APPENDIX J

Toxicological Reference Values (Ecological Health): Derivation of Mammalian and Avian Oral Toxicity Reference Values and Benchmarks for Community Receptors

Table of Contents

List of Tables

List of Figures

1.0 INTRODUCTION

This document outlines the methods and approaches used by Jacques Whitford to determine oral Toxicity Reference Values (TRVs) for mammals and birds and toxicity or regulatory benchmarks for plants, soil invertebrates, and aquatic and sediment biota to be used in Ecological Risk Assessment (ERA). Oral reference doses, the basis for TRV derivation, and toxicity or regulatory benchmarks were obtained from studies found in the primary scientific literature and published government documents (*e.g.,* Ontario Ministry of the Environment (MOE), United States Environmental Protection Agency (U.S. EPA), Canadian Council of Ministers of the Environment (CCME), Oak Ridge National Laboratory (ORNL)) and were used as the benchmark for which toxicological effects of contaminants of potential concern (COPC) could be judged. Preference was given to Provincial values where applicable, otherwise, sources and studies were assessed for reliability (*i.e.,* scientific confidence) and suitability for the ERA through scientific scrutiny and professional judgment.

2.0 UNCERTAINTY FACTORS

The preferred toxicological database that would support a wildlife receptor TRV should include a number of chronic or multi-generational exposure studies of relevant test species (*e.g.,* the ecological receptor of interest or a phylogenetically similar species) to appropriate chemical forms of the substance of interest. One or more relevant biological endpoints such as growth, reproductive effects, or survival should have been assessed. Databases that meet this requirement are available for some chemicals, but in most cases toxicity of contaminants to wildlife are assessed based on responses observed in animals whose body mass is not necessarily similar to wildlife receptors of interest. We suggest here that allometric scaling of dose, though not the only method, and not without its pitfalls, is a scientifically defendable and appropriate way to extrapolate chronic toxicity data between laboratory and wildlife species in ERA.

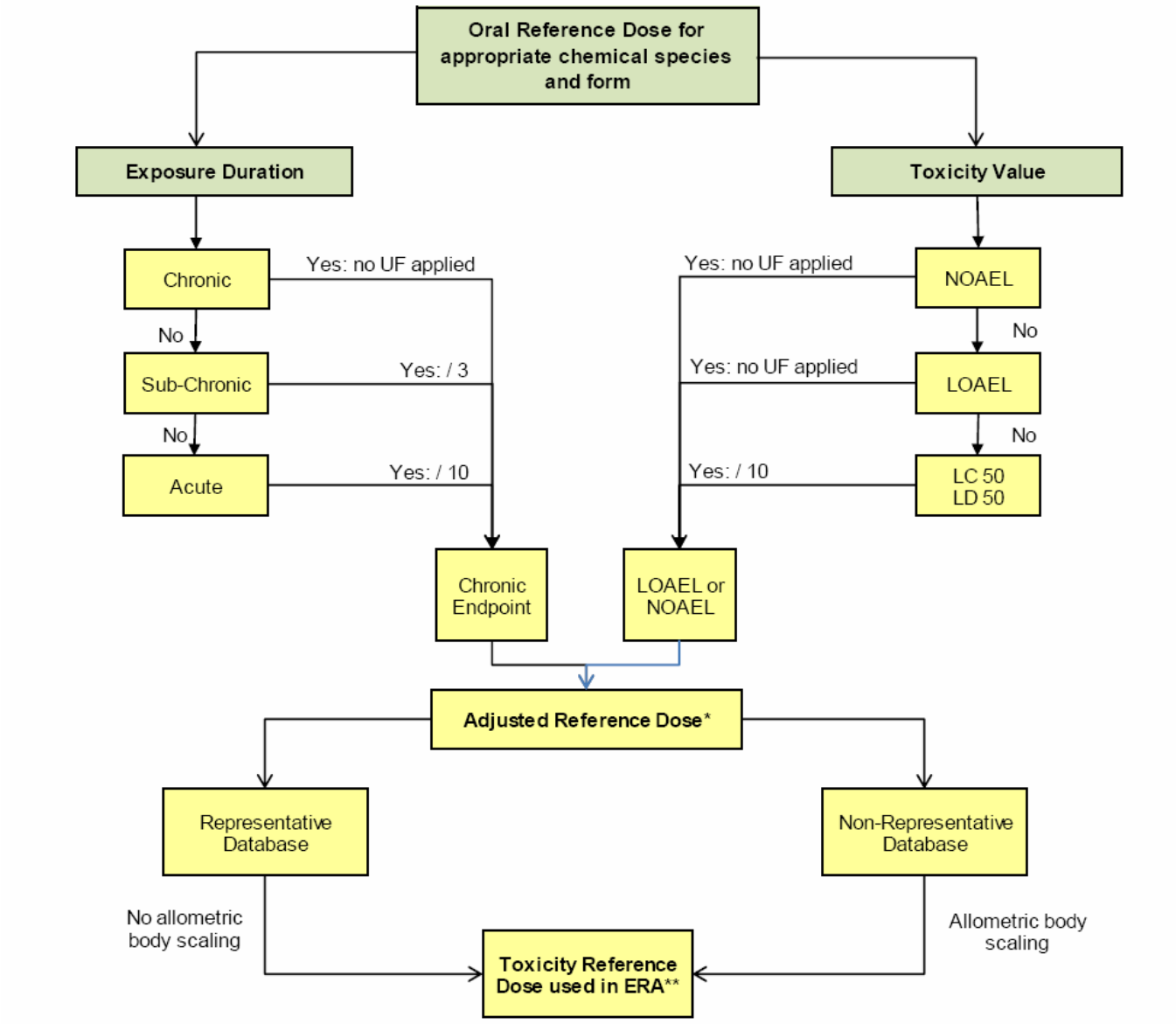
Uncertainty Factors in the toxicological literature are often applied as factors of 10; however, there is no well-defined scientific basis for this practice. As applied in human toxicology, UFs can build upon each other to levels that would be unreasonable for ERA. For example, the U.S. EPA (1994) employs a modification of guidelines for human toxicology proposed by the National Academy of Sciences (NAS 1977; 1980) as follows:

- use 10X when extrapolating from studies using healthy humans, to account for variation in sensitivity among members of the human population;
- use an additional 10X when extrapolating from long-term animal studies to humans;
- use an additional 10X when extrapolating from less than chronic studies on animals (less than chronic No-Observed Adverse Effect Level (NOAEL) to chronic NOAEL);
- use an additional 10X when deriving a reference dose from a LOAEL instead of a NOAEL, to account for the extrapolating uncertainty; and
- use professional judgment to determine another uncertainty factor between 0 and 10, depending upon the overall scientific uncertainty of the study and database not accounted for above.

Therefore, UFs in human toxicology can range up to 10^5. More recent documentation relevant to ERA practice (U.S. EPA 2002) recommends that UFs should range between 1 and 10, with "preferred" values of 1, 3 or 10 (the number 3 is identified as approximating half an order of magnitude on a log scale).

In ERA, toxicological extrapolations can include those between species, those that address the difference between short-term studies and chronic exposures, those related to the selection of toxicological endpoints, as well as other factors that may need to be addressed based upon the professional judgment of the practitioner. The Canadian Council of Ministers of the Environment (CCME, 1997) speaks to the important role of professional judgment in ERA.

Jacques Whitford prefers to use chronic LOAEL data derived from studies that assess reproductive, survival, or growth endpoints, as the basis for predicting wildlife population-level responses to contaminants. The LOAEL-based benchmark represents a threshold level at which adverse effects are likely to become evident (Sample *et al.*1996). The use of the LOAEL is appropriate since a TRV based on the LOAEL will be used as the denominator in the hazard quotient (HQ) calculation, and HQ values equal to or greater than one will be considered indicative of potential adverse environmental effects. In cases where no chronic LOAEL value is available, a NOAEL toxicity value may be selected, or UFs may be applied to other existing exposure and toxicological data using a tiered process to derive suitable ecological TRVs (Figure 2.1). When TRVs are based on U.S. EPA Ecological Soil Screening Levels (Eco-SSLs), NOAELs are often the selected endpoint, but can vary depending on the chemical (see section 4.0). The UF scheme outlined here is based on guidance provided by Ohio EPA (2003, 2008), U.S. EPA (2002), Sample and Arenal (1999) and the professional judgment of Jacques Whitford scientists.



*A NOAEL can be used if no appropriate LOAEL is available but the resultant RfD should be considered more conservative than if it was derived using a LOAEL. Refer to document text for details and application towards species at risk or conservation concern.

**No inter-class UF is used to derive TRVs (i.e., mammalian data is not adjusted for avian TRV).

Figure 2.1 Tiered Approach for the Application of Uncertainty Factors in ERA

2.1 UNCERTAINTY FACTORS FOR EXPOSURE DURATION

In cases where a search of scientific data indicates a lack of chronic studies for a particular contaminant, UFs may be applied to adjust toxicity data to a chronic exposure basis. Acute studies are those that are of short duration, generally less than one week. Sub-chronic exposures are of longer duration (generally less than 90 days), but may be considered equivalent to a chronic study if a critical life stage (such as the gestational period) is included. Chronic exposures would generally be greater than 90 days in length, exceeding 50% of the animal's lifespan or including a reproductive period. Jacques Whitford applies an UF of 3 (half an order of magnitude on a log scale) to adjust from sub-chronic to chronic, and 10 to adjust from acute to chronic. It should be noted that preference is given to longer duration exposure assessments in cases where published data are available, and acute data are relied on only when absolutely necessary.

2.2 UNCERTAINTY FACTORS FOR TOXICITY ENDPOINT

In cases where a search of scientific data indicates the absence of reproductive or other performance-based toxicity endpoints that would indicate a potential for adverse effects at the population level, other less sensitive toxicity endpoints may be considered. Where only a lethal dose (LD_{50}) is available, Jacques Whitford applies an UF of 10 (an order of magnitude) to estimate a LOAEL from LD_{50} data. Again, it should be noted that preference is always given to sub-lethal data, and lethal data are relied on only when absolutely necessary.

Jacques Whitford does not adjust NOAEL values upwards to estimate LOAEL values. Where the only chronic endpoint available is a NOAEL, it is used directly and reported as such in the discussion of uncertainties. Hazard quotient values based on the NOAEL may be permitted to exceed a value of 1.0 since the NOAEL is not an endpoint that signifies toxicological effects.

2.3 UNCERTAINTY FACTORS FOR BODY MASS

In ecological risk assessment (ERA), toxicity of contaminants to wildlife is typically assessed on the basis of toxicity data derived from relatively few species. Laboratory animals such as mice, rats, chickens, quail and ducks are most commonly used, and acute (*e.g.,* LD_{50}, LC_{50}) or chronic (*e.g.,* NOAEL, LOAEL) toxicity endpoints are measured. For use in ERA these laboratory data are often modified, and taxonomic (*e.g.,* genus, family, order) and body mass differences between lab and wildlife species accounted for, by using uncertainty factors with denominations of generally 1, 3, or 10 (US EPA, 1995; Duke and Taggart 2000; Ohio EPA 2003, 2008).

Aside from the use of uncertainty factors, a number of other methods have been used to extrapolate toxicity data (both acute and chronic) between species with different body masses. The application of acute-based extrapolation factors (derived using LD_{50}, HD_5 and standard deviation) to reproductive toxicity data (*e.g.,* Luttik *et al.* 2005), interspecies correlation estimation (ICE) models (Raimondo *et al.* 2007) and allometric scaling (Travis and White 1988; Chappell 1992; Mineau *et al.* 1996, Sample and Arenal 1999) have all been used. These methods are not exclusive of one another and combinations of methods can be applied. Each of these methods has positive and negative attributes, and none is without its drawbacks for extrapolating toxicity data between laboratory and wildlife species. For example, uncertainty factors between 1 and 10 are often arbitrarily assigned with no scientific basis; extrapolation factors require large statistical data sets; ICE models are restrained by limited chronic wildlife data; and there is incomplete chemical specific data to support scaling factors for allometric

relationships, especially for chemicals with daughter compounds that may be more toxic than the parent (Chappell 1992). Ultimately, the choice in method for use in ERA comes to scientific defensibility, practicality based on budgetary and time constraints, and professional judgment. Here we suggest support of the use of allometric scaling for both mammalian and avian receptors in ERA.

In the 1930s, Kleiber (1932) and Benedict (1938) conducted research to assess the relationship between metabolic rate and body mass for avian and mammalian species (Figure 2.2), leading to what is known as the "Kleiber Power Law"; that is body mass raised to the power of 0.75 can be used to predict metabolic rate. Mammals and birds of a wide range of body masses (*e.g.,* 20 g mouse and small birds up to ~3000kg elephants and ~20kg cassowary) were used in the derivation of the Law.

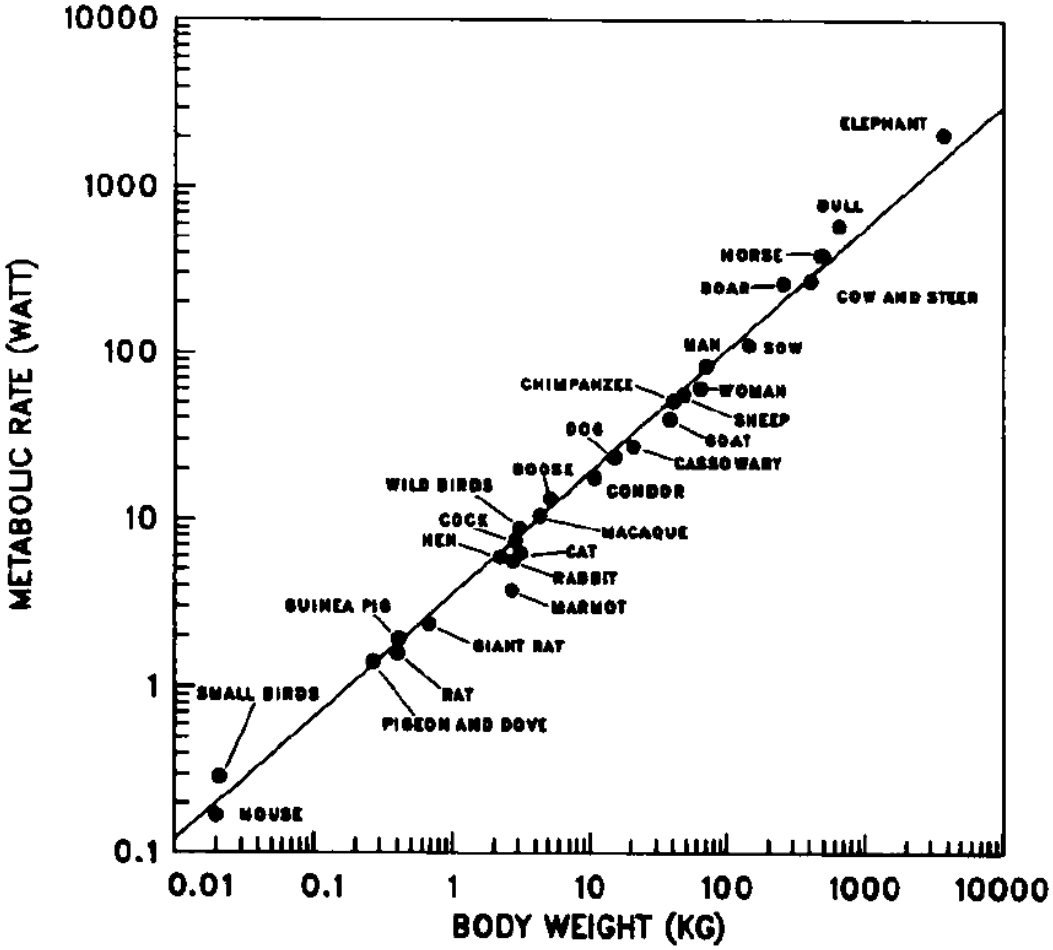


Figure 2.2 Relationship between metabolic rate and body weight, modified Travis and White (1988) and obtained from Schmidt-Nielson (1984). The slope of the line is not significantly different than 0.75.

The rationale to apply allometric scaling to toxicity is grounded to the observation that numerous physiological processes directly associated with metabolism (*e.g.,* uptake, distribution, biotransformation) are related with organismal responses to chemicals, and also follow a predictable relationship with body mass (Travis and White 1988; Chappell 1992; Mineau *et al.* 1996, Sample and Arenal 1999). The slope of the line linking toxicity to body mass is equivocal and extrapolations based on body mass raised to the power of 0.6 to 1.5 have all been suggested for use in toxicity scaling among species with different mass (Davidson *et al.* 1986; Travis and White 1988; Chappell 1992; Mineau *et al.* 1996, Sample *et al.* 1996, Sample and Arenal 1999). There is scientific support for all of

these allometric exponents, but in the cases where empirical evidence is not available for specific wildlife receptors and toxicants encountered in ERA, data from numerous sources support an allometric exponent between 0.60-0.80 (Chappell 1992), specifically body mass raised to the exponent of 0.75 (see below).

In a review of scaling of acute and sub-chronic toxicity data across mammalian species, Chappell (1992) cites two studies supporting a body mass exponent near 0.75. In a study by Krasovskii (1976) it was reported that the acute toxicity of 80-85% of over 100 chemicals could be related to body mass following the allometric scaling (*e.g.,* body mass raised to the power of 0.75). It should be noted, however, that though the relationship between toxicity and body mass held for 80-85% of chemicals tested, the predicted values differed from actual values in certain cases by a factor of up to 3 or 4. Mordenti (1986), using data produced by Freireich *et al.*(1966) that was based on comparisons of acute (measured) and sub-chronic (computed) toxicity data (LD_{10} and maximum tolerated dose, respectively) from mice, rats, dogs, monkeys and humans, reported that an average allometric exponent of 0.74 (range from 0.66-0.87, n=14) could be used to relate doses to body mass. In 1988, Travis and White recalculated the allometric exponent using the Freireich *et al.* (1966) data supplemented with further data from chemotherapy agents (Schein *et al.* 1970). The reanalysis showed that $BM^{0.75}$ was still an appropriate average scaling factor for the 27 compounds, with 95% confidence bounds around the average slope of 0.69 and 0.77. It can also be seen that a certain amount of intra-specifc chemical variation occurs, and as observed with the Krasovskii (1976) and Mordenti (1986) data, 0.75 was an appropriate "average" scaling factor (Figure 2.3).

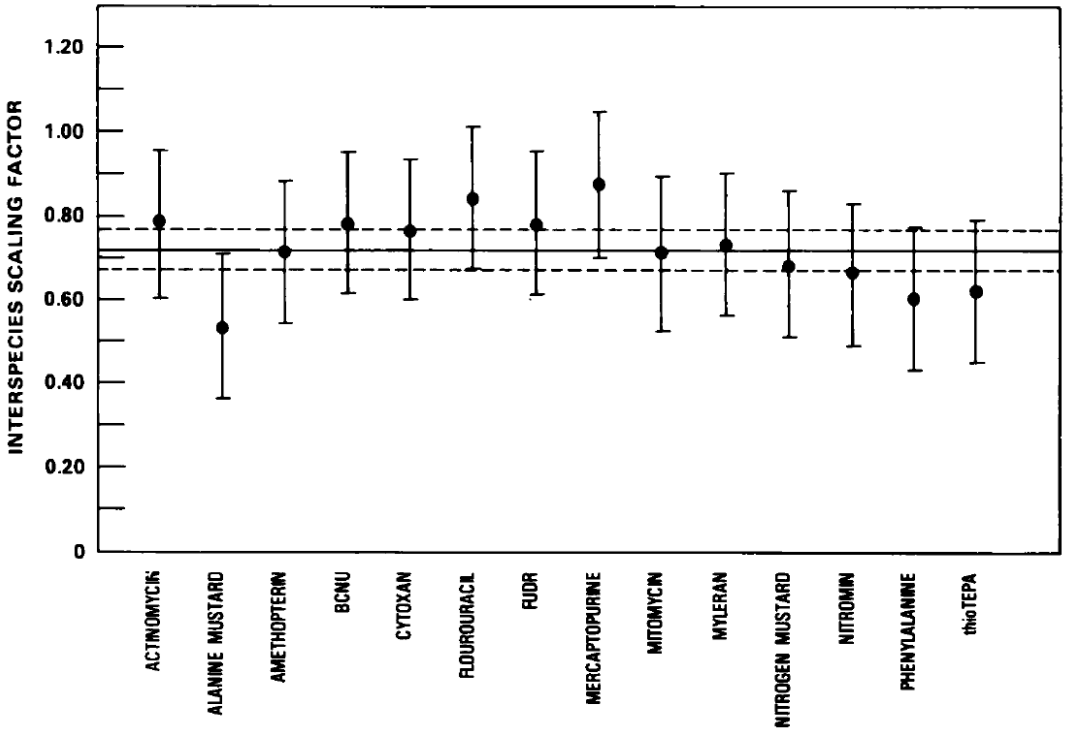


Figure 2.3 Chemicals assessed in Travis and White (1988). Each dot represents the slope of the line of inter-specific body mass vs. dose for each chemical. The solid horizontal line represents the average slope of 0.73 (not significantly different than 0.75). Reproduced from Travis and White (1988).

Unlike the case of mammals, only one study has assessed the use of scaling factors for avian toxicity (Mineau *et al.* 1996). The "Mineau scaling factor" (EPA 2005i) of 1.15 has been applied by US EPA in their T-REX model and by Sample *et al.* (1996) for the assessment of interspecies avian acute toxicity. Mineau *et al.* cautioned against applying acute scaling factors to chronic toxicity, however, the EPA and Environment Canada appear to use this scaling factor with both acute and chronic data (EPA 2005; Canada Gazette, 2008). However, based on the Kleiber Power Law and given that bird species with a wide range of body weights were used to derive it (along with various mammals), there is no apparent reason to exclude birds from body mass scaling with an exponent of 0.75.

Based on the evidence supporting an allometric scaling relationship of body mass raised to 0.75, toxicity reference values (TRVs) for wildlife receptors can be estimated based on laboratory data generated with species of different sizes based on the following equation:

$$TRV_{\text{receptor species}} = TRV_{\text{test species}} (BW_{\text{test species}} / BW_{\text{receptor species}})^{b}$$

In this case, b is equal to 1.0 minus the metabolic scaling factor of 0.75. In other words, b=0.25. Indeed, this is the approach used by Environment Canada and Health Canada for the derivation of mammalian TRVs (or critical toxicity values; CTVs) for chemical substances assessed under the Chemicals Management Plan (www.chemicalsubstanceschimiques.gc.ca), but like the EPA, they do not apply this scaling factor for avian species. A recent draft screening risk assessment for the chemical substance bisphenol-A can be used to illustrate the use of this allometric scaling approach (Canada Gazette, 2008).

Given this equation it can be expected that larger animals will have smaller TRVs than smaller animals. For example, a LOAEL commonly used in ERA for zinc is 320 mg/kg-bw/day based on reproductive endpoints resulting from dietary exposure studies with rats (approximate mass 0.35 kg) (Sample *et al.,* 1996). By using the equation above, a TRV for meadow voles, fox and deer can be calculated as roughly 544, 169, 89 mg/kg-bw/day, respectively. Using this method, larger animals have lower TRVs than that of the test species (*i.e.,* rat). The reason that smaller animals (birds and mammals) can deal with larger exposure doses is because the biological half-life of contaminants is shorter in smaller animals than larger animals because their higher metabolism per kilogram means that they are able to transform, metabolize, and remove contaminants from their bodies more rapidly than larger animals (Chappell 1992; Sample *et al.* 1996). In other words, smaller animals are able to tolerate higher doses better than larger animals, with the caveat that smaller species may be less able to tolerate toxicity if the toxic effect of the compound is produced by a metabolite (Chappell 1992; Sample *et al.* 1996). As can be seen in the case of the meadow vole (approximate mass 0.042 kg), the allometricallly scaled TRV is greater than that of the rat. If the allometric concept holds true, it should hold true in any direction, but to maintain conservatism in ERA, we suggest TRVs should not be scaled upward, and the TRV derived from the test species should be used for receptors smaller than that of the test species.

The application of allometric scaling to toxicity is not without its setbacks; allometric scaling is most useful for like chemicals with similar dispositions (Davidson *et al.* 1986; Chappell 1992) and may not be applicable to a wide range of chemicals; the use of allometric scaling may not be appropriate for chemicals that undergo biotransformation as the toxicity of metabolites may be different than parent compounds (Travis and White 1988); and almost all data linking allometric scaling to toxicity have done so using acute or sub-chronic data so the relationship to chronic data, though supported through concept, is lacking empirical support. These caveats need to be assessed prior to applying allometric scaling of dose in ERA.

2.4 UNCERTAINTY FACTORS FOR INDIVIDUAL RISK

In ERA, the focus of the assessment is normally to provide protection for wildlife at the population level and TRVs based on LOAELs are used in the calculation of risk. This is in contrast to human toxicology and human health risk assessment, where protection of individuals is of paramount concern. An exception to this occurs in ERA when federally or provincially designated species at risk or conservation concern are evaluated. To ensure that these species are afforded an appropriate level of protection in ERA, Jacques Whitford uses TRVs that are based on NOAELs; if NOAELs are not available and LOAELs are used in the calculation of risk, then the acceptable threshold for toxicity is modified downward from 1 to 0.33 (reduced by a half order of magnitude) in-line with guidance from Ohio EPA (2008).

3.0 ORAL REFERENCE DOSES

The following sections present the reference doses (mg/kg-bw/day) selected for use in the ERA model for each COPC for mammalian and avian receptors. For each reference dose, the supporting studies are discussed, a level of confidence in the reference dose is assigned, and where appropriate the promulgating agency is identified. Where appropriate, uncertainty factors were applied to oral reference doses for the derivation of toxicity reference values as outlined in the ERA. Table 1 presents the final TRVs for wildlife species, with any uncertainty factors and body-scaling applied, as necessary.

3.1 CONFIDENCE IN REFERENCE DOSE VALUES

Depending upon the number of studies reviewed, the test species evaluated, and the toxicity endpoints reported, the scientific confidence in each reference dose value identified is ranked as being high, medium or low. High confidence exists where the reference dose is based upon a large number of scientific studies, with several test species, and with chronic toxicity data that represent growth, reproductive, or survival endpoints. In general, these reference dose values would include datasets such as those reported by the U.S. EPA in the Eco-SSL series of reports. High confidence is also assigned to reference dose values that have gained general acceptance in the risk assessment community over a number of years, such as those presented in the Oak Ridge National Laboratory (ORNL) series of reports. A medium level of confidence would generally be assigned to reference dose values that are based on three or more studies, although the reported exposures may not have all been of chronic duration, used exposure routes other than food ingestion (e.g., drinking water exposure, which is less likely than exposure via food at the site), or may not necessarily have represented optimal toxicity endpoints. A low level of confidence would be assigned to reference dose values that are based on less than three studies, or where, due to studies being of short duration or reporting relatively insensitive toxicological endpoints, a large overall UF is applied to the source studies in order to estimate the reference dose value.

3.2 REFERENCE DOSE VALUES FOR INORGANICS

3.2.1 Antimony

The U.S. EPA has conducted an in-depth review of the toxicological literature pertaining to the effects of antimony on birds and mammals (U.S. EPA, 2005a). Eleven studies addressing effects on mammals

were identified and considered acceptable for consideration towards the selection of a TRV for the antimony Eco-SSL. An avian TRV was not selected by the U.S. EPA (2005a) for deriving and avian Eco-SSL, since no acceptable studies were identified addressing the effects of antimony to birds.

