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Long-Term Phosphorus Loading and Springtime Temperatures Explain Interannual Variability of Hypoxia in a Large Temperate Lake

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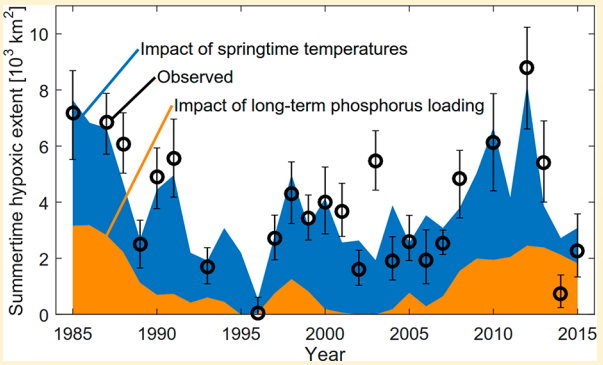
Supporting Information

ABSTRACT: Anthropogenic eutrophication has led to the increased occurrence of hypoxia in inland and coastal waters around the globe. While low dissolved oxygen conditions are known to be driven primarily by nutrient loading and water column stratification, the relative importance of these factors and their associated time scales are not well understood. Here, we explore these questions for Lake Erie, a large temperate lake that experiences widespread annual summertime hypoxia. We leverage a three-decade data set of summertime hypoxic extent (1985−2015) and examine the role of seasonal and long-term nutrient loading, as well as hydrometeorological conditions. We find that a linear combination of decadal total phosphorus loading from tributaries and springtime air temperatures explains a high proportion of the interannual variability in average summertime hypoxic extent (R^2 = 0.71). This result suggests that the lake responds primarily to long-term variations in phosphorus inputs, rather than springtime or annual loading as previously assumed, which is consistent with internal phosphorus loading from lake sediments likely being an important contributing mechanism. This result also demonstrates that springtime temperatures have a substantial impact on summertime hypoxia, likely by impacting the timing of onset of thermal stratification. These findings imply that management strategies based on reducing tributary phosphorus loading would take several years to reap full benefits, and that projected future increases in temperatures are likely to exacerbate hypoxia in Lake Erie and other temperate lakes.



INTRODUCTION

Human activities are impacting aquatic ecosystems in unprecedented ways, via changes ranging from the global to the local scale.[1] One of the manifestations of these impacts is the increased incidence of hypoxia, typically defined as a condition wherein dissolved oxygen (O_2) concentrations drop below 2 mg·L^{-1} and which is harmful to aquatic life. Hypoxia is of increasing concern across a variety of systems, ranging from estuaries to lakes.[2] One heavily studied system is Lake Erie, the warmest, shallowest, and most eutrophic of North America's Laurentian Great Lakes,[3] which together contain about a fifth of the global surface freshwater.[4] Because of its bathymetry, the central basin of the Lake Erie is the most prone to summer hypoxia, and there is evidence suggesting that some degree of bottom water hypoxia is naturally occurring.[5] However, it has been demonstrated that anthropogenic activity has exacerbated hypoxia for decades and that, since the mid-1990s, water quality in Lake Erie has once again been deteriorating.[6,7]

Identifying the main drivers of hypoxia is critical for setting up effective water management strategies, including nutrient load targets, and several studies have attempted to do so, both in Lake Erie and in other large temperate lakes. As in other freshwater systems, phosphorus (P) is the primary limiting nutrient for primary producers (e.g., algae, cyanobacteria, and macrophytes),[8,9] although there is some indication that nitrogen can also become limiting in the late summer.[10] The sedimentation of the produced biomass stimulates bacterial respiration and thus O_2 depletion in the hypolimnion.[6] For these reasons, a link between P loads from lake tributaries and hypoxia is clear.[6,7] It is less clear, however, which time scales are dominant, that is, whether springtime loading determines the severity of annual hypoxia, or, conversely, whether P sedimentation and subsequent long-term release via internal loading can be an important process influencing hypoxia.[11,12] In Lake Erie[3] and elsewhere,[8,13] it has been suggested that responses to changes in external P loads could take about a decade. In addition, hydroclimatic variability also affects lake hypoxia both indirectly and directly. Indirectly, more intense precipitation could for instance increase the P runoff from

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agricultural catchments.[14] Directly, warming temperatures can cause reduced O_2 solubility in water, increased microbial activity, and enhanced stratification (which hinders the downward flux of oxygen from surface waters),[2,15] while changes in wind patterns could also affect stratification.[16] Other hypotheses about key drivers, such as the intensity of wintertime diatom blooms in Lake Erie, have also been proposed.[17]

