

1 **Understanding interannual, decadal level variability in paralytic shellfish poisoning**
2 **toxicity in the Gulf of Maine: the HAB Index**

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1 **Abstract**

2 A major goal in harmful algal bloom (HAB) research has been to identify mechanisms
3 underlying interannual variability in bloom magnitude and impact. Here the focus is on
4 variability in *Alexandrium fundyense* blooms and paralytic shellfish poisoning (PSP) toxicity in
5 Maine, USA, over 34 years (1978 – 2011). The Maine coastline was divided into two regions -
6 eastern and western Maine, and within those two regions, three measures of PSP toxicity (the
7 percent of stations showing detectable toxicity over the year, the cumulative amount of toxicity
8 per station measured in all shellfish (mussel) samples during that year, and the duration of
9 measurable toxicity) were examined for each year in the time series. These metrics were
10 combined into a simple HAB Index that provides a single measure of annual toxin severity
11 across each region. The three toxin metrics, as well as the HAB Index that integrates them,
12 reveal significant variability in overall toxicity between individual years as well as long-term,
13 decadal patterns or regimes. Based on different conceptual models of the system, we considered
14 three trend formulations to characterize the long-term patterns in the Index – a three-phase
15 (mean-shift) model, a linear two-phase model, and a pulse-decline model. The first represents a
16 “regime shift” or multiple equilibria formulation as might occur with alternating periods of
17 sustained high and low cyst abundance or favorable and unfavorable growth conditions, the
18 second depicts a scenario of more gradual transitions in cyst abundance or growth conditions of
19 vegetative cells, and the third characterizes a “sawtooth” pattern in which upward shifts in
20 toxicity are associated with major cyst recruitment events, followed by a gradual but continuous
21 decline until the next pulse. The fitted models were compared using both residual sum of squares
22 and Akaike’s Information Criterion. There were some differences between model fits, but none
23 consistently gave a better fit than the others. This statistical underpinning can guide efforts to
24 identify physical and/or biological mechanisms underlying the patterns revealed by the HAB

1 Index. Although *A. fundyense* cyst survey data (limited to 9 years) do not span the entire interval
2 of the shellfish toxicity records, this analysis leads us to hypothesize that major changes in the
3 abundance of *A. fundyense* cysts may be a primary factor contributing to the decadal trends in
4 shellfish toxicity in this region. The HAB Index approach taken here is simple but represents a
5 novel and potentially useful tool for resource managers in many areas of the world subject to
6 toxic HABs.

7 **Keywords:** *Alexandrium fundyense*; harmful algal blooms; HABs; PSP; HAB Index

8

9 **1 Introduction**

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11 Harmful algal blooms (HABs) are a common threat to public health and coastal economies
12 throughout the world. In many affected areas, the phenomena are regular, annual events, but in
13 other cases, occur sporadically. Blooms vary in frequency, geographic extent, duration, shellfish
14 toxicity, and cell abundance. This multifaceted interannual variability poses significant
15 challenges to management programs that seek to protect fisheries resources and public health,
16 and as a result, it is often necessary to monitor shellfish and phytoplankton over broad areas and
17 long intervals of time, even in years when PSP outbreaks are spatially and/or temporally limited.

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19 A major goal for those conducting HAB research has therefore been to identify the
20 mechanisms underlying interannual variability in bloom magnitude and impact in a given region.
21 In some cases, these efforts examined atmosphere/ocean forcings such as the El Niño-Southern
22 Oscillation (ENSO; Maclean, 1989; Usup and Azanza, 1998; Azanza and Taylor, 2001; Moore et
al., 2008), or longer-term cycles, such as North Atlantic Oscillation (NAO; e.g. Alvarez-Salgado

1 et al., 2003), and the Pacific Decadal Oscillation (PDO; Erickson and Nishitani, 1985; Trainer et
2 al., 2003). White (1987) suggested a possible correlation between periods of greatest shellfish
3 toxicity in the Bay of Fundy (BOF) due to *A. fundyense* blooms and a cyclical phenomenon such
4 as an 18.6-year lunar cycle. Martin and Richard (1996) also examined data from the BOF using
5 five-, seven-, eight-, and ten-year moving average statistical techniques. No clear cycles were
6 observed, although other trends were suggested from the 51-year data set. These included
7 periods of higher toxicity in the mid-1940s, the early '60s, the late '70s, and mid-'90s. The
8 underlying causes of these toxicity regimes were not specified.

9 This study examines interannual variability in *A. fundyense*² blooms and the associated
10 PSP toxicity in the Gulf of Maine (GOM). Historically, toxicity was largely restricted to eastern
11 Maine and the BOF until 1972, when a massive, visible red tide of *A. fundyense* caused toxicity
12 in many western gulf areas for the first time (Hartwell, 1975). Since 1972, PSP toxicity has been
13 a recurrent problem throughout the region, including western gulf waters, and is responsible for
14 widespread shellfish harvesting closures and significant economic losses virtually every year
15 (Shumway et al., 1988; Jin et al., 2008). Since the major 1972 outbreak, annual toxicity has
16 varied extensively within the region, both in geographic extent and magnitude (McGillicuddy et
17 al., 2005b, 2011; Luerssen et al., 2005; Thomas et al., 2010; Horecka et al., this issue; Kleindinst
18 et al., this issue).

19 An important feature in *A. fundyense* population dynamics and toxin production is the
20 hypnozygote or resting cyst that is part of this organism's life history (Anderson, 1998). These
21 dormant cells fall from the water column during blooms, accumulating in the sediments
22 (Anderson and Wall, 1978; Anderson et al., 2005c) and near-bottom waters (Kirn et al., 2005;

² In this study, we have focused on the harmful algal species *Alexandrium tamarense* Group I, which we refer to as *A. fundyense*, the renaming proposed by Lilly et al. (2007).

1 Pilskaln et al., this issue; Butman et al., this issue) where they remain until conditions are
2 suitable for germination. Given the temperate climate of the GOM region and the virtual
3 absence of *A. fundyense* cells in the water column and rare occurrence of toxins in shellfish
4 during winter, blooms are heavily dependent on resting cysts for overwintering and eventual
5 bloom inoculation and recurrence. It is thus important to consider changes in cyst abundance in
6 this study of interannual variability in toxicity.

7 It is also important to understand the regional bloom dynamics and links to coastal
8 hydrography and cyst “seedbeds”. Conceptual models of *A. fundyense* bloom dynamics in the
9 GOM have been provided by Anderson et al. (2005c) and McGillicuddy et al. (2005a). Key
10 features are two cyst seedbeds - one in the BOF, and the other in mid-coast Maine offshore of
11 Casco and Penobscot Bays (Fig. 1; Anderson et al., 2005c; Anderson et al., this issue). Cysts
12 germinate from the BOF seedbed, causing recurrent *A. fundyense* blooms within a retentive eddy
13 at the mouth of the Bay that are self-seeding with respect to future outbreaks in that area (White
14 and Lewis, 1982; Aretxabaleta et al., 2008, 2009; Martin et al., this issue). Some cells escape the
15 retention zone and enter the eastern segment of the Maine Coastal Current (MCC) where they
16 bloom. Some of these are advected into the western GOM, while others undergo sexual
17 reproduction and fall to the bottom as cysts that accumulate in a feature hereafter termed the
18 mid-coast Maine seedbed near Casco and Penobscot Bays (Anderson et al., 2005c; Anderson et
19 al., this issue). The mid-coast Maine cysts (combined with vegetative cells from upstream
20 populations) contribute to blooms that cause toxicity in western portions of the gulf and possibly
21 offshore waters as well.

22 There are many factors that can regulate bloom size and the extent to which cells are
23 delivered to nearshore shellfish, including temperature, sunlight, nutrients, rainfall, and wind

1 speed and direction. It is thus not surprising that the geographic extent and magnitude of PSP
2 toxicity has been highly variable over the years that it has been monitored in Maine, New
3 Hampshire, and Massachusetts (Shumway et al., 1988; Bean et al., 2005; Kleindinst et al., this
4 issue). Here we describe a study that uses a simple approach to investigate interannual
5 variability in Maine shellfish toxicity. We collapsed 34 years of shellfish toxicity data from
6 weekly sampling at 160 stations in Maine into a novel "HAB Index" that can be used to
7 characterize the severity of PSP toxicity in a given year. We use this Index to identify decade-
8 level eras or regimes in toxicity in two Maine subregions. Given the significant relationship
9 between cyst abundance and the magnitude of subsequent blooms established in other studies
10 (McGillicuddy et al., 2011; Anderson et al., this issue), we hypothesize that the decadal
11 variability observed in Maine toxicity may reflect differences in regional cyst abundance,
12 although we recognize that this is based on a limited cyst abundance data set and will thus
13 require further testing.

14 **2 Methods**

16 *2.1 Blooms and shellfish toxicity*

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18 For the purposes of this paper, we have adopted an operational definition of the word
19 “bloom” to mean that the concentration of *A. fundyense* vegetative cells in contact with
20 nearshore shellfish populations is sufficient to cause detectable toxicity (≥ 40 μg saxitoxin
21 equivalents (STX eq.) per 100 g of shellfish tissue). We do this because the metrics used to
22 calculate the HAB Index all depend on shellfish toxicity measurements.

23 Throughout the years of this study, the state of Maine measured shellfish toxicity weekly at
24 160 stations along the coast during months that have a risk of PSP toxicity (generally March –
25 November), and closed harvesting when PSP toxin levels exceeded the action limit of 80 μg

1 STX eq. 100g⁻¹ shellfish meat. The limit of detection for the mouse bioassay is ~ 40 µg STX eq.
2 100g⁻¹ shellfish meat. Eleven species of shellfish were tested, with more than 3000 samples
3 analyzed annually. To reduce potential bias introduced by species-specific toxin uptake and
4 depuration rates (Bricelj and Shumway, 1998), we focus our HAB Index analysis on toxicity in
5 blue mussels – *Mytilus edulis*.

6 A PSP toxin-monitoring program has existed in the state since 1958, but in early years, the
7 number of stations sampled was limited and the schedule of sampling was sporadic (Hurst, 1975;
8 Shumway et al., 1988). It is only since 1977 that a consistent set of stations has been monitored
9 on a regular basis along the entire coast. We thus have a 35-year dataset from 160 stations that
10 extends to 2011. Note, however, that in 1977, there was an irregular sampling strategy, in that
11 no *Mytilus* samples were taken in eastern Maine until the end of September that year. Thus, 1977
12 is not included in our analysis and the total dataset spans 34 years. Throughout this long-term,
13 state monitoring program, PSP toxins in shellfish were measured using the AOAC mouse
14 bioassay (Association of Official Analytical Chemists, 1980).

15 2.2 *Maine subregions*

16 Hereafter, “eastern Maine” is used to describe the portion of the Maine coastline extending
17 from the Canadian border near the mouth of the BOF to the midpoint of Penobscot Bay (Fig. 1).
18 The “western Maine” subregion extends from Penobscot Bay to the New Hampshire border.
19 Note that this terminology is tied to the coastline of a single state, whereas other GOMTOX
20 studies that address the waters of the GOM have used EGOM to describe the subregion of the
21 Gulf extending from the BOF to Penobscot Bay, and WGOM to describe the subregion from
22 Penobscot Bay to Massachusetts waters (e.g. Anderson et al., this issue). The rationale for the
23 regional separation at Penobscot Bay is given below.

1 2.3 *The Maine HAB Index*

2 The HAB Index is calculated from records of PSP toxicity in *M. edulis* from 160
3 monitoring stations in Maine over the interval 1978-2011 (Maine Department of Marine
4 Resources; Table 1). The first parameter or metric in the Index is the percent of stations within
5 each subregion (i.e., eastern and western Maine) where detectable toxicity (i.e., >40 µg STX eq.
6 100g⁻¹) was measured for that year. These percentages were grouped into 5 equal bins (i.e., 0-
7 20%, 21-40%, etc.), and each bin assigned a number from 1 to 5. For the second parameter,
8 toxicity levels measured in *M. edulis* at each sampling station and time were summed over the
9 bloom season for all stations for each subregion, and then that total is divided by the number of
10 stations in that subregion and binned into ranges of 500 µg STX eq. 100g⁻¹ increments that are
11 then assigned a numerical value from 1 to 5. Thus “cumulative toxicity per station” is the second
12 parameter in the Index. Normalizing the cumulative toxicity to the number of stations in a
13 subregion allows the western and eastern subregions of Maine to be directly compared, as they
14 have different numbers of stations (90 and 70 respectively). The final Index parameter is the
15 duration of detectable mussel toxicity measured at stations affected by the widespread, coastal *A.*
16 *fundyense* blooms – i.e., excluding Lumbos Hole, Maine, an inland, isolated site where blooms
17 are initiated and develop in situ. The parameter represents the interval between the first
18 detectable toxicity at any station in either subregion and the last measurable toxicity at any
19 station within either subregion, with durations (total days) binned and assigned a numerical value
20 between 1 and 5.

