Bull. Environ. Contam. Toxicol. (1995) 54:676–682

Environmental Contamination and Toxicology

Use of Toxicity Identification Evaluation Procedures in the Assessment of Sediment Pore Water Toxicity from an Urban Stormwater Retention Pond in Madison, Wisconsin

M. Wenholz, R. Crunkilton

College of Natural Resources, University of Wisconsin–Stevens Point, Stevens Point, Wisconsin 54481, USA

Received: 13 September 1993/Accepted: 4 December 1994

Biological tests with sediments are an efficient means for evaluating sediment contamination because they provide information complimentary to chemical characterizations (Burton and Scott 1992). Recently, toxicity reduction and identification procedures, originally designed for characterizing complex effluents (USEPA 1991a) have been shown to be an effective way to evaluate toxicity of sediments (Giesy and Hoke 1990; Ankley et al. 1990; Schubauer-Berigan and Ankley 1991). Toxicity identification evaluations (TIEs) consist of three phases and are described by Burkhard and Ankley (1989). This study focused on the first phase of the TIE, which are manipulations designed to characterize classes of toxicants (USEPA 1991a).

This study examined effects of one type of urban pollution in sediments; toxicity of stormwater runoff contaminants which collect in urban retention pond sediments. Urban ponds may be significant non-point sources of trace metals to aquatic organisms (Liston and Maher 1986) and metals are often more highly concentrated in sediments than in the water column (Mathis and Cummings 1973; Solomons et al. 1987). The specific objective was to determine the biological effects of runoff contaminants found in interstitial pore water of sediments in an urban stormwater retention pond in Madison, Wisconsin to Ceriodaphnia dubia and to classify the type(s) of contaminant(s) causing toxicity through toxicity identification evaluations (TIEs). Chemical analyses were also performed on sediment pore water and overlying water to aid in characterizing contaminants.

Interstitial pore water was used in this study because 1) it is better than elutriates for predicting bulk sediment toxicity from the aqueous sediment fraction (Schubauer-Berigan and Ankley 1991; Ankley et al. 1991a), 2) pore water is a primary uptake route by many aquatic organisms (Burton 1991; Burton and Scott 1992), 3) common surface water test organisms (Daphnia magna and Ceriodaphnia dubia), which have well established data bases and are sensitive species can be used in pore water tests (Giesy and Hoke 1990; Burton 1991), and 4) toxicity caused by metals (Schuytema et al. 1984) and organics (Knezovich and Harrison 1988) can be predicted from pore water concentrations of contaminants.

MATERIALS AND METHODS

A stormwater retention pond (0.60 Ha) was constructed in 1981 within the city of Madison, Wisconsin. The drainage area (94 Ha) consists of medium density

Correspondence to: R. Crunkilton

residential housing (97%) and commercial use (3%). Traffic density in the drainage area is approximately 20,000 vehicles per day on arterial streets and 4,500 vehicles per day on collector and feeder streets. Sediment has accumulated in the pond at a rate of approximately 1 inch per year.

Six sediment samples were obtained in a random fashion on July 17, 1992 with an Ekman grab and composited in a 20-L pail. Sediment had an organic carbon content of 12% and was classified as a silty loam. Water depth and dissolved oxygen were recorded at each site (average depth of 1.1 m and average D.O. of 2.1 mg O_2/L). Overlying water in the pond was also collected and used for dilution purposes during testing. Samples were refrigerated at 4°C until used (2 weeks to 5 months). Overlying water used for determining metal concentrations in the water column was collected on April 18, 1993.

Aquatic life was limited in the retention pond. One goldfish (Carassius auratus) and several annelid worms were the only aquatic life found occurring in the pond. The pond had no submersed or emergent aquatic vegetation, but was attractive to avian species characteristically observed near wetlands.

The composited sediment was placed in 250-ml bottles and centrifuged for 30 min at 1800x. The supernatant was poured from the bottles, recentrifuged for 20 min at 1800x and refrigerated at 4°C until used (within 24 hr).

Initial toxicity was determined in a 48 hr acute test with Ceriodaphnia dubia. Treatments followed simple serial dilutions between 100% and 12.5% pore water. Dilution water came from the retention pond. Both lab culturing water and overlying retention pond water were used as controls in each test. Pore water samples were aerated for 10-15 min and allowed to warm to room temperature, as were the overlying water samples, before test dilutions were made. Tests were run at 25°C and ambient laboratory light. In each, two replicates of 10 C. dubia (< 24 hr old) were exposed per treatment. Organisms were fed yeast cerophyll trout food and Selenastrum capricornutum prior to the start of tests (USEPA 1991b). Acute toxicity data were fit to a probit model to calculate the LC50 (USEPA 1991b).

