



# COMPROMISING OUR CHILDREN

**CHEMICAL IMPACTS ON CHILDREN'S  
INTELLIGENCE AND BEHAVIOUR**

A WWF-UK Chemicals and Health Campaign Briefing  
June 2004



Compromising  
our Children –  
chemical impacts  
on children's  
intelligence  
and behaviour

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Campaign Briefing*

WWF-UK, June 2004



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# Foreword

Much of my life has been spent campaigning to try and right wrongs in our society. A large proportion of that time has been dedicated to helping children to be heard and protected from danger.

Reading this report I was struck by the significant threat that man-made chemicals in current use pose to our society, our children and our future. Unborn, developing babies and children may be disproportionately affected. This is unacceptable. We should not be using synthetic chemicals that have not been assessed for their safety.

Consumers need to be listened to - there is surely a need to replace the very worst chemicals with safer alternatives. We don't want chemicals that build up in our bodies and in the environment. Nor do we want chemicals that interfere with children's development or that affect their intelligence and behaviour. For the baby developing in the womb, no risk is acceptable.

In the 20<sup>th</sup> century man-made chemicals became an integral part of our lives and brought us many benefits. Now, in the 21<sup>st</sup> century, it is time for the chemical industry to become more responsible. They must recognise the problems associated with certain chemicals and eradicate them.

I urge everyone to join WWF, the Women's Institutes and The Co-operative Bank in calling for safer chemicals in everyday use.

A handwritten signature in black ink that reads "Esther Rantzen". The signature is written in a cursive, flowing style.

Esther Rantzen  
April 2004



# Introduction

Imagine how you'd feel if you woke up to find that mankind had foolishly managed to pollute the entire face of the Earth with toxic chemicals, and that those chemicals were not only contaminating wildlife species such as otters, whales and polar bears, but also people.

Then imagine how you'd feel if you discovered that pregnant women couldn't stop passing those chemicals on to the babies in their wombs, and that those chemicals were affecting the brain development and intelligence of children. Scary, isn't it? Yet that's what's happening.

Across Europe children are suffering from the effects of man-made chemicals. Chemical contaminants are accumulating in mothers and are passed on while the baby is in the womb. At "high background" levels (the upper range of the levels normally found) chemicals have harmed, and are continuing to harm, our children and are impairing their ability to make sense of their world.

But you don't need to take only WWF's word for it. A report for the World Health Organisation (1) concludes, "*exposure (particularly prenatal exposure) to certain endocrine disrupting chemicals (e.g. PCBs) can have adverse effects on neurological development... and behaviour... delays in...cognitive development have been found to be associated with neonatal PCB exposure.*"

WWF's biomonitoring survey in 2003 demonstrated that irrespective of where we live or what we do, we are all contaminated with a cocktail of toxic man-made chemicals. Some of these chemicals can adversely affect the brain and the wider nervous system. The most well-known are the ubiquitous pollutants, now banned, such as PCBs and DDT. Unfortunately, the chemical industry does not seem to have learned its lesson: there are many other man-made chemicals still being produced and used today in everyday products in the home and workplace. Data suggests that these may cause learning and behavioural difficulties.

In humans, the brain and nervous system are very vulnerable because development takes place over a long period. It begins early in the womb and continues through puberty. The developing brain is uniquely sensitive, and effects on brain function and coordination can occur in children at levels that would not cause permanent effects in an adult.

Unless we take action now, it may be that our children won't be as intelligent as they might be because of man-made chemicals. Worse still, they may develop behavioural problems. Our children are our future – and our future is under threat.

## Interference with our body's chemical messenger system

Some man-made chemicals can disrupt the hormone system, and can particularly interfere with the sex and thyroid hormones (2, 3). Thyroid hormones play an important role in orchestrating normal brain development and, along with sex hormones, guide sexual differentiation of the brain. A large number of man-made pollutants are either known to alter, or are suspected of altering, thyroid hormone function, and exposure of animals to such chemicals during early development can result in permanent effects on brain function (4).

While knowledge about a few neurotoxicants (substances that are toxic to the brain and the nervous system, such as lead and mercury) is good, very little is known about the neurotoxicity of the vast majority of chemicals. Past experience shows that effects of chemicals are often not fully understood until it is too late, when generations of children have already been exposed to unsafe levels. Exposure to neurotoxicants commonly found in the environment could account for a wide variety of cases of mental retardation currently classified as due to unknown causes.

This is a complex issue. Some scientists consider it likely that multiple effects may interact to produce observed effects (4). Whatever their precise mode of action, it is clear that some man-made chemicals can cause the wiring circuitry of the brain to go awry.

# Uncontrolled global experiment

The vast majority of the 70,000 or so man-made chemicals on the market in the EU today have never been adequately assessed for safety, and only a very small handful have ever been tested for their neurotoxic effects on developing offspring. A study of the data available on chemicals produced or imported in the highest quantities in the EU (approximately 2,500), found that around 70 per cent did not have data on developmental toxicity or teratogenicity (the ability to cause birth defects) (5). Similarly, a US study in 1998 estimated that of the 2,864 high production volume chemicals in the US, around 78 per cent had no information on developmental toxicity (6, 7).

Of particular concern are chemicals that are very persistent (meaning they remain in the environment for a long time and don't break down) and very bioaccumulative (meaning they build up in living things) that may also possess neurotoxic properties. Their potential to persist in the environment, including people's bodies, for long periods and their ability to adversely affect developing fetuses when contaminated women become pregnant, mean such chemicals pose a particular threat. They can be passed on to children through the placenta and during breast feeding. Unfortunately, many bioaccumulative chemicals have been banned too late to prevent effects from continuing in subsequent generations.

# Hidden dangers

Hazardous man-made chemicals that can interfere with hormone systems and which may affect the intelligence and behaviour of our children occur in many sometimes surprisingly familiar places.

## **INCINERATORS, POWER STATIONS AND FACTORIES**

Incinerators, coal and oil power stations, industrial combustion sources, chlorinated chemical factories, metal processing factories, and open burning and accidental fires are all sources of highly toxic chemicals called dioxins and furans (8). These are only accidentally produced, and arise when chlorinated compounds are incompletely burned. Our intake of dioxins is mainly through food. Particularly elevated levels may occur in food from areas where there are local emission sources, or when contamination of animal feed has occurred.

## **OLD TRANSFORMERS, PAINTS AND FRIDGES**

Until the 1970s, the electricity and mining industries made extensive use of chemicals called polychlorinated biphenyls (PCBs). Their ability to withstand heat meant that they were widely used as heat transfer fluids in capacitors, transformers and electrical switching gear. They also had some use in fridges, as lubricants, in paints and varnishes, as flame-retardants, in sealing materials in construction, in carbon-less copying paper, and as capacitors in strip lights and motorway lights. Although the production of PCBs was banned in the UK in 1978, the programme to collect them for “destruction” continues.

