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Exposure-Pathway Models Explain Causality in Whole-Sediment Toxicity Tests

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Measurements of lethal effect concentrations (LC50) and bioaccumulation following water-only and whole-sediment exposures of the amphipod, *Melita plumulosa*, and the bivalve, *Tellina deltoidalis*, to copper, were combined with bioenergetic-based kinetic models of exposure pathways to explain causality in whole-sediment toxicity tests. For both organisms, lethal body concentrations (LBCs) were greater for water-only exposures than for sediment exposures and indicated that the rate of copper accumulation and/or the mode of toxicity of copper assimilated were different for dissolved and particulate phases. The net assimilation of copper, expressed as a lethal exposure concentration (LEC) that was independent of the postexposure copper efflux, was shown to better explain the observed toxicity. The LEC of copper was the same for both water-only and whole-sediment toxicity tests. It is predicted that, for each species, a large range of effect concentrations may be measured for sediments having the same total copper concentration. These are conditional effect concentrations, as their value will be determined by total copper concentrations, partitioning (K_d) relationships (sediment properties), organism physiology (uptake rates from waters, assimilation efficiencies from solids), and organism feeding behavior (feeding selectivity). The importance of these factors to the development of sediment quality guidelines for metals based on species sensitivity distributions is discussed.

Introduction

Unlike most water quality guidelines, sediment quality guidelines (SQGs) are not based on clear cause−effect relationships (*1*−*3*). Globally, SQGs for metals vary over several orders of magnitude (*4*). This variation may be attributed to both the range of different approaches that have been used to calculate the SQGs and the quality and selection of data used in the calculations. Most SQGs have been derived from relationships between total sediment metal concentrations and field-observations (*5*) or derived as toxicity thresholds from toxicity test results with natural or spiked sediments (*2*) and do not consider factors influencing the bioavailable fraction of total metals in sediments. Attempts to incorporate information on metal bioavailability into SQGs by use of equilibrium partitioning (EqP) relationships, for example, AVS (acid-volatile sulfide) as a modifying factor, have had limited success due to the many other factors that influence metal bioavailability and toxicity (*4*, *6*).

To develop widely applicable SQGs for metals, a good understanding is needed of cause−effect relationships for a range of benthic organisms. The sensitivity of benthic organisms to metals in waters (pore waters and overlying waters) and sediments needs to be evaluated, and the relative importance of the different metal exposure pathways to the organisms needs to be understood. Furthermore, this understanding should extend to a range of sediments with varying properties (particulate phases, detritus, and food sources) and conditions that influence metal partitioning between sediment and water and metal speciation. Recent studies have indicated that metals in sediments (including food) were more important than pore water metals for body burdens in the amphipod, *Monoporeia affinis* (*7*).

Recently, the sensitivity of the amphipod, *Melita plumulosa,* and the bivalve, *Tellina deltoidalis*, to dissolved and particulate copper was evaluated using water-only and whole-sediment toxicity tests (*8*, *9*). These studies found that both dissolved and particulate copper will contribute to the toxicity of most naturally contaminated sediments. Bioenergetic-based kinetic models that describe copper bioaccumulation by *M. plumulosa* and *T. deltoidalis* during exposure to copper in water, sediments, and algae (food) were developed (*10*). These exposure pathway models highlighted the importance of the partitioning of copper between the sediments and waters (K_d), and organism feeding behavior (ingestion rates, selectivity of feeding), in controlling the total copper exposure these organisms would receive during whole-sediment toxicity tests.

This study uses the existing lethal effect concentration data (LC50s) and the models of copper bioaccumulation to develop cause−effect relationships for *M. plumulosa* and *T. deltoidalis* exposures to copper during water-only and whole-sediment toxicity tests. Copper accumulation predicted from the models was compared to copper body burdens measured following water and sediment exposures in toxicity tests. The toxicity of copper from water and sediment exposures was quantified in terms of lethal body concentrations (LBCs, net accumulation after considering uptake and efflux rates) and lethal exposure concentrations (LECs, the net assimilation; efflux not considered). The expression of copper toxicity by sediment-dwelling organisms is shown to be complex and dependent on sediment-water partitioning (K_d), organism feeding behavior, physiology of metal assimilation (from ingested solids), as well as total copper concentrations. The importance of these factors to the development of SQGs for metals based on species sensitivity distributions is discussed.

Methods

Test Organisms. The amphipod *Melita plumulosa* (family Melitidae) and bivalve *Tellina deltoidalis* (family Tellinidae) are both common in estuarine and marine environments throughout southeastern Australia (*9*). *M. plumulosa* were 2−3 month old adults (8−10 mm body length) obtained from laboratory-maintained cultures, and *T. deltoidalis* were young adults (5−10 mm in length) collected from the field, as described previously (*9*).

