**HMS AQUATOX Ecotoxicology Model Data Requirements**

To model ecotoxicology in HMS, the HMS AQUATOX chemical-fate model may be implemented so that chemical concentrations in the water column are available. Alternatively, chemical concentrations in the water column and/or sediment may be externally input to the model based on data, assumption, or alternative model calculation.

To model accumulation in organic matter, plants, and animals, those models may be implemented and the data requirements for those components met. Again, plant, animal, and organic-matter concentrations in the model may be externally input as an alternative.

Example JSON data files that include ecotoxicology models with and without external linkages may be found in the associated DOCS and TEST directories.

The following pages are excerpts from the relevant sections of the AQUATOX Release 3.2 Technical Documentation. The HMS ecotoxicology model was not changed from the AQUATOX Release 3.2 implementation and results were verified against AQUATOX Release 3.2 results.

**9. ECOTOXICOLOGY**

Unlike most ecological models, AQUATOX contains an ecotoxicology submodel that computes both lethal and sublethal acute toxic effects from the concentration of a toxicant in a given organism. Furthermore, because AQUATOX is an ecosystem model, it can simulate indirect effects such as loss of forage base, reduction in predation, and anoxia due to decomposition following a fish kill.

**Ecotoxicology: Simplifying Assumptions**

* Toxic effects of multiple chemicals are additive
* Sublethal effects levels of chemicals may be estimated as a fraction of lethal effects levels
* Regressions from one species to another are available regardless of the mode of action
* The external toxicity model assumes immediate toxic effect to a level of external exposure
* Cumulative toxicity considers differing tolerances in a population, but ignores inherited tolerance
* Resistance to lower doses is conferred for the lifetime of an animal and for one year for a plant.

User-supplied values for *LC50*, the concentration of a toxicant in water that causes 50% mortality, form the basis for a sequence of computations that lead to estimates of the biomass of a given organism lost through lethal toxicity each day. The sequence, which is documented in this chapter, is to compute:

* the internal concentration causing 50% mortality for a given period of exposure;
* the internal concentration causing 50% mortality after an infinite period of time based on an asymptotic concentration-response relationship;
* the time-varying lethal internal concentration of a chemical;
* the cumulative mortality for a given internal concentration;
* the biomass lost per day as an increment to the cumulative mortality.

The user-supplied *EC50*s, the concentrations in water eliciting sublethal toxicity responses in 50% of the population, are used to obtain factors relating the sublethal toxicities to the lethal toxicity. Because AQUATOX can simulate as many as twenty toxic organic chemicals simultaneously, the simplifying assumption is made that the toxic effects are additive.

**9.1 Lethal Toxicity of Compounds**

**Interspecies Correlation Estimates (ICE)**

Often *LC50* data will only be available for one or two of the many species that a user wishes to include in a simulation. To alleviate this problem, a substantial database of regressions (Interspecies Correlation Estimation, ICE) is available as developed by the US. EPA Office of Research and Development, the University of Missouri-Columbia, and the US Geological Survey (Asfaw and Mayer, 2003). At this time the Web-ICE database has over 2000 regressions with over 100 aquatic species as “surrogates” (Raimondo et al. 2007). Regressions may be made on the basis of species, families, or genera. The database also includes goodness of fit information for regressions so their suitability for a given application may be ascertained. Only statistically significant regressions are included in the database.

Using the ICE database and the following regression equation, the model can be parameterized to represent a complete food web.

 **(409)**

where:

*LC50Estimated* = estimated *LC50* (μg/L);

*Intercept* = intercept for regression (μg/L);

*Slope* = slope of the regression equation;

*LC50Observed* = observed *LC50* (μg/L).

The ICE database may be found at <https://www3.epa.gov/webice/>

Experimentally derived toxicity data for individual species should be used when available. However, ICE may then be used to estimate toxicity for species that have not yet been studied given a particular chemical.

**Internal Calculations**

In the default AQUATOX model, toxicity is based on the internal concentration of the toxicant in the specified organism. Many compounds, especially those with higher octanol-water partition coefficients, take appreciable time to accumulate in the tissue. Therefore, length of exposure is critical in determining toxicity. The same principles apply to organic toxicants and to both plants and animals.

The internal lethal concentration for a given period of exposurecan be computed from reported lethal toxicity data based on the simple relationship suggested by an algorithm in the FGETS model (Suárez and Barber, 1992):

 **(410)**

where:

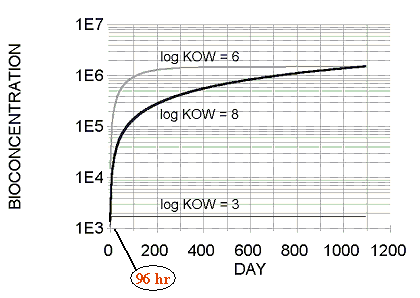
*InternalLC50* = internal concentration that causes 50% mortality;

*BCF* = bioconcentration factor (L/kg), see **(342)** to **(349)**; and

*LC50* = concentration of toxicant in water that causes 50% mortality (μg/L).

