Spreading Dead Zones and Consequences for Marine Ecosystems

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Dead zones in the coastal oceans have spread exponentially since the 1960s and have serious consequences for ecosystem functioning. The formation of dead zones has been exacerbated by the increase in primary production and consequent worldwide coastal eutrophication fueled by riverine runoff of fertilizers and the burning of fossil fuels. Enhanced primary production results in an accumulation of particulate organic matter, which encourages microbial activity and the consumption of dissolved oxygen in bottom waters. Dead zones have now been reported from more than 400 systems, affecting a total area of more than 245,000 square kilometers, and are probably a key stressor on marine ecosystems.

The visible ecosystem response to eutrophication is the greening of the water column as the algae and vegetation in coastal areas grow in direct response to nutrient enrichment. The most serious threat from eutrophication is the unseen decrease in dissolved oxygen (DO) levels in bottom waters, created as planktonic algae die and add to the flow of organic matter to the seabed to fuel microbial respiration (*1*). Hypoxia occurs when DO falls below ≤2 ml of O_2/liter, at which point benthic fauna show aberrant behavior—for example, abandoning burrows for exposure at the sediment-water interface, culminating in mass mortality when DO declines below 0.5 ml of O_2/liter (*2*). In most cases, hypoxia is associated with a semi-enclosed hydrogeomorphology that, combined with water-column stratification, restricts water exchange. More recently, dead zones have developed in continental seas, such as the Baltic, Kattegat, Black Sea, Gulf of Mexico, and East China Sea, all of which are major fishery areas.

Although the anthropogenic fertilization of marine systems by excess nitrogen has been linked to many ecosystem-level changes, there are natural processes that can lead to nutrient enrichment along continental margins that produce similar ecosystem responses. Coastal upwelling zones associated with the western boundary of continental landmasses are highly productive but are associated with severe hypoxia (<0.5 ml O_2/liter). These oxygen minimum zones (OMZs) occur primarily in the eastern Pacific Ocean, south Atlantic west of Africa, Arabian Sea, and Bay of Bengal, and are persistent oceanic features occurring in the water column at intermediate depths (typically 200 to 1000 m) (*3*). Where they extend to the bottom, the benthic fauna is adapted to DO concentrations as low as 0.1 ml of O_2/liter. This is in stark contrast to the faunal responses seen during recent eutrophication-induced hypoxic events in coastal and estuarine areas where DO concentrations this low led to mass mortality and major changes in community structure (*2*).

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Global Nature of Eutrophication-Induced Hypoxia

The worldwide distribution of coastal oxygen depletion is associated with major population centers and watersheds that deliver large quantities of nutrients (Fig. 1 and table S1). Most of these systems were not hypoxic when first studied, but it appears that from the middle of the past century, the DO concentrations of many coastal ecosystems have been adversely affected by eutrophication. The observed declines in DO have lagged about 10 years behind the increased use of industrially produced nitrogen fertilizer that began in the late 1940s, with explosive growth in the 1960s to 1970s (*4*). For marine systems with data from the first half of the 20th century, declines in oxygen concentrations were first observed in the 1950s in the northern Adriatic Sea (*5*), between the 1940s and 1960s in the northwestern continental shelf of the Black Sea (*6*), and in the 1980s in the Kattegat (*7*). Localized declines of DO levels were noted in the Baltic Sea as early as the 1930s, but it wasn't until the 1960s that hypoxia became widespread (*7*). Localized hypoxia had also been observed since the 1930s in the Chesapeake Bay (*8*) and since the 1970s in the northern Gulf of Mexico (*9*) and many Scandinavian coastal systems (*7*). Paleoindicators (foraminifera ratios and organic and inorganic compounds) show that hypoxia had not been a naturally recurring event in these ecosystems (*10*, *8*). The number of dead zones has approximately doubled each decade since the 1960s (fig. S1 and table S1).

Hypoxia tends to be overlooked until higher-level ecosystem effects are manifested. For example, hypoxia did not become a prominent environmental issue in the Kattegat until several years after hypoxic bottom waters were first reported and fish mortality and the collapse of the Norway lobster fishery attracted attention (*11*). Although hypoxia in the northern Gulf of Mexico has affected benthic communities over the past several decades, there is no clear signal of hypoxia in fishery landings statistics (*9*).