21 Numerical values (bin numbers) for these three parameters (Table 1) are then summed for
22 each year to give a single Index value for the western and eastern regions for that year (Table 2).
23 The Index can thus vary between 3 and 15. Note that on occasion, subjective decisions were

1 made on some of these parameters at some stations (such as when late-season toxicity persisted
2 through the winter in the absence of vegetative cells, thereby affecting the apparent start date of
3 toxicity the following year, and thus the duration calculations). Details of this type, as well as a
4 narrative on how different factors might affect the Index calculations each year are given in
5 online Supplementary Materials. Additional information about the annual PSP closures is given
6 in Kleindinst et al. (this issue) and in the associated Supplementary Materials for that paper.

7 2.4 *Statistical analyses*

8 Differences in the onset, duration, and termination of PSP toxicity were compared between
9 regions with a *t*-test performed using JMP 8.0 (SAS Institute, Cary, NC), with a significance
10 level of $\alpha=0.05$. Onset and termination data were square-transformed and the cumulative toxicity
11 data were log-transformed to improve normality and meet homoscedasticity. These
12 transformations produced homoscedastic data, and all but two datasets were normally distributed
13 (EGOM, onset; WGOM, termination). Duration data did not require transformation.

14 Time series data for the HAB Index were analyzed to characterize changes in the HAB
15 Index over the 34-year interval spanned by this study. Three different statistical models were
16 fitted to the HAB Index time-series: a two-phase linear model, a mean-shift model, and a pulse-
17 decline model. The goodness of fit for each model was measured by the residual sum of squares
18 (*RSS*) for each data set, and the first two models were compared using Akaike's Information
19 Criterion (AIC; Akaike, 1974) to determine which analysis best described temporal trends in
20 toxicity. Due to the parameter constraints of the third model, it was not possible to include it in
21 the AIC comparison. For these analyses, the eastern and western regions were examined
22 separately and together. Model formulations are described in detail below.

1 Let $Y_t, t = 1, 2, \dots, n$, be the time series of the HAB Index for a specific region. A general
2 statistical model for this time series is:

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$$Y_t = \mu_t + \varepsilon_t \quad (1)$$

5
6 where μ_t is the unknown time-varying mean or trend and ε_t is a normal error with mean 0 and
7 unknown variance σ^2 . Three trend models were considered. For the mean-shift model:

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$$\begin{aligned} \mu_t &= \mu(1) & 1 \leq t \leq \tau_1 \\ & \mu(2) & \tau_1 < t \leq \tau_2 \\ & \mu(3) & \tau_2 < t \leq n \end{aligned} \quad (2)$$

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11 Under this model, the mean of the HAB Index shifts between three levels at unknown change-
12 points τ_1 and τ_2 . This model represents a “regime shift” or multiple equilibria formulation as
13 might occur with alternating periods of sustained high and low cyst abundance or favorable and
14 unfavorable cell growth conditions.

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16 For the two-phase model:

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$$\mu_t = \beta_o + \beta_1 t + \beta_2 (t - \tau) I_\tau(t) \quad (3)$$

19 where $I_\tau(t) = 0$ for $t < \tau$ and $I_\tau(t) = 1$ for $t \geq \tau$. Under this model, the HAB index exhibits a linear
20 trend with slope β_1 through unknown time τ after which the slope changes to $\beta_1 + \beta_2$. This
21 model depicts a scenario of more gradual transitions in cyst abundance or growth conditions.

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For the pulse-decline model, the HAB index exhibits a linear decline with pre-change slope β_1 through unknown time τ , an upward shift at time $\tau + 1$, followed by a post-change linear decline with slope γ_1 through time n :

$$Y_t = \begin{cases} \beta_o - \beta_1 t + \varepsilon_t & 1 \leq t \leq \tau \\ \gamma_o - \gamma_1 (t - (\tau + 1)) + \varepsilon_t & \tau < t \leq n \end{cases} \quad (4)$$

with:

$$\begin{cases} \beta_1, \gamma_1 \geq 0 \\ \gamma_o \geq \beta_o - \beta_1 \tau \end{cases} \quad (5)$$

These parameter constraints ensure that the model has the hypothesized "sawtooth" pattern in which upward shifts in toxicity are associated with major cyst deposition events, followed by a gradual but continuous decline until the next pulse.

The mean shift model contains a total of 5 unknown trend parameters – $\mu(1), \mu(2), \mu(3), \tau_1, \tau_2$ – and the error variance σ^2 . The two-phase model contains 4 unknown trend parameters – $\beta_o, \beta_1, \beta_2, \tau$ – and the error variance σ^2 . The pulse-decline model contains 5 unknown trend parameters – $\beta_o, \beta_1, \tau, \gamma_o, \gamma_1$ – and the error variance σ^2 .

Briefly, for fixed values of the changepoints, the maximum likelihood (ML) estimates of the trend parameters for each of these models can be found by minimizing the *RSS* (i.e., the sum of squared differences between the observed and fitted values of the HAB Index) and the ML estimate of σ^2 is given by RSS / n where *RSS* is the minimized value of the *RSS*. In the case of the pulse-decline model, this minimization must be subject to the parameter constraints in (5).

The overall ML estimates can then be found by varying the changepoints until *RSS* is minimized.

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In statistical terminology, the three trend models are non-nested in the sense that they cannot be made equivalent by restricting parameter values. A standard approach to comparing non-nested models is through AIC. For normal models, this is given by:

$$AIC = n \log(RSS / n) + 2 p \quad (6)$$

where p is the number of parameters. The model with the smallest value of AIC is selected. When, as here, the number of observations is not much greater than the number of parameters, the small-sample version of the AIC is recommended (Burnham and Anderson, 2010). For normal models, this is given by:

$$AIC_c = AIC + (2 p (p + 1)) / (n - p - 1) \quad (7)$$

and again the model with the smallest value of AIC_c selected. The imposition of parameter constraints invalidates the use of the AIC for the pulse-decline model, so it was not included in this comparison.

3 Results

3.1 Toxicity Metrics

In this study, three metrics of toxicity were examined within each Maine subregion: the percent of stations showing detectable toxicity over the year, the cumulative amount of toxin per station measured in all mussel samples during that year, and the duration of measurable toxicity

1 (Tables 1, 2). Patterns evident in each of these time series are described in detail below.
2 Noteworthy features of toxicity and some additional commentary are available in online
3 Supplementary Materials.

4 Figure 2 shows the percent of stations with detectable levels of toxicity for each year
5 during the study period. Distinct differences are evident in the trends observed for the eastern
6 versus western Maine stations using this metric. Of the 70 stations in eastern Maine, a fairly
7 constant number were toxic from 1978 through 1995 (~30%). This interval was followed by a
8 decrease in the percent of toxic stations from 1996 to approximately 2000 (generally <10 %),
9 which in turn was followed by an increase in the mid 2000s to levels not observed previously (77
10 – 94 %). In western Maine, there were high percentages of toxic stations in the late 1970s and
11 early 1980s (70– 90%) decreasing to minimum values in the late 1990s (10 - 20 %). Beginning
12 in 2002, a rising trend in the percent of toxic stations is documented, reaching levels of 60 - 90 %
13 of the stations each year once again.

14 Consistent with the relatively moderate percent of toxic stations in eastern Maine from
15 1978 through 1995 (Fig. 2), the cumulative annual toxicity per station measured in that region
16 was also moderate over that interval (Fig. 3). In the late 1990s, this value dropped even lower,
17 and then increased from 2003 to 2011, though considerable variability is evident. In western
18 Maine, the cumulative toxicity was high in the late 1970s and '80s and low through the 1990s,
19 with an apparent increase after 2003, again with fluctuations in recent years. On average, the
20 cumulative toxicity per station was not significantly different between eastern and western Maine
21 ($p=0.66$), although if this comparison is made over the early high toxicity period of the Index
22 (1978-1990), western Maine stations were significantly more toxic than eastern Maine stations

1 (p=0.0003). For the 2003-2011 interval, there was no significant difference in cumulative
2 toxicity between west and east (p=0.1215).

3 The duration of the toxic events also varied considerably through time (Figs. 4, 5). In Fig.
4 4, the onset and termination of toxicity are shown separately for both western and eastern Maine.
5 The time interval between the two lines for each region indicates the duration of the outbreak for
6 a given year. The calculated duration of detectable toxicity for each year is given in Figure 5.
7 Once again, considerable variability is evident between individual years, but long-term trends are
8 evident in the record. In particular, the outbreaks in the late 1970s, 1980s, and 2000s have tended
9 to be longer than those in the 1990s, some of which were notably short. It is also apparent that
10 toxicity began significantly earlier in western Maine ($t= 3.84$, d.f.=66, $p=0.0003$), with an
11 average onset date of April 28 in the west versus May 15 in the east (17 days; Table 3).

12 The average date for termination of toxicity is also significantly earlier in western Maine
13 than in the east ($t= 3.06$, d.f.=66, $p=0.003$), August 9 versus September 7 (Table 3). The average
14 duration of toxicity was 105 days in the west, and 118 days in the east; however, this difference
15 was not statistically significant ($t= 1.13$, d.f.=66, $p=0.26$).

16 3.2 *The HAB Index*

17 Each of the toxicity metrics described above provides useful information, but it is also
18 informative to combine these metrics into a single measure that categorizes the overall severity
19 of PSP toxicity in Maine in a given year. This is termed the HAB Index (Fig. 6; Table 2), which
20 reveals differences between western and eastern Maine toxicity patterns and provides a single
21 metric of regional GOM toxicity. In the east, the Index values are lower than is the case in the
22 west, particularly in the first 13 years of the record. Over that interval (1978 – 1990), the Index

1 for the east averaged 6.8 (± 2.0 SD), whereas in the west, it was 11.2 (± 2.7). Over the entire 34
2 years of record, the eastern and western Indices averaged 6.8 (± 2.6) and 8.1 (± 3.5) respectively.

3 Three model formulations were considered in the analysis of trends in the HAB Index. The
4 general form of these models is shown in Figure 7. The first was a mean-shift approach in which
5 a constant mean changes to another mean with a distinct transition point or change point. This
6 approach is consistent with an interpretation of the patterns shown in Figure 7A-C as
7 representing an era of high toxicity from the late 1970s to the early 1990s, followed by an
8 interval of consistently low toxicity until the early 2000s, and then by a new era of high toxicity.
9 In this analysis, we constrained the mean-shift model to find two change points, with their
10 locations estimated from the data as part of the fitting.

11 The results from fitting the mean-shift model to time series of the HAB Index for western
12 and eastern Maine separately and together are presented in Table 4. These regions will be
13 referred to as western Maine, eastern Maine, and western plus eastern Maine, respectively.
14 Clearly, the results for the overall region should not be viewed as independent of those for the
15 two subregions. Change-point analysis delineates three eras of toxicity in the HAB Index. The
16 estimate $\hat{\tau}_1$ of the earliest change point was 1990 for both western Maine and western plus
17 eastern Maine and 1995 for eastern Maine only. Notably, the estimate $\hat{\tau}_2$ of the second or later
18 change point was 2002 for all three datasets. In Figure 7A-C, the original time series are shown
19 along with the fitted trends from the mean-shift model.

20 An alternative view of the HAB Index patterns is that toxicity was high in the late 1970s,
21 and then decreased steadily until the mid 1990s when a change point occurred, leading to an
22 increasing trend thereafter, continuing to the present. This V-shaped scenario was modeled
23 using the two-phase model with a single change point. Results are shown in Figures 7D-F and

1 Table 5. In this case, the estimates of the single change point are very close for the two Maine
2 subregions: 1997 for western Maine and western plus eastern Maine, and 1998 for eastern
3 Maine. In Figure 7D-F, the original time series are again shown along with the fitted trends from
4 the two-phase model.

5 The third model depicts a scenario in which pulses in toxicity are linked to major cyst
6 deposition events, after which toxicity gradually declines. For all three datasets the year 2002
7 was selected as the single fixed change point estimate τ indicating the timing of an upward shift
8 in toxicity. The pre-change slope β_1 for western and eastern Maine was -0.45 and -0.13,
9 respectively, while the post-change slope γ_1 was zero for western Maine and -0.05 for eastern
10 Maine. Results are shown in Figures 7G-I and Table 6.

11 The goodness of fit for each model was measured by the residual sum of squares (*RSS*) for
12 each data set. Based on minimal *RSS* values, the mean-shift model was the best fit for eastern
13 Maine, the two-phase model the best fit for western Maine, and the pulse-decline model best fit
14 the combined dataset.

15 The mean-shift and linear two-phase modeling approaches were further compared using
16 AIC_c (Table 7). On the basis of this criterion, the mean-shift model was again selected as the
17 best fit for eastern Maine and the two-phase model for western Maine and western plus eastern
18 Maine. The difference in AIC_c between these two models is small, especially for western and
19 eastern Maine combined, indicating that the two models essentially perform equally well.

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1 **4 Discussion**

2 Interannual variability is a hallmark of many HAB events worldwide. Here we document
3 toxin variability in Maine with 34 years of data from shellfish monitoring programs. An
4 informative HAB Index has been formulated that collapses large amounts of shellfish toxicity
5 data into a single measure proposed as indicative of annual severity. This Index and other
6 observations document long-term (decadal) trends in toxicity in both sections of the Maine
7 coastline. We know of no similar formulation that can incorporate large amounts of annual
8 monitoring data at multiple stations into a single index that can reveal annual and interannual
9 trends in toxicity. The Index represents a novel and practical tool for resource managers in many
10 areas of the world subject to toxic HABs.

