Estuaries Vol. 27, No. 4, p. 634–658 August 2004

Hypoxia in Chesapeake Bay, 1950–2001: Long-term Change in Relation to Nutrient Loading and River Flow

JAMES D. HAGY*, WALTER R. BOYNTON, CAROLYN W. KEEFE, and KATHRYN V. WOOD

University of Maryland Center for Environmental Science, Chesapeake Biological Laboratory, Post Office Box 38, Solomons, Maryland 20688

ABSTRACT: A 52-yr record of dissolved oxygen in Chesapeake Bay (1950–2001) and a record of nitrate (NO_3^-) loading by the Susquehanna River spanning a longer period (1903, 1945–2001) were assembled to describe the long-term pattern of hypoxia and anoxia in Chesapeake Bay and its relationship to NO_3^- loading. The effect of freshwater inflow on NO_3^- loading and hypoxia was also examined to characterize its effect at interannual and longer time scales. Year to year variability in river flow accounted for some of the observed changes in hypoxic volume, but the long-term increase was not due to increased river flow. From 1950–2001, the volume of hypoxic water in mid summer increased substantially and at an accelerating rate. Predicted anoxic volume (DO < 0.2 mg l^{-1}) at average river flow increased from zero in 1950 to 3.6 × 10^9 m^3 in 2001. Severe hypoxia (DO < 1.0 mg l^{-1}) increased from 1.6 × 10^9 to 6.5 × 10^9 m^3 over the same period, while mild hypoxia (DO < 2.0 mg l^{-1}) increased from 3.4 × 10^9 to 9.2 × 10^9 m^3. NO_3^- concentrations in the Susquehanna River at Harrisburg, Pennsylvania, increased up to 3-fold from 1945 to a 1989 maximum and declined through 2001. On a decadal average basis, the superposition of changes in river flow on the long-term increase in NO_3^- resulted in a 2-fold increase in NO_3^- loading from the Susquehanna River during the 1960s to 1970s. Decadal average loads were subsequently stable through the 1990s. Hypoxia was positively correlated with NO_3^- loading, but more extensive hypoxia was observed in recent years than would be expected from the observed relationship. The results suggested that the Bay may have become more susceptible to NO_3^- loading. To eliminate or greatly reduce anoxia will require reducing average annual total nitrogen loading to the Maryland mainstem Bay to 50 × 10^6 kg yr^{-1}, a reduction of 40% from recent levels.

Introduction

Depletion of dissolved oxygen (DO) from deep waters is a common feature in estuaries and other coastal systems where seasonal or permanent stratification of the water column restricts aeration of bottom waters by the atmosphere (e.g., Chesapeake Bay, Officer et al. 1984; Black Sea, Zaitsev 1992; Baltic Sea, de Jonge et al. 1994; Long Island Sound, Welsh et al. 1994; Gulf of Mexico, Rabalais et al. 1999, 2002). Hypoxia occurs when DO concentrations become sufficiently low to harm biota directly or to adversely affect normal ecological interactions. The DO concentration that defines hypoxia is nearly arbitrary since a continuous spectrum of effects has been observed as DO declines from not far below saturation concentrations to anoxia (complete absence of DO, Diaz and Rosenberg 1995). Behavioral and physiological responses can occur with moderate depression of DO, with detrimental effects on individuals and populations (Breitburg et al. 1997; Breitburg 2002). Critical DO thresholds below which severe effects occur vary. Ritter and Montagna (1999) identified 3 mg l^{-1} as a threshold for macrobenthos in Corpus Christi Bay, a higher value than the often-cited value of 2 mg l^{-1}. Some benthic animals often tolerate very low DO and may readily survive concentrations as low as 1 mg l^{-1}. Even the most hypoxia-tolerant benthic communities become degraded or afaunal as DO declines to less than 1 mg l^{-1} and to anoxia (Diaz and Rosenberg 1995). Biogeochemical processes change when anoxia develops, leading to efflux of hydrogen sulfide and inorganic nitrogen (N) and phosphorus (P) from sediments (Fenchel and Blackburn 1979; Cowan and Boynton 1996). Hydrogen sulfide helps maintain water column anoxia and is toxic to marine life. Additional nutrient inputs to the upper water column via upwelling provide positive feedback, contributing to further eutrophication and oxygen depletion. Given the spectrum of effects, a variety of levels were adopted here as definitions for hypoxia, with descriptive terms (e.g., severe, moderate) and specific definitions (e.g., DO < 2 mg l^{-1}) indicated as needed.

Moderate hypoxia (DO < 2 mg l^{-1}) to anoxia presently affects much to all of the below-pycnocline waters in the central one-third of Chesapeake Bay (102–222 km, Fig. 1) for most or all of the

* Corresponding author; current address: U.S. Environmental Protection Agency, National Health and Environmental Effects Research Laboratory, Gulf Ecology Division, 1 Sabine Island Drive, Gulf Breeze, Florida 32561; tele: 850/934-2455; fax: 850/934-2401; e-mail: hagy.jim@epa.gov

© 2004 Estuarine Research Federation

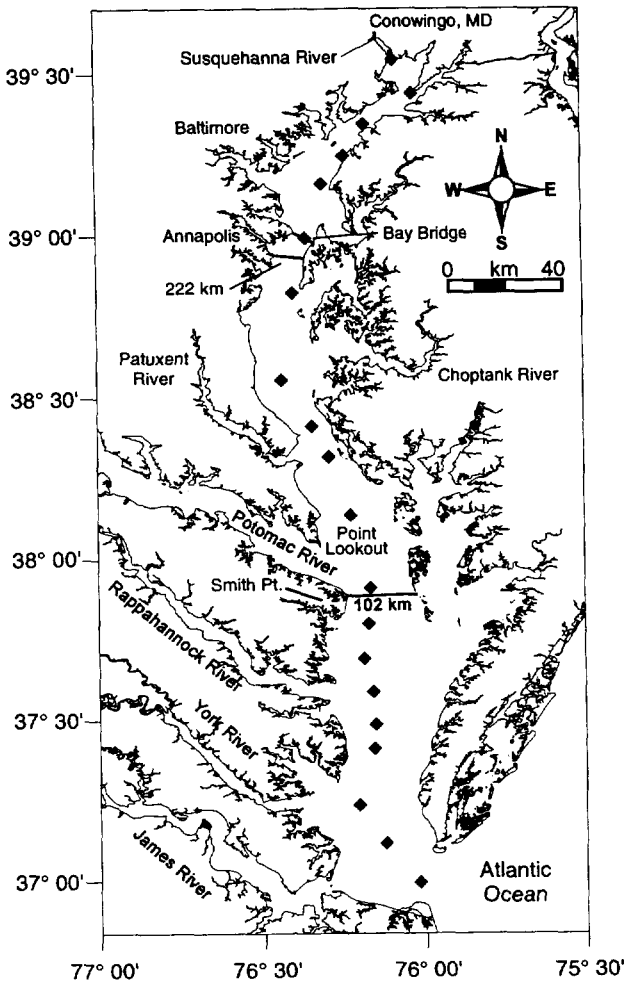


FIG. 1. A map of Chesapeake Bay showing major tributaries, important points of reference, the locations of Chesapeake Bay Water Quality Monitoring Program stations used in this study, and the boundaries between major regions of the Bay (Upper Bay > 222 km from Bay Mouth, Mid Bay 102–222 km, Lower Bay 0–102 km). The station identifications are (north to south): CB1.1, CB2.1, CB2.2, CB3.1, CB3.2, CB3.3C, CB4.1C, CB4.3C, CB4.4, CB5.1, CB5.2, CB5.3, CB5.4, CB5.5, CB6.1, CB6.2, CB6.3, CB6.4, CB7.3, CB7.4.

summer. Events in which a combination of wind-driven and tide-driven tilting of the pycnocline brings hypoxic waters into shallow areas or to the surface are common in the mesohaline Bay (Breitburg 1990; Sanford et al. 1990). Following an 18-hr event of this nature, divers observed that the sediment surface was littered with dead fish (Breitburg 2002). Degraded benthic communities in deeper portions of the mid Bay, and in adjacent shallower areas (Hagy 2002), also illustrate the harmful effects of Chesapeake Bay hypoxia. The extent of hypoxia in Chesapeake Bay is sufficient to cause significant ecological harm. There is a general recognition that hypoxia has probably increased in extent in Chesapeake Bay and that a long-term increase in the loading rates of N and P is probably the primary factor leading to the present condition (Environmental Protection Agency 2000). Management efforts directed toward reducing N and P loading are being implemented (Environmental Protection Agency [EPA] 2002). It remains unclear how much improvement in hypoxia can be expected from reductions in N and P loading. A major source of uncertainty is an inadequate knowledge of long-term trends in both hypoxia and nutrient loading rates.

Two well-known studies, published during the 1980s, provide the principal basis for the current understanding of trends in hypoxia since 1950 (Flemer et al. 1983; Seliger and Boggs 1988), although additional description and analysis can be found in other studies (Taft et al. 1980; Officer et al. 1984). Flemer et al. (1983) and Seliger and Boggs (1988) both estimated the volume of Bay water affected by hypoxia between 1950 and 1980 using data collected during summer surveys conducted by the Chesapeake Bay Institute in approximately half the years. One and occasionally two surveys were conducted each summer. Flemer et al. (1983) examined data for 13 yr during the period 1950–1980 and concluded that hypoxia increased dramatically. Consistent with the present view, they suggested that the increase in hypoxia could be attributed to increased nutrient inputs, noting a 2- to 3-fold increase in nitrate (NO_3^-) concentrations at a tidal fresh Chesapeake Bay station between 1965 and 1980. Seliger and Boggs (1988) examined the same DO data set, omitting data from 1972 and 1977, but with the addition of data from 1952, 1958, 1983, and 1984. In contrast to Flemer et al. (1983), they concluded that there was not sufficient evidence of a long-term increase in hypoxia. They explained most of the observed interannual differences in hypoxic volume in terms of interannual differences in spring freshwater discharge into the estuary and called into question the existence of any long-term trend.

The results of Seliger and Boggs (1988) documented the correlation between Chesapeake Bay hypoxia in summer and springtime discharge from the Susquehanna River. River flow-hypoxia relationships have also been found for other coastal systems (e.g., Rabalais et al. 1999; Rabalais and Turner 2001). Spring river flow is an extremely strong predictor of summer water column stratification in the middle Chesapeake Bay (Boicourt 1992; Hagy 2002), which affects vertical mixing of DO across the pycnocline (Hagy 2002), and is one of several important factors contributing to formation of hypoxia. Increased river flow also increases nutrient transport from rivers into the

coastal zone (e.g., Langland et al. 2001; Justić et al. 2003), contributing to a variety of biological and biogeochemical responses such as increased primary production and benthic nutrient recycling (Boynton and Kemp 2000). River flow as a predictor of hypoxia encompasses both direct physical effects and indirect biological effects associated with increased nutrient loading during high flow periods. The correlation between Susquehanna River discharge and nutrient loading since 1978, the period encompassing the most detailed nutrient loading estimates, is very strong, precluding statistical resolution of physical versus nutrient loading effects on hypoxia.

An analysis of nutrient (N and P) loading rates that resolves long-term trends, interannual differences, and seasonal patterns in nutrient loading rates to Chesapeake Bay would be useful because it would permit a direct analysis of the response of hypoxia to nutrient inputs. Loading from the Susquehanna River is of particular interest because this river contributes a large fraction of N loading to the system. Among the major tributaries to Chesapeake Bay, only the Susquehanna River flows directly into the mainstem of Chesapeake Bay. Loads from other rivers are substantially reduced, or even eliminated, by their respective tributary-estuaries before reaching the mainstem Bay (Boynton et al. 1995). Despite the availability of ample measurements of N concentration over time (as revealed in this study), studies of the Susquehanna River have described only a general pattern of increased loading over the long term. Based on an all-forest scenario for the precolonial period, Boynton et al. (1995) estimated that total nitrogen (TN) and total phosphorus (TP) loading rates to the Bay have increased 6.2-fold and 17.1-fold, respectively. National trends and trends within the northeastern United States suggest that N loading to Chesapeake Bay probably increased very rapidly from 1940 until sometime during 1970–1980, then stabilized (Smith et al. 1987; Jaworski et al. 1997; Howarth et al. 2002). Studies within the Chesapeake region generally match these larger trends. Flemer et al. (1983) observed a 2- to 3-fold increase in NO_3^- concentrations in the tidal fresh Bay between 1965 and 1980, suggesting that NO_3^- loading from the Susquehanna River increased similarly over that time. Jaworski et al. (1992, 1997) estimated that TN inputs from the Potomac River were very stable from 1906 until mid century, increased approximately 60% between 1954 and 1985, and subsequently decreased slightly. Hagy et al. (1998) estimated that TN loading rates at the fall line of the Patuxent River approximately doubled between 1960 and 1990 before decreasing substantially during the 1990s, principally due to N removal from sewage. One may suspect that N loading from the Susquehanna River may have increased 2- to 3-fold since 1950.

The objectives of this study are to document and analyze the changes in Chesapeake Bay hypoxia during the period 1950–2001 by combining the extensive data collected by the Chesapeake Bay Institute (1950–1980) and the Chesapeake Bay Monitoring Program (1984–2001). We also characterize changes in N loading from the Susquehanna River during the same period by assembling the available data describing N concentrations in the lower reaches of the river. Recognizing the potential for river flow and possibly other factors to modify the development of hypoxia in response to increased nutrient loading, we also identify and quantify factors other than N loading that explain additional variability in Chesapeake Bay hypoxia.

Methods

Study Area

Chesapeake Bay is a large, partially stratified estuary that extends 300 km from the mouth of the Susquehanna River to the Atlantic Ocean (Fig. 1). The oligohaline upper Bay has a mean depth of 4.5 m with a deeper (10 m) channel near the eastern margin. The mesohaline mid Bay has a deep (20–50 m) central channel flanked by shallower shoal areas to the east and west, giving it a deeper mean depth of 10 m. The polyhaline lower Bay is broader with a wide central channel region averaging 15 m depth as well as broad shoal areas on the flanks of the channel. The mean depth is 9 m.

The physical transport regime throughout most of the estuary is characterized by 2-layer gravitational circulation in which net up-estuary advection occurs below the pycnocline and net down-estuary advection occurs in the surface layer (Pritchard 1952). In the upper Bay, circulation is initially down-estuary at all depths but makes a transition to the 2-layer circulation characteristic of the rest of the Bay.

The watershed of the Chesapeake Bay encompasses 172,000 km^2. The Susquehanna River, the largest tributary to the Bay drains 71,200 km^2, accounting for 41% of the Chesapeake Bay watershed and >80% of the area draining directly into the mainstem Bay (Fig. 2; Hagy 2002). More than 98% of the Susquehanna River watershed lies upstream from a flow gauging and water quality monitoring station at Conowingo, Maryland. A monitoring station at Harrisburg, located upstream from Conowingo, includes 88% of the Susquehanna River watershed. Five tributary creeks and rivers entering the Susquehanna River below Harrisburg (West Conewago Creek, Swatara Creek, Conestoga

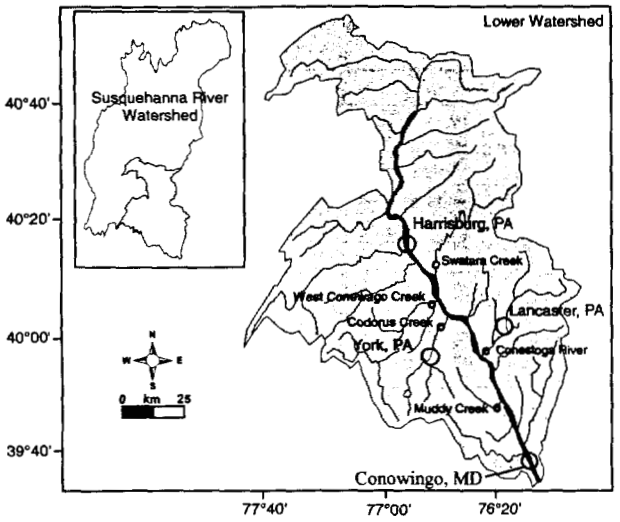


FIG. 2. The entire Susquehanna River watershed with the lower watershed enlarged in the inset. The locations of the Harrisburg, Pennsylvania, and Conowingo, Maryland, river gauging and water quality monitoring stations in smaller tributary rivers within the lower Susquehanna River watershed. The cities of Lancaster, York, and Harrisburg are major population centers in the region.

River, Codorus Creek, Muddy Creek) account for 8% of the overall watershed and 67% of the drainage area below Harrisburg.

