

Toxicant default guideline values for aquatic ecosystem protection

Bisphenol A in marine water

Technical brief July 2023

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Summary

The default guideline values (DGVs) and associated information in this technical brief should be used in accordance with the detailed guidance provided in the Australian and New Zealand Guidelines for Fresh and Marine Water Quality website (www.waterquality.gov.au/anz-guidelines).

Bisphenol A (BPA) is a widely used, high production volume industrial chemical. Major uses of BPA are as an intermediate compound in the manufacturing of polycarbonate plastic and epoxy resins, which are used as coatings to line the inside of food containers and beverage cans (Staples et al. 1998, ECB 2003, EC & HC 2008, OEHHA 2009, NCBI 2020).

In the environment, BPA chiefly partitions to water, with lesser amounts partitioning to soil and sediment (ECB 2003, OEHHA 2009). Following an initial lag period, biodegradation of BPA in water appears to be rapid (ECB 2003, EC & HC 2008, NCBI 2020). However, under anaerobic conditions, such as in anoxic or anaerobic sediment, BPA degradation can be slow, and long half-lives have been reported (Kang et al. 2007, EC & HC 2008).

With its widespread use, BPA has been detected in the environment in fresh, marine and estuarine surface water, groundwater, sediment, soil, leachates from landfill sites, and waste effluents from municipal and industrial waste treatment plants (EC & HC 2008, OEHHA 2009, Flint et al. 2012, NCBI 2020). Although BPA has been detected in fish, crabs, clams, mussels, squid and snails, it has a lowto-moderate potential to bioaccumulate in aquatic organisms (ECB 2003, Tsai 2006, EC & HC 2008).

BPA is a nonsteroidal xenoestrogen and endocrine disruptor that exhibits both oestradiol and antiandrogen activity (Kang et al. 2007, Flint et al. 2012). There is evidence that low level exposure to BPA, particularly at sensitive life cycle stages, can lead to permanent alterations in hormonal, developmental and reproductive capacities. Multigenerational effects of BPA exposure have been reported in fish and aquatic invertebrates (Sohoni et al. 2001, ECB 2003, Kang et al. 2007, EC & HC 2008, OEHHA 2009).

Low reliability default guideline values (DGVs) were derived using chronic EC5, EC10, LOEC and EC50 data and acute EC50 and LC50 data (converted to chronic negligible effect estimates) for eight species from four taxonomic groups, with a poor fit of the distribution to the toxicity data. The DGVs for 99%, 95%, 90% and 80% species protection are 0.04 μg/L, 0.63 µg/L, 2.2 μg/L, and 8.0 μg/L, respectively. However, some of the DGVs are below current analytical limits of reporting. Also, because the data span over four orders of magnitude, there is additional uncertainty in the DGVs, especially at the 99% species protection level. However, comparison of the DGVs with the available toxicity data indicated that the 95%, 90% and 80% species protection DGVs may not adequately protect some species.

If there are concerns that the DGV for a specific ecosystem condition and associated level of protection may not offer sufficient protection for key species (e.g. abalone) in the water body of interest, a conservative application of the DGVs may be warranted. For example, the 99% species protection DGV for BPA could be applied to a slightly-to-moderately disturbed ecosystem. As recommended in ANZG (2018), low reliability guideline values are typically not adequate to assess water quality, but can be used as interim values until more reliable values are derived. If used as interim values, they should always be used in conjunction with other lines of evidence.

1 Introduction

Bisphenol A (BPA) (CASRN 80-05-7), also known as 4,4'-isopropylidenediphenol, is a widely used, high production volume industrial chemical (ECB 2003, EC & HC 2008) with the chemical formula $(CH₃)₂C(C₆H₄OH)₂$. BPA is composed of two phenol rings connected by a methyl bridge, with two methyl functional groups attached to the bridge (Kang et al. 2007, NCBI 2020). Major uses of BPA are as:

- an intermediate compound in the manufacturing of polycarbonate plastic (used in a wide variety of products including water bottles)
- an intermediate compound in epoxy resins, which are used as coatings to line the inside of some food containers and beverage cans (Staples et al. 1998, ECB 2003, EC & HC 2008, OEHHA 2009, NCBI 2020).

Other products containing BPA include adhesives, powder paints, automotive lenses, protective window glazing, building materials, compact disks, optical lenses, thermal paper, and paper coatings. BPA is also produced through the biological reductive dehalogenation of tetrabromobisphenol A (TBBPA), a widely used brominated flame retardant (Kang et al. 2007, Flint et al. 2012).

