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Marine Dead Zones: Understanding the Problem

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Summary

An adequate level of dissolved oxygen is necessary to support most forms of aquatic life. While very low levels of dissolved oxygen (hypoxia) can be natural, especially in deep ocean basins and fjords, hypoxia in coastal waters is mostly the result of human activities that have modified landscapes or increased nutrients entering these waters. Hypoxic areas are more widespread during the summer, when algal blooms stimulated by spring runoff decompose to diminish oxygen. Such hypoxic areas may drive out or kill animal life, and usually dissipate by winter. In many places where hypoxia has occurred previously, it is now more severe and longer lasting; in others where hypoxia did not exist historically, it now does, and these areas are becoming more prevalent.

The largest hypoxic area affecting the United States is in the northern Gulf of Mexico near the mouth of the Mississippi River, but there are others as well. Most U.S. coastal estuaries and many developed nearshore areas suffer from varying degrees of hypoxia, causing various environmental damages. Research has been conducted to better identify the human activities that affect the intensity and duration of, as well as the area affected by, hypoxic events, and to begin formulating control strategies.

Near the end of the 105th Congress, the Harmful Algal Bloom and Hypoxia Research and Control Act of 1998 was signed into law as Title VI of P.L. 105-383. Provisions of this act authorize appropriations through NOAA for research, monitoring, education, and management activities to prevent, reduce, and control hypoxia. Under this legislation, an integrated Gulf of Mexico hypoxia assessment was completed in the late 1990s. In 2004, Title I of P.L. 108-456, the Harmful Algal Bloom and Hypoxia Amendments Act of 2004, expanded this authority and reauthorized appropriations through FY2008.

As knowledge and understanding have increased concerning the possible impacts of hypoxia, congressional interest in monitoring and addressing the problem has grown. The issue of hypoxia is seen as a search for (1) increased scientific knowledge and understanding of the phenomenon, as well as (2) cost-effective actions that might diminish the size of hypoxic areas by changing practices that promote their growth and development. This report presents an overview of the causes of hypoxia, the U.S. areas of most concern, federal legislation, and relevant federal research programs. This report will be updated as circumstances warrant.

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Introduction and Background

Hypoxia refers to a depressed concentration of dissolved oxygen in water. While definitions vary somewhat by region, it is generally agreed that hypoxia in a marine environment occurs seasonally when dissolved oxygen levels fall below 2-3 milligrams per liter. Normal dissolved oxygen concentrations in nearshore marine waters range between 5 and 8 milligrams per liter, and many fish species begin having respiratory difficulties at concentrations below 5 milligrams per liter. In extremely low oxygen environments, less tolerant marine animals cannot survive and either leave the area or die.[1] Mortality is especially likely for sedentary species. In addition, spawning areas and other essential habitat can be destroyed by the lack of oxygen. If these conditions persist, a so-called "dead zone" may develop in which little marine life exists.[2] The recovery of marine ecosystems following a hypoxic event has not been extensively studied.

Decreased concentrations of dissolved oxygen result in part from natural eutrophication when nutrients (e.g., nitrogen and phosphorus) and sunlight stimulate algal growth (e.g., algae, seaweed, and phytoplankton), increasing the amount of organic matter in an aquatic ecosystem over decades and centuries. As organisms die and sink to the bottom, they are consumed (decomposed) by oxygen-dependent bacteria, depleting the water of oxygen. When this eutrophication is extensive and persistent, bottom waters may become hypoxic, or even anoxic (no dissolved oxygen), while surface waters can be completely normal and full of life. Hypoxia is more likely to occur in coastal waters where the water column is stratified (i.e., layered) because of differences in temperature or salinity or both. Marine dead zones become most noticeable when and where natural eutrophication has been accelerated by human-influenced increases in nutrient loads.

Hypoxia often develops as a result of upwelled ocean waters, particularly along the western coast of the Americas. In many instances, cool, nutrient-rich, deep marine waters rise along the coastal margin and support massive algal blooms that lead to hypoxia. In other instances, upwelled deep water is simply devoid of oxygen. Eutrophication as a result of human activities usually results from non-point sources

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[1] Nancy N. Rabalais and R. E. Turner, eds., *Coastal Hypoxia: Consequences for Living Resources and Ecosystems*, Coastal and Estuarine Studies 58 (Washington, DC: American Geophysical Union, 2001), 454 pp.; Lisa A. Levin, "Oxygen Minimum Zone Benthos: Adaptation and Community Response to Hypoxia," *Oceanography and Marine Biology: An Annual Review*, v. 41 (2003): 1-45.

[2] Some molluscs and annelid worms are more tolerant of low oxygen conditions and can survive hypoxic episodes that last many weeks.

of nutrients (e.g., runoff from lawns and various agricultural activities including fertilizer use and livestock feedlots), point-source discharge from sewage plants, and emissions from vehicles, power plants, and other industrial sources.

