

EXPERT REPORT OF
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*In the Matter of Hero Lands Company,
LLC v. Chevron USA, Inc., et al.*

September 4, 2020

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1.0 Statement of qualifications

I am a Principal Toxicologist and Senior Vice President of the Health Sciences Division at CTEH® specializing in risk assessment, exposure assessment, toxicity evaluations, the evaluation of experimental design and methodologies, and assessing causal relationships between chemical exposure and disease. My educational background includes a B.S. in biochemistry with an emphasis in toxicology from Murray State University in 1993 and a Ph.D. in toxicology from The University of Georgia in 2000. I am also a Certified Industrial Hygienist (CIH) and Certified Safety Professional (CSP). I am a member of the Oil and Gas Working Group of the American Industrial Hygiene Association (AIHA), American Conference of Industrial Hygienists (ACGIH®), the American Society for Testing and Materials (ASTM) International – Subcommittee D18.26 on Hydraulic Fracturing, the Society of Toxicology (SOT), and the American College of Occupational and Environmental Medicine (ACOE). I am a member of the AIHA's Emergency Response Planning Guideline (ERPG) committee, which establishes exposure guidelines for communities after emergency chemical releases. My current duties at CTEH® include serving as a consulting toxicologist and industrial hygienist, providing guidance for risk assessment and remediation plans, leading responses to and providing toxicological support for hazardous materials incidents, and providing toxicological support to care providers and workers with potential chemical exposures. I have gained extensive knowledge and experience in the mechanisms and toxicities of a wide range of compounds including chlorinated hydrocarbons, volatile organic hydrocarbon solvents (VOCs), constituents associated with oilfield exploration and production, polycyclic aromatic hydrocarbons (PAHs), pesticides/herbicides, irritant gases, dioxins, and heavy metals. I have personally lead air monitoring and environmental sampling teams on over 50 hazardous materials incidents in the last 13 years, and I have remotely supported dozens more.

Toxicology, a blend of biology, chemistry, and medicine, is the science of the adverse effects of substances (e.g., chemicals, physical agents, and drugs) on biological systems including the effects, the recognition, and the mechanisms of a chemical-related disease. Whether a substance is a toxicant depends upon two inseparable criteria: 1) the intrinsic nature of the substance, and 2) the dose, or how much a substance the individual actually takes into their body. In toxicology, we study the dose-response of chemicals on biological systems, with emphasis on understanding the mechanisms of harmful effects.

I have attached a copy of my curriculum vitae and Rule 26 disclosure in **Appendix A**.

2.0 Basis of suit and understanding of allegations

The plaintiff, Hero Lands Company, LLC (Hero Lands), has filed suit based on allegations of contamination from historical oilfield activities within portions of the plaintiff's property located in Plaquemines Parish, Louisiana. Based on my review of the pleadings and plaintiff's experts' reports, it is my understanding that the plaintiffs are alleging that activities associated with oil and gas exploration and production (E&P) have resulted in contamination of portions of the soil and groundwater present within the Hero Lands property. As stated in the initial pleadings, these E&P operations "*damaged Plaintiff's Property by spilling and*

disposing of toxic and hazardous oilfield wastes on, in and adjacent to Plaintiff's Property" (Petition for Damages). Furthermore, plaintiff's experts opine, "*Contamination of site media, particularly surface soil and shallow groundwater has resulted from oil and salt water handling operations. Residual contaminants from those operations, including metals, salts, hydrocarbons, dioxin/furans equivalents and radionuclides pose an unacceptable health risk to human and ecological populations"* (Expert Report of William Rogers).

I have been asked as a toxicologist to evaluate the environmental sampling data collected by the plaintiff's and defendants' consultants relative to the property and determine if constituents at this site are present at levels which may pose a risk to human health. I have conducted this human health risk evaluation based on my education and training in toxicology and risk assessment and by utilizing a human health risk assessment framework consistent with the state-specific Louisiana Department of Environmental Quality (LDEQ) Risk Evaluation/Corrective Action Program (RECAP) and United States Environmental Protection Agency (USEPA) risk assessment methodologies and guidance.

3.0 Information reviewed and work conducted

In relation to this litigation, I have reviewed a substantial amount of material including, but not limited to, pleadings and environmental data including soil and groundwater samples collected by ICON Environmental Services, Inc. (ICON) working on behalf of the plaintiffs, and Environmental Resources Management (ERM) working on behalf of the defendants. In addition, I have reviewed reports from the plaintiff's and defendants' experts provided, scientific literature concerning the toxicology of the substances at issue (including but not limited to inorganics, metals, and petroleum hydrocarbons) and have reviewed appropriate risk assessment guidance (e.g., LDEQ RECAP and the USEPA). I conducted a site visit on the property on January 21, 2020. A list of the documents I have reviewed in this case is presented in **Appendix B**.

4.0 Location, description, and use of the Hero Lands property

The Hero Lands property is comprised of four tracts of land (designated as the NW tract, the SW tract, the NE tract, and the SE tract) encompassing approximately 155 acres within the town of Belle Chasse in Plaquemines Parish, Louisiana. The tracts are located in the Stella Oil and Gas Field along the western bank of the Mississippi River; oil production in the Stella Oil Field and within the Hero Lands property was developed in the 1940s. The Hero Lands property is located in Sections 16, 17, and 18 of Township 14 South, Range 24 East and Sections 2 and 3 of Township 15 South, Range 24 East. **Appendix C Figure C-1** presents the location of the Hero Lands property. The tracts are characterized by a combination of undeveloped land, levee and batture areas along the Mississippi River, oil and gas E&P activities, and industry associated with the Chevron Oronite Facility. The tracts of the Hero Lands property are bisected by the Louisiana (LA) Hwy 23. The NE and SE property tracts are bounded to the west by LA Hwy 23, the Mississippi River to the east and north (i.e., NE tract) and the Chevron Oronite Facility to the south (i.e., SE tract). The NW property tract is bounded to the east by LA Hwy 23 and residential areas to the west

and south. The SW property tract is bounded by LA Hwy 23 to the east, residential areas to the north, and undeveloped woodlands to the west and south. According to ordinance zoning details provided in the report of Keith Core, the zoning categories for the four property tracts include the following: SW tract as I-3: Heavy Industrial; SE tract as I-3: Heavy Industrial and FP: Flood Plain (Riverward); NW tract as C-3: General Commercial; and NE tract as I-3: Heavy Industrial and FP: Flood Plain.

Land use of the surrounding properties includes a mix of commercial, industrial, and residential properties. Historic and current land use of the Hero Lands property includes oil and gas E&P activities and possible recreational hunting. Whereas the property tracts are primarily used for oil and gas E&P activities, a single residential dwelling was reported along the highway on the NW property tract, and a trailer was observed near operation areas present on the SW property tract. As presented in **Appendix C Figure C-2**, the Hero Lands property is located within the DEQ subsegment basin 020601 (Intracoastal Waterway, Barataria, From Bayou Villars to Mississippi River) to the west and subsegment basis 070301 (Mississippi River, Mississippi, Monte Sano Bayou to Head of Passes). I have been asked to evaluate environmental sampling data relative to the tracts comprising the Hero Lands property.

Evaluating the current and future use of the property is an important component of the exposure pathway analysis to determine the potential risk to receptors (i.e., potentially exposed individuals) using the Hero Lands property. Use patterns for the Hero Lands property were abstracted from the plaintiff's deposition testimony, plaintiff's experts' reports and the defendants' experts' reports. Oil and gas E&P operations represent the historic, current, and likely future use of the property. Plaintiff experts indicate future land use may include residential development despite plaintiff deposition testimony indicating continued industrial activities.

5.0 Intended usage of the human health risk assessment process

Human health risk assessment was developed during the 1980's and was first codified by the National Research Council (NRC) in a publication referred to as the "Red Book," which forms the foundation of the risk assessment methodology adopted by the USEPA and states such as Louisiana. Risk assessment can be conducted in two ways, often referred to a "forward" or "reverse" (i.e., backward) risk assessment. Forward risk assessment, which is the methodology originally described in the Red Book, involves beginning with an exposure point concentration (i.e., some measure of the concentration of a constituent in an environmental medium) of a constituent of potential concern (COPC) combined with a series of exposure assumptions (i.e., exposure routes, frequencies and durations) to determine an individual's dose of that COPC for a given exposure scenario. This dose is then compared to health-based benchmarks to provide an estimate of the risk that may be associated with that specific exposure scenario. This methodology is advantageous, as it provides a theoretical risk value related to the given exposure scenario, and it can be tailored to a site-specific exposure scenario. However, this methodology is more labor intensive than reverse or backward risk assessment and involves a greater level of education, training, and professional judgement. This leads to the development of "reverse" risk assessment, which

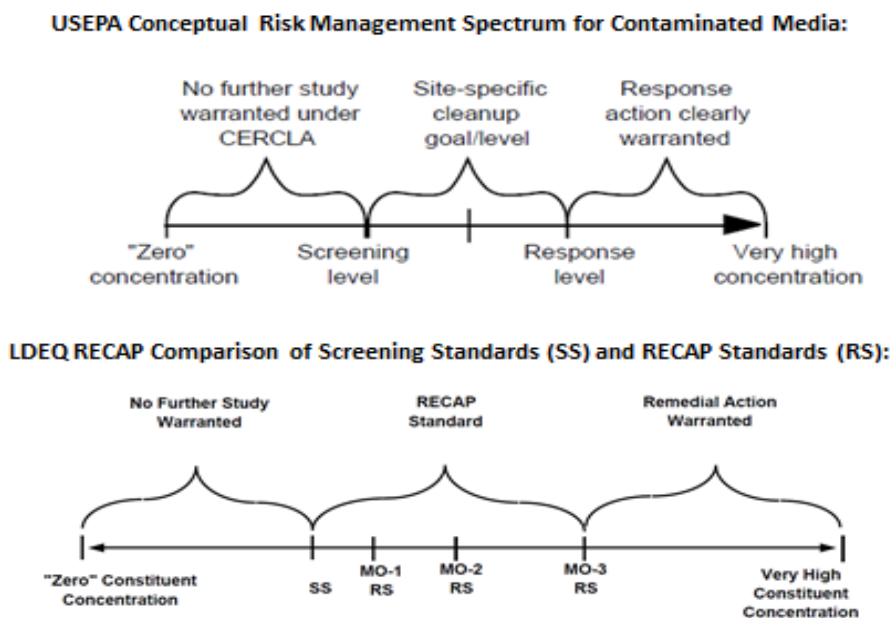
starts with first establishing the level of acceptable health risk and then working backward to determine the environmental concentration of the COPC that yields the acceptable health risk. Using this methodology, regulators and scientific bodies developed screening values that are published in generic “lookup” tables (e.g. LDEQ RECAP Table 1 and Table 2), allowing for the rapid screening of environmental sites by comparison of environmental COPC concentrations to published default values. This methodology forms the basis for risk-based cleanup programs such as the LDEQ RECAP. The advantages of this methodology include ease of use and less of a need for training and education in health sciences and the fate and transport of COPCs in the environment. The primary disadvantages of this methodology are that it is applicable only for the underlying exposure scenario based on default exposure assumptions (typically a residential or non-residential/industrial exposure scenario) and that it does not yield an estimated dose with which to calculate a site-specific health risk. To address the scenario-specific nature of the risk assessment approach, agencies such as the LDEQ have developed a tiered approach which allows for the incorporation of more site-specific information as one proceeds through higher tiers of the risk assessment. It should be noted, an extensive review of the toxicological basis and foundations of human health risk assessment can be found in **Appendix D**.

As with other states, the state of Louisiana uses the “reverse” or “backward” risk assessment approach to develop generic screening tables using a non-industrial (i.e., residential) or industrial exposure scenario to evaluate risks to human health and the environment posed by the presence of chemical constituents in the environment. The LDEQ’s RECAP is consistent with the USEPA risk assessment guidance; as noted by the LDEQ: “*RECAP is consistent with the Environmental Protection Agency’s (EPA) guidance on risk assessment*” (LDEQ, 2003b). As a general approach, the LDEQ uses a tiered or step-wise method for site evaluation to: “(1) *determine if corrective action is necessary for the protection of human health and the environment, and (2) identify constituent levels in impacted media that do not pose unacceptable risks to human health or the environment, i.e., RECAP Standards*” (LDEQ, 2003b).

The LDEQ’s RECAP is the state of Louisiana’s primary regulation governing remediation activities (LDEQ, 2003b). The LDEQ RECAP consists of a tiered framework composed of a Screening Option (SO) and three Management Options (e.g., the MO-1, MO-2, and MO-3). The Screening Options are comprised of Screening Standards (SS) applied to soil [e.g., Soil Screening Standard Non-industrial (residential), Soil_{SSNI}; Soil Screening Standard Industrial, Soil_{SSI}; the Soil Screening Standard protective of Groundwater, Soil_{SSGW}] and the Screening Standard for groundwater (e.g., Groundwater Screening Standard, GW_{SS}). For soil and groundwater the Screening Options are used to: “(1) *demonstrate that the COC concentration present in soil and/or groundwater does not pose a threat to human health or the environment and hence does not require further action at this time; (2) identify the [area of investigation] AOI and the COC for corrective action of soil and/or groundwater under the SO [Screening Option]; or (3) identify the AOI and the COC ... for soil and groundwater for further evaluation under a MO*” (LDEQ, 2003b). The tiered approach allows site evaluation and corrective action efforts to be tailored to site-specific conditions (LDEQ, 2003b). As stated in the RECAP: “*As the Management Option level increases, the approach becomes more site-specific*

[i.e., requires additional site specific data to evaluate constituent fate and transport] and, hence, the level of effort [and information] required to meet the objectives of the [Management] Option increases” (LDEQ, 2003b). The additional information may include further site evaluation, a more extensive exposure assessment, and use of sophisticated fate and transport models. Although the level of effort required for each Option varies, each Option achieves a common goal, which is “protection of human health and the environment” (LDEQ, 2003b). Stated another way, the Screening Options and all Management Options are designed to achieve the same goal, and no option is “safer” than the other. This concept is illustrated in Figure 5.1 demonstrating the USEPA’s spectrum of contamination which can be encountered at a site of interest and the conceptual range of risk management; the same diagram is adapted by the LDEQ regarding the comparison of Screening Standards to RECAP Standards. As evident by Figure 5.1, it is not until a MO-3 RECAP Standard is “exceeded” that remedial action is warranted, confirmatory sampling shall be conducted and closure and/or post-closure requirements shall be met (LDEQ, 2003b).

Figure 5.1: Comparison of USEPA and LDEQ RECAP conceptual risk management spectrum



Adapted from LDEQ (2003b) and USEPA (1996a)

To further expand on this concept, the Screening Options may be used to screen out areas of a property, media, or COCs¹ that do not warrant further evaluation as to limit the scope of the RECAP evaluation to

¹ As defined by LDEQ, 2003: Constituents of concern (COC) - solid waste and hazardous waste, as defined in LAC 33:V.109; industrial solid waste as defined in LAC 33:VII.115; hazardous substance, as defined in La. R.S. 30:2272; regulated substance, as defined in LAC 33:XI.103; pollutant as defined in La. R.S. 30:2004; wastes as defined in La. R.S. 30:2073; and pollutant, priority pollutant, and toxic substances, as defined in LAC 33:IX.107.

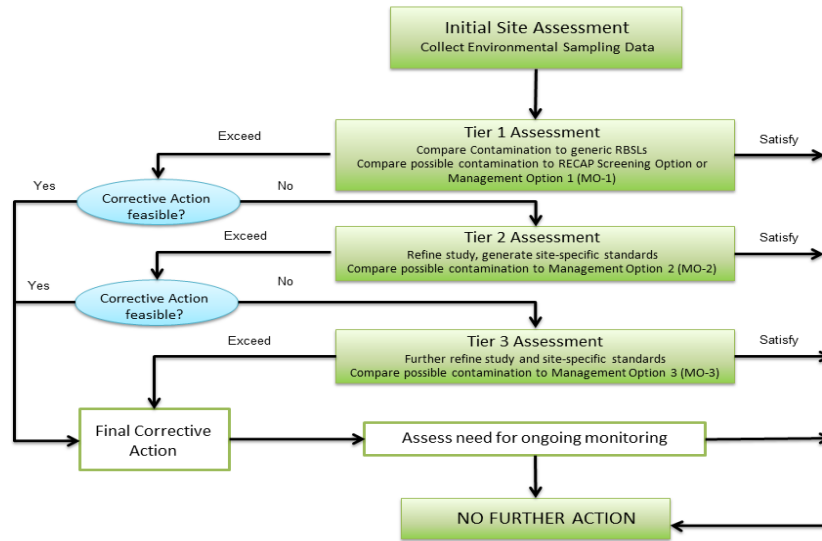
those areas/media/constituents of most concern. If the maximum constituent concentration(s) detected in soil and/or groundwater exceed the SS, then: (1) the area shall be managed under the SO; or (2) the area shall be evaluated under MO-1, MO-2, or MO-3. Under the MO-1, the LDEQ provides Department-derived RECAP Standards for soil and groundwater that are protective of human health and the environment (LDEQ, 2003b).

Similar to the SS, the *“Management Option 1 may be used to: (1) document that an AOI does not pose a threat to human health or the environment and hence, does not warrant further action at this time; (2) expeditiously manage an AOI defined by the presence of low constituent concentrations and standard exposure conditions; and/or (3) identify areas of a facility, media, or COC that warrant further evaluation so that the scope of the Management Option 2 (MO-2) or Management Option 3 (MO-3) evaluation can be limited to those areas/media/constituents most likely to pose risk. If a constituent-specific soil [area of interest concentration] AOIC or groundwater [compliance concentration] CC exceeds the MO-1 limiting RS [RECAP Standard], then the Submitter may: (1) remediate to the MO-1 limiting RS and comply with closure and/or post-closure requirements for MO-1; or (2) proceed with a MO-2 or MO-3 evaluation. The Submitter may elect to skip the MO-1 and proceed directly to MO-2 or MO-3”* if site specific information is available (LDEQ, 2003b).

The MO-2 allows for the development of soil and groundwater RECAP Standards (protective of human health and the environment) based on the use of site-specific data with analytical models to evaluate constituent fate and transport at the AOI. Under a MO-2 the site-specific evaluation is used in conjunction with *“default exposure assumptions and toxicity criteria”* to identify a site-specific MO-2 RECAP Standard. *“If the soil AOIC and groundwater CC for all COC are less than or equal to the site-specific MO-2 limiting RS, then typically, [no further action at this time] NFA-ATT is required for soil or groundwater.”* (LDEQ, 2003b). Furthermore, *“if a constituent-specific soil AOIC or groundwater CC exceeds a MO-2 limiting RS, the Submitter may: (1) remediate to the MO-2 limiting RS and comply with closure requirements for MO-2 (and post-closure requirements if warranted); or (2) proceed with a MO-3 evaluation”* (LDEQ, 2003b).

The MO-3 requires a more extensive exposure assessment and usage of sophisticated fate and transport models. The MO-3 allows for the development of site-specific RECAP Standards *“protective of human health and the environment under site specific conditions”* for all impacted media using site-specific exposure and environmental fate and transport data (LDEQ, 2003b). Guidance under MO-3 states: *“If the AOIC and groundwater CC detected at the AOI are less than or equal to the MO-3 limiting RS, then typically, NFA-ATT is required. If a constituent-specific AOIC or groundwater CC for a COC exceeds a MO-3 limiting RS, then: (1) the AOI shall be remediated to the MO-3 RS; (2) confirmatory sampling shall be conducted; and (3) closure and/or post-closure requirements shall be met”* (LDEQ, 2003b). Figure 5.2 corresponds to the tiered framework of the risk-based decision-making process adapted to include the associated tiers used under RECAP.

Figure 5.2: Risk-based corrective action flowchart incorporating LDEQ RECAP tiered assessment options



Adapted from Magaw and Nakles (2001)

The Screening Options presented under LDEQ RECAP provides a rapid screening tool during the early stages of a site investigation through the use of generic, default lookup tables with screening standards available in the LDEQ RECAP (Table 1 in LDEQ, 2003); furthermore, the Screening Option allows submitters to focus efforts for further assessment. By screening out areas of a site, the COCs of interest, and the exposure pathways for further evaluation, site managers can limit the necessary scope of the remedial investigation or risk assessment. However, there are several limitations presented by usage of the SO, including the inability to tailor the assessment to site-specific conditions (i.e., site usage or exposure parameters, groundwater classification, dilution factors, etc.), and the area of interest concentration (AOIC) is based on the maximum detection constituent concentration, not a measure of the likely upper-bound exposure [i.e., the 95 percent upper confidence limit on the arithmetic mean (95%UCL-AM²) concentration]. Thus, the conservative nature of the Screening Options often leads to the use of a higher tier of assessment (i.e., MO) if the remediating party so chooses. In addition, using this guidance for sites where residential land use assumptions do not apply, results in an over estimation of exposure and overly conservative screening levels (USEPA, 1996b).

In conclusion, “RECAP uses risk evaluation to: (1) determine if corrective action is necessary for the protection of human health and the environment, and (2) identify constituent levels in impacted media that do not pose unacceptable risks to human health or the environment, i.e., RECAP Standards [RS]” (LDEQ, 2003b). Thus, the Screening Options and Management Options are established for the protection of human health and are set at levels well below concentrations at which adverse health effects would be

² The 95%UCL-AM is the concentration most representative of the concentration that would occur over time as it would not be expected for an individual to spend their entire time at a single sampling location.

expected to occur. Thus, such risk assessment methodology can only be used to rule out the potential for adverse health effects and cannot be used to establish that a health risk exists.

The assessment of human health risks from exposure to environmental constituents cannot be exclusively based on the comparison of environmental sampling results to a screening level standard; site-specific information and site use patterns need to be considered when characterizing the risk to users of a property and the environment. As such, the exceedance of a screening level cannot serve as the sole foundation on which one can opine that site conditions represent a hazard. Nor can the mere presence of a chemical in environmental media imply that the level present is toxic. Whether a potential exposure represents a risk or is toxic is a function of the dose of the compound that would potentially be received (a function of the exposure concentration, exposure frequency, and exposure duration) and the compound's intrinsic toxicity.

The plaintiffs allege that substances from historic oil and gas E&P activities are present in the soil and groundwater which have been “contaminated” and/or “damaged” site media, and the defendants “knowingly disposing of toxic and hazardous substances onto Plaintiff’s Property, in failing to clean up said substances and stop its further migration, in allowing the migration of these substances to offsite properties, and in failing to properly maintain its facilities where these toxic and hazardous substances were transported, handled, stored and disposed of, each constitute “wanton or reckless disregard for public safety in the storage, handling, or transportation of hazardous or toxic substances” on the Hero Lands property based upon an incomplete assessment (Petition for Damages). Furthermore, plaintiff’s expert Dr. William J. Roger’s opines that “contamination of site media, particularly surface soil and shallow groundwater has resulted from oil and salt water handling operations. Residual contaminants from those operations, including metals, salts, hydrocarbons, dioxin/furan equivalents and radionuclides pose an unacceptable health risk to human and ecological populations.” It is important to note that the mere exceedance of a regulatory standard does not necessarily indicate that an actual risk exists or that corrective action is warranted to protect human health and the environment (LDEQ, 2003b; Risher and DeRosa, 1997; USEPA, 1996b). It should be noted that the plaintiff’s experts did not perform a proper risk evaluation and did not consider the current or likely future usage of the property and potential exposure scenarios likely to occur on the property. Additionally, plaintiff’s experts have not calculated a dose for a human receptor of any constituent; therefore, they cannot opine that constituents on site are present at levels that “can cause serious health related problems,” represent a “reckless disregard for public safety,” or “pose an unacceptable health risk” for any current or future user of the property.

The toxicological evaluation conducted in this report provides the appropriate assessment with regard to human health by following RECAP guidelines and utilizing site-specific information and data obtained from the site investigation. Furthermore, the toxicology of substances at issue has been well studied and evaluated in the scientific literature. Therefore, it is appropriate, scientifically and by regulation, to evaluate the potential human health risks based on the measured concentrations reported from environmental sampling data collected on the property and information regarding the actual land-use

scenario of the property, which dictates the potential exposure scenario. The available environmental sampling data collected were used to calculate doses for potential receptors present on the property consistent with the LDEQ RECAP, USEPA, and other scientifically accepted guidance. It is notable that the goal of regulatory risk assessment is to develop guidelines that are protective of human health without underestimating the actual risk. As uncertainties are inherent in the risk assessment process, conservative (i.e., health protective) assumptions are made that likely overestimate the true or actual risk to users or hypothetical users of the property. As a result, risk assessment cannot be used to establish whether toxicity will occur, but it can only be used to effectively rule out the possibility of adverse health effects associated with a given exposure.

Plaintiff's experts contend that the site exhibits "contamination" of inorganics, metals, petroleum hydrocarbons, dioxin/furan equivalents and naturally occurring radioactive material (NORM) caused by historical oil and gas E&P activities. The evaluation of NORM will be discussed by other experts. Regarding petroleum hydrocarbons, the plaintiff's experts reach these opinions by comparing total petroleum hydrocarbon (TPH) mixture data for gasoline range organics (TPH-GRO), diesel range organics (TPH-DRO), and oil range organics (TPH-ORO) to LDEQ RECAP Standards. The comparison of TPH-GRO, TPH-DRO, TPH-ORO to LDEQ RECAP Screening Option (SO) Screening Standards or Management Option-1 (MO-1) RECAP Standards is not a reliable method for evaluating the toxicological risk of petroleum hydrocarbon exposure to humans. To evaluate the toxicity of petroleum hydrocarbons and potential human health risks associated with petroleum hydrocarbon contamination, evaluation of petroleum hydrocarbons as aliphatic and aromatic hydrocarbon fractions is the appropriate methodology and is preferred by the LDEQ.

