7 Ecological Risk Assessment

This section provides practical guidance on addressing the unique challenges associated with conducting an ecological risk assessment (ERA) for TPH. Although an ERA may be warranted at TPH release sites with potentially complete exposure pathways for ecological receptors (see Conceptual Site Models), the approaches that have been used for TPH ERA vary by state and country with no single widely accepted TPH ERA guidance.

This section also presents tiered approaches for TPH ERAs, ranging from simple and qualitative to complex, site-specific, quantitative ecological risk evaluations. It describes types of TPH analytical data that may be appropriate for ERA, ranging from whole products to fractions to individual constituents, and provides a framework for selecting analytical data that are appropriate for the exposure media and receptors. This section also describes both model-based and toxicity testing–based methods for developing threshold toxicity values for TPH. Options and best practices for risk characterization are presented, as well as a practical overview of issues to consider and pitfalls to avoid in TPH ERAs. The proposed approach is generally consistent with USEPA guidance for ecological risk assessment USEPA 1997a.

7.1 ERA Process for TPH

TPH releases and exposures can range from single-event, petroleum product releases (e.g., crude oil spill into a marsh) to long-term discharges (e.g., diesel-contaminated groundwater seeping into a creek). ERAs typically evaluate a range of habitats, trophic levels, and, most importantly, representative species. Theoretically, each species could have a different response to TPH exposure. This, coupled with the fact that TPH is a complex mixture of numerous compounds that can change through time via physical weathering and/or biological transformation, makes TPH-focused ERAs particularly challenging.

The ecological effects of short-term and long-term releases are typically very different. During an accidental, single-event petroleum product release, the smothering of invertebrates or a reduction in the insulating properties of avian feathers is likely to be the primary risk mechanism in terms of immediate effects. Long-term discharges of TPH components to surface water are likely to have chronic effects on the receiving aquatic community that may be subtle and harder to quantify (relative to a petroleum product release such as a crude oil spill). This section focuses on the toxicological effects of short-term or long-term discharges rather than the physical effects associated with oil spills.

Additional information is available from National Oceanic and Atmospheric Administration (NOAA) guidance (https://oceanservice.noaa.gov/hazards/spills/) and other relevant materials, such as the International Oil Spill Conference Proceedings website (http://ioscproceedings.org), to address short-term spills, acute exposures, and their immediate aftermath.

Finally, elevated ecological risks in the narrow context of TPH toxicity do not always warrant remediation efforts that may themselves cause severe ecological disturbance or habitat destruction. Methods, such as Net Environmental Benefit Analysis, may be used to decide on the appropriate remedial strategies that will reduce risk and minimize collateral damage API 2013; USEPA 2006a

▼*Read more*

7.1.1 ERA Is a Tiered Process

Although the TPH States Survey reports that 24 states would require remediation of TPH-impacted sites based only on ecological risk (i.e., if human health risks were acceptable), an ERA may not always be necessary. Section 5.4 (Table 5-3) provides an overview of the exclusion/inclusion criteria for ERAs. This includes consideration of factors such as the size and location of the impacted area and the presence or absence of ecological receptors. State or federal regulatory concurrence would be necessary in individual cases because application of exclusion criteria varies among regulatory agencies.

If an ERA is warranted, it is typically conducted in a tiered manner with earlier, screening-level tiers employing conservative and generic exposure assumptions and screening criteria and later tiers using more site-specific information. In the early tiers of an ERA, it is standard practice to retain constituents that lack screening values for evaluation in a higher tier. As

Table 7-1 demonstrates, only a few jurisdictions have ecologically based TPH screening values. Therefore, many sites with detected TPH concentrations are likely to advance to a more complex, site-specific ERA.

Table 7-1. Summary of currently available ecologically based TPH screening values

Analyte	Sed. (ppm)	Soil (ppm)			Aquatic Habitat Goal (ppb)			Jurisdiction (Citation)
		Plants	Biota	Wildlife	Fresh	Marine	Estuarine	
TPH (diesel)	340-510	—	—	—	—	—	—	USA USEPA 2015b
TPH (residual)	3,600–4,400	—	—	—	—	—	—	
TPH (gasoline)	15–67	210–320	210–320	1100	1500	1500	—	Canada–Atlantic partnership Atlantic PIRI 2012a (see referenced document)
TPH (diesel)	25–112	150–2,500	150–2,500	9,800–16,000	100	100	—	
TPH (motor oil)	43–192	2,800–6,600	2,800–6,600	8,400	100	100	—	
Gasoline Range Organics	340–510	120	120	1,000–5,000	—	—	—	Washington WADOE 2016b, 2016a
Diesel Range Organics	—	1,600	240	2,000–6,000	—	—	—	
TPH (residual)	3,600–4,400	—	—	—	—	—	—	
TPH (gasoline)	—	—	—	—	440	3700	—	California CASWB-SFBR 2016a
TPH (Stoddard solvent)	—	—	—	—	640	640	—	
TPH (diesel)	—	—	—	—	640	640	—	
TPH	—	1,700	1,700	1,700	—	—	—	New Jersey NJDEP 2017a
TPH (gasoline)	—	—	—	—	500	3,700	500	Hawaii HIDOH 2017 (Appendix I, Table D-4a; only chronic values are shown; acute values are available in this reference)
TPH (middle distillates)	—	—	—	—	640	640	640	
TPH (residual fuels)	—	—	—	—	640	640	640	

Note: Sediments are saturated soils at the bottom of water bodies

In later ERA tiers, estimated receptor TPH doses are compared to toxicity reference values that may be expressed on a media concentration basis (e.g., LC50 [concentration that is lethal to 50% of the exposed organisms], EC50 [median concentration that elicits a response in 50% of the exposed organisms] in mg/L) or a body-weight-normalized basis (i.e., mg/kg BW-day). The availability of TPH toxicity data varies with receptor type, and it is important to understand the

assumptions and limitations of the toxicity information when using it in a risk evaluation.

Although this brief introduction makes the ERA process sound quite linear (i.e., screening-level ERA to site-specific ERA to remediation), the reader should be aware that there have been attempts to employ adaptive management methods into ERAs Stankey, Clark, and Bormann 2005; Williams 2011. ERA-based adaptive management methods yield an iterative process where results (and consequently risk) are updated periodically based on the inclusion of new data and/or monitoring information. This results in a reduction of uncertainty over time and can be especially useful for large and/or complex sites.

7.2 Current National and North American TPH ERA Practice

At this time, there is no national guidance for TPH ERA in the United States. However, TPH ERA screening guidance has been published by a few states and in Canada. The TPH States Survey indicates that 17 states follow publicly available guidance for conducting TPH ERAs. Of these, only New Jersey, Hawaii, Washington State, and California State Water Boards—San Francisco Bay Region (CASWB-SFBR) have developed ecologically based TPH screening levels (Table 7-1). Typically, these states have designated specific analytical methods to obtain TPH-based data (see TPH Analytical Methods and States Survey). Table 7-1 also provides TPH ERA screening levels for USEPA Region 4 and Canada.

▼*Read more*

7.2.1 Available Regulatory Screening Levels for TPH

Multiple approaches exist for establishing TPH ERA screening criteria, and a brief discussion of the approaches used for the Table 7-1 criteria is provided. The sources and links provided in Tables 7-8 through 7-10 may also be checked to obtain the most current version of available screening levels. These values are typically used at the screening-level tier to evaluate if a medium or site warrants additional evaluation. Screening levels should always be reviewed prior to use to confirm status, technical basis, assumptions, and limitations.

The Washington State freshwater sediment TPH standards were developed using the floating percentile model and acute toxicity tests (10-day *Chironomus* growth and/or mortality). The USEPA references the Washington State values (Table 7-1). Canada's TPH sediment screening values were derived using a combination of the PETROTOX model and an equilibrium partitioning–based approach.

Except for the New Jersey values, all soil screening values shown on Table 7-1 were derived via toxicity testing with various test organisms. The soil screening values for wildlife (Canada and Washington State) are based on toxicological testing and a wildlife exposure model. The only information that can be found related to the New Jersey soil screening value (1,700 mg/kg) was that it was established following a literature search and a review of the pertinent documents.

The surface water screening values in Table 7-1 (aquatic habitat goals) are all based on toxicity testing.

TPH screening levels have also been developed by nonregulatory sources, including API Pattanayek and DeShields 2004, Battelle 2007, Oak Ridge National Laboratories Efroymson, Sample, and Peterson 2004, and others or for specific projects such as Portland Harbor USEPA 2013b.

7.2.2 Canadian Approach

Both national and provincial guidelines for TPH ERA in Canada consider TPH fractions and individual compounds within the framework of different types of land uses and soil types.

At the national level, the Canadian Council of Ministers of the Environment (CCME) 2008 provides soil screening levels (called Canada-wide Petroleum Hydrocarbon standards) that are protective of plants and invertebrates for several TPH fractions (four carbon ranges, F1 through F4, roughly corresponding to gasoline, diesel, lubricants, tars, and waxes) based on toxicity testing and field studies. CCME also developed groundwater screening levels based on literature reviews and uptake modeling for livestock that may consume TPH-contaminated groundwater and for aquatic life that may be exposed to TPH after groundwater discharges to surface water. CCME 2010 also provides soil and water screening levels (called Environmental Quality Guidelines) for individual petroleum constituents (e.g., benzene, individual PAH compounds).

At the provincial level, the Atlantic states Atlantic PIRI 2012a, 2012b have developed TPH fraction screening levels for soil, sediment, groundwater, and surface water using a combination of other agencies' approaches, toxicity testing, equilibrium

partitioning, and Quantitative Structure-Activity Relationship (QSAR)–based models such as PETROTOX. British Columbia has developed surface water screening levels for some TPH fractions for the protection of aquatic life BCMOE 2018. Preference was given to new toxicity data for specific TPH fractions over surrogate data.

7.3 Exposure Assessment

This section describes the variety of factors that should be considered in exposure assessment for TPH ERA. It includes selection of appropriate TPH analytical data that can be used in the screening and site-specific tiers, the distribution of different TPH fractions among various environmental media, and their bioaccumulation potential. As part of both the screening and site-specific tiers, this section also describes approaches for identifying ecological receptors, their routes of exposure to TPH, and exposure dose estimation.

It is important to note that potential ecological exposure does not occur for all releases. For example, releases to areas that have no viable habitat (e.g., paved or gravel areas, manufacturing areas with minimal ecological receptors) and no potential for off-site transport to adjacent habitat areas likely do not warrant consideration for potential ecological risk (see Table 5-3).

▼*Read more*

7.3.1 Interrelationships Between TPH and Environmental Media

Ecological receptors are exposed to different groups of TPH compounds in different exposure media because TPH is a complex mixture of thousands of chemicals with varying physical and chemical properties. Depending on their habitat preferences, ecological receptors may be differentially exposed to the TPH compounds that are volatile, soluble, or sorbed to soil/sediments.

