the environment of POPs. Article 16 of the convention requires an effectiveness evaluation of the convention four years after its ratification.

The present study addresses New Zealand's obligations under the Stockholm Convention by aiming to estimate the levels of POPs in the New Zealand population, and to study trends over time, and thereby to evaluate whether the measures taken under the Stockholm Convention are effective in reducing the release of POPs into the environment. Baseline data for most POPs are available from two previous New Zealand breast milk surveys conducted 10 [1,2] and 20[3] years prior to the current survey (the 1988 and 1998 surveys). These studies showed that the levels of persistent organochlorine contaminants (dioxins, furans and organochlorine pesticides) in the milk of New Zealand women declined by about 70% over the ten year period from 1988 to 1998, and that overall the levels of organochlorine contaminants in New Zealanders were low relative to exposures in most other countries where similar studies had been done[3].

The present study follows on from the previous two breast milk surveys, obtaining data on current levels of POPs in human breast milk in New Zealand women aged 20-30. In addition to providing time trend data on the POPs included in the previous two breast milk surveys, this study also provides baseline data for new POPs, including several brominated compounds. The study also follows the guidelines of the fourth WHO-Coordinated Survey of human milk for persistent organic pollutants, specifying a pooled sample to be analysed by a reference laboratory so that it can contribute to a global monitoring programme. The study was commissioned by the Ministry of Health and conducted by the Centre for Public Health Research at Massey University.

1.1. Study aims

The study had the following aims:

- to obtain data on current levels of POPs in human breast milk in New Zealand;
- to compare these levels with previous levels and detect trends in POPs exposure;
- to measure for the first time polybrominated diphenylethers (PBDE) in breast milk in New Zealand;
- to use the collected New Zealand breast milk samples for inclusion in the fourth round of the WHO-coordinated study of human milk for POPs, thus providing an international comparison for levels of POPs;
- to study the determinants of elevated levels of POPs in breast milk in New Zealand;
- to provide recommendations for prioritising POPs for remedial action in New Zealand.

2. METHODS

2.1. Ethical approval

Ethical approval for the study was obtained from the Multi-Region Ethics Committee, reference MEC/06/10/119.

2.2. Study areas

The design of the study was largely modelled on that of the previous two surveys and the guidelines of the fourth WHO-Coordinated Survey of human milk for persistent organic pollutants, to optimise comparability over time and across countries. Four study areas were included in order to have representation of urban areas as well as rural areas of both the North and the South Islands. The 1988 and 1998 surveys included the following four regions: Auckland (urban North Island), Northland (rural North Island), Christchurch (urban South Island) and North Canterbury (rural South Island). The 2008 survey also included a rural and urban area in both the South and the North Island, but in this survey the urban North Island region was Wellington and the rural North Island region was the Wairarapa (figure 1). It was aimed to include 12-15 women from each of the four areas, yielding a total number of 50 women. Living in a rural area was defined as living more than 3 km from any town with a population of more than 2,500 people. Living in an urban area was defined as living within the boundaries of a city (Wellington or Christchurch). Potential participants were required to have lived in the study area for at least five years.

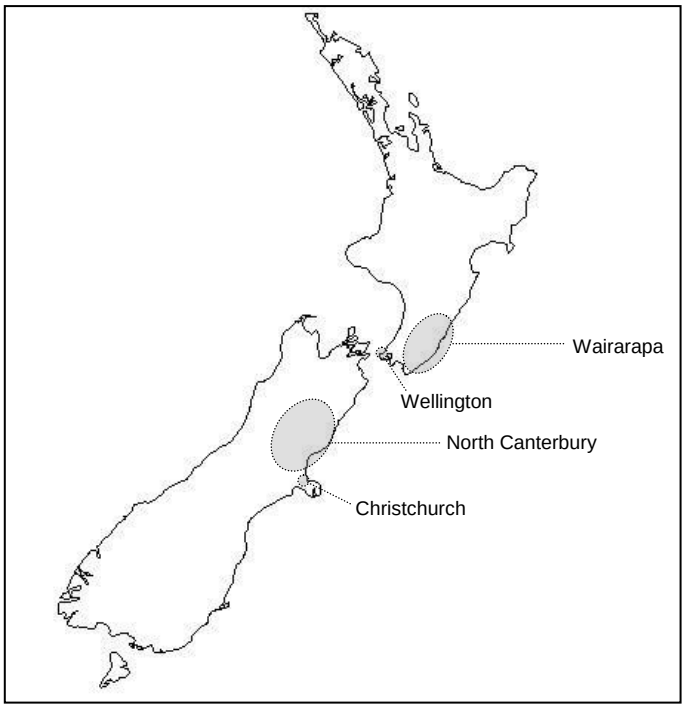


Figure 1. Map of New Zealand with the four study areas of the survey.

2.3. Field work

2.3.1. Recruitment of study participants

Research nurses at the Centre for Public Health Research established contacts with local midwives, lead maternity carers (LMCs), medical doctors (MDs) and breast feeding consultants, and asked for their assistance in recruiting pregnant women who fitted the inclusion criteria of the study. The main responsibility of the midwives/LMCs/MDs/breast feeding consultants was to identify eligible women and make first contact. The participants' recruitment instructions for LMCs are included in **appendix 2**. The actual recruitment, confirmation of eligibility and completion of the field work was done by the research nurse.

The role of the research nurses thus included: (i) liaising with the midwives/LMCs/MDs/breast feeding consultants; (ii) maintaining contact with the potential participating mothers; (iii) visiting the mothers to explain the breast milk collection procedure, and administering the study questionnaire; (iv) and visiting the mothers a second time to collect the breast milk samples (and collecting house dust samples[1]).

The initial approach to potential participating mothers was made in person by midwives/MDs/LMCs selected from the regions mentioned above, or during breast feeding classes. Women in their third trimester and who complied with the WHO selection criteria (table 1) were invited to participate in the study. These criteria are the same as those used for the 1988 and 1998 surveys and were modelled on the inclusion criteria of the fourth WHO-Coordinated Survey of human milk for persistent organic pollutants[4].

Table 1. Selection criteria for participating mothers.

(1) Primiparous, with singleton pregnancy;
(2) Aged 20-30 years;
(3) Apparently healthy mother and child, and 'normal' pregnancy (e.g. gestation >37 weeks, birth weight >2500 grams);
(4) Mother planning to exclusively breastfeed her child;
(5) Residential history: apart from holidays the mother had to have lived in the same area for the last five years;
(6) Freezer at home for storage of milk sample;

Potential participants showing interest in the study were provided with the information sheet of the study (included in **appendix 3**), which included a reply form with pre-paid envelope which the potential participant could use to obtain more information and indicate her interest.

[1] House dust samples were collected to assess the association between the levels of brominated flame retardants in house dust and breast milk, as part of an add-on study. Results are not yet available and will be reported separately in due course.

Once the research nurse received the reply form, she allocated a unique participant ID number to the potential participant. The research nurse then contacted the woman by phone to provide more information about the study. At this point, the research nurse also administered a short screening questionnaire to check eligibility according to the WHO criteria. The screening questionnaire is included in **appendix 4**. If all criteria were met, the research nurse further explained all aspects of the study, and informed the potential participant that she would be contacted again after the baby was born.

The research nurse remained in contact with the LMC of the pregnant women, in order to be informed about the delivery date, and any complications of the pregnancy that may have prevented participation in the study. After the baby's birth, the research nurse verified with the LMC whether (i) the child was fed exclusively on mother's milk; (ii) mother and child were healthy; (iii) the pregnancy was more than 37 weeks; and (iv) birth weight was more than 2,500 gram. If these criteria were met the mother was eligible to enrol in the study.

About 10 days after birth, the eligible mothers were sent a letter from the research nurse indicating that the research nurse would phone her in the next few days to arrange a time to come and visit. The research nurse then contacted the mother two to three weeks after the birth of their child, by phone. If the mother was still agreeable to participate, an appointment was made to visit the mother at home. Because the breast milk collection had to take place during the second month after birth, the first visit was scheduled to take place some time 3 to 6 weeks after birth.

2.3.2. First home visit (3-6 weeks postnatal)

The research nurse visited the mother at home at a time convenient for the mother. After discussing the study, the mother was asked to sign a consent form (**appendix 5**).

The research nurse provided the women with a breast milk sample collection kit, including one 250 ml glass storage container (RED top), to be used for the storage of the samples and eight smaller glass collection containers (BLUE tops) to be used to express the breast milk directly into (more details are included in **appendix 6**). All containers were provided cleaned by the laboratory. The lids were Teflon lined.

The use of the collection containers and the collection procedure was explained and the mother received an instruction sheet (included in **appendix 6**). During the first visit the research nurse also administered the study questionnaire (included in **appendix 7**). The first visit took on average 1 to 1 ½ hour.

The breast milk sample was collected by the mother during the second month after birth. The mother contacted the research nurse when a total of up to 250 ml of breast milk was collected and/or all of the collection containers had been

used. The milk was kept in the freezer at the home of the study participant until picked up by the research nurse. If the mother had any problems with or questions regarding the breast milk sampling, the mother could contact the research nurse by phone.

2.3.3. Second home visit (after milk sampling is completed)

The participating women collected a maximum of 250 ml of breast milk. After breast milk collection was completed, a second home visit was organised during which the research nurse picked up the milk samples. Samples were transported using ice packs and an insulated carrier to keep samples frozen. At the second home visit the research nurse also took dust samples from the house (using a vacuum cleaner) which will be used to determine levels of brominated flame retardants (results not included here).

The breast milk samples were stored at the Centre for Public Health Research in a -20°C freezer and sent in batches of 10-15 samples to AsureQuality. The laboratory set aside 10 ml of each sample for the WHO pooled sample (results not included here). Any remaining samples will be returned to the Centre for Public health Research for storage.

2.4. The POPs included in the study

The individual breast milk samples collected during the 2008 survey were analysed for the following analytes. Details are included in **Appendix 1 and Appendix 8**.

Dioxins
2,3,7,8-Tetrachlorodibenzo-*p*-Dioxin (2,3,7,8-TCDD);
1,2,3,7,8-Pentachlorodibenzo-*p*-dioxin (1,2,3,7,8-PeCDD);
1,2,3,4,7,8-Hexachlorodibenzo-*p*-dioxin (1,2,3,4,7,8-HxCDD);
1,2,3,6,7,8- Hexachlorodibenzo-*p*-dioxin (1,2,3,6,7,8-HxCDD);
1,2,3,7,8,9- Hexachlorodibenzo-*p*-dioxin (1,2,3,7,8,9-HxCDD);
1,2,3,4,6,7,8-Heptachlorodibenzo-*p*-dioxin (1,2,3,4,6,7,8-HpCDD);
Octachlorodibenzo-*p*-dioxin (OCDD).

Furans
2,3,7,8-Tetrachlorodibenzofuran (2,3,7,8-TCDF);
1,2,3,7,8-Pentachlorodibenzofuran (1,2,3,7,8-PeCDF);
2,3,4,7,8-Pentachlorodibenzofuran (2,3,4,7,8-PeCDF);
1,2,3,4,7,8-Hexachlorodibenzofuran (1,2,3,4,7,8-HxCDF);
1,2,3,6,7,8- Hexachlorodibenzofuran (1,2,3,6,7,8-HxCDF);
2,3,4,6,7,8- Hexachlorodibenzofuran (2,3,4,6,7,8-HxCDF);
1,2,3,7,8,9- Hexachlorodibenzofuran (1,2,3,7,8,9-HxCDF);
1,2,3,4,6,7,8-Heptachlorodibenzofuran (1,2,3,4,6,7,8-HpCDF);
1,2,3,4,7,8,9-Heptachlorodibenzofuran HpCDF (1,2,3,4,7,8,9-HpCDF);
Octachlorodibenzofuran (OCDF).

Polychlorinated biphenyls (PCBs)
PCB4&10; PCB15; PCB19; PCB28; PCB37; PCB44; PCB49; PCB52; PCB54; PCB70; PCB74; PCB77; PCB81; PCB99; PCB101; PCB104; PCB105; PCB110; PCB114; PCB118; PCB123; PCB126; PCB138; PCB153; PCB155; PCB156; PCB157; PCB167; PCB169; PCB170; PCB180; PCB183; PCB187; PCB188; PCB189; PCB194; PCB200; PCB202; PCB205; PCB206; PCB208; PCB209.

Organochlorine pesticides (OCPs) and metabolites
alpha-HCH (hexachlorocyclohexane, also known as BHC: benzene hexachloride); *beta*-HCH; *gamma*-HCH; *delta*-HCH (not a POP under the Stockholm Convention 2009); HCB (hexachlorobenzene); Aldrin; Dieldrin; heptachlor; heptachlor exo-epoxide (also known as heptachlor-epoxide); *alpha*-chlordane; *gamma*-chlordane; *p,p'*-DDT (dichlorodiphenyltrichloro ethane); *o,p'*-DDT; *p,p'*-DDD (dichlorodiphenyldichloroethane); *o,p'*-DDD; *p,p'*-DDE (dichlorodiphenyldichloroethylene); *o,p'*-DDE; Pentachlorobenzene; Endosulfan sulphate (not a POP under the Stockholm Convention 2009); Endrin; Endrin ketone; Endrin aldehyde; Mirex.

Polybrominated diphenyl ethers (PBDEs)
BDE47; BDE49; BDE66; BDE71; BDE77; BDE85; BDE99; BDE100; BDE119&120; BDE126; BDE138&166; BDE139; BDE140; BDE153; BDE154; BDE156&169; BDE171; BDE180; BDE183&175; BDE184; BDE191.

Additional polybrominated diphenyl ethers (PBDEs) not considered a POP under the Stockholm Convention 2009
BDE7; BDE15; BDE17; BDE28&33; BDE30; BDE196; BDE197; BDE201; BDE203; BDE204; BDE205; BDE206; BDE207; BDE208; BDE209.

Additional brominated flame retardants
BB153 (Hexabrominated biphenyl); DBDPE (Decabromodiphenylethane; not a POP under the Stockholm Convention 2009); HBB (Hexabromobenzene; not a POP under the Stockholm Convention 2009); PBEB (Pentabromoethylbenzene; not a POP under the Stockholm Convention 2009).

The lipid content of each of the samples was determined, and the levels of all POPs were expressed as picogram per gram lipid (or nanogram per gram lipid). **Appendix 8** lists the POPs that were included in this survey and whether they were also included in the 1988 and 1998 surveys.

2.5. Laboratory analyses

All laboratory analyses were performed at AsureQuality in Lower Hutt New Zealand, using the following methods:

Dioxins and furans

High-Resolution Gas Chromatography/High-Resolution Mass Spectrometry (HRGC/HRMS). Method 1613 Tetra- through Octa-Chlorinated Dioxins and Furans by Isotope Dilution HRGC/HRMS U.S. Environmental Protection Agency, October 1994.

