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The contribution of physical processes to inter-annual variations of hypoxia in Chesapeake Bay: A 30-yr modeling study

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Abstract

A numerical circulation model with a very simple representation of dissolved oxygen dynamics is used to simulate hypoxia in Chesapeake Bay for the 30-yr period 1984–2013. The model assumes that the biological utilization of dissolved oxygen is constant in both time and space in an attempt to isolate the role that physical processes play in modulating oxygen dynamics. Despite the simplicity of the model it demonstrates skill in simulating the observed inter-annual variability of hypoxic volume, capturing 50% of the observed variability in hypoxic volume (<2 mg L^{-1}) for the month of July and 58% of the observed variability for the month of August, over the 30-yr period. Model skill increases throughout the summer suggesting that physical processes play a more important role in modulating hypoxia later in the summer. Model skill is better for hypoxic volumes than for anoxic volumes. In fact, a simple regression based on the integrated January–June Susquehanna River nitrogen load can explain more of the variability in the observed anoxic volumes than the model presented here. Model results suggest that the mean summer (June–August) wind speed is the single-most important physical variable contributing to variations in hypoxic volumes. Previous studies have failed to document the importance of summer wind speed because they have relied on winds measured at Patuxent Naval Air Station, which does not capture the observed inter-annual variations in wind speed that are observed by stations that directly measure wind over the waters of Chesapeake Bay.

Over the last half-century, observations have documented that the deep bottom waters of Chesapeake Bay become depleted of dissolved oxygen (DO) for a significant fraction of the summer months (Hagy et al. 2004; Kemp et al. 2005; Murphy et al. 2011). The spatial and temporal extent of low DO (hypoxic) regions has a number of significant consequences for ecosystem health and function (Breitburg et al. 1997; Breitburg 2002). While the earliest direct observations of low DO (hypoxia) were made during the 1930s (Newcombe and Horne 1938), there is considerable evidence that the extent and severity of hypoxic conditions increased substantially during the early 1980s (Cooper and Brush 1991; Boesch et al. 2001; Liu and Scavia 2010). While it is generally accepted that this increase was the result of increased anthropogenic nutrient loads to the Bay, studies that attempt to statistically relate the inter-annual variations in hypoxic volume to nutrient loading often fail to explain the majority of the variability (Hagy et al. 2004; Scully 2010a; Murphy et al. 2011). These studies often explain a significantly larger fraction of the inter-annual variability of hypoxic volume when they consider physical factors such as variations in wind forcing.

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Even though these statistical studies provide evidence that both biological and physical processes play an important role in controlling hypoxia in Chesapeake Bay, quantifying the relative importance of these processes remains a significant challenge. This difficulty arises because there is considerable covariance between the biological and physical processes that modulate DO at both seasonal and inter-annual timescales. A clear example of this covariance is the strong correlation between nutrient inputs and river discharge. Increased river discharge delivers more nutrients to the system, providing the fuel for phytoplankton growth that ultimately leads to hypoxic conditions. Increased river discharge increases stratification and decreases vertical mixing, which also is generally thought to favor hypoxia (Officer et al. 1984). However, increased river discharge also increases the up-estuary advection of oxygen, which could favor reduced hypoxia (Li et al. 2015). Wind-driven processes physically alter the supply of DO to hypoxic waters through direct vertical mixing, modulating stratification (Scully et al. 2005), and driving axial and transverse advective fluxes (Scully 2010b; Li et al. 2015). Wind-driven processes also can alter the vertical and horizontal transport of nutrients and organic matter, which can substantially alter biological processes that impact hypoxia (Malone et al. 1986;

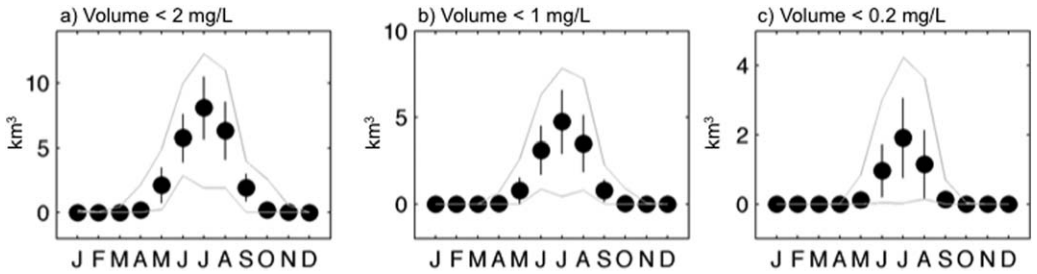

Fig. 1. Monthly averaged hypoxic volume for the entire model domain based on: (**a**) < 2 mg L^{-1} threshold, (**b**) < 1 mg L^{-1} threshold, and (**c**) < 0.2 mg L^{-1} threshold from the 30-yr simulation. Vertical lines represent ± 1 standard deviation and gray lines represent maximum and minimum monthly averages.

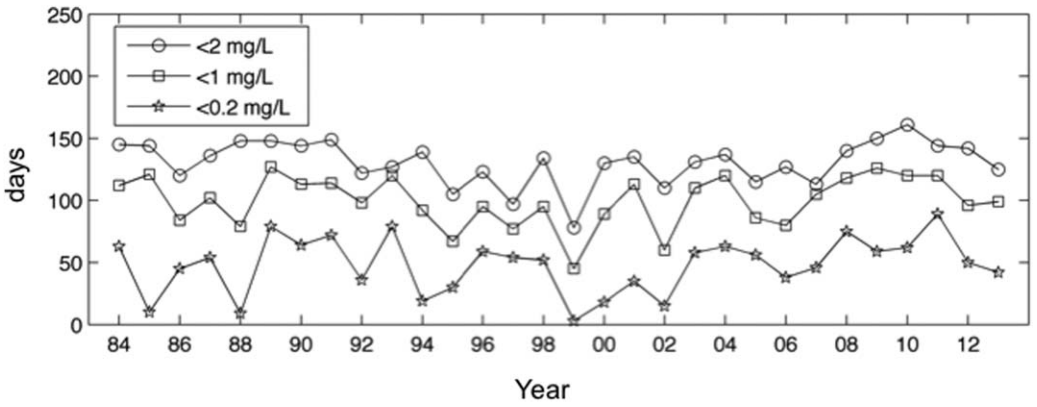

Fig. 2. Duration of the predicted occurrence of hypoxic conditions < 2 mg L^{-1} (circles), hypoxic conditions < 1 mg L^{-1} (squares), and anoxic conditions < 0.2 mg L^{-1} (stars) from 30-yr model simulation calculated from the entire model domain.

Lee et al. 2013). Much of our understanding of oxygen dynamics comes from studies that rely on statistical correlations to demonstrate the link between processes and inter-annual variations in hypoxic volumes. Yet, these correlations are confounded by the fact that oxygen dynamics respond to a number of inter-related biological and physical processes with complex and potentially nonlinear interactions.

Given the complex interactions between physical and biogeochemical processes, modeling studies provide a powerful tool for better understanding oxygen dynamics. Yet given the complexity of many coupled hydrodynamic and biogeochemical models, quantifying the relative importance of specific processes on hypoxia can be extremely challenging—even with a model. To try and address these complexities and isolate the role of physical processes, Scully (2013) introduced a highly simplified approach to modeling oxygen in Chesapeake Bay. This approach treats the biological processes that modulate oxygen as a single constant that is held constant in both time and space. Despite the simplicity and obvious limitations of this approach, it demonstrates skill in simulating the seasonal cycle of DO in the Bay and highlights the first order control that physical processes play in modulating oxygen dynamics. Further, by removing any variability caused by biological processes, this approach can be used to conduct detailed sensitivity studies that examine the role of physical processes in controlling hypoxia. Previous work using this model has focused primarily on simulating the seasonal cycle of oxygen and did not directly addressed inter-annual variability (Scully 2013). However, from both a scientific and management perspective, understanding the role of physical processes in contributing to inter-annual variations in hypoxia in the Bay is of greater interest. In this paper, the model of Scully (2013) is applied to a 30-yr period in order to try and quantify the importance of physical processes in modulating inter-annual variations in hypoxic volume.

Methods

The model used in this study is nearly identical to that used in Scully (2013). A brief description is provided here for clarity. The hydrodynamic model is based on the Chesapeake

