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PREDICTING THE TOXICITY OF NEAT AND WEATHERED CRUDE OIL: TOXIC POTENTIAL AND THE TOXICITY OF SATURATED MIXTURES

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Abstract—The toxicity of oils can be understood using the concept of toxic potential, or the toxicity of each individual component of the oil at the water solubility of that component. Using the target lipid model to describe the toxicity and the observed relationship of the solubility of oil components to log (K_{OW}), it is demonstrated that components with lower log (K_{OW}) have greater toxic potential than those with higher log (K_{OW}). Weathering removes the lower-log (K_{OW}) chemicals with greater toxic potential, leaving the higher-log (K_{OW}) chemicals with lower toxic potential. The replacement of more toxically potent compounds with less toxically potent compounds lowers the toxicity of the aqueous phase in equilibrium with the oil. Observations confirm that weathering lowers the toxicity of oil. The idea that weathering increases toxicity is based on the erroneous use of the total petroleum hydrocarbons or the total polycyclic aromatic hydrocarbons (PAHs) concentration as if either were a single chemical that can be used to gauge the toxicity of a mixture, regardless of its makeup. The toxicity of the individual PAHs that comprise the mixture varies. Converting the concentrations to toxic units (TUs) normalizes the differences in toxicity. A concentration of one TU resulting from the PAHs in the mixture implies toxicity regardless of the specific PAHs that are present. However, it is impossible to judge whether 1 μg/L of total PAHs is toxic without knowing the PAHs in the mixture. The use of toxic potential and TUs eliminates this confusion, puts the chemicals on the same footing, and allows an intuitive understanding of the effects of weathering.

Keywords—Polycyclic aromatic hydrocarbons Target lipid model Model validation Oil toxicity

INTRODUCTION

The purpose of this paper is to present and verify a method for predicting the aquatic and sediment toxicity of fresh and weathered crude oils. It is pointed out that the change in toxicity caused by weathering, which has been the subject of some debate [1,2], can be understood in terms of the relationship between toxicity and aqueous solubility of the components of oil. The method also can be applied to other hydrocarbon mixtures (e.g., coal tars).

The present paper describes the ideas behind the method and provides the basis for understanding the underlying principles. To predict the toxicity of complex mixtures, several problems arise. First, it is necessary to predict the aqueous and/or sediment concentrations of the components in oil in the presence of the oil phase. Second, it is necessary to predict the aqueous and/or sediment toxicity of the resulting concentrations of each component. Finally, it is necessary to predict the toxicity of the mixture of component concentrations. Comparisons to observed toxicity from aquatic and sediment exposures are provided to validate the proposed procedure.

Target lipid model

Petroleum is a complex mixture with many components, including straight-chain, branched, cyclic, monocyclic aromatic, and polycyclic aromatic hydrocarbons (PAHs). The majority exhibit a narcotic toxicity mode of action [3] that can be predicted using various quantitative structure–activity relationships. It is assumed that all the significant toxic components of the mixture are of these types. The octanol/water partition coefficient (K_{OW}) is the usual chemical property used in the quantitative structure–activity relationship. For a specific organism, the relationship between the lethal concentration to 50% of test organisms (LC50) for a series of chemicals and their K_{OW} values is

$$\log (\text{LC50}) = m \log (K_{OW}) + b \quad (1)$$

where $m \approx -1$ and b is the intercept of the equation (for review, see Hermens [4]). This relationship has been rationalized for fish by noting that the log(BCF)—where BCF is the bioconcentration factor, or the ratio of organism to aqueous concentration—increases with log(K_{OW}) with a slope $m \approx +1$. Thus, the critical body burden corresponding to the LC50, which is the product of the BCF and the LC50, is a constant, independent of the identity of the chemical [5]. This insight clarified the mechanism involved and pointed out the utility of using critical body burdens to explain narcotic toxicity.

The target lipid model (TLM) of narcotic toxicity [6,7] extends these ideas. It is based on the assumption, which has been validated by an extensive data analysis and more recent applications [8,9], that the slope m in Equation 1 is a universal constant, independent of organism identity and chemical classes. This would be the case if the target lipid (i.e., the site of action for narcotic toxicity) is chemically the same for all tested organisms. The critical body burdens, however, are different for different organisms, reflecting the differences in species sensitivity.

To apply the body burden model to organisms other than fish, a BCF relationship would be required for each organism. In the TLM, it is observed that the y-intercept b in Equation 1 can be interpreted as the lipid-normalized critical body burden C_L^* that corresponds to the observed endpoint, such as 50% mortality for the LC50 for the specific organism being considered. The critical body burdens derived from this pro-

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cedure are then used to calculate water and sediment criteria following the U.S. Environmental Protection Agency (U.S. EPA) guidelines [10]. As will become clear, the fact that the slope m is a universal constant greatly simplifies the analysis of the toxicity of mixtures.

The notation used is as follows: Organisms are indexed by i and chemicals by j, the superscript * is used to denote critical effects concentrations (e.g., LC50s), and the subscripts W, L, and S are used to denote water, lipid, and solid, respectively. The TLM equation that predicts the critical aqueous concentration is from Di Toro et al. [6]

$$\log (C^*_{W,ij}) = -0.945 \log (K_{OW,j}) + \log (C^*_{L,i}) + \Delta c_j \quad (2)$$

where $C^*_{W,ij}$ is the critical aqueous concentration (mmol/L; e.g., $C^*_{W,ij} = LC50_{ij}$ for a mortality endpoint), $m = -0.945$ the universal slope, $K_{OW,j}$ is the octanol/water partition coefficient for chemical j, and $C^*_{L,i}$ is the corresponding critical body burden (μmol/g octanol) for organism i derived from the intercepts (Eqn. 1). These estimated body burdens have been compared to measured body burdens (μmol/g lipid) and found to be comparable [6]. In addition, a correction Δc_j exists for chemical classes, such as PAHs, that exhibit additional toxicity when compared to baseline narcotics, and this correction ranges from zero (alkanes) to −0.263 (PAHs). The critical body burdens for LC50s range from $C^*_{L,i} = 286$ μmol/g octanol for *Tetrahymena elliotti* to 34.3 μmol/g octanol for *Mysidopsis bahia*. The critical body burden corresponding to the 5th-percentile organism sensitivity that is the final acute value (FAV) used in the U.S. EPA water-quality criteria derivation [10] is 35.3 μmol/g octanol. The median acute to chronic ratio is 5.09, so the chronic critical body burden is 6.94 μmol/g octanol [6,7]. Thus, the TLM provides a method of computing the toxicity of type I narcotics in general and of PAHs in particular.

Application to sediment toxicity

The equivalent equation for sediment toxicity can be derived using the equilibrium partitioning model [11]. This model is based on the observation that the sediment toxicity can be predicted by comparing the pore-water concentration to the critical concentration $C^*_{W,ij}$ in water-only exposures [12]. If an equilibrium is assumed to exist between the pore water and the organic carbon (OC) phase of the sediment, and if the pore-water toxicity is assumed to be predictable from the TLM (Eqn. 2), then the sediment concentration producing the same effect on the same organism is

$$C^*_{S,ij} = K_{OC,j} C^*_{W,ij} \quad (3)$$

where $C^*_{S,ij}$ is the OC-normalized critical sediment concentration (μmol/g OC), $K_{OC,j}$ is the OC partitioning coefficient (L/kg OC) for chemical j, and $C^*_{W,ij}$ is given by Equation 2. The value of $K_{OC,j}$ can be estimated [11] by

$$\log (K_{OC,j}) = 0.00028 + 0.983 \log (K_{OW,j}) \quad (4)$$

and the equation for predicting the critical sediment concentration using the TLM is

$$\log (C^*_{S,ij}) = 0.00028 + 0.038 \log (K_{OW,j}) + \log (C^*_{L,i}) + \Delta c_j \quad (5)$$

Note that to a close approximation, 0.00028 and 0.038 are approximately zero, and Equation 5 becomes

$$\log (C^*_{S,ij}) \cong \log (C^*_{L,i}) + \Delta c_j \quad (6)$$

so that the critical OC-normalized sediment concentration $C^*_{S,ij}$ for classes of chemicals for which Δc_j is constant (e.g., PAHs) is the same. This is the case because the critical lipid-normalized concentration $C^*_{L,i}$ is independent of the chemical identity j and depends only on the organism identity i.

Equations 2 and 5 predict the toxicity of the individual components of an oil mixture. It remains to determine the toxicity of the mixture. This is addressed in a subsequent section.

TOXIC POTENTIAL

Understanding of the toxicity of mixtures and of the effect of weathering is greatly aided by the concept of toxic potential first introduced by Bobra et al. [13]. Consider an immiscible, nonaqueous liquid phase made up of one component (e.g., benzene) in equilibrium with an aqueous phase. Define the toxic potential of that component as the toxicity of a saturated aqueous solution, a solution at the aqueous solubility of the component. It is the maximum concentration of free chemical that can be reached in an aqueous solution and, therefore, corresponds to the maximum toxicity that can be exerted.

It is convenient to use toxic units (TUs) to compare the toxic potential of different chemicals. An aqueous TU is defined as the ratio of the water-column concentration C_W and the critical concentration C^*_W:

$$TU = \frac{C_W}{C^*_W} \quad (7)$$

For example if the critical concentration is a LC50, then TU = C_W/LC50.