I have been asked as a toxicologist to evaluate environmental sampling data collected by the plaintiff's and defendants' consultants on the property and determine whether the levels of constituents present on-site are harmful to human health and represent a risk to public safety. I have also been asked to opine on the proper methodology to evaluate whether the levels of petroleum hydrocarbons present on site represent a harm to human health. This evaluation is based in part on calculating potential receptor doses to determine if an actual human health risk exists. My evaluation of site petroleum hydrocarbons is based on the aliphatic and aromatic hydrocarbon fractionation methodology (i.e., the "fractionation method"), which is the most defensible and scientifically reliable method to evaluate the health risk of petroleum hydrocarbons.

6.0 The "fractionation method" (aliphatic and aromatic hydrocarbon fractionation analysis) is the proper methodology for evaluation of human health risks from environmental exposure to petroleum hydrocarbons

I have been asked to assess the potential for adverse health effects from petroleum hydrocarbon exposure on the Hero Lands property and to opine on the various methods for assessing health risks from

environmental exposure to petroleum hydrocarbons. Historically, hydrocarbon-impacted soils and groundwater at oil and gas E&P sites were managed based on a total petroleum hydrocarbon (i.e., TPH-mixtures) approach. However, TPH-clean-up concentrations at E&P sites were not based upon risk to human health, but rather the protection of plants and water resources (API, 2001). More recently, health-based methods for determining risks from petroleum hydrocarbon exposure have evolved over time to be more precise and provide more accurate assessment of potential health risks. As it was clear that TPH-mixtures did not provide an accurate basis for the evaluation of potential human health risks at an E&P site, advances in research and risk assessment initiatives focused on developing and using a petroleum hydrocarbon fractionation-based method (e.g., aliphatic and aromatic hydrocarbon fractions) that allowed for grouping of hydrocarbon fractions based on physiochemical and toxicological properties. The hydrocarbon fractionation approach is the methodology preferred by LDEQ's RECAP and multiple other State and Federal agencies. As such, the aliphatic and aromatic hydrocarbon fractionation approach was designated as the appropriate methodology for evaluation of risks from exposure to petroleum hydrocarbons at the Hero Lands property.

6.1 Assessing health risks from petroleum hydrocarbon exposure – total petroleum hydrocarbons (TPH) mixtures vs. petroleum hydrocarbon fractionation

The current version of the RECAP regulation allows for assessing the health risks from petroleum hydrocarbon exposure using two methodologies; TPH-mixtures and petroleum hydrocarbon fractionation. Both methodologies rely on the same basic principle of applying the toxicity of one “indicator” or “surrogate” compound to a range of petroleum hydrocarbons. TPH-mixtures analysis is the first of the two methods developed and it involves grouping both aliphatic and aromatic hydrocarbons into rather wide groups or ranges based upon their carbon numbers. The TPH-mixture groupings are as follows:

- Gasoline Range Organics (TPH-GRO) (C6-C10)
- Diesel Range Organics (TPH-DRO) (C10-C28)
- Oil Range Organics (TPH-ORO) (>C28)

Screening values for each of these ranges are based upon the assumption that all compounds detected within a given TPH-mixture range have the same toxicity as the most potent compound in the range, which is designated as the surrogate compound for that range. For example, the screening concentration for TPH-ORO (>C28) is based upon the reference dose (RfD)³ for the compound pyrene (C₁₆H₁₀). As there were no previously developed RfDs for the TPH-ORO range, pyrene was selected as a conservative surrogate. However, the use of the RfD for pyrene greatly over predicts the toxicity of the longer-chain aliphatic and

³ Reference dose (RfD) - an estimate of a daily exposure level for a human population, including sensitive subpopulations, that is likely to be without an appreciable risk of deleterious effects during a lifetime (LDEQ, 2003b)

aromatic hydrocarbons that are contained with the TPH-ORO mixture range. Furthermore, pyrene is a 16-carbon compound and does not even fall within the carbon range for which TPH-ORO is designated (>C28).

The primary difference between the TPH-mixture methodology and the aliphatic and aromatic hydrocarbon fractionation methodology is that the fractionation methodology classifies petroleum hydrocarbons based upon their structure into separate aliphatic and aromatic hydrocarbon fractions and provides a smaller carbon range for each fraction. This is important, as aromatic hydrocarbons tend to have a greater toxicologic potency than aliphatic hydrocarbons of the same carbon number. The hydrocarbon fractionation methodology divides petroleum hydrocarbons into much smaller groupings and an appropriate surrogate compound is designated for each aliphatic or aromatic hydrocarbon fraction; thus, providing a much more precise measure of the potential health risks associated with petroleum hydrocarbons detected in the environment. The individual aliphatic and aromatic hydrocarbon fractions are given below:

- Aliphatic C6-C8
- Aliphatic >C8-C10
- Aliphatic >C10-C12
- Aliphatic >C12-C16
- Aliphatic >C16-C35
- Aromatic >C8-C10
- Aromatic >C10-C12
- Aromatic >C12-C16
- Aromatic >C16-C21
- Aromatic >C21-C35

Due to this increased precision and additional reasons discussed in-depth below, the aliphatic and aromatic hydrocarbon fractionation method is preferred over the TPH-mixture method for assessing health risks from petroleum hydrocarbon exposure.

6.2 Petroleum hydrocarbon fractionation is the preferred method for assessing health risks associated with petroleum hydrocarbon exposure

In keeping with RECAP (LDEQ, 2003b) which states: *“Petroleum-impacted soil and groundwater shall be assessed using the TPH Fraction and Indicator Approach as described by the TPH Criteria Working Group (TPHCWG)”* the petroleum hydrocarbon fractionation method is the proper and preferred method for the evaluation of health risks from petroleum hydrocarbon exposure. The TPH-mixture data (i.e., TPH-GRO, TPH-DRO, and TPH-ORO), as assessed by plaintiff’s experts, were considered as a screening tool, but were not included in the final assessment for evaluating health risks, as these data are not reflective of the actual petroleum hydrocarbon concentrations that may be present on a site and are not the most accurate or precise method to address toxicological risks from petroleum hydrocarbon exposure.

The LDEQ indicates its clear preference for petroleum fraction-specific results as evidenced in its response to a frequently asked question below:

Q: At an AOI impacted with TPH, the reported concentrations for TPH-G and TPH-D were above the SS. Therefore, additional samples were collected from the area of greatest

impact and analyzed using the fractionation method. All of the samples were ND for all of the fractions. Is it still necessary to address the TPH-G and TPH-D in the RECAP assessment?

A: Site management decisions should be based on the fractionation data (assuming it meets all QA/QC requirements) since this data is more specific and thus more representative of site conditions. (emphasis added) (LDEQ, 2012)

In addition, the use of aliphatic and aromatic fractionation methods is further validated in Appendix D of RECAP (LDEQ, 2003a; LDEQ, 2003b). Appendix D states:

If TPH fractionation data and TPH mixture data have both been collected at an AOI and the two data sets yield different conclusions concerning management of the AOI, then management decisions shall be based on the fractionation data since the fractionation method yields more specific information regarding the TPH constituents present and thus more accurately characterizes site conditions. (emphasis added) (LDEQ, 2003a; LDEQ, 2003b)

Most states recommend the use of fractionation-based methods for evaluation of petroleum hydrocarbons including but not limited to: Alaska (ADEC, 2000), Connecticut (CDEEP, 2012), Indiana (IDEM, 2010); Florida (FDEP, 2011), Massachusetts (MDEP, 2002), Minnesota (MPCA, 1999), Mississippi (MDEQ, 2002), Missouri (MDNR, 2013), Montana (MDEQ, 2016), New Jersey (NJDEP, 2008), New Mexico (NMED, 2014), Ohio (OHEPA, 2010), Oklahoma (ODEQ, 2012), Oregon (SODEQ, 2017), South Carolina (SHDHEC, 2001); Texas (TCEQ, 2010), Utah (UDEQ, 2012), Virginia (VDEQ, 2016), Washington (SOWDE, 2016), West Virginia (WVDEP, 1999), and Wyoming (WDEQ, 2016).

Federal risk assessment guidance also recommends the use of hydrocarbon fractionation. The USEPA Regional Screening Levels Frequent Questions provides details on the use of TPH and fractionation data in the use of human health risk assessments. As stated by the USEPA: *“TPH is a term intended to refer to the total mass of hydrocarbons present without identifying individual compounds”* (USEPA, 2020).

In order to conduct a human health risk assessment at a site with the presence of petroleum hydrocarbons, risk assessors need to know the chemical composition of the hydrocarbons present in site media. As such, the USEPA states: *“traditional TPH measurement techniques provide no specific information about the hydrocarbons that are detected. Because TPH is not a consistent entity, the assessment of health effects and development of toxicity values for mixtures of hydrocarbons are problematic”* (USEPA, 2020). The limitations of TPH-mixture analysis are evident; as such, the USEPA has recognized the need to use a fractionation-based approach and has adopted this methodology (USEPA, 2017b).

The USEPA Provisional Peer-Reviewed Toxicity Values (PPRTV) for Complex Mixtures of Aliphatic and Aromatic Hydrocarbons supports a fraction-based approach to risk assessment for complex mixtures of aliphatic and aromatic hydrocarbons (USEPA, 2009). The PPRTV approach is based on the fraction-based approach used by the Massachusetts Department of Environmental Protection (MADEP) and the Total Petroleum Hydrocarbon Criteria Working Group (TPHCWG). The MADEP and TPHCWG derive toxicity values for each fraction based on surrogate compounds selected to represent the toxicity of the respective aliphatic or aromatic fractions. The USEPA's rationale for adoption of the fractionation approach is based on several factors:

First, the development of the "fraction approach" by MADEP and TPHCWG represents the collective wisdom and scientific consensus of numerous scientists involved from governmental agencies, professional organizations, academia, and industry. Second, risk assessment of a chemical mixture, particularly one that is changing due to weathering, is a very difficult and complex issue. The "fraction approach" coupled with analytical information on complex mixtures of aliphatic and aromatic hydrocarbons from a given hazardous waste site, represents a reasonable, flexible, and best available methodology for risk assessment. Third, U.S. EPA scientists have employed computational chemistry and statistical methods to assess the fractionation scheme and found supporting evidence for selecting the fractions in this report. (USEPA, 2009)

The USEPA's preference for the fractionation methodology is evident as the PPRTV document reports:

"TPH is a loosely defined aggregate that depends on the method of analysis as well as the contaminating material; it represents the total mass of hydrocarbons without identifying individual compounds. As TPH is not a consistent entity, the assessment of health effects and development of toxicity criteria such as oral reference doses (RfDs) and slope factors for the complex mixture as a whole are problematic."

And,

"Thus, any attempt to assess the health effects of TPH from the individual hydrocarbon components is impractical because many of the known components lack appropriate toxicity data and criteria" (USEPA, 2009).

Toxicological data may be available for whole, unweathered hydrocarbon products; however, there are several limitations to compositional variability attributable to differences in the crude oils from which hydrocarbons are refined/produced. Furthermore, as stated in the PPRTV: *"Toxicity data for whole hydrocarbon products that are relatively heterogeneous are not necessarily applicable to the weathered materials or transport fractions to which exposure actually occurs"* (USEPA, 2009). As such an evaluation of site media for potential human-health risks that may be associated with site usage should involve

aliphatic and aromatic hydrocarbon fractionation data as this methodology permits an evaluation of the toxicological data relative to each fraction.

In addition to federal and state regulatory risk assessment guidance supporting the use of aliphatic and aromatic petroleum hydrocarbon fractions, many peer-reviewed scientific articles identify aliphatic and aromatic fractionation methodology as the appropriate method for evaluating and quantifying toxicological risks from petroleum hydrocarbons in site media (ATSDR, 1999a; Pinedo et al., 2012a; Pinedo et al., 2012b; Wang et al., 2012). Pinedo et al. (2012a) states: *“TPH determination does not allow a risk assessment of polluted soils, because risks are highly dependent on the hydrocarbon composition. A first separation between aliphatic and aromatic hydrocarbons is necessary in order to get the quantitative risk assessment.”* Pinedo et al. (2012b) states: *“TPH concentration is not a suitable parameter for risk assessment since it includes compounds with very different physicochemical and toxicological properties. TPH should be divided into fractions according to their physicochemical and toxicity properties to carry out a suitable risk assessment. Fractionation has been sorted in terms of aliphatic and aromatic compounds”* The Agency for Toxic Substances and Disease Registry reports: *“The ATSDR approach, as reflected in this profile, focuses on an assessment of the health effects of petroleum hydrocarbon transport fractions, as suggested by the TPHCWG”* (ATSDR, 1999b).

For the evaluation of petroleum hydrocarbons on site, aliphatic and aromatic hydrocarbon fraction data are used in the site human health risk evaluations, as the use of fractionation data is the scientifically accepted method for assessing potential health risks from exposure to petroleum hydrocarbons. Defense experts provided aliphatic and aromatic hydrocarbon fractionation data. However, the plaintiff’s experts relied on the analysis of TPH-mixtures (i.e., TPH-GRO, TPH-DRO, TPH-ORO). Thus, in contrast to using the best available scientific methodology, the plaintiff’s experts rely on TPH-mixture data in forming their opinions, the defendants’ experts have collected fractionation data which allows for the assessment of human health risks.

TPH-mixtures account for a wide range of loosely defined hydrocarbon aggregates that represent the total mass of a range of hydrocarbons without identifying specific compounds within the mixture. As TPH-mixtures are not a consistent homogeneous entity, the assessment of human health effects and development of toxicity criteria for such complex mixtures over such broad ranges of hydrocarbons is problematic.

Unlike the TPH-mixture data (i.e., TPH-GRO, TPH-DRO, TPH-ORO), aliphatic and aromatic hydrocarbon fractionation data account for the age and environmental weathering of petroleum hydrocarbons. In addition, hazard or risk of exposure to hydrocarbon fractionations is based on derived toxicity values for selected compounds within each fraction (i.e., surrogate compounds). The fractionation of petroleum hydrocarbons into aliphatic and aromatic hydrocarbons provides a more accurate estimate of the risk associated with a given sample, as fractionation is based on toxicological data from the respective

surrogate compound and is specific to the structural hydrocarbon family (i.e. aliphatic vs. aromatic hydrocarbons).

The MADEP was the first agency to evaluate human health risk on petroleum-impacted sites based on an evaluation of the differences in toxicity of the individual aliphatic or aromatic fractions (MADEP, 1994). Shortly afterwards, the TPHCWG was formed and recommended a “*fraction-based approach*” for assessing human health risks associated with TPH exposures (MADEP, 2002; MADEP, 2003; TPHCWG, 1996; TPHCWG, 1997; TPHCWG, 1998a; TPHCWG, 1998b; TPHCWG, 1999). Both the MADEP and the TPHCWG defined aliphatic and aromatic hydrocarbon fractions based on the expected environmental fate, structural similarity (i.e., carbon number), and derived toxicity values for surrogate compounds representative of the specific fractions. As such, both the MADEP and the TPHCWG recommend a “*fraction-based approach*” for assessing human health risks associated with petroleum hydrocarbon exposures. The TPHCWG identified 13 fractions based on the expected environmental behavior and toxicological data of individual petroleum compounds. These hydrocarbon fractions include six aliphatic fractions (C5-C6, >C6-C8, >C8-C10, >C10-C12, >C12-C16, and >C16-C35) and seven aromatic fractions (C6-C7, >C7-C8, >C8-C10, >C10-C12, >C12-C16, >C16-C21, and >C21-C35). The toxicity associated with these petroleum fractions was generated from toxicity studies on whole products, petroleum mixtures, and individual petroleum compounds. From this evaluation, the TPHCWG chose not to use the toxicity data from a single reference compound to represent the toxicity of each fraction and reviewed multiple reference/surrogate compounds within each fraction to develop conservative reference concentrations (RfCs) and reference doses (RfDs) that account for the uncertainty in the underlying toxicity database (TPHCWG, 1999).

Two primary methods have been developed to analyze petroleum hydrocarbon fractions in environmental media: the TPHCWG method and the MADEP extractable petroleum hydrocarbon (EPH)/volatile petroleum hydrocarbon (VPH) method. Both methods were developed to quantify a petroleum hydrocarbon mixture as discrete fractions that can be used in human health risk assessments based on toxicity or environmental behavior. Samples are first separated by aliphatic and aromatic compounds using silica gel prior to GC analysis and fraction quantification, allowing the identification of specific petroleum products and an assessment to the degree of weathering (USEPA, 2007).

As petroleum hydrocarbons are not a consistent entity, the assessment of health effects based on wide ranges of petroleum hydrocarbon as mixtures (i.e., TPH-GRO, TPH-DRO, and TPH-ORO) is problematic. The TPH-mixtures method encompasses a wide range of hydrocarbons including both aliphatic and aromatic hydrocarbons into the same mixture. As a result, the hydrocarbons present within these large ranges have widely differing chemical and physical properties which do not support their usage in human health risk assessments. As TPH-mixture analysis is not appropriate for use in human-health risk assessments, the fractionation-based approach was developed to reflect differences in the physiochemical properties of the aliphatic and aromatic hydrocarbons necessary to evaluate potential health risks to petroleum hydrocarbons. An overview of the aliphatic and aromatic hydrocarbon fractions and TPH-mixtures (i.e.,

TPH-GRO, TPH-DRO, and TPH-ORO) and their toxicological basis in the formation of RECAP Standards are presented in **Appendix E**.

7.0 Limitations of TPH-mixture analysis in human health risk assessments

Under USEPA risk assessment guidance, the hazard and health risk assessments conducted to support risk management decisions at contaminated sites require an understanding of the chemical composition of the hydrocarbons that are present in potentially contaminated media (USEPA, 2009). However, traditional TPH-mixture measurement techniques provide no specific information about the hydrocarbons that may be detected at a site. For instance, crude oil is categorized as a single substance, petroleum (CAS# 8002-05-9); however, this classification can be misleading as crude oil and other petroleum mixtures consist of a complex combination of hydrocarbons composed predominantly of aliphatic, alicyclic, and aromatic hydrocarbons (USEPA, 2009). Whereas commonalities exist in the identity of chemicals found in different types of crude oil, marked heterogeneity in the percent mass of constituent chemicals across crude oil types is not uncommon. Because petroleum hydrocarbons are not a consistent entity, the assessment of health effects and the development of toxicity values for mixtures of hydrocarbons are complex, and therefore petroleum hydrocarbon fractionation is the preferred method for assessing health risks from environmental exposure to petroleum hydrocarbons.

In addition, the analytical methods used in the laboratory to analyze petroleum hydrocarbons as TPH-mixtures can provide results which can be problematic for the evaluation of site media. For example, the primary method of TPH analysis is EPA 8015, consisting of gas chromatography (GC) with flame ionization detection (GC/FID). This method was originally developed to determine the concentrations of various nonhalogenated volatile and semi-volatile organic compounds and was modified to include the analysis of TPH gasoline range organics (TPH-GRO, C6-C10) and diesel range organics (TPH-DRO, C10-C28). As a GC-based method, EPA 8015 is able to provide information on product type and composition in addition to quantifying the total amount. However, sample matrix effects may be higher in this GC-FID method, and the identification of TPH as TPH-GRO and TPH-DRO may be complicated by an overlap of carbon number ranges for TPH-GRO and TPH-DRO; resulting in portions of the TPH-GRO range to be reported as TPH-DRO and vice versa (API, 2001). Overlap between the TPH-mixture ranges could be problematic as this could create inaccuracies in the concentrations reported in site media and an inaccurate representation of the true site conditions. Additionally, TPH-mixture methods are at risk of interference from non-petroleum compounds which can result in a bias in reported concentrations in site media as will be described below.

7.1 TPH-mixture analysis provides an imprecise estimate of health risks from petroleum hydrocarbon exposure

The use of TPH-mixture analysis to evaluate risks to human health from constituents in environmental media is imprecise and is not the methodology of choice as a result of two main deficiencies: 1) TPH-

mixture analysis fails to separate the petroleum hydrocarbons that may be present into aromatic and aliphatic hydrocarbon groupings; and 2) TPH-mixture analysis groups hydrocarbons into wide hydrocarbon ranges and applies toxicological criteria based on a surrogate compounds demonstrating the greatest inherent toxicity for compounds potentially present (or sometimes not present, as is the case with pyrene and TPH-ORO) within the respective TPH-mixture range.

To evaluate risks to human health, petroleum hydrocarbons should be separated into aliphatic and aromatic structural groupings. Aliphatic hydrocarbons contain carbon and hydrogen joined together in straight chains, branched chains, or non-aromatic rings. Aromatic hydrocarbons contain carbon and hydrogen atoms, where the carbon atoms form stable unsaturated cyclic compounds and can contain one or more benzene rings. Aromatic and aliphatic hydrocarbons of similar carbon number can have significant differences in toxicological properties due to their actions in the body. Under RECAP guidance, the RECAP Screening Standards or RECAP Standards for TPH-mixtures (i.e., TPH-GRO, TPH-DRO, and TPH-ORO) are calculated based on the aromatic hydrocarbon fraction with the most conservative (i.e., lowest) RfD (indicating the compound has the greatest toxicity), which is then used to calculate a Screening Standard or RECAP Standard for the entire TPH-mixture including the less toxic aliphatic hydrocarbons. For example, TPH-DRO (C10-C28) is represented by the oral reference dose (RfD_o) for aromatics >C16-21 and the inhalation reference dose (RfD_i) for aromatics >C10-C16. TPH-ORO (>C28) is represented by the RfD_o for Aromatics >C21-C25 (Adeniji et al., 2017). As a result, the use of TPH-mixture analysis becomes problematic as it is based on toxicological data established for specific aromatic hydrocarbons (which are also based on surrogate compounds designated as aromatic compounds) that may not even be present in the sample analyzed and greatly overestimate the toxicity of the aliphatic hydrocarbons that would likely contribute to the presence of detectable levels of a TPH-mixture present in site media. The hydrocarbon fractionation methodology separates aliphatic and aromatic hydrocarbons, eliminating this source of error.

Secondly, as detailed above, the TPH-mixture method divides petroleum hydrocarbons into three wide ranges, whereas the fractionation method divides petroleum hydrocarbons into ten different fractions. This provides a much better resolution of the hydrocarbon profile of a site and provides more precision in the estimation of health risks, as the surrogate compounds chosen for the narrower fractions more accurately represent the toxicity of the fraction, whereas this is not the case for the broad ranges of hydrocarbons included in the TPH-mixtures.

Additionally, TPH-mixture analysis groups hydrocarbons into wide hydrocarbon ranges based on toxicological criteria for surrogate compounds that typically contain a hydrocarbon number at the lower range of the respective TPH-mixture. As presented below, the surrogate compound often contains lower hydrocarbon numbers and quickly evaporates or is degraded due to weathering in the environment. As a result, TPH-mixture analysis does not account for weathering of petroleum hydrocarbons over time and overestimates the toxicity of the mixture if the sample collected is enriched with heavier hydrocarbon compounds that would remain after the weathering process. The hydrocarbon fractionation method

separates the aliphatic and aromatic compounds into small hydrocarbon ranges providing a more precise measure of the specific hydrocarbon range present in soil which allows for a more accurate toxicological evaluation.

7.2 TPH-mixture analysis does not account for weathering of petroleum hydrocarbons in the environment and standards derived for TPH-mixture analyses are derived based on surrogate compounds that may not be present in the sample

Weathering can have a significant impact on the composition of petroleum hydrocarbons in the environment. The weathering of petroleum hydrocarbons in the environment reduces the overall toxicity of the hydrocarbon mixtures. Weathering in the environment causes chemical and physical compositional changes to the petroleum hydrocarbon mixtures. For example, the more volatile petroleum components such as those with a carbon range of eight or below (e.g., hexane and benzene, toluene, ethylbenzene, and xylene compounds) may rapidly evaporate or degrade and will not be present in the weathered petroleum hydrocarbon mixture.

Despite the differing physical and chemical characteristics of crude oil, some generalizations can be made regarding its behavior in the environment. Freshly spilled crude oil, in which the composition of the oil residue resembles that of the unspilled source oil, will often release most of its volatile organics over hours to a few days when spilled in well-ventilated, warm environments. Previous studies have evaluated the presence of VOC air concentrations in the breathing zone above or in the direct vicinity of unweathered crude oil; a time course analysis from various crude oil releases demonstrates that VOCs released from crude oil typically dissipate over a period of a few days (Harrill et al., 2014; LDEQ, 2003b).

As the chemical and physical parameters of the crude oil change due to weathering, the potential toxicological hazards change as well. Due to changes associated with weathering in the environment, petroleum hydrocarbon mixtures may become toxicologically different from the original petroleum mixture. For example, for a direct inhalation pathway to exist (i.e., volatilization of constituents from water or soil), constituents must have a sufficient vapor pressure to volatilize from soil or surface water and become airborne at concentrations resulting in a meaningful dose to a receptor. Initially, the lighter components of crude oils (e.g., hexane, benzene, ethylbenzene, toluene, and xylenes), which fall in the range of gasoline-range organics (TPH-GRO) may rapidly volatilize and will not be present in a weathered mixture. Plaintiff's experts contend that the site is contaminated with gasoline, diesel, and oil range petroleum hydrocarbons. Although diesel range organics contain hydrocarbons with a wide range of vapor pressures, the more volatile compounds rapidly dissipate through the weathering process, leaving behind compounds with little or no volatility (Osuji et al., 2006; Stout et al., 2006; Thayer et al., 2001). Not only would the contaminant concentration decrease due to chemical degradation, dispersion of a finite mass of a volatile contaminant over a larger area would dilute the concentration to levels below those associated with adverse health effects. For example, aliphatic hydrocarbons in the 12-16 carbon range, (which would approximate the TPH-DRO range) have an extremely low vapor pressure of 0.000076 atm, and aliphatic hydrocarbons in the 16-35 carbon range (similar to the TPH-ORO range) have a vapor pressure of 0.0000011 atm. Aromatic hydrocarbons in these ranges have even lower vapor pressures

(ATSDR, 1999b). Thus, due to loss of the more volatile components, the weathered crude oil becomes less of an inhalation hazard from a toxicological standpoint. As TPH-mixture data does not provide any information on specific compounds or a breakdown of the aliphatic and aromatic hydrocarbon ranges, it cannot determine how much weathering a product has undergone.