The annual global production of BPA has increased significantly since the 1960s (Chen et al. 2002, Flint et al. 2012). In 2006, global production of BPA was reported to be 4 million tonnes, approximately one third of which was manufactured in the United States, and one quarter in Europe (Tsai 2006, EC & HC 2008). Global consumption of BPA in 2011 was predicted to exceed 5.5 million tonnes (Flint et al. 2012). BPA can enter the environment during production and processing, via various waste streams and spills, and during the use and disposal of products containing BPA. Flint et al. (2012) reported that, in 2008, over 500 tonnes of BPA was released to the environment from manufacture and processing, with another 1 300 tonnes released via incineration or wastewater treatment plants in the United States alone.

Under ambient conditions, BPA is a white solid, usually in the form of flakes or powder (ECB 2003, NCBI 2020). If released to air, a vapour pressure of 4.0x10⁻⁸ mmHg at 25°C indicates BPA will exist in both the vapour and particulate phases (NCBI 2020). BPA is short-lived in the atmosphere and is unlikely to be transported a long distance from its point of emission (ECB 2003).

In the environment, BPA mainly partitions to water, with lesser amounts partitioning to soil and sediment. Reported water solubility for BPA at ~25°C ranges from 120 mg/L to 300 mg/L, while reported log K_{oc} values range from 2.0 to 4.64 (ECB 2003, Tsai 2006, EC & HC 2008, NCBI 2020). In natural waters, BPA is not expected to volatilise, based on an estimated Henry's Law constant of 4.0 x 10^{-11} atm-m³/mol. As BPA lacks functional groups that hydrolyse under environmental conditions, it is not expected to undergo hydrolysis (ECB 2003, EC & HC 2008, NCBI 2020). Sensitised photo-oxidation may be an important fate process for BPA in sunlit natural waters (NCBI 2020).

Although some studies and screening tests show that BPA is non-biodegradable, other studies have found that BPA is readily biodegradable or inherently biodegradable. However, biodegradation appears to require an acclimation period to allow for the development of a microbial community capable of degrading BPA (ECB 2003, EC & HC 2008, NCBI 2020). In marine water under aerobic conditions, this acclimation period can be up to several weeks, after which degradation of BPA

appears to be rapid (Ying & Kookana 2003, Kang & Kondo 2005). Half-lives in surface water have been reported to range from 1 day to 15 days (ECB 2003, EC & HC 2008, NCBI 2020), with faster rates of photodegradation in the presence of dissolved organic matter and reactive oxygen species (Kang et al. 2007, OEHHA 2009). The process for BPA degradation in marine water may differ to that for freshwater. Some data indicate that BPA may persist longer in marine water than in freshwater, increasing the potential for adverse effects to occur (Sajiki & Yonekubo 2003, Kang & Kondo 2005). Under anaerobic conditions in natural water (including groundwater), little or no biodegradation of BPA may occur (Ying et al. 2003, Kang et al. 2007, EC & HC 2008). In anoxic estuarine sediment, BPA has been reported to be resistant to degradation (Voordeckers et al. 2002). Therefore, there is potential for accumulation of BPA in anoxic sediment pore water.

Primary biodegradation of BPA in an activated sludge treatment system with acclimated microbial populations has been reported to remove up to 99% of the BPA, although reduction rates in sewage treatment plants range from <1% to 99%, depending on whether secondary treatment is used (EC & HC 2008, NCBI 2020). The range in reduction rates likely reflects whether microbial organisms are acclimated to BPA. Major degradation products of BPA include 4-hydroxyacephenone and 4 hydroxybenzoic acid, which rapidly degrade to carbon dioxide and water (NCBI 2020). Although BPA can be rapidly degraded in biological waste treatment systems, detectable concentrations of BPA have been found in wastewater due to incomplete BPA removal during treatment from paper and plastic production plants and domestic sewage treatment plants (Kang et al. 2007, EC & HC 2008).

The primary route of BPA contamination to the aquatic environment is via effluent from wastewater treatment plants and leaching from landfill sites (Kang et al. 2007, EC & HC 2008). BPA has been detected in fresh, marine and estuarine surface water, sediment, groundwater and soil, and in municipal and industrial waste treatment streams (Crain et al. 2007, EC & HC 2008, OEHHA 2009, Flint et al. 2012, NCBI 2020). In fresh surface water, concentrations of BPA range from below the limits of reporting to 21 µg/L, although most concentrations were reported below 0.5 µg/L (ECB 2003, Tsai 2006, Kang et al. 2007, OEHHA 2009, NCBI 2020). There are fewer data on BPA concentrations in marine and estuarine systems. Where data were available, these indicate generally lower concentrations than in freshwater systems (Tsai 2006, Crain et al. 2007, OEHHA 2009). Reported concentrations of BPA in various media are as follows:

- up to 2.47 µg/L, with most concentrations at or below 0.2 µg/L, in marine water (Tsai 2006, Crain et al. 2007, OEHHA 2009)
- from <0.5 µg/kg to 1 630 µg/kg in freshwater sediment (ECB 2003, Kang et al. 2007)
- from <0.5 µg/kg to 53 µg/kg marine sediment (Tsai 2006)
- from 15 µg/L to 5 400 µg/L prior to treatment, and from 0.5 µg/L to 5.1 µg/L after treatment, in leachate from landfills (Kang et al. 2007).