Hypoxic zones frequently occur in coastal areas where rivers enter the ocean (e.g., estuaries). Rivers deliver fresh waters that are rich in nutrients to the saltier estuaries and coastal oceans. The fresh water is less dense than the salt water and typically flows across the top of the sea water. The fresh surface water effectively caps the more dense, saline bottom waters, retarding mixing, creating a two-layer system, and promoting hypoxia development in the lower, more saline waters. In the northern Gulf of Mexico, the greatest algal growth in surface waters occurs about a month after maximum river discharge, with hypoxic bottom water developing a month later.[3] Hypoxia is more likely to occur in estuaries with high nutrient loading and low flushing (i.e., low freshwater turnover).[4] Human activities that increase nutrient loading can increase the intensity, spatial extent, and duration of hypoxic events. Storms and tides may mix the hypoxic bottom water and the aerated surface water, dissipating the hypoxia.

Although the extent of effects of hypoxic events on U.S. coastal ecosystems is still uncertain, the phenomenon is of increasing concern in coastal areas. Several federal agencies are involved in analyzing the problem, including the U.S. Geological Survey (USGS), the National Oceanic and Atmospheric Administration (NOAA), and the U.S. Environmental Protection Agency (EPA). Legislation was enacted by the 105th Congress to provide additional authority and funding for research and monitoring to address these concerns. This authority was extended by the 108th Congress.

Hypoxic Areas

Hypoxic episodes have been recorded in all parts of the world, notably in partially enclosed seas and basins where vertical mixing is minimal, such as the Gulf of Mexico, Chesapeake Bay, the New York Bight, the Baltic Sea, and the Adriatic Sea. In March 2004, the U.N. Environment Program's (UNEP's) *Global Environment Outlook (GEO) Year Book 2003* reported 146 dead zones where marine life could not be supported due to depleted oxygen levels.[5] In 2006, UNEP's Global Programme of Action for the Protection of the Marine Environment from Land-based Activities reported that the frequency and intensity of coastal dead zones is rapidly

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[3] D. Justic et al., "Seasonal Coupling Between Riverborne Nutrients, Net Productivity and Hypoxia," *Marine Pollution Bulletin*, v. 26 (1993): 184-189.

[4] R. Turner and N. Rabalais, "Suspended Particulate and Dissolved Nutrient Loadings to Gulf of Mexico Estuaries," in *Biogeochemistry of Gulf of Mexico Estuaries*, T. Bianchi, J. Pennock, and R. Twilley, eds. (New York, NY: John Wiley & Sons, 1999), pp. 89-107.

[5] R. J. Diaz, J. Nestlerode, and M. L. Diaz, "A Global Perspective on the Effects of Eutrophication and Hypoxia on Aquatic Biota" in G. L. Rupp and M. D. White, eds., *Proceedings of the 7th International Symposium on Fish Physiology, Toxicology, and Water Quality*, Tallinn, Estonia, May 12-15, 2003, EPA 600/R-04/049 (Athens, GA: U.S. Environmental Protection Agency, Ecosystems Research Division, 2004); see Box 4 at [http://www.unep.org/geo/yearbook/yb2003/089.htm].

increasing and could reach 200 sites when a full list of newly identified sites is released in early 2007.[6] Hypoxia has become more frequent and widespread in shallow coastal and estuarine areas.[7] In addition, permanently hypoxic water masses (i.e., oxygen minimum zones) occur in the open ocean, affecting large seafloor surface areas along the continental margins of the eastern Pacific, Indian, and western Atlantic Oceans.[8]

About 21% to 43% of the area of the United States' estuaries have experienced a hypoxic event; more than half of the affected area is in the Mississippi/Atchafalaya River plume.[9] In the Mid-Atlantic region, 13 of 22 estuaries have experienced hypoxic/anoxic events.[10] Of these, the Long Island Sound, Chesapeake Bay, Choptank River, and the New York Bight experience the most serious annual episodes. In the South Atlantic region, hypoxic/anoxic episodes are generally brief,[11] with nearly two-thirds of this region's 21 estuaries experiencing some hypoxia/anoxia.[12] The Gulf of Mexico region experiences the highest rate of hypoxic/anoxic events, with almost 85% of this region's 38 estuaries experiencing episodes of hypoxia (including the Mississippi/Atchafalaya River plume).[13] The North Atlantic region is not as prone to hypoxic/anoxic events due to the generally low nutrient input (the result of lower population density) and high tidal flushing. However, areas adjacent to high population density (e.g., Cape Cod Bay and Massachusetts Bay) do experience oxygen depletion. In the Pacific region, hypoxia also occurs near population centers (e.g., San Diego Bay, Newport Bay, Alamitos Bay) or in areas of limited circulation, even where water temperatures are cold (e.g., Hood Canal, Whidbey basin/Skagit Bay).[14]

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[6] See UNEP press release, available at [http://www.unep.org/Documents.Multilingual/Default.asp?DocumentID=486&ArticleID=5393&l=en].

[7] R. J. Diaz and R. Rosenberg, "Marine Benthic Hypoxia: A Review of Its Ecological Effects and the Behavioral Responses of Benthic Macrofauna," *Oceanography and Marine Biology: An Annual Review*, v. 33 (1995): 245-303. (Hereafter referred to as "Marine Benthic Hypoxia.")

