



Contents lists available at ScienceDirect

Journal of Great Lakes Research

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

Commentary

Toxicity delayed in cold freshwaters?

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ARTICLE INFO

Article history:

Received 1 September 2014

Accepted 16 February 2015

Available online xxx

Communicated by Marlene Evans

Index words:

Delayed toxicity

Polar

Cold freshwaters

Risk assessment

ABSTRACT

There is increasing evidence of a delay in the manifestation of toxicity with exposure to contaminants and other stressors in polar (Arctic and Antarctic) marine environments. This phenomenon has not been shown to occur in cold (i.e., $<4^{\circ}\text{C}$) freshwater environments. Toxicity testing protocols have not been designed to investigate the existence of this phenomenon; toxicity testing is typically conducted in the laboratory at warmer than ambient cold water temperatures (i.e., $10\text{--}15^{\circ}\text{C}$) and for pre-determined time periods. The hypothesis of delayed toxicity in cold freshwaters is viable based on a review of available literature; this hypothesis should be tested. If this phenomenon occurs in cold freshwaters (i.e., northern, southern, and high altitude freshwaters), and if the exposure period is not adequate to account for a delayed response, sensitivity to tested toxicants under cold water conditions may not be adequately estimated, resulting in an underestimation of toxicity and an overestimation of predicted no effect concentrations.

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Introduction

Polar (cold water) fish and their prey are likely not more sensitive to contaminants than in temperate regions (Chapman and Riddle, 2005a). However, the relative sensitivity of polar marine and freshwater fish, and freshwater invertebrates to contaminants compared to their temperate and tropical counterparts remains to be fully assessed (deHoop et al., 2011). Toxicity tests with polar marine invertebrates indicate that sensitivities are similar to temperate marine invertebrates; however, toxic effects can take longer to manifest due to: 1) colder temperatures that retard metabolic activities and chemical reactions, and 2) smaller surface area-to-body mass ratios with larger fat reserves (Chapman and Riddle, 2005a; Zamora et al., 2015).

There is presently little evidence from field and laboratory studies in cold freshwater environments (e.g., large lakes) of the same lag time in expression of toxic effects found in marine environments. This lag may not occur in cold (i.e., $<4^{\circ}\text{C}$) freshwaters due to less constant cold water conditions (i.e., warmer temperatures in summer compared to polar marine environments). Alternatively, it may occur but not have been detected because freshwater toxicity testing is typically conducted at higher temperatures and for fixed time periods.

The purpose of the present Commentary is to suggest, from available literature, the possibility of delayed toxicity in cold freshwaters and to consider its potential significance for assessing and managing northern, southern, and high latitude cold water ecosystems exposed to stressors including contaminants.

Evidence for delayed toxicity in cold waters

Numerous studies have shown that polar (Arctic and Antarctic) marine invertebrates are generally slower to respond to contaminants including metals, polycyclic aromatic hydrocarbons (PAHs), and other contaminants, than closely related temperate species (King and Riddle, 2001; Jensen et al., 2008; Jensen and Carroll, 2010; Hjorth and Nielsen, 2011; Hansen et al., 2011, 2013; Payne et al., 2014; Zamora et al., 2015). As stated by Payne et al. (2014, p883), based on Peck (2002) and other authors, "The delayed response time of cold-adapted species has been linked to slower uptake kinetics, slower growth and development, lower metabolic rates, and higher lipid storage of organisms at low temperatures." Differences in toxicity responses between polar and temperate species have, in some cases, been ascribed to delayed onset of toxicity in the polar species (Chapman and Riddle, 2005a,b; Chapman et al., 2006; Zamora et al., 2015); however, in other cases the possibility of delayed toxicity was not investigated (Olsen et al., 2007; deHoop et al., 2011; Bach et al., 2014).

Pioneering studies of the 96-h acute toxicity of lead and zinc to epontic (i.e., under-ice) Arctic marine amphipods at ambient (cold) water temperatures found a surprising lack of effects even at concentrations of lead and zinc that would have caused total mortality of freshwater amphipods (Chapman and McPherson, 1993). Based on these studies Chapman (1993) suggested that Arctic marine invertebrates could be relatively insensitive to metals.

However, a repetition of this work in the Antarctic, as reported by Chapman and Riddle (2005a) found that marine amphipods tested in cold water for 10 days had similar sensitivity as temperate amphipods exposed for 4 days. In other words, toxicity was delayed as illustrated in Fig. 1. Zamora et al. (2015) similarly found that exposure periods of