BPA has a low-to-moderate potential to bioaccumulate in aquatic organisms, with log K_{OW} values ranging from 2.2 to 4.16 (ECB 2003, Tsai 2006, EC & HC 2008). Bioconcentration factors (BCFs) in marine and freshwater fish have been reported as 3.5–5.5 L/kg for rainbow trout (*Oncorhynchus mykiss*), 67.7 L/kg for carp (*Cyprinus carpio*), 73.4 L/kg for medaka (*Oryzias latipes*) and 38 L/kg for spotted halibut (*Varaspar variegates*) (ECB 2003, EC & HC 2008, NCBI 2020). Higher BCFs of 94– 182 L/kg have been reported for salmon (*Salmo salar* m. *sebago*) yolk-sac fry, suggesting greater accumulation of BPA in early life stages (Honkanen et al. 2004). In freshwater clams (*Pisidium*

amnicum) and frogs (*Rana temporaria*), BCFs of 110–144 L/kg and 131–147 L/kg, respectively, have been reported (ECB 2003, EC & HC 2008). Concentrations of BPA in freshwater biota have been reported up to 0.075 mg/kg (dry weight) in fish liver and 0.011 mg/kg in snails (OEHHA 2009). In marine biota (prawns, crabs, molluscs, squid and fish), concentrations up to 0.213 mg/kg have been reported (OEHHA 2009).

2 Aquatic toxicology

2.1 Mechanism of toxicity

BPA is a nonsteroidal xenoestrogen and endocrine disruptor that exhibits both oestradiol and antiandrogen activity in aquatic organisms following chronic exposures. BPA also has an active, but poorly understood, involvement in steroidal sex hormones in plant development and growth processes (Speranza 2010). Thus, the mode of action of BPA is known to affect both plants and animals (Speranza 2010).

2.2 Toxicity

A literature review of the effects of BPA on freshwater and marine organisms indicated less extensive research has been undertaken on marine organisms compared to freshwater organisms. The following summarises the effects of BPA that have been observed in aquatic organisms in either marine water or freshwater.

- Effects reported for fish include: inhibition of gonadal growth in males and females, vitellogenin induction, induction of apoptosis in testis cells, inhibition of spermatogenesis and reduced percentage of spermatocytes, embryonic deformities, and intersex.
- Effects reported for invertebrates include: premature metamorphosis of larvae, developmental inhibition, delayed larval emergence, altered sex ratios, reduced feeding behaviour, superfeminisation and imposex, oviduct rupture and morphological deformities (Kang et al. 2007, OEHHA 2009, Flint et al. 2012).

BPA is acutely toxic to aquatic organisms and adversely affects growth and development (Chen et al. 2002, Kang et al. 2007, EC & HC 2008, NCBI 2020). There is evidence that low level exposure to BPA, particularly at sensitive life stages, can lead to permanent alterations in hormonal, developmental or reproductive capacities (ECB 2003, EC & HC 2008). These data indicate that endocrine disruption may be the most sensitive endpoint of BPA, with many of the lowest effect concentrations for reproductive endpoints (e.g. vitellogenin induction, gonad development, sex ratios) occurring in the range of 1 µg/L to 1 mg/L in fish, aquatic invertebrates and frogs (Sohoni et al. 2001, ECB 2003, Kang et al. 2007, EC & HC 2008, OEHHA 2009). Vitellogenin is a precursor of egg-yolk proteins, and vitellogenin induction is one of the most widely studied biomarkers of BPA exposure (Kang et al. 2007).

As with other compounds that affect reproductive hormones, BPA can produce adverse effects following prolonged exposure at levels below those that usually elicit effects in standard toxicity tests (i.e. tests based on recognised methods that evaluate endpoints such as survival, reproduction and growth). Effects can also be apparent later in the life cycle following brief, low dose exposure at sensitive developmental stages, and on filial generations following parental exposure (EC & HC 2008). The marine acute toxicity data available were limited to macroinvertebrates and microinvertebrates. Macroinvertebrates were the most and least sensitive organisms, with a 12 hour EC50 of 30.7 μg/L for embryo development in the mollusc *Haliotis diversicolor supertexta* (Liu et al. 2011) and a 15 minute NOEC of 99 864 μg/L for fertilisation success in the sea urchin *Psammechinus miliaris* (Schafer et al. 2009). A single acute fish study (4 day LC50 for larvae of *Menidia menidia*) was identified (Alexander & Dill 1988); however, the reported salinity concentration was outside the defined range for marine studies, as specified in Warne et al. (2018).