The toxic potential is expressed in TUs and is denoted by $TU_{W,max}$. Because the aqueous phase is assumed to be in equilibrium with the pure liquid—the definition of a saturated solution—the aqueous concentration is $C_W = S_L$, where S_L is the water solubility of the nonaqueous liquid (e.g., the solubility of benzene). For this case, the TU concentration in the solution and, therefore, the toxic potential is

$$TU_{W,max} = \frac{S_L}{C^*_W} \quad (8)$$

where C^*_W is the critical concentration of the single compound (Eqn. 2). Thus, the acute toxic potential of a chemical is defined as the ratio of the maximum aqueous concentration (i.e., its aqueous solubility) to its LC50. Therefore, it is the maximum TU concentration that a chemical can achieve.

Example

Because the idea of toxic potential is central to understanding the effect of weathering on toxicity, two examples are presented. Consider benzene, a comparatively soluble constituent of oil with $S_L = 26$ mmol/L (2,000 mg/L). Because it is only moderately hydrophobic, with log (K_{OW}) = 2.0, it is not particularly toxic. It has a LC50 FAV of $C^*_W = 0.45$ mmol/L (35.5 mg/L). However, because its solubility (2,000 mg/L) is so much larger than its toxicity (35.5 mg/L), its toxic potential is $TU_{W,max}$ = (2,000 mg/L)/(35.5 mg/L) = 57.2, so a saturated solution of benzene has a TU concentration of nearly 60 TUs.

By contrast, phenanthrene, which is a solid at room temperature, is substantially less soluble ($S_S = 6.16$ μmol/L [1.1 mg/L]) and more hydrophobic, with log (K_{OW}) = 4.57. The LC50 FAV of phenanthrene is $C^*_W = 0.92$ μmol/L (0.165 mg/L), which is considerably less than that of benzene, and so phenanthrene is more toxic than benzene. However, its

toxic potential is $TU_{W,max}$ = (1.1 mg/L)/(0.165 mg/L) = 6.16, almost an order of magnitude lower than that of benzene. Thus, although phenanthrene is more toxic than benzene (LC50, 0.165 vs 35.5 mg/L), it is even more insoluble (S_S = 1.1 mg/L vs S_L = 2,000 mg/L), so its toxic potential is less than that of benzene. This may seem to be counterintuitive: A less toxic (higher LC50) chemical, benzene, has a higher toxic potential than a more toxic (lower LC50) chemical, phenanthrene. Because of the relationship between toxicity and solubility, however, this is, in fact, the case.

Subcooled liquid

An important correction needs to be made when mixtures are considered. When nonaqueous miscible liquids are mixed together, they usually remain liquids. However, mixtures of solids can liquefy via the mutual suppression of their melting points. This is the situation for petroleum, for which many of the components (e.g., the heavier PAHs) are solids at room temperature. In this case, the appropriate solubility at which to evaluate toxic potential for application to liquid mixtures is the subcooled liquid solubility—that is, the solubility of each component if it were a liquid at the temperature of interest. This is the appropriate solubility, because in a liquid hydrocarbon mixture, the components are, in fact, liquids. This hypothetical solubility for a single solid is computed using the relationship proposed by Yalkowsky [14]:

$$\frac{S_L}{S_S} = \exp[-6.8(1 - T_M/T)] \quad (9)$$

where T_M (K) and T is the temperature (K) of interest.

The distinction between the solid and subcooled solubility is important, because the difference is substantial. For phenanthrene, the subcooled solubility is S_L = 35.5 μmol (6.33 mg/L), so its toxic potential is higher ($TU_{W,max}$, 38.4 vs 6.16 for the solid). It is still approximately a factor of 1.5 lower than the toxic potential of benzene, with $TU_{W,max}$ = 57.2.

Comparisons

The example presented above is based on estimates of toxicity using the TLM. An analysis based on observed toxicity data is presented in this section. Figure 1 presents the observed *Pimephales promelas* and *Daphnia magna* LC50s and the corresponding subcooled liquid solubility data versus log (K_{OW}) for narcotic chemicals [6]. The regression lines for the LC50s have the universal narcosis slope. The solubility data also are fit with regression lines (parameters in Table 1). Table 1 also presents the toxic potential computed using the regression lines. This is compared to the toxic potential for each chemical where the individual data are averaged in log (K_{OW}) intervals. The mean and range of the data are presented. For *P. promelas*, the toxic potential decreases from $TU_{W,max} \cong 40$ to $TU_{W,max} \cong 10$ as log (K_{OW}) increases from 0.5 to 4.0. For *D. magna*, the decline is similar. Thus, the decrease in toxic potential as K_{OW} increases that is predicted by the TLM is, in fact, observed for narcotic chemicals in general.

A similar analysis is presented for PAHs in Figure 2. The LC50s for *P. promelas* and *D. magna* and the corresponding solubility data versus log (K_{OW}) for the solid PAHs are shown [6]. The regression lines for the LC50s have the universal narcosis slope. The toxic potential is much lower for the solids, because they have a lower solubility, which ranges from $TU_{W,max} \cong 15$ to $TU_{W,max} \cong 1.5$ for both species.

The data presented in Figure 1 include not only hydrocarbons but also other classes of organic chemicals that exhibit narcotic toxicity, such as ethers, ketones, and chlorinated hydrocarbons. An analysis of data for 66 aromatics and 11 alkanes found in petroleum for which subcooled liquid solubilities are available [15–17] is presented in Figure 3A. The regression line

$$\log (S_L) = 3.54 - 1.10 \log (K_{OW}) \quad (10)$$

is used together with the TLM estimates of toxicity (Eqn. 2) to compute the toxic potential of the PAH components of petroleum:

$$\log (TU_{W,max}) = \log (S_L) - \log (C^*_{W,ij}) \quad (11)$$

Substituting Equations 10 and 2 yields

$$\log (TU_{W,max}) = 3.54 - 1.10 \log (K_{OW}) - [-0.945 \log (K_{OW}) + \log (C^*_{L,i}) + \Delta c_j] \quad (12)$$

and simplifying yields

$$\log (TU_{W,max}) = -0.155 \log (K_{OW}) + 3.54 - \log (C^*_{L,i}) - \Delta c_j \quad (13)$$

This relationship for baseline narcotics ($\Delta c_j = 0$) and PAHs ($\Delta c_j = -0.263$) and for the FAV and final chronic value (FCV) critical body burdens $C^*_{L,i}$ = 35.3 and 6.94 μmol/g octanol, respectively, are presented in Figure 3B. Toxic potential decreases by fivefold over the range of log (K_{OW}) = 2–7. The PAHs are approximately a factor of two more potent because of Δc_j. The difference between acute and chronic toxic potential results from the median acute to chronic ratio of 5.09.

The negative slope of −0.155 in Equation 13, which results from the subcooled liquid solubility decreasing more rapidly (slope = −1.10) than the universal narcosis slope (slope = −0.945), causes the toxic potential to decrease as log (K_{OW}) increases. We shall see that this is the reason for the decrease in toxicity as petroleum weathers.

Mixtures

Predicting the toxicity of oils requires that the toxicity of mixtures of oil components can be predicted. It has been demonstrated for narcotics in general (for review, see Hermens [4]) and for PAHs in particular [7,18] that they are additive on a TU basis. For chemical j, $TU_{W,j}$ is the ratio of the water-column concentration $C_{W,j}$ and the critical concentration $C^*_{W,j}$ (Eqn. 2):

$$TU_{W,j} = \frac{C_{W,j}}{C^*_{W,j}} \quad (14)$$

The toxicity of the mixture is determined by the sum of the individual TUs:

$$TU = \sum_j TU_{W,j} \quad (15)$$

If TU = 1, then the mixture is predicted to be toxic. If the effects concentrations $C^*_{W,j}$ are LC50s, then a mixture with TU = 1 would be predicted to cause 50% mortality of the test organisms.

The remaining problem is the prediction of the aqueous concentrations $C_{W,j}$ for each of the j components in the petroleum in equilibrium with the solution. With only one component, such as benzene, then the concentration of benzene in the water phase would be $C_W = S_L$, as shown above, where S_L is the aqueous solubility of that component. For a mixture