In this Article, we address two crucial yet unanswered questions: (i) Which of the suspected drivers of hypoxia are most important as indicated by the available evidence? (ii) What are the most significant time scales at which nutrient pollution and hydroclimatology impact hypoxia? We seek to answer these questions with an approach that is innovative in that (i) we analyze a very long data set of hypoxic extent for a large lake, spanning three decades; (ii) we combine a parsimonious multiple regression approach and Bayesian model averaging to define the importance of the examined drivers; and (iii) we examine a broader range of possible environmental drivers and associated time scales building on recent investigations in Lake Erie and similar lakes. In particular, with regard to the last point, we include, among other variables, long-term cumulative nutrient loading to examine the potential role of phosphorus accumulation in lake sediments and subsequent re-release into the water column (i.e., internal loading),[8,9,11,12,18] and air temperature to examine the drivers and impact of lake thermal stability.[8,15,19,20]

MATERIALS AND METHODS

Long-Term Record of Hypoxic Extent. Average summertime horizontal hypoxic extent is an important indicator of eutrophication and represents the response variable for the analysis. For Lake Erie, this period of record spans 31 years (1985−2015) and contains 25 data points, because in situ observations in 1986, 1992, 1994, 1995, 2009, and 2011 were insufficient to establish a seasonal-average hypoxic extent.[7] To our knowledge, this is the longest record to date of hypoxic extent for a large temperate lake. Estimates of hypoxic extent are derived from O_2 profiles collected in the central basin of Lake Erie by the U.S. EPA Great Lake National Program Office, Environment Canada, and NOAA GLERL as described by Zhou et al.[21] Here we complement the data for 1985−2012 from Zhou et al.[7] with three additional years of hypoxic extent estimates based on O_2 data from EPA,[22] and processed using the same approach as Zhou et al.[21] The implemented approach also yields uncertainty bounds via Monte Carlo simulations. We focus on average hypoxic extent across August and September because these are the months during which Lake Erie hypoxia is most severe.

Long-Term Record of Explanatory Variables. The investigation of the driving factors of summer hypoxic extent is based on a set of candidate explanatory variables, also known as independent variables, covariates, predictors, ancillary, or auxiliary data. We consider riverine P loads and discharge, temperature, and wind direction and speed. Together they cover the main land management and hydroclimatic factors that have been hypothesized to influence hypoxia.

We include total phosphorus (TP), dissolved reactive phosphorus (DRP, a.k.a. soluble reactive phosphorus, SRP), and discharge data for four tributaries, namely the Maumee River, the River Raisin, the Sandusky River, and the Cuyahoga River, which are the major tributaries located upstream of the central basin and for which over three decades of observations are available. Additionally, we also consider monthly discharge estimates for the Detroit River from the U.S. Army Corps of Engineers available via NOAA GLERL.[23] Monthly loading estimates for the Detroit River are not available for the period of record, however (SI). Daily or subdaily tributary measurements of instantaneous concentration were obtained from the Heidelberg University National Center for Water Quality Research[24] and aggregated to monthly scales. While for years preceding 2013, monthly tributary data were directly obtained from Zhou et al.,[7] and for 2013 onward, in a manner similar to that of Obenour et al.,[25] we obtained monthly averages by multiplying average daily TP or DRP concentrations from Heidelberg with discharge data courtesy of the U.S. Geological Survey.[26] Gaps in concentration data on the order of days were filled by using the average of the ten nearest days with available data (as in Ho and Michalak[18]). Gaps in concentration data longer than one month were filled with a regression approach using as input the data from the closest tributary (as in Zhou et al.[7]). Specifically, missing monthly loads for the River Raisin were filled using a regression with loads from Maumee River, while missing loads for the Cuyahoga River were filled using a regression with Sandusky River loads.

Two types of monthly temperature data are used as candidate variables, namely mean air temperatures over Lake Erie and its drainage basins (Figure S1) from NOAA GLERL[4] and water temperatures from a NOAA NDBC buoy[27] located in the central basin. Temperature data from the POWER project at the NASA Langley Research Center[28] were used to fill gaps in the air and water (buoy) temperature data sets. Monthly wind duration by direction and wind speed were also compiled based on wind data from the NDBC buoy (SI). While for years preceding 2013 monthly wind data were directly obtained from Zhou et al.,[7] for 2013 onward we followed the same approach as reported therein. We also include the square of the average monthly speed as a candidate variable, because a quadratic relationship between hypoxic extent and wind speed has previously been observed.[7] Wind duration by direction is defined as the number of hours in a month during which the wind is blowing from one of the four intercardinal (NW, 270° to 360°, NE, 0° to 90°, SE, 90° to 180°, SW, 180° to 270°) directions. Unlike other data sources, buoy data are typically only available starting in April of each year.

