Marine Pollution Bulletin 109 (2016) 259–266

Contents lists available at ScienceDirect

Marine Pollution Bulletin

journal homepage: www.elsevier.com/locate/marpolbul

Developing a polycyclic aromatic hydrocarbon exposure dose-response model for fish health and growth

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ARTICLE INFO

Article history:
Received 22 January 2016
Received in revised form 26 May 2016
Accepted 28 May 2016
Available online 10 June 2016

Keywords:
Dose-response
Ecosystem
Oil spill
Modeling
PAH
Toxicity

ABSTRACT

One of the more important steps in understanding the ecosystem-level effects of anthropogenic disturbances on resident species is developing an accurate representation of the lethal and sub-lethal effects of these stressors. We develop methods for describing the impacts of oil on growth and mortality rates in fishes. We conducted a literature search to determine potential relationships between direct and indirect effects of exposure to oil, based on the frequency of lesions and body growth reduction. Data examining these effects with different exposure mediums were assessed and then input into four potential response models (a linear, step-wise, hockey-stick, and exponential model). We assessed the models using the Akaike Information Criterion. The most parsimonious and best fit model was the hockey-stick. This analysis will aid in identifying where future research on the impact of oil on fish should focus and also aid the development of ecosystem models on impacts of oil spills.
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1. Introduction

As the quantity and type of chemicals humans introduce into aquatic environments grows, a better understanding of species responses to exposures and factors that modulate it must be developed. As the number of oil extraction operations and corresponding oil spills increase, the specific need to understand how oil can impact organisms, especially fish, has become even more acute. The intensity of crude oil toxicity depends on the species or life stages involved, as well as the concentration and composition of the oil (Mosbech, 2002; McCay et al., 2004; Incardona et al., 2011a; McKenna et al., 2013). Exposure effects are further modified by the local climate, habitat, currents, and oil spill response efforts (Moore and Dwyer, 1974; Tjeerdema et al., 2013; Almeda et al., 2014).

Understanding both lethal and sub-lethal exposure effects is important to building a comprehensive view of the impacts from an oil spill. Direct mortality from hydrocarbons in oil is observed across a range of species (Rice et al., 1984; Lee and Page, 1997; Carls et al., 1999; Brown-Peterson et al., 2015), and sub-lethal effects are observed in some organisms (Moore and Dwyer, 1974; Lee and Page, 1997; Brown-Peterson et al., 2015). These toxic sub-lethal effects can include changes in behavior and predator-prey dynamics, growth impediment, and increased susceptibility to disease and parasitism (Moore and Dwyer, 1974). Sub-lethal effects may interact and persist after acute toxicological impacts have subsided. Exposure to oil can slow growth, decrease recruitment to the fishery, and increase susceptibility to predation (Moles & Norcross, 1998). The size-spectrum theory suggests that at a given age, larger individuals have a higher chance of survival due to lower predatory densities (Hare and Cowen, 1997), thus even a small decline in the ability of a juvenile fish to grow or store lipids can have a severe impact on their chance of survival (Meador et al., 2006). This suggests that growth reduction can have an indirect mortality effect on a population. Fecundity may also be affected by a decline in growth, as larger females produce larger eggs with larger yolk sacs, leading to faster larval growth (Kennedy et al., 2007).

Within the oil exposure literature, a large body of research has focused on the effects of polycyclic aromatic hydrocarbons (PAHs), key components of oil, on certain fish species. PAHs are thought to be major components in the toxicity of oil, partially because their metabolites have oxidative and carcinogenic properties (Moore and Dwyer, 1974; Lin and Tjeerdema, 2008; Incardona et al., 2011a). Brown et al. (1973) found a higher prevalence of stomach, skin, and liver tumors, especially hepatic neoplasms, in 16 species of fishes correlated with the presence of two PAHs (naphthalene and benzanthracene), crude oil, and other toxicants (e.g. chlorinated hydrocarbons) in the water column of the Fox River in Wisconsin. There is also strong evidence supporting a causal relationship between exposure to sediment PAHs and liver neoplasms and other liver lesions in marine species (Myers et al., 1991). A statistical analysis of these lesions supports a

