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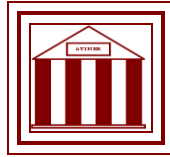
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Assessment of Hypoxia and its Relationship with Nutrient Loads and Wind, and Implication to the Chesapeake Bay Management

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Abstract

Statistics are applied to analyze the correlations of summer hypoxia in the Chesapeake Bay with watershed input and wind conditions based on nearly three decades of monitoring data. The Pearson correlation coefficients indicate that the averaged summer hypoxia has strong positive correlation with watershed nutrient load and discharge, and moderate negative correlation with summer average wind speed. Nutrient inputs and the subsequent decay of organic matter are the primary factor that controls the oxygen demand causing summer hypoxia, while episodic wind can partly erode stratification and hypoxia. The interannual variation of hypoxia is mainly controlled by watershed input, but wind plays an important role in modulating hypoxia, such as variations of hypoxic volumes in individual summer months. Although the extent of hypoxia reduction is different with different wind directions, a faster wind speed (above certain strength) causes stronger destratification and hypoxia reduction than weaker speeds, which is generally more important than the effect due to wind directions. Computer modelling is used to obtain dissolved oxygen conditions in finer temporal and spatial scales to supplement the discrete observations in scattered monitoring stations to better understand hypoxia development under episodic wind events, which enhances the understanding on physical relationships among the concerned constituents beyond the statistical analysis.

Keywords: Hypoxia, nutrient load, freshwater discharge, wind speed and directions, destratification, correlation analysis

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Introduction

Summer hypoxia in the Chesapeake Bay threatens living resources of the Bay. Excessive nutrient loads and the subsequent bloom of algae that elevate bottom oxygen demands are the main cause of the hypoxia (Officer et al. 1984; Harding et al, 1992). Controlling nutrient load has been focused on the Chesapeake management to reduce hypoxia (USEPA 2010). On the other hand, destratification by wind energy (Kato and Phillips, 1969) can increase dissolved oxygen (DO) in deep water and reduce hypoxia, as has been observed in the Chesapeake estuary and other estuarine and coastal regions (Melone et al. 1986; O'Donnell et al. 2008; Wilson et al. 2008). The relative importance of nutrient loads and wind affecting Chesapeake hypoxia has been given attention recently since it has significant implication to management (Scully 2010a; Murphy et al. 2011).

The Chesapeake Bay is oriented approximately north to south from the upper Bay to the mouth connecting to the Atlantic Ocean. It is a partially mixed estuary (Prichard, 1967), with net seaward (to the south) flow in surface waters and landward (to the north) flow of bottom saltier water. The extent of destratification by winds from different directions is different (Chen and Sanford 2009; Scully 2010a; Li & Li 2011) because of the two-layer circulation, channel bathymetry, Earth's rotation, etc. The relative importance on modulating hypoxia by wind direction and wind speed has also implications to management. Scully (2010b) attributes the sustained high hypoxia in the Chesapeake Bay in the past decades despite of nutrient reductions in past to a shift of wind that has been more westerly or southwesterly. However, Wang et al. (2013) found that wind speed over certain thresholds to be a more important factor than wind direction in many occasions. This paper focuses on the relative effects on hypoxia reduction by nutrient loads, wind speed, and wind direction.

Data and Method

We use observed data supplemented with model simulated values to analyze the response of hypoxia to nutrient load and wind, and applied statistics on historical data.

Hypoxia Estimated based on field observations: There are about 80 long-term monitoring stations of DO in the Chesapeake Bay, sampled once or twice each month since 1985. About 45 stations in the mainstem Bay and its lower tidal tributaries were selected to interpolate DO concentration spatially over the entire mainstem using the modified method of Wang et al. (2006) from the Chesapeake Bay 3-D interpolator (Bahner, 2001). Three levels hypoxia, i.e., DO<=0.2, <1, and <2 mg/l in the summer months (June to September) were assessed. The condition of low level of hypoxia (DO<=0.2 mg/l), also called anoxia (Jasinski, 2003; Wang et al. 2006) is the focus in this paper.

