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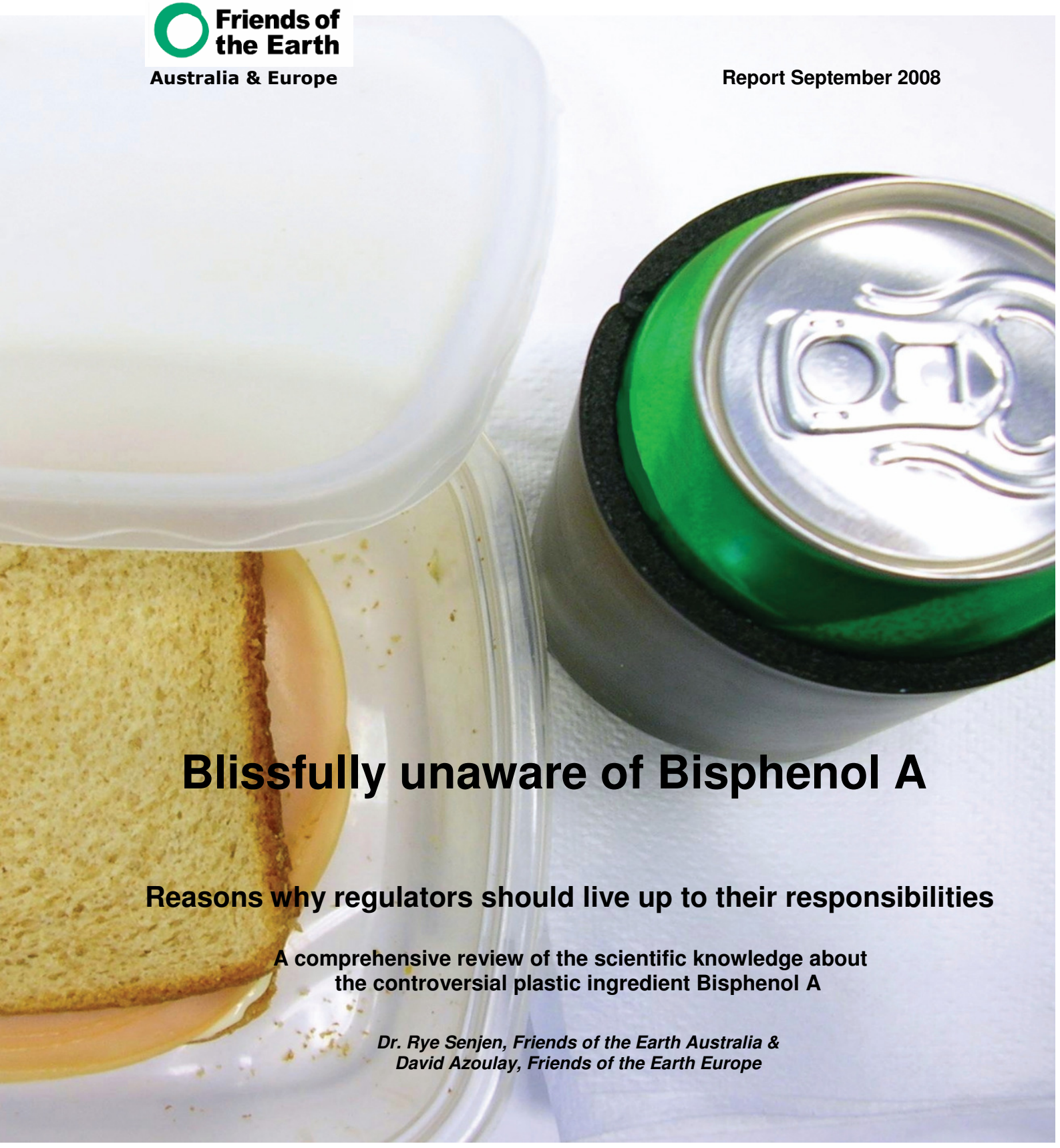

Blissfully unaware of Bisphenol A

Reasons why regulators should live up to their responsibilities

A comprehensive review of the scientific knowledge about the controversial plastic ingredient Bisphenol A

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Friends of the Earth Europe and Friends of the Earth Australia campaign for sustainable and just societies and for the protection of the environment, and are part of the world's largest grassroots environmental network, Friends of the Earth International.

Executive Summary

An ingredient of plastic is linked to many diseases of modern life
The use of plastics has become one of the defining characteristics of modern life. But many of the plastic products people use every day contain components that can prove harmful to human health and the environment.

One such component is a chemical called Bisphenol A BPA). BPA is one of the most widely used synthetic chemicals in the world and is a major component of plastic artifacts. Most of the clear, shatterproof plastics used in **baby bottles, food storage containers, small kitchen appliances and rigid water bottles** include this material. **It is also used in the lining of food, beer and soft drink cans.**

BPA has been known as an Endocrine Disruptor Chemical **(EDC)** since the 1930's and in the past 10 years, BPA exposure has been linked to a surprising number of diseases of modern life. An increasing number of scientific studies have implicated Bisphenol A in illnesses ranging from **infertility, obesity, breast and prostrate cancer,** to **diabetes, thyroid malfunction** and even **attention deficit syndrome.** These disorders have been observed even when exposure to BPA was in extremely low quantities (well below the traditional doses used in traditional toxicology)

Bisphenol A is everywhere and human exposure is continuous
BPA leaches from plastic consumer products are widely evidenced and contamination due to BPA production is considerable. BPA has been measured in freshwater, seawater, landfill sludge, air and dust particles. BPA has also been found to migrate from PVC panels into fresh fruit and vegetable grown in greenhouse conditions and from hoses and water storage tanks into drinking water.

There is broad scientific consensus that human exposure to, and contamination with, BPA is widespread around the world and at much higher levels than expected for a chemical supposed to be metabolized (i.e. broken down) in the human body within six hours. Numerous studies have found BPA in human serum, urine, amniotic fluid, follicular fluid, placental tissue, and umbilical cord blood. **All published surveys found highest concentration of BPA in children, the most sensitive population to BPA induced diseases and health problems.**

BPA regulation is based on flawed assumption and needs to be reviewed
Due to the growing body of scientific evidence and thanks to the continued efforts of civil society, the regulatory landscape for BPA in the US and Canada is gradually beginning to catch up with scientific research. Australia should not be left behind, but take into consideration the general scientific consensus and applying the precautionary principle to BPA.

Australia tends to follow overseas opinions, especially the EU and US regulatory decisions. However, the latest opinion of the European Food and Safety Agency (EFSA) published In early 2007 was as largely based on an industry funded study unpublished at the time, was assessed by a panel composed of food toxicologists, many with industry links and was compromised by a failure to invite expert on BPA or EDCs to provide their assessment.

By 2008 overseas regulatory opinions appear to have caught up with scientific consensus on the dangers of BPA: Canada and the US are conceding that BPA may cause harm and are reviewing regulations. Japanese regulators are reviewing its safety and in the interim are urging mothers to refrain from excessively heating baby bottles and are asking pregnant women to avoid eating too much canned food

Food Standards Australia and New Zealand (FSANZ) has declared that it is monitoring overseas opinion on Bisphenol A. Friends of the Earth Australia (FoEA) urges FZANS to start taking into account the general scientific consensus and to act on the basis of the precautionary principle when reassessing its opinion.

FoE Australia further insists that the overwhelming evidence in relation to the potential harmfulness of BPA even at extremely low dose, be considered. Australia should follow at least the lead of Canada.

It is essential that the latest and widespread scientific consensus be taken into account so that eventually BPA, as well as all endocrine disrupting chemicals (EDCs) are phased out from all consumer products as soon as possible.

Introduction

Bisphenol A (BPA) is one of the most widely used synthetic chemicals in the world and is used mainly in the production of polycarbonate plastics and epoxy resins. BPA can be found in the linings of food cans and lids, polycarbonate plastic water and food containers and shatter resistant baby bottles.

Extensive scientific evidence has identified BPA as an endocrine disrupting chemical (EDC) and implicates BPA in a host of adverse effects on humans and wildlife including developmental toxicity, carcinogenicity, and possibly neurotoxicity (Chapel Hill expert panel consensus statement 2007).

