BISPHENOL A: INFORMATION SHEET

HUMAN SAFETY: AN OVERVIEW

Summary

Bisphenol A (BPA) is a key industrial chemical used to make polycarbonate plastic, epoxy resins and other products. Following the four-step procedure recommended by the United States National Academy of Sciences (NRC, 1983), a safety assessment of BPA concludes that the potential human exposure to BPA from polycarbonate plastic and epoxy resin food contact applications is minimal and poses no known risk to human health. This conclusion is based on the following key points:

1) BPA is not carcinogenic and does not selectively affect reproduction or development. The No-Observed-Adverse-Effect-Level (NOAEL) for BPA, confirmed in multiple laboratory animal tests, is 50 mg/kg body weight/day;
2) The estimated dietary intake of BPA from polycarbonate plastic and epoxy resin food contact applications, based on the results of multiple migration studies with consistent results, is less than 0.000118 mg/kg body weight/day; and
3) This potential human exposure to BPA is more than 400 times lower than the maximum acceptable or "reference" dose for BPA of 0.05 mg/kg body weight/day established by the U.S. Environmental Protection Agency, which is derived from the NOAEL.

An independent analysis by the European Commission's Scientific Committee on Food (SCF), using a similar methodology, has confirmed the safety of polycarbonate plastic and epoxy resin food contact applications. The SCF estimated total dietary intake of BPA from all food contact sources to be in the range of 0.00048 to 0.0016 mg/kg body weight/day, which is below the Tolerable Daily Intake set by the SCF of 0.01 mg/kg body weight/day.

The use of polycarbonate plastic and epoxy resins for food contact applications has been and continues to be recognized as safe by the U.S. Food and Drug Administration, the European Commission's Scientific Committee on Food, the United Kingdom Food Standards Agency, the Japanese Ministry for Health, Labor and Welfare, and other regulatory authorities worldwide.

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The Four-Step Safety Assessment Procedure

Step 1: Hazard Identification

The objective of the hazard identification step is to qualitatively identify the health effects that may be associated with exposure to BPA.

HUMAN SAFETY ASSESSMENT OF BISPHENOL A / page 2

Carcinogenicity

The weight of scientific evidence from numerous studies, including two long-term studies, indicates that BPA is not carcinogenic (Haighton *et al*, 2002). Among these studies are lifetime exposure cancer bioassays conducted in rats and mice by the U.S. National Toxicology Program (NTP, 1982). There was no convincing evidence in either of the bioassays that BPA was carcinogenic. In addition, BPA is without mutagenic or genotoxic activity *in vivo.* These conclusions were also reached by the European Union in their comprehensive risk assessment on BPA (EU RAR, 2002).

Reproductive and Developmental Toxicity

In 1995, the Bisphenol A Toxicology Task Force of the Society of the Plastics Industry, Inc. completed a comprehensive review of available data on BPA (BATTF, 1995). Seven laboratory animal reproduction and development tests, including four conducted by the U.S. National Toxicology Program, were reviewed. These studies found no evidence that BPA selectively affects reproduction or development. Rather, effects on reproduction and development were observed only at doses of BPA so high that the health of the pregnant animal was compromised. These conclusions are consistent with those reported in the comprehensive European Union risk assessment on BPA (EU RAR, 2002).

Low-Dose Hypothesis

In recent years, a hypothesis has been advanced claiming that exposure to extremely low doses of certain substances could cause adverse health effects in humans, including disruption of normal hormonal functions. In science, a hypothesis is a limited statement regarding cause and effect that has not been confirmed through repeated experimental tests. According to this "low-dose hypothesis", health effects occur at doses far below levels previously determined to be safe using well-established toxicological procedures and principles. The hypothesis further asserts that the dose-response relationship for these substances is "non-monotonic", which means that health effects may only be observed at low doses while much higher doses result in no effects. The claimed non-monotonic dose-response relationship of the low-dose hypothesis is contrary to a fundamental principle of toxicology – "the dose makes the poison."