11 Three models for trend analysis were explored to define toxicity eras or regimes and their
12 associated change points within the HAB Index time series. In all cases, the models delineate
13 decade-level trends in toxicity, although the nature of the trends that are identified differ, as do
14 their implications. The difference between these three model formulations is evident in Figure 7,
15 but the statistical analysis suggests that none consistently gave a better fit than the others.
16 Nevertheless, the commonalities among the fitted models can now guide efforts to identify
17 physical and/or biological mechanisms underlying the patterns revealed by the HAB Index. As
18 discussed below, we hypothesize that major changes in the abundance of *A. fundyense* cysts may
19 be a primary factor underlying the decadal trends in shellfish toxicity.

20 4.1 *The Maine HAB Index*

21 In Maine, managers face the same problem as their counterparts worldwide – the need to
22 synthesize data from a large number of sampling stations over many years into a format that
23 facilitates comparative analysis. This would help to address common questions such as whether a

1 particular year is markedly different from those in the past, whether shellfish toxicity is
2 becoming more or less severe through time, whether there are trends that may be associated with
3 pollution, climate change, or other environmental factors, or whether monitoring policies should
4 be changed to reflect geographic differences or toxicity patterns.

5 One approach to this type of retrospective analysis is that of Thomas et al. (2010) who
6 used multivariate techniques to cluster monitoring stations along the GOM coast into multiple
7 station groups that behaved in a similar manner with respect to each of several metrics of
8 toxicity. Each metric yielded a different set of stations that grouped together, and each set of
9 station groups could then be analyzed against environmental parameters to look for causative
10 factors for the toxicity trends. That study provides a great deal of insight and detail, but the
11 analytical approach is complex and may be difficult to implement as a general management tool
12 in other regions. In a similar study using the same dataset, Nair et al. (2013) also divided the
13 coast into station clusters and looked for correlations with environmental and oceanographic
14 parameters. Here we took a simpler approach and divided the Maine coastline into two
15 subregions - eastern and western Maine. We then examined three measures of PSP toxicity over
16 34 years and combined them into a HAB Index (Fig. 6; Table 2) that provides a single measure
17 of annual severity of toxicity.

18 Our separation of the coast of the GOM into two regions is based on established
19 hydrographic features, including the offshore veering of the MCC near Penobscot Bay (e.g.,
20 Brooks, 1985; Lynch et al., 1997; Pettigrew et al., 2005), *Alexandrium* population distributions
21 documented during the ECOHAB – GOM program (Townsend et al., 2001; Anderson et al.,
22 2005d) as well as past patterns of shellfish toxicity in the region (e.g. Nair et al., 2013). In the
23 latter context, it has long been known that Penobscot Bay (Fig. 1) marks a boundary between a

1 region to the west that exhibits toxicity relatively early in the season compared to areas in eastern
2 Maine and the BOF (Hurst and Yentsch, 1981; Shumway et al., 1988; Bean et al., 2005;
3 Luerssen et al., 2005; Thomas et al., 2010). Both Thomas et al. (2010) and Nair et al. (2013)
4 used statistical approaches to identify multiple station clusters in the GOM that group together
5 and are generally in close geographic proximity. These workers conclude that many of the
6 factors that drive toxicity patterns in the GOM are regional with a separation near Penobscot
7 Bay, consistent with our western and eastern Maine subregions.

8 4.1.1 Individual Index metrics

9 Much can be learned from the individual metrics that comprise the HAB Index. For
10 example, toxicity typically begins 2.5 weeks earlier in the west than in the east and ends
11 approximately one month earlier (Figure 4, Table 3), such that toxicity duration is roughly the
12 same. This is consistent with McGillicuddy et al.'s (2005a) conceptual model of *Alexandrium*
13 bloom dynamics in the GOM in which warmer, early season temperatures in the west lead to
14 higher growth rates than in the east, and an earlier onset of toxicity.

15 Another general observation is that in the 1990s and early 2000s era when toxicity and thus
16 the HAB Index were low, bloom duration was short relative to other intervals (Figs. 4, 5). This
17 reflects early bloom terminations more than later onsets, as the average termination date over the
18 1990s in western Maine was July 5th, compared to September 15th for the 1978-1989 interval
19 (i.e., 60+ days later). The average dates of western Maine bloom onsets in the 1990s differed by
20 only 11 days versus the 1978 – '89 interval. The shorter blooms that occurred during the low
21 toxicity regime in the '90s thus started at nearly the same time of the year as the longer duration
22 ones of earlier and later years, but terminated earlier. This is probably because bloom initiation
23 is linked to cyst germination which is controlled by an internal clock in *A. fundyense* in the GOM

1 (Anderson and Keafer, 1987; Matrai et al., 2005). Germination onset would thus be relatively
2 invariant from year to year, leading to similar times for first detection of toxicity within a region.
3 The differences in termination dates in these toxicity regimes or eras suggests that it may be
4 possible to use the methods of Thomas et al. (2010) and Horecka et al. (this issue) to identify
5 physical mechanisms that resulted in shortened bloom duration (i.e., led to earlier termination)
6 during the 1990s or early 2000s, or conversely, lengthened them in the 1970s, 1980s, and mid to
7 late 2000s. Alternatively, earlier bloom termination may have been caused by chemical and
8 biological factors such as nutrient limitation and grazing of the *Alexandrium* population.

9 4.2 Trend analysis of the HAB Index

10 The composite HAB Indices for eastern and western Maine (Fig. 6) reveal significant
11 variability in overall toxicity between individual years. Based on visual inspection of the data –
12 two or three long-term, decadal patterns or regimes are evident in both subregions (Fig. 7). One
13 view is that the HAB Index for western Maine shows a 13-year period from 1978 to 1990 that
14 has high, mean toxicity (12 of 13 years at an Index ≥ 8), followed by a 14-year interval (1991-
15 2004) of low toxicity (14 of 14 Index values between 3 and 7), and thereafter high values of
16 toxicity from 2005 to the present (6 of 7 Index values ≥ 8). Similar trends are found in the eastern
17 Maine HAB Index. Another view is that of a V-shaped pattern in which high toxicity in the late
18 1970s and early 1980s decreases steadily to a low in the mid 1990s, and then increases thereafter.
19 Yet another view of the time series would be episodic pulses of high toxicity followed by a
20 gradual decline.

21 These interpretations were explored in detail through trend analysis to determine whether
22 the HAB Index time series best fitted a mean-shift model in which three eras of toxicity are
23 observed (high \rightarrow low \rightarrow high toxicity), a two-phase linear model (decreasing \rightarrow increasing

1 toxicity), or a third, pulse-decline model (high → declining → high → declining toxicity). Based
2 on the RSS, the mean-shift model was a better fit for eastern Maine, the two-phase model for
3 western Maine, and the pulse-decline model for western plus eastern Maine combined, though
4 the difference between the models is small (i.e., differed by < 20%). Any of the three models
5 could thus be used to characterize the time-series.

6 The underlying mechanisms and implications of the three model formulations, however,
7 are different. In the mean-shift model, major transitions from one toxicity era to another occur
8 over relatively short intervals (Fig. 7A-C, Table 4). The rapid decline in toxicity followed by an
9 interval of sustained, low levels is analogous perhaps to a disastrous year class of fish that affects
10 harvest yield for many years thereafter. It may thus be informative to examine environmental
11 data for the years around the time the mean shifts occurred, as this might identify important
12 environmental conditions that can either dramatically reduce *A. fundyense* abundance and
13 associated shellfish toxicity for an extended interval thereafter, or, conversely, initiate a new,
14 higher toxicity era. The years that might be worth scrutinizing in this regard would be 1990-1993
15 on the downside, and 2002-2004 on the upside (Figs. 6, 7). The HAB Index makes identification
16 of these critical time intervals relatively easy. Below we hypothesize that these transitions were
17 linked to major changes in cyst abundance.

18 Conversely, with the two-phase model, the decline in toxicity from the late 1970s to the
19 mid 1990s would be consistent with a gradual change in *A. fundyense* population size in
20 nearshore waters, which could arise from a variety of factors (declining cyst abundance (see
21 below), changing nutrient availability, other biological factors such as grazing), or it might
22 reflect systematic changes in the physical factors that regulate the delivery of offshore blooms to
23 shore. An analysis of the environmental factors that might correlate with this declining interval,

1 or with the increasing toxicity era that followed would require the multivariate approach of
2 Thomas et al. (2010), Nair et al. (2013), and Horecka et al. (this issue), and is beyond the scope
3 of this study.

4 The pulse-decline model could be explained by a major pulse of cyst deposition and
5 resulting high toxicity (McGillicuddy et al., 2011) that is followed by years in which that
6 inventory gradually declines, with a corresponding decline in toxicity. In this instance, the first
7 pulse probably occurred near 1972, the time of a major red tide and PSP outbreak in the region
8 (Hurst, 1975; Mulligan, 1975). Based on the model analysis, the second pulse occurred in the
9 2002 – 2004 time frame. Since then, there are too few years to reveal a declining trend in
10 western Maine, but a modest downward trend is evident for eastern Maine and the combined
11 region. More detail on the blooms for both of these pulse intervals are given in Section 4.5
12 below.

13 Examining the years preceding the 34-year dataset, another era (with low or undetectable
14 toxicity for at least 13 years from 1958 to 1971) can be postulated based on the general lack of
15 toxicity in that portion of coastal Maine over that interval. In eastern Maine, that time was
16 associated with geographically limited and infrequent toxicity. Specifically, Hurst (1975) points
17 out that the Maine biotoxin monitoring program began in 1958 after a PSP outbreak in New
18 Brunswick, Canada, but was focused only on eastern Maine. Closures due to PSP in eastern
19 Maine occurred in only 5 years out of 14 between 1958 and 1970 (Shumway et al., 1988).
20 Thereafter, testing was added in western portions of the state. In 1961, toxin was detected along
21 the entire Maine coast (Hurst, 1975), but all stations in western Maine, except those at two
22 offshore islands, had toxin levels below quarantine thresholds. Thereafter, there were no closures
23 in western Maine until 1972, when a visible *A. fundyense* red tide occurred, affecting the entire

1 Maine coast, as well as waters in New Hampshire and Massachusetts (Mulligan, 1975; Hartwell,
2 1975). The large scale of the 1972 outbreak led to an expanded monitoring program that includes
3 160 stations (Bean et al., 2005). Closures are now frequent in western Maine – occurring in over
4 80% of the 34 years studied here (Kleindinst et al., this issue). We believe it is important to
5 review and document these early toxicity data in Maine, as they support the view that in the 20
6 years before the HAB Index calculations began, toxicity was low in both eastern and western
7 Maine, marking another low toxicity era similar to the one observed in the 1990s.

8 4.3 *Upstream and downstream patterns*

9

10 Given the linkage between *A. fundyense* blooms in the BOF and those in eastern Maine
11 (Aretxabaleta et al., 2008; 2009), and the linkages between blooms in western Maine and those
12 in New Hampshire and Massachusetts Bay (Anderson, 1997), one wonders whether the patterns
13 indicated by the Maine HAB Index are reflected in toxicity patterns in these upstream and
14 downstream areas. Unfortunately, the data needed to calculate the HAB Index are not currently
15 available for Massachusetts or the BOF, but other measures of toxicity can be examined in this
16 context.

17 Toxicity in Massachusetts and southern New England waters is not initiated from
18 localized cyst germination (*A. fundyense* cyst abundances are low in that region; Anderson et al.,
19 2005c. this issue), but rather from the advection of established vegetative populations from
20 western Maine via the MCC (Anderson, 1997; Stock et al., 2005; Stock et al., 2007). It therefore
21 is not surprising that the PSP toxicity observed in Massachusetts Bay shows a pattern with
22 relatively frequent and high toxicity in the 1970s, '80s, and early '90s, but no toxicity from 1994
23 through 2004 (data not shown). Thereafter, toxicity in this region has been frequent but variable

1 in magnitude (McGillicuddy et al., 2011). These patterns are generally consistent with changes
2 in the western Maine HAB Index (Fig. 7A,D,G).

3 In the upstream region, the situation is more complex, as the *A. fundyense* blooms are
4 strongly influenced by a retentive gyre at the mouth of the BOF (Martin et al., this issue) and the
5 extent to which cells in the BOF escape into the eastern GOM (Aretxabaleta et al., 2008; 2009).
6 When the annual mean toxicity at multiple stations in the BOF was compared with the eastern
7 Maine HAB Index, no significant correlation was observed. This lack of correlation may reflect
8 the nature of interannual variability in the “leakiness” of the BOF, which Aretxabaleta et al.
9 (2009) argue depends primarily on the strength of the BOF gyre, which is in turn influenced by
10 stratification within the Grand Manan basin. Many other factors modulate the leakiness on a
11 variety of time scales (Aretxabaleta et al., 2008), including the residual tidal circulation, frontal
12 retention during stratified periods, wind stress, and interactions with the adjacent circulation of
13 the GOM. These forcings would all modulate the delivery of cells into eastern Maine, and may
14 thereby influence interannual variability in toxicity in downstream areas.

