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E-ARTICLE

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ABSTRACT: Phenological mismatch—maladaptive changes in phenology resulting from altered timing of environmental cues—is an increasing concern in many ecological systems, yet its effects on disease are poorly characterized. American lobster (*Homarus americanus*) is declining at its southern geographic limit. Rising seawater temperatures are associated with seasonal outbreaks of epizootic shell disease (ESD), which peaks in prevalence in the fall. We used a 34-year mark-recapture data set to investigate relationships between temperature, molting phenology, and ESD in Long Island Sound, where temperatures are increasing at 0.4°C per decade. Our analyses support the hypothesis that phenological mismatch is linked to the epidemiology of ESD. Warming spring temperatures are correlated with earlier spring molting. Lobsters lose diseased cuticle by molting, and early molting increases the intermolt period in the summer, when disease prevalence is increasing to a fall peak. In juvenile and adult male lobsters, September ESD prevalence was correlated with early molting, while October ESD prevalence was correlated with summer seawater temperature. This suggests that temperature-induced molting phenology affects the timing of the onset of ESD, but later in the summer this signal is swamped by the stronger signal of summer temperatures, which we hypothesize are associated with an increased rate of new infections. October ESD prevalence was ~80% in years with hot summers and ~30% in years with cooler summers. Yearly survival of diseased lobsters is <50% that of healthy lobsters. Thus, population impacts of ESD are expected to increase with increasing seawater temperatures.

Keywords: disease prevalence, disease impacts, life history, Crustacea, phenology, molting.

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Introduction

Environmental conditions are shifting in seasonality, variation, and intensity across the globe (e.g., Doney et al. 2012), raising concerns that altered phenology of life-history events resulting from temporal shifts in environmental cues may cause previously adaptive life-history strategies to become maladaptive. This phenomenon, called phenological mismatch, is particularly likely to disrupt interspecies interactions. For example, some bird migrations and nesting patterns are no longer timed with peak food availability, resulting in insufficient resources for parents and young (Visser and Both 2005); microzooplankton (copepods and heterotrophic dinoflagellates) are emerging earlier in some marine systems and not coinciding with the photoperiod-driven blooms of their primary diatom food sources, thereby altering spring bloom dynamics (Edwards and Richardson 2004); and flowering in some spring plants is no longer timed to maximize pollinator services, affecting both plants and pollinators (Kudo and Ida 2013).

Disease is one of the areas where the impacts of phenological mismatch are poorly understood (Campbell et al. 2015). Changes in environmental cues can alter the spatial and temporal overlap between susceptible stages of the host and virulent stages of the pathogen, thereby changing the length or severity of an epizootic event, the timing of transmission, or the likelihood that critical densities of infectious stages may be surpassed. This can potentially disrupt tightly coevolved host-pathogen interactions or facilitate the emergence of new or previously uncommon pathogens (Altizer et al. 2006).

For some marine taxa, infectious diseases are increasing in emergence, prevalence, or severity (Ward and Lafferty 2004). In the oceans, many of these diseases are environmental or opportunistic (Burge et al. 2013; Shields 2013). Opportunistic diseases occur when environmental stressors weaken the physical and physiological defenses of their hosts

(e.g., physical barriers to disease and immune responses), thereby allowing microbes to shift from commensal to pathogenic and potentially causing substantial morbidity or mortality. While host population density is a common factor influencing the prevalence of infectious diseases with density-dependent transmission, it is unclear how much population density influences opportunistic diseases that are thought to be caused by pathogens that have a ubiquitous distribution (Burge et al. 2013). Phenological mismatch may be associated with the prevalence of infectious diseases because many of the traits commonly affected by phenological mismatch (e.g., defenses and growth) are associated with epidemiological processes.

Temperature shifts are an obvious candidate for driving phenological mismatch in the oceans. Seawater temperature is an important cue for numerous life-history transitions. Many marine systems, particularly those at high latitudes, are undergoing rapid changes in seasonal patterns of seawater temperatures (Levitus et al. 2012; Pershing et al. 2015). Warming has been associated with marine disease outbreaks, including epizootic shell disease (ESD) and gaffkemia in the American lobster (Stewart et al. 1969; Glenn and Pugh 2006), *Hematodinium* outbreaks in snow crabs (Shields et al. 2007), sea star wasting disease in various asteroids (Eisenlord et al. 2016), withering syndrome in black abalone (Ben-Horin et al. 2013), and aspergillosis in sea fans (Ward et al. 2007). However, for many marine diseases, there are not enough data on environmental cues, phenology, or disease emergence to evaluate the role of altered phenology in epidemiological patterns.

One case where phenological mismatch may be altering patterns of a putatively opportunistic disease is ESD in the American lobster. This emerging disease is characterized by extensive pitting, erosion, and melanization of the cuticle (Smolowitz et al. 2005; Shields et al. 2012). These signs are caused by a dysbiotic association with a suite of chitinolytic bacteria on the cuticle (Chistoserdov et al. 2012; Meres et al. 2012). Typical of many opportunistic diseases, bacterial community composition on the epicuticle is similar on healthy and diseased lobsters from the same locations, although the relative abundances of species can differ substantially (Chistoserdov et al. 2012; Meres et al. 2012). Transmission of ESD between lobsters is challenging to achieve experimentally; however, the disease frequently manifests when lobsters are held in stressful situations or have compromised exoskeletons (Quinn et al. 2012; Shields 2013). This is suggestive that environmental condition, as opposed to pathogen exposure, is the limiting factor on disease emergence. Mortality due to the disease can be high. The estimated survival of moderately and severely diseased lobsters (i.e., with disease on more than 10% of their cuticle) is 30% that of healthy animals (Hoenig et al. 2017).

Prevalence of ESD is driven in part by temperature (Glenn and Pugh 2006; Castro and Somers 2012) and is highest when summer temperatures exceed 12°C (Glenn and Pugh 2006; Castro and Somers 2012). The exception to this pattern occurs when lobsters molt. Prevalence of ESD is lowest just after the molting season, which occurs in the late spring at lower latitudes (e.g., Long Island Sound, United States) and summer and early fall at higher latitudes (e.g., Bay of Fundy and Gulf of St. Lawrence, Canada; Krouse 1973; Briggs and Mushacke 1979; Comeau and Savoie 2001). If molting is successful, lobsters rid themselves of disease signs when they shed their infected exoskeleton (Castro and Angell 2000). This suggests that molting phenology can influence epidemiological processes. Molting in severely diseased lobsters can be fatal when the cuticle damage causes adherence between the new and old carapace (Stevens 2009). Both the disease and the molt cycle are associated with temperature, although these relationships are not well quantified (Glenn and Pugh 2006; Thakur et al. 2016).

In this study, we investigated patterns of molting and the prevalence of ESD in relation to changes in seasonal temperatures and lobster population size in Long Island Sound. This area is near the epicenter of the initial ESD outbreak, which started in 1996, and contains part of the Southern New England lobster stock, which has declined so much over the past 2 decades that it has been classified as collapsed (ASMFC 2015). We used a 34-year mark-recapture data set from this eastern Long Island Sound to examine and model the role of temperature, molting phenology, and population density in the epidemiology of ESD. We evaluated the following hypotheses about the roles that temperature, molting, and phenological mismatch are playing in what are now yearly outbreaks of ESD.