PCBs are also formed accidentally, and may be released along with dioxins during combustion processes where chlorine is involved. For example, unintentional sources of PCBs and/or dioxins include incinerators, power stations, thermal processes in the metallurgical industry, open waste burning, some chlorinated chemical factories, waste oil refineries, and vehicles burning leaded petrol. Food that may be particularly contaminated with PCBs and dioxins includes fish and fish oils, meat and animal fats, and milk and dairy produce. A study published in January 2004, which found high levels of PCBs and other man-made chemicals in Scottish salmon and salmon farmed elsewhere, highlights the fact that PCBs are a current and continuing contamination issue (79).

## **COMPUTERS, TVs, FURNITURE, CARS AND VIDEOS**

Computers, televisions, furniture, cars, videos, textiles and the like can all contain brominated flame retardant chemicals used to prevent fire starting or rapidly spreading. Preventing deaths from fire is undoubtedly necessary, but alternative methods and approaches are available. Making articles out of less flammable materials in the first place is one option; others are using better thermal barriers in electrical equipment or finding safer chemicals to slow the onset and spread of fire, as well as making better use of smoke alarms, fire escapes and sprinkler systems. The Swedish Rescue Services Agency has examined this issue and supports the phase-out of brominated flame retardants (9).

Some brominated compounds have very similar properties to PCBs. There is a real concern that these chemicals might act together with PCBs to produce additive or synergistic adverse effects.

In animals they seem to have similar effects to PCBs on learning behaviour (10, 11, 12). Alarming, the amount of certain brominated flame retardant chemicals in our bodies has increased dramatically over recent decades (13) and particularly high levels have been found in the UK and the US (14, 15). Despite the fact that these chemicals are now found to contaminate both people and wildlife, they are still being produced and released into the environment.

In the EU, the marketing of two of the three polybrominated diphenyl ether (PBDE) commercial flame retardants – pentabrominated diphenyl ether (penta-BDE) and octabrominated diphenyl ether (octa-BDE) – will be banned in August 2004. But despite the ban, the persistence of these chemicals means that we will have to live with them in our bodies for many years. Decabrominated diphenyl ether (deca-BDE) is used as a flame retardant in the plastics and textiles industries, and is still in widespread use. Moreover, brominated flame retardants (BFRs) are a chemically diverse group, and the fear must be that if and when the PBDE compounds are banned, other BFRs may simply take their place. Already, another brominated flame retardant chemical, HBCD (hexabromocyclododecane), which is in widespread use in a diverse range of products such as car cushions, insulation boards, textiles, mattress ticking, video housings and wire coatings, has also been found to affect learning and memory function in animals (16).

Apart from dietary intake, airborne dust particles contaminated with BFRs are probably a significant source of human exposure (see 17, 18). Indeed, a recent study found surprisingly high concentrations in house dust in the UK and Europe (19).

### **BOTTLES, CAN LININGS AND FILLINGS**

Many tin can linings, clear plastic re-usable water containers, baby feeding bottles and “white” dental fillings contain another hazardous chemical called bisphenol A (BPA). BPA is produced in high volumes and is the chemical used to make polycarbonate plastic. Low levels have been found in liquids stored in polycarbonate containers. Of particular concern is the leaching from baby feeding bottles that can lead to direct exposure of very young infants. BPA also leaches into food contained in tins lined with an epoxy-resin coating. Exposure to BPA and related substances can also arise from dental fillings and sealants (for review see (20)).

# Serious impacts

Man-made chemicals are affecting our children's intelligence and behaviour, compromising their ability to make sense of the world and affecting their movement skills.

Much of the existing evidence of chemical impacts on people focuses on ubiquitous pollutants such as PCBs. Researchers are reliant on epidemiological studies that by their nature can only be conducted after a number of years when sufficient data is available. Data is available for older chemicals, but for many newer ones, it is non-existent. We are therefore faced with an incomplete picture of the total impact of man-made chemicals on intelligence and behaviour. This does not mean that all man-made chemicals currently in use are harmless. What follows is a summary of the existing evidence.

## **DECREASED INTELLIGENCE AND ALTERED MOVEMENT SKILLS**

Effects on brain development associated with PCBs first came to light about 20 years ago when scientists began to see impacts on children born near Lake Michigan in North America. These children had been affected not by anything they had done, but by what their mothers had done. Their mothers had eaten fish contaminated by PCBs and this had affected the brain development of their children.

Effects on visual recognition were seen in babies exposed to higher levels of PCBs in the womb (21) and later tests showed that at four years of age these children did less well in short-term verbal memory tests predictive of learning ability (22). Studies elsewhere in the Great Lakes region backed up this data and found that PCBs were affecting children's mental development and intelligence (23). When the children from around Lake Michigan were re-examined at age 11, those with higher exposure to PCBs were three times as likely to have low average IQ scores and twice as likely to be at least two years behind in reading comprehension (24).

Table 1 summarises the impacts on children that have been linked to PCB exposure. It is not unfounded to suggest that similar effects might be caused by chemicals such as BFRs used in furniture and electronic goods (see above) that are structurally similar to PCBs.

PCBs have been shown many times to adversely affect neurological development. Studies in Michigan and New York, and in the Faeroe Islands, Germany, the Netherlands and Taiwan, have all shown negative associations between prenatal PCB exposure and cognitive function in infancy or childhood (for review see (25)). PCBs have also been shown to induce neuro-behavioural alterations in animals (26). Furthermore, in the US, middle-aged and older people with high levels of these PCB chemicals in their blood have impaired learning and memory (27).

**Table 1: Neurological effects on children associated with in-womb exposure to PCBs at high background levels**

Age	Effect
Babies	have increased startle responses and tremors, and pay less attention to visual and auditory stimuli (28).
Toddlers at 18 months	have less developed grasping, sitting, crawling, standing and walking skills (29).
Infants at age 3_	have decreased cognitive mental development (30).
Children at age 7	(from less than optimal home environments) show reduced cognitive development and delays in motor development (31).
Children age 7_	show altered gender-typical play behaviour – less masculinised play in boys and more masculinised play behaviour in girls (2) (see below).
Children at age 11	show deficits in memory and attention, decreased IQs and reduced reading comprehension (24).
Children at age 4_	show an inability to control inappropriate responses, which might be suggestive of Attention Deficit Hyperactivity Disorder (ADHD) (32) (see below).

While PCB exposure has been associated with effects on thyroid hormone levels in animals (4) and humans (33), it is still not clear whether neurological symptoms associated with such exposures are a direct result of thyroid disruption or some other mechanism (34, and for review see 35).

There is also evidence to suggest that dioxins might contribute to several adverse effects in people, including endometriosis, developmental neurobehavioural (cognitive) effects, developmental reproductive effects (sperm counts, female urogenital malformations) and immunotoxicity. The World Health Organisation (WHO) recommends a Tolerable Daily Intake (TDI) for dioxins and dioxin-like PCBs of 1-4 picograms WHO-TEQ (Toxic Equivalent Quotient) per kilogram body weight per day (36). People consuming relatively modest amounts of highly contaminated foods can certainly exceed these TDIs.