Statistical Analyses. We use multiple linear regression to model the interannual variability of hypoxic extent observed in the central basin of Lake Erie and select the combination of candidate variables that provides the best explanatory power. We focus only on linear models with up to two variables in order to maximize the robustness of the results and minimize the risk of overfitting. Candidate variables are categorized into five groups (Table 1). Group 1 represents the long-term cumulative P loads calculated over a window of one to 15 water years. For any given year, the loads were aggregated from the end of the current water year (September) backward. Given that tributary P monitoring began in October 1974 and the first estimate of hypoxia dates back to 1985, for cumulative loads requiring data prior to October 1974, we filled gaps by averaging all the available monthly load data (i.e., since October 1974) up to the year of interest. Group 2 represents same-year springtime P loads aggregated over one to six consecutive months. Group 3 includes seasonal discharge aggregated over one to six consecutive months from either the tributaries or the Detroit River or their sum (total discharge). Group 4 includes springtime air and water temperature data aggregated over one

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Table 1. Candidate Variables Used to Model the Interannual Variability in Summertime Hypoxia in Lake Erie

group	description	months[a]/years considered	selected variable[b]
1	cumulative TP and DRP loading from tributaries[c]	1−15 years	TP_{9y}
2	spring DRP loading from tributaries	January−July	
3	log of discharge from tributaries and Detroit River	January−July	
4	air and surface water temperature	January[d] or April−July	$T_{a,Mar-Apr}$
5	wind speed and wind duration by direction	April−July	

[a]All combinations of 1 to 6 consecutive months are considered. [b]Cumulative TP loading over nine water years (TP_{9y}, [Mg]) and average air temperature for March and April ($T_{a,Mar-Apr}$, [°C]). [c]n-year cumulative loads are calculated as the sum of the monthly loads going from September of the current year back to October of the previous n years. [d]While air temperature data are available all year long, water temperature data generally begin in April.

to six consecutive months. Group 5 consists of monthly wind variables.

We examine all combinations of variables, with the limitation that variables need to represent different mechanisms, and therefore be from two different groups. In addition to selecting the model with the highest overall explanatory power via ordinary least-squares (OLS), we also assess the robustness of the results through weighted least-squares (WLS), leave-one-out cross validation (LOOCV) and Bayesian model averaging (BMA). In WLS, we weigh each data point of the observed response by the reciprocal of the variance associated with the hypoxic extent estimates for each year (Figure 1). In LOOCV, one observation at a time is removed from the model calibration set, and the resulting model is used to predict the missing observation.[29] While the regression approach is prevalently frequentist, we also leverage a Bayesian framework to quantify the importance of each candidate variable based on its ability to explain the variance in the observed response, via BMA. In this approach, the importance of each variable is expressed as a posterior probability, which is equal to the sum of the posterior probabilities of all possible models containing the variable (e.g., eq 13 in Yadav et al.[30]). In other words, the importance of a variable is greater if the explanatory power of models including that variable is higher relative to other models.

RESULTS AND DISCUSSION

Explanatory Model for Hypoxia. We find that a linear combination of two variables, namely springtime air temperatures and multiyear cumulative TP loads, explains a high