H_1: Spring molting phenology is altered by temperature.

H_2: Spring molting reduces the risk of ESD.

H_3: ESD induces molting.

H_4: Prevalence of ESD is altered by spring molting phenology and summer temperatures.

Methods

A total of 217,466 lobsters were tagged by personnel at the Millstone Environmental Laboratory between 1982 and 2015. From May through October of each sampling year, 60 baited lobster traps were set and checked three times a week from 1982 through 2011 and twice a week after 2011.

Tagging locations were in eastern Connecticut near Jordan Cove, Niantic Bay, and Twotree Island (41°18′N, 72°10′W). Lobsters caught in traps were examined for carapace length, sex, reproductive condition, and ESD and then tagged with serially numbered, international orange, sphyrion tags and released at the site of capture (Landers et al. 2002). Retention of sphyrion tags by intermolt American lobsters is >96% (Moriyasu et al. 1995). Before release, lobsters were assigned to one of four disease states: healthy, no signs of ESD; mild disease, active ESD covering <10% of the carapace; moderate disease, active ESD covering 11%–50% of the carapace; and severe disease, active ESD covering >50% of the carapace. Lobsters recaptured during subsequent research surveys were examined for growth, reproductive state, and disease state and returned to the water at the recapture site. Bottom temperatures, continuously recorded by the Millstone Environmental Lab since January 1, 1976, from Niantic Bay (within 1.6 km of all sampling sites), were used in analyses involving temperature.

Data Exploration: Temperature, Molting, Disease, and Population Size

Before testing hypotheses, we characterized (1) multiyear trends in bottom temperatures, (2) the phenology of molting averaged across years, (3) the average seasonal trends in the prevalence of ESD, and (4) yearly trends in catch per unit effort (CPUE; a proxy for population size). Trends in bottom temperatures at Niantic Bay were calculated using average seasonal temperatures from 1976 to 2015. Seasons were defined by quarters (i.e., winter was January 1–March 31, spring was April 1–June 30, summer was July 1–September 30, fall was October 1–December 31). For each season, a linear regression was fit in order to determine the average rate of temperature change, the significance of this change, and the amount of variation explained by this trend.

Average monthly patterns in molting, ESD prevalence, and yearly trends in ESD were established by calculating the monthly proportions of diseased or molting lobsters and, for the first two calculations, averaging them across the years 1999–2015 (years when ESD was established at the study site). A lobster was considered to be diseased if it had any signs of ESD. A lobster was considered to be in a molting stage if it had a soft shell, a soft dorsal merus, soft carpus, or a notable crack along the midline of the carapace (indicative of imminent molting) at the time of tagging. Our methods likely underestimate molting because lobsters shelter immediately before and after molting (Karnofsky et al. 1989) and are therefore less likely to be caught. As such, we considered them indicative of trends in molting patterns but a large underestimate of the true molting activity. Adult males, adult females, and juveniles (carapace length <75 mm) were analyzed separately. For each of these groups, months were excluded when the sample size was <5, and a lowess curve (stiffness parameter $f = 2/3$) was fit to the resulting data. The majority of lobsters captured at the study site were male, and if they were female, they were not likely to be ovigerous. This is consistent with previous studies showing that females prefer cooler, deeper offshore waters than those found in shallow nearshore embayments during the summer (Jury and Watson 2013) and that ESD causes substantial mortality in ovigerous females relative to males (Hoenig et al. 2017). Because of their low capture rates, we excluded ovigerous females from this and all subsequent analyses. Only lobsters tagged for the first time were used in this and other analyses that did not account for recapture (i.e., in H_1 and H_4).

We calculated the average CPUE for two time periods—May through June (spring CPUE) and August through September (summer CPUE)—for each year of the study. We used two different methods to calculate CPUE in the spring and in the summer to reflect the possible effects of immersion time and trap saturation on our estimates. This was necessary because trap immersion time has been shown to influence lobster catch (Estrella and McKiernan 1989; Saila et al. 2002). Moreover, the immersion time for our sampling protocol changed slightly in 2012. In the first method for calculating CPUE (the saturated method), we assumed that traps saturated in less than the minimum immersion time. This was incorporated into calculations of CPUE by dividing the number of lobsters caught during these months by the product of the number of lobster traps checked during these sampling periods and 2.33 (the average immersion time if traps were checked three times per week; also the method used in this study until 2012) to give the number of lobsters per trap × day. The second method for calculating CPUE (the unsaturated method) was identical except that for the years 2012–2015 (when traps were only checked twice weekly), an average immersion time of 3.5 days was used in the calculations.

Hypothesis Testing

H_1: Spring molting phenology is altered by temperature.

The effect of temperature on the timing of peak molting was examined using linear regression. In order to determine the timing of the onset of molting, the proportion of animals that were in premolt stages (i.e., soft merus or carpus) was calculated for each week of each year from 1982 to 2015. We wanted to quantify the timing of spring molting and exclude data from the onset of fall molting, which in Long Island Sound can begin in late September for juveniles and adult males and October for adult females. Therefore, we

limited the data set to the first 11 weeks after May 1 for juveniles and adult males and the first 17 weeks after May 1 for adult females (which molt ~4 weeks later than juveniles and adult males). We then fit a cumulative distribution curve to these data for each year and interpolated between the closest points above and below 50% of the molting activity for the season to estimate the time period (in weeks) when peak molting activity occurred. Estimates from years where there were five or fewer lobsters observed in premolt stages were excluded (remaining sample size = 16, 11, and 32 for adult males, adult females, and juveniles, respectively). Potential environmental predictors were the average winter temperature, the minimum winter temperature, the maximum winter temperature, and the average May temperature. Each of these temperature metrics was included in a separate linear model, and models were run with and without interactions with life-history stage and sex. We then used Akaike information criterion corrected for finite sample sizes (AICc) to calculate the best-fitting environmental variables to predict the timing of peak molting.

H_2: Spring molting reduces the risk of ESD.

The factors that determine the probability of a tagged lobster being recaptured in the disease state were examined using logistic regression without overdispersion (e.g., Hoenig et al. 2017). Using data from recaptured lobsters, we examined whether the disease state at tagging, molting while at large, and time at large significantly contributed to the probability of being recaptured in a diseased state. Lobsters were considered to have molted if their carapace length at recapture increased by more than 2.5 mm (for details, see appendix, available online). Lobsters with molt increments >15 mm or <−2.5 mm were excluded from the analysis because those in the first group likely molted twice, whereas lobsters in the second group were likely measured incorrectly. Only data from tagged lobsters that were at large for 14–180 days in the years after ESD had fully emerged (1999–2015) were included in this analysis. Males and nonovigerous females were analyzed separately. For this analysis and the test of H_3, juveniles could not be analyzed separately from adults because these analyses require large data sets and the sample sizes for adults were relatively small.

H_3: ESD induces molting.