Polychlorinated biphenyls (PCBs)
High-Resolution Gas Chromatography/High-Resolution Mass Spectrometry (HRGC/HRMS). Method 1668B Chlorinated Biphenyl Congeners in Water, Soil, Sediment, Biosolids, and Tissue by HRGC/HRMS U.S. Environmental Protection Agency, November 2008.

Organochlorine pesticides
High-Resolution Gas Chromatography/High-Resolution Mass Spectrometry (HRGC/HRMS). Method 1699: Pesticides in Water, Soil, Sediment, Biosolids, and Tissue by HRGC/HRMS U.S. Environmental Protection Agency, December 2007.

Brominated flame retardants
High-Resolution Gas Chromatography/High-Resolution Mass Spectrometry (HRGC/HRMS). Method 1614 Brominated Diphenyl Ethers in Water, Soil, Sediment and Tissue by HRGC/HRMS, U.S. Environmental Protection Agency, August 2007.

For the purpose of quality assurance, prior to extraction each milk sample was fortified with internal standards containing isotopically-labelled ^{13}C analogs of most target analytes. Cleanup was performed using a combination of gel permeation chromatography and solid-phase chromatography to remove interfering components. Immediately prior to injection, a labelled injection standard was added to each extract and an aliquot of the extract was injected into the gas chromatograph (GC). The analytes were separated by the GC and detected by a high-resolution (≥10,000) mass spectrometer. Two exact m/z's (mass-to-charge ratios) for each analyte were monitored throughout a pre-determined retention time window. An individual analyte is identified by comparing the GC retention time and ion abundance ratio of two exact m/z's with the corresponding retention time of an authentic standard and the theoretical or acquired ion-abundance ratio of the two exact m/z's.

Quantitative analysis was performed in one of two ways using selected ion current profile (SICP) areas:

- For analytes for which a labelled analog is available, the GC/HRMS was multi-point calibrated and the concentration was determined using the isotope dilution technique.
- Analytes for which a labelled analog was not available, the GC/HRMS was multi-point calibrated and the concentration was determined using the internal standard technique. The labelled compounds were used as internal standards, affording recovery correction for all pesticides.

The quality of the analysis was assured through reproducible calibration and testing of the extraction, cleanup, and HRGC/HRMS systems.

The quality control acceptance criteria for internal standard recovery were 25-150%. Each batch of 10-15 samples included the analysis of one laboratory blank, which provided information on laboratory background levels of the target analytes. A spiked QA sample was also included with each batch of samples to confirm compliant method performance.

2.6. Data analysis

All questionnaire data were entered in a database and all the laboratory results were provided electronically by the analytical laboratory. Both were then imported into a single SAS database.

The levels of the individual dioxins (PCDDs), furans (PCDFs), and dioxin like PCBs were expressed as toxic equivalence values (TEQs) as follows: TEQ = ∑ (TEF x concentration). This was done using the most recently determined TEFs from 2005 as well as using the 1998 TEFs to ensure comparability with the previous survey. The WHO 1998 and 2005 TEFs are listed in table 2[5].

For each of the POPs, the mean, median, standard error, minimum and maximum value were determined for all study participants combined and by study area (Wellington; Wairarapa; Christchurch; North Canterbury). For those levels reported as being below the limit of detection (<LOD), half the LOD was assumed to be the level present in the sample. In order to provide insight into the potential impact on the mean levels of this assumption, the mean was also calculated under the assumption that the levels of reported as being <LOD were 0 (zero).

Table 2. The WHO 1998 and WHO 2005 TEF Values (from [5]).

Dioxins and furans	WHO 1998 TEF	WHO 2005 TEF
2,3,7,8-TCDD	1	1
1,2,3,7,8-PeCDD	1	1
1,2,3,4,7,8-HxCDD	0.1	0.1
1,2,3,6,7,8-HxCDD	0.1	0.1
1,2,3,7,8,9-HxCDD	0.1	0.1
1,2,3,4,6,7,8-HpCDD	0.01	0.01
OCDD	0.0001	0.0003
2,3,7,8-TCDF	0.1	0.1
1,2,3,7,8-PeCDF	0.05	0.03
2,3,4,7,8-PeCDF	0.5	0.3
1,2,3,4,7,8-HxCDF	0.1	0.1
1,2,3,6,7,8-HxCDF	0.1	0.1
2,3,4,6,7,8-HxCDF	0.1	0.1
1,2,3,7,8,9-HxCDF	0.1	0.1
1,2,3,4,6,7,8-HpCDF	0.01	0.01
1,2,3,4,7,8,9-HpCDF	0.01	0.01
OCDF	0.0001	0.0003

Polychlorinated biphenyls (PCBs)	WHO 1998 TEF	WHO 2005 TEF
PCB77	0.0001	0.0001
PCB81	0.0001	0.0003
PCB105	0.0001	0.00003
PCB114	0.0005	0.00003
PCB118	0.0001	0.00003
PCB123	0.0001	0.00003
PCB126	0.1	0.1
PCB156	0.0005	0.00003
PCB157	0.0005	0.00003
PCB167	0.00001	0.00003
PCB169	0.01	0.03
PCB189	0.0001	0.00003

The associations between population characteristics and measured levels of POPs were assessed through methods comparable to what was done in the 1998 survey. This involved making the following comparisons: urban vs. rural residency; North vs South Island residency; age 20-25 vs. 26-30; never vs ever smoking; 20-25 vs. 26-33 BMI; less than 6 kg vs. more than 6 kg weight gain during pregnancy defined as the difference in weight before pregnancy and at time of interview; 1.4%-3.5% vs. 3.6%-7.4% lipid content; and boys vs. girls. Standard t-tests were used to assess whether POPs levels were significantly different between the different groups.

3. RESULTS

3.1. The 2008 survey

3.1.1. The study population

Participant recruitment was planned to take one year (April 2007 to April 2008), but due to the small number of first time mothers that fitted the inclusion criteria of the study, the recruitment period was extended to 3 years (April 2007 to April 2010), during which a total of 39 women could be included in the study. The inclusion criteria that proved most difficult to meet were the age range of 20-30 years, as many first time mothers were either older or younger, and the set minimum duration of residency in the study area, which proved to be most difficult to meet for the rural areas. The difficulties in finding eligible women also proved to negatively affect the motivation of the midwives and LMCs to continue with the study, which was another factor slowing down recruitment. The study aimed to include 15 participants from each of the four study regions (Wellington, Wairarapa, Christchurch, North Canterbury), but due to the difficulties with meeting the inclusion criteria this could only be achieved for the Wellington region. As a result the two rural areas, particularly the North Canterbury area, are under-represented in the sample.

Of the total of 49 women initially recruited for the study, 10 could not participate either due to a limited milk supply (n=6), health problems of the baby (n=2), difficulty expressing milk manually (n=1), or personal problems (n=1).

The women expressed on average 30 ml per day (ranging between 2 ml and 100 ml) and collected the total sample over an average period of 14 days (between 3 and 38 days). The average total volume expressed by the 37 participants was 205 ml (ranging between 19 and 250). Two participants (1 from Wellington and 1 from the Wairarapa) did not provide sufficient breast milk to enable testing for all the compounds under study. These two samples were therefore only analysed for dioxins/furans/PCBs and not for OCPs and BFRs. The total number of samples available for the dioxin/furan/PCB analyses was therefore 39, and the total number of samples available for the OCPs and BFRs analyses was 37. When excluding the two small volume samples, the average sample volume was 215 ml for the 37 samples that could be tested for all analytes (ranging between 100 ml and 250 ml).

Table 3 describes the characteristics of the 39 participating mothers. The average age of the participants was 26.9 years. The majority of women were of European ethnicity. Maternal BMI was 26.1 kg/m^2 on average (ranging between 20.8 and 32.4), and the mean milk lipid concentration was 3.85% (ranging between 1.4% and 7.4%). The baby's weight was 3.5 kg on average (ranging between 2.4 kg and 4.3 kg). Of the 39 women 8 (20.5%) had ever smoked, of which almost all were ex-smokers at the time of interview. All but two had a diet including a variety of all foods. For both urban areas all women received their water from town supply, while in the rural area of the Wairarapa

roof collection was the most common source of water supply. None of the participants reported occupational exposure to sources of POPs.

Table 3. Characteristics of the study population of the 2008 survey (standard error of the mean between brackets), by study area.

	Wellington	Wairarapa	Christchurch	North Canterbury	all
Number of participants	17	10	9	3	39
North/South	North	North	South	South	
urban/rural	urban	rural	urban	rural	
maternal ethnicity (European:other)	15:2	10:0	9:0	3:0	37:2
mean maternal age (years)	27.7 (0.690)	26.9 (1.016)	25.0 (0.958)	28.8 (0.709)	26.9 (0.483)
mean maternal height (m)	1.65 (0.014)	1.69 (0.021)	1.69 (0.030)	1.70 (0.047)	1.67 (0.011)
mean maternal weight (kg)	71.7 (2.27)	73.6 (2.79)	71.6 (5.20)	79.7 (6.94)	72.8 (1.68)
mean maternal BMI (kg/m^2)	26.4 (0.83)	25.7 (0.865)	25.3 (1.37)	27.4 (0.94)	26.1 (0.52)
mean milk lipid concentration (%)	4.0 (0.35)	3.3 (0.30)	4.6 (0.39)	2.7 (0.62)	3.85 (0.21)
mean baby's birth weight (kg)	3.47 (0.095)	3.50 (0.129)	3.48 (0.180)	3.48 (0.228)	3.48 (0.067)
baby's sex (boy:girl)	8:9	6:4	5:4	0:3	19:20
smoking					
never (%)	82.4	80.0	77.8	66.7	79.5
ex (%)	11.8	20.0	22.2	33.3	18.0
current (%)	5.9	0.0	0.0	0.0	2.5
diet					
eat variety of all foods (%)	88.2	100.0	100.0	100.0	94.9
other (%)	11.8	0.0	0.0	0.0	5.1
water supply					
town supply (%)	100.0	0.0	100.0	66.7	71.8
roof collection (%)	0.0	70.0	0.0	0.0	18.0
private well or bore (%)	0.0	20.0	0.0	33.3	7.7
other (%)	0.0	10.0	0.0	0.0	2.6

3.1.2. Dioxins and furans

Table 4 lists the breast milk levels expressed in pg/g lipid for 7 dioxin congeners and 10 furan congeners. For two dioxin congeners (1,2,3,4,7,8-HxCDD and 1,2,3,7,8,9-HxCDD) the levels were below the detection limit for a majority of the samples. For only one furan congener levels were detected in all samples (2,3,4,7,8-PeCDF).

The total TEQ for the dioxins and furans combined (using 2005 WHO TEFs) added up to 3.54 pg TEQ/ g lipid, and ranged between a minimum of 1.39 pg TEQ/ g lipid and a maximum of 10.83 pg TEQ/ g lipid, showing an eight-fold difference between the lowest and highest TEQ.

Table 4. Dioxins and furans (PCDD/DFs) in breast milk (pg/g lipid) of 39 participants in the 2008 survey.

	WHO 2005 TEF	N	**Mean**	Median	SE	Minimum	Maximum	%<LOD
2,3,7,8-TCDD	1	39	**0.75**	0.70	0.05	0.29	1.72	3%
1,2,3,7,8-PeCDD	1	39	**1.57**	1.38	0.21	0.46	8.96	0%
1,2,3,4,7,8-HxCDD	0.1	39	**0.49**	0.41	0.04	0.24	1.12	79%
1,2,3,6,7,8-HxCDD	0.1	39	**2.87**	2.83	0.19	0.88	5.20	3%
1,2,3,7,8,9-HxCDD	0.1	39	**0.64**	0.54	0.05	0.18	1.39	62%
1,2,3,4,6,7,8-HpCDD	0.01	39	**5.44**	5.27	0.38	1.34	11.90	3%
OCDD	0.0003	39	**30.53**	24.90	2.56	12.40	97.40	0%
2,3,7,8-TCDF	0.1	39	**0.15**	0.11	0.02	0.06	0.47	97%
1,2,3,7,8-PeCDF	0.03	39	**0.19**	0.11	0.04	0.05	1.35	90%
2,3,4,7,8-PeCDF	0.3	39	**1.73**	1.71	0.10	0.81	3.54	0%
1,2,3,4,7,8-HxCDF	0.1	39	**0.64**	0.57	0.06	0.20	1.82	31%
1,2,3,6,7,8-HxCDF	0.1	39	**0.58**	0.50	0.05	0.15	1.93	36%
2,3,4,6,7,8-HxCDF	0.1	39	**0.38**	0.32	0.04	0.13	1.49	64%
1,2,3,7,8,9-HxCDF	0.1	39	**0.36**	0.32	0.03	0.09	0.85	100%
1,2,3,4,6,7,8-HpCDF	0.01	39	**1.72**	0.91	0.59	0.16	23.00	23%
1,2,3,4,7,8,9-HpCDF	0.01	39	**0.43**	0.39	0.03	0.16	1.02	100%
OCDF	0.0003	39	**0.54**	0.37	0.07	0.12	2.06	100%
1998 TEQ (including half LOD)		39	**3.89**	3.59	0.26	1.55	11.08	
2005 TEQ (including half LOD)		39	**3.54**	3.23	0.25	1.39	10.83	
1998 TEQ (excluding half LOD)		39	**3.71**	3.44	0.26	1.30	10.52	
2005 TEQ (excluding half LOD)		39	**3.37**	3.10	0.25	1.14	10.28	

Table 5 lists the mean levels of dioxins and furans by study area. The mean TEQ for the dioxins and furans (2005 TEFs, using a value of half the detection limit for samples with undetectable levels) was around 3 pg/g in the two urban areas (3.31 Wellington; 2.78 Christchurch) and between 4 and 5 pg/g in the two rural areas (4.42 Wairarapa; 4.26 North Canterbury). The difference in TEQ between the urban and rural samples was statistically significant (p=0.0168), with the mean TEQ in rural samples being 40% above the mean TEQ of the urban areas. There were no statistically significant differences between the TEQ levels of the North and South Island.

Table 5. Dioxins and furans (PCDD/DFs) in breast milk (pg/g lipid) of 39 participants in the 2008 survey, by study area.