[8] John J. Helly and Lisa A. Levin, "Global Distribution of Naturally Occurring Marine Hypoxia on Continental Margins," *Deep-Sea Research, Part I*, v. 51 (2004): 1159-1168.

[9] N. Rabalais, "Oxygen Depletion in Coastal Waters." *NOAA's State of the Coast Report.* (Silver Spring, MD: NOAA: 1998), National Picture, p. 4, [http://oceanservice.noaa.gov/websites/retiredsites/sotc_pdf/HYP.PDF]. (Hereafter referred to as "Oxygen Depletion in Coastal Waters.")

[10] S. Bricker, "NOAA's National Estuarine Eutrophication Survey: Selected Results for the Mid-Atlantic, South Atlantic and Gulf of Mexico Regions," *Estuarine Research Federation Newsletter*, v. 23, no. 1 (1997): 20-21. (Hereafter referred to as "NOAA's Estuarine Eutrophication Survey: Selected Results."); NOAA's Estuarine Eutrophication Survey, vol. 1: South Atlantic Region (Silver Spring, MD: National Ocean Service, Office of Ocean Resources Conservation and Assessment, 1996), p. 50.

[11] "Oxygen Depletion in Coastal Waters," Regional Contrasts, p. 2.

[12] "NOAA's Estuarine Eutrophication Survey: Selected Results," pp. 20-21.

[13] Ibid., pp. 20-21.

[14] "Oxygen Depletion in Coastal Waters," National Picture, p. 5.

Gulf of Mexico Dead Zone

The hypoxic zone in the northern Gulf of Mexico is the largest observed in the estuarine and coastal regions of the western hemisphere.[15] First recognized in the early 1970s, it is the largest and most hypoxic area in the United States. In summer 1993, following massive Mississippi River flooding, the dead zone covered more than 18,000 square kilometers (an area as large as the state of New Jersey); it reached its largest size in summer 2002 — 22,000 square kilometers (an area as large as the state of Massachusetts).[16] The area of hypoxia extends westward from the mouth of the Mississippi River to the upper Texas coast.[17] The seasonal shape and extent of the dead zone are mostly a function of the Mississippi/Atchafalaya River plume, the combined outflow from these two major rivers, and the biological processes it influences. The most reliable predictor of the size of the hypoxic zone is the nitrate-nitrogen load in the two months before the mid-summer mapping cruise.[18] This hypoxic zone generally occurs from May to September, but varies from year to year. After reaching a maximum size of 20,000 square kilometers in 1999, the dead zone covered a much smaller 4,400 square kilometers in 2000. Low velocity winds during the summer result in calm seas that maintain the stratified barrier between surface and bottom water layers. Only during weather disturbances, such as frontal passages, tropical storms, and hurricanes, does vertical mixing of these stratified layers occur. Increased winds and frontal storms in autumn vertically mix the water column, dissipating the hypoxia.[19] In the summer of 1998, this dead zone extended from very near shore (about 10-15 feet water depth) to deeper waters than are normally hypoxic (as much as 160 feet deep off the Mississippi River delta).[20]

Nutrient enrichment is the primary cause of eutrophication, of some algal blooms, and of hypoxia, and is believed to be a major factor in areas such as the northern Gulf of Mexico.[21] The Mississippi watershed drains 41% of the land area of the contiguous 48 states, including most of the farmbelt. Studies of the

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[15] Nancy N. Rabalais, R. E. Turner, and D. Scavia, "Beyond Science into Policy: Gulf of Mexico Hypoxia and the Mississippi River," *BioScience*, v. 52 (2002): 129-142.

[16] Nancy N. Rabalais, R. E. Turner, and W. J. Wiseman, Jr., "Hypoxia in the Gulf of Mexico, a.k.a. 'The Dead Zone,'" *Annual Review of Ecology and Systematics*, v. 33 (2002): 235-263. (Hereafter referred to as "Hypoxia, a.k.a. 'The Dead Zone.'")

[17] White House Office of Science and Technology Policy, Committee on Environment and Natural Resources, Hypoxia Work Group, *Gulf of Mexico Hypoxia Assessment Plan* (March 1998), p. 3. (Hereafter referred to as Hypoxia Work Group.)

[18] R. E. Turner, Nancy N. Rabalais, and D. Justic, "Predicting Summer Hypoxia in the Northern Gulf of Mexico: Riverine N, P and Si Loading," *Marine Pollution Bulletin*, v. 52 (2006): 139-148.

[19] In August-September 2005, the winds from Hurricanes Katrina and Rita likely promoted a somewhat earlier dissipation of the dead zone off the mouth of the Mississippi River, although Hurricane Cindy in early July 2005 resulted in a smaller mid-summer hypoxic zone than expected that summer.

[20] "Hypoxia, a.k.a. 'The Dead Zone.'"

[21] Hypoxia Work Group, p. 2.