The understanding of the characteristics of the hydrocarbon product in a given medium is important because it is likely predictive of its toxicity and impacts on ecological receptors. This is exemplified by research on the toxicity of oils in aquatic ecosystems relying on the concept of the water-accommodated fraction (WAF).

The WAF is created by mixing together a petroleum material (e.g., crude oil, fuels, lubricating fluids) with water and allowing the soluble components of the mixture to partition to water. As such, WAF is a mixture of short-chain monoaromatic hydrocarbons (BTEX and C3-, C4-, and C5-benzenes), 2- to 4-ring PAHs, and polar organic compounds (e.g., phenols and acids) Hokstad et al. 2000 and includes dissolved as well as any entrained neutrally buoyant free-phase droplets.

Similarly, the concept of water-soluble fraction (WSF) is also sometimes used in testing the toxicity of soil-water mixtures. Although the dissolved phase is generally most closely associated with bioavailability and effects on aquatic organisms and, as such, best quantifies the link between magnitude of exposure and risk, the use of WAF and WSF in toxicity tests may include both soluble and entrained components.

When a more detailed and specific analytical approach is taken, one of the major challenges associated with TPH is its complex mixture characteristic that changes over time. The fate and transport of TPH is discussed in detail in the TPH Fundamentals section. For example, fresh crude oil release sites involve short- and midchain hydrocarbons that may dissolve in the aqueous phase, volatilize into ambient air, rapidly move through soil, and float on water. Fresh TPH tends to have a greater overall potential for adverse effects on ecological receptors. In contrast, legacy petroleum sites with highly weathered materials contain hydrocarbons that tend to be immobile, insoluble, largely inert chemically and toxicologically, and typically not in direct contact with ecological receptors (e.g., buried in soil and sediment).

7.3.2 Expected TPH Distribution in Ecological Exposure Media

TPH analytes likely to be present in the exposure media are listed below, as described in Table 5-2.

- Surface water—Predominantly light aromatic hydrocarbons (e.g., BTEX) from original oil. May include hydrocarbons with pure chemical aqueous solubility (S)> 1 mg/L: C7 n-alkanes <C8 branched alkanes (selected); C12 alkylbenzenes; C13 alkyl-naphthalenes; C14 PAHs. Media/phase may include hydrosols (oil-in-water) or suspended sediments with sorbed chemicals, depending on the amount of mixing of the system and the sampling method. Dissolved-phase contaminants are also likely dominated by petroleum-related metabolites.
- Soil/sediment—Similar composition to original spilled or released oil. Includes up to approximately C32

hydrocarbons for systemic toxicity evaluation. Media/phase may include mobile NAPL or residual (immobile) oil trapped by capillary forces within the soil matrix. Unrefined petroleum (crude, condensate) may include heterogeneous organic chemicals (sulfur, nitrogen, oxygen).

- Ambient and burrow air—Predominately light volatile hydrocarbons. Includes chemicals with pure chemical vapor pressure ≥ 0.008 mm Hg or (equivalently) normal boiling point ≤ 270°C; C15 n-alkanes; C13 alkylbenzenes and naphtheno-benzenes. Ambient air may include significant non-source-related "background"; therefore, if any sampling is done, soil gas is recommended. Vapor sources may include methane (possibly advective) from adjacent methanogenic ebullition, as well as methane generated by source degradation.

7.3.3 Metabolites and TPH Degradation Compounds

TPH from weathered sources may have a greater potential for polar degradation compounds, also known as metabolites, to be present. Understanding the identity and potential toxicity of polar metabolites is a developing field and there are currently no well-established methods to incorporate these concerns into the framework of a typical ERA.

Polar metabolites are characterized by high solubility and predominantly fall into the middle distillate carbon range, in several different chemical classes. Therefore, the ecological receptors most likely to be exposed to polar metabolites in solution are aquatic biota. The potential for ecological receptors to be exposed to metabolites in air (i.e., burrow air) or in soil and sediment is considered negligible because metabolites appear to be primarily composed of soluble polar compounds Bekins et al. 2016; Zemo et al. 2017.

The inclusion of metabolites is not common practice in TPH ERA at this time, although some states and regional approaches have developed recommendations HIDOH 2017; CASWB-SFBR 2016a, 2016b. These agencies recommend the use of TPH analytical data without the use of silica gel cleanup (non-SGC data) for screening level assessments, based on the assumption that polar metabolites are equivalent in toxicity to their parent compounds. However, the uncertainties associated with such an assumption should be understood by the user as discussed in the TPH Fundamentals and Conceptual Site Models sections. These uncertainties include the facts that not all polar metabolite compounds are captured by the extractants used in SGC analysis, naturally occurring organic matter may be included in non-SGC results (unless robust background characterization is performed), and the toxicity of polar compounds may be exercised differently than the nonpolar parent compounds.

7.3.4 Exposure Routes for Ecological Receptors

Ecological receptors can be exposed to TPH in various exposure media through the same general exposure routes as typically evaluated in an ERA. However, in addition to the standard exposure routes of dermal contact, ingestion, and inhalation, TPH exposures also occur through a phenomenon known as oiling. Oiling is a major concern when evaluating TPH in direct contact with free product. Oiling is a common consequence of TPH releases resulting in potential harm to wildlife and birds, such as hemolytic anemia, decreased nutrient absorption, altered stress response, and decreased immune function Bursian et al. 2017from direct (acute toxicity from ingestion when trying to remove oil from fur or feathers) or indirect harm such as adverse effects from the loss of temperature control Bursian, Alexander, et al. 2017.

This exposure pathway is specific to TPH and is seldom seen in risk assessments related to other chemicals. In the event of the presence of free product following a release, this pathway needs to be considered. Tools for addressing oiling or the ecological effects of NAPL/product are beyond the scope of this section. Additional information can be obtained from the International Oil Spill Conference Proceedings website and the oil and chemical spill response resources provided by NOAA.

7.3.5 Bioaccumulation Potential of TPH

The potential for TPH to accumulate in biological tissues is generally considered to be low, with the exception of a few chemical subgroups, primarily consisting of PAHs.

Similar to the discussion in Section 6.8 for food chain bioaccumulation for human health, there are limited TPH bioaccumulation studies in aquatic and terrestrial ecosystems. Therefore, the focus lies on constituent groups such as PAHs for which some bioaccumulation data are available. In aquatic ecosystems, studies have demonstrated bioaccumulation of PAHs in fish and shellfish because PAHs partition into lipid-rich tissues and many organisms do not metabolize/depurate hydrocarbons efficiently Meador et al. 1995; Torres et al. 2012; Bleeker and Verbruggen 2009. Less is known about PAH bioaccumulation in terrestrial ecosystems. Studies have shown that vertebrates possess efficient metabolic and excretion mechanisms such that PAHs do not accumulate in tissues USEPA 2007a.

For TPH that are not PAHs, bioaccumulation potential can be estimated using partitioning equations. USEPA 2000a noted that bioaccumulation can be predicted for chemicals when the octanol-water partitioning coefficient (log K_{ow}) lies between 2 and 6. For compounds with a log K_{ow} greater than 6, uptake efficiency begins to decrease. Bioaccumulation values for soil-to-plants and soil-to-fish for six aliphatic and aromatic TPH fractions are available from the Risk Assessment Information System database maintained by Oak Ridge National Laboratory (www.rais.ornl.gov). However, these are modeled values based on estimated log K_{ow} for the fractions and cannot be verified by empirical data, partially due to naturally occurring oils in tissue. Due to the degree of uncertainty involved, using predictive models to estimate TPH bioaccumulation into tissue is not recommended. Overall, TPH bioaccumulation is unlikely to be a significant contributor to ecological risk and these evaluations are typically not used in risk management decision making.

7.3.6 Determination of Appropriate Fractions and Carbon Ranges for ERA

An effective ERA is best achieved by capturing the overall TPH concentration in a given exposure medium over a set of specific carbon ranges, from light volatiles to heavy oils, each with dissimilar physicochemical and toxicological properties. An effective ERA also incorporates a temporal component by considering both present and future conditions, given the fate and transport properties of a given TPH mixture.

Tables 7-2, 7-3, and 7-4 present the types of TPH analytes that may be available or analyzed for a particular site, including crude oils and refined petroleum products, operational fractions, fractional components, individual chemical classes, constituents and additives, and metabolites. These data may be used, as appropriate, in the screening and site-specific tiers of TPH ERA. Not all types of data are necessary or appropriate for every site or release.

When identifying carbon fractions and appropriate TPH analytes, the effects of solubility and toxicity should be factored into the appropriateness of carbon range selections for aqueous vs. solid media. Per Zemo et al. 2017, TPH fractions vary in solubility, with shorter chains being somewhat more soluble and longer chains largely insoluble. Given that aquatic toxicity by direct exposure (e.g., fish and invertebrates) is generally related to solubility, the aquatic assessment of TPH may be most warranted up to C16 Zemo et al. 2017 because solubility constraints may exceed toxicity thresholds at greater than C16. The solid media would need to be assessed for the full range of TPH analytes, although toxicity tends to decrease with increasing carbon number as demonstrated by the soil and sediment screening levels for the motor oil range (Table 7-1). Additional information is provided in the TPH Fundamentals and Conceptual Site Models sections.

Table 7-2. Product-based analytical data that can be used for ecological risk assessment

TPH Analyte	When To Use	Strengths	Limitations
1. Crude Oil and Refined Petroleum Products **Method:** 8015M			
Gasoline (GRO/ TPH-g)	Screening-level risk assessment; short-term and long-term evaluation of petroleum product spills and releases to soils and surface waters	Bulk TPH, based on Method 8015 data; consistent with many state-level TPH guidance approaches	Chemical composition may vary greatly based on source product composition and degree of weathering, and screening levels may not be appropriate; whole product analyses typically not done for contaminated site evaluation
Diesel (DRO/ TPH-d)			
Motor oil (RRO/ TPH-mo)			
Fuel oil			
Jet fuel A, B			
Crude oil			
Kerosene			
2. Operationally Defined Fractions **Method:** No specific methods, but partial analysis of individual constituents may be performed (e.g., 3510C extraction, followed by 8260 VOCs, 8270 SVOCs, 8310 (PAHs)			

TPH Analyte	When To Use	Strengths	Limitations
Water-accommodated fraction (WAF)	Site-specific risk assessment; short-term evaluation of petroleum product spills, long-term groundwater releases to surface waters, commingled multiproduct plumes	Direct measurement of aquatic toxicity when used in site-specific context	No readily available screening levels; potential for confounding factors; analytical data may not match toxicity test results
Water-soluble fraction (WSF)			

Table 7-3. Fraction-based analytical data that can be used for ecological risk assessment

