Toxikos
Toxicology Consultants

ABN 30 095 051 791
PO Box 74
Caulfield East
Victoria, 3145
Ph:+61 3 9569 3088
Fax: +61 3 9569 3618
rogerdrew@bigpond.com

Human Health Risk Assessment - Bell Bay Pulp Mill Effluent

Prepared for: Gunns Ltd.

Prepared by: Roger Drew and John Frangos,
Principal Consultants,
Toxikos Pty Ltd.

Toxikos Document: TR081205-RJF
July 2006

Roger Drew, Ph.D., D.A.B.T.
(Diplomate American Board of Toxicology)

John Frangos, M.Appl.Sc (Toxicology)

Disclaimer

This report was prepared by Toxikos Pty Ltd for Gunns Limited (Gunns, the 'Client'). The material in it reflects Toxikos' best judgement in the light of the information available to it at the time of preparation. However, as Toxikos cannot control the conditions under which this report may be used, Toxikos will not be responsible for damages of any nature resulting from use of or reliance upon this report. Toxikos' responsibility for the information herein is subject to the terms of engagement with Gunns.

About Toxikos Pty Ltd

Toxikos Pty Ltd is a consulting company formed on December 1st 2000 to provide clients with independent excellence in toxicology and health based risk assessment. Its charter is to assist industry and government make science based decisions regarding potential effects and management of environmental and occupational chemicals. For over twelve years, prior to and since the establishment of Toxikos, staff have provided toxicology and health risk assessment advice to clients in a wide range of industries and government in Australia, New Zealand and South Africa.

About the author: Dr Roger Drew is one of the Directors and Principal consultants of Toxikos Pty Ltd. He has primary degrees in biochemistry and pharmacology and postgraduate degrees in toxicology. Postdoctoral training was undertaken at the National Institutes of Health, National Cancer Institute in the USA and he spent twelve years teaching medical students and conducting toxicological research at Flinders University of South Australia. He was corporate Toxicologist to ICI/Orica Pty Ltd for twelve years before creating Toxikos Pty Ltd. Dr Drew is the only consultant toxicologist in Australia certified by the American Board of Toxicology.

Dr Drew has been a toxicology consultant to Australian Federal and State Authorities; a member of several standing expert committees of the National Health & Medical Research Council of Australia and the National Occupational Health and Safety Commission of Australia. He has been a member of many expert task groups of the World Health Organization for the International Programme on Chemical Safety.

Dr Drew was Adjunct Professor of Biochemical Toxicology at RMIT University and teaches various aspects of toxicology to undergraduate and postgraduate students at RMIT, Monash and Melbourne Universities. He is a member of several professional toxicology societies and is a recognised national and international expert in toxicology and risk assessment. He is currently on the editorial board of the international scientific journal "Regulatory Toxicology and Pharmacology" and a board member of the Australian National Research Centre for Environmental Toxicology.

Executive Summary

This report examines the potential for human health risks posed by effluent discharged into Bass Strait from an elemental chlorine free (ECF) kraft paper mill proposed for Bell Bay. Historically, kraft pulp mills have used elemental chlorine as the primary bleaching mechanism for wood pulp. This, combined with sub-optimal effluent treatment, lead to effluent from these mills being associated with polluting waterways.. Consequently fishing from some fresh and marine waters in the northern hemisphere down stream from the mills was banned. In addition, fish from some effluent receiving waters were tainted. However since the introduction, and refinement, of elemental chlorine free (ECF) bleaching, and enhanced biological treatment of effluent most of the fishing bans[1] have been lifted (Hagen et al. 1997, AET 2002) and fish tainting associated with pulp mills is largely a phenomenon of the past (Section 8 and Appendix 7).

The facility at Bell Bay is being designed as a current 'state of art' elemental chlorine free pulp mill. It is therefore inappropriate to use effluent information from mills that employed old, irrelevant technology to make predictions regarding the human health impact of effluent discharge to the ocean from the proposed Bell Bay mill.

It is however problematical to conduct a prospective health risk assessment for effluent that does not yet exist. Consequently the identity and concentration of substances in the mill effluent has been conservatively determined by the mill designers who used mass balance calculations to apportion potential components of effluent between various mill processes and waste streams. In general the mill designers consider they have over estimated the likely concentrations of substances in the effluent. This information was supplemented by Toxikos who created a list of substances that have historically been identified in pulp effluents even though they unlikely to be in the effluent from the Bell Bay mill. This was undertaken to minimise the chance of missing an important substance for consideration in the health risk assessment.

Together these approaches generated a 'candidate list of chemicals' that have in the past been associated with pulp mill effluents. With knowledge of the bleaching chemistry and effluent treatment to be used at the new mill this candidate list of chemicals was examined for substances likely to be in the Bell Bay effluent in an appreciable quantity. Such deliberations

[1] Since 1990 US authorities have removed 70% of the fish advisories placed on water bodies down stream of pulp mills (AET 2002).

lead to a large list of 'chemicals of interest' which were potential subject matter for the health risk assessment.

To assess the potential for human exposure to the effluent it was not considered realistic that recreational swimming or diving would occur near the ocean outfall because it is 3 km offshore in inhospitable seas and the ocean bed at that point (approximately 25m deep) is barren. The only plausible way the general public could be exposed to substances in the effluent is through consumption of biota that may have accumulated substances from the effluent. The candidate list of chemicals was therefore evaluated for bioaccumulation potential in fish. Shellfish were not considered because the proposed outfall area is physically incompatible for them to prosper and none were observed during baseline biota surveys (Aquenal 2005). Consumption of fish from around the effluent outfall is the only realistic and plausible means for the general public to be indirectly exposed to substances that may be in the discharged effluent.

Thus for humans to be exposed to chemicals in the effluent they must eat fish that have accumulated substances from the effluent. Such bioaccumulation is dependent upon four parameters, all of which must be satisfied for bioaccumulation to occur.

1. The substance must be a relatively small molecule.
2. It must be taken up by fish from water or sediment.
3. Organic substances must have high affinity for the fat of fish.
4. Compounds entering the fish must be poorly metabolised and/or not well excreted; if a chemical is readily excreted or easily/rapidly metabolised it cannot easily accumulate in the animal unless exposures are sufficient to overwhelm these removal mechanisms.

The candidate list of chemicals was therefore assessed for molecular size, bioconcentration potential, and fat solubility. Three substances [2] were found likely to bioaccumulate. However all are readily metabolised or excreted by fish and therefore are not 'chemicals of potential concern'.

In addition, the candidate list of chemicals was screened to determine if a reputable regulatory agency had identified a substance as being possibly bioaccumulative. If a chemical was considered as bioaccumulative by a regulatory agency, then it was automatically considered in the risk assessment. It should be noted however that although an agency had identified a chemical as potentially bioaccumulative in some situation somewhere in the world it may not be

[2] The substances are retene, chlororetene and fichetelite. Retene and fichetelite are possible biodegradation products of the abietic acid found naturally in wood.

so for the circumstances, or species potentially in contact with the Bell Bay mill effluent. Substances regarded as 'chemicals of potential concern' were dioxins/furans and the metals cadmium, selenium, mercury. These were identified from bioaccumulation notations in the ANZECC water quality guidelines but very little information is provided for the basis of the notation. Toxikos has therefore undertaken an evaluation of the literature for the circumstances which has lead then to be considered as potentially bioaccumulative. The assessment of these chemicals is described below.

Metals

It is well known that metals occur naturally in marine waters from non-anthropogenic sources. The biogeochemical processes which control metal accessibility to marine organisms and the mechanisms by which they are taken up and subsequently stored in the organisms are very complex. Unfortunately, addressing this complexity in a quantitative risk assessment is very data intensive and much of the required information is not available. However logic dictates that if within a very short distance of effluent discharge the theoretical incremental increase in water concentration of the metals is very small relative to the prevailing background seawater concentration then it is unlikely there will be significant increases in organism body burden over and above that which already occurs. Without a large incremental increase in body burden in organisms that may realistically be consumed by humans there is no material increase health risk to humans. Thus the first risk assessment consideration for metals is an evaluation of the circumstances which may realise the bioaccumulation potential of the chemicals of interest. Part of such an evaluation is a simple comparison of the predicted water concentrations at the perimeter of the initial dilution zone, the DZ100 [3],with background seawater concentrations of the metals. Knowledge of the biological processes controlling the accumulation of the metals and whether there is a large increase in seawater concentration will potentially drive the need for a quantitative risk assessment.

Selenium:

Selenium is an essential element required by fish and other animals for maintenance of normal biochemical functions. Its' uptake is controlled by homeostatic mechanisms and accumulation occurs when these are overwhelmed. This can happen when water concentrations are greater

[3] For the purposes of this risk assessment the discharge area of interest is that associated with a 1 in 100 dilution, called the DZ100. It is anticipated the DZ100 is a small area within 100m either side the diffuser at the end of the pipeline.

than 3 – 5 µg/L. Because the estimated selenium concentration at the margin of the DZ100 is within the background range measured around the world, and at least an order of magnitude less than the lowest water concentration associated with accumulation in fish, and less than the ANZECC water quality guidelines for protection of aquaculture, an incremental increase in selenium concentrations in fish around the outfall is unlikely.

It is concluded selenium discharged in the mill effluent will have negligible impact on humans eating fish caught at the outfall.

Cadmium:

Cadmium can be bioconcentrated in marine organisms if they are sedentary, such as mussel, and there is local cadmium pollution. While some tissues of fish are able to assimilate cadmium to relatively high levels, muscle has limited capacity. Accumulation to occur in fish muscle requires relatively high environmental concentrations; in addition this tissue is very slow to take uo cadmium and long exposure times are needed for fish muscle to accumulate appreciable levels. Furthermore when muscle does accumulate cadmium it quickly looses the metal when the fish transfers to cleaner water. The inability of muscle to take up cadmium and retain it is a reflection of relatively low amounts of metallothionein in the muscle. The implications for the human risk assessment are that fish which are temporarily within the initial dilution zone may not there long enough to accumulate cadmium into muscle.

A mass dilution of effluent cadmium gives a water concentration at the DZ100 of 0.012µg/L. While this level is not expected to result in marked incremental accumulation of cadmium because it is less than background concentrations around the world and the latter are not associated with cadmium accumulation by fish, it does not take into account the background seawater concentrations at the site which are recorded as being less than analytical detection limits of 0.2 µg/L (15 of 16 samples). In addition levels of cadmium in fish at the location have not been quantitated but are also less than analytical detection limits of 1 mg/kg fish (n = 20). It is noted however that existing levels of cadmium in fish are not recognised as potential cause for health concerns for humans.

Overall it is considered unlikely that humans will be affected by cadmium in the effluent but there is large uncertainty, due to lack of quantitative background data, associated with this opinion.

Mercury:

It is expected the effluent treatment processes of the Bell Bay mill will remove most if not all (detectable) mercury before discharge and, as with the other water concentration estimates, the concentration of mercury in the DZ100 is believed to be an over estimate. Mercury (Hg) is converted to methylmercury (MeHg) by miroflora at the sediment-water interface, and it is this form that is most readily bioaccumulated by marine organisms. The estimated Hg concentration at the fringe of the DZ100 is within the background range measured around the world and Australia, and tentatively supports the notion any incremental increase in MeHg concentrations in biota around the outfall will be minimal. There is however uncertainty associated with the estimation of local background seawater concentration used for this calculation. Consequently other assessment techniques have also been used to evaluate the potential impact of Hg in the discharged effluent on human health. Firstly, calculation of theoretical incremental increases in fish MeHg concentrations due to the effluent demonstrated total fish Hg levels would increase only slightly and would remain at a level approximately half of that specified in the Australia New Zealand Food Standards Code (FSANZ 2004) for Hg in fish. Secondly, conservative calculations of human intake of MeHg (background + incremental from effluent Hg) from fish showed that for all population sectors, high-end fish consumers who sourced all their fish from the effluent outfall would have MeHg intakes less than the relevant intake guideline for protection of health.

All these lines of assessment have lead to the conclusion that mercury discharged in effluent will have negligible human health impact.

Dioxins

Dioxin formation is very limited in the Bell Bay mill processes and as a result concentrations in the discharged effluent are anticipated to be non-detectable. However there is a residual small possibility of minute amounts of dioxins being present in the effluent. The maximum concentration of dioxins in the discharged effluent is estimated to be 0.074 pg TEQ/L which is significantly below analytical quantitation limits (approximately10 pg TEQ/L) and below RPDC[4] guideline limits (13 pg TEQ/L). In parts of the world where significant pollution of waterways has occurred, virtually all the dioxins partition into sediment. In locations where there has been high,

[4] RPDC is the Resource Planning and Development Commission (Tasmania).

point source contamination of sediment some types of fish can accumulate dioxin,however dioxins do not biomagnify between between sediment and fish or fish and their food.

The following conclusions regarding the bioaccumulation of dioxins by marine organisms have been formulated by review of the literature available for this project:

- Dioxins are not significantly bioaccumulated or biomagnified by fish.
- Fish living in local environments where dioxin concentrations are low also have low levels of dioxins.
- The theoretical increase in dioxin levels of fish after the effluent outfall becomes operational is markedly less than the analytical capability to measure the change.
- There will be no demonstrable increase in dioxin concentrations of fish that might reside around the ocean outfall.
- Animals higher in the food chain, including fish and seals, have relatively high metabolic and/or excretory capacity towards dioxins.

From the above information it could be argued that a detailed quantitative health risk assessment is not warranted for the dioxins discharged from the Bell Bay mill. However in order to adequately address stakeholder concerns regarding these substances a quantitative dioxin health risk assessment has been undertaken.

The general principle for assessing health impacts of dioxins in discharged mill effluent is to estimate the incremental dioxin monthly intake that results from eating fish caught in the area and add it to an estimate of monthly background intake of all dioxin like substances. Risk is characterised by comparing this sum with the monthly intake determined to be safe by the Australian Government Department of Health and Ageing (NHMRC 2002) (i.e. the tolerable monthly intake, the TMI). If the total monthly intake of dioxins is less than the TMI (70 pg TEQ/kg body weight/mth) then the risk of adverse health effects from dioxins in the Bell Bay mill effluent is very low.

Key assumptions in the risk assessment are:

- There is a constant dioxin water concentration at the DZ100.
- Dioxins discharged in effluent partition into sediment at the outfall.
- There is equilibrium between dioxin levels in the outfall environment and levels in fish.
- Fish spend the majority of their time at the outfall.
- All the fish eaten by humans comes from the outfall.
- People who eat the fish do so at the maximum rate identified for Tasmanians.
- There is maximum background intake of dioxins by people who eat fish from the outfall.

All the above assumptions individually over estimate the amount of effluent derived dioxin taken up by fish and consumed by humans. The calculated incremental increase in fish dioxin concentration is 0.18 pg TEQ/kg fish. This is much less than the analytical quantitation limits of 100 – 3,400 pg/kg for fish. The maximum incremental increase in dioxin intake by people (adults and children) eating the fish is 0.004 pg TEQ/kg bw/mth. This is an increase of 0.025% for person with high-end background intake and fish consumption.

Dioxins are formed by most combustion processes (e.g. cars, bush fires, burning waste) and are ubiquitous in our environment. For humans, food derived from animals (meat, dairy products, eggs, seafood etc) accounts for 95-99% of total background intake. Of this approximately 40% is from seafood (OCS 2004). The upper bound estimate[5] for background intake of all dioxin like compounds for Australians is 15.79 pg TEQ/kg bw/mth for adults and 37.74 pg TEQ/kg bw/mth for children. These background intakes do not materially change when the incremental increase of 0.004 pg TEQ/kg bw/mth from maximum consumption of fish assumed to be caught at the outfall is added.

Measurements of background dioxin levels in fish caught at the proposed ocean outfall site were less than quantitation limits. It should be noted the very low level of dioxin predicted to be in the discharged effluent cannot be measured. Similarly, the very small theoretical incremental increase in dioxin concentrations in fish cannot be measured. All these factors render it very difficult to corroborate the risk assessment experimentally, or with field observations once the

[5] The upper bound estimate for background intake of dioxins by Australians was sourced from the Department of Environment and Heritage's 'National Dioxins Program' risk assessment (OCS 2004). The upper bound estimate is the 95th percentile intake, but calculated using the analytical limit of detection for dioxin congers that were not detected in consumed food stuffs.

mill is in operation. It is highly unlikely a statistical difference can be shown between fish dioxin concentrations before and after the mill becomes operational. Since there will be no demonstrable change in dioxin levels of fish there is not quantifiable increase in dioxin intake by humans consuming the fish, and incremental health risks are negligible.

Because the risk assessment cannot be confirmed with field tests a sensitivity analysis was conducted by changing values of various parameters used for calculating the incremental ingestion of dioxins from fish. Increasing the assumed amount of dioxin in discharged effluent to that of the analytical quantitation limit[6] plus doubling the maximum ingestion rate of fish, or increasing the available dioxin in the sea bed 7 fold[7] had virtually no impact on the total amount[8] of dioxin ingested. In the sensitivity analysis dioxin intake increased from 15.79 pg TEQ/kg bw/month to 16.9 pg TEQ/kg bw/month

The anticipated dioxin concentrations in discharged mill effluent do not pose a health risk to people consuming fish caught in the vicinity of the outfall. The sensitivity analysis provides a high degree of confidence in this conclusion.

Tainting of seafood

The available information indicates commercial and recreational fishing does not occur in the vicinity of the proposed ocean outfall. In addition, there is a paucity of fish in the area and rapid dilution of the effluent is anticipated to occur. Food tainting is considered to have occurred if there is any change in food flavour, or if there is an unusual odour to the food. In the past pulp mill effluents discharged into fresh water systems have been associated with tainting of fish. However historical accounts of fish tainting should not be used to judge the potential of Bell Bay mill effluent to cause tainting.

Although it cannot be stated with certainty, it appears that poly-chlorination of natural phenols and resins in wood may have been primarily responsible for historic tainting of fish by pulp mill

[6] Increasing the amount of dioxin in the discharged effluent to the analytical detection limit (approximately 10 pg TEQ/L) increases the dioxin concentration in the receiving water 137 times.
[7] The amount of available dioxin in the seabed was increased by lowering the amount of organic carbon in the sediment in the modelling thereby allowing more 'free' unbound dioxin to be theoretically transferred to the lipid of fish.
[8] The 'total' amount of dioxins ingested is the incremental intake from fish plus the upper bound estimate of the 95th percentile of background intake for Australians for a person who consumes double the maximum amount of fish eaten by Australians and assuming all the fish is from the outfall.

effluent. Overall, it is considered that modern elemental chlorine free bleaching and modern effluent treatment technology intended to be installed at the Bell Bay pulp mill will virtually eliminate these substances.

On the whole it is considered there is little potential for tainting of fish in the outfall area and low potential for the public to be exposed to tainted fish. However there is sufficient uncertainty to support the RPDC requirement of taint testing of effluent, at least in the first few years of mill operation.

Recreational activities

The World Health Organisation and Australian authorities have published water quality criteria for protecting public health during recreational water use. These guidelines are intended for coastal and inland waters where the user comes into frequent direct contact with the water. Due to the harshness of the physical setting and scarceness of marine organisms recreational water use in the vicinity of the proposed outfall is unlikely, and has not been observed during marine survey work. Nonetheless, the estimated water concentrations of effluent constituents at the edge of the initial dilution zone do not exceed recreational water guidelines where they exist for a particular substance.

This page left intentionally blank

Contents

Acronyms and Abbreviations

ABS	Australian Bureau of Statistics
ADI	Acceptable Daily Intake
AET	Alliance for Environmental Technology
ANZECC	Australian and New Zealand Environment Conservation Council
ATSDR	Agency for Toxic Disease Registry (United States)
BAF	Bioaccumulation Factor
BCC	Bioaccumulative Chemicals of Concern
BCF	Bioconcentration Factor
BEKP	Bleached Eucalypt Kraft Pulp
BKME	Bleached Kraft Mill Effluent
BKP	Bleached Kraft Pulp
BMF	Biomagnification Factor
BSAF	Biota-to-Sediment Accumulation Factor . The ratio of the lipid-normalised concentration of a contaminant in tissue of an aquatic organism to its organic carbon-normalised concentration in surface sediment.
BW	Body Weight
C_{BS}	Bed sediment bulk density
C_F	COPC Concentration in fish
C_{Fish}	Concentration of contaminant in fish
$C_{Fish\ n}$	Contaminant concentration in top predator fish
C_l	Total concentration of a chemical in whole organism/tissue divided by the lipid fraction
C_s	Total concentration of a chemical in sediment
C_{sb}	Concentration of contaminant sorbed to bottom (bed) sediment
C_{soc}	Total concentration of a chemical in sediment divided by the fraction of organic carbon in sediment.
$C_{w\ tot}$	Total water concentration
ClO_2	Chlorine Dioxide
CoA	Co-enzyme A
CoPC	Chemical of Potential Concern
CR	Consumption Rate
D_l	Daily intake of contaminant
d_{bs}	Depth of upper benthic sediment layer
d_{wc}	Depth of water column
DEH	Department of Environment Heritage, Australian Government
EC	Environment Canada

ECF	Elemental Chlorine Free
EPA	Environmental Protection Agency (or Authority)
f_l	Lipid fraction
f_{oc}	Fraction of organic carbon in sediment
f_{bs}	Fraction of total water body contaminant concentration sorbed to bed sediment
FCM	Trophic level-specific food chain multiplier
[fish]	Contaminant concentration in fish tissue
GL	Giga Litre
HHRA	Human Health Risk Assessment
HOCl	Hypochlorous Acid
HQ	Hazard Quotient
I_{Fish}	Daily human intake of fish
IIS	Integrated Impact Statement
JP	Jaakko Pöyry Oy. The Finnish company designing the pupl mill.
K_{ow}	Log oil:water partition coefficient
Kd_{bs}	Bed sediment/sediment pore water partition coefficient
MeHg	Methylmercury
NAS	National Academy of Sciences
NEPC	National Environment Protection Council
OCS	Office of Chemical Safety
OC_{sed}	Fraction of organic carbon in bottom sediment
OSPAR	Oslo and Paris Conventions on the pollution of the North Sea, also known as the Convention for the Protection of the Marine Environment of the North East Atlantic
PBDD	Polybrominated dibenzodioxin
PBDF	Polybrominated dibenzofuran
PCB	Polychlorinated biphenyl
PCDD	Poly-Chlorinated Dibenzo-ρ-Dioxin
PCDD/F	Poly-Chlorinated Dibenzo-ρ-Dioxin/ Polychlorinated Dibenzofuran
PCDF	Polychlorinated Dibenzofuran
ppq	Parts per quadrillion
ppt	Parts per trillion
PTWI	Provisional Tolerable Weekly Intake
RPDC	Resource Planning and Development Commission (Tasmania)
TCDD	2,3,7,8 – Tetrachloro dibenzo-p-dioxin
TDI	Tolerable Daily Intake

TEQ	Toxicity equivalents. The relative toxicity of various dioxin congeners to that of 2,3,7,8-Tetrachlorodibenzo-*p*-dioxin.
TRS	Total Reduced Sulphur
θ_{bs}	Bed sediment porosity
US EPA	United States Environmental Protection Agency
WHO	World Health Organization
DZ100	Zone where mass dilution of 1 in 100 occurs

A comment on units:

Concentrations of dioxins are expressed as picogrammes (pg) (generally expressed as TEQ) per gramme (g) or kilogram (kg) of the medium (fish or sediment), or per litre (L) if water, or per cubic metre (m^3) if a gaseous medium.

1 pg is equal to 1/1,000,000,000,000 (1×10^{-12}) of a gramme (or one million millionth of a gramme).

Because a kilogram is 1,000 grams, 1 pg is one thousand million millionth of a kilogram (or 1/1,000,000,000,000,000 or 1×10^{-15} kg).

1 pg/L is 1×10^{-12} g/L.

This page left intentionally blank.

1. Introduction

1.1 General background

Gunns Limited (Gunns) intends to build and operate a paper pulp mill at Longreach, Bells Bay. The proposed mill will use elemental chlorine free (ECF) bleaching technology in the kraft process and will be 'state of art'. The proposed mill is being designed to achieve best practice environmental guidelines set by RPDC.

Biologically treated effluent (herein referred to as the 'final effluent') is intended to be transported from the mill via a land and sub-ocean pipeline ending at a point in Bass Strait where good dispersion characteristics of discharged effluent can be achieved. The end of the effluent pipeline will be located in the centre of a large area of coarse sand that currently has very limited marine organisms (Aquenal 2005). It will be at a depth of approximately 25m and about 3 km offshore from Five Mile Bluff (Figure 1.1), and will be fitted with a diffuser.

The diffuser will be purpose built to achieve an initial minimum mass effluent dilution of 100 fold within a short distance of about 100m of the release point (JP 2005a), this is called the 1:100 dilution zone and referenced in this report as DZ100 (see Section 3.2 for more information). Conceptually, the diffuser at the end of the pipeline will be approximately 200m long and will have a number of strategically placed holes to evenly release the effluent along its length. The effect is to release a curtain of effluent perpendicular to the prevailing cross current thereby achieving quick dilution. Detailed dispersion characteristics of the effluent are being examined by GHD (GHD 2005).

Gunns has contracted Toxikos Pty Ltd to perform a predictive human health risk assessment (HHRA) for the effluent that will be released into the ocean. In this HHRA the ocean effluent discharge is evaluated for:

- Impact on human health via potential accumulation of effluent constituents by biota which is consumed by humans.
- Possible tainting of seafood.
- Impact on human health due to recreation water use in the vicinity of the outfall.

To facilitate the assessment the pulp mill engineering designers, Jaakko Pöyry Oy, provided Toxikos with information on the type of constituents in the discharged mill effluent and their concentrations. Gunns provided additional information from their consultants as it came to hand. The proprietary information relied upon by Toxikos for conducting the health risk

assessment is listed at the beginning of the reference section of this report (Section 12). Toxikos has taken this information at face value.

It should be noted this assessment does not evaluate potential effects of the effluent on marine ecology. This is the subject of a separate report being prepared by GHD on behalf of Gunns.

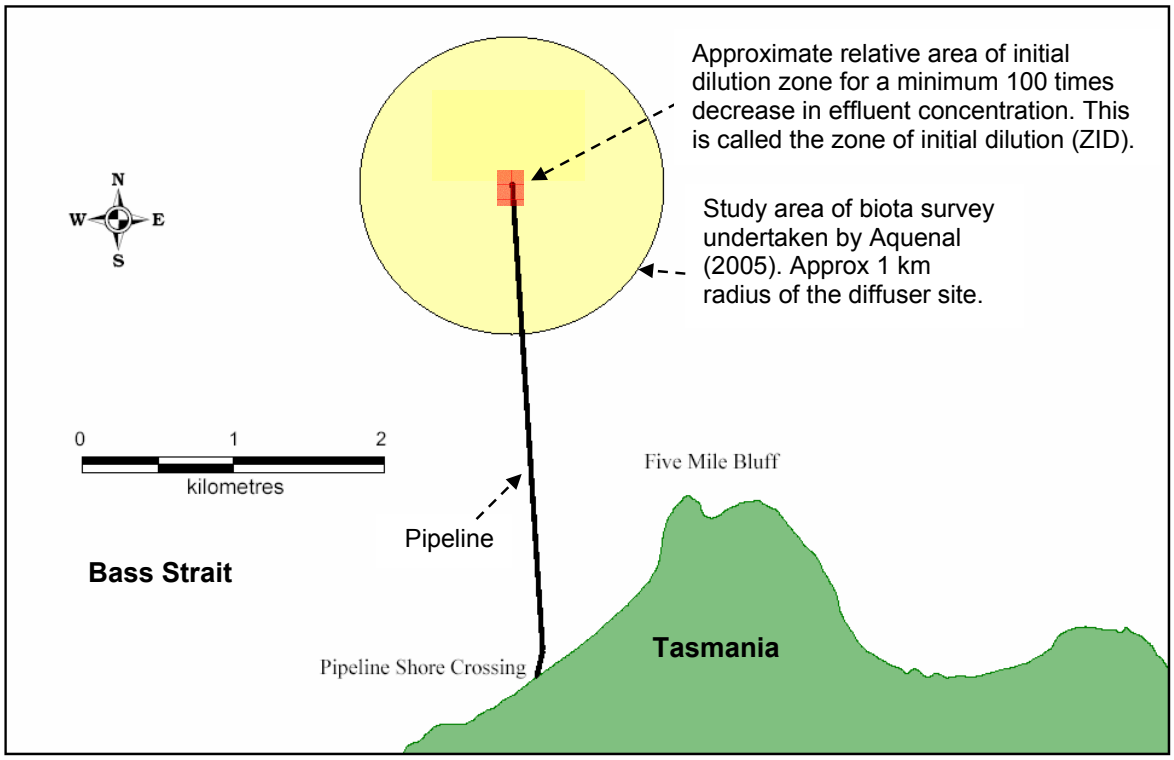


Figure 1.1: Schematic representation of the effluent pipeline and the area subject to the biota survey of Aquenal (2005) relative to the zone of initial dilution.
(Adapted from Aquenal 2005).

1.2 What is a health risk assessment?

A human health risk assessment (HHRA) is an analysis that uses information about environmental hazards, such as chemical substances, to calculate a theoretical level of risk for people who might be exposed to defined intensities of those hazards in the future. For the situation being evaluated in this report the hazards are natural and anthropogenic chemicals in

the waste effluent of the proposed mill. The natural chemicals are derived from the constituents of wood while the anthropogenic ones arise from products added during the pulping process. The risk assessment is used to inform regulatory officials, facility owners/managers and the public and to help determine strategies to ensure overall protection of human health should the proposed development proceed. Developments usually only occur when regulatory authorities are satisfied that appropriate controls have been put in place to ensure the future safety of the public.

It is important to note that a prospective risk assessment such as the one herein does not measure actual health effects that substances may have on human health because the development project has not yet taken place and as a result there are no data for exposure estimates. Such risk assessments are usually conducted by considering possible or theoretical community exposures from predicted concentrations of effluent concentrations. These are based on knowledge of previous effluent discharges from existing facilities, taking into consideration differences in the processes and engineering designs between the present proposal and existing mills. Adopting a precautionary approach, conservative safety margins are built into the risk assessment analysis to ensure protection of the public, and to account for lack of certainty in the actual concentrations of substances in the effluent from the proposed mill. Consequently any actual exposure, should it occur, is anticipated to be much lower than that used in the risk assessment analysis.

2. Structure of the report

Because this health risk assessment is for a facility that does not currently exist and exact details of effluent composition and concentration are unknown the risk assessment does not follow strictly the traditional structure of ‘hazard identification’, ‘exposure assessment’ and ‘risk characterisation’. Wherever possible, to make the report more readable and user friendly, detailed technical discussions supporting a particular aspect of the assessment or presentation of scientific information have been confined to Appendices. In some instances the main body of the report contains only a précis of the relevant appendix. Because of stakeholder interest in the possibility of dioxins being in the effluent and concern about dioxin release to the environment some detailed and contextual information is provided in the main body of the report on the bleaching chemistry and health effects of dioxins.

Although uncertainties associated with specific aspects of the health risk assessment are discussed at the time the particular topic is introduced in the report an overview uncertainty

analysis has been also been produced. In this analysis, an attempt has been made to confine the uncertainty discussion to the 'big picture' items and keep repetition to a minimum.

3. Risk assessment methodology

3.1 General

The overall methodology employed in this risk assessment is consistent with that of the Australian enHealth Council (enHealth 2002), the US Environmental Protection Agency (US EPA 1989, 2000a) and the US Agency for Toxic Substances and Disease Registry (ATSDR 1992a).

In summary, this risk assessment examines the potential for members of the general public to be exposed to chemicals that may be in the effluent. Although this risk assessment is quantitative there are aspects that are primarily of a screening nature due to the fact that it deals with risks for a hypothetical person who is maximally exposed, directly or indirectly, to the highest final effluent concentration that is reasonably expected to occur at the edge of the DZ100. Theoretically, human exposure (Section 6.1) to chemicals in the discharged effluent could occur by:

- Consumption of seafood if the chemical accumulates in the edible portions of marine animals living in the vicinity of the diffuser.
- Direct contact with seawater containing effluent, for example during recreational activities.

A significant hurdle to evaluating the health risks of effluent from a mill that is not yet constructed and in operation is the absence of detailed characterisation of effluent. This is made more acute by the realisation the Bell Bay mill will incorporate many engineering improvements not present in current best practice ECF mills. There is therefore uncertainty regarding the constituents and their concentrations in the Bell Bay mill effluent. This has been addressed by the process outlined in Figures 3.1 and 6.1, and making conservative assumptions (erring on the side of over estimation and caution) in the mass balance calculations used for determining possible realistic maximum concentrations in final effluent[9].

Although the Bell Bay mill predominately processes hardwood (eucalyptus) chips, it will nonetheless have production campaigns where pine chips are used for feedstock. Effluent

[9] Personal communication with Jaakko Pöyry (2005).

characteristics from pine and eucalyptus mills may be different, however for the purposes of this risk assessment where information on the different effluent constituent concentrations has been made available to Toxikos the exposure was assumed to be at the higher concentration.

The risk assessment does not evaluate in detail every possible component of the effluent. With hundreds of compounds potentially present (Section 4) it would be an impossible task to assess each individual chemical. Indeed common sense indicates exposure would not be expected to occur or be important for all compounds in the effluent. For example, a substance may be present at such low concentrations that the exposure is equivalent to background levels or less, or some substances may not have the necessary properties to be taken up and/or remain in fish.

Thus the risk assessment herein uses a number of screening procedures (Section 3.2) to decide which of the possible effluent components are important for detailed assessment. These are compounds that might be potential health threats to people who eat fish caught within the initial dilution zone and/or to people directly exposed to receiving waters close to the effluent outfall during recreational activities.

3.2 Zone of Initial Dilution – Assumptions and Implications

The diffuser at the end of the effluent pipe will be designed to achieve an effluent dilution of 100 fold within a short distance of release from the pipe[10]. The risk assessment addresses the human health impacts of incremental increases in seawater concentrations of effluent constituents at these assumed dilutions. Furthermore the diffuser is to be engineered so the target dilutions will be achieved within a short distance[11] of the diffuser, approximately 100m. The assessment therefore deals with incremental concentrations at the edge of the 1 in 100 zone of initial dilution, for ease of reference throughout the report this is termed the DZ100.

The 1:100 is dilution zone (DZ100) defined as the mass dilution of discharged effluent. The 1:100 dilution was selected based on the findings of eco-toxicological testing undertaken on representative effluent samples from similar operating pulp mills. The test reports concluded in reference to the 1:100 dilution mixing zone that assuming the effluent from the proposed pulp mill is the same as that sampled and tested from the representative mills, and that the sample was representative of effluent quality over time, then based on the eco-toxicology testing results, no acute or sub-acute lethal toxicity would be expected to be observed at the edge of the DZ100 mixing zone.

Within the relatively small area of the DZ100, effluent concentrations will be higher. This is not a significant issue for direct effects of the effluent in the human health risk assessment because the general population is only likely to be exposed to chemicals in effluent via secondary exposure mechanisms, such as consumption of fish that may have accumulated some effluent constituents. For those substances that have the necessary properties for bioaccumulation the extent of accumulation is determined by the average concentration to which the organism is exposed which in turn is governed by the concentrations prevailing over the fish's entire range and the time spent in various portions of the range, and by the rate of removal of the substance from the organism. Brief exposures to the higher concentrations that may occur within the DZ100 zone have relatively little impact on the final concentration achieved in the organism,

---

[10] Personal verbal communication Gunns Pty Ltd (2005), Jaakko Pöyry (2005) and GHD (2006). The diffuser will be 200m long with two rows of 15 cm holes in the upper half (in section at 10 & 2 o'clock) of the pipe which will be lying on the seabed.

[11] Estimates of the distances from the diffuser where the 1 in 100 dilution occurs will be determined by the hydrodynamic modelling being undertaken by GHD. The risk assessment herein has been conducted independent of the size of the DZ100, however the smaller the area of DZ100 relative to the foraging area of the fish the lower the probability that a fish will spend significant amounts of time within the DZ100. based on the assumption that the 1 in 100 dilution will be achieved within 100m of either side of the pipe the DZ100 area may be (100 + 100) x 200m = 40,000m^2 = 0.04 km^2.

especially if such exposures are small compared to the half life of the substance in the organism in question[12].

The DZ100 is different to the 'mixing zone' defined by RPDC (2004) and is likely to be smaller. The 'mixing zone' is defined in the RPDC (2004) emission limit guidelines for any new bleached eucalypt kraft mill in Tasmania as "a three dimensional area of the receiving waters around a point of discharge of pollutants within which it is recognised that the water quality objectives for the receiving waters may not be achieved (Sate Policy on Water Quality Management).

3.3 Screening procedures

The overall screening methodology is summarised in Figure 3.1. The first step was to determine what may be in the effluent (Section 4). Briefly, a list of effluent constituents was compiled by reviewing the literature. Any substance found to be reported in pulp mill effluent, regardless of age or type of mill, was regarded as being possibly present in the Bell Bay effluent (Appendix 1, Tables A1.1 & A1.2). This produced a *'candidate list of chemicals'.* This list was culled based on knowledge about the bleaching chemistry of chlorine dioxide, mill engineering modifications and advice from Jaakko Pöyry to produce a list of *'chemicals of interest'* for the Bell Bay effluent (Appendix 1, Table A1.3). When there was uncertainty about whether a historical effluent constituent could be present in the Bell Bay mill effluent it was assumed to be present. Final effluent concentrations for the *'chemicals of interest'* were determined as described in Appendix 1 (Table A1.3). These substances were then subjected to screening processes for bioaccumulation potential. In addition the predicted concentration of the chemicals of interest in the water column was screened against criteria for recreational water and criteria for tainting edible seafood as shown in Figure 3.1.

Screening for bioaccumulation in seafood:

The bioaccumulation screening methodology efficiently identifies effluent constituents which can be concentrated by aquatic organisms into either in muscle or fat and thus have the potential to pose a hazard to humans who consume the organisms. The screening methodology is described in detail in Section 5 (Figure 5.1) and is based on molecular weight, known bioaccumulation in marine organisms, and readiness of metabolism/excretion. Substances coming forward from this screening process were considered to be *'chemicals of potential concern'* and subject to a detailed human health risk assessment for exposure via seafood.

[12] The time it takes to reach steady state body burden concentrations is about 3 – 5 half lives. A half life is the time required for a given body concentration to decrease to half its initial value.

Screening for recreational activities:

Because the proposed outfall is approximately 3 km from shore in an area of extreme currents, the distance from shore, prevailing weather and lack of natural features near the outfall make it unlikely, although not impossible, that water contact recreational activities (e.g. swimming or diving) will occur in the initial dilution zone around the ocean outfall (i.e. within 70 - 100 m of the diffuser). Nevertheless, and primarily for completeness, chemicals assumed to be present in the effluent have been assessed in Section 7.2 against screening criteria developed by Australia (ANZECC 2000) and the World Health Organization (WHO 2003) for recreational water use. Recreational boating may occur near the outfall but it is not anticipated direct contact with high concentrations of effluent will occur unless a person falls overboard; in which case the situation will be as for a person swimming in the water.

Screening for tainting of seafood:

When some substances are taken up by organisms, even in small amounts, they can impart off-flavours and seriously affect the palatability of seafood. Such tainting substances potentially have deleterious impacts on aquaculture and wild-capture fishing industries (both commercial and recreational) if these activities occur close to effluent outfalls that release such substances. Indeed tainting has been an historical issue with some old pulp mills in the northern hemisphere. Although not strictly a health issue, substances assumed to be present in the final effluent of the Bell Bay mill were assessed against lists of threshold concentrations for chemicals in water above which tainting may occur [13] (refer Section 8).

[13] Guidelines for chemicals that cause tainting are available from ANZECC (2000), US EPA and various provinces of Canada, e.g. British Columbia. Guidelines could not be located from other authorities (Environment Canada, WHO, UK Department of Environment, Finnish Environment Industry and Danish Environmental Protection Agency). OSPAR (2002) provided a list of chemicals that are suspected to taint and also a list of chemicals that have been tested but found not to cause tainting. Compounds listed as not tainting are dimethylbenzenes, nitrophenols, dinitrophenols, β-pinene, phenol, toluene and xylene (OSPAR 2002). It is noted however some of these substances have ANZECC (2000) tainting guidelines. Consequently, for the purposes of this assessment, it has been assumed they may cause tainting even though experimental evidence suggests otherwise.

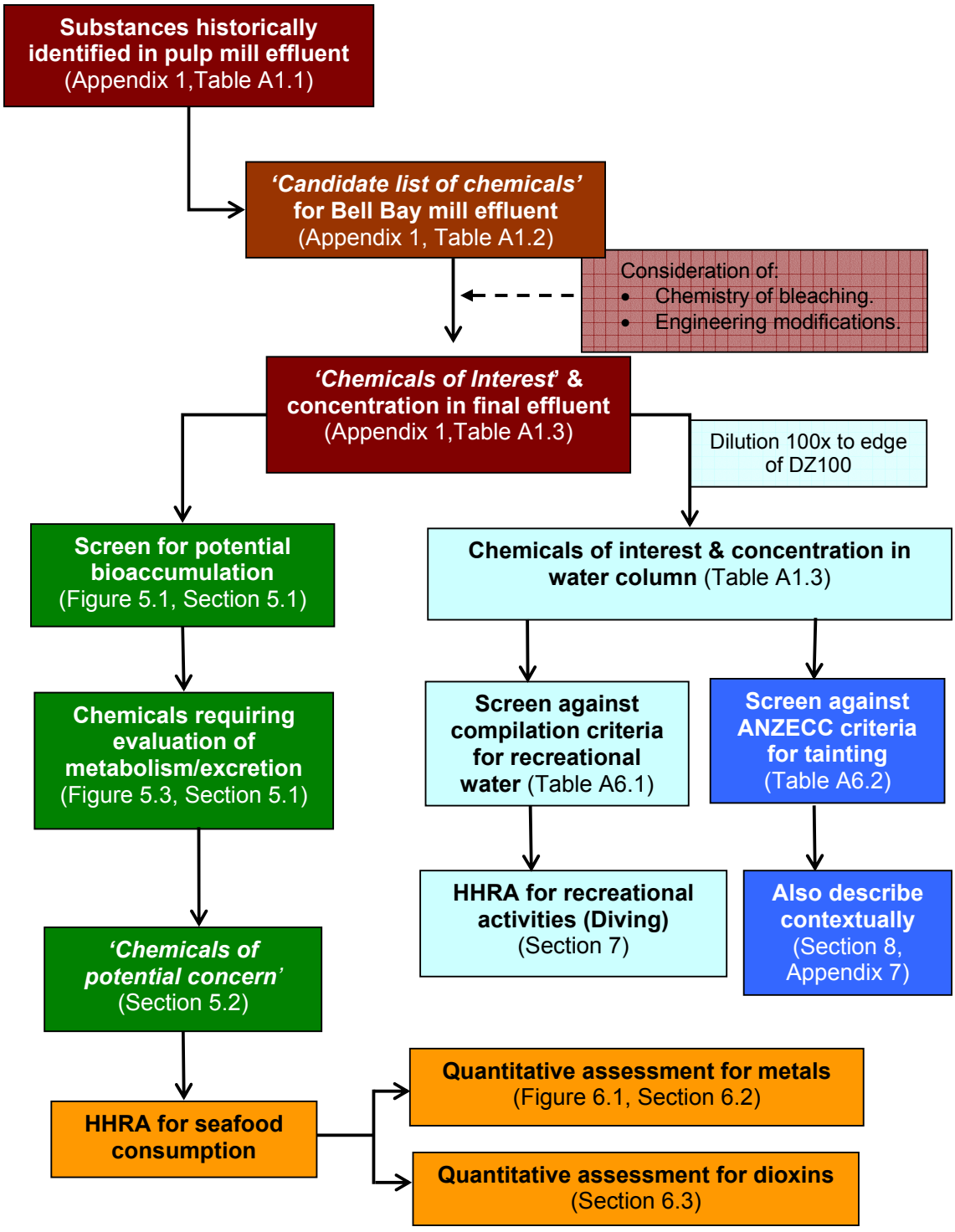


Figure 3.1: Overview of the screening processes for evaluating health impacts of effluent discharge to sea.

4. What's in the effluent?

4.1 Historical

Bleaching technology and effluent treatment expertise has changed dramatically over the last two decades. Thus, it is important to realise that effluents from different pulp mills are not necessarily comparable from one mill to the other or over time at a particular location because of engineering improvements. Figure 4.1 summarises and compares the effluent characteristics of historical and modern pulp mills.

Historically, pulping was a one stage process resulting in high residual lignin and elemental chlorine was used as the bleaching agent. These two factors resulted in large amounts of polychlorinated substances being formed and subsequently released in effluent. However modern pulp cooking methods produce low residual lignin[14], the improved (2-stage) delignification and oxygen prebleaching of modern mills further decreases the lignin content of the liquor entering the chlorine dioxide bleaching stage (Bright et al. 2003). This is important because the amount of chlorinated organic material in pulp mill effluent going to bio-treatment is determined by the lignin content of the prebleaching liquor. A markedly decreased lignin concentration prior to bleaching means much less chemical substrate is available to react with chlorine to produce polychlorinated substances (FEI 1996, Dahlman et al. 1996). In addition, historical process chemicals, particularly oil based defoamers with aromatic structures, also served as substrates for chemical reactions with elemental chlorine and were important for the formation of dioxins and furans in elemental chlorine pulp mills of the mid 1970s and 1980s (Gunthe 1998). These substances are no longer used. In addition to improvements in production processes there has been substantial enhancement effluent treatment prior to discharge that significantly reduces the chemical content of effluent.

Perhaps the most important advance in the pulp mill processing technology was the removal of elemental chlorine (Cl_2) as the primary bleaching agent and its replacement with chlorine dioxide (ClO_2). The chemical reactions of chlorine dioxide with lignin and other organic substances are completely different from that of elemental chlorine (Section 4.2). The combination of decreased chemical substrate (e.g. lignin and aromatic defoamers) together with different bleaching chemistry has dramatically reduced the formation of polychlorinated compounds and virtually eliminated dioxins and furans from modern pulp mill effluents[15] (Luthe

[14] The amount of lignin in pulp is measured as a 'kappa number'.
[15] Strömberg et al. (1996) analysed two ECF pulp mill effluents for polychlorinated dioxins and furans. They were only detected in one effluent and then at very low concentrations that the authors considered to be equivalent to background concentrations.

et al. 1992, FEI 1996, Shariff et al. 1996, Strömberg et al. 1996, EPA 1998, Bright et al. 2003). For example, for both chlorine and ECF pulp mills Stromberg et al. (1996) analysed effluent prior to it entering secondary treatment for polychlorinated compounds. In the ECF effluent tri- and tetra-chlorinated phenolics were not detected and the concentration of total chlorinated phenolic was reduced by 98%.

Table A1.1 in Appendix 1 contains a compilation of the chemicals historically found in pulp mill effluent. The list is not intended to be exhaustive however it does identify substances in effluent commonly reported in the literature. Table A1.2 of Appendix 1 is the same *'candidate list of chemicals'* but also provides the rationale, based on bleaching chemistry and engineering process modification[16], for determination of the *'chemicals of interest'* for Bell Bay mill effluent (Table A1.3).

[16] Outcomes of engineering process modifications were advised by Jaakko Pöyry.

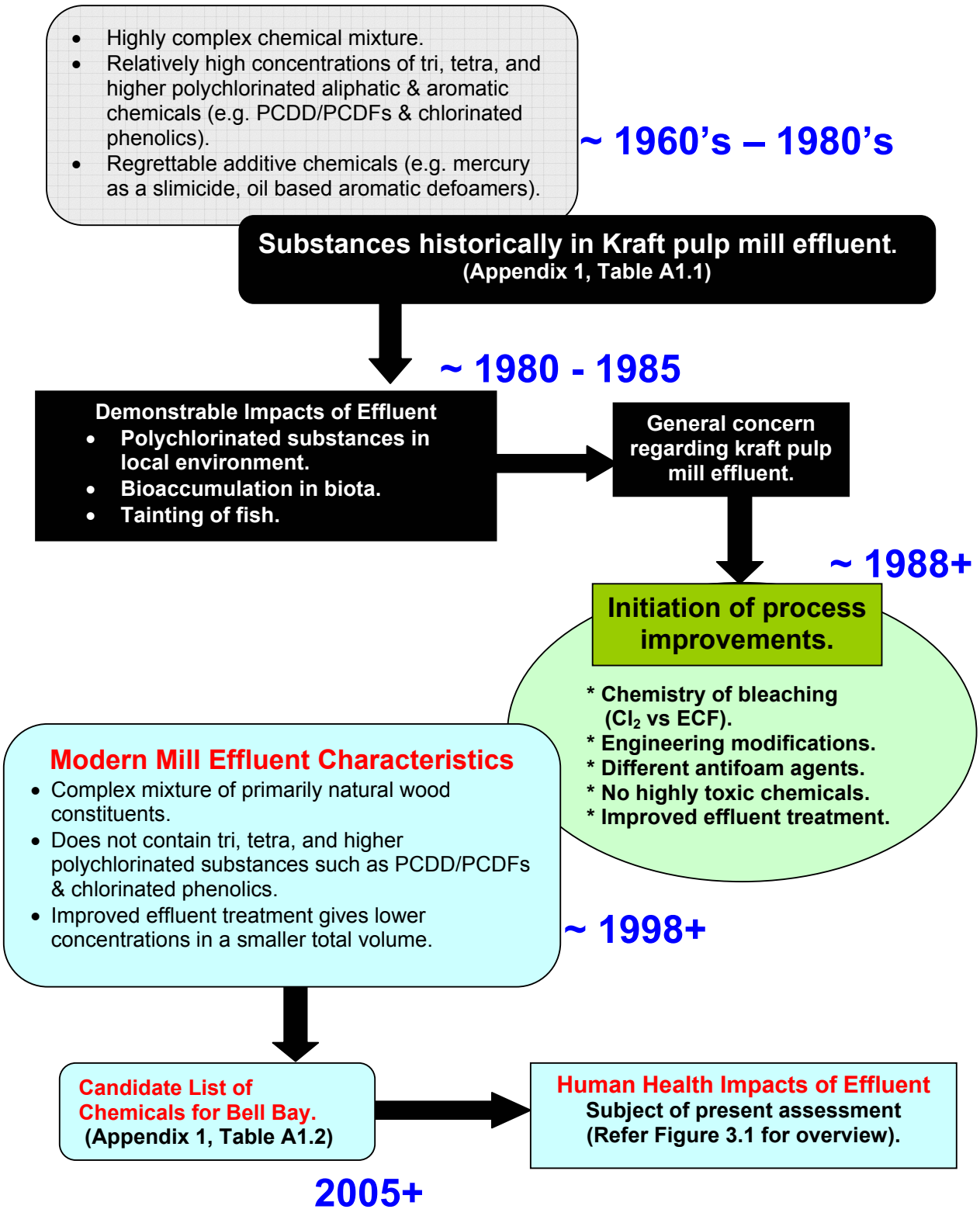


Figure 4.1: Characteristics of overseas historical pulp mill effluents versus effluent from proposed mill at Bell Bay.
Dates are indicative only

4.2 Bleaching chemistry of chorine dioxide

The publicity surrounding the environmental issues of dioxins in the effluent of pre- 1990 pulp mills has lead to a common perception that all kraft pulp mills release large amounts of dioxins into the environment. However, primarily because of the different bleaching chemistry of elemental chlorine (Cl_2) and chlorine dioxide (ClO_2) this is not the case for modern ECF mills. Since the difference in chemistry is fundamental to this fact, a brief review of the bleaching chemistry is provided in this section.

Elemental chlorine can directly react with the aromatic portion of lignin, or other aromatic chemicals, by electrophilic substitution or addition reactions which results in chlorine molecules being placed on the aromatic ring. Thus polychlorinated aromatic substances are formed.

On the other hand chlorine dioxide (ClO_2) reacts with the aromatic portion of lignin very differently than does Cl_2. Chlorine dioxide is a more powerful oxidant than Cl_2 and the aromatic rings are opened to yield carboxylic acids which are more water soluble. In this reaction hypochlorous acid (HOCl) is formed as a side product. In turn HOCl can react with double bonds (e.g. in the side chain of lignin) to further oxidise lignin; this reaction can produce chlorinated substances if substitution reactions occur. However the preferred reactions of HOCl with residual lignin are oxidative and the extent to which HOCl participates in electrophilic substitution reactions is significantly smaller than that of elemental chlorine. While the later reactions can be minimised by careful control of pH, some small amounts of mono- and di-chlorinated organic compounds can be formed.

Overall the amount of chlorinated substances produced in the bleaching process is much lower (by approximately 70%) with ClO_2 than with Cl_2 (Wallis et al. 1994). Higher polychlorinated compounds, especially dioxins, are virtually eliminated[17] by use of ClO_2; importantly only very small amounts of chlorinated aromatic compounds are produced (Luthe et al. 1992, Shariff et al.1996, Süss 2002, Bright et al. 2003). The data presented by Strömberg et al. (1996) show the reduction in the quantity of chlorinated phenolic compounds in ECF bleaching compared to Cl_2 bleaching is far greater than suggested by the overall AOX reduction[18]. The small amounts of chlorinated phenolic compounds that were detected in effluents from mills with ECF bleaching

[17] According to US EPA (1997) dioxin production is reduced by at least 96%.
[18] There is a 98% reduction in total chlorinated phenols vs 82% reduction in AOX (Strömberg et al. 1996).

had low level chlorination (mono- and di-chlorinated)[19]. Low chlorination reduces the persistence and potential of substances for bioaccumulation.

4.3 Candidate list of effluent constituents

The type and concentration of chemicals formed and released into effluent during pulp and paper production is dependent upon several factors. These include the wood species being pulped, the degree of spill control, the recovery of pulping liquor, the bleaching process, the type of chemical additives and the extent to which effluent is treated before being released. Nevertheless the composition and quantities of chemicals present in the final effluent (LaFleur 1996, EC 1991) can be broadly predicted on the basis of an understanding of the:

- sources of chemicals (wood and anthropogenic);
- mechanisms by which they are formed; and
- the efficiency of effluent treatment.

Notwithstanding the above, it is again noted that effluent constituents and quantities are affected by mill-to-mill differences in process technology and operations, differences in wood types and sources, plus chemical interactions among the different waste streams that may be specific for a given mill (Bright et al. 2003). There is therefore some uncertainty in predicting the composition of the Bell Bay effluent. The *'candidate list of chemicals'* potentially present in the final effluent was prepared by taking into account compounds previously measured/reported in pulp mill effluents regardless of type of mill or bleaching technology. It represents a conservative and cautionary list of chemicals.

Table 4.1 summarises the typical chemical classes of constituents of pulp mill effluent (based on literature review) and the percentage contribution that each stage of the process is expected to contribute to the final effluent for the Bell Bay mill. A more detailed compilation of the chemicals historically found in pulp mill effluent is in Appendix 1 (Table A1.1).

[19] The chlorinated phenolic compounds from ECF bleaching (~ 1g ptp) were exclusively mono-, and di-chlorine compounds but from elemental chlorine bleaching (~45g ptp) was 57% tri-, and tetra-chlorinated phenols (Strömberg et al. 1996). ptp = per tonne pulp.

Table 4.1: Summary of chemical classes historically found in pulp mill effluent.

Process Stage	Typical[1] Compounds		% in Final Effluent[2]
Raw material (i.e. wood components	Softwood (sw)	High molecular weight polysaccharides (alpha and hemi-cellulose) and lignins (p-hydroxyl propyl, guaiacol, syringyl propyl) [3].	96%
	Hardwood (hw)	High molecular weight polysaccharides (alpha and hemi-cellulose) and lignins (p-hydroxyl propyl, guaiacol, syringyl propyl) [3].	
	Raw material extractives[4]	Terpenes (hydrocarbons, alcohols and ketones), Sterols, Resin and fatty acids.	4%
Kraft Pulping	Aliphatic and aromatic compounds including alcohols, phenols, carboxylic acids and diacids, aldehydes, and reduced sulphur compounds (e.g. thiophenes).		
Bleaching	Chlorinated phenolics, chlorosulfones, chloroacetic acids, chloroacetones, chloro-resin/fatty acids/sterols, chloroform. Refer Section 4.2 for comments on formation of polychlorinated [5] substances (including dioxins).		

[1] Intended to be an indicative (not exhaustive) list of the types of compounds typically present in bleached Kraft pulp mills based on literature review LaFleur (1996), Bright et al. (2003), Dahlman et al. (1995), Sunito et al. (1988), Strömberg et al. (1996).

[2] Estimated composition by mass of the dissolved organic matter (DOM) in Bell Bay final effluent based on plant design characteristics (JP 2005a).

[3] The compositions of polysaccharides and lignins differ between hardwood and softwood. For instance softwood lignin consists primarily (>95%) of guaiacol propyl lignin while hardwood also contains significant amounts (approximately 20 – 40%) of syringyl propyl and guaiacol propyl lignin (LaFleur 1996).

[4] Chemicals which can be extracted with organic solvents are termed extractives.

[5] Several hundred low-molecular weight organic compounds have been identified in raw bleaching effluents (Sunito et al. 1988). These are not necessarily present in the treated effluent. The presence of polychlorinated substances cannot be entirely ruled out; however, if they are present they are expected at trace levels given that their formation requires the presence of elemental chlorine which itself is present only at trace levels.

4.4 Chemicals of interest in Bell Bay effluent

From the *'candidate list of chemicals'* (Tables A1.1 and A1.2, Appendix 1) that might be potentially present in the effluent of the Bell Bay mill the following groups of compounds were removed to derive the list of *'chemicals of interest'* in the effluent (Table A1.3, Appendix 1).

Classes of compounds removed from consideration of bioaccumulation potential were:

- Water Quality Parameters such as BOD, AOX, COD etc because they are summary water quality parameters, not individual compounds. They are not relevant or amenable for the human health risk assessment of effluent discharge to the ocean.
- Some polychlorinated compounds (i.e. tri-, tetra- etc) are considered 'virtually eliminated' on the basis of the chemistry of the bleaching process (Bright et al 2003, EC 1991)(see also Section 4.2).
- Brominated compounds because there is not a source of reactive bromine for these substances to be created. They were included on the *'candidate list'* because they were referenced by RPDC as requiring monitoring (RPDC 2004).

To enable the risk assessment herein, the mill designers (Jaakko Pöyry Oy) used its expert knowledge of the kraft process and mill design to estimate final effluent concentrations for either individual or classes of chemicals (JP 2005 c,d,e). This was done using mass balance equations where all sources of an effluent constituent were considered relative to effluent control and treatment efficiencies. This information was supplemented by Toxikos with information from the literature concerning known constituents of pulp mill effluent as described in Appendix 1. Table A1.3 in Appendix 1 summarises the assumed concentrations of effluent constituents for the Bell Bay mill effluent. These concentrations were used for assessment of recreational water use (Section 7) and tainting of fish flesh (Section 8).

It should be noted that while the estimated concentrations are not definitive, Toxikos is advised by Jaakko Pöyry that the concentration of any individual constituent in the final effluent is likely to be over-estimated rather than under-estimated.

5. Bioaccumulation screen of *'chemicals of interest'*

5.1 Screening criteria for bioaccumulation

In relation to marine organisms the term 'bioaccumulation' broadly refers to the accumulation of a chemical via direct transfer from the water column and/or sediment, plus accumulation through the diet. The ability to take up a chemical from either water or sediment, is called bioconcentration. In virtually all cases, for uptake via food to occur an organism at some position in the food chain bioconcentrates the chemical. Fat solubility of the chemical is an important, but not the sole determinant of bioconcentration. The amount of chemical accumulated in an organism via bioconcentration is the balance between uptake and removal

processes. The latter is dominated by an organism's ability to metabolise and/or excrete the chemical. In addition a chemical cannot bioconcentrate a chemical if it is too large to cross cell membranes (Connell 1998, di Giulio et al. 1995, US EPA 2000a).

Bioaccumulation (BAF) and bioconcentration factors (BCF) can be experimentally determined, in which case the balancing metabolism/excretion processes are incorporated as part of the empirically derived BAF or BCF. Unfortunately experimental BAFs and BCFs do not exist for all *'chemicals of interest'* in Table A1.3 (Appendix 1). However because there is a close association between fat solubility (measured as the log octanol water partition coefficient, log K_{OW}) and accumulation, BCFs for organic chemicals can be estimated using a software program from the US EPA (2000b) [20]. Although highly correlated with log K_{OW} the resulting calculated BCFs are not directly analogous with fat solubility and there is sufficient independence between these bioaccumulation indicators that both log K_{OW} and BCF can be used as screening criteria for bioaccumulation potential (US EPA 2000a, ANZECC 2000).

Thus log K_{OW} and BCFs have been used in screening the effluent *'chemicals of interest'* for bioaccumulation potential in a similar manner to how ANZECC (2000) have applied these parameters in deciding bioaccumulative potential in the Australian & New Zealand Guidelines for Fresh & Marine Water Quality. Thus substances that have a log K_{OW} of ≥ 4 and a BCF of ≥ 10,000 are considered to be bioaccumulative.

[20] The program is colloquially called EPISuite. It codifies the structure of the chemical to estimate the octanol water partition coefficient and then using general accumulation correlation relationships experimentally derived for chemical classes it estimates a BCF. Thus the estimated BCF inherently contains consideration of metabolism/excretion since the chemical class correlation equations are based on experimental determinations. Nevertheless the metabolism is not explicit for individual compounds and log K_{OW} dominates the calculated accumulation potential. It is likely therefore that the calculated BCF may over predict accumulation when a chemical is biotransformed (metabolised) and/or easily excreted by an aquatic organism (ANZECC 2000, Connell 1998, US EPA 2000a). In addition to this likely conservatism the algorithms in the software are based on bioconcentration data from warm water fish (fish preferences were fathead minnow>goldfish>sunfish>carp>>marine species), with data for fathead minnow being most prevalent (Meylan et al. 1999). Warm water fish generally have a higher lipid content than marine water species, the ocean outfall environment is also oligotrophic meaning fish in the area will have low lipid content because food of high calorific value is scarce. Consequently the calculated outputs of log K_{OW} and BCF from the US EPA algorithms will over estimate bioaccumulation potential for fish in the outfall area in Bass Straight. Furthermore because dietary accumulation, especially for high log K_{OW} chemicals, may have contributed to total uptake in some of the experimental data populating the algorithms it may be appropriate to consider the calculated BCFs as bioconcentration come bioaccumulation factors (Meylan et al. 1999). More information on the QSAR US EPA (2000b) EPISuite modelling is in Appendix 2.

For the purposes of this risk assessment, if an effluent constituent is considered bioaccumulative by ANZECC (2000) or US EPA (1995) then it was automatically regarded as a *'chemical of potential concern'* and was subjected to the human health risk assessment (HHRA) for seafood consumption. Unfortunately these regulatory authorities appear to have considered relatively few substances and in addition they prepared only positive lists. That is, lists of substances that meet the bioaccumulation criteria. Lists of compounds not conforming to the criteria are not available. Such compounds may not be bioaccumulative or may not have been considered. Hence all substances identified in Section 4.1 as being potentially present in the mill effluent have been screened against the bioaccumulation criteria.

Because metals are poorly fat soluble the US EPA (2000b) method of calculating BCFs has not been used; instead experimental BCFs as recommended by US EPA (2005a) have been adopted.

The screening methodology for bioaccumulation is summarised in Figure 5.1.

The first criteria relates to molecular size of the chemical. Substances with molecular weights greater than 1,000 are not readily absorbed across biological membranes (Nabholz et al. 1993). If absorption is unlikely then bioaccumulation is also unlikely as a substance only bioaccumulates if it enters the cell.

For most chemicals, it is expected that the rate of metabolism and elimination from the organism is greater than the rate of bioaccumulation. Consequently the calculated estimates of log K_{OW} and BCF (with no consideration of metabolism or elimination) will over predict bioaccumulation if a chemical is metabolised by an aquatic organism (ANZECC 2000, Connell 1998, US EPA 2000a). Therefore substances not listed as bioaccumulative by ANZECC or US EPA (1995) but which have a log K_{ow} of ≥ 4 and a BCF ≥ 10,000 are also evaluated for the extent to which they are biotransformed/metabolised by aquatic organisms. Studies on the metabolism and depuration rates of a compound or structurally similar compounds are considered on a case by case basis (refer Section 5.1 below).

Effluent constituents on the *"chemicals of interest' list* (Table A1.3) with bioaccumulative potential as described above and in Figure 5.1 are termed *'chemicals of potential concern'* and enter the HHRA for seafood consumption.

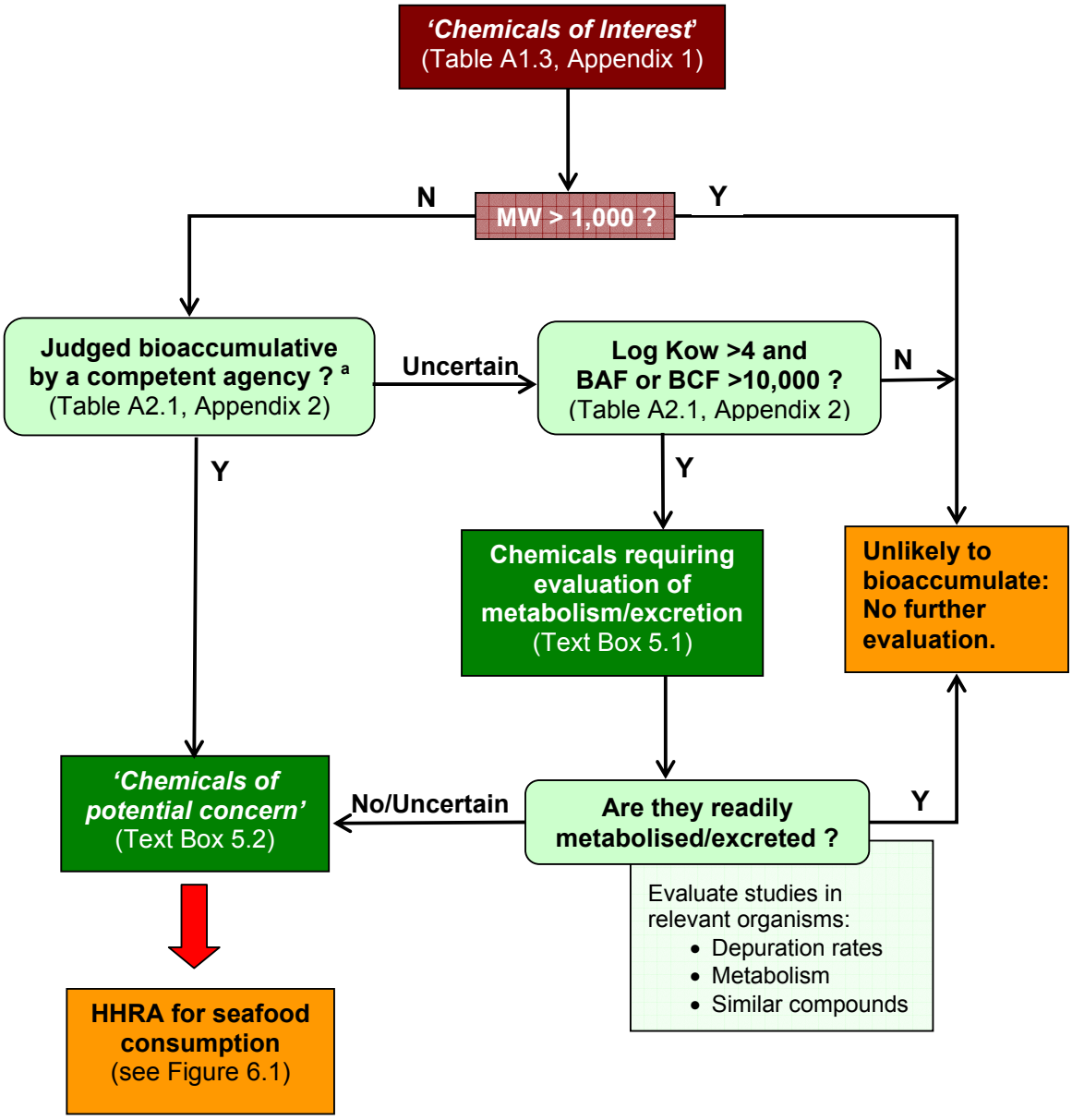


Figure 5.1: Determination of chemicals of potential concern.
[a] ANZECC (2000), US EPA (1995)

Analysis of bioaccumulation data

The detailed parameters (i.e. the log K_{OW} and BCF) for each *'chemical of interest'* are provided in Appendix 2, Table A2.1. A pictorial summary of this data is in Figure 5.2. From this chart it can be seen that although members of certain chemical classes (e.g. resin acids, fatty acids, chlorocymenes, sterols) have relatively high fat solubility (log K_{OW} >4), they do not have similarly high BCFs. When these two pieces of information are put together there are only four compounds (quadrant B of Figure 5.3), from the *'chemicals of interest'* list in Table A1.3, that satisfy the bioaccumulation screening criteria and also are not currently on competent authority lists for bioaccumulation. These compounds are chlororetene, fichtelite, and retene (all resin acids). In order to determine whether these substances should be regarded as chemicals of concern with respect to bioaccumulation, consideration of their ability to be metabolised and/or eliminated by marine organisms is required (see below).

