Sediment contaminant chemistry and toxicity of freshwater urban wetlands

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ABSTRACT

Wetlands provide many critical functions in urban ecosystems, including habitat for wetlanddependent fauna and enhancement of water quality. Interest in restoring or creating wetlands to enhance these functions is increasing due to the scale and extent of wetland loss and water quality problems associated with urbanization. One of the most pressing questions associated with urban wetland restoration is to what extent urban wetlands tend to concentrate contaminants, and if so, is there an associated risk to wildlife. The goal of this study was to better understand these potential risks, and the associated trade-offs with using wetlands to passively or actively treat urban runoff. Sediment toxicity, chemistry, and benthic community metrics were measured in 21 southern California wetlands that receive urban runoff as their primary water source. Benthic organisms in 18 of the 21 urban wetlands examined were considered to be at risk due to sediment contaminants. Most of the sites were either toxic to the amphipod Hyalella azteca, exceeded a sediment quality guideline, or both. Sediment chemistry and toxicity identification evaluation studies (TIEs) suggest that pyrethroid pesticides may have been responsible for much of the toxicity documented in this study. The mean Probable Effects Concentration quotient (mPECq; an index of degree of sediment contamination) was found to negatively correlate with benthic macroinvertebrate diversity in these wetlands suggesting that toxicity was affecting organisms at the base of the food chain in these wetlands. Sediment toxicity and chemistry concentrations in treatment wetlands were not significantly different than that observed in habitat wetlands.

INTRODUCTION

Over the past century, increasing urbanization of watersheds has resulted in a large loss and degradation of wetlands and riparian areas (Dahl 1990, Holland *et al.* 1995). Concurrent with the loss of wetland habitat, increased runoff from urbanized watersheds and discharges of point or nonpoint source pollution have created a demand for effective, low-cost solutions to improve surface water quality and attenuate storm flows. As a result, there is increasing interest in restoring, enhancing, and creating natural or constructed wetlands with multiple objectives, e.g., habitat support, treatment of nonpoint source pollution, flood attenuation, and recreation (Azous and Horner 2000).

Because urban runoff is recognized as a major source of contaminants (Characklis and Wiesner 1997, Paul and Meyer 2001) and because wetlands can accumulate contaminants over time, there is concern that the risk to wildlife from contaminant toxicity may outweigh the benefits that these urban wetlands provide as habitat (Helfield and Diamond 1997, Wren et al. 1997). Bioaccumulation of contaminants and toxicological effects on wildlife have been documented for wetlands receiving nonpoint source runoff (Ohlendorf et al. 1989, Schuler et al. 1990, Welsh and Maughan 1994, Glenn et al. 1999, Garcia-Hernandez et al. 2001). Impacts can include direct toxicity to algae and aquatic plants, wetland fauna including wetland invertebrates, amphibians, reptiles, fish, and birds, resulting in the loss of biodiversity and simplification of the food chain (Wren et al. 1997, Adamus et al. 2001). These impacts have been demonstrated for organophosphorus and pyrethroid pesticides (Katznelson et al. 1995, Harris et al. 1998), polyaromatic hydrocarbons (PAHs;

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Maltby *et al.* 1995), polychlorinated biphenyls (PCBs; Dunier and Siwicki 1993, Wren *et al.* 1997, Adamus *et al.* 2001;), and heavy and trace metals such as Hg, Pb, Zn, Cu, and Cd (Galli 1988, Yousef *et al.* 1990, Campbell 1994, Brown and Bay 2006). The presence and relative severity of effects depends on many site-specific factors, such as the wetland type, contaminant loading rates and concentrations, landscape position, hydrology, nature of sediment storage and transport, and structure of the biotic communities (Schueler 2000).

One of the nuances in assessing contaminant effects on wetlands is the lack of clear consensus on appropriate indicators of ecological condition (Pascoe 1993). While clearly no "gold standard" exists, sediments are a useful medium to measure contaminant exposure and its biological effects in wetlands. Three types of measurements are commonly used to integrate contaminant exposure and effects data in sediments, otherwise known as the "sediment quality triad": chemical analysis, toxicity tests and benthic macroinvertebrate community assessments (Long and Chapman 1985). Sediment contaminant concentrations are temporally less variable than water column concentrations, thus providing an integrated signal of contaminant exposure over time. Benthic macroinvertebrate community data, as well as sediment toxicity tests on macroinvertebrates, provide data on impacts to a key component at the base of the wetland food web (Adamus 2001). Toxicity tests provide a measure of bioavailability and toxicity of sediment contaminants from direct exposure, but are not complicated by many of the environmental factors that confound benthic community analysis or other measurements of contaminant effects in the field. Data from the sediment quality triad can be complemented by whole sediment TIEs, which serve to identify specific compounds or classes of compounds responsible for toxicity at the site.

The purpose of this study was to investigate contaminant exposure and effects in urban wetlands with respect to the following objectives: 1) quantify the sediment chemistry, toxicity and macroinvertebrate species richness of urban wetland sediments relative to reference wetlands; 2) determine the contaminants responsible for observed toxicity, as indicated by sediment chemistry and sediment TIEs); and 3) determine whether macroinvertebrate species richness, sediment contaminant concentrations or toxicity vary predictably as a function of degree of urbanization.

METHODS

General Study Approach

Sediment toxicity, chemistry and benthic macroinvertebrate richness were characterized for freshwater, urban wetlands created for varying objectives in southern California. The wetlands were located in six counties throughout southern California, including Santa Barbara, Los Angeles, Orange, San Bernardino, San Diego, and Imperial counties. Because no comprehensive maps of freshwater wetlands yet exist for this region, the 21 urban wetland sites and 2 reference selected for this study were identified from a master list of 43 wetland sites using information obtained from wetland owners, stakeholders, agency staff, and known projects. Sites were chosen to represent each of the three management objectives typically cited as the motivation for urban wetland restoration or creation: habitat creation, water quality improvement, or multiobjective.

Two types of wetland configurations were examined: wetland basins (depressional wetlands, usually perennial ponds that have extended retention of flows and sediment) and wetland channels (flowing channels, creeks or small streams with unrestricted outflow). The number of basin sites (12 sites) were somewhat higher than the number of channel sites (9 sites).

Characteristics of Study Sites

The study sites are located in a semi-arid Mediterranean-like climate, characterized by mild to cool wet winters, and warm to hot dry summers. The average annual rainfall is 38 cm, which accumulates mostly between December and March. Historically, freshwater wetlands in the region were seasonally wet, with groundwater serving an important role in sustaining wetland hydrology (Stein *et al.* 2007).

As the region has become urbanized, the wide variety of freshwater wetland habitats including wet meadows, sloughs, vernal pools, playas, etc. have been lost and perennial ponds now dominate remaining freshwater wetlands in southern California (Stein *et al.* 2007). Because of the high degree of regional groundwater withdrawals and the dominance of imported surface water from northern California and the Colorado River, many urban wetlands have become entirely dependent on surface water inputs. These surface water inputs are dominated by chronic nuisance flows from high density residential and commercial areas.