TPH Analyte	When to Use	Strengths	Limitations
3. Canadian TPH fractional components (aliphatic and aromatic combined) **Method:** CCME Tier 1 Reference Method (2008)			
C6–C10 (CWS) F1	Screening-level risk assessment; evaluation of long-term releases of petroleum products, remediation and monitoring	Allows consideration of environmental fate and transport properties in relation to exposure media; some screening levels available	May require modification of analytical methods to report these carbon ranges; screening levels available for soil, sediment, groundwater, and surface water for plants and invertebrates only; literature-based toxicity reference values available for livestock and some wildlife
C10–C16 (CWS) F2			
C16–C34 (CWS) F3			
4. TPH FRACTIONAL COMPONENTS (ALIPHATIC AND AROMATIC REPORTED SEPARATELY) **Method:** 11) Atlantic RBCA Tier 2 Reference Method (2016) 2) Specialized 2-dimensional GC analysis Whale et al. 2013; Comber et al. 2016			
C5–C6 (Ali only)	Site-specific risk assessment; Product life cycle risk evaluation, long-term releases to soil, sediments, groundwater, surface waters, air (occurrence of fractions varies by medium)	Allows evaluation of data using established mechanistic models and tools for aquatic toxicity; Could be modified to develop sediment screening levels;	Requires more advanced and specialized analytical methods that are not available through commercial laboratories; Cannot be used for terrestrial receptors at this time; Not commonly used in the United States at this time; very volatile fraction, not expected to be an ecological risk in water or soil.
C6–C8 (Ali or aro)			
C>8–C10 (Ali or aro)			
C>10–C12 (Ali or aro)			
C>12–C16 (Ali or aro)			
C>16–C21 (Ali or aro)			
C>21–C34 (Ali or aro)			

Table 7-4. Constituent-based analytical data that can be used for ecological risk assessment

TPH Analyte	Analytical Method(S)	When to Use	Strengths	Limitations
Chemical Group — Low molecular weight (LMW) PAHs, High molecular weight (HMW) PAHs	EPA 8270	Screening and site-specific risk assessment; releases of diesel and heavier petroleum products to soil, sediments, surface water; long-term remediation and monitoring needs	Allows evaluation of PAH-specific toxicity and potential for bioaccumulation for a range of media and receptors	Reliable toxicity values for PAHs not available for birds
Indicator/surrogate compound — BTEX	EPA 8260	Screening-level and site-specific risk assessment; releases of fresh gasolines and lighter petroleum products to surface waters, and soil vapor (burrowing mammals)	Allows chemical-specific evaluation; chemical-specific toxicity data readily available for many receptor types	Evaluation of volatile constituents is not relevant for ecological receptors in ambient air settings
Surrogate compound — Individual PAHs (e.g., Naphthalene, Pyrene)	EPA 8270			Reliable PAH toxicity data not available for certain receptor types, such as birds
Additive — MTBE, Tertiary amyl methyl ether (TAME)	EPA 8260	Screening-level and site-specific risk assessment; include as needed, depending on type and age of petroleum product released		
Additive — Tetraethyl lead	EPA 8270M and GC-ECD (electron capture detection)			
Interim degradation compounds — Naphthenic acids and other polar metabolites	No readily available analytical methods from commercial labs	When potential may exist for groundwater to enter surface water, during screening-level and site-specific evaluation	Most conservative approach to consideration of TPH degradation process; may be addressed at screening level by using non-SGC data, per some agency recommendations	Insufficient understanding of toxicity potential; no widely accepted regulatory guidance or requirements at this time; this area is under development

7.3.7 Quantitative vs. Qualitative Options for Exposure Assessment

Typically, ERAs are conducted in a quantitative manner and include calculation of exposures and risks ranging in complexity level from simple screening assessments to probabilistic multimedia and multiple lines of evidence evaluations. Most TPH sites would likely fall under the simpler, screening-level type assessments on a limited number of trophic levels (e.g., benthos, soil invertebrates, etc.) given that hydrocarbons readily degrade and are generally not persistent in the environment and/or the food webs/chains. Moreover, TPH ERAs would more likely focus on specific potential risk drivers or groups thereof (e.g., PAHs) for which toxicity reference values are better defined.

7.4 Toxicity Assessment

Toxicity assessment for ERAs can be challenging and highly variable because the assessment must consider toxicity to a wide range of receptors and groups and extrapolate toxicity data to protect populations or communities rather than individuals (except for specific cases). Additionally, for TPH, there is no single source of peer-reviewed or agency-approved

toxicity values for ecological receptors, which, in part, stems from the complexity of TPH toxicity as discussed below.

Reference toxicity values for ecological receptor communities are based on both No Effects and Lowest Effects levels USEPA 1997a. For plants, invertebrates and fish are typically reported as lethal concentrations (LC) and sub-lethal effective concentrations (EC) that affect some percentage of the exposed population, LC and EC refer to the exposure concentration of a chemical at which a health response is noted in the specified percentage of the population, e.g., mortality in 50% of test fish (LC50).When more protective values are desired, the LC or EC values may be reported to a lower percentage of the exposed population such as 10% or 25% (e.g, LC10, EC25). When reference toxicity values are reported for higher level receptors such as birds and mammals, the exposure dose is referred to a No Observed Adverse Effect Level (NOAEL) or Lowest Observed Adverse Effect Level (LOAEL).

The USEPA and most states do not specify a single recommended toxicity value for TPH and the associated chemicals for ecological risk. However, compilations of studies and toxicity values are available in various databases such as EcoTox and the Risk Assessment Information System, and the agencies may select certain values as the basis for the development of water quality criteria or ecological soil screening levels.

When available toxicity data are insufficient or inappropriate for use for risk assessment of TPH at a given site, product-specific or site-specific toxicity testing can provide a means to assess a site's unique TPH mixture or species sensitivity. However, toxicity testing of petroleum compounds has many limitations and technical challenges Singer et al. 2000; Redman and Parkerton 2015 that can confound results, and the presence of non-TPH chemical constituents or other stressors (e.g., nutrients) in site media can complicate site-specific testing. As an alternative, mechanistic models provide a means to develop toxicity thresholds or evaluate risk using site-specific conditions without some of the challenges of toxicity testing (see Overview of Toxicity Prediction by Mechanistic Models).

Figure 7-1 illustrates the two most common categories of methods for developing ecological toxicity threshold values for TPH and their associated uses: thresholds derived from mechanistic toxicity prediction models (which have been validated by laboratory test results), and thresholds derived from toxicity tests reported in the literature or from site-specific testing. Both model-based toxicity values and toxicity test-based toxicity values can be valuable in TPH ERA.

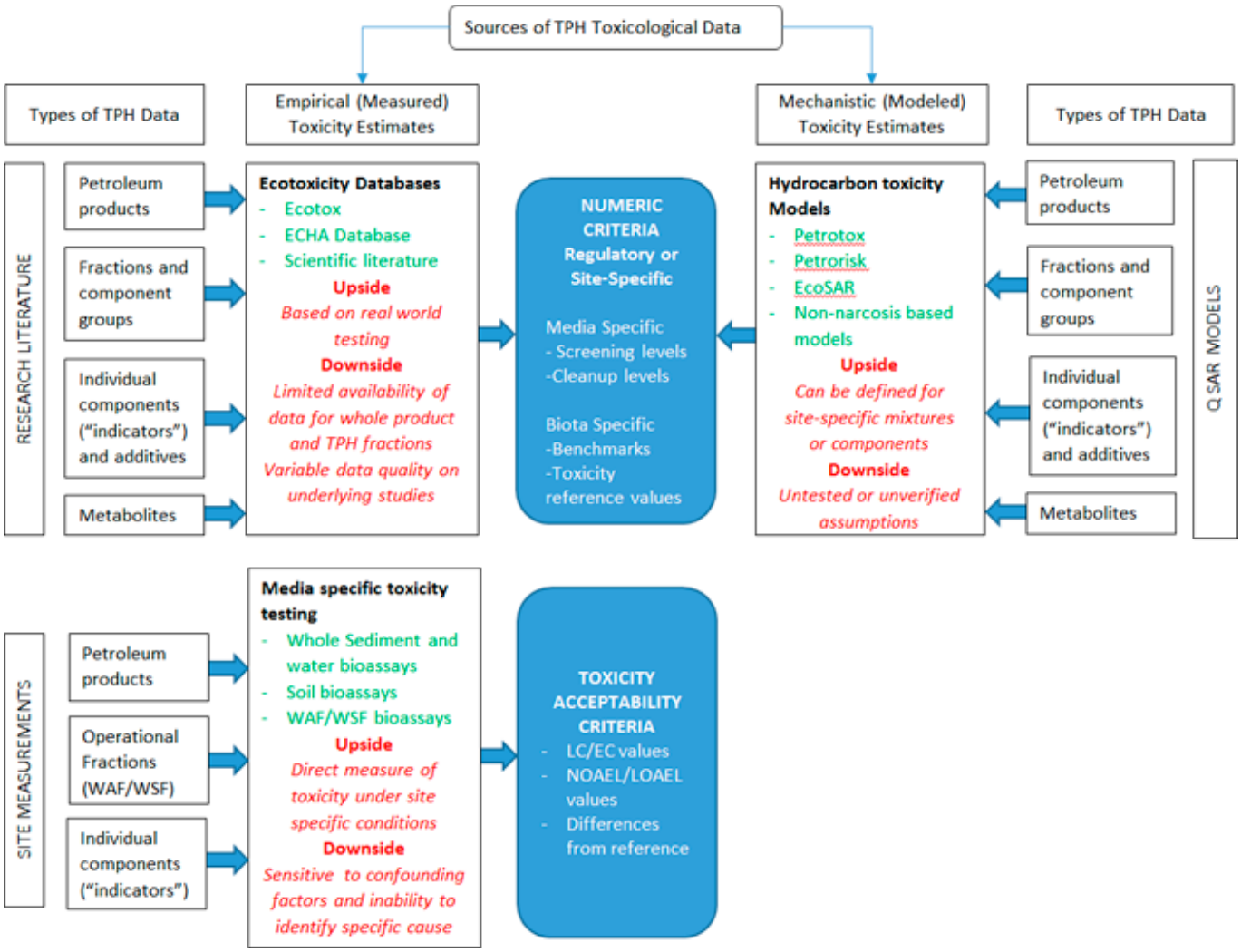

Figure 7-1. Sources, types, and uses of TPH toxicological data for ERA.
(Source: B. Bjorkman, GEI Consultants, 2018.)

7.4.1 Overview of TPH Toxicity to Various Taxa

TPH toxicity to ecological receptors depends on its hydrocarbon composition as well as the exposure pathway and duration of exposure Albers 2003. Additionally, TPH in the form of product (e.g., crude oil, heavy fuel oil) can cause physical and chemical toxicity. Toxicity associated with exposure to high concentrations of TPH typical of an accidental release is both physically and chemically mediated and is very different from the subtle and lower magnitude of effects associated with exposure to chronic low-level releases, which is primarily the result of chemical toxicity. Because this guidance is focused on evaluating the risk of the TPH fraction of petroleum and not the petroleum product itself, the focus of this section is on the chemical toxicity of TPH and the more extensively studied mechanisms of chemical toxicity upon which mechanistic toxicity models are based.