EDCs are chemicals that interfere and disrupt the physiological functioning of the hormonal (endocrine) or messaging system of humans and wildlife (see appendix for a primer on EDCs). The endocrine system is a complex network of glands, hormones and receptors that carefully regulates many bodily functions, including metabolism, immunity, behaviour and growth and development during childhood. For instance, the European Union has already identified over 200 EDCs available on the European market (Environment Directorate-General of the European Commission 2008).

Scientific consensus around the risks of EDCs has been building since 1991 with the Wingspread declaration that agreed on a connection between chemically-induced alterations in sexual development in wildlife and humans and culminating in late 2007 in the Chapel Hill expert consensus statement. The statement was delivered by 38 leading scientists in the field of EDCs and warned policymakers of potential adverse health effects of widespread exposure to BPA.

Unfortunately this expert consensus does not seem to have made its way to the Australian institutions yet.

1. What is Bisphenol A?

1.1. Bisphenol A is a key ingredient in making plastics

Bisphenol A (BPA, 2,2-bis(4-hydroxyphenyl) propane; CAS no. 80-05-7) is one of the most commonly used industrial chemicals in the world today. BPA is a key ingredient in the production of plastic polycarbonate materials. Making them strong and shatter proof, resistant to temperatures between 40 and 145 degrees Celsius, and resistant to many acids and oils. It is also an ingredient in epoxy resins, which are tough, resistant to many chemicals and adhere well to numerous surfaces. In addition, BPA is also used in a variety of minor applications, such as brake fluids, pesticides and polymerisation inhibitor and antioxidant in PVC (refer to Table 1 for a sample list of consumer products containing BPA). BPA began to be used in the production of polycarbonates in 1953. Potential environmental sources of BPA include contamination due to losses during production, leaching from landfill and consumer products, and presence in indoor air (Chapel Hill expert panel consensus statement 2007).

1.2. BPA production exceeds 3 million tonnes every year

In 2003 about 3 million tonnes of BPA were produced annually, ranking it among the highest-volume chemicals manufactured worldwide. Production of Bisphenol A is rising by about 6 - 7% per year (Market Publishers 2007). Output was predicted to reach over 4 million tons in 2006, and could be over 7 million tonnes by 2015 (China Chemical Industry News 2005). About a third of the worldwide annual production of BPA is used in the EU (Bro-Rasmussen 2006), with one factory in southern

Spain (GE Plastics) producing over 250,000 tonnes/year alone (Fernandez et al. 2007).

Most of the BPA produced is used in the manufacture of polycarbonate plastics (65% of global demand in 2001), with the remainder used in the production of epoxy resins (30%). Some BPA is used in the production of flame retardants, unsaturated polyester resins and polyacrylate, polyetherimide and polysulphone resins and other applications (ICIS 2007).

1.3. Major chemical companies are involved in BPA production worldwide

The main producers of BPA are Mitsubishi, Sunoco, Dow, Bayer and GE Plastics (CBGnetwork 2007, Bisphenol-A.org 2007, Sunoco 2008), but other chemical companies such as BASF, also produce significant quantities of the substance. BPA is a vital input ingredient for the production of polycarbonate plastics, and polycarbonate manufacture is big business. The Bayer Material Science Polycarbonate business unit had annual revenues of about 2.5 billion Euros in 2006 (Babe 2007). GE Plastics was acquired mid 2007 by SABIC (Saudi Arabian Basic Industry Corporation) for 11.6 billion $US. SABIC, a Saudi Arabian company is one of the top ten petrochemical companies and produces and sells the raw materials for the production of many oil based products, including basic chemicals, polymers, fertilisers and metals (Saudi Commerce and Economic Review 2007).

Table 1: Examples of consumer products containing Bisphenol A

Polycarbonate Plastics (65% of use)	Epoxy Resins (30% of use)	Other Uses (5% of use)
Impact-resistant glazing	Coatings	Pesticide formulations
Street-light globes	Food and beverage can linings	Antioxidant
Household appliance parts	Electrical laminates for printed circuit boards	Flame retardant
Components of electrical/electronic devices	Composites	Brake fluid
Compact discs	Adhesives	Rubber and PVC stabiliser
Automotive applications	Paints	Water supply pipes
Reusable bottles	Nail polish	Dental sealant.
Food and drink containers		Thermal paper additive
Sunglasses		Water main filters
Refrigerator shelving		Reinforced pipes floorings
Microwave ovenware		Electric insulators
Eating utensils		

Sources: Bro-Rasmussen 2006, Weise and Szabo 2008, Endocrine/Estrogen Letter 2003

Table 2: polycarbonate- producers, products and factory locations

Producer	Marketshare	Factory Location	Main Product
Bayer	**32%**	US (Sheffield, Pittburgh, Berlin, Newark, Baytown), Europe (Antwerp, Uerdingen, Domagen, Leverkusen, Filago) and Asia (Cuddalore, Map Ta Phut, Caojing and Hong Kong)	MAKROLON
GE Plastics (SABIC)	**29%**	Freeport (Texas, USA) and Stade (Germany) and Southern Spain	LEXAN®
Mitsubishi	**12%**	Japan, New Jersey (USA), China, Thailand	Lupilon, Novarex
Teijin	**11%**	Japan, Singapore, China, Japan, Singapore	PANLITE
Dow Chemicals	**9**	Mt. Vernon (Indiana; USA) Cartagena (Spain); and Bergen op Zoom (The Netherlands	CALIBRE® , PARABIS®

Sources: Babe 2007, Dow 2007, Sabic 2007, Teijin Chemicals 2007, Mitsubishi 2007

2. Documented adverse effects of BPA

2.1. BPA is a powerful endocrine disrupting chemical

BPA was recognised as early as the 1930s for its endocrine mimicking effects, well before it was used in industrial applications in the 1950s (Dodds and Lawson 1936). Since then, BPA has been implicated in human, mice and rat studies as a powerful endocrine disruptor.

For a long time BPA has been considered to be only a weak environmental oestrogen, but recent and repeated studies of molecular mechanisms of BPA action have shown that BPA can operate at very low concentrations in a variety of tissues (Vandenberg et al. 2007).

2.2. Endocrine toxicology is different from traditional toxicology

The problem with much of the research into endocrine disrupting chemicals (EDCs) is that it turns traditional toxicological thinking on its head. Toxicology works on the assumption that a threshold exists, below which a chemical has no effect on the body (the No Observable Adverse Effect Level **(NOAEL)**.

This idea comes from the belief that below this threshold the body's defence mechanisms are able to deal with the chemical. It is also assumed that as the dose increases so does the response – this is also known as a monotonic dose-response relationship or linear response.

However in the case of the endocrine system this assumption does not appear to hold. The endocrine system is a signaling system operating at all time, which is regulated at a number of different levels. When an endocrine disruptor gets absorbed by the body it interferes with this signaling system. It has been shown again and again that the disruption of the hormonal system by EDCs occurs at doses much lower then the NOAEL.

Furthermore, there is an ever-growing body of scientific research that shows that the relationship between dose and response can be a non-linear relationship. For example you may get a response at a very low dose, no response at a medium dose and again a response at a high dose.

To complicate things further, the type of interference can change with the amount of chemical that is added to the system. The timing and length of exposure also appear to affect the response. Additionally when several of these chemicals are mixed together, mixtures can interact additively or synergistically at concentrations that individually are insufficient to cause observable effects (Brian et al. 2005, Rajapakse et al. 2002). In case of estrogenic chemicals especially, it has been noted that "hazard assessments that ignore the possibility of joined action of estrogenic chemicals will almost certainly lead to significant underestimations of risk" (Silva et al. 2002).

