

# A Present for Life

hazardous chemicals  
in umbilical cord blood

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## Executive summary

Hazardous chemicals that are used in everyday household products end up in the bodies of unborn children via the mother. This study, conducted for Greenpeace and WWF-UK by TNO, analysed blood samples donated by a number of women and confirmed that hazardous chemicals are unwittingly passed from mother to child. Previous studies had already reported the presence of hazardous chemicals in human blood and tissues and the ability of some chemicals to pass the placenta. This study further confirms that known or suspected hazardous chemicals from eight chemical groups are commonly present in umbilical cord blood. These findings underscore the urgent need to provide mechanisms to replace such substances with safer alternatives.

In the past few years, studies conducted by Greenpeace and WWF found hazardous substances not only in house dust and rainwater, but also in human blood. For the present study samples of maternal (42) and umbilical cord (27) blood, donated for scientific research by volunteers, were analysed for the presence of eight chemical groups: artificial musks, alkylphenols, bisphenol-A, brominated flame retardants, perfluorinated compounds, phthalates, organochlorine pesticides and triclosan. The blood samples were taken at the University Hospital Groningen and analysed by TNO (Peters, 2005) as part of a joint Greenpeace/WWF-UK project.

Chemicals identified in these samples are used in countless products that we use every day, including computers, toys, perfume, T-shirts and shoes. There are more than 100,000 different chemicals currently available for commercial use. The major problem is that we know virtually nothing about their potentially adverse effects because of the way production, marketing and use of chemicals is regulated in Europe. Of the minority for which data about their hazards and use are publicly available, some are known to present significant problems for the environment and/or human health.

Particularly worrying are the hormone-disrupting chemicals, which may cause most damage during the vulnerable stages of development, that is during periods of rapid cell division, such as in early life and particularly when in the womb. A small disturbance in early development can have serious consequences in later life. PCBs and dioxins have already illustrated the potential for long-term, irreversible consequences of exposure to hazardous chemicals. However, the chemical industry continues to produce various chemicals with comparable properties.

Some brominated flame retardants, for instance, are suspected of causing learning and behavioural problems in offspring exposed in the womb. These chemicals can be found in many electronic products, in some plastics and in textiles. This study was only concerned with the widely used TBBP-A; the analysis of other brominated flame retardants will be published by University Hospital Groningen as part of an academic doctoral thesis in 2005. Although TBBP-A was found in only one cord blood sample, it was the first time this relatively new brominated flame retardant was detected in cord blood. As this study analysed only a limited number of samples, this result should be cause for serious concern.

Phthalates, one of the most omnipresent groups of chemicals and used mainly as softeners in PVC, were found in a lot of the maternal and cord blood samples. DEHP, the most commonly used plasticizer, was detected in 29 maternal and 24 cord blood samples. Some phthalates can be particularly damaging to the male reproductive tract, and are toxic to reproduction.

The commonly used artificial musk HHCb was found in almost all blood samples and at higher levels than the other artificial musks. Musk ambrette, a chemical banned for use in cosmetics in the EU since 1995, was still found in 15 maternal and 12 cord blood samples.

The alkylphenol compounds most commonly used to date are nonylphenol ethoxylates. These substances were extensively used in industrial cleaning agents, but this use has been banned in the EU. It should therefore be a matter of concern that it was found in 12 of the 17 cord blood samples that were analysed for this substance. This seems to be the first time nonylphenol has been reported in cord blood.

This study also quantifies the antibacterial agent triclosan in human blood; this chemical was found in almost 50% of the samples. DDT, the notorious pesticide that is banned for agricultural use worldwide but which is still used in some places to control malaria, was still found in virtually all blood samples. Similarly, the organochlorine by-product and pesticide hexachlorobenzene – also subject to a global ban - was found in the samples. Perfluorinated compounds like PFOS and PFOA, used to make non-stick pans and water repelling coatings, were present in all but one maternal blood sample. PFOS was detected in all cord blood samples and PFOA in half of them.

The research concludes that hazardous chemicals are common contaminants in both maternal and umbilical cord blood, indicating that these chemicals can pass from the mother to the baby across the placenta. How then can we better protect our children from exposure to such potentially harmful chemicals? The only answer is for governments to put in place mechanisms that will drive industry to replace those substances with safer alternatives.

The European Union is currently revising its chemicals policy. The proposed legislation is called REACH (Registration, Evaluation and Authorisation of Chemicals) and represents a once-in-a-lifetime opportunity to protect people from man-made chemicals. REACH is designed to address the lack of information about chemicals and to take precautionary action on the most problematic substances. But according to the current draft of REACH it will still be possible to get permission to continue using a hazardous chemical even if there is a safer alternative. There is a second major loophole: for hormone-disrupting chemicals it seems that prior authorization will only be required if there is already evidence of serious effects.

Greenpeace and WWF want the production and use of hazardous chemicals to be banned wherever less hazardous alternatives are available. Furthermore, industry (particularly the chemical industry) should dramatically increase their efforts to come up with such alternatives where they do not exist. REACH should push industry to innovate by requiring this substitution. In three interviews added to this study, scientists agree on the urgency of taking precautionary measures to reduce exposure to hazardous chemicals. Finally, this report includes eight fact sheets with detailed information about the substances on which this study focused.

## Introduction

**'No one is at liberty to produce products or materials that place humans - and especially children - at risk. The Precautionary Principle (...) must be used to help industry do what is right.'**

*Conclusions of the First Annual Conference for The Consortium for Fire Safety, Health and the Environment (2003)*

The chemical industry has enjoyed spectacular growth in the last century. There are now more than 100,000 different chemicals available on the market. Chemicals are incorporated into countless consumer products, some of which undoubtedly benefit our standard of living. But they also provide a source of daily exposure to a cocktail of hazardous chemicals.

Hazardous chemicals can be found everywhere. They are released into the environment at several points in their lifecycle and travel in the air and in water to even remote areas like the Alps and the Arctic. Some of the most hazardous chemicals do not break down easily and can accumulate throughout the food chain. It is therefore very likely that some of these substances will eventually end up in the human body or in animals.

Greenpeace and WWF have published various reports over the last few decades documenting the state of scientific knowledge on the distribution and possible effects of these chemical substances. In some cases, researchers have reported alarming correlations between exposure to hazardous chemicals and certain health and developmental effects in animals. An increasingly common and ever developing theme is the widespread presence of hazardous chemicals in the human body.

Undoubtedly there are many routes of exposure which contribute to the patterns of contamination observed. Food has long been thought to be the primary route of exposure for most persistent and bioaccumulative chemicals. However, in recent years greater attention has been given to the potential exposure directly through the use of products containing hazardous ingredients and indirectly through their contamination of the indoor environment.

In recent years, Greenpeace has analysed a range of everyday consumer products for the presence of a number of (potentially) hazardous chemicals and looked for these same chemicals in house dust and rainwater. The results add weight to the suspicion that these chemicals can 'leak' from products. Follow-up investigations by Greenpeace and others have sought to research the extent to which these chemicals actually end up in our bodies, by collecting and analysing blood samples from human volunteers.

The results of recent blood research projects by Greenpeace and WWF confirm that we all have hazardous chemicals in our blood, including chemicals that are contained in normal consumer products. Of particular concern is the impact of exposure to these substances on (unborn) children. The unprotected foetus is extremely vulnerable to hazardous chemicals. Mothers can unwittingly pass on these substances to their child during pregnancy and through breast feeding (which should not deter mothers from breast feeding as the benefits of breast feeding are still widely acknowledged). Exposure to small amounts of some chemicals during early development can lead to serious health consequences in adulthood.

Greenpeace Netherlands (in cooperation with University Hospital Groningen) and WWF-UK therefore initiated this current study, which investigated the presence of similar compounds in blood serum samples taken from Dutch mothers and new-born babies. In this report we present the conclusions of that research: many hazardous chemicals are present in maternal as well as cord blood. Exposure of the mother inevitably leads to exposure of the unborn child.

## 1. Chemical routes

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

*UK Royal Commission on Environmental Pollution (2003)*

DDT, PCBs and dioxins are among the most hazardous – and most researched – man-made chemicals that have ever been brought into our environment. These, along with a number of other chlorinated pesticide chemicals, have been officially classified as POPs (persistent organic pollutants) under the global Stockholm Convention, and are largely banned from intentional production and use. This United Nations Convention was adopted in 2001 and entered into force in May 2004.

### **Lost and found: persistent organic pollutants**

However, these twelve chemicals and chemical groups, sometimes referred to as the 'dirty dozen', make up only a small percentage of the total number of POPs. Many other persistent organic chemicals are still manufactured and used as ingredients in products for industrial, agricultural and/or consumer use. Chemicals like brominated flame retardants, alkylphenols, artificial musks and phthalates have become, as a result of their extensive use, widely distributed through the environment. They have even been found in regions and animals thought to be remote from sources of chemical contamination. For example, various brominated chemicals used as flame retardant additives in plastics and textiles have been found in the bodies of polar bears, wild falcons, sperm whales and human beings. Recent research indicates that hazardous chemicals can escape from consumer products during daily use, either directly to the air or in the form of contaminated dusts (Greenpeace Netherlands 2001 and 2003, Santillo et al. 2003a).

### **Ongoing presence**

Though deliberate production and use of the 'dirty dozen' POPs have been banned or severely restricted worldwide, these chemicals, in common with many still in use, are persistent. They do not easily break down or biodegrade and therefore remain in the environment for many decades with the concentrations declining only slowly, if at all. In 2003, WWF conducted a study of chemical contaminants in the blood of 155 volunteers in the UK, a country where PCBs were banned as far back as the 1970s (WWF-UK 2003). The continuing presence of PCBs in their blood illustrates how long persistent chemicals remain in the environment and what we might expect from other persistent chemicals such as brominated flame retardants.

### **Top of the food chain**

Most persistent and bioaccumulative chemicals eventually find their way into our bodies via the food chain. They are lipophilic (fat soluble) and therefore tend to build up in the fatty tissue of animals, a process known as bioconcentration. As one animal consumes another in the food chain the levels may become even higher (biomagnification). Consequently, predatory animals at the top of food chains tend to accumulate the highest levels of some of these hazardous chemicals. Humans too are vulnerable, as our diet includes other animals. The magnitude of biomagnification for PCBs was illustrated by a study of birds' eggs from Lake Ontario in the United States. The concentration of PCBs in the eggs was 25 million times higher than that in the lake water, having accumulated through the food chain to the fish on which the birds were feeding (Colborn et al. 1996).

### **Uncontrollable hazards**

Biomagnification factors for many other persistent chemicals may be somewhat lower than in the Lake Ontario example. But bioaccumulation commonly leads to greatly elevated internal exposure concentrations at some point in the food chain. Moreover, the risks of persistent and bioaccumulative substances, once they are released into the environment, are effectively uncontrollable. Even if the production and use, and therefore the release of such a chemical were to be stopped, existing levels of contamination cannot be removed from the environment or our bodies. They can only be monitored as they gradually decline over time.