Of the marine chronic toxicity data available, macroinvertebrates were the most sensitive species and dinoflagellates (*Prorocentrum cordatum* and *Margalefidinium polykrikoides*) were the least sensitive. Chronic toxicity values for macroinvertebrates ranged from a 25 day dietary exposure LOEC of 0.005 μg/L for the lobster *Homarus americanus* (Laufer et al. 2012) to a 4 day EC50 of 226.5 μg/L for the sea urchin, *Strongylocentrotus purpuratus* (Roepke et al. 2005). As the toxicity value for *H. americanus* was based on dietary exposure, it was not used in the DGV derivation. The next most sensitive species was the mollusc, *H. diversicolor supertexta*, with 96 hour embryo development EC5 values of 0.18 μg/L and 0.21 μg/L (Liu et al. 2011). The chronic toxicity of BPA to dinoflagellate species was approximately four orders of magnitude higher (i.e. less toxic); for example, 1 510 µg/L for *P. cordatum* (EC50, 3 day growth), and 3 470 µg/L for *M. polykrikoides* (EC10, 3 day growth). Chronic toxicity data for marine fish were limited to a 14 day LOEC of 200 μ g/L for body length and width in the medaka *Oryzias melastigma*; however, the reliability of this result is uncertain because only one exposure concentration was used, and no dose–response relationship was obtained (Huang et al. 2012).

There are no published multigenerational studies assessing the toxicity of BPA to marine species.

3 Factors affecting toxicity

Data indicate that BPA may persist longer in marine water compared to freshwater (Sajiki & Yonekubo 2003, Kang & Kondo 2005), which may have an influence on its toxicity. The available toxicity data suggest that BPA may be more toxic to some marine species compared to freshwater species. Effects for several marine species have been reported at concentrations below 1 µg/L (Marcial et al. 2003, Liu et al. 2011, Laufer et al. 2012), whereas there have been no effects for freshwater species reported at concentrations below 1 μ g/L (ECCC 2017). This is despite the fact that the marine toxicity data are based on shorter duration, partial life cycle studies (albeit for early life stages), whereas the freshwater data are mostly based on studies of chronic duration, including multigenerational exposures. Currently, there is no empirical evidence of abiotic factors, such as salinity, affecting the toxicity of BPA, and more data are required to determine whether BPA is generally more toxic to marine species than to freshwater species and whether there are any key toxicity modifying factors.

4 Default guideline value derivation

The DGVs were derived in accordance with the method described in Warne et al. (2018) and using Burrlioz 2.0 software.

4.1 Toxicity data used in derivation

The literature review and quality assessment and screening process identified permissible marine toxicity data consisting of 11 chronic toxicity values for six species and eight acute toxicity values for four species. These data were within the salinity range for establishing DGVs for marine water as specified by Warne et al. (2018).

A summary of the toxicity data (one value per species) and conversions used to calculate the DGVs for BPA in marine water is provided in **Error! Reference source not found.**. Further details on the data that passed the screening and quality assessment, including those used to derive the single species values used to calculate the DGVs, are presented in Appendix [A: Toxicity data that passed the](#page-15-1) screening and quality assessment [and were used to derive the default guideline values,](#page-15-1) [Table](#page-15-0) A 1. Details of the [data quality assessment](http://www.waterquality.gov.au/sites/default/files/documents/BPA-marine-DGVs-data-quality-assessment.xlsm) and th[e data that passed the quality assessment](http://www.waterquality.gov.au/sites/default/files/documents/BPA-marine-DGVs-data%20entry.xlsx) are provided as supporting information.

Table 1 Summary of single toxicity values, all species used to derive the default guideline values for BPA in marine water

Note: estimated chronic values are reported to no more than three significant figures.

a The measure of toxicity being estimated/determined: EC/LCx: x% effect/lethal concentration; LOEC: lowest observed effect concentration.

b Default conversion from chronic EC50 to chronic NOEC: chronic LC50 ÷ 5 = chronic NOEC.

c Actual chronic NOEC/EC10 or EC5.

d Default conversion from chronic LOEC to chronic NOEC: chronic LOEC ÷ 2.5 = chronic NOEC.

e Default conversion from acute EC/LC50 to chronic NOEC: acute EC/LC50 ÷ 10 = chronic NOEC.

f Value is the geometric mean of 96 hour EC5 values of 0.18 and 0.21 µg/L.

Studies that reported salinity within the range of 25–36‰, or that did not report salinity but used synthetic or natural marine water, were considered representative of marine conditions. Studies with salinity outside of the range of 25–36‰ were excluded from the derivation. This resulted in the exclusion of development and survival studies for the copepod *Acartia tonsa* (salinity 18‰)

(Andersen et al. 1999), the microalga *Skeletonema costatum*, the mysid *Mysidopsis bahia*, and the fish *Menidia* (salinity 20‰) (Alexander et al. 1988).