Mammals

The U.S. EPA (2005a) uses the highest NOAEL value which is lower than the lowest bounded LOAEL of studies reporting values based on reproduction, growth, or survival endpoints as the Eco-SSL TRV. Only studies presenting both NOAEL and LOAEL values are considered for this selection. The ecological model is based on LOAEL values when toxicological data permits. Therefore, the lowest bounded LOAEL from these studies was selected as the oral reference dose for TRV determination in the ecological model. This value (0.59 mg/kg-bw/day) was derived from a study by Rossi *et al.* (1987, in U.S. EPA, 2005a), in which female gestating rats were exposed to antimony (as antimony trichloride) in drinking water at concentrations of 0, 1, or 10 mg/L for 31 days. Adverse effects on progeny (reduced body weight) were observed at the 10 mg/L concentration only. Therefore, the 10 mg/L concentration is the LOAEL, and is considered a chronic value since exposure was administered during a critical lifestage (*i.e.*, reproduction). The chronic LOAEL is converted to a daily dose of 0.59 mg/kg-bw/day using water consumption and body weight reported by the study, and is selected for use as the reference dose for mammalian species in the ecological model. The collection of toxicological literature from which this LOAEL was selected included studies using rats and mice, and primarily acute and subchronic exposures, although some studies (including Rossi *et al.* 1987) included a reproductive period and are therefore considered chronic because exposure was received during a critical lifestage. This reference dose is assigned a medium level of confidence.

Birds

No acceptable avian toxicological studies were identified for use in the derivation of a reference dose, so no TRV could be established for this COPC.

3.2.2 Arsenic

The U.S. EPA has conducted an in-depth review of the toxicological literature pertaining to the effects of arsenic on birds and mammals (U.S. EPA, 2005b). Fifty-eight studies addressing effects on mammals, and four studies addressing effects on birds were identified and considered in the selection of a TRV for arsenic Eco-SSLs.

Mammals

The U.S. EPA (2005b) uses the highest NOAEL value which is lower than the lowest bounded LOAEL of studies reporting values based on reproduction, growth, or survival endpoints as the Eco-SSL TRV. Only studies presenting both NOAEL and LOAEL values are considered for this selection. The ecological model is based on LOAEL values when toxicological data permits. Therefore, the lowest bounded LOAEL from these studies was selected as the reference value for TRV determination in the ecological model. This value (1.66 mg/kg-bw/day) was derived from a study by Neiger and Osweiler (1989, in U.S. EPA, 2005b), in which dogs were exposed to arsenic (as sodium arsenite; 57.7% arsenic) in the diet at doses of 0, 0.88, 1.80, or 2.88 mg/kg-bw/day for 8 weeks. Adverse effects on growth (reduced body weight) were first observed at the 2.88 mg/kg-bw/day dose (as sodium arsenite). The value used in the ecological model is 1.66 mg/kg-bw/day as arsenic (corrected based on %arsenic in administered dose of sodium arsenite). The collection of toxicological literature from which this LOAEL was selected contained studies using a variety of test animals (*e.g.,* rat, mouse, rabbit, dog)

and exposure durations (acute, subchronic, and chronic), therefore this LOAEL value can be used for a variety of mammal species without additional uncertainty factors being applied, and there is a high level of confidence in this reference dose.

Birds

The U.S. EPA review and evaluation of primary toxicological literature pertaining to exposure of arsenic to birds resulted in only four studies which observed growth or reproduction endpoints (U.S. EPA, 2005b). The EPA uses the lowest NOAEL from these studies as the TRV for determining an Eco-SSL (NOAEL = 2.24 mg/kg-bw/day). The U.S. EPA assigned a low confidence rating to the study from which this value was extracted. The literature review for Eco-SSL TRVs is typically very thorough and the results generally provide a good representation of current toxicological knowledge for the chemical being reviewed.

Sample *et al.* (1996) provide results of two toxicological studies on the effects of arsenic on birds. Both studies were conducted by the U.S. Fish and Wildlife Service (U.S. FWS, 1964 and 1969). These studies were not included in the Eco-SSL document, presumably because the U.S. EPA only reviews studies from primary sources (*i.e.,* not U.S. FWS). The 1969 study by U.S. FWS, reported in Sample *et al.* (1996) provided arsenic to Brown-headed Cowbirds as copper acetoarsenite. It is not clear whether the presence of copper may have affected the results of this study. A chronic LOAEL from the U.S. FWS (1964) study is used as the reference value for TRV determination. In that study, arsenic was provided to Mallard ducks in feed as sodium arsenite for 128 days (chronic duration) at concentrations of 100, 250, 500, and 1,000 mg/kg. Mortality (12%) was observed in Mallards at the 250 mg/kg concentration (no mortality was observed at 100 mg/kg). Therefore, a chronic LOAEL is established at 250 mg/kg. This concentration corresponds to an arsenic daily dose of 12.84 mg/kg-bw/day, with a corresponding NOAEL of 5.14 mg/kg-bw/day. The LOAEL value from U.S. FWS (1964) and Sample *et al.* (1996) is retained as the reference dose, with a high level of confidence.

3.2.3 Barium

The U.S. EPA has conducted an in-depth review of the toxicological literature pertaining to the effects of barium on birds and mammals (U.S. EPA, 2005c). Ten studies addressing effects on mammals, and a single avian toxicity study were identified and considered in the selection of a TRV for barium Eco-SSLs.

Mammals

The ecological model uses the same TRV as the U.S. EPA uses for deriving an Eco-SSL that is protective of barium effects on mammals. Using the ten studies addressing effects on mammals, the U.S. EPA (2005c) applies the geometric mean of NOAELs from those studies which monitored growth or reproductive endpoints as the Eco-SSL TRV. This value (51.8 mg/kg-bw/day) is lower than any "bounded LOAEL" for studies monitoring growth, reproductive, or survival endpoints. The collection of toxicological literature from which this geometric mean was calculated contained studies using various exposure durations (*i.e.,* subchronic and chronic), and was therefore considered conservatively representative of chronic exposure without the application of uncertainty factors. Either rats or mice were the test subjects in each toxicological study considered for this geometric mean value. Therefore, body weight-based scaling factors are not applied since this dose is considered representative of small mammals, and conservative for larger mammals. This reference dose is assigned a high level of confidence.

Birds

The U.S. EPA review and evaluation of primary toxicological literature pertaining to exposure of barium to birds resulted in only one study considered appropriate for using towards the derivation of an avian Eco-SSL (U.S. EPA, 2005c). An avian TRV was not established, since the U.S.EPA requires at least three studies from two species as a minimum for deriving Eco-SSL values. The literature review for Eco-SSL TRVs is typically very thorough and the results generally provide a good representation of current toxicological knowledge for the chemical being reviewed. Sample *et al.* (1996) provides the results of one toxicological study addressing the effects of barium on birds. This study, by Johnson *et al.* (1960) is the same study identified by the U.S. EPA in the Eco-SSL avian toxicity review for barium (U.S. EPA, 2005c). Johnson *et al.* (1960) provided barium to one-day old chicks as barium hydroxide. Chicks were provided barium in the diet for four weeks (subchronic duration) at eight dose levels (doses successively doubled from 250 to 32,000ppm). Based on observations of mortality (5%), 4000 ppm was considered the subchronic LOAEL for this study. Using an estimated food consumption for two-week old chicks (U.S. EPA 1988; in Sample *et al.* 1996), and the mean body weight for chicks in the study (at day 14), this dose corresponds to a subchronic LOAEL of 416.53 mg/kg-bw/day. This value is used in the ecological model as the reference dose for TRV determination. Due to the paucity of toxicological information pertaining to the effects of barium to birds, a comparison of the interspecies sensitivity differences, or additional endpoints was not possible. Consequently, this reference dose is assigned a low level of confidence.

3.2.4 Beryllium

Mammals

The U.S. EPA has conducted an in-depth review of toxicological literature addressing the effects of beryllium exposure to mammals in an effort to derive Eco-SSL values (U.S. EPA, 2005d). Four studies were deemed acceptable for TRV determination by the U.S. EPA. The U.S. EPA uses the lowest NOAEL of studies observing growth or survival endpoints (no reproductive studies were considered acceptable) as the TRV for deriving an Eco-SSL. This value is from a study by Schroeder and Mitchener (1975; also reported by Sample *et al.* 1996) where beryllium (as beryllium sulfate) was provided to rats via drinking water for the lifetime of the rats at 0, or 5 mg/kg. Adverse effects were not observed on either survival or growth endpoints (only a transient decrease in male growth was observed). Based on the body weight reported in the study and the estimated water ingestion, this concentration corresponds to a chronic daily dose (NOAEL) of 0.532 mg/kg-bw/day, and is used in the ecological model as the reference dose for TRV determination with a high level of confidence.

Birds

No acceptable avian toxicological studies were identified for use in the derivation of a reference dose, so no TRV could be established for this COPC.

3.2.5 Boron

Mammals

The boron TRV selected for this ERA model for mammal species is based on the chronic NOAEL determined from studies performed by Weir and Fisher (1972), cited in Sample *et al*. (1996). The study was based on oral exposure of rats to boric acid or borax in food. The study endpoint was reproductive; rats exposed to doses of 1,170 ppm B as boric acid or borax became sterile, while no adverse effects were observed at doses of 117 or 350 ppm. Because the study duration extended over

3 generations and incorporated all life stages, the 1,170 ppm dose (93.6 mg/kg/day) was considered to be a chronic LOAEL, while 350 ppm (28 mg/kg/day) was considered a chronic NOAEL. The NOAEL of 28 mg/kg/day was selected for use as the daily dose for mammal species for this model.

Birds

The boron TRV selected for this ERA model for bird species is based on the chronic LOAEL determined from studies performed by Smith and Anders (1989), cited in Sample et al. (1996). The study was based on oral exposure of mallard ducks to boric acid in their diet. The study endpoint was reproductive. While ducks exposed to doses of 1000 ppm boric acid exhibited reduced egg fertility and duckling growth, increased duckling mortality and embryo mortality, no adverse reproductive effects were observed at other dose levels. Because the study considered exposure throughout reproduction, the 1000 ppm B dose (100 mg/kg/day) was considered to be a chronic LOAEL, while 288 ppm B (28.8 mg/kg/day) was considered a chronic NOAEL. The LOAEL of 100 mg/kg/day was selected for use as the daily dose for bird species for this model.

3.2.6 Cadmium

The U.S. EPA has conducted an in-depth review of the toxicological literature pertaining to the effects of cadmium on birds and mammals (U.S. EPA, 2005e). Of 1,953 studies examined, 145 studies addressed effects on mammals, and 35 addressed effects on birds, and were considered in the selection of a TRV for cadmium Eco-SSLs

Mammals

The ecological model is based on LOAEL values when toxicological data permits. The U.S. EPA presents a lowest bounded-LOAEL of 0.91 mg/kg/day based on the effects of cadmium chloride administered to juvenile sheep via feed for 163 days at dose rates of 0, 10.8, 29.4, 59.7, 111.12 mg/animal/day (Doyle, 1974). The U.S. EPA assigned the study from which this value was derived a total suitability score of 80 out of a possible 100. Adverse effects on growth (*i.e.,* body weight) were first observed at the 59.7 mg/animal/day dose rate, corresponding to 0.909 mg/kg-bw/day after accounting for sheep mass of 65.66 kg. The collection of toxicological literature from which this LOAEL was selected contained studies using several types of test animals (*e.g.,* mouse, rat, dog, sheep), and exposure durations (*i.e.,* acute, subchronic, chronic), therefore this LOAEL value has a high level of confidence and can be used for a variety of mammal species without additional uncertainty factors or allometric scaling being applied.

Birds

The U.S. EPA uses the geometric mean of NOAEL values from studies that monitored growth and reproductive endpoints. This value (1.47 mg/kg-bw/day) is lower than any of the reported LOAELs for these endpoints and mortality, and is used as the basis for TRV determination in the ecological model. The test animals in the studies used towards calculation of the geometric mean included chicken, mallard, quail, and wood duck, and the NOAEL carries a high level of confidence.

3.2.7 Chromium

The U.S. EPA (U.S. EPA, 2008) has conducted an in-depth review of the toxicological literature pertaining to the effects of trivalent Chromium (Cr^{3+}) on birds and mammals (U.S. EPA, 2008). Twenty studies addressing effects on mammalian species, and 13 addressing avian species were identified and considered in the selection of a TRV for chromium Eco-SSLs.

Jacques Whitford assumes that unless otherwise specified, the trivalent chromium and total chromium are equivalent, and that hexavalent chromium is not present unless specifically identified in analytical reports.

3.2.8 Trivalent (or Total) Chromium

Mammals

The U.S. EPA uses the geometric mean of NOAEL values from studies that monitored growth and reproductive endpoints. This value of 2.4 mg/kg-bw/day is lower than any of the reported LOAELs for these endpoints and mortality, and is used as the basis for TRV determination in the ecological model. The test animals in the studies used towards calculation of the geometric mean included mouse, rat, pig, and cattle. The reference dose carries a high level of confidence.

Birds

The U.S. EPA (2008) uses the geometric mean of NOAEL values from studies that monitored growth and reproductive endpoints. This value of 2.66 mg/kg-bw/day is lower than any of the reported LOAELs for these endpoints and mortality, and is used as the basis for TRV determination in the ecological model. The test animals in the studies used towards calculation of the geometric mean included Chicken, Turkey and Black Duck. The reference dose carries a high level of confidence.

3.2.9 Hexavalent Chromium

Mammals

The U.S. EPA uses the geometric mean of NOAEL values to derive a TRV. This value of 9.24 mg/kg-bw/day is lower than the lowest bounded LOAEL for studies assessing reproduction, growth and survival endpoints. The test animals in the studies used towards calculation of the geometric mean included mouse and rat. This reference dose carries a high level of confidence.

Birds

An avian TRV for hexavalent chromium was not derived by U.S. EPA (2008) because there were not enough study results that met the minimum requirements. Four studies which address the effects of hexavalent chromium on avian species were available but only one study reported adverse effects of hexavalent chromium on avian species (Asmatullah *et al.* 1999). Chickens were provided chromium as potassium dichromate in feed for 32 weeks at concentrations of 0, 250, and 500 mg/kg. Adverse reproductive effects were observed at the 250 mg/kg concentration. Given the reported body weight and food consumption of the test animals, this concentration corresponds to a daily dose (LOAEL) of 4.02 mg/kg-bw/day, and is used as the basis for TRV determination in the ecological model, with a medium level of confidence.

3.2.10 Cobalt

The U.S. EPA has conducted an in-depth review of the toxicological literature pertaining to the effects of cobalt on birds and mammals (U.S. EPA, 2005f). Twenty two studies addressing effects on mammals, and eleven avian toxicity studies were identified and considered in the selection of a TRV for cobalt Eco-SSLs.

Mammals

The ecological model uses the same TRV as the U.S. EPA uses for deriving an Eco-SSL that is protective of cobalt effects on mammals. Using the ten studies addressing effects on mammals, the U.S. EPA (2005f) applies the geometric mean of NOAELs from those studies which monitored growth or reproductive endpoints as the Eco-SSL TRV. This value (7.33 mg/kg-bw/day) is lower than any "bounded LOAEL" for studies monitoring growth, reproductive, or survival endpoints. The collection of toxicological literature from which this geometric mean was calculated contained studies using various exposure durations (*i.e.,* subchronic and chronic), and was therefore considered conservatively representative of chronic exposure without the application of uncertainty factors. The test subject used in the toxicological studies considered for this geometric mean value ranged in body size from 0.04 kg (mouse) to 99 kg (cow). Therefore, body weight-based scaling factors are not applied as this dose is considered applicable to the majority of mammalian species body weights. This reference dose is assigned a high level of confidence.

Birds

The ecological model uses the same TRV as the U.S. EPA uses for deriving an Eco-SSL that is protective of cobalt effects on birds. Using the eleven studies addressing effects on birds, the U.S. EPA (2005f) applies the geometric mean of NOAELs from those studies which monitored growth endpoints as the Eco-SSL TRV (no studies observing reproductive endpoints were identified). This value (7.61 mg/kg-bw/day) is lower than any "bounded LOAEL" for studies monitoring growth, or survival endpoints. The collection of toxicological literature from which this geometric mean was calculated contained only studies of sub-chronic exposure duration. Due to the lack of chronic or critical lifestage exposure, it was necessary to apply an uncertainty factor of 3 to the geometric mean LOAEL. Chickens or ducks, ranging in body weights from 0.11 kg to 1.63 kg, were the test subjects in each toxicological study considered for this geometric mean value. Therefore, body weight-based scaling factors are not applied since this dose is considered applicable to the body weights of the majority of avian species. The final reference is therefore 2.54 mg/kg-bw/day and is assigned a medium level of confidence.

3.2.11 Lead

The U.S. EPA (2005g) has conducted an in-depth review of the toxicological literature pertaining to the effects of lead on birds and mammals (U.S. EPA, 2005g). Of 2,429 studies examined, 219 studies addressed effects on mammals, and 54 addressed effects on birds, and were considered in the selection of a TRV for lead.

Mammals

The U.S. EPA (2005g) uses the highest bounded NOAEL value (4.7 mg/kg-bw/day) lower than the lowest bounded LOAEL of studies reporting values based on reproduction, growth, or survival endpoints as the Eco-SSL TRV. Only studies presenting both a NOAEL and LOAEL value (*i.e.* bounded) are considered for this selection. The Eco-SSL value of 4.7 mg/kg-bw/day is the value chosen for use in the ecological model as the reference dose, based on a study by Kimmel *et al.,* 1980. In the study, gestating rats were exposed to lead acetate in drinking water at doses of 0, 0.92, 4.7, 8.9 mg/kg-bw/day for 7 weeks. The effect on progeny weight was the endpoint. The collection of toxicological literature from which this NOAEL was selected contained studies using several types of test animals (*e.g.,* mouse, rat, rabbit, cattle), and exposure durations (*i.e.,* acute, subchronic, chronic), therefore this NOAEL value can be used for a variety of mammal species without additional uncertainty factors or allometric scaling being applied. This reference dose carries a high level of confidence.

Birds

The geometric mean NOAEL of 10.9 mg/kg/d for growth and reproduction was selected as the TRV for the ERA. Although the highest bounded NOAEL lower than the lowest bounded LOAEL (1.63 mg/kg-bw/day, U.S. EPA 2005g) is more conservative, it comes from a study were lead acetate (a highly bioavailable form of lead) was administered in feed, whereas the geometric mean is based on studies using various chemical forms of lead (*e.g.*, lead acetate, lead dichloride, lead nitrate). Moreover, the collection of toxicological literature from which this NOAEL was selected contained studies using several types of test animals (*e.g.,* quail, chicken, mallard, kestrel, turtle dove), and exposure durations (*i.e.,* acute, subchronic, chronic); therefore this NOAEL value can be used for a variety of bird species without additional uncertainty factors being applied. This reference dose is assigned a high level of confidence.

3.2.12 Mercury

Unless otherwise specified and supported by laboratory data, mercury in soils and sediments will be treated as inorganic mercury. Mercury in fish and other biota will be treated as organic or methyl mercury.

3.2.13 Inorganic Mercury

Mammals

The inorganic mercury reference dose used in the ecological model is based on the chronic NOAEL determined from studies performed by Aulerich *et al.* (1974), cited in Sample *et al.* (1996). The study was based on oral exposure of mink to mercuric chloride in food. The study endpoint was considered reproduction. Mink were exposed to mercury at a concentration of 10 mg/kg mercuric chloride in diet over a period of 6 months, during which they reproduced. Although kit weight was somewhat reduced, fertility and kit survival were not reduced. Because the study considered exposure through a reproductive period, the 10 mg/kg mercuric chloride dose (1.01 mg/kg-bw/day) was considered to be a chronic NOAEL. This reference dose is assigned a high level of confidence.

Birds

The mercury reference dose selected for this ERA model for bird species is based on the chronic LOAEL determined from studies performed Hill and Schaffner (1976), cited in Sample *et al.* (1996). The study was based on oral exposure of Japanese quail to mercuric chloride in feed. The study endpoint was considered reproduction. Quail were exposed to mercuric chloride at concentrations of 2, 4, 8, 16, and 32 mg Hg/kg diet over a period of 1 year. Although egg production increased with increasing Hg dose, fertility and hatchability were observed to decrease at 8 mg/kg diet. Based on the food ingestion rate and body weight of the Japanese quail the reference dose rate (based on the LOAEL) for avian species is estimated to be 0.9 mg/kg-bw/day, and the corresponding NOAEL is 0.45 mg/kg-bw/day. This reference dose is assigned a high level of confidence.

3.2.14 Methyl Mercury

Mammals

The methyl mercury reference dose selected for this ERA model for mammal species is based on the chronic LOAEL determined from a study performed by Verschuuren *et al.* (1976), cited in Sample *et al.* (1996). The study was based on oral exposure of rats to methyl mercury chloride in food. The study

endpoint was considered reproduction. Rats were exposed to mercury at concentrations of 0.1, 0.5 and 2.5 mg/kg methyl mercury chloride throughout 3 generations. Rats exposed to 2.5 mg/kg methyl mercury chloride exhibited reduced pup viability, however no adverse effects were observed at lower concentrations. Therefore the 2.5 and the 0.5 mg/kg methyl mercury chloride concentrations are considered to be a chronic LOAEL and NOAEL, respectively. Based on food ingestion and body weight, the 0.5 mg/kg LOAEL corresponds to a daily dose 0.16 mg/kg-bw/day while the corresponding NOAEL is 0.032 mg/kg-bw/day. The LOAEL is used in the ecological model as the reference dose for TRV determination representing the effects of methyl mercury on mammalian species. This reference dose is assigned a high level of confidence.

Birds

The methyl mercury reference dose selected for this ERA model for bird species is based on the chronic LOAEL determined from studies performed by Heinz (1979), cited in Sample *et al.* (1996). The study was based on oral exposure of Mallards to methyl mercuric dicyadiamide in diet. The study endpoint was considered reproduction. Mallards were exposed to methyl mercuric dicyadiamide in feed at 0.5 mg/kg total Hg over three generations. Significant effects, including reduced egg and duckling production were observed, and the 0.5 mg/kg concentration was considered to be a chronic LOAEL. Using body weight and food ingestion rate, the 0.5 mg/kg concentration was considered to correspond to a daily dose rate of 0.064 mg/kg-bw/day (Sample *et al.* 1996). This reference dose is assigned a high level of confidence.

3.2.15 Nickel

The U.S. EPA has recently conducted an in-depth review of the toxicological literature pertaining to the effects of nickel on birds and mammals (U.S. EPA, 2007a). Of 1,169 studies examined, 52 studies addressed effects on mammals, and 11 addressed effects on birds, and were considered in the selection of a TRV for nickel.

Mammals

The LOAEL selected as the reference value for TRV determination in the ecological model was derived from a study by Kakela *et al.,* (1999), as presented in the U.S. EPA nickel Eco-SSL database, and is the lowest bounded LOAEL for effects on growth and reproduction.

In the study, mice were exposed to nickel (as nickel chloride hexahydrate) via oral gavage at dose rates of 0, 10, 30, and 100 mg/L for 28 days. Adverse effects on reproduction (reduction in male and female fertility) were first observed at 30 mg/L. The equivalent dose, adjusted for ingestion rate and body mass, is 3.31 mg/kg-bw/day, and is used as the reference dose for TRV determination in the ecological model. The collection of toxicological literature from which this LOAEL was selected contained studies using several types of test animals (*e.g.,* mouse, rat, meadow vole, cattle) and exposure durations (*i.e.,* acute, subchronic, chronic), therefore this LOAEL value can be used for a variety of mammal species without additional uncertainty factors or allometric scaling being applied. This reference dose is assigned a high level of confidence.

Birds

The ecological model uses the same TRV as the U.S. EPA for deriving an Eco-SSL that is protective of nickel effects on birds. Using the 11 studies addressing effects on birds, the U.S. EPA (2007a) applies the geometric mean of NOAELs from those studies which monitored growth or reproductive endpoints as the Eco-SSL TRV. This value (6.71 mg/kg-bw/day) is lower than any LOAEL for studies monitoring

growth, reproductive, or survival endpoints in the U.S. EPA database. The collection of toxicological literature from which this geometric mean was calculated contained studies using Mallards and Chickens as test animals and primarily acute or subchronic exposures, but also included two studies of reproductive endpoints that had higher NOAEL and/or LOAEL values than the geometric mean NOAEL value. Therefore the value of 6.71 mg/kg-bw/day will be treated as equivalent to a chronic NOAEL. This reference dose is assigned a high level of confidence.

3.2.16 Selenium

The U.S. EPA has recently conducted an in-depth review of the toxicological literature pertaining to the effects of selenium on birds and mammals (U.S. EPA, 2007b). Of 1,734 studies examined, 132 studies addressed effects on mammals, and 69 addressed effects on birds, and were considered in the selection of a TRV for selenium.

Mammals

The selenium reference dose selected for this ERA model for mammal species is based on the chronic LOAEL determined from studies performed by Liu *et al.* (1994), cited in the U.S. EPA Eco-SSL. The study was based on oral exposure of juvenile rats to sodium selenite in feed. The study endpoint was considered growth. Rats were exposed to selenium at concentrations of 0, 0.51, 2.0, and 4.1 mg/kg over a period of 2 weeks *ad libitum*. While rats exposed to selenium at 2.0 mg/kg showed no adverse effects on growth, significant reduction in growth rates was noted in rats exposed to a concentration of 4.1 mg/kg. Therefore the 4.1 mg/kg and 2.0 mg/kg doses are considered to represent a chronic LOAEL and NOAEL, respectively. The chronic LOAEL of 4.1 mg/L was converted to a dose rate of 0.304 mg/kg-bw/day based on the body weight and food ingestion rate, and is selected for use as the reference dose for mammalian species in the ecological model. A total uncertainty factor of 3 was applied to this LOAEL, based on the sub-chronic exposure duration. The collection of toxicological literature from which this LOAEL was selected contained studies using several types of test animals (*e.g.,* mouse, rat, rabbit, cattle, pig) and exposure durations (*i.e.,* acute, subchronic, chronic), therefore this LOAEL value can be used for a variety of mammal species without allometric scaling being applied. This reference dose is assigned a high level of confidence.

Birds

The selenium reference dose selected for this ERA model for avian species is based on the chronic LOAEL determined from studies performed by Heinz *et al.* (1987), cited in Sample *et al.* (1996), and the U.S. EPA selenium Eco-SSL (U.S. EPA, 2007b). The study was based on oral exposure of mallards to sodium selenite in diet. The study endpoint was considered reproduction. Mallards were exposed to sodium selenite at 1, 5, 10, 25, and 100 mg/kg selenium in diet over a period of 78 days. As referenced in Sample *et al.,* (1996), mallards exposed to 100 mg/kg selenium showed reduced adult survival; exposure to 10 and 25 mg/kg selenium resulted in a significantly increased frequency of lethally deformed embryos; exposure to 5 mg/kg selenium resulted in no significant adverse effects. Therefore the 10 mg/kg and the 5 mg/kg diets are considered to represent a chronic LOAEL and NOAEL, respectively. The chronic LOAEL of 10 mg/kg in diet was converted to a dose rate of 1.0 mg/kg-bw/day based on the body weight and food ingestion rate. It should be noted that the U.S. EPA Eco-SSL considered the reproduction LOAEL from this study to be 25 mg selenium/kg feed, which is a less conservative value than that chosen for use in this assessment. Therefore, the more conservative value (1 mg/kg-bw/day; 10 mg/kg) based on Sample *et al.,* (1996) was chosen for use in the ecological model. This reference dose is assigned a high level of confidence.

3.2.17 Silver

Mammals

The U.S. EPA has conducted an in-depth review of the toxicological literature pertaining to the effects of silver on mammals (U.S. EPA, 2006). Thirteen studies addressing effects on mammals were identified and considered in the selection of a TRV for silver Eco-SSLs.