Sources of Data and Analytical Methods

This study was based on previously collected water quality, river discharge, and nutrient concentration data (Tables 1 and 2). The Chesapeake Bay Institute DO data (1950–1980) were collected by pumping water from depth and measuring DO using a modified Winkler titration (Carpenter 1965a). This has an estimated accuracy of 0.1% (Carpenter 1965b). The Chesapeake Bay Water Quality Monitoring Program (1985–2001) measured water temperature, salinity, and DO using multiparameter water quality monitoring instruments employing a polarographic oxygen probe (EPA 1993). The accuracy of oxygen measurements varied with the instrument used as well as daily calibration and use. Winkler titration was used daily for a subset of measurements to ensure accuracy to within ± 0.5 mg l^{-1} (EPA 1993). Experience with similar instruments suggests that accuracy was probably somewhat better.

An intermittent record of NO_3^- concentration in Susquehanna River waters at Harrisburg was assembled (Table 2). The 715 observations compiled provide an intermittent to regular record of NO_3^- from 1945 to 1995, plus several observations during 1902–1903. Water samples were obtained from surface water, but the river is very shallow and turbulent at this location. During 1945–1957, NO_3^- was monitored at a series of locations across the river, accessed via a highway bridge. We used the average value, but excluded data from the westernmost location. At low flow, this location typically had much higher (2 to 10 times) concentrations than other locations. We confirmed that a creek entering the river at this location heavily influenced NO_3^- concentrations (Langland personal communication). Beginning in 1973, a single value was recorded for about half of the sampling dates. This value was a composite sample, taken to be representative and comparable to the earlier data (Langland personal communication). The mean of all observations was used for a few dates on which as many as 10–15 samples were collected.

Monitoring of NO_3^- at Harrisburg concluded in 1995. Monitoring of NO_3^- at Conowingo, located downstream from Harrisburg (Fig. 2), began in 1978 and resulted in 817 observations during 1978–2001 (Table 2). The seasonally varying ratio of contemporaneous NO_3^- concentrations (1978–1995) was used to estimate concentrations at Harrisburg for 1995–2001, completing a 57-yr record.

TABLE 1. Data types, spatial resolution, temporal resolution, and sources of data.

Data Type (Period of Record)	Spatial Resolution	Temporal Resolution	Source
Chesapeake Bay Institute DO data (1950–1980)	~10 stations on Bay axis, 2–4 m vertical resolution with gaps	1–4 surveys per year, 1–2 per summer	Chesapeake Bay Institute
Chesapeake Bay Monitoring Program DO, salinity, temperature data (1984–2001)	~20 stations on Bay axis, 1 m vertical resolution	~20 surveys per year, biweekly during summer	USEPA (unpublished data), Chesapeake Bay Monitoring Program (unpublished data)
River flow, Conowingo Dam or Harrisburg, PA[1] (1900–2001)	1 location	Daily	U.S. Geological Survey (unpublished data)
NO_3^- concentrations	7 locations	Variable (see Table 2)	See Table 2

[1] Monthly mean Susquehanna River flow at Conowingo, Maryland, prior to 1968, when flow monitoring at Conowingo commenced, was estimated from the flow record at Harrisburg, Pennsylvania. The flow at Harrisburg includes >90% of the flow at Conowingo. The two flows are related with $r^2 > 0.99$ for the period 1968–2001.

TABLE 2. The sources of nitrate data obtained for sites in the Susquehanna River basin at Harrisburg, Pennsylvania, Conowingo, Maryland, and several additional tributaries to the Susquehanna River below Harrisburg.

Period	Obs.	Basin	Location	Source
1902–1903	9	Susquehanna	Harrisburg, PA	Leighton (1904)
1945–1957	165	Susquehanna	Harrisburg, PA	USGS (unpublished data), Reports[1]
1963–1972	88	Susquehanna	Harrisburg, PA	USGS[2]
1973–1995	453	Susquehanna	Harrisburg, PA	USGS[3]
1978–2001	817	Susquehanna	Conowingo, MD	USGS[3]
1984–1987	104	West Conewago Creek	Manchester, PA	USGS[3]
1986–1987	93	Swatara Creek	Hershey, PA	USGS[3]
1984–1993	258	Conestoga River	Conestoga, PA	USGS[3]
1985–1987	160	Codorus Creek	Pleasureville, PA	USGS[3]
1977–1981	160	Pequea Creek	Martic Forge, PA	USGS[3]
1994	2	Muddy Creek	Castle Fin, PA	USGS[3]

[1] U.S. Geological Survey (1947, 1949, 1950, 1952, 1953, 1954a, 1954b, 1955, 1956, 1957, 1960a,b).
[2] Data available from USGS http://water.usgs.gov/pa/nwis.
[3] Modern surface water quality data from the U.S. Geological Survey are available from the USGS at http://water.usgs.gov.

NO_3^- was monitored in five major tributaries of Susquehanna River below Harrisburg over varying periods, mostly during the 1980s. From 2 to 258 observations were recorded for each tributary (Table 2). NO_3^- data since 1978 were collected using field methods described by Ward and Harr (1990) and using automated colorimetric methods for the laboratory analysis of NO_3^- (EPA 1979). Standard manual colorimetric methods were used for the older data. Concentrations in the early portion of the record were reported as mg NO_3^- l^{-1} and were converted to mg NO_3^--N l^{-1}. Because of the similarity of the methods, and the fact that all the observed concentrations are well within the analytical limits of the methods used, all the data are believed to be comparable. All references to NO_3^- indicate NO_3^--N.

Historical N fertilizer use, land in farms, and human population data were compiled for the Susquehanna watershed for the period 1940–2000 by summing county level data for counties substantially within the Susquehanna watershed. For the watershed above Harrisburg, this included 26 counties in Pennsylvania and 6 counties in New York. For the watershed downstream of Harrisburg, this included 4 large counties in southern Pennsylvania. N fertilizer use data were obtained from the Pennsylvania Department of Agriculture (1963–1989, 1990–1997, 1998–1999). Land in farms data were obtained from U.S. Census Bureau (1952, 1961, 1972, 1981, 1989) and U.S. Department of Agriculture (1999). Population data were obtained from the U.S. Census Bureau (2003a,b; unpublished data).

Calculation of Hypoxic Volumes

Hypoxic volumes are defined as the volume of water with DO concentration below a defined level. Summer hypoxic volumes were calculated for each year from 1950 through 2001 for which data were available and for each of three levels: DO < 0.2 mg l^{-1} (near-anoxia), DO < 1.0 mg l^{-1} (severe hypoxia), and DO < 2.0 mg l^{-1} (moderate hypoxia). There was typically one cruise per summer in the Chesapeake Bay Institute data set, usually during July. Data from a July cruise were always used when available. In 1958, 1960, and 1972, data from August were used. In 1978, data from September were used (Table 3). To calculate hypoxic volume, the available mid channel oxygen data were interpolated to a 2-dimensional grid with grid cell dimensions of 1 m vertical by 1 nautical mile along the estuary axis (longitudinal). The grid corresponds to tabulated cross-sectional volumes for Chesapeake Bay (Cronin and Pritchard 1975), which were summed for cells with DO less than the specified maximum to compute hypoxic volume. This approach assumes DO isopleths are level (i.e., a flat oxycline), which this study found to be a reasonable assumption for Chesapeake Bay (e.g., Buzzelli et al. 2002).

The interpolation procedure involved a two-step process to account for the vastly smaller scales (m) and sharper DO gradients in the vertical dimension as compared to the horizontal dimension (km). First, linear interpolation was used to extrapolate the vertical DO profile at each station to a 1-m interval. Linear interpolation was then used to extrapolate horizontally at constant depth. This algorithm preserved expected horizontal and vertical gradients when sampling was thorough, as was always the case with the Chesapeake Bay Monitoring Program data (1984–2001). Sometimes such reasonable results were obtained with more sparse data; some particular data gaps led to obvious ar-

TABLE 3. Data sources, cruise dates, river flow, and calculated hypoxic volumes for Chesapeake Bay from 1950–2001.

Year	Dates	Jan–May Average River Flow ($m^3 s^{-1}$)	DO < 0.2 mg l^{-1}	DO < 1.0 mg l^{-1}	DO < 2.0 mg l^{-1}	Comment, Data Source
1950	July 14–19	1,966	0.00	0.98	2.17	Chesapeake Bay Institute (1950)
1952	July 15–August 6	2,370	1.93	3.47	5.78	Chesapeake Bay Institute (1954)
1957	July 23–26	1,625	0.51	1.84	2.86	(b), Chesapeake Bay Institute (1962a)
1958	August 6–22	2,080	0.41	4.76	7.37	Chesapeake Bay Institute (1962b)
1959	July 6–17	1,565	0.00	2.61	4.85	Chesapeake ay Institute (1962c)
1960	August 22–September 9	2,028	0.24	3.70	4.20	(c), Chesapeake Bay Institute (1962d)
1961	July 19–31	1,982	0.02	4.64	6.35	Chesapeake Bay Institute (1962e)
1962	July 24–August 5	1,674	0.00	1.41	3.90	Chesapeake Bay Institute (1962f)
1963	July 30–August 15	1,399	0.03	2.67	3.50	(e), Chesapeake Bay Institute (1963)
1965	July 1–6	1,122	0.00	0.08	2.34	(b), Whaley et al. (1966)
1968	July 8–10	1,165	0.75	3.46	4.78	(d), USEPA unpublished data
1969	July 7–10	1,021	0.74	1.67	2.44	Taylor and Cronin (1974)
1970	July 9–12	1,857	0.37	1.36	2.96	Taylor and Cronin (1974)
1972	August 27–31	1,998	3.70	5.12	7.64	(c), USEPA unpublished data
1973	June 25–29	1,830	0.83	2.76	4.93	USEPA unpublished data
1978	September 18–20	2,395	1.84	2.87	4.69	(c), Cronin et al. (1982)
1979	July 9–12	2,290	1.28	1.79	2.88	Cronin et al. (1982)
1980	July 23–August 2	1,506	2.20	3.52	5.03	Cronin et al. (1982)
1984	July Avg.	1,809	2.84	4.46	5.49	(a), USEPA unpublished data
1985	July Avg.	2,205	0.54	2.47	4.42	(a), USEPA unpublished data
1986	July Avg.	1,128	4.23	7.52	11.00	(a), USEPA unpublished data
1987	July Avg.	1,627	1.57	5.19	9.30	(a), USEPA unpublished data
1988	July Avg.	1,301	2.03	3.61	5.02	(a), USEPA unpublished data
1989	July Avg.	1,278	4.21	7.25	11.16	(a), USEPA unpublished data
1990	July Avg.	1,512	2.02	3.45	5.86	(a), USEPA unpublished data
1991	July Avg.	1,579	2.42	7.35	9.26	(a), USEPA unpublished data
1992	July Avg.	1,624	2.52	4.31	6.52	(a), USEPA unpublished data
1993	July Avg.	1,209	5.87	9.08	11.95	(a), USEPA unpublished data
1994	July Avg.	2,645	3.30	5.70	7.68	(a), USEPA unpublished data
1995	July Avg.	1,094	3.36	5.83	9.86	(a), USEPA unpublished data
1996	July Avg.	2,355	2.84	4.54	5.84	(a), USEPA unpublished data
1997	July Avg.	1,296	4.37	5.71	8.15	(a), USEPA unpublished data
1998	July Avg.	2,363	6.25	9.61	12.07	(a), USEPA unpublished data
1999	July Avg.	1,223	1.20	5.20	9.33	(a), USEPA unpublished data
2000	July Avg.	1,572	3.05	5.25	7.40	(a), USEPA unpublished data
2001	July Avg.	1,097	1.18	3.13	4.55	(a), USEPA unpublished data

(a) Average of two July cruises.
(b) No data south of Potomac River, but available data fully delineates the 1 mg l^{-1} and lower contours.
(c) Cruise dates later than likely period of maximum hypoxic volume.
(d) DO concentration in lower pycnocline and upper portion of lower layer poorly described. Pycnocline depth considered when interpolating DO profile.
(e) No data south of Potomac River. Data do not fully delineate DO contours greater than 0.2 mg l^{-1}. Hypoxic volumes for 1.0 and 2.0 mg l^{-1} are lower bounds.

tifacts. For example, when DO measurements were not made below the pycnocline, the extrapolated vertical profile will not reflect an oxycline, even when it is known to exist from stations immediately up-estuary or down-estuary. Problems with interpolation were evaluated using contour plots generated for each year prior to 1985.

To correct interpolation problems, adjustments were made to the interpolation procedure in three specific situations. When DO was not measured at the lower extent of the pycnocline, but was measured within the bottom mixed layer, the concentration within the bottom mixed layer was assumed to extend upward to the lower extent of the pycnocline. When DO was not measured within the bottom mixed layer, the DO profile within the bottom mixed layer was interpolated from adjacent up-Bay and down-Bay stations, rather than interpolating vertically through the pycnocline. Unless data were present to indicate otherwise, the DO contours near the northern extent of the channel trench were assumed to extend unmodified at constant depth to the northern limit of the trench. These modifications resulted in contoured DO distributions for sparse data sets that were structurally consistent with contoured DO distributions observed when sampling was thorough (1985–2001). The estimated distributions were plausible given the known water column structure and approximate physical transport regime.

The interpolation algorithm that was used to interpolate 1950–1980 DO profiles was also used to analyze the modern DO record (1984–2001). Adjustments for incomplete data were never needed because the sampling regime of the modern water quality monitoring program was thorough and consistent. Because two July cruises and corresponding hypoxic volume estimates were available for each year, the average of the two values was used to determine an estimate of July hypoxic volume for each year.

Statistical Methods

Two relatively complex multiple linear regression models that were used are described here to provide more detail. Simpler regression models, which require little explanation, are presented with the results.

The long-term change in hypoxic volume was evaluated using a multiple linear regression model that accounted for interannual differences in average river flow. Because the variability in hypoxic volumes increased with the mean, the data were log transformed. A small constant was added before transformation due to zero values in some years. Graphical analysis suggested that the rate of long-term change in the log of hypoxic volume increased over time. The data were fitted to the second-order polynomial

$$V' = \beta_0 + \beta_1 T + \beta_2 T^2 + \beta_3 Q \qquad (1)$$

where $V' = \ln(V + 1)$, V is the hypoxic volume, and T is the number of years since 1949. Average Susquehanna River flow, Q, was computed for January–May in each year. The optimal period of time over which to average river flow was evaluated by examining several periods. Asymmetric confidence bands for the predicted means were generated by computing confidence limits on the log-scale, then back-transforming.

An estimate of average NO_3^- concentration at Harrisburg was computed for each month from the intermittent record of NO_3^- using an analysis of covariance model, suggested by initial graphical analysis. The model fits seasonality in concentration, a seasonally varying long-term trend in concentration, and a joint-point, the time at which the long-term trends in concentration change. The model has the form

$$\begin{aligned} C = {} & \beta_0 + \beta_1 \sin(2\pi T) + \beta_2 \cos(2\pi T) \\ & + T[\beta_3 + \beta_4 \sin(2\pi T) + \beta_5 \cos(2\pi T)] \\ & + s(T - T_0)\{\beta_6 + \beta_7 \sin[2\pi(T - T_0)] \\ & + \beta_8 \cos[2\pi(T - T_0)]\} \end{aligned} \qquad (2)$$

where

$$s = \begin{cases} 0 & \text{when } T \le T_0 \\ 1 & \text{when } T > T_0, \end{cases}$$

T is the time elapsed (yr) since 1945, and T_0 is the location in time of the joint-point. The coefficients β_0, β_1, and β_2 have concentration units (mg l^{-1}), while $\beta_3 \ldots \beta_8$ are rates of change in concentration (mg l^{-1} yr^{-1}). The regression model describes a linear but seasonally varying change in concentration over time up to time T_0, when the seasonally varying slope may change. The rate of change in NO_3^- up to T_0 is $\beta_3 + \beta_4 \sin(2\pi T) + \beta_5 \cos(2\pi T)$, whereas after T_0 the slope changes to $\beta_3 + \beta_6 + (\beta_4 + \beta_7)\sin[2\pi(T - T_0)] + (\beta_5 + \beta_8)\cos[2\pi (T - T_0)]$. The joint-point, T_0, was estimated to the nearest integer by fitting the model using a range of values and selecting the T_0 that resulted in the lowest residual error. Discontinuity of model derivatives at the joint-point precluded use of nonlinear fitting procedures that have been suggested for fitting joint-point regression models (e.g., SAS Institute 1989). The disadvantage of the procedure used is that a confidence interval cannot be estimated for the date of the joint-point. Model residuals were analyzed for goodness-of-fit and regression model assumptions.

TABLE 4. Estimated parameters of the multiple regression relating Chesapeake Bay hypoxic volumes for 1950–2001 (10^9 m^3) to time (yr) and January–May average Susquehanna River flow (m^3 s^{-1}). The model is $V' = \beta_0 + \beta_1 T + \beta_2 T^2 + \beta_3 Q$, where $V' = \ln(V + 1)$, V is the hypoxic volume and T is the number of years since 1949. Significance of the long-term trend was determined by significance for the joint hypothesis H_0: $\beta_1 = 0$, $\beta_2 = 0$.

