

# PART 1 - Scientific Assessment

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# 1. Introduction

## 1.1 Declaration

Triclosan (phenol, 5-chloro-2-(2,4-dichlorophenoxy)-), CAS No 3380-34-5, was declared a Priority Existing Chemical for a full risk assessment under the *Industrial Chemicals (Notification and Assessment) Act 1989* (Cwlth) (the Act) by notice in the *Commonwealth Chemical Gazette* of 6 May 2003. The basis for the declaration was that triclosan has been shown, in laboratory studies, to be toxic to aquatic species, particularly to algae, which is the most sensitive species. The widespread use of triclosan in consumer products provides a number of pathways for the chemical to enter the environment. In addition, the chemical properties of triclosan indicate that it may be bioaccumulative and persistent in the environment. There are also reports that suggest incineration of textile products containing triclosan may result in the formation of dioxin-like substances.

## 1.2 Objectives

The objectives of this assessment are to:

- identify the extent of use of triclosan;
- characterise the human health hazards and environmental effects of triclosan;
- determine the potential occupational, public and environmental exposure to triclosan;
- determine the risk of adverse effects to the environment, workers and the general public resulting from exposure to triclosan; and
- make recommendations for minimizing environmental, occupational and public health risks, and for appropriate hazard communication measures, where applicable.

## 1.3 Sources of information

Consistent with these objectives, the report presents an extensive and critical evaluation of relevant information relating to the potential human health effects and environmental effects from exposure to triclosan.

Importers of triclosan and triclosan-containing products and some formulators provided relevant scientific data, including information on quantities imported into Australia, uses, physicochemical properties, human and environmental exposure, transport, handling, storage, manufacture and disposal, and toxicity (published and unpublished data). Information was also obtained from published papers identified in a comprehensive literature search of several online databases up to December 2006, and retrieved from other sources such as the 2002 review of triclosan antimicrobial resistance undertaken by the European Union (EU) Scientific Steering Committee (European Commission Health & Consumer Protection Directorate General, 2002b). With the exception of the studies contained in this

overseas report on antimicrobial activity, all primary sources of data were evaluated.

Data provided by applicants indicated no manufacture of triclosan occurs in Australia.

Quantities of triclosan in pesticides and veterinary medicines and therapeutic products were collected for this assessment, but no further assessment of these products were conducted as they are not within the scope of the ICNA Act. The Australian Pesticides and Veterinary Medicines Authority (APVMA) provided the information on pesticides and veterinary medicines containing triclosan imported into Australia and triclosan imported into Australia as raw material to be used in pesticide and veterinary medicine manufacture. Quantities of triclosan used for therapeutic purposes were identified in a survey undertaken by NICNAS in 2004 of registrants listed on the Australian Register of Therapeutic Goods (ARTG) as sponsors of products containing triclosan. Of the 30 registrants contacted 27 responded to the initial survey for import data for the calendar years 2001, 2002 and 2003. In contrast, 11 registrants responded to an updated survey for the calendar years 2004 and 2005 that was conducted in 2006.

Additionally, a telephone survey of companies using triclosan for industrial purposes in the textile and plastics industry was conducted in 2004. Four companies using triclosan to treat plastics and 11 companies using triclosan in textile treatments provided information.

The information obtained on industrial uses of triclosan together with that obtained on therapeutic, pesticide and veterinary uses resulted in a more accurate estimation of the total environmental load of triclosan in Australia and, thus, environmental risk assessment. The characterisation of health risks in Australia was based upon information on toxicology data, product specifications made by the applicants, and overseas use patterns and occupational exposure models.

Information to assist in this assessment was also obtained through site visits to workplaces involved in formulating triclosan into personal care and therapeutic products, and a workplace using triclosan for textile treatment. This assessment did not take into account any triclosan imported as part of finished plastic and textile articles, as no information was provided on the triclosan quantities in imported articles.

Additionally, NICNAS commissioned two projects for this assessment:

1. Determination of triclosan levels in national breast milk samples, which was undertaken by the National Centre for Environmental Toxicology, at the University of Queensland.
2. Evaluation, by a national expert on antimicrobial resistance, of studies on this issue published in scientific journals from 2002 to December 2005. These studies have been published since the 2002 EU review of antimicrobial resistance (European Commission Health & Consumer Protection Directorate General, 2002b).

## **1.4 Peer review**

During all stages of preparation, the report has been subject to internal peer review by NICNAS and the Australian Government Department of the Environment, Water, Heritage and the Arts (DEWHA). In addition, the Advisory Group on Chemical Safety peer reviewed the sections of the report describing the kinetics and metabolism of triclosan along with the models used to estimate occupational and public exposure, the methodology used for the human health risk characterisation, the NICNAS commissioned study to determine triclosan levels in national breast milk samples, and the potential for triclosan in breast milk to cause harm to breast-fed babies. The environmental sections of the report were peer reviewed overseas by Dr Helen Wilkinson of the United Kingdom Environmental Agency.

## **1.5 Report structure**

Part 1 of this report is the scientific assessment of the available data on triclosan. It examines the manufacture, importation and use of triclosan, the potential human and environmental exposure, and the potential human and environmental hazards associated with triclosan. It also characterizes the human and environmental risks associated with the use of triclosan and provides information on the current risk management practices.

Part 2 of the report contains detailed data which support the scientific assessment of Part 1.

## 2. Background

### 2.1 International perspective

The first US patent for triclosan was in 1964 (Merck, 1983) and triclosan has been marketed for over 30 years. The chemical is listed on the Organisation for Economic Cooperation and Development's (OECD) High Production Volume Chemicals list (OECD, 2004) and is being sponsored through the OECD SIDS program by Australia.

Triclosan is not listed under the Rotterdam Convention on the Prior Informed Consent Procedure for Certain Hazardous Chemicals and Pesticides in International Trade (Rotterdam Convention, Annex III, 2006). The Convention enables listed hazardous chemicals to be monitored and their trade controlled on a global scale. Triclosan is not listed under the Stockholm Convention on Persistent Organic Pollutants (UNEP, 2005). The Convention is an international treaty that Australia has ratified, and is aimed at restricting and ultimately eliminating the production, use, release and storage of persistent organic pollutants (POPs). Additionally, the World Health Organisation (WHO) has not set guidance values for triclosan levels in drinking water (WHO, 2006).

In the European Union the Cosmetics Directive (76/768/EEC) has set a maximum allowable concentration of 0.3% triclosan in cosmetic products (European Commission, 1999). In Japan, triclosan is included in the Standards for Cosmetics (as established by the Pharmaceutical Affairs Law, 1960), which sets a maximum allowable concentration of 0.1% triclosan in cosmetic products (Ministry of Health and Welfare Notification No. 331, 2000).

In Canada the use of chemicals in cosmetics is regulated via section 16 (Cosmetic Regulations) of the *Food and Drugs Act 1985*. Maximum allowable concentrations of 0.03% triclosan in mouthwash and 0.3% triclosan in other cosmetic products are allowed. In addition for all triclosan containing products, the concentration of the impurities 2,3,7,8-tetra-chlorodibenzo-p-dioxin and 2,3,7,8-tetra chloro dibenzofuran must not exceed 0.1 ng/g, the total concentration of all other chlorinated dibenzodioxin and dibenzofuran impurities must not be greater than 10 µg/g, and no other individual impurity should be greater than 5 µg/g (Health Canada, 2005).

Similarly, due to the potential for the formation of dioxins and dibenzofurans as unwanted low level trace by-products in triclosan (ECB, 2002), the United States Pharmacopoeia (USP) recommends concentration limits for the following impurities in triclosan: less than 10 µg/g for monochlorophenols; less than 10 µg/g for 2,4-dichlorophenol; less than 0.25 µg/g for 1,3,7-trichlorodibenzo-p-dioxin; less than 0.5 µg/g for 2,8-dichlorodibenzo-p-dioxin; less than 0.25 µg/g for 2,8-dichlorodibenzofuran; less than 0.5 µg/g for 2,4,8-trichlorodibenzofuran; less than 1 pg/g for 2,3,7,8-tetrachlorodibenzo-p-dioxin; and less than 1 pg/g for 2,3,7,8-tetra chlorodibenzofuran (USP, 2004). USP recommendations form the basis of enforcement actions by the U.S. Food and Drug Administration.

However the basis for the derived European, Japanese and Canadian maximum allowable concentration of triclosan in cosmetics, and American and Canadian impurities concentration limits, could not be determined.

## 2.2 Australian perspective

Triclosan is not manufactured in Australia. Imported triclosan has been reported for use as an antibacterial ingredient in personal care products; manufacture of carpet underlay, PVC swimming pool liners, chopping boards and textile fabric. In 1999, a total of 22 tonnes was notified to NICNAS as having been imported. The chemical is not reported on the Australian High Volume Industrial Chemicals list.

Triclosan is listed in the Australian Safety and Compensation Council's (ASCC) *List of Designated Hazardous Substances*, contained in the Hazardous Substances Information System (HSIS). Prior to the July 2008 HSIS update triclosan was classified as a hazardous substance with the risk phrase, 'Toxic by inhalation (R23)'. The source for this listing was a registration report by the Australian Pesticides and Veterinary Medicine Authority (ASCC, 2005). In July 2008, the triclosan hazard classification in the HSIS was updated to adopt the changes in Europe's 29<sup>th</sup> Adaptation to Technical Progress (ATP) to Directive 67/548/EEC (April, 2004). Due to this update triclosan is classified in the HSIS as 'Irritating to eyes and skin (R36/38)'.

An atmospheric occupational exposure standard has not been assigned for triclosan in the ASCC *Exposure Standards for Atmospheric Contaminants in the Occupational Environment* as provided by HSIS (ASCC, 2005). Triclosan is not specifically regulated for transport under the National Road Transport Commission's Dangerous Goods Code (ADG Code) (FORS, 1998).

Triclosan is not currently regulated for either public health or environmental purposes. The *Australian Drinking Water Guidelines* (National Health & Medical Research Council, 2004) do not stipulate a limit for triclosan in drinking water. Triclosan is not listed in the *Standard for Uniform Scheduling of Drugs and Poisons* (SUSDP No: 23, June 2008).

## 2.3 Assessments by other national or international bodies

The health and environmental effects of triclosan have recently been evaluated and its classification and labelling determined under EC Directive 67/548/EEC (Annex 1 of Directive 67548 EEC, 2005). Triclosan is classified in the European Union as a skin and eye irritant (with risk phrases R38 and R36 respectively) and dangerous to the environment (risk phrases R50 and R53).

The European Commission's Scientific Steering Committee (SSC) specifically reviewed antimicrobial resistance to triclosan in 2002 and concluded that "there is no convincing evidence that triclosan poses a risk to humans or to the environment by inducing or transmitting antibacterial resistance under current conditions of use" (European Commission Health & Consumer Protection Directorate-General, 2002a).

No international assessment of the health and/or environmental risk for triclosan has presently been carried out in the EU. However, in the European Union triclosan has been 'notified' under the Biocidal Products Directive (98/8/EC) in the following product types (PTs): human hygiene biocidal products (PT1); private and

public disinfectants (PT2); veterinary hygiene biocidal products (PT3); film preservatives (PT7); fibre, leather, rubber and polymerized materials preservatives (PT9). Under the Directive industry will be required to submit a data-package for triclosan to support its use in each of these product types. This will include an assessment of potential risks to human health and the environment. These data packages were due to be submitted to Denmark (the Rapporteur Member State) between 1 February and 31 July 2007 for PTs 1, 2 and 3 and between 1 May and 31 October 2008 for PTs 7 and 9 (Commission Regulation [EC] No 2032/2003, 2003).

Currently there are no restrictions in relation to the use of triclosan as a biocide in the European Union other than those placed on chemicals in relation to their classification and labelling under EC Directive 67/548/EEC.

A Preliminary Risk Assessment of the pesticide uses of triclosan was released by the US EPA in April 2008 for public comment. In addition, an aggregated risk assessment was conducted by the US EPA as part of the Reregistration Eligibility Decision (RED) Document using biological monitoring data for non-EPA regulated uses such as toothpaste, hand soaps and deodorants (US EPA, 2008).

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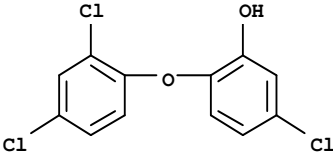
### 3. Identity, Properties, Analysis, Manufacture and Use

#### 3.1 Chemical identity

Triclosan is listed on the Australian Inventory of Chemical Substances (AICS) as:

Phenol, 5 chloro-2-(2,4-dichlorophenoxy)-. Synonyms, trade name and formula are shown in Table 3.1.

**Table 3.1 - Chemical identity**

Property	Value, name or structure
CAS No.:	3380-34-5
EINECS No.:	222-182-2
Synonyms:	Triclosan; 2,4,4' – trichloro-2'-hydroxydiphenyl ether; Ether, 2'-hydroxy-2,4,4'-trichlorodiphenyl; Phenyl ether, 2'-hydroxy-2,4,4'-trichloro-; 2',4',4'-Trichloro-2-hydroxydiphenyl ether; 2',4,4'-Trichloro-2-hydroxydiphenyl ether; 2'-Hydroxy-2,4,4'-trichlorodiphenyl ether; 2,2'-Oxybis(1',5'-dichlorophenyl-5-chlorophenol); 2-Hydroxy-2',4,4'-trichlorodiphenyl ether; 3-Chloro-6-(2,4-dichlorophenoxy)phenol; 4-Chloro-2-hydroxyphenyl 2,4-dichlorophenyl ether.
Trade Names:	CH 3565; Bacti-Stat soap; DP 300; Irgacare MP; Irgacide LP 10; Irgaguard B 1000; Irgasan; Irgasan CH 3565; Irgasan DP 30; Irgasan DP 300; Irgasan DP 3000; Irgasan PE 30; Irgasan PG 60; Microban Additive B; Microban B; NM 100; TCCP; THDP; Tinosan AM 100; Tinosan AM 110; Tinosan NW 500; Tinosan CEL Liquid; Ultrafresh NM 100; Vinyzene DP 7000; Yujiexin; Zilesan UW
Molecular Formula:	$C_{12}H_7Cl_3O_2$
Structural Formula:	
Molecular Weight:	289.54

## 3.2 Impurities and additives

Commercial grades of triclosan are typically over 99% pure. Impurities that may be present in trace amounts are:

- 2,4 Dichlorophenol
- 3-Chlorophenol
- 4-Chlorophenol
- 2,3,7,8 Tetrachlorodibenzo-p-dioxin
- 2,3,7,8 Tetrachlorodibenzo-furan
- 2,8-Dichlorbenzo-furan
- 2,8-Dichlorbenzo-p-dioxin
- 1,3,7 Trichlorodibenzo-p-dioxin
- 2,4,8 Trichlorodibenzo-furan

There are no permitted levels of impurities specified for triclosan in the British Pharmacopoeia (BP). Information on impurities of triclosan imported into Australia were compared to the maximum permitted levels of impurities in the US and Canada. The United States Pharmacopoeia (USP) sets a concentration limit for the following impurities in triclosan: less than 10 µg/g for monochlorophenols; less than 10 µg/g for 2,4-dichlorophenol; less than 0.25 µg/g for 1,3,7-trichlorodibenzo-p-dioxin; less than 0.5 µg/g for 2,8-dichlorodibenzo-p-dioxin; less than 0.25 µg/g for 2,8-dichlorodibenzofuran; less than 0.5 µg/g for 2,4,8-trichlorodibenzofuran; less than 1pg/g for 2,3,7,8-tetrachlorodibenzo-p-dioxin; and less than 1pg/g for 2,3,7,8-tetra chlorodibenzofuran (USP, 2004). Canada also regulates the dioxins and dibenzofurans in triclosan (see section 2.1).

From the data submitted, the imported grade of triclosan from the major local importer meets the specifications of the current edition of the USP. For the remaining local importers of triclosan, it could not be conclusively determined for three importers from the data submitted whether the imported grade of triclosan met the specifications of the current edition of the USP.

Evidence is available that some grades of triclosan traded commercially may not meet USP specifications. Analysis of triclosan manufactured by five different producers in India and a producer in China for the presence of 2,3,7,8-tetrachloro-dibenzo-p-dioxin and 2,3,7,8-tetrachloro-dibenzofuran indicated the samples did not meet USP specifications (Menoutis and Parisi, 2002). Levels of 2,3,7,8-tetrachloro-dibenzo-p-dioxin and 2,3,7,8-tetrachloro-dibenzofuran were observed to range from 17.2 to 1720 pg/g and 0.43 to 207.3 pg/g respectively.

## 3.3 Physical and chemical properties

### 3.3.1 Physical state

Triclosan appears as a white to off-white crystalline powder with a faint aromatic odour (Merck Index, 1983). The chemical is commercially available in solid form. The purity for triclosan for commercial use is 99% minimum (Ciba Specialty Chemicals, 2001a).

### 3.3.2 Physical properties

Few published data on the physical properties of triclosan could be located. Values derived from data provided by the applicants and where possible, published sources are summarised in Table 3.2.

**Table 3.2 - Physical properties of triclosan**

Property	Value	Reference
Melting point	54 <sup>0</sup> C to 57.3 <sup>0</sup> C	Merck Index (1983)
Decomposition temperature	280 <sup>0</sup> C to 290 <sup>0</sup> C	Fiege et al. (2000)
Density	1.55 g/cm <sup>3</sup> at 22 <sup>0</sup> C	Ciba-Geigy Limited (1990a)
Specific gravity	1.58 ± 0.03	Ciba Specialty Chemicals (2001a)
Solubility		
Water	0.001g/100g (1x10 <sup>-5</sup> g /mL) at 20 <sup>0</sup> C	Ciba Specialty Chemicals (2001a)
<i>n</i> -hexane	8.5 g/100g (0.085 g/mL) at 25 <sup>0</sup> C	
ammonium hydroxide	0.30 g/100g (0.003 g/mL) at 25 <sup>0</sup> C	
acetone	> 100 g/100g (> 1.0 g/mL) at 25 <sup>0</sup> C	
PKa (acid dissociation constant)	7.9	Merck Index (1983)
Vapour pressure	4 x 10 <sup>-6</sup> mm Hg (4 x 10 <sup>-4</sup> Pa) at 20 <sup>0</sup> C	Merck Index, (1983)
	2.6 x 10 <sup>-2</sup> mm Hg (2.6 Pa) at 100 <sup>0</sup> C	Fiege et al. (2000)
Partition coefficient ( <i>Log P<sub>ow</sub></i> )	4.8	Ciba-Geigy Limited (1990b)
Henry's Law Constant	0.000000005 (estimated) atm/m <sup>3</sup> mole at 25 <sup>0</sup> C	PBT Profiler (2004)
Autoignition temperature	> 350 <sup>0</sup> C	Ciba Specialty Chemicals (2001a)

### 3.3.3 Chemical properties

Triclosan is produced by treatment of 2,4,4'-trichloro-2'-methoxydiphenyl ether with aluminium chloride in benzene under reflux. Under extreme conditions such as high alkalinity and heat, conversion to chlorinated dibenzo-*p*-dioxins can occur (Fiege et al., 2000). The type and purity of the starting materials in the synthesis of triclosan will influence the extent of contamination by the impurities dioxins and dibenzofurans.

Triclosan is sparingly soluble in water, moderately soluble in dilute alkaline solutions, and readily soluble in most organic solvents. While triclosan in powder form is highly stable to intense radiation, solutions may show instabilities when exposed to intense UV-light radiation. Additionally, solutions are not stable to chlorine and have only moderate stability in the presence of oxidising compounds (Ciba Specialty Chemicals, 2001a).

Triclosan has some volatility in steam. When a suspension of 1000 mg triclosan in 800 ml water is distilled, 180 – 200 mg triclosan are found in the first 500 mL of the distillate (Ciba Specialty Chemicals, 2001a).

### **3.4 Methods of detection and analysis**

Various methods are described in the literature to analyse triclosan and more generally, organic halogen compounds and chlorophenols in a variety of media. Some of the main methods include High Performance Liquid Chromatography (HPLC) and Gas Chromatography/Mass Spectrometry (GC/MS). The methods of detection and analysis of triclosan are provided in **Part 2, Section 13**.

### **3.5 Manufacture, importation and use**

#### **3.5.1 Manufacture and importation**

Data provided by applicants indicated no manufacture of triclosan occurs in Australia. The APVMA provided the information on triclosan imported into Australia in pesticides and veterinary medicines or as a raw material to formulate them. Quantities of triclosan used for therapeutic purposes were identified in surveys undertaken by NICNAS in 2004 and 2006.

In addition, telephone and written surveys of a number of users of triclosan in the textile and plastics industries were conducted in 2004. Information in this chapter is based on data from these sources.

Triclosan is imported into Australia both as the raw chemical (>99% powder), as a liquid solution (10% to <20%), and as an ingredient in various products. Types of imported products containing triclosan include non-therapeutic personal care and cosmetic products, therapeutic goods, textile additives, plastics additives, and grout. In addition, it is probable that finished plastic and textile articles that have been manufactured with triclosan additives are imported into Australia.

Table 3.3 below shows the total amount of triclosan imported into Australia for the years 2001-2005. The figures do not take into account any triclosan imported as part of finished plastic and textile articles, as no information was provided on the triclosan quantities in imported articles. Articles such as these fall outside the scope of this assessment.

**Table 3.3 - Importation of triclosan into Australia annually from 2001 to 2005 (approximate)**

<b>Year</b>	<b>Raw triclosan (&gt;99% purity) (tonnes)</b>	<b>Triclosan contained in imported products (tonnes)</b>	<b>Total (tonnes)</b>
2001	27	3	30
2002	27	4	31
2003	26	2	28
2004	22	1	23
2005	20	1	21

Raw triclosan is imported into Australia as a powder (purity >99%) under the brand names Irgasan DP 300, Irgacare MP, Irgaguard B1000, Cansan TCH, and Triclosan USP25 in 20, 25 and 30 kg antistatic polyethylene lined fibreboard containers. Triclosan is also imported as 10%-<20% aqueous solutions, under the brand name Irgacide LP 10, in 30 kg blue plastic drums. The containers and drums are imported by sea freight and transported typically by road and rail within Australia directly to customers without being opened or re-packed by importers. Triclosan is occasionally stored in warehouses prior to delivery to customers.

Imported textile and plastic additives containing triclosan are stored by importers in licensed warehouses and mostly transported to customers unopened, although one importer formulates a plastic additive product using raw imported triclosan. Information from one importer indicates that personal care and cosmetic products are transferred from ships to trucks by cranes and forklifts, transported by road to the importer's warehouse, and thence to customers.

### **3.5.2 Uses in Australia**

Triclosan is imported into Australia both as the raw chemical and as an ingredient in various products. In Australia, triclosan is an ingredient in non-therapeutic cosmetic and personal care products, therapeutic products, veterinary products, pesticides, household and industrial cleaning products, grouting material, tile paint, and laminate paint. It is also incorporated in the manufacture of some plastics and textile products, and is probably present in some imported finished plastic and textile articles. Triclosan is used for its broad-spectrum anti-microbial activity against bacteria, as well as moulds and yeast.

### **3.5.3 Industrial uses**

#### **Cosmetic and personal care products**

Approximately 200 cosmetic and personal care end products containing triclosan were imported into Australia from 2001 - 2005. The concentration of triclosan in these formulations ranges from 0.00125% to 0.87% and equates to approximately 2 tonnes of triclosan imported annually. Cosmetic and personal care products ranging in size from 5 mL to 500 mL are imported in the form of soaps, creams, gels, sticks, liquids, and powders, in pre-packaged tubes, jars, and bottles.

It is estimated that about 45% – 59% of the total amount of triclosan imported annually into Australia (as 100% powder or 10% aqueous solution) is used in the formulation of personal care and cosmetic products. Therefore, the total amount of triclosan present in personal care and cosmetic products in Australia, from both imported finished products, and from products formulated within Australia, is estimated to be approximately 11 – 18 tonnes per annum (see Table 3.4).