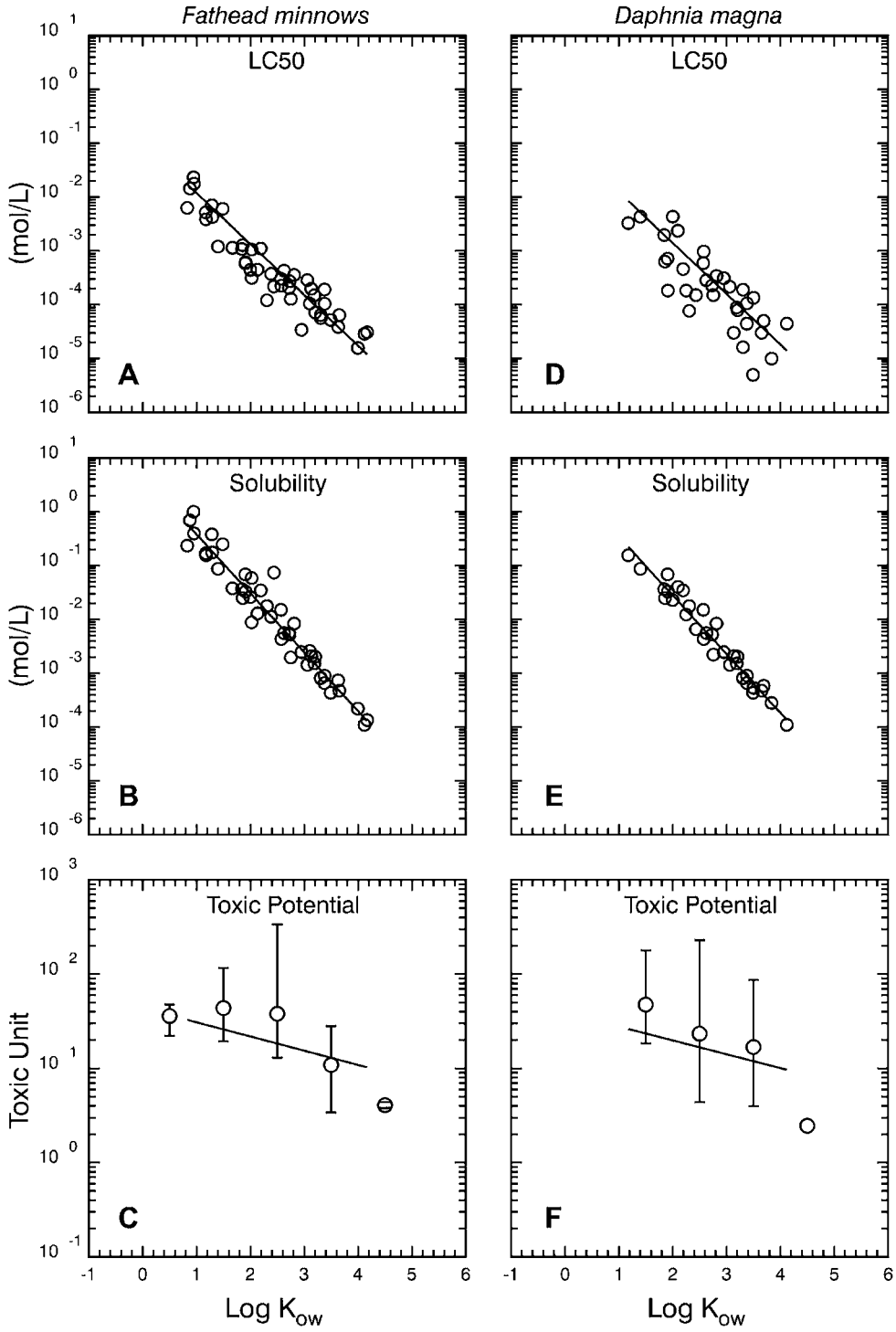

Fig. 1. Paired lethal concentrations to 50% test organisms (LC50s; **A** and **D**) and subcooled liquid solubility (**B** and **E**) versus log (K_{OW}) for narcotic chemicals for fathead minnow (*Pimephales promelas*) and *Daphnia magna*. Toxic potential = solubility/LC50 (**C** and **F**). The individual data points are averaged by log (K_{OW}) intervals. The mean value and the range are presented. Lines are computed using the regression-line slopes and intercepts given in Table 1. Data are from Di Toro et al. [6].

of components, the most straightforward assumption is that the mixture is an ideal mixture; therefore, the solubility of each component is described using Raoult's law [19]

$$C_{W,j} = x_j S_{L,j} \tag{16}$$

where x_j (mol/mol) is mole fraction of component j in the mixture and $S_{L,j}$ (mol/L) is the solubility of component j. For example, in an equimolar binary mixture, $x_j = 0.5$, and the concentration of each component would be at half the aqueous solubility of that component.

Hydrocarbon mixtures in general and oils in particular can exhibit departures from ideal behavior. This is accounted for by defining activity coefficients γ_j that correct Equation 16 appropriately [19]:

$$C_{W,j} = x_j \gamma_j S_j \tag{17}$$

Table 1. Regression line parameters

Species	*y* axis	Slope	Intercept	r^2
Narcotic chemicals				
Pimephales promelas	LC50[a] (mol/L)	−0.945[b]	−0.980	—
	Subcooled solubility (mol/L)	−1.096	0.677	0.945
	Toxic potential	−0.151[c]	1.65[b]	—
Daphnia magna	LC50 (mol/L)	−0.945[b]	−0.960	—
	Subcooled solubility (mol/L)	−1.092	0.631	0.956
	Toxic potential	−0.147[c]	1.59[b]	—
Polycyclic aromatic hydrocarbons				
Pimephales promelas	LC50 (mol/L)	−0.945[b]	−0.980	—
	Solid solubility (mol/L)	−1.323	1.048	0.882
	Toxic potential	−0.378[c]	2.03[b]	—
Daphnia magna	LC50 (mol/L)	−0.945[b]	−0.960	—
	Solid solubility (mol/L)	−1.571	2.093	0.880
	Toxic potential	−0.626[c]	3.05[b]	—

[a] LC50 = concentration lethal to 50% of organisms.
[b] Fixed at the universal narcosis slope.
[c] Computed as toxic potential = solubility − LC50 for both slope and intercept.

The γ_j values for the *Exxon Valdez* crude oil range from 0.71 to 3.72, with a median (standard deviation) of 1.46 (0.71). Thus, oil is nearly an ideal mixture, and the assumption that $\gamma_j = 1$ is not sufficiently inaccurate to invalidate the analysis presented below.

TOXICITY AND WEATHERING

To understand the consequences of weathering, consider the toxicity of a highly idealized oil, a binary mixture of benzene with concentration x_b (mol/mol) and phenanthrene with concentration x_p (mol/mol). The aqueous concentrations of benzene $C_{W,b}$ and phenanthrene $C_{W,p}$ are

$$C_{W,b} = x_b S_{L,b} \tag{18a}$$

$$C_{W,p} = x_p S_{L,p} \tag{18b}$$

where $S_{L,b}$ and $S_{L,p}$ are the aqueous solubility and subcooled solubility of benzene and phenanthrene, respectively. The toxicity of the mixture is

$$\mathrm{TU} = \frac{C_{W,b}}{C^*_{W,b}} + \frac{C_{W,p}}{C^*_{W,p}} \tag{19}$$

and substituting Equations 18a and 18b for the aqueous concentrations yields

$$\mathrm{TU} = \frac{x_b S_{L,b}}{C^*_{W,b}} + \frac{x_p S_{L,p}}{C^*_{W,p}} \tag{20}$$

Using the definitions of toxic potential (Eqn. 8) yields

$$\mathrm{TU} = x_b \mathrm{TU}^b_{W,max} + x_p \mathrm{TU}^p_{W,max} \tag{21}$$

where the superscripts b and p also denote benzene and phenanthrene, respectively. Thus, the toxicity of the binary mixture is sum of the toxic potential of each component weighted by its mole fraction in the oil. Note that this result can be generalized to a *N*-component mixture, such as petroleum:

$$\mathrm{TU} = \sum_{j=1}^{N} x_j \mathrm{TU}^j_{W,max} \tag{22}$$

For the two-component mixture being considered, the two mole fractions must sum to one ($x_b + x_p = 1$). Therefore, $x_p = 1 - x_b$, and the toxicity is

$$\mathrm{TU} = x_b \mathrm{TU}^b_{W,max} + (1 - x_b)\mathrm{TU}^p_{W,max} \tag{23}$$

Consider the situation in which benzene initially comprises almost all the oil (e.g., an unweathered oil). Then, $x_b \cong 1$, $x_p = 1 - x_b \cong 0$, and the initial toxicity is

$$\mathrm{TU}_{initial} \cong \mathrm{TU}^b_{W,max} \tag{24}$$

When weathering is complete, benzene has evaporated completely ($x_b \cong 0$), leaving behind phenanthrene, which now comprises the entire oil mixture ($x_p \cong 1$). Comparing the toxicity of the final oil to the initial oil, the result is that

$$\mathrm{TU}_{final} \cong \mathrm{TU}^p_{W,max} < \mathrm{TU}^b_{W,max} \cong \mathrm{TU}_{initial} \tag{25}$$

The final toxicity is less than the initial toxicity, because the toxic (potential of phenanthrene ($\mathrm{TU}^p_{W,max}$) is less than the toxic potential of benzene ($\mathrm{TU}^b_{W,max}$).

This is the key to understanding the effect of weathering. Weathering removes the more volatile, lower-molecular-weight components of the oil mixture, which are the components with the higher toxic potentials, and it leaves behind the higher-molecular-weight components with lower toxic potentials.

LC50s AND TOTAL PETROLEUM HYDROCARBON CONCENTRATIONS

From the analysis presented above, an aqueous phase in equilibrium with oil containing mostly low-K_{OW} components (e.g., benzene) clearly is more toxic than an aqueous phase in equilibrium with oil containing mostly high K_{OW} components (e.g., phenanthrene). Toxicity in this case is judged by the concentration of TUs in the aqueous phase: The higher the TU concentration, the higher the toxicity of the aqueous phase. Another measure, however, also can be used to evaluate the toxicity of the aqueous phase—namely, its volume.