Examples of toxicity end points of TPH, TPH fractions, and operational fractions for ecological receptors are summarized in Tables 7-5 and 7-6. This table highlights some of the reported end points, many of which are the basis for screening-level values and toxicity reference values, but is not a comprehensive list of all relevant toxicological end points or all end points reported in the literature. For the purposes of this table, WAF, WSF, and chemically enhanced water-accommodated fraction (CEWAF) are defined as described in Adams et al. 2017. The water-accommodated fraction (WAF) is generated by low-energy mixing of oil floating on water, with little disturbance of the surface slick. Test solutions contain dissolved hydrocarbons and potentially a small fraction of particulate oil. The water-soluble fraction is WAF that has been filtered or treated to remove all oil droplets and contains only truly soluble fractions of each test oil. CEWAF is generated by mechanical and chemical dispersion of floating oil. Test solutions contain dissolved hydrocarbons and a significant amount of particulate oil.

▼*Read more*

Table 7-5. Examples of TPH toxicity end points noted for aquatic biota.

Receptors	Toxicity End Points
Algae and phytoplankton	Changes in growth rate, photosynthesis, cell counts, survival, generation time, chlorophyll a, germination, community composition, and respiration in whole product and WAF/WSF/CEWAF studies Lewis and Pryor 2013
Aquatic (zooplankton) and benthic invertebrates	Smothering resulting in mortality in benthic invertebrates Teal and Howarth 1984, reduction in filtration and fecal production in copepods, possibly due to mechanical disruption/clogging of feeding apparatus and reduction in egg production and hatching due to droplet oil Nordtug et al. 2015 **Zooplankton:** Mortality, reduced or delayed growth and reproduction, developmental effects, changes in community composition exposed to WAF/WSF/ CEWAF Barron et al. 1999; Hemmer, Barron, and Greene 2011; Gardiner et al. 2013; Neff et al. 2000; Fucik, Carr, and Balcom 1994; Valentine and Benfield 2013 **Benthic invertebrates**: mortality, impaired feeding, decreased growth, development and recruitment from exposure to sediment-containing fresh and weathered product Lee et al. 2015 **Corals**: inhibition of fertilization, developmental effects, mortality, tissue damage, branch loss, bleaching observed in WAF/WSF/CEWAF exposures or post-spill observations NRC 2005; Beyer et al. 2016 **Bivalves**: Reduced growth, immune function impairment, decreased feeding, impaired embryo-larval development observed in WAF/WSF/CEWAF exposures Baussant et al. 2011; Clark et al. 2001; Kirby et al. 2007; Renzoni 1975; Luna-Acosta et al. 2011
Fish	**TPH fraction exposures**: Waxes in heavy fuel oil (HFO) were not chronically toxic, no increase in mortality or signs of blue sac disease (BSD) in trout; 3- and 4-ring PAHs in HFO caused increase in mortality and deformities; 2-ring PAHs in HFO caused a mild increase in mortality, BSD, and deformities with chronic treatment Bornstein et al. 2014 **WSF/WAF/CEWAF exposures**: Adults: Lesions, impaired immune function, reduced or delayed growth and reproduction Lee et al. 2015 Early life stages (embryo, larvae): Mortality, impaired cardiac function, impaired immune function, reduced respiration and heart rate, abnormal development, reduced growth, premature or delayed hatching, DNA alteration Lee et al. 2015

Table 7-6. Examples of TPH toxicity end points noted for terrestrial biota

Receptors	Toxicity End Points
Terrestrial vegetation	Mortality from smothering, reduced growth, reduced biomass, reduced photosynthesis, inhibition of seed germination in whole product studies or post-spill observations; mangroves are particularly sensitive compared to other marsh vegetation Lewis and Pryor 2013; Mendelssohn 2012; DeLaune and Wright 2011 Reduced shoot length, root length, and biomass with F1(fraction) (C6–C10) and F2 (C10–C16) of TPH, and less toxicity with F3 (F16–F34) and F4 (C34C50) CCME 2008
Terrestrial invertebrates	Mortality, reduced adult fecundity and number of juveniles—F1 (C6–10) and F2 (C10–16) were substantially more toxic than F3 (C16–C34) and F4 (C34C50) CCME 2008
Birds	Whole product: External exposure: Changes in metabolic rate and thermoregulation leading to hypothermia, behavioral changes, reduced hatching success (egg exposure), decreased buoyancy, drowning, suffocation, dehydration, starvation, lung irritation (inhalation of volatile components) NRC 2005; Lee et al. 2015; Bursian, Alexander, et al. 2017 Dietary exposure: Sub-lethal physiological effects such as hemolytic anemia, decreased nutrient absorption, altered stress response, decreased immune function Jenssen 1994; Bursian, K. M. Dean, et al. 2017

Receptors	Toxicity End Points
Mammals (aquatic and terrestrial)	Product exposure: Lung disease, physiological and pathological changes, disruption of thermoregulation and hypothermia (furred mammals), irritation of mucous membranes in eyes, nose, and mouth, mortality (physical fouling) Engelhardt 1983; Helm et al. 2014
Reptiles (aquatic and terrestrial)	Product exposure: Mortality, irritation of mucous membranes in eyes and mouth, lung disease, reduced hatching success (egg exposure) Lutcavage et al. 1995; Lee et al. 2015 Skin sloughing, necrosis, acanthosis, hyperkeratosis, and dermal hemorrhage (exposed to oil in water) Zychowski and Godard-Codding 2017
Livestock	Dietary exposure: Mortality; liver, gastrointestinal, hematological, and neurological effects Pattanayek and DeShields 2004

7.4.2 Mechanisms of Chemical Toxicity of TPH

The major mechanisms by which TPH compounds are understood to exert toxicity to ecological receptors include narcosis and phototoxicity. The chemical toxicity is considered to be additive in nature and described in more detail below.

▼*Read more*

7.4.2.1 Narcosis

Narcosis is defined as a general depression of biological activity from exposure to a nonspecifically acting toxicant. Narcosis due to TPH may be associated with both nonpolar compounds (narcosis I) and polar compounds (narcosis II) by mechanisms that are not necessarily similar and additive Roberts and Costello 2003. Hydrocarbons quantified for ERA by common analytical methods are considered nonpolar narcotics. The mode of nonpolar narcotic action is thought to be based on partitioning to and disruption of lipid-containing membranes by the narcotic chemical and subsequent cell function disruption/transport into the interior of the cell Schultz 1989. The potency of a narcotic chemical is related to its lipophilicity or tendency to partition into the lipids. A chemical's octanol-water partition coefficient, or K_{ow}, is used as a model for this behavior and, for narcosis, it correlates inversely with the concentration of the chemical required to cause narcosis, subject to model constraints Di Toro and McGrath 2000; Di Toro, McGrath, and Hansen 2000.

7.4.2.2 Phototoxicity

Phototoxicity (i.e., ultraviolet (UV)-enhanced, photoactivated, photoenhanced toxicity) is the increase in toxicity caused by exposure to UV radiation or light and a chemical. Photosensitization is the formation of excited triplet PAHs by photon absorption in the tissues of an organism, which can transfer energy to form reactive oxygen species, resulting in oxidative stress and oxidative damage Diamond 2003. Photomodification is the formation of new products from the oxidation of PAHs, which exhibit their own chemical toxicity. The relevance of phototoxicity to ecological risk at a site depends on the site-specific conditions, including penetration of UV light through the water column, photodegradation of PAHs in the water column, the sensitivity of the receptors of interest (e.g., presence of pigment to mitigate UV light penetration into the tissues), and their predicted accumulation of PAHs.

Phototoxicity is primarily considered applicable to relatively transparent aquatic invertebrates and early life stage fish (e.g., eggs, embryos, and larvae) located in the upper portion of the water column. For TPH, phototoxicity is generally thought to be associated with the PAH fraction Diamond 2003. Photoactivation of PAHs is dependent upon the UV wavelength and duration and intensity of exposure. Both UVB and UVA light in the range of 280–400 nm can reach aquatic systems. Most PAHs absorb UV light in the UVA range of 320–400 nm. There are two mechanisms of phototoxicity: photosensitization and photomodification.

7.4.2.3 Additivity and Toxic Units

Whether hydrocarbons in TPH range exhibit additive toxicity depends on the mechanism of action. For nonpolar narcosis, effects are observed once the concentration of total narcotic chemical in the lipid membrane reaches an organism's critical threshold, which is the same regardless of the identities of the narcotic chemicals in the lipid. This means that nonpolar narcosis is the result of the additive dose of each narcotic chemical present Di Toro, McGrath, and Hansen 2000. (Note: The differences in narcotic chemical potency are related to a chemical's ability to reach the lipid membrane [i.e., its lipophilicity].) Data support that phototoxicity is also additive Diamond 2003; Boese et al. 1999.

The additive nature of TPH mixture toxicity is commonly expressed using a toxic unit (TU) approach Di Toro and McGrath 2000. A TU is the concentration of a chemical/TPH fraction in a medium divided by the effects concentration (e.g., EC10, LC50) of that chemical/TPH fraction in that medium Di Toro and McGrath 2000. The toxicity of the mixture is the sum of the TUs (sum TU) for each individual chemical or TPH fraction. A sum TU of >1 suggests that the TPH mixture may pose a risk to the environment. Note that the environmental concentration in a TU calculation should not exceed the solubility of the chemical in the medium (e.g., aqueous solubility in freshwater).

7.4.3 Overview of Toxicity Prediction by Mechanistic Models

Predictive tools to estimate the ecological toxicity of TPH have been developed and used by regulatory agencies as well as professional and academic organizations. In general, predictive approaches use a combination of the physical and chemical properties of the chemicals and an assumed narcosis mode of action to predict the toxicity of a particular constituent or fraction. Acceptable concordance of model-predicted effects and no-effects concentrations with toxicity values derived from laboratory studies has been reported for test species for aquatic and sediment tests McGrath et al. 2005x; McGrath and Di Toro 2009; Redman et al. 2012; USEPA 2003. This section describes a few of the most commonly used predictive approaches.

A brief overview of the strengths and limitations associated with the models for prediction of TPH toxicity is provided in Table 7-7. It is important that the user be aware of the foundation and basis of the model of choice, the inherent assumptions and uncertainties, and the intended uses.