	Wellington		**Wairarapa**		**Christchurch**		**North Canterbury**	
	Mean	SE	**Mean**	SE	**Mean**	SE	**Mean**	SE
2,3,7,8-TCDD	**0.71**	0.08	**0.87**	0.07	**0.60**	0.08	**1.05**	0.16
1,2,3,7,8-PeCDD	**1.40**	0.09	**2.25**	0.76	**1.13**	0.18	**1.63**	0.08
1,2,3,4,7,8-HxCDD	**0.49**	0.06	**0.52**	0.07	**0.42**	0.07	**0.68**	0.13
1,2,3,6,7,8-HxCDD	**2.87**	0.25	**2.62**	0.35	**2.58**	0.42	**4.54**	0.41
1,2,3,7,8,9-HxCDD	**0.62**	0.08	**0.74**	0.11	**0.52**	0.08	**0.83**	0.26
1,2,3,4,6,7,8-HpCDD	**5.23**	0.42	**4.73**	0.65	**5.63**	1.01	**8.40**	1.80
OCDD	**30.52**	3.15	**21.05**	1.26	**38.29**	8.04	**38.89**	9.83
2,3,7,8-TCDF	**0.13**	0.02	**0.20**	0.05	**0.10**	0.01	**0.17**	0.05
1,2,3,7,8-PeCDF	**0.16**	0.03	**0.32**	0.12	**0.10**	0.01	**0.16**	0.04
2,3,4,7,8-PeCDF	**1.80**	0.13	**1.79**	0.22	**1.48**	0.23	**1.90**	0.26
1,2,3,4,7,8-HxCDF	**0.57**	0.06	**0.75**	0.14	**0.49**	0.08	**1.12**	0.13
1,2,3,6,7,8-HxCDF	**0.44**	0.04	**0.79**	0.14	**0.52**	0.11	**0.89**	0.16
2,3,4,6,7,8-HxCDF	**0.36**	0.04	**0.52**	0.12	**0.26**	0.04	**0.36**	0.09
1,2,3,7,8,9-HxCDF	**0.34**	0.05	**0.41**	0.05	**0.32**	0.04	**0.41**	0.10
1,2,3,4,6,7,8-HpCDF	**0.93**	0.15	**4.00**	2.18	**0.90**	0.19	**1.08**	0.40
1,2,3,4,7,8,9-HpCDF	**0.40**	0.05	**0.47**	0.06	**0.40**	0.03	**0.57**	0.06
OCDF	**0.59**	0.15	**0.63**	0.11	**0.34**	0.03	**0.58**	0.13
1998 TEQ (including half LOD)	**3.66**	0.23	**4.78**	0.80	**3.07**	0.42	**4.63**	0.22
2005 TEQ (including half LOD)	**3.31**	0.21	**4.42**	0.79	**2.78**	0.38	**4.26**	0.20
1998 TEQ (excluding half LOD)	**3.48**	0.24	**4.60**	0.77	**2.91**	0.44	**4.47**	0.16
2005 TEQ (excluding half LOD)	**3.12**	0.22	**4.24**	0.76	**2.62**	0.40	**4.10**	0.14

3.1.3. Polychlorinated biphenyls (PCBs)

Table 6 lists the results for PCBs. For 24 PCBs the levels of all the samples were within detection limits. The average toxic equivalency of the dioxin-like PCBs was 1.29 pg TEQ / g lipid and ranged between 0.58 and 3.68.

Table 6. PCBs in breast milk (pg/g lipid) of 39 participants in the 2008 survey.

Variable	WHO 2005 TEF	N	**Mean**	Median	SE	Minimum	Maximum	%<LOD
PCB4&10		39	**3.09**	2.66	0.28	0.84	9.15	92%
PCB15		39	**9.26**	6.70	1.77	1.57	67.00	23%
PCB19		36	**1.97**	1.80	0.16	0.50	4.72	81%
PCB28		39	**1369.56**	1160.00	171.05	374.00	6420.00	0%
PCB37		27	**11.43**	6.33	2.76	1.62	67.50	30%
PCB44		39	**33.80**	27.20	3.71	13.20	108.00	0%
PCB49		39	**25.19**	20.60	2.95	9.45	86.60	0%
PCB52		39	**96.92**	77.70	13.86	35.60	534.00	0%
PCB54		39	**2.24**	1.43	0.38	0.59	11.75	100%
PCB70		39	**27.76**	21.10	3.44	9.52	134.00	0%
PCB74		39	**1495.67**	1230.00	204.37	504.00	7890.00	0%
PCB77	0.0001	39	**2.39**	1.70	0.34	0.84	12.75	97%
PCB81	0.0003	39	**2.22**	1.68	0.32	0.83	12.20	100%
PCB99		39	**788.33**	655.00	63.89	247.00	2040.00	0%
PCB101		39	**114.95**	85.40	23.53	36.10	973.00	0%
PCB104		39	**0.81**	0.54	0.14	0.22	5.10	100%
PCB105	0.00003	39	**348.72**	302.00	35.12	136.00	1340.00	0%
PCB110		39	**44.40**	43.30	3.43	13.30	146.00	0%
PCB114	0.00003	39	**69.29**	62.30	5.99	20.10	216.00	0%
PCB118	0.00003	39	**1279.67**	1130.00	107.01	563.00	3880.00	0%
PCB123	0.00003	39	**21.90**	16.70	2.86	5.35	98.20	5%
PCB126	0.1	39	**10.09**	7.53	1.00	3.01	32.60	10%
PCB138		39	**4824.87**	4390.00	353.80	1180.00	10500.00	0%
PCB153		39	**5551.28**	4810.00	411.18	1330.00	13500.00	0%
PCB155		39	**1.66**	1.24	0.19	0.34	5.29	36%
PCB156	0.00003	39	**684.49**	658.00	44.37	138.00	1290.00	0%
PCB157	0.00003	39	**135.21**	125.00	9.54	31.90	305.00	0%
PCB167	0.00003	39	**180.64**	167.00	13.74	56.50	477.00	0%
PCB169	0.03	39	**6.66**	6.43	0.57	1.45	22.50	21%
PCB170		39	**1365.18**	1280.00	93.22	277.00	3210.00	0%
PCB180		39	**2912.07**	2810.00	221.72	92.70	7430.00	0%
PCB183		39	**308.94**	264.00	27.69	4.18	766.00	3%
PCB187		39	**773.13**	690.00	63.18	124.00	2000.00	0%
PCB188		39	**2.52**	1.75	0.40	0.53	14.30	95%
PCB189	0.00003	39	**52.85**	51.50	3.54	12.10	126.00	0%
PCB194		39	**287.68**	263.00	27.07	4.16	860.00	3%
PCB200		39	**1.52**	1.07	0.24	0.52	9.20	100%
PCB202		39	**53.95**	46.20	5.85	6.41	182.00	0%
PCB205		39	**7.70**	7.50	0.79	1.80	27.60	41%
PCB206		39	**26.63**	19.50	4.08	2.94	121.00	0%
PCB208		39	**12.19**	8.15	2.27	1.76	76.90	5%
PCB209		39	**29.96**	24.00	2.92	13.80	87.80	0%
1998 TEQ (incl half LOD)		39	**1.69**	1.37	0.13	0.73	4.40	
2005 TEQ (incl half LOD)		39	**1.29**	1.02	0.11	0.58	3.68	
1998 TEQ (excl half LOD)		39	**1.62**	1.36	0.14	0.36	4.40	
2005 TEQ (excl half LOD)		39	**1.19**	1.01	0.12	0.05	3.68	

Note: PCB 19 and PCB 37 could not be quantified in all samples (these analytes did not always meet the required method performance specs).

The highest levels were measured for PCB153, PCB138 and PCB180. There was approximately a 10-fold difference between the lowest and highest individual levels of PCBs 153 and 138, while there was an 80-fold difference between the highest and lowest level observed for PCB180.

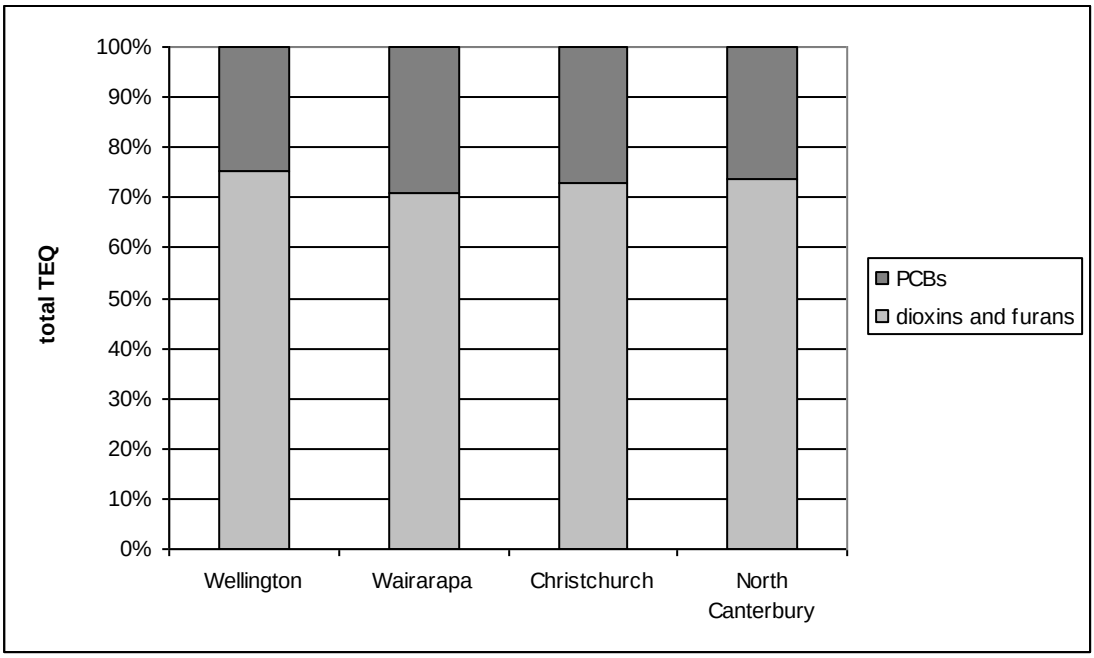
Table 7. PCBs in breast milk (pg/g lipid) of 39 participants in the 2008 survey, by study area.

	Wellington		Wairarapa		Christchurch		North Canterbury	
	Mean	SE	**Mean**	SE	**Mean**	SE	**Mean**	SE
PCB4&10	**3.47**	0.36	**3.41**	0.80	**2.05**	0.25	**3.04**	1.08
PCB15	**8.31**	1.57	**15.82**	6.05	**4.85**	0.65	**6.01**	0.76
PCB19	**2.39**	0.29	**1.90**	0.23	**1.35**	0.24	**1.80**	0.23
PCB28	**1879.88**	339.21	**918.90**	126.62	**905.78**	160.67	**1371.33**	247.48
PCB37	**9.72**	3.49	**19.39**	6.64	**5.54**	0.85	**5.48**	0.70
PCB44	**41.38**	6.26	**32.31**	8.87	**23.93**	2.60	**25.40**	4.25
PCB49	**30.68**	4.93	**24.41**	7.36	**17.77**	1.85	**18.93**	2.66
PCB52	**129.94**	27.23	**81.19**	22.23	**65.24**	6.84	**57.27**	7.52
PCB54	**2.68**	0.58	**2.31**	1.06	**1.58**	0.31	**1.50**	0.35
PCB70	**26.80**	3.89	**36.47**	11.45	**20.84**	2.10	**24.97**	2.38
PCB74	**2053.12**	419.33	**1224.80**	171.74	**826.67**	117.11	**1246.67**	182.79
PCB77	**2.35**	0.36	**3.41**	1.15	**1.54**	0.12	**1.74**	0.11
PCB81	**2.25**	0.33	**2.94**	1.10	**1.50**	0.11	**1.72**	0.09
PCB99	**895.65**	108.92	**695.70**	84.80	**638.67**	136.02	**938.00**	221.00
PCB101	**150.40**	52.06	**97.92**	20.52	**75.38**	8.47	**89.57**	6.22
PCB104	**0.70**	0.13	**1.21**	0.47	**0.64**	0.08	**0.66**	0.18
PCB105	**375.24**	66.30	**334.60**	44.34	**266.56**	45.69	**492.00**	167.53
PCB110	**42.67**	2.43	**52.34**	11.88	**36.87**	4.71	**50.37**	5.86
PCB114	**79.28**	10.71	**64.81**	9.25	**52.38**	11.25	**78.37**	8.94
PCB118	**1313.53**	185.71	**1344.50**	179.61	**981.33**	150.45	**1766.67**	496.97
PCB123	**20.49**	3.42	**31.16**	8.78	**13.54**	1.54	**24.07**	6.99
PCB126	**8.04**	1.09	**15.00**	2.81	**7.92**	0.88	**11.87**	2.63
PCB138	**4917.65**	521.84	**4886.00**	761.16	**4328.89**	874.70	**5583.33**	118.37
PCB153	**5690.59**	558.20	**5562.00**	855.83	**5002.22**	1146.37	**6373.33**	179.47
PCB155	**1.46**	0.20	**2.21**	0.55	**1.67**	0.34	**0.98**	0.48
PCB156	**701.53**	60.39	**714.10**	97.27	**593.22**	112.45	**763.00**	97.60
PCB157	**143.11**	15.11	**132.18**	16.15	**117.49**	24.03	**153.67**	21.40
PCB167	**178.19**	23.15	**198.91**	28.71	**153.17**	24.97	**216.00**	14.01
PCB169	**6.37**	0.50	**7.88**	1.72	**5.58**	1.25	**7.46**	0.69
PCB170	**1421.59**	114.69	**1286.40**	188.11	**1304.56**	282.89	**1490.00**	162.89
PCB180	**3124.12**	252.66	**2812.00**	395.33	**2522.30**	724.51	**3213.33**	433.33
PCB183	**336.94**	41.84	**268.42**	57.86	**289.81**	65.35	**342.67**	50.56
PCB187	**823.29**	88.42	**739.40**	120.07	**675.44**	176.47	**894.33**	103.98
PCB188	**1.69**	0.35	**4.23**	1.30	**2.00**	0.32	**3.16**	0.54
PCB189	**54.68**	4.07	**50.74**	6.56	**50.47**	11.55	**56.67**	8.28
PCB194	**351.24**	31.76	**220.12**	45.39	**258.02**	82.38	**241.67**	38.46
PCB200	**1.31**	0.23	**2.19**	0.79	**1.05**	0.19	**1.84**	0.60
PCB202	**67.26**	8.42	**39.96**	5.50	**45.89**	17.76	**49.30**	8.68
PCB205	**8.21**	0.96	**7.05**	1.23	**7.36**	2.69	**7.94**	1.21
PCB206	**34.57**	6.98	**18.28**	3.01	**22.13**	10.86	**23.03**	3.49
PCB208	**16.25**	4.21	**7.17**	0.98	**11.19**	5.48	**8.93**	1.00
PCB209	**33.35**	4.69	**26.29**	6.11	**30.96**	6.01	**20.03**	3.69
PCB1998TEQ (incl half LOD)	**1.51**	0.17	**2.21**	0.36	**1.36**	0.16	**2.00**	0.25
PCB2005TEQ (incl half LOD)	**1.08**	0.12	**1.82**	0.30	**1.03**	0.11	**1.52**	0.27
PCB1998TEQ (excl half LOD)	**1.46**	0.18	**2.00**	0.42	**1.36**	0.16	**2.00**	0.25
PCB2005TEQ (excl half LOD)	**1.02**	0.14	**1.55**	0.38	**1.01**	0.12	**1.52**	0.27

The mean PCB TEQ was around 1 pg/g in the two urban areas (1.08 Wellington; 1.03 Christchurch) and around 1.5 pg/g in the two rural areas (1.82 Wairarapa; 1.52 North Canterbury). The difference in PCB TEQ between the urban and rural samples was statistically significant (p=0.0021), with the mean TEQ in rural samples being 65% above the mean TEQ of the urban areas. There were no statistically significant differences between the North and South Island in PCB TEQ.