PCBs and dioxins may not be the only man-made chemicals implicated in the dumbing down of our children. Poly brominated diphenyl ethers (PBDEs), one class of BFRs, are another group of chemicals that have been reported to cause behavioural effects or learning deficits in animals (12, 37, 38). They all appear to have some effect on thyroid hormone or the thyroid gland, and to affect brain development. For example, several rodent studies indicate that commercially obtained penta-BDE (and pure tetra-BDE) can exert effects on thyroid hormones (39, for review see review 40). Similarly, octa-BDE can interfere with the thyroid hormone system (41). The evidence for the thyroid effects of deca-BDE is less clear, but the European risk assessment report suggests it may cause thyroid tumours in mice. While these effects are found at levels much higher than those to which humans are generally exposed, workers exposed to deca-BDE along with other brominated flame retardants exhibited a higher than normal prevalence of hypothyroidism (42). Other mammals are also vulnerable: for example, it has been found that

there is a significant relationship between thyroid hormone levels in the blood of grey seal weaned pups and juveniles, and PBDE levels in their blubber (43).

The increasing levels of these chemicals found in humans and wildlife underline the concerns regarding the reported effects on brain function and thyroid hormone action. Studies in Sweden showed that the sum of PBDE concentrations in breast milk increased 57-fold between 1972 and 1997 from 0.07 ng/g to 4.0 ng/g lipid, such that every five years the levels doubled (13). Levels have since declined in Sweden, but reports of work carried out at Lancaster University suggest much higher levels may be found in UK breast milk, with levels ranging from less than 1 ng/g to 69 ng/g lipid, with more than half the women having levels of 6ng/g or more (see 15).

Many pesticides have also been associated with effects on brain function and with thyroid disruption. The pesticides which are particularly under the spotlight with regard to neurotoxic effects include the organophosphates, DDT, pyrethroids and paraquat (44, 45, 46). A study in Mexico has shown startling effects in children believed to be exposed to high levels of pesticides in an area with intensive agriculture. A range of symptoms was seen, including poor hand and eye coordination, diminished memory, decreased physical stamina and decreased ability to draw a person, which is used as non-verbal measure of cognitive ability (47).

#### **ALTERED MASCULINE AND FEMININE BEHAVIOUR**

In addition to compromising children's ability to process information, chemicals may be affecting the developing nervous system in other ways. As well as affecting intelligence, it seems that dioxins and PCBs are also tampering with the male and female behaviour patterns of children. Such effects might be due to the ability of PCBs and dioxins to disrupt the sex hormones, as both these chemicals are known to have sex hormone-disrupting properties (2). The sex hormones not only influence reproduction, but also non-reproductive behaviour that shows sex differences (4).

In Europe, researchers studying Dutch children exposed to background levels of pollution found that the effects of prenatal exposure to PCBs were different for boys and girls. In boys, higher prenatal PCB levels were related to less masculinised play, whereas in girls, higher exposure was linked with more masculinised play. On the other hand, higher prenatal dioxin exposure was associated with more feminised play in boys as well as girls (2). While this work is controversial, these effects are alarming and warrant more research to verify and understand the full implications.

BPA is also known to have oestrogen (female sex hormone) mimicking properties, and as such is a hormone-disrupting chemical. In addition to effects on the uterus in animals, it is reported to cause reduced nursing behaviour (48), more masculinised play behaviour in females (49) and increased aggression in males (50), and to abolish the sex differences in open-field behaviour (51).

## **ATTENTION DEFICIT HYPERACTIVITY DISORDER**

Scientists now suspect that man-made chemicals may be contributing to a range of learning disabilities, including attention deficit hyperactivity disorder (ADHD). ADHD manifests itself as several symptoms including problems with paying attention and difficulty in controlling impulsive behaviour. It has been suggested that although many factors are liable to be implicated in causing ADHD, neurotoxic chemicals may also contribute to its incidence (52).

This is particularly worrying because the disorder known as ADHD is estimated to affect around one in 20 children in the US (53), and in a significant number of individuals, some symptoms may persist into adolescence and adulthood (52). In Britain, prescriptions of the drug Ritalin, used to treat ADHD, increased markedly during the latter part of the 1990s (54). The European Commission has registered its concern, and has warned that *“the occurrence of developmental disabilities, such as learning disabilities, intellectual retardation and attention deficit hyperactivity disorder is certainly large enough to constitute a significant public health problem”* (55).

Some studies suggest the involvement of chemicals. For example, “response inhibition” is frequently impaired in children with ADHD, and studies have shown a dose-dependent association between PCB levels in children and an inability to prevent inappropriate behavioural responses – which might be a predictor for ADHD. Brain (MRI) scans showed that children with sup-optimal development of certain areas of the brain seemed more vulnerable to the effects of PCBs. The smaller the splenium, (the back part of the bundle of fibres joining the two brain hemispheres) the larger the association between PCBs and response inhibition (32). Indeed, measurable effects on the brain seem to occur with ADHD – and patients with ADHD have been found to have smaller brain volumes than normal children (56). This gives weight to the suggestion that ADHD is a real, biologically-based phenomenon, and not just a disorder conjured up by “neurotic” parents.

## **AUTISM**

There is a concern that autism may be partly linked to chemical exposures, and that this developmental disorder has increased in recent years. Autism, a brain condition that is evident prior to three years of age, affects a person’s ability to form relationships and to behave normally in everyday life. There are no medical tests to determine whether a person has autism and diagnoses are based on observed behaviour. Autism is the term often used for the more severe cases, whereas the term autism spectrum disorders (ASD) includes “milder” forms of autism such as Asperger’s.

Studies of identical twins confirm a genetic component, and there is certainly a predisposition to autism condition in some families. Recent findings point to the possibility that the disorder spectrum is caused by a gene-environment interaction. Thus, it may be that to produce autism, it is necessary to have both susceptible genes, as well as some environmental (e.g. chemical) assault on these genes. Many chemicals have been mentioned as possibly playing a role. These include metals, some organochlorine and organobromine compounds, and some pharmaceuticals (see review (57)). One suggestion is that chemicals might cause damage around the time of neural tube closure in the womb, perhaps by disrupting retinoids (see 58, 59). Other researchers consider that differences in metabolism may be important (60). Mercury has been the focus of much concern, both with regard to infant exposure, due to its use as a preservative

in vaccinations, and with regard to exposure in the womb, largely due to mothers eating fish contaminated with mercury (61), and particularly because it seems that mercury levels in the umbilical cord of newborns are higher than in their mother's blood (62). A large study in Denmark did not find a link with postnatal exposure to mercury in the MMR (measles mumps and rubella) vaccine (63), but the US Institute of Medicine is reviewing all the evidence on the potential effects of mercury in vaccines and will report later in 2004 (64).

The increase in the frequency of the disorder certainly supports the suggestion of an environmental component. It seems that genetics loads the gun, but the environment pulls the trigger.

Many studies have suggested that rates have risen over the years, although some reviewers caution that increased recognition of the disorder, coupled with other factors, may account for some of the increase. Nevertheless, it does appear that autism spectrum disorders are more prevalent than previously thought, and may be found in around six children per 1,000 or one in 166 children (65). The National Autistic Society suggests it may be nearer one in 110 children and points out that two thirds of teachers surveyed in England and Wales felt there were now more children with autism spectrum disorders than just five years ago (66). Translated nationally, around half a million people may suffer from autism spectrum disorders. The rates of autism itself are lower and estimated to be 16.8 per 10,000, or one in 600 children (67, 68).