Hypoxia Estimated by Computer Models: The Chesapeake Bay Water Quality and Sediment Transport Model (WQSTM) (Cerco et al., 2010) is used to estimate hypoxia. The WQSTM is a peer reviewed and approved regulatory

model for the Chesapeake Bay water quality management. 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, 3 groups of phytoplankton, and related biochemical processes. The errors of estimated DO in the mainstem are 0.3 mg/l and -0.45 mg/l at depths of 6.7-12.8 m and greater than 12.8 m, respectively. Hourly hypoxic volume of the Bay is calculated by adding volumes of the model cells that have hourly average DO under the hypoxia thresholds.

The hypoxic volumes of the three levels DO thresholds, i.e., <=0.2 mg/l, <1 mg/l and <2 mg/l, are labelled as AV (i.e., anoxic volume), HV1 and HV2. We use the suffix '_mod' if the values are from modelled results, otherwise the values are from observation (though the suffix '_obs' is also used for observed).

Wind fields: Hourly wind observations at the Patuxent Naval Air Station, MD, USA, were collected. The counts, i.e., frequency, of hourly wind events for speed greater than certain thresholds and/or in certain directions were obtained. The events of 8 directions, i.e., N, NE, E, SE, S, SW, W, and NW, were filtered.

W*atershed discharge and nutrient loads:* Daily mean flows at the Conowingo USGS station of the Susquehanna River were collected. Daily TN and TP loads from the Susquehanna River were estimated based on bi-weekly total nitrogen (TN) and total phosphorus (TP) measurements and daily mean flows (Wang and Liker, 2008). In the correlation analysis, TN and TP load are composed into a single variable, 'nutrient index' (Wang et al. 2006), which equals (TN+10TP)/2 of the N and P weights, labelled SNP. Annual 'winter-spring' flow or load is calculated using their averaged values in January through May. The effective 'nutrient index' to the Bay's anoxic center was also estimated according to Wang et al. (2006) and is labelled ENP.

Statistical analysis on factors impacting hypoxia: Statistics are conducted to analyze correlations among summer hypoxia, winter-spring watershed input, winds, and other influencing variables. The statistics are on 15 years of data in 1991-2005, and in extended periods covering 1985-2012.

Results and Discussion

Inter-annual variation of hypoxia and related variables

Figure 1 shows inter-annual variation of summer anoxic volume (DO<=0.2 mg/l) and hypoxic volume (DO<1 mg/l), winter-spring watershed discharge and nutrient load index, and summer average wind speed in year 1985 to 2012. Table 1 lists correlation of anoxic volume with watershed inputs, winds, stratification strength (SS), deep water temperature (Wtmp) and air temperature (Atmp).

The summer hypoxia is highly correlated (r> 0.88) with winter-spring nutrient load as observed by other researchers (Hagy et al. 2004; Kemp et al.,

2005). The variations of interannual summer hypoxia well follow the variation of winter-spring nutrient load or flow (Fig. 1). The high winter-spring flow-load years 1993, 1996, 1998, and 2011 have high anoxic volumes, and the low flow-load years 2002 and 2012 have low anoxic volumes. The excessive nutrient load in early months of the year causes algal blooms in the spring and summer (Harding et al., 1992). The dead algae increase oxygen demand of bottom water in the summer. The several months-long residence time enables nutrient to have sustained influence on hypoxia through the summer (Murphy et al., 2011). The correlation between summer anoxic volume and average wind speed (Wspd) is -0.37. The years of high or low hypoxia do not always have low or high wind in the summer.

The nutrient load and flow are highly correlated (r=0.98), and they have similar strengths of correlation with summer hypoxia. In physical processes, besides carrying nutrient load, freshwater discharge affects stratification that also influences hypoxia, independent of nutrient's effect.