**Table 3.4 – Estimated amount of triclosan in cosmetic and personal care products (non-therapeutic) annually from 2001 to 2005.**

Year	Tonnes (approximate)		
	Present in imported finished end products	Formulated into products in Australia	TOTAL
2001	2.1	15.9	18.0
2002	1.8	14.3	16.1
2003	1.1	15.0	16.1
2004	0.9	9.9	10.8
2005	0.9	11.6	12.5

The following is a list of cosmetic and personal care product types containing triclosan marketed in Australia, compiled from data provided to NICNAS by industry for this assessment:

- Body sprays
- Underarm deodorants (spray, stick, roll-on)
- Feminine deodorants
- Colognes
- Foot and shoe deodorant sprays and talc
- Soaps, including liquid hand wash, shower and bath gels
- Face and skin cleansers, moisturisers, toners, exfoliants
- Facial masks
- Eye make-up
- Pre-wax skin wipes
- Baby wipes
- Skin purifying patches
- Anti-acne formulations
- Cuticle and nail conditioners
- Toothpaste
- Mouthwash
- Cotton buds
- Sunscreens
- Insect repellents

### **Use in household and industrial cleaning products**

Triclosan is present in a number of household and industrial-grade cleaning products formulated in Australia, at concentrations ranging from 0.04% – 0.30%. No household or industrial-grade cleaning products containing triclosan were imported into Australia. The total amount of triclosan used in Australia for the

formulation of these products for the years 2001 – 2003 is estimated to be at least 0.5 tonnes annually. Limited data was provided for 2004 and 2005, and so no estimates are made for these years. Products notified to NICNAS by industry for this assessment included:

- Dishwashing detergents
- A wool wash laundry detergent
- Bathroom surface cleaning products
- A commercial kitchen surface cleanser
- A hospital grade disinfectant/cleaner
- Floor mop cartridges

Household cleaning products are packed in 500 mL, 600 mL, 750 mL and 1 L containers, while the industrial-grade products are packed variously in a 750 mL trigger container, 500 mL, 1 L, 5 L, 15 L and 25 L containers.

### **Use in textile manufacture**

Textile additives containing triclosan are imported as liquids in 20 L, 25 kg, and 30 kg plastic containers and 200 L drums, and as a powder in 25 kg containers.

Triclosan is used in textiles to impart odour-protection properties to wool, synthetics, blends, and non-wovens by inhibiting the growth of bacteria and fungi on these surfaces, and to eliminate house dust mites from material.

Several products containing triclosan for use in textile manufacture have been imported into Australia from 2001 to 2005. These products contain triclosan at concentrations ranging from >1% to <20%. It is estimated less than 1 tonne of triclosan was used annually between 2001 and 2005 in these products. One formulator of a product for use in textile manufacture has been identified in Australia. This company uses an imported textile additive solution containing 1.25% triclosan as an ingredient in a chemical product used for coating fabric subsequently used in the manufacture of vertical blinds. No other formulators of textile additive products were identified.

The following is a list of textile end-use products manufactured in Australia that use a triclosan additive in their manufacture:

- Wool bedding
- Quilts (wool filling)
- Pillows (wool filling)
- Doona filling (polyester blend)
- Under-blankets
- Furniture upholstery
- Woollen goods and general textiles
- Towels
- Curtains, blinds
- Fashion, swimwear and sports apparel
- Hosiery
- Socks
- Shoe insoles

- Zippers
- Insulation bats

Application of triclosan to textile products is generally by inclusion in a dye bath, other treatment baths, or by padding. One manufacturer of polyester blend wadding sprays a triclosan solution on to batches of textile. The chemical coating treatment for vertical blind fabric is reported to be applied by a knife coating technique. Generally the products are completely applied and no waste is expected to be generated.

It is not known how much triclosan is imported into Australia in finished textiles, such as bedding and clothing. Such products are classed as 'articles' under the *Industrial Chemicals (Notification and Assessment) Act 1989* and as such are outside the scope of this report. A market and internet web page survey by the Danish Environmental Protection Agency for textile articles (Danish Environmental Protection Agency, 2003a) containing biocides, including triclosan, indicates that the following types of imported textile articles may contain triclosan: clothing for hospital workers, hospital bedding, sports clothing, socks, and tights. Triclosan is either built-in to the textile fibres during manufacture or applied as a coating by various techniques (Danish Environmental Protection Agency, 2003a). A NICNAS search of web pages marketing antibacterial products undertaken in 2004 indicated that the following types of products may also contain triclosan: mattress pads, pillows, sports clothing, shoes, underwear, socks, tights, gloves, hats, scarves, sleeping bags, pet beds, non-woven wipes, filters, and surgical type masks (Sterling Fibres, 2005; Safety and Security Centre, 2003; Manufacturabrasil, 2004).

### **Use in plastic manufacture**

Plastic additives containing triclosan are imported as liquid in 25 kg and 100 kg plastic drums, as granules in 20 kg plastic bags, and as pellets in 20 kg, 25 kg or 30 kg polyethylene lined fibreboard drums and 200 kg plastic lined drums with seal system.

Triclosan is used in plastics manufacture as an antimicrobial additive, to protect the articles from deterioration and from odours and discoloration.

Several products intended for this use have been imported between 2001 - 2005, containing triclosan at concentrations ranging from >1% - ≤ 10%. In addition, some plastic additive products, containing ≤ 5% triclosan, are formulated in Australia from imported raw triclosan. The total annual amount of triclosan used in plastics additives annually is estimated to be approximately 0.5 tonne.

Plastic end products manufactured in Australia using triclosan additives include various household moulded plastic products including:

- Food storage containers
- Wheelie bins
- Toilet seats
- Toilet tidy sets
- PVC carpet backing
- Swimming pool liners
- Toothbrushes, and

- Pet accessories such as litter trays, food bowls, and Frisbees.

Triclosan is also used in the manufacture of a cling wrap for export, which contains triclosan at 0.6 % concentration.

It is not known how much triclosan is imported into Australia already incorporated into finished plastic products. Some of the types of imported plastic products that could incorporate triclosan include: domestic and commercial kitchenware such as food storage bins, knives, cutting boards, sponges, appliances, gloves, kitchen and bathroom fixtures, medical devices, toys and high chairs, and flooring materials (Ciba Specialty Chemicals, 2001a).

### **Other industrial uses**

A product containing triclosan is being developed in Australia for use as an antimicrobial treatment agent for air conditioning heat exchange coils. The product, a spray-on aerosol containing 0.6% triclosan to be used by air conditioning service contractors, has not yet been released for sale. To date < 1 kg has been used annually in development.

There is very limited use of triclosan in grout, with one such product containing the chemical imported into Australia.

Triclosan is added to some oil-based paint formulated in Australia for interior use on tiles and laminates, as an antimicrobial agent, at a concentration of 1g/L. The amount of triclosan used annually for this purpose is <0.1 tonne.

### **3.5.4 Non-industrial uses**

#### **Therapeutic uses**

In 2003, triclosan was an ingredient in 84 therapeutic goods registered on the Australian Register of Therapeutic Goods (ARTG). For 56 of these products, triclosan is the 'active' ingredient, meaning that therapeutic claims are being made with respect to the triclosan as used in that product. For the remainder of the products (28), triclosan is listed on the ARTG as an 'excipient' ingredient, meaning that triclosan is not the ingredient in the product responsible for the making of therapeutic claims. Generally the triclosan in these latter products is considered as a preservative rather than a bactericide. The concentration of triclosan in therapeutic goods ranges from 0.5 mg/g to 20 mg/g.

As stated in Section 1.3, NICNAS conducted two surveys of the registrants of therapeutic goods containing triclosan listed on the ARTG in 2004 and 2006. Survey responses indicated that most therapeutic products are formulated in Australia from locally sourced triclosan. A small number (10) are imported as finished products. Twenty-eight of the 84 registered products were not being marketed at the time of the 2004 survey. From the survey responses, it is estimated that about 39% – 47% of the total amount of triclosan imported either as 100% raw powder or 10% aqueous solution per annum was utilised in the formulation of therapeutic products annually in the period 2001 – 2005. Less than one tonne per annum is imported as an ingredient in finished therapeutic products. Therefore, a total of approximately 10 - 13 tonnes of triclosan was formulated into therapeutic products or imported in finished therapeutic goods, per annum, for the period 2001 – 2005. This data is presented in Table 3.5.

**Table 3.5 - Estimated quantities of triclosan present in therapeutic products annually from 2001 to 2005.**

Year	Tonnes (approximate)		TOTAL
	Imported in finished products	Formulated in Australia	
2001	0.4	10.5	10.9
2002	0.7	12.3	13.0
2003	0.6	10.7	11.3
2004	0.3	9.2	9.5
2005	0.3	9.3	9.6

The following range of product types are represented:

- Medicated soaps
- Pimple creams
- Burn gels
- Toothpastes
- Insect repellents
- Antiseptic hand washes and barrier lotions
- Bath oil emollient
- Face washes,
- A pre-operative surgical liquid hand wash
- Lip balm
- Sunscreens
- Surface disinfectant

#### **Veterinary uses**

Triclosan is an ingredient in 22 products used for veterinary purposes: six pet shampoos, fifteen insect repellents, and a cattle teat ointment. The approximate total annual amount of triclosan used in these products is less than 66 kg per year.

#### **3.5.5 Overseas uses**

Overseas, triclosan is reported to be used in a similar range of products as reported in Australia, including cosmetic and personal care products, dermatological and topical care preparations for the skin, dentifrices and oral rinses, dishwashing and laundry detergents, fabric softeners, surface cleansers, and in textiles and plastics (Bhargava and Leonard, 1996; European Commission Health & Consumer Protection Directorate-General, 2002b; Ciba Specialty Chemicals, 2001b; US NPIRS, 2005).

Overseas uses reported in the literature but not reported in Australia include as an ingredient in toilet cleaners and as a biocide in cutting oils (Grattan et al., 1989).

Information from a technical brochure for a product marketed in Australia primarily for use as a textile additive states it can be: used as a laundry additive to be used with softener in the final rinse stage; added to pigment presscakes, dispersion and inks; used for carpet cleaning; and applied to synthetic and cellulosic sponges. These uses were not reported as occurring in Australia, and it is not known whether these are uses that occur overseas.

### Summary of uses

A summary of the uses of triclosan (industrial and non-industrial) is presented in Table 3.6.

**Table 3.6 - Estimated average annual distribution of triclosan in Australia by use category based on information provided for the assessment**

Use	Approximate average (tonnes)
Industrial	
Cosmetic/personal care products (non-therapeutic)	15
Household and industrial cleaning products*	<1
Textile additives	<1
Plastic additives	0.5
Therapeutic goods	11
Veterinary use	<0.1

\* Based on 2001 to 2003 data. The others are the average of 2001 to 2005 data.

Among the industrial sectors, the cosmetic/personal care sector is the major user of triclosan (Table 3.6), and the use appears to be declining over time (Table 3.4).

The range of uses of triclosan in Australia is very similar to the uses of triclosan overseas.

## 4. Human Exposure

### 4.1 Occupational exposure

Occupational exposure to triclosan may occur during transport, storage, repacking, formulation of personal care/cosmetic products, therapeutic products, cleaning agents and paints, treatment of textiles, plastic manufacture and/or during use of end products containing triclosan.

During occupational use of triclosan powder, solutions and triclosan-containing end-use products, the main exposure routes are dermal and inhalation, though ocular exposure may also occur.

In the absence of worker exposure data, exposure to triclosan (except for use of end products) was estimated using the Estimation and Assessment of Substance Exposure (EASE) model (version 2.0 for Windows) developed by the United Kingdom Health and Safety Executive (UK HSE). Occupational exposure during use of end products containing triclosan was estimated according to the European Commission's Technical Guidance Document on Risk Assessment (EC, 2003a). The estimated internal doses resulting from occupational inhalation and dermal exposure to triclosan and the integrated internal doses are summarised in Table 4.1.

**Table 4.1 - Internal dose levels for processes using triclosan**

Occupational Scenario	Triclosan	Inhalation (µg/kg bw/d)	Dermal (µg/kg/d)	Integrated internal dose* (µg/kg bw/d)
Repacking	100% powder	0.25-0.64 (with LEV) 0.64-6.36 (without LEV)	5.5-55	5.8-55.6 (with LEV) 6.1-61.4 (without LEV)
Formulation of end products	100% powder	15.9-39.7 (with LEV) 39.7-397 (without LEV)	85.5-855	101-895 (with LEV) 125-1252 (without LEV)
Textile	13.5% powder	2.14-5.36 (with LEV) 5.36-53.6 (without LEV)	11.5-115	13.6-120 (with LEV) 16.9-169 (without LEV)
	20% liquid	- **	17-170	17-170
Plastic manufacture	100% powder	15.9-39.7 (with LEV) 39.7-397 (without LEV)	85.5-855	101-895 (with LEV) 125-1252 (without LEV)
	10% liquid	- **	8.6-86	8.6-86
End use	0.3% maximum	- **	16.7	16.7

\*Integrated internal dose is the total dose following inhalation and dermal exposure for the activity undertaken.

\*\*Inhalation dose not calculated as considered negligible.

Although work processes with triclosan in the textile and plastic industry can involve high temperature heating which could result in vapour formation and an increased potential for inhalation exposure, most of the equipment used at high temperatures are closed systems. In addition, inhalation exposure is reduced where local exhaust ventilation (LEV) is present (Table 4.1). Furthermore, workers are

not required to perform any tasks around these 'heat' zones during these operations, and thus the potential for exposure to triclosan vapour is considered minimal.

The EASE model predicts that occupational tasks using 100% powdered triclosan result in the greatest exposure, and for each major occupational task (i.e. repackaging, formulation, and plastic manufacture) the EASE scenario that best describes the process resulted in total internal doses ranging from 5.8 - 895 µg/kg bw/day with LEV (Table 4.1). For textile treatment using 13.5% triclosan powder, the integrated internal dose is much lower, 13.6 - 120 µg/kg bw/day with LEV. Workers handling 10-20%<sup>4</sup> liquid forms of triclosan in the textile and plastic industries and handling end-use products containing triclosan are predicted to have lower occupational exposure ranging from 8.6 - 170 µg/kg bw/day (Table 4.1).

In the industrial setting, the real exposure level and subsequent internal dose that workers receive are likely to be lower as the estimations by EASE does not take into account Personal Protective Equipment (PPE) which were reported to be worn at all the sites surveyed by NICNAS. Consequently the use of PPE together with the use of mechanised, closed or partially enclosed work processes mean that the actual exposures are likely to be lower than that predicted by the EASE model.

A detailed analysis of occupational exposure to triclosan is provided in **Part 2, Section 14**. The occupational exposure calculations are detailed in **Appendix C**.

## **4.2 Public exposure**

### **4.2.1 Adults**

Exposure estimations indicate that of the industrial uses of triclosan, for adults in the general public the major source of exposure is likely to be from topical application of cosmetic and personal care products, though inhalation exposure following the use of household surface sprays can also contribute significantly to the overall body burden. However, it should be noted that for cosmetic and personal care products the predicted dermal exposure is based on the combined use of 17 products. The total maximum internal dose is estimated to be 578.1 µg/kg bw/day (Table 4.2). This is considered the worst-case scenario and is obtained from combined exposure through all potential routes of exposure. However, as individual use and hence exposure to these products will vary widely the actual exposure is likely to be significantly less in some sub-populations of the public who do not use these products on a regular daily basis.

In addition to the modelled data, limited measured exposure data are also available arising from the use of personal care products. Generally exposure to single and multiple personal care products containing triclosan resulted in steady state plasma levels of total triclosan less than 40 ng/mL, though higher levels up to 229 ng/mL were also occasionally seen.

The estimated internal doses in adults in the general public for various exposure scenarios following the use of products containing triclosan are presented in Table 4.2.

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<sup>4</sup> Liquid products used in the manufacture of textiles contain >1 % to < 20 % triclosan. For determining a worst-case exposure scenario for textile manufacture it is assumed that the product contains 20 % triclosan.

**Table 4.2 - Summary of internal dose levels in adults using exposure models**

<b>Public exposure scenario</b>	<b>Inhalation (µg/kgbw/day)</b>	<b>Dermal (µg/kgbw/day)</b>	<b>Oral (µg/kgbw/day)</b>	<b>Total exposure (µg/kg bw/day)</b>
Cosmetic & personal care products <sup>1</sup>	18.0 - 53.9	145.5	24.4	187.9-223.8
Household cleaning products <sup>1</sup>	1.7 – 349.5	0.34	ND	2.04-349.84
Article surfaces	3.8	ND	ND	3.8
Painted surfaces	ND <sup>2</sup>	0.56	ND	0.56
<b>Total internal dose</b>	<b>23.5 – 407.2</b>	<b>146.5</b>	<b>24.4</b>	<b>194.4-578.1</b>

<sup>1</sup> Determined for the maximum concentration of triclosan reported for each product type in Australia

<sup>2</sup> Included under article surfaces

ND=No data

#### 4.2.2 Children

Babies and young children are likely to be exposed to triclosan through use of cosmetic, personal care and household products. Furthermore, breast milk consumption may provide an additional source of exposure. When determining exposures in this assessment a baby is defined as a child less than one year old and exposures are determined in young children up to five years old.

No measured exposure data for babies and young children following use of consumer products containing triclosan was identified in the literature. Consequently, the same exposure models used to predict adult exposure to triclosan from consumer products have been used to predict exposure to babies and young children (see Appendix D). The use data available for these consumer products is for adults with no use data available in literature for babies and young children. Therefore, as a rough approximation of exposure and when considered 'reasonable', the adult use data has been used to predict exposure to babies and/or young children though it is recognised that the predicted values will be over-estimates and should be regarded as such.

With regards to breast milk consumption mean intake values and body weight are taken from the interim draft report of the US Environmental Protection Agency Child-Specific Exposure Factors Handbook (US EPA, 2002).

From the available data it is predicted that in young children the major source of exposure is likely to be from personal care products and accidental/intentional ingestion of toothpaste. For a worst-case scenario, where the exposure from all routes are combined, a maximum total internal dose of 105.3 and 71 µg/kg bw/day in a two and five year old respectively was estimated. For babies it was observed that for an exclusively breast-fed baby exposed to the highest concentration of triclosan detected in an Australian breast milk sample (19 ng/g of milk), the

internal dose of triclosan received was less (3.04 µg/kg bw/day – see Appendix D) than predicted from other sources of exposure in both babies and young children.

A summary of the predicted internal dose level in babies and young children is shown in Table 4.3.

**Table 4.3 -Summary of internal dose levels in babies and young children**

Public exposure scenario <sup>1</sup>	Inhalation (µg/kg bw/day)	Dermal (µg/kg bw/day)	Oral (µg/kg bw/day)	Total (µg/kg bw/day)
Baby				
<1 year	6.1	90.9	3.0 <sup>2</sup>	100.0
Young children				
2 years	5.3	84.5	15.5	105.3
5 years	4.3	56.4	10.3	71.0

<sup>1</sup> Determined for the maximum concentration of triclosan reported for each product type, and observed in a breast milk sample, in Australia

<sup>2</sup> This is the highest value determined in a 1-month old baby

Maternal and cord blood serum samples received from the Academic Hospital of Groningen, Netherlands were analysed for selected man-made chemicals including triclosan (Peters, 2005). Triclosan was detected in approximately half of the samples analysed (16 out of 39 maternal blood and 8 out of 17 cord blood samples). In maternal blood the concentration of triclosan ranged from 0.1 to 1.3 ng/g serum and in cord blood from 0.5 to 5.0 ng/g serum (limit of detection <0.1 ng/g serum). The levels of triclosan in cord blood were higher than in maternal blood.

Three quarters of the urine samples (2517) collected from the US general population (age 6 years and older from 2003 – 2004) contained free and/or conjugated triclosan (95<sup>th</sup> percentile = 459.0 µg/L). Concentrations differed by age and socio-economic status but not by race/ethnicity and sex. The concentrations of triclosan appeared to be highest during the third decade of life and among people with the highest household income (Clafat et al., 2007).

A detailed analysis of the exposure of the general public to triclosan is provided in **Part 2, Section 15**.

## 5. Environmental Exposure

Triclosan is widely used in Australia, particularly in consumer applications (personal care products) and therapeutic products that entail discharge to sewer, and to natural surface waters after treatment.

### 5.1 Environmental fate

The water solubility of triclosan is low (10 mg/L) but environmentally significant as the solubility allows triclosan to be transported in solution. Triclosan is stable to hydrolysis, but can be regarded as inherently biodegradable in aerobic aquatic environments because of its susceptibility to microbial metabolism. Degradation of triclosan in soil incubated under aerobic conditions proceeds primarily via the formation of methyl triclosan and significant amounts of bound residues. Some mineralization of the residues is observed. In aerobic aquatic systems, triclosan dissipates rapidly from the water phase by degradation and adsorption to the sediment. In both compartments, it degrades to numerous minor metabolites, bound residues and carbon dioxide. Photolysis also contributes to the loss of triclosan from sunlit surface waters. In contrast to its degradation in aerobic environments, triclosan degrades very slowly and is persistent under anaerobic conditions, for example in soil and sediment.

A minor metabolite, methyl triclosan, forms during aerobic treatment of sewage and is discharged in sewage effluent together with residues of triclosan. This metabolite occurs at much lower concentrations than triclosan, but is more persistent and bioaccumulative.

Consistent with its low water solubility, triclosan can sorb strongly to soils and sediment.

### 5.2 Environmental release

The release of triclosan to the sewage system as a result of its use in personal care products will result in its partitioning to both the aqueous effluent, which is subsequently discharged to receiving waters, and to sludge (nutrient rich organic matter, also known as biosolids).

#### 5.2.1 Aqueous environment

A recent Australian screening study determined the concentrations of triclosan in the effluent from nineteen sewage treatment plants (8 from South Australia (SA), 5 from Queensland, 2 from the Australian Capital Territory (ACT), 1 from Western Australia (WA) and 3 from Victoria) which ranged from 23 ng/L to 434 ng/L with mean and median concentration of 142 and 108 ng/L, respectively. A follow up study on five of these sewage treatment plants in SA and WA indicated substantial removal (72-93%), with influent concentrations of 573-845 ng/L reducing to 60-159 ng/L in effluents discharged to surface waters. There is uncertainty as to whether these data are reflective of larger sewage treatment plants serving major urban populations, for example in Melbourne and Sydney, but these Australian data are comparable to or slightly below overseas measurements.

Recent Australian monitoring in five rivers/estuaries receiving effluents from sewage treatment plants in Queensland has found triclosan at concentrations up to 75 ng/L near the sewage outfall (Ying and Kookana, 2007). The range of concentrations detected was 21-75 ng/L in 2004 and 14-60 ng/L in 2005. Respective effluent concentrations were 51-222 ng/L and 45-187 ng/L. Concentrations were reduced at upstream and downstream sampling locations, both about 200 m from the outfall, particularly during 2005 when sampling occurred under summer conditions conducive to rapid degradation of triclosan. The authors of this study caution that riverine concentrations could exceed 75 ng/L during drought conditions as there would be limited dilution of the discharged effluent. Overseas measurements are comparable to or slightly higher than the Australian data.

Concentrations of triclosan entering and leaving Australian sewage treatment plants have been estimated, based on the assumptions that 96% (Ciba Specialty Chemicals, 1998a) of the import volume is discharged to sewer, and 28-39% (estimated using the SimpleTreat 3.0 model assuming that triclosan is inherently biodegradable or not biodegradable, respectively) of this amount discharged in treated effluent (Table 5.1) for the various levels of sewage treatment possible in Australia. The estimates obtained are about ten to twenty times higher than the measured values listed above. This adds to the uncertainty as to whether the limited available Australian data are truly representative, and indicates a need for further monitoring.