Of the thirteen studies identified by the U.S. EPA for mammalian studies involving silver, only one, performed by Van Vleet*.,* (1976) was selected for use in deriving a mammal TRV for this ERA. This TRV, a LOAEL of 60.2 mg/kg-day, (the lowest LOAEL from growth studies), is based on growth effects (change in body weight) of juvenile pigs (sex not reported) sub-chronically exposed to silver via diet. A total uncertainty factor of 3 was applied to this LOAEL, based on the sub-chronic exposure duration. This reference dose is assigned a low level of confidence.

Birds

The U.S. EPA has conducted an in-depth review of the toxicological literature pertaining to the effects of silver on birds (U.S. EPA, 2006). Seven studies addressing effects on birds were identified and considered in the selection of a TRV for silver Eco-SSLs.

Of the seven studies identified by the U.S. EPA for avian studies involving silver, only one, performed by Jensen *et al.,* (1974) was selected for use in deriving an avian TRV for this ERA. This TRV, a LOAEL of 20.2 mg/kg-day, (the lowest LOAEL from growth studies), is based on growth effects (change in body weight) of juvenile turkeys (male and female) sub-chronically exposed to silver via diet. A total uncertainty factor of 3 was applied to this LOAEL, based on the sub-chronic exposure duration. This reference dose is assigned a low level of confidence.

3.2.18 Thallium

Mammals

The thallium TRV selected for this ERA model for mammal species is based on the sub-chronic LOAEL determined from studies performed by Formigli *et al*. (1986), cited in Sample et al. (1996). The study was based on oral exposure of rats to thallium sulphate in water. The study endpoint was considered reproduction (male testicular function). Rats were exposed to thallium sulphate at a dose level of 10 ppm TI over a period of 60 days. Rats exposed to thallium sulphate at a dose level of 10 ppm TI displayed reduced sperm motility. Because the study considered exposures of only 60 days, the 10 ppm dose (0.74 mg/kg/day) is considered a sub-chronic LOAEL and was selected for use as the daily dose for mammal species for this model. An uncertainty factor of 3 was applied for the conversion from a sub-chronic LOAEL to a chronic LOAEL, resulting in a chronic LOAEL of 0.15 mg/kg/day used in this model for mammal species.

Birds

The thallium TRV selected for this ERA model for avian species is based on an acute LD50 (34.6 mg/kg) value cited in Schafer et al. (1972). The study was based on oral exposure of starlings to thallium sulphate administered in propylene glycol via gavage. The study endpoint was mortality. Because the study considered only acute exposures, the 34.6 mg/kg dose is assigned an uncertainty factor of 100 for the conversion from an acute LD50 to a chronic LOAEL, resulting in a chronic LOAEL of 0.346 mg/kg/day used in this model for avian species.

3.2.19 Tin (Inorganic)

Mammals

The reference dose selected for this ERA (44 mg/kg/day) was based on the average NOAEL from two sub-chronic dosing studies (FDA 1972 and Theuer *et al.* 1971, as cited in ATSDR 2005b) where female rats, mice and hamsters were given either stannous chloride, tin fluoride or sodium pentachlorostannite in food (available *ad libitum*) or water (gavage) during gestational days 0-20. A NOAEL of 31 mg/kg/day (stannous chloride) based on absence of reproductive effects (i.e., number of corpora lutea and implantation and resorption sites) in rats, mice and hamsters was identified in the FDA (1972) study. This dose was also identified as the NOAEL for developmental effects based on the lack of significant changes on fetal weight, the number of live or dead fetuses, and the incidence of external and internal malformations. In the Theuer e*t al.* study, exposure of rats of up to approximately 45 mg tin/kg/day (sodium pentachlorostannite) or 56 mg tin/kg/day (tin fluoride) in the diet had no significant effect on the number of resorptions or placental weight or on average fetal weight or the number of live fetuses per litter. Though these studies were sub chronic in nature, they were conducted during a reproductive period and are, therefore, considered chronic because exposure was received during a critical lifestage. This reference dose is assigned a medium level of confidence.

Birds

No acceptable avian toxicological studies were identified for use in the derivation of a reference dose, so no TRV could be established for this COPC.

3.2.20 Vanadium

The U.S. EPA has conducted an in-depth review of the toxicological literature pertaining to the effects of vanadium on birds and mammals (U.S. EPA, 2005h). Of 916 studies examined, 48 studies addressed effects on mammals, and 36 addressed effects on birds and were considered in the selection of a TRV for vanadium.

Mammals

The ecological model is based on LOAEL values when toxicological data permits. Therefore the lowest LOAEL from these studies was selected as the reference dose for TRV determination in the ecological model. This value (5.11 mg/kg-bw/day) was derived from a study by Daniel and Lillie (1938), in which rats were exposed to vanadium (as sodium metavanadate) in the diet at doses of 0, 0.44, 1.03, 5.11, 9.78, and 19.0 mg/kg-bw/day for 10 weeks. Adverse effects on growth (reduced body weight) were first observed at the 5.11 mg/kg-bw/day dose. The collection of toxicological literature from which this LOAEL was selected included studies using rats and mice, and primarily acute and subchronic exposures, although some studies included a reproductive period and are therefore considered chronic because exposure was received during a critical lifestage. This reference dose is assigned a high level of confidence.

Birds

The vanadium reference dose selected for this ERA for bird species is based on the subchronic LOAEL determined from studies performed by Hill (1979), as cited in the U.S. EPA vanadium Eco-SSL (2005h). The U.S. EPA selected the NOAEL from this study as the vanadium TRV for avian species. The study by Hill (1979) was based on oral exposure of 1-day old chicks to sodium metavanadate at concentrations of 0, 3, 6 and 12 mg/kg in food over a period of 5 weeks, *ad libitum*. The study endpoint

considered was body weight changes. Adverse effects on growth were first observed at the 12 mg/kg dose. Based on food consumption and body mass of the studied chicks, the maximum concentration of 12 mg/kg was converted to a subchronic LOAEL of 0.688 mg /kg-bw/day. An uncertainty factor of 3 was applied for the conversion from a sub-chronic LOAEL to a chronic LOAEL. This reference dose is assigned a high level of confidence.

3.2.21 Zinc

The U.S. EPA has conducted an in-depth review of the toxicological literature pertaining to the effects of zinc on birds and mammals (U.S. EPA, 2007c). Of 10,410 studies examined, 99 studies addressed effects on mammals, and 53 addressed effects on birds and were considered in the selection of a TRV for zinc.

Mammals

The zinc reference dose used in the ecological model for mammalian species is based on the chronic LOAEL (lowest bounded) determined from studies undertaken by Miller *et al.,* (1989), cited in the U.S. EPA zinc Eco-SSL (2007c). The study was based on oral exposure of cattle to zinc sulfate. The study endpoint was considered reproduction. Cattle were exposed to zinc sulphate at concentrations of 0, 1,000, and 2,000 mg/kg for 14 weeks during gestation. Progeny of cattle exposed to zinc at 2,000 mg/kg displayed decreased progeny growth rates, while no effects were observed on calves from cattle exposed to 1,000 mg/kg. Therefore the 2,000 mg/kg and the 1,000 mg/kg doses are considered to be a chronic LOAEL and NOAEL, respectively. A chronic LOAEL of 75.9 mg/kg-bw/d was estimated from the 2,000 mg/kg concentration in feed based on the body weight and feeding rate, and was selected for use as the reference dose for mammalian species in the ecological model. Because this chronic LOAEL is the lowest bounded value in the zinc Eco-SSL database which considers many test species and study durations, allometric scaling and additional uncertainty factors do not need to be applied. This reference dose is assigned a high level of confidence.

Birds

The zinc reference dose used in the ecological model for avian species is based on the subchronic LOAEL (lowest bounded) determined from studies performed by Gibson *et al*. (1986), cited in the U.S. EPA zinc Eco-SSL (2007c). The study was based on oral exposure of chickens (hens) to zinc acetate in diet. The study endpoint was considered reproduction. Hens were exposed to zinc sulphate at concentrations of 0, 114.7, 133, 162.1, 107.4, and 135.9 mg/day over a period of 10 weeks. Hens exposed to 133 mg zinc/day in diet exhibited significant effects on reproduction, while exposure to 114.7 mg zinc/day in diet resulted in no adverse effects. Therefore the 133 and 114.7 mg zinc/day concentrations are considered to be a subchronic LOAEL and NOAEL, respectively. A subchronic LOAEL of 66.5 mg/kg-bw/d was estimated from the 133 mg zinc/day concentration in feed based on the body weight and feeding rate, and was selected for use as the reference dose for avian species in the ecological model. Because this subchronic LOAEL is the lowest bounded value in the zinc Eco-SSL database which considers many test species and study durations, and considers reproductive effects, allometric scaling and additional uncertainty factors do not need to be applied. This reference dose is assigned a high level of confidence.

3.3 REFERENCE DOSE FOR POLYCYCLIC AROMATIC HYDROCARBONS

Polycyclic Aromatic Hydrocarbons (PAHs) are a class of compounds consisting of two or more aromatic ring structures. The primary source of PAHs in the environment is from the extraction, refinement and combustion of petroleum or petroleum products (U.S. EPA, 2007d). PAHs typically exist in the environment as complex mixtures. Isolation and characterization of the individual compounds comprising the mixture is often very difficult.

PAHs may be categorized into two groups: low molecular weight compounds consisting of two or three aromatic rings, and high molecular weight compounds consisting of four or more aromatic rings. Health Canada acknowledges this categorization, but assesses health risks for PAH compounds individually (CEPA, 1993). The assessment of potential human health risks, particularly carcinogenic risk from PAH exposure is typically done using a relative potency approach where each compound is assigned a carcinogenic potency relative to benzo(a)pyrene, one of the most toxic PAHs, but this approach is not appropriate for ecological receptors.

In June, 2007 the U.S. EPA released a report: *Ecological Soil Screening Levels for Polycyclic Aromatic Hydrocarbons* (US EPA, 2007d). In this document the U.S. EPA develops toxicological benchmarks for ecological receptors using the low and high molecular weight categorization, rather than following an individual compound approach. The U.S. EPA acknowledges that the optimal approach may be to use a toxic equivalency scheme, but current data limitations preclude this. The assessment of PAHs as two groups of compounds (low and high molecular weight) also has significant benefits. This approach simplifies the inclusion of less well known PAHs, where compound-specific data are not available.

To derive Eco-SSLs for terrestrial wildlife, the U.S. EPA considers toxicological data for constituents of each grouping, but the grouping process leads to a focus on the more toxic compounds, and in addition the overall process is influenced by the availability of data for a relatively small number of compounds (particularly naphthalene in the low molecular weight range and benzo(a)pyrene in the high molecular weight range).

Mammals

Low Molecular Weight PAHs

The U.S. EPA (2007d) identified 76 toxicological results, from studies that met stringent criteria for suitability towards deriving Eco-SSLs. For those results that are based on reproduction or growth endpoints, the geometric mean of NOAEL values was calculated as 170 mg/kg-bw/day. This value was higher than the lowest bounded LOAEL for these endpoints and consequently, the U.S. EPA adopted a value of 65.5 mg/kg-bw/day, the highest bounded NOAEL that is below the lowest bounded LOAEL for growth, reproduction, or survival, as the TRV. Jacques Whitford considers this approach to be too strongly driven by a single study in the context of a large data set and weight of evidence approach. The value selected by the U.S. EPA is also specific for the chemical 1-naphthalenacetic acid, which is not a mainstream PAH. Jacques Whitford therefore considers the U.S. EPA value to be too conservative to be representative of all low molecular weight PAHs and has selected instead the geometric mean of NOAEL values (*i.e.,* 170 mg/kg-bw/day) as the reference dose for the ecological model. The collection of toxicological literature from which this NOAEL was selected included studies using rabbits, mice and rats, and primarily subchronic exposures, although some studies included a reproductive period and are therefore considered chronic because exposure was received during a critical lifestage. Therefore this NOAEL value will be used for a variety of mammal species without additional uncertainty factors being applied. This reference dose value is assigned a high level of confidence.

High Molecular Weight PAHs

The U.S. EPA (2007d) identified 45 toxicological results based on various endpoints that met criteria for high molecular weight PAHs. The geometric mean NOAEL value of results assessing growth and reproduction endpoints was 18 mg/kg-bw/day. This value is higher than the lowest bounded LOAEL for these endpoints and consequently the U.S. EPA adopted a value of 0.615 mg/kg-bw/day based on a study of benzo(a)pyrene, the highest bounded NOAEL that is below the lowest bounded LOAEL for growth, reproduction, or survival. Jacques Whitford considers this value to be too conservative to be representative of all high molecular weight PAHs (*i.e.,* those having four or more rings) and has selected instead the geometric mean of NOAEL values (*i.e.,* 18 mg/kg-bw/day) as the reference dose for the ecological model. The collection of toxicological literature from which this NOAEL was selected included studies using mice and rats, and guinea pigs and primarily subchronic and chronic exposures. Therefore this NOAEL value will be used for a variety of mammal species without additional uncertainty factors being applied. This reference dose value is assigned a high level of confidence.

Birds

The U.S. EPA (2007d) identified nearly 5,500 papers with possible toxicity data for either birds or mammals. Of those meeting Eco-SSL acceptability criteria (46 papers), only two contained data concerning avian species. To derive TRVs, the U.S. EPA requires at least three studies, and a minimum of two species. Therefore, Eco-SSLs were not derived for birds due to data limitations. However, during the Eco-SSL literature review it was observed that for the compounds that had toxicological results for bird species, mammals were always more sensitive (Kapustka, 2004). On the basis of this observation, it has been suggested that mammalian TRVs can be assumed to be protective of avian species also (Kapustka, 2004), and Jacques Whitford will follow this approach for PAHs.

3.4 DIOXINS AND FURANS

Mammals

The reference dose for total dioxins and furans (as 2,3,7,8 TCDD equivalent) for mammalian receptors was obtained from Sample *et al.* (1996), based on a study by Murray *et al.* (1979). Rats were exposed to TCDD in their diet at doses of 0.000001, 0.00001, and 0.0001 mg/kg-bw/day over three generations. Effects on fertility and neonatal survivorship were assessed as chronic endpoints and a chronic LOAEL of 0.00001 mg/kg-bw/day was established. This reference dose is assigned a high level of confidence.

Birds

The reference dose for dioxins and furans (as 2,3,7,8 TCDD equivalents) for avian receptors was obtained from Sample *et al.* (1996), based on a study by Nosek *et al,* (1992). A chronic LOAEL based on reproduction of ring-necked pheasants was estimated to be 0.00014 mg/kg-bw/day. This reference dose value is assigned a medium level of confidence since TCDD was administered to birds via intraperitoneal injection.

3.5 OTHER ORGANIC COMPOUNDS

3.5.1 Carbon Tetrachloride

Mammals

The carbon tetrachloride reference dose used in the ecological model is based on a chronic NOAEL determined from studies performed by Alumot *et al.* (1976), cited in Sample *et al.* (1996). The study was based on oral exposure of rats to carbon tetrachloride in food. The study endpoint was considered reproduction. Rats were exposed to carbon tetrachloride at concentrations of either 80 or 200 ppm in food over a period of two years, during which they reproduced. No significant adverse effects were observed during this study. The upper concentration (200 ppm) is therefore considered a chronic NOAEL, and corresponds to a daily dose of 16 mg/kg-bw/day (based on estimated body weight and food consumption; U.S. EPA 1988, in Sample *et al.* 1996). An ATSDR toxicological profile is available for this compound, and provides a summary of results from studies of oral exposure to mammals (2005a). No adverse effects were reported at less than 16 mg/kg-bw/day for any endpoint (lowest chronic LOAEL is 47 mg/kg-bw/day for systemic effects). The NOAEL from Alumot *et al.* (1976) is used as the reference dose for carbon tetrachloride in the ecological model, and is assigned a medium level of confidence.

Birds

No acceptable avian toxicological studies were identified for use in the derivation of a reference dose, so no TRV could be established for this COPC.

3.5.2 Chloroform

Mammals

The chloroform reference dose used in the ecological model is based on a subchronic LOAEL determined from studies performed by Palmer *et al.,* (1979), cited in Sample *et al.* (1996). This study was based on oral exposure of rats to chloroform via gavage, at dose rates of 15, 30, 150, and 410 mg/kg-bw/day for 13 weeks. Gonadal atrophy was seen in both male and female rats at the highest dose, and so this value was considered to be the subchronic LOAEL. To account for the subchronic exposure duration a total uncertainty factor of 3 was applied to this value to arrive at a mammalian TRV of 136.6 mg/kg-bw/day. This value is used in the ecological model, and is assigned a high level of confidence.

Birds

No acceptable avian toxicological studies were identified for use in the derivation of a TRV. Therefore, no avian reference dose can be established for this COPC.

3.5.3 Dichlorobenzene

Dichlorobenzene (DCB) exists as one of three isomers: 1,2-DCB; 1,3-DCB; and 1,4-DCB. Most of the information on health effects of DCBs is from studies of 1,2-DCB and 1,4-DCB. Very little is known about the health effects of 1,3-DCB, especially in wildlife receptors, but they are likely to be similar to those of the other DCB isomers.

Breathing or ingesting any of the DCB isomers caused harmful effects on the livers of laboratory animals. Animal studies also found that 1,2-DCB and 1,4-DCB caused effects on the kidneys and blood, and that 1,3-DCB caused thyroid and pituitary effects. There is no clear evidence that 1,2-DCB or 1,4-DCB impair reproduction or fetal development in animals at levels below those that also cause serious maternal health effects, although there is an indication that 1,4-DCB can affect development of the nervous system after birth (ATSDR 2006).

Mammals

The 1,2-DCB reference dose is based on a chronic LOAEL determined from a study by the National Toxicology Program (NTP, 1985; in CEPA, 1993). Groups of 50 male and female rats and mice were administered 0, 60 or 120 mg/kg-bw of 1,2- DCB daily by gavage in corn oil, 5 days per week for 103 weeks (NTP, 1985). Adverse renal effects were observed at the 120 mg/kg-bw/day dose level in rats, and a of males, particularly in the high-dose group, was noted at the end of the study. The equivalent dose, adjusted to 7 days per week (*i.e.,* 87.5 mg/kg-bw/day) is considered to be a chronic LOAEL.

No mortality or overt signs of toxicity occurred in male or female Sprague-Dawley rats that were exposed to 1,3-DCB in corn oil by gavage at doses as high as 588 mg/kg-bw/day for 90 consecutive days (McCauley *et al.* 1995; as cited in ATSDR 2006). Endocrine, hepatic, hematological and body weight effects were observed at various dose levels. However, the significance of these effects for deriving an ecological TRV is questionable. Consequently, the highest dose (588 mg/kg-bw/day) is considered to be a subchronic LOAEL.

ATSDR (2006) provides results of one chronic orally administered reproductive toxicity study for 1,4-DCB (NTP, 1987). Mice and rats were given 1,4-DCB via oral gavage for 5 days per week over a period of two years. No reproductive effects were observed at the highest dose (600 mg/kg-bw/day). The equivalent dose, following adjustment to 7 days per week (428.6 mg/kg-bw/day) is considered to be a chronic NOAEL.

Taking into consideration the available information for 1,2-, 1,3-, and 1,4-DCB, the reference dose used for unspecified dichlorobenzenes in the ecological model is the chronic LOAEL value of 87.5 mg/kg-bw/day based on decreased survival of male rats. A medium level of confidence is assigned to this reference dose.

Birds

No acceptable avian toxicological studies were identified for use in the derivation of a TRV, and therefore no avian reference dose can be established for this COPC.

3.5.4 Dichloromethane

Mammals

The dichloromethane reference dose used in the ecological model is based on a chronic LOAEL determined from studies performed by the National Coffee Association (NCA, 1982), cited in Sample *et al.* (1996). This study was based on oral exposure of rats to dichloromethane in drinking water, and monitored several systemic endpoints. Groups of rats (85 /sex/dose) were exposed to dichloromethane at nominal doses of either 5, 50, 125, or 250 mg/kg in drinking water over a period of two years. Based on observations of liver histopathology, a LOAEL was determined at the 50.0 mg/kg-bw/day dose. This nominal dose correlates to actual daily doses of 52.58 and 58.32 mg/kg-bw/day for males and females respectively (IRIS, 1988). No oral toxicity studies observing reproductive endpoints have been identified for this compound. An ATSDR toxicological profile for dichloromethane is available, and indicates that the LOAEL derived by the NCA (1982) is conservative compared to effects thresholds for other chronic mammalian (oral) toxicity studies (ATSDR, 2000). The LOAEL for male rats (52.58 mg/kg-bw/day) from the NCA, (1982) is used as the reference dose for dichloromethane in the ecological model, and is assigned a high level of confidence.

Birds

No acceptable avian toxicological studies were identified for use in the derivation of a TRV, therefore no avian reference dose can be established for this COPC.

3.5.5 Hexachlorobenzene

Mammals

The hexachlorobenzene reference dose selected for this ERA model for mammal species is based on a study performed by Arnold *et al.* (1985). The study was based on oral exposure of parental (F0) and F1 generation of rats to hexachlorobenzene in feed at 0, 0.32, 1.6, 8, and 40 mg/kg over a period of 130 weeks (F0 exposed for 3 months and 50 F1 pups exposed for 130 weeks post weaning). There were no treatment related effects on growth, food consumption or survival in F0 or F1 generations but the F0 viability index was reduced at the 40 mg/kg dose. Given that both a sub-chronic LOAEL (change in F0 viability) and chronic NOAEL (no treatment related effects at highest dose in F1) can be determined, the chronic NOAEL (40 mg/kg, or 3.2 mg/kg/day based on the food ingestion rate and body weight of rats), was used as the reference dose rate for mammalian species. This reference dose is assigned a medium level of confidence.

Birds

The hexachlorobenzene reference dose selected for this ERA model for bird species is based on the chronic LOAEL determined from studies performed Vos *et al.* (1971), cited in Sample *et al.* (1996). The study was based on oral exposure of Japanese quail to mixed isomers of hexachlorobenzene in feed. The study endpoint was considered reproduction. Quail were exposed to hexachlorobenzene at concentrations of 1, 5, 20, and 80 mg hexachlorbenzene/kg diet over a period of 90 days. Egg volume and hatchability were observed to decrease at 20 mg/kg diet. Based on the food ingestion rate and body weight of the Japanese quail the reference dose rate (based on the LOAEL) for avian species is estimated to be 2.25 mg/kg-bw/day. This reference dose is assigned a high level of confidence.

3.5.6 Pentachlorobenzene

Mammals

The pentachlorobenzene reference dose selected for this ERA model for mammal species is based on the chronic LOAEL determined from studies performed Linder *et al.* (1980). The study was based on oral exposure of rats to pentachlorobenzene in feed. The study endpoint was considered hepatic and renal toxicological effects. Rats were exposed to pentachlorobenzene at concentrations of 0, 125, 250, 500, and 1000 mg pentachlorbenzene/kg diet over a period of 180 days. At the 125 mg/kg dosage, hepatotoxic effects were observed. Based on the food ingestion rate and body weight of rats, the reference dose rate (based on the sub-chronic LOAEL) for mammalian species is estimated to be 8.3 mg/kg-bw/day. An uncertainty factor of 3 is applied to this LOAEL to convert it from a sub-chronic to a chronic value. This reference dose is assigned a low level of confidence.

Birds

No acceptable avian toxicological studies were identified for use in the derivation of a TRV, therefore no avian reference dose can be established for this COPC.

3.5.7 Pentachlorophenol

The U.S. EPA has conducted an in-depth review of the toxicological literature pertaining to the effects of pentachlorophenol on birds and mammals (U.S. EPA, 2007e). Sixteen studies addressing effects on mammals, and three studies addressing effects on birds were identified and considered in the selection of a TRV for pentachlorophenol Eco-SSLs.

Mammals

The ecological model uses the same TRV the U.S. EPA uses for deriving an Eco-SSL that is protective of pentachlorophenol effects on mammals. Using the sixteen studies addressing effects on mammals, the U.S. EPA (2007e) applies the geometric mean of NOAELs from those studies which monitored growth or reproductive endpoints as the Eco-SSL TRV. This value (8.42 mg/kg-bw/day) is lower than any "bounded LOAEL" for studies monitoring growth, reproductive, or survival endpoints. The collection of toxicological literature from which this geometric mean was calculated contained studies using various exposure durations (*i.e.,* subchronic and chronic), and was therefore considered conservatively representative of chronic exposure without the application of uncertainty factors. Rats, mice, mink or sheep were the test subjects in each toxicological study considered for this geometric mean value. Therefore, body weight-based scaling factors are not applied since this dose is considered representative of small and large mammals. This reference dose is assigned a high level of confidence.

Birds

Of the three studies identified by the U.S. EPA for avian studies involving pentachlorophenol, only one, performed by Prescott *et al.,* (1982) was selected for use in deriving an avian TRV for this ERA. This TRV, a LOAEL of 22.5 mg/kg-day, is based on growth effects (change in body weight) of juvenile chickens sub-chronically exposed to pentachlorophenol via diet. A total uncertainty factor of 3 was applied to this LOAEL, based on the sub-chronic exposure duration. This reference dose is assigned a high level of confidence.

3.5.8 Terphenyl, o-

Mammals

No acceptable mammalian toxicological studies were identified for use in the derivation of a reference dose, so no TRV could be established for this COPC.

Birds

No acceptable avian toxicological studies were identified for use in the derivation of a reference dose, so no TRV could be established for this COPC.

3.5.9 Tetrachlorobenzene, 1,2,4,5-

Mammals

The TRV selected for this ERA model for mammal species is based on the sub-chronic LOAEL determined from studies performed by the National Toxicology Program (NTP) (1991) where male and female F344/N rats and B6C3F1 mice were given to 1,2,4,5-tetrachlorobenzene for 13 weeks at doses between 0 and 2000 ppm in corn oil. The no-observed-effect level (NOEL) from the study, based on thyroid lesions, was 30 ppm for male and female rats and 300 ppm for male and female mice. The next

highest dose, 100 ppm and 1000 ppm, respectively, can be considered the LOAEL. The lower of the two LOAELs, 100 ppm, converted to a sub-chronic LOAEL of 7 mg/kg/day based on body weight and food intake rates of the study animals, was selected for use in the ERA as the daily dose for mammal species. An uncertainty factor of 3 has been applied to this LOAEL based on the sub-chronic nature of the study. A medium level of confidence is assigned to this TRV.

Birds

No acceptable avian toxicological studies were identified for use in the derivation of a TRV, therefore no avian reference dose can be established for this COPC.

3.5.10 Tribromomethane (Bromoform)

Mammals

The tribromomethane TRV selected for this ERA model for mammal species is based on the chronic NOAEL determined from studies performed by the National Toxicology Program (NTP) (1989). The study was based on oral exposure of rats and mice to tribromomethane in corn oil. The study endpoint was reproductive; male and female mice exposed to doses of 200 mg/kg/day (the highest dose administered in the study), for five days a week over a two year period exhibited no adverse effects on fertility or reproductive performance. The U.S. EPA IRIS has adopted the sub-chronic LOAEL of 50 mg/kg/day from the NTP study for the derivation of a human health oral TRV however the LOAEL is based on cancer-related endpoints which are not relevant to this ERA.

The chronic NOAEL of 200 mg/kg/day corrected to 142.8 mg/kg/day (200 mg/kg/day x 5/7) was selected for use in the ERA as the daily dose for mammal species. Due to the paucity of additional studies to corroborate this value, a low level of confidence is assigned to this TRV.

Birds

No acceptable avian toxicological studies were identified for use in the derivation of a reference dose, so no TRV could be established for this COPC.