Mississippi and Atchafalaya Rivers indicate that dissolved nitrogen levels have tripled and phosphorus levels have doubled since 1960, fueling algal growth and the resultant dead zone.[22]

Research suggests that fertilizer leaching and runoff from upriver agricultural sources may be the main sources of nutrients. For example, USGS states that 56% of the Mississippi River's nutrient loading results from fertilizer runoff, with an additional 25% of the Mississippi River nitrogen coming from animal manure (municipal and solid wastes account for 6%, atmospheric deposition for 4%, and unknown sources for 9%).[23] Analysis of cores of sediments underlying the hypoxic area reveals historic information on the Mississippi River watershed, indicating that surface water productivity has increased and bottom water oxygen stress has worsened since the early 1900s, with the most dramatic changes occurring since the 1950s — a change strongly correlated with increased use of commercial fertilizers in the watershed.[24] Hypoxia in the northern Gulf of Mexico was not an obvious or widespread phenomenon prior to the early 1970s,[25] but became so since then with the advent of heavy use of artificial fertilizers and changed agricultural practices. Several studies show a direct relationship between river-born nutrients, the high rates

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[22] R. E. Turner and N. Rabalais, "Changes in Mississippi River Quality This Century — Implications for Coastal Food Webs," *BioScience*, v. 41, no. 3 (1991): 140-147; R. E. Turner and Nancy N. Rabalais, "Suspended Sediment, C, N, P, and Si Yields from the Mississippi River Basin," *Hydrobiologia*, v. 511 (2004): 79-89; R. E. Turner, et al., "Future Aquatic Nutrient Limitations," *Marine Pollution Bulletin*, v. 46 (2003): 1032-1034.

[23] R. H. Meade, ed., *Contaminants in the Mississippi River, 1987-92*, Circular 1133 (Denver, CO: U.S. Geological Survey, 1995), 140 pp.; D. A. Goolsby et al., *Flux and Sources of Nutrients in the Mississippi-Atchafalaya River Basin*, Topic 3 Report for the Integrated Assessment of Hypoxia in the Gulf of Mexico, NOAA Coastal Ocean Program Decision Analysis Series No. 17 (Silver Spring, MD: NOAA Coastal Ocean Program, 1999).

[24] T. A. Nelson et al., "Time-Based Correlation of Biogenic, Lithogenic and Authigenic Sediment Components with Anthropogenic Inputs in the Gulf of Mexico NECOP Study Area," *Estuaries*, v. 17 (Dec. 1994): 873; B. J. Eadie et al., "Records of Nutrient-Enhanced Coastal Productivity in Sediments from the Louisiana Continental Shelf." *Estuaries*, v. 17 (Dec. 1994): 754-765; N. Rabalais et al., "Nutrient Changes in the Mississippi River and System Responses on the Adjacent Continental Shelf," *Estuaries*, v. 19 (1996): 386-407; S. Gupta et al., "Seasonal Oxygen Depletion in Continental-Shelf Waters of Louisiana: Historical Record of Benthic Foraminifers," *Geology*, v. 24 (1996): 227-230; and R. E. Turner and N. Rabalais, "Coastal Eutrophication Near the Mississippi River Delta," *Nature*, v. 368 (1994): 619-621.

[25] N. Chen et al., "Historical Trends of Hypoxia on the Louisiana Shelf: Applications of Pigments as Biomarkers," *Organic Geochemistry*, v. 32 (2001): 543-561; D. Justic, N. N. Rabalais, and R. E. Turner, Modeling the Impacts of Decadal Changes in Riverine Nutrient Fluxes on Coastal Eutrophication Near the Mississippi River Delta," *Ecological Modeling*, v. 152 (2002): 33-46; L. E. Osterman, "Benthic Foraminifers from the Continental Shelf and Slope of the Gulf of Mexico: An Indicator of Shelf Hypoxia," Estuarine, *Coastal and Shelf Science*, v. 58 (2003): 17-35; and N. N. Rabalais et al., "Ecosystem History of Mississippi River-Influenced Continental Shelf Revealed Through Preserved Phytoplankton Pigments,: *Marine Pollution Bulletin*, v. 49 (2004): 537-547.

of phytoplankton production, and subsequent Gulf of Mexico hypoxia.[26] However, questions remain as to how much of the river's nitrogen might come from natural soil mineralization, what effects floods have on nutrient transport, and how much nitrogen may be contributed by coastal land loss, estimated at 25 square miles per year.[27]

Although studies have found that more than 70% of the total nitrogen transported to the Gulf of Mexico by the Mississippi River originates above the confluence of the Ohio and Mississippi Rivers,[28] focusing on nitrogen runoff per unit area identifies other areas where more concentrated nutrient runoff occurs.[29] Although the lower Mississippi basin (which drains parts of Tennessee, Arkansas, Missouri, Mississippi, and Louisiana) is responsible for only 23% of the nitrogen delivered to the Gulf, some scientists believe that nitrogen removal and/or runoff prevention strategies should focus on this area because of its much greater relative nitrogen contribution[30] and likely more economically efficient nitrogen removal. Researchers estimate that the benefits of nutrient controls in this lower basin could be twice as effective as implementing them in upstream basins.[31] Others dispute that approach, believing that nitrogen removal is much more effective in small streams (i.e., headwaters in drainages) than in large rivers. They contend that, while an area

