Risk Analysis and Sediment Delineation Upstream of a Dam Slated for Probable Removal

Bright ideas. Sustainable change. RAMBOLL

AUTHORS

Phyllis Fuchsman, Katrina Leigh, Kyle Fetters, *Jennifer Bell Taylor*, Renee Sandvig, Stephen Song, Pieter Booth, Ramboll

INTRODUCTION

Contaminated sediments were discovered in an impounded river that received stormwater discharges from a former manufacturing facility, following investigations related to possible removal of the impoundment's dam. For various reasons, it was necessary to understand human health and ecological risks related to the contaminated sediments before local officials reached a decision on whether to remove or repair the dam.

Objective

Conduct a human health risk assessment (HHRA) and an ecological risk assessment (ERA) for both current conditions and a future dam removal scenario. Identify which area of the site drives risks in both scenarios.

Primary contaminants of interest

- Metals: lead, zinc, cadmium, mercury, antimony
- PAHs
- PCBs

Current scenario

- Recreators wading and fishing in the river
- Benthic invertebrates, fish and aquatic-feeding wildlife

Future scenario

- River level drops and sediments become soil
- Recreators using park/open space
- Plants, soil invertebrates and terrestrial wildlife

ANALYSIS

To ensure that delineation of contaminated sediments was commensurate with needs of the HHRA and ERA, sediment and soil screening levels were derived based on the site-specific exposure scenarios to be evaluated (instead of using the state's generic residential soil screening levels).

In the HHRA, cumulative cancer risk and hazard index estimates were calculated for every sediment sample location. The ecological investigation included chronic sediment toxicity testing paired with bulk sediment and porewater analyses, as well as biota tissue sampling. Literature-based analyses to support the future risk scenario included updating wildlife toxicity values for PAHs and lead and developing methods to estimate bioaccumulation of antimony in earthworms, bioaccumulation of lead in bird blood, and mercury speciation in terrestrial plants and invertebrates.

In this poster, we focus on lines of evidence which are directly measured rather than modeled because they provided new insights into risk analysis.

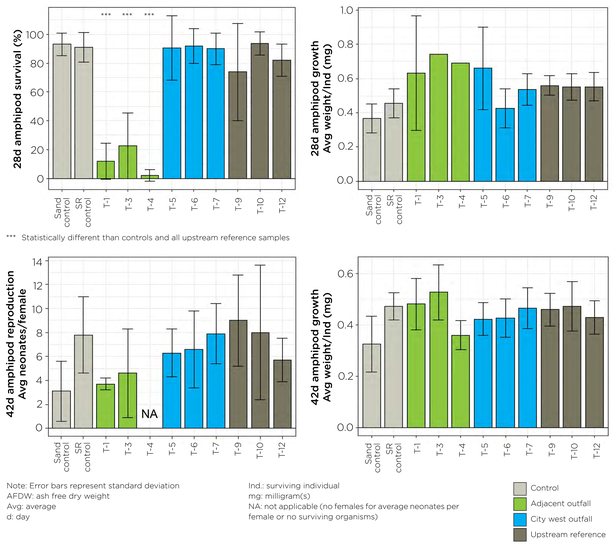
Sediment toxicity testing

Sediment toxicity was evaluated using two sediment bioassays, the 42-day *Hyalella azteca* (amphipod) survival, growth and reproduction test (Figure 1) and the 20-day *Chironomus dilutus* (midge) survival and growth test (Figure 2). Test methods followed standard guidance (USEPA 2000, ASTM E 1706). The toxicity test results indicate that sediments near the outfall (T-1, T-3, T-4) were most likely to cause toxicity (Figure 3).

