THE INFLUENCE OF SEDIMENT SULFIDE ON THE STRUCTURE OF SOUTH FLORIDA SEAGRASS COMMUNITIES

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ABSTRACT

Sediment porewater sulfide affects the structure of South Florida seagrass communities because it is abundant, it is toxic, and because sulfide tolerance varies among seagrass species. In this paper, we present four datasets which illustrate the influence of sulfide on the structure and species composition of subtropical seagrass communities: 1. Porewater sulfide measurements made during seagrass die-off episodes in Florida Bay during 1990 and 2000 which show that porewater sulfide concentrations in active die-off areas (4-10 mM) far exceed normal concentrations throughout Florida Bay (<2 mM); 2. Field experiments which raised porewater sulfide concentrations up to 12 mM resulting in complete mortality of *Thalassia testudinum*, some loss of *Syringodium filiforme*, and almost no mortality of *Halodule wrightii*; 3. Guano-addition experiments performed in the early 1990's by Fourqurean et al. (1995), which we re-interpret to show that porewater sulfide concentration changes might contribute to species shifts from *Thalassia* to *Halodule* around bird roosting sites, and 4. Sediment sulfide concentrations measured in nine estuaries of the eastern Gulf of Mexico that show potentially toxic sediment sulfide concentrations in several estuaries.

We suggest that *Thalassia* is much less tolerant of high porewater sulfide concentrations than *Halodule* because *Thalassia* has a much higher ratio of below-ground:above-ground biomass. Any physical or chemical process which increases sediment sulfide concentrations can therefore cause sub-lethal, chronic stress which reduces *Thalassia* productivity. Higher concentrations can result in acute, lethal sulfide stress. Sulfide stress might also occur without changes in porewater sulfide concentrations if the oxygen balance of seagrasses is affected by decreased photosynthetic or oxygen transport capacity due to shading by phytoplankton, epiphytes, or resuspended sediments. Pathogens, physical injury, and grazing organisms can also reduce oxygen transport from seagrass leaves to roots and rhizomes, setting the stage for hypoxic stress and sulfide toxicity.

Recent measurements of porewater sulfide concentrations in several west Florida estuaries suggest that the problem of sulfide toxicity is not limited to seagrasses growing in carbonate sediments. In fact, sulfide toxicity might play a synergistic role in recent seagrass losses in Tampa Bay. Seagrass restoration efforts should consider sediment sulfide levels and sulfide tolerance of seagrass species in transplant projects.

INTRODUCTION

Although seagrasses are vital components of the nearshore ecosystem (Zieman 1982; Nelson 1992), drastic declines in the distribution and abundance of seagrass communities have occurred in many estuaries throughout the Gulf of Mexico (Lewis et al. 1982; Pulich and White 1991), the United States (Orth and Moore 1983; Dennison et al. 1993), and the world (Cambridge et al. 1986). In most cases, concurrent declines in water quality have been blamed for seagrass loss. Declines in water quality and clarity, in turn, often result from anthropogenic nutrients which stimulate estuarine phytoplankton blooms. In Tampa Bay, an 80% decline in seagrass cover over the past 100 years has been largely attributed to deterioration of water clarity (Lewis et al. 1982). Aggressive efforts to reduce nitrogen loading to Tampa Bay have resulted in improved water clarity and increases in seagrass cover between 1990 and 1996 (Johansson and Greening 2000).

Recent studies have shown that sediment sulfide concentrations can also act alone or synergistically to cause chronic, sublethal or acutely lethal stress on seagrasses (Carlson et al. 1994; Goodman et al. 1995; Erskine and Koch 2000). Sulfide is

produced naturally in anaerobic marine sediments by heterotrophic bacteria which use sulfate as a terminal electron acceptor in breakdown of organic matter (Goldhaber and Kaplan, 1975). Because seagrass sediments typically have high organic matter content, sulfate reduction rates in seagrass sediments are higher than in unvegetated marine sediments (Carlson et al. 1994, Holmer and Nielsen 1997). Sulfide is also a potent cytotoxin, irreversibly binding enzymes involved in electron transport for both photosynthesis and respiration (see review by Bagarinao 1992). Sulfide also causes hypoxia in seagrass roots and rhizomes by reacting with photosynthetically-produced oxygen diffusing from leaves to below-ground tissue. Marine plants and animals vary in their ability to tolerate sulfide, using a variety of avoidance strategies to exclude sulfide and accommodation strategies to detoxify sulfide (see review by Bagarinao 1992). However, the tolerance limits of seagrasses can be exceeded if sulfide accumulates to toxic levels in sediment porewater.