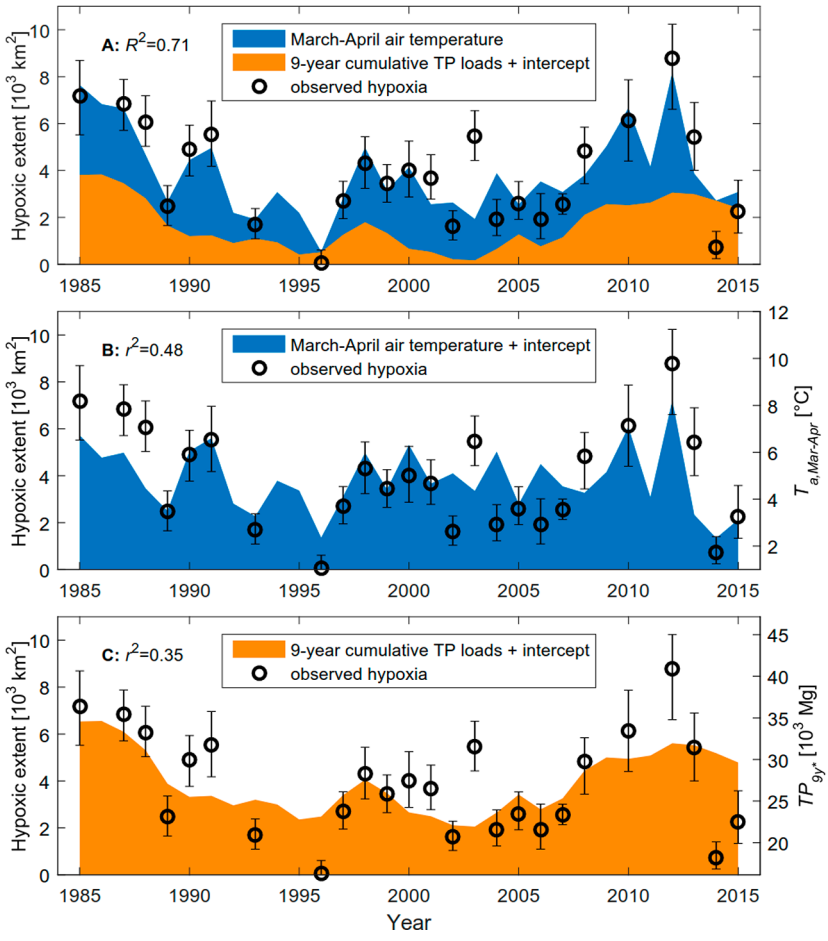

Figure 1. Comparison of the observed time series of average August−September hypoxic extent, including 95% confidence intervals, with the time series of (A) the model in eq 1 and (B, C) the individual predictors included in the model. Temperatures in (A) are minimum-deviated for visualization purposes, such that the contribution of $T_{a,Mar-Apr}$ is null for the coldest year.

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Table 2. Posterior Probabilities of Individual Variables Based on BMA[30] of All Possible Two-Variable Models

air temperature		annual TP loads		logarithm of tributary discharge		wind duration	
month aggregation	posterior probability[a]	year aggregation	posterior probability	month aggregation	posterior probability	month, direction	posterior probability
Feb–Mar	0.04	6-year	0.01	Jan–Jun	0.01	Jul, NW	0.02
Feb–Apr	*0.08*	7-year	*0.06*	Apr–Jun	0.01		
Feb–May	*0.07*	8-year	*0.12*	May–Jun	0.01		
Feb–Jun	0.03	9-year	**0.25**				
Feb–Jul	*0.05*	10-year	*0.06*				
Mar	*0.07*	11-year	*0.13*				
Mar–Apr	**0.38**	12-year	*0.05*				
Mar–May	*0.10*	13-year	0.03				
Mar–Jun	*0.05*	14-year	0.02				
Mar–Jul	*0.08*						
all aggregations[b]	**0.95**	all aggregations	**0.75**				

[a]Only variables with a posterior probability $\mathbb{P}$ above 1% are listed here. $\mathbb{P}$ is a measure of the importance of a variable in explaining the observed variability in hypoxic extent, when considering all possible sets of two candidate variables. Typeface indicates the relative probabilities: bold type for $\mathbb{P} \geq 0.15$, italic type for $0.15 > \mathbb{P} \geq 0.05$, and plain type for $0.05 > \mathbb{P} > 0.01$. [b]Note that water temperature is not included because none of its aggregations has $\mathbb{P} > 0.01$.

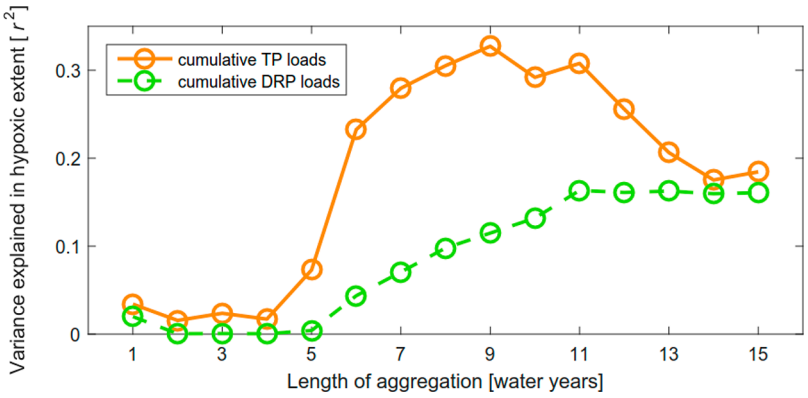

Figure 2. Explanatory power of cumulative phosphorus loads integrated over different durations. TP_{9y} is the best explanatory variable among those in group 1 (Table 1), and, its modified version, TP_{9y*}, is also part of the two-variable model with the overall highest explanatory power (eq 1, Figure 1).

