

**Stormwater Runoff Water Quality Newsletter
Devoted to Urban/Rural Stormwater Runoff
Water Quality Management Issues**

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Several previous editions of the Newsletter have discussed the potential role of aquatic plant nutrients in urban and rural stormwater runoff and wastewater discharges in the stimulation of algae and other aquatic plants that adversely impact water quality (Newsletters 1-2, 1-5, 4-3/4, 5-1, 6-1, 6-2, 7-6/7, 9-1/2, 9-7, 9-8, 9-10, 10-1, 10-2, 10-4, and 10-5, all of which are available at <http://www.gfredlee.com/newsindex.htm>). This edition of the Newsletter expands the discussion of the impacts of aquatic plant nutrients (nitrogen and phosphorus) to specifically address the role of aquatic plant nutrients in support of algal growth that leads to DO depletion in waterbodies, which can, in turn, lead to aquatic life toxicity in the sediment and near the sediment/water interface. It also discusses the importance of considering this role in attempting to control sediment toxicity associated with heavy metals, organics, and other constituents.

Role of Nutrients as a Cause of Oxygen Demand and Sediment Toxicity

Figure 1 schematically presents the relationships between aquatic plant nutrients and dissolved oxygen depletion. It shows how nutrients, via their role in DO depletion, can

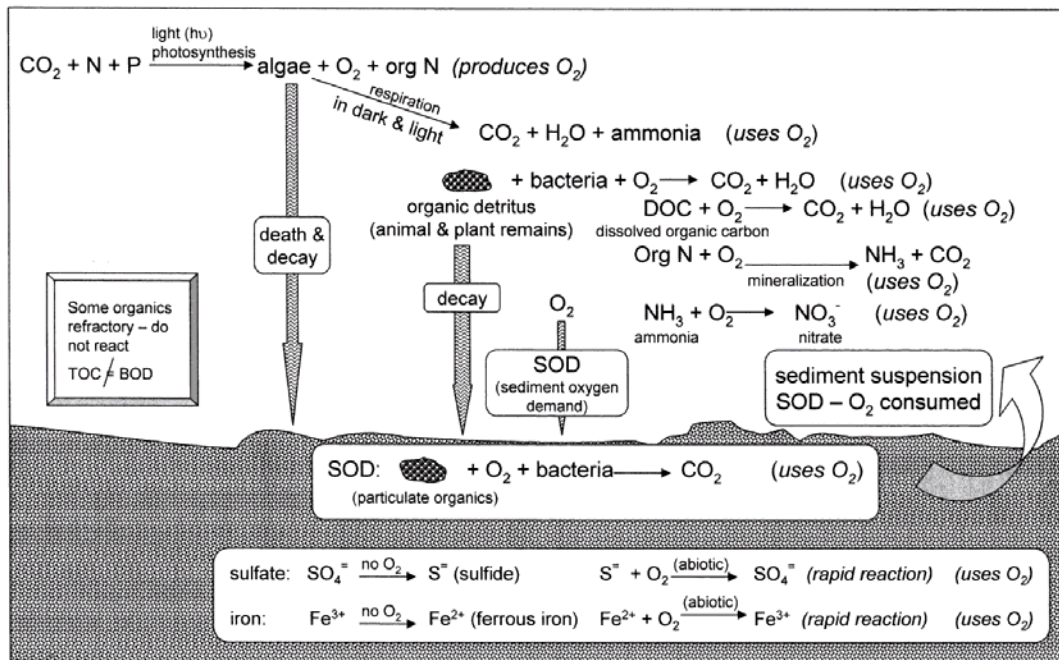


Figure 1. Algae & Organic Detritus as Sources of Oxygen Demand

be a driving factor in sediment toxicity caused by low DO, hydrogen sulfide, and ammonia. In short, nutrient additions drive the production of planktonic algae, which die and settle to the sediments. Bacterial decomposition of the dead algae consumes dissolved oxygen. Bacterial decomposition of accumulated algae and other detrital material in the sediment can cause anoxic (oxygen-free) conditions in the sediment, which in turn can result in the reduction of sulfate to sulfides, and ferric to ferrous iron. The accumulation of ferrous iron and sulfides in sediments can form iron sulfides/polysulfides which give many sediments a black color and cause them to exert high, rapid oxygen demand when suspended in the watercolumn. (These issues were discussed in Newsletters 10-4 and 10-5.) The low DO of sediments, and the hydrogen sulfide and ammonia that accumulate in sediments, are the most pervasive causes of sediment toxicity to aquatic life. As part of the US EPA studies of the characteristics of coastal sediment quality for the EMAP-estuaries Virginian Province – 1992, Strobel *et al.* (1992) reported that the most common cause of sediment degradation was low dissolved oxygen.