### **Dietary exposure in the Arctic**

People who are dependent on a diet rich in fat, such as the Inuit peoples of Canada and Greenland who eat seals and whales, appear to accumulate high concentrations of hazardous chemicals in their bodies. The Canadian epidemiologist, Dr Eric Dewailly, who was testing for hazardous chemicals in the breast milk of women from industrialised towns, found that the breast milk of his control group (Inuit women) contained seven times as much PCBs. Between 1994 and 2001, Dr Dewailly's research group also analysed 251 samples of blood from the umbilical cord of recently born Inuit children. They reported high doses of PCBs, DDT and other hazardous chemicals (Dallaire et al. 2003). Another large collaborative study analysed umbilical cord blood and breast milk of Inuit peoples from the Arctic regions of eight countries. The results revealed, for example, that the average concentrations of PCBs and mercury in residents of remote villages of Greenland were 20 to 50 times higher than in people inhabiting urban areas in the USA and Europe (AMAP, 2003). Thankfully, it seems that over time the levels of these pollutants have been declining, but the worry is that we are not now preventing the use of other persistent and bioaccumulating compounds.

### **Evidence of effects**

The effects of chemicals are extremely hard to comprehend. The factors that define the behaviour of hazardous chemicals are complex, and the ecosystems and living organisms in which they end up are just as complicated. This makes it difficult to provide hard evidence of hazardous effects. All too often it is not even possible to state clearly what the likely effects of long-term exposure to one particular chemical substance will be. When considering exposure to a combination of two or more chemicals, it becomes even more difficult to speculate on the nature, scale and severity of adverse effects (Axelrad et al. 2002, 2003). And yet, in real life, this is just the sort of exposure we face every day. Furthermore, researchers inevitably depend on epidemiological studies that, by their nature, can only be conducted after years of exposure, when sufficient data are available. These studies only ever yield correlative links rather than definitive evidence of cause-effect relationships.

### **Low doses**

Only relatively recently have scientists begun to unravel the effects of long-term exposure to low doses of chemicals, as opposed to the high doses which were previously examined (Dorey, 2003). They are now also researching the effects of combinations of chemical substances. What is becoming increasingly obvious through this work is how little we know about the possible environmental and health effects of the majority of chemicals on the global market. At the same time, however, some of the emerging evidence concerning chemicals used in everyday products gives substantial cause for concern. It is now clear, for example, that many chemicals can act together and have a cumulative effect.



## 2. From mother to child

**'The foetus is the weak link when it comes to exposure to hazardous substances.'**

*Dr Vyvyan Howard (2004)*

Hazardous chemicals can end up in the bodies of unborn children via the mother. Research shows that mothers pass some chemicals to their child both during pregnancy and during breastfeeding. Persistent lipophilic chemicals that have accumulated in the bodies of women over their lifetime are released as fat reserves are mobilised, for instance when they are pregnant or breast feed their children. From studies carried out on animals it appears that phthalates, for example, can pass across the placenta (Srivasta et al. 1989).

### **Chemicals in breast milk**

Phthalates have also been found in breast milk (Dostal et al. 1987, Parmar et al. 1985). Additional concerns were raised by a study of Swedish breast milk in which concentrations of the brominated flame retardant pentabromodiphenyl ether (penta-BDE) doubled every five years over a period of 25 years from 1972 (Merionyte et al. 1999). More recently, the concentrations have declined as a result of tighter controls on their use. Synthetic musk compounds (Rimkus et al. 1994) and nonylphenols also appear in breast milk (Guenther et al. 2002).

### **Benefits of breastfeeding**

It is widely acknowledged that breastfeeding confers substantial benefits on babies, in the form of vital nutrients and antibodies passed from the mother to baby, especially in the first few months of life. It also helps the bonding process between mother and child. Therefore, in spite of concerns regarding chemical contamination, the advice from scientists and health professionals is to continue breastfeeding. Rather than being a reason to stop breastfeeding, the current presence of chemical contaminants in breast milk illustrates the urgent need to tackle chemical pollution at source.

### **Body burden**

Nonylphenols and Bisphenol-A have both been found in the umbilical cords of newborn babies (Takada et al. 1999, Schönfelder et al. 2002) and in the amniotic fluid (Ikezuki et al. 2002), suggesting that these chemicals can pass across the placenta. The biomonitoring study recently conducted by WWF, involving 155 people from the UK, found that women commonly had lower amounts of PCBs in their bodies than men. The more children the woman had given birth to, the lower the PCB concentration (WWF-UK 2003). This suggests that a significant proportion of the body burden of persistent chemicals accumulated by the mothers may have been lost during pregnancy, including a proportion which would inevitably have passed to the developing child.

### **Hormone disrupting**

The hormone-disrupting effects of hazardous chemicals remain a potentially severe threat. In recent decades, the concern about chemicals with hormone disrupting properties has escalated, as several wildlife species have certainly been affected by exposure to endocrine disrupting chemicals (EDCs). The possible effects of chemical substances with so-called 'oestrogenic' or 'anti-androgenic' activity on the hormone system has been of particular concern in the last decade. These chemical substances mimic the effects of female sex hormones (oestrogens) or block the action of male sex hormones (androgens) and can seriously disturb reproduction in animals. For example, if male embryos of rats or mice are exposed to chemicals such as bisphenol-A, DDT or vinclozilin (a fungicide), such exposure can negatively affect the development of male sex organs (Skakkebaek et al. 2001).

### **Vulnerable development**

Recent studies have investigated how chemical substances such as PCBs can influence the thyroid gland and the pituitary gland. The thyroid gland plays a fundamental role in the brain development of the child, even before birth. Disrupting the action of the thyroid gland or the hormone it produces can impact on the development of the brain and the nervous system both before and after birth (Zoeller et al. 2002). The unprotected foetus is extremely vulnerable to hormone-disrupting chemicals. What may seem to be small impacts during development can lead to serious life-long consequences for the health of an adult.

### **Control system**

Our hormone system is a complex regulatory system. Almost every process in the human body is related to hormones. They regulate the growth and development of the body and influence its defence systems. Hormones play an especially vital role in the first stages of development of a new born human being or animal. A diversity of commonly used hazardous chemicals appear to have the ability to disrupt the hormone system, either by mimicking or blocking the effect of hormones or by interfering with their production or breakdown. The consequences can be drastic; exposure to hormone-disrupting chemicals has been linked to adverse effects on developing reproductive organs, disturbance of the immune system and even neurological, behavioural and learning problems.

### 3. Sick of chemicals

**'There is a worrying increase in health problems that can be partially explained by our use of chemicals, such as growing numbers of hyperactive children, dramatically dropping sperm counts; increases in testicular cancer, breast cancer and other types of cancer.'**

*Stavros Dimas, European Commissioner for the Environment (2005)*

It is impossible to establish with certainty that our exposure to hazardous chemicals has contributed substantially to the trends outlined in the statement above. Nevertheless, the indications of such a contribution are too numerous to be ignored (Dorey, 2003). Very recently, for example, the presence of elevated levels of phthalates such as DEHP in house dust has been correlated with an increased incidence of asthma, eczema and rhinitis in children (Bornehag et al. 2004).

#### **Developmental problems**

Various researchers have studied the long-term effects of exposure to PCBs and dioxins on children. Studies involving children in the Netherlands (Patandin et al. 1999, ten Tusscher 2002) and Germany (Walkowiak et al. 2001) have confirmed that deficits in cognitive function and learning ability in children can be correlated with 'high background levels' of these chemicals in certain European cities. While such deficits in European children have sometimes been described as 'subtle', there could be unknown consequences related to their future intellectual ability (Feeley et al. 2000). The significance of even a small drop in average ability across a population should not be underestimated.

#### **Deficient lung function**

Long-term studies have also shown that the greater the exposure around the time of birth, the more severe the deficiency in lung function and the greater the deficit in brain development (Ten Tusscher, 2002). Similarly, other studies involving Dutch children revealed that higher exposure for children around the time of their birth meant a lower motor and mental development (Vreugdenhil, 2002).

#### **New chemicals, similar effects**

PCBs and dioxins may not be the only man-made chemicals implicated in affecting the brain development of our children. Some researchers suspect that certain brominated flame retardants, which are used in many electrical and electronic appliances, disrupt brain development early in life. The structure of these chemicals, termed PBDEs, is remarkably similar to PCBs. PCBs have been found to cause immune suppression, altered sexual development, cancer, delayed brain development and lower IQ, and were banned in 1976. It has also been suggested that they are linked to behavioral problems like hyperactivity in humans. As with PCBs, exposure to PBDEs may be particularly harmful during a critical window of brain development during pregnancy and early childhood (WWF, June 2004).

#### **Learning deficits**

Researchers have reported that PBDEs are able to cause behavioural effects or learning deficits in animals (Eriksson et al. 2001, Viberg et al. 2001, Viberg et al. 2003). Several studies with rodents indicate that some PBDEs can exert effects on thyroid hormones (Hallgren et al. 2001) or thyroid hormone systems (Zhou et al. 2001). A significant relationship has also been reported between thyroid hormone levels in the blood of grey seal weaned pups and juveniles and the levels of some PBDEs in their blubber (Hall et al. 2003). Evidence for the thyroid effects of deca-BDE, which is still commercially important in Europe, is not clear. Nevertheless this chemical has been shown to cause effects on brain function in animals (Viberg et al. 2003).

**Effects on brain function**

Other chemicals have also been associated with effects on brain function and with thyroid disruption. Some pesticides, especially organophosphates, DDT, pyrethroids and paraquat, are particularly under the spotlight with regard to neurotoxic effects (Eskenazi et al. 1999, Dorner et al. 2002, Eriksson, 1997). Bisphenol-A, a substance frequently used in plastics, is known to have oestrogen (female sex hormone)-mimicking properties. It is reported to cause sex changes (Stoker et al. 2003), reduced nursing behaviour (Palanza et al. 2002), more masculine play behaviour in females (Dessi-Fulgheri et al. 2002) and increased aggression in male animals (Kawai et al. 2003). Furthermore, bisphenol-A has been found to abolish the sex differences in open-field behaviour (Kubo et al. 2003). Scientists also suspect that man-made chemicals may be contributing to a range of learning disabilities, including attention deficit hyperactivity disorder (ADHD), which are becoming increasingly prevalent in children. Although many factors are liable to be implicated in causing ADHD, neurotoxic chemicals may be contributing to its incidence (Rice, 2000).

## 4. Blood testing projects

**‘The pollution may perhaps be negligible where one individual is concerned, but if you assess these kinds of findings at a population level then they definitely cause a measurable difference.’**

*Professor Greet Schoeters (2004)*

Greenpeace has recently reported the presence of hazardous chemicals in house dust (Greenpeace Netherlands, 2001, Santillo et al. 2003a) and in rainwater (Greenpeace Netherlands, 2003). The logical next question is: ‘to what extent are these same hazardous chemicals also ending up in our bodies’? Greenpeace and WWF have conducted a number of biomonitoring studies, focusing in particular on the UK and the Netherlands, in order to provide further scientific data to answer this question.