The low-dose hypothesis is largely based on several small-scale experimental studies that report reproductive or developmental effects in mice or rats from low doses of BPA. Several attempts to confirm the hypothesis by repeating these initial experiments have shown that the results cannot be replicated, which indicates that the hypothesis is not valid. More importantly, definitive large-scale experiments using accepted protocols have also found no evidence for reproductive or developmental effects from low doses of BPA. Consequently, a number of independent scientific bodies, after reviewing all available evidence, have concluded that the low-dose hypothesis is unproven (BPA INFO, 2002a).

HUMAN SAFETY ASSESSMENT OF BISPHENOL A / page 3

The most definitive tests of the validity of the low-dose hypothesis for BPA are two large-scale reproductive and developmental toxicity studies using accepted protocols. Both of these studies clearly demonstrated the absence of a low-dose effect of BPA.

The most comprehensive of these is a three-generation study conducted at the Research Triangle Institute (now RTI International) under the direction of Dr. Rochelle Tyl (Tyl *et al*, 2002). In this study, Sprague-Dawley rats were fed a diet containing BPA at levels from 0 to 7500 parts per million, yielding approximate intakes of 0, 0.001, 0.02, 0.3, 5, 50 or 500 mg/kg body weight/day. Exposures were continued until adulthood of the third-generation offspring and a wide variety of relevant endpoints were evaluated. Analysis of the data for all of the endpoints for the parental and three offspring generations revealed no evidence of a low-dose effect of BPA. This exceptionally powerful study, which complied with Good Laboratory Practice (GLP) standards, clearly demonstrated the absence of low-dose effects for BPA.

In a similar study commissioned by the Japanese National Institute of Health Sciences and carried out by the Chemical Compound Safety Research Institute (Ema *et al*, 2001), Crj:CD (SD) IGS rats were dosed orally by stomach tube over two generations. The doses applied were 0, 0.2, 2.0, 20 or 200 µg/kg body weight/day of BPA. Analysis of the data for the parental and two offspring generations revealed no evidence of a low-dose effect of BPA, which is fully consistent with the results of the Tyl study.

The lack of low-dose effects in the definitive large-scale studies and the inability to replicate low-dose effects reported in small-scale studies demonstrates that the low-dose hypothesis for BPA cannot be valid. A series of independent bodies have comprehensively reviewed the evidence for and against low-dose effects and have consistently reached this same conclusion.

In 2000, the U.S. National Toxicology Program (NTP) conducted an independent scientific peer review of the evidence for and against "low-dose endocrine disruptor" effects (NTP, 2001). In their overall conclusion, the Bisphenol A Subpanel stated:

> "There is credible evidence that low doses of BPA can cause effects on specific endpoints. **However, due to the inability of other credible studies in several different laboratories to observe low dose effects of BPA, and the consistency of these negative studies, the Subpanel is not persuaded that a low dose effect of BPA has been conclusively established as a general or reproducible finding.** In addition, for those studies in which low dose effects have been observed, the mechanism(s) is uncertain (i.e., hormone related or otherwise) and the biological relevance is unclear." (emphasis added)

In 2002, the European Commission completed a comprehensive risk assessment on BPA, which includes a review of evidence for and against low-dose effects (EU, 2002). The European Commission's Scientific Committee on Toxicity, Ecotoxicity and the

HUMAN SAFETY ASSESSMENT OF BISPHENOL A / page 4

Environment (CSTEE, 2002) independently reviewed the Risk Assessment Report (RAR) and stated:

> "[A] number of studies using non-standard protocols have reported effects of bisphenol A administration on development using substantially lower doses than the studies performed according to testing guidelines. The RAR critically describes the many weaknesses (lack of repeatability, problems with experimental design and statistical evaluation, poor reporting) of the low dose studies. The CSTEE agrees with the conclusion of the RAR that there is no convincing evidence that low doses of bisphenol A have effects on developmental parameters in offspring and remarks that effects observed are not adverse."