The previous hypothesis was focused on whether molting could alter the disease state of a lobster. This hypothesis is focused on the opposite interaction: can disease induce molting? To address this hypothesis, we used a logistic regression to analyze the probability of molting while at large as a function of carapace length at tagging, disease state at tagging, time of tagging, and the interaction between disease state at tagging and time of tagging. Time of tagging was divided into three 2-month blocks in order to examine whether season could interact with the effects being examined. Because larger lobsters molt less frequently than smaller ones (Aiken 1980), we included carapace length at tagging as a covariate. Males and nonovigerous females were analyzed separately. The data set included records from the years 1999–2015 and lobsters that were at large for at least 14 days. As in the previous analysis, lobsters that were at large more than 180 days were excluded.

H_4: Prevalence of ESD is altered by spring molting phenology and summer temperatures.

In Long Island Sound, seasonal patterns of ESD show that for adult males and juvenile lobsters, ESD prevalence is lowest in June and July and increases through October (Hoenig et al. 2017). For adult females, there are two smaller peaks; ESD prevalence peaks in June, decreases in July and August, and increases again in September and October. (It should be noted that this data set does not include samples from November through April.) Therefore, in this analysis, we focused on understanding correlations between temperature and molting phenology on the prevalence of ESD in September and October for adult male and juvenile lobsters and in September for adult female lobsters. Because of low sample sizes, we did not evaluate factors correlated with October prevalence of ESD in adult females. In all of these analyses, we included population size (represented by CPUE) as a predictor of ESD prevalence.

To identify factors correlated with the prevalence of ESD in September and October for adult males and juveniles, we fit multiple linear models evaluating the additive effects of maturity and sex (juvenile, adult female, or adult male), the proportion of lobsters caught in May that were observed molting, various temperature metrics, and summer CPUE (as a proxy for population density) on the prevalence of ESD. All continuous predictor variables were centered and standardized in order to reduce issues of collinearity. Because the mechanism for how temperature cues alter disease prevalence is not well understood, we included one of the following temperature metrics in each model: average summer temperature, average temperature for September of that year, or number of days in the summer exceeding 20°C. The suites of models predicting September and October prevalence of ESD were evaluated using AICc. In cases where multiple models had a good fit to the data (ΔAICc from the best-fitting model <2), we calculated full and conditional averaged models. The process to model ESD prevalence in adult females in September was similar, with the exception that we used the proportion of animals molting in June as a predictor, since molting occurred later in females. All other predictors were the same.

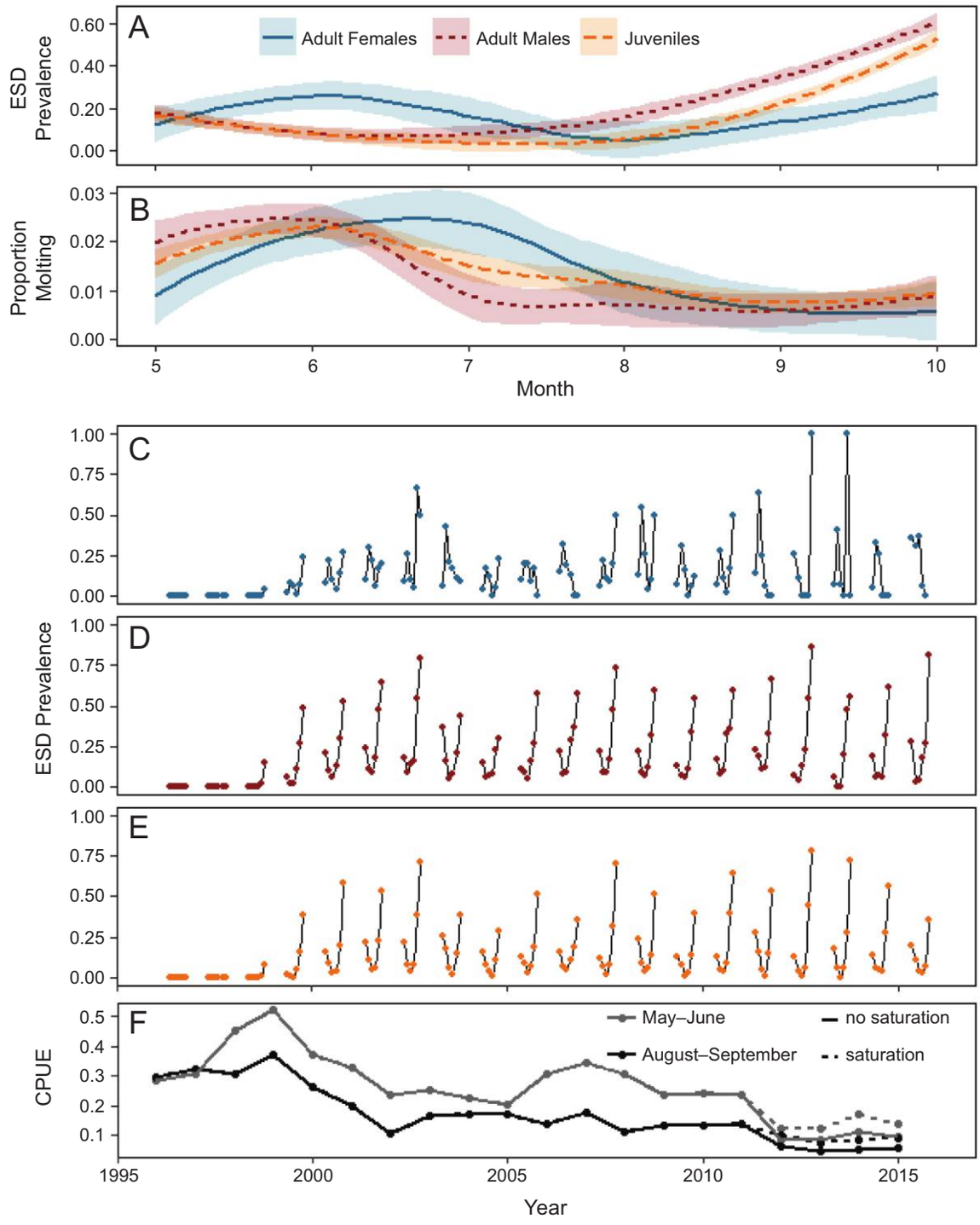

Figure 1: Seasonal and yearly phenologies of epizootic shell disease (ESD), molting, and lobster population density in Niantic Bay. Seasonal phenology of ESD (*A*) and molting (*B*) are averaged across years from 1999 to 2015 and fit with a lowess curve ($f = 2/3$). Shading indicates standard errors around the mean. Yearly patterns in prevalence of ESD are depicted separately for adult female (*C*), adult male (*D*), and juvenile (*E*) lobsters. Catch per unit effort (CPUE; a proxy for population density) for the spring and summer are depicted two ways: first, assuming that lobster traps are saturated after 2.3 days, and second, assuming that they do not saturate before the traps are sampled (*F*).

For all analyses of H_4, only data from 1999–2015 were used in order to focus on years when ESD had become endemic in the lobster population. Only lobsters tagged for the first time were used in this analysis in order to meet the assumption of independence.