The amount of sulfide which accumulates in seagrass bed sediments depends on a number of physical and chemical characteristics. Tidal currents, wave action, and sandy sediments facilitate exchange of sediment porewater with the overlying water column, resulting in oxidation or export of sulfide produced by bacteria. In contrast, sulfide concentrations are generally higher in quiescent areas with fine-grained sediments. Siliceous, terrigenous sediments typically contain high concentrations of iron (up to 5%) which bind sulfide as pyrite or iron monosulfide. Biogenic carbonate sediments, such as those in Florida Bay, the Florida Keys, and Biscayne Bay, however, have very low iron concentrations, resulting in porewater sulfide concentrations which are considerably higher than those typically found in Central and North Florida seagrass beds.

In keeping with the theme of this conference "Seagrass Management: It's not just nutrients," we suggest that sediment porewater sulfide concentrations also influence the species composition, survival, and growth of seagrass beds in South Florida. If this is the case, two important ramifications for seagrass management should be considered: 1. human activities which affect organic matter and sediment accumulation in seagrass beds might increase seagrass sulfide stress; 2. Sediment sulfide levels might affect the survival and growth of transplanted seagrass beds.

We draw on data from four projects to examine the role that sulfide plays in determining the species composition, survival, and growth of South Florida seagrass communities: Sediment sulfide data collected in studies of Florida Bay seagrass die-off over the last 12 years provide powerful, albeit circumstantial, evidence that high sulfide concentrations kill turtle grass. We also performed experiments in Florida Bay seagrass beds to examine the response of *Thalassia testudinum*, *Halodule wrightii*, and *Syringodium filiforme* to elevated sulfide concentrations, and we re-sampled experiments which examined the effects of bird guano on seagrass beds (Fourqurean et al. 1995). Finally, we compared sulfide concentrations in seagrass beds of nine estuaries located along the coast of the Gulf of Mexico from the Chandeleur Islands to Florida Bay.

STUDY AREA AND METHODS

Florida Bay Seagrass Die-Off Studies. We have measured porewater sulfide concentrations associated with die-off episodes of *Thalassia* since 1988. Two major episodes have occurred in that time period. The first episode occurred between 1987 and 1991, affecting over 10,000 ha. of seagrass beds, primarily in the central and western portions of Florida Bay (Robblee

et al. 1991). The second episode has occurred since 1998 in a much smaller area near Barnes Key near the southern edge of Florida Bay, and it is still ongoing.

In the initial die-off studies, we collected small (60 cm^3) cores from three die-off patches in each of three basins affected by die-off (Rankin Lake, Johnson Key Basin, and Rabbit Key Basin). Three replicate cores were collected in February, June, and October each year from dead areas, fringes of die-off patches, and surviving seagrass surrounding the die-off patches. Cores were submersed in chilled seawater for transport, and porewater sulfide was determined in the laboratory using a sulfide ion-specific electrode (Carlson et al. 1994). Samples were collected from die-off patches in 1989 and 1990. Additional porewater sulfide samples were also collected from equilibrium-dialysis samplers deployed in a die-off patch and in an adjacent, surviving grass bed in Johnson Key Basin in fall 1990.

Porewater sulfide sampling was also carried out quarterly between 1994 and 1996 at 24 sites throughout Florida Bay using the same coring and analysis techniques. Quarterly sampling of porewater sulfide at the Barnes Key die-off site began in October 1999 and is continuing.

Sediment Perfusion Experiments. As part of our investigation into the role of sulfide in seagrass die-off in Florida Bay, we have carried out a series of experiments to determine the response of seagrasses to elevated porewater sulfide concentrations. We removed the bottoms from 15 5-gallon plastic buckets and inserted them into the sediments of dense, nearly-monospecific beds of *Thalassia testudinum*, *Halodule wrightii*, and *Syringodium filiforme* near Man O'War Key in the western bay in June 1990. Each bucket isolated a column of sediment and seagrass from the surrounding bed; rhizomes around the perimeter of each bucket were severed to prevent translocation between seagrass shoots inside and outside the buckets. After allowing seagrasses to recover from bucket insertion for one month, we randomly selected three buckets for each seagrass species to serve as bucket controls and three buckets to receive glucose amendments. Three "perfusers" (porous polyethylene tubes containing 140 g glucose) were inserted into the sediments of each glucose-amended bucket. Three pieces of PVC pipe (1" nominal diameter) were inserted into each of the control buckets. A porous polyethylene porewater sampler was also inserted in the center of each bucket to allow monthly collection of sediment porewater for sulfide and pH determination. Buckets were harvested eight weeks after the perfusers were installed in the buckets. All plant material retained on a 1 mm sieve was retained for determination of live and dead above- and below-ground biomass. Seagrass shoots were classified as live, new-dead, or old-dead, counted, and measured.

Bird Stake Experiments. Several stakes were installed as bird roosts on Cross Bank by J. Fourqurean and G. Powell in 1981 (Powell et al. 1989, Fourqurean et al. 1995). Approximately two years after stakes were installed, five stakes were pushed down into the sediment so they could no longer serve as bird roosts. In July 1991, ten years after the experiment began, we collected duplicate sediment cores adjacent to each of twelve stakes: four active bird roosts, four stakes which had been pushed into the sediments, and four control sites. Cores were transported in chilled seawater and analyzed using a sulfide ion-specific electrode as described above.