**Table 5.1 - Predicted surface water triclosan concentrations using SimpleTreat, reported triclosan removal rates, various levels of wastewater treatment and the estimated Australian triclosan introduction quantity**

Level of Treatment	Removal rate (%) <sup>*</sup>	PEC Freshwater (triclosan, ng/L)	PEC Marine (triclosan, ng/L)
Untreated wastewater	---	14500-17400	1450 - 1740
Primary Treatment	2-96	581-17000	58 - 1700
Secondary Treatment			
Trickling Filter	58-96	581-7300	581 - 730
Activated Sludge	55-99	145-7820	14.5 - 782
Activated sludge (SimpleTreat)	61-72	4070-6780	407 - 678
Tertiary treatment	87-≥99	≤145-2260	≤14.5 - 226

<sup>\*</sup> Removal rate obtained from literature sources. Freshwater and marine PEC values obtained by dividing the estimated effluent concentration by receiving environment dilution factors of 1 and 10, respectively.

## 5.2.2 Terrestrial environment

Triclosan can be substantially removed by adsorption to biosolids during sewage treatment. The rate of removal is highly variable and dependant on the type and level of treatment of the effluent. These triclosan containing biosolids may be added to soil as an ameliorant. The terrestrial environment may also be exposed to

triclosan through irrigation using triclosan containing effluent. The predicted concentrations from these uses are summarized below in Table 5.2.

Recent Australian data from sewage treatment plants in South Australia and Western Australia indicate that biosolids may contain 0.090-16.790 mg/kg triclosan on a dry weight basis. Again, there is some uncertainty as to whether these data are reflective of other parts of Australia, but they are comparable to overseas measurements. The predicted concentration in soil amended with biosolids approaches but does not exceed 1 mg/kg, while limited data indicate that actual levels will be much lower than predicted.

A detailed analysis of the environmental exposure is provided in **Part 2, Section 16**.

**Table 5.2 -Soil PECs resulting from use of biosolids and a soil conditioner and treated effluent for irrigation**

Parameter	Environment Australia (2003) STP model		ASTE (2004) and Dillon (2000) model	
Estimated quantity of triclosan to sewer (kg/y)	26000		26000	
Estimated quantity of triclosan to sewer (mg/y)	$2.60 \times 10^{10}$		$2.60 \times 10^{10}$	
Estimated fraction in sludge based on SimpleTreat model (%)	55% <sup>a</sup>	61% <sup>b</sup>	55% <sup>a</sup>	61% <sup>b</sup>
Estimated sludge triclosan conc. (mg/kg dry wt)	95.6	106	79.9	88.6
Soil Application Rate (tonnes/ha/year)			10	
PEC <sub>Soil</sub> biosolid application (mg/kg dry wt)	0.735	0.815	0.614	0.681
Influent Concentration (µg/L)	17400	17400	14500	14500
Overall Removal Rate (%)	61% <sup>a</sup>	72% <sup>b</sup>	61% <sup>a</sup>	72% <sup>b</sup>
Concentration in effluent (µg/L)	6.78	4.86	5.66	4.07
Waste water application rate to land (m/ha/year)			1.0	
PEC <sub>Soil</sub> irrigation (mg/kg dry wt)	0.0521	0.0374	0.0436	0.0313

Notes: The SimpleTreat model output refers only to an activated sludge treatment process. a: Assumes no biodegradation; b: Assumes inherently biodegradable.

## 6. Human Health Hazard Assessment

### 6.1 Kinetics and metabolism

Numerous human and animal studies are available on the toxicokinetics of triclosan following both oral and dermal exposure and these are summarized below.

#### 6.1.1 Oral and dermal route

##### Absorption

Following oral administration of triclosan, absorption from the gastrointestinal tract is rapid and extensive in both humans and animals. Data in one study in humans indicates absorption to be at least 97% while comparative oral and intravenous studies in rodents indicate absorption to be from 70% to 'virtually complete'. Consequently, for the purposes of this risk assessment, absorption is considered to be 100% following oral administration in humans. Following dermal application of triclosan-containing products, absorption in humans was generally at least 3% to 7%, though at least 14% was observed in one volunteer for a 12 h exposure. Animal data indicates that the extent of triclosan absorption is dependent on the formulation applied. A number of studies in the rat indicate absorption to be 21% to 28% following application in ethanol-based, soap suspension and cream formulations, while skin biopsy and in vitro evidence suggest that the rate of dermal absorption is less in humans than animals. Thus, it is considered that dermal absorption in humans is 14%, as this may be observed in some individuals. In vitro dermal absorption studies using human skin preparations and various formulations containing triclosan showed dermal absorption values for triclosan ranging from 11-20% in these formulations (US EPA, 2008). Additionally, limited buccal absorption was also seen in humans. Following normal toothpaste use absorption was up to 14% of the amount that would be absorbed if an equivalent dosage of triclosan were ingested.

The US EPA evaluated dermal absorption studies on triclosan or its formulations and estimated a dermal absorption value of around 20% for rat skin and possibly a lower value for human skin. The US EPA report stated that additional verification is needed for determination of dermal absorption of triclosan (US EPA, 2008).

##### Distribution

Triclosan was rapidly removed from the blood, and metabolism data indicate extensive first pass metabolism following absorption from the gastrointestinal tract. The half-life of elimination for orally administered triclosan ranged from approximately 13 to 29 h in humans compared to 10 to 15 h in rats, 8 to 12 h in mice and 25 to 32 h in hamsters. In rodents, radioactivity was widely distributed to organs and tissues following oral or dermal exposure to <sup>14</sup>C- or <sup>3</sup>H-triclosan. Well-perfused and excretory organs such as liver, lung, kidney, gastrointestinal tract and gall bladder showed highest levels following oral and dermal absorption in rodents. Additionally, evidence is available in the mouse that suggests that the liver is a specific target organ. Triclosan has also been detected in human breast milk

samples at levels ranging from below the limit of quantification to 19 ng/g milk. However, due to pronounced first pass metabolism the bioavailability of unconjugated triclosan is likely to be very limited following oral exposure. Enterohepatic circulation has been demonstrated in rats, while limited evidence is available for such in mice and hamsters.

### **Metabolism**

The major metabolic pathways in humans and animals involve glucuronide and sulphate conjugation. Data in rodents indicates that the liver has a high conjugating capacity for triclosan, while human and animal data demonstrate triclosan is metabolised to the glucuronide and sulphate conjugate in the skin. The relative proportion of these metabolites varies depending on plasma steady state of triclosan and these conjugates combined, with higher concentrations resulting in a shift from predominantly glucuronide- to predominantly sulphate- conjugates in rodents and humans. No difference in metabolic patterns was seen between different human racial groups. In humans and rodents triclosan glucuronide and triclosan are predominantly found in the urine and faeces respectively.

### **Excretion**

The major route of excretion is via the urine with the faeces being of secondary importance in humans, hamsters, rabbits and primates following oral exposure, whilst the reverse was seen in rats, mice and dogs. The available dermal data, in rats and rabbits, indicates the same predominant routes of excretion. In humans up to 87% of the administered dose was excreted in the urine and elimination was relatively rapid; the majority of the dose was excreted by 72 h post dose. Though a significant difference was observed in the rate of elimination between some Negroid (black) volunteers compared to Caucasians (white), there are no data available to explain why this difference was observed. However, the human oral and dermal data provide no evidence of a bioaccumulation potential. Likewise, the tissue distribution data in rats and hamsters following single and repeated dosing provides no evidence of bioaccumulation in these species, though there is limited evidence in mice that retention of triclosan and/or its metabolites may occur in the liver.

The observance of triclosan and/or its metabolites in human breast milk indicates potential excretion in breast milk. However, the data do not allow a reliable quantitative determination to be made on the potential dose excreted by this route following exposure to triclosan. The first pass metabolism and relatively rapid elimination of triclosan, though, suggest that the potential for transfer to the foetus and bioaccumulation may be limited.

### **Inhalation route**

There are no data on the toxicokinetics of triclosan following inhalation exposure. However, the observation of clinical signs of toxicity such as muscle spasms seen in a repeat inhalation study in the rat indicates that absorption via the inhalation route can occur, but the data do not allow a quantitative estimation of absorption to be made. Furthermore, because first pass metabolism would not take place following exposure by this route, bioavailability of triclosan is likely to be substantially greater than is associated with the oral route, or the dermal route where metabolism of triclosan to its conjugates has been demonstrated in the skin.

A detailed analysis of the kinetics and metabolism is provided in **Part 2, Section 17**.

## **6.2 Effects on laboratory animals**

### **6.2.1 Acute toxicity**

The most recent and well-conducted LD50 study indicates that triclosan has low acute toxicity by the oral route (LD50 >5000 mg/kg bw), though there is evidence from older and less well reported studies that it is moderately toxic and produces nephrotoxicity. No clinical signs of toxicity were observed following a 4 h exposure to an aerosol of 0.15 mg triclosan/L, which was the highest technically achievable rat respirable concentration used in this study. The LC50 was greater than 0.15 mg/L. Due to this very low dose tested in this study it is not possible to derive a conclusion about the acute inhalation toxicity of triclosan. However, in a repeat dose inhalation toxicity study in rats, more than 50% rats died after a single 2-h exposure to 1300 mg triclosan/m<sup>3</sup> air. Therefore, LC50 for triclosan is considered as <1300 mg/m<sup>3</sup> or <1.3 mg/L. Limited evidence is available that triclosan is of low acute toxicity by the dermal route (LD50 >9300 mg/kg bw for a slurry with propylene glycol) and that its acute toxicity is increased if administered intravenously.

### **6.2.2 Irritation**

The available data shows that triclosan produces both skin and eye irritation in studies in rabbits but is not phototoxic in a study in guinea-pigs. Respiratory tract irritation was observed in rats exposed to triclosan in the repeat dose inhalation toxicity study and therefore, triclosan is considered a respiratory irritant.

### **6.2.3 Sensitisation**

The available data indicate that at most triclosan possesses a very weak skin sensitisation potential in studies conducted in guinea-pigs.

### **6.2.4 Repeat dose toxicity**

#### **Inhalation**

In the only available 21-day inhalation study conducted in rats (2 h/day nose only exposure), clinical signs of toxicity and death in the high dose animals at 1300 mg triclosan/m<sup>3</sup> air (in 10% ethanol) indicate systemic toxicity. More than 50% rats in the highest dose group died (11 out of 18) on the first two days of the experiment, after a single 2 h exposure, compared to no deaths in other treatment groups or the control group exposed to 10% ethanol. All other observed treatment related effects are due to local irritation for which a NOAEC of 0.05 mg/L was identified.

#### **Oral**

Studies are available in the mouse, rat, hamster, rabbit, dog and baboon.

A NOAEL could not be identified in the 13-week mouse study, although a LOAEL of 25 mg/kg bw/day was identified based on effects on haematology parameters, relative liver weight and total cholesterol in both sexes. However, while the mouse is the most sensitive species, there is evidence that (unlike the rat and hamster) it is

sensitive to peroxisome proliferator type effects that are not considered relevant to a human health risk assessment. Consequently, a NOAEL of 40 mg/kg bw/day (m) and 56 mg/kg bw/day (f) was identified from a two-year carcinogenicity study in the rat based on clinical chemistry changes, together with histopathological changes in the liver in males and a trend for reduced body weight gain in females.

## **Dermal**

Local irritant effects have been clearly seen in animal studies. A NOAEL of 7.5 and 3.5 mg/kg bw/day was identified in 14-day studies in male and female rats, respectively. No systemic toxicity was seen in the rat studies and the only available robust dog study. However, histological changes to the liver were seen in two 14-day studies in the mouse, with a NOAEL of 20 and 24 mg/kg bw/day identified in males and females respectively.

In a 90-day rat study, no treatment related effects were seen on mortality, clinical signs of toxicity, body weight gain, food or water consumption, haematology, clinical chemistry or organ weight. Coagulative necrosis of the liver, focal cortical tubular degeneration of the kidney and microscopic changes to the bladder were seen at necropsy in a small number of animals. In the absence of a dose response effect these observations were not considered treatment related. Occult blood was seen in the urine of 3 to 4 males per group at 40 mg/kg bw/day and above, including the recovery group, and 2 females in the 40 mg/kg bw/day and recovery group. However, as the significance of this finding is unknown, it is not considered to provide reliable evidence of systemic toxicity based on the weight of evidence, and a NOAEL of 80 mg/kg bw/day is identified. For local irritant effects a NOAEL could not be identified, and thus the LOAEL was 10 mg/kg bw/day.

### **6.2.5 Genotoxicity – in vitro**

Negative results have been seen in numerous studies in bacteria, with only a single weakly positive result seen in a briefly reported study at a very high dose level. Similarly, no robust evidence of an in vitro mutagenic activity was seen in studies in fungi or mammalian cells. Both a positive and negative chromosome aberration study is available, while only negative results were seen in Unscheduled DNA Synthesis (UDS) assays. Thus, the weight of evidence (a single robust positive chromosome aberration assay from numerous in vitro studies) does not indicate a significant genotoxic potential.

### **6.2.6 Genotoxicity – in vivo**

In vivo, negative results have been seen in a number of bone marrow chromosome aberration and micronucleus studies. Both a negative and positive result has been seen in the mouse spot test though there are limitations in the methodology employed in both studies. Similarly, there are limitations in methodology in the available studies in germ cells that were all negative. Therefore, there is no robust evidence of a genotoxic potential in vivo.

### **6.2.7 Carcinogenicity**

Neither of the carcinogenicity bioassay conducted in the rat or hamster provided evidence of a carcinogenic potential.

### 6.2.8 Fertility

No evidence of an effect on fertility was seen in a two-generation dietary study in the rat. Furthermore, data from numerous repeat dose studies of 90-day or longer duration that examined the reproductive organs support the finding of the only available fertility study.

### 6.2.9 Developmental toxicity

No evidence of a developmental effect was seen with triclosan in robust studies in the rat at doses that produced marked maternal toxicity. Additionally, no evidence of a postnatal developmental effect was seen in this species. Similarly, no evidence of a developmental effect was seen at a level that produced marked maternal toxicity in the only available study in the rabbit. In the mouse, the only treatment related finding of delayed ossification of forelimb phalanges is considered to be a secondary non-specific consequence of maternal toxicity. Therefore, the available data in animals provide no evidence that triclosan has a direct effect on development. The NOAEL for both developmental and maternal toxicity is 50 mg/kg bw/day.

### 6.2.10 Antimicrobial resistance

#### Recent laboratory studies

Data from recent laboratory studies of resistance mechanisms, sometimes in clinical isolates, more usually with laboratory generated variants, have demonstrated the following four resistance mechanisms, which have been manipulated both genetically and biochemically in the laboratory:

- mutational change in gene(s) encoding the target enzyme EAR such that binding of triclosan and thus inhibition of EAR and fatty acid synthesis does not occur<sup>5</sup>;
- mutational changes leading to overproduction of EAR within the cell such that some molecules of EAR escape the inhibitor and thus remain active<sup>5</sup>;
- impermeability of the bacterial cell envelope such that triclosan does not readily penetrate beyond this outer layer of the cell<sup>6</sup>; and
- effects on efflux pumps that pump triclosan (usually along with other xenochemicals such as antibiotics and other biocides) from the cell such that the triclosan concentration does not rise to a damaging level within the cell<sup>5</sup>.

Though there is sometimes disagreement as to which mechanism(s) is primarily responsible for triclosan resistance, and whether more than one may be functioning, resistance mechanisms involving multidrug efflux pumps and outer envelope impermeability are likely to give rise to cross resistance towards other biocides and antibiotics.

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<sup>5</sup> A form of selected resistance.

<sup>6</sup> A form of intrinsic resistance.

## Studies from clinical or natural settings

Overall these recent environmental studies, and analyses of clinical isolates from collections taken over the course of time, point to the conclusion that the use of biocides (including triclosan) in homes and hospitals has not lead to any notable selection of antibiotic resistance in bacteria, nor where it has been examined, to cross resistance.

## Physiological fitness of bacteria resistant to biocides and antibiotics

By varied measures of physiological fitness, mutants of both *Pseudomonas aeruginosa* and *Stenotrophomonas maltophilia* that overproduce multi-drug efflux pumps were found to be significantly less fit than their parental wild types in a number of important regards. The mechanism by which this occurs remains for the moment a matter for speculation.

## Overall conclusions regarding antibiotic resistance

The studies up to 2002 reviewed both by the European Union Scientific Steering Committee (see EC Health & Consumer Protection Directorate-General, 2002a) and those studies published between 2002 and 2005 reviewed here, published since 2002, show that triclosan resistance is readily generated in a range of bacterial species (both gram positive and negative) by selection under laboratory conditions in partially inhibitory concentrations of triclosan. In contrast, triclosan resistant mutants are present at low frequency in natural (including clinical) isolates of gram-positive and gram-negative bacteria. Furthermore, investigations to determine if the frequency of triclosan resistance has risen as a result of exposure of natural populations of bacteria to this biocide have shown minor or no change. Therefore, the data suggest that conditions exist in laboratory experiments that differ in some important way(s) (unidentifiable from the experiments reported) from conditions which natural populations experience.

Hence, overall, the new studies available since the EU review provide no evidence that triclosan poses a risk to humans or to the environment by inducing or transmitting antibacterial resistance under current conditions of use. Though the recent limited number of studies do not resolve specific technical/use issues identified by the SSC (see EC Health & Consumer Protection Directorate-General, 2002a), and therefore the relationship between the use of biocides and the development of clinically relevant antimicrobial resistance should be kept under regular review.

A detailed analysis of the effects on laboratory animals and other test systems is provided in **Part 2, Section 18**.

## 6.3 Effects on human health

### 6.3.1 Skin irritation

There is evidence available that triclosan produces skin irritation in studies conducted with human volunteers, however, there was no evidence of phototoxicity.

### 6.3.2 Sensitisation

Although there is potential widespread consumer exposure to triclosan only a small number of case studies have been reported where humans who are not atopic have demonstrated positive reactions to triclosan. The available data indicate that at most triclosan possesses a very weak skin sensitisation potential.

There is also only very limited evidence for photo-sensitisation by triclosan in healthy volunteers or those with dermatological conditions.

### 6.3.3 Repeat dose toxicity

There was no evidence of a treatment related effect in a human tolerance study with oral doses of triclosan up to 30 mg/day for 15 days and 30 mg/day for 52 days. There has also been no evidence of adverse effects in human studies examining the use of personal care products containing triclosan.

A detailed analysis of the effects on human health is provided in **Part 2, Section 19**.

## 6.4 Regulatory classifications for workplace based hazards

The classification of the health effects of triclosan has been conducted according to the *Approved Criteria for Classifying Hazardous Substances* (the Approved Criteria) (NOHSC, 2004) or, in the case of physicochemical hazards, the *Australian Code for the Transport of Dangerous Goods by Road and Rail* (ADG Code) (FORS, 1998).

The Approved Criteria are cited in the *National Model Regulations for the Control of Workplace Hazardous Substances* (NOHSC, 1994c) and provide the mandatory criteria for determining whether or not a workplace chemical is hazardous.

### 6.4.1 Physicochemical hazards

Triclosan does not meet the ADG Code (FORS 1998) for classification as a dangerous good on the basis of physicochemical hazards.

### 6.4.2 Occupational health hazards

#### Acute toxicity

Based on the available animal data triclosan does not meet the Approved Criteria (NOHSC, 2004) for classification for acute oral and dermal toxicity.

The acute inhalation toxicity data in rats is limited. The dose used in the acute inhalation study was low with no deaths reported (LC50 >0.15 mg/L). However, based on effects seen after a single exposure in a repeat dose inhalation toxicity study, triclosan meets the Approved Criteria (NOHSC, 2004) for classification as 'Toxic by inhalation (R23)'.

#### Irritation and corrosive effects

Based on the human and/or animal data triclosan meets the Approved Criteria (NOHSC, 2004) for classification as irritating to eyes (R36), respiratory system (R37) and to skin (R38).

### **Sensitising effects**

Based on the available human and animal data triclosan does not meet the Approved Criteria (NOHSC, 2004) for classification as a skin sensitiser.

### **Effects from repeated or prolonged exposure**

Based on the available animal data triclosan does not meet the Approved Criteria (NOHSC, 2004) for classification as causing serious damage to health by prolonged exposure through inhalation, ingestion or dermal contact.

### **Genotoxicity**

Based on the available in vitro and animal data triclosan does not meet the Approved Criteria (NOHSC, 2004) for classification as a mutagen.

### **Carcinogenicity**

Based on the available animal data triclosan does not meet the Approved Criteria (NOHSC, 2004) for classification as a carcinogen.

### **Reproductive effects**

Based on the available animal data triclosan does not meet the Approved Criteria (NOHSC, 2004) for classification as a reprotoxicant, as a developmental toxicant, or for lactational effects.

## **6.5 Classification and labelling for public health hazards**

### **Scheduling in the SUSDP**

The acute oral toxicity of triclosan is greater than 5000 mg/kg bw in rats, and acute dermal toxicity is  $\geq 9300$  mg/kg bw in rabbits. Triclosan has a LC50 of less than 1300 mg/m<sup>3</sup> in rats with 2 h nose-only exposure (expected to be  $\leq 650$  mg/m<sup>3</sup> with 4 h exposure). It is a skin and eye irritant in rabbits. The eye irritation effects were not completely reversible by day 7 (mean score for cornea opacity = 1 on day 7).

Overall based on the toxicity profile, triclosan meets the Interim Guidelines of the NDPSC for scheduling (TGA, 2008).

Triclosan is used widely in a number of consumer products. Based on its inhalation toxicity and irritation effects, inclusion of triclosan in the Standard for the Uniform Scheduling of Drugs and Poisons (SUSDP) is considered appropriate with cut-offs and/or exemptions for consumer products.

Details of the hazard classification based on the above are provided in **Part 2, Section 20**.

## 7. Environmental Hazard Assessment

Ecotoxicity data for several trophic levels (animals and plants) from aquatic and terrestrial environments were available; however, the data were limited in quantity in all categories.

### 7.1 Wildlife

Based on the data available for two standard test species, triclosan is slightly toxic to birds by the oral route of exposure, with the lowest toxicity data for the bobwhite quail of LD50 862 mg/kg bw (single oral dose).

### 7.2 Terrestrial invertebrates

Triclosan exhibited very slight toxicity to earthworms, with a recorded no-observed-effect concentration (NOEC) of 1026 mg/kg dry wt. Triclosan did not effect the soil microbial processes of respiration and nitrification at concentration up to 2 mg/kg of dry soil.

### 7.3 Terrestrial plants

In soils, triclosan is toxic to plants when grown in sandy soil (time-weighted average (TWA) NOEC for cucumber 65 µg/kg); however, toxicity was less (TWA NOEC for cucumber 446 µg/kg) when grown in sandy loam. The attenuation of phytotoxicity is potentially due to the higher organic matter content of the sandy loam soil binding to the triclosan.

### 7.4 Aquatic organisms

Triclosan is very toxic to freshwater aquatic organisms with LC50 or EC50 values <1 mg/L (Mensink et al., 1995). From the limited data available, freshwater algae are the most sensitive species (lowest NOEC 0.5-0.69 µg/L and 72-96 h EC50 0.7-1.4 µg/L). Morphological analysis generally indicated enlarged cell sizes when algae were exposed to concentrations  $\geq 2.2$  µg/L. Data from two algae studies involving the assessment of recovery post-exposure indicates that algae growth resumed, hence, triclosan is algistatic rather than algicidal at concentrations up to 13 µg/L.

Data for freshwater algae indicates that ecotoxicity decreases slightly in the presence of dissolved organic matter (*S. subspicatus* E<sub>r</sub>EC50 3.5 µg/L), perhaps with adsorption to organic matter reducing bioavailability.

In both acute and chronic tests with freshwater invertebrates, EC50 values increase as pH increases, and triclosan is much more toxic to freshwater animals in neutral or acidic waters than in alkaline waters. For example, in a 7 d test with *C. dubia*, the maximum acceptable toxicant concentration (MATC) at pH 7 was ~30 times less than at pH 8.5. The above mentioned data for freshwater algae were obtained from toxicity tests performed under alkaline pH conditions (pH  $\geq 7.5$ ) and given the higher toxicity of triclosan to other aquatic animals under neutral to acidic

conditions, the toxicity values for algae may under-estimate algal toxicity through the full environmental pH range.