15 4.4 *Underlying mechanisms*

16 Although the dataset examined in the Thomas et al. (2010) study was shorter than the 34
17 years (1978 – 2011) that we utilized, these authors described what was, in effect, a mean-shift
18 pattern in toxicity. That study tried to correlate toxicity patterns with interannual environmental
19 variables (e.g., temperature, river discharge, salinity, wind stress), but only western Maine
20 stations showed correlations with any tested environmental metric. There, positive correlations
21 were observed between toxicity and early season wind stress driving onshore Ekman transport;
22 negative correlations were found with wind stress driving offshore transport, and with summer
23 cross-shelf surface temperature gradients. Similar conclusions were drawn from the analysis of
24

1 the same dataset by Nair et al. (2013). The correlations between toxicity and wind direction
2 stems from the tendency of downwelling-favorable winds to bring toxic *Alexandrium* cells to
3 shore, and upwelling-favorable winds to move them offshore and away from shellfish have been
4 noted in multiple other studies (e.g., Franks and Anderson 1992a,b; McGillicuddy et al., 2003;
5 Anderson et al., 2005a). Such relationships have also been noted for the western Irish coast by
6 Raine et al. (2010).

7 Thomas et al. (2010) reported a temporal autocorrelation in their data, suggesting that
8 processes with time scales longer than a year are important in controlling toxicity in the GOM.
9 Our study suggests that this factor may be cyst abundance. This hypothesis is based on the
10 significant and positive relationship we have documented between fall *A. fundyense* cyst
11 abundance and bloom magnitude the following year (McGillicuddy et al., 2011; Anderson et al.,
12 this issue). Although our cyst survey data do not span the same interval as the 34 years of
13 toxicity records, it is nevertheless possible to compare cyst abundance and the HAB Index for the
14 years that are in common to determine if a relationship exists between the two. The first large-
15 scale cyst survey for the GOM conducted in 1997 (Anderson et al., 2005c) yielded very low cyst
16 abundance compared to eight subsequent surveys from 2004 - 2011 (Anderson et al., this issue).
17 When scaled to represent a common cyst-sampling domain among years, the 1997 cyst
18 abundance was 1/11th of what it was in 2009, for example. This low abundance occurred within
19 the decade when the HAB Index indicates there was low-level toxicity in both the eastern and
20 western subregions (Fig. 6). The significantly higher cyst abundances recorded from 2004 –
21 2011 (Anderson et al., this issue) coincide with the era of renewed high toxicity.

22 Unfortunately, very limited quantitative cyst data are available in the GOM prior to the
23 1997 survey, although two surveys provide insights. Anderson and Keafer (1985) measured *A.*

1 *fundyense* cyst abundances in the WGOM in 1983 and 1984. The northernmost stations of that
2 sampling effort (Massachusetts Bay to Portsmouth, NH) were located in the same general area as
3 the westernmost stations sampled during the 1997 large-scale survey. Counts from 1983 and
4 1984 were markedly (>10X) higher than those at similar locations in that 1997 survey. For
5 example, the mean cyst concentrations from 6 stations offshore of Portsmouth, New Hampshire
6 were 664 cysts cm⁻³ in 1983 (0-2 cm) and 711 cysts cm⁻³ in 1984 (0-2 cm) vs. < 50 cysts cm⁻³
7 observed in the 0-1 cm layer from that same area during the large-scale survey in 1997.
8 Furthermore, when the 0-1 cm layer was repeatedly sampled at a single station in that area
9 (station 29; latitude 43.00N longitude 70.31W) over a 4-year period that spanned 1984 to 1987,
10 the *A. fundyense* cyst counts ranged from a minimum of 318 cysts cm⁻³ to a maximum of 992
11 cysts cm⁻³ with a mean of 584 cysts cm⁻³ observed (n=14), more than a order of magnitude
12 higher than any cyst concentration observed near Station 29 in 1997.

13 Although these 1983, 1984 data are obviously limited in scale, we note that an ongoing
14 statistical analysis of *A. fundyense* cyst abundance patterns from nine years of large-scale surveys
15 in the GOM demonstrates that there is a generic cyst distribution pattern in the region, with cyst
16 abundance at individual stations varying in a consistent pattern from year to year relative to the
17 surrounding stations (A. Solow, unpub. data). If that same spatial pattern were present during
18 the time of the high relative cyst abundance measurements in 1983 and 1984, which seems
19 likely, those values would be representative of the broader regional cyst distribution.

20 We thus hypothesize that the decade-level trends in regional toxicity evident in the HAB
21 Index (Figs. 6, 7) correspond to major differences in cyst abundance over that same interval. The
22 data in support of this hypothesis are limited, but can be further tested in the future.

1 4.5 *Conceptual model of historical toxicity patterns*

2 Figure 8 provides a conceptual model of the historical toxicity patterns and their
3 relationship with both measured and hypothesized cyst abundance and distribution in the region.
4 We acknowledge that portions of this model are speculative, as they depend on either postulated
5 cyst abundance before the times of major cyst surveys, or limited cyst survey data, as for 1983
6 and 1984. Nevertheless, we feel it is worthwhile to advance a long-term conceptual model that
7 updates our view of the bloom and toxin dynamics of this GOM system, and that can guide
8 future studies.

9 We argue that the first toxicity era or regime is the “pre-1972” interval in which toxicity
10 was high and recurrent in the BOF (as it has been for hundreds of years (Medcof et al., 1947;
11 Needler, 1949; Prakash et al., 1971)), sporadic and infrequent in eastern Maine, and essentially
12 non-existent in western Maine, New Hampshire, or Massachusetts (Hurst, 1975; Shumway et al.,
13 1988). No cyst surveys were conducted in that interval (in fact the cyst of *A. fundyense* had not
14 yet been discovered; Dale, 1977; Anderson and Wall, 1978), but we hypothesize that the BOF
15 cyst seedbed was much as it is today, and that the mid-coast Maine seedbed was sparse or non-
16 existent. This supposition can perhaps be tested someday using the fossil cyst record.

17 The contrast in toxicity between the years before and after the 1972 GOM red tide is
18 remarkable. With no closures in the west, and only sporadic closures in the east prior to that year
19 (Hurst, 1975; Shumway et al., 1988), *A. fundyense* blooms have since become an annual
20 phenomenon along the entire coast, including western Maine. Several studies indicate that the
21 1972 event deposited *A. fundyense* cysts in high abundance in the western gulf region, creating
22 what we now refer to as the mid-coast Maine seedbed, which was sustained through time with
23 the recurrent *A. fundyense* blooms of the 1970s and ‘80s (e.g., Mulligan, 1975; Anderson, 1997).

1 The only quantitative cyst abundance surveys during that interval were from the 1983 and 1984
2 small-scale surveys in the WGOM (Anderson and Keafer, 1985), which documented high *A.*
3 *fundyense* cyst abundances. The corresponding toxicity pattern for that era was one of continued
4 PSP outbreaks in the BOF, and frequent and high toxicity events in eastern and western Maine,
5 as well as in downstream New Hampshire and Massachusetts. Subsequently, we theorize that
6 there was either a gradual decline in cyst abundance over a decade or more during this interval
7 (the two-phase and pulse-decline models), or a precipitous drop (mean-shift model) occurring
8 around 1990. We believe this low toxicity and low cyst abundance interval persisted until the
9 early 2000s, consistent with the low cyst concentrations in the 1997 large-scale cyst survey
10 (Anderson et al., 2005c).

11 For reasons that are not well understood (Anderson et al., this issue), cyst abundance
12 increased greatly between the 1997 and the 2004 surveys. This may relate to unusually high *A.*
13 *fundyense* cell concentrations observed in both the BOF and the EGOM in the fall of 2003. In
14 the BOF, cell densities were the highest observed in more than two decades at Wolves Island
15 (Martin et al., this issue). Also, high cyst abundance in 2004 was the main explanation for a
16 major regional *A. fundyense* bloom in 2005 (He et al., 2008), extending from eastern Maine to
17 southern Massachusetts and its offshore islands (Anderson et al., 2005b). Those high levels of
18 cyst abundance have persisted to this writing, fluctuating 2-5-fold among years, but never falling
19 to the low level reported in 1997 (Anderson et al., this issue). Toxicity has generally been high
20 but variable, as documented in the western and eastern Maine HAB Indices (Figs. 6, 7). The
21 new regime of high toxicity that began in 2002 - 2004 may be similar to that experienced after
22 the 1972 red tide, which was followed by 17 years of frequent and high toxicity events (Fig. 6),
23 possibly in a gradually decreasing trend. If history is giving us a view of the future, the GOM

1 may have entered a new regime of high cyst abundance and high or gradually declining shellfish
2 toxicity that could last for a decade or more.

3 **Acknowledgements**

4 This paper is dedicated to John Hurst, who established and ran the Maine Biotoxin
5 monitoring program for over 40 years. His insights and wisdom on the regional *A. fundyense*
6 blooms are legendary, built on years of keen observations and inquiry. We can only hope that the
7 concepts presented here meet with his approval, though he undoubtedly would repeat a phrase
8 we heard often: “It sure takes you scientists a long time to answer a simple question.” We
9 gratefully acknowledge the thoughtful comments of several anonymous reviewers who greatly
10 improved this manuscript.

11 Research support provided through the Woods Hole Center for Oceans and Human
12 Health, National Science Foundation (NSF) Grants OCE- 1128041 and OCE-1314642; and
13 National Institute of Environmental Health Sciences (NIEHS) Grant 1-P50-ES021923-01, the
14 ECOHAB Grant program through NOAA Grants NA06NOS4780245 and NA09NOS4780193,
15 the MERHAB Grant program through NOAA Grant NA11NOS4780025, the PCMHAB Grant
16 program through NOAA Grant NA11NOS4780023, and funding through the states of ME, NH,
17 and MA. Funding for J.L. Martin was provided by Fisheries and Oceans Canada. This is
18 ECOHAB contribution #735; MERHAB contribution #168; PCMHAB contribution #7.

19

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1

2 **Table 1. Metrics of the Maine HAB Index (see Methods sec. 2.3).**

3

Bin #	% of toxic stations	Cumulative toxicity per station ($\mu\text{g STX eq. } 100\text{g}^{-1}$ shellfish meat)	Duration of toxicity (days)
1	0-20	0-500	0-50
2	21-40	501-1,000	51-100
3	41-60	1,001-1,500	101-150
4	61-80	1,501-2,000	151-200
5	81-100	>2,000	>200

4

5

Table 2. The Maine HAB Index and constituent metrics of toxicity.

Year	% of Toxic Stations (West)	% of Toxic Stations (East)	Cumulative toxicity per station (West)	Cumulative toxicity per station (East)	Duration (West)	Duration (East)	Total Index (West)	Total Index (East)
1978	102	43	4116	399	206	94	15	6
1979	91	13	1889	118	162	97	13	4
1980	91	64	4134	1096	206	262	15	12
1981	92	26	2081	397	143	198	13	7
1982	68	36	755	343	149	135	9	6
1983	69	29	1105	355	126	140	10	6
1984	83	41	1752	401	106	114	12	7
1985	31	30	273	416	112	127	6	6
1986	92	31	5757	1015	134	195	13	9
1987	63	20	804	130	177	150	10	5
1988	73	33	823	192	96	114	8	6
1989	89	46	1769	778	130	136	12	8
1990	77	30	573	374	106	134	9	6
1991	52	27	160	182	92	65	6	5
1992	30	30	48	126	70	134	5	6
1993	59	33	533	723	72	134	7	7
1994	34	29	59	144	37	58	4	5
1995	40	31	123	509	59	93	5	6
1996	10	11	7	25	63	79	4	4
1997	20	9	26	7	24	22	3	3
1998	24	20	86	29	51	50	5	3
1999	6	10	4	20	24	58	3	4
2000	59	4	433	6	52	56	6	4
2001	9	47	9	322	72	84	4	6
2002	22	21	35	54	87	92	5	5
2003	36	63	96	980	164	123	7	9
2004	53	77	256	2112	148	156	7	13
2005	72	54	1374	527	114	119	10	8
2006	89	50	529	230	96	103	9	7
2007	68	43	158	264	107	104	8	7
2008	71	87	412	1470	77	142	7	11
2009	100	94	1946	3379	140	141	12	13
2010	32	66	65	869	92	171	5	10
2011	72	57	629	450	77	134	8	7

Table 3. Average onset and termination dates (\pm SD) and duration of PSP toxicity in eastern and western Maine

Parameter	Date or duration
West onset	April 28 (\pm 17)
West termination	August 9 (\pm 42)
West duration (days)	105 (\pm 47)
East onset	May 15 (\pm 27)
East termination	September 7 (\pm 30)
East duration (days)	118 (\pm 48)

Table 4. Estimated parameters for mean-shift model fitted to the HAB Index time series data: mean for each interval ($\hat{\mu}$), change-point years ($\hat{\tau}_1, \hat{\tau}_2$), and residual sum of squares (RSS).

