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Response to Humphreys' (2007) Comments on Hose GC (2005) Assessing the Need for Groundwater Quality Guidelines for Pesticides Using the Species Sensitivity Distribution Approach.

LETTER TO THE EDITOR

Response to Humphreys' (2007) Comments on *Hose GC (2005) Assessing the Need for Groundwater Quality Guidelines for Pesticides Using the Species Sensitivity Distribution Approach.*

The increasing dependence of society on groundwater to meet industrial, agricultural, and domestic water needs threatens the integrity of groundwater ecosystems. Coincident with the increasing pressure for water extraction is an increasing threat of aquifer contamination from industrial and agricultural chemicals and nutrients. Consequently, there exists a strong need for water quality guidelines that are specific to the protection of groundwater ecosystems.

Recently, I derived water quality guidelines for groundwater ecosystems (Hose 2005) using the species sensitivity distribution (SSD) approach (Posthuma *et al.* 2002a). To achieve this I used acute toxicity data for a reduced suite of taxa in order to simulate the truncated biodiversity of groundwater ecosystems (Gibert and Deharveng 2002). In the absence of sufficient toxicity data for true hypogean fauna, I used the acute toxicity data for epigean taxa in these groups as a surrogate for groundwater taxa.

As I point out in Hose (2005), and as amplified in some detail by Humphreys (2007), the fauna inhabiting in groundwater (hypogean) ecosystems are physiologically very different from those in surface (epigean) ecosystems. Humphreys (2007) agrees with limiting the species used in the SSD analyses to those commonly found in groundwater ecosystems. However, he questions whether it is appropriate to use toxicity data for surface fauna as a measure of acute toxicity on groundwater organisms and cautions against its "uncritical general acceptance." In particular, Humphreys (2007) argues that groundwater organisms typically have (1) low metabolic rates, (2) are adapted to a low energy, low PO_2 environment and, (3) have greater fat stores than epigean relatives, and as a result, they will respond differently to toxicants.

In this letter I argue that the approach taken in Hose (2005) is a valid preliminary risk assessment for groundwater ecosystems. I then show through a short review of toxicity literature that the factors identified by Humphreys (2007) variably, sometimes negligibly, and unpredictably affect the response of hypogean organisms to toxicants. Consequently, there is little evidence of a consistent or systematic bias in using the SSD approach. I conclude by emphasizing that although our understanding of the physiology of hypogean organisms and their responses to toxicants remains limited, and toxicity data remain sparse, the approach I have taken (Hose 2005) is essential if we are to move forward in the management of groundwater quality and ecosystem protection.

Humphreys' (2007) concerns are appropriately directed. Indeed, no risk assessment should be adopted nor water quality guidelines applied without critical reflection. Toxicity data for species in the target ecosystem *should* underpin risk assessments and the setting of guidelines for that ecosystem (Forbes and Calow 2002), but sufficient toxicity data are currently lacking for groundwater ecosystems. In lieu of sufficient data, the use of guideline values for surface ecosystems is currently recommended to protect groundwater ecosystems in Australia (ANZECC/ARMCANZ 2000). My approach betters the simple application of surface water quality guidelines because it truncates the suite of taxa used toward those in the target ecosystem, which is of critical importance in shaping of SSD outcomes (Duboudin *et al.* 2004). Nevertheless, this approach and its numerous assumptions (outlined in Hose 2005) should be scrutinized in the context of the site and conditions to which any guidelines are to be applied.

In a tiered risk assessment framework, the approach taken by Hose (2005) is commonly used to provide a preliminary characterization of ecological effects (*e.g.*, Warren-Hicks *et al.* 2002). Indeed, much of ecotoxicology and risk assessment is based on applying toxicity data from laboratory-cultured species to natural ecosystems where those taxa do not necessarily occur, under the assumption that the test species represent the likely responses of some taxa in that environment (Kooijman 1987). For example, applying toxicity data for freshwater species to marine systems is commonplace throughout the ANZECC/ARMCANZ (2000) guidelines, and elsewhere (*e.g.*, Leung *et al.* 2001), because there is insufficient data from marine species to derive a specific trigger value for many toxicants (Warne 2001). In such cases, the derived water quality guidelines may be considered as interim values for marine systems until more specific toxicity data become available and appropriate criteria derived (Warne 2001).