DO Threshold (model r^2)	*Intercept*, β_0	Linear Trend, β_1	Quadratic Trend, β_2	Trend Significance, $P(H_0: \beta_1 = 0, \beta_1 = 0)$	River Flow-dependence, β_3 $P(H_0: \beta_3 = 0)$
< 0.2 mg l^{-1} (r^2 = 0.68)	−0.90	$3.96(10)^{-2}$	$-1.52(10)^{-4}$	<0.01	$4.60(10)^{-4}$ ($p < 0.01$)
< 1.0 mg l^{-1} (r^2 = 0.49)	0.41	$1.58(10)^{-2}$	$8.94(10)^{-5}$	<0.01	$3.20(10)^{-4}$ ($p < 0.05$)
< 2.0 mg l^{-1} (r^2 = 0.45)	1.15	$7.89(10)^{-3}$	$1.99(10)^{-4}$	<0.01	$1.99(10)^{-4}$ ($p \approx 0.09$)

NO_3^- loading at Harrisburg was computed on a monthly interval as the product of model-predicted NO_3^- concentration and the average discharge rate of the Susquehanna River at Harrisburg during the respective month, a simplification of the basic approach of Cohn et al. (1989). The average discharge in each month was computed from the record of daily discharge.

Results

Hypoxia in Chesapeake Bay, 1950–2001

Summer hypoxic volumes in Chesapeake Bay increased significantly and dramatically from 1950 to 2001 and also depended significantly on winter–spring (January–May) average discharge from the Susquehanna River (Tables 3 and 4, Fig. 3). At any point in the time series, expected hypoxic volume was higher in years with high winter–spring river flow (Fig. 3). Average river flow for seasonal periods other than winter–spring, such as spring (April–May; Seliger and Boggs 1988) or late winter (January–March), explained less of the variance. Incorporating additional terms in the regression model to accommodate several river flow averages also did not improve the model. Residual plots indicated that the model had approximately homoscedastic and normally distributed residuals and did not exhibit obvious lack of fit. Residuals were not autocorrelated (Durbin-Watson $D \approx 2$).

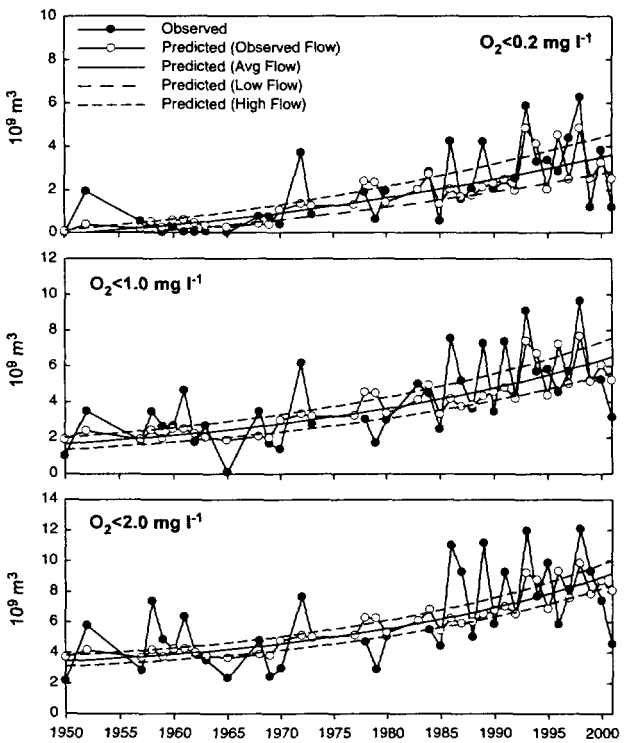


FIG. 3. Calculated summertime hypoxic volumes for Chesapeake Bay during 1950–2001 as reported in Table 3 and corresponding model predictions calculating using observed river flow (Eq. 1). Trend lines indicate model-predicted (e.g., 1) hypoxic volumes assuming low winter–spring average Susquehanna River flow (1,300 m^3 s^{-1}), average flow (1,700 m^3 s^{-1}), and high flow (2,100 m^3 s^{-1}). Note the differences in y-axis scales.

The extent and nature of the increase in hypoxic volume depended on the definition of hypoxia. Because river flow affected the extent of hypoxia at any point in time, river flow also affected percentage increases in hypoxic volume over the study period. At average winter–spring river flow during the period (1,700 m^3 s^{-1}), the expected volume of near-anoxic water (DO < 0.2 mg l^{-1}) increased from zero in 1950 to 3.6×10^9 m^3 (95% CI: 2.4×10^9 to 5.3×10^9) in 2001 (Fig. 3). The estimated volume of severely hypoxic water (DO < 1.0 mg l^{-1}) increased 3.8-fold from 1.7×10^9 m^3 (0.77×10^9 to 3.0×10^9 m^3) to 6.5×10^9 m^3 (4.6×10^9 to 9.2×10^9). The estimated volume of moderately hypoxic water (DO < 2.0 mg l^{-1}) increased 2.7-fold from 3.4×10^9 m^3 (2.1×10^9 to 5.3×10^9) to 9.2×10^9 m^3 (6.8×10^9 to 12×10^9). The volume of water affected by moderate hypoxia increased more than the volume of near-anoxic water, even though the proportional increase was smaller.

The long-term increase in hypoxic volume is expressed by the combination of the linear and quadratic trends. Statistical significance of the long-term trend was tested via the joint hypothesis that $\beta_2 = 0$ and $\beta_3 = 0$ (in Eq. 1). Statistically significant ($p < 0.01$) long-term trends were found for each level of hypoxia. A nearly linear increase was observed in near-anoxic volume. For severe and moderate hypoxia, however, the quadratic trend was an important component of the overall trend. The

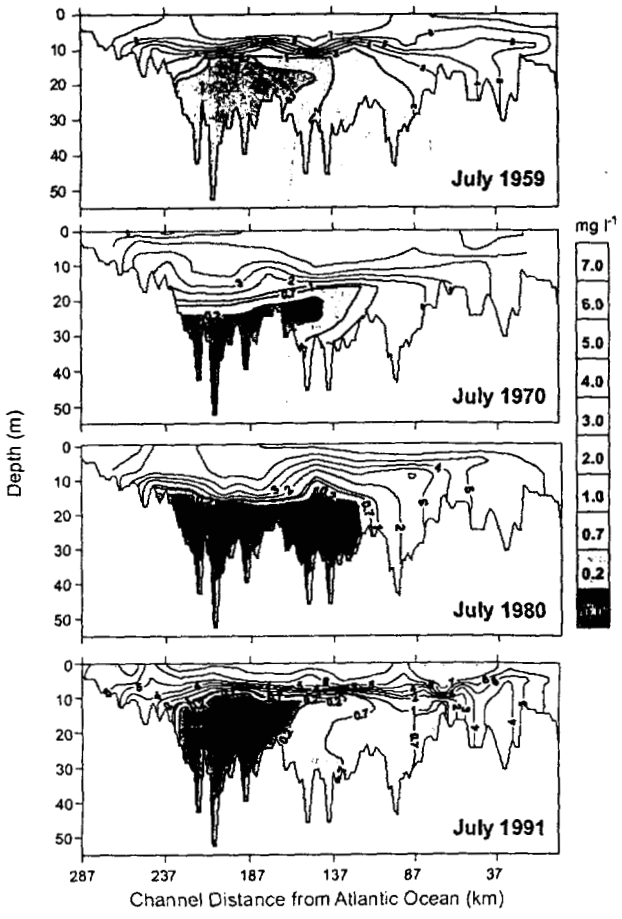


FIG. 4. Summer dissolved oxygen profiles in Chesapeake Bay during four years with near average January–May Susquehanna River flow (approximately 1,700 $m^3 s^{-1}$). Data for 1959 are from the Chesapeake Bay Institute Data Report #41 (1962c). Data for 1970 are from Taylor and Cronin (1974). Data for 1980 are from Cronin et al. (1982). Data for 1991 were obtained from the Chesapeake Bay Water Quality Monitoring Program.

rate of increase in moderate and severe hypoxia has increased in recent years (Fig. 3).

The spatial distribution of hypoxia expanded southward during 1950 through 2001 from a small area at the landward limit of the mesohaline Bay to eventually encompass the entire mesohaline Bay and a portion of the polyhaline Bay in Virginia. Summer DO profiles for years with approximately average spring river flow illustrate the pattern of change without the confounding effect of river flow variations (Fig. 4). In July 1959, the 1 mg l^{-1} DO contour intersected the channel bottom at 140 km from the Atlantic Ocean, between Point Lookout, Maryland, and the mouth of the Patuxent River (Figs. 1 and 4). There were no DO observations less than 0.2 mg l^{-1}. The presence of near anoxic or anoxic water distinguished the July 1970 DO profile from the 1959 profile. In 1980, the 1 mg l^{-1} contour extended down-estuary to approximately 100 km from the Atlantic Ocean (Smith Point, Virginia), while the 2 mg l^{-1} contour had changed little from 1959. The 0.2 mg l^{-1} contour was greatly expanded such that in most places the 0.2 mg l^{-1} contour was very close to the 1.0 mg l^{-1} contour. In 1991, both the 1.0 and 0.2 mg l^{-1} contours extended closer to the water surface in the mesohaline region as compared to 1980. Of particular note was the extension of the 1.0 and 2.0 mg l^{-1} contours to include areas further to the south, approximately 60 km from the Atlantic Ocean near the mouth of the Rappahannock River (Figs. 1 and 4). In some recent years, notably 1998 and 1999, the hypoxic zone expanded even further to the south. In 1998, a year with high winter–spring Susquehanna River flow, the 0.2 mg l^{-1} contour extended to between 60 and 240 km from the Atlantic Ocean, or from the Bay Bridge to as far south as the Rappahannock River, and included all the waters up to 10 m depth. In 1999, a year with low river flow, near-anoxic water in the mesohaline was greatly reduced, but a region with DO < 2.0 mg l^{-1} extended to within 30 km of the Atlantic Ocean, separated from the mesohaline Bay by a region of normoxic (i.e., DO > 2.0 mg l^{-1}) water. In the low flow year of 2001, the region affected by hypoxia contracted considerably to include only Maryland waters.

Although the 2-dimensional, map-view distribution of DO in bottom waters was not measured, the benthic area affected by hypoxia can be inferred from the bathymetric profile given the same assumptions used in computing hypoxic volume, namely that isopleths of DO up to 2 mg l^{-1} maintain constant depth across the channel into the littoral zone. Available cross-channel profiles indicate that this assumption is approximately valid on average, which is suitable for the purposes of this study (Buzzelli et al. 2002). During 1950 through 2001, the ratio of calculated hypoxic volume to hypoxic benthic area, essentially the mean depth of the hypoxic volume, was 5 to 7 m. This ratio did not change significantly over the long term. The area of benthic habitat affected by seasonally persistent hypoxia has increased in direct proportion to the volume of water affected by hypoxia.

Spatial and Temporal Dimensions of Hypoxia

Sufficient water quality data were available for 1985–2001 to characterize not only the extent of hypoxia in mid summer, but also development of hypoxia in late spring and decline of hypoxia in late summer or early fall. The following describes these patterns, as observed in this 17-yr record. In mid Chesapeake Bay, bottom water DO concentrations declined monotonically and linearly begin-

TABLE 5. Estimated rate of DO decline in bottom waters at station CB4.3C (Fig. 1) during the spring period of linear DO decline and the estimated date on which bottom waters reached anoxia. Bottom waters at CB4.3C became anoxic every summer and remained anoxic until early fall.

Year	Rate of DO Decline (mg l^{-1} d^{-1})	Onset of Anoxia (Date)
1985	0.103	June 20
1986	0.096	June 22
1987	0.146	May 28
1988	0.182	May 26
1989	0.093	July 5
1990	0.119	May 20
1991	0.089	June 6
1992	0.095	July 3
1993	0.184	May 21
1994	0.100	June 3
1995	0.075	July 3
1996	0.107	June 10
1997	0.085	June 28
1998	0.196	May 2
1999	0.114	June 14
2000	0.141	May 15
2001	0.121	June 2

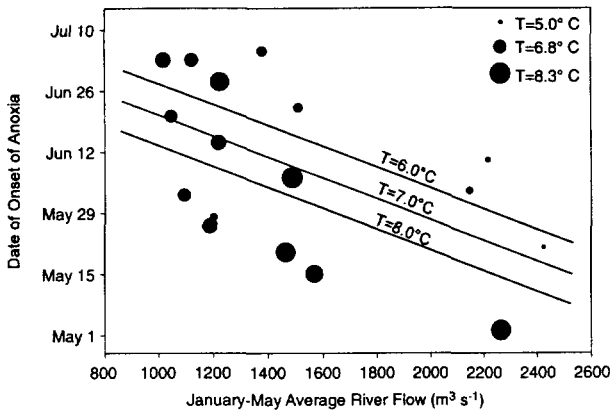


FIG. 5. The relationship between January–May average Susquehanna River flow, March–April average bottom water temperature, and the date of onset of anoxia in bottom water at station CB4.3C during 1985–2001. Plot symbol size is linearly scaled to indicate average water temperature (°C). Regression lines indicate the relationship between the date of onset of anoxia and river flow given a particular water temperature.

ning in late spring and always reached anoxia by early summer. The Chesapeake Bay Water Quality Monitoring Program reports minimum DO equal to 0.2 mg l^{-1} due to measurement uncertainty, but the presence of sulfide in the water column at these deep sites indicates a complete absence of DO (Kemp et al. 1992). While an orderly decline in bottom water DO concentration appears to have been a consistent feature of DO dynamics in the mid Chesapeake Bay for some time, having also been observed in 1938 (Newcombe et al. 1938), 1970 (Officer et al. 1984), 1977 (Taft et al. 1980), and 1979 (station 834F, EPA unpublished data), consistent depletion to anoxia appears to be a relatively new feature. Rates of DO decline and dates on which anoxia first occurred were estimated via regression (i.e., slope and x-intercept, respectively) using the 3 to 5 DO observations that described the spring decline in DO each year (Table 5). These regressions generally had $r^2 > 0.95$. The rate of spring DO depletion during 1985–2001 varied between 0.075 mg l^{-1} d^{-1} (1995) and 0.196 (1998) and averaged 0.12 mg l^{-1} d^{-1}. In an average year, depletion of bottom water oxygen from winter levels to anoxia occurred over a period of about 75 d. The linear DO decline tended to occur more rapidly when it was initiated later in spring and more slowly when it began earlier in the year. The onset of anoxia was not a simple function of either the rate of DO depletion or the date depletion was initiated. The date of onset of anoxia varied substantially, from May 2 through July 5 (Table 5), and was significantly correlated with winter–spring average river flow such that anoxia occurred earlier in the year when winter–spring river flow was above average (Fig. 5). In addition, among four years with river flow >2,000 m^3 s^{-1}, anoxia occurred much earlier when March–April average water temperature was high (e.g., 8.3°C in 1998) as compared to years when water temperature in the same period averaged <6°C (1993, 1994, and 1996). The multiple regression relating date of anoxia to river flow and early spring (March–April) average water temperature ($p < 0.05$) is:

$$D = 250 - 0.024Q - 7.4T \qquad (3)$$

where D is the date on which anoxia first occurred (expressed as number of days since December 31), Q is the January–May average river flow (m^3 s^{-1}) and T is the March–April average bottom water temperature at CB4.3C ($r^2 = 0.38$, Fig. 5). In this regression, the effect of river flow is significant ($p < 0.05$), but the effect of early-spring water temperature must be considered speculative ($p < 0.1$). The observed 1,380 m^3 s^{-1} range in average spring river flow was associated with an expected 33-d range in the date of onset of anoxia (Eq. 3). Similarly, the observed 3.2°C range in March–April average bottom water temperature (5.0–8.3°C) would be associated with a 24-d range in the date of onset of anoxia (Eq. 3).