Region	$\hat{\mu}(1)$	$\hat{\tau}_1$	$\hat{\mu}(2)$	$\hat{\tau}_2$	$\hat{\mu}(3)$	<i>RSS</i>
Western Maine	11.2	1990	4.75	2002	8.1	138.8
Eastern Maine	6.5	1995	4.14	2002	9.4	109.6
Western + Eastern Maine	17.9	1990	9.58	2002	17.6	336.1

Table 5. Estimated parameters for two-phase model fitted to the HAB Index time series data: trend parameters $\hat{\beta}_o, \hat{\beta}_1, \hat{\beta}_2$, change-point year $\hat{\tau}$, and residual sum of squares *RSS*.

Region	$\hat{\beta}_o$	$\hat{\beta}_1$	τ	$\hat{\beta}_2$	<i>RSS</i>
Western Maine	14.7	-0.54	1997	0.94	121.5
Eastern Maine	7.7	-0.15	1998	0.63	135.3
Western + Eastern Maine	22.6	-0.71	1997	1.54	363.2

Table 6. Estimated parameters for pulse-decline model fitted to the HAB Index time series data: trend parameters $\hat{\beta}_0, \hat{\beta}_1, \hat{\beta}_2$, $\hat{\gamma}_0, \hat{\gamma}_1$, change-point year $\hat{\tau}$, and residual sum of squares RSS .

Region	$\hat{\beta}_0$	$\hat{\beta}_1$	$\hat{\tau}$	$\hat{\gamma}_0$	$\hat{\gamma}_1$	RSS
Western Maine	14.0	-0.45	2002	8.1	0	127.0
Eastern Maine	7.6	-0.13	2002	9.6	-0.05	114.4
Western + Eastern Maine	21.5	-0.06	2002	17.8	-0.05	322.1

Table 7. AIC_c values calculated for the mean-shift and two-phase models for each dataset.

Region	AIC_c for mean- shift model	AIC_c for two- phase model
Western Maine	63.1	55.3
Eastern Maine	54.9	56.9
Western + Eastern Maine	93.0	92.7

Figure legends

Figure 1. Conceptual model map of *Alexandrium fundyense* regional blooms in the Gulf of Maine. Areas enclosed with dashed lines denote cyst seedbeds that provide inoculum cells. Major current systems are shown with solid arrows (black = Maine Coastal Current (MCC); grey = Gulf of Maine Coastal Plume (GMCP; Keafer et al., 2005). Possible *A. fundyense* transport pathways (separate from the major current systems) are shown with dashed red arrows. Red-shaded areas represent regions of growth and transport of motile *A. fundyense* cells. Modified from Anderson et al. (2005c). The dividing line between WGOM and EGOM is shown at Penobscot Bay, as described in the text. Abbreviations: MCC –Maine Coastal Current; GMCP – Gulf of Maine Coastal Plume; MB – Massachusetts Bay; BOF – Bay of Fundy; MA – Massachusetts; NH – New Hampshire; ME – Maine; NS – Nova Scotia.

Figure 2. Percent of stations on the Maine coast with detectable levels of PSP mussel toxicity. Eastern Maine (open circles); western Maine (closed circles).

Figure 3. Cumulative toxicity per station measured in *Mytilus edulis* in western Maine (closed circles) and eastern Maine (open circles) for each year.

Figure 4. Onset and termination dates for toxicity detection in Maine –western Maine (A), eastern Maine (B).

Figure 5. Duration of detectable toxicity for western (closed circles) and eastern (open circles) Maine.

Figure 6. HAB Indices for western Maine (A), eastern Maine (B), and combined (C).

Figure 7. Mean-shift (A-C), two-phase (D-F), and pulse-decline (G-I) models fitted to time series of the HAB Index for eastern and western Gulf of Maine separately, and together.

Figure 8. Conceptual model of historical toxicity patterns and their relationship with measured and hypothesized cyst abundance and distribution in the Gulf of Maine region. The shapes used for the cyst seedbeds are not intended to be accurate but only represent general features. Note that the size of the cyst beds in the first two panels is based on inference rather than direct measurement because large-scale cyst surveys were not conducted in those years. Nevertheless, the distribution depicted is inferred from toxicity data and other observations.

Supplementary Materials – Maine toxicity – noteworthy details and commentary. Toxicity in $\mu\text{g STX } 100\text{g}^{-1}$.

Year	Notes	West		East	
		Onset	End	Onset	End
1977	<p>1977 is not an ideal year to use in the HAB Index, for several reasons:</p> <ol style="list-style-type: none"> 1) The western Maine “onset” was triggered by a relatively isolated bloom initiated at Lumbos Hole, which did not appear to spread and maintain intensity in the rest of the western Maine coast until a massive spike appeared at Monhegan Is. On Oct 7, 1977; the bulk of the “Biomass Score” for western Maine in 1977 occurred from Oct 7 to Nov 2, which makes the “Duration” metric appear artificially long, at 196 days, when the majority of the toxin load was delivered in only 27 days. 2) There seems to have been an irregular sample strategy in eastern Maine in 1977, in that no <i>Mytilus</i> samples were taken in eastern Maine until Sep 28, while <i>Mya</i> samples were taken consistently for most of the summer, showing toxin in that species as early as Jul 19. This produces artificially lowered metrics for all three categories, since only <i>Mytilus</i> is used to calculate them. <p>The general characteristics of the 1977 HAB event in Maine could be summarized as beginning in late summer in eastern Maine, and hitting an unusual spike in western Maine in the fall. Despite the “official” onset of low-level, isolated toxicity in April, it would be reasonable to consider 1977 as a “fall bloom” year for western Maine. Heaviest impacts in western Maine were limited to Monhegan Island, tips and outer areas of peninsulas, and along the southern Maine coast. No evidence of intrusion or significant residence time in far-inland areas.</p> <p>Residual toxin detected in other molluscs (snails and surf clams) sampled in January of 1977 suggests that there was a late bloom and/or high toxin levels at the</p>	Apr 21	Nov 2	Sep 26	Nov 5

Year	Notes	West		East	
	end of the 1976 season. Note, however, that detoxification rates are very slow in surf clams, and thus making interpretations of bloom duration difficult.				
		<i>Onset</i>	<i>End</i>	<i>Onset</i>	<i>End</i>
1978	<p>Eastern Maine patterns were a little unusual, with <i>Mytilus</i> toxicity showing up early for the area (May), and especially high levels showing up in the coastal area just east of MDI, around the Schoodic peninsula, with levels there far surpassing any in Cobscook Bay at any point in the season, which is unusual. The high scores for the Cobscook Bay area did not appear until much later in the summer (late July), which is a more typical seasonal pattern for the area.</p> <p>Scores at Monhegan Island reached extremely high levels (>11,000) in late June. The rest of western Maine experienced a severe bloom in 1978, with scores reaching extremely high levels and intruding far up into inland areas during that same period of time (late June).</p> <p>There was an unexplained spike at the Boothbay Harbor lab float station on May 5 (5,162), which does not appear to track with subsequent samples from that station or from the surrounding area. Other causes should be considered for that score (e.g. perhaps the Culture Collection had an accidental discharge, or other experiments at the DMR wet lab were impacting the area).</p> <p>Residual toxicity measured in January of 1978 in <i>Mya</i> in eastern Maine and <i>Modiolus</i> in western Maine suggests late input from the 1977 bloom season.</p>	Mar 3	Sep 24	May 16	Aug 17
		<i>Onset</i>	<i>End</i>	<i>Onset</i>	<i>End</i>
1979	<p>Nothing noteworthy about season or patterns; Monhegan Island did not get any toxicity greater than 108 µg.</p> <p>Residual toxin detected in other molluscs (snails and surf clams) sampled in January of 1979 suggests that there was a late bloom and/or high toxin levels at the end of the 1978 season. Note, however, that</p>	Apr 18	Sep 26	May 4	Aug 8

Year	Notes	West		East	
	detoxification rates are very slow in surf clams, and thus making interpretations of bloom duration difficult.				
		<i>Onset</i>	<i>End</i>	<i>Onset</i>	<i>End</i>
1980	<p>Although toxicity was still measured until December at Pemaquid Point, this area was exposed to the most extreme toxicity along the entire coast, and the residual toxicity in December does not likely represent a vegetative input. The date of November 13 is being used to denote the end of the bloom input, because the Head Beach station showed a >60% reduction in toxicity on that date, with only residual levels persisting after that date until mid-December.</p> <p>After a fairly “normal” start to the season, with low to average scores showing up in late April and May, western Maine had a stunning spike in toxicity scores around the Pemaquid peninsula and the surrounding areas in late August and early September of 1980 (high scores >10,000 µg).</p> <p>Eastern Maine showed toxicity in <i>Mytilus</i> in a January 1980 sample in Cobscook Bay, which represents some sort of low-level toxicity input during the winter or early spring in that region; residual toxicity from 1979 is unlikely, since that station and others around it were tested in August 1979 as toxin-free. Additionally, low-level toxicity is consistent in early spring in this area of eastern Maine, therefore the “onset” date of toxicity is being accepted as January 29, 1980. Eastern Maine also exhibited a fall toxicity spike in early September. A “fall bloom” was present for both regions of the state in 1980.</p>	Apr 22	Nov 13	Jan 29	Oct 17
		<i>Onset</i>	<i>End</i>	<i>Onset</i>	<i>End</i>
1981	Toxicity measured in January and February at the western Maine stations of Head Beach and Pemaquid Point are capturing residual toxicity from 1980, since these stations remained toxic in testing through December of 1980. Because of this, the “onset” date for western Maine in 1981 is adjusted to April 14, when toxicity first appeared at the Cundy’s Harbor station.	Apr 14	Sep 3	Mar 8	Sep 21

Year	Notes	West		East	
	<p>Toxicity was measured in <i>Mya</i> from eastern Maine stations in January 1981, which was present at the end of 1980. In addition, toxicity continued to persist in <i>Mya</i> samples in eastern Maine through December of 1981.</p> <p>Extremely high residual toxin (>3,000) in surf clams from western Maine tested in January 1981 indicates impacts from a late bloom and/or high toxicity event in 1980. Note, however, that detoxification rates are very slow in surf clams, and thus making interpretations of bloom duration difficult.</p>				
		<i>Onset</i>	<i>End</i>	<i>Onset</i>	<i>End</i>
1982	<p>There is an unexplained high toxin score at the Widgeon Cove station in Harpswell (western Maine) of 1,620 µg on March 17, 1982. Since there are no other toxin scores from any other areas in western Maine at that time, this data point is not considered in the 1982 HAB Index data set.</p> <p>Toxin measured in <i>Mya</i> samples from several stations around Cobscook Bay in January and February of 1982 are showing residual toxicity from late 1981.</p> <p>Residual toxin (>600) in surf clams from western Maine tested in January 1982 indicates impacts from a late bloom and/or high toxicity event in 1981.</p>	May 4	Sep 29	May 17	Sep 28
		<i>Onset</i>	<i>End</i>	<i>Onset</i>	<i>End</i>
1983		Apr 21	Aug 24	May 3	Sep 19
		<i>Onset</i>	<i>End</i>	<i>Onset</i>	<i>End</i>
1984		Apr 19	Aug 2	May 7	Aug 28
		<i>Onset</i>	<i>End</i>	<i>Onset</i>	<i>End</i>
1985		May 21	Sep 9	May 21	Sep 24
		<i>Onset</i>	<i>End</i>	<i>Onset</i>	<i>End</i>