Triclosan was highly toxic to sediment dwelling organisms (the midge *Chironomus tentans* and the freshwater amphipod *Hyalella azteca*) when exposed through the water column. In contrast, exposure of the the midge *Chironomus riparius* to triclosan through spiked sediment showed no effect at concentrations up to 100 mg/kg dry sediment.

Limited data are available for the toxicity of triclosan to marine organisms. The available data indicates that triclosan is highly toxic to grass shrimp with larvae being the most sensitive life stage. Triclosan is also very highly toxic to the marine bacterium *Vibio fischeri*.

None of the aquatic toxicity tests undertaken investigated or discussed the actual mode of toxic action of triclosan in the aquatic organisms affected.

Preliminary data indicate that triclosan (or metabolite) is not potently estrogenic to freshwater fish but it may be weakly estrogenic, anti-estrogenic or androgenic.

## 7.5 Micro-organisms

Triclosan is an antimicrobial compound to many bacteria, fungi, moulds and yeasts. Some species are resistant to triclosan and others are able to use it as a sole carbon source. Effects occur in sensitive micro-organisms at concentrations of  $\geq 0.01$  ppm.

The limited data available indicate that effect levels of triclosan on activated sewage sludge micro-organisms vary depending on the level of acclimation. A concentration of 2 mg/L inhibited activated sludge micro-organisms that had not been acclimated to triclosan; however, the same concentration had no effect on acclimated organisms. Laboratory-derived IC50 values range from 20-239 mg triclosan/L based on carbon dioxide (CO<sub>2</sub>) evolution and glucose utilisation. Triclosan ( $\geq 2$  mg/L) had a slight effect on chemical oxygen demand (COD) removal under laboratory conditions, but had a major inhibitory effect on the nitrification process. Anaerobic sludge digestion was significantly inhibited at a concentration of 10 mg/L. A NOEC for sewage microbes was not available.

A detailed analysis of the effects on organisms in the environment is provided in **Part 2, Section 21**.

# 8. Human Health Risk Characterisation

## 8.1 Health risk characterisation methodology

A margin of exposure methodology is used frequently in international assessments to characterise risks to human health (EC, 2003a). The risk characterisation is conducted by comparing quantitative information on exposure to the NOAEL/NOAEC and deriving a Margin of Exposure (MOE) as follows:

1. Identification of critical effect(s).
2. Identification of the most appropriate/reliable NOAEL (if available) for the critical effect(s).
3. Where appropriate, comparison of the estimated or measured human dose or exposure (EHD) to provide a Margin of Exposure (MOE):  
$$\text{MOE} = \text{NOAEL}/\text{EHD}$$
4. Characterisation of risk, by evaluating whether the MOE indicates a concern for the human population under consideration.

The MOE methodology was used for characterising occupational and public health risk following exposure to triclosan.

The MOE provides a measure of the likelihood that a particular adverse health effect will occur under the conditions of exposure. As the MOE increases, the risk of potential adverse effects decreases. In deciding whether the MOE is of sufficient magnitude, expert judgment is required. Such judgments are usually made on a case-by-case basis, and should take into account uncertainties arising in the risk assessment process such as the completeness and quality of the database, the nature and severity of effect(s) and intra/inter species variability.

The MOE methodology was used for characterising occupational and public health risk following exposure to triclosan.

## 8.2 Occupational risk

### 8.2.1 Occupational exposures

All triclosan used in Australia is imported either as powder or liquid solution. Triclosan is also present in imported end-use products. Exposure to triclosan may occur during:

- Repacking;
- Formulation of personal care/cosmetic products, cleaning agents, and paints;
- Treatment of textiles;
- Plastics manufacture; and

- Use of triclosan-containing end-use products.

Potential routes of exposure to triclosan in the occupational setting are via inhalation and dermal contact. The likelihood of exposure by ingestion in occupational settings is expected to be low.

Exposure monitoring data were not available for levels of triclosan in the air for the different occupational exposure scenarios. Consequently, the UK EASE model (version II) was used to determine both inhalation and dermal exposure.

### 8.2.2 Critical health effects

Triclosan is of low acute toxicity via the oral and dermal routes. The limited acute inhalation toxicity data indicate a LC50 of less than 1300 mg/m<sup>3</sup> (equivalent to 1.3 mg/L) in rats for a 2 h exposure period. The critical health effects from acute exposure to triclosan are skin, eye and respiratory irritation. Skin irritation was observed in both humans and animals following dermal application. Only data in experimental animals are available for eye irritation, which was observed following instillation of triclosan into the eyes of rabbits.

For repeat dose toxicity, the available human data provide no reliable information to identify a robust NOAEL or profile the systemic toxicity of triclosan. Animal data are available for the oral, dermal and inhalation routes of exposure. Overall, the data from rodent studies indicates that the principal effect following ingestion and topical application of triclosan are hepatic effects, with local irritation often seen at the site of contact.

While the mouse is the most sensitive species, there is evidence that (unlike the rat and hamster) it is sensitive to peroxisome proliferator type effects in the liver that are not considered a risk to human health. Consequently, for ingestion a NOAEL of 40 mg/kg bw/day was identified in the rat for mild clinical chemistry and/or haematology changes, with hepatocyte hypertrophy and hepatocyte vacuolisation in cells from males only. In this species no reliable evidence of systemic toxicity was seen in a dermal study up to the top dose of 80 mg/kg bw/day. Irritation of the nasal tract and changes in clinical chemistry parameters were seen in rats in the only repeat dose inhalation study following exposure to an aerosol of triclosan in 10% ethanol at doses more than 50 mg/m<sup>3</sup> air.

Triclosan did not cause in vivo genotoxicity, carcinogenicity, reproductive or developmental toxicity in rodents.

### 8.2.3 Risk estimates

#### Physicochemical hazards

Triclosan is a non-flammable powder that does not undergo autoignition and has no evidence of an explosive property. It has a melting point of 54 °C to 57.3 °C and decomposition occurs at 280 °C to 290 °C.

Triclosan is stable under normal storage conditions, although solutions are not stable to chlorine and have only moderate stability in the presence of oxidising compounds. Triclosan itself has no oxidising properties. Triclosan powder, like other dusts, may be explosive if ignited when present at a critical concentration in air.

Based on the properties of triclosan the risk from physicochemical hazards during storage and handling of triclosan is considered to be low.

### **Acute risks due to occupational exposure**

The toxicological profile of triclosan indicates that contact with the raw material or concentrated solutions may result in skin and eye irritation. Inhalation of triclosan powder may cause toxicity and irritation to the respiratory tract.

Workplace activities related to triclosan are repacking, formulation, use in textile treatment, plastics manufacture and use of end use products.

Formulation of products containing triclosan is essentially an enclosed, automated process with closed mixing tanks employed.

Similarly at most workplaces textile treatment is essentially an enclosed, automated process. Solutions used contain triclosan at less than 20% concentration, with the exception of one workplace that used a powder containing 13.5% triclosan. At a wool processing plant site with daily exposure, a solution containing 3% or less triclosan is used. Similarly, cleaning of baths through which fabrics or articles are passed in the treatment of textiles is of little concern as the triclosan is very dilute at this point and the baths are first emptied of solution.

Manufacture of polyolefin masterbatches using raw triclosan occurs on a periodic basis. Other plastic manufacturing processes use solutions or plastic pellets containing triclosan. There is little potential for exposure where triclosan is used encapsulated in a plastic matrix, while when solutions are used the maximum concentration of triclosan is up to 10%.

The risk of acute effects such as inhalation toxicity, skin, eye and respiratory irritation during the processes described above is expected to be low due to periodic rather than daily exposure and use of engineering controls such as local exhaust ventilation (LEV) at some workplaces. In addition the reported use of PPE, such as safety goggles and gloves, at some sites would minimise exposure. Nevertheless, there is the possibility of accidental spills as manual handling procedures are employed during repacking, at some stages of formulation such as transfer of raw material to another container or mixing vessel, and during textile treatment such as transfer of triclosan solutions to another container, mixing vessel or dye machine. The manual handling stages are of short duration and potential exposure would normally be minimal. Therefore, overall, the potential for exposure to triclosan is low. Consequently, the risk to workers from handling triclosan is considered to be low.

### ***End-use products***

Workers may be exposed to triclosan through the use of commercial personal care and cleaning products containing the chemical. In addition to normal usage, acute exposure of end users to triclosan may occur following accidental spillage. Exposure would not be significant as the maximum concentration of triclosan identified in an occupational end-use product was only 0.3%. Consequently, the risk of acute effects such as inhalation toxicity, skin, eye and respiratory irritation is assessed as low.

### ***Secondary transfer***

Manual handling of triclosan and triclosan products occurred during the majority of the identified uses. In addition to the risk of inhalation toxicity, skin, eye and respiratory irritation from acute exposure to triclosan and/or products containing triclosan following direct skin contact and contact with air borne particles, secondary transfer from hands or gloves may also occur. The risk of secondary transfer is expected to be higher in the following situations:

- use of raw material or products containing high percentages of triclosan; and
- where PPE is not used.

PPE was reported to be used at all sites surveyed, and the risk of acute effects from secondary transfer is expected to be low.

### **Chronic risks**

There are no Australian or overseas worker health effects monitoring data available during repacking, formulation, treatment of textiles, and plastic manufacture or for use of end products. Consequently, the UK EASE model has been used to predict exposure, and a NOAEL of 40 mg/kg bw/day was selected for effects on the liver in rats (as described in Section 8.2.2) to calculate MOE for risk assessment. Table 8.1 provides MOEs calculated using the EASE model and the appropriate formulae are detailed in Appendix C.

In deciding whether the MOE is of sufficient magnitude for these occupational scenarios expert judgment is required taking into account the risk assessment process, the nature and severity of effect(s) on which the NOAEL is based, and intra/inter species variability.

With regards to the nature and severity of effects on which the NOAEL is based, not only are the critical effects seen in the two-year rat carcinogenicity study considered minimal but the minor histopathological changes seen in hepatic cells in males only were not consistently seen at the identified NOAEL (40 mg/kg bw/day) in interim sacrifice groups throughout the study. Furthermore, no increase in the severity of these histopathological changes was seen with dose (Part 2, Section 18.4). However, while the effects seen are minimal and the mechanism by which changes arise and their significance for human health is not clear, they cannot be dismissed as being of no significance. With regard to inter species variability there are no data to suggest that humans are more sensitive than animals.

**Table 8.1 – Calculated margins of exposure (MOE) for effects on the liver for each occupational exposure scenarios**

Process		Estimated body burden from		Combined body burden (µg/kg bw/day)	MOE (based on NOAEL of 40 mg/kg bw/day)
		Inhalation exposure (µg/kg bw/day)	Dermal exposure (µg/kg bw/day)		
Repacking (100% powder)	With LEV	0.25 - 0.64	5.6 - 55	5.8 – 55.6	6897 - 719
	Without LEV	0.64 – 6.36		6.1 – 61.4	6557 - 651
Formulation of end-use products (100% powder)	With LEV	15.9 – 39.7	85.5 - 855	101 - 895	396 - 44.7
	Without LEV	39.7 - 397		125 - 1252	320 - 31.9
Treatment of textiles (13.5% powder)	With LEV	2.14 – 5.36	11.5 - 115	13.6 – 120	2941 - 333
	Without LEV	5.36 – 53.6		16.9 – 169	2367 - 237
Treatment of textiles (assumed to be a 20% liquid) <sup>1</sup>	With LEV	-	17 - 170	17 - 170	2353 - 235
	Without LEV	-			
Plastic manufacture (100% powder)	With LEV	15.9 – 39.7	85.5 - 855	101 - 895	396 - 44.7
	Without LEV	39.7 - 397		125 - 1252	320 - 31.9
Plastic manufacture (10% liquid)	With LEV	-	8.6 - 86	8.6 - 86	4651 - 465
	Without LEV	-			
Use of end-use products (0.3%)	With LEV	-	16.7	16.7	2395
	Without LEV	-			

<sup>1</sup> Liquid products used in the manufacture of textiles contain >1% to < 20% triclosan. For determining a worst-case exposure scenario/MOE for textile manufacture it is assumed that the product contains 20% triclosan.

A MOE of 100 or greater is usually not considered a flag for concern as it represents the conservative default uncertainty factors of 10 each for both intra- and inter- species variability used for risk characterisation.

The MOEs for repacking, treatment of textiles, and plastics manufacture using 10% triclosan solution, with and without the use of LEV, were all >100 indicating that the risk of chronic effects to workers from repeated handling of triclosan in these scenarios is low. The MOE for the use of end-use products was also >100. Inhalation exposure was not estimated for the use of end-use products. Inhalation exposure may occur if commercial spray cleaning products are used. However, the triclosan concentration reported in cleaning products ranged from 0.04 % - 0.30%.

MOEs < 100 were identified for two scenarios. The MOE for formulation ranged from 44.7 – 396 with LEV and 31.9 – 320 without LEV, as did that for plastic manufacture when using raw triclosan powder.

The lower levels of the MOE in these ranges are considered likely to be overestimates. However, as the predictive dermal model is less developed than the inhalation model and its outputs should be regarded as no more than approximations. Furthermore, the semi-automated processes for formulation and plastic manufacture mean that dermal exposure is only likely during manual handling procedures such as transfer of raw material to another container or mixing vessel in the case of the formulation process and as these procedures are of short duration exposure would be minimal. Additionally, EASE does not quantify the actual protection provided by PPE, such as gloves. Risk of chronic effects will therefore be less at workplaces where PPE are used. Consequently, it is considered that the MOEs will be at the higher levels of the predicted range (i.e. >100) and the risk of chronic effects to workers in the formulation and plastics manufacture industry from repeated exposure to triclosan is low.

#### **8.2.4 Uncertainties in occupational risk estimates**

Uncertainties in any risk characterisation process arise from inadequate information, assumptions made during the process and variability in experimental conditions. The uncertainties inherent in the characterisation of risk for triclosan arise mainly from inadequate data and include:

- absence of representative atmospheric monitoring;
- absence of dermal exposure data;
- lack of data on the health effects of triclosan in humans following repeated exposures; and
- use of a default oral NOAEL for determination of MOE estimates as no reliable evidence of systemic toxicity was seen in dermal studies in a suitable animal model.

In addition, the assumptions used in EASE modelling add uncertainties to the risk characterisation.

#### **8.2.5 Areas of concern**

Risk characterisation has indicated that under occupational conditions the risk to workers of adverse health effects such as inhalation toxicity, skin, eye and respiratory irritation and chronic effects is low. However, the risk of skin, eye and respiratory irritation are likely to increase during accidental spills or leaks of triclosan and/or products containing high concentrations of triclosan, especially where PPE is not used.

### **8.3 Public health risk**

#### **8.3.1 Public exposure - Adults**

Exposure may occur through the use of consumer products containing triclosan. The major exposure scenarios are:

- Use of cosmetic and personal care products containing triclosan;
- Use of household products containing triclosan; and
- Use of articles containing triclosan.

Potential routes of exposure are via inhalation and dermal contact. The likelihood of exposure by ingestion is expected to be low in adults.

Measured exposure data are limited for consumer exposure scenarios. Some data are available for repeated use of cosmetic and personal care products. Consequently, exposure models have been used to predict consumer exposure to triclosan during use of various categories of products (see Part 2, Section 15.3) and, as conducted for occupational risk, a MOE methodology has been used by comparing quantitative information on exposure to the NOAEL/NOAEC and deriving a MOE (see Section 8.1 for further details).

The limited measured data available has been used to undertake risk characterisation by an alternative method, namely, comparing it with levels of total triclosan (i.e. triclosan and its metabolites) in the plasma at the identified NOAEL/NOAEC.

MOE = plasma levels at NOAEL/plasma level following use of consumer product.

As stated previously in sub-section 8.1:

- as the MOE increases the risk of potential adverse effects decreases;
- in deciding whether the MOE is of sufficient magnitude, expert judgment is required; and
- the critical health effects from acute exposure to triclosan are inhalation toxicity, skin, eye and respiratory irritation, and hepatic effects from repeated exposure.

### **8.3.2 Public health risk estimates - Adults**

#### **Acute risks**

The potential for acute inhalation toxicity, skin, eye and respiratory irritation could arise as a result of consumer use of:

- cosmetic and personal care products containing triclosan; and
- household products containing triclosan.

The concentration of triclosan in cosmetics and personal care products ranges from < 0.01 - 0.5% and in household goods ranges from 0.04 – 0.3%. At these low concentrations the risk of the irritant effects and inhalation toxicity of triclosan is not expected.

Additionally, textile and plastic articles containing triclosan do not present a risk for inhalation toxicity or skin, eye and respiratory irritation.

#### **Risks from repeated exposure**

Potential concerns for repeat dose toxicity arise from those consumer exposure scenarios that involve repeated exposure to triclosan. The use of cosmetic, personal care and household products and articles containing triclosan can all occur on a daily basis and so are relevant.

The calculations of consumer exposure for cosmetic and personal products, and household cleaning products using estimated data (see Part 2, Section 15.3 and 15.4

respectively) are based on the maximum levels of triclosan in each type of product in Australia. Furthermore a worst-case exposure scenario has been determined, that assumed a person was exposed to all possible types of product (e.g. deodorant, body lotion, facial mask etc) containing triclosan. Consequently, it is recognised that the determined body burdens and subsequent MOE range presented are unlikely to be applicable to all consumers (i.e. MOE's would be greater).

### **Measured plasma levels - cosmetic and personal care products**

There is potential for minor hepatic effects to arise as a result of repeated use of cosmetic and personal care products (see Section 8.2.3 chronic effects). Limited studies are available measuring plasma levels of total triclosan in volunteers following repeated use of triclosan-containing cosmetic and personal care products. Such studies are considered the most appropriate as repeated use allows steady state plasma levels to be reached. A NOAEL of 41 µg/mL (level in animal plasma) for hepatic effects was used to estimate MOE for repeated use of cosmetic and personal care products. A summary of appropriate studies with estimated MOEs derived from measured data are presented in Table 8.2.

The lowest MOE was 179 in male volunteers following use of a soap containing 1% triclosan. However, a MOE of 1367 – 2050 was obtained following use of an unspecified bath product containing a comparable amount of triclosan (0.75%). Similarly, while MOEs of 258 and 311 were seen in female and male volunteers following use of a toothpaste containing 0.3% triclosan, MOEs of 1595 – 2000 were seen following use of a dentifrice containing 0.28% triclosan.

Thus, considerable variation has been seen in plasma steady state levels of total triclosan in volunteers following single use of similar products containing similar concentrations of triclosan. Though the majority of the studies, including a multiple product use study with triclosan concentrations ranging from 0.28% – 0.75%, indicate MOEs >1000 which are considered satisfactory, there is evidence of MOEs of approximately 180 – 310 in some studies. Considering the nature of the health effect used to derive the NOAEL (minor histopathological changes in hepatic cells in male rats that were not consistently seen in interim sacrifice groups throughout a carcinogenicity study), all the derived MOEs are considered to indicate a low risk of chronic effects following repeated use of consumer products containing triclosan.

However, the above data does raise a potential concern, namely, that MOEs lower than those observed may be possible in some individuals through combined use of many products containing triclosan, and/or products containing relatively high concentrations of triclosan.

**Table 8.2 – Calculated margins of exposure (MOE) in adults for effects on the liver from measured exposure to cosmetic and personal care products**

Product	Plasma level of total triclosan in humans (ng/mL)	MOE based on plasma levels <sup>1</sup>
<i>Single product use</i>		
Hand wash (1%)	229 <sup>2</sup> 158 <sup>3</sup>	179 259
Unspecified bath product (0.75%)	Approx 20 - 30	2050 - 1367
Dentifrice (0.2%)	26.7	1536
Dentifrice (0.28%)	22.7 <sup>4</sup> 25.7 <sup>5</sup> 20.5 <sup>6</sup>	1806 1595 2000
Toothpaste (0.3%)	132 <sup>2</sup> 159 <sup>3</sup>	311 258
<i>Multiple product use</i>		
Soap bar (0.75%) Deodorant (0.39%) Dentifrice (0.28%)	36.7 <sup>4</sup> 34.3 <sup>5</sup>	1117 1195

<sup>1</sup> Calculation based on animal plasma level of total triclosan at the NOAEL: plasma level of 41 µg/mL which is the combined average of male and female values observed at 3, 6, 12, 18 and 24 months.

<sup>2</sup> Maximum level observed in male volunteers

<sup>3</sup> Maximum level observed in female volunteers

<sup>4</sup> Maximum level observed in Caucasian (white) volunteers

<sup>5</sup> Maximum level observed in Negroid (black) volunteers

<sup>6</sup> Maximum level observed in Mongoloid (oriental) volunteers

### **Risks from repeated exposure to cosmetic and personal care products using estimated exposure data**

Information on triclosan concentrations in cosmetic and personal care products was available, however, there was no information on Australian use patterns. Consequently, use patterns and exposure models were adopted from the EU technical guidance document on risk assessment (EC, 2003a) and guidance note for the testing of cosmetic ingredients and their safety evaluation (SCCNFP, 2003). The estimated MOEs are presented in Table 8.3.

**Table 8.3 – Calculated margins of exposure (MOE) in adults for effects on the liver from estimated exposure to cosmetic and personal care products**

Inhalation (µg/kg bw/day)	Dermal (µg/kg bw/day)	Oral (µg/kg bw/day)	Combined body burden (µg/kg bw/day)	MOE (based on NOAEL of 40 mg/kg bw/day)
18.0 - 53.9	145.5	24.4	187.9 - 223.8	212.9 - 178.7

The body burden level used to estimate MOE is derived from a worst-case scenario, where exposure is assumed to all products containing triclosan by all exposure routes. The estimated MOE range is therefore, a worst-case range. It is expected that for the majority of the population the MOE will generally be at the higher end of the predicted range. However, there may be a subgroup of consumers who repeatedly use multiple products containing triclosan. In this subgroup the MOE may be at the lower end of the predicted range. The estimated MOE range is considered to indicate a low risk by chronic effects following repeated use of consumer products containing triclosan in the majority of the population.

### **Estimated risk from repeated exposure - household cleaning products**

Information was only available on triclosan levels in Australian household cleaning products, and the use patterns and exposure models were adopted from the EU technical guidance document on risk assessment (EC, 2003a). These estimated MOEs are presented in Table 8.4.

**Table 8.4 – Calculated margins of exposure (MOE) in adults for effects on the liver from estimated exposure to household cleaning products**

<b>Inhalation (µg/kg bw/day)</b>	<b>Dermal (µg/kg bw/day)</b>	<b>Oral (µg/kg bw/day)</b>	<b>Combined body burden (µg/kg bw/day)</b>	<b>MOE (based on NOAEL of 40 mg/kg bw/day)</b>
1.7 - 349.5	0.34	ND	2.04 - 349.84	19608 - 114.3

The estimated MOE range is a worst-case scenario based on exposure to a number of household cleaning products and is unlikely to be reflective of normal use. Exposure to all the cleaning products is also unlikely to occur on a daily basis. It is therefore considered that the realistic MOE is likely to be at the higher end of the predicted range. Consequently, the risk of chronic effects from the use of household products containing triclosan is low.

### **Estimated risks from repeated exposure – articles**

Minimal data on triclosan levels in articles in use in Australia is available. Furthermore, potential dermal and oral exposure from articles containing triclosan could not be determined as no data on the migration of triclosan from such are available. Dermal exposure is based only on contact with painted surfaces. Inhalation exposure was calculated using the OECD Environmental Directorate model (OECD, 1993) and is likely to be an overestimate. However, the derived MOE (see Table 8.5) still indicates that the risk of chronic effects from the use of articles containing triclosan is low.