The time-integrated hypoxic volume summarizes both the extent of hypoxic water in the Bay and the fraction of the year that hypoxic conditions persist (Fig. 6). To provide some context for interpretation of these values, it is helpful to identify the likely range that might be observed. In general, seasonally persistent hypoxia occurs only in the below-pycnocline waters of the Bay, which en-

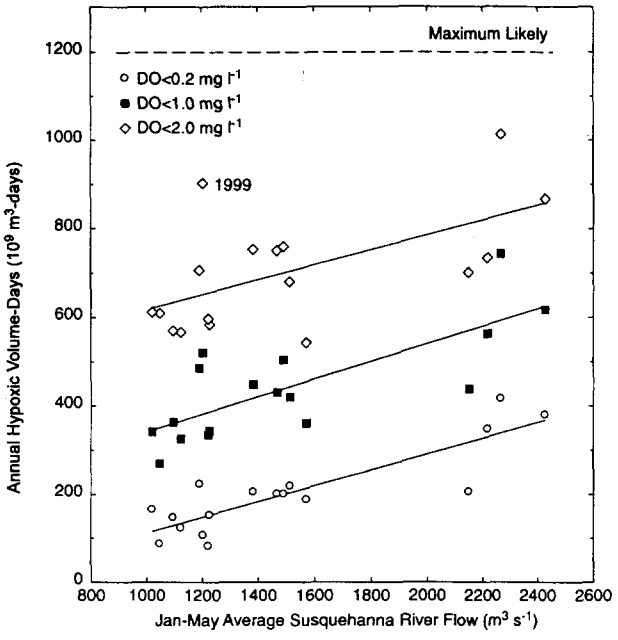


FIG. 6. The relationship between January–May average Susquehanna River flow and time-integrated hypoxia (annual hypoxic-volume-days), where hypoxia is defined as either dissolved oxygen less than 0.2, 1.0, or 2.0 mg l^{-1}. The period encompassed is 1985–2001. Hypoxia was well above the expected value in 1999. The maximum likely hypoxia refers to a scenario where hypoxia first appears in mid May and increases to affect all below-pycnocline waters in the Bay, then decreases gradually and disappears in mid September.

compass about 25% of the total volume, or 12.75 $\times$ 10^9 m^3. Hypoxia occurred most frequently from mid May to mid September, about 120 d. Hypoxic volume generally increased gradually to a mid summer maximum, then decreased gradually. This pattern can be quantified as the area under the graph of hypoxic volume versus time, roughly a half-ellipse with an area of 1.2 $\times$ 10^{12} m^3-d. This quantity reflects a likely worst-case scenario for hypoxia in Chesapeake Bay, where hypoxia affects all the below-pycnocline waters in the Bay for all of the period in which hypoxia is likely to occur. During 1985–2001, moderate hypoxia (DO < 2.0 mg l^{-1}) was 45–84% (average = 59%) of this value. Near-anoxia was 7–25% (average = 17%) of this value. The temporal and spatial extent of hypoxia was significantly greater in years with high winter–spring average river flow ($p < 0.01$, Table 6, Fig. 6). The extent of near-anoxia was most strongly related to river flow ($r^2 = 0.75$), while the extent of moderate hypoxia was more variable ($r^2 = 0.35$). Within the time series of these estimates (17 yr, as opposed to the entire time series of hypoxic volume in Fig. 3), there was no apparent increase or decrease over time in hypoxic-volume days that was not attributable to river flow.

Nitrate Concentration and Load at Harrisburg

We assembled a record of NO_3^- concentrations for the Susquehanna River at Harrisburg spanning 1903–2001. The final 57 yr of the record (1945–2001) were sufficient to estimate NO_3^- loading at Harrisburg in each year, while 9 measurements during late fall 1902 through summer 1903 provided a less certain, but still useful, perspective on NO_3^- concentrations early in the century.

River flow was above average in spring 1903, with several periods of particularly high flow. A peak NO_3^- concentration of 1.08 mg l^{-1} was observed following the first flow peak, but concentrations later in spring were 0.27–0.44 mg l^{-1}, with an overall springtime average of 0.55 mg l^{-1}. In summer and fall, concentrations averaged 0.3 mg l^{-1}. While the spring concentrations are similar to spring observations for the 1940s, the summer average is 35% less than the average in mid century.

NO_3^- concentration at Harrisburg increased during 1945 to 2001, with the extent of the increase varying according to season (Fig. 7). The increase in concentration was greatest during winter (December–February), somewhat less during spring (March–April), and least during summer (June–August). The highest concentrations appear to have occurred in the late 1980s and were particularly high in 1989, a year in which high river flow occurred in late spring. Somewhat lower concentrations prevailed in the subsequent 6 yr of the record at Harrisburg (1990–1995; Fig. 7).

NO_3^- concentrations at Harrisburg during 1995–2001 were estimated from the ratio of NO_3^- concentration at Conowingo to the concentration at Harrisburg. This ratio was as high as 2 during late summer (Fig. 8), indicating the presence of high-concentration NO_3^- sources between Harris-

TABLE 6. Regressions relating hypoxic-volume-days (10^{10} m^3-d) for Chesapeake Bay for 1985–2001 to January–May average Susquehanna River flow (m^3 s^{-1}). The three regressions reflect hypoxic volume calculated using the three different DO levels: < 0.2, 1.0, or 2.0 mg l^{-1}.

DO Threshold	Intercept (± SE)	Slope (± SE)	
< 0.2 mg l^{-1}	−6.8 (4.2)	0.018 (0.0027), $p < 0.01$	$r^2 = 0.75$, n = 17
< 1.0 mg l^{-1}	14 (6.8)	0.020 (0.0043), $p < 0.01$	$r^2 = 0.58$, n = 17
< 2.0 mg l^{-1}	45 (9.2)	0.017 (0.0059), $p = 0.012$	$r^2 = 0.35$, n = 17

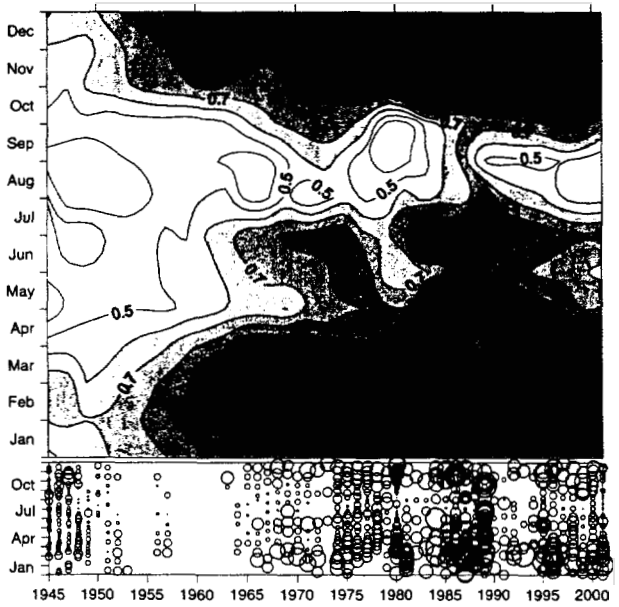


FIG. 7. Seasonal and long-term (1945–2001) distribution of nitrate concentration (mg NO_3^-N l^{-1}) at Harrisburg, Pennsylvania, illustrated via contours (upper panel) and temporal distribution of raw observations (lower panel). The size of the plot symbol in the lower panel indicates concentration. Concentrations for 1945–1995 were measured at the Harrisburg monitoring station. Concentrations for 1995–2001 were estimated from values obtained at the Conowingo monitoring station (Fig. 8).

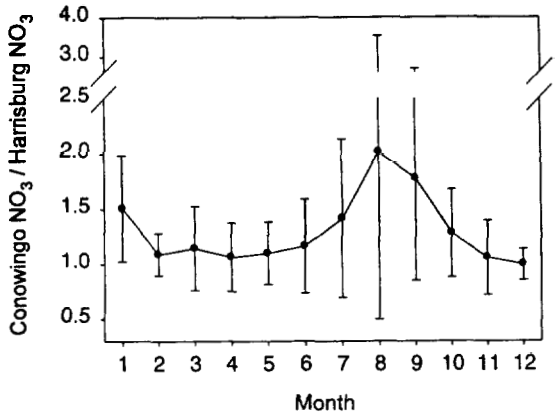


FIG. 8. The seasonal pattern in the ratio of NO_3^- concentration at Conowingo, Maryland, to NO_3^- concentration at Harrisburg, Pennsylvania, computed from the period of overlapping records (1978–1995).

burg and Conowingo and seasonally lower concentrations in the mainstem of the river (Fig. 7). Compared to summer, these sources were proportionately less important during the high flow period of November–May, when the Conowingo:Harrisburg NO_3^- ratio was only slightly greater than 1.0 (Fig. 8). NO_3^- concentrations at Harrisburg, estimated using these monthly ratios, continued the downward trend that appeared to have begun in 1990 (Fig. 7).

Very high NO_3^- concentrations in the largest gauged tributaries to the Susquehanna River between Harrisburg and Conowingo (Table 2, Fig. 2) can likely explain the increased ratio of concentrations at Conowingo to concentrations at Harrisburg during summer, when flow along the mainstem of the Susquehanna is at a seasonal minimum and concentrations are also at a seasonal minimum. While the average summer NO_3^- concentration at Harrisburg in recent years was 0.5 to 0.7 mg l^{-1}, the average of available measurements was higher for (in decreasing order of drainage area) West Conewago Creek (2.2 mg l^{-1}), Swatara Creek (3.1 mg l^{-1}), Conestoga River (6.7 mg l^{-1}), Codorus Creek (3.1 mg l^{-1}), and Muddy Creek (4.0 mg l^{-1}). These basins collectively account for 67% of the drainage area between Harrisburg and Conowingo. Average flow and NO_3^- concentrations show that during summer the confluence of the highly NO_3^--enriched Conestoga River and the Susquehanna River added only 3% to the average Susquehanna flow but increased NO_3^- concentration by up to 20%. If sufficient data were available, a similar analysis for the other major tributaries would likely account for much of the remaining difference that was observed. While an estimate of the Conowingo:Harrisburg ratio for NO_3^- was needed to estimate concentrations at Harrisburg for 1995–2001, it is worthwhile to point out that summer concentration estimates are relatively less important for addressing NO_3^- loading to the Bay because much higher winter–spring river flow and NO_3^- concentrations account for much of the annual NO_3^- load (see later discussion).

A multiple regression modeling the 1945–2001 Harrisburg NO_3^- time-series as a function of seasonally varying long-term trends (Eq. 2, Table 7) explained about half of the overall variance (r^2 = 0.54) with residuals consistent with model assump-

TABLE 7. Parameter estimates (± SE) and statistical significance for the regression parameters of Eq. 2, describing the long-term and seasonal changes in NO_3^- concentration at Harrisburg, Pennsylvania, from 1945–2000. ne = not estimated, ns = not significant.

Parameter	Estimate	Significance, $P(H_0: \beta_i = 0)$
T_0	44 (ne)	ne
β_0 (mg l^{-1})	0.47 (0.023)	<0.01
β_1 (mg l^{-1})	0.016 (0.03)	ns
β_2 (mg l^{-1})	0.13 (0.03)	<0.01
β_3 (mg l^{-1} yr^{-1})	0.014 (0.00073)	<0.01
β_4 (mg l^{-1} yr^{-1})	0.0034 (0.00096)	<0.01
β_5 (mg l^{-1} yr^{-1})	0.0046 (0.0011)	<0.01
β_6 (mg l^{-1} yr^{-1})	−0.034 (0.0038)	<0.01
β_7 (mg l^{-1} yr^{-1})	−0.017 (0.0053)	<0.01
β_8 (mg l^{-1} yr^{-1})	−0.015 (0.0054)	<0.01

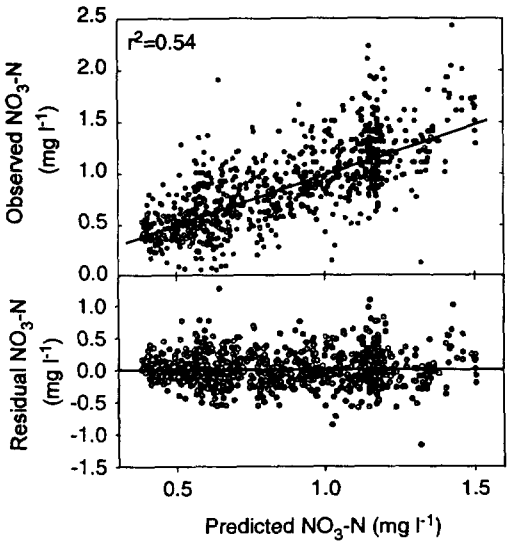


FIG. 9. Predicted (via Eq. 2) versus observed NO_3^- concentration during 1945–2001 in the Susquehanna River at Harrisburg, Pennsylvania (upper panel), and predicted NO_3^- concentration versus residual error (lower panel).

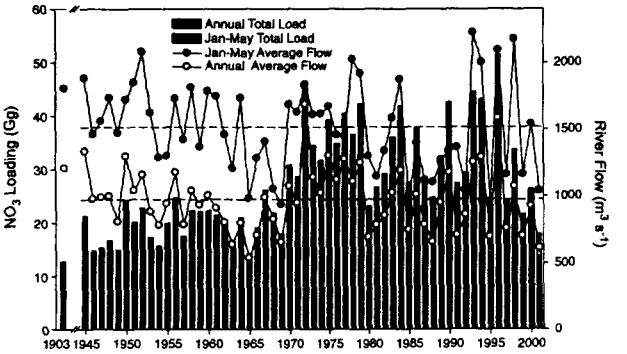


FIG. 10. January–May and annual total NO_3^- loading, and January–May and annual average flow from the Susquehanna River at Harrisburg, Pennsylvania, during 1903 and 1945–2001. Dotted lines indicate 1945–2001 means of winter–spring and annual average river flow. NO_3^- data for 1903 were obtained from Leighton (1904). Annual NO_3^- loading for 1903 could not be estimated due to insufficient data.

tions (Fig. 9). Model results suggested that average NO_3^- concentrations peaked in 1989 and provided the following estimates of average concentration and rates of change. Concentrations in 1945 averaged 0.46 mg l^{-1}, with only slightly higher values in winter–spring than in summer. Over the ensuing 44 yr, annual mean NO_3^- at Harrisburg increased at an average rate of 0.014 mg l^{-1} yr^{-1}, reaching a maximum annual mean concentration of 1.1 mg l^{-1}, a 2.4-fold increase. After 1989, concentrations decreased at a relatively more rapid rate of 0.034 mg l^{-1} yr^{-1} reaching 0.86 mg l^{-1} in 2001. Winter–spring average concentrations increased more rapidly (up to 0.02 mg l^{-1} yr^{-1}) from 1945–1989, reaching an average winter–spring concentration of 1.3 mg l^{-1}, 2.7-times 1945 levels in winter–spring. Winter–spring concentrations subsequently declined to 0.94 mg l^{-1} by 2001, remaining 2-fold greater than 1945 concentrations.

The long-term pattern of NO_3^- loading was strongly affected by interannual and interdecadal differences in river discharge. During 1945–1960, river flow varied around the long-term average. Below-average flow prevailed in most years during the 1960s (1961–1969) and 1980s (1980–1989), whereas above-average river flow prevailed during most of the 1970s (Fig. 10). Extreme high and low flow characterized the 1990s. While a relatively constant increase in average NO_3^- concentration was observed during 1945–1969, annual NO_3^- loading changed little, averaging 20 Gg yr^{-1} (Gg = 10^9 g) as river flow declined from average to drought conditions in the 1960s. During the 1970s, higher flow returned, increasing NO_3^- loading to an average of 37 Gg yr^{-1}, a nearly 2-fold increase (Fig. 10). Decadal average NO_3^- loading never subsequently exceeded this maximum. During the 1980s, as NO_3^- concentrations continued to increase, average NO_3^- loading decreased slightly to 31 Gg yr^{-1} due to lower average river flow. Despite decreasing NO_3^- concentrations in the 1990s, average NO_3^- loading increased to 33 Gg in the 1990s as several years of record river flow contributed very high loads. High river flow through most of the year in 1996 contributed to a new record NO_3^- load, exceeding the 1972 record load that resulted from the summer flood following Tropical Storm Agnes. The three highest winter–spring NO_3^- loads in 1945–2001 occurred in conjunction with floods in 1993, 1994, and 1996 (Fig. 10).

Nutrient loading by the Susquehanna River at Conowingo (Fig. 2) has been estimated since 1978 using detailed field data and well-developed estimation procedures (Langland et al. 2001). Average NO_3^- loading at Harrisburg (this study) was highly correlated with average NO_3^- loading estimates for Conowingo ($n = 20$, $p < 0.01$). We developed functional (model II) regressions relating annual mean and winter–spring NO_3^- loading at Conowingo ($L_{NO_3,C}$) to corresponding average loading rates at Harrisburg ($L_{NO_3,H}$):

Annual:

$$L_{NO_3,C} = 1.89 + 1.36L_{NO_3,H} \quad (r^2 = 0.97) \quad (4)$$

Winter–Spring:

$$L_{NO_3,C} = -0.42 + 1.39L_{NO_3,H} \quad (r^2 = 0.98) \quad (5)$$

The regression slopes, being substantially great-

er than 1, reflect the fact that river flow is 10% higher at Conowingo as compared to Harrisburg and that NO_3^- concentrations are also higher at Conowingo (Fig. 8).