Studies with a >10-fold difference in the exposure concentrations were excluded from the DGV derivation. This resulted in the exclusion of development endpoints for the copepod *Tigriopus japonicus* (Marcial et al. 2003) and fertilisation success endpoints for the sea urchin *P. miliaris* (Schafer et al. 2009). In addition, endpoints for growth in the fish *O. melastigma* from Huang et al. (2012) were excluded, as the experiment only considered one exposure concentration and, therefore, a dose–response could not be obtained.

Where only one acceptable toxicity value was available for a species, that value was selected for the final dataset used to calculate the DGVs. For species with more than one acceptable toxicity value available, the value selected for the final dataset was in accordance with Warne et al. (2018). Overall, eight species were considered for the final dataset. These species included: three sea urchins, two dinoflagellates, two crustaceans, and one mollusc (abalone). The toxicity data used for these eight species comprised one chronic LOEC value, two chronic EC50 values, one chronic EC10 value, one geometric mean calculated from two EC5 values, and three acute EC/LC50 values. The toxicity data for these species spanned over four orders of magnitude. Chronic toxicity data were available for five species from three taxonomic groups—one taxonomic group short of the minimum requirement for using the species sensitivity distribution (SSD) method (Warne et al. 2018). Therefore, the dataset needed to be supplemented with acute toxicity data (converted to chronic estimates) for the two crustacean species to attain the minimum taxonomic representation of four taxonomic groups. Also, although chronic toxicity data were available for the sea urchin *P. lividis*, an acute EC50 of 388 µg/L (converted to 38.8 µg/L) was used instead. The acute EC50 was similar to the lowest reliable no/low effect estimate of 362 µg/L (EC5, embryo development) (Özlem & Hatice 2008), but resulted in a more conservative value once converted to a chronic no/low effect equivalent. This was considered appropriate because data from Özlem and Hatice (2008) suggested effects on egg fertilisation and embryos could occur at concentrations below the chronic EC5 of 362 µg/L.

Modality checks on the dataset were performed according to the method stipulated in Warne et al. (2018), with the details of the assessment provided in Appendix [B: Modality assessment for](#page-16-0) [bisphenol A.](#page-16-0) The weight of evidence assessment indicated that there were insufficient data to determine if the dataset was bimodal or multimodal and, hence, it supported use of the data for eight species for derivation of the DGVs.

4.2 Species sensitivity distribution

The cumulative frequency (species sensitivity) distribution (SSD) of the eight acute (converted) and chronic BPA marine water toxicity data reported in **Error! Reference source not found.** is shown in [Figure](#page-11-0) 1. The model was judged to provide a poor fit to the data, specifically at the lower and upper tails of the distribution. The implications of the poor fit of the lower tail are addressed in Section [4.3.](#page-11-1)

4.3 Default guideline values

It is important that the DGVs [\(Table](#page-11-2) 2) and associated information in this technical brief are used in accordance with the detailed guidance provided in the Australian and New Zealand Guidelines for Fresh and Marine Water Quality website (ANZG 2018).

The DGVs for 99%, 95%, 90% and 80% species protection are shown i[n Table](#page-11-2) 2. Some of the DGVs may be below current analytical limits of reporting for BPA. However, the available toxicity data indicate that toxic effects can occur below the current limits of reporting. ANZG (2018) (see [Accounting for local conditions\)](https://www.waterquality.gov.au/anz-guidelines/guideline-values/default/water-quality-toxicants/local-conditions#default-guideline-values-and-analytical-detection-limits) provides guidance on what to do if guideline values are below analytical detection limits.

a The DGVs were derived using the Burrlioz 2.0 software and rounded to either one or two significant figures.

The DGVs were compared to the permissible chronic and acute toxicity data (that had been converted where necessary to represent chronic negligible effect data) (i.e. 19 values for eight species). The DGVs for 99%, 95%, 90% and 80% species protection were protective of (i.e. lower than) 100%, 76%, 76% and 65% of the toxicity values, respectively, indicating that the theoretical protection offered by the 95%, 90% and 80% species protection DGVs may be inadequate. This is reflective of the poor model fit at the lower tail of the SSD [\(Figure](#page-11-0) 1). However, all the values that exceeded the 95% and 90% species protection DGVs were from one species, the abalone *H. diversicolor supertexta*, which was two orders of magnitude more sensitive than the next most sensitive species, the copepod *T. japonicus* and the three echinoderms (**Error! Reference source not found.**, [Figure](#page-11-0) 1). If there are concerns that the DGV for a specific ecosystem condition and associated level of protection may not offer sufficient protection for key species (e.g. abalone) in the water body of interest, a conservative application of the DGVs may be warranted. For example, the 99% species protection DGV of 0.04 µg/L could be applied to a slightly-to-moderately disturbed ecosystem (also see additional guidance on DGV application in Section [4.4\)](#page-12-0).