The CSTEE further remarked, "a number of high quality studies on the reproductive and developmental effects of bisphenol A are already available and do not support low-dose effects."

Also in 2002, the Japanese Ministry of Economy, Trade and Industry released a hazard assessment of BPA (METI, 2002). In regard to the need for risk assessment or other measures, the Ministry stated:

> "Though it is necessary to collect further information on so-called 'low dose effects' represented by BPA from academic point of view, it seems unnecessary to take any specific measure other than the above, considering the view expressed by NTP Low Dose Effect Panel that the low dose effect of BPA at present is a phenomenon observed under considerably limited experimental conditions and it is hardly considered to be the general phenomenon."

In addition to these assessments specifically on BPA, other independent bodies (e.g., U.S. Environmental Protection Agency, Japanese Ministry of Health, Labor and Welfare) have reached similar conclusions on the validity of the low-dose hypothesis in general (BPA INFO, 2002a).

These weight-of-evidence assessments, the lack of low-dose effects in definitive large-scale studies, and the inability to replicate low-dose effects reported in small-scale studies all support the conclusion that the low-dose hypothesis is not valid.

Step 2: Dose-Response Assessment

The objective of the dose-response assessment is to define the relationship between exposure level (dose) with the frequency and severity of any health effects associated with exposure to BPA. Specific outcomes from this step are identification of a No-Observed-Adverse-Effect-Level (NOAEL) from animal studies, which can then be used as the basis for calculation of a "reference dose" or other similar values. The reference dose is defined by the U.S. Environmental Protection Agency as an estimate of a daily

HUMAN SAFETY ASSESSMENT OF BISPHENOL A / page 5

oral exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime (EPA, 1993).

Based on the results of the lifetime exposure cancer bioassays conducted by the U.S. National Toxicology Program, the U.S. Environmental Protection Agency selected 50 mg/kg body weight/day as the basis for the reference dose. The reference dose is calculated by dividing the selected dose of 50 mg/kg body weight/day by a safety factor of 1000, which results in a reference dose of 0.05 mg/kg body weight/day (EPA, 1993).

The results of the two multi-generation studies designed to look for low-dose effects of BPA (Ema *et al*, 2001; Tyl *et al*, 2002) support the use of the 50 mg/kg body weight/day dose for calculating a reference dose. No low-dose effects were observed in either study.

As part of a comprehensive risk assessment on BPA, the European Union reviewed all available toxicity data, including evidence for low-dose effects, and also concluded that the NOAEL is 50 mg/kg body weight/day (EU RAR, 2002).

Likewise, the Bisphenol A Toxicology Task Force estimated the No-Observed-Adverse-Effect-Level (NOAEL) for BPA to be 50 mg/kg body weight/day based on the dose-response results observed in the seven reproduction and development tests and the lifetime exposure cancer bioassays (BATTF, 1995).

STEP 3: EXPOSURE ASSESSMENT

The objective of the exposure assessment step is to estimate the level of potential human exposure to BPA. The exposure estimate can then be compared with the reference dose to determine if the potential exposure to BPA results in any risk to human health.

Potential human exposure to BPA is primarily from intake of foods and beverages that have been held in polycarbonate plastic containers or packaged in metal cans coated with an epoxy resin. For both polycarbonate plastic and epoxy resins, potential human exposure is based on measured migration data.

Polycarbonate Food and Beverage Containers

Many researchers have studied the potential for trace levels of BPA to migrate from polycarbonate into food and beverages under conditions typical for uses of polycarbonate products. These studies include ones conducted by government agencies in the US, Europe and Japan, as well as studies conducted by academic researchers and by industry.

These studies generally show that, under typical use conditions, the potential migration of BPA into food is extremely low. Migration testing under conditions that are typical of

HUMAN SAFETY ASSESSMENT OF BISPHENOL A / page 6

how polycarbonate products are actually used indicates that migration of BPA, when it is detected, is generally less than 5 parts per billion.