Comparison of Gulf Coast Estuaries. Porewater sulfide concentrations were measured at 25–30 sites in nine estuaries

distributed between the Chandeleur Islands and Florida Bay. At each site, we collected 60-cm^3 cores and measured sulfide using an ion-specific electrode.

RESULTS AND DISCUSSION

Florida Bay Seagrass Die-Off Studies. Vertical profiles of sediment porewater sulfide concentrations of surviving *Thalassia* beds and die-off patches at Johnson Key Basin differed in several ways (Figure 1). Sulfide concentrations were low near the sediment surface and increased with depth in the sediments for both profiles. In the surviving grass bed, however, maximum sulfide concentrations ~3 mM occurred at a depth of 8–10 cm, corresponding roughly to the depth where most rhizomes are located. Sulfide concentrations in the die-off patch increased to values near 1.5 mM at a depth of 20 cm in the sediments. Low porewater sulfide concentrations of the die-off patch sediments probably resulted from depletion of seagrass organic matter in the year that elapsed between the time that seagrass died and the time we sampled. Previous studies have shown that sediment sulfide concentrations increase dramatically during die-off episodes and then decline slowly over 12 to 18 months (Carlson et al. 1994).

During initial die-off investigations in 1989 and 1990, mean porewater sulfide concentrations in sediments of Johnson Key Basin, Rabbit Key Basin and Rankin Lake were 1.6 to 1.8 mM (Table 1A). Concentrations varied seasonally with concentrations generally lower than 1 mM in spring and summer. Concentrations in

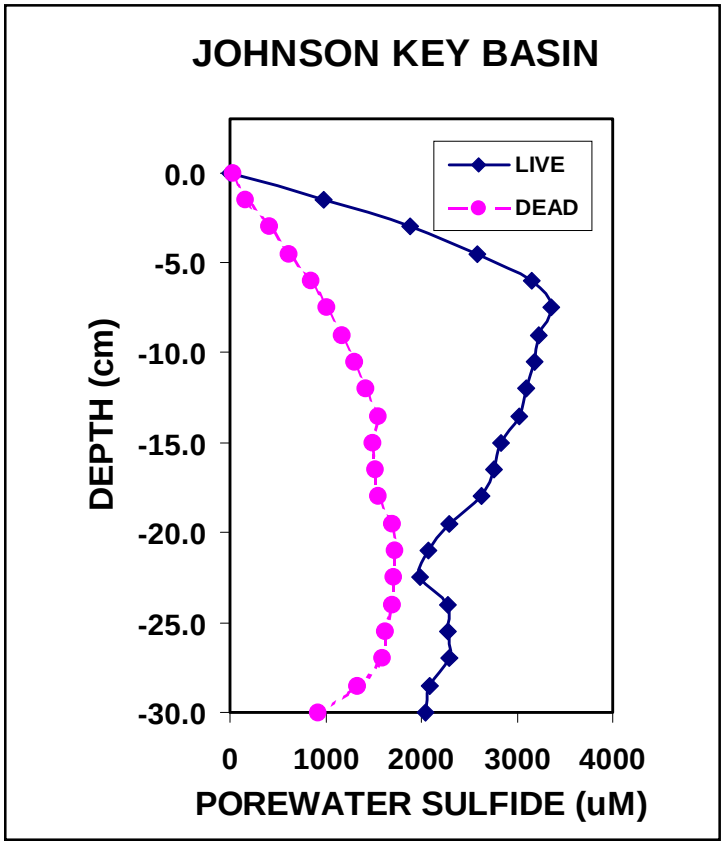

Figure 1. Vertical profile of porewater sulfide in die-off patch sediments and adjacent surviving *Thalassia* testudinum bed, Johnson Key Basin, fall 1990.

Table 1. Florida Bay sediment porewater sulfide concentrations,1989 to 1999. Data are mM (mmol S= per liter porewater). Within each grouping (season, year, or region), values with the same letter subscript are not significantly different. Barnes Key 1999 data are means followed by standard deviation values in parentheses.

A. Initial Die-Off Period 1989–1991		
Basin averages	Johnson Key Basin	1.7 (1.4)
	Rabbit Key Basin	1.6 (1.2)
	Rankin Lake	1.8 (1.7)
Seasonal averages	June 1989	0.52 (0.09)
	October 1989	2.30 (0.69)
	February 1990	0.86 (0.34)
	June 1990	0.88 (0.24)
	October 1990	3.80 (0.89)
B. Baywide averages 1994–1996		
Annual averages	1994	0.62 a
	1995	0.59 a
	1996	0.71 a
Seasonal averages	Spring	0.54 b
	Fall	0.73 a
Regional averages	North-central	0.10 a
	Western	0.75 b
	Southeast	0.48 c
	Northeast	0.47 c
	South-central	0.47 c
Barnes Key site	May 1994	0.53
	October 1994	0.90
	May 1995	0.66
	October 1995	0.56
	April 1996	0.43
	September 1996	0.76
	May 1997	0.68
C. Barnes Key die-off site, October 1999		
	Old die-off	2.70 (0.92)
	Dense *Thalassia*	3.30 (0.55)
	New die-off	4.70 (1.10)