TPH-mixture analyses do not account for physical and compositional changes of petroleum hydrocarbons in the environment over time. Furthermore, the TPH-mixture methodology applies to a wide range of hydrocarbons that combine both aliphatic and aromatic hydrocarbons into the same mixture. A few generalities can be made regarding the use of surrogate compounds for TPH-mixtures and how weathering affects the site evaluation of petroleum hydrocarbons if TPH-mixtures (i.e., TPH-GRO, TPH-DRO, or TPH-ORO) are used. As indicated earlier, the surrogate compounds for TPH-mixtures are based on the most toxic compound containing a carbon number that falls within the respective TPH-mixture range. For example, naphthalene ($C_{10}H_8$) contains ten carbon atoms and is used as one of the surrogate compounds for TPH-DRO. However, this is not always the case as TPH-ORO (>C28) is based on the surrogate compound pyrene, a 16-carbon compound, which actually falls within the TPH-mixture range for TPH-DRO (C10-C28) but is used as the surrogate compound for TPH-ORO (>C28). As a result, the wide range of hydrocarbons represented by TPH-mixtures have standards which are derived by a surrogate compound (typically the compound with the greatest inherent toxicity) that greatly overestimates the toxicological risk. Furthermore, the surrogate compounds for a given TPH-mixture are often those with the lowest number of hydrocarbons compared to those with higher hydrocarbons within the respective TPH-mixture range, or as indicated with TPH-ORO even fewer carbon atoms than the given TPH-mixture range for which it is used. These surrogate compounds, such as naphthalene, are most likely to evaporate or degrade, leaving behind only the compounds with the greatest number of carbons (and less inherent toxicity) in site media. When the TPH-mixture method assumes that all hydrocarbon compounds within a mixture have the same toxicity as the lowest molecular weight compounds (which may not be present in site media due to evaporation or degradation), the risk and toxicity of what is actually present in site media is overestimated. This is because a soil concentration of TPH-GRO, TPH-DRO, or TPH-ORO is treated as if the entire sample is comprised of the surrogate compound, which often does not reflect the actual chemical composition of the sample in site media over time, as the surrogate compound may not even be detected within site media where detections of TPH-mixtures occur. In conclusion, the use of TPH-mixture analysis may result in comparing site sample results to a RECAP Screening Standard for TPH-GRO, TPH-DRO, or TPH-ORO that is derived from toxicological data from a surrogate compound which may not even be present in the sample analyzed.

7.3 TPH-mixture results are influenced by the presence of non-petroleum hydrocarbon sources

TPH-mixture data are also subject to influence from non-petrogenic sources, particularly when the sample does not undergo a cleanup step to remove non-polar compounds. Extractable hydrocarbons and non-hydrocarbons from naturally occurring biogenic material can result in over-estimations of petroleum hydrocarbons in environmental samples because standard TPH-mixture analyses do not differentiate between petroleum hydrocarbons and background organic material (Wang et al., 2009). Thus, non-

petroleum sources may yield false-positive TPH-mixture results in the analysis that can exceed regulatory standards. Solvent extraction of soils using standard USEPA methodology can yield hydrocarbons and non-hydrocarbons with the same boiling range as petroleum hydrocarbons. This can result in an overestimation of the concentration of TPH-mixtures (Stout and Uhler, 2003). One of the most common sources of background organic material in soil and sediment samples is vascular plant debris (Stout and Uhler, 2003; Wang et al., 2009). Stout and Uhler (2003) note: *“The extractable component within soils and sediments containing plant debris can be significant, particularly in moist, highly vegetated environments where peat or other organic-rich soils accumulate (or had in the past).”* For example, the TPH content of various biological materials was reported by TPHCWG (Weisman, 1998) as follows:

- Fresh Pine needles: 16,000 ppm
- Pine bark: 2,400 ppm
- Pine needle compost: 1,200 ppm
- Maple tree seeds: 7,100 ppm
- Oak leaves dried: 18,000 ppm
- Grass, dried: 14,000 ppm
- Gall nuts: 9,700 ppm

For these reasons, measurements of petroleum hydrocarbons as *“total petroleum hydrocarbon”* mixtures (i.e., TPH-DRO or TPH-ORO) are not preferred for evaluating human exposure and risk. For example, the ASTM indicates:

X2.5.3 Use of TPH or TOC Measurements in Risk Assessments— Various chemical analysis methods commonly referred to as “Total Petroleum Hydrocarbons” (TPH) or “Total Organic Content” (TOC) are often used during an initial site assessment to focus future investigations toward particular compounds and/or media. These methods usually determine the total amount of hydrocarbons present as a single number, and give no information on the types of hydrocarbons present. Such TPH or TOC methods may be useful in screening assessments where the whole product toxicity approach is appropriate to determine the need for further sampling. In general, these measurements should not be used for risk assessments, because the general measure of TPH or TOC provides insufficient information about the amounts of individual compounds present to accurately characterize potential risk. More information on petroleum hydrocarbons is available from the Total Petroleum Hydrocarbon Criteria Working Group (TPHCWG) effort. Refs (11-14). (ASTM, 2004)

7.4 TPH-mixture standards cannot be used as a bright-line above which adverse health effects are possible

The LDEQ RECAP Soils_{SSni} for TPH-ORO serves as an example of how a detectable level of TPH-ORO above the Soils_{SSni} cannot be used to opine a risk to human health exists. For example, the LDEQ chose pyrene (the most toxic constituent) as the representative surrogate compound for establishing the standard for TPH-ORO (Edwards et al., 1997; LDEQ, 2003b). The RfD for pyrene is based on a feeding study of pyrene in laboratory rodents that produced a NOAEL (i.e., no observable adverse effects level) of 75 mg/kg-day pyrene in the diet. The USEPA derived a RfD of 0.03 mg/kg-day based on this NOAEL value (USEPA, 2017a), which was used by LDEQ to derive the RECAP Soils_{SSni}. When one calculates the daily dose of TPH-ORO an individual may receive if exposed to impacted soil at the concentration of the Soils_{SSni} for TPH-ORO (i.e., 180 mg/kg), the total dose of TPH-ORO from both ingestion and dermal routes of exposure is 0.0029 mg/kg-day. When compared to the daily dose in laboratory animals used to derive the Standard Screening (i.e., the NOAEL of 75 mg/kg-day), a dose which produced no adverse health effects, the dose at the Soils_{SSni} is almost 26,000 times less than the no-effect dose of pyrene measured in laboratory animals. It is notable that the lowest dose of pyrene producing effects in the laboratory animal study is 125 mg/kg-day, which is 43,000 times greater than the dose an individual would receive at the Soils_{SSni} of 0.0029 mg/kg-day. Thus, not only does using pyrene as a surrogate for the derivation of screening standards for TPH-ORO overestimate the risk to human health, TPH-mixture results may be compared to a standard based on a surrogate compound that is not present in the mixture, and therefore would considerably overestimate the possible risk to human health.

8.0 Site characterization

8.1 Oil and gas well survey

The Louisiana Department of Natural Resources (LDNR) Strategic Online Natural Resources Information System (SONRIS) database indicates 85 oil and gas wells have been reported within a one-mile radius of the Hero Lands property, of which the following designations are reported: three Act 404 orphan wells (no product specified), seven active oil producing wells, one active injection community salt water disposal well, one active injection nonhazardous industrial well, five active injection produced salt water wells, 21 dry and plugged wells (no product specified); four PA-35 temporary inactive wells, six permit expired wells, five plugged and abandoned gas wells, 18 plugged and abandoned oil wells, six plugged and abandoned wells (no product specified), one reverted to single completion well (no product specified), one shut-in dry hole future utility well (no product specified), three shut-in productive future utility gas wells, one shut-in productive future utility oil well, and two unable to locate no plugged and abandoned oil well.

According to the report of Calvin Barnhill, a total of 39 wellbores were drilled within the Hero Lands boundary, of which 14 are currently classified as active (e.g., not plugged and abandoned): five active productive wells, three LDNR status 33, five active SWD walls, and one orphan well. Barnhill reported 21

wells that have been plugged and abandoned and three dry hole wells within the Hero Lands property. A map depicting the active and historical oil and gas E&P wells within a 1-mile boundary in relation to the Hero Lands property tracts is present in **Appendix C Figure C-3**.

8.2 Groundwater well survey

Based on a query of the LDNR SONRIS database, there is one abandoned domestic water well, one active irrigation water well, one active oil and gas rig supply water well, five piezometer water wells, and 61 monitoring water wells within one mile of the Hero Lands Property. There are 119 plugged and abandoned water wells (including monitoring, piezometer, rig supply, and test holes) within one mile of the Hero Lands Property. With the exception of monitoring and piezometer wells, all water wells are reported at a depth equal to or greater than 250 feet below ground surface (bgs) and are screened within the Gramercy Aquifer, Mississippi River Alluvial Aquifer Surficial Confining, and New Orleans Aquifer system surficial confining unit. Within the site boundary, there are four monitoring wells, two plugged and abandoned rig supply, and two plugged and abandoned monitoring wells. No water wells within the property or within one mile of the property boundary are used for potable purposes. A map depicting registered water wells within a one-mile radius of the Hero Lands property is presented in **Appendix C Figure C-4**.

8.3 Exposure pathway analysis

A key part of the risk assessment process is the determination of the degree to which an individual may be exposed to an impacted medium. If there is no exposure to a given medium, there can be no dose, and thus no risk of adverse health effects. This process is known as an exposure pathway analysis.

An exposure pathway is made up of four elements:

- A source and mechanism of chemical exposure;
- A retention or transport medium;
- A point of potential human contact with the impacted medium, and;
- An exposure route at the contact point.

As stated by the USEPA (1989), *“An exposure pathway describes the course a chemical or physical agent takes from the source to the exposed individual. An exposure pathway analysis links the sources, locations, and types of environmental releases with population locations and activity patterns to determine the significant pathways of human exposure.”*

8.3.1 Soil exposure pathway analysis

Soil exposure pathways may include incidental ingestion or dermal contact with impacted soil. Based on plaintiff’s experts’ reports, the defendants’ experts’ reports, and the plaintiff’s deposition testimony the historical and current use of the Hero Lands Property includes active and historical industrial activities (oil and gas E&P activities and potential recreational activities. As reported by the plaintiffs, the future land

usage of the property will continue to be industrial activities. Mr. Allen Hero testified that the intent was for current operations on the property to continue (A. Hero: p. 134-145). Mr. George Alfred Hero IV testified that potential future use for the site would be development into an industrial site (G. Hero: p. 293). Under current and future site conditions, it is possible that a person using the property for these purposes may have direct contact with soil. Thus, the soil exposure pathway was further evaluated.

8.3.2 Groundwater exposure pathway analysis

Based on the water well survey presented in Section 8.2 of this report, no water wells are used on the Hero Lands property as a source of potable drinking water. A complete characterization of the groundwater present within the Hero Lands property is provided in the expert reports prepared by ERM. Three water-bearing zones were sampled beneath the Hero Lands property. These zones consisted of the A-zone – screened within the upper 30 feet bgs; the B-zone – screened between 20 and 52 feet bgs; and the C-zone – screened between 76 and 86 feet bgs. The results of testing in A-zone reported low yield and water quality consistent with groundwater classification 3 (GW₃) as classified by ERM. B-zone and C-zone are reported as meeting groundwater class 2 (GW₂) criteria according to ERM. As there are no active water supply wells used for potable purposes on the Hero Lands property and no registered or known uses of the shallow A, B, or C-zones for potable purposes, the groundwater exposure pathway within the Hero Lands property is deemed incomplete. Furthermore, public water supply is provided to the Hero Lands property and surrounding areas by the Belle Chasse public water system. A full groundwater analysis with comparison of site constituent concentrations to RECAP Standards is provided in the expert report of Ms. Angela Levert.

8.3.3 Consumption of Wildlife

The plaintiff's experts have alleged that possible human exposure to chemical constituents may occur through the ingestion of wildlife. Plaintiff's expert Dr. William Rogers states: *"Any contamination that gets into the local food chain has the potential to be ingested by hunters, fishermen, and others who frequent the property and consume wildlife that are harvested there. The chance of contaminated wildlife ingestion is further increased by the presence of commonly consumed game animals and other forms of wildlife, all of which can be found on the property."* And, *"The culture and living habits of rural populations in Louisiana are unique in that many tend to "live off the land" more than other metropolitan populations of Louisiana. Thus, residents of all ages from this community are more likely to be exposed than the average Louisiana citizen to contaminants by ingesting biota from on-site, and from exposure to contaminants potentially migrating from the property."*

There is no evidence to suggest that the consumption of wildlife from the Hero Lands property or adjacent properties represents a significant exposure pathway or that this occurs at all. Wildlife are mobile, and the geographic extent of the alleged media contamination on the Hero Lands property is small in comparison to the potential area in which wildlife may travel. No testing has been conducted or evidence presented indicating that any alleged constituents on the Hero Lands property would accumulate in the

tissues of wildlife to any degree that would represent a significant exposure pathway. For instance, studies show that polycyclic aromatic hydrocarbons (PAHs, primary constituents of crude oil) are rapidly metabolized and do not tend to bioaccumulate at high concentrations in wild vertebrates despite their high lipid solubility (Douben, 2003; Eisler, 1987). As noted by the USEPA, the bioavailability of contaminants in soils incidentally ingested by wildlife is rarely considered because of the difficulty in making such measurements (USEPA, 2004). As such, constituents detected in soil would not accumulate in wildlife, and therefore do not represent a significant exposure pathway. Thus, the consumption of wildlife from the Hero Lands property is considered an insignificant exposure pathway based on lack of evidence that it would contribute significantly to a receptor's exposure to constituents at the site.

9.0 Site investigation activities on the Hero Lands property

A complete discussion of the site investigation activities, investigation procedures, methods, and results is provided in the reports of ICON and ERM. Site characterization, exposure pathway analysis, conceptual site models, and screening of environmental sampling data relative to LDEQ RECAP Screening Standards and RECAP Standards are presented in detail in the expert report of Ms. Angela Levert. Plaintiff's experts have used an inappropriate methodology in reaching opinions that "contamination" exists on the Hero Lands property that represents "unacceptable risks" to users of the property. As discussed above, regulatory values such as RECAP Standards can only be used to rule out the potential for adverse health effects and cannot be used to determine actual health risks. The estimation/calculation of health risks from a given exposure can only be performed by first estimating the dose of a constituent received by a given receptor (i.e. individual) and then comparing this dose to some type of health-based comparison value. I have conducted this analysis and the results are detailed below.

9.1 Results of soil risk assessment

For soil, an initial screening of the data compared the maximum soil concentrations from each of the designated property tracts (i.e., NW tract, NE tract, SE tract, and SW tract) to direct contact industrial soil screening standards ($Soil_{SSI}$) to identify constituents in soil warranting further evaluation under RECAP. Reported concentrations for all constituents reported as wet-weight and/or dry-weight fall below their respective industrial soil screening standards ($Soil_{SSI}$). Arithmetic average concentrations of arsenic in soil reported as wet-weight and/or dry-weight fall below state-specific background levels adopted by LDEQ for unrestricted land use. In accordance with RECAP guidance, the arithmetic average concentration is used for comparison to the RECAP Standard based on background concentrations (i.e., arsenic). A full RECAP assessment is provided in the report of Ms. Angela Levert. Despite all constituent concentrations on the four property tracts being reported below their respective industrial soil screening standards, I have conducted dose calculations under an industrial exposure scenario for arsenic, barium, and the total

TCDD TEQ⁴. For purposes of calculating a dose, I have considered both wet and dry-weight concentrations of constituents in soil to address hypothetical risks to potential receptors accessing the property under an industrial land use scenario. In addition to evaluating an industrial land use scenario, I have evaluated constituents under a residential exposure scenario as presented in **Appendix F**. For the sake of completeness, I have also conducted a risk assessment under industrial and residential exposure scenarios for exceedances in off-site sampling locations as presented in **Appendix G**. Exposure point concentrations (EPCs) used in the dose calculations are provided below.

Table 9.1.1: Exposure point concentrations (wet-weight)

Analyte		Wet-weight concentration (mg/kg)	Sample location	Depth (bgs)
Arsenic	site max	22.9	BC-1	4-6'
	maximum sample location average	18.9	BC-1	4-6'
	maximum tract average	5.1	SW Tract	0-18'
	maximum tract 95% UCL (0-2')	8.0	SW Tract	0-2'
Barium	site max	5,573	SB-13	0-2'
	maximum sample location average	5,143	SB-14R	2-4'
	maximum tract 95% UCL (0-2')	3,207	SW	0-2'
Total TCDD TEQ	site max	0.0000069	BC-16R	2-4'

Table 9.1.2: Exposure point concentrations (dry-weight)

Analyte		Wet-weight concentration (mg/kg-dry)	Sample location	Depth (bgs)
Arsenic	site max	27.3	SB-13	0-2'
	maximum sample location average	18.5	BC-1	4-6'
	maximum tract average	5.6	SW tract	0-18'
	maximum tract 95% UCL (0-2')	9.0	SW tract	0-2'
Barium	site max	9,320	SB-13	0-2'
	maximum sample location average	6,280	SB-13	0-2'
	maximum tract 95% UCL (0-2')	2,545	SW tract	0-2'
Total TCDD TEQ	site max	0.0000094	BC-16R	2-4'

In addition to calculating doses based on the arithmetic averages, doses were also calculated using 95% UCLs. Per LDEQ RECAP, the “AOIC shall be represented by: (1) the maximum constituent concentration

⁴ Toxicity Equivalence. Evaluation of tetrachlorodibenzo-para-dioxin (TCDD) and dioxin like compounds (DLC) includes consideration of the cancer risks and non-cancer effects of these contaminants. Toxicity equivalence factors (TEFs) are used as a measure of the toxicity of the DLCs relative to TCDD. Concentrations of DLCs measured in media are modified by TEFs to determine the dose of each DLC in a medium that is equivalent to a dose of TCDD. The modified DLC dose are expressed in terms of TCDD toxicity equivalence (TEQ). The total TCDD TEQ is the sum of the individual DLC's TEQs.

(SO, MO-1, MO-2, and MO-3) detected at the AOC/AOI... or (2) The 95 percent upper confidence limit on the arithmetic mean (95%UCL-AM) constituent concentration (MO-1, MO-2, and MO-3) detected at the AOI" (LDEQ, 2003b). The 95 percent upper confidence limit on the arithmetic mean (95% UCL-AM) for arsenic and barium both in wet-weight and dry-weight for surface soil within the 0-2' bgs range for each tract are presented below. For purposes of conducting dose calculations, the maximum 95% UCL-AM for both wet-weight and dry-weight among the four property tracks was used as a conservative approach. Outputs for 95% UCL calculations are presented in **Appendix H**.

Table 9.1.3: 95% UCL-AM concentrations by tract (0-2' bgs; wet-weight)

Analyte	Tract	Concentration (mg/kg)	UCL Calculation
Arsenic	NE	5.79	95% Student's-t UCL
	NW	NA	NA
	SE	7.57	95% Student's-t UCL
	SW	7.98	95% Modified-t UCL
Barium	NE	NA	NA
	NW	NA	NA
	SE	1,425	95% Student's-t UCL
	SW	3,207	95% H-UCL

Table 9.1.4: 95% UCL-AM concentrations by tract (0-2' bgs; dry-weight)

Analyte	Tract	Concentration (mg/kg-dry)	UCL Calculation
Arsenic	NE	8.05	95% Student's-t UCL
	NW	NA	NA
	SE	9.07	95% Student's-t UCL
	SW	9.04	95% Adjusted Gamma UCL
Barium	NE	NA	NA
	NW	NA	NA
	SE	1,815	95% Student's-t UCL
	SW	2,545	95% Adjusted Gamma UCL

As presented in the tables above, the reported concentrations for all constituents reported as wet-weight and/or dry-weight (arsenic reported as tract average) fall below their respective industrial soil screening standards (Soil_{ssi}). Furthermore, the 95% UCL-AM concentrations for arsenic and barium in soil from the 0-2' bgs depth range are reported below the MO-1 Soil_i and the Soil_{ni} RECAP Standards. Thus, under LDEQ RECAP guidance, all the respective constituents (reported as wet-weight or dry-weight) evaluated in site soil on the Hero Lands property fall below their respective industrial RECAP Standards (accounting for potential additive effects). As such, there is no risk to human health from exposure to soil constituents on site under the current and likely future industrial land use of the property, as well as a residential exposure scenario.

To further evaluate the potential for adverse health effects from exposure to constituents present on the property, I have performed dose calculations for potential receptors using the property. Dose calculations were conducted for an adult industrial receptor, as the property is used for industrial purposes. Dose calculations were conducted using methodology consistent with methodology employed by the LDEQ RECAP and USEPA.

9.1.1 Soil noncancer dose calculations

Arsenic, barium, and the total TCDD TEQ were included in the toxicological risk evaluation. RECAP provides guidance for the use of wet-weight data in comparison to direct contact standards. This method was confirmed by LDEQ for risk evaluations previously submitted and approved under RECAP MO-3. The USEPA requires use of dry-weight concentrations for evaluation of a direct contact pathway. For sake of completeness, the dose calculations for the direct contact pathway were conducted using both wet-weight and dry-weight results. Maps of the soil sample locations within the Hero Lands property can be found in **Appendix C Figure C-5**.

Dose calculations were conducted for arsenic, barium, and total TCDD TEQ. Dose calculations were derived for a hypothetical adult industrial scenario. Calculated doses were then compared to health-based toxicity benchmarks, including the RfDs, NOAELs, LOAELs, or benchmark doses (BMDs) that form the basis for the RECAP Standards, to determine whether the estimated doses calculated for each of the constituents exceeded toxicological benchmark levels.

Doses were calculated based on ingestion, dermal contact, and inhalation of soil particulates. Calculations of estimated doses for the industrial exposure scenarios are provided below. Exposure parameters used in the dose calculations are provided in **Appendix C**.

Table 9.1.5: Comparison of an adult industrial dose to health-protective benchmarks and toxicity effect levels (wet-weight)

Analyte		Total Intake (mg/kg-day)	RfD (mg/kg-day)	Daily Dose fold-below RfD	NOAEL (mg/kg-day)	Daily Dose fold-below NOAEL	LOAEL (mg/kg-day)	Daily Dose fold-below LOAEL
Arsenic	site max	9.76E-06	3.00E-04	31	8.00E-04	82	1.40E-02	1,434
	maximum sample location average	8.04E-06	3.00E-04	37	8.00E-04	100	1.40E-02	1,742
	maximum tract average	2.17E-06	3.00E-04	138	8.00E-04	368	1.40E-02	6,438
	maximum tract 95% UCL (0-2')	3.40E-06	3.00E-04	88	8.00E-04	235	1.40E-02	4,114
Barium	site max	2.39E-03	2.00E-01	84	6.30E+01	26,405	8.40E+01	35,207
	maximum sample location average	2.20E-03	2.00E-01	91	6.30E+01	28,613	8.40E+01	38,150
	maximum tract 95% UCL (0-2')	1.37E-03	2.00E-01	146	6.30E+01	45,886	8.40E+01	61,181
Total TCDD TEQ	site max	4.12E-12	7.00E-10	170	NA	NA	2.00E-08	4,850

Table 9.1.6: Comparison of an adult industrial dose to health-protective benchmarks and toxicity effect levels (dry-weight)

Analyte		Total Intake (mg/kg-day)	RfD (mg/kg-day)	Daily Dose fold-below RfD	NOAEL (mg/kg-day)	Daily Dose fold-below NOAEL	LOAEL (mg/kg-day)	Daily Dose fold-below LOAEL
Arsenic	site max	1.16E-05	3.00E-04	26	8.00E-04	69	1.40E-02	1,203
	maximum sample location average	7.89E-06	3.00E-04	38	8.00E-04	101	1.40E-02	1,775
	maximum tract average	2.40E-06	3.00E-04	125	8.00E-04	333	1.40E-02	5,832
	maximum tract 95% UCL (0-2')	3.86E-06	3.00E-04	78	8.00E-04	208	1.40E-02	3,632
Barium	site max	3.99E-03	2.00E-01	50	6.30E+01	15,789	8.40E+01	21,052
	maximum sample location average	2.69E-03	2.00E-01	74	6.30E+01	23,432	8.40E+01	31,243
	maximum tract 95% UCL (0-2')	1.09E-03	2.00E-01	184	6.30E+01	57,821	8.40E+01	77,095
Total TCDD TEQ	site max	5.62E-12	7.00E-10	125	NA	NA	2.00E-08	3,560

The tables above demonstrate that the calculated doses for the respective constituents for an adult industrial exposure scenario are below health protective RfDs, concentrations representing no adverse effect levels (i.e., NOAELs or BMDs), and below concentrations where toxicological effects are reported (i.e., LOAELs or BMDs). As such these concentrations in soil from the property do not represent levels that would be associated with a risk of adverse non-cancer health effects for an adult industrial exposure scenario following direct contact.

9.1.2 Soil cancer risk calculations

As arsenic and dioxins (i.e., 2,3,7,8-TCDD) are classified as human carcinogens, an assessment of theoretical cancer risks was conducted based on the wet-weight and dry-weight arsenic and the total TCDD TEQ concentrations presented in Section 9.1, which included site maximum values, maximum sample location averages, maximum tract averages, or 95%UCL-AMs. The calculated cancer risk includes exposure via inhalation, ingestion, and dermal contact.

Theoretical cancer risks associated with exposure to arsenic/dioxins in soil were calculated as follows:

$$Risk = LADI \times SF$$

where:

Risk = a unitless probability (e.g. 2E-5) of an individual developing cancer over a 70 year lifetime.

