The Aquatic Toxicity of Dissolved Hydrocarbon Gases and the Effect of High Pressure on Narcosis

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Abstract

Releases of oil and gas to aquatic environments may result in exposure of aquatic organisms to petroleum hydrocarbons. Methods for evaluating potential toxicity arising from exposure to the liquid hydrocarbons are well developed. The target lipid model (TLM) and toxic unit approach assume that each hydrocarbon acts via narcosis and the toxicity is additive. In the case of releases from oil and gas operations in the deep sea, dissolved hydrocarbon gases may be present. The TLM has not been validated for hydrocarbon gases. A complicating factor is the marked increase in the aqueous solubility of hydrocarbon gases as hydrostatic pressure increases with increasing depth. Thus, the exposure of aquatic organisms to dissolved phase gases is also expected to increase with increasing depth. Further, elevated pressure has been shown to reverse narcotic effects on biota (including bacteria, mammals and aquatic organisms). Pressures required for a reversal of effects in the lab are high (circa 100 – 150 atm), but commensurate with water depths and pressures that exist in the vicinity of some well heads (commonly at 3,000 to 10,000 feet, or a total pressure of 100 – 300 atm). A literature review was conducted to determine (1) if dissolved hydrocarbon gases contribute to toxicity via narcosis, and (2) the degree to which elevated hydrostatic pressure might mitigate narcotic effects on aquatic organisms. An approach for applying the TLM to account for these effects was also developed. The present study describes how the TLM has been extended to quantitatively address both the aquatic hazard posed by dissolved gases and the role of pressure in mitigating the narcotic effects of dissolved gaseous and liquid hydrocarbons. Results indicate that the TLM can be applied to dissolved gases based on the octanol-water partition coefficient of the gas and an adjustment factor to the critical target lipid body burden can be used to account for the potential influence of elevated pressure on the toxicity of dissolved hydrocarbon liquids and gases with increasing depth. This study provides a framework for predicting the effects of dispersed oil and gas released in deep sea environments.

Do gaseous hydrocarbons behave as narcotics?

Meyer (1937) determined inhalation 50% effect concentrations (EC50s) for mice exposed to gaseous hydrocarbons in air.

- The EC50s in air decrease as octanol-water partition coefficient increases (light blue bars, left to right)
- At the inhalation EC50s, air-equilibrated hydrocarbon concentrations in olive oil, used as a surrogate for organism lipid, are nearly constant (dark blue bars)
- This shows that these compounds are acting by a non-specific mode of action and that methane, the smallest hydrocarbon gas, behaves as a narcotic

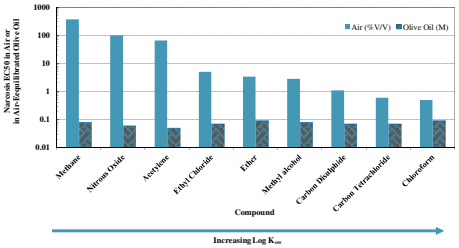


Figure 1. Experimental data from toxicity test with mice (Meyer 1937).

Target Lipid Model and Computation of HC5

The Target Lipid Model (TLM) provides a method for deriving water quality criteria for hydrocarbons. It assumes that

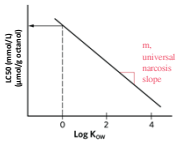
- organism lipid is the site of action of narcosis
- octanol is a surrogate for organism lipid
- each hydrocarbon acts via narcosis and toxicity is additive

The TLM equation to compute critical acute aqueous concentration is

$$\log(C_W) = m\log(K_{OW}) + \log(C_L^*) + \Delta c$$

where:

m = universal slope that describes the relationship for all species
C_W = Acute water effect concentration, $mmol/L$
C_L^* = Species specific critical target lipid body burden (CTLBB), $\frac{\mu mol}{g_{oct}}$
Δc = Chemical class adjustment



The TLM was applied to compute Hazard Concentrations (HC5) intended to protect 95% of species

$$\log(HC5) = E[m]\log(K_{OW}) + E[\log(C_L^*)] + \Delta c - E[\log(ACR)] - k_z\sqrt{V[m]\log(K_{OW})^2 + V[\log(C_L^*)] + E[\log(ACR)]}$$

Where:

$E[m]$ = mean of universal narcosis slope
$E[\log(C_L^*)]$ = mean of the log CTLBB
$E[\log(ACR)]$ = mean of the log ACR
k_z = 95% confidence sample size dependent extrapolation factor
$V[m]$ = variance of universal narcosis slope
$V[\log(C_L^*)]$ = variance of the log CTLBB
$V[\log(ACR)]$ = variance of the log ACR