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[26] F. H. Sklar and R. E. Turner, "Characteristics of Phytoplankton Production Off Barataria Bay in an Area Influenced by the Mississippi River," *Marine Science*, v. 24 (1981): 93-106; S. E. Lohrenz, M. J. Dagg, and T. E. Whitledge, "Enhanced Primary Production at the Plume/Oceanic Interface of the Mississippi River," *Continental Shelf Research*, v. 7 (1990): 639-664; S. E. Lohrenz et al., "Variations in Primary Productivity of Northern Gulf of Mexico Continental Shelf Waters Linked to Nutrient Inputs from the Mississippi River," *Marine Ecology Progress Series*, v. 155 (1997): 435-454; D. Justic, Nancy N. Rabalais, and R. E. Turner, "Effects of Climate Change on Hypoxia in Coastal Waters: A Doubled CO_2 Scenario fo the Northern Gulf of Mexico," *Limnology and Oceanography*, v. 41, no. 5 (1996): 992-1003.

[27] D. Malakoff, "Death by Suffocation in the Gulf of Mexico," *Science*, v. 281 (July 10, 1998): 190-192. (Hereafter referred to as "Death by Suffocation.")

[28] R. Alexander, R. Smith, and G. Schwarz, "The Regional Transport of Point and Nonpoint-Source Nitrogen to the Gulf of Mexico," *Proceedings of the First Gulf of Mexico Hypoxia Management Conference*, Kenner, LA (Dec. 5-6, 1995), pp. 127-133 (hereafter referred to as "Regional Transport"); R. H. Meade, ed., *Contaminants in the Mississippi River, 1987-92,* Circular 1133 (Denver, CO: U.S. Geological Survey, 1995), 140 pp.; D. A. Goolsby et al., *Flux and Sources of Nutrients in the Mississippi-Atchafalaya River Basin*, Topic 3 Report for the Integrated Assessment of Hypoxia in the Gulf of Mexico, NOAA Coastal Ocean Program Decision Analysis Series No. 17 (Silver Spring, MD: NOAA Coastal Ocean Program, 1999).

[29] Of this total, 39% is contributed by the upper and central Mississippi River basins (which include Minnesota, Wisconsin, Iowa, Missouri, and Illinois), 22% by the Ohio River basin, and 11% by the Missouri River basin.

[30] Nitrogen runoff for the lower Mississippi basin is 2,072 kilograms of nitrogen per square kilometer per year compared to 708 kilograms of nitrogen per square kilometer per year for the upper Mississippi basin and 437 kilograms of nitrogen per square kilometer per year for the Ohio River basin.

[31] "Regional Transport," p. 131.

with higher yield per area may seem like a suitable place to focus management attention, focus should be directed to upstream areas where the total yield (regardless of the yield per area) is greater.[32] Workshops and conferences have identified strategies for implementing nutrient controls in the lower Mississippi basin.[33]

Many farming interests maintain that evidence has not proven that agricultural practices are the primary contributors to the development of the Gulf of Mexico dead zone.[34] Some farmers dispute that they contribute substantially to creating a dead zone that is as much as 1,000 miles away. They argue that their goal is to keep as much as possible of the applied nutrients on their land, since any nutrients that wash away represent wasted money. On the other hand, it is estimated that as much as half of the applied nutrients are lost to surface or ground water and to the air, resulting in approximately $750 million in excess nitrogen (calculated as fertilizer cost) entering the Mississippi River each year.[35]

Impacts of the Gulf of Mexico Dead Zone on Fishing. The Gulf of Mexico supports important, easily accessible commercial and recreational fisheries, bringing in almost $2.9 billion annually in retail sales to Louisiana and supporting almost 50,000 jobs.[36] These highly productive fisheries are the direct result of the input of nutrients from the Mississippi River watershed. Several studies have linked fishery effects, including declines in shrimp yields, with hypoxic episodes and areas in the Gulf. Evidence suggests that the dead zone forces fish and shrimp further offshore as well as into shallow nearshore areas, and reduces the area of essential habitat.[37] Hypoxia increases stress on aquatic ecosystems and may decrease

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[32] R. B. Alexander, R. A. Smith, and G. E. Swartz, "Effect of Stream Channel Size on the Delivery of Nitrogen to the Gulf of Mexico," *Nature*, v. 403 (2000): 758-761; R. B. Alexander and R. A. Smith, "Trends in the Nutrient Enrichment of U.S. Rivers During the Late 20th Century and Their Relation to Changes in Probable Stream Trophic Conditions," *Limnology and Oceanography*, v. 51 (2006): 639-654.

[33] For example, see C. L. Cordes and B. A. Vairin, eds., *Workshop on Solutions and Approaches for Alleviating Hypoxia in the Gulf of Mexico*, NWRC Special Report 98-02 (Lafayette, LA: U.S. Geological Survey, 1998), 53 p.

[34] C. David Kelly, "Hypoxia Issue Paints a Murky Picture," *Voice of Agriculture, American Farm Bureau* (Sept. 29, 1997), at [http://www.fb.org/index.php?fuseaction=newsroom.focusfocus&year=1997&file=fo0929.html].

[35] "Death by Suffocation," pp. 190-192.