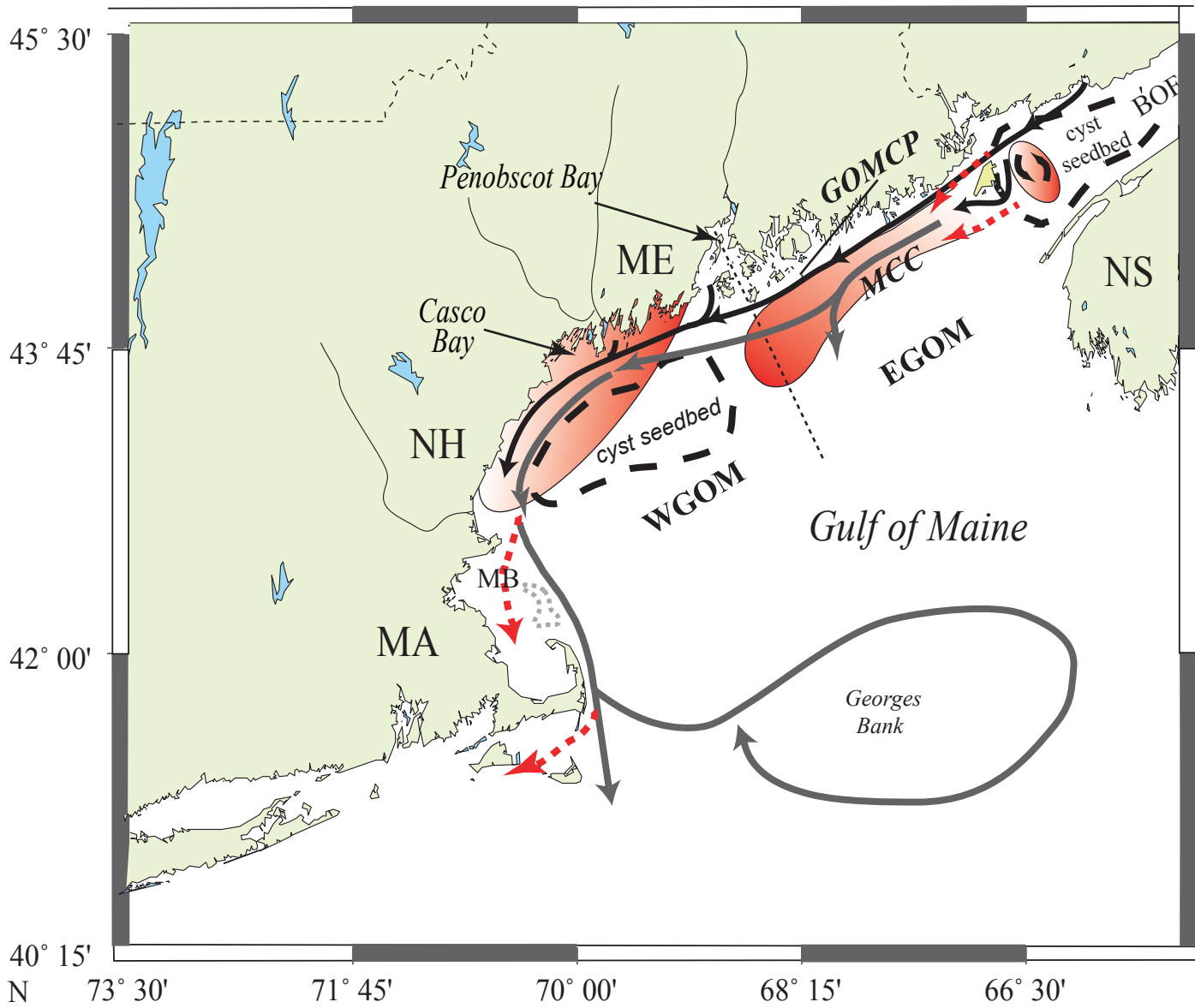
Year	Notes	West		East	
1986	There is an unexplained high toxin score at the Ewin Narrows station in Harpswell (western Maine) of 203 µg on February 8, 1986. Since there are no other toxin scores from any other areas in western Maine at that time, and subsequent samples at that station in the following weeks are negative, this data point is not considered in the 1986 HAB Index data set.	Apr 14	Sep 25	Mar 13	Sep 23
		<i>Onset</i>	<i>End</i>	<i>Onset</i>	<i>End</i>
1987	Although the duration of this event looks “normal”, the bulk of the toxicity spikes in western Maine occurred in late August and early September 1987, and it would be reasonable to categorize this year as a fall bloom for western Maine.	Apr 13	Oct 6	May 6	Oct 2
		<i>Onset</i>	<i>End</i>	<i>Onset</i>	<i>End</i>
1988		May 2	Aug 5	May 1	Aug 22
		<i>Onset</i>	<i>End</i>	<i>Onset</i>	<i>End</i>
1989		May 15	Sep 21	May 17	Sep 29
		<i>Onset</i>	<i>End</i>	<i>Onset</i>	<i>End</i>
1990		Apr 10	Jul 24	May 14	Sep 24
		<i>Onset</i>	<i>End</i>	<i>Onset</i>	<i>End</i>
1991		Apr 16	Jul 16	Jun 10	Aug 13
		<i>Onset</i>	<i>End</i>	<i>Onset</i>	<i>End</i>
1992		May 1	Jul 9	May 19	Sep 29
		<i>Onset</i>	<i>End</i>	<i>Onset</i>	<i>End</i>
1993		May 3	Jul 13	May 17	Sep 27
		<i>Onset</i>	<i>End</i>	<i>Onset</i>	<i>End</i>
1994		May 23	Jun 28	Jun 6	Aug 2

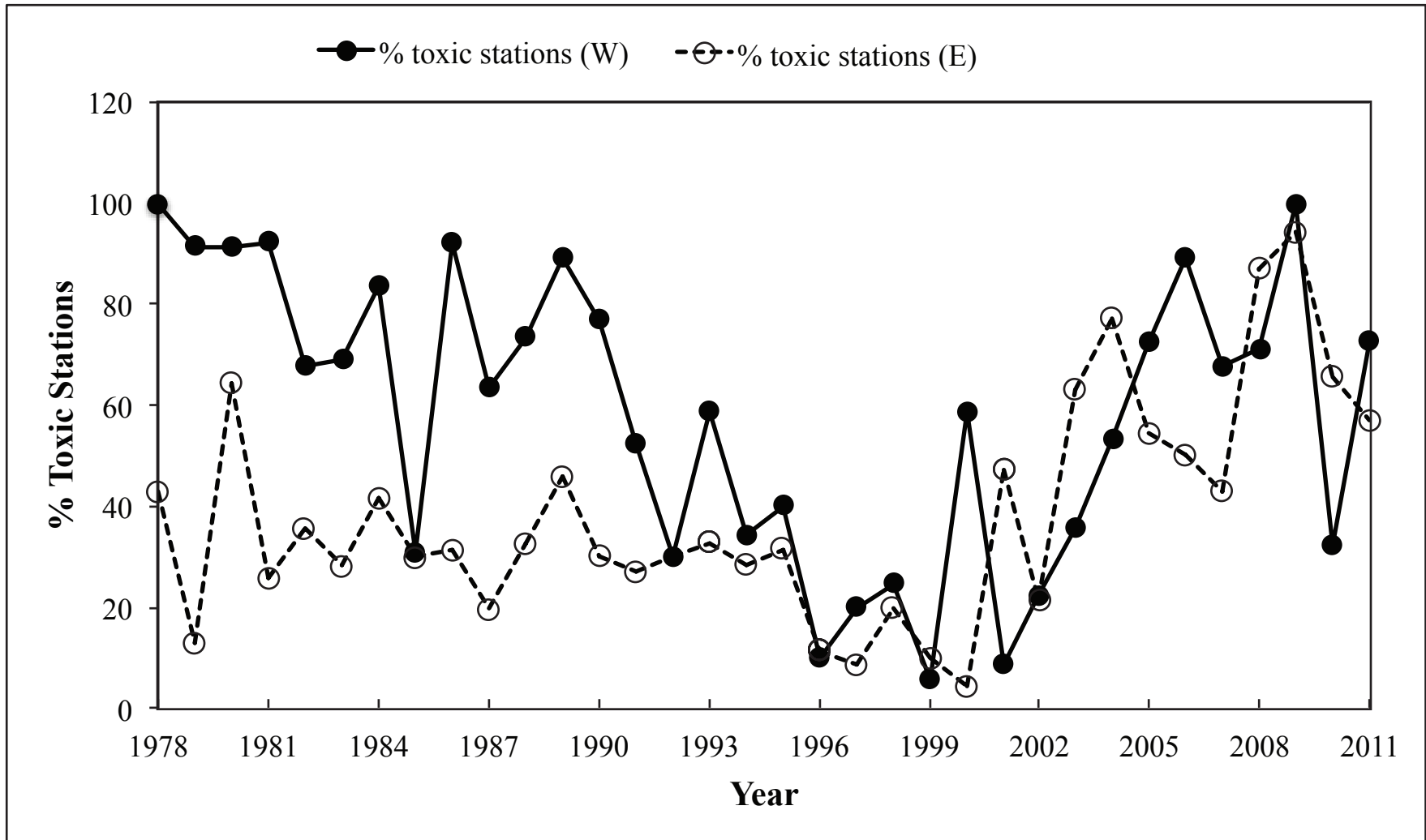
Year	Notes	West		East	
		<i>Onset</i>	<i>End</i>	<i>Onset</i>	<i>End</i>
1995		May 2	Jun 29	May 15	Aug 15
		<i>Onset</i>	<i>End</i>	<i>Onset</i>	<i>End</i>
1996		Apr 25	Jun 26	Jun 17	Sep 3
		<i>Onset</i>	<i>End</i>	<i>Onset</i>	<i>End</i>
1997		May 27	Jun 19	Jun 24	Jul 14
		<i>Onset</i>	<i>End</i>	<i>Onset</i>	<i>End</i>
1998		May 11	Jun 30	May 25	Jul 13
		<i>Onset</i>	<i>End</i>	<i>Onset</i>	<i>End</i>
1999		May 10	Jun 2	Jun 7	Aug 3
		<i>Onset</i>	<i>End</i>	<i>Onset</i>	<i>End</i>
2000	Unusual year due to no toxin measured at all in Cobscook Bay/far eastern Maine; what happened in Canada that year? Low impact from the BOF?	May 1	Jun 21	Jun 6	Jul 31
		<i>Onset</i>	<i>End</i>	<i>Onset</i>	<i>End</i>
2001		May 24	Aug 3	May 30	Aug 21
		<i>Onset</i>	<i>End</i>	<i>Onset</i>	<i>End</i>
2002	No changes or comments.	Apr 23	Jul 18	May 13	Aug 12
		<i>Onset</i>	<i>End</i>	<i>Onset</i>	<i>End</i>
2003	A mild year in western Maine, with “normal” season and spatial patterns, but eastern Maine had a late start, with the bulk of their toxicity appearing in the fall. This was a fall bloom year for eastern Maine. Also interesting to note that <i>Arctica</i> samples were taken in eastern Maine all season, and showed no detectable toxicity until mid-September, when scores jumped from negative to >2,000. Indicates a strong downwelling event in eastern Maine in the fall.	May 11	Oct 21	Jul 7	Nov 6

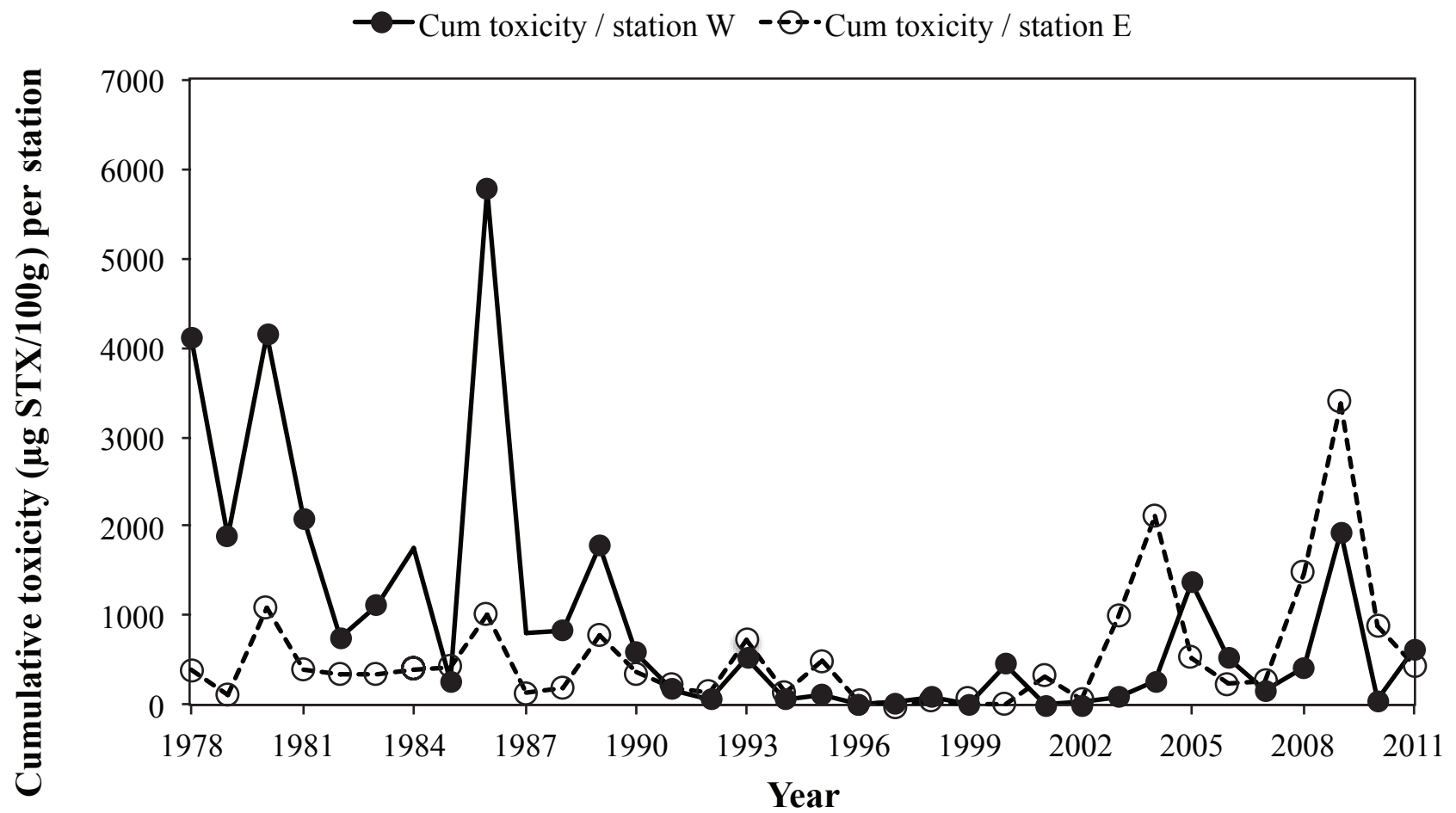
Year	Notes	West		East	
		<i>Onset</i>	<i>End</i>	<i>Onset</i>	<i>End</i>
2004	Largest toxicity inputs for eastern Maine slightly later than usual, from mid-July to late August. Also, <i>Arctica</i> samples showed residual toxin from 2003 through spring and early summer, with toxicity levels degrading over the summer to very low levels (<80 µg), but a new pulse of toxicity appeared in <i>Arctica</i> in early September, indicating a strong down-welling event in eastern Maine in early fall.	Apr 20	Sep 14	May 11	Oct 13
		<i>Onset</i>	<i>End</i>	<i>Onset</i>	<i>End</i>
2005	Although eastern Maine experienced a “mild to average” season, western Maine experienced toxicity levels and inshore intrusion of toxicity that had not been documented at these levels for nearly two decades, since the 1980s. Also of note, <i>Arctica</i> showed no detectable toxicity most of the summer, through mid-July, until they showed a moderate spike of toxicity in late July, indicating a strong downwelling event in eastern Maine on or around July 28.	May 2	Aug 23	May 4	Aug 30
		<i>Onset</i>	<i>End</i>	<i>Onset</i>	<i>End</i>
2006	Incorporated dealer database and buoy data into this data set, since similar data were already merged into the main PSP data sets in previous years, and was used in calculated HAB Index values. The addition of so many MER buoys in 2006, with a dense spatial coverage, would create an artificial boost to both “Num of Stations” and “Biomass Score” in western Maine, if all points were used; therefore, MER buoy stations were clustered in groups that were spatially similar. The groupings are clustered as follows: 4/5, 10/11, 12/13, 14-18, 19/20, 22/23, 24/26, and 27/28. These groups are treated as a single station in the “Num of Stations” count, and the “Biomass Score” is averaged from each of these groups when scores were recorded in the same week.	Apr 19	Jul 23	May 11	Aug 21