### **European check up**

In 2003 WWF-UK presented the results of analyses of blood samples from 155 volunteers for three groups of chemicals: organochlorine pesticides (including DDT), PCBs and PBDEs (WWF-UK, 2003). One year later, the results of blood sample analyses from 47 volunteers in 17 European countries were published (WWF, April 2004). These samples were analysed for the same groups of chemicals, as well as for two additional brominated flame retardants (HBCD and TBBP-A) and for a number of phthalate esters and perfluorinated chemicals. Both studies found that the blood of every person tested contained a cocktail of hazardous chemicals. The degree of contamination varied widely between volunteers from the different European countries.

### **Chemical footprints in blood**

A study conducted for Greenpeace Netherlands in 2004 found comparable results for a number of chemicals, notably brominated flame retardants, phthalates, artificial musks, organotin compounds, alkylphenols and alkylphenol ethoxylates and Bisphenol-A (Greenpeace Netherlands, November 2004, Meijer et al. 2004). The study, carried out by the University Hospital Groningen in cooperation with TNO (Peters, 2004), determined the presence of these chemicals in blood samples from 91 volunteers in the Netherlands. Professor Sauer, who led the project, concluded: ‘There are chemicals present in the blood of all 91 participants. It does not matter where they live, how old they are or what job they have. We also found that the amounts vary from very low to very high. Some people are obviously more exposed than others.’

### **Family biomonitoring**

In October 2004 WWF-UK published a blood testing report containing analyses of seven chemical groups in the blood of 33 volunteers from seven British families (WWF-UK 2004). The volunteers in each family spanned three generations, with ages ranging from 9 to 88 years. All three generations tested were found to be contaminated by a cocktail of hazardous man-made chemicals. More chemicals and higher levels of certain chemicals were found in some children than in their parents or grandparents.

### **Cord blood**

This current blood testing project, commissioned jointly by Greenpeace Netherlands and WWF-UK, investigated the presence of hazardous chemicals in maternal and cord blood samples. The samples, 42 maternal blood serum and 27 cord blood serum samples, were taken at the University Hospital Groningen. TNO-MEP analysed the samples for the following chemicals: brominated flame retardant TBBP-A, phthalates, artificial musks, bisphenol-A, alkylphenols, organochlorine pesticides (DDT), triclosan and perfluorinated compounds (Peters, 2005). The results clearly show the presence of these chemicals in the blood serum samples from both mother and child. Levels of certain phthalates, artificial musks,

organochlorine pesticides, triclosan and the perfluorinated chemicals are of particular interest and concern. Furthermore, nonylphenol and TBBP-A were detected in cord blood serum, apparently for the first time.

## 5. Chemicals in maternal and cord blood: the test results

### **Alkylphenols**

Commonly used alkylphenol compounds include nonylphenols (NPs) and octylphenols (OPs) and their ethoxylates, particularly nonylphenol ethoxylates (NPEs). NPEs were extensively used as additives in plastics and as surface-active ingredients in industrial detergents and emulsifiers. They were used in textile and carpet cleaning and as emulsifiers in solvents and are still used in some agricultural pesticides.

Once released into the environment, APEs can degrade back to APs, which are to some extent persistent, bioaccumulative and toxic to aquatic life. Both APEs and APs are widely distributed in fresh and marine waters. NPs were found in a variety of foods (Guenther et al. 2002), in rainwater (Peters, 2003) and in house dust (Santillo et al. 2003). Alkylphenols can mimic natural oestrogen hormones and have been shown to alter the sexual development in some organisms, for example in fish (Jobling et al. 1996). Research in mice has shown that NPs have an effect on the male sex organs and the quality of sperm (Kyselova et al. 2003).

In the earlier Greenpeace study NPs were found in 16 of the 91 samples and OPs in only two samples (Greenpeace Netherlands, November 2004). In this study no OPs were found, which is not surprising since they are rarely found in biological or environmental samples. Some analytical problems were encountered when testing for the presence of NPs in maternal blood samples. The two maternal blood samples in which NPs could be identified may well therefore be an underestimate of the number of samples containing this compound at levels above detection limits.

There were no similar problems with the analysis of the cord blood samples. In that case, NPs were found in 12 of the 17 samples in concentrations ranging from 0.5 to 7.5 ng/g of serum. That is within the range of concentrations reported in the previous Greenpeace study. Furthermore, this seems to be the first time that NPs have been reported in cord blood.

### **Artificial musks**

Artificial musks are used to replace natural aromas and are added to many products like washing agents, soap and cosmetics (OSPAR, 2004). The best known are nitromusks like musk xylene (MX) and musk ketone (MK), though these are increasingly being replaced by polycyclic musks like tonalide (AHTN) and galaxolide (HHCB). Due to their persistence and extensive use artificial musks have become widely distributed throughout the environment.

There is increasing evidence that some musks can interfere with hormone communication systems in fish (Schreurs et al. 2004), amphibians (Dietrich et al. 2004) and mammals (Bitsch et al. 2002, Schreurs et al. 2002). MX can cause cancer in mice if they are exposed to high concentrations; this led the scientific committee for cosmetic products in the EU to tighten the EU standards for MX in cosmetics ((EU Scientific Committee, 1999).

In a blood study conducted by Greenpeace Netherlands, MK and MX were found in 9 and 6 of the 91 samples respectively (Peters, 2004). However, HHCB (found in all samples) and AHTN (found in 88 of the 91 samples) were found most frequently and at the highest levels. HHCB was also the most common and abundant artificial musk compound found in the current study (in 38 maternal and 26 cord blood samples). The concentrations are about half of those found in the earlier Greenpeace blood study.

The concentrations of HHCB and AHTN found in maternal and cord blood samples are more or less comparable. If used to estimate musk concentrations on a lipid basis (assuming

0.65% lipid content), the serum musk concentrations also compare well with lipid-normalised levels previously reported for human milk and adipose tissue (Rimkus et al. 1996, Zehringer et al. 2001). Musk ambrette was found in 15 samples of maternal blood and 12 cord blood samples. As this chemical has been banned in EU cosmetics since 1995, these findings suggest long-term persistence of the substance in our environment.

### **Bisphenol-A**

Many tin can linings, clear plastic re-usable water containers, baby feeding bottles and white dental fillings are made from polymers that can release bisphenol-A (BPA) or related compounds during use. BPA is a widely used intermediate in the production of epoxy resins, polycarbonate plastics and flame retardants.

Bisphenol-A is the building block or monomer used to form polycarbonate, a plastic which is used in situations where it can come into contact with food. Non-polymerised BPA may be released from the polycarbonate. BPA was found in canned food (Goodson et al. 2002), due to migration of material on the inside of the can to the food.

BPA was found in about 40% of the blood samples in the previous Greenpeace study (Greenpeace Netherlands, November 2004). A study of BPA in the blood of pregnant women and in placental tissue and umbilical cord blood has shown that exposure levels to BPA were similar to those suggested to be toxic to reproductive organs of male and female offspring in animal studies (Schönfelder et al. 2002). In another study, the chemical was also reported in maternal serum at concentrations of 0.21 to 0.79 ng/g and in cord blood serum at 0.45 to 0.76 ng/g (Kuroda et al. 2003).

The range of concentrations found in this study is similar: 0.5 to 1.7 ng/g serum. BPA was found in 6 of the 39 maternal blood samples analysed for this study and in only one sample of cord blood, at a concentration of 1.3 ng/g serum. This differs from the previous study by Kuroda et al. in which BPA was detected in all 9 samples of cord blood analysed.

### **Brominated flame retardants**

Brominated flame retardants prevent products from easily catching fire. They can be found in a range of industrial and electrical appliances, vehicles, insulation boards, textiles, carpets and wire coatings. Researchers have found traces of these chemicals in cormorants, whales, mountain trout and breast milk (Greenpeace Netherlands, 2004). Preventing deaths from fire is essential, but alternative (less hazardous) chemicals, methods and approaches are available (Greenpeace, 2005, Santillo et al. 2003b).

Brominated flame retardants are persistent chemicals, some of which can disrupt hormones. There are well-founded suspicions that some of them can disrupt development in early life and thereby cause effects on brain function in children. Two of the three PBDE flame retardants were banned in the EU in 2004, but other brominated flame retardants, including deca-PBDE (Viberg et al. 2003) and HBCD (Eriksson et al. 2002), have also been found to affect learning and memory function in animals.

In this study the analysis of brominated flame retardants was confined to TBBP-A because other brominated flame retardants were already investigated by the University Hospital Groningen and their results will be published later in 2005 as part of an academic doctoral thesis. TBBP-A is mainly used as a flame retardant in epoxy polymers such as printed circuit boards in electronic equipment like computers and television sets. TBBP-A *in vitro* studies indicate toxic effects on the immune system and thyroid (Darnerud et al. 2001, 2003). The chemical displays oestrogenic and thyroid disrupting properties in some studies (Meerts et al. 2001, Kitamura et al. 2005a, Kitamura et al. 2005b).



A recent WWF study found this substance in about half of the 47 blood samples (WWF, April 2004). In this study TBBP-A was found in 9 of the 42 maternal blood samples, in concentrations ranging from 0.06 to 0.19 ng/g serum. The brominated flame retardant was found in only one of the cord blood samples analysed, in a concentration of 0.05 ng/g. As far as we know this is the first time TBBP-A has been detected in cord blood serum.

### **Organochlorine pesticides**

Well-known compounds such as DDT and its metabolites belong to the group of organochlorine pesticides. These chemicals were widely used in the past all over the world. Although their manufacture and application are now largely prohibited or restricted in industrialized western countries, they can still be found in the environment, in wildlife and in humans due to their persistence.

As DDT bioaccumulates in animal fat, most humans are exposed to this substance primarily through their food. Residues have been found in human blood, serum and breast milk. Reproductive disorders are well documented in animal studies, and exposure is also linked to human developmental disorders. The main DDT component, p,p'-DDT, is classified as possibly carcinogenic to humans (DHHS, 1998).

As in other studies, HCB, p,p'-DDE and p,p'-DDT were found in the majority of samples (75% or more), in maternal as well as cord blood. Although this frequency is comparable to other studies, the concentrations of p,p'-DDE found in maternal blood in the current study (0.33-1.9 ng/g serum) are somewhat lower than those reported in previous studies (Covaci et al. 2002, WWF, April 2004). However, the concentrations of p,p'-DDT (0.09-1.5 ng/g serum) were similar to or slightly higher than those in the WWF study (WWF, April 2004).

In cord blood, concentrations of p,p'-DDE (0.15-0.83 ng/g serum) were within the range of those reported for Belgian samples (Covaci et al. 2002). Concentrations were also comparable with that study for HCB in both maternal and cord blood. Compared to previous studies a high number of maternal blood samples contained o,p'-DDD and p,p'-DDD, while these metabolites were found in only a few of the cord blood samples. The absence of DDDs in cord blood may be a detection limit problem in view of the fact that the HCB, p,p'-DDE and p,p'-DDT concentrations in cord blood were about half those in the maternal blood and the detection limits in this study were relatively high.

### **Perfluorinated compounds**

Two of the main types of perfluorinated compounds (PFCs) are PFOS and PFOA. PFCs are heat stable and they repel water as well as oil. Because of these properties PFCs are used in myriad applications, such as non-stick pans and stain/water repelling coatings for clothing, furniture and paper. Typical brand names are Teflon, Gortex, Stainmaster and - until recently - Scotchgard (EPA, 1999).