[36] Southwick Associates, *The Economic Benefits of Fisheries, Wildlife and Boating Resources in the State of Louisiana* (Arlington, VA: March 1997), 21 pp.

[37] J. A. Downing et al., *Gulf of Mexico Hypoxia: Land-Sea Interactions*, Council for Agricultural Science and Technology, Task Force Report No. 134 (1999), 40 p.; N. N. Rabalais and R. E. Turner, eds., *Coastal Hypoxia: Consequences for Living Resources and Ecosystems*, Coastal and Estuarine Studies 58 (Washington, DC: American Geophysical Union, 2001), 454 p.; J. K. Craig, L. B. Crowder, and T. A. Henwood, "Spatial Distribution of Brown Shrimp (*Farfantepenaeus aztecus*) on the Northwestern Gulf of Mexico Shelf: Effects of Abundance and Hypoxia," *Canadian Journal of Fisheries and Aquatic Science*, v. 62 (2005): 1295-1308; J. K. Craig and L. B. Crowder, "Hypoxia-Induced Habitat Shifts and Energetic Consequences in Atlantic Croaker and Brown Shrimp on the Gulf of Mexico

(continued...)

biological diversity in areas experiencing repeated and severe hypoxia.[38] Crowding of marine life into restricted habitat also may lead to indirect consequences through altered competition and predation interactions. In addition, hypoxia may delay or impede the offshore migration of older, larger shrimp, preventing shrimp trawlers from selectively targeting larger shrimp for harvest.

While it is unclear what specific effects the dead zone has on Gulf fisheries, the occurrence of this dead zone may force fishing vessels to change their normal fishing patterns, possibly expending more time and fuel to harvest their catch. One study has concluded that any increase in fishing expenses could drive marginal operators out of business. Other potential impacts on Louisiana fisheries include concentration of fishing effort in other areas, resulting in localized overfishing; damage to essential habitat, and possible decreased future production; shellfish mortality, if hypoxic conditions impinge on barrier island beaches and coastal bay waters; localized mortality of finfish and shellfish in shoreline areas; and decreased growth due to reduced food resources in the sediments and water column.[39] The National Oceanic and Atmospheric Administration, Center for Sponsored Coastal Ocean Research, initiated a series of research projects in the northern Gulf of Mexico in 2003 to better understand the effects of hypoxia on fishery resources.

Chesapeake Bay

Hypoxic conditions have been recognized in Chesapeake Bay for many years.[40] In 2003, Virginia Institute of Marine Science (VIMS) scientists found a 250-square-mile area of hypoxic water in upper Chesapeake Bay at depths below about 20 feet, from north of Annapolis nearly to the mouth of the Potomac River.[41] The low oxygen levels were attributed to large nitrogen and phosphorus nutrient inputs,[42] likely carried into the bay by runoff from above-average winter snowfall and spring

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[37] (...continued)
Shelf," *Marine Ecology Progress Series*, v. 294 (2005): 79-94.

[38] "Marine Benthic Hypoxia," pp. 285-287; N. N. Rabalais and R. E. Turner, eds., *Coastal Hypoxia: Consequences for Living Resources and Ecosystems*, Coastal and Estuarine Studies 58 (Washington, DC: American Geophysical Union, 2001), 454 pp.

[39] J. Hanifen et al., "Potential Impacts of Hypoxia on Fisheries: Louisiana's Fishery-Independent Data," *Proceedings for the First Gulf of Mexico Hypoxia Management Conference*, Kenner, LA (Dec. 5-6, 1995), pp. 87-100; N. N. Rabalais and R. E. Turner, eds., *Coastal Hypoxia: Consequences for Living Resources and Ecosystems*, Coastal and Estuarine Studies 58 (Washington, DC: American Geophysical Union, 2001), 454 pp.

[40] Denise L. Breitburg, "Episodic Hypoxia in Chesapeake Bay: Interacting Effects of Recruitment, Behavior, and Physical Disturbance," *Ecological Monographs*, v. 62, no. 4 (1992): 525-546; James D. Hagy et al., "Hypoxia in Chesapeake Bay, 1950-2001: Long-Term Change in Relation to Nutrient Loading and River Flow," *Estuaries*, v. 27, no. 4 (2004): 634-659.

[41] See the VIMS press release at [http://www.vims.edu/newsmedia/press_release/hypoxia.html].

[42] For more information on nutrient input to Chesapeake Bay, see [http://chesapeake.usgs.gov/features.html].

rains. This runoff was able to pick up nutrients that had accumulated in surrounding soils during four consecutive years of dry weather. These nutrients stimulate large summertime algal blooms in the bay, reducing dissolved oxygen in bay waters when these organisms sink to the bottom and decompose. In 2006, the total hypoxic area in the bay was smaller than in previous years, possibly because spring rainfall was close to the lowest on record, resulting in less runoff and reduced nutrient input to the bay.[43]

Oregon Coast

Although permanently hypoxic water masses (oxygen minimum zones) affect large seafloor surface areas along the continental margin of the eastern Pacific Ocean, no dead zone events had been reported in the nearshore waters off the Oregon coast prior to 2002. Unlike the dead zones in estuarine systems that are caused, in large part, by excessive nutrient runoff from land, the Oregon dead zone forms along the open coast. Coastal winds drive ocean currents that upwell nutrient-rich, but oxygen-poor, waters from the deep sea onto the shallow reaches of the continental shelf. This upwelling of nutrients fuels phytoplankton blooms that eventually sink and decompose to further reduce oxygen levels in the already low-oxygen waters along the seafloor. Hypoxic zones along the Oregon coast form seasonally and can begin in late spring/early summer in response to the onset of upwelling-favorable winds from the north. This hypoxia can persist through the summer and ultimately recedes during the fall when winds again shift direction and promote ocean currents that flush low-oxygen water off the continental shelf.