**Table 8.5 – Calculated margins of exposure (MOE) in adults for effects on the liver from estimated exposure to articles**

Inhalation (µg/kg bw/day)	Dermal (µg/kg bw/day)	Oral (µg/kg bw/day)	Combined body burden (µg/kg bw/day)	MOE (based on NOAEL of 40 mg/kg bw/day)
3.8	0.56	No Data	4.4	9090

***Summary of estimated risks from repeated exposure in adults***

Table 8.6 below provides a summary of the data including an overall body burden and MOE when major exposure scenarios are combined. In determining an overall total body burden and MOE for cosmetic and personal care products estimated exposure data has been used, as values are reported in µg/kg bw/day as for other scenarios. Additionally for cosmetic and personal care products, though the MOEs derived from measured data were generally >1000 some studies had values similar to the estimated range.

**Table 8.6 – MOEs in adults for exposure scenarios to triclosan**

Public exposure scenario	Combined body burden (µg/kg bw/day)	MOE (based on NOAEL of 40 mg/kg bw/day)
Cosmetic & personal care products	187.9 - 223.8	212.9 - 178.7
Household cleaning products	2.04 - 349.84	19608 - 114.3
Article surfaces	4.4	9090
<b>All exposure scenarios combined</b>	193.7 - 577.4	206.5 - 69.3

Individual public exposure scenarios showed MOE ranges greater than 100, indicating low risk (Table 8.6). The higher value in the predicted MOE range for all exposure scenarios combined is similar to that for cosmetic and personal care products, indicating that the greatest contributor to the overall body burden are from the use of such products. The lower MOE value less than 100 in the predicted MOE range for combined scenario (MOE = 69.3) indicates that there could be risks when using cosmetic and personal care products, and household cleaning products together with exposure to article surfaces by the same person.

**8.3.3 Public exposure - children**

This section focuses on babies and young children up to five years old. Exposure to this sub-group may occur from direct contact with consumer products containing triclosan. The major exposure scenarios are:

- Use of personal care products containing triclosan; and
- Use of articles containing triclosan.

In addition, exposure to triclosan may also occur via breast milk in breast-fed babies.

Potential routes of exposure are via inhalation and dermal contact, and the oral route from the sucking or mouthing of textile articles and/or breast-feeding.

As for consumer exposure to adults, measured exposure data is limited and exposure models have been used to predict exposure to various categories of products. Additionally, a MOE methodology was undertaken.

#### 8.3.4 Risk estimates - children

##### Acute risks

As for adults (see Section 8.3.1) the risk of inhalation toxicity, skin, eye and respiratory irritation as a result of exposure to personal care products is low. Articles containing triclosan present a low risk.

##### Risks from repeated exposure

Limited data are available on the concentration of triclosan and patterns of use for triclosan containing products that may be used on children. In determining exposure to these products assumptions have been made regarding application volume to children (see Section 4.2.2 for more details). Consequently, it is likely that the predicted exposure levels may be over-estimates.

##### *Estimated risks from repeated exposure - personal care products*

Dermal exposure was estimated for use of a body lotion on babies and children up to five years old. Additionally, oral exposure was estimated from use of toothpaste in children between one and five years. The data is presented in Table 8.7.

**Table 8.7 -Calculated margin of exposure (MOE) in children for effects on the liver from estimated exposure to cosmetic and personal care products**

Age (years)	Dermal ( $\mu\text{g}/\text{kg}$ bw/day)	Oral ( $\mu\text{g}/\text{kg}$ bw/day)	Combined body burden ( $\mu\text{g}/\text{kg}$ bw/day)	MOE (based on NOAEL of 40 mg/kg bw/day)
<1	85	N/A	85	471
2	84	15.5	99.5	402
5	56	10.3	66.3	603

The estimated MOEs are for a worst-case scenario, as the maximum concentration of triclosan in Australian consumer goods likely to be used by children have been taken to determine body burdens. The estimated MOEs for all the age groups are >100 and are based on the minor health effects observed in animals at the selected NOAEL of 40 mg/kg bw/d. Thus, these MOEs for children are considered to indicate a low risk of chronic effects following repeated use of multiple consumer products containing triclosan.

### *Estimated risks from repeated exposure – articles*

The OECD Environmental Directorate model (OECD, 1993) was used to determine exposure from a textile or plastic article based on the chemical's vapour pressure. However, no data is available on the leaching of triclosan from articles that would allow potential oral or dermal exposure to be predicted. It is considered that oral and dermal exposure to triclosan through use of articles containing triclosan will be low and, thus, the contribution to the total body burden is likely to be negligible. Dermal exposure to triclosan through contact with painted surfaces has been estimated based on 100% availability from the thin films. The data is presented in Table 8.8 and the derived MOEs indicate that the risk of chronic effects from exposure to articles containing triclosan is low.

**Table 8.8 - Calculated margin of exposure (MOE) in children for effects on the liver from estimated exposure to articles**

Age (years)	Body burden - inhalation (µg/kg bw/day)	Body burden - inhalation (µg/kg bw/day)	Body burden - total - (µg/kg bw/day)	MOE (based on NOAEL of 40 mg/kg bw/day)
Infant				
<1	7.40	5.90	13.30	3007
Children				
2	5.27	0.46	5.73	6981
5	4.28	0.42	4.70	8511

### *Estimated risks from repeated exposure – breast milk*

Exposure was determined in exclusively breast-fed babies based on the maximum level of total triclosan observed in an Australian breast milk study (see Appendix E). The data is presented in Table 8.9 and the derived MOEs indicate that the risks of chronic effects from exposure through breast milk containing triclosan is low.

**Table 8.9 - Margin of exposure (MOE) in children for effects on the liver from the maximum level of total triclosan measured in Australian breast milk**

Age (month)	Body burden - oral - (µg/kg bw/day)	MOE (based on NOAEL of 40 mg/kg bw/day)
1	3.04	13 158
2	2.46	16 260
3	2.22	18 018
4	2.10	19 048

### *Summary of estimated risks from repeated exposure in children*

Table 8.10 below provides a summary of the data including an overall body burden and MOE when major exposure scenarios are combined. As stated previously the derived MOEs are likely to be overestimates. As for adults, it can be seen that the predicted MOE for all exposure scenarios combined is similar to that for cosmetic and personal care products, indicating that the greatest risk by far comes from the use of cosmetic and personal care products. Furthermore, Table 8.10 also indicates that the lowest risk for children is from exposure through breast-milk containing triclosan.

**Table 8.10 – MOEs in children for exposure scenarios to triclosan**

Source of exposure	Age (years)	Combined body burden ( $\mu\text{g}/\text{kg bw}/\text{day}$ )	MOE (based on NOAEL of 40 mg/kg bw/day)
Cosmetic & personal care products	<1	85	471
	2	99.5	402
	5	66.3	603
Breast milk	<1	3.04	13 158
Article surfaces	<1	13.30	3007
	2	5.73	6981
	5	4.70	8511
<b>All sources of exposure combined</b>	<1	95.44	395
	2	104.77	380
	5	70.58	564

#### **8.3.5 Uncertainties in public risk estimates**

Uncertainties involved in the public health risk characterisation for both adults and children result from database limitations. There is a lack of Australian data on the use patterns of consumer products containing triclosan to allow a realistic exposure assessment. Additionally there is very limited measured data, meaning that generally exposure models have been used to determine the sources of exposure that represent the greatest risk to consumers. Such models are not as reliable as measured data as they mostly use conservative assumptions.

#### **8.3.6 Areas of concern**

The available information indicates that public use of triclosan products and hence potential exposure is widespread. The risk characterisation has indicated that under normal conditions of consumer use the risk of adults and children being exposed to levels of triclosan that would lead to adverse health effects such as inhalation toxicity, skin, eye and respiratory irritation and chronic effects is low. Of all the sources of exposure the risk estimation indicated that the lowest MOEs, using modelled data, were from combined use of triclosan containing products (cosmetic and personal care products, cleaning products and exposure to article surfaces).

However, the use patterns of triclosan-containing products vary greatly among individuals.

Some studies in humans show a high level of exposure following use of a single cosmetic or personal care product. This raises concerns that chronic health effects may potentially occur in some individuals through the combined use of a range of cosmetic and personal care products containing triclosan, or use of certain products containing relatively high concentrations of triclosan. However, these limited incidences are not reflective of general consumer exposure.

#### **8.4 Biological monitoring data**

The population-based biological monitoring data are believed to be a more accurate predictor of aggregate exposure because not only are the data triclosan specific, they are also based on actual consumer use of the various triclosan products as they naturally co-occur (US EPA, 2008). However, there are uncertainties in the biological monitoring data.

In the US EPA aggregated risk assessment, the population-based biological monitoring data based on spot urine concentrations were obtained from the National Health and Nutrition Survey. Based on the results at the mean and 99<sup>th</sup> percentile, the aggregated risk to triclosan from all uses did not trigger a risk of concern (using an oral NOAEL of 30 mg/kg bw/d in baboons MOEs were >100 even for the most conservative dose and conversion method) (US EPA, 2008).

## 9. Environmental Risk Characterisation

### 9.1 Environmental release and degradation

This section provides a characterisation of risks to the Australian environment from use of triclosan as an anti-microbial agent. A risk quotient (RQ) approach has been used to predict the risk to aquatic organisms, terrestrial (soil-dwelling) organisms, and wildlife associated with aquatic and terrestrial environments. To predict an acceptable environmental risk using the RQ approach, the quotient of the predicted environmental concentration (PEC) divided by the predicted no effect concentration (PNEC) needs to be 1 or less (i.e.  $RQ \leq 1$ ).

#### 9.1.1 Environmental release of triclosan

Environmental release of triclosan is unlikely during importation, storage and transportation. Containers of triclosan will be transported directly from port facilities to several industrial facilities throughout Australia for use in the manufacture of various products. In addition, triclosan in finished products will be imported (in ready-to-use products and articles) and distributed throughout Australia for consumer use. Accidental spills, leaks and catastrophic mechanical failure during a transport accident are the most likely reasons for environmental release. Engineering controls (e.g. container specifications) and emergency clean-up procedures (i.e. spill response instructions on Material Safety Data Sheet and label) will limit the impact on the environment of such incidents.

Triclosan is incorporated into cosmetics and personal care products that after application may wash off directly into natural surface waters, such as during bathing and primary aquatic recreational activities. Due to the uncertainty in estimating environmental release by this pathway, no predicted environmental concentration (PEC) in surface waters has been derived. Greater risks to the aquatic environment would be more likely in populated areas during peak usage times, and in surface waters with limited flow or longer hydraulic residence time (e.g. ponds, lakes).

#### 9.1.2 Wastewater treatment plant systems

The majority of triclosan used in Australia will eventually be washed off or otherwise enter the Australian sewerage system where it will mix with a wide range of chemical and biological constituents typically found in wastewater. Most wastewater treatment plants (WWTPs) and sewage treatment plants (STPs) rely on microbial processes to enable treatment and degradation of wastewater constituents. In general, wastewater with inhibitory levels of contaminants may reduce or cease the treatment plant's ability to degrade wastewater constituents, potentially resulting in the release of poorly treated wastewater into the environment in effluent and sludge with potential adverse impacts.

Although triclosan has anti-microbial properties, the international literature indicates that microbial biodegradation processes (e.g. secondary treatment) enable significantly greater treatment and degradation of triclosan relative to primary treatment processes. This indicates that the communities of sewage microorganisms within the STPs tested were capable of treating some if not most

of the triclosan during operational conditions. In addition, variable rates of biodegradation of triclosan, particularly through mineralisation to CO<sub>2</sub>, have been demonstrated over time in laboratory tests involving sewage sludge microorganisms exposed to triclosan at concentrations in the range of 10-5000 µg/L, and in one long-term study with a triclosan concentration of 500 000 µg/L (Table 16.3). Furthermore, specific microorganisms have been identified in sewage sludge that are capable of partially mineralising triclosan through a series of co-metabolic steps.

However, biodegradation studies indicate that the rate of biodegradation of triclosan is generally proportional to exposure time and inversely proportional to triclosan concentration, and the degradation rate probably also depends on the microbial community and whether it has been acclimatised to triclosan as well as the environmental conditions (e.g. aerobic versus anaerobic; Table 16.3). At elevated concentrations, triclosan may potentially inhibit sewage microbes, species or communities. At concentrations of up to 20000 µg/L, triclosan was not readily or inherently biodegradable under aerobic conditions and the inability to degrade triclosan is attributed to inhibition of microbial growth as a 3-hour IC<sub>50</sub> of 20000 µg/L has been reported. Inhibition of growth, CO<sub>2</sub> evolution and nitrification have been reported under aerobic conditions at concentrations ≥600 µg/L; however, some studies show that inhibition at an exposure concentration of 2000 µg/L may be relatively minor if microbes have been pre-exposed to low concentrations of triclosan. A lag phase before degradation has also been demonstrated in some microbial degradation studies.

For triclosan, anaerobic microbial degradation, a process used in many STPs, is slow relative to aerobic biodegradation. In one study, ~91% of the extractable residues remained as <sup>14</sup>C-triclosan after 147 days of incubation with anaerobic inoculum and anaerobic conditions (Springborn Laboratories Inc., 1994a). Microbial analysis of the sludge during the study indicated that the sludge microbes remained viable.

Based on the estimated use and disposal pattern for triclosan in Australia, a mean annual wastewater concentration for triclosan in the Australian sewerage system of 14500-17400 ng/L (see Section 16.5) has been estimated. Recently, triclosan levels have been measured in the influent of five Australian STPs in concentrations ranging from 573 to 845 ng/L, and triclosan concentrations in secondary and tertiary treated effluents have been observed in the range 23-434 ng/L (with a median measured concentration of 108 ng/L and a mean of 142 ng/L) from 19 STPs (Ying and Kookana, 2007). This compares with a range of <100-740 ng/L in effluent from three primary treated STPs measured in the mid 90s. As a comparison, the available monitoring data from international sources indicate triclosan concentrations in untreated wastewater in the range of <100-562000 ng/L and secondary and tertiary treated effluents of 10-2700 ng/L (see Table 16.54). Hence, estimated concentrations of triclosan in Australian sewage systems are at the lower end of the range of concentrations measured internationally. However the study by Ying and Kookana (2007) did not cover many urban STPs (none in the largest urban areas in Australia) and as a result the full range of triclosan concentrations in influent and effluent across Australia is not yet clear.

STP monitoring studies indicate that a relatively high rate of removal of triclosan (95%) can be achieved after secondary (activated sludge and trickling filter) treatment processes even when the influent triclosan concentration was 7500-

21900 ng/L (Sabaliunas et al., 2003). However, in another STP, removal efficiency of only 35%-42% was achieved after secondary treatment (aerobic digestion) of influent containing triclosan in the range of 30100-37800 ng/L, though at the same STP, a lower influent triclosan concentration of 1300-2600 ng/L resulted in a higher treatment efficiency (69%). At relatively low influent concentrations, the available data from STP monitoring do not indicate a particular trend of decreasing treatment efficiency with increasing triclosan concentration; however, at higher influent concentrations, treatment efficiency apparently declines. Although this apparent trend may potentially be related to inhibition of the microbial processes by triclosan (as discussed above), it is not possible to extrapolate between STPs due to the different treatment processes and environmental conditions and other factors may have also resulted in the lower treatment efficiency of triclosan when at relatively high influent concentrations. The available data has been collected from a range of treatment processes across a number of countries. The measured removal rate for five STPs (3 tertiary and 2 secondary) ranged between 72-93%. However, given that this is a small sample of the nearly 900 STPs across Australia it is uncertain how representative this would be of the wider Australian sewage treatment system. Consequently, the following assessment has been conducted based on the ranges removed that have been observed in international data.

## 9.2 Aquatic risk

### 9.2.1 Predicted No Effect Concentration (PNEC) for triclosan

Although triclosan has been used in Australia and internationally for many years, and has been discharged into aquatic receiving environments (freshwater, estuarine, marine) in treated wastewaters and other products, no published freshwater or marine water or sediment quality guidelines were available from the Australian or international literature for triclosan for the protection of aquatic ecosystems. Consequently, predicted no effect concentrations (PNECs) have been derived in this assessment from the ecotoxicity data available using recognised Australian methodology for deriving guidelines (ANZECC and ARMCANZ, 2000).

While no published guidelines are available, several other authors/agencies have also derived PNECs or equivalent values for triclosan for the protection of freshwater organisms:

- The Danish Environmental Protection Agency (Danish EPA, 2003b), using the European Union principles for derivation of Predicted No Effect Concentrations (PNECs), derived a  $PNEC_{\text{aquatic}}$  of 0.05  $\mu\text{g/L}$  for triclosan by dividing the lowest available NOEC, for a freshwater alga (0.5  $\mu\text{g/L}$ ; RCC, 1995), by a safety factor of 10.
- The quality and reliability of the algae toxicity data available for triclosan have been reviewed elsewhere by Hanstveit and Hamwijk (2003) and the NOEC of 0.69  $\mu\text{g/L}$  (*S. subspicatus*; ABC Laboratories, 1997a) was considered the most reliable value in their view, stating that the lower NOEC of 0.5  $\mu\text{g/L}$  (RCC, 1995) was from a test which had discrepancies between the control and acetone control that cast doubt on the incubation conditions used. A PNEC of 0.069  $\mu\text{g/L}$  was derived by dividing this NOEC by an assessment factor of 10.

- The abovementioned  $\text{PNEC}_{\text{freshwater}}$  of 0.069  $\mu\text{g/L}$  ( $\sim 0.07 \mu\text{g/L}$ ) is also referred to in a Briefing Note prepared by the Environment Agency (2004) and a report of the fate of triclosan in wastewater treatment (Thompson et al. 2005).

Recent work by Veldhoen et al. (2006) on the effects of triclosan on the development of tadpoles of the North American bullfrog, *Rana catesbeiana* demonstrated alterations in gene expression at concentrations of 0.03  $\mu\text{g/L}$  triclosan. Precocious hormonally-induced metamorphosis was recorded after exposure to concentrations of  $0.15 \pm 0.03 \mu\text{g/L}$  triclosan. In isolation, this work is not considered sufficient demonstration that exposure to such concentrations during development in the wild would result in lasting adverse effects such as reduced survivorship. Nevertheless, these early results do indicate the potential for interference with thyroid hormones at extremely low concentrations.

While this study in isolation is considered insufficient to determine the regulatory endpoint, these data contribute to the weight of evidence that adverse effects are likely to occur at concentrations below those measured in the field.

### 9.2.2 Risk to freshwater ecosystems

Aquatic toxicity data are available for four freshwater taxa with 13 species (acute studies) and three taxa with 10 species (chronic studies).

The aquatic toxicity data available indicate that the green algae (*Scenedesmus subspicatus*) and the cyanobacterium (*Anabaena flos-aquae*) are relatively sensitive to the adverse effects of triclosan (refer Table 21.5). A  $\text{PNEC}_{\text{freshwater}}$  of 0.05  $\mu\text{g/L}$  (50 ng/L) is adopted for this assessment based on dividing the NOEC, for the freshwater alga *Scenedesmus subspicatus* (0.5  $\mu\text{g/L}$ ; RCC, 1995), by a safety factor of 10, in line with the Danish Environmental Protection Agency (Danish EPA, 2003b). This is in agreement with the PNEC of 58 ng/L determined using the protective concentration of 0.29  $\mu\text{g/L}$  to protect 95% of species derived using BurrliOZ modelling and applying an assessment factor of 5 in line with the EC Technical Guidance Document (Appendix H). It is also a compromise with the PNEC of 20 ng/L which would be derived from the lowest reported NOEC for green alga (0.2  $\mu\text{g/L}$  for the green alga *Pseudokirchneriella subcapitata*) and applying the same assessment factor of 10.

The derived risk quotients in Table 9.1 based on the predicted environmental concentrations near STP outlets indicate that the current use rate of triclosan presents an unacceptable risk to freshwater organisms. A risk to aquatic organisms is indicated for each type of wastewater treatment considered, from primary to tertiary; moreover, in each case this is so even at the lowest predicted concentration. It should be noted that higher levels of wastewater treatment are applied where the receiving water is freshwater. Unacceptable risk quotients are also predicted based on the limited Australian measured triclosan levels for untreated wastewater, treated effluent and surface waters. The risk quotients for the measured Australian data are much lower than those for the predicted concentrations.

**Table 9.1 - Risk Quotients ( $PEC_{\text{Freshwater}}/PNEC_{\text{Freshwater}}$ ) for freshwater ecosystems based on removal rates for sewage treatment levels**

Level of Treatment	Removal rate (%) <sup>a</sup>	$PEC_{\text{Freshwater}}$ (triclosan, $\mu\text{g/L}$ )	$PNEC_{\text{Freshwater}}$ (triclosan, $\mu\text{g/L}$ )	$\frac{PEC_{\text{Freshwater}}}{PNEC_{\text{Freshwater}}}$
Untreated wastewater	---	14.5-17.4	0.050	290-347
Primary Treatment	2-96	0.581-17	0.050	12-341
Secondary Treatment				
Trickling Filter	58-96	0.581-7.3	0.050	12-146
Activated Sludge	55-99	0.145-7.82	0.050	3-156
Activated sludge (SimpleTreat)	61-72	4.07-6.78	0.050	81-136
Tertiary treatment	87- $\geq$ 99	$\leq$ 0.145-2.26	0.050	3-45
<b>Measured Australian Data</b>				
		$PEC_{\text{Freshwater}}$ (triclosan $\mu\text{g/L}$ )	$PNEC_{\text{Freshwater}}$ (triclosan, $\mu\text{g/L}$ )	$\frac{PEC_{\text{Freshwater}}}{PNEC_{\text{Freshwater}}}$
Untreated Effluent		0.573-0.845	0.050	11-17
Treated Effluent		0.023-0.740	0.050	0.46-14.8
Surface Waters		0.014-0.070	0.050	0.28-1.4
International surface waters		$\leq$ 2.300 <sup>b</sup>	0.050	$\leq$ 46

a Removal rate obtained from literature sources (see Section 16.5). Freshwater PEC values obtained by dividing the estimated effluent concentration by receiving environment dilution factors of 1. Data are from Table 16.6.

b excludes levels near manufacturing facility

### 9.2.3 Risk to marine ecosystems

Many large sewerage systems discharge into marine environments, but there is a paucity of aquatic toxicity data for triclosan to marine organisms. In the absence of adequate marine toxicity data, the  $PNEC_{\text{freshwater}}$  of 0.05  $\mu\text{g/L}$  is adopted as a  $PNEC$  for marine waters for this assessment. This approach is supported by a preliminary review of comparative freshwater and marine ecotoxicity data by ECETOC (2003).

**Table 9.2 - Risk Quotients ( $PEC_{\text{Marine}}/PNEC_{\text{Marine}}$ ) for marine ecosystems based on removal rates for sewage treatment levels**

Level of Treatment	Removal rate (%)*	$PEC_{\text{Marine}}$ (triclosan, $\mu\text{g/L}$ )	$PNEC_{\text{Marine}}$ (triclosan, $\mu\text{g/L}$ )	$\frac{PEC_{\text{Marine}}}{PNEC_{\text{Marine}}}$
Untreated wastewater	---	1.5-1.7	0.050	29-34.7
Primary Treatment	2-96	0.058-1.7	0.050	1.2-34.1
Secondary Treatment				
Trickling Filter	58-96	0.058-0.73	0.050	1.2-14.6
Activated Sludge	55-99	0.015-0.78	0.050	0.3-15.6
Activated sludge (SimpleTreat)	61-72	0.41-0.68	0.050	8.1-13.6
Tertiary treatment	87- $\geq$ 99	$\leq$ 0.015-0.23	0.050	0.3-4.5
<b>Measured Australian Data</b>				
		$PEC_{\text{Marine}}$ (triclosan $\mu\text{g/L}$ )	$PNEC_{\text{Marine}}$ (triclosan, $\mu\text{g/L}$ )	$\frac{PEC_{\text{Marine}}}{PNEC_{\text{Marine}}}$
Untreated Effluent		0.0573-0.0847	0.050	1.1-1.7
Treated Effluent		0.0023-0.0740	0.050	0.046-1.48
Surface Waters		0.0014-0.0070	0.050	0.028-0.14

\* Removal rate obtained from literature sources (see Section 16.5.2). Marine PEC values obtained by dividing the estimated effluent concentration by receiving environment dilution factors of 10.