## A real and pervasive problem

Exposure to man-made chemicals at levels that can adversely affect the nervous system of our children is commonplace. Commenting on our exposure to dioxins and dioxin-like PCBs, WHO experts have recognised that “*subtle effects may already occur in the general population in developed countries at current background levels of two to six picograms/kilogram body weight*” (69).

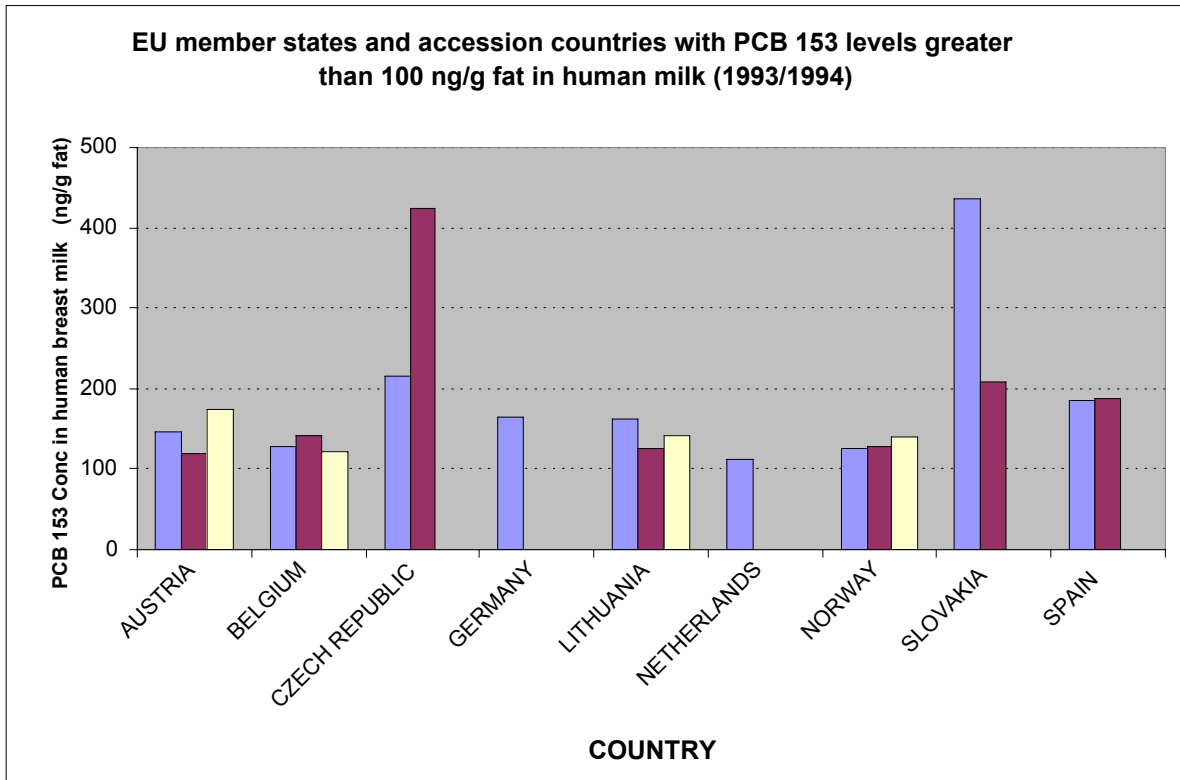
Similarly, for the non-dioxin like PCBs, it seems that the levels found in some mothers have exceeded those that are associated with effects on brain development in their infants. Scientists have tried to convert the data in the various studies into comparable forms, by comparing calculated or measured levels of a certain type of PCB, called PCB 153 (70). They found that the PCB concentration associated with adverse effects on neurological function in the study of infants and mothers living around Lake Ontario in the US seemed to be around 40 ng PCB 153 per gram of maternal serum lipid, while levels in mothers of children affected in Rotterdam were around 100 ng per gram of maternal serum lipid. See Table 2.

**Table 2: Median maternal serum levels of PCB 153 – associated with adverse neurological effects**

Study	Location	Specimen years	median serum concentration PCB 153 (ng/g lipid)
Darvill et al.,2000(23)	Oswego, New York County, Lake Ontario, US	1991-1994	40
Patandin et al., 1999 (30)	Rotterdam and Groningen, Netherlands	1990-1992	100
Walkowiak et al., 2001 (74)	Düsseldorf, Germany	1993-1995	140

Studies coordinated by the WHO have revealed that many places in the industrialised world have background levels of PCBs similar or higher than these, and this warrants some concern. While comprehensive EU-wide data is lacking, studies suggest that in certain areas of Britain, (71) and in some industrialised cities in Austria, Belgium, Germany, Italy, the Netherlands and Spain (see Figure 1), some women were contaminated with levels of PCBs that were not widely dissimilar to those found in the women who gave birth to children with neurological impacts. The highest levels in European women have been found in the Danish Faeroe Islands, (where neurological effects were linked mostly with mercury rather than PCBs) (25) and in some eastern European countries such as the Czech Republic and Slovakia (70,72,73).

Figure 1: EU member states and accession countries with PCB 153 levels greater than 100 ng/g fat in pooled human milk samples taken in 1993 or 1994 (71).



NB: The different bars for each country relate to different locations of pooled samples in that country.

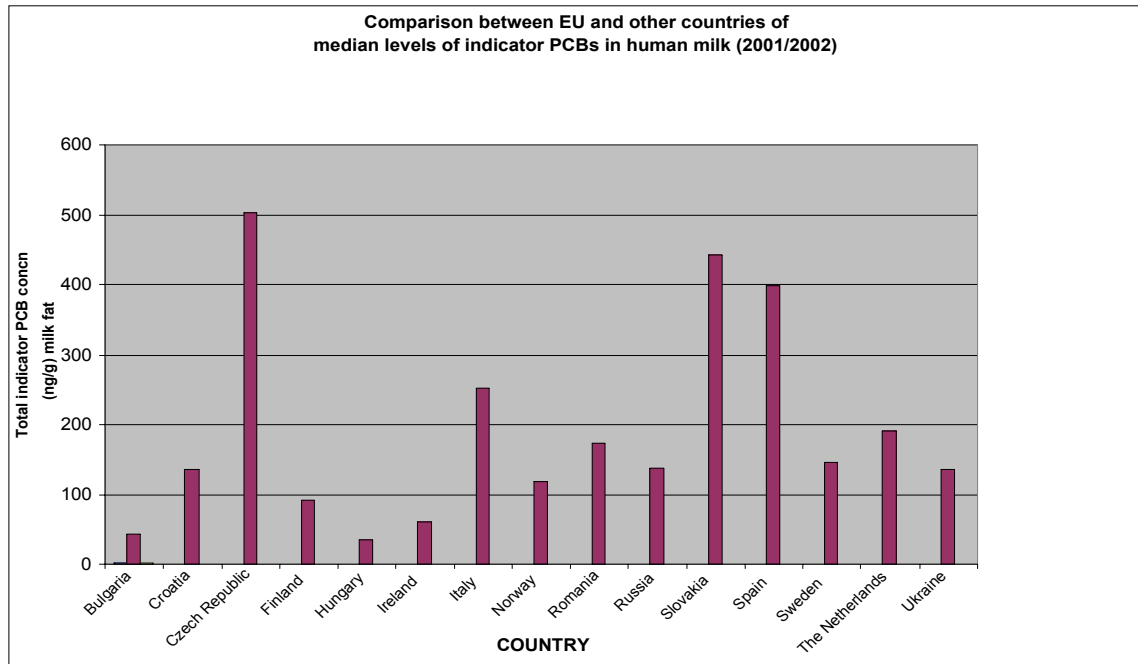


Figure 2: Comparative levels of indicator PCBs (28, 52, 101, 138, 153 and 180) in human milk in 2001-02