proportion of the interannual variability in late-summer hypoxic extent H [10^3 km^2] in 1985–2015 (Figure 1). The two variables that together provide the highest explanatory power are the average air temperature for March and April ($T_{a,Mar–Apr}$, [°C]) and cumulative TP loading over nine water years (TP_{9y}, [Mg]). The BMA analysis also confirms that these two variables are the most important in explaining hypoxia across all possible combinations of predictor variables (Table 2). This result suggests that spring temperatures and long-term loading are more important factors than previously assumed. Indeed, most of previous studies on Lake Erie[7,31,32] did not consider these variables. We additionally explore the impact of truncating the cumulative TP loading term prior to the end of the current water year in September, while keeping full water years for the other eight years. We find that truncating this term in April of the modeled year, to coincide with the air temperature term, results in an almost identical R^2 (R^2 = 0.703 for nine full water years vs R^2 = 0.705 with the last water year truncated at the end of April). Because this latter version has the additional advantage of enabling prediction of late summer hypoxia by the end of April of each year, more than three months in advance of the peak hypoxic season, we present the version with the cumulative TP loading term truncated at the end of April of the year of interest here (TP_{9y*}, Figure 1)

$$H = 0.901 \cdot T_{a,Mar–Apr} + 2.88 \times 10^{-4} \cdot TP_{9y*} - 8.67 + \varepsilon \quad (1)$$

where ε are random errors assumed to be independently and normally distributed, with zero mean and estimated standard deviation equal to 1.24 [10^3 km^2]. The estimated standard deviations of the regression coefficients are 0.175 [10^3 km^2·(°C)$^{-1}$], 0.706 × 10^{-4} [10^3 km^2·(Mg)$^{-1}$], and 1.98 [10^3 km^2], respectively. Coefficients and errors in the regression model of hypoxic extent (eq 1) are estimated via OLS, and a LOOCV using this model yielded R^2 = 0.61. A sensitivity analysis using WLS rather than OLS yielded a model with the same two explanatory variables, only with slightly different regression coefficients:

$$H = 0.994 \cdot T_{a,Mar–Apr} + 2.72 \times 10^{-4} \cdot TP_{9y*} - 9.02 + \varepsilon \quad (2)$$

This model has R^2 = 0.68 and its fit is illustrated in Figure S2 (equivalent of Figure 1). The R^2 here indicates the proportion of variance explained in the untransformed response space and is sometime[33] designated as "pseudo-R^2_{WLS}". The LOOCV, BMA, and WLS demonstrate the robustness of the model and the roles of long-term phosphorus loading and springtime temperatures. We find that while spring temperatures are strongly associated with year-to-year variations of hypoxia, the cumulative loads explain its long-term trends (Figure 1).

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Whereas the data provide strong support for springtime air temperature and multiyear cumulative TP loading as the key explanatory factors, other combinations of months between February and July (air temperature) and other cumulative loading windows of between 7 and 12 years (TP loading) also provide strong explanatory power (Table 2). This is not surprising given the collinearity among variables. We therefore focus the discussion that follows broadly on multiyear cumulative TP loading and springtime temperatures, rather than on the specific time spans that form the single "best" model.

Long-Term P Loads. Anthropogenic nutrient loading has long been discussed as the main driver of O_2 depletion in lakes.[31] Interestingly, several studies have attempted to link single-year P loads to hypoxia, yet empirical results have shown little association between the two phenomena. In Lake Erie, for instance, Burns et al.[32] found only a mild correlation between hypolimnetic oxygen depletion rates and annual TP load (r^2 = 0.19), while Rucinski et al.[34] reported no correlation between water column oxygen demand and annual TP load (r^2 = 0.01). Similarly, here we find that single-year TP (r^2 = 0.03) and DRP (r^2 = 0.02) loading (Figure 2) are weak predictors of hypoxic extent.