Ecosystem-level change is rarely the result of a single factor, and several forms of stress typically act in concert to cause change. The shallow northwest continental shelf of the Black Sea provides an example of a system stressed by eutrophication-driven hypoxia in combination with other stressors, including overfishing and the introduction of invasive species, all of which led to drastic reductions in demersal fisheries. Nutrient inputs declined in the 1990s, hypoxia disappeared, and ecosystem services recovered; however, nutrient inputs are again rising as agriculture expands and a return to hypoxic conditions may be imminent (*12*). The key to reducing dead zones will be to keep fertilizers on the land and out of the sea. For agricultural systems in general, methods need to be developed that close the nutrient cycle from soil to crop and back to agricultural soil (*13*).

Degrees of Hypoxia

The most common form of eutrophication-induced hypoxia, responsible for about half the known dead zones, generally occurs once per year, in the summer after spring blooms—when the water is warmest and stratification is strongest—and lasts until autumn (table S1). The usual ecosystem response to seasonal oxygen depletion is mortality of benthic organisms followed by some level of recolonization with the return of normal oxygen conditions. Higher-level trophic transfer from the benthos is limited by seasonal hypoxia and can occur only when normal DO conditions prevail (*2*).

Periodic oxygen depletion has been observed in about a quarter of systems reported as hypoxic and may occur more often than seasonally, but this tends to be less severe, lasting from days to weeks. Many smaller systems, such as the York River in the Chesapeake Bay (*2*), are vulnerable to periodic hypoxia because local weather events and spring neap-tidal cycles influence stratification intensity. Diel cycles that influence production and respiration can also cause hypoxia that lasts only hours but has a daily reoccurrence (*14*). The margins of seasonal dead zones may also experience periodic hypoxic events influenced by wind and tides (*15*).

Another 17% of the systems reported as hypoxic experience infrequent episodic oxygen depletion, with less than one event per year, sometimes with years elapsing between events. Episodic oxygen depletion is the first signal that a system has reached a critical point of eutrophication, which, in combination with physical processes that stratify the water column, tips the system into hypoxia. In 1976, a single hypoxic event in the New York Bight that covered about 1000 km^2 caused mass mortality of demersal fishes and benthos and blocked the northward migration of bluefish (*Pomatomus saltatrix*) (*16*). Many systems experience episodic hypoxia be-

fore the onset of seasonal hypoxia, such as in the northern Adriatic, Pomeranian Bay, and the German Bight. Paleoindicators and models from the northern Gulf of Mexico also support this pattern of occurrence.

Because eutrophication increases the volume of organic matter that reaches the sediments, there is a tendency for hypoxia to increase in time and space. In systems prone to persistent stratification, oxygen depletion may also persist. This type of persistent hypoxia accounts for 8% of dead zones, including the Baltic Sea, the largest dead zone in the world, as well as many fjordic systems.

Progression of Hypoxia

Coastal hypoxia seems to follow a predictable pattern of eutrophication first enhancing the deposition of organic matter, which in turn promotes microbial growth and respiration and produces a greater demand for oxygen. DO levels become depleted if the water column stratifies. In the second phase, hypoxia occurs transiently, accompanied by mass mortalities of benthic animals. With time and further buildup of nutrients and organic matter in the sediments, a third phase is initiated, and hypoxia becomes seasonal or periodic, characterized by boom-and-bust cycles of animal populations. If hypoxia persists for years and organic matter and nutrients accumulate in the sediments, a fourth phase is entered, during which the hypoxic zone expands, and as the concentration of DO continues to fall, anoxia is established and microbially generated H_2S is released. This type of threshold response has been documented in the Gulf of Mexico (*17*), Chesapeake Bay (*8*), and Danish waters (*18*).

The critical point in the response trajectory of an ecosystem to eutrophication is the appearance of severe seasonal hypoxia. Although some level of nutrient enrichment is a positive force in enhancing an ecosystem's secondary productivity and, to a point, fishery yields (*19*), eutrophication and seasonal hypoxia favor only benthic species with opportunistic life histories, shorter life spans, and smaller body sizes (*2*).