It has been known for many years that PFCs accumulate in the environment and they have been detected far from manufacturing plants in birds, marine plants and mammals. PFOS has been reported most frequently and was also found in human serum (Kärman et al. 2004, Kannan et al. 2004). Both PFOS and PFOA have previously been found in cord blood (Tittlemier et al. 2004, Inoue et al. 2004). Adverse effects have been reported in mammals and aquatic organisms (Hekster et al. 2003, Berthiaume et al. 2002, Hu et al. 2002) following exposure to certain PFCs. PFOS and PFOA cause a wide range of toxic effects on the liver of exposed laboratory rats (Berthiaume et al. 2002). Endocrine disruption and developmental effects were caused by PFOS after administration of relatively high doses to rats (Lau et al. 2004).

In blood, PFOS and PFOA are assumed to bind to plasma proteins, and thus analysis of whole blood samples is considered most representative (Jones et al. 2003, Han et al. 2003). In the current study the chemicals were determined in serum samples. The results should therefore be interpreted only as a qualitative indication of the presence of these compounds in the blood samples.

Both PFOS and PFOA were found in virtually every maternal blood serum sample. Estimated serum concentrations ranged from 0.2 to 4.2 ng/g serum for PFOA and from 0.1 to 1.3 ng/g serum for PFOS. The PFOA results for cord blood serum were comparable with those for maternal blood. For PFOS both frequency and concentrations in cord blood were lower than those in maternal blood.

### **Phthalates**

Most of the phthalates that are produced are used as plasticizers to increase the flexibility of countless PVC products like toys, vinyl flooring and electricity cables. Phthalates are also used as solvents or fixing agents in perfume, body lotion and other cosmetics. DEHP is the most commonly used plasticizer but is nowadays gradually being replaced by other phthalates like DINP. The phthalate DEP is used in a wide range of personal care products and cosmetics. It rapidly penetrates the skin and becomes widely distributed around the body following each exposure (WHO, 2003). Phthalates have become one of the most ubiquitous chemicals in the global environment. Both DEHP and DINP were measured in significant concentrations in rainwater (Peters, 2003).

Some phthalates are reproductive toxicants and particularly affect the testes (Allsop et al. 1997, Swan et al. 2005). Researchers have also shown a correlation between premature breast development in girls younger than eight years old and the concentrations of the phthalate DEHP in their blood (Colon et al. 2000). Other research suggests that exposure to some phthalates affects the sexual development of baby boys at exposure rates currently seen in the US (Swan et al. 2005).

Latini et al. found DEHP and/or MEHP (the metabolite of DEHP) in 88% of the cord blood samples they analysed (Latini et al. 2003). In the current study, DEHP was again the most commonly found phthalate, in 29 maternal and in 24 cord blood samples. The maximum DEHP concentrations found in this study were 5559 ng/g serum for maternal blood and 4004 ng/g serum for cord blood. As in the earlier Greenpeace Netherlands study (Greenpeace Netherlands, November 2004) DINP was found in only a small number of samples.

Monitoring the metabolites of phthalates is probably a more accurate way of determining exposure, as it excludes the possibility of contamination and shows the amount that has actually passed through the body. In the study being reported on here, only the parent diesters were analysed, but nevertheless this provides a useful snapshot indication of possible exposure.

### **Triclosan**

Triclosan is a commonly used antibacterial and antimicrobial agent (Glaser, 2004). Triclosan has been incorporated into many common consumer products like toothpaste, deodorants, cosmetics, textiles, toys and antibacterial soaps and detergents. Due to the increasing use of these products over the last thirty years triclosan is regularly being found in the environment.

Traces of triclosan have been found in water, sediment and fish (Okumura, 1996). Studies have indicated that triclosan is environmentally persistent and acutely toxic to biota. Its partial breakdown product methyl-triclosan is even more persistent and exhibits a potential for

bioconcentration (Böhmer et al. 2004, Balmer et al. 2004). EC Directive 67/548 classifies triclosan as 'very toxic to aquatic organisms'.

Studies showed that triclosan affects liver enzymes in rats and that this could contribute to its toxicity (Hanioka et al. 1996, 1997). One study has demonstrated multidrug resistance (MDR) conveyed by triclosan to *Pseudomonas aeruginosa* (Chuanchuen et al. 2004), which is a cause of death in many hospital-acquired infections due to its intrinsic resistance to many antibiotics.

Since triclosan has a potential for bioconcentration it's likely to end up in human beings. In the current study triclosan was found in approximately half of both the maternal and cord blood samples. For maternal blood the concentrations ranged from 0.1 to 1.3 ng/g serum, while those for cord blood ranged from 0.5 to 5.0 ng/g serum. The frequency of occurrence in maternal and cord blood is comparable, but the levels in cord blood seem to be higher than those in maternal blood.

## Conclusions

We all have hazardous chemicals in our blood. Studies conducted by Greenpeace and WWF clearly indicate that our bodies are contaminated with man-made chemicals like organochlorine pesticides, PCBs, phthalates, brominated flame retardants and artificial musks. These are chemicals that are contained in many consumer products and are therefore part of our daily lives. But these substances can 'leak' from the products and sooner or later end up in the environment and in our bodies.

The results of this Greenpeace/WWF-UK study show that not only adults but even unborn children are exposed to hazardous chemicals. Mothers unwittingly pass on these hazardous substances to their children. The chemicals are released from mothers' fat tissue during breastfeeding and can pass the placenta.

How likely is it that the chemicals found at the concentrations reported are causing adverse effects on the growth and development of the unborn child? We cannot be sure – in fact, it is unlikely that we will ever be sure. Additional research is certainly necessary. However, it is already possible to conclude that exposure of the developing foetus to a continuous low dose of a complex mixture of persistent, bioaccumulative and bioactive chemicals is a serious cause for concern. All possible steps should be taken on a precautionary basis to avoid such exposure in the womb. This can only be done by controlling the exposure of the mother to these chemicals – and that means eliminating particularly hazardous substances from the everyday products we use and, ultimately, from the environment in which we live.

The European Commission now regards the occurrence of developmental and learning disabilities as a 'significant public health problem'. Yet for most chemicals on the market today there is a lack of safety information, particularly about their ability to cause developmental toxicity, in other words where toxic substances affect the developing offspring at doses where they would not produce effects in mothers (WWF, June 2004).

It surely can't be wise to burden ourselves and our children with such a chemical legacy. Furthermore, there are alternative substances and technologies for many hazardous chemicals. Large companies such as furniture retailer IKEA and clothing chain Hennes & Mauritz have been using these alternatives for years. Electronics giants like Samsung, Nokia and Sony also decided to phase out hazardous substances. Yet many manufacturers still choose to use these chemicals because it's easier, out of ignorance, or simply because they believe it is cheaper.

Many hazardous chemicals are used needlessly, simply because there is no legislative or economic reason for substitution. But Greenpeace and WWF feel that companies should be obliged to replace hazardous chemicals with less hazardous, and preferably non-hazardous, alternatives where such alternatives are available.

While companies should voluntarily change to safer alternatives, few companies are jumping at the prospect. For this reason, governments must make it mandatory for manufacturers to use alternatives, and to develop them if no safer alternative is currently available.

European chemicals regulations are currently being completely overhauled and new legislation will be passing through the European Parliament in 2005. The proposed regulation known as REACH – the Registration, Evaluation and Authorisation of Chemicals - provides a once-in-a-generation opportunity to effectively protect people and the environment from the effects of hazardous substances. However, the proposals aren't tough enough as they stand, as the authorisation process will fail to ensure that chemicals of very high concern are phased out even when safer alternatives are available.

Although the framework (REACH) and mechanism (Authorisation) are in place, as it stands the draft legislation persists with 'adequate control' as the regulatory paradigm. Human exposure to what are considered by some to be 'acceptable levels' of chemicals of very high concern - such as chemicals that cause endocrine disruption or which build up in our bodies - seems set to continue.

Children and wildlife have a right not to be contaminated. And parents have a right to expect that products that are used in the home are as safe as possible. Greenpeace and WWF do not accept that the continuing exposure to a cocktail of hazardous chemicals can ever be considered 'safe'. Not for adults and most certainly not for the unborn child and developing infants.

**What Greenpeace and WWF want:**

- An obligation to phase out the production and use of chemicals that accumulate in wildlife, humans or the environment, and those that disrupt hormones.
- An obligation to substitute hazardous chemicals with safer alternatives.
- Complete disclosure of substances used in manufacturing processes and the composition of products, including the effects and properties of chemicals.
- Make industry accountable for the impacts of their products.
- Make importers meet the same standards as manufacturers in the EU.

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## Interviews

PROFESSOR PIETER SAUER  
**CHEMICALS TRACED IN BLOOD**

*'Our children are being exposed to polluting chemicals, though we have hardly any information on the long-term effects.'*

*P.J.J. Sauer M.D., Professor of Pediatrics, Beatrix Children's Hospital, University Hospital Groningen, has been researching the effects of polluting chemicals on children for more than 15 years. Prof. Sauer led the research project initiated by Greenpeace Netherlands in 2004 in which blood samples from 91 volunteers were analysed for 6 chemical groups. His research group collected the maternal and umbilical cord blood samples that were analysed by TNO.*

### **What strikes you most, looking at the test results?**

What is remarkable is that even substances that are less persistent can pass the placenta and end up in umbilical cord blood. Obviously the continuous daily intake of these substances is so high that they are transferred to the foetus, even when they are less persistent. Apparently, every chemical that is introduced into the environment on a large scale can enter the unborn child.

### **But it seems that the foetus is less polluted than the mother.**

That is not really true. Levels of water-soluble compounds present in the mother and the foetus are almost equal. It is different for lipid-soluble chemicals. A foetus has relatively little body fat compared to adults. So when we compare levels of lipid-soluble chemicals, the foetal levels per (kilo)gram of fat are similar to those of the mother.

### **Are these results comparable to the blood research project in 2004?**

'Certainly. In 2004 we also found polluting chemicals in the blood of all 91 participants. It didn't matter where they lived, how old they were or what job they had. The amounts found varied from very low to very high, and that's what I find particularly worrying. Evidently some people are more far more exposed than others.

### **What are the effects on children's health?**

As a pediatrician myself, I am particularly worried about the long-term effects. We all know that unborn or new-born children are most vulnerable to exposure to polluting chemicals. But we don't know what the long-term effects of these substances are, simply because there is hardly any information available. Yet our children are being exposed to these chemicals. What happens to children who are exposed at a very young age when they grow older? What are the possible effects on future generations?

### **The chemical industry tells us not to worry...**

I think you shouldn't be too quick in saying that there isn't anything wrong here. If a chemical can be traced back to the blood and you see hazardous effects in animals, where is the proof that such a chemical is not dangerous? You can only know that if you have examined a chemical very thoroughly and that includes examining the long-term effects. I would therefore turn the question around and ask: what proof is there that it doesn't cause harm?

### **If you were in charge what would you do?**

I think it is important that legislation is introduced which makes it mandatory for companies to research a chemical before they use it. I wouldn't ban existing products but would demand that the manufacturers quickly come up with a sound toxicological report about their long-

term effects. If they fail to do so I would withdraw the product from the market. Without a decent toxicological report a chemical should not be allowed onto the market.