3.5.11 Trichlorobenzene, 1,2,4-

Mammals

The TRV selected for this ERA model for mammal species is based on the chronic LOAEL determined from studies performed by the Robinson *et al.* (1981). The study was based on oral exposure of rats (F0 and F1 generation) to 1,2,4-trichlorobenzene in drinking water. The study endpoint was effects on adrenal gland weights of progeny over several generations; rats were exposed to doses of 1,2,4-trichlorobenzene at concentrations of 0, 25, 100, or 400 mg/kg. Rats exposed to a dose of 400 mg/kg exhibited significant increases in adrenal gland weights. A number of other endpoints of toxicity were also assessed during this study (e.g., changes in organ mass, fertility, food and water intake, blood chemistry) and did not appear to vary in relation to exposure dose. 400 mg/kg, converted to a chronic LOAEL of 53.6 mg/kg/day (based on the mass and food intake rates of rat from the study), was selected for use in the ERA as the daily dose for mammal species. No uncertainty factors have been applied to this LOAEL, as the study was considered chronic due to the inclusion of a sensitive lifestage (reproduction). A medium level of confidence is assigned to this TRV.

Birds

No acceptable avian toxicological studies were identified for use in the derivation of a TRV, therefore no avian reference dose can be established for this COPC.

3.5.12 Trichloroethane, 1,1,1-

Mammals

The 1,1,1-Trichloroethane reference dose selected for this ERA model for mammalian species is based on a chronic oral gavage study performed by Lane *et al.* (1982), cited in Sample *et al.* (1996). 1,1,1-trichloroethane was administered to mice via drinking water for two generations (greater than one year) at concentrations of 0, 0.58, 1.75, or 5.83 mg/mL, and various reproductive endpoints were measured. No adverse effects were observed at any dose level. The upper concentration is therefore considered a chronic NOAEL, and corresponds to a daily dose of 1000 mg/kg-bw/day (based on a body weight of 0.035 kg, and a study-estimated water consumption of 6 mL/day).

The selection of this reference dose is based on a review of several studies for 1,1,1-trichloroethane. The ATSDR toxicological profile for 1,1,1-trichloroethane (2006b) provides a tabular summary of results for numerous oral toxicity tests of mammalian toxicity (*i.e.,* primarily rats and mice). The reference dose based on a chronic NOAEL established by Lane *et al.* (1982) is a conservative value in comparison to other effects levels for reproduction, development, or mortality endpoints presented in ATSDR (2006b). The ATSDR (2006b) presents results for one study by the National Cancer Institute (NCI), (1977) which observed adverse effects (i.e. mortality) to rats from exposure to less than 1000 mg/kg-bw/day (the NOAEL value reported by Lane *et al.* (1982)). The NCI (1977) study provided 1,1,1-trichloroethane to rats via oral gavage at two doses (750 or 1500 mg/kg-bw/day) for 78 weeks (5 days/week). Increased mortality was observed in both dose groups. However, the U.S. EPA (2007f) reports that results of the NCI test were likely influenced by the presence of chronic murine pneumonia (*i.e*. a bacterial infection) in both treated and control animals.

The NOAEL-based reference dose reported by Lane *et al.* (1982) of 1000 mg/kg-bw/day is therefore considered a conservative value, and is assigned a high level of confidence.

Birds

No acceptable avian toxicological studies were identified for use in the derivation of a TRV. Therefore, no avian reference dose can be established for this COPC.

3.5.13 Trichlorofluoromethane

Mammals

The trichlorfluoromethane reference dose selected for this ERA model for mammalian species is based on a chronic oral gavage study performed by the National Cancer Institute (NCI, 1978). Trichlorofluoromethane was provided to rats and mice five days per week at two dosages (50 animals/species/sex/dose) for 78 weeks. A significant increase in mortality (compared to controls) was observed in both sexes for rats, and in female mice at the 488 mg/kg/day dose. The value is adjusted to 349 mg/kg/day to represent a continuous daily dose (seven days per week), and is selected for use as the reference dose for mammalian species in the ecological model. It is assumed that this dose was the lowest administered dose since a NOAEL was not reported (in IRIS, 1992). This reference dose was based on the consideration of a single study, due to the limited availability of chronic oral

toxicity data for trichlorofluoromethane. Consequently, a low level of confidence is assigned to this value.

Birds

No acceptable avian toxicological studies were identified for use in the derivation of a TRV. Therefore, no avian reference dose can be established for this COPC.

Final mammalian and avian TRVs, with uncertainty factors and body-scaling applied, as appropriate, are listed below in Table 1.

Table 1: Final Mammalian and Avian TRVs used in this Assessment (mg/kg-bw/day)

COPC	Eastern Cottontail Rabbit	Masked Shrew	Meadow Vole	Mink	Muskrat	Red Fox	White-tailed Deer	American Robin	Belted Kingfisher	Great Blue Heron	Mallard Duck	Red-tailed Hawk	Wild Turkey
Polycyclic Aromatic Hydrocarbons													
Low Molecular Weight PAHs													
Acenaphthene	170	170	170	170	170	170	170	--	--	--	--	--	--
Acenaphthylene	170	170	170	170	170	170	170	--	--	--	--	--	--
Anthracene	170	170	170	170	170	170	170	--	--	--	--	--	--
Fluoranthene	170	170	170	170	170	170	170	--	--	--	--	--	--
Fluorene	170	170	170	170	170	170	170	--	--	--	--	--	--
Phenanthrene	170	170	170	170	170	170	170	--	--	--	--	--	--
High Molecular Weight PAHs													
Benz(a)anthracene	18	18	18	18	18	18	18	--	--	--	--	--	--
Benzo(a)pyrene	18	18	18	18	18	18	18	--	--	--	--	--	--
Benzo(e)pyrene	18	18	18	18	18	18	18	--	--	--	--	--	--
Benzo(a)fluorene	18	18	18	18	18	18	18	--	--	--	--	--	--
Benzo(b)fluorene	18	18	18	18	18	18	18	--	--	--	--	--	--
Benzo(b)fluoranthene	18	18	18	18	18	18	18	--	--	--	--	--	--
Benzo(g,h,i)perylene	18	18	18	18	18	18	18	--	--	--	--	--	--
Benzo(k)fluoranthene	18	18	18	18	18	18	18	--	--	--	--	--	--
Chrysene	18	18	18	18	18	18	18	--	--	--	--	--	--
Dibenz(a,c)anthracene	18	18	18	18	18	18	18	--	--	--	--	--	--
Dibenz(a,h)anthracene	18	18	18	18	18	18	18	--	--	--	--	--	--
Indeno(1,2,3-cd)pyrene	18	18	18	18	18	18	18	--	--	--	--	--	--
Perylene	18	18	18	18	18	18	18	--	--	--	--	--	--
Pyrene	18	18	18	18	18	18	18	--	--	--	--	--	--
Dioxins and Furans													
2,3,7,8-TCDD Equivalent	7.35E-06	1.00E-05	1.00E-05	8.01E-06	7.40E-06	5.28E-06	2.76E-06	1.40E-04	1.40E-04	1.15E-04	1.35E-04	1.37E-04	9.78E-05
PCB													
Aroclor 1254 (Total PCBs)	0.223	0.680	0.517	0.244	0.225	0.161	0.084	1.80	1.80	1.47	1.73	1.76	1.26
Chlorinated Monocyclic Aromatics													
1,2-Dichlorobenzene	34.8	87.5	80.4	37.9	35.0	25.0	13.1	--	--	--	--	--	--
1,2,4-Trichlorobenzene	39.4	53.6	53.6	42.9	39.6	28.3	14.8	--	--	--	--	--	--
1,2,4,5-Tetrachlorobenzene	1.71	2.33	2.33	1.87	1.73	1.23	0.64	--	--	--	--	--	--
Pentachlorobenzene	2.03	2.77	2.77	2.22	2.05	1.46	0.76	--	--	--	--	--	--
Hexachlorobenzene	2.35	3.20	3.20	2.56	2.37	1.69	0.88	2.25	2.25	1.15	1.35	1.37	0.98
Pentachlorophenol	8.42	8.42	8.42	8.42	8.42	8.42	8.42	7.50	7.50	6.90	7.50	7.50	5.89
Chlorinated Solvents and Derivatives													
Carbon Tetrachloride	11.8	16.0	16.0	12.8	11.8	8.4	4.4	--	--	--	--	--	--
Chloroform	100	137	137	109	101	72	38	--	--	--	--	--	--
Dichloromethane	36.7	50.0	50.0	40.1	37.0	26.4	13.8	--	--	--	--	--	--
Trichlorofluoromethane (Freon 11)	256	349	349	280	258	184	96	--	--	--	--	--	--
Chlorinated Alkanes/Alkenes													
1,1,1-Trichloroethane	413	1000	955	450	416	297	155	--	--	--	--	--	--
Other Organics													
Bromoform	56.8	142.8	131.3	61.9	57.1	40.8	21.4	--	--	--	--	--	--
O-Terphenyl	--	--	--	--	--	--	--	--	--	--	--	--	--
Inorganics													
Antimony	0.435	0.592	0.592	0.474	0.438	0.313	0.164	--	--	--	--	--	--
Arsenic	1.66	1.66	1.66	1.66	1.66	1.66	1.17	12.8	12.8	10.5	12.4	12.5	9.0
Barium	51.8	51.8	51.8	51.8	51.8	51.8	51.8	139	132	67	79	80	57
Beryllium	0.424	0.532	0.532	0.463	0.427	0.305	0.160	--	--	--	--	--	--

Table 1: Final Mammalian and Avian TRVs used in this Assessment (mg/kg-bw/day)

COPC	Eastern Cottontail Rabbit	Masked Shrew	Meadow Vole	Mink	Muskrat	Red Fox	White-tailed Deer	American Robin	Belted Kingfisher	Great Blue Heron	Mallard Duck	Red-tailed Hawk	Wild Turkey
Boron	20.6	28.0	28.0	22.4	20.7	14.8	7.7	100	100	82	96	98	70
Cadmium	0.910	0.910	0.910	0.910	0.910	0.910	0.910	1.47	1.47	1.47	1.47	1.47	1.47
Chromium (Total)	2.40	2.40	2.40	2.40	2.40	2.40	2.40	2.66	2.66	2.66	2.66	2.66	2.66
Chromium VI	9.24	9.24	9.24	9.24	9.24	9.24	9.24	4.02	4.02	3.70	4.02	4.02	3.16
Cobalt	7.33	7.33	7.33	7.33	7.33	7.33	7.33	2.54	2.54	2.54	2.54	2.54	2.54
Lead	4.70	4.70	4.70	4.70	4.70	4.70	4.70	10.9	10.9	10.9	10.9	10.9	10.9
Mercury - Inorganic	0.965	1.010	1.010	1.010	0.971	0.693	0.363	0.900	0.900	0.458	0.540	0.547	0.391
Methyl Mercury	0.118	0.160	0.160	0.128	0.118	0.084	0.044	0.064	0.064	0.052	0.062	0.062	0.045
Nickel	3.31	3.31	3.31	3.31	3.31	3.31	3.31	6.71	6.71	6.71	6.71	6.71	6.71
Selenium	0.101	0.101	0.101	0.101	0.101	0.101	0.101	1.00	1.00	0.82	0.96	0.98	0.70
Silver	20.1	20.1	20.1	20.1	20.1	20.1	20.1	6.73	6.73	4.97	5.85	5.93	4.24
Thallium	0.183	0.247	0.247	0.200	0.184	0.132	0.069	0.335	0.286	0.146	0.171	0.174	0.124
Tin	44	44	44	44	44	44	44	--	--	--	--	--	--
Vanadium	3.76	5.11	5.11	4.09	3.78	2.70	1.41	0.229	0.217	0.111	0.130	0.132	0.094
Zinc	75.9	75.9	75.9	75.9	75.9	75.9	75.9	66.5	66.5	66.5	66.5	66.5	66.5

"-- " – Suitable TRV could not be identified for this COPC.

4.0 COMMUNITY BENCHMARKS

4.1 PHYTOTOXICITY AND SOIL INVERTEBRATE SCREENING BENCHMARKS

For certain COPC, phytotoxicity and soil invertebrate benchmarks have not been established by the various agencies mentioned in the introduction which prevents a quantitative assessment of these chemicals. Many of the values chosen for screening benchmarks are the full depth generic site condition standards (non-potable groundwater condition) developed by the MOE (2004), but only those standards based on an ecotoxicity criterion (*i.e.,* a Netherlands "C-value" or a value from another agency, typically a soil quality guideline published by the CCME) for 1) coarse-grain soils; and 2) from the most conservative value between residential/parkland and commercial/industrial land uses. U.S. EPA and ORNL values were also reviewed where available. The U.S. EPA (http://www.epa.gov/ecotox/ecossl/) publishes a number of interim Eco-SSL values for various metals and organics. The ORNL has released two documents addressing soil toxicity to terrestrial vegetation (Efroymson *et al.,*1997a) and soil litter invertebrates (Efroymson *et al.* 1997b).

The Netherlands "C-value" for soil remediation

In the Netherlands, C-values have been replaced by intervention values, which incorporate both a human and ecological component (Swartjes, 1999). The C-values selected by the MOE represent the ecological component of the current intervention values. These ecological intervention values represent a soil concentration, at which adverse ecological effects are expected to occur. Above this concentration, soils are considered "seriously contaminated" (VROM, 2000).

Each ecological intervention value is developed using toxicity studies on ecosystem species, and microbial and enzymatic processes. Priority is given to NOAELs and LOAELs, however lethal effects concentration are also used (divided by an uncertainty factor of 10). Toxicity data for species from a wide range of taxonomic groups are considered, including several plant and invertebrate phyla. Avian and mammalian toxicity is also considered for those substances where a potential for secondary exposure exists.

MOE-adopted soil quality values

As an alternative to the Netherlands "C-value" the MOE often adopts another soil quality guideline to represent the ecotoxicity criterion used towards the development of the full depth generic site condition standards. Most frequently this value is a CCME ecological soil quality guideline (SQG). These SQG are based on soil contact using toxicity data from studies on both plants and invertebrates. For agricultural land uses, soil and food ingestion toxicity to birds and mammals is also considered.

4.2 PHYTOXICITY BENCHMARKS

4.2.1.1 Sulphur Dioxide Effects on Plants

The International Union of Forest Association (IUFRO) first developed air quality standards for protection of vegetation that were primarily based on field observations in 1978. The protection of trees in good growing conditions was established to be at 50 µg/m^3 and at 25 µg/m^3 for trees in poor sites (Wentzel, 1983). These values were based on field evidence (*i.e*. phytotoxicity symptoms) derived from

Finland and mountain regions of Central Europe for coniferous species which are much more sensitive to SO_2 under cold temperatures. The more recent guidelines (WHO, 2000) are based on either field or experimental data. In particular, the critical level of 50 µg/m^3 is based on an analysis of experimental data to identify the lowest concentration producing and adverse effect in a study performed by Bell (1985) with perennial ryegrass.

High concentrations of sulphur dioxide can produce acute injury in vegetation, which is first observed on the plant foliage. The plant foliage is more sensitive than stems, buds or reproductive parts (Legge and Krupa 2002). The most prevalent phytotoxicity symptoms occur in the form of foliar necrosis, even after relatively short duration exposure. Depending on the plant species, the necrotic areas can vary in colour from white to brown to black.

The main pathway of entry for sulphur dioxide into a leaf is through the stomata. Once inside the leaf, the sulphur dioxide dissolves in toxic compounds, which can cause partial or wider stomatal opening (Mansfield and Pearson 1997). These can lead to depression of the photosynthetic activity and transpirational water loss.

The acute effects are generally less important in the field than chronic effects, which result from long-term exposure to much lower concentrations, with periodic intermittent and random peak levels (Krupa 1996). Long-term exposure may induce chronic injury symptoms such as marginal or interveinal chlorosis in broad leaf plants and premature fall colouration and premature leaf abscission (Legge et al. 1998). Often reduced growth and yield and increased senescence have been reported without the development of visible foliar injury symptoms. The effects of sulphur dioxide on vegetation are determined by both biotic (plant stage of growth, nutrient status, insects and disease) and abiotic factors (air and soil temperature, humidity, radiation, precipitation).

4.1.1.1 Nitrogen Dioxide Effects on Plants

As described in Garner *et al.* (1989), typically, nitrogen oxides are rarely found in concentrations high enough to cause visible injury to vegetation. Typical concentrations of nitrogen oxides in ambient air are around 20 ppb (40 µg/m^3) in urban areas of North America, compared to typical concentrations of 10 ppb (20 µg/m^3) in near-urban or rural forests (U.S. EPA, 1993). In forested ecosystems which are away from urban sources, concentrations of nitrogen oxides are typically at or below the detection limit of <3 ppb (<6 µg/m^3). At low concentrations, NOx can stimulate growth of vegetation; however at higher concentrations growth can be reduced (WHO 2000).

Nitrogen-containing air pollutants, in particular NO_2, have been reported to cause leaf damage. However, the concentrations at which phytotoxicity symptoms were reported to occur due to nitrogen oxides exposures were usually very high (Taylor *et al*. 1975). Most important it is the combination of other air pollutants such as sulphur dioxide and ozone that cause a higher degree of phytotoxicity.

Nitrogen oxides uptake in terrestrial plants is occurring mostly through stomatal diffusion from atmosphere into the intercellular spaces of the leaf, while smaller amounts are being taken up through deposition and passage through the cuticle (Kerstiens, 1996). Nitrogen oxides can then be involved in the nitrogen metabolism of plants and affect regulatory mechanisms such as enzymes production and the nitrate/nitrite reduction pathways (Stulen *et al*. 1998).

Nitrogen oxides can affect vegetation indirectly, via chemical reactions in the atmosphere, or directly after being deposited on vegetation, soil or water. The indirect pathway refers to the atmospheric chemistry of NO and NO_2 which are of key importance in the production and removal of tropospheric

ozone (O_3) (Mansfield, 2002). The direct impact of airborne nitrogen is due to toxic effects, eutrophication and acidification.

Symptoms such as chlorosis, browning or bleaching between the leaf veins, especially near margins have been reported (Malhotra and Blauel, 1980) in deciduous trees species at acute NO_2 concentrations.

The World Health Organization (2000) describes two different types of effect threshold: critical levels and critical loads. The critical level (CLE) is the concentration in the atmosphere above which direct adverse effects on receptors, such as plants, ecosystems or materials, may occur. CLEs are expressed in terms of exposure (µg/m^3 and exposure duration), while critical load CLOs are expressed in terms of deposition (kg N/ha per year). As reported by the WHO (2000) the critical level (CLE) for NOx (NO + NO_2, expressed as NO_2 in µg/m^3) is 30 µg/m^3 as an annual mean and 75 µg/m^3 as a 24-hour mean. The critical load (CLO) was established at 30 kg /ha year. The CLE was primarily used in this ERA for the evaluation of the NO_2 effects emissions on the vegetation.

4.1.1.1 Fluorides Effects on Plants

Fluorine and many fluorides such as hydrogen fluoride have been reported to cause foliar injuries in areas where emissions from certain industrial processes exist such as the manufacture of aluminum, bricks, glass and steel. The predominant pathway by which gaseous fluoride enters the plant is diffusion through the leaf stomata (pores) where it then dissolves in water and concentrates in the leaf margins and tip. It is therefore these areas that typically are the first to show visible injury. Leaves are generally most sensitive to fluoride when they are young; once fully developed they may be much more resistant. Where exposure is periodic, symptoms may reflect this, as only those leaves that are at the sensitive stage of development when the exposure occurs will develop injury.

Davison (1986) reported that when the diffusion through the aqueous phase of the mesophyll from the substomatal is the rate limiting factor, HF will be absorbed at a greater rate than other gaseous pollutants because of its lower molecular weight and greater solubility in water. The particulate F that is deposited on the leaf surface can slowly penetrate into the leaf, depending on the particle size, solubility of the material, relative humidity and the presence of free water on the foliar surface.

The visible foliar symptoms induced by exposure to HF depend primarily on the type of species of plant and secondarily on the concentration and duration of the exposure time (Weinstein *et al*., 1998).

Exposure to a high concentration of fluorides causes necrosis (tissue death) of part, or even the whole, of the leaf. The initial stages vary with species, and both the speed of development of the symptoms and their appearance depend on the weather. In most monocotyledonous (narrow-leaved species including grasses and lilies) plants, the initial symptom is the development of chlorosis (yellowing) at the tips and margins of elongating leaves. In dicotyledonous (broad-leaved) species the initial symptom of fluoride effects on leaves is usually chlorosis of the tip, which later extends downward along the margins and inward toward the midrib and deformation such as a downward cupping. Continued exposure may lead to the tip becoming necrotic and falling off, leaving the leaf notched.

Based on a literature search of air quality standards for gaseous fluoride two different approaches were identified. The first was to limit the acceptable concentrations of fluoride in vegetation which would be protective of adverse effects both on the plants themselves and also of livestock which would feed on the plants. For example, in the state of Maryland, the standard specifies maximum concentrations of 100 ppm F in deciduous trees and shrubs, 50 ppm F in washed samples of the current year's growth of

conifers and evergreen trees and shrubs, and 75 ppm F in previous season's growth of conifers. The maximum concentration for field crops (washed) is 30 ppm F, while forage (unwashed) varies from 80 ppm F for the average of samples collected during one month, 60 ppm F for the average of any two consecutive months, and 35 ppm F for the average samples collected in 1 year.

The second approach to reduce the risk of fluoride effects on vegetation is the development of standards of acceptable levels of HF in the air which reduced the risk of adverse effects on vegetation (Scholl, 1971). These levels would protect against acute effects, short-term exposures and also longer term exposures during which accumulation could occur. The suggested acceptable limits range from 1.6 – 10.0 µg/m^3 for a 24 hour averaging period, 0.4 – 2.5 µg/m^3 for a one month averaging period, and 0.25 – 1.2 µg/m^3 for a seven month averaging period (McCune & Weinstein, 2002).

4.1.1 Polycyclic Aromatic Hydrocarbons

Acenaphthene

An established phytotoxicity benchmark or suitable toxicity data could not be identified for acenaphthene in a review of scientific literature or current regulatory documents.

Acenaphthylene

An established phytotoxicity benchmark or suitable toxicity data could not be identified for acenaphthylene in a review of scientific literature or current regulatory documents.

Anthracene

A soil concentration of 40 mg/kg is used as the phytotoxicity benchmark for anthracene in the ecological model. This value is the Netherland C-Level value (updated in 1993), and represents the ecotoxicity component of the full depth generic site condition standard (non-potable groundwater condition) developed by the MOE for anthracene (2004).

Fluoranthene

A soil concentration of 40 mg/kg is used as the phytotoxicity benchmark for fluoranthene in the ecological model. This value is the Netherland C-Level value (updated in 1993), and represents the ecotoxicity component of the full depth generic site condition standard (non-potable groundwater condition) developed by the MOE for fluoranthene (2004). The ecotoxicity component is the lowest of all components for fluoranthene, and is therefore also used as the generic standard.

Fluorene

An established phytotoxicity benchmark or suitable toxicity data could not be identified for fluorene in a review of scientific literature or current regulatory documents.

Phenanthrene

A soil concentration of 40 mg/kg is used as the phytotoxicity benchmark for phenanthrene in the ecological model. This value is the Netherland C-Level value (updated in 1993), and represents the ecotoxicity component of the full depth generic site condition standard (non-potable groundwater condition) developed by the MOE for phenanthrene (2004).

Benzo(a)anthracene

A soil concentration of 40 mg/kg is used as the phytotoxicity benchmark for benzo(a)anthracene in the ecological model. This value is the Netherland C-Level value (updated in 1993), and represents the ecotoxicity component of the full depth generic site condition standard (non-potable groundwater condition) developed by the MOE for benzo(a)anthracene (2004). It is also the lowest of the health criteria available for benzo(a)anthracene, and is therefore selected as the MOE generic standard (for all land uses).

Benzo(a)pyrene

A soil concentration of 40 mg/kg is used as the phytotoxicity benchmark for benzo(a)pyrene in the ecological model. This value is the Netherland C-Level value (updated in 1993), and represents the ecotoxicity component of the full depth generic site condition standard (non-potable groundwater condition) developed by the MOE for benzo(a)pyrene (2004). The ecotoxicity component is the lowest of all components for benzo(a)pyrene, and is therefore also used as the generic standard.

Benzo(e)pyrene

An established phytotoxicity benchmark or suitable toxicity data could not be identified for benzo(e)pyrene in a review of scientific literature or current regulatory documents.

Benzo(a)fluorene

An established phytotoxicity benchmark or suitable toxicity data could not be identified for benzo(a)fluorene in a review of scientific literature or current regulatory documents.

Benzo(b)fluorene

An established phytotoxicity benchmark or suitable toxicity data could not be identified for benzo(b)fluorene in a review of scientific literature or current regulatory documents.

Benzo(b)fluoranthene

An established phytotoxicity benchmark or suitable toxicity data could not be identified for benzo(b)fluoranthene in a review of scientific literature or current regulatory documents.

Benzo(g,h,i)perylene

A soil concentration of 40 mg/kg is used as the phytotoxicity benchmark for benzo(g.h,i)perylene in the ecological model. This value is the Netherland C-Level value (updated in 1993), and represents the ecotoxicity component of the full depth generic site condition standard (non-potable groundwater condition) developed by the MOE for benzo(g.h,i)perylene (2004). The ecotoxicity component is the lowest of all components for benzo(g.h,i)perylene, and is therefore also used as the generic standard.

Benzo(k)fluoranthene

A soil concentration of 40 mg/kg is used as the phytotoxicity benchmark for benzo(k)fluoranthene in the ecological model and it represents the ecotoxicity component value for benzo(k)fluoranthene. This value is the Netherland C-Level value (updated in 1993), and represents the ecotoxicity component of the full depth generic site condition standard (non-potable groundwater condition) developed by the MOE for benzo(k)fluoranthene (2004).

Chrysene

A soil concentration of 40 mg/kg is used as the phytotoxicity benchmark for chrysene in the ecological model. This value is the Netherland C-Level value (updated in 1993), and represents the ecotoxicity component of the full depth generic site condition standard for Chrysene.

Dibenz(a,c)anthracene

An established phytotoxicity benchmark or suitable toxicity data could not be identified for dibenz(a,c)anthracene in a review of scientific literature or current regulatory documents.

Dibenz(a,h)anthracene

An established phytotoxicity benchmark or suitable toxicity data could not be identified for dibenz(a,h)anthracene in a review of scientific literature or current regulatory documents.

Indeno(1,2,3-cd)pyrene

A soil concentration of 40 mg/kg is used as the phytotoxicity benchmark for indeno(1,2,3-cd)pyrene in the ecological model. This value is the Netherland C-Level value (updated in 1993), and represents the ecotoxicity component of the full depth generic site condition standard (non-potable groundwater condition) developed by the MOE for indeno(1,2,3-cd)pyrene (2004). The ecotoxicity component is the lowest of all components for indeno(1,2,3-cd)pyrene, and is therefore also used as the generic standard.

Perylene

An established phytotoxicity benchmark or suitable toxicity data could not be identified for perylene in a review of scientific literature or current regulatory documents.

Pyrene

An established phytotoxicity benchmark or suitable toxicity data could not be identified for pyrene in a review of scientific literature or current regulatory documents.

4.1.2 Dioxins and Furans

An established phytotoxicity benchmark or suitable toxicity data could not be identified for dioxins and furans in a review of scientific literature or current regulatory documents.

4.1.3 Total PCBs

The soil concentration used as the phytotoxicity benchmark in the ecological model for total PCBs is 40 mg/kg. This value is a screening benchmark published by the Oak Ridge National Laboratory (Efroymson *et al.* 1997a), on the basis of effects observed in plants grown in 40 ppm PCB surface soil (Strek and Weber (1980); in Efroymson *et al.* 1997a).

4.1.4 Other Organics

Dichlorobenzene

A soil concentration of 30 mg/kg is used as the phytotoxicity benchmark for dichlorobenzene in the ecological model. This value is a Netherlands “C-value” and represents the ecotoxicity criterion used

towards selection of the MOE full depth generic site condition standard for non-potable groundwater conditions.