Most of the urban wetlands sampled in this study have been engineered to some degree. Twelve of the 21 sites were modified from existing or historical wetland areas (e.g., widened, deepened, or features such as berms or channels have been added to slow down or re-direct the flow), while five of the sites were created from upland areas. The remaining four sites are historic wetlands. The two reference sites selected for this study are located in natural areas that do not receive runoff from point or nonpoint contaminant sources (Sespe Creek, Mojave River marsh). Three other reference sites were targeted for inclusion in the study, but were dry at the time of sampling.

Field Methods

All sites were sampled for benthic macroinvertebrates (BMI) between April and June 2007 and for sediment contaminants and toxicity between October and November 2007 (Figure 1). BMI sampling characterized the relative abundance and diversity of invertebrates, with approximately equal effort given to the sampling of all major habitat types. Methods used were based on California Department of Fish and Game (CDFG) bioassessment procedures for lakes and ponds (ABL 1996) and flowing waters and channels (Harrington and Born 2000, ABL 2003). Taxonomy was conducted at the family level.

Sediment contaminant chemistry and toxicity samples were collected at a location nearest to the primary source of urban runoff in order to characterize the greatest potential effect. As such, the sediment from this location probably represents the highest levels of contamination and toxicity in these wetlands. The top 10 cm sediment were collected by wading from shore with a shovel, or with a Ponar grab sampler from an inflatable row boat. Sediment from multiple grabs was composited in the field, and distributed to a precleaned 4-oz glass jar for chemical analysis, or to four 1-L polyethylene jars for toxicity analysis. All containers were held on ice until distributed to the analytical laboratories.

Percent imperviousness was used as a proxy for degree of urbanization in the land use within its upstream catchment. The area defined by the catchment was modeled based on either a 30-m or 10-m digital elevation model using the ARC Hydro component of ARC INFO Version 9.1 (ESRI 2006). The 2001 National Land Cover Database (NLCD Imperviousness data layer) was used to estimate the percent imperviousness within the upstream catchment.

Laboratory Analysis

Chemistry

Samples for trace metals were analyzed by Inductively Coupled Plasma Mass Spectrometry (ICPMS) using EPA Method 6020m (Table 1). Samples for organic constituents (PAHs, PCBs, organochlorine pesticides, organophosphate pesticides, synthetic pyrethroids) were analyzed by Gas Chromatography/Mass Spectrometry (GC/MS) using EPA Method 8270c. Total organic carbon concentrations were determined by EPA Method 415.1. Chemical analyses of the samples were conducted by CRG Labs, Torrance CA.

Toxicity

All sediment samples were tested for toxicity using the H. azteca and Chironomus tentans 10-day survival tests following procedures outlined by USEPA (2000). The tests were conducted in 1-L glass jars containing 2 cm of sediment (approximately 150 ml) and 800 ml of water. Five replicates were used for each sample and the control. Sediments were sieved through a 0.5-mm screen to remove resident organisms and debris. The sediment was then added to the jars and overlying water (Culligan system treated) one day before the addition of animals in order to provide a 24-hour equilibration period. All test chambers were supplied with light aeration (1 - 2 bubbles per second). After equilibration, 10 juvenile H. azteca (7 - 10 days old) and 10 C. tentans (2nd to 3rd instar) were added to each beaker to start the test. Both species were added to each exposure chamber for the testing, instead of conducting separate exposures. Previous experiments have documented successful conduct of tests with both species together, minimizing concern that this modification would affect test results (Nautilus unpublished data). The exposures were conducted at 20°C. A source of food was provided during the test by adding 1 ml of a mixture of ground Tetramin® flakes (0.02 grams (g) per 100 ml Culligan) per chamber approximately every 3 days during the testing period. At the end of the exposure period, the sediment from the beakers was passed through a sieve to recover the animals, and the number of surviving animals counted. Water quality parameters (temperature, pH, dissolved oxygen and conductivity) were measured daily in surrogate water quality



Figure 1. Locations of the wetland sites sampled. County borders have been added for reference.

beakers for both pore water and overlying water in the sediment tests. Ammonia was measured at both the beginning and end of the exposure period. A 96-hour water only Cu reference toxicant test was conducted as a positive control with both species.

Sediment TIEs

Ten sample locations that exhibited toxicity during the October/November 2007 sampling events were selected for subsequent testing with preliminary TIEs. The TIE treatments for this evaluation specifically targeted pyrethroid pesticides, based on available chemistry data of the samples tested. Samples were re-collected from these 10 sites in April and May 2008. The TIEs were conducted on both whole sediment and pore water, and toxicity was evaluated using H. azteca. Methods to identify toxicity related to pyrethroids were primarily based on that described in Amweg and Weston (2007) and Weston and Amweg (2007). The two primary TIE treatments tested included addition of porcine carboxylesterase enzyme (CEE; 2.5 units esterase activity/ml) and piperonyl butoxide (PBO; 200 µg/L). Carboxylesterases are enzymes that hydrolyze ester-containing compounds (e.g., pyrethroids) into relatively non-toxic products, and have been found

to successfully inhibit toxicity from synthetic pyrethroids (Wheelock et al. 2004, Weston and Amweg 2007). PBO has been found to be an effective metabolic inhibitor of organophosphorous (OP) pesticide metabolism, thus decreasing toxicity due to this class of compounds (Ankley et al. 1991). In addition, PBO can enhance the toxicity of pyrethroid pesticides (Amweg and Weston 2007). Baseline whole sediment and pore water tests were performed concurrent to TIE treatments to compare the response in untreated samples to responses obtained after treatment manipulations. All TIE procedures were performed on undiluted sediment and pore water. Bovine serum albumin (BSA) was added as a method control, to account for reduced toxicity due to potential non-specific binding of various compounds (including pyrethroids) to the protein matrix in the CEE treatment. BSA was added at protein concentrations equivalent to the CEE additions (143 mg/L).

To both conserve sample and accommodate the large scale of testing required for this assessment, a modified test volume and number of replicates was employed (whole sediment: 80-ml sediment with 140-ml overlying water in 250-ml glass beakers using 3 to 5 replicates per treatment; pore water:

Table	1.	Reporting	levels	and	analysis	methods	for
chemical constituents measured.							

Analyte	Reporting Level	Method			
Metals (µg/g)					
As	0.05	EPA 6020m			
Cd	0.05	EPA 6020m			
Cr	0.05	EPA 6020m			
Cu	0.05	EPA 6020m			
Fe	5.00	EPA 6020m			
Pb	0.05	EPA 6020m			
Hg	0.05	EPA 6020m			
Ni	0.05	EPA 6020m			
Se	0.05	EPA 6020m			
Zn	0.05	EPA 6020m			
Organic Contaminants (ng/g)					
Organochlorine Pesticides ¹	5, 50	EPA 8270Cm			
PCBs ²	5, 20	EPA 8270Cm			
Organophosphate Pesticides ³	2-16	EPA 8270Cm			
Synthetic Pyrethroid Pesticides ⁴	25	EPA 8270Cm			
PAHs ⁵	5	EPA 8270Cm			

¹ Organochlorine pesticides include: 4,4'-DDD, 2,4'-DDD, 2,4'-DDE, 2,4'-DDT, 4,4'-DDE, 4,4'-DDT, aldrin, BHC-alpha, BHC-beta, BHC-delta, BHC-gamma (Lindane), chlordane-alpha, chlordane-gamma, cis-nonachlor, dieldrin, endosulfan sulfate, endosulfan-II, endosulfan-II, endrin aldehyde, endrin ketone, heptachlor, heptachlor epoxide, methoxychlor, mirex, oxychlordane, toxaphene, and trans-nonachlor.

