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Effect of host background and environmental conditions on disease susceptibility and mortality in the northern bay scallop, *Argopecten irradians irradians*

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Since 2019, outbreaks of an apicomplexan parasite caused catastrophic mortalities of adult northern bay scallops (*Argopecten irradians irradians*) in New York, USA. Anecdotal observations suggested different levels of mortality between different scallop stocks and mortality outbreaks appeared to occur in years displaying positive temperature anomalies. In this study, field and laboratory experiments were designed to evaluate the effect of environmental conditions and host (scallop) background on disease susceptibility and mortality. Wild and aquacultured scallops originally sourced from the same area were deployed in lantern nets (suspended in the water column) or oyster culture bags (on bottom) at two different enzootic sites showing contrasting environmental conditions. Subsets of wild and aquacultured stocks were also used in laboratory experiments exposing scallops to different temperatures and dissolved oxygen levels, mimicking different environmental scenarios. Results showed higher mortality in aquacultured scallops compared to wild scallops, especially in the more stressful conditions. Laboratory experiments showed that the trajectories of disease development and mortality were significantly affected by initial disease levels and environmental factors. For instance, high temperature and low dissolved oxygen were shown to favor disease development when initial disease levels were low (wild scallops) but also appear to lead to an overall reduction in disease via culling of the most heavily infected scallops when initial disease levels and mortality during the experiment were high (aquacultured scallops). Altogether, these findings underline the impact of environmental conditions, in particular temperature, on host-parasite interactions and suggest the potential existence of more resilient scallop stocks.

Keywords Mollusk, Disease, Apicomplexa, Marosporida, Climate, Temperature, Bivalve

The northern bay scallop, *Argopecten irradians irradians*, is an iconic resource in New York, where it serves as the official state shell. It used to support a multi-million-dollar fishery industry in the state until 2018. Since 2019, catastrophic and recurrent mortality of adult scallops have occurred annually throughout the Peconic Estuary (east end of Long Island, New York), decimating the population and the commercial fishery; this collapse led the United States Department of Commerce to declare the bay scallop in the Peconic Estuary a fishery disaster[1]. These mortality events were systematically associated with annual outbreaks of an undescribed parasite that disrupts the tissues of infected animals. Genetic analysis showed that the parasite belongs to a newly defined class of the *Apicomplexa*, the *Marosporida*[2]. The parasite, provisionally dubbed bay scallop Marosporida (BSM), has a tropism for kidney tissues, but is also capable of colonizing and altering a broad range of scallop tissues including the adductor muscle, gonad and gills[2]. Disease prevalence and intensity have a clear seasonal signature and are strongly correlated with scallop mortality trends. The parasite appears to be acquired by juveniles during their first summer but at low intensity, and the disease worsens dramatically during the following summer with significant increase in parasite numbers and activity (cell division), disrupting scallop tissues and decimating

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adults before they enter the fishery that fall. Anecdotal field observations suggested possible differences in mortality trends between different scallop stocks, and scallop mortalities associated with BSM have been so far only reported in New York, while this scallop subspecies is thought to range from Massachusetts to New Jersey (also introduced to Maine, Canada and China for aquaculture)[3,4]. Evaluation of temperature trends showed that disease outbreaks and mortalities in NY were associated with warmer-than-normal temperatures, particularly in spring and early summer[2].

All members of the Apicomplexa are parasitic (excluding *Nephromyces* sp.), and members of the Marosporida (i.e., *Aggregata* sp., *Rhytidocystis* sp., *Margolisiella* sp., and *Merocystis* sp.) infect various molluscan species, including octopuses (Colunga-Ramirez et al., 2025), whelks[5] and scallops[6–8]. Previous studies in other scallop species attempted to link mortality outbreaks to increasing water temperatures, but as elegantly formulated by Kristmundsson[9], "*these conclusions are in many cases highly speculative*", underlining a lack of understanding of the impact of ecologically-relevant temperatures on scallop health especially in the context of apicomplexan diseases. Previous research in the Icelandic scallop (*Chlamys islandica*) showed that *Merocystis kathae* (Marosporida, Apicomplexa) can induce significant mortality although ecological drivers of these outbreaks remain obscure[10,5] and may include biotic (e.g., host density and genetic diversity) or abiotic factors (e.g., temperature), leading these authors to conclude that "*which factors could drive such a process are hard to currently ascertain*".

The influence of host genetic background on disease development is recognized in virtually all terrestrial and marine organisms[11–13]. In many bivalve species, disease resistance/tolerance in some individuals has served to develop selective breeding programs to mitigate viral[14], bacterial[15] and protozoan[16,17] pathogens. In parallel, changes in disease dynamics (e.g., prevalence, intensity, geographic distribution) are directly related to environmental conditions in many marine organisms. Factors such as rising ocean temperatures, hypoxia, acidification and pollution can compromise host immune systems, enhance pathogen virulence, and create favorable conditions for disease transmission and development. For example, elevated sea temperatures have been linked to increased outbreaks of diseases in corals and shellfish[18,19]. Additionally, pollution and acidification can stress marine organisms, making them more susceptible to pathogens[20,21]. Furthermore, hypoxic conditions, often exacerbated by climate change, can impair immune responses in marine invertebrates, increasing their vulnerability to infections[22]. As climate change continues to alter marine environments, the frequency and severity of disease outbreaks in marine ecosystems are expected to rise, posing significant challenges to biodiversity and marine resource management.

In this study, complementary field experiments and laboratory studies were used to assess if and how host background and environmental conditions, including temperature and dissolved oxygen level, may influence disease development and mortality in bay scallops. Results highlight the impact of temperature on disease progress and mortality and support the potential existence of more resilient stocks. The findings are interpreted in the context of the very limited current knowledge on factors regulating apicomplexan infections in marine invertebrates.