DR GAVIN TEN TUSSCHER  
**HEALTH EFFECTS ON CHILDREN**

*'I think the precautionary principle is an inherent part of my profession as doctor.'*

*Dr Gavin ten Tusscher is specialising as paediatrician. He is also a member of a Technical Working Group advising the European Commission on biomonitoring of children within the framework of the new European chemicals policy. He obtained his Ph.D. degree from the University of Amsterdam in 2002 with a thesis on the long-term effects of dioxin exposure on children.*

**Were you surprised that the Greenpeace/WWF-UK study found man-made chemicals in cord blood?**

No, it is no surprise that so many chemicals have been found, even in the placenta. But it still remains shocking. We are exposing our children to chemicals that should not be present in the human body. What is even more unsettling is the fact that we know so very little about the health effects on individuals of exposure to these chemicals. Dioxins have been studied for decades and we are only now beginning to understand their effects on the human body.

**Your research group has studied the effects of dioxin exposure on children.**

We have followed a group of around 60 normal, healthy children that were born in the early 1990s in the Netherlands. We know what the dioxin exposure of the children was in the womb and what their exposure was after birth through breast milk. They were examined at birth, at the age of two-and-a-half and between seven and twelve years of age; we have ALWAYS found negative effects. Currently we are doing follow-up research with this group of children who are now 14 to 19 years of age.

**What kind of health effects?**

Firstly, we observed reduced lung function. The higher the exposure in the womb and after birth, the lower the lung function in later childhood. This is the most alarming effect, I think, because the lung function normally begins to decline from the age of about 25. If a child reaches adulthood with an already impaired lung function, it may lead to major pulmonary problems in later life. We also discovered effects on the immune system and the children displayed an inhibited production of blood platelets.

**You also found indications of deficits in brain development.**

Yes, we used ultra-modern and very sensitive equipment in our research (MEG) to measure the speed with which the brain reacts to certain signals. For the children in our group who were exposed to a relatively high dioxin level before and after birth, the average deficit in brain development appeared to be approximately three years. A higher exposure also seems to result in an increase in behavioural problems and aggressiveness.

**How worried do you think we should be?**

I would not advise people to worry, but I would recommend that they put pressure on policymakers to change legislation and penalise offenders. We need to demand change in order to protect our children and our children's children. If you look at the results of our research, the negative health effects for the average individual are so slight that they are barely noticeable, but if you view them on a population level they are frightening.

**Are you a supporter of the precautionary principle?**

Yes, I feel it is an inherent part of my role as a doctor. As a doctor you not only need to treat illness, you also have to try and prevent illness.

*'The foetus is the "weak link" when it comes to exposure to hazardous substances'*

*Dr. Vyvyan Howard is Head of Research in the Developmental Toxicology Research Group at the University of Liverpool, UK. He is a well-known and frequently quoted toxicopathologist, whose work includes research into the effects of hazardous substances on the foetus.*

### **The 'weak link'. Why?**

The foetus and infant are the most vulnerable members of society when it comes to exposure to hazardous substances. We're all walking around with a complex mixture of chemicals in our body. Yet these chemicals appear to have the maximum impact on the foetus in the womb. In the past, we thought that the uterus protected the unborn child against all external threats. But it turns out that most of these substances have no difficulty in crossing the placental barrier.

### **What makes the foetus so vulnerable?**

At the most critical stage of life, which is development, changes occur at exposure levels thousands of times lower than the safety limits that were set a few years ago. New studies show that many bulk chemicals that we thought were safe are actually biologically active and can disrupt human systems, including endocrine systems. They don't work by having an acute toxicity effect, they work by hijacking development. In the uterus, these chemicals can disrupt important cell signalling functions in the developing body. And that can have important implications.

### **Like what?**

For instance, I think there's lots of evidence that pollutants could be a major factor in the rising incidence of cancer in the developed countries. Just after World War II there was a one in four chance of getting cancer in the western world and now it's less than one in three. For men in the USA it's very nearly one in two. We're also seeing increases in the incidence of certain types of cancer among children and young people, for instance cancer of the testis in the male.

### **How can we determine which chemicals are risky?**

Chemicals are enormously complex. In theory, a single pesticide, toxaphene, can exist in 62,000 different forms. We simply don't have the tools to analyse all of them. There are so many chemical compounds that we just don't know which chemical does what in the complete soup. And they are ubiquitous, there's no escaping them. I think, with the level of complexity of exposure, that we have little else to resort to other than the precautionary principle. These pollutants should not be in the foetus.

### **Is that what REACH, the new European chemicals policy initiative, can achieve?**

REACH is at least an attempt to reduce exposure. A big problem is the serious lack of information. With pesticides we have that information and we can rank them by hazard. For bulk chemicals in general, we have little or no information. Either the research hasn't been done or the producers say it's confidential. Under REACH the information will become public and then we will be able to make comparative hazard assessments, so people will be able to make choices.



**FACT SHEETS**

Brominated flame retardants, phthalates, alkylphenols, bisphenol-A, artificial musks, triclosan, perfluorinated compounds and organochlorine pesticides are known or suspected hazardous substances. Once they enter the environment some of these chemicals persist for lengthy periods and can build up in living tissue and the food chain. Most of these chemicals are suspected of interfering with chemical processes in animals and humans.

Although the conclusive scientific evidence for specific effects is sometimes difficult to obtain, there is overwhelming evidence for most of these chemicals that they can mimic or block the actions of hormones. Some may weaken the immune system, cause learning difficulties and reproductive disorders. Of most concern is their potential impact on the development of the unborn child.

## ALKYLPHENOLS

**Alkyphenols (APs) are non-halogenated chemicals, manufactured almost exclusively to produce alkylphenol ethoxylates (APEs), a group of non-ionic surfactants.**

### PRODUCTION AND USE

Commonly used alkylphenol compounds include nonylphenols (NPs) and octylphenols (OPs), and their ethoxylates, particularly nonylphenol ethoxylates (NPEs). NPEs were extensively used as additives in plastics and as surface-active ingredients in industrial detergents and emulsifiers. They were used in textile and carpet cleaning and as emulsifiers in solvents and are still used in some agricultural pesticides. A small proportion is used in other products, for example as ingredients in personal care products and possibly in glues and sealants, though information is extremely scarce (1).

In Europe it is thought that for most of their former uses APEs have now been replaced by alcohol ethoxylates, which appear to have a much more favourable environmental profile. Other NP derivatives have been used as antioxidants in some plastics (2), although the scale of such uses has not been reported.

### ENVIRONMENTAL DISTRIBUTION

Both APEs and APs are widely distributed in fresh and marine waters, accumulating in particular in sediments, in which these persistent compounds accumulate. Because of releases to water, APEs and APs have also been common components of sewage sludge, including sludge used on the land.

Research into levels in wildlife remains very limited. There have been reports of significant levels in fish and aquatic birds downstream from sites where APEs are manufactured and/or used. Both NPs and OPs are known to accumulate in the tissues of fish and other organisms and to biomagnify through the food chain (1).

Recent research demonstrated the widespread presence of NPs in a variety of foods in Germany (2). Little is known about the extent and consequences of direct exposure from use in consumer products. Both NP and OP residues have been found in house dust (3). A Greenpeace study (2003) showed that NPs were ubiquitously present in rainwater, perhaps reflecting continued use of NPEs at that time. OPs were found in a limited number of rainwater samples (4).

### EFFECTS

The main hazards associated with APEs result from their partial degradation to shorter-chain ethoxylates and to the parent APs themselves, both of which are toxic to aquatic organisms. The EU risk assessment for NPs identified significant risks to the aquatic environment and to the soil through the then current uses of NPEs, but also to higher organisms resulting from the accumulation of NPs through the food chain (1). With respect to human exposure through use in consumer products, the CSTE (5) observed '*a serious lack of measured data for NP*'.

The most widely recognised hazard associated with APs is their ability to mimic natural oestrogen hormones, which has been shown to alter sexual development in some organisms, for example the feminisation of fish. This is thought to have contributed significantly to the widespread changes in sexual development and fertility among fish in UK rivers (6). Exposure of male rainbow trout (*Oncorhynchus mykiss*) to four different alkylphenolic chemicals caused synthesis of vitellogenin, a process normally dependent on endogenous oestrogens, and a concomitant inhibition of testicular growth (7). In laboratory experiments with the freshwater snail (*Marisa cornuarietis*) and marine prosobranch (*Nucella lapillus*) it induced a complex syndrome of alterations referred to as superfemales (8).

Research with mice has shown that NPs have an effect on the male sex organs, the quality of sperm and the fertility of parents and descendants (9). Studies with mice also showed that NPs can increase the levels of certain antibodies and messenger chemicals, which are themselves implicated in allergic reactions (10).

APs can cross the placenta and have been found in the umbilical cords of babies (11) and NPs are found in breast milk (2). Hazards to human health remain unclear, although recent studies describe effects on mammalian sperm function (12, 13), while DNA damage in human lymphocytes has also recently been documented (14). Preliminary studies show that NPs may disrupt the human immune system by adversely affecting groups of white blood cells.

## EXISTING CONTROLS

In 1992 parties to the Oskar Convention decided to phase out NPEs in cleansing agents (15). In 1998 the OSPAR Commission agreed on the target of cessation of discharges, emissions and losses of all hazardous substances to the marine environment by 2020. NPs/NPEs were included on the first list of chemicals for priority action towards achieving this target (16). NPs have also been included as a 'priority hazardous substance' under the EU Water Framework Directive (17). A decision on the prioritisation of OPs/OPEs under this Directive is still under consideration.

According to Directive 2003/53/EC, as of January 2005 products containing more than 0.1% NPs or NPEs may no longer be placed on the market within Europe, with some minor exceptions, principally for 'closed loop' industrial systems (18).

Very little specific information exists regarding the scale and diversity of use of NPs and their derivatives in consumer products up to that date and, consequently, regarding our exposure to them over the years. Furthermore, it is also difficult to establish the extent to which OPs and other alkylphenols and their derivatives remain in use within Europe. The new European chemicals policy – REACH – is expected to come into force in 2006. This could further restrict the production and use of environmentally harmful chemical substances such as APs.

## ALTERNATIVES

APEs can be substituted by other substances, for example alcohol ethoxylates, though direct substitution may be complex in some situations.

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## ARTIFICIAL MUSKS

**Synthetic musks are man-made compounds that are used to replace natural musk. They are added to many everyday products, including laundry detergents, air fresheners, hand creams, soaps and perfumes (1).**

### PRODUCTION AND USE

Synthetic musks encompass three chemical groups: nitro musks, polycyclic musks and macro cyclic musks. Due to toxicological concerns, nitro musk production has been in decline in Europe for a number of years. Only two nitro musks are of importance today: musk xylene (MX) and musk ketone (MK). These, along with the two polycyclic musks, galaxolide (HHCB) and tonalide (AHTN), account for 95% of the European market for synthetic musks (1).

