

# Relative Importance of Nutrient Load and Wind on Regulating Interannual Summer Hypoxia in the Chesapeake Bay

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**Abstract** To analyze the correlations of summer anoxia/hypoxia in the Chesapeake Bay with watershed input and wind conditions, statistics were applied to nearly three decades of monitoring data. The results of Pearson correlation coefficients, multivariate regression analysis, and cluster analysis indicate that anoxia/hypoxia has a strong positive correlation with nutrient load and a moderate negative correlation with wind speed. Physical relationships among the relevant constituents were analyzed. Nutrient loads and the subsequent decay of organic matter are the primary factors that control the oxygen demand causing summer anoxia and hypoxia, while episodic wind can partially erode stratification and reduce anoxia/hypoxia. Although the extent of anoxia/hypoxia reduction differs with wind direction, higher wind speeds result in more destratification and anoxia/hypoxia reduction than lower wind speeds and are more important than the effect of wind direction. The influences of freshwater discharge, stratification, and temperature were also analyzed. Computer modeling results were used to obtain dissolved oxygen conditions at finer temporal and spatial scales to supplement the scattered and discrete observations from monitoring stations and for better understanding of anoxia/hypoxia development under episodic wind events.

**Keywords** Hypoxia and anoxia · Nutrient load · Freshwater discharge · Wind speed · Wind direction · Destratification · Chesapeake Bay

## Introduction

Summer hypoxia and anoxia in the Chesapeake Bay threatens Bay living resources. Excessive nutrient loads in the winter and spring have impacts on algal blooms in the spring and summer (Officer et al. 1984; Harding et al. 1992). After and during bloom conditions, dead algae increase oxygen demand. Hypoxia or anoxia begins to appear in the deep water of the Bay in late spring or early summer when water temperatures and stratification increase and diminishes in fall and winter when temperatures decrease and stratification weakens (Testa and Kemp 2012). Nutrient and freshwater inputs to the Bay can have a residence time of several months or longer in the estuary (Kemp et al. 2005), and the winter-spring inputs can affect the eutrophication processes through the summer. Correlation of winter-spring, i.e., January to May, nutrient load and summer, i.e., June to September, hypoxia has been recognized (Hagy et al. 2004; Wang et al. 2006). Controlling nutrient load inputs to the Chesapeake has been the focus of management actions to reduce hypoxia (CEC 2000; USEPA 2010).

On the other hand, physical processes such as destratification by wind (Kato and Phillips 1969) can increase dissolved oxygen (DO) in deep water and reduce hypoxia as observed in the Chesapeake Bay (Malone et al. 1986; Scully 2010a; Testa and Kemp 2012), Long Island Sound (O'Donnell et al. 2008), and other estuaries or coastal zones (DiMarco et al. 2010). For example, despite New York City's upgraded nitrogen control of point sources, the long-term decline in July/August summer minima bottom DO concentration in western Long Island Sound was explained to be due

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to the decreased wind ventilation of bottom DO in later years (Wilson et al. 2008). Based on statistics on 1950–2007 data, Scully (2010b) found that more of the interannual variation in the volume of hypoxic water of the Chesapeake Bay was explained by wind direction than any other variable, emphasizing wind direction in mediating the variation of interannual hypoxia in the Bay. A study based on 1985–2012 data by Wang et al. (2013a) suggested nutrient loads to be more important than wind in controlling interannual summer hypoxia, though both nutrient load and wind are important.

Researchers have also recognized that the effect on hypoxia varies by wind direction (Scully et al. 2005; Chen and Sanford 2009; Scully 2010a; Li and Li 2011; Wang et al. 2013a). Because the Chesapeake's main channel is north–south oriented, the south and north winds have longer fetches and greater influence in reducing hypoxia than west or east winds. Note that a south wind is defined in this article as a wind that originates in the south and blows north. The partially mixed estuary has a net flow seaward (to the south) in surface water and up-estuary (to the north) of bottom saltier water (Prichard 1967). The south wind blows against the net flow direction of the surface current and thus causes greater destratification than the north wind (Li and Li 2011), and at the scale of the entire Bay, a south wind generally reduces more anoxia (Scully 2010a) and has a greater impact on the central Bay's anoxic waters (Wang and Wang 2012) in episodic wind events. Scully (2010a, b) observed sustained high hypoxia in the Chesapeake Bay in the past decade despite nutrient load reductions in recent years and attributed its cause to be due to a shift of wind direction from more south-southeasterly to more westerly. Scully (2010b) emphasized wind direction over wind speed and concluded that slight changes in wind direction can have significant impacts on water quality. In contrast, Wang et al. (2013a) showed that in most cases, wind speed is more important than wind direction in modulating hypoxia, though the extent of hypoxia reduction differs with different wind direction for the same wind speed and duration.

The relative importance of nutrient loads and wind on Chesapeake hypoxia (including anoxia) has been given attention recently because it has significant implications to management of Chesapeake water quality (Scully 2010a; Murphy et al. 2011; Lee et al. 2013). A clear understanding of the factors controlling the interannual variations of summer hypoxia is needed. This paper provides statistical analyses of the relative effects on Chesapeake hypoxia by nutrient loads, wind speed, and wind direction using nearly three decades of observed data. Other factors besides nutrient loads and wind that may affect hypoxia such as stratification and temperature were also evaluated.