Toxicity identification evaluation (TIE) tests use organisms to detect the presence of toxicants. The procedure includes an assessment of the toxicity of the original sample followed by a series of physical and chemical manipulations designed to reduce or eliminate toxicity. Toxicity identification tests performed were slight modifications of standard protocol (USEPA 1991a). The modifications were changes in the volume of sample used for each test due to the small amount of sample available when using pore water extractions. The procedures consisted of pH adjustment to 3 and 11 followed by filtration, aeration, and C-18 (octadecyl) solid phase extraction; oxidation/reduction with sodium thiosulfate (1.608 g/l) and EDTA (0.438 g/l) chelation (water hardness determines concentration of EDTA used); and a graduated pH test where pH was increased. The initial pH (pHi) for all tests was 8.1. Following pH adjustment to 3 and 11 (pH 9 for C-18 extraction) and physical or chemical treatment, sample pH was returned to pHi with 1.0, 0.1, and 0.01 N NaOH and HCl prior to toxicity testing. Approximately 200 ml of pore water extract was filtered through the C-18 column (Baker Chemical Co.). Two aliquots of 40 ml were collected after 25 and 150 ml of pore water had been passed through the C-18 filter. These two aliquots were used for toxicity testing and the remainder of the filtrate was discarded. All chemicals were reagent grade or higher. All tests, except for the graduated pH test, were performed with 5 C. dubia (<24 hr) in 30-ml plastic cups filled with 10-ml of test solution. There were 2 replicate cups per treatment. The graduated pH test was performed with 5 C. dubia (<24 hr) in 6-ml capped vials filled completely with test solution to exclude air which could alter

pH. There were four replicates for each treatment. Baseline toxicity tests at pHi were performed with each manipulation to check for changes in toxicity between the time the initial toxicity test and the TIE tests were performed. All tests lasted 48 hr.

Total ammonia and pH were measured with an Orion ISE meter (model 920A) with an Orion ammonia ion electrode. The concentration of unionized ammonia in the pore water sample (as mg NH_3-N/l) was calculated from the amount of total ammonia present and pH (Emerson et al. 1975). Calcium and Mg hardness was measured as $CaCO_3$ (HACH Chemical Co.). Dissolved oxygen was measured with a Model 57 Yellow Springs Inc. oxygen meter. Total metals (Cd, Cu, Zn, Cr, Hg, Pb, Ni and Fe) from centrifuged pore water and overlying water samples were prepared following Method 3111 APHA (1989) and read on a Varian Spectr AA 20 spectrophotometer with GTA-96 graphite tube atomizer.

RESULTS AND DISCUSSION

The initial LC50 was 62% pore water with a 95% confidence interval between 57% and 65%. There was no mortality in dilution water (overlying pond water and lab culture water) controls.

Baseline tests were run at three concentrations of pore water [(100%, LC50 (62%), and .5 x LC50 (31%)] and two controls (overlying pond water and lab culture water) for the pH adjustment tests. Mortality (80%) in the baseline test at pHi was observed only at the 100% pore water concentration, therefore, all further discussion pertains only to this concentration (Fig. 1). Adjustment to pH 3 removed toxicity (0% mortality), while adjustment to pH 11 did not (90 % mortality). Large flocs of chemical precipitate formed when pH was either dropped to pH 3 or raised to pH 11. The floc in the pH 11 treatment redissolved when the pH was readjusted to pHi. The floc remained in the pH 3 treatment after readjustment to pHi.

There were no mortalities after filtration of both the pH 3 and pH 11 treatments. Acute toxicity remained high (70% mortality) in the baseline test which was a filtered sample at pHi without pH adjustment.

Aeration at pH 3 removed toxicity (0% mortality) and greatly reduced toxicity (10% mortality) at pH 11, but toxicity was not reduced in the aerated sample at pHi.

No mortalities were observed after post C-18 (octadecyl) solid phase extraction column collections in both pH 3 and pH 9 treatments. Acute toxicity remained in the post C-18 solid phase extraction column collections in the pHi treatments (90% and 100%, respectively).