Predicted Effect Concentrations of Gaseous Hydrocarbons

- TLM-predicted EC50s for *Daphnia magna* are in good agreement with extrapolated values from EC50-K_{ow} relationship for higher molecular weight alkanes.
- Predicted EC50s for methane and ethane are greater than their aqueous solubility. As such, at STP conditions saturated solutions of these gases should produce less than a 50% response to *D. magna*.
- This prediction is in agreement with observations of no effects of methane on tadpoles (Overton, 1901), a species of similar sensitivity.

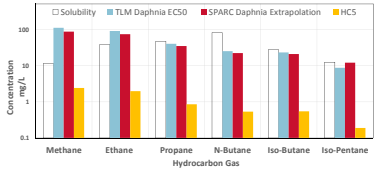


Figure 2

Pressure Protection and Gaseous Pressurizing Agents

Elevated pressure reverses narcotic effects on aquatic organisms (Johnson & Flagler, 1950).

- Dose-response data for effect of urethane on loss of righting reflex (LRR) in tadpoles (Figure 3)
  - At 1 atm hydrostatic pressure, EC50 ~ 14 mM urethane (filled circles)
  - At 130 atm, EC50 ~ 28 mM urethane (unfilled circles)
  - ➢ Increase in pressure results in higher EC50 → pressure protection
  - At 110 atm, EC50 ~20 mM urethane when gaseous pressure agent (GPA) is helium
  - At 30 atm, EC50 ~8 mM urethane when GPA is nitrogen
  - ➢ Degree of pressure protection varies with pressuring agent

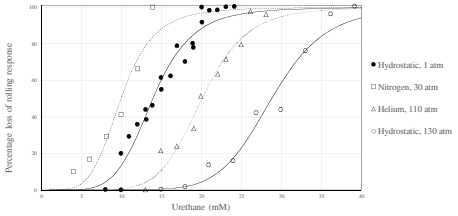


Figure 3. Data from Dodson et al., 1985

Pressure protection has been interpreted in the context of the critical volume hypothesis (Miller et al., 1973; Dodson et al. 1985). That is, narcosis occurs if the lipid membrane accumulates chemical and expands to a critical amount. The effect is reversed by the compression provided by elevated pressure.

- Urethane toxic units (TU_u) needed to maintain a 50% response as test chamber pressure increases (Fig.4). TU is computed as the ratio of urethane concentration needed to maintain a 50% response at elevated pressure divided by the urethane EC50 at 1 atmosphere total pressure.
  - With hydrostatic pressure, urethane TU increase with increasing pressure → pressure protection
  - When a GPA such as helium or neon is used, urethane TU still increases with increasing pressure but not as much as with hydrostatic pressure because the GPA adds TU to the system, so less urethane is needed to produce the same effect.
  - When a GPA such as hydrogen, nitrogen or argon is used urethane TU < 1.0 as pressure increases. These GPAs are relatively toxic and even less urethane is needed than at P_T=1. The contribution of these GPAs to narcosis is greater than pressure protection provided by increase in pressure.

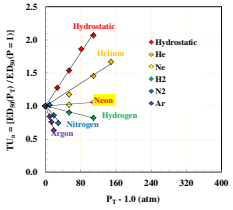


Figure 4

The GPA EP50s (GPA pressure associated with a 50% response, without another narcotic present) were measured by Dodson et al. (1985). These EP50s can be used to compute the GPA toxic units (TU_g = P_T/EP50) and to add them to the urethane TU (TU_u) for the preceding experiments. Now, the total narcotic effect (anesthetic + GPA) is reflected by the Y-axis values and the data begin to collapse to a single line (Figure 5A).

A limitation of using directly measured EP50s is that the elevated GPA pressure causing toxicity is concurrently providing pressure protection.

- Thus the measured EP50 is an "apparent EP50" rather than an "intrinsic EP50" (reflecting GPA toxicity alone, without pressure protection).
- Hydrostatic pressure and GPA test results both reflect expansion due to the anesthetic (urethane) and compression due to pressure
  - GPA results also reflect expansion due to GPA dissolution in the membrane.
  - The difference in the toxic response obtained by these two test methods is indicative of a GPA's *intrinsic toxicity (due to membrane expansion alone)*, rather than its *apparent toxicity (reflecting expansion offset by compression)*.