In 2006, the Oregon coastal dead zone was significantly larger (more than 3,000 square kilometers), thicker, longer lasting, and lower in oxygen concentration than in previous years, extending along the ocean floor from Cape Perpetua (Florence) in the south to Cascade Head (Lincoln City) in the north, as close to shore as the 50-foot depth.[44] Underwater surveys of the 2006 event revealed complete disappearance of fish from important habitats in addition to near-complete mortalities of benthic invertebrates that are important in coastal food-webs.[45] The appearance and growth of this dead zone is attributed to fundamental, but not well understood, changes in ocean conditions off the Oregon coast.[46] The severity of the 2006 dead zone appears to reflect (1) changes in ocean and atmosphere conditions that include the strongest declines in offshore source-water oxygen content on record, (2) an exceptional shift in coastal wind patterns that has greatly enhanced upwelling currents, and (3) the persistence of low-oxygen water and phytoplankton blooms along the coast. An analogous dead zone along the open coast of Washington State may also be developing.

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[43] See "State of the Bay 2006" at [http://wrc.iewatershed.com/chesapeake/SOTB_2006.pdf].

[44] For more information, see [http://www.piscoweb.org/research/oceanography/hypoxia] and [http://oregonstate.edu/dept/ncs/newsarch/2006/Oct06/deadzoneends.html].

[45] See [http://oregonstate.edu/events/newsevents/deadzone_video.html].

[46] Brian A. Grantham, et al., "Upwelling-Driven Nearshore Hypoxia Signals Ecosystem and Oceanographic Changes in the Northeast Pacific," *Nature*, v. 429 (June 17, 2004): 749-754.

While controlling upwelling-caused hypoxic zones is unlikely, increasing the ability to predict and understand the severity and consequences of future hypoxic events will be necessary for managing and ameliorating the social and economic effects of ecosystem changes along the Oregon coast and beyond.

Policy and Management Efforts

Since a temporary, yet severe, hypoxic event could result in significant mortality or injury to marine mammals, fish, and other aquatic species, many have deemed better understanding and consistent monitoring of hypoxic phenomena necessary. NOAA initiated the Nutrient Enhanced Coastal Ocean Productivity (NECOP) program in 1989 to study the effects of nutrient discharges on U.S. coastal waters. This study found a clear link between nutrient input, enhanced primary production (i.e., algal and plant growth), and hypoxic events in the northern Gulf of Mexico.[47]

In response to a January 1995 petition from the Sierra Club Legal Defense Fund (currently known as Earthjustice Legal Defense Fund) on behalf of 18 environmental, social justice, and fishermen's organizations, the Gulf of Mexico Program[48] held a conference in December 1995 to outline the issue and identify potential actions. Following that conference, Robert Perciasepe, Assistant EPA Administrator for Water, convened an interagency group of senior Administration officials (the "principals group") to discuss potential policy actions and related science needs. Subsequently, this "principals group" created a Mississippi River/Gulf of Mexico Watershed Nutrient Task Force. Additionally, the White House Office of Science and Technology Policy's Committee on Environment and Natural Resources (CENR) conducted a Hypoxia Science Assessment at the request of EPA. The CENR assessment was peer-reviewed, made available for public comment, and submitted to the task force to assist in developing policy recommendations and a strategy for addressing hypoxia in the northern Gulf of Mexico.

In response to an integrated scientific assessment of hypoxia in the northern Gulf of Mexico by the multi-agency Watershed Nutrient Task Force,[49] a Plan of Action for addressing hypoxia was released in January 2001.[50] This plan's environmental objective is to reduce the five-year running average of the dead zone's area to 5,000 square kilometers or less by the year 2015. Estimates based on water-quality measurements and streamflow records indicate that a 40% reduction in total nitrogen flux to the Gulf is necessary to return to average loads comparable to those

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[47] NOAA, Coastal Ocean Program Office, *Nutrient-Enhanced Coastal Ocean Productivity, Proceedings of 1994 Synthesis Workshop* (1995), p. 119; see also *Estuaries*, v. 17, no. 4 (Dec. 1994): 729-911.

[48] The Gulf of Mexico Program is a cooperative federal-state effort beginning after Congress, through P.L. 102-178, designated 1992 as the Year of the Gulf of Mexico. For additional information on this program, see [http://www.epa.gov/gmpo/].

[49] See [http://www.nos.noaa.gov/products/pubs_hypox.html].