Importantly for groundwater ecosystems, the approach taken by Hose (2005), and the resulting water quality guidelines, give no less protection than is currently provided by the broad application of water quality guidelines for surface waters (ANZECC/ARMCANZ 2000). With the exception of atrazine, the PC95 values (from which water quality guidelines are derived) for the surrogate groundwater fauna were less than or not significantly different from those derived for surface water fauna (Hose 2005).

Humphreys (2007) rightly recognizes that any comparison of surface and groundwater fauna is likely to be confounded by phylogenetic differences unless closely related species occur in each group. Within the context of the SSD approach, the phylogenetic relationships among taxa in the epigean and hypogean groups are not a major concern. What is important is that the range of sensitivities of the surrogate groundwater fauna used is representative of the range of sensitivities of true groundwater fauna (Posthuma *et al.* 2002b). If this is the case, then the approach taken in Hose (2005) is justified.

The SSD approach does not compare species assemblages *per sé*, but uses the sensitivity distribution of the assemblage to determine a concentration that is protective of a specified proportion of species (Posthuma *et al.* 2002a), which may then be compared. Consequently, I do not show (Hose 2005) that hypogean species are more (or less) sensitive than epigean fauna, but rather I show that the assemblage of fauna in groundwater ecosystems contains (at least) some fauna that are more

(or less) sensitive than the fauna in the epigean assemblage. It is the more sensitive species occupying the left hand side of the sensitivity distribution that influence the resulting water quality guideline value, particularly if asymmetrical distributions are fitted.

The overall lower metabolic rates of hypogean fauna may invalidate the use of surface water fauna as a surrogate *if* the lower metabolic rate confers differential sensitivity to toxicants. Simćić *et al.* (2005) showed that the hypogean amphipod *Niphargus rhenorhodanensis* had a lower respiratory rate, and consequently a lower metabolic rate than the epigean amphipod *Gammarus fossarum*. However, Plénet (1999) and Canivet *et al.* (2001) showed *G. fossarum* to be more sensitive than *N. rhenorhodanensis* to Cu, Zn, and As over 10–12 day exposures. However, both species were equally sensitive to Cr (Canivet *et al.* 2001). In contrast, Mosslacher (2000) showed that the hypogean isopod, *Proasellus slavus vindobonensis*, had a low respiratory rate, and was more sensitive to toxicants than two epigean copepods with higher respiratory rates, although this comparison was confounded by coarse phylogenetic matching.

Analyses at an assemblage level also suggest that a lower metabolic rate does not necessarily lead to greater sensitivity to pesticides. If we assume that because the ambient temperature of the tropics is greater than in temperate areas, the fauna of tropical areas will have generally higher metabolic rates. Interestingly, Maltby *et al.* (2005) show no significant difference in the water quality guideline (HC5) values derived for these groups. In this case, the relative metabolic rates of taxa in the assemblage are not directly related to their sensitivity distributions. However, the overall variability in the relationship between metabolic rate and sensitivity at the assemblage and population levels (described earlier) supports Humphreys' suggestion that the direction of the bias caused by using epigean species for risk assessment is unknown.

The low respiratory and metabolic rates of hypogean taxa may lead to reduced uptake of toxicants (Plénet 1999), particularly those that are actively accumulated and for which the rate of uptake is metabolically dependent. The corollary is that a low metabolic rate also infers limited capacity for organisms to metabolize or depurate toxins. Although rates of uptake and elimination may be balanced for toxins that are actively accumulated, the uptake of toxicants that are accumulated passively, such as metals (Rainbow and Dallinger 1993), may exceed the low metabolic capacity of hypogean organisms for depuration or immobilization. This scenario would make hypogean species more susceptible to toxicant exposure than epigean relatives.