Baseline tests were run at three pore water concentrations (100%, LC50 (62%), and 50%) and 2 controls (overlying pond water and lab culture water) for the oxidant/reduction and EDTA chelation tests. Mortality (100%) was observed only at the 100% pore water concentration (Fig. 2). Both oxidant/reduction and EDTA chelation tests used gradient additions of stock standards to 100% pore water samples. Toxicity was slightly reduced (100% to 70-90%) in the higher sodium thiosulfate additions (0.4, 0.6, 0.8, and 1.0 ml) of the oxidant/reduction test. The lowest sodium thiosulfate treatment (0.2 ml) resulted in 100% mortality. Toxicity was reduced slightly (100% to 80-90%) at intermediate concentrations of EDTA.

The graduated pH test showed an increase in toxicity of 100% pore water with an increase in pH (pH 7 = 0% mortality, pH 8 = 10.5% mortality, pH 8.5 = 90% mortality). Knowing that the baseline pHi was 8.1, it appears that a sharp pH

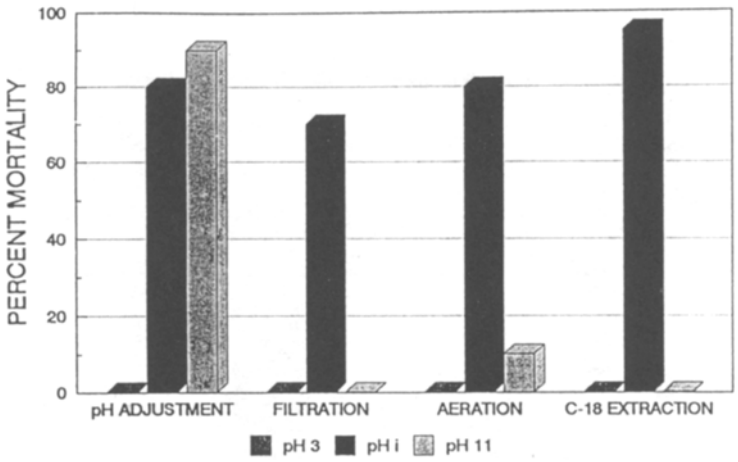

Figure 1. Bioassay results following pH adjustment of the 100% pore water. pHi is 8.1.

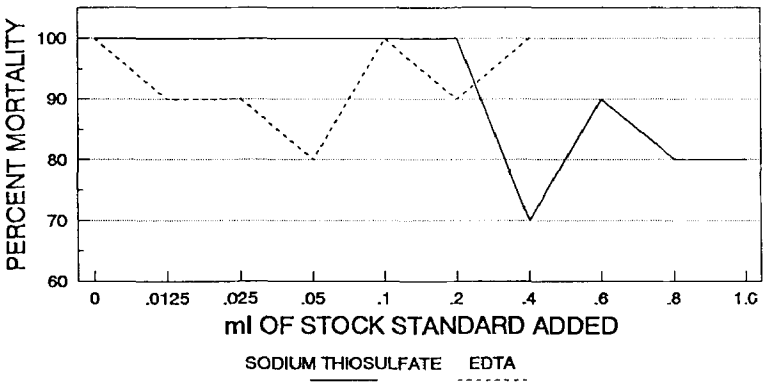

Figure 2. Bioassay results following oxidant/reduction and EDTA chelation treatments of 100% pore water

gradient (i.e. 0.1 - 0.2 units) drastically changes the toxicity of the pore water.

Nearly all metals which were analyzed were at much higher concentrations in the pore water than in the overlying water column (Table 1). Two of the tested metals (copper and zinc) had pore water concentrations higher than published acute toxicity criteria for surface water in the state of Wisconsin (Table 1). These criteria were derived from equations published by the Wisconsin DNR (1989) which take into account water hardness which was 170 mg $CaCO_3$/l in pore water. Iron in pore water greatly exceeded surface water criteria (American Fisheries Society 1979). The unionized ammonia concentration in the pore water was 0.599 mg/l NH_3-N,

which is also above the criteria limit of 0.37 mg/l at a pH of 8.0 and 25°C for warmwater organisms (USEPA 1984).

Table 1. Metal concentrations (mg/L) in pore water and overlying water and the acute toxicity criteria value.

Metal	Pore water concentration	Overlying water concentration	Acute toxicity criteria value
Cd	<0.002	<0.002	0.052 [2]
Cu	0.060	<0.010	0.027 [2]
Zn	0.195	0.210	0.161 [2]
Cr	N/A	<0.020	2.88[a]/0.014[b] [2]
Hg	<0.0002	0.0003	0.0000012 [1]
Pb	0.078	0.010	0.332 [2]
Ni	0.089	<0.020	1.681 [2]
Fe	20.100	0.098	1.00 [1]

[1] = EPA water quality criteria (American Fisheries Society 1979)
[2] = Wisconsin DNR water quality criteria (1989)
[a] = Total recoverable chromium (+)
[b] = Total recoverable chromium (+6)

