BISPHENOL A - *INFORMATION SHEET*

THE COMPOUND

Bisphenol A (BPA) is a chemical used primarily as a monomer in the production of polycarbonate plastics and epoxy resins. Polycarbonate plastics are rigid plastics sometimes used for food contact materials, including drink bottles. Epoxy resins are used for lining tin cans. Three major types of resin are used for food cans where the food is sterilised in the can namely; epoxyphenolic, PVC organosol and polyester phenolic. Of these coatings, the epoxyphenolic is the most important and is used where the can is made of two or three pieces, particularly shallow draw, 2 piece cans such as those used for canned fish. BPA is a starting substance used in the manufacture of most types of epoxy resins but is not normally present in PVC organosol coatings.

SOURCES

BPA may occur in food due to migration of incompletely polymerised or depolymerised BPA monomer from:

- Epoxy resin coatings of canned foods. BPA is more likely to migrate into oily or fatty foods, such as meat, fish and coconut cream (Scientific Panel on Food Additives Flavourings Processing Aids and Materials in Contact with Food, 2006; Thomson and Grounds, 2005).
- Polycarbonate infant feeding bottles and drinking bottles. Migration from new bottles is very low, but can increase with repeated use, due to cleaning treatments (Environment Canada, 2008).
- Coatings for wine storage vats (Scientific Panel on Food Additives Flavourings Processing Aids and Materials in Contact with Food, 2006).
- Polycarbonate tableware and food storage containers. Migration from these sources appear to be negligible (Scientific Panel on Food Additives Flavourings Processing Aids and Materials in Contact with Food, 2006).

POTENTIAL HEALTH EFFECTS

BPA is weakly estrogenic – it is able to mimic the female sex hormone, 17β-estradiol, but requires much higher concentrations to achieve the same effect. Consequently, the main effects of interest for BPA are developmental and reproductive toxicity.

Much of the science related to potential human health effects of BPA is contentious. Two pivotal issues are:

- The toxicokinetics of BPA. Most toxicity studies on BPA have been carried out on rodents (rats and mice). Studies suggest that BPA is metabolised very differently in humans than in rodents (Scientific Panel on Food Additives Flavourings Processing Aids and Materials in Contact with Food, 2008). BPA is rapidly converted to a non-estrogenic conjugate in humans and is excreted in that form, almost entirely via urine. In rodents, the same conjugation occurs, but the conjugated form is recirculated and converted back into free BPA.

- Low dose effects. While most studies on experimental animals have shown effects only at exposure levels far in excess of those experienced by humans, some have shown effects at very low doses, including effects on organ weights, tissue architecture, receptor expression and behaviour (Norwegian Scientific Committee for Food Safety (VKM), 2008). The European Food Safety Authority (EFSA) assessment noted that many of these studies of low dose effects suffered from methodological problems and their findings were at odds with large, well-conducted studies (Scientific Panel on Food Additives Flavourings Processing Aids and Materials in Contact with Food, 2006)

The 2006 EFSA assessment concluded that the most sensitive effect seen in large, well-conducted studies was liver toxicity, with reproductive toxicity observed at dose levels an order of magnitude higher (Scientific Panel on Food Additives Flavourings Processing Aids and Materials in Contact with Food, 2006; Tyl *et al.*, 2008; Tyl *et al.*, 2002).

Little human epidemiological evidence is available on health effects of BPA. A recent cross-sectional epidemiological study has reported associations between urinary BPA and coronary heart disease and diabetes in humans (Lang *et al.*, 2008). Urinary BPA is indicative of exposure in the previous 24 hours and is not necessarily representative of exposure over the period when these diseases may develop. EFSA have reviewed this study and concluded that it does not provide sufficient proof of a causal link (EFSA, 2008).