The chemical industry produced musk compounds on a large scale particularly in the 1980s and the first half of the 1990s. The world production of synthetic musk was estimated in 1998 to be 7,000 tonnes per year, mostly the polycyclic. Of this amount 2,000 tonnes is produced in Europe (2). PFW Aroma Chemicals in the Netherlands is one of the largest producers of polycyclic musks in the world.

The worldwide production of nitro musk compounds is between 1,000 and 2,500 tonnes, of which MX and MK account for 90%. In Europe it is estimated that around 50 to 300 tonnes of MX and 110 tonnes of MK are produced annually.

### ENVIRONMENTAL DISTRIBUTION

Synthetic musks are persistent chemicals. Due to their extensive use in products such as washing agents, soap and cosmetics they have become widely distributed in the environment, especially in aquatic and marine systems (2, 3, 4) but also in the atmosphere (5) and inside buildings (6).

From the use in human household products synthetic musks are released into the atmosphere and wastewater. The compounds are released to the aquatic environment from wastewater treatment plants with effluents (7). A recent review of nitro musks and polycyclic musks in the Nordic countries found particularly high concentrations of polycyclic musk compounds in sewage sludge. Polycyclic musks were also frequently reported in blue mussels and rainwater. Nitro musk compounds were only exceptionally found in Norway, but some sewage samples from Denmark and one from Sweden also contained musk ambrette (MA) (7).

A Dutch study found synthetic musk compounds in rainwater. The nitro musk MA, which has been banned in EU cosmetics since 1995, was found at 34% of the rainwater collection points (5). This may suggest long-term environmental persistence or contribution from ongoing use. The highest levels of AHTN were found in the centre of the Netherlands, where producer PFW Aroma Chemicals is located.

Human skin is exposed directly to musk compounds from many everyday products. Synthetic musks can concentrate in living tissues; indeed, musks used in perfumes have also been found to contaminate human blood and breast milk (8, 9).

### EFFECTS

The long-term effects of musk compounds on humans are largely unknown. Whereas acute toxicity of synthetic musks to mammals seems to be relatively low, insufficient data are available to evaluate the hazards of long-term, low level exposure. This is especially true for the polycyclic musks, including AHTN and HHCB, and their metabolites. Some effects on reproductive and foetal development have been observed in rats, though so far only at levels

far higher than ambient exposure levels from consumer products and environmental contamination (10).

There is increasing evidence emerging that some nitro musks and polycyclic musks, including those commonly used in perfumes, may be capable (either as parent compounds or as metabolites) of interfering with hormone communication systems in fish (11), amphibians (12) and mammals (13, 14). For example, research carried out *in vitro* in 1999 indicated that the hormone system of frogs and fish may be more sensitive to breakdown products of MX and MK than to the parent compounds themselves (15). Recent Dutch research also showed that polycyclic musk compounds can be hormone disrupting in fish (11).

Although the oestrogenic activity exhibited by HHCB and AHTN in mammals is relatively weak, anti-oestrogenic effects have been observed for the same compounds at concentrations more than a hundred times lower (14). Statistical associations have been reported between MX and MK levels in the blood and the occurrence of certain gynaecological conditions in women (16), although no causal relationship has been established.

MX can cause cancer in mice if they are exposed to high concentrations; this led the scientific committee for cosmetic products in the EU to tighten the EU standards for MX in cosmetics (17). New research suggests that some synthetic musks may exacerbate the effects of exposure to other toxic chemicals (18). HHCB, AHTN and MX were all found to inhibit the defences of cells taken from mussel gills to multiple toxic exposure by inhibiting the activity of proteins that normally prevent uptake of xenobiotic agents. As well as illustrating a potential toxicological issue associated with musks, this work shows how standard toxicity tests do not provide a comprehensive picture of the effects a compound may have upon release into the environment.

## **BAN**

MA is not permitted in the EU in cosmetic products due to its phototoxic or neurotoxic properties. The European Commission regulated some of the uses of MX and MK in 2002 and 2003 (19).

In 1998, the OSPAR Commission agreed on the target of cessation of discharges, emissions and losses of all hazardous substances to the marine environment by 2020. MX was included on the first list of chemicals for priority action towards achieving this target (20).

## **ALTERNATIVES**

Nitro musk compounds are often substituted by polycyclic musk compounds. A recent OSPAR document concluded that MX should be replaced by substitutes with a more favourable environmental profile (1). OSPAR stated that polycyclic musks 'should not be promoted as suitable substitutes for nitromusks because, although not actually regarded as PBT substances according to the criteria of the EC technical guidance document, they have unfavourable characteristics'.

Although there is still very little information available about macrocyclic musks, an initial assessment suggests these compounds are more environmentally benign. OSPAR provisionally suggests that macro cyclic musks may be acceptable substitutes, but that would need to be confirmed on the basis of results of field studies before they can be recommended as environmentally acceptable substitutes (1).

Other alternatives for synthetic musks are based on natural products such as flowers and herbs.

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## **BISPHENOL-A**

**Bisphenol-A (BPA) is widely used in the production of epoxy resins, polycarbonate plastics and flame retardants and as an additive in other plastics.**

### **PRODUCTION AND USE**

BPA is one of the chemicals with the highest volumes of production worldwide; global BPA capacity in 2003 was 2,214,000 metric tonnes (1). Most BPA is used to make polycarbonate plastic and epoxy resins (2). Polycarbonates are used for compact disks, reusable beverage containers (e.g. water cooler bottles and baby bottles), mobile phone housings, safety glazing, motorcycle windshields, medical devices and roofing panels.

Epoxy resins are used as plastic coatings in the food-packing industry, as well as in adhesives, laminates, structural composites and protective coatings. Bisphenol-A-based resins are also used in human health applications such as filling materials or sealants in dentistry.

### **ENVIRONMENTAL DISTRIBUTION**

BPA was found in the leachate from a landfill in Germany (3), in treated wastewater effluent and in drinking water (4). BPA is metabolized in river water by bacteria, but the metabolites also show some oestrogenic activity (5). Research confirms the leaching of oestrogenic BPA monomers and/or related compounds from dental composites (6) and from lacquer-coated cans (7).

Studies suggest major differences between species in the metabolism and excretion of BPA; e.g. slowly by rats, but more rapidly by humans (8). BPA was identified in 95% of urine samples from people in the USA examined by the Centers for Disease Control (CDC) (1).

### **EFFECTS**

BPA is an EU category 3 reproductive toxicant. BPA binds to oestrogen receptors in a range of human cell lines and is oestrogenic in animals (9).

A recent review of studies of low-dose effects of BPA found that 94 of the 115 publications reviewed reported significant effects. In 31 reported animal studies, significant effects occurred at levels below the USA predicted 'safe' or reference dose of 50 µg/kg/day BPA. Effects include alterations in brain chemistry and structure, behaviour, the immune system, enzyme activity, the male reproductive system, and the female reproductive system in a variety of animals, including snails, fish, frogs and mammals (1).

A study of BPA in the blood of pregnant women and in placental tissue and umbilical cord blood has shown that exposure levels to BPA were similar to those suggested as being toxic to reproductive organs of male and female offspring in animal studies (10). A recent epidemiological study among Japanese women indicates that BPA may be related to ovarian disease in women (11).

### **EXISTING CONTROLS**

There is genuine concern about endocrine disruptors but little has been done to reduce exposure or phase out the use of chemicals like BPA. The OSPAR Commission has acknowledged the need to include endocrine disruptors on its List of Substances of Possible Concern, but this has not yet been done (12).

### **ALTERNATIVES**

In many cases it is likely that there are alternative materials for BPA-based materials and products. A recent study found that the urinary BPA levels in Japanese students in 1999



were significantly lower than in 1992. The researchers speculated that changes made to the interior coating of beverage cans in Japan may explain these findings (13).

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## **BROMINATED FLAME RETARDANTS**

**Brominated flame retardants are used to prevent combustion or retard the spread of flames in a variety of plastics, textiles and other materials.**

### **PRODUCTION AND USE**

Although more than seventy brominated compounds or groups are in use as flame retardants, three chemical groups dominate current usage: the PBDEs, HBCD and brominated bisphenols (especially TBBP-A) (1). Brominated flame retardants can be found in a range of industrial and electrical appliances, vehicles, lighting and wiring. But they are also used in textiles including carpets and furnishings, and in packaging and insulating materials such as polystyrene (1).

The worldwide industrial use of brominated flame retardants in 2001 was 204,740 tonnes, of which Asia accounted for 58%, the USA for 29% and Europe for 12%. TBBP-A accounted for 58% of the volume used, PBDEs for 33% and HBCD for 8% (2). Brominated flame retardants are produced by several companies, including Great Lakes Chemical, Albemarle, Atochem and the Dead Sea Bromine Group. The British producer Great Lakes Chemical announced in 2004 that it would cease production of penta- and octa-BDEs (3).

### **ENVIRONMENTAL DISTRIBUTION**

Brominated flame retardants are generally persistent and bioaccumulative chemicals, which are ubiquitously present in the environment. They have been found even in remote areas, e.g. in sperm whales which find their food deep in the oceans (4). Most of the available data concern PBDEs and PBBs, although use of the latter has now largely ceased (5). There are fewer data for the other brominated flame retardants in common use, although recent research suggests that HBCD contamination might also be a widespread phenomenon (6).

Brominated flame retardants were found in 30% of the samples taken of Dutch rainwater. HBCD had a peak concentration close to Terneuzen, where it is produced by Broomchemie, a subsidiary of the Dead Sea Bromine Group (7).

The primary route of exposure for humans may be through food, but other sources of exposure may also be significant, including direct contact with products in which flame retardants have been applied. PBDEs, HBCD and TBBP-A have been detected in indoor air and/or dust in the workplace (8, 9). Concentrations in the blood correlated to some extent with contact with, among other things, computers in the office environment (10). Brominated flame retardants were also found in house dust in the UK and in eight European parliament buildings (11, 12).

PBDEs have several times been reported as common contaminants in humans (13, 14, 15, 16). Compared to the levels found a couple of decades ago, the concentrations of PBDEs in human breast milk and blood have increased (17, 18).

Irrespective of the chemical form of the brominated flame retardant used, if products containing these compounds are burned extremely toxic brominated dioxins and furans can be produced (19).

### **EFFECTS**

Although their mechanisms of toxicity are gradually being elucidated, the long-term, low-dose toxicity of brominated flame retardants generally remains poorly described (12).

In a recent review of available data on HBCD, PBDE and TBBP-A scientists concluded that the existing information raises concerns, but the toxicology database is inadequate to truly understand the risks (20). The review noted that in order to better understand the risks from

exposure to brominated flame retardants studies should focus on the congeners, and potentially their metabolites and/or breakdown products, that are present in people and wildlife.

While their acute toxicity is considered to be low, chronic exposure (especially in the womb) to brominated flame retardants has been shown to interfere with brain and skeletal development in rats (21). This may in turn lead to permanent neurological effects (22). Common metabolites of the PBDEs, as well as TBBP-A, are reported to interfere with the binding of thyroid hormones (23, 24), increasing the potential for diverse effects on growth and development.

Some PBDEs and TBBP-A have been reported to be capable of binding to oestrogen receptors and eliciting some oestrogenic responses in human cell lines *in vitro* (25) and one study for TBBP-A has found an oestrogenic response *in vivo* (26). TBBPA has also been found to affect thyroid hormones in *in-vitro* assays (27). Genotoxic effects for both PBDEs and HBCD in mammalian cell lines have been reported (28).