October of 1989 and 1990 were 2.3 and 3.8 mM, respectively, coinciding with *Thalassia* die-off events. In active die-off patches, Carlson et al. (1994) measured porewater sulfide concentrations over 13 mM. Although much of the sulfide production in active die-off patches is fueled by microbial decomposition of dying roots and rhizomes, elevated porewater sulfide concentrations (1.5–1.8 mM) preceded die-off by three months at one monitored site in Johnson Key Basin in 1990, indicating that events which stimulated sediment sulfide production in summer might have caused die-off directly or made *Thalassia* vulnerable to other stressors.

Between 1994 and 1996, no large seagrass die-off episodes occurred in Florida Bay and porewater sulfide concentrations sampled at 24 sites throughout the Bay ranged between 0.47 mM and 1.1 mM (Table 1B). Highest concentrations occurred in the north-central region of the Bay (1.1 mM), followed by the western region (0.75 mM). The southeast, south-central, and northeast regions all had porewater sulfide concentrations less than 0.5 mM.

These values were significantly lower than mean concentrations measured in the western Bay during the first major die-off episode (Table 1A). During this same period, surface water salinity in Florida Bay dropped from hypersaline values to 25–35 ppt (Everglades National Park Marine Monitoring Program, unpublished data). Although the Barnes Key die-off episode has cast doubt about the importance of hypersalinity as a contributing cause for *Thalassia* die-off episodes, the drop in porewater sulfide values from 1.8 to 1.1 mM in the north-central region and a decline from 1.7 to 0.75 mM in the western Bay over the time period between 1989–90 and 1994-96 is statistically significant. The drop might indicate a regional-scale influence—climatic, hydrographic, or biological—which had contributed to *Thalassia* die-off in 1987–91 declined during this period.

During the period 1994–96, annual variation in porewater sulfide concentrations was not significant, but fall values (0.73 mM) were significantly higher than spring (0.54 mM). Both sampling periods (1989–90 and 1994–96) exhibited the same seasonal trends suggesting that processes operating in the fall cause elevated porewater sulfide concentrations. As noted above, *Thalassia* die-off episodes have also occurred during the fall suggesting a link between the seasonal dynamics of sediment sulfide and *Thalassia* die-off. The Bay-wide survey data support this link, but they also indicate that a moderate rise in porewater sulfide concentrations during fall is not sufficient by itself to cause die-off. The processes that cause the seasonal cycle in porewater sulfide concentrations are not known but Yarbro and Carlson (1989) suggested that hypoxic stress of *Thalassia* might occur in fall as the combined result of declining day length and warm water temperatures.

During the 1994–96 period, porewater sulfide concentrations at one sampling point near Barnes Key fluctuated between 0.43 and 0.9 mM. When a new episode of *Thalassia* die-off occurred in this area in fall 1999, porewater sulfide concentrations over 6 mM were measured (Table 1C). Even surviving *Thalassia* beds had porewater sulfide concentrations over 3 mM, suggesting the potential for sulfide stress, although no visible stress symptoms were observed.

Sediment Perfusion Experiments. Addition of glucose to bucket sediments caused significant increases in porewater sulfide, indicating that sulfate reduction rates in Florida Bay sediments are limited by the availability of labile organic matter rather

than by sulfate. Initial porewater sulfide concentrations measured May 16, 1991 ranged from 0.8 to 1.2 mM with no significant difference among species or treatments (Table 2). Final concentrations varied markedly among treatments. Glucose-amended buckets of all three species had porewater sulfide concentrations between 10 mM and 13 mM. Concentrations within control buckets ranged from 2 mM in *Halodule* beds to 2.5 mM in *Syringodium* beds, to 3.5 mM in *Thalassia* beds. Sediments outside the buckets had the lowest sulfide concentrations, generally less than 1 mM.

Seagrass survival was inversely related to sediment sulfide concentrations (Table 2). Lowest mortality (5%) occurred in outside control plots in *Thalassia* beds and in control buckets in *Halodule* beds. Mortality in control buckets in *Syringodium* and *Thalassia* beds (8% and 15%, respectively) was slightly higher, indicating that the process of severing rhizomes and installing buckets does kill some seagrass shoots.

Seagrass mortality in glucose-amended sediments of *Halodule* and *Syringodium* beds (5% and 12%, respectively) was slightly, but not significantly, higher than controls. *Thalassia* mortality in glucose-amended buckets was approximately 70%, much higher than in control buckets. We interpret these results to indicate that *Thalassia* is very sensitive to elevated sulfide concentrations, *Syringodium* is less sensitive, and *Halodule* is relatively insensitive to elevated sulfide concentrations in the range of 10–13 mM, values which were measured in sediments of active die-off patches.