Water-soluble fraction LC50

An equivalent measure of toxicity is the volume fraction of the aqueous phase that causes a 50% effect. Consider a sample of oil that is equilibrated with water to form the water-soluble fraction (WSF). A series of volume fractions f_{WSF} (e.g., 1, 2, 5, . . . , 50, and 100%) are tested, and the fraction f^*_{WSF} that produces the 50% effect is determined. For convenience, denote this volume fraction as LC50(WSF) = f^*_{WSF}. The smaller the LC50(WSF), the more toxic the WSF, because it takes a smaller volume fraction to produce the effect.

The LC50(WSF) can be computed using TUs. Let TU_{WSF} be the TU concentration of the WSF. Then, the toxicity of a volume fraction f_{WSF} is $\mathrm{TU}_{WSF} f_{WSF}$. The LC50 occurs when the TU concentration equals one, so

$$\mathrm{TU}_{WSF} f^*_{WSF} = 1 \tag{26}$$

where f^*_{WSF} is the volume fraction LC50 and

$$\mathrm{LC50(WSF)} = f^*_{WSF} = \frac{1}{\mathrm{TU}_{WSF}} \tag{27}$$

Therefore, the toxicity of a WSF also can be correctly evaluated using the LC50(WSF) volume fraction. It is the inverse of the TU concentration of the WSF.

Total petroleum hydrocarbon LC50

Traditionally, the toxicity of an aqueous phase in equilibrium with an oil sample has been related to the sum of the concentrations of the components in the aqueous phase without

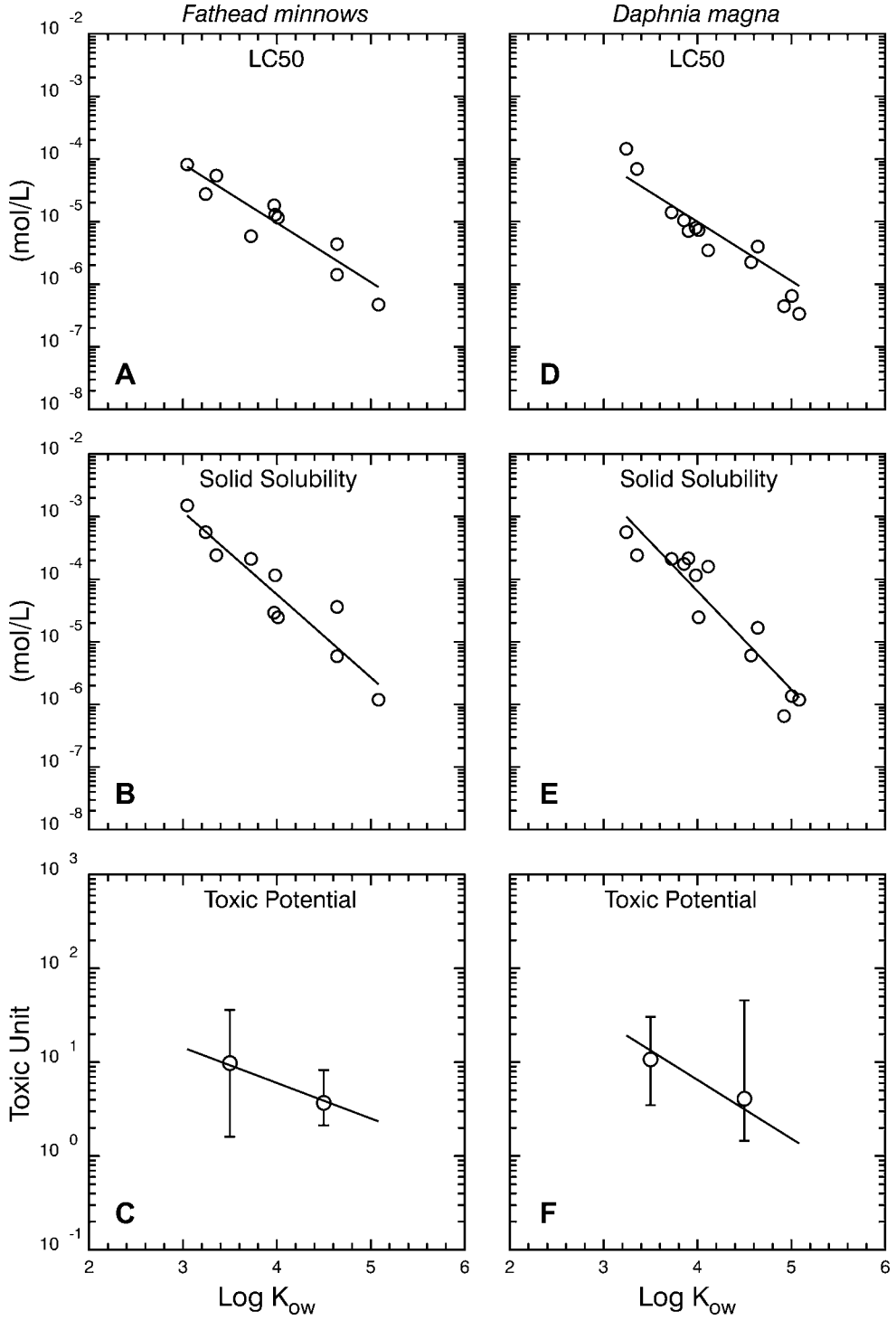

Fig. 2. Paired lethal concentrations to 50% test organisms (LC50s; **A** and **D**) and solid solubility (**B** and **E**) versus log (K_{ow}) for polycyclic aromatic hydrocarbons (PAHs) for fathead minnow (*Pimephales promelas*) and *Daphnia magna*. Toxic potential = solubility/LC50 (**C** and **F**). The individual data points are averaged by log (K_{ow}) intervals. The mean value and range presented. Lines are computed using the regression-line slopes and intercepts given in Table 1. Data are from Di Toro et al. [6].

regard to their identity. For example, one could sum the concentrations of all the components on a concentration (mg/L) basis and refer to the concentration of total petroleum hydrocarbons (TPHs). Then, the toxicity of the aqueous phases in equilibrium with the oil samples would be judged in terms of the TPH LC50, denoted by LC50(TPH).

Unfortunately, this procedure is completely misleading in general, because it is natural to assume that the lower the LC50(TPH), the more toxic the mixture. However, this is not necessarily the case. A lower LC50(TPH) does not necessarily correspond to a more toxic mixture, because the composition of the mixture can change from less toxically potent to more toxically potent components. This change is not necessarily reflected in the TPH concentration, because TPH is a sum of concentrations without regard to their identities or toxicities.

To demonstrate how misleading the LC50(TPH) is, this

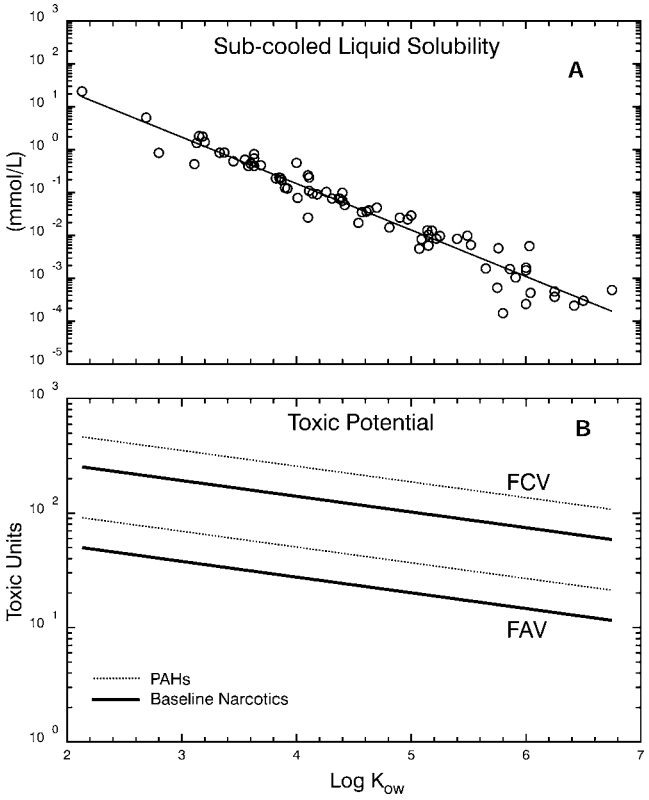

Fig. 3. (**A**) Sub-cooled liquid solubilities versus log (K_{OW}) for 66 aromatics and 11 alkanes [15–17]. (**B**) Toxic potential versus log (K_{OW}) (Eqns. 10–12) for baseline narcotics and polycyclic aromatic hydrocarbons (PAHs) for final acute value (FAV) and final chronic value (FCV) end points.

value is computed below using the model equations derived above. For the benzene–phenanthrene case, the result, derived in the *Appendix,* is

$$\text{LC50(TPH)}_{\text{final}} = C^*_{\text{W,p}} < C^*_{\text{W,b}} = \text{LC50(TPH)}_{\text{initial}} \quad (28)$$

That is, LC50(TPH) of the final aqueous phase (i.e., the aqueous phase in equilibrium with the weathered oil) is less than the LC50(TPH) of the aqueous phase in equilibrium with the nonweathered oil. As demonstrated above, however, the initial WSF, saturated with benzene, is more toxic than the final WSF, saturated with phenanthrene. So, although the final LC50(TPH) is, in fact, less that the initial LC50(TPH), it is incorrect to conclude that the final WSF is more toxic than the initial WSF. In fact, just the opposite is true.

