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The aquatic hazard of hydrocarbon liquids and gases and the modulating role of pressure on dissolved gas and oil toxicity

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ABSTRACT

Hydrostatic pressure enhances gas solubility and potentially alters toxicity and risks of oil and gas releases to deep-sea organisms. This study has two primary objectives. First, the aquatic hazard of dissolved hydrocarbon gases is characterized using results of previously published laboratory and field studies and modeling. The target lipid model (TLM) is used to predict effects at ambient pressure, and results are compared to effect concentrations derived from extrapolation of liquid alkane hazard data. Second, existing literature data are used to quantify and predict pressure effects on toxicity using an extension of the TLM framework. Results indicate elevated pressure mitigates narcosis, particularly for sensitive species. A simple adjustment is proposed to allow TLM-based estimates of acute effect and TLM-derived HC5 values (concentrations intended to provide 95% species protection) for oil or gas constituents to be calculated at depth. Future applications, and opportunities and challenges for providing validation, are discussed.

1. Introduction

Releases of oil and gas during exploration, production, or transport of crude or refined petroleum substances pose well-known risks to aquatic biota. Most work directed at quantifying these risks has focused on the fate and effects of liquid oil spills at or near the water surface (Jewett et al., 1999; Peterson, 2001; Rice et al., 2001; Page et al., 2002; Peterson et al., 2003; IER, 2010; County of Santa Barbara, 2011). Acute toxicity resulting from exposure to the lighter, more water-soluble aromatic components of oil spilled at the surface are commonly attributed to narcosis (Brocksen and Bailey, 1973; Brodersen, 1987). Such effects can be quantitatively described using the critical body residue (CBR) concept which assumes that observed effects can be related to the internal concentration of hydrocarbons within an aquatic organism (Di Toro et al., 2000; French-McCay, 2002; McGrath and Di Toro, 2009). Due to the more volatile nature and faster biodegradation of monoaromatic hydrocarbons (MAHs), polyaromatic hydrocarbons (PAHs) tend to be more persistent and have often been the focus of evaluating chronic risks of longer term oil spill related exposures (French-McCay, 2016). Such chronic effects can involve different modes of toxic action beyond narcosis (Hose et al., 1996; Carls et al., 1999; Incardona et al., 2004; Rhodes et al., 2005).

In addition to liquid hydrocarbon spills, releases may involve gaseous hydrocarbons, including methane and lesser amounts of other low molecular weight alkanes (Valentine et al., 2010; Joye et al., 2011; Socolofsky et al., 2015). When released at or near the water surface, similar to volatile liquid aliphatic constituents, dissolution and subsequent exposure in the water column will be limited due to rapid volatilization to the atmosphere. It is for this reason that these dissolved aliphatic hydrocarbon gases have typically been neglected in exposure and effects assessments. The situation differs for deep-sea releases, where both the partial pressure and residence time of gas bubbles in the water column are increased. Thus, dissolved gas concentrations may be much higher than their solubility limits under standard pressure conditions near the surface, thereby increasing exposures to deep-sea organisms. Such an increase in dissolved gas exposures has been documented in association with deep-sea releases of oil when real-time measurements have been made to track plumes. For example, methane is ubiquitous and present at relatively low concentrations in aquatic settings throughout the world, but dissolved methane concentrations may be markedly increased in proximity to deep-sea releases (Camilli et al., 2010; Yvon-Lewis et al., 2011). However, the relative importance of dissolved gases in contributing to potential adverse effects in such releases remains an open question.

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The 2010 Deepwater Horizon blowout in the Gulf of Mexico highlighted the need to better understand risks to aquatic life associated with deep-sea hydrocarbon releases (Valentine et al., 2010; Joye et al., 2011; Thibodeaux et al., 2011). One unique aspect of deep-sea release scenarios is the elevated pressures that characterize such environments. Pressure may modulate exposure by enhancing the solubility of gases. One need only refer to the seminal investigations of narcotic effects on tadpoles, by Hans Meyer (1899) and Charles Overton (1901), to gain insight into this consideration. For example, Overton (1901) provided the earliest known mention of how effects of gases should be accentuated by the increase in their aqueous solubility with increasing pressure. He reported that tadpoles, exposed to saturated solutions of methane or ethane were unaffected at standard temperature and pressure conditions, and he posited that effects due to methane should be expected at 18 to 30 atm (Overton, 1901). In contrast, pressure has a much less pronounced effect on the solubility of liquid hydrocarbons (Gros et al., 2016). Alternatively, dissolved gas exposures can be reduced through clathrate (an ice-like crystalline structure that incorporates trapped gases) formation under high pressure and low temperature deep-sea conditions. Pressure may also influence hazard and the need to better understand the role of pressure on oil toxicity has been recognized (Thibodeaux et al., 2011; Mestre et al., 2014). However, there is a paucity of empirical toxicity data, a reflection of the inherent difficulty of collecting, culturing and testing deep-sea species under high pressures.