For compounds with a *LogKOW* in excess of 5 the usual 96-hr toxicity exposure does not reach steady state, so a time-dependent *BCF* is used to account for the actual internal concentration at the end of the toxicity determination. This is applicable no matter what the length of exposure (Figure 158, based on Figure 144).

Figure 158: Bioconcentration factor as a function of time and KOW



The internal concentration causing 50% mortality after an infinite period of exposure, *LCInfinite,* can be computed by:

 **(411)**

where:

*k2* = elimination rate constant (1/d); and

*ObsTElapsed* = exposure time in toxicity determination (h).

Essentially this equation determines the asymptotic toxicity relationship and provides the model with a constant toxicity parameter for a given compound.

The model estimates *k2*, see **(364)** and **(354)**, assuming that this *k2* is the same as that measured in bioconcentration tests; good agreement has been reported between the two (Mackay et al., 1992). The user may then override that estimate by entering an observed value. The *k2* can be calculated off-line based on the observed half-life:

 **(412)**

where:

*t½*  = observed half-life.

Based on the Mancini (1983) model, the lethal internal concentration of a toxicant for a given exposure period can be expressed as (Crommentuijn et al. (1994):

 **(413)**

where:

*LethalConc* = tissue-based concentration of toxicant that causes 50% mortality (ppb or μg/kg);

*LCInfinite* = ultimate internal lethal toxicant concentration after an infinitely long exposure time (ppb);

*TElapsed* = period of exposure (d).

The longer the exposure the lower the internal concentration required for lethality.

Exposure is limited to the lifetime of the organism:

 **(414)**

where:

*LifeSpan* = user-defined mean lifetime for given organism (d).

Based on an estimate of time to reach equilibrium (Connell and Hawker, 1988),

 **(415)**

The fraction killed by a given internal concentration of toxicant is best estimated using the time-dependent *LethalConc* in the cumulative form of the Weibull distribution (Mackay et al., 1992; see also Christensen and Nyholm, 1984):

 **(416)**

where:

*CumFracKilled* = fraction of organisms killed per day (g/g d),

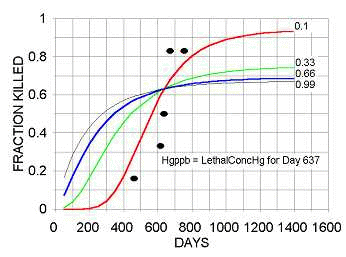
*PPB* = internal concentration of toxicant (μg/kg), see **(310)**; and

*Shape* = parameter expressing variability in toxic response (unitless).

As a practical matter, if *CumFracKilled* exceeds 95%, then it is set to 100% to avoid complex computations with small numbers. By setting organismal loadings to very small numbers, seed values can be maintained in the simulation.

This formulation is preferable to the empirical probit and logit equations because it is simple and yet based on mechanistic relationships. The *Shape* parameter is important because it controls the spread of mortality. The larger the value, the greater the distribution of mortality over toxicant concentrations and time. Mackay et al. (1992) found that a value of 0.33 gave the best fit to data on toxicity of 21 narcotic chemicals to fathead minnows. This value is used as a default in AQUATOX, but it can be changed by the user. Although mercury is not currently modeled, data on MeHg toxicity shows that the *Shape* parameter may take a value less than 0.1 (Figure 154).

Figure 159. The effect of *Shape* in fitting the observed (McKim et al., 1976)  
cumulative fraction killed following continued exposure to MeHg



The biomass killed per day is computed by disaggregating the cumulative mortality. Think of the biomass at any given time as consisting of two types: biomass that has already been exposed to the toxicant previously, which is called *Resistant* because it represents the fraction that was not killed; and new biomass that has formed through growth, reproduction, and migration and has not been exposed to a given level of toxicant and therefore is referred to as *Nonresistant*. Then think of the cumulative distribution as being the total *CumFracKilled,* which includes the *FracKilled* that is in excess of the cumulative amount on the previous day if the internal concentration of toxicant increases. A conservative estimate of the biomass killed at a given timeis computed as:

 **(417)**

where:

*Poisoned* = biomass of given organisms killed by exposure to toxicant at given time (g/m3 d);

*Resistant* = fraction of biomass not killed by previous exposure (frac);

*FracKilled* = fraction killed per day in excess of the previous fraction (g/g d);

*Nonresistant =* biomass not previously exposed; the biomass in excess of the resistant biomass (g/m3) = *(1-Resistant)·Biomass*.