Correlation between summer anoxia and wind speeds

Table 2 is correlation coefficients between summer anoxic volume and counts of summer wind events for speeds greater than 2, 3, 4, 5, 6, 7 and 8 m/s as symbols Cwd2 through Cwd8, and average wind speed (Wspd).

Cwd4 has the highest correlation with anoxic volume than other wind counts, indicating the winds with speeds greater or equal to 4 m/s influence anoxic volume more than the winds with lower speeds. The study from Chen and Sanford (2009) suggested wind speed at 2 m/s has little effect on destratification and hypoxia. Note that, Cwd2 includes all events of speed>2 m/s. The main contributions to Cwd2 to have negative correlation with hypoxia (r=-0.28) may be from the events of higher speeds. However, winds of very high speed may not occur at comparable frequency around the DO observation dates among the years. The wind count only include high speeds, such as Cwd7, has weaker correlation with summer anoxia than Cwd4 does, although stronger winds influence more. The 6-8 sampling events in the summer in different years may meet with different strengths of wind, therefore, the wind effect on hypoxia reduction cannot be well represented, therefore, only medium strength (r= -0.37) of correlation is obtained between the observed summer anoxia and average wind speed. Greater negative correlation (r= -0.53) is obtained using modeled data, in which both summer anoxia and average wind speed were averaged from hourly values.

Correlation of summer anoxia with wind directions

Table 3 is the correlation of anoxic volume and counts of wind events in 8 wind directions based on 1991-2005 data for speeds equal and greater than 5 m/s as the post-fix 5 after direction symbols. The N, NE, and E winds have moderate high negative correlation with anoxic volume (r= -0.38 ~ -0.52), while the south wind has a positive correlation with hypoxia (r=0.24). This does not mean that north wind reduces anoxic volume while south wind increases anoxia, because extensive research revealed that in the Chesapeake

Bay, under moderate high speed, either southerly or northerly wind can reduce stratification and hypoxia, and in many cases south wind reduces more hypoxia than north wind under a specified speed (Chen and Sanford 2009; Li and Li 2011). The aforementioned correlations between the frequency of wind direction and hypoxia are basically a reflection of the correlation between wind speed and hypoxia. The following is our reasoning. In the summer of 1997 to 2005, the stronger wind events (speed >=5 m/s) are more northerly than southerly, as we see high positive correlation between average wind speed (Wspd) and frequency of north wind (N5, r=0.88), but negative correlation between Wspd and frequency of south wind (S5, r=-0.34). Because wind speed has negative correlation with summer anoxia (r= -0.37), thus we see the apparent negative correlation of northerly wind frequency (N5) and anoxic volume and weak positive between southerly wind frequency (S5) and anoxic volume. The occurrences of southerly winds in different summers are not consistent with the variations of anoxic volume may be the other reason causing the said weak correlation with anoxia. Comparing to the effect on hypoxia by nutrient load or wind speed, the differences in the impact on hypoxia by wind directions are less important to the interannual variation of hypoxia, though some directions such as south winds can have greater impact than other directions.

Model simulated hypoxia under different wind directions and durations

Fig. 2 is model simulated anoxic volume changes relative to no wind for 4 wind directions. The wind started at 8 o'clock on August 10, 1996, and the anoxic volume was about 12.4 km^3 at no wind. Fig. 2a is for wind speed at 8 m/s that lasts about 2 days. Anoxic volume reduces rapidly at the beginning of wind, and reaches the lowest at third day, i.e., 0.5 day after wind stops. The difference in anoxic volume reductions between south and north winds is about 1.2 km^3 and between south and west winds is 2.2 km^3. They are 12% and 22%, respectively, of the averaged reduction by winds from the no-wind. Due to sediment oxygen demands and re-stratification, the anoxic volume gradually recovers to the pre-wind condition after wind calms. It recovered 75% after one week.