NB: The values in the two figures are not directly comparable, as Figure 1 shows only PCB 153 values and Figure 2 shows total indicator PCB values. Studies suggest that typically PCB 153 makes up roughly around 43 per cent of total *indicator* PCBs (72).

Studies have shown that brain development in children living in industrialised European countries has been affected by chemicals that have accumulated in their mothers and are passed on from the mother while the baby is in the womb. Levels of PCBs in maternal and placental cord blood have been used to compare the abilities of the children with high exposures in the womb, with the abilities of children with lower exposures. Studies on children in the Netherlands (30) and Germany (74) have confirmed fears that in certain European cities widespread harm appears to have been caused to children simply by “high background” levels of these chemicals. While the observed neurodevelopmental deficits in European children have sometimes been described as “subtle”, there could be unknown consequences related to their future intellectual ability (75).

Comparing the levels of PCB breast milk contamination shown in Figure 1 with the PCB levels listed in Table 2 gives some indication of the likelihood of effects. However, the values cannot be directly compared because they show levels of PCB 153 in human milk and human serum respectively. The ratio of PCBs in milk lipid to serum lipid may be around 1.3:1 (see 70).

The data shown in Figure 2 suggests that children in many of the countries listed, particularly infants born to the most highly exposed women in the Czech Republic, Italy, the Netherlands, Romania, Russia, Slovakia and Spain, are likely to be suffering from adverse effects.

In the UK, the only government-conducted survey of PCB levels in human milk analysed samples taken in 1993-94. However, due to serious doubts about the accuracy of the analysis, it is difficult to compare the range of levels of PCBs found in these women with the levels found in women from other countries where effects on infants have been noted. The results of this study that were published in the 1996 WHO report should be totally ignored, because they were based on provisional data and the laboratory had technical difficulties (76). Furthermore, a subsequent re-analysis and report of the levels found in these samples (77) also appears to be unreliable. The raw data provided shows that the levels of PCB 153 reported were proportionately so low compared with the total indicator PCB levels, that this author considers the data to be flawed.

Overall, the UK government’s strategy for evaluating *in utero* and early life exposure to harmful chemicals has been inadequate and lacking in proper oversight. However, studies conducted by Lancaster University provide a clearer insight into the range of PCB contaminant levels found in the UK population. In 1990 and early 1991, 115 Welsh breast milk samples were collected and screened for 50 PCB congeners (71). Our analysis is that the levels reported in this study are not dissimilar to those found in other studies where effects were reported, so that effects on brain function are considered likely to have occurred in the infants of the mothers with the highest levels of contamination. A subsequent study of breast milk, funded by Lancaster University, is expected to be published in due course. In discussing or reporting the contamination of breast milk, it should be remembered that it is prenatal exposure that is likely to blame for any effects, with the analysis of breast milk serving to provide a useful indication of the level of this earlier exposure. Breast-feeding is considered to be beneficial, and should be encouraged.

## Cost

There are great emotional costs to the families of children with impaired brain function. The heartache of parents with children suffering from behavioural disorders and learning disabilities is immense, as is the burden on the children themselves. Responding to the needs of affected children is also costly in financial terms.

Take autism as an example. Even with an assumed prevalence of just five children per 10,000 (which is three times less than current estimates of the prevalence of autism (68) and at least 10 times less than the number of children estimated to be affected by autism spectrum disorders), the estimated annual societal cost of autism in the UK exceeds £11 billion, and the lifetime cost for a person with autism exceeds £12.4 million (78). If autism spectrum disorders were included, this figure would increase dramatically, highlighting the huge costs of autism and autism spectrum disorders to society as a whole.

Estimating the proportion of the disease burden caused by exposure to toxic chemicals is difficult. However, the National Academy of Sciences in the US has estimated the fraction of neurobehavioural disorders that may be attributed to environmental factors. They considered that three per cent were caused directly by toxic environmental exposures, and another 25 per cent caused by interactions between genetic susceptibility and environmental factors, defined very broadly – so a total of 28 per cent caused wholly or partly by environmental factors. In the US, it has been estimated that within this total, some 10 per cent are at least partly caused by toxic exposures, not including alcohol or tobacco or drugs of abuse. Looking only at mental retardation – defined as IQ below 85, and cerebral palsy and autism – a cost of US\$9.2 billion per year was arrived at for that proportion caused by toxic chemicals of human origin in environmental media (79). This estimate specifically excluded the effects of lead, alcohol and tobacco. As a very approximate estimate, taking the rates of disease and costs incurred as similar to those in the US, this would equate to an annual cost in Britain of approximately £1 billion. Therefore, looking just at mental retardation, and cerebral palsy and autism, a very rough estimated figure of £1 billion per year might be the cost in Britain of the effects of man-made toxic chemicals (excluding lead) on brain development in children.

It should also be noted that the above cost calculation does not include the potential effects of pollutants such as PCBs that may cause a few points reduction in IQ. However, even such subtle effects may take a high financial toll. For example, it was noted that Salkever calculated that the loss of one IQ point was associated with an overall reduction in lifetime earnings of 2.39 per cent (79). Furthermore, on a larger, national scale, it has been said that in this information age, the wealth of a nation is directly correlated with developmental health and aggregate intelligence (80).

A study in the US (79) considered that the costs of childhood diseases of environmental origin are likely to increase if children's exposures to inadequately tested chemicals are allowed to continue.

Better regulation of chemicals could produce an overall financial saving to society. An independent study undertaken for WWF-UK concluded that the value of the health benefits that

would accrue from implementing the proposed new and tighter EU chemicals regulation known as REACH – the Registration, Evaluation and Authorisation of Chemicals – would significantly exceed the costs of implementing such legislation (81).

# Chemicals and life

It is clear that hazardous industrial chemicals dramatically affect our quality of life. Children and wildlife have a right not to be contaminated. Parents have a right to expect that the products they buy for their home and the toys they buy for their children are safe and harmless. Man-made chemicals should have adequate safety data and should be neither bioaccumulative nor persistent, unless persistence is a required property – as in some construction materials. No unnecessary risk is acceptable where our children and our future are concerned, and where safer alternatives are available.