² PCBs include: Aroclor 1016, 1221, 1232, 1242, 1248, 1254, 1260, PCB congener 18, 28, 31, 33, 37, 44, 49, 52, 66, 70, 74, 77, 81, 87, 95, 97, 99, 101, 105, 110, 114, 118, 119, 123, 126, 128+167, 138, 141, 149, 153, 156, 157, 158, 168, 168+132, 169, 170, 177, 180, 183, 187, 189, 194, 200, 201, 206.

³ OP pesticides include: bolstar (sulprofos), chlorpyrifos, coumaphos, demeton, diazinon, dichlorvos, dimethoate, disulfoton, ethoprop (ethoprofos), fenchlorophos (ronnel), fensulfothion, fenthion, guthion, malathion, merphos, mevinphos (phosdrin), parathion-methyl, phorate, tetrachlorovinphos (stirophos), tokuthion, and trichloronate.

⁴ Pyrethroid pesticides include: allethrin, permethrin, bifenthrin, cyfluthrin, cypermethrin, deltamethrin, fenpropathrin, lamda cyhalothrin, prallethrin, and pyrethrins.

1-methylphenanthrene, PAHs include: 1-methylnaphthalene, 2.3.5trimethyInaphthalene, 2,6-dimethylnaphthalene, 2-methylnaphthalene. acenaphthene. acenaphthylene, anthracene. benz[a]anthracene, benzo[a]pyrene, benzo[b]fluoranthene, benzo[e]pyrene, benzo[g,h,i]perylene, benzo[k]fluoranthene, biphenvl. chrvsene. dibenz[a,h]anthracene. dibenzothiophene, fluoranthene, fluorene. indeno[1,2,3-c,d]pyrene, naphthalene, pervlene, and phenanthrene, pyrene,

150-ml glass beakers with 75-ml sample using 3 or 4 replicates per treatment). Light aeration was supplied to each replicate test chamber at a rate of 1 to 2 bubbles per second. A 0.5-in square 1.0-mm Nitex screen was added to each pore water replicate to serve as a substrate for the amphipods. Whole sediment exposures were conducted over a period of 4 to 11 days, depending on the magnitude of the amphipod response observed. Single whole sediment replicates were ended periodically during the exposure period (from 96 hours to 10 days) to assess the magnitude of response. This was performed in an attempt to capture a meaningful separation among treatments when toxicity was observed, as both over-

whelming and limited toxic responses often make it difficult to evaluate treatment effectiveness. Pore water exposures were conducted over an eight-day period, with survival recorded daily. Pore water was obtained by centrifuging whole sediment at $3480 \ge g$ for 15 minutes.

Control water consisted of Culligan-filtered water (hardness 200 to 300 mg/L CaCO₃). Control sediment was obtained from a reference location in Yaquina Bay, Oregon, and washed with DI water prior to use. Control water and sediment were tested untreated (laboratory controls) and also underwent each of the applied TIE manipulations (method controls) to assess potential effects related to the procedures themselves.

Data Analysis

The sediment chemistry measurements were assessed by comparing the values to freshwater Probable Effects Concentrations (PECs; MacDonald *et al.* 2000; Table 2). The PEC thresholds are intended to identify contaminant concentrations above which harmful effects on sediment dwelling organisms are expected to occur frequently. PEC quotients (PECq) were calculated by dividing each measured concentration by its PEC value. A PECq >1.0 is considered potentially harmful. The mPECq was calculated following MacDonald *et al.* (2000) as the average PECq for 10 contaminants, including As, Cd, Cr, Cu, Pb, Ni, Zn, total PAHs, PCBs, and sum of dichlorodiphenyethylenes (DDEs).

No PEC value exists for Se, therefore concentrations of Se were assessed by comparing measured values with the observed effects threshold summarized by Van Derveer and Canton (1997; Table 2). Pyrethroid pesticides were assessed by comparing measured concentrations with the mean LC50 values (concentrations that cause a 50% reduction in survival, Table 2). There are no PEC thresholds for this class of compounds. The LC50 values were derived from sediment exposure experiments with H. azteca (Amweg et al. 2005). The LC50 values were used in this study to help interpret the toxicity data, but not to assess the wetland sites; because these LC50 values were derived from a single study, they do not carry the same weight to assess the sites as the PEC values, which were derived from data collected from many studies, encompassing several sediment types, and included several biological endpoints. A mean pyrethroid quotient was calculated using the same approach used for the mPECq; the concentrations for Table 2. Freshwater sediment quality guidelines used to evaluate the sediment chemistry concentrations MacDonald *et al.* (2000). An observed effects threshold of 4 mg/kg was used for Se (Van Presser *et al.* 2004). The pyrethroid pesticide LC50 value for bifenthrin = 4.5μ g/kg, for cyfluthrin = 13.7μ g/kg, for deltamethrin = 9.9μ g/kg, for esfenvalerate = 24μ g/kg, for lambda-cyhalothrin = 5.6μ g/kg, and for permethrin = 90μ g/kg (Amweg *et al.*, 2005).

Analyte	Probable Effects Concentration (PEC)
Heavy and Trace Metals (mg/kg, dry wt)	
As	33
Cd	5
Cr	111
Cu	149
Pb	128
Hg	1
Ni	49
Se	4
Zn	459
Organochlorine Pesticides (µg/kg, dry v	vt)
Chlordane	18
Sum DDD	28
Sum DDE	31
Sum DDT	63
DDT, total	572
Dieldrin	62
Endrin	207
Heptachlor epoxide	16
Lindane	5
PCBs, total (µg/kg, dry wt)	676
PAHs (µg/kg, dry wt)	
Anthracene	845
Benzo[a]pyrene	1450
Benz[a]anthracene	1050
Chrysene	1290
Fluorene	536
Fluoranthene	2230
Naphthalene	561
Phenanthrene	1170
Pyrene	1520
PAHs, total	22800

the individual pyrethroid compounds were divided by their respective LC50, and the average quotient was then calculated for each site.

The toxicity data were assessed with Analysis of Variance (ANOVA) and Dunnett's multiple comparison test, or unpaired one-way Student's t-tests. The survival data were arcsin square root transformed prior to analysis. ANOVA on ranks was used when data were non-normally distributed, and an ANOVA with Bonferroni correction was used when there were unequal replicates. Samples were considered toxic if they were significantly different and <80% of the control value.

Spearman correlation was used to examine the relationships between benthic macroinvertebrate taxa richness, sediment contaminant chemistry, toxicity, project objective, and percent imperviousness of land use in the upstream catchment land use

RESULTS

Benthic Macroinvertebrates

The BMI taxa richness scores varied from 4 to 11 in the basin wetlands, relative to scores of 10 to 11 in the 2 basin reference sites. In channel wetlands, the number of taxa ranged from 2 to 15 in the channel wetlands, compared to 12 taxa found at the channel reference site.