3.1.4. Total TEQ

For all 4 study areas about three quarters of the total TEQ was attributable to dioxins/furans and one quarter to PCBs (see figure 2). The total TEQ (including dioxins, furans and dioxin-like PCBs) was 4.39 pg/g for Wellington, 6.24 pg/g for the Wairarapa, 3.81 pg/g for Christchurch and 5.78 pg/g for North Canterbury.



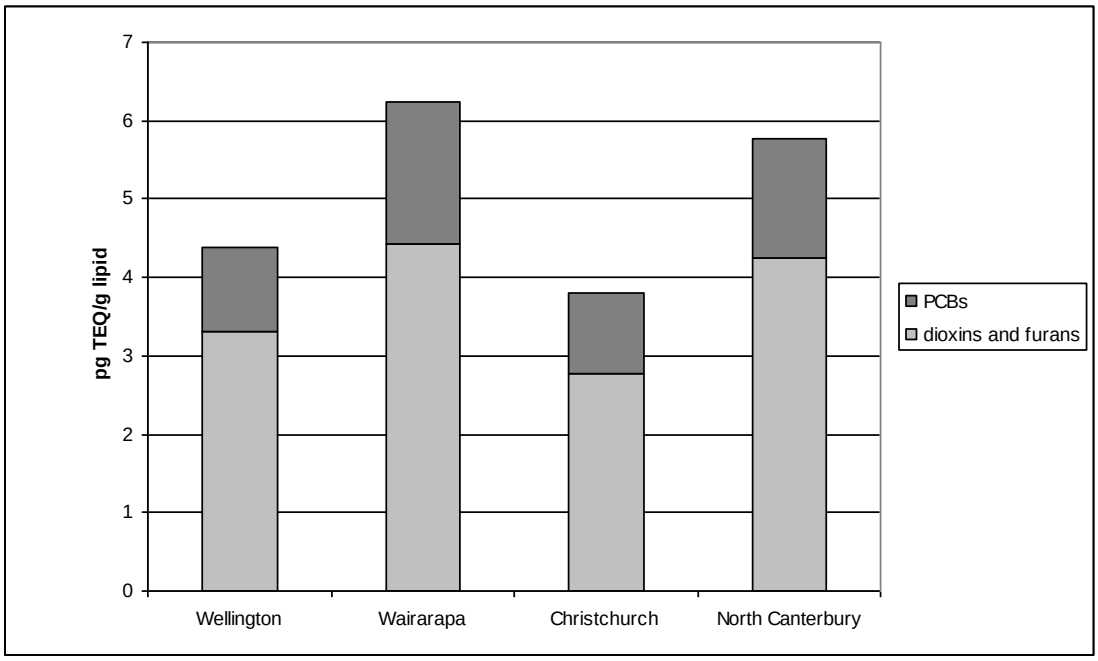


Figure 2. The total TEQ for dioxins/furans and PCB, by study area.

3.1.5. Organochlorine pesticides (OCPs)

Table 8 lists the results for the organochlorine pesticides. The volume of two samples was not sufficient to be included in these analyses and thus a total of 37 samples were analysed for OCPs. The following compounds were detected in all 37 samples: *beta*-HCH, HCB, dieldrin, heptachlor-epoxide, *p,p'*-DDT, *o,p'*-DDT, *p,p'*-DDD, *p,p'*-DDE, mirex.

Table 8. Organochlorine pesticides and metabolites in breast milk (ng/g lipid) of 37 participants in the 2008 survey.

	N	**Mean**	Median	SE	Minimum	Maximum	%<LOD
alpha-HCH	37	**0.049**	0.049	0.009	0.017	0.346	5%
beta-HCH	37	**8.432**	8.432	3.463	1.160	132.000	0%
gamma-HCH	37	**0.218**	0.218	0.040	0.057	1.310	27%
delta-HCH	37	**0.072**	0.072	0.014	0.019	0.295	100%
HCB	37	**6.763**	6.763	0.621	2.940	25.700	0%
aldrin	37	**0.009**	0.009	0.001	0.003	0.033	97%
dieldrin	37	**10.138**	10.138	1.197	1.140	28.300	0%
heptachlor	37	**0.013**	0.013	0.001	0.006	0.030	11%
heptachlor-epoxide	37	**0.465**	0.465	0.037	0.133	1.200	0%
alpha-chlordane	37	**0.077**	0.077	0.007	0.021	0.213	100%
gamma-chlordane	37	**0.060**	0.060	0.005	0.014	0.149	8%
p,p'-DDT	37	**5.023**	5.023	0.407	1.400	13.000	0%
o,p'-DDT	37	**0.556**	0.556	0.093	0.084	2.980	0%
p,p'-DDD	37	**0.123**	0.123	0.008	0.035	0.243	0%
o,p'-DDD	37	**0.024**	0.024	0.002	0.005	0.071	24%
p,p'-DDE	37	**378.946**	378.946	41.877	113.000	995.000	0%
o,p'-DDE	37	**0.164**	0.164	0.046	0.031	1.750	5%
pentachlorobenzene	37	**0.558**	0.558	0.360	0.060	13.500	78%
endosulfansulfate	37	**0.064**	0.064	0.009	0.011	0.307	65%
endrin	37	**0.018**	0.018	0.002	0.005	0.053	78%
endrin ketone	37	**0.062**	0.062	0.009	0.016	0.351	100%
endrin aldehyde	37	**0.093**	0.093	0.008	0.028	0.241	100%
mirex	37	**0.231**	0.231	0.022	0.076	0.773	0%

There was an approximately 10-fold difference between the lowest and highest individual levels of most OCPs, while for *beta*-HCH the difference was 114-fold, due to 1 outlier (from the Wellington region) for which a level of 132 ng/g was measured. If this outlier is excluded a 10-fold difference between the lowest and highest level was also observed for *beta*-HCH.

Table 9 lists the mean levels of the OCPs by study area.

Table 9. Organochlorine pesticides and metabolites in breast milk (ng/g lipid) of 37 participants in the 2008 survey, by study area.

	Wellington		Wairarapa		Christchurch		North Canterbury	
	Mean	SE	**Mean**	SE	**Mean**	SE	**Mean**	SE
alpha-HCH	**0.08**	0.02	**0.03**	0.00	**0.02**	0.00	**0.03**	0.00
beta-HCH	**13.81**	7.91	**4.22**	1.02	**3.74**	0.74	**6.49**	1.64
gamma-HCH	**0.33**	0.08	**0.18**	0.04	**0.10**	0.01	**0.09**	0.01
delta-HCH	**0.12**	0.03	**0.05**	0.01	**0.03**	0.00	**0.03**	0.00
HCB	**7.68**	1.35	**6.12**	0.60	**5.76**	0.51	**6.82**	0.93
aldrin	**0.01**	0.00	**0.01**	0.00	**0.01**	0.00	**0.01**	0.00
dieldrin	**7.65**	1.67	**13.99**	2.27	**6.80**	0.68	**21.87**	3.98
heptachlor	**0.01**	0.00	**0.01**	0.00	**0.01**	0.00	**0.02**	0.00
heptachlor-epoxide	**0.46**	0.06	**0.47**	0.08	**0.45**	0.08	**0.47**	0.08
alpha-chlordane	**0.08**	0.01	**0.06**	0.01	**0.08**	0.01	**0.07**	0.02
gamma-chlordane	**0.05**	0.00	**0.06**	0.01	**0.06**	0.01	**0.10**	0.03
p,p'-DDT	**4.68**	0.71	**5.56**	0.85	**4.50**	0.65	**6.78**	0.62
o,p'-DDT	**0.66**	0.18	**0.62**	0.17	**0.27**	0.02	**0.68**	0.19
p,p'-DDD	**0.11**	0.01	**0.12**	0.01	**0.12**	0.01	**0.18**	0.02
o,p'-DDD	**0.02**	0.00	**0.03**	0.01	**0.02**	0.00	**0.03**	0.01
p,p'-DDE	**295.19**	59.44	**458.22**	94.46	**324.56**	40.54	**751.00**	139.34
o,p'-DDE	**0.26**	0.10	**0.11**	0.02	**0.06**	0.01	**0.10**	0.03
pentachlorobenzene	**1.09**	0.83	**0.15**	0.03	**0.17**	0.06	**0.10**	0.01
endosulfansulfate	**0.07**	0.01	**0.05**	0.01	**0.05**	0.01	**0.13**	0.09
endrin	**0.01**	0.00	**0.03**	0.00	**0.01**	0.00	**0.03**	0.01
endrin ketone	**0.09**	0.02	**0.05**	0.01	**0.03**	0.00	**0.04**	0.00
endrin aldehyde	**0.11**	0.01	**0.11**	0.02	**0.05**	0.01	**0.06**	0.01
mirex	**0.24**	0.03	**0.20**	0.03	**0.18**	0.02	**0.40**	0.18

When comparing the OCP levels between the urban and the rural areas, there was a general pattern of higher levels in the rural areas compared to the urban areas. This difference was only statistically significant for dieldrin (7.34 ng/g urban; 15.96 ng/g rural; p=0.0003) and *p,p'*-DDE (305.76 ng/g urban; 531.42 ng/g rural; p=0.0096). There were no statistically significant differences in OCP levels between the urban areas of the North and South Island.

3.1.6. Brominated flame retardants

Table 10 lists the measured levels of the BDEs as well as BB153 (Hexabrominated biphenyl), DBDPE (Decabromodipenylethane; not a POP under the Stockholm Convention 2009), HBB (Hexabromobenzene; not a POP under the Stockholm Convention 2009), and PBEB (Pentabromoethylbenzene; not a POP under the Stockholm Convention 2009). The volume of two samples was not sufficient to be included in these analyses and thus a total of 37 samples were analysed for brominated flame retardants.

Table 10. Brominated flame retardants in breast milk (pg/g lipid) of 37 participants in the 2008 survey.

	N	**Mean**	Median	SE	Minimum	Maximum	%<LOD
BDE7	37	**2.59**	2.09	0.30	0.38	10.50	100%
BDE15	37	**131.56**	77.30	18.11	19.10	458.00	0%
BDE17	37	**2.40**	2.13	0.21	0.57	5.63	24%
BDE28&33	37	**218.65**	172.00	26.08	48.80	751.00	0%
BDE30	37	**1.16**	0.94	0.11	0.18	2.98	100%
BDE47	37	**2541.97**	2030.00	257.46	317.00	7710.00	0%
BDE49	37	**25.56**	21.40	2.73	6.51	96.40	0%
BDE66	37	**30.87**	25.30	3.73	5.39	103.00	3%
BDE71	37	**1.55**	1.47	0.11	0.43	3.19	100%
BDE77	37	**2.92**	0.98	0.74	0.46	20.00	76%
BDE85	37	**48.59**	37.70	6.23	2.21	168.00	5%
BDE99	37	**532.11**	486.00	51.85	66.20	1290.00	0%
BDE100	37	**535.75**	419.00	64.21	70.80	1820.00	0%
BDE119&120	37	**2.27**	2.41	0.14	0.87	4.53	100%
BDE126	37	**2.54**	2.17	0.24	0.81	7.50	100%
BDE138&166	37	**6.70**	5.25	0.68	1.35	17.85	100%
BDE139	37	**19.13**	16.40	2.08	4.56	59.40	11%
BDE140	37	**7.69**	6.92	0.86	0.87	25.00	32%
BDE153	37	**717.14**	488.00	102.07	142.00	3820.00	0%
BDE154	37	**37.00**	33.00	3.61	6.54	101.00	0%
BDE156&169	37	**2.80**	2.44	0.29	0.71	9.30	100%
BDE171	37	**6.37**	4.18	1.01	1.10	35.00	51%
BDE180	37	**4.80**	3.57	0.72	1.20	24.10	76%
BDE183&175	37	**65.81**	40.70	14.15	8.01	512.00	0%
BDE184	37	**4.26**	4.11	0.49	0.55	13.40	32%
BDE191	37	**3.71**	3.55	0.45	0.61	18.10	100%
BDE196	37	**15.55**	12.50	1.81	2.53	43.90	8%
BDE197	37	**125.15**	102.00	12.25	35.30	320.00	0%
BDE201	37	**23.42**	20.40	2.25	5.94	60.30	0%
BDE203	37	**18.11**	15.80	1.75	3.00	45.00	16%
BDE204	37	**3.78**	3.35	0.33	1.58	9.75	100%
BDE205	37	**6.20**	5.60	0.46	2.15	13.65	100%
BDE206	37	**28.60**	15.90	6.24	2.86	195.00	14%
BDE207	37	**85.19**	64.50	10.98	25.90	337.00	0%
BDE208	37	**24.29**	15.40	4.64	1.75	133.00	14%
BDE209	37	**353.75**	183.00	91.89	65.30	3140.00	3%
DBDPE	36	**113.53**	90.00	13.03	15.85	325.50	97%
BB153	37	**148.06**	85.10	29.89	18.00	956.00	0%
HBB	37	**21.72**	19.80	2.01	6.38	73.70	0%
PBEB	37	**1.02**	1.04	0.08	0.39	3.10	46%

Note: for 1 sample DBDPE could not be determined, but it was determined (typically as not detected) in all other samples.

The highest levels were measured for BDE47, followed by BDE153, BDE99, BDE100 and BDE209. For most BDEs the range of individual breast milk levels was wide, with, on average, the highest levels being 25 times higher than the lowest measured levels.

Table 11 lists the mean levels of the BFRs by study region. The levels tended to be higher in the urban South Island compared to the urban North Island but these differences were not statistically significant. Statistically significantly

higher levels were observed for the urban areas compared to the rural areas for BDE184, BDE196, BDE197, BDE201, BDE203 and BDE207.

Table 11. Brominated flame retardants in breast milk (pg/g lipid) of 37 participants in the 2008 survey, by study area.