In the past, many persistent and bioaccumulative chemicals, such as DDT (82) and PCBs, have been banned too late to prevent damage. Now there is an ever more urgent need for action. Many more persistent and bioaccumulative chemicals, which take a very long time to break down in the environment and build up in living things, are in use today. Such chemicals should be phased out, irrespective of their currently known toxicity, because it is almost impossible to predict and test for the long-term effects of low-level exposures that may take years to appear in humans and other long-lived animals. If we get it wrong, it is our children who will pay the price.

The EU is negotiating new chemical legislation to regulate industrial chemicals. This is a once in a generation opportunity to create a safer future for our children and wildlife. WWF is calling for the legislation to:

- phase out chemicals that are persistent and bioaccumulative;
- phase out endocrine-disrupting chemicals; and
- substitute these chemicals with safer alternatives and allow their continued use only where there is an overwhelming societal need, where no safer alternatives exist, and where measures to minimise exposure are put in place.

## REFERENCES

1. IPCS (International Programme on Chemical Safety) (2002). Global Assessment of the State-of-The-Science of endocrine disruptors. World Health Organisation (WHO).
2. Vreugdenhil H J, Slijper F M E, Mulder PG H, and Weisglas-Kuperus N (2002). Effects of Perinatal Exposure to PCBs and Dioxins on Play Behaviour in Dutch Children at School Age. *Environmental Health Perspectives*, Vol. 110 (10), pp593-598.
3. Porterfield S P (2000). Thyroidal dysfunction and environmental chemicals-potential impact on brain development. *Environmental Health Perspectives*, 108 (Suppl3), June, pp433-438.
4. Schantz S, and Widholm J (2001). Cognitive effects of endocrine-disrupting chemicals in animals. *Environmental Health Perspectives*, 109(12), pp1197-1206.
5. Allanou R, Hansen BG, van der Bilt Y (1999). Public Availability of Data on EU High Production Volume Chemicals. EUR 18996 EN. European Commission Joint Research Centre, Italy.
6. US EPA (1998). Chemical Hazard Data Availability Study: What do We Really Know about the Safety of HPV Chemicals? EPA's baseline of hazard information that is readily available to the public. EPA, Office of Pollution Prevention and Toxics (OPPTs), Washington DC.
7. Goldman L and Koduru S (2000). Chemicals in the environment and developmental toxicity to children: A public health and policy perspective. *Environmental Health Perspectives*, 108 (Suppl 3), pp443-448.
8. DEFRA (Department for the Environment Food and Rural Affairs) (2002). Dioxins and dioxin-like PCBs in the UK Environment: Consultation Document. October. DEFRA, London.
9. Albinson, Björn (2002). Alternative ways to achieve fire safety. Aug 2002. The Swedish Rescue Services Agency.
10. KEMI (Swedish National Chemicals Inspectorate) (1999). Phase out of PBDEs and PBBs, Report No 2/99, p35. KEMI, Solna, Sweden.
11. Eriksson P (1998). Perinatal Developmental Neurotoxicity of PCBs, Report 4897, KEMI (Swedish Environmental Protection Agency) Sweden.

12. Eriksson P, Jakobsson E, Fredriksson A (2001). Brominated flame retardants: A novel class of developmental neurotoxicants in our environment. *Environmental Health Perspectives*. 109(1), Sept, pp903-908.
13. Noren K and Meironyte D (2000). Certain organochlorine and organobromine contaminants in Swedish human milk in perspective of past 20-30 years. *Chemosphere* 40, pp1111-1123.
14. Hites RA (2004) Polybrominated Diphenyl Ethers in the Environment and in People: A Meta-Analysis of Concentrations. *Environmental Science and Technology*. Web release date: 8 January.
15. Kanasakie L (2003). Why do people's PBDE levels vary widely? *Environ.Sci.Technol.* 1;37(9), pp164A-165A.
16. Eriksson P, Viberg H, Fischer C, Wallin M, Fredriksson A (2002). A comparison on developmental neurotoxic effects of hexabromocyclododecane, 2,2',4,4',5,5'-hexabromodiphenyl ether (PBDE 153) and 2,2',4,4',5,5'-hexachlorobiphenyl (PCB 153). Abstract no 488. In *DIOXIN 2002*.
17. Sjodin A, Patterson D G Jr, Bergman A (2003). A review on human exposure to brominated flame retardants - particularly polybrominated diphenyl ethers. *Environ Int.* Sep;29(6), pp829-839.
18. Jakobsson K, Thuresson K, Rylander L, Sjodin A, Hagmar L, Bergman A (2002). Exposure to polybrominated diphenyl ethers and tetrabromobisphenol A among computer technicians. *Chemosphere*. Feb;46(5), pp709-716.
19. Santillo D, Labunska I, Davidson H, Johnston P, Strutt M and Knowles O (2003) Consuming Chemicals. Hazardous chemicals in house dust as an indicator of chemical exposure in the home. Greenpeace. UK
20. Lyons G (2000). Bisphenol A: A known endocrine disruptor. WWF-UK.
21. Jacobson S W, Fein G G, Jacobson J L, Schwartz P M, Dowler J (1985). The effect of intrauterine PCB exposure on visual recognition memory. *Child Dev* 56, pp853-860.
22. Jacobson J L, Jacobson S W, Humphrey H E. (1990). Effects of in utero exposure to polychlorinated biphenyls and related contaminants on cognitive functioning in young children. *J Pediatr* Jan;116(1), pp38-45.
23. Darvill T, Lonky E, Reihman J, Stewart P, Pagano J (2000). Prenatal exposure to PCBs and infant performance on the Fagan Test of Infant Intelligence. *Neurotoxicology* 21, pp1029-1038.
24. Jacobson L and Jacobson SW (1996). Intellectual impairment in children exposed to polychlorinated biphenyls in utero. *N Engl J Med* 335 (11), pp783-789.