LADI = lifetime average daily intake averaged over 70 years (mg/kg-day)

SF = oral and dermal slope factor, expressed in (mg/kg-day)⁻¹

The resulting cancer risks are expressed in scientific notation (e.g., 1.0E-04 to 1.0E-06) and refer to additional lifetime cancer risks of one cancer in 10,000 persons to one cancer in 1,000,000 persons. For example, a calculated theoretical lifetime cancer risk of 1.0E-05 (or 1 in 100,000) indicates that if 100,000 people were exposed to a potentially carcinogenic chemical throughout their 70-year lifetimes, one of the 100,000 individuals would theoretically develop cancer from the lifetime of exposure. The USEPA generally considers total lifetime cancer risks between 1.0E-04 and 1.0E-06 as acceptable for exposures to multiple chemicals with potential carcinogenic effects. Cancer risk calculations for an adult industrial exposure scenario are presented below; exposure parameters and calculations are presented in **Appendix C**.

Table 9.1.7: Summary of cancer risk from soil (wet-weight; adult industrial exposure scenario)

Analyte		EPC (mg/kg)	Cancer Risk
Arsenic	site max	22.9	5.24E-06
	maximum sample location average	18.9	4.31E-06
	maximum tract average	5.1	1.16E-06
	maximum tract 95% UCL (0-2')	8.0	1.82E-06
Total TCDD TEQ	site max	0.0000069	1.91E-07

Table 9.1.8: Summary of cancer risk from soil (dry-weight; adult industrial exposure scenario)

Analyte		EPC (mg/kg-dry)	Cancer Risk
Arsenic	site max	27.3	6.24E-06
	Maximum sample location average	18.5	4.23E-06
	maximum tract average	5.6	1.29E-06
	maximum tract 95% UCL (0-2')	9.0	2.07E-06
Total TCDD TEQ	site max	0.0000094	2.61E-07

As demonstrated above, the calculated cancer risks for adult industrial exposure scenarios for the various wet and dry-weight concentrations fall within or below the USEPA acceptable risk range of 1.0E-04 and 1.0E-06. To put these risks into perspective, a discussion of lifetime cancer probabilities is relevant. Unfortunately, the development of cancer is a major public health problem worldwide and is the second leading cause of death in the United States. The lifetime probability of being diagnosed with invasive cancer is slightly higher for men (39.7% or 0.397) than for women (37.6% or 0.376) (Siegel et al., 2018). Using an adult industrial exposure scenario, which assumes the individual would be exposed to arsenic at the respective concentration for 250 days a year for 25 years, based on the wet-weight 95% UCL-AM for the 0-2' bgs range, would result in an increased theoretical total cancer risk for arsenic of 1.82E-06 (0.000182% or 0.00000182). For the site maximum wet-weight total TCDD TEQ concentration, the increased theoretical total cancer risk is 1.91E-07 (0.0000191% or 0.000000191). These theoretical upper bound calculated cancer risks pale in magnitude compared to the population cancer risk in the United States. Thus, these calculated risks fall within the acceptable risk ranges established by the USEPA and include numerous health-protective uncertainties (i.e., conservative exposure parameters) that are inherent in the risk calculation process. As such, these concentrations do not represent a risk to human health.

In summary, the calculation of a theoretical upper-bound cancer risk for arsenic and the total TCDD TEQ concentrations detected on the property indicated that these risks fall within or below the 1E-04 to 1E-06 range deemed acceptable by the USEPA. It should be noted that an individual would not be expected to

spend their entire time on the property at one location. As such, using a maximum reported value from one sample location would significantly overestimate the risk to an individual present on the property.

10.0 Toxicology of chlorides in soil

Chlorides in soil are essentially non-toxic and do not pose a direct human health risk. Chloride is considered an essential nutrient; it is one of the major minerals in the human body and helps maintain the balance of bodily fluid levels by working in concert with both sodium and potassium. Healthy 19 to 50-year-old adults should consume 1.5 grams of sodium and 2.3 grams of chloride each day (i.e., 3.8 grams of salt) to replace the amount lost daily on average through sweat and to achieve a diet that provides a sufficient amount of essential nutrients (IOM, 2004).

A healthy level of chlorides in the diet is 2.3 grams or 2,300 milligrams. To put this into perspective, a comparison can be made between the recommended dietary intake of chlorides and the level of chlorides one may consume via incidental soil ingestion at the site. The residential incidental soil ingestion rate for a child is 200 mg/day and 100 mg/day for an adult. Assuming the soil on the property is 100% composed of chlorides, which it is not, the average daily incidental ingestion rate of chlorides for an adult would be 23 times lower than the 2,300 mg/day recommended intake of chlorides.

11.0 Comments on the toxicological evaluation report of Dr. William Rogers

I have reviewed the plaintiff's expert report entitled: *"Toxicological Evaluation and Risk Assessment Associated with Oil and Gas Operations on Hero Lands Company, LLC Property Within Stella Oil and Gas Field, Plaquemines Parish, Louisiana"* prepared by Dr. William J. Rogers. My critique of this report is focused on the toxicological evaluation prepared by Dr. Rogers, who opines that *"residual contaminants from [oil and salt water handling operations], including metals, salts, hydrocarbons, dioxin/furan equivalents and radionuclides pose an unacceptable health risk to human and ecological populations"* and that these contaminants have the potential to bioconcentrate to levels which pose *"an unacceptable risk to both human and ecological consumers"*. Additionally, Dr. Rogers opines that the site and/or adjacent property has the potential of being used for residential and recreational purposes. Following review of Dr. Rogers' risk assessment and toxicological evaluation, I have arrived at the following opinions:

Dr. Rogers' opinion that users of the property would be at an "unacceptable health risk" from exposure to site constituents is based on an invalid comparison to various screening values.

Dr. Rogers opines: *"Contamination of site media, particularly surface soil and shallow groundwater has resulted from oil and salt water handling operations. Residual contaminants from those operations, including metals, salts, hydrocarbons, dioxin/furan equivalents and radionuclides pose an unacceptable health risk to human and ecological populations."* Dr. Rogers appears to base his opinions regarding human health on comparisons of constituent concentrations measured on site to various screening standards. As stated by Rogers: *"I have also used accepted soil/sediment ecological screening values as well as screening values recommended and included in the Louisiana Department of Environmental*

Quality's Risk Evaluation and Corrective Action Program (RECAP) remediation standards (RS) and Statewide Order 29B." First and foremost, as mentioned below, and in **Appendix D**, one cannot use screening standards to conclude that exposure to any given environmental constituent will result in an actual health risk. Dr. Rogers is comparing site maximum concentrations to RECAP Standards and simply reporting the fold exceedance of each constituent. This once again is not a scientifically valid comparison and results in creating the appearance of a potential health risk, when in fact there is none.

The remaining exposure pathways (i.e., ingestion of biota from on-site) identified by Dr. Rogers are not considered under the RECAP Screening Option Screening Standards or RECAP MO-1 Standards. The RECAP document states:

The SS do not address the following pathways: inhalation of soil particulates, the inhalation of volatile emissions from soil to an enclosed structure, the inhalation of volatile emissions from groundwater to an enclosed structure, the ingestion of surface water, the inhalation of volatiles from surface water, dermal contact with surface water, the ingestion of sediment, dermal contact with sediment, the inhalation of volatiles from sediment, or the ingestion of biota (recreational or subsistence fishing and/or fish/shellfish propagation or production; meat or dairy production; agricultural crop production). If one or more of these pathways are of concern at an AOC, they shall be addressed under a MO. (LDEQ, 2003b).

and;

The MO-1 RS do not address the following pathways: inhalation of particulates, the ingestion of surface water, the inhalation of volatiles from surface water, dermal contact with surface water, the ingestion of sediment, dermal contact with sediment, the inhalation of volatiles from sediment, or the ingestion of biota (recreational or subsistence fishing and/or fish/shellfish propagation or production; meat or dairy production; agricultural crop production). If any of these pathways are of concern at an AOC, they shall be addressed under MO-2 or MO-3. (LDEQ, 2003b)

Dr. Rogers did not conduct the requisite analysis to address these additional exposure pathways. Thus, the basis for Dr. Rogers' opinion that site constituents represent an "unacceptable health risk" to humans is not scientifically or toxicologically valid and renders his opinion methodologically flawed and unreliable.

Dr. Rogers misuses risk assessment to opine that unacceptable human health risks exist on the property now and in the future.

In this matter, Dr. Rogers has relied on a flawed application of risk-based screening levels to conclude that constituents present at the site "pose an unacceptable health risk to human and ecological populations." Risk assessment is designed to be protective of human health, but not predictive of the actual incidence of disease. The health protective basis of risk assessment is discussed in detail in **Appendix D** of this report.

Risk assessment cannot be used to establish disease causation or determine the actual risk of adverse health effects. Dr. Rogers compares the maximum detected concentration of a given constituent to a regulatory standard and, if it exceeds this value, concludes that the constituent poses “*an unacceptable health risk*” to humans. This is not the intent of risk-based screening guidance.

Risk assessments and risk-based screening standards provide regulatory agencies and risk managers with the ability to make informed decisions on hazardous site cleanup strategies that ensure overall protection of human health and the environment. However, a risk assessment is not intended to predict actual risk of the occurrence of adverse health effects associated with exposure to hazardous substances at a site. Instead, risk assessment methodology provides a health-protective estimate of the maximum risks potentially associated with a site. Regulators realize that uncertainty is inherent in the risk assessment process, and in an effort to ensure that they err on the side of public health, many overestimated exposure assumptions are incorporated into the risk assessment process. This results in health protective guidelines but does not provide an accurate depiction of the true human health risk.

As such, risk assessment cannot be used to predict the incidence of health effects or to state that an actual health risk exists, as Dr. Rogers has done in this case. Therefore, Dr. Rogers’ conclusion that constituents at the site pose “*an unacceptable health risk*” to humans is without proper methodological foundation.

Dr. Rogers does not provide sufficient scientific support for his theory that users of the Hero Lands property or nearby properties are experiencing increased health risks through consumption of wildlife and other biota that are present on the property.

Dr. Rogers states: “*Any contamination that gets into the local food chain has the potential to be ingested by hunters, fisherman, and others who frequent the property and adjacent properties and consume wildlife that are harvested there. The chance of contaminated wildlife ingestion is further increased by the presence of commonly consumed game animals and other forms of wildlife, all of which can be found on and adjacent to the property.*”

Dr. Rogers provides no scientific support for his statements regarding the potential uptake of contaminants through the ingestion of wildlife and biota on the Hero Lands property, nor has he conducted any evaluation to establish that this phenomenon is in fact occurring. There are several key steps in such an evaluation that Dr. Rogers has apparently not conducted. These would include: 1) the measurement of the concentrations of the constituents of interest present in biota allegedly being consumed by users of the property; 2) estimating the amount of fish and/or game consumed by any individual users of the property; 3) estimating the frequency of consumption of fish and game from the property; and 4) estimating the number of years an individual would consume fish and game from the property. To my knowledge, Dr. Rogers has not compiled any of this and other necessary information in order to properly assess this exposure pathway. Thus, there is no evidence to suggest that the consumption of biota from the property represents any health risk to users of the property or other nearby properties.

Dr. Rogers inappropriately classifies Secondary Maximum Contaminant Levels for chlorides as human health regulatory standards.

In Dr. Rogers' expert report, he uses secondary maximum contaminant levels (SMCLs) for chlorides in his comparison of groundwater concentrations to MCLs and RECAP screening standards to conclude these constituents represent a health risk to users of the property. In Dr. Rogers' Toxicological Effects Summary of Sodium/Salts he states: *"Concentrations of chlorides in Site groundwater exceeded the EPA Secondary MCL by a factor of 297X."* Dr. Rogers' misleads the reader by suggesting the exceedance of an SMCL value increases the likelihood that an adverse effect will occur. SMCLs are non-enforceable drinking water guidelines established *"... to assist public water systems in managing their drinking water for aesthetic considerations, such as taste, color and odor."* (USEPA, 2018). SMCLs are not health-based standards, but instead are guidelines to ensure potability and utility of drinking water for domestic purposes. The USEPA has established SMCLs for 15 contaminants including: aluminum, chloride, color, copper, corrosivity, fluoride, foaming agents, iron, manganese, odor, pH, silver, sulfate, TDS, and zinc. As stated in the 1979 USEPA National Secondary Drinking Water Regulations: *"the presence of too great a concentration of chloride ions in drinking water can result in two undesirable effects. First the consumer may detect an objectionable taste in the water. Second, corrosion of the pipes in hot water systems may occur"* (USEPA, 1979). Thus, the SMCLs for chlorides cannot not serve as a basis for Dr. Rogers to claim that these constituents represent a health risk and that at *"higher levels, symptoms of salt toxicity include anxiety and hypersensitivity, followed by loss of coordination and collapse"* and he provides no effort to explain what these levels are or calculate a dose for potential receptors using or consuming this water.

Dr. Rogers claims that exposure to the site poses an "unacceptable health risk to humans;" however, he has not conducted the proper analysis to reach this conclusion.

In the toxicological sciences and in human health risk assessment, the risk of adverse health effects, or the theoretical human health risks are a function of the inherent toxicity of the compound and the dose of the compound. In other words, one cannot determine the risk if one does not know the dose. In this instance, Dr. Rogers has not calculated any dose for any human receptor; therefore, he cannot opine with any reasonable degree of scientific or toxicological certainty that any health risk, whether theoretical or actual, exists. Additionally, much of his exposure is based on pathways that he has insufficient information to quantify, such as ingestion of wildlife and other biota that have been exposed to site media. Thus, without calculating a dose for any actual or theoretical human receptor on the property, Dr. Rogers cannot opine that a health risk exists.

12.0 Conclusions

This report describes an evaluation of the environmental sampling data from the Hero Lands property utilizing risk assessment and toxicology methodologies consistent with LDEQ RECAP and USEPA to determine what, if any, potential human health risks are associated with constituent concentrations measured on the property. Based on this analysis, I have reached the following opinions:

1. Concentrations of constituents measured in soil on the Hero Lands property do not represent a risk to human health. For example, the reported levels of the arsenic, barium, and dioxins measured in soil samples from the property are not harmful to human health and do not present a risk of adverse health effects to current or future users of the property.
2. As there are no active water supply wells used for potable purposes on the Hero Lands property and no registered or known uses of the shallow A, B, or C-zones for potable purposes, the groundwater exposure pathway within the Hero Lands property is deemed incomplete.
3. The most defensible and scientifically accurate method to evaluate the health risks from environmental exposure to petroleum hydrocarbons is the aliphatic and aromatic hydrocarbon fractionation methodology (i.e., the “fractionation method”).
4. Plaintiff’s experts have chosen to use the total petroleum hydrocarbon mixture methodology to assess health risks from petroleum hydrocarbon exposure. There are several limitations associated with TPH-mixture analysis (i.e., TPH-GRO, TPH-DRO, and TPH-ORO) that make this method unreliable for site risk assessment purposes.
5. There are no data indicating the constituent concentrations present on the property have accumulated in biota, or that consumption of biota represents a risk to users of the property.

I declare that the foregoing opinions are true and correct to a reasonable degree of scientific and toxicological certainty.

I reserve the right to amend the opinions in this report should additional information become available.

Respectfully Submitted:



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Appendix D: Risk assessment and toxicology

- D.1.0 Toxicology forms the basis of human health risk assessment D-Error! Bookmark not defined.
- D.2.0 Foundation of human health risk assessment and United States Environmental Protection Agency (USEPA) Risk-Based Corrective Action (RBCA) program..... D-Error! Bookmark not defined.
- D.3.0 Risk assessment process..... D-Error! Bookmark not defined.
- D.4.0 The tiered risk-based decision-making framework..... D-Error! Bookmark not defined.
- D.5.0 RECAP evaluation D-Error! Bookmark not defined.
- D.6.0 Screening standards do not represent clean-up standards ... D-Error! Bookmark not defined.
- D.7.0 Human health basis for RECAP standards D-Error! Bookmark not defined.
- D.8.0 Health protective basis of RECAP soil standards..... D-Error! Bookmark not defined.
- D.9.0 Exposure assumption and equations used in the derivation of RECAP Standards: Protective basis of RECAP soil standards D-Error! Bookmark not defined.
- D.10.0 Intended usage of the regulatory human health risk assessment process..D-Error! Bookmark not defined.



D.1.0 Toxicology forms the basis of human health risk assessment

Toxicology is the science that studies the adverse effects of chemical or physical agents (toxicants) on living organisms. A toxicant is any agent that can produce adverse effects on a biological system. This general definition of a toxicant highlights the fact that exposure to any substance can produce an adverse effect at a high enough dose. This fundamental principle of toxicology was coined by the Swiss philosopher, Paracelsus, who stated:

All substances are poisons; there is none which is not a poison. The right dose differentiates a poison from a remedy. (Klaassen, 2013)

Toxicologists evaluate and attempt to measure or predict the extent of these effects and the intrinsic properties of the toxicants that produce them. The correlation between different dose levels and the effects they may elicit on a biological system is referred to as the dose-response relationship. This relationship is considered the most fundamental concept in toxicology (Klaassen, 2013). Since all substances can be toxic, evaluation of exposure conditions and dose levels (i.e., exposure science) to assess safety is crucial in identifying risk. Thus, an understanding and assessment of human health risks from such substances depends on an understanding of the magnitude and duration of exposure to these substances of concern.

Controlled animal tests are often conducted to evaluate the toxicity of chemicals. This type of testing is common in the field of toxicology, as controlled laboratory studies allow for the evaluation of the potency, and dose-response relationship chemicals have on living organisms. The dose-response relationship accounts for toxicant presence in target tissues at sufficient concentrations and for a sufficient period of time to elicit an adverse effect. Typically, a range of doses is included in a study in order to capture the full range of response, which ranges from the No Observed Adverse Effects Level (NOAEL – the highest dose tested that doesn't elicit an adverse effect), the Lowest Observed Adverse Effects Level (LOAEL – the lowest dose tested that elicits an adverse effect), and a maximal effect or response (MTD – Maximally Tolerated Dose).

D.2.0 Foundation of human health risk assessment and United States Environmental Protection Agency (USEPA) Risk-Based Corrective Action (RBCA) program

In the 1970s, regulatory agencies and public health officials recognized the need to develop and refine the evaluation of toxicological risks (in a quantitative manner) to determine whether chemically-exposed populations are at significant risk of harm (Faustman and Omenn, 2013). Health risk assessment characterizes health risks to humans and ecological receptors from chemical contaminants and other stressors that may be present in the environment (USEPA, 2017e). In the early 1950's, scientists developed methods to determine exposure levels to chemicals in food that were protective of human health, based

on available animal toxicity data. This effort led to the development of Acceptable Daily Intakes (ADI), which took into consideration relative sensitivities within human populations, as well as sensitivities or variability in response between humans and experimental animals. Since ADI values were intended to be protective (rather than predicative) of human health, ADI values were typically set at 1/100^{th5} of the most sensitive chronic animal NOAEL (Rodricks, 2007). USEPA later adopted this approach and applied it to exposure to chemicals in all other environmental media. Toxicologists and epidemiologists were key players in the development of exposure and risk assessment methodologies, which incorporate information regarding the toxic properties of chemicals and contaminants (Federal Judicial Center, 2011). The National Research Council’s Committee on the Institutional Means for Assessment of Health Risk stated that a human health risk assessment should include *“a description of the potential adverse health effects based on an evaluation of results of epidemiologic, clinical, toxicological, and environmental research (hazard identification); extrapolation from those results to predict the type and estimate the extent of health effects in humans under given conditions of exposure (dose–response assessment); judgments regarding the number and characteristics of persons exposed at various intensities and durations (exposure assessment); summary judgments on the existence and overall magnitude of the public-health problem; and characterization of the uncertainties inherent in the process of inferring risk (risk characterization)”* (Federal Judicial Center, 2011).

The first objective of risk assessment is to estimate the probability that an adverse effect will occur if exposure to a chemical occurs at a sufficiently high concentration and at a sufficient exposure duration to elicit that chemical’s toxicity. In general terms, risk can be qualitatively expressed by the following equation, showing that risk is dependent upon both toxicity and exposure:

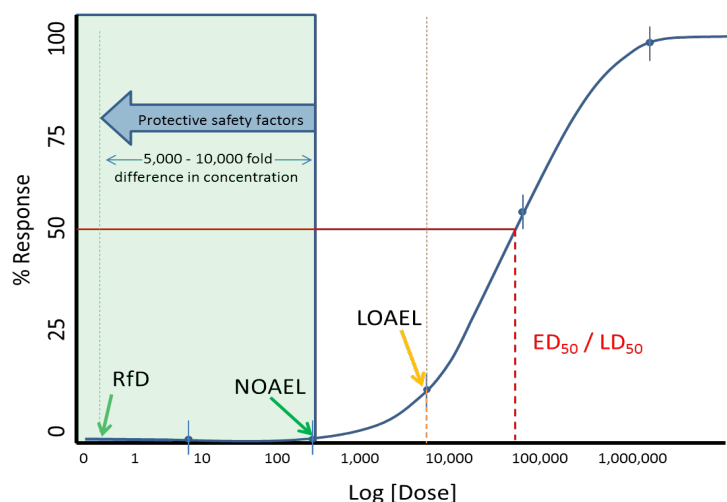
$$\text{Risk} \approx \text{Toxicity} \times \text{Exposure}$$

The second objective of risk assessment is to establish safe exposure levels for humans. Throughout its implementation, refinement of the process of dealing with uncertainties in human toxicity has led to the inclusion of numerical uncertainty factors (UFs) to account for scientific uncertainty and safety factors (SFs) to indicate policy judgments that go beyond scientific uncertainties. The USEPA defined the RfD or Reference Concentration (RfC) as the magnitude of a daily exposure to the human population (including sensitive subgroups), with uncertainty spanning perhaps an order of magnitude, that are likely to be without an appreciable risk of deleterious effects during a lifetime⁶. Often, exceedances of RfD exposures are misinterpreted as *“unacceptable”* or *“unsafe”*. However, RfDs are not bright-line indicators of safe vs. unsafe exposures, and do not necessarily indicate that an adverse health effect will occur. The health-protective nature of the RfD is indicated by UFs that are typically applied to the lowest LOAEL or highest NOAEL observed in controlled laboratory studies, including multiple factors of 10 for extrapolation from sub-acute to chronic exposures, across routes of exposure, animal to human pharmacokinetic and

$\left(\frac{\text{NOAEL}}{(10 \times 10)} \right)$
⁶ USEPA RfD definition: <http://www.epa.gov/iris/rfd.htm>

pharmacodynamic characteristics, possible sensitive human populations, and quality or limitations of the available toxicity database. As such, it is not uncommon to see RfDs that are up to 10,000-fold less than the NOAEL or LOAEL observed in animal studies, often resulting in a very conservative RfD that overestimates true toxic potential. Use of RfDs along with exposure parameters based on a reasonable maximum exposure (RME)⁷ result in conservative health-protective screening levels. Figure D.1 illustrates how RfDs are derived from NOAEL and LOAEL values of a dose-response curve from a controlled laboratory animal study. Responses can be adverse changes in a biological measurement (i.e., % change in body weight gain) or a proportion of affected study subjects within each dose group. In Figure D.1, the dose which elicits an effect of 50% of the maximal response is the Effective Dose 50 (ED50). It is important to understand that at low enough exposures, a threshold can be observed below which a chemical does not produce an observable adverse effect, likely due to an insufficient internal dose of a toxicant to overcome normal physiologic, protective mechanisms (Dybing et al., 2002).

Figure D.1: Health-protective nature of RfDs



D.3.0 Risk assessment process

The risk assessment process is scientific in nature and dependent upon several factors: 1) how much of the chemical is present in an environmental medium (e.g., soil, water, air); 2) how much contact (exposure) an individual or ecological receptor has with the contaminated environmental medium; and 3) the inherent toxicity of the chemical (USEPA, 2017a). In order to conduct a risk assessment, a framework is necessary to evaluate information about chemical substances obtained from scientific studies and site environmental data to estimate a level of risk for people who might be exposed to these substances. Risk assessment is built on the framework of four basic steps as recommended by the National Academy of

⁷ Concept of RME is discussed in the Human-health Basis for RECAP Standards Section 7.0 of Appendix D.

Sciences including: (1) hazard identification, (2) dose-response assessment, (3) exposure analysis, and (4) characterization of risk (NRC, 1983). These basic steps are described below:

- 1. Hazard identification** – Identification of the chemicals suspected to pose a health hazard, quantification of the concentrations of constituents present in the environment, a description of the toxicity that is caused by the constituents, and evaluation of the conditions in which toxicity may be expressed in humans. Information from this step is derived from environmental monitoring data and from epidemiological and animal studies (NRC, 1994).
- 2. Dose-response assessment** – Represents an evaluation in which the inherent toxicity of a constituent may be manifested in an exposed individual, based on a quantitative relationship between the dose of the constituent and human response. The variability between individuals should be noted and assessed as marked variation may exist among humans (e.g., susceptibility between young and elderly people) following similar exposures (NRC, 1994). A key aspect of this paradigm is to determine the potential magnitude of exposure and the probability of adverse effects.
- 3. Exposure assessment** – Determination of the population that may be exposed to the chemical of concern (COC), identifying the routes in which exposure can occur, and estimating the magnitude, duration, and doses that people might receive as a result of their exposure. The goal of exposure assessment is to quantify those amounts and time periods of exposure to a chemical constituent (e.g., the dose). As stated in the Reference Manual on Scientific Evidence (Federal Judicial Center, 2011), *“Ultimately the dose incurred by populations or individuals is the measure needed by health experts to quantify risk of toxicity.”* Thus, in the absence of quantifying a dose, the risk assessment is incomplete, and the risk of toxicity cannot be evaluated. Simply evaluating the potential routes in which a chemical constituent can enter the body does not enable one to quantify risk. A key aspect of this paradigm is determining the extent to which an exposure actually occurs.
- 4. Risk characterization** – This step of the process involves the integration of information from the hazard identification, dose-response assessment, and exposure assessment to estimate the likelihood and magnitude of risk. It should be noted that an appropriate risk characterization should include a full discussion on the relative uncertainties associated with the estimates of risk (GAO, 2001). The result of the risk characterization is *“...a conservative estimate that is likely to overestimate the true risks posed by the site. In reality, the true risk will most likely be much lower than the estimated risk”* (Magaw and Nakles, 2001). Using this paradigm, risk assessments aid regulatory agencies and risk managers in making informed decisions on hazardous site cleanup strategies that ensure overall protection of human health and the environment.