Sediments and Waters. Sediments with the same physicochemical properties were used for the whole-sediment toxicity tests and to develop the bioenergetic-based kinetic models that describe copper bioaccumulation by *M. plumulosa* and *T. deltoidalis* (*8*−*10*). The sediments were hydrous (68% water), silty (99% particles <63 μm), pH 7.3 ± 0.2, salinity 29 ‰, and sub-oxic (acid-volatile sulfide <0.5 μmol/g, redox

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TABLE 1. Acute Toxicity of Dissolved and Particulate Copper to *M. plumulosa* and *T. deltoidalis* (*8*)

	dissolved Cu, μg/L	
water-only	LC50 (95% CL)	NOEC
M. plumulosa	180 (30−260)	80
T. deltoidalis	130 (90−150)	70

	particulate Cu, μg/g[a]	
sediments	LC50 (95% CL)	NOEC
M. plumulosa	1300	520
T. deltoidalis	1020	650

[a] Dissolved copper concentrations were below the NOEC of 10-day water-only exposures.

potential = −40 ± 50 mV). The sediment had 12 ± 2% organic carbon (loss on ignition) and acid-extractable (30 min, 1 M HCl) metal concentrations (in μg/g) of 6000 (Fe), 50 (Mn), 160 (Zn), 66 (Pb), 30 (Cu), 4 (Ni), and <1 (Cd) (*11*). Pore water metal concentrations of all potentially toxic metals (Ag, As, Cd, Cr, Cu, Hg, Ni, Pb, Se, Zn) were low (<3 μg/L) and well below concentrations that cause effects in water-only exposures (*1*). Sediments were stored in the dark at 4 °C for a maximum of 30 days prior to use. Seawater (32.5‰ salinity) was collected from Cronulla, Sydney, NSW, and filtered through 0.45 μm membrane filters.

The copper-spiked sediments used in the toxicity tests were prepared using the procedures described by Simpson et al. (*11*) and were equilibrated for 14 days before use. Although the ^{64}Cu radioisotope (carrier free, $t_{1/2}$ = 12.7 h) used to determine the copper uptake pathways (development of the copper bioaccumulation model) was equilibrated with sediments for only 20 h before commencing experiments, the equilibration of this copper (<0.03 μg/g) occurred rapidly in these sediments due to their large copper binding capacity (*11*). Equilibrium partitioning calculations using the measured pore water, overlying water, and sediment copper concentrations indicated that K_d values ([sediment copper]/[filterable copper], L/kg) were 2 × 10^4 to 4 × 10^5 L/kg in the toxicity test experiments and (2−7) × 10^4 L/kg for ^{64}Cu in the uptake pathway experiments (confirming ^{64}Cu equilibration with the sediments).

Copper Toxicity Data. Effect concentration data (LC50s, no observable effect concentrations (NOECs)) for exposures of *M. plumulosa* and *T. deltoidalis* to dissolved and sediment copper were taken from Batley et al. (*8*) (Table 1). These data correspond to 10-day acute toxicity tests with copper-spiked seawater (30 ‰ salinity) and 10-day acute toxicity tests with copper-spiked estuarine sediments made using standard procedures (*9*). The toxicity tests were comprised of five replicate treatments (exposure containers) containing 20 amphipods or 15 bivalves. In the standardized procedures, the water-only toxicity test period is 4-days and organisms are not fed (*9*). To determine 10-day water-only effect concentrations for *T. deltoidalis*, it was necessary to feed organisms (marine diatom *Phaeodactylum tricornutum*, 5 × 10^5 cells/mL) to obtain adequate survival (unpublished results).

During sediment tests, dissolved copper concentrations in pore waters and overlying waters were generally <20 μg/L and always below the NOECs of 70−80 μg/L determined for the water-only exposures (*8*). Any toxicity observed in the sediment exposures was therefore attributed to particulate copper. In past studies of organism sensitivity to metal-spiked sediments, toxicity has often been due to pore water and overlying water metals that are present at unrealistically high concentrations (mg/L ranges) when compared to naturally contaminated sediments (low μg/L ranges) (*12*−*14*).

The organisms *M. plumulosa* and *T. deltoidalis* have sensitivity similar to copper in water and sediment toxicity tests (Table 1). Measured (0.45 μm filterable) dissolved copper concentrations were used to calculate effect concentrations (e.g., LC50s). The 95% confidence limits for the LC50s were large, typifying the high degree of variability observed for replicated tests (*8*). For *T. deltoidalis*, the 4-day and 10-day LC50s were 180 (110−240) and 130 (90−150) μg/L for water-only toxicity tests, where organisms were fed during the 10-day tests (unpublished data).