NO_3^- loading accounted for 70% of TN loading at Conowingo. NO_3^- loading at Harrisburg was also strongly correlated with TN loading at Conowingo ($n = 20$, $p < 0.01$). Therefore, we developed model II regressions to relate TN loading at Conowingo ($L_{TN,C}$) to NO_3^- loading at Harrisburg ($L_{NO_3,H}$):

Annual:

$$L_{TN,C} = -0.16 + 1.99L_{NO_3,H} \quad (r^2 = 0.90) \quad (6)$$

Winter–Spring:

$$L_{TN,C} = -2.75 + 2.03L_{NO_3,H} \quad (r^2 = 0.94) \quad (7)$$

These regressions provide a means to estimate loading targets for the current monitoring location, Conowingo, on the basis of historical loading estimates for the Susquehanna River at Harrisburg.

Hypoxic Volume and Nitrate Loading

During 1950–2001, mid summer hypoxic volume in Chesapeake Bay was positively correlated with winter–spring NO_3^- loading from the Susquehanna River (Fig. 11, see Table 8 for statistical details). While this result was expected, closer examination revealed unexpected complexity. Foremost was the observation that Susquehanna River NO_3^- loading did not explain as much variability in hypoxic volumes as river flow and a long-term second-order (i.e., quadratic) trend over time (Table 4). Because Chesapeake Bay hypoxia was hypothesized to have increased in response to increased N loading, one might have expected that NO_3^- loading would predict hypoxic volumes best. Adding average river flow to regressions that included NO_3^- loading, on the theory that river flow modifies the ecosystem response to NO_3^- loading, did not improve these models ($p > 0.05$). This result can be understood in statistical terms. Whereas the modeled temporal trends included an accelerating increase in hypoxic volume, NO_3^- concentrations began to decline in 1990 in opposition to the continued increase in hypoxic volumes (Fig. 3). Models that predicted hypoxic volume from NO_3^- loading underpredicted hypoxia in the most recent years (Fig. 11).

The relationship between time and expected hypoxic volume given a fixed level of nutrient loading can be explored via the semi-partial correlation coefficient, which describes the correlation between one variable and another variable that has been corrected for its correlation with a third variable. The quantity $r_{T(V'|L_{NO_3,H})}$ quantifies the correlation between time and hypoxic volume after the effect of NO_3^- loading at Harrisburg on hypoxia has been removed. For log-transformed hypoxic volume (DO < 1.0 mg l^{-1}), $r_{T(V'|L_{NO_3,H})} = 0.47$ ($p < 0.01$), meaning that the expected hypoxic volume given a particular NO_3^- loading rate increased over time (Fig. 11). The squared semi-partial correlation coefficient, $r^2_{T(V'|L_{NO_3,H})}$, indicates that time explains an additional 22% of the vari-

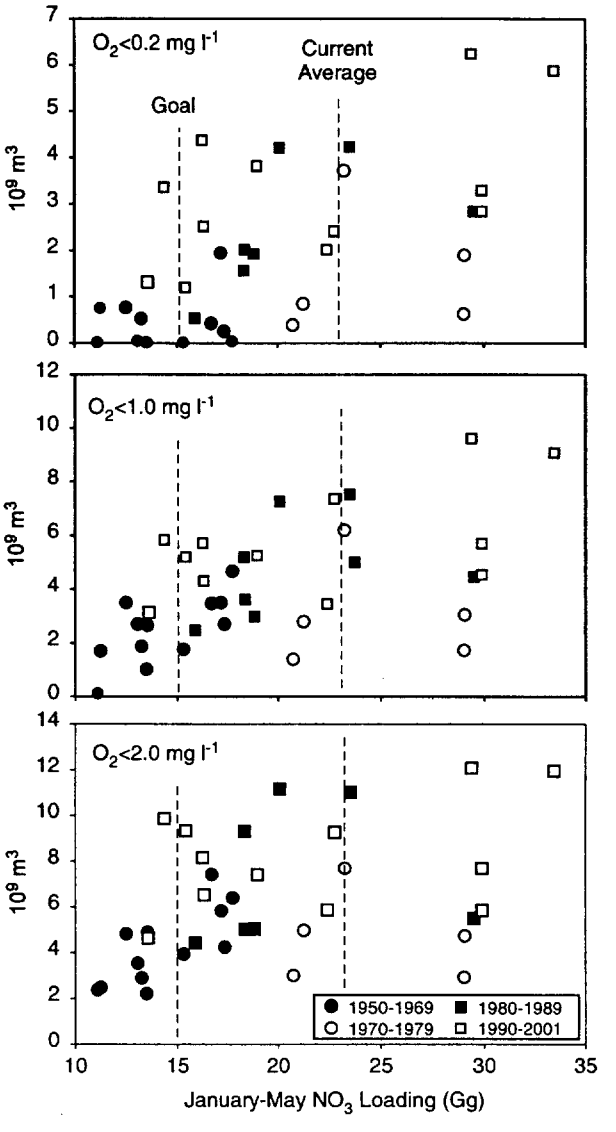


FIG. 11. The relationship between January–May total NO_3^- loading at Harrisburg, Pennsylvania, and hypoxic volume in Chesapeake Bay (1950–2001). Plot symbols indicate decadal (or longer) periods in the record (1950–1969, 1970–1979, 1980–1989, 1990–2000). Vertical lines indicate the present (1990–2001) average winter–spring NO_3^- load and the target (Goal) NO_3^- load that may be required to eliminate or nearly eliminate anoxia.

TABLE 8. Parameters of the regression $V' = (\beta_0 + \beta_2) + ; B_1L_{NO3,H} + \beta_3T$, where V' is hypoxic volume, transformed as in Eq. 5, $L_{NO3,H}$ is the winter–spring total NO_3^- loading at Harrisburg (Gg season^{-1}, 151 d), and T is years since 1949. In order to emphasize the assumed importance of NO_3^- loading, the model was fitted sequentially, by fitting $V' = \beta_0 + \beta_1L_H + \varepsilon$, then fitting $\varepsilon = \beta_2 + \beta_3T + \varepsilon'$, then substituting the second model into the first.

DO Threshold (overall r^2)	Intercept, $\beta_0 + \beta_2$	NO_3^- Load Effect, β_1 (partial r^2)	Time-dependent Residual Trend, β_3 (partial r^2)
DO < 0.2 mg l^{-1} ($r^2 = 0.62$, $p < 0.01$)	−0.88	0.060 ($r^2 = 0.36$, $p < 0.01$)	0.020 ($r^2 = 0.37$, $p < 0.01$)
DO < 1.0 mg l^{-1} ($r^2 = 0.44$, $p < 0.01$)	0.36	0.040 ($r^2 = 0.25$, $p < 0.01$)	0.013 ($r^2 = 0.22$, $p < 0.01$)
DO < 2.0 mg l^{-1} ($r^2 = 0.37$, $p < 0.01$)	1.02	0.071 ($r^2 = 0.17$, $p = 0.01$)	0.012 ($r^2 = 0.25$, $p < 0.01$)

ance in hypoxia beyond that explained by NO_3^- loading.

Semi-partial regressions quantify the increase in hypoxia expected over time given a particular N loading rate. These predict that one expects no anoxia in 1950 (i.e., T = 1) when integrated winter–spring NO_3^- loading at Harrisburg ($L_{NO_3,H}$) is <15 Gg (Table 8). In 1995 (T = 46) we expect anoxia to affect 1.3×10^9 m^3 of deep water given the same NO_3^- load. Factors other than increased NO_3^- loading account for this quantity of anoxic water. One may also say that if winter–spring NO_3^- loading were reduced to 15 Gg, that this amount of anoxia would persist today, even though the Bay would have enjoyed no anoxia at this loading rate in 1950. Expected anoxia at average flow in 1995, based on trends analysis (reflecting the actual loading rate) is 3.0×10^9 m^3. Increased NO_3^- loading accounts for 57% of the anoxia expected at average flow in 1995, while other factors must explain the additional long-term increase.

Discussion

Trends in Chesapeake Bay Hypoxia, 1950–2001

The results of this study show that the volume of water affected by hypoxia in mid summer in Chesapeake Bay increased dramatically during 1950–2001 and that this long-term trend was modulated by interannual differences in average winter–spring river flow (Tables 3 and 4, Fig. 3). The increase in hypoxic volume appears to have accelerated, especially in the case of moderate hypoxia (DO < 2.0 mg l^{-1}).

Two major features of the long-term trend are of particular interest. Extensive summer anoxia now occurs regardless of winter–spring river flow (Table 3, Fig. 3). Prior to 1968, anoxia appears to have been limited to years of high winter–spring river flow. Even in high flow years, summer anoxia was previously limited in spatial extent compared to recent years. Because of the particularly adverse consequences of anoxia, this change marked an important shift in the ecological history of Chesapeake Bay (see discussion of sedimentary record, below). The second important feature of the long-term trend is the massive expansion, largely since 1980, of the volume and area of the Bay affected by moderate hypoxia (DO < 2.0 mg l^{-1}; Tables 3 and 4, Fig. 3). Hypoxia now affects areas of the Bay that were formerly unaffected, even in high flow years. The newly affected areas include high-mesohaline and polyhaline regions of the Bay, including Virginia waters (Fig. 4). In 1999, a year characterized by severe drought in Maryland, but not in Virginia, hypoxia occurred in Virginia waters, even though hypoxia in Maryland was milder than usual. This pattern of expansion to the south corresponds directly to the long-term increase in phytoplankton biomass, in which the largest increases have occurred in the southern Bay (Harding and Perry 1997).

Earlier studies of hypoxia in Chesapeake Bay (Flemer et al. 1983; Seliger and Boggs 1988) may have reached equivocal results due to issues concerning interpolation method, inclusion or exclusion of data from individual years, definitions of hypoxia, and the means by which river flow effects were included in the analysis. This study overcomes these limitations in part through a larger data set spanning a large range of hydrologic conditions. Data since 1984 easily resolve the major spatial patterns in DO along the main axis of the Bay. We also computed and analyzed hypoxic volumes for several DO thresholds, avoiding the limitations of a single, arbitrary choice. Like the earlier studies, this study used data collected on an axial transect of the Bay that do not fully characterize the 3-dimensional pattern of hypoxia. Data collected on lateral transects of the mid Bay (U.S. Environmental Protection Agency, unpublished data) show variability in DO at constant depths across the horizontal axis of the Bay. High-resolution DO profiles collected on lateral transects using an undulating towed instrument platform also demonstrate the presence of complex, temporally variable 3-dimensional water column structure in Chesapeake Bay

(Chesapeake Bay Land Margin Ecosystem Research Program unpublished data). When evaluating only the location of DO contours less than 2.0 mg l^{-1}, the available evidence suggests that no systematic bias is introduced by computing hypoxic volumes using axial (i.e., 2-dimensional) data. This approach is preferred for continuity with the older data and because most of the lateral data can provide only a limited characterization of the complex 3-dimensional water quality structure. Most importantly, the major conclusions regarding the long-term trends in hypoxia are probably insensitive to choice of 2-dimensional or 3-dimensional analysis, or any reasonable choice of interpolation method, because the magnitude of the documented change is so large.

While the increase in hypoxia during 1950–2001 was very clear, a continued upward trend during 1985–2001 was not evident when one only considers observations from those years, the period covered by the higher-resolution Chesapeake Bay Monitoring Program data set. Within this shorter record, the only significant predictor of hypoxic volume was winter–spring average river flow, where the highest summer hypoxic volumes occurred when spring river flow was highest (Fig. 6). This raises the question of whether the long-term increase in hypoxic volume may have in fact stabilized in recent years, even as within the context of the long-term trend it appears that hypoxia continued to increase more rapidly than ever. An important factor to consider is the potential for subtle changes in trends or responses to external forcing to be resolved against a highly variable background, even with a long time series. Since much variability remains unexplained, ecological changes may not be resolved until a substantial response has occurred over a reasonably long period. In this regard, the data provide conclusive evidence for long-term change over the entire time series, but less conclusive evidence for differences in trends across subsets of the record. Decadal-scale patterns in ecological forcing appear to exacerbate this problem by introducing statistical problems. For example, average river flow increased during 1985–2001, principally due to several years of very high river flow in the 1990s (1993, 1994, 1996, 1998). As a result, long-term trends, river flow effects, and nutrient loading effects are largely confounded within this period. The longer record provides a different perspective. In the 1950–2001 time series, recent record high hypoxic volumes (e.g., 1993, 1998) contribute to the upward trend in hypoxic volume (Fig. 3) and are not explained by the high river flow in those years (Table 3). In the shorter time series (1985–2001) these extreme events drive the estimated response of hypoxia to river flow (Fig. 6). This study leaves open the possibility that trends in hypoxic volume may have changed in ways that cannot be resolved. It seems reasonable to ask whether recent observations depart significantly downward from the expectation derived from the longest time series available, rather than whether recent observations indicate a trend when considered in isolation. In this light, there does not yet appear to be substantial evidence that hypoxia has begun to stabilize or decrease. While an optimist may view the most recent observations reported in this study (1999–2001) as the genesis of a decreasing trend, such optimism may be tempered by the extensive hypoxia reported in 2003 (U.S. Environmental Protection Agency unpublished data).

Analyses of temporal trends in time series must consider the statistical independence of observations. The same question is relevant in ecological terms. Namely, do high nutrient load, river flow, and hypoxia in one year contribute to higher hypoxia in a subsequent year? Including prior year NO_3^- loading or hypoxia in regressions did not significantly improve predictions, suggesting that development of summer hypoxia does not depend substantially on hypoxia or nutrient loading in the previous year. The lack of any significant lag effect of loading or hypoxia during the 1990s, when large changes in river flow occurred in concurrent years, supports this conclusion (Figs. 3 and 6). These results do not address whether longer-term eutrophication-driven ecological change could have affected the sensitivity of Chesapeake Bay to nutrient-driven eutrophication and hypoxia. For example, benthic communities can suffer mass mortality due to anoxia and require an extended recovery period to develop mature communities that include older individuals of long-lived species. Similarly, seagrass beds recover slowly. Ecological feedback effects at these longer-time scales will not be realized at annual time scales and cannot be detected through simple autocorrelation. Such responses might be realized after several years of above-average flow (e.g., 1993–1994) or below-average flow (e.g., 1999–2001), or in response to decadal-scale patterns (e.g., dry period in late 1980s).

Early DO Data and the Sedimentary Record

Hypoxia and some anoxia were observed during summer in Chesapeake Bay between Patuxent River and Annapolis, Maryland, as early as the 1930s (Newcombe and Horne 1938; Newcombe and Lang 1939; Newcombe et al. 1939; Officer et al. 1984). At 50 m depth near Patuxent River (38°18′N, 76°25′W) bottom waters were fully oxygenated on July 7, 1936, anoxic on August 3, 1936, and hypoxic on August 10 and 20, 1936. In the

following year, moderate hypoxia (1.9 mg l^{-1}) was first observed at the same location on May 7, 1937, then DO declined to anoxia on July 29, 1937. No anoxia was observed in 1938, but hypoxia was found from May 18 through August 30. Moderate to severe hypoxia was also observed near Annapolis in 1938 (Newcombe et al. 1938), suggesting that hypoxia extended continuously between Patuxent River and Annapolis. The 1936–1938 observations of hypoxia were made over a range of spring hydrologic conditions, from above-average river flow in 1936–1937 to below-average flow in 1938, the year no anoxia was observed. Chesapeake Bay was vulnerable to summer hypoxia and some anoxia as early as the 1930s, with levels modulated by river flow. Investigators in the late 1930s recognized the extent, severity, and possible significance of summer hypoxia in Chesapeake Bay. Truitt (1937, p. 13) stated: "It has been shown that, during summer, an extensive bottom stratum of water that is very low in oxygen content extends over a wide section of the Bay." Truitt (1938, p. 9) reported that "studies are in progress which aim to show how important these so-called 'oxygen deserts' are in the economy of the Bay."

Geochemical and microfossil evidence obtained in recent years from sediment cores has provided further evidence for the long-term development of eutrophication and hypoxia in Chesapeake Bay (Cooper and Brush 1991, 1993; Helz et al. 2000; Karlsen et al. 2000; Zimmerman and Canuel 2000; Adelson et al. 2001; Colman and Bratton 2003). These studies corroborate the view assembled here and provide additional information. Studies of the sedimentary record used multiple markers for dating strata in cores and a remarkable array of markers for ecological change. These include sediment organic carbon and biogenic silica content, iron, sulfur, osmium, and molybdenum geochemistry, lipid biomarkers, and pollen, diatom, and foraminiferan microfossils. Results indicate that some ecological change began shortly following European settlement, particularly increased sedimentation (Cooper and Brush 1993; Helz et al. 2000), but that eutrophication and increased hypoxia was largely limited to the 20th century. Sulfur and lipid biomarker records show that hypoxia increased from 1934 to 1948, coincident with increased deposition of phytoplankton-derived and microbially-derived organic matter (Zimmerman and Canuel 2000). Foraminiferan fossils and molybdenum geochemistry indicate that anoxia was not prevalent until the late 1960s to early 1970s (Karlsen et al. 2000; Adelson et al. 2001), consistent with this study. Sedimentary records also confirm that anoxia was first prevalent at the landward limit of the deep-circulation of Chesapeake Bay and then expanded to the south (Adelson et al. 2001).