Data and code associated with the above analyses are available in the Dryad Digital Repository: http://dx.doi.org/10.5061/dryad.n102b1s (Groner et al. 2018). Code is also available as supplementary material online.

Results

Data Exploration

Since 1976, the average bottom temperature in Long Island Sound has increased by 1.6°C (0.4°C per decade; fig. A1; figs. A1–A3 are available online). The weakest and most variable trend in temperature occurred in the winter, with average bottom temperatures increasing by 0.34°C per decade (linear regression; $t_{38} = 2.2$, $P < .05$, $R^2 = 0.11$). In the spring, the average bottom temperature increased by 0.40°C per decade ($t_{38} = 4.0$, $P < .005$, $R^2 = 0.30$). In the summer and fall, the average bottom temperatures increased by 0.45°C ($t_{38} = 6.0$, $P < .00001$, $R^2 = 0.48$) and 0.43°C ($t_{38} = 3.8$, $P < .0005$, $R^2 = 0.27$) per decade, respectively.

From 1999 through 2005, ESD prevalence for adult males and juveniles was highest in October and lowest in June and July (fig. 1*A*). For adult females, disease prevalence had two peaks, one in June and another in October. The average peak in molting activity for adult males and juveniles occurred in late May and early June, whereas for adult females it occurred in mid- to late June (fig. 1*B*). Prevalence of ESD from May through October showed a characteristic check mark–shaped trend in each year for adult males and juveniles (fig. 1*C*–1*E*). The prevalence of ESD in October was highest for juveniles and adult males in 2012 when it was 78% and 85%, respectively. Prevalence of ESD in October was lowest for adult males and juveniles in 2004. ESD was not observed in adult females in October 2005 or 2006. The pattern in females was more complex. Because of low sample sizes, the prevalence of ESD in adult females could not be calculated in October 2008 and 2010–2015 and in September 2011–2013. The highest October disease prevalence for females (among years when it could be calculated) occurred in 2007, when it was 50%.

Lobster density, as measured by CPUE, varied considerably during the time span of this study (fig. 1*F*), increasing until 1999, after which it fell by more than 50% over the course of 4 years (55% for spring CPUE, 67% for summer CPUE). CPUE was fairly constant between 2002 and 2011. It dropped in 2012 (by 73%–85% relative to 1999 levels, depending on the method used) and remained low thereafter. During most years, spring CPUE was higher than summer CPUE.

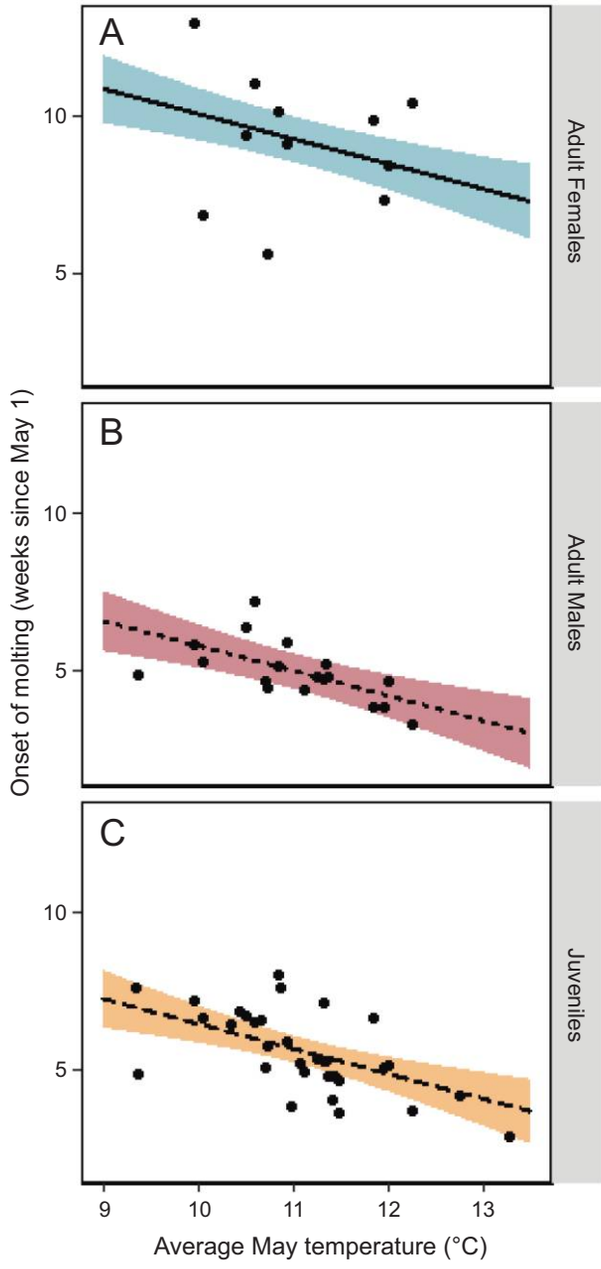

Figure 2: Effect of May temperatures on molting phenology for adult female (*A*), adult male (*B*), and juvenile (*C*) lobsters. Peak molting (measured in weeks since May 1) in all three groups occurs earlier when the winter is warmer. Shading indicates 95% confidence intervals; circles indicate yearly means in the onset of molting activity. Data are from 1982 through 2015.

Hypothesis Testing

H_1: Spring molting phenology is altered by temperature.

The onset of molting occurred earlier in warmer years (fig. 2). The best-fitting model for the onset of molting included additive effects of the average May temperature, maturity, and sex (i.e., juvenile, adult male, or adult female) as predictors. The onset of the premolt stage for adult females was later than that for juveniles and adult males by 3.6 ± 0.4 weeks ($t_3 = 8.4$, $P < .0001$). There was no difference between adult males and juveniles in the phenology of the onset of molting ($t_3 = -1.8$, $P = .08$). For all lobsters, molting occurred earlier when the average May temperature was warmer. For every 1°C increase in the average May temperature, molting was 0.8 weeks, or about 6 days earlier ($t_3 = -4.0$, $P = .0002$). Differences in sample sizes may explain the much larger variance in the predictions for adult females. The data set consisted of 14,846 adult females, 26,629 adult males, and 103,108 juvenile lobsters. This model explained much of the variation in the data (adjusted $R^2 = 0.61$). A similar model, which was identical except that it included average winter temperature instead of average May temperature, was also a good fit for the data (ΔAICc = 0.4). No other models had a ΔAICc from the best model that was <2.

H_2: Spring molting reduces the risk of ESD.

The probability of being diseased at recapture was influenced by molting, time at large, disease state at tagging, and the interactions among these variables (table 1; fig. 3). For both males and females that were healthy when tagged, the probability of disease at recapture was close to zero if they were recaptured within a month of tagging. The probability of becoming diseased increased with time at large, such that if males did not molt, their probability of disease at recapture approached 100% after 6 months at large. This trend also occurs in females. For those males and females that were tagged when healthy and molted while at large, the probability of disease at recapture increased with time at large, but it never exceeded 20%.