The derived risk quotients in Table 9.2 based on the predicted environmental concentrations indicate that the current use rate of triclosan also presents an unacceptable risk to marine organisms, though the risk quotients are significantly lower than the corresponding freshwater situation. Risk is only marginal from treated effluent based on the available although quite limited Australian figures. While significant quantities of Sydney's wastewater (~75%) are discharged to the marine environment after receiving only high flow primary treatment, Table 9.2 includes data from three of the major plants, including Malabar STP which is responsible for the highest value. However, the risk is acceptable to the Australian marine environment once a minimum level of dilution has taken place.

#### 9.2.4 Risk to sediment dwelling organisms

Toxicity data are available for two freshwater taxa with 2 species. The most sensitive organism is the amphipod *Hyalella azteca* which has a reported LC50 of 200  $\mu\text{g/L}$  (Dussault et al. 2008). Based on this endpoint and applying an assessment factor of 1000 (based on the lack of data) the  $PNEC_{\text{sediment}}$  for sediment dwelling organisms has been determined to be 0.2  $\mu\text{g/L}$ . It is inappropriate to use the result of 100 mg/kg for exposure to spiked sediment as aquatic organisms will be exposed through the water column and also the triclosan used in this study was strongly bound to the sediment. The derived risk quotients in Table 9.3 based on the predicted environmental concentrations near STP outlets indicate that the current use rate of triclosan presents an unacceptable risk to sediment dwelling

organisms. A risk to sediment dwelling organisms is indicated for each type of wastewater treatment considered, from primary to tertiary; moreover, in each case this is so even at the lowest predicted concentration. Once again, it should be noted that higher levels of wastewater treatment are applied where the receiving water is freshwater. Unacceptable risk quotients are also predicted based on the limited Australian measured triclosan levels for untreated wastewater and treated effluent. However, the risks based on the measured levels in Australian surface waters are acceptable.

**Table 9.3 - Risk Quotients ( $PEC_{SDO}/PNEC_{SDO}$ ) for Sediment Dwelling Organisms (SDO) based on removal rates for sewage treatment levels**

Level of Treatment	Removal rate (%) <sup>a</sup>	$PEC_{SDO}$ (triclosan, $\mu\text{g/L}$ )	$PNEC_{SDO}$ (triclosan, $\mu\text{g/L}$ )	$\frac{PEC_{SDO}}{PNEC_{SDO}}$
Untreated wastewater	---	14.5-17.4	0.2	72-87
Primary Treatment	2-96	0.581-17	0.2	2.9-85
Secondary Treatment			0.2	
Trickling Filter	58-96	0.581-7.3	0.2	2.9-36
Sludge				
Activated	55-99	0.145-7.82	0.2	0.72-39
Activated sludge (SimpleTreat)	61-72	4.07-6.78	0.2	20-34
Tertiary treatment	87-≥99	≤0.145-2.26	0.2	0.72-11.3
<b>Measured Australian Data</b>				
		$PEC_{SDO}$ (triclosan $\mu\text{g/L}$ )	$PNEC_{SDO}$ (triclosan, $\mu\text{g/L}$ )	$\frac{PEC_{SDO}}{PNEC_{SDO}}$
Untreated Effluent		0.573-0.845	0.2	2.86-4.22
Treated Effluent		0.023-0.740	0.2	0.115-3.7
Surface Waters		0.014-0.070	0.2	0.07-0.35
International surface waters		≤2.300 <sup>b</sup>	0.2	2.86-4.22

a Removal rate obtained from literature sources (see Section 16.5). Sediment Dwelling Organisms PEC values obtained by dividing the estimated effluent concentration by receiving environment dilution factors of 1. Data are from Table 16.6.

b excludes levels near manufacturing facility

Sediment dwelling organisms also include microbial populations that have been shown to play an important role in the recycling of essential elements such as carbon, nitrogen and phosphorus (Alongi 1994 and Costanzo et al. 2005). The continual discharge of triclosan has the potential to disrupt these microbial populations possibly affecting the recycling of these nutrients.

In addition, there are data to suggest that triclosan accumulates in sediments that are distant from catchment sources (Singer et al., 2002), as well as the persistence of triclosan in sediments.

### 9.3 Terrestrial risk

Most of the triclosan used each year will, after use, be sent to sewer where treatment in STPs is expected to account for degradation of much of the triclosan present. However, a fraction entering the sewerage system is expected to partition in the sludge phase with a proportion remaining in the treated water. This is supported by international monitoring data from a range of unit processes. Hence, terrestrial organisms may be exposed to triclosan through contact with STP sludge or treated water containing triclosan. The following sections look at the risk associated with exposure to these sources of triclosan in the environment.

#### 9.3.1 Risk associated with sludge and biosolids from STPs containing triclosan

Traditionally, sewage sludge was disposed of to landfill or incinerated and these practices continue in parts of Australia (e.g. incinerated in the Australian Capital Territory). However, an increasing proportion is being reclaimed as biosolids and re-used for soil conditioning (see Appendix A). For example, in Sydney in 2002-3, Sydney Water Corporation captured solids to the equivalent of ~51000 dry tonnes of biosolids of which 100% was used for soil conditioning applications in agriculture (60%), forestry (20%-35%), land rehabilitation, landscaping and horticulture (5%-20%). The use of biosolids as a soil conditioning agent results in the exposure of soil dwelling organisms such as earthworms as well as crops through their roots and seeds, to triclosan.

Limited data is available for soil dwelling organisms; NOEC values are available for six plant species (Table 21.2) and earthworms (1026 mg/kg dry wt; Section 21.2). Based on this data, an assessment factor of 50 has been adopted for this assessment. The lowest available NOEC is for cucumber of 96 µg/kg (dry wt) (see Table 21.2) resulting in a PNEC of 1.92 µg/kg dry wt. This endpoint is less conservative than the PNEC<sub>soil</sub> of 0.096 µg/kg (dry wt) derived by the Danish Environmental Protection Agency (2003b), based on dividing the lowest available NOEC by a safety factor of 1000. This PNEC was considered preliminary due to the lack of soil ecotoxicity data available.

The predicted soil (PEC<sub>soil</sub>) concentrations outlined in Section 16.5.7 (Table 16.63) derived for use of STP biosolids as a soil conditioner have been used to estimate the risk to soil dwelling organisms resulting from this practice (Table 9.3).

The calculated PEC<sub>soil</sub>/PNEC<sub>soil</sub> ratios presented in Table 9.3 for the predicted biosolids concentrations indicate potential risks to soil dwelling organisms through the use of sewage sludge contaminated with triclosan as a soil conditioner when used at a rate of 10 tonnes of biosolids per hectare per annum. The PEC<sub>soil</sub>/PNEC<sub>soil</sub> ratios derived from the Australian measured data range from an acceptable risk at the lowest measured concentration (0.07 µg/kg dry weight) to an unacceptable risk at the highest measured concentration (129 µg/kg dry weight), with the large range reflecting that of the measured concentrations. Ying and Kookana (2007) reached a similar conclusion based on their data, but with an assessment factor of 1000 (RQ ≤1360).

**Table 9.4 - Risk Quotients ( $PEC_{Soil}/PNEC_{Soil}$ ) for soil dwelling organisms based on the use of sewage sludge as a soil conditioner.**

Parameter	Environment Australia (2003) STP model		ASTE (2004) and Dillon (2000) model <sup>a</sup>		Measured Australian Data <sup>d</sup>	
Estimated fraction in sludge based on SimpleTreat model (%)	55% <sup>b</sup>	61% <sup>c</sup>	55% <sup>b</sup>	61% <sup>c</sup>	-	-
$PEC_{Soil}$ (biosolid application) ( $\mu\text{g}/\text{kg}$ dry wt)	735	815	614	681	0.07	129
Lowest NOEC ( $\mu\text{g}/\text{kg}$ dry wt)	96	96	96	96	96	96
Assessment Factor	50	50	50	50	50	50
$PNEC_{soil}$ ( $\mu\text{g}/\text{kg}$ dry wt)	1.92	1.92	1.92	1.92	1.92	1.92
$PEC_{soil}/PNEC_{soil}$	383	424	320	355	0.36	67

Notes: The SimpleTreat model output refers only to an activated sludge treatment process. a. Based on a sludge generation rate of 100 kg/ML of wastewater. b. Assumes inherently biodegradable. c. Assumes no biodegradation. d. Ying and Kookana (2007).

Consequently, the calculated risk quotients for both the predicted and some of the measured levels of triclosan in biosolids indicate an unacceptable risk when biosolids from STPs are used as soil ameliorants. Given the small amount of the Australian data available it is unclear how representative this is of the levels in biosolids produced across Australia.

### 9.3.2 Risk associated with triclosan containing effluent from STPs for irrigation

The irrigation of crops with sewage effluent containing triclosan will also result in exposure to soil dwelling organisms. The risk to soil dwelling organisms as a result of this practice has been determined based on the  $PEC_{soil}$  for irrigation presented in Table 16.63 and the  $PNEC_{soil}$  determined above. These are summarised below in Table 9.5.

**Table 9.5 - Risk Quotients ( $PEC_{Soil}/PNEC_{Soil}$ ) for soil dwelling organisms based on the use of treated effluent for irrigation.**

Parameter	Environment Australia (2003) STP model		ASTE (2004) and Dillon (2000) model <sup>d</sup>		Measured Australian Data	
$PEC_{Soil}$ (irrigation) ( $\mu\text{g}/\text{kg}$ dry wt)	52.1 <sup>a</sup>	37.4 <sup>a</sup>	43.6 <sup>a</sup>	31.3 <sup>a</sup>	0.18 <sup>b</sup>	5.7 <sup>b</sup>
$PNEC_{soil}$ ( $\mu\text{g}/\text{kg}$ dry wt)	1.92	1.92	1.92	1.92	1.92	1.92
$PEC_{soil}/PNEC_{soil}$	27.1	19.5	22.7	16.3	0.09	2.97

<sup>a</sup>. See Table 16.63 <sup>b</sup>. Determined in an analogous manner to the values derived in Table 16.63 with the range of measured triclosan effluent concentrations of 0.023-0.740  $\mu\text{g}/\text{L}$  from Ying and Kookana (2007).

Once again the generated  $PEC_{soil}/PNEC_{soil}$  of Table 9.5 indicate that there is a potential risk to soil dwelling organisms at a total application rate of 1 m depth irrigation water per hectare per annum, including the higher end of the range of measured Australian data. The peak soil concentration is likely to be significantly reduced by degradation between irrigation events and deeper movement into the soil. While a potential risk is still indicated (risk quotients approximately 2-3) if it is assumed the triclosan from a single irrigation event delivering 10 cm is adsorbed in the surface 10 cm of soil, based on PECs estimated from modelling, the risk would be acceptable based on PECs estimated from measured Australian biosolid levels. Recent studies indicate that triclosan will degrade relatively rapidly in aerobic soils which would mitigate potential risks. However, triclosan will persist if the soil is anaerobic.

### 9.3.3 Risks to water-associated wildlife

#### Derivation of the toxicity reference values (TRVs) for birds and mammals

Effects on algae and other aquatic organisms indicated above (Table 9.1) may be sufficient to indirectly impact higher organisms through reduction in their food supply. The potential for direct toxicity from exposure to triclosan to arise in wildlife should also be considered. This may arise from dietary exposure (exacerbated by bioaccumulation in the food chain), from drinking surface water, or from incidentally or accidentally ingesting sediment.

Due to data limitations, avian and mammalian oral toxicity reference values (TRVs) have been derived using an assessment (safety) factor (AF) approach rather than more sophisticated and accurate statistical approaches that are data intensive. The Approximation Approach of United States Army Center for Health Promotion and Preventive Medicine (USACHPPM, 2000) has been adopted for this assessment to derive TRVs as this method is applicable to small sets of toxicity data. The methodology is scientifically based, and is an important internationally published guide that describes the rationale and methods for deriving wildlife TRVs.

Studies are available for the mouse, rat, hamster rabbit, dog and baboon. The data indicate that the mouse is the most sensitive species to the systemic toxicity of triclosan. A LOAEL of 25 mg/kg bw/day was identified from a 13-week study in mice for effects on haematology parameters, relative liver weight and total cholesterol in both sexes.

An avian repeat dose oral toxicity data of adequate quality is available for triclosan and has been used to derive the avian TRV. The five day repeat dose in diet oral NOAEL of 179 mg TCS/kg bw/day for Bobwhite quail, based on the study by Bio-Life Associates (1993c), has been selected to derive the avian TRV for triclosan as this was the highest dose tested that resulted in no adverse effects (mortality).

Oral TRVs derived for the assessment of risks from exposure to triclosan by mammals and birds and the aquatic TRV have been presented in Table 9.6. A high level of confidence of wildlife health protection is afforded by the derived TRVs for triclosan.

Wildlife TRVs are expressed as an acceptable dose of chemical by a specific exposure route (e.g. oral, inhalation, or dermal) or as an acceptable environmental media concentration (e.g. mg/kg of soil). The derived TRVs have been used in an analogous manner to PNEC values in the risk quotient approach to predict the risks to mammalian and avian wildlife.

**Table 9.6 - Derived mammalian and avian oral TRVs for triclosan (TCS)**

<b>Taxa and Toxicity Data</b>	<b>Reference</b>	<b>AF</b>	<b>Derived TRV (mg TCS/kg bw/day)</b>
<u>Mammals (mg TCS/kg bw)</u>			
Sub-Chronic LOAEL: 25 mg TCS/kg bw/day	Chapter 18	20	1.25
<u>Birds (mg TCS/kg bw)</u>			
Acute NOAEL (Bobwhite mortality): 179 mg TCS/kg bw/day	Bio-Life Associates (1993c)	30	6.0

AF = Assessment Factor

### Wildlife exposure

In general, wildlife may potentially be exposed to one or more environmental media (e.g. surface waters, sediments, soils, air), each of which may potentially contain triclosan, and multi-media exposure may occur concurrently (e.g. oral, dermal and/or inhalation). Triclosan has a high affinity to lipids and a high propensity for bioaccumulation, as indicated by biological tissue residue monitoring conducted in other parts of the world, and wildlife may be exposed to triclosan through consumption of foods (food chain or secondary exposure).

Total exposure to environmental media by wildlife may be estimated using the following model equation:

$$\text{Exposure}_{\text{total}} = \text{Exposure}_{\text{oral}} + \text{Exposure}_{\text{dermal}} + \text{Exposure}_{\text{inhalation}} \quad (\text{Eq. 1})$$

In the above equation, oral exposure routes are considered more likely to occur or be relatively more significant for triclosan. Although all potential pathways for exposure to triclosan have been considered in this assessment, oral exposure routes are of greatest importance (e.g. food consumption, drinking water, incidental sediment ingestion). Triclosan is not volatile and inhalation exposure is unlikely to be a significant exposure pathway for wildlife. Although dermal absorption of triclosan can potentially occur, there is considerable uncertainty in estimation of dermal uptake rates by wildlife from exposure to solutions containing triclosan. In general, features such as oily fur and feathers and toughened skin, are likely to reduce the potential for skin contact with environmental media and absorption (Sample et al., 1997).

For this assessment, it is assumed that wildlife obtain all of their food and water within the area of contamination based on the calculated PEC values (Appendix B Tables B-2 to B-5). The derived PECs for food intake are based on the consumption of biota allowing for a BCF of 5000 based on data for fish (see Section 16.4.2), which may be different for the organisms in the wildlife diet. Further, wildlife that have home ranges of size greater than the area of contamination will likely have less exposure than animals with smaller home ranges. Exposure may be seasonal or intermittent for migratory species of wildlife relative to sedentary species. In addition, their prey may move in and out of contaminated areas, thereby the potential for bioaccumulation of triclosan in prey may be less than for sedentary prey.

### **Avian and mammalian risk**

The risk to birds and mammals for the ingestion of triclosan through the intake of water, food and sediment has been estimated by comparing the PECs for the various routes of exposure (See Appendix B Tables B-3, B-4, B-5 and B-6) with the TRVs of 6.0 and 1.25 triclosan/kg bw/day derived for birds and mammals respectively.

As a worst case, wildlife triclosan intake rates by birds and mammals during drinking have been estimated using the upper value PEC surface water values ( $\leq 17.4 \mu\text{g/L}$ ) and the wildlife exposure model equations for surface water ingestion (see Appendix B). Using this method, the maximum bird and mammal intake rates of triclosan by the drinking water exposure route are  $\leq 0.005 \text{ mg/kg bw/day}$ .

Wildlife triclosan intake rates by birds and mammals from incidental or intentional sediment ingestion have been estimated using PEC sediment and the wildlife exposure model equations for sediment ingestion (see Appendix B). Using this method, the maximum bird and mammal intake rates of triclosan by the sediment exposure route are  $\leq 3.6 \text{ mg/kg bw/day}$ .

Triclosan in the aquatic environment has the potential to bioaccumulate in aquatic organisms and food chain exposure by wildlife may occur. Estimated wildlife dietary intake of triclosan has been presented in Tables B-2, B-3, B-4 and B-5 (Appendix B) based on a BCF of 5000 (derived from fish data). This is considered to be highly protective. Dietary exposure for birds to triclosan is estimated in the range of 3.3-126 mg/kg bw/day (freshwater) and 0.3-12.6 mg/kg bw/day (marine) depending on taxa, weight and the discharge source. The food chain potentially provides a significantly greater level of exposure than other routes of exposure evaluated. A similar scenario may be expected for mammals.

The calculated risk quotients (PEC/TRV) for the total oral intake (sum of the water, food and sediment exposure routes) are presented below (Table 9.7). The risk quotients for the various modes of intake are presented in Appendix G. The risk quotients for drinking water of various levels of water treatment are all below 0.01 and the maximum risk quotient for sediment intake was 0.6, indicating that these modes of intake are not expected to present a risk to wildlife.

The risk quotients in Table 9.7 indicate that at worst-case predicted concentrations for each type of sewerage treatment, there is a potential risk for birds and mammals ingesting biota from downstream of STP outfalls at all levels of treatment except for those exposed to tertiary treated wastewater discharging to the marine environment. Calculations based on the maximum measured concentration in Australian surface waters indicate there is not a potential risk for birds that are solely dependent on the aquatic compartment for their food and water intake. Similar calculations for mammals indicate a potential risk for mammals that are solely dependent on the freshwater aquatic environment for their food and water intake. However, most Australian mammals are not solely dependent on the freshwater aquatic compartment for both their food and water intake and are therefore unlikely to reach the above levels of exposure. The exception to this may be the platypus.

The potential risk to platypuses has been evaluated using the mammalian TRV and the potential exposure as determined in Section 16.5.10. The risk quotients for total exposure (water, sediment and biota) are in the range 4.6-7.6 for the predicted secondary treatment effluent scenarios, the minimum level of treatment expected for discharges to inland rivers suggesting potential risk to platypuses. However, calculations based on the maximum measured concentration in surface waters of 0.070 mg/L (Ying and Kookana 2007) yields risk quotients of 0.5-0.7 indicating no potential risk to platypus. It should be noted that the PECs derived for food exposure are based on extrapolating a BCF of 5000 from fish data to the freshwater invertebrates that make up the bulk of the platypus diet.

**Table 9.7 - Estimated Risk Quotients (PEC/TRV) for birds and mammals (0.01-1.0 kg bw) potentially exposed to freshwater and marine ecosystems containing triclosan released in STP effluent, based on removal rates for sewage treatment levels**

Effluent Source	Body weight (kg live wt)	Birds Total Oral PEC/TRV		Mammals Total Oral PEC/TRV	
		Freshwater	Marine	Freshwater	Marine
Untreated wastewater	0.01 kg	21.6	2.2	54.8	5.5
	0.1 kg	9.7	1.0	43.5	4.3
	1.0 kg	4.3	0.4	34.5	3.5
Primary Treatment	0.01 kg	21.2	2.1	53.7	5.4
	0.1 kg	9.5	0.9	42.6	4.3
	1.0 kg	4.2	0.4	33.9	3.4
Trickling Filter	0.01 kg	9.1	0.9	23.0	2.3
	0.1 kg	4.1	0.4	18.3	1.8
	1.0 kg	1.8	0.2	14.5	1.5
Activated Sludge	0.01 kg	9.7	1.0	24.6	2.5
	0.1 kg	4.4	0.4	19.6	2.0
	1.0 kg	2.0	0.2	15.5	1.6
Activated sludge (SimpleTreat)	0.01 kg	8.4	0.8	21.4	2.1
	0.1 kg	3.8	0.4	17.0	1.7
	1.0 kg	1.7	0.2	13.5	1.3
Tertiary treatment	0.01 kg	2.8	0.3	7.1	0.7
	0.1 kg	1.3	0.1	5.7	0.6
	1.0 kg	0.6	0.06	4.5	0.4
Measured Australian Data	0.01 kg	0.9	0.09	2.3	0.2
	0.1 kg	0.4	0.04	1.9	0.2
	1.0 kg	0.2	0.02	1.5	0.1

#### 9.4 Risk from degradation products

Little or no toxicity data is available for the triclosan degradation products. Hence, it is not possible to quantify the risks associated with these compounds. However, these compounds are only formed in small quantities from triclosan and any steps put in place to mitigate the potential risks posed by triclosan will mitigate any potential risks associated with the degradation products.

#### 9.5 Data gaps

The following significant data gaps were identified when undertaking the risk characterisation.

##### Monitoring data

Triclosan has been used in Australia for many years with most eventually being disposed to sewer. There is some local monitoring data (Ying and Kookana, 2007) which indicates that it is found in sewage influent (five samples; 573 to 845 ng/L), effluent (19 samples; 23 to 434 ng/L) and in surface waters near STP outfalls (five rivers, 14 to 75 ng/L) at levels exceeding the freshwater PNEC of 50 ng/L. Earlier

data indicate levels of <100-740 ng/L in primary treated effluents from three major Sydney STPs. However, apart from these the identities as well as details of sewage treatment and receiving waters are not known. There is also limited Australian STP monitoring data, and environmental monitoring data, for the methylated or chlorinated derivatives of triclosan. A survey during this assessment found that none of the major Australian wastewater utilities routinely monitor wastewaters, effluent or receiving environments/ecological receptors (e.g. shellfish, fish) for triclosan.

Triclosan has a high affinity to sediments, where it may be stable in anaerobic conditions in the long term based on international sediment core dating research, and biota; however, no sediment or tissue monitoring studies have been undertaken for triclosan or methyl-triclosan in Australia.

Triclosan has a high affinity to STP sludge; however, limited monitoring data are available on the concentration of triclosan in these sludges, or in biosolids, used as a soil fertility conditioner in Australia.

Triclosan is known to photolyse to various chlorinated derivatives including 2,7/2,8-dichlorodibenzo-p-dioxin (DCDD). No STP or field monitoring data were available on the occurrence of these dioxins in effluent or surface waters in the aquatic environment. Very limited toxicity data are available for these compounds as well.

### **Microbial effects**

Triclosan is widely used due to its known anti-microbial properties. However, there is a paucity of microbial toxicity data or field monitoring data on the ecological effects of triclosan.

### **Toxicity data**

While there are toxicity data for triclosan for a few species of mammals and birds (none for reptiles or amphibians), there is a paucity of data for native Australian wildlife for triclosan, and no toxicity data for the methylated product of triclosan. Ethical constraints on vertebrate testing will mean this gap is unlikely to be filled. International studies indicate that the methylated product of triclosan, has a higher affinity to bioaccumulate and is present in biota at comparatively higher concentrations. Further, comparisons between Australian native species and standard test species have shown that where data is available the variation is within a factor of 10. Therefore, this would be accounted for within the interspecies variation allowed for in the assessment factors applied in determining the PNECs.