ESTIMATES OF DIETARY EXPOSURE

The average dietary exposure to BPA by New Zealanders from consumption of canned foods has been estimated as 0.008 µg/kg body weight/day, based on actual measurements of BPA in canned foods and New Zealand dietary recall data (Thomson and Grounds, 2005). This is substantially below the TDI assigned in Europe of 50 µg/kg body weight/day.

Conservative exposure assessment for European populations were in the range 0.2 (breast-fed infants) to 13 (formula-fed infant at worst concentration of BPA in infant formula) µg/kg body weight/day (Scientific Panel on Food Additives Flavourings Processing Aids and Materials in Contact with Food, 2006). FDA conservative estimates for the US are lower at 0.2-2.4 µg/kg body weight/day for infants (FDA, 2008).

BPA exposure can also be assessed by analysis of urine for metabolites. Urinary analysis data from the US and Japan suggests dietary exposure to BPA of the order of 0.04-0.2 µg/kg body weight/day (Calafat *et al.*, 2005; LaKind and Naiman, 2008; Miyamoto and Kotake, 2006).

All exposure estimates are well below the ADI of 50 µg/kg body weight/day.

FACTORS INFLUENCING RISK

Migration of BPA from the can lining into canned food appears to largely occur during the canning process. The duration of can storage, temperature of storage and the condition of the can (dented or undented) have little impact on the final BPA concentration of the food (Goodson *et al.*, 2004).

SAFETY ASSESSMENTS

BPA has been the subject of a number of safety assessments in recent years, including:

- a European Union environmental and human health assessment (European Communities, 2008) and a follow-up assessment of four studies on developmental neurotoxicity (Norwegian Scientific Committee for Food Safety (VKM), 2008);
- an EFSA assessment (Scientific Panel on Food Additives Flavourings Processing Aids and Materials in Contact with Food, 2006) and a follow-up assessment of the toxicokinetics of BPA (Scientific Panel on Food Additives Flavourings Processing Aids and Materials in Contact with Food, 2008);
- an environmental and human health screening assessment by Environment Canada (Environment Canada, 2008); and
- an assessment by the US National Toxicology Program (NTP) (National Toxicology Program, 2008)
- a US Food and Drug Administration assessment for use in food contact applications (FDA, 2008). This assessment is currently in draft form and FDA is preparing a response to review of the document conducted by the FDA Science Board.

With respect to human health, the EU assessment concluded that "There is at present no need for further information and/or testing and for risk reduction measures beyond those which are being applied already".

EFSA set a Tolerable Daily Intake (TDI) for bisphenol A of 0.05 mg/kg body weight/day (50 µg/kg body weight/day). This was based on effects on the liver in animal studies. It was concluded that reproductive effects occurred at doses ten-fold higher than those at which liver effects were seen.

Both the Environment Canada and Norwegian assessments signalled concerns related to possible behavioural neurotoxicity

NTP summarises assessments on a five-point scale of concern, where the mid-point is 'some concern'. Their summary for bisphenol A was:

- *some concern* for effects on the brain, behavior, and prostate gland in foetuses, infants, and children at current human exposures to bisphenol A.
- *minimal concern* for effects on the mammary gland and an earlier age for puberty for females in foetuses, infants, and children at current human exposures to bisphenol A.
- *negligible concern* that exposure of pregnant women to bisphenol A will result in foetal or neonatal mortality, birth defects, or reduced birth weight and growth in their offspring.

- *negligible concern* that exposure to bisphenol A will cause reproductive effects in non-occupationally exposed adults and *minimal concern* for workers exposed to higher levels in occupational settings.

SAFETY AND REGULATORY LIMITS

Safety limits are levels of dietary exposure that are without appreciable risk for a lifetime of exposure. Regulatory limits define the maximum amount of a substance that is permitted in a particular food.

Source	Limit Type	Limit
Safety Limits		
No safety limits have been set for BPA in New Zealand or by the Joint FAO/WHO Expert Committee on Food Additives		
Regulatory Limits		
No regulatory limits have been set for BPA in New Zealand or Australia		

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