Seasonal Development of Hypoxia

The seasonal pattern of hypoxia and anoxia is an important aspect of DO dynamics. Late spring patterns of DO depletion and even formation of anoxia does not appear to have changed dramatically. DO declined in spring much the same in the late 1930s as recently (Newcombe and Horne 1938; Newcombe et al. 1939; Officer et al. 1984), sometimes reaching hypoxia or anoxia during May. Once reaching anoxia DO subsequently increased within a short period, whereas today anoxia persists throughout the summer. Weekly DO measurements at a Patuxent River site in 1944–1946 (Beaven 1947) showed that DO declined to anoxia on June 1, 1944, but increased the following week and continued to increase each week for the remainder of the summer. Adequate organic matter has been available in late spring to support DO depletion since at least the 1930s. Given increased organic matter production in recent years, oxygen demand in the mid Bay is probably no more substrate-limited in spring than in earlier years. The surprising 2-mo variability in the timing of DO depletion is most likely regulated principally by physical factors: water column stratification and water temperature (Fig. 5). Historical recovery of DO during summer suggests that metabolic rates became substrate limited by early summer (Beaven 1947). Even in the modern eutrophic Bay, the seasonal maximum sediment oxygen demand precedes the seasonal temperature maximum (Kemp and Boynton 1992). Summer rates remain limited by the quantity and quality of organic substrates, but the extent of substrate limitation is clearly less than in earlier years. The similarity between springtime DO dynamics in the 1930s and the modern Bay may be attributed to similarity in physical controls, while the dramatic differences in the persistence and extent of hypoxia and anoxia is likely attributable to nutrient enrichment and the resulting ecological changes.

Spatial patterns in DO in summer, rather than temporal patterns in spring are the best indicator of eutrophication-related changes in anoxia, particularly for tracking historical developments and the response of hypoxia to management. Springtime DO depletion may change in response to large-scale climate cycles or global warming, which is expected to bring warmer winter temperatures and increased winter–spring river discharge to the mid Atlantic region (Najjar 1999). Following a warm, wet winter, hypoxia may occur as much as 2 mo earlier than in years with low winter–spring river flow and low springtime temperatures (Fig. 5).

Significance of Increased Hypoxia

Hypoxia on the northwest shelf of the Black Sea has been blamed for eliminating 100–200 tons of benthic macrofauna and demersal fishes km^{-2} and with reducing sturgeon and turbot catches (Zaitsev 1992). No studies have shown that a decline in commercial fisheries in Chesapeake Bay has occurred as a direct result of increased hypoxia and anoxia, although a modeling study has quantified the extent to which food web shifts associated with hypoxia may limit food resources available to demersal fish (Hagy 2002; Baird et al. In press). Simpler measures also show the significance of increased Chesapeake Bay hypoxia. In an average flow year around 1950 (as predicted by modeled trends) severe hypoxia (DO < 1.0 mg l^{-1}) affected 38% of the below-pycnocline volume of the mid Bay and 13% of below-pycnocline waters Baywide. By 2001, hypoxia affected more than 100% of mid Bay below pycnocline waters (i.e., hypoxia spilled into the lower Bay) and 52% of below-pycnocline waters Baywide. Hypoxia affected a much smaller fraction of the overall mainstem Bay volume (i.e., including the surface layer). In the early 1950s, only 3% was affected, while this amount increased to 13% by 2001. While a relatively small fraction of the overall Bay volume is affected by hypoxia, hypoxia may be very important in the mid Bay.

Benthic and pelagic habitats at all depths may have had adequate oxygen to remain productive as late as the 1950s, even if deeper benthic habitats were limited to hypoxia-tolerant species and subject to periodic hypoxia-induced mortality in high flow years (e.g., 1952). By 1980, deep-water macrobenthos was likely absent in the mid Bay. The presence of near-anoxic water near the pycnocline (Fig. 6) may have increased the impact of aperiodic events bringing below-pycnocline waters onto shallower flanks of the Bay (Breitburg 1990; Sanford et al. 1990). While benthic communities can potentially survive moderate hypoxia until normoxic conditions return by using a variety of adaptive strategies (Diaz et al. 1992), mortality is more rapid under severe hypoxia and especially in the presence of hydrogen sulfide (Diaz and Rosenberg 1995). While aperiodic events bringing below-pycnocline waters into shallower habitats have probably always occurred, the detrimental effects of these events may now be much greater due to anoxia. This mechanism could account for more degraded benthos in shallow normoxic areas adjacent to hypoxic areas (Hagy 2002).

Increased anoxia has affected the biogeochemistry of the Bay, perhaps accounting in part for the accelerating increase in hypoxic volume. The P efflux from anoxic mesohaline sediments in mesohaline Chesapeake Bay is 50–150 μmol m^{-2} h^{-1}, while P efflux from oxic sediments is apparently negligible (Cowan and Boynton 1996). For the estimated anoxic sediment area in the 1990s, 5.7×10^8 m^2, this amounts to an additional $2.1–6.3 \times 10^4$ kg P d^{-1}, ~3–10 times recent annual average P loading rates from Susquehanna River to Chesapeake Bay. Anoxia prevents coupled nitrification-denitrification, which occurs at significant rates in warm, organic rich, but oxic sediments (Seitzinger 1988). Summertime ammonia (NH_4^+) efflux from the mesohaline Bay sediments in recent years was substantially greater than elsewhere in the Bay (Cowan and Boynton 1996). Assuming a reasonable potential for summertime denitrification is 50–100 μmol m^{-2} h^{-1} (Seitzinger 1988), the additional NH_4^+ efflux associated with anoxia amounts to 9.6×10^3 to 1.9×10^4 kg N d^{-1}, or 10% of the annual average and 11–23% of summer average Susquehanna River TN loading rates for 1984–1996. Both N and P fluxed from sediments to the lower water column are isolated from the phytoplankton community by the pycnocline, but transfer to the euphotic zone occurs through upwelling, limited turbulent diffusion across the pycnocline, and transverse circulation (Malone et al. 1986). Regenerated N attributable to anoxia may be especially important due to the close spatial proximity of this source to the N-limited phytoplankton community (Fisher et al. 1992) relative to more distant riverine sources.

Historical Trends in Nitrogen Loading

The NO_3^- record beginning in 1945 describes a nearly linear long-term increase in NO_3^- concentration at Harrisburg from 1945 to 1989, when concentrations began to decline (Table 7, Fig. 7). We examined how changes in N inputs to the watershed may have contributed to this trend, using an N budget for the 1990s (Boyer et al. 2002) as a model for the analysis. Boyer et al. (2002) concluded that the largest sources of N are presently atmospheric N deposition (27%), N fixation in agricultural lands (28%), and imported food and animal feed (26%). Other sources, including N fertilizer application (15%), account for a smaller fraction of inputs. Data we assembled reveal that N fertilizer application and atmospheric N deposition increased significantly (Fig. 12) and likely contributed to increased N export. Agricultural land uses decreased 50% (Fig. 12). An initial analysis suggests that crops (or pasture) that fix little or no N (e.g., pasture, corn, wheat) decreased, while N-fixing crops (e.g., alfalfa hay, soybeans) may have increased (U.S. Department of Agriculture, unpublished data). Agricultural N fixation appears to have changed very little. We did not

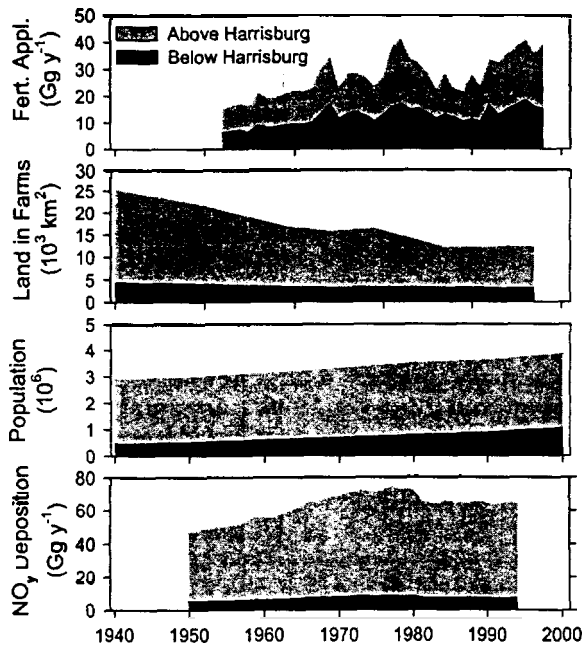


FIG. 12. Commercial nitrogen fertilizer use, farm land, including cultivated land and pasture, human population, and atmospheric deposition of nitrogen oxides. Stacked areas indicate total for the entire Susquehanna River watershed. Land in farms and fertilizer use for above Harrisburg exclude the fraction of the watershed in New York, about 25% of the watershed above Harrisburg. Apparent decline in fertilizer use from 1982–1992 may reflect systematic underreporting of fertilizer sales during this period. Sources: Pennsylvania Department of Agriculture (1963–1989, 1990–1997, 1998–1999), U.S. Census Bureau (1952, 1961, 1972, 1981, 1989, 2003a, 2003b); U.S. Department of Agriculture (1999). Atmospheric NO_y deposition computed from NO_x emissions and regression model from Jaworski et al. (1997).

attempt the complex computation of net importation of food and feed (Boyer et al. 2002), however, we examined trends in the human population (Fig. 12) and animal populations (not shown) to estimate the change in their dietary N needs. While the composition of the animal population changed (e.g., fewer dairy cows, more hogs and chickens), the aggregate dietary N requirements changed very little, provided that the estimates of per capita N requirements used by Boyer et al. (2002) are valid for 1950. The increase in dietary N requirements of the human population is small relative to other changes (assuming 5 kg person^{-1} yr^{-1}; Boyer et al. 2002). We suspect that, given decreased pasture in the watershed and national trends toward the use of animal feeding operations and imported feed (National Research Council 2000), the current rate of importation of animal feed (Boyer et al. 2002) represents a substantial increase relative to 1950. Further work is needed to reduce uncertainty and reach more quantitative conclusions. It appears that atmospheric N deposition and fertilizer application both contributed significant increases in N inputs to the watershed since 1950. We are less certain of the trends in importation of food and feed; this could have contributed a larger increase in N inputs than the other N sources combined.

From our NO_3^- loading estimates (Fig. 10) and our Eq. 6, we computed that the streamflow export of TN from the Susquehanna watershed in 1950 was approximately 30 Gg yr^{-1}, about 40% of the estimate of Boyer et al. (2002) for the 1990s. Assuming initially that among the N inputs to the watershed only atmospheric NO_y deposition and N fertilizer application (Fig. 12) were different in 1950 from current estimates (Boyer et al. 2002), we compute that the watershed exported only 12% of TN inputs via the river. If net food and feed importation was zero in 1950, this figure increases to 16%. Both estimates are much less than the current 23% export (Boyer et al. 2002). The long-term increase in N export appears to reflect both increased N inputs to the watershed and a substantial decrease in the fraction of N inputs stored or lost within the watershed.

The decline in N concentrations during the 1990s likely reflects as many factors as the long-term increase, potentially including decreased atmospheric deposition since 1980 (Fig. 12). A detailed study of changes in the watershed during this period attribute decreased N concentrations to implementation of agricultural best management practices, especially in the lower watershed, which help prevent loss of N from the land to surface waters (Sprague et al. 2000).

Nitrogen Loading and Chesapeake Bay Hypoxia

The long-term increase in hypoxia occurred concurrently with a long-term increase in NO_3^- loading (Figs. 3 and 10) and chlorophyll *a* concentrations (Harding and Perry 1997). Substantial levels of anoxia first appeared in 1970 (Fig. 3), coincident with the dramatic increase in NO_3^- loading associated with a return to high river flow levels (Fig. 10). Correlations between NO_3^- loading and hypoxic volumes (Fig. 11) further illustrate the broad relationship between NO_3^- loading and hypoxia over the past 50 yr. In view of known mechanisms relating N loading to patterns of eutrophication and hypoxia, one may infer that N loading contributed to eutrophication and the long-term increase in hypoxia in Chesapeake Bay, a pattern widely observed elsewhere (Boesch 2002).

The observed relationships between NO_3^- loading and hypoxia presented some unexpected results. The degree of correlation is relatively low, although statistically significant. While this may be

seen as undermining the value of the relationships as predictive tools, one must recognize the inherent variability of hypoxia. For example, hypoxia in 1996, the year with the highest loading rate in the record (Fig. 10), was less extensive than in either the low flow year preceding it (1995) or the flood year 2 yr later (1998, Fig. 3). Although complex water quality simulation models may be able to better account for the differences between these years than simpler models, it is questionable whether such simulations can accurately predict all the changes that might occur to the ecosystem if loading were returned to 1950s loading levels. These retrospective relationships provide a valuable addition to the manager's toolbox, which may be used to suggest loading targets or to corroborate or qualify conclusions reached through other means.

Another unexpected result was that river flow did not account for additional unexplained variance in the relationship between NO_3^- loading and hypoxia. We had expected that an extended record of NO_3^- loading would allow us to resolve physically-controlled (i.e., via stratification) vs. biologically controlled (i.e. via nutrient loading) mechanisms by which river flow affects hypoxia. We hypothesized that less hypoxia would occur when a given N loading rate occurred at low river flow (late in the record) versus average or high river flow (earlier in the record). We found instead that when effects associated with NO_3^- loading were explained, river flow did not explain further differences. Moreover, at a given NO_3^- loading rate, more hypoxia occurred late in the record (Table 8, Fig. 11). Even as NO_3^- concentrations decreased during the 1990s (Fig. 7; Sprague et al. 2000), hypoxia increased in both high and low flow years relative to the 1980s.

We examined and discounted several alternative explanations. We considered whether NO_3^- or TN loading from the Susquehanna River to the Bay at Conowingo increased, while loading and concentration at Harrisburg decreased. Our results and those of Sprague et al. (2000) confirmed that changes in loading were similar at both sites since 1978. We also considered the possibility that N loading from other sources increased total loading, while loading from the Susquehanna basin, the largest source, was stable. We also found that this was unlikely. TN loading to the Maryland mainstem Bay in recent years is ~80 Gg yr^{-1} (Boynton et al. 1995), of which the Susquehanna River contributes approximately 60 Gg yr^{-1} (computed from U.S. Geological Survey estimates, Langland et al. 2001), or 75%. While changes to other sources could have a significant effect, they would have to be proportionately large to have a substantial effect on Chesapeake Bay. The decade of the 1990s was a period of rapid economic development and population growth, particularly in the suburban counties surrounding Washington, D.C. and Baltimore. In 9 suburban counties in this area, population grew by 646,000 or 20% (U.S. Census Bureau unpublished data). N loading from the major western shore tributaries was either unchanged or decreased (Sprague et al. 2000). In the Potomac River basin, increases in loads from urban sources were offset by reductions from agricultural sources while in the smaller Patuxent River basin, large reductions in point source loadings reduced the fall line N load by up to 66% (Sprague et al. 2000). Atmospheric N inputs directly to the surface of the Bay most likely contribute <10% of the overall input (Boynton et al. 1995; Wang et al. 1997). Although estimated changes in deposition directly to the Bay were not found, estimates for watershed areas immediately surrounding the Bay suggest any increase in atmospheric N loading is limited to <5% (Sprague et al. 2000), limiting the effect on overall loading to <1%.

A remaining hypothesis, which cannot be discounted, is that the repeated occurrence of very high flow and N loading (1993, 1994, 1996, 1998) led to a persistent increase in the susceptibility of the Bay to eutrophication and hypoxia. Such changes may explain the long-term pattern of increased hypoxia relative to N loading (Table 8, Fig. 11). Increased susceptibility could result from loss of filtration capacity associated with the now depauperate benthic communities in the mid Bay (Hagy 2002) and the near elimination of oyster populations from the mainstem Bay (Smith 2001). Submerged aquatic vegetation, known to trap nutrients and sediment in shallow littoral zones, has also declined significantly in the period since 1965 (Orth and Moore 1984), potentially contributing to increased susceptibility of the Bay to hypoxia.

Changes in the stoichiometric ratios of nutrients may also have contributed to eutrophication. For example, while dissolved silica has not increased in rivers across the world, N and P have increased (Justic et al. 1995). Such changes favor increased phytoplankton production in formerly N or P limited systems and increases the potential for silica limitation. The latter may promote noxious nondiatom phytoplankton or contribute to increased sinking of diatoms (Conley and Malone 1992).