As discussed by Lee and Jones-Lee (1996), regulatory agencies typically ignore the role of low DO (and nutrients as causes of low DO), hydrogen sulfide, and ammonia in causing sediment toxicity, and limit the focus of sediment toxicity testing to that caused by traditionally considered “pollutants” including heavy metals, and organics such as pesticides, PCBs, and PAHs. In fact, the US EPA's recommended approach for testing sediment toxicity includes aeration of the test solution in order to keep the test organisms alive. This step removes, without recognition or consideration, the toxicity caused by low DO and some reduced forms of contaminants such as sulfides. Based on an overview survey of aquatic toxicity testing laboratories in northern California, I found that some laboratories test sediment samples without removal of ammonia and simply report the toxicity found. Others treat the sediments by repeated dilutions until the ammonia concentration is decreased to below toxic levels, or remove ammonia through ion exchange. The dilution/removal of ammonia prior to testing could significantly alter the manifestation of toxicity due to other constituents present in the sediment that are, in fact, toxic under environmental conditions.

Removal of ammonia, coupled with the addition of oxygen to the sediment during the testing procedure, makes results of sediment toxicity testing as typically practiced in accord with US EPA procedures, unreliable for estimating the magnitude of sediment toxicity under ambient conditions and assessing the causes of sediment toxicity that could be affecting the beneficial uses of a waterbody. Further, inadequate attention to the role of nutrients/algae in contributing to sediment toxicity through low DO can result in the overlooking of the ultimate cause of the manifested toxicity problem, and the misdirecting of toxicity evaluation and management. Inadequate attention to low DO and its causes is, therefore, a fundamental flaw in the evaluation, regulation, and management of sediment quality.

Significance of Ammonia in Causing Sediment Toxicity

An illustration of how narrow focus on the “traditional” pollutants such as heavy metals, pesticides, and other anthropogenic organics, can mislead sediment toxicity evaluation is

provided by our work on the toxicity of waterway sediments slated for dredging, as discussed by Jones and Lee (1988). The sediments in waterways in urban and industrialized areas typically contain a variety of chemical contaminants that could have adverse impacts on the aquatic organisms in those waters. In the mid- to late-1970s Lee directed an extensive laboratory and field study as part of the US Army Corps of Engineers Waterways Experiment Station Dredged Material Research Program to evaluate the contaminant release from, and toxicity of, sediments dredged from US waterways and disposed of in open water, and to develop testing protocols and criteria guidance for dredged sediment evaluation and management. More than 30 chemical parameters, including heavy metals, chlorinated hydrocarbon pesticides, PCBs, and nutrients, were measured in the sediments from about two dozen locations across the US.