Humphreys (2007) argues that because groundwater fauna inhabit a low energy, low PO_2 environment, they may be particularly disadvantaged relative to surface fauna if toxicant exposure induces an increase in respiratory and metabolic rates. It may be particularly difficult for hypogean fauna to reclaim an oxygen debt in the hypoxic groundwater environment. However, hypogean fauna are well able to deal with hypoxia (Malard and Hervant 1999). Consequently, hypoxia is unlikely to exacerbate changes in metabolic rate due to toxicant exposure because hypoxia is likely to be within the range of tolerance of many hypogean taxa. Mosslacher (2000) showed that neither the sensitivity of hypogean species to toxicants, nor their respiratory rates varied significantly under normoxic and hypoxic conditions. Unfortunately the interaction of these factors was not tested.

It appears that any interaction between respiratory rate and toxicant exposure in hypogean organisms may depend on the exposure regime. For example, Hopkins *et al.* (2003) found no change in standing metabolic rate between mosquito fish that were exposed to Hg and those that were not. This contrasts to earlier work showing a change in metabolic rate induced by acute Hg exposure (Tatara *et al.* 2001). Hopkins *et al.* (2003) suggest that changes to gills caused by acute exposure caused an increase in metabolic rate, but chronic exposures that did not cause gill damage did not cause a change in metabolic rate. In extrapolating this to groundwater ecosystems, the metabolic and respiratory consequences of exposure for hypogean fauna may be related to the exposure concentration and duration. Unfortunately, pesticides have been found at acutely toxic concentrations in groundwater (Hose 2005), hence increased respiration and implications of hypoxia may be significant.

Fat stores in aquatic organisms may act as a sink for lipid soluble toxicants, and the lipid content of an individual can influence its bioaccumulation (Kooijman 2003). For example, fish with a high lipid content accumulated more pentachlorophenol and PAHs, and had lower elimination rate constants than those with lower lipid content (van den Heuvel *et al.* 1991). However, this did not translate to a difference in LC50 values between high- and low-lipid content groups (van den Heuvel *et al.* 1991). In contrast, Geyer *et al.* (1993) showed a positive linear relationship between the log LC50 value for dieldren and the lipid content of mosquito larvae, suggesting that animals with greater lipid content can store and tolerate higher dieldren doses than animals with a lower fat content.

For hypogean invertebrates, greater fat stores imply that the retention of toxicants may be higher than in epigean species that have lower lipid contents, but the toxic response lesser. However, data on the influence of fat stores on toxicity is equivocal and is likely to be dependent on the toxicant, and its mode of uptake and place of storage in the animal (Connell 2001). Despite having slower uptake rates, the potential for bioaccumulation by lipid rich hypogean animals may be exacerbated by the high fat content and because of their often greater life spans and thus longer exposure compared to epigean species (see Plénet 1999). Although I make no attempts to discuss long-term (chronic) exposures and impacts (Hose 2005), these may be most relevant for hypogean fauna because their lower metabolic rates necessitate much longer exposures for toxic effects to be evident than for surface fauna (Plénet 1999).

Predicting the response of groundwater fauna to toxicants is difficult given the unique physiology of hypogean fauna, the low energy and PO_2 environment, and the toxicant-specific attributes such as mode of action and fate in the hypogean environment. Consequently, it is difficult to predict the relative response of surface and groundwater fauna to a toxicant, and there is little evidence available to demonstrate a systematic bias in using toxicity data for epigean fauna as a surrogate in the SSD approach. Indeed, the use of toxicity data for epigean taxa as a model in the absence of data for true groundwater taxa is the only appropriate approach available at this time. The discussion provided by Humphreys (2007) has highlighted important considerations for toxicity testing if we are to address the current dearth of groundwater toxicity data. His comments also highlight gaps in our knowledge of hypogean physiology that, if filled, will allow us to better model the likely impacts of hypogean fauna based on toxicity data for epigean relatives (*e.g.*, Traas *et al.* 2002).

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The combination of improved modeling of epigean data, and new knowledge on the sensitivities of true hypogean fauna sensitivities will provide increasingly reliable SSDs and ultimately more robust assessments of ecological risk for groundwater ecosystems.

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