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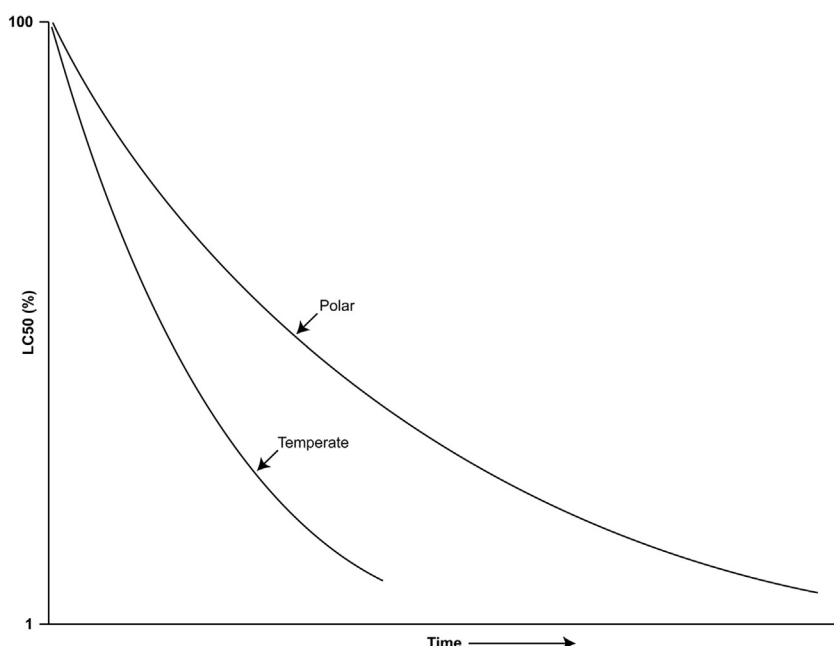


Fig. 1. Toxicity manifests more slowly in colder waters. Polar and temperate marine amphipods show similar sensitivity to contaminants (LC50s, lethal concentration to 50% of exposed organisms) but with slower manifestation in Polar marine waters. Adapted from Chapman and Riddle (2005a).

48–96 h, commonly used in toxicity tests with temperate and tropical species, are too short for polar organisms to respond and stated (p267) “This study highlights the need for longer exposure periods in toxicity tests with slow responding Antarctic biota in order to generate relevant sensitivity data for inclusion in site-specific environmental quality guidelines for Antarctica.”

Poulsen et al. (2011) observed accelerated developmental timing in Antarctic krill larvae exposed to p,p'-DDE up to 9 days. However, in a delayed toxicity experiment, comparing larvae exposed for only 5 days with prolonged monitoring (>2 weeks) of larvae in clean seawater revealed delayed effects including mortality, delayed and unsuccessful development.

Lotufo et al. (2000) demonstrated varying toxicokinetics and apparent toxicological sensitivity for two freshwater amphipod species exposed to DDT and its metabolites at different ambient temperatures (4 °C versus 18–21 °C). While toxicokinetics were slow at 4 °C compared to 18–21 °C, steady state bioconcentration factors were relatively high in the colder water. Experiments of varying exposure durations (4, 10, 28 days) demonstrated increasing toxicity with time in both species in the colder water.

Cold temperatures can potentially increase the persistence of a variety of contaminants in aquatic ecosystems by decreasing mixing rates, evaporation, dissolution, and biodegradation. The sensitivity of aquatic organisms to contaminants can be affected by the cold waters in which they live. Cold waters result in physiological adaptations, and in reduced chemical reactions both outside and within organisms (Chapman and Riddle, 2005a). Some species enter dormancy at low temperatures; as noted by Li et al. (2014, p1564) for a copepod and a rotifer, “Such metabolic depression responses in these zooplanktons could reduce their uptake of the chemical and hence minimize the chemical toxicity at low temperatures.” These authors also noted that (p. 1571) toxicities of four chemicals for these two zooplanktons “increased significantly with increasing temperatures, but not with decreasing temperatures”.

So what if toxicity is delayed in cold waters?

As documented in the previous section, there is good evidence that manifestation of toxicity can be delayed in cold marine waters. Warmer

temperatures generally result in increased toxicity of environmental contaminants (e.g., Heugens et al., 2001; Noyes et al., 2009; Kimberly and Salice, 2013; Zhou et al., 2014; Laetz et al., 2014). Temperature variability can also increase the sensitivity of aquatic organisms to toxicants (Kimberly and Salice, 2014) and, conversely, toxicants can increase the sensitivity of organisms to temperature (Little and Seebacher, 2015).

Consequently, it is not unreasonable to consider the possibility that delayed manifestation of toxicity could also occur in cold freshwater ecosystems typical of northern, southern, and high altitude freshwater environments such as large lakes. Toxicity testing protocols have not been designed to investigate the existence of this phenomenon; testing is typically conducted in the laboratory for set time periods at warmer than ambient cold water temperatures.

Whether or not a toxicant elicits a response in an aquatic toxicity test is a function of the concentration of a contaminant and duration of exposure. Generally, a greater proportion of organisms in a toxicity test will show a specified effect as test duration increases. Toxicity thresholds therefore shift downward with time (i.e., the longer the test duration, the lower the concentration of contaminant required to elicit a response). This continues until time (test duration) is no longer a factor and the incipient threshold has been reached. At this point, all exposed organisms that are going to respond have responded.

Exposure duration is likely very important for toxicity tests with cold freshwater species actually tested in cold freshwaters. The spatial extent and temporal duration of cold temperatures may vary within lakes, for example related to the hypolimnion of large lakes. The spatial and temporal durations of cold temperatures for biota within lakes will also vary. As noted previously, some animals are inactive in the winter and during this time they cannot control their temperature exposure. Other animals such as zooplankton and winter spawning fish (e.g., burbot) are active throughout the winter and thus have the capacity to move between temperature gradients.

Because temperature affects toxicity, Zhou et al. (2014, p20) stated that “toxicity tests should be conducted at...environmentally realistic temperatures”; similar recommendations were made by Li et al. (2014). Low temperatures will reduce contaminant uptake rates, while adaptation to cold will reduce metabolic rates (Clarke and Peck, 1991; Peck, 2002). Greater energy stores in the form of lipids can further

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