Material and methods

Scallop collection and field experiment

Experimental protocols used in this study were reviewed and approved by Stony Brook University (NY, USA) in accordance with institutional, state, national, and international guidelines for the ethical use of animals in research. The experimental workflow (field deployments and laboratory experiments) is presented in Fig. 1A. Adult scallops from 2 genetic backgrounds (naturally-recruited wild scallops: 56.7 ± 4.6 mm in height; aquacultured, i.e., hatchery-produced, scallops: 54.5 ± 3.8 mm in height, produced by Cornell Cooperative Extension and maintained in longlines) were collected from Orient Harbor, NY (41.138078, -72.316327) on June 24, 2020. Each stock was divided into different groups and immediately re-deployed in four replicate lantern nets (0.5 m diameter, 1.2 cm mesh size, 5 layers, suspended in the water column) and ADPI bags (91.5 cm x 40.5 cm x 11.5 cm rectangular cages having a 1 cm square mesh; deployed on the bottom) in Flanders Bay (40.923889, −72.616111) and in Orient Harbor, adjacent to the site of wild scallop collections (Fig. 1B). These two locations were chosen because they depict different environmental conditions; as Flanders Bay is confined at the west end of the Peconic estuary, it experiences lower dissolved oxygen (DO) and higher temperature levels compared to Orient Harbor, which is located closer to the open sea. Temperature and DO at each site were recorded hourly using loggers (miniDOT, Precision Measurement Engineering, Inc., Vista, California, USA) attached to one bag and one lantern net at each site. For logistical reasons related to the COVID-19 pandemic, the loggers were deployed 1 month after scallop deployment. The last data retrieval was at the end of the field experiment (October 2020) except for the logger attached to the lantern net in Flanders Bay (environmental data collection stopped on 8/28/2020 as the logger was lost due to a storm after that sampling). An initial sample of 30 scallops from each group (wild and aquacultured) was collected before deployment (Time 0 on 6/24/20) and a minimum of 20 live scallops were sampled from each group/site/gear on 7/29/20 and 8/17/20 to evaluate BSM prevalence and intensity. Pathology samples from scallops deployed in lantern nets in Orient Harbor were also collected on 9/15/20 and 10/19/20. Mortality for all groups was recorded at the end of the field experiment (10/19/20).

Laboratory experiments

Temperature experiment

In parallel to field deployment, subsets of the same wild and aquacultured scallops collected on June 24, 2020, were also used in a laboratory experiment at the Stony Brook Southampton Marine Station (Fig. 1B) where they were exposed to three temperature regimes. Scallops were acclimated for one week before divided into nine replicate tanks (25 scallops per tank) placed in water baths held under conditions similar to initial field conditions (22°C, salinity of 28). Each replicate tank received a constant flow of natural seawater (~50L/hr). At that point, temperature in each water bath was gradually increased to one of three temperature treatments (three

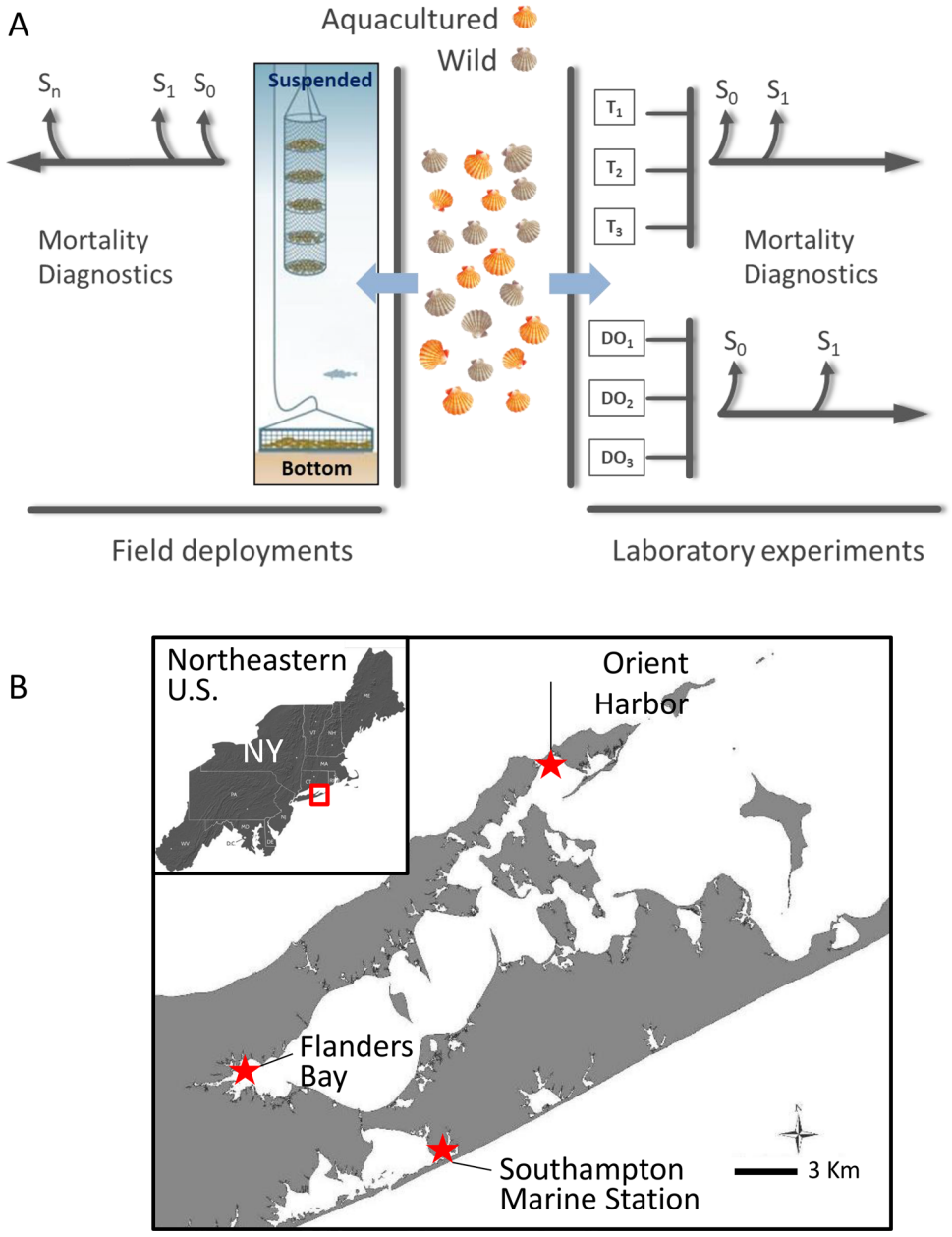

Fig. 1. (**A**) Schematic representation of the experimental workflow and (**B**) maps showing the locations of the deployment sites in Orient Harbor and Flanders Bay, and the Stony Brook Southampton Marine Station where the laboratory experiments were performed. Different samplings were made (represented by S_0 to S_n) throughout the experiments which were performed between June and October 2020. Map generated in R version 4.3.0 using the "rnaturalearth" package version 1.2.0 (https://cran.r-project.org/web/packages/rnaturalearth/index.html).

replicate tanks per treatment): 22°C (no change, control), 25°C (~1°C every 4 days) and 28°C (~1°C every 2 days). These temperatures were selected as they represent early to mid-summer conditions in various areas of the bay. Temperature manipulation and maintenance were performed using 200W titanium fish tank heaters and TECO aquarium chillers (TK-2000) connected to Inkbird aquarium controllers (ITC-306T). Target temperatures were maintained for 1 month. Mortality was recorded twice a day, and dead animals were immediately removed from the tanks. Live scallops (10 from each replicate tank, totaling 30 per group per treatment) were sampled at the end of the experiments to assess disease development.