One difference among these three seagrass species which might account for their differing sulfide tolerance is their below-: above-ground biomass ratios. Species which have a proportionally large investment in belowground tissue might be more vulnerable to sulfide toxicity than a species with less belowground biomass. Up to 80% of *Thalassia* biomass is roots and rhizomes, while *Syringodium* and *Halodule* roots and rhizomes typically comprise less than 30% and 20% respectively of total plant biomass (Kenworthy and Thayer 1984). In our experiments, *Thalassia*, the species with the greatest fraction of

Table 2. Porewater sulfide concentrations and seagrass shoot mortality in sediment perfusion experiments, summer 1991. Shoot mortality is calculated as the percent of shoots present at the beginning of the experiment within each bucket.

		TREATMENT:		
PARAMETER		OUTSIDE CONTROLS	CONTROL BUCKETS	GLUCOSE-AMENDED BUCKETS
Initial Porewater Sulfide Concentration (mM)	*Thalassia* beds	—	1.2	1.2
	Syringodium beds	—	1.0	1.0
	Halodule beds	—	0.8	0.8
Final Porewater Sulfide Concentration (mM)	*Thalassia* beds	1.0	3.5	13.0
	Syringodium beds	—	2.5	11.0
	Halodule beds	—	2.0	10.0
Shoot Mortality	*Thalassia* beds	5%	15%	70%
	Syringodium beds	—	8%	12%
	Halodule beds	NA	5%	5%

belowground biomass, was the most vulnerable to sulfide toxicity. *Halodule*, the species with the lowest ratio of below-:above-ground tissue, was least affected by elevated sediment sulfide concentrations.

Our results are consistent with those of Goodman et al. (1995) who found that photosynthesis and growth of *Zostera marina* was inhibited by experimentally-elevated sulfide concentrations. Our data are also remarkably consistent with Terrados et al. (1999), who found that sucrose addition to sediments in a Phillipine seagrass bed resulted in significant mortality and slower growth in *Thalassia hemprichii* shoots, while *Halodule uninervis* and *Syringodium isoetifolium* survival and growth did not differ significantly from controls.

In contrast, laboratory experiments by Erskine and Koch (2000) found that short-term (48-hour) exposure of *Thalassia testudinum* to sulfide concentrations up to 10 mM caused lower leaf elongation rates and lower adenylate charge ratios, but the effects were found to be reversible. They concluded that sulfide could not "initiate rapid die-off episodes of *T. testudinum* in Florida Bay." However, their results were influenced by the short-term exposures used in their experiments and by removal of root and rhizome tissue from *Thalassia* shoots prior to sulfide exposure. In the natural environment, *Thalassia* roots and rhizomes are continuously exposed to sulfide, and natural *Thalassia* beds have high root:shoot ratios.

One additional result of the perfusion experiments is noteworthy: glucose amendments not only increased sediment sulfide concentrations but they also caused sediment pH values to decline from approximately 7.5 to values below 6.5 (Figure 2). Becaused the pK1 of H_2S is 7.0, the 1.0 unit decline in pH causes a three-fold increase in the fraction of total porewater sulfide present as H_2S (Goldhaber and Kaplan 1975). Hydrogen sulfide (H_2S) has been shown to be more toxic than bisulfide (HS-) because H_2S penetrates plant tissues more easily. Not only did sediment sulfide concentrations increase 10-fold in glucose-amended sediments, but the fraction of gaseous H_2S also increased by a factor of three, resulting in a 30-fold increase of the most toxic fraction of the sediment sulfide pool.

Because all experimental studies to date have shown deleterious effects of sulfide on seagrass survival and growth, we conclude that chronic sulfide toxicity is at least a synergistic influence on the die-off of *Thalassia* in Florida Bay. It is also likely that acute toxicity caused by elevated sulfide concentrations is sufficient by itself to cause die-off.

Bird Stake Experiments. Previous studies (Powell et al. 1989, Fourqurean et al. 1995) found that seagrass communities adjacent to bird roosts changed over a period of several years from *Thalassia* dominated beds to monospecific beds of *Halodule*. They concluded that, under nutrient-rich conditions caused by bird guano deposition, *Halodule* outcompeted *Thalassia*. When we resampled bird roosts on Cross Bank in 1991, we found that, in addition to higher sediment nutrient concentrations, sites where birds actively roosted had elevated sediment sulfide concentrations (Figure 3). In fact, mean porewater sulfide concentrations adjacent to active roosting stakes were 2 mM. Adjacent to stakes where birds could no longer roost, mean porewater sulfide concentrations were 1.2 mM. Control sites had porewater concentrations of approximately 0.8 mM. We also found that porewater ammonia and porewater sulfide concentrations in seagrass sediments were positively correlated (Figure 4).