25. Schantz S, Widholm J, Rice D (2003). Effects of PCB exposure on neuropsychological function in children. *Environmental Health Perspectives*, 111, pp357-376.
26. Faroon O, Jones D, de Rosa C (2001). Effects of polychlorinated biphenyls on the nervous system. *Toxicol. Ind. Health*, Sept 16(7-8), pp305-333.
27. Schantz S, Gasior D, Polverejan E, McCaffrey R J, Sweeney A M, Humphrey H E B, Gardiner J C (2001). Impairments of Memory and Learning in Older Adults Exposed to Polychlorinated Biphenyls Via Consumption of Great Lakes Fish. *Environmental Health Perspectives*, 109 (6), June, pp605-611.
28. Health Canada (1997). State of Knowledge Report on Environmental Contaminants and Human Health in the Great Lakes Basin. ISBN 0-662-26-169-0, Ministry of Public Works and Government Services, Canada.
29. Huisman M, Koopman-Esseboom C, Lanting C I, van der Paauw C G, Tuinstra L G, Fidler V, Weisglas-Kuperus N, Sauer PJ, Boersma E R, Touwen B C (1995). Neurological condition in 18-month-old children perinatally exposed to polychlorinated biphenyls and dioxins. *Early Hum Dev Oct 2;43(2)*, pp165-176.
30. Patandin S, Lanting C I, Mulder P G H, Boersma E R, Sauer P J J, Weisglas-Kuperus N (1999). Effects of environmental exposure to polychlorinated biphenyls and dioxins on cognitive abilities in Dutch children at 42 months of age. *J Pediatr*.134, pp33-41.
31. Vreugdenhil H J, Lanting C I, Mulder P G, Boersma E R, Weisglas-Kuperus N (2002). Effects of prenatal PCB and dioxin background exposure on cognitive and motor abilities in Dutch children at school age. *J Pediatr*. Jan;140(1), pp48-56.
32. Stewart P, Fitzgerald S, Reihman J, Gump B, Lonky E, Darvill T, Pagano J, Hauser P (2003). Prenatal PCB exposure, the corpus callosum, and response inhibition. *Environmental Health Perspectives*, 111(13) Oct, pp1670-1677.
33. Koopman-Esseboom C, Morse D C, Weisglas-Kuperus N, Lutkeschipholt I J, Van der Paauw CG, Tuinstra L G, Brouwer A, Sauer P J (1994). Effects of dioxins and polychlorinated biphenyls on thyroid hormone status of pregnant women and their infants. *Pediatr. Res.*, Oct;36(4), pp468-473.
34. Winneke G, Walkowiak J, Lilienthal H. (2002). PCB-induced neurodevelopmental toxicity in human infants and its potential mediation by endocrine dysfunction. *Toxicology*, Dec 27;181-182, pp161-165.
35. Renner R (2003). NIEHS News: NIEHS funded research pursues thyroid findings. *Environmental Health Perspectives*, 111 (12), September.
36. Van Leeuwen R and Younes M (1998). WHO revises the tolerable daily intake for dioxins. *Dioxin 98 Symposium*. Also in *Food Additives & Contaminants*, 17(4) of April 2000 (ISSN 0265-203X).

37. Viberg H, Fredriksson A, Jakobsson E, Örn U, Eriksson P (2001). Neonatal exposure to hexabromodiphenyl ether (PBDE 153) affects behaviour and cholinergic nicotinic receptors in brain of adult mouse. Poster presentation, Second International Workshop on Brominated Flame Retardants, May 14-16, Stockholm University, Sweden, pp275-278 (See also EU Risk Assessment Report for octa-PBDE).
38. Viberg H, Fredriksson A, Jakobsson E, Orn U, Eriksson P. (2003) Neurobehavioral derangements in adult mice receiving decabrominated diphenyl ether (PBDE 209) during a defined period of neonatal brain development. *Toxic Sci. Nov*; 76(1):112-20.
39. Hallgren S, Sinjari T, Hakansson H, Darnerud P O (2001). Effects of polybrominated diphenyl ethers (PBDEs) and polychlorinated biphenyls (PCBs) on thyroid hormone and vitamin A levels in rats and mice. *Arch Toxicol Jun*;75(4), pp200-208.
40. Olsson P-E, Borg B, Brunstrom B, Hakansson H, Klasson-Wehler E (1998). Endocrine disrupting substances, ISBN 91-620-4859-7, Swedish EPA, Stockholm.
41. Zhou T, Ross DG, DeVito M J, Crofton K M (2001). Effects of short-term *in vivo* exposure to polybrominated diphenyl ethers on thyroid hormones and hepatic enzyme activities in weanling rats. *Toxicological Sciences*, 61, pp76-82.
42. EU RAR (2002). European Union Risk Assessment Report for Deca – bis(pentabromophenyl ether – CAS No: 1163 19 5, EUR 20402, European Chemicals Bureau, Italy. For this and other RARs see: <http://ecb.jrc.it/existing-chemicals/>
43. Hall A J, Kalantzi O I, and Thomas G O (2003). Polybrominated diphenyl ethers (PBDEs) in grey seals during their first year of life – are they thyroid hormone endocrine disrupters? *Environmental Pollution* 126(1), pp29-37.
44. Eskenazi B, Bradman A and Castorina R (1999). Exposures of children to organophosphate pesticides and their potential adverse health effects. *Environmental Health Perspectives*, 107 (Suppl 3), pp409-419.
45. Dorner G, Plagemann A (2002). DDT in human milk and mental capacities in children at school age: an additional view on PISA 2000. *Neuroendocrinol Lett.*, Oct-Dec;23(5-6), pp427-431.
46. Eriksson P (1997). Developmental neurotoxicity of environmental agents in the neonate. *Neurotoxicology*.18 (3), pp719-726.
47. Guillette E A, Meza M M, Aquilar M G, Soto A D, Enedina I (1998). An anthropological approach to the evaluation of preschool children exposed to pesticides in Mexico. *Environmental Health Perspectives*, 106, pp347-353.

48. Palanza P, Howdeshell KL, Parmigiani S, vom Saal FS (2002). Exposure to a low dose of bisphenol A during fetal life or in adulthood alters maternal behaviour in mice. *Environmental Health Perspectives*, 110 (Suppl 3), June 2002, pp415-422.
49. Dessi-Fulgheri F, Porrini S, Farrabollini F (2002). Effects of perinatal exposure to bisphenol A on play behaviour of female and juvenile rats. *Environmental Health Perspectives*, June 110 (Suppl 3), pp403-407.
50. Kawai K, Nozaki T, Nishikata H, Aou S, Takii M, Kubo C (2003). Aggressive Behavior and Serum Testosterone Concentration during the Maturation Process of Male Mice: The Effects of Fetal Exposure to Bisphenol A. *Environmental Health Perspectives* Feb;111(2), pp175-178.
51. Kubo K, Arai O, Omura M et al (2003). Low dose effects on bisphenol A on sexual differentiation of the brain and behaviour in rats. *Neuroscience Research*, 45(3), pp345-356.
52. Rice D (2000). Parallels between Attention Deficit Hyperactivity Disorder and Behavioral Deficits Produced by Neurotoxic Exposure in Monkeys. *Environmental Health Perspectives*. Vol 108 (Supplement 3), June, pp405-408.
53. Goldman L, Genel M, Bezman R, Slanetz P (1998). Diagnosis and treatment of attention deficit hyperactivity disorder in children and adolescents. *JAMA*, 279(14), pp1100-1107.
54. Panorama 10 April 2000:  
see [www.wsws.org/articles/2000/apr2000/rit-a25.shtml](http://www.wsws.org/articles/2000/apr2000/rit-a25.shtml)
55. CEC (2003). Commission of the European Communities: A European Environment and Health Strategy, Brussels , 11.6.2003 COM (2003) 338 final. [www.europa.eu.int/comm/press\\_room/presspacks/health/pp\\_health\\_en.htm](http://www.europa.eu.int/comm/press_room/presspacks/health/pp_health_en.htm)
56. Castellanos F X, Lee P P, Sharp W, Jeffries N O, Greenstein D K, Clasen L S, Blumenthal J D, James R S, Ebens C L, Walter J M, Zijdenbos A, Evans A C, Giedd J N, Rapoport J L (2002). Developmental trajectories of brain volume abnormalities in children and adolescents with attention-deficit/hyperactivity disorder. *JAMA*, Oct 9, 288(14), pp1740-1748.
57. Wakefield Julie (2002) New Centers to Focus on Autism and Other Developmental Disorders. *Environmental Health Perspectives* 110 (1) January, pA20.
58. London E and Etzel R (2000). The environment as an etiologic factor in autism: A new direction for research, *Environmental Health Perspectives*, 108(Suppl3), pp401-404.
59. Megson MN (2000). Is autism a G-alpha protein defect reversible with natural vitamin A? *Med Hypotheses*. June;54(6), pp979-983.