1,2,4-Trichlorobenzene

A soil concentration of 30 mg/kg is used as the phytotoxicity benchmark for 1,2,4-trichlorobenzene in the ecological model. This value is a Netherlands "C-value" and represents the ecotoxicity criterion used towards selection of the MOE full depth generic site condition standard for non-potable groundwater conditions.

1,2,4,5-Tetrachlorobenzene

An established phytotoxicity benchmark or suitable toxicity data could not be identified for 1,2,4,5-tetrachlorobenzene in a review of scientific literature or current regulatory documents.

Pentachlorobenzene

An established phytotoxicity benchmark or suitable toxicity data could not be identified for pentachlorobenzene in a review of scientific literature or current regulatory documents.

Hexachlorobenzene

A soil concentration of 30 mg/kg is used as the phytotoxicity benchmark for hexachlorobenzene in the ecological model. This value is a Netherlands "C-value" and represents the ecotoxicity criterion used towards selection of the MOE full depth generic site condition standard for non-potable groundwater conditions.

Pentachlorophenol

The U.S. EPA has recently conducted an in-depth review of the toxicological literature pertaining to the effects of pentachlorophenol on plants (U.S. EPA, 2007e). Of 43 studies examined, four studies met all 11 U.S. EPA Study Acceptance Criteria, and were considered in the selection of a benchmark for pentachlorophenol. The U.S. EPA defines a pentachlorophenol screening benchmark for plants of 5 mg/kg (U.S. EPA 2007e).

Based on Ontario specific guidance, a soil concentration of 5 mg/kg is used as the phytotoxicity benchmark for pentachlorophenol in the ecological model. This value is a Netherlands "C-value" and represents the ecotoxicity criterion used towards selection of the MOE full depth generic site condition standard for non-potable groundwater conditions.

Carbon Tetrachloride

An established phytotoxicity benchmark or suitable toxicity data could not be identified for carbon tetrachloride in a review of scientific literature or current regulatory documents.

Chloroform

An established phytotoxicity benchmark or suitable toxicity data could not be identified for chloroform in a review of scientific literature or current regulatory documents.

Dichloromethane

An established phytotoxicity benchmark or suitable toxicity data could not be identified for dichloromethane in a review of scientific literature or current regulatory documents.

Trichlorofluoromethane (Freon 11)

An established phytotoxicity benchmark or suitable toxicity data could not be identified for trichlorofluoromethane in a review of scientific literature or current regulatory documents.

1,1,1-Trichloroethane

An established phytotoxicity benchmark or suitable toxicity data could not be identified for 1,1,1-trichloroethane in a review of scientific literature or current regulatory documents.

Tribromomethane (Bromoform)

An established phytotoxicity benchmark or suitable toxicity data could not be identified for tribromomethane in a review of scientific literature or current regulatory documents.

o-Terphenyl

An established phytotoxicity benchmark or suitable toxicity data could not be identified for o-terphenyl in a review of scientific literature or current regulatory documents.

4.1.5 Inorganics

Antimony

The U.S. EPA has recently conducted an in-depth review of the toxicological literature pertaining to the effects of antimony on plants (U.S. EPA, 2005a). Of 12 studies examined, only one study met all 11 U.S. EPA Study Acceptance Criteria, and was considered in the selection of a benchmark. As the result of a lack of data, the U.S. EPA was not able to define an antimony screening benchmark for plants (U.S. EPA 2005a).

Based on Ontario specific guidance, the soil concentration used as the phytotoxicity benchmark in the ecological model for antimony is 20 mg/kg. This value represents the ecotoxicity criteria for the full depth generic site condition standard (non-potable groundwater condition) developed by the MOE for antimony (2004).

Arsenic

The U.S. EPA has recently conducted an in-depth review of the toxicological literature pertaining to the effects of arsenic on plants (U.S. EPA, 2005b). Of 171 studies examined, 26 studies met all 11 U.S. EPA Study Acceptance Criteria, and were considered in the selection of a benchmark. The U.S. EPA defines an arsenic screening benchmark for plants of 18 mg/kg (U.S. EPA 2005b).

Based on Ontario specific guidance, the soil concentration used as the phytotoxicity benchmark in the ecological model for arsenic is 20 mg/kg. This value represents the ecotoxicity criteria for the full depth generic site condition standard (non-potable groundwater condition) developed by the MOE for arsenic (2004).

Barium

The U.S. EPA has recently conducted an in-depth review of the toxicological literature pertaining to the effects of barium on plants (U.S. EPA, 2005c). Of 30 studies examined, only three studies met all 11 U.S. EPA Study Acceptance Criteria, and were considered in the selection of a benchmark. As the result of a lack of data, the U.S. EPA was not able to define a barium screening benchmark for plants (U.S. EPA 2005c).

Based on Ontario specific guidance, a soil concentration of 750 mg/kg is used as the phytotoxicity benchmark for barium in the ecological model. This value represents the ecotoxicity criteria for the full depth generic site condition standard (non-potable groundwater condition) developed by the MOE for barium (2004). The ecotoxicity criteria is the lowest of all components for barium, and is therefore also used as the generic standard. The MOE adopted this value from the CCME, where 750 mg/kg is the interim soil remediation criteria value for agricultural land use (CCME, 2007).

Beryllium

The U.S. EPA has recently conducted an in-depth review of the toxicological literature pertaining to the effects of barium on plants (U.S. EPA, 2005d). Of 32 studies examined, only three studies met all 11 U.S. EPA Study Acceptance Criteria, and were considered in the selection of a benchmark. As the result of a lack of data, the U.S. EPA was not able to define a beryllium screening benchmark for plants (U.S. EPA 2005d).

Based on Ontario specific guidance, the soil concentration used as the phytotoxicity benchmark in the ecological model for beryllium is 4 mg/kg. This value represents the ecotoxicity criteria for the full depth generic site condition standard (non-potable groundwater condition) developed by the MOE for beryllium (2004).

Boron

A soil concentration of 30 mg/kg is used as the phytotoxicity benchmark for boron in the ecological model, and represents the 97.5 percentile of the sample population background concentration for Old Urban Parkland / Rural Parkland, as defined by the MOE (1993). This value was deemed applicable based on close corroboration with evidence provided in the MOEE 1996 rationale document, which states that generally less than 5% of total soil B is found in plant available forms (Gupta, 1979; in MOEE, 1996). It has been well established that availability of boron to plants is strongly associated with the hot-water soluble fraction, which usually ranges from 0.4 to 4.7% of total boron (Gupta and McLeod 1982). Because soil concentrations utilized in this assessment are based on total boron, and assuming that the 1.5 mg/kg MOE benchmark (MOE, 1996) for available boron accounts for 4.7% of the total soil boron, a simple scaling up of the benchmark was performed to arrive at 32 mg/kg, which is very close to the 30 mg/kg benchmark chosen for this assessment.

Cadmium

The U.S. EPA has recently conducted an in-depth review of the toxicological literature pertaining to the effects of cadmium on plants (U.S. EPA, 2005e). Of 716 studies examined, 62 studies met all 11 U.S. EPA Study Acceptance Criteria, and were considered in the selection of a benchmark. The U.S. EPA defines a cadmium screening benchmark for plants of 32 mg/kg (U.S. EPA 2005e).

Based on Ontario specific guidance, a soil concentration of 12 mg/kg is used as the phytotoxicity benchmark for cadmium in the ecological model. This value is a Netherlands “C-value” and represents the ecotoxicity criterion used towards selection of the MOE full depth generic site condition standard for non-potable groundwater conditions (MOE, 2004).

Chromium III (Total)

The U.S. EPA has recently conducted an in-depth review of the toxicological literature pertaining to the effects of chromium (all forms) on plants (U.S. EPA, 2008). Of 150 studies examined, 11 studies met

all 11 U.S. EPA Study Acceptance Criteria, and were considered in the selection of a benchmark. A lack of adequate data precluded the derivation of a plant toxicity benchmark (U.S. EPA 2008).

A soil concentration of 750 mg/kg is used as the phytotoxicity benchmark for total chromium in the ecological model. This value represents the ecotoxicity criteria for the full depth generic site condition standard (non-potable groundwater condition) developed by the MOE for chromium (2004). The ecotoxicity component is the lowest of all criteria for chromium, and is therefore also used as the generic standard. The MOE adopted this value from the CCME, where 750 mg/kg is the interim soil remediation criteria value for agricultural land use (CCME, 2007).

Chromium VI

A soil concentration of 8 mg/kg is used as the phytotoxicity benchmark for hexavalent chromium in the ecological model. This value represents the ecotoxicity criteria for the full depth generic site condition standard (non-potable groundwater condition) developed by the MOE for chromium (2004). The ecotoxicity component is the lowest of all criteria for hexavalent chromium, and is therefore also used as the generic standard. The MOE adopted this value from the CCME, where 8 mg/kg is the interim soil remediation criteria value for agricultural land use (CCME, 2007).

Cobalt

The U.S. EPA has recently conducted an in-depth review of the toxicological literature pertaining to the effects of cobalt on plants (U.S. EPA, 2005f). Of 152 studies examined, four studies met all 11 U.S. EPA Study Acceptance Criteria, and were considered in the selection of a benchmark. The U.S. EPA defines a cobalt screening benchmark for plants of 13 mg/kg (U.S. EPA 2005f).

Based on Ontario specific guidance, the soil concentration used as the phytotoxicity benchmark in the ecological model for cobalt is 40 mg/kg. This value represents the ecotoxicity criteria for the full depth generic site condition standard (non-potable groundwater condition) developed by the MOE for cobalt (2004).

Lead

As part of the Eco-SSL literature search process, a total of 439 papers were selected for detailed review. Of those papers acquired, 30 studies were used to derive the U.S. EPA Eco-SSL (2005g). The Eco-SSL is the geometric mean of the MATC values for four test species (loblolly pine, red maple, berseem clover and rye grass) under three different test conditions (pH and % OM) and is equal to 120 mg/kg dw (U.S. EPA 2005g); this value was used in the ecological model as the screening benchmark.

Mercury (Total)

A soil concentration of 10 mg/kg is used as the phytotoxicity benchmark for total mercury in the ecological model. This value is a Netherlands “C-value” and represents the ecotoxicity criterion used towards selection of the MOE full depth generic site condition standard for non-potable groundwater conditions.

Methyl Mercury

A soil concentration of 10 mg/kg is used as the phytotoxicity benchmark for total mercury in the ecological model. This value is a Netherlands “C-value” and represents the ecotoxicity criterion used

towards selection of the MOE full depth generic site condition standard for non-potable groundwater conditions.

Nickel

The U.S. EPA has recently conducted an in-depth review of the toxicological literature pertaining to the effects of nickel on plants (U.S. EPA, 2007a). Of 252 studies examined, 26 studies met all 11 U.S. EPA Study Acceptance Criteria, and were considered in the selection of a benchmark. The U.S. EPA defines a nickel screening benchmark for plants of 38 mg/kg (U.S. EPA 2007a).

Based on Ontario specific guidance, a soil concentration of 150 mg/kg is used as the phytotoxicity benchmark for nickel in the ecological model. This value is the MOE ecotoxicity criterion used towards selection of the MOE full depth generic site condition standard for non-potable groundwater conditions (MOE, 2004).

Phosphorus

An established phytotoxicity benchmark or suitable toxicity data could not be identified for phosphorus in a review of scientific literature or current regulatory documents.

Selenium

The U.S. EPA has recently conducted an in-depth review of the toxicological literature pertaining to the effects of selenium on plants (U.S. EPA, 2007b). Of 184 studies examined, 16 studies met all 11 U.S. EPA Study Acceptance Criteria, and were considered in the selection of a benchmark. The U.S. EPA defines a selenium screening benchmark for plants of 0.52 mg/kg (U.S. EPA 2007b).

Based on Ontario provincial guidance (MOE, 2004), a recommended total soil selenium concentration of 10 mg/kg is given for the protection of ecological receptors. This value of 10 mg/kg was used in the ecological model as the screening benchmark for terrestrial plants.

Silver

The U.S. EPA has recently conducted an in-depth review of the toxicological literature pertaining to the effects of silver on plants (U.S. EPA, 2006). Of 61 studies examined, two studies met all 11 U.S. EPA Study Acceptance Criteria, and were considered in the selection of a benchmark. The U.S. EPA defines a silver screening benchmark for plants of 560 mg/kg (U.S. EPA 2006).

Based on Ontario specific guidance, a soil concentration of 20 mg/kg is used as the phytotoxicity benchmark for silver in the ecological model. This value is the MOE ecotoxicity criterion used towards selection of the MOE full depth generic site condition standard for non-potable groundwater conditions.

Thallium

The soil concentration used as the phytotoxicity benchmark in the ecological model for thallium is 1 mg/kg. This value is a screening benchmark published by the Oak Ridge National Laboratory (Efroymson *et al.* 1997a), on the basis of toxic effects observed in plants grown in 1 ppm thallium surface soil (Kabata-Pendias and Pendias, 1984; in Efroymson *et al.* 1997a).

Tin (Inorganic)

The soil concentration used as the phytotoxicity benchmark in the ecological model for tin is 500 mg/kg. This value is a screening benchmark published by the Oak Ridge National Laboratory (Efroymson *et al.*

1997a), on the basis of effects observed in plants grown in 5 ppm tin surface soil (Romney *et al.*, 1975; in Efroymson *et al.* 1997).

Vanadium

The U.S. EPA has recently conducted an in-depth review of the toxicological literature pertaining to the effects of vanadium on plants (U.S. EPA, 2005h). Of 73 studies examined, two studies met all 11 U.S. EPA Study Acceptance Criteria, and were considered in the selection of a benchmark. A lack of adequate data precluded the derivation of a plant toxicity benchmark (U.S. EPA 2005h).

Based on Ontario specific guidance, a soil concentration of 200 mg/kg is used as the phytotoxicity benchmark for vanadium in the ecological model. This value is the MOE ecotoxicity criterion used towards selection of the MOE full depth generic site condition standard for non-potable groundwater conditions.

Zinc

The U.S. EPA has recently conducted an in-depth review of the toxicological literature pertaining to the effects of zinc on plants (U.S. EPA, 2007c). Of 680 studies examined, 78 studies met all 11 U.S. EPA Study Acceptance Criteria, and were considered in the selection of a benchmark. The U.S. EPA defines a zinc screening benchmark for plants of 160 mg/kg (U.S. EPA 2007c).

Based on Ontario specific guidance, a soil concentration of 600 mg/kg is used as the phytotoxicity benchmark for vanadium in the ecological model. This value is the MOE ecotoxicity criterion used towards selection of the MOE full depth generic site condition standard for non-potable groundwater conditions.

4.2 SOIL INVERTEBRATE BENCHMARKS

4.2.1 Polycyclic Aromatic Hydrocarbons

Acenaphthene

The U.S. EPA has recently conducted an in-depth review of the toxicological literature pertaining to the effects of pentachlorophenol on soil invertebrates (U.S. EPA, 2007d). Of 94 papers acquired for possible inclusion in the benchmark derivation process, 16 studies were considered in the selection of a benchmark for low molecular weight PAHs (LMH PAHs), and six for high molecular weight PAHs (HMW PAHs). Based on these studies, the U.S. EPA defines screening benchmarks of 29 mg/kg and 18 mg/kg for LMW PAHs and HMW PAHs, respectively. The Eco-SSLs are the geometric mean of the Maximum Allowable Toxicant Concentration (MATC) and EC_{10} (concentration impacting 10% of the study population) values for several test species (springtail, potworm, earthworms) under different test conditions (pH and % organic matter) (U.S. EPA 2007d).

The screening benchmark selected for use in the ecological model for acenaphthene is thus 29 mg/kg (U.S. EPA, 2007d).

Acenaphthylene

The screening benchmark selected for use in the ecological model for acenaphthylene is 29 mg/kg (U.S. EPA, 2007d).

Anthracene

A soil concentration of 40 mg/kg is used as the soil invertebrate benchmark for anthracene in the ecological model. This value is a Netherlands “C-value” and represents the ecotoxicity criterion used towards selection of the MOE full depth generic site condition standard for non-potable groundwater conditions.

Fluoranthene

A soil concentration of 40 mg/kg is used as the soil invertebrate A soil concentration of 40 mg/kg is used as the phytotoxicity benchmark for fluoranthene in the ecological model. This value is a Netherlands “C-value” and represents the ecotoxicity criterion used towards selection of the MOE full depth generic site condition standard for non-potable groundwater conditions. It is also the lowest of the health criteria available for fluoranthene, and is therefore selected as the MOE generic standard (for all land uses).

Fluorene

The screening benchmark selected for use in the ecological model for fluorene is 29 mg/kg (U.S. EPA, 2007d).

Phenanthrene

A soil concentration of 40 mg/kg is used as the soil invertebrate benchmark for phenanthrene in the ecological model. This value is a Netherlands “C-value” and represents the ecotoxicity criterion used towards selection of the MOE full depth generic site condition standard for non-potable groundwater conditions. It is also the lowest of the health criteria available for phenanthrene, and is therefore selected as the MOE generic standard (for all land uses).

Benzo(a)anthracene

A soil concentration of 40 mg/kg is used as the soil invertebrate benchmark for benzo(a)anthracene in the ecological model. This value is a Netherlands "C-value" and represents the ecotoxicity criterion used towards selection of the MOE full depth generic site condition standard for non-potable groundwater conditions. It is also the lowest of the health criteria available for benzo(a)anthracene, and is therefore selected as the MOE generic standard (for all land uses).

Benzo(a)pyrene

A soil concentration of 40 mg/kg is used as the soil invertebrate benchmark for benzo(a)pyrene in the ecological model. This value is a Netherlands "C-value" and represents the ecotoxicity criterion used towards selection of the MOE full depth generic site condition standard for non-potable groundwater conditions. The ecotoxicity component is the lowest of all components for benzo(a)pyrene, and is therefore also used as the generic standard.

Benzo(e)pyrene

The screening benchmark selected for use in the ecological model for benzo(e)pyrene is 18 mg/kg (U.S. EPA, 2007d).

Benzo(a)fluorene

The screening benchmark selected for use in the ecological model for benzo(a)fluorene is 18 mg/kg (U.S. EPA, 2007d).

Benzo(b)fluorene

The screening benchmark selected for use in the ecological model for benzo(b)fluorene is 18 mg/kg (U.S. EPA, 2007d).

Benzo(b)fluoranthene

The screening benchmark selected for use in the ecological model for benzo(b)fluoranthene is 18 mg/kg (U.S. EPA, 2007d).

Benzo(g,h,i)perylene

A soil concentration of 40 mg/kg is used as the soil invertebrate benchmark for benzo(g,h,i)perylene in the ecological model. This value is a Netherlands "C-value" and represents the ecotoxicity criterion used towards selection of the MOE full depth generic site condition standard for non-potable groundwater conditions. It is also the lowest of the health criteria available for benzo(g,h,i)perylene, and is therefore selected as the MOE generic standard (for all land uses).

Benzo(k)fluoranthene

A soil concentration of 40 mg/kg is used as the soil invertebrate benchmark for benzo(k)fluoranthene in the ecological model. This value is a Netherlands "C-value" and represents the ecotoxicity criterion used towards selection of the MOE full depth generic site condition standard for non-potable groundwater conditions.

Chrysene

A soil concentration of 40 mg/kg is used as the soil invertebrate benchmark for chrysene in the ecological model. This value is a Netherlands "C-value" and represents the ecotoxicity criterion used towards selection of the MOE full depth generic site condition standard for non-potable groundwater conditions.

Dibenz(a,c)anthracene

The screening benchmark selected for use in the ecological model for dibenz(a,c)anthracene is 18 mg/kg (U.S. EPA, 2007d).

Dibenz(a,h)anthracene

The screening benchmark selected for use in the ecological model for dibenz(a,h)anthracene is 18 mg/kg (U.S. EPA, 2007d).

Indeno(1,2,3-cd)pyrene

A soil concentration of 40 mg/kg is used as the soil invertebrate benchmark for indeno(1,2,3-cd)pyrene in the ecological model. This value is a Netherlands "C-value" and represents the ecotoxicity criterion used towards selection of the MOE full depth generic site condition standard for non-potable groundwater conditions. The ecotoxicity component is the lowest of all components for indeno(1,2,3-cd)pyrene, and is therefore also used as the generic standard.

Perylene

The screening benchmark selected for use in the ecological model for perylene is 18 mg/kg (U.S. EPA, 2007d).

Pyrene

The screening benchmark selected for use in the ecological model for pyrene is 18 mg/kg (U.S. EPA, 2007d).

4.2.2 Dioxins and Furans

The screening benchmark selected for use in the ecological model for dioxins and furans is 0.5 mg/kg, based on a chronic 85 day study performed by Reinecke and Nash, (1984, cited in U.S. EPA, 1999). No earthworm mortality was observed at the 5 mg/kg concentration. The U.S. EPA applied an uncertainty factor of 10 to this value because earthworm mortality was the only endpoint and no statistical analysis was performed on the data.

4.2.3 Total PCBs

The screening benchmark selected for use in the ecological model for dioxins and furans is 2.51 mg/kg, based on an acute median LC_{50} earthworm study performed by Rhett *et al.*, (1989, cited in U.S. EPA, 1999), where a value of 251 mg/kg was derived. The U.S. EPA applied an uncertainty factor of 100 to this value.

4.2.4 Other Organics

Dichlorobenzene

A soil concentration of 30 mg/kg is used as the soil invertebrate benchmark for dichlorobenzene in the ecological model. This value is a Netherlands "C-value" and represents the ecotoxicity criterion used

towards selection of the MOE full depth generic site condition standard for non-potable groundwater conditions.

1,2,4-Trichlorobenzene

A soil concentration of 30 mg/kg is used as the soil invertebrate benchmark for 1,2,4-trichlorobenzene in the ecological model. This value is a Netherlands "C-value" and represents the ecotoxicity criterion used towards selection of the MOE full depth generic site condition standard for non-potable groundwater conditions.

1,2,4,5-Tetrachlorobenzene

The soil invertebrate benchmark used in the ecological model to address exposure to 1,2,4,5-tetrachlorobenzene in soil is 10 mg/kg, based on a benchmark proposed for 1,2,3,4-tetrachlorobenzene. This benchmark was recommended by Efroymson *et al*. (1997b) based on five studies performed on two earthworm species by van Gestel *et al*. (1991). Of the five studies reviewed, the adverse effects concentrations (LC50) ranged from 75 mg/kg to 223 mg/kg soil. A safety factor of 5 was applied to the lowest LC50 value (75 mg/kg) to obtain the recommended benchmark value of 10 mg/kg soil. Confidence in this benchmark is low because of the paucity of data and because it is based on a lethal endpoint.

Pentachlorobenzene

The soil invertebrate benchmark used in the ecological model to address exposure to pentachlorobenzene in soil is 20 mg/kg. This benchmark was recommended by Efroymson *et al*. (1997b) based on four studies performed on two earthworm species by van Gestel *et al*. (1991). Of the four studies reviewed, the adverse effects concentrations (LC50) ranged from 72 mg/kg to 223 mg/kg soil. A safety factor of 5 was applied to the lowest reported LC50 value (115 mg/kg) to obtain the recommended benchmark value of 20 mg/kg soil. Confidence in this benchmark is low because of the paucity of data and because it is based on a lethal endpoint.

Hexachlorobenzene

A soil concentration of 30 mg/kg is used as the soil invertebrate benchmark for hexachlorobenzene in the ecological model. This value is a Netherlands "C-value" and represents the ecotoxicity criterion used towards selection of the MOE full depth generic site condition standard for non-potable groundwater conditions.

Pentachlorophenol

The U.S. EPA has recently conducted an in-depth review of the toxicological literature pertaining to the effects of pentachlorophenol on soil invertebrates (U.S. EPA, 2007e). Of 58 studies examined, 22 studies met all 11 U.S. EPA Study Acceptance Criteria, and were considered in the selection of a benchmark for pentachlorophenol. The U.S. EPA defines a pentachlorophenol screening benchmark for soil invertebrates of 31 mg/kg (U.S. EPA 2007e).

Based on Ontario specific guidance, a soil concentration of 5 mg/kg is used as the soil invertebrate benchmark for pentachlorophenol in the ecological model. This value is a Netherlands "C-value" and represents the ecotoxicity criterion used towards selection of the MOE full depth generic site condition standard for non-potable groundwater conditions.

Carbon Tetrachloride

There is limited data available addressing the effects of carbon tetrachloride exposure to soil invertebrates. The soil invertebrate toxicity benchmark for carbon tetrachloride is based on a microflora toxicity study by Walton *et al.* (1989; cited in Efroymson 1997b). Walton *et al.* (1989) observed a 21% decrease in respiration when exposed to 1000 mg/kg carbon tetrachloride in soil. This effect was observed in sandy loam soil, but not in silt loam. A total uncertainty factor of 100 was applied to this value for extrapolation from effects on microflora to that of terrestrial invertebrates. This 10 mg/kg soil concentration is used as the soil invertebrate toxicity benchmark for carbon tetrachloride.

Chloroform

An established soil invertebrate benchmark or suitable toxicity data could not be identified for chloroform in a review of scientific literature or current regulatory documents.

Dichloromethane

An established soil invertebrate benchmark or suitable toxicity data could not be identified for dichloromethane in a review of scientific literature or current regulatory documents.

Trichlorofluoromethane

An established soil invertebrate benchmark or suitable toxicity data could not be identified for trichlorofluoromethane in a review of scientific literature or current regulatory documents.

Trichloroethane, 1,1,1-

An established soil invertebrate benchmark or suitable toxicity data could not be identified for 1,1,1-trichloroethane in a review of scientific literature or current regulatory documents.

Bromoform (Tribromomethane)

An established soil invertebrate benchmark or suitable toxicity data could not be identified for bromoform in a review of scientific literature or current regulatory documents.

o-Terphenyl

An established soil invertebrate benchmark or suitable toxicity data could not be identified for o-terphenyl in a review of scientific literature or current regulatory documents.

4.2.5 Inorganics

Antimony

The U.S. EPA has recently conducted an in-depth review of the toxicological literature pertaining to the effects of antimony on soil invertebrates (U.S. EPA, 2005a). Of 7 studies examined, three study met all 11 U.S. EPA Study Acceptance Criteria, and was considered in the selection of a benchmark. The U.S. EPA defines an antimony screening benchmark for soil invertebrates of 78 mg/kg (U.S. EPA 2005a).

Based on Ontario specific guidance, the soil concentration used as the soil invertebrate benchmark in the ecological model for antimony is 20 mg/kg. This value represents the ecotoxicity criteria for the full depth generic site condition standard (non-potable groundwater condition) developed by the MOE for antimony (2004).

Arsenic

The U.S. EPA has recently conducted an in-depth review of the toxicological literature pertaining to the effects of arsenic on soil invertebrates (U.S. EPA, 2005b). Of 35 studies examined, only one met all 11 U.S. EPA Study Acceptance Criteria, for consideration in the selection of a benchmark. The U.S. EPA was unable to define an arsenic soil invertebrate screening value based on a lack of adequate data (U.S. EPA 2005b).

Based on Ontario specific guidance, the soil concentration used as the soil invertebrate benchmark in the ecological model for arsenic is 20 mg/kg. This value represents the ecotoxicity criteria for the full depth generic site condition standard (non-potable groundwater condition) developed by the MOE for arsenic (2004).

Barium

The U.S. EPA has recently conducted an in-depth review of the toxicological literature pertaining to the effects of barium on soil invertebrates (U.S. EPA, 2005c). Of 152 studies examined, only four studies met all 11 U.S. EPA Study Acceptance Criteria, and were considered in the selection of a benchmark. The U.S. EPA defines a barium screening benchmark for soil invertebrates of 330 mg/kg (U.S. EPA 2005c).