New biomass is considered vulnerable, ignoring the possibility of inherited tolerance. It is assumed for purposes of risk analysis that resistance is not conferred for an indefinite period. In animals elapsed exposure time is capped at the average life span, which is a parameter in the animal record. However, it is assumed that resistance persists in the population until the end of the growing season. Macrophytes can live for an entire growing season, and algae usually reproduce asexually as long as conditions are favorable. However, winter die-back does occur in most macrophytes, and many algae will switch to sexual reproduction under unfavorable conditions, especially triggered by light and temperature. As a simplifying assumption for both animals and plants, in the northern hemisphere January 1 is taken as being the date at which exposure and resistance are reset; in the southern hemisphere (denoted by negative latitude in the site record) July 1 is the reset date. On this date, the variables *Resistant, FracKilledPrevious,* and *TElapsed* are all set to zero.

**9.2 Sublethal Toxicity**

Organisms usually have adverse reactions to toxicants at levels significantly below those that cause death. In fact, the lethal to sublethal ratio is commonly used to quantify this relationship. The user supplies observed *EC50* values, which can then be used to compute AFs (application factors). For example:

 **(418)**

where:

*EC50Growth* = external concentration of toxicant at which there is a 50% reduction in growth (μg/L);

*AFGrowth* = sublethal to lethal ratio for growth (unitless); and

*LC50* = external concentration of toxicant at which 50% of population is killed (μg/L).

If the user enters an observed *EC50* value, the model provides the option of applying the resulting *AF* to estimate *EC50*s for other organisms. The computations for AFPhoto and AFRepro are similar:

 **(419)**

 **(420)**

where:

*EC50Photo* = external concentration of toxicant at which there is a 50% reduction in photosynthesis (μg/L);

*AFPhoto* = sublethal to lethal ratio for photosynthesis (unitless);

*EC50Repro* = external concentration of toxicant at which there is a 50% reduction in reproduction (μg/L); and

*AFRepro* = sublethal to lethal ratio for reproduction (unitless).

Because of the nature of these application factors, sublethal effects cannot be calculated (using internal calculations) unless LC50 parameters are included in the model.

Similar to computation of lethal toxicity in the model, sublethal toxicity is based on internal concentrations of a toxicant. Often sublethal effects form a continuum with lethal effects and the difference is merely one of degree (Mackay et al., 1992). Regardless of whether or not the mode of action is the same, the computed factors relate the observed effect to the lethal effect and permit efficient computation of sublethal effects factors in conjunction with computation of lethal effects. Because AQUATOX simulates biomass, no distinction is made between reduction in a process in an individual and the fraction of the population exhibiting that response. The commonly measured reduction in photosynthesis is a good example: the data only indicate that a given reduction takes place at a given concentration, not whether all individuals are affected. The factor enters into the Weibull equation to estimate reduction factors for photosynthesis, growth, and reproduction:

 **(421)**

 **(422)**

 **(423)**

where:

*FracPhoto* = reduction factor for effect of toxicant on photosynthesis (unitless);

*RedGrowth* = factor for reduced growth in animals (unitless);

*RedRepro* = factor for reduced reproduction in animals (unitless);

*PPB* = internal concentration of toxicant (μg/kg), see **(310)**;

*LethalConc* = tissue-based conc. of toxicant that causes mortality (μg/kg), see **(413)**;

*AFPhoto* = sublethal to lethal ratio for photosynthesis (unitless, default of 0.10);

*AFGrowth* = sublethal to lethal ratio for growth in animals (unitless, default of 0.10);

*AFRepro*  = sublethal to lethal ratio for reproduction in animals (unitless, default of 0.05);

*Shape* = parameter expressing variability in toxic response (unitless, default of 0.33).

The reduction factor for photosynthesis, *FracPhoto*, enters into the photosynthesis equation (Eq. **(35)**) and it also appears in the equation for the acceleration of sinking of phytoplankton due to stress (Eq. **(69)**).

The variable for reduced growth, *RedGrowth*, is arbitrarily split between two processes, ingestion (Eq. **(91)**), where it reduces consumption by 20%:

 **(424)**

and defecation (Eq. **(97)**), where it increases the amount of food that is not assimilated by 80%:

 **(425)**

These have indirect effects on the rest of the ecosystem through reduced predation and increased production of detritus in the form of feces.