Regressions of benthic macroinvertebrate data showed a declining condition in benthic macroinvertebrate community structure with increasing % imperviousness in the upstream catchment. For basins, BMI taxa richness, insect richness, % intolerant species, and % dominant species, % Chironomid midges and % Odonata were all significantly correlated with % imperviousness at an $\alpha = 0.05$. For channels, only a subset of these was significant (BMI taxa richness, insect richness, and % intolerant species). No significant relationships were found by wetland objective (habitat only, multipurpose, treatment only) for BMI metrics in basin or channel wetlands (Figure 2).

Sediment Chemistry

Most of the urban wetlands had at least one constituent that exceeded a PEC (Table 3). Of the 21 urban wetlands, PEC thresholds were exceeded at 13 sites (62%; Figure 3). For most sites, there were only one or two constituents that exceeded the PEC thresholds, rather than a mixture of contaminants. This was the case for 11 of the 13 sites with PEC exceedances. The most number of PEC exceedances to occur at a site was five. The two reference wetland sites did not have any constituents exceeding the PEC thresholds.

The constituents exceeding the PECs varied among the sites. The most prevalent contaminant was Cd, exceeding the PEC at six sites, followed by Ni and DDE (each elevated at four sites), chlordane (three sites), and Zn (two sites). Fluoranthene, pyrene, Pb, and Cu exceeded their respective PEC



Figure 2. Distribution of BMI taxa richness in urban study sites for basins and channel wetlands relative to reference data. Note that these reference sites were dry at the time of sediment contaminant and toxicity sampling, so other reference sites were chosen.

at one site each. The incidence of contamination from heavy metals was approximately equal to organics, with PEC exceedances at seven and eight sites, respectively.

Pyrethroid pesticide contamination was prevalent at most urban wetland sites (Table 3; Figure 3). Measured values exceeded reported LC50 values at 13 urban wetlands (62%) by as much as a factor of 137. The highest values were reported at Sims Pond, which receives golf course runoff, and WetCAT North, which receives residential area runoff. Pyrethroid concentrations were below the method detection limit at the two reference wetlands. Organophosphorus pesticide concentrations were below the method detection limit at all sites.

Six locations had sediment concentrations that exceeded the preliminary observed effects threshold for Se (Table 3). All sites that exceeded this threshold were in the San Diego Creek watershed – the primary freshwater drainage to Upper Newport Bay – or sites within the local drainage basin of the Bay. Exceedances ranged from a factor 1.2 at UCI Pond 3 to a factor of 14 at Big Canyon Marsh and Big Canyon Riparian wetlands.

Contaminant concentrations that were correlated to the amount of urbanization of the catchment are those typically associated with transportation infrastructure (% imperviousness of the catchment area). Sediment concentrations of Cu, Pb, Zn, total PAHs and cypermethrin were all significantly correlated (p-value < 0.05) with % impervious area (r = 0.43, r = 0.53, r = 0.51, r = 0.43, r = 0.59, respectively). Sediment contaminant concentrations were not found to significantly differ by wetland objective. Wetlands with habitat as the main objective did not have significantly lower concentrations of contaminants than wetlands used to improve water quality, or those wetlands with habitat and water quality as equally important objectives.

One measure of benthic organism community was related to sediment contamination (Figure 4). There was a significant decrease in benthic macroinvertebrate diversity with increasing mPECq values (r = -0.62, p<0.01). All other BMI were not significantly correlated. The mean pyrethroid quotient was not correlated with benthic community measures (r = -0.09 BMI richness).

Toxicity

About half of the treatment wetland sites (10 out of 21) were toxic to *H. azteca* survival (Table 3; Figure 5). Only two sites were toxic to *C. tentans* survival (Big Canyon Riparian and WetCAT North). Because *H. azteca* survival was the much more sensitive test, results for amphipod survival were used throughout the remainder of the study to further identify causes of toxicity.

Toxicity to *H. azteca* was not related to wetland objective. Amphipod survival in sediments from wetlands with water quality as the primary objective was not significantly different than the survival of amphipods exposed to sediments from habitat wetlands, or the multipurpose wetlands (p = 0.42). In addition, amphipod toxicity was not related to the degree of urbanization (measured as % imperviousness of the catchment area, p = 0.52) or BMI community indices (p = 0.95).

Contaminants Likely Responsible for Toxicity

Based on concentrations and TIE results, pyrethroids are likely responsible for decreased sur-

Table 3. Summary of constituents exceeding sediment quality guideline thresholds, and an indication of toxicity at each of the study sites. nt = not tested. Partial = pyrethroids implicated in the toxicity, but other contaminants may have also been responsible. Inconclusive = the samples collected for TIE testing were no longer toxic.

Wetland Site	Constituents Exceeding PEC Thresholds	Pyrethroids Exceeding Mean Sediment LC50 for <i>H. azteca</i>	Exceed Se Observed Effect Threshold	<i>H. azteca</i> Acute Toxicity	<i>C. tentans</i> Acute Toxicity	Indication of Pyrethroid Toxicity by TIEs
Arroyo Seco Channel	None	Bifenthrin, permethrin	No	Yes	No	Partial
Ballona Fresh Water Marsh	None	Bifenthrin	No	No	No	nt
Big Canyon Marsh	Cd	Bifenthrin	Yes	Yes	No	Yes
Big Canyon Riparian	Cd, Ni	Bifenthrin, permethrin	Yes	Yes	Yes	Yes
Brawley Wetlands	Sum DDE	None	No	No	No	nt
Camino Real Bioswale	None	Bifenthrin	No	Yes	No	Yes
Crown Valley Parkway Riparian	Cd, Ni	Bifenthrin, L-cyhalothrin, permethrin	No	Yes	No	Partial
Dairy Mart Ponds	Sum DDE	None	No	No	No	nt
IRWD Carlson Marsh	Cd	None	Yes	No	No	nt
IRWD Pond A	Sum DDE	Bifenthrin	Yes	Yes	No	Inconclusive
IRWD Pond 6	Sum DDE	None	Yes	No	No	nt
Lewis Center Marsh	None	None	No	No	No	nt
Madrona Marsh	Pb, Zn, chlordane	Bifenthrin, L-cyhalothrin, permethrin	No	No	No	nt
Mojave River Marsh	None	None	No	No	No	nt
Old Mission Creek	None	Bifenthrin	No	No	No	nt
San Elijo Marsh	None	Bifenthrin, permethrin	No	Yes	No	Yes
Sespe Creek	None	None	No	No	No	nt
Sims Pond	Fluoranthene, pyrene	Bifenthrin, cyfluthrin, L-cyhalothrin, permethrin	No	Yes	No	Yes
UCI Pond 11	None	None	No	No	No	nt
UCI Pond 3	None	None	Yes	No	No	nt
Valeta Street Marsh	Chlordane	Bifenthrin, L-cyhalothrin, cyfluthrin, permethrin	No	Yes	No	Yes
Wet CAT East	Cd, Ni	None	No	No	No	nt
Wet CAT North	Cd, Cu, Ni, Zn, chlordane	Bifenthrin, cyfluthrin, L-cyhalothrin, permethrin	No	Yes	Yes	Yes

vival observed at all 10 sites that were toxic to amphipods (Figure 6). Pyrethroids were elevated at most sites. There was agreement between pyrethroid exceedance and the toxicity threshold (both exceeded, or neither exceeded) at 18 out of the 21 urban wetlands (86%). Moreover, the mean pyrethroid quotient for the toxic sites was significantly greater than for the nontoxic sites (p <0.01), and amphipod survival was negatively correlated with the mean pyrethroid quotient (p <0.01, r = -0.68).