Typically the waterway sediments investigated were collected near urban industrial areas and were therefore likely to have been contaminated by chemical constituents in domestic wastewaters, urban stormwater runoff, industrial wastewater and stormwater runoff, and runoff from upstream agricultural areas. Contaminant release from the sediments was evaluated in elutriate test protocols designed to simulate worst-case open water disposal of hydraulically dredged sediments. Further, as part of evaluating the reliability of laboratory-based test results, contaminant release and receiving water characteristics were monitored during a variety of types of open water disposal operations involving evaluated sediments (see Lee *et al.*, 1978 and Jones and Lee, 1978 for details, and the summary in Newsletter 10-5). The sediments were also subjected to laboratory toxicity testing in a manner developed for the conditions being investigated, using the epibenthic grass shrimp (*Palaemonetes pugio*) for the marine systems and daphnids (*Daphnia magna*) for freshwater systems, in toxicity testing systems containing settled sediment. This sediment toxicity testing was conducted by G. Mariani (1979) as part of his PhD dissertation at the University of Texas at Dallas (Lee *et al.*, 1978 and Jones and Lee, 1978).

As expected, it was found that many of the sediments were laden with a wide variety of heavy metals, chlorinated hydrocarbon pesticides, PCBs, and nutrients. However, with the exception of ammonia, these “traditional” contaminants were not typically released with vigorous mixing with site water. As shown in the partial data set included in Table 1, the sediments from a number of the waterways did show some level of toxicity under the elutriate toxicity test conditions. (Those conditions simulated those that exist in hydraulic dredge discharge without dilution in the ambient waters). There was no identifiable relationship between the sediment concentration of the various individual (or collective) heavy metals and chlorinated hydrocarbon pesticides measured, and the mortality found in the elutriate toxicity tests for the more than 250 elutriate tests run during the study.

Further, of all the chemical contaminants measured in the sediments and elutriates, only ammonia was released in elutriate tests in potentially significant amounts that could cause toxicity. The heavy metals and other potentially toxic constituents measured were not released to the test water in amounts that would be expected to be toxic; nor were they in forms available to test organisms in contact with the settled sediments. These test results

were in keeping with the finding that these constituents were also not released during actual dredged sediment disposal operations monitored during our field studies in conjunction with the elutriate testing.

TABLE 1—Summary of acute toxicity for selected U.S. waterway sediments Ref. (9).

Sediment Location	Percent Surviving ^a at 96 h— Sediment Percentage					
	5%		10%		20%	
	(A B) ^b	\bar{X}	(A B)	\bar{X}	(A B)	\bar{X}
Mobile Bay, AL		100		...		100
San Francisco Bay, CA						
Rodeo Flats	(90,100)	95	(70,70)	70	(60,80)	70
Mare Island	(100,90)	95	(80,90)	85	(80,80)	80
Oakland Inner Harbor		100		...		100
Los Angeles Harbor, CA						
Buoy A-7 (Site 1)	(70,80)	75		...	(40,...)	40
Buoy C-2 (Site 2)	(90,100)	95		...	(90,100)	95
Bridgeport, CT	(80,70)	75	(50,40)	45		...
Norwalk River, CT						
North Site		100		...		100
South Site		100		...	(90,90)	90
Stamford, CT						
West Branch		100		...	(90,100)	95
Apalachicola, FL						
Site 1		100		...	(80,100)	90
Site 5	(90,90)	90		...	(80,80)	80
Menominee River, MI		100		...	(40,30)	35
Upper Mississippi River, St. Paul, MN		100		...		100
Hudson River, NY						
Foundry Cove— <i>P. pugio</i>		(100,90)	95
— <i>D. magna</i>		(90,80)	85
New York-New Jersey Harbors						
Perth Amboy Anchorage	(80,70)	75		...	(80,70)	75
Perth Amboy Channel	(60,80)	70		...	(40,40)	40
Bay Ridge Channel	(100,50)	75		...	(100,90)	95
Perth Amboy Channel—Site 1		(90,80,90)	87
—Site 2		(60,60,60)	60
—Site 3		(80,80,90)	83
Ashtabula Harbor, Lake Erie, OH						100
Newport, RI						
Offshore		100		Reproduction occurred		100
Corpus Christi, TX						
Site 3	(90,80)	85	(80,100)	90	(90,90)	90
Houston Ship Channel, TX						
Site 2		100		...	(90,90)	90
Site 3		100		...		100
Morgan's Point, TX	(90,90)	90	(80,80)	80	(70,60)	65
Port Lavaca, TX		100		100		100
Galveston Bay Entrance Channel, TX						
Buoy 1	(90,90)	90		100	(90,80)	85
Buoy 9		...		100		...
Buoy 11		100		100	(90,90)	90
Texas City Channel, TX						
Site 1		100		100		80
Site 2	(90,90)	90	(90,90)	90	(80,80)	80
Site 3	(90,90)	90	(90,90)	90	(80,80)	80
Site 4	(80,100)	90	(90,70)	80	(90,80)	85
Site 5		100		100		100
Site 6		100		100	(90,100)	95
Bailey Creek, VA (4/76)		100		...	(80,100)	90