Copper Bioaccumulation Data. Copper bioaccumulation was determined for organisms surviving at the end of the toxicity tests. For water-only toxicity tests using *T. deltoidalis*, copper bioaccumulation was only determined for the 4-day tests because during 10-day water-only tests ingestion of copper-contaminated food will have affected bioaccumulation. Following toxicity tests, organisms were washed and transferred to seawater for 24 h for depuration before analysis of copper tissue concentrations. A depuration period of 24 h was chosen to allow the depuration of most nonassimilated copper while minimizing the loss of assimilated (body residue) copper and was based on the study of King et al. (*10*). After depuration, organisms were rinsed in seawater and then blotted dry on filter paper (Whatman Ashless 42) to remove excess water. Organisms were weighed wet and reweighed after drying at 60 °C for 24 h. Organisms, including the shell for *T. deltoidalis*, were placed individually in 5 mL polyethylene vials and digested in high purity HNO_3 (Trace Pur Merck, 0.5 mL/0.1 g dry weight) for 24 h at 22 ± 4 °C, and then for 20 min in a domestic microwave oven (1100 W; 10% power). After cooling, H_2O_2 was added to each vial (0.5 mL/0.1 g dry weight), and the digestion process was repeated (24 h at 22 ± 4 °C, and then for 20 min by microwave). Cooled samples were diluted with deionized water (Milli-Q, Millipore) to a final volume of 10 mL/0.1 g dry weight, and metal concentrations were determined by ICP-AES (Spectroflame EOP, calibrated against matrix matched standards). For quality control purposes, each batch of organisms analyzed included one blank and samples of the reference material TORT-2 (National Research Council Canada). Recoveries of copper in the TORT reference material were 102 ± 7 μg/g, as compared to the certified value of 106 ± 0.6 μg/g.

Model of Copper Exposure Pathways and Bioaccumulation. A bioenergetic-based kinetic model (*15*, *16*) was used to describe the copper exposure pathways and bioaccumulation of copper by *M. plumulosa* and *T. deltoidalis* (*10*). In this model, copper bioaccumulation by the organisms from filtration of water or ingestion of sediments was described by the following first-order equation:

$$dC_O/dt = (k_{u-W} \cdot C_w - k_{e-W} \cdot C_{O-W}) + (AE \cdot IR \cdot C_S - k_{e-S} \cdot C_{O-S}) \quad (1)$$

and at steady state by the equation:

$$C_{O-SS} = (k_{u-W} \cdot C_w / k_{e-W}) + (AE_S \cdot IR \cdot C_S / k_{e-S}) \quad (2)$$

where C_O is the amount of copper taken up by the organism (μg/g dry weight) for an exposure time, t (days), C_{O-SS} is the metal concentration in the organisms at steady state (μg/g dry weight), k_{u-W} is the uptake rate constant from the dissolved phase (L/g/day), C_W is the copper concentration in the dissolved phase (μg/L), AE is the copper assimilation efficiency from the ingested particles (%), IR is the ingestion rate of the organism (g/g/day), and C_S is the copper concentration in the ingested particle (C_S, μg/g). The model assumed that uptake from dissolved and sediment sources was additive. Growth of the adult *M. plumulosa* and *T. deltoidalis* during 10 days was negligible, and a growth parameter was not necessary in the model. Parameters for

TABLE 2. Bioenergetic-Based Kinetic Model Parameters for Copper Bioaccumulation by *M. plumulosa* and *T. deltoidalis* (*10*)

model parameter	units	*M. plumulosa*	*T. deltoidalis*
k_u-water	L/g/day	0.115	0.190
k_{ew}-water	/day	0.17	0.11
AE-sediment	%	7.8	30
AE-algae	%	33	49
k_{ef}-sediment	/day	0.30	0.20
k_{ef}-algae	/day	0.25	0.16
ingestion rate	g/g/day	0.20	0.08
K_d	L/kg	1×10^4 to 5×10^5	

the model are shown in Table 2. The cumulative error associated with the model parameters was estimated to be ~30% (*10*).

The model was used to predict the time required to the reach the steady-state body concentration (C_{O-SS}) for water and sediment exposures. For the 10-day water and sediment exposures, the model was used to predict the copper body burdens of the organisms (both uptake and efflux rates considered) and the total copper assimilated by the organisms (only uptake rates considered). Copper body burdens (μg/g dry weight) were calculated as the sum of background copper initially present in the test organisms and the predicted copper accumulation by the organisms (during 10 days). The total copper assimilated by the organisms (during 10 days) was used as a measure of the organism's exposure to bioavailable copper and was calculated using the equation:

$$E = ((k_{u-W} \cdot C_w) + (AE_S \cdot IR \cdot C_S)) \cdot t \quad (3)$$

where E is the organism's exposure to bioavailable copper (μg/g dry weight) and $t = 10$ days.

The lethal body concentration (LBC50) was calculated as the copper body burden of the organism at the LC50 for the water or sediment exposures (Table 1). The lethal exposure concentration (LEC50) was calculated as the organism's exposure (E) to bioavailable copper at the LC50. Both the LBC and the LEC were dependent on the copper uptake rate from each exposure source (dissolved or particulate); however, only the LBC was dependent on the copper efflux rate, postexposure.