TEXT BOX 5.1:

The screening process for bioaccumulation (i.e. MW, log K_{OW} & BCF) identified only three effluent constituents with bioaccumulation potential.

Chlororetene
Fichtelite } ***….. Resin acids***
Retene

All these substances are readily metabolised and excreted in fish and therefore with respect to bioaccumulation are <u>not</u> considered as chemicals of concern in Bell Bay mill effluent.

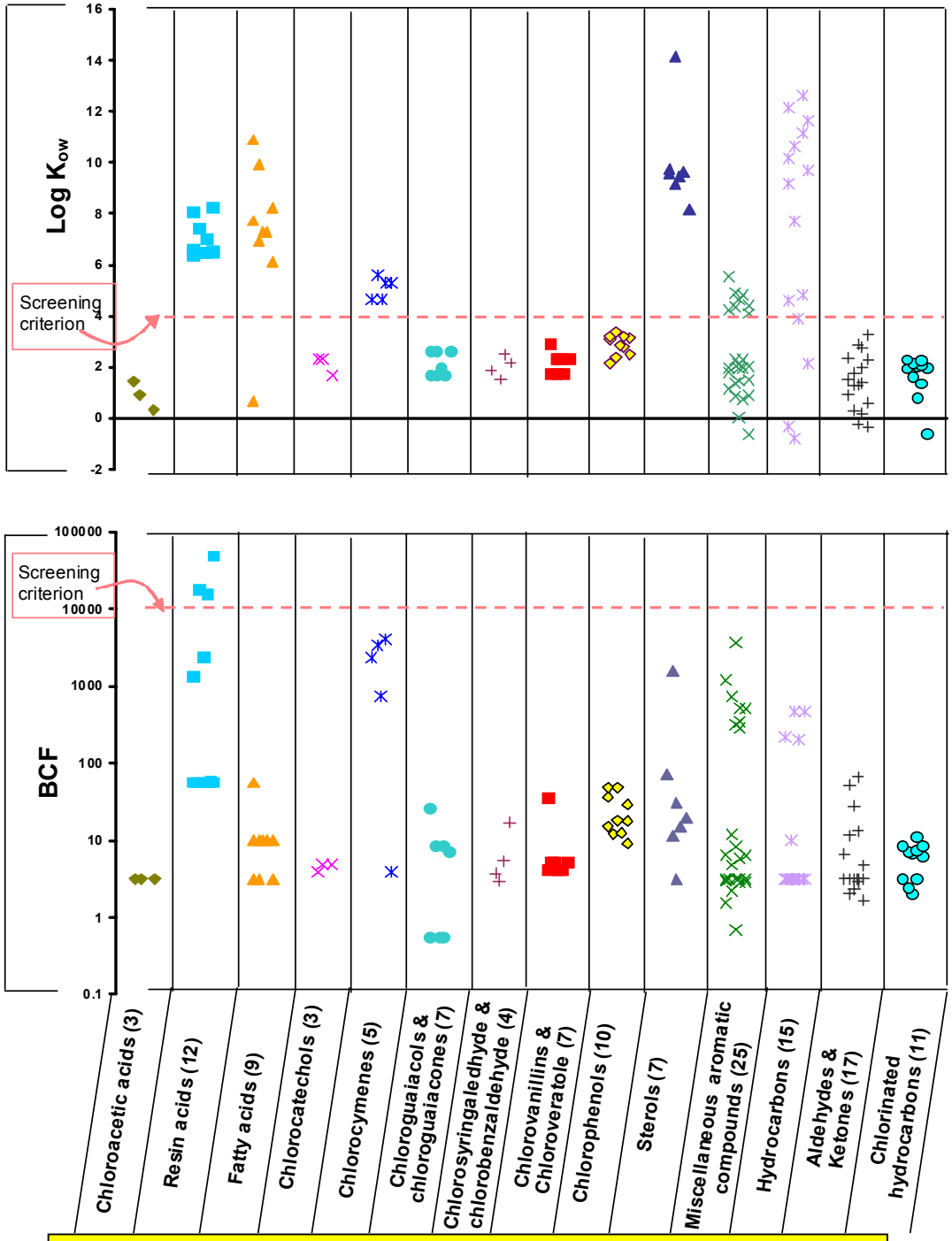


Figure 5.2: Log Kow values and BCFs for organic compounds of interest in the final effluent.

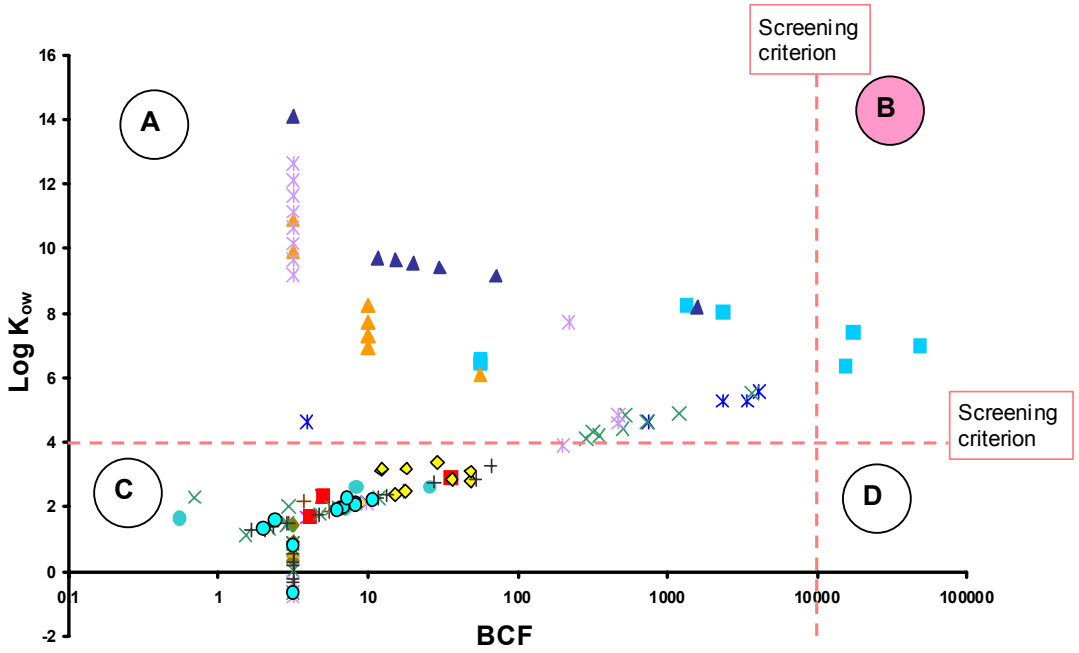


Figure 5.3: Screening for bioaccumulation of organic constituents.

Legend

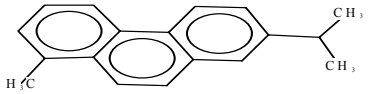
- Chloroacetic acids
- Resin acids
- Fatty acids
- Chlorocatechols
- Chlorocymenes
- Chloroguaiacols & chloroguaiacones
- Miscellaneous aromatic compounds
- Chlorosyringaldehydes & chlorobenzaldehyde
- Chlorovanillins & chloroveratole
- Chlorophenols
- Sterols
- Hydrocarbons
- Aldehydes & Ketones
- Chlorinated Hydrocarbons

Chemicals in quadrant B exceed the screening criteria of log K_{OW} >4 and BCF>10,000 and are hence potentially bioaccumulative. According to Figure 5.1 they therefore require evaluation of metabolism/ excretion.

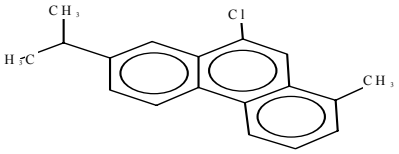
The chemicals are: Retene (a resin acid)
Fichtelite (a resin acid)
Chlororetene (a resin acid)

The three substances identified as being potentially bioaccumulative by the screening process occur in wood and are naturally present in the environment and aquatic organisms (Mcleay 1987). Retene and fichtelite are possible biodegradation products of abietic acid (Leppanen and Oikari 1999). In addition all are, or are expected to be rapidly metabolised by fish (Oikari et al. 1987, Gravato and Santos 2002, Burggraaf et al. 1996, Mcleay 1987, Muir and Servos 1996).

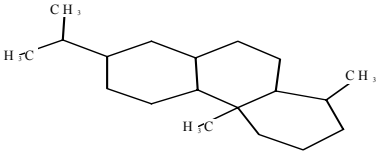
Retene



Chlororetene



Fichtelite



Within organisms resin acids and their environmental degradation products are readily biotransformed in the liver by the oxidative cytochrome P450 enzyme system responsible for normal lipid metabolism (di Giulio et al. 1995). These oxidative enzymes are present in nearly all aquatic organisms but in general the amount and activity in fish is higher than in invertebrate species (di Giulio et al. 1995). Following exposure of fingerling rainbow trout to retene two metabolites were detected in fish liver (bile). The parent compound was also distributed to bile and was rapidly excreted with a half life of approximately 14 hours (Oikari et al. 2002). Although specific studies on the metabolism of fichtelite and chlororetene were not located, they are expected to be metabolised in a similar fashion as retene based on their structural similarities. The chlorine atom on chlororetene will not interfere with side chain demethylation or oxidation of other aromatic rings.

It is also worth noting that although resin acids are a group of diterpene organic compounds that are very abundant in the resin canal of coniferous trees (Gravato and Santos 2001), the pulp process is being designed to result in at least 95% degradation of the resin acids during the wastewater treatment processes and therefore will only be present in the final effluent at very low concentrations [21].

[21] Personal communication with Jaakko Pöyry (2005).

5.2 Chemicals of potential concern

The compounds identified in the initial screening procedure for bioaccumulation are readily metabolised and consequently are not regarded as being bioaccumulative and chemicals of concern for the Bell Bay mill.

TEXT BOX 5.2:

In relation to bioaccumulation, the chemicals of potential concern for Bell Bay effluent are:

Cadmium
Mercury
Selenium
Dioxins and furans

These were identified from bioaccumulation notations in Water Quality Guidelines (ANZECC 2000).

Agency deliberations: Of the many compounds identified as being of potential interest for the Bell Bay effluent (Table A1.3), only four are regarded by ANZECC (2000) as being bioaccumulative. According to the scheme outlined in Figure 5.1, they are *a priori* chemicals of concern in the effluent and subject to the human health risk assessment for seafood.

The compounds are cadmium, mercury, selenium and dioxins/furans (PCDD/PCDFs).

6. HHRA for effluent discharge

6.1 Exposure considerations

The present assessment is exclusively focused on human health impacts of the Bell Bay effluent discharge to Bass Strait (i.e. it does not consider ecological impacts to marine organisms).

People who may be directly exposed to the effluent at the ocean outfall include workers associated with ocean outfall operations and those using the water for recreational activities, e.g. boating, swimming and diving (see Section 7). A separate report is being prepared on occupational hazard and risks to workers at the Bell Bay mill therefore workers associated with ocean outfall operations are not included in the present assessment. Recreational boaters and people fishing from boats at the outfall are not considered to be directly exposed to the effluent as it is anticipated they will not be in the water. The location (approximately 3 km offshore) and unsympathetic seas make it extremely unlikely a person will be swimming in close proximity of the outfall. Similarly the featureless coarse sandy ocean bottom surrounding the outfall and the relative lack of biota, especially scallops and abalone, make it very unlikely diving will occur near the outfall. Nevertheless for completeness, a screening risk assessment has been performed for these 'incomplete' water contact exposure pathways using Australian guidelines for recreational waters. (Section 7 with details in Appendix 6).

Potential indirect exposure to effluent constituents could plausibly occur by consumption of fish that may have accumulated the constituents.

Human consumption of shellfish (scallops and abalone) from the area has not been considered because they do not exist at the site. Furthermore, the Aquenal (2005) survey of the diffuser site describes the sea bottom as dominated by well sorted coarse sand indicative of the area being subjected to frequent high levels of water movement. Aquenal consider the lack of shellfish in the area is reflective of the lack of shelter from predators and wave action in the open sandy habitat. Toxikos is of the opinion the existing physical attributes which are detrimental to colonisation by shellfish will not be altered by the presence of the outfall.

Traditionally an exposure assessment involves the determination of the magnitude, frequency, extent, character and duration of exposures (enHealth 2002, NEPC 1999). Hence the fisherman exposure scenario requires the following broad tasks to be performed:

- Determination of chemicals in effluent likely to be accumulated, and their concentrations (see Section 5),
- Determination of the amount of chemical accumulated by fish likely to be caught in the outfall area.
- The frequency and extent fish from the area will be consumed.
- Judgement of the health impact of the calculated intake of effluent chemical.

Depending of the dispersive characteristics of the effluent, the theoretical potential zone of impact of the effluent discharge could be quite large however the area closest to the outfall will have the highest concentration of effluent chemicals. The higher the water and/or sediment concentration of effluent chemical the greater is the likelihood of significant concentrations being bioaccumulated in fish, when consumed these present the greatest risk to humans. Consequently the present assessment has focussed on identifying risks to humans within the DZ100 approximately 100 m around the diffuser.

Ideally a risk assessment of this nature should make some allowance for the territorial range of fish and their temporal movement[22] in and out of the initial dilution zone (US EPA 2005c). These factors obviously have significant impact on the exposure a fish may have to effluent and its likelihood of accumulating effluent chemicals. A small and infrequent amount of time spent in the dilution zone will not result in large amounts of uptake of chemicals and additionally time spent away from the diffuser allows the organism to metabolise and excrete any chemical which it may have bioaccumulated. Nevertheless implicit in the calculations of this risk assessment is the assumption that fish will reside at the ocean outfall most of their lives in order to accumulate chemicals to steady state[23]. This is considered conservative and to over predict the amount of chemical that may be in edible fish flesh.

Furthermore no allowance is made regarding human consumption of fish from sources other than from the vicinity of the ocean outfall. This will over predict human intake of any effluent chemical that might be in fish.

---

[22] One way of accounting for the reality that organisms do not uniquely reside in a polluted area is to incorporate a 'temporal use factor' (TUF) relating the time spent foraging at the site to that outside. TUFs are used to incorporate site specific factors that limit the time ecological receptors are expected to be present at the site (Ohio EPA 2003, Oregon DEQ 2000). The present risk assessment assumes fish reside for their entire lifetimes at the edge of the DZ100.

[23] Note this assumption is implied in the screening methodology for metals and in the calculations for dioxin because account has not been taken in either process of time spent inside or outside the zone of initial dilution, nor of the concentration gradient from the centre of the DZ100 to the 1 in 100 perimeter. The embedded explicit assumption is that fish spend all their time at the edge of the initial dilution where effluent constituent concentrations are constantly at 1/100th of that in the discharged effluent.

Additionally the number of people potentially affected is relatively small. A report on the recreational fishing activities in Tasmania (Henry and Lyle 2003) indicates approximately 1% of the 125,000 recreational fisher persons catch fish off-shore in private boats with the vast majority of these fishing on the Eastern and Southern coasts, as opposed to the northern coastal location of the outfall.

Chemicals of potential concern for human health identified in Section 5.2 (Text Box 5.2) are some metals and dioxins. These substances have quite different chemical and physical properties and are therefore considered separately in Sections 6.2 and 6.3.

6.2 Risk assessment for bioaccumulative metals

Due to the prospect of bioaccumulation the effluent constituents of potential concern are the metals cadmium (Cd), mercury (Hg) and selenium (Se), and dioxins (Section 5, Text Box 5.2).

The pulp mill effluents contain trace amounts of metals because they are present in low amounts in wood (Skipperud et al. 1998). In order to estimate the final effluent concentration of trace metals, samples of plantation and native eucalypt, and of pine wood chips representative of those to be used at the Bell Bay pulp mill were analysed for their metal concentration. Jaakko Pöyry (JP 2005e) used this data and information from the preliminary designs for the mill to conservatively estimate final effluent concentrations for the metals.

It is important to recognise that metals occur normally in marine waters from non-anthropogenic sources. The biogeochemical processes which control metal accessibility to marine organisms and the mechanisms by which they are taken up and subsequently stored are very complex (Phillip and Rainbow 1989, Boudou et al. 1998, Chapman et al. 2003, US EPA 2004). Unfortunately addressing this complexity in a quantitative risk assessment is very data intensive and much of the required information is not available. Thus the first risk assessment consideration for metals is an evaluation of the circumstances which may realise their bioaccumulation potential. Part of such an evaluation is a simple comparison of the predicted water concentrations at the perimeter of the initial dilution zone, the DZ100 [24], with background concentrations of the metals. Knowledge of the biological processes controlling the

[24] For the purposes of this risk assessment the dilution area of interest is that associated with a 1 in 100 dilution, called the DZ100. It is anticipated the DZ100 is a small area within 100m either side the diffuser at the end of the pipeline.

accumulation of the metals and whether there is a large incremental increase in receiving waters will potentially drive the need for a quantitative risk assessment.

If there is not a large, demonstrable incremental increase in metal concentration at the DZ100 then it is unlikely there will be a meaningful increase in organism body burden over and above that which currently occurs and consequently there will be no increased risk to humans consuming the organisms. Thus the first risk assessment consideration for metals is a simple comparison between the predicted effluent metal concentrations at the edge of the DZ100 and the background concentrations of the metals in the receiving seawater. A quantitative risk assessment assessing fish body burden and human intakes is pursued when metal concentrations from effluent discharge significantly increase the prevailing background water concentrations and there is likely to be a demonstrable increase in fish body burden of the chemical. The logic is summarised in Figure 6.1.

Data on metal concentrations in water, sediment and marine organisms from the proposed diffuser site (within a 1 km radius of the site) are available from Aquenal (2005). In addition, Toxikos conducted a literature review for background concentrations of Cd, Hg and Se in marine waters, specifically excluding reported information from areas that may have been impacted by anthropogenic sources (Appendix 4). These data are considered and reported in the sections below which address the metals of potential concern.

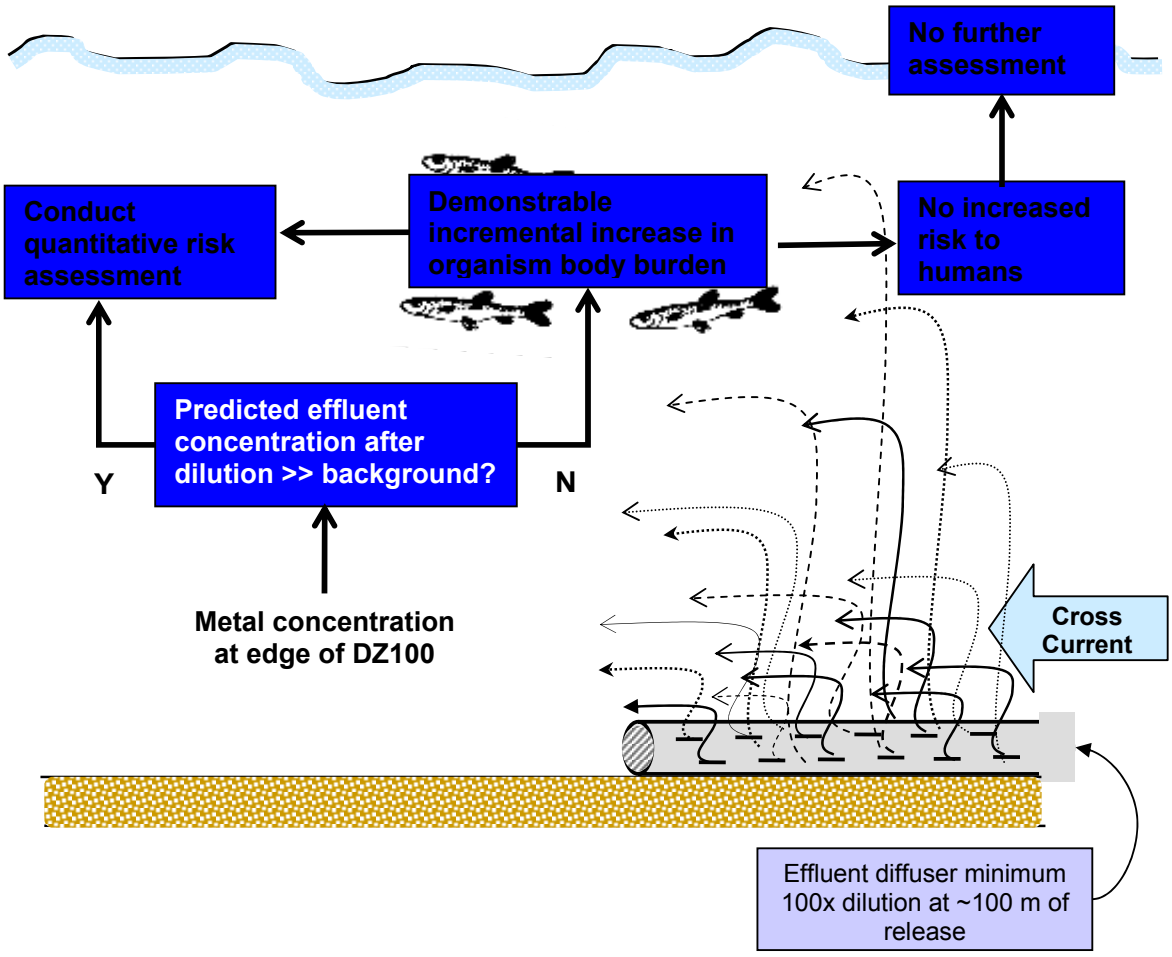


Figure 6.1: Summary of qualitative risk assessment process for bioaccumulative metals.

6.2.1 Cadmium

While cadmium has variable tendency to bioaccumulate in marine organisms bioconcentration can be significant for bivalves. ANZECC (2000) record a bioaccumulation factor of 10,000 - 20,000 which led them to conclude cadmium has bioaccumulation potential. Similar order of magnitude bioaccumulation factors are implied by Cossa (1988) who reported that concentrations of cadmium in the Gironde Estuary in France of between 0.2 µg/L and 0.4 µg/L

- **Cadmium is not readily accumulated by fish muscle.**
- **High water concentrations are needed.**
- **Long exposures are required.**
- **Cadmium is quickly lost from fish muscle.**

were associated with mussel concentrations of between 12 mg/kg and 37 mg/kg, these tissue concentrations were much higher than the 0.6 to 3.3 mg/kg found by Cossa (1988) in a worldwide survey of cadmium in mussel. It is noted the marine survey conducted by Aquenal (2005) did not find bivalves and there was a scarcity of scallops and abalone. It is also considered the physical nature of the site will remain inhospitable to shellfish after the outfall is operational.

In contrast to mussel, fish are not avid accumulators of cadmium (Jarvinen and Ankley 1999, ANZECC 2000). This may be because cadmium residues in fish muscle reach steady-state only after long exposure periods and muscle loses accumulated Cd when the fish moves to clean water (Sangalang and Freeman 1979, de Conto Cinier 1999). Pharmacokinetic modelling of data showed that while whole body cadmium levels (represented by blood, gill, gut wall, liver and alimentary canal contents but not muscle) in trout reached steady state in approximately 50 days, the concentrations in kidney did not attain steady state even after 350 days (Thomann et al. 1997). Whole body depuration occurred with an initial half life of approximately 6 days followed by a longer terminal phase with a half life of approximately 24 days [25], on the other hand kidney concentrations continued to increase during depuration. This is consistent with the experimental data of de Conto Cinier (1999) who showed exposure of carp to high concentrations of water borne cadmium resulted in concentrations of the metal increasing sharply in kidney and liver, but not muscle. For muscle, cadmium concentrations only became significant after 106 days. After 127 days exposure to 53 µg/L the cadmium concentration in kidney was 4 fold higher than in liver and 50 fold higher than muscle; at 443 µg/L these ratios were 2 and 100 respectively. With depuration however the loss of accumulated cadmium was rapid and immediate in muscle but no decrease was observed for liver and kidney. Similar results have been found for Japanese eels (Yang et al. 1996), zebrafish (Wicklund et al. 1988) and girella (Kuroshima 1987).

Metallothionein is generally regarded as a high affinity sink for some non-essential metals (Segnar and Braunbeck 1998). Cadmium is avidly bound by metallothionein and tissue

[25] These depuration half lives are graphical estimates made by Toxikos from Figure 7 of Thomann et al. (1997).

sequestering of cadmium in aquatic animals, as in mammals (Goyer and Clarkson 2001) and sea birds (Stewart et al 1996, Elliott et al. 1992, Elliot and Scheuhammer 1997), correlates with metallothionein concentration and inducibility which is highest in liver and kidney but low in muscle (Roesijadi 1992). This explains the relative inability for fish muscle to accumulate and retain cadmium, cadmium accumulated by muscle quickly redistributes to liver and kidney. Unlike marine predators humans do not usually eat the liver and kidneys of fish, but rather cut the fish into fillets of muscle.

It is concluded that cadmium does not easily accumulate in fish muscle. High concentrations in the aquatic environment and long term exposure are required before significant concentrations are observed in fish muscle (de Conto Cinier 1999, Kraal et al. 1995, Papoutsoglou and Abel 1988).

The concentration of cadmium in effluent is conservatively estimated to be 1.2 µg/L. With a mass dilution of 1: 100 the concentration at the fringe of the DZ100 would be 0.012 µg/L. While this concentration is markedly less than those associated with accumulation of cadmium in fish flesh (de Conto Cinier 1999, Kraal et al. 1995, Papoutsoglou and Abel 1988) it does not take into account background levels of cadmium in seawater.

The predicted concentration of 0.012 µg effluent cadmium/L after mass dilution at the edge of the DZ100 is clearly below the range of cadmium concentrations of <0.2-0.4 µg/L measured by Aquenal (2005) at the proposed location[26]. Elsewhere in the world background concentrations for marine waters have been measured to be between 0.001-0.7 µg/L with Australia tending towards the upper end of the range (HHRA Table A4.1, Appendix 4). Unfortunately quantitative data for cadmium in seawater and fish from the proposed site of the outfall are not available, these measurements being recorded as less than analytical detection limits by Aquenal (2005).

Conclusions:

From the above information it appears that cadmium can be bioconcentrated in marine organisms if they are sedentary, such as mussel, and there is local cadmium pollution. While some tissues of fish are able to assimilate cadmium to relatively high levels, muscle has limited capacity. This tissue requires relatively high environmental concentrations and is very slow to reach appreciable cadmium concentrations. Nevertheless when muscle does accumulate cadmium it quickly looses the metal when the fish transfers to cleaner water. The implications

[26] All seawater samples except one had Cd levels less than the analytical detection limit of 0.2 µg/L, and all fish samples were less than detection limit of 1 mg/kg (Aquenal 2005).

for the human risk assessment are that fish which are temporarily within the initial dilution zone may not there long enough to accumulate cadmium into the muscle.

A mass dilution of the effluent cadmium gives a water concentration at the DZ100 of 0.012µg/L. While this level is not expected to result in marked incremental accumulation of cadmium because it is less than background concentrations around the world and the latter are not associated with cadmium accumulation by fish, it does not take into account the background seawater concentrations at the site which are recorded as being less than analytical detection limits of 0.2 µg/L (15 of 16 samples). In addition levels of cadmium in fish at the location have not been quantitated but are less than analytical detection limits of 1 mg/kg fish (n = 20). Existing levels of cadmium in fish are not recognised as causing issues for humans.

Overall it is considered unlikely that humans will be affected by cadmium in the effluent but there is large uncertainty, due to lack of quantitative background data, associated with this opinion.

6.2.2 Mercury

A detailed evaluation of mercury accumulation by fish is provided in Appendix 8. It is expected the effluent treatment processes of the Bell Bay mill will remove most if not all (detectable) mercury before discharge to Bass Strait and that the estimates from Jaakko Pöyry are over predictions (JP 2005i).

Mercury (Hg) in the aquatic environment exists mainly as inorganic mercury or in methylated forms (ANZECC 2000, Morel et al. 1998). The major components of mercury in seawater are complexes of the divalent inorganic form, Hg^{++} (ANZECC 2000). Under certain conditions, inorganic mercury in sediments and water can be biologically converted into methylated mercury by microbes (Morel et al. 1998, Mauro et al. 2002, ANZEEC 2000).

Methylmercury (MeHg) can easily penetrate the biological membranes of microorganisms and is efficiently accumulated by these organisms by covalently binding to protein sulphydryl groups. Subsequent accumulation in aquatic food chains is mainly due to ingestion of MeHg containing microorganisms (Morel et al. 1998). Fish bioaccumulation factors for MeHg are consequently very high (US EPA 2001, ANZECC 2000). The biomagnification of methylmercury through the food chain is exemplified by the increasing fraction of MeHg of the total mercury in aquatic organisms as one moves up the trophic layers. The fraction of total mercury as MeHg in

aquatic organisms is 15% in primary producers , 30% in zooplankton and 95% in fish (Watras and Bloom 1992).

The covalent binding of MeHg to protein results in a long half-life for elimination of about two years (Wiener and Spry 1996). Given steady environmental concentrations, MeHg concentrations in individuals of a given fish species tend to increase with age as a result of the slow elimination and increased intake due to changes in trophic position that often occurs as a fish grows to larger size (i.e. fish eat more, and bigger prey as they grow larger) . Therefore, older fish typically have higher mercury concentrations in the tissues than younger fish of the same species (UNEP 2002).

The net methylation rate of mercury in sediment can strongly influence the amount of MeHg that is produced and available for accumulation and retention by aquatic organisms. While much is generally known about mercury bioaccumulation and biomagnification, the process is nonetheless extremely complex and involves complicated biogeochemical cycling and ecological interactions. As a result, although accumulation/magnification can be observed, the extent of mercury biomagnification in fish is not easily predicted across different geographic locations and local data is important in assessing the impact of point sources.

At the edge of the DZ100, the 100 fold mass dilution will result in a Hg concentration of approximately 0.003 µg/L. While this is within the range of Australian coastal and open ocean waters the existing background concentration of Hg in the receiving water should also be taken into consideration. Unfortunately an accurate quantitation of Hg in the water around the proposed diffuser site is unavailable; recent seawater analyses by Aquenal (2006) placed the Hg concentration at less than the analytical detection limit of 0.1 µg/L. The Department of Primary Industry, Water and the Environment (DPIWE 2006) found the concentration of mercury in seawater to be below detection (<0.05 µg/L) at Hebe Reef and other locations in the Tamar estuary[27]. In Appendix 4 background concentrations of Hg for Australian coastal waters are recorded to be <0.001 – 0.02 µg/L (DEH 1995). Assuming the Hg level at the diffuser site is at the midpoint of this range the concentration would be approximately 0.01 µg/L.

[27] The Australian and New Zealand Environmental Conservation Council (ANZECC) have established a water quality guideline (WQG) for mercury in marine waters of 0.1 µg/L. The WQG is intended for the protection of aquatic organisms however ANZECC consider that the use of the WQG designed for the protection of areas of high conservation value (i.e. 99th percentile level of protection) to be precautionary for bioaccumulation and biomagnification in aquatic organisms.

Using this value for the existing background seawater Hg concentration gives a 1:100 dilution of effluent Hg when seawater is the diluent as follows:

$$[(1 \times 0.275\ \mu g/L) + (99 \times 0.01\ \mu g/L)] \div 100 = 0.013\ \mu g/L$$

This assumed DZ100 concentration of 0.013 µg/L is within the concentration range reported for Australian coastal waters and it might therefore be expected that, because the discharged Hg reaches assumed background concentrations very quickly, existing fish Hg concentrations will not alter very much as a result of effluent discharge. However there is a great deal of uncertainty associated with the above background Hg concentration assumption; the calculation should only be used to provide a rough indication of the possible impact of discharged effluent Hg on Hg concentrations in sea water surrounding the diffuser. The higher the background seawater Hg concentration the less influence there will be from effluent Hg.

To supplement the tacit implication of no impact on human health from the above consideration of effluent dilution, the theoretical incremental increase in fish MeHg levels due to discharged Hg has been calculated (Appendix A8.2). The theoretical incremental increase was then added to the results of measurements of existing Hg in fish to enable comparison with the Australian food standard for Hg in fish (Appendix A8.3). In addition, total Hg intake (background + incremental) by humans consuming fish sourced from the proposed outfall has been calculated for comparison with health based intake standards[28] protective of human health.

Thus the incremental increase in fish MeHg concentration due to discharged Hg in effluent is:

$$IC_{FISH} = (Hg_{EFF} \times C_{Hg\text{-}MeHg}) \times BAF$$
$$= 0.00275\mu g/L \times 0.03 \times 320{,}000\ L/kg$$
$$= 26.4\ \mu g/kg \text{ wet weight fish } (0.026\ mg/kg \text{ fish}).$$

Where:
- IC_{FISH} = Incremental increase in fish MeHg due to effluent.
- Hg_{EFF} = Hg concentration at DZ100 (0.00275 µg/L) due to effluent.
- $C_{Hg\text{-}MeHg}$ = Conversion of effluent Hg to MeHg (3%, see Appendix A8.2).
- BAF = Bioaccumulation Factor for MeHg (320,000 L/kg; see Appendix A8.1).

[28] The health based guidelines for MeHg intake fro protection of human health are called provisional tolerable weekly intakes (PTWI).

In two rounds of sampling, April 2005 and February 2006, a total of 39 fish of different varieties have been caught within 250m to the east and west of the proposed diffuser site and analysed for Hg concentration (Aquenal 2005, GHD 2006a).

The concentration of Hg in the 39 fish was 0.22 ± 0.15 mg/kg (mean ± SD, n = 39). Twenty four of the fish had Hg levels below the analytical detection limit[29] hence to calculate the statistics the level of Hg was assumed to be at half the detection limit for these animals. The distribution of Hg in fish is shown in Figure A8.1 in Appendix 8. If the discharged Hg in the effluent caused an incremental increase in fish Hg content of 0.026 mg/kg as conservatively calculated above, the average fish Hg concentration will increase from 0.22 mg/kg to 0.25 mg/kg. The consequence of this incremental increase on the distribution pattern of Hg in fish is also shown in Figure A8.1 in Appendix 8. It should be noted an incremental increase in fish Hg of this magnitude will not be statistically demonstrable if measurements are conducted on fish with techniques usually employed for monitoring fish Hg levels. This is especially so given the estimates of Hg in the effluent are considered to be over predictions and a high conversion of Hg to MeHg is assumed in the calculations for estimating incremental increases in fish Hg.

In relation to compliance with the 0.5 mg/kg Hg fish standard of the Food Standards Australia and New Zealand (FSANZ 2006) the average of the new distribution for Hg in fish will be 50% of the standard and will therefore remain compliant with the standard.

MeHg intake by humans consuming fish sourced from the outfall assumed all fish eaten came from the outfall and that the maximum empirically derived conversion of effluent Hg to MeHg was operable in the vicinity of the outfall. Furthermore the calculations were performed for the average, 95th percentile and maximum fish intakes of various population sectors (Appendix A8.4). None of the calculated intakes of MeHg exceeded the relevant[30] health based PTWI for women of child bearing age, for the general population or for children between 2 – 6 years old. Given the conservative assumptions used in the calculations it is concluded the incremental human health impact from Hg in the discharged effluent is negligible.

---

[29] The analytical detection limit (DL) for Hg in fish was 0.1, 0.2 or 0.5 mg/kg depending on the analytical run. The assignment of 0.5 DL for fish with Hg analytical non-detects in the calculation of statistics was done according to the respective detection limit for the batch within which the specific non-detect fish resided.

[30] Because the foetus is more sensitive to the harmful effects of MeHg than are adults FSANZ has applied two separate upper safe levels of dietary intake for their risk assessments (FSANZ 2004). The first is a PTWI considered to be protective of the general population and a lower level considered to be protective of the foetus. The level set to protect the foetus is 1.6 µg MeHg/kg bw/week and is approximately half the level used for the general population (3.3 µg MeHg/kg bw/week).

It is noted that existing background intakes dominate the overall intake of MeHg from fish by providing 90% of MeHg intake after the effluent outfall becomes operational.

Conclusion

The estimated mercury concentration at the fringe of the initial dilution zone (DZ100) is within the background range measured around the world and Australia, and tentatively supports the notion any incremental increase in MeHg concentrations in biota around the outfall will be minimal. There are however large uncertainties associated with input parameters for this process. Consequently other assessment techniques have also been used to evaluate the potential impact of Hg in the discharged effluent on human health. Firstly theoretical incremental increases in fish MeHg concentrations due to Hg in effluent demonstrated total fish Hg levels would increase only slightly and would remain at a level approximately half of that specified in the Australia New Zealand Food Standards Code (FSANZ 2004). Secondly conservative calculations of human intake of MeHg (background + incremental from effluent Hg) showed that within all population sectors, high-end fish consumers sourcing all their fish intake from around the effluent outfall would have intakes less than the relevant health guideline for the population sector.

All these lines of assessment have lead to the conclusion that the incremental human health impact from Hg in the discharged effluent is negligible.

It is noted that existing background intakes dominate the overall intake of MeHg from fish, background sources provide 90% of the MeHg intake.

6.2.3 Selenium

Selenium is an essential micronutrient in animals. Trace concentrations are required for normal growth and development, and at moderate water concentrations of selenium homeostatic regulation maintains body burdens within normal physiological ranges (Hamilton 2004, ANZECC 2000). Fabris et al. (2005) studied selenium concentrations in edible tissue of snapper, flathead, lobster and abalone in coastal waters of Victoria. Although the concentration of selenium in water or sediment was not reported an important observation was the concentration of selenium was approximately uniform at ~0.5 µg/g in all finfish populations examined and was consistent with previous studies cited by the author for a large number of fish species obtained from near-shore environments of Australia. The explanation being that for

essential elements biochemical uptake mechanisms maintain constant tissue concentrations (e.g. Skinner et al. 2004) therefore at low water concentrations, presumably at or about background, bioaccumulation is not expected.

Whole body half-lives of selenium range from 20 to 30 days for small fish, however in whole adult flathead, minnow, muscle of rainbow trout, sub-adult bluegill and large-mouth bass it was 50-60 days and greater than 100 days in large razorback sucker (Hamilton 2004).

- **Selenium is an essential element.**
- **Fish uptake controlled by homeostatic mechanisms.**
- **Accumulation only when homeostatic processes overwhelmed.**
- **Accumulation requires concentrations >3 – 5 µg/L.**
- **Water concentration due to effluent 0.075µg/L.**
- **Water concentration at site <0.2 – 2 µg/L.**

Selenium can bioaccumulate 100 - 30,000 times in organisms at the base of food webs, i.e. in aquatic plants and invertebrates eaten by fish (Lemly 1999, ANZECC 2000, Sappington 2002, Hamilton 2004). In experimental and field studies conducted in ponds and isolated river channels of the selenium contaminated Colorado River concentrations in zooplankton were proportional to the waterborne selenium concentrations ranging from 2.2-9.5 µg/L. Notably part of the selenium taken up by zooplankton was probably waterborne organo-selenium compounds released from living and/or decaying algae (Hamilton 2005).

A series of experiments in lakes in Sweden confirmed that selenium bioaccumulated in fish via the food chain if waterborne selenium concentrations were greater than 3-5 µg/L (Paulsson and Lundbergh 1989, 1991, 1994, Lindqvist et al. 1991). Similarly studies in Canada concluded selenium was accumulated through the food chain of fish and recommended aquaculture “additions” of selenium be limited to 1 µg/L (Rudd et al. 1980, Turner and Rudd 1983, Turner and Swick 1983).

The estimated concentration of effluent derived selenium at the periphery of the DZ100 around the outfall is 0.075 µg/L. This is within the range for coastal waters around Australia (<0.01-0.08 µg/L) and world wide sea water (0.009-0.45 µg/L) (Appendix 4). Data is not available for the selenium concentration of seawater local to the proposed outfall. However if it is assumed the concentration is at the high end of that for coastal Australian waters then at the edge of the

DZ100 the concentration[31] of selenium will be 0.15 µg/L. Thus the predicted selenium concentration attributable to mill effluent at the DZ100 is much less than water concentrations identified as resulting in bioaccumulation of selenium by fish. Furthermore the concentrations are less than the 10 µg/L ANZECC (2000) guidelines for protection of aquaculture and human consumption of seafood.

Conclusion

Selenium is an essential element required by fish and other animals for maintenance of normal biochemical functions. Its' uptake is controlled by homeostatic mechanisms and accumulation occurs when these are overwhelmed. This can happen when water concentrations are greater than 3 – 5 µg/L. Because the estimated selenium concentration at the margin of the DZ100 is within the background range measured around the world and at least an order of magnitude less than the lowest water concentration associated with accumulation in fish, and less than the ANZECC water quality guidelines for protection of aquaculture, it is concluded an incremental increase in selenium concentrations in biota around the outfall is unlikely. Consequently there will be negligible impact on human health.

[31] Detection limits for selenium in seawater in analysis conducted by Aquenal (2005) were 2 µg/L, results were <2 – 2 µg/L and are therefore not informative. Assuming background concentration is 0.08 µg/L then a 1:100 dilution of effluent with this seawater will give rise to a concentration at the edge of the DZ100 of [(1 x 7.5 µg/L) + (99 x 0.08)]/100 = 0.15 µg/L.

6.3 Risk assessment for dioxins

6.3.1 Is a quantitative risk assessment for dioxins necessary?

The term 'dioxins' is used to describe a group of environmentally persistent halogenated aromatic hydrocarbons that includes polychlorinated dibenzodioxins (PCDDs), polychlorinated dibenzofurans (PCDFs), polybrominated dibenzodioxins (PBDDs) and polybrominated dibenzofurans (PBDFs). Historically only chlorinated dioxins/furans [32] have been identified in pulp mill effluent. Other dioxin-like substances not found in pulp mill effluent are the polychlorinated biphenyls (PCBs). These compounds have been widely used in manufacturing, electronics and power industries and the PCBs are now globally distributed and can be found in tissues of many marine mammals. Although they are chemically very different from dioxins their environmental and human health risks are usually expressed as dioxin equivalents (see Section 6.3.2); this can create confusion and the perception that dioxins are present when in fact it dioxins may be at very low concentrations in the organism. PCBs have significant potential to bioaccumulate and biomagnify through marine food webs but dioxins do not. The difference between the bioaccumulation of PCBs versus dioxins in marine mammals is due to dioxins being metabolised whereas PCBs are not. Thus reports of high concentrations of dioxin equivalents in blubber or flesh of marine mammals relate to bioaccumulation of PCBs and not dioxins (see discussion below). PCBs are not formed in the Bell Bay pulping process and no PCBs are released into the environment.

✓ Dioxin formation is very limited in Bell Bay mill processes.

✓ Dioxins in discharge effluent are anticipated to be non-detectable.

✓ Dioxins do not biomagnify between fish and their food or between fish and sediment.

Conclude a quantitative HHRA is not required for the fisherman scenario.

Nevertheless a HHRA is undertaken using worst case assumptions to address stakeholder concerns.

The process chemistry of modern pulp mills not conducive for dioxin formation (Section 4). This, coupled with the fact that any dioxins formed will partition to the sludge of the treatment ponds, results in the dioxin content of effluent from most modern mills being either less than analytical detection limits (JP 2005d). Consequently only very low amounts of dioxin like material are expected to be in the discharged Bell Bay mill effluent.

[32] Throughout this document the term 'dioxin' or 'dioxins' collectively refers to all polychlorinated dibenzodioxins (PCDDs) and dibenzofurans (PCDFs) congeners.

In an Australian ecological risk assessment conducted for the Department of Environment and Heritage, Gatehouse (2004) makes an overview comment that available field-based aquatic bioaccumulation studies generally show a progressive increase in tissue dioxin like material from low to high trophic levels. This statement appears to be contradictory to detailed investigations of dioxin bioaccumulation, notably in the Great Lakes, where biomagnification of TCCD through the food chain "*is significant between fish and fish-eating birds but not between fish and their food, or fish and sediment"* (Gatehouse 2004) [Indicating no bioaccumulation by fish to greater levels than is in their environment].

The latter conclusion by Gatehouse (2004) is supported by Australian studies in which biosediment accumulation factors[33] (BSAF's) for a variety of fish species and bivalves have been empirically determined to be less than unity (Gatehouse 2004, discussed in more detail below), and by studies not reviewed by Gatehouse (2004) but described later in this section.

Potential bioaccumulation of dioxins in transplanted cultured mussels from an ocean outfall at the Ninety Mile Beach (in Bass Strait) in Victoria was investigated by Haynes et al. (1995). The outfall is situated 1.2 km offshore in 15m of water and at the time was discharging approximately 40 ML/day of secondary treated complex effluent. The effluent consisted of domestic and light industrial waste waters (~16 ML/day), effluent from a bleached kraft pulp and paper mill (~15 ML/d) plus oil and gas production waters from the Bass Strait region (~9ML/d). Mussels were deployed in approximately 16m of water at the outfall, 1, 7 and 10 km down current from the outfall and at a control site 50 km up-current. Mussel dioxin profiles were similar to those in mussels grown in unpolluted seawater and the authors concluded "*tissue concentrations of dioxins and furans in deployed mussels provided no evidence that bioaccumulation of dioxins discharged from the ocean outfall was occurring".*

The fish's food is the most important exposure source for uptake of dioxins; direct uptake from water via gills and skin is negligible due to very low dioxin water solubility (Gatehouse 2004). According to Gatehouse (2004) benthic feeding fish (bottom dwellers and demersal feeders) consistently contain more dioxins than other fish. The relationship between sediment concentration and fish tissue concentration is therefore more important than the relationship between water column concentration and fish tissue concentration (i.e. the bioaccumulation

[33] The biosediment accumulation factor (BSAF) for TCDD is the ratio of contaminant concentration in the organism (normalised for lipid content) to the concentration in dry weight sediment (normalised for organic carbon content). Because it is empirically derived from field data it takes into consideration dioxin accumulation that occurs not only by direct transfer from water or sediment to the organism but also from the food the organism eats and therefore potential biomagnification.

factor BAF is more important than the bioconcentration factor, BCF). This is consistent with the fact that in polluted waterways around the world virtually all the dioxins partition into sediment.

The relationship between sediment dioxin concentration and fish tissue dioxin concentration is determined from field trials and is called the biota-sediment accumulation factor (BSAF). It is simply the ratio of the concentration in the fish to the concentration in sediment after both media have been normalised for certain factors.

Because BSAFs are based on field data, the values also incorporate the effects of chemical bioavailability and uptake from all segments of the animal's environment; including sediment, water column, and food web. Other factors such as metabolism, depuration, biomagnification, fish growth effects and others are inherently accounted for in the BSAF (Hendricks et al. 1998, Burkhard and Lukasewyez 2000, US EPA 2004, US EPA 1995, Cook and Burkhard 1998). Because the BSAFs are empirically determined they are particularly useful for chemicals, such as dioxins, which may be detectable in fish tissues and sediments but are difficult to detect or measure precisely in the water column. For this reason US EPA (2004) consider the BSAF for dioxins to be a more reliable measure of bioaccumulation potential than bioaccumulation factors (BAFs) and/or bioconcentration factors (BCFs).

BSAFs are specific for the locations from which sediment and fish samples were obtained. In order to make the BSAF applicable to other locations the BSAF needs to be made location specific for dioxin sediment concentration, sediment organic carbon content, and location specific dioxin concentration in fish and the lipid content of the fish.

As a component of the Australian National Dioxins Program, PCDD/PCDFs were measured in sediment and biota from a number of sites around Australia and BSAFs calculated for bivalves and various Australian species of fish (Gatehouse 2004, Müeller 2004). Commercial fishermen supplied fish samples that were caught in close proximity to the sediment sampling locations. The BSAFs for fish were calculated by dividing the fish tissue TCDD concentrations by the average surface sediment TCDD concentration in the catching vicinity. Gatehouse (2004) normalised the BASF according to the lipid content of the fish and the organic carbon content of sediment from the locality from which the fish was caught using the data provided in Müeller (2004). All BSAF for dioxins were much less than 1.0. This result is consistent with most fish BSAFs reported in the literature and indicates dioxins are not bioaccumulated to concentrations higher than are in sediment (Gatehouse 2004).

The lack of significant bioconcentration does not however mean fish do not take up dioxins from their environment. In locations where there has been high, point source contamination of sediment some fish have been found to have higher levels of dioxins compared to fish from in non-polluted areas, however dioxins do not biomagnify between fish and their food or between fish and sediment (Gatehouse 2004).

Dioxins are considered for a quantitative risk assessment herein because ANZECC (2000) has nominated 2,3,7,8-TCDD as being bioaccumulative (Section 5.2 of HHRA). The ANZECC bioaccumulative opinion for dioxins is not well documented but is apparently founded on "*elevated dioxin levels found in fish, shellfish and sediments in some localised urban and industrial areas in Australia (Thompson et al. 1992) and overseas (US EPA 1984a, CCREM 1987, Palmer et al. 1988)".* ANZECC (2000) do not provide discussion of the content of the references they cite nor any specific technical information relating to bioaccumulation of dioxins.

The US EPA, CCREM and Palmer references quoted by ANZECC (2000) are not readily available. However the Thompson et al. (1992) reference relates to *very high local pollution* in Homebush Bay where large amounts (between 200-300 tonnes) of dioxin-contaminated wastes were produced between 1949 and 1976 by industry on the banks of the Bay. Subsequent surveys in the 1980s showed mean concentrations of TCDD varied from <4 to 181 pg/g - wet wt fish depending on species and 29 to 116 pg/g in invertebrates. Surface sediments, down to 50mm depth, contained an average of 9.6±18 pg TCDD/g dry weight sediment. As a result of these surveys, fishing has been banned in Homebush Bay (Rubinstein & Wicklund 1991). These data suggest bioaccumulation of TCDD from high point source contamination of sediment into some types of fish. On face value the data appear at variance to other studies. It is noted however the mean concentration data reported by Thompson et al. (1992) has very large standard errors and it is not possible to determine whether the fish have bioconcentrated dioxins to a greater extent than is present in their environment.

The US EPA (2000) dioxin reassessment makes some general observations in relation to the data it reviewed:

- *"For fish, the concentrations of CDDs and CDFs are dependent on the exposure level, fat content, living habits, and the degree of movement of the species. Comparatively high fat-content bottom fish, collected close to the contaminant source, generally have*

the highest CDD/CDF levels; whereas, lower fat content, non-stationary fish have much lower concentrations, even in the vicinity of the contaminant source.

- *The US National Dioxin Study indicated that the levels of 2,3,7,8-TCDD in fish from the Great Lakes Region were higher than those from urban areas. Comparable levels were detected in whole bottom feeders and predators from the Great Lakes Region".* [This latter statement signify's lack of biomagnification of dioxins].

Conclusion:

The information discussed above collectively supports the view that:

- dioxins are not significantly bioaccumulated or biomagnified by fish or bivalves.
- fish living in local environments where dioxin concentrations are low also have low levels of dioxins.

Direct evidence for lack of biomagnification of dioxins through trophic levels of a food web has recently been provided by Wan et al. (2005). In an investigation of a food web in Bohai Bay [34], north China, these workers found lipid concentrations of low chlorinated 2,3,7,8- substituted dioxins and furans did not exhibit significant trends with trophic layers. In fact the concentrations of higher chlorinated dioxins and furans declined significantly with increasing trophic levels. There were however significant positive relationships between trophic layer and accumulation of PCBs. Given the similarity in fat solubility between dioxins and PCBs the authors concluded the difference in trophic transfer (none or little for dioxins but positive for PCBs) is mainly due to their different metabolic transformation rates in the higher trophic levels.

The difference between PCBs and dioxins in bioaccumulation potential is also seen in marine megafauna. Muir et al. (1996) studied beluga whales in the St Lawrence seaway; in males the geometric mean concentration for TCDD TEQ for PCBs was 1070 pg/kg blubber but that for dioxins only 0.1 pg/kg. Other studies in different whales and porpoise reported by Muir et al. (1996) showed similar relationships. Based on the ratio of mirex to TCDD in Lake Ontario and the assumption of similar transport and accumulation properties, the authors considered the TCDD concentration in beluga blubber should be 5 – 6 orders of magnitude (10^5 to 2 x 10^6) times higher than measured.

[34] Bohai Bay is an enclosed inner sea in north China. It is a highly developed economic area and about 1 billion tons of waste water have been discharged into the bay. The study analysed 9 dioxins, 11 furans and 12 PCBs in samples of phytoplankton/seston, zooplankton, 3 invertebrate species, 6 fish species and one marine mammal.

In relation to seals, the Swedish Dioxin Survey measured concentrations of 17 dioxin and furan congeners in tissues of several fish species, in fish eating birds, and in marine mammals[35] obtained from different sites along the Swedish coast. While there were geographical differences between dioxin/furan levels and patterns in fish, which could be linked to specific point sources of dioxins/furans, and levels were higher in fish eating birds compared to their prey, dioxin concentrations in seals did not indicate biomagnification (de Wit et al. 1992). This conclusion is further supported by a study (Bignert et al. 1989) of four seal species[36] from widely different areas around the Scandinavian Peninsula that are variously impacted (or non-impacted) by anthropogenic sources of dioxins The study showed no substantial species or spatial differences in levels of dioxins/furans. Among marine mammals highly persistent organochlorines (e.g. DDT or PCBs) normally increase with increasing age, this relationship was not observed for dioxin/furan concentrations in seals (Bignert et al. 1989), a phenomenon also seen in other studies (Addison et al. 2005). The results are consistent with expectations for a substance that is not highly bioaccumulative and does not biomagnify, and are explained by rapid metabolism of dioxins/furans by seals.

De Swart et al. (1995) fed two groups of approximately 1 year old harbour seals for two years with fish from two sources. One source had approximately ten fold more TCDD TEQ than the other. While at the end of the two years there was a 3½ difference in blubber concentration between the groups of seals, in both groups the levels of dioxins were lower than in the fish fed to them and estimated body burdens were less than 0.1% of the cumulative intakes of the compounds. These data show dioxins in fish are not readily assimilated by seals and the authors suggest seals may have an efficient mechanism for either excreting or metabolising dioxins.

Blubber of harbour seals from the Strait of Georgia, British Columbia, Canada (in 1991 and 1992) contained higher concentrations of PCDD/F than did samples from Quatisino Sound on western Vancouver Island (Addison et al. 2005, Addison & Ross 2001). Historically the Strait of Georgia received effluent from six coastal pulp mills which used elemental chlorine in the bleaching process with poor effluent control systems, they also used a wood-chip feedstock that had been preserved with pentachlorophenol (Addison & Ross 2001). The Strait of Georgia receives treated and untreated industrial and domestic wastes from surrounding communities. Thus the effluent quality and quantity discharged into the Strait of Georgia was quite different from that to be discharged by the proposed Bell Bay mill. In contrast Quatsino Sound receives

[35] Blubber of grey, harbour and ringed seals.
[36] Grey seal, common seal, harp seal and ringed seal.

effluents from some mining operations and a single sulphite pulp mill which has only used mild chlorine bleaching and a wood feedstock free of PCDD/F precursors. The total dioxin levels in harbour seals in Strait of Georgia were approximately 5 to 8 times those in Quatisino Sound; the latter were 10-30 pg/g blubber lipid wet wt which is comparable with data recorded from uncontaminated sites in eastern Canada, western Europe and the Arctic (Addison et al. 2005). Although still different from the Bell Bay mill processes, which will use no elemental chlorine in its bleaching process, the combination of mine and sulphite pulp mill effluent into Quatisino Sound did not result in increased dioxin concentrations in seals.

As is the case with fish, *relatively high* concentrations of dioxins in a local environment can result in higher concentrations of dioxins in marine mammals compared with those from pristine areas. This does not mean however dioxins in mammals in the polluted area are the result of biomagnification through the food chain.

Conclusion:
The weight of evidence of the studies reviewed above indicates dioxins are not avidly bioaccumulated and are not biomagnified by marine mammals.

Thus for the Bell Bay mill effluent dioxins are not expected to be detectable due to improved process chemistry and effluent treatment compared to mills of the 1980s. Furthermore dioxins appear not to be avidly biomagnified from sediment or food by fish and are not biomagnified through the food chain. These specifics indicate a quantitative risk assessment, based on the twin perceptions of high levels of dioxins in effluent and biomagnification by fish is not warranted. Nevertheless, in order to address stakeholders' perceptions concerning dioxins in kraft mill effluent a quantitative risk assessment for these compounds has been undertaken herein.

Belief that kraft pulp mills cause significant dioxin environmental contamination plus conviction by some stakeholders that dioxins biomagnify and are 'super' toxic generate emotive arguments. Consequently specific effort has been taken in this risk assessment to provide appropriate information on dioxin toxicity and the benchmark used for judging human health impacts of dioxin exposure.

6.3.2 What are dioxins?

Polychlorinated dibenzo-p-dioxins (PCDDs) and polychlorinated dibenzofurans (PCDFs) are collectively called dioxins. Co-planar polychlorinated biphenyls (co-planar PCBs) possess toxicity similar to that of dioxins and are called dioxin-like compounds. Dioxin or dibenzo furan molecules consist of two benzene rings joined together by oxygen atom(s) with various amounts of chlorine or hydrogen atoms attached in the numbered positions of Figure 6.1 There are 75 kinds of PCDDs, 135 PCDFs and more than 10 co-planar PCBs. The different types of dioxins are called congeners.

Dioxins have no known industrial use but occur as unwanted by-products of some industrial and combustion processes such as metal smelting and burning wastes and fuel. In contrast PCBs have been used for a variety of industrial purposes including heat transfer agents, dielectric fluids for capacitors and transformers, plasticisers and paint additives (Safe 1990). Dioxins are ubiquitous in the Australian environment (e.g. Gaus et al. 2001), it has been estimated that bushfires contribute at least 20 – 30% of the total release of dioxin-like compounds to the Australian environment. Thus humans have been exposed to low levels of dioxins and human metabolism coping for thousands of years (OCS 2004). More than 95% of intake of dioxins by the general public is primarily via the diet, with the majority of this due to fatty foods derived from animals and fish/shell fish (Dijen Liem et al. 2000, Llobet et al. 2000, WHO 2000).

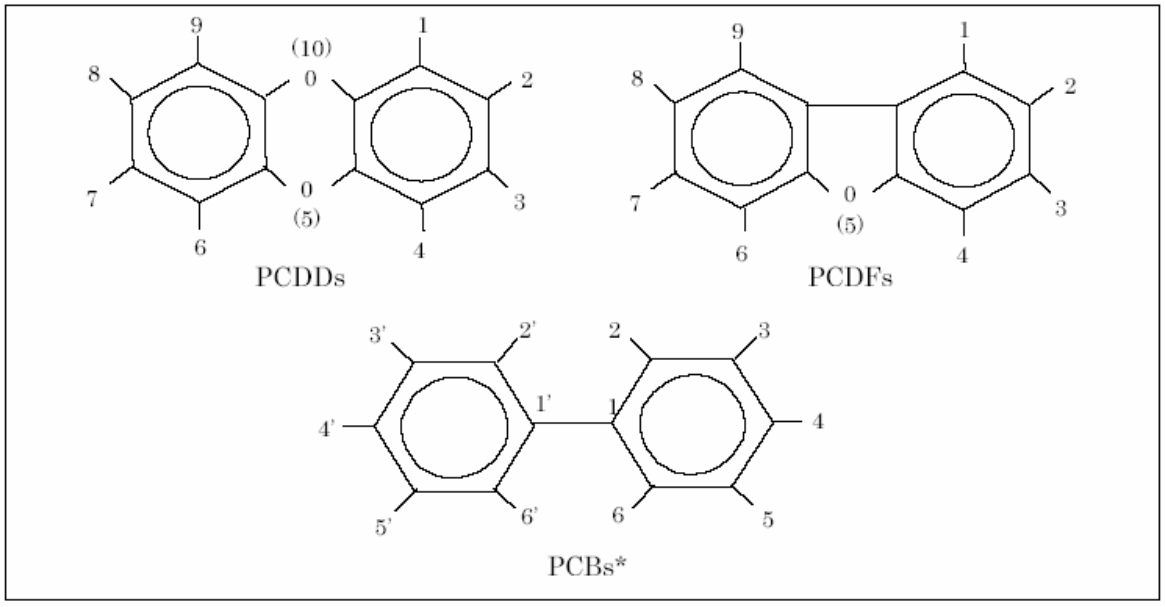


Figure 6.1: Structures of dioxins, dibenzo furans and PCBs.

Human exposure to dioxins in the environment or in food is invariably to a complex mixture of many dioxins and furans however the degree of toxicity of dioxins varies from compound to compound. Only a subset of dioxin congeners produce toxicity. The tetrachlorinated dibenzo-p-dioxin with chlorine atoms attached in the 2, 3, 7 and 8 positions (2,3,7,8-TCDD usually simplified to TCDD) is known to possess the highest toxic potency and toxic effects of this congener have been the most studied. Because dioxin congeners that cause toxicity appear to do so via a common mode of biological action (binding to a specific receptor inside cells [37]), it is possible to rank the toxicity of various dioxins, furans and co-planar PCBs relative to the toxicity of TCDD. Thus the 'toxic' dioxins are assigned a 'Toxicity Equivalency Factor' (TEF) relative to TCDD according to their ability to bind to the receptor and elicit the activated receptor mediated biochemical and toxic responses. The TEFs developed by the World Health Organisation (van den Berg et al. 1998) are widely accepted as being the most appropriate for human risk assessment and have been adopted by Australian authorities (OCS 2004). They are however heavily dependent upon the biochemical responses elicited in rat tissues, especially the relative ability of the congeners to induce cytochrome P450 mediated enzyme activities [38]. Development of a recent refined database of potency estimates for dioxin-like compounds indicates that on balance the TEF value recommended by the WHO (van den Berg et al. 1998) are in the upper range of potencies based on different endpoints (Haws et al. 2006), they do not however necessarily represent the maximum value. In addition, as discussed in Section 6.3.4, humans are at least ten times less sensitive than rats and the assigned TEFs over estimate risk to humans.

The toxic potency of a dioxin mixture is estimated by multiplying the mass concentration of each individual congener by its respective TEF, the sum of the products provides the TCDD toxic

---

[37] A biological receptor is a protein either on or within a cell that has a specific three dimensional structure allowing a substance to tightly bind to it, an oft referred analogy is a lock and key where the receptor is the lock. When the receptor is thus occupied it changes shape and is able to then initiate a number of biological processes. A substance that activates the receptor is called an agonist but one that binds to it but does not cause the required conformational changes required for activation is called an antagonist. The receptor for dioxins is the aryl hydrocarbon receptor, abbreviated to *Ah* receptor. Activation of the *Ah* receptor causes activation of tyrosine kinase (an enzyme pivotal to other enzyme networks and receptor signalling) and also increases the transcription of a variety of genes and regulation of other gene networks. The strength of receptor activation and the ensuing responses is dependent upon the concentration of dioxins in the tissue (i.e. the extent of exposure) and their ability to bind to the receptor; this determines the number of receptors activated. Activation of the *Ah* receptor in animals by relatively large doses of TCDD can result in endocrine and paracrine disturbances and alterations in cell functions including growth and differentiation. Different dioxin congers have different ability to activate the *Ah* receptor. Thus in a dioxin mixture a weak agonist bound to the receptor may block the action of a stronger congener.

[38] Cytochrome P450 consists of a family of enzymes, the one that is most responsive to TCDD is CYP1A1. Levels of this enzyme are often measured by ethoxyresorufin-*O*-deethylase activity (EROD) or CYP1A1 mRNA.

equivalence (TEQ) for the mixture. Thus TEF values for individual congeners in combination with their chemical concentration can be used to calculate the total TCDD toxic equivalent concentration (TEQs) contributed by all dioxin-like congeners using the following equation assuming dose additivity.

$$TEQ = \sum (PCDD_i \times TEF_{WHO\ i}) + \sum (PCDF_i \times TEF_{WHO\ i}) + \sum (PCB_i \times TEF_{WHO\ i})$$

……**Equation 1**

The equation assumes there will be no competition between antagonists [39] , weak agonists and full agonists for binding to and activation of the *Ah* receptor. However the presence of weak agonists or antagonists in a dioxin/PCB mixture will interfere with the molecular action of the high potency (i.e. full agonists) components and the toxicity of the mixture will be less than that of the same mass exposure of the full agonists alone (Schwarz and Appel 2005). Equation 1 however assumes additivity and hence likely overestimates the toxicity of the mixture.

Exposure to dioxins is expressed as the amount (usually in picogram, pg) of dioxin TEQ in the exposure media, e.g. pg TEQ/m^3 if exposure is via air, or pg TEQ/kg if exposure is through food or soil. Dioxins in the Bell Bay effluent are estimated as pg TEQ/L of effluent.

6.3.3 Effluent dioxin concentrations

PCDD/Fs are not expected to be present in the Bell Bay effluent at detectable levels (RPDC 2004 Vol 2 p 5, JP 2005d). This prediction can be made with confidence given that the reaction scheme for the formation of PCDD/F during pulp processing is known (JP 2005d, See Section 4.2).

- **Maximum predicted dioxin in discharged effluent is 0.074 pg TEQ/L.**
- **RPDC limit is 13 pg TEQ/L.**
- **Analytical quantitation limit is approx 10 pg TCDD/L.**

It is widely acknowledged that dioxin levels in pulp mill effluents have been greatly reduced following the introduction of ECF technology (Luthe et al. 1992, FEI 1996, Shariff et al. 1996, US EPA 1998, Bright et al. 2003). When introducing new rules for the substitution of elemental chlorine with chlorine dioxide the US EPA (1997) stated benefits would include at least a 96% reduction of dioxins in effluents, as well as removal of dioxin-related fish consumption

[39] An antagonist is a substance that can bind to the *Ah* receptor but will not elicit any responses, a partial agonist can bind to the receptor and will cause varying degrees of response. On the other hand a full agonist is a substance (e.g. 2,3,7,8-TCDD) that can bind to the receptor and cause maximum response.

advisories in fresh- water ways downstream of pulp and paper mills. A review of analytical results for laboratory and mill bleaching [40] showed 2,3,7,8-TCDD was not found in the raw pulp or effluents[41] when chlorine dioxide replaced elemental chlorine (Shariff et al. 1996). Therefore, replacement of Cl_2 with ClO_2 in the first stage of bleaching results in 'virtual elimination' [42] of 2,3,7,8-TCDD.