	Wellington		Wairarapa		Christchurch		North Canterbury	
	Mean	SE	**Mean**	SE	**Mean**	SE	**Mean**	SE
BDE7	**2.52**	0.33	**1.77**	0.26	**3.26**	0.99	**3.34**	0.95
BDE15	**116.03**	28.43	**145.76**	41.86	**141.12**	37.82	**143.13**	34.14
BDE17	**2.21**	0.30	**2.35**	0.51	**2.96**	0.35	**1.85**	0.70
BDE28&33	**186.07**	29.22	**211.31**	55.02	**273.48**	77.58	**250.00**	20.66
BDE30	**1.31**	0.20	**1.18**	0.26	**0.88**	0.13	**1.18**	0.26
BDE47	**2528.38**	310.37	**2139.89**	583.01	**2951.11**	682.13	**2593.33**	839.47
BDE49	**22.58**	2.31	**26.68**	6.27	**29.83**	8.71	**25.23**	2.86
BDE66	**26.67**	3.95	**33.90**	10.23	**34.63**	9.52	**32.97**	5.41
BDE71	**1.38**	0.11	**1.28**	0.17	**1.83**	0.28	**2.38**	0.55
BDE77	**1.61**	0.57	**5.23**	2.65	**3.41**	0.99	**1.47**	0.36
BDE85	**47.77**	7.41	**43.89**	15.46	**56.88**	15.65	**42.20**	21.33
BDE99	**561.00**	76.17	**402.69**	116.43	**607.00**	109.00	**541.67**	154.16
BDE100	**461.94**	58.56	**509.53**	146.82	**674.11**	180.50	**593.00**	287.47
BDE119&120	**2.20**	0.17	**1.80**	0.25	**2.61**	0.33	**3.04**	0.61
BDE126	**2.73**	0.39	**2.38**	0.58	**2.42**	0.47	**2.38**	0.39
BDE138&166	**5.98**	0.72	**5.70**	1.32	**7.98**	1.89	**9.68**	2.98
BDE139	**16.49**	2.41	**19.86**	4.71	**23.26**	5.81	**18.57**	3.89
BDE140	**7.11**	1.05	**7.42**	1.98	**9.74**	2.22	**5.43**	1.67
BDE153	**654.38**	89.88	**433.44**	60.86	**827.33**	117.50	**1572.33**	1124.53
BDE154	**36.99**	5.24	**29.74**	7.02	**46.93**	8.79	**29.07**	4.59
BDE156&169	**2.61**	0.34	**2.36**	0.60	**2.56**	0.37	**5.86**	1.80
BDE171	**5.70**	0.93	**4.13**	1.14	**10.75**	3.34	**3.48**	0.58
BDE180	**4.00**	0.51	**3.39**	0.93	**7.88**	2.49	**4.05**	0.67
BDE183&175	**45.14**	7.71	**43.24**	13.14	**129.16**	51.30	**53.63**	20.15
BDE184	**4.22**	0.60	**2.69**	0.50	**6.72**	1.32	**1.85**	0.31
BDE191	**3.05**	0.31	**2.83**	0.49	**5.53**	1.61	**4.41**	0.73
BDE196	**15.67**	2.61	**10.22**	1.75	**23.24**	4.55	**7.83**	2.39
BDE197	**126.31**	16.78	**86.42**	10.34	**179.90**	32.07	**70.90**	14.58
BDE201	**25.27**	3.21	**15.03**	1.64	**31.96**	5.75	**13.16**	2.85
BDE203	**20.05**	2.59	**13.16**	1.72	**24.06**	3.92	**4.79**	0.55
BDE204	**3.31**	0.41	**3.53**	0.79	**4.11**	0.55	**6.01**	1.58
BDE205	**5.32**	0.66	**6.10**	0.88	**7.41**	1.06	**7.53**	1.54
BDE206	**29.90**	8.97	**16.26**	3.75	**43.20**	19.50	**14.92**	6.05
BDE207	**86.35**	13.89	**46.61**	4.68	**129.56**	33.02	**61.60**	15.33
BDE208	**27.14**	7.63	**12.46**	2.81	**35.83**	12.28	**9.96**	4.28
BDE209	**339.88**	108.29	**231.47**	51.78	**559.67**	325.17	**176.80**	46.72
DBDPE	**108.59**	17.88	**77.00**	14.65	**142.66**	35.05	**186.25**	35.25
BB153	**202.21**	56.62	**123.36**	53.43	**71.59**	21.59	**162.73**	112.66
HBB	**18.12**	2.36	**30.14**	6.24	**20.21**	1.95	**20.20**	3.82
PBEB	**0.99**	0.09	**1.26**	0.25	**0.83**	0.11	**1.01**	0.03

3.2. Comparison with the 1988 and 1998 surveys

Table 12 lists the population characteristics of the three consecutive breast milk surveys conducted in 1988, 1998 and 2008.

Table 12. Comparison of participants and their babies in the 3 studies (standard error between brackets).

	1988		1998		2008	
	rural	urban	rural	urban	rural	urban
Number of participants	18	20	18	35	13	26
Ethnicity (European:other)			14:4	26:9	13:0	24:2
Mean maternal age (years)	25.7 (0.72)	26.7 (0.65)	26.6 (0.57)	26.9 (0.49)	27.4 (0.81)	26.7 (0.61)
Mean maternal height (m)	1.60 (0.01)	1.62 (0.02)	1.66 (0.01)	1.68 (0.01)	1.69 (0.02)	1.66 (0.01)
Mean maternal weight (kg)	59.6 (1.6)	63.5 (2.7)	70.5 (2.9)	74.4 (2.7)	75.1 (2.7)	71.7 (2.1)
Mean maternal BMI (kg/m^2)	22.9 (0.64)	24.2 (0.97)	25.5 (0.97)	26.4 (0.92)	26.1 (0.70)	26.1 (0.70)
Mean babies' birth weight (kg)	3.39 (0.085)	3.43 (0.090)	3.37 (0.059)	3.52 (0.084)	3.49 (0.11)	3.48 (0.09)

The mean age of the participants was similar over all 3 surveys. The height of the participants of the 2008 survey was greater than the 1988 survey but similar to the height of the participants of the 1998 survey. A similar pattern was observed for the weight of the participants, being very similar between the two most recent surveys while being lower in the first survey of 1988.

3.2.1. Lipid content of the breast milk

The previous two surveys of 1988 and 1998 had observed a difference in milk lipid concentration between urban and rural women, with the lipid concentration being higher for urban women.

Table 13. Lipid concentrations (%) in the breast milk of urban and rural women from the 3 studies.

	1988			1998			2008		
Study area:	n	**mean**	SE	n	**mean**	SE	n	**Mean**	SE
Auckland	11	**4.04**	0.30	20	**4.38**	0.26			
Northland	10	**2.74**	0.24	16	**3.53**	0.27			
Christchurch	9	**4.11**	0.54	15	**4.56**	0.34	9	**4.60**	0.39
North Canterbury	8	**2.76**	0.23	2	**3.62**	0.34	3	**2.70**	0.63
Wellington							17	**3.96**	0.35
Wairarapa							10	**3.34**	0.30
Urban	20	**4.07**	0.28	35	**4.46**	0.21	26	**4.18**	0.27
Rural	18	**2.75**	0.16	18	**3.54**	0.24	13	**3.19**	0.27
Total	**38**	**3.45**	**0.20**	**53**	**4.15**	**0.17**	**39**	**3.85**	**0.21**

The mean lipid concentration in the 39 samples was 3.85%, and as in the previous two surveys, in the 2008 survey the milk lipid levels in urban women was higher than the milk lipid levels in rural women (p=0.0242).

3.2.2. Dioxins and Furans

The comparison of the two previous breast milk studies indicated that over the 10 years between 1988 and 1998, the levels of POPs in New Zealanders (females aged 20-30) had decreased by approximately 70%. This comparison could only be made for the compounds that were analysed and detected in both surveys. These included 12 PCDDs/PCDFs, 6 PCBs and 6 organochlorine pesticides.

For these compounds we compared the 2008 levels with the 1988 and 1998 levels. For an additional 5 PCDDs/PCDFs, 27 PCBs and 5 organochlorine pesticides the 2008 levels could only be compared with the 1998 levels, because these were not included in the 1988 survey.

Table 14 presents the levels of the analytes that were included and detected in two or more of the 3 breast milk surveys, as well as the percentage difference between 1988-1998 and 1998-2008.

Table 14. The mean levels of dioxins and furans as reported for the 3 studies.

		1988		1998		2008		% difference	
	2005 WHO TEF	**mean**	<LOD (%)	**mean**	<LOD (%)	**mean**	<LOD (%)	(1988-1998)	(1998-2008)
PCDDs and PCDFs (pg/g)									
2,3,7,8-TCDD	1	**5.12**	5%	**1.22**	0%	**0.75**	3%	-76.2%	-38.5%
1,2,3,7,8-PeCDD	1	**7.3**	0%	**2.53**	2%	**1.57**	0%	-65.3%	-37.9%
1,2,3,4,7,8-HxCDD	0.1			**1.13**	23%	**0.49**	79%		-56.6%
1,2,3,6,7,8-HxCDD	0.1			**7.29**	0%	**2.87**	3%		-60.6%
1,2,3,7,8,9-HxCDD	0.1	**5.95**	11%	**2.28**	2%	**0.64**	62%	-61.6%	-71.9%
1,2,3,4,6,7,8-HpCDD	0.01	**52**	0%	**13**	19%	**5.44**	3%	-74.9%	-58.2%
OCDD	0.0003	**209**	0%	**67.9**	6%	**30.53**	0%	-67.6%	-55.0%
2,3,7,8-TCDF	0.1	**0.86**	14%	**0.2**	57%	**0.15**	97%	-76.7%	-25.0%
1,2,3,7,8-PeCDF	0.03	**0.25**	81%	**0.19**	79%	**0.19**	90%	-24.9%	0.0%
2,3,4,7,8-PeCDF	0.3	**5.42**	3%	**2.16**	2%	**1.73**	0%	-60.2%	-19.9%
1,2,3,4,7,8-HxCDF	0.1			**1.01**	19%	**0.64**	31%		-36.6%
1,2,3,6,7,8-HxCDF	0.1			**0.95**	19%	**0.58**	36%		-38.9%
2,3,4,6,7,8-HxCDF	0.1			**0.46**	30%	**0.38**	64%		-17.4%
1,2,3,7,8,9-HxCDF	0.1	**0.34**	100%	**0.16**	98%	**0.36**	100%	-54.1%	125.0%
1,2,3,4,6,7,8-HpCDF	0.01	**7.19**	0%	**1.2**	26%	**1.72**	23%	-83.3%	43.3%
1,2,3,4,7,8,9-HpCDF	0.01	**0.33**	100%	**0.2**	98%	**0.43**	100%	-39.1%	115.0%
OCDF	0.0003	**3.17**	97%	**0.68**	79%	**0.54**	100%	-78.4%	-20.6%
Total TEQ 1998*		**16.7**		**5.31**				-68.1%	
Total TEQ 2005 **				**5.92**		**3.54**			-40.2%

* (incl half LOD. only including the 1988 congeners)

** (incl half LOD, including all congeners)

The levels of TCDD in the 2008 survey were 38.5% below those reported for the 1998 survey. The levels of the other dioxin congeners had also decreased

over time, with the percentage difference between 1998 and 2008 ranging between -38% and -72%.

For most furan congeners that were detectable in a majority of the samples, levels decreased between 1998 and 2008. The levels of 1,2,3,7,8,9-HxCDF, 1,2,3,4,6,7,8-HpCDF and 1,2,3,4,7,8,9-HpCDF appeared to have increased compared to the previous survey in 1998, but it should be noted that the percentage below the limit of detection was high for these analytes. For those samples below the limit of detection, half the limit of detection is assumed, thus for these analytes the average levels are largely driven by the limit of detection, which are not the same for the 1998 and 2008 surveys. For example, for 1,2,3,7,8,9-HxCDF the average reported limit of detection was 0.3 pg/g lipid for the 1998 survey while for the 2008 survey this was 0.7 pg/g lipid. The limits of detection for the 2008 survey may be higher because the volume of sample analysed in the 2008 survey was split across more analytes. The resulting differences in detection limit are likely to explain the apparent higher 2008 levels compared to the 1998 levels for 1,2,3,7,8,9-HxCDF, 1,2,3,4,6,7,8-HpCDF and 1,2,3,4,7,8,9-HpCDF.

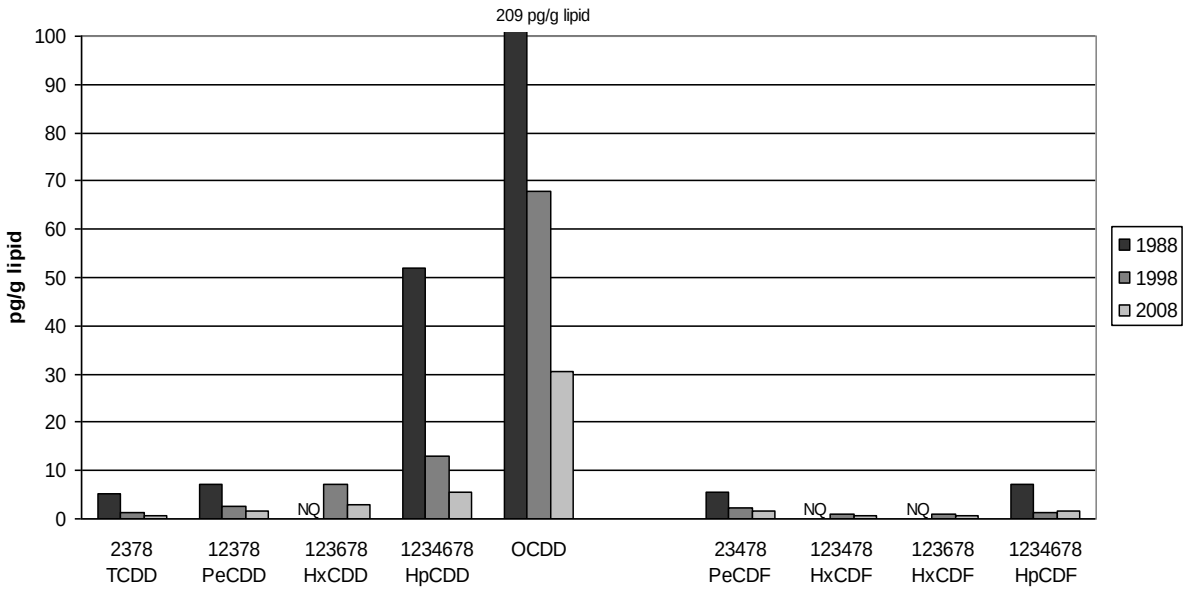


Figure 3. Mean levels of individual dioxins and furans as reported for the 3 studies (only listing those analytes that were detected in the majority of samples).

The total TEQ for dioxins and furans decreased by 40% between 1998 and 2008. The decrease in dioxin and furan levels over time is presented graphically in figure 3. This only includes those congeners that were detected in more than 20% of the samples. It should be noted that the 1988 OCDD level is off the scale of the graph. NQ indicates the congener was not quantified in the 1988 survey.

3.2.3. Polychlorinated biphenyls (PCBs)

For almost all the PCB congeners that were included in both the 1998 and 2008 survey a decrease in levels over time was observed (table 15). The percentage decrease ranged between 33% and 63% for those congeners that were detected in all samples of both surveys. Only for PCB209 the 2008 levels were above the 1998 levels, but this comparison is less reliable because PCB209 was only detected in 1 of the 1998 samples and for the other samples half the limit of detection was assumed.