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http://dx.doi.org/10.1016/j.marpolbul.2016.05.072
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morphologically identifiable progression ultimately leading to hepatic neoplasms, or liver tumors (Myers et al., 1991). The most supported causative PAHs for liver lesions are higher in weight, often pyrogenic (Vethaak et al., 1996; Myers et al., 1998). Burning oil in response to a spill increases the relative amount of higher weight PAHs in the system, increasing the risk of these lethal impacts.

Predicting the impacts of oil spills on fish populations is further complicated by the lack of data relating direct exposure concentrations to changes in life rates. Many studies, dating back to Moore and Dwyer (1974) have approximated the oil threshold at which a risk of health impacts begins, but not the shape of the organismal or population response. Some studies only compare oiled with non-oiled sites, and do not consider the degree of exposure (Khan and Nag, 1993; Jewett et al., 1995; Marty et al., 2003). Others use biliary fluorescent aromatic compounds (FACs) as an indicator of oil exposure, which have been documented in the laboratory to have a positive correlation with exposure dose (Collier and Varanasi, 1991). However, there are many factors that influence these FAC concentrations and caution should be used when interpreting these results from field investigations (Lee and Anderson, 2005). These examples highlight the difficulty in defining the relationship between oil exposure concentration and organismal response.

Dose-response curves, which predict how organisms and populations respond to increasing toxin exposure, offer a chance to connect lethal and sub-lethal impacts of oil. They also may be integrated into ecosystem models in order to predict long-term changes in food webs due to spills. The goal of this article is to parameterize best fit dose response models relating petrogenic PAH or oil exposure values to growth and mortality rates, which can then be used to predict responses of organisms to oil spills. Developing dose-response curves will allow direct estimates of oil impacts on organisms to be developed and tested. However, dose-response curves may take multiple shapes. To assess our current knowledge of how oil exposure may impact species, we gathered data on petrogenic PAH exposure and impacts on fish species from the literature and tested best fits against several common response models. This assessment and modeling exercise will also aid in identifying gaps in current empirical studies and target future work.

2. Methods

2.1. Literature search

Given the paucity of data related directly to oil exposure on juveniles and adults, we conducted a literature search to identify studies where an impact on organism health or growth was correlated with PAH exposure. We recognize the existence of literature on oil effects on larvae and embryos (e.g. Incardona et al., 2005, 2008, 2011b, 2015; Carls et al., 2008), however they were not included in this study as there is a difference in response during this stage as well as many ecosystem models do not explicitly model this life stage. Our search was initially guided by the review of PAH impacts on fishes by Collier et al. (2013), however many of the studies reviewed were pyrogenic in nature, therefore the search was expanded upon to locate other relevant studies focused on petrogenic sources. Lee and Page (1997) provide another review of oil spill impacts on sub-tidal regions, however many field studies relating to the Exxon Valdez spill are unable to quantify oil exposure. While we originally intended to develop different dose-response curves for juvenile and adult fishes, and for pelagic, demersal and epibenthic habitat use, it is not possible at this time due to limited ecotoxicological data. Thus, we grouped all relevant data found in the literature search into one general fish grouping for the purpose of establishing the shape of the dose-response curve (although we note that the overall magnitude of effect is established through other means and may reflect differential PAH uptake rates due to habitat use). Ecotoxicological studies in progress by Gulf of Mexico Research Initiatives (GOMRI) researchers (see http://gulfresearchinitiative.org/consortia/), may soon provide data necessary to discriminate between different life stages and habitat use.