Dissolved oxygen experiment

A second laboratory experiment was performed to evaluate the effect of DO on BSM development and mortality in wild and aquacultured scallops. Adult wild (56.1 ± 1.7 mm in height) and aquacultured (52.0 ± 3.9 mm) scallops were collected from Orient Harbor (same site as above) and submitted to three target DO levels (2, 4 and

8 mg/L; three replicate tanks per level, 12 scallops per group per tank). These levels represent the typical range of DO levels across scallop habitats in the Peconic Estuary. The 8 mg/L condition was achieved by bubbling ambient air into each replicate tank. The reduced DO conditions (4 and 2 mg/L) were attained by bubbling mixtures of pure N_2 and air via the use of gas proportioners (Cole Parmer® Flowmeter system, multitube frame, Vernon Hills, IL, USA) adjusted to reach the desired oxygen levels. Temperature was adjusted to 25°C by maintaining experimental tanks in water baths fitted with heaters and chillers connected to electronic controllers as described above. Temperature and DO were monitored twice a day using a YSI meter. Water in experimental tanks was renewed continuously as for the temperature experiment although the flow was reduced to ~12L/hr to enable better control of DO levels, and the tanks were fitted with biological filter units to help remove biological waste. Because the flow-through rate of natural seawater was relatively low, experimental scallops were supplemented with algae paste daily (LPB frozen shellfish diet, Reed Mariculture, Campbell, CA, USA). The experiment was run for 54 days, and scallop monitoring and sampling followed the procedures described for the temperature experiment.

BSM prevalence and intensity

BSM prevalence and intensity were assessed using fresh kidney preparations following the protocol described by Pales Espinosa et al.[2]. Randomly collected scallops from field and laboratory experiments were cleaned, measured and dissected for evaluation. A piece of the kidney (~100mg) was collected, placed in 24 well microplates with 1 ml sterile seawater and crushed using a sterile plastic pellet pestle. The fresh preparation was then observed using an inverted microscope (the whole well screened using a 10x objective couple with a 10x eyepiece). Parasite intensity was ranked on a scale ranging from 0 to 3 (BSM score, 0.5 incrementation) based on the relative abundance of BSM cells[2].

Data analysis

Scallop survivorship data from the field experiment (collected at the end of the experiment) was assessed using a Cochran-Mantel-Haenszel test. Survivorship data from the lab experiments (collected daily) were evaluated using a Kaplan-Meier survival analysis in conjunction with log-rank comparisons. Disease intensity data was evaluated using Mann-Whitney rank sum tests (two-sample comparisons) or ANOVA on ranks (for multiple sample comparisons) followed by Dunn's post-hoc test when appropriate. All statistical analyses were performed in SPSS version 28 and differences were considered significant at $p<0.05$.

Results

Field experiment

Temperature and dissolved oxygen

Temperature and DO levels in each site are presented in Supplementary data 1. The logger attached to the lantern net in Flanders Bay was lost in late August and hence the full data series in that site is only available from the logger attached to the ADPI bags. The average temperatures in ADPI bags and lantern nets in Orient Harbor and ADPI bags in Flanders Bay were 22.0 ± 3.1°C (range: 15.4–27.5°C), 22.0 ± 3.2°C (15.5–27.7°C) and 22.4 ± 3.9°C (15.2–29.1°C), respectively. Paired comparisons (ΔT) between the locations were also assessed (Fig. 2). Seawater temperature (ADPI) was markedly higher in Flanders Bay as compared to Orient Harbor ADPI throughout late July and August ($\Delta T > 0$ in 96.4% of the time points; $\Delta T > 2$°C in 10.8% of the time points). This trend was reversed after mid-September (09/14/20) as water temperature was generally lower in Flanders Bay compared to Orient Harbor ($\Delta T < 0$ in 75.2% of the time points; $\Delta T < -1$°C in 6.8% of the time points). At Orient Harbor, temperature was generally higher in the lantern nets compared to the ADPI bags (65.6% of the experiment timeframe; Fig. 2). In contrast, temperatures measured in Flanders Bay between late July and late August did not show any consistent differences between lantern nets and ADPI bags. Overall, temperature recorded in ADPI bags and lantern nets in Flanders Bay showed about 19% and 8% more time points with a temperature above 24°C compared to the two corresponding series of temperature data measured at the Orient Harbor site (Supplementary data 2). Differences between sites were more obvious at higher temperature thresholds with Flanders Bay presenting 492% and 207% more time points with a temperature greater than 26°C compared to the data sets collected from Orient Harbor ADPI bags and lantern nets, respectively.

In parallel, the average DO in Orient Harbor (ADPI bags), Orient Harbor (lantern nets) and Flanders Bay (ADPI bags) were 7.0 ± 1.0 mg/L (range: 2.8–11.5 mg/L), 7.1 ± 0.9 mg/L (4.1–11.7 mg/L) and 6.7 ± 1.2 mg/L (2.5–10.6 mg/L), respectively (Supplementary data 1). Paired comparisons (ΔDO) between the data series showed that differences in DO fluctuated markedly (likely as a result of photosynthesis and respiration), making it difficult to characterize one site as compared to another (Fig. 3). Nevertheless, Flanders Bay (ADPI bags) generally displayed lower DO levels compared to Orient Harbor (ADPI bags). Overall, only the ADPI treatments represented hypoxia conditions, with DO levels lower than 4 mg/L (2.1% and 3.6% of the data points for Orient Harbor and Flanders Bay, respectively; Fig. 3 and Supplementary data 2).

Scallop mortality

Scallop genetic lineage (wild vs. aquaculture), deployment method (ADPI bags vs. lantern nets) and deployment site (Orient Harbor vs. Flanders Bay) all had significant impacts on mortality. For instance, mortality was significantly higher among aquacultured scallops (range: 43.0% to 90.1%) compared to wild scallops (5.5% to 36.3%), regardless of the site and the deployment gear (Fig. 4). Similarly, mortality was significantly higher for scallops deployed in ADPI bags in Flanders Bay (90.1% and 36.3% for aquacultured and wild scallops, respectively) compared to those deployed at the same site in lantern nets (61.7% and 11.5% for aquacultured and wild scallops, respectively). No significant differences were detected in survivorship between deployment methods for scallops deployed in Orient Harbor (Fig. 4). Finally, scallops deployed in Flanders Bay systemically