There is also a lack of data available for the toxicity of triclosan and methyl-triclosan to sediment dwelling organisms. There have been triclosan related effects observed on the development of tadpoles of the North American bullfrog, *Rana catesbeiana* although the biological significance of these results is unknown.

Further investigation of the effects of triclosan on soil micro-organisms and nutrient recycling should be carried out given the potential for significant quantities of triclosan to find its way into the terrestrial environment due to the use of biosolids for soil amelioration.

## 9.6 Conclusions

The annual import volume and use pattern of triclosan has been used to estimate potential levels of triclosan entering Australian STPs. The estimated potential levels between 14.5-17.4 µg/L are 17-20 times higher than the highest observed influent concentration (0.845 µg/L), but these data are quite limited. The estimated concentrations are at the lower end of those observed overseas (<0.10-562 µg/L). These influent levels have been used to derive PECs for Australian freshwater and marine environments based on varying levels of wastewater treatment. For freshwater, the predicted levels range between ≤0.1-15.2 µg/L, are consistent with the observed levels overseas of 0.01-269 µg/L, but higher than the limited Australian effluent data (0.023-0.74 µg/L) that are available. Measurements of triclosan in Australian surface waters have found triclosan levels between 0.014 and 0.075 µg/L.

Based on the predicted concentrations and the use of triclosan and subsequent release to the Australian sewage system at current levels of use, is likely to result in concentrations of the chemical within natural waterways which indicate potential risk to aquatic ecosystems at all levels of wastewater treatment. Algae and aquatic plants are the most susceptible organisms. However, if the levels obtained close to the outfall from five Australian rivers are representative of all Australian conditions then the risk is only marginal. Given the significant difference between the predicted and limited measured levels it is important to determine how reflective the measured data is of the wider Australian environment. However, based on the limited measured Australian data it is likely that the growth of sensitive algal species downstream of some sewage outfalls is inhibited by exposure to triclosan residues in the discharged effluent. As the distance from the outfall increases, the level of triclosan is expected to be reduced through a combination of photolysis and absorption (see Section 16.5.5), and at least with ocean outfalls through further dilution of the effluent plume. The distance downstream of the point of release triclosan before levels fall below harmful levels has not been estimated in this assessment, but it is noted that in Europe triclosan has been detected at levels >50 ng/L approximately 20 km downstream (see Figure 16.13).

There is potential for indirect effects on birds and mammals to occur near STP outlets resulting from effects of triclosan on their food supply, and also direct toxicity arising primarily through food consumption based on the predicted levels of triclosan in surface waters. However, the endpoint on which the mammalian wildlife TRV value is based is a sub-chronic endpoint and therefore highly protective, and the avian value is also considered conservative. Furthermore, direct toxicity has been assessed assuming bioaccumulation occurs in the entire diet to a similar extent to that found in laboratory studies with fish, and that triclosan concentration in water is at the upper end of predicted concentration ranges for various types of sewage treatment, which results in a very conservative assessment. When the highest measured concentration in Australian surface waters is used the potential effects are predicted for mammals and not for birds. However, as noted above, the predictions are based on the mammals being solely dependent on the freshwater aquatic environment for food. This is unlikely to be the case for most Australian mammals with the exception of the platypus. Calculations based on platypus specific data and the maximum measured Australian surface water

concentration indicate that there is an acceptable level of risk to platypuses living in the vicinity of a sewage outfall.

A potentially unacceptable risk toward soil dwelling organisms as a result of the use of biosolids (as soil conditioners) or effluent (for irrigation) from STP has also been indicated based on the levels of triclosan potentially present in the sludge and effluent.

**DRAFT**

# 10. Current Human Health Risk Management

## 10.1 Occupational health and safety

### 10.1.1 Assessment of current control measures

According to the *National Model Regulations for the Control of Workplace Hazardous Substances* (NOHSC, 1994c), exposure to hazardous substances should be prevented or, where this is not practicable, adequately controlled so as to minimise risks to health and safety. The *National Code of Practice for the Control of Workplace Hazardous Substances* (NOHSC, 1994) provides further guidance in the form of a hierarchy of control strategies, namely:

- Elimination;
- Substitution;
- Isolation;
- Engineering controls;
- Safe work practices; and
- Personal protective equipment (PPE).

These measures are not mutually exclusive and effective control usually requires a combination of these strategies.

#### **Elimination and substitution**

Elimination is the removal of a chemical from a process and should be the first option considered in minimising risks to health. In situations where it is not feasible or practical to eliminate the use of a chemical, substitution should be considered. Substitution includes replacing with a less hazardous substance or the same substance in a less hazardous form.

Triclosan is not manufactured in Australia but is imported both as the raw chemical and as an ingredient in products. Of all the applicants providing data, only one was actively seeking to replace triclosan in their product with a less hazardous chemical. Therefore, elimination and substitution do not appear to be being locally pursued. Substitution and elimination of triclosan may not have been considered as it is less hazardous than a number of other antimicrobial agents.

#### **Isolation**

Isolation as a control measure involves separation of the process from employees by distance or the use of barriers or enclosure to prevent exposure. In this regard, the following controls were identified for triclosan and products containing triclosan:

- Stored in original containers in a cool dry ventilated chemical storage area, not segregated and no special storage precautions taken;

- Polymer pellets are stored above ground, with restricted access to employees; and
- All raw materials, triclosan included, are stored in a banded dangerous goods area segregated from the rest of the site.

### **Engineering controls**

Engineering controls are plant or processes which minimise the generation and release of hazardous substances. They include enclosure or partial enclosure, local exhaust ventilation and automation of processes.

The engineering controls in place to prevent exposure to triclosan vary and include:

- Triclosan is delivered to site in 25 kg fibre drums and dispensed from a laminar flow dispensing booth into plastic bags, which are then dispensed as needed to be mixed with other ingredients to make bulk product;
- Weighing of triclosan in a ventilated and extracted dispensing area. At the manufacturing area material are added to the mixer under local extraction and the batch is filled into containers through a sealed system;
- Mechanical air extraction at point of weighing and point of addition of triclosan to product;
- During weighing and dispensing activities, a stand-alone dust extraction system is used to collect air-borne particles; and
- Automated mixing systems.

### **Safe work practices**

Safe work practices have an important role in reducing exposure to triclosan. Work practices vary and include:

- Restricted access to triclosan, reducing the number of employees exposed;
- The mixture is left to stand for 24 h before transport by road to contract filler. Safety and emergency procedures are detailed in the relevant Material Safety Data Sheet (MSDS) located within the dispensary;
- Triclosan is weighed out in a ventilated room;
- For weighing out and dispensing, a plastic bag is placed into another plastic bag and the chemical is dispensed into this double plastic bag arrangement to minimise the risk of puncturing and hence exposure; and
- Workers follow written standard operating procedures, which cover receipt and storage, dispensing, cleaning equipment and waste disposal. The chemical's MSDS is required to have been read and understood by those handling triclosan. Following weighing and/or dispensing, overalls and gloves are immediately disposed of as hazardous waste.

### **Personal protective equipment (PPE)**

PPE is used to minimise exposure to or contact with chemicals. PPE should be used in conjunction with other controls and not as a replacement. Where other control measures are not practicable or adequate to control exposure, then PPE

should be used. From the information submitted some companies used PPE alone whilst others used PPE in combination with engineering controls. Furthermore, submitted details on PPE varied with some companies simply advising wearing of “appropriate” PPE whilst other detailed the type of equipment to be used.

For workers handling triclosan the PPE used are mainly to protect the hands, face and eyes. In addition, inhalation of dusts is also minimised by appropriate PPE. Instructions given for PPE were:

- Waterside transport and warehouse workers wear coats, overalls and heavy duty gloves;
- During weighing, operators wear overalls, safety glasses, gloves and appropriate respiratory protections (e.g. a face mask or dust mask fitted with particulate dust cartridge);
- Workers wear overalls, protective eyewear, footwear, face masks etc in accordance with Workcover requirements;
- Workers wear PPE including overalls, PVC gloves, safety glasses and appropriate respiratory protection. Handled as per supplier’s MSDS;
- Workers follow written standard operating procedures and wear P3 Powered Air Purifying Respiratory protection which includes a hood, disposable overalls, and impervious gloves; and
- Prior to formulation of the product, workers wear gloves, eyeglasses, protective clothing, boots and organic respirator. After formulation, workers wear all these PPE except for the respirator.

### **10.1.2 Hazard communication**

#### **Labels**

The *National Code of Practice for the Labelling of Workplace Substances* (NOHSC, 1994a) is applicable to labels for workplace substances. Labels of consumer products are required to comply with the *Standard for the Uniform Scheduling of Drugs and Poisons* (SUSDP) (NDPSC, 2003). Triclosan is currently not listed in the SUSDP. Triclosan is classified as a hazardous substance in the Australian Safety and Compensation Council (ASCC) Hazardous Substances Information System (HSIS).

Labels submitted for assessment were assessed for requirements under the *National Code of Practice for the Labelling of Workplace Substances* (NOHSC, 1994a). The assessment took the form of a qualitative appraisal, which included the following categories of information:

- Substance identification;
- Hazard category/signal word;
- ADG Code classification/packaging group;
- Details of manufacturer or supplier;
- Risk information (or phrase);
- Safety information (or phrase);

- Information on spills/leaks or fires; and
- Reference to MSDS.

Triclosan is classified as a hazardous substance in HSIS, and depending on the concentration, labels for products containing triclosan should contain the following hazard classification, risk and safety phrases based on the HSIS classification prior to July 2008:

**Classification of mixtures containing triclosan as presented in HSIS prior to July 2008:**

<b>Triclosan Concentration</b>	<b>Risk Phrases</b>	<b>Classification of Mixtures</b>
$\geq 25\%$	R23	Toxic
$3\% \leq \text{conc} < 25\%$	R20	Harmful

The most appropriate safety phrases are:

- S38: In case of insufficient ventilation, wear suitable respiratory equipment
- S45: In case of accident or if you feel unwell seek medical advice immediately (show the label where possible)
- S60: This material and its container must be disposed of as a hazardous waste

This assessment supports the additional classification for triclosan.

Additional risk and safety phrases may be applicable in products depending on the presence of other hazardous ingredients.

The labels provided by applicants were assessed against the classification appearing in the HSIS prior to July 2008. No safety phrases were given under the HSIS for triclosan prior to July 2008.

A total of four labels for triclosan raw material were provided (two for different grades from the same company). One label gave the concentration though all identified the substance. Three labels included the UN number of 3077 and of those, two gave a packaging class of 9. The EU risk phrases (for skin and eye irritation) were given in three and no label gave the risk phrase as provided in HSIS prior to July 2008 (toxic by inhalation). Therefore, the ADG Class for toxicity (6.1) or the relevant UN number was not provided on any label. Although no safety phrases are mandated all labels presented adequate safety precautions/instructions. Information on spills was provided on three labels.

## **MSDS**

Under NOHSC *National Model Regulations for the Control of Workplace Substances* (NOHSC 1994c) and the corresponding State and Territory legislation, suppliers are required to provide MSDS to their customers for all hazardous substances. Employers must ensure that a MSDS, prepared in accordance with the NOHSC *National Code of Practice for the Preparation of Material Safety Data Sheets* (NOHSC 1994b), is readily accessible to employees with potential exposure to triclosan used in the workplace. A sample MSDS for triclosan prepared in accordance with this Code is provided in Appendix I. This sample MSDS is for

guidance only. Under the NOHSC MSDS Code, manufacturers and importers have the responsibility of compiling their own MSDS and to ensure information is up-to-date and accurate.

In April 2003, NOHSC declared under the NOHSC Act, the *National Code of Practice for the Preparation of Material Safety Data Sheets 2nd Edition* (NOHSC, 2003) (MSDS Code). The MSDS Code forms part of the Hazardous Substances Framework and the revision addressed various technical elements and facilitates Australia remaining consistent with international approaches to hazard communication. The major focus of the revised MSDS Code however is to incorporate the information provisions of the *National Standard for the Storage and Handling of Workplace Dangerous Goods* (NOHSC, 2001). Notification of the declaration appeared in the *Commonwealth Government Notices Gazette* of 23rd July 2003 and the *Commonwealth Chemical Gazette* of 5th August 2003.

In declaring the MSDS Code, NOHSC decided that it should not come into effect under Commonwealth, State and Territory regulations until 24 April 2006 to minimise the impact on industry and allow time for the Commonwealth, States and Territories to amend their regulations. The 2nd Edition of the MSDS Code is available on the ASCC web site at:

[http://www.ascc.gov.au/ohslegalobligations/nationalstandards/COP\\_MSDS.htm](http://www.ascc.gov.au/ohslegalobligations/nationalstandards/COP_MSDS.htm)

A number of MSDS for triclosan and triclosan-containing products were provided for assessment. MSDSs provided for assessment fall into four main categories:

- 1) Triclosan raw chemical
- 2) Triclosan containing industrial products
- 3) Triclosan containing consumer products; and
- 4) Triclosan containing articles.

The content and format of MSDSs for raw chemical were assessed according to NOHSC *National Code of Practice for the Preparation of Material Safety Data Sheets 2nd Edition* (NOHSC, 2003). This assessment focused on the adequacy of the information provided in relation to the 'core' elements; product identification, health hazard information; precautions for use and safe handling information. The quality/adequacy of information presented in MSDS for triclosan is summarised in Appendix J.

Numerous types of products containing triclosan at varying concentrations are available. Due to the large number of product MSDS and the inability to identify a product reflective of standard use containing a 'typical' concentration of triclosan, no assessment was undertaken on MSDSs for triclosan containing products.

MSDS for products containing other hazardous substances in addition to triclosan should address the hazards of all ingredients/residues, taking into account combined/additive effects of chemicals where relevant.

### **Assessment of MSDS for triclosan raw material**

A total of seven MSDS from five suppliers were provided for assessment. Appendix J provides a summary of this assessment against 'core' elements as described above. In general all but one MSDS attempted to cover the majority of

core elements but there was inconsistency in the information provided between the MSDSs. One supplier produced two MSDSs for the raw material under different product names. In these MSDSs differences between the two documents were found in the following sections: materials to avoid, ingredients, health effects, first aid, precautions for use, personal protection, storage and transport, spills and disposal, fire/explosion hazard. In most cases the differences were that one MSDS provided more detailed information than the other, however in the first aid section, one document stated that vomiting should not be induced whilst the other document provided no statement on vomiting.

Another importer had simply copied the MSDS produced by their overseas supplier and that MSDS was totally inadequate (see MSDS number 6 at Appendix J). In terms of the core elements, only product identification and formulation were adequately addressed in this MSDS.

With regard to the hazardous nature triclosan was classified in Australia in the ASCC HSIS prior to July 2008 as:

R23 – Toxic by inhalation.

The source for this listing was an assessment by the Australian Pesticides and Veterinary Medicine Authority (APVMA).

All MSDS addressed health effects, however, they were inadequate in that none indicated the then Australian classification as in the HSIS prior to July 2008. Instead, the EU classification ‘Irritating to eyes and skin (R36/38)’ was provided. ASCC updated the HSIS in July 2008 to adopt the EU classification of R36/38 for triclosan.

The following is a discussion of the key findings of this assessment.

### ***Product identification***

This was adequately covered in all but two MSDSs, one of which provided only the product name.

Triclosan is not specifically listed in the ADG Code. Due to its moderate inhalation toxicity (HSIS classification prior to July 2008 and NICNAS classification in this report), triclosan powder (100%) falls under Class 6.1 (Toxic substances), packaging class III (Substances presenting low danger) and UN number 2811 (toxic, solid, organic) (see recommendation 2a).

The other forms of triclosan imported to Australia (liquids and pellets) should have the appropriate UN number (solid or liquid) depending on the concentration of triclosan. Class 6.1 applies only if the estimated LC50 value (1 hour) falls within the ADG Code (2007) classification range for inhalation toxicity ( $\leq 4.0$  mg/L).

Triclosan is highly toxic (acute and chronic) to some aquatic species. If the LC50 value (1 hour) of triclosan liquids or solids falls outside the Class 6.1 classification range for inhalation toxicity ( $> 4$  mg/L), Class 9 (Miscellaneous dangerous substances and articles) and UN number 3077 (environmentally hazardous substance, liquid, not otherwise specified) or 3082 (environmentally hazardous substance, solid, not otherwise specified) is applicable to triclosan (ADG Code, 2007).

### ***Health hazard information***

This information was poorly covered in that the ASCC classification (prior to July 2008) (R23) was not given in any MSDS. One MSDS gave no risk phrases whilst the remaining MSDSs listed the EU classification, with one MSDS including the additional risk phrase R37: 'Irritating to respiratory system'.

### ***Precautions for use***

Overall the information on personal protective equipment was considered satisfactory but again one MSDS gave no information under this element.

### ***Safe handling information***

Adequate information was provided on storage and transport. Handling of spills and disposal and fire/explosion hazards was adequately covered in five of the seven MSDS.

## **10.1.3 Education and training**

Guidelines for the induction and training of workers exposed to hazardous substances are provided in the National Commission's *National Model Regulations for the Control of Workplace Hazardous Substances* (NOHSC 1994c) (the Model Regulations). Under these regulations, employers are obliged to provide training and education to workers handling hazardous substances.

The Model Regulations stipulate that training and induction should be appropriate for the workers concerned. It is important that each workplace implement a program that is suitably designed to accommodate the needs of different workers.

It is important that training be given to the workers at induction and repeated at regular intervals to reinforce the information. Review of training and education needs for workers on a regular basis is useful.

Information obtained for assessment indicates that very few importers and/or formulators of triclosan or triclosan containing products have written instructions or formal training for workers, as only six companies have a training program in place. Furthermore, while ongoing training reinforces what was taught at initial induction it appears from the data submitted that training was not ongoing.

Information provided stated:

- Staff are trained generally in good manufacturing practice under the supervision of the Quality Assurance Manager;
- Staff using triclosan are instructed to wear protective clothing and follow good hygiene practices;
- Storemen, samplers and operators are trained in chemical and manual handling and records of training kept;
- Workers receive training in accessing information from MSDSs and on safe handling of chemicals;
- Workers are trained in safe handling of hazardous substances; and

- Written standard operating procedures mandate training that is supervised by the Training Team Leader but no further details are provided.

## **10.2 Occupational monitoring and regulatory controls**

### **10.2.1 Atmospheric monitoring**

Under the NOHSC Model Regulations (NOHSC, 1994c), employers are required to carry out an assessment of the workplace for all hazardous substances, the methodology of which is provided in the NOHSC *Guidance Note for the Assessment of Health Risks Arising from the Use of Hazardous Substances in the Workplace* (NOHSC, 1994d). When assessment indicates that the risk of exposure via inhalation is significant, atmospheric monitoring should be conducted to measure levels of the hazardous substances in the workplace as a precursor to the implementation of suitable control measures to reduce exposure. Subsequent monitoring is also required to ensure that such measures are effective. No atmospheric monitoring programs for triclosan in the workplace have been identified. Triclosan was classified as Toxic by inhalation (i.e. R23) in the ASCC HSIS prior to July 2008.

### **10.2.2 Occupational exposure standards**

Triclosan is not listed in the NOHSC *Exposure Standards for Atmospheric Contaminants in the Occupational Environment* (NOHSC, 1995) nor do any overseas exposure standards exist.

### **10.2.3 Health surveillance**

In accordance with NOHSC Model Regulations (NOHSC, 1994c), employers have a responsibility to provide health surveillance in those workplaces where the workplace assessment indicates that exposure to a hazardous substance may lead to an identifiable substance-related disease or adverse health effect. Triclosan is not listed in Schedule 3 (list of substances requiring health surveillance) and as such there are no formal requirements for health surveillance programs for exposed workers.

### **10.2.4 National transportation regulations**

The Australian Dangerous Goods Code (ADG Code) (FORS, 1998) sets out various requirements relating to the transport of dangerous goods by road or rail. Triclosan is not specifically listed in the ADG Code. According to the MSDSs from three suppliers, the solid form of the chemical fits into the ADG Code category of “environmentally hazardous substance, solid, n.o.s.” and has the UN Number 3077 associated with it.

However, due to its inhalation toxicity (HSIS classification prior to July 2008 and NICNAS classification in this report), triclosan powder (1-h LC50 of 2.6 mg/L estimated from 4-h LC50 of 0.65 mg/L) falls under ADG Code Class 6.1 and UN number 2811 – toxic, solid, organic.

The other forms of triclosan imported to Australia (liquids and pellets) should have the appropriate UN number (solid or liquid) depending on the concentration of triclosan. Class 6.1 applies only if the estimated LC50 value (1 hour) falls within the ADG Code (2007) classification range for inhalation toxicity ( $\leq 4.0$  mg/L).

Triclosan is highly toxic (acute and chronic) to some aquatic species. If the LC50 value (1 hour) of triclosan liquids or solids falls outside the Class 6.1 classification range for inhalation toxicity (> 4 mg/L), Class 9 (Miscellaneous dangerous substances and articles) and UN number 3077 (environmentally hazardous substance, liquid, not otherwise specified) or 3082 (environmentally hazardous substance, solid, not otherwise specified) is applicable to triclosan (ADG Code, 2007).

Considering that liquid triclosan formulations imported to Australia contains only <20% triclosan, those would fall under UN number 3082 (Class 9) based on the acute and chronic toxicity to aquatic life.

### 10.3 Public health regulations

#### **Australian Drinking Water Guidelines and SUSDP**

Triclosan is not listed in the *Australian Drinking Water Guidelines* (NHMRC, 2004) or in the *Standard for the Uniform Scheduling of Drugs and Poisons* (SUSDP) (NDPSC, 2003). However, given the acute toxicity profile of triclosan and the potential for consumer exposure to products containing triclosan, some public health regulatory controls may be warranted.

#### **Cosmetics**

The majority of cosmetic products used in Australia contain 0.3% or less of triclosan. However, some products, such as shower/bath gels, body washes, face washes and face masks, can contain up to 0.5% triclosan. There is no Australian standard limiting the amount of triclosan allowed in cosmetic products. In contrast the EU, Canada and Japan have all set maximum allowable concentrations for triclosan in cosmetic products (see sub-section 2.1).

#### **Labels for consumer products**

Forty-five companies submitted data on consumer products containing triclosan, which fell into three broad categories and amounted to 383 products. The categories were:

1. Cosmetics, including toothpastes, deodorants/antiperspirants, perfumes, body washes and moisturisers;
2. Disinfectants and surface cleaners, including bathroom cleaners and anti-mould products; and
3. Articles, including cling wrap and cotton buds

The number of cosmetic products by far exceeded the numbers of other products.

Labels for consumer products were provided by 19 companies and covered 114 cosmetics and 4 surface cleaners. Of these 118 labels provided, 77 listed triclosan as an ingredient. As triclosan is not listed in the SUSDP there are no specific labelling requirements for consumer goods that contain the chemical (as opposed to industrial products containing triclosan which must be labelled according to the classification in the HSIS).

# 11. Current Environmental Risk Management

## 11.1 Environmental regulatory controls

This section provides information with reference to international initiatives on the environmental regulatory controls in Australia applicable to triclosan.

In summary, the management of environmental pollution and waste in Australia is regulated through individual State and Territory regulatory systems rather than at a national level and each State and Territory has legislative frameworks and strategies for managing emissions and environmental pollution to air, land and waters.

## 11.2 Control of major hazard facilities

According to the *National Standard for the Control of Major Hazard Facilities* (NOHSC, 2002), triclosan is not one of the specifically identified chemicals that must be considered when determining whether a site is a major hazard facility.

## 11.3 Aquatic ecosystems management

The Australian water quality guidelines (ANZECC/ARMCANZ, 2000), established under the National Water Quality Management Strategy, provide water and sediment quality guidelines (trigger levels) for freshwater and marine ecosystems throughout Australia. The guidelines provide a decision-tree framework for the assessment and management of risks from chemicals to water and sediment quality.

