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Continental Shelf Hypoxia: Some Nagging Questions

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COMMENTARY

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CONTINENTAL SHELF HYPOXIA: SOME NAGGING QUESTIONS.—The near-shore bottom waters of large areas of the continental shelf of the northwest Gulf of Mexico (GoM) suffer from hypoxia every summer. This hypoxia (dissolved oxygen < 2 mg liter⁻¹ or 1.4 ml liter⁻¹) results from a combination of vertical stratification of the water column and intensified oxygen use from the waters near the seafloor. Excessive nitrate causes eutrophication in many oceanic habitats, accompanied in some situations by bottom water hypoxia, and it has thus been suggested that reducing nitrate in the Mississippi River would reduce the extent and intensity of hypoxia off Louisiana (Turner and Rabalais, 1994; Diaz and Rosenberg, 1995; Committee on Environment and Natural Resources, 2000; Howarth, 2001; Rabalais et al., 2002a). The idea, however, that nitrate is the sole culprit, whose control will ameliorate hypoxia, is not shared universally. The purpose of this letter is to evaluate the seemingly opposing viewpoints.

Hypoxia is a stressful condition in aquatic ecosystems. Oxygen this low is deleterious to metazoan marine life, in general, and can have disastrous effects on an ecosystem, although organisms in upwelling systems seem to survive short exposures quite well (e.g., Verheye, 1991; Rabalais and Turner, 2001). The size of the region affected in the northwest GoM varies from year to year depending on the volume of freshwater delivered by the Mississippi and Atchafalaya rivers and on the nitrogen content of the effluent. Since 1985 the mean area affected each summer has changed from about 8,000 to 9,000 km² during 1985–92 to about 19,000 km² in 1999, although the increase has not been linear or sustained (Battaglia and Goolsby, 2001; Ferber, 2001). A sudden increase in area in 1993 coincided with a major flood of the Mississippi (Rabalais et al., 1998). State and federal agencies, in association with regional academic institutions, have invested heavily in multidisciplinary programs describing hypoxia in the northwest GoM. Monitoring under the leadership of Nancy Rabalais at the Louisiana Universities Marine Consortium has provided more than a decade of seasonal data that illustrate an obvious relationship among river flow, nitrate loading, stratification of the water column, and hypoxia (Rabalais et al.,

1991, 1994, 1996, 1998). Other programs, such as the Louisiana–Texas Physical Oceanography program (Murray, 1998; support from the Minerals Management Service of the Department of the Interior) and the Nutrient Enhanced Coastal Ocean Productivity program (Rabalais et al., 1999; support from National Oceanic and Atmospheric Administration), have been carried out over the coastal shelf adjacent to the Mississippi delta and provide considerable information on a broad spectrum of hypoxia-related processes.

Mississippi River nutrient loading causes localized phytoplankton blooms south of Terrebonne Bay (90.5°W, Fig. 1), which then sink and decompose. This results in hypoxia (Turner and Rabalais, 1991, 1994; Diaz and Rosenberg, 1995) when combined with physical water-column stratification. The particulate organic material that is produced near the surface sinks into deep water and onto the seafloor sediments, where respiration consumes most if not all of the oxygen dissolved in the seawater. The oxygen at depth cannot be replenished by mixing because of intense stratification of the water column. The stratification is also caused by the river: freshwater pouring into the GoM on the surface is less dense than the seawater below it; it floats on the surface and the difference in densities between the deep salty water and the surface freshwater impedes mixing of the layers. Within a month or so of such conditions, the near-bottom water becomes hypoxic at a very linear rate (Rabalais et al., 1991; Rowe, 2001).

There is concern among marine scientists and coastal zone managers that hypoxia is on the increase worldwide (Diaz and Rosenberg, 1995). The hypoxia on the Louisiana shelf has prompted recommendations from marine ecologists to limit nitrogen loading in the Mississippi River drainage basin, thus hopefully diminishing the eutrophication that produces the hypoxia. This concern has spread to soil scientists who are considering what management practices might best be used to decrease the losses of nitrogen to the drainage basin from agriculture.