Results and Discussion

Copper Bioaccumulation during Toxicity Tests. Body burdens measured following exposure of organisms to control waters and sediments containing no added copper (<3 μg/L and <20 μg/g, respectively) exhibited considerable variability. For *M. plumulosa*, copper body burdens in control treatments were (mean ± standard error, $n = 20$) 90 ± 4, 136 ± 6, and 91 ± 6 μg/g (dry weight) in water-only exposures and 94 ± 4, 83 ± 1, 82 ± 3, 101 ± 3, and 90 ± 3 μg/g in sediment exposures. For *T. deltoidalis*, copper body burdens in control treatments were (mean ± standard error, $n = 15$) 157 ± 15, 215 ± 22, and 151 ± 21 μg/g (dry weight) in water-only exposures and 130 ± 22, 151 ± 19, 180 ± 17, 144 ± 14, and 193 ± 14 μg/g in sediment exposures. For the bioaccumulation model, background copper concentrations of 90 μg/g in *M. plumulosa* and 165 μg/g in *T. deltoidalis* were used.

The survival of *M. plumulosa* and *T. deltoidalis* decreased with increasing dissolved and particulate copper concentrations. At copper concentrations above the LC50s, body burdens were generally more variable between replicate toxicity tests, and impaired function of surviving organism may have impeded depuration before organisms were analyzed for copper. Body burdens were determined following 10-day sediment tests for both species and 10-day water-only tests for *M. plumulosa* and 4-day water-only tests for *T. deltoidalis*. Measured body burdens of copper in *M. plumulosa* and *T. deltoidalis* following water-only and sediment exposures are shown in Figure 1.

According to the bioaccumulation model, the time required by the organisms to reach the predicted steady-state body burden (C_{O-SS}, eq 2) was 37 days for *M. plumulosa* and 40 days for *T. deltoidalis* during water-only exposures and 25 days for *M. plumulosa* and 35 days for *T. deltoidalis* during sediment exposures. At the completion of the 10-day toxicity tests, the body concentrations were predicted to have been 80% (water) and 95% (sediment) of C_{O-SS} for *M. plumulosa* and 68% (water) and 87% (sediment) of C_{O-SS} for *T. deltoidalis*. At the completion of the 4-day water-only toxicity tests for *T. deltoidalis*, the predicted body concentrations were just 48% of C_{O-SS}.

Predicted body burdens of copper in *M. plumulosa* and *T. deltoidalis* following water-only and sediment exposures are shown in Figure 1. For *T. deltoidalis*, only 4-day copper body burdens were determined for water-only exposures; however, predicted body burdens are shown for 4- and 10-day exposures. During exposures to dissolved copper concentrations of 50−200 μg/L copper, both *M. plumulosa* and *T. deltoidalis* accumulated more copper than that predicted by the model. Rapid adsorption of copper to the organisms surfaces was not considered in the calculation of the copper uptake rate constant (from water) used in the model (*10*) and may account for some of this discrepancy.

For both organisms, reasonable agreement was observed between measured and predicted copper body burdens following sediment exposures. During the whole-sediment toxicity tests, dissolved copper concentrations in pore waters and overlying waters were below the NOECs for the water-only exposures but were sufficient to contribute to measured copper bioaccumulation. For these tests, the copper bioaccumulation model indicated that dissolved copper would have contributed 17−32% and 14−17% of the copper accumulated by *M. plumulosa* and *T. deltoidalis*, respectively.

Lethal Body Concentrations of Copper. For nonessential and nonregulated metals, the expression of toxicity on a body burden basis has often been a better indicator of biological effects than the use of total sediment concentrations (*2*, *17*, *18*). For the metals Cd, Ni, and Pb, relationships between sediment toxicity and critical body burdens were found to be useful for predicting toxicity to the freshwater amphipod *Hyalella azteca* (*2*). Toxicity to *H. azteca* caged above the sediments and pore water, and with overlying water Cd, Ni, and Pb concentrations comparable to water-only effect concentrations, indicated that the toxicity was due to dissolved metal and not metals in the solid phase (*18*). Copper and zinc appeared to be regulated by *H. azteca*, and toxicity could not be related to body concentration (*2*, *18*, *19*).

The relationships between the observed toxicity (LC50) and measured and model copper body burden were used to calculate lethal body concentrations (LBC50s) for *M. plumulosa* and *T. deltoidalis* (Figure 1). For both organisms, LBCs determined from measured and predicted copper body burdens were greater for water-only exposures than for sediment exposures. This indicated that there may be different excretion processes (rate of efflux) and/or different modes of toxicity for copper assimilated from the dissolved and particulate phases.

