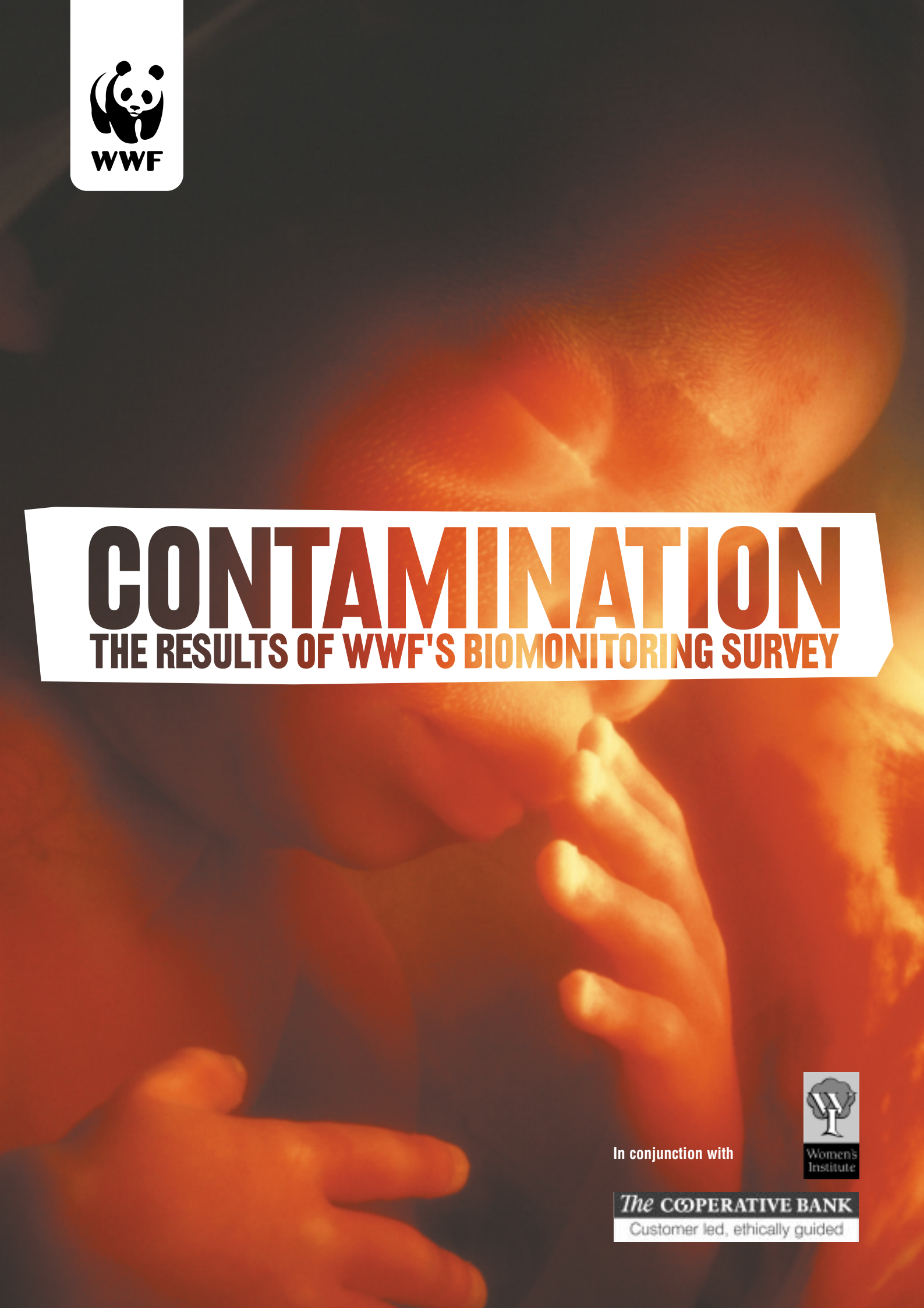

CONTAMINATION

THE RESULTS OF WWF'S BIOMONITORING SURVEY

WWF

In conjunction with

Women's Institute

The Co-operative Bank
Customer led, ethically guided

This advertisement appeared in the media in 2002 but was banned by the Advertising Standards Authority (ASA), even though it agreed with WWF that man-made chemicals are causing widespread contamination of humans and wildlife. Chemical industry lobby groups had complained that the advertisement overstated the risk of chemicals to human health – but the ASA rejected all complaints relating to the scientific and technical content of the advertisement. Industry representatives disputed whether:

- **more than 300 man-made chemicals are present in human bodies;**
- **all these chemicals are present in the foetus;**
- **man-made chemicals are more dangerous than naturally occurring ones;**
- **presence of these chemicals is dangerous even at low levels;**
- **pollutants are found in intensively farmed food; and**
- **man-made chemicals are linked to birth defects in humans.**

The ASA found WWF's scientific research to be above reproach on all fronts and rejected every technical complaint. But despite being ruled factually accurate and being in the public interest, the advertisement was nevertheless banned on the grounds that it was "unduly alarming".

WWF is perturbed that, by asking the ASA to ban this advertisement, the chemical lobby has managed to influence what the public is allowed to know about chemical contamination. The lobby is now attempting to weaken the proposed EU chemical legislation which, as a result, could end up providing no additional public protection.

Given the concern expressed by international organisations such as the European Commission and the World Health Organisation about the potential harm from man-made chemicals, this report makes unnerving reading. We are facing an uncontrolled global experiment where humans and wildlife are being exposed to man-made synthetic chemicals that have the potential to harm.
It is time to wake up to this threat and ensure that exposure to such chemicals is controlled – and, where necessary, that they are banned.

The advertisement is shown in full on the inside back cover.

CONTAMINATION

THE RESULTS OF WWF'S BIOMONITORING SURVEY NOVEMBER 2003

The authors have used all reasonable endeavours to ensure that the content of this report, the data compiled, and the methods of calculation and research are consistent with normally accepted standards and practices. However, no warranty is given to that effect nor any liability accepted by the authors for any loss or damage arising from the use of this report by WWF-UK or by any other party.

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WWF-UK National Biomonitoring Survey 2003

Where chemicals are found in elevated concentrations in biological fluids such as breast milk, they should be removed from the market immediately.

Royal Commission on Environmental Pollution, 2003

Often the weakest link in determining whether observed adverse effects in humans and/or wildlife are linked to EDCs is the absence of adequate exposure data…

Data on the magnitude and trends of global human or wildlife exposure is limited. Potential sources of exposure are through contaminated food, contaminated groundwater, combustion sources, and contaminants in consumer products. Information on exposure during critical development periods is generally lacking.

The exposure data sets that exist are primarily for various environmental media (air, food, water) rather than the most relevant internal exposure (blood, tissue). Limited exceptions are human breast milk and adipose tissue samples. Worldwide, in spite of large expenditures of money, time and effort, comparable data sets for assessing exposures to EDCs for humans or wildlife are not available. Such information is essential to adequately evaluate exposure/response relationships in field and epidemiology studies and to use these relationships to produce credible risk assessments.

World Health Organisation, 2002

Contents

Executive summary

WWF visited 13 locations in England, Northern Ireland, Scotland and Wales in the summer of 2003 and took blood samples from 155 volunteers. Lancaster University analysed the samples for 78 chemicals – 12 organochlorine pesticides (including DDT and lindane), 45 PCB congeners and 21 polybrominated diphenyl ethers (PBDE) flame retardants, including those found in the commercially traded penta-, octa- and deca-BDEs.

WWF believes that this survey provides the first data on the concentrations of PCBs, organochlorine pesticides and PBDEs in the UK population's blood serum. Further, these results form the most comprehensive and largest data-set of organohalogen chemical concentrations in humans in the UK in the last 10 years at least. In addition, the survey is the first which tries to link findings of chemical contamination to people's lifestyles.

FINDINGS

- Every person tested is contaminated by a cocktail of known highly toxic chemicals which were banned from use in the UK during the 1970s and which continue to pose unknown health risks.
- We found 70 (90 per cent) of the 78 chemicals we looked for in the survey. The highest number of chemicals found in any one person was 49 – nearly two thirds (63 per cent) of the chemicals looked for.
- Every person is contaminated by chemicals from each group: organochlorine pesticides, PCBs and PBDEs (flame retardants).
- The highest concentration of any chemical found was 2,557 ng/g (ng/g – parts per billion) of the DDT metabolite p,p'-DDE. The use of DDT was banned in the UK more than 20 years ago.
- The most frequently detected chemicals were PCB congeners 99 and 118 and the DDT metabolite p',p-DDE, which were detected in all but one of the 155 volunteers.
- Ten chemicals were found in more than 95 per cent of volunteers (PCB congeners 99, 118, 138, 153, 156, 170, 180, 194, PBDE 153 and the organochlorine pesticides _-HCH and p',p-DDE).
- This is the first survey to identify the widespread contamination of non-occupationally exposed people to the deca-BDE brominated flame retardant product. Worryingly, the highest levels found in our non-occupationally exposed volunteers were very similar to those observed in Sweden of people occupationally exposed to deca-BDE.
- We are being contaminated daily by "unregulated" chemicals of unknown toxicity, such as the deca-BDE flame retardant. Since there is a dearth of knowledge on the levels of brominated flame retardants in the UK population, it is not possible to determine any trend in contaminant levels.
- PCB contamination is gradually decreasing from levels found in the UK 10 years ago – which indicates that strong regulations work.

- Small numbers of people continue to be exposed and contaminated with high levels of certain chemicals, although median levels of some chemicals are decreasing compared with some earlier studies.
- Volunteers tested in Nottingham had the highest median level of total chemical contamination of the chemicals we looked for. They also had the highest median level of PCBs, organochlorine pesticides and of DDT and its metabolites. Further regional findings are presented in Appendix 1.
- The lifestyle questionnaire identified two factors which significantly affected the level of contamination of individual chemicals:
    - older people have higher levels of PCBs in their blood; and
    - women have lower levels of certain PCBs than men and the levels appear to reduce in relation to the number of children they carried and breast-fed. These differences seem to be related to women "off-loading" some of their chemical burden to their children.

CONCLUSIONS

Learning the lessons*?*

UK and EU legislators and chemical regulators have not learned the lessons from past experiences of the adverse effect of persistent and bioaccumulative chemicals on people and wildlife. Current legislation is not adequate, since the use of such chemicals is still allowed. Persistent and bioaccumulative chemicals that have long since been banned continue to contaminate people in the UK, and are now augmented by other chemicals with similar properties which are still being produced and used today – for instance, certain brominated flame retardants.

In WWF's view, the only way to stop this contamination, and the threat to future generations, is to prevent the marketing of chemicals that are found in elevated concentrations in biological fluids such as breast milk. This is also one of the primary recommendations of *Chemicals in Products,* the Royal Commission on Environmental Pollution report published in June 2003.

The Current Opportunity - REACH

Bioaccumulative chemicals in use now should be urgently phased out, using existing legislation. The proposed new EU chemicals regulation known as REACH – the Registration, Evaluation and Authorisation of Chemicals – provides a once in a generation opportunity to secure adequate controls for these substances. Hazardous chemicals, such as the very persistent and very bioaccumulative chemicals (vPvBs) and the endocrine disrupting chemicals (EDCs), should be subject to prior authorisation under the REACH proposals. This would mean that in the first place, there would be a presumption against the use of such chemicals.

To secure the phase-out of these chemicals, however, the current draft of the REACH legislation needs to be improved. Authorisation to use hazardous chemicals should only be granted when there is no safer alternative *and* an overwhelming societal need for them.

The effects of a ban

This survey shows that effective chemical regulations which ban the manufacture and use of hazardous chemicals can eventually start to reduce levels of human contamination. For instance, this survey failed to find the organochlorine pesticide chlordane which has been banned from

use in the UK. However, the process of reducing exposure takes many years and, in some instances, many decades. We found that the levels of PCB contamination are reducing, despite the fact that exposure is still occurring more than 20 years after they were banned in the UK. We also found contamination by DDT, lindane (_-HCH) and hexachlorobenzene (HCB) which have been banned in the UK. This should be an impetus for phasing out chemicals with similar properties.

Lifestyle factors affecting levels of contamination

The lifestyle survey was able to identify only a limited number of statistically significant factors to explain the different levels of contamination. We showed that levels of PCB contamination increased with a person's age, but that women significantly reduced their contamination by passing it on to their children during pregnancy and breast feeding.

It is not unexpected that the lifestyle survey was only able to identify a limited number of contamination risk factors, because the sample number is relatively small and contamination may occur via many different routes – the food chain or indoor air, for example – and could occur on a single occasion. Furthermore, a recent Greenpeace study, *Consuming Chemicals (2003)*, found widespread chemical contamination of house dust in the UK. The levels of chemicals (mg/g – parts per thousand) were far higher than those found in blood (ng/g – parts per billion). This contamination is likely to be due to the fact that many hazardous man-made chemicals are used in household consumer products. For a number of chemicals, especially some of the brominated flame retardants, house-dust might be a significant route of exposure. Penta-BDE, for example, was found in each of the 100 samples analysed, and deca-BDE was the PBDE found at the highest concentrations. We could not take dust contamination into account in our lifestyle survey. If house dust is a significant source of exposure to chemicals, this might explain why the factors considered in our survey – those traditionally associated with exposure to chemicals, such as diet – found only limited associations with levels of contamination.

RECOMMENDATIONS

The number, types and concentrations of chemicals found in the survey, and by extrapolation the UK population, are deplorable. We are still no nearer having definitive explanations for people's exposures to most chemicals. It appears to be a lottery, or even "Russian roulette", as to whether, where, when, how and to what extent we are exposed to chemicals that accumulate in our bodies and potentially interfere with our hormone systems. More needs to be done to protect ourselves and future generations of people and wildlife from this insidious threat. In particular, WWF believes that:

1. The UK government should do all in its power to protect future generations of humans and wildlife by ensuring that REACH requires persistent, bioaccumulative and other hazardous chemicals to be removed from the market. Such measures would reduce the continuing exposure of people and the environment. In particular, the government should support strict conditions for authorising chemicals under REACH. This must include:
    - supporting the inclusion of very persistent and very bioaccumulative (vPvB) chemicals (those likely to be found in biological fluids such as blood and breast milk) and EDCs into the prior authorisation scheme of REACH; and

- prohibiting the use of these chemicals of very high concern, such as vPvBs and EDCs, if safer alternatives exist and there is no overwhelming societal need for them.

2. The best route of protection is to introduce better control of hazardous chemicals so that humans and wildlife are not contaminated in the first place. Chemicals with undesirable properties should be taken off the market. Where this "gatekeeper" approach fails, there should be adequate monitoring to determine the levels of chemicals in the environment and their effects. The UK government should therefore set up coordinated biomonitoring programmes to determine trends in the levels of hazardous chemical in humans, wildlife and the environment. These programmes should be integrated into the risk assessment process so that the detection of chemicals in monitoring surveys should be considered unacceptable and would initiate rapid investigation and the phase-out of a chemical, if appropriate.

3. The detection of deca-BDE in the UK public (identified for the first time in this survey) is sufficient evidence to recommend that it should be subject to a similar EU ban to that recently agreed for the penta- and octa- brominated flame retardants.

1 Introduction

The contamination of the environment by man-made hazardous chemicals will probably not come as a surprise to most people. Over the years, WWF has highlighted the global nature of chemical contamination. Now, from polar bears in the once pristine Arctic through to seals and dolphins around the UK's coast, wildlife throughout the world is contaminated.

But chemical contamination isn't only a global or even a local issue: it is now very much a personal issue. Everyone – not least the next generation – should have the right to a clean, healthy and uncontaminated body so that they achieve their maximum potential without the ever-present worry of their lives being blighted by exposure to chemicals.

Some surveys show that up to 30 per cent of our food is contaminated by man-made hazardous chemicals. Others show that the air and dust in our homes is also contaminated. This survey takes the next logical step by investigating the presence and levels of certain hazardous man-made chemicals in the bodies of a cross-section of people in the UK.

This biomonitoring survey was commissioned as part of WWF's Chemicals and Health Campaign to determine the levels of 78 industrial chemicals in the blood of 155 volunteers around the UK. Its objectives are:

- to determine the concentrations of a range of organohalogen persistent and bioaccumulative chemicals in human blood; and
- to investigate links between chemical concentrations and people's life history or lifestyle parameters.

OUR CHEMICAL ENVIRONMENT

We are now living in an environment contaminated by increasingly large amounts of industrial chemicals, the vast majority of which have not been tested for their effects on human health. The global production of chemicals has increased from 1 million tonnes in 1930 to 400 million tonnes today. Some 100,000 different substances are registered in the EU market, 10,000 of which are marketed in volumes of more than 10 tonnes and a further 20,000 at 1-10 tonnes.

We are exposed to these through the air we breathe, the food we eat and the water we drink. We are exposed to chemicals released directly into the environment from industry, agriculture or other sources of environmental pollution such as vehicle and diesel exhaust, incinerators and tobacco smoke. In addition, many commercial products manufactured by industry and used in or around the home contain chemicals that pose a potential risk to humans.

Very few of these chemicals have been tested for adverse health effects. Only 14 per cent of EU high production volume chemicals have even the minimum "base-set" amount of data and 21 per cent have no data at all (Allanou et al 1999).

Although we know very little about the relationship between exposure and health risk for most of these chemicals, this is not always the case. We know that organochlorines, a class of chlorine-containing compounds including polychlorinated biphenyls (PCBs) and certain pesticides such as DDT, tend to persist in the environment and become concentrated in animal tissues. Many organochlorines have the ability to disrupt the endocrine system, the body's hormonal signalling system which is crucially important for regulating reproduction and development. The developing foetus, infant and child are particularly vulnerable to many of

these compounds. Birth defects and developmental disabilities are increasingly common, and chemical toxicants are known to play a role in causing some of these conditions.

WWF is particularly concerned that very persistent (vP) (those that aren't broken down in the environment and therefore linger for long periods of time), very bioaccumulative (vB) chemicals (those that build up in the tissues of living organisms) and endocrine disrupting chemicals (EDCs) are not adequately addressed in the new regulations. These types of chemicals are of particular concern because once released into the environment, they cannot be recalled like products on a supermarket shelf. Instead, they will persist and build up in people, wildlife and the environment, and may reach levels that cause adverse effects. For example:

- polar bears, seals and dolphins are suffering decreased immune system function due to the immuno-toxic effects of accumulated PCBs;
- dog-whelk populations crashed in the UK and other parts of the world due to tributyltin (TBT), which is used in antifouling paints on ship hulls to prevent organisms from growing on the bottom of boats (female dog-whelks were masculinsed and unable to reproduce); and
- the populations of many birds of prey in the UK fell as a result of DDT, which caused their eggshells too thin.

WWF is not alone in wanting urgent action to stop our exposure to such hazardous chemicals:

- In May this year, 60 European human health and environmental scientists signed a declaration highlighting the urgent need to reduce human exposure to persistent and bioaccumulative chemicals and endocrine disrupting chemicals; and
- in June this year, the Royal Commission on Environmental Pollution published a report recommending to the government that "*where chemicals are found in elevated concentrations in biological fluids such as breast milk, they should be removed from the market immediately*".

POLICY CONTEXT

Current Chemical Regulations

The current system in Europe for regulating chemicals is widely acknowledged to be inadequate, failing and in need of overhaul. Among the tens of thousands of industrial chemicals marketed in Europe are around 140 that have been prioritised for evaluation and risk reduction because of their hazardous nature. Nevertheless, over the 10 years since their identification, fewer than 20 substance evaluations have been completed and fewer still have been the subject of regulatory action to limit the known threat.

Most chemicals analysed for this survey were slowly phased out in a piecemeal fashion, country by country, before they were finally subjected to widespread international bans. Experience with the world's "dirty dozen" chemicals serves to highlight the inadequate protection we get from known toxic chemicals.

Persistent Organic Pollutants (POPs) are defined as being persistent, bioaccumulative and able to travel great distances. After years of painfully slow negotiation, the POPs Convention was finally signed by more than 100 countries in Stockholm in 2001 – yet two years later, neither the UK nor the EU has ratified the treaty. Four chemicals that we analysed (DDT, PCBs, HCB and chlordane) are POPs.

This illustrates the slow pace of regulation to protect our lives and the environment from some of the world's most hazardous chemicals. Often it may take many years from the first warning signs to a chemical being adequately regulated.

New Chemical Regulation Proposals (REACH)

The EU is debating a new chemical strategy in an attempt to address legislative failures and inadequacies in the system. Under the proposals, industry will have to provide more safety data on the hazardous properties of their chemicals.

The EU review also presents an important opportunity to phase out chemicals of very high concern. The proposals could help establish a robust system of regulation that protects present and future generations from exposure to toxic chemicals. However, as they stand, the proposals aren't tough enough, as the authorisation process will fail to ensure that chemicals of very high concern – such as very persistent, very bioaccumulative (vPvB) and hormone (or endocrine) disrupting chemicals (EDCs) – are phased out when safer alternatives are available. It is important that the new EU chemical regulation requires the phase-out of such chemicals, and their mandatory substitution with safer alternatives. Phasing out is the best way effectively to reduce and eventually stop our exposure to hazardous chemicals.

If the EU strengthens the proposals as we outline above, the new legislation will yield a more progressive, precautionary and science-based chemicals policy, which will encourage industry to innovate in order to produce greener and safer products.

HEALTH EFFECTS OF CHEMICALS

For the vast majority of chemicals in use, and some of the chemicals we tested for such as the PBDE flame retardants, the data to assess their safety is hopelessly inadequate. But that is not so in the case of PCBs, which are known carcinogens and reproductive and neurological toxicants: indeed, new studies are frequently appearing in scientific literature which show that PCBs are able to produce subtle adverse effects at lower levels than previously thought.

Furthermore, ongoing developments in understanding how chemicals exert their toxic effects show that the current system of a substance by substance risk assessment does not adequately predict their risks. For instance, chemical risk assessments do not take account of the fact that:

- no chemical is ever present as a single contaminant – we are all exposed to a cocktail of chemicals, and there is therefore a potential for interaction between chemicals; and
- foetuses are exquisitely sensitive to chemicals, so that exposure in the womb can produce adverse effects at lower concentrations than would affect adults.

Taken together, this means that there are great uncertainties surrounding what might be considered a safe level of exposure to hazardous man-made chemicals, especially when they persist in the body for long periods. While WWF does not claim that exposure to a certain chemical at a certain concentration will cause a particular adverse effect in a particular individual, neither do we accept that continuing exposure, especially of foetuses, to a cocktail of hazardous chemicals can be considered "safe".

We should learn the lessons of our recent experience of chemicals such as DDT and PCBs. In WWF's view, the phase-out of the use of very persistent and very bioaccumulative chemicals

and of EDCs, and their substitution with safer alternatives, is the only way to stop the contamination of future generations of humans and wildlife.

Biomonitoring

Biomonitoring is the measurement of exogenous chemicals (those from external sources), their metabolites (breakdown products), or their reaction products in blood, urine, breast milk, fat, hair or other tissue. Biomonitoring is the preferred and most direct way of estimating the amount of a man-made chemical absorbed into the body. Biomonitoring has several uses. It can:

- determine levels of contamination in a population;
- determine trends in exposure over time, between age groups and in various countries;
- identify new chemicals of concern;
- help prioritise action on chemicals; and
- determine whether chemical control measures are effective.

Examples of the use of biomonitoring include checking the amount of alcohol in a person's breath, looking for the level of drugs in an athlete's urine, or the level of lead in a child's blood.
The total amount of chemicals present in the body is called the "body burden". The relationship of chemical body burden to human health is largely unknown, but biomonitoring is an important tool for human health scientists, epidemiologists and environmental scientists to identify the presence of chemicals and their effects on biological processes, individuals and populations. In fact, some of the most toxic chemicals have been detected by observing effects in wildlife or humans while analysing biological samples, rather than by assessment techniques in the laboratory. Some countries have begun national biomonitoring studies to determine the chemical body burden of the population and to investigate any potential links to diseases.

2 Methodology

VOLUNTEERS

Our budget allowed us to survey 155 volunteers, recruited from England, Northern Ireland, Scotland and Wales. They were 18 years of age or older, not knowingly pregnant and did not have a known medical condition that would preclude them from the survey.

Fifty (32 per cent) were male and 105 (68 per cent) female. The age range was 22 to 80 with a median of 40.5 years. Both these factors make this survey quite different from a breast-milk study, where subjects are exclusively females of a certain age range.

Among those tested were Michael Meacher MP, former Minister for the Environment, and Margot Wallström, the EU Environment Commissioner.

CHOICE OF BIOLOGICAL MATERIAL

Much thought was given to the choice of what should be analysed. Breast milk, adipose (fat) tissue and blood were all considered. WWF is keen to stress that contamination of the body is a matter of great importance to all people of all ages, so we chose to analyse chemicals in the lipid (fat) component of blood. Because comparatively little information is available on blood contamination, we felt this would be a useful addition to the scientific literature.

There is evidence that in a given body there is a standard relationship between the levels of PCBs or OCPs in adipose and blood serum or plasma (expressed as a chemical concentration per gram lipid). This also appears to be true for PCBs between human milk and blood. It is also considered likely that PBDEs follow this pattern, at least for the less brominated congeners. Therefore the levels of these chemicals detected in blood serum (with the exception of deca-BDE (BDE-209) may be extrapolated to those in adipose or milk fat.

CHOICE OF CHEMICALS

Chemicals were chosen on the basis of technical feasibility and relevance to the issue of regulating persistent, bioaccumulative and endocrine disrupting chemicals. Several chemicals are well known for both their persistence and bioaccumulation in the environment. These include:

Polychlorinated biphenyls (PCBs)

PCBs are a group of man-made chemicals first manufactured in the 1920s. They occur in 209 different forms, known as congeners. Once in the environment, PCBs do not readily break down and therefore remain for very long periods of time. PCBs can enter the air by evaporation from soil and water and can be carried long distances. They are now found all over the world – for example in Arctic snow and sea water, far away from where they were released into the environment. They enter the bodies of small organisms and fish in water and are then ingested by other animals that eat these aquatic creatures as food. PCBs especially accumulate in fish and marine mammals such as seals and whales, reaching levels that may be many millions of times higher than in water. PCB levels are highest in animals high up the food chain. Particularly high levels have been found in polar bears.

Now, nearly everyone in industrial countries has been exposed to PCBs because they are found throughout the environment, and people are likely to have detectable amounts of PCBs in their blood, fat and breast milk.

Organochlorine pesticides (OCPs) – for example, DDT, HCB and HCHs

We analysed for a range of OCPs which are typically older types of pesticides. They are characterised as very persistent and bioaccumulative, and toxic. In the UK they were generally banned from use in the 1970s, but even so, many are still widely found in the environment.

Polybrominated diphenyl ether (PBDE) flame retardants

These are a family of structurally-related chemicals used as flame retardants. They have been used widely as a safeguard against fire taking hold quickly in products. However, they have a high potential for uptake and accumulation by organisms, are now widely dispersed in the environment and have been found in human breast milk as well as in the tissues of several animal species. Both penta- and octa-commercial BDE flame retardant products have recently been banned in the EU because they were found to be widespread in humans and wildlife and because of uncertainties about their toxicity. However, they are still used elsewhere in the world, including the US.

Deca-BDE (BDE-209)

Deca-BDE is related to the less brominated penta- and octa- BDEs. Although it has been found in humans in a limited number of studies, particularly of occupationally exposed people in Sweden, we did not know whether we would find it in our "non-occupationally exposed" survey population. In the end, we discovered it in more than five per cent of volunteers.

PERSONAL AND LIFESTYLE QUESTIONNAIRE

Volunteers were asked to complete a lifestyle questionnaire concerning:

- gender;
- age;
- level of body fat (measured as body mass index);
- diet (vegan, dairy and egg eating vegetarian, fish-eating vegetarian or omnivore;
- proportion of diet that was organic;
- hours of computer usage per day;
- weight – gain, loss or stable;
- place of longest residence – northern or southern England, Scotland, Wales or Northern Ireland;
- recent purchase of consumer products likely to contain brominated flame retardants – for example, a new carpet, mattress, sofa or car; and
- the number of children carried by women and the total period spent breast feeding.

3 Results and discussion

WWF believes that this survey provides the first data on the concentrations of PCBs, organochlorine pesticides and PBDEs in human blood serum in the UK population. These results also form the most comprehensive data-set of organohalogen chemical concentrations in humans in the UK in the last 10 years at least. In addition, the survey is the first to link findings of chemical contamination to people's lifestyles.

Table 1: Summary of the Chemical Findings of the Whole Survey

	Minimum	Maximum	Median
All chemicals analysed (ng/g lipid)	46	3100	340
Total PBDE flame retardants (ng/g lipid)	0.63	420	5.6
Total PCBs (ng/g lipid)	14	670	170
Total organochlorine pesticides (ng/g lipid)	7.1	2700	130
Total HCH pesticide (ng/g lipid)	Not detected	120	15
Total DDT and metabolites (ng/g lipid)	1.3	2600	100
Number of chemicals	9	49	27

ORGANOCHLORINE PESTICIDES

The predominant pesticides detected in almost all samples were HCB, p,p'-DDE, p,p'-DDT and β-HCH. However, o,p'-DDD, o,p'-DDT, o,p'-DDE, p,p'-DDD, α-HCH and gamma (γ)-HCH (lindane) were rarely detected. None of the samples was found to contain any α-chlordane or gamma (γ)-chlordane.

DDT

In most instances the concentration p,p'-DDE (the predominant metabolite of DDT) greatly exceeded (by more than an order of magnitude) the p,p'DDT concentration, indicating that exposure to the DDT pesticide was either through the indirect route (through the diet, for example) or some time in the past. In seven cases, however, the concentration of p,p'-DDE was less than ten times the concentration of p,p'-DDT, which may indicate more recent exposure. Six samples came from volunteers who had spent time in areas where malaria is prevalent (DDT is in current use in some malarial areas), although other volunteers who had also spent time in malarial areas did not show high DDT concentrations relative to DDE. However, DDT has been banned from use in many countries and alternatives are now used. This could explain the finding.

β-HCH

The median level of β-HCH found in serum in this survey was very similar to that found in surveys of UK breast milk in 2001 and 2003, but was slightly lower than that found in a similar study in 1997/8.

POLY BROMINATED DIPHENYL ETHER (PBDE) FLAME RETARDANTS

BDEs 47, 99, 100, 153, 154 and 183 were most regularly detected in the serum samples. The major constituents of the commercial flame retardant products are for:

- penta: BDEs 47, 99, 100, 153 and 154
- octa: BDEs 153 and 183

This indicates widespread contamination of the public in the UK to both penta- and octa-BDE flame retardants.

PCBS

PCBs 153, 180 and 138 were the dominant congeners (in that order), each contributing more than 10 per cent to the median total PCB concentration. This pattern has been seen regularly in the past.

PERSONAL AND LIFESTYLE ANALYSIS

The data from the questionnaire was used to investigate potential relationships between types and levels of contamination and personal and lifestyle factors. It should be noted that there were not large numbers of people in all groups.

Because too many chemicals were detected to test for trends for each one individually, 'indicative' PCBs and PBDEs congeners were selected. If a trend was identified for PCB118, for example, this should generally be assumed to be true for other pentachlorobiphenyls.

Gender

Significant differences were noted between the sexes in the concentrations of the following chemicals:

Total HCH, PCB118 and BDEs 100 and 153.

Age

There is a correlation between the concentrations of certain chemicals and age. The following chemicals were more likely to be found at higher concentrations in older people:

Total HCH, p,p'-DDT, p,p'-DDE, and the PCB congeners 118, 153, 180 and 194.

In addition, the concentration of PCB congeners 153, 180 and 194 appears to be affected by the age and gender of adults. We investigated whether the levels of chemicals in mothers was related to the number of children they carried.

Number of children carried

If the number of children carried is taken into account, there is evidence of a slight tendency for the mothers of more children to have slightly lower concentrations in their age range. This was true for HCH, BDE153 and PCBs 118, 180 and 194. This appears to support the suggestion that women may "offload" to their children some of their body-burden of some of the more persistent chemicals during pregnancy and lactation.

Significant differences in the concentrations of certain chemicals seemed to be related to where people lived at the time of the survey and where they lived for the longest period of time.

CORRELATIONS IN CONCENTRATIONS OF DIFFERENT CHEMICAL GROUPS

The correlation between the levels of the different chemicals groups was investigated. It appears that, roughly speaking, the concentrations in humans of "old use" chemicals – such as PCBs and OCPs, which have been banned in the UK for some decades – correlate well with each other, indicating the likelihood that the volunteers are exposed through similar patterns, such as through food. However, the levels of PBDEs did not correlate with them, indicating that they are likely to reach humans through different exposure patterns, such as that identified in a recent Greenpeace study, *Consuming chemicals (2003)*. This highlighted a potential novel route of exposure when widespread and significant chemical contamination of house dust was found in a UK survey. It is possible that house dust might be a significant route of exposure to many people and might swamp other sources of exposure for some chemicals, especially brominated flame retardants. Penta-BDE was found in every one of the 100 samples analysed, and deca-BDE was the PBDE found at the highest concentrations. The levels of chemicals found in the dust (mg/g – parts per million) were far higher than the levels found in blood (ng/g – parts per billion). This contamination is likely to be due to the fact that many hazardous man-made chemicals are used in household consumer products.

TEMPORAL TRENDS AND INTERNATIONAL CONTEXT

As there is a lack of UK data on the concentration in human serum of each chemical tested in this survey, for comparison's sake data of concentrations in human adipose and milk from other published studies is included.

Chemical levels found in this survey closely match the results found in a study of breast milk from women in London and Lancaster between 2001 and 2003. This adds some weight to the suggestion that the findings of our survey are a reasonable representation of the level of contamination in the general UK population.

PCBs

UK

The levels of PCBs found in this survey closely match the results found in a study of breast milk from women in London and Lancaster between 2001 and 2003. However, they are significantly lower than those found in a previous survey of breast milk and adipose taken in Wales in 1990/91. This seems to indicate reducing levels of PCB exposure in the UK, following the ban in the 1970s.