For males and females that were diseased at tagging, the probability of being diseased at recapture remained high for lobsters that did not molt. For males, it was close to 80% and was not significantly affected by time at large. For females, it was close to 90% soon after tagging and decreased with time at large. This decrease may be an artifact of a small sample size; there were no recaptures after the first 3 months. In contrast, for males that were diseased at tagging and molted while at large, the probability of disease at recapture was ~10% initially and increased to 30% after 6 months. For females that were diseased at tagging and molted while at large, the probability of disease at recapture was 0% initially and remained at that level. No diseased females were recaptured after the first 3 months. Data from 1,994 females and 6,594 males were used in this analysis.

H_3: ESD induces molting.

Recapture data were available from 6,594 males and 1,994 nonovigerous females for analysis of the probability of molting (table 2; fig. 4). Both males and females had a higher probability of molting if they were diseased; however, this decreased with increasing size. For males, the molting probability was significantly higher in May and June relative to other months. There were no significant interactions between disease and time of tagging. For females, there was an interaction between disease and time of tagging such that the effect of disease on the molting probability was significantly higher in July and August than in other months.

Table 1: Results of logistic regression assessing the probability of a lobster being diseased at recapture 14–180 days after tagging

	Males				Nonovigerous females			
	Estimate	SE	z	P	Estimate	SE	z	P
Intercept	**−3.51**	**.08**	**−41.6**	**<.0001**	**−3.69**	**.19**	**−19.8**	**<.0001**
Time at large	**14.19**	**.45**	**31.3**	**<.0001**	**12.82**	**1.25**	**10.3**	**<.0001**
Molted	.32	.39	.8	.421	−.82	.81	−1.0	.31
Disease at tagging	**5.20**	**.26**	**20.2**	**<.0001**	**6.53**	**.71**	**9.3**	**<.0001**
Time × molted	**−9.88**	**1.59**	**−6.2**	**<.0001**	−5.46	3.37	−1.6	.11
Time × disease	**−17.62**	**2.02**	**−8.7**	**<.0001**	**−24.20**	**6.09**	**−4.0**	**<.0001**
Molted × disease	**−4.69**	**.89**	**−5.3**	**<.0001**	**−6.05**	**3.07**	**−2.0**	**.05**
Time × molted × disease	**17.29**	**4.26**	**4.1**	**<.0001**	17.56	20.84	.8	.40

Note: Predictor variables include time at large, whether molting occurred between tagging and recapture, disease state at tagging, and interactions between these variables. Males and females were analyzed separately. The baseline for comparison is healthy lobsters that did not molt. Significant effects are in bold.

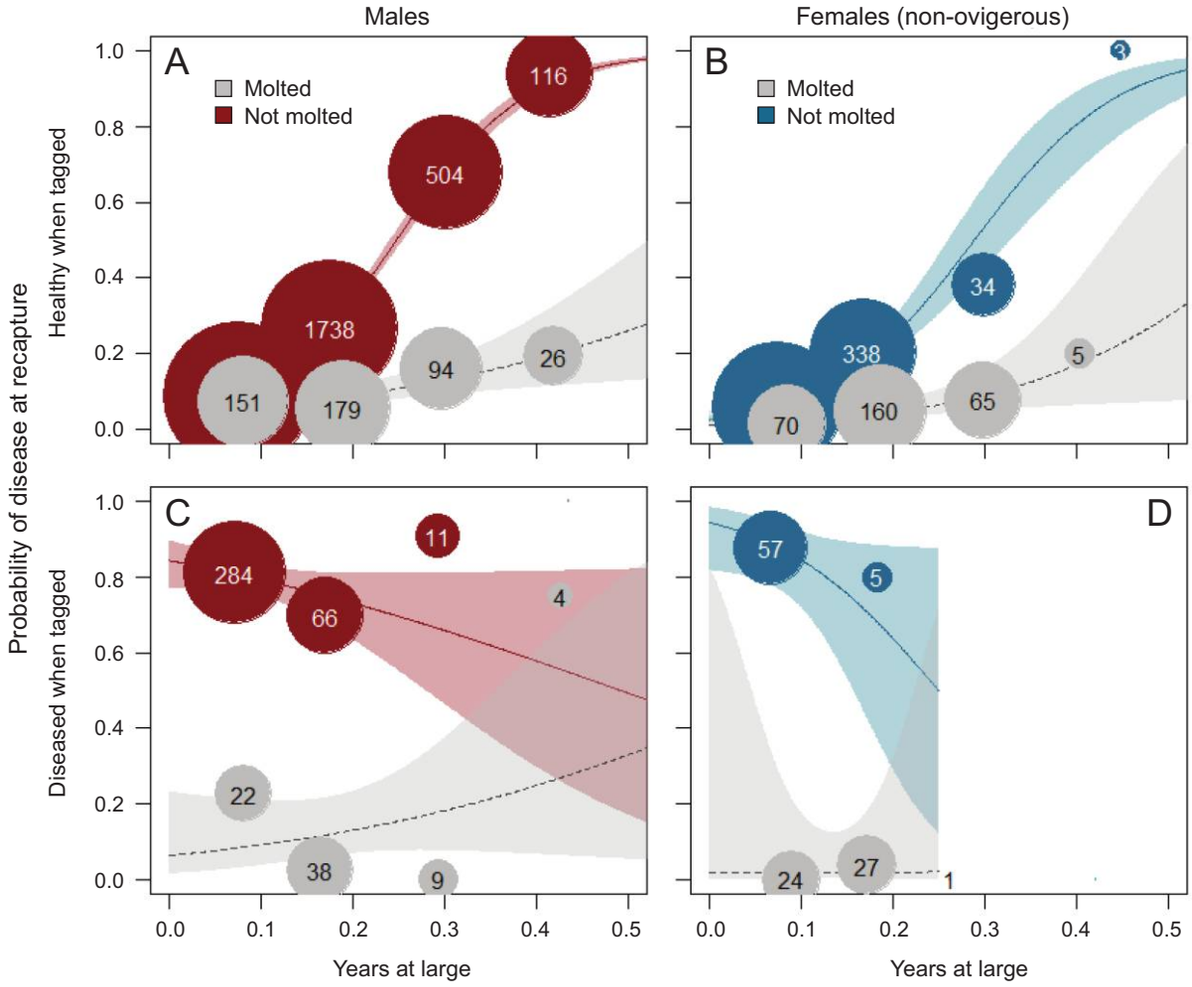

Figure 3: Relationships between initial disease state, molting, and time at large on disease state at recapture. Males and nonovigerous females were analyzed separately. Circles indicate aggregations of actual data points and are proportional to the sample size (numbers inside circles); lines and shading indicate predictions and 95% confidence intervals from logistic regression, respectively. Note that for *D* there were no samples after 0.25 years at large.

H_4: Prevalence of ESD is altered by spring molting phenology and summer temperatures.