We acknowledge that there is convincing evidence that hypoxia frequency, duration, and spatial extent during the last several decades are correlated directly with characteristics of the river plume (see the above references of Rabalais et al.). Surveys since 1985 illustrate

Fig. 1. The Louisiana-Texas coastline in the Gulf of Mexico, showing places mentioned in the text, the region generally affected by hypoxia, and the estimated boundaries of our regions A, B, and C. In the hypoxic zone, light shading indicates regions affected <50% of the time during annual summer surveys, dark shading regions affected >50% of the time (redrawn from Rabalais et al., 1999).

the positive relationship among nitrate concentrations, water volume, and hypoxia. One would infer a simple cause and effect. The eutrophication paradigm has now become so elegantly simple that it is hard not to believe, even though it is acknowledged that other inorganic nutrients and light are also important (Dortch and Whitedge, 1992). Add nitrate to a wide spectrum of marine ecosystems and the surface waters bloom with phytoplankton. The phytoplankton is then eaten by zooplankton grazers. Eutrophication occurs when detrital material (particulate organic matter such as fecal pellets along with dead or dying phytoplankton) sinks into near-bottom water where the commensurate decay (respiration) consumes dissolved oxygen faster than it can be resupplied (Rowe, 2001).

Our concern is that this is an oversimplification. 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? We know that oxygen declines below the pycnocline; we even know the rate pretty well. We suggest, however, that correlation is not the cause. To what degree can we rely on our present understanding of the paradigm? Is fertilization by nitrate the lone culprit? Can we be confident that nitrate loading really intensifies organic loading below the pycnocline? What other processes are we ignoring that may have as much or more effect on bottom water oxygen concentrations? It has been pointed out that there also is extensive loading by fluvial organic matter (Carey et al., 1999) in the form of dissolved compounds and particulate

detritus. Carey et al. (1999) strongly implied that the oxygen demand of this material could be as important as the eutrophication (overproduction of phytoplankton) linked to river-borne nitrate.

First, let us consider the information available that firmly supports the paradigm, as reviewed in Carey et al. (1999). It is well established that nitrate enters the GoM in high concentrations in the freshwater plume of the Mississippi (Bratkovich et al., 1994). Initially, photosynthesis is light limited because of high concentrations of suspended silt and clay. As this sediments out or is diluted by saline Gulf water, the light transmission increases and the phytoplankton bloom (Smith and Hitchcock, 1994; Lohrenz et al., 1997). It is generally acknowledged that primary production in the Gulf is nitrate limited, even adjacent to the Mississippi freshwater plume (Walsh et al., 1989). Just outside the turbid plume, primary production can range from around $1 \text{ g C m}^{-2} \text{ d}^{-1}$ in the fall and winter, when river flow is low and the days are short, to as much as $5 \text{ g C m}^{-2} \text{ d}^{-1}$ in spring and early summer, when river flow is high and the days are long (Lohrenz et al., 1997). A reasonable fraction (mean of ca. $600 \text{ mg C m}^{-2} \text{ d}^{-1}$) of this material sinks out of the surface waters as particulate matter (Redalje et al., 1994), whereas several hundred milligrams of carbon per square meter per day is metabolized by heterotrophic organisms in the water column and at the bottom (Dortch et al., 1994; Rowe, 2001). The rates of respiratory metabolism in the water column combined with those at the bottom can easily account for the observed rate of decline in bot-

tom water oxygen in the area over time (Dortch et al., 1994). Cores of the sediments suggest that gradual changes have occurred within the last half century in step with increases in the use of commercial fertilizers (Nelsen et al., 1994), and organic carbon accumulation in the sediments correlates with an increase in nitrate loading in the water column (Eadie et al., 1994), thus confirming the paradigm, presumably.

Now let us address the weaknesses in this oversimplified scenario. A fundamental requirement, as we see it, is the vertical stratification that is imposed by the freshwater of the river. Hypoxia correlates as well with the strength of the density gradient as it does with nitrate (Wiseman et al., 1997). This intense pycnocline virtually eliminates the vertical exchange of water between the fresh, and hence less dense, surface zone supersaturated in oxygen and the saline deep water. The greater the freshwater flow, the more abrupt the density discontinuity, the greater the area of shelf affected, and the more widespread the hypoxia. Thus, one could equally contend that freshwater is the culprit and not nitrate. Carey et al. (1999) have shown that there has been an apparent increase in the flow of the Mississippi at Vicksburg of about 11% since 1967 when compared with the previous 15-yr mean. Their figure 18 suggests that there was a drop in flow of about 7% around 1870, based on the first 50 yr of the record; thus, long-term variations in flow may be a routine occurrence in the basin.