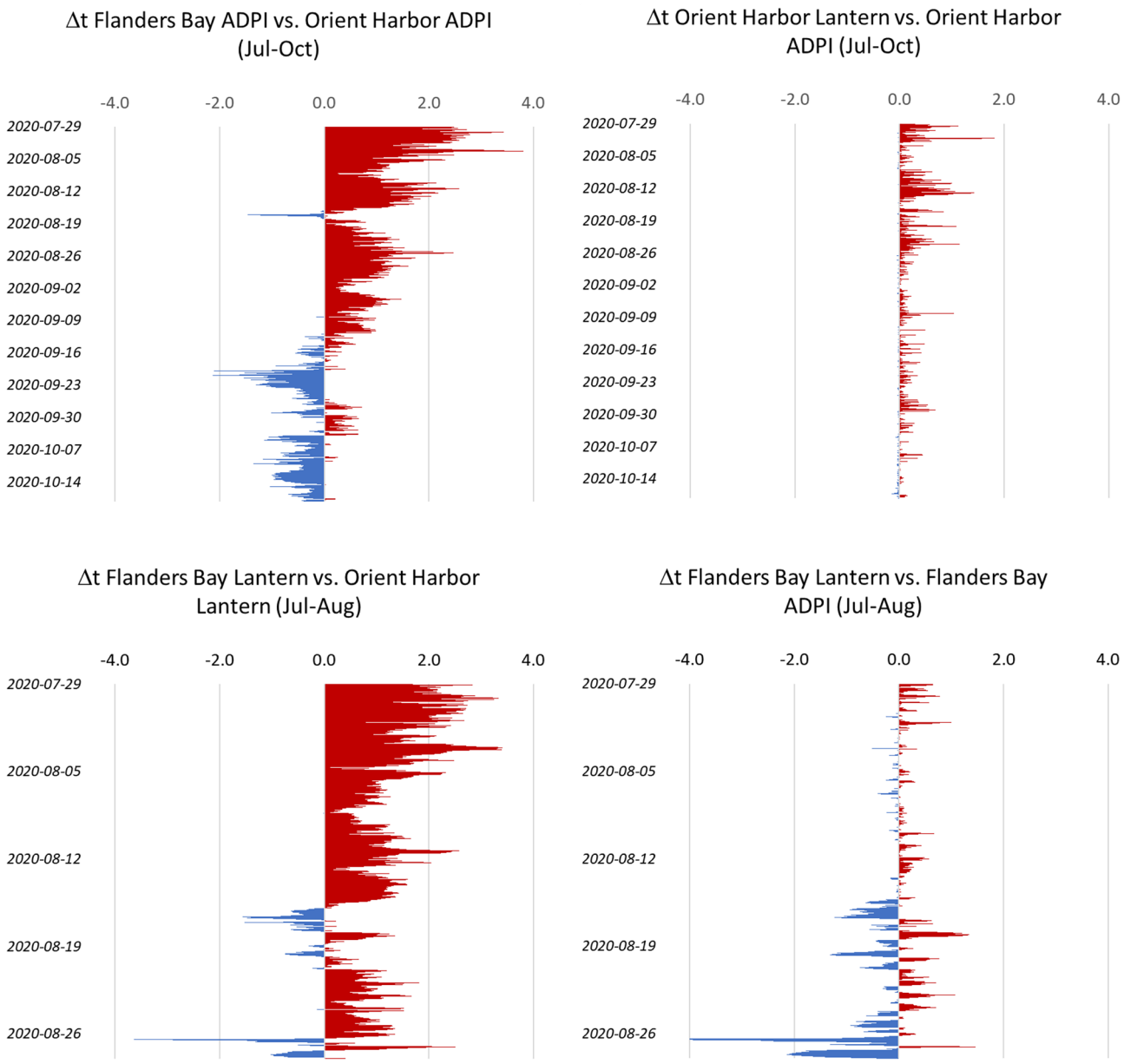

Fig. 2. Differential temperature (ΔT, °C) between Flanders Bay and Orient Harbor for the two deployment methods (lantern nets and ADPI bags) at each site. Positive values indicate that the first member of the pair had higher levels than the second member (and vice versa) for that given date.

displayed higher mortality than their counterparts (same scallop lineage, same gear) held in Orient Harbor, although differences were statistically different only for scallops held in ADPI bags.

Parasite levels

At the beginning of the field experiment (T0, 06/24/2020), BSM scores in wild scallops ranged from 0.5 (very light infection) to 2 (moderate infection), averaging 1.40 ± 0.42 (mean ± SD, Fig. 5), with an overall prevalence of 100%. In contrast, BSM scores in the aquacultured scallops ranged from 1 (light infection) to 3 (severe infection), averaging 2.12 ± 0.47, but also with an overall prevalence of 100%. Statistical analysis showed that BSM scores at T0 were significantly lower in wild scallops compared to the aquacultured cohort (Mann-Whitney rank sum test, $p<0.001$).

BSM scores in wild scallops deployed in Flanders Bay using ADPI bags remained relatively stable through July (~1 month of deployment, 1.50 ± 0.67) and August (1.23 ± 0.38, ANOVA on ranks, $p > 0.05$; Fig. 5). In the lantern nets, however, BSM scores were significantly lower in August (0.95 ± 0.36; Mann-Whitney rank sum test) compared to T0. Due to logistical constraints, no disease sampling was performed in July for that group).