2.3. The "low dose" issue

The "low dose" effect of BPA has now been well established. **By the end of 2006 149 out of 176 (93%) peer reviewed scientific studies showed that low doses of BPA can cause adverse effects.** Out of 27 studies which showed no adverse effects 13 were industry funded, while the rest used rats that were unsuitable because of their insensitivity to estrogenic chemical, including BPA (vom Saal 2006). By the end of 2007 a further 19 peer reviewed laboratory studies on BPA "low dose" effects were published, all showed harmful effects (Senjen 2008). For example, one study showed that a low dose of BPA (relevant to human exposure) produced a 70% higher growth rate in prostate cancer cells than a 100 time higher doses (Wetherill et al 2002).

Traditional toxicology also assumes that the relevant safety standard should apply to adults, however numerous studies have shown that BPA exposure in the womb or during early childhood may be the most damaging. For instance, one study showed that in-utero exposure of mice to an environmentally relevant dose of 25 µg/kg body weight resulted in a 70% higher growth rate in breast cancer

cells than a 10 time higher doses of 250 µg/kg body weight of an adult (Markey et al. 2001).

What is a low dose effect?
In the last ten years, many different experiments, both in vivo and in vitro, have shown that the adverse effect of EDCs occur at much lower doses than traditionally assumed. This has become known as the "low dose effect". Industry and some government still dispute the reality of the "low dose" effect, but there is increasing scientific evidence supporting its validity. In the context of laboratory animal studies "low doses" means the administration of doses below those used in traditional toxicological studies conducted for risk assessment purposes. These are any doses below the so-called Lowest Adverse Effect Level (LOEAL).In the case of BPA the lowest dose examined for traditional toxicological risk assessment was 50mg per kg /per day, established in the 1980s (Wetherill 2007).

How the Lowest Adverse Effect Level (LOEAL) is used to calculate the EU reference dose

The LOEAL is still used as the basis for calculating the current US and EU reference or No Observable Adverse Effect Level **(NOEAL)** dose. This dose is considered safe for humans to ingest on a daily basis and is typically 1000 times smaller than the LOAEL. In the case of BPA it is 50µg/per kg/per day (Chapel Hill Bisphenol A expert panel consensus statement 2007).

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2.4. BPA is a known and proven endocrine disruptor

There is ample evidence to attest that BPA binds selectively to endocrine receptors. Recent research has shown that BPA can also alter the ability of the body to make and metabolise hormones and alter hormone concentrations in the blood. Additionally BPA changes tissue enzymes and hormone receptors, and interacts with a variety of hormone response systems (for a review see Richter et al 2007). Furthermore, recent research has shown that BPA can stimulate the (only recently discovered) oestrogen receptors in the cell membrane at incredibly low concentrations, e.g. parts per trillion (for example see: Quesada et al. 2005, Walsh et al. 2005, Wozniak et al. 2005, Zsarnovszky et al. 2005).

2.5. BPA is not just an oestrogen inhibitor

The BPA estrogenic effect is well documented, however BPA's effects are not just limited to the inhibition, enhancement mimicry of endogenous estrogen and/or disruption of estrogen receptor action. BPA also has a number of other effects, including: effects on the androgen systems (which regulates the growth, development, and function of the male reproductive system), disruption of thyroid hormone function, diverse influences on development, differentiation and function of the central nervous system and potentially adverse influences on the immune system. Furthermore, the bioavailability or expression of endogenous steroid hormones may be limited and modified by BPA exposure. Recent

research has also shown that early interference and exposure to this chemical can transmit and express itself later in life and across generations (as reviewed in Wetherill et al. 2007, see also below § 2.7.)

2.6. BPA may cause cancer

According to a 2007 scientific review on the cancer causing potential of BPA by Keri et al. (2007) and published in Reproductive Toxicology, BPA exposure has been associated in animal studies with increased cancer of the haematopoietic system (e.g. marrow, spleen, tonsils, and lymph nodes), and a significant increase in interstitial cell tumours of the prostate. Additionally, animal studies have also shown that early life exposure to BPA increases the risk of breast and prostate cancer (Soto et al 2008). The weight of evidence points towards BPA increasing cancer susceptibility through developmental reprogramming when exposure occurs during foetal or early childhood development. Furthermore, a Spanish study (Fernandez et al. 2007) published in late 2007 investigated the level of BPA and its chlorinated derivatives in adipose tissue of women. BPA was above the limit of detection (LOD) in 11 out of 20 samples (55%).This is the first report of BPA being found in adipose tissue in humans.

2.7. BPA can alter how genes are expressed

When mice were fed BPA before, during and after pregnancy, their resulting offspring had yellow instead of brown coats and were obese. This is particularly significant as obesity may result in a higher susceptibility to cancer and diabetes (Dolinoy et al. 2007). This study is part of a growing body of scientific evidence that investigates how certain factors such as hormones or environmental factors can alter how genes are expressed (i.e. turned on or off) and how this can lead to an increased risk of disease (the field of study known as epigenetics). It has now become undisputable that environmental factors such as diet, life experiences and exposure to certain synthetic chemicals such as BPA can influence gene expression. This new study provides concrete evidence that BPA can alter how genes are expressed, by removing the protective molecules that normally prevent genes from being turned on at the wrong time or in the wrong tissue. It also shows that certain periods during pregnancy may be more 'harmful' for the foetus.

2.8. Low doses of BPA may affect your grandchildren adversely

A study on pregnant mice published in early 2007 suggests that "low dose" BPA exposure affects maturing eggs and additionally continues to affect the offspring produced from these eggs (Susiarjo et al. 2007). The study found that the estrogenic effect occurs at a much earlier stage of egg development than previously thought and resulted in an abnormal number of chromosomes in the eggs. The study also uncovered a multigenerational effect: when exposed fetuses reached adulthood, they contained a significantly higher number of chromosomally abnormal eggs and embryos. To put it another way: "low-dose" BPA exposure during early pregnancy could result in an increased number of chromosomally abnormal grandchildren.

To put it into the words of one of the authors of the study: "In the course of studies to assess the effects of BPA on the mouse oocyte, we have uncovered a novel ''grandmaternal'' effect: "low dose" exposure to BPA during pregnancy disturbs oocyte development in unborn female foetuses. When these foetuses reach adulthood, the perturbations are translated into an increase in chromosomally abnormal eggs and embryos. Thus, "low-dose" BPA exposure during pregnancy has multigenerational consequences; it increases the likelihood of chromosomally abnormal grandchildren." (Susiarjo et al. 2007). But do these results translate into worrying consequences for humans? Interestingly an earlier study has already made an association between serum BPA levels and recurrent miscarriages in humans (Sugiura-Ogasawara et al. 2005). The additional 2007 study provides more concrete evidence.

The experiments clearly show that environmental exposure to chemicals can affect the process of cell division in mammals. But it also shows that key health issues may only become apparent after two successive generations. This presents problems for decision-making and regulating bodies. A study based on currently accepted statistical and scientific principals, such as sufficient and representative numbers of test subjects, would

require assessing a large representative sample of human females of reproductive age (perhaps 2000 of them), as well as assessing their female children, and subsequently their grandchildren (Hawley and Warburton 2007). Apart from the obvious ethical implications, the process of collecting the data would prove very onerous. Given the wide acceptance of the precautionary principle, it does appear more sensible in the case of BPA to actually apply it, rather than wait for another 30 years to confirm what is already implicated from many hundreds peer reviewed studies.

3. How are humans exposed to BPA

3.1. Introduction

It is now obvious and indisputable that BPA can have adverse effects on human health even at low doses. But how exactly does BPA transfer from all of our consumer goods (see Table 1) to our body systems? First of all, the potential overall environmental contamination due to BPA production is considerable and largely unacknowledged. BPA has been measured in freshwater, seawater, landfill leachates, air, and dust particles. Total emissions of BPA in Europe in 1999 were estimated at 2.1 tonnes into the air, 199 tons into water and 30 tonnes into soil (Directoraat- Generaal Rijkswaterstaat. Ministerie vanVerkeer enWaterstaat 2001). As the production of BPA has since doubled one should assume that emissions have done the same. However, these figures are hardly sufficient to account for the BPA levels found in our bloodstream and overall system, by all available surveys. Again, we have to turn to the extensive sets of evidence showing that BPA is leached from countless consumer products, food contact materials and is quite massively released into the environment during its production.

