COMMENTARY

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CONTINENTAL SHELF HYPOXIA: SOME COMPELLING ANSWERS.—In a commentary in this journal entitled "Continental Shelf Hypoxia: Some Nagging Questions," Rowe and Chapman (2002) expressed concern about oversimplification of the degree to which land-based nutrients, particularly nitrate, are the cause of the widely publicized oxygen depletion in the northern Gulf of Mexico. They ask, "What if nitrate loading is not the only factor controlling hypoxia on the Louisiana continental shelf? What if turning down the nitrate in the river has little or no effect on the hypoxia?"

In my considered view, there are already clear answers to both questions. Regarding the first question, it is well recognized that nitrate loading is not the only factor responsible for hypoxia but that an increase in nitrate loading is the predominant cause of the long-term increase in its extent and severity. To answer the second question, there is compelling evidence and reasoning that reducing nitrate loading will significantly reduce hypoxia.

I have had a good vantage point from which to observe the development of science in relation to these questions during the past 20 yr. In the mid-1980s, I was involved in originating the first concerted research on Gulf hypoxia, including struggling with how to address the same questions about the factors responsible for hypoxia that Rowe and Chapman now raise. After 1990, I was no longer an active participant and focused much of my attention on scientific research, monitoring, and assessment related to the abatement of similar eutrophication phenomena in the Chesapeake Bay. In 1999, I served as a member of the editorial board responsible for peer reviews of six technical reports, examining various aspects of the Gulf hypoxia issue for the Committee on Environment and Natural Resources of the President's National Science and Technology Council. This provided me intimate knowledge of the evidence, arguments, and criticisms addressed by Rowe and Chapman.

Regarding the factors controlling hypoxia, it appears either that Rowe and Chapman did not read very carefully the literature they cited or they elected to discount it for reasons not stated. Specifically, the role of freshwater discharge, stratification, and circulation on the formation and maintenance of hypoxia has been extensively addressed by Wiseman et al. (1997) and Rabalais et al. (1999), among other publications. Stratification, particularly resulting from lower-density surface waters freshened by river discharges, is a critical element of the conditions causing bottom-water hypoxia. The greater the freshwater discharge, the stronger and more extensive is the stratification. Similarly, the long retention time of bottom waters, which results from low current velocities and the propensity for summer flow reversal, has been recognized as a factor of hypoxia by the same authors. As Rowe and Chapman put it, because of these physical characteristics, the Louisiana shelf is "primed to become hypoxic." No one would doubt that if this continental shelf received only minor freshwater discharges or had highly energetic tidal currents that broke down density stratification or reduced residence time, there would be little hypoxia. On the other hand, nutrient enrichment of coastal systems that are similarly physically susceptible has been responsible for the onset or intensification of benthic hypoxia in many parts of the world (Diaz and Rosenberg, 1995).

Physical features and forces are also undoubtedly significant causes of the year-to-year variability in the severity, extent, and persistence of hypoxia. High river discharges increase stratification, whereas wind mixing due to storms and hurricanes can diminish it. However, regarding the effects of land-based nutrient inputs, the operative question is the degree to which the increased input of nitrate has affected hypoxia beyond what might be attributable to the physical effects of freshwater discharge variability. The Committee on Environment and Natural Resources (2000) addressed this question in its integrated assessment based on the six technical component reports mentioned above. It concluded, "River discharge and nitrate concentrations, and sediment core data, provide almost 100 yr of record for this system. On that time scale, there is no indication that climate factors override the impacts of human activities in the basin. Average annual flow in the Mississippi River increased 30 percent between 1955–70 and 1980–96, compared to the 300 percent increase in nitrate flux over this period." Biological and chemical indicators in the sediment record clearly show that primary production increased (incorpo-

ration of marine-source organic carbon and biogenic silica in the sediments doubled) and hypoxia intensified (hypoxia-tolerant benthic foraminifera increased) after the 1950s despite the fact that high river discharges had also occurred during the earlier part of the century.

Increased water runoff not only affects stratification on the shelf but also increases the amount of nitrate flushed to the Gulf. Using two different approaches, Donner et al. (2002) and Justić et al. (2003) agreed that 20–25% of the increased nitrate flux between the mid-1960s to the mid-1990s is attributable to greater runoff and river discharge, with the rest due to increased nitrogen loading on the landscape. Simply stated, a wetter climate drives more of the nonpoint source loadings of nutrients (predominantly from agriculture) downstream.