Table 15. The mean levels of PCBs (ng/g lipid) as reported for the 3 studies.

		1988 (n=38)			1998 (n=52)			2008 (n=39)			% difference	
	TEF (2005)	**mean**	SE	<LOD (%)	**mean**	SE	<LOD (%)	**mean**	SE	<LOD (%)	(1988-1998)	(1998-2008)
PCB#44					**0.08**	NR	89%	**0.034**	0.004	0%		-57.8%
PCB#49					**0.05**	NR	90%	**0.025**	0.003	0%		-49.6%
PCB#52					**0.16**	NR	81%	**0.097**	0.014	0%		-39.4%
PCB#70					**0.05**	NR	89%	**0.028**	0.003	0%		-44.5%
PCB#74		**3.91**	0.35	100 %	**2.25**	0.17	0%	**1.496**	0.204	0%	-42.5%	-33.5%
PCB#77	0.0001				**0.01**	NR	94%	**0.002**	0.000	97%		-76.1%
PCB#81	0.0003				**0.01**	NR	94%	**0.002**	0.000	100%		-77.8%
PCB#99					**1.44**	0.09	0%	**0.788**	0.064	0%		-45.3%
PCB#101					**0.2**	NR	67%	**0.115**	0.024	0%		-42.5%
PCB#105	0.00003				**0.78**	0.06	0%	**0.349**	0.035	0%		-55.3%
PCB#110					**0.11**	NR	89%	**0.044**	0.003	0%		-59.6%
PCB#114	0.00003				**0.16**	0.01	0%	**0.069**	0.006	0%		-56.7%
PCB#118	0.00003	**7.17**	0.56	95%	**3.22**	0.24	0%	**1.280**	0.107	0%	-55.1%	-60.3%
PCB#123	0.00003				**0.15**	NR	92%	**0.022**	0.003	5%		-85.4%
PCB#126	0.1				**0.02**	NR	40%	**0.010**	0.001	10%		-49.6%
PCB#138		**27.1**	1.46	0%	**9.64**	0.85	0%	**4.825**	0.354	0%	-64.5%	-49.9%
PCB#153		**43.9**	2.8	0%	**9.76**	0.81	0%	**5.551**	0.411	0%	-77.8%	-43.1%
PCB#156	0.00003				**1.28**	0.12	0%	**0.684**	0.044	0%		-46.5%
PCB#157	0.00003				**0.23**	0.02	0%	**0.135**	0.010	0%		-41.2%
PCB#167	0.00003				**0.46**	0.04	0%	**0.181**	0.014	0%		-60.7%
PCB#169	0.03				**0.02**	NR	87%	**0.007**	0.001	21%		-66.7%
PCB#170		**7.44**	0.57	87%	**3.69**	0.27	0%	**1.365**	0.093	0%	-50.3%	-63.0%
PCB#180		**18.1**	5.26	26%	**5.88**	0.37	0%	**2.912**	0.222	0%	-67.6%	-50.5%
PCB#183					**0.74**	0.05	0%	**0.309**	0.028	3%		-58.3%
PCB#187					**1.84**	0.12	0%	**0.773**	0.063	0%		-58.0%
PCB#189	0.00003				**0.11**	NR	34%	**0.053**	0.004	0%		-52.0%
PCB#194					**0.8**	0.22	0%	**0.288**	0.027	3%		-64.0%
PCB#202					**0.1**	0.01	0%	**0.054**	0.006	0%		-46.1%
PCB#206					**0.05**	NR	60%	**0.027**	0.004	0%		-46.7%
PCB#208					**0.02**	NR	64%	**0.012**	0.002	5%		-39.1%
PCB#209					**0.02**	NR	90%	**0.030**	0.003	0%		49.8%
2005 PCBTEQ incl half LOD					**0.0028**			**0.0013**	0.0001			-53.8%

The decline in PCB levels over time is graphically represented in figure 4, only including those congeners that were detected in all samples of the 1998 and

2008 surveys. NQ indicates that the congener was not quantified in the 1988 survey.

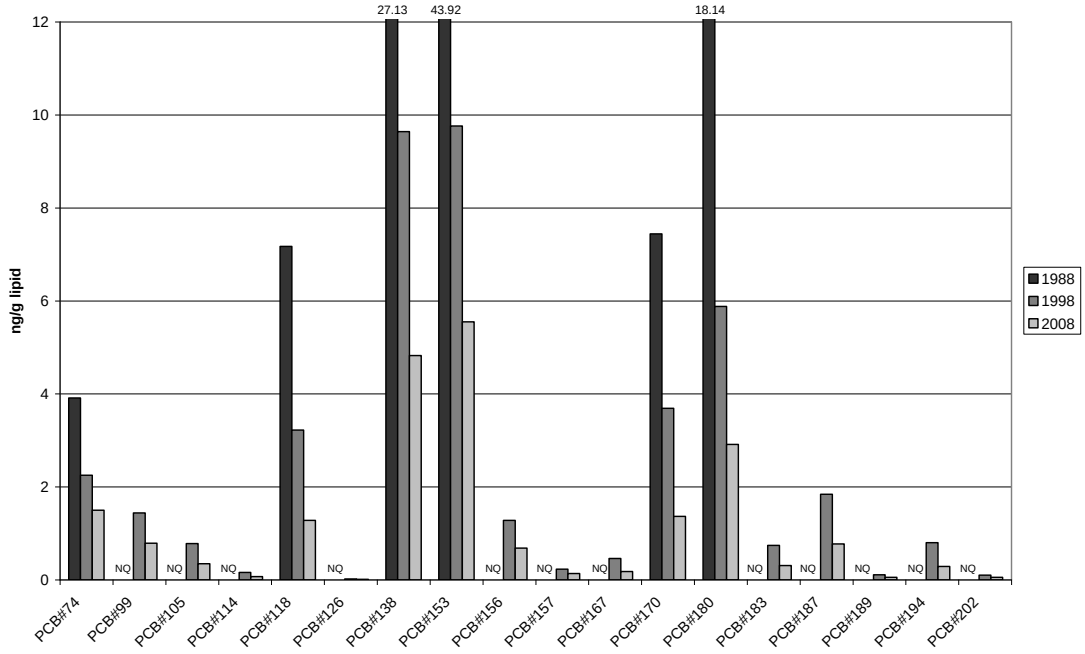


Figure 4. Mean levels of individual PCBs as reported for the 3 studies (only listing those analytes that were detected in all 1998 and 2008 samples).

3.2.4. Total TEQ

The total TEQ of all dioxin-like compounds (including dioxins, furans and dioxin-like PCBs) was calculated for the 1998 and 2008 survey using 2005 TEFs. The total TEQ could not be quantified for the 1988 survey because not all congeners contributing to the TEQ were determined in that survey.

Figure 5 shows the total TEQ of the 1998 and 2008 survey and the relative contributions of dioxins/furans and PCBs. This indicates that the total TEQ has decreased from 8.7 pg TEQ/g to 4.8 pg TEQ/g between 1998 and 2008. The contribution of PCBs to the total TEQ has decreased from 32% to 27%, as a result of the larger decrease in the PCB TEQ relative to the decrease in dioxin/furan TEQ.

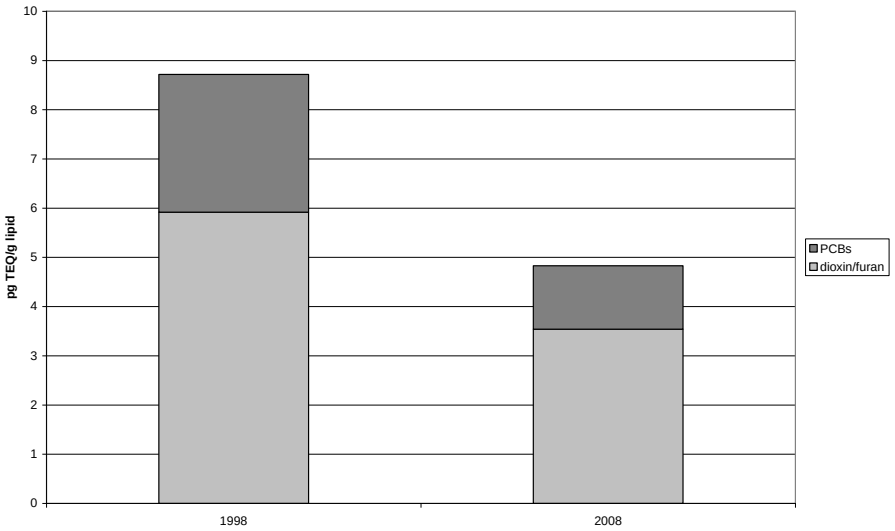


Figure 5. The total TEQ for dioxins/furans and PCBs (using 2005 TEFs) reported for the 1998 and 2008 surveys).

3.2.5. Organochlorine pesticides (OCPs)

Table 16 lists the mean levels of OCPs that were included in at least 2 surveys. For all OCPs the levels of the 2008 surveys were below the levels of the 1998 survey, with the percentage decrease ranging between -34% and -90%.

Table 16. The mean levels of OCPs (ng/g lipid) as reported for the 3 studies.

	1988 (n=38)			1998 (n=52)			2008 (n=37)			% difference	
	mean	SE	<LOD (%)	**mean**	SE	<LOD (%)	**mean**	SE	<LOD (%)	(1988-1998)	(1998-2008)
alpha-HCH				**0.19**	0.02	4%	**0.05**	0.01	5%		-74%
beta-HCH	**11.2**	1.22	95%	**16.3**	2.11	0%	**8.43**	3.46	0%	45%	-48%
gamma-HCH				**0.6**		62%	**0.22**	0.04	27%		-64%
HCB	**31.5**	3.82	3%	**10.6**	0.47	0%	**6.76**	0.62	0%	-67%	-36%
dieldrin	**47.4**	3.84	0%	**15.4**	1.07	0%	**10.14**	1.20	0%	-67%	-34%
heptachlor-epoxide				**4.69**	1.08	0%	**0.47**	0.04	0%		-90%
gamma-chlordane				**0.33**		96%	**0.06**	0.01	8%		-82%
o,p'-DDT	**19.3**	6.89	95%	**4.36**	1.82	0%	**0.56**	0.09	0%	-78%	-87%
p,p'-DDT	**78**	13.2	0%	**25.6**	9.46	0%	**5.02**	0.41	0%	-67%	-80%
p,p'-DDE	**1929**	253	0%	**626**	43.6	0%	**378.95**	41.88	0%	-68%	-39%

Figures 6 and 7 show the mean levels over time of those OCPs that were detected in all of the samples in the 1998 and 2008 surveys. Note that the

1988 level of *p,p*'-DDT is off the scale of the graph. NQ signifies the compound was not quantified in the 1988 survey.

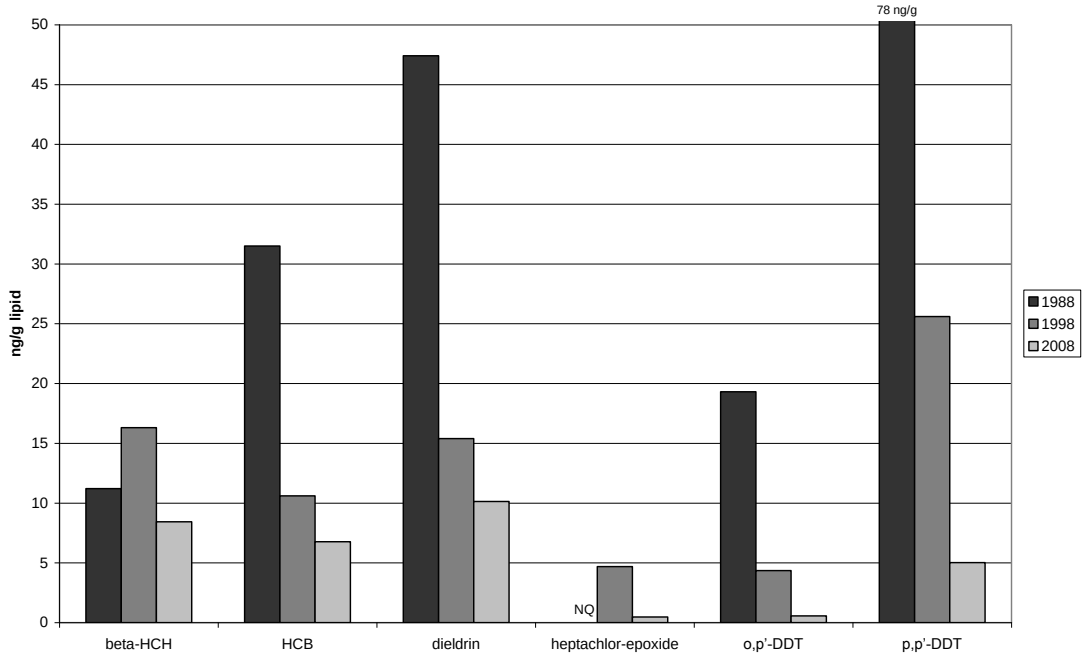


Figure 6. Mean levels of individual OCPs as reported for the 3 studies (only listing those compounds that were detected in all samples of the 1998 and 2008 surveys).

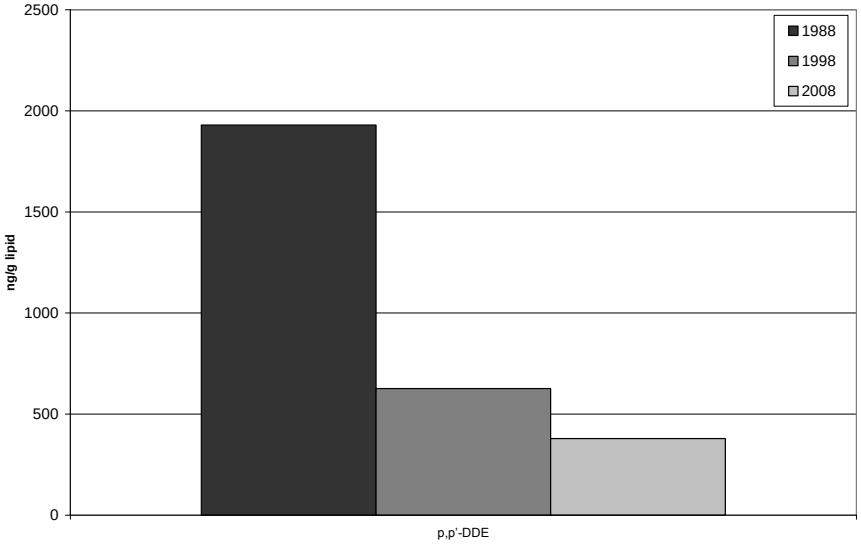


Figure 7. Mean levels of *p,p'*-DDE as reported for the 3 studies.