EU

The levels found in this survey, while of the same order of magnitude (100s of ng/g lipid), are significantly lower than levels detected in human serum from Belgium and the Netherlands in four studies in 1991, 1995 and 1999. This may indicate that people's exposure to PCBs in these countries is slowly decreasing due to the ban on their use in the 1980s.

Much of the EU biomonitoring data found in the literature comes from Belgium. Belgium recently introduced a national biomonitoring programme following scandals regarding the contamination of animal feed with dioxins in 1999 and the contamination of chicken feed with PCBs in 2002.

Organochlorine pesticides

pp-DDE

UK

The levels of the DDT metabolite (p,p' DDE), were very similar to those found in a UK survey of human breast milk taken from volunteers in London and Lancaster between 2001 and 2003. However, the levels were significantly lower than those found in breast milk and adipose samples in the UK and Wales between 1990 and 1998. 'This seems to indicate reducing levels of exposure to DDT.

EU

The only comparable studies found were those conducted in Belgium between 1999 and 2002. The levels found in serum in the UK in this survey are of the same order of magnitude (100s of ng/g lipid), but significantly lower than the levels found in serum in Belgium in 1999.

HCB

UK

The levels of HCB found in serum in this survey are very similar to those found in human breast milk from London and Lancaster between 2001 and 2003. However, they are significantly lower (an order of magnitude lower) than levels of HCB found in UK breast milk in 1997. This might indicate reducing levels of HCB exposure in the UK, following the ban of the use of HCB in the 1970s.

EU

The levels of HCB found in serum in this survey are very similar to those found in the results of one survey – but an order of magnitude lower than the levels found in another survey – of serum in Belgium in 1999. Given the paucity of data, it is difficult to draw any conclusions about whether the levels of HCB contamination in the UK are currently the same or lower than levels found in Belgium.

β-HCH

UK

The median level of β-HCH found in serum in this survey was the same as that found in a survey of breast milk taken from Lancaster and London in 2001 and 2003, but slightly lower than that found in breast milk found in a UK study in 1997/8. These latter two studies found maximum levels far higher than those found in our survey. This provides some limited evidence to suggest that levels of exposure to β-HCH are decreasing.

Polybrominated Diphenyl Ether flame retardants (PBDEs)

The presence and levels of PBDEs seems to be different from the older PCBs and OCPs, suggesting different patterns of exposure. This is supported by the recent Greenpeace study. In addition, the composition of PBDE flame retardant contamination in our survey is quite different from that seen in other studies and varies significantly from location to location. This suggests that the source of exposure to PBDE flame retardants is different in the UK from that seen previously, and that it varies from location to location. The lifestyle questionnaire information is unable to identify any common exposures. These findings may be due to the UK's very stringent fire safety laws that require higher BFR content in products, or it might reflect the banning of certain PBDE flame retardants in Sweden several years ahead of the EU ban which the UK is now introducing.

Penta-BDE

EU

The concentrations of total PBDE congeners – representing the penta-BDE technical product found in this survey – are very similar to concentrations found in other European countries in recent years, and rather higher than were found in the late 1970s and early 1980s in Scandinavia.

The PBDE concentrations (and their distribution in the population) in this survey were similar to those found in the general population in Sweden in 2002. However, the range of penta-BDE levels detected in this survey was quite broad: in particular, a small number of people had high exposure to these chemicals. Both showed that approximately five per cent of the populations had more than five times the median PBDE concentration. This may indicate a "normal PBDE exposure" population and a "higher PBDE exposure" population in each country. Unfortunately, the source of the higher exposure was not evident from the personal lifestyle information collected. This might be because we did not consider exposure from house dust, which a recent Greenpeace study found may be a significant source of exposure.

Octa-BDE

The BDE congeners indicative of octa-BDE brominated flame retardant usage was found in this survey at a similar median concentration to several studies in Sweden between 1997 and 2000.

BDE-209 (deca)

We found that seven per cent (n=11) of volunteers from five towns in England – 50 per cent of English locations – contained the deca-BDE flame retardant. This indicates the UK population's continuing exposure to this chemical. Worryingly, the highest levels found in our "non-

occupationally exposed" volunteers were very similar to those of people occupationally exposed to deca-BDE in Sweden.

EU
BDE-209 was found at a higher median concentration than in Sweden, but with a similar overall range of concentrations. However, the highest concentrations found in the UK were similar to those found in samples from occupationally exposed people (workers in the flame-retarded rubber industry) in the Swedish study.

While the source of exposure of our volunteers was not evident from their personal lifestyle information, Greenpeace found that deca-BDE was the most abundant brominated flame retardant in its 2003 UK national house dust survey. It is possible that household consumer products treated with this chemical are responsible for this finding.

ASSOCIATION BETWEEN LIFESTYLE AND EXPOSURE

Generally, the lifestyle questionnaire identified few statistically significant trends in people's lifestyle habits to explain the results. This is not totally unexpected since the sample number is statistically small. Furthermore, *Consuming chemicals (2003)*, the Greenpeace report, found widespread chemical contamination of house dust, which might be a significant route of exposure to many people and might swamp other sources of exposure for some chemicals.

We are therefore still no nearer having definitive explanations for people's exposures to most chemicals. It appears to be a lottery, or even "Russian roulette", as to where, when, how and to what extent people are exposed to chemicals that persist and bioaccumulate in their bodies and interfere with their hormone system. What is abundantly clear, therefore, is the need for tighter controls of such chemicals to ensure that we minimise our risk of exposure.

However, the statistical analysis does identify the following:

- older people have higher levels of PCBs in their blood; and.
- women have lower levels of certain PCBs than do men, and the levels appear to reduce in relation to the number of children the women carried and breast-fed. These differences seem to be related to women "offloading" some of their chemical burden to their children.

The statistical analysis of our small survey is unable to show any significant difference in contamination levels between those eating vegan, vegetarian or omnivorous diets. Despite this, it is possible to reduce exposure to certain chemicals by altering one's lifestyle – especially diet. Government pesticide residue surveys have recently identified the presence of DDT and its metabolites in salmon sold for food, and PCBs are also often found in fish as well as meat. Moreover, the levels found in some fish and fish oil supplements could lead to levels of exposure above "tolerable" daily intakes identified by the World Health Organisation. This has forced the UK government to advise pregnant women to limit their intake of such products.

HEALTH EFFECTS

This survey focused purely on the presence of a range of chemicals. It is not an epidemiological survey (it does not investigate the potential adverse health effects of the presence of these chemicals) because several other studies have investigated such associations. Elevated levels of the different chemicals analysed here have been linked to a range of adverse health effects in

humans and/or wildlife such as cancers, immune deficiencies, neurological problems (behavioural disorders and depressed intelligence), depressed muscle coordination and reduced birth weight leading to increased infant mortality. Some of these effects have been observed at concentrations currently found as "background concentrations" in some EU countries including the Netherlands. For further information see WWF's *Chemicals and Health in Humans* briefing report. (WWF 2003)

While WWF does not claim that exposure to a certain chemical at a certain concentration will cause a particular adverse effect in a particular individual, due to the uncertainties concerning how chemicals can exert their toxicity, we do not accept that continuing exposure, especially of foetuses, to a cocktail of hazardous chemicals can be considered "safe".

Phasing out the use of very persistent and very bioaccumulative chemicals and of EDCs, and their substitution with safer alternatives, is the only way to stop the insidious threat of such chemicals and the contamination of future generations of humans and wildlife.

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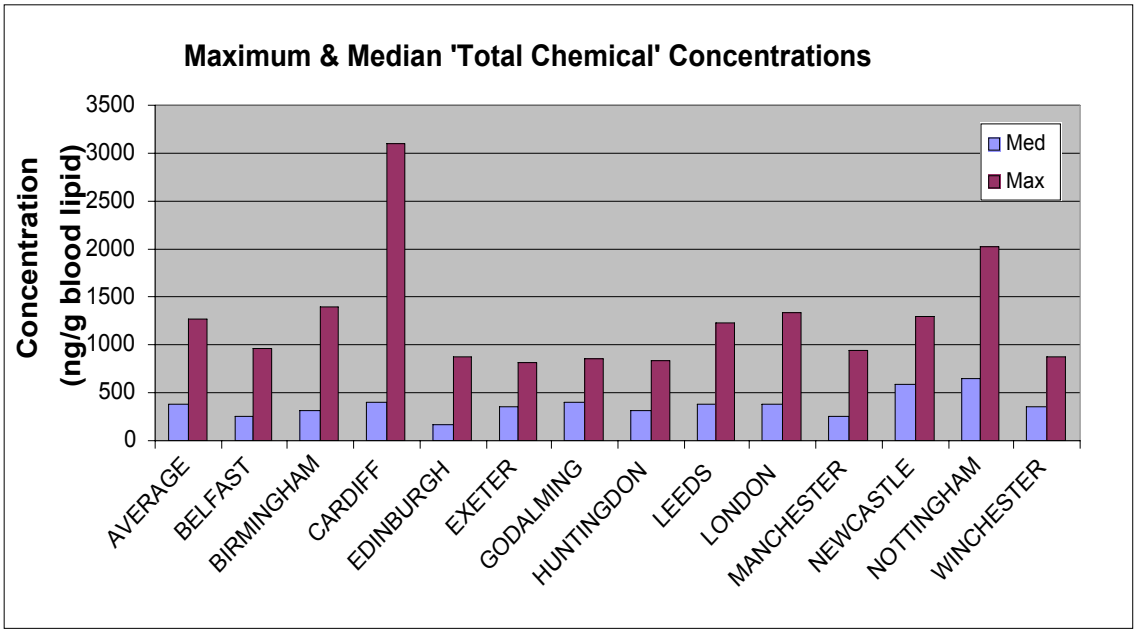
Appendix 1: Median and Maximum Chemical Concentrations by Location

Below is a brief summary of observations at each of the sample locations. Too much should not be read into these observations because the number of volunteers tested at each location is small (n=10). Neither do we claim that the volunteers are representative of each town, (in fact some people tested did not live in the town where the samples were taken). In particular, it should be noted that each group is not necessarily comparable in terms of age or gender mix.

Appendix 1 Table 1: Total Chemical Concentration (of those analysed)

	Median Concentration (ng/g lipid)	Maximum Concentration (ng/g lipid)
AVERAGE	367	1272
BELFAST	253	957
BIRMINGHAM	311	1385
CARDIFF	388	3105
EDINBURGH	176	871
EXETER	359	820
GODALMING	402	862
HUNTINGDON	311	842
LEEDS	374	1227
LONDON	369	1330
MANCHESTER	240	939
NEWCASTLE	591	1301
NOTTINGHAM	646	2024
WINCHESTER	346	869



Cardiff

A person tested in Cardiff was contaminated with the highest level of the chemicals tested.

Edinburgh

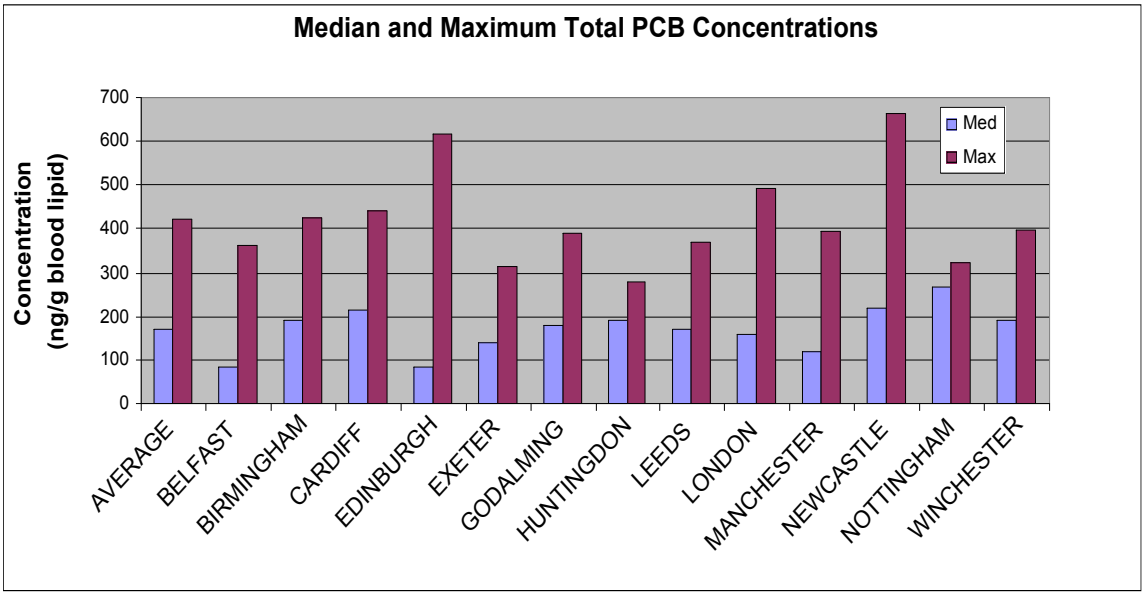
People tested in Edinburgh contained the lowest median level of total chemical contamination of the chemicals we analysed for.

Nottingham

People tested in Nottingham contained the highest median level of total chemical contamination of the chemicals we analysed for.

Appendix 1 Table 2: Total PCB Concentration

	Median Concentration (ng/g lipid)	Maximum Concentration (ng/g lipid)
AVERAGE	170	421
BELFAST	85	360
BIRMINGHAM	189	427
CARDIFF	214	441
EDINBURGH	85	618
EXETER	138	316
GODALMING	178	388
HUNTINGDON	190	277
LEEDS	173	371
LONDON	160	494
MANCHESTER	121	392
NEWCASTLE	219	665
NOTTINGHAM	266	321
WINCHESTER	190	397



Belfast

People tested in Belfast contained the lowest median level of total PCBs.

Edinburgh

People tested in Edinburgh contained the second lowest median level of total PCBs

Newcastle

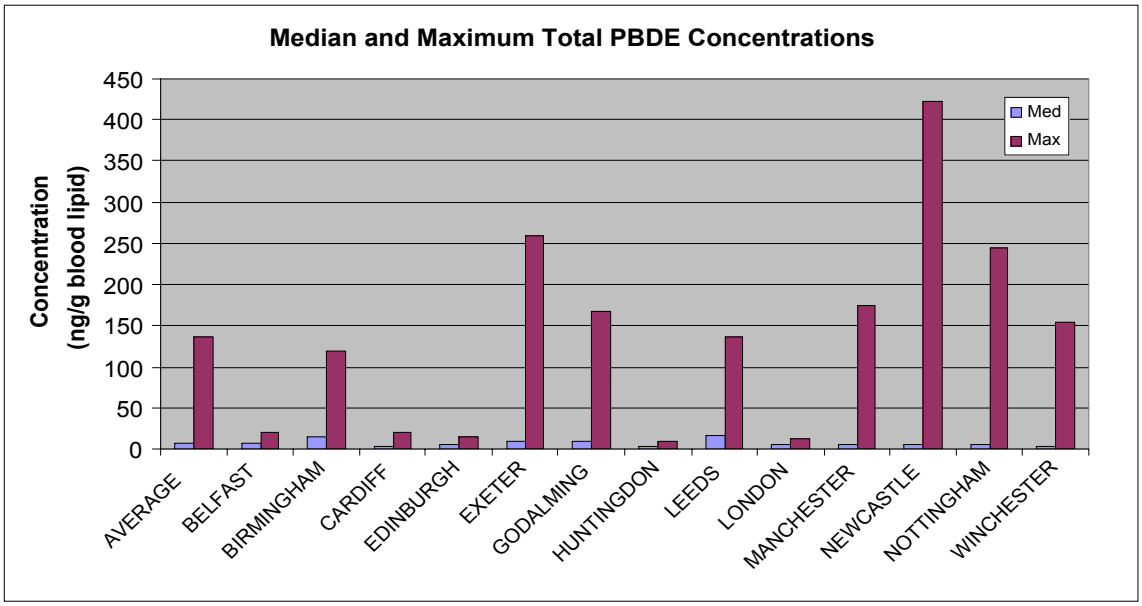
People tested in Newcastle contained the highest level of total PCB contamination.

Nottingham

People tested in Nottingham contained the highest median level of PCB contamination.

Appendix 1 Table 3: Total PBDE Concentrations

	Median Concentration (ng/g lipid)	Maximum Concentration (ng/g lipid)
AVERAGE	7	135
BELFAST	7	20
BIRMINGHAM	15	120
CARDIFF	4	21
EDINBURGH	5	16
EXETER	9	259
GODALMING	9	167
HUNTINGDON	4	9
LEEDS	17	137
LONDON	5	13
MANCHESTER	5	174
NEWCASTLE	5	422
NOTTINGHAM	6	244
WINCHESTER	4	155



Leeds

People tested in Leeds contained the highest median level of total PBDE flame retardant contamination.

Newcastle

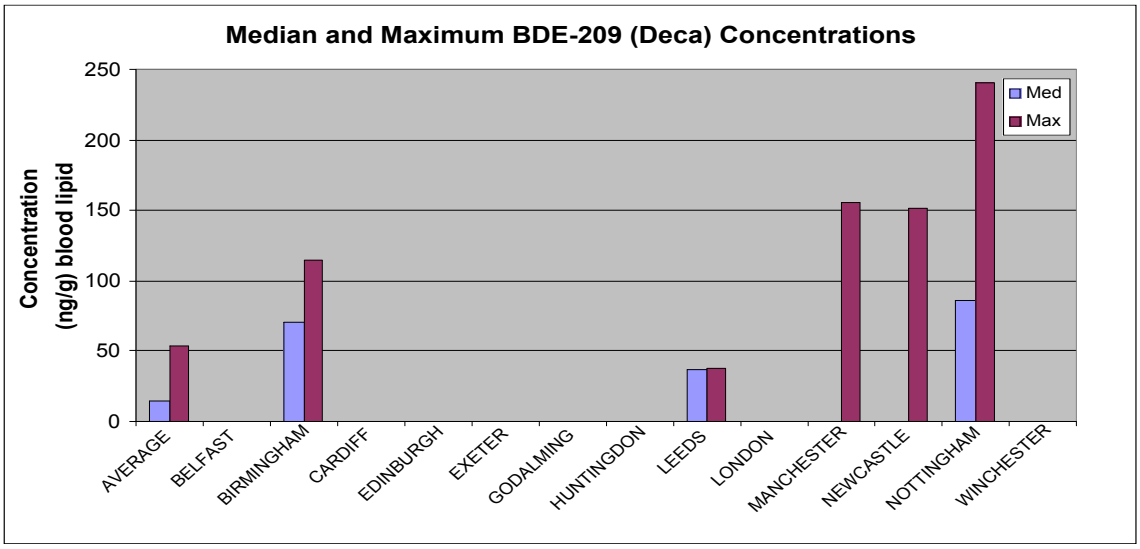
People tested in Newcastle contained the highest level of total PBDE flame retardant contamination.

Winchester

People tested in Winchester contained the lowest median level of total PBDE flame retardant contamination.

Appendix 1 Table 4: BDE-209 (deca) Concentrations

	Median Concentration (ng/g lipid)	Maximum Concentration (ng/g lipid)
AVERAGE	15	54
BELFAST	0	0
BIRMINGHAM	70	115
CARDIFF	0	0
EDINBURGH	0	0
EXETER	0	0
GODALMING	0	0
HUNTINGDON	0	0
LEEDS	37	37
LONDON	0	0
MANCHESTER	0	155
NEWCASTLE	0	151
NOTTINGHAM	86	241
WINCHESTER	0	0



Manchester

People tested in Manchester contained the highest median level of deca-BDE flame retardant contamination.

Nottingham

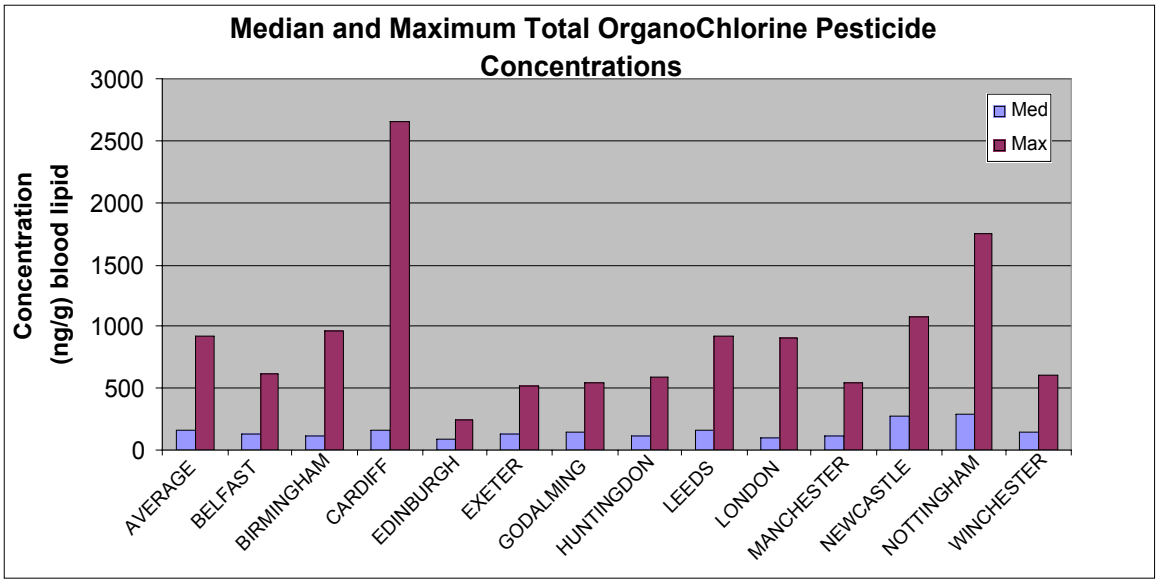
People tested in Nottingham contained the highest level of deca-BDE flame retardant contamination.

Birmingham

More people tested in Birmingham were contaminated with deca-BDE than in any other location. Four people were contaminated and they contained the second highest median level of deca- contamination.

Appendix 1 Table 5: Total Organochlorine Pesticide Concentrations

	Medium Concentration (ng/g lipid)	Maximum Concentration (ng/g lipid)
AVERAGE	152	917
BELFAST	136	620
BIRMINGHAM	119	961
CARDIFF	161	2654
EDINBURGH	86	247
EXETER	134	513
GODALMING	149	540
HUNTINGDON	110	583
LEEDS	161	917
LONDON	103	903
MANCHESTER	120	541
NEWCASTLE	272	1078
NOTTINGHAM	286	1754
WINCHESTER	143	606



Cardiff

A person tested in Cardiff contained the highest level of organochlorine pesticide contamination.

Edinburgh

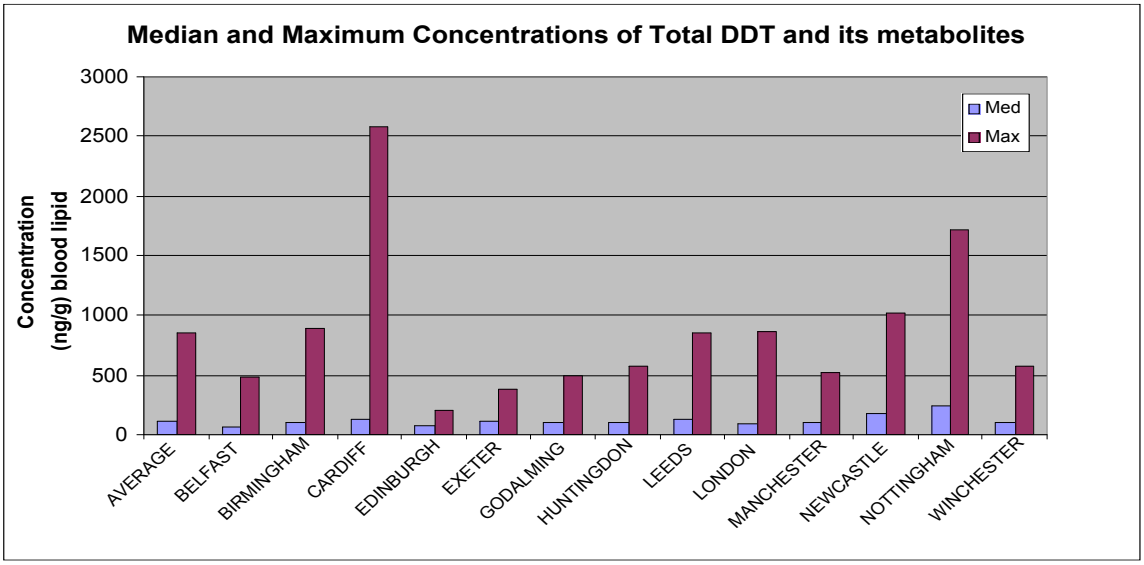
People tested in Edinburgh contained the lowest median level of the organochlorine pesticides we tested for.

Nottingham

People tested in Nottingham contained the highest median level of organochlorine pesticides we tested for.

Appendix 1 Table 6: Concentrations of Total DDT and Metabolites

	Median Concentration (ng/g lipid)	Maximum Concentration (ng/g lipid)
AVERAGE	116	858
BELFAST	59	485
BIRMINGHAM	102	892
CARDIFF	132	2579
EDINBURGH	74	209
EXETER	112	384
GODALMING	103	496
HUNTINGDON	96	578
LEEDS	123	848
LONDON	93	860
MANCHESTER	97	519
NEWCASTLE	172	1019
NOTTINGHAM	243	1715
WINCHESTER	105	570



Cardiff

A person tested in Cardiff had the highest level of total DDT and metabolites.

Nottingham

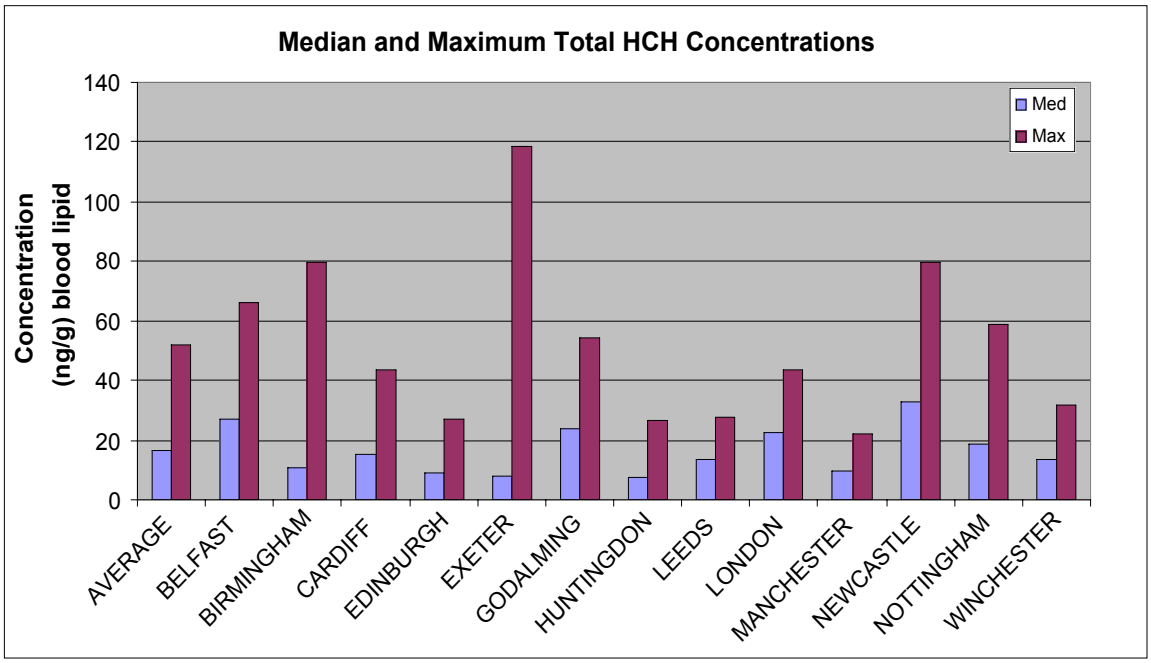
People tested in Nottingham contained the highest median level of total DDT and metabolites.

Belfast

People tested in Belfast contained the lowest median level of total DDT and metabolites.

Appendix 1 Table 7: Concentrations Total HCH Pesticides (including lindane).

	Median Concentration (ng/g lipid)	Maximum Concentration (ng/g lipid)
AVERAGE	16	52
BELFAST	27	66
BIRMINGHAM	11	79
CARDIFF	15	44
EDINBURGH	9	27
EXETER	8	118
GODALMING	24	54
HUNTINGDON	7	27
LEEDS	13	28
LONDON	22	43
MANCHESTER	10	22
NEWCASTLE	33	80
NOTTINGHAM	18	59
WINCHESTER	13	32



Exeter

A person tested in Exeter contained the highest level of total HCH pesticides (including lindane).

Newcastle

People tested in Newcastle contained the highest median level of total HCH pesticides.

Huntingdon

People tested in Huntingdon contained the lowest median level of total HCH pesticides.

Appendix 2: Summary of Results by Location

Belfast

People tested in Belfast contained:

- the highest median concentration of the pesticide HCB;
- the second highest median levels of total HCH pesticides (including lindane) after Newcastle;
- the lowest median level of total PCBs; and
- the lowest median level of total DDT and metabolites.

Birmingham

More people tested in Birmingham were contaminated with deca-BDE than in any other location. Four people were contaminated. They also contained the second highest median level of contamination with deca-BDE.

Cardiff

A single person tested in Cardiff had:

- the highest level of the chemicals we tested for;
- the highest level of the organochlorine pesticides we tested for; and
- the highest level of total DDT and metabolites.

Edinburgh

People tested in Edinburgh contained:

- the lowest median level of total chemical contamination of the chemicals we analysed for;
- the lowest median level of organochlorine pesticides we tested for; and
- the second lowest median level of total PCBs.

Exeter

A person tested in Exeter was the most contaminated with total HCH pesticides (including lindane).

GODALMING, Surrey (WWF-UK headquarters)

A person tested in Godalming contained the highest number of chemicals detected (49) and people tested in Godalming contained the highest median number of chemicals.

Huntingdon

People tested in Huntingdon contained the lowest median level of total HCH-pesticides (including lindane).

Leeds

People tested in Leeds contained the highest median level of total PBDE flame retardants.

London

A person tested in London contained the third highest level of total PCB contamination.

Manchester

People tested in Manchester contained the highest median level of deca-BDE flame retardant.

Newcastle

People tested in Newcastle contained:

- the highest median level of total HCH pesticides (including lindane);

Individuals tested in Newcastle contained

- the highest level of total PBDE flame retardants; and
- the highest level of PCBs.

Nottingham

People tested in Nottingham contained:

- the highest median level of total chemical contamination of the chemicals we looked for;
- the highest median level of PCBs;
- the highest median level of organochlorine pesticides we tested for and
- the highest median level of DDT and its metabolites.

In addition, the person containing the highest level of deca-BDE flame retardant was tested in Nottingham.

Winchester

People tested in Winchester had the lowest median level of total PBDE flame retardants.

Appendix 3: Lancaster University Analytical Report

A study of organohalogen chemicals in human blood from around the United Kingdom

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November 2003

Executive Summary

OBJECTIVES OF THE STUDY:

1 To determine the concentrations of a range of organohalogen chemicals in human blood in the UK

2 To investigate links between chemical concentrations and life history or lifestyle parameters.

Blood samples were taken from 154 volunteers at 13 locations in the UK in March, June and July 2003. One sample was also taken in Brussels, Belgium, in July 2003. The serum from each sample was analysed for 45 PCBs, 12 organochlorine pesticides and 21 PBDEs.

The total PCB concentrations ranged from 14 to 670 ng/g lipid, with a median of 100 ng/g lipid. PCBs 153, 180 and 138 (in that order) were the dominant congeners, each contributing more than 10% to the median total PCB concentration, as has regularly been seen in the past.

HCB, p,p'-DDE, p,p'-DDT and β-HCH were the most commonly detected organochlorine pesticides and were dominant in almost all samples. The range of concentrations found for HCB was relatively small, whilst the range of concentrations found for p,p'-DDE was the largest of any chemical analysed.

BDEs 47, 99, 100, 153, 154 and 183 were most regularly detected in the serum samples. It should be noted that BDEs 47, 99, 100, 153 and 154 are present as the major constituents of the pentaBDE commercial flame retardant product, whereas BDEs 153 and 183 are the dominant constituents of the octaBDE commercial flame retardant product.

There was a correlation between PCB concentration and age, with a significant difference in gradient between males and females (older females having lower concentrations than older males, but older people, in general, having higher concentrations that younger people). We also found that the UK population in 2003 had similar PBDE concentrations, and a similar concentration distribution, to that found in the general population in Sweden in 2002, with approximately 5% of the population having more than five times the median PBDE concentration. This is somewhat surprising because of the history of PBDE production and use in the UK compared to Sweden.

This study was, we believe, the first survey of blood serum concentrations of PCBs, organochlorine pesticides and PBDEs in the UK. The product of the study is a large and valuable data-set which may be used as an indicator of human exposure to this range of chemicals in the UK, for comparison to other regions, and may be used to determine patterns of exposure to certain chemical groups dependent on lifestyle.

Background

A study was performed to investigate the concentrations of a range of organohalogen chemicals in human blood taken from volunteers in different parts of the UK in March, June and July 2003.

The objectives of the study were:

1) To determine the concentrations of a range of organohalogen chemicals in human blood in the UK
2) To investigate links between chemical concentrations and life history or lifestyle parameters.

Methods

A pilot study was performed to determine the best sampling and analytical methods and media for this project, and was the subject of a previous report. The choice of serum as the matrix for analysis, the sample treatment in the field and laboratory and the use of the chosen sampling devices (not to introduce interference or contamination) were verified as suitable for this project in the pilot study.