Year	Notes	West		East	
	<p>Also, experimental “intertidal” buoys were not included in these metrics. These standardizations resulted in a change from “64” to “80” for the “Num of Stations” metric.</p> <p>In a previous data table, a sample result at Goose Rocks Beach was mistakenly recorded as “<i>Mytilus</i>” when it was actually a “<i>Spisula</i>” sample, which set the end date of the western Maine bloom on August 11. The actual end date is July 23 at the Ogunquit R station, which changes the “Duration” metric from 115 days to 96 days.</p>				
		<i>Onset</i>	<i>End</i>	<i>Onset</i>	<i>End</i>
2007	See notes for 2006 regarding dealer database and how to handle MER buoy stations. These same adjustments will be carried forward to all years past this point.	Apr 30	Aug 14	May 30	Sep 10
		<i>Onset</i>	<i>End</i>	<i>Onset</i>	<i>End</i>
2008	Fifteen additional buoy stations were added to Cobscook Bay this year. Because these stations were distributed across a wide area, with no spatial overlap, all of the data from these sites will be included in the HAB Index, without adjustments.	Apr 24	Jul 9	May 5	Sep 23
		<i>Onset</i>	<i>End</i>	<i>Onset</i>	<i>End</i>
2009	Although the 2009 seasonal length and toxicity patterns were fairly “average” for both regions of the state, there was an unusual spike in toxicity in early and mid-July that resulted in the bulk of toxicity in the “Biomass Score”, reaching a level in that metric that has not been reported since the 1980s for western Maine, and breaking all “Biomass Score” records for eastern Maine.	Apr 22	Sep 8	May 18	Nov 5
		<i>Onset</i>	<i>End</i>	<i>Onset</i>	<i>End</i>
2010	<p>Western Maine had an unusually early start to the season, but it was otherwise unremarkable, and ended by mid-summer.</p> <p>The eastern Maine “Biomass Score” is artificially</p>	Mar 22	Jun 21	Apr 26	Oct 13

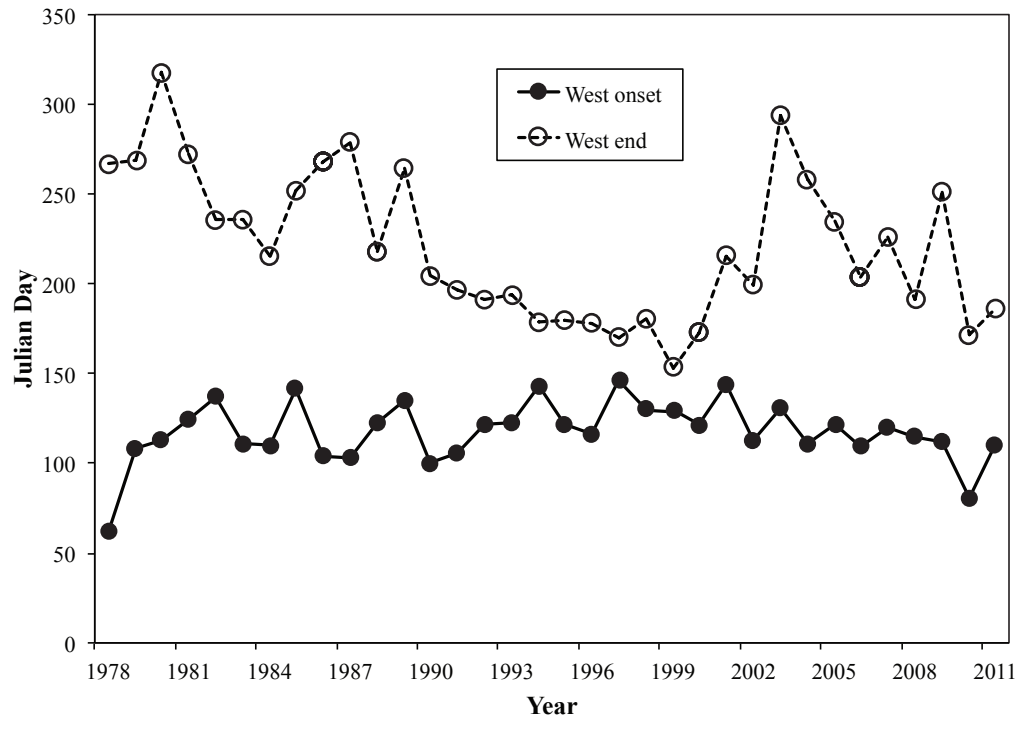
Year	Notes	West		East	
	high due to an extraordinary spike at Mount Desert Rock in early July (>14,000).				
		<i>Onset</i>	<i>End</i>	<i>Onset</i>	<i>End</i>
2011		Apr 20	Jul 5	May 9	Sep 19