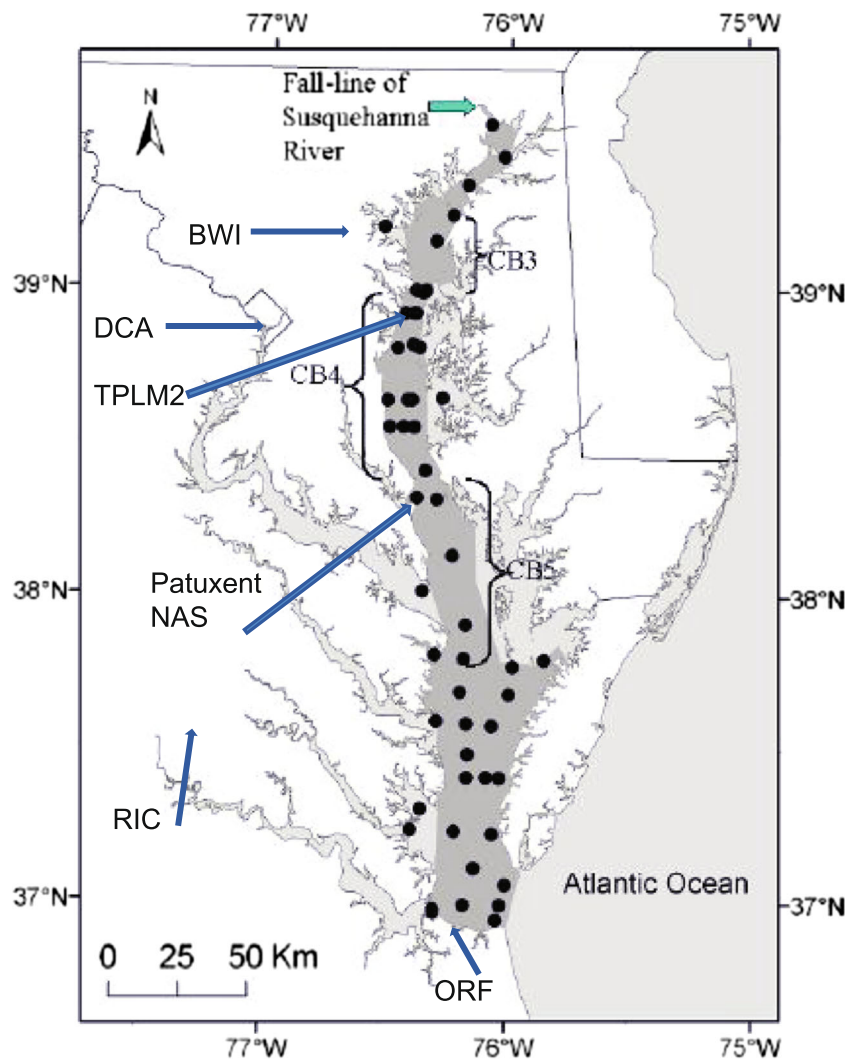
## Methods

### Data Collection

**Hypoxia Assessments** There are about 100 long-term monitoring stations in the Chesapeake estuary for DO, salinity, water temperature, and other water quality parameters, sampled at 1-m depth intervals once or twice each month since 1985 by the Chesapeake Bay Program. About 50 stations in the mainstem Bay and the lower end of tidal tributaries (Fig. 1) were selected to interpolate DO concentration spatially over the entire mainstem Bay for individual monitoring cruises using the modified method of Wang et al. (2006) from the Chesapeake Bay 3D interpolator (Bahner 2001). Only the cruises that sampled at least 40 of the 50 stations and included all main deep-channel stations were utilized. Generally, six cruises were selected for each summer: one in late June or early September and two in July or August. Three levels of hypoxia, i.e., DO <0.2 mg/l (near anoxia), <1 mg/l (severe hypoxia), and <2 mg/l (moderate hypoxia), were assessed (Hagy et al. 2004; Scully 2010a; Murphy et al. 2011) in the summer months of June to September. Depending on the reported detectable DO concentrations, some sampling cruises may have an estimated zero anoxic volume when using a DO <0.2-mg/l limit but nonzero estimated anoxic volume when using DO ≤0.2 mg/l (Wang et al. 2006). Therefore, DO ≤0.2 mg/l was used (Jasinski et al. 2005) to define the near anoxia condition and hereafter calling it “anoxia” in this paper. The water volumes for the three levels of hypoxia, i.e., DO ≤0.2, <1, and <2 mg/l, are labeled as AV, HV1, and HV2, respectively. If there is no DO threshold associated with the word hypoxia in the text, then hypoxia is meant in the broadest sense, i.e., all three levels of hypoxia including anoxia. This paper focuses on the lowest level of hypoxia, i.e., anoxia, in the mainstem Bay.

The Chesapeake Bay Water Quality and Sediment Transport Model (WQSTM) (Cercio et al. 2010) was used to estimate hypoxia to supplement the scattered observations. The WQSTM is a peer-reviewed and approved regulatory model used for the Chesapeake Bay water quality management. A model cell approximately represents 1 km×1 km horizontally with 1.5-m depth. Its hydrodynamic module simulates estuarine circulation, considering freshwater input and wind effects. The water quality module simulates 36 state variables including various species of nutrients, three types of phytoplankton, and related biochemical processes. Model forcing functions include daily flow and nutrient loads from the watershed model (USEPA 2010; Shenk and Linker 2013) and hourly wind from five stations including (1) the Patuxent Naval Air Station (Patuxent NAS), (2) Baltimore-Washington International Airport (BWI) or Thomas Point, MD (TPLM2), (3) Norfolk International Airport (ORF), (4) Reagan Airport (DCA), and (5) Richmond International Airport (RIC). The

**Fig. 1** Chesapeake Bay estuary with long-term monitoring stations (solid circles) used to calculate anoxic/hypoxic volumes for the mainstem Bay



errors of estimated DO in the mainstem are 0.3 and  $-0.45$  mg/l at depths of 6.7–12.8 m and greater than 12.8 m, respectively. The conditions of 1991–2005 were simulated. Hourly hypoxic volumes of the Bay were calculated by adding volumes of the model cells that had hourly average DO under the three hypoxia threshold levels. Daily hypoxic volumes (of the three levels) were averaged from the daytime period of 9 a.m. to 3 p.m. to better compare with the collected times of the observed monitoring data. The suffix “\_mod” is used for modeled anoxic/hypoxic volumes, AV, HV1, and HV2, otherwise the anoxic/hypoxic volume estimates are from observation. In some cases, the suffix “\_obs” is also used for observed for clarity purposes.

**Strength of Stratification and Deep-Water Temperature** Salinity and water temperature were also measured during the sampling cruises. Water density,  $\rho$ , is calculated using the SigmaT method developed by Woods Hole Oceanographic Institute (<http://globec.whoi.edu/globec-dir/sigmat-calc-fortran.html>). The square of the Brunt-Vaisala

frequency (Knauss 1997) or  $N^2$  is calculated for each 1-m interval:  $N^2 = (g/\rho)(d\rho/dz)$ , where  $\rho$  is density,  $g$  is the acceleration of gravity, and  $z$  is depth. The maximum  $N^2$  in a vertical profile is used to represent stratification or pycnocline strength, similar to the approach of Murphy et al. (2011). Station CB4.1C is at the center of the Bay’s hypoxic region and is one of the Bay’s deepest monitoring stations. Stratification strength (SS) and water temperatures in deep water (DWtmp), i.e., depths  $> 20$  m, at station CB4.1C were obtained from the bi-weekly monitoring data.

**Wind Fields and Air Temperatures** Hourly wind and daily air temperature observed at the Patuxent NAS were collected from NOAA National Climate Data Center. Missing wind data of less than 12 h were filled in by interpolation from observed values, while missing data of greater than 12 h were filled with the observations from BWI. The counts, i.e., frequency, of winds in individual hours for speeds greater than certain thresholds and/or from certain directions were obtained. The aggregate winds that have a common character, such

as in certain directions and/or certain speeds, in a defined period were called *wind events*. The winds from eight directions, i.e., N, NE, E, SE, S, SW, W, and NW, were filtered according to the compass degrees between  $-22.51 \sim 22.50$ ,  $22.51 \sim 67.50$ ,  $67.51 \sim 112.50$ ,  $112.51 \sim 157.50$ ,  $157.51 \sim 202.50$ ,  $202.51 \sim 247.50$ ,  $247.51 \sim 292.50$ , and  $292.51 \sim 337.5$ , respectively. This paper analyzed three types of wind counts, including (1) counts of hourly winds in all directions with speeds greater or equal to a particular value, (2) counts of hourly winds from only a particular direction at all speeds, and (3) counts of hourly winds from only a particular direction, and with wind speeds greater or equal to a particular speed (Table 1, abbreviations 11 to 13). Wind data from Thomas Point were also analyzed, and the results of the correlation analyses on anoxia and wind speeds and directions were consistent with the Patuxent NAS results.

**Watershed Discharge and Nutrient Loads** Daily mean flows at the Conowingo station of the Susquehanna River were collected from the USGS National Water Information System. Based on the bi-weekly total nitrogen (TN) and total phosphorus (TP) measurements and

daily mean flows at the station, daily TN and TP loads from the Susquehanna River were estimated using the method of Wang and Linker (2008). In the correlation analysis, TN and TP loads were composed into a single variable, the *nutrient load index* (Koroncai et al. 2003; Jasinski et al. 2005; Wang et al. 2013b), which equals  $(TN+10TP)/2$  and is labeled SNP. Annual “winter-spring” flow or load is used which is the average flow or load from January through May.

#### Data Analysis on Factors Impacting Hypoxia

Time-series plots were used to visually inspect the variations of hypoxic volume and its influencing constituents. Statistics were applied to analyze correlations among them. Most of the statistics were applied to data in the period 1991–2005 because consistent observed data for most variables of interest for this period can be obtained. There were also modeled DO data available for this period as well. Statistics on extended periods, e.g., 1985 to 2012 and 1950 to 2001 periods, were also conducted. Table 1 lists abbreviations for the constituents in the statistics.

The correlations of hypoxia with its influencing factors, or constituents, were conducted separately for summer average hypoxia and July hypoxia. For the constituents representing the antecedent hypoxia events, such as watershed inputs of flow and nutrient loads, the values of their January to May average were used in the analyses of both summer hypoxia and July hypoxia. On the other hand, for the constituents that reflect the Chesapeake’s ambient condition, such as stratification, wind, and temperature, their average values in summer were used for the statistics of summer hypoxia, and average July values were used for July hypoxia statistics.

Cluster analyses were conducted on anoxic volume and its possible influencing variables, such as flow, nutrient load, stratification strength, temperature, average wind speeds, and frequency of wind direction. Tree diagrams were generated based on the *proportion of variance explained* among the variables to show the affinities among them, which supplemented the correlation analysis.