Several other constituents also exceeded available effects-based guideline values in 7 of the 10 sites that had amphipod toxicity. Heavy metals exceeded PEC thresholds at four of the toxic sites, with Cd exceedances at all four sites, followed by Ni (three sites), Cu and Zn (one site each). Almost an equal number of sites, however, had elevated metal concentrations but no associated toxicity (three wetlands). Sediment DDE concentrations exceeded the PEC value at four sites, but only one of these sites was toxic to amphipods. Likewise, the chlordane PEC was exceeded at three sites, with associated toxicity at only one of these sites.

Sediment TIE results confirm that pyrethroid pesticides are a major component of sediment toxici-

ty in these wetlands. Among the 10 sites in which toxicity was originally measured in the fall of 2007, toxicity was observed in the baseline samples for 8 of the 10 whole sediment exposures and 7 of the 10 pore water exposures in April 2008 (Table 3). Pyrethroid pesticides could be attributed to most, if not all, of the observed toxicity to H. azteca in seven samples tested (Table 3). Other contaminants may have been responsible for part of the toxicity in the samples where toxicity was only partially reduced by the CEE treatment (Arroyo Seco Channel, Crown Valley Parkway Riparian). Because toxicity was no longer observed in the Irvine Ranch Water District (IRWD) Pond A baseline sample collected for TIE, the source of the toxicity is inconclusive in the first sediment sample collected from this site.

DISCUSSION

Risk to Benthic Organisms from Sediment Contaminants

The habitat quality of a wetland is directly related to its water source (Mitsch and Gosselink 1993). Many urban wetlands are directly dependent on wet



Figure 3. Sediment concentrations of select contaminants at each site. The dashed line indicates the Probable Effects Concentration (metals, DDE, chlordane), or the average sediment LC50 for *H. azteca* from Amweg *et al.*, 2005 (bifenthrin, L-cyhalothrin). The LC50 for bifenthrin = $4.5 \mu g/kg$. Nondetects were replaced with half the method detection limit.

and dry weather runoff to sustain their wetland hydrology, particularly in urban environments where water is a scarce resource (Everts 1997, Lahr 1997). Conversely, this urban water source provides a source of contaminants that can be harmful to the wildlife that depend upon the wetland for existence. The results of this study indicate that the benthic organisms in 18 of the 21 urban wetlands examined were at risk due to sediment contaminants. Most of these sites were either toxic to *H. azteca*, or exceeded a sediment quality guideline, or both. Sediment contaminant concentration was also found to significantly correlate with decreased benthic macroinvertebrate diversity in these wetlands. Sediment TIE studies indicate that pyrethroid pesticides are a major component of toxicity to benthic organisms in urban freshwater wetlands found in southern California. BMI Richness vs MPECq



Figure 4. Relationship between mPECq and BMI richness. The vertical dashed line is the threshold above which toxicity is expected.

Several previously published studies support the conclusion that urban wetlands are impacted to various degrees by toxic contaminants. Maltby *et al.* (1995) found that runoff from highways into a small

stream resulted in high concentrations of water and sediment metals and hydrocarbons, which correlated with changes in benthic community structure. Within these sites, sediment toxicity studies showed a slight reduction of survival of amphipods, with fractionation and toxicity confirmation studies indicating that most of the toxicity was due to hydrocarbons (Maltby et al. 1995, Boxall and Maltby 1997). Galli (1988) and Yousef et al. (1990) both found that macroinvertebrate communities in sediments of stormwater ponds were low in diversity with an assemblage characteristic of high pollution stress (chironomid and tubificid worms and dipteran midge larvae). Karouna-Reiner and Sparling (1997), in a study of trace metal bioaccumulation in stormwater ponds in Maryland, detected Cu, Pb, Zn and occasionally Cd in tissues of snails, damselflies and other macroinvertebrates, but found no acute toxicity to amphipods exposed to sediments from these ponds. Most of these studies noted the severity of bioaccumulation and toxicity could be expected to be highly variable among wetlands and a function of many site-specific factors, including the mix of contami-



Figure 5. Survival of *H. azteca* and *C. tentans* exposed to wetland sediments. * = significantly different and <80% of control value.



Figure 6. TIE results for three representative sites. Carboxyl esterase (CEE) removed most of the toxicity in the samples from Camino Real, with no reduction in toxicity from the bovine serum albumin treatment, indicating the toxicity in the baseline sample was related to exposure to pyrethroids. Toxicity was only somewhat reduced by CEE in the Crown Valley Parkway whole sediment sample, suggesting the toxicity was partially related to pyrethroids. For the WetCAT North samples, CEE did not reduce toxicity in the whole sediments, but did appear to be effective in reducing the toxicity of the pore water. Results for the pore water testing represent the 96-hour exposure experiments, except for Camino Real, which represent the 192-hour exposures. CEE = 2.5 U/ml carboxyl esterase enzyme. BSA = 143 mg/L bovine serum albumin. PBO = $200 \mu \text{g/l}$ piperonyl butoxide.

nants introduced, contaminant loading over time, hydraulic residence time, sediment bulk characteristics, and age of the wetland (Schueler 2000).

Sediment contaminant concentrations and toxicity could also be expected to vary both spatially and temporally within a wetland (Wren *et al.* 1997, Carapeto and Purchase 2000, Mallin *et al.* 2002). In this study, sediment samples were taken in a location proximal to the primary source of urban runoff. As contaminant concentration and toxicity could be expected to vary, at minimum, as a function of distance from the source of contaminants, the results of the study are likely to represent, spatially, a conservative estimate of risk for the wetland. Some differences were also apparent in the seasonality of toxicity observed at these sites. Of the 10 sites in which toxicity was found in November 2007, 7 sites retained toxicity in April 2008 sampling periods. This variability could be due to a variety of factors (i.e., seasonal pesticide use within the watershed, flow patterns, changes in grain size distribution, loss or burial of previously contaminated sediments, and buildup of chemicals during antecedent dry periods). To better define the appropriate protocol for costeffectively capturing risk to wildlife from sediment contamination and toxicity, additional information about how sediment contaminant concentrations vary seasonally and spatially within wetlands is required. This information would also help identify management options to mitigate that risk.

Potential Contaminants Responsible for Toxicity

Sediment chemistry and chemical-specific TIE methods suggest pyrethroids may have been responsible for much of the toxicity documented in this study. Three lines of evidence support this assertion. First, concentrations of pyrethroids were elevated at all 10 sites that were toxic to H. azteca. Second, the mean pyrethroid quotient was negatively correlated with amphipod survival; other contaminants were elevated, and could also have contributed to toxicity at a subset of the sites evaluated. Third and most importantly, whole sediment and pore water TIEs demonstrated that pyrethroid pesticides were responsible for most, if not all, of observed toxicity in 7 of 10 samples tested. In addition, there is at least one line of evidence suggesting that some fraction of observed toxicity in the remaining three samples tested might also be related to pyrethroids.