Some of the most notable examples include studies conducted by the:

1. U.S. Food and Drug Administration (FDA) on baby bottles, water bottles and cut portions of baby bottles under "typical/normal use" conditions (Biles *et al*, 1997);
2. U.K. Ministry of Agriculture, Fisheries and Food (MAFF) on baby bottles subjected to 20-50 cycles of cleaning, sterilizing and simulated use (Mountfort *et al*, 1997; MAFF, 1997);
3. U.K. Department of Trade and Industry (DTI), Consumer Affairs Directorate on baby bottles handled under "realistic worst-case conditions of use" (Earls *et al*, 2000);
4. Japanese National Institute of Health Sciences (NIHS) on tableware and baby bottles under conditions representative of normal consumer use (Kawamura *et al*, 1998); and
5. Society of the Plastics Industry, Inc. (SPI) on polycarbonate discs under the most rigorous conditions recommended by FDA (Howe and Borodinsky, 1998).

These studies are not identical in design but all aimed to measure the potential migration of BPA into foods and beverages under temperature and time conditions considered to be typical of how polycarbonate products are actually used. Considered together, these studies cover a complete range of polycarbonate food contact products and end-use conditions, which provides reassurance that the collective results fully represent the potential migration of BPA into foods and beverages. The results of these studies are briefly summarized below in reference to the type of polycarbonate product or article that was tested. More detailed information is also available (BPA INFO, 2002b).

Baby Bottles: Each of the studies conducted by the government agencies included or focused entirely on baby bottles. In most cases, new baby bottles were studied under well-characterized laboratory conditions. Migration was measured into infant formula, fruit juice or a range of solvents to simulate food. In each case, migration of BPA from new baby bottles, when detected, was less than 5 parts per billion.

Water Bottles: In the US FDA study, water from several 5-gallon polycarbonate bottles from a bottled water supplier was analyzed with a detection limit of 0.05 parts per billion. In water that had been stored in the bottles for up to 39 weeks, BPA was found at extremely low levels ranging from 0.1 to 4.7 parts per billion.

Tableware: The Japanese NIHS study evaluated several mugs and ricebowls along with a measuring cup. No BPA was detected above the 0.5 part per billion limit of detection when 3 of 5 articles were exposed to either water (95^{o}C for 30 minutes) or 20% ethanol (60^{o}C for 30 minutes). Migration of BPA was observed from the other 2 articles, but only at levels below 5 parts per billion.

HUMAN SAFETY ASSESSMENT OF BISPHENOL A / page 7

Molded Discs/Cut Pieces: In addition to evaluation of whole baby bottles, the US FDA study also tested migration from baby bottles that had been cut into pieces and immersed in food simulating solvents. For both simulants tested, the amount of BPA detected was estimated to be equivalent to migration of approximately 2 ng/ml (equal to 2 parts per billion) from a whole baby bottle.

The Society of the Plastics Industry, Inc. conducted a study (Howe and Borodinsky, 1998) to measure migration from molded discs that were prepared from a blend of polycarbonate resin from three American manufacturers. The three resins were blended and pressed into small discs such that all surfaces were similar to that of a finished polycarbonate product. The study was conducted according to procedures developed by the US FDA (FDA, 1995, revised 2002) and performed using storage time and temperature conditions recommended by the US FDA. No BPA migration was detected in any of the samples with a 5 part per billion limit of detection.

Using procedures recommended by the U.S. Food and Drug Administration for estimating exposures from food-contact applications (FDA, 2002), and a migration value of 5 parts per billion, the estimated dietary intake of BPA from polycarbonate food and beverage containers is less than 0.0125 micrograms (0.0000125 milligrams) per kilogram body weight per day (BPA INFO, 2002b).

Epoxy Resin Can Coatings

The potential for trace levels of BPA to migrate from epoxy resin can coatings into foods and beverages under typical use conditions has been studies by several researchers. The two most comprehensive studies are briefly summarized below and more detailed information is also available (BPA INFO, 2002c).