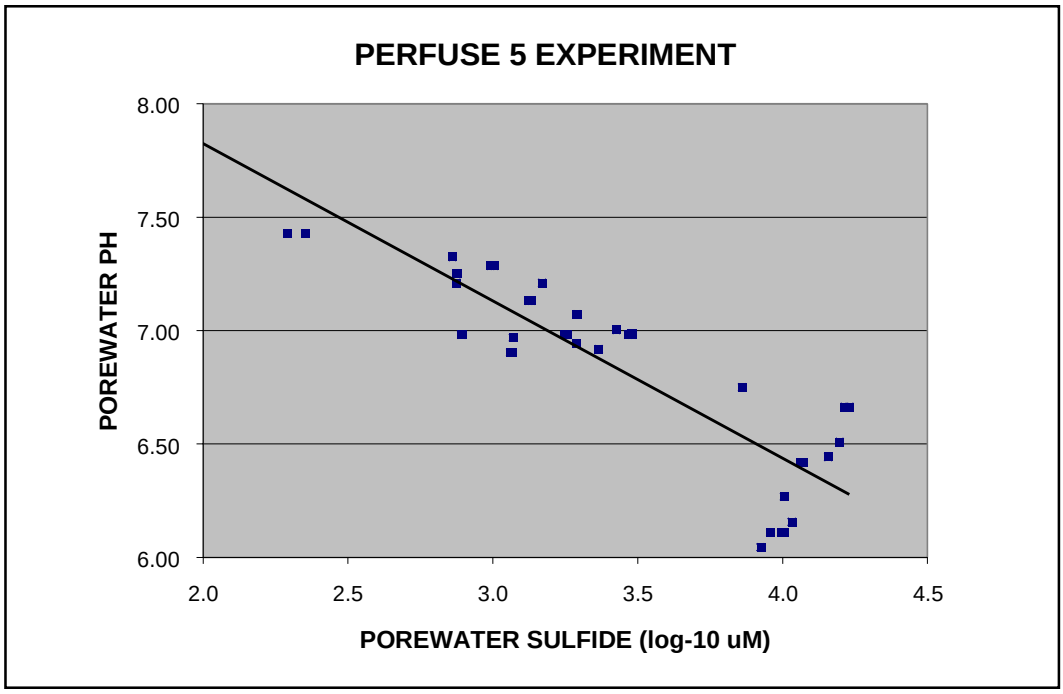

Figure 2. Relationship of sediment porewater pH and sulfide in sediment perfusion experiments.

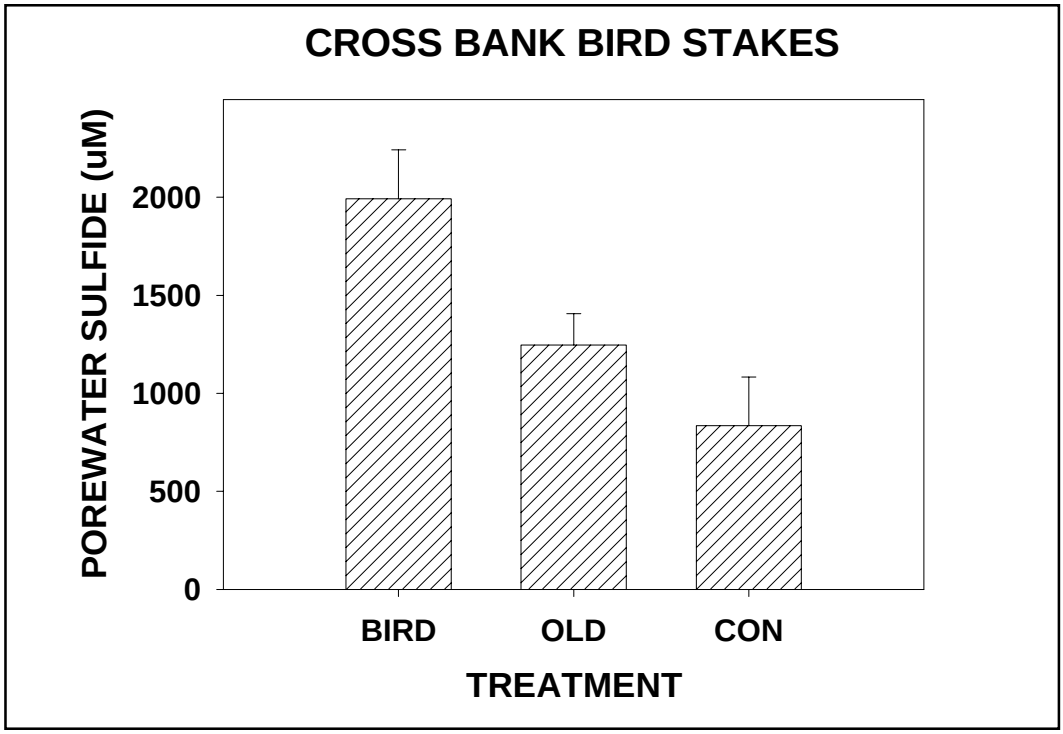

Figure 3. Effect of bird guano on sediment porewater sulfide concentrations. See Fourqurean et al. 1995 for experimental details.