SAMPLING

Blood samples were taken by trained medical professionals from 154 volunteers at 13 locations in the UK in March, June and July 2003. One sample was also taken in Brussels, Belgium, in July 2003. 40 ml whole blood was taken from each volunteer. The locations were (the code used is shown in brackets): Belfast (BEL), Birmingham (BIR), Brussels (BRUSS), Cardiff (CAR), Edinburgh (EDIN), Exeter (EXE), Godalming (Surrey) (WWF), Huntingdon (HUN), Leeds (LEE), London (LON), Manchester (MAN), Newcastle (NEW), Nottingham (NOT) and Winchester (WIN).

The BD Vacutainer® system was used to take all samples, and blood samples were taken into serum tubes (with clotting agent and polymer separator). Samples were mixed by inverting the tubes immediately after blood collection, and, after an appropriate time for coagulation to occur, centrifuged at 3000 rpm for 10 min, at the sampling site, to separate the serum from the blood cells.

As soon as possible after separation the serum samples were frozen to –20°C and posted to Lancaster University. All samples were still frozen upon receipt at Lancaster University, where they were defrosted and the serum transferred to clean glass containers before being frozen to –20 °C until analysis.

ANALYSIS

Sample analysis was performed at Lancaster University using methods based on those developed by Hovander *et al.* (2000), briefly entailing modification of the sample with HCl and Isopropanol, followed by extraction with a hexane:MTBE mixture. Samples were then cleaned using concentrated sulphuric acid, followed by gel permeation chromatography, before analysis

by GC-MS.

Samples were analysed for 45 PCBs (PCBs 18, 22, 28, 31, 41/64, 44, 49, 52, 54, 60/56, 70, 74, 87, 90/101, 95, 99, 104, 105, 110, 114, 118, 123, 138, 141, 149, 151, 153, 155, 156, 157, 158, 167, 170, 174, 180, 183, 187, 188, 189, 194, 199 and 203) using a GC-quadrupole MS system (Finnigan TRACE) in EI mode. 12 organochlorine pesticides (α-chlordane, γ-chlordane, HCB, o,p'-DDD, p,p'-DDD, o,p'-DDE, p,p'-DDE, o,p'-DDT, p,p'-DDT, α-HCH, β-HCH and γ-HCH) were also analysed using the Finnigan TRACE GC-MS in EI mode. 21 PBDEs (BDEs 17, 28, 32, 35, 37, 47, 49, 71, 75, 77, 85, 99, 100, 119, 138, 153, 154, 166, 181, 183 and 190) were analysed using a GC-quadrupole MS system (Fisons MD800 or Finnigan TRACE) in NCI mode, using ammonia as the reagent gas. BDE209 was analysed using a GC-high resolution MS (Micromass Autospec Ultima) in EI mode, using a resolution of at least 10,000.

QUALITY CONTROL

All samples were spiked with ^{13}C-labelled recovery standards before extraction. The standards used included ^{13}C-labelled PCBs 28, 52, 101, 138, 153, 180 and 209 and ^{13}C-labelled BDE209. Concentrations were not corrected for the recoveries of these standards, but the recoveries were monitored to assess the effectiveness of the analytical methods. ^{13}C-labelled PCB recoveries averaged 79-84 % and ^{13}C-labelled BDE209 recovery averaged 107%.

Laboratory blanks, consisting of purified water, were analysed at a ratio of 1 for each ten samples analysed, and blank concentrations were subtracted from the concentrations found in each sample before application of the detection limit.

Control samples, consisting of the BD Vacutainer® tubes used for sample collection were prepared using purified water. After drawing the water into the tube the water was decanted and treated as a normal sample for analysis. 1 control sample was prepared for each 25 blood samples analysed.

One in-house reference material, consisting of homogenised seal blubber, was analysed for each 25 blood samples. Concentrations of selected analytes were compared to a control chart based on previous analyses of the reference material, and did not exceed the specified upper or lower action limit concentrations. (The seal blubber used to prepare the reference material was taken from a stranded seal which had been found dead on a UK beach, and collected by a sea mammal research centre in the UK as part of their ongoing stranded sea mammal collection programme.

DETECTION LIMITS

For chemicals detected in the blank samples the method detection limit was defined as three times the standard deviation of the blank value. In the absence of detectable concentrations in the blank samples the method detection limit was defined as the instrument detection limit (the amount of chemical required to achieve a signal to noise ratio of at least 10).

Results

The organohalogen chemicals concentrations found in the serum samples are summarised in Tables 1 and 2. The entire PCB data set is shown in Appendix 1 Tables 1-22 and the entire pesticide and PBDE data set is shown in Appendix 1 Tables 23-44. There is evidence, as is usual for pollutant concentrations in humans, that the concentrations of chemicals in the blood samples do not conform to a Normal distribution, so the data has been summarised using the median, ranges and quartile values (rather than using the mean and standard deviation, which require that the data conform to a Normal distribution). Note that values below the detection limit were excluded when calculating the minimum, median and quartile values and that quartile values were not calculated for chemicals found in less than 10 samples. The data is summarised according to sampling location in Appendix 2.

Table 1 – Summary of detected PCB concentrations found in blood serum and plasma (N = Number of detected values)

PCB	Minimum ng/g lipid	Maximum ng/g lipid	Median ng/g lipid	25th Percentile *Only for N>10*	75th Percentile *Only for N>10*	N (out of 155)
18	0.52	2.9	1.1	0.74	1.6	15
22	0.44	4.8	1.1	0.72	2.1	19
28	0.85	14	2.3	1.7	3.2	129
31	0.50	10	1.2	0.82	2.8	56
41/64	0.51	21	1.5	0.98	4.2	20
44	0.30	1.6	0.53	0.44	0.73	28
49	0.41	3.9	1.4	0.91	2.4	30
52	0.26	4.7	0.57	0.49	0.7	68
54			0.51			1
70	0.30	19	0.82	0.55	1.9	55
74	0.35	27	4.4	2.7	8.3	145
87	0.33	57	2.7	0.59	8.4	12
90/101	2.1	9.0	3.5	2.8	5.7	13
95	1.4	8.5	2.0	1.8	3.1	17
99	0.67	17	3.2	2.2	5.1	154
105	0.36	6.5	1.5	0.88	2.5	115
110	0.45	2.2	0.99			6
114	0.31	9.2	0.82	0.68	1.1	78
118	0.52	29	6.2	3.6	11	154
123	0.36	0.66	0.58			4
138	5.7	110	28	20	48	150
141	2.0	3.6	2.6			5
149	4.3	12	10			8
151	1.9	6.7	4.6	2.8	5.3	10
153	8.7	170	42	28	71	153
156	0.59	18	4.8	3.1	7.7	152
157	0.37	3.8	1.3	0.83	1.8	122

158	0.27	5.1	0.77	0.50	1.2	69
167	0.39	6.1	1.9	1.1	3.1	135
170	1.4	61	13	8.6	21	151
174	1.1	6.8	1.7			9
180	4.3	160	33	21	53	152
183	1.4	16	4.4	3.0	5.9	105
187	0.66	57	7.9	5.2	12	130
189	0.48	1.2	0.78			8
194	0.42	39	5.2	3.0	8.0	148
203	0.79	37	6.1	5.1	8.6	71

Table 2 – Summary of detected organochlorine pesticide, PBDE and total chemical group concentrations found in blood serum and plasma (N = Number of detected values)

Pesticide	Minimum ng/g lipid	Maximum ng/g lipid	Median ng/g lipid	25th Percentile *Only for N>10*	75th Percentile *Only for N>10*	N (out of 155)
HCB	5.4	72	14	10	24	109
o,p'-DDD	1.6	49	7.9	7.2	21	13
o,p'-DDT	0.73	8.4	1.3	0.93	1.7	19
o,p'-DDE	0.92	3.9	1.5			7
p,p'-DDD	0.49	20	1.3			9
p,p'-DDE	15	2600	100	59	210	154
p,p'-DDT	0.80	73	3.3	1.9	6.0	141
α-HCH	0.98	23	4.1	1.7	6.3	23
β-HCH	1.9	80	12	7.2	22	151
γ-HCH	6.8	110	18	13	23	17
PBDE						
17	0.20	0.30	0.25			2
28	0.16	9.8	0.31	0.25	0.46	42
32	0.21	11	0.30	0.25	0.67	16
35	0.41	1.1	0.98			3
37	0.17	0.42	0.33			4
47	0.40	180	1.3	0.76	2.3	105
49	0.27	1.7	0.58			3
66	0.35	3.2	0.83	0.71	1.1	23
71			0.26			1
75			0.30			1
77	0.64	17	5.7			6
85	0.92	5.3	2.1			4
99	0.36	150	1.2	0.64	2.2	63
100	0.25	390	0.82	0.59	1.1	143
119	0.43	1.4	0.67			5
138	0.47	1.6	0.82			3
153	0.39	87	1.7	1.2	2.4	153
154	0.17	4.4	0.68	0.49	0.90	133
166	0.32	2.9	1.6			2
181			0.89			1
183	0.19	1.8	0.59	0.42	0.76	85
209	35	240	83	37	140	11
All chemicals analysed	**46**	**3100**	**340**	**220**	**590**	
Total PBDE	**0.63**	**420**	**5.6**	**3.4**	**8.2**	
Total PCB	**14**	**670**	**170**	**100**	**270**	
All OC pesticides analysed	**7.1**	**2700**	**130**	**83**	**270**	
Total HCH	**nd**	**120**	**15**	**7.7**	**29**	
Total DDT & metabolites	**1.3**	**2600**	**100**	**61**	**210**	
No. of chemicals found	**9**	**49**	**27**	**23**	**30**	

nd = not detected

Discussion

We believe these are the first concentrations of PCBs, organochlorine pesticides and PBDEs reported from a survey of human blood serum in the UK, and form the biggest data-set of concentrations of organohalogen chemicals in humans in the UK in the last 10 years.

It has been reported in the past that PCB, organochlorine pesticides and PCDD/F concentrations are similar, when expressed on a lipid normalised basis (chemical concentration per gramme lipid in any tissue), in human adipose and blood serum or plasma (Schecter *et al.*, 1991; Mussalo-Rauhamaa *et al.*, 1991; Wingfors *et al.*, 1998) and also, for PCBs, between human milk and blood (Schecter *et al.*, 1998). Although there is no evidence currently available we believe that it is very likely that PBDEs also follow this pattern, at least for the less brominated congeners, because they have similar physical-chemical properties to the other chemicals studied. Since there is a lack of human serum concentration data for the UK for all of the chemicals tested in this study, we have, therefore, included published concentrations in human adipose and milk in the discussion. We have less confidence in making the assumption that blood and other tissue concentrations will be similar on a lipid basis for BDE209, due to it's very high octanol-water partition coefficient, low lipid solubility and high molecular weight, but for this chemical there are data for blood from other countries available of which we have made use.

PCBS

The total PCB concentrations (defined as the sum of all congeners detected) ranged from 14 to 670 ng/g lipid, with a median of 100 ng/g lipid. PCBs 153, 180 and 138 (in that order) were the dominant congeners, each contributing more than 10% to the median total PCB concentration as has regularly been seen in the past.

Some previously published concentrations of PCBs in the western European population are summarised in Table 3. The main criterion for inclusion of data in the table was the comparability of data presentation (i.e. range and median or geometric mean, as the arithmetic mean concentration alone is little use for comparative purposes). The surveys of PCBs in the UK population were the only ones found which had been previously published. The median total PCB concentration is approximately a factor of 5 lower than reported in adipose samples taken from corpses (predominantly male) in Wales in 1990/91 (Duarte-Davidson *et al.*, 1994a) and approximately a factor of 2.5 lower than in milk-fat samples from Wales in the same period (Duarte-Davidson *et al.*, 1994b). The range of total PCB concentrations is somewhat broader in relation to the median in this study than was found in either of the Welsh studies. Assuming that lipid based concentrations for different tissues are comparable (as discussed above) the decrease in concentration seen between 1990/1 and this study is likely to be due to a reduction in environmental (and particularly human foodstuffs) concentrations over the past decade. The median and range of values found in this study are, however, very close to the geometric mean and range of concentrations found in human breast milk-fat collected in London and Lancaster between December 2001 and January 2003 (Kalantzi *et al.*, 2003). The median total PCB concentration found in this study is 2 to 3 times lower than found in studies of human serum (from women aged 50-65 in Koppen *et al.*, 2002 and from serum of pregnant women in Covaci *et al.*, 2002) and adipose from a varied population (Chu *et al.*, 2003) in other European studies (between 1991 and 2002) quoted in

Table 3. The cause of the difference in PCB concentrations between this study and those in Europe cannot be determined, but may be due to the different characteristics of the sample population in each study.

Table 3 – PCB concentrations previously found in samples from the European population

Region	Year	Sample Type	Total PCB concentration ng/g lipid *median (range)*	Reference
Wales	1990-1	Adipose	850 (260-2600)	Duarte-Davidson *et al.*, 1994a
Wales	1990-1	Milk	440 (140-1700)	Duarte-Davidson *et al.*, 1994b
London & Lancaster	2001-3	Milk	120* (2.6-530)	Kalantzi *et al.*, 2003
UK	2003	Serum	170 (14-670)	This study
Belgium	1999	Serum	350 (110-1000)†	Covaci *et al.*, 2002
Belgium	1999	Serum	530 (450-680**)	Koppen *et al.*, 2002
Belgium	2002	Adipose	480‡ (190-1100)	Chu *et al.*, 2003
Netherlands	1991	Serum	550†‡	from Covaci *et al.*, 2002
Netherlands	1995	Serum	450†‡	from Covaci *et al.*, 2002

* = Geometric mean;
‡ = Arithmetic mean;
† = Calculated assuming 0.5% lipid in blood;
** = 95th Percentiles

ORGANOCHLORINE PESTICIDES

α-Chlordane and γ-chlordane were not detected in any of the samples analysed, and of the other organochlorine pesticides analysed HCB, p,p'-DDE, p,p'-DDT and β-HCH were most commonly detected and were dominant in almost all samples.

The range of concentrations found for HCB was relatively small, whilst the range of concentrations found for p,p'-DDE was the largest of any chemical analysed. The median concentrations of HCB, p,p'-DDE, p,p'-DDT and β-HCH were very close to geometric mean concentrations found in milk samples taken from London and Lancaster between 2001 and 2003 (Kalantzi *et al.*, 2003) although the ranges of values found in this study are slightly narrower than found by Kalantzi *et al.*. Organochlorine pesticide concentrations found in other studies of western Europeans are summarised in Table 4. It can be seen that p,p'-DDE concentrations found in this study are between a factor of 1.1 and 9.9 lower than found in other studies conducted since 1990, and in particular that concentrations are somewhat lower than recently found in Belgium. The cause of the difference in concentrations between this study and the other studies quoted in Table 4 cannot be determined, but may be due to the different characteristics of the sample population in each study, and in the UK may be due to a reduction in concentration with time. HCB concentrations in this study were rather lower than found in the UK in 1997 and β-HCH concentrations in this study were rather lower than found either in the UK in 1997 or in Belgium in 1999.

The pesticides o,p'-DDD, o,p'-DDT, o,p'-DDE, p,p'-DDD, __HCH and __HCH were detected in few samples. In all cases the p,p'-DDE concentration greatly exceeded (by more than an order of magnitide) the p,p'DDT concentration, indicating that exposure to the DDT pesticide was either through the indirect route (e.g. through the diet) or some time in the past. In seven cases, however, the concentration of p,p'-DDE was less than ten times the concentration of

p,p'-DDT, which may indicate more recent exposure. Six out of these samples came from volunteers who had spent time in areas where malaria is prevalent (DDT is in current use in some malarial areas), although other volunteers who had also spent time in malarial areas did not show high DDT concentrations relative to DDE.

Table 4 – Organochlorine pesticide concentrations previously found in samples from the European population

Region	Year	Sample Type	Concentration (ng/g lipid) Median (Range)			Reference
			p,p'DDE	HCB	β-HCH	
Wales	1990-1	Adipose	990 (110-6400)			Duarte-Davidson *et al.*, 1994a
Wales	1990-1	Milk	310 (35-6000)			Duarte-Davidson *et al.*, 1994b
UK	1997-8	Milk	280 (<60-4000)	250 (<12-330)	50 (<8-750)	Harris *et al.*, 1999
London & Lancaster	2001-3	Milk	110* (1.7-1600)	14* (0.3-180)	16* (0.1-1500)	Kalantzi *et al.*, 2003
UK	2003	Serum	100 (15-2600)	14 (5.4-72)	12 (1.9-80)	This study
Belgium	1999	Serum	310 (110-4100)†	36 (12-100)†		Covaci *et al.*, 2002
Belgium	1999	Serum	870 (730-1200**)	110 (90-132**)		Koppen *et al.*, 2002
Belgium	2002	Adipose	480‡ (84-1800)			Chu *et al.*, 2003

= Geometric mean;
‡ = Arithmetic mean;
† = Calculated assuming 0.5% lipid in blood;
** = 95th Percentiles

PBDES

The six PBDE congeners most regularly detected in the serum samples were BDEs 47, 99, 100, 153, 154 and 183. It should be noted that BDEs 47, 99, 100, 153 and 154 are present as the major constituents of the pentaBDE commercial flame retardant product, whereas BDEs 153 and 183 are the dominant constituents of the octaBDE commercial flame retardant product. The median concentration of BDE153 was higher than the median concentration of BDE47, which is unusual compared to other published data on humans, but was also found in Belgian adipose samples by Covaci *et al.*, 2002 and in serum from some occupational groups in Sweden between 1997 and 2000 (Jakobsson *et al.*, 2003). A similar pattern was found in milk-fat samples from Lancaster in the UK taken between 2001 and 2003 (Kalantzi, personal communication). The average PBDE congener pattern, calculated as the mean of the percentage contribution of BDEs 47, 99, 100, 153, 154 and 183 to the sum of those six congeners in each sample, is shown in Figure 1. Approximately 20% of the individual samples had congener patterns similar to those seen in other human studies (BDE47 dominating, followed by BDEs 153, 99, 100 and 154, usually in that order) and these samples were unevenly distributed through the sample locations (the locations and proportion of samples with this pattern were: Huntingdon (0%), Cardiff (8%), Nottingham (9%), Winchester (10%), London (11%), Edinburgh (17%), Manchester (18%), Newcastle (22%), Exeter (27%), Belfast (30%), Godalming (33%), Birmingham (50%) and Leeds (60%)). This may indicate groups of people who are exposed to PBDEs from different sources, or at different times, but none of the personal information currently available appeared to explain the difference in PBDE patterns noted. It is possible that, since new pentaBDE product usage has now ceased in the UK, metabolism of certain congeners (dependant on the bromination pattern and bromination level) is now having a marked impact on the pattern seen in certain

people whose exposure may have been reduced in recent years, whilst others may still be exposed to a relatively 'fresh' intake of PBDEs (dominated by BDE47) due to their living or working environment. It is also possible that, with the recent phasing out of the pentaBDE flame retardant product (the major source of BDEs 47, 99, 100, 153 and 154) exposure to the octaBDE product (the major constituents of which are BDEs 183 and 153) may be a more important source of BDEs for some individuals. However, these hypotheses cannot be tested within this project. The values and the distribution of the concentrations of the sum of BDEs 47, 99, 100, 153 and 154 were very similar to that found in Swedish blood samples (from the general population) by van Bavel et al. (2002), with approximately 5% of samples from this UK study and the Swedish study having concentrations higher than 30 ng/g lipid (more than five times the median value). This may indicate that there is a 'normal PBDE exposure' population and a 'higher PBDE exposure' population within each country, but the source of the higher exposure is not evident from the personal information provided.

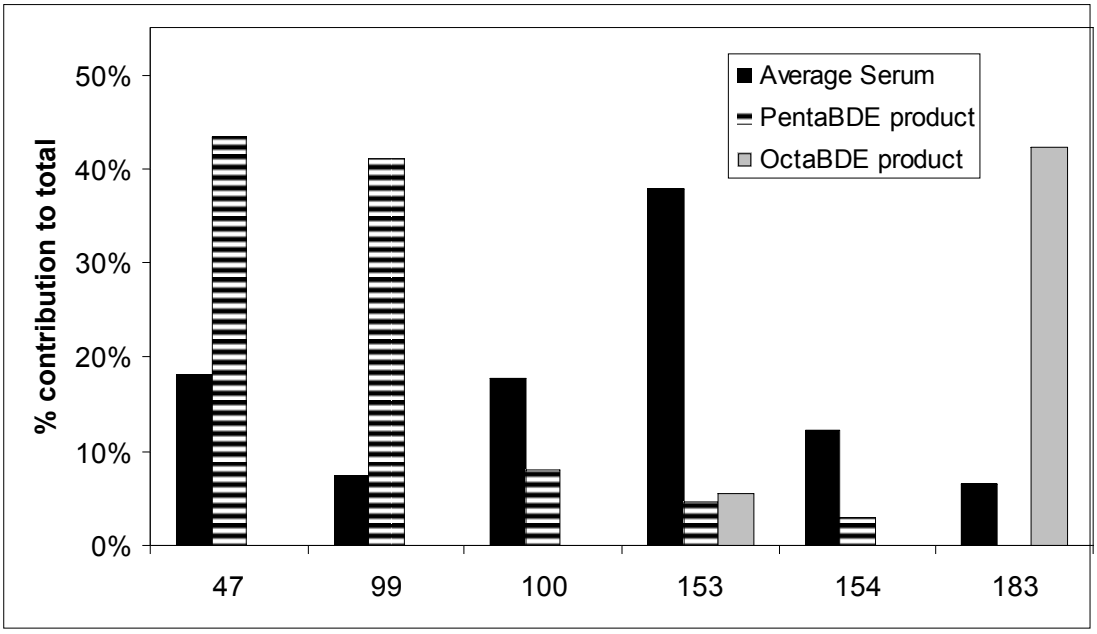

Figure 1 – PBDE congener profile for serum and the pentaBDE (Huber and Ballschmitter, 2001) and octaBDE (EU, 2003) products (NB. individual octaBDE and nonaBDE congeners also make large contributions to the octaBDE product)

BDE209 was found in 11 samples, ranging in concentration from close to the detection limit (3 samples) to a maximum of 240 ng/g lipid. (Note that the concentration in ng/g lipid should be divided by 0.958 to give the concentration expressed in pmol/g lipid, used in some publications). The samples with detectable levels of BDE209 came from Newcastle (1 sample), Birmingham (4 samples), Nottingham (3 samples), Manchester (1 sample) and Leeds (2 samples).

Table 5 – PBDE concentrations previously found in samples from the European population

Region	Year	Sample Type	Concentration (ng/g lipid) Median (Range)			Reference
			ΣBDE	BDE183	BDE209	
London & Lancaster	2001-3	Milk	6.6* (0.3-69)			Kalantzi *et al.*, 2002
UK	2003	Serum	4.6 (0.52-420)	0.59 (0.19-1.8)[a]	83 (35-240)[b]	This Study
Sweden	2001-2	Blood	4.9‡[c]			Van Bavel *et al.*, 2002
Belgium	2000	Adipose	4.8‡			Covaci *et al.*, 2002
Finland	1994-8	Milk	2.1‡			Strandman *et al.*, 2000
Germany	1985 1990 1995 1999	Blood	3.1‡[d] 3.6‡[d] 3.7‡[d] 3.9‡[d]			Schröter-Kermani *et al.*, 2000
Norway	1977 1986 1995 1999	Serum	0.44‡ 1.1‡ 3.1‡ 3.1‡			Thomsen *et al.*, 2002
Sweden	1972 1980 1990 2000	Milk	0.07‡ 0.45‡ 1.2‡ 2.6‡			Summarised in Sjödin *et al.*, 2003
Sweden	1997 1998 2000 2000 1999 1997 1997 2000	Serum		11 (3.0-25) ED <0.38 (<0.38-1.6) CB <0.48 (<0.48-1.1) RM <1.9 RW 1.2 (0.23-6.1) CT 0.23 (<0.02-1.3) C 0.15 (0.029-0.37) CL <0.38 AB	4.8 (<0.29-9.5) ED 2.3 (<0.96-5.6) CB 28 (1.2-140) RM 34 (6.7-280) RW 1.5 (<0.96-6.8) CT <0.67 (<0.67-7.7) C <0.67 (<0.67-3.7) CL 2.4 (0.92-9.3) AB	Jakobsson *et al.*, 2003

^ = sum of BDEs 47, 99, 100, 153 and 154 unless otherwise indicated;
= Geometric mean;
‡ = Arithmetic mean;
† = Calculated assuming 0.5% lipid in blood;
** = 95th Percentiles;
[a] = only detected values used (N = 85);
[b] = only detected values used (N = 11);
[c] = sum of BDE47, 99, 153;
[d] = BDE47 only;
ED = Electronics dismantlers;
CB = Circuit board recycling workers;
RM = Rubber mixers;
RW = Rubber wire production;
CT = Computer technicians;
C = Clerks;
CL = Cleaners;
AB = Abattoir workers

PBDE concentrations in humans in western Europe have been summarised in Table 5. It can be seen that the total PBDE (congeners 47, 99, 100, 153, 154 – representing the pentaBDE technical product) concentrations found in this study are very similar to concentrations found in other European countries in recent years, and that they are rather higher than were found in the late 1970s and early 1980s in Scandinavia. We have generally assumed that the general populace of the UK, which has had the strictest furniture flame retardant legislation in the world for some years, would be significantly more exposed to the pentaBDE product (used until recently as the main flame retardant for polyurethane foam, used in furniture, for example) than people living in other countries. This appears not to be the case, although the range of detected values is quite broad, which may be due to a small number of people having particular exposure to these chemicals, either at home or through their occupation. Of the two large (highly brominated) BDE congeners analysed, which are indicative of octaBDE and decaBDE usage, respectively, BDE183 was found in this study at a similar median concentration, and with a similar range, as found in recent studies in Sweden, whereas BDE209 was found at a somewhat higher median concentration than found in Sweden, but with a similar overall range of concentrations. It should be noted that the highest BDE209 concentrations reported in the Swedish study were samples taken from occupationally exposed people (workers in the flame retarded rubber industry), and are similar to those found in this study even though the people with the highest concentrations in this study were not working in industries particularly noted for BDE209 use.

STATISTICAL ANALYSIS

The questionnaire results were transferred into a spreadsheet and, where appropriate, the answers were divided into a small number of discrete bands. The entire data-set, formed by the chemical concentration data and the questionnaire data, was transferred into SPSS (a statistical software package) for statistical analysis.

As is common in surveys of chemical pollutants in animals, there was evidence that the chemical concentrations did not conform to the Normal distribution, but were closer to the log-Normal distribution. Chemical concentration data were therefore transformed to their natural logarithms (ln) for some of the statistical analyses (this is stated when performed). To reduce the complexity of the analysis one PCB was chosen to represent each chlorination level represented in the PCBs analysed (PCBs 28, 52, 118, 153, 180 and 194). Six PBDEs were also chosen as representative of the technical flame retardant products used in the UK (BDEs 47, 99, 100, 153, 154 and 183), and the OC pesticides which were most commonly detected (total HCH, p,p'-DDE, p,p'-DDT and HCB) were included in the analysis.

Descriptive statistics

155 volunteers provided samples, of which 50 were male and 105 female. The volunteers were aged from 22 to 80, with a median of 40.5. The body mass index (BMI) of the volunteers ranged between 17.7 and 42.8, with a median of 23.4. Computer usage ranged from 0 to 12 hours per day, with an average of 4.3 hours. 10 volunteers reported that they were gaining weight, 17 that they were losing weight, and the remainder had a stable weight. 42 volunteers reported that they had spent most of their lives living in northern England, 71 in the south, 13 in Scotland, 16 in Wales, 11 in Northern Ireland and 1 abroad. 20% of volunteers had bought a new carpet, mattress, sofa or car in the last year, 45% between 1 and 3 years ago, 20 % between 4 and 10 years ago and the remainder more than 10 years ago. 1 volunteer classed themself as vegan, 19 as vegetarian, 23 as vegetarians who eat fish, and 111 as omnivores. 50% of the volunteers estimated that between 1 and 25% of their diet was

organically produced, 16% said between 25 and 50%, 7 % between 50 and 75%, and 6% said greater than 75%. 54 of the female volunteers had not had a child, 11 had had one, 18 had had 2 children, 11 had 3 and 6 had 4 or more children. The majority of women breastfed all of their children.

Correlation of chemical concentrations

To investigate the correlation of different chemicals they were grouped, according to historic usage pattern, as Total DDT and metabolites (TotDDX), Total HCHs (TotHCH), Total PCBs (TotPCB), HCB and Total PBDEs (TotBDE). All correlations were tested at the 95% confidence level. It was found that HCB correlated well with TotDDX and TotHCH and that TotPCB also correlated well with TotDDX and TotHCH, but not with HCB. TotBDE did not correlate well with any other chemical group. This shows, roughly speaking, that concentrations in humans of 'old use' chemicals, which have been banned in the UK for some decades, correlate well with each other (indicating that they are likely to have reach the volunteers through similar exposure patterns, histories and routes), whilst the new use chemicals (PBDEs) do not correlate with them (indicating that they are likely to have reached humans through different exposure patterns, histories or routes).

Effect of lifestyle and personal data on chemical concentration

Each of the selected chemicals (log transformed) was split into groups defined by the coding of each question in the questionnaire. The means of these groups were compared to see whether the lifestyle or personal data may have an impact on the chemical concentration. The significant results are presented in Table 6.

Table 6 – Significant (at the 95% confidence level) differences between chemical concentrations for personal or lifestyle groupings

Lifestyle parameter	Chemicals with significant differences between groupings
Current Location	Total HCH; HCB; BDEs 154, 183; PCB28
Sex	Total HCH; BDEs 100, 153; PCB118
Age	Total HCH; p,p'-DDT, p,p'-DDE; PCBs 118, 153, 180, 194
BMI	Total HCH; BDE154; PCB118
Place resided in the longest	HCB; BDE154; PCBs 28, 118, 153, 180, 194
No. of children carried	HCH; BDE153; PCBs 118, 180, 194

It should be noted that some of the groups for some parameters represented few volunteers, and that some of the questions asked may give answers which correlate to some degree with other parameters, but are otherwise unrelated (e.g. as age increases it will be more likely that women will have had more children, so the impact of age may be seen in the significance of difference in groups for the number of children carried, and vice versa). We will concentrate on the parameters which had the most complete data-sets (i.e. all, or most, groups with reasonable numbers of volunteers) – excluding those that had insufficient data – and which may be expected to have the greatest impact on chemical concentrations.

The box-plots of the chemicals separated by groupings in the 'place resided in the longest' category were inspected, and showed that for PCBs (see Figure 2) and BDE154 all of the UK regions had similar ranges and median concentrations. However, for HCB (Figure 3) Northern Ireland showed the highest median concentration, followed by northern England, although all regions had similar concentration ranges.

(*N.B.* Ranges were compared including the outliers isolated by the statistical program used, since this function could not be switched off.)

It appears, from scatterplots of chemicals against age, that the following chemicals are more likely to be found at higher concentrations in older people: p,p'-DDT, p,p'-DDE, PCB118, PCB153, PCB180 and PCB194. The effect of age on the concentration of PCB153, PCB180 and PCB194 appears to be different for males and females (see Figure 4).

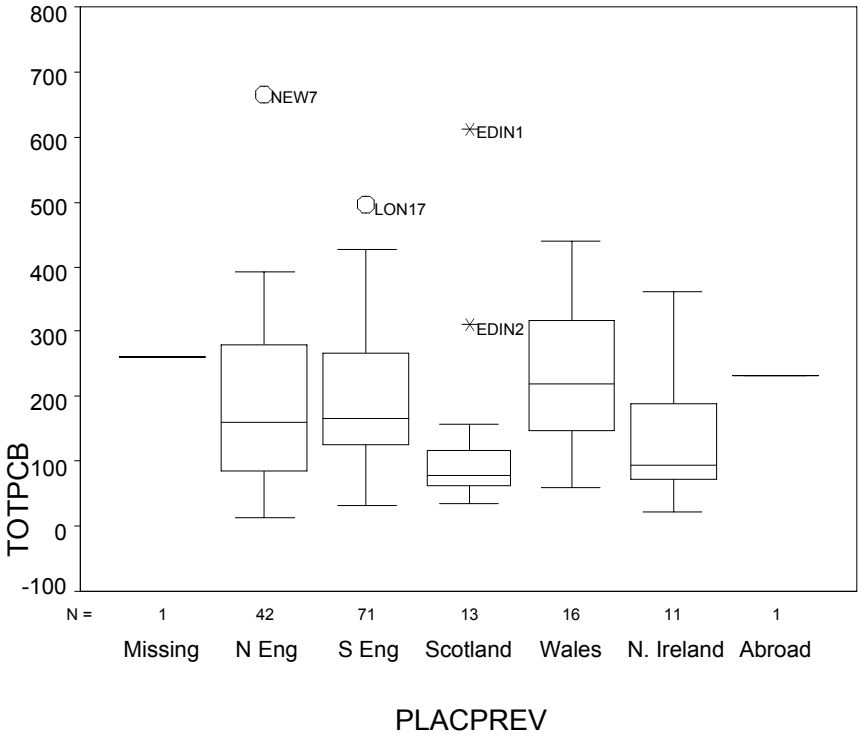

Figure 2 – Boxplot of total PCB concentration and the place resided in the longest ('Missing' = Sample LON6 - no data available for previous residences).