Conversely, decadal P loads are shown here to be more strongly associated with hypoxic extent, which is consistent with long-term "memory" effects observed in some European lakes.[8,13] Earlier studies have shown that for both deep and shallow temperate lakes, a large fraction of the external P loading can be buried in sediments and gradually reintroduced over several years via internal loading. Our finding is also consistent with modeling[18] and experimental[12] studies of algal blooms in Lake Erie showing that a combination of P retained from loads of previous years and recent allochthonous P loads can together contribute to eutrophication events. As shown in Figure 2, the correlation with hypoxic extent reaches a maximum for an aggregation window of approximately a decade. In general, long-term TP loading appears more strongly associated with hypoxia than DRP, explaining twice the variance in hypoxic extent. TP accounts both for phosphorus that is immediately bioavailable (whether dissolved or particulate) and for the more stable fraction of the phosphorus load. Once settled into lake-bottom sediments, a portion of the nonbioavailable fraction can undergo biogeochemical transformation making it more bioavailable, especially under hypoxic conditions.[11,35] Consequently, cumulative TP load accounts both for the long-term release of bioavailable phosphorus retained through primary production and sedimentation and for more stable phosphorus that becomes more bioavailable over longer periods, in part through exposure to hypoxic conditions.[9]

Springtime Temperatures. Climatic variability has been recognized as an important factor impacting lake hypoxia and confounding the relationship between nutrient inputs and O_2 depletion.[2,7,8,15] Higher temperatures, in particular, are expected to exacerbate lake hypoxia via multiple physical and biochemical processes, a crucial one being the reinforcement and prolongation of lake stratification.[8,20,36] Stratification in dimictic lakes such as Lake Erie typically begins in spring when the water surface temperature increases above 4°C, creating a warmer layer of water on the top of the denser hypolimnion. The hypolimnion typically warms up much more slowly and to a lesser extent, maintaining a spring temperature slightly above 4°C.[15,16,37] The surface water is instead more strongly influenced by air temperatures.[19] This implies that higher spring air temperatures are likely to lead to an earlier onset of lake stratification and a higher thermal stability (related to the temperature difference between the epilimnion and hypolimnion).[20,38] Longer and stronger stratification leads to reduced transport of O_2 to the hypolimnion, and thus a higher risk or severity of hypoxia.[8,16,19]

To corroborate the hypothesis that warmer springs correlate with hypoxic extent because they lead to early onset of stratification, we use daily lake-wide surface temperatures from NOAA CoastWatch[39] to ascertain the day-of-year of onset of stratification. This data set has not been included among the candidate variables because data collection only began in 1995. The onset of stratification is defined as the first day of the year for which the water surface reaches a temperature of 5°C and does not go below this threshold until after the summer season. This definition is consistent among others with Schertzer et al.[37] who discussed that, in the context of the Great Lakes, summer stratification typically commences when lake surface reaches a temperature greater than 4°C. As shown in Figure 3, we observe that in years with warmer springs, thermal stratification begins earlier and hypoxic extent is generally larger. Interannual variability in summertime hypoxic extent is

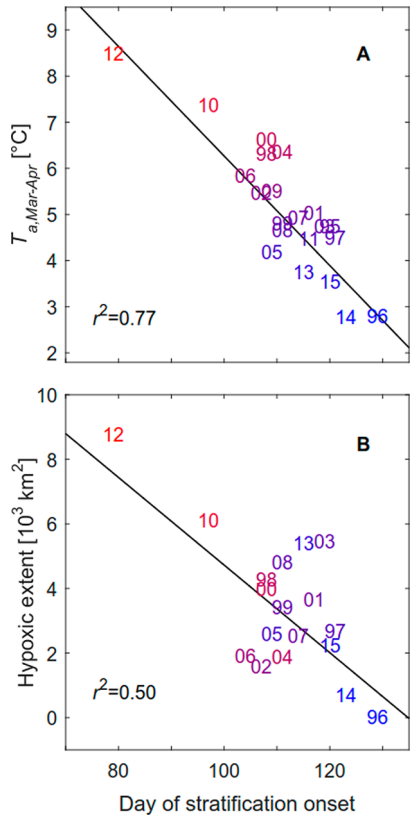

Figure 3. Relationships between onset of thermal stratification, air temperature, and hypoxic extent. The linear relationship between day-of-year of onset of thermal stratification and both spring temperatures and summertime hypoxic extent is strongly significant ($p \leq 0.001$). Data points are displayed as a number representing their year, with color progressing from blue for the coldest year to red for the warmest one. One of the two predictors of the model in eq 1 is strongly associated with day of stratification onset (A), which in turn is strongly associated with hypoxia (B). Because of the difference in data availability, years in panels A and B do not overlap completely.