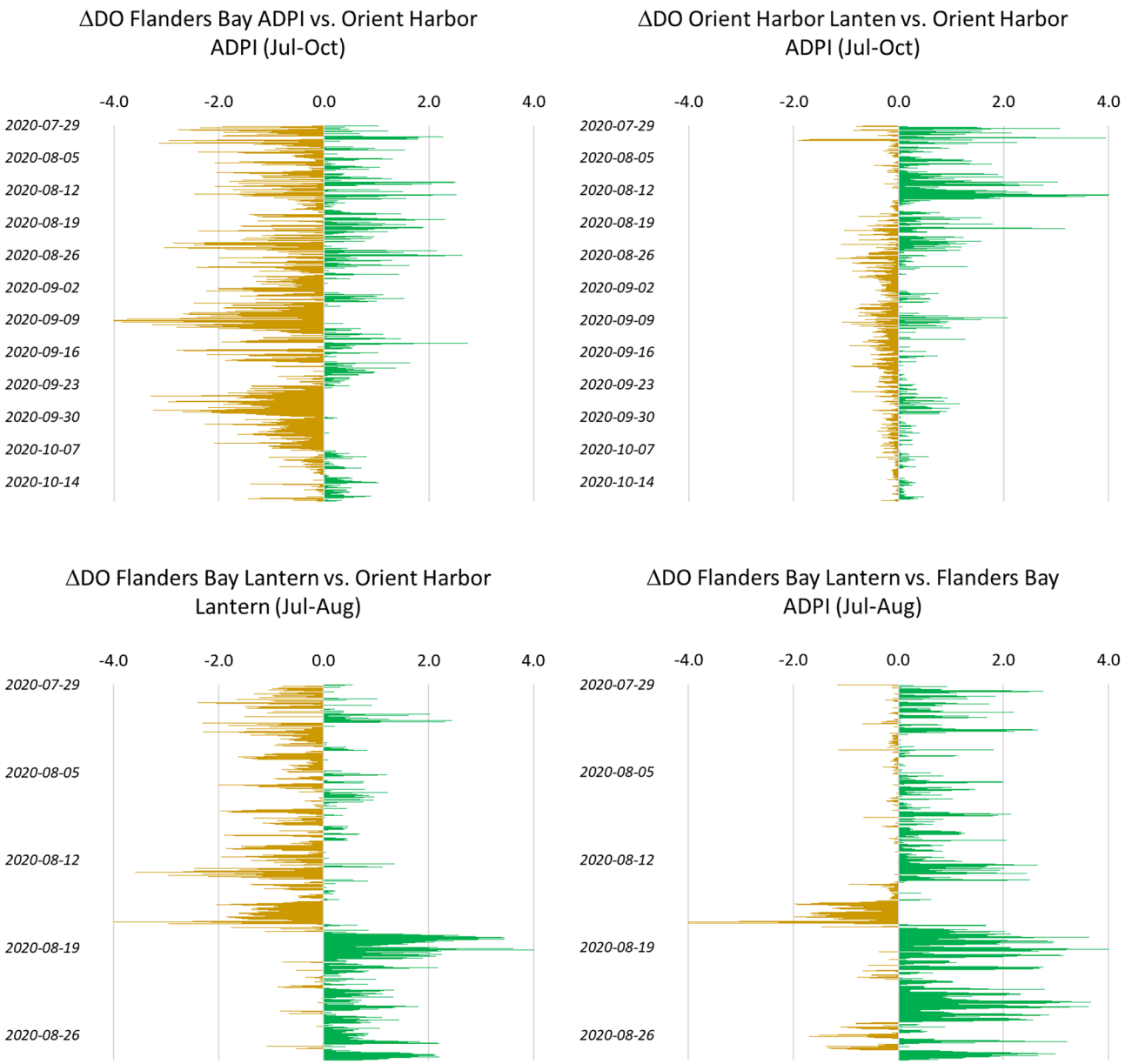

Fig. 3. Differential dissolved oxygen (ΔDO, mg/L) between Flanders Bay and Orient Harbor for the two deployment methods (lantern nets and ADPI bags) at each site. Positive values indicate that the first member of the pair had higher levels than the second member (and vice versa) for that given date.

For wild scallops deployed in Orient Harbor in ADPI bags, BSM scores were significantly higher in July (2.35 ± 0.29, Fig. 5) compared to T0 (1.40 ± 0.42), before slightly decreasing in the August sampling (1.83 ± 0.65; no difference between August and either one of the two prior samplings). In parallel, BSM scores of wild scallops deployed in lantern nets were significantly higher in July (2.20 ± 0.73) and August (2.08 ± 0.63) respectively) compared to T0, before abating during the September (1.40 ± 0.74) and October (1.79 ± 0.61) samplings.

BSM scores in aquacultured scallops deployed in Flanders Bay in ADPI bags were significantly lower in July (1.23 ± 0.30) and August (1.21 ± 0.64) compared to T0 (2.12 ± 0.47; Fig. 5). Similar trends were observed in the lantern nets, although at a slower pace as BSM scores reached 1.74 ± 0.79 in July before decreasing significantly in the August sampling (0.76 ± 0.45).

For aquacultured scallops deployed in Orient Harbor in ADPI bags, BSM scores were slightly lower in July (1.91 ± 0.90, Fig. 5) compared to T0. After 2 months, BSM scores further decreased (1.65 ± 0.49) and differences with those at T0 became significant. In the lantern nets, BSM scores were relatively stable between June and August (2.32 ± 0.65), before decreasing during the September sampling (1.70 ± 0.55; significant difference between the August and September samplings).

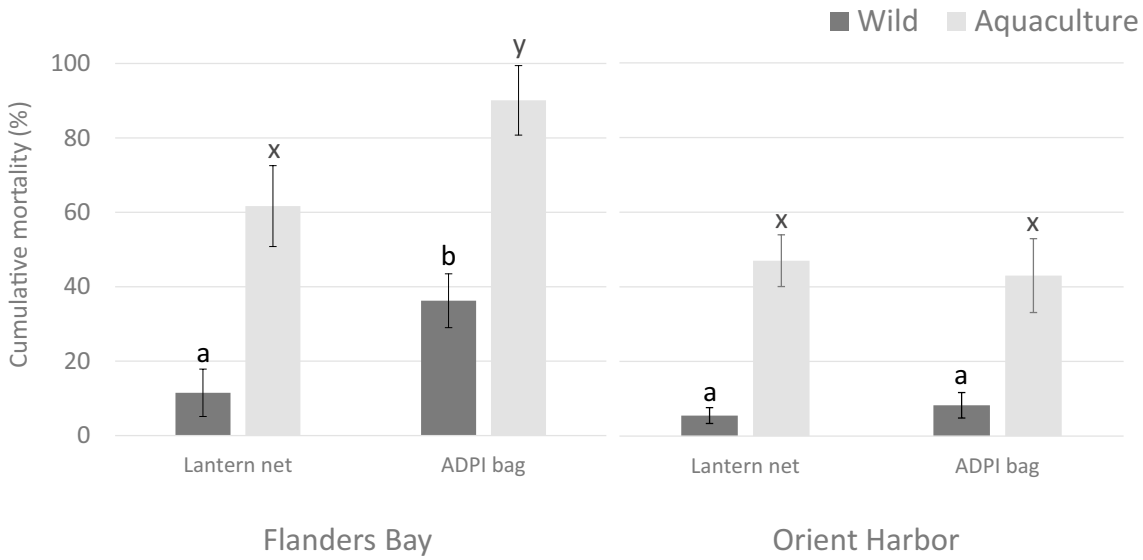

Fig. 4. Cumulative mortality (%) of scallops deployed at each site. Letters indicate significant differences in mortality between sites and deployment methods within each scallop lineage (wild or aquacultured; Cochran-Mantel-Haenszel test, p<0.05).

Laboratory experiments

Temperature experiment

Temperature level significantly affected mortality of infected scallops. After 1 month of exposure, the cumulative mortality of aquacultured scallops reached 47.4%, 43.9% and 89.5%, when exposed to 22°C, 25°C and 28°C, respectively. Death probability was significantly higher in scallops maintained at 28°C compared to the two other treatments (log-rank test, p<0.00001, Fig. 6). The cumulative mortality of wild scallops reached 14.0%, 12.3% and 52.6%, when exposed to 22°C, 25°C and 28°C, respectively, and the death probability was also significantly higher at 28°C compared to the two other treatments (log-rank test, p<0.003, Fig. 6). Statistical comparison between the strains showed higher death probability among aquacultured than wild scallops for each of the three temperature treatments (log-rank test, p<0.007).

BSM scores in fresh kidney preparations from aquacultured scallops tended to decrease with increasing temperatures but were not statistically different from those held for 1 month at 22°C (2.28 ± 0.83), 25°C (1.89 ± 0.68) or 28°C (1.75 ± 0.69), or from those evaluated at T0 (2.12 ± 0.47, Fig. 6; ANOVA on ranks, p = 0.08). In wild scallops, BSM scores generally increased over the 1-month experiment period for scallops held at all three temperatures compared to T0 (1.40 ± 0.42). In contrast to aquacultured scallops, BSM scores in wild scallops tended to be higher at higher temperatures, reaching 1.64 ± 0.72 at 22°C, 2.00 ± 0.91 at 25°C, and 2.16 ± 0.69 at 28°C; however, differences were significant only when comparing T0 and 28°C (Dunn's post-hoc test, p<0.05).