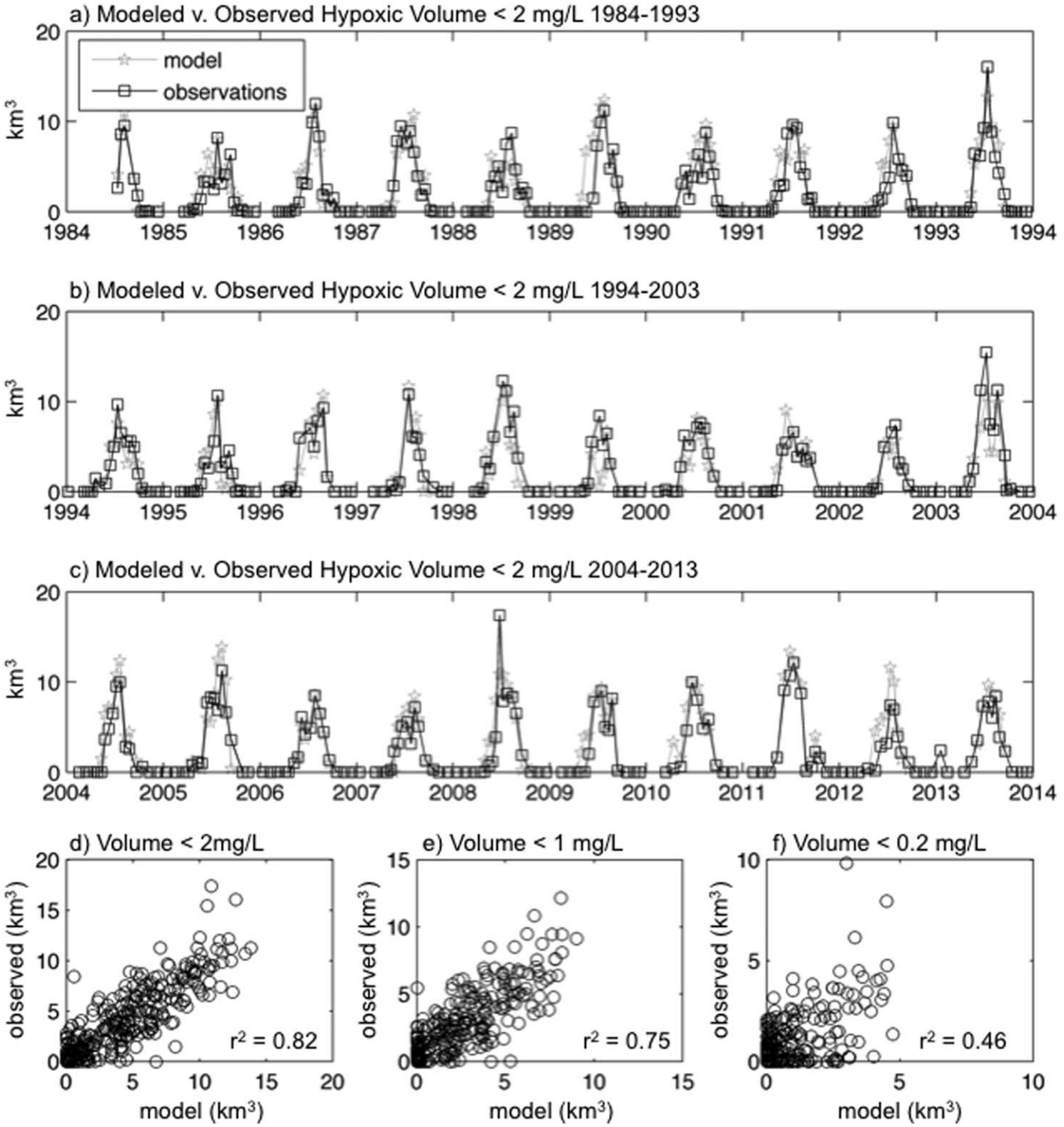

Fig. 3. Comparison of the predicted hypoxic volume < 2 mg L^{-1} (gray stars) to the observed (black squares) volume based on individual CBP cruises for (**a**) 1984–1993, (**b**) 1994–2003, and (**c**) 2004–2013. Scatter plots comparing the model prediction and observed hypoxic volumes for (**d**) < 2 mg L^{-1} threshold, (**e**) < 1 mg L^{-1} threshold, and (**f**) < 0.2 mg L^{-1} threshold. Reported correlations are all significant at $p < 0.05$.

Bay Regional Ocean Modeling System (ROMS) Community Model (ChesROMS) (Xu et al. 2012). The 150 by 100 model domain has 20 vertical sigma levels and includes the nine largest tributaries to the Bay, as well as the shelf region immediately adjacent to the Bay mouth. Model forcing includes river discharge derived from the United States Geological

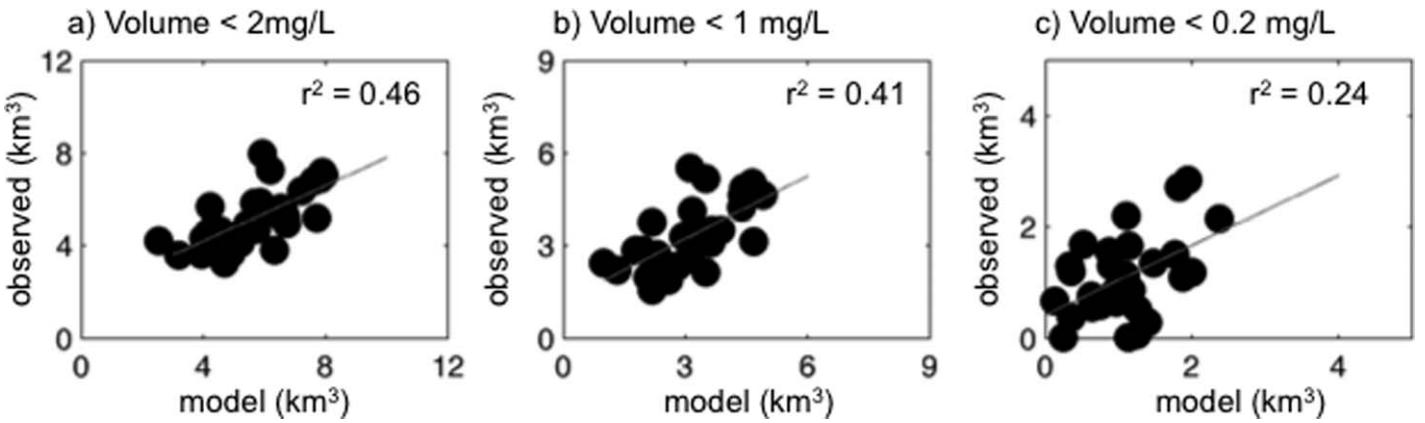

Fig. 4. Comparison of the modeled and observed mean summer (June–August) hypoxic volume for (**a**) < 2 mg L^{-1} threshold, (**b**) < 1 mg L^{-1} threshold, and (**c**) < 0.2 mg L^{-1} threshold. Straight line represents the best fit least squares regression. Reported correlations are all significant at $p < 0.05$.

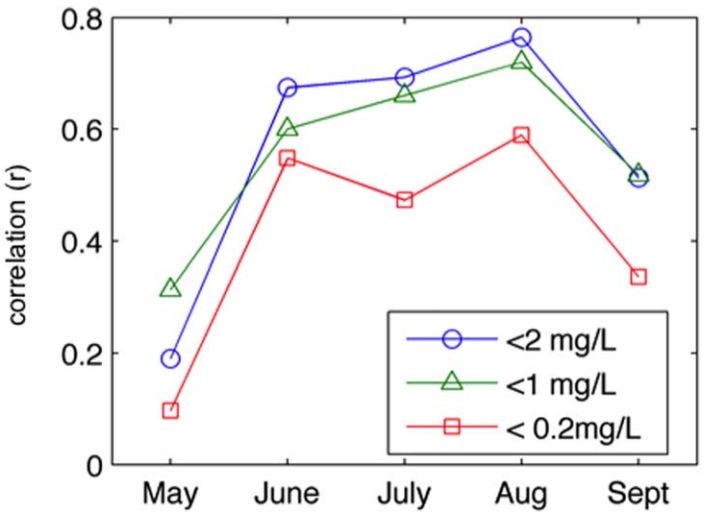

Fig. 5. Correlation coefficient (r), between the 30-yr modeled hypoxic volumes and those derived from the CBP data as a function of month. Circles represent < 2 mg L^{-1} threshold, triangles represent < 1 mg L^{-1} threshold, and squares represent < 0.2 mg L^{-1} threshold. [Color figure can be viewed at wileyonlinelibrary.com]

Survey (USGS) gauging stations, tidal constituents derived from the Advanced Circulation (ADCIRC) model, observed non-tidal water level (Duck, NC and Lewes, DE), temperature and salinity at the oceanic boundary from the World Ocean Atlas 2001, and surface atmospheric forcing (short wave solar radiation, long wave radiation, rainfall, surface air humidity, pressure, temperature, and 10 m winds) from the National Center for Environmental Prediction (NCEP) North American Regional Reanalysis (NARR) model. Unlike in Scully (2013), but consistent with other modeling studies of Chesapeake Bay (Testa et al. 2014; Li et al. 2015), surface winds from the NARR model are used instead of the observed winds from the National Data Buoy Center (NDBC) Thomas Point Light (TPL) station (NDBC station TPLM2). Wind measurements at TPL began in 1986 and there are numerous gaps in the data making their use impractical for a 30-yr simulation.

As in Scully (2013), DO is introduced into the model as a passive tracer. Inside the estuarine portion of the domain, a spatially and temporally constant oxygen consumption of 1.4×10^{-4} mmoleO_2 m^{-3} s^{-1} (0.39 mmoleO_2 m^{-3} d^{-1}) is prescribed, which is 20% smaller than the value used in Scully (2013). This value is essentially a tuning parameter that was selected to best match the modeled hypoxic volume with the observations presented in Bever et al. (2013) for the year 2004. The smaller value used in this study was needed to produce roughly the same volume of hypoxic water when using the NARR winds, which are weaker than the observed TPL winds (see Fig. 13). For simplicity, the surface oxygen concentration is set to saturation values based on the modeled surface temperature and salinity. Dissolved oxygen concentrations at both the oceanic and river boundaries are fixed to saturation values and DO values are not allowed to become negative, essentially imposing a respiration rate of zero for anoxic conditions. For computational reasons, each model year is run separately, initiated from the final time step from the simulation of the previous year. The model was spun up beginning in 1979, but our analysis focuses on the period from 1984 to 2013 because the first Chesapeake Bay Program (CBP) water quality cruise was conducted in July 1984, providing a consistent data set for comparison.

A detailed skill assessment of this model is beyond the scope of this paper. However, it is worth noting that a comprehensive skill assessment that compared numerous models to CBP DO data found no statistical difference between the skill of models that use a full biogeochemical representation of oxygen dynamics and the simple model used here (Irby et al. 2016). This is true for bottom DO, surface DO, and the strength and location of the vertical DO gradient. Instead of focusing on a detailed comparison of the model to individual CBP stations, in this paper we compare simulated hypoxic

Table 1. Correlation coefficient (r) between the modeled average summer (June–August) hypoxic volumes (< 2 mg L^{-1}, < 1 mg L^{-1}, and < 0.2 mg L^{-1}) for 1984–2013 and the January–June Susquehanna River discharge, the January–June Susquehanna River total nitrogen load, the June–August wind speed from NARR model, the June–August modeled Bay-wide water temperature, and the percent duration of June–August winds from eight equally spaced compass directions from NARR model. Significant correlations (< 0.05) are denoted with asterisk.