3.2. Food packaging is one of the major sources of BPA exposure for humans

According to available studies, the amount of leaching of BPA from food packaging is related to the type of food or liquid, temperature and heating time. Leaching rates under normal conditions of use have been measured for food containers and bottles, epoxy resins (can coatings), baby bottles, take-away food containers and plastic wraps (see table 3). Leachates into food products have been detected in vegetables, fish, fruit (including fresh), instant coffee, powdered milk and baby formula, milk (all canned) as well as honey.

For instance, a 2008 study tested the amount of BPA released from polycarbonate bottles used to store water and other beverages for consumption. The chemical was found to migrate from polycarbonate water bottles, irrespective of whether or not the bottle had been previously used. When filled with boiling water the rate of BPA migration from the bottle into the water increased by 15 to 55-fold. Migration also increased over time and after 7 days the concentration of BPA amounted to 250ng per standard cup of water. While by itself the actual amount leached is not large, remember that, as an endocrine disruptor, BPA starts having adverse effects and interact with other EDCs at extremely low doses. Furthermore, although amounts are small, BPA leachates undoubtedly contribute to the total "EDC-burden" to which most consumers are exposed (Le et al. 2008).

3.3. BPA contamination of drinking water is widespread

BPA has also been shown to migrate from PVC hoses and water storage tanks, further contributing to contamination of drinking water. The migration rate of BPA into water may furthermore be exacerbated by residual chlorine in the water (Fernandez 2007). BPA has also been detected in many rivers in Europe. A 2001 study investigating BPA levels in water found levels ranging up to 16ng/l in river samples and 2ng/l in drinking water (Kuchand and Ballschmitter 2001).

3.4. BPA can even be found in fresh food

Another unexpected source of BPA may be fresh fruit and vegetable grown under green house conditions. A 2007 Japanese study reported BPA in fresh strawberries and a 2003 Italian study found 250-1000 ng/g of BPA in 8 out of 14 fresh vegetables (Vivacqua et al. 2003).The most likely source of BPA in the case of fresh fruit and vegetables are PVC panels used for the walls of greenhouses (however this has not been positively confirmed), with BPA migrating into the fruit and vegetables via the atmosphere (Sajiki et al. 2007). The amount of BPA found in fresh food was twice as high as that found migrating from cardboard take-away containers and in the same range as potential migration levels from microwaving polycarbonate containers. What is significant is the indication that even fresh food that had no direct contact with BPA may still contain it. This again points to the fact that there may be a number of 'unexpected' and as yet unrecognised sources that contribute to our overall BPA load.

3.5. Ink/toner and thermographic printing products contain BPA

Printing ink, toners and thermographic printing products may all contain BPA (Danish EPA 2007). During the process of recycling, waste paper is frequently bleached with sodium hypochlorite which may lead to formation of chlorinated BPA derivatives. These derivatives have been found to be 28 times more estrogenic than the non-chlorinated Bisphenol A products (Fukazawa et al. 2002, as cited by Danish EPA 2007). BPA is also used in the production of "direct thermal transfer printing" which produces low resolution and relatively low permanence printing results such as airline, event and cinema tickets, online lottery and gaming tickets, labels and point of sale applications such as checkout receipts. Toners are used commonly in copying and non-impact printing processes, such as office copiers, plain paper fax machines, digital printers and copiers. Various manufacturers (Xerox, Lexmark) use BPA derivatives in these toners, for example, in the form of Bisphenol A polyester resin. Printing inks are applied as thin films on paper, paper board, metal sheets and metallic foil, plastic films and moulded plastic articles, textiles and glass etc. Some may not contain BPA, however many others do (Danish EPA 2007).

3.6. BPA is also found in many dental products

Leaching of BPA from dental products has also been well demonstrated. BPA is used to create resin-based preventive sealants, adhesives and restorative materials (Vandenberg et al. 2007). Research published in 2006 has shown that BPA exposure from dental sealants is detectable and measurable in both saliva and urine of exposed individuals following initial application. Again, the levels detected after application of the sealant, have been shown to produce adverse oestrogen-mediated effects in rodents (Joskow et al. 2006).

3.7. Current levels of BPA in adult and children show harmful effects

Taking into account animal models and the greater rate of BPA clearance from the body in humans versus rodents, a recent review paper on human exposure to BPA (Vandenberg et al. 2007) argues that current human exposure levels are likely to cause adverse effects on human cell and organ functions, because:

- "Humans are exposed to BPA at a much higher level than has been estimated from known exposure sources; and/or
- humans are exposed through multiple routes, making the metabolic response different from that observed in animal models; and/or
- metabolism of BPA following chronic, low-dose exposure is not predicted by the acute high-dose studies used to generate the current pharmacokinetic models.
- many adverse responses have been observed in human and animal cells at and below concentrations of 0.23 ng/ml, which is the median human blood levels of unconjugated BPA (e.g. not metabolised and thus biologically active)".

It is now undisputable that human exposure to BPA is worldwide and widespread. Numerous surveys have measured BPA levels in human serum, urine, amniotic fluid, follicular fluid, placental tissue, and umbilical cord blood (Vandenberg et al. 2007). Most foetuses, children and adults in the developed world will record about 0.3–4.4 ppb- (parts per billion or 0.3 - 4.4 ng/ml) of BPA in tissues and fluids (Chapel Hill expert panel consensus statement 2007).

A 2008 study investigated the BPA levels of women trying to conceive. The BPA excretion levels of the 10 women who did become pregnant increased by 33 percent during pregnancy. This may be due to the changes brought about by pregnancy which affect a woman's ability to metabolise, distribute and/or clear BPA from the body. While the number of participants is too small to be statistically significant, the data may indicate that the foetus is exposed to much higher concentrations of BPA than previously thought. Additionally timing of exposure is also critical.

Several studies have shown that the foetus is thought to be most at risk when exposed to BPA (Mahalingaiah et al. 2008, Dolinoy et al. 2007 study). The inability of newborn mice to adequately deal with exposure to BPA was confirmed by a 2008 study. This study reported that when newborn mice and adult mice were exposed to BPA, the newborns had significantly higher levels of BPA in their blood. The reason for this may be the substantially lower levels of the enzymes needed to breakdown BPA. Preliminary data indicate that human infants

also have lower levels of these enzymes when compared to adult humans. This adds further evidence to the maxim in pediatrics that "babies are not little adults" and that regulators need to take into account the possibility that chemicals have an increased adverse impact on the health of foetuses, infants and children (Taylor et al. 2008). Newborns may also be exposed to BPA via breast milk. Three independent peer reviewed studies found levels of up to 0.97 ng/ml (as cited by Vandenberg et al. 2007). This means that a newborn baby could be exposed to approximately up to 1000 ng or 1 µg per litre of breast milk consumed.
A study published in October 2007, that examined the levels of BPA in urine in a representative sample of the US population (over 2500 participants sampled between 2004 and 2006) showed that 92.6% of the US population had detectable levels of BPA in their bodies, with total concentrations ranging from 0.4 µg/l to 149 µg/l. The highest concentration of BPA was found in children, followed by adolescents, adult females and finally males (Calafat et al. 2007). This confirmed an earlier study conducted in the US in 2000 (Weise and Szabo 2007) and indicates that humans are continually exposed to BPA, despite BPA not being persistent i.e.: it is metabolised or "broken down" in a human body within 6 hours (Vandenberg et al. 2007).

Case Study: Could BPA in cardboard take-way containers cause cancer?

A new study raises concerns about synergic effects of chemicals in cardboard containers used for food containers. The 2007 study confirmed a new source of BPA exposure: pizza boxes, potato chip containers and paper bags for take-away sandwiches (Lopez-Espinoza et al. 2007). The study investigated cardboard containers collected from four EU countries: Belgium, Italy, Portugal, and Spain. When the cardboard was subjected to aqueous extraction, 90% of the obtained solution induced human breast cancer cells to grow in culture. The aqueous extraction contained both BPA and the phthalates DBP and DEHP (used as plasticisers).