Carboxylesterase was successful at reducing toxicity in seven of the eight toxic whole sediments and five of the seven toxic pore water samples. PBO was successful at enhancing toxicity in 5 of the 10 sediments, and 8 of the 10 pore water samples tested. These trends are consistent with that expected for pyrethroid pesticides based on known modes of action and prior studies (Wheelock et al. 2004 and 2006, Anderson et al. 2006, Phillips et al. 2006, Amweg and Weston 2007, Weston and Amweg 2007). Due to the specificity of these two treatments, and overall success and consistent patterns observed, the combined CEE and PBO treatments provided substantial confidence in identifying pyrethroid-related toxicity, using a very targeted cost-effective approach. Sediment and pore water are extremely complex matrices that can make isolating and identifying specific organic contaminants challenging. The effectiveness of any one treatment will depend on a variety of potential matrix related effects, thus emphasizing the importance of using a weight of evidence approach that employs multiple

treatments in both whole sediment and pore water. While this, along with available analytical data, is highly suggestive that pyrethroids were the primary class of toxicants of concern, Phase II and III TIE steps would be required to further identify and confirm specific causal agents, as well as the proportion of toxicity attributable to each in cases where more than one toxicant may be present (USEPA 1993a, 1993b, 2007).

Toxicity from pyrethroid pesticides has been observed in sediments and surface waters in a number of locations in California (Amweg et al. 2005, Weston et al. 2004, Weston et al. 2005). As a class of contaminants, synthetic pyrethroid pesticides are now a large percentage of the residential and commercial pesticide use in the United States (Weston et al. 2005). Pyrethroids adsorb to soil particles, so sediments would be expected to be the main repository of these compounds (Gan et al. 2005). Pyrethroid pesticides were expected to bring less inadvertent runoff toxicity than the organophosphorus pesticides they replaced, because of the assumption that their association with soil would make retention on site easier. However, pyrethroids are known to be highly toxic to aquatic organisms (Maund et al. 2002). Observations of pyrethroid-associated toxicity in aquatic sediments indicate that increased scrutiny of pyrethroid use is needed. In particular, bifenthrin, found at high concentrations in numerous sediments in this study, is a restricted use pesticide commonly used in structural pest control, fire ant control, and vector control in aquatic habitats (Weston et al. 2005). Because of its restricted use status, targeted outreach and control of bifenthrin use is of high priority and warranted in southern California given the widespread pyrethroid toxicity found in wetland sediments in this study. Pyrethroid pesticides are clearly a regional if not national issue for wetland managers that deserve particular attention. It is important to mitigate these risks in light of the importance that urban wetlands serve as habitat and for stormwater management, particularly in arid climates (Everts 1997, Lahr 1997).

Implications of Toxicity to Higher Level Wetland Organisms

Adverse effects on higher level wetland organisms (e.g., amphibian, birds, fish, etc.) can occur via bioaccumulation, direct toxicity or via impact from alterations in the food web (Cooper 1991, Dunier and Siwicki 1993, Campbell 1994, Wren *et al*.

1997). In this study, an index of degree of sediment contamination (mPECq) was found to significantly correlate with benthic macroinvertebrate diversity in these wetlands. Macroinvertebrates are a critical link in the food web of wetlands, providing the link between primary producers, detrital trophic organisms, and higher level consumers such as birds, fish, and amphibians (Ludwa and Richter 2000). The effects of urban runoff on resident macroinvertebrate species diversity and community structure have been well documented. Overall, these results can be summarized as: 1) decreasing overall taxa richness, 2) eliminating or reducing taxa belonging to scraper and shredder functional feeding groups relative to collector functional feeding groups, 3) reducing the taxa richness and relative abundance of orders Ephemeroptera, Plecoptera, Odonata, and Trichoptera (sensitive orders often used as a basis for stream biometrics), and 4) reducing or eliminating certain chironomid taxa, with an increase in the abundance of tolerant Chironomids, oligochaetes, and gastropods (Galli 1988, Yousef et al. 1990, Ludwa and Richter 2000).

In this study, the magnitude of this alteration is difficult to document because of the limited data on reference sites. Few remaining reference sites for the "basin" class of wetlands exist in southern California (Stein et al. 2007). Of these remaining wetlands, most were dry during the sampling period, which occurred during the driest rainfall year on record for southern California. For the two reference sites sampled, sediments were not toxic to test organisms, and did not exceed sediment quality guidelines for any of the contaminants analyzed. Using the reference sites for comparison, urban wetlands tended to have greater concentrations of metals. For example, all 21 of the urban wetlands had greater concentrations of Cu and Zn (ranging by a factor of 1.2 to 24.4 times the reference value for Cu, and 1.1 to 12.6 times the value for Zn), 20 sites had greater Cd and Ni concentrations (3 to 356 times the Cd value, and 1.4 to 21.4 times for Ni), and 19 sites had greater concentrations of Pb (1.1 to 31.3 times greater than the reference site values). Thus, though limited, the sediment chemistry and toxicity data from the reference sites show a clear distinction from the majority of urban sites.

Factors Associated With Sediment Contamination and Toxicity

Most of the contaminants that were correlated with increasing urbanization (% imperviousness of

the catchment area) are associated with automobile use and transportation infrastructure (Drapper *et al.* 2000). While there was a correlation with urbanization, these constituents were rarely present at potentially toxic levels; there were only a few instances where these contaminants exceeded their respective PEC. These less-than-toxic concentrations may have partially accounted for the lack of correlation between amphipod toxicity and urbanization. However, a more likely explanation is that other contaminants responsible for the toxicity (including pyrethroids) were not correlated with imperviousness.

One key management question posed is whether treatment wetlands, whose primary management objective is water quality enhancement, present a higher risk to wetland organisms from contaminants than habitat or multipurpose wetlands (wetland sites in which habitat and water quality enhancement functions are equally important). Study results show no significant difference in benthic macroinvertebrate, sediment contaminant concentrations or toxicity among urban wetlands by objective. This result is similar to that found for several other indicators of habitat quality including benthic macroinvertebrate and bird diversity (Sutula et al. 2008). The lack of significant differences by objective may be due to the profound equalizing effect that a broad range of urban stressors can have on wetlands. These stressors, including eutrophication, excessive sedimentation, invasive species, habitat fragmentation and disturbance, etc. can confound effects of contaminant toxicity, making trends difficult to identify (Azous and Horner 2000).

LITERATURE CITED

Aquatic Bioassessment Laboratory (ABL). 1996. California Lentic Bioassessment Procedure (Biological Sampling for Lakes and Reservoirs). California Department of Fish and Game, Water Pollution Control Laboratory. Rancho Cordova, CA.

ABL. 2003. California Stream Bioassessment Procedure (Protocol Brief for Biological and Physical/Habitat Assessment in Wadeable Streams). California Department of Fish and Game, Water Pollution Control Laboratory. Rancho Cordova, CA.