An alternative to use of test results from the field of aquatic toxicology is to consider research in the field of anesthesiology. In this case, elevated pressure has been used to gain insight into the underlying mechanisms of anesthesia. Further, because this research frequently focuses on narcotic effects on aquatic organisms, such studies inform our understanding of how pressure influences the effects of dissolved hydrocarbons on deep-sea organisms. Johnson and coworkers (Johnson et al., 1942a, 1942b; Johnson and Eyring, 1948; Johnson and Flagler, 1950, 1951; Johnson et al., 1954) are often credited with having completed the earliest demonstrations that an increase in total pressure may actually reverse narcotic effects. These early pressure-reversal results led to further investigations of the underlying mechanism of narcosis. For example, Miller and coworkers (Johnson and Miller, 1970; Miller et al., 1973; Dodson et al., 1985) used newts and tadpoles to demonstrate the antagonistic effect of pressure on narcosis (i.e., pressure reversal of narcosis), and to quantify how such effects may be related to changes in volume of a lipid bilayer thought to be associated with the central nervous system of the tadpole.

Others working with different models of lipid membranes have found that administration of a narcotic leads to an increase in fluidity of the lipid phase, a change that is causally related to narcotic effects (Trudell et al., 1975; Mountcastle et al., 1978). This finding is explained on the basis of thermodynamic principles. That is, chemical accumulation alters the solid-liquid transition state of lipid membranes. It *reduces* the melting point, thereby disrupting nerve pulse transmission (e.g., Ebel et al., 2001; Kharakoz, 2002, 2008; Heimburg and Jackson, 2005, 2007a, 2007b; Graesbøll et al., 2014; Heimburg, 2014). The results of pressure reversal studies are consistent with this explanation, because an increase in pressure increases the melting point, thereby restoring nerve pulse transmission. Note that this thermodynamic rationale is consistent with membrane expansion as a result of chemical accumulation, even though expansion may simply be a response associated with fluidization and a narcotic effect, but not necessarily causally related to it. Thus, because changes in both membrane volume and transition temperature are associated with a change in membrane narcotic concentration, these alternative hypotheses are not mutually exclusive and it is unclear which, if either, provides a correct mechanistic explanation of narcosis. Further, they do not preclude the possibility that an alternative physiological explanation, such as the mean excess volume hypothesis (Matubayasi and Ueda, 1983) or the frequently espoused protein binding hypothesis (Franks and Lieb, 1982, 1987; Franks, 2006), might be correct. As a result, in spite of an extensive body of research, a definitive mechanistic explanation of narcosis has proven elusive. Further details about these early studies of pressure and narcosis, and more recent thermodynamically inspired investigations, are included in Supplemental Information 01 (SI 01).

The Target Lipid Model (TLM), which is based on the critical body residue concept, has been developed and validated for predicting acute effects resulting from exposure to individual hydrocarbons (Di Toro et al., 2000; Burgess et al., 2013) and petroleum related mixtures assuming concentration addition (McGrath et al., 2005, 2009; Redman et al., 2012). When used in conjunction with empirically derived acute to chronic ratios (ACRs) and a 95% level of protection for characterizing the acute species sensitivity distribution (SSD), the TLM may be used to estimate concentrations protective of chronic effects (McGrath and Di Toro, 2009; Redman et al., 2014a). Therefore, the TLM provides a valuable framework for evaluating both the acute and chronic risks of oil and petroleum related substances, a framework within which both the composition and concentration of dissolved hydrocarbon components can be quantitatively considered (Redman and Parkerton, 2015). However, although the TLM has been used to predict the toxicity of dissolved liquid hydrocarbons, it has not previously been used to predict the toxicity of dissolved hydrocarbon gases. Further, the TLM does not consider the potential role of elevated pressure on toxicity in deep-sea settings.

There are two main objectives of this study. The first is to characterize the toxicity of dissolved gases to aquatic life. This was accomplished by completing a review of lab and field studies that characterize effects on aquatic life that result from exposure to dissolved gases. The ability of the TLM to predict the acute toxicity effect concentrations for dissolved hydrocarbon gases at ambient pressure is then tested by comparing the predictions to dissolved gas effect concentrations estimated by extrapolation of toxicity data for liquid alkanes. The second objective is to characterize the influence of elevated pressure on the aquatic toxicity of narcotic substances. Empirical pressure-dependent aquatic toxicity studies on narcotic substances from the literature are reviewed. An extension of the TLM framework is then described and used to evaluate the compiled aquatic toxicity datasets. It is shown that use of hydrostatic pressure and different gaseous pressurizing agents (GPAs) to achieve elevated pressure differentially influence the anesthetic effect and that, in the context of the proposed framework, consideration of the narcotic action of the GPA can resolve these inconsistencies. Based on this analysis, an approach is proposed for use of the TLM to account for the role of pressure on the predicted toxicity of both dissolved hydrocarbon gases and liquids. Potential limitations of the approach and opportunities for validation are also discussed.

2. Results

This section summarizes information on the aquatic hazards of dissolved hydrocarbon gases, how elevated pressure influences exposure levels and effects of both gaseous and liquid hydrocarbons, and how these factors may be quantified for modeling purposes.

2.1. The aquatic hazard of dissolved hydrocarbon gases

The physical-chemical characteristics of dissolved hydrocarbon gases are presented first to facilitate the interpretation of toxicity data in the context of a chemical accumulation-based toxicity model. Laboratory and field toxicity data are also summarized and used to infer their mode of action. The limited effects data are supplemented with model predictions of the expected toxicity of these gases.

2.1.1. Physical chemical characteristics

The aquatic hazard of dissolved compounds is commonly related to and may be inferred from their physical-chemical characteristics (e.g., SI 02). Thus, it is useful to consider some of the pertinent characteristics