Dissolved oxygen experiment

During the entire course of the experiment, the average water temperature was 24.7 ± 1.1°C (Supplementary data 3). The water temperature was higher than 26°C from 08/11/21 to 08/18/21 (end of the experiment). The targeted dissolved oxygen levels of 2, 4 and 8 mg/L averaged 2.7 ± 0.9 mg/L, 4.7 ± 0.9 mg/L and 7.5 ± 0.7 mg/L, respectively. Survivorship of aquacultured and wild scallops remained high (>95%) for the largest fraction of the experiment, including for the hypoxia treatment, and mortality only started to increase around day 45. After 54 days (08/18/21), the cumulative mortality among aquacultured scallops reached 61.1%, 27.8% and 8.3% for the 2, 4 and 8 mg/L treatments, respectively. In parallel, the cumulative mortality reached 61.1%, 25.0% and 8.3% in wild scallops from the 2, 4 and 8 mg/L treatments, respectively. Statistical analysis showed no difference between aquacultured and wild scallops. In contrast, within each strain, mortality was significantly higher in the 2 mg/L treatment compared to the two other oxygen levels (Fig. 7; log-rank test, p<0.05).

BSM scores in kidney fresh preparations from aquacultured scallops averaged 2.05 ± 0.69 at T0, with 100% prevalence (Fig. 7). No significant changes were noted after 54 days in the 2 mg/L (2.35 ± 1.00) and 4 mg/L (2.45 ± 0.51; ANOVA on rank, p>0.05) treatments. However, scallops held in the 8 mg/L treatment (2.75 ± 0.55) yielded significantly higher BSM scores than the other two treatments and T0 scallops (Dunn's post-hoc test, p<0.05). In wild scallops, BSM was not detected in the kidney fresh preparations at T0 (BSM score = 0) and significant increases in BSM scores were noted for all treatments (Dunn's test, p<0.05, Fig. 7). These were 1.44 ± 0.73, 1.16 ± 0.59 and 1.20 ± 0.36 for the 2 mg/L, 4 mg/L and 8 mg/L treatments, respectively.

Discussion

Results from field and laboratory experiments showed that the trajectories of disease development and mortality of *A. irradians irradians* are significantly affected by scallop background and environmental factors. For instance, a main finding of the study was the remarkable difference in initial disease levels between aquacultured and

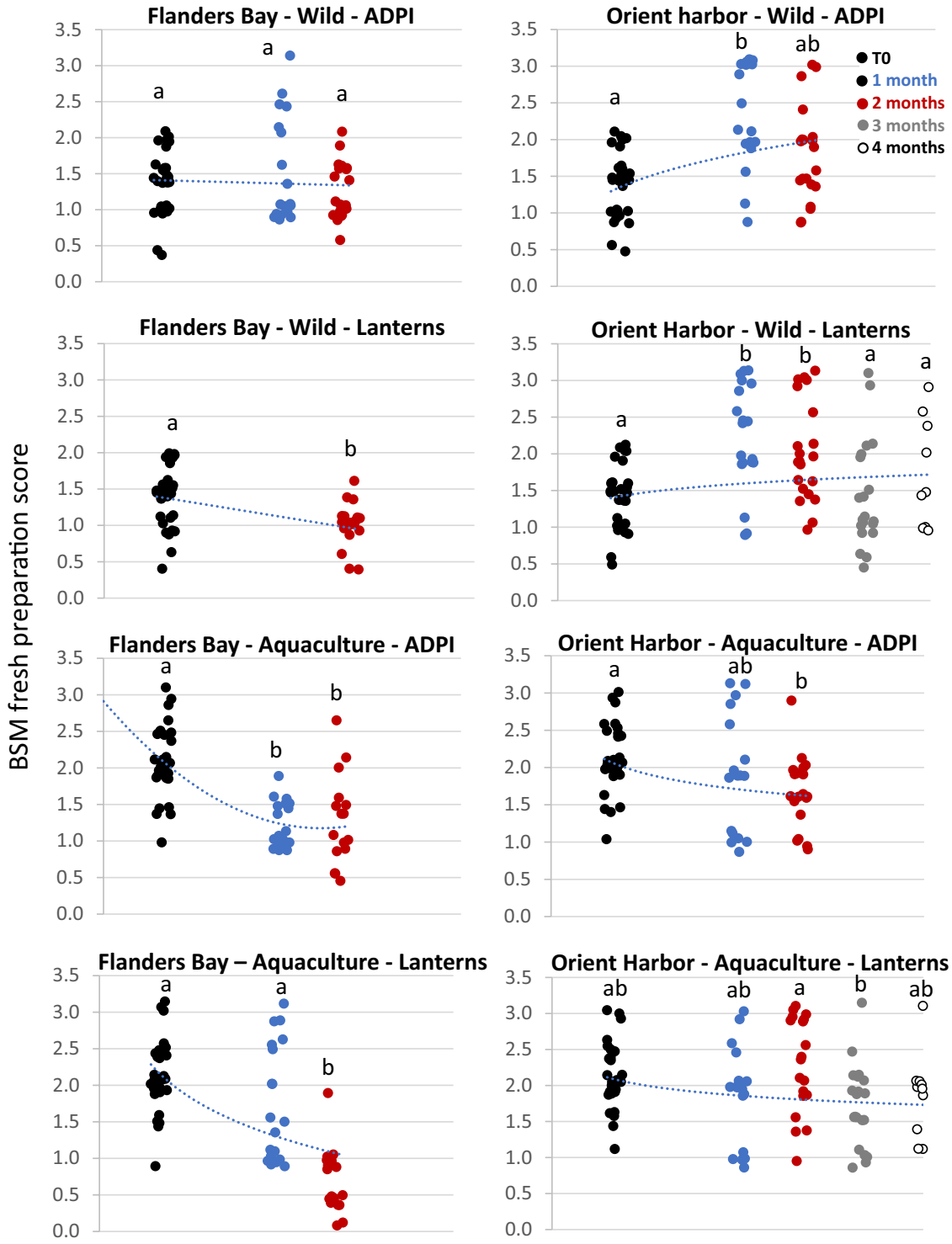

Fig. 5. BSM scores (infection intensity) in scallops (wild and aquacultured) deployed in Flanders Bay or Orient Harbor using ADPI bags or lantern nets between June and October 2020. For each plot, letters indicate significant differences between the sampling dates (ANOVA on ranks followed by Dunn's test, $p<0.05$).