Table 7-7. Summary of advantages and limitations for ecotoxicity modeling tools

Description/Usage	Advantages	Limitations
Narcosis Target Lipid Model		
Most commonly applied in sediment risk assessment	Generally accepted by agencies after discussion	There may be multiple modes of toxic action in addition to narcosis, some of which can be accounted for by applying an acute to chronic ratio (ACR).
PETROTOX		
Primarily for surface water; can be adjusted for groundwater discharge to surface water by selection of input options; should use most current version of model and user manual	Includes acute and chronic toxicity data for 42 freshwater and saltwater test species of algae, invertebrates, and fish; uses robust species sensitivity distribution approach; can back-calculate acceptable TPH levels for any desired percentage of exposed taxa	Primarily intended to model toxicity for product-based scenarios; requires some model adjustments to apply to contaminated site evaluation; works best with high resolution fractional data; recommend discussion with agencies prior to use; not all agencies accept species sensitivity distribution approach; chronic effects are estimated more by the use of ACRs than chronic toxicity data
PETRORISK		
Can be used to estimate multimedia ecological and human health risks for exposure to petroleum products during the product life cycle; incorporates physical/chemical model properties from PETROTOX	Similar to PETROTOX	Similar to PETROTOX
ECOSAR/EPI Suite		

Description/Usage	Advantages	Limitations
Narcosis Target Lipid Model		
ECOSAR estimates acute and chronic toxicity based on narcosis model; uses EPI Suite to develop physical/chemical properties for fate and transport and intermedia transfer calculations	Generally accepted by agencies; includes TPH aliphatic and aromatic hydrocarbons; most useful and robust for freshwater algae, invertebrates, and fish;	Aquatic receptors only; very little information for saltwater species; requires match-up between analytical data and chemical classes included in the program
QSAR		
Similar to ECOSAR	Accepted by agencies	Similar to ECOSAR, some QSAR models and applications have extensive learning/knowledge requirements

▼*Read more*

7.4.3.1 Predictive Model Descriptions

Narcosis-based modeling used in conjunction with equilibrium partitioning modeling predicts the toxicity of TPH based on chemical properties, primarily the octanol-water partition coefficient (K_{ow}), as well as the aqueous solubility and other related parameters that determine the aqueous (i.e., bioavailable) concentrations of hydrocarbons to which an organism would be exposed under specific conditions. Narcosis theory states that the toxic effect (e.g., mortality) occurs when the molar concentration of narcotic chemical(s) inside the body (or target lipid) reaches a critical threshold and that the threshold is species-specific. The differences in toxic potential of narcotic chemicals such as PAHs and other petroleum hydrocarbons are primarily due to variations in the chemical attributes affecting the transport of the chemical into the body and, ultimately, to cellular sites of adverse action (e.g., target lipid).

Thus, the TPH constituents that have the greatest potential for ecological toxicity and/or bioaccumulation are those constituents or fractions that are soluble enough, are persistent enough externally and internally, and have sufficient affinity for partitioning into living tissues (lipid transfer). These tend to be the constituents that fall into the carbon range of approximately C9–C16 for both aliphatic and aromatic fractions. Some toxicity has also been noted for the C5–C9 range. Aliphatic petroleum constituents exceeding the C16 carbon range are generally considered to be relatively more inert (low solubility and large molecular size) and their toxic effects are far less significant.

7.4.3.2 Narcosis Target Lipid Model

The narcosis target lipid model (TLM) was developed by Di Toro et al. 2000 to predict the toxicity to aquatic organisms of nonpolar chemicals that act via narcosis. The TLM is a Quantitative Structure-Activity Relationship (QSAR) model that relates the species-specific, target lipid threshold of narcotic chemical for a toxic effect to the aqueous concentration of an individual chemical or chemical mixture that would result in that effect based on the log K_{ow} of the chemical(s). Some of the key assumptions of the model are that the target lipid is the site of toxic action within the organism, octanol is an appropriate surrogate for lipid, and the target lipid has generally the same physical-chemical properties in all organisms McGrath, Parkerton, and Di Toro 2004.

Although narcosis may affect all organisms, including birds and mammals, it has been applied mostly to predict the toxicity of nonpolar organics, including petroleum hydrocarbons, to aquatic species, which include algae, invertebrates, and fish. Numerous researchers have demonstrated reasonable agreement between TLM-based toxicity predictions and standard toxicity test results such as LC50s and EC50s for acute toxicity Redman et al. 2012; Redman et al. 2017; Swigert et al. 2014; Knap et al. 2017. They have also evaluated the utility of applying ACRs to acute narcotic toxicity to predict chronic and sub-lethal toxicity Redman et al. 2012; Redman, Parkerton, Comber, et al. 2014; Redman, Parkerton, Paumen, et al. 2014; McGrath and Di Toro 2009. The model has been used to predict the toxicity of individual chemicals as well as petroleum mixtures.

Di Toro et al. 2000 and the USEPA 2003 extended the TLM to develop Final Chronic Values and sediment quality guidelines for PAHs by applying equilibrium partitioning theory USEPA 2003, which states, in this case, that a nonpolar chemical would partition between a benthic organism, sediment pore water, and organic carbon. The USEPA Equilibrium Partitioning Sediment Benchmarks (ESBs) for PAH mixtures at petroleum sites have been shown to be protective of benthic organisms

USEPA 2003; Kreitinger et al. 2007; McDonough et al. 2010.

7.4.3.2.1 PETROTOX

Detailed information regarding PETROTOX can be found at https://www.concawe.eu/reach/petrotox.

> ***Example: Use of PETROTOX to Develop Aquatic Toxicity Benchmarks for TPH Fractions***
>
> Atlantic PIRI (2012a, b) developed acute (LC50) and chronic No Observed Effects Level (NOEL) aquatic toxicity benchmarks for several aliphatic and aromatic fractions for 42 species using the PETROTOX model. Using these data, species sensitivity distributions were created to select species representing the 5th and 50th percentile in chronic sensitivity and 20th percentile in acute sensitivity. Aquatic toxicity benchmarks for each fraction for these species were used to develop ecological screening levels for the protection of freshwater and marine aquatic life for BTEX, gasoline, diesel, and #6 lube oil. For additional details, refer to Atlantic PIRI 2012a, 2012b

The PETROTOX model is a program developed by Redman et al. 2012, and Conservation of Clean Air and Water in Europe (CONCAWE) to estimate the toxicity of TPH. PETROTOX uses a three-phase partitioning model (water, product, air) to estimate exposure concentrations of TPH fractions in water linked to the TLM to develop effects concentrations in water associated with a target effect level such as narcosis/mortality. A useful feature of PETROTOX is that it identifies when a toxic concentration may actually exceed the solubility limit of a given TPH. PETROTOX features two types of petroleum compositional input data: high and low resolution. High resolution input is based on two-dimensional gas chromatography data and allows for input of mass fraction across 16 structural classes of hydrocarbons separated into user-defined carbon ranges. Low-resolution input is designed for input of mass fractions of aliphatic and aromatic hydrocarbons separated into user-defined boiling point ranges. A user who desires risk characterization at the most detailed level of TPH data would select the high-resolution inputs, although such data typically require research-level laboratory instrumentation that is typically not available at a commercial scale. The low resolution input scale allows risk characterization for fractions that can be tailored to the needs of a specific project.

7.4.3.2.2 PETRORISK

Further information regarding PETRORISK can be found at https://www.concawe.eu/reach/petrorisk CONCAWE 2017.

PETRORISK is a spreadsheet tool developed for CONCAWE that performs environmental risk assessments for TPH using principles provided by the European Chemicals Agency (ECHA). It is designed to evaluate environmental exposure and ecological risks for a wide range of petroleum products, ranging from naphtha (gasoline), kerosene, and gas oils, to heavy fuel, lubricant oils, and hydrocarbon-based solvents. The spreadsheet tool can evaluate risks associated with different stages in the product life cycle. For example, the ecological risks can be evaluated at the production, formulation, and distribution stages, as well as for generic uses in industrial, professional, and consumer use sectors. The toxicity values are based on predicted no-effect concentrations derived from the TLM McGrath, Parkerton, and Di Toro 2004. Petroleum compositional input data required for PETRORISK is the same as for PETROTOX.

7.4.3.2.3 ECOSAR/EPI Suite

Detailed information about ECOSAR/EPI Suite can be found on the USEPA website: https://www.epa.gov/tsca-screening-tools/ecological-structure-activity-relationships-ecosar-predictive-model.

The USEPA has developed predictive tools to provide users with estimates of physical/chemical properties, as well as environmental fate of various chemicals, such as those included in TPH. The USEPA's EPI Suite is one such screening-level tool. The EPI Suite is a Windows-based tool developed by USEPA and Syracuse Research Corp. Included in EPI Suite is a module called ECOSAR.

ECOSAR is a QSAR-based tool that predicts aquatic toxicity of known chemicals or substances with characterized physicochemical properties, a feature useful when addressing complex mixtures such as TPH. It was developed to predict aquatic toxicity primarily for the rapid assessment of industrial chemicals under the Toxic Substances Control Act in 2012 and updated in 2017 USEPA 2017b. ECOSAR contains a library of class-based QSARs for predicting aquatic toxicity, overlaid with an expert decision tree for selecting the appropriate chemical class. ECOSAR Version 1.11 is programmed to identify 111 chemical classes and allows access to 704 QSARs for numerous end points and organisms. ECOSAR is useful for TPH chemicals because these chemicals fall under the category of neutral organic chemicals that are nonionizable and nonreactive and act via simple nonpolar narcosis generally thought of as a reversible, drug-induced loss of consciousness

(general anesthesia or narcosis). This toxicological response is often referred to as baseline toxicity Franks and Lieb 1990; Veith and Broderius 1990 and is the overarching concept in ecological risk assessment of TPH.

7.4.3.2.4 Organization for Economic Co-operation and Development (OECD) QSAR Toolbox

Detailed information can be found on the OECD website:

http://www.oecd.org/chemicalsafety/risk-assessment/oecd-qsar-toolbox.htm

QSAR models can also be used to predict biological activity based on structures of chemicals. The OECD has developed QSAR Toolbox, which is a software application intended for the use of governments, the chemical industry, and other stakeholders in filling gaps in (eco)toxicity data needed for assessing the hazards of chemicals. QSAR Toolbox can accommodate individual TPH substances or specific mixtures. The modeling exercise allows for the development of predicted toxicity profiles for untested target chemicals by inputting information on their presumed chemical structure. The program then retrieves information from known chemicals with similar structures and properties to predict toxicity effects to a variety of test species for the untested target compound. A number of helpful tutorials are provided.

7.4.3.3 Use of Indicator and Surrogate TPH Compounds

Toxicity assessment based on the concepts of indicator and surrogate chemicals is often used in risk assessment, but they are not identical and serve different purposes.

For chemical mixtures, an indicator chemical(s) is often used to account for the main toxic chemical of the mixture. That is, if the risk from the indicator chemical is acceptable, then risk from the mixture is also assumed to be acceptable. An example of this is the use of PAHs as indicator compounds for assessment of the toxicity of petroleum substances in sediments. However, the USEPA and others have demonstrated that sediment toxicity at several sites is conservatively predicted by bulk sediment PAH concentrations, and the estimation of pore water concentrations using equilibrium partitioning theory, or better still the direct measurement of freely dissolved PAH concentrations in sediment pore water, can provide a more accurate indication of toxicity to benthic organisms USEPA 2003, 2012b; Kreitinger et al. 2007; McDonough et al. 2010.