The freshwater will continue to flow even if it contains considerably less nitrate. There are examples of such stratification-induced hypoxia from elsewhere, and even freshwater input does not seem necessary. For example, the Agulhas Bank, south of Africa, is a wide continental shelf with a mean depth of about 100–120 m. During summer, there is essentially no freshwater input from the few small rivers along the southern coast of South Africa, but hypoxia can develop close to the coast because of the strong stratification induced by solar heating in a region where water movement is very low (Chapman and Shannon, 1987). In this area, the change in temperature across the thermocline can be 12 °C in less than 10 m (Schumann and Beekman, 1984), and it is this density interface that causes the hypoxia and not the phytoplankton production, which, at 20–40 mg C m⁻³ hr⁻¹ (Carter et al., 1987), is similar to rates observed in the Mississippi plume.

Another requirement for hypoxia to develop

is that the water below the pycnocline should be quiescent, with only a slow replacement time. This is the case off the Mississippi; Murray et al. (1998) report that current velocities in the subpycnocline layer are only about 1–3 cm sec⁻¹ in summer. In fact, the physical oceanography of the delta region is primed to become hypoxic because of the way the current flow varies during the year. Most of the year, the general flow is “down coast” (from Louisiana to the west), but during summer it reverses and flows “up coast” (Murray et al., 1998). This causes water flowing west from the Mississippi and Atchafalaya rivers to pile up against the water flowing east from the Texas shelf and provides additional time for respiration processes in both the water and sediment to reduce the oxygen content of the water column.

Although we have a reasonable idea of the rates at which oxygen is consumed during phytoplankton respiration off the Mississippi delta, there are additional mechanisms that lead to the consumption of oxygen that need to be quantified. Microbial respiration of terrestrial particulate (Trefry et al., 1994) and dissolved organic matter (Lopez-Veneroni and Cifuentes, 1994) that is also introduced in high concentrations by the river may be important. There is reason to believe that biogenic terrestrial detritus is substantially more reactive than thought previously (Amon and Benner, 1996; Sun et al., 1997; Mayer et al., 1998). Another probable fate of oxygen is reaction with the reduced end products of anaerobic metabolism (Rowe et al., unpubl. data). This includes sulfide, Fe⁺⁺, Mn⁺⁺, and NH₄⁺ (Morse and Rowe, 1999; Rowe et al., 2002) and dissolved organic matter (Lopez-Veneroni and Cifuentes, 1994). It is generally presumed that in oxic environments the total sediment community oxygen consumption, measured with in situ benthic incubation chambers or aboard ship in core incubations, integrates both aerobic and anaerobic metabolism. This is presumed because the metabolic by-products of anaerobic respiration are oxidized by free oxygen, and this consumption in incubation experiments can thus be used to calculate the rates of anaerobic respiration. However, when the bottom water oxygen is limiting, the oxygen consumption is markedly reduced or eliminated and the end products can diffuse out of the mud into the water column. There they consume any remaining free oxygen available, and any residual reducing agents can be advected westward under the influence of the prevailing circulation into the region south of Terre-

TABLE 1. Sediment community oxygen consumption (SCOC) rates and their equivalent carbon respiration rates at four sites between the Mississippi delta and the Texas coast.

Site	Depth (m)	Temperature (°C)	Longitude (°W)	SCOC (mmol O ₂ m ⁻² d ⁻¹)	Carbon respiration rate (mg C m ⁻² d ⁻¹)
Delta	15	20.0	89.5	22.7	232
	15	28.1	89.5	18.7	191
	21	28.1	89.4	40.1	409
	22	27.2	89.8	56.4	575
Zone 2	19	19.3	90.8	25.7	262
	20	20.0	90.5	60.0	612
	20	28.6	90.5	16.6	169
Flower Gardens Bank	27	20.0	93.8	20.2	206
	28	26.0	93.8	29.9	305
Port Aransas	27	28.8	96.5	27.1	276
	27	28.8	96.5	33.1	338
	27	28.8	96.5	35.0	357

bonne Bay or further west, where they can continue to deplete dissolved oxygen levels.

Is the photosynthesis rate that is attributed to nitrate loading really that high? In a limited spatial sense in close proximity to the plume, it is (Lohrenz et al., 1997), but in terms of the area affected by hypoxia and beyond, we contend that it is not. It is true that, compared with the mean rates in the GoM as a whole (100–200 g C m⁻² yr⁻¹, see Walsh et al., 1989), the river-stimulated rates are high. But compared with many “normal” coastal environments in many areas of the world (Walsh et al., 1989), the plume is relatively modest in both yearly average rates and areal coverage. The GoM’s problem as a whole is not too much nitrate but too little nitrate. The bulk of the nitrate in the Gulf is below the euphotic zone and hence has a minor effect on productivity. Mixing of nitrate up into the euphotic zone during the winter gives rise to a seasonal doubling of plant pigments (Müller-Karger et al., 1991), but this increase is modest when compared with many other large ecosystems.