4.4 Reliability classification

The BPA marine water DGVs have a low reliability classification (Warne et al. 2018) based on the outcomes for the following three criteria:

- Sample size—eight (good)
- Type of toxicity data—chronic and converted acute
- SSD model fit-poor (Burr Type III model).

As recommended in ANZG (2018), low reliability guideline values are typically not adequate to assess water quality, but can be used as interim values until more reliable values are derived. If used as interim values, they should always be used in conjunction with other lines of evidence. Moreover, the BPA toxicity data span over four orders of magnitude, which typically increases the uncertainty in the DGVs, especially at the 99% species protection level. Consequently, the 99% species protection DGV should be treated with additional caution.

Glossary

Appendix A: Toxicity data that passed the screening and quality assessment and were used to derive the default guideline values

Table A 1 Summary, toxicity data that passed the screening and quality assurance processes, BPA in marine water

a The measure of toxicity being estimated/determined: EC/LCx: x% effect/lethal concentration; LOEC: lowest observed effect concentration

b Values used as reported here for the DGV derivation unless otherwise indicated.

c For the DGV derivation, reported chronic EC50 value was divided by default conversion factor of 5, as per Warne et al. (2018).

d For the DGV derivation, reported chronic LOEC value was divided by default conversion factor of 2.5, as per Warne et al. (2018).

e For the DGV derivation, reported acute EC/LC50 value was divided by default conversion factor of 10, as per Warne et al. (2018).

f A geometric mean of 0.19 µg/L was estimated from the 0.18 µg/L and 0.21 µg/L EC5 values at 96 hour exposure for *Haliotis diversicolor supertexta.*

Appendix B: Modality assessment for bisphenol A

A modality assessment was undertaken for BPA toxicity to marine species according to the four questions stipulated in Warne et al. (2018). These questions and their answers are listed below.

Is there a specific mode of action that could result in taxa-specific sensitivity?

BPA is a nonsteroidal xenoestrogen and endocrine disruptor that exhibits both oestradiol and antiandrogen activity in aquatic organisms following chronic exposures. BPA also has an active, but poorly understood, involvement in steroidal sex hormones in plant development and growth processes (Speranza 2010). Therefore, based on mode of action alone, there was no clear reason to suspect large differences in taxa-specific sensitivity.

Does the dataset suggest bimodality?

Visual representation of the data, calculation of the bimodality coefficient (BC), and consideration of the range in the effect concentrations are recommended lines of evidence for evaluating whether bimodality or multimodality of the dataset is apparent. This is discussed as follows.

- The histogram of the raw effect concentration SSD data [\(Figure](#page-16-1) B 1) could be interpreted as positively right skewed, typical of concentration-based data (Warne et al. 2018). The logtransformed histogram generally follows a normal distribution [\(Figure](#page-16-1) B 1).
- Data that span large ranges (>4 orders of magnitude) indicate potential for underlying bimodality or multimodality (Warne et al. 2018); the BPA data span over four orders of magnitude.
- When the BC is greater than 0.555, it indicates that the data do not follow a normal distribution and may be bimodal; the BC of the log-transformed data is 0.22, which does not support bimodality.

Based on these lines of evidence, the distribution of the log-transformed dataset is generally in accordance with a unimodal distribution.

Figure B 1 Histogram, raw (left) and log-transformed (right) data

Do data show taxa-specific sensitivity (i.e. through distinct groupings of different taxa types)? The mode of action of BPA is known to affect both plants and animals (Speranza 2010); the potential for taxa-specific sensitivity in the data was examined using box plots of the SSD data with the grouping variable phyla and major organism types (noting that no plant data were identified for this DGV derivation).

As shown in [Figure](#page-17-0) B 2, echinoderms and molluscs appear to be the most sensitive. However, as there are only three echinoderms and one mollusc, it is difficult to draw robust conclusions.

Organisms considered to be taxonomically different

Organisms considered to be taxonomically different

Figure B 2 Box plots, raw (left) and log-transformed (right) data grouped by organisms considered to be taxonomically different

Is it likely that indications of bimodality or multimodality or distinct clustering of taxa groups are not due to artefacts of data selection, small sample size, test procedures, or other reasons unrelated to a specific mode of action?

Due to the small sample size, it is not possible to discern trends in the data and if such trends are artefacts of data selection, test procedures, or other reasons unrelated to a specific mode of action. The weight of evidence is insufficient to support a conclusion of bimodality or multimodality; therefore, all eight species identified in preparation of the SSD were retained for the final dataset.

References

Alexander, HC & Dill, DC 1988. Bisphenol A: Acute aquatic toxicity. *Environmental Toxicology and Chemistry*, 7, 19–26.