wild scallops (50% higher BSM scores in aquacultured scallops at T0, Mann-Whitney rank sum test, $p<0.001$; Fig. 5) and subsequent survivorship in the field (540% higher mortality in aquacultured scallops on average with a range of 250% to 850% depending on site and deployment gear; Fig. 4). These results strongly suggest that initial disease levels represent the most important driver of mortality although the causes of this initial difference in disease intensity between both stocks remain unclear. Both aquacultured and wild scallops were collected from Orient Harbor and were therefore submitted to the same overall oceanographic conditions

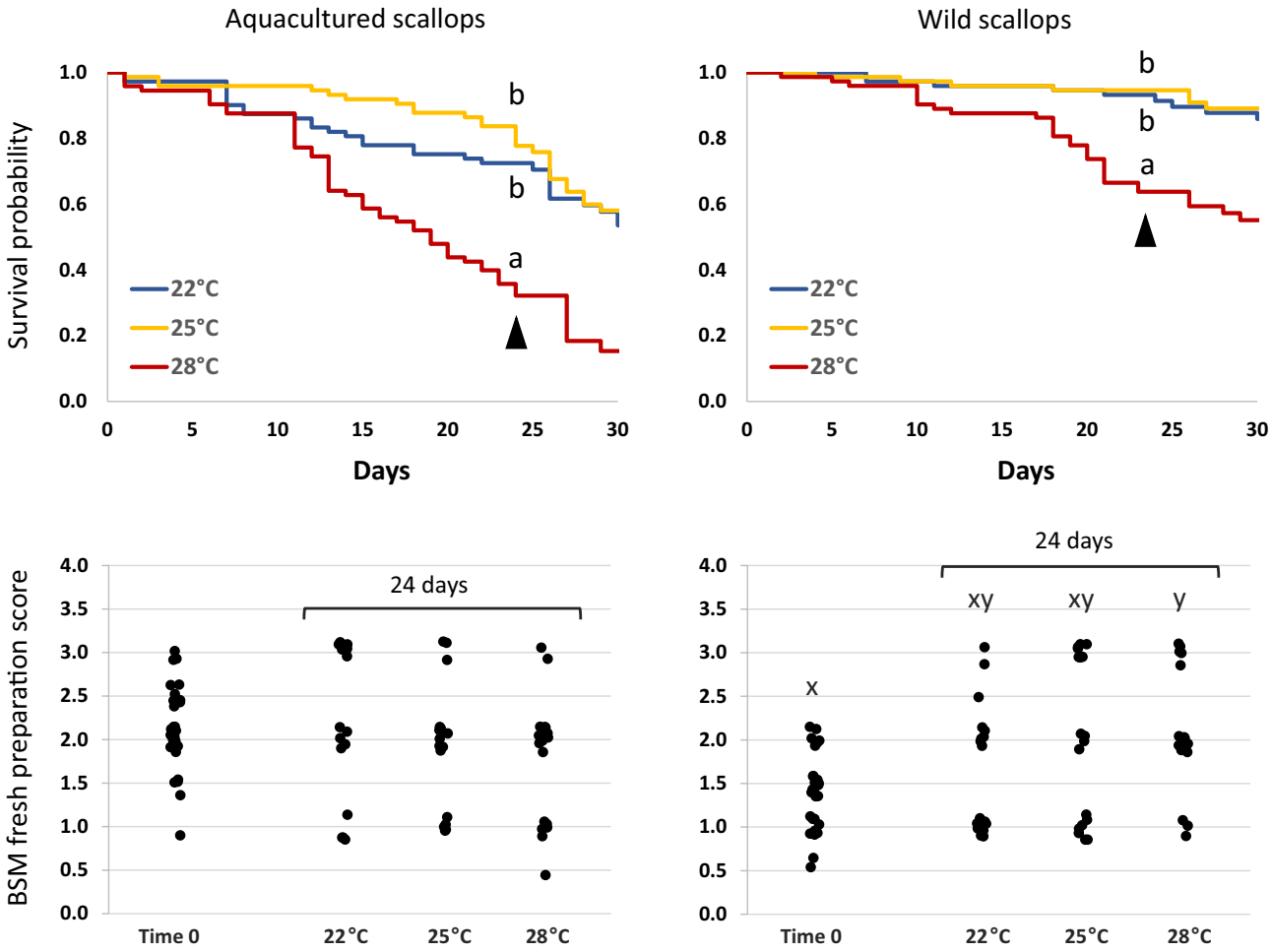

Fig. 6. Survival probability (Kaplan-Meier estimates, top) and BSM scores (bottom) in wild and aquacultured scallops exposed to different temperatures. The arrowheads indicate the sampling time point for kidney collection and BSM scoring. For each plot, different letters indicate significant differences between treatments (a and b: log-rank test, $p<0.003$; x and y: Dunn's post-hoc test, $p<0.01$).

even though environmental conditions in the specific collection sites were not exactly the same. For instance, while wild scallops were harvested from the bottom by diving, aquacultured scallops were maintained in lantern nets suspended in the water column. Temperature data from Orient Harbor during summer showed limited differences between suspended lantern nets and ADPI bags deployed on the bottom (Fig. 2), which is not surprising given that the harbor is relatively shallow (~4–7 m in depth) and well-mixed, prohibiting the establishment of a stable thermocline. In parallel, dissolved oxygen levels measured during summer were lower (i.e., more stressful) at the bottom (Fig. 3), which contradicts lower disease levels measured among wild scallops as one might expect higher disease levels in more stressful environments (although the opposite can also be true as discussed below). Nevertheless, no data is available about the contrast between environmental conditions in the water column and the bottom during the months that preceded scallop sampling, which may have affected disease development. Furthermore, it is also possible that potential differences in parasite abundance between the water column and the bottom (not assessed) may impact disease pressure and resulting scallop mortalities. Therefore, it cannot be ruled out that differences in environmental conditions between the water column and the bottom may have somewhat contributed to the difference in disease levels between aquacultured and wild scallops at T0.

Beyond prevailing environmental conditions, other important differences between aquacultured and wild scallops exist. For instance, aquacultured scallops are reared in confined environments and are stocked in high density (compared to wild scallops) throughout their entire life. Our preliminary data suggest that BSM is monoxenous, and higher stocking densities may facilitate disease transmission. In parallel, shellfish aquaculture conditions are associated with intended (e.g., fast growth) and unintended selection that may influence disease resistance or tolerance if immunologically weak individuals are inadvertently selected (i.e., uncontrolled genetic selection in the hatchery). For example[23] showed that aquaculture practices in the eastern oyster induce the selection of genetic markers that may be related to development and growth in a confined environment and that are highly specific, enabling the segregation of wild and aquacultured stocks along a broad geographic range spanning Maine to North Carolina. Alternatively, selection could also occur among wild scallops to eliminate