There is little evidence that oxygen demand on the seafloor or in the water column is exceptionally high below the plume. Few comparisons have been made with other areas of the shelf, including downstream of the plume. In Table 1, we show sediment community oxygen consumption rates for several areas of the shelf. The rates, which include their carbon equivalents, suggest that overall respiration is low and that there is essentially no difference in respiration between the delta and the Texas coast. The data were taken during periods when hypoxia was not occurring. Although the highest individual rate was found in zone 2, analysis of variance shows that these data are

indistinguishable from each other, regardless of longitude. Reducing the data to a common temperature makes little difference to the analysis. During periods of hypoxia (not shown), the respiration rates are even lower, even in zone 2, where they might be expected to be higher if the simple paradigm of hypoxia holds. In fact, oxygen consumption shuts down when oxygen disappears (Morse and Rowe, 1999; Rowe, 2001).

If an increase in oxygen demand cannot be demonstrated after the surface water bloom, maybe the paradigm is oversimplified. Total heterotrophic metabolism, including sulfate reduction (Morse and Rowe, 1999; Rowe et al., unpubl. data), is high when all the common terminal electron acceptors are added up. This may be a function of the rapid accumulation of terrestrially derived material near the delta and not of phytoplankton production. It is true that it has been proposed that anaerobic metabolism may be high and may release reduced metabolic end products into the water column (Rowe et al., 2002), but the organics fueling this mixture of processes may have little to do with nitrate loading. The release of reactive oxygen-consuming compounds may find its cause in a combination of stratification by freshwater, loading by terrestrially derived organics, and rapid accumulation of river-borne clay and silt. Nitrate, we must caution, has not been demonstrated to cause any of these phenomena. Nitrate’s guilt is by implication only.

We are not saying in this comment that nitrate is not a potentially important contributor to the Mississippi River–associated eutrophication and hypoxia. Certainly, we agree that the best management practice in fertilizer appli-

Fig. 2. A revised paradigm describing the controls on hypoxia in the three regions discussed in the text.

cation is the ideal to strive for. We believe that the evidence against nitrate is not conclusive. It is risky to demand rapid remediation by the agricultural industry if it will have little effect on the size and intensity of the hypoxic zone.

It seems clear from the above that there is considerable evidence that nitrate alone is not the sole cause of the hypoxia but that physical and other biogeochemical processes are also important. We, therefore, put forward a new idea that includes all three aspects. This is shown conceptually in Figure 2. Our idea is that there are three zones around the mouth of the Mississippi (and presumably also the Atchafalaya, although the wetlands near the river mouth may strongly affect zones 1 and 2 here). The first (brown) zone, nearest the river mouth, is controlled by the deposition of sedimentary material. Light penetration is low, as is primary production, but most of the particulate organic carbon brought down by the river is deposited here in association with clay flocculation. This leads to high rates of sediment accumulation and metabolism, which is essentially anaerobic and leads, in turn, to the production of ammonium, sulfides, and reduced iron and manganese species. The western boundary of this zone is generally somewhere near 90°W.

The second (green) zone is the region of highest surface water primary production and

occurs immediately offshore—alongshore from zone 1. Here, the light penetration is increased because of the flocculation–deposition in zone 1 and dissolved nitrate and silicate concentrations are high, and this leads to rapid diatom growth. According to the paradigm, these are deposited rapidly. Some deposition may be in the form of zooplankton fecal pellets, particularly at the outer edge of this zone. Initial decomposition is aerobic, but if production continues in the euphotic zone, this can be accompanied by anaerobic decomposition in the sediments, including denitrification, and this could be expected to increase as hypoxia intensifies. This zone probably extends offshore to about 28.5°N and west to about 91.5°W.