Andersen, HR, Halling-Sorensen, B & Kusk, KO 1999. A parameter for detecting estrogenic exposure in the copepod *Acartia tonsa*. *Ecotoxicology and Environmental Safety*, 44, 56–61.

ANZG 2018. Australian and New Zealand Guidelines for Fresh and Marine Water Quality. Australian and New Zealand Governments and Australian state and territory governments, Canberra, ACT, Australia, [https://www.waterquality.gov.au/anz-guidelines.](https://www.waterquality.gov.au/anz-guidelines)

Bosnjak, I, Borra, M, Iamunno, F, Benvenuto, G, Ujevic, I, Buselic, I, Roje-Busatto, R & Mladineo, I 2014. Effect of bisphenol A on P-glycoprotein-mediated efflux and ultrastructure of the sea urchin embryo. *Aquatic Toxicology*, 156, 21–29.

Chen, M-Y, Michihiko, I & Masanori, F 2002. Acute toxicity, mutagenicity, and estrogenicity of bisphenol-A and other bisphenols. *Environmental Toxicology*, 17, 1, 80–86.

Crain, DA, Eriksen, M, Iguchi, T, Jobling, S, Laufer, H, LeBlanc, GA & Guillette Jr, LJ 2007. An ecological assessment of bisphenol-A: Evidence from comparative biology. *Reproductive Toxicology*, 24, 225– 239.

Ebenezer, V & Ki, J-S 2012. Evaluation of the sub-lethal toxicity of Cu, Pb, bisphenol A and polychlorinated biphenyl to the marine dinoflagellate *Cochlodinium polykrikoides*. *Algae*, 27, 1, 63– 70.

EC & HC 2008. Screening Assessment for the Challenge. Phenol, 4,4'-(1-methylethylidene)bis- (Bisphenol A). Chemical Abstracts Service Registry Number 80-05-7. Environment Canada and Health Canada, October 2008.

ECB 2003. European Union Risk Assessment Report. Bisphenol A (CAS No. 80-05-7). European

ECB 2003. [European Union Risk Assessment Report. Bisphenol A. CAS No. 80-05-7. EINECS No. 201-](https://ec.europa.eu/jrc/en/publication/eur-scientific-and-technical-research-reports/european-union-risk-assessment-report-bisphenol-cas-no-80-05-7-einecs-no-201-245-8) [245-8.](https://ec.europa.eu/jrc/en/publication/eur-scientific-and-technical-research-reports/european-union-risk-assessment-report-bisphenol-cas-no-80-05-7-einecs-no-201-245-8) European Chemicals Bureau. Accessed September 2015.

ECCC 2017. [Canadian Environmental Protection Act, 1999: Federal Environmental Quality Guidelines,](https://www.canada.ca/en/environment-climate-change/services/evaluating-existing-substances/federal-environmental-quality-guidelines-bisphenol-a.html) [Bisphenol A.](https://www.canada.ca/en/environment-climate-change/services/evaluating-existing-substances/federal-environmental-quality-guidelines-bisphenol-a.html) Environment and Climate Change Canada.

Flint, S, Markle, T, Thompson, S & Wallace, E 2012. Bisphenol A exposure, effects, and policy: A wildlife perspective. *Journal of Environmental Management*, 104, 19–34.

Guo, R, Ebenezer, V & Ki, J-S 2012. Transcriptional responses of heat shock protein 70 (Hsp70) to thermal, bisphenol A and copper stresses in the dinoflagellate *Prorocentrum minimum*. *Chemosphere*, 89, 512–520.

Hirano, M, Ishibashi, H, Matsumura, N, Nagao, Y, Watanabe, N, Watanabe, A, Onikura, N, Kishi, K & Arizono, K 2004. Acute toxicity responses of two crustaceans, *Americamysis bahia* and *Daphnia magna*, to endocrine disruptors. *Journal of Health Science*, 50, 97–100.

Honkanen, JO, Holopainen, IJ & Kukkonen, JVK 2004. Bisphenol A induces yolk-sac oedema and other adverse effects in landlocked salmon (*Salmo salar* m. *sebago*) yolk-sac fry. *Chemosphere*, 55, 187– 196.

Huang, Q, Fang, C, Chen, Y, Wu, X, Ye, T, Lin, Y & Dong, S 2012. Embryonic exposure to low concentration of bisphenol A affects the development of *Oryzias melastigma* larvae. *Environmental Science and Pollution Research*, 19, 2506–2514.

Kang, J-H & Kondo, F 2005. Bisphenol A degradation in seawater is different from that in river water. *Chemosphere*, 60, 1288–1292.

Kang, J-H, Aasi, D & Katayama, Y 2007. Bisphenol A in the aquatic environment and its endocrinedisruptive effects on aquatic organisms. *Critical Reviews in Toxicology*, 37, 607–625.