Differences in the predicted steady-state body burden (C_{O-SS}) and the time required to reach steady state caused by different exposure pathways (dissolved or particulate sources) have important implications for the use of body burden data for predicting when metal-induced toxicity may be expected. Although body concentrations of metals change slowly, organisms living in sediments with the same total metal concentration may have a wide range of metal body burdens due to small-scale sediment heterogeneity that

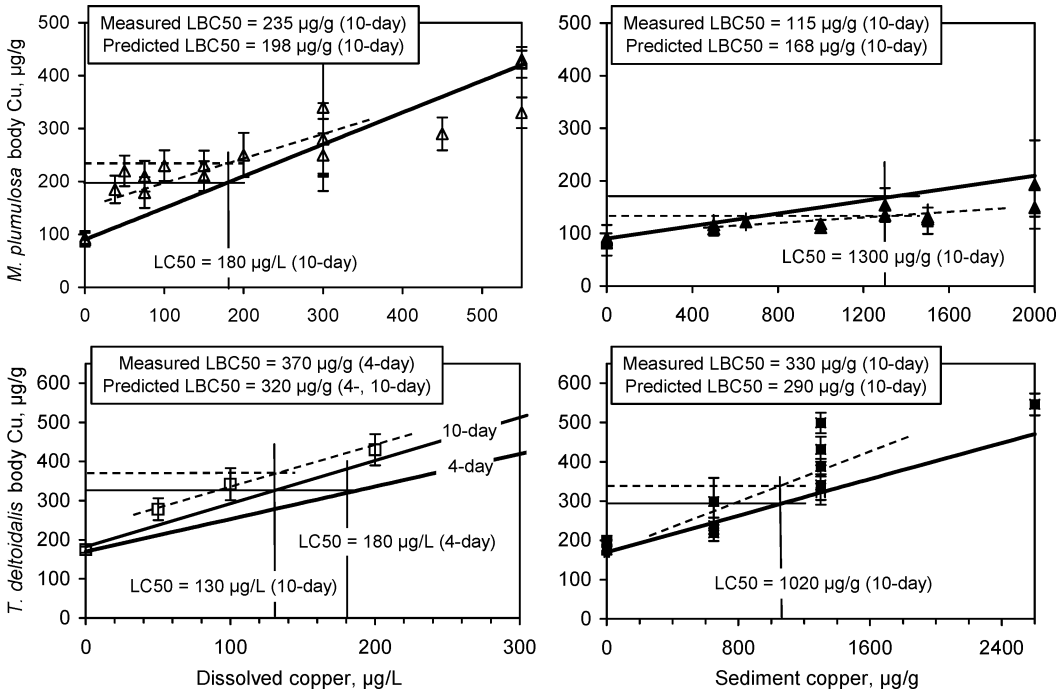

FIGURE 1. Copper bioaccumulation by *M. plumulosa* and *T. deltoidalis* during water-only and sediment toxicity tests. The symbols represent the copper body burdens measured at the end of the toxicity tests (errors bars are SE). Sediment tests were 10 days for both species. Water-only tests were 10 days for *M. plumulosa* and 4 days for *T. deltoidalis*. The diagonal lines represent the measured (dashed) and predicted body burdens (solid). The water and sediment LC50 concentrations (Table 1) are shown, together with calculated lethal body concentrations (LBC50).

changes the partitioning of metals between dissolved and particulate phases. For water-only exposures for *T. deltoidalis*, the model predicted LBC50s of 319 and 323 μg/g for 4-day and 10-day exposures, respectively (Figure 1). This indicated that exposure time should not effect the LBC for this species. The affect of exposure time on metal accumulation and metal toxicity deserves further investigation for a range of species exhibiting different exposure pathways.

The importance of considering exposure pathways when evaluating biological effects was recently highlighted by Hook and Fisher (*20*). Their studies demonstrated that, while the toxicity of silver to zooplankton occurred at dissolved concentrations of > 10 μg/L (much greater than most waters), zooplankton feeding on algae grown in waters containing 0.05 μg/L silver can cause sublethal effects. When silver toxicity was expressed on the basis of zooplankton body burden, toxicity occurred at much lower body burdens for exposure to silver from food (algae) than for exposure to silver from the dissolved phase.

Relating Copper Exposure to Effects in Whole-Sediment Toxicity Tests. In the following section, we hypothesize that toxicity occurs not due to the net accumulated copper (i.e., the LBC), but due to the organism's net exposure to bioavailable copper. The organism's exposure to bioavailable copper is defined as the amount of copper that an organism assimilates into its body, as calculated in eq 3. The exposure to bioavailable copper is a function of the dissolved (pore water and overlying water) and particulate (sediment) copper concentrations and the organism's feeding behavior (ingestion rate) and physiology (uptake rate from water, assimilation efficiency of ingested particles) and is, therefore, specific to the sediment and the organism. The efflux of assimilated copper is not considered in the calculation of *E*, as it was necessary to calculate the net bioaccumulation of copper by the organisms (eqs 1 and 2).