Although no Australian trigger values are available for triclosan or its methylated or chlorinated products, aquatic toxicity data are available for triclosan and have been utilised in this assessment to develop water quality benchmark level (predicted no effect concentrations, PNECs). Each State and Territory has legislative frameworks and strategies for managing water and sediment pollution.

## 11.4 Disposal and waste treatment

Each Australian State and Territory provides statutory controls on waste generation and management. Triclosan-containing materials classified as wastes should be sent to licensed waste disposal contractors in accordance with State and Territory requirements. No specific waste disposal guidelines, standards or management issues were identified for triclosan wastes. Due to the ecotoxicity of triclosan product, care should be exercised in disposing of contaminated wastes to avoid pollution of the environment.

In some States/Territories, waste disposal licences are required to be held by waste contractors managing triclosan wastes. In NSW, transporters conveying triclosan waste in quantities greater than 200 kg per load or waste facilities treating triclosan wastes require a licence under the *Protection of the Environment Operations Act 1997* issued by the NSW Environment Protection Authority.

Although no specific waste disposal guidelines, standards or management issues were identified for triclosan, 11 companies provided detailed disposal information. Most companies indicated that there was no routine disposal of waste chemical, as batch sizes are determined so as to use full containers of triclosan. If disposal was needed the following were given as the methods used:

- Cardboard packaging is recycled and inner empty triclosan plastic bag disposed of at approved landfill. If product waste occurred it is disposed of as condemned stock, that is collected by a waste collection agency and taken to an EPA licensed waste facility;
- Mixing vessels and filling machines are routinely washed. The washings are processed in a waste treatment plant, the treated water pumped to the sewer and sludge disposed of by a licensed waste operator;
- Waste is released to sewers (estimated total annual release 20 kg);
- Spills swept up and then flushed to an onsite liquid waste treatment plant and;
- Workers follow written standard operating procedures. Spills are treated as hazardous waste. The chemical is swept and placed in a plastic bag that is sealed and then encased in a fibre container. A label identifying the hazardous waste is affixed to the outside of the fibre container and an outside contractor is called to collect and dispose of the waste by high temperature incineration. The waste is tracked by giving the contractor a hazardous waste handover certificate and after destruction the contractor must supply a Certificate of Destruction that is filed on site. The empty cardboard drums in which triclosan was shipped are recycled and the plastic liner is disposed of at approved landfill.

## **11.5 Emergency procedures**

### **Handling and storage incidents**

Recommendations for dealing with spills involving solids and aqueous solutions are provided in MSDSs and are similar for both forms and state:

- Shovel into approved disposal container;
- Vacuum contaminated area;
- Avoid creating dusty conditions;
- Spills to be promptly removed;
- Prevent material from entering sewers, waterways or low areas; and
- Report large spills to local environmental authorities.

However, one importer of triclosan raw material provided specific procedures for spills on site:

- Isolate spill or leak area immediately;
- Warn other personnel;
- Wear appropriate PPE before touching damaged containers;

- If safe to do so, for liquids, contain with suitable absorbent material and prevent spilled material from spreading to drain, watercourse or soil. Use sand or earth to make a dam to contain spill;
- If safe to do so, for powders, dampen down first, scoop up, then absorb/vacuum up all remaining residue using small quantities of water and detergent if necessary;
- Place collected spilled material into a sealable container and label “Waste Environmentally Hazardous Solid, UN No3077”;
- Do not wash down residue to drains. Dampen down area and repeat clean-up and containerization of spilled material until all residue is collected; and
- Dispose as chemical waste according to State/Territory waste disposal regulations.

Recommendations for fighting fires involving solids and aqueous solutions are provided in MSDSs and are the same for both forms and state:

- Use self contained breathing apparatus; and
- Extinguish using carbon dioxide, dry chemical, foam, or water.

### **Road transport incidents**

Procedures provided by an importer of the raw chemical for spills during road transport include the following:

- Stop vehicle engine and turn off electrical equipment;
- No smoking, no naked lights, or sources of ignition in immediate area;
- Warn other traffic;
- Send message to fire brigade and police. Tell location, material, UN No., quantity, condition of vehicle and emergency contact;
- Inform emergency services (e.g. Fire Brigade, Police, Environment Protection Agency) of incident location, substance, UN Number, quantity, container type, condition of vehicle and emergency contact;
- Stop leak if safe to do so, wear chemical resistant gloves, boots and protective clothing;
- Prevent spilled material from spreading to drain, watercourse or soil. Use sand or earth to make a dam to contain spill. Absorb liquid to suitable absorbent material (e.g. dry sand or earth). Collect spilled substance in a sealable container and label waste with the UN number;
- Do not wash down residues to drains. Dampen down area and repeat clean-up and containerization of spilled material until all residues are collected; and
- Dispose of waste according to instructions from EPA

Procedures provided by an importer of the raw chemical for fires during transport are:

- Carry out actions described for full emergencies;
- If a minor fire extinguish using dry powder or foam extinguisher; and
- Major fire handled by emergency services - wear chemical resistant gloves, boots and protective clothing. Use dry powder, foam or water fog. Use sand or earth to make a dam. Prevent contaminated fire fighting water from spreading to drain, watercourse or soil.

### **Maritime incidents**

For managing spills and leaks of triclosan during maritime transportation, the procedures followed will depend on the type, extent and location of the spill incident and whether the spill is contained within the ship or released to the marine environment. Recommendations include the following:

- Initiate ship chemical emergency procedures;
- Identify the source of the spill or leak and isolate the area immediately;
- Stop the leak if safe to do so, (e.g. reposition the container). Wear chemical-resistant gloves, boots and protective clothing. Clean-up spill as per above recommendations (*Handling and storage incidents*);
- Prevent spilled material from spreading. Use sand or earth to make a dam to contain spill. Absorb liquid to suitable absorbent material (e.g. dry sand or earth). Collect spilled substance in a sealable container and label waste with the UN number. Do not wash down residues;
- If product has been released off-ship, identify the location of the spill or leak (e.g. GPS co-ordinates), quantity, container type, weather conditions and current. Warn other shipping traffic in the area;
- Inform emergency services (e.g. Dockyard chemical safety officer, Fire Brigade, Police, Australian Maritime Safety Authority, Environment Protection Agency) of incident location, substance, UN Number, quantity, container type, condition of ship and emergency contact; and
- Dispose of any containerised waste in accordance with state/territory waste disposal regulations by incineration.

## 12. Discussion and Conclusions

### 12.1 Importation and use

Triclosan is not manufactured in Australia. It is imported into Australia in various forms, such as the raw chemical (>99% powder), a liquid solution (10% to <20%), plastic pellets and as an ingredient in various products (see below). The total amount of triclosan imported annually into Australia has decreased each year from 30 tonnes in 2001 to 21 tonnes in 2005.

The main occupational use of triclosan in Australia is in the formulation of personal care and cosmetic products, therapeutic products and cleaning agents. Other major uses of triclosan are in the treatment of textiles and plastics manufacture. A minor use is in the formulation of some oil-based paints for interior use on tiles and laminates. Triclosan is included in many consumer products because of its antimicrobial activity. Consumer uses of triclosan in Australia include cosmetic and personal care products, therapeutic products, veterinary products, pesticides, household and cleaning products.

It is possible that several polychlorodibenzo-p-dioxins and polychlorodibenzofurans may be found as low-level trace by-products in triclosan. The trace levels are dependent on the starting materials and reaction conditions. This led to the United States Food and Drug Administration (via the United States Pharmacopoeia) and Health Canada to set concentrations limits for these impurities in triclosan.

The European Union, Canada and Japan have set maximum allowable concentration limits for triclosan in cosmetic products.

A summary of the health and environmental hazards, and the potential risk to workers and the public are discussed in the following sections.

### 12.2 Human health hazards and risks

#### 12.2.1 Human health hazards

In humans, triclosan is rapidly and completely absorbed from the gastrointestinal tract while a lower rate is seen for dermal absorption. It is also rapidly removed from the blood, and extensive first pass metabolism occurs following oral administration. The major metabolic pathways in humans and animals involve glucuronide and sulphate conjugation, and metabolism to these conjugates has also been observed in the skin. In humans, excretion is relatively rapid. Though a significant difference was observed in the rate of elimination between some Negroid (black) volunteers compared to Caucasians (white), there are no data available to explain this difference. The major route of excretion being the urine, while the faeces is of secondary importance. The human oral and dermal data provide no evidence of a bioaccumulation potential. Additionally, enterohepatic circulation has been demonstrated in rats, while limited evidence is available in mice and hamsters.

Triclosan has low acute oral and dermal toxicity in animals. The limited inhalation toxicity data in rats indicate moderate toxicity. Both animal and human data indicate it is a skin irritant, and a study in rabbits indicates it is an eye irritant. The repeat dose inhalation toxicity study in rats showed irritation effects to the respiratory tract. Data from both humans and animals indicate that triclosan has at most a very weak skin sensitisation potential. No data on respiratory sensitization are available.

Systemic toxicity was observed following repeated exposure to triclosan in oral and dermal animal studies. No reliable human data are available. Animal data indicates that the liver is the target organ following ingestion of triclosan, with hepatocyte hypertrophy and hepatocyte vacuolization in cells observed. While the mouse is the most sensitive species there is evidence that (unlike the rat and hamster) it is sensitive to peroxisome proliferator type effects in the liver that are not considered a risk to human health. Similar effects on the liver were seen in dermal studies.

A number of in vitro and in vivo genotoxicity studies are available, and, although some positive results were obtained, overall, there is no evidence of an in vivo genotoxic potential. Oral carcinogenicity studies in the rat and hamster provide no evidence of a carcinogenic potential. No effects on fertility were seen in a 2-generation study in the rat, and there was no evidence of teratogenicity in developmental toxicity studies conducted in rats and rabbits.

Triclosan is listed in the Office of the Australian Safety & Compensation Council's (ASCC) *List of Designated Hazardous Substances*, contained in the Hazardous Substances Information System (HSIS). Prior to July 2008, triclosan was classified as a hazardous substance in the HSIS with the risk phrase, 'Toxic by inhalation (R23)'. ASCC updated the HSIS in July 2008 to adopt the Europe's 29<sup>th</sup> Adaptation to Technical Progress (ATP) to Directive 67/548/EEC (April, 2004). With this update in July 2008, triclosan is now on the HSIS with the risk phrase, 'Irritating to eyes and skin (R36/38)'. Based on the current assessment and according to the *Approved Criteria for Classifying Hazardous Substances* (NOHSC, 2004), triclosan is classified as 'Toxic by inhalation (R23)' and 'Irritating to eyes, respiratory system and skin (R36/37/38)'.

Triclosan is not listed in the *Standard for Uniform Scheduling of Drugs and Poisons* (SUSDP). However, the acute toxicity profile of triclosan suggests that it could be considered for listing in the SUSDP.

### 12.2.2 Occupational health and safety risks

Workers may be potentially exposed to triclosan by skin and eye contact and inhalation. The likelihood of exposure by ingestion in occupational settings is expected to be low. Similarly, the low vapour pressure of triclosan means that the main route of exposure is likely to be via the dermal route. However, there is potential for inhalation exposure when using triclosan powder.

Exposure during importation and storage is unlikely except in the case of accidental breakage of containers and spillage of triclosan powder or liquid. The potential for exposure following accidental spillage of plastic pellets containing triclosan is low, as the triclosan itself is encapsulated in the plastic matrix. The major occupational exposure scenarios are formulation of personal care, cosmetic and cleaning products, treatment of textiles and plastics manufacture. In these

scenarios, enclosed automated processes are reported to be used. Exposure is expected to be low as generally the process is periodic, engineering controls such as local exhaust ventilation are reported to be in place at most work sites and personal protective equipment (PPE), such as safety goggles and gloves, are reported to be worn at some sites. Consequently, the risk of acute effects such as inhalation toxicity, skin, eye and respiratory irritation is low, though the risk would increase for accidental spills or leaks of triclosan and/or products containing high concentrations of triclosan, especially where personal protective equipment is not used in the clean up of spills. Exposure would not be significant during use of commercial cleaning products containing triclosan, as the maximum concentration of triclosan identified in an occupational end-use product was 0.3%.

However, no occupational monitoring data for triclosan are available in Australia or reported in the literature. Therefore, the Estimation and Assessment of Substance Exposure (EASE) model was used to predict inhalation and dermal exposure. For chronic effects, a Margin of Exposure (MOE) approach was undertaken for risk characterisation using a NOAEL of 40 mg/kg bw/day identified in a 2-year study for effects on the liver in the rat. The lowest MOE ranges determined were 32–320 for formulation/plastic manufacture with 100% triclosan. However, it is considered that the MOEs for these scenarios will be at the higher end of the predicted range (i.e. > 100 for formulation/plastic manufacture) and the risk of chronic effects to workers from repeated exposure to triclosan is low. The determination of a low risk is based on the nature and severity of effects seen in repeated dose studies in animals. The histopathological changes observed in hepatic cells were minor and seen only in male rats. In addition, these changes were not seen consistently throughout the carcinogenicity study and, there are no data to suggest that humans are more sensitive than animals. Additionally, EASE does not take into account the use of PPE and it is considered that the use of closed or partially enclosed automated work processes and other engineering controls and PPE mean that the actual exposures are likely to be lower than that predicted by the EASE model.

MSDS and labels for imported raw triclosan were assessed qualitatively against the NOHSC MSDS and Labelling Codes. In general, labels were lacking information on the concentration (i.e. purity) of triclosan. No label gave the risk phrase for inhalation toxicity as provided in the HSIS, but all except one label gave the risk phrases for eye and skin irritation. All labels provided adequate safety precautions/instructions. There was inconsistency in the information provided between the MSDS, generally relating to the level of detailed information provided. However, overall, information on product identification, precautions for use, and safe handling were adequate. The risk phrases for eye and skin irritation were provided in all but one MSDS, though one included the additional risk phrase R37: 'irritating to respiratory system'. A sample MSDS for triclosan is included in Appendix I.

Due to the large number of product MSDS and the inability to identify a product reflective of standard use containing a 'typical' concentration of triclosan, no assessment was undertaken on MSDS for triclosan containing products.

### **12.2.3 Public health risks**

Public exposure can occur through the use of consumer products containing triclosan. The major exposure scenarios are from the use of consumer products

containing triclosan such as cosmetic and personal care products, household cleaning products and from textile articles containing triclosan. Given the types of triclosan containing products available to the public the main route of exposure is likely to be dermal, though oral exposure may occur through accidental or incidental ingestion of lip balm, toothpaste or mouthwash formulations, and inhalation exposure may occur through breathing aerosols generated from the use of cosmetic, personal care or cleaning products. Additionally, oral exposure may potentially occur in young children and babies through the sucking or mouthing of textile/plastic articles. The detection of triclosan and/or its metabolites in human breast milk samples indicates a further potential source of exposure in breast-feeding babies.

For acute health effects such as inhalation toxicity, skin, eye and respiratory irritation the risk is considered to be low due to the low concentration of triclosan in consumer products. Additionally, accidental ocular exposure is expected to occur only infrequently. Textile and plastic articles do not present a risk for irritation.

Measured exposure data are limited for the consumer exposure scenarios. Some data are available for repeated use of cosmetic and personal care products. Consequently, various exposure models have been used to predict consumer exposure to various categories of products. The absence of data on the leaching of triclosan from articles prevents the potential dermal and oral exposure to be determined from such. As for occupational risk characterisation, an MOE approach was undertaken for chronic effects. A worst-case exposure scenario was determined with the exposure models and MOEs were determined using the maximum level of triclosan detected for each type of product in Australia and with exposure to all possible types of products for that exposure scenario.

All MOE ranges derived from exposure models indicated that the risk of chronic effects from repeated exposure to consumer products containing triclosan is low, as in addition to being worst-case scenarios and thus likely to be overestimates, the nature and severity of the effects seen in animals are minor and there are no data to suggest that humans are more sensitive than animals. The lowest MOE ranges in both adults and young children/babies using modelled data, and hence greatest potential risk of an adverse effect, was for exposure to cosmetic and personal care products: 179-213 in adults; 471 in babies less than 1 year old; 402 in 2 year old children; and 603 in 5 year old children. In adults, similar MOE ranges were seen in some volunteer studies (i.e. measured data) using a single cosmetic or personal care product containing triclosan: 179–311<sup>7</sup>. This measured data raise a concern that cannot be completely dismissed, that is, the risk of chronic effects may potentially increase to levels that cause concern in some individuals through combined use of many cosmetic and personal care products containing triclosan, and/or use of such products containing relatively high concentrations of triclosan.

This assessment indicates that the lowest potential source of exposure to babies, and hence the lowest risk of an adverse effect, is from triclosan in breast milk.

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<sup>7</sup> For the measured data, the majority of MOEs following use of a single cosmetic or personal care product were greater than 1000.

## 12.3 Environmental hazards and risks

### 12.3.1 Environmental hazards

Limited ecotoxicity data were available for several trophic levels (animals and plants) from aquatic and terrestrial environments.

In the aquatic environment, triclosan is very toxic to freshwater aquatic organisms such as *Daphnia* and fish. From the limited data available, freshwater algae are the most sensitive species (NOEC = 0.2-0.69 µg/L and 72-96 h EC<sub>50</sub> = 0.53-1.44 µg/L). Recent research has indicated that effects on hormonally-induced metamorphosis of tadpoles can occur at concentrations around the predicted no effect concentration (PNEC). However, the biological significance of these effects is currently unclear, particularly as higher concentrations showed no effect.

In both acute and chronic tests with freshwater invertebrates, EC50 values increase as pH increases, and triclosan is much more toxic to freshwater animals in neutral or acidic waters than in alkaline waters. There was a paucity of data for marine organisms, which precludes conclusions on toxicity for this compartment.

Both triclosan and its methylated derivative methyl-triclosan have a high potential to bioaccumulate in aquatic organisms (Log Pow values of 4.8 and 5.2, respectively, indicating partitioning to lipids). Bioaccumulation potential is also evident from laboratory-scale bioconcentration factor (BCF) studies and field monitoring studies. However, no data were available that correlate tissue concentrations with effect levels.

In the terrestrial environment, data for two standard test species indicates that triclosan is slightly toxic to birds by the oral route of exposure, with a LD<sub>50</sub> of 862 mg/kg bw in bobwhite quail. In soils, triclosan is toxic to plants when grown in sandy soil (time-weighted average (TWA) NOEC for cucumber 65 µg/kg); however, toxicity was less (TWA NOEC for cucumber 446 µg/kg) when grown in sandy loam. The attenuation of phytotoxicity is potentially due to the higher organic matter content of the sandy loam soil binding to triclosan.

Triclosan is also slightly toxic to earthworms. No other terrestrial invertebrate toxicity data were available, and no data were available on the effects of triclosan on soil microbial process (e.g. respiration, nitrification).

The limited data available indicate that effect levels of triclosan on activated sewage sludge micro-organisms can vary depending on the level of acclimation, but can significantly reduce their ability to remove ammonia as well as their nitrification capacity for several days at least.

For assessing potential risks to the environment, the annual import volume and use pattern of triclosan was used to estimate potential levels of triclosan entering Australian STPs. The estimated amount is between 14.5-17.4 µg/L. The estimated concentrations are consistent with those observed overseas (<0.10-562 µg/L). These influent levels were used to derive predicted environment concentrations for Australian freshwater and marine environments based on varying levels of wastewater treatment. For freshwater, the predicted levels range between ≤ 0.1-15.2 µg/L, which are consistent with the observed levels overseas of 0.01-269 µg/L. However, limited measured Australian data for the levels of triclosan in some sewage effluent and biosolids indicate that measured levels are at the lower end of internationally observed values. Notably, the measured data do not include

the larger STPs or any NSW STPs. Consequently, it is difficult to extrapolate these data to all freshwater ecosystems in Australia.

### **12.3.2 Environmental risks from release to the aquatic environment**

There is potential for indirect effects on birds and mammals to occur near STP outlets as a consequence of the effects of triclosan on their food supply, and also direct toxicity arising primarily through food consumption based on the predicted levels of triclosan in surface waters. Based on exposure and toxicity data, the risks to birds and most Australian mammals (with the possible exception of the platypus) are considered to be acceptable. Modelled data indicate potentially unacceptable risks to mammals such as the platypus that subsist exclusively on water-based organisms such as fish (from bioaccumulation in food). To refine the risk estimate, platypus specific data was compared to the maximum measured Australian surface water concentration taken from five rivers in Queensland, and indicated that there is an acceptable level of risk to platypuses. However, these surface water measurements were only conducted in Queensland and do not cover the full range of urban STPs in Australia, particularly the larger STPs.

Modelled data indicate a potentially unacceptable risk to freshwater organisms for each type of wastewater treatment. Available data for concentrations of triclosan in surface waters near STPs in five rivers in Queensland indicate that concentrations are likely to be lower than those predicted through modelling, but confirms that triclosan is still present at levels which could potentially result in adverse effects on algae. These surface water measurements are unlikely to be representative of broader environmental concentrations, which could potentially be much higher around other, larger STPs. Because no data were submitted for sediment-dwelling organisms, it is not possible to determine potential effects in this particular compartment. As dilution is high in ocean outfalls, risks to marine species are considered acceptable.

### **12.3.3 Environmental risks from release to the terrestrial environment**

Both modelled and measured data indicate that triclosan is present in biosolids at levels which, when applied to soil, may result in adverse effects on plants. While some evidence is also available which points to its persistence in treated soils, a standard laboratory study indicates that triclosan degrades rapidly in aerobic soils. Although modelled data indicate potential risks to soil dwelling organisms from irrigation by effluent water, limited measured data indicate that risks to plants from irrigation are acceptable.

Overall, in the absence of additional data indicating concentrations downstream of representative STPs in Australia, it is not possible currently to exclude the possibility of unacceptable risks to certain species such as algae. Algae form an important food source for numerous other organisms. Potentially unacceptable risks to soil dwelling organisms from the use of biosolids (as soil conditioners) or effluent (for irrigation) from STPs also currently cannot be excluded.

## **12.4 Data gaps**

For the purposes of human health and environmental risk assessment, this report identified a number of data gaps.

The environmental assessment identified the following monitoring data gaps:

- lack of comprehensive Australian data on the concentration of triclosan in representative STP effluents and surface waters;
- lack of Australian data on the concentration of methylated or chlorinated derivatives of triclosan in representative STP effluents and surface waters;
- lack of measured data on the concentration of triclosan and its methylated or chlorinated derivatives in aquatic sediments;
- lack of comprehensive data on the concentration of triclosan and its methylated or chlorinated derivatives in STP sludge, or in biosolids, used as soil conditioners in Australia; and
- lack of field monitoring and microbial toxicity data on the ecological effects of triclosan.

The environmental assessment identified the following data gaps for toxicity:

- lack of toxicity data for triclosan for native Australian wildlife, and no toxicity data for the methylated products of triclosan;
- limited data on the toxicity of triclosan and no data for methyl-triclosan to sediment dwelling organisms;
- lack of data on the effects of methyl-triclosan on soil micro-organisms and nutrient recycling; and
- limited data regarding effects of triclosan on marine organisms and no data for methyl-triclosan.

The human health assessment identified the following monitoring and toxicity data gaps:

- absence of representative atmospheric monitoring in formulation plants;
- absence of dermal exposure data;
- absence of data on the leaching of triclosan from textile and plastic articles;
- lack of data on the health effects of triclosan in humans following repeated exposure; and
- use of a default oral NOAEL for determination of MOE estimates as no reliable evidence of systemic toxicity was seen in dermal studies in a suitable animal model.

The assumptions used in EASE modelling also add uncertainties to the human risk characterisation.

Furthermore, while it is concluded that there is no risk to humans or the environment with regard to antimicrobial resistance to triclosan, it is recognized that there is limited information on:

- The prevalence of triclosan resistant organisms in clinical environments;
- The exact mechanisms of antibacterial action of triclosan;
- The kinetics of triclosan antibacterial resistance mechanisms and their possible transferability; and

- The fate of triclosan in the environment, the rate and extent of degradation of triclosan and the anti-microbial activity of degradates or low concentrations in the environment.

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