60. Alberti A, Pirrone P, Elia M, Waring R H, Romano C (1999). Sulphation deficit in low-functioning autistic children: a pilot study. *Biol Psychiatry* Aug 1; 46(3), pp420-424.
61. Hornig M (2004). Evidence to the US Institute of Medicine, vaccines and autism. See [www.iom.edu/project.asp?id=4705](http://www.iom.edu/project.asp?id=4705) and see Michael Day, UK Sunday Telegraph 15 February 2004.
62. Stern A H and Smith A E (2003). An assessment of the cord blood:maternal blood methylmercury ratio: Implications for risk assessment. *Environmental Health Perspectives* 111(12), pp1465-1470.
63. Madsen K M, Lauritsen M B, Pedersen C B, Thorsen P, Plesner A M, Andersen P H, Mortensen P B (2003). Thimerosal and the occurrence of autism: negative ecological evidence from Danish population-based data. *Pediatrics*. Sep;112(3 Pt 1), pp604-606.
64. The Immunization Safety Review Committee (ISR) is a project within the Institute of Medicine that addresses current and emerging vaccine-safety concerns. A meeting on 9 February 2004 addressed the issue of vaccines and autism and will publish its findings. See [www.iom.edu/project.asp?id=4705](http://www.iom.edu/project.asp?id=4705)
65. Charman T (2002). The prevalence of autism spectrum disorders. Recent evidence and future challenges. *Eur Child Adolesc Psychiatry*, Dec 11(6), pp249-256.
66. NAS (National Autistic Society) (2002), *Autism in schools: crisis or challenge*. May.
67. Chakrabarti S and Fombonne E (2001). Pervasive developmental disorders in preschool children. *JAMA.*, June 27, 285(24), pp3141-3142.
68. Szatmari P (2003). The causes of autism spectrum disorders. *BMJ* 326 January, pp173-174.
69. World Health Organisation (1998). WHO Press Release WHO/45, 3 June. WHO experts re-evaluate health risks from dioxins. WHO, Geneva.
70. Longnecker M P, Wolff M S, Gladen B C, Brock J W, Grandjean P, Jacobson J L, Korrick S A, Rogan W J, Weisglas-Kuperus N, Hertz-Picciotto I, Ayotte P, Stewart P, Winneke G, Charles M J, Jacobson S W, Dewailly E, Boersma E R, Altshul L M, Heinzow B, Pagano J J, Jensen A A (2003). Comparison of polychlorinated biphenyl levels across studies of human neurodevelopment. *Environmental Health Perspectives*, Jan;111(1), pp65-70.
71. Duarte-Davidson R, Wilson S C, Jones K C (1994). PCBs and other organochlorines in human tissue samples from the Welsh population. *Experimental Pollution*, Vol 84(1), pp69-87.
72. WHO (1996). Levels of PCBs, PCDDs and PCDFs in human milk, World Health Organisation, Denmark.

73. van Leeuwen R and Malisch R (2002). Results of the third round of the WHO – coordinated exposure study on the levels of PCBs, PCDDs and PCDFs in human milk. *Organohalogen Compounds*, Vol 56, pp311-316.
74. Walkowiak J, Wiener JA, Fastabend A, Heinzow B, Kramer U, Schmidt E, Steingruber H J, Wundram S, Winneke G (2001). Environmental exposure to polychlorinated biphenyls and quality of the home environment: effects on psychodevelopment in early childhood. *The Lancet*, Nov 10;358 (9293), p160.
75. Feeley M and Brouwer A (2000). Health risk to infants from exposure to PCBs, PCDDs and PCDFs. *Food Addit Contam.*, 17(4), pp325-333.
76. Harrison N (1998). Pers.comm. letter from Dr Nigel Harrison of the Food Contaminants Division, MAFF dated 30 July 1998.
77. MAFF (1997). Dioxins and polychlorinated biphenyls in foods and human milk. *Food Surveillance Information Sheet*, No 105.
78. Jarbrink K and Knapp M (2001). The economic impact of autism in Britain, *Autism*. Mar;5(1), pp7-22.
79. Landrigan P J, Schechter C B, Lipton J M, Fahs M C, Schwartz J (2002). Environmental pollutants and disease in American children: Estimates of morbidity, mortality, and costs for lead poisoning, asthma, cancer and developmental disabilities. *Environmental Health Perspectives*, 110(7) July, pp721-728.
80. Keating P and Hertzman C (1999). *Developmental health and the wealth of nations*, New York, Guildford Press.
81. Pearce D and Koundouri P (2003). *The social costs of chemicals: The costs and benefits of future chemicals policy in the European Union*, WWF-UK, Godalming.
82. Longnecker M P, Klebanoff M A, Zhou H, Brock J W (2001). Association between maternal serum concentration of the DDT metabolite DDE and preterm and small-for-gestational-age babies at birth. *The Lancet*, Nov 17, 358 (9294), p1732.
83. Hites R A, Foran J A, Carpenter D O, Hamilton M C, Knuth B A and Schwager S J (2004) Global assessment of organic contaminants in farmed salmon. *Science*, 303, pp226-229.







**TAKE ACTION: IF YOU WOULD LIKE TO SUPPORT WWF'S CHEMICALS AND HEALTH CAMPAIGN AND TAKE ACTION FOR A SAFER FUTURE FOR WILDLIFE AND PEOPLE, PLEASE CALL 01483 860869 FOR A CAMPAIGN LEAFLET, OR VISIT [WWW.WWF.ORG.UK/CHEMICALS](http://WWW.WWF.ORG.UK/CHEMICALS)**

## WWF'S CHEMICALS AND HEALTH CAMPAIGN

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

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

## CAMPAIGNING TOGETHER

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



As the largest women's organisation in England and Wales, the National Federation of Women's Institute is working for a safer future for our children and grandchildren.

[www.womens-institute.co.uk](http://www.womens-institute.co.uk)



Through its Customers Who Care campaign, The Co-operative bank is calling for the phase out of persistent and bioaccumulative chemicals.

[www.co-operativebank.co.uk/cwc](http://www.co-operativebank.co.uk/cwc)



The mission of WWF – the global environment network – is to stop the degradation of the planet's natural environment and to build a future in which humans live in harmony with nature, by:

- conserving the world's biological diversity
- ensuring that the use of renewable resources is sustainable
- promoting the reduction of pollution and wasteful consumption

**Taking action for a living planet**

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