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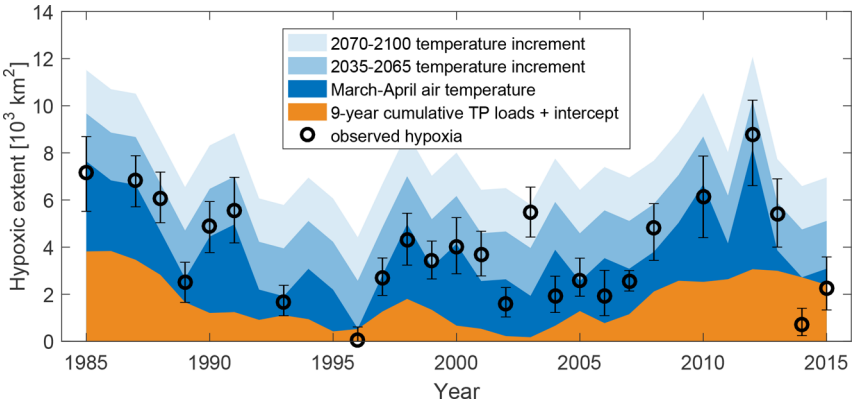

Figure 4. Temporal evolution of observed and modeled hypoxic extent as in Figure 1A. Additionally, we show, in lighter shades of blue, the modeled hypoxic extent that we would expect as a result of average warming projected for the Lake Erie watershed (Figure S1) for the mid- and late-century, assuming the same loads as in the period 1976–2015. Future March–April temperatures are calculated by adding to the observed temperatures a constant increase (2.3 and 4.3°C for mid- and late-century, respectively) derived from CMIP5 model projections under the RCP8.5 scenario (details in the SI).

consistently associated with corresponding variability in springtime air temperature. Indeed, air temperature is the best single predictor of hypoxia, with March to May temperature alone explaining 49% of the interannual variability in hypoxic extent over the examined period. (The March to April temperatures included in the best two variable model explain 48% of the variability.) Interestingly, monthly surface water temperatures are less correlated to hypoxic extent relative to air temperatures. (For instance, April–July water temperature explains only 21% of hypoxic extent variability.) This evidence provides substantial support to the hypothesis that air temperature influences hypoxia mainly by determining the timing of onset of springtime thermal stratification rather than by simply affecting the long-term average water temperature. Indeed the higher explanatory power of the date of onset of stratification relative to that of monthly or seasonal water surface temperatures themselves suggests that the "stratification onset" hypothesis is more plausible relative to other possible mechanisms connecting warmer springs to more severe hypoxia, such as enhanced biological activity,[2,15] which might instead be significant for longer-term climatic shifts. In other words, the timing of surface water temperatures exceeding a threshold linked to the establishment of thermal stability appears to be more important for hypoxia than the average temperature itself.

Other Possible Influential Factors. While the proposed parsimonious model can explain 71% of the observed variability in seasonally averaged hypoxic extent, studying outliers can provide insights into additional contributing factors. Out of the 25-year record of hypoxic extent, there are three years that are less consistent with the model: 2003, 2004, and 2014. These extents can in part be explained in light of the drivers proposed by Zhou et al.[7] In particular, the small hypoxic extents in 2004 and 2014 were likely caused by unusually high wind stress in July, which would have served to reoxygenate the bottom of the lake.[16,37] Indeed, average July wind speeds were the second and third highest over the examined period, respectively, and the square of July wind speed (a simple proxy for wind stress[40]) is mildly and negatively related to hypoxic extent over the full examined period (r^2 = 0.16, p = 0.05). Conversely, the larger hypoxic event in 2003 is more difficult to explain. One hypothesis could be that very high springtime (May–July) DRP loading (307 Mg, representing the second highest value over the examined period, almost triple the median of 106 Mg) played a role, although the model performed well during other years with high spring loading. Note that by excluding the outlier in 2003 the variables in eq 1 would yield a model with R^2 = 0.82. In addition to the above-mentioned variables, we also find that tributary discharge is negatively related to hypoxic extent across the examined period (r^2 = 0.37, p = 0.001 for the natural logarithm of April–June discharge), similarly to what had previously been observed in other studies.[7,41] Although lower in overall importance when compared to air temperature and long-term P loads based on the analysis presented in Table 2, discharge does figure among the best fitting two-variable models. While it is unlikely that the variability in discharge of the studied rivers can significantly affect lake circulation and residence times,[7] it is plausible that low discharge is associated with other hydrometeorological conditions, including higher temperatures. For instance, we observe that the negative linear relationship between January–June discharge and March–April air temperature is substantial (r^2 = 0.16, p < 0.02). Lower January–June discharges can be linked to warmer springs by considering that evapotranspiration is temperature-dependent.[42]