For TPH in soils, surrogate chemicals with similar chemical structures to the other chemicals in the mixture or fraction are also often used in ERAs when toxicity values for the detected chemicals are lacking. Surrogate chemicals are used to account for the toxicity of the TPH mixture or a TPH fraction. For example, USEPA developed Ecological Soil Screening Limits (Eco-SSLs) for low molecular weight (LMW) and high molecular weight (HMW) PAHs using surrogate chemical data. Naphthalene is similar to other LMW PAHs in chemical reactivity, environmental fate, and toxicological effects. Therefore, naphthalene screening levels and toxicity reference values (TRVs) were used as surrogates for all LMW PAHs and applied to the total concentration of LMW PAHs. Likewise, screening levels and TRVs developed for a HMW PAH, such as benzo(a)pyrene or 7,12-dimethylbenz(a)anthracene, were used to approximate toxicity of all HMW PAHs on equipotent basis. Although some approaches may select the most toxic chemical to represent an entire fraction, such a conservative approach may overestimate risks, especially when sufficient analytical data are available regarding the presence or absence of the selected compounds Vedagiri and Curren 2014.

7.4.3.4 Toxicity of Polar Degradation Products to Ecological Receptors

Approaches to evaluating the toxicity of polar degradation compounds are still evolving due to lack of data on their potential toxicity and persistence. For a detailed discussion about the nature of polar degradation products and challenges of evaluating their toxicity see Section 6.10.

As noted in TPH Fundamentals, polar degradation products are primarily produced by biodegradation but can also be produced by chemical degradation such as photooxidation. For ecological receptors, photooxidized products can be formed in environmental media (i.e., photomodification) or within the tissues of an organism (i.e., photosensitization), potentially resulting in phototoxicity. In this section, the focus is on new polar, photooxidized degradation products produced in environmental media that have their own inherent properties and toxicity.

Environmentally relevant studies on the ecotoxicity of polar metabolites are still developing, especially for refined product releases. Although some studies are available regarding the potential ecological toxicity of some of the polar degradation products Rogers et al. 2002; Mao et al. 2009a; Melbye et al. 2009; Neff et al. 2000; Thomas, Donkin, and Rowland 1995; Wolfe et al. 1995, only limited data are available and research is ongoing to fill some of these data gaps Lundegard and Knott 2001; Mao et al. 2009a, 2009b; Zemo, O'Reilly, et al. 2013; Zemo et al. 2017. Results from these studies suggest that

overall toxicity appears to decrease with increased transport distance or with degree of weathering (time). A literature review of these studies has recently been published in a State of California guidance document CASWB-SFBR 2016b.

Two approaches are currently used to assess the toxicity of polar degradation products to ecological receptors. The first approach is to assume that polar degradation products in a TPH mixture have toxicity that is similar to or less than the toxicity of the parent hydrocarbons CASWB-SFBR 2016a; HIDOH 2017. Under this approach, sample data generated by analytical methods that do not selectively remove polar compounds (i.e., without the use of silica gel cleanup, or non-SGC TPH data) are directly compared to TPH screening levels for toxicity. This approach is typically recommended for initial site screening purposes.

The second approach is to separate polar degradation products and petroleum hydrocarbons in environmental samples and conduct toxicity testing on the extracts to assess the toxicity of site-specific mixtures. Soils and sediments contain naturally occurring polar organic compounds, particularly if the media are rich in organic material, and these compounds should be considered when evaluating the toxicity of polar degradation products in soils and sediments at petroleum release sites. A comparison of the toxicity observed at background or reference sites with similar site characteristics to that of site samples may provide information about the contribution of naturally occurring polar organic compounds to toxicity of site samples.

A third approach may be possible in the future and could reduce the need for toxicity testing of site media. This approach, as is described for human health risk assessment in Section 6, assigns toxicity factors to individual polar degradation products, which then represent the toxicity of chemical structural families of degradation products Zemo, O'Reilly, et al. 2013. The primary limitation of this approach for assessment of toxicity to ecological receptors is that data are limited to determine and assign toxicity factors that would represent the different classes of receptors or even the most common classes of ecological receptors (e.g., fish, aquatic invertebrates), but research is ongoing to fill these data gaps.

7.4.3.5 Toxicity Testing Methods

Toxicity testing methods are available to evaluate TPH toxicity in the context of releases that have already occurred, as well as to predict toxicity if a release were to occur in the future. In general, there are more established toxicity testing methods for TPH impacts to aquatic and benthic species than for terrestrial species.

7.4.3.5.1 TPH Toxicity Testing

Ecological toxicity associated with TPH can be directly evaluated using a variety of test media, species, and experimental design/conditions. A vast array of toxicity tests is available. They have been used for testing of various exposure media, such as soils, bulk sediments, surface water, pore water, WAFs, and WSFs. The nature of the source TPH may be fresh or weathered, whole products or residues, fractions or individual constituents. The test species may include terrestrial plants and seedlings, terrestrial invertebrates, such as earthworms, and aquatic and benthic species, such as algae, invertebrates, and fish. Both freshwater and saltwater test species have been used.

Given the complex mixture of TPH, interpreting toxicity tests can be challenging. Toxicity tests must be conducted such that the actual exposure concentration and composition can be measured to extrapolate the data to site-specific conditions. This is particularly important when testing the toxicity of petroleum products to aquatic organisms where the dissolved hydrocarbons in the WSF or WAF may be challenging to quantify Redman and Parkerton 2015. Although there is no one compendium of recommended test species and procedures for toxicity testing of petroleum hydrocarbons, guidelines for standardized aquatic toxicity test methods for petroleum substances have been developed and published Redman and Parkerton 2015; Aurand and Coelho 2005; Singer et al. 2000; Barron and Ka'aihue 2003.

Guidelines for toxicity testing of chemical constituents in environmental media, such as water, soil, or sediments, have been developed by numerous international and national regulatory agencies, including the OECD 2017 and USEPA 1994. In designing site-specific toxicity tests, it is useful to include reference locations (locations not impacted by the TPH release or that of other hazardous substances) to account for natural variability that might impact test results and lead to a potential false positive (i.e., conclusion that TPH release causes toxicity) in regards to test interpretation.

7.4.3.5.2 Whole Effluent Toxicity Testing

There may be concerns about TPH toxicity to aquatic biota residing in surface water if TPH-containing groundwater were to discharge into surface water bodies. Whole Effluent Toxicity (WET) testing can be performed at TPH sites to assess the spill-related aquatic toxicity of contaminated groundwater discharging to surface water CASWB-SFBR 2016a; Zemo, O'Reilly, et al. 2013; Chakrabarti 2018. WET testing measures the combined effect of a complex effluent (mixture) where it is typically infeasible to measure all individual compounds (and where single-chemical toxicity information is lacking). WET testing

methods are a component of the National Pollutant Discharge Elimination System (NPDES) permit program, authorized by the Clean Water Act, that controls water pollution by regulating point source discharges into waters of the United States SETAC 2004; USEPA 1991, 2000a. Specific considerations for WET testing of TPH mixtures include testing a background groundwater sample (unimpacted by the TPH spill in an upgradient or nearby location that has similar hydrogeologic and vegetative characteristics) and monitoring the TPH concentration of the groundwater during the test protocol to evaluate whether there is significant loss through volatilization or biodegradation. Site-specific examples of WET testing can be found in the literature Chakrabarti 2018.

7.4.4 Toxicity Basis for Screening and Site-Specific ERA

There is intentional difference in the level of representativeness and conservatism associated with the toxicity criteria and values used at the screening-level and site-specific level of risk assessments.

7.4.4.1 Toxicity Basis TPH Screening Levels

Screening levels for ecological risk assessment are discussed in Section 7.2. Regulatory screening values for TPH are available for surface water, and to a lesser degree, for soil and other media. The screening levels are generally based on direct toxicity, such as protection of algae, invertebrates, and fish that may be directly exposed to TPH in surface water. Soil screening levels for the protection of terrestrial vegetation have been published in Canada CCME 2008. The sources and links provided in Tables 7-8 through 7-10 may also be checked to obtain the most current version of available toxicity data for screening levels. It is noted that the information and sources shown in these tables may change considerably over time as new data become available.

7.4.4.2 Toxicity Reference Values for Site-Specific ERA

The availability of toxicity data for TPH greatly influences the depth and breadth of ecological risk assessment that can be performed. Ecological toxicity data are available and constantly updated for various TPH fractions (see Table 7-8). The sources of information include government agencies, industry associations, academic research, and other technical and "gray" literature.

Tables 7-8 through 7-10 provide a listing of sources and relevant analytical constituents for each of the products, fractions, and constituents for which ecological toxicity information is available for both freshwater (FW) and saltwater (SW) species. This summary is based on data that were readily available online as of January 2017. Because toxicity data are constantly being updated and reinterpreted, the purpose of this table is not to provide a master compilation of toxicity reference values, but rather to identify sources where the most up-to-date information may be available for the constituent and species of interest. The information provided should be used with caution and with an understanding of the assumptions and uncertainties. It is recommended that the primary literature also be accessed and reviewed, as appropriate, by an experienced ecotoxicologist/ecological risk assessor.

▼*Read more*

Table 7-8. Sources of available ecological toxicity values for TPH products and fractions

TPH Analyte	Receptor/Citation (See Table 7-10 for citations)
Gasoline	Aquatic algae (FW/SW) – a/a,b; aquatic invertebrate (FW/SW) – a,b/a,b,f; fish (FW/SW) –a/a,f; terrestrial plants –f; terrestrial invertebrates – f; mammals –f
Diesel	Aquatic algae (FW/SW) – a/a,b; aquatic plants (FW/SW) – □; aquatic invertebrate (FW/SW) – a,b/a,b; fish (FW/SW) – a/a,b; terrestrial plants – f; avian – f
Motor oil	Aquatic algae (FW/SW) – a/a; aquatic invertebrate (FW/SW) – a/a; fish (FW/SW) –a/a
Fuel oil	Aquatic invertebrate (FW/SW) – b/b; fish (FW/SW) – □/b
Jet fuel A, B	□

TPH Analyte	Receptor/Citation (See Table 7-10 for citations)
Crude oil	Aquatic algae (FW/SW) – f/b,f; aquatic plants (FW/SW) – f/f; aquatic invertebrate (FW/SW) – b,f/b,f; fish (FW/SW) – b,f/b,f; terrestrial plants – f; terrestrial invertebrate – f; avian – f; mammals – f
Kerosene	Aquatic algae (FW/SW) – f/□; terrestrial plants – f; terrestrial invertebrate – f
Other	Aquatic algae (FW/SW) – c.1/c.1; aquatic plants (FW/SW) – c.1/c.1; aquatic invertebrate (FW/SW) – c.1/c.1; fish (FW/SW) – c.1/c.1; terrestrial plants – c.3,j; terrestrial invertebrate – c.3,j
Fractions	
C6–C10	Terrestrial plants – a; terrestrial invertebrate – a; avian – a; mammals – a
C10–C16	Terrestrial plants – a; terrestrial invertebrate – a; avian – a; mammals – a
C16–C34	□
C34–C50	□
Aliphatic/Aromatic	
C5–C6	Aquatic algae (FW/SW) – a/a; aquatic invertebrate (FW/SW) – a/a; fish (FW/SW) – a/a
C6–C8	Aquatic algae (FW/SW) – a/a; aquatic invertebrate (FW/SW) – a/a; fish (FW/SW) – a/a
C>8–C10	Aquatic algae (FW/SW) – a/a; aquatic invertebrate (FW/SW) – a/a; fish (FW/SW) – a/a
C>10–C12	Aquatic algae (FW/SW) – a/a; aquatic invertebrate (FW/SW) – a/a; fish (FW/SW) – a/a
C>12–C16	Aquatic algae (FW/SW) – a/a; aquatic invertebrate (FW/SW) – a/a; fish (FW/SW) – a/a
C>16–C21	Aquatic algae (FW/SW) – a/a; aquatic invertebrate (FW/SW) – a/a; fish (FW/SW) – a/a
C>21–C34	Aquatic algae (FW/SW) – a/a; aquatic invertebrate (FW/SW) – a/a; fish (FW/SW) – a/a