Linear regressions were performed using the observed anoxic volume in the summer or in July as the dependent variable (called the estimator). Independent variables (called regressors) were winter-spring nutrient loads plus other influencing constituents such as the strength of stratification, wind speed, and air or deep-water temperature in the summer or in July. The SAS software was used for the statistical analysis. Augmenting the methods here and the results presented below is an additional online [supplementary material](#) with results from additional statistic tests.

**Table 1** Description of symbols

	Abbreviations	Explanation of abbreviations
1	AV_obs	Observed anoxic volume (for $DO \leq 0.2$ mg/l)
2	HV1_obs	Observed hypoxic volume for $DO < 1.0$ mg/l
3	HV2_obs	Observed hypoxic volume for $DO < 2.0$ mg/l
4	AV_mod	Modeled anoxic volume (for $DO \leq 0.2$ mg/l)
5	HV1_mod	Modeled hypoxic volume for $DO < 1.0$ mg/l
6	HV2_mod	Modeled hypoxic volume for $DO < 2.0$ mg/l
7	Flow	Flow from the Susquehanna River ( $m^3$ ) (Jan–May average)
8	SNP	Nutrient load index from Susquehanna River (Jan–May average)
9	Wspd	Average wind speed (in the summer or individual months) for all speeds
10	Wspd#	Average wind speed for speeds $\geq \#$ m/s (in the summer or individual months)
11	Cwd#	Counts of hourly winds (in all directions) for speeds $\geq \#$ m/s
12	c*	Counts of hourly winds blowing from *, e.g., N, direction for all speeds
13	c*#	Counts of hourly winds blowing from * direction for speeds $\geq \#$ m/s. For example, cN5 is counts of wind events blowing from the north that have speeds $\geq 5$ m/s.
14	SS	Strength of stratification
15	DWtmp	Average deep-water temperature (in $^{\circ}C$ ) in depth $> 20$ m
16	Atmp	Average air temperature (in $^{\circ}C$ )



## Results

### Time-Series Plot of Hypoxia Variation and Its Influencing Constituents

**Interannual Variation of Summer Hypoxia and Other Constituents** Figure 2 plots the summer average anoxic volume ( $\text{km}^3$ , for  $\text{DO} \leq 0.2 \text{ mg/l}$ ) and hypoxic volume ( $\text{km}^3$ , for  $\text{DO} < 1 \text{ mg/l}$ ), winter-spring watershed discharge and nutrient load index, and summer average wind speeds in years 1985 to 2012. The interannual variations of summer hypoxic and anoxic volumes are comparable to those by Jasinski (2003); MASC (2006); IAN UMCES (2010), and MDDNR (2012). The fluctuation of summer anoxia and hypoxia is consistent with the variation of the winter-spring nutrient loads or flow, suggesting that nutrient loads are an important factor influencing summer anoxia and hypoxia (Officer et al. 1984).

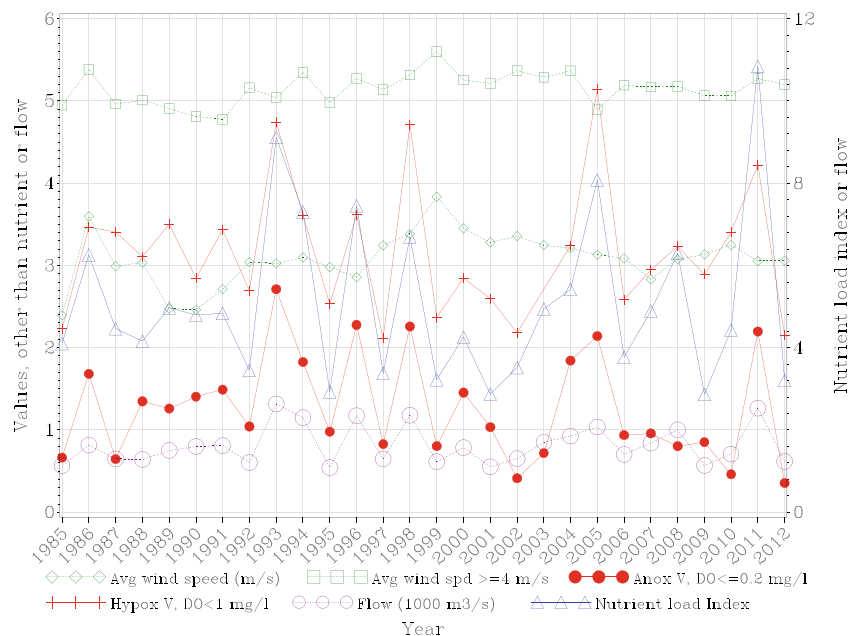
**Anoxic Volumes in Finer Time Steps and the Associated Wind** The open circles in Fig. 3 are observed anoxic volume ( $\text{km}^3$ , for  $\text{DO} \leq 0.2 \text{ mg/l}$ ) in the summer (June through September) sampling cruises during the years 1991–2005. The crosses represent modeled daily anoxic volume during daylight hours in the summer, which are mostly in agreement with the observed anoxic volumes. The observed anoxic volumes are obtained through interpolation of field DO measurements from scattered monitoring stations during the 4 to 7 days of a sampling cruise. Therefore, the anoxic volumes derived from the two methods are not directly comparable. The small dots signify wind speed  $\geq 6 \text{ m/s}$ . Usually, July and August had higher anoxia than June and September, which could be related to higher temperatures and/or weaker winds. In some years, August had higher anoxia volume than July did, such as

1996 and 2005 when August had weaker winds. In some years, July had higher anoxia such as in 1993, 1998, and 2004, when July had weaker winds.

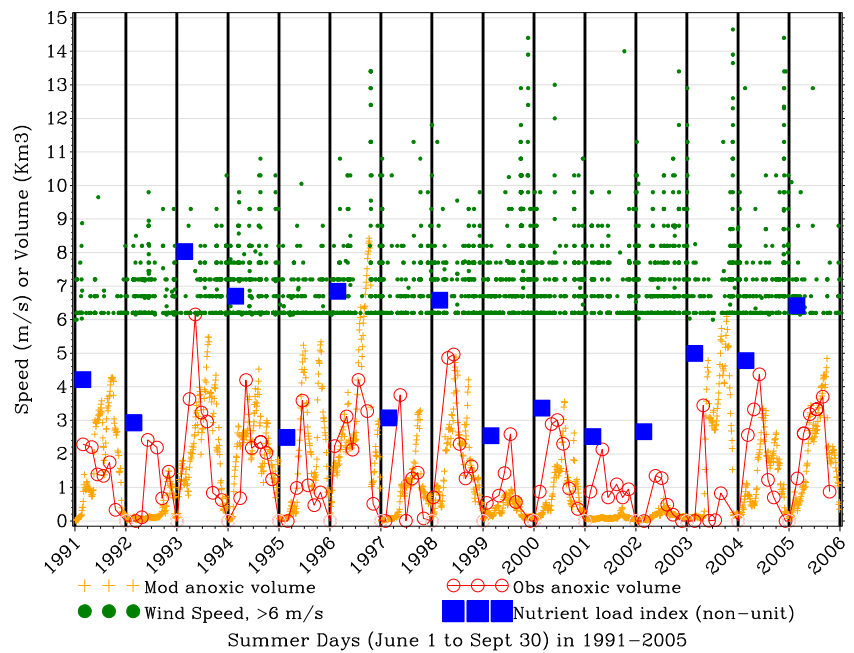
Figure 4 focuses on 1995–1998. The year 1996 had about twice nutrient load as either 1995 or 1997, but 1996 had a lower July average hypoxic volume of  $\text{DO} < 1 \text{ mg/l}$  (Fig. 4). This could be due to wind effect, i.e., July 1996 had more frequent strong winds with speeds of  $6 \text{ m/s}$  or greater (accounting winds in all directions). This is further shown in Fig. 5 where the wind events of speeds  $> 4$  and  $> 8 \text{ m/s}$  were more frequent in July 1996 (Fig. 5, B2) than they were in July 1995 (Fig. 5, A2) or July 1997 (Fig. 5, C2). Meanwhile, for the deeper pool of waters with anoxia ( $\text{DO} \leq 0.2 \text{ mg/l}$ ), July 1996 had higher anoxic volume. This means that the wind had less effect on deep channel water and its anoxic volume, and thus, July 1996 still had a greater anoxic volume than July 1995 or 1997. On the other hand, June of 1996 (Fig. 5, B1) had fewer high wind events than June of 1995 (Fig. 5, A1) or 1997 (Fig. 5, C1). This led June 1996 to have significantly higher hypoxic ( $\text{DO} < 1 \text{ mg/l}$ ) and anoxic volumes than June of 1995 and 1997 (Fig. 4). Hence, the average summer hypoxic and anoxic volumes were higher in 1996 than in 1995 and 1997 (Fig. 2), which were controlled mainly by nutrient loads, but the variable effects of wind on anoxia or hypoxia can be seen over individual sampling cruises (Fig. 3) or individual month values (Fig. 4).