Toxicity in the pore water appeared to be caused by multiple factors. Toxicity was eliminated or reduced when the pH was lowered to pH 3 or raised to pH 11 and filtered. Changes in pH affect solubility, polarity, stability and speciation of a compound, thereby affecting its bioavailability and toxicity (USEPA 1991a). C-18 extraction did not reduce toxicity (beyond the reduction observed by pH manipulation and filtration prior to C-18 extraction), therefore, nonpolar organics or nonpolar metal chelates are unlikely contributors to toxicity. Because several of the tested metals exceeded acute toxicity criteria, it appears that some elimination/reduction of toxicity was due to adsorption of toxicants (perhaps metals) to the precipitated floc when the pH was adjusted. Mortality was high (90%) in the pH 11 treatment. When this treatment was readjusted to pHi with HCl, the floc that had formed when base (NaOH) was added to raise the pH to 11, dissolved and again resulted in toxicity. Filtration at pH 11 removed this toxicity, therefore, it appears that the toxicity was associated with substances bound to the floc. The floc remained and no mortalities were observed in the pH 3 treatment after it was readjusted to pHi. It appears the substance(s) contributing to toxicity were irreversibly altered by a drop in pH and bound to the floc.

Further evidence for metal toxicity was shown by a reduction in toxicity following sodium thiosulfate and EDTA addition. Sodium thiosulfate acts as a chelating agent for some cationic metals and EDTA is a chelator for Cd, Cu, Fe, Pb, Ni, and Zn

(USEPA 1991a). The graduated pH test may also suggest metal toxicity. Zinc, nickel and cadmium have been shown to be more toxic at pH 8.5 than pH 6.5, while copper was shown to be more toxic at pH 6.5 than pH 8.5 (USEPA 1991a). The concentrations of Zn and Cu in our pore water samples exceeded the acute toxicity criteria for these metals. We cannot rule out zinc as a contributor to toxicity at higher pH values in the graduated pH test because toxicity was not completely removed at higher pH values following aeration (which should volatilize unionized ammonia).

Ammonia also appeared to contribute to the toxicity of pore water. Mortality increased as pH of the 100% pore water was increased. The unionized form of ammonia predominates at higher pH values (Emerson et al. 1975) and is more toxic than the ionized form to C. dubia.

The concentration of iron in pore water greatly exceeded criteria (American Fisheries Society 1979) set for freshwater aquatic life (Table 1) and may also have played a role in the toxicity of the pore water. However, since few analyses were performed to determine the specific physical and chemical nature of the sediments such as sorption kinetics, acid volatile sulfides, manganese oxide, and sulfate concentration, it is impossible to determine the exact adsorption and partitioning activities of iron or other metals in the test sediments. Although several metals exceeded acute toxicity thresholds, acid volatile sulfides may eliminate toxicity due to the "free" or soluble form of the metal ion by forming a metal-sulfide precipitate (Di Toro et al. 1990; Ankley et al. 1991b; Ankley et al. 1993). Further studies are needed to determine the adsorptive nature and partitioning of metals in the test sediments.

In conclusion, toxicity of the sediment pore water from the stormwater retention pond in Madison, Wisconsin appears to be caused by ammonia, and to a lesser extent, metals. Biological and chemical evidence suggests Zn and Fe, and possibly Cu, are the chief metal contaminants, however, further study would be needed for confirmation. Strong evidence for metal toxicity appeared to be obscured by the presence of ammonia. These metals are common contaminants in surface runoff from urban areas and high concentrations appear to be retained in the sediments of stormwater detention basins (Liston and Maher 1986). Elevated ammonia concentrations are commonly associated with anoxic conditions within sediments, which agrees with the low D.O. and high organic carbon readings. Low concentrations of ammonia may interact with other sediment-associated contaminants, thus complicating interpretation of sediment toxicity tests (Ankley et al. 1990). New methodology needs to be developed to deal with the phenomenon of ammonia interactions with other potential contaminants in sediment samples. Further studies should be conducted to more clearly assess the nature of the toxicity associated with sediments in the stormwater retention pond.

Acknowledgments. This project was partially funded by two student research grants from the University of Wisconsin-Stevens Point. The authors would like to specially thank the Environmental Task Force state lab at UW-Stevens Point for performing metal analyses, Brad Roost for helping with the pH reduction tests, Roger Bannerman for providing technical information concerning the retention pond, and the Green Bay Metropolitan Sewerage District and the National Oceanic and Atmospheric Administration for aiding in the preparation of this paper.

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