Because DDE is the major metabolite of DDT, the relative proportion of DDT and DDE detected in breast milk can be an indication of the length of time since exposure. The proportion of *p,p'*-DDT was therefore compared with the *p,p'*-DDT+*p,p'*-DDE levels. This indicated that in 1988 the *p,p'*-DDT

represented 4% of total *p,p'*-DDT+*p,p'*-DDE. In 1998 this was also 4%. In 2008 this had decreased to 1%, indicated that the contribution of the parent compound had decreased in relation to its metabolite.

3.3. Population characteristics in relation to POPs levels

The study population was categorized in groups according to various characteristics, and the POPs levels were compared. Student t-tests were applied to determine whether differences were statistically significant.

3.3.1. Dioxins, furans and dioxin-like PCBs

The total TEQ for dioxins/furans as well as PCBs was higher in rural areas, with the difference being statistically significant (p=0.0168 and p=0.0021) (see table 17). The mean dioxin/furan levels were slightly higher for the North Island when comparing the urban areas only, but this difference was not statistically significant. Although the age range of the study participants was narrow (20-30 years) the higher age group had higher levels of both dioxins/furans and dioxin-like PCBs. This difference was statistically significant for the PCB TEQ (p=0.0178).

Table 17. Comparison of mean levels of furan/dioxin TEQ and PCB TEQ across study population groups.

	mean	Std Err	**mean**	Std Err	*p-value (t-test)*
by urban/rural	**urban**		**rural**		
dioxins and furans 2005 WHOTEQ (incl half LOD)	**3.12**	0.19	**4.38**	0.60	**0.0168*
dioxins and furans 2005 WHOTEQ (excl half LOD)	**2.95**	0.20	**4.21**	0.58	**0.0151*
PCBs 2005 WHOTEQ (incl half LOD)	**1.06**	0.09	**1.75**	0.24	**0.0021*
PCBs 2005 WHOTEQ (excl half LOD)	**1.02**	0.10	**1.54**	0.29	**0.0389*
by North/South (only urban areas)	**North**		**South**		
dioxins and furans 2005 WHOTEQ (incl half LOD)	**3.31**	0.21	**2.78**	0.38	*0.2005*
dioxins and furans 2005 WHOTEQ (excl half LOD)	**3.12**	0.22	**2.62**	0.40	*0.2430*
PCBs 2005 WHOTEQ (incl half LOD)	**1.08**	0.12	**1.03**	0.11	*0.7738*
PCBs 2005 WHOTEQ (excl half LOD)	**1.02**	0.14	**1.01**	0.12	*0.9483*
by age group	**20-25**		**26-30**		
dioxins and furans 2005 WHOTEQ (incl half LOD)	**3.06**	0.66	**3.78**	0.19	*0.1837*
dioxins and furans 2005 WHOTEQ (excl half LOD)	**2.86**	0.63	**3.62**	0.19	*0.1548*
PCBs 2005 WHOTEQ (incl half LOD)	**0.93**	0.10	**1.47**	0.15	**0.0178*
PCBs 2005 WHOTEQ (excl half LOD)	**0.71**	0.09	**1.43**	0.16	**0.0038*
by baby's sex	**boy**		**girl**		
dioxins and furans 2005 WHOTEQ (incl half LOD)	**3.39**	0.44	**3.69**	0.27	*0.5534*
dioxins and furans 2005 WHOTEQ (excl half LOD)	**3.20**	0.43	**3.53**	0.28	*0.5257*
PCBs 2005 WHOTEQ (incl half LOD)	**1.25**	0.14	**1.34**	0.17	*0.6900*
PCBs 2005 WHOTEQ (excl half LOD)	**1.20**	0.15	**1.18**	0.19	*0.9257*
			Continued on next page		

Table 17. *Continued*

	mean	Std Err	mean	Std Err	*p-value (t-test)*
by BMI	**20-25**		**26-33**		
dioxins and furans 2005 WHOTEQ (incl half LOD)	**3.56**	0.41	**3.52**	0.27	*0.9313*
dioxins and furans 2005 WHOTEQ (excl half LOD)	**3.39**	0.39	**3.34**	0.27	*0.9276*
PCBs 2005 WHOTEQ (incl half LOD)	**1.38**	0.17	**1.18**	0.13	*0.3725*
PCBs 2005 WHOTEQ (excl half LOD)	**1.24**	0.19	**1.13**	0.14	*0.6809*
by lipid content (only urban areas)	**1.4%-3.5%**		**3.6%-7.4%**		
dioxins and furans 2005 WHOTEQ (incl half LOD)	**3.25**	0.43	**3.07**	0.21	*0.6761*
dioxins and furans 2005 WHOTEQ (excl half LOD)	**2.99**	0.45	**2.93**	0.22	*0.8939*
PCBs 2005 WHOTEQ (incl half LOD)	**1.26**	0.24	**0.98**	0.07	*0.1472*
PCBs 2005 WHOTEQ (excl half LOD)	**1.19**	0.26	**0.94**	0.08	*0.2501*
by smoking status	**never**		**ex/current**		
dioxins and furans 2005 WHOTEQ (incl half LOD)	**3.30**	0.18	**4.49**	0.99	*0.0574*
dioxins and furans 2005 WHOTEQ (excl half LOD)	**3.14**	0.19	**4.27**	0.96	*0.0645*
PCBs 2005 WHOTEQ (incl half LOD)	**1.34**	0.12	**1.12**	0.26	*0.4483*
PCBs 2005 WHOTEQ (excl half LOD)	**1.25**	0.13	**0.95**	0.32	*0.3307*
by weight gain during pregnancy	**<6kg**		**6 kg or more**		
dioxins and furans 2005 WHOTEQ (incl half LOD)	**3.39**	0.22	**3.87**	0.49	*0.3722*
dioxins and furans 2005 WHOTEQ (excl half LOD)	**3.22**	0.23	**3.69**	0.47	*0.3736*
PCBs 2005 WHOTEQ (incl half LOD)	**1.28**	0.11	**1.21**	0.16	*0.7144*
PCBs 2005 WHOTEQ (excl half LOD)	**1.28**	0.11	**1.00**	0.18	*0.2036*

The differences between never and ever smokers for the dioxin/furan TEQ was marginally statistically significant, with ever smokers having a higher dioxin/furan TEQ compared to never smokers. This pattern was also observed in the 1998 survey but the differences were, as in the 2008 survey, not statistically significant. There were no statistically significant differences for the other population characteristics (table 17).

3.3.2. Organochlorine pesticides (OCPs)

As for the dioxins/furans and dioxin-like PCBs, the main population characteristics associated with OCP levels were urban/rural area and age (see table 18). The mean levels of dieldrin (insecticide and metabolite of aldrin) and *p,p'*-DDE (the major metabolite of DDT) were markedly higher in rural areas when compared to urban areas (p=0.0003 and p=0.0096 respectively).

For all OCPs under study except dieldrin, the mean level in the older age group (25-30) was higher than the mean level in the younger (20-25) age group. This difference was statistically significant for heptachlor-epoxide, *o,p'*-DDT, *p,p'*-DDE and mirex (p=0.0194, p=0.0371, p=0.0183 and p=0.0201 respectively). Smoking was not related to OCP levels, in contrast to the 1998 survey, which observed statistically significantly higher levels of heptachlor-epoxide for ever smokers compared to never smokers. None of the other population characteristics were associated with measured OCP levels.

Table 18. Comparison of mean levels of OCPs across study population groups.

	mean	Std Err	**mean**	Std Err	*p-value (t-test)*
by urban/rural	**urban**		**rural**		
beta-HCH	**10.18**	5.10	**4.27**	0.88	*0.4738*
HCB	**6.99**	0.89	**6.30**	0.49	*0.6088*
Dieldrin	**7.34**	1.08	**15.96**	2.14	**0.0003*
heptachlor-epoxide	**0.46**	0.05	**0.47**	0.06	*0.8835*
p,p'-DDT	**4.62**	0.50	**5.87**	0.66	*0.1527*
o,p'-DDT	**0.52**	0.12	**0.63**	0.13	*0.5744*
p,p'-DDD	**0.12**	0.01	**0.14**	0.01	*0.1728*
p,p'-DDE	**305.76**	40.23	**531.42**	84.91	**0.0096*
mirex	**0.22**	0.02	**0.25**	0.05	*0.5162*
by North/South (only urban areas)	**North**		**South**		
beta-HCH	**13.81**	7.91	**3.74**	0.74	*0.3547*
HCB	**7.68**	1.35	**5.76**	0.51	*0.3131*
Dieldrin	**7.65**	1.67	**6.8**	0.68	*0.7177*
heptachlor-epoxide	**0.46**	0.06	**0.45**	0.08	*0.9238*
p,p'-DDT	**4.68**	0.71	**4.50**	0.65	*0.8622*
o,p'-DDT	**0.66**	0.18	**0.27**	0.02	*0.1267*
p,p'-DDD	**0.11**	0.01	**0.12**	0.01	*0.5232*
p,p'-DDE	**295.19**	59.44	**324.56**	40.54	*0.7341*
mirex	**0.24**	0.03	**0.18**	0.02	*0.1396*
by age group	**20-25**		**26-30**		
beta-HCH	**3.20**	0.58	**11.27**	5.28	*0.2718*
HCB	**5.26**	0.28	**7.58**	0.91	*0.0752*
Dieldrin	**10.33**	1.98	**10.03**	1.53	*0.9084*
heptachlor-epoxide	**0.35**	0.03	**0.53**	0.05	**0.0194*
p,p'-DDT	**4.15**	0.54	**5.50**	0.54	*0.1164*
o,p'-DDT	**0.30**	0.02	**0.70**	0.14	**0.0371*
p,p'-DDD	**0.11**	0.01	**0.13**	0.01	*0.4050*
p,p'-DDE	**247.15**	21.67	**450.33**	58.89	**0.0183*
mirex	**0.16**	0.01	**0.27**	0.03	**0.0201*
by baby's sex	**Boy**		**girl**		
beta-HCH	**10.89**	6.75	**5.84**	0.74	*0.4741*
HCB	**7.27**	1.16	**6.23**	0.37	*0.4115*
Dieldrin	**9.15**	1.31	**11.18**	2.05	*0.4049*
heptachlor-epoxide	**0.46**	0.05	**0.47**	0.05	*0.8481*
p,p'-DDT	**5.21**	0.63	**4.83**	0.52	*0.6422*
o,p'-DDT	**0.61**	0.15	**0.50**	0.10	*0.5632*
p,p'-DDD	**0.13**	0.01	**0.11**	0.01	*0.2180*
p,p'-DDE	**388.42**	54.84	**368.94**	65.28	*0.8199*
mirex	**0.23**	0.03	**0.23**	0.03	*0.9033*
			Continued on next page		

Table 18. *Continued.*

	mean	Std Err	**mean**	Std Err	*p-value (t-test)*
by BMI	**20-25**		**26-33**		
beta-HCH	**10.50**	6.10	**5.72**	0.76	*0.5013*
HCB	**7.18**	1.05	**6.22**	0.42	*0.4508*
Dieldrin	**10.49**	1.49	**9.68**	2.01	*0.7421*
heptachlor-epoxide	**0.46**	0.05	**0.47**	0.05	*0.9717*
p,p'-DDT	**4.96**	0.57	**5.11**	0.59	*0.8617*
o,p'-DDT	**0.58**	0.14	**0.52**	0.12	*0.7302*
p,p'-DDD	**0.13**	0.01	**0.11**	0.01	*0.1947*
p,p'-DDE	**372.33**	57.11	**387.63**	63.38	*0.8594*
mirex	**0.24**	0.02	**0.22**	0.04	*0.7856*
by lipid content (only urban areas)	**1.4%-3.5%**		**3.6%-7.4%**		
beta-HCH	**21.02**	15.87	**5.08**	0.74	*0.1491*
HCB	**8.27**	2.54	**6.38**	0.60	*0.3342*
Dieldrin	**6.79**	1.64	**7.60**	1.42	*0.7331*
heptachlor-epoxide	**0.40**	0.05	**0.49**	0.06	*0.3813*
p,p'-DDT	**4.76**	1.21	**4.55**	0.50	*0.8547*
o,p'-DDT	**0.72**	0.33	**0.42**	0.10	*0.2718*
p,p'-DDD	**0.13**	0.02	**0.11**	0.01	*0.2474*
p,p'-DDE	**226.13**	29.24	**343.24**	55.86	*0.1798*
mirex	**0.23**	0.03	**0.22**	0.03	*0.7689*
by smoking status	**never**		**ex/current**		
beta-HCH	**9.43**	4.26	**4.14**	1.19	*0.5566*
HCB	**7.14**	0.74	**5.16**	0.65	*0.2182*
Dieldrin	**10.57**	1.35	**8.30**	2.61	*0.4658*
heptachlor-epoxide	**0.49**	0.04	**0.37**	0.10	*0.2034*
p,p'-DDT	**5.37**	0.47	**3.55**	0.55	*0.0799*
o,p'-DDT	**0.60**	0.11	**0.37**	0.08	*0.3461*
p,p'-DDD	**0.12**	0.01	**0.11**	0.02	*0.5194*
p,p'-DDE	**371.70**	41.08	**410.00**	143.04	*0.7256*
mirex	**0.22**	0.02	**0.27**	0.09	*0.3635*
by weight gain during pregnancy	**<6kg**		**6 kg or more**		
beta-HCH	**4.63**	0.71	**13.54**	7.93	*0.2434*
HCB	**6.30**	0.40	**7.66**	1.34	*0.3161*
Dieldrin	**11.17**	1.89	**9.29**	1.77	*0.4762*
heptachlor-epoxide	**0.50**	0.05	**0.42**	0.06	*0.3000*
p,p'-DDT	**4.70**	0.52	**5.53**	0.72	*0.3456*
o,p'-DDT	**0.42**	0.07	**0.75**	0.19	*0.0928*
p,p'-DDD	**0.12**	0.01	**0.13**	0.01	*0.4562*
p,p'-DDE	**356.89**	53.59	**407.31**	76.85	*0.5876*
mirex	**0.20**	0.02	**0.28**	0.04	*0.0953*

3.3.3. Brominated flame retardants

Table 19 shows the differences in concentration between different groups for the five most commonly measured and reported BDEs. Other BFRs were also studied, and results are presented in the text for those which showed statistically significant differences ($p_{t\text{-}test}<0.05$).

There were no statistically significant differences in mean BDE levels between urban and rural areas for the five most common BDEs, but statistically significantly higher levels were observed for the urban areas for BDE184, BDE196, BDE197, BDE201, BDE203 and BDE207.

There were no statistically significant differences in BDE levels between the North and the South Island for the 5 most common BDEs, or any of the other brominated analytes (only including urban areas). Age also did not affect the BDE levels, with the BDE levels of the 20-25 year olds being very similar to those of the 25-30 year olds.