For 10-day water and sediment toxicity tests, the predicted exposure, *E*, to bioavailable copper for *M. plumulosa* and *T. deltoidalis* is shown in Figure 2 along with calculated lethal exposure concentrations (LEC50s). For both organisms, the calculated LEC50 values were similar for both water and sediment exposures and indicated that *E* (the bioavailable copper exposure) from water and sediment sources caused the same lethal effects to these organisms. These calculations indicate that understanding the processes that affect the "net exposure" (reflected in rate of uptake) may be more useful than understanding "net accumulation" in predicting toxic effects of metals to organisms. Although there is no practical way of directly measuring *E*, by modeling *E* we can obtain a better understanding of how metal exposure pathways may influence the observed effects (e.g., toxicity) and improve experimental design to studies these processes.

Effect of Sediment Properties on Copper Exposure Pathways and Toxicity. Sediment metals may be partitioned to a wide range of materials (e.g., organic matter, iron hydroxides, sulfides). It is well recognized that partitioning between sediments and pore waters is greatly affected by sediment properties (*11*, *13*). The distribution of metals between sediments and pore waters or overlying waters can be described by a partition coefficient, K_d = [sediment]/[water] (in L/kg). For naturally copper-contaminated estuarine/marine sediments, K_d values are typically from 1×10^3 to 5×10^5 L/kg, which covers the range from sands to silty, organic-rich sediments (i.e., from low to high densities of metal binding sites). K_d is influenced by a variety of factors, including (i) the speciation of the metals in the sediments (e.g., metal binding to AVS, particulate organic matter, and iron and manganese oxyhydroxides − influenced by redox potential, E_h), (ii) ions that compete for metal binding sites (e.g., H^+ (pH), Ca^{2+}, Mg^{2+} (hardness/salinity), Fe^{2+}, Mn^{2+} (sub-oxic sediments)), and (iii) soluble metal-complexing ligands (e.g., dissolved organic carbon, chloride). Disturbances to the sediments (by organisms and by laboratory manipulations) will also affect partitioning. Mixing will cause changes in oxidation state of previously redox-stratified sediment components ($Fe(II)/Fe(OH)_3/FeS$) and affect K_d

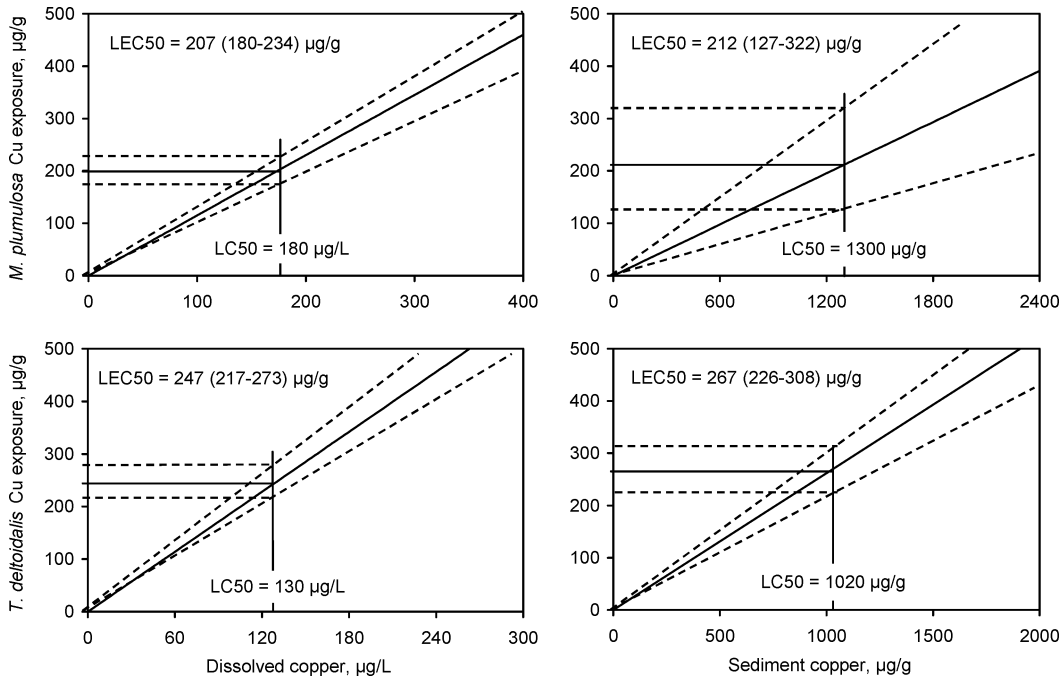

FIGURE 2. Calculated copper exposures (*E*) of *M. plumulosa* and *T. deltoidalis* during 10-day water-only and whole-sediment toxicity tests. Exposure = ($k_{u-W} \cdot C_w + AE_S \cdot IR \cdot C_S$) × 10 (days) (µg Cu/g organism tissue dry weight). The diagonal lines represent the calculated exposure (solid) and model parameter ranges (dashed). The water and sediment LC50 concentrations (Table 1) are shown, and lethal exposure concentrations (LEC50) are calculated.