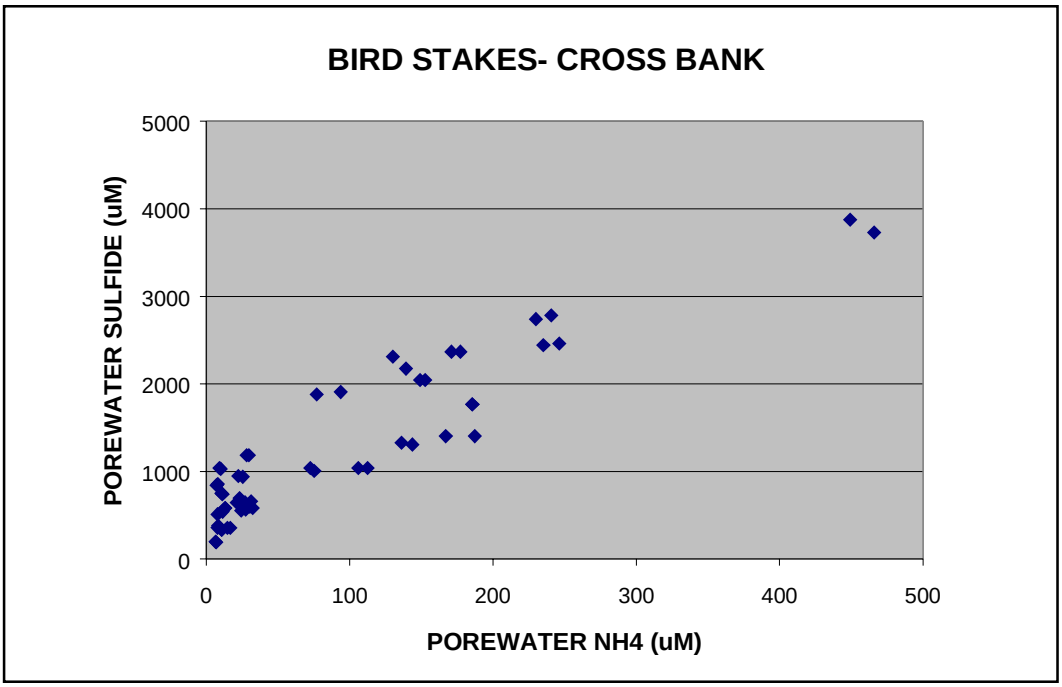

Figure 4. Relationship of porewater ammonia and porewater sulfide concentrations in sediments receiving bird guano.

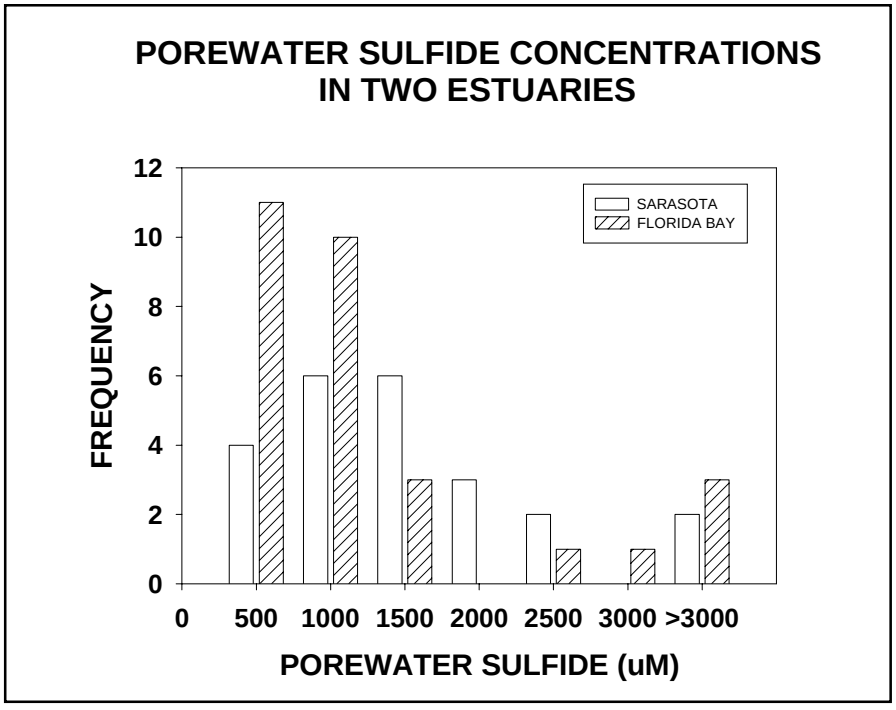

Figure 5. Frequency distribution of porewater sulfide concentrations in biogenic carbonate sediments of Florida Bay (Rabbit Key Basin) and siliceous, terrigenous sediments of Sarasota Bay.