The risk assessment process has generally followed two different paths – one for the assessment of cancer risks and the other for assessment of noncancer risks. For cancer risks, the Linear No Threshold (LNT) model historically assumed that there is no threshold dose below which a carcinogen would confer zero probability of an adverse effect, and that, at low doses, the risk of tumor development decreases linearly

to zero at zero dose. A major factor in support of this model was the ease with which this model could be applied toxicologically. On a cancer dose-response curve, a straight line is drawn from the lowest statistically significant response to the origin. The “steepness”, or slope, of this line represents the carcinogenic potency of the chemical at low exposure levels. The basis of the LNT model was derived from studies suggesting a linear dose response for ionizing radiation-induced genetic mutations. This concept became accepted by radiation geneticists and national/international advisory committees for risk assessment of ionizing radiation-induced mutations/cancer. The US National Academy of Sciences (NAS) Committee on Biological Effects of Atomic Radiation (BEAR 1)/ Genetics Panel believed there was no safe exposure to ionizing radiation for reproductive cells, with an increased risk of mutation from a single ionization event. However, recent discoveries of correspondence between Genetics Panel members suggest that their data and policy decisions may have been based more on funding self-interests than on objective interpretation of the data (Calabrese, 2014). The USEPA Carcinogen Assessment Group (CAG) endorsed the LNT model based on support of epidemiological studies related to ionizing radiation, cigarette smoking, and lung cancer. This model later evolved to a linear multi-stage model to account for toxicological data at high doses. Thus, the emergence of the linear dose-response strategies for current chemical cancer risk assessment practices among federal regulators was based on empirical evidence that radiation-induced cancer had no threshold (Calabrese, 2013; Williams et al., 2000). However, the levels of chemicals in the environment are often too low to produce excess cancer in epidemiological studies. As these epidemiological studies are not always available, cancer risks are often extrapolated from animal cancer bioassays that typically include two doses (and to a lesser extent three or more doses); these doses are often several orders of magnitude higher than doses expected under normal human exposure scenarios with positive tumor formation typically occurring at the highest dose (USEPA, 1998b).

Conversely, noncancer risk estimates have been largely developed under the assumption that a threshold dose does exist below which no adverse effect would be expected (GAO, 2001; Klaassen, 2013; Williams et al., 2000). Noncancer risks are typically presented as a ratio of the estimated exposure to a toxicant and the level at which no adverse effects are expected, such as the RfD or RfC. This ratio is referred to as the Hazard Quotient (HQ). When the HQ is less than 1, no adverse effects are expected as a result of that particular exposure; however, if the HQ is greater than 1, it is possible (but not necessarily likely) that adverse health effects could occur. The HQ value is not a probability that adverse health effects will actually occur, and it is unlikely that the magnitude of the HQ will be proportional to risk⁸. Since calculated RfDs and RfCs can represent values 10,000-fold lower than the observed NOAEL, it is important to emphasize that a HQ exceeding 1 does not necessarily mean that adverse effects will occur.

When there are simultaneous exposures to chemicals that act through similar mechanisms of toxicity, the HQ’s are summed to derive the total hazard index (THI). In assessing noncancer risk, the THI can be expressed as the sum of all hazard quotients for toxicants that affect the same target organ or organ

⁸ Hazard Quotient Definition: <http://www.epa.gov/nata/gloss1.html>

system. Since different toxicants may cause adverse health effects through similar mechanisms, it is reasonable to combine the HQ's that share a toxicity mechanism or affect a common target organ.

Regarding chemical risk assessment there are two directions of assessment used to characterize risk: 1) a "forward" risk assessment which is used to quantify the actual health risks posed by exposure to environmental contaminants and 2) a "reverse" risk assessment which is used to establish allowable or "health-protective" exposure concentrations (e.g., screening standards, maximum contaminant levels; Ofungwu, 2014). In a forward risk assessment, assumptions regarding exposure characteristics, including incidental or accidental ingestion of soil, dermal contact, and body weights, are combined with exposure concentrations to which it is assumed the receptor may be exposed. The dose-response relationships of the contaminants are combined with the exposure concentrations and the estimated exposure characteristics to estimate the potential cancer and/or noncancer health risks that could result from exposure to the contaminants (Ofungwu, 2014).

A reverse risk assessment is used to determine allowable concentrations of a chemical to which exposure can occur. In general, a reverse risk assessment can be qualitatively expressed by the equation:

$$\text{Allowable Exposure} = \text{Acceptable Risk} / \text{Toxicity}$$

To establish a health protective screening level, exposure characteristics are developed using statistical analyses, data on exposed population behavior, and predicted site use. These health protective screening levels often assume a risk of 1×10^{-6} for the development of cancer or a risk hazard quotient of 1.0 for noncancer as acceptable risk levels deemed by the USEPA (1996b). These risk parameters are combined with estimated toxicity of the chemical to back-calculate an allowable exposure concentration (or screening standard) for the contaminant, which is designed to be health-protective in nature (Ofungwu, 2014). Examples of exposure concentrations derived in this manner include USEPA Regional Screening Levels, USEPA Soil Screening Levels, the USEPA drinking water standards and ambient water quality criteria, the Texas Commission on Environmental Quality (TCEQ) Protective Concentration Levels, and LDEQ Screening Option and Management Option Standards.

The process of using exposure concentrations derived by reverse risk assessment to guide cleanup of contaminated sites is referred to as risk-based decision making (RBDM) or RBCA. The initial framework for RBDM was developed by the USEPA in response to requirements set forth in the Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA) of 1980; however, the framework of RBDM has been refined over the years (Magaw and Nakles, 2001).

In the 1980s and 1990s, as the number of underground storage tank (UST) releases increased, regulatory pressure was on UST implementing agencies to derive cleanup requirements for these UST release sites. To address the cleanup of UST sites, corrective action programs were developed with the support of the USEPA to streamline the RBDM processes. In 1995, the USEPA recognized the use of RBDM in UST corrective action programs in an effort to ensure that environmental cleanup efforts are protective of

human health and the environment, and are based on the application of sound science, are flexible, and cost-effective (USEPA, 1995). The incorporation of RBDM into the UST corrective action process has resulted in what is known as RBCA. The ASTM has issued a detailed scientific and technical basis for RBCA that provides a flexible, technically defensible framework for corrective action applicable to a wide range of sites and chemical(s) of concern. The development of this guide was driven by the necessity to cost-effectively and expeditiously manage UST sites. The framework is based on a tiered approach with increasing data collection and analysis integrating site assessment and response actions with human health and ecological risk assessment to determine the need for remedial action and to tailor corrective action to site-specific conditions (ASTM, 2004). The majority of states in the US follow a site evaluation process based on RBCA. As stated by ATSM: *“The USEPA has endorsed the use of risk-based decision-making in underground storage tank (UST) corrective action programs. ASTM, in cooperation with USEPA, is providing training on the RBCA process to over forty-nine state UST agencies. Over thirty states are in the process of implementing a RBCA program”* (ASTM, 2017). The State of Louisiana has adopted a RBCA framework through the promulgation of the Risk Evaluation/Corrective Action Program (RECAP) regulations.

D.4.0 The tiered risk-based decision-making framework

As the use of RBDM process for site management where chemical releases may have occurred can require a substantial investment of technical and financial resources, tiered strategies have been developed as a cost-effective strategy for addressing potential site contamination. Not only does RBDM act as a mechanism for the protection of human health and the environment, but also serves as a framework to efficiently and cost-effectively manage a chemical release site. As stated by the USEPA (1995):

In addition, risk-based decision-making can provide a coherent decision-making framework to help keep transaction costs under control. Thus, while risk-based decision-making can be as protective of human health and the environment as other approaches, it offers a scientifically sound and administratively effective way to respond to the pressures for timely action at large numbers of sites and efficient use of both public and private resources.

RBDM is consistent with USEPA policies and regulations in the cleanup of environmental contamination. As stated by the Office of Solid Waste and Emergency Response Directive (OSWER) 9610.17 (USEPA, 1995):

Risk-based decision-making is a mechanism for identifying necessary and appropriate action throughout the corrective action process. Depending on known or anticipated risks to human health and the environment, appropriate action may include site closure, monitoring and data collection, active or passive remediation, containment, or institutional controls. In all cases, the objective is the same, i.e., to ensure that adequate

protection of human health and the environment is provided. The availability of options such as allowing contamination to remain in place or using institutional controls to prevent exposure will depend on applicable State and local laws and regulations.

This approach is evident within the RECAP. The tiered RBCA process is as follows:

Tier 1 – Chemical concentrations may be compared to generic Tier 1 RBSLs. The RBSLs are chemical-specific concentrations in environmental media that are considered protective of human health. These screening levels are often derived by state or federal regulatory agencies using generic and very conservative exposure assumptions (i.e., over-predictive of likely exposures) using standardized equations combining exposure information assumptions with USEPA toxicity data. The tiered approach begins with this initial state, Tier 1, and uses basic site assessment data and involves a comparison of the concentrations of chemicals in different environmental media to predetermined Tier 1 RBSLs. Site concentrations below the Tier 1 RBSLs do not pose a significant risk to human health or the environment and no remedial action is necessary. If chemical concentrations exceed the Tier 1 RBSLs, the site manager has the option to remediate to a Tier 1 level or, alternatively, progress to conduct further site evaluation under a Tier 2 or Tier 3 analysis (ASTM, 2004; Magaw and Nakles, 2001).

Tiers 2 and 3 –The cleanup goals of a Tier 2 and 3 analyses are generally higher than the Tier 1 analysis as the generic exposure assumptions used in the Tier 1 levels are replaced with more site-specific exposure assumptions and data. Tier 2 and 3 assessments require increasing levels of data collection and analysis. The resulting Tier 2 or 3 cleanup levels are often higher (i.e., allow a higher constituent concentration to remain in place). This is not because they are less protective of human health or the environment; in fact, all three tiers of risk analysis provide an equal level of health protection. The use of more site-specific data and exposure assumptions further refines the likelihood and extent of exposure, and often results in achieving target risk levels at higher environmental media exposure concentrations. Using this tiered approach, a site manager has flexibility to forego a detailed risk characterization of a site-specific Tier 2 or 3 analysis and can proceed directly to more cost-effective actions that may involve meeting conservatively low, generic cleanup goals as in a Tier 1 assessment (ASTM, 2004; Magaw and Nakles, 2001).

The usage of these tiers in the RBCA process aids in establishing cleanup goals and allows the site manager to choose the most effective method of site management. As stated by the USEPA (1995):

Risk-based cleanup goals can be either generic or site-specific. Generic goals based on conservative assumptions about factors that may influence human and environmental exposures can be developed for contaminants generally present at UST release sites. Such generic cleanup goals can be designed to provide adequate protection of human health and the environment in the great majority of corrective action cases. Their use generally will cut down on site-specific data collection and analysis and thus expedite corrective

action. There are sites where it will be more cost-effective to gather site-specific data and set site-specific cleanup goals based on exposure and risk assessment methodology. Where conditions are similar to those used to establish the applicable generic cleanup goals, site-specific goals may not be significantly different, and the costs of the additional data collection and analysis may negate any savings associated with site-specific goals. UST implementing agencies also should consider the administrative costs of negotiating and overseeing the implementation of site-specific goals as they design and develop a risk-based process. EPA believes that a balance can be achieved between the costs and benefits of employing such a process.

Thus, the tiered approach is designed to allow multiple decision points for the responsible party, allowing flexibility to implement RBDM to comply with regulatory mandates and follow a process that ultimately provides protection of human health and the environment. Furthermore, the tiered approach can save resources by helping to determine which areas do not require additional regulatory attention early in a process. It should also be noted (see discussion below) that the RBDM process is designed for site cleanup purposes and is not designed to produce bright-line values above which an actual health risk exists, nor can it be used to predict the actual risk of disease in an individual or a population.

D.5.0 RECAP evaluation

To evaluate the need for remedial action in the state of Louisiana, a standard approach is used to evaluate human health risks to achieve protection of human health and the environment. The LDEQ's RECAP is the state of Louisiana's primary regulation governing remediation activities (LDEQ, 2003). As a general approach, the LDEQ uses a tiered or step-wise method as mentioned above for site evaluation to determine the level of remedial effort. As noted by the LDEQ: "RECAP is consistent with the Environmental Protection Agency's (EPA) guidance on risk assessment" (LDEQ, 2003).

The LDEQ RECAP consists of a tiered framework composed of a Screening Option (SO) and three Management Options (MO-1, MO-2, and MO-3). The Screening Options is comprised of Screening Standards (SS) applied to soil [i.e., Soil Screening Standard non-industrial (residential), Soil_{SSni}; Soil Screening Standard Industrial, Soil_{SSi}; the Soil Screening Standard protective of groundwater, Soil_{SSGW}] and groundwater (i.e., Groundwater Screening Standard, GW_{SS}). For soil and groundwater, the Screening Options is used to:

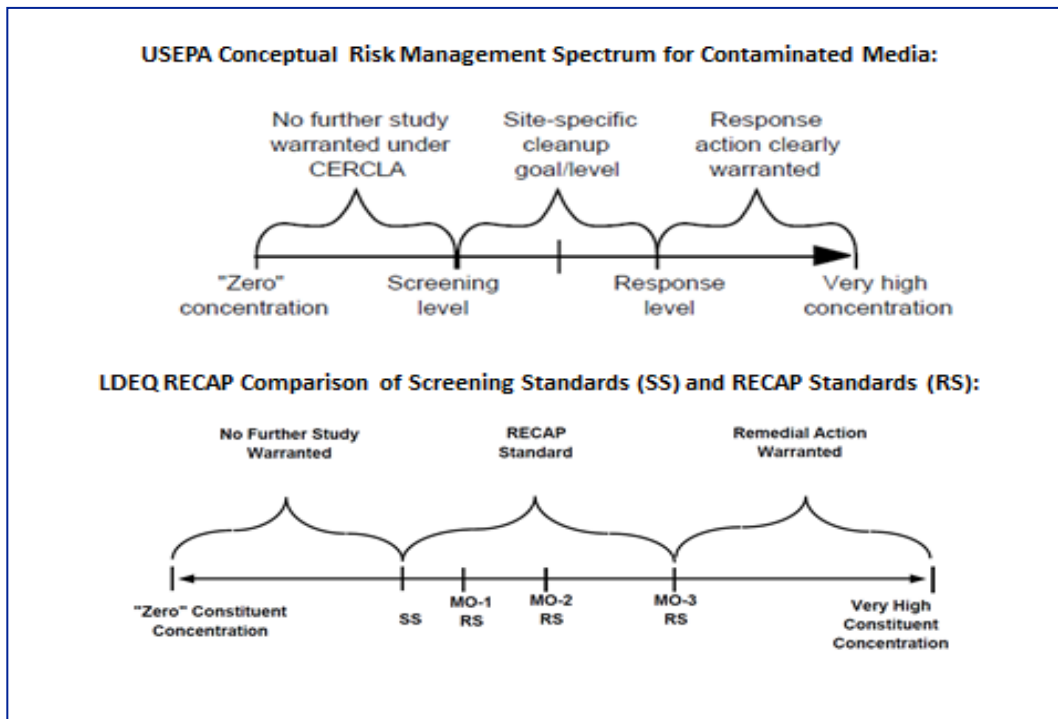
(1) demonstrate that the COC concentration present in soil and/or groundwater does not pose a threat to human health or the environment and hence does not require further action at this time; (2) identify the AOI and the COC for corrective action of soil and/or groundwater under the SO [Screening Option]; or (3) identify the AOI and the COC ... for soil and groundwater for further evaluation under a MO." (LDEQ, 2003)

The tiered approach allows site evaluation and corrective action efforts to be tailored to site-specific conditions (LDEQ, 2003). As stated in the RECAP:

As the Management Option level increases, the approach becomes more site-specific [i.e., requires additional site specific data to evaluate constituent fate and transport] and, hence, the level of effort [and information] required to meet the objectives of the [Management] Option increases (LDEQ, 2003).

The additional information may include further site evaluation, a more extensive exposure assessment, and use of sophisticated fate and transport models. Although the level of effort required for each Option varies, each Option achieves a common goal, which is “protection of human health and the environment” (LDEQ, 2003). Stated another way, the Screening Options and all MOs are designed to achieve the same goal, and no option is “more” safe than the other. This concept is illustrated in Figure D.2 demonstrating the USEPA’s spectrum of contamination which can be encountered at a site of interest and the conceptual range of risk management; the same diagram is adapted by the LDEQ regarding the comparison of Screening Standards to RECAP Standards. As evident by Figure D.2, it is not until an MO-3 RECAP Standard is “exceeded” that remedial action is warranted, confirmatory sampling shall be conducted and closure and/or post-closure requirements shall be met (LDEQ, 2003).

Figure D.2: Comparison of USEPA and LDEQ RECAP conceptual risk management spectrum



Adapted from LDEQ, 2003 and USEPA, 1996a

To further expand on this concept, the Screening Options may be used to screen out areas of a property, media, or COCs⁹ that do not warrant further evaluation as to limit the scope of the RECAP evaluation to those areas/media/constituents of most concern. If the maximum constituent concentration(s) detected in soil and/or groundwater exceed the SS, then: (1) the area shall be managed under the SO; or (2) the area shall be evaluated under MO-1, MO-2, or MO-3. Under the MO-1, the LDEQ provides Department-derived RECAP Standards for soil and groundwater that are protective of human health and the environment (LDEQ, 2003).

Similar to the SS, the *“Management Option 1 may be used to: (1) document that an AOI does not pose a threat to human health or the environment and hence, does not warrant further action at this time; (2) expeditiously manage an AOI defined by the presence of low constituent concentrations and standard exposure conditions; and/or (3) identify areas of a facility, media, or COC that warrant further evaluation so that the scope of the Management Option 2 (MO-2) or Management Option 3 (MO-3) evaluation can be limited to those areas/media/constituents most likely to pose risk. If a constituent-specific soil [area of interest concentration] AOIC or groundwater [compliance concentration] CC exceeds the MO-1 limiting RS [RECAP Standard], then the Submitter may: (1) remediate to the MO-1 limiting RS and comply with closure and/or post-closure requirements for MO-1; or (2) proceed with a MO-2 or MO-3 evaluation. The Submitter may elect to skip the MO-1 and proceed directly to MO-2 or MO-3”* if site specific information is available (LDEQ, 2003).

The MO-2 allows for the development of soil and groundwater RECAP Standard (protective of human health and the environment) based on the use of site-specific data with analytical models to evaluate constituent fate and transport at the AOI. Under an MO-2, the site-specific evaluation is used in conjunction with *“default exposure assumptions and toxicity criteria”* to identify a site-specific MO-2 RECAP Standard. *“If the soil AOIC and groundwater CC for all COC are less than or equal to the site-specific MO-2 limiting RS, then typically, no further action at this time (NFA-ATT) is required for soil or groundwater.”* (LDEQ, 2003) Furthermore, *“if a constituent-specific soil AOIC or groundwater CC exceeds a MO-2 limiting RS, the Submitter may: (1) remediate to the MO-2 limiting RS and comply with closure requirements for MO-2 (and post-closure requirements if warranted); or (2) proceed with a MO-3 evaluation.”* (LDEQ, 2003)

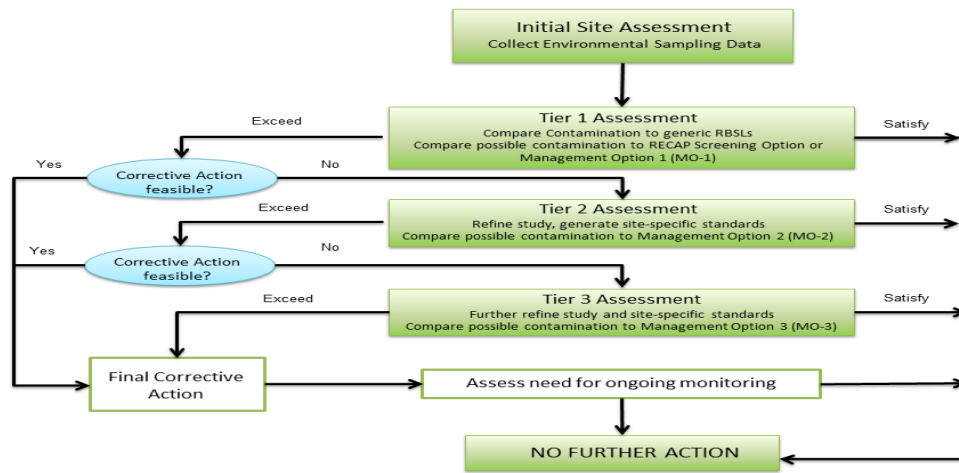
The MO-3 requires a more extensive exposure assessment and usage of sophisticated fate and transport models. The MO-3 allows for the development of site-specific RECAP Standard *“protective of human health and the environment under site specific conditions”* for all impacted media using site-specific exposure and environmental fate and transport data (LDEQ, 2003). Guidance under MO-3 states:

⁹ As defined by LDEQ, 2003: Constituents of concern (COC) - solid waste and hazardous waste, as defined in LAC 33:V.109; industrial solid waste as defined in LAC 33:VII.115; hazardous substance, as defined in La. R.S. 30:2272; regulated substance, as defined in LAC 33:XI.103; pollutant as defined in La. R.S. 30:2004; wastes as defined in La. R.S. 30:2073; and pollutant, priority pollutant, and toxic substances, as defined in LAC 33:IX.107.

If the AOIC and groundwater CC detected at the AOI are less than or equal to the MO-3 limiting RS, then typically, NFA-ATT is required. If a constituent-specific AOIC or groundwater CC for a COC exceeds a MO-3 limiting RS, then: (1) the AOI shall be remediated to the MO-3 RS; (2) confirmatory sampling shall be conducted; and (3) closure and/or post-closure requirements shall be met (LDEQ, 2003).

Figure D.3 corresponds to the tiered framework of the risk-based decision-making process adapted to include the associated tiers used under RECAP.

Figure D.3: Risk-based corrective action flowchart incorporating LDEQ RECAP tiered assessment options



Adapted from Magaw and Nakles, 2001

D.6.0 Screening standards do not represent clean-up standards

For soil, under USEPA guidance, Screening Standards do not represent national clean-up standards [emphasis added] (USEPA, 1996a). The intended usage of a Screening Standards is implied in its very name “Screening;” Screening Standards are used for the purposes of identifying and defining areas, contaminants, and conditions at a particular site that represent levels below which no further attention is required. Soil Screening Standards can be used as Preliminary Remediation Goals (PRGs) which are risk-based values that provide an initial reference point, which may be used for the “establishment” of site specific clean-up levels (USEPA, 1996b). Where contaminant concentrations exceed the Screening Standards, further evaluation and investigation are warranted, but not necessarily remedial activities (USEPA, 1996b). This is further addressed under the USEPA Regional Screening Levels (RSLs) User’s Guide:

*The [USEPA] screening levels (SLs) presented in the Generic Tables are chemical-specific concentrations for individual contaminants in air, drinking water and soil that may warrant further investigation or site cleanup. ... **It should be emphasized that SLs are not***

cleanup standards. *We also do not recommend that the [Regional Screening Levels] RSLs be used as cleanup levels for Superfund Sites until the recommendations in EPA’s Supplemental Guidance to Risk Assessment Guidance for Superfund, Volume I, Part A ... have been addressed. SLs should not be used as cleanup levels for a Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA) site until the other remedy selections identified in the relevant portions of the National Contingency Plan (NCP), 40 CFR Part 300, have been evaluated and considered. (USEPA, 2016; USEPA, 2017d)*

Similar to the usage of Screening Standards under USEPA guidance, the Screening Options presented under LDEQ RECAP provides a rapid screening tool during the early stages of a site investigation through the use of lookup tables with Screening Standards available in the LDEQ RECAP (Table 1 in LDEQ, 2003); furthermore, the Screening Option allows submitters to focus efforts for further assessment. By screening out areas of a site, the COCs of interest, and the exposure pathways for further evaluation, site managers can limit the necessary scope of the remedial investigation or risk assessment. There are also several limitations presented by usage of the SO, including the inability to tailor the assessment to site-specific conditions (i.e., groundwater classification, dilution factors, etc.), and the area of interest concentration is based on the maximum detection constituent concentration, not a measure of the likely upper-bound exposure (i.e., the 95 percent upper confidence limit on the arithmetic mean [95%UCL-AM¹⁰] concentration). Thus, the conservative nature of the Screening Options often leads to a higher tier of assessment (i.e., MO) if the remediating party so chooses. In addition, using this guidance for sites where residential land use assumptions do not apply, results in an over estimation of exposure and overly conservative screening levels (USEPA, 1996a).

In conclusion, RECAP uses risk evaluation to: “(1) determine if corrective action is necessary for the protection of human health and the environment, and (2) identify constituent levels in impacted media that do not pose unacceptable risks to human health or the environment, i.e., RECAP Standards (RS)” (emphasis added) (LDEQ, 2003). Thus, the Screening Options and Management Options are established for the protection of human health and are set at levels well below concentrations at which adverse health effects would be expected to occur.

D.7.0 Human health basis for RECAP standards

The human health basis of each tier (i.e., SO, MO-1, MO-2, and MO-3 RECAP Standards) under RECAP closely follows USEPA guidelines.