Fig. 2b is for wind speed at 6 or 5 m/s that last for 1 hour. At wind speed=6 m/s, the anoxic volume reduces 0.2 to 0.4 km^3 at the first 1 hour, which are 1.6% to 3.2% from the no-wind condition. The anoxic volume recovers about 80% in 2 days – much faster than that by the 8 m/s wind of 2-day duration. The difference in anoxic volume reductions is mostly less than 0.1 km^3 between south wind and north or west wind. The anoxia reduction by south wind of 5 m/s is much less than that by west wind of 6 m/s. The anoxic volume gets a greater change by changing 1 m/s speed than by changing wind direction without speed change. Fig. 2 also indicates that wind of longer duration has a greater impact on hypoxia.

DO, hypoxic volume and their associated wind in finer time steps

The circles in Figure 3 are observed anoxic volume (DO<=0.2 mg/l) in

summer (June through September) sampling cruises in years 1991-2005. The pluses are 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 in scattered monitoring stations in 4-7 days of a sampling cruise. Therefore, the anoxic volumes from the two methods are not directly comparable. Figure 3 also plots observed hypoxic volumes (DO<1 mg/l, in bigger dark dots). The small dots are wind speed>=6 m/s. The modelled anoxic\hypoxic volume responses to wind: usually depresses after high wind events.

Figure 4 is wind speed and directions for wind>=6 m/s. The high wind events in the summer of 1997-2005 are more prevailed from north, northeast and northwest, though there are many events blowing from south (and southwest or southeast) at speeds < 4 m/s which are excluded from the figure.

The 1996 and 1998 years have similar nutrient loads, but July hypoxia in 1998 is about twice that of 1996. This is because in the month of July, 1996 has more frequent wind events of >=4 m/s than 1998 (242 vs. 181, accounting for all directions, Table 4). The higher speed events (i.e., >= 4 m/s) in 1996 are mainly in W, NW, and SW directions, as well as N and E, while, the frequency of southerly wind in July is more in 1998 than that in 1996, e.g., 118 vs. 100 (for all speeds). The more reduction of July hypoxia in 1996 cannot be explained simply by wind direction, since under the same speed, westerly wind usually has weaker destratification than southerly wind. In this case, wind speed is more important than wind directions in hypoxia reduction.

The wind's effect on June hypoxia is almost in the opposite pattern (Fig. 3). 1996 has greater June anoxia and hypoxia than 1998, which is concordant to the fewer frequent June winds of speed > 4 (or 6) m/s in 1996 than in 1998.

Correlation analysis using a longer period of data

The analysis on 15 years of data provides useful information on the inter-annual change of hypoxia. Analyses using data in expanded periods were also performed. They are based on the 1991-2005 data and added additional data year-by-year forward to 2012 and backward to 1986. The correlations among the variables in Table 1 vary less than 0.1. If 1985 is included, the r's reduce about 0.1-0.2. These are due to the additional years having different patterns or trends from the base years for the variables.

The lower correlation when adding 1985 to the data of recent years is coincident to the observation by Conley et al. (2009) who found 1986 to be a significant breakpoint in the Bay hypoxia trend. Kemp et al (2005) detected 2 separate significant relationships between hypoxia and nitrate loading for 1950–1979 and 1980–2001, with similar slopes but different intercepts, implying that the Bay has become less able to assimilate N inputs.

Hagy at el. (2004) collected July hypoxia from 1950 to 2001. If applied with the Scully (2010b) method, i.e., using May-July wind and counts of all wind events of > 2 m/s to conduct the correlation using Hagy et al. (2004) July hypoxia and winter-spring flow in 1950-2001, different results from Tables 1