### **EXISTING CONTROLS**

In 1998, the OSPAR Commission agreed on the target of cessation of discharges, emissions and losses of all hazardous substances to the marine environment by 2020. Brominated flame retardants were included on the first list of chemicals for priority action towards achieving this target (29). OSPAR has since reviewed opportunities for action in relation to the PBDEs and HBCD, but is awaiting the outcome of assessments within the European Union (EU) before developing specific measures (30).

In its background document on TBBP-A in 2004, OSPAR concluded that substitution with safer alternatives that pose less risk to the environment should be encouraged, as well as the development of such substitutes (31).

In 2003 the EU agreed to phase out PBBs and all PBDEs from electrical and electronic equipment by 1 July 2006 under the EU Restrictions on Hazardous Substances (ROHS) Directive (32). However, an exemption for deca-BDE is still under discussion and the directive does not cover the use of other brominated flame retardants, e.g. TBBP-A.

Since August 2004 the EU has banned products containing octa- and penta-BDE from the European market (33). While substantial data gaps remain and prevent completion of the assessment of deca-BDE, EU Member States have so far agreed to voluntary reductions of emissions from factories while further testing proceeds (34). The EU also recently imposed testing and information requirements on the importers or manufacturers of TBBP-A (35).

### **ALTERNATIVES**

Danish and Swedish government studies showed that effective alternatives are available for applications of brominated flame retardants which are less harmful for the environment (36). A number of electronics companies have already committed themselves to phasing out all brominated flame retardants and have introduced a range of products on the market that do not contain these chemicals (37).

**PBDE:** POLYBROMINATED DIPHENYL ETHER - **HBCD:** HEXABROMOCYCLODODECANE - **TBBP-A:** TETRABROMOBISPHENOL-A - **PBB:** POLYBROMINATED BIPHENYL

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## DDT

**DDT (dichloro-diphenyl-trichloroethane) is a pesticide that was used intensively worldwide in the 1950s and 1960s, both in agricultural production and for malaria control. Concerns about impacts on wildlife populations - particularly predatory birds - led to the phasing out of DDT in many countries in the 1970s.**

### PRODUCTION AND USE

The term 'DDT' refers to technical DDT, which is a mixture of several compounds and may not always have the same composition. The main component is p,p'-DDT, though it also contains a variable mix of other DDT and DDE compounds (1, 2). DDT was first widely used during the Second World War to control disease-carrying insects.

The use of DDT in agriculture has been banned globally since 2004 by the Stockholm Convention, but countries can ask for an exemption for certain uses (3). An estimated 19 countries (mainly in Africa) are currently using DDT to fight malaria, and another 6 used it until recently. Of the 91 countries that signed the Stockholm Convention, 31 requested exemptions to use DDT to control malaria (4).

Accurate production data are difficult to establish. DDT is probably still produced in India and China. Hindustan Insecticides Limited (HIL) is the government-owned company responsible for production in India (5, 6, 7). In China DDT is produced by Shenzhen Jiangshan Commerce and Industry Corporation (4). Another manufacturer, for whom the current status is not certain, is P.T. Montrose Pesticido Nusantara (Indonesia) (5, 6). Unnamed producers are also thought to be operating in China, Mexico, Russia, South Korea and the former Soviet Union (8).

### ENVIRONMENTAL DISTRIBUTION

The main ingredient, p,p'-DDT, is broken down in the environment or in the body to p,p'-DDE and smaller quantities of other chemicals. p,p'-DDE is more persistent both in the body and the environment than p,p'-DDT (9). It is responsible for most of the observed toxic effects, unless there has been recent exposure to technical DDT.

Most humans are exposed to DDT primarily through their food because the substance can bioaccumulate in animal fat. DDT and DDE residues have been documented in the food supply of many countries around the world (10). Particularly high levels of DDT have been documented among indigenous people in the Arctic who eat traditional foods (e.g. seals, narwhal whales) (4).

Residues have been found in human blood, serum and breast milk in many countries around the world. Levels of DDT found in humans have dropped significantly in those countries that have long since banned its widespread use in agriculture (11).

### EFFECTS

In wildlife, DDT primarily causes population decline through reproductive failure, though it may also kill highly exposed birds directly (12, 13, 14). Since DDT was banned in many countries in the early 1970s, many of the bird populations facing extinction at that time have recovered. However, present DDE levels in the Arctic are still causing significant egg shell thinning (15). There is also strong evidence to suggest that DDT may have played a role in the change in the sex ratio of several North American gull populations in the post-DDT era, which resulted in an overabundance of females (16).

DDT is moderately to slightly toxic to mammals (1, 7, 17). The primary target of DDT is the nervous system, and it has caused chronic effects on the nervous system, liver, kidneys and

immune systems in experimental animals (1, 18). There is also evidence that DDT causes reproductive effects in test animals, including reduced fertility (1).

Laboratory studies clearly show that DDT-related chemicals have sex hormone-disrupting properties (19). However, it is difficult to determine the precise causal mechanism of the effects seen in the wild. It is not even known for certain whether the well-known eggshell-thinning effects of DDT are caused by hormone disruption.

Exposure is linked to human developmental disorders and reproductive disorders are well documented in animal studies. Recent studies have also linked exposure to reduced lactation in nursing women. DDE levels in American women have also been linked with increased risks of premature delivery and reduced infant birth weight (20).

The IARC classified p,p'-DDT as possibly carcinogenic to humans (group 2B) and the US Department of Health and Human Services regards it as being 'reasonably anticipated to be a human carcinogen' (2).

### **EXISTING CONTROLS**

The UN Stockholm Convention, that entered into force in May 2004, agreed on a worldwide phase-out of twelve persistent organic pollutants (POPs), including DDT. However, exemptions of limited duration are allowed for DDT for disease vector control (21). DDT is also controlled under numerous other international legal instruments.

### **ALTERNATIVES**

For malaria control, DDT is sprayed on the walls inside homes in areas where mosquitoes are known to be present. Mosquitoes develop resistance to DDT (4). Alternatives to DDT for malaria vector control already exist, including - but not limited to - alternative chemical agents. These include synthetic pyrethroids, although these agents themselves may not be free from concerns regarding human and environmental toxicity. Like DDT, they will never be universally effective. Other controls are physical measures to destroy breeding grounds, physical barriers and prophylactic medicines. Countries that have moved away from the use of DDT to control malaria use a combination of drugs, bed nets treated with synthetic pyrethroids and the application of chemicals to breeding areas or houses. It is likely that a combination of alternative strategies will remain necessary in order to provide protection for human health.

WWF has documented the experience in the Kheda district in India, where non-chemical approaches were demonstrated to be cost-effective. In the Philippines the national program has relied on treated bed nets and the spraying of alternative chemicals (22).

A recent review of indoor residual house spraying concluded that DDT is still shown to be an effective malarial control chemical in southern Africa, but that new drug therapies (artemisinin-based combinations) have also been implemented to good effect in the Republic of South Africa (23).

Successful malaria control remains a major challenge in many areas where public health programs are under-funded and the cheapest drugs are no longer effective. More effective anti-malaria programs are needed, with increased funding for research and field application of alternative control methods.

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## **PERFLUORINATED COMPOUNDS**

**Perfluorinated compounds (PFCs) are thermal and chemically stable polyfluorinated organic compounds (1) that are used in a wide variety of applications.**

### **PRODUCTION AND USE**

PFCs are used in protective coatings for carpets, textiles, leather, paper and board, but also in fire-fighting foams and as polymerisation aids. Furthermore, they are used as speciality surfactants in cosmetics, electronics, etching, medical use and plastics (2).

PFCs generally have side groups attached, such as carboxylic acids or sulfonic acids. This compound group comprises two major classes of PFCs: perfluorinated carboxylates or perfluoroalkyl carboxylates (PFCAs, which include PFOA and fluorotelomer alcohols) and perfluorinated sulfonates or perfluoroalkyl sulfonates (PFOS-related) (3).

PFCs have been produced since 1956 by 3M Company. The production of PFOS-related chemicals in the USA was 3,000 tonnes in 2000 (4). Under increased public and governmental pressure, 3M decided in 2000 to cease the manufacture of PFOS-related chemicals by 2003 (5, 6). They have largely been replaced with other, less bioaccumulative, perfluorinated alkyl sulphonates.

Other companies that produce PFCs include AsahiGlass (Japan), AtoFina (France), Clariant (Germany), Daikin (Japan) and DuPont (United States) (3, 7). DuPont makes PFOA from which Teflon, a non-stick coating used for saucepans, is manufactured (8).

### **DISTRIBUTION IN THE ENVIRONMENT**

PFCs enter the environment during manufacturing and use. They have been detected in indoor and outdoor air, in rivers, lakes and groundwater, in wastewater treatment effluent, in landfills and in the marine environment (4). PFCs are persistent in the environment (9) and there is no known route of complete degradation either in the environment or by living organisms.

PFCs accumulate in the blood, liver and gall bladder of living organisms (3, 10), from where they can accumulate in the food chain (11). PFOS is a widespread contaminant in living organisms, including the tissues of aquatic and terrestrial living organisms such as humans. Other PFCs, including PFOA, are also found in living organisms, although often to a lesser extent than PFOS (1).

Various PFCs have been found in human blood serum from a number of countries, including USA, Poland, Japan, Korea, Malaysia, Belgium, Brazil, Italy, Colombia and India (12). Overall, the results of studies have suggested that levels of exposure to PFOS vary quite a lot both within and between countries. The highest mean levels were found in the USA and Poland, and the lowest in India. Other studies have also investigated the level of PFOS in human blood in the USA (5, 13), northern Canada (14), Japan (15, 16) and Sweden (17).

Recent research commissioned by the Norwegian Pollution Control Authority found PFOS in 70% of the blood samples of pregnant Norwegian and Russian women in the Arctic. Although the number of samples was limited, the results confirm that PFOS is a substance that is transported over long distances: there are no known local sources of these chemicals (18).

A Canadian study found PFOS and PFOA in cord blood plasma, which shows that the developing infant is exposed in the womb (14). PFOS in cord blood serum was also found in a Japanese study, where it was concluded that foetuses in Japan may be exposed to relatively high levels of PFOS (19).

Two human milk samples were analysed for PFCs, including PFOS, PFOA and PFOSA (13). In one sample only PFPeA, at 1.56 ng/ml, was found, and in the other sample only PFHxA, at 0.82 ng/ml (= 1 billionth gram per millilitre). The study suggested that PFCs may therefore not be as prevalent in human milk as in blood. Nevertheless, studies with a much larger sample size are needed to confirm this since the sample size was rather small.

## **EFFECTS**

PFCs are persistent in the environment and some have the potential to bioaccumulate in the blood and liver of living organisms. Adverse effects have been reported in mammals and aquatic organisms following exposure to certain PFCs. A review of aquatic toxicity studies, most of them published by industry, reported that PFOS is moderately acutely toxic and slightly chronically toxic to aquatic organisms (2).