For soil and groundwater exposures, the RECAP Screening Options Screening Standards and MO-1 RECAP Standards are based on a number of default assumptions chosen to be protective of human health (i.e.,

¹⁰ *The 95%UCL-AM is the concentration most representative of the concentration that would occur over time as it would not be expected for an individual to spend their entire time at a single sampling location.*

overestimating exposure and potential toxicity to err on the side of public safety). Although the default Screening Options Screening Standards and MO-1 RECAP Standards can be used in place of MO-2 and MO-3 RECAP Standards which are based on the use of site-specific fate and transport data, it is important to note that the site-specific RECAP Standards (i.e., MO-2 and MO-3) provide health protective target risk levels in line with USEPA risk assessment guidance through better defining potential exposures at the property. The assumptions used to calculate RECAP Standards are consistent with the USEPA's Superfund concept of RME based on a non-industrial (residential) and/or industrial setting. The RME is defined as the highest exposure that is reasonably expected to occur at a site (USEPA, 1989), and RECAP applies standard default RME assumptions under the SO, MO-1, and MO-2 scenarios (LDEQ, 2003). In estimating the RME, conservative values for intake and duration of exposure are used (USEPA, 1991b), taking into account exposure via ingestion, dermal, and inhalation pathways. The RME estimate for various exposure pathways includes many conservative and upper-bound parameter values and assumptions (e.g., upper 95th confidence limit on water ingested and upper-bound duration of occupancy of a single residence; (USEPA, 1989)). The USEPA states: *"The intent of the RME is to estimate a conservative exposure case (i.e., well above the average case) that is still within the range of possible exposures"* (USEPA, 1989). The resulting standards represent concentrations of contaminants that are designed to be health protective of exposures in a non-industrial (residential) and/or industrial setting. The RECAP Standards under the MO-1 and MO-2 are also based on default RME exposure assumptions representative of an RME scenario for non-industrial (residential) and/or industrial/commercial land usage. Under the MO-3, site-specific RME assumptions approved by the LDEQ shall be applied for non-industrial (residential) and/or industrial/commercial land uses [in the absence of site-specific exposure data, default RME assumptions shall be used] (LDEQ, 2003). It is important to note that the estimates of the RME use professional judgment, as specific values identified should be regarded as general recommendations, and could change based on site-specific information (USEPA, 1989).

D.8.0 Health protective basis of RECAP soil standards

As stated in LDEQ, 2003, the methodologies and exposure assumptions used for the development of the Screening Standards and the RECAP Standards are consistent with the current USEPA guidelines:

- Risk Assessment Guidance for Superfund, Volume 1 Human Health Evaluation Manual, Part A, RAGS-A (USEPA, 1989);
- Risk Assessment Guidance for Superfund, Volume I Human Health Evaluation Manual, Part B Development of Risk-Based Preliminary Remediation Goals, RAGS-B (USEPA, 1991b);
- Soil Screening Guidance, SSG (USEPA, 1996a; USEPA, 1996b);
- Risk Assessment Guidance for Superfund Volume I: Human Health Evaluation Manual Part E Supplemental Guidance Dermal Risk Assessment Final Version (USEPA, 2004);
- Supplemental Guidance for Developing Soil Screening Levels for Superfund Sites (USEPA, 2001).

It should be noted that the current RECAP guidance was promulgated in 2003; thus, some of the aforementioned USEPA guidelines may have been updated since 2003. Updated USEPA guidance documents were used as resources to supplement previous USEPA guidance and are cited in the Reference Section of this report. Based on the aforementioned USEPA guidelines, the generic non-industrial (residential) $Soil_{SSni}$ and $Soil_{ni}$ standards presented in RECAP are calculated from the same equations used in site-specific methodology but are based on a number of default exposure assumptions. These default assumptions are “chosen to be protective of human health for most site conditions” (USEPA, 1996a). As such, standards based on default assumptions “are expected to be generally more conservative than site-specific levels” [i.e., Screening Options and MO-1 Standards] (USEPA, 1996a). The standardized equations used to calculate these values are designed to address human exposure pathways in a non-industrial (residential) or industrial setting consistent with the concept of RME.

D.9.0 Exposure assumption and equations used in the derivation of RECAP Standards: Protective basis of RECAP soil standards

It should be noted that multiple assumptions are made with regards to the use of risk-based decision-making for site management. As there tends to be limited data available to conduct a risk-based site evaluation, there is generally a need to make basic assumptions to calculate risk. These assumptions may be in regard to the inherent toxicity of the chemical(s) of concern or the duration, frequency, and extent of potential exposures.

As mentioned earlier, the exposure assumptions used to calculate these standards are consistent with the concept of RME. Compared to the average or median exposure, the “high-end” estimate of exposure that is reasonably expected to occur at a site is represented as the RME (USEPA, 2001). The following will address the methods used to calculate screening levels for non-industrial (residential)/industrial exposure pathways, along with the technical basis and limitations associated with their usage. Some of the assumptions used by the USEPA and LDEQ RECAP are presented in Table D.1:

Table D.1: USEPA and LDEQ RECAP Standard default exposure parameters

Definition	Units	Symbol	USEPA Non-Industrial (Residential) Value		USEPA Industrial Value	RECAP Non-Industrial (Residential) Values		RECAP Industrial Value
			Adult ¹	Child ¹	Adult ¹	Adult ²	Child ²	Adult ²
Ingestion Rate, Soil	mg/day	IR _{soil}	100	200	50/100/330*	100	200	50
Soil to skin Adherence	mg/cm ²	AF	0.07	0.2	0.12/0.3**	0.07	0.2	0.2
Skin Surface Area	cm ²	SA	6,032	2,690	3,470	5,700	2,800	3,300
Body Weight	kg	BW	80	15	80	70	15	70
Exposure Frequency	days/yr	EF	350	350	250	350	350	250

Definition	Units	Symbol	USEPA Non-Industrial (Residential) Value		USEPA Industrial Value	RECAP Non-Industrial (Residential) Values		RECAP Industrial Value
			Adult ¹	Child ¹	Adult ¹	Adult ²	Child ²	Adult ²
Exposure Duration	years	ED	20	6	25	24	6	25
Averaging Time (noncarcinogens)	years	AT	20	6	25	24	6	25

¹(USEPA, 2017a); ²(LDEQ, 2003); *Indoor/Outdoor/Construction Worker Soil Ingestion rate; **Worker/Construction Worker Soil Adherence Factor

The LDEQ RECAP provides department-derived Screening Options based Screening Standards and MO-1 RECAP Standards for soil and groundwater for non-industrial (residential) and industrial land use scenarios. As presented in LDEQ (2003), non-industrial land is defined as;

Any property that does not meet the exclusive definition of an industrial property.... Such properties may be residential, farming (livestock or vegetative), or undeveloped lands that are not included in the industrial property description (privately-owned lands, wetlands, state and national parks). Non-industrial sites shall be managed through comparison with non-industrial standards and/or remediated to non-industrial standards.

and,

Residential exposure scenarios and assumptions should be used whenever there are or may be occupied residences on or adjacent to the site. Under this land use, residents are expected to be in frequent, repeated contact with contaminated media. The contamination may be on the site itself or may have migrated from it. The assumptions in this case account for daily exposure over the long term and generally result in the highest potential exposures and risk. (emphasis added) (USEPA, 1991a)

Industrial land is defined as;

...any property not currently used for human habitation on a permanent or temporary/intermittent basis having the following North American Industry Classification System (NAICS) ... major group numbers 11-21; 22 (except 22131); 23-56 inclusive; 61 (except 61111, 61121, 61131); 62 (except 62211, 62221, 62231, 62311, 62322, 62331, 623312, 62399, 62411, and 62441); 71 (except 71219); 72 (except 721191, 721211 and 72131); 81 (except 81411); and 92 (except 92214). Industrial property shall include any block(s) or lot(s) of land controlled by the same owner or operator that are vacant land(s) found within or beside developed land(s). For leased lands, industrial property includes the leasehold and any containers, vessels, tanks, or any other contrivances or units that provide for the management of COC to or from the leasehold. (LDEQ, 2003)

and,

Under this type of land use, workers are exposed to contaminants within a commercial area or industrial site. These scenarios apply to those individuals who work on or near the site. Under this land use, workers are expected to be routinely exposed to contaminated media. Exposure may be lower than that under the residential scenarios, because it is generally assumed that exposure is limited to 8 hours a day for 250 days per year. (USEPA, 1991a)

As previously discussed above, screening levels are developed based on equations using reverse risk assessment methodologies that back-calculate an acceptable contaminant concentration from an acceptable target risk (carcinogen) or HQ (for noncarcinogens) based on conservative, health protective parameters. As an example, the equations for the Soil Screening Standards for the Screening Options and soil RECAP Standards for a MO-1, MO-2, and MO-3 in regard to a non-industrial (residential) or industrial land use scenario for a carcinogenic/noncarcinogenic inorganic constituent are presented below:

Non-industrial (Residential):

Soil_{SSni} or Soil_{ni}– Carcinogenic Effects – Inorganic Constituents (mg/kg):

$$= \frac{TR \times AT_c \times 365 \frac{\text{days}}{\text{yr}}}{EF_{ni} \times \left[\left(\frac{1}{SF_o} \times 10^{-6} \frac{\text{kg}}{\text{mg}} \times IRS_{adj} \right) + \left(SF_o \times 10^{-6} \frac{\text{kg}}{\text{mg}} \times ABS \times IRD_{adj} \right) \right]}$$

Soil_{SSni} or Soil_{ni}– Noncarcinogenic Effects – Inorganic Constituents (mg/kg):

$$= \frac{THQ \times BW_c \times AT_{nc} \times 365 \frac{\text{days}}{\text{yr}}}{EF_{ni} \times ED_c \times \left[\left(\left(\frac{1}{RfD_o} \right) \times 10^{-6} \frac{\text{kg}}{\text{mg}} \times IRS_c \right) + \left(\left(\frac{1}{RfD_o} \right) \times SA_c \times AF_c \times ABS \times 10^{-6} \frac{\text{kg}}{\text{mg}} \right) \right]}$$

Parameter	Definition	Input Value			
		Screening Option (SO)	Management Option 1 (MO-1)	Management Option 2 (MO-2)	Management Option 3 (MO-3)
Soil _{SSni} or Soil _{ni}	Non-industrial (residential) risk-based chemical concentration in soil (mg/kg)	--	--	--	--
TR	Target excess individual lifetime cancer risk (unitless)	10 ⁻⁶	10 ⁻⁶	10 ⁻⁶	10 ⁻⁶
THQ	Target hazard quotient (unitless)	0.1	1	1	1
SF _o	Oral cancer slop factor ((mg/kg-day) ⁻¹)	CS	CS	CS	CS
RfD _o	Oral reference dose (mg/kg-day)	CS	CS	CS	CS

Parameter	Definition	Input Value			
		Screening Option (SO)	Management Option 1 (MO-1)	Management Option 2 (MO-2)	Management Option 3 (MO-3)
BW _c	Average child body weight ages 1-6 (kg)	15	15	15	15
AT _{nc}	Averaging time – noncarcinogens, (yr)	6	6	6	6
AT _c	Averaging time – carcinogens (yr)	70	70	70	70
EF _{ni}	Exposure frequency, non-industrial (residential) (days/yr)	350	350	350	350
ED _c	Child exposure duration ages 1-6 (yr)	6	6	6	6
IRS _{adj}	Age-adjusted non-industrial (residential) soil ingestion rate (mg/kg)	114	114	114	114
IRS _c	Child soil ingestion rate ages 1-6 (mg/day)	200	200	200	200
IRD _{adj}	Age-adjusted dermal contact rate (mg-yr/kg-day)	360	360	360	360
SA _c	Child skin surface area (cm ² /day)	2,800	2,800	2,800	2,800
AF _c	Child soil-to-skin adherence factor (mg/cm ²)	0.2	0.2	0.2	0.2
ABS	Dermal absorption factor (unitless)	CS	CS	CS	CS

CS=Chemical Specific (LDEQ, 2003)

Industrial:

Soil_{ssi} or Soil_i– Carcinogenic Effects – Inorganic Constituents (mg/kg):

$$= \frac{TR \times BW_a \times AT_c \times 365 \frac{\text{days}}{\text{yr}}}{EF_i \times ED_i \times \left[\left(SF_o \times 10^{-6} \frac{\text{kg}}{\text{mg}} \times IRS_i \right) + \left(SF_o \times SA_i \times AF_i \times ABS \times 10^{-6} \frac{\text{kg}}{\text{mg}} \right) \right]}$$

Soil_{ssi} or Soil_i– Noncarcinogenic Effects – Inorganic Constituents (mg/kg):

$$= \frac{THQ \times BW_a \times AT_{ni} \times 365 \frac{\text{days}}{\text{yr}}}{EF_i \times ED_i \times \left[\left(\frac{1}{RfD_o} \times 10^{-6} \frac{\text{kg}}{\text{mg}} \times IRS_i \right) + \left(\frac{1}{RfD_o} \times 10^{-6} \frac{\text{kg}}{\text{mg}} \times SA_i \times AF_i \times ABS \right) \right]}$$

Parameter	Definition	Input Value			
		Screening Option (SO)	Management Option 1 (MO-1)	Management Option 2 (MO-2)	Management Option 3 (MO-3)
Soil _{ssi} or Soil _i	Industrial risk-based chemical concentration in soil (mg/kg)	--	--	--	--
TR	Target excess individual lifetime cancer risk (unitless)	10 ⁻⁶	10 ⁻⁶	10 ⁻⁶	10 ⁻⁶

Parameter	Definition	Input Value			
		Screening Option (SO)	Management Option 1 (MO-1)	Management Option 2 (MO-2)	Management Option 3 (MO-3)
THQ	Target hazard quotient (unitless)	0.1	1	1	1
SF _o	Oral cancer slope factor ((mg/kg-day) ⁻¹)	CS	CS	CS	CS
RfD _o	Oral reference dose (mg/kg-day)	CS	CS	CS	CS
BW _a	Average adult body weight (kg)	70	70	70	70
AT _{ni}	Averaging time – noncarcinogens, industrial (yr)	25	25	25	Site-specific (25)
AT _c	Averaging time – carcinogens (yr)	70	70	70	70
EF _i	Industrial exposure frequency (days/yr)	250	250	250	Site-specific (250)
ED _i	Industrial exposure duration (yr)	25	25	25	Site-specific (25)
IRS _i	Industrial soil ingestion rate (mg/kg)	50	50	50	Site-specific (50)
SA _i	Skin surface area for an industrial worker (cm ² /day)	3,300	3,300	3,300	Site-Specific (3,300)
AF _i	Soil-to-skin adherence factor for an industrial worker (mg/cm ²)	0.2	0.2	0.2	Site-Specific (0.2)
ABS	Dermal absorption factor (unitless)	CS	CS	CS	CS

CS=Chemical Specific (LDEQ, 2003)

The non-industrial (residential) exposure pathway is based on many conservative exposure assumptions. For example, the incidental ingestion of soil is based on an exposure frequency (EF) of 350 days/year for the non-industrial (residential) setting. This value has been argued as being over-conservative even for RME estimates. National travel data were evaluated to determine if an accurate number of “days spent at home” could be reported; however, no conclusions were drawn from literature (USEPA, 1991a). Thus, the common assumption is that the working members of a household take two weeks of vacation per year, which supported a value of 15 days per year spent away from home resulting in a non-industrial (residential) exposure frequency of 350 days/year (USEPA, 1991a). The USEPA recommends using this default value in the absence of site-specific information and states: “this assumption may overestimate EF” (USEPA, 1998a). Furthermore, this upper-bound estimate does not account for time in which individuals may spend at places including work and/or school. For example, these default assumptions would greatly overestimate the risk associated with an individual with full-time employment away from their residence. If one considers a full-time employee works 40 hours a day for 50 weeks a year this equates to 2,000 hours a year, which is the equivalent to approximately 83 days (2,000 hours/year divided

by 24 hours/day) in which the resident is not at their home. In other words, an individual with a full-time job would likely only have an EF of approximately 267 days.

For chronic exposures, the default intake and duration assumptions represent individuals living in a small town or other non-transient community; whereas, exposure to individuals in a transient community are assumed to be shorter; thus, having a lower risk (USEPA, 1996b). In terms of the exposure duration (ED) for adult residents, some default parameters assume an individual to live in the same home for 30 years. In the USEPA Exposure Factors Handbook, this value is presented as the 90th percentile for time spent at one residence (USEPA, 2011). Based on the Monte Carlo method used to simulate residential occupancy periods by Johnson and Capel (1992) if the current age of an individual is 3 years old (for both genders) a residential occupancy period of 22 years represents the 99th percentile. This means that the probability of an individual currently 3 years of age living longer than 22 years at the same location is less than 1%. Furthermore, if we were to use this same Monte Carlo model to represent residential occupancy periods for an individual less than three years old, the 99th percentile for residential occupancy period would be significantly less than 22 years. Thus, it is unlikely that an individual will live at the same residence for a 30-year period.

Conservatism is also built into soil ingestion rates. When characterizing the RME for non-industrial (residential) exposures to soil, the USEPA and the LDEQ RECAP assume upper-bound soil ingestion rates of 200 mg/day for young children (1 to 6 years of ages) and 100 mg/day for older children and adults. These general soil ingestion rates were based on studies conducted prior to 1997 and discussed in EPA's Exposure Factors Handbook (USEPA, 2011); however, more recent studies reported in peer-reviewed literature indicate these daily rates are overestimated (AMEC, 2003). Two studies, published by the authors of the studies upon which the USEPA has based its upper-bound estimates (Calabrese et al., 1989; Stanek and Calabrese, 1995a; Stanek and Calabrese, 1995b) provide the most objective information for use in deriving high-end estimates of daily soil intake. The most recent of the studies, as described by Stanek and Calabrese (2000), has several improvements in study design and analytical procedures, including: a relatively large study group (n=64 children); improved particle size measurements; longer study duration; randomized participants; use of relevant age group and a random sample of the population for that group; and better control for input/output error. The soil ingestion ranges reported by Stanek and Calabrese (2000) for these children were:

- A 95th percentile soil ingestion rate of 106 mg/day (when evaluated over a 365-day period);
- An arithmetic mean soil ingestion rate of 31 mg/day; and
- A median (50th percentile) soil ingestion rate of 17 mg/day.

A study of soil ingestion rates in adults by Stanek et al. (1997) included a number of methodological improvements over the initial study in which adult ingestion rates were based, such as: a larger number of subjects and duration of participation; an improved study design and fecal sampling; improved selection of soil tracers; a broader range of soil ingestion validation; and additional assessments on

particle size of soil ingested. In Stanek et al. (1997) one of the subjects had an unusually high soil ingestion estimate (2 grams) on the first day of the study week. On this day, the subject reported 4 times higher freeze-dried fecal weight than that of any other day, suggesting that his excretion on that day reflected a 304-day accumulation instead of ingestion for one day. As a consequence, the 95th percentile ingestion rate from this study (331 mg/day) driven by the result from this one subject is “*uncertain, unstable, and artificially inflated*” (Calabrese and Baldwin, 2003). Regarding these circumstances Calabrese’s group recommended the use of the upper 75th percentile value, which was 49 mg/day, as the basis for an upper bound soil ingestion rate of 50 mg/day for adults and older children.

The author of the original studies in which the soil ingestion rates are based – Calabrese et al. (1989), Stanek and Calabrese (1995a); Stanek and Calabrese (1995b), and Calabrese et al. (1990) stated: “*I believe that these rates are overstated and can be significantly improved by reliance on newer soil ingestion studies from our group, which used improved methodologies*” (Calabrese and Baldwin, 2003). As stated in AMEC (2003):

Adoption of these more recent data would be consistent with EPA’s Guidelines for Ensuring and Maximizing the Quality, Objectivity, Utility, and Integrity of Information Disseminated by the Environmental Protection Agency (EPA, 2002), which identify information suitable for inclusion in risk assessments as ‘the best available science and supporting studies conducted in accordance with sound and objective scientific practices, including, when available, peer reviewed science and supporting studies.

As numerous studies show that incidental ingestion of soil is common among children 6 years old and younger (Calabrese et al., 1989; Davis et al., 1990; van Wijnen et al., 1990) an age-adjusted soil ingestion factor is used by RECAP, which takes into account the difference in daily soil ingestion rates, body weights, and exposure duration for children from 1 to 6 years of age and others from 7 to 31 years of age. The elevated intake rate of soil by children combined with their lower body weight will lead to a lower (i.e., more conservative) risk-based soil concentration compared to an adult-only assumption.

Thus, the use of conservative exposure parameters leads to screening values based on assumptions that overestimate the likely magnitude of exposure, and result in a subsequent overestimate of the actual risk, if any, for a given exposure scenario. By maximizing these exposure parameters in the exposure scenario, a risk assessor can evaluate the upper bound of potential exposures in a population (if indeed this exposure truly exists in the population). The intended usage of the RME is expressed in the Federal Register (USEPA, 1988) which states:

A legitimate use of worst-case scenarios is to determine if the exposure or risk is low enough even at this extreme so as to dismiss concern for this scenario. It is not legitimate to use a worst-case scenario to prove that there in fact exists a concern in a real population. (emphasis added)

D.10.0 Intended usage of the regulatory human health risk assessment process

The uncertainties associated with a human health risk assessment are well known. Regulatory agencies recognize this uncertainty and adopt assumptions that are conservative (meaning their exposure assumptions and toxicity constants ensure an overestimate of the true risk) to determine “human-health protective” exposure estimates - not to predict actual exposure outcomes. These precautionary assumptions are common for initial screening assessments (i.e., use of Screening Standards), when the primary goal is to determine if the presence of a constituent represents a potential health risk and if further evaluation is necessary. In other words:

...the focus of federal agencies’ “risk” assessments can sometimes be characterized more accurately as safety assessment [i.e., estimating an exposure level below which no significant risk will occur] rather than as risk assessment [i.e., simply describing the likelihood of a risk]. (GAO, 2001)

The goal of the regulatory risk assessment scheme is to not underestimate the actual risk, and so conservative assumptions are chosen when uncertainties exist in the data that are incorporated into the risk assessment. This conservative (i.e., health-protective) approach related to assumptions of exposure, dose, and toxicity support the regulatory scheme because regulators are looking for calculations that will help ensure they have developed exposure guidelines that are protective of human health. As stated in Federal Judicial Center (2011):

Risk assessment is not an exact science. It should be viewed as a useful framework to organize and synthesize information and to provide estimates on which policymaking can be based. In recent years, codification of the methodology used to assess risk has increased confidence that the process can be reasonably free of bias; however, significant controversy remains, particularly when actual data are limited and generally conservative default assumptions are used.

and,

...an example of conservative default assumptions can be found in Superfund risk assessment. EPA has determined that Superfund sites should be cleaned up to reduce cancer risk from 1 in 10,000 to 1 in 1,000,000. A number of assumptions can go into this calculation, including conservative assumptions about intake, exposure frequency and duration, and cancer-potency factors for the chemicals at the site.

A risk assessment provides a health protective estimation of the maximum risks potentially associated with a site but does not provide an accurate depiction of the true human health risk or predict actual health effects that hazardous substances at a site may have on an individual. It is clear that regulatory agencies recognize the overestimation of the risk assessment process:

It should be emphasized that the linearized multistage procedure leads to a plausible upper limit to the risk that is consistent with some proposed mechanisms of carcinogenesis. Such an estimate, however, does not necessarily give a realistic prediction of the risk. The true value of risk is unknown, and may be as low as zero. The range of risks, defined by the upper limit given by the chosen model and the lower limit which may be stated as low as zero, should be explicitly state. (emphasis added) (USEPA, 1986)

The risk assessment process is protective of human health on many levels. For example, the USEPA publishes toxicity values for hundreds of chemicals that serve as the toxicological underpinnings of regulatory human health risk assessments. As previously discussed, these toxicity values are intended to protect even the most sensitive individuals in the general population and are derived by dividing the no-observed-adverse effect levels¹¹ (NOAEL) or lowest-observed-adverse-effect level¹² (LOAEL) in the most sensitive laboratory animal species by “uncertainty factors” designed to account for differences in responses between animals and humans and other sources of uncertainty in the model. Therefore, these toxicity constants are health protective in nature and by design are likely to overestimate the hazard posed by a given dose of a chemical. Similar health protection is built into the calculations used to derive cancer slope factors in the USEPA Integrated Risk Information System (IRIS) database (USEPA, 2017a). The USEPA IRIS database provides information to help evaluate human risk from exposure to environmental contaminants. As stated in the USEPA IRIS preamble, IRIS values cannot be used to provide accurate predictions of actual human health risks:

In general IRIS values cannot be validly used to accurately predict the incidence of human disease or the type of effects that chemical exposures have on humans. This is due to the numerous uncertainties involved in risk assessment, including those associated with extrapolations from animal data to humans and from high experimental doses to lower environmental exposures. The organs affected and the type of adverse effect resulting from chemical exposure may differ between study animals and humans. In addition, many factors besides exposures to a chemical influence the occurrence and extent of human disease. (emphasis added) (USEPA, 2012)

Therefore, as summarized by the USEPA, IRIS toxicity values are intended to protect the public from adverse health risks rather than predict actual human disease resulting from chemical exposure; this typically overestimates the true risk of the exposure.

Furthermore, from the former USEPA risk assessment scientists,

¹¹A no-observed-adverse effect levels (NOAEL) is an experimentally determined dose at which there was no statistically or biologically significant indication of the toxic effect of concern (USEPA, 2017c)

¹²A lowest-observed-adverse-effect-level (LOAEL) is the lowest exposure level at which there are biologically significant increases in frequency or severity of adverse effects between the exposed population and its appropriate control group (USEPA, 2017b).