This is not the first time BPA has been implicated in the induction of breast cancer cell growth. Several other scientific studies have also reported estrogenic activity of low concentrations of BPA in MCF-7 breast cancer cells (these cells are used as a model for the study of human breast cancer). The source of the BPA in the paper was initially puzzling. However BPA is frequently used in the production of printer inks, and waste paper from offices is a commonly used in the production of recycled paper. Nine out of ten of the card board take-away containers contained recycled paper (often not labeled as such).

There was no direct causal link established between BPA and the carcinogenic effects of cardboard containers; instead the investigators suspected that BPA, perhaps in synergy with the phtalates, produced the cancer inducing effect. Exposure to phthalates has been implicated in many health problems e.g. early puberty in girls, premature delivery, poor sperm quality and infertility in men, genital birth defects and reduced testosterone production in boys, to name a few. The European Union and many other countries have restricted the use of phthalates in children's toys and cosmetics.

Table 3: major sources of BPA dietary and food contact exposure

Sources of exposure	Level found in Product	Reference	Comment
Baby bottles	▪ 9.6 ng/ml leaching level ▪ 2560 ng/in^2 product level	Brede et al. (2003) Wong et al (2005)	Exposure increased significantly after repeated use
Polycarbonate plastic bottle	1 ng/ml after 7 days new bottle, ambient water 0.7ng/ml after 7 days used bottle , ambient water 3.84 -7.67ng/ml after heating new bottle, 1.92 ng/ml after heating, used bottle	Le et al. (2008)	55 fold increase when filled with boiling water
Microwave plastic containers	30 µ/g in product, potential leaching level 6.5 µ/g of food	Nerin et al. (2003)	Leaching increased with heating of containers
poly vinylidine dichloride plastic wraps	Up to 483 mg/kg film 30.7 µg/dm^2 leaching level	Lopez-Cervantes et al. (2003)	Leaching observed when in contact with water, olive oil, acetic acid
Card board for take-away food	BPA detected in 45% of samples Average of 115 ng/g of cardboard	Lopez-Espinosa et al. (2007)	40 containers from 4 EU countries (Belgium, Italy, Portugal, and Spain)
Paper towels from recycled paper	24.1 µ/g in product	Vingaard et al. (2000) Ozaki et al. (2004)	Virgin paper contained significantly less BPA
Polycarbonate plastic tubing	4.8 ng/ml leaching level	Sajiki et al (2003, 2004)	Leaching levels greatest in river water
Canned food lining	Up to 102 ng/ml leaching level in tuna fish and other fatty foods	13 studies in total, Mungula-Lopez et al (2006)	Including vegetables, fish, fruit, instant coffee, powdered milk and baby formula milk
Fresh food	2 ng/g fresh strawberries 250-1000 ng/g in fresh vegetables	Sajiki et al. (2007) Vivacqua et al. (2003)	From PVC in glass house panels via air?

Sources as cited by Vandenberg et al. 2007, Lopez-Espinoza et al. 2007, Le et al. 2008.

3.8. Levels of BPA observed are higher than expected

Considering the numerous documented sources of BPA exposure and the fact that BPA is not biopersistent, there appears to be a discrepancy between the known sources of human exposure to BPA and the much higher levels measured in human tissues and fluids (Vandenberg et al. 2007). While each exposure source may only contribute a relatively small amount in itself, exposure is clearly widespread and occurs through many different routes.

A number of studies have shown that air and dust are a further source of exposure to BPA. For instance, 86% of homes surveyed in the US contained BPA in the air, ranging from 0.2 to 17.6µg/g (Rudel et al. 2003). Studies have estimated that human exposure ranges from less the 1µg/kg/day to almost 5 µg/kg/day or 0.325 mg/day/adult on average (Vandenberg et al. 2007). A study published in 2007, which investigated the exposure to BPA of 257 pre-school children in two US states to BPA found that 50% of indoor air, surfaces and hand wipes, 83% of solid and 68% liquid foods contained BPA. Potential total exposure levels to BPA were up to 1.570 µg /kg/day per child (Wilson et al. 2007). This constant and continuous exposure accounts for the BPA levels found in our bodies by all available bio monitoring surveys,

Little research exists to explain what effect this continuous low level exposure to BPA may have on the general population and the environment. Takeuchi et al. (2004) describes, for example, a relationship between elevated BPA blood levels and polycystic ovary disease

(PCOS) in Japanese women, and in 2005 Sugiura-Ogasawara et al. reported a relationship between blood levels of BPA and recurrent miscarriage also in Japanese women. Vom Saal and Hughes (2006) points out that the findings from these studies are consistent with studies that show harm from BPA to animals at BPA blood levels within or below those detected in human blood.

However, in this context, the 2007 Chapel Hill expert consensus conference that brought together 38 leading BPA researchers is confident that given existing data:

- "Human exposure to BPA is variable, and exposure levels covers over a broad range in tissues and fluids in foetuses, children and adults.
- Human exposure is likely to be continuous, unlike exposure in most laboratory animal studies of BPA pharmacokinetics.
- The commonly reported **circulating levels in humans exceed the circulating levels extrapolated from acute exposure studies in laboratory animals.**
- **BPA levels in the foetal mouse exposed to BPA by maternal delivery of 25 mg kg−1, a dose that has produced adverse effects in multiple experiments, are well within the range of unconjugated BPA levels observed in human fetal blood"** (Chapel Hill expert panel consensus statement 2007).

Scientists broadly agree: endocrine disrupting chemicals are dangerous
Following a review of over a hundred peer reviewed scientific studies, it appears that there is an undisputed consensus on the following (see also appendix 1):

- Exposure to EDCs is ubiquitous and worldwide.
- Many synthetic chemicals (including pesticides) in widespread use are being identified as EDCs.
- Low dose exposure to EDCs may have a much greater and/or different effect than higher doses (low-dose effect), turning conventional toxicological wisdom on its head.
- All chemically mediated messaging systems in the body are liable to be disrupted by EDCs, causing numerous adverse affects.
- There are many serious human health impacts including negative effects on adults, foetuses, as well as intergenerational effects.

To summarise these conclusions by leading BPA experts: BPA levels found in human blood are universal and at a level that have produced adverse effects in laboratory animals.

The Chapel Hill expert panel consensus stated that
"It is essential for the precautionary principle to be applied because scientific certainty will be difficult to establish due to the complexity of the endocrine/messaging system and the wide ranging effects of EDCs. Scientific certainty is clouded by bias towards false negatives, industry influence, and the impossibility to find non-contaminated research subjects and environments" (Chapel Hill expert panel consensus statement 2007).

Is the EU tolerable intake level for BPA exceeded on a daily basis?
Recent scientific results indicate an intake of up to 100 mg/day/adult of BPA (Vandenberg et al. 2007). This implies that the daily intake of the average person (assume body weight of 70kg) is approximately 30 times higher than the newly established acceptable European Bisphenol A Tolerable Daily Intake (TDI -an estimate of the amount of a substance that can be ingested daily without risk) of 0.05 milligram/kg body weight It is furthermore important to note that this BPA TDI already represents a five fold increase from the previous assessment made in 2002 and is solely based on a at the time not peer reviewed industry funded study

4. The way forward

4.1. Will the Australia be left behind in precautionary BPA regulation?
Despite strident efforts by the plastic and chemical industry the regulatory landscape for BPA is finally catching up with scientific research. In the USA, a 2008 draft report by the National Toxicology Program (part of NIEH) acknowledged for the first time "some concern" that BPA may affect neural and behavioural development "in foetuses, infants, and children at current human exposures." Concerns were also expressed about the risk of cancer, diabetes and other serious health problems in adults, while early puberty in girls and hyperactivity were some of the acknowledged possible "developmental disturbances". (NTP 2008). This was the first time a US government agency has expressed any concern about possible health risks associated with BPA. Interestingly this report considered many studies rejected by an earlier panel and additionally reviewed more than 400 studies published between April 2007 and February 2008 (Layton 2008).

In April 2008 Canada Health released its draft report on the impacts of BPA with a focus on newborns and infants up to 18 months of age. The report concluded that that the gap between exposure and effect of BPA on the under 18 month age group is not large enough to be considered safe. As a result the government of Canada intends to ban polycarbonate baby bottles and to develop stringent migration targets for BPA in infant formula cans. Additionally the report also noted that BPA at low levels can harm fish and aquatic organisms over time and that it is found in wastewater and sludge treatment plants (Health Canada 2008).