Kiyomoto, M, Kikuchi, A, Unuma, T & Yokota, Y 2006. Effects of ethynylestradiol and bisphenol A on the development of sea urchin embryos and juveniles. *Marine Biology*, 149, 57–63.

Laufer, H, Baclaski, B & Koehn, U 2012. Alkylphenols affect lobster (*Homarus americanus*) larval survival, molting and metamorphosis. *Invertebrate Reproduction and Development*, 56, 1, 66–71.

Lee, K-W, Raisuddin, S, Hwang, D-S, Park, HG & Lee, J-S 2007. Acute toxicities of trace metals and common xenobiotics to the marine copepod *Tigriopus japonicus*: Evaluation of its use as a benchmark species for routine ecotoxicity tests in Western Pacific coastal regions. *Environmental Toxicology*, 22, 532–538.

Liu,Y, Tam, NFY, Guan, Y, Yasojima, M, Zhou, J & Gao, B 2011. Acute toxicity of nonylphenols and bisphenol A to the embryonic development of the abalone *Haliotis diversicolor supertexta*. *Ecotoxicology*, 20, 6, 1233–1245.

Marcial, HS, Hagiwara, A & Snell, TW 2003. Estrogenic compounds affect development of harpacticoid copepod *Tigriopus japonicus*. *Environmental Toxicology and Chemistry*, 22, 12, 3025– 3030.

NCBI 2020. PubChem Compound Summary for CID 6623, Bisphenol A. PubChem database, National Center for Biotechnology Information, Bethesda, MD, USA.

OEHHA 2009[. Toxicological Profile for Bisphenol A.](http://www.opc.ca.gov/webmaster/ftp/project_pages/MarineDebris_OEHHA_ToxProfiles/Bisphenol%20A%20Final.pdf) Office of Environmental Health Hazard Assessment, California Environmental Protection Agency.

Özlem, ÇA & Hatice, P 2008. Effects of bisphenol A on the embryonic development of sea urchin (*Paracentrotus lividus*). *Environmental Toxicology*, 23, 387–392.

Roepke, TA, Snyder, MJ & Chen, GN 2005. Estradiol and endocrine disrupting compounds adversely affect development of sea urchin embryos at environmentally relevant concentrations. *Aquatic Toxicology*, 71, 155–173.

Sajiki, J & Yonekubo, J 2003. Leaching of bisphenol A (BPA) to seawater from polycarbonate plastic and its degradation by reactive oxygen species. Chemosphere, 51, 55–62.

Schafer, S, Bickmeyer, U & Koehler, A 2009. Measuring Ca²⁺-signalling at fertilization in the sea urchin Psammechinus miliaris: Alterations of this Ca²⁺-signal by copper and 2,4,6-tribromophenol. *Comp. Biochem. Physiol. Part C: Toxicol. Pharmacol.*, 150, 2, 261–269.

Sohoni, P, Tyler, CR, Hurd, K, Caunter, J, Hetheridge, M, Williams, T, Woods, C, Evans, M, Toy, R, Gargas, M & Sumpter, JP 2001. Reproductive effects of long term exposure to bisphenol A in the fathead minnow (*Pimephales promelas*). *Environmental Science and Technology*, 35, 14, 2917–2925.

Speranza, A 2010. Into the world of steroids. *Plant Signaling & Behavior*, 5, 8, 940–943.

Staples, CA, Dome, PB, Klecks, GM, O'Block, ST & Harris, LR 1998. A review of the environmental fate, effects and exposures of bisphenol A. *Chemosphere*, 36, 10, 2149–2173.

Tsai, W-T 2006. Human health risk on environmental exposure to bisphenol-A: A review. *Journal of Environmental Science and Health, Part C: Environmental Carcinogenesis and Ecotoxicology Reviews*, 24, 2,225–255.

Voordeckers, JW, Fennell, DE, Jones, K & Häggblom, MM 2002. Anaerobic biotransformation of tetrabromobisphenol A, tetrachlorobisphenol A, and bisphenol A in estuarine sediments. *Environmental Science and Technology*, 36, 696–701.

Warne, MStJ, Batley, GE, van Dam, RA, Chapman, JC, Fox, DR, Hickey, CW & Stauber, JL 2018. Revised Method for Deriving Australian and New Zealand Water Quality Guideline Values for Toxicants – update of 2015 version. Prepared for the revision of the Australian and New Zealand Guidelines for Fresh and Marine Water Quality. Australian and New Zealand Governments and Australian state and territory governments, Canberra, 48 pp.

Ying, G-G & Kookana, RS 2003. Degradation of five selected endocrine-disrupting chemicals in seawater and marine sediment. *Environmental Science and Technology*, 37, 1256–1260.

Ying, G-G, Kookana, RS & Dillon, P 2003. Sorption and degradation of selected five endocrine disrupting chemicals in aquifer material. *Water Research*, 37, 3785–3791.