Most risk estimates are calculated to be protective of human health, rather than predictive of actual toxicity. For example, cancer potency factors calculated by the U.S. EPA are presented as the 95% upper confidence limit on the dose-response curve, rather than the maximum likelihood estimate. EPA goes on to say that risk assessors believe the actual cancer risk to be somewhere below this upper confidence limit, and that it would be as low as zero. This is an acknowledgement of the uncertainty inherent in the process of cancer risk assessment, which is a function of both cross-species and high-dose extrapolation. (*emphasis added*) (Felter and Dourson, 1998)

The intended goal of the regulatory process was not to develop true estimates of human risk, but rather to identify risk levels below which no adverse health effects are expected to occur, for reasons of health protection and safety. The resulting risk estimates are therefore valid for only one purpose: that of excluding risks that are too small to be of potential health concern. In other words, it is illogical to apply a methodology that over-predicts risk as a basis for determining whether the perceived risk from an exposure will induce a specific effect. This is because one knows the true risk is lower and may still be insufficient to produce the effect. On the other hand, if a methodology over-predicts risk, this method can be useful in eliminating the potential for adverse effects because if the perceived risk is too low to support concern for harm, the inherent overestimation of the risk assessment process makes these low risks even less likely to be an issue than calculated. Such risks can therefore be dismissed. This feature of regulatory risk assessments (i.e., their conservative, health protective nature) has been pointed out in published literature. For example:

...this approach to risk characterization is highly simplistic and ought to be confined to a screening-level type of analysis. Assuming that all carcinogens and non-carcinogens have additive effects implies that all compounds have the same target organs or mechanisms of action, that the interactions between compounds at trace levels are, in fact, additive and that all effects are of equal severity. If the results of a typically conservative analysis are below the de minimis risk level of 1×10^{-6} (cancer) or 1.0 (non-cancer), one can be reasonably certain that no actual health risks exist. Risk estimates above the de minimis risk levels, however, cannot be taken of evidence of a risk because of the conservative assumptions inherent in most risk assessments. Providing these point estimates as the sole focus of the risk characterization creates a false impression of precision without an understanding of the uncertainty inherent in the process. These risk estimates should never be treated or presented as predictions of future happenings or medical diagnoses, as often happens, but rather as decision –making tools for regulatory purposes. (Gargas et al., 1999)

The health protective nature of risk assessment is often misunderstood, and it is mistakenly believed that risk assessment is predictive of a “true” human health risk. This misunderstanding is often exhibited by those with limited understanding of the “health-protective” methodologies, which form the basis of

human health risk assessment. Thus, the risk assessment process results in the formulation of health protective guidelines but is not predictive of risks. Unfortunately, this distinction is often lost on the public and on many of those who communicate risks to the public. As Felter and Dourson (1998) note:

Somewhere between the steps of risk assessment and risk management, however, the concept of risk estimates as inherently imprecise has been lost. This is probably due to a number of reasons, one of which is likely because the risk manager has to communicate with a public that wants to know with some certainty and precision what the risks from exposure to hazardous substances actually are (and in rather succinct terms), rather than hearing the risks described more appropriately as scientific judgments that are, by their very nature, imprecise.

The result is that these risks are often viewed as true health risks. This concern has been voiced by toxicologists and risk assessors:

...the government gives mixed messages through its use of predicted worst-case risks. For example, both the U.S. Environmental Protection Agency (USEPA) and National Academy of Sciences (NAS), through their publications of worst-case risk numbers for pesticide residues in food, leave the strong impression that the public can expect along the order of thousands of deaths per year from consuming current levels of regulated pesticide residues in and on food. There is no valid scientific support whatever for such an implication. (Scheuplein, 1992)

and,

Despite the fact that the stated definition of EPA's RfD and RfC¹³ includes a statement that the risk value has "uncertainty spanning perhaps an order of magnitude," or that the cancer potency factors are 95% upper confidence limits based on one statistical model, risk management decisions are often made as if these risk estimates are precise point estimates. (Felter and Dourson, 1998)

Furthermore, because risks are exaggerated to be health-protective in nature, risk assessment cannot be used to establish a causal effect; however, it is useful to effectively rule out the possibility of health risks associated with a given exposure. As noted by the California Environmental Protection Agency (2001):

¹³Reference concentration (RfC) - The reference concentration is an estimate (with uncertainty spanning perhaps an order of magnitude) of a continuous inhalation exposure to the human population (including sensitive subgroups that include children, asthmatics, and the elderly) that is likely to be without an appreciable risk of deleterious effects during a lifetime. It can be derived from various types of human or animal data, with uncertainty factors generally applied to reflect limitations of the data used (USEPA, 2017)

People sometimes think that a risk assessment will tell them whether a current health problem or symptom was caused by exposure to a chemical. This is not the case.

Thus, due to its health protective nature, whereby risks are exaggerated to provide increased margins of safety, risk assessment cannot be used to predict the incidence of health effects. However, since risk assessment provides upper bound risk estimates, it is particularly useful to effectively rule out the possibility of health risks associated with a given exposure. Thus, the methodology and guidance used by the USEPA provide a scientific foundation for the determination of health-protective constituent levels in the environmental media and provide a consistent basis for site assessment and risk-based decision-making regarding the corrective action process.

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Appendix E: Toxicological basis for aliphatic and aromatic hydrocarbon fractionations and TPH-mixtures

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E.1.0 RECAP TPH fraction-specific and indicator approach

The Louisiana Department of Environmental Quality (LDEQ) Risk Evaluation/Corrective Action Program (RECAP) recommends the TPH Fraction and Indicator approach developed by the Total Petroleum Hydrocarbon Criteria Working Group (TPHCWG) to assess petroleum impacted groundwater and soil. This approach is based on the assessment of both individual petroleum constituents identified as “indicators” and aliphatic and aromatic hydrocarbon fractions characterized by fraction-specific toxicity data.

The RECAP guidelines for assessing petroleum impacted environments utilize oral reference doses (RfD_o) and inhalation reference doses (RfD_i) determined by reference values developed by the TPHCWG. The reference dose is an estimate of a daily exposure level that is likely to be without an appreciable risk of deleterious effects during a lifetime (LDEQ, 2003b). These reference values are based on the best available toxicity data for the individual compounds and mixtures which best represent the composition of each fraction.

The RfD_o and RfD_i for each fraction as defined by the RECAP guidelines and the TPHCWG are compared in Table E.1 below.

Table E.1 Comparison of RECAP and TPHCWG reference doses

Analyte	RECAP				TPHCWG		
	RfD _o mg/kg-day	Reference	RfD _i mg/kg-day ¹	Reference	RfD _o mg/kg-day	RfC mg/m ³	RfD _i mg/kg-day ¹
Aliphatics C5-C6 ²	NA	NA	NA	NA	5.00	18.40	5.3
Aliphatics >C6-C8	5.00	TPHCWG	5.3	TPHCWG	5.00	18.40	5.3
Aliphatics >C8-C10	0.10	TPHCWG	0.30	TPHCWG	0.10	1.00	0.30
Aliphatics >C10-C12	0.10	TPHCWG	0.30	TPHCWG	0.10	1.00	0.30
Aliphatics >C12-C16	0.10	TPHCWG	0.30	TPHCWG	0.10	1.00	0.30
Aliphatics >C16-C35	2.00	TPHCWG	2.00	*	2.00	NA	NA
Aromatics >C7-C8 ^{2,3}	NA	NA	NA	NA	0.20	0.40	0.11
Aromatics >C8-C10	0.04	TPHCWG	0.06	TPHCWG	0.04	0.20	0.06
Aromatics >C10-C12	0.04	TPHCWG	0.06	TPHCWG	0.04	0.20	0.06
Aromatics >C12-C16	0.04	TPHCWG	0.06	TPHCWG	0.04	0.20	0.06
Aromatics >C16-C21	0.03	TPHCWG	0.03	*	0.03	NA	NA
Aromatics >C21-C35	0.03	TPHCWG	0.03	*	0.03	NA	NA
TPH-GRO (C6-C10) ⁴	0.04	RECAP	0.06	RECAP			
TPH-DRO (C10-C28) ⁴	0.03	RECAP	0.06	RECAP			
TPH-ORO (>C28) ⁴	0.03	RECAP	NA	*			

NA – Not available. *No inhalation toxicity available; oral toxicity used to assess inhalation exposure (LDEQ, 2003b). ¹To compare to the RfDi values listed in RECAP Appendix D (LDEQ, 2003b), the conversion of TPHCWG RfC values to mg/kg-day was calculated by multiplying the RfC in mg/m³ by the inhalation rate of an adult (ages 7-31; 20 mg³/day) divided by the average body weight of an adult (ages 7-31; 70 kg) (USEPA, 2011). ²Reference values for aliphatics C5-C6 and aromatics >C7-C8 are included in TPHCWG Volume IV. However, RECAP guidelines evaluate toluene, xylene, and ethylbenzene in lieu of the aromatic fractions >C5-C7 and >C7-C8 and do not include the aliphatic fraction C5-C6 (LDEQ, 2003b; TPHCWG, 1996). ³Table 1 of TPHCWG Volume IV lists 0.4 mg/m³ (0.11 mg/kg-day) as the RfDi for the aromatic fraction >C7-C8, which is based on the RfDi for toluene provided by the USEPA IRIS. In Section IV.A.2 of TPHCWG Volume IV, the recommended RfDi is 1 mg/m³ (0.29 mg/kg-day), which is stated as being "protective for the entire range of compounds within this fraction." As the more conservative value, 0.4 mg/m³ will be used as the RfC for this fraction. ⁴The RfD values for TPH-GRO, TPH-DRO, and TPH-ORO were determined by the aliphatic or aromatic fraction with the most conservative (i.e., health-protective) RfD within each division. TPH-GRO is represented by the RfD values for aromatics >C8-C10. TPH-DRO is represented by the RfD values for aromatics >C16-21 and the RfDi for aromatics >C10-C16. TPH-ORO is represented by the RfD values for aromatics >C21-C25. (LDEQ, 2003b).

Based on information provided in Table E.1, the RECAP Screening Standards and RECAP Standards for the TPH-mixtures (i.e., TPH-GRO, TPH-DRO, and TPH-ORO) are calculated based on the aliphatic or aromatic hydrocarbon fraction with the most conservative (i.e., lowest) RfD, which is then used to calculate a Screening Standard or RECAP Standard for the entire TPH-mixture. Thus, TPH-GRO is represented by the RfD for aromatics >C8-C10. TPH-DRO is represented by the RfD_o for aromatics >C16-21 and the RfD_i for aromatics >C10-C16. TPH-ORO is represented by the RfD_o for aromatics >C21-C25 (LDEQ, 2003b). Thus, the use of TPH-mixture analysis becomes problematic as they are based on toxicological data established for specific aromatic hydrocarbon fractions that may not even be present in the sample analyzed.

E.2.0 Basis of aliphatic and aromatic hydrocarbon fraction specific RfDs

The fraction-specific RfDs were developed by assessing the available toxicity data for the individual compounds and mixtures present in each hydrocarbon fraction. As outlined in TPHCWG Volume IV, this was accomplished by deriving a reference value from the no-observed-adverse effect levels¹⁴ (NOAEL) or lowest-observed-adverse-effect level¹⁵ (LOAEL), uncertainty factors (UF), and modifying factors (MF). NOAELs and LOAELs are determined using available critical studies of chronic oral exposure and chronic inhalation exposure. The oral NOAELs and LOAELs for the aliphatic and petroleum hydrocarbon fractions are provided in Table E.2 below.

Table E.2 Aliphatic and aromatic hydrocarbon fraction specific oral NOAELs and LOAELs*

Analyte	NOAEL (mg/kg-d)	LOAEL (mg/kg-d)
Aliphatic Hydrocarbon Fractions		
Aliphatics C5-C6	NA	NA
Aliphatics >C6-C8	NA	NA
Aliphatics >C8-C10	100	500
Aliphatics >C10-C12	100	500
Aliphatics >C12-C16	100	500
Aliphatics >C16-C35	200	2,000
Aromatic Hydrocarbon Fractions		
Aromatics >C7-C8	97.7	291
Aromatics >C8-C10	300	300
Aromatics >C10-C12	300	300
Aromatics >C12-C16	300	300
Aromatics >C16-C21	75	125
Aromatics >C21-C35	75	125

* (TPHCWG, 1996); NA: Not available.

¹⁴A no-observed-adverse effect levels (NOAEL) is an experimentally determined dose at which there was no statistically or biologically significant indication of the toxic effect of concern (USEPA, 2017c).

¹⁵A lowest-observed-adverse-effect-level (LOAEL) is the lowest exposure level at which there are biologically significant increases in frequency or severity of adverse effects between the exposed population and its appropriate control group (USEPA, 2017a).

Where chronic oral and inhalation exposure studies were unavailable, the available studies were modified with the application of uncertainty factors to determine a reference value.

As defined by the USEPA (2017d), uncertainty factors are:

One of several, generally 10-fold, default factors used in operationally deriving the RfD and RfC from experimental data. The factors are intended to account for (1) variation in susceptibility among the members of the human population (i.e., inter-individual or intraspecies variability); (2) uncertainty in extrapolating animal data to humans (i.e., interspecies uncertainty); (3) uncertainty in extrapolating from data obtained in a study with less-than-lifetime exposure (i.e., extrapolating from subchronic to chronic exposure); (4) uncertainty in extrapolating from a LOAEL rather than from a NOAEL; and (5) uncertainty associated with extrapolation when the database is incomplete.

Modifying factors are defined by the USEPA (2017b) as:

A factor used in the derivation of a reference dose or reference concentration. The magnitude of the MF reflects the scientific uncertainties of the study and database not explicitly treated with standard uncertainty factors (e.g., the completeness of the overall database). A MF is greater than zero and less than or equal to 10, and the default value for the MF is 1.

RfD_o values are calculated using the following equation:

$$\text{RfD}_o = \text{NOAEL}(\text{or LOAEL})/(\text{UF} \times \text{MF})$$

RfD_i values are calculated by first adjusting the available NOAEL (or LOAEL) for continuous exposure and dosimetric differences across species. Thus, RfD_i values are calculated using the following series of equations:

- (1) **NOAEL_{ADJ} = E x D (hour/24 hours) x W (days/7 days)**; in which NOAEL_{ADJ} is the NOAEL adjusted for continuous exposure, E is the exposure level, D is the number of hours exposed, and W is the number of days of exposure.
- (2) **NOAEL_{HEC} = NOAEL_{ADJ} x [(H_{b/g})_A/(H_{b/g})_H]**; in which NOAEL_{HEC} (human equivalent NOAEL) is the NOAEL adjusted for dosimetric differences across species to a human equivalence concentration and H_{b/g})_A/(H_{b/g})_H is the ratio of the blood:gas partition coefficient of the animal in the study to the blood:gas partition coefficient for humans. If these values are unknown, the default value for H_{b/g})_A/(H_{b/g})_H is 1.
- (3) **RfC = NOAEL_{HEC} (or LOAEL)/(UF x MF)**; in which RfC is expressed in mg/m³.
- (4) **RfD_i = RfC x IRA_a/(BW_a)**; in which the RfD_i is expressed in mg/kg-day, BW_a is the assumed body weight of an adult (70 kg), and IRA_a is the assumed inhalation rate of an adult (20 m³/day) as defined by the USEPA Exposure Factors Handbook.

Under LDEQ RECAP guidance, these health protective RfD_o and RfD_i values are then used for the calculation of soil and groundwater Screening Standards and RECAP Standards as presented in Appendix H of RECAP (LDEQ, 2003b).

E.3.0 Toxicological basis for aliphatic and aromatic hydrocarbon fraction-specific reference values

The following sections discuss the development of RfD_o and RfD_i reference values for the aliphatic and aromatic hydrocarbon fractions as outlined in TPHCWG Volume IV. TPHCWG presents these values as RfD (oral reference dose; mg/kg-day) and RfC (inhalation reference concentration; mg/m³). To compare to the RfD_i values listed in RECAP Appendix D (LDEQ, 2003a), the conversion of TPHCWG RfC values (mg/m³) to RfD_i (mg/kg-day) is calculated by multiplying the RfC by the inhalation rate of an adult (ages 7-31; 20 m³/day) divided by the average body weight of an adult (ages 7-31; 70 kg) (USEPA, 2011).

Aliphatics C5-C6 and >C6-C8

At the time of publication of TPHCWG Volume IV, n-hexane was the only compound in the C5-C8 aliphatic fractions with an RfC developed by the USEPA. In 1990, the RfC for n-hexane in the IRIS database was published as 0.2 mg/m³, which was developed based on a neurotoxicity study in humans. Since then, the USEPA published a new RfC on the IRIS database of 0.7 mg/m³, which was developed based on a subchronic rat inhalation study. This change in critical study was due to the identification of new literature and more recent data regarding potential co-exposure in the human neurotoxicity study. As this information was published after the publication of the TPHCWG guidance, all calculations and considerations regarding n-hexane in the development of recommended reference values by the TPHCWG were performed using the more conservative RfC of 0.2 mg/m³.

The USEPA has not defined an oral RfD for n-hexane due to a lack of appropriate data. As such, the TPHCWG calculated an RfD for n-hexane of 0.06 mg/kg-day using the RfC value available at the time of publication, an assumed body weight of 70 kg, an assumed inhalation rate of 20 m³/day, and 100% absorption (USEPA, 2011).

Because n-hexane has a unique toxicity and is present at relatively low levels in petroleum products (0.05-7.0% in gasoline, 0.7-1.8% in crude oil, and 0.06-15.7% in naphtha from petroleum refinery streams), the TPHCWG determined that using n-hexane RfD values for the entire fraction overestimated the potential health risks of the other compounds. Thus, the TPHCWG used datasets from n-heptane, a compound structurally similar to n-hexane, and commercial hexane, a solvent mixture containing hexane isomers, to develop recommended RfD values.

Pharmacokinetic studies to quantitate the neurotoxic risk of n-heptane to n-hexane have suggested that the neurotoxic risk of n-heptane is at least 38-times lower than n-hexane. Assuming an RfD for n-hexane of 0.06 mg/kg-day, the TPHCWG calculated a RfD of 2 mg/kg-day for n-heptane, which is 38 times higher than the value for n-hexane. Other C5-C8 compounds have not been shown to cause neurotoxicity, meaning that n-heptane can be considered an appropriate surrogate for the C5-C8 fraction (excluding n-hexane, which has separate RfD values).

Commercial hexane, a solvent containing approximately 53% n-hexane, has been studied under Section 4 of the Toxic Substance Control Act. Based on two chronic bioassay studies for commercial hexane, the TPHCWG calculated an RfC of 18.4 mg/m³, which can then be used to calculate an RfD of 5 mg/kg-day.

At the time of publication, data on cyclohexane had not yet been published for the calculation of reference values, though the TPHCWG suggested that the data may impact the reference values for this fraction and should be examined upon release. Since the publication of the TPHCWG guidelines, the USEPA has published a RfC value of 6 mg/m³ on the IRIS database. Using the same calculations as above, this can be used to estimate an RfD of 1.7 mg/kg-day.

Based on the available RfD information, the composition of petroleum products in this carbon range, and the level of conservatism inherent in RfD development, the TPHCWG determined that the RfD value of 5 mg/kg-day and RfC value of 18.4 mg/m³ (converted to RfD_i of 5.3 mg/kg-day) developed for commercial hexane are appropriate for all situations regarding this carbon fraction with the exception of circumstances involving high purity n-hexane, which would be detectable using analytical methodology. This determination does not consider the RfD values of cyclohexane, which were published after the TPHCWG recommendations.

Aliphatics >C8-C10, >C10-C12, and >C12-C16

Minimal toxicity data is available on individual components within the C9-C16 aliphatic ranges. As such, toxicity studies on Jet Propellant 8 (JP-8; C9-C16) and dearomatized petroleum streams were used to develop RfD values for these fractions. The determination of reference values for these fractions is weighted in favor of data from the dearomatized petroleum streams, as petroleum streams have 0.1-1.5% aromatic content compared to up to 20% aromatic content in JP-8.

RfD

Multiple oral studies on dearomatized petroleum streams resulted in calculated RfD values of 0.1 mg/kg-day. The oral study for JP-8 resulted in a calculated RfD of 0.75 mg/kg-day. As 0.1 mg/kg-day is a more conservative estimate, the TPHCWG determined that this RfD is considered representative of this fraction and is protective of systemic toxicity and developmental/reproductive endpoints.

RfC

Various inhalation studies (both subchronic and developmental) for dearomatized petroleum streams and JP-8 resulted in calculated RfC values of 0.9-1.0 mg/m³. The TPHCWG determined that a RfC of 1.0 mg/m³, which is converted to an RfD_i of 0.3 mg/kg-day, is considered representative of this fraction and is protective of systemic toxicity and developmental/reproductive endpoints.

Aliphatics >C16-C21 and >C21-C35

The TPHCWG recommended that reference values for this fraction be developed using toxicity data for white mineral oil, which is a complex of highly refined aliphatic mineral hydrocarbons with virtually no aromatic components or other contaminants. Reference values for white mineral oils are developed using the results of a rat toxicity study conducted by the British Industrial Biological Research Association (BIBRA).

RfD

The toxicity study on white mineral oils indicated that results appear to be related to molecular weight. As such, the TPHCWG developed two RfD values: 2 mg/kg-day for TPH fractions containing aliphatic fractions C17-C34 based on the NOAEL for low molecular weight mineral oils, and 20 mg/kg-day for TPH fractions containing aliphatic fractions >C34 based on the NOAEL for high molecular weight mineral oils. LDEQ RECAP uses an RfD of 2 mg/kg-day, the more conservative of the two RfD values for white mineral oil.

RfC

The TPHCWG did not suggest RfC values for TPH fractions >C16-C35; these heavier compounds have low volatilization potential, and inhalation is not likely to be a significant method of exposure.

Aromatics C5-C8

RfD

The RfD of 0.2 mg/kg-day listed by the TPHCWG for the C5-C8 aromatics fraction is based on the RfD values of six of the seven petroleum compounds identified in this fraction: toluene (0.2 mg/kg-day), ethylbenzene (0.1 mg/kg-day), styrene (0.2 mg/kg-day), and xylenes o-, m-, and p- (2.0 mg/kg-day). Though ethylbenzene had the most conservative RfD, the TPHCWG determined that an RfD of 0.2 mg/kg-day is appropriate because (a) the RfD for ethylbenzene is on the same order of magnitude as the RfD for toluene and styrene and (b) the relative portion of ethylbenzene to toluene is ten times lower in most unweathered products.

RfC

The RfC of 0.4 mg/m³ listed by the TPHCWG for the C5-C8 aromatics fraction is based on the RfC values for toluene (0.4 mg/m³), ethylbenzene (1 mg/m³), and styrene (1 mg/m³) available on IRIS at the time of publication. Since the publication of the TPHCWG guidance, the USEPA has updated the recommended RfC value for toluene and published a recommended RfC value for xylenes on the IRIS database. The RfC for toluene, previously based on a 1990 occupational study, has been updated to 5 mg/m³ based on multiple newer human studies with newer methodologies. Xylenes, which did not have a published RfC at the time of the TPHCWG publication, have a listed RfC of 0.1 mg/m³ based on a rat inhalation study.

Aromatics >C8-C10, >C10-C12, and >C12-C16

RfD

The RfD_o determined by the TPHCWG for the aromatic fractions >C8-C10, >C10-C12, and >C12-C16 is based on the RfD values for isopropylbenzene (cumene, 0.04 mg/kg-day), acenaphthene (0.06 mg/kg-day), biphenyl (0.05 mg/kg-day), fluorene (0.04 mg/kg-day), anthracene (0.3 mg/kg-day), fluoranthene (0.04 mg/kg-day), naphthalene (0.04 mg/kg-day), pyrene (0.03 mg/kg-day), and naphthalenes/methylnaphthalenes (0.03 mg/kg-day) available at the time of publication. The fraction-specific RfD was recommended to be 0.04 mg/kg-day, which was equal to the RfD values of isopropylbenzene, naphthalene, fluorene, and fluoranthene. With the exception of the RfD values for

pyrene and naphthalenes/methylnaphthalenes at 0.03 mg/kg-day, this was the most conservative of the available RfD values at the time of publication.

Since the publication of the TPHCWG guidance, updated RfD values have been added to the IRIS database for isopropylbenzene, naphthalene, and biphenyl. While isopropylbenzene and biphenyl were updated to RfD values a full order of magnitude higher than the previous values, the IRIS RfD value for naphthalene is listed as 0.02 mg/kg-day based upon a subchronic oral rat study. The current RfD values listed on IRIS for isopropylbenzene, biphenyl, and naphthalene should be taken into account when considering the fraction-specific RfD value for aromatic fractions >C8-C10, >C10-C12, and >C12-C16.

RfC

The RfC determined by the TPHCWG for aromatic fractions >C8-C10, >C10-C12, and >C12-C16 is based on the limited RfC data available for compounds in this carbon range. At the time of publication, RfC values had been published for two compounds within this carbon range: isopropylbenzene (0.09 mg/m³) and naphthalene (0.0013 mg/m³). As these compounds are not representative of the entire range, the TPHCWG used additional inhalation studies on C9 aromatics to supplement RfC calculations. Mice and rat inhalation studies on C9 aromatics resulted in the calculation of two RfC values: 1.3 mg/m³ and 0.2 mg/m³. The TPHCWG concluded that the data on the C9 aromatic mixtures was representative of these fractions, as it represents more compounds than single compound information, and determined that the more conservative value of 0.2 mg/m³, which translates into an RfD_i of 0.06 mg/kg-day would be representative of the entire >C8-C16 fraction.

It is worth noting that updated RfC values for isopropylbenzene (0.4 mg/m³) and naphthalene (0.003 mg/m³) were published on the IRIS database after the publication of the TPHCWG guidance. Both updated RfC values are higher than their previously published values.

Aromatics >C16-C21 and >C21-C35

RfD

At the time of publication, there were no previously developed RfD values for chemicals in this carbon range. As such, the TPHCWG determined that pyrene (C₁₆H₁₀) would be used as a surrogate for the fraction RfD. Pyrene has a lower carbon number than any compound in this fraction, which means that an RfD developed using pyrene will be a conservative value to represent the whole fraction. The RfD value for pyrene is 0.03 mg/kg-day.

RfC

At the time of publication, there were no previously developed RfC values for chemicals in this carbon range. Additionally, the TPHCWG determined that the development of RfC values for this fraction was inappropriate, as *“compounds within this carbon range are not volatile and inhalation will not be a relevant exposure pathway”* (TPHCWG, 1996).