Years 1996 and 1998 had similar winter-spring nutrient loads. The anoxic or hypoxic volume in July 1998 was about twice that in July 1996 (Fig. 4). July 1998 had fewer strong wind events (Fig. 5, D2) than July 1996 (Fig. 5, B2) did, but July 1998 had more southerly winds, an indication that wind speed can be more important than direction with respect to its influence on hypoxia.

**Fig. 2** Summer average anoxic and hypoxic volumes, wind speed, and winter-spring watershed nutrient load and freshwater discharge in 1985–2012



**Fig. 3** Observed anoxic volume ( $\text{DO} \leq 0.2 \text{ mg/l}$ , open circles), modeled daily anoxic volume (crosses), and hourly wind speed ( $\geq 6 \text{ m/s}$ , small dots) in summer months (June–September, as the four minor tick-marked intervals) in 1991–2005. There are six observed anoxic volumes and six hypoxic volumes in a summer; the first is in late June, the second and the third are in July, the fourth and the fifth are in August, and the sixth is in early September. The squares denote the averaged winter-spring nutrient load index (unitless)



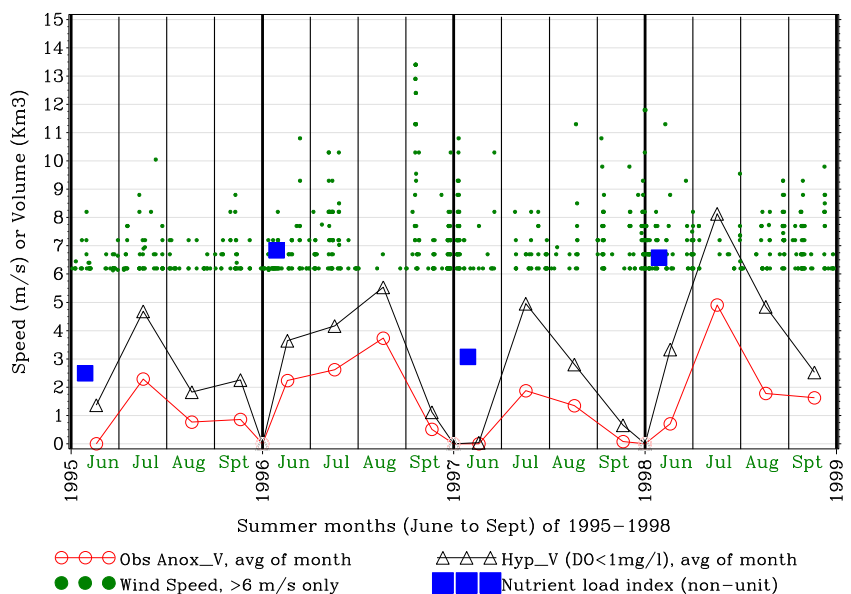
#### Statistics of Anoxic Volume and Its Influencing Constituents Based on 1991–2005 Data

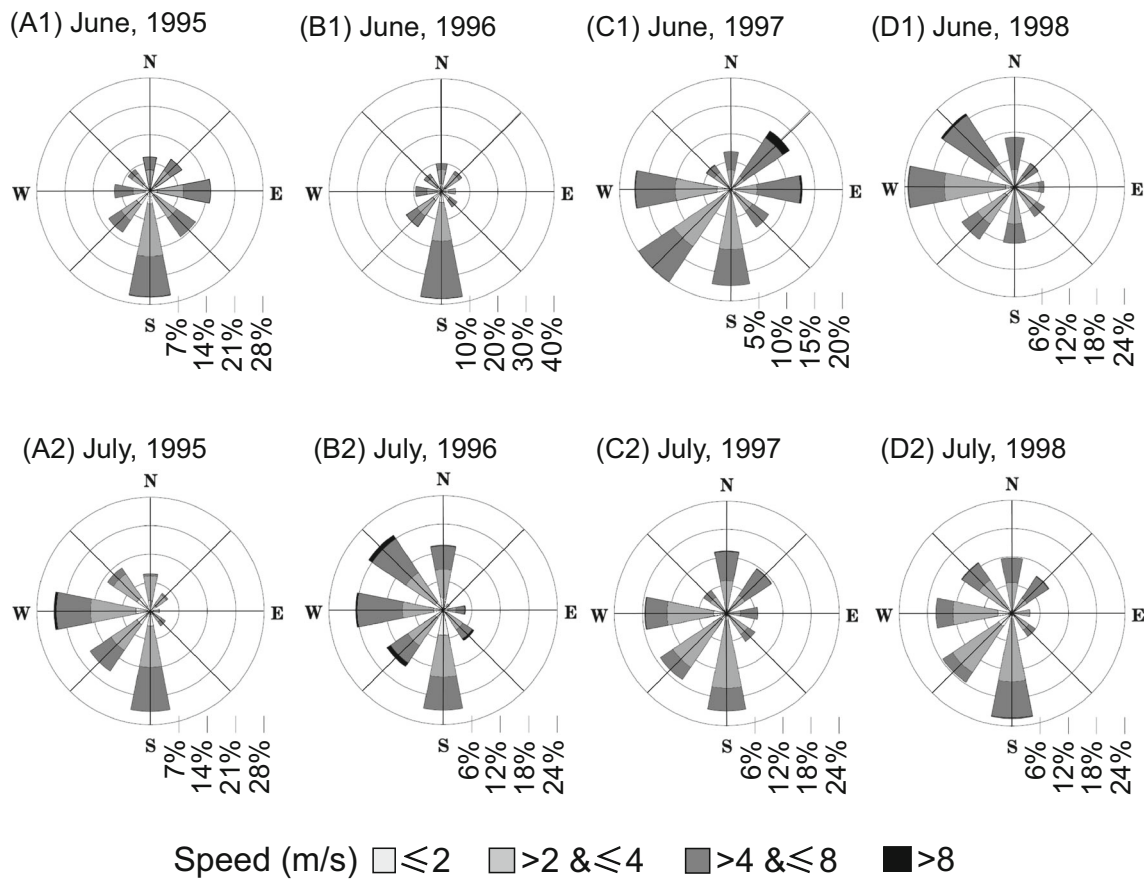
Table 2 lists correlation coefficients ( $r$ ) of July and summer anoxic volume with its influencing constituents based on 1991–2005 data ( $n=15$ ). The  $p$  values are not listed in the tables of correlation coefficient (Tables 2, 3 and 4) but can be generalized as the following:  $p < 0.002$  in the  $r > 0.7$  cases;  $p < 0.05$  in the  $r > 0.5$  cases;  $p < 0.25$  in  $r = 0.3 \sim 0.49$  cases;  $p$  reaches 0.4 at low  $r$ . Thus, there is greater confidence on correlations with  $r > 0.7$ . Note that the observed anoxic volume, strength of stratification, and water

temperatures have only one or two observations in a month, while other components are averaged from daily or hourly observations.