	Jan–Jun Susquehanna River discharge	Jan–Jun Susquehanna nitrogen load	Jun–Aug Wind speed (NARR)	Jun–Aug Bay water temperature (model)	Percent duration summer wind from north	Percent duration summer wind from northeast	Percent duration summer wind from east	Percent duration summer wind from southeast	Percent duration summer wind from south	Percent duration summer wind from southwest	Percent duration summer wind from west	Percent duration summer wind from northwest
<2 mg L^{-1}	0.58*	0.56*	−0.76*	0.28	−0.21	−0.41*	0.03	0.18	−0.20	−0.15	0.43*	0.48*
<1 mg L^{-1}	0.58*	0.55*	−0.74*	0.30	−0.18	−0.36*	0.06	0.21	−0.23	−0.19	0.39*	0.49*
<0.2 mg L^{-1}	0.51*	0.53*	−0.67*	0.32	−0.15	−0.29	0.07	0.18	−0.24	−0.21	0.38*	0.48*

volumes to those derived from the CBP data as published by Bever et al. (2013). This data set has been extended through the end of 2013 for this analysis. Between the months of May and September there are typically two CBP water quality cruises per month. Because data are collected over the entire main stem of the Chesapeake Bay and its tributaries from multiple vessels, each "cruise" usually spans several days. The interpolated hypoxic volumes of Bever et al. (2013) generally represent average conditions over a 4–7 d period. In order to compare the model output to this data set, modeled hypoxic volumes were averaged over the period defined by the first and last day of any given cruise. To compare these data with the results of Bever et al. (2013), modeled hypoxic volumes were defined based on the thresholds < 2 mg L^{-1} and < 1 mg L^{-1}, and the modeled anoxic volume is defined based on the threshold < 0.2 mg L^{-1}. Deleterious effects to biota can begin at higher concentrations than those considered here (Vaquer-Sunyer and Duarte 2012), but for consistency with previous studies (Hagy et al. 2004; Scully 2010a; Murphy et al. 2011; Bever et al. 2013) we utilize these thresholds.

Results

Consistent with the results of Scully (2013), results from this 30-yr simulation demonstrate a well-defined seasonal cycle of hypoxia in every year simulated (Fig. 1). Hypoxic conditions are predicted to begin on average in early May, peak in mid-July before finally mixing away in mid-September. Both the onset and termination of hypoxia are predicted to vary considerably, with hypoxic conditions (<2 mg L^{-1}) beginning in early April and lasting until late October in some years. The model predicts that anoxic conditions will occur on average every summer, but anoxic conditions do not always persist throughout the entire summer. The duration of hypoxia and anoxia varies considerably in the model results (Fig. 2). The duration of hypoxic conditions (<2 mg L^{-1}) varies from a maximum of 161 d to a minimum of 78 d and the duration of anoxic conditions (<0.2 mg L^{-1}) varies from a maximum of 89 d to a minimum of 3 d. These results demonstrate that physical processes have the potential to contribute significantly to inter-annual variability in both the spatial extent and duration of hypoxia.

Figure 3 shows a comparison of the observed and modeled hypoxic volume (<2 mg L^{-1}) for all cruises from 1984 to 2013. Also shown are scatter plots comparing the predicted hypoxic volumes to the observed volumes based on all three definitions of hypoxia. Compared at this time scale, the model demonstrates skill in predicting hypoxic volume and explains over 80% of the variance in the time series of hypoxic volume (<2 mg L^{-1}). Model skill is worse for anoxic volume ($r^2 = 0.46$). The variance at this time scale (i.e., the individual cruise) is dominated by the seasonal cycle and does not directly address the variability at inter-annual time

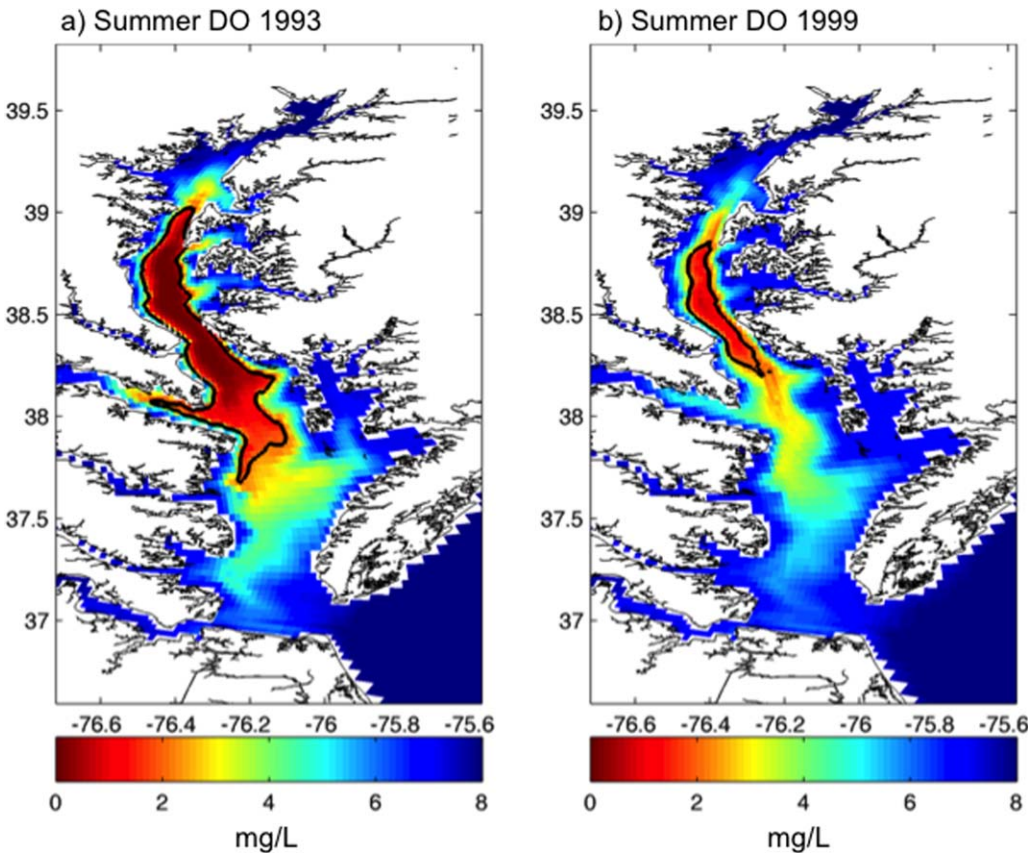

Fig. 6. Comparison of the mean summer (June–August) bottom DO concentration for (**a**) 1993 and (**b**) 1999. Thick black line represents the 2 mg L^{-1} contour.

scales. Figure 4 compares the observed and predicted hypoxic volumes for all three thresholds averaged over the months of June, July, and August for each year. For all three thresholds, the modeled hypoxic volumes are significantly ($p < 0.05$) correlated with the observations. The strongest correlation between the model and observations is for the hypoxic volume based on the 2 mg L^{-1} threshold ($r^2 = 0.46$) with the weakest correlation between the modeled and observed anoxic volumes ($r^2 = 0.24$).

Comparing hypoxic volumes for the individual months when hypoxia is typically observed shows that model skill generally increases throughout the summer (Fig. 5). The strongest correlation is for the month of August and the weakest correlation is for the month of May. The modeled and observed hypoxic volumes for the month of May are not significantly correlated for any of the thresholds considered. Correlations for September are significant ($p < 0.05$), but values are lower than for August. Over the period 1984–2013, a simple model with no biological variability can explain 50% and 58% of the variance in the observed hypoxic volume (<2 mg L^{-1}) for the months of July and August, respectively. We interpret the increasing correlations throughout the summer to indicate that physical processes play a more important role in modulating DO during the later summer months. Presumably early in the summer, hypoxia is driven by the respiration of spring phytoplankton-derived organic matter, which cannot be captured by this simple model. These results contradict the work of Murphy et al. (2011) who found that hypoxic volumes in the early summer exhibited a long-term trend that could be explained by the long-term trend in stratification, while the late summer trend was consistent with decreased nitrogen loading.

The variable that explains most of the inter-annual variability in hypoxic volume (all definitions) predicted by the numerical model is the summer (June–August) wind speed (Table 1). The second most correlated variable is the January–June Susquehanna River discharge. These two variables can explain roughly 70% of the variance in hypoxic volume as predicted by the model. Hypoxic volumes are negatively correlated with summer wind speed and positively correlated with Susquehanna River discharge. The positive correlation with river discharge is in contrast to the results of Li et al. (2015) who found that increased river discharge decreased integrated hypoxic volumes slightly. Correlations with the mean summer water temperature are positive, but not significant at $p < 0.05$. For the 30-yr time series of hypoxic

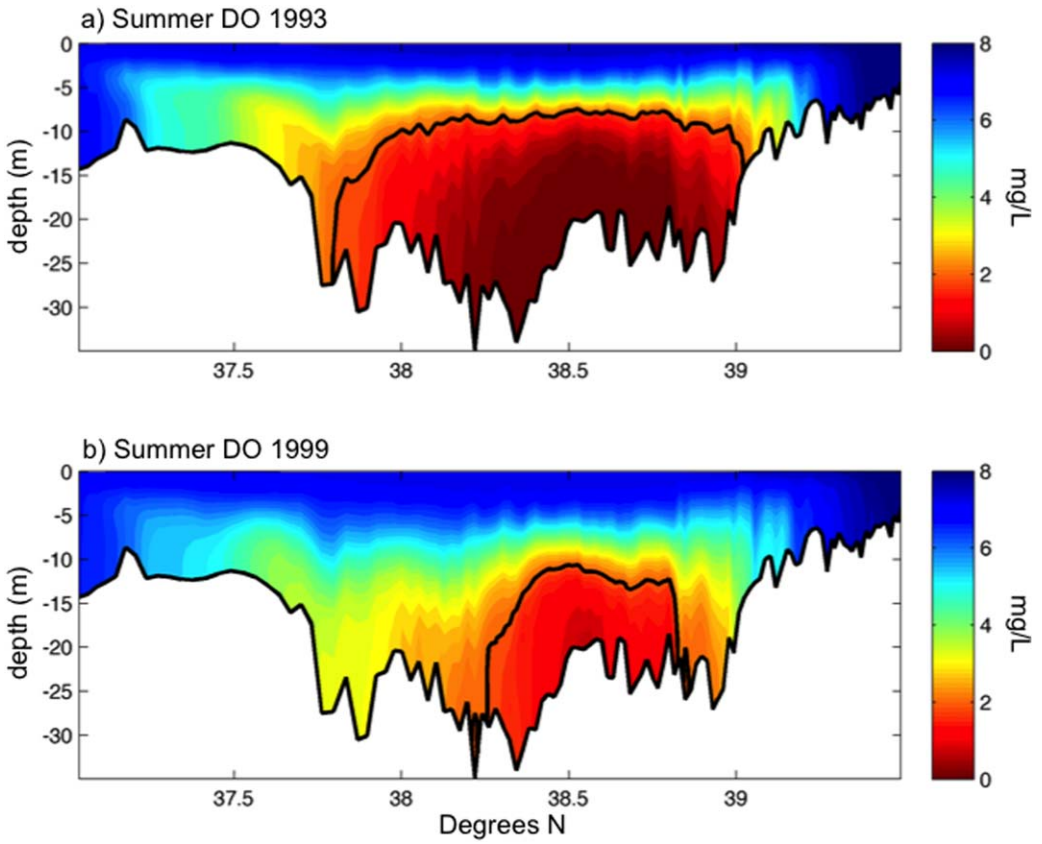

Fig. 7. Comparison of the mean summer (June–August) thalweg DO concentration for (**a**) 1993 and (**b**) 1999. Thick black line represents the 2 mg L^{-1} contour.