Our porewater sulfide and ammonia data suggest another interpretation of these results. If sediment microbial communities in general, and sulfate-reducing bacteria specifically, are nutrient-limited, then guano addition stimulates heterotrophic microbial activity in the sediments. Porewater sulfide concentrations double, and, because *Halodule* is more tolerant to sulfide than *Thalassia, Thalassia* is slowly replaced by *Halodule*.

Surveys of Gulf Coast Estuaries. We originally anticipated that sulfide toxicity was a phenomenon limited to the iron-poor biogenic carbonate sediments of Florida Bay and the Florida Keys. However, in our survey of several estuaries, we found that, while mean sulfide concentrations were less than 2 mM in all estuaries, some estuaries had individual sampling stations with values greater than 2 mM (Figure 5). For example, seven sampling points at Perdido Key near Pensacola and four in Sarasota Bay had sulfide concentrations greater than 2 mM, a value that our work and other studies support as a toxic threshold, at least for *Thalassia testudinum*.

CONCLUSIONS

Our research suggests four conclusions: 1. Acute sulfide toxicity can kill *Thalassia testudinum*; 2. The acute toxicity threshold for *Thalassia* is between 2 mM and 3 mM; 3. *Halodule wrightii* tolerates much higher sulfide concentrations; and 4. Chronic sulfide toxicity causes shifts in seagrass species composition. Like other plant species, *Thalassia* and other seagrasses are probably affected by both direct and indirect sulfide toxicity effects. The direct, cytotoxic effects result from the reaction of sulfide with enzymes required for photosynthesis and respiration. Indirect toxicity effects are caused by hypoxia when photosynthetically-produced oxygen oxidizes sulfide which enters roots and rhizomes.

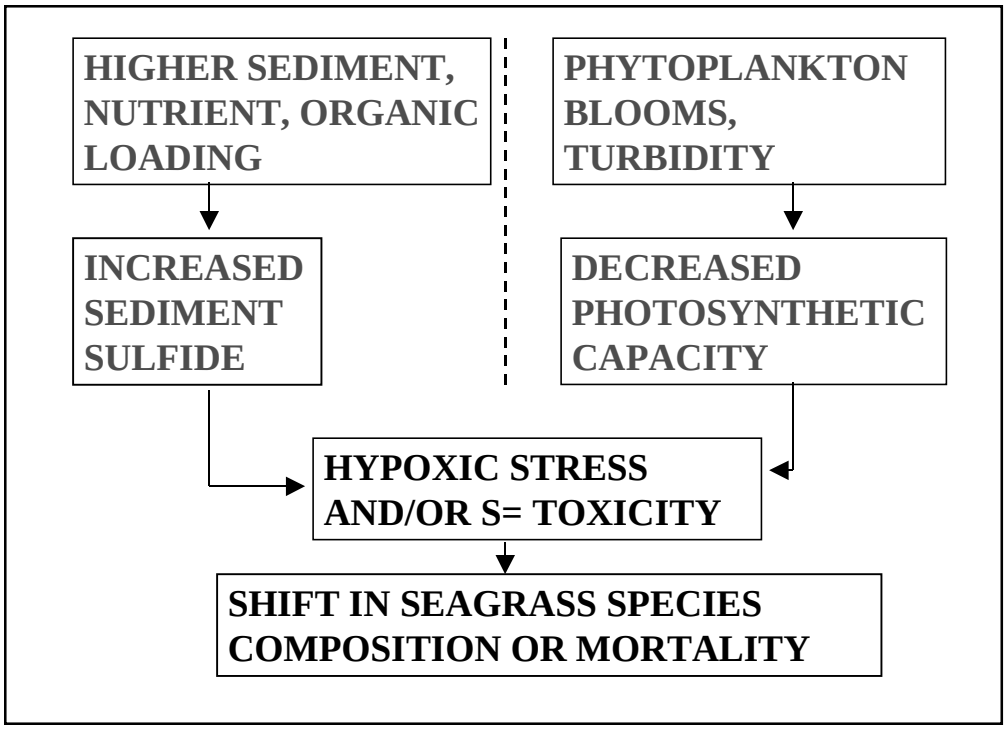

Figure 6. Factors affecting the vulnerability of seagrasses to hypoxia and sulfide toxicity.

Oxygen production and transport within plants is the key to resistance to hypoxia and sulfide toxicity, and seagrass survival depends on a balance between the plant's oxygen supply and sediment porewater sulfide as shown in Figure 6. Any process which causes elevation of sediment sulfide increases hypoxia or sulfide toxicity in seagrasses. Sulfide toxicity can also be increased by factors which decrease seagrass photosynthesis. If the balance between the internal oxygen supply of seagrasses is shifted slightly, seagrass species with higher sulfide tolerance might replace less tolerant species over a period of years.

ACKNOWLEDGEMENTS

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