"When it comes to Canada's environment, you can't put a price on safety," said Minister Baird. "Not only are we finding out about the health impacts of Bisphenol A, but the environmental impacts as well. That's why our Government will be moving forward and will work with the provinces and stakeholders to keep Bisphenol A out of our environment, and take the necessary measures to ensure its safe use and disposal." (Source: Health Canada 2008).

This is the first time in the history of BPA that any government has seriously considered banning products containing this chemical. Many retailers in North America (including Wal Mart and major Canadian and outdoor retailers) have not waited for the final government regulation and have started to remove polycarbonate baby and water bottles from their shelves (Austin 2008).

In July 2008 the Japanese health ministry instructed the Food Safety Commission to assess the safety of bisphenol A. with particular reference to children's health. The review is expected to take at least one year and in the interim the Japanese ministry has urged mothers to refrain from excessively heating baby bottles and pregnant women to avoid eating too much canned (Jiji Press English News Service 2008).

Australia should not be left behind by failing to take into consideration the general scientific consensus and applying the precautionary principle to BPA.

4.2. BPA regulation is outdated and heavily influenced by industry
Judging by the reluctance of various government agencies in the EU to take the necessary step towards eliminating BPA at least from food contact materials and then slowly from all products, the chemical and plastics industry is continuing to disseminate and financially support misinformation, apply pressure on government agencies and scientific panels and populate scientific panels with people that share their misguided opinions.

The case of BPA is rather reminiscent of the tobacco industry campaign that aimed to deny the health hazards of smoking. Conflict of interest associated with scientific research has been well and extensively documented (Sass 2006, Hayes 2004, Barrow and Conrad 2006). Manufacturing doubt is one of the methods used by industry to advance their economic and political causes (Ong and Glatz 2001). The most recent and relevant examples being the line of argument that chemically induced animal tumours are not relevant to human risk assessment (Melnick et al. 2007). In the specific case of BPA the tactic appears to have

been to deny, delay and/or dismiss research on low dose effects, primarily by conducting industry studies that somehow were unable to replicate “low dose” effects.

As numerous independent “low dose” studies found effects on hormone sensitive tissues and systems below safety standards, the industry begun to argue that the reported results did not apply to humans, due to the different physiological characteristics of humans and animals. The overall effect has been an industry-led effort to determine what constitutes legitimate, relevant, and reliable scientific research and to delay proper regulation of dangerous chemical substances for as long as possible (Vogel 2008.

For instance, a study conducted by Rochelle Tyl (Tyl 2002), from the US Triangle Institute of North Carolina found no reproductive or developmental effect after 8000 Sprague-Dawley rats were fed a diet containing a variety of levels of BPA (from very low to very high). But there were serious questions regarding the validity of this study. Most damming perhaps, the strain of rats chosen by Tyl were naturally unresponsive to BPA. Additionally no positive control was used, which would have confirmed whether the animals were able to respond sensitively to a test of reproduction and development (Hillman 2003). The study was financed by the plastics industry.

Another study financed by the plastics industry and again conducted by Rochelle Tyl, apparently formed the cornerstone to the early 2007 EFSA decision to increase the lower limit of BPA fivefold. This study was finally available in May 2008 (Tyl et al. 2008).It apparently found no negative effects over two generations of mice when they were feed BPA. At the time of the EFSA review in 2006 this study was unpublished and had not been subjected to peer review by other independent scientists (as is a common practice in order to test the validity of results and the experimental design). It should have never formed the basis of the EFSA review, as at this point in time it was impossible to assess the validity of any claims and if the experiment were conducted in an unbiased manner (Roegner 2007).

4.3. BPA and the European Food and Safety Agency

The European Food Safety Authority (EFSA) AFC Panel (food additives, flavourings, processing aids and materials in contact with food) released its opinion on dietary exposure to BPA in early 2007. The purpose of the opinion was to evaluate the effects of BPA on reproduction and the endocrine system in relation to food contact materials. It is of some interest to note:

- An industry funded study, unpublished at the time of the EFSA review was used as the major source to come to the panel’s decision on the ‘safety’ of BPA (Roegner 2007).
- The panel failed to invite experts on “low dose” BPA effects or endocrine disruptors to provide their opinion.
- The panel was almost entirely composed of (food) toxicologists, several with industry links, including the plastics industry and an industry funded NGO (see appendix 5 for a listing of members and their questionable links to industry).

Considering al the facts detailed in this report and given the inherent flaws in the methodology and thinking that has led to this opinion, it is not really a surprise to note that the EFSA opinion, in stark contrast to the most recent and broad scientific consensus, actually recommended raising the acceptable BPA levels in this last opinion. The previous opinion (published in 2002) had set the level of acceptable TDI at 0.01 mg/kg body weight. The new ruling amounts to a five fold increase.

It appears that the EU scientific assessment process has so far lacked proper governance, transparency and guidelines and may be unduly influenced by industry interest.

5. Life after BPA

5.1 Encouraging alternatives

BPA is used in hundreds of consumer products and phasing it out will not be an easy task. As a first step it is essential that the use of polycarbonate plastics in all food contact materials (e.g. can coatings, lids of glass containers, polycarbonate bottles), as well as materials that come in contact with water, be banned immediately. But what are the alternatives?

Currently 90% of all cans are coated with epoxy resins containing BPA. Polyester and polyamides are obvious alternatives, and they may possibly be less harmful. However, other plastics alternatives such as polyacrylate and polymerised rosin (a solid resin obtained from pine trees) alternatives may be even more hazardous. There are currently several barriers preventing polyester coatings from being used more widely, such as limited chemical resistance, shorter shelf life and adherence, they are also more expensive. Polyester-based coatings are already in use for some non-corrosive food cans such as meats (Danish EPA 2007).

Plastic alternatives to polycarbonates can have similar properties to polycarbonate. For instance thermoplastic polyamide, such as Grilamid TR (EMS Chemie AG) is UV, high chemical and stress crack resistant and hence can be used for baby bottles. Main suppliers of polyamide in Europe include EMS Chemie, BASF, Rhone- Poulenc and DuPont (Danish EPA 2007). One baby bottle maker (Born Free) uses polyethersulfone rather than polycarbonate. The material is four to five times as expensive as polycarbonate.

In late 2007 a new alternative to polycarbonate plastics emerged: Tritan. Copolyester, manufactured by Eastman Chemicals. It is claimed to have all the advantages of polycarbonate plastics but contains no Bisphenol A. Tritan is more expensive than polycarbonate, increasing the retail price by at least 10%. The three major plastic drink bottle producers: Camelbak, Nalgene, Alladin, and Vita-mix (blender containers) all claim they will use Tritan in their new products. (Austin 2008).

Nevertheless, the environmental and health effects of any alternatives will need to be carefully researched, and while they may not have endocrine disrupting effects, they may well have yet unknown, other harmful side effects.

As development of alternatives to plastics may be a lengthy and costly, it is imperative that producers and users of hazardous chemicals commit to researching and adequately funding this type of research now. The research should take into consideration both chemical and non-chemical (technological) solutions.

An important alternative to plastic is of course glass, especially for food and water storage. Glass was traditionally used for many food storage applications and could again replace many polycarbonate plastics.

5.2 Encouraging companies to change

An important aspect of turning public and government opinion in relation to BPA around is taking action in the market place. If everybody or a large proportion of consumers stopped buying baby bottles or other products containing BPA, manufactures would get the message very quickly. Similarly if an increasing number of people asked their local supermarket or retailer to stop selling products containing BPA, eventually they will respond and this will have a ripple on effect to the manufacturers.