Guthe (1998) reviewed Canadian pulp mill effluent releases between 1988 and 1994. The significance of this period is that Canadian pulp mills introduced process improvements in the early 1990s including oxygen delignification and a higher share of chlorine dioxide of the total active chlorine in bleaching. The survey information showed 2,3,7,8-TCDD/TCDF effluent discharges were reduced more than 99% during the period 1988 through 1994.

Although PCDD/Fs are expected to be below analytical detection in the Bell Bay mill effluent (see below) it is theoretically possible some congeners may still be formed at very low concentrations. In order to provide a conservative and cautionary estimate for this risk assessment JP(2005d) assumed the Bell Bay mill processes will result total dioxin TEQ generation of the order of 10% of that measured in the 1990s in Sweden and Canada. This assumption and mass balance apportionment to emission streams yielded a final estimated concentration for dioxins of 0.074 pg TEQ/L for the Bell Bay mill effluent. Analytical detection limits for dioxins in effluent range from 0.3 – 9 pg TCDD/L (Shariff et al. 1996) to up to 10 pg TCDD/L (ALS Environmental 2005).

The estimated maximum concentration of 0.074 pg/L of dioxins in discharged effluent is well below the RPDC guideline for the final effluent (at point of discharge) of 10 pg/L and 30 pg/L for 2,3,7,8-TCDD and 2,3,7,8-TCDF respectively, these limits are equivalent to 13 pg TEQ/L [43]. In addition, based on the US and Canadian observations of 96% and >99% reductions in dioxins, the final effluent TEQ concentration is considered to be a theoretical worst case concentration.

Assuming a minimum dilution of 100 in the immediate vicinity of the ocean outfall, the resulting theoretical dioxin water concentration is 0.00074 pg TEQ/L. For the purposes of this present

---

[40] The data from pulp production mills comprised 163 samples from 9 Canadian, 6 U.S. and 2 Swedish mills where ClO_2 had completely replaced Cl_2 in the first stage of bleaching.
[41] Detection limits for TCDD in bleached pulp were 0.1-0.3 pg/g pulp (ppt) and for effluent 0.3-9 pg/L.
[42] Virtual elimination was defined by Shariff et al. (1996) when a chemical cannot be detected in 100% of samples analysed using the best detection techniques available at the time.
[43] The WHO TEF for 2,3,7,8-TCDD is 1.0 and for 2,3,7,8-TCDF is 0.1 (Van den Berg et al. 1998). Hence for the RPDC limits of 10 pg/L TCDD and 30 pg/L TCDF and following Equation 1:
TEQ = (10 x 1) + (30 x 0.1) = 13 pgTEQ/L.

report it is assumed the various dioxin congeners making up the total TEQ behave in the environment and food chain as if they were 2,3,7,8 –TCDD.

6.3.4 Dioxin toxicity and health guideline

Dioxin Toxicity:

Adverse effects reported in animals following administration of dioxins include immunotoxicity, endometriosis in Rhesus monkeys and developmental and behavioural effects in offspring of treated monkeys. Developmental effects have also been observed in treated rats. The most sensitive effect, i.e. the one occurring at the lowest dioxin exposure, was decreased sperm production and sexual feminisation in male off-spring of exposed rats sensitive to TCDD. TCDD is carcinogenic in several species but does not damage DNA (NHMRC 2002, OCS 2004).

In humans the data, mostly from relatively highly exposed populations, indicate a variety of subtle biochemical responses may occur. These include induction of hepatic enzymes, changes in hormonal levels and reduced glucose tolerance. However, these effects are of unknown clinical significance, and may or may not indicate a toxic response or potential for toxic response (OCS 2004). Of the many health effects evaluated in exposed adult populations, many were transient and not observed when exposure ceased. Human studies have failed to provide compelling evidence for endometriosis. The most consistently observed effect following high dose exposure is chloracne and other skin conditions. There is also some evidence that high paternal exposure to TCDD may be associated with the birth of more girls than boys. From animal cancer experiments with TCDD and occupational studies, plus an understanding of the plausibility of a common mechanism of action for animals and humans the International Agency for Research on Cancer (IARC) has concluded TCDD is carcinogenic to humans (NHMRC 2002, OCS 2004).

There is compelling data that in animals and humans for a common mechanism of action for the biochemical and toxicological effects, i.e. binding to and activation of the *Ah* receptor. Thus results of animal experiments are used to predict the possibility of health effects in humans that have not been observed in human studies. This is the basis for establishing a health guideline for dioxin intake by humans that is regarded by authorities as being safe (see below).

Human sensitivity:

According to WHO (van Leeuwen et al. 2000) data for *Ah* receptor binding affinity and responses directly dependent on *Ah* receptor activation suggest humans may be less susceptible to dioxin than the 'responsive' rodent strains often used in experimental studies. Conversely other biochemical or cellular effects suggest comparable susceptibility, however these latter effects are not associated with adverse health and their clinical significance is largely unknown (OCS 2004). Hays et al. (1997) evaluated the relative susceptibility of humans and rats for cancer using several dose metrics applied to the pivotal rat bioassay (Kociba et al. 1978, Goodman and Sauer 1992) and the US National Institute of Occupational Safety and Health (NIOSH) cohort (Fingerhut et al. 1991). Both these studies had available data on biological dose (blood lipid or adipose tissue TCDD levels) and cancer response. The authors concluded humans are much less sensitive than rats to the carcinogenic effects of TCDD. Others have also suggested that humans are less or no more susceptible to the toxic effects of TCDD and hence exposure of the general population to environmental levels of dioxins should not be of concern (Kimbrough 1990, Leung et al. 1990). More recent comparisons of cytochrome P450 (CYP1A1) induction by TCDD in fresh hepatocytes from human donors, rats and rhesus monkeys indicates that humans are about 10 – 100 times less sensitive than are rats (Silkworth et al. 2005). Since the TEFs for dioxin congeners are in large part based on the responsiveness of the rat to *Ah* –receptor mediated biochemical responses it suggests the TEF allocation for congeners may be over estimating the risk to humans by at least an order of magnitude.

A recent review of the molecular structure, function and dose-response data for the human *Ah*-receptor indicates the human receptor shares key mutations with a mouse strain that compared to sensitive rat strains is relatively unresponsive to TCDD. Binding of TCDD to human *Ah*-receptor is approximately an order of magnitude lower than that observed with *Ah*-receptors of sensitive rodents. The TCDD binding data and molecular structure information support the hypothesis that the human *Ah*-receptor is less functional than the *Ah*-receptor of the more sensitive laboratory animals upon which the TEFs are based (Connor and Aylward 2006).

Health Guideline:

To emphasise the relatively long time frames required for exposure to dioxin like substances before human health effects are

- **Health guideline for dioxin intake = 70 pg TEQ/kg bw/month**

likely to occur the Australian NHMRC/TGA recommend [44] (NHMRC 2002) a 'Tolerable Monthly Intake' (TMI) of 70 pg TEQ/kg bw; this is instead of the more common 'Tolerable Daily Intake' recommended for most other substances. The TMI is a monthly intake of dioxins and dioxin like PCBs that can occur over 40 - 50 years such that the body burden associated with adverse health effects is not achieved. The TMI is based on accumulated body burdens in experimental animals associated with subtle adverse effects and a safety factor of about 10 fold is incorporated for humans. That is the TMI is an intake that can pragmatically be considered safe.

In 1990 the World Health Organization (WHO) established a tolerable daily intake (TDI) for PCDD/PCDF of 10 TEQ/kg bw/d. Re-evaluation of the TDI in 1998 (WHO 1998) resulted in a lowering of the TDI to 1 - 4 pg TEQ/kg bw/d. The maximal tolerable intake is 4 pg TEQ/kg bw/d but the target is reduction of intake to below 1 pg TEQ/kg bw/d. More recently the National Health and Medical Research Council of Australia (NHMRC 2002) have endorsed the Australian Department of Health and Aged Care recommendation for a TDI of 70 pg TEQ/kg bw/month (this is equivalent to 2.3 pg TEQ/kg bw/d) for dioxin like substances, this in turn takes into consideration the revaluations and recommendations of the European Commission (EC-SCF 2001) and JECFA (2001).

Because of the wide variation in elimination of PCDD/PCDF and dioxin-like PCBs between species, the WHO (1998) TDI was established by using the body burden of TEQ in animals rather than the daily intake. In a number of animal studies the sensitive adverse endpoints (hormonal, reproductive and developmental) occurred within a narrow range of body burdens i.e. 10-50 ng TEQ/kg bw. The human daily intake that would result in an equivalent body burden was calculated to be 14-37 pg/kg/d (i.e. this represents a calculated human low observed adverse effect level [LOAEL]). WHO (1998) considered an uncertainty factor of 10 was sufficient to convert this human LOAEL to a TDI, i.e. to a level at which it is anticipated humans will not experience adverse health effects from having that quantity of dioxin like material in their bodies.

The uncertainty factor of 10 was based on the following rationale. Since differences in toxicokinetics (i.e. absorption, metabolism and elimination) are inherently accounted for by using body burden rather than dose it was considered that an uncertainty factor for differences

[44] The TGA recommendation for a tolerable monthly intake of dioxin-like substances for Australians is based on deliberations of the WHO (1998), EC-SCF(2001) and JECFA (2001) and was endorsed by the NHMRC on 24th October 2002. The guideline was established through the NHMRC process to ensure national acceptability. The report upon which the guideline is based underwent public consultation processes and was subject to external review before finalisation. This health reference value for dioxin like substances is the appropriate value for use in risk assessments for Australia.

in toxicokinetics between species was not required. It was noted by the WHO working group that the animal 'no-effect' body burdens were within a factor of 2-3 of the animal 'effect' body burdens, hence a lower uncertainty factor than the traditional factor of 10 for conversion of LOAEL to NOAEL was warranted. In addition, the working group noted that for many of the effects observed experimentally, humans are less sensitive than animals so the full uncertainty factor based on the traditional presumed assumption of higher sensitivity of humans to a chemical was not required. There does however remain some uncertainty regarding human sensitivity to dioxins. This, together with the fact that different components of a dioxin mixture have different half lives in the body, prompted the WHO use an overall composite factor of 10 to account for the uncertainties.

Thus by applying an uncertainty factory of 10 to the range of animal LOAELs of 14-37 pg TCDD equivalents /kg bw/d a TDI, expressed as a range, of 1-4 WHO-TEQ pg/kg bw was established for dioxins and dioxin like compounds. The NHMRC (2002) acknowledge this range in their proposal for a TDI for PCDDs/PCDFs in Australia, and have embraced the WHO methodology for calculating toxicity equivalent factors (Van den Berg et al 1998, WHO 1998).

There have been additional risk assessments of TCDD recently conducted by the European Commission (EC-SCF 2001) and JECFA (2001). These organisations have recommended the tolerable intake of dioxin like compounds be based on long term exposures and have suggested exposure standards that are close to the mid range of the WHO (1998) 1-4 TEQ pg/kg bw/d. These recommendations are 14 TEQ pg/kg bw/week (EC-SCF 2001) and 70 TEQ pg/kg bw/month (JECFA 2001). These convert to 2 and 2.3 TEQ pg/kg bw/d respectively. All organisations have reviewed the same data but have used different processes to derive their recommended exposure standards; it is informative that approximately the same recommendations have been made.

The NHMRC (2002) report a principal finding of the US EPA's evaluation of dioxins on human health (US EPA 2000c) that although dioxins can initiate biochemical and biological events resulting in the potential for a spectrum of cancer and non-cancer responses in animals, *"there is currently no clear indication of increased disease in the general population attributable to dioxin-like compounds"*. This is important because dioxins are ubiquitous in the environment, they are formed during any combustion process (car engines, waste incineration, wood fires, bush fires etc), and hence exposure and accumulation of dioxins in the body cannot be avoided.

6.3.5 The dioxin health risk assessment

6.3.5.1 Thresholds

An important aspect of the risk assessment for dioxins is that the toxic effects of dioxins have a threshold exposure (or dose) below which no adverse health effect will occur. This is the fundamental premise underpinning the establishment of the TMI health guideline (see Section 6.3.4).

Dioxins can cause both non-cancer and cancer effects. It is widely accepted that thresholds exist for the non-cancer effects (ECSCF 2001, JECFA 2001, FSA 2001, NHMRC 2002, OCS 2004). However the US EPA (2003), contrary to other regulatory agencies around the world, has adopted a policy of using a linearised low-dose mathematical model for estimating cancer risks from small dioxin exposures. Such a model assumes no threshold for the cancer effects and implies any dose carries with it a statistical likelihood of cancer for those exposed. This dose response model is usually reserved for risk assessment of substances that cause cancer by direct damage to DNA, i.e. genotoxic substances. Although dioxins are animal multisite carcinogens they are not genotoxic and hence are not initiators of cancer. However they are tumour promoters (OCS 2004). In addition to promoting cancer initiated by genotoxic agents, dioxins also appear to cause cancer in targeted tissues through *Ah* receptor activation and hormonal imbalances, and also perhaps by inducing the metabolism of procarcinogens (Pohl et al. 2002). These biological mechanisms indicate thresholds exist for dioxin induced cancer. The animal and human carcinogenicity data for TCDD has recently been reviewed by Popp et al. (2006) who concluded the level of certainty for a non-linear cancer dose response was substantial because there is concordance of many lines of evidence and consistency of repeated observations pointing to non-linearity.

Thus both mechanistically and experimentally, the weight of evidence robustly supports a non-linear dose response for the carcinogenic effects of dioxins (i.e. the data supports the existence of a threshold for the cancer effects). It is noted the US EPA has been criticised for its policy position for assuming linearity (Kayajanian 2002, Phol et al. 2002, Popp et al. 2006). The World Health Organization, the Australian NHMRC and Office of Chemical Safety (OCS), scientists advising the US EPA and others support the concept of a non-linear dose response for dioxins and cancer (SAB 1995, van Leeuwen et al. 2000, ECSCF 2001, JECFA 2001, FSA 2001, NHMRC 2002, OCS 2004, Schwarz and Appel 2005).

This risk assessment does not follow the US EPA approach of calculating cancer risks from dioxin exposure. Consistent with other epigenetic carcinogens and the deliberations of most

international authorities, we consider a practical threshold exists for the cancer effects of dioxins and that the TMI established by the WHO and Australian authorities provides protection against cancer as well as non-cancer health effects (see Section 6.3.3). In fact, the reproductive and hormonal effects in experimental studies seem to occur at lower body burdens than required for cancer (Pohl et al. 2002, OCS 2004).

6.3.5.2 General principle for the dioxin HHRA

The general principle for assessing health impacts of dioxins in discharged mill effluent is to estimate an incremental dioxin monthly intake from eating fish caught in the area and add it to an estimate of background intake of all dioxin like substances. Risk is characterised by comparing this sum with the monthly intake determined to be safe by the Australian Government Department of Health and Ageing (NHMRC 2002) (i.e. the tolerable monthly intake, or TMI). If the total monthly intake of dioxins is less than the TMI then the risk of adverse health effects from dioxins in the Bell Bay mill effluent is very low.

The risk assessment for dioxins is schematically outlined in Figure 6.2 and in its simplest mathematical form is expressed as follows:

$$M_{I\ TOTAL} = M_{I\ FISH} + M_{I\ BKGD} < TMI \quad \text{.....Equation 2}$$

Where:

$M_{I\ FISH}$ = Monthly Intake of dioxins (incremental) from ingestion of fish that have potentially taken up dioxins from the Bell Bay mill effluent. This requires knowledge of the concentration in fish (see Appendix 5 for details) and the amount of fish consumed (see Section Appendix 5 for details).

$M_{I\ BKGD}$ = Monthly background Intake of all dioxin like compounds including PCBs (see Section 6.3.5.4 & Appendix 5 for details).

TMI = Tolerable Monthly Intake (see Section 6.3.4).

All terms have the units of pg dioxin TEQ/kg body weight/month (abbreviated to pg TEQ/kg/mth).

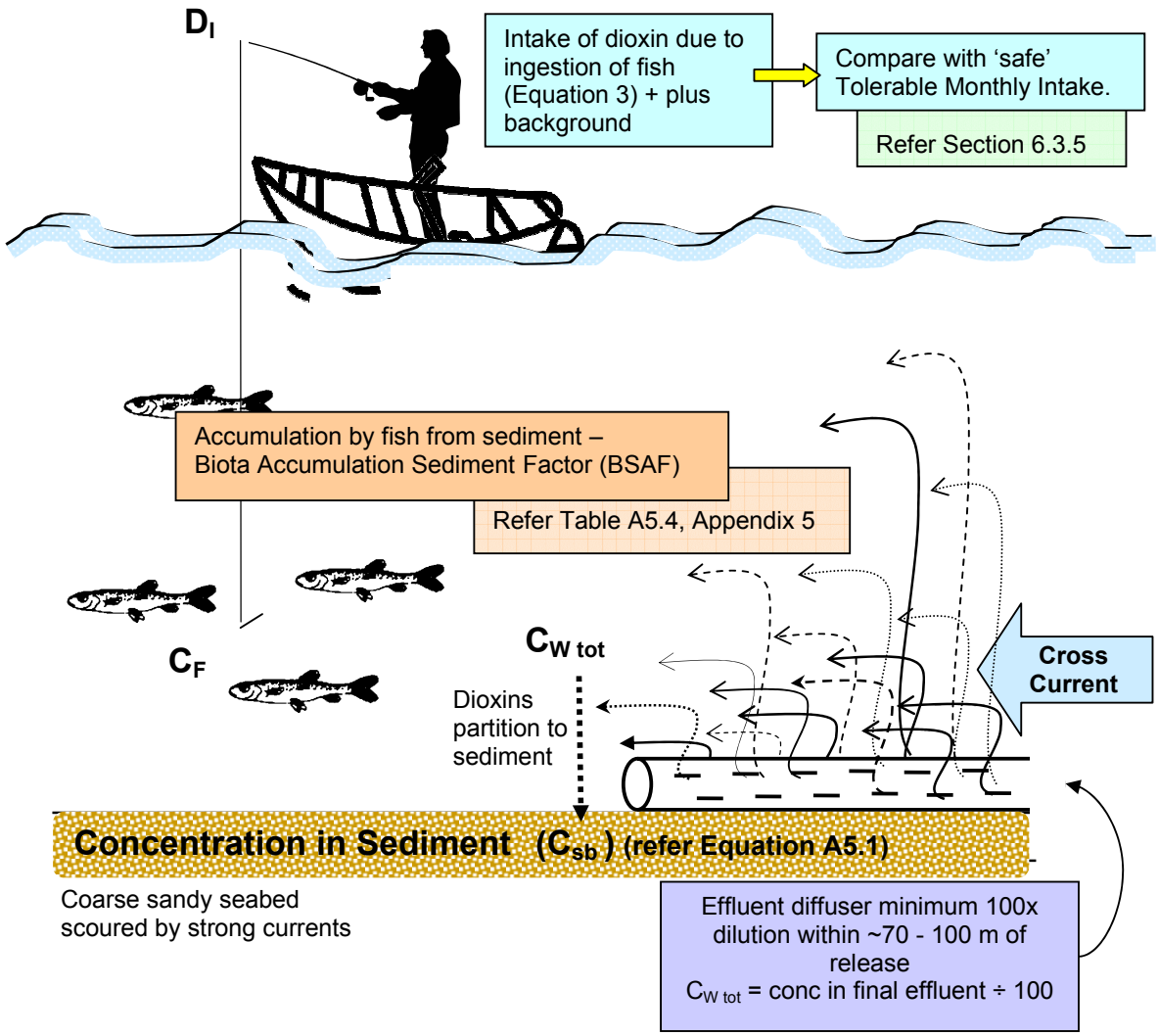


Figure 6.2: Schematic overview of dioxin exposure pathway for an angler eating fish caught at the ocean outfall.
Equations A5.1 and A5.2 plus descriptions thereof in Appendix 5.

6.3.5.3 Key assumptions in the exposure assessment

1. Dioxins have long biological half lives, in humans approximately 7 years (NHMRC 2002), and it takes many years[45] for steady state body burdens to be achieved. This is essentially true also for food production animals, though the life spans of most food production animals is much less than the time required to achieve steady state body burdens. Nevertheless it is assumed in the calculations herein that steady state has been achieved between dioxin concentrations at the initial dilution zone, the amount accumulated by marine organisms in the effluent outfall area (i.e. fish) and the amount consumed by humans.

Key assumptions:

- **Steady state between dioxin in the outfall environment and fish.**
- **Majority of dioxins in effluent partition into sediment near the outfall.**
- **Fish spend majority of their time at the outfall.**
- **Fish are consumed at maximum rate.**
- **Fish from other sources not eaten.**
- **Maximum background intake of dioxins.**

ALL OVER-ESTIMATE INTAKE

2. The assumption of steady state between environmental concentrations of dioxins near the outfall and fish also has embedded within it the assumption that fish will spend the majority of their time at the edge of the DZ100. This assumption is clearly unrealistic and highly conservative.

3. Dioxins have very low water solubility, and partition into sediment which acts as the environmental sink (US EPA 2000). It is assumed all dioxins in the discharged effluent will partition into sediment near the outfall (see Appendix 5).

4. People will catch fish from the outfall area and consume them at the maximum consumption rate noted for Australians (see Appendix 5).

5. Fish from sources other than the ocean outfall are not eaten.
6. The person consuming the fish is also an individual who has the highest background body burden of dioxins (see Appendix 5).

[45] The rule of thumb for quickly estimating time to steady state body burden is that it takes 3 – 5 half lives, hence on the assumption of a 7 year half life time to steady state is approximately 20- 35 years.

6.3.5.4 Background dioxin intake for Australians

A pivotal aspect of the risk assessment for dioxin-like substances is for background intakes to be included. The Australian Government has recently published estimates for background intake of dioxin-like substances for Australians (OCS 2004). The estimated total background intakes from all sources of exposure for dioxins and furans for Australian adults are summarised in Table 6.1. Intake from food accounts for 95-99% of the total intakes, and of this approximately 40% is from seafood (OCS 2004). This risk assessment conservatively uses the upper bound total intake estimates for Australians are used.

Table 6.1: Estimated total intakes of dioxins and dioxin-like compounds for Australians[a]

Total Intake (pg TEQ/kg bw/month)		
Population sector	Lower bound	Upper bound
Adults	3.89	**15.79** [b]
Young children	7.22	**37.74** [b]

[a] Data taken from OCS 2004 (Table 3-32 therein).

[b] The values are upper bound intake estimates of the 95th percentile total intake estimates from all media (air, food, soil and food). The term upper bound relates to the fact that where a dioxin or PCB congener was not detected the intake was calculated assuming it was present at a level equal to the analytical non-detect level (OCS 2004). These upper bound estimates therefore represent a worst case assumption for background intakes of dioxin-like substances and have been used in the risk calculations below.

6.3.5.5 Incremental dioxin fish concentrations

- **Incremental dioxin increase in fish = 0.18 pg TEQ/kg fish.**

Background concentrations of dioxins in fish (flathead, wrasse and perch) at the proposed ocean outfall site were less than quantitation limits of 0.1 – 3.4 pg/g tissue wet weight for congeners grouped according to degree of chlorination [46] (Aquenal 2005). The calculated incremental increase in dioxins from the assumed maximum effluent dioxin concentration is 0.18 pg TEQ/kg fish (i.e. 0.00018 pg TEQ/g), substantially less than the analytical quantitation limits. The calculations and rationale leading to the incremental dioxin estimate for fish are provided in Appendix 5.

[46] The dioxin data contained in Aquenal (2005) do not allow calculation of background TEQ concentrations in fish.

6.3.5.6 Incremental human dioxin intake from fish

Equation 2 contains a term ($M_{I\ FISH}$) for the incremental monthly increase in dioxin intake due to ingestion of fish caught at the effluent outfall. This can be estimated using the following Equation 3.

$$\text{Monthly incremental Intake} = \frac{\text{Dioxin concentration in fish x monthly consumption of fish}}{\text{body weight}}$$

$$M_{I\ FISH}\ (\text{pg/kg bw/mth}) = \frac{C_F\ (\text{pg/kg fish}) \times CR\ (\text{kg/month})}{BW\ (\text{kg})} \quad \text{.....Equation 3}$$

Where:

$M_{I\ FISH}$ = Monthly intake of dioxins from fish (pg TEQ/kg bw/mth).
C_F = Concentration of dioxin in fish = 0.18pg TEQ/kg fish (see Appendix 5).
CR = Consumption rate of fish (kg/month). Sourced from the Australian Bureau of Statistics, summarised in Table 6.2 and detailed in Appendix 5.
BW = Body weight: Adult 70kg, child 15kg (enHealth 2003).

Within the general population, fish consumption is variable. Consequently the risk assessment has determined potential incremental dioxin intakes ($M_{I\ FISH}$) for the average, upper 95th percentile and maximum rates of monthly fish consumption for adults and children as recorded by the Australian Bureau of Statistics (ABS 1999, Appendix 5). Table 6.2 summarises the statistics for fish consumption and the corresponding calculated incremental monthly dioxin intake assuming an incremental increase in fish dioxin concentration of 0.18 pg TEQ/kg fish (see Appendix 5 for details). Interestingly there is no difference between age groups when the fish consumption is normalised for body weight (maximum consumption for adults and children is 0.023 kg fish/kg bw/mth). Consequently, the incremental dioxin intake is the same across age groups.

An example calculation for the incremental dioxin intake using the maximum fish consumption for an adult is presented below.

$$M_{I\ FISH} = \frac{C_F\ (\text{pg/kg}) \times CR\ (\text{kg/mth})}{BW\ (\text{kg})} \quad \text{.....Equation 3}$$

$$= \frac{0.18 \times 1.6}{70}$$

$$= 0.004\ \text{pg TEQ/kg bw/mth}$$

Table 6.2: Human incremental dioxin monthly intake from fish.

Population Group		Fish Consumption [a] (CR)(kg/person/mth)			Body Weight (kg)	Dioxin Intake pg TEQ/kg bw/month [d]		
		Avg	95%	Max		Avg	95%	Max
Adult [b] (Tasmanian)		0.94	1.4	**1.6**	70	0.002	0.004	**0.004**
Child [c] (Australian)	Male	0.21	0.32	**0.36**	15.5	0.002	0.004	**0.004**
	Female	0.20	0.30	**0.37**	15.3	0.002	0.004	**0.004**

[a] Fish consumption data was sourced from the Australian Bureau of Statistics (ABS 1999, Appendix 5, Table A5.3).
[b] The ABS data does not separate Tasmanian adult fish consumption by gender, values are for Tasmanians above 19 years.
[c] Child fish consumption values were not found for Tasmanians, data for the general Australian population (2 to 3 year old).
[d] The dioxin concentration in fish caught at the site is estimated to be 0.18 pg TEQ/kg wet weight (see Appendix 5 for calculation details for this value). An example calculation is provided above.

6.3.5.7 Total human dioxin intake

The total monthly dioxin intake from all exposure routes (ingestion of fish caught at the outfall plus background) is presented in Table 6.3. The total intake is dominated by background estimates. The theoretical incremental increase in human intake of dioxins due to the effluent discharged to sea is less than 0.05% of background intakes.

Table 6.3: Theoretical human total intake of dioxin-like substances

Population Group	Incremental dioxin intake [a] (pg TEQ/kg bw/mth)			Background Intake[b] (pg TEQ/kg bw/mth)		Total Intake [c] (pg TEQ/kg bw/mth)	
	Avg	95%	Max	Lower bound (LB)	Upper bound (UB)	Avg + LB	Max + UB
Adult (Tasmanian)	0.002	0.004	0.004	3.89	15.79	**3.89**	**15.79**
Child (Australian)	0.002	0.004	0.004	7.22	37.74	**7.22**	**37.74**

[a] The incremental intake is from Table 6.2. Note the number of significant figures in the values above do not imply a certain level of accuracy in the estimations; rather they have been included only to allow for transparency in the mathematical accounting.
[b] Background intakes are from Table 6.1 and originate from OCS (2004, Tables 3-32 and 3-33 therein). It should be noted the background intakes incorporate dioxin and dioxin-like compound background concentrations that may be in fish due to accumulation of dioxins from sources other than the discharged mill effluent, also included in the background intake are PCBs.
[c] Total intakes are calculated by simple addition of incremental intake + background intake. Values in the table are for the addition of average incremental dioxin intake plus the lower bound estimate of background intake, and for the maximum incremental intake added to the upper bound background intake. The latter sum is a reasonable maximum estimate of total dioxin intake.

6.3.5.8 Risk characterisation and uncertainty

- ✓ **The possibly of low dioxin concentrations in discharged mill effluent does <u>not</u> pose a health risk to people consuming fish caught in the vicinity of the outfall.**
- ✓ **There is a high level of confidence in the above statement.**

The lifetime intake of dioxin-like substances by humans determined by health authorities to be without adverse health effects (i.e. the safe intake level) is 70 pg TEQ/kg bw/month (Section 6.3.4). This dioxin risk assessment has made a number of conservative assumptions regarding possible exposure of fish to effluent, dioxin uptake by fish, and consumption of fish by humans that collectively bias the assessment towards markedly over estimating human exposure to dioxins in the discharged effluent. Nevertheless the estimated total child intake of dioxins, including background, arising from a theoretical maximum dioxin TEQ concentration in the discharged effluent is 54% of the health guideline [47]. Most of the calculated exposure (>99%) is associated with background intakes. Other sectors of the population (adults and lower fish eating groups) have much lower monthly intakes of dioxins. It is concluded the anticipated dioxin concentrations in discharged mill effluent do not pose a health risk to people consuming fish caught in the vicinity of the outfall.

It should be noted the Bell Bay mill is to be designed to virtually eliminate dioxins in effluent, hence the final effluent dioxin concentration is expected to be well below analytical quantitation limits. Indeed, the estimated dioxin TEQ concentration in discharged effluent (0.074 pg TEQ/L) used in the risk assessment is also significantly below analytical quantitation limits (~10 pg/L). This presents an issue in verifying the outcomes of the risk assessment since the assumed very low level of dioxin in the discharged effluent cannot be measured. Similarly, the very small theoretical incremental increase in dioxin concentrations in fish cannot be measured. It is therefore very difficult to corroborate the risk assessment experimentally or with field observations once the mill is in operation. The best that is likely to be achieved is that dioxin effluent concentrations are shown to be below analytical detection limits (how far below?) and there will be no demonstrable change in background fish dioxin concentrations after the outfall becomes functional.

[47] The maximal total intake is ~ 38 pg TEQ/kg/mth for a child (Table 6.3), the lifetime health guideline is 70 pg TEQ/kg/mth. [38/70 x 100 = 54%]

To address these issues, a sensitivity analysis has been performed. The risk assessment calculations have been performed assuming the effluent dioxin concentration could be *at* the analytical quantitation limit [48] of 10 pg TCDD/L , i.e. ~135 times greater than the likely maximum concentration advised by Jaakko Pöyry (JP 2005d). Furthermore, the calculations have also been executed using the suspect high fish intake of 100g/d for recreational fishermen noted in enHealth (2003) (see Table A5.1 of Appendix 5). This is approximately twice the maximum fish consumption used in the risk assessment. The results of this sensitivity analysis are summarised in Table 6.4. It can be seen that a further increase of approximately 250 fold [49] in incremental dioxin intake has very little impact on the maximal total dioxin intake which remains well below the dioxin health guideline.

The estimation of the amount of dioxin that may be accumulated by fish from sediment is sensitive to the organic carbon (OC) content of the sediment (Equation A5.3 of Appendix 5). OC data for the ocean outfall site was not determined by Aquenal (2005). Consequently the mean value for marine sediments around Australia was applied to the risk assessment. Because the outfall site is described as being coarse sand there is a possibility the OC may be lower than that used in the risk assessment; lower sediment OC means greater transfer of dioxin from sediment into the lipid of fish. The influence of OC on the risk calculations have been addressed by completing the calculations for the equations in Text Box A5.1 (Appendix 5) with the lowest sediment OC measured by Muëller (2004), i.e. 0.048% instead of the mean 0.32%. The result of this was to increase the theoretical maximum total intake of dioxin of an adult person at the top end of the spectrum for fish consumption from 15.79 pg TEQ/kg/mth to 15.81 pg TEQ/kg/mth; i.e. by only approximately 0.2% (Table 6.4).

Consequent to the above considerations there is a high degree of confidence in the conclusion of no dioxin related adverse health effects from eating fish caught in the outfall area.

[48] In risk assessments of environmental exposures to dioxins where measurements cannot quantitate the concentration of congeners it is usual to conduct the assessment assuming the congener may be present at 50% of the analytical quantitation limit. However since quantitation limits differ between congeners and vary with media matrixes the sensitivity analysis in Table 6.4 has been done using 100% of the quantitation limit. This removes some of the uncertainty regarding what the quantitation limit may be (see Section 6.3.3).

[49] The most likely maximum incremental intake of dioxin from fish is 0.004 pgTEQ/kg/mth. With the assumptions of increased effluent concentration to 10 pg TEQ/L and increased fish consumption to 100 g/adult/day the incremental dioxin intake rises from 0.004 pg TEQ/kg/mth to ~1 pg/kg/mth, an increase of 250 times.

Table 6.4: Sensitivity analysis for adult dioxin TEQ intakes.

Risk assessment assumptions [a, i]	Max incremental dioxin intake from fish (pg /kg bw /mth)	Upper bound background intake (pg/kg bw /mth)	Max total intake [b] (pg/kg bw /mth)
As per main text: EC: 0.074 pg TEQ/L [c] FC: 54 g/person/d [d] OC: 0.32% [g]	**0.004** (from Table 6.2) [0.03% background]	15.79 (from Table 6.1)	**15.79** (from Table 6.3) [23% TMI]
EC: 0.074 pg TEQ/L [c] FC: 54 g/person/d [d] OC: 0.048% [h]	**0.028** [0.2% background]	15.79	**15.82** [23% TMI]
EC: 10 pg TEQ/L [e] FC: 54 g/person/d OC: 0.32%	**0.57** [3.6% background]	15.79	**16.36** [23% TMI]
EC: 10 pg TEQ/L FC: 100 g/person/d [f] OC: 0.32%	**1** [6.3% background]	15.79	**16.79** [24% TMI]

[a] EC = Effluent Concentration.
FC = Fish Consumption, average daily intake throughout life.
OC = Organic Carbon.

[b] Values in this column should be compared with the health guideline of 70 pg TEQ/kg bw/mth. Please note that the number of significant figures in these values does not imply a level of accuracy in the calculations, the significant figures are driven by the background concentrations reported in OCS (2004).

[c] Estimated by Jaakko Pöyry (JP 2005d) to be maximum likely dioxin concentration in effluent.

[d] Maximum consumption of fish by a Tasmanian adult (ABS 1999, Table A5.1 Appendix 5).

[e] Assumed analytical quantitation limit of dioxins in effluent. Note the RPDC limits for TCDD & TCDF in mill effluent are 13 pg TEQ/L (see Footnote 23).

[f] From a 1977 survey reported in en Health (2003) which enHealth regard as an over estimation (see Table A5.1 Appendix 5).

[g] Mean of the 12 marine sediment samples collected from around Australia (Muëller 2004).

[h] The lowest organic carbon content of Australian marine sediments measured by (Muëller 2004); range 0.048% to 1.4%, median 0.11%, mean 0.32%. A low OC results in greater transfer of dioxin from sediment into fish because there is more unbound dioxin available.

[i] Note it is highly unlikely the dioxin effluent concentration will be at the quantitation limit and a person will consume 100g fish each day for a protracted period or source the fish solely from the outfall area - hence the low sediment organic carbon content has not been joined with these high end values in the sensitivity analysis. In addition the sensitivity analysis has not been performed for a child because dioxin effects are related to body burden which takes ~20 – 35 years to achieve.

6.4 Discussion and conclusions

Bioaccumulative metals

A number of metals are likely to be present in discharged Bell Bay mill effluent because they are natural constituents of wood. Of these, cadmium, mercury and selenium are thought by ANZECC (2000) to be potentially bioaccumulative in some circumstances and hence have been subject to detailed consideration in this risk assessment. A qualitative evaluation of the literature

pertaining to bioaccumulation of cadmium and selenium showed bioaccumulation of these metals by fish at the ocean outfall is not a concern.

Estimates of cadmium, mercury and selenium concentrations in effluent at the edge of the DZ100, which is assumed to be a relatively short distance from the diffuser, are less than background concentrations in marine waters, and for cadmium and selenium less than baseline concentrations measured at the site. It therefore follows that marine organisms will not take up these substances to any greater extent than what currently occurs at background concentrations. If there is no incremental increase in metal body burden of edible species due to the effluent discharge, then there is no incremental increase in health risk to humans consuming those species.

It is therefore concluded the low levels of cadmium, mercury and selenium in effluent do not pose a health risk to people consuming fish caught in the region of the outfall.

Dioxins

Although the Bell Bay mill processes and effluent treatments are designed to virtually eliminate dioxin formation and its presence in discharged effluent, there is a residual possibility of low amounts of dioxins being in the effluent. However the concentration of dioxins is predicted to be substantially below the RPDC guidelines and analytical quantitation limits for effluent. The quantitative risk assessment conducted herein showed, even at maximum likely concentrations in effluent, that the theoretical incremental uptake of dioxins by fish was tiny. Consequently the total intake of dioxins by people eating fish caught in the region is much less than the intake considered by health authorities to be safe, even if large amounts of fish were consumed over a long period of time.

It is concluded with a high level of confidence that the possibly of low dioxin concentrations in discharged mill effluent does not pose a health risk to people consuming fish caught in the vicinity of the outfall.

7. HHRA for recreational water use

- **Recreational water use in the vicinity of the outfall is unlikely.**
- **The estimated water concentrations of effluent constituents do not exceed guidelines where they exist.**

Both the WHO (2003) and ANZECC (2000) have published water quality guideline values for the protection of public health for recreational water use. In the main, these guidelines are intended for coastal and inland waters used for activities -

- in which the user comes into frequent direct contact with water, either as part of the activity or accidentally; for example, swimming, diving or surfing (primary contact);
- that generally have less-frequent body contact with the water; for example, boating or fishing (secondary contact).

The guidelines for chemical contaminants in water are based on drinking water guidelines using the reasoning that varying extents of accidental ingestion may occur during the recreational activity (WHO 2003).

Recreational activities in the vicinity of the proposed effluent outfall site were not observed during any of the survey work done to-date. Recreational dive charters operate around Hebe Reef (north of George Town) and more than 5 km west of the diffuser site. Seal watching tours operate from George Town and take tourists out to Tenth Island to observe the seal colony on the island. The nearest dive charter operator to the effluent outfall is based at Low Heads however the likelihood of diving at the outfall is considered very remote because the site is barren, there is low species abundance, the current strong and seas inhospitable.

Although not observed, probably because of the paucity of fish, it is possible that boat fishing may occur in the vicinity of the outfall. Nevertheless the prevailing seas and weather suggest it would be unlikely. However given the possibility of the occurrence of recreational boating and fishing the effluent, following initial dilution, was assessed against health based screening criteria for recreational water use.

Table A1.3 contains the list of chemicals of interest determined in Section 4.4. The comparison of contaminant concentration at the boundary of the initial dilution zone with recreational water guideline concentrations is contained in Table A6.1 of Appendix 6. None of the concentrations

of these chemicals following initial dilution exceeded their respective recreational water guideline value contained in WHO (2003) and ANZECC (2000).

8. Tainting of seafood

Tainting is defined as any change in flavour or odour that is unusual when compared to other products (Poels et al. 1988). It is normally associated with off-flavour rather than improvement to the flavour of seafood. Guidelines for chemicals that cause tainting are available from ANZECC (2000), US EPA (2005b) and various provinces of Canada, e.g. British Colombia ((BC MoE 1998). The guidelines [50] provide lists of compounds that have been identified as causing tainting in seafood and the threshold at which tainting can occur, but do not describe the basis for deriving thresholds.

It is unlikely commercial seafood will be caught within the initial dilution zone because of its relatively small size, however recreational fishing may occur. The estimated concentration of each chemical of interest at the edge of the DZ100 was assessed against the available tainting guideline values. None of the chemicals of interest following initial dilution exceed any of their respective tainting guidelines (see Table A7.1 in Appendix 7). However only 18 of 163 compounds of interest have a tainting guideline.

In order to further assess the potential for tainting a brief contextual review of literature associating pulp mill effluent with fish tainting was conducted (see Appendix 7). Only three articles were obtained that had been published in the last ten years. However in this period there have been many advances in pulp and paper mill technology that have markedly changed both the nature and quantity of effluent released to the environment. Furthermore there are substantial differences between mills of the same era in relation to technological processes, effluent constituents and environmental impacts. Consequently use of historic instances of tainting to predict potential tainting by effluent discharged from the more technologically advanced Bell Bay mill requires careful consideration.

[50] Food or water tainting guidelines could not be located from other regulatory agencies (Environment Canada, Europa Environment, WHO, UK Department of Environment, Finnish Environment Industry and Danish Environmental Protection Agency). OSPAR provided a list of chemicals that are suspected to taint and a list of chemicals that have been tested and found not to cause tainting. Compounds listed as not tainting are dimethylbenzenes, nitrophenols, dinitrophenols, β-pinene, phenol, toluene and xylene (OSPAR, 2002). Some of these compounds have guidelines according to ANZECC (2000) and therefore are expected to taint. Consequently the OSPAR list was rejected.

Although it is difficult to draw comparisons between the Bell Bay final effluent and reports of historical tainting in the literature, some general statements from the literature review can be made:

- The literature is replete with examples of fish tainting associated with pulp mill effluents.
- Most papers reporting pulp mill effluents as the cause of fish tainting were published pre-1993. Approximately 25 papers were identified with publication dates prior to 1993[51] and only 5 papers post-1993 (Craig 1993, Mosse and Kowarsky 1995, Redenbach et al. 1997, Lowell et al 2003, Lowell et al 2005). Effluent treatment processes particularly biological treatment processes have been implemented and/or improved to meet regulations on effluent discharge implemented around 1991-1994 in the Canada, Sweden, and the US and it likely that these improvements in effluent treatment have contributed to the reduction in reporting of tainting downstream to pulp mills. Environment Canada has recently acknowledged that improvements in effluent treatment processes have resulted in either elimination or substantially reduction of the tainting ability of individual pulp mill effluents (Lowell et al. 2005).
- Very few studies on the pulp mill effluents and tainting were found since the introduction of ECF plants in the early 1990s. This is suggestive that the historical problem of fish tainting is not characteristic of modern mill effluents. However the environmental effects monitoring programme of pulp mills conducted by Environment Canada during 2000 – 2004 found the effluent from one mill was tainting fish although the intensity of taint was less than observed in the previous cycle of monitoring conducted in the late 1990s (Lowell et al. 2003, 2005). The relevance of this information for the Bell Bay mill is difficult to gauge as specific information on the mill and its location are not provided.
- Almost all identified reports have occurred in freshwater environments. The only study identified that investigated tainting from a pulp and paper source discharging into a marine environment was for the Australian Maryvale mill and did not report tainting of fish (flathead) (Mosse and Kowarsky 1995). The lack of tainting data for pulp mill effluent discharges to sea may reflect there are relatively few pulp mills discharging to the marine environments. Most mills in the northern hemisphere discharge to freshwater environments. However, unlike a river or lake mixing system where the potential for dispersion of contaminants is limited the proposed ocean effluent discharge for Bell Bay mill effluent will result in relatively rapid mixing within the initial dilution zone and

[51] Swabey 1965, Wells 1967, Shumway and Chadwick 1971, Shumway and Palensky 1973, Cook et al 1973, Langford 1974, Langer and Nassichuk 1975, Bell and Kallman 1976, Whittle and Flood 1977, Liem et al 1977, Brouzes et al 1978, Rogers 1978, Findlay and Naish 1979, Gordon et al 1980, Weinbauer et al 1980, Kuusi and Suihko 1981, Miettinen et al 1982, Paasivirta 1982, Kovacs 1982, Paasivirta 1983, Persson 1988, Jardine 1992, Lindsay and Heil 1992, Paasivirta 1992.

subsequent dispersal at very low concentrations over a wide area, decreasing the likelihood of tainting.

- A variety of substances in pulp mill effluent have historically been investigated as possible causes of tainting. It may be that no one substance or class of substances are solely responsible. Nevertheless the literature suggests that in the past alky phenols and chlorinated anisoles and veratoles were implicated, with the latter being the most likely candidates for causing tainting.

Conclusions

Historical accounts of fish tainting should not be used to judge the potential of Bell Bay mill effluent to cause tainting. The available information indicates commercial and recreational fishing does not occur in the vicinity of the proposed ocean outfall. In addition, there is a paucity of fish in the area. Rapid dilution of the effluent is anticipated to occur so fish further afield will not be affected.

Although it cannot be stated with certainty, it appears from the literature review in Appendix 7 that poly-chlorination of natural phenols and resins may be primarily responsible for historic tainting of fish by pulp mill effluent discharged into freshwater systems. Overall it is considered modern elemental chlorine free bleaching and the modern effluent treatment technology to be installed at the Bell Bay pulp mill will virtually eliminate substances thought to be associated with fish tainting.

In summary, there is little potential for the public to be exposed to tainted fish. However there is sufficient uncertainty to support the RPDC requirement of taint testing of effluent, at least in the first few years of mill operation.

9. Consequences on regional aquaculture and commercial fishing

According to Aquenal (2005) there are no aquaculture activities in the vicinity of the outfall site. The nearest marine farm is located at Long Reach in the Tamar Estuary, where salmon and rainbow trout are harvested. Toxikos is also aware that other marine farms exist several kilometres along the coast to the east of Five Mile Bluff. There is also very little commercial fishing in the central Bass Strait area. The main commercial fishing methods identified for central Bass Strait are gillnetting, scallop dredging and lobster trapping as well as some squid jigging (Butler et al 2002). Southern rock lobster (*Jasus edwardsii*), greenlip abalone (*Haliotis laevigata*) and blacklip abalone (*Haliotis rubra*) were identified at Five Mile Bluff and/or Stony Head by Edgar (1998) but not in quantities. The primary species fished commercially in Bass Strait water by gillnetting are ling, spotted warehou, gummy shark, school shark, elephant shark, saw shark, and whiskery shark (Larscombe et al. 2002).

The potential for bioaccumulation in fish has been assessed in Sections 5 and 6 of this report. There are only relatively few substances in the effluent that have the potential to bioaccumulate, for most of these open ocean background levels are achieved within a very small distance (approximately 100 metres) from the diffuser site. In addition, the risk assessment for dioxins showed essentially zero incremental human risk from consuming fish caught in the vicinity of the outfall. It therefore follows that these substances are very unlikely to impact on distant aquaculture and commercial fishing. Consequently a human health risk assessment for aquaculture and commercial fishing activities is not warranted.

Similarly, because it is considered there is little likelihood of fish being caught near the ocean outfall being tainted due to the mill effluent, it is very remote that distant aquaculture will be affected.

10. General uncertainty analysis

The risk assessment herein involves a number of steps[52] each of which incorporates the use of assumptions and simplifications to manage uncertainty or lack of knowledge about the correct parameter value. Without such assumptions and simplifications it would not be possible to evaluate the potential for health effects. Although uncertainties in the risk assessment may influence its accuracy, reliability and interpretation, the assumptions used to cope with the uncertainties err on the side of safety and therefore bias the evaluation to over estimate of health risk. This is appropriate for a prospective assessment for possible impacts on public health. It must be realised however the conservatism entrenched within any single parameter value is at least additive, most times multiplicative, with other conservatisms. Consequently the cumulative conservatism integrated into the assessment can be very large. This is especially so when gross, unrealistic default parameters are used *in lieu* of measured data.

In the main body of the risk assessment, uncertainties associated with specific assumptions are discussed at that time. This section (Table 10.1) contains a general qualitative discussion of the major uncertainties and their potential influence on the health risk assessment. The 'big picture' uncertainties fall into the following major categories.

- Those associated with effluent characterisation (i.e. identification and concentration of effluent constituents).
- Assumptions in the screening process.
- Contaminant specific uncertainties.
- Characterisation related uncertainties.

[52] E.g. identifying final effluent constituents, concentration estimation of effluent constituents, screening for bioaccumulation potential, and ingestion of fish caught within the vicinity of the outfall.

Table 10.1: Uncertainties and potential affect on health risk assessment outcome.

Uncertainty/Assumption	Comment	Effect on Risk Assessment
Identification of constituents likely to be present in the final effluent. In the absence of actual data there is uncertainty regarding what substances will be in the effluent. Some effluent components may not have been identified.	Candidate substances and associated chemical classes considered in the risk assessment included those historically identified in pulp mill effluent around the world. Constituents in effluent from mills processing different types of wood (including eucalypt and pine) were considered. The candidate chemicals were assessed for likelihood of being in effluent according to engineering design & production processes at the Bell Bay mill. A list of 'chemicals of interest' was thus produced. Constituents regarded as readily degradable were included in the assessment even though many of these are likely to be completely removed during biological treatment.	It is possible some individual effluent constituents have not been identified. However the likelihood they will have characteristics significantly different to those subject to the screening process is low as the major source of compounds in effluent is the wood itself. The major classes of compounds and representative compounds from each class extracted from wood are included. Hence an individual chemical within the classes, but perhaps not named in the 'chemicals of interest' will have minimal impact on the health risk assessment because all such compounds did not survive the screen for bioaccumulation.
Estimation of constituent concentrations. The mill has not yet been built hence there is uncertainty regarding the concentration of effluent constituents. The concentration of some chemicals in the effluent may not have been appropriately estimated. Process variability and hence effluent variability is not known at this time. Estimation of effluent concentration in the receiving waters may be wrong.	Concentrations for individual chemicals or classes of chemicals have been estimated by Jaakko Pöyry Oy based on mass balances given knowledge of the kraft process and the BB mill design. This information was supplemented by Toxikos with information from the literature. Estimated concentrations are based on mass balances at the design production capacity of the mill. The mill will not always operate at this capacity. The minimum dilution design criterion for the outfall diffuser was assumed. However dilution is expected to be greater than this for most of the time. For the dioxin HRA fish intake calculations were also done assuming effluent TEQ concentration was at the analytical detection limit, i.e. 135 times the anticipated concentration.	Estimated concentrations are not definitive; however Toxikos is of the opinion that the concentration of any individual constituent in the final effluent and receiving water is likely to be over-estimated rather than under-estimated. The incremental TEQ intake increased from 0.004 to 0.57 pg TEQ/kg/mth (3.6% of maximum background) but total intake was only 23.1% of the TMI (up from 22.6%, Section 6.3.5.8). Thus, in relation to total TEQ intake, the HRA is not especially sensitive to assumed TEQ concentration in effluent.

Uncertainty/Assumption	Comment	Effect on Risk Assessment
Screening for bioaccumulation potential. Not all chemicals of potential concern have been identified by the screening process. Lipid solubility may be under estimated and thus chemicals were falsely eliminated for bioaccumulation potential.	The bioaccumulation potential of many low molecular weight organic chemicals is predicted based on their ability to partition from water or food to fat (i.e. are fat soluble). Only a small proportion of the chemicals of interest had high lipid solubility. Of these only a few are known to bioaccumulate because most can be efficiently metabolised and excreted by fish. The screening process relies on estimates of log K_{ow} & BCF most of which are calculated values predicted from a software program. Algorithms for calculated BCF values are based on bioconcentration data from warm water fish (preferences fat head minnow>goldfish>sunfish>carp >> marine species), with data for fathead minnow being most prevalent (Meylan et al. 1999). Warm water fish have higher lipid content than marine species. Since the ocean outfall environment is oligotrophic fish in the area will have low lipid content because food of high calorific value is scarce. Consequently for any given compound the calculated log K_{OW} and BCF from US EPA algorithms will over estimate bioaccumulation potential for fish in the outfall area. For dioxins the highest or highest mean value for accumulation by fish was selected.	The screening process is unlikely to underestimate the potential for bioaccumulation. Thus it is unlikely any 'chemical of interest' was falsely eliminated from being potentially included in a detailed quantitative assessment.

Uncertainty/Assumption	Comment	Effect on Risk Assessment
Background concentrations	For the majority of natural extractives present in the final effluent background exposures are unknown but are likely to be tiny.	This has virtually no impact on the health risk assessment as the natural extractives are readily metabolised and excreted by fish and hence won't accumulate.
Assessment for accumulation of some metals by fish relies on predicted concentrations at the edge of the initial dilution zone being less than, or the same as background concentrations. Background concentrations may be over estimated thereby playing down accumulation potential.	Data for the specific ocean location has been used for background concentrations of Cd & Se (Aquenal 2005). For Hg global background concentrations from areas not impacted by industry were used. For metals considered these background concentrations are rapidly achieved within the initial dilution zone.	It is unlikely outfall specific background concentrations for Hg will be markedly less than the general global oceans. Not using location specific data for Hg is considered to have minimal impact on the health risk assessment.
Background intake of dioxins may not be representative for northern Tasmanians.	Total intake of dioxin like substances is dominated by background intake. Hence error in estimating background intakes could have noticeable effect on total intake estimations. Nevertheless the best available data for upper bound background intake has been used.	Error in background TEQ intake does not affect the conclusion that adverse health effects are unlikely. Even using upper bound background estimates for 95th percentile background estimations, the calculated total intakes in the risk assessment are appreciably less than the 'safe' intake level.

Uncertainty/Assumption	Comment	Effect on Risk Assessment
Dilution assumptions		
The assumed dilution of 100x at the edge of the DZ100 may not be achieved.	For the purposes of the risk assessment the zone of initial dilution was defined as the area where 100x dilution occurred.	The risk assessment has been conducted on the assumption a 100x dilution will be achieved within quite a short distance of the diffuser. This minimises the body of water where fish can spend more time exposed to higher effluent constituent concentrations. If this is not achieved then the qualitative HRA for metals may need to be revisited.
The target dilution of 100x might occur a long way from the diffuser thereby creating a large body of water where the effluent concentration is higher than that assumed in the risk assessment.	The diffuser is to be designed so the 100x dilution is achieved within a short distance of effluent release. The draft hydrodynamic modelling report of GHD (2006) concluded that with the exception of AOX the modelling shows all outfall constituents will be diluted to satisfactory concentrations within a distance of 125 m.	
The risk assessment has been conducted assuming 100x dilution, i.e. at the edge of the initial zone of dilution. Fish may spend significant amounts of time within the DZ100 because of increased nutrients and therefore be exposed to higher concentrations of nutrients.	Because the zone of initial dilution is quite small (the draft hydrodynamic report suggests ~125m) it is considered unlikely fish will spend significant amounts of time in waters of higher concentration even if there are high nutrient levels in the water. For example they will not breed near the outfall. It is more likely time will be spent at lower concentrations. We have however not conducted a literature review on the life cycle of local fish, nor one to substantiate the assumption that fish are attracted to, and remain in the very close proximity of pulp mill effluent ocean outfalls. Notwithstanding the above, if fish were to stay very close to the outfall and accumulate metals we consider that because the human health effects of the metals concerned are associated with long term high intake, there would not be sufficient fish stocks within the DZ100 to sustain the catch and intake levels.	If the assumption that fish are attracted to, and remain for substantial periods of time within the zone of initial dilution is supported then the qualitative risk assessment will need to be re-evaluated.

Uncertainty/Assumption	Comment	Effect on Risk Assessment
Species uncertainty The screening process is specific for fish and may not be applicable to other seafood acquired from the vicinity of the outfall.	Bioconcentration of various metals is a natural phenomenon. Hence a health risk assessment for shellfish consumption after the outfall becomes operational requires this background information. However bioaccumulation in shellfish is not addressed because several attempts to retrieve shellfish during the biological survey failed (Aquenal 2005).	It is very unlikely shellfish from the vicinity of the outfall will be collected and consumed.
Once the outfall becomes operational the abundance and variety of organisms may change such that it becomes plausible significant amounts of fish or shellfish could be caught and consumed.	Incremental intake of dioxin TEQ was determined using maximum total fish intake levels for Tasmanians (i.e. for all forms of fish food including prepared fish). It assumed no fish intake from other sources than from the vicinity of the outfall. For some distance around the outfall the environment is physically unfavourable for shellfish habitation. This situation is unlikely to change after the outfall becomes operational. Like fish, shellfish do not biomagnify dioxins. BSAFs are less than 1.	Fish consumption is distinctly overestimated. Even when doubling the maximum total fish intake of Australians the incremental increase is ~0.4 pg TEQ/kg/mth, total TEQ intake (including assuming 137x more dioxin in the effluent) is ~4 fold less than the TMI (Table 6.4). Hence if additional fish were caught there will be minimal impact on the HRA conclusions.
	From ABS (1999) ~27% of all fish eaten by Tasmanian adults is finfish (fresh fish) and 2.9% 'crustacea & molluscs' (taken to be shellfish for uncertainty analysis purposes). Hence if all the shellfish consumed by a person was from the outfall it may increase the assumed seafood consumption from the area by ~10%. The risk assessment assumes the amount of fish caught from the outfall is equal to the maximum amount of all seafood (all sub-categories) eaten.	Not considering dioxin uptake into shellfish and their consumption has no impact on the conclusions of the HRA conclusions.

Uncertainty/Assumption	Comment	Effect on Risk Assessment
Risk characterisation related uncertainties		
Comparison of concentration of trace metals in the final effluent to background concentrations is not valid because the metals entering the marine environment are from an anthropogenic source.	The concentrations of metals in the final effluent were estimated by JP based on metal analysis of the eucalyptus feed stock, expert knowledge of the mill process and metal partitioning between various waste streams. There is no material difference between background metal speciation and that in the effluent. Their environmental fates will consequently be similar.	The estimates for metals are considered conservative and are likely to overestimate the actual concentrations in the final effluent.
	Relative to analysis of a single composite effluent sample from an overseas mill using similar processes as proposed for Bell Bay Mill, metal concentration estimates are higher suggesting they are likely to be conservative.	
There is uncertainty regarding the uptake of dioxins by fish, consequently dioxin risks from ingestion of fish may be under predicted.	Dioxins are not expected to be discharged at measurable levels.	Assumptions that effluent TEQ could be at the limit of analytical detection has no impact on the dioxin HRA conclusions.
	It is assumed TEQ content of fish is at steady state equilibrium with sediment and food web TEQ. This necessitates fish to reside at the ocean outfall most of the time and over predicts the amount in edible flesh.	Fish uptake of TEQ is more likely to be over estimated rather than under estimated.
	Fish and shellfish do not biomagnify dioxins. BSAFs are less than 1.	
	Decreasing sediment organic carbon (OC) in fish uptake calculations increases TEQ transfer into fish. Assuming ~7 fold less OC increased incremental intake from fish 20 fold but had no impact on total TEQ intake because it is very small compared to background intakes (Section 6.3.5.8).	Conservative exposure assumptions regarding fish consumption overestimate incremental dioxin intake.
	No allowance is made regarding human consumption of fish from sources other than from the ocean outfall.	
	Doubling the maximum fish intake by humans had negligible impact on total TEQ intake calculations.	

11. Overall conclusions

A number of substances are likely to be present in discharged Bell Bay mill effluent because they are natural constituents of wood. Some of these may be modified to varying extents by the pulping process. However there are very few substances in the treated discharged effluent that are expected to bioaccumulate in fish. Those considered by regulatory authorities to have potential for bioaccumulation in some circumstances are cadmium, selenium or mercury, and dioxin/furans.

Metals

The specific situation at the proposed effluent outfall indicates it is very unlikely selenium or cadmium will accumulate in organisms at the site. In addition, because the estimated cadmium, selenium or mercury concentrations from effluent discharge at the perimeter of the initial dilution zone are within the background range measured for the proposed location and/or around the world, it is concluded there will be no incremental increase in concentrations of these metals in biota around the outfall. Consequently there is no additional human health risk.

Dioxins

Although the Bell Bay mill processes and effluent treatments are designed to virtually eliminate dioxin formation and their presence in discharged effluent, there is a residual possibility of low amounts of dioxins being in the effluent. However, the concentration is anticipated to be substantially below the RPDC guidelines and analytical quantitation limits for effluent. The quantitative risk assessment conducted herein showed, even at maximum likely concentrations in effluent the theoretical incremental uptake of dioxins by fish was minuscule. Subsequently the total intake of dioxins by people eating fish caught in the region is much less than the intake considered by health authorities to be safe, even if large amounts of fish were consumed for a long time.

It is concluded with a high level of confidence that the possibly of low dioxin concentrations in discharged mill effluent does <u>not</u> pose a health risk to people consuming fish caught in the vicinity of the outfall.

Recreational

The World Health Organisation and Australian authorities have published water quality criteria for protecting public health during recreational water use. These guidelines are intended for coastal and inland waters where the user comes into frequent direct contact with the water. Recreational water activities in the vicinity of the proposed effluent outfall site were not noted during any of the survey work done to-date. However, it is possible boat fishing may occur in the vicinity of the outfall even though the prevailing seas and weather, and lack of fish, suggest it would be unlikely. Nonetheless, the estimated water concentrations of effluent constituents at the perimeter of the initial dilution zone do not exceed guidelines where they exist for a particular substance.

Tainting

Tainting is considered to have occurred if there is any change in food flavour or odour that is unusual. In the past pulp mill effluents discharged into fresh water systems have been associated with tainting of fish. However historical accounts of fish tainting should not be used to judge the potential of Bell Bay mill effluent to cause tainting.

Although it cannot be stated with certainty it appears that poly-chlorination of natural phenols and resins may be primarily responsible for historic tainting of fish by pulp mill effluent. Overall, it is considered modern elemental chlorine free bleaching and the state of art effluent treatment technology to be installed at the Bell Bay pulp mill will virtually eliminate these substances.

On the whole, there is little potential for tainting of fish in the outfall area and low potential for the public to be exposed to tainted fish. However there is sufficient uncertainty to support the RPDC requirement of taint testing of effluent, at least in the first few years of mill operation.

12. References

Many references were obtained and read during literature searches for this project, those with an asterisk (*) are cited in the report.

The following information was supplied by Gunns or its consultants:

ALS Environmental (2005). Laboratory report for measurement of halogenated organics. Performed by Leway & Co Environmental Services. Performed for ALS Environmental & Gunns Ltd. 10th June 2005.

*ALS Environmental (2005). Laboratory report for analytical results for dioxins and furans. Performed by ALS Environmental. Prepared for Gunns Ltd. 9th June 2005.

ALS (2005). Analysis of water sample. Performed by ALS Environmental. 5th June 2005.

ALS Environmental (2005). Report of water effluent for AA#1 Pulp mill final effluent. Performed by ALS Environmental. 17th June 2005.

*Aquenal (2005). Aquenal (2005). Gunns Ltd Pulp Mill. Marine biological and pollutant survey at the proposed outfall site.

Ecotox (2005). Toxicity assessment of a pulp mill effluent for the proposed Tasmanian pulp mill. Final report. June 2005. 98 pages.

FSA (2001). COT statement on the tolerable daily intake for dioxins and dioxin-like poly chlorinated biphenyls. Food Standards Agency, United Kingdom.

*GHD (2005). Projects GHD Subject Draft Report Volume 3 –Water Supply Pipeline from Trevallyn Dam. Effluent Pipeline from the Pulp Mill to Five Mile Bluff and Ocean Outfall Construction Camp. Gunns Limited. October 2005.

*GHD (2006). Gunns Pulp Mill EIS Hydrodynamic Modelling Report. March 2006.

*GHD (2006a). Excel spreadsheet titled “Gunns Monitoring Program and Results”. Worksheet “Pulp Mill. Project. Effluent Outfall Monitoring Program”. Provided by GHD by email 28/06/06. This spreadsheet provides additional sampling results for compounds in fish in the vicinity of the proposed diffuser site.

*JP (2005a). Compliance of the final effluent quality with ANZECC guidelines. Prepared for Gunns Ltd. Ref No: 16B0104 – E0016. June 10th 2005. Jaakko Pöyry Oy.

*JP (2005b). Bioassay and chemical analysis results of the Advance Agro BEKP Mill effluent. Prepared for Gunns Ltd. Ref. No 16B0104. 16 June, 2005. pages 18 2. Jaakko Pöyry Oy.

*JP (2005)c. Excel spreadsheet titled “Roger Effluent Loads & WWTP Design.Base.Case.15.06.05, worksheet FE.DOM. Prepared by Jaakko Pöyry Oy sent by Gunns Ltd 18/11/05. This spreadsheet details the estimated composition of dissolved organic matter (DOM) in Final Effluent.

*JP (2005d). Excel spreadsheet titled “Roger NSI.Emissions.November.05.New.” Three worksheets are cited in the report; “I-TEQ” - details the estimate for concentration of total PCDD/Fs at Bell Bay, “CPhs” – details the estimate for total chlorinated phenolics expected in the final effluent at Bell Bay, “Extractives” – details the estimates of final effluent concentrations for substances extracted from wood including resin and fatty acids. Prepared by Jaakko Pöyry Oy sent by Gunns Ltd 18/11/05.

*JP (2005)e. Excel spreadsheet titled “Effluent Loads and WWTP Design.HMS.Dec.05”. One worksheet is cited in the report; “HMEms.New” - details the estimate for concentration of trace metals in the final effluent based on analysis of trace metals in plantation, native eucalypts and pine and the water balance of the Bell Bay mill. Prepared by Jaakko Pöyry Oy sent by Gunns Ltd 19/12/05.

JP (2005f). Bioassay and Chemical Analysis Results of the Advance Agro BEKP Mill Effluent. Prepared for Gunns Ltd. Ref. No 16B0104. 16 June, 2005. pages 18

JP (2005g). Environmental Control: Kraft pulp mill health risks and prevention. Preliminary report. Prepared for Gunns Ltd. Ref. No 16B0104-E0026. 26 September, 2005. pages 30

JP (2005h). Environmental control. Kraft pulp mill health risks and prevention. Prepared for Gunns Ltd. Ref. No 16B0104-E0026. 26 September 2005.

*JP (2005i). email titled “Final effluent content of mercury” Personal Communication via email 24/1/05 with Jaakko Pöyry Oy

References Obtained from the Literature

*ABS (1999). National Nutrition Survey: Foods Eaten. Australia 1995. Report No: 4804. Prepared by McLennan, W. and Podger, A. for the Australian Bureau of Statistics (ABS).

*Addison, R.F. and Ross, P., S. (2001). Organochlorine (OC) compounds in British Columbia marine mammals – An overview of trends and concerns. Proceedings of Puget Sound Research Conference 2001.

*Addison, R.F., Ikonomou, M.G., Ferandez, M.P. and Smith, T.G. (2005). PCDD/F and PCB concentrations in Arctic ringed seals *(Phoca hispida)* have not changed between 1981 and 2000. Science of the Total Environment 351-352:301-311.

*AET (1996). AET Cluster Rule Submission. Submitted to the US EPA August 14, 1996. Alliance for Environmental Technology (www.aet.org).

*AET (2002). Eco-system recovery: Liftings of Fish Consumption Advisories Dioxin Downstream of US Pulp and Paper Mills. Alliance for Environmental Technology. (www.aet.org/reg_market_news/press_release/2002/ecosys_2002.html).

AMAP (1998). Artic pollution issues. Arctic Monitoring and Assessment Program Assessment Report. http://www.amap.no/

*ANZECC (2000). Australian and New Zealand guidelines for fresh and marine water quality, 2000. Australian and New Zealand Environment and Conservation Council. Environment Australia.