Based on Ontario specific guidance, a soil concentration of 750 mg/kg is used as the soil invertebrate benchmark for barium in the ecological model. This value represents the ecotoxicity criteria for the full depth generic site condition standard (non-potable groundwater condition) developed by the MOE for barium (2004). The ecotoxicity criteria is the lowest of all components for barium, and is therefore also used as the generic standard. The MOE adopted this value from the CCME, where 750 mg/kg is the interim soil remediation criteria value for agricultural land use (CCME, 2007).

Beryllium

The U.S. EPA has recently conducted an in-depth review of the toxicological literature pertaining to the effects of barium on soil invertebrates (U.S. EPA, 2005d). Of six studies examined, only three studies met all 11 U.S. EPA Study Acceptance Criteria, and were considered in the selection of a benchmark. The U.S. EPA defines a beryllium screening benchmark for soil invertebrates of 40 mg/kg (U.S. EPA 2005d).

Based on Ontario specific guidance, the soil concentration used as the soil invertebrate benchmark in the ecological model for beryllium is 4 mg/kg. This value represents the ecotoxicity criteria for the full depth generic site condition standard (non-potable groundwater condition) developed by the MOE for beryllium (2004).

Boron

A soil concentration of 30 mg/kg is used as the soil invertebrate benchmark for boron in the ecological model, and represents the 97.5 percentile of the sample population background concentration for Old Urban Parkland / Rural Parkland, as defined by the MOE (1993). This value was deemed applicable based on close corroboration with evidence provided in the MOEE 1996 rationale document, which states that generally less than 5% of total soil B is found in bioavailable forms (Gupta, 1979; in MOEE, 1996). It has been well established that bioavailability of boron is strongly associated with the hot-water soluble fraction, which usually ranges from 0.4 to 4.7% of total boron (Gupta and McLeod 1982). Because soil concentrations utilized in this assessment are based on total boron, and assuming that the 1.5 mg/kg MOE benchmark (MOE, 1996) for available boron accounts for 4.7% of the total soil

boron, a simple scaling up of the benchmark was performed to arrive at 32 mg/kg, which is very close to the 30 mg/kg benchmark chosen for this assessment.

Cadmium

The U.S. EPA has recently conducted an in-depth review of the toxicological literature pertaining to the effects of cadmium on soil invertebrates (U.S. EPA, 2005e). Of 239 studies examined, 32 studies met all 11 U.S. EPA Study Acceptance Criteria, and were considered in the selection of a benchmark. The U.S. EPA defines a cadmium screening benchmark for soil invertebrates of 140 mg/kg (U.S. EPA 2005e).

Based on Ontario specific guidance, a soil concentration of 12 mg/kg is used as the soil invertebrate benchmark for cadmium in the ecological model. This value is a Netherlands "C-value" and represents the ecotoxicity criterion used towards selection of the MOE full depth generic site condition standard for non-potable groundwater conditions (MOE, 2004).

Chromium III (Total)

The U.S. EPA has recently conducted an in-depth review of the toxicological literature pertaining to the effects of chromium (all forms) on soil invertebrates (U.S. EPA, 2008). Of 31 studies examined, 4 studies met all 11 U.S. EPA Study Acceptance Criteria, and were considered in the selection of a benchmark. A lack of adequate data precluded the derivation of a soil invertebrate toxicity benchmark (U.S. EPA 2008).

A soil concentration of 750 mg/kg is used as the soil invertebrate benchmark for total chromium in the ecological model. This value represents the ecotoxicity criteria for the full depth generic site condition standard (non-potable groundwater condition) developed by the MOE for chromium (2004). The ecotoxicity component is the lowest of all criteria for chromium, and is therefore also used as the generic standard. The MOE adopted this value from the CCME, where 750 mg/kg is the interim soil remediation criteria value for agricultural land use (CCME, 2007).

Chromium VI

A soil concentration of 8 mg/kg is used as the soil invertebrate benchmark for hexavalent chromium in the ecological model. This value represents the ecotoxicity criteria for the full depth generic site condition standard (non-potable groundwater condition) developed by the MOE for chromium (2004). The ecotoxicity component is the lowest of all criteria for hexavalent chromium, and is therefore also used as the generic standard. The MOE adopted this value from the CCME, where 8 mg/kg is the interim soil remediation criteria value for agricultural land use (CCME, 2007).

Cobalt

The U.S. EPA has recently conducted an in-depth review of the toxicological literature pertaining to the effects of cobalt on soil invertebrates (U.S. EPA, 2005f). Of 11 studies examined, no studies met all 11 U.S. EPA Study Acceptance Criteria, and were considered in the selection of a benchmark. The U.S. EPA was thus not able to define a soil invertebrate screening benchmark (U.S. EPA 2005f).

Based on Ontario specific guidance, the soil concentration used as the soil invertebrate benchmark in the ecological model for cobalt is 40 mg/kg. This value represents the ecotoxicity criteria for the full depth generic site condition standard (non-potable groundwater condition) developed by the MOE for cobalt (2004).

Lead

As part of the Eco-SSL literature search process, a total of 179 papers were selected for further review. Of those papers acquired, four were eligible for the Eco-SSL derivation. The Eco-SSL is the geometric mean of the MATC values for one test species (a springtail) under three different test conditions (pH) and is equal to 1,700 mg/kg dw; this value was used in the ecological model as the screening benchmark (U.S. EPA, 2005g).

Mercury (Total)

A soil concentration of 10 mg/kg is used as the soil invertebrate benchmark for total mercury in the ecological model. This value is a Netherlands "C-value" and represents the ecotoxicity criterion used towards selection of the MOE full depth generic site condition standard for non-potable groundwater conditions.

Methyl Mercury

A soil concentration of 10 mg/kg is used as the soil invertebrate benchmark for total mercury in the ecological model. This value is a Netherlands "C-value" and represents the ecotoxicity criterion used towards selection of the MOE full depth generic site condition standard for non-potable groundwater conditions.

Nickel

The U.S. EPA has recently conducted an in-depth review of the toxicological literature pertaining to the effects of nickel on soil invertebrates (U.S. EPA, 2007a). Of 46 studies examined, 9 studies met all 11 U.S. EPA Study Acceptance Criteria, and were considered in the selection of a benchmark. The U.S. EPA defines a nickel screening benchmark for soil invertebrates of 280 mg/kg (U.S. EPA 2007a).

Based on Ontario specific guidance, a soil concentration of 150 mg/kg is used as the soil invertebrate benchmark for nickel in the ecological model. This value represents the ecotoxicity criterion used towards selection of the MOE full depth generic site condition standard for non-potable groundwater conditions.

Phosphorus

An established soil invertebrate benchmark or suitable toxicity data could not be identified for phosphorus in a review of scientific literature or current regulatory documents.

Selenium

The U.S. EPA has recently conducted an in-depth review of the toxicological literature pertaining to the effects of selenium on soil invertebrates (U.S. EPA, 2007b). Of 33 studies examined, 11 studies met all 11 U.S. EPA Study Acceptance Criteria, and were considered in the selection of a benchmark. The U.S. EPA defines a selenium screening benchmark for soil invertebrates of 4.1 mg/kg (U.S. EPA 2007b).

Based on Ontario specific guidance, a soil concentration of 10 mg/kg is used as the soil invertebrate benchmark for selenium in the ecological model. This value represents the ecotoxicity criteria for the full depth generic site condition standard (non-potable groundwater condition) developed by the MOE for selenium (2004).

Silver

The U.S. EPA has recently conducted an in-depth review of the toxicological literature pertaining to the effects of silver on soil invertebrates (U.S. EPA, 2006). Of 61 studies examined, two studies met all 11 U.S. EPA Study Acceptance Criteria, and were considered in the selection of a benchmark. The U.S. EPA could not define a silver screening benchmark for soil invertebrates based on a lack of adequate data (U.S. EPA 2006).

Based on Ontario specific guidance, a soil concentration of 20 mg/kg is used as the soil invertebrate benchmark for silver in the ecological model. This value represents the ecotoxicity criteria for the full depth generic site condition standard (non-potable groundwater condition) developed by the MOE for silver (2004).

Thallium

An established soil invertebrate benchmark or suitable toxicity data could not be identified for thallium in a review of scientific literature or current regulatory documents.

Tin

There is limited data available addressing the effects of tin exposure to soil invertebrates. The soil invertebrate toxicity benchmark for tin is based on an microflora enzyme inhibition (arylsulfatase) study by Al-Khafaji and Tabatabai, 1979; cited in Efroymson 1997b. Al-Khafaji and Tabatabai (1979) observed a decrease in arylsulfatase activity when exposed to 2698 mg/kg tin in soil. Effects were less severe in soils with greater organic carbon and clay content. The soil concentration of 2000 mg/kg is used as the soil invertebrate toxicity benchmark for tin. Confidence in this benchmark is low due to the limited amount and type of data available.

Vanadium

The U.S. EPA has recently conducted an in-depth review of the toxicological literature pertaining to the effects of vanadium on soil invertebrates (U.S. EPA, 2005h). Of 6 studies examined, no studies met all 11 U.S. EPA Study Acceptance Criteria, required for consideration in the selection of a benchmark. A lack of adequate data precluded the derivation of a soil invertebrate toxicity benchmark (U.S. EPA 2005h).

Based on Ontario specific guidance, a soil concentration of 200 mg/kg is used as the soil invertebrate benchmark for vanadium in the ecological model. This value represents the ecotoxicity criteria for the full depth generic site condition standard (non-potable groundwater condition) developed by the MOE for vanadium (2004). The ecotoxicity criteria is the lowest of all components for vanadium, and is therefore also used as the generic standard. The MOE adopted this value from the CCME, where 200 mg/kg is the interim soil remediation criteria value for agricultural and residential/parkland land use (CCME, 2007).

Zinc

The U.S. EPA has recently conducted an in-depth review of the toxicological literature pertaining to the effects of zinc on soil invertebrates (U.S. EPA, 2007c). Of 162 studies examined, 26 studies met all 11 U.S. EPA Study Acceptance Criteria, and were considered in the selection of a benchmark. The U.S. EPA defines a zinc screening benchmark of 120 mg/kg (U.S. EPA 2007c).

Based on Ontario specific guidance, a soil concentration of 600 mg/kg is used as the soil invertebrate benchmark for zinc in the ecological model. This value represents the ecotoxicity criteria for the full

depth generic site condition standard (non-potable groundwater condition) developed by the MOE for zinc (2004).

4.3 AQUATIC TOXICITY BENCHMARKS

4.3.1 Organics with no Prescribed PWQO

Most of the organic COPC in the ecological model may be categorized as narcotic chemical based on their presumed mode of action. Type 1 narcotic chemicals are broadly defined as all non-ionic organic chemicals which exert toxic effects via narcosis, and includes PAHs and some other related compounds such as chlorinated solvents. Di Toro *et al.* (2000) provide a representative listing of chemicals included in an acute toxicity database for Type 1 narcotic chemicals. All of these compounds should be viewed as having additive toxicity (Bradbury *et al.* 1989; Hermens *et al.* 1989; Verhaar *et al.* 1992; Di Toro et al, 2000, Di Toro and McGrath, 2000; Di Toro *et al.* 2007).

The generalized toxicity model for Type 1 narcotic chemicals outlined by Di Toro and co-workers is defined as the Target Lipid Model (TLM) and can be applied to both water and sediments (using the equilibrium partitioning or EqP approach). The TLM has been validated by comparison to experimental results for crude oil and PAHs in water and sediment (Di Toro *et al.* 2000, 2007, Di Toro and McGrath, 2000) and gasoline in water (McGrath *et al.* 2005).

Toxicity of Type 1 Narcotic Chemicals in Water

The toxicity of Type 1 narcotic chemicals, which include PAHs and other chemicals, should be treated as additive (Bradbury *et al.* 1989; Hermens *et al.* 1989; Verhaar *et al.* 1992; Di Toro et al, 2000, Di Toro and McGrath, 2000; Di Toro *et al.* 2007). The following sections demonstrate how this additive toxicity can be evaluated for aquatic biota, based on the concept of Toxic Units (TU; Di Toro *et al.* 2007).

The relationship between the LC_{50} for Type 1 narcotic chemicals (mmol/L) and the K_{OW} value for fish is approximately (Di Toro *et al.* 2000 Eq. 1):

$$\log(LC_{50}) \approx - \log(K_{OW}) + 1.7 \quad .$$

From Di Toro *et al.* (2000 Eq. 3), the critical body burden corresponding to lethality is:

$$C^*_{Org} = BCF \times LC_{50} \quad .$$

The BCF varies with K_{OW}, so that (Di Toro *et al.* 2000 Eq. 4):

$$\log(BCF) = \log(K_{OW}) - 1.3 \quad .$$

Therefore, the critical body burden corresponding to the LC_{50} can be calculated by combining the toxicity and the bioaccumulation equations as:

$$\begin{aligned} \log(C^*_{Org}) &= \log(BCF) + \log(LC_{50}) \\ &\approx \log(K_{OW}) - 1.3 - \log(K_{OW}) + 1.7 \\ &\approx 0.4 \quad . \end{aligned}$$

Or,

$C^*_{Org} \approx 2.5$ mmol/kg wet weight (Di Toro *et al.* 2000 Eq. 6).

Assuming that the lipid fraction of fish and invertebrates is approximately 5%, then:

C^*_{Org} = 50 mmol/kg lipid (which can also be defined as C^*_L)

Note that although initially based on fish, this is, in principle, a universal concentration, applicable to all aquatic biota, and broadly validated for protozoa, coelenterates, polychaetes, crustaceans, mollusks, insects, fish and amphibians for a broad range of Type 1 narcotic chemicals (Di Toro *et al.* 2000).

From this concentration, and from considerations of multi-component organic compound solubility in water and sediment pore water, it is possible to derive models that can account for the toxicity of hydrocarbon mixtures in water and sediment, for a broad range of aquatic biota. This is done and validated by Di Toro *et al.* (2000), Di Toro and McGrath (2000), McGrath *et al.* (2005) and Di Toro *et al.* (2007), and is identified as the Target Lipid Model (TLM).

Di Toro *et al.* (2007 Eq. 1) note that:

$$\log(LC_{50}) = m \log(K_{OW}) + b \quad ,$$

where *m* has the value of ≈ -1 and *b* has the value of ≈ 1.7 for fish. It has been further shown that the slope (m) of approximately -1 (actually defined as -0.945 ± 0.014 by Di Toro *et al.* 2000) is universal across K_{OW} values (*i.e.,* for a broad range of narcotic chemicals) and independent of organism identity, and that the intercept (*b*), which varies according to individual species sensitivity and toxic endpoint, can be interpreted as the lipid-normalized critical body burden, C^*_L, that corresponds to an observed endpoint such as 50% mortality or LC_{50} (Di Toro *et al.* 2007).

Families of lines can be defined, each having the same slope (-0.945), but having different intercepts, to represent different endpoints (such as acute or chronic lethality) for different organisms. Di Toro *et al.* (2007) evaluate a variety of different species in order to define the 5th percentile of sensitivity (as required by U.S. EPA, and as recently recommended by CCME in estimating water quality criteria). Based upon multi-species evaluation (including protozoa, coelenterates, polychaetes, crustaceans, mollusks, insects, fish and amphibians), critical lipid concentrations for the more sensitive (5th percentile) receptors are defined as follows:

$C^*_{L,\ FAV}$ = 35.3 mmol/kg lipid, where FAV indicates Final Acute Value; and

$C^*_{L,\ FCV}$ = 6.94 mmol/kg lipid, where FCV indicates Final Chronic Value, and an acute:chronic ratio (ACR) of 5.09 is assumed.

Note, however, that the analysis (Di Toro *et al.* 2000) also showed that some classes of narcotic chemicals are systematically considered to be more toxically potent than others, and correction factors are introduced to the "universal" values given above as follows (Table 2):

Table 2: Chemical Class Correction Factors (relative to baseline narcotics), and Critical Tissue Concentrations for Sensitive (5th percentile) Aquatic Biota

Baseline	Halogenated Baseline	Ketones	Halogenated Ketones	PAHs	Halogenated PAHs
Chemical Class Correction Factor (Di Toro et al. 2000)					
1.000	0.570	0.569	0.324	0.546	0.311
Log Chemical Class Correction Factor (Di Toro et al. 2000)					
0	-0.244	-0.245	-0.489	-0.263	-0.507
Final Acute Value (acute effects - lethality), mmol/kg lipid					
35.3	20.1	20.1	11.4	19.3	11.0
Final Chronic Value (LOAEL), mmol/kg lipid					
6.94	3.96	3.95	2.25	3.79	2.16

Notes: baseline narcotic chemicals include aliphatics, ethers, alcohols, and most aromatics (*i.e.,* the conventional BTEX and TPH substances). Acute to Chronic Conversion Assumes ACR = 5.09.

Evaluating Aqueous Toxicity

For each substance (j), the critical water concentration at which an adverse effect will occur is:

$$\log(C^*_{W,j}) = -0.945 \log(K_{OW,j}) + (\log(C^*_L) + \Delta c_j) ,$$

where K_{OW},j is the octanol-water partition coefficient for substance j, and the term $(\log(C^*_L) + \Delta c_j)$ is the critical effects concentration in the organism lipid fraction (mmol/kg lipid) from Table 1, here taken to be based on the Final Chronic Value for baseline narcotics (*i.e.,* 6.94 mmol/kg lipid baseline narcotics). This value is the 5th percentile in a species sensitivity distribution (SSD), and can therefore be considered generally protective of aquatic resources, in a manner consistent with Provincial Water and Sediment Quality Guidelines for the protection of aquatic life. Note that in the equation above, the relevant non-logged values are:

$$C^*_{L,\,FAV} = 35.3 \text{ mmol/kg lipid}$$

$$C^*_{L,\,FCV} = 35.3 \div 5.09 = 6.94 \text{ mmol/kg lipid.}$$

In log notation, $C^*_{L,\,FCV}$ becomes:

$$\log(C^*_{L,\,FCV}) = 1.548 - 0.707 = 0.841, \text{ and FCV} = 10^{(0.841)} = 6.94 \text{ mmol/kg lipid.}$$

Note that the correction term, Δc_j, has values that range from zero (in log units; log(1) = 0) for baseline narcotic chemicals, to -0.507 for halogenated PAHs. However, the most commonly applied correction factor will be $\Delta c_j = \log(0.546) = 0.263$, for PAHs.

Therefore, the critical water concentrations for acute exposures of sensitive species are based on the following equations:

$\log(C^*_{W,j}) = -0.945 \log(K_{OW,j}) + 0.578$, for PAHs; and
$\log(C^*_{W,j}) = -0.945 \log(K_{OW,j}) + 0.597$, for chlorinated solvents in this assessment.

4.3.2 Organics with a Prescribed PWQO

When a PWQO existed for a given organic compound, this value was preferentially used over the TLN Model described above. Where both an original and revised PWQO existed, the revised value was taken as the benchmark (as is also the case for inorganics).

Anthracene

The total anthracene reference benchmark of 8.0×10^{-7} mg/L for freshwater receptors, is based on a generic wildlife protection value of 8.0×10^{-4} µg/L (MOE, 1999). This threshold it is given a moderate level of confidence.

Fluoranthene

The total fluoranthene reference benchmark of 8.0×10^{-7} mg/L for freshwater receptors, is based on a generic wildlife protection value of 8.0×10^{-4} µg/L (MOE, 1999). This threshold it is given a moderate level of confidence.

Fluorene

The total fluorene reference benchmark of $2.0x10^{-4}$ mg/L for freshwater receptors, is based on a generic wildlife protection value of 0.2 µg/L (MOE, 1999). This threshold it is given a moderate level of confidence.

Phenanthrene

The total phenanthrene reference benchmark of $3.0x10^{-5}$ mg/L for freshwater receptors, is based on a generic wildlife protection value of 0.3 µg/L (MOE, 1999). This threshold it is given a moderate level of confidence.

Benz(a)anthracene

The total benz(a)anthracene reference benchmark of $8.0x10^{-7}$ mg/L for freshwater receptors, is based on a generic wildlife protection value of $8.0x10^{-4}$ µg/L (MOE, 1999). This threshold it is given a moderate level of confidence.

Benzo(g,h,i)perylene

The total benzo(g,h,i)anthracene reference benchmark of $2.0x10^{-8}$ mg/L for freshwater receptors, is based on a generic wildlife protection value of $2.0x10^{-5}$ µg/L (MOE, 1999). This threshold it is given a moderate level of confidence.

Benzo(k)fluoranthene

The total benzo(k)fluoranthene reference benchmark of $2.0x10^{-7}$ mg/L for freshwater receptors, is based on a generic wildlife protection value of $2.0x10^{-4}$ µg/L (MOE, 1999). This threshold it is given a moderate level of confidence.

Chrysene

The total chrysene reference benchmark of $1.0x10^{-7}$ mg/L for freshwater receptors, is based on a generic wildlife protection value of $1.0x10^{-4}$ µg/L (MOE, 1999). This threshold it is given a moderate level of confidence.

Dibenz(a,h)anthracene

The total dibenz(a,h)anthracene reference benchmark of $2.0x10^{-6}$ mg/L for freshwater receptors, is based on a generic wildlife protection value of $2.0x10^{-3}$ µg/L (MOE, 1999). This threshold it is given a moderate level of confidence.

1,2-Dichlorobenzene

The total 1,2-dichlorobenzene reference benchmark of $2.5x10^{-3}$ mg/L for freshwater receptors, is based on a generic wildlife protection value of 2.5 µg/L (MOE, 1999). This threshold it is given a moderate level of confidence.

1,2,4,5-tetrachlorobenzene

The total 1,2,4,5-tetrachlorobenzene reference benchmark of $1.5x10^{-4}$ mg/L for freshwater receptors, is based on a generic wildlife protection value of 0.15 µg/L (MOE, 1999). This threshold it is given a moderate level of confidence.

Hexachlorobenzene

The total hexachlorobenzene reference benchmark of $6.5x10^{-6}$ mg/L for freshwater receptors, is based on a generic wildlife protection value of $6.5x10^{-3}$ µg/L (MOE, 1999). This threshold it is given a moderate level of confidence.

Pentachlorophenol

The total pentachlorophenol reference benchmark of $5.0x10^{-4}$ mg/L for freshwater receptors, is based on a generic wildlife protection value of 0.5 µg/L (MOE, 1999). This threshold it is given a moderate level of confidence.

1,1,1-Trichloroethane

The total 1,1,1-trichloroethane reference benchmark of $1.0x10^{-2}$ mg/L for freshwater receptors, is based on a generic wildlife protection value of 10 µg/L (MOE, 1999). This threshold it is given a moderate level of confidence.

Bromoform

The total bromoform reference benchmark of 0.06 mg/L for freshwater receptors, is based on a generic wildlife protection value of 60 µg/L (MOE, 1999). This threshold it is given a moderate level of confidence.

4.3.3 Dioxins and Furans

The 2,3,7,8-TCDD Equivalent reference benchmark of $1.0x10^{-8}$ mg/L for freshwater receptors (Grimwood and Dobbs 1995) is based on the NOEL value from exposure of early life stage rainbow trout in an experimental setting to 2,3,7,8-TCDD and a literature review of the ecotoxicology of dioxins and furans. This threshold it is given a moderate level of confidence.

4.3.4 Total PCBs

The total PCBs reference benchmark of $1.0x10^{-6}$ mg/L for freshwater receptors, is based on a generic wildlife protection value of $1.0x10^{-3}$ µg/L (MOE, 1999), considering the bioaccumulative nature of PCBs through the trophic web and toxicity to a wide variety of organisms (molluscs, fish, invertebrates). This threshold it is given a moderate level of confidence.

4.3.5 Inorganics

Antimony

The antimony reference benchmark of 0.02 mg/L for freshwater receptors, is based on a generic wildlife protection value of 20 µg/L (MOE, 1999). This threshold it is given a moderate level of confidence.

Arsenic

The arsenic reference benchmark of 0.005 mg/L for freshwater receptors, is based on a generic wildlife protection value of 5 µg/L (MOE, 1999). This threshold it is given a moderate level of confidence.

Barium

The aquatic toxicity benchmark used in the ecological model for barium is 0.22 mg/L. This value is a Maximum Permissible Concentrations (MPC) derived by Crommentuijn *et al.* (1997) for the Netherlands National Institute of Public Health and the Environment. The MPC is calculated by adding a background surface water concentration to the estimated Maximum Permissible Addition (MPA). The

MPA is the maximum concentration that can be added to the background, while maintaining a desired level of protection (depending on availability of toxicity data). The MPA of 0.15 mg/L was derived using the modified EPA-method by applying a factor of 100 on an LC50 for *Daphnia magna.* The MPA value was then added to an estimated Netherlands background surface water concentration of 0.073 mg/L (dissolved) barium, to obtain the 0.22 mg/L MPC value used in the ecological model.

Beryllium

The beryllium reference benchmark of 0.011 mg/L for freshwater receptors, is based on a generic wildlife protection value of 11 µg/L (MOE, 1999). This threshold it is given a moderate level of confidence.

Boron

The boron reference benchmark of 0.2 mg/L for freshwater receptors, is based on a generic wildlife protection value of 200 µg/L (MOE, 1999). This threshold it is given a moderate level of confidence.

Cadmium

The cadmium reference benchmark of $5x10^{-4}$ mg/L for freshwater receptors, is based on a generic wildlife protection value of 0.5 µg/L (MOE, 1999). This threshold it is given a moderate level of confidence.

Chromium III (Total)

The total chromium reference benchmark of 0.0089 mg/L for freshwater receptors, is based on a generic wildlife protection value of 8.9 µg/L (MOE, 1999). This threshold it is given a moderate level of confidence.

Chromium VI

The chromium VI reference benchmark of 0.001 mg/L for freshwater receptors, is based on a generic wildlife protection value of 1 µg/L (MOE, 1999). This threshold it is given a moderate level of confidence.

Cobalt

The cobalt reference benchmark of 0.0009 mg/L for freshwater receptors, is based on a generic wildlife protection value of 0.9 µg/L (MOE, 1999). This threshold it is given a moderate level of confidence.

Lead

The lead reference benchmark of 0.005 mg/L for freshwater receptors, is based on a generic wildlife protection value of 5 µg/L (MOE, 1999). This threshold it is given a moderate level of confidence.

Mercury –Inorganic

The inorganic mercury reference benchmark of $2x10^{-4}$ mg/L for freshwater receptors, is based on a generic wildlife protection value of 0.2 µg/L (MOE, 1999). This threshold it is given a moderate level of confidence.

Methyl Mercury

The aquatic toxicity benchmark used in the ecological model for methyl mercury is 0.00002 mg/L. This value is a MPC derived by Crommentuijn *et al.* (1997) for the Netherlands National Institute of Public

Health and the Environment. The MPC is calculated by adding a background surface water concentration to the estimated MPA. The MPA is the maximum concentration that can be added to the background, while maintaining a desired level of protection (depending on availability of toxicity data). For methyl mercury, chronic aquatic toxicity (NOEC) data was available from more than four different aquatic taxa, which permitted the construction of the species sensitivity distribution. From this distribution, the MPA was estimated based on protection of 95% of aquatic species (0.00001 mg/L), and assuming 0% bioavailability of the background concentration. The MPA value was then added to an estimated Netherlands background surface water concentration of 0.00001 mg/L (dissolved) mercury, to obtain the 0.00002 mg/L MPC value used in the ecological model.

Nickel

The nickel reference benchmark of 0.025 mg/L for freshwater receptors, is based on a generic wildlife protection value of 25 µg/L (MOE, 1999). This threshold it is given a moderate level of confidence.

Phosphorus

The phosphorus reference benchmark of 0.03 mg/L for freshwater receptors, is based on a generic wildlife protection value (MOE, 1999) which is protective of excessive macrophyte growth in rivers and streams. Excessive macrophyte growth is shown to deplete dissolved oxygen and degrade overall habitat quality for fish and other aquatic receptors. This threshold it is given a moderate level of confidence.