**Correlation of Anoxic Volume with Nutrient Load and Freshwater Discharge from the Watershed** The summer anoxia is highly correlated ( $r=0.89$ ) with the winter-spring nutrient load, a finding which is in agreement with Hagy et al. (2004), indicating that a significant effect on summer anoxia is due to the winter-spring watershed load. The correlation between summer anoxic volume and average wind speed (Wspd) is  $-0.37$ .

**Fig. 4** Observed monthly anoxic volume ( $\text{DO} \leq 0.2 \text{ mg/l}$ , open circles) and hypoxia volume ( $\text{DO} < 1 \text{ mg/L}$ , triangles), and hourly wind speed ( $\geq 6 \text{ m/s}$ , small dots) in summer of 1995–1998. The monthly anoxic/hypoxic volumes are the average from one or two sampling cruises in the month (Fig. 3). The squares denote the averaged winter-spring nutrient load index (unitless)





**Fig. 5** Wind roses for June and July of 1995–1998

Nutrient load is the product of concentration and flow and is highly correlated to discharge flow (Cohn et al. 1992). The correlation between the winter-spring flow and the winter-spring nutrient load is strong ( $r=0.9$ , not tabulated). Therefore, the strengths of correlations between anoxic volume and flow or nutrient load are similar (Table 2). Thus, flow can be used to substitute for nutrient load to analyze the effect on hypoxia in the Chesapeake. In addition, flow is easier to obtain than nutrient loads, and its seasonal or annual values are usually estimated more accurately because of daily, or more frequent, observations of flows. In the correlation analysis in the extended period when complete nutrient load information is unavailable, flow is used as a substitute for load. In physical processes, freshwater inflow also influences hypoxia through stratification.

**Correlation of Anoxic Volume with Stratification Strength** There is weak correlation ( $r=0.29$  to  $0.5$ ) between monthly average flow and CB4.1C's stratification strength in the same month or with a lag of 1 to 3 months (table omitted for brevity) and also between January and May average flow and CB4.1C's stratifications strength in late spring to mid-summer months of the year (table omitted for brevity). The winter-spring flow had a considerable effect on stratification in early summer but a weak effect in late summer, consistent with the findings of Murphy et al. (2011) who used average stratification strength in the mainstem. The summer anoxic volume has a low correlation with stratification ( $r=0.23$ ) but has a stronger correlation with winter-spring river discharge ( $r=0.89$ ) and nutrient loads ( $r=0.89$ ) (Table 2). The high correlation of the winter-spring flow and the summer anoxic volume is mainly due to the effect by the flow-related nutrient

**Table 2** Correlations of anoxic volume (AV) with its influencing constituents based on 1991–2005 data ( $n=15$ )

		Jan–May average		July or summer average					
		SNP	flow	Wspd	Wspd5	Cwd4	SS	DWtmp	Atmp
AV_Obs	Summer	0.89	0.89	−0.37	−0.25	−0.42	0.23	−0.15	0.33
	July	0.75	0.82	−0.36	−0.10	−0.42	0.04	−0.20	0.17

**Table 3** Correlation coefficients of summer anoxic volume with counts of summer wind events that are above certain speed thresholds in 1991–2005 ( $n=15$ )

	Wspd	Cwd2	Cwd3	Cwd4	Cwd5	Cwd6	Cwd7	Cwd8
AV_obs	-0.37	-0.28	-0.41	-0.42	-0.33	-0.24	-0.21	-0.39
Wspd	1.00	0.48	0.67	0.67	0.66	0.62	0.72	0.75

load and less by the flow-related stratification. Although stratification is necessary and important to maintain hypoxia, the interannual changes in hypoxia are more influenced by the amounts of nutrient load than by the variations of strength of stratification. Freshwater discharge also affects the residence time of nutrients in the estuary, and subsequently on anoxia; however, this was not analyzed in this work.

**Correlation of Anoxia with Temperature** The Bay's anoxia occurs in the summer. Higher temperature in the summer reduces oxygen solubility, promotes oxidation of organics, and enforces the bottom to surface density stratification because the bottom water has lower temperatures than the surface waters in the summer. Strong stratification can prevent heat exchange between the deep water and the warmer upper water and the air, leading to a negative correlation of stratification strength and DWtmp ( $r=-0.15$  to  $-0.29$ , table omitted for brevity). The stratification strength has a positive correlation with summer anoxic volume ( $r=0.23$ , Table 2). This could cause the apparent negative correlation between anoxic volume and DWtmp ( $r=-0.15$ ), although it is impracticable to draw definitive conclusions based on the weak correlation. Meanwhile, the summer average air temperature (Atmp) in individual years reflects the temperature in the entire Bay system and, thus, has a positive correlation with summer anoxic volume (though weak,  $r=0.33$ ). Since the interannual changes in summer air temperature are small, a strong correlation between air temperature and anoxic volume is difficult to discern from the observed data.

**Correlation Between Anoxia and Wind Speeds** Table 3 lists correlation coefficients between summer anoxic volume and counts of summer hourly winds for speeds greater than or equal to 2, 3, 4, 5, 6, 7, and 8 m/s as symbols Cwd2 through Cwd8 and average wind speed (Wspd). The Cwd2 metric is the count of hourly winds that had speed  $\geq 2$  m/s, as is the case for Cwd3 through Cwd8.

The Cwd4 has the highest correlation with anoxic volume, suggesting that the winds of speeds greater than or equal to 4 m/s influence anoxic volume more than the winds of lower speeds. Wind speed of 2 m/s has little effect on destratification and hypoxia (Chen and Sanford 2009). The main contribution to Cwd2's negative correlation with anoxia ( $r=-0.28$ ) could be from the winds of higher speeds. High-speed wind events, such as  $>7$  m/s, are infrequent, and the timing of their occurrence and DO sampling in the Chesapeake is unlikely. Thus, the wind counts involving only high speeds, e.g., Cwd7, have weaker correlations with summer anoxia than do the wind counts also including some lower speeds, e.g., Cwd4, although it is expected that stronger winds actually have a stronger influence on anoxia than weaker winds do.

DO at the anoxic zone can increase, i.e., anoxia is reduced, promptly in strong wind events (Wang et al. 2013a). However, only a medium strength of correlation is obtained between the observed summer anoxic volume (AV\_obs) and the average wind speed (Wspd) ( $r=-0.37$ , Table 2) or the count of hourly winds of speed  $>4$  m/s (Cwd4,  $r=-0.42$ ). One of the reasons is that the six to eight sampling events in the summer in different years may meet with different wind strengths, and therefore, the wind effect on anoxia reduction is poorly represented in the samples.

**Correlation of Anoxic Volume with Wind Directions** Table 4 is the correlation of summer anoxic volume and counts of hourly winds for eight compass directions based on 1991–2005 data for speeds equal to or greater than 5 or 2 m/s, as indicated by the number 5 or 2 following the symbols of wind direction.

**Results from Linear Regression** In the presented regressions (Tables 5 and 6), the nutrient load index (SNP) is always a regressor, either as a single regressor or with one or more additional regressor(s). The residuals of the annual anoxic volume predictions ( $\text{Residual} = \text{AV}_{\text{predict}} - \text{AV}_{\text{obs}}$ ) are accessed. For either the summer or July anoxic volume

**Table 4** Correlation of summer anoxic volume with counts of wind events of speed  $\geq 5$  and 2 m/s, respectively, for eight compass directions in summer (1991–2005) ( $n=15$ )

	cN5	cNE5	cE5	cSE5	cS5	cSW5	cW5	cNW5
AV_obs	-0.38	-0.52	-0.52	0.24	0.24	0.05	-0.03	0.41
Wspd	0.88	0.87	0.31	-0.28	-0.34	-0.12	-0.14	0.11
	cN2	cNE2	cE2	cSE2	cS2	cSW2	cW2	cNW2
AV_obs	-0.11	-0.53	-0.30	0.24	0.19	-0.29	-0.03	0.37
Wspd	0.56	0.58	0.06	-0.27	-0.27	0.24	-0.16	-0.12



**Table 5** Statistics of the results from regressions that predict summer and July anoxic volume by nutrient load and other influencing constituents using 1991–2005 data ( $n=15$ )