*ANZECC (2000b). Australian and New Zealand Guidelines for Fresh and Marine Water Quality. Volume 2, Aquatic Ecosystems –Rationale and Background Information. Ch. 8.3 Toxicants. Australian and New Zealand Environment and Conservation Council. October 2000 (www.deh.gov.au/water/quality/nwqms/volume2.html).

*Apte, S.C., Bately, G.E., Szymczak, R., Rendell, P.S., Lee, R. and Waite, T.D. (1998). Baseline trace metal concentrations in New South Wales coastal waters. Marine and Freshwater Research 49:203-214 as cited in ANZECC (2000b). Australian and New Zealand Guidelines for Fresh and Marine Water Quality. Volume 2, Aquatic Ecosystems –Rationale and Background Information. Ch. 8.3 Toxicants. Australian and New Zealand Environment and Conservation Council. October 2000 (www.deh.gov.au/water/quality/nwqms/volume2.html).

*Aquarium Reference (2005). *Pseudolabrus psittaculus* Species Summary (www.aquariumreference.org).

Arnot, J.A. and Gobas, F.A.P.C. (2004). A food web bioaccumulation model for organic chemicals in aquatic ecosystems. Environmental Toxicology and Chemistry. 23:2343-2355.

Assmuth, T. and Jalonen, P. (2004). Risks and management of dioxins and dioxin-like compounds in Baltic Sea fish: An integrated assessment. Draft. Nordic Council of Ministers.

* ATSDR (1992). Public Health Assessment Guidance Manual. U.S. Department of Health and Human Services, Public Health Service, Agency for Toxic Substances and Disease Registry.

*ATSDR (1996). Toxicological Profile for Selenium (Update). U.S. Department of Health and Human Services, Public Health Service, Agency for Toxic Substances Disease Registry, Atlanta, GA

*ATSDR (1998b). Toxicological Profile for Chlorinated Dibenzo-p-dioxins (CDDs). U.S. Department of Health and Human Services, Public Health Service, Agency for Toxic Substances and Disease Registry. (Update) (PB/99/121998)..

Atwell L., Hobson, K.A. and Welch, H.E. (1998). Biomagnification and bioaccumulation of mercury in an arctic marine food web: insights from stable nitrogen isotope analysis. Canadian Journal of Fisheries and Aquatic Sciences. 55: 1114-1121.

*Australian Standard (2005). Sensory analysis. Method 2.2. Specific methods – Triangle test. Standard Number 2542.2.2. Standards Australia, Commonwealth of Australia.

*Australian Standard (1988). Sensory analysis of foods. Part 2. Specific Methods. Method 2.3. Rating. Standard Number 2542.2.3. Standards Association of Australia, Commonwealth of Australia.

Barwick, M. and Maher, W. (2003). Biotransference and biomagnification of selenium copper, cadmium, zinc, arsenic and lead in a temperate seagrass ecosystem from Lake Macquarie Estuary, NSW, Australia. Marine Environmental Research. 56: 471-502.

BC MoE (1998). Water quality guidelines (criteria) reports: Chlorophenols. Environmental Protection Division, Water, Air and Climate Change Branch. British Columbia Ministry of Environment. http://www.env.gov.bc.ca/wat/wq/BCguidelines/chlorophenols/chlorophenol.html Accessed December 2005.

Bell, L.M. and Kallman, R.J. (1976). The Kitimat river estuary. Status of environmental knowledge to 1976. Special estuary series No. 6. Fisheries and Marine Service, Environment Canada. West Vancouver, B.C. As cited in McLeay 1987.

*Bignert, A., Olsson, M., Bergqvist, P.,A., Bergek, S., Rappe, C., de Wit, C. (1989). Polychlorinated dibenzo-p-dioxins (PCDD) and dibenzo-furans (PCDF) in seal blubber. Chemosphere 1989: 19:551– 6.

*Boudou, A., Inze, B., Lemaire-Gony, S., Maury-Brachet, R., Odin, M. and Ribeyre, F. (1998). Metal Bioaccumulation in Freshwater Systems: Experimental Study of the Actions and Interactions Between Abiotic and Contamination Factors. Chapter 15 from Schüürmann, G. and Markert, B. (1998). Ecotoxicology. Ecological Fundamentals, Chemical Exposure, and Biological Effects. John Wiley & Sons Inc and Spektrum Adaemischer Verlag Co-Publication.

*Boyle, E.A., Scalter, F. and Edmond, J.M. (1976). On the marine geochemistry of cadmium. Nature 263:42-44 as cited in WHO (1992). EHC 135 –Cadmium environmental aspects (http://www.inchem.org/documents/ehc/ehc/ehc135.htm).

*Bright, D.A., Hodson, P.V., Lehtinen, K.J., McKague, A.B., Rodgers, J.H. and Soloman, K. R. (2003). Evaluation of ecological risks associated with the use of chlorine dioxide for the bleaching of pulp: Scientific progress since 1993. In Environmental Impacts of Pulp and Paper Waste Streams, Stuthridge, T., van den Hueval, M.R., Marvin, N.A., Slade, A.H. and Gifford, J. (Ed). Setac Press, 2003.

*Brouzes, R.J.P., Liem, A.J., and Naish, V.A. (1978). Protocol for fish tainting bioassay. CPAR Rep. No. 775-1. Environmental Protection Service, Ottawa, Ontario. As cited in McLeay, 1987.

*Bruland, K.W. (1983). Trace elements in seawater. In “Chemical Oceanography”, Vol 8, eds JP Riley & R Chester, Academic Press, London, 157-220 as cited in ANZECC (2000b). Australian and New Zealand Guidelines for Fresh and Marine Water Quality. Volume 2, Aquatic Ecosystems – Rationale and Background Information. Ch. 8.3 Toxicants. Australian and New Zealand Environment and Conservation Council. October 2000 (www.deh.gov.au/water/quality/nwqms/volume2.html).

Brumley, C.M., Haritos, V.S., Ahokas, J.T. and Holdway, D.A. (1996). Metabolites of chlorinated syringaldehydes in fish bile as biomarkers of exposure to bleached eucalypt pulp effluents. Ecotoxicology and Environmental Safety. 33: 253-260.

Brumley, C.M., Haritos, V.S., Ahokas, J.T., and Holdway, D.A. (1998). The effects of exposure duration and feeding status on fish bile metabolites: Implications for biomonitoring. Ecotoxicology and Environmental Safety. 39: 147-153

Buhler, D.R. and Williams, D.E. (1988). The role of biotransformation in the toxicity of chemicals. Aquatic Toxicology. 11: 19-28.

Burger, J. (2002). Consumption patterns and why people fish. Environmental Research Section A. 90: 125-135.

*Burggraaf, S., Langdon, A.G., Wilkins, A.L. and Roper, D.S. (1996). Accumulation and depuration of resin acids and fichtelite by the freshwater mussel *Hyridella menziesi*. Environmental Toxicology and Chemistry 15(30:369-375.

*Burkhard, L.P. and Lukasewycz, M.T. (2000). Some bioaccumulation factors and biota-sediment Accumulation factors for polycyclic aromatic hydrocarbons in lake trout. Environ. Toxicol. Chem. 19:1427-1429.

*Butler, A., Althaus, F., Furlani, D. and Ridgway, K. et al (2002). Assessment of conservation values of the Bass Strait sponge beds area. CSIRO marine Research. Report to Environment Australia. As cited in Aquenal (2005): Gunns Ltd Pulp Mill. Marine biological and pollutant survey at the proposed outfall site.

Campbell, L.M., Norstrom,R.J. Hobson, K.A., Muir, D.C.G. Backus, S. and Fisk, A.T. (2005). Mercury and other trace elements in a pelagic Arctic marine food web (Northwater Poynya, Baffin Bay). Science of the Total Environment. 351-352: 247-263.

*CCREM (1987). Canadian water quality guidelines. Canadian Council of Resource and Environment Ministers, Ontario. . As cited in ANZECC (2000).

Chapman, P.M. (1999). Selenium – A potential time bomb or just another contaminant. Human and Ecological Risk Assessment. 5(6): 1123-1138. Abstract only.

*Chapman, P.M., Wang, F., Janssen, C.R., Goulet, R.R. and Kamunde, C.N. (2003). Conducting ecological risk assessments of inorganic metals and metalloids: current status. Human and Ecological Risk Assessment 9(4):641-697.

Clarke, K.E., Gobas, F.A.P.C. and Mackay, D. (1990). Model of organic chemical uptake and clearance by fish from food and water. Environmental Science and Technology. 24: 1203-1213.

*Connell, D.W. (1998). Bioaccumulation of Chemicals by Aquatic Organisms. Chapter 14 from Schüürmann, G. and Markert, B. (1998). Ecotoxicology. Ecological Fundamentals, Chemical Exposure, and Biological Effects. John Wiley & Sons Inc and Spektrum Adaemischer Verlag Co-Publication.

*Connor, K.T. and Aylward, L.L. (2006). Human response to dioxin: Aryl hydrocarbon receptor (AhR) molecular structure, function, and dose-response data for enzyme induction indicate an impaired human AhR. J. Toxicol. Environ. Health, Part B, 9: 146 – 171.

* Cook, P. M. and Burkhard, L. P. (1998). Development of bioaccumulation factors for protection of fish and wildlife in the Great Lakes. In: *National Sediment Bioaccumulation Conference Proceedings*. U.S. Environmental Protection Agency, Office of Water. Washington, DC. EPA 823-R-002 (cited in USEPA 2000a).

* Cook, W.H., Farmer, F.A., Kristiansen, O.E., Reid, K., Reid, J., Rowbottom, R. (1973). The effect of pulp and paper mill effluents on the taste and odour of the receiving water and the fish therein. Pulp Paper Mag. Can. 74: 97-106.

*Cossa, D. (1988). Cadmium in *Mytilus* spp.: Worldwide survey and relationship between seawater and mussel content. Marine Environmental Research 26:265-284. As cited in ANZECC (2000b). Australian and New Zealand Guidelines for Fresh and Marine Water Quality. Volume 2, Aquatic Ecosystems –Rationale and Background Information. Ch. 8.3 Toxicants. Australian and New Zealand Environment and Conservation Council. October 2000 (www.deh.gov.au/water/quality/nwqms/volume2.html).

*Craig, G.R. and Stasiak, M. (1993). Fish tainting chemicals – separation, isolation and identification of compounds from pulp and paper effluent. 14th Annual Meeting (SETAC) – Ecological Risk assessment: Lessons Learned? Abstract only.

*Croce, B., Stagg, R.M. and Smith,M. (1995). Effects of exposure to a combination of [beta]-naphthoflavone and resin acids on biotransformation enzymes in Atlantic salmon parr *(Salmo salar)*. Aquatic Toxicology 32(2-3):161-176.

*CSIRO (1996). Port Phillip Bay as cited in ANZECC (2000). Australian and New Zealand Guidelines for Fresh and Marine Water Quality. Volume 2, Aquatic Ecosystems –Rationale and Background Information. Ch. 8.3 Toxicants. Australian and New Zealand Environment and Conservation Council. October 2000 (www.deh.gov.au/water/quality/nwqms/volume2.html).

*Currie, R.S., Fairchild, W.L., Holoka, M.I.H. and Muir, C.G. (2000). Long-term fate and bioavailability of 2,3,7,8-tetrachlorodibenzofuran in littoral enclosures. Environmental Toxicology and Chemistry 19(6):1491-1500.

*DAFF (2005). The gummy shark. Department of Agriculture Forestry and Fisheries. The Bureau of Rural Sciences and the Fisheries Research and Development Corporation (1993). Reproduced from The Australian Fisheries Resources (www.edaff.gov.au).

*Dahlman, O.B., Reimann, A.K., Stromberg, L.M. and Mörck, R.E. (1995). High molecular weight effluent materials from modern ECF and TCF bleaching. Tappi Journal 78: 99 – 109.

*DEH (1995). The State of the Marine Environment Report for Australia Technical Annex 2: Heavy metals and tributyltin in Australian coastal and estuarine waters. Department of the Environment, Sport & Territories, Canberra.

*de Conto Cinier, C., Petit-Ramel, M., Faure, R., Garin, D. and Bouvet, Y. (1999). Kinetics of cadmium accumulation and elimination in carp *Cyprinus carpio* tissues. Comparative Biochemistry and Physiology Part C. 122: 345-352.

*de Swart, R.L., Ross, P.S., Timmerman, H.H., Hijman, W.C., de Ruiter, E.M., Liem, A.K.D., Brouwer, A., van Loveren, H., Reijnders, P.J.H., Vos, J.G.and Osterhaus, A.D.M.E. (1995). Short term fasting does not aggravate immunosuppression in harbour seals (*phoca vitulina*) with high body burdens of organochlorines. Chemosphere 31: 4289 – 4309.

*deWit, C., Jannson, B., Bergek, M., Hjelt, M., Rappe, C., Olsson, M., and Andersson, O. (1992). Polychlorinated dibenze-p-dioxin and polychlorinated dibenzofuran levels in fish and fish-eating wildlife in the Baltic sea. Chemosphere 25:185 – 188.

*Di Giulio, R.T., Benson, W. H., Sanders B. M., and van Veld, P.A . (1995). Biochemical mechanisms: metabolism, adaption, and toxicity. In 'Fundamentals of Aquatic toxicology' Ed Rand, G., M. Chapter 17 Taylor & Francis, p 523-560.

*Dix, T. G. and Martin, A. (1975). Sand Flathead (Platycephalus bassensis), an indicator species for mercury pollution in Tasmanian waters. Marine Pollution Bulletin: 6(9) 142-143.

Dos Santos, I.R., Silva-Filho, E.V., Schaefer, C., Sella, S.M., Silva, C.A., Gomes, V., Passos, M.J. and Ngan, P.V. (2005). Baseline mercury and zinc concentrations in terrestrial and coastal organisms of Admiralty Bay, Antartica. Environmental Pollution. In press.

*EA (1998). National Pollutant Inventory. Emission Estimation Technique Manual for Pulp and Paper Manufacturing. Environment Australia August 1998.

*EA/MoHW (1999). Report on Tolerable Daily Intake (TDI) of Dioxins and related Compounds (Japan). Environmental Health Committee of the Central Environment Council, Environment Agency. Living Environment Council and Food Sanitation Investigation Council, Ministry of Health and Welfare, Government of Japan.

*EA/MoHW (2001). Dioxins. Ministry of the Environment, Office of Dioxins Control, Administration Division, Environment Management Bureau, Government of Japan.

*EA/MoHW (2003). Dioxins. Office of Dioxins Control, Environment Management Bureau, Ministry of the Environment, Government of Japan.

*Eaton, A. (1976). Marine geochemistry of cadmium. Marine Chem. 4: 141-154 as cited in Fergusson, J.E. (1990) The Heavy Elements: Chemistry, Environmental Impact and Health Effects. Pergamon Press, Headington Hill Hall, Oxford. P. 178.

*Ebens, R.J. and Shacklette, H.T. (1982). Geochemistry of some rocks, mine spoils, stream sediments, soils, plants, and waters in the western energy region of the conterminous United States. U.S. Geol. Surv. Prof. Paper 1237. U.S. Govt. Printing Off., Washington D.C. as cited in Eisler, R (2000). Handbook of Chemical Risk Assessment Health Hazards to Humans, Plants, and Animals. Volume 1. Metals. Chapter 31 –Selenium. Lewis Publishers. Boca Raton, Florida.

*EC (1991). Priority Substances List Assessment Report No. 2 for Effluents from Pulp Mills Using Bleaching Environment Canada, Health Canada.

EC (1999). Compilation of EU Dioxin Exposure and Health Data. Task 1- Member State Legislation and Programmes. Prepared by Petersen, A. for European Commission DG Environment and UK Department of the Environment, Transport and the Regions. October 1999.

*Edgar, G.J. and Shaw, C. (1995). The production and trophic ecology of shallow-water fish assemblages in southern Australia. II. Diets of fishes and trophic relationships between fishes and

benthos at Western Port, Victoria. Journal of Experimental Marine Biology and Ecology 194: 83-106.

* Edgar, G.J. (1997). Australian Marine Life - the plants and animals of temperate waters. Reed Books Australia cited in Aquenal (2005).

*EC-SCF (2001). Opinion of the Scientific Committee on Food (SCF) on the Risk Assessment of Dioxins and Dioxin-like PCBs in Food. Update based on new scientific information available since the adoption of the SCF Opinion of 22nd November 2000. EC Health and Consumer Protection Directorate- General. Document CS/CNTM/DIOXIN/20 final. Adopted on 30th May 2001.

*Elliot, J.E., Scheuhammer, A.M., Leighton, F.A. and Pearce, P.A. (1992). Heavy-metal and metallothionein concentrations in Atlantic Canadian seabirds. Archives of Environmental Contamination and Toxicology. 22(1): 63-73.

*Elliot, J.E. and Scheuhammer, A.M. (1997). Heavy metal and metallothionein concentrations in seabirds from the Pacific Coast of Canada. Marine Pollution Bulletin. 34(10): 794-801.

*enHealth (2002). Environmental Health Risk Assessment. Guidelines for Assessing Health Risks from Environmental Hazards. Department of Health and Aging and enHealth Council, Commonwealth of Australia.

* enHealth (2003). Australian exposure assessment handbook. Consultation draft. enHealth Council. http://www.health.gov.au/internet/wcms/publishing.nsf/Content/health-pubhlth-strateg-envhlth-exposure.htm-copy3/$FILE/exposure.pdf.

*EUROPA (1976). Bathing water quality directive 76/160/EEC. Council Directive of 8 December 1975. Europa Environment (http://www.europa.eu.int/water/water-bathing/directiv.html).

*Fabris, G.J., Monahan, C.A. and Batley, G.E. (1999). Heavy metals in waters and sediments of Port Phillip Bay, Australia. Mar. Freshwater Res. 50:503-513.

*Fabris, G.J., Turoczy, N.J. and Stagnitti, F. (2005). Trace metal concentrations in edible tissue of snapper, flathead, lobster, and abalone from coastal waters of Victoria, Australia. Ecotoxicology and Environmental Safety. In Press, Corrected Proof, Available online 29 January 2005.

*Farmer, F.A., Neilson, H.R. and Esar, D. (1973). Flavour evaluation by triangle and hedonic scale tests of fish exposed to pulp mill effluents. Can. Inst. Food Sci. Technol. J. 6: 12-16. As cited in Kenefick et al 1995.

*FEI (1996). Chapter 2: Technical development and environmental implications. In 'The Finnish Environment Report No.17 , The Finnish Environment Institute.

Felipe, Z.S., Beckett, R. and Hart, B. (2001). Ecological risk assessment (ERA) in Derwent Estuary, Tasmania, Australia: A critical evaluation of the problem formulation phase of the ERA process. Conference Proceedings of the Marine Education Society Australasia. http://www.mesa.edu.au/aaee_conf/San%20Felipe-Beckett-Hart.PDF.

*Findlay, D.M., Naish, V.A. (1979). Nature and sources of tainting in a kraft mill. CPAR Rep No 775-2. Environmental Protection service, Ottawa, Ontario. As cited in McLeay 1987.

*Fisk, A.T., Norstrom, R. J., Cymbalisty, C. C. and Muir D. C.B. (1998). Dietary accumulation and depuration of hydrophobic organochlorines: Bioaccumulation parameters and their relationship with the octanol/water partition coefficient. Environmental Toxicology and Chemistry 17: 951-961.

*Fitzgerald, W.F. (1979). Distribution of mercury in natural waters. P161-173 in JO Nriagu (ed.). The biogeochemistry of Mercury in the Environment. Elsevier/North-Holland Biomedical Press, NY as cited in Eisler, R (2000). Handbook of Chemical Risk Assessment Health Hazards to Humans, Plants, and Animals. Volume 1. Metals. Chapter 5 –Mercury. Lewis Publishers. Boca Raton, Florida.

*Fleisher, M., Arofim, A.F., Fassett, D.W., Hammond, P., Shacklette, H.T., Nisbet, I.C.T. and Epstein, S. (1974). Environmental impact of cadmium: A review by the panel on hazardous trace substances. Environmental Health Perspectives 7:253-323 as cited in Fergusson, J.E. (1990). The Heavy Elements: Chemistry, Environmental Impact and Health Effects. Pergamon Press, Headington Hill Hall, Oxford. P. 177.

Fragoso, N.M., Hodson, P.V., Kozin, I.S., Brown, R.S., Parrott, J.L: (1999). Kinetics of mixed function oxygenase induction and retene excretion in retene exposed rainbow trout (*oncorhynchus mykiss)*. Environmental Toxicology and Chemistry. 18: 2268-2274.

Franke, C. Studinger, G., Berger, G., Böhling, S., Bruckmann, U., Cohors-Fresenborg, D., Jöhncke, U. (1994). The assessment of bioaccumulation. Chemosphere. 29: 1501-1514.

*Frost, D.V. and Ingvolstad, D. (1975). Ecological aspects of selenium and tellurium in human and animal health. Chem. Scr. 8A:96-107 as cited in Eisler, R (2000). Handbook of Chemical Risk Assessment Health Hazards to Humans, Plants, and Animals. Volume 1. Metals. Chapter 31 – Selenium. Lewis Publishers. Boca Raton, Florida.

*Gatehouse, R. (2004). Ecological risk assessment of dioxins in Australia –technical report no. 11. Government, Department of the Environment and Heritage May 2004 (http://www.deh.gov.au/settlements/publications/chemicals/dioxins/report-11/index.html).

*Gaus, C., Papke, O., Dennison, N., Haynes, D., Shaw, G.R., Connell, D.W. and Muller, J.F. (2001). Evidence for the presence of a widespread PCDD source in coastal sediments and soils from Queensland, Australia. Chemosphere. 43: 549-558.

Goodman, D.G., and Sauer, R.M. (1992). Hepatotoxicity and carcinogenicity in female sprague-dawley rats treated with 2,3,7,8-Tetrachlorodibenzo-*p*-dioxin (TCDD): A pathology working group reevaluation. Regulatory Toxicology and Pharmacology. 15: 245-252.

*Gordon, M.R., Mueller, J.C. and Walden,C.C. (1980). Effect of biotreatment on fish tainting propensity of bleached kraft whole mill effluent. Trans. Tech. Sect. Canada. Pulp. Paper Assoc. Vol 6. TR 2-8. As cited in McLeay 1986.

*Goyer, R.A. and Clarkson, T.W. (2001). Toxic effects of metals. In Casarett and Doull's Toxicology: The basic science of poisons. 6th edition. Editors: C. D. Klaassen. McGraw-Hill, Sydney. 811-867.

*Gravato, C. and Santos, M.A. (2002). Juvenile sea bass liver biotransformation and erythrocytic genotoxic responses to pulp mill contaminants. Ecotoxicology and Environmental Safety 53(1):104-112.

Gravato, C. and Santos, M.A. (2002). Juvenile sea bass liver biotransformation and erythrocytic genotoxic responses to resin acids. Ecotoxicology and Environmental Safety. 53: 104-112.

*Gutiérrez, A., Romero, J. and del Río, J.C. (2001). Lipophilic extractives in process waters during manufacturing of totally chlorine free kraft pulp from eucalypt wood. Chemosphere. 44: 1237-1242.

Gutiérrez, A. and del Río, J.C. (2001). Gas chromatography/ mass spectrometry demonstration of steryl glycosides in eucalypt wood, Kraft pulp and process liquids. Rapid Communications in Mass Spectrometry. 15: 2515-2520.

*Hamasaki, T., Nagaawe, H., Yoshioka, Y. and Sato, T. (1995). Formation, distribution and ecotoxicity of methylmetals of tin, mercury, and arsenic in the environment. Crit Rev Environ Sci Technol 25: 45-91 as cited in Eisler, R (2000). Handbook of Chemical Risk Assessment Health Hazards to Humans, Plants, and Animals. Volume 1. Metals. Chapter 5 –Mercury. Lewis Publishers. Boca Raton, Florida.

*Hamilton, S.J. (2004). Review of selenium toxicity in the aquatic food chain. Science of the Total Environment 326:1-31.

*Hamilton, S.J. (2005). Selenium impacts on razorback sucker, Colorado river, Colorado I. Adults. Ecotoxicology and Environmental Safety, 61: 7-31.

Hanninen, E., Pekkanen, M., Kiiskila, E. and Valttila, O. (1996). Towards zero effluent pulp and paper production. APPITA 1996: 749-756.

*Hawaii Coral Reef Network. Family Monacanthidae (www.coralreefnetwork.com).

*Haws, L.C., Su, S.H., Harris, M., de Vito, M.J., Walker, N.J., Farland, W.H., Finley, B. and Birnbaum, L. (2006). Development of a refined database of mammalian relative potency estimates for dioxin-like compounds. Toxicological Sciences. 89(1): 4-30.

*Haynes, D., Mosse, P. and Oswald, L. (1995). The use of transplanted cultured mussels (*Mytilus edulis*) to monitor pollutants along the Ninety Mile Beach, Victoria, Australia – II. Polychlorinated dibenzo-*p*-dioxins and dibenzofurans. Marine Pollution Bulletin. 30(12): 834-839.

Hays, S.M., Aylward, L.L., Karch, N.J., and Paustenbach, D.J. (1997). The relative susceptibility of animals and humans to the carcinogenic hazard posed by exposure to 2,3,7,8-TCDD: An analysis using standard and internal measures of dose. Chemosphere. 34(5-7): 1507-1522.

Haynes, D., Leeder, J. and Rayment, P. (1997). A comparison of the bivalve species *Donax deltoides* and *Mytilus edulis* as monitors of metal exposure from effluent discharges along the Ninety Mile Beach, Victoria, Australia. Marine Pollution Bulletin. 34(5): 326-331.

*Hendriks, A.J., Pieters, H. and de Boer, J. (1998). Accumulation of metals, polycyclic (halogenated) hydrocarbons, and biocides in zebra mussel and eel from the Rhine and Meuse rivers. Environ. Toxicol. Chem 17:1885-1898.

*Henry, G.W. and Lyle, J.M. (2003). The National Recreational and Indigenous Fishing Survey. Tasmanian Aquaculture & Fisheries Institute, University of Tasmania. FRDC Project No. 99/158. July 2003. www.affa.gov.au .

*Hewitt, M., Carey, J.H., Dixon, G. and Munkittrick, K.R. (1996). Examination of bleached kraft mill effluent fractions for potential inducers of mixed function oxygenase activity in rainbow trout. In Environmental Fate and Effects of Pulp and Paper Mill Effluents. Servos , M.,R., Munkittrick, K.,R., Carey, J.,H., and van der Kraak, G.,J. (Ed). St Lucie Press (1996).

*Higgins, H.W. and Mackey, D.J. (1987). Role of *Ecklonia radiata* (C. Ag.) J. Agardh in determining trace metal availability in coastal. I. Total trace metals. Australian Journal of Marine and Freshwater Research 38:307-316 as cited in ANZECC (2000). Australian and New Zealand Guidelines for Fresh and Marine Water Quality. Volume 2, Aquatic Ecosystems –Rationale and Background Information. Ch. 8.3 Toxicants. Australian and New Zealand Environment and Conservation Council. October 2000 (www.deh.gov.au/water/quality/nwqms/volume2.html)

Holdway, D.A., Brennan, S.E. and Ahokas, J.T. (1995). Short review of selected fish biomarkers of xenobiotic exposure with an example using fish hepatic mixed-function oxidase. Australian Journal of Ecology. 20(10): 34-44.

Irvine, G.M., Clark, N.B. and Recupero, C. (1997). Extended delignification of mature and plantation eucalypt wood. CSIRO National Pulp Mills Research Program. Technical Report Series. No. 23. Commonwealth of Australia.

*Jardine, C.G. (1992). Public evaluation of fish tainting from pulp and paper mill discharges. Wat. Sci. Tech. 25: 57-64.

*Jarvinen and Ankley (1999). Linkage of effects to tissue residues: Development of a comprehensive database for aquatic organisms exposed to inorganic and organic chemicals. SETAC Technical Publication Series, SETAC Press, Pensacola FL.

Jaakko Pöyry Pty Ltd (1995). A study of water quality for a hypothetical Western Australian bleached eucalypt kraft pulp mill. CSIRO National Pulp Mills Research Program. Technical Report Series. No. 12. Commonwealth of Australia.

*Javitz, H. (1980). Seafood consumption data analysis. SRI International. Final report prepared for EPA Office of Water Regulations and Standards. EPA Contract 68-01-3887. As cited in USEPA (1997).

*JECFA (2001). 57th Meeting of the Joint FAO/WHO Expert Committee on Food Additives (JECFA), Rome, 5 – 14 June 2001. Summary and Conclusions, Annex 4, pp 24-40. Full report published in Food Additive Series 48 as “Polychlorinated Dibenzodioxins, Polychlorinated Dibenzofurans, and Co-planar Polychlorinated Biphenyls” (2002). pp 451-664.

Jones, M., Monahan, C., Richardson, D., Kearney, P. and Nixon, C. (2001). An ecological risk assessment from a cold caustic soda and thermo mechanical pulp mill. APPITA 2001: 125-129.

Jordan, A., Samson, C., Lawler, M. and Halley, V. (2003). Assessment of seabed habitats and heavy metals in Cornelian bay. Tasmanian Aquaculture and Fisheries Institute, University of Tasmania.

Karels, A., Markkula, E. and Oikari, A. (2001). Reproductive, biochemical, physiological and population responses in perch (*perca fluviatilis* L.) and roach (*rutilus rutilus* L.) downstream of two elemental chlorine-free pulp and paper mills. Environmental Toxicology and Chemistry. 20(7): 1517-1527.

*Kayajanian, G.M. (2002). The j-shaped dioxin dose response curve. Ecotoxicology and Environmental Safety. 51: 1-4.

*Kenefick, S.L., Low, N.J., Hrudey, S.E. and Brownlee, B.G. (1995). A review of off flavour tainting of drinking water and fish by pulp mill effluents. Wat. Sci. Tech. 31:55-61.

*Kovacs, T.G. (1982). The effect of bleached kraft effluent on the aquatic environment. Prepared for Weyerhauser Canada Ltd. Kamloops B.C. by Pulp and Paper Research Institute of Canada. As cited in Kenefick et al 1995.

Kondo, T., Yamamoto, H., Tatarazako, N., Kawabe, K., Koshio, M. Hirai, N. and Morita, M. (2005). Bioconcentration factor of relatively low concentrations of chlorophenols in Japanese medaka. Chemosphere. 61: 1299-1304.

*Korte, F. (1983). Ecotoxicology of cadmium: General review. Exotoxicol Environ Safety 7:3-8 (as cited in Eisler, R (2000). Handbook of Chemical Risk Assessment. Health Hazards to Humans, Plants, and Animals. Volume 1. Metals. Lewis Publishers, CRC Press, Boca Raton, Florida).

*Korte (1983). Ecotoxicology of cadmium: General overview. Ecotoxicol. Environ. Saf. 7: 3-8 (as cited in WHO (1992). EHC 135 –Cadmium environmental aspects). (http://www.inchem.org/documents/ehc/ehc/ehc135.htm).

*Kostamo, A., Holmbom, B. and Kukkonene, J.V.K. (2004). Fate of wood extractives in wastewater treatment plants at kraft pulp mills and mechanical pulp mills. Water Research 38: 972-982.

*Kraal, M.H., Kraak, M.H.S., de Groot, C.J. and Davids, C. (1995). Uptake and tissue distribution of dietary and aqueous cadmium by carp (*Cyprinus carpio*). Ecotoxicology and Environmental Safety. 31: 179-183.

Kuehl, D.W., Butterworth, B.C., DeVita, W.M. and Sauer, C.P. (1987). Environmental contamination by polychlorinated dibenzo-*p*-dioxins and dibenzofurans associated with pulp and paper mill discharge. Biomedical and Environmental Mass Spectrometry. 14: 443-447.

*Kuroshima, R. (1987). Cadmium accumulation and its effect on calcium metabolism in the girella *Girella punctata* during a long-term exposure. Bulletin of the Japanese Society of Scientific Fisheries. 53(3):445-450. As cited in de Conto Cinier et al (1999).

*Kuusi, T. and Suihko, M. (1983). Occurrence of various off-flavours in fish in Finland from 1969 to 1981. Wat. Sci. Tech. 15: 47-58.

*LaFleur, L.E.(1996). Sources of pulping and bleaching derived chemicals in effluents. In "Environmental Fate and Effects of Pulp and Paper Mill Effluents". Servos ,M.R., Munkittrick, K.R., Carey, J.H., and van der Kraak, G.J. (Ed). St Lucie Press, Delray Beach, Florida (1996).

*Langer, O.E. and Nassichuk, M.D. (1975). Selected biological studies of the Thompson river system. Environment Canada. Technical Report Series No. PAC/T-75-22. As cited in Kenefick et al 1995.

*Langford, R.W. (1974). Data review of biological and chemical effects of the Crestbrook pulp mill effluent on the Kootenay river. Fish and Wildlife Branch, B.C. Ministry of Environment. Victoria B.C. As cited in McLeay 1987.

*Larscombe, J., Brooks, K., Charlambou, C., Fenton, M., Fisher, M. and Summerson, R. (2002). Marine matters –atlas of marine activities and coastal communities in South Australia's South East Marine Region. Bureau of Rural Science, Canberra as cited in Aquenal (2005). Gunns Ltd Pulp Mill. Marine biological and pollutant survey at the proposed outfall site.

Leeming, R. and Nichols, P.D. (1998). Determination of the sources and distribution of sewage and pulp-fibre-derived pollution in the Derwent Estuary, Tasmania, using sterol biomarkers. Marine Freshwater Research. 49: 7-17.

*Lemly A., D. (1999). Selenium transport and bioaccumulation in aquatic ecosystems: A proposal for water quality criteria based on hydrological units. Ecotoxicology and Environmental Safety 42: 150–156.

Leppänen, H. and Oikari, A. (1999). Occurrence of retene and resin acids in sediments and fish bile from a lake receiving pulp and paper mill effluents. Environmental Toxicology and Chemistry 18(7): 1498-1505.

Liem, A.J., Naish, V.A., Rowbootom, R.S. (1977). An evaluation of the effect of inplant treatment studies on the abatement of air wand water pollution from a hardwood kraft pulp mill. CPAR Rep. No. 484-1. Environmental Protection Service, Ottawa, Ontario. As cited in McLeay 1987.

*Liem, A.K.D., Fürst, P. and Rappe, C. (2000). Exposure of populations to dioxins and related compounds. Food Additives and Contaminants. 17(4): 241-259.

*Lindqvist, O., Johansson, K., Aastrup, M., Andersson, A., Bringmark, L., Hovsenius, G., Hakanson, L., Iverfeldt, A., Meili, M. and Timm, B., 1991. Mercury in the Swedish environment – recent research on causes, consequences and corrective methods. Water Air Soil Pollution 55: pp. xi–261. As cited in Hamilton (2004).

Lindsay, R.C. and Heil, T.P. (1992). Flavor tainting of fish in the Upper Wisconsin river caused by alkyl- and thio-phenols. Wat. Sci. Tech. 25:35-40.

Livingstone, D. R. (1998). The fate of organic xenobiotics in aquatic ecosystems: Quantitative and qualitative differences in biotransformation by invertebrates and fish. Comparative Biochemistry and Physiology Part A 120: 43-49.

*Llobet, J.M., Domingo, J.L., Bocio, A., Casas, C., Teixido, A. and Muller, L. (2003). Human exposure to dioxins through the diet in Catalonia, Spain: Carcinogenic and non-carcinogenic risk. Chemosphere. 50: 1193-1200.

Lorber, M. (2002). A Pharmacokinetic Model for Estimating Exposure of Americans to Dioxin-Like Compounds in the Past, Present, and Future. Sci. Total. Environ. 288: 81-95.

*Lowell, R.B., Ring, B., Pastershank, G., Walker, S., Trudel, L., Hedley, K. (2005). National assessment of pulp and paper environmental effects monitoring data: Findings from cycles 1 through 3. National Water Research Institute, Burlington, Ontario. NWRI Scientific Assessment Report Series No 5. 40p.

*Lowell, R.B., Ribey, S.C., Ellis, I.K., Porter, E.L., Culp, J.M., Grapentine, L.C., McMaster, M.E., Munkittrick, K.R., Scroggins, R.P. (2003). National assessment of the pulp and paper environmental effects monitoring data. National Water Research Institute, Burlington, Ontario. NWRI Contribution No 03-521.

*Luthe, C.E., Wrist, and Berry, R.M. (1992). An evaluation of the effectiveness of dioxins control strategies on organochlorine effluent discharges from the Canadian bleached chemical pulp industry. Pulp & Paper Canada 93(9): 40 – 49.

Mackay, D., Shiu, W. Y. and Ma, K. C. (1992). Polynuclear aromatic hydrocarbons, polychlorinated dioxins and dibenzofurans. Illustrated handbook of physical-chemical properties and environmental fate for organic chemicals. Lewis Publishers, London.

*Mackey, D.J. (1984). Trace metals and productivity of shelf waters off north west Australia. Australian Journal of Marine and Freshwater Research 35:505-516 as cited in ANZECC (2000b). Australian and New Zealand Guidelines for Fresh and Marine Water Quality. Volume 2, Aquatic Ecosystems –Rationale and Background Information. Ch. 8.3 Toxicants. Australian and New Zealand Environment and Conservation Council. October 2000 (www.deh.gov.au/water/quality/nwqms/volume2.html).

*Mackey et al. (1996). Bathurst Harbour & Old River, Tasmania as cited in ANZECC (2000) Australian and New Zealand Guidelines for Fresh and Marine Water Quality. Volume 2, Aquatic Ecosystems –Rationale and Background Information. Ch. 8.3 Toxicants. Australian and New Zealand Environment and Conservation Council. October 2000 (www.deh.gov.au/water/quality/nwqms/volume2.html).

*Marine Discovery Research Centre (1997). Barber Perch –Family Serranidae (www.woodbridge.tased.edu.au).

*Marine Discovery Research Centre (2001). Leatherjackets –Family Monacanthidae (www.woodbridge.tased.edu.au).

* Mauro, J.B.N., Guimaraes, J.R.D., Hintelmann, H., Watras, C.J., Haak, E.A. and Coelho-Souza, S.A. (2002). Mercury methylation in macrophytes, periphyton, and water comparative studies with stable and radio-mercury additions. Analytical and Bioanalytical Chemistry 374, 983–989.

McGeer, J. C., Brix, K. V., Skeaff, J. M., deForest, D. K., Brigham, S. I., Adams, W. J. and Green, A. (2003). Inverse relationship between bioconcentration factor and exposure concentrations for

metals: Implications for hazard assessment of metals in the aquatic environment. Environmental Toxicology and Chemistry. 22(5): 1017-1037.

McMaster, M. E., Parrott, J. L. and Hewitt, L. M. (2004). A decade of research on the environmental impacts of pulp and paper mill effluents in Canada (1992-2002). National Water Research Institute, Environment Canada, Canada. 62p

*Mcleay D. (1987). Aquatic Toxicity of Pulp and Paper Mill Effluent: A Review, Report EPS 4/PF/1, Environment Canada, Canada (1987).

Meylan, W.M., Howard, P.H., Boethling, R.S., Aronson, D., Printup, H. and Gouchie, S. (1999). Improved method for estimating bioconcentration/bioaccumulation factor from octanol/water partition coefficient. Environmental Toxicology and Chemistry. 18 (4): 664-672.

*Mondon, J.A., Nowak, B.F. and Sodergren, A. (2001). Persistent organic pollutants in oysters *Crassostrea gigas* and sand flathead *Platycephalus bassensis* from Tasmanian estuarine and coastal waters. Marine Pollution Bulletin 42(2):157-161.

*Moore, J.W. and Ramamoorthy, S. (1984). Heavy metals in natural waters: Applied monitoring and impact assessment: Springer-Verlag as cited in Fergusson, J.E. (1990). The Heavy Elements: Chemistry, Environmental Impact and Health Effects. Pergamon Press, Headington Hill Hall, Oxford. P. 178-179.

*Morato, T., Encarnacion, S., Gros, M.P. and Menezes, G. (2003). Diets of thornback ray (*Raja clavate*) and tope shark (*Galeorhinus galeus*) in the bottom longline fishery of the Azores, north-eastern Atlantic. Fisheries Bulletin 101:590-602.

* Morel F., M., M., Kraepiel A., M., L., and Amyot M (1998). The chemical cycle and bioaccumulation of mercury. Annual Review of Ecology and Systematics. 29: 543-567.

*Mosse, P.R.L., and Kowarsky, J. (1995). Testing an effluent for tainting of fish – The Latrobe Valley ocean outfall. Water (Australia). 22: 20-22.

*Muir, D.C.G., Ford, C.A., Rosenberg, F.B. Norstrom, R.J., Simon, M. and Beland, P. (1996). Persistent organochlorines in beluga whales (*delhinapterus leucas*) from the St Lawrence river estuary- I. Concentrations and patterns of specific PCBs, chlorinated pesticides and polychlorinated Dibenzo-p-dioxins and dibenzofurans. Environmental Pollution 93: 219 – 234.

*Muir,D.C.G. and Servos, M.R. (1996). Bioaccumulation of bleached kraft pulp mill related organic chemicals by fish. In “Environmental Fate and Effects of Pulp and Paper Mill Effluents”. Servos ,M.R., Munkittrick, K.R., Carey, J.H., and van der Kraak, G.J. (Ed). St Lucia Press, Delray Beach, Florida (1996).

*Müeller, J., Muller, R., Goudkamp, K., Shaw, M., Mortimer, M., Haynes, D., Paxman, C., Hyne, R., McTaggart, A., Burniston, D., Symons, R. & Moore, M. (2004). Dioxins in aquatic environments in Australia –technical report no.6. Australian Government, Department of the Environment and Heritage (DEH) May 2004 (http://www.deh.gov.au/settlements/publications/chemicals/dioxins/report-6/index.html).

*Nabholz, J., V., Miller, P., and Zeeman, M. (1993). Environmental risk assessment of new chemicals under the toxic substances control act (TSCA) section five. In 'Environmental Toxicology and Risk Assessment', ASTM STP 1179, Eds Landis, W., G., Hughes, J., S., and Lewis, M., A. American Society for Testing and Materials, Philadelphia, 1993 p40-55.

*NAS (1976). Selenium. Comm. Med. Biol. Eff. Environ Poll., Div. Med. Sci., Natl. Res. Counc., National Academy of Sciences, Washington, D.C. as cited in Eisler, R (2000). Handbook of Chemical Risk Assessment Health Hazards to Humans, Plants, and Animals. Volume 1. Metals. Chapter 31 –Selenium. Lewis Publishers. Boca Raton, Florida.

*National Marine Fisheries Service (NMFS). (1993). Data tapes for the 1993 NMFS provided to U.S. EPA, National Center for Environmental Assessments. As cited in USEPA (1997).

Nelson, P.J., Chin, W.J. and Grover, S.G. (1998). Bleaching studies on eucalypt kraft pulps. CSIRO National Pulp Mills Research Program. Technical Report Series. No. 26. Commonwealth of Australia.

*NHMRC (2002). Dioxins: Recommendation for a Tolerable Monthly Intake for Australians. National Health and Medical Research Council, Commonwealth of Australia.

Nichols, J. W. (2001). Use of indicators in ecological risk assessments for persistent, bioaccumulative toxicants. Human and Ecological Risk Assessment. 7(5): 1043-1057.

*Nishimura, M., Konishi, S., Matsunaga, K., Hata, K. and Kosuga, H. (1983). Mercury concentration in the ocean. J. Oceanogr Soc Japan 39:2951-3300 as cited in Eisler, R (2000). Handbook of Chemical Risk Assessment Health Hazards to Humans, Plants, and Animals. Volume 1. Metals. Chapter 5 –Mercury. Lewis Publishers. Boca Raton, Florida.

*Niragu, J.O. (1980). Cadmium in the Environment. Part 1: Ecological Cycling. John Wiley, NY as cited in Eisler, R. (2000). Handbook of Chemical Risk Assessment. Health Hazards to Humans, Plants, and Animals. Volume 1. Metals. Lewis Publishers, CRC Press, Boca Raton, Florida.

NSR Environmental Consultants (2001). Boyer Mill: Ecological risk assessment. Executive summary. www.nsrenv.com.au/downlaods/boyer-era.pdf

OCS (2004). Human Health Risk Assessment of Dioxins in Australia, National Dioxins Program Technical Report No. 12. Office of Chemical Safety, Australian Government Department of Health and Ageing, Australian Government Department of the Environment and Heritage, Commonwealth of Australia.

OEHHA (2000). Technical support document for exposure assessment and stochastic analysis. Office of Environmental Health and Hazard Assessment, California. http://oehha.org/air/hot_spots/pdf/Stoch4f.pdf

*Ohio EPA (2003). Guidance for Conducting Ecological Risk Assessments DERR - 00 - RR - 031 February 2003 State of Ohio Environmental Protection Agency Division of Emergency and Remedial Response. http://www.epa.state.oh.us/derr/rules/RR-031.pdf

Oikari, A., Ånäs, E., Kruzynski, G. and Holmbom, B. (1984). Free and conjugated resin acids in the bile of rainbow trout, *salmo gairdneri*. Bulletin of Environmental Contamination and Toxicology. 33: 233-240.

*Oikari, A. and Kunnamo-Ojala,T. (1987). Tracing of xenobiotic contamination in water with the aid of fish bile metabolites: A field study with caged rainbow trout *(Salmo gairdneri)*. Aquatic Toxicology 9(6):327-341. 5.1.2

*Oikari, A., Fragoso, N., Leppänen, H., Chan, T., Hodson, P.V. (2002). Bioavailability to juvenile rainbow trout (*oncorynchus mykiss)* of retene and other mixed-function oxygenase-active compounds from sediment. Environmental Toxicology and Chemistry. 21: 121-128.

* OSPAR (2002). OSPAR list of substances/compound liable to cause taint. OSPAR Convention for the Protection of the Marine Environment of the North-East Atlantic. Meeting of the Offshore Industry Committee. Annex 9.
http://www.sft.no.arbeidsomr/petroleum/dokumenter/Anex09_Taint_list.doc

*Oregon DEQ (2000). Guidance for Ecological Risk Assessment: Levels I, II, III, IV. Oregon Department of Environmental Quality Waste Management & Cleanup Division Cleanup Policy & Program Development Section http://www.deq.state.or.us/wmc/documents/eco-3.pdf

*Paasivirta, J., Knuutinene, J., Tarhanen, J., Kuokkanen, T., Surma-Aho, K., Paukku, R., Kääriäinen, H., Lahtiperä, M. and Veijanen, A. (1983). Potential off-flavour compounds from chloro-bleaching of pulp and chlorodisinfection of water. Wat Sci. Tech. 15: 97-104.

*Paasivirta, J., Klein, P., Knuutila, M., Knuutinen, J., Lahtiperä, M., Paukku, R., Veijanen, A., Welling, L., Vuorinen, M. and Vuorinen, P.J. (1987). Chlorinated anisoles and veratroles in fish. Model compounds. Instrumental and sensory determination. Chemosphere. 16: 1231-1241.

*Paasivirta, J., Rantalainen, A-L., Welling, L., Herve, S., Heinonen, P. (1992). Organochlorines as environmental tainting substances: Taste panel study and chemical analyses of incubated mussels. Wat Sci Tech. 25: 105-113.

*Pacheco, M. and Santos, M.A. (2002). Biotransformation, genotoxic, and histopathological effects of environmental contaminants in European eel *(Anguilla anguilla L.)*. Ecotoxicology and Environmental Safety 53(3):331-347.

*Palmer, F.H., Sapudar, R.A., Heath, J.A., Richard, N.J. and Bowes, G.W. (1988). Chlorinated dibenzo-p-dioxin and dibenzofuran contamination in California from chlorophenol wood preservative use. Report No. 88-5WQ, Division of Water Quality, California State Water Resources Control Board. As cited in ANZECC (2000).

*Pao, E.M., Fleming, K.H., Guenther, P.M., and Mickle, S.J. (1982). Foods commonly eaten by individuals: amount per day and per eating occasion. U.S. Department of Agriculture. Home Economics Report No. 44. As cited in USEPA (1997).

*Papoutsoglou, S.E. and Abel, P.D. (1988). Sublethal toxicity and accumulation of cadmium in *Tilipea aurea*. Bulletin of Environmental Contamination and Toxicology. 41(3): 404-411.

*Pastershank, G.M., and Muir, D.C.G. (1995). Contaminants in environmental samples: PCDDs and PCDFs downstream of bleached kraft mills, Peace and Athabasca Rivers, 1992. Northern River Basins Study Project Report 44. Northern River Basins Study, Edmonton, AB, Canada. As cited in Currie et al (2000).

*Paulsson, K., and, Lundbergh, K. (1989). The selenium method for treatment of lakes for elevated levels of mercury in fish. The Science of the Total Environment, 87-88: 495-507.

*Paulsson, K. and Lundbergh, K., (1991). Treatment of mercury contaminated fish by selenium addition. Water Air Soil Pollution, 56, 833–841. As cited in Hamilton (2004).

*Paulsson K, Lundbergh K. (2004). Selenium treatment of mercury-contaminated water systems. In: Proceedings of Selenium-Tellurium Development Association's Fifth International Symposium, Brussels, Belgium, 1994, pp. 287–290. As cited in Hamilton (2004).

Payne, S., Holsen, T. and Grimberg, S. (2002). The influence of water level fluctuations on mercury biochemical cycling. Civil and Environmental Engineering. University of Clarkson.

*Persson, P-E. (1984). Uptake and release of environmentally occurring odorous compounds by fish. Water. Res. 18: 1263-1271.

*Persson, P.E. (1988). Off-flavours in fish from waters around a pulp and paper mill in Finland. Verh. Internat. Verein. Limnol. 23: 1751-1755. As cited in Kenefick et al 1995.

*Poels, C.L.M., Fischer, R., Fukawa, K., Howgate, P., Maddock, B.G., Persoone, G., Stephenson, R.R., Bontinck, W.J. (1988). Establishment of a test guideline for the evaluation of fish tainting. Chemosphere. 17(4): 751-765.

*Pohl, H.R., Hicks, H.E., Jones, D.E., Hansen, H. and De Rosa, C.T. (2002). Public health perspectives on dioxin risks: Two decades of evaluations. Human and Ecological Risk Assessment. 8(2): 233-250.

*Popp, J.A., Crouch, R.L. and McConnell, E.E. (2006). A weight-of-evidence analysis of the cancer dose-response characteristics of 2, 3, 7, 8- tetradibenzodioxin (TCDD). Toxicological Sciences. 89(2): 361-369.

Povari, P. (2003). Sources and fate of mercury in aquatic ecosystems. Monographs of the Boreal Environment Research. 23. Finnish Environment Institute, Finland.

*Prothro (1993). as cited in ANZECC (2000). Australian and New Zealand Guidelines for Fresh and Marine Water Quality. Volume 2, Aquatic Ecosystems –Rationale and Background Information. Ch. 8.3 Toxicants. Australian and New Zealand Environment and Conservation Council. October 2000 (www.deh.gov.au/water/quality/nwqms/volume2.html)

Rand, G.M. (1995). Fundamentals of Aquatic Toxicology. Effects, Environmental Fate, and Risk Assessment. Second Edition. Taylor & Francis, Washington, D.C.

*Raspor, B. (1980). Distribution and speciation of cadmium in natural waters as cited in Nriagu, J.O. Ed. (1980). Cadmium in the environment. Part 1. Ecological Cycling. Wiley P 147-236. As cited in

Fergusson, J.E. 1990, The Heavy Elements: Chemistry, Environmental Impact and Health Effects. Pergamon Press, Headington Hill Hall, Oxford. P. 178).

*Redenbach, A.E. (1997). Sensory evaluation of fish exposed to pulp and paper mill effluent: A case study of methods used for environmental effects monitoring. Wat. Sci. Tech. 35: 357-363.

*Robberecht, H. and Von Grieken, R. (1982). Selenium in environmental waters: determination, speciation and concentration levels. Talanta 29: 823-844 as cited in Eisler, R (2000). Handbook of Chemical Risk Assessment Health Hazards to Humans, Plants, and Animals. Volume 1. Metals. Chapter 31 –Selenium. Lewis Publishers. Boca Raton, Florida.

*Roesijadi, G. (1992). Metallothioneins in metal regulation and toxicity in aquatic animals. Aquatic Toxicology. 22: 81-114.

*Rogers, I.H. (1978). Environmental effects of terpenoid chemicals: a review. J. Amer. Oil Chem. Soc. 55: 113A-118A. (As cited in McLeay 1987).

*RPDC (2004). Recommended environmental emission limit guidelines for any new bleached eucalypt kraft pulp mill in Tasmania. Volume 2, Resource Planning and Development Commission, Tasmania. August 2004.

*RPDC (2005). Proposed bleached kraft pulp mill in Northern Tasmania by Gunns Limits. Report on Gunns' Revised Project Scope and the Draft Scope Guidelines for the Integrated Impact Statement (IIS). Resources Planning and Development Commission, Hobart, Tasmania. November 2005.

*Rubinstein, N. & Wicklund, J. (1991). Dioxin contamination of sediment and marine fauna in Homebush Bay. State Pollution Control Commission, Sydney.

*Rudd, J.W.M., Turner, M.A., Townsend, B.E., Swick, A. and Furutani, A., (1980). Dynamics of selenium in mercury-contaminated experimental freshwater ecosystems. Can J Fish Aquat Sci 37: 848–857. As cited in Hamilton (2004).

*SAB (1995). Review of the Office of Research and Development's Reassessment of Dioxin and Dioxin-Like Compounds by the Dioxin Reassessment Review Committee. An EPA SAB Report: A Second Look at Dioxin. Science Advisory Board of the US EPA, September 29, 1995.

*Safe, S. (1990). Polychlorinated biphenyls (PCBs), dibenzo-p-dioxins (PCDDs), dibenzofurans (PCDFs) and related compounds: Environmental and mechanistic considerations which support the development of toxic equivalency factors (TEFs). Crit. Rev. Toxicol. 21: 51 – 58.

* Sangalang, G. B. and Freeman, H. C. (1979). Tissue uptake of cadmium in brook trout during chronic sublethal exposure. Arch Environ Contam Toxicol, 8:77-84. (As cited in Thomann et al. 1997).

*Sappington, K. G. (2002). Development of aquatic life criteria for selenium: A regulatory perspective on critical issues and research needs. Aquatic Toxicology. 57: 101-113.

*Schutz, D.F. and Turekian, K.K. (1965). The investigation of geographical and vertical distribution of several trace elements in seawater using neutron activation analysis. Geochim Cosmochim Eta

29:259-313. (As cited in ATSDR 2003). Toxicological Profile for Selenium. Agency for Toxic Substances and Disease Registry (http://www.atsdr.cdc.gov/toxprofiles/tp92.html)

*Schüürmann, G. and Markert, B. (1998). Ecotoxicology. Ecological Fundamentals, Chemical Exposure, and Biological Effects. John Wiley & Sons Inc and Spektrum Adaemischer Verlag Co-Publication.

Schwarz, M., and Appel, K.E. (2005). Carcinogenic risks of dioxin: Mechanistic considerations. Regulatory Toxicology and Pharmacology. 43: 19-34.

*Sea-Ex Australia (2005). Flathead. Australian Fish and Seafood Photographs and Information. (www.sea-ex.com/fishphotos/flathead.htm).

*Segner, H. and Braunbeck, T. (1998). Cellular response profile to chemical stress. In Ecotoxicology. Editors: G. Schüürmann and B. Markert. John Wiley & Sons, Brisbane. 521-569.

Servos, M.R., Munkittrick, K.R., Carey, J.H. and Van der Kraak, G.J. (1996). Environmental Fate and Effects of Pulp and Paper Mill Effluents. St. Lucie Press, Delray Beach, Florida.

*Servos, M.R., Muir, D.C.G., and Webster, G.R.B. (1992). Environmental fate of polychlorinated dibenzo-*p*-dioxins in lake enclosures. Canadian Journal of Fisheries and Aquatic Science. 49: 722-734. As cited in Currie et al (2000).

*Sharrif A., Ahlborg U., Axegard P., Rappe C. and van Heiningen A (1996). An Assessment of the Formation of 2378-TCDD and 2378-TCDF when Chlorine Dioxide is the Oxidizing Agent in the First Stage of Chemical Pulp Bleaching. Report prepared for the Alliance for Environmental Technology (http://www.aet.org/reports/technical/exec_sum.html#sec2).

*Shiu, W-Y., Ma, K-C., Varhackova, D. and Mackay, D. (1994). Chlorophenols and alkylphenols: A review and correlation of environmentally relevant properties and fate in an evaluative environment. Chemosphere. 29:1155-1224.

*Shumway, D.L. and Chadwick, G.G. (1971). Influence of kraft mill effluent on the flavour of salmon fish. Water Research. 5(11):997-1003.

*Shumway, D.L. and Palensky, J.R. (1973). Impairment of the flavour of fish by water pollutants. U.S. Environmental Protection Agency Report EPA-R3-73-010. Washington, D.C. As cited in Persson 1984.

*Silkworth, J.B., Koganti, K., Possolo, A., Zhao, M. and Hamiltion, S.B. (2005). Comparison of TCDD and PCB CYPIA induction sensitivities in fresh hepatocytes from human donors, Sprague-Dawley rats and rhesus monkeys and HepG2 cells. Tox. Sci. 87:508 – 519.

*Skipperud, L., Salbu, B. and Hagebo, E. (1998). Transfer, pathways, enrichment and discharge of trace elements in the pulp industry measured by means of neutron activation analysis. Science Total Environment 216:133-146.

Skipperud, L., Salbu, B. and Hagebø, E. (1998). Speciation of trace elements in discharges from the pulp industry. Science Total Environment. 217: 251-256.

*Skinner, C., Turoczy, N. J., Jones, P. J., Barnett, D. and Hodges, R. (2004). Heavy metal concentrations in wild and cultured blacklip abalone (Haliotis Rubra Leach) from southern Australian waters. Food Chem. 85, 351-356.

Smith, J.W. and Sprague, J.B. (1996). Compatibility of pulping effluents with the aquatic environment. Kraft mill effluent need not harm the receiving water. Pulp & Paper Canada 93(6): 53 – 57.

Solomon, K., Bergman, H., Huggett, R., Mackay, D. and McKague, B. (2005). A review and assessment of the ecological risks associated with the use of chlorine dioxide for the bleaching of pulp. Alliance for Environmental Technology. Accessed 30th November 2005. http://www.aet.org/scienc_of_ecf/eco_risk/risk1.html.

*Stewart, F.M., Furness, R.W. and Monteiro, L.R. (1996). Relationships between heavy metal and metallothionein concentrations in lesser black-backed gulls, *Larcus fuscus*, and Cory's shearwater, *Calonectris diomedea*. Archives of Environmental Contamination and Toxicology. 30(3): 299-305.

*Stromberg, L., Morck, R., de Sousa F. and Dahlman, O. (1996). Effects of internal process changes and external treatment on effluent chemistry. In “Environmental Fate and Effects of Pulp and Paper Mill Effluents”. Servos ,M.R., Munkittrick, K.R., Carey, J.H., and van der Kraak, G.J. (Ed). St Lucie Press, Delray Beach, Florida.

*Stuthridge, T.R., Anderson, S.M., Gifford, J.S., Robinson, M.J. and Straus, D.L. (1997). Bioaccumulation of dehydroabietic acid in rainbow trout *(onchorhynchus mykiss)*: Role of biotransformation enzymes. Water Science and Technology. 35(2-3):365-372.

*Süss, H.U. (2002). Bleaching agents. In “Ulmann’s Encylopedia of Industrial Chemistry”, Wiley-VCH Verlag GmbH & Co.

*Swabey, Y.H. (1965). Report on a study of the flavour of fish from the Spanish river and adjacent North Channel. Ontario Water Resources Commission. Toronto, Ontario. (As cited in Kenefick et al. 1995).

*Tavendale, M.H., McFarlane, P.N., Mackie, K.L., Wilkins, A.L. and Langdon, A.G. (1997). The fate of resin acids-1. The biotransformation and degradation of deuterium labelled dehydroabietic acid in anaerobic sediments. Chemosphere 35(10):2137-2151.

*Thomann, R. V., Shkreli, F. and Harrison, S. (1997). A pharmacokinetic model of cadmium in rainbow trout. Environmental Toxicology Chemistry. 16(11): 2268-2274.

Thompson, G.,B., Chapman, J.,C. & Richardson, B.,J. (1992). Disposal of hazardous wastes in Australia: Implications for marine pollution. Marine Pollution Bulletin: 25, 155−162.

Toda, E. (2004). Draft emission scenario on pulp and paper industry kraft pulp mills. Organisation for Economic Co-operation and Development, Vienna.

*Tsang, A.M. and Klepeis, N.E. (1996). Results tables from a detailed analysis of the National Human Activity Pattern Survey (NHAPS) response. Draft Report prepared for the U.S.

Environmental Protection Agency by Lockheed Martin, Contract No. 68-W6-001, Delivery Order No. 13. (As cited in US EPA 1997).

*Turner, M.A. and Rudd, J.W.M. (1983). The English-Wabigoon River system: III. Selenium in lake enclosures: its geochemistry, bioaccumulation, and ability to reduce mercury bioaccumulation. Can J Fish Aquatic Science: 40, 2228–2240. As cited in Hamilton (2004).

*Turner, M.A. and Swick, A.L. (1983). The English-Wabigoon River system: IV. Interaction between mercury and selenium accumulated from waterborne and dietary sources by northern pike (Esox lucius). Can J Fish Aquatic Science: 40, 2241–2250. As cited in Hamilton (2004).

*UK (1996). Risk Assessment of Dioxin Releases from Municipal Waste Incineration Processes. Her Majesty's Inspectorate of Pollution. Department of the Environment, HIMP – Commissioned Research. DOE Reference: HMIP/CPR2/41/1/181.

Ullrich, S. M., Tanton, T. W. and Abdrashitova, S. A. (2001). Mercury in the aquatic environment: a review of factors affecting methylation. Critical Reviews in Environmental Science and Technology 31(3): 241-293. (Abstract only).

*UNEP (2002). Global mercury assessment. Inter-Organizational Programme for the Sound Management of the Environment, United Nations Environment Programme (UNEP).

* US EPA (1989). Risk Assessment Guidance for Superfund. Volume I: Human Health Evaluation Manual (Part A). Washington, DC: U.S. Environmental Protection Agency, Office of Emergency and Remedial Response. EPA/540/1-89/001.

*US EPA (1984). Ambient water quality criteria for 2,3,7,8-tetrachlorodibenzo-p-dioxin, EPA, Washington DC. As cited in ANZECC (2000).

*US EPA (1995). Final Water Quality Guidance for the Great Lakes System. Final Water Quality Guidance for the Great Lakes System Final Rule. Federal Register: March 23, 1995, Page 15365-15366. 40 CFR 9, 122, 123, 131, and 132.

*US EPA (1997). Press Release Titled *"EPA Eliminates Dioxin, Reduces Air And Water Pollutants From Nation's Pulp And Paper Mills"* For Release: Friday, November 14, 1997 United States Environmental Protection Agency
http://yosemite.epa.gov/opa/admpress.nsf/b1ab9f485b098972852562e7004dc686/cec7ccca2be74f2e8525654f00682f23?OpenDocument

*US EPA (1997b). Exposure Factors Handbook. Volume 1: General Factors. EPA/600/8-89/043, May 1989. Update to Exposure Factors Handbook. EPA/600/P-95/002Fa, August 1997. Office of Research and Development, National Center for Environmental Assessment, U.S. Environmental Protection Agency.

*US EPA (1998). National Emission Standards for Hazardous Air Pollutants for Source Category: Pulp and Paper Production; Effluent Limitations Guidelines, Pretreatment Standards, and New Source Performance Standards: Pulp, Paper, and Paperboard Category. Federal Register: April 15, 1998, 63(72):18503-18552.

*US EPA (2000a). Methodology for deriving ambient water quality criteria for the protection of human health (2000). Office of Water. U.S. Environmental Protection Agency. Report No: EPA-822-B-00-004.

*US EPA (2000b). Estimation program interface (EPI) suite. U.S. Environmental Protection Agency Office of Pollution Prevention Toxics and Syracuse Research Corporation. Washington, D.C.

*US EPA (2000c). Exposure and Health Assessment for 2,3,7,8 – Tetrachlorodibenzo-p-dioxin (TCDD) and Related Compounds. National Centre for Environmental Assessment Office of Research and Development, U.S. Environmental Protection Agency.

*US EPA (2000d). Exposure and human health re-assessment for 2,3,7,8 – tetrachlorodibenzo-p-dioxin (TCDD) and related compounds. Draft final report. Office of Research and Development. U.S. Environmental Protection Agency. Washington, D.C. Report Number: EPA 600/P-00/001 Bc. As cited in US EPA (2005).

*US EPA (2001). Water quality criterion for the protection of human health: methylmercury. Office of Science and Technology, Office of Water, U.S. Environmental Protection Agency. Washington.

*US EPA (2004a). Exposure and Health Assessment for 2,3,7,8 – Tetrachlorodibenzo-p-dioxin (TCDD) and Related Compounds. National Centre for Environmental Assessment Office of Research and Development, U.S. Environmental Protection Agency. National Academy of Sciences Review Draft.

US EPA (2004b). Framework for inorganic metals risk assessment. Peer review draft. Risk Assessment Forum, U.S. Environmental Protection Agency. Washington.

US EPA (2005). Fish tissue criterion for methylmercury to protect human health document. Water Quality Criteria, Office of Water, U.S. Environmental Protection Agency. Washington.

*US EPA (2005a). Human health risk assessment protocol for hazardous waste combustion facilities. Volume 2 Appendix B and also HHRAP companion database. U.S. Environmental Protection Authority. Washington, D.C. (http://www.epa.gov/epaoswer/hazwaste/combust/riskvol.htm#volume2).

*US EPA (2005b). Current national recommended water quality criteria. U.S. Environmental Protection Agency. Last updated 26th May 2005. Accessed December 2005. http://www.epa.gov/waterscience/criteria/wqcriteria.html .

*US EPA (2005c). Human health risk assessment protocol for hazardous waste combustion facilities. Volume 2 Appendix C. U.S. Environmental Protection Authority. Washington, D.C. (http://www.epa.gov/epaoswer/hazwaste/combust/riskvol.htm#volume2).

*van den Berg, M., Birnbaum, L., Bosveld, A. T. C., Brunstrom, B., Cook, P., Feeley, M., Giesy, J.P., Hanberg, A., Hasegawa, R., Kennedy, S.W., Kubiak, T., Larsen, J.C., van Leeuewen, F.X.R., Liem, A.K.D., Nolt, C., Peterson, R.E., Poellinger, L., Safe, S., Schrenk, D., Tillitt, D., Tysklind, M., Younes, M., Waern, F. and Zacharewski, T. (1998). Toxic Equivalency Factors (TEFS) for PCBs, PCDDs, PCDFs for Humans and Wildlife. Environ. Health Persp. 106: 775-792.

Van der Oost, R., Beyer, J., Vermeulen, N.P.E. (2003). Fish bioaccumulation and biomarkers in environmental risk assessment: a review. Environmental Toxicology and Pharmacology. 13: 57-149. Abstract only.

van Leeuwen, F.X.R., Feeley, M., Schrenk, D., Larsen, J.C., Farland, W., and Younes, M. (2000). Dioxins: WHO's tolerable daily intake (TDI) revisited. Chemosphere. 40: 1095-1101.

*Verta, M., Ahtiainen, J., Nakari, T., Langi, A., and Talka, E. (1996). The effect of waste constituents on the toxicity of TCF and ECF pulp bleaching effluents. In Environmental Fate and Effects of Pulp and Paper Mill Effluents. Servos , M.,R., Munkittrick, K.,R., Carey, J.,H., and van der Kraak, G.,J. (Ed). St Lucie Press, Delray Beach, Florida, 1996.