volumes (<1 mg L^{-1} and 2 mg L^{-1}), statistically significant negative correlations are found for the percent duration of summer winds from the northeast. Both hypoxic and anoxic volumes exhibit significant ($p < 0.05$) positive correlations with the duration of winds from the west and northwest and negative correlations with duration of winds from northeast. The positive correlations with the duration of winds from the west is consistent with the analysis of Scully (2010a) who found that the percent duration of summer winds from the west was an important variable in controlling hypoxic volumes for the time period (1950–2007).

It is worth noting that even though the model contains no biological variability, the modeled time series of hypoxic volumes are significantly correlated with observed nitrogen loading. This is the result of the tight coupling between the integrated nitrogen loading and the Susquehanna River discharge and illustrates the difficulty in separating the contribution of variables that have significant co-variance. In this model, there is no biologic response to nutrient loading and the positive correlation between river discharge and hypoxic volume is consistent with increased stratification leading to decreased vertical mixing. Both Scully (2013) and Li et al. (2015) suggest that hypoxic volumes are relatively insensitive to changes in river discharge because of the compensatory relationship between increased up-Bay advective flux of oxygen and decreased vertical mixing associated with increased river discharge.

To highlight the inter-annual differences in DO distribution, we compare the predicted average summer (June–August) DO concentration for 1993 and 1999 (Figs. 6, 7). In 1999, the mean summer wind speed was the second strongest during the 30-yr period and the January–June river discharge was the fourth lowest. In contrast, 1993 had the second highest average January–June river discharge and third weakest average summer winds. The high winds and low river discharge in 1999 limited the bottom extent of low DO compared to 1993. The overall length of hypoxic water (<2 mg L^{-1}) expanded from 88 km in 1999 to 146 km in 1993. Similarly, the effective width of the hypoxic zone (<2 mg L^{-1}) in 1993 is nearly double that of 1999 and hypoxic water spreads laterally into the lower Potomac River (PR).

Empirical orthogonal function (EOF) analysis is used to examine the inter-annual variability of the summer DO

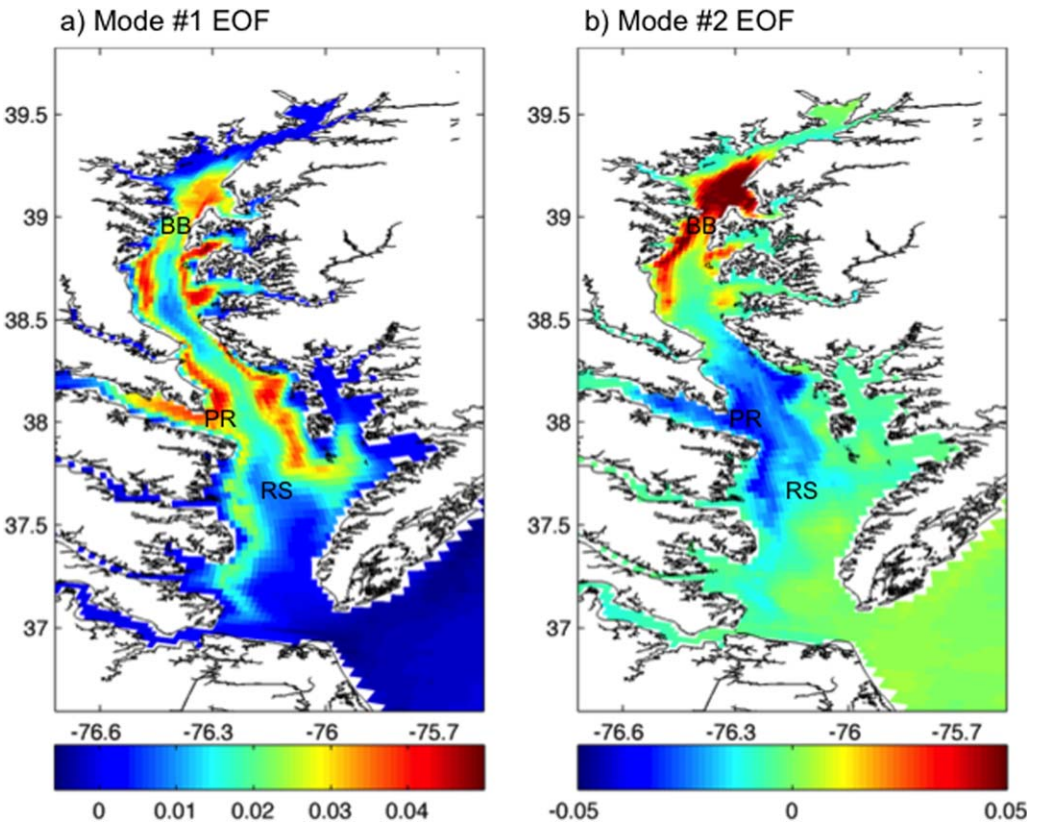

Fig. 8. The (**a**) first and (**b**) second mode EOF for summer (June–August) mean bottom DO concentration for the 30-yr simulation. First mode explains 70% of the variance and second model explains 13% of the variance. The location of the BB, mouth of PR, and RS are denoted on the figure.

concentration. Both the bottom DO and profiles of DO from the deepest location in the estuarine cross-section along the length of the Bay (e.g., the thalweg) are averaged over the months of June through August for each year and the EOF analysis is performed on the resulting 30-yr time series. The first and second modes from the thalweg EOF analysis are highly correlated with the first and second modes from the EOF analysis of bottom DO, and represent essentially the same modes of variability. The first and second modes from the EOF analysis explain roughly 70% and 13% of the variance, respectively, for both bottom DO and the thalweg distribution of DO. The first mode of the bottom DO EOF is characterized by high variance in DO concentration centered roughly on the 8 m isobath surrounding the deep channel (Fig. 8). This is a region where there is a strong DO gradient and generally coincides with the location where the pycnocline intersects the bottom, on average. For the thalweg distribution, the mode 1 EOF has the greatest variability in the pycnocline with greater variability in the northern portion of the Bay near the Bay Bridge (BB) where the bathymetry shoals (Fig. 9). The first mode is significantly correlated with both the averaged summer (June–August) wind speed ($r = 0.64$) and the January–June Susquehanna River discharge ($r = -0.60$). Increased wind mixing generally erodes hypoxic water and shifts the location where the pycnocline intersects the bottom into deeper water, consistent with the positive correlation with mode 1. Under increased river discharge, the location where the pycnocline intersects the bottom moves into shallower water, resulting in a negative correlation between river discharge and the variability of mode 1. This suggests that the lateral and vertical expansion of hypoxic water in response to increased river discharge is more important than the increase in the advective flux at the southern limit of the hypoxic zone. Mode 1 is generally positive everywhere suggesting that regions of high variability are in phase, increasing or decreasing at the same time.

In contrast, mode 2 is characterized by both positive and negative values and represents a north-south shift in the oxygen field where variations in bottom DO north and south of roughly 38.5°N are out of phase (Figs. 8, 9). Our interpretation is that this mode represents the response to bathymetrically controlled convergence/divergence caused by increased river discharge. We hypothesize that elevated river discharge leads to strong surface convergence south of the mouth of the PR, where the deep channel shoals from over 40 m to 15 m onto Rappahannock Shoal (RS). Strong surface