The September prevalence of ESD for juvenile and adult male lobsters was strongly correlated with life stage and molting phenology in May and weakly correlated with summer temperatures and summer CPUE (calculated using the saturated method; table 3; fig. 5*A*–5*C*). There were four models with $\Delta\text{AICc} < 2$. Of these, all four included terms for life stage and molting phenology, and three included terms for summer CPUE and/or summer temperature. In both the conditional and full averaged models, ESD prevalence was 13% ± 3.3% (mean ± SE) higher in adult males than in juveniles ($z = 3.7$, $P < .001$ in both models). The proportion of lobsters molting in May was positively correlated with ESD prevalence such that predicted ESD prevalence in a year with an early molting phenology (5% observed molting in May, >30% predicted ESD prevalence) was double the predicted ESD prevalence in a year with a late molting phenology (1% observed molting in May, 15% predicted ESD prevalence; $z = 2.3$, $P = .02$ in both conditional and full averaged models). ESD prevalence increased with the number of days that exceeded 20°C in the previous summer; however, this effect was not significant in either the full averaged model or the conditional averaged model ($P \geq .1$). Summer CPUE was negatively correlated with the prevalence of ESD in September; however, this effect was nonsignificant in both the full and the conditional averaged models ($P > .1$). The R^2 of the best-fitting model was 0.46, suggesting that as yet unidentified factors are also influencing prevalence of ESD in September.

The October prevalence of ESD for juvenile and adult male lobsters was strongly correlated with summer temper-

Table 2: Results of logistic regression assessing probability of molting for lobsters at large for 14–180 days as a function of size (carapace length), disease state (healthy or diseased), and time of tagging (divided into 2-month intervals)

	Males				Nonovigerous females			
	Estimate	SE	z	P	Estimate	SE	z	P
Intercept	**9.80**	**.61**	**16.18**	**<.0001**	**10.00**	**.83**	**12.04**	**<.0001**
Carapace length (mm)	**−.17**	**.009**	**−19.53**	**<.0001**	**−.166**	**.012**	**−13.52**	**<.0001**
Diseased	**1.57**	**.17**	**9.49**	**<.0001**	**1.56**	**.26**	**5.92**	**<.0001**
July–August	**−.62**	**.13**	**−4.77**	**<.0001**	**−.94**	**.17**	**−5.40**	**<.0001**
September–October	**−1.25**	**.465**	**−2.70**	**.007**	**−2.79**	**1.01**	**−2.75**	**.003**
Diseased × July–August	−14.63	230.68	−.06	.949	**2.37**	**.54**	**4.39**	**<.0001**
Diseased × September–October	−1.13	.78	−1.44	.151	1.88	1.31	1.44	.151

Note: Interactions for disease state (diseased or healthy) and time of tagging were also included. Males and nonovigerous females of all sizes were analyzed separately. The baseline model was for healthy lobsters that were tagged in May or June. Positive estimates indicate an increased probability of molting. Significant effects are in bold.

ature and weakly correlated with life stage, molting phenology in May, and summer CPUE (table 3; fig. 5*D*–5*F*). There were eight models with $\Delta\text{AICc} < 2$. Of these, all eight included a term for summer temperature, and four models included terms for molting phenology, life stage, and/or summer CPUE. In both the average and the full models, ESD prevalence was positively correlated with the number of days that exceeded 20°C in the previous summer. In years with only 50 days exceeding 20°C, an October prevalence of 40% was predicted, while in years with 90 days exceeding 20°C, an October prevalence of 65% was predicted ($z = 2.7$, $P < 0.01$ in both conditional and full averaged models). Disease prevalence was higher in adult males than in juveniles, positively correlated with molting phenology (earlier molting increased disease prevalence) and negatively correlated with summer CPUE; however, none of these terms was significant in the full or conditional averaged models (all $P > 0.05$). The R^2 of the best-fitting model was 0.37, suggesting that unidentified factors are also influencing prevalence of ESD in October.

Our models did not identify any factors that were correlated with September ESD prevalence in adult females. The null model was the best-fitting model, and there were no other models with $\Delta\text{AICc} < 2$ relative to this model. It should be noted that 5 years of data were rejected from use in this model because of the low sample sizes, and while the other years had $n > 5$, the sample sizes were still low relative to those of juveniles and adult males. Full and conditional averaged models for prevalence of ESD in September and October for juveniles and adult males are available in table A1, available online.

Discussion

Our analyses of a 34-year mark-recapture data set revealed complex relationships between temperature, molting phenology, and ESD in the American lobster. While largely correlative, the results of this study are consistent with the hypothesis that temperature-induced changes to molting phenology lead to a maladaptive phenology in the context of this emerging disease. Molting is critical for maintaining the health of disease-free lobsters and for recovery of lobsters with ESD. Warmer spring temperatures cue lobsters to molt earlier in the late spring and summer (Waddy and Aiken 1991; Tlusty and Metzler 2012), thereby allowing disease prevalence to increase throughout the summer and fall, uninterrupted by molting activity. The effect of molting phenology on disease prevalence is detectable in juvenile and adult male lobsters in September; however, by October, disease prevalence is more strongly correlated with summer temperatures, specifically the number of days exceeding 20°C. Indeed, 2012, the year with the greatest number of days exceeding 20°C during the study period (90 days), had the highest October ESD prevalence of all years for both juveniles and adult males. (The prevalence of ESD in October 2012 for females could not be calculated because of low sample size.) This suggests that molting phenology alters the onset of disease, but this signal becomes swamped by that of summer temperatures, which we hypothesize influences incidence, or the rate that new infections occur. The consequences of molting phenology and warmer summers on ESD prevalence are not trivial. Previous estimates in Long Island Sound calculate that the yearly survival rate of mildly diseased lobsters is 45% that of healthy lobsters (Hoenig et al. 2017). During years with warm winters and summers, disease prevalence has reached ~80% in juvenile and adult lobsters. Assuming a background natural mortality of 0.15 (ASMFC 2015) and that all lobsters reached only a mild disease state (a conservative assumption), yearly mortality would be expected to exceed 45% in years with high disease prevalence.

We could not identify any factors correlated with fall ESD prevalence in adult females. This is likely due to the low sample sizes for the fall months; females migrate to cooler deeper water at this time (Cowan et al. 2007). Nonetheless, the pattern of relatively low fall prevalence of ESD in adult females