Another opportunity to force the issue on BPA is shareholder activism. For instance in the US over 24 shareholder resolutions have queried publicly held corporations about their use or sale of potentially toxic chemicals in the last two years. Responses by companies to these shareholder actions have been hostile or, at best, rather modest. For instance, Whole Foods Market, the largest retail chain of natural foods supermarkets in the US with a turnover of over $US4.7 billion in 2006, initially opposed a 2006 resolution requesting the removal of all products containing BPA from its shelves. Eventually it bowed to pressure and stopped merchandising kid's cups and baby bottles containing BPA (Spivake 2007).

More proactively, in 2005 Patagonia, a well known US outdoor equipment retailer, stopped selling polycarbonate bottles, followed in December 2007, by Mountain Equipment Co-op. Mountain Equipment Co-op, Canada's largest camping and outdoor retailer, pulled most food and beverage containers made of polycarbonate plastic off the shelves awaiting further information from the Canadian government. Mountain Equipment Co-op cited possible health risks as the reason for their action. Baby-bottle maker Playtex claimed in April 2008 that it will phase out bottles containing BPA .

While some governments around the world have set safety limits for BPA exposure, many are outdated, rely on scientific research that is years old assumptions about toxicology that need to be revised according to the latest scientific findings.

CONCLUSION

FoE Australia believes that evidences of BPA exposure are widespread and abundant and urgent action to reduce human BPA exposure is needed.

FoE Australia urges companies to phase out BPA from all consumer products and assign the necessary means to developing safe and efficient alternative to BPA.

FoE Australia encourages consumers and retailers to pressure their own retailers and providers to ensure the complete disappearance of BPA from all consumer products.

Time for critical decision has come

FOE Australia urges the Australian Government to review its opinion on BPA taking into account the latest scientific evidence and to act on the basis of precautionary principle.

Quick Guide: What YOU can do to reduce BPA exposure and minimize its adverse effects

Food Packaging and Storage: choose safe options

- Store food in glass, ceramic or stainless steel containers.
- Buy fresh and local produce; try to avoid fruit and vegetables grown in greenhouses.
- If you need to use plastic that comes into contact with food, choose safer options where possible: Suitable plastics are those with a recycling code no.1 (, (xx or PETE), no. 2 (high density poly ethelyene or HDPE), recycling code no.4 (low-density polyethylene or LDPE) and no.5 (polypropylene PP)

Take care to avoid polycarbonate plastics (PC) as much as possible

Avoid plastics with recycling code no. 3, 6 or 7.

- No.7 (other plastics) may contain Bisphenol A and are best avoided.
- Additionally take care and avoid PVC (no.3) and polystyrene (no.6), as the possible residues (vinyl chloride and styrene) may also be harmful (Mutti et. al 1984, Benignus 2005). PVC has negative environmental and health impacts during production, use and after disposal, for instance when waste is incinerated.

Avoid heating foods or drinks in plastic containers

- Avoid heating all plastics, irrespective of their recycling numbers. If you need to store heated food or liquid in plastic containers, wait until it has cooled down.

Avoid canned food and foods grown in plastic greenhouses.

Although it is not always possible to identify fruits and vegetables grown in plastic greenhouses, eating seasonal products can be first step and a good way to avoid food grown under plastic greenhouses.

Caution: food wrapping

- Meats, cheeses, and other commercially-wrapped foods in delis/speciality food shops and standard food shops / supermarkets may be wrapped in PVC, which we recommend you avoid.
- Some of the commercial wraps sold for home use are made from polyethylene (no.4).

Caution: unlabelled could mean unsafe

- Many plastic items are unlabelled and the only way to find out what they are made of is by contacting the manufacturer. We encourage you to do so and to express your concerns.
- In the absence of information: **avoid using plastics where possible**. The safer alternatives are glass and stainless steel.

Take care with all plastic products

- Take plastic products to recycling stations where possible.
- Ask your dentist to use dental sealants that do not contain BPA.

Engage with retailer and producers

- Always read the labels. Sometimes a company declares that their product is free of BPA.
- Ask you retailer to stop using and selling polycarbonate food contact materials.
- Contact the manufacturer and ask them whether the food contact material contains BPA.

How to ensure your baby and infant have minimal exposure to BPA

Babies and infants are especially at risk from low dose BPA exposure. Here are some suggestions:

Feeding

- Breastfeed whenever possible for as long as possible. Breast milk is the optimal food for your baby, the World Health Organisation (WHO) recommends six months of exclusive breastfeeding and continued breastfeeding thereafter until two years or longer, so no need for infant formula and bottles
- If you need to use infant formula, choose a powdered one, as liquid formulas have higher levels of BPA and use glass bottles or cups for feeding.
- Use as few cans as possible.
- Do not use ready-to-eat liquid formulas in metal cans.
- Avoid liquid formulas that are in rigid and transparent plastic containers marked with PC.
- When expressing breast milk, use breast pumps, shields and jars and bags that are BPA free.

Baby bottles/sippy cups:

- **Use glass or plastic baby bottles that are labelled "Bisphenol A-free" or made of polyethylene, polypropylene or polyamide** (National Childbirth Trust UK 2008)

Teats, 'dummies' or pacifiers

- Choose teats/ 'dummies' or pacifiers made from silicon. They are the most durable and inert options.

Ask your local childcare centre to get rid of all polycarbonate food contact and food storage materials

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Plastic Code Quick Guide

Avoid: 3,6,7

No.3-PVC (Polyvinyl chloride)
No.6-PS (Poly Styrene)
No.7- PC (Poly Carbonate)9

Probably Safe: 1,2,4,5

No.1-PET (Polyethylene terephthalate)
No.2-HDPE (High-density Polyethylene)
No.4-LDPE (Low-density Polyethylene)
No.5-PP (Polypropylene)

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Appendix 1: A primer on endocrine disrupting chemicals

Endocrine disrupting chemical: a brief introduction

The endocrine system is a complex network of glands, hormones and receptors that carefully regulates many bodily functions, including our metabolism, immunity, behaviour and growth and development during childhood. Hormones play a complex role in the human body and mind and regulate our response to disease, reproduction and even influence our behaviour and relationships with each other, e.g. mother child bonding (Environment Directorate-General of the European Commission 2008).

The endocrine system is a messaging system: The glands secrete hormones, which act as the chemical messages and are transported by the bloodstream. Hormones are received by receptors, which will detect and react to specific hormones in particular cell/tissue types. This mechanism functions very much like a lock and key. Malfunctioning of the endocrine system may trigger diseases including diabetes, thyroid diseases, obesity and some cancers (NRDC 1998).

What are endocrine disruptors?

EDCs are chemicals that may cause adverse health effects by altering the function of the endocrine system by either modifying the action of or mimicking hormones produced by the body itself (WHO 2002). However these chemicals have a different and more complex impact on our bodies than many other chemicals. The same chemical may at different doses, halt or stimulate the production of a particular hormone or even change the way the hormone travels through the body. In the last twenty years concerns over the effect of these chemicals have continued to grow, despite industry and governments frequently denying their apparent effects. Well known human EDCs include diethylstilbesterol (the drug DES), dioxins, PCBs, and DDT, but many other chemicals, particularly pesticides and plasticisers, are suspected to function as endocrine disruptors (Environment Directorate-General of the European Commission 2008).

How do endocrine disruptors function?

Some EDC's can disturb the stabilising mechanisms of the body or initiate processes at an unexpected time during a person's life cycle. A number of different mechanisms how this can occur have been proposed:

- They may imitate the natural hormone, bind to the receptor sites, but cause unexpected results.
- They may physically block the binding of the natural hormone to its receptor.
- They may alter the amount of natural hormone present in the blood by binding to transport proteins.
- They may affect the synthesis or breakdown rate of the natural hormone, thereby disrupting the metabolic process of the body. (Environment Directorate-General of the European Commission 2008).

How do endocrine disruptors affect humans and animals?

Both humans and wildlife are clearly affected by EDCs. Countless wildlife studies, including studies on molluscs, crustaceans, fish, reptiles, birds and mammals have shown that exposure to environmental chemicals can lead to endocrine disruption in these species. Some of the effects of EDCs on wildlife include abnormalities and impaired reproductive performance in some species, changes in immunity and behaviour and skeletal deformities. EDC have also been implicated in many changes in human health patterns over recent decades, including declining sperm counts in some geographical regions, increased numbers of male children born with genital malformations, and increases in certain types of cancer that are known to be sensitive to hormones as well as impairment in neural development and sexual behaviour (Environment Directorate-General of the European Commission 2008).