The Society of the Plastics Industry, Inc. (SPI) conducted a study of BPA migration from a variety of can coatings into food simulating solvents (Howe *et al*, 1998). The test conditions used for this study were those recommended by the U.S. Food and Drug Administration (FDA, 1995, revised 2002) to exaggerate the normal canning processes used. No detectable migration of BPA from the three beverage/beer can coatings tested was found with an analytical method sensitive to 5 parts per billion. The average level of BPA migration from the 14 cans food cans tested was 37 parts per billion.

Based on these results and using procedures recommended by the U.S. Food and Drug Administration for estimating exposures from food-contact applications (FDA, 2002), the estimated dietary intake of BPA from canned foods and beverages lined with epoxy coatings is less than 0.11 micrograms (0.00011 milligrams) per kilogram body weight per day (Howe *et al*, 1998).

In 2001, the UK Food Standards Agency (FSA) reported the results of a survey of BPA content in canned foods and beverages purchased in the UK (UK FSA, 2001; Goodson *et*

al, 2002). The levels of BPA found in foods and beverages are generally consistent with the results reported by SPI (Howe *et al*, 1998), which were measured in food simulating solvents. In all but one canned beverage, BPA was not detected at a limit of detection of 2 parts per billion, which is below the 5 parts per billion limit of detection in the SPI study. In most of the canned food samples, BPA, when detected, was found at levels comparable to or lower than the average level of 37 ppb for food cans reported in the SPI study. For all canned foods and beverages, the UK FSA estimated an average upper bound BPA level of 21.7 parts per billion. Using a different methodology that assumes higher consumption of canned foods and beverages than the FDA methodology, the UK FSA estimated a BPA dietary intake of 0.36 to 0.38 micrograms per kilogram body weight per day.

Step 4: Risk Characterization

The objective of the risk characterization step is to determine if the potential exposure to BPA, as estimated in the exposure assessment step, will result in any risk to human health.

Based on the results of migration studies and procedures recommended by the U.S. Food and Drug Administration, the estimated total dietary intake of BPA from polycarbonate food and beverage containers and from epoxy can coatings totals less than 0.118 micrograms (0.000118 milligrams) per kilogram of body weight per day. The total estimated dietary exposure to BPA from polycarbonate food and beverage containers and from epoxy can coatings is more than 400 times lower than the reference dose of 0.05 milligrams per kilogram body weight per day.

The Scientific Committee on Food (SCF), which is an independent advisory committee to the European Commission on food safety matters, has recently evaluated the safety of BPA from all food contact sources including polycarbonate plastic and epoxy resin coatings. The SCF set a Tolerable Daily Intake (TDI) for BPA of 0.01 milligrams per kilogram body weight per day after a comprehensive review of all robust scientific data covering all aspects of toxicity. Similar to the EPA reference dose, the TDI represents a lifetime exposure level that is considered to be safe. Based on the existing migration data, the total exposure to BPA from all food contact sources was estimated to be in the range of 0.00048 to 0.0016 milligrams per kilogram body weight per day for adults and infants respectively, which is below the TDI value set by the SCF. This assessment confirms that polycarbonate plastic and epoxy resin food contact applications are safe for use and pose no known risk to human health (SCF, 2002)

Conclusion

The potential human exposure to BPA is more than 400 times lower than the U.S. EPA reference dose. This minimal level of exposure to BPA poses no known risk to human health.

HUMAN SAFETY ASSESSMENT OF BISPHENOL A / page 9

The use of polycarbonate plastic and epoxy resins for food contact applications has been and continues to be recognized as safe by the U.S. Food and Drug Administration, the European Commission Scientific Committee on Food, the United Kingdom Food Standards Agency, the Japanese Ministry for Health, Labor and Welfare, and other regulatory authorities worldwide.

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HUMAN SAFETY ASSESSMENT OF BISPHENOL A / page 10

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