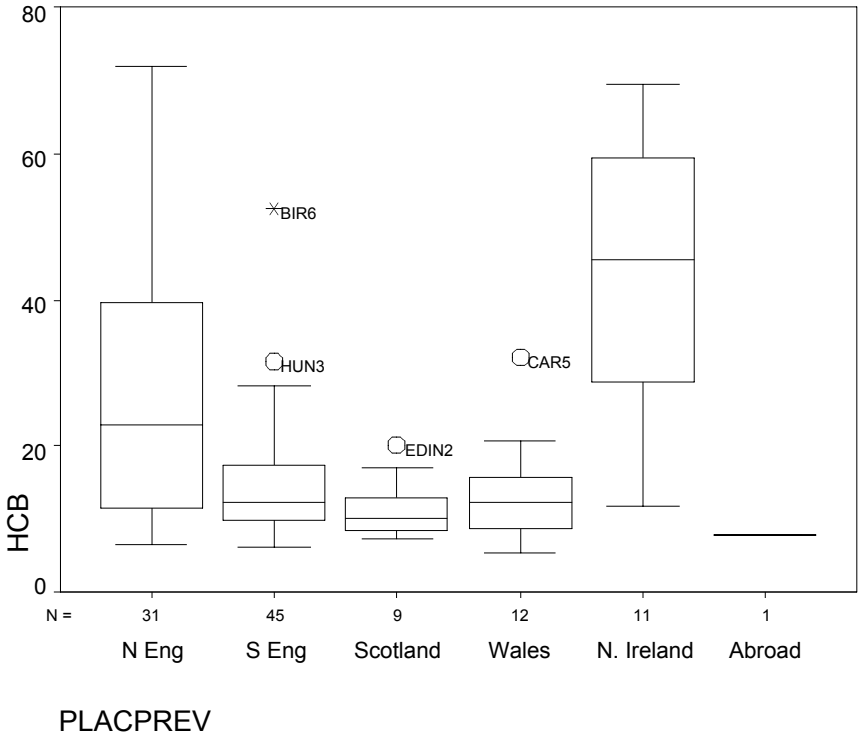

Figure 3 – Boxplot of HCB concentration and the place resided in the longest.

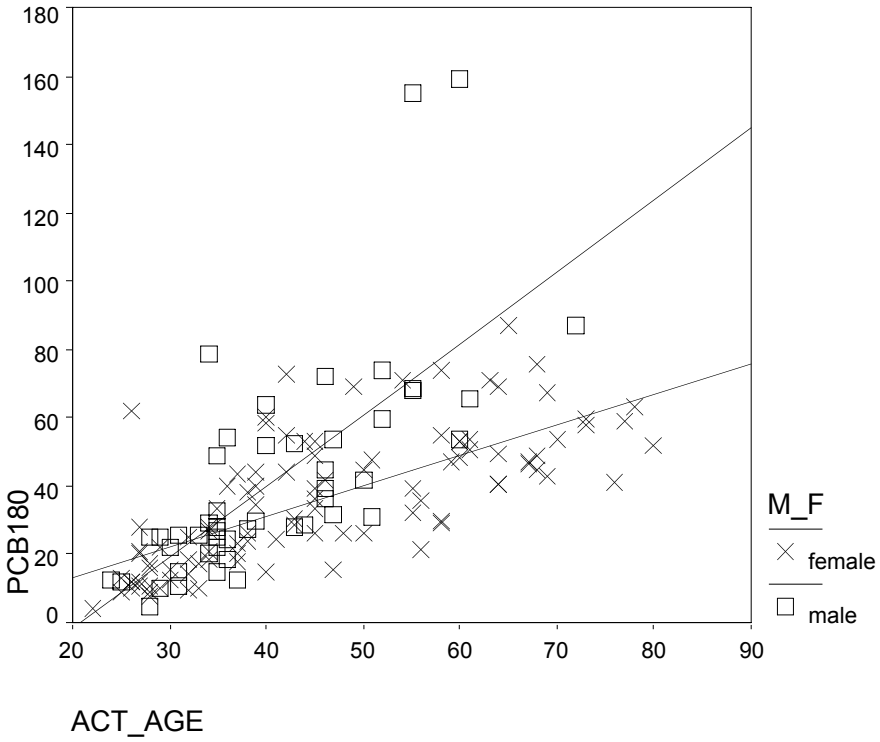

Figure 4 – Concentration of PCB180 plotted against age, with the sexes differentiated

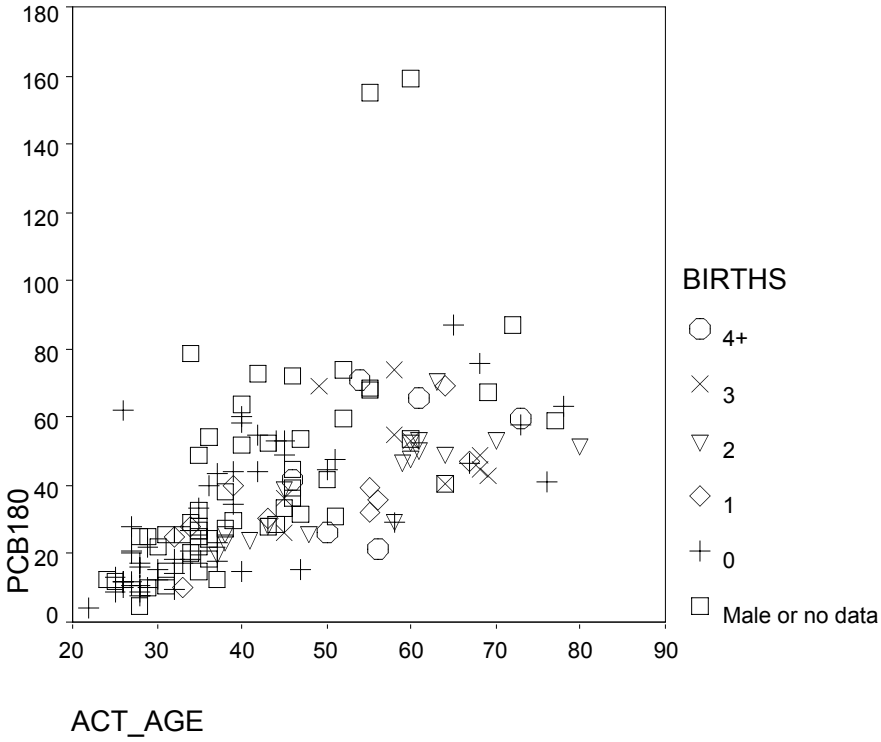

Figure 5 – Concentration of PCB180 plotted against age, with the number of children carried differentiated

To explain this observation it may be postulated that older women have 'off-loaded' some of their body-burden of the more persistent chemicals to their children during pregnancy and lactation (see Alcock *et al.*, 2000), and when the same plot is made with the number of children carried differentiated (Figure 5) there is evidence of a slight tendency for the mothers of more children to have slightly lower concentrations in their age-range.

A straight line model can be fitted to the PCB concentrations against age to determine whether the effect of sex on the change in PCB concentration is statistically significant. Performing this on the selected PCBs showed that for women there is a significant reduction in the increase in PCB concentration with age for PCB118, PCB153, PCB180 and PCB194. It should be pointed out, however, that the concentrations of PCBs in the temperate environment have been shown to have been generally decreasing since they were banned in most countries, in the 1970s, and therefore that the future concentration difference between men and women may not match that observed here.

Principal Components Analysis

In another attempt to find patterns in the data which might be used to elucidate effects of particular lifestyle or personal parameters principal components analysis (PCA) was performed on the data but did not produce any informative results.

Concluding remarks

This study was, we believe, the first survey of blood serum concentrations of PCBs, organochlorine pesticides and PBDEs in the UK. The product of the study is a large and valuable data-set which may be used as an indicator of human exposure to this range of chemicals in the UK, for comparison to other regions, and may be used to determine patterns of exposure to certain chemical groups dependent on lifestyle. In particular, we have shown a correlation between PCB concentrations and age, with a significant difference in gradient between males and females. We have also found that the UK population in 2003 had similar PBDE concentrations, and a similar concentration distribution, to that found in the general population in Sweden in 2002, with approximately 5% of the population having more than five times the median PBDE concentration. This is somewhat surprising because of the history of PBDE production and use in the UK compared to Sweden.

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APPENDICES

APPENDIX 1

Concentrations of PCBs, PBDEs and organochlorine pesticides in serum samples

APPENDIX 2

Data summarised according to sampling location

APPENDIX 3

PCB numbers used

APPENDIX 4

PBDE numbers used

APPENDIX 5

Glossary

Appendix 1 – Concentrations of PCBs, PBDEs and organochlorine pesticides in serum samples

Appendix 1, Table 1 - PCB concentrations found in serum samples

Sample	WWF 1	WWF 2	WWF 3	WWF 4	WWF 5	WWF 6	WWF 8
PCB	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid
18	0.72	1.4	1.6	2.2	2.9	0.52	<0.3
22	0.6	1	1.7	2.5	2.7	<0.3	0.44
28	3.3	3.2	7.6	7.2	10	1.9	2.2
31	1.5	3	3.4	4.6	6.3	<0.9	<1.0
41/64	<6.0	<6.2	<5.4	19	<7.9	<4.2	<4.7
44	0.58	0.61	0.96	1.1	1.5	0.43	0.48
49	<0.48	0.66	0.68	1.1	1.6	0.53	<0.38
52	<0.48	<0.49	1.2	1	1.9	<0.33	<0.38
54	<0.48	<0.49	<0.43	<0.48	<0.63	<0.33	<0.38
60/56	<11	<12	<81	<94	<15	<8.	<9.
70	<0.48	<0.49	0.86	0.94	1.4	<0.33	<0.38
74	4.1	1.6	9.2	6.7	3.8	2.2	8
87	9.8	4.3	4	27	57	<1.4	<1.6
90/101	<5.4	<5.6	7	<5.4	9	<3.8	<4.2
95	<5.1	<5.2	5.1	<5.1	8.5	<3.5	<4.0
99	1.8	1.7	4.4	5.1	2.6	1.8	2.1
104	<0.48	<0.49	<0.43	<0.48	<0.63	<0.33	<0.38
105	1.5	1.3	3.8	3.1	2	1.4	0.91
110	<1.4	<1.5	2	<1.4	2.1	<1.0	<1.1
114	0.55	<0.4	1.2	0.76	<0.6	0.44	0.72
118	6.9	4.1	17	11	6.1	4.9	5.7
123	<0.48	<0.49	0.53	<0.48	0.64	<0.33	<0.38
138	21	11	53	50	18	26	23
141	<1.0	<1.0	2	<1.0	2	2.6	<0.8
149	<8.0	<8.3	9.8	<8.0	11	7.1	<6.3
151	<3.6	<3.7	5.1	<3.6	6.7	<2.5	<2.8
153	33	16	89	81	29	40	54
155	<0.48	<0.49	<0.43	<0.48	<0.63	<0.33	<0.38
156	3.7	1	9.8	8.6	1.1	3.7	6.4
157	1.1	<0.4	2.7	1.7	<0.6	1.2	1.3
158	0.52	0.84	1.3	0.92	1.3	1	1.2
167	1.4	0.66	4.6	3.1	1.3	0.96	2.1
170	11	3.2	27	28	5.2	13	17
174	1.5	<0.9	1.1	<0.9	1.5	3.9	<0.7
180	25	7.6	69	64	12	30	40
183	5.9	2.5	15	14	4.5	10	7.9
187	2.2	1.4	5.7	5.3	2.3	4	2.6
188	<0.48	<0.49	<0.43	<0.48	<0.63	<0.33	<0.38
189	0.55	<0.4	1.2	0.96	<0.6	<0.3	0.68
194	3.8	1.5	11	11	1.5	5.1	6.2
199	<0.48	<0.49	<0.43	<0.48	<0.63	<0.33	<0.38
203	3.2	2.5	8.7	9	2	4.3	5.1

Appendix 1, Table 2 - PCB concentrations found in serum samples

Sample	WWF 10	WWF 11	WWF 13	WWF 14	WWF 15	WWF 16	WWF 17	WWF 18
PCB	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid
18	0.6	1.2	1.1	1.3	2.3	0.9	<0.6	<0.4
22	0.71	2.1	0.92	1.7	3.9	1.8	1.1	0.72
28	3.4	5.5	3.7	2.3	11	1.8	3	2.6
31	1.1	4.2	2.3	1	8.1	1.2	<1.8	<1.2
41/64	<4.1	<10.	<5.8	<14.	<6.7	<5.1	<8.1	7.1
44	0.35	<0.8	0.51	<0.6	1.6	0.46	0.9	0.43
49	0.41	0.97	0.72	0.87	1.3	0.54	<0.65	<0.43
52	0.33	<0.87	0.55	<0.67	1.9	<0.4	<0.65	<0.43
54	<0.33	<0.87	<0.46	<0.67	<0.53	<0.4	<0.65	<0.43
60/56	<8.	<18	<56	<8.	<87	<9.	<85	<75
70	<0.33	<0.87	<0.46	2.1	1.7	<0.4	<0.65	<0.43
74	10	3.9	7.7	2.6	12	3.5	3.6	11
87	<1.4	<3.7	<1.9	<0.6	3.9	<1.7	<2.8	<1.8
90/101	<3.7	<9.8	<5.2	3.5	7.2	<4.5	<7.3	<4.8
95	<3.5	<9.2	<4.9	2	<5.7	<4.3	<6.9	<4.5
99	8.6	3.4	5.4	1.3	5.4	2.8	3.7	4.9
104	<0.33	<0.87	<0.46	<0.67	<0.53	<0.4	<0.65	<0.43
105	6.4	2.3	3.2	<0.6	3.6	1.6	2.2	3.7
110	<1.0	<2.6	<1.4	1.3	<1.6	<1.2	<2	<1.3
114	1.3	<0.8	0.91	<0.6	1.4	<0.4	<0.6	0.91
118	25	6.9	14	3.2	15	5	4.3	13
123	<0.33	<0.87	<0.46	<0.67	<0.53	0.66	<0.65	<0.43
138	70	20	50	16	56	27	22	32
141	<0.7	<1.8	<1	<0.6	<1.1	3.6	<1.4	<0.9
149	<5.5	<14.	<7.7	4.3	<8.9	10	<10.	<7.1
151	<2.5	<6.5	<3.4	1.9	4.5	4.5	<4.9	<3.2
153	109	33	73	23	87	40	38	49
155	<0.33	<0.87	<0.46	<0.67	<0.53	<0.4	<0.65	<0.43
156	10	3.5	7.3	2.1	9	2.2	3.5	5.3
157	1.8	1	2	1.6	2.4	0.54	1.2	1.1
158	1.4	1.5	0.87	<0.6	1.6	1.3	1.1	0.99
167	4.2	1.5	2.9	<0.6	4	1.7	0.67	2.3
170	27	9.8	19	8.8	25	9.7	11	15
174	<0.6	<1.7	<0.9	1.7	1.2	5.1	<1.2	<0.8
180	62	25	44	22	60	22	29	33
183	15	3.4	10	5	16	8.8	5.2	5.5
187	6.3	2.1	4.7	1.3	5.9	4.8	3.3	3.4
188	<0.33	<0.87	<0.46	<0.67	<0.53	<0.4	<0.65	<0.43
189	0.98	<0.8	0.48	<0.6	0.84	<0.4	<0.6	<0.4
194	8	3.8	5.8	4.7	11	3.2	4.8	4.4
199	<0.33	<0.87	<0.46	<0.67	<0.53	<0.4	<0.65	<0.43
203	5.9	3.3	5	2.3	9.7	3.4	4.6	4

Appendix 1, Table 3 - PCB concentrations found in serum samples

Sample	WWF 19	WWF 20	WWF 21	LON1	LON2	LON3	LON4
PCB	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid
18	<0.5	<0.4	<0.4	0.74	<1.2	<1.4	<1.3
22	0.86	<0.4	0.46	0.77	<1.2	<1.4	<1.3
28	2.9	2.4	5	2.7	2.9	3.2	4.1
31	<1.4	<1.2	<1.2	2.4	2	2	3.9
41/64	<6.3	<5.5	<5.4	1.9	4.1	4.2	8
44	<0.5	<0.4	<0.4	0.52	0.57	0.77	0.77
49	<0.5	<0.44	<0.43	<0.85	<1.53	<1.7	<1.6
52	<0.5	<0.44	<0.43	0.8	0.78	0.88	1.4
54	<0.5	<0.44	<0.43	<0.3	<0.54	<0.59	<0.56
60/56	<12	<10	<10	<41	<60	<76	<21
70	<0.5	<0.44	<0.43	0.41	0.75	0.78	0.86
74	3.2	1.6	16	3.6	6.7	6.6	3.7
87	<2.1	<1.9	<1.8	<0.3	<0.5	<0.5	<0.5
90/101	<5.7	<5	<4.9	<1.6	<2.8	<3.2	<3.0
95	<5.4	<4.7	<4.6	<1.2	<2.1	<2.4	<2.3
99	3.5	1.3	7.8	4.1	4.5	3.8	2.5
104	<0.5	<0.44	<0.43	<0.3	<0.54	<0.59	<0.56
105	2.2	1.4	5	1.5	3.4	3.3	1.8
110	<1.5	<1.3	<1.3	<0.4	<0.7	<0.8	<0.7
114	<0.5	<0.4	1.2	0.69	1.2	1.2	0.8
118	7	2.9	21	4.3	9.9	8.8	5.3
123	<0.5	<0.44	<0.43	<0.3	<0.54	<0.59	<0.56
138	27	5.7	57	47	41	39	28
141	<1.1	<0.9	<0.9	<1.3	<2.4	<2.7	<2.5
149	<8.4	<7.4	<7.2	<5.0	<9.1	<10.	<9.5
151	<3.8	<3.3	<3.2	<1.9	<3.5	<3.8	<3.6
153	45	8.7	82	76	65	62	51
155	<0.5	<0.44	<0.43	<0.3	<0.54	<0.59	<0.56
156	3.8	0.59	7.1	8.5	8.1	7.1	6
157	0.9	<0.4	1.7	1.9	2.2	1.9	1.8
158	0.57	0.59	0.56	<0.3	0.61	0.9	<0.5
167	1.9	0.47	4	1.7	2.9	3	2.2
170	13	1.4	21	25	20	17	19
174	<0.9	<0.8	<0.8	<1.7	<3.0	<3.4	<3.2
180	33	4.3	53	59	45	40	79
183	8.4	<0.9	12	4.5	3.4	3.4	5.2
187	2.8	0.66	6.4	12	11	9	33
188	<0.5	<0.44	<0.43	<0.3	<0.54	<0.59	<0.56
189	<0.5	<0.4	0.73	<0.3	<0.5	<0.5	<0.5
194	5.9	3.6	6.8	11	7.2	5.1	39
199	<0.5	<0.44	<0.43	<0.3	<0.54	<0.59	<0.56
203	4.7	0.79	7.3	7.2	<5.8	<6.4	37

Appendix 1, Table 4 - PCB concentrations found in serum samples

Sample	LON5	LON6	LON7	LON8	LON9	LON10	LON11
PCB	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid
18	<1.0	<1.3	<0.9	<0.8	<1.1	<1.0	<7.8
22	<1.0	<1.3	<0.9	<0.8	<1.1	<1.1	<7.9
28	2.1	<1.5	<1.0	2.3	2.1	<1.2	<8.8
31	1.2	<0.8	<0.5	0.56	1.2	0.76	8.7
41/64	3.8	0.81	1	1.1	2.2	0.97	<3.3
44	0.53	0.58	<0.3	<0.3	<0.4	<0.4	<3.3
49	<1.23	<1.62	<1.09	<1.05	<1.38	<1.31	<9.52
52	0.51	0.57	<0.38	0.4	0.51	<0.46	4.7
54	<0.43	<0.57	<0.38	<0.37	<0.48	<0.46	<3.33
60/56	<15	<69	<14	<15	<22	<17	<12
70	0.52	<0.57	<0.38	0.37	<0.48	<0.46	4.6
74	5.1	5.5	4.2	5.7	5.2	3.1	6.9
87	0.84	<0.5	<0.3	<0.3	<0.4	<0.4	<3.3
90/101	<2.3	<3.0	<2.0	<1.9	<2.6	<2.4	<18.
95	<1.7	<2.3	<1.5	<1.5	<1.9	<1.8	<13.
99	2.3	4.3	1.8	5.2	4.2	1.9	<3.3
104	<0.43	<0.57	<0.38	<0.37	<0.48	<0.46	<3.33
105	1.9	3.3	1.2	2.9	2.5	2	<3.3
110	<0.5	<0.7	<0.5	<0.4	<0.6	<0.6	<4.4
114	0.74	1	0.59	0.63	<0.4	0.75	<3.3
118	6	12	3.5	10	6.6	7	5.5
123	<0.43	<0.57	<0.38	<0.37	<0.48	<0.46	<3.33
138	23	45	20	32	25	21	<52.
141	<1.9	<2.6	<1.7	<1.6	<2.2	<2.1	<15.
149	<7.3	<9.6	<6.4	<6.2	<8.2	<7.8	<56.
151	<2.8	<3.7	<2.4	<2.4	<3.1	<3.0	<21.
153	39	70	35	46	35	34	<54.
155	<0.43	<0.57	<0.38	<0.37	<0.48	<0.46	<3.33
156	4.4	9.1	4.6	4	3.8	4.3	3.9
157	1.4	2.4	1.3	1.2	1.3	1.3	<3.3
158	0.44	<0.5	0.4	0.8	<0.4	<0.4	<3.3
167	2	3.3	1.4	2	1.4	2.1	<3.3
170	11	23	13	10	7.9	10	<19.
174	<2.4	<3.2	<2.1	<2.1	<2.7	<2.6	<19.
180	29	55	31	23	17	25	<33.
183	<2.1	3.3	2	3.3	<2.4	<2.2	<16.
187	5.3	14	5.4	5.2	5.4	4.2	<24.
188	<0.43	<0.57	<0.38	<0.37	<0.48	<0.46	<3.33
189	<0.4	<0.5	<0.3	<0.3	<0.4	<0.4	<3.3
194	3.9	8.9	5.1	2.7	1.7	3	<4.4
199	<0.43	<0.57	<0.38	<0.37	<0.48	<0.46	<3.33
203	<4.6	<6.1	<4.1	<3.9	<5.2	<5	<36.

Appendix 1, Table 5 - PCB concentrations found in serum samples

Sample	LON12	LON13	LON14	LON15	LON16	LON17	LON18
PCB	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid
18	<1.1	<0.9	<1.2	<1.2	<1.4	<1.0	<1.0
22	<1.2	<0.9	<1.2	<1.2	<1.4	<1.1	<1.0
28	2.7	2.3	1.6	3.3	<1.6	9.5	1.5
31	0.98	<0.6	<0.7	1.2	<0.9	0.72	<0.6
41/64	1.7	<0.4	<0.5	<0.5	<0.6	<0.4	<0.4
44	<0.5	<0.4	<0.5	0.53	<0.6	0.62	<0.4
49	<1.44	<1.19	<1.45	<1.51	<1.77	<1.32	<1.31
52	0.59	<0.42	<0.51	0.83	<0.62	0.86	<0.46
54	<0.51	<0.42	<0.51	<0.53	<0.62	<0.46	<0.46
60/56	<19	<98	<19	<20	<23	<47	<17
70	0.55	<0.42	<0.51	0.78	<0.62	<0.46	<0.46
74	4.7	9.4	2.9	11	1.7	22	2.9
87	<0.5	<0.4	<0.5	<0.5	<0.6	0.53	<0.4
90/101	<2.7	<2.2	<2.7	<2.8	<3.3	<2.5	<2.4
95	<2.0	<1.7	<2.0	<2.1	<2.5	<1.8	<1.8
99	2.5	4.3	2.4	10	2.8	11	3
104	<0.51	<0.42	<0.51	<0.53	<0.62	<0.46	<0.46
105	2.9	3.1	1.1	3.8	<0.6	4	1.1
110	<0.6	<0.5	<0.6	<0.7	<0.8	<0.6	<0.6
114	1.1	0.92	<0.5	1.3	<0.6	1.5	<0.4
118	7.1	18	4.3	13	2.2	23	5.6
123	<0.51	<0.42	<0.51	<0.53	<0.62	<0.46	<0.46
138	22	41	13	79	20	88	32
141	<2.3	<1.9	<2.3	<2.4	<2.8	<2.1	<2.1
149	<8.5	<7.0	<8.6	<9.0	<10.	<7.8	<7.8
151	<3.3	<2.7	<3.3	<3.4	<4.0	<3.0	<3
153	31	58	18	126	32	123	43
155	<0.51	<0.42	<0.51	<0.53	<0.62	<0.46	<0.46
156	6.6	7.9	2.2	10	3	14	4.7
157	1.7	1.9	0.51	2.6	0.68	3.1	0.87
158	0.65	0.53	<0.5	2	<0.6	0.7	0.58
167	1.6	4.1	0.97	3.7	0.99	5.8	2.3
170	8.5	21	5.9	29	8.7	38	14
174	<2.8	<2.3	<2.9	<3.0	<3.5	<2.6	<2.6
180	19	49	13	72	22	87	28
183	<2.5	3	<2.5	9.5	<3.0	8.1	3.1
187	4.8	8.7	<3.7	16	<4.6	28	5.6
188	<0.51	<0.42	<0.51	<0.53	<0.62	<0.46	<0.46
189	<0.5	<0.4	<0.5	<0.5	<0.6	<0.4	<0.4
194	2.4	8.1	1.2	10	2.4	14	2.1
199	<0.51	<0.42	<0.51	<0.53	<0.62	<0.46	<0.46
203	<5.4	<4.5	<5.5	8.3	<6.7	11	<4.9

Appendix 1, Table 6 - PCB concentrations found in serum samples

Sample	LON19	HUN1	HUN2	HUN3	HUN4	HUN5	HUN6
PCB	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid
18	<0.9	<0.6	<1.3	<0.8	<0.8	<0.9	<0.7
22	<0.9	<0.6	<1.3	<0.9	<0.8	<0.9	<0.7
28	4	1.1	<1.4	6.2	1.4	<1.0	1.5
31	<0.5	<0.4	<0.8	<0.5	<0.5	<0.5	<0.4
41/64	<0.3	<0.2	<0.5	<0.3	<0.3	<0.3	<0.3
44	<0.3	<0.2	<0.5	<0.3	<0.3	<0.3	<0.3
49	<1.1	<0.8	<1.57	<1.07	<1.02	<1.11	<0.93
52	0.41	<0.28	<0.55	0.62	<0.36	<0.39	<0.33
54	<0.38	<0.28	<0.55	<0.38	<0.36	<0.39	<0.33
60/56	<14	<10	<21	<14	<13	<14	<12
70	<0.38	<0.28	<0.55	<0.38	<0.36	<0.39	<0.33
74	19	6.5	1.9	19	8.8	2.6	5.8
87	<0.3	<0.2	<0.5	<0.3	<0.3	<0.3	<0.3
90/101	<2.0	<1.5	<2.9	<2.0	<1.9	<2.1	<1.7
95	<1.5	<1.1	<2.2	<1.5	<1.4	<1.5	<1.3
99	7.1	3.3	1.6	8.2	2.8	10	2.7
104	<0.38	<0.28	<0.55	<0.38	<0.36	<0.39	<0.33
105	2.2	1.1	<0.5	4	1.5	0.77	1.3
110	<0.5	<0.3	<0.7	<0.5	<0.4	<0.5	<0.4
114	1.1	0.74	<0.5	1.1	0.75	<0.3	0.54
118	14	7	2.7	23	12	3.6	7.6
123	<0.38	<0.28	<0.55	<0.38	<0.36	<0.39	<0.33
138	48	39	14	51	41	55	28
141	<1.7	<1.2	<2.5	<1.7	<1.6	<1.7	<1.5
149	<6.5	<4.7	<9.3	<6.3	<6.0	<6.5	<5.5
151	<2.5	<1.8	<3.5	<2.4	<2.3	<2.5	<2.1
153	72	56	23	64	60	70	44
155	<0.38	<0.28	<0.55	<0.38	<0.36	<0.39	<0.33
156	7.7	7.8	2.9	7.2	8.3	4.9	5.1
157	1.8	1.8	0.72	1.6	1.9	1.1	1.1
158	0.39	<0.2	<0.5	0.57	<0.3	5	<0.3
167	3.5	2.5	1.2	3.8	3.8	1.3	1.9
170	22	21	8.1	19	24	18	15
174	<2.2	<1.6	<3.1	<2.1	<2.0	<2.2	<1.8
180	53	49	21	41	54	45	36
183	5	3	<2.7	5	3	9.8	2.7
187	9.4	12	<4.0	9.6	12	11	10
188	<0.38	<0.28	<0.55	<0.38	<0.36	<0.39	<0.33
189	<0.3	<0.2	<0.5	<0.3	<0.3	<0.3	<0.3
194	7.6	8.9	2.5	6.1	8.2	6.9	6.5
199	<0.38	<0.28	<0.55	<0.38	<0.36	<0.39	<0.33
203	5.8	6	<5.9	5.3	5.7	7.1	4.5

Appendix 1, Table 7 - PCB concentrations found in serum samples

Sample	HUN7	HUN8	HUN9	HUN10	EXE1	EXE2	EXE3
PCB	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid
18	<0.9	<0.9	<1.1	<1.3	<1.1	<0.7	<1.1
22	<0.9	<0.9	<1.1	<1.3	<1.1	<0.7	<1.1
28	<1.1	<1.0	1.9	<1.5	1.7	0.91	<1.2
31	<0.6	<0.5	<0.7	<0.8	<0.7	<0.5	<0.7
41/64	<0.4	<0.3	<0.4	<0.5	<0.4	<0.3	<0.4
44	<0.4	<0.3	<0.4	<0.5	<0.4	<0.3	<0.4
49	<1.19	<1.12	<1.35	<1.64	<1.38	<0.94	<1.33
52	<0.42	<0.39	<0.47	<0.57	<0.48	<0.33	<0.47
54	<0.42	<0.39	<0.47	<0.57	<0.48	<0.33	<0.47
60/56	<15	<27	<10	<22	<18	<12	<17
70	<0.42	<0.39	<0.47	<0.57	<0.48	<0.33	<0.47
74	2.2	2.1	2.9	3	7.9	1.9	8.4
87	<0.4	<0.3	<0.4	<0.5	1.5	<0.3	<0.4
90/101	<2.2	<2.1	<2.5	<3.1	<2.6	<1.7	<2.5
95	<1.7	<1.6	<1.9	<2.3	<1.9	<1.3	<1.9
99	2.7	2.8	2.3	2.4	4.4	1.9	3.6
104	<0.42	<0.39	<0.47	<0.57	<0.48	<0.33	<0.47
105	0.88	0.71	1.4	0.96	1.6	0.69	1.1
110	<0.5	<0.5	<0.6	<0.7	<0.6	<0.4	<0.6
114	<0.4	<0.3	<0.4	<0.5	1	<0.3	0.89
118	4.4	3.4	7	4.5	10	2.8	7.5
123	<0.42	<0.39	<0.47	<0.57	<0.48	<0.33	<0.47
138	22	32	19	21	54	14	42
141	<1.9	<1.8	<2.1	<2.6	<2.2	<1.5	<2.1
149	<7.0	<6.6	<8.0	<9.7	<8.2	<5.6	<7.9
151	<2.7	<2.5	<3.0	<3.7	<3.1	<2.1	<3.0
153	33	53	28	31	81	17	72
155	<0.42	<0.39	<0.47	<0.57	<0.48	<0.33	<0.47
156	3.7	6.6	3.5	3.4	11	2.1	9.5
157	0.8	1.4	0.74	0.75	2.9	0.45	2
158	<0.4	<0.3	<0.4	<0.5	<0.4	<0.3	<0.4
167	1.3	1.2	1.7	1.5	4.1	0.63	3.1
170	12	22	11	11	30	6.3	27
174	<2.3	<2.2	<2.7	<3.2	<2.7	<1.8	<2.6
180	29	52	26	26	71	13	66
183	2.4	2.8	<2.3	<2.8	3.3	<1.6	3.8
187	5.5	10	4.9	<4.2	14	3	11
188	<0.42	<0.39	<0.47	<0.57	<0.48	<0.33	<0.47
189	<0.4	<0.3	<0.4	<0.5	<0.4	<0.3	<0.4
194	5.4	10	5.1	4.1	11	1.6	12
199	<0.42	<0.39	<0.47	<0.57	<0.48	<0.33	<0.47
203	<4.5	5.2	<5.1	<6.2	6.1	<3.5	7.2

Appendix 1, Table 8 - PCB concentrations found in serum samples

Sample	EXE4	EXE5	EXE6	EXE7	EXE8	EXE9	EXE10
PCB	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid
18	<1.1	<1.4	<0.5	<1.1	<1.2	<1.0	<1.2
22	<1.1	<1.4	<0.5	4.8	<1.3	<1.0	<1.2
28	3.3	<1.5	1.1	4.9	<1.4	<1.2	<1.3
31	1.3	<0.9	<0.3	<0.7	<0.8	<0.6	<0.7
41/64	<0.4	<0.5	<0.2	<0.5	<0.5	<0.4	<0.5
44	<0.4	<0.5	<0.2	<0.5	<0.5	<0.4	<0.5
49	<1.33	<1.69	<0.68	<1.42	<1.56	<1.3	<1.47
52	0.73	<0.59	0.26	0.7	<0.55	<0.46	<0.52
54	<0.47	<0.59	<0.24	<0.5	<0.55	<0.46	<0.52
60/56	<17	<22	<10	<19	<33	<17	<46
70	0.52	<0.59	<0.24	<0.5	<0.55	<0.46	<0.52
74	3.4	2.2	2.7	5.9	3.3	5.2	1
87	0.66	<0.5	<0.2	<0.5	<0.5	<0.4	<0.5
90/101	<2.5	<3.2	<1.3	2.9	<2.9	<2.4	<2.7
95	<1.9	<2.4	<0.9	<2.0	<2.2	<1.8	<2.1
99	2	1.8	3.2	6.7	2.9	5.1	0.94
104	<0.47	<0.59	<0.24	<0.5	<0.55	<0.46	<0.52
105	0.82	0.63	1	3.1	1.2	1.9	<0.5
110	<0.6	<0.7	<0.3	<0.6	<0.7	<0.6	<0.6
114	<0.4	<0.5	0.31	0.7	<0.5	<0.4	<0.5
118	4.9	2.6	4.8	15	6.2	10	<0.5
123	<0.47	<0.59	<0.24	<0.5	<0.55	<0.46	<0.52
138	20	17	23	55	23	39	<8.0
141	<2.1	<2.7	<1.1	<2.2	<2.5	<2.1	<2.3
149	<7.9	<10.	<4.0	<8.4	<9.3	<7.7	<8.7
151	<3.0	<3.8	<1.5	<3.2	<3.5	<2.9	<3.3
153	33	29	34	82	36	54	11
155	<0.47	<0.59	<0.24	<0.5	<0.55	<0.46	<0.52
156	4	3.3	3.5	6.8	3.7	5.2	1.9
157	0.74	0.64	0.72	1.6	0.8	1.4	<0.5
158	<0.4	<0.5	0.36	1.2	<0.5	0.54	<0.5
167	1.6	1.1	0.96	3.5	1.9	2.7	<0.5
170	12	10	11	22	12	16	4.8
174	<2.6	<3.4	<1.3	<2.8	<3.1	<2.6	<2.9
180	28	26	26	53	33	38	12
183	<2.3	<2.9	2.2	5.5	<2.7	3.9	<2.5
187	4.9	5	6.1	16	7.4	8.4	<3.8
188	<0.47	<0.59	<0.24	<0.5	<0.55	<0.46	<0.52
189	<0.4	<0.5	<0.2	<0.5	<0.5	<0.4	<0.5
194	4.5	4.4	4.4	7.8	5.8	4.6	1.2
199	<0.47	<0.59	<0.24	<0.5	<0.55	<0.46	<0.52
203	<5.0	<6.4	2.8	<5.4	<5.9	<4.9	<5.6