For all five most common BDEs, levels were higher for those with a lower BMI with the difference being marginally statistically significant for BDE 99 and BDE 154. Statistically significantly higher levels for the low BMI group were in addition observed for BDE28&33, BDE49 and BDE66, compared to the low BMI group.

Table 19. Comparison of mean levels of selected BDEs across study population groups.

	mean	Std Err	**mean**	Std Err	*p-value (t-test)*
by urban/rural	**urban**		**rural**		
BDE 47	**2680.56**	309.97	**2253.25**	470.04	*0.4450*
BDE 99	**577.56**	61.38	**437.43**	93.83	*0.2104*
BDE 100	**538.32**	75.59	**530.40**	125.03	*0.9549*
BDE 153	**716.64**	71.94	**718.17**	285.67	*0.9945*
BDE 154	**40.57**	4.61	**29.57**	5.28	*0.1569*
by North/South (only urban areas)	**North**		**South**		
BDE 47	**2528.38**	310.37	**2951.11**	682.13	*0.5243*
BDE 99	**561.00**	76.17	**607.00**	109.00	*0.7272*
BDE 100	**461.94**	58.56	**674.11**	180.5	*0.1833*
BDE 153	**654.38**	89.88	**827.33**	117.5	*0.2570*
BDE 154	**36.99**	5.24	**46.93**	8.79	*0.3101*
by age group	**20-25**		**26-30**		
BDE 47	**2505.31**	548.16	**2561.83**	273.84	*0.9183*
BDE 99	**551.17**	109.13	**521.79**	55.76	*0.7911*
BDE 100	**548.83**	137.31	**528.67**	67.93	*0.8833*
BDE 153	**707.54**	116.53	**722.33**	145.94	*0.9460*
BDE 154	**41.03**	8.44	**34.82**	3.29	*0.4201*
			Continued on next page		

Table 19. *Continued.*

	mean	Std Err	**mean**	Std Err	*p-value (t-test)*
by baby's sex	**boy**		**girl**		
BDE 47	**2414.16**	316.90	**2676.89**	417.40	*0.6169*
BDE 99	**505.06**	66.46	**560.67**	81.70	*0.5991*
BDE 100	**502.62**	85.46	**570.72**	98.21	*0.6031*
BDE 153	**528.89**	50.52	**915.83**	194.87	*0.0569*
BDE 154	**34.69**	4.52	**39.44**	5.77	*0.5188*
by BMI	**20-25**		**26-33**		
BDE 47	**2923.81**	385.30	**2040.81**	280.35	*0.0894*
BDE 99	**615.67**	75.41	**422.45**	59.64	*0.0640*
BDE 100	**622.10**	97.97	**422.43**	67.66	*0.1250*
BDE 153	**724.86**	82.90	**707.00**	213.97	*0.9324*
BDE 154	**42.83**	5.60	**29.36**	3.27	*0.0638*
by lipid content (only urban areas)	**1.4%-3.5%**		**3.6%-7.4%**		
BDE 47	**2371.75**	571.38	**2825.88**	374.98	*0.5060*
BDE 99	**477.38**	115.73	**624.71**	71.54	*0.2718*
BDE 100	**473.13**	143.43	**569.00**	90.56	*0.5652*
BDE 153	**644.88**	128.64	**750.41**	88.23	*0.5054*
BDE 154	**33.14**	8.22	**44.06**	5.52	*0.2778*
by smoking status	**never**		**ex/current**		
BDE 47	**2488.03**	297.03	**2773.14**	513.50	*0.6707*
BDE 99	**505.81**	55.52	**644.86**	137.16	*0.3001*
BDE 100	**505.29**	69.58	**666.29**	164.23	*0.3331*
BDE 153	**703.27**	121.30	**776.57**	157.78	*0.7829*
BDE 154	**34.86**	3.75	**46.19**	10.23	*0.2244*
by weight gain during pregnancy	**<6kg**		**6 kg or more**		
BDE 47	**2687.06**	419.73	**2285.38**	349.06	*0.4736*
BDE 99	**589.90**	84.04	**456.81**	65.82	*0.2296*
BDE 100	**579.21**	104.52	**434.38**	77.21	*0.2832*
BDE 153	**913.28**	192.27	**499.56**	63.34	*0.0607*
BDE 154	**42.52**	5.97	**28.06**	3.53	*0.0520*

The BDE levels were also higher for women who gained less than 6 kg over pregnancy, compared to those gaining more than 6 kgs. The differences were marginally statistically significant for BDE153 and BDE154, and statistically significant for BDE171, BDE180, BDE183&175 and BDE191. BDE levels were higher for breast milk samples with a higher lipid content for the 5 most common BDEs, but this difference was not statistically significant. For BDE30, BDE156&169 and DBDPE the levels were statistically significantly higher in the women with lower lipid levels. There were no statistically significant differences in levels between ever and never smokers for any of the BFRs except for BDE15 of which the levels were higher for ever smokers.

4. DISCUSSION

The strength of this study is its comparability in study design and recruitment procedures with the two previous breast milk surveys conducted in New Zealand 10 and 20 years prior to the current survey. The participant inclusion criteria have however been increasingly difficult to meet over time. In particular, the age criteria set for the study (first time mothers aged 20-30) were already difficult to meet in the 1998 survey and proved to be even more difficult in the 2008 survey. However, considering that age is such a strong determinant of levels of dioxins, furans, PCBs and OCPs, changing the selection criteria would have compromised the validity of the comparison with previous surveys and the subsequent assessment of time trends. Maintaining the strict inclusion criteria has ensured that the population characteristics of the 2008 survey were very similar to those of the 1998 survey.

This study showed that in breast-feeding first time mothers aged 20-30 years in New Zealand the breast milk levels of dioxins/furans, PCBs and OCPs have continued to decline over the last decade. The previous survey reported a decline of 70% of the dioxins/furans over the period 1988-1998 and here we report a further decline of 40% over the period 1998-2008. Over this period, the levels of dioxin-like PCBs have declined more sharply, by 54%. The decline in levels of selected OCPs (*alpha*-HCH, *beta*-HCH, *gamma*-HCH, HCB, dieldrin, heptachlor-epoxide, *gamma*-chlordane, *o,p'*-DDT, *p,p'*-DDT, *p,p'*-DDE) ranged between 34% and 90% over the same period.

It is generally difficult to compare time trends with those observed in other countries, as few countries have conducted repeated surveys using methods that would allow such comparisons, and the time periods addressed differ between different countries. A mini-monograph on time trends and regional variability of chemical contaminants in breast milk, was published in 2002 [6]. It reported that coordinated WHO studies in Europe from 1986 to 1993 showed an average decrease in dioxin levels of approximately 35%, with consistently higher levels in industrial areas. Sweden, for which extensive time trend data is available, reported a downward trend in average breast milk levels over 25 years, with a relatively steep decline from the 1970s to the mid-1980s [6] and a further decline over the period 1996-2005 of 6-9% per year for dioxins, 3-6% per year for furans and 4-9% per year for PCBs[7]. Between 1997 and 2007 the dioxin/furan TEQ declined from approximately 8 pg TEQ/g lipid to approximately 5 pg/g lipid[7] for Sweden, compared to 5.9 pg TEQ/g lipid to 3.5 pg TEQ/g lipid over 1998-2008 reported here for New Zealand. The percentage decrease over this time period is therefore comparable between Sweden and New Zealand, although the actual concentrations in New Zealand are more than 25% below the Swedish levels. For Sweden, levels of PCB153 declined from approximately 70 ng/g lipid to 30 ng/g lipid over a 10 year period between 1997 and 2007 [7], compared to a decline in PCB153 levels from 9.8 to 5.6 ng/g lipid in the 10-year period between 1998 and 2008 in New Zealand. Again, the percentage decline in levels for PCB 153 was comparable between Sweden and New Zealand, but New Zealand levels were considerably lower compared to the Swedish levels. In countries where

OCPs have been banned for several decades, as is the case for New Zealand, the average breast-milk levels of OCPs have generally decreased substantially [6], a pattern again observed for this study. The current study therefore indicates that New Zealand's time trends of POPs are similar to those observed for comparable countries, but also provides evidence that a continued decrease in POPs levels (dioxins/furans, PCBs and OCPs) is not only present in countries with high baseline levels, but also in regions where the breast milk levels of POPs are relatively low by international comparison.

This survey has also provided New Zealand baseline data for brominated flame retardants (BFRs), in particular PBDEs. These results suggest, as did a previous study using serum samples[8], that New Zealand's levels of these compounds are low in comparison with Australia and the US, but are comparable with, or in some cases higher than, those observed in some European countries. For example, the PBDE levels in Australia[9] are about twice as high as those measured for New Zealand, while the levels measured for Belgium[10] are about half the New Zealand levels. For the US[11,12] BDE breast milk levels are 3 to 6 times the levels found in New Zealand. This suggests that New Zealand is an area with moderate to low BDE levels. Because this is the first study to measure these compounds New Zealand breast milk, time trend data cannot be provided. Studies from other countries have, however, indicated that breast milk levels of these compounds have been increasing over time [7], as many of the BFRs are still in use. Continued monitoring of these emerging POPs is therefore paramount.

The congener profile of PBDEs in breast milk was dominated by BDE47, BDE153, BDE99 and BDE100, which is consistent with that observed in for example Australia[9] and other background exposed populations[13]. These congeners are components of the commercial mixture "pentabrom", which has primarily been used as an additive in polyurethane foams, where up to 30% of the weight of the foam can be accounted for by this flame retardant. Pentabrom was banned in Europe at the turn of the century and voluntarily withdrawn in the United States in 2004 because of the persistence in the environment and unknown safety [14]. PBDEs have not been produced or imported as a raw product in New Zealand, and human exposure may therefore have occurred and continue to occur via imported goods containing "pentabrom" or other flame retardants. BDE209 also appeared to be prominently present in the New Zealand breast milk samples, which points towards exposure to the commercial mixture "decaBDE" or "DBDE" of which BDE209 is the main component. It is used as an additive flame retardant primarily in electrical and electronic equipment and continues to be widely used.[14] BDE209 is not considered a POP under the Stockholm Convention as it is less likely to bioaccumulate due to its short half-life[15,16]. Its short half-life however suggests that the BDE209 levels in breast milk reported here may be due to recent exposure to flame retardants containing BDE209. It should be noted that serum levels of BDE209 may be considerably higher (i.e. 10-fold) than those measured in breast milk, because of the suggested less efficient transfer of higher brominated congeners from blood to breast milk compared to the lower brominated congeners[11,17]. The breast milk levels for BDE209 as

reported here are therefore likely to underestimate the actual body burden of this compound.

This survey, as well as the 1998 survey, also investigated whether certain population characteristics are associated with POPs levels. As repeatedly observed in other countries [6], age was a strong determinant of breast milk levels of dioxins/furans, PCBs and OCPs, with higher age being associated with higher levels of POPs. This pattern could even be observed within the relatively narrow age range (20-30) of the women included. This illustrates the importance of including women of comparable age if surveys are to be repeated, and studying time trends is one of the main objectives. The strong association with age can be attributed to the accumulation of POPs in the body over time, as well as the trend that older generations have generally been exposed to higher levels than younger generations in regions where POPs levels have declined over time.

In the 2008 survey higher levels of dioxins/furans, PCBs and OCPs were observed for rural areas compared to urban areas. This pattern was not as strong in the 1998 survey. Other countries have generally reported higher levels of dioxins/furans and PCBs for industrial areas. In this survey substantially higher levels of *p,p'*-DDE and dieldrin ($p<0.05$) were observed for the rural areas when compared to the urban areas. These differences are likely due to the environmental contamination of rural areas where these pesticides were used in the past.

Differences in PBDE levels between subgroups followed a different pattern than that observed for dioxins/furans, PCBs and OCPs. In particular, there was a lack of an association between age and PBDE levels, which has also been reported by other studies from Germany [18], the US [12] and Sweden [7], although the Swedish study did observe higher levels of BDE153 being associated with a higher age. Studies from Asia also reported an age-dependency for BDE153, although the direction of the association was different for different populations [19]. The levels of BDEs tended to be higher for the low BMI group (20-25 kg/m^2) compared to the high BMI group (26-33 kg/m^2). Similarly, less weight gain over pregnancy was associated with higher levels of some BDEs. This inverse association between weight indicators and BDE levels has also been observed for Swedish women [7] and the authors of the study hypothesized that the inverse association could be due to a "dilution" effect of BDEs, and that weight loss could result in the mobilisation of BDEs from body fat. Another study from the US however observed higher breast milk levels of BDEs among mothers with high compared to normal BMI[14].

Urban or rural residency was not associated with levels of the four most abundant BDEs (BDE47, BDE99, BDE100, BDE153), but statistically significantly higher levels were observed for the urban areas for BDE184, BDE196, BDE197, BDE201, BDE203 and BDE207. The exposure pathways for BDEs are not fully understood, but it has been suggested that besides diet, indoor air and particularly dust[20] is an important source of BDEs. The determination of PBDEs in the house dust samples taken from the houses of

the participants of the 2008 survey may provide further insights into why levels of selected BDEs are higher in urban areas.

Finally, the results of this study combined with those of the two previous surveys conducted 10 and 20 years previously, illustrate the value of the continued monitoring of POPs and the inclusion of emerging POPs in such surveys.

5. CONCLUSIONS

- Over the period 1998-2008, dioxin and furan levels in breast milk have declined by 40% in breast feeding women aged 20-30 years in New Zealand; it is likely that similar declines have occurred in the general New Zealand population.

- Over the period 1998-2008, the levels of dioxin-like PCBs have declined by 54%.

- The decline in levels of OCPs (*alpha*-HCH, *beta*-HCH, *gamma*-HCH, HCB, dieldrin, heptachlor-epoxide, *gamma*-chlordane, *o,p'*-DDT, *p,p'*-DDT, *p,p'*-DDE) ranged between 34% and 90% over the period 1998-2008.

- The New Zealand levels of dioxins/furans, PCBs and OCPs remain low by international standards.

- This survey has provided baseline data for breast milk levels of brominated flame retardants (BFRs) including PBDEs: the New Zealand levels of these compounds are moderate to low by international standards.

- As observed in the previous two surveys, the lipid content of the breast milk was higher in urban areas compared to rural areas.

- Levels of dioxins/furans, PCBs and OCPs tended to be higher in rural areas compared to urban areas.

- Levels of some PBDEs were higher in urban areas compared to rural areas.

- The age of the mother was positively associated with the breast milk levels of dioxins/furans, PCBs and OCPs.

- The age of the mother was not associated with the breast milk levels of brominated flame retardants including PBDEs.

- Body mass index (BMI) was inversely associated with the levels of some PBDEs.

- There were no statistically significant differences in POPs levels (dioxins/furans, PCBs, OCPs or BFRs), between the South and the North Island of New Zealand.

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