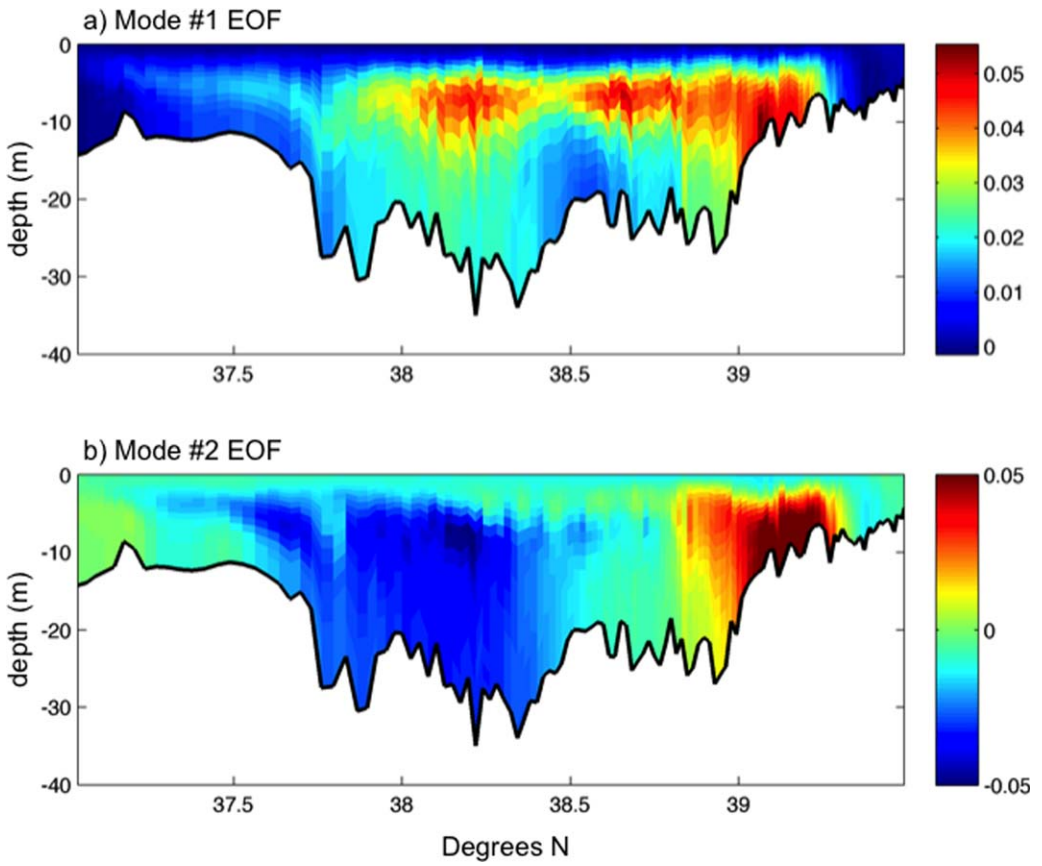

Fig. 9. The (**a**) first and (**b**) second mode EOF for summer (June–August) mean thalweg DO concentration for the 30-yr simulation. First mode explains 70% of the variance and second model explains 13% of the variance.

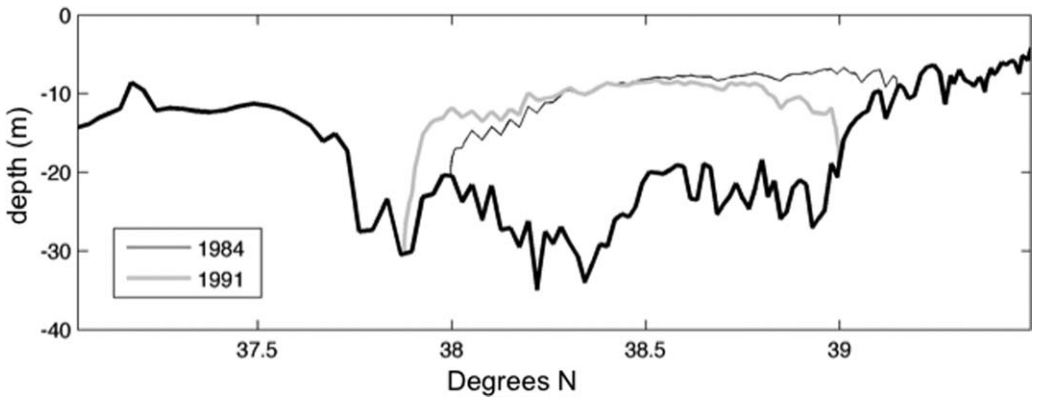

Fig. 10. Comparison of the location of the thalweg 2 mg L^{-1} mean summer DO contour for 1984 (years with lowest mode #2 EOF score) and 1991 (year with highest mode #2 EOF score).

convergence caused by this rapid decrease in bathymetry is intensified during high river discharge conditions and causes significant downward advection and mixing of surface oxygen (M. E. Scully, Mixing of DO in Chesapeake Bay driven by the interaction between wind driven circulation and estuarine bathymetry, Journal of Geophysical Research: Oceans, unpubl. in press.). Similarly, south of the BB where the thalweg depth increases from less than 15 m to over 40 m, there is strong surface divergence in the residual along-channel flow, leading to upwelling of low-oxygen bottom waters and decreasing oxygen concentration in the upper layer.

The downwelling near RS and the upwelling south the BB is enhanced by increased river discharge and causes the oxycline

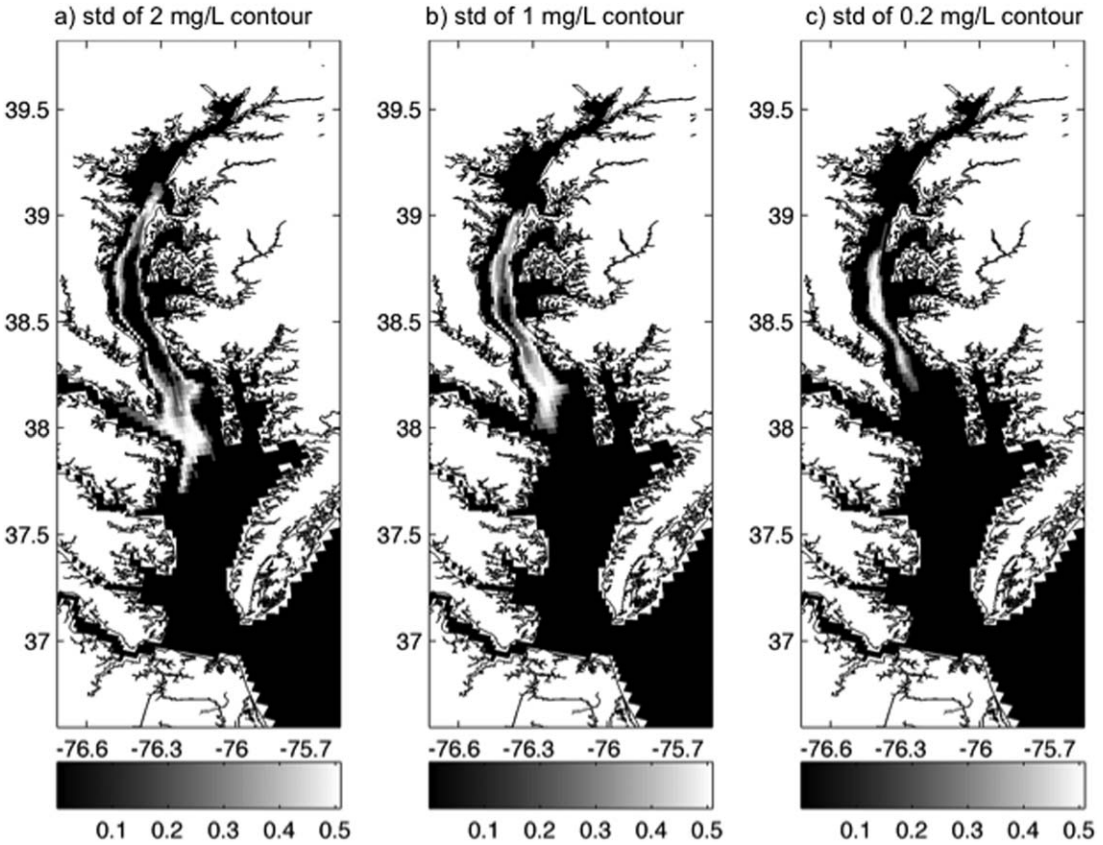

Fig. 11. Standard deviation of the location of the summer (June–August). (**a**) 2 mg L^{-1} bottom DO contour; (**b**) 1 mg L^{-1} bottom DO contour; (**c**) 0.2 mg L^{-1} bottom DO contour.

to tilt up toward the north. This is illustrated by comparing the location of the 2 mg L^{-1} contour for 1984 (year with lowest EOF #2 score) with 1999 (year with highest EOF #2 score) (Fig. 10). The second mode is negatively correlated with the June–August Susquehanna River discharge ($r = -0.71$) suggesting that when summer river discharge is high, the region of low oxygen water shifts northward. This pattern of variability and the negative correlation with river discharge is opposite to what is expected if increased river discharge resulted in a simple down-estuary displacement of the low DO region. Li et al. (2015) found that increased river discharge resulted in increased up-Bay oxygen flux at the southern end of the hypoxic zone, which is consistent with the northward displacement of the 2 mg L^{-1} contour in response to increased river discharge, but does not explain the northward and upward shift near the BB.

In the southern portion of the Bay, the model predicts that the horizontal bottom DO gradient is relatively weak in the along channel direction (e.g., Figs. 6, 7). As a result, even relatively small changes in the bottom DO concentration in this region can result in large shifts in the location of the southern boundary of hypoxic water (<2 mg L^{-1}). This area does not appear as a region of high DO variance in either the bottom or thalweg mode #1 EOF (Figs. 8a, 9a). However if we quantify the variance in the location of the 2 mg L^{-1} contour, the biggest changes from summer-to-summer are in this region between 37.75°N and 38.25°N (Fig. 11). In the middle section of the Bay, the model predicts that the maximum difference in the vertical position of the 2 mg L^{-1} contour is only about 3 m from summer-to-summer (Fig. 12). Similarly, the modeled width of the hypoxic zone in the middle of the Bay exhibits very little variability from year-to-year, and nearly all of the variability in the size of the hypoxic zone is caused by changes in its length. Unlike the location of the 2 mg L^{-1} contour, which varies mostly around the periphery of the deep channel, the location of the 0.2 mg L^{-1} contour is limited mostly to the deep bottom waters between 38.3°N and 38.9°N. In this region, the average summer oxygen concentration is nearly always less than 2 mg L^{-1} and it is the core of the hypoxic zone predicted by the model.