Embryos are often more sensitive to toxicants, although reproductive failure may occur for various reasons. As a simplification, the factor for reduced reproduction, *RedRepro*, is used only to increase gamete mortality (Eq. **(126)**) beyond what would occur otherwise:

 **(426)**

By modeling sublethal and lethal effects, AQUATOX makes the link between chemical fate and the functioning of the aquatic ecosystem- a pioneering approach that has been refined over the past twenty years, following the first publications (Park et al., 1988; Park, 1990).

Sloughing of periphyton and drift of invertebrates also can be elicited by toxicants. For example, sloughing can be caused by a surfactant that disrupts the adhesion of the periphyton, or an invertebrate may release its hold on the substrate when irritated by a toxicant. Often the response is immediate so that these responses can be modeled as dependent on dissolved concentrations of toxicants with an available sublethal toxicity parameter, as in the equation for periphyton sloughing:

 **(427)**

where:

*DislodgePeri, Tox* = periphyton sloughing due to given toxicant (g/m3 d);

*MaxToxSlough* = maximum fraction of periphyton biomass lost by sloughing due to given toxicant (fraction/d, 0.1);

*ToxicantWater* = concentration of toxicant dissolved in water (μg/L); see **(300)**;

*EC50Dislodge* = external concentration of toxicant at which there is 50% sloughing (μg/L); and

*BiomassPeri* = biomass of given periphyton (g/m3); see **(33)**.

Likewise, drift is greatly increased when zoobenthos are subjected to stress by sublethal doses of toxic chemicals (Muirhead-Thomson, 1987), and that is represented by a saturation-kinetic formulation that utilizes an analogous sublethal toxicity parameter :

 **(428)**

where:

*ToxicantWater* = concentration of toxicant in water (μg/L);

*DriftThreshold* = the concentration of toxicant that initiates drift (μg/L); and

*EC50Growth* = concentration at which half the population is affected (μg/L).

These terms are incorporated in the respective periphyton washout **(72)** and zoobenthos drift **(130)** equations.

**9.3 External Toxicity**

Chemicals that are taken up very rapidly and those that have an external mode of toxicity, such as affecting the gills directly, are best simulated with an external toxicity construct. AQUATOX has an alternative computation for *CumFracKilled,* when calculating toxic effects based on external concentrations, using the two-parameter Weibull distribution as in Christiensen and Nyholm (1984):

 **(429)**

where:

*z*  = external concentration of toxicant (g/L);

*CumFracKilled* = cumulative fraction of organisms killed for a given period of exposure

(fraction/d), applied to equation **(417)**;

*k* and *Eta* = fitted parameters describing the dose response curve.

Rather than require the user to fit toxicological bioassay data to determine the parameters for *k* and *Eta,* these parameters are derived to fit the LC50 and the slope of the cumulative mortality curve at the LC50 (in the manner of the RAMAS Ecotoxicology model, Spencer and Ferson, 1997):

 **(430)**

 **(431)**

where: *slope*  = slope of the cumulative mortality curve at LC50 (unitless).

LC50 = concentration where half of individuals are affected (g/L).

AQUATOX can assume that each chemical’s dose response curve has a distinct shape, relevant to all organisms modeled. In this manner, a single “slope factor” parameter describing the shape of the Weibull curve can be entered in the chemical record rather than requiring the user to derive slope parameters for each organism modeled. (Note, this is different than the *shape* parameter used for internal toxicity.) However, animal and plant-specific slope factors may also be entered in the animal and plant chemical-toxicity databases. If these values are left blank or zero, the value from the chemical record is used. Otherwise the organism-specific factor is used. The units for this factor are the same as those for the chemical underlying data (the slope at EC50 multiplied by the EC50 in ug/L).

As shown below, the slope of the curve at the LC50 is both a function of the shape of the Weibull distribution and also the magnitude of the LC50 in question. Figure 160 shows two Weibull distributions with identical shapes, but with slopes that are significantly different due to the scales of the x axes.

Figure 160. Weibull distributions with identical shapes, but different slopes.

**0.01**



For this reason, rather than have a user enter “the slope at LC50” into the chemical record, AQUATOX asks the user to enter a “slope factor” defined as “the slope at LC50 multiplied by LC50.” In the above example, the user would enter a slope factor of 1.0 and then, given an LC50 of 1 or an LC50 of 100, the above two curves would be generated.

When modeling toxicity based on external concentrations, organisms are assumed to come to equilibrium with external concentrations (or the toxicity is assumed to be based on external effects to the organism).

Unlike the internal model, application factors are not used to estimate sublethal effects when calculating external toxicity. Therefore, EC50 parameters do not need to be paired with LC50 values to calculate sublethal effects