The sediments and water from the Perth Amboy Channel and Anchorage in New Jersey, and Bay Ridge Channel in New York showed some of the highest elutriate-test toxicity of those sediments evaluated. At the time of the study in the mid-1970s, that area of the New York/New Jersey harbor received approximately 12 million m³ per day of industrial and domestic wastewaters from New York and New Jersey sources, much of which was discharged without treatment; the sediments there provided a sink for many of the chemical contaminants contained in those discharges and from the upstream discharges to

the Hudson River and several New Jersey rivers. Table 2 presents the bulk chemical concentrations of heavy metals in the New York Harbor sediments we evaluated and shows that if released to the water in available forms, many could cause acute aquatic life toxicity.

Table 2. Heavy Metal Concentrations, New York Harbor Area Sediments (mg/kg dry wt)

Sample Designation	Manganese		Cadmium		Chromium		Zinc		Nickel		Lead		Copper		Iron*		Mercury		Arsenic	
	\bar{X}	SD	\bar{X}	SD	\bar{X}	SD	\bar{X}	SD	\bar{X}	SD	\bar{X}	SD	\bar{X}	SD	\bar{X}	SD	\bar{X}	SD	\bar{X}	SD
Perth Amboy Channel	99	72 ^a	1.7	0.6	18	17	140	127	11.6	9.1	8.9	0.3	380	39	14	5	3.44	0	15.1	12.6
Perth Amboy Anchorage	245	111	3.3	2.5	69	33	97	94	30	20	84	67	487	102	15	7	4.77	0.18	57.5	17.9
Bay Ridge Channel	183	126	6.9	3.4	3.2	2.9 ^a	103	95	2.5	0.7	48	67	257	128	16	10	2.21	0.07	<5	0

NOTE: Mean and standard deviation calculated from triplicate analyses, except for mercury and arsenic, which were calculated from duplicate analyses.
*g/kg

Because ammonia was released however, and new information had been developed on the toxicity of ammonia to grass shrimp (Hall *et al.*, 1978) the mortality found in the New York/New Jersey elutriate toxicity tests was revisited in the 1980s by R. A. Jones as part of a New Jersey Sea Grant-supported project (Jones and Lee, 1988). The organism survival after 96 hrs' exposure was plotted as a function of the un-ionized ammonia concentration found in each of the respective elutriate tests. The relationship that emerged is presented in Figure 2. This figure shows a decrease in the survival of the test organisms after 96-h exposure with an increase in concentration of un-ionized ammonia measured in the test system. Further, the concentration of un-ionized ammonia found in those systems in which the 96-h survival was about 50% was on the order of 0.3 to 0.5 mg N/L, about the level of 96-h LC50 to grass shrimp for ammonia reported by Hall *et al.* (1978). While these results are not conclusive, the toxicity exhibited by the Perth Amboy and Bay Ridge sediments may be due to the release of ammonia, with little or no effect of the myriad other potentially toxic chemicals in the sediments. These results also indicate that there is, for this data set, a relationship between the concentration of un-ionized ammonia in the sediment/water mixtures (elutriates) and the mortality of the grass shrimp in the toxicity tests.