	Regressors	SNP			Second regressor			$R^2$	Adj $R^2$	Std_rsd of AV
		Coeff	SE	$p$ value	Coeff	SE	$p$ value			
Regression for summer anoxic volume predict	SNP	0.298	0.0412	<0.0001	N/A	N/A	N/A	0.802	0.787	0.308
	Multivariate*	0.319	0.0539	<0.0001	See Table 6			0.840	0.776	0.276
	SNP+Wspd4	0.293	0.0430	<0.0001	−0.257	0.4236	0.555	0.808	0.776	0.303
	SNP+Wspd5	0.293	0.0422	<0.0001	−0.304	0.3698	0.427	0.812	0.781	0.299
	SNP+Cwd4	0.290	0.0465	<0.0001	−0.0013	0.0029	0.673	0.805	0.772	0.305
	SNP+SS	0.303	0.0447	<0.0001	−11.56	31.023	0.716	0.804	0.771	0.306
	SNP+Atmp	0.320	0.0472	<0.0001	−0.111	0.1173	0.364	0.816	0.785	0.297
	SNP+DWtmp	0.305	0.0435	<0.0001	0.060	0.0929	0.528	0.809	0.777	0.302
Regression for July anoxic volume predict	SNP	0.440	0.1086	0.0014	N/A	N/A	N/A	0.558	0.533	0.812
	Multivariate*	0.487	0.1317	0.0041	See Table 6			0.591	0.427	0.781
	SNP+Wspd4	0.443	0.1161	0.0024	0.151	1.1364	0.890	0.559	0.485	0.811
	SNP+Wspd5	0.440	0.1141	0.0023	0.0044	1.3852	0.998	0.558	0.484	0.812
	SNP+Cwd4	0.402	0.1140	0.0042	−0.005	0.0073	0.314	0.595	0.528	0.777
	SNP+SS	0.448	0.1136	0.0019	−21.41	46.870	0.655	0.566	0.493	0.805
	SNP+Atmp	0.479	0.1207	0.0019	−0.181	0.2275	0.441	0.580	0.510	0.791
	SNP+DWtmp	0.438	0.1169	0.0028	−0.011	0.1805	0.954	0.558	0.484	0.812

Residual = predicted − observed. The sum and mean of residuals are nearly zero in all regressions. The ranges of the residuals are about −0.65 to 0.35 in summer AV prediction, and about −1 to 1.7 in July AV prediction.

Regressors for the multivariate regression, SNP + Wspd5 + SS + Atmp, see Table 6

SE standard error in coefficient estimate, Std\_rsd standard deviation of residuals in anoxic volume (AV) prediction, N/A not applicable

predictions, adding an additional regressor besides SNP slightly increases  $R^2$  and reduces the standard deviation of Residual (Std\_rsd). The multivariate regression has the lowest Std\_rsd and highest  $R^2$ . Consistent with Murphy et al. (2011),

**Table 6** Supplemental statistics for the multivariate regression in Table 5

		Regressors				Observed AV
		SNP	Wspd5	SS	Atmp	
Regression for summer anoxic volume predict (Intercept = 6.920)	Coeff	0.319	−0.533	5.031	−0.167	N/A
	SE of coeff	0.0539	0.5024	39.814	0.1285	N/A
	$p$ value of coeff	<0.0001	0.314	0.902	0.222	N/A
	Min input value	2.87	5.60	0.0096	22.3	0.35
	Max input value	9.11	6.42	0.0183	25.3	2.71
	Mean input value	5.17	6.08	0.0137	23.6	1.26
	Std of input values	2.07	0.24	0.0030	0.83	0.64
Regression for July anoxic volume predict (Intercept=8.838)	Coeff	0.487	−0.505	−25.23	−0.197	N/A
	SE of coeff	0.1317	1.5813	52.427	0.2536	N/A
	$p$ value of coeff	0.004	0.756	0.641	0.456	N/A
	Min input value	2.87	5.68	0.0078	23.3	1.10
	Max input value	9.11	6.23	0.0262	26.8	4.91
	Mean input value	5.17	5.98	0.0156	25.2	2.53
	Std of input values	2.07	0.17	0.0050	1.10	1.22

N/A not applicable, SS stratification strength, Atmp average air temperature, Wspd wind speed

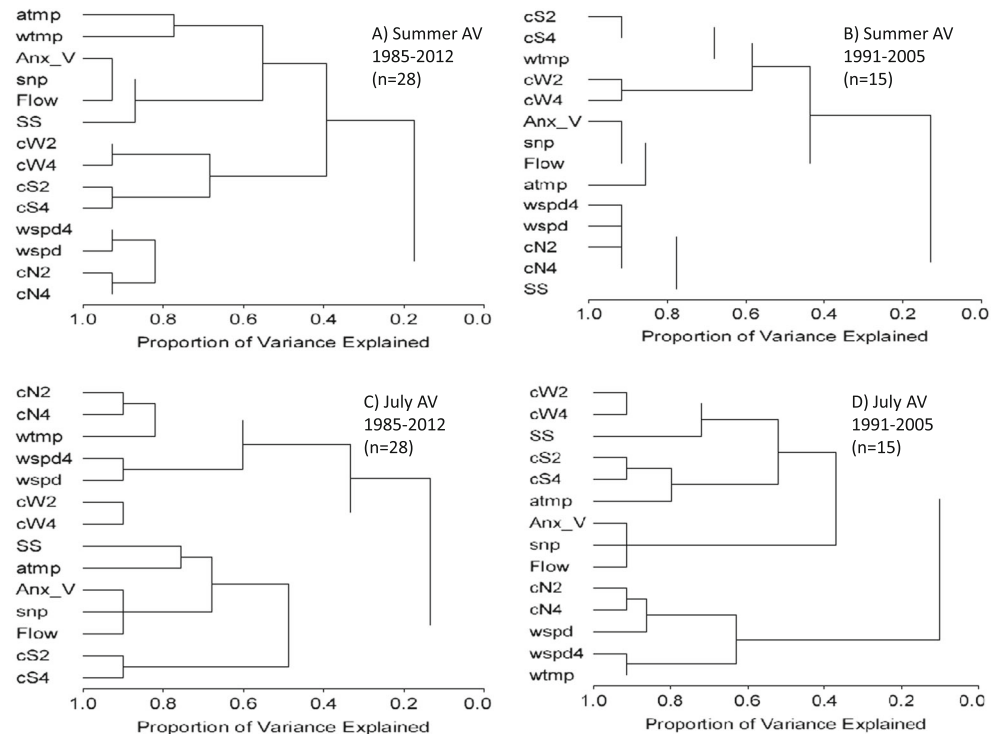
the results suggest that factors other than nutrients could influence summer or July hypoxia. The  $p$  values are low for SNP but higher in other variables. The low  $p$  value and positive slopes of SNP suggest nutrient load to be the most important predictor of anoxia. Strong winds, e.g., Wspd5, reduce anoxia (with slope =  $-0.3$  to  $-0.5$ ). The prediction on summer anoxia is better than the prediction on July anoxia. The Std\_rsd is higher, and  $R^2$  is lower in the July AV predictions than in the summer AV predictions. This may be due to high variability of some anoxia-influencing factors at the monthly scale, such as wind. This suggests that summer average anoxia metric may be better than July anoxia to represent the interannual variation of anoxia.

#### Statistics on Data in 1985–2012 Period and Comparison to the Results from the 1991–2005 Data

The correlations presented in Tables 2, 3 and 4 are based on the 1991–2005 data. Similar correlation coefficients were computed for all years of data from 1986 to 2012 in a stepwise manner adding 1 year at a time. The correlation coefficients including more years of data vary less than 0.1 from the values in Table 2. If 1985 is included, the correlations among them reduce about 0.1–0.2. The lower correlation upon adding 1985 data is coincident to the observation by Conley et al. (2009) who found 1986 to be a significant break point in the Chesapeake hypoxia trend. Nevertheless, the correlations based on the 1991–2005 data generally hold to the recent years as well as earlier periods, as can be further verified by cluster analysis.