This connection between hypoxia and increased nitrate loading on the Louisiana continental shelf is hardly surprising. Dramatic manifestations of nutrient overenrichment, including hypoxia, loss of seagrasses, and increased frequency of harmful algal blooms, have also been documented in a number of well-studied coastal ecosystems in North America, Europe, and Japan between 1960 and 1980, coincident with the fivefold increase in the use of manufactured fertilizers during the 20-yr period (Boesch, 2002). Given this worldwide pattern and the tripling of Mississippi River nitrate loads, associated with a sevenfold increase in fertilizer nitrogen inputs in the Mississippi–Atchafalaya Basin (Goolsby et al., 1999), it would be even more surprising if there was no increase in hypoxia on the "primed to become hypoxic" Louisiana shelf.

By now, managers and technical analysts have moved beyond the second question of Rowe and Chapman to ask by how much must nitrate loading be reduced to accomplish a significant reduction in hypoxia? In 2000, a task force representing eight federal agencies, nine states, and two tribal governments adopted a general goal of reducing on the average the area experiencing hypoxia in the northern Gulf of Mexico by two-thirds (to 5,000 km^2; Rabalais et al., 2002b), estimating that this would probably require a 30% reduction in nitrogen inputs from the Mississippi River.

In another article cited by Rowe and Chapman, Rabalais et al. (2002a) describe a mass balance eutrophication model of the shelf that simulated the effects of nutrient load reductions for 3 yr of record. With the same freshwater discharge rate as observed for these years, a 30% reduction in nitrogen loading was estimated to result in a 35–90% increase in average bottom dissolved oxygen concentration depending on the year. If they had actually read this article, how could Rowe and Chapman state that "one could equally contend that freshwater is the culprit and not nitrate"—at least not without counterargument? More recently, Scavia et al. (2003) presented a statistical model that relates the extent of Gulf hypoxia to river nitrogen load and a simple parameterization of ocean dynamics with a high degree of fidelity. Using this model in hind cast, they concluded that extensive regions of low oxygen were not common before the mid-1970s. Justić et al. (2002) reached a similar conclusion using a mathematical model simulating bottom-water oxygen content related to Mississippi River discharge and nitrate flux. In forecast, Scavia et al. (2003) predict that reductions in nitrogen loads of 35–45% would be required to meet the task force goals for reducing the scale of hypoxia.

If one does not trust models and needs a practical example of the effects of nutrient load reduction on continental shelf hypoxia, consider the Black Sea. The northwestern shelf of the Black Sea—not its deep hypoxic basin—is physically prone to hypoxia like the Louisiana shelf. Both are broad and microtidal, receive the discharges of large rivers, and have modest currents driven primarily by winds and buoyancy differences. In contrast to the Louisiana shelf, this shelf was well studied by Soviet and Eastern European scientists from the 1950s. Nutrient concentrations in the lower Danube River began to increase after the 1960s, and because of regular surveys, we can say for sure that large-scale hypoxia first occurred in 1973 (Zaitsev, 1999). The situation suddenly changed after 1989–90, when Eastern European countries embarked on an abrupt transition to market economies and fertilizer use decreased by half. Within 5 yr the total nitrogen discharge of the Danube had been reduced by nearly half, and in 1996, hypoxia was virtually nonexistent for the first summer since 1973 (Mee, 2001).

Rowe and Chapman repeat the assertions of a report prepared for Fertilizer Institute (Carey et al., 1999) that fluvial organic matter delivered by the Mississippi and Atchafalaya rivers could be as important as the plankton production fueled by river-borne nutrients. The integrated assessment addressed this issue and reported, "Scientists reviewed the evidence about the role of [land-derived] organic carbon at a December 1999 meeting and agreed that it is a relatively small factor driving hyp-

oxia—nitrogen-driven carbon production is approximately an order of magnitude greater." Notably, the contributing author of the organic carbon section of the Carey et al. (1999) report joined the consensus. In a book review immediately after the Rowe and Chapman article in this journal, the author (Pennock, 2002) found that the National Research Council report he reviewed presented "a clear argument for the role that nitrogen plays in nutrient over-enrichment of estuarine and coastal waters." Although there are large quantities of organic carbon discharged onto the shelf by the rivers, most of it is refractory or is deposited near the river mouths. A mole of reactive nitrogen, on the other hand, is repeatedly recycled to stimulate the fixation of many more moles of highly labile carbon by marine phytoplankton over a broader area. Furthermore, the delivery of organic carbon by the rivers has decreased during the last half of the 20th century at the same time that nitrate delivery increased threefold.