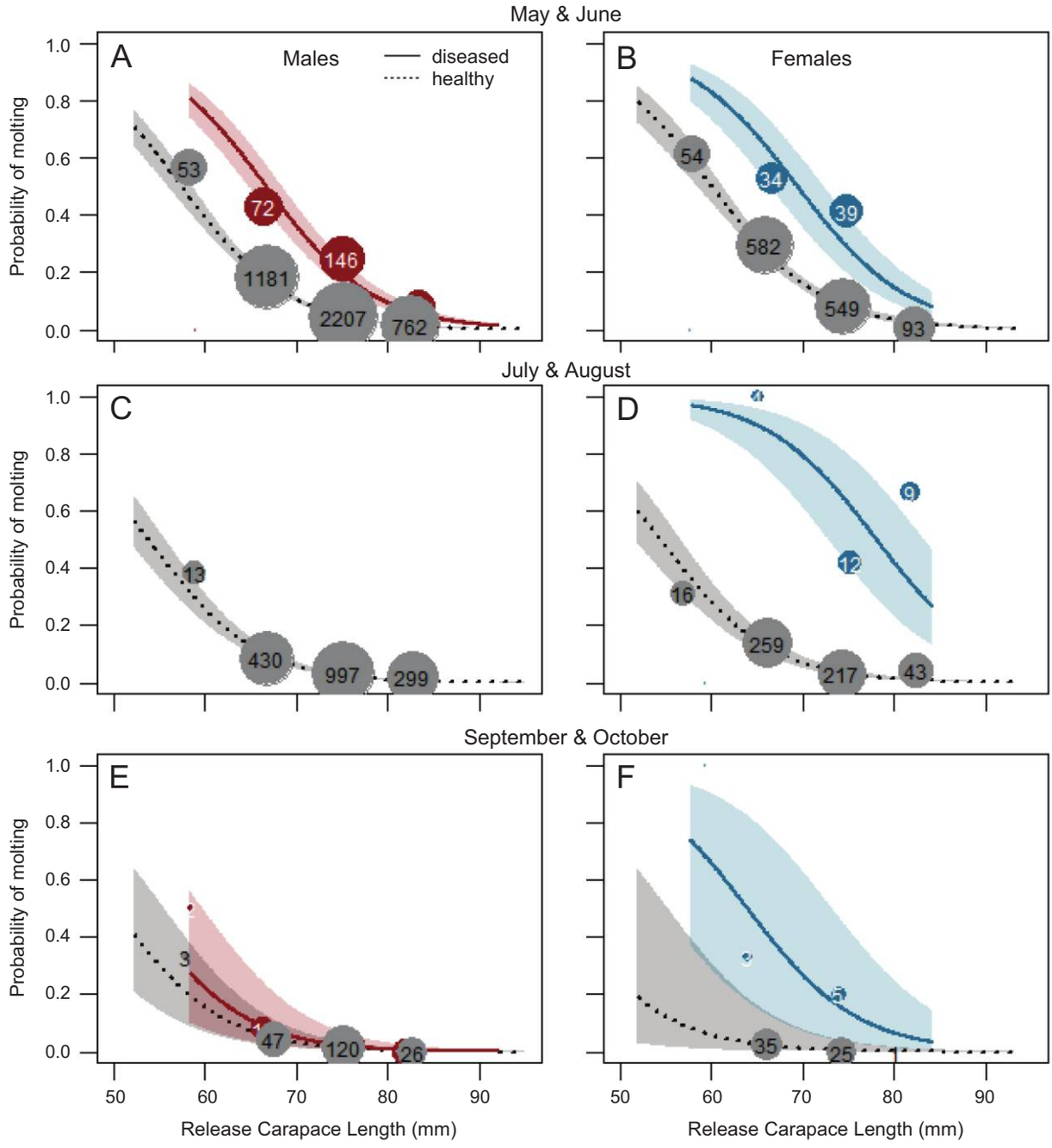

Figure 4: Probability of molting as a function of size at release, disease state, time of tagging, and sex. Males (*A*, *C*, *E*) and nonovigerous females (*B*, *D*, *F*) were analyzed separately. Circles indicate aggregations of actual data points and are proportional to the sample size (numbers inside circles). Shading indicates 95% confidence intervals.

compared with other life stages is consistent with our hypothesis that molting phenology affects disease prevalence; adult females molt on average 3 weeks later than adult males and juveniles. As a result, there is a shorter intermolt period in the summer for disease transmission to occur, and disease prevalence peaks at a lower level in the fall. Reproduction is tightly linked to the molt cycle in female lobsters. Further research is necessary to understand trade-offs between molting and fecundity in the context of ESD (Waddy and Aiken 1991).

Our findings suggest that warming temperatures facilitate disease through two mechanisms. First, warmer spring temperatures cause lobsters to molt earlier in the spring,

Table 3: Best models predicting the prevalence of epizootic shell disease in juvenile and adult male lobsters in September and October

	df	AICc	ΔAICc	Weight
September:				
Life stage + May molting + hot days	5	258.8	0	.34
Life stage + May molting + CPUE	5	259.2	.41	.27
Life stage + May molting + hot days + CPUE	6	259.7	.95	.21
Life stage + May molting	4	260.0	1.23	.18
October:				
Life stage + hot days + CPUE	5	275.2	0	.155
Life stage + hot days + May molting + CPUE	6	275.3	.07	.149
Life stage + hot days +	4	275.3	.08	.149
Life stage + hot days + May molting	5	275.4	.13	.145
Hot days	3	276.0	.77	.105
Hot days + summer CPUE	4	276.0	.78	.105
Hot days + May molting	4	276.2	.93	.097
Hot days + May molting + CPUE	5	276.2	.97	.095

Note: In both cases, models tested additive effects of May molting and a variety of summer temperature metrics, including the number of days exceeding 20°C (hot days) and summer catch per unit effort (CPUE; calculated using the unsaturated method). Juvenile lobsters include all individuals with a carapace length <75 mm. May molting refers to the standardized proportion of lobsters that were caught in any molt stages the previous May. Weight refers to the relative likelihood of a model and was used in model averaging. Model selection was based on minimizing corrected Akaike information criterion (AICc) values.

giving the new exoskeleton longer exposure to ESD during the summer and fall. Warmer summer temperatures are also correlated with an increased in ESD prevalence in the summer and fall, well after lobsters have molted. This suggests that incidence (the rate of new infections over time) is higher during warm summers. The first mechanism is correlated with patterns of disease in September, while the second is correlated with patterns of disease in October. Our data show that peak molting can shift by as much as 4 weeks in eastern Long Island Sound as a result of temperature shifts, and this allows ESD prevalence to increase throughout the summer and fall without being reset by molting. During the summer, bottom temperatures in eastern Long Island Sound exceeded 20°C for as many as 90 days in a year, and the amount of warm days occurring in the summer is correlated with disease prevalence during the epidemic peak in October. Temperatures above 20°C are considered stressful to lobsters. Indeed, lobsters exposed to experimental temperature treatments at 23°C showed numerous physiological signs of stress, including reduced phagocytosis, hemolymph acidosis, and other signs of altered hemolymph chemistry (Dove et al. 2005). Lab experiments demonstrate that ESD progression accelerates at warm temperatures (18°C), with lesions growing at approximately three times the rate of those at cooler temperatures (6° and 12°C; Barris et al. 2018). Positive correlations between stressful temperatures and disease prevalence are typical of opportunistic diseases, which emerge when the host is stressed (Burge et al. 2013; Shields 2013).

Developmental stage also plays a role in the complex relationship between molting and ESD. Juvenile lobsters (<50 mm carapace length) typically molt twice or more in a year, with the second molt occurring in the fall or winter (Harding et al. 1983). In contrast, adults (>100 mm carapace length) typically molt only once per year or not at all, depending on size and temperature regime (Aiken 1980). Whether warming temperatures are altering the timing of the late fall/winter molt among juveniles in Long Island Sound remains to be investigated and requires winter sampling of lobsters; however, other studies show that warm temperatures can delay fall molting activity (Tlusty and Metzler 2012; Thakur et al. 2016). If this occurs in Long Island Sound, it further increases the intermolt period allowing disease transmission and progression to occur in the summer and fall without interruption.