The model results indicate that the mean summer wind speed is the primary physical variable that contributes to this variability. Previous studies that have examined long-term trends in observed hypoxic volume in Chesapeake Bay have not noted a significant negative correlation between summer wind speed and hypoxic volume (Scully 2010a; Murphy et al. 2011; Lee et al. 2013; Zhou et al. 2014). These

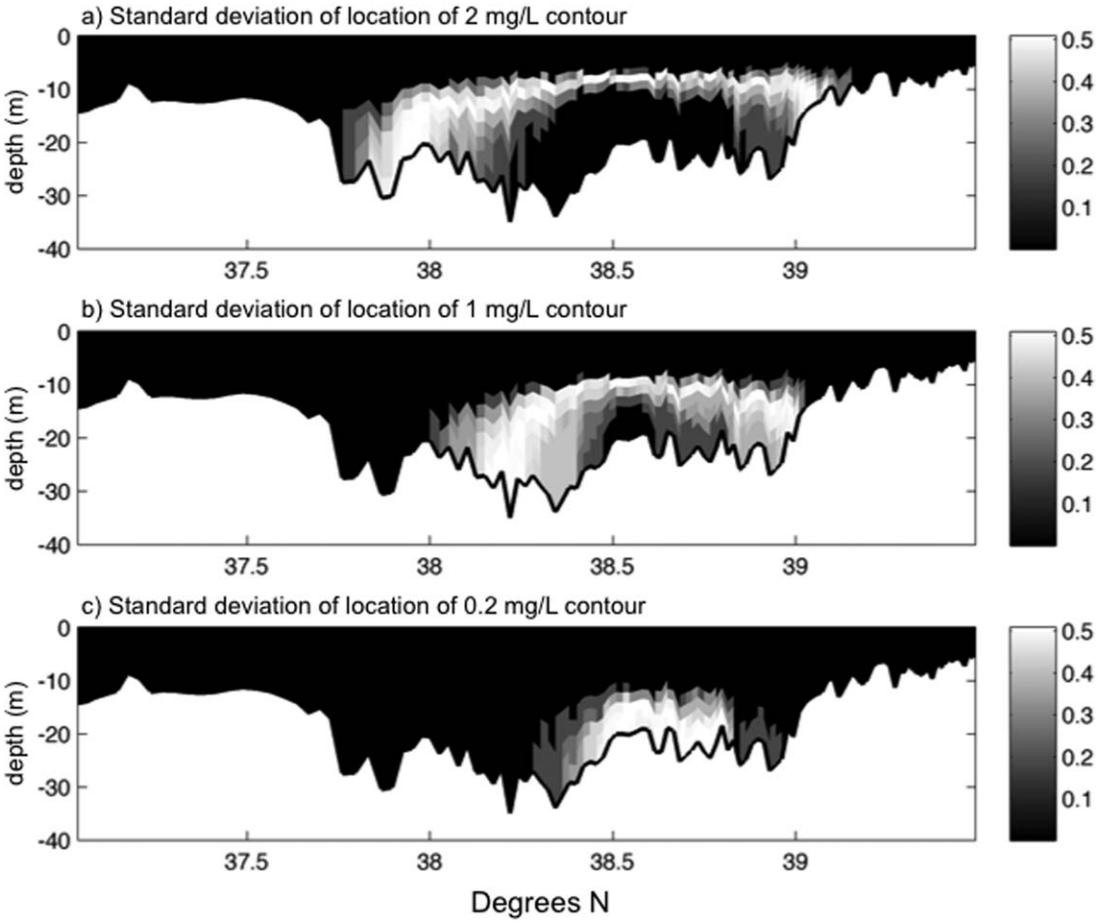

Fig. 12. Standard deviation of the location of the summer (June–August). (**a**) 2 mg L^{-1} thalweg DO contour; (**b**) 1 mg L^{-1} thalweg DO contour; (**c**) 0.2 mg L^{-1} thalweg DO contour.

studies have used the winds measured at the Patuxent Naval Air Station (PNAS) and not the winds from TPL, which are measured over water. We perform a correlation analysis on the observed time series of summer hypoxic volumes derived from the CBP data similar to that performed on the model output, but using the observed water temperature and winds from TPL (Table 2). Data were not available at TPL until 1986, so the analysis is limited to 28 yr (1986–2013). Consistent with the results from the numerical simulations, the observed hypoxic and anoxic volumes are significantly correlated with the observed January–June Susquehanna River discharge. Unlike previous studies, we find that the observed hypoxic volumes (<1 and <2 mg L^{-1}) are significantly correlated with the observed summer wind speed. A simple multiple regression that includes mean summer wind speed and January–June Susquehanna River discharge can explain roughly 60% of the variance in the observed time series of hypoxic and anoxic volumes, which is similar to the variance explained in the model. We note that hypoxic volumes are not significantly correlated with summer wind speed measured at PNAS, consistent with previous studies.

Correlations between nitrogen loading and hypoxic volumes (<2 mg L^{-1}) are similar between the model and observations despite the fact that the model has no response to this loading. This suggests that much of the variance in the observed hypoxic volumes could simply be explained by the physical response to increased stratification driven by Susquehanna flow. In contrast, the correlation between the observed anoxic volumes and nitrogen loading is much stronger in the observations than in the model suggesting that the core of anoxic water is primarily caused by the response to nutrient loading. In fact, the summer anoxic volumes from 1984 to 2013 are more strongly correlated with the integrated January–June Susquehanna River nitrogen load than either the numerical model results, or any other variable. Unlike the analysis of the model results, the river discharge generally explains more of the inter-annual variability in the observed hypoxic volumes than the summer wind speed. While there are a number of potential explanations for this, we suggest that the greater correlation between the observed hypoxic volumes and river discharge reflects the fact that increased river discharge also brings

Table 2. Correlation coefficient (r) between the observed (CBP data as published in Bever et al. [2013]) average summer (June–August) hypoxic volumes (<2 mg L^{-1}, <1 mg L^{-1}, and <0.2 mg L^{-1}) for 1986–2013 and the January–June Susquehanna River discharge, the January–June Susquehanna River total nitrogen load, the June–August wind speed from TPL, the June–August water temperature at TPL, and the percent duration of June–August winds from eight equally spaced compass directions measured at TPL. Significant correlations (< 0.05) are denoted with asterisk.

	Jan–Jun Susquehanna River discharge	Jan–Jun Susquehanna nitrogen load	Jun–Aug Wind speed (TPL)	Jun–Aug Bay water temperature (TPL)	Percent duration summer wind from north	Percent duration summer wind from northeast	Percent duration summer wind from east	Percent duration summer wind from southeast	Percent duration summer wind from south	Percent duration summer wind from southwest	Percent duration summer wind from west	Percent duration summer wind from northwest
<2 mg L^{-1}	0.67*	0.61*	−0.48*	0.00	0.15	−0.39*	0.02	0.18	−0.14	0.14	0.05	0.25
<1 mg L^{-1}	0.74*	0.66*	−0.42*	−0.02	0.08	−0.42*	−0.01	0.15	−0.05	0.15	0.03	0.27
<0.2 mg L^{-1}	0.81*	0.86*	−0.14	−0.21	0.07	−0.21	−0.16	0.16	0.00	−0.17	0.11	0.34

increased nutrient loads. The stronger correlation between the observed hypoxic volumes and river discharge is most likely because there is both a physical and biological response to this forcing.

Hypoxic and anoxic volumes are not significantly correlated with the observed mean summer water temperature at TPL (Table 2), generally consistent with the results from the 30-yr model simulation. In contrast to the modeling results of Scully (2013), which suggest that the seasonal variation of temperature plays an important role in the seasonal cycle of hypoxia, both the model and observations suggest that inter-annual variations in water temperature do contribute significantly to inter-annual variations in hypoxia at the time scales considered here. At longer time scales, it has been suggested that increasing water temperature could enhance hypoxic volume (Kemp et al. 2009). Kaushal et al. (2010) show that the observed annual water temperatures at Solomons, MD have increased over the period from 1938 to 2006, with a relatively rapid increase around 1985. This increase in 1985 is not apparent in the data from TPL.

Correlations between the observed hypoxic volumes and observed percent duration of winds from the west and northwest are not significant, as they are in the model. There are some important differences between the modeled and observed winds. The overall magnitude of the observed summer winds at TPL is over 45% stronger than in the NARR model for the grid nearest TPL, on average. Additionally, the observations suggest that summer winds from the south are more common and stronger relative to other directions, than is captured by the model (Fig. 13). The observed hypoxic volumes are not significantly correlated with mean average summer wind speed from the model (NARR), suggesting that deficiencies in the modeled winds could degrade the prediction of hypoxic volume at inter-annual time scales.

Clearly, the lack of any biological variability is a major shortcoming of this simple model. However, deficiencies in the model's ability to accurately capture the hydrodynamics also adversely impact its ability to model oxygen. In an attempt to quantify the model's deficiencies, the residuals are examined. Here, the residuals are defined as the difference between the observed summer hypoxic volumes and the variability in the numerical model that is statistically correlated with the observations. A simple least squares fit between the observed summer hypoxic volumes and the numerical model results is done to determine and remove the variability in the observations that is correlated with the numerical results. The correlation between the residuals and various forcing parameters is given in Table 3. For forcing parameters that are not included in the model (like nitrogen load), or are based directly on observations (like river discharge), correlations are performed between the residuals and the observed variable. For the other variables (water temperature and wind speed and direction), the residuals are

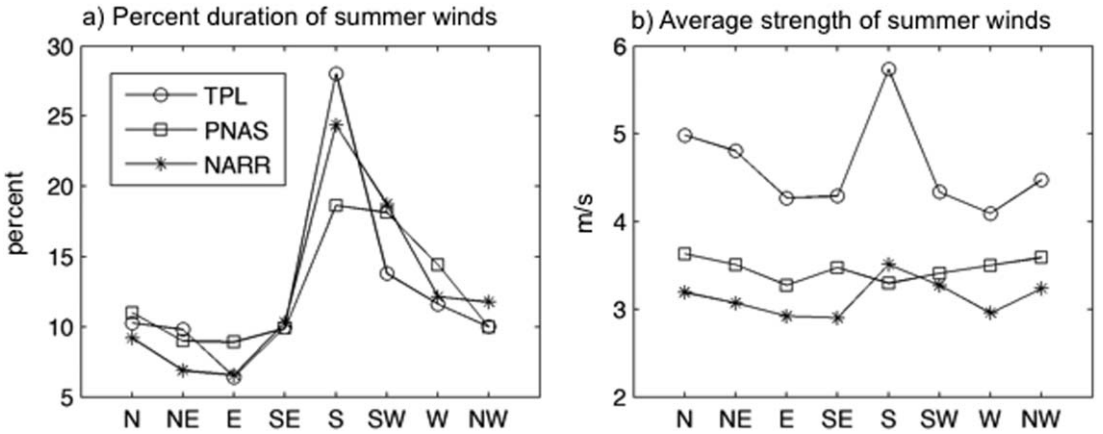

Fig. 13. Comparison of the (**a**) percent duration of summer (June–August) winds and (**b**) strength of summer wind as a function of wind direction for TPL, PNAS, and the NCEP NARR model. All winds have been adjusted to 10 m assuming neutral stability.

correlated with the difference between the observed and the modeled forcing. Because the observed wind speed and water temperature are measured at TPL, we restrict this analysis to the period when data from TPL were available (1986–2013).