Therefore using the LC50(TPH) is completely misleading. It is true that weathering lowers the LC50(TPH), but it is not true that as a consequence, the toxicity of the WSF increases. The misunderstanding that weathering increases toxicity result from use of the LC50(TPH) and to interpret the TPH, or total PAH concentration, as if it were the concentration of one toxic chemical. If TPH were a specific toxic chemical, then a lower LC50 would imply increased toxicity. However, TPH is not a specific toxic chemical; therefore, it is entirely inappropriate for use as a measure of toxicity

This problem arises in three recent papers regarding oil toxicity, two discussing weathering [1,20] and one discussing volatilization [2]), in which it is incorrectly concluded that the loss of the lighter, low-K_{OW} material and the subsequent increase in the heavier, high-K_{OW} material in the aqueous phase

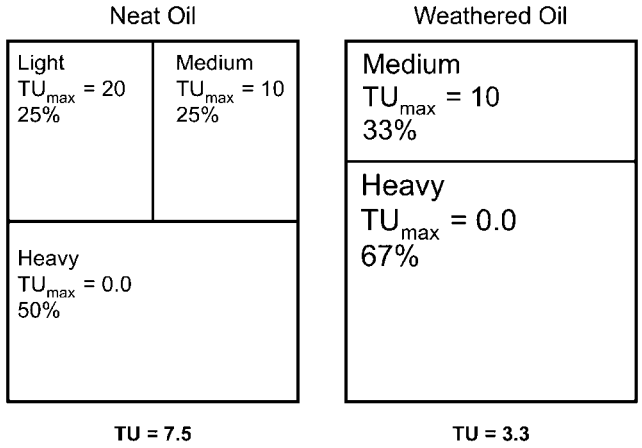

Fig. 4. An example of the composition change in fresh and weathered oils. The light, medium, and heavy fractions are considered. The toxic potential is TU_{max}, and the weight percentages are given. The resulting aqueous toxic units (TUs) are listed.

results in an increased toxicity of the mixture. The data from the first of these papers will be examined in more detail below.

WEATHERING DECREASES TOXICITY

Computational example

An example computation of the effect of weathering on toxicity is presented in Figure 4. It is assumed initially that the fresh oil is composed of 25% (mol fraction) low-K_{OW} compounds, 25% intermediate-K_{OW} compounds, and 50% heavy-molecular-weight compounds. The toxic potentials are assumed to be TU_{max} = 20, 10, and 0, respectively, for the three representative classes. The aqueous-phase toxicity of the WSF is the weighted sum of the toxic potential and the mole fraction in the mixture (Eqn. 22): TU = 20(0.25) + 10(0.25) + 0(.50) = 7.5. Weathering is assumed to remove all the light fraction. The resulting oil mole fractions change in response to this loss. The intermediate component increases from 0.25 to 0.33 mole fraction. Thus, more of that fraction will dissolve into the aqueous phase. The aqueous-phase toxicity that results is TU = 10(0.33) + 0(0.67) = 3.3. Despite the increase of the contribution of the intermediate phase from $TU_{intermediate}$ = 10(0.25) = 2.5 to $TU_{intermediate}$ = 10(0.33) = 3.3, the loss of the more toxic, lighter-phase contribution TU_{light} = 20.(0.25) = 5 causes the toxicity to be reduced from $TU_{initial}$ = 7.5 to TU_{final} = 3.3.

The most straightforward explanation of the reduction in toxicity is to realize that weathering removes the more toxically potent chemicals, which are replaced with less toxically potent chemicals. The replacement occurs on a mole fraction basis—for example, the increase of 25 to 33% of the intermediate-log (K_{OW}) compounds in the above example. The inevitable result is a decrease in toxicity.

The discussion and hypothetical example presented above are intended to explain and clarify the mechanisms of toxicity reduction by weathering. In the following sections, data from three toxicity experiments and one oil spill are discussed to validate these ideas.

WSF toxicity to D. magna

The toxicity of three oils at various stages of weathering was determined by Bobra et al. [13] using laboratory exposures to *D. magna*. The oils were weathered by partial evaporation. The results (Fig. 5) demonstrate that the LC50(TPH) does,

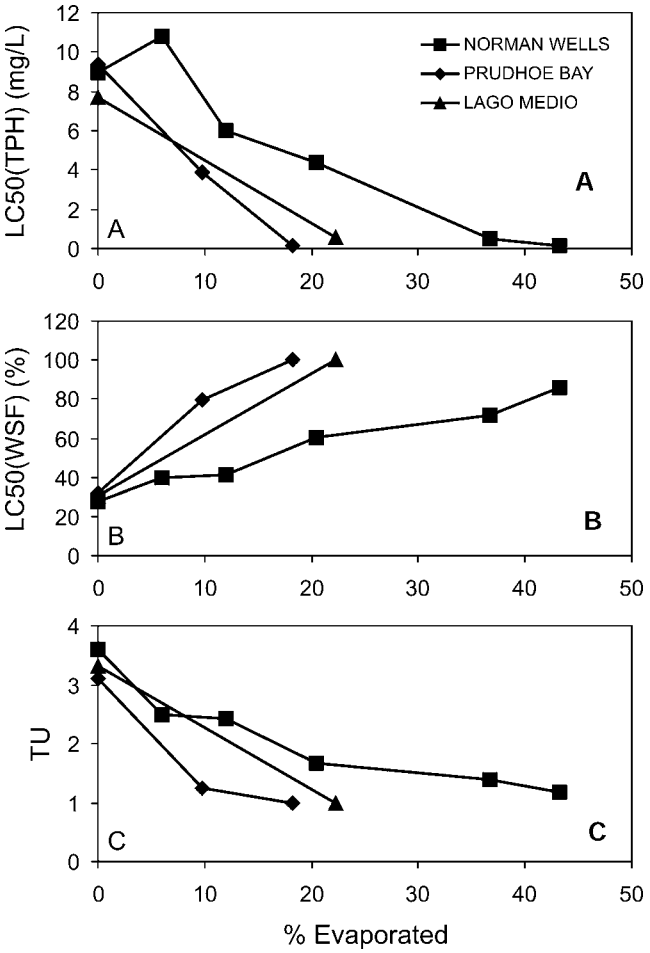

Fig. 5. Lethal concentration to 50% test organisms (LC50) on a total petroleum hydrocarbon basis (**A**), LC50 as a percentage of the aqueous phase (**B**), and toxic unit (TU) concentration (**C**) versus percentage of the oil evaporated [13]. Norman Wells, Prudhoe Bay, and Lago Medio were the crude oils used in the toxicity tests. TPH = total petroleum hydrocarbon; WSF = water-soluble fraction.

indeed, decrease as weathering increases, as expected (Eqn. 28). However, the toxicity is actually decreasing. The LC50(WSF) is increasing; therefore, the TU concentration is decreasing. In fact, at a certain point in the weathering sequence, the aqueous phase is no longer toxic, and the LC50s could no longer be determined (LC50(WSF) $>$ 100%, TU $<$ 1). Thus, weathering reduced the toxicity until the mixture could no longer cause mortality even though the LC50(TPH) was decreasing (Fig. 5A). This experiment clearly points out the fallacy of using LC50 values that are based on total hydrocarbon concentrations to judge toxicity.

Exxon Valdez *WSF toxicity to* P. promelas

The toxicity of WSFs of neat (unweathered) and naturally weathered *Exxon Valdez* Alaska (USA) North Slope crude oil to *P. promelas* was evaluated as follows: Neat oil was collected from the *Exxon Valdez* oil tanker 7 d after the tanker ran aground in Prince William Sound (AK, USA). Naturally weathered oil was collected approximately five months after the tanker grounded. Water-soluble fractions were prepared from 10:1 (water:oil) solutions for the neat and weathered oils. Each WSF was analyzed for BTEX (benzene, toluene, ethylbenzene, and xylene), biphenyl, 19 parent PAHs, and 21 alkylated homologues of parent PAHs. A listing of the compounds is provided in Table 2. Although dibenzothiophene is a heterocyclic compound that contains sulfur and is not considered to be a type I narcotic compound [3], it and its alkylated homologues were included in the analysis for completeness, but they were not predicted to exert any significant toxicity. Six dilutions of the WSFs were made for use in toxicity testing. The measured concentrations for each chemical were converted to TUs (Eqns. 2 and 14) using the critical body burden for fathead minnows [6] (Table 2). The TU of the WSF was computed by summing the TUs of each component in the mixture (Eqn. 15).

The measured total concentration in the undiluted WSF from the neat oil was approximately 19.8 mg total measured compounds/L, of which greater than 98% were from the BTEX compounds. In comparison, the measured total concentration in the undiluted WSF from the weathered oil was 0.78 mg total measured compounds/L, of which 67% were from the BTEX compounds. The decrease in concentration results from the loss of the more soluble components, particularly the light BTEX compounds, through weathering.