Ecosystem Responses

The effect of seasonal hypoxia on biomass and annual secondary production is well documented (*2*, *9*). What is not well understood is how hypoxia affects the habitat requirements of different species or the resilience of an ecosystem. Pelagic species will experience habitat compression when hypoxia makes deeper, cooler water unavailable in the summer (*15*) or overlaps with nursery habitat (*9*). For example, the spawning success of cod in the central Baltic is hindered by hypoxic water at the halocline (70 to 80 m), the depth where salinity is high enough to provide buoyancy for cod eggs (*20*). Similar habitat compression occurs when sulphide is generated in sediments. In this case, as the redox potential discontinuity layer is compressed close to the sediment-water interface, deeper-dwelling species, including the key bioturbators that control pore-water chemistry (*21*), are eliminated. The presence of Fe^{3+} and Mn^{4+} in the sediment may buffer the system and reduce the formation of poisonous H_2S. Reduced bioturbation associated with hypoxia also alters sedimentary habitats by disrupting nitrification and denitrification. Hence, under hypoxic conditions, instead of nitrogen being removed as N_2 by denitrification, ammonia and ammonium together with phosphorus are the main fluxes out of reduced sediments (*8*, *22*) and may stimulate further primary production.

Habitat compression and the loss of fauna as a result of hypoxia have profound effects on ecosystem energetics and function as organisms die and are decomposed by microbes. Ecosystem models for the Neuse River estuary (*23*), Chesapeake Bay (*24*), and Kattegat (*25*) all show hypoxia-enhanced diversion of energy flows into microbial pathways to the detriment of higher trophic levels (Fig. 2). Only under certain circumstances will demersal fish predators be able to consume stressed benthic prey, because their tolerance to low oxygen concentration tends to be less (~3 to 4 ml of O_2/liter) than that of the benthic fauna. Thus, it is only within a narrow range of conditions that hypoxia facilitates upward trophic transfer. As the benthos die, microbial pathways quickly dominate energy flows. Ecologically important places, such as nursery and recruitment areas, suffer most from energy diversion into microbial pathways because hypoxia tends to occur in summer, when growth and predator energy demands are high.

Missing Biomass

Areas within ecosystems exposed to long periods of hypoxia have low annual secondary production and typically no benthic fauna. Estimates of the missing biomass in Baltic dead zones that are now persistently hypoxic are ~264,000 metric tons of carbon (MT C) annually (*7*) and represent ~30% of total Baltic secondary production (*26*). Similarly, estimates for the Chesapeake Bay indicate that ~10,000 MT C is lost because of hypoxia each year, representing ~5% of the Bay's total secondary production (*27*). If we estimate that ~40% of benthic energy should be passed up the food chain in the Baltic (*28*) and 60% in the Chesapeake Bay (*26*), when hypoxic conditions prevail, 106,000 MT C of potential food energy for fisheries is lost in the Baltic and 6,000 MT C in