*Wallis, A., Smith, T., and Wearne R. (1993a). Characterisation of the major components (by mass) of selected effluent from bleaching eucalypt kraft pulp. Technical Report Series No. 10. National Pulp Mills Research Program CSIRO Division of Forest Products.

Wallis, A. (1994). Characterisation of high molecular weight material in filtrates from bleaching eucalypt kraft pulps. Technical Report Series No. 4. National Pulp Mills Research Program. CSIRO Division of Forest Products, Commonwealth of Australia: 23.

*Wallis, A., Smith, T., and Wearne R. (1993b). Determination of chlorinated phenols in effluent from bleached eucalypt kraft mills. Technical Report Series No. 2. National Pulp Mills Research Program. CSIRO Division of Forest Products, Commonwealth of Australia.

Wallis, A.F.A. (1995). A review of the organic extractives in eucalypt woods for pulp production. Technical Report Series. No. 15. National Pulp Mills Research Program. CSIRO Division of Forest Products, Commonwealth of Australia.

*Wan, Y., Hu,J., Yang, M. An, L., An, W., Jin, X., Hattori, T. and Itoh, M. (2005). Characterisation of trophic transfer for polychlorinated Dibenzo-p-dioxins, dibenzofurans, non- and mono-ortho polychlorinated biphenyls in the marine food web of Bohai Bay, North China. Envir. Sci.and Technol. 39: 2417-2425.

* Watras, C., J., and Bloom, N., S. (1992). Mercury and methylmercury in individual zooplankton: implication for bioaccumulation. Limnology and Oceanography 37: 1313-1318.

Webster, E., Cowan-Ellsberry, E. and McCarty, L. (2004). Putting science into persistence bioaccumulation and toxicity evaluations. Environmental Toxicology and Chemistry. 23(10): 2473-2482.

*Weinbauer, J.D., Thiel, D.A., Kaczynski, V.W., and Martin, C.S. (1980). Receiving stream fisheries studies relative to secondary treated pulp mill effluents. TAPPI. 63(10): 121-125.

*Wells, D.L. (1967). Report on the flavour of whitefish from Nipigon Bay (1967). Ontario Water Resources Commission. Toronto, Ontario. As cited in Kenefick et al. (1995).

*West, P.C., Fly, M.J., Marans, R. and Larkin, F. (1989). Michigan sport anglers fish consumption survey. A report to the Michigan Toxic Substance Control Commission. Michigan Department of Management and Budget Contract No. 8720141. (As cited in US EPA 1997).

*West, P.C., Fly, J.M. Marans, R., Larkin, F. and Rosenblatt, D. (1993). 1991-92 Michigan sport anglers fish consumption study. Prepared by the University of Michigan, School of Natural Resources for the Michigan Department of Natural Resources, Ann Arbor, MI. Technical Report No. 6. May. (As cited in US EPA 1997).

*Whittle, D.M. and Flood, K.W. (1977). Assessment of acute toxicity, growth impairment, and flesh tainting potential of a bleached kraft mill effluent on rainbow trout (*Salmo gairdneri).* J. Fish. Res. Boar. Can. 34: 869-878.

*Whittle, K.J., Hardy, R., Holden, A.V., Johnston, R. and Pentreath, R.J. (1977). Occurrence and fate of organic and inorganic contaminants in marine animals. Ann. N.Y. Acad. Sci 298:47-79. (As cited in Eisler, R (2000). Handbook of Chemical Risk Assessment Health Hazards to Humans, Plants, and Animals. Volume 1. Metals. Chapter 31 –Selenium. Lewis Publishers. Boca Raton, Florida).

*Wiener J., G., & Spry D., J. (1996). Toxicological significance of mercury in freshwater fish. Chapter 13 in Environmental contaminants in wildlife: Interpreting tissue concentrations, ed WN Beyer, GH Heinz & AW Redmon-Norwood, CRC Press, Boca Raton, FL, 297−339. (As cited in UNEP 2002).

*WHO (1989). Environmental Health Criteria No. 86 -Mercury environmental aspects. World Health Organization, Geneva.

*WHO (1998). Assessment of the Health Risks of Dioxins: Re-evaluation of the Tolerable Daily Intake (TDI). Executive summary of the WHO Consultation, May 25 – 29, Geneva, Switzerland.

WHO (2000). Air Quality Guidelines for Europe. 2nd Edition. World Health Organization Regional Publications, European Series Number 91. http://www.euro.who.int/document/e71922.pdf

*WHO (2003). Guidelines for safe recreational water environments. Volume 1: Coastal and fresh waters. World Health Organization. http://www.who.int/water_sanitation_health/bathing/srwg1.pdf

*WHO (2004). Guidelines for drinking-water quality. 3rd edition. Volume 1: Recommendations. World Health Organization. http://www.who.int/water_sanitation_health/dwq/GDWQ2004web.pdf

*Wicklund, A., Runn, P. and Norrgren, L. (1988) Cadmium and zinc interactions in fish – Effects of zinc on the uptake, organ distribution, and elimination of CD-109 in the zebrafish, *Bracydanio rerio*. Archives of Environmental Contamination and Toxicology. 17(3): 345-354. (As cited in de Conto Cinier et al. 1999).

Wilson, S. K., Murray, J. L. and Huntington, H. P. (1998). Assessment report: Arctic pollution issues. Arctic Monitoring and Assessment Programme. Oslo.

Wilson, N. D., Price, P. S. and Paustenbach, D. J. (2001). An event-by-event probabilistic methodology for assessing the health risks of persistent chemicals in fish: A case study at the Palos Verdes shelf. J Toxicology Environmental Health Part A. 62: 595-642.

Wong, C. S., Capel, P. D. and Nowell, L. H. (2001). National-scale, field-based evaluation of the biota-sediment accumulation factor model. Abstract only. Environmental Science Technology. 35: 1709-1715.

*Yang, H.N. and Chen, H.C. (1996). Uptake and elimination of cadmium by Japanese eel, *Anguilla japonica*, at various temperatures. Bulletin of Environmental Contamination and Toxicology. 56: 670-676.

*Yaws, C.L. (2003). Yaws handbook of thermodynamic and physical properties of chemical compounds. Electronic Book, Knovel. Release date February 1, 2003.

*Yearsley, G.K., Last, P.R. and Ward, R.D. (1999). Australian Seafood Handbook. An identification guide to domestic species. CSIRO Marine Research, Australia.

Yu, M.-H. (2005). Environmental toxicology. Biological and health effects of pollutants. CRC Press, London.

Zhuang, W., McKague, B., Reeve, D. and Carey, J. H. (2003). Identification and confirmation of traces of chlorinated fatty acids in fish downstream of bleached kraft pulp mills by gas chromatography with halogen-specific detection. Journal of Chromatography A. 994: 137-157.

Appendix 1: What's in the Bell Bay effluent?

A1.1 Historical candidate list of effluent constituents

Whether kraft mills are processing softwood or hardwood, a vast number of chemicals have been identified in mill effluent over the last two decades. However many are naturally found in wood, or their breakdown products, and are likely to be in effluent only at tiny concentrations. While RPDC (2004) have stipulated performance criteria for effluent these criteria do not necessarily account for the endpoints (bioaccumulation and human health, fish tainting, water recreation and human health) being addressed in this assessment. For this risk assessment there was therefore a need to determine more comprehensively what chemicals might be present, and at what concentrations, in the effluent from the Bell Bay mill. Patently this is a difficult exercise since the mill has not yet been constructed and there is no effluent to be analysed. In addition the mill is designed to be state of art and therefore data from other mills not representative of the Bell Bay mill effluent.

To address these problems Toxikos conducted a non-exhaustive literature search and used the information, primarily from reviews[53], to identify substances commonly reported to be in effluent. This was done regardless of the age, bleaching or effluent treatment processes of the mill. The philosophy behind this approach was to capture and address as many of the effluent constituents as possible that historically have been found in kraft mill effluents and/or have been perceived to be an issue for human health. The process yielded a *'candidate list of chemicals'* for the Bell Bay effluent (Tables A1.1 & A1.2). Jaakko Pöyry Oy assisted in this task. The *'candidate list of chemicals'* was then culled according to likelihood of a particular chemical being in Bell Bay effluent to create a *'chemicals of interest'* list for the Bell Bay mill (Table A1.3). Again Jaakko Pöyry Oy assisted and also provided estimates of final effluent concentrations for major organic classes and individual metals (JP 2005 c,d,e).

Effluent constituents and quantities are affected by mill-to-mill differences in process technology and operations, differences in wood types and sources, plus chemical interactions among the different waste streams that may be specific for a given mill. There is therefore uncertainty in

[53] For example LaFleur (1996), Bright et al. (2003), Dahlman et al. (1995), Sunito et al. (1988), Strömberg et al. (1996).

predicting the composition of the Bell Bay effluent. Similarly the predictions of effluent constituent concentrations are uncertain and Jaakko Pöyry advise that concentrations are over predictions. The uncertainty in concentration of chemical concentration has been further addressed in the quantitative portions of this risk assessment by assuming very conservative (erring on the side of over prediction) concentrations for individual effluent components (see Section A1.3).

Table A1.1: *'Candidate chemicals'* historically identified in pulp mill effluent

Chemical	Reference [a]	Chemical	Reference [a]
Primary Discharge		**Other aromatic compounds**	
Absorbable organic halide AOX	RPDC (2004)	**Sterols**	
Biological oxygen demand (BOD_5)	RPDC (2004)	beta-sitostanol	Verta et al (1996)
Chemical oxygen demand (COD)	RPDC (2004)	beta-sitosterol	Verta et al (1996)
Chlorate	RPDC (2004)	Fucosterol	Guttiérrez et al (2001)
Colour	RPDC (2004)	Betulinol	Verta et al (1996)
pH	JP (2005b)	Campesterol	Verta et al (1996)
Total dissolved solids (TDS)	JP (2005b)	Stigmasterol	Bright et al (2003)
Total organic carbon (TOC)	JP (2005b)	Squalene (steroid hydrocarbon)	Guttiérrez et al (2001)
Total suspended solid	RPDC (2004)	**Miscellaneous aromatics**	
		2,4-Dimethylphenol	JP (2005a)
High molecular weight substances	JP (2005a)	2-Methylphenol	JP (2005a)
		2-Nitrophenol	JP (2005a)
Metals		3-Methylphenol	JP (2005a)
Aluminium	JP (2005a)	4-Methylphenol	JP (2005a)
Antimony	JP (2005e)	Phenol	JP (2005a)
Arsenic (III &V)	JP (2005a)	Dichloroacetovanillone	LaFleur (1996)
Barium	JP (2005b)	a-pinene	Bright et al (2003)
Beryllium	JP (2005e)	Benzene	Bright et al (2003)
Boron	JP (2005a)	b-pinene	Bright et al (2003)
Cadmium	JP (2005a)	1,2,4-Trichlorobenzene	Hewitt et al (1996)
Calcium	JP (2005b)	Camphene	Bright et al (2003)
Chromium (III & VI)	JP (2005a)	Dehydrojuvabione	Bright et al (2003)
Cobalt	JP (2005a)	Juvabione	Bright et al (2003)
Copper	JP (2005a)	Dichloromethylene furanones	Bright et al (2003)
Iron	JP (2005b)	Chlorodimethyl naphtalenes	Bright et al (2003)
Lead	JP (2005a)	Alkylchlorophenanthrenes	Bright et al (2003)
Magnesium	JP (2005b)	4-chloro-3-hydroxy-2H-pyran-2-one	Bright et al (2003)
Manganese	JP (2005a)	5,5-dichloro-6,6-dihydroxy-2-methoxy-2-cyclohexene-1,4-dione	Bright et al (2003)

Chemical	Reference [a]
Mercury	JP (2005a)
Molybdenum	JP (2005a)
Nickel	JP (2005a)
Selenium	JP (2005a)
Silicon	JP (2005e)
Sodium	JP (2005a)
Tin	JP (2005a)
Vanadium	JP (2005e)
Zinc	JP (2005a)
Non metallic inorganics	
Ammonia	JP (2005a)
Chloride	JP (2005b)
Hydrogen sulphide	JP (2005a)
Nitrate	JP (2005a)
Sulphate	JP (2005a)
Thiolignin	JP (2005a)
Thiosulphate	JP (2005a)
Organic acids	
Chloroacetic acids	
Monochloroacetic acid	EA (1998)
Dichloroacetic acid (including methyl ester)	Bright et al (2003), EC (1991)
Trichloroacetic acid	Bright et al (2003)
Resin acids	
14-chloro dehydroabeitic acid	Hewitt et al (1996)
12,14-dichloro hydroabietic acid	Hewitt et al (1996)
Abietine	Verta et al (1996)
Arakine	Verta et al (1996)
Chlororetene	Bright et al (2003)
dehydroabietic acid	Verta et al (1996)
Fichtelite	Bright et al (2003), EC (1991)
Isopirame	Verta et al (1996)
Levopirame	Verta et al (1996)
Neoabietine	Verta et al (1996)

Chemical	Reference [a]
Dichloro protocatechualdehyde	LaFleur (1996)
3-methoxy-5-dichloromethylene -2(H5)furanone	Bright et al (2003)
Aniline	JP (2005a)
2,4-Dichloroaniline	JP (2005a)
2,5-Dichloroaniline	JP (2005a)
3,4-Dichloroaniline	JP (2005a)
2,4,6-trichloroanisole	Bright et al (2003)
3-methoxycatechol	LaFleur (1996)
p-cymene	Bright et al (2003)
p-hydroxybenzaldehyde	LaFleur (1996)
Syringol	Gutiérrez et al (2001)
Dioxins (PCDD/PCDFs not PCBs or PCBB/PCBFs) TEQ (refer notes below)	RPDC (2004)
Miscellaneous	
Hydrocarbons	
Methanol	EA (1998)
Ethanol	JP (2005c)
3-carene	Bright et al (2003)
Carbon disulphide	EA (1998)
Hexane (C6)	Bright et al (2003)
Pentadecane (C15)	EC (1991)
Octadecane (C18)	Hewitt et al (1996)
Nonadecane (C19)	Hewitt et al (1996)
cosane (C20)	Hewitt et al (1996)
heneicosane (C21)	Hewitt et al (1996)
eicosane (22)	Hewitt et al (1996)
tricosane (C23)	Hewitt et al (1996)
tetracosane (C24)	Hewitt et al (1996)
pentacosane (C25)	Hewitt et al (1996)
Limonene	Bright et al (2003)
Styrene	Bright et al (2003)
Toluene	Bright et al (2003)
m-&p-xylene	Bright et al (2003)
o-xylene	Bright et al (2003)

Chemical	Reference [a]	Chemical	Reference [a]
Palustrine	Verta et al (1996)	**Aldehydes & Ketones**	
pimaric acid	Hewitt et al (1996)	Acetaldehyde	Bright et al (2003)
Retene	Bright et al (2003)	Pentanal	Bright et al (2003)
Trichloropterostilbene	Hewitt et al (1996)	Hexanal	Bright et al (2003)
Fatty acids		Heptanal	Bright et al (2003)
(2E,4Z)-Hexadienedioic acid monomethyl ester	Bright et al (2003)	Octanal	Bright et al (2003)
Behene	Verta et al (1996)	Nonanal	Bright et al (2003)
Lignoserine	Verta et al (1996)	Acetone	Bright et al (2003)
Linoleine	Verta et al (1996)	1,1-dichloroacetone	LaFleur (1996)
Myristine	Verta et al (1996)	Pentachloroacetone	Bright et al (2003)
Oleine	Verta et al (1996)	2-butanone	Bright et al (2003)
Palmitine	Verta et al (1996)	2-pentanone	Bright et al (2003)
Stearine	Verta et al (1996)	2-hexanone	Bright et al (2003)
Linoleic acid	Bright et al (2003)	2-heptanone	Bright et al (2003)
		2-octanone	Bright et al (2003)
Chlorinated natural phenolics		2-nonanone	Bright et al (2003)
4-chlorocatechol	Bright et al (2003)	Hydroquinone	Bright et al (2003)
3,4-dichlorocatechol	Bright et al (2003)	4-methyl-2-pentanone	Bright et al (2003)
3,5-dichlorocatechol	EC (1991)	Dichlorocyclohexendione	Bright et al (2003)
3,4,5-trichlorocatechol	EC (1991)	**Trihalomethanes**	
Tetrachlorocatechol	EC (1991)	Bromodichloromethane	JP (2005a)
2-chloro-p-cymene	Bright et al (2003)	Bromoform	JP (2005a)
5-chloro-o-cymene	Bright et al (2003)	Chloroform	RPDC (2004)
2,3-dichloro-p-cymene	Bright et al (2003)	Dibromohloromethane	JP (2005a)
2,5-dichloro-p-cymene	Hewitt et al (1996)	**Chlorinated Hydrocarbons**	
2,6-dichloro-m-cymene	Bright et al (2003)	Chloromethane	EA (1998)
2,3,6-trichloro-p-cymene	Bright et al (2003)	Dichloromethane	Bright et al (2003)
Tetrachloro-p-cymene	Bright et al (2003)	Carbon tetrachloride	Bright et al (2003)
4-chloroguaiacol	Bright et al (2003)	1,1-Dichloropropane	JP (2005a)
3,4-dichloroguaiacol	Hewitt et al (1996)	1,3-Dichloropropane	JP (2005a)
4,5-dichloroguaiacol	Hewitt et al (1996)	1,1,1-Trichloroethane	JP (2005a)
4,6-dichloroguaiacol	Hewitt et al (1996)	1,1,2-Trichloroethane	JP (2005a)
3,4,5-Trichloroguaiacol	EC (1991)	1,1,2,2-Tetrachloroethane	JP (2005a)
4,5,6-Trichloroguaiacol	EC (1991)	Pentachloroethane	JP (2005a)
Tetrachloroguaiacol	EC (1991)	Hexachloroethane	JP (2005a)
2-chloroaceto guaiacones	Wallis et al (1993b)	Chloroethylene	JP (2005a)
5-chloroaceto guaiacones	Wallis et al (1993b)	1,1-Dichloroethylene	JP (2005a)
6-chloroaceto guaiacones	Wallis et al (1993b)	1,2-Dichloroethylene (cis & trans)	JP (2005a)
2-Monochloro syringaldehyde[c]	Wallis et al (1993b)	1,3-dichloropropene (cis & trans)	JP (2005a)
2,6-dichloro	Wallis et al (1993b)	Trichloroethylene	JP (2005a)

Chemical	Reference [a]	Chemical	Reference [a]
syringaldehyde[c]			
2-Chloro-3,4,5-trimetoxybenzaldehyde	Wallis et al (1993b)	Tetrachloroethylene	JP (2005a)
2,6-Dichloro-3,4,5-trimetoxybenzaldehyde	Wallis et al (1993b)	3-chloropropene	JP (2005a)
4,5,6-trichloro trimethoxybenzene	EC (1991)	1,1-dichlorodimethyl sulfone	Bright et al (2003)
2-chlorovanillin	Wallis et al (1993b)	1,1,3-trichlorodimethyl sulfone	EC (1991)
5-chlorovanillin	Bright et al (2003)	Chlorohydroxypyrone[b]	Wallis et al (1993a)
6-chlorovanillin	Bright et al (2003)	tetrachlorothiophene	Bright et al (2003)
2,5-dichlorovanillin	Wallis et al (1993b)	Hexachlorocyclo pentadiene	Hewitt et al (1996)
2,6-dichlorovanillin	Wallis et al (1993b)		
5,6-dichlorovanillin	Hewitt et al (1996)		
2,5,6-trichlorovanillin	Wallis et al (1993b)		
Dichloroveratrole	EC (1991)		
3,4,5-Trichloroveratrole	Bright et al (2003)		
1,2,3,4-tetrachloro-5,6-veratrole	EC (1991)		
Chlorophenols			
2-Monochlorophenol	JP (2005a)		
3-Monochlorophenol	JP (2005a)		
4-Monochlorophenol	JP (2005a)		
2,3-Dichlorophenol	JP (2005a)		
2,4-Dichlorophenol	JP (2005a)		
2,5-Dichlorophenol	JP (2005a)		
2,6-Dichlorophenol	JP (2005a)		
3,4-dichlorophenol	JP (2005a)		
3,5-dichlorophenol	JP (2005a)		
2,3,4-trichlorophenol	JP (2005a)		
2,3,5-trichlorophenol	JP (2005a)		
2,3,6-trichlorophenol	JP (2005a)		
2,4,5-trichlorophenol	JP (2005a)		
2,4,6-trichlorophenol	JP (2005a)		
2,3,4,5-tetrachlorophenol	JP (2005a)		
2,3,4,6-tetrachlorophenol	JP (2005a)		
2,3,5,6-tetrachlorophenol	JP (2005a)		
Pentachlorophenol	JP (2005a)		
4-Chloro-3-methylphenol	JP (2005a)		

[a] Hierarchical reference system consisting of RPDC 2004, JP 2005a, JP 2005b, Wallis et al. 1993a, Wallis et al. 1993b, Bright et al. 2003, EA 1998, EC 1991, LaFleur 1996, Verta et al. 1996, Hewitt et al. 1996, JP 2005c, JP 2005e.

[b] Wallis et al. (1993a) characterised the major components by mass of effluent from experimental bleaching eucalypt kraft pulp. The article states major lipophilic compounds identified in the experimental bleaching filtrates were those which have previously been identified in filtrates from bleaching pulps from other wood species.

[c] Noted by Wallis et al. (1993b) as the major chlorinated phenols in bleached (ECF) eucalypt pulp effluent.

A1.2 Candidate list of effluent constituents for Bell Bay mill

From the *'historical/candidate list of chemicals'* (Tables A1.1) that might be potentially present in the effluent of the Bell Bay mill the following groups of compounds were removed to derive a list of *'chemicals of interest'* (Table A1.3). Table A1.1 is reproduced as Table A1.2 but with the rationale for why some chemicals are not of interest for the Bell Bay mill.

The compounds removed from Tables A1.1 & A1.2 were:

- Water Quality Parameters because they are not relevant, or amenable, for the human health risk assessment of effluent discharge to the ocean.
- Polychlorinated compounds (i.e. tri-, tetra- etc) are considered to be 'virtually eliminated' on the basis of the chemistry of the bleaching process (Bright et al 2003, EC 1991)(see also Section 4.2). Strömberg et al. (1996) found very small amounts of chlorinated phenolic compounds in ECF effluents and these were only monochlorinated and dichlorinated compounds, no tri- or tetra chlorinated phenolic compounds were detected. Polychlorinated dioxins were not removed from the *'historical/candidate'* list for two reasons. The first is that they have been detected in ECF pulp mill effluent albeit at very low, assumed background, concentrations (Strömberg et al. 1996). The second is that the potential presence of dioxins in effluent is of such concern to many stakeholders that it is prudent to retain them on the list of chemicals of interest.
- Amongst the polychlorinated compounds taken off the candidate list chloroform ($CHCl_3$) deserves special mention because RPDC (2004) has requested it be measured in effluent. During Cl_2 bleaching chloroform can be formed during both the acidic and basic stages of bleaching. Some older mills used to have a hypochlorite bleaching stage and removal of this stage has radically reduced the amount of chloroform in effluent. Furthermore chloroform is effectively removed in effluent treatment plants, most likely by evaporation (Strömberg et al. 1996).
- Brominated compounds because there is not a source of reactive bromine for these substances to be created. They were included on the *'candidate list'* because they were referenced by RPDC as requiring monitoring (RPDC 2004).
- Chlorate was not detected in any of the biologically treated effluents examined by Strömberg et al. (1996). Apparently chlorate is very effectively converted to chloride in the anoxic zones which precede aerobic effluent treatments.

Table A1.2: *'Candidate chemicals'* for Bell Bay final effluent

Substances historically identified in effluent	Screened from candidate list of chemicals based on [a]:			Chemical of Interest[b]
	High MW	Bleaching & pulping process	Not relevant for present assessment	
Primary Discharge				
Adsorbable organic halide AOX			✓[a]	**N**[b]
Biological oxygen demand (BOD_5)			✓	**N**
Chemical oxygen demand (COD)			✓	**N**
Chlorate			✓	**N**
Colour			✓	**N**
pH			✓	**N**
Total dissolved solids (TDS)			✓	**N**
Total organic carbon (TOC)			✓	**N**
Total suspended solid			✓	**N**
High molecular weight substances	✓[a]			**N**
Metals				
Aluminium				**Y**[b]
Antimony				**Y**
Arsenic (III &V)				**Y**
Barium				**Y**
Beryllium				**Y**
Boron				**Y**
Cadmium				**Y**
Calcium			✓	**N**
Chromium (III & VI)				**Y**
Cobalt				**Y**
Copper				**Y**
Iron				**Y**
Lead				**Y**
Magnesium			✓	**N**
Manganese				**Y**
Mercury				**Y**
Molybdenum				**Y**
Nickel				**Y**
Selenium				**Y**
Silicon				**Y**
Sodium			✓	**N**

Substances historically identified in effluent	Screened from candidate list of chemicals based on [a]:			Chemical of Interest[b]
	High MW	Bleaching & pulping process	Not relevant for present assessment	
Tin				Y
Vanadium				Y
Zinc				Y
Non metallic inorganics				
Ammonia				Y
Chloride			✓	N
Hydrogen sulphide				Y
Nitrate				Y
Sulphate			✓	N
Thiolignin				Y
Thiosulphate				Y
Organic acids[e]				
Chloroacetic acids[e]				
Monochloroacetic acid[c]				Y
Dichloroacetic acid (includes methyl ester)				Y
Trichloroacetic acid				Y
Resin acids[e]				
14-chlorodehydroabeitic acid				Y
12,14-dichlorohydroabietic acid				Y
Abietine				Y
Arakine				Y
Chlororetene				Y
dehydroabietic acid				Y
Fichtelite				Y
Isopirame				Y
Levopirame				Y
Neoabietine				Y
Palustrine				Y
pimaric acid				Y
Retene				Y
Trichloropterostilbene		✓[a]		N
Fatty acids[d,e]				
(2E,4Z)-Hexadienedioic acid monomethyl ester				Y
Behene				Y
Lignoserine				Y
Linoleine				Y
Myristine				Y
Oleine				Y

Substances historically identified in effluent	Screened from candidate list of chemicals based on [a]:			Chemical of Interest[b]
	High MW	Bleaching & pulping process	Not relevant for present assessment	
Palmitine				Y
Stearine				Y
Linoleic acid				Y
Chlorinated natural phenolics[e]				
4-chlorocatechol				Y
3,4-dichlorocatechol				Y
3,5-dichlorocatechol				Y
3,4,5-trichlorocatechol		✓		N
Tetrachlorocatechol		✓		N
2-chloro-p-cymene				Y
5-chloro-o-cymene				Y
2,3-dichloro-p-cymene				Y
2,5-dichloro-p-cymene				Y
2,6-dichloro-m-cymene				Y
2,3,6-trichloro-p-cymene		✓		N
Tetrachloro-p-cymene		✓		N
4-chloroguaiacol				Y
3,4-dichloroguaiacol				Y
4,5-dichloroguaiacol				Y
4,6-dichloroguaiacol				Y
3,4,5-Trichloroguaiacol		✓		N
4,5,6-Trichloroguaiacol		✓		N
Tetrachloroguaiacol		✓		N
2-chloroacetoguaiacones				Y
5-chloroacetoguaiacones				Y
6-chloroacetoguaiacones				Y
2-Monochlorosyringaldehyde				Y
2,6-dichlorosyringaldehyde				Y
2-Chloro-3,4,5-trimetoxybenzaldehyde				Y
2,6-Dichloro-3,4,5-trimetoxybenzaldehyde				Y
4,5,6-trichlorotrimethoxybenzene		✓		N
2-chlorovanillin				Y
5-chlorovanillin				Y
6-chlorovanillin				Y
2,5-dichlorovanillin				Y
2,6-dichlorovanillin				Y
5,6-dichlorovanillin				Y
2,5,6-trichlorovanillin		✓		N

Substances historically identified in effluent	Screened from candidate list of chemicals based on [a]:			Chemical of Interest[b]
	High MW	Bleaching & pulping process	Not relevant for present assessment	
Dichloroveratrole				Y
3,4,5-Trichloroveratrole		✓		N
1,2,3,4-tetrachloro-5,6-veratrole		✓		N
Chlorophenols[e]				
2-Monochlorophenol				Y
3-Monochlorophenol				Y
4-Monochlorophenol				Y
2,3-Dichlorophenol				Y
2,4-Dichlorophenol				Y
2,5-Dichlorophenol				Y
2,6-Dichlorophenol				Y
3,4-dichlorophenol				Y
3,5-dichlorophenol				Y
2,3,4-trichlorophenol		✓		N
2,3,5-trichlorophenol		✓		N
2,3,6-trichlorophenol		✓		N
2,4,5-trichlorophenol		✓		N
2,4,6-trichlorophenol		✓		N
2,3,4,5-tetrachlorophenol		✓		N
2,3,4,6-tetrachlorophenol		✓		N
2,3,5,6-tetrachlorophenol		✓		N
Pentachlorophenol		✓		N
4-Chloro-3-methylphenol				Y
Other aromatic compounds				
Sterols				
beta-sitostanol				Y
beta-sitosterol				Y
Fucosterol				Y
Betulinol				Y
Campesterol				Y
Stigmasterol				Y
Squalene (steroid hydrocarbon)				Y
Miscellaneous aromatics[e]				
2,4-Dimethylphenol				Y
2-Methylphenol				Y
2-Nitrophenol				Y
3-Methylphenol				Y
4-Methylphenol				Y
Phenol				Y
Dichloroacetovanillone				Y

Substances historically identified in effluent	Screened from candidate list of chemicals based on [a]:			Chemical of Interest[b]
	High MW	Bleaching & pulping process	Not relevant for present assessment	
a-pinene				Y
Benzene				Y
b-pinene				Y
1,2,4-Trichlorobenzene		✓		N
Camphene				Y
Dehydrojuvabione				Y
Juvabione				Y
Dichloromethylenefuranones				Y
chlorodimethylnaphtalenes				Y
Alkylchlorophenanthrenes				Y
4-chloro-3-hydroxy-2H-pyran-2-one[c]				Y
5,5-dichloro-6,6-dihydroxy-2-methoxy-2-cyclohexene-1,4-dione				Y
Dichloroprotocatechualdehyde				Y
3-methoxy-5-dichloromethylene -2(H5)furanone				Y
Aniline				Y
2,4-Dichloroaniline[f]		✓		N
2,5-Dichloroaniline[f]		✓		N
3,4-Dichloroaniline[f]		✓		N
2,4,6-trichloroanisole		✓		N
3-methoxycatechol				Y
p-cymene				Y
p-hydroxybenzaldehyde				Y
Syringol				Y
Dioxins (PCDD/PCDFs not PCBs or PCBB/PCBFs) I-TEQ (refer notes below)				Y
Miscellaneous				
Hydrocarbons[e]				
Methanol				Y
Ethanol				Y
3-carene				Y
Carbon disulphide				Y
Hexane (C6)				Y
Pentadecane (C15)				Y
Octadecane (C18)				Y
Nonadecane (C19)				Y
cosane (C20)				Y

Substances historically identified in effluent	Screened from candidate list of chemicals based on [a]:			Chemical of Interest[b]
	High MW	Bleaching & pulping process	Not relevant for present assessment	
heneicosane (C21)				Y
eicosane (C22)				Y
tricosane (C23)				Y
tetracosane (C24)				Y
pentacosane (C25)				Y
Limonene				Y
Styrene		✓		N
Toluene		✓		N
m-&p-xylene		✓		N
o-xylene		✓		N
Aldehydes & Ketones[e]				
Acetaldehyde				Y
Pentanal				Y
Hexanal				Y
Heptanal				Y
Octanal				Y
Nonanal				Y
Acetone				Y
1,1-dichloroacetone				Y
Pentachloroacetone		✓		N
2-butanone				Y
2-pentanone				Y
2-hexanone				Y
2-heptanone				Y
2-octanone				Y
2-nonanone				Y
Hydroquinone				Y
4-methyl-2-pentanone				Y
Dichlorocyclohexendione				Y
Trihalomethanes				
Bromodichloromethane		✓		N
Bromoform		✓		N
Chloroform		✓		N
Dibromohloromethane		✓		N
Chlorinated Hydrocarbons[e]				
Chloromethane				Y
Dichloromethane				Y
Carbon tetrachloride		✓		N
1,1-Dichloropropane				Y
1,3-Dichloropropane				Y

Substances historically identified in effluent	Screened from candidate list of chemicals based on [a]:			Chemical of Interest[b]
	High MW	Bleaching & pulping process	Not relevant for present assessment	
1,1,1-Trichloroethane		✓		**N**
1,1,2-Trichloroethane		✓		**N**
1,1,2,2-Tetrachloroethane		✓		**N**
Pentachloroethane		✓		**N**
Hexachloroethane		✓		**N**
Chloroethylene				**Y**
1,1-Dichloroethylene				**Y**
1,2-Dichloroethylene (cis & trans)				**Y**
1,3-dichloropropene (cis & trans)				**Y**
Trichloroethylene		✓		**N**
Tetrachloroethylene		✓		**N**
3-chloropropene				**Y**
1,1-dichlorodimethylsulfone				**Y**
1,1,3-trichlorodimethylsulfone		✓		**N**
Chlorohydroxypyrone				**Y**
tetrachlorothiophene		✓		**N**
Hexachlorocyclopentadiene		✓		**N**

[a] Ticks within the screening columns provide the reason for removal of chemicals from the present assessment. The chemical receives a tick if it has a high molecular weight, is not likely to be formed in the current bleaching and pulping process or is not relevant for human health.

[b] Within the chemical of interest column, Y refers to those that are chemicals of interest and N refers to those that are removed during screening, therefore are not chemicals of interest. To remove a chemical, there must be a tick within the screening columns.

[c] Jaako Poyry 2005c state the organic acids are comprised mainly acetic acids.

[d] Jaako Poyry 2005c state that fatty acids are 95% removed from the final effluent, therefore 5% remains in the final effluent.

[e] Many of these substances are ready biodegradable and are likely to be removed during biological treatment, however it was assumed a small proportion will survive biological treatment.

[f] According to JP (2005) this compound is not expected to be present or will only be present in trace amounts.

A1.3 Chemicals of interest for Bell Bay mill

Jaakko Pöyry Oy have used their expert knowledge of the kraft process and mill design to estimate final effluent concentrations for either individual or classes of chemicals (JP 2005 c,d,e,f) This was done using mass balance equations where all sources of contribution for an effluent constituent were considered relative to effluent control and treatment efficiencies. This information was supplemented by Toxikos with information from the literature, as described below, to assign concentration values for each effluent constituent assumed to be possibly present in the Bell Bay effluent. Estimates of the total concentration of a class of chemical are considered by Jaakko Pöyry Oy to be conservative, i.e. over estimates. Additional conservatism has been layered over this by the Toxikos default assumption that any one member of a class of chemicals could be present at a concentration at least 20% of the total estimated by Jaakko Pöyry Oy, however where information suggests the proportion is higher this has been adopted.

Table A1.3 contains is the list of *'chemicals of interest'* together with their estimated concentration in final effluent. It should be noted the estimated concentrations are not definitive, far from it; however Toxikos is of the opinion that the concentration of any individual constituent in the final effluent is likely to be over-estimated rather than under-estimated. Below is the brief rational used for assigning a concentration to members of major chemical classes.

Resin acids

A final effluent concentration for total resin acids of 250 µg/L was provided by Jaako Pöyry Oy (2005c) based on processing *Pinus radiata*. Resin acids are natural carboxylic acid constituents of wood. Softwood and hardwood are expected to contain different compounds and different ratios of resin acids, with softwood generally having the greater amount. At least 13 resin acids are considered to be possibly present in the Bell bay effluent; an estimation of the concentration of each compound has not been attempted. Instead a default conservative assumption has been made that the concentration of an individual resin acid could be as high as 20% of the total resin acids estimated in the final effluent (250 µg/L). That is each could be at 0.2 x 250 µg/L = 50 µg/L.

Fatty acids

It is not possible with our current knowledge to identify and assign concentrations to individual fatty acids present in the final effluent. Therefore for the purposes of the present assessment the default assumption that an individual fatty acid in Table A1.3 (n = 9) could have a concentration of 20% of

the total concentration of fatty acids (190 µg/L) estimated by Jaakko Pöyry Oy to be in the final effluent. That is each fatty acid could be at 0.2 x 190 µg/L = 38 µg/L, rounded to 40 µg/L.

Chloroacetic acids

Three chlorinated acetic acids (monochloroacetic acid, dichloroacetic acid and trichloroacetic acid) were identified in effluent from hardwood pulp mills (Strömberg et al. 1996). The mono- and di-chloroacetic acids are very effectively removed (96 – 100%) in activated sludge plants and aerated lagoons, however efficient removal of tri-chloroacetic acid required longer retention times. Wallis et al. (1994a) simulated bleaching Australian eucalyptus kraft pulp in the laboratory using either chlorine, chlorine dioxide or a mixture of chlorine dioxide/chlorine as bleaching agents. Analysis for chlorinated aliphatic acids showed only these chlorinated acetic acids were formed. With chlorine dioxide, dichloroacetic acid was the most abundant (66% of total) of the three. Both Strömberg et al. (1996) and Wallis et al. (1994a) report chloroacetic acid effluent loads were reduced with ECF bleaching compared to Cl_2.

Since Jaakko Pöyry (JP 2005c) state the organic acids present in the final effluent are mostly acetic acids together with other unknown small molecular weight organic acids it has been assumed the difference between total organic acids and the sum of the resin plus fatty acid content of effluent are entirely chloroacetic acids. That is:

- Total organic acids = chloroacetic acids (n=3) + total resin acids + total fatty acids.
  Substituting concentrations provided by Jaakko Pöyry (2005c,d).
  4,500 µg/L = chloroacetic acids (n=3) + 250 µg/L + 190 µg/L
  Thus total chloroacetic acids = 4,060 µg/L
- The concentration of each chloroacetic acid is assumed to be:

Total chloroacetic acids (4,060 µg/L) x 0.3 = ~1,350 µg/L for each chloroacetic acid.

Aromatic Compounds (chlorinated phenolics, sterols, and miscellaneous aromatic compounds)

Jaakko Pöyry (JP 2005c) considers aromatic compounds make up 5% of the total low molecular weight substances in the final effluent. An estimate of 25 µg/L is provided for chlorinated phenolics (JP 2005d) but not for other compounds. Chlorinated natural phenolics are the biggest contributors (assumed to be 95%) to the total chlorinated phenolics with simple chlorophenols likely to be present in small quantities (5%). We therefore assume the concentration of total chlorinated natural phenolics is 24 µg/L and total chlorophenols (1 µg/L). As a conservative default assumption the

proportion any individual chlorinated phenolic compound in the final effluent could be as high as 20% of the total concentration for its class (i.e. 5 µg/L for chlorinated natural phenolics and 0.2 µg/L chlorophenols).

Sterols

Jaakko Pöyry (JP 2005c) considers aromatic compounds, other than natural phenolics which have become chlorinated and the chlorophenols will in total be at a concentration of approximately 475 µg/L. These 'other aromatic compounds' are comprised of sterols and miscellaneous aromatics in Table A1.3. Sterols occur both in softwood and hardwood species, often β-sitosterol is the dominating species (Strömberg et al. 1996, Gutierrez et al. 2001). Because the sterols are lipophilic they can survive treatment processes (Kostamo et al. 2004). Hence for the purposes of estimating a final effluent concentration of sterols a conservative default assumption was made that 90% of the 'other aromatic compounds' present in the final effluent will be sterols. Therefore final effluent concentration for total sterols is 428 µg/L. As a conservative default assumption the proportion of an individual sterol compound in the final effluent could be as high as 20% of the estimated total sterol concentration (i.e. 86 µg/L rounded to 90 µg/L).

Miscellaneous aromatic compounds

The intention of kraft pulping is to remove the bulk of lignin while minimising the degradation of cellulose. The chemicals formed from the reaction of lignin with the pulping liquors are generally polar in nature and include phenolic compounds and related compounds such as those identified in the Tables A1.1 & A1.2. For the purposes of estimating a final effluent concentration for the other aromatic compounds it was assumed 10% would not be sterols. Since Jaakko Pöyry (JP 2005c) consider there may be 475 µg/L of total aromatic compounds the miscellaneous aromatics (n= 25) listed in Table A1.3 may be cumulatively present at 10% of the total (i.e. ~ 48 µg/L) and individually as high as 20% of this concentration, i.e. ~ 10 µg/L.

Polychlorinated aromatic compounds (dioxins/furans)

Polychlorinated aromatic compounds are virtually eliminated (i.e. present below sensitive detection limits) from the effluent of ECF mills (refer Section 4.2). This prediction is made with reasonable confidence because the chemical reaction scheme for the formation of PCDD/F during pulp processing is known (JP 2005a) and they either have not been detected in ECF effluent or only at very low, at background, concentrations. Jaakko Pöyry (JP 2005d) believe dioxins in the final effluent of the Bell Bay mill will be below analytical limits of detection. Nevertheless because of the

presence of trace amounts of elemental chlorine in the chlorine dioxide bleaching process it is theoretically possible that they may still be formed. Jaakko Pöyry (JP 2005d) consider the Bell Bay mill bleaching and effluent treatment processes will result in at least a 90% reduction of PCDD/F compared to the concentrations measured in the 1990s in Sweden and Canada. On this basis the final effluent concentration of PCDD/F is assumed to be 0.074 pg TEQ/L.

Note that the US EPA state that a 96% reduction in PCDD/F production can be expected with the introduction of chlorine dioxide bleaching (US EPA 1997). Therefore the final effluent concentration of 0.074 pg total PCDD/PCDF I-TEQ/L is considered a theoretical worst case estimate.

Miscellaneous

Pulping by-products in effluent also include a wide variety of chlorinated and/or non chlorinated aliphatic alcohols and hydrocarbons. An estimate of the total quantity of these compounds was provided by Gunns for the Bell Bay mill. As a conservative default assumption the proportion of each compound in the final effluent could be as high as 20% of the total in Table A1.3 estimated by Jaakko Pöyry (JP 2005c) for the class.

Table A1.3: *'Chemicals of interest'* in Bell Bay effluent

Chemicals of interest	Estimated concentration in final effluent (µg/L)	Estimated concentration in initial dilution zone (µg/L)[a]	Reference
Metals			
Aluminium	600	6	JP 2005e
Antimony	1.4	0.014	JP 2005e
Arsenic (III &V)	2.9	0.029	JP 2005e
Barium	6.1	0.06	JP 2005e
Beryllium	1.5	0.015	JP 2005e
Boron	23	0.23	JP 2005e
Cadmium	1.2	0.012	JP 2005e
Chromium (III & VI)	20	0.20	JP 2005e
Cobalt	3	0.03	JP 2005e
Copper	20	0.20	JP 2005e
Iron	817	8.17	JP 2005e
Lead	3	0.03	JP 2005e
Manganese	817	8.17	JP 2005e
Mercury	0.3	0.003	JP 2005e
Molybdenum	1.4	0.014	JP 2005e
Nickel	28	0.28	JP 2005e
Selenium	7.5	0.075	JP 2005e
Silicon	570	0.570	JP 2005e
Tin	13.8	0.138	JP 2005e
Vanadium	1.4	0.014	JP 2005e
Zinc	84.7	0.847	JP 2005e
Non metallic inorganics			
Ammonia	< 455	< 4.55	JP 2005a
Hydrogen sulphide			
Nitrate	< 18,181	< 182	JP 2005a
Thiolignin			
Thiosulphate			
Organic acids	4,500	45	JP 2005c
Chloroacetic acids	4,060	41	Estimate
Monochloroacetic acid	1,350[b]	13.5	Estimate
Dichloroacetic acid (includes methyl ester)	1,350[b]	13.5	Estimate
Trichloroacetic acid[c]	1,350[b]	13.5	Estimate
Resin acids (n=13)	**250**	**2.50**	**JP 2005c**
14-chlorodehydroabeitic acid	50[d]	0.50	Estimate
12,14-dichlorohydroabietic acid	50[d]	0.50	Estimate

Chemicals of interest	Estimated concentration in final effluent (µg/L)	Estimated concentration in initial dilution zone (µg/L)[a]	Reference
Abietine	50[d]	0.50	Estimate
Arakine	50[d]	0.50	Estimate
Chlororetene	50[d]	0.50	Estimate
dehydroabietic acid	50[d]	0.50	Estimate
Fichtelite	50[d]	0.50	Estimate
Isopirame	50[d]	0.50	Estimate
Levopirame	50[d]	0.50	Estimate
Neoabietine	50[d]	0.50	Estimate
Palustrine	50[d]	0.50	Estimate
pimaric acid	50[d]	0.50	Estimate
Retene	50[d]	0.50	Estimate
Fatty acids	**190**	**1.90**	**JP 2005c**
(2E,4Z)-Hexadienedioic acid monomethyl ester	40[d]	0.40	Estimate
Behene	40[d]	0.40	Estimate
Lignoserine	40[d]	0.40	Estimate
Linoleine	40[d]	0.40	Estimate
Myristine	40[d]	0.40	Estimate
Oleine	40[d]	0.40	Estimate
Palmitine	40[d]	0.40	Estimate
Stearine	40[d]	0.40	Estimate
Linoleic acid	40[d]	0.40	Estimate
Chlorinated natural phenolics	**24 (total)**	**0.24 (total)**	**JP 2005c**
4-chlorocatechol	5[d]	0.05	Estimate
3,4-dichlorocatechol	5[d]	0.05	Estimate
3,5-dichlorocatechol	5[d]	0.05	Estimate
2-chloro-p-cymene	5[d]	0.05	Estimate
5-chloro-o-cymene	5[d]	0.05	Estimate
2,3-dichloro-p-cymene	5[d]	0.05	Estimate
2,5-dichloro-p-cymene	5[d]	0.05	Estimate
2,6-dichloro-m-cymene	5[d]	0.05	Estimate
4-chloroguaiacol	5[d]	0.05	Estimate
3,4-dichloroguaiacol	5[d]	0.05	Estimate
4,5-dichloroguaiacol	5[d]	0.05	Estimate
4,6-dichloroguaiacol	5[d]	0.05	Estimate
2-chloroacetoguaiacones	5[d]	0.05	Estimate
5-chloroacetoguaiacones	5[d]	0.05	Estimate
6-chloroacetoguaiacones	5[d]	0.05	Estimate
2-Monochlorosyringaldehyde	5[d]	0.05	Estimate
2,6-dichlorosyringaldehyde	5[d]	0.05	Estimate
2-Chloro-3,4,5-trimetoxybenzaldehyde	5[d]	0.05	Estimate
2,6-Dichloro-3,4,5-trimetoxybenzaldehyde	5[d]	0.05	Estimate

Chemicals of interest	Estimated concentration in final effluent (µg/L)	Estimated concentration in initial dilution zone (µg/L)[a]	Reference
2-chlorovanillin	5[d]	0.05	Estimate
5-chlorovanillin	5[d]	0.05	Estimate
6-chlorovanillin	5[d]	0.05	Estimate
2,5-dichlorovanillin	5[d]	0.05	Estimate
2,6-dichlorovanillin	5[d]	0.05	Estimate
5,6-dichlorovanillin	5[d]	0.05	Estimate
Dichloroveratrole	5[d]	0.05	Estimate
Chlorophenols	**1 (total)**	**0.01 (total)**	**JP 2005c**
2-Monochlorophenol	0.2[d]	0.002	Estimate
3-Monochlorophenol	0.2[d]	0.002	Estimate
4-Monochlorophenol	0.2[d]	0.002	Estimate
2,3-Dichlorophenol	0.2[d]	0.002	Estimate
2,4-Dichlorophenol	0.2[d]	0.002	Estimate
2,5-Dichlorophenol	0.2[d]	0.002	Estimate
2,6-Dichlorophenol	0.2[d]	0.002	Estimate
3,4-dichlorophenol	0.2[d]	0.002	Estimate
3,5-dichlorophenol	0.2[d]	0.002	Estimate
4-Chloro-3-methylphenol	0.2[d]	0.002	Estimate
Other aromatic compounds	**475 (total)**	**4.75 (total)**	**JP 2005c**
Sterols	**428**	**4.28**	**Estimate**
beta-sitostanol	90[d]	0.90	Estimate
beta-sitosterol	90[d]	0.90	Estimate
Fucosterol	90[d]	0.90	Estimate
Betulinol	90[d]	0.90	Estimate
Campesterol	90[d]	0.90	Estimate
Stigmasterol	90[d]	0.90	Estimate
Squalene (steroid hydrocarbon)	90[d]	0.90	Estimate
Miscellaneous aromatics	**48 (10% of total)**	**0.48 10% of total**	**JP 2005c**
2,4-Dimethylphenol	10[d]	0.10	Estimate
2-Methylphenol	10[d]	0.10	Estimate
2-Nitrophenol	10[d]	0.10	Estimate
3-Methylphenol	10[d]	0.10	Estimate
4-Methylphenol	10[d]	0.10	Estimate
Phenol	10[d]	0.10	Estimate
Dichloroacetovanillone	10[d]	0.10	Estimate
a-pinene	10[d]	0.10	Estimate
Benzene	10[d]	0.10	Estimate
b-pinene	10[d]	0.10	Estimate
Camphene	10[d]	0.10	Estimate
Dehydrojuvabione	10[d]	0.10	Estimate
Juvabione	10[d]	0.10	Estimate
Dichloromethylenefuranones	10[d]	0.10	Estimate

Chemicals of interest	Estimated concentration in final effluent (µg/L)	Estimated concentration in initial dilution zone (µg/L)[a]	Reference
chlorodimethylnaphtalenes	10[d]	0.10	Estimate
Alkylchlorophenanthrenes	10[d]	0.10	Estimate
4-chloro-3-hydroxy-2H-pyran-2-one	10[d]	0.10	Estimate
5,5-dichloro-6,6-dihydroxy-2-methoxy-2-cyclohexene-1,4-dione	10[d]	0.10	Estimate
Dichloroprotocatechualdehyde	10[d]	0.10	Estimate
3-methoxy-5-dichloromethylene -2(H5)furanone	10[d]	0.10	Estimate
Aniline	10[d]	0.10	Estimate
3-methoxycatechol	10[d]	0.10	Estimate
p-cymene	10[d]	0.10	Estimate
p-hydroxybenzaldehyde	10[d]	0.10	Estimate
Syringol	10[d]	0.10	Estimate
Dioxins (PCDD/PCDFs not PCBs or PCBB/PCBFs) I-TEQ	**0.074 pg/l (total)**	**0.00074 pg/l (total)**	**JP 2005c**
Miscellaneous	**60 (total)**	**0.60 (total)**	**JP 2005c**
Hydrocarbons			
Methanol	12[d]	0.12	Estimate
Ethanol	12[d]	0.12	Estimate
3-carene	12[d]	0.12	Estimate
Carbon disulphide	12[d]	0.12	Estimate
Hexane (C6)	12[d]	0.12	Estimate
Pentadecane (C15)	12[d]	0.12	Estimate
Octadecane (C18)	12[d]	0.12	Estimate
Nonadecane (C19)	12[d]	0.12	Estimate
cosane (C20)	12[d]	0.12	Estimate
heneicosane (C21)	12[d]	0.12	Estimate
eicosane (C22)	12[d]	0.12	Estimate
tricosane (C23)	12[d]	0.12	Estimate
tetracosane (C24)	12[d]	0.12	Estimate
pentacosane (C25)	12[d]	0.12	Estimate
Limonene	12[d]	0.12	Estimate
Aldehydes & Ketones			
Acetaldehyde	12[d]	0.12	Estimate
Pentanal	12[d]	0.12	Estimate
Hexanal	12[d]	0.12	Estimate
Heptanal	12[d]	0.12	Estimate
Octanal	12[d]	0.12	Estimate
Nonanal	12[d]	0.12	Estimate

Chemicals of interest	Estimated concentration in final effluent (µg/L)	Estimated concentration in initial dilution zone (µg/L)[a]	Reference
Acetone	12[d]	0.12	Estimate
1,1-dichloroacetone	12[d]	0.12	Estimate
2-butanone	12[d]	0.12	Estimate
2-pentanone	12[d]	0.12	Estimate
2-hexanone	12[d]	0.12	Estimate
2-heptanone	12[d]	0.12	Estimate
2-octanone	12[d]	0.12	Estimate
2-nonanone	12[d]	0.12	Estimate
Hydroquinone	12[d]	0.12	Estimate
4-methyl-2-pentanone	12[d]	0.12	Estimate
Dichlorocyclohexendione	12[d]	0.12	Estimate
Chlorinated Hydrocarbons			
Chloromethane	12[d]	0.12	Estimate
Dichloromethane	12[d]	0.12	Estimate
1,1-Dichloropropane	12[d]	0.12	Estimate
1,3-Dichloropropane	12[d]	0.12	Estimate
Chloroethylene	12[d]	0.12	Estimate
1,1-Dichloroethylene	12[d]	0.12	Estimate
1,2-Dichloroethylene (cis & trans)	12[d]	0.12	Estimate
1,3-dichloropropene (cis & trans)	12[d]	0.12	Estimate
3-chloropropene	12[d]	0.12	Estimate
1,1-dichlorodimethylsulfone	12[d]	0.12	Estimate
Chlorohydroxypyrone	12[d]	0.12	Estimate

[a] A dilution factor of 100 is applied to the final effluent concentration to calculate the concentration at the edge of the DZ100.

[b.] The total of chloroacetic acids was divided by three to determine the concentration of each individual chloroacetic acid.

[c] Footnote on trichloroethylene

[d] Default assumption used in the assessment: individual compounds in a group are present at 20% each (i.e. 0.2 x total).

A1.4 References

Bright, D.A., Hodson, P.V., Lehtinen, K.J., McKague, A.B., Rodgers, J.H. and Soloman, K. R. (2003). Evaluation of ecological risks associated with the use of chlorine dioxide for the bleaching of pulp: Scientific progress since 1993. In Environmental Impacts of Pulp and Paper Waste Streams, Stuthridge, T., van den Hueval, M.R., Marvin, N.A., Slade, A.H. and Gifford, J. (Ed). Setac Press, 2003.

EA (1998). National Pollutant Inventory. Emission Estimation Technique Manual for Pulp and Paper Manufacturing. Environment Australia August 1998.

EC (1991). Priority Substances List Assessment Report No. 2 for Effluents from Pulp Mills Using Bleaching Environment Canada, Health Canada.

Gutiérrez, A., Romero, J., del Río, J.C. (2001). Lipophilic extractives in process waters during manufacturing of totally chlorine free kraft pulp from eucalypt wood. Chemosphere. 44: 1237-1242.

Hewitt, M., Carey, J.,H., Dixon, G., and Munkittrick, K.,R. (1996). Examination of bleached kraft mill effluent fractions for potential inducers of mixed function oxygenase activity in rainbow trout. In Environmental Fate and Effects of Pulp and Paper Mill Effluents. Servos , M.,R., Munkittrick, K.,R., Carey, J.,H., and van der Kraak, G.,J. (Ed). St Lucie Press (1996).

JP (2005a) Compliance of the final effluent quality with ANZECC guidelines. Prepared for Gunns Ltd. Ref No: 16B0104 – E0016. June 10th 2005. Jaakko Pöyry Oy. 1.2 & Table 4.3

JP (2005b). Bioassay and chemical analysis results of the Advance Agro BEKP Mill effluent. prepared for Gunns Ltd. Ref. No 16B0104. 16 June, 2005. pages 18 2. Jaakko Pöyry Oy. Table 4.3

JP (2005c). Spreadsheet received from Jaako Pöyry Pty Ltd. titled *"roger NSI.Emissions"*. Received by email Friday, November 18, 2005

JP (2005e). received from Jaako Pöyry Pty Ltd. titled *"Effluent Loads and WWTP Design.HMs.Dec.05.xls"*. Received by email Monday, December 19, 2005

LaFleur, L.,E,.(1996). Sources of pulping and bleaching derived chemicals in effluents. In Environmental Fate and Effects of Pulp and Paper Mill Effluents. Servos , M.,R., Munkittrick, K.,R., Carey, J.,H., and van der Kraak, G.,J. (Ed). St Lucie Press, Delray Beach, Florida (1996).

RPDC (2004). Recommended environmental emission limit guidelines for any new bleached eucalypt kraft pulp mill in Tasmania. Volume 2, Resource Planning and Development Commission, Tasmania. August 2004.

Verta, M., Ahtiainen, J., Nakari, T., Langi, A., and Talka, E. (1996). The effect of waste constituents on the toxicity of TCF and ECF pulp bleaching effluents. In Environmental Fate and Effects of Pulp and Paper Mill Effluents. Servos , M.,R., Munkittrick, K.,R., Carey, J.,H., and van der Kraak, G.,J. (Ed). St Lucie Press, Delray Beach, Florida, 1996.

Wallis, A., Smith, T., and Wearne R. (1993). Characterisation of the major components (by mass) of selected effluent from bleaching eucalypt kraft pulp. Technical Report Series No. 10. National Pulp Mills Research Program CSIRO Division of Forest Products.

Wallis, A., Smith, T., and Wearne R. (1993). Determination of chlorinated phenols in effluent from bleached eucalypt kraft mills. Technical Report Series No. 2. National Pulp Mills Research Program. CSIRO Division of Forest Products, Commonwealth of Australia.

Appendix 2: Estimation of octanol-water partion coefficient and biocentration factors

The screening methodology for bioaccumulation is summarised in Section 5 and in particular Figure 5.1 of the main body of the report. The screening approach is intended to identify effluent constituents which have properties for bioaccumulation in aquatic organisms and thus pose a potential hazard to humans who consume those organisms. The magnitude of bioaccumulation by aquatic organisms varies widely depending on the chemical but can be extremely high for some highly persistent and hydrophobic chemicals that undergo limite metabolism/excretion by organisms. Since there are many *'chemicals of interest'* potentially present in the Bell Bay effluent a risk assessment based on bioaccumulation in seafood requires a screening phase to differentiate between chemicals that obviously have no potential to bioaccumulate and those that do.

Bioconcentration potential can be predicted by combination of the following three factors (ANZECC 2000):

1. ***Log K_{OW}:*** Bioconcentration occurs following passive diffusion from water or sediment across the absorptive membranes of aquatic organisms to be deposited in fatty tissue. Octanol is a reasonable surrogate for biota lipid for compounds having log K_{OW} values from 2 to about 6.5. However the different chemical nature of octanol and biota lipid may result in differences in bioconcentration behaviour even for substances with specific active chemical groups or may be ionised at physiological or environmental pH's even though they may have a log K_{OW} between 2 and 6.5 (Connell 1998).

   It is noted the relationship between octanol-water partition coefficient and bioconcentration does not apply to inorganic chemicals, and the bioavailability of substances with certain specific active groups, or ionic substances is chemical-specific.

   The K_{OW} values used for the bioaccumulation screening were obtained from either authoritative reference sources or estimated using a quantitative structure activity software program (EPISuite version 3.12, 2004, US EPA 2000b). The references used and estimation techniques are provided in Section A2.1 and Table A2.1 contains the parameter values for each chemical of interest used in the estimation. The EPISuite software provides an estimate of the logK_{ow} value and where available also provides experimental logK_{ow}

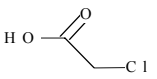
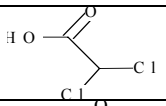
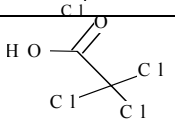
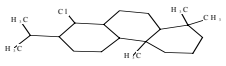
value(s) for a compound; for substances for which there was an experimental and calculated value available, the highest was selected so as not to under estimate the potential for bioaccumulation.

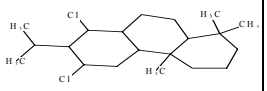
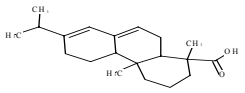
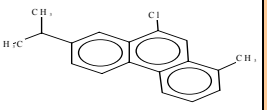
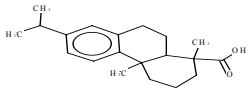
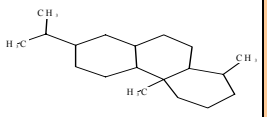
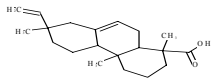
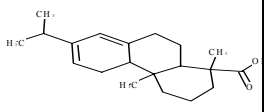
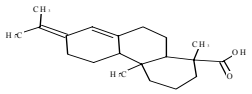
2. ***BCF (bioconcentration factor)*** refers to the accumulation in aquatic organisms resulting directly from uptake from water or sediment. Experimental BCF values for metals have been adopted from US EPA (2005) who reviewed field measured and experimental data for adequacy. For a particular metal the higher geometric mean of these data sets was chosen for the screening parameters. Experimental BCFs are not available for all the organic chemicals of interest hence they were estimated using a quantitative structure activity software program (EPISuite version 3.12, 2004, US EPA 2000b). The estimation technique is discussed in Section A2.2 below.

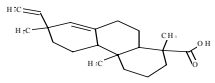
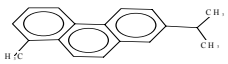
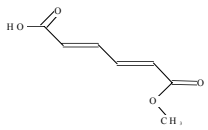
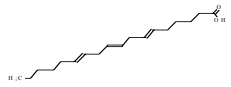
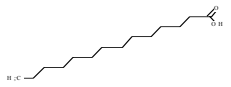
3. ***Metabolism/excretion:*** For most chemicals, it is expected that metabolism and elimination outstrips bioaccumulation. Because the calculated estimates of log K_{OW} and BCF may be over estimates of bioaccumulation potential, substances with log K_{OW} >4 and BCF > 10,000 in Table A2.1 below are further evaluated for the degree to which they are biotransformed/metabolised by aquatic organisms.