Selenium

The selenium reference benchmark of 0.1 mg/L for freshwater receptors, is based on a generic wildlife protection value of 100 µg/L (MOE, 1999). This threshold it is given a moderate level of confidence.

Silver

The silver reference benchmark of $1x10^{-4}$ mg/L for freshwater receptors, is based on a generic wildlife protection value of 0.1 µg/L (MOE, 1999). This threshold it is given a moderate level of confidence.

Thallium

The silver reference benchmark of $3x10^{-4}$ mg/L for freshwater receptors, is based on a generic wildlife protection value of 0.3 µg/L (MOE, 1999). This threshold it is given a moderate level of confidence

Tin (Inorganic)

The aquatic toxicity benchmark used in the ecological model for tin is 0.18 mg/L, published by the U.S. EPA (Region 5, 2003).

Vanadium

The vanadium reference benchmark of 0.006 mg/L for freshwater receptors, is based on a generic wildlife protection value of 60 µg/L (MOE, 1999). This threshold it is given a moderate level of confidence

Zinc

The zinc reference benchmark of 0.02 mg/L for freshwater receptors, is based on a generic wildlife protection value of 20 µg/L (MOE, 1999). This threshold it is given a moderate level of confidence.

4.4 SEDIMENT TOXICITY BENCHMARKS

4.4.1 Organics with no Prescribed SQG

Refer to aquatic benchmarks section for a more complete discussion of the Target Lipid Model, and the approach used to evaluate the toxicity of organic COPC to freshwater and sediment dwelling organisms. This method of benchmark derivation was used only when an applicable Sediment Quality Guideline was not available.

Toxicity of Type 1 Narcotic Chemicals in Sediment

The evaluation of organic COPC to sediment dwelling organisms follows a similar approach to that applied for other freshwater receptors (Di Toro and McGrath, 2000), since it is the concentration of organic substances in sediment pore water that is considered to be available and potentially toxic to aquatic life (*i.e.,* the equilibrium partitioning or EqP approach). Sediment narcotic chemical concentrations are based on organic carbon (OC) normalization, where typical sediment organic carbon fractions may range from 0.001 to 0.05 (and can be defined by the user, provided data are available to support a specific selection), with a default value of 0.01 (1% organic carbon).

Two simple relationships can be exploited as follows. First (Di Toro and McGrath 2000 Eq. 6 and 7),

$$C_{S,OC} = K_{OC} \times C_W \text{ , where } \log(K_{OC}) = 0.00028 + 0.983 \log(KOW) \text{ .}$$

Second (Di Toro and McGrath 2000, Eq. 9 and 10),

$$\log(C_{SQG}) = \log(K_{OC}) + \log(FCV) \text{ , where } \log(FCV) = \log(C^*_L) - 0.945 \log(K_{OW}) \text{ .}$$

Then by substitution and simplification (Di Toro and McGrath 2000 Eq. 12):

$$\log(C_{SQG}) = 0.00028 + \log(C^*_L) + 0.038 \log(K_{OW})$$

This is the desired equation which provides a critical sediment concentration (C_{SQG}, mmol/kg OC), based on C^*_L (as for water above, final acute and chronic values are 35.5 and 6.94 mmol/kg lipid for baseline hydrocarbons; with adjustments to reflect higher toxicity of other chemical groups, such as PAHs); and K_{OW} (L/kg, a fundamental property of each substance).

Toxic unit concentrations in sediment can then be defined, much as they are for water:

$$TU_{S,j} = C_{S,OC,j} / C_{SQG,j} \text{ , and } TU = \Sigma TU_j \text{ ,}$$

where $TU_{S,j}$ is the toxic units in sediment from substance j; $C_{S,OC,j}$ is the OC normalized sediment concentration of substance j, and $C_{SQG,j}$ is the critical effect concentration or sediment quality guideline for substance j. Then the total sediment TU present can be estimated as the sum of the individual substance TU.

4.4.2 Organics with a Prescribed SQG

Anthracene

The sediment toxicity benchmark used in the ecological model for anthracene is 0.22 mg/kg. This value is obtained from the Ontario Ministry of the Environment's *Guidelines for Identifying, Assessing, and Managing Sediments in Ontario* (MOE, 2008). This concentration is assumed to be protective over time to all benthic receptors, and is used with a medium level of confidence.

Fluoranthene

The sediment toxicity benchmark used in the ecological model for fluoranthene is 0.75 mg/kg. This value is obtained from the Ontario Ministry of the Environment's *Guidelines for Identifying, Assessing, and Managing Sediments in Ontario* (MOE, 2008). This concentration is assumed to be protective over time to all benthic receptors, and is used with a medium level of confidence.

Fluorene

The sediment toxicity benchmark used in the ecological model for fluorene is 0.19 mg/kg. This value is obtained from the Ontario Ministry of the Environment's Ontario Ministry of the Environment's *Guidelines for Identifying, Assessing, and Managing Sediments in Ontario* (MOE, 2008). This concentration is assumed to be protective over time to all benthic receptors, and is used with a medium level of confidence.

Phenanthrene

The sediment toxicity benchmark used in the ecological model for phenanthrene is 0.56 mg/kg. This value is obtained from the Ontario Ministry of the Environment's Ontario Ministry of the Environment's *Guidelines for Identifying, Assessing, and Managing Sediments in Ontario* (MOE, 2008). This concentration is assumed to be protective over time to all benthic receptors, and is used with a medium level of confidence.

Benz(a)anthracene

The sediment toxicity benchmark used in the ecological model for benz(a)anthracene is 0.32 mg/kg. This value is obtained from the Ontario Ministry of the Environment's Ontario Ministry of the Environment's *Guidelines for Identifying, Assessing, and Managing Sediments in Ontario* (MOE, 2008). This concentration is assumed to be protective over time to all benthic receptors, and is used with a medium level of confidence.

Benzo(a)pyrene

The sediment toxicity benchmark used in the ecological model for benzo(a)pyrene is 0.37 mg/kg. This value is obtained from the Ontario Ministry of the Environment's Ontario Ministry of the Environment's *Guidelines for Identifying, Assessing, and Managing Sediments in Ontario* (MOE, 2008). This concentration is assumed to be protective over time to all benthic receptors, and is used with a medium level of confidence.

Benzo(g,h,i)perylene

The sediment toxicity benchmark used in the ecological model for benzo(g,h,i)perylene is 0.17 mg/kg. This value is obtained from the Ontario Ministry of the Environment's Ontario Ministry of the Environment's *Guidelines for Identifying, Assessing, and Managing Sediments in Ontario* (MOE, 2008). This concentration is assumed to be protective over time to all benthic receptors, and is used with a medium level of confidence.

Benzo(k)fluoranthene

The sediment toxicity benchmark used in the ecological model for benzo(k)fluoranthene is 0.24 mg/kg. This value is obtained from the Ontario Ministry of the Environment's Ontario Ministry of the Environment's *Guidelines for Identifying, Assessing, and Managing Sediments in Ontario* (MOE, 2008).

This concentration is assumed to be protective over time to all benthic receptors, and is used with a medium level of confidence.

Chrysene

The sediment toxicity benchmark used in the ecological model for chrysene is 0.34 mg/kg. This value is obtained from the Ontario Ministry of the Environment's Ontario Ministry of the Environment's *Guidelines for Identifying, Assessing, and Managing Sediments in Ontario* (MOE, 2008). This concentration is assumed to be protective over time to all benthic receptors, and is used with a medium level of confidence.

Dibenz(a,h)anthracene

The sediment toxicity benchmark used in the ecological model for dibenzo(a,h)anthracene is 0.06 mg/kg. This value is obtained from the Ontario Ministry of the Environment's Ontario Ministry of the Environment's *Guidelines for Identifying, Assessing, and Managing Sediments in Ontario* (MOE, 2008). This concentration is assumed to be protective over time to all benthic receptors, and is used with a medium level of confidence.

Indeno(1,2,3-cd)pyrene

The sediment toxicity benchmark used in the ecological model for indeno(1,2,3-cd)pyrene is 0.2 mg/kg. This value is obtained from the Ontario Ministry of the Environment's Ontario Ministry of the Environment's *Guidelines for Identifying, Assessing, and Managing Sediments in Ontario* (MOE, 2008). This concentration is assumed to be protective over time to all benthic receptors, and is used with a medium level of confidence.

Pyrene

The sediment toxicity benchmark used in the ecological model for pyrene is 0.49 mg/kg. This value is obtained from the Ontario Ministry of the Environment's Ontario Ministry of the Environment's *Guidelines for Identifying, Assessing, and Managing Sediments in Ontario* (MOE, 2008). This concentration is assumed to be protective over time to all benthic receptors, and is used with a medium level of confidence.

4.4.3 Dioxins and Furans

The 2,3,7,8-TCDD Equivalent reference benchmark for freshwater sediment community receptors is based on the NOEC concentration of 0.025 mg/kg for survival and growth of amphipods (Barber *et al.* 1998). This benchmark value is given a moderate level of confidence.

4.4.4 Total PCBs

The sediment toxicity benchmark used in the ecological model for total PCBs is 0.07 mg/kg. This value is obtained from the Ontario Ministry of the Environment's Ontario Ministry of the Environment's *Guidelines for Identifying, Assessing, and Managing Sediments in Ontario* (MOE, 2008). This concentration is assumed to be protective over time to all benthic receptors, and is used with a medium level of confidence.

4.4.5 Inorganics

Unless otherwise noted inorganic contaminant sediment TRV benchmarks were based on "Lowest Effects Level" guidelines from the Ontario Ministry of the Environment's Ontario Ministry of the Environment's *Guidelines for Identifying, Assessing, and Managing Sediments in Ontario* (MOE, 2008). Otherwise, guidelines were preferentially chosen from the National Institute for Public Health and the Environment, The Netherlands (hereafter, RIVM). The RIVM guidelines were preferred for use as sediment TRVs over the CCME (2003) sediment quality guidelines because of criticism expressed about the approach (co-occurrence) used to derive the CCME benchmarks (Von Stackelberg and Menzie 2002, Borgmann 2003). The RIVM guidelines are based on aquatic ecotoxicological data and equilibrium partitioning theory (Lijzen *et al.* 2001), an approach also recommended by others (Leung *et al.* 2005).

Among the RIVM guidelines, SRAeco (Serious Risk Addition) values adjusted by a factor of 10 were preferred over MPA (Maximum Permissible Addition) values. MPA values are based on the 5th percentile of the reported chronic NOAEL distribution of a substance for a diversity of test species. This, according to Struijs *et al.* (1997), is assumed to be protective of 95% of species. However, the MPA value is considered overly conservative as a benchmark because it uses the 5% percentile of the NOAEL and not of LOAEL values, and in many cases approaches or is below background concentrations (Struijs *et al.* 1997).

If chronic NOAEL data for 4 or more taxonomic groups were available, then the SRAeco was based on the geometric mean of the NOAEL distribution. If chronic NOAEL data for fewer than 4 taxonomic groups were available, then the SRAeco values are based on the lower of the following two values: the geometric mean of the acute toxicity (LC50) values divided by a correction factor of 10; and the geometric mean of the NOAEL values. Therefore, the SRAeco value is no higher than the geometric mean of the NOAEL distribution. Following the standard protocol for addressing uncertainty in toxicity benchmarks (Faustman and Omenn, 1996; Lijzen *et al.*, 2001), an uncertainty factor of 10 was further assessed against the SRAeco value before adding this to the background value (Cb, Struijs *et al.* 1997), when available, to determine a chronic exposure benchmarks used in the HHERA. These sediment benchmarks are expected to provide a reasonable level of protection for freshwater benthic organisms.

Antimony

The sediment toxicity benchmark used in the ecological model for antimony is 4303 mg/kg. This value is the sum of $1/10^{th}$ the SRAeco value of 43000 mg/kg and the background concentration (Cb) value of 3 mg/kg obtained from van Vlaardingen *et al.* (2005) for the Netherlands National Institute of Public Health and the Environment. The SRA (Serious Risk Addition) values are based on a minimum of toxicity data for four taxa and are statistically derived as the geometric mean of effects levels. The SRAeco is the Serious Risk Addition for ecological receptors and the lowest of the SRAacute and SRAchronic values. To obtain a chronic LOAEL equivalent, the SRAeco was then divided by 10. Toxicity data for three taxa were found, the SRAeco is calculated as the geometric mean of the chronic data.

Arsenic

The sediment toxicity benchmark used in the ecological model for arsenic is 6 mg/kg. This value is obtained from the Ontario Ministry of the Environment's Ontario Ministry of the Environment's *Guidelines for Identifying, Assessing, and Managing Sediments in Ontario* (MOE, 2008). This

concentration is assumed to be protective over time to all benthic receptors, and is used with a medium level of confidence.

Barium

The sediment toxicity benchmark used in the ecological model for barium is 2455 mg/kg. This value is the sum of 1/10th the SRAeco value of 23000 mg/kg and the background concentration (Cb) value of 155 mg/kg obtained from van Vlaardingen *et al.* (2005) for the Netherlands National Institute of Public Health and the Environment. The SRA (Serious Risk Addition) values are based on a minimum of toxicity data for four taxa and are statistically derived as the geometric mean of effects levels. The SRAeco is the Serious Risk Addition for ecological receptors and the lowest of the SRAacute and SRAchronic values. To obtain a chronic LOAEL equivalent, the SRAeco was then divided by 10.

Beryllium

The sediment toxicity benchmark used in the ecological model for beryllium is 5.4 mg/kg. This value is the sum of 1/10th the SRAeco value of 42 mg/kg and the background concentration (Cb) value of 1.1 mg/kg obtained from van Vlaardingen *et al.* (2005) for the Netherlands National Institute of Public Health and the Environment. The SRA (Serious Risk Addition) values are based on a minimum of toxicity data for four taxa and are statistically derived as the geometric mean of effects levels. The SRAeco is the Serious Risk Addition for ecological receptors and the lowest of the SRAacute and SRAchronic values. To obtain a chronic LOAEL equivalent, the SRAeco was then divided by 10.

Boron

An established sediment toxicity benchmark or suitable toxicity data could not be identified for boron in a review of scientific literature or current regulatory documents.

Cadmium

The sediment toxicity benchmark used in the ecological model for cadmium is 0.6 mg/kg. This value is obtained from the Ontario Ministry of the Environment's Ontario Ministry of the Environment's *Guidelines for Identifying, Assessing, and Managing Sediments in Ontario* (MOE, 2008). This concentration is assumed to be protective over time to all benthic receptors, and is used with a medium level of confidence.

Chromium III (Total)

The sediment toxicity benchmark used in the ecological model for total chromium is 26 mg/kg. This value is obtained from the Ontario Ministry of the Environment's Ontario Ministry of the Environment's *Guidelines for Identifying, Assessing, and Managing Sediments in Ontario* (MOE, 2008). This concentration is assumed to be protective over time to all benthic receptors, and is used with a medium level of confidence.

Chromium VI

The sediment toxicity benchmark used in the ecological model for chromium (VI) is 4004 mg/kg. This value was derived from chronic (28 d) exposure of *Hyalella azteca* to spiked sediment producing with a LOEL survival endpoint (Besser *et al.* 2004). This concentration was based on the Columbia Wetlands (CW) sediment, which had an intermediate total organic carbon content of approximately 2%. In this sediment, the benchmark (94 umol/g = 4794 mg/kg) had a 79% 28 d survival compared to 91 to 95%

for lower concentrations and controls. As *Hyalella azteca* is considered a sensitive benthic species, the benchmark of 4004 mg/kg Cr (VI) is expected to provide protection for freshwater benthic species.

Cobalt

Minor modifications were made to portions of the previous MOE sediment guidance document to either update sections with current information (*e.g.*, update the section on legislation to replace Open Water Disposal Guidelines with Lakefill Guidelines). Previous open water disposal guidelines listed cobalt, with a benchmark of 50 mg/kg. According to more recent guidance by the MOE (2003) document, cobalt lakefill guidelines are not available for unconfined fill into open waters, (although a value is available for confined fill (*i.e.* within retaining walls, etc.), and so an Ontario specific value sediment value could not be used.

Therefore, the sediment toxicity benchmark used in the ecological model for cobalt is 459 mg/kg. This value is the sum of $1/10^{th}$ the SRAeco value of 4,500 mg/kg and the background concentration (Cb) value of 9 mg/kg obtained from van Vlaardingen *et al.* (2005) for the Netherlands National Institute of Public Health and the Environment. The SRA (Serious Risk Addition) values are based on a minimum of toxicity data for four taxa and are statistically derived as the geometric mean of effects levels. The SRAeco is the Serious Risk Addition for ecological receptors and the lowest of the SRAacute and SRAchronic values. To obtain a chronic LOAEL equivalent, the SRAeco was then divided by 10.

Lead

The sediment toxicity benchmark used in the ecological model for lead is 31 mg/kg. This value is obtained from the Ontario Ministry of the Environment's Ontario Ministry of the Environment's *Guidelines for Identifying, Assessing, and Managing Sediments in Ontario* (MOE, 2008). This concentration is assumed to be protective over time to all benthic receptors, and is used with a medium level of confidence.

Mercury (Inorganic)

The sediment toxicity benchmark used in the ecological model for inorganic mercury is 0.2 mg/kg. This value is obtained from the Ontario Ministry of the Environment's Ontario Ministry of the Environment's *Guidelines for Identifying, Assessing, and Managing Sediments in Ontario* (MOE, 2008). This concentration is assumed to be protective over time to all benthic receptors, and is used with a medium level of confidence.

Methyl mercury

The sediment toxicity benchmark used in the ecological model for methylmercury is 1.4 mg/kg. This value is a MPC derived by Crommentuijn *et al.* (1997) for the Netherlands National Institute of Public Health and the Environment. The MPC is calculated by adding a background surface water concentration to the estimated MPA. The MPA is the maximum concentration that can be added to the background, while maintaining a desired level of protection (depending on availability of toxicity data). The MPA of 1.1 mg/kg was derived using equilibrium partitioning (MPA(sed)) using the MPA for freshwater and the log Kp(sed/w). The MPA value was then added to an estimated Netherlands background surface water concentration of 0.4 mg/kg (dissolved) methylmercury, to obtain the 1.4 mg/kg MPC value used in the ecological model.

Nickel

The sediment toxicity benchmark used in the ecological model for nickel is 16 mg/kg. This value is obtained from the Ontario Ministry of the Environment's Ontario Ministry of the Environment's *Guidelines for Identifying, Assessing, and Managing Sediments in Ontario* (MOE, 2008). This concentration is assumed to be protective over time to all benthic receptors, and is used with a medium level of confidence.

Phosphorus

The sediment toxicity benchmark used in the ecological model for phosphorus is 600 mg/kg. This value is obtained from the Ontario Ministry of the Environment's Ontario Ministry of the Environment's *Guidelines for Identifying, Assessing, and Managing Sediments in Ontario* (MOE, 2008). This concentration is assumed to be protective over time to all benthic receptors, and is used with a medium level of confidence.

Selenium

The sediment toxicity benchmark used in the ecological model for selenium is 14.7 mg/kg. This value is the sum of 1/10th the SRAeco value of 140 mg/kg and the background concentration (Cb) value of 0.7 mg/kg obtained from van Vlaardingen *et al.* (2005) for the Netherlands National Institute of Public Health and the Environment. The SRA (Serious Risk Addition) values are based on a minimum of toxicity data for four taxa and are statistically derived as the geometric mean of effects levels. The SRAeco is the Serious Risk Addition for ecological receptors and the lowest of the SRAacute and SRAchronic values. To obtain a chronic LOAEL equivalent, the SRAeco was then divided by 10.

Silver

Minor modifications were made to portions of the previous MOE sediment guidance document to either update sections with current information (*e.g.*, update the section on legislation to replace Open Water Disposal Guidelines with Lakefill Guidelines). Previous open water disposal guidelines listed silver, with a benchmark of 0.5 mg/kg. According to more recent guidance by the MOE (2003) document, silver lakefill guidelines are not available for unconfined fill into open waters, (although a value is available for confined fill (*i.e.* within retaining walls, etc.), and so an Ontario specific value sediment value could not be used. An appropriate sediment screening benchmark could be found for silver.

Thallium

The sediment toxicity benchmark used in the ecological model for thallium is 2 mg/kg. This value is the sum of 1/10th the SRAeco value of 140 mg/kg and the background concentration (Cb) value of 1 mg/kg obtained from van Vlaardingen *et al.* (2005) for the Netherlands National Institute of Public Health and the Environment. The SRA (Serious Risk Addition) values are based on a minimum of toxicity data for four taxa and are statistically derived as the geometric mean of effects levels. The SRAeco is the Serious Risk Addition for ecological receptors and the lowest of the SRAacute and SRAchronic values. To obtain a chronic LOAEL equivalent, the SRAeco was then divided by 10.

Tin (Inorganic)

The sediment toxicity benchmark used in the ecological model for inorganic tin is 14,819 mg/kg. This value is the sum of 1/10th the SRAeco value of 148,000 mg/kg and the background concentration (Cb) value of 19 mg/kg obtained from van Vlaardingen *et al.* (2005) for the Netherlands National Institute of Public Health and the Environment. The SRA (Serious Risk Addition) values are based on a minimum of toxicity data for four taxa and are statistically derived as the geometric mean of effects levels. The

SRAeco is the Serious Risk Addition for ecological receptors and the lowest of the SRAacute and SRAchronic values. To obtain a chronic LOAEL equivalent, the SRAeco was then divided by 10.

Vanadium

The sediment toxicity benchmark used in the ecological model for vanadium is 98 mg/kg. This value is the sum of 1/10th the SRAeco value of 560 mg/kg and the background concentration (Cb) value of 42 mg/kg obtained from van Vlaardingen *et al.* (2005) for the Netherlands National Institute of Public Health and the Environment. The SRA (Serious Risk Addition) values are based on a minimum of toxicity data for four taxa and are statistically derived as the geometric mean of effects levels. The SRAeco is the Serious Risk Addition for ecological receptors and the lowest of the SRAacute and SRAchronic values. To obtain a chronic LOAEL equivalent, the SRAeco was then divided by 10.

Zinc

The sediment toxicity benchmark used in the ecological model for zinc is 120 mg/kg. This value is obtained from the Ontario Ministry of the Environment's Ontario Ministry of the Environment's *Guidelines for Identifying, Assessing, and Managing Sediments in Ontario* (MOE, 2008). This concentration is assumed to be protective over time to all benthic receptors, and is used with a medium level of confidence.

5.0 REFERENCES

Agency for Toxic Substances and Disease Registry (ATSDR). 2000. Toxicological profile for Methylene Chloride. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service.

Agency for Toxic Substances and Disease Registry (ATSDR). 2005. Toxicological profile for Carbon Tetrachloride. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service.

Agency for Toxic Substances and Disease Registry (ATSDR). 2005b. Toxicological profile for Tin and Tin Compounds. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service

Agency for Toxic Substances and Disease Registry (ATSDR). 2006. Toxicological profile for Dichlorobenzenes. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service.

Agency for Toxic Substances and Disease Registry (ATSDR). 2006b. Toxicological profile for 1,1,1-Trichloroethane. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service.

Al-Khafaji, A.A., and M.A. Tabatabai. 1979. "Effects of trace elements on arylsulfatase activity in soils." *Soil Sci*. 127(3):129-133.

Alumot, E. (Olomucki), E. Nachtomi, E. Mandel, and P. Holstein. 1976. Tolerance and acceptable daily intake of chlorinated fumigants in the rat diet. *Fd. Cosmet. Toxicol.* 14: 105-110.

Arnold, D.L., C.A. Moodie, S.M. Charbonneau, *et al.* 1985. Long-term toxicity of hexachlorobenzene in the rat and the effect of dietary Vitamin A. Fd. Chem. Toxic. 23(9): 779-793.

Asmatullah, Asma, A., Latif, A., and Shakoori, A. R. 1999. Effect of hexavalent chromium on egg laying capacity, hatchability of eggs, thickness of egg shell and post-hatching development of gallus domesticus. *Asian-Australasian Journal of Animal Sciences.* 12(6): 944-950.

Aulerich, R. J., R. K. Ringer, and S. Iwamoto. 1974. Effects of dietary mercury on mink. Arch. Environ. Contam. Toxicol. 2: 43-51.

Barber, T.R., D.J. Chappie, D.J. Duda, P.C. Fuchsman and B.L. Finley. 1998. Using a spiked sediment bioassay to establish a no-effect concentration for dioxin exposure to the amphipod Ampelisca abdita. Env. Toxicol. Chem. 17: 420-424.

Bell, J.N.B. 1985. SO2 effects on the productivity of grass species. In: *The Effects of Accumulation of Air Pollutants in Forest Ecosystems*. (W.E. Winner, H.A. Mooney and R.A. Goldstein, eds.). pp. 209-266. Stanford University Press, Stanford.

Benedict, F.G. 1938. Vita energetics: A study in comparative basal metabolism. Carnegie Institution Publications. #503: 1-215.

Besser, J. M., W. G. Brumbaugh, N. E. Kemble, T. W. May, and C. G. Ingersoil. 2004. Effects of sediment characteristics on the toxicity of chromium (III) and Chromium (IV) to the amphipod *Hyalella azteca*. *Environmental Science and Technology* 38:6210–16.

Borgmann, U. 2003. Derivation of cause-effect based sediment quality guidelines. Can. J. Fish. Aquat. Sci. 60:352-360.

Bradbury, S., R. Carlson and T. Henry. 1989. Polar narcosis in aquatic organisms. Aquat. Toxicol. Hazard Assess. 12: 59-73.

Canada Gazette. 2008. Draft Screening Assessment for Phenol, 4,4'-(1-methylethylidene)bis- (80-05-7). Part I, Vol. 142 No. 16 (available online at http://www.chemicalsubstanceschimiques.gc.ca/challenge-defi/batch-lot_2_e.html)

Canadian Environmental Protection Act (*CEPA*). 1993. Priority Substances List Assessment Report. Dichlorobenzenes. Health Canada, Ottawa, ON.

CCME (Canadian Council of Ministers of the Environment). 1997. A Framework for Ecological Risk Assessment: Technical Appendices. March 1997.

CCME (Canadian Council of the Ministers of the Environment). 2003. Canadian Environmental Quality Guidelines, Summary Table. Chapter 6, Canadian Sediment Quality Guidelines for the Protection of Aquatic Life. Winnipeg.

CCME (Canadian Council of the Ministers of the Environment). 2007. Canadian soil quality guidelines for the protection of environmental and human health: summary tables. Updated September, 2007. In: Canadian Environmental Quality Guidelines, 1999, Canadian Council of Ministers of the Environment, Winnipeg.

Chappell, W.R. 1992. Scaling toxicity data across species. Environmental Geochemistry and Health. 14(3): 71-80.

Crommentuijn, T., M.D. Polder and E.J. van de Plassche. 1997. Maximum permissible concentrations and negligible concentrations for metals, taking background concentrations into account. National Institute of Public Health and the Environment, Bilthoven.

Daniel, EP and Lillie, RD. 1938. experimental vanadium poisoning in the whit rat/. *Public Health Rep.* 53: 765.

Davidson, I.W.F., Parker, J.C., and Beliles, R.P. 1986. Biological basis for extrapolation across mammalian species. Regulatory Toxicology and Pharamacology. 6: 211-237.

Davison, A.W. 1986. Pathways of fluoride transfer in terrestrial ecosystems. In: Coughtrey, P.J., Martin, M.H., Unsworth, M.H. (eds) Pollutant Transport and Fate in Ecosystems. Blackwell Scientific Publications, Oxford, pp.193-210.