Adamus, P., T.J. Danielson and A. Gonyaw. 2001. Indicators for Monitoring Biological Integrity of Inland Freshwater Wetlands: A Survey of North American Literature (1990-2000). US Environmental Protection Agency, Office of Water, Wetlands Division. Washington, DC.

Amweg, E.L. and P.D. Weston. 2007. Whole sediment toxicity identification evaluation tools for pyrethroid insecticides: I. Piperonyl Butoxide addition. *Environmental Toxicology and Chemistry* 26:2389-2396.

Amweg, E.L., D.P. Weston and N.M. Ureda. 2005. Use and toxicity of pyrethroid pesticides in the Central Valley, California, USA. *Environmental Toxicology and Chemistry* 24:966-972.

Anderson, B.S., B.M. Phillips, J.W. Hunt, K. Worcester, M. Adams, N. Kapellas and R.S. Tjeerdema. 2006. Evidence of pesticide impacts in the Santa Maria River watershed, California, USA. *Environmental Toxicology and Chemistry* 25:1160-1170.

Ankley, G.T., J.R. Dierkes, D.A. Jensen and G.S. Peterson. 1991. Piperonyl butoxide as a tool in aquatic toxicological research with organophosphorous insecticides. *Ecotoxicology and Environmental Safety* 21:266-274.

Azous, A. and R. Horner. 2000. Wetlands and Urbanization. CRC Press. Boca Raton, FL.

Birch, G.A., C. Matthai, M.S. Fazeli and J.Y. Suh. 2004. Efficiency of a constructed wetland in removing contaminants from stormwater. *Wetlands* 24:459-466.

Boxall, A.B.A. and L. Maltby. 1997. The effects of motorway runoff on freshwater ecosystems. *Archives of Environmental Contamination and Toxicology* 33:9-16.

Brown, J. and S. Bay. 2004. Organophosphorus pesticides in the Malibu Creek watershed. pp. 94-102 *in*: S.B. Weisberg and D. Elmore (eds.), Southern California Coastal Water Research Project 2003-2004 Biennial Report. Westminster, CA.

Brown, J. and S. Bay. 2006. Assessment of best management practice (BMP) effectiveness for reducing toxicity in urban runoff. pp. 207-226 *in*: S.B. Weisberg and D. Elmore (eds.), Southern California Coastal Water Research Project 2005-2006 Biennial Report. Westminster, CA.

Campbell, K.R. 1994. Concentrations of heavy metals associated with urban runoff in fish living in

stormwater treatment ponds. *Archives of Environmental Contamination and Toxicology* 27:352-356.

Carapeto, C. and D. Purchase. 2000. Distribution and removal of cadmium and lead in a constructed wetland receiving urban runoff. *Bulletin of Environmental Contamination and Toxicology* 65:322-329.

Characklis, G.W. and M.R. Wiesner. 1997. Particles, metals, and water quality in runoff from large urban watershed. *Journal of Environmental Engineering* 123:753-759.

Cooper, K. 1991. Effects of pesticides on wildlife. *in*: W.J. Hays and E.R. Law (eds.), Handbook of Pesticide Toxicology. Volume One. Academic Press. San Diego, CA.

Dahl, T.E. 1990. Wetland Losses in the United States 1780's to 1980's. US Fish and Wildlife Service. Washington, DC.

Drapper, D., R. Tomlinson and P. Williams. 2000. Pollutant concentrations in road runoff: Southeast Queensland case study. *Journal of Environmental Engineering* 126:313-320.

Dunier, M. and A.K. Siwicki. 1993. Effects of pesticides and other organic pollutants in the aquatic environment on immunity of fish: a review. *Fish and Shellfish Immunology* 3:423-438.

Environmental Systems Research Institute, Inc. (ESRI). 2006. ARC GIS Software Version 3.2. Redlands, CA.

Everts, J.W. 1997. Ecotoxicology for risk assessment in arid zones: Some key issues. *Archives of Environmental Contamination and Toxicology* 32:1-10.

Galli, F.J. 1988. A Limnological Study of an Urban Stream Stormwater Management Pond and Stream Ecosystem. George Mason University. Fairfax, VA.

Gan, J., S.L. Lee, W.P. Liu, D.L. Haver and J.N. Kabashima. 2005. Distribution and persistence of pyrethroids in runoff sediments. *Journal of Environmental Quality* 34:836-841.

Garcia-Hernandez, J., K.A. King, A.L. Velasco, E. Shumilin, M.A. Mora and E.P. Glenn. 2001. Selenium, selected inorganic elements, and organochlorine pesticides in bottom material and

biota from the Colorado River Delta. *Journal of Arid Environments* 49:65-89.

Glenn, E.P., J. Garcia, R. Tanner, C. Congdon and D. Luecke. 1999. Status of wetlands supported by agricultural drainage water in the Colorado River Delta, Mexico. *Hortscience* 34:39-45.

Harrington, J. and M. Born. 2000. Measuring the Health of California Streams and Rivers: A Methods Manual for Water Resource Professionals, Citizen Monitors, and Natural Resources Students. 2nd ed. Sustainable Land Stewardship International Institute. Sacramento, CA.

Harris, M.L., C.A. Bishop, J. Struger, M.R. Van Den Heuvel, G.J. Van Der Kraak, D.G. Dixon, B. Ripley, and J.P. Bogart. 1998. The functional integrity of Northern Leopard Frog (Rana pipiens) and Green Frog (Rana clamitans) populations in orchard wetlands. *Environmental Toxicology and Chemistry* 17:1338-1350.

Helfield, J.M. and M.L. Diamond. 1997. Use of constructed wetlands for urban stream restoration: A critical analysis. *Environmental Management* 21:329-341.

Holland, C.C., J.E. Honea, S.E. Gwin and M.E. Kentula. 1995. Wetland degradation and loss in the rapidly urbanizing area of Portland, Oregon. *Wetlands* 15:336-345.

Horner, R., A. Azous, K. Richter, S.A. Cooke, L. Reinelt and K. Ewing. 2000. Wetlands and stormwater management guidelines. pp. 299-324 *in*: R. Horner (ed.), Wetlands and Urbanization. CRC Press. Boca Raton, FL.

Karouna-Renier, N.K. and D.W. Sparling. 1997. Toxicity of stormwater treatment pond sediments to *Hyalella azteca* (Amphipoda). *Bulletin of Environmental Contamination and Toxicology* 58:550-557.

Katznelson, R., W.T. Jewell and S.L. Anderson. 1995. Spatial and temporal variations in toxicity in an urban-runoff treatment marsh. *Environmental Toxicology and Chemistry* 14:471-482.

Lahr, J. 1997. Ecotoxicology of organisms adapted to like in temporary freshwater ponds in arid and semi-arid regions. *Archives of Environmental Contamination and Toxicology* 32:1-10. Long, E.R. and P.M. Chapman. 1985. A sediment quality triad: measures of sediment contamination, toxicity and infaunal community composition in Puget Sound. Marine Pollution Bulletin 16:405-415.