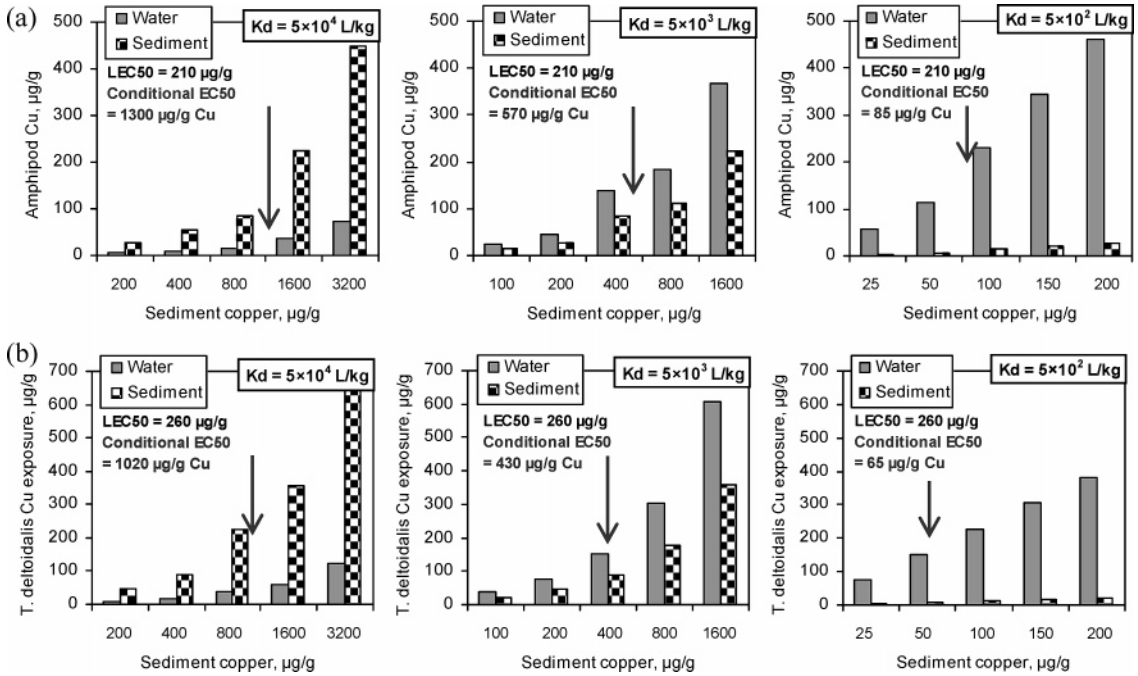

FIGURE 3. Effect of K_d (5×10^4, 5×10^3, 5×10^2 L/kg) on effect concentrations (EC_{50}) for (a) *M. plumulosa* and (b) *T. deltoidalis*. The conditional EC_{50} values are calculated as the sediment copper concentration that results in a lethal exposure concentration (LEC50) of 210 (*M. plumulosa*) and 260 (*T. deltoidalis*) µg Cu accumulated/g organism.

through the subsequent reactions of these new phases (*11*, *21*).

As discussed earlier, the predicted lethal exposure concentration (LEC50) appeared to be independent of the exposure pathway (Figure 2). However, the concentration of total copper in the sediments required to reach the LEC50 will be strongly influenced by K_d. Figure 3 illustrates the effect of K_d on the predicted exposure (*E*) that *M. plumulosa* will receive from water and sediment exposure pathways. For sediments with the same total copper concentrations, decreases in K_d from 5×10^4 L/kg (silty sediment, similar to that used in these studies) to 5×10^2 L/kg (sandy sediment, with low copper binding capacity) do not affect the exposure organisms receive from sediments; however, the exposure from the water (pore water and overlying water) increases as dissolved concentrations increase. Consequently, to achieve the same LEC, lower total copper concentrations are required in sediments with lower K_d values (Figure 3). All effect concentrations (e.g., LC50s) determined from total sediment copper concentrations should therefore be considered as "conditional" EC50 values, as they vary with sediment properties (K_d). According to the modeling, for

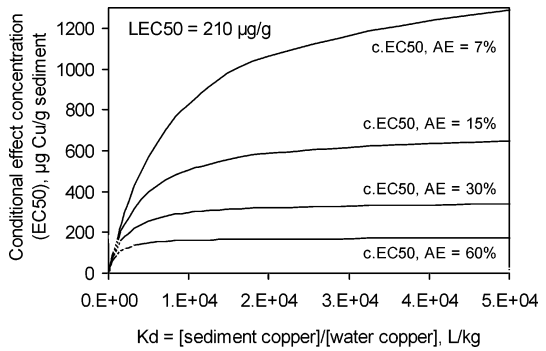

FIGURE 4. Effect of metal assimilation efficiency (AE) and sediment-water partitioning (K_d) on the conditional effect concentrations determined for *M. plumulosa* exposed to copper-contaminated sediments with a range of copper-binding phases. The conditional EC50 values were calculated using the bioaccumulation model and a lethal exposure concentration (LEC50) of 210 μg/g.

sediments with K_d values of 5×10^4, 5×10^3, and 5×10^2 L/kg, the LEC50 for *M. plumulosa* would be achieved at conditional EC50 concentrations of 1300, 570, and 85 μg/g Cu, respectively (Figure 3). Changes in sediment properties are predicted to have similar effects on the exposure and toxicity of copper to *T. deltoidalis* (Figure 3).