Figure 6a, b shows tree diagrams from a cluster analysis on summer anoxic volume and its influencing variables using 1985–2012 ( $n=28$ ) and 1991–2005 ( $n=15$ ) observed data. The tree diagram on the 1985–2012 data (Fig. 6a) is consistent with the correlation analysis. The anoxic volume (Anx\_V), Susquehanna nutrient load (SNP), and flow are grouped with only a short distance among them, with “proportion of variance explained” = 0.95, indicating a high positive correlation among them. The SS and the Atmp also have a positive but weaker impact on anoxia, i.e., having a larger distance from Anx\_V, and with a lower proportion of variance explained, 0.85 and 0.55, respectively. The group of the bottom four variables (Wspd, Wspd4, cN2, and cN4) is highly tied, since many higher speed wind events in 1997–2012 were northerly, and cN2 or cN4 have high positive correlations with Wspd (Table 4). This group is separated from the Anx\_V group, having proportion of variance explained  $<0.2$ , and they have an adverse effect on, i.e., reduce, anoxia. Many southerly winds have lower speeds and have a negative correlation with Wspd (Table 4) and thus have a weak positive correlation with anoxic volume. Accordingly, the tree diagram shows a weak tie of anoxic volume with the counts of southerly wind (cS2 and cS4), having a proportion of variance explained of 0.4. In general, the tree diagram is consistent to the correlation analysis. The tree diagram based on the 1991–2005 data (Fig. 6b) also displays similar affinities among the variables, though with minor deviations such as the adverse impact on anoxia by stratification strength. Figure 6c, d shows tree diagrams for July anoxic volume and its influencing variables using the 1985–2012 and 1991–2005 data. The affinities among the

**Fig. 6** Tree diagrams for anoxic volume and its influencing variables. **a** For summer anoxic volume, using 1985–2012 data ( $n=28$ ), **b** for summer anoxic volume, using 1991–2005 data ( $n=15$ ), **c** for July anoxic volume, using 1985–2012 data ( $n=28$ ), and **d** for July anoxic volume, using 1991–2005 data ( $n=15$ )



variables are consistent to the correlation analysis relating July anoxic volume found in Table 2.

### Comparing Three Levels of Hypoxia

The correlations of watershed load and flow with observed hypoxic volume of DO <1 or <2 mg/l are about 0.1 to 0.2 lower than their correlations with anoxic volume of DO ≤0.2 mg/l. This could be due to the interaction of the depths of the various pools of hypoxic and anoxic water and the ability of wind to influence these different pools. The average depths of the upper boundaries of hypoxia/anoxic waters for DO <2, <1, and ≤0.2 mg/l at station CB4.1C in summertime of 1985–2012 were 12.2, 13.1, and 19.7 m, respectively. The average depth of the maximum stratification at CB4.1C was 11.4 m. The episodic winds' effects vary in sampling cruises. The destratification by direct wind mixing propagates from the surface to deeper depths (Chen and Sanford 2009) and usually has a greater influence on water at shallower depths. Anoxic water is generally at deeper depths than the hypoxic waters and is less vulnerable to mixing associated with moderate wind stress. This may be a reason for the greater correlation of watershed load with anoxic volume than with hypoxic volumes of higher DO threshold.

## Discussion

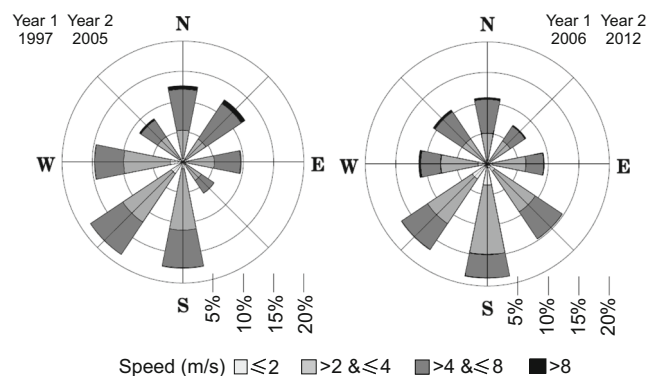
### Wind Speed Versus Direction in Influencing Hypoxia

The time-series plots of anoxic/hypoxic volume of 1996–1998 (Fig. 4) and the wind-rose plots (Fig. 5) suggest that wind speed is more important than wind direction in affecting anoxia/hypoxia. This conclusion is consistent with the correlation analysis: The summer anoxic volume has a moderate negative correlation with average wind speed (Wspd,  $r = -0.37$ ) and counts of stronger winds (e.g., Cwd4,  $r = -0.42$ ) (Tables 2 and 3). In the analyses, the anoxic or hypoxic volume and the wind in the same season were used, because the anoxia/hypoxia influenced by episodic winds can return to the approximate original volume in a few days after the wind weakens. If May and June's winds were used with July's wind to relate to July's hypoxia, different conclusions could be drawn, as in Scully (2010a).

The above conclusion is not simply based on correlation analysis, since the  $p$  and  $r$  values only tell the significance of the correlation for the variables in the specifically selected sample which may fail to indicate whether they have a cause and effect relationship (Browner and Newman 1987; Taylor 1990). Table 4 shows that for winds of speeds >5 m/s, the north and northeast winds had moderately high negative correlations with anoxic volume ( $r = -0.38 \sim -0.52$ ), while south

winds had a positive correlation with anoxic volume ( $r = 0.24$ ). But in this case, it would be incorrect to infer that a cause and effect relationship between wind direction and anoxic volume exists, i.e., the north winds reduce anoxia while the south winds increase anoxia. In fact, in many cases of episodic wind events, a south wind reduced more hypoxia than a north wind under the same wind speed (Chen and Sanford 2009; Scully 2010a; Li and Li 2011). The aforementioned case of apparent correlation of wind direction and anoxia is basically a reflection of the correlation between wind speed and anoxia. Figure 7a, b shows wind roses from hourly winds in summers of 1997–2005 and 2006–2012. The stronger winds (speed >8 and >4 m/s) were more northerly, while weaker winds were predominantly southerly. In fact, this pattern generally held in each individual year of the 1997–2012 period (figures omitted for brevity). Thus, there is a high positive correlation ( $r = 0.88$ ) between average wind speed (Wspd) and the frequency of the north wind (cN5) but a negative correlation ( $r = -0.34$ ) between Wspd and the frequency of the south wind (cS5) (Table 4). Because wind speed has a negative correlation with summer anoxia ( $r = -0.37$ , Table 2) and wind speed has a negative correlation with the southerly wind frequency (cS5), therefore, a weak positive correlation between the southerly wind frequency (cS5) and the anoxic volume is seen.

Similarly, the  $r$  values from a correlation analysis may not reflect a cause and effect relation if a key component in the data set is mixed from different periods that were in different environments or assessed by different methodologies. For example, the methods assessing July anoxic/hypoxic volumes were different before and after 1985 (Hagy et al. 2004), and an ecosystem shift in 1986 has been postulated (Conley et al. 2009): Two separate significant relationships between hypoxia and nitrate loading were detected for 1950–1979 and 1980–2001 (Kemp et al. 2005). A regression using the combined pre- and post-1985 July hypoxia yielded high correlation of hypoxia with westerly winds ( $r = 0.71$ ,  $p < 0.05$ ) but moderate correlation with nutrient load ( $r = 0.44$ ) or flow ( $r = 0.24$ ) (Scully 2010b), which is inconsistent with the results of this



**Fig. 7** Wind roses for all hourly winds in summer of 1997–2005 and 2006–2012

article. This could possibly be due to the different time periods averaged for wind and hypoxia. The rationale for inclusion of the preceding month's winds of May and June in a correlation analysis of July hypoxia in Scully (2010b) is unclear because of the relatively rapid influence that wind has on hypoxia.