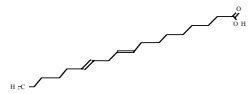
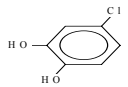
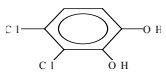
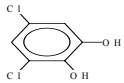
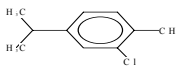
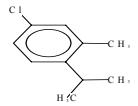
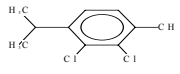
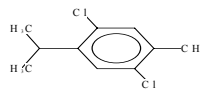
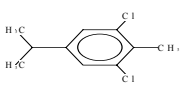
Table A2.1: *'Chemicals of interest'* screened for bioaccumulation

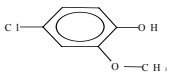
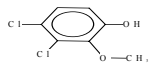
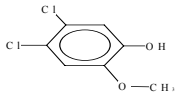
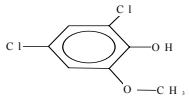
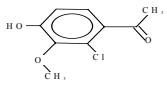
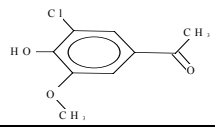
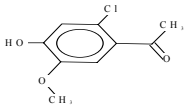
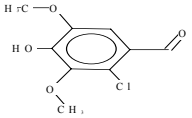
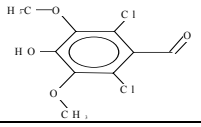
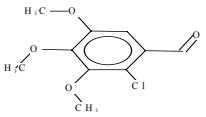
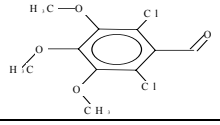
Chemicals of interest[a]	Judged Bioaccumulative by competent authority[b] (✓=yes, ×=no, ? =uncertain)	EPISuite		Log K_{ow}	BCF
		SMILES code	Structure	(reference footnote)	(reference footnote)
Metals					
Aluminium	×	NR[c]	NR		
Antimony	×	NR	NR		**40**[g]
Arsenic (III &V)	×	NR	NR	**N/A**[d]	**114**[g]
Barium	×	NR	NR	**N/A**	**633**[g]
Beryllium	×	NR	NR		**62**[g]
Boron	×	NR	NR	**N/A**	
Cadmium	✓[e]	NR	NR	**N/A**	**907**[g]
Calcium	×	NR	NR		
Chromium (III & VI)	×	NR	NR	**N/A**	**19**[g]

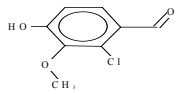
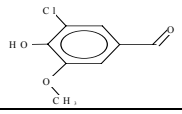
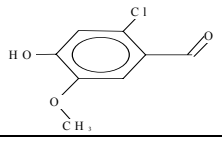
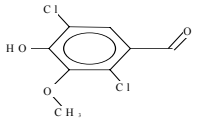
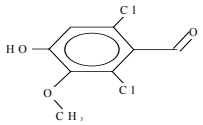
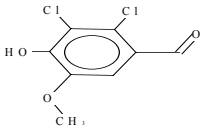
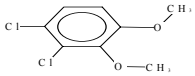
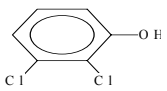
Chemicals of interest[a]	Judged Bioaccumulative by competent authority[b] (✓=yes, ×=no, **? =uncertain)**	EPISuite		Log K_{ow} (reference footnote)	BCF (reference footnote)
		SMILES code	Structure		
Cobalt	×	NR	NR		
Copper	×	NR	NR	**N/A**	
Iron	×	NR	NR	**N/A**	
Lead	×	NR	NR	**N/A**	**0.09[g]**
Magnesium	×	NR	NR		
Manganese	×	NR	NR	**N/A**	
Mercury	✓	NR	NR	**N/A**	**6,800,000[f]**
Molybdenum	×	NR	NR	**N/A**	
Nickel	×	NR	NR	**N/A**	**78[g]**
Selenium	✓	NR	NR	**N/A**	**129[g]**
Silicon	×	NR	NR		
Sodium	×	NR	NR		
Tin	×	NR	NR	**N/A**	
Vanadium	×	NR	NR		
Zinc	×	NR	NR	**N/A**	**2060[g]**
Non metallic inorganics					
Ammonia	×	NR	NR	**N/A**	
Hydrogen sulphide		NR	NR		
Nitrate	×	NR	NR		
Thiolignin		NR	NR		
Thiosulphate		NR	NR		
Organic acids					
Chloroacetic acids					
Monochloro acetic acid	?	OC(=O)CCL		**0.34[j]**	**3.16[j]**
Dichloroacetic acid	?	OC(=O)C(CL)CL		**0.92[k]**	**3.16[j]**
Trichloroacetic acid	?	OC(=O)C(CL)(CL)CL		**1.44[j]**	**3.16[j]**
Resin acids					
14-chloro dehydroabeitic acid	?	C1CCC2(C)C3CCC(C(C)C)C(CL)C3CCC2C1(C)(C)		**8.05[j]**	**2363[j]**

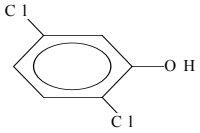
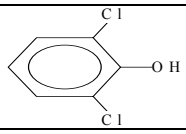
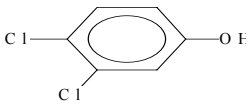
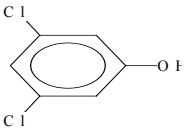
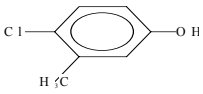
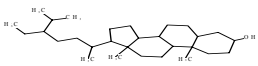
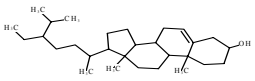
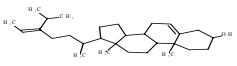
Chemicals of interest[a]	Judged Bioaccumulative by competent authority[b] (✓=yes, ×=no, **? =uncertain)**	EPISuite		Log K_{ow} (reference footnote)	BCF (reference footnote)
		SMILES code	Structure		
12,14-dichloro hydroabietic acid	?	C1CCC2(C)C3CC(CL)C(C(C)C)C(CL)C3CCC2C1(C)(C)		**8.23[j]**	**1337[j]**
Abietine	?	OC(=O)C1(C)CCCC2(C)C3CCC(C(C)C)=CC3=CCC21		**6.46[j]**	**56.23[j]**
Arakine	?	Structure could not be found			
Chlororetene	?	Cc1cccc2c3ccc(C(C)C)cc3c(CL)cc21		**6.99[j]**	**48,400[j]**
dehydroabietic acid	?	OC(=O)C1(C)CCCC2(C)c3ccc(C(C)C)cc3CCC21		**6.52[j]**	**56.33[j]**
Fichtelite	?	CC1CCCC2(C)C3CCC(C(C)C)CC3CCC21		**7.41[j]**	**17,500[j]**
Isopirame	?	OC(=O)C1(C)CCCC2(C)C3CCC(C)(C=C)CC3=CCC21		**6.45[j]**	**56.23[j]**
Levopirame	?	OC(=O)C1(C)CCCC2(C)C3CC=C(C(C)C)C=C3CCC21		**6.46[j]**	**56.23[j]**
Neoabietine	?	OC(=O)C1(C)CCCC2(C)C3CCC(=C(C)C)C=C3C CC21		**6.59[j]**	**56.23[j]**

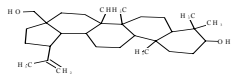
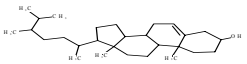
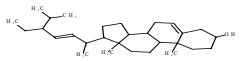
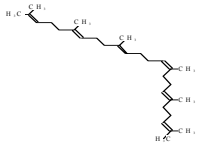
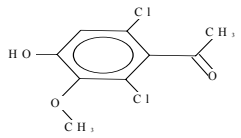
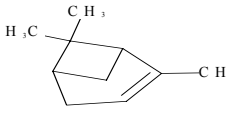
Chemicals of interest[a]	Judged Bioaccumulative by competent authority[b] (✓=yes, ×=no, **? =uncertain)**	EPISuite		Log K_{ow} (reference footnote)	BCF (reference footnote)
		SMILES code	**Structure**		
Palustrine	?	Has a double bond between ring groups, therefore can't SMILES, approximately similar to levopimaric acid		**6.45**[j]	**56.23**[j]
pimaric acid	?	OC(=O)C1(C)CCCC2(C)C3CCC(C)(C=C)C=C3CCC21		**6.45**[j]	**56.23**[j]
Retene	?	CC(C)c1cc2ccc3c(C)cccc3c2cc1		**6.35**[j]	**15,400**[j]
Fatty acids					
(2E,4Z)-Hexadienedioic acid monomethyl ester	?	OC(=O)C=CC=C C(=O)OC		**0.67**[j]	**3.16**[j]
Behene	?	OC(=O)CCCCCCCCCCCCCCCCCCCCCC		**9.91**[j]	**3.16**[j]
Lignoserine	?	CCCCCCCCCCCCCCCCCCCCCCCC(=O)O		**10.89**[j]	**3.16**[j]
Linoleine	?	CCCCCC=CCC=CCC=CCCCCC(=O)O		**7.3**[j]	**10**[j]
Myristine	?	CCCCCCCCCCCCCCC(=O)O		**6.11**[k]	**56.23**[j]
Oleine	?	CCCCCCCCC=CCCCCCCCC(=O)O		**7.73**[j]	**10**[j]

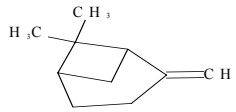
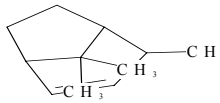
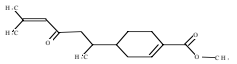
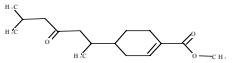
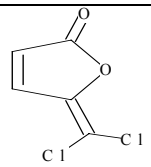
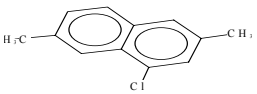
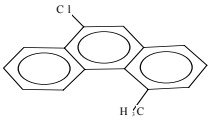
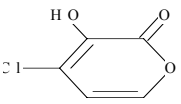
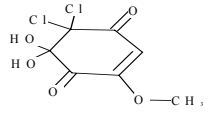
Chemicals of interest[a]	Judged Bioaccumulative by competent authority[b] (✓=yes, ×=no, ? =uncertain)	EPISuite		Log K_{ow} (reference footnote)	BCF (reference footnote)
		SMILES code	Structure		
Palmitine	?	CCCCCCCCCCCCCCCC(=O)O		**6.96[j]**	**10[j]**
Stearine	?	CCCCCCCCCCCCCCCCCC(=O)O		**8.23[k]**	**10[j]**
Linoleic acid	?	CCCCCC=CCC=CCCCCCCC(=O)O		**7.5[j]**	**10[j]**
Chlorinated natural phenolics					
4-chlorocatechol	?	c1cc(O)c(O)cc1(CL)		**1.68[j]**	**3.90[j]**
3,4-dichloro catechol	?	Oc1c(O)c(CL)c(CL)cc1		**2.32[j]**	**4.87[j]**
3,5-dichloro catechol	?	Oc1c(O)c(CL)cc(CL)c1		**2.32[j]**	**4.87[j]**
2-chloro-p-cymene	?	Cc1c(CL)cc(C(C)C)cc1		**4.64[j]**	**747[j]**
5-chloro-o-cymene	?	Cc1c(C(C)C)ccc(CL)c1		**4.64[j]**	**3.9[j]**
2,3-dichloro-p-cymene	?	Cc1c(CL)c(CL)c(C(C)C)cc1		**5.29[j]**	**3428[j]**
2,5-dichloro-p-cymene	?	Cc1c(CL)cc(C(C)C)c (CL)c1		**5.6[k]**	**4093[j]**
2,6-dichloro-m-cymene	?	Cc1c(CL)cc(C(C)C)cc1(CL)		**5.29[j]**	**2343[j]**

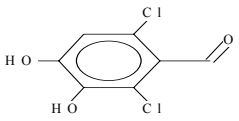
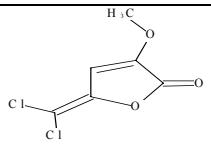
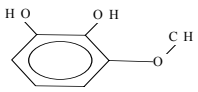
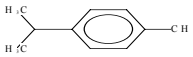
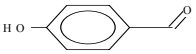
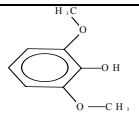
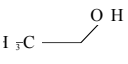
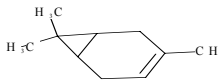
Chemicals of interest[a]	Judged Bioaccumulative by competent authority[b] (✓=yes, ×=no, **? =uncertain)**	EPISuite		Log K_{ow} (reference footnote)	BCF (reference footnote)
		SMILES code	Structure		
4-chloroguaiacol	?	Oc1c(OC)cc (CL)cc1		**1.98[j]**	**7[j]**
3,4-dichloro guaiacol	?	Oc1c(OC)c (CL)c(CL) cc1		**2.63[j]**	**8.37[j]**
4,5-dichloro guaiacol	?	Oc1c(OC)cc (CL)c (CL)c1		**2.63[j]**	**25.72[j]**
4,6-dichloro guaiacol	?	Oc1c(OC)cc (CL)c c1(CL)		**2.63[j]**	**8.37[j]**
2-chloroaceto guaiacones	?	CC(=O)c1c (CL)c(OC)c(O) cc1		**1.66[j]**	**0.55[j]**
5-chloroaceto guaiacones	?	CC(=O)c1cc(O C)c(O)c(CL)c1		**1.66[j]**	**0.55[j]**
6-chloroaceto guaiacones	?	CC(=O)c1cc (OC)c(O)cc1 (CL)		**1.66[j]**	**0.55[j]**
2-Monochloro syringaldehyde[b]	?	C(=O)c1c(CL) c(OC)c(O)c (OC)c1		**1.52[j]**	**2.98[j]**
2,6-dichloro syringaldehyde[b]	?	C(=O)c1c(CL) c(OC)c(O)c (OC)c1(CL)		**2.17[j]**	**3.72[j]**
2-Chloro-3,4,5-trimetoxybenzal dehyde	?	C(=O)c1c(CL) c(OC)c(OC)c (OC)c1		**1.87[j]**	**5.45[j]**
2,6-Dichloro-3,4,5-trimetoxybenzal dehyde	?	C(=O)c1c(CL) c(OC)c(OC)c (OC)c1(CL)		**2.51[j]**	**17.09[j]**

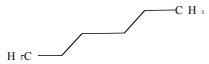
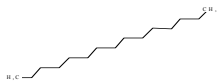
Chemicals of interest[a]	Judged Bioaccumulative by competent authority[b] (✓=yes, ×=no, **? =uncertain)**	EPISuite		Log K_{ow} (reference footnote)	BCF (reference footnote)
		SMILES code	Structure		
2-chlorovanillin	?	C(=O)c1c(CL)c(OC)c(O)cc1		**1.7[j]**	**4.06[j]**
5-chlorovanillin	?	C(=O)c1cc(OC)c(O)c(CL)c1		**1.7[j]**	**4.06[j]**
6-chlorovanillin	?	C(=O)c1cc(OC)c(O)cc1(CL)		**1.7[j]**	**4.06[j]**
2,5-dichloro vanillin	?	C(=O)c1c(CL)c(OC)c(O)c(CL)c1		**2.34[j]**	**5.07[j]**
2,6-dichloro vanillin	?	C(=O)c1c(CL)c(OC)c(O)cc1(CL)		**2.34[j]**	**5.07[j]**
5,6-dichloro vanillin	?	C(=O)c1cc(OC)c(O)c(CL)c1(CL)		**2.34[j]**	**5.07[j]**
Dichloro veratrole	?	COc1c(OC)c(CL)c(CL)cc1		**2.93[j]**	**35.79[j]**
Chlorophenols					
2-Monochloro phenol	×	NR	NR	**2.15[l]**	**9.03[j]**
3-Monochloro phenol	×	NR	NR	**2.5[l]**	**17.78[o]**
4-Monochloro phenol	×	NR	NR	**2.39[l]**	**15.14[o]**
2,3-Dichloro phenol	×	Oc1c(CL)c(CL)ccc1		**3.15[n]**	**12.21[j]**
2,4-Dichloro phenol	×	NR	NR	**3.2[n]**	**12.4[h]**

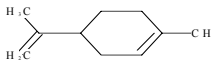
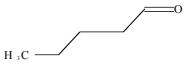
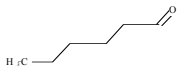
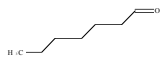
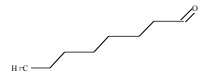
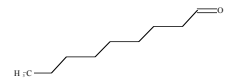
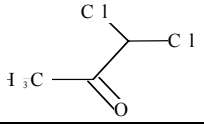
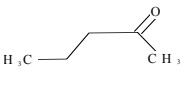
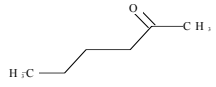
Chemicals of interest[a]	Judged Bioaccumulative by competent authority[b] (✓=yes, ×=no, **? =uncertain)**	EPISuite		Log K_{ow} (reference footnote)	BCF (reference footnote)
		SMILES code	Structure		
2,5-Dichloro phenol	×	Oc1c(CL)ccc (CL)c1		**3.2**[n]	**18.04**[j]
2,6-Dichloro phenol	×	Oc1c(CL)ccc c1(CL)		**2.86**[n]	**36.3**[o]
3,4-dichloro phenol	×	Oc1cc(CL)c(C L)cc1		**3.37**[n]	**29.11**[j]
3,5-dichloro phenol	×	Oc1cc(CL)cc (CL)c1		**2.80**[j]	**48.7**[j]
4-Chloro-3-methylphenol	?	Oc1cc(C)c (CL)cc1		**3.1**[i]	**48.6**[h]
Other aromatic compounds					
Sterols					
beta-sitostanol	?	OC1CCC2(C) C3CCC4(C)C (C(C)CCC(C C)C(C)C)CCC 4C3CCC2C1		**9.73**[j]	**11.65**[j]
beta-sitosterol	?	OC1CCC2(C) C3CCC4(C)C (C(C)CCC(CC)C(C)C)CCC4 C3C C=C2C1		**9.65**[j]	**15.25**[j]
Fucosterol	?	OC1CCC2(C) C3CCC4(C)C (C(C)CCC(=C C)C(C)C)CCC 4C3CC=C2C1		**9.56**[j]	**19.96**[j]

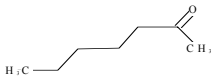
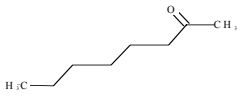
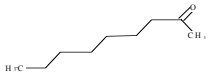
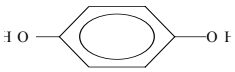
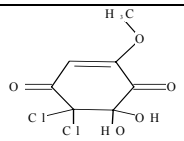
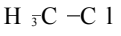
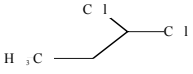
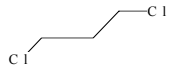
Chemicals of interest[a]	Judged Bioaccumulative by competent authority[b] (✓=yes, ×=no, **? =uncertain)**	EPISuite		Log K_{ow} (reference footnote)	BCF (reference footnote)
		SMILES code	Structure		
Betulinol	?	OC1CCC2(C)C3CCC4C5C(C(=C)C)CCC5(CO)CCC4(C)C3(C)CCC2C1(C)(C)		**8.18**[j]	**1,582**[j]
Campesterol	?	OC1CCC2(C)C3CCC4(C)C(C(C)CCC(C)C(C)C)CCC4C3CC=C2C1		**9.16**[j]	**71.78**[j]
Stigmasterol	?	OC1CCC2(C)C3CCC4(C)C(C(C)C=CC(CC)C(C)C)CCC4C3CC=C2C1		**9.43**[j]	**30.04**[j]
Squalene (steroid hydrocarbon)	?	CC(C)=CCCC(C)=CCCC(C)=CCCC=C(C)CCC=C(C)CCC=C(C)C		**14.12**[j]	**3.16**[j]
Miscellaneous aromatic compounds					
2,4-Dimethylphenol	×	NR	NR	**2.3**[m]	**11.8**[h]
2-Methylphenol	?	NR	NR	**1.95**[m]	**6.33**[h]
2-Nitrophenol	?	NR	NR	**1.79**[i]	**4.77**[h]
3-Methylphenol	?	NR	NR	**1.96**[m]	**6.44**[h]
4-Methylphenol	?	NR	NR	**1.96**[m]	**5.79**[h]
Phenol	×	NR	NR	**1.46**[m]	**2.85**[h]
Dichloro acetovanillone	?	CC(=O)c1c(CL)c(OC)c(O)cc1(CL)		**2.31**[j]	**0.69**[j]
a-pinene	?	CC1=CCC2C(C)(C) C1C2		**4.83**[k]	**523**[j]
Benzene	×	NR	NR	**2.13**[m]	**8.26**[h]

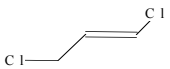
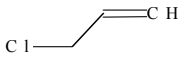
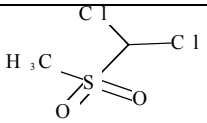
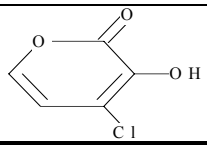
Chemicals of interest[a]	Judged Bioaccumulative by competent authority[b] (✓=yes, ×=no, **? =uncertain)**	EPISuite		Log K_{ow} (reference footnote)	BCF (reference footnote)
		SMILES code	Structure		
b-pinene	?	C=C1CCC2C(C)(C) C1C2		**4.35[j]**	**319[j]**
Camphene	?	CC1C=CC2C(C)(C) C1CC2		**4.63[j]**	**733[j]**
Dehydrojuvabione	?	COC(=O)C1=CCC(C(C)CC(=O)C=C(C)C)CC1		**4.42[j]**	**504[j]**
Juvabione	?	COC(=O)C1=CCC(C(C)CC(=O)CC(C)C)CC1		**4.22[j]**	**351[j]**
Dichloro methylene furanones	?	O1C(=C(CL)CL)C=CC1(=O)		**0.74[j]**	**3.16[j]**
Chlorodimethyl naphtalenes	?	Cc1cc(CL)c2cc(C)ccc2c1		**4.91[j]**	**1,200[j]**
Alkylchloro phenanthrenes	?	c1cc(C)c2c3ccccc3c(CL)cc2c1		**5.54[j]**	**3,659[j]**
4-chloro-3-hydroxy-2H-pyran-2-one	?	o1ccc(CL)c(O)c1(=O)		**-0.64[j]**	**3.16[j]**
5,5-dichloro-6,6-dihydroxy-2-methoxy-2-cyclohexene-1,4-dione	?	C1=C(OC)C(=O)C(O)(O)C(CL)(CL) C1(=O)		**1.51[j]**	**2.89[j]**

Chemicals of interest[a]	Judged Bioaccumulative by competent authority[b] (✓=yes, ×=no, **? =uncertain)**	EPISuite		Log K_{ow} (reference footnote)	BCF (reference footnote)
		SMILES code	**Structure**		
Dichloroprotocatechualdehyde	?	C(=O)c1c(CL)c(O)c(O)cc1(CL)		**2.04[j]**	**2.95[j]**
3-methoxy-5-dichloromethylene -2(H5)furanone	?	O=C1OC(=C(CL)CL)C=C1(OC)		**0.03[j]**	**3.16[j]**
Aniline	×	NR	NR	**0.9[m]**	**3.16[h]**
3-methoxy catechol	?	COc1cccc(O)c1(O)		**0.86[j]**	**3.16[j]**
p-cymene	?	Cc1ccc(C(C)C)cc1		**4.1[m]**	**286[j]**
p-hydroxy benzaldehyde	?	O=Cc1ccc(O)cc1		**1.35[m]**	**2.19[j]**
Syringol	?	Oc1c(OC)cccc1(OC)		**1.15[m]**	**1.53[j]**
Dioxins (I-TEQ)	✓				
Miscellaneous					
Hydrocarbons					
Methanol	?	NR	NR	**-0.77[m]**	**3.16[h]**
Ethanol	?	CCO		**-0.31[m]**	**3.16[j]**
3-carene	?	CC1=CCC2C(C)(C)C2C1		**4.61[j]**	**470[j]**
Carbon disulphide	×	NR	NR	**2.14[m]**	**9.86[h]**

Chemicals of interest[a]	Judged Bioaccumulative by competent authority[b] (✓=yes, ×=no, **? =uncertain)**	EPISuite		Log K_{ow} (reference footnote)	BCF (reference footnote)
		SMILES code	Structure		
Hexane (C6)	?	CCCCCC		**3.9[m]**	**201[j]**
Pentadecane (C15)	?	CCCCCCCCCCCCCCC		**7.71[j]**	**219[j]**
Octadecane (C18)	?	CCCCCCCCCCCCCCCCCC		**9.18[j]**	**3.16[j]**
Nonadecane (C19)	?	CCCCCCCCCCCCCCCCCCC		**9.67[j]**	**3.16[j]**
cosane (C20)	?	CCCCCCCCCCCCCCCCCCCC		**10.16[j]**	**3.16[j]**
heneicosane (C21)	?	CCCCCCCCCCCCCCCCCCCCC		**10.65[j]**	**3.16[j]**
eicosane (22)	?	CCCCCCCCCCCCCCCCCCCCCC		**11.15[j]**	**3.16[j]**
tricosane (C23)	?	CCCCCCCCCCCCCCCCCCCCCCC		**11.64[j]**	**3.16[j]**
tetracosane (C24)	?	CCCCCCCCCCCCCCCCCCCCCCCC		**12.13[j]**	**3.16[j]**

Chemicals of interest[a]	Judged Bioaccumulative by competent authority[b] (✓=yes, ×=no, **? =uncertain)**	EPISuite		Log K_{ow} (reference footnote)	BCF (reference footnote)
		SMILES code	Structure		
pentacosane (C25)	?	CCCCCCCCCCCCCCCCCCCCCCCCC		**12.62[j]**	**3.16[j]**
Limonene	?	CC1=CCC(C(=C)C) CC1		**4.83[j]**	**470[j]**
Aldehydes & Ketones					
Acetaldehyde	?	NR	NR	**-0.34[m]**	**3.16[h]**
Pentanal	?	CCCCC=O		**1.31[l]**	**2.03[j]**
Hexanal	?	CCCCCC=O		**1.78[m]**	**4.68[j]**
Heptanal	?	CCCCCCC=O		**2.29[l]**	**11.56[j]**
Octanal	?	CCCCCCCC=O		**2.78[l]**	**27.61[j]**
Nonanal	?	CCCCCCCCC=O		**3.27[l]**	**65.96[j]**
Acetone	?	NR	NR	**-0.24[m]**	**3.16[h]**
1,1-dichloro acetone	?	CLC(CL)C(=O)C		**0.2[l]**	**3.16[j]**
2-butanone		NR	NR	**0.3[m]**	**3.16[h]**
2-pentanone	?	CCCC(=O)C		**0.91[m]**	**3.16[j]**
2-hexanone	?	CCCCC(=O)C		**1.38[m]**	**2.31[j]**

Chemicals of interest[a]	Judged Bioaccumulative by competent authority[b] (✓=yes, ×=no, **? =uncertain)**	EPISuite		Log K_{ow} (reference footnote)	BCF (reference footnote)
		SMILES code	Structure		
2-heptanone	?	CCCCCC(=O)C		**1.98[m]**	**6.68[j]**
2-octanone	?	CCCCCCC(=O)C		**2.37[m]**	**13.33[j]**
2-nonanone	?	CCCCCCCC(=O)C		**2.88[m]**	**52.2[j]**
Hydroquinone	?	Oc1ccc(O)cc1		**0.59[m]**	**3.16[j]**
4-methyl-2-pentanone	?	NR	NR	**1.31[m]**	**1.67[h]**
Dichlorocyclo hexendione	?	O=C1C(OH)(OH)C(CL)(CL)C(=O)C=C1(OC)		**1.51[j]**	**2.89[j]**
Chlorinated Hydrocarbons					
Chloromethane	?	CCL		**1.97[m]**	**6.56[j]**
Dichloro methane	×	NR	NR	**1.25[m]**	**2[h]**
1,1-Dichloro propane	×	CCC(Cl)Cl		**2.25[j]**	**10.76[j]**
1,3-Dichloro propane	×	ClCCCCl		**2[m]**	**6.92[j]**
Chloroethylene	×	NR	NR	**1.62[l]**	**2.39[h]**
1,1-Dichloro ethylene	×	NR	NR	**2.13[m]**	**8.26[h]**
1,2-Dichloro ethylene (cis & trans)	×	NR	NR	**2.09[m]**	**8.26[h]**

Chemicals of interest[a]	Judged Bioaccumulative by competent authority[b] (✓=yes, ×=no, ? =uncertain)	EPISuite		Log K_{ow} (reference footnote)	BCF (reference footnote)
		SMILES code	Structure		
1,3-dichloro propene (cis & trans)	×	CLCC=CCL		**2.29**[j]	**7.30**[j]
3-chloropropene	×	CLCC=C		**1.93**[l]	**6.14**[j]
1,1-dichloro dimethylsulfone	×	C(CL)(CL)S (=O)(=O)C		**0.81**[j]	**3.16**[j]
Chlorohydroxy pyrone[a]	?	OC1=C(CL)C =COC1(=O)		**-0.64**[j]	**3.16**[j]

[a] Refer to Table A1.2 and A1.3.
[b] Refer to Figure 5.2 for a description of the screening methodology. Competent authorities utilised were ANZECC (2000) and US EPA (2000a). The screening criteria of Log K_{ow} of >4 and BAF/BCF of 10,000 is based on ANZECC (2000).
[c] NR = not required (chemicals could not be run on EPISuite or results were available in primary references.
[d] N/A = not applicable
[e] ANZECC considers cadmium to be bioaccumulative in marine environments only (ANZECC 2000).
[f] The BCF value is the BAF for methylmercury as cited by US EPA 2005 and not elemental mercury.
[g] Values for metal BCFs from US EPA HHRAP (2005).
[h] Estimate provided by US EPA (2005).
[i] Cited in US EPA (2005).
[j] Estimated by US EPA (2000).
[k] Experimental value provided by US EPA (2000).
[l] Estimate provided by Yaws (2003).
[m] Experimental value provided by Yaws (2003).
[n] Value provided by Shiu et al (1994) based on review of calculated and experimental values.
[o] Experimental value provided by Shiu et al (1994).

A2.1 Estimation of n-octanol water partition coefficient.

Values for log K_{OW} were obtained from primary sources (e.g. Yaws 2003, US EPA 2004, Shiu 1994 and Howard 1990) or estimated using the US EPA EPISuite (2000) KOWWIN program. The KOWWIN program uses atom or fragment contribution to determine the log K_{OW} for a compound. Compounds are divided into fragments. Each nonhydrogen atom (e.g. carbon, nitrogen, oxygen, sulphur) is defined as the core element for a fragment. The exact fragment is

determined by the atoms attached to the core. Some functional groups (e.g. carbonyl, cyano, nitrate) are also treated as core atoms. Connections to the core atom are defined either specifically or generally using a hierarchical system determined by Meylan and Howard (1995). Each fragment is assigned a coefficient based on regression analysis of experimental log K_{OW} values. Some fragments have correction factors to improve the estimation of log K_{OW} for more complex atoms. Correction factors were determined by comparing the log K_{OW} calculated from estimates of atoms alone and the measured values. Meylan and Howard (1995) calculated the equation for log K_{OW} based on 2351 compounds (Equation A1.1). To calculate the K_{OW} compounds are broken down into fragments and the number of times a fragment appears in a compound is noted. Coefficients and correction factors are assigned to each fragment and are summed together to give the log K_{OW}. This method was validated against a database of 6055 compounds. The accuracy of the model against the validation set gives a value of r^2 equal to 0.943, a standard deviation of 0.408 and a mean error 0.31. The validation data set was different to that used to generate the model, therefore the validation is representative of the accuracy in estimating unknown log K_{OW}. Of the entire data only 1.47% have an order of magnitude error in K_{OW}.

$$\log K_{ow} = \sum (f_i n_i) + \sum (c_j n_j) + 0.229 \qquad \text{.....Equation A2.1}$$

where

f_i	=	coefficient for each atom/ fragment
n_i	=	number of times the atom/ fragment appears
c_j	=	coefficient for each correction factor
n_j	=	number of times the correction factor is applied

A2.2 Estimation of bioconcentration factors.

Bioconcentration factors (BCFs) were obtained using the BCFWIN program in the US EPA EPISuite. Estimations are based on the assumption that bioconcentration is driven by thermodynamic partitioning between water and biological lipid, which is modelled using n-octanol (Meylan et al, 1999). To create a model for BCF estimation a database of 727 fish BCF were obtained along with measured or estimated log K_{ow} values (if estimation was required it was performed using KOWWIN). BCF values were excluded for 33 compounds based on a lack of reliable log K_{ow} or unknown validity in the measurement of the BCF value. Compounds were split according to ionic nature. For non-ionic compounds, the relationship between log K_{ow} and logBCF was defined by four different relationships (log K_{OW}<1; 1<log K_{OW}<7; 7<log K_{OW}<10.5; logK_{ow}>10.5). For logK_{ow} values between 1 and 7 the relationship was determined to be linear with certain functional groups requiring the addition of a correction factor. The correction factors are only added once for any functional group (Equation 2).

$$\log BCF = 0.77 \log K_{ow} - 0.7 + \sum F_i \qquad \text{.....Equation A2.2}$$

where

F_i = correction factor for a functional group

For values of logK_{ow}<1 the logBCF value ranged from 0 to 1 therefore a constant logBCF of 0.5 was assumed. For values of logK_{ow} above 7 a linear relationship was obtained for non-ionic compounds (Equation 3) that is similar to that obtained in Equation 2.

$$\log BCF = -1.37 \log K_{ow} + 14.4 + \sum F_i \qquad \text{.....Equation A2.3}$$

where

F_i = correction factor for a functional group

Equation A2.3 estimates negative logBCF values for logK_{ow}>10.5. This is not realistic so the equation output is truncated at logBCF=0.5 and consequently all logK_{ow} values greater than 10.5 are assumed to have a logBCF of 0.5. The correlation for 610 non-ionic compounds gives an r^2

value of 0.73, with a standard deviation of 0.67 and a mean error of 0.48 when validated against the database used to define the equations.

Compounds defined as ionic were carboxylic acids, sulfonic acids and salts and quaternary ammonium compounds. Originally phenols and anilines were defined as ionic compounds, however they were well described by the non-ionic equations and were redefined as non-ionic compounds. A linear model was not found to be representative of the relationship between $logK_{ow}$ and logBCF for ionic compounds. For $logK_{ow}$ values less than 5 the logBCF was found to lie between 0 and 1, hence a value of 0.5 was chosen as the logBCF. One exception is long alkyl chain compounds (≥11 carbons) which had logBCF values close to 2, consequently the recommended logBCF for these compounds is 1.85. There were very few logBCF values for ionic compounds with $logK_{ow}$ greater than 5, consequently the following guidelines (Table 1) were determined by assuming that the logBCF increases with $logK_{ow}$ up to 7 and then declines.

Table A2.1: Guidelines for estimation of logBCF for ionic compounds with $logK_{ow}$ > 5

$LogK_{ow}$	Log BCF
5 – 6	0.75
6 – 7	1.75
7 – 9	1.0
>9	0.5

The correlation of estimation for the ionic compounds when compared to the measured values gives a r^2 value of 0.62, with a standard deviation of 0.41 and a mean error of 0.31. The overall correlation of the logBCF estimation process (Table 2) gives an r^2 of 0.74, a standard deviation of 0.65 and a mean error of 0.47.

Table A2.2: Summary of logBCF estimation technique.

Non-ionic compounds		Ionic compounds	
$logK_{ow}$	logBCF	$logK_{ow}$	logBCF
<1	0.5	<5	0.5
1 – 7	$0.77 \log K_{ow} - 0.7 + \sum F_i$	<5 with ≥11 alkyl carbons	1.85 (lower limit)
>7	$-1.37 \log K_{ow} + 14.4 + \sum F_i$	5 – 6	0.75
>10.5	0.5	6 – 7	1.75
		7 – 9	1.0
		>9	0.5

A2.3 References

ANZECC (2000). Australian and New Zealand guidelines for fresh and marine water quality, 2000. Australian and New Zealand Environment and Conservation Council. Environment Australia.

Boethling, R.S., Lynch, D.G., Jaworska, J.S., Tunkel, J.L., Thom, G.C., Webb, S. (2004). Using Biowin^{TM}, Bayes, and batteries to predict ready biodegradability. Environmental Toxicology and Chemistry. 23(4): 911-920.

Burggraaf, S., Langdon, A.G., Wilkins, A.L. and Roper, D.S. (1996). Accumulation and depuration of resin acids and fichtelite by the freshwater mussel *Hyridella menziesi*. Environmental Toxicology and Chemistry 15(30:369-375.

di Giulio, R.,T., Benson, W., H., Sanders B., M., and van Veld, P.,A . (1995). Biochemical mechanisms: metabolism, adaptation, and toxicity. In 'Fundamentals of Aquatic toxicology' Ed Rand, G., M. Chapter 17 Taylor & Francis, p 523-560.

Connell, D.W. (1998) Bioaccumulation of Chemicals by Aquatic Organisms. Chapter 14 from Schüürmann, G. and Markert, B. (1998). Ecotoxicology. Ecological Fundamentals, Chemical Exposure, and Biological Effects. John Wiley & Sons Inc and Spektrum Adaemischer Verlag Co-Publication.

Howard, P.H. (1990). Handbook of environmental fate and exposure data for organic chemicals. Volume 1. Large production and priority pollutants. Lewis Publishers. Michigan, USA.

Gravato, C. and Santos, M.A. (2002). Juvenile sea bass liver biotransformation and erythrocytic genotoxic responses to pulp mill contaminants. Ecotoxicology and Environmental Safety 53(1):104-112.

Gutiérrez, A., Romero, J., del Río, J.C. (2001). Lipophilic extractives in process waters during manufacturing of totally chlorine free kraft pulp from eucalypt wood. Chemosphere. 44: 1237-1242.

Mcleay D. (1987). Aquatic Toxicity of Pulp and Paper Mill Effluent: A Review, Report EPS 4/PF/1, Environment Canada, Canada (1987).

Meylan, W.M., Howard, P.H., Boethling, R.S., Aronson, D., Printup, H. and Gouchie, S. (1999). Improved method for estimating bioconcentration/bioaccumulation factor from octanol/water partition coefficient. Environmental Toxicology and Chemistry. 18 (4): 664-672.

Meylan, W.M. and Howard, P.H. (1995). Atom/fragment contribution method for estimating octanol-water partition coefficients. Journal of Pharmaceutical Sciences. 84(1): 83-92.

Muir and Servos (1996). Bioaccumulation of bleached kraft pulp mill related organic chemicals by fish. In "Environmental Fate and Effects of Pulp and Paper Mill Effluents". Servos ,M.R., Munkittrick, K.R., Carey, J.H., and van der Kraak, G.J. (Ed). St Lucie Press, Delray Beach, Florida (1996).

Oikari, A. and Kunnamo-Ojala,T. (1987). Tracing of xenobiotic contamination in water with the aid of fish bile metabolites: A field study with caged rainbow trout *(Salmo gairdneri)*. Aquatic Toxicology 9(6):327-341. 5.1.2

Oikari, A., Fragoso, N., Leppänen, H., Chan, T., Hodson, P.V. (2002). Bioavailability to juvenile rainbow trout (*oncorynchus mykiss)* of retene and other mixed-function oxygenase-active compounds from sediment. Environmental Toxicology and Chemistry. 21: 121-128.

Shiu, W-Y., Ma, K-C., Varhackova, D. and Mackay, D. (1994). Chlorophenols and alkylphenols: A review and correlation of environmentally relevant properties and fate in an evaluative environment. Chemosphere. 29:1155-1224.

Tunkel, J., Howard, P.H., Boethling, R.S., Stiteler, W., Loonen, H. (2000). Predicting ready biodegradability in the Japanese Ministry of International Trade and Industry test. Environmental Toxicology and Chemistry. 19: 2478-2485.

US EPA (2000b). Estimation program interface (EPI) suite. U.S. Environmental Protection Agency Office of Pollution Prevention Toxics and Syracuse Research Corporation. Washington, D.C.

USEPA (2005). Human health risk assessment protocol for hazardous waste combustion facilities. HHRAP companion database. U.S. Environmental Protection Authority. Washington, D.C. http://www.epa.gov/epaoswer/hazwaste/combust/riskvol.htm#volume2.

Yaws, C.L. (2003). Yaws handbook of thermodynamic and physical properties of chemical compounds. Electronic Book, Knovel. Release date February 1, 2003.

This page left intentionally blank

Appendix 3: Data from JP on final effluent constituents and concentrations.

Appendix 3 contains data provided by Gunns Ltd and their consultants, in order to estimate the concentrations of constituents present in the final effluent. The following references are cited:

- JP (2005)c. Excel spreadsheet titled "Roger Effluent Loads & WWTP Design.Base.Case.15.06.05, worksheet FE.DOM. Prepared by Jaakko Pöyry Oy sent by Gunns Ltd 18/11/05. This spreadsheet details the estimated composition of dissolved organic matter (DOM) in Final Effluent. **Table A3.1 below**

- JP (2005)d. Excel spreadsheet titled "Roger NSI.Emissions.November.05.New." Three worksheets are cited in the report; "I-TEQ" - details the estimate for concentration of total PCDD/Fs at Bell Bay, "CPhs" – details the estimate for total chlorinated phenolics expected in the final effluent at Bell Bay, "Extractives" – details the estimates of final effluent concentrations for substances extracted from wood including resin and fatty acids. Prepared by Jaakko Pöyry Oy sent by Gunns Ltd 18/11/05. **Tables A3.2, A.3.3 and A3.4 below.**

- JP (2005)e. Excel spreadsheet titled "Effluent Loads and WWTP Design.HMS.Dec.05". One worksheet is cited in the report; "HMEms.New" - details the estimate for concentration of trace metals in the final effluent based on analysis of trace metals in plantation, native eucalypts and pine and the water balance of the Bell Bay mill. Prepared by Jaakko Pöyry Oy sent by Gunns Ltd 19/12/05.

Estimates in Tables A3.1-A3.3 and Sections A3.1 and A3.2 were provided as either a concentration in µg/L or as kg or g/Adt (air dried tonne). In order to convert kg/Adt to µg/L, it is assumed that the volume of effluent will be 22 kL/Adt. For example, to calculate the total organic acid concentration in final effluent from Table A3.1 below;

Estimated total resins:	0.097 kg/Adt
Volume of effluent:	22 kL/Adt
Estimated concentration	0.0044 kg/L = **4409 µg/L**

Table A3.1 – Estimated Composition of Dissolved Organic Matter (JP 2005c)
(items in bold were used in Appendix 1 to estimate final effluent concentrations)

Estimated Composition of Dissolved Organic Matter (DOM) in Final Effluent			
			kg/Adt
1. Total Estimated DOM			6.00
2. High Molecular Weight Substances (MW > 1000)	% of total	96.13	5.77
The HMWS comprise carbohydrates and oxidized, partly chlorinated lignin derivatives. Amount of phenolic elements in this material is very low or well below 5 %			
3. Low Molecular Weight Substances (MW < 1000)	% of total	3.87	0.23
The main substance groups are:			
Organic Acids*/	**% of total**	**42**	**0.097**
Aromatic Compounds/**	**% of total**	**5**	**0.012**
Neutral Compounds*/**	**% of total**	**53**	**0.123**

**/ Mostly acetic and other small molecular weight organic acids*
***/ Estimated amount of chlorinated phenolics is well below 50 % of total*
****/ Mostly methanol, ethanol, aldehydes, ketones, and sulfones*

References

Dahlman O.B., Reiman A.K., Stromberg L.M., Morck R.E.: Vol.78, No. 12 Tappi Journal, Dec.1995
Tana J. and Lehtinen K-J.: The Finnish Environment, Publ. No. 17, 1996

A3.1 – Estimated Composition of PCDD/PCDF (JP 2005d)

ASSESSMENT OF PCDD/PCDF EMISSIONS OF THE BELL BAY PULP MILL

1. PCDD/PCDF Emissions from BKP Mills using Elemental Chlorine Bleaching

1.1 Introductory Remarks

In the mid 1980's USEPA found detectable amounts of PCDD/DF in BKPM effluents originating from chlorine bleaching. As a consequence, the so-called "104 Mill Study" was implemented in the US in the late 1980's. Similar studies were implemented also in Canada and in Scandinavia. It was concluded that elemental chlorine based bleaching generated measurable amounts of PCDD/PCDF. Subsequently, the chemistry of chlorine bleaching was studied in detail to better understand the process conditions promoting the generation of PCDD/DF and to develop techniques to eliminate the emissions.

1.2 PCDD/DF Generation in Bleaching of Pulp by Using Elemental Chlorine

Based on the late 1980's and 1990's studies the average PCDD/PCDF-emissions from BKP mills were as follows:

Estimated I-TEQ Emissions	**g I-TEQ/a**	**BEKP Prod., Adt/a**	**micro-g I-TEQ/Adt**	
USA (USEPA/NCASI 104-Mill Study, 1989)	100	20000000	5	3 (early 1990's)
Canada (CPPA, 1991)	39.5	10400000	3.80	
Canada (Environment Canada, 1999)	11.4	11000000	1.04	
Sweden (Naturvardsverket, 1993)	7	6000000	1.17	
Typical Distribution of I-TEQ:	**% of total**	Avg. USA, Sweden, and Canada, micro-g/Adt		2.25
Gaseous Emissions (incl.power boilers)	20			
Liquid Effluents and Sludge	40			
Pulp	40			

The US and Canadian emissions in the late 1980's and 1991, resp. were clearly higher than the corresponding Canadian and Swedish emissions later in the 1990's. Then the Swedish mills and the Canadian mills already used more advanced technologies, including oxygen delignification and a higher share of chlorine dioxide of the total active chlorine in bleaching. Simultaneously, by the early 1990's, the US pulp industry had started using oxygen delignification and higher percentage share of chlorine dioxide in bleaching. This had resulted in a about 35-40 % reduction of the specific PCDD/DF emission approximately to the same level as in Canada in 1991. Hence, the specific emission used for the US mills is 3 micro-g/Adt.

2. PCDD/PCDF Emissions of Modern ECF Pulp Mills

The key process improvements to achieve additional reduction of PCDD/DF emissions are:
1. Modified cooking and two-stage oxygen delignification to achieve very low residual lignin in pulp going to final bleaching. The residual lignin in hardwood pulp as Kappa-number is currently 8-10. In the BAT-level mills 15 years ago the kappa number used to be about 12-15. The average additional reduction has been about 35

%.

2. Very efficient washing of unbleached pulp to minimise the carryover of black liquor and O2-stage solids to final bleaching. The brownstock washing efficiency in modern mills is about 99 % as compared to about 98.5 % in older mills. The carryover of black liquor and O2-stage solids containing possible dioxin precursors has been reduced by about 35 %.
3. 100 % substitution of chlorine dioxide for chlorine in final bleaching. In addition, chlorine dioxide generated in the most modern ClO2-plants contains only traces of by-product chlorine. Hence, the possibility of any substitution and addition reactions with the aromatic precursors of PCDD/PCDF's in O2-delignified pulp is minimal.to be 0.15- 0.2, whereas in modern mills it is well below 0.1. cannot be ruled out. The possible trace amounts of PCDD/PCDF generated in modern ECF bleaching may be estimated by assuming that there is a direct correlation between the improved key process variables and the generation of PCDD/PCDF.
4. Due to the low unbleached kappa, the total charge of chlorine per unit of O2-delignified pulp (so-called chlorine multiple) is at present considerably lower than in the BAT bleaching about 15 years ago. At that time the chlorine multiple used to be 0.15-0.2, whereas in modern mills it is well below 0.1.

As the result of the above process improvements, the modern ECF bleaching does not generate measureable amounts of polychlorinated dioxins and furans. However, the presence of PCDD/PCDF at concentrations below the detection limits cannot be ruled out. The possible trace amount of PCDD/PCDF generated in modern ECF bleaching may be estimated by assuming that there is a direct correlation between the improved key process variables and the generation of PCDD/PCDF.

Based on this approach the following "key process coefficients" as compared to the early 1990's technology could be used:

1. Reduction of PCDD/PCDF precursors due to modified cooking, two-stage oxygen delignification, and improved post oxygen washing: **k1 = 0.4** (35 % lower kappa and brownstock washing loss)
2. Elimination of the use of element chlorine in final bleaching and mitigation of chlorine multiple. **k2 = 0.25** (Active chlorine: Modern 40 kg/Adt (100 % ClO2, 2 % Cl2 in ClO2); early 1990's 50 kg/Adt (50%/50 % Cl2/ClO2, 20 % Cl2 in ClO2))
3. Combined Reduction Potential of k1 and k2, % : **90**

In addition to the process improvements, the external effluent treatment systems are also able to remove PCDD/DF more effectively than conventional treatment plants. The removal efficiency can be about 25-50 % better than before due to more efficient secondary clarification, which reduces the amount of biosolids in the final effluent.

The combined effect of the process factors would be about 90 % reduction of PCDD/PCDF generation in modern ECF bleaching as compared to BAT-level bleaching processes used about 15 years ago. Hence, the total I-TEQ generation in a modern BHKP mill could be in order of 10 % of the specific emissions measured in the 1990's in Sweden and in Canada

By assuming that the total emission is distributed between the pulp, liquid effluents, and gaseous emissions as presented above, the I-TEQ-emissions of the Bell Bay Mill producing 1100000 Adt/a is estimated as follows:

Estimated I-TEQ-Emissions from the Bell Bay BEKP Mill					
Total Emission, g I-TEQ/a			0.248	Balance Concentration	
Gaseous Emission, g I-TEQ/a			0.050	ng/Nm3	0.0058
	% of total	20		FG ,Nm3/Adt	7811
Final Effluent, g I-TEQ/a			0.074	pg/l	3.376

	% of total	40		Flow, kl/Adt	20
	Red.in ETP,%	25			
Pulp, g I-TEQ/a			0.099	micro-g/Adt	0.090
	% of total	40			
I-TEQ with Effluent Sludge, g/a			0.025	ng/BDkg	1.61
		Total,g I-TEQ/a	0.248		

References:

1. NCASI and USEPA: 104 Mill Study, 1989
2. RPDC Guidelines, October 1994
3. Generation of TCDD/TCDF as function of chlorine multiple (Axegard, 1988)
4. The aquatic environmental impact of pulping and bleaching operations-an overview, (Tana and Lehtinen, 1996)
5. Pulp & Paper Canada 93:9 (1992), pp.T239-T248
6. Pulp & Paper Canada 93:6 (1992), pp T157-T161
7. Unpublished mill measurements in Europe, Asia, South America, and Australia

A3.2 – Estimated Composition of Chlorinated Phenolic Substances (JP 2005d)

Generation of Chlorinated Phenolic Substances in Modern BEKP Mills

1. Introductory Remarks

The generation of chlorinated phenolic compounds in pulp bleaching has been known for many decades. Their environmental importance became more obvious in the late 1960 and 1970's after the accumulation of persistent chlorinated organics, especially pesticides, into various foodwebs became a global issue.

A small part of AOX comprises chlorinated phenolics. In conventional elemental chlorine bleaching of hardwood pulp typical ChP-emissions used to be about 20-30 g/Adt at unbleached kappa 15-20. The main chemical groups found were chlorinated phenols, vanillins, guaiacols, catechols, syringols, and syringealdehydes.

At low chlorine multiples (<0.1) mono- and di-chlorinated CPh compounds dominate, while at higher Cl-multiples tri- and tetra-chlorinated compounds become more dominant.

In addition to chlorine multiple the kappa number of unbleached pulp has a substantial impact on the formation of CPh. The generation of CPh seems to be directly proportional to the unbleached kappa number.

2. Impacts of Modern Pulping and Bleaching on Generation of Chlorinated Phenolics

2.1 Low Kappa Cooking and Oxygen Delignification

In the modern cooking process of hardwood pulps kappa numbers 15-20 can be achieved. An additional reduction of at least 50 % can then be achieved in the subsequent two-stage oxygen delignification. Consequently, the kappa number of oxygen delignified pulp to final bleaching is 8-10.

2.2 ECF Bleaching

100 % substitution of chlorine dioxide for chlorine results in a substantial reduction of the generation of chlorinated phenolics. The formation of polychlorinated phenolics is practically eliminated and the total emission is reduced by about 80 %, or in direct proportion to the reduction of chlorine charge on pulp.

The observed residual amounts of CPh in modern hardwood ECF mill effluents are well below 5 g/Adt.

This observation is consistent with the changes in the key process variables; ie. reduction of kappa by about 50 % and reduced chlorine charge by about 80 %. Hence the expected total reduction-% would be about 90 % as compared to conventional bleaching based on elemental chlorine.

3. Impact of Biological Effluent Treatment

Chlorinated phenolics are reduced further in biological treatment of the effluent. In conventional aerated lagoons the observed reduction efficiencies have been about 50-60 %, but in modern activated sludge plants the reduction efficiency is up to about 90 %. Part of the reduction is due to biological and physical-chemical degradation, while part is due to adsorption/absorption phenomena on the excess biosludge.

4. Estimated Emission of Chlorinated Phenolics in the Final Effluent

Based on the improvements in the key process variables and the observed efficiency of modern activated sludge plants it is estimated that the total load of chlorinated phenolics in the final effluent of the Bell Bay Pulp Mill would be in order of <0.5-1 g CPh/Adt. **The estimated average load is 0.5 g/Adt, or about 25 micro-g/l in the effluent pumped to Bass Strait.**

5. References

1. Jaakko Poyry Internal Data Banks
2. Tana, J. and Lehtinen, K-J, 1996: The Aquatic Environmental Impact of Pulping and Bleaching Operations-An Overview
The Finnish Environment Publ. No. 17, 1996
3. SSVL, 1978; Klorid i Atervinningssystem
4. Wong A., 1980: Chlorinated Organics in Kraft Bleaching Effluents. ICWW, Stockholm, 1980

Table A3.2– Estimated Composition of Resin and Fatty Acids (JP 2005d)

Extractives Balance during *Pinus radiata* Production					Draft/HJ	08.06.05
Avg. Bleached Pulp Production, Adt/d						2040
Yields, %	Blowline	47	Post-O2	45.4	Bleached	43.8
Pine Chips to Digester, BDt/d						4219
Input of Extractives						
Total Extractives in Fresh Wood, % of BD weight						3.5
Total Extractives in Fresh Wood, t/d						147.669
Extractives in Chips to Digester, kg/Adt			Storage Losses, %		20	57.902

	Resin Acids, kg/Adt	% of total	45	26.056
	Fatty Acids, kg/Adt	% of total	35	20.266
	Neutral Extractives, kg/Adt	% of total	10	5.79
	Volatile Extractives, kg/Adt	% of total	10	5.79
Extractives to Weak Black Liquor, Turpentine System and to NCG System				
Resin Acids, kg/Adt		Washing Loss, %	1	25.795
Fatty Acids, kg/Adt		Washing Loss, %	1	20.063
Neutral Extractives, kg/Adt		Washing Loss, %	1	5.732
Volatile Extractives, kg/Adt		To Turpentine System, %	90	0.579
Volatile Extractives to Turpentine Decanter, kg/Adt				5.211
Raw Turpentine, kg/ADt		Separation efficiency, %	90	4.69
Turpentine to NCG-Gas System, kg/Adt				1.1
Resin Acids, Fatty Acids, and Neutral Extractives to Tall Oil System				
Crude Soap to Off-Site Tall Oil Cooking, kg/Adt		Soap Separation Efficiency, %	80	41.272
Crude Soap to Recovery Boiler with HBL, kg/Adt				10.318
Extractives Carry-over to Bleaching and Process Effluent				
Resin Acids, kg/Adt				0.261
Fatty Acids, kg/Adt				0.203
Neutral Extractives, kg/Adt				0.058
Total to Final Bleaching, kg/Adt				0.521
Removal by Chemical Oxidation in O2- and Do-Stages, kg/Adt		Efficiency,%	50	0.261
Discharge of Extractives to Raw Process Effluent, kg/ADt				0.261
	Resin Acids, kg/Adt			0.117
	Fatty Acids, kg/Adt			0.091
	Neutral Extractives, kg/Adt			0.026
Removal in the Effluent Treatment Plant				
Resin Acids, kg/Adt		Removal Efficiency,%	95	0.111
Fatty Acids, kg/Adt		Removal Efficiency,%	95	0.087
Neutral Extractives, kg/Adt		Removal Efficiency,%	95	0.025
Extractives in the Final Effluent and at the Border of the Initial Mixing Zone in the Sea				
Final Effluent Flow, kl/d				47744
Resin Acids in Final Effluent, kg/d				11.96
Resin Acids in Final Effluent, mg/l				**0.25**
Theoretical Maximum Conc. of Resin Acids in Sea Water, micro-g/l */		Dil.Factor	100	2.5

Fatty Acids in Final Effluent, kg/d	9.3
Fatty Acids in Final Effluent, mg/l	**0.19**
Fatty Acids in Sea Water, micro-g/l	1.95
Neutral Extractives in Final effluent, kg/d	2.7
Neutral Extractives in Final effluent, mg/l	0.06
Neutral Extractives in Sea Water, micro-g/l	0.56

*/ Target "No-Effect" concentration for resin acids < 5 micro-g/l (ref. J.Tana and K-J Lehtinen, 1996)

Extractives in the Effluent and Sea Water over a 5-d Pinus radiata Campaign

Total HRT in the Effluuent Treatment Plant, days	Total Volume, kl	135000	2.83
Step Signal Concentration of Total Extractives in Raw Effluent, mg/l			11.13

	Total Extractives	Resin Acids	
Day	Tot.Extr.FE,mg/l	FE, mg RA/l*/	SW,micro-g RA/l
1	0.165	0.074	0.744
2	0.282	0.127	1.268
3	0.363	0.164	1.635
4	0.421	0.189	1.894
5	0.461	0.208	2.076
6	0.324	0.146	1.459
7	0.16	0.072	0.721
8	0.056	0.025	0.25
9	0.014	0.006	0.061
10	0.002	0.001	0.01

*/ FE= Final effluent, SW= Sea Water

Table A3.3– Estimated Composition of Metals (JP 2005e)

Heavy Metal Balances Based on Tasmanian Wood Analysis 15.12.05/HJ 16B0104

1. Heavy Metal Contents of Wood

Element	**Sample** DS0201, Plant.Euca	DS0202, Native Euca	DS0203, *P. radiata*
Mo, mg/kg	0.1	0.1	0.1
P, mg/kg	57	37	74
Sb, mg/kg	0.1	0.1	0.1
Si, mg/kg	16	20	37
Sn, mg/kg	1	1	1
Al, mg/kg	17	17	39
As, mg/kg	0.1	0.2	0.2

B, mg/kg	2.4	3	1.7
Ba, mg/kg	1.4	1.3	0.5
Be, mg/kg	0.1	0.1	0.1
Cd, mg/kg	0.1	0.1	0.1
Co, mg/kg	0.1	0.1	0.2
Cr, mg/kg	2	1.1	1.4
Cu, mg/kg	0.9	1	1.4
Fe, mg/kg	30	28	53
Mn, mg/kg	27	15	53
Ni, mg/kg	1.7	1.6	1.9
Pb, mg/kg	0.1	0.2	0.2
Se, mg/kg	0.5	0.5	0.5
V, mg/kg	0.1	0.1	0.1
Zn, mg/kg	1.8	2.4	5.6
Hg, mg/kg	0.02	0.02	0.02
Ca, mg/kg	284	148	323
K, mg/kg	479	227	542
Mg, mg/kg	106	74	148
Na, mg/kg	114	62	27
Moisture, %	8	8	10

2. Input with Wood, g/ADBt			
Yield, % (bleached pulp)	52	47	44
Element	**Sample** DS0201, Plant.Euca	DS0202, Native Euca	DS0203, *P. radiata*
Mo, g/Adt	0.188	0.208	0.227
P, g/Adt	107.232	77.012	168.182
Sb, g/Adt	0.188	0.208	0.227
Si, g/Adt	30.100	41.628	84.091
Sn, g/Adt	1.881	2.081	2.273
Al, g/Adt	31.982	35.384	88.636
As, g/Adt	0.188	0.416	0.455
B, g/Adt	4.515	6.244	3.864
Ba, g/Adt	2.634	2.706	1.136
Be, g/Adt	0.188	0.208	0.227
Cd, g/Adt	0.188	0.208	0.227
Co, g/Adt	0.188	0.208	0.455
Cr, g/Adt	3.763	2.290	3.182
Cu, g/Adt	1.693	2.081	3.182
Fe, g/Adt	56.438	58.279	120.455
Mn, g/Adt	50.794	31.221	120.455
Ni, g/Adt	3.198	3.330	4.318
Pb, g/Adt	0.188	0.416	0.455
Se, g/Adt	0.941	1.041	1.136
V, g/Adt	0.188	0.208	0.227
Zn, g/Adt	3.386	4.995	12.727
Hg, g/Adt	0.038	0.042	0.045

Ca, g/Adt	534.281	308.048	734.091
K, g/Adt	901.129	472.479	1231.818
Mg, g/Adt	199.415	154.024	336.364
Na, g/Adt	214.465	129.047	61.364

3. Distrubution between Ubl. Pulp and Causticizing Solids

Outlet from POW, % of Gross Input	10	Steady State HMs to POW,% of net input	159
Outlet from Causticizing,% of Gross Input	70		

Element	**Sample** DS0201, Plant.Euca		DS0202, Native Euca		DS0203, *P. radiata*	
	Ubl.Pulp	Caust.Solids	Ubl.Pulp	Caust.Solids	Ubl.Pulp	Caust.Solids
Mo, %	15.9	84.1	15.9	84.1	15.9	84.1
P, %	15.9	84.1	15.9	84.1	15.9	84.1
Sb, %	15.9	84.1	15.9	84.1	15.9	84.1
Si, %	15.9	84.1	15.9	84.1	15.9	84.1
Sn, %	15.9	84.1	15.9	84.1	15.9	84.1
Al, %	15.9	84.1	15.9	84.1	15.9	84.1
As, %	15.9	84.1	15.9	84.1	15.9	84.1
B, %	15.9	84.1	15.9	84.1	15.9	84.1
Ba, %	15.9	84.1	15.9	84.1	15.9	84.1
Be, %	15.9	84.1	15.9	84.1	15.9	84.1
Cd, %	15.9	84.1	15.9	84.1	15.9	84.1
Co, %	15.9	84.1	15.9	84.1	15.9	84.1
Cr, %	15.9	84.1	15.9	84.1	15.9	84.1
Cu, %	15.9	84.1	15.9	84.1	15.9	84.1
Fe, %	15.9	84.1	15.9	84.1	15.9	84.1
Mn, %	15.9	84.1	15.9	84.1	15.9	84.1
Ni, %	15.9	84.1	15.9	84.1	15.9	84.1
Pb, %	15.9	84.1	15.9	84.1	15.9	84.1
Se, %	15.9	84.1	15.9	84.1	15.9	84.1
V, %	15.9	84.1	15.9	84.1	15.9	84.1
Zn, %	15.9	84.1	15.9	84.1	15.9	84.1
Hg, %	15.9	84.1	15.9	84.1	15.9	84.1
Ca, %	15.9	84.1	15.9	84.1	15.9	84.1
K, %						
Mg, %	15.9	84.1	15.9	84.1	15.9	84.1
Na, %						

4. Element Concentration in Raw Effluent

	Plantation Euca		**Native Euca**		*Pinus radiata*	
Raw Effl. Amount, kl/Adt	22.991		23.812		23.401	
Solids Amount, t/Adt		0.04		0.04		0.04
Element	Raw Effluent	Causticizing Solids	Raw Effluent	Caust. Solids	Raw Effluent	Caust. Solids
Mo, micro-g/l	1.299	3957	1.387	4378	1.542	4780

P, micro-g/l	740.334	2255285	513.360	1619697	1140.801	3537157
Sb, micro-g/l	1.299	3957	1.387	4378	1.542	4780
Si, micro-g/l	207.813	633063	277.492	875512	570.400	1768579
Sn, micro-g/l	12.988	39566	13.875	43776	15.416	47799
Al, micro-g/l	220.801	672629	235.868	744185	601.233	1864177
As, micro-g/l	1.299	3957	2.775	8755	3.083	9560
B, micro-g/l	31.172	94959	41.624	131327	26.208	81259
Ba, micro-g/l	18.184	55393	18.037	56908	7.708	23900
Be, micro-g/l	1.299	3957	1.387	4378	1.542	4780
Cd, micro-g/l	1.299	3957	1.387	4378	1.542	4780
Co, micro-g/l	1.299	3957	1.387	4378	3.083	9560
Cr, micro-g/l	25.977	79133	15.262	48153	21.583	66919
Cu, micro-g/l	11.689	35610	13.875	43776	21.583	66919
Fe, micro-g/l	389.650	1186992	388.489	1225717	817.060	2533369
Mn, micro-g/l	350.685	1068293	208.119	656634	817.060	2533369
Ni, micro-g/l	22.080	67263	22.199	70041	29.291	90819
Pb, micro-g/l	1.299	3957	2.775	8755	3.083	9560
Se, micro-g/l	6.494	19783	6.937	21888	7.708	23900
V, micro-g/l	1.299	3957	1.387	4378	1.542	4780
Zn, micro-g/l	23.379	71220	33.299	105061	86.331	267677
Hg, micro-g/l	0.260	791	0.277	876	0.308	956
Ca, micro-g/l	3688.682	11236861	2053.440	6478789	4979.442	15439214
K, micro-g/l						
Mg, micro-g/l	1376.762	4194040	1026.720	3239395	2281.602	7074315

5. Removal to EBS and Concentration in Final Effluent

EBS-Amount, BDkg/Adt			3.85	**Plant. Euca**	**Native Euca**	***P.radiata***
				Conc.in FE	**Conc.in FE**	**Conc.in FE**
Element		Element in EBS	In EBS,g/Adt	**micro-g/l**	**micro-g/l**	**micro-g/l**
Mo, mg/kg	(EBS)	1	0.004	1.132	1.226	1.377
P, mg/kg	(EBS)			740.334	513.360	1140.801
Sb, mg/kg	(EBS)	1	0.004	1.132	1.220	1.377
Si, mg/kg	(EBS)			207.813	277.492	570.400
Sn, mg/kg	(EBS)	10	0.038	11.316	12.202	13.773
Al, mg/kg	(EBS)			220.801	235.868	601.233
As, mg/kg	(EBS)	0.9	0.003	1.148	2.624	2.935
B, mg/kg	(EBS)	20	0.077	27.827	38.279	22.921
Ba, mg/kg	(EBS)	10	0.038	16.511	16.364	6.065
Be, mg/kg	(EBS)	0.5	0.002	1.215	1.304	1.459
Cd, mg/kg	(EBS)	2	0.008	0.964	1.053	1.213
Co, mg/kg	(EBS)	1	0.004	1.132	1.220	2.919
Cr, mg/kg	(EBS)	10	0.038	24.304	13.590	19.939

Cu, mg/kg	(EBS)	10	0.038	10.017	12.202	19.939	
Fe, mg/kg	(EBS)			389.650	388.489	817.060	
Mn, mg/kg	(EBS)			350.685	208.119	817.060	
Ni, mg/kg	(EBS)	10	0.038	20.408	20.527	27.648	
Pb, mg/kg	(EBS)	1	0.004	1.132	2.608	2.919	
Se, mg/kg	(EBS)	1	0.004	6.327	6.770	7.544	
V, mg/kg	(EBS)	1	0.004	1.132	1.220	1.377	
Zn, mg/kg	(EBS)	10	0.038	21.706	31.627	84.688	
Hg, mg/kg	(EBS)	0.2	0.001	0.226	0.244	0.275	
Ca, mg/kg	(EBS)			3688.682	2053.440	4979.442	
K, mg/kg	(EBS)						
Mg, mg/kg	(EBS)			1376.762	1026.720	2281.602	
Na, mg/kg	(EBS)						

Appendix 4: Background concentrations of metals in marine environments

Table A.4.1 provides information on typical marine water background concentrations of cadmium, mercury, and selenium found in Australia, and elsewhere. The information was obtained primarily from reviews. Although efforts have been made to avoid citing values from environments contaminated by anthropogenic sources interrogation of the primary literature has not been undertaken.

Table A.4.1 also contains the results of sediment and biota sampling at the outfall site for cadmium, mercury and selenium. Where available local (i.e. Tamar estuary and Bass Strait) background concentrations for sediment and biota are also included.

Table A4.1 Background concentrations of cadmium, mercury and selenium in the marine environment

Metal	Location	Species (where applicable)	Sample size	Mean	Range of values	Reference
Water µg/L						
Cadmium	Bell Bay mill – outfall site	NA[a]	16		<0.2-0.4 (<0.2, n=14)	Aquenal (2005)
	Marine Australia	NA	NR[b]		0.002-0.7	Apte et al 1998 Mackey (1984)
	Costal waters Australia	NA	NR		0.002-0.07	DEH (1995)
	Pacific ocean (3-5000 m)	NA	NR		0.0008-0.012	Moore & Ramamoorthy (1984)
	Marine world	NA	NR		0.001-1.1	Bruland (1983)
	Open-ocean sea water	NA	NR		0.01-0.1	Korte (1983)
	Ocean	NA	NR	0.06		Niragu (1980)
	Sea water	NA	NR	<0.1		Korte (1983)
	Marine USA	NA	NR		0.01-0.2	Protho (1993)
	Coastal sea water	NA	NR	<0.05		Korte (1983)
	Open-ocean surface	NA	NR	0.005		Boyle et al (1976)
	North Atlantic Ocean	NA	NR	0.06	0.02-0.15	Eaton (1976)
	North Atlantic Ocean (50-100m)	NA	NR		0.02-0.15	Moore & Ramamoorthy (1984)
	Indian Ocean (surface)	NA	NR	0.07		Moore & Ramamoorthy (1984)
	Estuary Australia	NA	NR		0.002-0.026	Mackey et al (1996) CSIRO (1996)
	Estuary Australia	NA	NR		0.51-1.2	Higgins & Mackey (1987)
Mercury	Coastal waters Australia	NA	NR		<0.001-0.02	DEH (1995)
	Open ocean	NA	NR		0.0005-0.003	WHO (1989)
	Open ocean	NA	NR		0.002-0.03	Moore & Ramamoorthy (1984)
	Open ocean	NA	NR	0.0053	0.0031-0.0075	Nishimura et al (1983)
	Open ocean	NA	NR		<0.01	Fitzgerald (1979)
	Costal sea water	NA	NR		0.002-0.015	WHO (1989)

Metal	Location	Species (where applicable)	Sample size	Mean	Range of values	Reference
Water µg/L (cont'd)						
Mercury (cont'd)	Coastal sea water	NA	NR		<0.02	Fitzgerald (1979)
	Estuary Australia	NA	NR		0.0017	CSIRO (1996)
	Estuary World	NA	NR		0.0007-0.003	CSIRO (1996)
	Estuarine seawater	NA	NR		<0.05	Fitzgerald (1979)
Selenium	Bell Bay mill – outfall site	NA	16		<2-2 (< 2 n=13)	Aquenal (2005)
	Australia	NA	NR		< 0.5	ANZECC (2000b)
	Costal waters Australia	NA	NR		<0.01-0.08	DEH (1995)
	Sea water worldwide	NA	NR		0.009-0.045	Frost & Ingvolstad (1975) Ebens & Shacklette (1982)
	Ocean	NA	NR	0.09		Schutz & Turekian (1965)
	Sea water worldwide	NA	NR		0.09-0.45	Whittle et al (1977)
	Sea water California	NA	NR		0.058-0.08	Robberect & von Grieken (1982)
	Sea water Japan	NA	NR		0.04-0.08	Robberect & von Grieken (1982)
	Sea water worldwide	NA	NR		0.09-<6.0	NAS (1976)
Sediment (mg/kg sediment)						
Cadmium	Bell Bay mill – outfall site	NA	8		<1-2 (<1, n=7)	Aquenal (2005)
	Marine	NA	NR		0.03-1	Korte (1983)
	North Atlantic	NA	NR		0.04-1.88	Fleisher (1974)
	North Atlantic	NA	NR		0.13-0.21	Eaton (1976)
Mercury	Bell Bay mill – outfall site	NA	8		<0.1-0.1 (<0.1, n=5)	Aquenal (2005)
	Marine	NA	NR		0.05-0.08	Hamasaki et al (1995)
	Ocean	NA	NR		0.02-0.1	WHO (1989)
Selenium	No literature references were found					

Metal	Location	Species (where applicable)	Sample size	Mean	Range of values	Reference
Biota (mg/kg tissue)						
Cadmium	Bell Bay mill – outfall site	Flathead	8	NA	<1 mg/kg in all sampled fish	Aquenal (2005)
	Bell Bay mill – outfall site	Common gurnard perch.	7	NA	<1 mg/kg in all sampled fish	Aquenal (2005)
	Bell Bay mill – outfall site	Blue-throated wrasse.	3	NA	<1 mg/kg in all sampled fish	Aquenal (2005)
Mercury	Bell Bay mill – outfall site	Flathead	8		<0.1-0.4	Aquenal (2005)
	Bass Strait – Bell Bay mill – outfall site	Common gurnard perch.	7		<0.1-0.6	Aquenal (2005)
	Bell Bay mill – outfall site	Blue-throated wrasse.	3		<0.1-0.2	Aquenal (2005)
	Upper Tamar	Flathead	12	0.03	0.02-0.07	Dix et al (1975)
	Middle Tamar	Flathead	12	0.04	0.03 -0.07	Dix et al (1975)
	Lower Tamar	Flathead	12	0.06	0.05-0.10	Dix et al (1975)
	Anderson Bay	Flathead	12	0.09	0.02-0.33	Dix et al (1975)
	Georges Bay	Flathead	12	0.05	0.02-0.10	Dix et al (1975)
	Great Oyster Bay	Flathead	12	0.09	0.05-0.20	Dix et al (1975)
	Schouten Passage	Flathead	12	0.06	0.02-0.12	Dix et al (1975)
	Spring Bay	Flathead	12	0.11	0.03 -0.22	Dix et al (1975)
Selenium	Bell Bay mill – outfall site	Flathead	8	NA	< 5 mg/kg in all sampled fish	Aquenal (2005)
	Bell Bay mill – outfall site	Common gurnard perch.	7	NA	< 5 mg/kg in all sampled fish	Aquenal (2005)
	Bell Bay mill – outfall site	Blue-throated wrasse.	3	NA	< 5 mg/kg in all sampled fish	Aquenal (2005)

[a] NA = not applicable

[b] NR = not reported

A4.1 References

ANZECC (2000b). Australian and New Zealand Guidelines for Fresh and Marine Water Quality. Volume 2, Aquatic Ecosystems –Rationale and Background Information. Ch. 8.3 Toxicants. Australian and New Zealand Environment and Conservation Council. October 2000 (www.deh.gov.au/water/quality/nwqms/volume2.html).