For hypoxic volume < 2 mg L^{-1}, the residuals are not significantly correlated with the observed January–June nitrogen loading, suggesting that the lack of biological response to nutrient loading is not the primary reason the model and data disagree. In contrast, for anoxic volume there is a strong positive correlation between the residuals and nitrogen loading, demonstrating that much of the variability in the observations that is not captured by the model is due to biological processes. The residuals for both definitions of hypoxic volume are more strongly correlated with differences between the observed and modeled wind forcing than for nitrogen loading. One interpretation of this result is that the spatial and temporal resolution of the NARR modeled winds do not adequately capture the details of the wind forcing over Chesapeake Bay and that more accurate wind forcing would improve model prediction of hypoxic volume.

Given the dominant role that the summer wind speed plays in controlling hypoxia predicted by the model, it is worth briefly discussing the consistency between various observations of wind measured around Chesapeake Bay. Previous studies have found a dependence of hypoxic volume on wind direction, but have not found a significant correlation between summer hypoxic volume and mean summer wind speed (Scully 2010a; Murphy et al. 2011; Lee et al. 2013; Zhou et al. 2014). All of these studies have relied on the winds from PNAS, which are not measured over water. In addition to TPL (1986–present), wind speed measurements over water have been made at the Cove Point LNG Pier (NDBC station COVM2) since 2007, York River Light (YRL) (NDBC station YKRV2) since 2006, and at both the Rappahannock Light (RPL) (NDBC station RPLV2) and Chesapeake Bay Bridge Tunnel (CBBT) (NDBC station CBBV2) since 2005. While these records are much shorter than those from PNAS and TPL, it is instructive to compare the mean summer winds measured at these stations. As the correlations in Table 4 demonstrate, there is generally strong correspondence between the winds at TPL and the other stations that measure wind speed over water. In contrast, the correlations between summer wind speed at PNAS and all the stations that measure wind speed over water are either low, or in a number of cases, negative. In all cases the observed winds over water are negatively correlated with the observed hypoxic volumes, while the mean summer wind speed measured at PNAS exhibits essentially no correlation to the observed volumes. While interpreting the statistics of such short records must be done with caution, these data seem to indicate that the winds at PNAS do not capture the inter-annual variations in summer wind speed that are observed at the stations that measure wind speed over water.

All of the stations that measure wind speed over water demonstrate that the dominant summer wind direction is from south or southwest. Similarly, the dominant summer wind direction measured at PNAS is from the south (Fig. 13). At most of the stations that measure wind speed over water, the strongest winds during the summer are also from either the south or southwest. In contrast, at PNAS the strongest winds during the summer are from the north and northwest. At TPL, summer winds from the south are ~25% stronger than the other wind directions. At PNAS, summer winds from the south are ~5% weaker than the other directions. These results call into question the reliability of the winds measured at PNAS.

Discussion

The model used here assumes that the biological utilization of oxygen is constant in time and space and includes no biological production of oxygen. In Chesapeake Bay, primary production and respiration have clear temporal and

Table 3. Correlation coefficient (r) between model residuals for average summer (June–August) hypoxic volumes (< 2 mg L^{-1}, < 1 mg L^{-1}, and < 0.2 mg L^{-1}) for 1986–2013 and the January–June Susquehanna River discharge, the January–June Susquehanna River total nitrogen load, the difference between the TPL (observed) and NARR (modeled) June–August wind speed, the difference between TPL (observed) and simulated June–August water temperature, and the difference between TPL (observed) and NARR (modeled) percent duration of June–August winds from eight equally spaced compass direction. Significant correlations (< 0.05) are denoted with asterisk. Model residuals are defined as the difference between the observed summer hypoxic volumes and the variability in the numerical model that is statistically correlated with the observations.

	Jan–Jun Susquehanna River discharge	Jan–Jun Susquehanna nitrogen load	Jun–Aug Wind speed (TPL-NARR)	Jun–Aug Bay water temperature (TPL-model)	Percent duration summer wind from north	Percent duration summer wind from northeast	Percent duration summer wind from east	Percent duration summer wind from southeast	Percent duration summer wind from south	Percent duration summer wind from southwest	Percent duration summer wind from west	Percent duration summer wind from northwest
<2 mg L^{-1}	0.38*	0.32	−0.37	0.01	0.44*	0.04	0.25	0.07	−0.19	−0.06	−0.09	−0.54*
<1 mg L^{-1}	0.48*	0.41*	−0.30	0.02	0.38*	0.11	0.20	−0.01	−0.13	0.04	−0.10	−0.50*
<0.2 mg L^{-1}	0.61*	0.69*	0.19	−0.07	0.07	0.26	−0.15	−0.27	−0.29	0.25	0.30	0.00

spatial variability (e.g., Smith and Kemp 2014) and DO concentration in the euphotic zone often exceeds 100% saturation. The model used in this study does not account for any of these processes. Yet, a detailed skill assessment that compared numerous models to CBP DO data found no statistical difference in skill between models that used a full biogeochemical representation of oxygen dynamics and the simple model used here (Irby et al. 2016). This does not imply that these biogeochemical processes are not important, but that accurately modeling these complex processes is extremely difficult. It also highlights the important role that physical processes play in controlling the temporal and spatial distribution of oxygen in Chesapeake Bay. The model used here is not intended to accurately represent the biogeochemistry that drives hypoxia. Rather, the model is designed to try and isolate the contributions by physical processes.

While that is the intent, it is important to point out that there is an inherent coupling between biological and physical processes that may not be accurately accounted for in this model. Biogeochemical processes contribute to the evolution of oxygen gradients, which in turn will influence how both vertical mixing and horizontal advection supply oxygen to hypoxic regions. This is clearly illustrated by the results of Li et al. (2015) who demonstrate that seasonal variations in water column respiration rate significantly alter the horizontal advection of DO into the southern hypoxic zone of Chesapeake Bay. Although a complete evaluation of these complex interactions is beyond the scope of this paper, it is instructive to examine the sensitivity of the model results to the prescribed oxygen utilization rate. To that end, the 30-yr simulation was run using a rate of 2.1×10^{-4} $mmoleO_2\ m^{-3}\ s^{-1}$, a 50% increase over the base model run. Increasing the biological consumption of oxygen leads to substantial increases in the observed hypoxic volumes during the summer months (Fig. 14). The simulations with the higher utilization rate still predict a clear seasonal cycle with hypoxia largely absent during the winter months, highlighting the importance of physical processes on the seasonal cycle. The duration of hypoxia predicted by the model increases, with the months of April and October experiencing hypoxia more consistently than the simulations with the lower utilization rate.

Increases in hypoxic volume that are the result of the increased biological oxygen utilization are largely the result of the seaward expansion of the summer hypoxic zone (Fig. 15). By comparison, both the vertical and lateral changes in the hypoxic region are relatively modest (Fig. 16). The seaward expansion of the hypoxic zone in response to increased biological oxygen utilization is consistent with the model results of Testa et al. (2014). Their results suggest that higher nitrogen loads lead to increases in water column production and respiration in the seaward regions of the Bay during summer months, which led to the southward expansion of hypoxia. Interestingly, this is also the region where the

Table 4. Correlation coefficient (*r*) between mean summer (June–August) wind speed measured at various stations around Chesapeake Bay and the NARR model. Stations where wind speed is measured over water include Cove Point LNG pier (COV), YRL, RPL, CBBT, and TPL. Winds at PNAS are not measured over water. Data from the NARR model are taken from the grid location nearest TPL. The duration of available measurements is indicated for each station and correlations are based on available data. Bold font is used to highlight negative correlations.

COV (2007–2013)	YRL (2006–2013)	RPL (2005–2013)	CBBT (2007–2013)	TPL (1986–2013)	NARR (1984–2013)	
0.31	**−0.07**	**−0.28**	**−0.24**	0.24	0.51	PNAS (1984–2013)
	0.84	0.67	0.68	0.63	0.85	COV (2007–2013)
		0.86	0.80	0.81	0.81	YRL (2006–2013)
			0.69	0.81	0.68	RPL (2005–2013)
				0.55	0.44	CBBT (2007–2013)
					0.59	TPL (1986–2013)

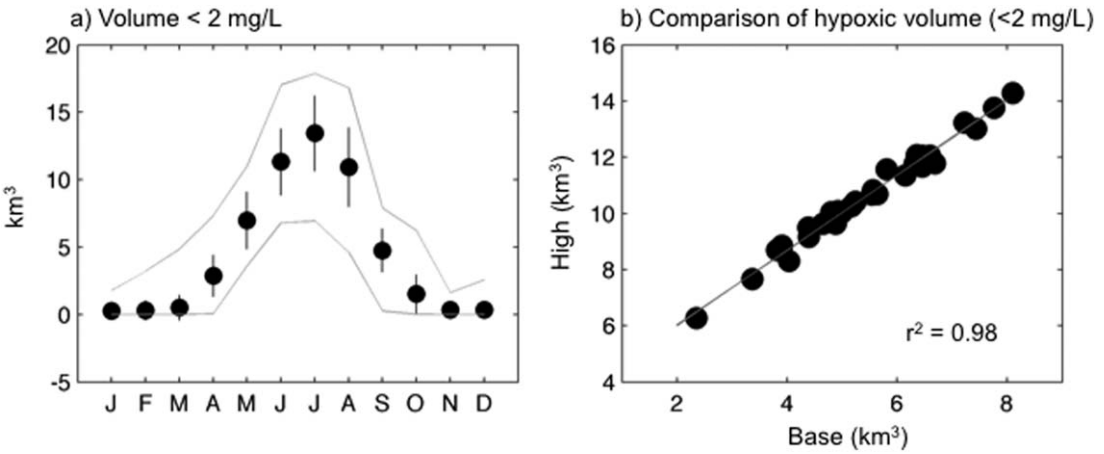

Fig. 14. (**a**) Predicted monthly averaged hypoxic volume (< 2 mg L^{-1}) for the 30-yr simulation where the oxygen utilization rate is increased to 2.1 × 10^{-4} mmoleO_2 m^{-3} s^{-1}. Vertical lines represent ± 1 standard deviation and gray lines represent maximum and minimum monthly averages. (**b**) Comparison of the summer (June–August) hypoxic volume (< 2 mg L^{-1}) for the base simulation where the oxygen utilization rate is 1.4 × 10^{-4} mmoleO_2 m^{-3} s^{-1} (*x*-axis) with the simulation where the oxygen utilization rate is increased to 2.1 × 10^{-4} mmoleO_2 m^{-3} s^{-1} (*y*-axis). The best fit linear regression is shown by the fitted line. Reported correlation is significant at $p < 0.01$.