Appendix 2: Scientific consensus on the dangers endocrine disruption (adapted and expanded from http://www.ourstolenfuture.org/Consensus/consensus.htm)

Declaration	Key Message
Chapel Hill Bisphenol A Expert Panel Consensus Statement 2007	Thirty-eight of the world's leading scientific experts on Bisphenol A, a known EDC, have warned policymakers of potential adverse health effects of the widespread exposure to this chemical
Vallombrosa 2005	Vallombrosa Consensus Statement concludes that environmental contaminants including EDCs are responsible for compromising human fertility
Prague Declaration 2005	Prague Declaration on Endocrine Disruption urges precautionary approach
International Programme on Chemical Safety (NIEHS-WHO), 2002	Global Assessment of the State-of-the-Science of Endocrine Disruptors justifies concern about possible human health impacts
US National Toxicology Program, 2000	Scientific peer review of “low-dose” studies confirms adverse effects and concludes that “low-dose” considerations must be integrated into regulatory science
The Royal Society 2000	Endocrine disrupting chemicals (EDCs) - **"**Regulations cannot be 'put on hold' until all the evidence has been collected.**"**
Yokohama 1999	The Effects of Endocrine Disruptors in Living Things stresses the need to initiate investigations into human health hazards caused by endocrine disruptors, in the meantime deems it important to take a precautionary approach
National Research Council 1999	Hormonally-active Agents in the Environment – the report demonstrated that the risks, while not proven, are both serious and highly plausible.
Erice 1995	Environmental endocrine disrupting chemicals have neural, endocrine and behavioural effects. The authors were confident that every pregnant woman in the world has endocrine disruptors in her body that are transferred to the foetus. She also has measurable concentrations of endocrine disruptors in her milk that are transferred to the infant.
Wingspread 1995-II	Chemically-induced alterations in the developing immune system: the wildlife/human connection
Wingspread 1995-I	Chemically-induced alterations in functional development and reproduction of fishes
Wingspread 1993	Environmentally induced alterations in development: a focus on wildlife.
Wingspread 1991	Chemically-induced alterations in sexual development: the wildlife/human connection

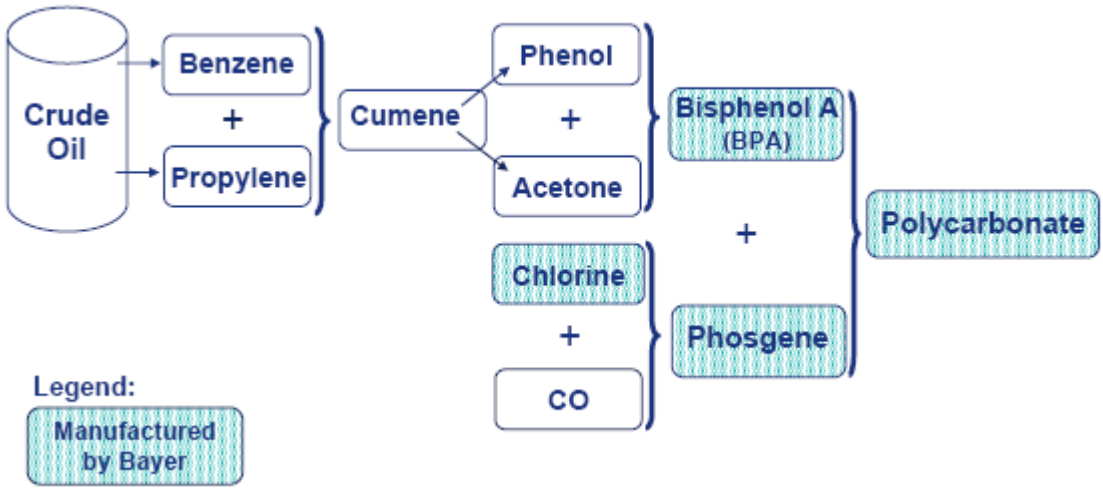
Appendix 3: How exposure and concentration are measured

How exposure and concentration are measured

Ppb: Parts per billion. A measurement that is used to specify the concentration (by volume) of a dissolved material at high dilution. For instance 1 ppb represents 1 microgram of a substance per litre of water (µg/l).
1mg – one milligram is one thousandth of a gram or 10–3 g
1µg – one microgram is a one millionth of a gram or 10–6 g
1 ng/l – one nanogram per litre is 0.001 microgram per litre or 10–9 g

Appendix 4: Production information

Raw materials for Polycarbonates include:



Source: Babe 2007

Appendix 5: Excerpt of the list of EFSA committee and their affiliations and subject areas

Source: http://www.efsa.europa.eu/en/science/afc/afc_members.html

Name	Area of Expertise / current affilitation	Potential conflicts of interest
Dr. Fernando Aguilar	Food toxicologist French Food Safety Agency	▪ Worked for Nestle, ▪ Spouse still working for Nestle
Prof. Herman Autrup	Toxicologist Institute of Public Health, University of Aarhus	▪ Greenfacts* board member ▪ Member of advisory board to CEFIC
Dr. Susan Barlow (chair of committee)	Toxicologist/former UK bureaurcrat, now self employed	▪ Consultant to Unilever, Tesco, GNT, Grant /Son ▪ Greenfacts* member, work included drafting papers including on endocrine disruptors ▪ Husband CEFIC European Chemical Industry Council consultant
Prof. Wolfgang Dekant	Toxicologist Institute of Toxicology, University of Wuerzburg	▪ Contracts for undisclosed private companies (Hoechst? Clariant?) ▪ Financial support by undisclosed industry organisation to write articles ▪ Actively opposes low dose bpa research: ▪ http://www.efsa.europa.eu/EFSA/General/10_30_W_Dekant_21Nov,0.pdf ▪ http://www.bfr.bund.de/cm/232/bisphenol_a_hazard_and_health_risk_assessment_of_a_food_contact_material.pdf)
Prof Karl-Heinz Engel	Food Chemist-Technologist Chair, General Food Technology, Technical University Munich	▪ Contracts from Degussa, Kraft, Suedzucker, Frey and Lau, Dr. Willmar Schwabe GMBH, T. Hasegawa Japan, indirect Monsanto, Symrise, Ajinomoto
Prof Ivonne Rietjens	Food toxicologist Prof Toxicology, Wageningen University, Netherlands	▪ Research collababoration TNO Zeist, ▪ Consultant / research with Nestle ▪ Member of expert panel of flavour and extract manufacters association (FEMA) ▪ Advisory boards Nanotox BV private
Prof Paul Tobback	Food process engineering Emiritus Professor, Belgium	▪ Member of scientific committee of Belgian food industry assoc, ▪ Consultant to Carrefour, SGS S&SC
Prof Fidel Toldra	Food Chemist Prof Meat Science Group, CSIC, Spain	▪ Vanquera meat industry grant ▪ Various private meat promotion NGOs
Dr Frank Sullivan	Toxicologist Consultant	▪ Husband of Susan Barlow ▪ CEFIC consultant ▪ AD hoc expert ▪ DOI had been removed from EFSA website

Background on "Greenfacts"
GreenFacts, formerly the GreenFacts Foundation, is an international non profit organisation founded in 2001 in Brussels, Belgium. It is primarily funded by industrial companies such as Solvay (a Belgian chemicals company, which has made the information it disseminates the subject of some criticism.

In 2006 Greenfacts had a total budget of over EUR 500,000, with over 50% coming from industrial companies such as Carrefour (a European supermarket group), CEFIC (the European Chemical Industry Council), Euro Chlor, PlasticsEurope, the European Crop Protection Association, GlaxoSmithKline Biologicals, Proctor & Gamble, Raffinerie Tirlemontoise (a sugar company), Suez and Total Petrochemicals, Solvay and Ferrari Textiles. In 2007 additional corporate sponsors included Cumerio, DSM, Floridienne and Umicore. (Sourcewatch 2008).

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