The finding that wind speed is more important than direction in modulating anoxia is also supported by the cluster analysis (Fig. 6), as well as model simulations illustrated in Fig. 8. The anoxic volume in the mainstem Bay was about  $9.6 \text{ km}^3$  in the simulated no-wind condition (Fig. 8). The simulated anoxia reduction by a south wind of 5 m/s is less than that of a west wind at 6 m/s. A 20 % increase in wind speed yields a greater anoxia reduction than switching wind direction from westerly to southerly without a change of wind speed. The above relationship is approximately held by wind speeds ranging from 4 to 10 m/s that last for a few hours. The differences in anoxia reduction among wind directions become larger with wind of longer duration (or greater speeds) such as winds lasting 2 days. Compared to nutrient loads or wind speed, wind direction has relatively less influence on the interannual variation of anoxia. The most important condition required for wind destratification is for the speed of the wind to be sufficient.

#### Nutrient Load Versus Wind in Regulating Interannual Hypoxia and Implications for Management

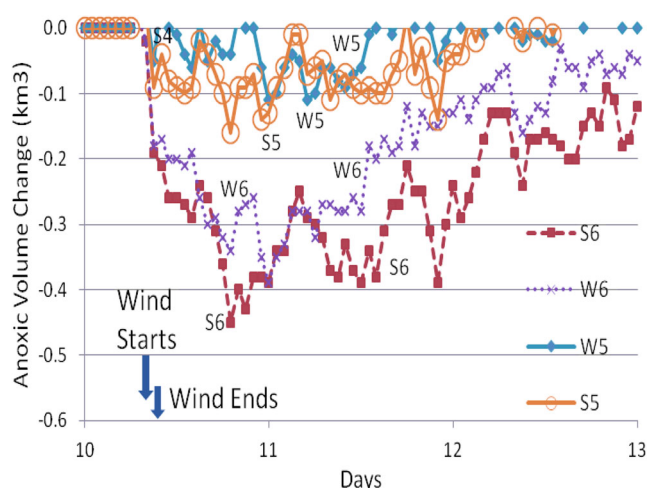
Nutrient loads have two main contributions to anoxia. First, the organic nutrient load is a direct oxygen demand, and secondly, the inorganic nutrient loads support primary production and consequently increase oxygen demand from decaying algae in deep waters and bottom sediment. The timing of the input and generation of the two sources of oxygen demand may vary interannually. Relationships

between chlorophyll-*a* concentrations and nutrient loads were found to be diverse in different time scales or localities (Jordan et al. 1991). Although algal blooms contribute to hypoxia, Wang et al. (2006) obtained a weak correlation ( $r < 0.3$ ) between the selected metrics of anoxia and chlorophyll-*a*.

Model experiments showed that under consistently applied initial conditions, the anoxia reduction is greater by a 20 % increase in simulated wind speed than by a 20 % decrease in simulated winter-spring nutrient load. This suggests that wind can play an important role in modulating anoxia in individual wind events, especially with winds of high speed. However, the interannual variation of summer anoxia approximately follows the interannual variation of the winter-spring nutrient loads and flow (Fig. 2). The low anoxia years are not observed to have extremely high winds nor do the high anoxia years have extremely low winds. This indicates that the interannual anoxia responds mainly to nutrient loads, consistent with the strong correlation of nutrient loads and anoxia (Table 2). The weaker correlation of wind with anoxia (Table 2) in the interannual scale is due to four reasons: (1) nutrient load is a primary, intrinsic factor leading to anoxia; (2) the relative magnitude of wind variation in different summers is not as big as the interannual variation of nutrient load (Fig. 2); (3) a high nutrient load year usually is accompanied by high flows which could result in greater stratification and better preserve anoxia; and (4) wind conditions vary relative to different monitoring events in a year. Thus, the wind conditions affecting the monitored DO could be poorly represented by the averaged wind data used in the correlation analysis. Nevertheless, wind strongly influences hypoxia in some cases, especially for the hypoxia of higher DO thresholds (e.g.,  $\text{DO} < 1$  or  $< 2 \text{ mg/l}$ ), as presented in Figs. 4 and 5 on comparing hypoxia and winds in individual months in 1996, 1995, and 1997.

Scully (2010b) observed a reduction of frequency of southerly winds in the past two to three decades, implying that the Bay would be subjected to a more severe hypoxic problem in later years if nutrient load remains unchanged in those years. However, such a trend in wind fields may only alter the hypoxia in the Chesapeake Bay to a certain degree. The interannual variation of nonpoint load is highly modulated by hydrology, which varies among the years and influences the interannual variation of hypoxia significantly. Thus, we still observed lower anoxic volumes in some recent years such as 2010 and 2012 (Fig. 2) which had low nutrient loads.

In the studied summer periods, extreme wind events, such as hurricanes, rarely occurred, and when they did, they were mainly in late September or later and not in the selected DO monitoring period of June to mid-September. In the peak summer period, when respiration and oxygen demands were strong, anoxia/hypoxia could gradually recover to the pre-wind condition because of re-stratification. Therefore, nutrient loads (plus stratification by freshwater input) were the primary



**Fig. 8** Model simulated changes of anoxic volume versus the no-wind condition by southerly and westerly winds. The wind started at hour 4 on day 10 in August 1996 and lasted for 1 h. The numbers 6 and 5 following the direction symbols indicate the speed (at m/s)



factors causing summer hypoxia, while wind was a secondary force that episodically modulated the nutrient-induced hypoxia. Thus, we see a higher correlation of anoxic volume with watershed load (SNP,  $r=0.89$ ), and a moderately low negative correlation of anoxic volume with average wind speed (Wspd,  $r=-0.37$ ) or frequency of stronger winds (Cwd4,  $r=-0.42$ ) (Table 2). The high affinity of SNP with Anx\_V in the cluster analysis and the good explanation of SNP on anoxic volume in the regression experiments support the finding that nutrient loads are the key factor controlling interannual anoxia. Thus, summer average hypoxia represents the integrated and lasting effect of loads and helps avoid the noise of brief wind events.

Wind varies among the summer months and is important in modulating anoxia/hypoxia variations during these months. The different wind's effects among summer months caused the correlation of the nutrient load with individual months' (such as July) anoxic volume to be weaker ( $r=0.75$ , Table 2) than the correlation of the nutrient load with the summer average anoxic volume ( $r=0.89$ ). Nevertheless, it is necessary and useful to assess anoxia/hypoxia in individual months especially July and August to capture the worst DO conditions. Since wind can significantly modify the hypoxic condition and wind varies frequently, more frequent observations on DO could provide more representative conditions. On the other hand, caution is needed when using the DO observed during or soon after a high wind event.

## Conclusions

Both watershed loads and wind have significant impacts on Chesapeake summer hypoxia (including anoxia). The increase of oxygen demand and respiration in the water column and sediment due to watershed nutrient loads are the key factors influencing summer hypoxia in the Chesapeake Bay. On the other hand, wind can erode stratification and reduce hypoxia by exchanging the oxygen-rich surface water with the deep hypoxic water. Based on the past three decades of observation, the interannual changes of anoxia and hypoxia in the Chesapeake Bay are mainly controlled by the nutrient loads from the watershed, while the influence of interannual changes in wind conditions plays a secondary role.

Wind conditions, e.g., speed and direction, vary at short time scales, causing within-month variation in hypoxia. However, the relative magnitude of the variation of its summer average in different years is not as large as the interannual variation of nutrient loads. Although brief wind events cause destratification and reductions in anoxia and hypoxia, conditions may return to pre-wind levels after the wind weakens. These factors cause the observed summer anoxia and hypoxia to have a weaker correlation with the wind rather than with the nutrient load.

Although the degree of anoxic or hypoxic volume reductions are different with different wind directions at a specific wind speed, the magnitude of wind speed (once above a certain threshold) is generally more important than wind direction in episodically decreasing stratification and reducing hypoxia.

Considering the scattered monitoring events and episodic winds typical of the Chesapeake and other coastal waters, the application of a summer-averaged anoxic/hypoxic metric could better reflect the Chesapeake management assessment of anoxia/hypoxia and its response to nutrient loads rather than an individual month's value. In addition to a summer-averaged metric, July and August anoxia or hypoxia should also be analyzed because they represent the worst DO months in a year.

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