Although the long-term pattern of P loading could not be documented, loading likely increased from historic levels until actions were taken to reduce loads (Boynton et al. 1995). Management action in the early 1980s reduced TP and orthophosphate (PO_4^{3-} or DIP) loading from the Susquehanna River dramatically (Sprague et al. 2000;

Langland et al. 2001) as N loading continued to increase. As a result, the average dissolved inorganic nitrogen:dissolved inorganic phosphorus (DIN:DIP) loading ratio (atomic) increased from 120 in 1979 to 350 during 1988–1998. The period of highest DIN:DIP loading ratio corresponded to the period of most extensive hypoxia. We hypothesize that increased DIN:DIP ratio could affect the spatial and temporal distribution of phytoplankton production, affecting the subsequent response of hypoxia. Increased P limitation would likely exacerbate P limitation in the freshwater reaches of the Bay (Fisher et al. 1992) and promote increased N concentration and primary production down-estuary, a pattern observed by Harding and Perry (1997). The ecological consequences of effecting large changes in the ratios of nutrients through management actions warrant further investigation.

Restoration and Loading Targets

The long-term record of NO_3^- loading and hypoxia assembled here affords the opportunity to suggest a N loading target that, if attained, should lead to acceptable levels of hypoxia. To devise such a loading target first requires a definition of a reasonable hypoxia reduction goal. Given that hypoxia has been present in some form for much of the 20th century, the goal of completely eliminating hypoxia from deep waters of the Bay is probably unreasonable. Due to the particularly severe ecological consequences of anoxia, including geochemical feedbacks contributing to eutrophication, eliminating or nearly eliminating anoxia is a desirable goal that from an historical perspective may also be achievable. Water quality criteria for DO for Chesapeake Bay require DO > 1 mg l^{-1} even in the deepest waters, but implementation guidelines recommend considering extreme events when evaluating possible nonattainment (EPA 2003). This goal is consistent with DO conditions observed during 1950–1970. During this time, winter–spring NO_3^- loading averaged 15 Gg with annual NO_3^- loading averaging 20 Gg yr^{-1}. During 1970–2000, winter–spring and annual NO_3^- loading averaged 22 Gg and 34 Gg yr^{-1}, respectively (Fig. 11). The historical level of NO_3^- loading at which anoxia was relatively uncommon may be a reasonable N loading goal (Fig. 11). This would represent a reduction of current winter–spring NO_3^- loading by 32% and annual NO_3^- loading by 41%. Based on the relationships between NO_3^- loading at Harrisburg and TN loading at Conowingo (Eqs. 10 and 11), the target levels for TN loading at Conowingo would be 28 Gg for winter–spring TN loading and 40 Gg yr^{-1} for annual TN loading. Assuming TN loading from all sources to the Maryland mainstem Bay increased in proportion to Susquehanna River TN loading, proportional reductions would be needed to achieve the hypoxia reduction target. Given the rate of TN loading of 81 Gg yr^{-1} estimated for the Maryland mainstem Bay by Boynton et al. (1995) and the target 41% reduction, the TN load from all sources to this portion of the Bay should be reduced to 48 Gg yr^{-1}. This N loading reduction target is surprisingly close to the goal set forth in the 1987 Chesapeake Bay Agreement (EPA 1987, p. 3) to "achieve by the year 2000 at least a 40% reduction of nitrogen and phosphorus entering the main stem of the Chesapeake Bay." Note that this reduction goal is for the total load, not the controllable load as was proposed in the years following the 1987 Chesapeake Bay Agreement.

This study shows that while hypoxia was related over the long term to nutrient loading, interannual variability associated with river flow, climate, and weather patterns, and perhaps internal ecosystem dynamics, is considerable. In response to protracted nutrient enrichment, the ecosystem may have become more vulnerable to development of hypoxia and anoxia (e.g., Fig. 11). Restoration must proceed with persistence. Suggestions that current levels of hypoxia and anoxia are a natural feature of Chesapeake Bay are demonstrably false. Assertions that hypoxia does not have significant detrimental effects on living resources in the Bay are also dubious (Hagy 2002).

In upcoming years, restoration efforts will face mounting challenges regarding management of hypoxia. Not only do increasing human pressures on the landscape make it difficult to limit nutrient loading rates, climate change may also affect hypoxia in Chesapeake Bay. Najjar (1999) concluded that annual discharge from the Susquehanna River could be expected to increase 24% in response to an expected doubling of atmospheric CO_2 and associated global warming. This study shows that both increased river flow and warmer winter water temperatures hasten the formation of anoxia in Chesapeake Bay during late spring and promote the maintenance of hypoxia during summer. Innovative and aggressive environmental management efforts will be needed to achieve needed nutrient loading reductions.

Acknowledgments

This manuscript was derived in part from the Ph.D. dissertation of J. D. Hagy, submitted to the University of Maryland College Park. We are grateful to Ph.D. committee members W. M. Kemp, T. J. Miller, E. Russek-Cohen, and R. E. Ulanowicz for helpful discussions and for reviews of the dissertation. We are grateful for the assistance of C. Peets of the U.S. Department of Agriculture, National Agricultural Statistics Service library, N. Collins and J. Breitsman of the Pennsylvania Department of Agriculture, and C. V. Miller of the USGS in Baltimore. R. Greene

and N. Rabalais provided helpful reviews of late drafts of the manuscript. Two anonymous reviewers provided extensive and very helpful comments that significantly improved the final manuscript. This research was supported by the EPA Chesapeake Bay Program and a grant from the National Science Foundation Land-Margin Ecosystem Research Program (DEB-9412113). This is contribution number GED-03-2494 of the U.S. Environmental Protection Agency, National Health and Environmental Effects Laboratory, Gulf Ecology Division, Gulf Breeze, Florida and contribution number UMCES 3777 of the University of Maryland Center for Environmental Science.

Literature Cited

Adelson, J. M., G. R. Helz, and C. V. Miller. 2001. Reconstructing the rise of recent coastal anoxia; molybdenum in Chesapeake Bay sediments. *Geochimica et Cosmochimica Acta* 65: 237–252.

Baird, D., R. R. Christian, C. H. Peterson, and G. A. Johnson. In press. Consequences of hypoxia on estuarine food web and ecosystem function: Energy diversion from consumers to microbes. *Ecological Applications*

Beaven, G. F. 1947. Hydrographic data at Sandy Point, Patuxent River, Maryland, 1942–1947. CBL Reference Series 47-1. Chesapeake Biological Laboratory, Solomons, Maryland.

Boesch, D. F. 2002. Challenges and opportunities for science in reducing nutrient over-enrichment of coastal ecosystems. *Estuaries* 25:886–900.

Boicourt, W. C. 1992. Influences of circulation processes on dissolved oxygen in the Chesapeake Bay, p.7–59. *In* D. E. Smith, M. Leffler, and G. Mackiernan (eds.), Oxygen Dynamics in the Chesapeake Bay. A Synthesis of Recent Research. Maryland Sea Grant College, College Park, Maryland.

Boyer, E. W., C. L. Goodale, N. A. Jaworski, and R. W. Howarth. 2002. Anthropogenic nitrogen sources and relationships to riverine export in the northeastern U.S.A. *Biogeochemistry* 57/58:137–169.

Boynton, W. R., J. H. Garber, R. Summers, and W. M. Kemp. 1995. Inputs, transformations, and transport of nitrogen and phosphorus in Chesapeake Bay and selected tributaries. *Estuaries* 18:285–314.

Boynton, W. R. and W. M. Kemp. 2000. Influence of river flow and nutrient loads on selected ecosystem processes. A synthesis of Chesapeake Bay data, p. 269–298. *In* J. E. Hobbie (ed.), Estuarine Science: A Synthetic Approach to Research and Practice. Island Press, Washington, D.C.

Breitburg, D. L. 1990. Nearshore hypoxia in the Chesapeake Bay: Patterns and relationships among physical factors. *Estuarine, Coastal and Shelf Science* 30:593–609.

Breitburg, D. 2002. Effects of hypoxia, and the balance between hypoxia and enrichment, on coastal fishes and fisheries. *Estuaries* 25:767–781.

Breitburg, D. L., T. Loher, C. A. Pacey, and A. Gerstein. 1997. Varying effects of low dissolved oxygen on trophic interactions in an estuarine food web. *Ecological Monographs* 67:489–507.

Buzzelli, C. P., R. A. Luettich, Jr., S. P. Powers, C. H. Peterson, J. E. McNinch, J. L. Pinckney, and H. W. Paerl. 2002. Estimating the spatial extent of bottom-water hypoxia and habitat degradation in a shallow estuary. *Marine Ecology Progress Series* 230:103–112.

Carpenter, J. H. 1965a. The Chesapeake Bay Institute technique for the Winkler dissolved oxygen method. *Limnology and Oceanography* 10:141–143.

Carpenter, J. H. 1965b. The accuracy of the Winkler method for dissolved oxygen analysis. *Limnology and Oceanography* 10: 135–140.

Chesapeake Bay Institute. 1950. Cruises 5 and 6, May 20, 1950–May 25, 1950; July 14, 1950–July 19, 1950. Data Report 4. Chesapeake Bay Institute, The Johns Hopkins University, Baltimore, Maryland.

Chesapeake Bay Institute. 1954. Cruise 12, July 15–August 6, 1952. Reference 54-5. Data Report 18. Chesapeake Bay Institute, The Johns Hopkins University, Baltimore, Maryland.

Chesapeake Bay Institute. 1962a. Bay Cruise 14, July 23–26, 1957. Reference 62-1. Data Report 33. Chesapeake Bay Institute, The Johns Hopkins University, Baltimore, Maryland.

Chesapeake Bay Institute. 1962b. Bay Cruise 18, August 6–22, 1958. Reference 62-6. Data Report 38. Chesapeake Bay Institute, The Johns Hopkins University, Baltimore, Maryland.

Chesapeake Bay Institute. 1962c. Bay Cruise 21, July 6–17, 1959. Reference 62-9. Data Report 41. Chesapeake Bay Institute, The Johns Hopkins University, Baltimore, Maryland.

Chesapeake Bay Institute. 1962d. Bay Cruise 24, August 22–September 9, 1960. Reference 62-12. Data Report 44. Chesapeake Bay Institute, The Johns Hopkins University, Baltimore, Maryland.

Chesapeake Bay Institute. 1962e. Bay Cruise 27, July 19–August 1, 1961. Reference 62-15. Data Report 47. Chesapeake Bay Institute, The Johns Hopkins University, Baltimore, Maryland.

Chesapeake Bay Institute. 1962f. Bay Cruise 28, July 24–August 7, 1962. Reference 62-16. Data Report 48. Chesapeake Bay Institute, The Johns Hopkins University, Baltimore, Maryland.

Chesapeake Bay Institute. 1963. Bay Cruise 29, March 11–19, 1963; Bay Cruise 30, July 30–September 11, 1963; Bay Cruise 31, October 21–24, 1963. Reference 63-3. Data Report 50. Chesapeake Bay Institute, The Johns Hopkins University, Baltimore, Maryland.

Cohn, T. A., L. L. DeLong, E. J. Gilroy, R. M. Hirsch, and D. K. Wells. 1989. Estimating constituent loads. *Water Resources Research* 25:937–942.

Colman, S. M. and J. F. Bratton. 2003. Anthropogenically induced changes in sediment and biogenic silica fluxes in Chesapeake Bay. *Geology* 31:71–74.

Conley, D. J. and T. C. Malone. 1992. Annual cycle of dissolved silicate in Chesapeake Bay: Implications for the production and fate of phytoplankton biomass. *Marine Ecology Progress Series* 81:121–128.

Cooper, S. R. and G. S. Brush. 1991. Long-term history of Chesapeake Bay anoxia. *Science* 254:992–996.

Cooper, S. R. and G. S. Brush. 1993. A 2,500-year history of anoxia and eutrophication in Chesapeake Bay. *Estuaries* 16: 617–626.

Cowan, J. L. W. and W. R. Boynton. 1996. Sediment-water oxygen and nutrient exchanges along the longitudinal axis of Chesapeake Bay: Seasonal patterns, controlling factors and ecological significance. *Estuaries* 19:562–580.

Cronin, W. B. and D. W. Pritchard. 1975. Additional statistics on the dimensions of Chesapeake Bay and its tributaries: Cross-section widths and segment volumes per meter depth. Reference 75-3. Special Report 42. Chesapeake Bay Institute, The Johns Hopkins University, Baltimore, Maryland.

Cronin, W. B., W. R. Taylor, and M. Mallonee. 1982. Suspended sediment distribution and related data September 1978–October 1980. Chesapeake Bay Institute Open File Report 27. Chesapeake Bay Institute, The Johns Hopkins University, Baltimore, Maryland.

de Jonge, V. N., W. Boynton, C. F. D'Elia, R. Elmgren, and B. L. Welsh. 1994. Responses to developments in eutrophication in four different North Atlantic estuarine systems, p. 179–196. *In* K. R. Dyer and R. J. Orth (eds.), Changes in Fluxes in Estuaries: Implications from Science to Management. Olsen and Olsen, Fredensborg, Denmark.

Diaz, R. J., R. J. Neubauer, L. C. Schaffner, L. Pihl, and S. P. Baden. 1992. Continuous monitoring of dissolved oxygen in an estuary experiencing periodic hypoxia and the effect of

hypoxia on macrobenthos and fish. *Science of the Total Environment, Supplement 1992* 1055–1068.

DIAZ, R. J. AND R. ROSENBERG. 1995. Marine benthic hypoxia: A review of its ecological effects and the behavioral responses of benthic macrofauna. *Oceanography and Marine Biology Annual Review* 33:245–303.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1979. Methods for chemical analysis of water and wastes, 1979. EPA-600/4-79-020. U.S. Environmental Protection Agency, Environmental Monitoring and Support Laboratory, Cincinnati, Ohio.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1987. 1987 Chesapeake Bay Agreement. U.S. Environmental Protection Agency, Chesapeake Bay Program Office, Annapolis, Maryland.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1993. Guide to using the Chesapeake Bay Program Water Quality Monitoring Data. U.S. Environmental Protection Agency, Chesapeake Bay Program Office, Annapolis, Maryland.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 2000. Chesapeake 2000. U.S. Environmental Protection Agency, Chesapeake Bay Program Office, Annapolis, Maryland.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 2002. The State of the Chesapeake Bay. A Report to the Citizens of the Bay Region. U.S. Environmental Protection Agency, Annapolis, Maryland.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 2003. Ambient water quality criteria for dissolved oxygen, water clarity and chlorophyll-*a* for Chesapeake Bay and its tidal tributaries. U.S. Environmental Protection Agency Region III Chesapeake Bay Program Office, Annapolis, Maryland, Region III Water Protection Division, Philadelphia, Pennsylvania, and Office of Water, Office of Science and Technology, Washington, D.C.

FENCHEL, T. AND T. H. BLACKBURN. 1979. Bacteria and Mineral Cycling. Academic Press, New York.

FISHER, T. R., E. R. PEELE, J. W. AMMERMAN, AND L. W. HARDING, JR. 1992. Nutrient limitation of phytoplankton in Chesapeake Bay. *Marine Ecology Progress Series* 82:51–63.

FLEMER, D. A., G. B. MACKIERNAN, W. NEHLSEN, V. K. TIPPIE, R. B. BIGGS, D. BLAYLOCK, N. H. BURGER, L. C. DAVIDSON, D. HABERMAN, K. S. PRICE, AND J. L. TAFT. 1983. Chesapeake Bay: A profile of environmental change. U.S. Environmental Protection Agency, Chesapeake Bay Program, Annapolis, Maryland.

HAGY, J. D. 2002. Eutrophication, hypoxia and trophic transfer efficiency in Chesapeake Bay. Ph.D. Dissertation, University of Maryland at College Park, College Park, Maryland.

HAGY, J. D., W. R. BOYNTON, AND M. M. WEIR. 1998. Estimating nitrogen and phosphorus loads for Patuxent River, p. 184–211. *In* W. R. Boynton and F. M. Rohland (eds.), Maryland Chesapeake Bay Water Quality Monitoring Program, Ecosystem Processes Component, Level One Report No. 15. Interpretive Report. [UMCES]CBL 98-073. Maryland Department of Natural Resources, Annapolis, Maryland.

HARDING, JR., L. W. AND E. S. PERRY. 1997. Long-term increase of phytoplankton biomass in Chesapeake Bay, 1950–1994. *Marine Ecology Progress Series* 157:39–52.

HELZ, G. R., J. M. ADELSON, C. V. MILLER, J. C. CORNWELL, J. M. HILL, M. HORAN, AND R. J. WALKER. 2000. Osmium isotopes demonstrate distal transport of contaminated sediments in Chesapeake Bay. *Environmental Science and Technology* 34:2528–2534.