Apte, S.C., Bately, G.E., Szymczak, R., Rendell, P.S., Lee, R. and Waite, T.D. (1998). Baseline trace metal concentrations in New South Wales coastal waters. Marine and Freshwater Research 49:203-214 as cited in ANZECC (2000b). Australian and New Zealand Guidelines for Fresh and Marine Water Quality. Volume 2, Aquatic Ecosystems –Rationale and Background Information. Ch. 8.3 Toxicants. Australian and New Zealand Environment and Conservation Council. October 2000 (www.deh.gov.au/water/quality/nwqms/volume2.html).

Aquenal (2005). Aquenal (2005). Gunns Ltd Pulp Mill. Marine biological and pollutant survey at the proposed outfall site.

Boyle, E.A., Scalter, F. and Edmond, J.M. (1976). On the marine geochemistry of cadmium. Nature 263:42-44 as cited in WHO (1992). EHC 135 –Cadmium environmental aspects (http://www.inchem.org/documents/ehc/ehc/ehc135.htm).

Bruland, K.W. (1983). Trace elements in seawater. In Chemical oceanography, Vol 8, eds JP Riley & R Chester, Academic Press, London, 157-220 as cited in ANZECC (2000b). Australian and New Zealand Guidelines for Fresh and Marine Water Quality. Volume 2, Aquatic Ecosystems –Rationale and Background Information. Ch. 8.3 Toxicants. Australian and New Zealand Environment and Conservation Council. October 2000 (www.deh.gov.au/water/quality/nwqms/volume2.html).

CSIRO (1996). Port Phillip Bay as cited in ANZECC (2000). Australian and New Zealand Guidelines for Fresh and Marine Water Quality. Volume 2, Aquatic Ecosystems –Rationale and Background Information. Ch. 8.3 Toxicants. Australian and New Zealand Environment and Conservation Council. October 2000 (www.deh.gov.au/water/quality/nwqms/volume2.html).

DEH (1995). The State of the Marine Environment Report for Australia Technical Annex 2: Heavy metals and tributyltin in Australian coastal and estuarine waters. Department of the Environment, Sport & Territories, Canberra.

Dix, T., G., Martin, A., Ayling, G.M., Wilson, K.C. and Ratowsky, D.A. (1975). Sand Flathead (Platycephalus bassensis), an indicator species for mercury pollution in Tasmanian waters. Marine Pollution Bulletin: 6(9) 142-143.

Eaton, A. (1976). Marine geochemistry of cadmium. Marine Chem. 4: 141-154 as cited in Fergusson, J.E. (1990) The Heavy Elements: Chemistry, Environmental Impact and Health Effects. Pergamon Press, Headington Hill Hall, Oxford. P. 178.

Ebens, R.J. and Shacklette, H.T. (1982). Geochemistry of some rocks, mine spoils, stream sediments, soils, plants, and waters in the western energy region of the conterminous United States. U.S. Geol. Surv. Prof. Paper 1237. U.S. Govt. Printing Off., Washington D.C. as cited in Eisler, R (2000). Handbook of Chemical Risk Assessment Health Hazards to Humans, Plants, and Animals. Volume 1. Metals. Chapter 31 –Selenium. Lewis Publishers. Boca Raton, Florida.

Fitzgerald, W.F. (1979). Distribution of mercury in natural waters. P161-173 in JO Nriagu (ed.). The biogeochemistry of Mercury in the Environment. Elsevier/North-Holland Biomedical Press, NY as cited in Eisler, R (2000). Handbook of Chemical Risk Assessment Health Hazards to Humans, Plants, and Animals. Volume 1. Metals. Chapter 5 –Mercury. Lewis Publishers. Boca Raton, Florida.

Fleisher, M., Arofim, A.F., Fassett, D.W., Hammond, P., Shacklette, H.T., Nisbet, I.C.T. and Epstein, S. (1974). Environmental impact of cadmium: A review by the panel on hazardous trace substances. Environmental Health Perspectives 7:253-323 as cited in Fergusson, J.E. (1990). The Heavy Elements: Chemistry, Environmental Impact and Health Effects. Pergamon Press, Headington Hill Hall, Oxford. P. 177.

Frost, D.V. and Ingvolstad, D. (1975). Ecological aspects of selenium and tellurium in human and animal health. Chem. Scr. 8A:96-107 as cited in Eisler, R (2000). Handbook of Chemical Risk Assessment Health Hazards to Humans, Plants, and Animals. Volume 1. Metals. Chapter 31 – Selenium. Lewis Publishers. Boca Raton, Florida.

Hamasaki, T., Nagaawe, H., Yoshioka, Y. and Sato, T. (1995). Formation, distribution and ecotoxicity of methylmetals of tin, mercury, and arsenic in the environment. Crit Rev Environ Sci Technol 25: 45-91 as cited in Eisler, R (2000). Handbook of Chemical Risk Assessment Health Hazards to Humans, Plants, and Animals. Volume 1. Metals. Chapter 5 –Mercury. Lewis Publishers. Boca Raton, Florida.

Higgins, H.W. and Mackey, D.J. (1987). Role of *Ecklonia radiata* (C. Ag.) J. Agardh in determining trace metal availability in coastal. I. Total trace metals. Australian Journal of Marine and Freshwater Research 38:307-316 as cited in ANZECC (2000). Australian and New Zealand Guidelines for Fresh and Marine Water Quality. Volume 2, Aquatic Ecosystems –Rationale and Background Information. Ch. 8.3 Toxicants. Australian and New Zealand Environment and Conservation Council. October 2000 (www.deh.gov.au/water/quality/nwqms/volume2.html)

Korte, F. (1983). Ecotoxicology of cadmium: General review. Exotoxicol Environ Safety 7:3-8 (as cited in Eisler, R (2000). Handbook of Chemical Risk Assessment. Health Hazards to Humans, Plants, and Animals. Volume 1. Metals. Lewis Publishers, CRC Press, Boca Raton, Florida).

Korte (1983). Ecotoxicology of cadmium: General overview. Ecotoxicol. Environ. Saf. 7: 3-8 (as cited in WHO (1992). EHC 135 –Cadmium environmental aspects). (http://www.inchem.org/documents/ehc/ehc/ehc135.htm).

Mackey, D.J. (1984). Trace metals and productivity of shelf waters off north west Australia. Australian Journal of Marine and Freshwater Research 35:505-516 as cited in ANZECC (2000b). Australian and New Zealand Guidelines for Fresh and Marine Water Quality. Volume 2, Aquatic Ecosystems –Rationale and Background Information. Ch. 8.3 Toxicants. Australian and New Zealand Environment and Conservation Council. October 2000 (www.deh.gov.au/water/quality/nwqms/volume2.html).

Moore, J.W. and Ramamoorthy, S. (1984). Heavy metals in natural waters: Applied monitoring and impact assessment: Springer-Verlag as cited in Fergusson, J.E. (1990). The Heavy Elements: Chemistry, Environmental Impact and Health Effects. Pergamon Press, Headington Hill Hall, Oxford. P. 178-179.

NAS (1976). Selenium. Comm. Med. Biol. Eff. Environ Poll., Div. Med. Sci., Natl. Res. Counc., National Academy of Sciences, Washington, D.C. as cited in Eisler, R (2000). Handbook of Chemical Risk Assessment Health Hazards to Humans, Plants, and Animals. Volume 1. Metals. Chapter 31 –Selenium. Lewis Publishers. Boca Raton, Florida.

Niragu, J.O. (1980). Cadmium in the Environment. Part 1: Ecological Cycling. John Wiley, NY as cited in Eisler, R. (2000). Handbook of Chemical Risk Assessment. Health Hazards to Humans, Plants, and Animals. Volume 1. Metals. Lewis Publishers, CRC Press, Boca Raton, Florida.

Nishimura, M., Konishi, S., Matsunaga, K., Hata, K. and Kosuga, H. (1983). Mercury concentration in the ocean. Jour Oceanogr Soc Japan 39:2951-3300 as cited in Eisler, R (2000). Handbook of Chemical Risk Assessment Health Hazards to Humans, Plants, and Animals. Volume 1. Metals. Chapter 5 –Mercury. Lewis Publishers. Boca Raton, Florida.

Prothro (1993). as cited in ANZECC (2000). Australian and New Zealand Guidelines for Fresh and Marine Water Quality. Volume 2, Aquatic Ecosystems –Rationale and Background Information. Ch. 8.3 Toxicants. Australian and New Zealand Environment and Conservation Council. October 2000 (www.deh.gov.au/water/quality/nwqms/volume2.html)

Raspor, B. (1980). Distribution and speciation of cadmium in natural waters as cited in Nriagu, J.O. Ed. (1980). Cadmium in the environment. Part 1. Ecological Cycling. Wiley P 147-236 as cited in Fergusson, J.E. (1990). The Heavy Elements: Chemistry, Environmental Impact and Health Effects. Pergamon Press, Headington Hill Hall, Oxford. P. 178.

Robberecht, H. and Von Grieken, R. (1982). Selenium in environmental waters: determination, speciation and concentration levels. Talanta 29: 823-844 as cited in Eisler, R (2000). Handbook of Chemical Risk Assessment Health Hazards to Humans, Plants, and Animals. Volume 1. Metals. Chapter 31 –Selenium. Lewis Publishers. Boca Raton, Florida.

Schutz, D.F. and Turekian, K.K. (1965). The investigation of geographical and vertical distribution of several trace elements in seawater using neutron activation analysis. Geochim Cosmochim Eta 29:259-313 as cited in ATSDR (2003). Toxicological Profile for Selenium. Agency for Toxic Substances and Disease Registry (http://www.atsdr.cdc.gov/toxprofiles/tp92.html)

Whittle, K.J., Hardy, R., Holden, A.V., Johnston, R. and Pentreath, R.J. (1977). Occurrence and fate of organic and inorganic contaminants in marine animals. Ann. N.Y. Acad. Sci 298:47-79 as cited in Eisler, R (2000). Handbook of Chemical Risk Assessment Health Hazards to Humans, Plants, and Animals. Volume 1. Metals. Chapter 31 –Selenium. Lewis Publishers. Boca Raton, Florida.

WHO (1989). EHC 86 -Mercury environmental aspects. World Health Organization. (http://www.inchem.org/documents/ehc/ehc/ehc086.htm).

This page left intentionally blank

Appendix 5: Determination of fish consumption and dioxin fish content

A5.1 Review and selection of fish consumption rates

The daily intake or dose of dioxin from the consumption of fish caught by recreational anglers requires knowledge of the amount of fish ingested by people as an average intake (i.e. consumption rate of fish as kg fish/person/month refer to Equation 3, Section 6). In selecting conservative yet realistic maximum fish consumption rates for Tasmanians the following information sources were consulted:

- Australian Bureau of Statistics National Nutrition Survey 1995 (1999),
- enHealth Exposure Assessment Handbook (2003),
- US EPA Exposure Factors Handbook (1997) and
- US EPA Methodology for Deriving Ambient Water Quality Criteria for the Protection of Human Health (2000).

Table A5.1 is a comparison of fish consumption averages for Australia and USA included is data for Tasmania. Fish consumption rates for the risk assessment were sourced from the latest Australian National Nutrition Survey conducted in 1995 and 1996 and reported by the Australian Bureau of Statistics in 1999 (ABS 1999). Data in the survey was obtained predominantly from 24 hour recall of foods consumed and a food frequency questionnaire. The survey was conducted across all states and the data was reported by the ABS according to age, region, economic indicators and birthplace.

Data from ABS (1999) is available as:

- Mean daily intake per person and reported as total 'fish and seafood products and dishes' with the following sub-categories:
    - fin fish (excluding canned),
    - crustacean and molluscs (excluding canned),
    - packed (canned and bottled) fish and seafood,
    - fish and seafood products,
    - mixed dishes with fish and seafood as the major component, or

Fish consumption rates used in the risk assessment are total fish product consumptions reported by ABS (1999) for 'fish and seafood products and dishes' and are therefore inclusive of consumption of the sub-groups named above. For the site in question it could be argued that only

the consumption of fin-fish is relevant and using data for total fish product and meal consumption will over estimate likely intake of fish caught in the vicinity of the outfall. For an adult Tasmanian finfish consumption is 27% of the total consumption of seafood in all sub-categories.

The data included in Tables A5.1 (average daily consumption) and A5.2 (serve size) are for the population groups with the largest mean or maximum consumption for all fish and seafood products reported in ABS (1999). The 95th percentile was calculated by Toxikos based on the standard error for the mean provided by ABS (1999). The ABS considers mean consumptions with relative standard errors greater than 25% are not reliable and should be used with caution. This applies to the child data in Tables A5.1 and A5.2, nevertheless the data were used in the risk assessment because no other information sources consulted contained data for 2 – 3 year olds. Information is provided on US population fish consumption for contextual comparison. It should be noted that the 95th percentile is not used in the risk assessment calculations but rather the maximum, the 95th percentile is only provided for contextual information.

Table A5.1: Average daily fish consumption rates for Australia & US

Reference	Comments	Mean	95%ile	Max
		(g/person/day)		
General population				
ABS (1999)	2-3 yr female (Australian)	**6.5** [a]	**10**	**12**[c]
ABS (1999)	2-3 yr male (Australian)	**6.9** [a]	**11**	**12**[c]
ABS (1999)	45-60 yr male (Australian)	33	39	42
ABS (1999)	Australian adult (>19 yr)	26	27	28
ABS (1999)	Tasmanian adult (>19 yr)	**31**	**46**	**54**[c]
US EPA (1997)	0-9 yr females	6.1	17.3	-
US EPA (1997)	0-9 yr males	6.3	15.8	-
US EPA (1997)	General population	20.1	53	-
Recreational fishers				
enHealth (2003)	Australian [b]	**100** [b]	-	-
US EPA (1997)	60-69 yr male anglers	24.4	61.1	-
US EPA (1997)	Marine anglers	7.2	26.0	-
US EPA (1997)	Freshwater anglers	17	-	-
US EPA (1997)	General fishers	29.4	-	-
US EPA (2000)	Subsistence fishers	142.4	-	-

[a] Relative standard error is between 25 and 50% and ABS (1999) suggest the values be used with caution.
[b] This value is from a 1977 survey and the draft enHealth Exposure Assessment Handbook (enHealth 2003) notes the fish intake levels from this survey for other consumers were at variance with the National Dietary Survey of 1983 (and from the table also from the data of 1995, ABS 1999). enHealth (2003) state because the 1977 survey was conducted only over one week with a small population of subjects it has over estimated fish consumption. This value has therefore not been used in the primary risk assessment, it has however been incorporated into a sensitivity analysis (see the *Risk Characterisation* of Section 6.3.3).
[c] Data used in the primary risk assessment herein.

enHealth (2003) recommends exposure factors for fish consumption for leisure anglers but these are founded in an old survey conducted in 1977. This survey showed an average consumption of 513g per week for leisure anglers compared to 610g per week for the general population. The enHealth approximates the consumption to 100g per day. They state this is likely to be an overestimate due to the small population size and the short survey time (2003). In addition enHealth (2003) note the 1977 survey results are at odds with the data collected in the National Dietary Survey of 1983. Consequently the fish consumption value for leisure anglers cited by enHealth (2003) has not been used in the risk assessment; however it has been incorporated into a sensitivity analysis (see the Risk Characterisation Section 6.3.3).

A national recreational and indigenous fishing survey has been conducted (Henry and Lyle 2003) however an estimate of mean daily fish consumption for recreational anglers was not provided.

Fish consumption data for the USA is provided in Tables A5.1 and A5.2 for contextual purposes. The US EPA (1997) used information gleaned by the U.S. Department of Agriculture Continuing Study of Food Intakes by Individuals from 1989 to 1991 to calculate the daily consumption of fish from responses provided by participants over three consecutive days.

The US EPA also provides consumption rates for recreational fishermen and a brief summary of some of the major studies supporting the data included in Table A5.1 is below.

West et al. (1989), using short term and usual eating survey questionnaires, found general recreational fishermen consumed approximately 29.4g/d of which 14g/d was recreational catch.

The National Marine Fisheries Service completed a series of telephone and direct interviews to determine the number of fishers who consume ocean fish (NMFS 1993) with age group analysis performed by Javitz (1980). The study required households to record any fish meals during a month; 94% of the population were found to consume fish. The data for the age group with the highest fish consumption (60 – 69 year old) are presented in Table A5.1 under the US EPA (1997) entry.

A default value for fish consumption in subsistence fisherman is assumed by the US EPA to be 142.4g/d (US EPA 2000). This is based on information obtained between 1994 and 1996 from the

U.S. Department of Agriculture Continuing Study of Food Intakes by Individuals. However prior to using defaults the US EPA recommends using local data, data reflecting a similar geographic area or population group, or the use of national surveys. The US subsistence fisherman data are not relevant to the present exposure scenario, especially given the large distance to nearest boat landing, weather conditions in Bass Strait and the existence of Australian and Tasmanian consumption data.

Size of fish meals:

Information on the size of individual fish meals for similar demographics as in Table A5.1 is in Table A5.2. The data has been taken from ABS (1999) and the US EPA exposure factors handbook (US EPA 1997). The US EPA information is based on an analysis performed by Pao et al. (1982) for consumption over three consecutive days. The data provided in the Table A5.2 is for the age group with the greatest consumption per eating occasion.

Table A5.2: Fish meal sizes.

Reference	Comments	Median	95%ile	Max
		(g)		
ABS (1999)	2-3 year old female (Australian)	47.5 [a]	56	61
ABS (1999)	2-3 year old male (Australian)	63.3 [a]	75	81
ABS (1999)	12-15 year old male (Australian)	148.0	167	176
ABS (1999)	Australian adult (>19 years)	100	101	102
ABS (1999)	Tasmanian adult (>19 years)	126.5	147	157
US EPA (1997)	3-5 year old male/females	70	170	240
US EPA (1997)	19-34 year old male	149	362	643
US EPA (1997)	General population	129	326	
US EPA (2000)	Subsistence fishers	142.4	-	-

[a] Relative standard error is between 25 and 50% and ABS (1999) suggests the values be used with caution.

Fish consumption data for the risk assessment:

The chosen fish consumption values are presented in Table A5.3 and transformed to fish consumption in kg/person per month for use in calculations of human dioxin intake. The consumption rates highlighted in Table A5.1 were selected because they are likely to reflect long term consumption patterns which are of relevance for attainment of dioxin body burden.

The data for Tasmanian adults were used because they were higher than the Australian average. However no data were available for fish consumption by Tasmanian children consequently values for Australian children aged 2 to 3 years were selected.

Table A5.3: Fish consumption rates selected for exposure assessment

Population Group		Fish Consumption g/person per day [e]			Fish Consumption kg/person per month[a]		
		Avg	**95%**	**Max**	**Avg**	**95%**	**Max**
Adult (Tasmanian > 19 years)[b]		31	46	54	0.94	1.4	**1.6**
Child (Australian[c] 2-3 years old)	Male [d]	6.9	11	12	0.21	0.32	**0.36**
	Female [d]	6.5	10	12	0.20	0.30	**0.37**

[a] Months are assumed to have 30 days.
[b] Values are rounded and not separated by gender.
[c] Tasmanian values not available, therefore Australian values for 2 to 3 years old used.
[d] Relative standard error is between 25 and 50% and ABS (1999) suggest the values be used with caution.
[e] From Table A5.1.
[f] Bolded values are used in risk calculations.

A5.2 Determination of incremental dioxin fish concentration

A summary of the steps and equations used to determine incremental increases in fish due to the discharge of mill effluent is summarised in Text Box A5.1 and Table A5.4.

Only the predicted effluent dioxin concentration is available for determining an effluent related increase in fish dioxin levels. A notional water column dioxin concentration can be assumed within the small DZ100 by dividing the dioxin effluent concentration by the minimal dilution design requirements (100 fold) for the diffuser.

The US EPA (2004) considers that practically all dioxin in the water column will partition into sediment. For this risk assessment it is assumed the dioxin water concentration at the edge of the DZ100 will be constant and equilibrium will be established with sediment concentrations. To convert the assumed constant water column concentration into an equilibrium sediment concentration the general approach of the US EPA (2005) has been followed.

Fish may potentially acquire some of the dioxin in the sediment. The accumulation is dependent upon location specific characteristics of the sediment and the species of fish – these attributes are incorporated into location and species specific biota sediment accumulation factors (BSAF) that are empirically determined. From Tasmanian fishing information (Henry and Lyle 2003) and the Aquenal (2005) report it was considered flathead are the species most likely to be caught by recreational anglers near the outfall. The dioxin BSAF for this species was determined from Australian field studies reported in Gatehouse (2004) and verified by independent calculations using the parameter data supplied in Müeller (2004).

The BSAF was then used to calculate the dioxin concentration in fish according to an equation from the US EPA (2005) that relates the dioxin concentration in sediment with the lipid content of the specific fish species and organic carbon content of local sediment.

A summary of the parameters and the values adopted for the risk assessment is in Table A5.4. Additional discussion is provided in the text below.

Text Box A5.1: Equation summary for risk assessment of dioxins

In order to calculate the concentration of dioxins in fish ($\mathbf{C_F}$), from a given total water column concentration ($\mathbf{C_{wtot}}$) a number of calculations briefly outlined below are required:

1. Estimation of equilibrium sediment concentration (C_{sb}) from water column concentration (C_{wtot}). The approach adopted is from the US EPA (2005) for a water body whose dioxin concentration is at steady state with input sources. Sediment concentration is dependent on the equilibrium partition coefficient ($\mathbf{Kd_{bs}}$) of dioxin between sediment pore water and sediment particles. It is also dependent on the depth of the local water column ($\mathbf{d_{wc}}$) and site adjustment is made for local bed sediment porosity ($\mathbf{\Theta_{bs}}$) and bulk density ($\mathbf{C_{BS}}$).

$$C_{sb} = [(f_{bs} \times C_{wtot} \times Kd_{bs})/(\theta_{bs} + Kd_{bs} \times C_{BS})] \times [(d_{wc} + d_{bs})/d_{bs}] \quad \text{.....Equation A5.1}$$

2. Determination of the relationship between dioxin sediment concentration and the concentration in fish (i.e. the BSAF). This has been estimated using the same approach as Gatehouse (2004) and data from the Australian dioxins program in which concentrations of TCDD in sediment and fish from a number of locations around Australia were measured (Muëller 2004). The data is specific for the locations from which samples were obtained and in order to make it applicable to other sites adjustment is required for location specific dioxin sediment concentration ($\mathbf{C_{s\ LS}}$), sediment organic carbon ($\mathbf{OC_{sed\ LS}}$) content, location specific concentration in fish ($\mathbf{C_{F\ LS}}$) and the lipid content of the fish ($\mathbf{f_{lipid\ LS}}$) of interest (flathead). (The LS sub-script denotes 'location specific').

3. Calculation of the concentration in fish (C_F). The calculation method is from US EPA (2005). It relates the concentration in sediment (C_{sb}, from Equation A5.1) and the BSAF with the lipid fraction (f_{lipid}) in fish likely to be caught by anglers (flathead) at the outfall and the organic carbon in sediment at the location (OC_{sed}).

$$C_F = \frac{C_{sb} \times f_{lipid} \times BSAF}{OC_{sed}} \quad \text{..... Equation A5.3}$$

4. Calculation of the monthly intake ($\mathbf{M_{I\ FISH}}$, [pg/kg bw/month]). The amount of dioxin TEQ ingested is calculated (Section 6.3.5 of main text) assuming a person exclusively eats fish that spend all their time in the initial dilution zone of the proposed outfall site and are at steady state with sediment concentrations in that area. Ingestion of fish from other sources is not considered.

$$M_{I\ FISH} = \frac{C_F \text{ (pg/kg fish)} \times \text{consumption rate (kg fish/month)}}{\text{body weight (kg)}} \quad \text{..... Equation A5.4}$$

Table A5.4: Summary of equation parameters

Parameter Symbol	Value	Description / Reference
Parameters from equation A5.1		
C_{sb}	0.35 pg TEQ / kg	Sediment concentration. Estimated value from Equation 1.
f_{bs}	1 (unitless)	Fraction of total contaminant discharged to water body partitioning to bed sediment (unitless). According to US EPA (2004) practically all dioxins relatively rapidly distribute to sediment.
C_{wtot}	0.00074 pg TEQ/L	Total water concentration, assumed to be the TEQ estimate for discharged mill effluent with 100x dilution.
Kd_{bs}	292,000 (unitless)	Partition coefficient of TCDD between bed sediment particulates & bed sediment pore water. This is a compound specific value obtained from US EPA (2005).
Θ_{bs}	0.6 (unitless).	Bed sediment porosity at outfall. Default US EPA value of 0.6 adopted (US EPA 2005).
C_{BS}	1.0 g/cm^3	Bed sediment bulk density at outfall. US EPA (2005) expects this value to be reasonable in most cases.
d_{wc}	26 metres	Depth of water column (m). According to Aquenal(2005) and based on barthymetric survey the site of the proposed diffuser will be approx. 26 metres below surface. However it is assumed the vertical mixing zone may occur to the surface.
d_{bs}	0.03 metres	Depth of upper benthic sediment layer. A default 0.03m has been adopted based on the median of values cited by US EPA (2005).
Parameters from equation A5.2		
C_F	pg TEQ/kg fish	Concentration of contaminant in fish (pg TEQ/kg fish). To be calculated.
BSAF	0.09 (unitless)	Biota-sediment accumulation factor (BSAF)-the relationship between sediment dioxin concentration and fish tissue dioxin concentration determined from field trials. It is calculated as the ratio of the lipid-normalised concentration of a substance in tissue of an aquatic organism to its organic carbon-normalised concentration in surface sediment. Refer to Table A5.5.
f_{lipid}	0.02 (unitless)	Fraction of lipid in fish. The lipid content of Australian fish varied between 0.4% and 6.4% with a mean of approximately 2% (Müeller et al. 2004). The lipid content of sampled flathead was 0.82% to 3.4%. For the species present at the outfall site lipid content is 0.5-0.9% (Yearsley 1999). A value of 2% (0.02) has been adopted for the risk assessment.
OC_{sed}	0.0032 (unitless)	Fraction of organic carbon in bottom sediment. The organic carbon (OC) content was not measured at the proposed outfall site (Aquenal 2005). The mean OC fraction in a recent survey of dioxin levels in Australian marine sediments was 0.32% (range 0.048% to 1.4%) (Muëller 2004). The proposed site for the ocean outfall is described as 'pure sand' and therefore is expected to have a low fraction of OC (Aquenal 2005). In the absence of measured data, the mean value of 0.32% (0.0032) for Australian marine sediments was used. However in the sensitivity analysis an OC of 0.048% was also applied (Section 6.3.5.8).

Estimation sediment dioxin concentration

According to US EPA (2004) practically all dioxins present in the water column will be deposited to sediment (C_{sb}). Dioxins have very low water solubility and in effluent will be absorbed onto particulate matter. Dioxins absorbed onto particulates tend to rapidly redistribute in the environment by settling out to bottom sediments (Servos et al. 1992). In an enclosed lake which received single or multiple additions of TCDD sorbed onto bed sediment it was observed whole water [54] concentration declined rapidly with a first order half life of 1.7 – 1.9 days (Currie et al. 2000).

The US EPA (2005) have developed a procedure (Equation A5.1) for determining the sediment concentration in water bodies receiving dioxin from direct air borne deposition, surface water run off and erosion. The method relies on the partitioning of dioxin between the water column and sediment when the water column concentration is at equilibrium with the dioxin input sources and removal mechanisms (vaporisation and sediment burial). In the risk assessment herein the US EPA (2005) methodology has been applied assuming the zone of initial dilution is the receiving water body and the effluent discharge is the sole input source. A tacit assumption in applying the US EPA (2005) methodology is the dioxin concentration in water of the initial dilution zone is constant and at equilibrium with the sediment concentration. In this risk assessment this has been assumed to be the concentration at the boarder of the 1 in 100 initial dilution zone. Thus the sediment concentration has been calculated assuming a total constant concentration in the water column of 0.00074 pg TEQ/L (i.e. the TEQ concentration in discharged effluent but diluted 100 fold). The impact of dioxin concentrations137 times higher is assessed in Section 6.3.5.8.

Sediment concentration is dependent on the equilibrium partition coefficient (Kd_{bs}) of dioxin between sediment pore water and sediment particles. It is also dependent on the depth of the local water column (d_{wc}) and site adjustment is made based on local bed sediment porosity (Θ_{bs}) and bulk density (C_{BS}).

[54] Whole water concentration includes TCDD sorbed to particulates, plus that associated with dissolved organic carbon and the fraction freely dissolved in water.

$$C_{sb} = [(f_{bs} \times C_{wtot} \times Kd_{bs})/(\Theta_{bs} + Kd_{bs} \times C_{BS})] \times [(d_{wc} + d_{bs})/d_{bs}]$$

……Equation A5.1

Where

f_{bs} = Fraction of total contaminant discharged to water body that is sorbed to bed sediment (unitless). According to US EPA (2004) practically all dioxins relatively rapidly distribute to sediment. Therefore the fraction sorbed to sediment is assumed to be 1.

$C_{w\,tot}$ = Total water concentration (pg TEQ/L), assumed to be the TEQ estimate for Bell Bay effluent with 100x dilution, i.e. 0.00074 pg TEQ/L.

Kd_{bs} = Partition coefficient of TCDD between bed sediment particulates and bed sediment pore water. This is a compound specific value and according to the US EPA (2005) the value for TCDD is 2.92 E+05.

Θ_{bs} = Bed sediment porosity (unitless). A default value of 0.6 is adopted (US EPA 2005).

C_{BS} = Bed sediment bulk density (g/cm^3). Default value of 1.0. The US EPA (2005) expects this value to be reasonable in most cases.

d_{wc} = Depth of water column (m). According to Aquenal the depth of the proposed ocean outfall is approximately 26 metres and vertical mixing is anticipated for this depth.

d_{bs} = Depth of upper benthic sediment layer a default 0.03m has been adopted based on the median of values cited by US EPA (2005).

Based on Equation A5.1 and the above assumptions the bed sediment concentration is calculated to be 0.35 pg/kg, viz:

$$C_{sb} = [f_{bs} \times C_{wtot} \times Kd_{bs}/(\Theta_{bs} + Kd_{bs} \times C_{BS})] \times [(d_{wc} + d_{bs})/d_{bs}]$$
$$= [1 \times 0.00074 \times 292{,}000/(0.6 + 292{,}000 \times 1)] \times [(26 + 0.03)/0.03]$$
$$= 0.64 \text{ pg/kg}$$

Selection of a biota-sediment accumulation factor

Dioxins sorbed to suspended and dissolved organic matter can be ingested and accumulated by sediment-dwelling organisms and general concern is they may be transferred through the food chain to fish at higher trophic levels. The fish's food is the most important exposure source for uptake of dioxins; direct uptake from water via gills and skin is negligible due to very low dioxin water solubility (Gatehouse 2004). According to Gatehouse (2004) benthic (bottom dwellers or demersal) feeding fish consistently contain more dioxins than other fish. The relationship between sediment concentration and fish tissue concentration is therefore more important than the relationship between water column concentration and fish tissue concentration (i.e. the bioaccumulation factor BAF).

The relationship between sediment dioxin concentration and fish tissue dioxin concentration is determined from field trials and is called the biota-sediment accumulation factor (BSAF).

The nomenclature “biota-sediment accumulation factor” implies accumulation may be primarily, if not exclusively, from sediment however because BSAFs are based on field data the values also incorporate the effects of chemical bioavailability from all segments of the local environment, the food web structure, metabolism, biomagnification, fish growth plus other factors (Hendricks et al. 1998, Burkhard and Lukasewyez 2000, US EPA 2004, US EPA 1995, Cook and Burkhard 1998). Because the BSAF approach is empirically determined it is particularly useful for chemicals which are detectable in fish tissues and sediments but are difficult to detect or measure precisely in the water column. For this reason US EPA (2004) consider the BSAF for TCDD to be a more reliable measure of bioaccumulation potential than bioaccumulation factors (BAFs) and/or bioconcentration factors (BCFs).

BSAFs are specific for the locations from which sediment and fish samples were obtained. In order to make the BSAF applicable to other locations the BSAF needs to be made location specific for dioxin sediment concentration ($C_{s\ LS}$), sediment organic carbon ($OC_{sed\ LS}$) content, and location specific dioxin concentration in fish ($C_{F\ LS}$) and the lipid content of the fish ($f_{lipid\ LS}$)[55]. Thus BSAFs are usually reported as values normalised for these parameters.

As a component of the Australian National Dioxins Program, PCDD/PCDFs were measured in sediment and biota from a number of sites around Australia and BSAFs calculated for bivalves and various Australian species of fish (Gatehouse 2004, Müeller 2004). Commercial fishermen supplied fish samples that were caught in close proximity to the sediment sampling locations. However the number of sediment samples available for each area was small, ranging between one to three (Müeller 2004). The concentration ratios for fish were calculated by dividing the fish tissue TCDD concentrations by the average surface sediment TCDD concentration in the catching vicinity. Gatehouse normalised the BASF according to the lipid content of the fish and the organic carbon content of sediment from the locality from which the fish was caught using the data provided in Müeller (2004). Unfortunately the normalised BSAFs are not reported as tabulated numerical values but are presented graphically (Gatehouse, figure 3.6). Because it is difficult to accurately

[55] The sub-script notation of ‘LS’ denotes ‘Location Specific’.

determine the numerical values from the presentation in Gatehouse (2004) Toxikos has used the equation of Gatehouse (2004) and the same data source (i.e. Müeller 2004) to calculate the normalised BSAFs presented by Gatehouse (2004).

Table A5.5 summarises the Toxikos calculated BSAF values for TCDD in fish, these correspond to the values provided in Figure 3.6 of Gatehouse (2004). It is evident that they are all below 1 and hence signify no biomagnification by fish. Also included in Table A5.5 are BSAF values from overseas literature and the recommended US EPA (2005) normalised BSAF for TCDD in fish. These have been included to support the fact that dioxin BSAF values are generally less than 1 and also as a check for the calculations undertaken. It should also be noted that the data of Gatehouse (2004) includes BSAFs for molluscs, these are also less than unity and signify dioxins are not biomagnified by these organisms.

The most commonly caught species of fish by Tasmanian anglers is the flathead (a bottom dweller) and the BSAF chosen from Table A5.5 for calculating dioxin fish concentrations in Equation A5.3 was the highest of those available for this species. It is noted the flathead data obtained in the Australian National Dioxins Program were all caught in an estuary, consequently they will tend to have higher lipid content than their marine counterparts and therefore a higher BSAF. The chosen BSAF may therefore tend to over estimate the transfer of dioxin from sediment to fish.

The BSAF chosen for the present assessment is 0.046, this is the highest value calculated for flathead, the species most likely to be caught at the outfall site.

Table A5.5: Biota-sediment accumulation factors for 2,3,7,8-TCDD

Species	$C_{F\ LS}$ (pgTEQ/ kg fish)	$f_{lipid\ LS}$	$C_{F\ lipid}$ (pgTEQ/ kg fish)	Location	$C_{S\ LS}$ (pgTEQ/ kg sed)	F_{OC}	$C_{S\ OC}$ pg/kg sed	BSAF[b]
Port Jackson Bream Acanthopagrus *sp.*	0.078	0.046	1.69	Sydney Port Jackson West (ES2A) Estuarine	39	0.019	2052	0.0008
Port Jackson Flathead *Platycephalus sp.*	<0.02[a]	0.013	0.77	Sydney Port Jackson West (ES2A) Estuarine	39	0.019	2052	0.0004
Melbourne Region Green Backed Flounder *Rhombosolea tapirina*	<0.005[a]	0.019	0.13	Melbourne Lower Yarra River (ES1B)	<0.2[a]	0.0046	22	0.006
Port Jackson King George Whiting *Sillaginoides punctata*	0.011	0.012	0.92	Sydney Port Jackson West (ES2B) Estuarine	45	0.053	849	0.001
Melbourne Region Flathead *Platycephalus sp.*	0.0072	0.009	0.79	Melbourne Lower Yarra River (ES1A) Estuarine	0.7	0.041	17	**0.046** [b]
Melbourne Region King George Whiting *Sillaginoides punctata*	0.02	0.017	1.18	Melbourne Lower Yarra River (ES1B) Estuarine	<0.2[a]	0.0046	22	0.054
Gippsland Lakes short finned eel *Anguilla australis*	0.015	0.064	0.23	Gippsland Lakes (ESA1) Estuarine	<0.08[a]	0.015	2.67	0.09
Derwent River Australian Salmon Arripis *sp.*	<0.02[a]	0.062	0.16	Hobart Lower Derwent R (ES1A) Estuarine	<0.04[a]	0.028	0.71	0.2
US EPA (2005) recommended value for Human Health Risk Assessment	Presented as a homologue group value for TetraPCDD and PCDFs. Values were obtained from US EPA (2000) and were measured, or estimated using a whole fish lipid content of 7% & organic content of 3%.							0.09
Lake Ontario	Variety of fish species including; lake trout, brown trout, yellow perch, white perch, smallmouth bass, smelt, and slimy sculpin (cited in Gatehouse 2004). Note that the fish lipid contents of lakes are typically higher (approx 7%) than those of marine species (1%).							Range 0.03-0.12

Shaded rows represent the species chosen to represent those at the outfall and the bold value the BSAF for use in the risk assessment.

[a] Calculated at half detection limit by Müeller et al. (2004).

[b] The data in the Table is from Müeller (2004) and the BSAF values calculated using the same equation as for Australian National Dioxins Program (Gatehouse 2004), viz:

$$BSAF = C_{F\ lipid} \div C_{S\ OC} \quad \text{.....Equation A5.2}$$

$C_{F\ lipid} = C_{F\ LS} \div f_{lipid\ LS}$ The fish concentration normalised for lipid ($C_{F\ lipid}$) is calculated by dividing the total concentration in locally caught fish tissue ($C_{F\ LS}$) by the lipid tissue fraction ($f_{lipid\ LS}$).

$C_{S\ OC} = C_{S\ LS} \div f_{SOC\ LS}$ The sediment concentration normalised for organic carbon ($C_{S\ OC}$) is calculated by dividing the total dry weight dioxin concentration in sediment ($C_{S\ LS}$) by the dry weight sediment organic carbon fraction ($f_{SOC\ LS}$).

Calculation of dioxin concentration in fish

The calculation method of the US EPA (2005) has been used to estimate the concentration of dioxin in fish tissue from a sediment concentration (Equation A5.2). The equation [56] utilises the bed sediment concentration from Equation A5.1 and the 'normalised' BSAF from Table A5.5, site specificity is incorporated into the calculation by considering the lipid fraction of fish likely to be caught at the effluent outfall (Appendix 8) and an assumption regarding the organic carbon content of the sediment at the outfall.

$$CF = \frac{BSAF \times C_{sb} \times f_{lipid}}{OC_{sed}} \quad \text{.....Equation A5.3}$$

Where:

C_F = Concentration of contaminant in fish (pg contaminant/kg fish)

C_{sb} = Concentration of contaminant sorbed to bottom (bed) sediment (pg contaminant/kg bed sediment). Value calculated to be 0.69pg/kg from Equation A5.1.

f_{lipid} = Fraction of fish lipid (unitless). Since the accumulation of dioxin occurs in lipid, a correction factor to estimate the overall tissue concentration is needed since fish consumption refers to fish flesh and not just the lipid. Australian National Dioxins Program (Müeller et al. 2004) included measurement of the lipid fraction of fish collected for dioxin analysis. The lipid content of all fish caught varied between 0.4% and 6.4% with a mean of approximately 2%. In this program the lipid content of sampled flathead was 0.82% to 3.4% (mean 1.5%, 23 locations). Note not all the lipid data from Müeller et al. (2004) is included in Table A5.5. Table A5.6 includes the average lipid content of fish observed by Aquenal (2005) to be present at the outfall. According toYearsley (1999) the lipid content of these species is between 0.5-0.9%. For this risk assessment a fish lipid concentration of 2% (0.02) has been assumed which, from the information above will overestimate fish dioxin concentrations.

BSAF = Biota-to-sediment accumulation factor (unitless). The value chosen for the BSAF is 0.046 (Table A5.5).

OC_{sed} = Fraction of organic carbon in bottom sediment (unitless). The organic carbon content was not measured as part of the outfall site survey conducted Aquenal (2005). However data is provided in the Australian National Dioxins Program (Muëller 2004). The mean total organic carbon fraction from all sampled sites was 0.32% (range 0.048% to 1.4%). The proposed site for the ocean outfall is expected to have a low fraction of organic carbon as it is described as a 'pure sand' (Aquenal 2005). In the absence of measured data, the mean value for Australian marine sediments of 0.0032 has been used for the present assessment.

---

[56] The equation of the US EPA (2005) can be derived from Equation A5.2 (Footnote to Table A5.5) viz:

$BSAF = C_{F\ lipid} \div C_{S\ OC}$

where $C_{F\ lipid} = C_{F\ LS} \div f_{lipid\ LS}$ and $C_{S\ OC} = C_{S\ LS} \div f_{SOC\ LS}$, substituting in the equation yields,

$BSAF = (C_{F\ LS} \div f_{lipid\ LS}) / (C_{S\ LS} \div f_{SOC\ LS})$, solving for the fish dioxin concentration gives,

$C_{F\ LS} = BSAF \times f_{lipid\ LS} \times C_{S\ LS} / f_{SOC\ LS}$ which is the same as Equation A5.2

where parameter symbols for local sediment dioxin concentration ($C_{S\ LS}$) = the bed sediment concentration (C_{sb}) and symbol for the fraction of local sediment as organic carbon ($f_{SOC\ LS}$) = organic carbon in sediment (OC_{sed}).

Substituting these parameter symbols yields,

$CF = BSAF \times C_{sb} \times f_{lipid} / OC_{sed}$,i.e. Equation A5.3

Substituting values into Equation A5.3, the concentration in fish was calculated to be 0.18 pg/kg.

$$C_F = \frac{C_{sb} \times f_{lipid} \times BSAF}{OC_{sed}}$$

$$= \frac{0.64 \text{ pg/kg} \times 0.02 \times 0.046}{0.0032}$$

$$= 0.18 \text{ pg/kg}$$

Fish species at effluent outfall location

Aquenal (2005) conducted a field survey of the marine environment at the proposed Gunns Ltd pulp mill outfall site between the 4th and 22nd April 2005. A range of investigation techniques were used but of particular relevance to fish were baited fish video surveys and a literature review. According to Aquenal (2005) the most commonly recorded species during the baited video surveys was degen's leatherjacket, followed by barber perch. Some velvet leatherjacket, sand flathead, and red cod were also recorded as solitary individuals. However during collection of fish using handlines the most commonly caught species was sand flathead, followed by common gurnard perch, with smaller number of rosy wrasse and blue-throated wrasse.

Collection of shellfish from the outfall site was unsuccessfuly attempted on several occasions during the Aqenal survey. It is therefore concluded there are insufficient shellfish occurring naturally around in the area of the outfall to allow their effective collection by diving, particularly considering the depth of the survey area (Aquenal 2005, p17).

The Aqenal report (2005) describes fish densities as generally low during baited video surveys. Out of 50 surveys performed, fish were recorded in 37 cases while no fish were detected in the remaining 13 surveys, the latter including surveys performed at the immediate diffuser site. The ocean bottom at the diffuser site is dominated by well sorted coarse sands which is indicative of frequent high levels of water movement. Currents in the open sandy habitat combined with lack of shelter from predators are consistent with the paucity of flora and fauna observed in the Aquenal survey (GHD 2005).

Table A5.6: Description of species within 1 km of ocean outfall (see Figure 1)

Principal Species/ Group	Habitat	Main Diet	Adult Weight (kg)	Adult Length (cm)	Average oil content (%)
Sand Flathead *Platycephalus Arenarius*[a]	Demersal. Bottom-dwelling on soft substrates of the inner continental shelf of tropical and warm temperate Australia in depths of 5-60 m[a]	small fish, crabs, prawns and other small crustaceans, octopus, squid and worms[b]	0.3-0.6[a]	30-42[a]	0.8 [a]
Leatherjacket *Monacathidae*[a] (Degen's lj, velvet lj)	Dermersal and pelagic on the continental shelf and upper slope, often near reefs and sponge beds, to depths of 200 m [a]	Small invertebrates[c] Omnivorous: primarily gastropods, sponges, tunicates, heart urchins and algae[d]	0.8[a]	38[a]	0.5 (for the *Nelusetta ayraudi species*)[a]
Barber Perch -*Caesioperca rasor* Red Guarnand Perch -*Helicolenus percoides*[a]	Sheltered and moderately exposed reef (2-100m depth)[e] Demersal on the shelf and slope off southern Australia in 10-800m depth [a]	Small invertebrates[e]	0.8[a]	< 26[e] < 35[a]	0.5[a]
Wrasse blue-throated -*Notolabrus tetricus* rosy -*Pseudolabrus psittaculus*	Demersal in kelps, seagrasses and over coral and rocky reefs on the continental shelf to at least 200m [a] Found at water depths of 2 -220m [e]	Small invertebrates.[f]	0.3-2.0[a]	25-50[a] Maximum size: 20 cm[e]	0.5 (for parrotfish, *Scarus ghobban*)[a]
Red Cod (Preudophycis bachus)	Coastal and inner continental shelf to depths of 160 m. Most abundant in less than 60 m depth[a]	Crustaceans , cephalopods & fish[g]	0.8-1.5[a]	40-50[a]	

Principal Species/ Group	Habitat	Main Diet	Adult Weight (kg)	Adult Length (cm)	Average oil content (%)
School shark -*Galeorhinus galeus*[a]	Demersal and midwater over the continental shelf and upper slope from inshore to at least 600 m and probably deeper while young sharks use bays and estuaries as nursery areas [a]	Fish make up most of the diet with some crustaceans and celphalopods[h]	6-12[a]	100-130[a]	0.9[a]
Gummy shark *Mustelus antarcticus*[i]	Demersal on the continental shelf and upper slope from shallow estuaries to depths of about 400m [a]	Cephalopods (squid and octopus) make up 36% of the diet, crustaceans 25% and bony fish 11%. The remaining 28% consists of 12 other classes of organism and unidentifiable material [i]	5[a]	100-120[a]	0.9[a]

[a] Yearsley (1999), [b] Sea-Ex Australia (2005), [c] Marine Discovery Research Centre (2001), [d] Hawai'i Coral Reef Network (2001), [e] Marine Discovery Research Centre (1997), [f] Aquarium Reference (2005), [g] Edgar and Shaw (1995), [h] Morato et al. (2003), [i] The Bureau of Rural Sciences and the Fisheries Research and Development Corporation (1993) in DAFF (2005).

References for Appendix 5.

ABS (1999). National Nutrition Survey: Foods Eaten. Australia 1995. Report No: 4804. Prepared by McLennan, W. and Podger, A. for the Australian Bureau of Statistics (ABS).

Aquarium Reference (2005). *Pseudolabrus psittaculus* Species Summary (www.aquariumreference.org).

Aquenal (2005). Aquenal (2005). Gunns Ltd Pulp Mill. Marine biological and pollutant survey at the proposed outfall site.

Burkhard, L.P. and Lukasewycz, M.T. (2000). Some bioaccumulation factors and biota-sediment Accumulation factors for polycyclic aromatic hydrocarbons in lake trout. Environ. Toxicol. Chem. 19:1427-1429.

Cook, P., M. and Burkhard, L., P. (1998). Development of bioaccumulation factors for protection of fish and wildlife in the Great Lakes. In: *National Sediment Bioaccumulation Conference Proceedings*. U.S. Environmental Protection Agency, Office of Water. Washington, DC. EPA 823-R-002 (cited in USEPA 2000a).

Currie, R.S., Fairchild, W.L., Holoka, M.I.H., and Muir, C.G. (2000). Long-term fate and bioavailability of 2,3,7,8-tetrachlorodibenzofuran in littoral enclosures. Environmental Toxicology and Chemistry. 19(6): 1491-1500.

DAFF (2005). The gummy shark. Department of Agriculture Forestry and Fisheries. The Bureau of Rural Sciences and the Fisheries Research and Development Corporation (1993). Reproduced from The Australian Fisheries Resources (www.edaff.gov.au).

Edgar, G.J. and Shaw, C. (1995). The production and trophic ecology of shallow-water fish assemblages in southern Australia. II. Diets of fishes and trophic relationships between fishes and benthos at Western Port, Victoria. Journal of Experimental Marine Biology and Ecology 194: 83-106.

enHealth (2003). The Australian Exposure Assessment Handbook. Consultation Draft. Prepared by Langley, A., Sabrodo, L., Taylor, A., Dal Grande, E. and Mangas, S. Commonwealth of Australia. http://www.health.gov.au/internet/wcms/publishing.nsf/content/health-pubhlth-strateg-envhlth-exposure.htm/$FILE/exposure.pdf

Gatehouse, R. (2004). Ecological risk assessment of dioxins in Australia –technical report no. 11. Government, Department of the Environment and Heritage May 2004 (http://www.deh.gov.au/settlements/publications/chemicals/dioxins/report-11/index.html).

GHD (2005). Projects GHD Subject Draft Report Volume 3 –Water Supply Pipeline from Trevallyn Dam. Effluent Pipeline from the Pulp Mill to Five Mile Bluff and Ocean Outfall Construction Camp. Gunns Limited. October 2005.

Hawai'i Coral Reef Network. (2001). Family Monacanthidae (www.coralreefnetwork.com).

Hendriks, A.J., Pieters, H., de Boer, J. (1998). Accumulation of metals, polycyclic (halogenated) hydrocarbons, and biocides in zebra mussel and eel from the Rhine and Meuse rivers. Environ. Toxicol. Chem 17:1885-1898.

Henry, G.W. and Lyle, J.M. (2003). The national recreational and indigenous fishing survey. Australian Government Department of Agriculture, Fisheries and Forestry. Commonwealth of Australia.

Javitz, H. (1980). Seafood consumption data analysis. SRI International. Final report prepared for EPA Office of Water Regulations and Standards. EPA Contract 68-01-3887. (As cited in US EPA 1997).

Müeller, J., Muller, R., Goudkamp, K., Shaw, M., Mortimer, M., Haynes, D., Paxman, C., Hyne, R., McTaggart, A., Burniston, D., Symons, R. & Moore, M. (2004). Dioxins in aquatic environments in Australia –technical report no.6. Australian Government, Department of the Environment and Heritage (DEH) May 2004 (http://www.deh.gov.au/settlements/publications/chemicals/dioxins/report-6/index.html).

Marine Discovery Research Centre (1997). Barber Perch –Family Serranidae (www.woodbridge.tased.edu.au).

Marine Discovery Research Centre (2001). Leatherjackets –Family Monacanthidae (www.woodbridge.tased.edu.au).

Morato, T., Encarnacion, S., Gros, M.P. and Menezes, G. (2003). Diets of thornback ray (*Raja clavate*) and tope shark (*Galeorhinus galeus*) in the bottom longline fishery of the Azores, northeastern Atlantic. Fisheries Bulletin 101:590-602.

National Marine Fisheries Service (NMFS) (1993). Data tapes for the 1993 NMFS provided to U.S. EPA, National Center for Environmental Assessments. (As cited in US EPA 1997).

Pao, E.M., Fleming, K.H., Guenther, P.M., and Mickle, S.J. (1982). Foods commonly eaten by individuals: amount per day and per eating occasion. U.S. Department of Agriculture. Home Economics Report No. 44. (As cited in US EPA 1997).

Sea-Ex Australia (2005). Flathead. Australian Fish and Seafood Photographs and Information. (www.sea-ex.com/fishphotos/flathead.htm).

Servos, M.R., Muir, D.C.G., and Webster, G.R.B. (1992). Environmental fate of polychlorinated dibenzo-*p*-dioxins in lake enclosures. Canadian Journal of Fisheries and Aquatic Science. 49: 722-734. As cited in Currie et al (2000).

US EPA (1995). Final Water Quality Guidance for the Great Lakes System. Final Water Quality Guidance for the Great Lakes System Final Rule. Federal Register: March 23, 1995, Page 15365-15366. 40 CFR 9, 122, 123, 131, and 132.

US EPA (1997). Exposure Factors Handbook. U.S. Environmental Protection Agency. http://www.epa.gov/ncea/pdfs/efh/front.pdf

US EPA (2000a). Methodology for deriving ambient water quality criteria for the protection of human health (2000). Office of Water. U.S. Environmental Protection Agency. Report No: EPA-822-B-00-004.

US EPA (2000b). Exposure and human health re-assessment for 2,3,7,8 – tetrachlorodibenzo-p-dioxin (TCDD) and related compounds. Draft final report. Office of Research and Development. U.S. Environmental Protection Agency. Washington, D.C. Report Number: EPA 600/P-00/001 Bc. (As cited in US EPA, 2005).

US EPA (2004). Exposure and Health Assessment for 2,3,7,8 – Tetrachlorodibenzo-p-dioxin (TCDD) and Related Compounds. National Centre for Environmental Assessment Office of Research and Development, U.S. Environmental Protection Agency. National Academy of Sciences Review Draft.

US EPA (2005). Human health risk assessment protocol for hazardous waste combustion facilities. Volume 2 Appendix B and also HHRAP companion database. U.S. Environmental Protection Authority. Washington, D.C. (http://www.epa.gov/epaoswer/hazwaste/combust/riskvol.htm#volume2).

West, P.C., Fly, M.J., Marans, R., and Larkin, F. (1989). Michigan sport anglers fish consumption survey. A report to the Michigan Toxic Substance Control Commission. Michigan Department of Management and Budget Contract No. 8720141. (As cited in USEPA 1997).

Yearsley, G.K., Last, P.R. and Ward, R.D. (1999). Australian Seafood Handbook. An identification guide to domestic species. CSIRO Marine Research, Australia.

Appendix 6: Comparison with guidelines for recreational waters.

A6.1 Screening against recreational waters guideline values

The estimated concentration of each chemical of interest at the edge of the initial dilution zone was assessed against guideline values for primary recreational activities (swimming or diving). It is unlikely that direct water conract recreational activities will occur within the initial dilution zone. None of the chemicals of interest in Table A1.3 following initial dilution exceed any their respective recreational waters guideline value. Table A6.1 details the comparison for each chemical of interest and their respective guideline values.

Table A6.1: *'Chemicals of interest'* screened against recreational guidelines.

Chemical of interest	Effluent concentration after dilution (µg/L)	ANZECC [a] recreational guideline (µg/L)	WHO [b]/ Europa [c] recreational guidelines (µg/L)
Metals			
Aluminium	6	200	
Antimony	0.014		200[b]
Arsenic (III &V)	0.029	50	100[b]
Barium	0.06	1,000	7,000[b]
Beryllium	0.015		
Boron	0.23	1,000	5,000[b]
Cadmium	0.012	5	30[b]
Chromium (III & VI)	0.20	50	500[b]
Cobalt	0.03		
Copper	0.20	1,000	20,000[b]
Iron	8.17	300	
Lead	0.03	50	100[b]
Manganese	8.17	100	4,000[b]
Mercury	0.003	1	10[b]
Molybdenum	0.014		700[b]
Nickel	0.28	100	200[b]
Selenium	0.075	10	100[b]
Silicon	0.570		
Tin	0.138		
Vanadium	0.014		
Zinc	0.847	5,000	
Non metallic inorganics			

Chemical of interest	Effluent concentration after dilution (µg/L)	ANZECC [a] recreational guideline (µg/L)	WHO [b]/ Europa [c] recreational guidelines (µg/L)
Ammonia	< 4.55	10	
Hydrogen sulphide			
Nitrate	< 182	10,000	500,000[b]
Thiolignin			
Thiosulphate			
Organic acids	45		
Chloroacetic acids	41		
Monochloroacetic acid	13.5		
Dichloroacetic acid (includes methyl ester)	13.5		
Trichloroacetic acid	13.5		
Resin acids	**2.50**		
14-chlorodehydroabeitic acid	0.50		
12,14-dichlorohydroabietic acid	0.50		
Abietine	0.50		
Arakine	0.50		
Chlororetene	0.50		
dehydroabietic acid	0.50		
Fichtelite	0.50		
Isopirame	0.50		
Levopirame	0.50		
Neoabietine	0.50		
Palustrine	0.50		
pimaric acid	0.50		
Retene	0.50		
Fatty acids	**1.90**		
(2E,4Z)-Hexadienedioic acid monomethyl ester	0.40		
Behene	0.40		
Lignoserine	0.40		
Linoleine	0.40		
Myristine	0.40		
Oleine	0.40		
Palmitine	0.40		
Stearine	0.40		
Linoleic acid	0.40		
Chlorinated natural phenolics	**0.24 (total)**	2 (total phenolics)	≤5 (total phenolics)[c]
4-chlorocatechol	0.05		
3,4-dichlorocatechol	0.05		
3,5-dichlorocatechol	0.05		
2-chloro-p-cymene	0.05		

Chemical of interest	Effluent concentration after dilution (µg/L)	ANZECC [a] recreational guideline (µg/L)	WHO [b]/ Europa [c] recreational guidelines (µg/L)
5-chloro-o-cymene	0.05		
2,3-dichloro-p-cymene	0.05		
2,5-dichloro-p-cymene	0.05		
2,6-dichloro-m-cymene	0.05		
4-chloroguaiacol	0.05		
3,4-dichloroguaiacol	0.05		
4,5-dichloroguaiacol	0.05		
4,6-dichloroguaiacol	0.05		
2-chloroacetoguaiacones	0.05		
5-chloroacetoguaiacones	0.05		
6-chloroacetoguaiacones	0.05		
2-Monochlorosyringaldehyde	0.05		
2,6-dichlorosyringaldehyde	0.05		
2-Chloro-3,4,5-trimetoxybenzaldehyde	0.05		
2,6-Dichloro-3,4,5-trimetoxybenzaldehyde	0.05		
2-chlorovanillin	0.05		
5-chlorovanillin	0.05		
6-chlorovanillin	0.05		
2,5-dichlorovanillin	0.05		
2,6-dichlorovanillin	0.05		
5,6-dichlorovanillin	0.05		
Dichloroveratrole	0.05		
Chlorophenols	**0.01 (total)**	2 (total phenolics)	≤5 (total phenolics)[c]
2-Monochlorophenol	0.002		
3-Monochlorophenol	0.002		
4-Monochlorophenol	0.002		
2,3-Dichlorophenol	0.002		
2,4-Dichlorophenol	0.002		
2,5-Dichlorophenol	0.002		
2,6-Dichlorophenol	0.002		
3,4-dichlorophenol	0.002		
3,5-dichlorophenol	0.002		
4-Chloro-3-methylphenol	0.002		
Other aromatic compounds	**4.75 (total)**		
Sterols	**4.28**		
beta-sitostanol	0.90		
beta-sitosterol	0.90		
Fucosterol	0.90		
Betulinol	0.90		

Chemical of interest	Effluent concentration after dilution (µg/L)	ANZECC [a] recreational guideline (µg/L)	WHO [b]/ Europa [c] recreational guidelines (µg/L)
Campesterol	0.90		
Stigmasterol	0.90		
Squalene (steroid hydrocarbon)	0.90		
Miscellaneous aromatics	**0.48 10% of total**		
2,4-Dimethylphenol	0.10	**2 (total phenolics)**	**≤5 (total phenolics)[c]**
2-Methylphenol	0.10	**2 (total phenolics)**	**≤5 (total phenolics)[c]**
2-Nitrophenol	0.10	**2 (total phenolics)**	**≤5 (total phenolics)[c]**
3-Methylphenol	0.10	**2 (total phenolics)**	**≤5 (total phenolics)[c]**
4-Methylphenol	0.10	**2 (total phenolics)**	**≤5 (total phenolics)[c]**
Phenol	0.10	**2 (total phenolics)**	**≤5 (total phenolics)[c]**
Dichloroacetovanillone	0.10		
a-pinene	0.10		
Benzene	0.10	10	100[b]
b-pinene	0.10		
Camphene	0.10		
Dehydrojuvabione	0.10		
Juvabione	0.10		
Dichloromethylenefuranones	0.10		
chlorodimethylnaphtalenes	0.10		
Alkylchlorophenanthrenes	0.10		
4-chloro-3-hydroxy-2H-pyran-2-one	0.10		
5,5-dichloro-6,6-dihydroxy-2-methoxy-2-cyclohexene-1,4-dione	0.10		
Dichloroprotocatechualdehyde	0.10		
3-methoxy-5-dichloromethylene -2(H5)furanone	0.10		
Aniline	0.10		
3-methoxycatechol	0.10		
p-cymene	0.10		
p-hydroxybenzaldehyde	0.10		
Syringol	0.10		
Dioxins (PCDD/PCDFs not PCBs or PCBB/PCBFs) I-TEQ	0.00074 pg/l (total)		

Chemical of interest	Effluent concentration after dilution (µg/L)	ANZECC [a] recreational guideline (µg/L)	WHO [b]/ Europa [c] recreational guidelines (µg/L)
Miscellaneous	**0.60 (total)**		
Hydrocarbons			
Methanol	0.12		
Ethanol	0.12		
3-carene	0.12		
Carbon disulphide	0.12		
Hexane (C6)	0.12		
Pentadecane (C15)	0.12		
Octadecane (C18)	0.12		
Nonadecane (C19)	0.12		
cosane (C20)	0.12		
heneicosane (C21)	0.12		
eicosane (C22)	0.12		
tricosane (C23)	0.12		
tetracosane (C24)	0.12		
pentacosane (C25)	0.12		
Limonene	0.12		
Aldehydes and ketones			
Acetaldehyde	0.12		
Pentanal	0.12		
Hexanal	0.12		
Heptanal	0.12		
Octanal	0.12		
Nonanal	0.12		
Acetone	0.12		
1,1-dichloroacetone	0.12		
2-butanone	0.12		
2-pentanone	0.12		
2-hexanone	0.12		
2-heptanone	0.12		
2-octanone	0.12		
2-nonanone	0.12		
Hydroquinone	0.12		
4-methyl-2-pentanone	0.12		
Dichlorocyclohexendione	0.12		
Chlorinated hydrocarbons			
Chloromethane	0.12		
Dichloromethane	0.12		200[b]
1,1-Dichloropropane	0.12		400 (1,2-dichloropropane)[b]
1,3-Dichloropropane	0.12		400 (1,2-dichloropropane)[b]
Chloroethylene	0.12		3[b]
1,1-Dichloroethylene	0.12	0.3	300[b]

Chemical of interest	Effluent concentration after dilution (µg/L)	ANZECC [a] recreational guideline (µg/L)	WHO [b]/ Europa [c] recreational guidelines (µg/L)
1,2-Dichloroethylene (cis & trans)	0.12		500[b]
1,3-dichloropropene (cis & trans)	0.12		200[b]
3-chloropropene	0.12		
1,1-dichlorodimethylsulfone	0.12		
Chlorohydroxypyrone	0.12		

[a] ANZECC (2000)

[b.] WHO recreational guidelines were calculated by multiplying the drinking water guideline (WHO 2004) by 10 according to the information provided in the WHO (2003).

[c] Europa (1976).

A6.1 References

ANZECC (2000). Australian and New Zealand guidelines for fresh and marine water quality, 2000. Australian and New Zealand Environment and Conservation Council. Environment Australia.

EUROPA (1976). Bathing water quality directive 76/160/EEC. Council Directive of 8 December 1975. Europa Environment (http://www.europa.eu.int/water/water-bathing/directiv.html).

WHO (2003). Guidelines for safe recreational water environments. Volume 1: Coastal and fresh waters. World Health Organization. http://www.who.int/water_sanitation_health/bathing/srwg1.pdf

WHO (2004). Guidelines for drinking-water quality. 3rd edition. Volume 1: Recommendations. World Health Organization. http://www.who.int/water_sanitation_health/dwq/GDWQ2004web.pdf

Appendix 7: Pulp mill effluent and fish tainting

Screening against tainting of seafood guideline values

Guidelines for chemicals that cause tainting are available from ANZECC (2000), US EPA (2005) and various provinces of Canada, e.g. British Columbia (BC MoE 1998). Guidelines could not be located from other regulatory agencies (Environment Canada, Europa environment, WHO, UK Department of Environment, Finnish Environment Industry and Danish Environmental Protection Agency) [57]. The estimated concentration of each chemical of interest at the periphery of the initial dilution zone was assessed against guideline values for tainting of seafood. It is unlikely that commercial seafood will be caught in this area however recreational fishing may occur. As can be seen in Table A7.1, none of the chemicals of interest following initial dilution (i.e. within approximately 100m of the outfall diffuser) exceed any of their respective tainting guidelines. However there are very few guidelines available for the chemicals of interest.

[57] OSPAR provided a list of chemicals that are suspected to taint and a list of chemicals that have been tested and found not to cause tainting. Compounds listed as not tainting are dimethylbenzenes, nitrophenols, dinitrophenols, β-pinene, phenol, toluene and xylene (OSPAR, 2002). Some of these compounds have guidelines according to ANZECC (2000) and therefore are expected to taint. Consequently this list was rejected.

Table A7.1: *'Chemicals of interest'* screened for tainting.