Hyalella azteca (amphipod)

Chironomus dilutus (midge)

Figure 1. Sediment toxicity test results for the amphipod, *Hyalella azteca*

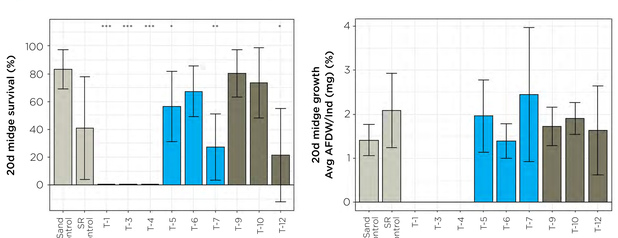


*** Statistically different from controls and all upstream reference samples

Note: Error bars represent standard deviation
AFDW: ash free dry weight
Avg: average
d: day

Ind.: surviving individual
mg: milligram(s)
NA: not applicable (no females for average neonates per female or no surviving organisms)

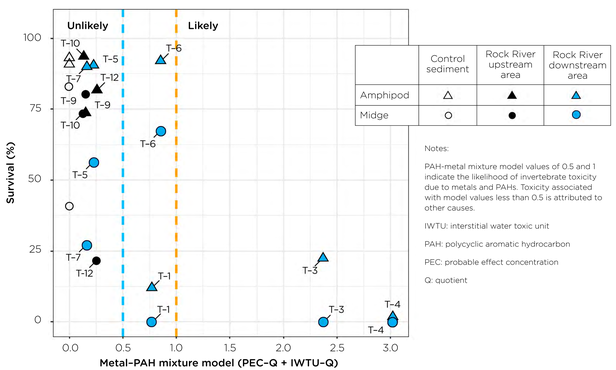
Figure 2. Sediment toxicity test results for the midge, *Chironomus dilutus*



* Statistically different than sand control only
** Statistically different than sand control and 2 of 3 upstream reference samples (T-9, T-10)
*** Statistically different than sand control and all upstream reference samples

Note: Error bars represent standard deviation

Figure 3. Interpretation of metal-PAH mixtures as a predictor of site-specific sediment toxicity



Notes:

PAH-metal mixture model values of 0.5 and 1 indicate the likelihood of invertebrate toxicity due to metals and PAHs. Toxicity associated with model values less than 0.5 is attributed to other causes.

IWTU: interstitial water toxic unit

PAH: polycyclic aromatic hydrocarbon

PEC: probable effect concentration

Q: quotient

SEM-AVS analysis

The most important factors controlling bioavailability for divalent metals in sediment are the concentrations of acid volatile sulfide (AVS) and organic carbon (OC) relative to simultaneously extracted metals (SEM) in the sample (USEPA 2005). In all toxicity test samples, the concentration of "excess" metals (SEM-AVS) normalized to fractional organic carbon content (fOC) was less than 130 µmol/gOC, thus predicting no toxicity due to metals (Table 1).

Table 1. Concentrations of total and bioavailable metals reported for toxic sediment samples

Location	Total cadmium (mg/kg)	Total lead (mg/kg)	Total zinc (mg/kg)	Organic carbon-normalized excess SEM[a] (µmol/g OC)
T-1	0.7	162	173	-466
T-3	6.3	1380	1170	1
T-4	2.5	592.0	640	-215

Non-detected SEM and AVS are included as one-half the quantitation limit.

[a] Organic carbon-normalized excess SEM = (∑SEM-AVS)/foc. Per USEPA (2005a), toxicity in certain sediments containing benthic invertebrates due to metals is very unlikely at concentrations below 130 µmol/g OC and very likely at concentrations above 3,000 µmol/g OC.

Yet, no other chemicals adequately explained toxicity in sediments containing elevated total metals. Other chemicals evaluated included PAHs and mercury in porewater, and PCBs, SVOCs, VOCs, pyrethroid pesticides, and other metals in bulk sediment.

What does the SEM-AVS track record tell us about this apparent discrepancy?