Di Toro, D. M. and J. A. McGrath. 2000. "Technical Basis for Narcotic Chemicals and Polycyclic Aromatic Hydrocarbon Criteria. II. Mixtures and Sediments." Environmental Toxicology and Chemistry 19.8 (2000): 1971-82.

Di Toro, D. M., J. A. McGrath, and D. J. Hansen. 2000. "Technical Basis for Narcotic Chemicals and Polycyclic Aromatic Hydrocarbon Criteria. I. Water and Tissue." Environmental Toxicology and Chemistry 19.8 (2000): 1951-70.

Di Toro, D. M., J. A. McGrath, and W. A. Stubblefield. 2007. "Predicting the toxicity of neat and weathered crude oil: Toxic potential and the toxicity of saturated mixtures." Environmental Toxicology and Chemistry 26.1 (2007): 24-36

Doley, D. 1986. Plant-fluoride Relationships, An Analysis with Particular Reference to Australian Vegetation. Botany Department University of Queensland. Inkata Press. Melbourne.

Doyle, J. J., Pfander, W. H., Grebing, S. E., and Pierce, J. O. 1974. effect of dietary cadmium on growth, cadmium absorption and cadmium tissue levels in growing lambs. *J. Nutr.* 104(2): 160-166.

Duke, L.D. and Taggart, M. 2000. Uncertainty factors in screening ecological assessments. *Environmental Toxicology and Chemistry*. 19(6):1668-1680.

Efroymson, R.A., M.E. Will, G. W. Suter and A.C. Wooten. 1997a. Toxicological benchmarks for screening contaminants of potential concern for effects on terrestrial plants: 1997 revision. Oak Ridge National Laboratory report ES/ER/TM-85/R3.

Efroymson, R.A., M.E. Will and G. W. Suter. 1997b. Toxicological benchmarks for screening contaminants of potential concern for effects on soil and litter invertebrates and heterotrophic processes: 1997 revision. Oak Ridge National Laboratory report ES

Faustman E. M. and Omenn G. S. 1996. Risk assessment, in *Casarett and Doull's Toxicology: The Basic Science of Poisons* 5th ed. (Klaassen CD ed) pp 75-88, McGraw-Hill, New York.

FDA. 1972. Teratologic evaluation of FDA 71-33 (stannous chloride). Washington, DC: U.S. Food & and Drug Administration. PB221780.

Formigli, L., R. Scelsi, P. Poggi, C. Gregotti, A. DiNucci, E. Sabbioni, L. Gottardi, and L. Manzo. 1986. Thallium-induced testicular toxicity in the rat. *Environ. Res*. 40: 531-539.

Freireich, E.J., Gehan, E.A., Rall, D.P., Schmidt, L.H., and Skipper, H.E. 1966. Quantitative comparison of toxicity of anticancer agents in mouse, rat, hamster, dog, monkey and man. Cancer Chemotherapy Reports. 50:219-244.

Garner, J.H.B., T. Pagano, and E.B. Cowling. 1989. An evaluation of the role of ozone, acid deposition, and other airborne pollutants in the forests of eastern North America. General Technical Report SE-59. Southeastern Forest Experiment Station, Forest Service, U.S. Department of Agriculture.

Gibson, S. W., Stevenson, Mary H., and Jackson, N. 1986. comparison of the effects of feeding diets supplemented with zinc oxide or zinc acetate on the performance and tissue mineral content of mature female fowls. *Br. Poult. Sci. (1986)* 27(3): 391-402.

Grimwood, M.J. and Dobbs, T.J. 1995. A review of the aquatic ecotoxicology of polychlorinated dibenzo-p-dioxins and dibenzofurans. Environmental toxicology and water quality. 10: 57-75.

Gupta U.C. and J.A. Mcloed. 1981. Plant and Soil Boron as Influenced by Soil pH and Calcium Sources on Podzol Soils. Soil Sci. 131: 20 - 25.

Gupta, U.C. 1979. Boron nutrition of Crops. In: Advances in Agronomy. Vol. 31. Brady, N.C. (ed.). Academic Press Inc. New York. Pp273- 307.

Heinz, G. H. 1979. Methyl mercury: reproductive and behavioral effects on three generations of mallard ducks. J. Wildl. Mgmt. 43: 394-401.

Heinz, G. H., D. J. Hoffman, A. J. Krynitsky, and D. M. G. Weller. 1987. Reproduction in mallards fed selenium. *Environ. Toxicol. Chem*. 6: 423-433.

Hermens, J. 1989. Quantitative structure-activity relationships in environmental pollutants. pp. 111-162 in: Hutzinger, O., ed., Handbook of Environmental Chemistry. Vol. 2E, Reactions and Processes. Springer-Verlag, Berlin.

Hill, C. H. 1979. the effect of dietary protein levels on mineral toxicity in chicks. *J Nutr.* 109(3): 501-7.

Hill, E. F. and C. S. Schaffner. 1976. Sexual maturation and productivity of Japanese Quail fed graded concentrations of mercuric chloride. Poult. Sci. 55: 1449-1459.

Integrated Risk Information System (IRIS) 1988. National Center for Environmental Assessment, Washington, DC. Accessed 2008 from http://www.epa.gov/ncea/iris/subst/0070.htm3.

Integrated Risk Information System (IRIS) 1992. National Center for Environmental Assessment, Washington, DC. Accessed 2008 from http://www.epa.gov/ncea/iris/subst/0120.htm.

Jensen, L. S., Peterson, R. P., and Falen, L. 1974. Inducement of Enlarged Hearts and Muscular Dystrophy in Turkey Poults with Dietary Silver. *Poultry Science. 53(1): 57-64.*

Johnson, D., Jr., A. L. Mehring, Jr., and H. W. Titus. 1960. Tolerance of chickens for barium. *Proc. Soc. Exp. Biol. Med*. 104: 436-438.

Kabata-Pendias, A., and H. Pendias. 1984. Trace Elements in Soils and Plants. CRC Press, Inc. Boca Raton, Florida.

Kakela, R., A. Kakela, and H. Hyvarinen. 1999. effects of nickel chloride on reproduction of the rat and possible antagonistic role of selenium. *Comp. Biochem. Physiol. Part C: Pharmacol., Toxicol. Endocrinol.* 123C(1): 27-37 .

Kapustka, L.A. 2004. Establishing Eco-SSLs for PAHs: Lessons Revealed from a Review of Literature in Exposure and Effects to Terrestrial Receptors. *Human and Ecological Risk Assessment,* 10:2, 185-205.

Kerstiens G. 1996. Plant Cuticles – an integrated Functional Approach. Bios Scientific Publishes.

Kimmel, C. A., Grant, L. D., Sloan, C. S., and Gladen, B. C. 1980. Chronic Low Level Lead Toxicity in the Rat. 1. Maternal Toxicity and Peri Natal Effects. *Toxicol. Appl. Pharmacol.* 56(1): 28-41.

Kleiber, M. 1932. Body size and metabolism. Hilgardia 6: 315-332.

Krasovskii, G.N. 1976. Extrapolation of experimental data from animals to man. Environmental Health Perspectives. 13: 51-58.

Kruppa, S. V. 1996. The role of atmospheric chemistry in the assessment of crop growth and productivity. In: Plant Response to Air Pollution (Eds. M. Yunus and M. Iqbal), pp 35-73. John Wiley & Sons, Chichester, UK.

Lane, R. W., B. L. Riddle, and J. F. Borzelleca. 1982. Effects of 1,2-dichloroethane and 1,1,1-trichloroethane in drinking water on reproduction and development in mice. *Toxicol. Appl. Pharmacol*. 63: 409-421.

Legge, A.H. and S. V. Krupa. 2002. Effects of sulphur dioxide. In: Air Pollution and Plant Life (Eds. J. Bell and M. Treshow), pp. 135-162. John Wiley & Sons, Chichester, UK.

Legge, A.H., Jager H. and S.V. Kruppa. 1998. Sulphur Dioxide. In: Recognition of Air Pollution injury to vegetation: A pictorial atlas, 2nd Edition (Eds. R.B. Flagler), pp.3-1-1-42. Air and Waste Management Association, Pittsburgh, PA.

Leung K. M. Y., A. Bjorgesaeter, J. S. Gray, W. K. Li, G. C. S. Lui, Y. Wang, P. K. S. Lam. 2005. Deriving sediment quality guidelines from field-based species sensitivity distributions. *Environ Sci Technol* 39:5148-5156.

Lijzen J. P. A., Baars A. J., Otte P. F., Rikken M. G. J., Swartjes F. A., Verbruggen E. M. J., van Wezel A. P., 2001. 'Technical Evaluation of the Intervention Values for Soil/Sediment and Groundwater, Human and Ecotoxicological Risk Assessment and Derivation of Risk Limits for Soil, Aquatic Sediment and Groundwater.' Report 711701 023. RIVM; National Institute of Public Health and the Environment Bilthoven, Netherlands.

Linder, R., Scotti, T., Goldstein, J., McElroy, K., & Walsh, D. 1980. Acute and subchronic toxicity of pentachlorobenzene. J. Environ. Pathol. Toxicol., 4(5-6): 183-196.

Liu, J. Z., Zhang, B. Z., and Milner, J. A. 1994. dietary selenite modifies glutathione metabolism and 7,12-dimethylbenz(a)anthracene conjugation in rats. *J. Nutr.* 124(2): 172-180.

Luttik, R., Mineau, P., Roelofs, W. 2005. A review of interspecies toxicity extrapolation in birds and mammals and a proposal for long-term toxicity data. Ecotoxicology 2005, 14, 817-832.

Malhotra, S.S.; Blauel, R.A. 1980. Diagnosis of air pollutant and natural stress symptoms on forest vegetation in western Canada. North. For. Res. Cent., Can. For. Serv., Environ. Can. Inf. Rep. NOR-X-228. 84 p.

Mansfield T.A and M. Pearson. 1997. Disturbances in stomatal behaviour in plants exposed to air pollution. In: Plant Response to Air Pollution (Eds. M. Yunus and M. Iqbal), pp 179-194. John Wiley & Sons, Chichester, UK.

Mansfield, T.A. 2002. Nitrogen oxides: old problems and new challenges. In: Air Pollution and Plant Life (Eds. J. Bell and M. Treshow), pp. 120-133. John Wiley & Sons, Chichester, UK.

McCauley PT, Robinson M, Daniel FB, *et al.* 1995. Toxicity studies of 1,3-dichlorobenzene in Sprague-Dawley rats. Drug Chem Toxicol 18(2 & 3):201-221.

McCune, D.C. and L.H. Weinstein. 2002. Effects of fluorides. In: Air Pollution and Plant Life (Eds. J. Bell and M. Treshow), pp. 163-171. John Wiley & Sons, Chichester, UK.

McGrath, J.A., T.F. Parkerton, F.L. Hellweger and D.M. Di Toro. 2005. Validation of the narcosis target lipid model for petroleum products: gasoline as a case study. Environ. Toxicol. Chem. 24: 2382-2394.

Miller, W. J., Amos, H. E., Gentry, R. P., Blackmon, D. M., Durrance, R. M., Crowe, C. T., Fielding, A. S., and Neathery, M. W. 1989. long-term feeding of high zinc sulfate diets to lactating and gestating dairy cows. *J Dairy Sci.* 72(6): 1499-508.

Mineau, P, Collins, B.T., and Baril, A. 1996. On the use of scaling factors to improve interspecies extrapolation of acute toxicity in birds. Regulatory Toxicology and Pharamacology. 24: 24-29.

MOE (Ontario Ministry of the Environment), 1993. Ontario Typical Range of Chemical Parameters in Soil, Vegetation, Moss Bags, and Snow. December 1993.

MOE (Ontario Ministry of the Environment), 1999. Water Management. Policies Guidelines Provincial Water Quality Objectives of the Ministry of Environment and Energy.

MOE (Ontario Ministry of the Environment), 2003. Standards Development Branch, Environmental Monitoring and Reporting Branch. Fill Quality Guidelines for Lakefilling in Ontario. March, 2003.

MOE (Ontario Ministry of the Environment). 2004. Soil, Ground Water and Sediment Standards for Use Under Part XV.1 of the Environmental Protection Act. Table 3: Full depth generic site condition standards in a non-potable groundwater condition . March, 2004.

MOE (Ontario Ministry of the Environment), 2008. Guidelines for Identifying, Assessing, and Managing Contaminated Sediments in Ontario: An Integrated Approach. May 2008.

MOEE (Ontario Ministry of the Environment and Energy). 1996. Rationale for the development and application of generic soil, ground water and sediment criteria for use at contaminated site in Ontario. Appendix B3. Rationale for ecotoxicity-based soil criteria

Mordenti, J. 1986. Doseage regimen design for pharmaceutical studies conducted in animals. Journal of Pharmaceutical Sciences. 75: 853-857.

Murray, F.J., F.A. Smith, K.D. Nitschke, C.G. Humiston, R.J. Kociba, and B.A.Schwetz. 1979. “Three-Generation Reproduction Study of Rats Given 2,3,7,8-Tetrachlorodibenzo-p-dioxin (TCDD) in the Diet.” Toxicology and Applied Pharmacology. Volume 50. Pages 241-252.

NAS (National Academy of Sciences). 1977. Drinking Water and Health, Vol. 1. Published by the National Research Council, Washington, DC.

NAS (National Academy of Sciences). 1980. Drinking Water and Health, Vol. 3. Published by the National Research Council, Washington, DC.

NCA (National Coffee Association). 1982. 24-Month chronic toxicity and oncogenicity study of methylene chloride in rats. Final Report. Prepared by Hazleton Laboratories America, Inc., Vienna, VA. (Unpublished)

NCI (National Cancer Institute). (1977) Bioassay of 1,1,1-trichloroethane for possible carcinogenicity. National Institutes of Health, U.S. Department of Health and Human Services, Bethesda, MD; Carcinogenesis Technical Report Series 3. Available from the National Technical Information Service, Springfield, VA; PB265082.

NCI (National Cancer Institute). 1978. Bioassay of trichlorofluoromethane for possible carcinogenicity. Report. No. 106, PHS/NIH, DHEW Publ. No. 78- 1356.

Neiger, R. D. and G. D. Osweiler. 1989. Effect of subacute low level dietary sodium arsenite on dogs. *Fund. Appl. Toxicol*. 13: 439-451.

Nosek, J.A., S.R. Craven, J.R. Sullivan, S.S. Hurley, and R.E. Peterson. 1992. “Toxicity and Reproductive Effects of 2,3,7,8-Tetrachlorodibenzo-p-dioxin in Ring-Necked Pheasant Hens.” Journal of Toxicology and Environmental Health. Volume 35. Pages 187-198.

NTP (National Toxicology Program). 1985. Carcinogenesis studies of 1,2-Dichlorobenzene (CAS No. 95-50-1) in F344/N rats and B6C3F1 mice (gavage studies). Research Triangle Park, N.C., Department of Health and Human Services, Public Health Service, National Institutes of Health. NTP TR 261.

NTP (National Toxicology Program). 1987. Toxicology and carcinogenesis studies of 1,4-dichlorobenzene in F344/N rats and B6C3F1 mice (gavage studies). Research Triangle Park, N.C., Department of Health and Human Services, Public Health Service, National Institutes of Health. NTP TR 319.

NTP (National Toxicology Program). 1989. Toxicology and Carcinogenesis Studies of Tribromomethane (Bromoform) in F344/N Rats and B6C3F_1 Mice (Gavage Studies). Research Triangle Park, N.C., Department of Health and Human Services, Public Health Service, National Institutes of Health. NTP TR 319.

NTP (National Toxicology Program). 1991. Toxicity Studies of 1,2,4,5-tetrachlorobenzene in F344/N rats and B6C3F1 mice (feed studies). Research Triangle Park, N.C., Department of Health and Human Services, Public Health Service, National Institutes of Health. NTP TR 319.

Ohio EPA. 2003. Ecological Risk Assessment Guidance Document. State of Ohio EPA DERR-00-RR-031.

Ohio EPA. 2008. Ecological Risk Assessment Guidance Document. State of Ohio EPA DERR-00-RR-031.

Palmer, A.K., A.E. Street, F.J.C. Roe, et al. 1979. Safety evaluation of toothpaste containing chloroform. II. Long term studies in rats. J. Environ. Path. Toxicol. 2: 821-833.

Prescott, C. A., Wilkie, B. N., Hunter, B., and Julian, R. J. 1982. Influence of a purified grade of pentachlorophenol on the immune response of chickens. Am. J. Vet. Res. (1982) 43(3): 481-7.

Raimondo, S., Mineau, P., and Barron, M. G. 2007. Estimation of Chemical Toxicity to Wildlife Species Using Interspecies Correlation Models. Environ. Sci. Technol. 41: 5888-5894.

Reinecke, A.J., and R.G. Nash. 1984. "Toxicity of 2,3,7,8-TCDD and Short-Term Bioaccumulation by Earthworms (Oligochaeta)." *Soil Biology Biochem*istry. Volume 16. Pages 45-49. As cited in U.S. Fish and Wildlife Service. 1986. *Dioxin Hazards to Fish, Wildlife, and Invertebrates: A Synoptic Review*. Biological Report 85 (1.8). May.

Rhett, G., and others. 1989. "Rate and Effects of PCB Accumulation on *Eisenia foetida*." U.S. Army Corps of Engineers. Waterways Experiment Station. Vicksburg, Mississippi. September 21.

Robinson, K.S., R.J. Kavlock, N. Chernoff and E. Gray. 1981. Multi- generation study of 1,2,4-trichlorobenzene in rats. J. Toxicol. Environ. Health. 8: 489-500.

Romney, E. M., J. D. A. Wallace, and G. V. Alexander. 1975. Response of bush bean and barley to tin applied to soil and to solution culture. *Plant Soil* 42:585-589.

Rossi, F., R. Acampora, C. Vacca, S. Maione, M. G. Matera, R. Servodio, and E. Marmo. 1987. Prenatal and postnatal antimony exposure in rats: effect on vasomotor reactivity development of pups. *Teratog. Carcinog. Mutagen.* 7(5): 491-496.

Sample, B.E. and C.A Arenal. 1999. Allometric models for interspecies extrapolation of wildlife toxicological data. Bull. Environ. Contam. Toxicol. 62:653-663.

Sample, B.E., D.M. Opresko, and G.W. Suter II. 1996. Toxicological Benchmarks for Wildlife: 1996 Revision. Oak Ridge National Laboratory, Oak Ridge, TN. ES/ER/TM-86/.

Schafer, E.W., 1972. The acute oral toxicity of 369 pesticidal, pharmaceutical and other chemicals to wild birds. Toxicol Appl. Pharmacol, 21: 315-330.

Schein, P.S., Davis, R.D., Carter, S., Newman, J., Schein, D.R., and Rall, D.P. 1970. The evaluation of anticancer drugs in dogs and monkeys for the prediction of qualitative toxicities to man. Clinical Pharmacology and Therapeutics. 11: 3-40.

Scholl, G. 1971. Die Immissionsrate von Fluor in Pflanzen als Massstab fur eine Immissions-begrenzung. (with discussions by Hill, A.C., Weinstein, L.H.) VDI-Ber. 164: 39-52.

Schmidt-Nielson, K. 1984. *Scaling: Why is Animal Size So Important?* Cambridge University Press. Cambridge. 1984.

Schroeder, H. A and M. Mitchener. 1975. Life-term studies in rats: effects of aluminum, barium, beryllium, and tungsten. J. Nutr. 105: 421-427.

Smith, G. J. and V. P. Anders. 1989. Toxic effects of boron on mallard reproduction. *Environ. Toxicol. Chem*. 8: 943-950.

Strek, J. H. and J. B Weber. 1980. Absorption and translocation of polychlorinated biphenyls (PCBs) by weeds. *Proc. South. Weed Sci. Soc.* 33:226-232.

Struijs, J., van de Meent, D., Peijnenburg, W.J.G.M., van den Hoop, M.A.G.T., and Crommentuijn, T. 1997. Added Risk Approach to Derive Maximum Permissible Concentrations for Heavy Metals: How to Take Natural Background Levels into Account. Ecotoxicology and Environmental Safety. 37:112-118.

Stulen, I., Perez-Soba, M., De Kok, L.J. and Van der Eerden, L. 1998. Impact of gaseous nitrogen deposition on plant functioning. New Phytologist 139, 61 -70.

Swartjes, F.A. 1999. Risk-based Assessment of Soil and Groundwater Quality in the Netherlands: Standards and Remediation Urgency. Risk Analysis 19(6): 1235-1249.

Taylor, O.C., Thompson, C.R. Tingey, D.T. and Reinert T.A. 1975. Oxides of nitrogen. In: Responses of Plants to Air Pollution (Eds. J. B. Mudd and T.T. Kozlowski), pp 121-139.

Theuer RC, Mahoney AW, Sarett HP. 1971. Placental transfer of fluoride and tin in rats given various fluoride and tin salts. J Nutr 101:525-532.

Travis, C.C. and White R.K. 1988. Interspecific scaling of toxicity data. Risk Analysis. 8:119-125

U.S. EPA. 1988. Recommendations for and documentation of biological values for use in risk assessment. Environmental Criteria and Assessment Office, Cincinnati, OH. EPA/600/6-87/008.

U.S. EPA. 1993. National Ambient Air Quality Standards. http://www.epa.gov/air/criteria.html. Website access February 9, 2009.

US EPA. 1995. *Great Lakes Water Quality Initiative Technical Support Document for Wildlife Criteria.* Office of Water. EPA-820-B-95-009.

U.S. EPA. 1999. Screening Level Ecological Risk Assessment Protocol for Hazardous Waste Combustion Facilities. Available at: http://www.epa.gov/combustion/ecorisk.htm

U.S. EPA. 2002. A Review of the Reference Dose and Reference Concentration Processes. Risk Assessment Forum. EPA/630/P-02/002F.

U.S. EPA Region 5. 2003. Region 5 RCRA Corrective Action Ecological Screening Values. www.epa.gov/reg5rcra/ca/edql.htm.

U.S. EPA. 2005a. Ecological Soil Screening Levels for Antimony, Interim Final. http://www.epa.gov/ecotox/ecossl/

U.S. EPA. 2005b. Ecological Soil Screening Levels for Arsenic. Interim Final http://www.epa.gov/ecotox/ecossl/

U.S. EPA. 2005c. Ecological Soil Screening Levels for Barium, Interim Final. http://www.epa.gov/ecotox/ecossl/

U.S. EPA. 2005d. Ecological Soil Screening Levels for Beryllium, Interim Final. http://www.epa.gov/ecotox/ecossl/

U.S. EPA. 2005e. Ecological Soil Screening Levels for Cadmium, Interim Final. http://www.epa.gov/ecotox/ecossl/

U.S. EPA. 2005f. Ecological Soil Screening Levels for Cobalt, Interim Final. http://www.epa.gov/ecotox/ecossl/

U.S. EPA. 2005g. Ecological Soil Screening Levels for Lead, Interim Final. http://www.epa.gov/ecotox/ecossl/

U.S. EPA. 2005h. Ecological Soil Screening Levels for Vanadium, Interim Final. http://www.epa.gov/ecotox/ecossl/

U.S. EPA. 2005i. User's Guide T-REX Version 1.2.3 (*T*errestrial *R*esidue *EX*posure model). Available on-line at http://www.epa.gov/oppefed1/models/terrestrial/trex_usersguide.htm

U.S. EPA. 2006. Ecological Soil Screening Levels for Silver, Interim Final. http://www.epa.gov/ecotox/ecossl/

U.S. EPA. 2007a. Ecological Soil Screening Levels for Nickel, Interim Final. http://www.epa.gov/ecotox/ecossl/

U.S. EPA. 2007b. Ecological Soil Screening Levels for Selenium, Interim Final. http://www.epa.gov/ecotox/ecossl/

U.S. EPA. 2007c. Ecological Soil Screening Levels for Zinc, Interim Final. http://www.epa.gov/ecotox/ecossl/

U.S EPA. 2007d. Ecological Soil Screening Levels for Polycyclic Aromatic Hydrocarbons (PAHs). Interim Final. Office of Solid Waste and Emergency Response. Washington, DC. Available online at: http://www.epa.gov/ecotox/ecossl/

U.S. EPA. 2007e. Ecological Soil Screening Levels for Pentachlorophenol, Interim Final. http://www.epa.gov/ecotox/ecossl/

U.S. EPA. 2007f. Toxicological review of 1,1,1-Trichloroethane (CAS No. 71-55-6). Integrated Risk Information System (IRIS). National Center for Environmental Assessment, Washington, DC; EPA/635/R-03/013. Available online at http://www.epa.gov/iris.

U.S. EPA. 2008. Ecological Soil Screening Levels for Chromium, Interim Final. http://www.epa.gov/ecotox/ecossl/

U.S. Fish and Wildlife Service (U.S. FWS). 1969. Bureau of sport fisheries and wildlife. Publication 74, pp. 56–57.

U.S. Fish and Wildlife Service (U.S. FWS). 1964. Pesticide-wildlife studies, 1963: a review of Fish and WildlifeService investigations during the calendar year. FWS Circular 199.

van Gestel, C.A.M., W.-C. Ma, and C.E. Smit. 1991. "Development of QSAR's in terrestrial ecotoxicology: Earthworm toxicity and soil sorption of chlorophenols, chlorobenzenes and dichloroaniline." Sci. Total. Environ. 109/110:589-604.

Van Vlaardingen, P.L.A., Posthumus, R., and Posthuma-Doodeman, C.J.A.M. 2005. Environmental Risk Limits for Nine Trace Elements. RIVM report 601501029/2005.

Van Vleet, J. F. 1976. Induction of Lesions of Selenium Vitamin E Deficiency in Pigs Fed Silver. *American Journal of Veterinary Research.* 37(12): 1415-1420.

Verhaar, H., C. Van Leeuwen and J. Hermens. 1992. Classifying environmental pollutants. 1. Structure-activity relationships for prediction of aquatic toxicity. Chemosphere 25: 471-491.

Verschuuren, H. G., R. Kroes, E. M. Den Tonkelaar, J. M. Berkvens, P. W. Helleman, A. G. Rauws, P. L. Schuller, and G. J. Van Esch. 1976. Toxicity of methyl mercury chloride in rats. II. Reproduction study. Toxicol. 6: 97-106.

von Stackelberg, K. and C. Menzie. 2002. A cautionary note on the use of species presence and absence data in deriving sediment quality criteria. *Environmental Toxicology and Chemistry* 21(2):466-472.

Vos, J. G., H. L. Van Der Maas, A. Musch, and E. Ram. 1971. Toxicity of hexachlorobenzene in Japanese quail with special reference to porphyria, liver damage, reproduction, and tissue residues. *Toxicol. Appl. Pharmacol*. 18: 944-957.

VROM (Netherlands ministry of housing, spatial planning, and the environment), 2000. Circular on target values and intervention values for soil remediation. DBO/1999226863.

Walton, B.T., T.A. Anderson, M.S. Hendricks, and S.S. Talmage. 1989. Physiochemical properties as predictors of organic chemical effects on soil microbial respiration. Environ Toxicol Chem 8:53-63.

Weinstein, D.A., Samuelson, L.J. and Arthur, M.A. 1998. Comparison of the response of red oak *(Quercus rubra)* seedlings and mature trees to ozone exposure using simulation modeling. *Environmental Pollution* 102: 307-320

Weir, R. J., and R. S. Fisher. 1972. Toxicological studies on borax and boric acid. *Toxicol. Appl. Pharmacol*. 23: 351-364.

Wentzel, K.F. 1983. IUFRO studies on maximal SO_2 emissions standards to protect forests. In: The Effects of Accumulation of Air Pollutants in Forest Ecosystems. (B. Ulrich and J. Pankrath, eds.), pp. 295-302. D Reidel, Dordrecht.

WHO (World Health Organization). 2000. Air Quality Guidelines for Europe, 2nd Edition. WHO Regional Publications, European Series, No. 91.