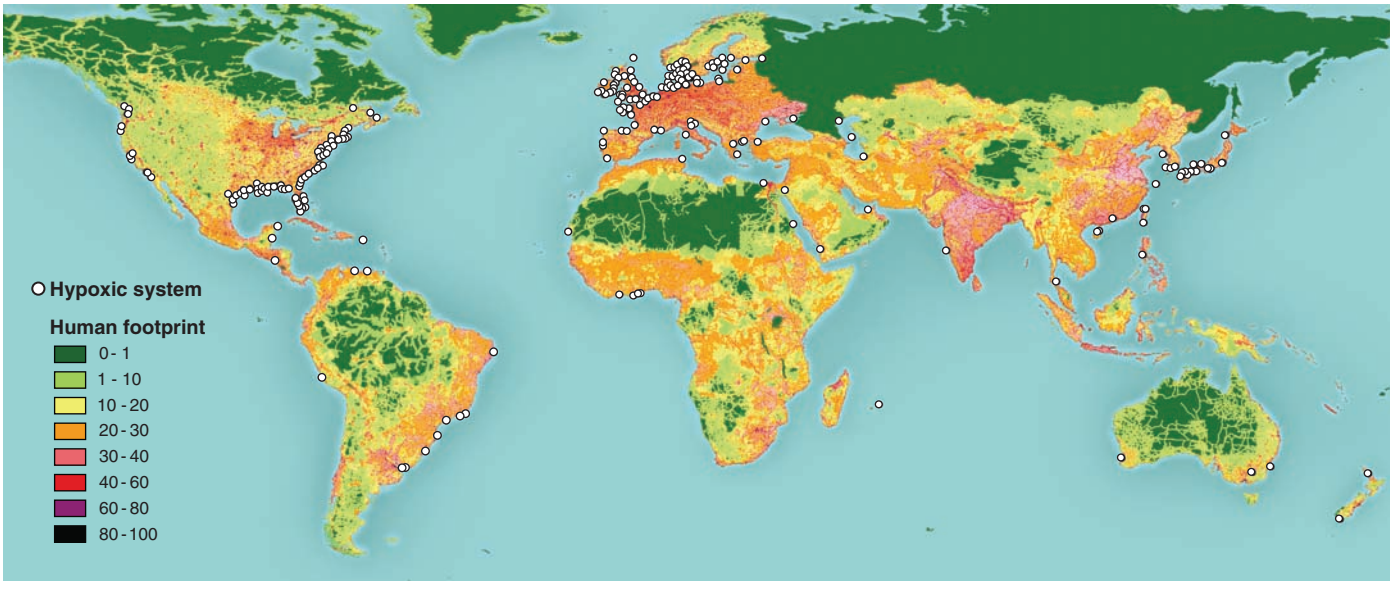

Fig. 1. Global distribution of 400-plus systems that have scientifically reported accounts of being eutrophication-associated dead zones. Their distribution matches the global human footprint [the normalized human influence is expressed as a percent (*41*)] in the Northern Hemisphere. For the Southern Hemisphere, the occurrence of dead zones is only recently being reported. Details on each system are in tables S1 and S2.

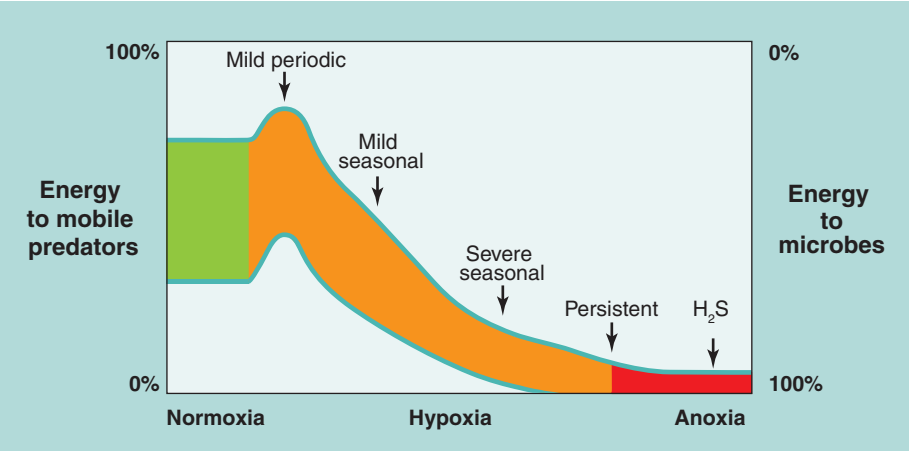

Fig. 2. Conceptual view of how hypoxia alters ecosystem energy flow. The green area indicates the range of energy transferred from the benthos to higher-level predators under normoxia, typically 25 to 75% of macrobenthic carbon. As a system experiences mild or periodic hypoxia, there can be a pulse of benthic energy to predators. This "windfall" is typically short-lived and does not always occur. With declining oxygen, higher-level predation is suspended, benthic predation may continue, and the proportion of benthic energy transferred to microbes rapidly increases (orange). Under persistent hypoxia, some energy is still processed by tolerant benthos. Microbes process all benthic energy as hydrogen sulphide, and anoxia develops (red).

the Chesapeake Bay, respectively. In areas of the Gulf of Mexico that experience severe seasonal hypoxia, benthic biomass is reduced by as much as 1.4 MT C/km^2 (*9*); assuming a 60% transfer efficiency, this is equivalent to approximately 17,000 MT C of lost prey to demersal fisheries.

Is the production lost during periodic hypoxia made up by the ecosystem during normal conditions, or partly compensated for by higher secondary production outside the dead zone? The latter seems to occur in the Baltic, where secondary production outside the dead zones has doubled as a result of eutrophication (*26*); but if the dead zones were eliminated, the Baltic would be more productive by at least a third to a half, assuming that organic matter was processed through benthos instead of by microbes. In Chesapeake Bay, because hypoxia dissipates after about 3 months, the entire area affected is returned to production by recruitment (*27*). Aerial estimates of missing biomass for about a third of the world's dead zones (table S1) indicate that as much as 343,000 to 734,000 MT C is displaced over a total area of 245,000 km^2 as a result of hypoxia.