and 3 were yielded: weaker correlations between July hypoxia and wind speed (r=+/- 0.1) or flow (r=0.2). On wind direction, it yielded moderate high correlation (r=0.6-0.7, p<0.05) between frequency of west wind and July hypoxia, and moderate negative correlation (r=-0.45) between frequency of southeast wind and July hypoxia. When the data were split into two periods (1950-1982 and 1985-2001), the correlations between hypoxia and river discharge were doubled, but the correlation between hypoxia and west wind frequency reduced 2/5 in each sub-period data. The split samples may increase data consistency among years in each subset, therefore, generally increase correlations among the variables that have physical links, such as hypoxia and river discharge. As pointed out by Browner and Newman (1987) and Taylor (1990), p and r values can only tell the significance of the correlation for the variables in the specifically selected sample, but does not tell whether they have a cause-and-effect relationship. The significant reduction of correlation between hypoxia and frequency of westerly wind in subsamples suggests that the high r and low p based on the data of entire period may not be highly related to their physical links. The wind events including May and June may not be appropriate to perform correlation with July hypoxia, because hypoxia may recover a few days after wind calms. The apparent high correlation derived from the lumped two periods of hypoxia data and the out-of-phased wind may not reflect their strong links in physical processes. Therefore, the importance of wind direction on modulating the interannual hypoxia variations based on such lumped data should be discounted. Together with other evidence presented in this article, we believe that the strength of winds (disregarding direction) is more important than direction in the overall cases, though a sustained southerly wind could reduce hypoxia more than a westerly wind.

Nutrient load versus wind in regulating interannual hypoxia, and implication to management

The variations of interannual summer hypoxia follow the variation of winter-spring nutrient load or flow (Fig. 1). The maximum difference in winter-spring loads among the years is over 100% of the mean. The fluctuation of interannual load has a potential to cause fluctuation of summer hypoxia. Due to long residence time (several months) in the estuary, nutrient and freshwater input in winter-spring can affect the eutrophication processes through the summer.

The maximum difference in average speeds or frequencies of wind of speed >=4 m/s in summertime among the years is about 20% of the mean, smaller than nutrient variations. The wind's reduction on the nutrient-induced hypoxia among different years is less pronounced by comparing to nutrient's variation. Note: in our analyzed data, hurricanes occurred only in late September of a few years, thus extreme events were excluded. In peak summer, hypoxia could recover to the pre-wind condition in the calm period (Fig. 2) because of re-stratification and excessive oxidation from the bottom oxygen demand. The inter-summers variations of the episodic wind events do not follow the variation pattern of winter-spring loads but vary among months in a summer, thus reduce wind's correlation with summer hypoxia in the annual scale.

Nevertheless, wind is important on modulating hypoxia variation among months within a summer.

Therefore, nutrient load (plus stratification by freshwater input) is the primary factor causing summer hypoxia, while wind partly reduces hypoxia episodically, as we see A) high correlation of summer (average) anoxic volume with watershed load (r=0.89), B) moderate low negative correlation (r=-0.4) between summer hypoxia and average wind speed or frequency of higher wind events (Tables 1 and 2), and C) nutrient load to have stronger correlation with summer hypoxia than with individual months' (such as July) hypoxia (e.g., r=0.89 vs. r=0.71).

Currently, the Bay partners use the average hypoxic volume from 6-8 monitoring cruises in each summer to evaluate Bay's water quality and to compare inter-annual hypoxia. Since wind can significantly modify hypoxic condition and wind varies frequently, more frequent observations could provide more representative conditions. For protecting living resources, we are better off using the DO observations that avoid big storms and the periods soon after a big storm to address the anoxic problem. The current practice in the Chesapeake Bay-wide long-term DO monitoring is considered acceptable, because it usually avoids strong storms. Thus, the monitored DO shows good correlation with watershed load. Although the averaged summer hypoxia better explains interannual variations, assessing hypoxia in July or other months is meaningful to capture the worst DO condition. Comparing to the hypoxic volume of DO<1 or <2 mg/l, the anoxic volume (DO<=0.2 mg/l) at lower depth is affected less by wind but may show sensitively due to greater percent change from the initial smaller values. It may be a better index to evaluate the influence on DO by nutrient load.