These calculations provide a mechanistic understanding of why individual species of organisms show a large variability in their sensitivity to sediments that have the same total metal concentrations but differing sediment properties. Besser et al. (*13*) showed that increasing the amount of organic matter in sediments increases the partitioning of cadmium and copper to the sediments and lowers the toxicity of the sediments to the freshwater amphipod, *Hyalella azteca*. Riba et al. (*22*) showed that the lethal toxicity of Cd, Cu, Pb, and Zn in sediments to the estuarine clam *Ruditapes philippinarum* increased as the overlying water pH decreased. The changes in sediment toxicity to these organisms may be explained by changes in the metal exposure occurring due to changes in metal partitioning between the pore water and sediment phases. The calculations also explain why poorly equilibrated metal-spiked sediments that have unrealistically high pore water metal concentrations are generally more toxic than naturally contaminated sediments with the same total metal concentrations (*21*, *23*).

Effect of Organism Physiology on Metal Exposure from Sediments. As well as sediment properties influencing metal partitioning (K_d), the speciation of the particulate phase (e.g., sulfides, organic matter, iron hydroxides) will also influence the assimilation of ingested particles. With respect to sediment exposure pathways, the degree of assimilation of each metal from each sediment phase will depend on the organism's physiology (e.g., gut passage time, gut chemistry) as well as the properties of the sediment phase (*24*−*26*). The influence of the metal assimilation efficiency (AE) of an organism on copper exposure pathways and conditional EC50 values was calculated for sediments with a range of metal partitioning properties (K_d) using the model for *M. plumulosa* (Figure 4). The model predicted that, as the AE of the organism increases, the importance of the sediment exposure (ingestion) pathway increases; that is, the sediments become more "toxic" and the conditional EC50 decreases. For sediments with the same total metal concentrations, relationships between conditional effect concentrations and K_d and AE are predicted to be nonlinear. The sediment K_d affects the amount of metal in the pore water and, therefore, primarily influences the exposure from dissolved contaminants. The metal assimilation efficiency (AE) of the organism affects the exposure an organism receives from particulate contaminants that are accumulated through the digestive system (*24*−*26*). Consideration of both sediment K_d values and organism AEs will therefore be necessary to make quantitative estimations of the toxicity of different sediments to benthic organisms.

Use of Species Sensitivity Distributions (SSD) for Developing Sediment Quality Guidelines. A favored approach for developing water quality guidelines is the use of species sensitivity distributions (SSDs) (*1*). Limitations and misuses of this type of approach for metals have recently been highlighted (*27*).

The present study indicates that, for each species of benthic organism, a large range of effect concentrations may be measured for different sediments with the same total metal concentration. These are conditional effect concentrations, as their value will be determined by total copper concentrations, partitioning (K_d) relationships (sediment properties), organism physiology (uptake rates from waters, assimilation efficiencies from solids), and organism feeding behavior (feeding selectivity). As relationships between the conditional effect concentrations and K_d and AE are nonlinear, SSD approaches for developing SQGs would be applicable only for sediments with similar properties. Using data in SSDs from sediments with vastly differing properties may cause large errors in estimated effect thresholds because of differences in organism exposure pathways (K_d and AE influences).

Extrapolation to Field Environments. The heterogeneity present in most sediments and the existence of microenvironments that establish their local partitioning conditions limit the accuracy of equilibrium partitioning relationships for predicting pore water metal concentrations (*28*). Our current knowledge of the biogeochemistry of organism−sediment relationships suggests that the conditions conducive to thermodynamic equilibrium partitioning may be rare (*29*). Because physicochemical equilibria are disrupted due to animal activity at small spatial (micrometers to millimeters) and temporal (seconds to minutes) scales, greater considerations need to be given to short-term kinetics (*21*, *29*).

The importance of sediment heterogeneity and dynamics extends beyond metal partitioning. The availability of food (e.g., detritus, algae) to organisms will also vary spatially and temporally in sediments. *M. plumulosa* and *T. deltoidalis* assimilate a greater percent of copper from algae (food) than from sediments (*10*). De Haas et al. (*30*) observed that increased amounts of food partially masked the toxic effects of copper on the midge *Chironomus riparius* in whole sediment bioassays. The availability of different food sources and the selectivity of organism feeding will affect organism exposure and toxic effects that influence data interpretation and risk assessment outcomes.

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