- In many cases, the SEM-AVS model **accurately predicts** benthic invertebrate responses to metal-contaminated sediments (eg USEPA 2005; Burton et al 2005; Besser et al 2008, 2010, 2013; MacDonald et al 2009; Nguyen et al 2011; Chandler et al 2014).
- The (SEM-AVS)/OC model can **overestimate risk** of toxicity in cases where for binding to iron and manganese oxides and oxyhydroxides limits metal bioavailability in oxidized sediments (Costello et al 2011, 2015).
- Sediment handling and laboratory toxicity testing conditions can cause oxidation of sulfide, changing the AVS content of the sediment and increasing metal bioavailability (Simpson and Spadaro 2011, Simpson et al 2012, De Jong et al 2012a, 2012b). In this case, measurements of (SEM-AVS)/OC that accurately represent field conditions may **underpredict laboratory toxicity**.
- Measurements of (SEM-AVS)/OC taken at both the start and completion of toxicity testing have sometimes shown **underprediction of toxicity** specifically for invertebrate species that build burrows and irrigate them with overlying water (Besser et al 2013, Chandler et al 2014). This can be a sampling artifact if oxidation occurs at the **spatial scale of the organism** (eg 0-1 cm depth) but SEM-AVS sampling occurs over a larger depth interval including reduced sediments (Vandegehuchte et al 2013).

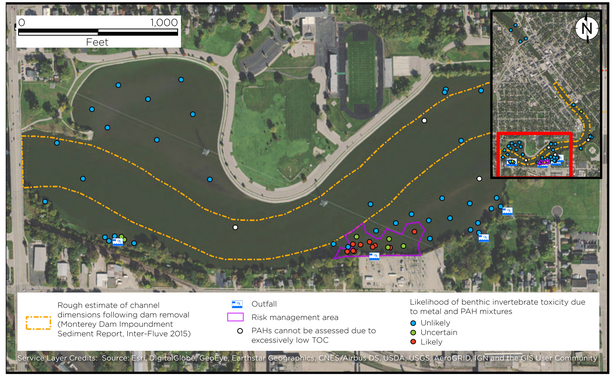
We hypothesized that our toxicity test samples became oxidized during the chronic toxicity tests, resulting in increased bioavailability compared to conditions in the river. Thus, a lesson learned was to measure SEM-AVS in test sediments at both the start and end of chronic sediment toxicity tests. For this investigation, however, we judged that the laboratory oxidation phenomenon provided a reasonably conservative approximation of potential future conditions in the event that contaminated sediments became exposed to air following dam removal, potentially resulting in toxicity to soil organisms.

DELINEATION

In the HHRA, a point-by-point risk characterization demonstrated that the only locations with unacceptable risk were limited to an area near the main stormwater outfall.

For the ERA, sediment collected near the main stormwater outfall was toxic to midges and amphipods in chronic laboratory tests. Delineation was accomplished using mean probable effect concentration (PEC) quotients for metals in bulk sediment and calculation of PAH toxic units using site-specific organic carbon-water partition coefficients. The resulting sediment management footprint was also found to be protective for fish and wildlife under current and potential future conditions.

Figure 4. Spatial distribution of metal-PAH mixtures and toxicity predictions for surface sediments



REFERENCES

Besser et al 2008. Arc Environ Contam Toxicol 54:557–570.
Besser et al 2010. USDOI, USGS. 08-NRDAR-04.
Besser et al 2013. Environ Toxicol Chem 32(11):2495–2506.
Burton et al Environ Toxicol Chem 24(3):541–553.
Chandler et al 2014. Environ Sci Tech 48:12893–12901.
Costello et al 2011. Environ Sci Tech 45:5798–5805.
Costello et al 2015. Environ Sci Tech 49:6926–6933.
De Jonge et al 2012a. Water Research 46:2205–2214.
De Jonge et al 2012b. Water Research 46: 3387–3397.
MacDonald et al 2009. USEPA.
Nguyen et al. 2011 Environ Toxicol Chem 30(1):162–172.
Simpson et al. 2011 Environ Sci Tech 30(10):2326–2334.
Simpson et al 2012. Chemosphere 88(8):953–961.
USEPA 2000. EPA/600/R-99/064.
USEPA 2005. EPA-600-R-02-00.
Vandegehuchte et al 2013. Environ Toxicol Chem 32:1048–1059.