Appendix 1, Table 9 - PCB concentrations found in serum samples

Sample	EXE11	CAR1	CAR2	CAR3	CAR4	CAR5	CAR6
PCB	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid
18	<0.9	<1.1	<0.8	<1.2	<1.1	<0.9	<1.4
22	<0.9	<1.1	<0.8	<1.2	<1.1	<0.9	<1.4
28	2.7	<1.3	1.2	1.5	1.8	2.1	2.3
31	<0.6	<0.7	<0.5	<0.7	<0.7	0.73	<0.9
41/64	<0.4	<0.5	<0.3	<0.5	<0.4	<0.4	<0.6
44	<0.4	<0.5	<0.3	<0.5	<0.4	<0.4	<0.6
49	<1.15	<1.42	<1.02	<1.45	<1.33	<1.14	<1.75
52	<0.4	<0.5	0.43	<0.51	<0.47	0.57	0.71
54	<0.4	<0.5	<0.36	<0.51	<0.47	<0.4	<0.61
60/56	<27	<19	<13	<19	<17	<15	<23
70	<0.4	<0.5	<0.36	<0.51	<0.47	<0.4	<0.61
74	12	1.6	5.7	3.5	9.6	15	4.9
87	<0.4	<0.5	<0.3	<0.5	<0.4	0.57	<0.6
90/101	<2.1	<2.6	<1.9	<2.7	<2.5	<2.1	<3.3
95	<1.6	<2.0	<1.4	<2.0	<1.9	<1.6	<2.5
99	7.9	2	7	4.3	3.5	17	3.7
104	<0.4	<0.5	<0.36	<0.51	<0.47	<0.4	<0.61
105	3.4	0.93	1	1.1	1.5	3.2	1.9
110	<0.5	<0.6	<0.4	<0.6	<0.6	<0.5	<0.8
114	0.81	<0.5	0.7	<0.5	0.69	1.1	<0.6
118	20	3.3	5.8	4.9	11	20	11
123	<0.4	<0.5	<0.36	<0.51	<0.47	<0.4	<0.61
138	48	17	65	30	39	96	38
141	<1.8	<2.2	<1.6	<2.3	<2.1	<1.8	<2.8
149	<6.8	<8.4	<6.0	<8.6	<7.9	<6.8	<10.
151	<2.6	<3.2	<2.3	<3.3	<3.0	<2.6	<4
153	62	28	97	40	56	121	64
155	<0.4	<0.5	<0.36	<0.51	<0.47	<0.4	<0.61
156	6.6	4	9.2	4.2	6.3	9.5	6.7
157	1.6	0.89	2.1	0.94	1.3	2.4	1.7
158	0.56	<0.5	0.74	<0.5	<0.4	3.2	<0.6
167	3.1	1.1	2.2	1.4	3.1	4.6	3.5
170	17	14	27	13	18	28	21
174	<2.3	<2.8	<2.0	<2.9	<2.6	<2.3	<3.5
180	40	36	69	28	44	68	73
183	5.2	<2.4	6.6	3.4	3.2	11	4.7
187	11	8	16	7.8	9.6	18	22
188	<0.4	<0.5	<0.36	<0.51	<0.47	<0.4	<0.61
189	<0.4	<0.5	<0.3	<0.5	<0.4	<0.4	<0.6
194	6.6	10	11	4.5	5.6	9.2	22
199	<0.4	<0.5	<0.36	<0.51	<0.47	<0.4	<0.61
203	5.7	<5.3	8.1	<5.5	<5.0	9.2	18

Appendix 1, Table 10 - PCB concentrations found in serum samples

Sample	CAR7	CAR8	CAR9	CAR10	CAR11	CAR12	CAR13
PCB	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid
18	<1.1	<0.8	<0.8	<0.7	<1.0	<1.6	<0.8
22	<1.1	<0.8	<0.8	<0.7	<1.0	<1.6	<0.8
28	3	2.8	1.2	0.86	1.9	<1.8	2.1
31	1.3	<0.5	<0.5	<0.4	<0.6	<1.0	<0.5
41/64	<0.4	<0.3	<0.3	<0.3	<0.4	<0.6	<0.3
44	0.5	<0.3	<0.3	<0.3	<0.4	<0.6	<0.3
49	<1.35	<1.06	<1.03	<0.88	<1.25	<1.96	<1.06
52	0.85	0.57	0.46	<0.31	<0.44	<0.68	<0.37
54	<0.47	<0.37	<0.36	<0.31	<0.44	<0.68	<0.37
60/56	<42	<14	<13	<11	<16	<38	<14
70	0.73	<0.37	<0.36	0.39	<0.44	<0.68	<0.37
74	7	10	5.4	2.3	9.8	4.4	4
87	<0.4	<0.3	<0.3	<0.3	<0.4	<0.6	<0.3
90/101	3	<2	2.1	<1.6	<2.3	<3.7	<2
95	2.3	<1.5	1.7	<1.2	<1.7	<2.8	<1.5
99	5.9	4.7	2.7	2.3	5.9	2.5	2.6
104	<0.47	<0.37	<0.36	<0.31	<0.44	<0.68	<0.37
105	2.5	2.7	1.7	0.91	1.7	0.98	0.56
110	<0.6	<0.5	<0.4	<0.4	<0.5	<0.9	<0.5
114	0.95	0.74	0.5	<0.3	0.83	<0.6	<0.3
118	7.8	16	9.5	3.6	10	3.8	7.3
123	<0.47	<0.37	<0.36	<0.31	<0.44	<0.68	<0.37
138	54	38	28	13	47	23	27
141	<2.1	<1.7	<1.6	<1.4	<2.0	<3.1	<1.7
149	<8.0	<6.2	<6.1	<5.2	<7.4	<11.	<6.3
151	<3.0	<2.4	<2.3	<2.0	<2.8	<4.4	<2.4
153	80	55	47	17	68	33	41
155	<0.47	<0.37	<0.36	<0.31	<0.44	<0.68	<0.37
156	7.3	6.2	4.7	1.7	7.1	4.2	4
157	1.8	1.4	1.2	0.44	1.8	0.97	0.85
158	1.1	0.44	<0.3	<0.3	0.44	<0.6	<0.3
167	3.1	3.2	2.5	0.7	2.9	1.5	2.2
170	21	19	14	4.6	19	12	12
174	<2.7	<2.1	<2.0	<1.7	<2.5	<3.9	<2.1
180	53	43	36	10	47	28	31
183	5.3	4.1	2.2	<1.5	5	<3.4	2.7
187	12	9.1	9.2	<2.3	12	7.3	7
188	<0.47	<0.37	<0.36	<0.31	<0.44	<0.68	<0.37
189	<0.4	<0.3	<0.3	<0.3	<0.4	<0.6	<0.3
194	7.5	7.5	5.9	1	6.9	4.4	4.3
199	<0.47	<0.37	<0.36	<0.31	<0.44	<0.68	<0.37
203	5.6	5.7	<3.9	<3.3	5	<7.4	<4.0

Appendix 1, Table 11 - PCB concentrations found in serum samples

Sample	MAN1	MAN2	MAN3	MAN4	MAN5	MAN6	MAN7
PCB	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid
18	<0.9	<1.0	<0.7	<0.7	<0.8	<1.1	<0.9
22	<0.9	<1.0	<0.7	<0.7	<0.8	<1.1	<0.9
28	2	<1.2	2	2.4	2.2	<1.3	2.8
31	<0.6	<0.6	0.73	0.63	<0.5	<0.7	<0.6
41/64	<0.4	<0.4	1.3	<0.3	<0.3	<0.4	<0.4
44	<0.4	<0.4	<0.3	<0.3	<0.3	<0.4	<0.4
49	<1.13	<1.3	<0.95	<0.89	<0.97	<1.41	<1.16
52	<0.4	<0.46	0.54	0.39	0.42	<0.49	0.61
54	<0.4	<0.46	<0.33	<0.31	<0.34	<0.49	<0.41
60/56	<25	<17	<12	<40	<11	<18	<15
70	<0.4	<0.46	0.38	<0.31	<0.34	<0.49	<0.41
74	3.4	1.3	7.9	3.8	2.5	2.7	9.2
87	<0.4	<0.4	<0.3	<0.3	<0.3	<0.4	<0.4
90/101	<2.1	<2.4	<1.7	<1.6	<1.8	<2.6	<2.1
95	<1.6	<1.8	<1.3	<1.2	<1.3	<2.0	<1.6
99	2.1	1.3	2.4	2.5	2.8	2.7	11
104	<0.4	<0.46	<0.33	<0.31	<0.34	<0.49	<0.41
105	0.54	<0.4	2.3	1.1	0.88	<0.4	2.2
110	<0.5	<0.6	<0.4	<0.4	<0.4	<0.6	<0.5
114	<0.4	<0.4	0.92	<0.3	<0.3	<0.4	0.79
118	4.7	1.5	7.6	7.3	4.7	2.2	12
123	<0.4	<0.46	<0.33	<0.31	<0.34	<0.49	<0.41
138	13	<7.1	28	20	23	18	79
141	<1.8	<2.1	<1.5	<1.4	<1.5	<2.2	<1.8
149	<6.7	<7.7	<5.6	<5.3	<5.7	<8.3	<6.9
151	<2.6	<2.9	<2.1	<2.0	<2.2	<3.2	<2.6
153	18	8.8	44	27	32	24	106
155	<0.4	<0.46	<0.33	<0.31	<0.34	<0.49	<0.41
156	1.6	0.81	5.7	2.9	3	2.6	8.9
157	<0.4	<0.4	1.4	0.69	0.47	<0.4	1.9
158	<0.4	<0.4	<0.3	<0.3	0.37	<0.4	0.93
167	0.74	<0.4	2.2	1.5	1.1	0.83	3.3
170	4.3	<2.6	14	9	10	6.7	29
174	<2.2	<2.6	<1.9	<1.7	<1.9	<2.8	<2.3
180	10	<4.6	32	22	24	16	69
183	<1.9	<2.2	2	2.1	2.6	<2.4	10
187	<2.9	<3.3	6.6	4.7	6.6	5	23
188	<0.4	<0.46	<0.33	<0.31	<0.34	<0.49	<0.41
189	<0.4	<0.4	<0.3	<0.3	<0.3	<0.4	<0.4
194	0.76	<0.6	5	3.7	3.6	1.5	12
199	<0.4	<0.46	<0.33	<0.31	<0.34	<0.49	<0.41
203	<4.3	<4.9	<3.6	<3.4	<3.7	<5.3	11

Appendix 1, Table 12 - PCB concentrations found in serum samples

Sample	MAN8	MAN9	MAN10	MAN11	BRUSS1	EDIN1	EDIN2
PCB	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid
18	<1.1	0.94	0.89	<1.1	<1.0	<0.7	<1.1
22	<1.1	<0.6	<0.8	<1.1	<1.0	<0.7	<1.1
28	1.3	3.2	3.1	1.5	2.3	2.5	2.2
31	<0.7	1	1.1	<0.7	0.94	0.6	<0.7
41/64	<0.4	<0.2	<0.3	<0.4	<0.4	<0.3	<0.4
44	<0.4	0.3	0.37	<0.4	<0.4	0.33	<0.4
49	<1.4	0.92	1.4	<1.32	<1.27	<0.85	<1.37
52	<0.49	0.54	0.57	<0.46	0.6	0.65	0.58
54	<0.49	<0.28	<0.37	<0.46	<0.45	<0.3	<0.48
60/56	<60	<10	<10	<17	<17	<11	<18
70	<0.49	<0.28	<0.37	<0.46	0.62	0.45	<0.48
74	6	4.6	2.8	3.8	4.4	6.3	6.7
87	<0.4	<0.2	<0.3	<0.4	<0.4	<0.3	<0.4
90/101	<2.6	<1.5	<1.9	<2.5	<2.4	3.6	3.9
95	<2.0	<1.1	<1.5	<1.9	<1.8	3.2	3.3
99	3.6	3.5	2.1	3.5	3.7	5.6	6.1
104	<0.49	<0.28	<0.37	<0.46	<0.45	<0.3	<0.48
105	<0.4	1.1	0.79	0.56	3.9	2.8	2.1
110	<0.6	<0.3	<0.4	<0.6	<0.6	0.71	0.66
114	<0.4	0.34	<0.3	<0.4	0.86	0.89	0.57
118	6.9	7.2	5.9	5.4	11	15	7
123	<0.49	<0.28	<0.37	<0.46	<0.45	<0.3	<0.48
138	29	25	18	26	43	72	61
141	<2.2	<1.3	<1.6	<2.1	<2.0	<1.3	3.5
149	<8.3	<4.7	<6.2	<7.8	<7.5	5.3	11
151	<3.2	<1.8	<2.3	<3.0	<2.9	2.9	4.6
153	41	37	26	34	65	128	81
155	<0.49	<0.28	<0.37	<0.46	<0.45	<0.3	<0.48
156	3.4	4.2	2.9	3.2	6.6	11	7.6
157	0.61	0.75	0.65	0.55	1.4	2.3	2
158	0.62	0.43	<0.3	<0.4	1	0.43	1.1
167	1.8	1.6	1.1	1.1	3.1	3.9	1.9
170	10	12	6.3	9.3	20	55	24
174	<2.8	<1.6	<2.1	<2.6	<2.5	<1.7	5.2
180	27	29	16	21	40	155	55
183	3.5	3	<1.8	2.5	4.1	16	7.7
187	5.9	5.6	3.4	6	13	57	18
188	<0.49	<0.28	<0.37	<0.46	<0.45	<0.3	<0.48
189	<0.4	<0.2	<0.3	<0.4	<0.4	<0.3	<0.4
194	2.4	5.3	1.5	1.9	5.6	39	8.1
199	<0.49	<0.28	<0.37	<0.46	<0.45	<0.3	<0.48
203	<5.3	4	<3.9	<5.0	<4.8	29	5.8

Appendix 1, Table 13 - PCB concentrations found in serum samples

Sample	EDIN3	EDIN4	EDIN5	EDIN6	EDIN7	EDIN8	EDIN9
PCB	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid
18	<0.6	<0.7	<1.3	<0.9	<1.0	<1.2	<0.6
22	<0.6	<0.7	<1.3	<0.9	<1.0	<1.2	<0.6
28	1.4	1.5	<1.5	1.4	1.2	2.3	0.85
31	0.64	0.62	<0.8	<0.6	<0.6	0.96	<0.3
41/64	0.87	<0.3	<0.5	<0.4	<0.4	<0.5	<0.2
44	<0.2	<0.3	0.64	0.53	<0.4	<0.5	<0.2
49	<0.8	<0.89	<1.64	<1.16	<1.24	<1.49	<0.72
52	0.32	0.56	<0.57	<0.41	<0.44	0.52	<0.25
54	<0.28	<0.31	<0.57	<0.41	<0.44	<0.52	<0.25
60/56	<15	<11	<22	<18	<16	<20	<49
70	0.3	<0.31	<0.57	<0.41	<0.44	0.65	<0.25
74	1.7	2.7	2.7	1.1	2.7	2.9	1.7
87	<0.2	<0.3	<0.5	<0.4	<0.4	<0.5	<0.2
90/101	<1.5	3.2	<3.1	<2.1	<2.3	<2.8	<1.3
95	<1.1	3	<2.3	<1.6	<1.7	<2.1	<1.0
99	1.6	2	2	0.84	2.8	1.6	2.3
104	<0.28	<0.31	<0.57	<0.41	<0.44	<0.52	<0.25
105	1.3	0.59	<0.5	<0.4	<0.4	0.7	0.53
110	<0.3	0.45	<0.7	<0.5	<0.5	<0.7	<0.3
114	0.35	0.34	<0.5	<0.4	<0.4	<0.5	<0.2
118	3	3.8	3.2	1.3	3.9	4.3	2.8
123	<0.28	<0.31	<0.57	<0.41	<0.44	<0.52	<0.25
138	7.2	18	15	6.7	20	14	13
141	<1.2	<1.4	<2.6	<1.8	<2	<2.4	<1.1
149	<4.7	<5.2	<9.7	<6.9	<7.4	<8.8	<4.3
151	<1.8	2.5	<3.7	<2.6	<2.8	<3.4	<1.6
153	10	29	23	11	28	20	18
155	<0.28	<0.31	<0.57	<0.41	<0.44	<0.52	<0.25
156	1.1	3.1	2	1.8	2.4	2.1	1.7
157	0.37	0.6	<0.5	<0.4	<0.4	<0.5	<0.2
158	0.34	<0.3	<0.5	<0.4	<0.4	<0.5	0.27
167	0.59	1.1	1.1	<0.4	0.88	1.1	0.57
170	1.8	9.5	6	4.1	7.1	5.8	4.5
174	<1.6	<1.7	<3.2	<2.3	<2.5	<2.9	<1.4
180	4.7	24	16	11	18	14	11
183	<1.4	2.2	<2.8	<2.0	<2.1	<2.6	1.4
187	<2.0	4.7	4.5	<3.0	5	<3.8	2.1
188	<0.28	<0.31	<0.57	<0.41	<0.44	<0.52	<0.25
189	<0.2	<0.3	<0.5	<0.4	<0.4	<0.5	<0.2
194	0.42	4.2	1.3	1.6	2.1	1.1	1.1
199	<0.28	<0.31	<0.57	<0.41	<0.44	<0.52	<0.25
203	<3.0	<3.3	<6.2	<4.4	<4.7	<5.6	<2.7

Appendix 1, Table 14 - PCB concentrations found in serum samples

Sample	EDIN10	EDIN11	EDIN12	NOT1	NOT2	NOT3	NOT4
PCB	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid
18	<1.1	<0.9	<0.8	<1.1	<1.1	<1	<0.7
22	<1.1	<0.9	<0.9	<1.1	<1.1	<1.0	<0.7
28	1.8	3.5	1.1	1.9	1.6	3.2	0.89
31	1.3	<0.5	<0.5	<0.7	<0.7	<0.6	0.62
41/64	<0.4	<0.3	<0.3	<0.4	<0.5	<0.4	1
44	<0.4	<0.3	<0.3	<0.4	<0.5	0.42	<0.2
49	<1.39	<1.12	<1.08	<1.32	<1.42	<1.21	<0.84
52	0.57	0.49	<0.38	<0.46	<0.5	0.55	<0.29
54	<0.49	<0.39	<0.38	<0.46	<0.5	<0.42	<0.29
60/56	<18	<15	<14	<17	<31	<31	<35
70	0.52	0.56	<0.38	<0.46	<0.5	0.69	<0.29
74	1.7	5.3	4.1	2.2	1.6	8	11
87	<0.4	<0.3	<0.3	<0.4	<0.5	<0.4	<0.2
90/101	<2.6	<2.1	<2.0	<2.5	<2.6	<2.2	<1.5
95	<2	<1.6	<1.5	<1.9	<2.0	<1.7	<1.2
99	1.6	3.4	2.6	1.5	0.71	9.6	3.7
104	<0.49	<0.39	<0.38	<0.46	<0.5	<0.42	<0.29
105	<0.4	1.6	0.51	<0.4	<0.5	4.2	2
110	<0.6	<0.5	<0.5	<0.6	<0.6	<0.5	<0.4
114	<0.4	0.47	<0.3	<0.4	<0.5	0.74	1.2
118	0.52	8.4	2.9	2.6	0.71	19	9.5
123	<0.49	<0.39	<0.38	<0.46	<0.5	<0.42	<0.29
138	16	29	15	10	8.1	59	45
141	<2.2	<1.8	<1.7	<2.1	<2.2	<1.9	<1.3
149	<8.3	<6.6	<6.4	<7.8	<8.4	<7.2	<5.0
151	<3.1	<2.5	<2.4	<3.0	<3.2	<2.7	<1.9
153	28	42	21	15	15	85	72
155	<0.49	<0.39	<0.38	<0.46	<0.5	<0.42	<0.29
156	2.9	4	2.2	1.3	2.6	7.7	9.1
157	<0.4	0.7	<0.3	<0.4	<0.5	1.3	2.2
158	<0.4	<0.3	<0.3	<0.4	<0.5	0.94	<0.2
167	0.61	2	0.56	0.81	<0.5	3.5	3.3
170	9.4	12	6.7	4	6.8	23	20
174	<2.8	<2.2	<2.1	<2.6	<2.8	<2.4	<1.6
180	27	30	15	9.5	20	54	48
183	<2.4	3	<1.8	<2.3	<2.4	7.5	3.3
187	5.1	7.8	4.5	<3.4	<3.6	15	7.5
188	<0.49	<0.39	<0.38	<0.46	<0.5	<0.42	<0.29
189	<0.4	<0.3	<0.3	<0.4	<0.5	<0.4	<0.2
194	4.4	4.7	2	<0.6	2.8	8.5	7.3
199	<0.49	<0.39	<0.38	<0.46	<0.5	<0.42	<0.29
203	<5.3	<4.2	<4.1	<5.0	<5.3	6.7	5.1

Appendix 1, Table 15 - PCB concentrations found in serum samples

Sample	NOT5	NOT6	NOT7	NOT8	NOT9	NOT10	NOT11
PCB	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid
18	<1.0	<1.1	<0.6	<1.0	<0.8	<1.0	<0.8
22	<1.0	<1.1	<0.6	<1.0	<0.8	<1.0	<0.8
28	7.8	1.9	2.5	2.5	2.4	2.2	1.9
31	1.3	<0.7	0.5	<0.6	<0.5	<0.6	<0.5
41/64	<0.4	<0.4	<0.2	<0.4	<0.3	<0.4	<0.3
44	<0.4	<0.4	<0.2	<0.4	<0.3	<0.4	<0.3
49	<1.25	<1.4	<0.79	<1.22	<0.98	<1.26	<1.04
52	0.79	<0.49	0.38	0.65	<0.34	<0.44	0.5
54	<0.44	<0.49	<0.28	<0.43	<0.34	<0.44	<0.36
60/56	<38	<18	<10	<16	<13	<16	<13
70	0.66	2	<0.28	<0.43	<0.34	<0.44	0.82
74	15	13	13	10	11	1.5	1.7
87	<0.4	<0.4	<0.2	<0.4	<0.3	<0.4	<0.3
90/101	<2.3	<2.6	<1.4	<2.3	<1.8	<2.3	<1.9
95	<1.7	<2.0	<1.1	<1.7	<1.4	<1.8	1.7
99	5.1	8.1	7.2	5.2	4.4	1.3	0.93
104	<0.44	<0.49	<0.28	<0.43	<0.34	<0.44	<0.36
105	0.52	1.5	2.9	1.6	0.38	<0.4	<0.3
110	<0.5	<0.6	<0.3	<0.5	<0.4	<0.5	<0.4
114	0.54	1.4	1.1	0.83	0.64	<0.4	<0.3
118	6	18	22	12	15	1.6	1.5
123	<0.44	<0.49	<0.28	<0.43	<0.34	<0.44	<0.36
138	39	62	61	55	42	9.3	11
141	<2.0	<2.2	<1.2	<1.9	<1.5	<2.0	<1.6
149	<7.4	<8.3	<4.6	<7.2	<5.8	<7.4	<6.1
151	<2.8	<3.2	<1.8	<2.7	<2.2	<2.8	<2.3
153	67	90	80	83	75	13	18
155	<0.44	<0.49	<0.28	<0.43	<0.34	<0.44	<0.36
156	8.4	9.4	9.1	8.4	7.8	3.6	2
157	1.4	2.3	1.7	1.5	1.6	<0.4	<0.3
158	1.6	<0.4	0.52	<0.4	<0.3	<0.4	<0.3
167	1.5	4.4	4.2	3	3.7	<0.4	0.39
170	25	24	23	23	21	5.3	5.8
174	<2.5	<2.8	<1.5	<2.4	<1.9	<2.5	<2.0
180	63	59	52	58	50	9.2	13
183	4.4	5.5	6	4.9	4.5	<2.1	<1.8
187	11	12	12	16	13	<3.2	3.1
188	<0.44	<0.49	<0.28	<0.43	<0.34	<0.44	<0.36
189	<0.4	<0.4	<0.2	<0.4	<0.3	<0.4	<0.3
194	10	6.4	6.9	8.4	7.2	0.87	1.4
199	<0.44	<0.49	<0.28	<0.43	<0.34	<0.44	<0.36
203	7.1	<5.3	5.8	5.7	5.3	<4.7	<3.9

Appendix 1, Table 16 - PCB concentrations found in serum samples

Sample	WIN1	WIN2	WIN3	WIN4	WIN5	WIN6	WIN7
PCB	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid
18	<1.0	<1.0	<0.9	<1	<1.1	<1.2	<0.9
22	<1.1	<1.0	<0.9	<1.0	<1.1	<1.2	<0.9
28	5.3	2.1	1.2	2	<1.2	4.2	1.3
31	1.2	<0.6	<0.6	0.8	<0.7	<0.7	<0.5
41/64	0.51	1.2	<0.4	<0.4	<0.4	<0.5	0.51
44	<0.4	<0.4	<0.4	<0.4	<0.4	<0.5	<0.3
49	<1.31	<1.3	<1.16	<1.2	<1.32	<1.46	<1.09
52	0.68	0.49	<0.41	<0.42	<0.46	0.82	0.51
54	<0.46	<0.46	<0.41	<0.42	<0.46	<0.51	<0.38
60/56	<32	<93	<15	<58	<18	<19	<14
70	1.6	0.53	0.87	<0.42	<0.46	<0.51	1.9
74	5.4	11	8.9	2.2	2	27	2.1
87	<0.4	<0.4	<0.4	<0.4	<0.4	<0.5	<0.3
90/101	<2.4	<2.4	<2.1	<2.2	<2.5	<2.7	<2.0
95	<1.8	<1.8	<1.6	<1.7	<1.9	<2.0	<1.5
99	3.3	9	5.8	1.9	3.9	6.4	2.3
104	<0.46	<0.46	<0.41	<0.42	<0.46	<0.51	<0.38
105	0.75	6.5	1.1	<0.4	<0.4	0.77	0.4
110	<0.6	<0.6	<0.5	<0.5	<0.6	<0.6	<0.5
114	<0.4	1.4	0.78	<0.4	<0.4	0.58	<0.3
118	7.1	22	12	2.3	3.4	5.9	3.9
123	<0.46	<0.46	<0.41	<0.42	<0.46	<0.51	<0.38
138	23	72	50	15	36	27	21
141	<2.1	<2.1	<1.8	<1.9	<2.1	<2.3	<1.7
149	<7.8	<7.7	<6.9	<7.1	<7.8	<8.6	<6.5
151	<3.0	<2.9	<2.6	<2.7	<3.0	<3.3	<2.5
153	34	117	78	22	59	38	37
155	<0.46	<0.46	<0.41	<0.42	<0.46	<0.51	<0.38
156	4.5	9	7.3	2.4	6.6	3.6	3.8
157	0.71	2.4	1.6	<0.4	1.4	<0.5	<0.3
158	<0.4	1.4	<0.4	<0.4	<0.4	<0.5	<0.3
167	1.8	6.1	3.3	0.54	0.99	0.8	0.85
170	11	26	19	6.7	19	11	16
174	<2.6	<2.6	<2.3	<2.4	<2.6	<2.9	<2.1
180	27	68	49	15	52	25	42
183	<2.2	6.5	4.7	<2.1	3.4	3.5	3.4
187	5.3	17	11	3.2	12	7.8	8.9
188	<0.46	<0.46	<0.41	<0.42	<0.46	<0.51	<0.38
189	<0.4	<0.4	<0.4	<0.4	<0.4	<0.5	<0.3
194	3.6	9.5	6	1.5	8	3.4	9.4
199	<0.46	<0.46	<0.41	<0.42	<0.46	<0.51	<0.38
203	<5	7.1	<4.4	<4.5	5.3	<5.5	6.4

Appendix 1, Table 17 - PCB concentrations found in serum samples

Sample	WIN8	WIN9	WIN10	BIR1	BIR2	BIR3	BIR4
PCB	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid
18	<1.4	<0.7	<0.8	<0.8	<0.9	<0.9	<0.8
22	<1.4	<0.7	<0.8	<0.9	<0.9	<0.9	<0.8
28	<1.5	1.7	2.5	1.5	1.7	2.8	3.6
31	<0.9	<0.4	<0.5	<0.5	<0.5	<0.6	<0.5
41/64	<0.5	<0.3	<0.3	<0.3	<0.3	<0.4	<0.3
44	<0.5	<0.3	<0.3	<0.3	<0.3	<0.4	<0.3
49	<1.7	<0.9	<1.01	<1.07	<1.11	<1.17	<1.03
52	<0.59	0.39	0.62	<0.38	<0.39	<0.41	<0.36
54	<0.59	<0.31	<0.35	<0.38	<0.39	<0.41	<0.36
60/56	<22	<12	<13	<14	<14	<51	<13
70	<0.59	0.53	<0.35	0.41	<0.39	0.44	0.74
74	2.7	7.9	8.7	4.9	4.7	5.1	15
87	<0.5	<0.3	<0.3	<0.3	<0.3	<0.4	<0.3
90/101	<3.2	<1.7	<1.9	<2.0	<2.1	<2.2	<1.9
95	<2.4	<1.2	1.5	<1.5	<1.5	<1.6	<1.4
99	1.6	3.4	4.8	2.2	3.9	3.7	9.8
104	<0.59	<0.31	<0.35	<0.38	<0.39	<0.41	<0.36
105	<0.5	0.61	0.89	<0.3	<0.3	<0.4	4.1
110	<0.8	<0.4	<0.4	<0.5	<0.5	<0.5	<0.4
114	<0.5	0.74	0.78	0.57	0.4	<0.4	1.2
118	2.8	9.7	10	4.9	5.7	6.3	29
123	<0.59	<0.31	<0.35	<0.38	<0.39	<0.41	<0.36
138	11	38	50	27	44	36	80
141	<2.7	<1.4	<1.6	<1.7	<1.7	<1.8	<1.6
149	<10.	<5.3	<6	<6.4	<6.6	<6.9	<6.1
151	<3.8	<2.0	<2.3	<2.4	<2.5	<2.6	<2.3
153	16	55	71	43	82	54	105
155	<0.59	<0.31	<0.35	<0.38	<0.39	<0.41	<0.36
156	1.4	7.7	8.1	7.1	9.1	6.1	10
157	<0.5	1.4	1.5	1.2	2	1.9	2.5
158	<0.5	0.38	<0.3	<0.3	<0.3	<0.4	1.3
167	<0.5	3	2.2	1.8	1.8	2.2	6
170	5.5	20	22	17	30	19	30
174	<3.4	<1.8	<2.0	<2.1	<2.2	<2.3	<2.0
180	12	47	54	45	87	49	74
183	<2.9	3	4.8	<1.8	4.8	2.9	8.5
187	<4.4	9.7	14	8.7	18	9.8	23
188	<0.59	<0.31	<0.35	<0.38	<0.39	<0.41	<0.36
189	<0.5	<0.3	<0.3	<0.3	<0.3	<0.4	<0.3
194	<0.7	7.9	8	7.7	18	6.5	12
199	<0.59	<0.31	<0.35	<0.38	<0.39	<0.41	<0.36
203	<6.4	4.8	5.3	<4.0	10	<4.4	11