inter-annual variability due to physical processes is greatest (e.g., Figs. 11, 12). In both the vertical and across Bay directions, there are very strong gradients in the vertical supply of oxygen via turbulent mixing that are associated with the location of the pycnocline. As a result, even relatively large changes in biological oxygen utilization do not result in large changes in the vertical or lateral location of hypoxic water during the summer. In contrast, the southern limit of the hypoxic zone is characterized by relatively weak horizontal gradients in oxygen (e.g., Fig. 7), which presumably are associated with weak gradients in the physical supply. Thus, small changes in either the physical supply of oxygen or its biological utilization will result in large horizontal excursions of the location of the hypoxic zone.

Despite the fact that increasing the oxygen utilization rate changes the physical distribution of DO (e.g., Figs. 15, 16), the predicted inter-annual variability in hypoxic volume remains virtually unchanged when the biological utilization in the model is increased. The correlation between the 30-yr time series of summer hypoxic volume < 2 mg L^{-1} for the simulations with high and low biological utilization is extremely high ($r^2 = 0.98$) (Fig. 14). The other thresholds have similarly high correlations. While this does not directly address the role of spatial variations in biological processes, it does suggest that the inter-annual variability driven by physical processes is not sensitive to the spatial distribution of DO.

The conclusion of this paper is not that biological variability is unimportant. The sensitivity to the imposed oxygen utilization rate clearly highlights that the biogeochemical response to nutrient loading is the underlying process that results in hypoxia. Inter-annual variations in this

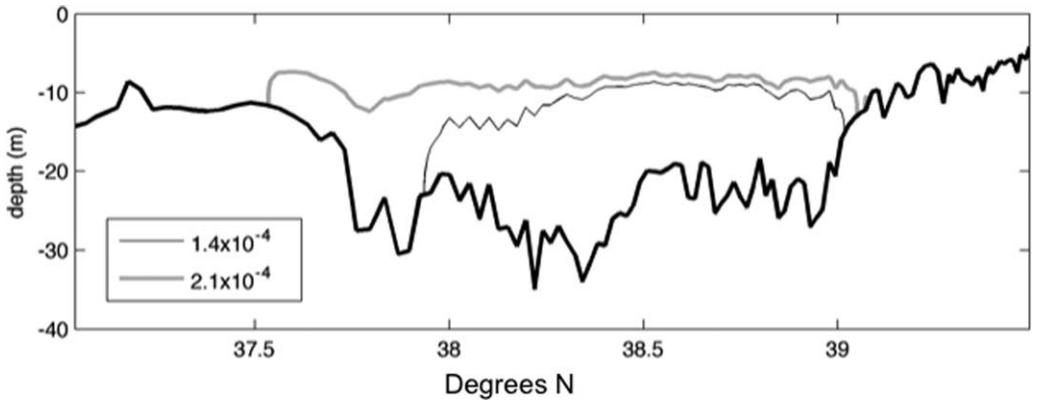

Fig. 15. Comparison of thalweg location of the mean summer (June–August) 2 mg L^{-1} oxygen contour for the base model run with a oxygen utilization rate of 1.4×10^{-4} mmoleO_2 m^{-3} s^{-1} (thin black line) with the 30-yr simulation where the oxygen utilization rate was increased to 2.1×10^{-4} mmoleO_2 m^{-3} s^{-1} (thick gray line).

rate will clearly contribute to inter-annual variations in hypoxic volume. The results from the correlation analysis presented above clearly demonstrate that inter-annual variations in the observed anoxic volumes are strongly related to nutrient loading. However, the results presented above also demonstrate that observed hypoxic volumes are statistically related to both nutrient loads and physical forcing, and a simple model with no biological variability captures over half the observed variance in hypoxic volume for the months of July and August for the period 1984–2013. From a management perspective, quantifying the role of physical processes is important to a better understanding of the relationship between hypoxic volume and nutrient loading. Physical processes are not the only reason for the decoupling between nutrient loading and hypoxia. Kemp et al. (2009) conclude that a number of both physical and ecological processes can shift the relationship between hypoxia and nutrient loading, and that hypoxia in large open systems like Chesapeake Bay responds to changes in nutrient loading in nonlinear ways.

Historical data document that the observed hypoxic volumes normalized by spring nitrogen loading increased significantly in the early 1980s (e.g., Hagy et al. 2004). This shift could be the result of fundamental changes to the ecosystem, long-term changes in the atmospheric forcing, or both (Kemp et al. 2009). Atmospheric forcing from the NARR model only is available beginning in 1979. Without accurate atmospheric forcing prior to 1979, it is not possible to use this model to assess the role that changes in physical forcing may have played in contributing to this increase. Scully (2010a) documented that decadal-scale oscillations in the strength of the summer Bermuda high pressure system favored greater incidence of summer winds from the west over Chesapeake Bay beginning around 1980. It was hypothesized that this shift contributed to the increase in hypoxic volume that occurred in the early 1980s. The model results presented above support the occurrence of greater hypoxic volume in association with increased duration of winds from the west. However, both wind speed and river discharge explain more of the modeled variability than differences in wind direction. Thus, it is unlikely that this model would capture the large increases in hypoxic volume that occurred around 1980, but longer simulations with accurate atmospheric forcing could better address this hypothesis.

Conclusions

The results presented above suggest that a relatively simple model with no biological variability can capture roughly half of the observed variability in hypoxic volume for the months of July and August over the period 1984–2013. Model skill increases during the summer, peaking in August suggesting physical processes play a more important role in modulating hypoxia later in the summer. Model skill is much better for hypoxic volumes than for anoxic volumes. In fact, a simple regression based on the integrated January–June Susquehanna River nitrogen load can explain more of the variability in anoxic volume than the model presented here. Our interpretation of these results is that physical forcing plays a much more important role in controlling hypoxic volumes than anoxic volumes in Chesapeake Bay. Our results support the notion that the biologic response to the delivery of nutrients via river discharge in the spring leads to the development of anoxic water in summer. However, physical processes result in mixing at the edges of this anoxic zone and appear to play an important role in controlling the extent of hypoxic water. This mixing is primarily done by the wind. Previous studies have failed to document the importance of summer wind speed because they have relied on winds measured at PNAS, which does not capture the observed inter-annual variations in wind speed that are

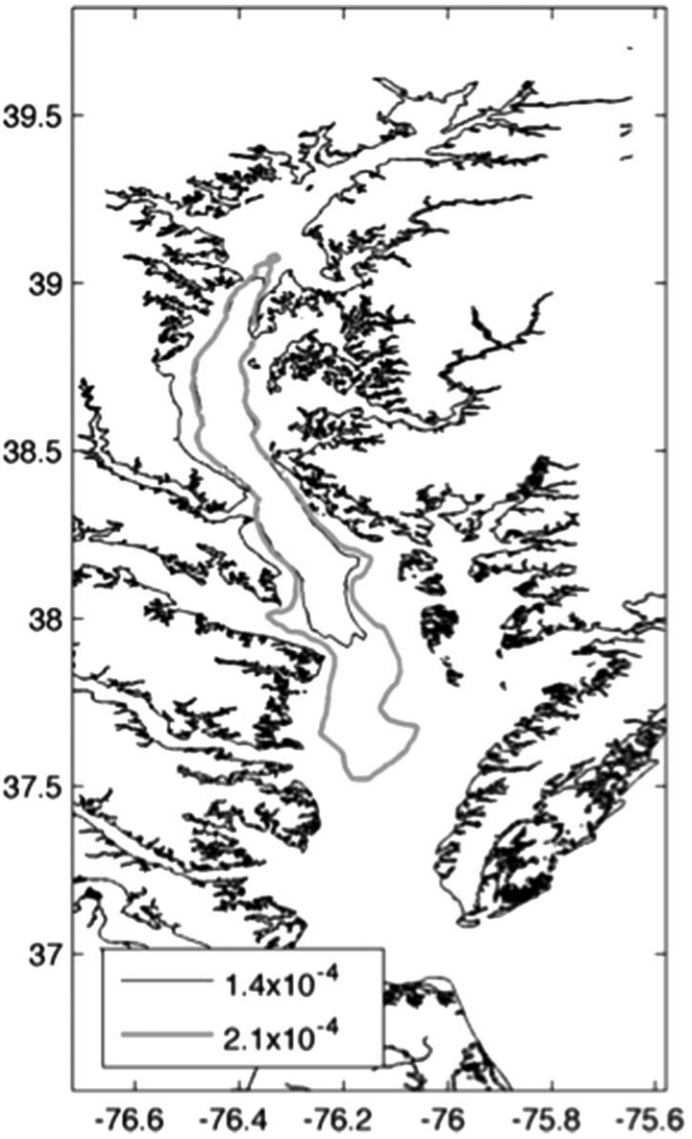

Fig. 16. Comparison of the location of the mean summer (June–August) 2 mg L^{-1} bottom oxygen contour for the base model run with a oxygen utilization rate of 1.4×10^{-4} mmoleO_2 m^{-3} s^{-1} (thin black line) with the 30-yr simulation where the oxygen utilization rate was increased to 2.1×10^{-4} mmoleO_2 m^{-3} s^{-1} (thick gray line).

observed by stations that directly measure wind over the waters of Chesapeake Bay.

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Conflict of Interest

None declared.

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