The duration of seasonal hypoxia then becomes the primary factor affecting ecosystem energy flows. Within most systems that have strong seasonal cycles, increases in populations are related to recruitment events timed to take advantage of the input of new organic matter, usually a spring or autumn bloom; populations normally decline from a combination of resource depletion and predation (*29*). Thus, the shorter the interval between recruitment and the onset of hypoxia, the greater the negative effect on the upward flow of energy in the food chain. During persistent hypoxia, there is a drastic reduction in secondary production, and microbes remineralize virtually all organic matter.

Recovery

By the end of the 20th century, oxygen depletion of marine systems had become a major worldwide environmental problem, with only a small fraction (4%) of the 400-plus systems that had developed hypoxia exhibiting any signs of improvement (table S1). These improvements in DO were related to reductions in three factors: organic and nutrient loadings, stratification strength, and freshwater runoff.

From 1973 to 1990, the hypoxic zone on the northwestern continental shelf of the Black Sea had expanded to 40,000 km^2; however, since 1989, the loss of fertilizer subsidies from the former Soviet Union reduced nutrient loading by a factor of 2 to 4, with the result that, by 1995, the hypoxic zone had gone (*12*). As oxygen levels normalized, ecosystem function improved, and the benthic fauna started to recolonize but have not recovered to prehypoxic levels. In the Gulf of Finland, a decrease in water-column stratification occurred between 1987 and 1994, which improved DO conditions and facilitated the return of benthic fauna (*7*); however, with the return of stratification, conditions have again deteriorated.

In the northern Gulf of Mexico, the occurrence and extent of the dead zone are tightly coupled with freshwater discharge from the Mississippi River, which delivers large quantities of nutrients from U.S. agricultural activities. During years with low river flow, the area of hypoxia shrinks to <5000 km^2, only to increase to >15,000 km^2 when river flow is high (*30*).

The management of nutrients and carbon inputs has virtually eliminated dead zones from several systems, including the Hudson and East Rivers in the United States and the Mersey and Thames Estuaries in England (*31*, *32*). However, in other systems, such as the Chesapeake Bay, the management of nutrient input has not improved DO. Nevertheless, the management of sewage and pulp mill effluents has led to many small-scale reversals in hypoxia (table S1).

The key factors in determining the degree of ecosystem degradation are the duration of exposure and DO concentration. It may take years to recover from severe hypoxia and, moreover, the tolerance to oxygen depletion of mature community species may not mirror that of the opportunistic species that are the first colonizers. The benthos of many coastal areas may be reestablished by larval recruitment and succession, as described in the Pearson-Rosenberg model (*33*); however, the pattern of species that establish during recovery will not be the same as the pattern of species loss during DO deterioration, and consequently a hysteresis-like response will be observed (Fig. 3). A pronounced hysteresis-like response was doc-

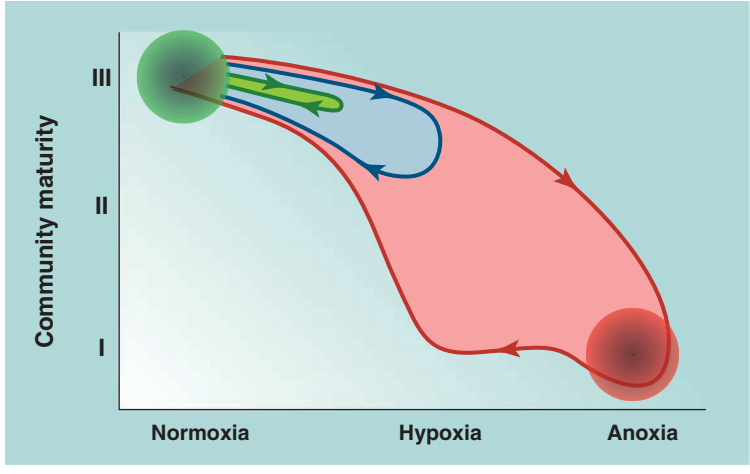

Fig. 3. Generalized pattern of benthic community response to hypoxia (*34*). As DO declines to <0.7 ml of O_2/liter and extends through time, mass mortality of both equilibrium (stage III) and opportunistic (stage I) species occurs (red). If anoxia is reached, benthos are eliminated. The recovery path from severe hypoxia is different than the decline path because of the hysteresis-like progression of successional dynamics. When exposed to mild hypoxia, mortality is moderate, and the recovery path is closer to the response path (blue) as fauna restart from midsuccessional stage II. When exposed to intermediate oxygen conditions, the response is minor (green) and not hysteresis-like.