The work of Jones and Lee in the 1980s was the first to show that ammonia is a potentially significant toxicant in aquatic sediments. Today it is recognized by those conducting sediment toxicity tests that ammonia can be an important toxicant in aquatic sediments, especially marine sediments. Any sediment quality evaluation should include consideration of the potential role of ammonia as a cause of toxicity. Sediment toxicity testing should always include measurement and reporting of the concentration of total and un-ionized ammonia in the sediment and test water before any ammonia removal or dilution in preparation for organism exposure. Further, as discussed below, it is important to give consideration to the possibility that ammonia toxicity could occur in sediments that have been "remediated" to remove more traditionally considered pollutants.

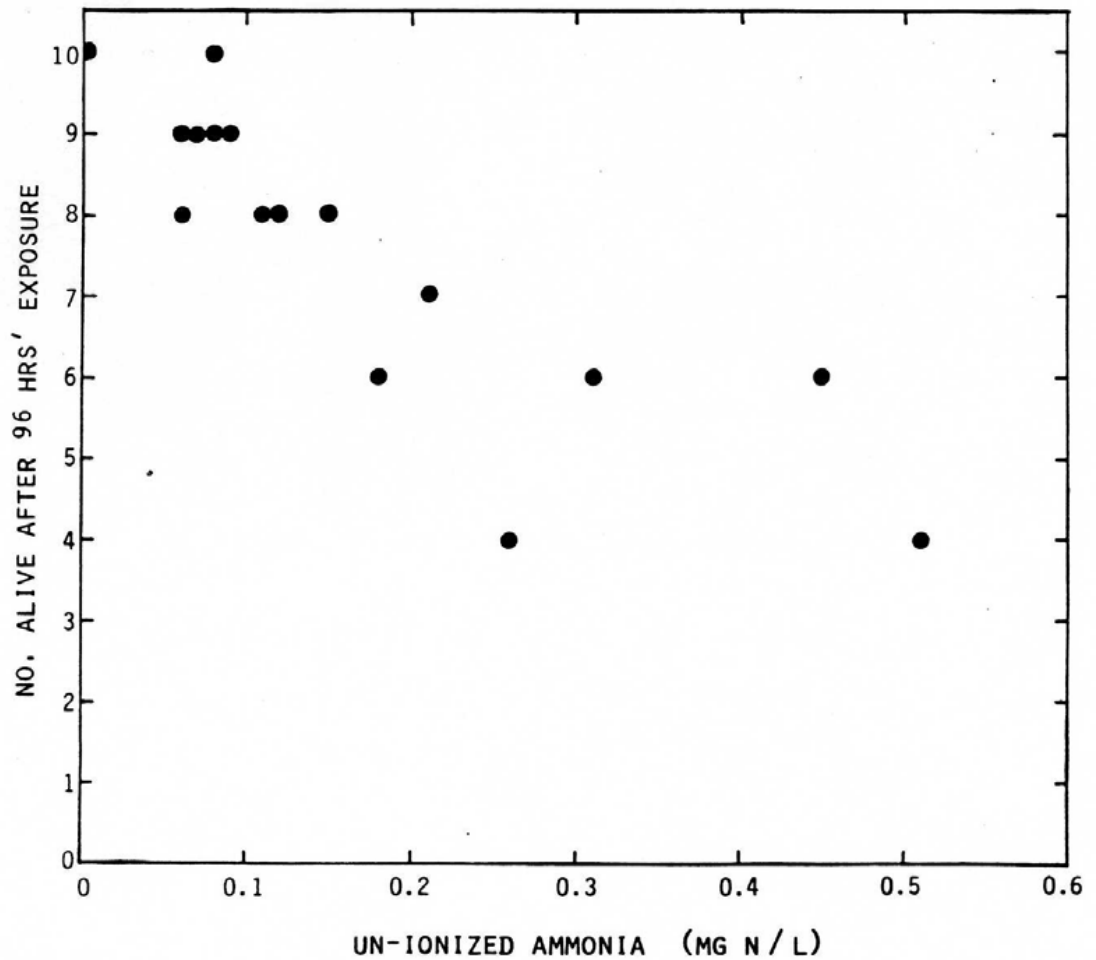


Figure 2. Survival of Grass Shrimp in Elutriate Toxicity Tests as a Function of Elutriate Ammonia Concentration – New York Harbor Sediments.