Discussion and Implications for Management. Here, we show for the first time that long-term P loads and springtime temperatures explain a large fraction of the interannual variability in seasonally averaged hypoxic extent in Lake Erie. The main strengths of this study are that (i) we use three decades of observations at lake scale, which is a very long horizon compared to studies that focus on explaining hypoxia for one or a few years,[9,11,15,16,36,43] and (ii) we use a parsimonious regression approach in which we model the evolution of hypoxia with only two variables, while probabilistically computing the importance of each candidate variable.

The evidence provided in this investigation supports previous hypotheses on the impact of nutrient runoff and hydroclimatic factors on hypolimnic O_2 levels in Lake Erie. Additionally, we are able to ascertain the temporal scales that appear most important in explaining interannual variability in summertime hypoxia and provide a model that can be used to forecast the severity of late summer (August–September) hypoxia in Lake Erie by the end of April each year, providing actionable information for water managers and stakeholders.

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We find that tributary nutrient loads may affect lake quality on decadal scales, far longer than those previously investigated (months to one year),[6,7] which is consistent with recent hypotheses about the potential importance of internal loading and multiannual nutrient recycling.[3,8,12,18,35] Also interesting is the finding that cumulative TP explains approximately twice as much variability in hypoxic extent as cumulative DRP. This is a large-scale corroboration of field studies finding that, in hypoxic conditions, some of the stable particulate fraction of TP can be released and become bioavailable.[9,11]

We also find that the influence of seasonal air temperatures on lake thermal stability is an important explanatory factor for the severity of hypoxia. This is consistent with findings that rising temperatures prolong and reinforce stratification and thus hypoxia both in Lake Erie[16,36] and in other similar systems.[2,8] In fact, the model in eq 1 can be combined with temperature projections from global climate models to anticipate the effect of changing springtime temperatures on hypoxia. In Figure 4 we show what the hypoxic extent could have been over the recorded period, had average temperatures been akin to those expected in the middle (2035−2065) and end of the century (2070−2100) under the CMIP5 RCP8.5 "business-as-usual" scenario.[44] These estimates are derived by computing the increase in the mean March−April temperature for each future period relative to the modeled temperatures over the historical period (1985−2015), adding this increase to the recorded temperature for March−April 1985−2015, and using the modified temperature record in eq 1 (details in SI).

Each degree of warming corresponds to an approximately 900 km^2 increase in expected hypoxic extent (eq 1). All other factors remaining equal, we find that the average hypoxic extent by the middle of the century could be similar to the fifth largest recorded historical event. By the end of the century, the average extent could be almost as large as the largest recorded event to date. These estimates are likely to be conservative because they do not account for other mechanisms responding to long-term warming, such as rising hypolimnetic temperatures[8,20] leading to increased oxygen depletion rates[15] nor for indirect impacts of climate change such as higher loads resulting from the expected increases in precipitation in the region.[14,45]

In summary, via empirical modeling we identify specific time scales, factors, and mechanisms that explain trends in Lake Erie hypoxia. Overall, our results indicate that both local direct impacts (connected to land use and management) and global indirect ones (connected to climate variability and change) play an important role in controlling lake O_2 depletion. These findings have two main implications for water and nutrient management: first, any reductions in phosphorus loading may take longer than previously expected to reap full benefits and, second, a warming climate could partially offset benefits associated with loading reductions via earlier onset of stratification.

■ ASSOCIATED CONTENT

Supporting Information

The Supporting Information is available free of charge on the ACS Publications website at DOI: 10.1021/acs.est.7b04730.

Description of temperature gap-filling; analyses of Detroit River loading; description of the temperature projections; map of the Lake Erie watersheds; time series of hypoxic extent modeled with eq 2; comparison of loading estimates for the Detroit River; comparison of temperature projections and observations; and climate models used for temperature projections (PDF)

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Notes

The authors declare no competing financial interest.

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