The TUs associated with the undiluted WSFs from the neat and weathered oils separated into BTEX (log K_{OW} = 2–3.2) and log (K_{OW}) classes (low: $<$4.5; mid: 4.5$-$6; high: $>$6) are shown in Figure 6A. The BTEX component accounts for 67% of the toxicity in the neat oil WSF, compared to 11% of the toxicity in the weathered oil WSF. Therefore, BTEX compounds are important contributors to the toxicity of neat oil. As the oil is weathered, the lighter fraction is removed, and the heavier fraction comes into solution. The TUs from the heavier material (mid- and high-K_{OW} fractions in Fig. 6A) are higher in the weathered sample than in the nonweathered sample. However, these compounds have lower toxic potentials and, therefore, exert less toxicity. Therefore, the total toxicity of the oil sample decreases with weathering. The predicted total TUs in the WSFs decrease from the neat oil (TU = 0.72) to weathered oil (TU = 0.29), suggesting that the neat oil is approximately 2.5-fold more toxic compared with the weathered oil. Figure 6B presents the percentage mortality versus TUs that confirms the predictions of the TLM. The neat oil WSF (predicted TU = 0.72) was observed to be toxic, and the weathered oil (predicted TU = 0.29) was observed to be nontoxic.

Sediment toxicity to Ampelisca abdita

The third example is a field monitoring study investigating the effects of an oil spill that occurred in January 1996 from the North Cape barge [21]. Approximately 3,000,000 L of No. 2 fuel oil were spilled into Rhode Island Sound near Matunuck (RI, USA). The concentrations of PAHs at two locations in the Harbor of Refuge were followed for nine months after the spill. At these two sites, samples of sediment were collected for toxicity testing, and 96-h sediment bioassays were performed using the amphipod *A. abdita*.

The concentrations of 33 PAHs were measured in the sediments at 2, 6, 13, 33, 62, 132, 189, and 270 d postspill. The measured compounds included 17 parent PAHs and 16 alkylated homologues of parent PAHs. A listing of the specific PAHs is provided in Table 2. The sediment effect concentrations were computed using Equation 5, in which the $C^*_{L,i}$ for *A. abdita* is 12.2 μmol/g octanol [7]. The individual effect concentrations on an OC basis for the PAHs are similar (the reason has been discussed previously by Di Toro and McGrath [7]) and range from 8.9 to 12 μmol/g OC (Table 2). The

Table 2. Specific compounds and their effect concentrations used in laboratory and field examples[a]

Chemical	Molecular weight (g/mol)	Log K_{OW}	Chemical class correction	*Promelas promelas* FAV used in neat and weathered oil analysis, LC50 96-h (mmol/L)[b]	*Ampelisca abdita* C_S^* used in oil spill analysis (μmol/g OC)[c]
Benzene	78.11	2.00	0	1.35E+00	—
Toluene	92.14	2.62	0	3.51E−01	—
Ethylbenzene	106.17	3.06	0	1.35E−01	—
m+*p* Xylene	106.17	3.20	0	9.94E−02	—
o-Xylene	106.17	3.13	0	1.16E−01	—
Biphenyl	154.21	3.97	0	1.88E−02	—
Naphthalene	128.2	3.36	−0.263	3.86E−02	8.9
C1-naphthalenes	142.2	3.80	−0.263	1.47E−02	9.3
C2-naphthalenes	156.2	4.20	−0.263	6.15E−03	9.6
C3-naphthalenes	170.3	4.66	−0.263	2.26E−03	10.0
C4-naphthalenes	184.3	5.10	−0.263	8.68E−04	—
Acenaphthylene	152.2	3.45	−0.263	3.15E−02	—
Acenaphthene	154.2	3.84	−0.263	1.35E−02	—
Fluorene	166.2	4.21	−0.263	6.05E−03	9.6
C1-fluorenes	180.3	4.72	−0.263	1.99E−03	10.1
C2-fluorenes	194.3	5.20	−0.263	6.99E−04	10.5
C3-fluorenes	208.3	5.70	−0.263	2.35E−04	11.0
Anthracene	178.2	4.53	−0.263	3.00E−03	9.9
Phenanthrene	178.2	4.57	−0.263	2.75E−03	9.9
C1-phenanthrenes/antracenes	192.3	5.04	−0.263	9.98E−04	10.4
C2-phenanthrenes/antrhacenes	206.3	5.46	−0.263	3.97E−04	10.7
C3-phenanthrenes/anthracenes	220.3	5.92	−0.263	1.46E−04	11.2
C4-phenanthrenes/anthracenes	234.3	6.32	−0.263	6.11E−05	11.6
Dibenzothiophene	184.2	4.53	−0.263	3.00E−03	9.9
C1-dibenzothiophene	198.3	4.96	−0.263	1.18E−03	10.3
C2-dibenzothiophene	212.3	5.42	−0.263	4.33E−04	10.7
C3-dibenzothiophene	226.3	5.89	−0.263	1.56E−04	11.2
Fluoranthene	202.3	5.08	−0.263	8.99E−04	10.4
Pyrene	202.3	4.92	−0.263	1.28E−03	10.2
C1-fluoranthenes/pyrenes	216.3	5.48	−0.263	3.80E−04	10.8
C2-fluoranthenes/pyrenes	230.3	6.15	−0.263	8.84E−05	—
C3-fluoranthenes/pyrenes	244.3	6.60	−0.263	3.32E−05	—
Benzo[*a*]anthracene	195.0	5.89	−0.263	1.56E−04	11.2
Chrysene	228.3	5.71	−0.263	2.29E−04	11.0
C1-chrysenes	242.3	6.14	−0.263	9.03E−05	11.4
C2-chrysenes	256.3	6.43	−0.263	4.82E−05	11.7
C3-chrysenes	270.4	6.94	−0.263	1.58E−05	—
C4-chrysenes	284.4	7.36	−0.263	6.35E−06	—
Benzo[*b*]fluoranthene	252.3	6.27	−0.263	6.87E−05	11.5
Benzo[*k*]fluoranthene	252.3	6.29	−0.263	6.50E−05	11.6
Benzo[*e*]pyrene	252.3	6.44	−0.263	4.70E−05	11.7
Benzo[*a*]pyrene	252.3	6.11	−0.263	9.64E−05	11.4
Perylene	252.3	6.44	−0.263	4.70E−05	11.7
Indeno[1,2,3-*c,d*]pyrene	276.3	6.72	−0.263	2.55E−05	12.0
Dibenz[*a,h*]anthracene	278.4	6.71	−0.263	2.60E−05	12.0
Benzo[*ghi*]perylene	276.3	6.51	−0.263	4.07E−05	11.8

[a] C_S^* = critical sediment concentration; FAV = final acute value; LC50 = concentration lethal to 50% of organisms; OC = organic carbon; — = not measured.

[b] Log FAV = (−0.945)(log K_{OW}) + log C_L^* + Δc, where C_L^* = 105 μmol/g octanol = critical target lipid body burden (CTLBB) for *P. promelas*.

[c] Log C_S^* = 0.00028 + 0.038 log (K_{OW}) + log C_L^* + Δc, where C_L^* = 12 μmol/g octanol = CTLBB for *A. abdita*; Δc = 0, because toxicity data used to derive CTLBB were for polycyclic aromatic hydrocarbons.

sediment TU for each compound was computed by dividing the measured concentration normalized to OC by the specific effect concentration (Eqn. 5). The OC concentration ranged from approximately 0.2 to 2.6%, with an average of 0.6%, at station 1, and from 0.5 to 2%, with an average of 1.3%, at station 2 over the monitoring period (J. Latimer, U.S. EPA, Narragansett, RI, personal communication). The total TUs for each sample were then computed by summing the TUs of the individual components.

The total TUs in sediments from stations 1 and 2 as a function of time are shown in Figure 7A. At the both stations, the total TUs initially increased to a peak level of 9.3 and 22, respectively, and then declined with time. Nine months after the spill, the TUs were less than 1.0 in both sediments, which demonstrates that as the oil weathers naturally over time in the sediment, its toxicity decreases. The filled and unfilled symbols in Figure 7A correspond to those sediment samples for which greater than or less than 50% mortality, respectively, to *A. abdita* was observed. In each case, the TLM correctly predicted that mortality would (TU > 1) or would not (TU < 1) occur.