Figure 1 shows a decrease in the averaged summer wind speed from 1998 to 2012. It is mainly contributed by fewer wind events of speed >= 4 m/s, including fewer southerly wind events. This implies that the Bay would be subjected to more severe hypoxic problem in later years if they have the same nutrient load. Nevertheless, since 2012 is one of the lowest hypoxia year due to low load (Fig. 1), indicating the nutrient is still the main factor governing interannual hypoxia.

Higher temperature in the summer reduces oxygen solubility, promotes oxidation and reduces DO in the bottom water, as well as enforces the bottom-to-surface density stratification due to greater surface-to-bottom thermal gradient. However, Table 1 shows that the summer hypoxia has weak negative correlation with deep water temperature (Wtmp, r=-0.2). Deep water temperature in the summer is greatly impacted by stratification that prevents heat exchange between deep water and the warmer upper water and the air. The positive correlation of stratification (Strat) and summer hypoxia (r=0.5, Table 1) and negative correlation of stratification and Wtmp (r=-0.15 to -0.29, table omitted) causes the apparent negative correlation between Wtmp and hypoxia. While the summer average air temperature (Atmp) in individual years reflects the temperature in the entire Bay system, thus we see positive correlation (though weak, r=0.17) with summer anoxic volume. Therefore, air temperature

has more significance than deep water temperature on inter-annual variation of hypoxia. As the result of global warming, the Bay could be subjected to more severe hypoxic problem.

Conclusions

   Both watershed load and wind have significant impact on Chesapeake summer hypoxia, but in opposite directions. The increase of sediment oxygen demand due to watershed nutrient load is the key factor leading summer hypoxia in the Chesapeake Bay. The hypoxia can be maintained in deep stratified water, which prevents exchange with surface water. The discharge accompanied with high watershed load enhances stratification and summer hypoxia. Wind erodes stratification and reduces hypoxia. Based on the past three decades observation, the internannual changes of hypoxia in the Chesapeake Bay is mainly controlled by load from the watershed, while, the influence by the interannual changes in wind conditions is less significant.

   Wind conditions (e.g., speed and direction) vary in the time scale of hours, causing variations of hypoxia among months in a summer, which influence could be pronounced episodically. However, wind occurs naturally, and the magnitude of its variation in different summers is not as big as the interannual variation of nutrient load. While, the wind's destratification and hypoxia reduction can usually be recovered in a few days. There is no fix pattern of wind condition in the DO monitoring events among different years. The combination of these factors causes the observed summer average hypoxia to have weaker correlation with wind than with nutrient load.

   Although the reductions of hypoxic volume are different by different wind directions at a specific wind speed, the magnitude of wind speed (greater than certain threshold) is generally more important than wind direction in eroding stratification and hypoxia. There is certain impact due to decreased frequency of southerly winds in summertime in the last two decades, which plays a less significant role in controlling interannaual hypoxia than the variations in frequency of stronger wind events.

   Considering the scatted monitoring events and episodic winds, summer averaged hypoxia value may better reflect the Bay's DO problem and its relation to nutrient load than individual month's value. However, July or August hypoxia should also be analyzed since it may represent the worst DO period in a year.

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Table 1. *Correlations of anoxic volume (AV) with its influencing variables based on 1991-2005 data*

		Jan-May average			July or Summer average				
		ENP	SNP	Flow	Strat	Wspd	Cwd4	Wtmp	Atmp
AV_	July	0.77	0.71	0.82	0.20	-0.36	-0.42	-0.20	0.17
_Obs	Summer	0.83	0.89	0.89	0.50	-0.37	-0.42	-0.15	0.33
AV_	July	0.85	0.78	0.85	0.40	-0.66	-0.54	-0.24	0.34
_Mod	Summer	0.81	0.77	0.78	0.39	-0.53	-0.48	-0.29	0.36

Table 2. *Correlation coefficients of summer anoxic volume with counts of summer wind events that are over certain speed thresholds*

	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
AV_mod	-0.53	-0.25	-0.43	-0.47	-0.40	-0.33	-0.32	-0.34
Wspd	1.00	0.48	0.67	0.67	0.66	0.62	0.72	0.75