The third (blue) zone, which we believe is the largest and most affected by the volume of water delivered by the rivers, is one where stratification plays the most important role. The euphotic zone is nitrogen limited (data show that nitrate concentrations in the upper layer decay very quickly away from the river mouths), and production is controlled by regenerated nitrogen. Respiration at the bottom is essentially aerobic, with little particulate organic carbon deposition and little denitrification. However, the oxygen content of the bottom layer is already reduced because of the prevailing westward flow, and it is possible for the oxygen to become reduced to less than 2 mg liter⁻¹ by

Fig. 3. Ten-year record (1990–99) of flow in the Mississippi River at Tarbert Landing, LA. The thin solid line shows the actual flow, whereas the thicker solid line and the dotted lines show the 70-yr mean flow and the standard deviation about the mean.

the continuing respiration demand. Depending on river discharge and the local wind regimen, this zone can extend as far west as the Texas coast. Although hypoxia is rarely found west of 94°W, oxygen concentrations well below saturation are found as far west as Galveston Bay (Harper et al., 1981; Murray, 1998; Nowlin et al., 1998).

All three zones are affected by river input, but zone 1 will be affected least and zone 3 will be affected most by the changes in freshwater volume. The strength of the pycnocline affects zones 2 and 3, but zone 2 is controlled more by the incoming nitrate and silicate and will therefore vary less than zone 3 as the volume of freshwater impinging on the region varies.

Is there any evidence for this hypothesis? We believe that the sudden increase in the area affected by hypoxia after the 1993 flood demonstrates this point well. The interesting point about the flood was not just the total volume of water delivered by the Mississippi but the fact that the runoff rate was high (relative to the long-term mean) throughout the summer. The 1990s were, in fact, generally years of above-average flow (Fig. 3), and most of these years showed secondary peaks in flow during the June–Aug. period. The peaks not only supplied additional nutrients during the summer, when primary and secondary productions usu-

ally decrease (Qureshi, 1995), but they also served to keep the water column stable and decrease gas exchange. In addition, the large amount of runoff would have served to add much higher amounts of suspended matter than usual, with the likely result that the bottom in both zones 1 and 2 became covered with a layer of sediment that kept the bottom oxygen concentrations at or close to zero for the rest of the summer. Although the water column would be reoxygenated during the winter, the sediment itself would remain anoxic until all the reducing agents in it are used up and would continue to release reducing compounds into the water column as described earlier. Because the 1993 flood was so large when compared with normal flow rates, it might well take several years for the reducing agents associated with it to leach out of the system. This could also contribute to the large region affected by hypoxia during the rest of the 1990s.

High sedimentation rates overload the system's ability to oxidize incoming material. If we are correct, it suggests that even if nitrate runoff is reduced considerably in the upstream region of the watershed, it will be many years before we can see the effects in the coastal waters of Louisiana. Rabalais et al. (2002b) have noted this point and have suggested an adap-

tive management strategy, coupled with a public education outreach program. We endorse this approach.

We have suggested that there are additional mechanisms that cause hypoxia in addition to the commonly accepted view that it is nitrate, and only nitrate, that controls the extent of the hypoxic zone. We strongly support the continued monitoring of river flow volume, nitrate loading, and hypoxia by Rabalais and others, which, if anything, needs to be enhanced. This is important because at present only one survey of the extent of the hypoxic region is carried out each year. We, therefore, have little idea of the rate at which it is generated. In addition, a series of experiments needs to be conducted in the field to test the relative importance of the alternative possibilities that we have listed. The independence of (1) stratification, (2) fluvial organic matter loading, and (3) oxidation of metabolic by-products should be considered in the experimental designs. Such experiments should be carried out in and below the plumes of both the Mississippi and Atchafalaya rivers because it seems that the retention of much of the wetland area adjacent to the mouth of the Atchafalaya may have a considerable effect on the size of zones 1 and 2 here. If this is the case, it may have ramifications for controlling the water flow and hence the size of the hypoxic region.

Are there alternative remedial actions besides or in addition to lowering nitrate loading? Mississippi water has been channelized to ever-increasing degrees since the 1700s (Gunter, 1952; Clay, 1986; Carey et al., 1999). This aids navigation and prevents losses to floods. A companion result is that less and less water returns to adjacent wetlands, swamps, salt marshes, and estuaries, all of which take up inorganic nutrients during photosynthesis, recycle organic matter, and trap lithogenic particulates. Can strategies be devised for returning some fraction of the river water to natural wetlands? Can the water, with its suite of inorganic nutrients, buoyancy, organic matter, and sediment suspension, be put to some alternative use? Such diversions might be costly to shipping, but alternatives might have to be considered if reduction in nitrate turns out to have little or no effect.

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