Appendix 1, Table 18 - PCB concentrations found in serum samples

Sample	BIR5	BIR6	BIR7	BIR8	BIR9	BIR10	LEE1
PCB	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid
18	<0.7	<0.8	<0.7	<1.0	<1	<1	<0.7
22	<0.8	<0.8	<0.7	<1.0	<1.0	<1.0	<0.7
28	2.4	4.6	2.1	1.4	1.8	1.7	1.8
31	<0.5	1.4	<0.4	<0.6	<0.6	<0.6	<0.4
41/64	<0.3	<0.3	<0.3	<0.4	<0.4	<0.4	<0.2
44	<0.3	<0.3	<0.3	<0.4	<0.4	<0.4	<0.2
49	<0.95	<0.98	<0.87	<1.3	<1.21	<1.21	<0.84
52	0.41	0.73	<0.31	<0.45	<0.42	0.5	<0.29
54	<0.33	<0.34	<0.31	<0.45	<0.42	<0.42	<0.29
60/56	<12	<13	<11	<17	<16	<16	<11
70	0.61	0.8	<0.31	<0.45	<0.42	<0.42	<0.29
74	16	18	3.4	2.1	3.5	4	10
87	<0.3	<0.3	<0.3	<0.4	<0.4	<0.4	<0.2
90/101	<1.8	<1.8	<1.6	<2.4	<2.2	<2.2	<1.5
95	1.4	2.1	<1.2	2	<1.7	<1.7	<1.2
99	5.5	8.3	4	1.9	2.7	4.5	5.5
104	<0.33	<0.34	<0.31	<0.45	<0.42	<0.42	<0.29
105	1.7	2.5	0.86	<0.4	1.2	<0.4	<0.2
110	<0.4	<0.4	<0.4	<0.6	<0.5	<0.5	<0.4
114	0.93	0.66	<0.3	<0.4	<0.4	<0.4	<0.2
118	16	17	5.7	2.5	6	6.9	13
123	<0.33	<0.34	<0.31	<0.45	<0.42	<0.42	<0.29
138	54	65	26	26	28	28	48
141	<1.5	<1.5	<1.4	<2.0	<1.9	<1.9	<1.3
149	<5.6	<5.8	<5.2	<7.7	<7.1	<7.2	<5.0
151	<2.1	<2.2	<2	<2.9	<2.7	<2.7	<1.9
153	82	85	37	37	41	36	63
155	<0.33	<0.34	<0.31	<0.45	<0.42	<0.42	<0.29
156	9.8	11	5.2	4.4	5.7	3.1	6.4
157	1.8	1.9	0.46	1.1	0.69	<0.4	0.49
158	<0.3	<0.3	<0.3	<0.4	<0.4	<0.4	0.37
167	3.9	3.3	0.98	0.82	1.9	1.3	2.1
170	19	20	9	13	14	5.9	18
174	<1.9	<1.9	<1.7	<2.6	<2.4	<2.4	<1.6
180	58	55	25	39	34	20	46
183	5.5	5.9	3.2	2.9	2.7	2.6	5.5
187	12	14	5.2	8.8	6.9	6.4	10
188	<0.33	<0.34	<0.31	<0.45	<0.42	<0.42	<0.29
189	<0.3	<0.3	<0.3	<0.4	<0.4	<0.4	<0.2
194	8.5	7.4	3.5	7.1	5	2	7.1
199	<0.33	<0.34	<0.31	<0.45	<0.42	<0.42	<0.29
203	6.7	5.8	<3.3	<4.9	<4.5	<4.6	6.3

Appendix 1, Table 19 - PCB concentrations found in serum samples

Sample	LEE2	LEE3	LEE4	LEE5	LEE6	LEE7	LEE8
PCB	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid
18	<1.2	<1.0	<1.1	<0.7	<1.0	<1.0	<0.7
22	<1.2	<1.0	<1.1	<0.7	<1.0	<1.0	<0.7
28	<1.3	<1.1	<1.2	1.1	1.7	5.2	1.8
31	<0.7	<0.6	<0.7	<0.4	<0.6	1.1	1.8
41/64	<0.5	<0.4	<0.4	<0.3	<0.4	<0.4	<0.3
44	<0.5	<0.4	<0.4	<0.3	<0.4	<0.4	<0.3
49	<1.47	<1.23	<1.34	<0.92	<1.29	2.2	2.3
52	<0.52	<0.43	<0.47	<0.32	0.49	0.7	<0.33
54	<0.52	<0.43	<0.47	<0.32	<0.45	<0.45	<0.33
60/56	<19	<16	<17	<12	<17	<17	<12
70	<0.52	<0.43	<0.47	<0.32	<0.45	<0.45	<0.33
74	2.6	4.6	2.8	1.8	3.2	12	4.4
87	<0.5	<0.4	<0.4	<0.3	<0.4	<0.4	<0.3
90/101	4.4	<2.3	<2.5	<1.7	<2.4	2.7	<1.7
95	3	1.8	<1.9	1.8	<1.8	1.9	<1.3
99	2.7	6.4	1.2	1.3	2.7	8.3	2.5
104	<0.52	<0.43	<0.47	<0.32	<0.45	<0.45	<0.33
105	<0.5	<0.4	<0.4	<0.3	<0.4	2.3	<0.3
110	<0.6	<0.5	<0.6	<0.4	<0.6	<0.6	<0.4
114	<0.5	<0.4	<0.4	<0.3	<0.4	1	<0.3
118	2.7	11	3.4	2.5	5.7	15	1.8
123	<0.52	<0.43	<0.47	<0.32	<0.45	<0.45	<0.33
138	40	42	13	12	25	64	40
141	<2.3	<1.9	<2.1	<1.4	<2.0	<2.0	<1.5
149	12	<7.3	<7.9	<5.4	<7.7	<7.5	<5.5
151	5.7	<2.8	<3.0	<2.1	<2.9	4.7	<2.1
153	63	59	24	15	38	91	65
155	<0.52	<0.43	<0.47	<0.32	<0.45	<0.45	<0.33
156	8	7.5	<0.4	2.9	<0.4	14	9.9
157	0.91	0.84	0.68	<0.3	0.59	2.2	2.1
158	<0.5	<0.4	<0.4	<0.3	<0.4	2.2	<0.3
167	<0.5	1.3	1.5	0.57	1.1	3.5	1.4
170	18	14	7	3.5	9.7	22	22
174	6.8	<2.4	<2.6	<1.8	<2.6	<2.5	<1.8
180	47	39	20	11	31	71	76
183	5.8	4.3	<2.3	<1.6	3.2	8.5	2.9
187	16	8.4	<3.4	4.4	7.6	15	14
188	<0.52	<0.43	<0.47	<0.32	<0.45	<0.45	<0.33
189	<0.5	<0.4	<0.4	<0.3	<0.4	<0.4	<0.3
194	7.4	5.7	2.5	0.93	3.9	9.4	12
199	<0.52	<0.43	<0.47	<0.32	<0.45	<0.45	<0.33
203	<5.6	6.3	7.5	<3.5	<4.9	10	10

Appendix 1, Table 20 - PCB concentrations found in serum samples

Sample	LEE9	LEE10	NEW1	NEW2	NEW3	NEW4	NEW5
PCB	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid
18	<0.9	<1.0	<1.0	<1.2	<1.0	<1.3	<0.8
22	<0.9	<1.0	<1.0	<1.2	<1.0	<1.3	<0.8
28	1.7	2.9	3	3.2	14	3.8	14
31	<0.6	0.92	0.88	<0.8	<0.6	3.5	8.8
41/64	<0.4	<0.4	<0.4	<0.5	<0.4	<0.5	<0.3
44	<0.4	<0.4	<0.4	<0.5	<0.4	<0.5	<0.3
49	1.4	<1.25	<1.22	<1.51	<1.24	2.8	2.3
52	<0.41	0.48	0.59	<0.53	<0.43	0.57	0.62
54	<0.41	<0.44	0.51	<0.53	<0.43	<0.55	<0.34
60/56	<15	<16	<16	<20	<16	<21	<13
70	<0.41	<0.44	1.1	<0.53	0.58	<0.55	1.9
74	1.4	4.3	8.9	8.2	24	9.1	0.35
87	<0.4	<0.4	<0.4	<0.5	<0.4	<0.5	<0.3
90/101	2.7	<2.3	<2.3	<2.8	<2.3	<3	<1.8
95	<1.6	<1.7	<1.7	<2.1	<1.7	<2.2	<1.4
99	2.2	2.7	3.3	5.3	9.9	4.9	1.1
104	<0.41	<0.44	<0.43	<0.53	<0.43	<0.55	<0.34
105	0.52	<0.4	0.77	<0.5	4.8	<0.5	0.52
110	<0.5	<0.5	<0.5	<0.7	<0.5	<0.7	<0.4
114	<0.4	<0.4	0.81	<0.5	0.81	<0.5	<0.3
118	2.2	5.9	8.5	9.2	27	6.4	1.4
123	<0.41	<0.44	<0.43	<0.53	<0.43	<0.55	<0.34
138	21	21	33	43	57	50	9.4
141	<1.8	<2.0	<1.9	<2.4	<2	<2.5	<1.5
149	<6.9	<7.4	<7.2	<8.9	<7.3	<9.4	<5.8
151	<2.6	<2.8	<2.8	<3.4	<2.8	<3.6	<2.2
153	33	29	49	64	69	76	16
155	<0.41	<0.44	<0.43	<0.53	<0.43	<0.55	<0.34
156	<0.4	3.3	7	6.9	7.1	11	1.2
157	1.3	0.44	1.3	1	1.2	1.3	<0.3
158	0.64	<0.4	<0.4	<0.5	0.48	<0.5	0.48
167	<0.4	0.53	2.6	2.5	3.8	2.5	<0.3
170	8.6	5.9	15	13	13	20	6.1
174	<2.3	<2.5	<2.4	<3.0	<2.4	<3.1	<1.9
180	26	18	40	44	40	55	15
183	2.2	2.3	2.2	4.2	6.3	4.4	<1.7
187	7.2	<3.2	8.8	8.7	11	16	2.7
188	<0.41	<0.44	<0.43	<0.53	<0.43	<0.55	<0.34
189	<0.4	<0.4	<0.4	<0.5	<0.4	<0.5	<0.3
194	4.1	1.7	5.6	4.8	4.7	8.2	3.1
199	<0.41	<0.44	<0.43	<0.53	<0.43	<0.55	<0.34
203	<4.4	<4.7	<4.6	<5.7	6.1	9.2	<3.7

Appendix 1, Table 21 - PCB concentrations found in serum samples

Sample	NEW6	NEW7	NEW8	NEW9	BEL1	BEL2	BEL3	BEL4
PCB	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid
18	<1.0	<0.7	<0.8	<1.2	<1.3	<1.1	<1.2	<0.7
22	<1.0	<0.7	<0.8	<1.2	<1.3	1.2	<1.2	<0.7
28	3.8	3.1	2.9	2.1	6.8	13	3	1.9
31	6.5	<0.4	0.61	<0.8	7.9	10	1.9	0.74
41/64	<0.4	<0.3	<0.3	<0.5	<0.5	<0.5	<0.5	<0.3
44	<0.4	<0.3	<0.3	<0.5	<0.5	<0.5	<0.5	<0.3
49	1.3	1.1	2.3	2.9	3	1.8	3.9	2.6
52	<0.43	0.44	0.43	<0.53	<0.56	0.52	<0.52	<0.3
54	<0.43	<0.3	<0.35	<0.53	<0.56	<0.5	<0.52	<0.3
60/56	<16	<11	<13	<20	<21	<19	<19	<11
70	1.1	7.5	8	2.7	1.1	4	3	3.1
74	0.9	<0.3	<0.3	<0.5	0.67	<0.5	<0.5	<0.3
87	<0.4	0.33	<0.3	<0.5	<0.5	<0.5	<0.5	<0.3
90/101	<2.3	<1.6	<1.9	<2.8	<3.0	<2.6	<2.8	<1.6
95	<1.7	<1.2	<1.4	<2.1	<2.3	<2.0	<2.1	<1.2
99	0.67	7.9	4.1	0.88	0.76	2.1	2.1	2.5
104	<0.43	<0.3	<0.35	<0.53	<0.56	<0.5	<0.52	<0.3
105	0.69	2.1	0.36	<0.5	<0.5	1.4	<0.5	0.51
110	<0.5	<0.4	<0.4	<0.7	<0.7	<0.6	<0.7	<0.4
114	<0.4	1	1.3	1.5	<0.5	9.2	4.9	3.8
118	0.61	16	6.5	1.1	0.58	7.3	3.5	3.5
123	<0.43	<0.3	<0.35	<0.53	<0.56	<0.5	<0.52	<0.3
138	22	105	53	9.3	<8.7	22	14	26
141	<1.9	<1.4	<1.6	<2.4	<2.5	<2.2	<2.3	<1.4
149	<7.2	<5.1	<6.0	<8.9	<9.5	<8.4	<8.8	<5.1
151	<2.7	<1.9	<2.3	<3.4	<3.6	<3.2	<3.3	<1.9
153	12	172	83	16	<9.2	32	18	43
155	<0.43	<0.3	<0.35	<0.53	<0.56	<0.5	<0.52	<0.3
156	12	18	13	3.8	0.63	3.7	2.8	4.4
157	1.1	3.8	1	1.6	<0.5	<0.5	<0.5	0.54
158	1.5	1.4	0.5	2	<0.5	0.77	<0.5	0.32
167	<0.4	4.7	2.1	<0.5	<0.5	1.7	<0.5	<0.3
170	4.2	61	25	<3.0	<3.2	9.8	3.6	15
174	<2.4	<1.7	<2.0	<3.0	<3.2	<2.8	<2.9	<1.7
180	10	159	59	8.9	<5.7	26	10	41
183	<2.1	9.4	4.1	<2.6	<2.8	<2.4	<2.5	2.5
187	<3.1	43	13	<3.9	<4.1	6.7	<3.8	7.2
188	<0.43	<0.3	<0.35	<0.53	<0.56	<0.5	<0.52	<0.3
189	<0.4	<0.3	<0.3	<0.5	<0.5	<0.5	<0.5	<0.3
194	1.5	28	8.3	<0.7	<0.7	4.3	<0.6	7.4
199	<0.43	<0.3	<0.35	<0.53	<0.56	<0.5	<0.52	<0.3
203	<4.6	19	11	<5.7	<6.1	<5.4	<5.6	8.5

Appendix 1, Table 22 - PCB concentrations found in serum samples

Sample	BEL4	BEL5	BEL6	BEL7	BEL8	BEL9	BEL10
PCB	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid
18	<0.7	<0.8	<1.0	<0.8	<0.7	<1.4	<1.1
22	<0.7	<0.8	<1.0	<0.8	<0.7	<1.4	<1.1
28	1.9	2.6	<1.1	1.1	4.4	2.1	3.5
31	0.74	<0.5	<0.6	<0.5	<0.5	1.2	3.4
41/64	<0.3	21	<0.4	<0.3	<0.3	<0.6	<0.4
44	<0.3	<0.3	<0.4	<0.3	<0.3	<0.6	<0.4
49	2.6	1.1	<1.28	<0.96	2	2.4	2.7
52	<0.3	<0.37	<0.45	<0.34	0.51	<0.6	0.69
54	<0.3	<0.37	<0.45	<0.34	<0.33	<0.6	<0.47
60/56	<11	<14	<17	<12	<12	<22	<17
70	3.1	12	1.5	2.7	19	3.3	1.4
74	<0.3	0.92	<0.4	<0.3	0.54	<0.6	<0.4
87	<0.3	<0.3	<0.4	<0.3	<0.3	<0.6	<0.4
90/101	<1.6	<2	<2.4	<1.8	<1.7	<3.2	<2.5
95	<1.2	<1.5	<1.8	<1.3	<1.3	<2.4	<1.9
99	2.5	6.3	1.4	2.2	6.4	3.5	1.3
104	<0.3	<0.37	<0.45	<0.34	<0.33	<0.6	<0.47
105	0.51	0.94	<0.4	<0.3	3.5	0.75	<0.4
110	<0.4	<0.5	<0.6	<0.4	<0.4	<0.8	<0.6
114	3.8	1.1	0.47	<0.3	<0.3	1.5	<0.4
118	3.5	9.3	1.8	4	20	3.6	1.6
123	<0.3	<0.37	<0.45	<0.34	0.36	<0.6	<0.47
138	26	55	12	17	57	15	<7.2
141	<1.4	<1.7	<2.0	<1.5	<1.5	<2.7	<2.1
149	<5.1	<6.3	<7.6	<5.7	<5.6	<10.	<7.9
151	<1.9	<2.4	<2.9	<2.2	<2.1	<3.9	<3.0
153	43	89	20	28	81	21	14
155	<0.3	<0.37	<0.45	<0.34	<0.33	<0.6	<0.47
156	4.4	11	2.3	4.5	6.9	3	1
157	0.54	1.3	<0.4	<0.3	1.4	<0.6	<0.4
158	0.32	1.1	<0.4	0.81	1.1	<0.6	0.51
167	<0.3	1.3	<0.4	<0.3	3.4	<0.6	<0.4
170	15	28	7.3	7.2	21	5.5	4.5
174	<1.7	<2.1	<2.5	<1.9	<1.9	<3.4	<2.6
180	41	74	18	18	53	12	13
183	2.5	5.6	<2.2	<1.6	4.5	<2.9	<2.3
187	7.2	17	<3.3	5.2	15	<4.4	<3.4
188	<0.3	<0.37	<0.45	<0.34	<0.33	<0.6	<0.47
189	<0.3	<0.3	<0.4	<0.3	<0.3	<0.6	<0.4
194	7.4	12	3.3	2.8	8.3	0.8	2.1
199	<0.3	<0.37	<0.45	<0.34	<0.33	<0.6	<0.47
203	8.5	12	7.8	<3.6	7.2	<6.4	<5.0

Appendix 1, Table 23 – Pesticide and PBDE concentrations found in serum samples

Sample	WWF 1	WWF 2	WWF 3	WWF 4	WWF 5	WWF 6	WWF 8
Pesticide	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid
α-chlordane	<0.95	<0.99	<0.86	<0.96	<1.2	<0.67	<0.75
γ-chlordane	<0.95	<0.99	<0.86	<0.96	<1.2	<0.67	<0.75
HCB	14	6.5	12	21	12	8.6	9.3
o,p'-DDD	<0.95	<0.99	<0.86	<0.96	<1.2	<0.67	<0.75
o,p'-DDT	<0.95	<0.99	<0.86	<0.96	<1.2	<0.67	<0.75
o,p'-DDE	<0.95	<0.99	<0.86	<0.96	<1.2	<0.67	<0.75
p,p'-DDD	<0.95	<0.99	<0.86	<0.96	<1.2	<0.67	<0.75
p,p'-DDE	49	47	139	192	59	57	364
p,p'-DDT	1.1	<0.9	6.6	2.6	<1.2	0.8	29
α-HCH	<0.95	<0.99	1.1	1.3	<1.2	<0.67	1.7
β-HCH	9.1	3.4	5.8	15	17	8.1	14
γ-HCH	16	11	47	13	25	<3.9	<4.4
PBDE							
17	<0.24	<0.25	<0.22	<0.24	<0.31	<0.17	<0.19
28	0.25	<0.2	<0.2	<0.2	<0.3	<0.1	<0.1
32	<0.24	11	0.31	0.29	<0.31	<0.17	0.24
35	<0.24	<0.25	<0.22	<0.24	<0.31	<0.17	<0.19
37	<0.24	<0.25	<0.22	<0.24	<0.31	0.17	<0.19
47	2	4.4	2	2.6	5.1	2.4	1.2
49	0.27	<0.2	<0.2	<0.2	<0.3	<0.1	<0.1
66	0.71	<0.2	3.2	2.5	<0.3	<0.1	<0.1
71	<0.24	<0.25	<0.22	<0.24	<0.31	<0.17	<0.19
75	0.3	<0.2	<0.2	<0.2	<0.3	<0.1	<0.1
77	0.86	<0.2	<0.2	<0.2	<0.3	<0.1	<0.1
85	1.2	<0.2	<0.2	<0.2	<0.3	<0.1	<0.1
99	1.7	1.1	0.4	1	1.5	0.67	0.76
100	0.69	0.59	0.32	0.56	0.59	0.45	0.28
119	0.88	<0.2	<0.2	<0.2	<0.3	<0.1	<0.1
138	0.47	<0.2	<0.2	<0.2	<0.3	<0.1	<0.1
153	3.2	87	2.2	3.6	1.1	1.9	1.2
154	2.7	0.39	0.92	1	<0.3	0.51	0.7
166	<0.24	<0.25	<0.22	<0.24	<0.31	<0.17	<0.19
181	0.89	<0.2	<0.2	<0.2	<0.3	<0.1	<0.1
183	1.4	0.42	0.27	0.28	0.49	<0.1	0.27
190	<0.24	<0.25	<0.22	<0.24	<0.31	<0.17	<0.19
209	<25	<26	<23	<25	<33	<17	<20

Appendix 1, Table 24 – Pesticide and PBDE concentrations found in serum samples

Sample	WWF 10	WWF 11	WWF 13	WWF 14	WWF 15	WWF 16	WWF 17
Pesticide	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid
α-chlordane	<0.66	<1.7	<0.92	<1.6	<1.0	<0.81	<1.3
γ-chlordane	<0.66	<1.7	<0.92	<1.6	<1.0	<0.81	<1.3
HCB	15	17	21	8.6	20	15	11
o,p'-DDD	<0.66	<1.7	<0.92	<1.6	<1.0	<0.81	<1.3
o,p'-DDT	0.93	<1.7	<0.92	<1.6	<1.0	<0.81	<1.3
o,p'-DDE	1.5	<1.7	<0.92	<1.6	2.9	<0.81	3.9
p,p'-DDD	<0.66	<1.7	<0.92	20	<1.0	<0.81	<1.3
p,p'-DDE	112	71	205	39	268	59	191
p,p'-DDT	8.3	<1.7	3.3	<1.6	3.3	1.5	16
α-HCH	4.1	<1.7	1.6	<1.6	4.2	2	23
β-HCH	14	16	18	2.1	27	14	8.5
γ-HCH	20	<10	<5.4	<1.6	14	<4.7	<7.6
PBDE							
17	<0.16	<0.43	<0.23	<1.68	<0.27	<0.2	<0.32
28	<0.1	<0.4	<0.2	<1.6	<0.2	3	<0.3
32	<0.16	0.55	0.28	<1.68	0.34	0.21	0.33
35	<0.16	<0.43	<0.23	<1.68	<0.27	<0.2	<0.32
37	<0.16	<0.43	<0.23	<1.68	0.42	0.29	<0.32
47	1.4	2.5	1.1	<1.6	1.5	106	3.3
49	<0.1	<0.4	<0.2	<1.6	<0.2	0.58	<0.3
66	<0.1	<0.4	<0.2	<1.6	<0.2	<0.2	<0.3
71	<0.16	<0.43	<0.23	<1.68	<0.27	<0.2	<0.32
75	<0.1	<0.4	<0.2	<1.6	<0.2	<0.2	<0.3
77	<0.1	<0.4	<0.2	<1.6	<0.2	<0.2	<0.3
85	<0.1	<0.4	<0.2	<1.6	<0.2	3	<0.3
99	<0.1	<0.4	<0.2	<1.6	0.38	29	2.1
100	0.39	0.56	0.42	<1.6	0.49	14	0.69
119	0.45	<0.4	<0.2	<1.6	<0.2	0.43	<0.3
138	<0.1	<0.4	<0.2	<1.6	<0.2	<0.2	<0.3
153	0.91	5.2	2.4	2.4	2	8.1	5.7
154	0.17	0.46	0.8	<1.6	1.7	2	0.7
166	<0.16	<0.43	<0.23	<1.68	<0.27	<0.2	<0.32
181	<0.1	<0.4	<0.2	<1.6	<0.2	<0.2	<0.3
183	0.19	0.53	0.3	0	0.33	0.33	1.6
190	<0.16	<0.43	<0.23	<1.68	<0.27	<0.2	<0.32
209	<17	<46	<24	0	<28	<21	<34

Appendix 1, Table 25 – Pesticide and PBDE concentrations found in serum samples

Sample	WWF 18	WWF 19	WWF 20	WWF 21	LON1	LON2	LON3
Pesticide	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid
α-chlordane	<0.85	<1.01	<0.88	<0.87	<0.6	<1.0	<1.1
γ-chlordane	<0.85	<1.01	<0.88	<0.87	<0.6	<1.0	<1.1
HCB	15	10	7.8	22	9.8	<9.0	15
o,p'-DDD	<0.85	<1.01	<0.88	<0.87	<0.6	<1.0	<1.1
o,p'-DDT	<0.85	<1.01	<0.88	<0.87	<0.6	<1.0	<1.1
o,p'-DDE	1.1	<1.01	<0.88	<0.87	0.92	<1.0	1.7
p,p'-DDD	<0.85	<1.01	<0.88	<0.87	<0.6	<1.0	<1.1
p,p'-DDE	133	81	44	487	165	100	176
p,p'-DDT	5.4	2	2	9.1	43	3.8	6.2
α-HCH	3.8	1.1	0.98	<0.87	<0.6	<1.0	<1.1
β-HCH	25	7.2	6.3	22	21	10	23
γ-HCH	<5.0	21	<5.1	<5.1	7.8	19	21
PBDE							
17	<0.21	<0.25	<0.22	<0.22	<0.15	<0.27	<0.3
28	<0.2	<0.2	<0.2	<0.2	<0.1	<0.2	<0.3
32	0.27	0.28	0.24	<0.22	<0.21	<0.37	<0.41
35	<0.21	<0.25	<0.22	<0.22	<0.15	<0.27	<0.3
37	0.36	<0.25	<0.22	<0.22	<0.15	<0.27	<0.3
47	1.9	3	6.5	2.1	2.9	<0.5	0.75
49	<0.2	<0.2	<0.2	<0.2	<0.1	<0.2	<0.3
66	<0.2	<0.2	0.35	<0.2	<0.1	<0.2	<0.3
71	0.26	<0.25	<0.22	<0.22	<0.15	<0.27	<0.3
75	<0.2	<0.2	<0.2	<0.2	<0.1	<0.2	<0.3
77	<0.2	<0.2	<0.2	<0.2	<0.1	<0.2	<0.3
85	<0.2	<0.2	<0.2	<0.2	<0.1	<0.2	<0.3
99	0.65	<0.2	3.1	0.6	0.42	<0.6	<0.7
100	0.49	0.25	0.93	0.49	0.9	1.1	1.4
119	<0.2	<0.2	<0.2	<0.2	<0.1	<0.2	<0.3
138	<0.2	<0.2	<0.2	<0.2	<0.1	<0.2	<0.3
153	1.9	3	1.3	2.4	1.7	1.7	2.4
154	1	0.9	0.43	0.34	0.61	1	1.2
166	<0.21	<0.25	<0.22	<0.22	<0.15	<0.27	<0.3
181	<0.2	<0.2	<0.2	<0.2	<0.1	<0.2	<0.3
183	0.9	1.4	0.45	0.38	0.55	<0.2	<0.3
190	<0.21	<0.25	<0.22	<0.22	<0.15	<0.27	<0.3
209	<22	<27	<23	<23	<26	<46	<51

Appendix 1, Table 26 – Pesticide and PBDE concentrations found in serum samples

Sample	LON4	LON5	LON6	LON7	LON8	LON9	LON10
Pesticide	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid
α-chlordane	<1.1	<0.86	<1.14	<0.76	<0.73	<0.97	<0.92
γ-chlordane	<1.1	<0.86	<1.14	<0.76	<0.73	<0.97	<0.92
HCB	<9.5	<7.2	<9.6	<6.4	13	<8.2	<7.8
o,p'-DDD	<1.1	<0.86	<1.14	<0.76	<0.73	<0.97	<0.92
o,p'-DDT	<1.1	0.94	<1.14	<0.76	<0.73	<0.97	<0.92
o,p'-DDE	<1.1	<0.86	<1.14	<0.76	<0.73	<0.97	<0.92
p,p'-DDD	<1.1	<0.86	<1.14	<0.76	<0.73	<0.97	<0.92
p,p'-DDE	63	62	86	61	223	124	72
p,p'-DDT	<1.1	2.3	<1.1	4.4	7.3	16	4.9
α-HCH	<1.1	<0.86	<1.14	<0.76	<0.73	<0.97	<0.92
β-HCH	3.2	9.9	16	9.5	23	12	4.5
γ-HCH	25	<9.3	<12.	<8.3	<8	<10.	18
PBDE							
17	<0.28	<0.21	<0.28	<0.19	<0.18	<0.24	<0.23
28	<0.2	<0.2	<0.2	<0.1	<0.1	<0.2	<0.2
32	<0.39	<0.3	<0.39	<0.26	<0.25	<0.33	<0.32
35	<0.28	<0.21	<0.28	<0.19	<0.18	<0.24	<0.23
37	<0.28	<0.21	<0.28	<0.19	<0.18	<0.24	<0.23
47	<0.6	<0.4	1.2	<0.4	0.43	2	<0.4
49	<0.2	<0.2	<0.2	<0.1	<0.1	<0.2	<0.2
66	<0.2	<0.2	<0.2	<0.1	<0.1	<0.2	<0.2
71	<0.28	<0.21	<0.28	<0.19	<0.18	<0.24	<0.23
75	<0.2	<0.2	<0.2	<0.1	<0.1	<0.2	<0.2
77	<0.2	<0.2	<0.2	<0.1	<0.1	<0.2	<0.2
85	<0.2	<0.2	<0.2	<0.1	<0.1	<0.2	<0.2
99	<0.6	<0.5	<0.7	<0.4	0.52	<0.6	<0.5
100	1.2	0.83	1.1	0.84	0.79	0.89	0.86
119	<0.2	<0.2	<0.2	<0.1	<0.1	<0.2	<0.2
138	<0.2	<0.2	<0.2	<0.1	<0.1	<0.2	<0.2
153	2.6	1.2	1.5	2.1	1.7	2.1	0.73
154	1.1	0.69	1.2	0.82	0.64	0.8	0.63
166	<0.28	<0.21	<0.28	<0.19	<0.18	<0.24	<0.23
181	<0.2	<0.2	<0.2	<0.1	<0.1	<0.2	<0.2
183	<0.2	<0.2	<0.2	0.75	0.6	<0.2	<0.2
190	<0.28	<0.21	<0.28	<0.19	<0.18	<0.24	<0.23
209	<48	<37	<49	<33	<31	<42	<40

Appendix 1, Table 27 – Pesticide and PBDE concentrations found in serum samples

Sample	LON11	LON12	LON13	LON14	LON15	LON16	LON17
Pesticide	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid
α-chlordane	<6.67	<1.01	<0.83	<1.0	<1.0	<1.2	<0.92
γ-chlordane	<6.67	<1.01	<0.83	<1.0	<1.0	<1.2	<0.92
HCB	<56.	<8.5	16	<8.6	12	<10.	23
o,p'-DDD	<6.67	<1.01	<0.83	<1.0	<1.0	<1.2	<0.92
o,p'-DDT	<6.67	<1.01	<0.83	<1.0	2.5	<1.2	1.7
o,p'-DDE	<6.67	<1.01	<0.83	<1.0	<1.0	<1.2	<0.92
p,p'-DDD	<6.67	1.6	<0.83	<1.0	1.1	<1.2	<0.92
p,p'-DDE	83	69	100	45	846	59	495
p,p'-DDT	9.3	8.9	1.5	2.1	11	1.6	13
α-HCH	<6.67	<1.01	<0.83	<1.0	<1.0	<1.2	<0.92
β-HCH	<6.6	<1.0	30	4.4	30	3.3	33
γ-HCH	<72.	<11	<9.0	<11.	<11.	<13.	<10.
PBDE							
17	<1.67	<0.25	<0.21	<0.25	<0.26	<0.31	<0.23
28	<1.6	<0.2	<0.2	<0.2	<0.2	<0.3	<0.2
32	<2.3	<0.35	<0.29	<0.35	<0.36	<0.43	<0.32
35	<1.67	<0.25	<0.21	<0.25	<0.26	<0.31	<0.23
37	<1.67	<0.25	<0.21	<0.25	<0.26	<0.31	<0.23
47	<3.5	<0.5	<0.4	1	4.7	<0.6	0.68
49	<1.6	<0.2	<0.2	<0.2	<0.2	<0.3	<0.2
66	<1.6	<0.2	<0.2	<0.2	<0.2	<0.3	<0.2
71	<1.67	<0.25	<0.21	<0.25	<0.26	<0.31	<0.23
75	<1.6	<0.2	<0.2	<0.2	<0.2	<0.3	<0.2
77	<1.6	<0.2	<0.2	<0.2	<0.2	<0.3	<0.2
85	<1.6	<0.2	<0.2	<0.2	<0.2	<0.3	<0.2
99	<4.1	<0.6	<0.5	1.9	1.6	<0.7	<0.5
100	<1.6	1	0.97	1.7	1.9	1.2	1.4
119	<1.6	<0.2	<0.2	<0.2	<0.2	<0.3	<0.2
138	<1.6	<0.2	<0.2	<0.2	<0.2	<0.3	<0.2
153	6.1	1.8	1.3	1.5	2.3	1.6	1.9
154	<1.6	1.2	0.8	0.7	1.3	1.1	0.91
166	<1.67	<0.25	<0.21	<0.25	<0.26	<0.31	<0.23
181	<1.6	<0.2	<0.2	<0.2	<0.2	<0.3	<0.2
183	<1.6	<0.2	<0.2	<0.2	0.99	<0.3	0.83
190	<1.67	<0.25	<0.21	<0.25	<0.26	<0.31	<0.23
209	<29	<43	<36	<44	<46	<53	<40

Appendix 1, Table 28 – Pesticide and PBDE concentrations found in serum samples

Sample	LON18	LON19	HUN1	HUN2	HUN3	HUN4	HUN5
Pesticide	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid
α-chlordane	<0.92	<0.77	<0.56	<1.1	<0.75	<0.71	<0.78
γ-chlordane	<0.92	<0.77	<0.56	<1.1	<0.75	<0.71	<0.78
HCB	8.2	19	11	<9.3	31	17	<6.5
o,p'-DDD	<0.92	<0.77	<0.56	<1.1	<0.75	<0.71	<0.78
o,p'-DDT	<0.92	0.93	<0.56	<1.1	<0.75	<0.71	1.3
o,p'-DDE	<0.92	<0.77	<0.56	<1.1	<0.75	<0.71	<0.78
p,p'-DDD	<0.92	<0.77	<0.56	<1.1	<0.75	<0.71	<0.78
p,p'-DDE	40	304	80	53	313	154	574
p,p'-DDT	1.8	6.9	1.9	1.5	6.7	4.4	3.1
α-HCH	<0.92	<0.77	<0.56	<1.1	<0.75	<0.71	<0.78
β-HCH	5.1	37	11	5.9	27	25	4.7
γ-HCH	<9.9	<8.3	<6.1	<11.	<8.1	<7.7	<8.4
PBDE							
17	<0.23	<0.19	<0.14	<0.27	<0.19	<0.18	<0.19
28	<0.2	0.29	<0.1	<0.2	<0.1	<0.1	<0.1
32	<0.32	0.71	<0.19	<0.38	<0.26	<0.24	<0.27
35	<0.23	<0.19	<0.14	<0.27	<0.19	<0.18	<0.19
37	<0.23	<0.19	<0.14	<0.27	<0.19	<0.18	<0.19
47	0.51	<0.4	<0.3	<0.5	<0.4	<0.3	1.1
49	<0.2	<0.1	<0.1	<0.2	<0.1	<0.1	<0.1
66	<0.2	<0.1	0.44	0.83	0.75	<0.1	0.56
71	<0.23	<0.19	<0.14	<0.27	<0.19	<0.18	<0.19
75	<0.2	<0.1	<0.1	<0.2	<0.1	<0.1	<0.1
77	<0.2	<0.1	<0.1	<0.2	<0.1	<0.1	<0.1
85	<0.2	<0.1	<0.1	<0.2	<0.1	<0.1	<0.1
99	<0.5	<0.4	<0.3	<0.6	<0.4	<0.4	1.8
100	0.91	0.68	0.5	0.93	0.64	0.55	1.1
119	<0.2	<0.1	<0.1	<0.2	<0.1	<0.1	<0.1
138	<0.2	<0.1	<0.1	<0.2	<0.1	<0.1	<0.1
153	1.1	1.3	0.85	1.5	0.89	0.9	1.2
154	0.54	0.6	0.47	0.68	0.52	0.46	0.82
166	<0.23	<0.19	<0.14	<0.27	<0.19	<0.18	<0.19
181	<0.2	<0.1	<0.1	<0.2	<0.1	<0.1	<0.1
183	<0.2	0.73	<0.1	<0.2	0.63	<0.1	1
190	<0.23	<0.19	<0.14	<0.27	<0.19	<0.18	<0.19
209	<39	<33	<24	<47	<32	<30	<33