Table 3. *Correlation of summer anoxic volume with count of wind event in summer for wind directions at speed>=5 m/s*

	N5	NE5	E5	SE5	S5	SW5	W5	NW5	Cwd5
AV_obs	-0.38	-0.52	-0.52	0.24	0.24	0.05	-0.03	0.41	-0.27
AV_mod	-0.61	-0.71	-0.27	0.09	0.42	-0.13	0.25	0.38	-0.40
Wspd	0.88	0.87	0.31	-0.28	-0.34	-0.12	-0.14	0.11	0.66
Cwd5	0.57	0.56	0.10	0.19	0.32	0.43	0.35	0.36	1.00

Table 4. *Counts of wind event in 8 directions for 2 speed-ranges in June 25-July 24, 1996 and 1998*

Wind directions		N	NE	E	SE	S	SW	W	NW	All dir
Speed ranges	<4 m/s	149	31	28	37	78	44	53	56	476
	>=4 m/s	32	7	12	18	22	37	61	53	242
1996, all speeds Sum		181	38	40	55	100	81	114	109	718
Speed ranges	<4 m/s	161	18	20	20	79	99	91	43	531
	>=4 m/s	28	14	5	9	39	25	34	33	187
1998, all speeds Sum		189	32	25	29	118	124	125	76	718

Figure 1. *Summer average anoxic and hypoxic volume, wind speed, and winter-spring watershed nutrient load and freshwater discharge in 1985-2012*
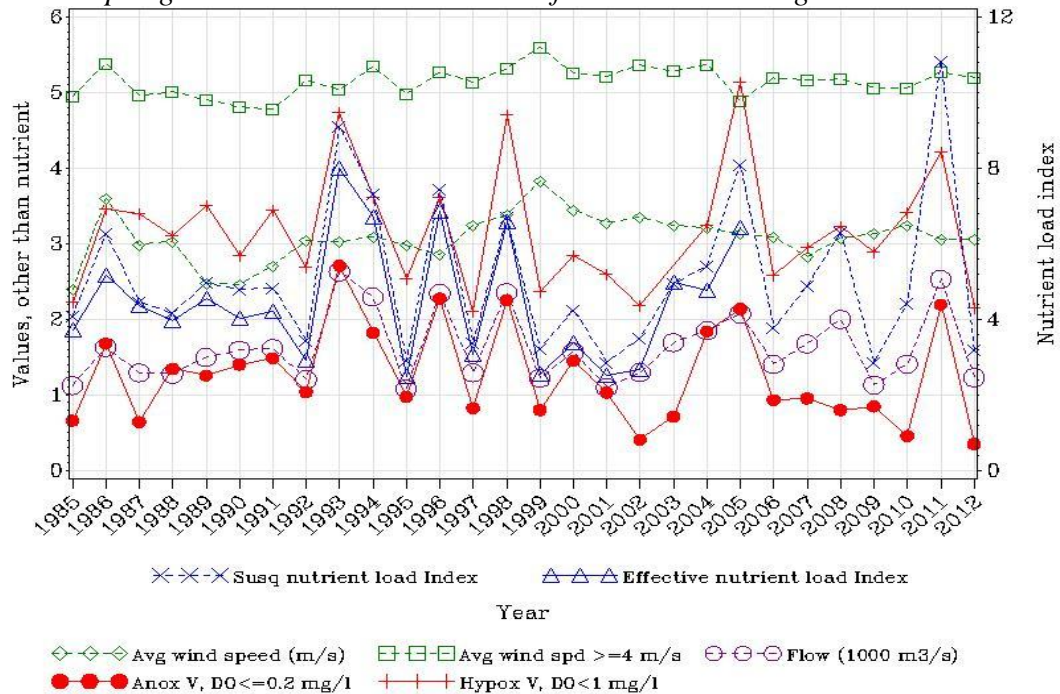
Figure 2. *Model simulated changes of anoxic volume versus no-wind by artificial wind directions. The wind starts at hour 8 on Day 10 in August, 1996. A) wind lasts for 2 and half days at speed =8 m/s, B) wind lasts for 1 hour, and the post-fix numbers 6 and 5 indicate wind speed (at m/s).*
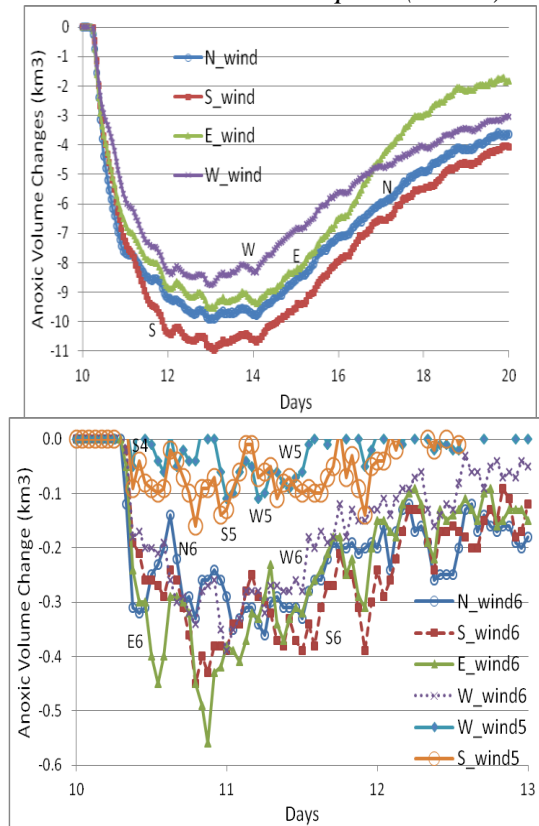