- *Intrinsic toxicity* may be quantified by the difference in slopes between the GPA and hydrostatic pressure responses on Figure 4.
  - This difference (Δ_g ~ TU/atm) is a measure of the degree of GPA attenuation of pressure reversal in comparison to pressure reversal achieved by hydrostatic pressure alone.
  - The intrinsic EP_{50} of a GPA is equivalent to $1/\Delta_g$ (~atm/TU).
- Using intrinsic EP50s to re-plot the data of Figure 5A, collapses the data to a single line (Figure 5B).

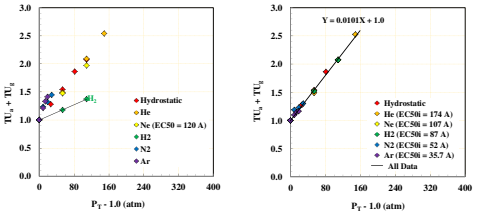


Figure 5 A & B

- Collapsing the hydrostatic and GPA data about a single line isolates the beneficial effect of pressure due to the GPA from its narcotic effect.
- The slope of this line (0.0101 TU/atm) yields a quantitative measure of this beneficial effect. It is incorporated into the TLM for predictive purposes.

Adjusting the CTLBB to account for Pressure

The simplest way to represent how pressure influences narcosis for modeling purposes is to adjust the organism-specific CTLBB to account for pressure-related effects (Paquin et al., 2017). Considering the tadpole results in Figure 5B, the slope of the toxicity data is 0.0101 TU/atm. For 50% narcotic response, the CTLBB for tadpoles is approximately 100 $\mu mol/g_{oct}$ per TU under standard conditions. For an increase in 1 atm of pressure, the CTLBB has to increase by 1.01 $\mu mol/g_{oct}$

$$\frac{100\ \mu mol\ goct}{TU} \times (0.0101\ TU/atm) \times (1\ atm) = 1.01\ \mu mol/g_{oct}$$

Thus, for a depth of 1300 feet ~ 130 atm, CTLBB associated with a 50% response at elevated pressure would increase by approximately 131 $\mu mol/g_{oct}$, which results in a total CTLBB of 231 $\mu mol/g_{oct}$

$$CTLBB_{Elevated\ pressure} = CTLBB_{1atm}(1 + S*P_T)$$

$$231\ \mu mol/g_{oct} = 100\ \mu mol/g_{oct}(1 + (0.0101*130))$$

Figure 6 summarizes the tadpole CTLBB adjustment factors estimated for all studies considered, using both hydrostatic pressure and GPAs (1.34 $\mu mol/g_{oct}$/atm for hydrostatic tests and 1.09 $\mu mol/g_{oct}$/atm for GPA tests). These results are generally consistent with an independent estimate based on thermodynamics and the effect of anesthetic accumulation and pressure on membrane transition temperature (1.54 $\mu mol/g_{oct}$/atm; Heimburg and Jackson, 2007). The mean value for the latter three estimates is 1.32 $\mu mol/g_{oct}$/atm (dashed line).

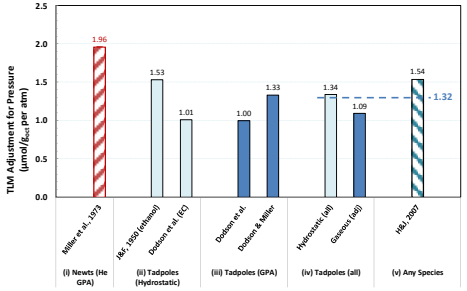


Figure 6

Pressure and Species Sensitivity Distribution (SSD)

The impact of pressure on the CTLBB species sensitivity distribution (SSD) for a dataset of 38 shallow-water aquatic species is shown in Figure 8A (data compiled by McGrath et al. 2004). The lower SSD is the CTLBB at 1 atmosphere. For a CTLBB adjustment of 1.32 $\mu mol/g_{oct}$/atm, a total pressure of 200 atmospheres (ΔP_T = 199 atm) corresponding to a depth of 2000 m should increase the CTLBB by approximately 265 $\mu mol/g_{oct}$. It follows that the SSD at P_T = 200 atm (red dots on Figure 7A) increases by 8.8-fold for the most sensitive species at the low end of the SSD (from 34.3 to 299 $\mu mol/g_{oct}$, ~ HC5) and by only 1.7-fold for the least sensitive species at the high end of the SSD (from 366 to 631 $\mu mol/g_{oct}$). Thus, the more sensitive species are predicted to benefit more from the protective effect of pressure than less sensitive species.