Chemicals of interest	Effluent concentration after dilution (µg/L)	ANZECC tainting guideline (µg/L)	US EPA organoleptic water quality criteria (µg/L)
Metals			
Aluminium	6		
Antimony	0.014		
Arsenic (III &V)	0.029		
Barium	0.06		
Beryllium	0.015		
Boron	0.23		
Cadmium	0.012		
Chromium (III & VI)	0.20		
Cobalt	0.03		
Copper	0.20	1,000	1,000
Iron	8.17		
Lead	0.03		
Manganese	8.17		
Mercury	0.003		
Molybdenum	0.014		
Nickel	0.28		
Selenium	0.075		
Silicon	0.570		
Tin	0.138		
Vanadium	0.014		
Zinc	0.847	5,000	5,000
Non metallic inorganics			
Ammonia	< 4.55		
Hydrogen sulphide			
Nitrate	< 182		
Thiolignin			
Thiosulphate			
Chlorate	19		
Organic acids	45		
Chloroacetic acids	41		
Monochloroacetic acid	13.5		
Dichloroacetic acid (include methyl ester)	13.5		
Trichloroacetic acid	13.5		
Resin acids	**2.50**		
14-chlorodehydroabeitic acid	0.50		

Chemicals of interest	Effluent concentration after dilution (µg/L)	ANZECC tainting guideline (µg/L)	US EPA organoleptic water quality criteria (µg/L)
12,14-dichlorohydroabietic acid	0.50		
Abietine	0.50		
Arakine	0.50		
Chlororetene	0.50		
dehydroabietic acid	0.50		
Fichtelite	0.50		
Isopirame	0.50		
Levopirame	0.50		
Neoabietine	0.50		
Palustrine	0.50		
pimaric acid	0.50		
Retene	0.50		
Fatty acids	**1.90**		
(2E,4Z)-Hexadienedioic acid monomethyl ester	0.40		
Behene	0.40		
Lignoserine	0.40		
Linoleine	0.40		
Myristine	0.40		
Oleine	0.40		
Palmitine	0.40		
Stearine	0.40		
Linoleic acid	0.40		
Chlorinated natural phenolics	**0.24 (total)**		
4-chlorocatechol	0.05		
3,4-dichlorocatechol	0.05		
3,5-dichlorocatechol	0.05		
2-chloro-p-cymene	0.05		
5-chloro-o-cymene	0.05		
2,3-dichloro-p-cymene	0.05		
2,5-dichloro-p-cymene	0.05		
2,6-dichloro-m-cymene	0.05		
4-chloroguaiacol	0.05	8 (Guaiacol)	

Chemicals of interest	Effluent concentration after dilution (µg/L)	ANZECC tainting guideline (µg/L)	US EPA organoleptic water quality criteria (µg/L)
3,4-dichloroguaiacol	0.05	8 (Guaiacol)	
4,5-dichloroguaiacol	0.05	8 (Guaiacol)	
4,6-dichloroguaiacol	0.05	8 (Guaiacol)	
2-chloroacetoguaiacones	0.05		
5-chloroacetoguaiacones	0.05		
6-chloroacetoguaiacones	0.05		
2-Monochlorosyringaldehyde	0.05		
2,6-dichlorosyringaldehyde	0.05		
2-Chloro-3,4,5-trimetoxybenzaldehyde	0.05		
2,6-Dichloro-3,4,5-trimetoxybenzaldehyde	0.05		
2-chlorovanillin	0.05		
5-chlorovanillin	0.05		
6-chlorovanillin	0.05		
2,5-dichlorovanillin	0.05		
2,6-dichlorovanillin	0.05		
5,6-dichlorovanillin	0.05		
Dichloroveratrole	0.05		
Chlorophenols	**0.01 (total)**		
2-Monochlorophenol	0.002	0.1 - 15	0.1
3-Monochlorophenol	0.002		0.1
4-Monochlorophenol	0.002	0.1	0.1
2,3-Dichlorophenol	0.002		0.04
2,4-Dichlorophenol	0.002	0.1 - 14	0.3
2,5-Dichlorophenol	0.002	20	0.5
2,6-Dichlorophenol	0.002	30	0.2
3,4-dichlorophenol	0.002	0.3	0.3
3,5-dichlorophenol	0.002		
4-Chloro-3-methylphenol	0.002	20 – 3,000	3,000
Other aromatic compounds	**4.75 (total)**		
Sterols	**4.28**		

Chemicals of interest	Effluent concentration after dilution (µg/L)	ANZECC tainting guideline (µg/L)	US EPA organoleptic water quality criteria (µg/L)
beta-sitostanol	0.90		
beta-sitosterol	0.90		
Fucosterol	0.90		
Betulinol	0.90		
Campesterol	0.90		
Stigmasterol	0.90		
Squalene (steroid hydrocarbon)	0.90		
Miscellaneous aromatics	**0.48** **10% of total**		
2,4-Dimethylphenol	0.10	400	400
2-Methylphenol	0.10	400	
2-Nitrophenol	0.10		
3-Methylphenol	0.10	200	
4-Methylphenol	0.10	100	
Phenol	0.10	1,000 - 10,000	300
Dichloroacetovanillone	0.10		
a-pinene	0.10		
Benzene	0.10		
b-pinene	0.10		
Camphene	0.10		
Dehydrojuvabione	0.10		
Juvabione	0.10		
Dichloromethylenefuranones	0.10		
chlorodimethylnaphtalenes	0.10		
Alkylchlorophenanthrenes	0.10		
4-chloro-3-hydroxy-2H-pyran-2-one	0.10		
5,5-dichloro-6,6-dihydroxy-2-methoxy-2-cyclohexene-1,4-dione	0.10		
Dichloroprotocatechualdehyde	0.10		
3-methoxy-5-dichloromethylene -2(H5)furanone	0.10		
Aniline	0.10		
3-methoxycatechol	0.10		
p-cymene	0.10		
p-hydroxybenzaldehyde	0.10		

Chemicals of interest	Effluent concentration after dilution (µg/L)	ANZECC tainting guideline (µg/L)	US EPA organoleptic water quality criteria (µg/L)
Syringol	0.10		
Dioxins (PCDD/PCDFs not PCBs or PCBB/PCBFs) I-TEQ	0.00074 pg/l (total)		
Miscellaneous	**0.60 (total)**		
Hydrocarbons			
Methanol	0.12		
Ethanol	0.12		
3-carene	0.12		
Carbon disulphide	0.12		
Hexane (C6)	0.12		
Pentadecane (C15)	0.12		
Octadecane (C18)	0.12		
Nonadecane (C19)	0.12		
cosane (C20)	0.12		
heneicosane (C21)	0.12		
eicosane (C22)	0.12		
tricosane (C23)	0.12		
tetracosane (C24)	0.12		
pentacosane (C25)	0.12		
Limonene	0.12		
Aldehydes and ketones			
Acetaldehyde	0.12		
Pentanal	0.12		
Hexanal	0.12		
Heptanal	0.12		
Octanal	0.12		
Nonanal	0.12		
Acetone	0.12		
1,1-dichloroacetone	0.12		
2-butanone	0.12		
2-pentanone	0.12		
2-hexanone	0.12		
2-heptanone	0.12		

Chemicals of interest	Effluent concentration after dilution (µg/L)	ANZECC tainting guideline (µg/L)	US EPA organoleptic water quality criteria (µg/L)
2-octanone	0.12		
2-nonanone	0.12		
Hydroquinone	0.12		
4-methyl-2-pentanone	0.12		
Dichlorocyclohexendione	0.12		
Chlorinated hydrocarbons			
Chloromethane	0.12		
Dichloromethane	0.12		
1,1-Dichloropropane	0.12		
1,3-Dichloropropane	0.12		
Chloroethylene	0.12		
1,1-Dichloroethylene	0.12		
1,2-Dichloroethylene (cis & trans)	0.12		
1,3-dichloropropene (cis & trans)	0.12		
3-chloropropene	0.12		
1,1-dichlorodimethylsulfone	0.12		
Chlorohydroxypyrone	0.12		

Contextual information on fish tainting

Tainting is defined as any change in flavour or odour in a particular batch of food that is unusual when compared to other products or previous batches (Poels et al. 1988). Although tainting is generally perceived as detrimental this is not necessarily so since it is defined as any change, positive or negative. A comparison of the effluent 'chemicals of interest' against guidelines for tainting did not identify any compounds present in the initial dilution zone at concentrations likely to cause tainting. However only 18 of the 163 chemicals of interest listed in Table A1.3 had tainting guidelines. To qualitatively assess the overall likelihood of fish/shell fish tainting by the discharged Bell Bay mill effluent a literature review was conducted to provide contextual information.

Environmental and general science databases [58] were searched for the words "taint" or "organoleptic" combined with "pulp" or "mill" or "effluent". The references cited within relevant

[58] The databases searched were Medline, Toxline, Biological Abstracts, PIRA, Science Direct, CSA and Web of Science.

papers and bibliographic lists provided further literature. The web sites of many major international regulatory environmental agencies were also searched using the words taint or organoleptic.

The results of the literature search are summarised in Table A7.4. It was found that most articles on tainting and associated with pulp mills were published between the 1970s and early 1990s. Only three articles were obtained that had been published in the last ten years. However in this period there have been many advances in pulp and paper mill technology that have markedly changed both the nature and quantity of effluent released to the environment. Consequently use of historic instances of tainting to predict potential tainting from the 'state of art' Bell Bay mill effluent requires careful consideration.

Although it is difficult to draw comparisons between the Bell Bay final effluent and historical reports of tainting as both the process chemistry and environmental control strategies in the pulp industry have substantially improved (refer Section 4) and considerable differences in processes can exist between different mills. However some general statements can be made:

- Almost [59] all reports of tainting have occurred in freshwater environments. The only study found that investigated potential tainting from a pulp and paper source discharging to a marine environment did not report tainting of fish (flathead) (Mosse and Kowarsky 1995).
- Most papers reporting pulp mill effluents as the cause of fish tainting were published pre-1993. Approximately 25 papers were identified with publication dates prior to 1993[60] and only 5 papers post-1993 (Craig 1993, Mosse and Kowarsky 1995, Redenbach et al. 1997, Lowell et al. 2003, Lowell et al. 2005).
- Improvements in effluent treatment processes have resulted in either elimination or extensive reduction of the tainting ability of individual pulp mill effluents (Lowell et al. 2005).

One of the issues with the published literature is the pulp mill processes are often poorly documented making it difficult to associate tainting with certain types of plants or processes. Field tainting studies are performed downstream from effluent release points or in laboratory based

[59] An Environment Canada report authored by McLeay (1987) cites an unavailable Fisheries and Marine Service report (Bell & Kallman 1976) in which tainting of fish in an estuarine environment at about the start-up time of a pulp mill is alleged, however McLeay (1987) points out no factual evidence was presented in the report.

[60] Swabey 1965, Wells 1967, Shumway and Chadwick 1971, Shumway and Palensky 1973, Cook et al 1973, Langford 1974, Langer and Nassichuk 1975, Bell and Kallman 1976, Whittle and Flood 1977, Liem et al 1977, Brouzes et al 1978, Rogers 1978, Findlay and Naish 1979, Gordon et al 1980, Weinbauer et al 1980, Kuusi and Suihko 1981, Miettinen et al 1982, Paasivirta 1982, Kovacs 1982, Paasivirta 1983, Persson 1988, Jardine 1992, Lindsay and Heil 1992, Paasivirta 1992.

experiments. Results are given as a percentage by volume of effluent causing tainting or, in some cases, are linked to certain components of the effluent. For a variety of purposes the traditional effluent monitoring parameters such as BOD, COD, and AOX provide an indication of the 'strength' of the effluent and allow different effluents to be roughly compared prior to dilution. Unfortunately these parameters were not provided in the tainting literature making judgment of effluent relativities difficult as the original 'strength' of the effluent is unknown.

Chlorate

Although chlorate was not detected in any of the biologically treated effluents examined by Stromberg et al. (1996) the mill designers have nominated a concentration of 1.9 mg/L as potentially being present in the discharged effluent. Thus at the DZ100 the concentration could be about 20 µg/L. Tainting guidelines were not found for chlorate and a literature search[61] did not yield any information.

Chlorates are very water soluble , range 7 – 100g/100ml water and consequently have very limited solubility in non-polar solvents. A log K_{OW} was not located. It is therefore quite unlikelt that chlorate will accumulate in aquatic organisms.

The taste and odour threshold for ClO_2 in water as cited by WHO (2005) and HC(2005) is 0.4 mg/L, this seems to be based on a Russian study that is not available. Since ClO_2 dissolved in water will form chlorate this odour/taste threshold may inform on the likelihood of chlorate taste effects in fish from discharged chlorate in the effluent. The water ClO_2 odour/taste threshold is 20 times higher than the concentration of chlorate at the DZ100, which together with the presumed low fat solubility suggests there may be little likelihood of tainting of fish.

[61] The search was conducted using the word "chlorate" AND "taste OR taint*OR organoleptic on the following data bases. Web of Science, Medline, Environmental Pollution and Control Management, biological abstracts.

Taint testing

The general procedure for taint testing is schematised in Figure A7.1. Rainbow trout is the species recommended for testing because it has a moderate fat content, is ready available and is used in other ecotoxicological testing. Rainbow trout can acclimatise to fresh or sea water, therefore testing can be performed in either environment. However flathead have been used in testing the effluent of Australian pulp mills (Mosse and Kowarsky 1995). Studies performed in fresh and sea water fish environments have been shown to produce similar results (Poels et al. 1988).

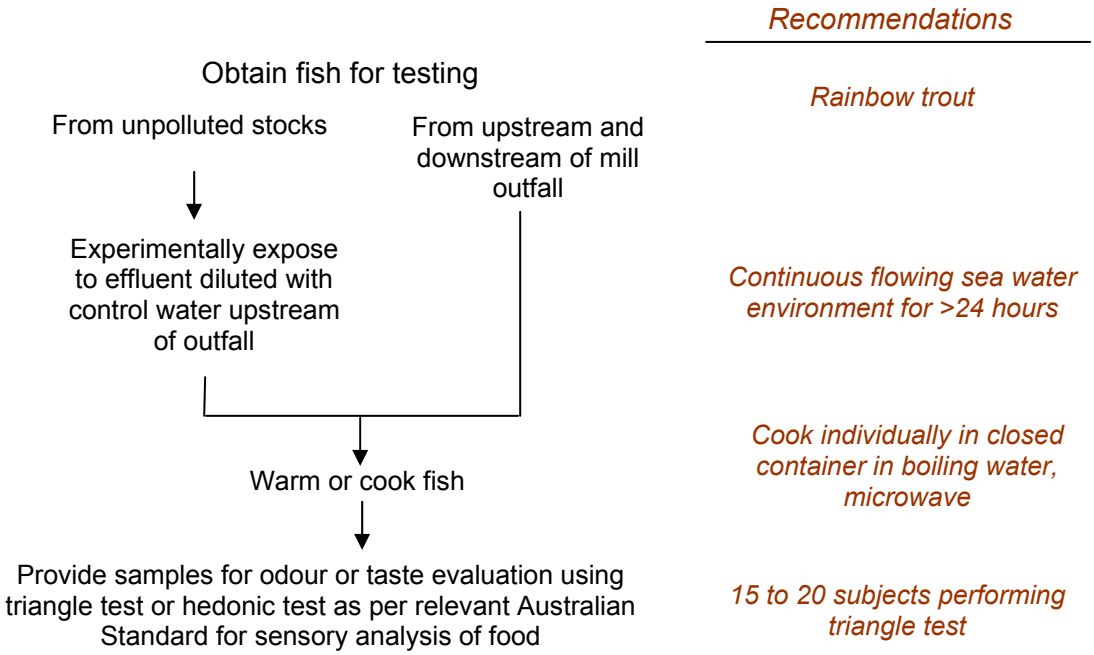


Figure A7.1: Scheme for taint testing and recommendations suggested by Poels et al. (1988).

Because taste is markedly influenced by smell Poels et al. (1988) evaluated testing using taste and smell, or smell alone. The results were similar with both conditions and the authors concluded either odour or taste can be used as the evaluation parameter for tainting. The recommended evaluation technique for sensory analysis of food by either taste or smell is the triangle test (Australian Standards 2005). Each panel member receives three samples (two are the same and

the third is different) and has to report which sample is different. They may be asked to indicate whether the different sample is better or worse (Poels et al. 1988). A table is provided by the Australian Standards (2005) of the minimum number of panel correct responses for a difference to be scored between 'treated' and 'control' samples.

Very few of the literature reports on tainting by mill effluent followed the above test and evaluation recommendations. Australian Standards (1988) recommends a large linear scoring scale (e.g. between 7 and 15 categories, from strongly like to strongly dislike) to minimise biased results. However studies often used a scale of less than 5 and in some cases only measured the extent of dislike of the sample.

Compounds likely to cause tainting

Many constituents of mill effluents have been suggested to cause tainting. These include chlorinated compounds (Persson 1984, Paasivirta et al.1992), phenolics (Lindsay and Heil 1992, Paasivirta et al. 1983), resin acids (Findlay and Naish 1979), organosulphur compounds (Rogers 1978, Findlay and Naish, 1979) and monoterpenes (Rogers 1978, Findlay and Nash 1979, Craig 1993). However very little analytical evidence exists to confirm the historical tainting associated with pulp mill effluent is primarily due to these compounds (McLeay 1987). However improvements in analytical analysis have enabled comparision of trace organic concentrations of substances with taste panel evaluations. These have associated chlorophenols and alkylphenols with tainting and are reported in more detail below (Paasivirta et al. 1987, Lindsay and Heil 1992).

Lindsay and Heil (1992) collected fish from a section of the Upper Wisconsin River industrialized with pulp and paper manufacturing industries. Fish were vacuum packaged and frozen prior to analysis. Samples were split, part was steam distilled to obtain volatile compounds that were analysed with gas chromatography and mass spectrometry. Taste tests were performed on the other sample portion using a rank analysis of off-flavour intensity and overall preference of fish. They identified alkylphenols and thiophenols in fish lead to a chemical-petroleum-phenolic flavour. Intentional tainting of fish with alkylphenols yielded concentrations of individual phenols required to produce an off-flavour (Table A7.2). Alkyl phenols are suggested to have synergistic interactions, allowing low concentrations to produce tainting. The only chlorinated phenol present was 2,4,6-

trichlorophenol and it was only present in one sample, therefore it was not considered the cause of tainting of fish in the Upper Wisconsin River.

Table A7.2: Critical concentrations of phenolics required to cause different tastes in fish.

Compound	Concentration (µg/kg) [b]	Flavour
2,4-diisopropylphenol	3	Phenolic, musty
2,4-diisopropylphenol	30	Sharp, petroleum-like, anise-like
3-isopropylphenol	100	Petroleum-like
Carvacrol	100	Petroleum-like
Thiophenol	20	Paper-like/ Cardboard-like
2-isopropylphenol	100	Paper-like/ Cardboard-like
3-isopropylphenol	10	Paper-like/ Cardboard-like

[a] Data from Lindsay and Heil 1992.
[b] Concentration added to fish mince before cooking.

Other studies have suggested that chlorinated compounds, e.g. chlorinated natural phenols or their breakdown products may cause tainting. Paarsivita et al. (1987) collected fish from three lakes in Finland and subjected them to gas chromatogram analysis and taste testing. Twenty people were used for taste test analysis to rank fish pieces from 0 (very good) to 3 (very bad). Samples from above the outfall received taste ratings of 0.5 – 1.0, those 5km downstream rated 1.4 – 2.0 and those 60km downstream rated between 0.6 – 1.0. The most significant correlation between the taste value and chlorinated phenols in the fish was for the sum of all the polychlorophenols (Table A7.3). The authors suggest that the tainting is due to the presence of polychlorinated anisoles and polychlorinated veratoles as these are present at the same concentrations as the polychlorinated phenols but have lower taste thresholds (Paarsivita et al. 1987). A similar conclusion was reached for pulp mill associated mussel tainting in lakes of Finland (Paarsivita et al. 1992) and fish by chlordisinfection of water (Paarsivita et al. 1983).

Polychlorinated anisoles and veratoles are not expected to be present in the Bell Bay effluent because formation polychlorinated phenols are virtually eliminated in Bell Bay pulp mill effluent (refer Section 4).

Table A7.3: Relationship between polychlorinated compounds in fish and taste.

Site	Concentration (ng/g)		Taste
	SPCP [b]	SANIS [c]	

Upstream of mill discharge	2.2-6.1	0.2	0.5-1.0
5km downstream of discharge	11.5-73.6	16.3-40.3	1.4-2.0
60km downstream of discharge	10.0-37.7	0.2-5.3	0.6-1.0

[a] Data from Paasivirta et al. (1987).

[b] SPCP= sum of polychlorinated phenols (includes tri, tetra and penta chlorinated phenols).

[c] SANIS = sum of chloroanisoles (includes tri, tetra and penta-chlorinated phenols).

Effluent concentrations causing tainting

Very few studies on the pulp mill effluents and tainting were found since the introduction of ECF plants in the early 1990s. This is suggestive that the historical problem of fish tainting is not characteristic of modern mill effluents.

In reports documenting an association between pulp mill effluent and fish tainting minimal information on the mill processes have been provided, this prevents meaningful comparison between those mills and the proposed Bell Bay mill. Nonetheless it can be inferred from the publication dates of the studies (field and experimental data being generated a few years earlier) that the tainting was associated with mill processes that have since been upgraded to prevent the problem. There is however an Environment Canada report (Lowell et al. 2005) that indicates at one pulp mill in Canada was causing fish tainting during 2000 – 2004, specific information for this mill was not provided in the report. In addition it is noted the Bell Bay mill will be releasing effluent to a marine environment whereas the majority of tainting studies have been performed in freshwater systems that are relatively enclosed. Only one report investigating fish tainting from pulp mill effluent discharged into marine environment was located in the literature search, this was for the Australian mill at Maryvale mill and no evidence of tainting was found (Mosse and Kowarsky 1995, described in greater detail below).

The Mosse and Kowarsky (1995) study was for a combined ocean outfall that contained secondary treated effluent from the Maryvale pulp and paper mill, plus domestic and light industrial waste from several small towns plus formation water from the Longford gas plant. Sand flathead from Port Phillip Bay were experimentally exposed to continuous flow of effluent at concentrations of 1.3% and 2.5% in sea water for 72 hours. These concentrations were chosen on the basis that 1.3% was a 75 fold dilution and also the minimum design criterion for the outfall diffuser, 2.5% was chosen as an extreme case being a 40 fold dilution and roughly one half the design dilution requirement of the diffuser. At the end of the exposure fish were filleted and frozen. Partly thawed flesh was minced,

aliquots wrapped in aluminium, warmed to 60^0C in a water bath and assessed for odour tainting by a panel of 18 people using the triangle test according to Australian Standard AS 2542.2.2. Panellists were required to select the odd sample, whether the smell was better or worse and provide a written comment. There was no evidence that fish exposed to either concentration of effluent could be distinguished from control fish exposed only to sea water. No agreement was seen in the assessment of whether the results were more or less pleasant with the only responses for the higher concentration being reported as being possibly different or more pleasant in odour. The minority of panellists who 'correctly' identified the effluent treated fish reported that the odour of these fish was slightly stronger than that of the controls, but they did not indicate a clear preference for either exposed or control fish. It is noted however the warmed fish flesh samples were able to cool down during the testing which may have possibly compromised the results (Mosse and Kowarsky, 1995).

From the data summarised in Table A7.4 it is apparent that effluents tested up to the late 1990s were able to cause tainting of fish at concentrations of approximately 1% or more, its appears that a threshold for tainting for this era effluent may be about 0.1%. However as noted by several authors there are very large differences between mills (e.g. Persson 1984, Jardine 1992, Redenbach 1997) hence it may not be possible to define a generic concentration for these historic effluents that is unlikely to cause tainting.

Rainbow trout were tested *in situ* at two pulp mill sites in Canada and yellow perch were netted and assessed from one of these sites. The first site was downstream of a pulp mill which had been upgraded in 1983 to include oxygen delignification, secondary waste treatment and steam stripping condensate towers. Testing was performed in close proximity to the mill. The second mill was upgraded in 1972 with an effluent diffuser and a clarifier to reduce the effect on fish tainting. Due to the rapid flowing water at the second mill the test fish were caged approximately 1000m downstream and a 1 in 10,000 dilution occurred prior to the contaminant reaching the test site. Fish remained *in situ* 72 hours before removal and freezing. Sensory evaluation of odour, using the triangle test, were performed panels of 8 and 17 members. Results showed no flavour tainting in the rainbow trout at either site. However the yellow perch netted at the second site were found to be tainted (correctly identified in 49 of 59 exposures) and the odour was described as "sulphur dioxide", "petroleum smell" and "stagnant water/ earthy smell" (Jardine 1992).

Analysis has been performed both on whole mill effluents and on components of the effluent. Tainting thresholds are lowest for the condensates, with tainting occurring at concentrations as low as 0.007%. This component constitutes approximately 8% of the total effluent (Findlay and Naish 1979). Similarly, Farmer et al. (1973) and Cook et al. (1972) suggested tainting was caused by kraft mill effluent condensates, digester primary and secondary foul condensates and recovery furnace flue gas condensates.

In Canada, pulp and paper mills need to conduct environmental effects monitoring (EEM) as part of the requirements of the *Pulp and Paper Effluent Regulations* under the *Fisheries Act*. EEM studies typically consist of a benthic invertebrate survey, a fish population survey, and an analysis of possible impacts on fish usability. The latter includes assessment of fish tainting downstream from effluent discharges. In Cycle 2 of the National programme only two mills conducted tainting studies. Results showed tainting in both environments. At one mill the effluent was determined to be the cause of the tainting, however the tainting couldn't be linked to the second mill and may be the result of other industrial discharges in the region (Lowell et al. 2003). Further testing was performed in Cycle 3 of the program on the same sites. One site was found to have no tainting whereas the other site was still found to taint. However the intensity of the tainting was decreased as a result of mill process and effluent treatment improvements (Lowell et al. 2005). Details of the tests and the mills are not provided in the Environment Canada reports authored by Lowell et al. (2003, 2005).

Tainting by a treated unbleached kraft pulp mill was observed at very low concentrations (Redenbach 1997). Eulachon[62] and rainbow trout were exposed for three hours to various concentrations of effluent concentration. Taste tests were performed by 8 to 35 people using a double triangle test. Results showed that tainting occurred at 0.08% with an exposure loading density of 10 to 20g/L

[62] Eucalons are smelts; they have common name of candlefish because they are so high in oil content that they can be dried, fitted with a wick through the mouth and used as a candle (Fisheries and Oceans Canada http://www.pac.dfo-mpo.gc.ca/ops/fm/herring/eulachon/default_e.htm).

Table A7.4: Concentration of effluent causing tainting.

Description	Tainting (Y/N)	Conc (% v/v)	Comment	Reference
Whole mill effluent				
Treated unbleached effluent	Y	0.08	- laboratory test on spawning eulachon and rainbow trout - exposed for 3 to 240 hours - triangle test for difference	Redenbach 1997
Combination of secondary treated bleached kraft pulp mill effluent, industrial and domestic light waste, formation water from oil mill	N	2.5	- laboratory test on sand flathead - held for 72 hours - triangle test - exposure in sea water	Mosse and Kowarsky 1995
Unbleached kraft effluent	Y	NR	- test on oolichan (migratory marine smelt) - taint correlated with a-pinene, cumene and thujane	Crage and Stasiak 1993
Not specified		NR	- Cycle 2 of environmental effects monitoring Canada - two mills tested and found to taint (results form one mill could not confirm that the mill was the cause of the tainting) - Cycle 3 of environmental effects monitoring Canada - same mills tested - one showed no tainting - other mill continued to taint but decreased intensity due to mill process and effluent treatment improvements	Lowell et al. 2005 Lowell et al. 2003
Whole kraft mill effluent	TTC[a]	0.1		Findlay and Naish, 1979
Untreated bleached kraft whole mill effluent	Y	0.2-0.8	- exposed for 4 hours - triangle test	Gordon et al 1980
Untreated bleached kraft mill	Y	1	- laboratory test on rainbow trout - exposed for 24 or 96 hours - hedonic test (0 to 10) by six people - 1% ranked 6.6 and 2.5% ranked 3.6 (control 8.0)	Miettinen et al. 1982
Bleached kraft whole mill effluent	Y	1	- laboratory test on rainbow trout - triangle test	Liem et al. 1977

Untreated unbleached kraft mill effluent	Y	1-2	- laboratory test on salmon - held for 96 or 72 hours - effluent from settling lagoon - 0 (least off flavour) – 6 (most off flavour) ranking -control 0.87 – 1.9 - greater than 1% 1.8 – 3.4	Shumway & Chadwick 1971
Treated (aerated lagoons) bleached kraft whole mill effluent	Y	2-2.9	- exposed for 4 hours - triangle test	Gordon et al. 1980
Treated (activated sludge) bleached kraft mill	Y	2.5	- laboratory test on rainbow trout - exposed for 24 or 96 hours - hedonic test (0 to 10) by six people - no tainting at 1% - 2.5% ranked 6.2 (control 8.0)	Miettinen et al. 1982
Biologically treated unbleached kraft mill effluent	N	2.9	- laboratory test on salmon - held for 96 or 72 hours - effluent from settling lagoon - 0 (least off flavour) – 6 (most off flavour) ranking - biological floc treatment in laboratory	Shumway and Chadwick 1971
Treated (steam stripping) bleached kraft mill	Y	3	- laboratory test on rainbow trout - exposed for 48 or 144 hours - hedonic test - no tainting at concentration of 2% - steam stripped treatment of effluent	Whittle and Flood 1977
Treated bleached kraft whole mill effluent	N	4	- laboratory test on rainbow trout - exposed for 8 or 48 hours - hedonic test	Langer and Nassichuk, 1975
Bleached kraft whole mill effluent	Y	5	- laboratory test on rainbow trout - triangle test	Liem et al. 1977
Bleached kraft whole mill effluent	Y	5	- laboratory test on perch - triangle test	Liem et al. 1977
Kraft waste water	Y	5-7	- laboratory test on rainbow trout - hedonic test	Shumway and Palensky 1973
Kraft effluent, biologically stabilised	Y	5-8	- laboratory test on rainbow trout - hedonic test	Shumway and Palensky, 1973

Untreated kraft mill effluent	Y	5–10	- laboratory test on perch - held for 7 days to up to 6 weeks - freshwater - triangle test - no tainting at 0.33%	Cook et al. 1973
Bleached kraft mill effluent/ mechanical pulping effluent	Y	NR[b]	- test on pike, pickerel - fish from small river	Swabey 1965
Bleached kraft mill effluent/ mechanical pulping effluent	Y	NR	- test on whitefish - distance from outfall 1.5km - fish from freshwater bay	Wells 1967
Bleached kraft mill effluent/ mechanical pulping effluent	N	NR	- test on whitefish - distance from outfall 10km - fish from freshwater bay	Wells 1967
Bleached kraft pulp mill	Y	NR	- test on trout, whitefish - fish from river - tainting was not demonstrated to be caused by pulp mill effluent	Langford 1974
Bleached kraft mill	Y	NR	- test on trout - fish from river - distance from outfall is less than 1km - tainting was not demonstrated to be caused by pulp mill effluent	Langer and Nassichuk 1975
Treated unbleached kraft effluent	Y	NR	- estuary environment - report of tainting following pulp mill introduction - no factual evidence to document	Bell and Kallman 1976
Historic bleached kraft/ integrated bleached kraft/groundwood mill	N	NR	- test on perch	Liem et al. 1977
Not specified	Y	NR	- organosuphur compounds suggested to cause tainting - no analytical proof available	Rogers 1978

Bleached kraft/ mechanical/ sulphite pulp mill	Y	NR	- test on pike - fish from river - tainting was not demonstrated to be caused by pulp mill effluent - hedonic test 0 (same or better) – 4 (repulsive taste) - samples had slight taste impairment compared to control fish - palatability by itself was good - fish upstream had higher taste impairment compared to those downstream of the effluent release, therefore non-point source pollution had a greater adverse effect	Weinbauer et al. 1980
Fish from around Finland	Y	NR	- 18 species of fish were captured in Finnish waters - hedonic test (0 completely unfit for consumption – 10 excellent); 5 considered limit of acceptance - 5 – 57% (species dependent) were unacceptable due to kraft pulp mill effluents - brackish water fishes non specific off flavour - fresh water fishes highest off flavour due to kraft pulp mill effluent	Kuusi and Suihko 1981
Bleached kraft mill	Y	NR	- test on white fish - fish from river - tainting was not demonstrated to be caused by pulp mill effluent	Kovacs 1982
Bleached pulp mill	Y	NR	- test on pike - hedonic scale 1 (no off flavour) to 4 (strong off flavour) - correlation between hexachlorobenzene, trichlorophenol, tetrachlorophenol and off taste	Paasivirta et al. 1983
Kraft pulp mill with biological purification plant	Y	NR	- test on pike and burbot - hedonic test from 0 (very good) to 3 (very bad) - distance 5 – 10 km below pulp mill - taste value of 1.4 – 2.0 - correlated with polychlorinated phenolics, polychlorinated anisoles and polychlorinated veratoles - at 60km from pulp mill no difference between control and sample was observed	Paasivirta et al. 1987
Kraft mill effluent/ sulfite mill effluent	Y	NR	- test on perch, pike, bream, roach - fish from lake - distance from outfall is 0 – 17.5km	Persson 1988

Kraft pulp mill with biological purification plant	Y	NR	- test on mussels - hedonic test (0 no off flavour to 3 very bad taste) - incubations of mussels for 4 weeks at site - significantly higher taste (1.94 vs 1.47) at site 5 km downstream of pulp mill - no difference in off taste observed 60km downstream of pulp mill - tainting was expected to result from the presence of chloroanisoles, chloroveratoles - correlations between tainting and chemical concentration were also observed for chlorophenols, PCBs and chlorocymenes	Paasivirta et al. 1992
Unknown number and type of mills	Y	NR	- test on pike, black crappie, yellow perch, bullhead - fish collected from Upper Wisconsin river - hedonic test for off-flavour intensity (1 none to 7 extremely pronounced) & overall preference (1 – dislike extremely to 7 like extremely) - increase in off-flavour intensity from 2.34 (control area) to 3.97-4.53 (downstream regions) - decrease in preference from 4.99 (control area) to 2.98-3.48 (downstream regions) - taint correlated with alkylphenols and thiophenols	Lindsay and Heil 1992
Bleached kraft mill	N	NR	- testing performed on trout - fish from river - distance from outfall is 1km	Jardine 1992
Bleached kraft mill	N	NR	- testing performed on trout - fish from river - distance from outfall is 2km	Jardine 1992
Bleached kraft mill	Y	NR	- testing performed on perch - fish from river - distance from outfall is 12km	Jardine 1992
Mill effluent components/processes				
Recovery (condensates & scrubber effluent) from whole kraft mill effluent	TTC[a]	0.007-1		Findlay and Naish, 1979
Kraft foul condensates	Y	0.05	- laboratory test in rainbow trout	Brouzes et al 1978

Evaporator condensate from untreated kraft mill effluent	Y	0.7	- laboratory test on perch - held for 7 days to up to 6 weeks - freshwater - triangle test	Cook et al 1973
Bleach plant effluent from whole kraft mill effluent	TTC[a]	>1		Findlay and Naish, 1979
Vanillin plant main sewer from untreated kraft mill effluent	N	1.0	- laboratory test on perch - held for 7 days to up to 6 weeks - freshwater - triangle test	Cook et al 1973
Digester foul water condensate from untreated kraft mill effluent	Y	1.1	- laboratory test on perch - held for 7 days to up to 6 weeks - freshwater - triangle test - no tainting at 0.55%	Cook et al 1973
West sewer kraft mill from untreated kraft mill effluent	N	1.2	- laboratory test on perch - held for 7 days to up to 6 weeks - freshwater - triangle test	Cook et al 1973
Unbleached pulp thickener from untreated kraft mill effluent	N	1.2	- laboratory test on perch - held for 7 days to up to 6 weeks - freshwater - triangle test	Cook et al 1973
Kraft bleach plant from untreated kraft mill effluent		1.2	- laboratory test on perch - held for 7 days to up to 6 weeks - freshwater - triangle test - one sample showed tainting whereas the second sample showed no tainting	Cook et al 1973
Causticizing effluent from untreated kraft mill effluent	N	1.4	- laboratory test on perch - held for 7 days to up to 6 weeks - freshwater - triangle test	Cook et al 1973

[a] TTC = tainting threshold concentration, [b] NR = not reported.

A7.6 References

ANZECC (2000). Australian and New Zealand guidelines for fresh and marine water quality. Australian and New Zealand Environment and Conservation Council. October 2000.

Australian Standard (2005). Sensory analysis. Method 2.2. Specific methods – Triangle test. Standard Number 2542.2.2. Standards Australia, Commonwealth of Australia.

Australian Standard (1988). Sensory analysis of foods. Part 2. Specific Methods. Method 2.3. Rating. Standard Number 2542.2.3. Standards Association of Australia, Commonwealth of Australia.

BC MoE (1998). Water quality guidelines (criteria) reports: Chlorophenols. Environmental Protection Division, Water, Air and Climate Change Branch. British Columbia Ministry of Environment. http://www.env.gov.bc.ca/wat/wq/BCguidelines/chlorophenols/chlorophenol.html. Accessed December 2005.

Bell, L.M. and Kallman, R.J. (1976). The Kitimat river estuary. Status of environmental knowledge to 1976. Special estuary series No. 6. Fisheries and Marine Service, Environment Canada. West Vancouver, B.C. As cited in McLeay 1987.

Brouzes, R.J.P., Liem, A.J., and Naish, V.A. (1978). Protocol for fish tainting bioassay. CPAR Rep. No. 775-1. Environmental Protection Service, Ottawa, Ontario. As cited in McLeay, 1987.

Cook, W.H., Farmer, F.A., Kristiansen, O.E., Reid, K., Reid, J., Rowbottom, R. (1973). The effect of pulp and paper mill effluents on the taste and odour of the receiving water and the fish therein. Pulp Paper Mag. Can. 74: 97-106.

Craig, G.R., Stasiak, M. (1993). Fish tainting chemicals – separation, isolation and identification of compounds from pulp and paper effluent. 14th Annual Meeting (SETAC) – Ecological Risk assessment: Lessons Learned? Abstract only.

Findlay, D.M., Naish, V.A. (1979). Nature and sources of tainting in a kraft mill. CPAR Rep No 775-2. Environmental Protection service, Ottawa, Ontario. (As cited in McLeay 1987).

Farmer, F.A., Neilson, H.R., Esar, D. (1973). Flavour evaluation by triangle and hedonic scale tests of fish exposed to pulp mill effluents. Can. Inst. Food Sci. Technol. J. 6: 12-16. As cited in Kenefick et al 1995.

Gordon, M.R., Mueller, J.C., Walden,C.C. (1980). Effect of biotreatment on fish tainting propensity of bleached kraft whole mill effluent. Trans. Tech. Sect. Canad. Pulp. Paper Assoc. Vol 6. TR 2-8. As cited in McLeay 1986.

Jardine, C.G. (1992). Public evaluation of fish tainting from pulp and paper mill discharges. Wat. Sci. Tech. 25: 57-64.

Kenefick, S.L., Low, N.J., Hrudey, S.E. and Brownlee, B.G. (1995). A review of off flavour tainting of drinking water and fish by pulp mill effluents. Wat. Sci. Tech. 31:55-61.

Kovacs, T.G. (1982). The effect of bleached kraft effluent on the aquatic environment. Prepared for Weyerhauser Canada Ltd. Kamloops B.C. by Pulp and Paper Research Institute of Canada. As cited in Kenefick et al 1995.

Kuusi, T. and Suihko, M. (1983). Occurrence of various off-flavours in fish in Finland from 1969 to 1981. Wat. Sci. Tech. 15: 47-58.

Langer, O.E. and Nassichuk, M.D. (1975). Selected biological studies of the Thompson river system. Environment Canada. Technical Report Series No. PAC/T-75-22. As cited in Kenefick et al 1995.

Langford, R.W. (1974). Data review of biological and chemical effects of the Crestbrook pulp mill effluent on the Kootenay river. Fish and Wildlife Branch, B.C. Ministry of Environment. Victoria B.C. As cited in McLeay 1987.

Liem, A.J., Naish, V.A., Rowbootom, R.S. (1977). An evaluation of the effect of inplant treatment studies on the abatement of air wand water pollution from a hardwood kraft pulp mill. CPAR Rep. No. 484-1. Environmental Protection Service, Ottawa, Ontario. As cited in McLeay 1987.

Lindsay, R.C. and Heil, T.P. (1992). Flavor tainting of fish in the Upper Wisconsin river caused by alkyl- and thio-phenols. Wat. Sci. Tech. 25:35-40.

Lowell, R.B., Ring, B., Pastershank, G., Walker, S., Trudel, L., Hedley, K. (2005). National assessment of pulp and paper environmental effects monitoring data: Findings from cycles 1 through 3. National Water Research Institute, Burlington, Ontario. NWRI Scientific Assessment Report Series No 5. 40p.

Lowell, R.B., Ribey, S.C., Ellis, I.K., Porter, E.L., Culp, J.M., Grapentine, L.C., McMaster, M.E., Munkittrick, K.R., Scroggins, R.P. (2003). National assessment of the pulp and paper environmental effects monitoring data. National Water Research Institute, Burlington, Ontario. NWRI Contribution No 03-521.

McLeay, D. (1987). Aquatic toxicity of pulp and paper mill effluent: a review. Environment Canada. Report No. EPS 4/PF/1.

Mosse, P.R.L., and Kowarsky, J. (1995). Testing an effluent for tainting of fish – The Latrobe Valley ocean outfall. Water (Australia). 22: 20-22.

OSPAR (2002). OSPAR list of substances/compound liable to cause taint. OSPAR Convention for the Protection of the Marine Environment of the North-East Atlantic. Meeting of the Offshore industry Committee. Annex 9.
http://www.sft.no.arbeidsomr/petroleum/dokumenter/Anex09_Taint_list.doc

Paasivirta, J., Knuutinene, J., Tarhanen, J., Kuokkanen, T., Surma-Aho, K., Paukku, R., Kääriäinen, H., Lahtiperä, M. and Veijanen, A. (1983). Potential off-flavour compounds from chloro-bleaching of pulp and chlorodisinfection of water. Wat Sci. Tech. 15: 97-104.

Paasivirta, J., Klein, P., Knuutila, M., Knuutinen, J., Lahtiperä, M., Paukku, R., Veijanen, A., Welling, L., Vuorinen, M. and Vuorinen, P.J. (1987). Chlorinated anisoles and veratroles in fish. Model compounds. Instrumental and sensory determination. Chemosphere. 16: 1231-1241.

Paasivirta, J., Rantalainen, A-L., Welling, L., Herve, S., Heinonen, P. (1992). Organochlorines as environmental tainting substances: Taste panel study and chemical analyses of incubated mussels. Wat Sci Tech. 25: 105-113.

Persson, P-E. (1984). Uptake and release of environmentally occurring odorous compounds by fish. Water. Res. 18: 1263-1271.

Persson, P.E. (1988). Off-flavours in fish from waters around a pulp and paper mill in Finland. Verh. Internat. Verein. Limnol. 23: 1751-1755. As cited in Kenefick et al 1995.

Poels, C.L.M., Fischer, R., Fukawa, K., Howgate, P., Maddock, B.G., Persoone, G., Stephenson, R.R., Bontinck, W.J. (1988). Establishment of a test guideline for the evaluation of fish tainting. Chemosphere. 17(4): 751-765.

Redenbach, A.E. (1997). Sensory evaluation of fish exposed to pulp and paper mill effluent: A case study of methods used for environmental effects monitoring. Wat. Sci. Tech. 35: 357-363.

Rogers, I.H. (1978). Environmental effects of terpenoid chemicals: a review. J. Amer. Oil Chem. Soc. 55: 113A-118A. As cited in McLeay 1987.

Shumway, D.L. and Chadwick, G.G. (1971). Influence of kraft mill effluent on the flavour of salmon fish. Water Research. 5(11):997-1003.

Shumway, D.L. and Palensky, J.R. (1973). Impairment of the flavour of fish by water pollutants. U.S. Environmental Protection Agency Report EPA-R3-73-010. Washington, D.C. As cited in Persson 1984.

Swabey, Y.H. (1965). Report on a study of the flavour of fish from the Spanish river and adjacent North Channel. Ontario Water Resources Commission. Toronto, Ontario. As cited in Kenefick et al 1995.

US EPA (2005). Current national recommended water quality criteria. U.S. Environmental Protection Agency. Last updated 26th May 2005. Accessed December 2005. http://www.epa.gov/waterscience/criteria/wqcriteria.html.

Weinbauer, J.D., Thiel, D.A., Kaczynski, V.W., and Martin, C.S. (1980). Receiving stream fisheries studies relative to secondary treated pulp mill effluents. TAPPI. 63(10): 121-125.

Wells, D.L. (1967). Report on the flavour of whitefish from Nipigon Bay 1967. Ontario Water Resources Commission. Toronto, Ontario. As cited in Kenefick et al 1995.

Whittle, D.M. and Flood, K.W. (1977). Assessment of acute toxicity, growth impairment, and flesh tainting potential of a bleached kraft mill effluent on rainbow trout (*Salmo gairdneri).* J. Fish. Res. Boar. Can. 34: 869-878.

Appendix 8: Considerations on accumulation of mercury and human intake.

The assessment of the human health impact from potential accumulation of mercury (Hg) in fish and their subsequent consumption has been conducted in a number of different ways:

- Comparison of the concentration of Hg at the edge of the DZ100 with concentrations in coastal waters of Australia. This approach was hampered by lack of quantitative information for sea water concentrations of Hg in the region of the DZ100.
- The form of mercury that is taken up by biota is methylmercury (MeHg). Hence the incremental MeHg in fish has been estimated and how this might alter current background MeHg fish levels relative to the Food Standards of Australia and New Zealand (FSANZ) standard for fish mercury concentrations.
- Calculation of human intake of food MeHg (background + incremental from effluent) and comparison with the intake considered by FSANZ (2004) considered safe for the developing foetus.

A8.1 Changes in DZ100 mercury concentrations

The highest estimated concentration of Hg in discharged effluent from pulped native eucalypt, plantation eucalypt or pine is 0.275 µg/L (JP 2005e, Section 5 of Table A3.3 in Appendix 3). The estimate was made by the mill designers and is based on analysis of pooled wood chip samples that are representative of feed stock for the Bell Bay mill and application of conservative mass balance assumptions. The form of mercury in the effluent is not known, but is likely to be mercuric salts (i.e. Hg^{++}).

At the edge of the DZ100, the 100 fold mass dilution will result in a Hg concentration of approximately 0.003 µg/L. While this is within the range of Australian coastal and open ocean waters the existing background concentration of Hg in the receiving water should also be taken into consideration. Unfortunately an accurate quantitation of Hg in the water around the proposed diffuser site is unavailable; recent seawater analyses by GHD (2006a) placed the Hg concentration at less than the analytical detection limit of 0.1 µg/L. The Department of Primary Industry, Water and the Environment ( ) found the concentration of mercury in water to be below detection (<0.05 µg/L) at Hebe Reef and other locations in the Tamar

estuary[63]. In Appendix 4 background concentrations of Hg for Australian coastal waters are recorded to be <0.001 – 0.02 µg/L (DEH 1995). Assuming the Hg level at the diffuser site is at the midpoint of this range the concentration would be approximately 0.01 µg/L.

Using this value as the existing background seawater Hg concentration gives a 1:100 dilution of the effluent Hg using seawater as the diluent as follows:

$$[(1 \times 0.275\ \mu g/L) + (99 \times 0.01\ \mu g/L)] \div 100 = 0.013\ \mu g/L$$

This assumed DZ100 concentration is within the concentration range reported for Australian coastal waters and it might therefore be expected that background fish Hg concentrations will not alter very much. However there is a great deal of uncertainty associated with the background Hg concentration assumption; the calculation should only be used to provide a rough indication of the possible impact of discharged effluent Hg on Hg concentrations in seawater surrounding the diffuser. The higher the background seawater Hg concentration the less influence there will be from effluent Hg. The reverse is true if background Hg concentrations are very low.

[63] The Australian and New Zealand Environmental Conservation Council (ANZECC) have established a water quality guideline (WQG) for mercury in marine waters of 0.1 µg/L. The WQG is intended for the protection of aquatic organisms however ANZECC consider that the use of the WQG designed for the protection of areas of high conservation value (i.e. 99th percentile level of protection) to be precautionary for bioaccumulation and biomagnification in aquatic organisms.

A8.2 Incremental increases in MeHg in fish

Calculation of the potential increase in Hg uptake by fish due to Hg in the effluent requires several pieces of information to come together:

- Determination of the effluent derived incremental increase of the bioavailable form of Hg (i.e. MeHg) at the DZ100.
- Identification of a bioaccumulation factor (BAF) for the bioavailable form of Hg.
- Calculation of the incremental increase of Hg in fish using the Equation A8.1.

$$IC_{FISH} = (Hg_{EFF} \times C_{Hg\text{-}MeHg}) \times BAF \quad \text{.....Equation A8.1}$$

Where:

IC_{FISH} = Incremental increase in fish MeHg due to effluent.

Hg_{EFF} = Hg concentration at DZ100 (0.00275 µg/L) due to effluent.

$C_{Hg\text{-}MeHg}$ = Conversion of effluent Hg to MeHg (3%; see below).

BAF = Bioaccumulation Factor for MeHg (320,000 L/kg; see below).

$C_{Hg\text{-}MeHg}$: Nearly all mercury that accumulates in fish is methylmercury (MeHg). Inorganic mercury, which is less efficiently absorbed and more readily eliminated from the body than MeHg, does not tend to bioaccumulate (US EPA 1997). Thus the amount of mercury in discharged effluent available for bioaccumulation is the proportion of mercury that is converted to methylmercury.

A literature search for the proportion of inorganic Hg converted to MeHg in either open ocean or coastal waters revealed a range of 0.03% to 3% based on empirical observations (Mason et al. 1999, Mason & Sullivan 1998, Benoit et al. 1998, Ullrich et al. 2001, Rolfhus and Fitzgerald 1995). For conservative calculation of the amount of MeHg that may be formed from Hg discharged in effluent the top of the observational range was chosen, i.e. 3%.

BAF: The bioaccumulation factor (BAF) is defined as the ratio of MeHg concentration in fish flesh divided by the concentration of dissolved MeHg in the water column. The BAF represents the accumulation of Hg in fish of a specific trophic level from both direct uptake from water and uptake from the food web (i.e. it includes consideration of sediment and sediment dwelling organisms).

A database of bioaccumulation factors has been established by the US EPA (1997) using data either directly obtained from field studies (most of the field studies were conducted in the Great Lakes region of the USA) or obtained indirectly by estimating a BAF from the bioconcentration factor (i.e. transfer factor of mercury from water column only) and 'food chain multiplier' (a factor to account for food chain exposure to mercury). The data was then used to derive probability distributions of bioaccumulation factors for fish (US EPA 1997).

The US EPA (1997) considered the variability in BAF values either directly measured or indirectly estimated and considered the median value of the directly measured BAF was most representative for Hg accumulation in fish in fresh water lake environments. For foraging/predator fish the median BAF was 1,600,000 dry-weight which at 80% water content of fish translates to a BAF of 320,000 on a wet weight basis. The same BAF value has been used by Environment Canada to establish tissue residue guidelines for the protection of wildlife consuming fish (EC 2001) and also by the US EPA in establishing tissue residue guidelines for human consumption of fish (US EPA 2001).

Thus the incremental increase in MeHg concentration due to discharged Hg in effluent is:

$IC_{FISH} = (Hg_{EFF} \times C_{Hg\text{-}MeHg}) \times BAF$ ……**Equation A8.1**

= 0.00275µg/L x 0.03 x 320,000 L/kg

= 26.4 µg/kg wet weight fish (0.026 mg/kg fish).

A8.3 Potential change in measured baseline fish MeHg levels

In two rounds of sampling, April 2005 and February 2006, a total of 39 fish of different varieties have been caught within 250m to the east and west of the proposed diffuser site and analysed for Hg concentration (Aquenal 2005, GHD 2006a). A statistical description of the results is in Table A8.1.

Although 3 of 39 fish[64] (7.7%) had Hg concentrations higher than the FSANZ (2006) standard[65] of 0.5 mg/kg (Figure A8.1) the standard is intended to be applied to the average of 5 or 10 fish from a commercial catch. The average concentration of Hg in the 39 fish was 0.22 mg/kg which complies with the FSANZ (2006) Hg standard for seafood consumption. Twenty four of the 39 fish had Hg levels below the analytical detection limit[66] hence to calculate the statistics in Table A8.1 half detection limit was assumed.

Table A8.1: Statistical descriptors[a] for background concentrations of mercury in fish.

Statistical descriptor	Concentration (mg/kg)
Mean	0.22
Standard deviation	0.15
Upper 95% confidence limit	0.27
Number of samples	39
Number of fish with Hg< DL	24
Maximum	0.6
Minimum (0.5 of lowest DL) [a]	0.05
Median	0.2
Geometric mean	0.18

[a] DL = Detection limit. To calculate the statistics half detection limit (0.5 DL) was assumed for fish whose Hg level was < DL.

[64] Two of the three fish whose Hg levels were greater than the FSANZ standard were flathead, the other was a perch.

[65] The Australia New Zealand Food Standards Code (FSANZ 2004) prescribes two maximum levels for mercury in fish – a level of 1.0 mg Hg/kg for fish that are known to contain high concentrations of Hg (e.g. swordfish, southern bluefin tuna, barrmundi, ling, orange roughy, rays and shark). A level of 0.5 mg/kg is imposed for all other species of fish, crustacean and molluscs.

[66] The analytical detection limit (DL) for Hg in fish was 0.1, 0.2 or 0.5 mg/kg depending on the analytical run. The assignment of 0.5 DL for fish with Hg analytical non-detects in the calculation of statistics was done according to the respective detection limit for the batch within which the specific non-detect fish resided.

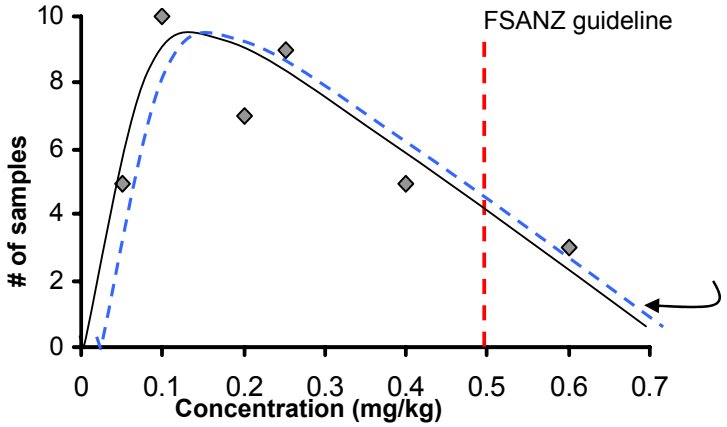


Figure A8.1: Background fish concentrations (mg/kg) and estimated impact of effluent Hg compared to the FSANZ guideline
The blue dashed line represents the estimated increase in fish MeHg concentration due to the effluent. The effluent is expected to increase the concentration of Hg by 0.026mg/kg (Section 8.2).

The FSANZ standard is for the average of 5 or 10 fish sampled from a commercial catch.

From Section A8.2 the estimated incremental increase in fish MeHg concentration is 0.026 mg/kg. This may increase the existing background concentrations of Hg in fish by approximately 12%, i.e. the fish Hg distribution in Figure A8.1 is likely to shift slightly to the right and the mean concentration increase from 0.22 mg/kg to 0.25 mg/kg. In relation to compliance with the FSANZ 0.5 mg/kg Hg fish standard the average of the new distribution will be 50% of the standard and will therefore remain compliant with the standard.

A8.4 Intake of MeHg by humans consuming fish

The determination of human intake of MeHg from fish after the effluent discharge becomes operational requires information on the existing concentration of Hg (assumed to be MeHg) in fish (Table A8.1 and Figure A8.1), the incremental increase in fish Hg due to the effluent (Section A8.2) and fish consumption patterns (Appendix 5). Because the health based guideline for intake of MeHg in Australia is the average intake over a week the monthly consumption information in Appendix 5 needs to be converted to a weekly consumption.

To ensure there is appropriate conservatism in the calculations the following was assumed:

- All the fish consumed by a person was sourced from the outfall. No fish was consumed that was bought from commercial outlets.
- Background fish Hg concentrations were equivalent to the upper 95th percentile level of the concentrations of 39 fish measured to date (Table A8.1). It should be noted 24 of 39 fish had Hg concentrations less than the analytical detection limit and that 0.5 detection limit was assumed for these fish when calculating the statistics.
- The incremental increase in fish concentration is as calculated in Section A8.2. This is founded on an assumed effluent Hg concentration of 0.275 µg/L that is regarded by the mill designers as being worst case. Based on Swedish data[67] the concentration of Hg in effluent could be less than 0.016 µg/L, i.e. approximately 20 times less than that assumed for calculating the incremental increase in fish Hg levels.
- For the amount of MeHg that may be formed from effluent Hg the maximum observed conversion of 3% was used (Section A82).
- The 95th percentile fish concentration was married with various statistical estimates of fish consumption in order to provide an indication of the most likely or 'average' intake of MeHg relative to the worst case scenario.

For judging the likely health impact of MeHg intake the calculated intakes (background plus incremental due to effluent Hg) were compared to the MeHg provisional tolerable weekly intake (PTWI) for women of child bearing age (1.6 µg/kg bw/week) and to the PTWI of 3.3 µg/kg bw/week for the Australian general population (2 years and above) and children aged 2 – 6 years (FSANZ 2004). The PWTI for women and the general population is different because the MeHg induced health effect of concern is subtle neurodevelopment impairment in the foetus. A brief description of the derivation of the PTWI for women of child bearing age is provided in Section A8.6. FSANZ consider the PTWI to be the amount of MeHg that can be consumed safely.

[67] Email to Toxikos from ,Jaakko Pöyry Oy dated 24/11/2005.

The calculations for estimating the human intake of MeHg from fish after the effluent outfall becomes operational are consitent with the calculations and parameters used for dioxin intake from fish (Section 6.3.5.6 and Appendix 5). Thus MeHg intake is calculated from:

$$WI_{FISH}\ (\mu g/kg\ bw/week) = C_F\ (mg/kg\ fish) \times CR\ (kg\ fish/kg\ bw\ per\ week) \times 10^3$$

…… **Equation A8.2**

Where:

WI_{FISH} = Weekly intake of mercury from fish (µg/kg bw/week).

C_F = Concentration of mercury in fish (mg/kg fish)

= $C_{F\ BKGD} + C_{F\ INCREM}$ [$C_{F\ BKGD}$ is the upper 95th percentile of existing fish Hg concentrations, Table 8.1 = 0.27 mg/kg fish]
[$C_{F\ INCREM}$ is the incremental increase in fish Hg from the discharged effluent = 0.026 mg/kg fish].

= 0.27 + 0.026

= 0.3 mg/kg fish.

CR = Consumption rate of fish (kg fish/kg bw/week). Sourced from the Australian Bureau of Statistics (Variable as perTable A8.2).

10^3 = Conversion of mg/kg bw/week to µg/kg bw/week.

Table A8.2 contains data for fish consumption by various sectors of the Australian population as reported in Appendix 5 and converted to consumption rate per kg body per week as required for Equation A8.2.

Table A8.3 shows the human MeHg intake levels for various fish consumption rates for different sectors of the Australian population as calculated from Equation 8.2. A sample calculation is below for adult females at the 95th percentile fish consumption rate:

$$WI_{FISH} = C_F\ (mg/kg\ fish) \times CR\ (kg\ fish/kg\ bw\ per\ week)$$

$$= 0.3 \times 0.0035 \times 10^3$$

= 1 µg/kg bw/week (1 µg/kg bw/week)

Table A8.2: Fish consumption rates.

Population Group		Fish Consumption [a] (g/person/day)			Fish Consumption [a] (CR) (kg fish/kg bw/week)		
		Avg	95%	Max	Avg	95%	Max
Adult [b] (Tasmanian)		31	46	54	0.003	0.005	0.006
Adult Male (Australian)		32.8	38.9	42	0.003	0.004	0.004
Adult Female (Australian)		25.7	32.9	37.1	0.003	0.003	0.004
Child [c] (Australian)	Male	6.9	11	12	0.003	0.005	0.005
	Female	6.5	10	12	0.003	0.005	0.005

[a] Fish consumption data was sourced from the Australian Bureau of Statistics (ABS 1999, Appendix 5, Table A5.3) and converted from grams of fish per day to kilograms of fish per kilogram bodyweight per week (multiply by 7 to convert from 1 day to a week and divide by 10^3 to convert grams to kilograms). That is the (daily fish consumption x 7 ÷ 10^3) ÷ bodyweight. Average body weight of 67 and 66 kg for males and females [FSANZ 2004], 15.5 and 15.3 kg for boys and girls [enHealth 2004] were used.

[b] The ABS data does not separate Tasmanian adult fish consumption by gender, values are for Tasmanians above 19 years.

[c] Child fish consumption values were not found for Tasmanians, data for the general Australian population (2 to 3 year old).

Table A8.3: Human mercury weekly intake from fish.

Population Group		MeHg Intake [a, b, c] (µg/kg bw/week)			PTWI
		Avg	95%	Max	
Adult [b] (Tasmanian)		0.9	1.4	1.8	- [d]
Adult Male (Australian)		1.0	1.2	1.3	**3.3**
Adult Female (Australian)		0.8	1.0	1.2	**1.6**
Child (Australian)	Male	0.9	1.4	1.6	**3.3**
	Female	0.9	1.4	1.6	

[a] Weekly MeHg intakes were calculated using Equation A8.2 and the respective fish consumption data from Table A8.2.

[b] Total fish mercury concentration (background + incremental) is estimated to be 0.3 mg/kg wet weight (Section A8.2).

[c] To each of the estimated MeHg intakes from fish can be added the non-fish MeHg intakes estimated by FSANZ (2004) of 0.02, 0.01 and 0.21µg/kg bw/week for an adult male, adult female and child respectively.

[d] Because fish consumption rates for Tasmanians are not stratified according to gender it is difficult to assign the relevant PTWI to this group.

A8.5 Information on the PTWI for MeHg

The toxic effects of MeHg on the nervous system are are well documented, effects in adults occur at much higher doses than those required for effects in children after *in utero* exposure. The developing nervous system is regarded as the most sensitive target for toxicity with the critical exposure period being during *in utero* development when the brain is developing very rapidly. In the foetal brain, MeHg has the capability of disrupting normal patterns of cell migration and divison. At low levels of exposure the effects are subtle and are similar to mild learning disabilities, careful administration of sensitive neurobehavioural and nueropsychogical tests is required for the effects to be detected.

The Australian PTWI for MeHg in women of child bearing age is 1.6µg/kg bw/week and for the general population is 3.3 µg/kg bw/week (FSANZ 2004). The PTWI for women is based on the evaluation performed by Joint FAO/ WHO Expert Committee on Food Additives (JECFA 2003a). In their re-evaluation of MeHg JECFA established the PTWI based on neurodevelopmental toxicity observed in cohort studies of children whose *in utero* exposure to MeHg was the result of high maternal fish consumption rates (JECFA 2003a). Such mercury exposure was associated with changes in the child's neuropsychological behaviour in the areas of language, attention and memory, and, to a lesser extent, memory related to the visual perception of space and motion (JECFA 2003b). Two studies were used by JECFA, one was for Seychelles Islanders from which a no observed effect level (NOEL) was identified, the other study was for inhabitants of the Faroes Islands from which a bench mark dose (BMD) was estimated. Both populations have a dietary dependence on fish and marine mammals, and both studies used Hg maternal hair concentration as the surrogate for medium term mercury exposure. The average of the NOEL and BMD for neurodevelopmental toxicity was calculated by JECFA to be 14 mg/kg of MeHg in maternal hair.

The average 'NOEL' was converted, using a pharmacokinetic relationship between maternal hair concentration and dose, to obtain a daily intake of 1.5µg/kg bw/day associated with the NOEL. A total uncertainty factor of 6.4 was applied to the converted dose to give a TDI of 0.23µg/kg/day. The uncertainty factor consisted of a factor of 3.2 for interindividual pharmacokinetic variability and a factor of 2 for the interindividual variability present within the conversion of a methylmercury concentration in hair to a methylmercury concentration in blood. No uncertainty factor was considered by JECFA to be required to account for variation amongst sensitive sub-populations as the NOEL was based on exposure during the critical life-stage, resulting in a sensitive toxicological endpoint in two diverse populations. Because the health

effect of concern arises from long term exposures the TDI was converted to a provisional weekly intake (PTWI) of 1.6µg/kg bw/week (JECFA 2003a).

A8.6 Conclusions

From Table 8.3 it can be seen that none of the calculated intakes of MeHg exceed the relevant health based PTWI. Given the conservative assumptions used in the calculations it is concluded the incremental health impact from Hg in the discharged effluent is negligible.

It is noted that existing background intakes dominate the overall intake of MeHg from fish by providing 90% of fish derived MeHg intake after the effluent outfall becomes operational.

A8.7 References for Appendix 8

*ABS (1999). National Nutrition Survey: Foods Eaten. Australia 1995. Report No: 4804. Prepared by McLennan, W. and Podger, A. for the Australian Bureau of Statistics (ABS).

*Aquenal (2005). Aquenal (2005). Gunns Ltd Pulp Mill. Marine biological and pollutant survey at the proposed outfall site.

*Benoit, J.M., Gilmour, C.C., Mason, R.P. and Riedel, G.S. (1998). Behaviour of mercury in the Patuxent River estuary. Biogeochemistry, 40: 249-265.

*DPIWE (2006). Spreadsheet from GHD titled “Data for Tamar River Water Quality Model”. Includes concentrations of metals in water at various locations around the Tamar River including Hebe Reef. Received 06/02/06.

*EC (2001). Ambient water quality guidelines for mercury. Overview report – First update. Ministry of Environment Government of British Columbia. http://www.env.gov.bc.ca/wat/wq/BCguidelines/mercury/mercury.html

*FSANZ (2004). Mercury in fish. Further Information. Food Standards Australia and New Zealand. http://www.foodstandards.gov.au/_srcfiles/FS_Mercury_in_fish_final.pdf

*GHD (2006a). Excel spreadsheet titled “Gunns Monitoring Program and Results”. Worksheet “Pulp Mill. Project. Effluent Outfall Monitoring Program”. Provided by GHD by email 28/06/06. This spreadsheet provides additional sampling results for compounds in fish in the vicinity of the proposed diffuser site.

*JECFA (2003a). Joint FAO/ WHO expert committee on food additives. 61st meeting. Rome, 10-19 June 2003. World Health Organisation. http://www.who.int/ipcs/food/jecfa/summaries/en/summary_61.pdf

*JECFA (2003b). Methylmercury (addendum). WHO Food Additive Series: 52. World Health Organisation. http://www.inchem.org/documents/jecfa/jecmono/v52je23.htm#eva

*JP (2005e). Excel spreadsheet titled “Effluent Loads and WWTP Design.HMS.Dec.05”. One worksheet is cited in the report; “HMEms.New” - details the estimate for concentration of trace metals in the final effluent based on analysis of trace metals in plantation, native eucalypts and pine and the water balance of the Bell Bay mill. Prepared by Jaakko Pöyry Oy sent by Gunns Ltd 19/12/05.

*Mason, R.P., Lawson, N.M., Lawrence, A.L., Leaner, J.J., Lee, J.G.and Sheu, G.R. (1999). Mercury in the Chesapeake Bay. Marine Chemistry, 65: 77-96.

*Mason, R. P. and Sullivan, K., A. (1998). Mercury and methylmercury transport through an urban watershed, Water Res. 32: 321-330.

*Rolfhus, K.R., and Fitzgerald W., F. (1995). Linkages between atmospheric mercury deposition and the methylmercury content of marine fish. Water, Air and Soil Pollution, 80:291-297.

*Ullrich S.M., Tanton, T.W. and Abdrashitova S.A. (2001). Mercury in the aquatic environment: A review of factors affecting methylation. Critical Reviews in Environmental Science and Technology, 31: 241-293.

*US EPA (1997). Mercury Study Report to Congress Volume III: Fate and Transport of Mercury in the Environment. United States Environmental Protection Agency EPA-452/R-97-005 December 1997 http://www.epa.gov/ttn/oarpg/t3/reports/volume3.pdf

*US EPA (2001). Water quality criterion for the protection of human health: methylmercury. Office of Science and Technology, Office of Water, U.S. Environmental Protection Agency. Washington. http://www.epa.gov/waterscience/criteria/methylmercury/mercappa.pdf