umented in Gullmarsfjord, Sweden, which suffered hypoxia for about half a year, during which time the fauna was eliminated in deeper areas and diversity and abundance were reduced to less than one-third at medium depths (*34*). Within 2 years, the benthic community had recovered to the same community composition and density that had existed before the hypoxic event (Fig. 3). In this fjord, sedimentary redox conditions had not become intensely reducing, and rapid colonization occurred by larvae from benthic communities in adjacent undisturbed areas. Should hypoxia prevail for more than 5 years, recovery would be prolonged (*35*) and the hysteresis-like response exaggerated, as was recently observed in the Black Sea, where recovery of the benthos after hypoxia in 1994 was still not complete in 2004 (*36*).

Prospects for Change

Further expansion of dead zones will depend on how climate change affects water-column stratification and how nutrient runoff affects organic-matter production. General circulation models predict that climate change alone will deplete oceanic oxygen by increasing stratification and warming as well as by causing large changes in rainfall patterns (*37*), enhancing discharges of fresh water and agricultural nutrients to coastal ecosystems. For example, climate predictions for the Mississippi River basin indicate a 20% increase in river discharge, which will elevate nutrient loading and lead to a 50% increase in primary production, a 30 to 60% decrease in subpycnocline DO, and an expansion of the oxygen-depleted area (*38*). Conversely, if the climate becomes stormier and stratification decreases because of increased mixing, the risk of oxygen depletion will decline. Tropical storms and hurricanes influence the duration, distribution, and size of the Gulf of Mexico dead zone in a complex way. In 2005, four hurricanes (Cindy, Dennis, Katrina, and Rita) disrupted stratification and aerated bottom waters. After the first two storms, stratification was reestablished and hypoxia reoccurred, but the total area was a fourth less than predicted from spring nitrogen flux. The other two hurricanes occurred later in the season and dissipated hypoxia for the year (*30*).

Climate change also has the potential to expand naturally occurring OMZs into shallower coastal waters (*3*), damaging fisheries and affecting energy flows in the same way that eutrophication-driven hypoxia does. There is currently about 1,148,000 km^2 of seabed covered by OMZs (<0.5 ml of O_2/liter), and a small change in oceanographic processes could lead to a major expansion of these zones. Areas at greatest risk for expanding OMZs encompass the western continental shelves of South America, Africa, and the Indian subcontinent, where extensive OMZ and upwelling areas already exist. The development of dead zones along the western coast of other countries is highly likely if wind patterns shift and cause stronger upwelling. This effect might explain the recent development of a dead zone off the coast of Oregon (*39*). Furthermore, there is a possibility that increased loadings of terrestrial nutrients have contributed to an expansion of the OMZ on the western Indian continental shelf (*40*).

The weight of evidence indicates that in preindustrialized times, most coastal and offshore ecosystems never became hypoxic except in natural upwellings. However, measuring the effects of hypoxia on ecosystems is complicated by many factors, not least of which is the inadequate data on historic trends in DO concentrations and faunal populations, as well as the combined effects of multiple stressors, including fishing and habitat loss. It is the recurring nature of hypoxia that alters an ecosystem's state and prevents full recovery of function.

Currently, hypoxia and anoxia are among the most widespread deleterious anthropogenic influences on estuarine and marine environments, and now rank with overfishing, habitat loss, and harmful algal blooms as major global environmental problems. There is no other variable of such ecological importance to coastal marine ecosystems that has changed so drastically over such a short time as DO. We believe it would be unrealistic to return to preindustrial levels of nutrient input, but an appropriate management goal would be to reduce nutrient inputs to levels that occurred in the middle of the past century, before eutrophication began to spread dead zones globally.

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Supporting Online Material

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Fig. S1
Tables S1 and S2
References

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