From the results of our literature review, we tested the relationship between PAH exposure and lesion and tumor frequency (a proxy for mortality) as well as growth for several reported species. Lesion frequency was chosen as a proxy for mortality due to the documented progression from lesions to tumors (Myers et al., 1991), indicating an overall decline in the health of the fish. We do not assume direct mortality from this decline in health, but rather an increase in likelihood of mortality from any source due to reduced health and behavioral changes (Moore and Dwyer, 1974; Moles & Norcross, 1988). Overall, four studies were used relating to lesion or similar pathology frequency data, located on the liver, pancreas, gills, or externally, and seven studies were used relating to growth reduction. To examine the growth response after PAH exposure, the species included were Pink Salmon (*Oncorhynchus gorbuscha*), Inland Silversides (*Menidia beryllina*), Yellowfin Sole (*Limanda aspera*), Rock Sole (*Lepidopsetta bilineata*), Pacific Halibut (*Hippoglossus stenolepis*), Chinook Salmon (*Oncorhynchus tshawytscha*), Turbot (*Scophthalmus maximus*), Zebrafish (*Danio rerio*), Southern Flounder (*Paralichthys lethostigma*) (Moles and Rice, 1983; Gundersen et al., 1996; Moles and Norcross, 1998; Meador et al., 2006; Morales-Nin et al., 2007; Vignet et al., 2014; Brown-Peterson et al., 2015). Literature growth values were not uniform in units reported, so available data were converted to reflect a proportional reduction in biomass growth rate. This was done through a data transformation, which divided each individual growth response by a reference rate (which is represented by the growth rate under the lowest PAH concentration tested). This resulted in a low value for organisms exhibiting slow growth. All values were then subtracted from one to represent the proportional difference in growth from the reference rate. Using this method, all growth rate data points fall between 0 and 1, where a value of 0 would be no reduction in growth relative to the reference rate and 0.5 would be 50% reduction in growth. Exposure concentrations were also not uniform, and included petrogenic hydrocarbons, total PAH, fuel oil concentration, total aromatic hydrocarbons, and tPAH50 (sum of 50 aromatic hydrocarbons). Units were converted to μg/L water, μg/kg wet sediment, or μg/kg food pellet.

For lesion frequency some of the species included were Zebrafish (*Danio rerio*), Southern Flounder (*Paralichthys lethostigma*), and Alligator Gar (*Atractosteus spatula*) (Larcher et al., 2014; Brown-Peterson et al., 2015; Omar-Ali et al., 2015). The final data set included came from a

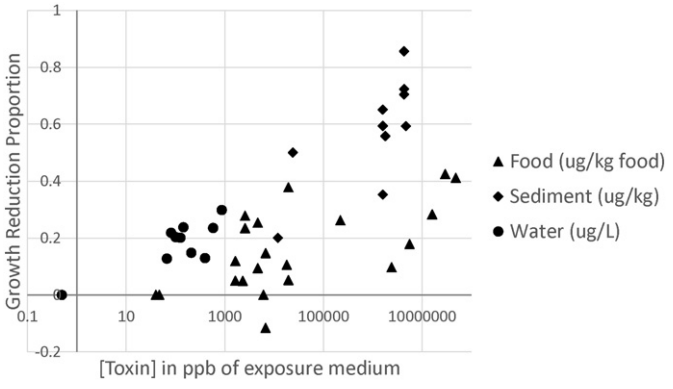

Fig. 1. All literature data used in the analyses relating PAH exposure to growth reduction. The x-axis is shown on a log scale of toxicant concentration in ppb for different exposure mediums (μg/L water, μg/kg wet sediment, or μg/kg food). Toxicant concentrations were reported as total petrogenic hydrocarbons, total PAH, fuel oil concentration, total aromatic hydrocarbons, or tPAH50 (total concentration of 50 PAH) (Moles and Rice, 1983; Gundersen et al., 1996; Moles and Norcross, 1998; Meador et al., 2006; Morales-Nin et al., 2007; Vignet et al., 2014; Brown-Peterson et al., 2015). The y-axis is a proportional reduction in growth for each data point, obtained by comparing it to the control or lowest-exposure data point in that study. The legend illustrates the exposure medium.

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