Ludwa, K.A. and K.O. Richter. 2000. Emergent macroinvertebrate communities in relation to watershed development. pp. 47-67 *in*: R. Horner (ed.), Wetlands and Urbanization. CRC Press. Boca Raton, FL.

MacDonald, D.D., C.G. Ingersoll and T.A. Berger. 2000. Development and evaluation of consensusbased sediment quality guidelines for freshwater systems. *Archives of Environmental Contamination and Toxicology* 39:20-31.

Mallin, M.A., S.H. Ensign, T.L. Wheeler and D.B. Mayes. 2002. Pollutant removal efficacy of three wet detention ponds. *Journal of Environmental Quality* 31:654-660.

Maltby, L., D.M. Forrow, A.B.A. Boxall, P. Calow and C.I. Betton. 1995. The effects of motorway runoff on freshwater ecosystems. Environmental Toxicology and Chemistry 14:1079-1101.

Maund, S.J., M.J. Hamer, M.C.G. Lane, E. Farrelly, J.H. Rapley, U.M. Goggin and W.E. Gentle. 2002. Partitioning, bioavailability, and toxicity of the pyrethroid insecticide cypermethrin in sediments. Environmental Toxicology and Chemistry 21:9-15.

Mitsch, W.J. and J.G. Gosselink. 1993. Wetlands. 2nd ed. Van Nostrand Press. New York, NY.

National Irrigation Water Quality Program (NIWQP). 1998. Guidelines for interpretation of the biological effects of the selected constituents in biota, water, and sediment. National Irrigation Water Quality Program information report No. 3. US Department of the Interior. Reston, VA.

Ohlendorf, H.M., R.L. Hothem and D. Welsh. 1989. Nest sucess, cause-specific nest failure, and hatchability of aquatic birds at selenium-contaminated Kesterson Reservoir and a reference site. *Condor* 91:787-796

Pascoe, G.A. 1993. Wetland risk assessment. *Environmental Toxicology and Chemistry* 12:2293-2307.

Paul, M.J. and J.L. Meyer. 2001. Streams in the urban landscape. *Annual Review of Ecological Systems* 32:333-365.

Phillips, B.M., B.S. Anderson, J.W. Hunt, S. Huntley, R.S. Tjeerdema, N. Kapellas and K. Worcester. 2006. Solid-phase sediment toxicity identification evaluation in an agricultural stream. *Environmental Toxicology and Chemistry* 25:1671-1676.

Schueler, T.R. 2000. Environmental Impact of Stormwater Ponds. Article 79 *in*: T. Schueler and H. Holland (eds.), The Practice of Watershed Protection. Center for Watershed Protection. Ellicott City, MD.

Schuler, C.A., R.G. Anthony and H.M. Ohlendorf. 1990. Selenium in wetlands and waterfowl foods at Kesterson Reservoir, California, 1984. *Archives of Environmental Contamination and Toxicology* 19:845-853.

Stein, E.D., S. Dark, T. Longcore, N. Hall, M.Beland, R. Grossinger, J. Casanova and M. Sutula.2007. Historical ecology and landscape change of the San Gabriel River and floodplain. Technical Report 499. Southern California Coastal Water Research Project. Costa Mesa, CA.

Sutula, M., J. Brown, E. Fetscher, M. Mattson, S. Madon, G. Santolo, E. Byron and C. Stransky. 2008. Habitat value and treatment effectiveness of freshwater urban wetlands. Southern California Coastal Water Research Project. Costa Mesa, CA.

United States Environmental Protection Agency (USEPA). 1993a. Methods for Aquatic Toxicity Identification Evaluations - Phase II Toxicity Identification Procedures for Samples Exhibiting Acute and Chronic Toxicity. EPA/600/R-92/080. USEPA. Washington, DC.

USEPA. 1993b. Methods for Aquatic Toxicity Identification Evaluations - Phase III Toxicity Conformation Procedures for Samples Exhibiting Acute and Chronic Toxicity. EPA/600/R-92/081. USEPA. Washington, DC.

USEPA. 2000. Methods for Measuring the Toxicity and Bioaccumulation of Sediment-associated Contaminants with Freshwater Invertebrates (2nd ed.). EPA 600/R-99/064. USEPA. Washington, DC.

USEPA. 2007. Sediment Toxicity Identification Evaluation (TIE) Phases I, II, and III Guidance

Document EPA/600/R-07/080. USEPA. Washington, DC.

Van Derveer, W.D. and S.P. Canton. 1997. Selenium sediment toxicity thresholds and derivation of water quality criteria for freshwater biota of western streams. *Environmental Toxicology and Chemistry* 16:1260-1268.

Welsh, D. and O.E. Maughan. 1994. Concentrations of selenium in biota, sediments, and water at Cibola National Wildlife Refuge. *Archives of Environmental Contamination and Toxicology* 26:452-458.

Weston, D.P. and E.L. Amweg. 2007. Whole sediment toxicity identification evaluation tools for pyrethroid insecticides: II Esterase addition. Environmental Toxicology and Chemistry 26:2397-2404.

Weston, D.P., R.W. Holmes, J. You and M.J. Lydy. 2005. Aquatic toxicity due to residential use of pyrethroid insecticides. *Environmental Science and Technology* 39:9778-9784.

Weston, D.P., J. You and M.J. Lydy. 2004. Distribution and toxicity of sediment-associated pesticides in the agriculture-dominated waterbodies of California's Central Valley. *Environmental Science and Technology* 38:2752-2759.

Wheelock, C.E., J.L. Miller, M.J. Miller, S.J. Gee, G. Shan and B.D. Hammock. 2004. Development of toxicity identification evaluation procedures for pyrethroid detection using esterase activity. *Environmental Toxicology and Chemistry* 23: 2699-2708.

Wheelock, C.E., J.L. Miller, M.J. Miller, B.M. Phillips, S.A. Huntley, S.J. Gee, R.S. Tjeerdema and B.D. Hammock. 2006. Use of carboxylesterase activity to remove pyrethroid-associated toxicity to *Ceriodaphnia dubia* and *Hyalella azteca* in toxicity identification evaluations. *Environmental Toxicology and Chemistry* 25:973-984.

Wren, C.D., C.A. Bishop, D.L. Stewart and G.C. Barrett. 1997. Wildlife and contaminants in constructed wetlands and stormwater ponds: current state of knowledge and protocols for monitoring contaminant levels and effects in wildlife. Technical Report 269. Canadian Wildlife Service. Ontario, Canada. Yousef, Y.M., M. Wanielista, J. Dietz, L. Yin and M. Brabham. 1990. Final Report-Efficiency Optimization of Wet Detention Ponds for Urban Stormwater Management. University of Central Florida, Florida Department of Environmental Regulation. Orlando, FL.

ACKNOWLEDGEMENTS

The authors thank Nick Miller, Andrew Fields and Greg Lyons of SCCWRP for their assistance in collecting the sediment samples. The authors also thank Nathan Hall for assistance with the toxicity and TIE testing efforts, Sarah Douglass for helping with the figures, and Adrienne Cibor and Dr. Howard Bailey (all from Nautilus) for their review and comments. In addition, the authors would like to express appreciation for funding provided by the Los Angeles Regional Water Quality Control Board, Agreement Number 04-090-554-0.