PFOS and PFOA cause a wide range of toxic effects on the liver of exposed laboratory rats, including hepatic (liver) peroxisome proliferation (20) and induction of enzymes associated with  $\beta$ -oxidation of fatty acids and lowering of serum cholesterol.

PFOS and PFOA have been found to inhibit the communication system between cells (gap junction intercellular communication) in both mammalian cell lines *in vitro* and in rats. Disruption of the process results in abnormal cell growth and function and is associated with tumour promotion. In addition, chronic (long-lasting) disruption of gap junction intercellular communication could lead to neurological, cardiovascular, reproductive and endocrinological dysfunction (21).

Endocrine disruption and developmental effects were caused by PFOS after administration of comparatively high doses to rats (22). The developmental effects include reduction of fetal weight, cleft palate, anasarca (edema), delayed ossification of the bones, cardiac abnormalities and death in new-borns. Significant increases in liver weight in new-borns exposed to PFOS *in utero* was also reported (23). It was suggested that the developing liver is a potential target for PFOS action. In addition, pups that survived beyond the first few days had growth retardation (a reduction in body weight gain) and had reduced serum levels of thyroxine, a thyroid hormone. The results on growth retardation suggested that PFOS may interfere with cellular or functional maturation of target organs, possibly via alteration of thyroid hormones.

One study of mortality among humans who were occupationally exposed to PFOA reported that the risk of mortality from prostate cancer increased the longer the duration of the work (24). These findings were, however, based on only four deaths among exposed workers. The researchers suggested that further research was needed into the risk of prostate cancer from PFOA exposure.

## **EXISTING CONTROLS**

The US EPA is drafting a risk assessment on PFOA, which is due to be published in 2005. It also recently filed a claim against DuPont, the principle manufacturer of PFOA in the USA, seeking penalties against it for withholding the results of human blood sampling documenting levels of PFOA in individuals near a DuPont facility in West Virginia (25).

The European Commission is currently drafting proposals to restrict the marketing and use of PFOS. In June 2004, the UK government announced unilateral action to phase out PFOS and related compounds (6).

**PFOS:** perfluorooctane sulphonate - **PFOA:** perfluorooctanoic acid - **PFPeA:** perfluoropentanoic acid – **PFHxA:** perfluorohexanesulphonate – **PFOSA:** perfluorooctane sulfonamide

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## **PHTHALATES**

***Ninety percent of all phthalates are used as softeners in PVC products like toys, rain clothing, floor covering, vinyl wallpaper and electrical cables. Other applications include use in paints, glues, printing inks and cosmetics.***

### **PRODUCTION AND USE**

DEHP is the most commonly used plasticizer. Other widely used phthalates are DINP and DIDP. DBP and DEP are used as softeners, but also as solvents or fixatives in cosmetics (2). DEP is used in a wide range of personal care products as a solvent and vehicle for fragrances and other cosmetic ingredients. Another application of DEP is to render the alcohol used in cosmetics unfit to drink (1).

In Western Europe about one million tonnes of phthalates are produced each year, of which approximately 900,000 tonnes are used to plasticize PVC (polyvinyl chloride). DEHP accounts for about 30% of the volume produced (3).

### **ENVIRONMENTAL DISTRIBUTION**

As a result of their high production volume and widespread use, phthalates have become among the most ubiquitous chemicals in the global environment (2). They do not easily biodegrade and can, in some cases, accumulate in the environment and in organisms. In a TNO study commissioned by Greenpeace Netherlands in 2003, the phthalates DEHP and DINP were measured in significant concentrations in rainwater (4).

Since DEP is an ingredient of perfumes and other personal care products it appears that inhalation of DEP may be a significant route of exposure (5). Absorption through the skin is also likely to be a contributory factor. Scientists found high concentrations of phthalate breakdown products in women's urine, to which the use of nail varnish and perfume may have made a significant contribution (6).

Small children may have a higher exposure to phthalates than adults via toys, household furnishings and floor coverings. In a urine study in the USA, the highest concentrations of metabolites for a number of phthalates were found in the urine of children aged between 6 and 11 (7). Hospital patients may be exposed through the use of PVC in medical equipment. The phthalate DEHP in PVC blood and infusion bags has been shown to leach into the blood (8).

### **EFFECTS**

Long-term exposure of rats to the phthalate DINP led to increases in liver and kidney weight (9). It is possible that some phthalates can cause cancer. For example, exposure to DEHP has been linked to liver cancer in rodents (10).

The main concern about phthalates, e.g. DEHP, DBP and BzBP, is that they have an anti-androgenous effect. Exposure during pregnancy can affect the development of the testicles and sperm production (11, 12, 13). One study reported that girls with early breast development (6 months - 8 years) often had phthalates in their blood serum in much higher concentrations than their age contemporaries in the control group (14).

DEP has generally been considered to have a low overall toxicity, but newly emerging evidence raises significant concerns regarding its safety. DEP is rapidly metabolized in the human body to its monoester form (MEP), which has been reported at up to thirty times higher concentrations in human urine than metabolites of any other phthalate ester (15). The

highest levels were found in women, possibly reflecting differences in frequency of use of personal care products (16).

Changes to the DNA of human sperm cells were found to be more prevalent in individuals who also show high levels of MEP in their urine (15); further studies are necessary to determine if there is a causal relationship. Other research has identified a possible link between exposure to two phthalate metabolites, namely MEP and MBP (monobutyl phthalate), measured in urine samples and restricted lung function in adult men (17). A recent study found an association between male genital development and some phthalate metabolites, including MEP and MBP. These findings support the hypothesis that prenatal phthalate exposure at environmental levels can adversely affect male reproductive development in humans (18).

### **EXISTING CONTROLS**

In 1998, the OSPAR Commission agreed on the target of cessation of discharges, emissions and losses of all hazardous substances to the marine environment by 2020. DBP and DEHP were included on the first list of chemicals for priority action towards achieving this target (19).

The use of phthalates in soft PVC children's toys has been an issue of major concern because children can ingest these softeners by sucking and biting on the toys. At the end of 1999 the EU unanimously agreed to a three-month ban of certain phthalates in toys which could be put in the mouth and in childcare articles for the under-threes. This ban was repeatedly extended and was therefore actually in place for years.

In July 2005 the European Parliament decided to a permanent ban on the use of DEHP, DBP and BBP in all toys and childcare articles. The EP also agreed to ban the phthalates DINP, DIDP and DNOP in toys and childcare articles that can be put in the mouth whether or not they are intended for this use (20).

The new European chemicals legislation REACH is expected to come into force in 2006. If regulators show willing it would provide an excellent opportunity to phase out other uses of these chemicals.

### **ALTERNATIVES**

Phthalates are mainly used in PVC. The best solution is to replace PVC products with other materials: linoleum, tiles, wood or carpet on the floor instead of vinyl. The majority of alternatives are widely available. Electricity cables made from polypropylene (PP) function just as well as PVC. Alternatives for soft PVC toys are toys made of less harmful plastics or cloth toys. PVC-free and DEHP-free alternatives are available for almost every use of PVC in the health care setting. DEP could be replaced as an alcohol denaturant or its use abolished.

<rijtje kikap in klein corps>

**BBzP:** butyl benzyl phthalate - **DEHP:** di-(2-ethylhexyl) phthalate – **DEP:** diethyl phthalate - **DIBP:** di-iso-butyl phthalate - **DIDP:** di-iso-decyl phthalate - **DINP:** di-iso-nonyl phthalate – **DMP:** dimethyl phthalate – **DOP:** di-n-octyl phthalate - **DBP:** di-n-butyl phthalate - **DCHP:** diphenyl phthalate

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## TRICLOSAN

**Triclosan is a commonly used antibacterial agent for products including detergents, soaps, cosmetics, deodorants, toothpastes and mouthwashes (1, 2).**

### PRODUCTION AND USE

Triclosan, known as 5-chloro-2-(2,4-dichlorophenoxy)phenol (3), is used as an antibacterial agent in products like mattress pads, food cutting boards, shoes and sportswear (4). Fibers and polymers impregnated with Triclosan have names such as Ultra-Fresh, Amicor, Microban, Monolith, Bactonix and Sanitized (1).

### DISTRIBUTION IN THE ENVIRONMENT

Triclosan is a relatively stable, lipophilic compound (5). Triclosan can enter the aquatic environment in significant quantities via effluent from wastewater treatment plants (5, 6). A Swedish study found that methyl triclosan is formed from triclosan during processing at such plants (5). It was detected at consistently lower concentrations than triclosan itself, but is more persistent than triclosan.

Triclosan has also been detected in the bile of wild fish that were living downstream from wastewater treatment plants in Sweden (1). It was also found in caged experimental fish placed in the same environment. A Swiss study detected methyl triclosan in fish (7). The study noted that results were consistent with previous research in Japan that had reported methyl triclosan in fish in the Tama River.

Triclosan was also found in blood plasma in a Swedish study (8). High levels of triclosan were found in human breast milk in another Swedish study (1).

### EFFECTS

EU Directive 67/548 classifies Triclosan as 'very toxic to aquatic organisms' and it is bioaccumulative. Under certain conditions, triclosan is converted to various by-products by heat and UV irradiation (9), including some di- and tri-chlorinated dibenzo-p-dioxins. But these congeners are not among the most toxic and bioaccumulative forms of dioxin.

Triclosan is reported to be toxic to rainbow trout and to the aquatic invertebrate *Daphnia magna* (1). Other studies suggest that triclosan is highly toxic to the early life stages of fish (medaka), and that some metabolites may be weakly oestrogenic (10). A major concern of triclosan contamination in environmental surface waters is its toxicity to certain algae (4).

Triclosan has been shown to be a skin irritant in rabbits (11) and a contact allergen (12, 13). Studies showed that triclosan affected liver enzymes in rats and that this could contribute to its toxicity (9, 14).

One study has demonstrated multidrug resistance (MDR) conveyed by triclosan to *Pseudomonas aeruginosa* (15), a bacterial strain of foremost clinical importance. *Pseudomonas aeruginosa* is a cause of death in many hospital-acquired infections due to its intrinsic resistance to many antibiotics. The results of this study raised the notion that widespread and unregulated use of triclosan may compound antibiotic resistance.

### EXISTING CONTROLS

In Sweden, the use of phenolic antibacterial substances like triclosan in hospitals was abandoned several years ago since they were considered unnecessary in practice (1).

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**For background information see the following reports:**

- 'Chemical Legacy, Contamination of the Child', C Dorey/Greenpeace UK 2003
- 'The Poison Link, The story nobody wants to hear', Greenpeace Netherlands April 2004
- 'Compromising our Children, chemical impacts on children's intelligence and behaviour', WWF-UK 2004
- 'Chemical footprints in blood', Greenpeace Netherlands 2004
- 'Sick of chemicals – a review of the evidence', Chemical Reaction and the European Public Health Alliance Environment Network 2005
- 'Man-Made Chemicals in Maternal and Cord Blood' - RBJ Peters/TNO 2005

**Or check the following websites:**

- [www.greenpeace.org/chemicals](http://www.greenpeace.org/chemicals)
- [www.panda.org/campaign/detox](http://www.panda.org/campaign/detox)
- [www.chemicalreaction.org](http://www.chemicalreaction.org)