The toxicological basis and surrogate compounds for each of the aliphatic and aromatic hydrocarbon fractions and TPH-mixtures are summarized in Table E.3

Table E.3: TPHCWG* Surrogate/indicator compounds for aliphatic and aromatic hydrocarbon fractions

Analyte	RfD	RfC	Surrogate/Indicator Compounds	RfD mg/kg-day	RfC mg/m ³
Aliphatic and Aromatic Hydrocarbon Fractions					
Aliphatics C5-C6 Aliphatics >C6-C8	5.0	18.4	n-hexane n-heptane Commercial hexane	0.06 [†] 2 5 [†]	0.2 7.6 18.4
Aliphatics >C8-C10 Aliphatics >C10-C12 Aliphatics >C12-C16	0.1	1.0	C10-C11 C7-C11 JP-8(C9-C16) C9-C12 C10-C13 C11-C17	NA NA 0.75 0.1 0.1 0.1	0.9 1 1 NA NA NA
Aliphatics >C16-C35	2.0 (C17-C34) 20 (>C35)	NA‡ NA‡	White mineral oils (C17-C34) White mineral oils (>C35)	2 20	NA‡ NA‡
Aromatics C7-C8	0.2	0.4	Ethylbenzene Styrene Toluene* Xylenes (m-, o-, and p-)	0.1 0.2 0.2 2	1 1 0.4 NA
Aromatics >C8-C10 Aromatics >C10-C12 Aromatics >C12-C16	0.04	0.2	Isopropylbenzene (cumene) Naphthalene Acenaphthene Biphenyl Fluorene Anthracene Fluoranthene Pyrene C9 Aromatics Naphthalenes/Methylnaphthalenes	0.04 0.04 0.06 0.05 0.04 0.3 0.04 0.03 NA 0.03	0.09 0.0013 NA NA NA NA NA NA 0.2 NA
Aromatics >C16-C21 Aromatics >C21-C35	0.03	NA‡	Pyrene	0.03	NA‡

NA: Not available. *The values in this table are the values used by the TPHCWG and do not include changes to the RfD and RfC values published after the TPHCWG guidelines were established. †Calculated from RfC. ‡TPHCWG does not derive a RfC or RfD for aliphatics >C16-C35 or aromatics >C16-C35 because "the development of an inhalation RfC from this fraction was determined to be inappropriate because the compounds in this carbon range are not volatile and inhalation will not be a relevant exposure pathway" (TPHCWG, 1996).

E.4.0 Toxicological basis for TPH-mixtures (TPH-GRO, TPH-DRO, TPH-ORO)

RECAP guidelines differ from the TPHCWG in that RECAP guidelines include the assessment of TPH-mixtures for gasoline-range organics (TPH-GRO; carbon ranges C6-C10), diesel-range organics (TPH-DRO; carbon ranges C10-C28), and oil-range organics (TPH-ORO; carbon ranges >C28). For these mixtures, the aliphatic or aromatic fraction with the most conservative RfD or RfC was used to represent the entire TPH-mixture. TPH-GRO is represented by the RfD values for the aromatics fraction >C8-C10 (RfD_o of 0.04

mg/kg-day; RfD_i of 0.06 mg/kg-day) which is based on the derived RfD for naphthalene/methyl naphthalenes (with consideration of other chemicals in this range) of 0.04 mg/kg-day and a RfC for C9 aromatics of 0.2 mg/m³. TPH-DRO is represented by the RfD values for the aromatics fraction >C16-C21 (RfD_o of 0.03 mg/kg-day; RfD_i of 0.06 mg/kg-day) which is based on the RfD for pyrene of 0.03 mg/kg-day and the RfD_i value (0.06 mg/kg-day) for the aromatics fraction >C10-C16 (the RfD_i value is based on the RfC for C9 aromatics of 0.2 mg/m³). TPH-ORO is represented by the RfD values for the aromatics fraction >C16-C35 (RfD_o of 0.03 mg/kg-day; RfD_i - NA) which is based on the RfD for pyrene of 0.03 mg/kg-day. RECAP does not define an RfD_i for the aliphatic and aromatic fractions >C16-C35 as the TPHCWG states that “the development of an inhalation RfC from this fraction was determined to be inappropriate because the compounds in this carbon range are not volatile and inhalation will not be a relevant exposure pathway” (TPHCWG, 1996).

The toxicological basis and surrogate compounds for each of the TPH-mixtures (i.e., TPH-GRO, TPH-DRO, and TPH-ORO) are summarized in Table E.4.

Table E.4: Surrogate/indicator compounds for TPH-mixtures (TPH-GRO, TPH-DRO, and TPH-ORO)

Analyte	RfD _o mg/kg-day	RfD _i mg/kg-day	Surrogate/Indicator Compounds	RfD mg/kg-day	RfC mg/m ³
Total Petroleum Hydrocarbon Mixtures					
TPH-GRO (C6-C10)	0.04	0.06	Naphthalene/Methylnaphthalenes C9 Aromatics	0.04 NA	0.0013 0.2
TPH-DRO (C10-C28)	0.03	0.06	Naphthalene/Methylnaphthalenes C9 Aromatics	0.04 NA	0.0013 0.2
TPH-ORO (>C28)	0.03	NA	Pyrene	0.03	NA

NA: Not available.

References

- LDEQ (2003a) *Appendix D: Louisiana Department of Environmental Quality*.
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Appendix F: Residential soil risk assessment

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F.1.0 Residential soil risk assessment

In addition to evaluating an industrial land use scenario as outlined in my report, I have also evaluated constituents under a non-industrial (i.e., residential) exposure scenario. Calculating a dose using the exposure assumptions incorporated into the non-industrial standards represents the most conservative approach to assessing health risks associated with the property, as it assumes the greatest amount of exposure to site constituents.

Dose calculations were derived for a hypothetical resident using sampling data (reported as wet-weight and dry-weight) for the respective constituents. The exposure point concentrations used to calculate the doses were derived from the reported wet or dry-weight concentrations presented below.

Table F-1: Exposure point concentrations (wet-weight)

Analyte		Wet-weight concentration (mg/kg)	Sample location	Depth (bgs)
Arsenic	site max	22.9	BC-1	4-6'
	maximum sample location average	18.9	BC-1	4-6'
	maximum tract average	5.1	NW Tract	0-18'
	maximum tract 95% UCL (0-2')	8.0	SW Tract	0-2'
Barium	site max	5573	SB-13	0-2'
	maximum sample location average	5143	SB-14R	2-4'
	maximum tract 95% UCL (0-2')	3207	SW	0-2'
Total TCDD TEQ	site max	0.0000069	BC-16R	2-4'

Table F-2: Exposure point concentrations (dry-weight)

Analyte		Wet-weight concentration (mg/kg-dry)	Sample location	Depth (bgs)
Arsenic	site max	27.3	SB-13	0-2'
	maximum sample location average	18.5	BC-1	4-6'
	maximum tract average	5.6	NW tract	0-18'
	maximum tract 95% UCL (0-2')	9.0	SW tract	0-2'
Barium	site max	9320	SB-13	0-2'
	maximum sample location average	6280	SB-13	0-2'
	maximum tract 95% UCL (0-2')	2545	SW tract	0-2'
Total TCDD TEQ	site max	0.0000094	BC-16R	2-4'

F.2.0 Soil noncancer risk calculations – child residential

The tables below present a comparison of theoretical child residential doses based on wet-weight and dry-weight concentrations to health-protective benchmarks and toxicity effect levels. Doses were calculated based on ingestion, dermal contact, and inhalation of soil particulates. Dose calculations were conducted for a hypothetical child residential receptor, as this represents the most conservative potential exposure scenario. Only the child receptor is considered since the child is assumed to have exposures that

are higher than those of an adult when considered on a body weight basis. For this reason, the daily intake calculated for a child resident was used to evaluate the non-cancer health effects associated with exposures to arsenic and barium within site soil. Dose calculations were conducted using methodology consistent with LDEQ RECAP and USEPA.

Table F-3: Comparison of a child residential dose to health-protective benchmarks and toxicity effect levels (wet-weight)

Analyte		Total Intake (mg/kg-dry)	RfD (mg/kg-d)	Daily Dose fold-below RfD	NOAEL (mg/kg- day)	Daily Dose fold-below NOAEL	LOAEL (mg/kg- day)	Daily Dose fold-below LOAEL
Arsenic	site max	2.00E-04	3.00E-04	1.5	8.00E-04	4	1.40E-02	70
	Maximum sample location average	1.65E-04	3.00E-04	2	8.00E-04	5	1.40E-02	85
	maximum tract average	4.46E-05	3.00E-04	7	8.00E-04	18	1.40E-02	314
	maximum tract 95% UCL (0-2')	6.98E-05	3.00E-04	4	8.00E-04	11	1.40E-02	201
Barium	site max	7.13E-02	2.00E-01	3	6.30E+01	884	8.40E+01	1,179
	maximum sample location average	6.58E-02	2.00E-01	3	6.30E+01	958	8.40E+01	1,277
	maximum tract 95% UCL (0-2')	4.10E-02	2.00E-01	5	6.30E+01	1,536	8.40E+01	2,049
Total TCDD TEQ	site max	9.56E-11	7.00E-10	7	NA	NA	2.00E-08	209

Table F-4: Comparison of a child residential dose to health-protective benchmarks and toxicity effect levels (dry-weight)

Analyte		Total Intake (mg/kg-dry)	RfD (mg/kg-d)	Daily Dose fold-below RfD	NOAEL (mg/kg- day)	Daily Dose fold-below NOAEL	LOAEL (mg/kg- day)	Daily Dose fold-below LOAEL
Arsenic	site max	2.39E-04	3.00E-04	1.3	8.00E-04	3	1.40E-02	59
	maximum sample location average	1.62E-04	3.00E-04	2	8.00E-04	5	1.40E-02	87
	maximum tract average	4.92E-05	3.00E-04	6	8.00E-04	16	1.40E-02	284
	maximum tract 95% UCL (0-2')	7.91E-05	3.00E-04	4	8.00E-04	10	1.40E-02	177
Barium	site max	1.19E-01	2.00E-01	2	6.30E+01	529	8.40E+01	705
	maximum sample location average	8.03E-02	2.00E-01	2	6.30E+01	785	8.40E+01	1,046
	maximum tract 95% UCL (0-2')	3.25E-02	2.00E-01	6	6.30E+01	1,936	8.40E+01	2,581
Total TCDD TEQ	site max	1.30E-10	7.00E-10	5	NA	NA	2.00E-08	154

The tables above demonstrate that the calculated doses for the respective constituents for a child residential exposure scenario are below health protective RfDs, concentrations representing no adverse effect levels (i.e., NOAELs or BMDs), and below concentrations where toxicological effects are reported (i.e., LOAELs or BMDs). As such these concentrations in soil from the property do not represent levels that would be associated with a risk of adverse non-cancer health effects for a child residential exposure scenario following direct contact.

F.3.0 Soil cancer risk calculations - residential

As arsenic and dioxins (i.e., 2,3,7,8-TCDD) are classified as human carcinogens, an assessment of theoretical cancer risks was conducted based on the wet-weight and dry-weight arsenic and the total TCDD TEQ concentrations, which included site maximum values, maximum sample location averages, maximum tract averages, or 95% UCL-AMs. The calculated cancer risk includes exposure via inhalation, ingestion, and dermal contact and represents the cumulative cancer risk for a lifetime spent on the property.

Theoretical cancer risks associated with exposure to arsenic/dioxins in soil were calculated as follows:

$$Risk = LADI \times SF$$

where:

Risk = a unitless probability (e.g. 2E-5) of an individual developing cancer over a 70 year lifetime.

LADI = lifetime average daily intake averaged over 70 years (mg/kg-day)
 SF = oral and dermal slope factor, expressed in (mg/kg-day)⁻¹

The resulting cancer risks are expressed in scientific notation (e.g., 1.0E-04 to 1.0E-06) and refer to additional lifetime cancer risks of one cancer in 10,000 persons to one cancer in 1,000,000 persons. For example, a calculated theoretical lifetime cancer risk of 1.0E-05 (or 1 in 100,000) indicates that if 100,000 people were exposed to a potentially carcinogenic chemical throughout their 70-year lifetimes, one of the 100,000 individuals would theoretically develop cancer from the lifetime of exposure. The USEPA generally considers total lifetime cancer risks between 1.0E-04 and 1.0E-06 as acceptable for exposures to multiple chemicals with potential carcinogenic effects. Cancer risk calculations for a lifetime residential exposure scenario are presented below.

Table F-5: Summary of cancer risk from soil (wet-weight; residential exposure scenario)

Analyte	EPC (mg/kg)	Cumulative Child & Adult Cancer Risk
Arsenic	site max	22.9
	Maximum sample location average	18.9
	maximum tract average	5.1
	maximum tract 95% UCL (0-2')	8.0
Total TCDD TEQ	site max	0.0000069
		1.35E-06

Table F-6: Summary of cancer risk from soil (dry-weight; residential exposure scenario)

Analyte	EPC (mg/kg-dry)	Cumulative Child & Adult Cancer Risk
Arsenic	site max	27.3
	Maximum sample location average	18.5
	maximum tract average	5.6
	maximum tract 95% UCL (0-2')	9.0
Total TCDD TEQ	site max	0.0000094
		1.84E-06

As demonstrated above, the calculated cancer risks for lifetime residential scenarios for the various wet and dry-weight concentrations fall within the USEPA acceptable risk range of 1.0E-04 and 1.0E-06. To put these risks into perspective, a discussion of lifetime cancer probabilities is relevant. Unfortunately, the development of cancer is a major public health problem worldwide and is the second leading cause of death in the United States. The lifetime probability of being diagnosed with invasive cancer is slightly higher for men (39.7% or 0.397) than for women (37.6% or 0.376) (Siegel et al., 2018). Using a lifetime residential exposure scenario, which assumes the individual would be exposed to arsenic at the respective concentration for 350 days per year for a total of 36 years (six years as child; 30 years as adult), based on the wet-weight 95%UCL-AM for the 0-2' bgs range, would result in an increased theoretical total cancer

risk for arsenic of 1.16E-05 (0.00116% or 0.0000116). For the site maximum wet-weight the total TCDD TEQ concentration, the increased theoretical total cancer risk is 1.35E-06 (0.000135% or 0.00000135). These theoretical upper bound calculated cancer risks pale in magnitude compared to the population cancer risk in the United States. Thus, these calculated risks fall within the acceptable risk ranges established by the USEPA and include numerous health-protective uncertainties (i.e., conservative exposure parameters) that are inherent in the risk calculation process. As such, these concentrations do not represent a risk to human health.

In summary, the calculation of a theoretical upper-bound cancer risk for arsenic and the total TCDD TEQ concentrations detected on the property indicated that these risks fall within or below the 1E-04 to 1E-06 range deemed acceptable by the USEPA. It should be noted that an individual would not be expected to spend their entire time on the property at one location. As such, using a maximum reported value from one sample location would significantly overestimate the risk to an individual present on the property.

F.4.0 References

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Appendix G: Off-site soil risk assessment

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G.1.0 Off-site soil risk assessment

In addition to evaluating on-site exposure scenarios, I have also conducted a risk assessment for constituents exceeding screening standards in samples collected outside the NE property tract boundaries. The exposure point concentrations used to calculate the doses were derived from the reported wet or dry-weight concentrations presented below.

Table G-1: Exposure point concentrations (wet-weight)

Analyte	Wet-weight concentration		Sample location	Depth (bgs)
		(mg/kg)		
Aliphatic >C10-C12	site max	6,000	BC-8	4-6'
	95% UCL (0-10')	1,240	NA	0-10'
Aliphatic >C12-C16	site max	12,200	BC-8R2	4-6'
	95% UCL (0-10')	13,015	NA	0-10'
Aliphatic >C16-C35	site max	30,800	BC-8R2	4-6'
	95% UCL (0-10')	10,441	NA	0-10'
Aromatic >C8-C10	site max	810	BC-8R2	4-6'
	95% UCL (0-10')	309	NA	0-10'
Aromatic >C21-C35	site max	3,980	BC-8	4-6'
	95% UCL (0-10')	2,204	NA	0-10'

Table G-2: Exposure point concentrations (dry-weight)

Analyte	Dry-weight concentration		Sample location	Depth (bgs)
		(mg/kg-dry)		
Arsenic	site max	15.3	BC-7	10-12'
	area average (0-10')	5.65	NA	0-10'
	95% UCL (0-10')	7.61	NA	0-10'
Aliphatic >C10-C12	site max	5,162	BC-8	2-4'
	95% UCL (0-10')	1,100	NA	0-10'
Aliphatic >C12-C16	site max	10,858	BC-8R2	4-6'
	95% UCL (0-10')	11,900	NA	0-10'
Aliphatic >C16-C35	site max	27,412	BC-8R2	4-6'
	95% UCL (0-10')	9,032	NA	0-10'
Aromatic >C8-C10	site max	721	BC-8R2	4-6'
	95% UCL (0-10')	269	NA	0-10'
Aromatic >C21-C35	site max	3,259	BC-8	2-4'
	95% UCL (0-10')	1,857	NA	0-10'

G.2.0 Soil noncancer risk calculations

The tables below present a comparison of theoretical doses based on wet-weight and dry-weight concentrations to health-protective benchmarks and toxicity effect levels. Doses were calculated based on ingestion, dermal contact, and inhalation of soil particulates. Dose calculations were conducted under adult industrial and child residential exposure scenarios for constituents exceeding their respective

industrial or non-industrial screening standards. Dose calculations were conducted using methodology consistent with LDEQ RECAP and USEPA.

Table G-3: Comparison of an adult industrial dose to health-protective benchmarks and toxicity effect levels (wet-weight)

Analyte		Total Intake (mg/kg-dry)	RfD (mg/kg-d)	Daily Dose fold-below RfD	NOAEL (mg/kg- day)	Daily Dose fold-below NOAEL	LOAEL (mg/kg- day)	Daily Dose fold-below LOAEL
Aliphatic >C12-C16	site max	5.22E-03	1.00E-01	19	1.00E+02	19,147	5.00E+02	95,733
	95% UCL (0-10')	5.57E-03	1.00E-01	18	1.00E+02	17,948	5.00E+02	89,738
Aliphatic >C16-C35	site max	3.06E-02	2.00E+00	65	2.00E+02	6,538	2.00E+03	65,381
	95% UCL (0-10')	1.04E-02	2.00E+00	193	2.00E+02	19,287	2.00E+03	192,869

Table G-4: Comparison of an adult industrial dose to health-protective benchmarks and toxicity effect levels (dry-weight)

Analyte		Total Intake (mg/kg-dry)	RfD (mg/kg-d)	Daily Dose fold-below RfD	NOAEL (mg/kg- day)	Daily Dose fold-below NOAEL	LOAEL (mg/kg- day)	Daily Dose fold-below LOAEL
Arsenic	site max	6.52E-06	3.00E-04	46	8.00E-04	123	1.40E-02	2,146
	area average	2.41E-06	3.00E-04	125	8.00E-04	332	1.40E-02	5,811
	95% UCL (0-10')	3.25E-06	3.00E-04	92	8.00E-04	246	1.40E-02	4,313
Aliphatic >C12-C16	site max	4.65E-03	1.00E-01	22	1.00E+02	21,513	5.00E+02	107,565
	95% UCL (0-10')	5.09E-03	1.00E-01	20	1.00E+02	19,629	5.00E+02	98,146
Aliphatic >C16-C35	site max	2.72E-02	2.00E+00	73	2.00E+02	7,346	2.00E+03	73,462
	95% UCL (0-10')	8.97E-03	2.00E+00	223	2.00E+02	22,296	2.00E+03	222,957

Table G-5: Comparison of a child residential dose to health-protective benchmarks and toxicity effect levels (wet-weight)

Analyte		Total Intake (mg/kg-dry)	RfD (mg/kg-d)	Daily Dose fold- below RfD	NOAEL (mg/kg- day)	Daily Dose fold-below NOAEL	LOAEL (mg/kg- day)	Daily Dose fold-below LOAEL
Aliphatic >C10-C12	site max	7.67E-02	1.00E-01	1.3	1.00E+02	1,304	5.00E+02	6,518
	95% UCL (0-10')	1.59E-02	1.00E-01	6	1.00E+02	6,307	5.00E+02	31,537
Aliphatic >C12-C16	site max	1.56E-01	1.00E-01	--	1.00E+02	641	5.00E+02	3,205
	95% UCL (0-10')	1.66E-01	1.00E-01	--	1.00E+02	601	5.00E+02	3,005
Aliphatic >C16-C35	site max	5.04E-01	2.00E+00	4	2.00E+02	397	2.00E+03	3,968
	95% UCL (0-10')	1.71E-01	2.00E+00	12	2.00E+02	1,170	2.00E+03	11,705
Aromatic >C8-C10	site max	1.04E-02	4.00E-02	4	3.00E+02	28,968	3.00E+02	28,968
	95% UCL (0-10')	3.95E-03	4.00E-02	10	3.00E+02	75,935	3.00E+02	75,935
Aromatic >C21-C35	site max	6.51E-02	3.00E-02	--	7.50E+01	1,151	1.25E+02	1,919
	95% UCL (0-10')	3.61E-02	3.00E-02	--	7.50E+01	2,079	1.25E+02	3,466

Table G-6: Comparison of a child residential dose to health-protective benchmarks and toxicity effect levels (dry-weight)

Analyte		Total Intake (mg/kg-dry)	RfD (mg/kg-d)	Daily Dose fold- below RfD	NOAEL (mg/kg- day)	Daily Dose fold-below NOAEL	LOAEL (mg/kg- day)	Daily Dose fold-below LOAEL
Arsenic	site max	1.34E-04	3.00E-04	2	8.00E-04	6	1.40E-02	105
	area average	4.94E-05	3.00E-04	6	8.00E-04	16	1.40E-02	283
	95% UCL (0-10')	6.66E-05	3.00E-04	5	8.00E-04	12	1.40E-02	210
Aliphatic >C10-C12	site max	6.60E-02	1.00E-01	2	1.00E+02	1,515	5.00E+02	7,576
	95% UCL (0-10')	1.41E-02	1.00E-01	7	1.00E+02	7,110	5.00E+02	35,551
Aliphatic >C12-C16	site max	1.39E-01	1.00E-01	--	1.00E+02	720	5.00E+02	3,602
	95% UCL (0-10')	1.52E-01	1.00E-01	--	1.00E+02	657	5.00E+02	3,286
Aliphatic >C16-C35	site max	4.49E-01	2.00E+00	4	2.00E+02	446	2.00E+03	4,458
	95% UCL (0-10')	1.48E-01	2.00E+00	14	2.00E+02	1,353	2.00E+03	13,531
Aromatic >C8-C10	site max	9.22E-03	4.00E-02	4	3.00E+02	32,548	3.00E+02	32,548
	95% UCL (0-10')	3.44E-03	4.00E-02	12	3.00E+02	87,226	3.00E+02	87,226
Aromatic >C21-C35	site max	5.33E-02	3.00E-02	--	7.50E+01	1,406	1.25E+02	2,344
	95% UCL (0-10')	3.04E-02	3.00E-02	1	7.50E+01	2,468	1.25E+02	4,113

The tables above demonstrate that the calculated doses for the respective constituents for a child residential exposure scenario are below health protective RfDs, concentrations representing no adverse effect levels (i.e., NOAELs or BMDs), or below concentrations where toxicological effects are reported (i.e., LOAELs or BMDs). As such these concentrations in soil from the property do not represent levels that would be associated with a risk of adverse non-cancer health effects for a child residential exposure scenario following direct contact.

G.3.0 Soil cancer risk calculations

As arsenic is classified as a human carcinogen, an assessment of theoretical cancer risks was conducted based on the dry-weight arsenic concentrations presented above. Wet-weight concentrations of arsenic did not exceed applicable screening standards; thus, dose calculations were performed only for dry-weight concentrations. The calculated cancer risk includes exposure via inhalation, ingestion, and dermal contact and represents the cumulative cancer risk for a lifetime spent on the property.

Theoretical cancer risks associated with exposure to arsenic/dioxins in soil were calculated as follows:

$$Risk = LADI \times SF$$

where:

Risk = a unitless probability (e.g. 2E-5) of an individual developing cancer over a 70 year lifetime.

LADI = lifetime average daily intake averaged over 70 years (mg/kg-day)

SF = oral and dermal slope factor, expressed in (mg/kg-day)⁻¹

The resulting cancer risks are expressed in scientific notation (e.g., 1.0E-04 to 1.0E-06) and refer to additional lifetime cancer risks of one cancer in 10,000 persons to one cancer in 1,000,000 persons. For example, a calculated theoretical lifetime cancer risk of 1.0E-05 (or 1 in 100,000) indicates that if 100,000 people were exposed to a potentially carcinogenic chemical throughout their 70-year lifetimes, one of the 100,000 individuals would theoretically develop cancer from the lifetime of exposure. The USEPA generally considers total lifetime cancer risks between 1.0E-04 and 1.0E-06 as acceptable for exposures to multiple chemicals with potential carcinogenic effects. Cancer risk calculations for a lifetime residential exposure scenario are presented below.

Table G-7: Summary of cancer risk from soil (dry-weight; adult industrial scenario)

Analyte	EPC (mg/kg)	Cumulative Cancer Risk
Arsenic	site max	3.50E-06
	maximum location average	1.29E-06
	maximum tract average	1.74E-06

Table G-8: Summary of cancer risk from soil (dry-weight; residential exposure scenario)

Analyte	EPC (mg/kg)	Cumulative Child & Adult Cancer Risk
Arsenic	site max	2.22E-05
	maximum location average	8.18E-06
	maximum tract average	1.10E-05

As demonstrated above, the calculated cancer risks for the industrial and residential scenarios for the various dry-weight concentrations fall within the USEPA acceptable risk range of 1.0E-04 and 1.0E-06 and include numerous health-protective uncertainties (i.e., conservative exposure parameters) that are inherent in the risk calculation process.