For lobsters with ESD, molting more frequently increases opportunities to shed diseased cuticle, but larger lobsters are unlikely to molt more than once per year; hence, the shift in phenology puts them at risk to infection. Although lobsters already molt more frequently as juveniles, our results suggest that ESD further stimulates juvenile molting, which provides the short-term benefit of improving health. Previous research suggests that the mechanism for increased molting in response to disease is due to disease-induced upregulation of molting genes (Tarrant et al. 2012) and the molting hormone ecdysone (Laufer et al. 2005). Costs of molting while diseased have not been found in males; however, diseased females have a smaller molt increment than healthy females (DENC 2016). Further work is required to quantify additional physiological outcomes of diseased-induced molting on survival and fecundity. Recent models suggest that molting when diseased may decrease overall health over time as a result of accumulated physiological stress (Tlusty et al. 2014). If ESD advances to a severe state, which would be predicted with larger intermolt periods during the summer and fall, lobsters may have an increased risk of death during molting because severe lesions can adhere to the developing cuticle of the new instar, preventing completion of the molt (Stevens 2009). Although we did not analyze ovigerous females, using the same data set Hoenig et al. (2017) found that prevalence was highest in this group, presumably because they only molt every 2 years. This raises concerns that disease-related mortality in this critical life stage may be reducing overall fecundity in this

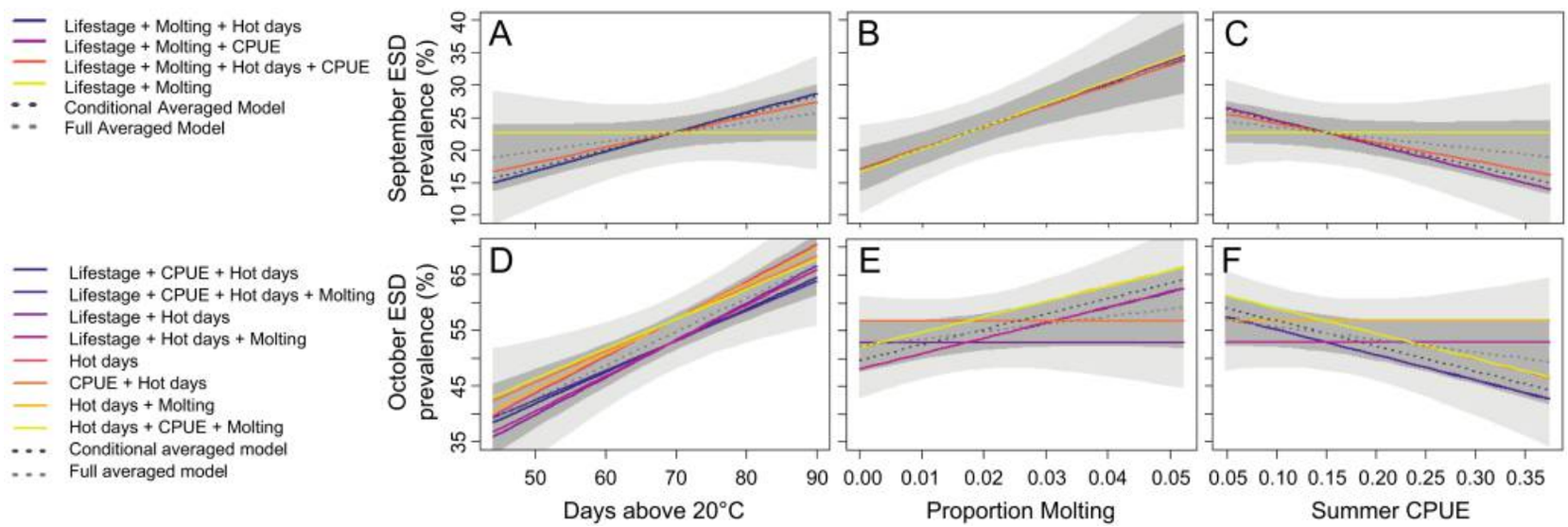

Figure 5: In September, the prevalence of epizootic shell disease (ESD) in juveniles is strongly correlated with molting phenology such that earlier molting in the spring results in higher prevalence. Summer temperature and catch per unit effort (CPUE) are weakly correlated with September prevalence of ESD. In October, the prevalence of ESD is more strongly correlated with hotter summers, in particular the number of summer days exceeding 20°C, while molting phenology and CPUE are weakly correlated with ESD prevalence. Multiple models were fit to September and October prevalence data. All models with corrected Akaike information criterion <2 as well as the conditional and full averaged models are displayed. Light shading indicates 2 SDs from the full averaged model, and dark shading indicates 1 SD from the full averaged model.

population, and this is reflected in poor recruitment indices (Wahle et al. 2009).

While population density is frequently correlated with disease transmission, we found little support for any effect of population density on ESD. Our analyses showed weak negative correlations between summer CPUE and September and October disease prevalence in juvenile and adult male lobsters. The most probable explanation for this pattern is that increasing summer temperatures are correlated with both increased disease rates and migration of lobsters to cooler waters. Indeed, the number of summer days above 20°C and CPUE was correlated in warm years but not cool years. The lack of an effect of population density is typical of opportunistic disease such as ESD, where changes to the host immune system as opposed to density-dependent transmission are hypothesized to regulate disease prevalence (Burge et al. 2013).

Decreasing population size in the southern New England lobster stock is a major concern (Howell 2012; Boudreau et al. 2015; Wahle et al. 2015). Recruitment has been low since 1998, and natural mortality rates have increased over time (Castro and Somers 2012; Howell 2012; Hoenig et al. 2017). Seasonal outbreaks of ESD and temperature stress are considered the main drivers of these losses (Howell 2012; Shields 2013). Long Island Sound has been warming at ~0.4°C per decade over the past 4 decades, and these trends are predicted to continue (e.g., Maynard et al. 2016). Recently, increases in the prevalence of ESD in the largest lobster stock in the United States, the Gulf of Maine stock, has raised concerns that the disease is expanding northward (Steneck et al. 2011; Reardon et al. 2018). Summer temperatures in the Gulf of Maine are forecasted to reach levels conducive to ESD within the next few decades, under business as usual climate projections (Maynard et al. 2016). Our research suggests that the effect of ESD on this fishery largely depends on future spring and summer seawater temperatures. If warming temperatures will lead to less molting during the summer (when lobsters are most susceptible to ESD) and more molting in the spring, then disease may increase substantially. Notably over the past 14 years, ESD prevalence in the Gulf of Maine has increased by nearly two orders of magnitude, from <0.01% to 0.7%, with focal levels ~5.0% (Reardon et al. 2018). Future research evaluating the role of temperature, molting phenology, and ESD will be important for understanding the spread of this disease into new regions.

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American lobsters (*Homarus americanus*) caught in Long Island Sound. Pitting and erosion on the carapace of the lobster on the left is indicative of epizootic shell disease. The presence of white barnacles indicates that this lobster has not molted recently. An orange tag, used for mark-recapture, is visible on the healthier lobster on the right. Photo credit: John Swenarton.