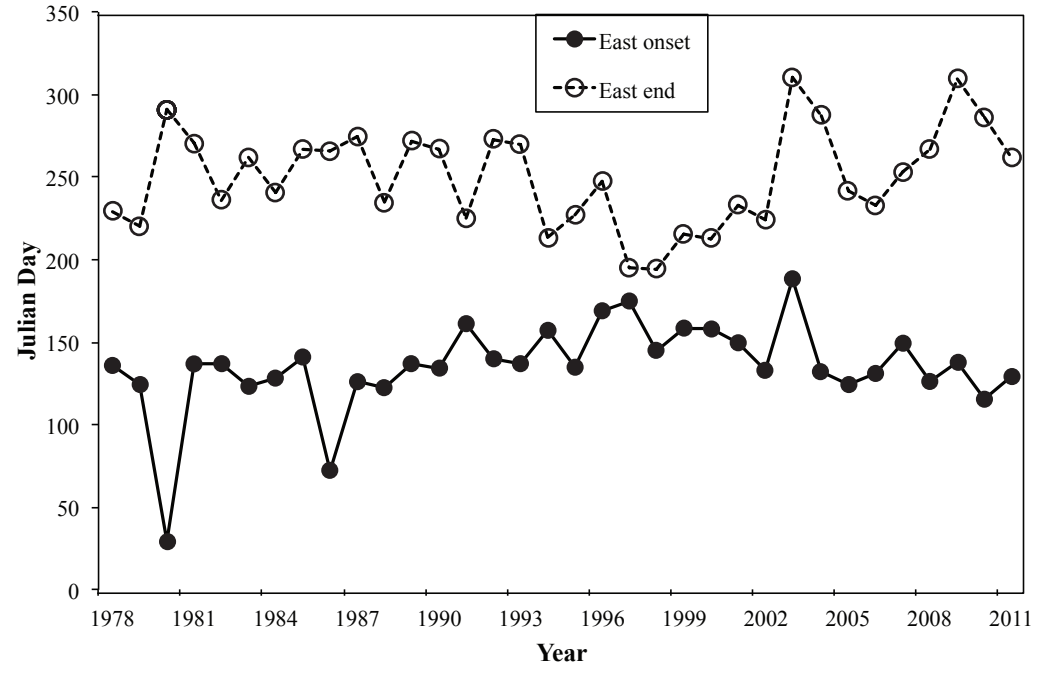


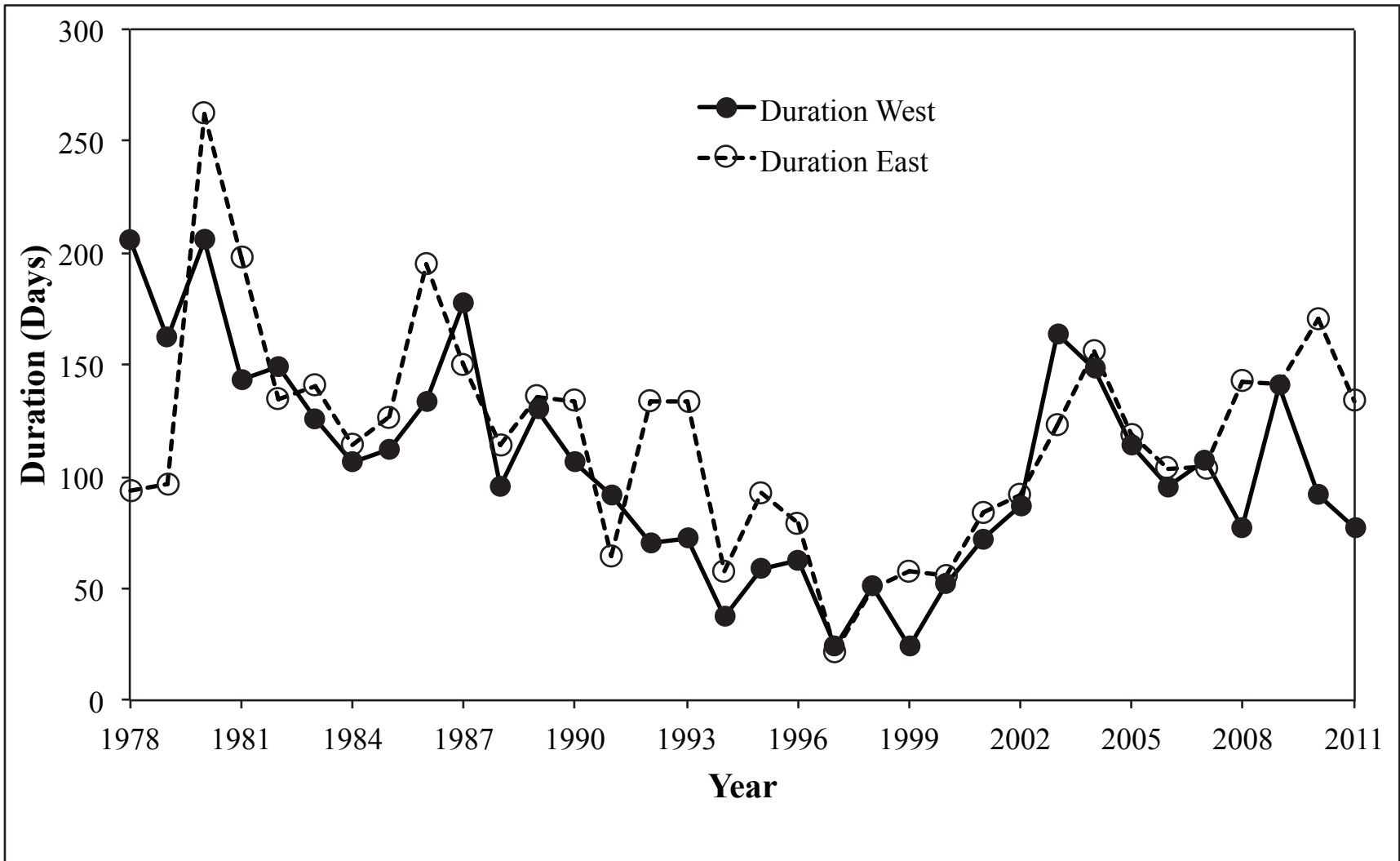


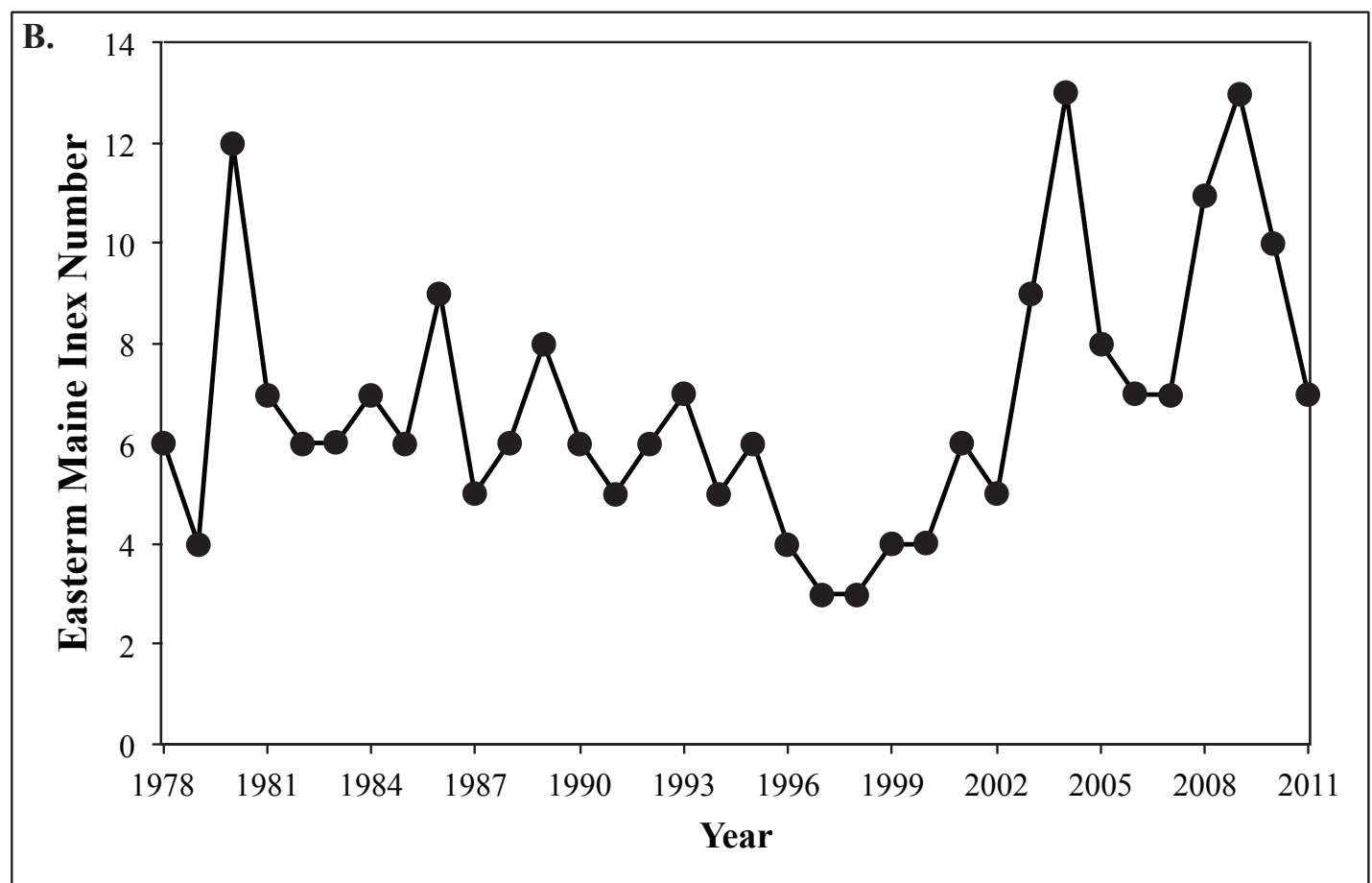
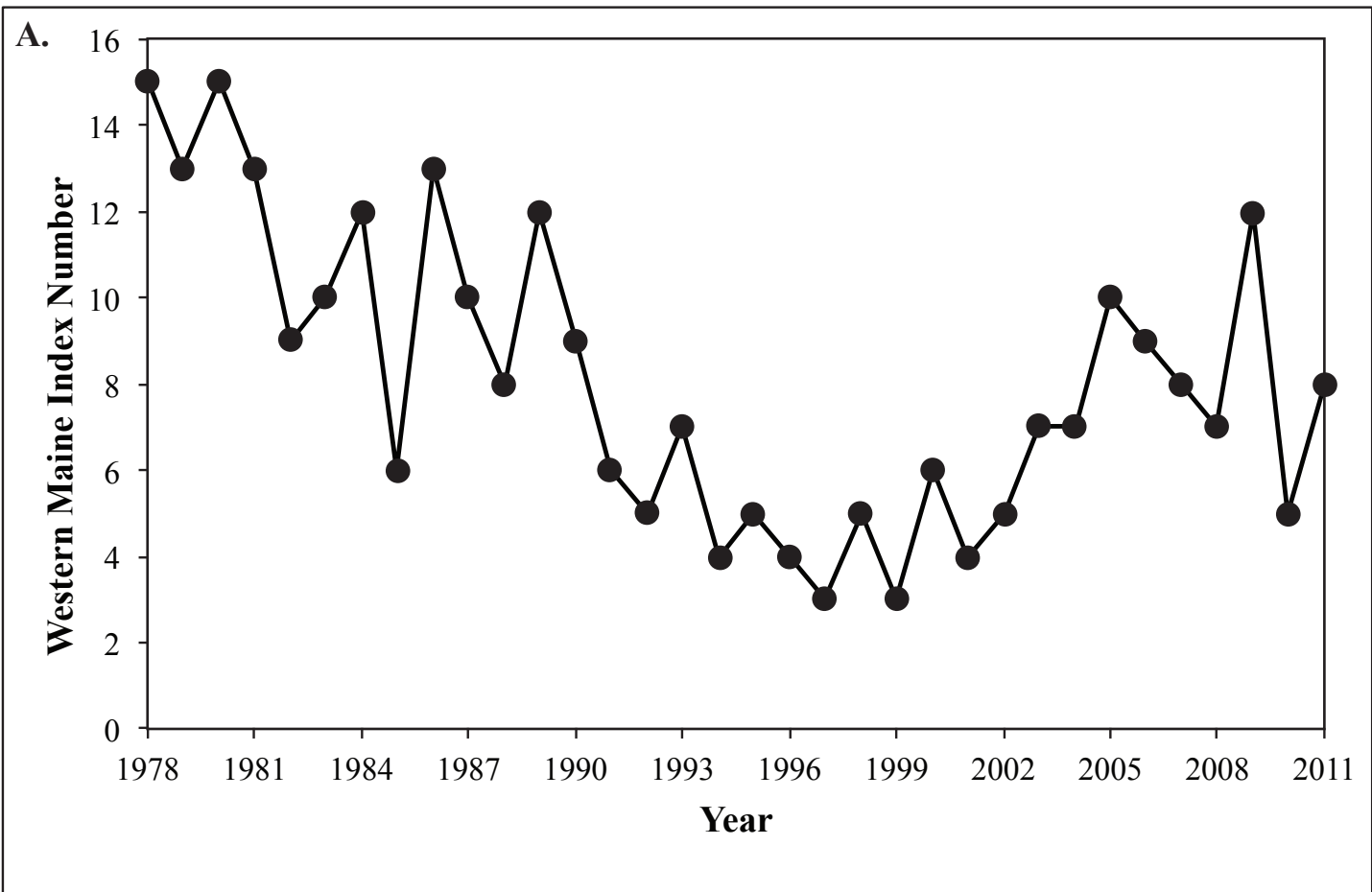
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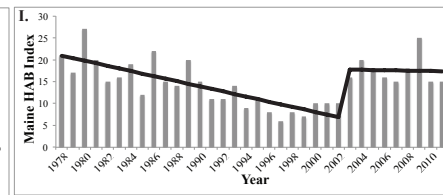
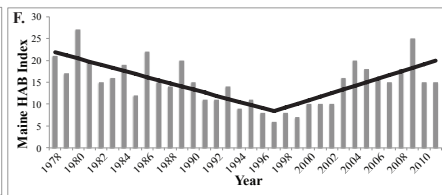
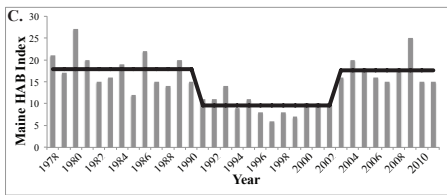
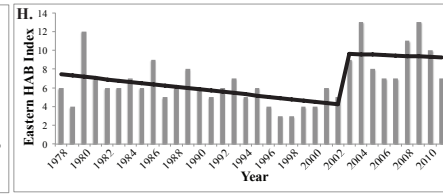
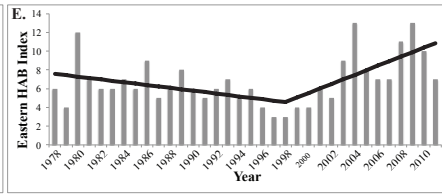
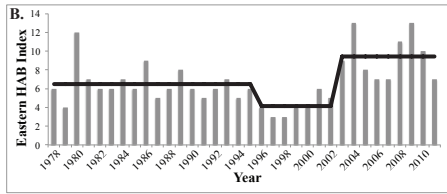
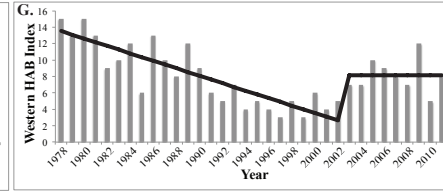
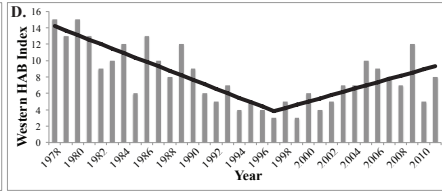
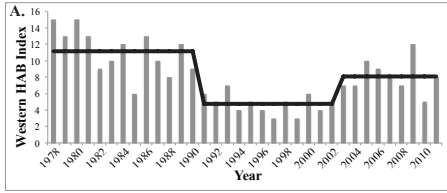


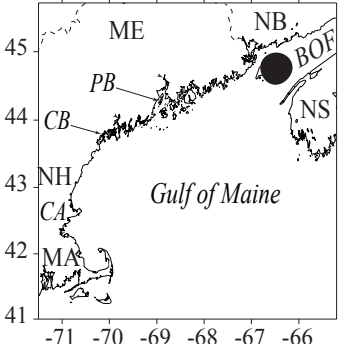
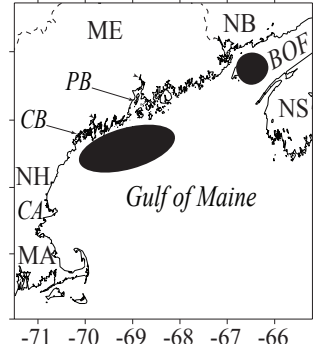
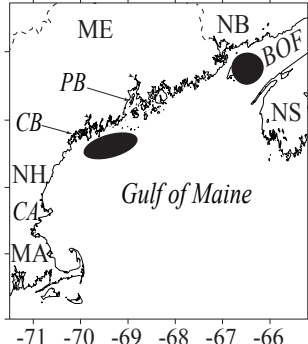
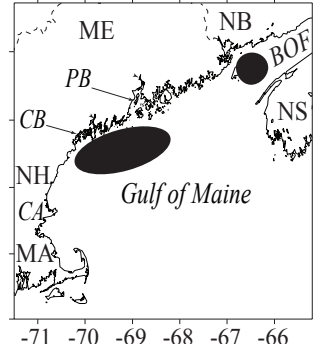
B.









Pre-1972	1972-mid-90s	mid-90s-2003	2003-2011
 <p>Cysts presumed to be present in Bay of Fundy (BOF) seedbed, but not abundant in western GOM (WGOM).</p>	 <p>Cysts presumed to accumulate in WGOM seedbed due to 1972 bloom and subsequent blooms.</p>	 <p>Hypothesized gradual (or precipitous) decline in WGOM seedbed leads to low abundance of WGOM cysts for over a decade.</p>	 <p>Cysts abundant in WGOM once again.</p>
<p>Recurrent toxicity in BOF; sporadic closures in eastern Maine (ME); toxicity low or non-existent most years in western ME.</p>	<p>Recurrent toxicity in BOF and eastern ME; start of 70's and 80's regime of frequent and high toxicity in western ME.</p>	<p>Recurrent toxicity in BOF; change to regime with low toxicity in western and eastern ME.</p>	<p>Recurrent toxicity in BOF; begin new regime of high toxicity in western and eastern ME.</p>
<p>HAB Index data unavailable.</p>	<p>HAB Index commences in 1978. Mean-shift model identifies period of early, high toxicity for western ME (1978-91) and eastern ME (1978-96). Two-phase and pulse-decline models define this period as one of gradually declining toxicity for both regions.</p>	<p>Mean-shift model identifies this period of low toxicity in ME, ending in 2003 for both regions. Two-phase model identifies 1998 (western ME) and 1999 (eastern ME) as change-point years marking the shift from an era of declining toxicity to one of increasing toxicity. Pulse-decline model defines 2002 as changepoint between decline phase and new pulse.</p>	<p>Mean-shift model identifies this period as one of renewed high toxicity, commencing in 2003 for both ME regions. Two-phase linear model defines this period as one of gradually increasing toxicity, continuing to the end of the HAB Index time series. Pulse-decline model indicates a pulse in 2003. Subsequent years show a sustained high level of toxicity or the beginning of a gradual decline.</p>