The TLM was then used to compute the HC5 (McGrath et al., 2004, Redman et al., 2012). This calculation assumes that the HC5 depends on the CTLBB Species Sensitivity Distribution, which changes with pressure, while the Acute to Chronic Ratio distribution is assumed to be independent of pressure. This analysis yielded the following relationship for adjusting the HC5 to account for pressure (Figure 7B):

$$\frac{HC5_P}{HC5_{STP}} = 1 + 0.019(P - 1atm)$$

Where:

$HC5_{STP}$ = HC5 at standard conditions (1 atm)
$HC5_P$ = HC5 at pressure of interest
P = total hydrostatic pressure of interest (atm)

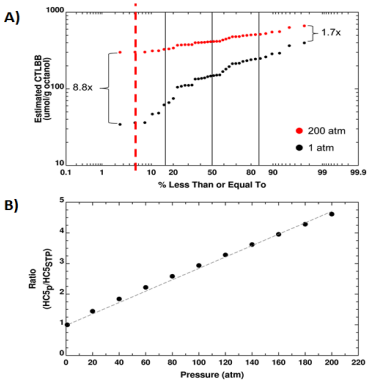


Figure 7

Summary

- TLM predictions of hydrocarbon gas toxicity suggest that saturated solutions of methane and ethane would not be toxic at STP. Reliable toxicity data are not available for these hydrocarbons gases. The high volatility and explosive nature of these gases present challenges in obtaining reliable data.
- An increase in pressure appears to offset the effect of narcosis in lab toxicity studies.
- Based on a critical analysis of pressure-dependent toxicity studies, the observed protective effect afforded by pressure was translated into an increase in the CTLBB per atm of pressure of 1.32 $\mu mol/g_{oct}$
- Based on this analysis, the target lipid model can be extended to calculate pressure-dependent water quality objectives (HC5s) for dissolved hydrocarbons to support risk assessment.
- Model uncertainties are related to extrapolating limited laboratory test results with amphibians (mostly tadpoles) to deep-sea organisms under field conditions.
- If future toxicity tests at elevated pressure are conducted:
  - Unexposed controls should be included to account for physical effects of pressure alone and relative sensitivities of un-acclimated and pressure-acclimated test organisms.
  - The influence of exposure duration on pressure reversal and pressure protection warrants study.
  - The assumption regarding lack of pressure dependence on the ACR should be confirmed.

References

Dodson, B.A., Z.W. Furmaniuk Jr., and K.W. Miller, 1985. "The Physiological Effects of Hydrostatic Pressure are Not Equivalent to those of Helium Pressure on *Rana pipiens*," Journal of Phys, 362: 233-244.

Heimburg, T., and A.D. Jackson, 2007. "The Thermodynamics of General Anaesthesia," *Biophysical Journal*, 92: 3159-3165.

Johnson, F.H., and E.A. Flagler, 1950. "Hydrostatic Pressure Reversal of Narcosis in Tadpoles," *Science*, 112: 91-93.

McGrath, J., T. Parkerton and D.M. Di Toro, 2004. "Application of the Narcosis Target Lipid Model to Algal Toxicity and Deriving Predicted-No-Effect Concentrations," *Environmental Toxicology and Chemistry*, 23: 2503-2517.

Meyer, K.W., 1937. "Contributions to the Theory of Narcosis," Transactions Faraday Society, 33: 1062-1064.

Miller, K.W., W.D.M. Paton, R.A. Smith and E.B. Smith, 1973. "The Pressure Reversal of General Anesthesia and the Critical Volume Hypothesis," *Molecular Pharmacology*, 9: 131-143.

Overton, C.E., 1901. *Studies of Narcosis*, a translation by R.L. Lipnick (ed.), first edition published in 1991 by Chapman and Hall Ltd. and Wood Library-Museum of Anesthesiology, 191 pp.

Paquin , P.R., J.McGrath, C. Fanelli and D.M. Di Toro, 2017. "The Aquatic Toxicity of Hydrocarbon Liquids and Gases and the Effect of High Pressure on Exposure and Narcotic Effects," in preparation.

Redman, A., T. Parkerton, J. McGrath and D.M. Di Toro, 2012. "PETROTOX: An Aquatic Toxicity Model for Petroleum Substances," *Environmental Toxicology and Chemistry*, 31: 2498-2506.

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