Sediment Quality Evaluation

The extensive data set on the variety of potential pollutants and aquatic life toxicity developed by Lee *et al.* (1978) demonstrated that the total concentration of potential pollutants (such as heavy metals, pesticides, etc.) in sediments is unreliable for predicting sediment toxicity. Given this, it was astonishing to find that Long and Morgan (1991) made extensive use of the Lee et al. database on the total concentration of a variety of pollutants in sediments and sediment toxicity in developing their “co-occurrence”-based sediment quality guidelines. It was well-recognized at the time their guidelines were first published, as it is today, that co-occurrence-based sediment quality guidelines are fundamentally flawed as a basis for assessing sediment quality. It is also important to note that in using the Lee et al. database, Long and Morgan did not include their data on

sediment ammonia (the constituent most likely responsible for the sediment toxicity measured by Lee et al.) in developing their sediment quality guidelines.

We have published extensively on the inappropriate use of the “co-occurrence” approach in sediment quality evaluation. Our papers and reports, as well as references to the wider literature, on this issue are available on our website

(<http://www.gfredlee.com/psedqual2.htm>). Lee and Jones-Lee (2004) discussed why sediment chemical composition (such as the total concentration of a potential toxicant, as well as the cumulative concentration of potential toxicants) should not be used in sediment quality evaluation since it can readily lead to erroneous conclusions regarding the cause of toxicity and inappropriate sediment remediation procedures.

The state of California Water Resources Control Board (SWRCB) is in the process of developing sediment quality objectives for the state’s enclosed bays and estuaries. Information on that effort is available at

<http://swrcb2.waterboards.ca.gov:8765/query.html?qt=Sediment%20Quality%20Objectives>. Unfortunately, the SWRCB’s triad approach for sediment quality evaluation, as is currently being developed, includes total concentrations along with sediment toxicity and altered organism assemblages. While the SWRCB staff recognizes that there is no relationship between the total or collective concentration of individual pollutants and sediment toxicity, it is still advocating inclusion of total concentrations as a key parameter(s) in identifying whether a sediment is potentially significantly adverse to the beneficial uses of a waterbody. If this approach is adopted by the SWRCB, it can result in erroneous sediment quality evaluations which could, in turn, lead to large-scale public and private expenditures for sediment “remediation” and source control while failing to address sediment-associated contaminants such as ammonia, low DO, sulfide, and others, which, while not included in the evaluation approach, are in fact causing real, significant water quality problems.

Lee and Jones-Lee (2004) also discussed how chemical information should be used in sediment quality evaluation. They recommended that sediment toxicity and organism assemblages, relative to what should be present based on habitat characteristics, serve as the primary components of sediment quality evaluation. The total concentration of chemicals, individually or collectively, should not be included in such an evaluation. Chemical information should only be used in the identification of the cause of toxicity through appropriately conducted toxicity identification evaluation (TIE) procedures. A comment that is sometimes made by those with limited aquatic chemistry backgrounds about this is that there is no standard TIE procedure for sediments. Those who understand the aquatic chemistry of sediments and the complexity of the chemical processes that can take place in aquatic sediments that govern manifestation of sediment toxicity know that it is not possible for individuals with limited chemistry backgrounds to cook-book through TIE procedures to determine the cause of sediment toxicity or altered organism assemblages. Such investigations should be conducted by those with extensive sediment aquatic chemistry backgrounds.