HOWARTH, R. W., E. W. BOYER, W. J. PABICH, AND J. N. GALLOWAY. 2002. Nitrogen Use in the United States from 1961–2000 and Potential Future Trends. *Ambio* 31:88–96.

JAWORSKI, N. A., P. M. GROFFMAN, A. A. KELLER, AND J. C. PRAGER. 1992. A watershed nitrogen and phosphorus balance: The upper Potomac River basin. *Estuaries* 15:83–95.

JAWORSKI, N. A., R. W. HOWARTH, AND L. J. HETLING. 1997. Atmospheric deposition of nitrogen oxides onto the landscape contributes to coastal eutrophication in the northeast U.S. *Environmental Science and Technology* 31:1995–2004.

JUSTIĆ, D., N. N. RABALAIS, R. E. TURNER, AND Q. DORTCH. 1995. Changes in nutrient structure of river-dominated coastal waters: Stoichiometric nutrient balance and its consequences. *Estuarine, Coastal and Shelf Science* 40:339–356.

JUSTIĆ, D., R. E. TURNER, AND N. N. RABALAIS. 2003. Climatic influences on riverine nitrate flux: Implications for coastal marine eutrophication and hypoxia. *Estuaries* 26:1–11.

KARLSEN, A. W., T. M. CRONIN, S. E. ISHMAN, D. A. WILLARD, R. KERHIN, C. W. HOLMES, AND M. MAROT. 2000. Historical trends in Chesapeake Bay dissolved oxygen based on benthic foraminifera from sediment cores. *Estuaries* 23:488–508.

KEMP, W. M. AND W. R. BOYNTON. 1992. Benthic-pelagic interactions: Nutrient and oxygen dynamics, p. 149–221. *In* D. E. Smith, M. Leffler, and G. Mackiernan (eds.), Oxygen Dynamics in the Chesapeake Bay: A Synthesis of Recent Research. Maryland Sea Grant College, College Park, Maryland.

KEMP, W. M., P. A. SAMPOU, J. GARBER, J. TUTTLE, AND W. R. BOYNTON. 1992. Seasonal depletion of oxygen from bottom waters of Chesapeake Bay: Roles of benthic and planktonic respiration and physical exchange processes. *Marine Ecology Progress Series* 85:137–152.

LANGLAND, M. J., R. E. EDWARDS, L. A. SPRAGUE, AND S. YOCHUM. 2001. Summary of trends and status analysis for flow, nutrients, and sediments at selected nontidal sites, Chesapeake Bay Basin, 1985–99. Open-File Report 01-73. U.S. Geological Survey, New Cumberland, Pennsylvania.

LEIGHTON, M. O. 1904. Quality of water in the Susquehanna River drainage basin. U.S. Geological Survey Water-Supply and Irrigation Paper 108. U.S. Government Printing Office, Washington, D.C.

MALONE, T. C., W. M. KEMP, H. W. DUCKLOW, W. R. BOYNTON, J. H. TUTTLE, AND R. B. JONAS. 1986. Lateral variation in the production and fate of phytoplankton in a partially stratified estuary. *Marine Ecology Progress Series* 32:149–160.

NAJJAR, R. 1999. The water balance of the Susquehanna River Basin and its response to climate change. *Journal of Hydrology* 219:7–19.

NATIONAL RESEARCH COUNCIL. 2000. Clean Coastal Waters: Understanding and Reducing the Effects of Nutrient Pollution. Committee on the Causes and Management of Coastal Eutrophication, Ocean Studies Board and Water Science and Technology Board, Commission on Geosciences, Environment, and Resources, National Academy Press, Washington, D.C.

NEWCOMBE, C. L. AND W. A. HORNE. 1938. Oxygen-poor waters of the Chesapeake Bay. *Science* 88:80–81.

NEWCOMBE, C. L., W. A. HORNE, B. B. SHEPHERD, AND A. G. LANG. 1938. Hydrographic data of Chesapeake Bay and Patuxent River, Maryland collected in 1938. CBL Reference Series 38–1. Chesapeake Biological Laboratory, Solomons, Maryland.

NEWCOMBE, C. L., W. A. HORNE, AND B. B. SHEPHERD. 1939. Studies on the physics and chemistry of estuarine waters in Chesapeake Bay. *Journal of Marine Research* 2:87–116.

NEWCOMBE, C. L. AND A. G. LANG. 1939. The distribution of phosphates in the Chesapeake Bay. *Proceedings of the American Philosophical Society* 81:393–420.

OFFICER, C. B., R. B. BIGGS, J. L. TAFT, L. E. CRONIN, M. A. TYLER, AND W. R. BOYNTON. 1984. Chesapeake Bay anoxia: Origin, development, and significance. *Science* 223:22–27.

ORTH, R. J. AND K. MOORE. 1984. Distribution and abundance of submerged aquatic vegetation in Chesapeake Bay: An historical perspective. *Estuaries* 7:531–540.

PENNSYLVANIA DEPARTMENT OF AGRICULTURE. 1963–1989. Reported Fertilizer Consumption, 1962 to 1988. Pennsylvania Department of Agriculture, Harrisburg, Pennsylvania.

PENNSYLVANIA DEPARTMENT OF AGRICULTURE. 1990–1997. Fertil-

izer Distributed by County, 1989 to 1996. Pennsylvania Department of Agriculture, Harrisburg, Pennsylvania.

PENNSYLVANIA DEPARTMENT OF AGRICULTURE. 1998–1999. Total Fertilizer and Nutrients by County, 1997 and 1998. Pennsylvania Department of Agriculture, Harrisburg, Pennsylvania.

PRITCHARD, D. W. 1952. Salinity distribution and circulation in the Chesapeake estuarine system. *Journal of Marine Research* 11: 106–123.

RABALAIS, N. N. AND R. E. TURNER. 2001. Hypoxia in the Northern Gulf of Mexico: Description, causes and change, p. 1–36. *In* N. N. Rabalais and R. E. Turner (eds.), Coastal Hypoxia: Consequences for Living Resources and Ecosystems. Coastal and Estuarine Studies 58. American Geophysical Union, Washington, D.C.

RABALAIS, N. N., R. E. TURNER, D. JUSTIĆ, Q. DORTCH, AND W. J. WISEMAN, JR. 1999. Characterization of Hypoxia. Topic 1 Report for the Integrated Assessment on Hypoxia in the Gulf of Mexico. NOAA Coastal Ocean Program Decision Analysis Series No. 15. National Oceanic and Atmospheric Administration, Silver Spring, Maryland.

RABALAIS, N. N., R. E. TURNER, AND D. SCAVIA. 2002. Beyond science into policy: Gulf of Mexico hypoxia and the Mississippi River. *Bioscience* 52:129–142.

RITTER, C. AND P. A. MONTAGNA. 1999. Seasonal hypoxia and models of benthic response in a Texas bay. *Estuaries* 22:7–20.

SANFORD, L. P., K. G. SELLNER, AND D. L. BREITBURG. 1990. Covariability of dissolved oxygen with physical processes in the summertime Chesapeake Bay. *Journal of Marine Research* 48: 567–590.

SAS INSTITUTE. 1989. SAS/STAT® User's Guide, Version 6, 4th edition, Volume 2. SAS Institute, Inc., Cary, North Carolina.

SEITZINGER, S. P. 1988. Denitrification in freshwater and coastal marine ecosystems: Ecological and geochemical significance. *Limnology and Oceanography* 33:702–724.

SELIGER, H. H. AND J. A. BOGGS. 1988. Long term pattern of anoxia in the Chesapeake Bay, p. 570–583. *In* M. Lynch and E. C. Krome (eds.), Understanding the Estuary: Advances in Chesapeake Bay Research. Publication Number 129. Chesapeake Research Consortium, Solomons, Maryland.

SMITH, G. F. 2001. A characterization of oyster habitat in mesohaline Chesapeake Bay. Ph.D. Dissertation, University of Maryland, College Park, Maryland.

SMITH, R. A., R. B. ALEXANDER, AND M. G. WOLMAN. 1987. Water-quality trends in the nation's rivers. *Science* 235:1607–1615.

SPRAGUE, L. A., M. J. LANGLAND, S. E. YOCHUM, R. E. EDWARDS, J. D. BLOMQUIST, S. W. PHILLIPS, G. W. SHENK, AND S. D. PRESTON. 2000. Factors affecting nutrient trends in major rivers of the Chesapeake Bay watershed. Water Resources Investigations Report 00-4218. U.S. Geological Survey, Richmond, Virginia.

TAFT, J. L., W. R. TAYLOR, E. O. HARTWIG, AND R. LOFTUS. 1980. Seasonal oxygen depletion in Chesapeake Bay. *Estuaries* 3: 242–247.

TAYLOR, W. R. AND W. B. CRONIN. 1974. Plankton Ecology Project, Station Data, AESOP Cruises, April 1969–April 1971. Reference 74-6. Chesapeake Bay Institute Special Report 38. Chesapeake Bay Institute, The Johns Hopkins University, Baltimore, Maryland.

TRUITT, R. V. 1937. Report of Chesapeake Biological Laboratory. Contribution No. 22. Chesapeake Biological Laboratory, Solomons, Maryland.

TRUITT, R. V. 1938. Report of Chesapeake Biological Laboratory. Contribution No. 30. Chesapeake Biological Laboratory, Solomons, Maryland.

U.S. CENSUS BUREAU. 1952. Farms, Farm Characteristics, Livestock and Products, Crops, Fruits, Values. Middle Atlantic States, Volume 1, Part 2. U.S. Census of Agriculture: 1950. Bureau of the Census, U.S. Department of Commerce, Washington, D.C.

U.S. CENSUS BUREAU. 1961. Farms, Farm Characteristics, Livestock and Products, Crops, Fruits, Values. Pennsylvania Counties, Volume 1, Part 9. U.S. Census of Agriculture: 1959. Bureau of the Census, U.S. Department of Commerce, Washington, D.C.

U.S. CENSUS BUREAU. 1972. Pennsylvania Summary Data, Volume 1, Area Reports. Part 9. 1969 Census of Agriculture. Bureau of the Census, U.S. Department of Commerce, Washington, D.C.

U.S. CENSUS BUREAU. 1981. Pennsylvania State and County Data, Volume 1, Part 38. 1978 Census of Agriculture. Bureau of the Census, U.S. Department of Commerce, Washington, D.C.

U.S. CENSUS BUREAU. 1989 Series Part 38. 1987 Census of Agriculture. Bureau of the Census, U.S. Department of Commerce, Washington, D.C.

U.S. CENSUS BUREAU. 2003a. Table CO-EST2002-01-42. Pennsylvania County Population Estimates: April 1, 2000 to July 1, 2002. Population Division, U.S. Census Bureau, Washington, D.C.

U.S. CENSUS BUREAU. 2003b. Table CO-EST2002-02-36. New York County Population Estimates: April 1, 2000 to July 1, 2002. Population Division, U.S. Census Bureau, Washington, D.C.

U.S. DEPARTMENT OF AGRICULTURE. 1999. Pennsylvania State and County Data. Volume 1, Geographic Area Series Part 38. 1997 Census of Agriculture. National Agricultural Statistics Service. U.S. Department of Agriculture, Washington, D.C.

U.S. GEOLOGICAL SURVEY. 1947. Quality of Surface Waters of the United States 1944. Geological Survey Water-Supply Paper Number 1022. U.S. Government Printing Office, Washington, D.C.

U.S. GEOLOGICAL SURVEY. 1949. Quality of Surface Waters of the United States 1945. Geological Survey Water-Supply Paper Number 1030. U.S. Government Printing Office, Washington, D.C.

U.S. GEOLOGICAL SURVEY. 1950. Quality of Surface Waters of the United States 1946. Geological Survey Water-Supply Paper Number 1050. U.S. Government Printing Office, Washington, D.C.

U.S. GEOLOGICAL SURVEY. 1952. Quality of Surface Waters of the United States 1947. Geological Survey Water-Supply Paper Number 1102. U.S. Government Printing Office, Washington, D.C.

U.S. GEOLOGICAL SURVEY. 1953. Quality of Surface Waters of the United States 1948. Geological Survey Water-Supply Paper Number 1132. U.S. Government Printing Office, Washington, D.C.

U.S. GEOLOGICAL SURVEY. 1954a. Quality of Surface Waters of the United States 1949. Geological Survey Water-Supply Paper Number 1162. U.S. Government Printing Office, Washington, D.C.

U.S. GEOLOGICAL SURVEY. 1954b. Quality of Surface Waters of the United States 1950. Geological Survey Water-Supply Paper Number 1186. U.S. Government Printing Office, Washington, D.C.

U.S. GEOLOGICAL SURVEY. 1955. Quality of Surface Waters of the United States 1951. Geological Survey Water-Supply Paper Number 1197. U.S. Government Printing Office, Washington, D.C.

U.S. GEOLOGICAL SURVEY. 1956. Quality of Surface Waters of the United States 1952. Geological Survey Water-Supply Paper Number 1250. U.S. Government Printing Office, Washington, D.C.

U.S. GEOLOGICAL SURVEY. 1957. Quality of Surface Waters of the United States 1953. Geological Survey Water-Supply Paper Number 1290. U.S. Government Printing Office, Washington, D.C.

U.S. GEOLOGICAL SURVEY. 1960a. Quality of Surface Waters of the United States 1956. Geological Survey Water-Supply Paper

Number 1450. U.S. Government Printing Office, Washington, D.C.

U.S. GEOLOGICAL SURVEY. 1960b. Quality of Surface Waters of the United States 1957. Geological Survey Water-Supply Paper Number 1520. U.S. Government Printing Office, Washington, D.C.

WANG, P., J. STORRICK, AND L. C. LINKER. 1997. Chesapeake Bay Watershed Model Application and Calculation of Nutrient and Sediment Loadings, Appendix D: Phase IV Chesapeake Bay Watershed Model Precipitation and Meteorological Data Development and Atmospheric Nutrient Deposition. Report of the Modelling Subcommittee, August, 1997. Chesapeake Bay Program Office, Annapolis, Maryland.

WARD, J. R. AND C. A. HARR. 1990. Methods for collection and processing of surface-water and bed-material samples for physical and chemical analyses. Open-File Report 90-140. U.S. Geological Survey, Reston, Virginia.

WELSH, B. L., R. I. WELSH, AND M. L. DIGIACOMO-COHEN. 1994. Quantifying hypoxia and anoxia in Long Island Sound, p. 131–137. In K. R. Dyer and R. J. Orth (eds.), Changes in Fluxes in Estuaries: Implications From Science To Management. Olsen and Olsen, Fredensborg, Denmark.

WHALEY, R. C., J. H. CARPENTER, AND R. L. BAKER. 1966. Chesapeake Bay Institute, Special Report 12, Reference 66-4. Chesapeake Bay Institute, The Johns Hopkins University, Baltimore, Maryland.

ZAITSEV, Y. P. 1992. Recent changes in the trophic structure of the Black Sea. *Fisheries Oceanography* 1:180–189.

ZIMMERMAN, A. R. AND E. A. CANUEL. 2000. A geochemical record of eutrophication and anoxia in Chesapeake Bay sediments: Anthropogenic influence on organic matter composition. *Marine Chemistry* 69:117–137.

SOURCES OF UNPUBLISHED MATERIALS

CHESAPEAKE BAY LAND MARGIN ECOSYSTEM RESEARCH PROGRAM. Unpublished data. University of Maryland Center for Environmental Science, Cambridge, Maryland.

LANGLAND, M. Personal Communication. U.S. Geological Survey, 215 Limekiln Road, New Cumberland, Pennsylvania 17070.

U.S. CENSUS BUREAU. Unpublished data. Population of Counties by Decennial Census: 1900 to 1990. Population Division, U.S. Bureau of Census, Washington, D.C. <http://www.census.gov/population/cencounts>

U.S. DEPARTMENT OF AGRICULTURE. Unpublished data. Agricultural Statistics Data Base. National Agricultural Statistics Service. <http://www.nass.usda.gov>

U.S. ENVIRONMENTAL PROTECTION AGENCY (USEPA). Unpublished data. Chesapeake Bay Program Office, Chesapeake Bay Water Quality Monitoring Program, Annapolis, Maryland. <http://www.chesapeakebay.net>

U.S. ENVIRONMENTAL PROTECTION AGENCY (USEPA). Unpublished data. Chesapeake Bay Program Office, Chesapeake Bay Water Quality Monitoring Program, Annapolis, Maryland. <http://www.chesapeakebay.net>

U.S. GEOLOGICAL SURVEY. Unpublished data. Surface Water Data. Reston, Virginia. <http://water.usgs.gov>

Received, October 10, 2003
Revised, January 29, 2004
Accepted, March 30, 2004