Another issue raised by Rowe and Chapman is the role of oxidation of reduced metabolic by-products (e.g., sulfide) diffusing from the seabed as a contributing factor in the depletion of dissolved oxygen in bottom waters. Undoubtedly, this oxidation does contribute to depleted oxygen concentrations downdrift. However, what is the source of the organic carbon undergoing anaerobic decomposition in the sediments in the first place? Only near depocenters adjacent to the river mouths is it likely to be fluvial organic matter. Elsewhere, plankton in overlying waters produces most of the readily decomposable organic matter in the sediments. In other words, the initial production of these anaerobically decomposed organics has a lot to do with nitrate loading.

The three-zone concept of Rowe and Chapman regarding controls on hypoxia with distance from the river source includes some elements of reality but misrepresents some important processes. The brown water, first zone, does not extend as far from the river mouths as they depict. Hypoxic conditions seldom overlie areas of high sediment accumulation, where the surface sediments are typically well oxidized and turbidity limits the production of phytoplankton. The green water, second zone, is more extensive and, as the authors suggest, characterized by deposition of bioseston and higher bottom water and sediment respiration. As bottom-water oxygen is depleted, reducing conditions may extend to the sediment surface (commonly observed as black surface sediments of sediments covered with *Beggiatoa*). Nitrification of ammonium leads to oxygen starvation, causing denitrification rates to be reduced because of the lack of nitrate (Childs et al., 2002), resulting in the flux of ammonium, as well as phosphate, back toward the photic zone, where it refuels primary production—a positive feedback intensifying hypoxia (Boesch et al., 2001). In addition, the simple east–west zonation of Rowe and Chapman ignores the role of the coastal boundary layer—the nearshore zone of vertically well-mixed water—in transport, reintroduction of nutrient-rich bottom waters, and maintenance of high primary productivity along the inshore edge of the hypoxic zone.

Rowe and Chapman ask, "Are there alternate remedial actions besides or in addition to lowering nitrate loading?" Specifically, they ask, "Can strategies be devised for returning some fraction of the river water to natural wetlands?" Indeed, this was a major focus of the integrated assessment (Committee on Environment and Natural Resources, 2000). Of course, the principal way that wetlands would help stem hypoxia is by reducing the loading of nitrate and other nutrients to the Gulf by serving as sinks for these nutrients. In an article resulting from one of the technical components of the integrated assessment, Mitsch et al. (2001) estimated that diversion of river flow to help rebuild the wetlands of the Mississippi and Atchafalaya deltas could reduce the nitrogen load to the Gulf by at the most 3–6%. So even with this strategy fully implemented reducing nutrient losses at the source would still be required.

Rowe and Chapman challenge "the commonly accepted view that it is nitrate, and only nitrate, that controls the extent of the hypoxic zone." Although there is compelling evidence and rationale that reducing the delivery of nitrate to the Gulf is essential to reduce the extent of the hypoxic zone, it is equally clear that it is not only nitrate that matters. One should not disregard the delivery of other nutrients, including phosphorus and silica, which have been changing as well, resulting in complex effects on hypoxia and the trophodynamics of the ecosystem (Justić et al., 1995). Nutrient control strategies require integrated approaches to these multiple nutrients. How humans affect the timing and location of the delivery of freshwater to the Gulf also matters a lot. This fact should be taken into account as one contemplates river diversions and changes in flow allocations among distributaries.

Rowe and Chapman attempt to muddy the waters with their nagging questions, but like

the muddied waters of the Mississippi as they enter the Gulf, these questions cease to nag as light penetrates and evidence and reason prevail just like nutrients and phytoplankton.

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