As in the previous examples, the decrease in toxicity with time results from loss of the more soluble components. The fractional TUs associated with low (<4.5)-, mid (4.5–6)-, and

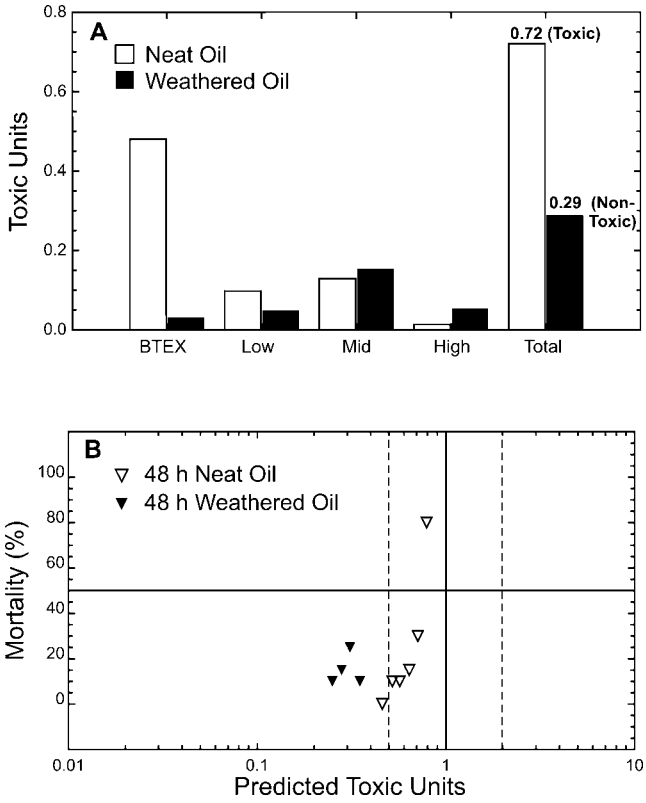

Fig. 6. (**A**) Comparison of toxic units from undiluted water-soluble fractions (WSFs) of neat (open bars) and weathered (filled bars) Alaska North Slope crude oil. The toxic units are shown for BTEX (benzene, toluene, ethylbenzene, and xylene) compounds and low (log (K_{OW}) < 4.5), mid (log (K_{OW}) 4.5–6.0), and high (log (K_{OW}) > 6.0), K_{OW} polycyclic aromatic hydrocarbons (PAHs). Also shown are the total toxic units from all compounds measured. (**B**) The 48-h mortality to fathead minnow (*Pimephales promelas*) as a function of predicted total toxic units for neat oil (open triangle) and weathered oil (filled triangles).

high (>6.0)-log (K_{OW}) compounds are shown in Figure 7B and C for stations 1 and 2, respectively. As the oil is weathered, the lighter log (K_{OW}) fraction is removed; its mole fraction in the oil is decreasing. This is observed in sediments from both stations, where the lighter fraction initially accounts for approximately 20 to 25% of the total TU and then decreases to the point at which it has no toxicological significance. The TUs from the heavier log (K_{OW}) material increases as the material is weathered because of an increase in mole fraction. The toxic contribution from the mid-log (K_{OW}) compounds is relatively constant, ranging from 75 to 90% in the oil. In these sediments, the lighter material with greater toxic potential is removed via natural weathering and is being replaced by the heavier material with less toxic potential. Therefore, the total toxicity of the sediments decreases with weathering.

Chronic toxicity to Clupea pallasi

Carls et al. [1] computed concentrations affecting 50% of organisms (EC50s) for various biological responses in Pacific herring (*C. pallasi*) based on total PAH concentration (μg TPAH/L) for less weathered (LWO) and more weathered (MWO) crude oils (Fig. 8A). In this experiment, the exposure waters were generated by passing water through contaminated gravel. To simulate a weathering effect, the gravel from the LWO exposures was reused in the MWO exposures. With the exception of egg death, the EC50s expressed as μg TPAH/L

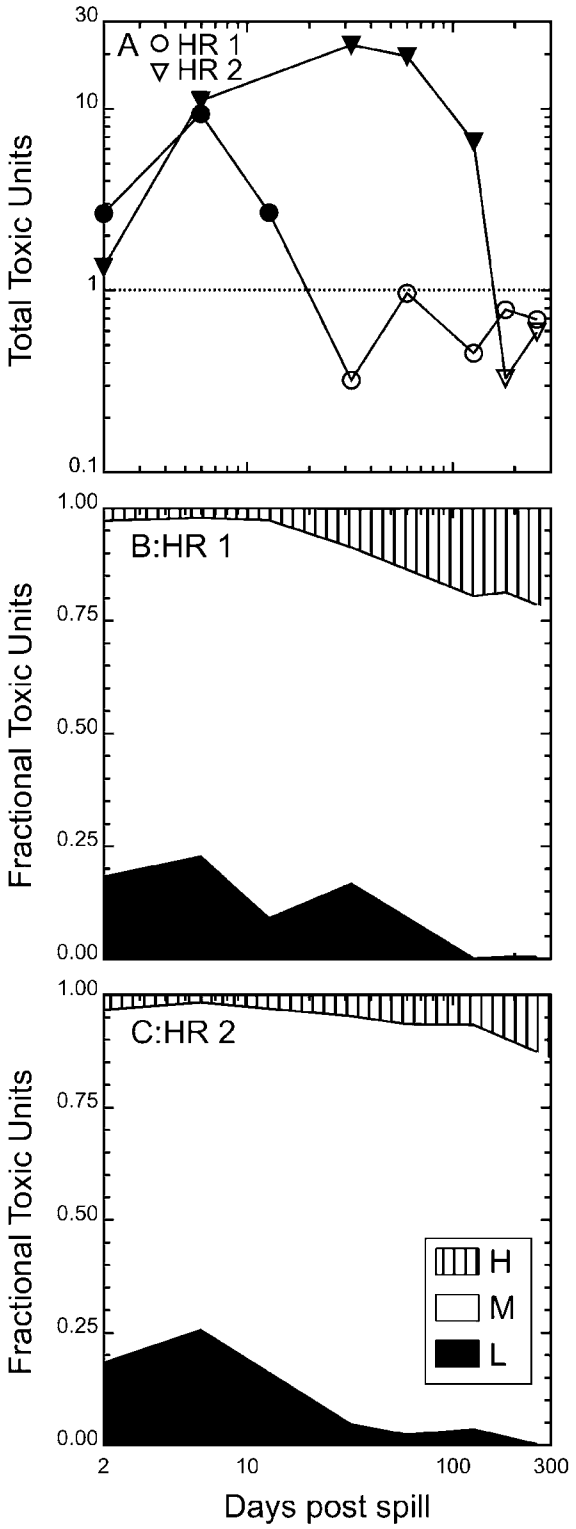

Fig. 7. Concentration of total polycyclic aromatic hydrocarbons (PAHs) normalized to toxic units (TUs) collected in sediment samples at stations HR 1 and HR 2 as a function of time after the North Cape oil spill [21]. Total TUs units (**A**) are shown, as are fractional TUs from PAHs with low (L; <4.5), mid (M; 4.5–6.0), and high (H; > 6.0) log (K_{OW}) values in samples collected at station HR 1 (**B**) and station HR 2 (**C**). In (**A**), filled symbols indicate greater than 50% mortality was observed in bioassays, and unfilled symbols indicate less than 50% mortality. Dashed line indicates TU = 1.

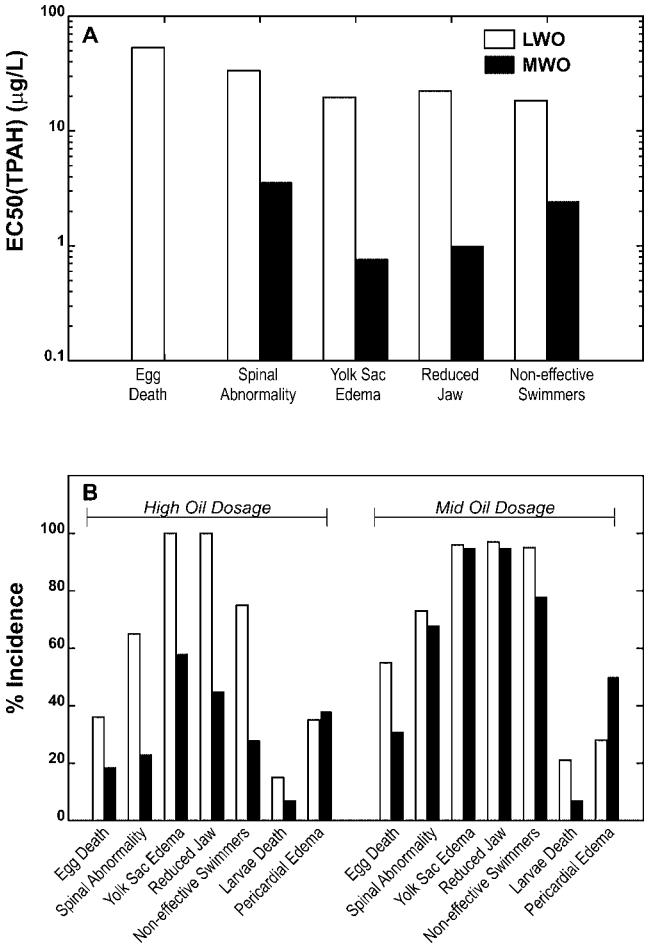

Fig. 8. Comparison of concentrations affecting 50% of test organisms (EC50s; μg total polycyclic aromatic hydrocarbons [PAH]/L; **A**) and percentage incidences (**B**) for various biological responses from less weathered oil (LWO) and more weathered oil (MWO). Data shown are for the high- and mid-level oil treatment [1].

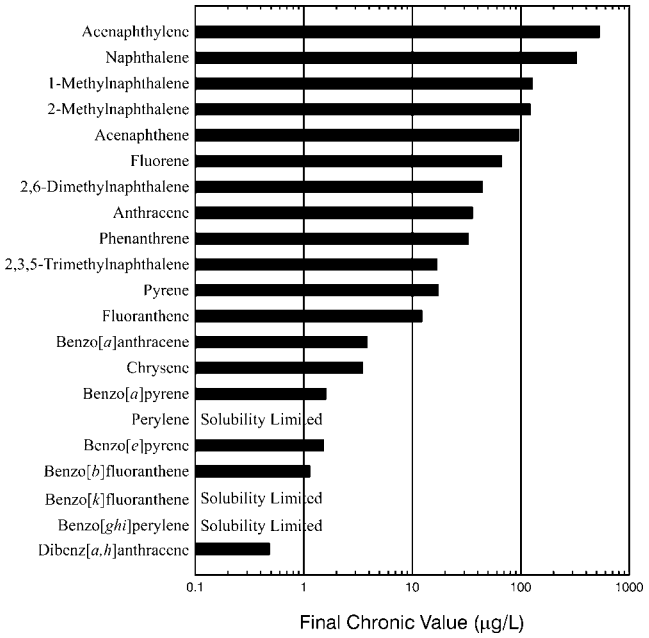

Fig. 9. Target lipid model final chronic values for various polycyclic aromatic hydrocarbons [6].

for the effects from the MWO treatments are lower. For egg death, an EC50 from the MWO treatment could not be calculated because of insufficient response, presumably as a result of the decrease in toxicity.