Figure 3. *Observed anoxic volume (DO<=0.2 mg/l, circles)) and hypoxia volume (DO<1 mg/L, bigger dots), modeled daily anoxic volume (pluses) and hourly wind speed (>= 6 m/s, small dots) in summer months (June-Sept) in 1991-2005. The annual winter-spring averaged nutrient load index (stars, non-unit)*
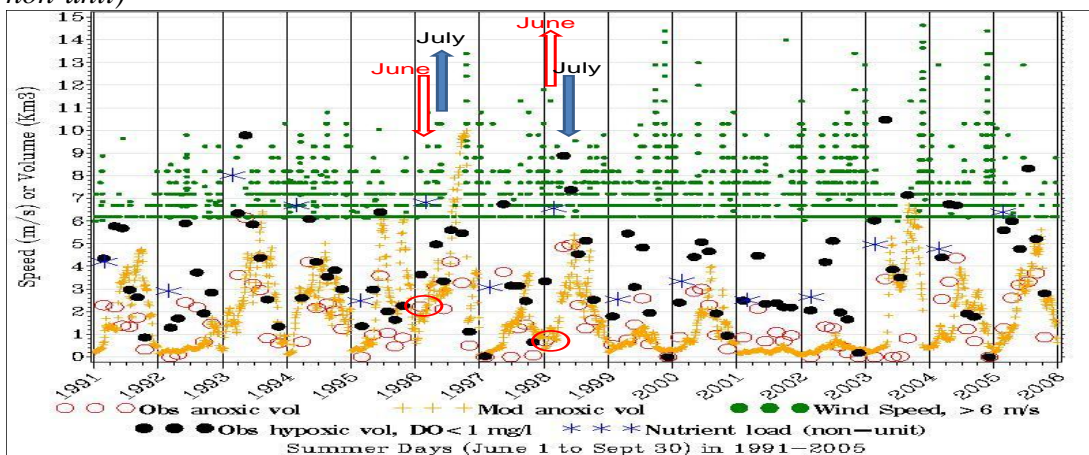
Figure 4. *Wind speed (dots, in m/s) and directions (circles, wind coming in degrees from north) for speed>=6 m/s in summer (June-September) of 1991-2005*