[50] See [http://www.epa.gov/msbasin/taskforce/pdf/actionplan.pdf].

during 1955-1970.[51] Model simulations suggest that, short of this 40% reduction, nutrient load reductions of about 20%-30% would result in a 15%-50% increase in dissolved oxygen concentrations in bottom waters. Strategies selected focus on encouraging voluntary, practical, and cost-effective actions; using existing programs, including existing state and federal regulatory mechanisms; and following adaptive management. A reassessment of progress on implementing this action plan was initiated in 2005.[52]

A key consideration is the level and duration of the necessary reduction in excess nutrients from watersheds. Many agricultural lands have been saturated with nutrients for many years, and it may take a long time to "cycle out" excess nitrogen and phosphorus, even if application rates are reduced.[53] While some believe this problem may have no fast solutions and any management regime considered will need to recognize that progress or improvement may not be apparent for years or even decades, others suggest that improved agricultural practices in efficient application of chemical fertilizers and prevention of soil erosion could yield immediate and measurable benefits. One model suggests that a 12% to 14% reduction in the use of fertilizer on croplands in the Mississippi River basin would reduce the net anthropogenic nitrogen inputs by about 30%, without any loss in crop production. Various policy options for modifying agriculture practices continue to be discussed.[54]

Because nonpoint sources are major contributors to the problem at the mouth of the Mississippi River system, many believe the Clean Water Act is the appropriate legal framework for addressing future nutrient inputs. Under §319 of the Clean Water Act, Louisiana and other states have initiated nonpoint-source[55] control programs. These programs seek to combine local, state, and federal agency resources to address pollution from nonpoint sources within each state.[56] To effectively address concerns, however, nonpoint-source programs would need to be encouraged, funded, and implemented throughout the Mississippi River watershed. Under §303 of the Clean Water Act, states must identify water-quality-limited segments of their waters that are not meeting standards, and then establish total maximum daily loads (TMDLs) for each listed water and each pollutant (e.g., nutrients) that is not meeting

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[51] D. Scavia, et al., "Predicting the Response of Gulf of Mexico Hypoxia to Variations in Mississippi River Nitrogen Load," *Limnology and Oceanography*, v. 48 (2003): 951-956.

[52] See [http://www.epa.gov/msbasin/taskforce/reassess2005.htm].

[53] "Death by Suffocation" (citing Don Goolsby, USGS, Denver, CO), pp. 190-192.

[54] For example, see Suzie Greenhalgh and Amanda Sauer, "Awakening the Dead Zone: An Investment for Agriculture, Water Quality, and Climate Change," *World Resources Institute Issue Brief* (February 2003), 24 p.

[55] Originating from land use activities; sediment, organic and inorganic chemicals, and biological, radiological, and other toxic substances are carried to lakes and streams by surface runoff.

[56] D. Sabin and J. Boydstun, "Louisiana Activities and Programs in Nutrient Control and Management," *Proceedings of the First Gulf of Mexico Hypoxia Management Conference,* Kenner, LA (Dec. 5-6, 1995), pp. 196-198.

current water quality standards. In addition, agricultural research and educational outreach/assistance to farmers might complement regulatory efforts.

Congress took note of the hypoxia problem in 1997 when the conference report on FY1998 Department of the Interior appropriations (H.Rept. 105-337) directed the USGS to give priority attention to hypoxia in its FY1999 budget. Near the end of the 105th Congress, provisions of the Harmful Algal Bloom and Hypoxia Research and Control Act of 1998 were incorporated into the Coast Guard Authorization Act of 1998. This measure was signed into law as P.L. 105-383 on November 13, 1998; Title VI authorized appropriations through NOAA to conduct research, monitoring, education, and management activities for the prevention, reduction, and control of hypoxia, harmful algal blooms, *Pfiesteria*, and other aquatic toxins. In 2004, Title I of P.L. 108-456, the Harmful Algal Bloom and Hypoxia Amendments Act of 2004, expanded this authority and reauthorized appropriations through FY2008.

Appropriations and Funding

National Ocean Service. Hypoxia research is regularly funded through appropriations to the National Ocean Service — part of the National Oceanic and Atmospheric Administration in the Department of Commerce — in their Extramural Research account under *National Centers for Coastal Ocean Science*. For FY2007, NOAA requested $6 million for extramural research for grants related to harmful algal bloom and hypoxia forecasting.

Department of Agriculture. In the last few years, the U.S. Department of Agriculture's Cooperative State Research, Education, and Extension Service has provided a special research grant of around $220,000 annually to Iowa State University's Leopold Center for Sustainable Agriculture for a project to define and implement new methods and practices in farming that reduce impacts on water quality and the hypoxia problem in the Gulf of Mexico.

Environmental Protection Agency. In the last few years, the Environmental Protection Agency's Environmental Programs and Management account has provided a specific authorization of around $125,000 annually for the Missouri Department of Agriculture's Hypoxia Education and Stewardship Project. This effort seeks to educate Missouri producers about hypoxia and encourage use of practices that will reduce the amount of nitrogen lost through leaching and/or evaporation. The EPA has also funded research on Gulf of Mexico hypoxia through its Gulf Breeze Laboratory.