Table 7-9. Sources of available ecological toxicity values for TPH constituents

Chemical Classes	Receptor/Citation (See Table 7-10 for explanation of citations)
LMW PAHs	Terrestrial invertebrate – l; mammals – l
HMW PAHs	Terrestrial invertebrate – l; mammals – l
Individual Constituents	
Benzene	Aquatic algae (FW/SW) – a,b,c.1,d,f,g,h,i/a,b,c.1,d,g; aquatic plants (FW/SW) – c.1,d,g,h,i/c.1,d,g; aquatic invertebrate (FW/SW) – b,c.1,d,f,g,h,i/b,f,c.1,g,d; amphibian – b,f,g; Fish (FW/SW) – b,c.1,d,f,g,h,i/b,c.1,d,f,g; terrestrial plants – a,c.3,f,g,i,k.1; terrestrial invertebrate – a,c.3,f,g,i,k.1; avian – a; mammals – a,f
Ethylbenzene	Aquatic algae (FW/SW) – a,b,c.1,d,f,g,h,i/a,b,c.1,d,f,g; aquatic plants (FW/SW) – c.1,d,g,i/c.1,d,g; aquatic invertebrate (FW/SW) – b,c.1,d,f,g,h,i/b,f,c.1,g,d; fish (FW/SW) – b,c.1,d,f,g,h,i/b,c.1,d,f,g; terrestrial plants – a,c.3,f,i,k.2; terrestrial invertebrate – a,c.3,f,g,i,k.2; avian – a; mammals – a,f

Chemical Classes	Receptor/Citation (See Table 7-10 for explanation of citations)
Toluene	Aquatic algae (FW/SW) – a,c.1,d,f,g,i/a,b,c.1,d,f,g; aquatic plants (FW/SW) – c.1,d,g,i/c.1,d; aquatic invertebrate (FW/SW) – f,g,c.1,i,d/b,f,g,c.1,d; amphibian – f; fish (FW/SW) – b,c.1,d,f,g,i/b,c.1,d,f,g; terrestrial plants – a,c.3,f,i,g,k.3; terrestrial invertebrate – a,c.3,f,g,i,k.3; avian – a; mammals – a,f
Xylenes	Aquatic algae (FW/SW) – a,b,c.1,f,i/a,c.1,f; aquatic plants (FW/SW) – c.1,i/c.1; aquatic invertebrate (FW/SW) – b,c.1,f,i/b,c.1,f; amphibian – b,f; fish (FW/SW) – b,c.1,f,i/b,c.1,f; terrestrial plants – a,c.3,f,i,k.4; terrestrial invertebrate – a,c.3,f,i,k.4; avian – a,f; mammals – a,f
Individual PAHs	Aquatic algae (FW/SW) – b,c.1,d,f,g,h,i/c.1,f,g; aquatic plants (FW/SW) – c.1,d,f,g,h,i/c.1,f,g; aquatic invertebrate (FW/SW) – b,c.1,c.2,d,e,f,g,h,i/b,f,c.1,e,g,c.2,e; amphibian – g; fish (FW/SW) – b,c.1,d,e,f,g,h,i/b,c.1,e,f,g; terrestrial plants – c.3,f,i,l; terrestrial invertebrate – c.3,f,i,l; avian – f; mammals – f
Formerly Used TPH Additives	
MTBE	Aquatic algae (FW/SW) – c.1,d,f/c.1,d; aquatic plants (FW/SW) – c.1,d/c.1,d; aquatic invertebrate (FW/SW) – f,c.1,d/f,c.1,d; amphibian – f; fish (FW/SW) – c.1,d,f/c.1,d,f
TAME	Aquatic algae (FW/SW) – f/□; aquatic invertebrate (FW/SW) – f/□; fish (FW/SW) – f/□
Tetraethyl lead	Aquatic algae (FW/SW) – f/f; aquatic invertebrate (FW/SW) – f/f; fish (FW/SW) – f/ f; avian – f; mammals – f
OTHER COMPOUNDS	
Naphthenic acids	□

Notes: FW – Freshwater; SW – Saltwater

Table 7-10. Web links to sources of ecological toxicity values

Citation	Web Link
a–Atlantic PIRI 2012b. Scientific Rationale to Support the Adoption/Development of Tier 1 Ecological Screening Levels for Soil, Surface Water, Groundwater and Sediment. July.	Atlantic PIRI
b–Australian and New Zealand Environment and Conservation Council ANZECC (2000). Australian and New Zealand Guidelines for Fresh and Marine Water Quality. Revision 2018.	ANZECC 2000, rev 2018
c–British Columbia Ministry of Environment (BCMoE) 2018. Contaminated Sites Regulation. July. B.C. Reg. 375/96.	**.1** CSR Schedule 3.2 **.2** CSR Schedule 3.4 **.3** CSR Schedule 3.1
d–Canadian Council of Ministers of the Environment (CCME) 2010. Canadian Environmental Quality Guidelines.	CCME2010
e–USEPA 2003. Procedures for the Derivation of Equilibrium Partitioning Sediment Benchmarks (ESBs) for the Protection of Benthic Organisms: PAH Mixtures. EPA-600-R-02-013. Office of Research and Development. Washington, D.C. USEPA 2003	EPA 2003
f–USEPA 2018a. EcoTox Database. Last update June 7, 2018. USEPA 2018a	EcoTox
g–European Chemicals Agency (ECHA) 1994-2007. Information from Existing Substances Regulation (ESR).	ECHA-ESR
h–Official Journal of the European Union (EU) 2008. Directives: on Environmental Quality Standards in the Field of Water Policy. Directive 2008/105/EC	EU 2008
i–National Institute for Public Health and the Environment (NIPHE) 2001. Technical Evaluation of the Intervention Values for Soil/Sediment and Groundwater. RIVM report 711701 023.	Intervention Levels
j–CCME. 2008. Canada-Wide Standard for Petroleum Hydrocarbons (PHC) in Soil: Scientific Rationale Supporting Technical Document. January	PHC

Citation	Web Link
k–CCME. 2007. Canadian Soil Quality Guidelines for the Protection of Environmental and Human Health	**.1** SQGes–benzene **.2** SQGes– ethylbenzene **.3** SQGes—toluene **.4** SQGes– xylenes
l–USEPA 2007a. Ecological Soil Screening Levels for Polycyclic Aromatic Hydrocarbons (PAHs). OSWER Directive 9285.7-78. Office of Solid Waste and Emergency Response. Washington, D.C.	Eco-SSLs PAHs
□	No readily available information

Searchable databases that provide toxicity data by chemical and test species of interest are also available, including two of particular importance:

- EcoTox (ecotoxicology knowledge base maintained by USEPA)
- ECHA database, 1994–2007

Additionally, information is also available at other sources, including:

- Oak Ridge National Laboratory maintains a comprehensive database of ecological screening values
- American Petroleum Institute's High Production Volume (HPV) Challenge Program Testing
- NOAA's CAFÉ: Chemical Aquatic Fate and Effects Database

The availability of ecological toxicity data varies with the type of TPH and the ecological receptor. In general, more data are available for aquatic receptors than for terrestrial receptors. Toxicity data from laboratory-based testing are more available for individual constituents (e.g., BTEX), certain chemical groups (PAHs), and whole products than for TPH fractions. Toxicity values developed for fractions (whether or not they are separated into aliphatic and aromatic fractions) may be based on the interpreted combinations of modeling and toxicity testing results where the assumed physical and chemical properties of individual fractions are applied to whole product testing to estimate fraction-specific toxicity values. Limited toxicity data are available for amphibians, birds, and mammals.

7.5 Risk Characterization

In ecological risk assessment USEPA 1997a, the risk characterization integrates the exposure (Section 7.3) and toxicity assessments (Section 7.4). For general guidance on methods and approaches to risk characterization for ecological risk assessment, see the references noted in Section 7.1.

7.5.1 Screening-Level Risk Characterization

Screening-level ecological risk assessment relies on comparison of site data against default or site-specific screening values. At the screening level, maximum or statistical estimates of TPH concentrations in the media of interest are compared to their corresponding screening levels. The lack of exceedances or the number and magnitude of exceedances of the screening levels are used to determine whether the evaluation can terminate at the screening level (i.e., no threats to ecological receptors and no further evaluation warranted) or if a site-specific risk assessment should be undertaken (i.e., there may be potential for adverse effects and additional evaluation is warranted).

7.5.2 Site-specific Level Risk Characterization

Site-specific ERA identifies and develops toxicity data and exposure parameters for identified receptors and assessment end points at the site, and estimates risk based on hazard quotients by comparing media concentrations to measured or estimated media toxicity values and/or ingested doses compared to toxicity reference values. Section 7.4 provides detailed information on toxicity criteria, methods, and approaches.

7.5.3 Multiple Lines of Evidence Approach in Site-Specific TPH ERA

Multiple Lines of Evidence (MLOE) Approach

The MLOE approach serves to increase the robustness of and level of confidence in a risk assessment. When all lines

of evidence point to similar trends and findings, there is greater confidence in the overall conclusions of the ERA. In TPH ERA, the MLOE approach may include using several types of appropriate TPH data (e.g., product-level, fraction-level, and indicator compounds data) and several risk characterization methods (e.g., predicted hazard quotients, toxicity tests, and population and community-level impact metrics). When assuming that the toxicity of TPH compounds or fractions is additive, it is important to avoid "double-counting" of hazards in the assessments (see Sections 7.5 and 7.6).

Risk characterization for site-specific risk assessment introduces **site-specific lines of evidence and measurement end points** to address the specific nature of the TPH issues at the site. Table 7-11 presents general options and recommendations. Applying more than one line of evidence to a complex site can be helpful in understanding the potential impacts of TPH mixtures.