Appendix 1, Table 29 – Pesticide and PBDE concentrations found in serum samples

Sample	HUN6	HUN7	HUN8	HUN9	HUN10	EXE1	EXE2
Pesticide	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid
α-chlordane	<0.65	<0.83	<0.79	<0.95	<1.1	<0.97	<0.66
γ-chlordane	<0.65	<0.83	<0.79	<0.95	<1.1	<0.97	<0.66
HCB	9.3	<7.0	<6.6	<8.0	<9.7	13	<5.6
o,p'-DDD	<0.65	<0.83	<0.79	<0.95	<1.1	<0.97	<0.66
o,p'-DDT	<0.65	<0.83	<0.79	<0.95	<1.1	<0.97	<0.66
o,p'-DDE	<0.65	<0.83	<0.79	<0.95	<1.1	<0.97	<0.66
p,p'-DDD	<0.65	<0.83	<0.79	<0.95	<1.1	<0.97	<0.66
p,p'-DDE	196	108	74	60	69	97	45
p,p'-DDT	7.4	1.7	1.2	2.9	1.3	4.5	1.6
α-HCH	<0.65	<0.83	<0.79	<0.95	<1.1	<0.97	<0.66
β-HCH	19	6	4.7	7.6	7.4	19	4.6
γ-HCH	<7.1	<9.0	<8.5	<10.	<12.	<10.	<7.1
PBDE							
17	<0.16	<0.21	<0.2	<0.24	<0.29	<0.24	<0.16
28	0.28	<0.2	<0.2	<0.2	<0.2	<0.2	<0.1
32	<0.22	<0.29	<0.27	<0.33	<0.39	<0.33	<0.23
35	<0.16	<0.21	<0.2	<0.24	<0.29	<0.24	<0.16
37	<0.16	<0.21	<0.2	<0.24	<0.29	<0.24	<0.16
47	0.8	1.4	<0.4	0.87	<0.6	0.63	0.54
49	<0.1	<0.2	<0.2	<0.2	<0.2	<0.2	<0.1
66	<0.1	<0.2	<0.2	0.87	<0.2	1	0.62
71	<0.16	<0.21	<0.2	<0.24	<0.29	<0.24	<0.16
75	<0.1	<0.2	<0.2	<0.2	<0.2	<0.2	<0.1
77	<0.1	<0.2	<0.2	<0.2	<0.2	<0.2	<0.1
85	<0.1	<0.2	<0.2	<0.2	<0.2	<0.2	<0.1
99	<0.4	0.61	<0.4	<0.5	1.9	4.6	2.3
100	0.72	1.3	0.58	1	0.85	<0.2	0.65
119	<0.1	<0.2	<0.2	<0.2	<0.2	<0.2	<0.1
138	<0.1	<0.2	<0.2	<0.2	<0.2	<0.2	<0.1
153	1.5	4.4	1.9	1.6	1.9	1.3	0.39
154	0.72	0.84	0.84	<0.2	1.5	1.4	0.78
166	<0.16	<0.21	<0.2	<0.24	<0.29	<0.24	<0.16
181	<0.1	<0.2	<0.2	<0.2	<0.2	<0.2	<0.1
183	0.55	0.8	0.69	0.76	<0.2	1.1	<0.1
190	<0.16	<0.21	<0.2	<0.24	<0.29	<0.24	<0.16
209	<28	<36	<34	<41	<49	<42	<28

Appendix 1, Table 30 – Pesticide and PBDE concentrations found in serum samples

Sample	EXE3	EXE4	EXE5	EXE6	EXE7	EXE8	EXE9
Pesticide	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid
α-chlordane	<0.93	<0.93	<1.1	<0.48	<1	<1.0	<0.91
γ-chlordane	<0.93	<0.93	<1.1	<0.48	<1	<1.0	<0.91
HCB	12	<7.9	<10.	6.2	11	<9.2	9.3
o,p'-DDD	<0.93	<0.93	<1.1	<0.48	<1	<1.0	<0.91
o,p'-DDT	<0.93	<0.93	<1.1	1.3	1.5	<1.0	<0.91
o,p'-DDE	<0.93	<0.93	<1.1	<0.48	1.2	<1.0	<0.91
p,p'-DDD	<0.93	<0.93	<1.1	0.49	<1	<1.0	<0.91
p,p'-DDE	167	78	134	105	376	70	135
p,p'-DDT	4.5	2.2	1.9	4.8	4.7	2.8	6.1
α-HCH	<0.93	<0.93	<1.1	<0.48	<1	<1.0	<0.91
β-HCH	16	5.7	5.5	8.1	8.7	4.8	8.2
γ-HCH	<10.	<10.	<12.	<5.2	110	<11.	<9.9
PBDE							
17	<0.23	<0.23	<0.3	0.3	<0.25	<0.27	<0.23
28	<0.2	0.31	0.37	9.7	<0.2	<0.2	<0.2
32	<0.32	<0.32	<0.41	<0.17	<0.34	<0.38	<0.31
35	<0.23	<0.23	<0.3	<0.12	<0.25	<0.27	<0.23
37	<0.23	<0.23	<0.3	<0.12	<0.25	<0.27	<0.23
47	0.69	<0.5	1.8	179	0.6	1.1	0.54
49	<0.2	<0.2	<0.3	1.7	<0.2	<0.2	<0.2
66	0.87	1.1	<0.3	1.4	<0.2	<0.2	<0.2
71	<0.23	<0.23	<0.3	<0.12	<0.25	<0.27	<0.23
75	<0.2	<0.2	<0.3	<0.1	<0.2	<0.2	<0.2
77	<0.2	<0.2	<0.3	<0.1	<0.2	<0.2	<0.2
85	<0.2	<0.2	<0.3	5.3	<0.2	<0.2	<0.2
99	<0.5	6.3	1.5	4	<0.6	3.6	<0.5
100	1.2	<0.2	1.7	36	1.4	1.1	0.79
119	<0.2	<0.2	<0.3	<0.1	<0.2	<0.2	<0.2
138	<0.2	<0.2	<0.3	0.82	<0.2	1.5	<0.2
153	4.9	1.8	3	15	1.7	2.1	2
154	1.2	1.4	0.96	4.4	0.98	1.9	<0.2
166	<0.23	<0.23	<0.3	<0.12	<0.25	2.9	<0.23
181	<0.2	<0.2	<0.3	<0.1	<0.2	<0.2	<0.2
183	<0.2	<0.2	<0.3	0.7	0.9	<0.2	<0.2
190	<0.23	<0.23	<0.3	<0.12	<0.25	<0.27	<0.23
209	<40	<40	<51	<20	<43	<47	<39

Appendix 1, Table 31 – Pesticide and PBDE concentrations found in serum samples

Sample	EXE10	EXE11	CAR1	CAR2	CAR3	CAR4	CAR5
Pesticide	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid
α-chlordane	<1.0	<0.81	<0.99	<0.71	<1.0	<0.93	<0.8
γ-chlordane	<1.0	<0.81	<0.99	<0.71	<1.0	<0.93	<0.8
HCB	<8.7	11	<8.4	12	<8.6	12	32
o,p'-DDD	<1.0	<0.81	<0.99	<0.71	<1.0	<0.93	<0.8
o,p'-DDT	<1.0	<0.81	<0.99	<0.71	<1.0	<0.93	3.5
o,p'-DDE	<1.0	<0.81	<0.99	<0.71	<1.0	<0.93	<0.8
p,p'-DDD	<1.0	<0.81	<0.99	<0.71	<1.0	<0.93	<0.8
p,p'-DDE	21	371	48	546	129	123	2557
p,p'-DDT	1.1	2.5	2.9	2.7	3.3	5.1	19
α-HCH	<1.0	<0.81	<0.99	<0.71	<1.0	<0.93	<0.8
β-HCH	1.9	19	2	30	13	30	44
γ-HCH	<11.	<8.7	<10.	<7.7	<11.	<10.	<8.7
PBDE							
17	<0.26	<0.2	<0.25	0.2	<0.25	<0.23	<0.2
28	<0.2	<0.2	<0.2	<0.1	<0.2	<0.2	0.43
32	<0.36	<0.28	<0.34	<0.24	<0.35	<0.32	<0.28
35	<0.26	<0.2	<0.25	<0.18	<0.25	<0.23	0.41
37	<0.26	<0.2	<0.25	<0.18	<0.25	<0.23	<0.2
47	1.1	0.92	0.61	1.9	0.83	0.55	3.3
49	<0.2	<0.2	<0.2	<0.1	<0.2	<0.2	<0.2
66	<0.2	<0.2	<0.2	<0.1	<0.2	<0.2	<0.2
71	<0.26	<0.2	<0.25	<0.18	<0.25	<0.23	<0.2
75	<0.2	<0.2	<0.2	<0.1	<0.2	<0.2	<0.2
77	<0.2	<0.2	17	<0.1	<0.2	<0.2	<0.2
85	<0.2	<0.2	<0.2	<0.1	<0.2	<0.2	<0.2
99	<0.6	<0.5	<0.6	0.58	<0.6	<0.5	1.8
100	1	0.72	0.97	1.2	0.92	1	1.5
119	<0.2	<0.2	<0.2	<0.1	<0.2	<0.2	<0.2
138	<0.2	<0.2	<0.2	<0.1	<0.2	<0.2	<0.2
153	<0.2	1.2	2	2.2	2.2	1.6	1.7
154	<0.2	<0.2	<0.2	0.7	0.81	0.84	0.88
166	<0.26	<0.2	<0.25	<0.18	<0.25	<0.23	<0.2
181	<0.2	<0.2	<0.2	<0.1	<0.2	<0.2	<0.2
183	<0.2	<0.2	<0.2	<0.1	<0.2	<0.2	<0.2
190	<0.26	<0.2	<0.25	<0.18	<0.25	<0.23	<0.2
209	<44	<35	<43	<30	<44	<40	<34

Appendix 1, Table 32 – Pesticide and PBDE concentrations found in serum samples

Sample	CAR6	CAR7	CAR8	CAR9	CAR10	CAR11	CAR12
Pesticide	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid
α-chlordane	<1.2	<0.94	<0.74	<0.72	<0.62	<0.87	<1.3
γ-chlordane	<1.2	<0.94	<0.74	<0.72	<0.62	<0.87	<1.3
HCB	<10.	8.5	17	8.1	5.4	13	<11.
o,p'-DDD	<1.2	<0.94	<0.74	<0.72	<0.62	<0.87	<1.3
o,p'-DDT	<1.2	<0.94	<0.74	<0.72	<0.62	<0.87	<1.3
o,p'-DDE	<1.2	<0.94	<0.74	<0.72	<0.62	<0.87	<1.3
p,p'-DDD	<1.2	<0.94	<0.74	<0.72	<0.62	<0.87	<1.3
p,p'-DDE	74	157	153	92	45	571	75
p,p'-DDT	2.2	3.5	5	1.8	2.3	3.9	4.1
α-HCH	<1.2	<0.94	<0.74	<0.72	<0.62	<0.87	<1.3
β-HCH	6.6	19	24	6.4	8.6	29	11
γ-HCH	<13.	13	<8.0	<7.8	6.8	<9.5	<14.
PBDE							
17	<0.31	<0.24	<0.18	<0.18	<0.15	<0.22	<0.34
28	0.39	<0.2	0.21	0.21	0.21	0.26	<0.3
32	<0.42	<0.32	<0.25	<0.25	<0.21	<0.3	<0.47
35	<0.31	<0.24	<0.18	<0.18	<0.15	<0.22	<0.34
37	<0.31	<0.24	<0.18	<0.18	<0.15	<0.22	<0.34
47	<0.6	<0.5	<0.4	<0.3	<0.3	0.7	<0.7
49	<0.3	<0.2	<0.1	<0.1	<0.1	<0.2	<0.3
66	<0.3	<0.2	<0.1	<0.1	<0.1	<0.2	<0.3
71	<0.31	<0.24	<0.18	<0.18	<0.15	<0.22	<0.34
75	<0.3	<0.2	<0.1	<0.1	<0.1	<0.2	<0.3
77	<0.3	<0.2	<0.1	<0.1	<0.1	<0.2	<0.3
85	<0.3	<0.2	<0.1	<0.1	<0.1	<0.2	<0.3
99	<0.7	<0.5	<0.4	<0.4	<0.3	<0.5	<0.8
100	1	0.87	0.71	0.62	0.64	0.95	1.1
119	<0.3	<0.2	<0.1	<0.1	<0.1	<0.2	<0.3
138	<0.3	<0.2	<0.1	<0.1	<0.1	<0.2	<0.3
153	3	1.8	1.3	1.7	1.1	1.3	2.2
154	1.3	0.9	0.69	0.55	<0.1	<0.2	1
166	<0.31	<0.24	<0.18	<0.18	<0.15	<0.22	<0.34
181	<0.3	<0.2	<0.1	<0.1	<0.1	<0.2	<0.3
183	<0.3	0.8	<0.1	<0.1	<0.1	<0.2	<0.3
190	<0.31	<0.24	<0.18	<0.18	<0.15	<0.22	<0.34
209	<53	<41	<32	<31	<26	<38	<59

Appendix 1, Table 33 – Pesticide and PBDE concentrations found in serum samples

Sample	CAR13	MAN1	MAN2	MAN3	MAN4	MAN5	MAN6
Pesticide	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid
α-chlordane	<0.74	<0.79	<0.91	<0.66	<0.62	<0.68	<0.99
γ-chlordane	<0.74	<0.79	<0.91	<0.66	<0.62	<0.68	<0.99
HCB	8.7	<6.7	<7.7	14	9.7	6.5	<8.3
o,p'-DDD	<0.74	<0.79	<0.91	<0.66	<0.62	<0.68	<0.99
o,p'-DDT	<0.74	1.4	<0.91	<0.66	<0.62	<0.68	<0.99
o,p'-DDE	<0.74	<0.79	<0.91	<0.66	<0.62	<0.68	<0.99
p,p'-DDD	<0.74	<0.79	<0.91	<0.66	<0.62	<0.68	<0.99
p,p'-DDE	140	123	24	129	47	96	95
p,p'-DDT	1.7	5.7	1	5	1.5	1.4	2
α-HCH	<0.74	<0.79	<0.91	<0.66	<0.62	<0.68	<0.99
β-HCH	11	9.6	3	19	12	5.4	22
γ-HCH	<8.0	<8.6	<9.9	<7.2	<6.8	<7.4	<10.
PBDE							
17	<0.19	<0.2	<0.23	<0.17	<0.16	<0.17	<0.25
28	0.25	1.7	<0.2	<0.1	0.2	0.2	<0.2
32	<0.26	<0.27	<0.31	<0.23	<0.22	<0.23	<0.34
35	<0.19	<0.2	<0.23	<0.17	<0.16	<0.17	<0.25
37	<0.19	<0.2	<0.23	<0.17	<0.16	<0.17	<0.25
47	1.4	9.7	0.58	0.4	0.91	2.1	<0.5
49	<0.1	<0.2	<0.2	<0.1	<0.1	<0.1	<0.2
66	<0.1	<0.2	0.88	<0.1	<0.1	0.81	<0.2
71	<0.19	<0.2	<0.23	<0.17	<0.16	<0.17	<0.25
75	<0.1	<0.2	<0.2	<0.1	<0.1	<0.1	<0.2
77	<0.1	0.64	<0.2	<0.1	<0.1	<0.1	<0.2
85	<0.1	0.92	<0.2	<0.1	<0.1	<0.1	<0.2
99	1.6	2.6	<0.5	0.62	2.6	0.75	<0.6
100	0.93	5.2	0.73	0.78	0.51	0.77	0.64
119	<0.1	<0.2	<0.2	<0.1	<0.1	<0.1	<0.2
138	<0.1	<0.2	<0.2	<0.1	<0.1	<0.1	<0.2
153	2.4	16	0.95	1.6	0.83	1	0.76
154	0.61	0.69	0.34	0.75	0.39	0.43	0.58
166	<0.19	<0.2	<0.23	<0.17	<0.16	<0.17	<0.25
181	<0.1	<0.2	<0.2	<0.1	<0.1	<0.1	<0.2
183	0.63	0.76	0.71	0.5	0.4	0.46	<0.2
190	<0.19	<0.2	<0.23	<0.17	<0.16	<0.17	<0.25
209	<32	<34	<39	<28	<27	<29	<42

Appendix 1, Table 34 – Pesticide and PBDE concentrations found in serum samples

Sample	MAN7	MAN8	MAN9	MAN10	MAN11	BRUSS1	EDIN1
Pesticide	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid
α-chlordane	<0.81	<0.98	<0.56	<0.73	<0.93	<0.89	<0.59
γ-chlordane	<0.81	<0.98	<0.56	<0.73	<0.93	<0.89	<0.59
HCB	11	<8.3	12	7.7	8.1	7.8	17
o,p'-DDD	<0.81	<0.98	<0.56	<0.73	<0.93	<0.89	<0.59
o,p'-DDT	<0.81	<0.98	<0.56	<0.73	<0.93	<0.89	0.73
o,p'-DDE	<0.81	<0.98	<0.56	<0.73	<0.93	<0.89	<0.59
p,p'-DDD	<0.81	<0.98	<0.56	<0.73	<0.93	1.2	<0.59
p,p'-DDE	515	181	138	60	88	91	203
p,p'-DDT	3.3	1.9	2.6	1.6	2.5	7.6	4.4
α-HCH	<0.81	<0.98	<0.56	<0.73	<0.93	<0.89	<0.59
β-HCH	11	8.4	12	8.7	8.1	<0.8	22
γ-HCH	<8.8	<10.	<6.1	<7.9	<10.	<9.7	<6.4
PBDE							
17	<0.2	<0.25	<0.14	<0.18	<0.23	<0.22	<0.15
28	<0.2	<0.2	<0.1	<0.1	0.43	<0.2	0.25
32	<0.28	<0.34	<0.19	<0.25	<0.32	<0.31	<0.2
35	<0.2	<0.25	<0.14	<0.18	<0.23	<0.22	<0.15
37	<0.2	<0.25	<0.14	<0.18	<0.23	<0.22	<0.15
47	1.2	<0.5	0.48	<0.3	0.69	<0.4	0.5
49	<0.2	<0.2	<0.1	<0.1	<0.2	<0.2	<0.1
66	1	1.2	0.63	0.78	<0.2	<0.2	<0.1
71	<0.2	<0.25	<0.14	<0.18	<0.23	<0.22	<0.15
75	<0.2	<0.2	<0.1	<0.1	<0.2	<0.2	<0.1
77	<0.2	<0.2	<0.1	<0.1	<0.2	<0.2	<0.1
85	<0.2	<0.2	<0.1	<0.1	<0.2	<0.2	<0.1
99	0.57	<0.6	<0.3	<0.4	13	<0.5	<0.3
100	0.82	0.74	0.52	0.56	<0.2	1.6	0.88
119	<0.2	<0.2	<0.1	<0.1	<0.2	<0.2	<0.1
138	<0.2	<0.2	<0.1	<0.1	<0.2	<0.2	<0.1
153	2	1.7	0.98	1.1	2.1	1.6	3.4
154	0.65	0.62	0.37	0.36	2	0.72	0.79
166	<0.2	<0.25	<0.14	<0.18	<0.23	<0.22	<0.15
181	<0.2	<0.2	<0.1	<0.1	<0.2	<0.2	<0.1
183	0.63	0.78	0.4	0.51	0.77	0.59	0.55
190	<0.2	<0.25	<0.14	<0.18	<0.23	<0.22	<0.15
209	<35	<42	<24	<31	155	<38	<25

Appendix 1, Table 35 – Pesticide and PBDE concentrations found in serum samples

Sample	EDIN2	EDIN3	EDIN4	EDIN5	EDIN6	EDIN7	EDIN8
Pesticide	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid
α-chlordane	<0.96	<0.56	<0.62	<1.1	<0.81	<0.87	<1.04
γ-chlordane	<0.96	<0.56	<0.62	<1.1	<0.81	<0.87	<1.04
HCB	20	<4.7	8.3	11	<6.8	8.7	<8.8
o,p'-DDD	<0.96	<0.56	<0.62	<1.1	<0.81	<0.87	<1.04
o,p'-DDT	<0.96	<0.56	<0.62	<1.1	<0.81	<0.87	<1.04
o,p'-DDE	<0.96	<0.56	<0.62	<1.1	<0.81	<0.87	<1.04
p,p'-DDD	<0.96	<0.56	<0.62	<1.1	<0.81	<0.87	<1.04
p,p'-DDE	192	81	79	53	22	63	34
p,p'-DDT	8.7	3.9	2.1	1.6	2.3	3.5	2.3
α-HCH	<0.96	<0.56	<0.62	<1.1	<0.81	<0.87	<1.04
β-HCH	27	3.9	11	10	2.7	7.7	7
γ-HCH	<10.	<6.1	<6.7	<12.	<8.8	<9.4	<11.
PBDE							
17	<0.24	<0.14	<0.16	<0.29	<0.2	<0.22	<0.26
28	0.64	<0.1	<0.1	<0.2	0.26	0.22	<0.2
32	<0.33	<0.19	<0.21	<0.39	<0.28	<0.3	<0.36
35	<0.24	<0.14	<0.16	<0.29	<0.2	<0.22	<0.26
37	<0.24	<0.14	<0.16	<0.29	<0.2	<0.22	<0.26
47	1.3	0.64	<0.3	0.84	2.2	1.4	<0.5
49	<0.2	<0.1	<0.1	<0.2	<0.2	<0.2	<0.2
66	<0.2	<0.1	<0.1	<0.2	<0.2	<0.2	<0.2
71	<0.24	<0.14	<0.16	<0.29	<0.2	<0.22	<0.26
75	<0.2	<0.1	<0.1	<0.2	<0.2	<0.2	<0.2
77	<0.2	<0.1	<0.1	<0.2	<0.2	<0.2	<0.2
85	<0.2	<0.1	<0.1	<0.2	<0.2	<0.2	<0.2
99	0.64	0.36	<0.3	<0.7	<0.5	<0.5	<0.6
100	0.7	1.1	0.49	1.3	5.4	1	<0.2
119	<0.2	<0.1	<0.1	<0.2	<0.2	<0.2	<0.2
138	<0.2	<0.1	<0.1	<0.2	<0.2	<0.2	<0.2
153	3.1	1.8	1.2	1.6	6.4	1.5	<0.2
154	1.5	2	0.5	0.55	1.3	0.53	0.52
166	<0.24	<0.14	<0.16	<0.29	<0.2	<0.22	<0.26
181	<0.2	<0.1	<0.1	<0.2	<0.2	<0.2	<0.2
183	0.92	0.73	0.43	0.65	<0.2	0.51	0.69
190	<0.24	<0.14	<0.16	<0.29	<0.2	<0.22	<0.26
209	<41	<24	<27	<49	<35	<37	<45

Appendix 1, Table 36 – Pesticide and PBDE concentrations found in serum samples

Sample	EDIN9	EDIN10	EDIN11	EDIN12	NOT1	NOT2	NOT3
Pesticide	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid
α-chlordane	<0.51	<0.98	<0.78	<0.76	<0.93	<0.99	<0.85
γ-chlordane	<0.51	<0.98	<0.78	<0.76	<0.93	<0.99	<0.85
HCB	7.3	<8.2	13	10	<7.8	<8.4	12
o,p'-DDD	<0.51	<0.98	<0.78	<0.76	20	<0.99	<0.85
o,p'-DDT	<0.51	<0.98	<0.78	0.87	8.4	<0.99	1.7
o,p'-DDE	<0.51	<0.98	<0.78	<0.76	<0.93	<0.99	<0.85
p,p'-DDD	<0.51	<0.98	<0.78	<0.76	<0.93	<0.99	1.8
p,p'-DDE	44	57	113	171	25	<11.	701
p,p'-DDT	3.3	2.2	3.4	3.1	1.9	1.3	11
α-HCH	<0.51	<0.98	<0.78	<0.76	<0.93	<0.99	<0.85
β-HCH	7.8	6.5	18	19	11	5.8	20
γ-HCH	<5.5	<10.	<8.5	<8.2	<10.	<10.	<9.2
PBDE							
17	<0.13	<0.24	<0.2	<0.19	<0.23	<0.25	<0.21
28	0.28	<0.2	<0.2	<0.1	<0.2	0.45	0.43
32	<0.17	<0.34	<0.27	<0.26	<0.32	<0.34	<0.29
35	<0.13	<0.24	<0.2	<0.19	<0.23	<0.25	<0.21
37	<0.13	<0.24	<0.2	<0.19	<0.23	<0.25	<0.21
47	2.3	<0.5	<0.4	1.3	0.52	<0.5	1.8
49	<0.1	<0.2	<0.2	<0.1	<0.2	<0.2	<0.2
66	<0.1	<0.2	<0.2	<0.1	<0.2	<0.2	<0.2
71	<0.13	<0.24	<0.2	<0.19	<0.23	<0.25	<0.21
75	<0.1	<0.2	<0.2	<0.1	<0.2	<0.2	<0.2
77	<0.1	<0.2	<0.2	<0.1	<0.2	<0.2	<0.2
85	<0.1	<0.2	<0.2	<0.1	<0.2	<0.2	<0.2
99	1.2	<0.6	0.92	1	<0.5	<0.6	0.77
100	0.86	0.65	1.1	0.87	0.65	1.4	1.3
119	<0.1	<0.2	<0.2	<0.1	<0.2	0.67	<0.2
138	<0.1	<0.2	<0.2	<0.1	<0.2	<0.2	<0.2
153	1.2	1.7	1.3	0.92	1	0.72	2.2
154	0.35	0.48	0.8	0.57	0.46	0.54	0.86
166	<0.13	<0.24	<0.2	<0.19	<0.23	<0.25	<0.21
181	<0.1	<0.2	<0.2	<0.1	<0.2	<0.2	<0.2
183	0.43	0.6	0.76	0.48	0.7	0.51	0.78
190	<0.13	<0.24	<0.2	<0.19	<0.23	<0.25	<0.21
209	<22	<42	<34	<32	241	<43	<36

Appendix 1, Table 37 – Pesticide and PBDE concentrations found in serum samples

Sample	NOT4	NOT5	NOT6	NOT7	NOT8	NOT9	NOT10
Pesticide	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid
α-chlordane	<0.59	<0.87	<0.98	<0.55	<0.85	<0.68	<0.88
γ-chlordane	<0.59	<0.87	<0.98	<0.55	<0.85	<0.68	<0.88
HCB	20	13	44	36	23	39	<7.4
o,p'-DDD	<0.59	<0.87	<0.98	<0.55	<0.85	<0.68	<0.88
o,p'-DDT	<0.59	<0.87	<0.98	<0.55	<0.85	<0.68	<0.88
o,p'-DDE	<0.59	<0.87	<0.98	<0.55	<0.85	<0.68	<0.88
p,p'-DDD	<0.59	<0.87	1.3	<0.55	<0.85	<0.68	<0.88
p,p'-DDE	212	236	696	451	462	1642	18
p,p'-DDT	4	7.1	12	9.7	17	73	0.92
α-HCH	<0.59	<0.87	<0.98	<0.55	<0.85	<0.68	<0.88
β-HCH	37	31	59	56	18	<0.6	4.8
γ-HCH	<6.4	<9.5	<10.	<6.0	<9.2	<7.4	<9.5
PBDE							
17	<0.15	<0.22	<0.25	<0.14	<0.21	<0.17	<0.22
28	<0.1	0.62	<0.2	<0.1	0.69	<0.1	<0.2
32	<0.2	<0.3	<0.34	<0.19	<0.29	0.87	<0.3
35	<0.15	<0.22	<0.25	<0.14	<0.21	0.98	<0.22
37	<0.15	<0.22	<0.25	<0.14	<0.21	<0.17	<0.22
47	0.62	1.3	0.56	<0.3	1.3	<0.3	<0.4
49	<0.1	<0.2	<0.2	<0.1	<0.2	<0.1	<0.2
66	<0.1	0.81	<0.2	<0.1	<0.2	<0.1	<0.2
71	<0.15	<0.22	<0.25	<0.14	<0.21	<0.17	<0.22
75	<0.1	<0.2	<0.2	<0.1	<0.2	<0.1	<0.2
77	<0.1	<0.2	<0.2	<0.1	<0.2	<0.1	<0.2
85	<0.1	<0.2	<0.2	<0.1	<0.2	<0.1	<0.2
99	0.48	1	<0.6	<0.3	<0.5	<0.4	<0.5
100	0.9	1.1	0.7	0.37	1.5	0.49	0.6
119	<0.1	<0.2	<0.2	<0.1	<0.2	<0.1	<0.2
138	<0.1	<0.2	<0.2	<0.1	<0.2	<0.1	<0.2
153	2.5	4	0.87	0.8	2.2	0.92	0.94
154	0.65	0.78	0.62	0.39	0.68	0.41	0.38
166	<0.15	<0.22	<0.25	<0.14	<0.21	<0.17	<0.22
181	<0.1	<0.2	<0.2	<0.1	<0.2	<0.1	<0.2
183	0.41	0.49	0.64	0.46	0.7	0.43	0.63
190	<0.15	<0.22	<0.25	<0.14	<0.21	<0.17	<0.22
209	<25	72	86	<24	<37	<29	<38

Appendix 1, Table 38 – Pesticide and PBDE concentrations found in serum samples

Sample	NOT11	WIN1	WIN2	WIN3	WIN4	WIN5	WIN6
Pesticide	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid
α-chlordane	<0.73	<0.92	<0.91	<0.81	<0.84	<0.93	<1.0
γ-chlordane	<0.73	<0.92	<0.91	<0.81	<0.84	<0.93	<1.0
HCB	9.7	16	14	19	10	<7.8	9.8
o,p'-DDD	<0.73	7.4	<0.91	9.3	<0.84	<0.93	<1.0
o,p'-DDT	<0.73	<0.92	1	0.9	2.6	<0.93	<1.0
o,p'-DDE	<0.73	<0.92	<0.91	<0.81	<0.84	<0.93	<1.0
p,p'-DDD	<0.73	<0.92	1.3	<0.81	<0.84	<0.93	<1.0
p,p'-DDE	35	78	383	556	35	99	166
p,p'-DDT	1.3	5.8	9	4.5	<0.8	1.8	5
α-HCH	<0.73	<0.92	<0.91	<0.81	<0.84	<0.93	<1.0
β-HCH	10	17	16	17	10	9.6	7.9
γ-HCH	<7.9	<10.	<9.9	<8.8	<9.1	<10.	<11.
PBDE							
17	<0.18	<0.23	<0.23	<0.2	<0.21	<0.23	<0.26
28	<0.1	0.44	0.52	<0.2	0.4	<0.2	0.75
32	<0.25	<0.32	<0.31	<0.28	<0.29	<0.32	<0.35
35	<0.18	<0.23	<0.23	<0.2	<0.21	<0.23	<0.26
37	<0.18	<0.23	<0.23	<0.2	<0.21	<0.23	<0.26
47	1.8	0.88	9.8	<0.4	<0.4	<0.5	1.7
49	<0.1	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2
66	<0.1	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2
71	<0.18	<0.23	<0.23	<0.2	<0.21	<0.23	<0.26
75	<0.1	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2
77	<0.1	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2
85	<0.1	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2
99	0.73	<0.5	2	<0.5	<0.5	<0.5	0.94
100	0.62	0.89	2.9	0.46	0.46	0.78	1.4
119	<0.1	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2
138	<0.1	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2
153	1.6	1.8	2.1	0.98	0.85	1.6	2.2
154	0.4	0.9	2	0.57	0.55	0.73	0.55
166	<0.18	<0.23	<0.23	<0.2	<0.21	<0.23	<0.26
181	<0.1	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2
183	0.38	0.62	0.98	0.52	<0.2	<0.2	0.62
190	<0.18	<0.23	<0.23	<0.2	<0.21	<0.23	<0.26
209	<31	<40	<39	<35	<36	<40	<44