Because the EC50s for MWO are lower than the EC50s for LWO, it appears that weathering increases toxicity, which was the conclusion offered by those authors. However, a comparison of the biological responses from the two highest oil doses (Fig. 8B) shows that the percentage incidences of the biological effects are lower for the MWO—the single exception is a slight increase in pericardial edema. This clearly demonstrates that the MWO is less toxic than the LWO. The incorrect conclusion offered by the authors—that weathering increases toxicity—was caused by the incorrect use of total PAH EC50s to judge toxicity.

Conclusion

The data in these examples clearly demonstrate that the observed toxicity decreases as the degree of weathering increases. The fraction of the more soluble, lower-molecular-weight components with higher toxic potential is removed, allowing the fraction of the less soluble, higher-molecular-weight components with lower toxic potential to increase. Therefore, the toxicity of the material as a whole decreases. The data from these examples also demonstrate that the toxicity of complex mixtures of petroleum, both in aqueous and sediment samples, can be correctly predicted using the TLM, TU additivity, and for sediments, the equilibrium partitioning model [11].

RESIDUAL AQUEOUS PAH CONCENTRATIONS AND A "NEW APPRECIATION" FOR OIL TOXICITY

In a recent paper by Peterson et al. [22], a ''new emerging appreciation for oil toxicity'' is offered that claims oil is toxic to fish at PAH levels in the ppb (μg/L) range. Supporting evidence for these low concentrations is from field assessments and laboratory studies conducted after the *Exxon Valdez* oil spill that showed long-term chronic effects from exposure to PAHs were caused by the less soluble, heavier, more persistent PAHs. Peterson et al. compared this emerging appreciation to the old paradigm, in which oil toxicity was based on short-term acute toxicity tests using more soluble, more volatile, lighter PAHs and for which mortality occurred in the ppm (mg/L) range.

This new appreciation for aquatic toxicity from oil, however, is not new at all. The TLM presented by Di Toro et al. [6] included FCVs for individual PAHs that are reproduced in Figure 9. These ranged from 528 μg/L for acenaphthylene to 0.48 μg/L for dibenzo[*a,h*]anthracene. These FCVs are the concentrations of the individual PAHs that would be predicted to cause long-term effects. As discussed previously, the predicted toxicity varies as a function of log (K_{OW}) such that toxicity increases with increasing log (K_{OW}). Therefore, the heavier PAHs have lower FCVs, and for the very heavy PAHs that have a log (K_{OW}) greater than 6.0, the FCVs are in the range of 1 μg/L. Note that some PAHs (i.e., perylene, benzo[*k*]fluoranthene, and benzo[*ghi*])perylene) are not predicted to be toxic by themselves, because their predicted FCV is above their water solubility. These PAHs would contribute to the total toxicity when present in liquid mixtures, however, because of their lower subcooled liquid solubility.

The main difference between the paradigm of Peterson et al. [22] and the TLM [6,7] is the normalization of the aquatic

toxicity of PAHs in mixtures. In the Peterson et al. paradigm, the aquatic toxicity of three- to five-ringed PAHs is assumed to be the same. This is incorrect, however, because it assumes that the aquatic toxicity of the individual PAHs is similar. If the PAH of concern is the three-ringed phenanthrene (log (K_{OW}) = 4.57), then a concentration of 1 μg/L would not be chronically toxic. In the TLM, the conversion of the individual PAHs concentrations to TUs normalizes the differences in aquatic toxicity. Based on the TLM, it is not unexpected that chronic effects are observed for PAH concentrations near 1 μg/L, when the heavier PAHs are present. For low levels of residual oils, the heavier PAHs may be present at ng/L to μg/L levels. Each PAH will contribute to the toxicity. For example, if benzo[*a*]pyrene (FCV = 1.6 μg/L) is present at 0.2 μg/L, it would contribute 0.125 TU. By itself, benzo[*a*]pyrene would not be toxic, but if the same sample also contained seven more PAHs, each with a similar log (K_{OW}) at a similar concentration, then the sample would be predicted to be toxic.

This ''new appreciation'' is based on the same misunderstanding that leads to the incorrect conclusion that weathering increases toxicity. The toxicity of the sample depends on which PAHs are present. For example, assigning a total PAH concentration of 1 μg/L as toxic is meaningless, because the toxicity of individual PAHs varies. Converting the concentrations to TUs normalizes the differences in toxicity. A concentration of 1 TU from total PAHs present in the sample implies toxicity. A concentration of 1 μg/L of total PAHs can be either toxic or nontoxic; no judgment is possible.

CONCLUSIONS

The toxicity of the components of oil can be understood using the concept of toxic potential. As log (K_{OW}) increases, so does the toxicity—but the solubility decreases more rapidly. Therefore, the toxic potential (i.e., the ratio of solubility to toxicity) decreases. Thus, lower log (K_{OW}) compounds are more toxically potent than higher log (K_{OW}) chemicals. Weathering removes the lower log (K_{OW}) chemicals from the oil; they are replaced, in a mole fraction sense, with higher log (K_{OW}) chemicals. The replacement of more toxically potent compounds with less toxically potent compounds lowers the toxicity.

The idea that weathering increases toxicity is based on the erroneous use of the TPH concentration as though it were a single chemical compound. If TPH were a single chemical, then a comparison of its LC50 concentration among various weathered and nonweathered oils is rational. Because the identity of the compounds making up TPH is changing, however, comparisons of LC50s are completely misleading and can lead to wrong conclusions—in particular, that weathering increases toxicity. The use of toxic potential and TUs eliminates this confusion, puts all the chemicals on the same footing, and allows an intuitive understanding of the effects of weathering.

The ''new emerging appreciation'' of PAH toxicity is not new at all. The FCVs for the heavier PAHs are expected to be in the range of 1 μg/L. It is incorrect, however, to assume that 1 μg/L of total PAHs would be chronically toxic; it depends entirely on the composition of the PAHs making up the mixture. Therefore, total hydrocarbon or total PAH concentration should not be used to quantify the toxicity of a mixture of PAHs. Rather, TUs should be used to normalize the different aqueous toxicity from the different PAHs present in mixtures.

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APPENDIX

This appendix discusses derivation of the LC50(TPH). All abbreviations and terms are as defined in the text.

The TPH concentration is calculated as

$$\mathrm{TPH} = C_{\mathrm{W,b}} + C_{\mathrm{w,p}} = x_{\mathrm{b}} S_{\mathrm{L,b}} + x_{\mathrm{p}} S_{\mathrm{L,p}} \tag{A1}$$

Clearly, the initial total concentration is larger than the final concentration, because benzene is much more soluble than phenanthrene. Thus,

$$\mathrm{TPH}_{\mathrm{initial}} = S_{\mathrm{L,b}} > S_{\mathrm{L,p}} = \mathrm{TPH}_{\mathrm{final}}$$

To calculate the LC50(TPH) values, consider the initial WSF. The volume fraction required to produce a 50% effect requires a dilution of $1/\mathrm{TU}_{\mathrm{intiial}}$ (Eqn. 27). The LC50 based on the concentration of benzene ($\mathrm{TU}_{\mathrm{initial}}$) in the initial WSF and the volume fraction that produces toxicity ($1/\mathrm{TU}_{\mathrm{initial}}$; Eqn. 24) is

$$\mathrm{LC50(TPH)}_{\mathrm{initial}} = \frac{\mathrm{TPH}_{\mathrm{initial}}}{\mathrm{TU}_{\mathrm{initial}}} = \frac{S_{\mathrm{L,b}}}{\mathrm{TU}_{\mathrm{initial}}}$$

$$= \frac{S_{\mathrm{L,b}}}{S_{\mathrm{L,b}}/C^{*}_{\mathrm{W,b}}} = C^{*}_{\mathrm{W,b}} \tag{A3}$$

This is expected, because the definition of $C^{*}_{\mathrm{W,b}}$ is the LC50 for benzene. Therefore, the LC50 of the initial aqueous phase (i.e., the solution containing almost entirely benzene) is the LC50 for benzene.

Similarly the final LC50 is

$$\mathrm{LC50(TPH)}_{\mathrm{final}} = C^{*}_{\mathrm{W,p}} \tag{A4}$$

which is the LC50 for phenanthrene. Because phenanthrene has a lower LC50 than benzene, the result is

$$\mathrm{LC50(TPH)}_{\mathrm{final}} = C^{*}_{\mathrm{W,p}} < C^{*}_{\mathrm{W,b}} = \mathrm{LC50(TPH)}_{\mathrm{initial}} \tag{A5}$$

and the final LC50(TPH) is less than the initial LC50(TPH).