Table 7-11. Key lines of evidence for site-specific TPH ecological risk characterization

Media and Receptors	TPH Applicability	Best Practices
Line of Evidence: Calculation of TPH hazard quotients		
Ingested dose to **higher trophic level receptors** in **aquatic** and **upland** environments	Relies on defining appropriate **toxicity reference values** and **exposure parameters** for the TPH exposure	Generally recognized and straightforward method. Easy to interpret. Multiple toxicity criteria from diverse sources and absence of generally applied criteria require careful identification and justification of selected values.
Line of Evidence: Food web uptake and biotransfer		
Higher trophic level receptors (predators) indirectly exposed via prey	Limited applicability. Biotransfer of TPH difficult to evaluate. Evaluation often based on known bioaccumulative components, such as PAHs	Food web uptake is usually part of site-specific risk assessment for individual chemicals. However, not easily measured or defined for TPH and therefore not recommended except for PAHs in aquatic environments and in special circumstances.
Line of Evidence: Toxicity testing and bioaccumulation studies		
Water and sediment: applicable and generally accepted for **media-specific and receptor-specific applications** Soil: less common, but frequently applied to specific receptor classes such as rooting plants and soil invertebrates	In surface: **water toxicity testing**, **WAF toxicity tests**. In sediment: **sediment and pore water bioassays.** In soil: **plant and invertebrate toxicity testing** All toxicity tests subject to confounding factors and lack of specificity.	Generally recognized and applicable approach. Toxicity testing is effective in identifying adverse effects from TPH mixtures. The selection of the appropriate toxicity test is site- and situation-specific, depending on the site of impact of TPH (sediment, surface water, soil). Interpretation of toxicity tests complicated by confounding factors and may not be highly specific to TPH, therefore well-designed test objectives and protocols are critical.
Line of Evidence: Population and/or community metrics		
Aquatic (benthic and fish) community metrics accepted and in widespread use	Susceptible to confounding factors and lack of specificity when multiple stressors are present.	Community metrics of effect are primarily used in well-defined aquatic settings. Limited applicability to TPH risk assessment due to the lack of well-defined measures differentiating TPH-related effects from other stressors. Generally not recommended for terrestrial environments at this time.

7.5.4 Ecological Risk Characterization Considerations for TPH

This section outlines key concepts for considering TPH in the risk characterization and summarizes them in Table 7-12 for screening-level assessments and Tables 7-13 and 7-14 for site-specific risk assessments.

Table 7-12. Screening-level risk characterization—issues and best practices

Screening-Level Risk Assessment
Issue: Exclusion criteria and need for eco risk assessment **Summary:** Not all petroleum release sites require ecological evaluation. Exclusion criteria can be used in many jurisdictions to exclude evaluation of TPH impacts if certain conditions are met. (Table 5-3) **Best Practices:** Follow local jurisdiction regulations on ecological exclusion criteria. Consider if evaluation of indicators of TPH, such as BTEX and PAHs, may adequately address the ecological concerns.
Issue: Multiple modes of action **Summary:** Where free product is present, the concern of non-chemical-specific adverse effects from oiling, physical contact, and indirect effects from ambient oxygen deprivation and suffocation from root blockage or deoxygenation of waters, or ingestion of bulk (acutely toxic) quantities from preening may be significant. **Best Practices:** Consider if direct contact with TPH free product is relevant to the site. Adverse physical effects from direct contact are generally not included in TPH screening or toxicity criteria. Site-specific evaluation may be more useful for physical effects. In the absence of free product, the use of standard or modeled screening levels (for screening level) and toxicity reference values (for site-specific risk) is likely to be adequate to address the ecological risk of TPH. If free product is present, site-specific evaluation will likely be more appropriate in addition to standard dose-based evaluation, especially in aquatic risk assessment where floating, emulsified, entrained, or undissolved TPH may be present and resulting in direct toxicity effects.
Issue: Defining ecological screening-level criteria **Summary:** There are relatively few ecological screening levels for TPH, as discussed in Section 7.2. These screening levels are frequently nonspecific as to the specific mixture or product under evaluation or are based on non-risk-based criteria. **Best Practices:** The options for initial screening include screening against generic TPH criteria, recognizing that the screening may say little about ecological risk, and proceed to additional site-specific tiers to further evaluate the issue if found to be exceeded. Screening analytical data against individual components of the TPH mixture, such as PAHs for heavier TPH and BTEX for lighter TPH, may be an option or requirement in certain jurisdictions. This practice does not represent best practice, as it underestimates risk from other components of the mixture but may be appropriate in limited circumstances.
Issue: Non-risk-based criteria **Summary:** Screening levels for TPH may not be based on risk-based considerations. Such screening levels do not provide any information on whether there is an ecological risk, and it should not be assumed that there is ecological risk solely on such considerations. **Best Practices:** The source and relevance of screening levels should be reviewed. The use of TPH screening levels based on regulatory cleanup limits or on ad hoc values based on human health or analytical method and narrative criteria, such as the absence of visible sheens, may be appropriate depending on jurisdiction, but risk characterization should consider that it could under- or overestimate ecological risk.

Table 7-13. Site-specific risk characterization—issues and best practices for toxicity criteria

Site-Specific Risk Assessment
Issue: Aquatic toxicity criteria and approaches **Summary:** There is limited toxicity data for complete hydrocarbon mixtures, and these are focused on site-specific evaluation of marine exposures to WAFs. Models such as PETROTOX are limited to data for individual chemicals and fine-grained hydrocarbon “blocks” (see Overview of Toxicity Prediction by Mechanistic Models). The use of exposure models in aquatic systems can be a powerful tool. **Best Practices:** Use of toxicity screening levels based on site-specific TPH conditions is growing in acceptability, especially at petroleum release sites, and can be a powerful tool where jurisdictions accept the approach. Specialized analytical data may be required for these approaches. See Section 7.4.3 for more information on the use of such models.

Site-Specific Risk Assessment
Issue: Use of tissue criteria **Summary:** The evaluation of biological tissue concentrations (e.g., bird eggs, muscle tissue, or liver concentrations) is sometimes used in site-specific ecological risk assessment. Although this has wide applicability for many chemicals, its applicability to TPH is questionable. Bioavailability and biochemistry of the components of a TPH mixture vary widely, and no single “TPH” measure can be defined or considered to be applicable across the food chain. It is conceptually difficult to posit a biochemical mode of action for TPH. TPH as an analytical end point is difficult to analyze and interpret in biological tissue due to interference from the organism’s lipids and biochemical pathways and large variations in metabolism of TPH components in biological tissue. **Best Practices:** It is recommended that TPH measurements in tissue not be considered in ecological risk assessment. Evaluation of specific components, such as PAHs, may be appropriate, especially where the concern is bioaccumulation and impacts to higher trophic-level organisms, but does not necessarily represent TPH as a whole.

Table 7-14. Site-specific risk characterization—issues and best practices for TPH TRVs

Issue: Defining Appropriate TRVs
Summary: The partitioning models for TPH in aquatic systems are inapplicable or insufficient in ecological risk assessment for terrestrial receptors (exposures to soil and the terrestrial food chain) and aquatic food webs (e.g., aquatic mammals exposed to the aquatic food chain). Evaluation of ecological risk requires the definition of applicable TRVs to allow the ingested dose line of evidence to be developed. Relatively few toxicity studies, based on a low number of individual chemicals and tested on a few standard test organisms (predominantly rats and mice) under laboratory conditions, serve as the base for TPH. Therefore, TPH TRVs for the entire mixture may rely on just a few toxicity study results, implying that the entire mixture or fraction has equivalent and additive toxicity as the test compound used as surrogate. **Best Practices:** Best practice for using TPH TRVs in site-specific risk assessment may be ranked as follows: 1. Apply literature or regulator-recommended TRV(s) and apply as part of a standard ingested dose risk calculation. When determining the TRV: 1. Avoid “double counting” by subtracting any contribution from individually evaluated components of the TPH (must have been analyzed by the same method) 2. Consider how the TRV would apply to the site TPH mixture in terms of toxicity, bioavailability, and bioaccumulation and discuss as part of uncertainty 3. Report TPH-specific risk separately and not additively to individual chemicals 2. In the absence of applicable literature or regulator-recommended TRVs, use representative TRVs based on surrogate chemicals. Conduct an ingested dose risk-based calculation for each identified component of the TPH mixture, especially the more toxic components. The risk, when added, might be considered representative of the total risk from ingesting the TPH. If applying this approach, consider the following in uncertainty discussion: 1. Underestimating risk from unevaluated components of the mixture that may possess higher toxicity than expected. Although it is generally accepted that the aromatics, especially the common PAHs, supply a major portion of the toxicity compared to aliphatics, there may be other chemicals of concern present. 2. For food web uptake calculations, this approach may be more appropriate because there are generally no standard TPH bioaccumulation and biotransfer factors. 3. In site-specific risk assessment, the shortcomings of using default or surrogate toxicity data may be overcome by focusing on toxicity tests and community studies. 1. Toxicity testing can be a productive approach but also relies on test organisms as surrogates for the organisms of concern, and becomes more challenging when dealing with larger wildlife. Although toxicity testing may provide a good snapshot, the effect of spatial and temporal heterogeneity in the field, and differences in bioavailability between field and toxicity testing conditions need to be considered. 2. Community studies, comparing the on-site community or population to an expected or reference community, can be a useful line of evidence in aquatic environments. Such studies can be time-consuming but can help demonstrate that the observed situation is no different from a similar situation without the TPH impact.

7.5.5 Uncertainty Analysis

Uncertainties are integral to all risk assessment and particularly so for ecological risk assessment, considering the variability of real-world ecological systems and the unknowns inherent in evaluating a large class of chemicals as is the case with TPH. In the uncertainty section, the risk characterization results are evaluated in terms of confidence in the outcome based on the uncertainties, confidence levels, and applicability of the conceptual site model, exposure assumptions, toxicity assumptions, and underlying data. The evaluation of uncertaintiesan be narrative and may include qualitative or quantitative sensitivity analysis based on alternate assumptions to bound the estimates.

▼*Read more*

Table 7-15 summarizes some key uncertainties inherent to TPH ERA to consider when evaluating or planning risk assessments.

Table 7-15. Effect of uncertainties on TPH ERA

Key Uncertainty Issue	**Probable Impact on Risk Estimates (Over/Under)**	**Typical Magnitude**
Representativeness of fractions, components and/or surrogates of TPH	✓/✓	Unknown, may be large for weathered or degraded TPH
Completeness of CSM (chronic vs. acute, indirect exposure pathways, relevant biological scale of exposure to receptors)	/✓	Moderate
Sample and site spatial and temporal heterogeneity, effect of weathering	/✓	Small to large
Screening levels (representative of TPH mix, risk-based source, applicable end points)	✓/✓	Small to large
Nonadditivity of TPH risk and TPH component double counting	✓/	Small to moderate
Bioavailability (TPH weathering, mobility, solubility, presence of free product)	✓/✓	Small to large
Toxicity reference value applicability and relevance of selected TPH data types	✓/✓	Small to large
Toxicity test representativeness to underlying exposure mechanisms, especially when TPH + non-TPH mixtures	✓/✓	Small to moderate
Additional direct or indirect impacts from TPH (oiling, direct contact, indirect changes to habitat)	/✓	Small (but may be large in oil spill situations)