Appendix 1, Table 39 – Pesticide and PBDE concentrations found in serum samples

Sample	WIN7	WIN8	WIN9	WIN10	BIR1	BIR2	BIR3
Pesticide	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid
α-chlordane	<0.77	<1.1	<0.63	<0.71	<0.75	<0.78	<0.82
γ-chlordane	<0.77	<1.1	<0.63	<0.71	<0.75	<0.78	<0.82
HCB	8.7	<10.	24	17	8.9	10	10
o,p'-DDD	7.9	<1.1	2.3	7.7	1.6	<0.78	<0.82
o,p'-DDT	1.1	<1.1	<0.63	<0.71	1.1	<0.78	<0.82
o,p'-DDE	<0.77	<1.1	<0.63	<0.71	<0.75	<0.78	<0.82
p,p'-DDD	<0.77	<1.1	<0.63	<0.71	<0.75	<0.78	<0.82
p,p'-DDE	88	37	101	317	70	253	80
p,p'-DDT	5.4	1.8	3.3	7.5	4.9	8.4	4.1
α-HCH	<0.77	<1.1	<0.63	<0.71	<0.75	<0.78	<0.82
β-HCH	3.2	4.6	32	30	6.2	3.3	5.3
γ-HCH	<8.3	<12.	<6.8	<7.6	<8.2	<8.4	<8.9
PBDE							
17	<0.19	<0.3	<0.16	<0.18	<0.19	<0.19	<0.21
28	<0.1	<0.3	0.16	<0.1	0.2	0.3	<0.2
32	<0.26	<0.41	<0.22	<0.24	<0.26	<0.27	<0.28
35	<0.19	<0.3	<0.16	<0.18	<0.19	<0.19	<0.21
37	<0.19	<0.3	<0.16	<0.18	<0.19	<0.19	<0.21
47	1.1	<0.6	<0.3	<0.3	1.4	6.6	<0.4
49	<0.1	<0.3	<0.1	<0.1	<0.1	<0.1	<0.2
66	<0.1	1.6	0.79	<0.1	<0.1	<0.1	<0.2
71	<0.19	<0.3	<0.16	<0.18	<0.19	<0.19	<0.21
75	<0.1	<0.3	<0.1	<0.1	<0.1	<0.1	<0.2
77	<0.1	<0.3	<0.1	<0.1	<0.1	<0.1	<0.2
85	<0.1	<0.3	<0.1	<0.1	<0.1	<0.1	<0.2
99	<0.4	147	<0.3	<0.4	0.47	2.2	<0.5
100	0.68	<0.3	0.57	0.26	1.2	3.6	1.1
119	<0.1	1.4	<0.1	<0.1	<0.1	<0.1	<0.2
138	<0.1	<0.3	<0.1	<0.1	<0.1	<0.1	<0.2
153	1.2	0.77	0.78	0.96	2.8	7.7	1.5
154	0.52	1.7	0.33	0.18	0.41	0.65	0.24
166	<0.19	<0.3	<0.16	<0.18	<0.19	<0.19	<0.21
181	<0.1	<0.3	<0.1	<0.1	<0.1	<0.1	<0.2
183	0.43	1.8	0.38	<0.1	<0.1	0.22	<0.2
190	<0.19	<0.3	<0.16	<0.18	<0.19	<0.19	<0.21
209	<33	<51	<27	<30	35	<33	<35

Appendix 1, Table 40 – Pesticide and PBDE concentrations found in serum samples

Sample	BIR4	BIR5	BIR6	BIR7	BIR8	BIR9	BIR10
Pesticide	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid
α-chlordane	<0.72	<0.67	<0.69	<0.61	<0.91	<0.84	<0.85
γ-chlordane	<0.72	<0.67	<0.69	<0.61	<0.91	<0.84	<0.85
HCB	27	28	52	12	<7.7	<7.1	<7.1
o,p'-DDD	<0.72	<0.67	<0.69	<0.61	<0.91	<0.84	<0.85
o,p'-DDT	<0.72	<0.67	<0.69	<0.61	<0.91	<0.84	<0.85
o,p'-DDE	<0.72	<0.67	<0.69	<0.61	<0.91	<0.84	<0.85
p,p'-DDD	<0.72	<0.67	<0.69	<0.61	<0.91	<0.84	<0.85
p,p'-DDE	647	889	580	89	91	63	107
p,p'-DDT	8.4	2.5	7.4	4.6	<0.9	3.5	2.6
α-HCH	<0.72	<0.67	<0.69	<0.61	<0.91	<0.84	<0.85
β-HCH	39	41	79	12	11	4.8	9.9
γ-HCH	<7.8	<7.2	<7.4	<6.6	<9.8	<9.2	<9.2
PBDE							
17	<0.18	<0.17	<0.17	<0.15	<0.23	<0.21	<0.21
28	<0.1	<0.1	0.38	0.19	<0.2	<0.2	<0.2
32	<0.25	<0.23	<0.24	<0.21	<0.31	<0.29	<0.29
35	<0.18	<0.17	<0.17	<0.15	<0.23	<0.21	<0.21
37	<0.18	<0.17	<0.17	<0.15	<0.23	<0.21	<0.21
47	1.1	1.9	4	4.5	0.57	0.98	2.2
49	<0.1	<0.1	<0.1	<0.1	<0.2	<0.2	<0.2
66	<0.1	<0.1	<0.1	<0.1	<0.2	<0.2	<0.2
71	<0.18	<0.17	<0.17	<0.15	<0.23	<0.21	<0.21
75	<0.1	<0.1	<0.1	<0.1	<0.2	<0.2	<0.2
77	<0.1	<0.1	<0.1	<0.1	<0.2	<0.2	<0.2
85	<0.1	<0.1	<0.1	<0.1	<0.2	<0.2	<0.2
99	<0.4	1.1	2.2	0.4	<0.5	<0.5	<0.5
100	0.61	0.67	0.85	1.5	0.47	0.43	0.66
119	<0.1	<0.1	<0.1	<0.1	<0.2	<0.2	<0.2
138	<0.1	<0.1	<0.1	<0.1	<0.2	<0.2	<0.2
153	0.91	0.72	1.1	1.9	1.8	2.1	1.4
154	0.3	0.4	0.22	<0.1	0.45	0.68	<0.2
166	<0.18	<0.17	<0.17	<0.15	<0.23	<0.21	<0.21
181	<0.1	<0.1	<0.1	<0.1	<0.2	<0.2	<0.2
183	<0.1	<0.1	<0.1	<0.1	<0.2	<0.2	0.23
190	<0.18	<0.17	<0.17	<0.15	<0.23	<0.21	<0.21
209	<31	115	58	<26	<39	83	<36

Appendix 1, Table 41 – Pesticide and PBDE concentrations found in serum samples

Sample	LEE1	LEE2	LEE3	LEE4	LEE5	LEE6	LEE7
Pesticide	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid
α-chlordane	<0.59	<1.0	<0.86	<0.94	<0.64	<0.91	<0.89
γ-chlordane	<0.59	<1.0	<0.86	<0.94	<0.64	<0.91	<0.89
HCB	9.9	<8.7	<7.3	<7.9	<5.4	11	50
o,p'-DDD	<0.59	<1.0	<0.86	<0.94	<0.64	<0.91	<0.89
o,p'-DDT	<0.59	<1.0	<0.86	<0.94	<0.64	<0.91	<0.89
o,p'-DDE	<0.59	<1.0	<0.86	<0.94	<0.64	<0.91	<0.89
p,p'-DDD	<0.59	<1.0	<0.86	<0.94	<0.64	<0.91	<0.89
p,p'-DDE	170	87	160	50	46	57	294
p,p'-DDT	1.2	1.6	1.7	0.95	<0.6	1.9	2.5
α-HCH	<0.59	<1.0	<0.86	<0.94	<0.64	<0.91	<0.89
β-HCH	25	9.6	9.1	12	4.4	19	28
γ-HCH	<6.4	<11.	<9.3	<10.	<7.0	<9.8	<9.7
PBDE							
17	<0.15	<0.26	<0.22	<0.23	<0.16	<0.23	<0.22
28	<0.1	<0.2	<0.2	<0.2	<0.1	<0.2	<0.2
32	<0.2	<0.36	<0.3	<0.32	<0.22	<0.31	<0.31
35	<0.15	1.1	<0.22	<0.23	<0.16	<0.23	<0.22
37	<0.15	<0.26	<0.22	<0.23	<0.16	<0.23	<0.22
47	<0.3	4.3	2.3	2.1	0.78	16	18
49	<0.1	<0.2	<0.2	<0.2	<0.1	<0.2	<0.2
66	<0.1	<0.2	<0.2	<0.2	<0.1	<0.2	<0.2
71	<0.15	<0.26	<0.22	<0.23	<0.16	<0.23	<0.22
75	<0.1	<0.2	<0.2	<0.2	<0.1	<0.2	<0.2
77	<0.1	<0.2	<0.2	<0.2	<0.1	<0.2	<0.2
85	<0.1	<0.2	<0.2	<0.2	<0.1	<0.2	<0.2
99	<0.3	2.2	0.93	1.8	0.44	1.3	8.1
100	0.32	0.72	0.82	0.65	0.49	1.3	4.6
119	<0.1	<0.2	<0.2	<0.2	<0.1	<0.2	<0.2
138	<0.1	<0.2	<0.2	<0.2	<0.1	<0.2	<0.2
153	1.9	2.4	1.5	1.5	0.98	1.8	4.4
154	0.55	0.84	0.6	0.49	0.51	0.37	1.3
166	<0.15	<0.26	<0.22	<0.23	<0.16	<0.23	<0.22
181	<0.1	<0.2	<0.2	<0.2	<0.1	<0.2	<0.2
183	<0.1	1.3	0.32	0.4	0.28	<0.2	0.45
190	<0.15	<0.26	<0.22	<0.23	<0.16	<0.23	<0.22
209	36	<44	<37	<40	<28	<39	<38

Appendix 1, Table 42 – Pesticide and PBDE concentrations found in serum samples

Sample	LEE8	LEE9	LEE10	NEW1	NEW2	NEW3	NEW4
Pesticide	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid
α-chlordane	<0.65	<0.81	<0.87	<0.86	<1.0	<0.87	<1.1
γ-chlordane	<0.65	<0.81	<0.87	<0.86	<1.0	<0.87	<1.1
HCB	48	31	22	40	27	72	62
o,p'-DDD	<0.65	<0.81	<0.87	<0.86	<1.0	<0.87	<1.1
o,p'-DDT	<0.65	<0.81	<0.87	<0.86	<1.0	<0.87	<1.1
o,p'-DDE	<0.65	<0.81	<0.87	<0.86	<1.0	<0.87	<1.1
p,p'-DDD	<0.65	<0.81	<0.87	<0.86	<1.0	<0.87	<1.1
p,p'-DDE	846	101	120	154	1010	669	239
p,p'-DDT	1.8	4.6	20	3.1	8.3	7.2	4.4
α-HCH	<0.65	<0.81	<0.87	<0.86	<1.0	<0.87	<1.1
β-HCH	22	14	13	31	33	80	13
γ-HCH	<7.1	<8.8	<9.5	<9.3	<11.	<9.4	<12.
PBDE							
17	<0.16	<0.2	<0.22	<0.21	<0.26	<0.22	<0.28
28	<0.1	0.3	0.32	<0.2	0.51	<0.2	<0.2
32	0.23	<0.28	<0.3	<0.29	<0.36	<0.3	<0.38
35	<0.16	<0.2	<0.22	<0.21	<0.26	<0.22	<0.28
37	<0.16	<0.2	<0.22	<0.21	<0.26	<0.22	<0.28
47	<0.3	1.2	9.1	<0.4	0.73	1.2	4.5
49	<0.1	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2
66	<0.1	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2
71	<0.16	<0.2	<0.22	<0.21	<0.26	<0.22	<0.28
75	<0.1	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2
77	<0.1	<0.2	7.1	<0.2	<0.2	4.3	<0.2
85	<0.1	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2
99	<0.4	<0.5	16	<0.5	<0.6	<0.5	0.73
100	0.25	0.65	100	<0.2	0.63	0.38	0.62
119	<0.1	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2
138	<0.1	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2
153	1.2	2	2.7	0.63	2.4	0.5	3.6
154	0.85	0.36	0.71	<0.2	0.34	<0.2	<0.2
166	<0.16	<0.2	0.32	<0.21	<0.26	<0.22	<0.28
181	<0.1	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2
183	0.32	0.43	<0.2	<0.2	<0.2	<0.2	<0.2
190	<0.16	<0.2	<0.22	<0.21	<0.26	<0.22	<0.28
209	37	<35	<38	<37	<45	<37	151

Appendix 1, Table 43 – Pesticide and PBDE concentrations found in serum samples

Sample	NEW5	NEW6	NEW7	NEW8	NEW9	BEL1	BEL2
Pesticide	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid
α-chlordane	<0.69	<0.85	<0.61	<0.71	<1.0	<1.1	<0.99
γ-chlordane	<0.69	<0.85	<0.61	<0.71	<1.0	<1.1	<0.99
HCB	24	25	26	50	43	42	34
o,p'-DDD	<0.69	<0.85	<0.61	13	49	7.2	7.2
o,p'-DDT	<0.69	<0.85	<0.61	<0.71	<1.0	<1.1	<0.99
o,p'-DDE	<0.69	<0.85	<0.61	<0.71	<1.0	<1.1	<0.99
p,p'-DDD	<0.69	3.3	<0.61	<0.71	<1.0	<1.1	<0.99
p,p'-DDE	25	15	252	157	28	15	51
p,p'-DDT	0.82	<0.8	4.4	1.5	<1.0	2.3	1
α-HCH	18	4.1	2.9	7.2	19	<1.1	2.6
β-HCH	16	7	21	43	38	16	18
γ-HCH	<7.5	<9.2	<6.6	<7.7	13	<12.	<10.
PBDE							
17	<0.17	<0.21	<0.15	<0.18	<0.26	<0.28	<0.25
28	<0.1	<0.2	<0.1	1	0.28	<0.2	<0.2
32	<0.24	<0.29	<0.21	<0.24	<0.36	0.75	<0.34
35	<0.17	<0.21	<0.15	<0.18	<0.26	<0.28	<0.25
37	<0.17	<0.21	<0.15	<0.18	<0.26	<0.28	<0.25
47	2.1	<0.4	2.3	0.4	0.57	<0.6	<0.5
49	<0.1	<0.2	<0.1	<0.1	<0.2	<0.2	<0.2
66	<0.1	<0.2	<0.1	<0.1	<0.2	<0.2	<0.2
71	<0.17	<0.21	<0.15	<0.18	<0.26	<0.28	<0.25
75	<0.1	<0.2	<0.1	<0.1	<0.2	<0.2	<0.2
77	<0.1	<0.2	<0.1	<0.1	<0.2	<0.2	<0.2
85	<0.1	<0.2	<0.1	<0.1	<0.2	<0.2	<0.2
99	23	<0.5	0.49	<0.4	<0.6	<0.6	<0.6
100	393	1.3	0.97	<0.1	0.31	0.54	<0.2
119	<0.1	<0.2	<0.1	<0.1	<0.2	<0.2	<0.2
138	<0.1	<0.2	<0.1	<0.1	<0.2	<0.2	<0.2
153	2.6	1.2	4.9	1.7	2.5	1.5	1.3
154	0.49	<0.2	1.5	0.5	<0.2	0.36	<0.2
166	<0.17	<0.21	<0.15	<0.18	<0.26	<0.28	<0.25
181	<0.1	<0.2	<0.1	<0.1	<0.2	<0.2	<0.2
183	1.4	<0.2	<0.1	<0.1	<0.2	<0.2	1.2
190	<0.17	<0.21	<0.15	<0.18	<0.26	<0.28	<0.25
209	<29	<37	<26	<30	<46	<48	<43

Appendix 1, Table 44 – Pesticide and PBDE concentrations found in serum samples

Sample	BEL3	BEL4	BEL5	BEL6	BEL7	BEL8	BEL9	BEL10
Pesticide	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid	ng/g lipid
α-chlordane	<1.0	<0.61	<0.74	<0.89	<0.67	<0.66	<1.1	<0.93
γ-chlordane	<1.0	<0.61	<0.74	<0.89	<0.67	<0.66	<1.1	<0.93
HCB	69	48	45	22	23	69	68	51
o,p'-DDD	<1.0	<0.61	<0.74	<0.89	<0.67	28	21	<0.93
o,p'-DDT	<1.0	<0.61	<0.74	<0.89	<0.67	<0.66	<1.1	<0.93
o,p'-DDE	<1.0	<0.61	<0.74	<0.89	<0.67	<0.66	<1.1	<0.93
p,p'-DDD	<1.0	<0.61	<0.74	<0.89	<0.67	<0.66	<1.1	<0.93
p,p'-DDE	54	248	245	41	59	446	82	41
p,p'-DDT	<1.0	1.7	1.1	<0.8	<0.6	12	4.1	1
α-HCH	6.3	4.6	8.3	<0.89	<0.67	5.6	3.9	4.5
β-HCH	30	29	27	10	15	60	31	7.6
γ-HCH	<11.	<6.6	<8.0	<9.7	<7.3	<7.2	<13	<10.
PBDE								
17	<0.26	<0.15	<0.19	<0.22	<0.17	<0.17	<0.3	<0.23
28	<0.2	<0.1	<0.1	<0.2	0.25	<0.1	<0.3	<0.2
32	<0.36	<0.21	<0.26	<0.31	<0.23	<0.23	<0.41	<0.32
35	<0.26	<0.15	<0.19	<0.22	<0.17	<0.17	<0.3	<0.23
37	<0.26	<0.15	<0.19	<0.22	<0.17	<0.17	<0.3	<0.23
47	0.67	1	3.2	0.9	<0.3	11	3.6	1.2
49	<0.2	<0.1	<0.1	<0.2	<0.1	<0.1	<0.3	<0.2
66	<0.2	<0.1	<0.1	<0.2	<0.1	<0.1	<0.3	<0.2
71	<0.26	<0.15	<0.19	<0.22	<0.17	<0.17	<0.3	<0.23
75	<0.2	<0.1	<0.1	<0.2	<0.1	<0.1	<0.3	<0.2
77	<0.2	<0.1	<0.1	14	<0.1	<0.1	<0.3	<0.2
85	<0.2	<0.1	<0.1	<0.2	<0.1	<0.1	<0.3	<0.2
99	<0.6	<0.3	<0.4	<0.5	<0.4	2.2	<0.7	<0.5
100	0.8	0.51	0.64	<0.2	<0.1	2.3	1.4	0.39
119	<0.2	<0.1	<0.1	<0.2	<0.1	<0.1	<0.3	<0.2
138	<0.2	<0.1	<0.1	<0.2	<0.1	<0.1	<0.3	<0.2
153	1.3	4.6	2.6	1.9	0.62	4.2	1.7	5.6
154	<0.2	0.67	0.34	0.56	<0.1	0.28	<0.3	<0.2
166	<0.26	<0.15	<0.19	<0.22	<0.17	<0.17	<0.3	<0.23
181	<0.2	<0.1	<0.1	<0.2	<0.1	<0.1	<0.3	<0.2
183	<0.2	<0.1	<0.1	<0.2	<0.1	0.42	<0.3	<0.2
190	<0.26	<0.15	<0.19	<0.22	<0.17	<0.17	<0.3	<0.23
209	<45	<26	<32	<38	<29	<28	<51	<40

Appendix 2 – Data summarised according to sampling location

Appendix 2, Table 1 – Concentration (in ng per g lipid) summary for GODALMING

	Minimum	Maximum	Median	25th percentile	75th percentile
				only for N>10	Only for N>10
'Total' chemical burden	110	862	402	252	597
Total PBDE burden	2.4	167	8.8	6.2	13
Total PCB burden	36	388	178	143	303
'Total' OC pesticide burden	61	540	149	90	249
Total HCH burden	2.1	54	24	16	31
Total DDT & metabolite burden	46	496	103	59	205
No. of chemicals found	28	49	35	31	42

Appendix 2, Table 2 – Concentration (in ng per g lipid) summary for LONDON

	Minimum	Maximum	Median	25th percentile	75th percentile
				Only for N>10	Only for N>10
'Total' chemical burden	126	1330	369	210	452
Total PBDE burden	2.2	13	4.7	3.9	5.8
Total PCB burden	34	494	160	123	270
'Total' OC pesticide burden	52	903	103	77	245
Total HCH burden	0.0	43	22	7.3	30
Total DDT & metabolite burden	42	860	93	66	197
No. of chemicals found	9	36	26	24	31

Appendix 2, Table 3 – Concentration (in ng per g lipid) summary for HUNTINGDON

	Minimum	Maximum	Median	25th percentile	75th percentile
				Only for N>10	Only for N>10
'Total' chemical burden	144	842	311	207	440
Total PBDE burden	1.9	9.3	4.3	3.6	5.9
Total PCB burden	79	277	190	119	243
'Total' OC pesticide burden	60	583	110	78	223
Total HCH burden	4.7	27	7.5	5.9	17
Total DDT & metabolite burden	54	578	96	71	192
No. of chemicals found	18	28	24	22	27

Appendix 2, Table 4 – Concentration (in ng per g lipid) summary for EXETER

	Minimum	Maximum	Median	25th percentile	75th percentile
				Only for N>10	Only for N>10
'Total' chemical burden	59	820	359	226	500
Total PBDE burden	2.1	259	9	4	10
Total PCB burden	32	316	138	116	266
'Total' OC pesticide burden	24	513	134	82	180
Total HCH burden	1.9	118	8	5	17
Total DDT & metabolite burden	22	384	112	77	157
No. of chemicals found	12	37	25	23	27

Appendix 2, Table 5 – Concentration (in ng per g lipid) summary for CARDIFF

	Minimum	Maximum	Median	25th percentile	75th percentile
				Only for N>10	Only for N>10
'Total' chemical burden	128	3105	388	287	494
Total PBDE burden	2	21	4	3	7
Total PCB burden	58	441	214	148	288
'Total' OC pesticide burden	53	2654	161	90	201
Total HCH burden	2	44	15	11	30
Total DDT & metabolite burden	47	2579	132	79	161
No. of chemicals found	19	33	26	22	27

Appendix 2, Table 6 – Concentration (in ng per g lipid) summary for MANCHESTER

	Minimum	Maximum	Median	25th percentile	75th percentile
				Only for N>10	Only for N>10
'Total' chemical burden	46	939	240	195	338
Total PBDE burden	2	174	5.0	3.8	6.7
Total PCB burden	14	392	121	88	147
'Total' OC pesticide burden	28	541	120	92	166
Total HCH burden	3.0	22	10	8.2	12
Total DDT & metabolite burden	25	519	97	76	138
No. of chemicals found	14	33	28	24	30

Appendix 2, Table 7 – Concentration (in ng per g lipid) summary for EDINBURGH

	Minimum	Maximum	Median	25th percentile	75th percentile
				Only for N>10	Only for N>10
'Total' chemical burden	84	871	176	133	293
Total PBDE burden	1.2	16	5.1	4.5	6.6
Total PCB burden	38	618	85	70	128
'Total' OC pesticide burden	27	247	86	65	162
Total HCH burden	2.7	27	9.0	6.8	18
Total DDT & metabolite burden	24	209	74	53	131
No. of chemicals found	19	38	25	22	29

Appendix 2, Table 8 – Concentration (in ng per g lipid) summary for NOTTINGHAM

	Minimum	Maximum	Median	25th percentile	75th percentile
				Only for N>10	Only for N>10
'Total' chemical burden	71	2024	646	243	971
Total PBDE burden	2.0	244	5.5	4.2	45
Total PCB burden	48	321	266	61	306
'Total' OC pesticide burden	7.1	1754	286	62	650
Total HCH burden	0.0	59	18	8.1	34
Total DDT & metabolite burden	1.3	1715	243	46	594
No. of chemicals found	17	34	28	23	29

Appendix 2, Table 9 – Concentration (in ng per g lipid) summary for WINCHESTER

	Minimum	Maximum	Median	25th percentile	75th percentile
				Only for N>10	Only for N>10
'Total' chemical burden	136	869	346	270	586
Total PBDE burden	1.4	155	3.6	2.7	7.5
Total PCB burden	54	397	190	143	251
'Total' OC pesticide burden	43	606	143	111	332
Total HCH burden	3.2	32	13	8.3	17
Total DDT & metabolite burden	37	570	105	93	292
No. of chemicals found	17	34	27	23	29

Appendix 2, Table 10 – Concentration (in ng per g lipid) summary for BIRMINGHAM

	Minimum	Maximum	Median	25th percentile	75th percentile
				Only for N>10	Only for N>10
'Total' chemical burden	247	1385	311	271	992
Total PBDE burden	2.8	120	15	3.6	60
Total PCB burden	123	427	189	151	319
'Total' OC pesticide burden	71	961	119	101	608
Total HCH burden	3.3	79	11	5.6	32
Total DDT & metabolite burden	66	892	102	86	506
No. of chemicals found	21	32	26	22	28

Appendix 2, Table 11 – Concentration (in ng per g lipid) summary for LEEDS

	Minimum	Maximum	Median	25th percentile	75th percentile
				Only for N>10	Only for N>10
'Total' chemical burden	112	1227	374	250	470
Total PBDE burden	3.5	137	17	6.6	39
Total PCB burden	59	371	173	104	246
'Total' OC pesticide burden	50	917	161	91	198
Total HCH burden	4.4	28	13	10	21
Total DDT & metabolite burden	46	848	123	66	169
No. of chemicals found	20	34	25	23	27

Appendix 2, Table 12 – Concentration (in ng per g lipid) summary for NEWCASTLE

	Minimum	Maximum	Median	25th percentile	75th percentile
				Only for N>10	Only for N>10
'Total' chemical burden	136	1301	591	421	982
Total PBDE burden	0.6	422	4.7	3.6	10
Total PCB burden	53	665	219	85	300
'Total' OC pesticide burden	54	1078	272	189	319
Total HCH burden	11	80	33	24	50
Total DDT & metabolite burden	18	1019	172	77	257
No. of chemicals found	22	31	27	23	28

Appendix 2, Table 13 – Concentration (in ng per g lipid) summary for BELFAST

	Minimum	Maximum	Median	25th percentile	75th percentile
				Only for N>10	Only for N>10
'Total' chemical burden	108	957	253	173	458
Total PBDE burden	0.9	20	6.7	2.9	7
Total PCB burden	21	360	85	72	171
'Total' OC pesticide burden	73	620	136	99	297
Total HCH burden	10	66	27	15	35
Total DDT & metabolite burden	25	485	59	45	211
No. of chemicals found	17	33	22	18	28

Appendix 2, Table 14 – Concentration (in ng per g lipid) summary for ENGLAND (combined data for all English locations)

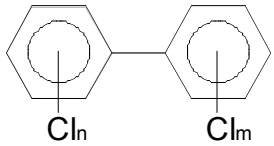
	Minimum	Maximum	Median	25th percentile	75th percentile
				Only for N>10	Only for N>10
'Total' chemical burden	46	2024	360	241	615
Total PBDE burden	0.6	422	5.8	3.8	11
Total PCB burden	14	665	169	119	273
'Total' OC pesticide burden	7.1	1754	140	83	274
Total HCH burden	0.0	118	15	7.7	29
Total DDT & metabolite burden	1.3	1715	107	66	237
No. of chemicals found	9	49	27	23	30

Appendix 3 – PCB numbering used

PCB No.	PCB Name
18	2,2’,5-Trichlorobiphenyl
22	2,3,4’-Trichlorobiphenyl
28	2,4,4’-Trichlorobiphenyl
31	2,4’,5-Trichlorobiphenyl
41/64	2,2’,3,4-Tetrachlorobiphenyl / 2,3,4’,6-Tetrachlorobiphenyl
44	2,2’,3,5’-Tetrachlorobiphenyl
49	2,2’,4,5’-Tetrachlorobiphenyl
52	2,2’,5,5’-Tetrachlorobiphenyl
54	2,2’,6,6’-Tetrachlorobiphenyl
60/56	2,3,4,4’-Tetrachlorobiphenyl / 2,3,3’,4’-Tetrachlorobiphenyl
70	2,3’,4’,5-Tetrachlorobiphenyl
74	2,4,4’,5-Tetrachlorobiphenyl
87	2,2’,3,4,5’-Pentachlorobiphenyl
90/101	2,2’,3,4’,5-Pentachlorobiphenyl / 2,2’,4,5,5’-Pentachlorobiphenyl
95	2,2’,3,5’,6-Pentachlorobiphenyl
99	2,2’,4,4’,5-Pentachlorobiphenyl
104	2,2’,4,6,6’-Pentachlorobiphenyl
105	2,3,3’,4,4’-Pentachlorobiphenyl
110	2,3,3’,4’,6-Pentachlorobiphenyl
114	2,3,4,4’,5-Pentachlorobiphenyl
118	2,3’,4,4’,5-Pentachlorobiphenyl
123	2’,3,4,4’,5-Pentachlorobiphenyl
138	2,2’,3,4,4’,5’-Hexachlorobiphenyl
141	2,2’,3,4,5,5’-Hexachlorobiphenyl
149	2,2’,3,4’,5’,6-Hexachlorobiphenyl
151	2,2’,3,5,5’,6-Hexachlorobiphenyl
153	2,2’,4,4’,5,5’-Hexachlorobiphenyl
155	2,2’,4,4’,6,6’-Hexachlorobiphenyl
156	2,3,3’,4,4’,5-Hexachlorobiphenyl
157	2,3,3’,4,4’,5’-Hexachlorobiphenyl
158	2,3,3’,4,4’,6-Hexachlorobiphenyl
167	2,3’,4,4’,5,5’-Hexachlorobiphenyl
170	2,2’,3,3’,4,4’,5-Heptachlorobiphenyl
174	2,2’,3,3’,4,5,6’-Heptachlorobiphenyl
180	2,2’,3,4,4’,5,5’-Heptachlorobiphenyl
183	2,2’,3,4,4’,5’,6-Heptachlorobiphenyl
187	2,2’,3,4’,5,5’,6-Heptachlorobiphenyl
188	2,2’,3,4’,5,6,6’-Heptachlorobiphenyl
189	2,3,3’,4,4’,5,5’-Heptachlorobiphenyl
194	2,2’,3,3’,4,4’,5,5’-Octachlorobiphenyl
199*	2,2’,3,3’,4,5,6,6’-Octachlorobiphenyl
203	2,2’,3,4,4’,5,5’,6-Octachlorobiphenyl

* = sometimes numbered 200

The PCB structure is $C_{12}H_{10-(n+m)}Cl_{(n+m)}$, where (n + m) = 1-10

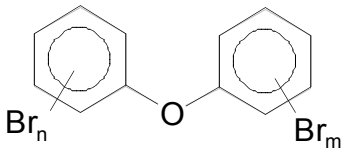


On each ring the position number starts at the carbon where the rings are attached.

Appendix 4 – PBDE numbers used

PBDE Number	PBDE Name
17	2,2',4-Tribromodiphenyl ether
28	2,3',5'-Tribromodiphenyl ether
32	2,4,6-Tribromodiphenyl ether
35	3,3',4-Tribromodiphenyl ether
37	3,4,4'-Tribromodiphenyl ether
47	2,2',4,4'-Tetrabromodiphenyl ether
49	2,2',4,5'-Tetrabromodiphenyl ether
66	2,3',4,4'-Tetrabromodiphenyl ether
71	2,3',4',5'-Tetrabromodiphenyl ether
75	2,4,4',5-Tetrabromodiphenyl ether
77	3,3',4,4'-Tetrabromodiphenyl ether
85	2,2',3,4,4'-Pentabromodiphenyl ether
99	2,2',4,4',5-Pentabromodiphenyl ether
100	2,2',4,4',6-Pentabromodiphenyl ether
119	2,3',4,4',5-Pentabromodiphenyl ether
138	2,2',3,4,4',5'-Hexabromodiphenyl ether
153	2,2',4,4',5,5'-Hexabromodiphenyl ether
154	2,2',4,4',5,6'-Hexabromodiphenyl ether
166	2,3,4,4',5,6-Hexabromodiphenyl ether
181	2,2',3,4,4',5,6-Heptabromodiphenyl ether
183	2,2',3,4,4',5',6-Heptabromodiphenyl ether
190	2,3,3',4,4',5,6-Heptabromodiphenyl ether
209	2,2',3,3',4,4',5,5',6,6'-Decabromodiphenyl ether

The PBDE structure is $C_{12}H_{10-(n+m)}OBr_{(n+m)}$, where (n + m) = 1-10



On each ring the position number starts at the carbon attached to the bridging oxygen atom.

Appendix 5 – Glossary

Adipose	Body fat tissue
Body mass Index	Body weight (kg) divided by height (m) squared: a general measure of one's 'fatness'
Chlorination / Bromination level	The number of chlorine/bromine atoms in a single molecule of a particular chemical (e.g. PCB153 has a chlorination level of 6 – it has 6 chlorine atoms per molecule)
Congener	An individual chemical out of a group of closely related chemicals (e.g. PCB153 is a congener in the PCB chemical 'family')
Correlate / correlation	A connection between two or more things, often one in which one of them causes or influences the other
Geometric mean	The mean (average) of log-transformed data
Limit of detection	The lowest quantity reliably detected in a sample
Median	The middle value in a set of values arranged in order of size
Normal distribution	A distribution following a symmetrical, bell-shaped frequency curve
Not detected	Below the limit of detection
Octanol-water partition coefficient	The ratio of the equilibrium concentrations of a chemical in octanol and water
Organohalogen / Organochlorine	Organic chemical whose molecules contain halogen/chlorine atoms
Principal component analysis	A statistical method which attempts to identify underlying variables, or factors, that explain the pattern of correlations within a set of observed variables
Serum	The straw coloured liquid separated from clotted blood after centrifugation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The womb should be the safest place on earth. But today our bodies are contaminated with over 300 man-made chemicals, to which our great-grandparents were never exposed. Many of these pollutants are found in intensively-farmed food or everyday products and some have been linked with birth defects in people and wildlife.
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WWF'S CHEMICALS AND HEALTH CAMPAIGN

Along with wildlife around the world, we are being subjected to an uncontrolled and dangerous global experiment. Exposure to hazardous man-made chemicals is putting us all at risk. Our children and wildlife are especially vulnerable. WWF's Chemicals and Health campaign is seizing a once in a lifetime opportunity to put an end to this threat, by asking people to help us ensure forthcoming European chemicals legislation brings chemicals under control.

WWF is calling for hazardous man-made chemicals to be properly regulated – replaced where safer alternatives exist, or banned where necessary.

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WWF has joined forces with two campaign partners, the National Federation of Women's Institutes and The Co-operative Bank.

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www.co-operativebank.co.uk/cwc

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Taking action for a living planet

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