Overall Significance of Sediment Toxicity

Lee and Jones Lee (1996) pointed out that there are many waterbodies whose sediments exhibit high degrees of toxicity due to low DO, ammonia, and hydrogen sulfide associated with fluxes of nutrients to the waterbody, i.e., eutrophic waterbodies, but yet support outstanding warmwater fisheries. It is evident that sediment toxicity does not necessarily significantly adversely impact those beneficial uses of those waterbodies. As Lee and Jones-Lee pointed out, there is a very poor understanding of the relationship between aquatic life toxicity in sediments and adverse impacts on the beneficial uses of waterbodies.

Toxicity due to low DO, sulfides, and ammonia has been found to complicate the evaluation of the water quality significance of sediment-associated pollutants such as pyrethroid-based pesticides. The US EPA recommends that the amphipod, *Hyallela azeta*, be used as a standard sediment toxicity test organism. *Hyallela* occurs naturally in some waterbodies. R. Homes (personal communication, 2007) of the California Central Valley Regional Water Quality Control Board staff, reported on the Weston *et al.* (2005) studies of sediment toxicity due to pyrethroid-based pesticides in urban streams in Roseville, CA. Homes stated,

“The Hyallela sediment test method (US EPA, 2000) requires aeration if DO is below 2.5 mg/L. We encountered DO levels in the 0.5 - 1.0 mg/L in late summer at some sites in our Roseville study - which compared toxicity with resident Hyallela distribution. Some sites were not toxic and physical habitat appeared suitable but Hyallela abundance was low compared to other sites - these sites were also correlated with very low DO.

The abundance of resident H. azteca was correlated with pyrethroid TUs (Figure 4b; $p < 0.05$; Spearman rank correlation). Sediments containing more than one TU of pyrethroids had few or no resident H. azteca. Densities were quite variable at sites having less than one TU, presumably due to factors other than pyrethroid concentrations. The distributions of resident H. azteca are consistent with the patterns of sediment pyrethroid concentrations and toxicity test results, but the patterns are confounded by other habitat factors, for example, the low dissolved oxygen concentrations in some regions of the system. The low input of water in the summer results in low current speeds, and with the accumulation of decaying riparian vegetation in the bottom of the creeks, dissolved oxygen levels can be low (measured at 1.0-7.6 mg/L in Pleasant Grove Creek, 3.6-7.8 mg/L in South Branch, and 0.5-4.5 mg/L in Kaseberg Creek.”

These results demonstrate the complexities of aquatic systems and the difficulties in using even an appropriate sediment quality triad (toxicity, benthic organism assemblages, and appropriate consideration of chemical composition) to evaluate the role of anthropogenic pollutants such as pesticides as a cause of impaired beneficial uses of waterbodies, without considering the role of DO and other potential toxicants such as ammonia and hydrogen sulfide. As discussed by Lee and Jones-Lee (1996) there is need to conduct studies to gain an understanding of the implications of sediment toxicity and altered benthic organism assemblages to the beneficial uses of waterbodies. Particular emphasis should be placed on evaluating the potential significance of low DO as a cause of sediment toxicity.

An issue that should be considered in sediment quality management is whether the sediments of an area contain sufficient rapidly-exertable oxygen demand stored in the

form of reduced iron and sulfides to cause toxic conditions at the sediment/water interface, or when the sediments are stirred into the watercolumn during storms or at other times. While the sediment/water interfaced may normally be oxic under quiescent conditions, this area could become toxic to aquatic life due to occasional suspension of sediment and the depletion of DO that results. Under these conditions large amounts of money could be spent to control the concentrations of traditional pollutants in the sediments, yet leave the aquatic life-related beneficial uses still degraded due to the occasional low DO conditions. In order to determine if this type of situation occurs it is necessary to conduct focused monitoring of the DO at the sediment/water interface during and shortly after conditions that lead to sediment suspension. If sediment suspension leads to low DO conditions, it will be necessary to control the input of aquatic plant nutrients that lead to algal development and other sources of constituents that lead to accumulation of rapid oxygen demand in the sediment.

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