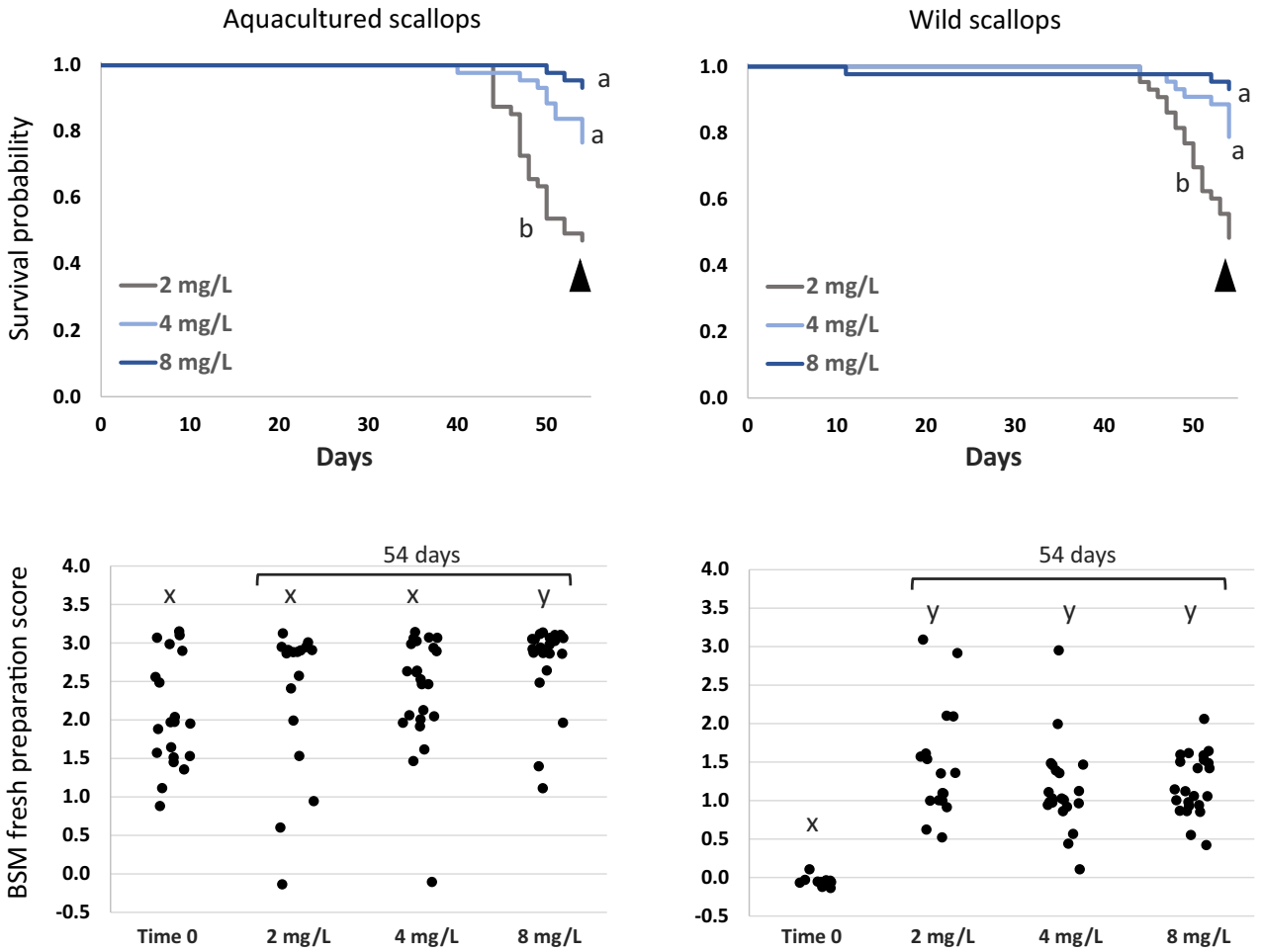

Fig. 7. Survival probability (Kaplan-Meier estimates, top) and BSM scores (bottom) in wild and aquacultured scallops exposed to different levels of dissolved oxygen. The arrowheads indicate the sampling time point for kidney collection and BSM scoring. For each plot, different letters indicate significant differences between treatments (a and b: log-rank test, $p<0.01$; x and y: Dunn's post-hoc test, $p<0.05$).

the weakest animals and favor the survivorship of the most robust individuals. For instance, dissolved oxygen was markedly lower at the bottom than in the water column and may have contributed to the elimination of the most sensitive (or, for that matter, the most heavily infected) individuals, leaving animals with the lowest disease levels to enter the study.

While these scenarios cannot be fully excluded, the most compelling cause of difference in performance between wild and aquacultured scallops is that of genetic differences between both groups as demonstrated by a companion study that deployed and monitored the same stocks in Flanders Bay for 4 months (June-October 2020)[24]. In that study, high-throughput single nucleotide polymorphism analysis was used to show that wild and aquacultured scallops were genetically distinct at the beginning of the study (T0), and that some genotypes that were present only among aquacultured stocks were gradually eliminated as summer progressed, leaving survivor scallops that were genetically similar to wild individuals[24]. Remarkably, specific shifts in allele frequencies were noted in genes associated with response to pathogens and kidney metabolism, highlighting the role of BSM as a selective pressure during these mortalities.

It should be noted that our definition of "wild" and "aquacultured" stocks does not necessarily imply restrictive genetic segregation as broodstocks used for aquaculture production of each generation are collected from the environment and aquacultured scallops are also annually released to the environment to enhance "wild" populations[25,26]. Therefore, strong genetic overlap between both stocks is expected but differences noted in the initial genetic composition[24] and field and lab performance (this study) are, nevertheless, remarkable and may be due in part to the breeding strategies used (until recently) for aquaculture. For instance, during the scallop production process, the genitors are usually collected in embayments where they are abundant and easy to find, suggesting "refuge area" characterized by low mortality pressure. Therefore, their progeny may inherit these characteristics and, when deployed in unfavorable areas, they succumb due to strong selective pressure exerted by disease and stressful summer conditions. The higher mortality observed in the most stressful environment (Flanders Bay as opposed to Orient Harbor) supports this scenario. The conclusions would not be surprising

since a genetic basis of resistance/resilience has been previously reported in many bivalve species against a broad range of biological[16,27–32] and environmental[33-35] stressors.

Mortality of infected scallops was also highly impacted by prevailing environmental factors, particularly temperature. Warm seawater temperature anomalies reported in the Peconic Estuary since 2019 have been previously suggested as a contributing factor to BSM epizootics and mortalities[2,36]. The impact of environmental conditions on the development and progression of diseases, particularly those induced by opportunistic facultative pathogens (e.g., many bacterial infections), is well known in marine invertebrates. For example, stressful environmental conditions (e.g., high temperature, low dissolved oxygen, or both) have been directly linked to outbreaks caused by vibrios in corals[37,38], crustaceans[39-42]), and mollusks[43–46]. In contrast, studies focusing on the impact of environmental conditions on members of the Apicomplexa in marine invertebrates are more rare and conclusions often derive from seasonal variations in disease prevalence instead of direct evidence from laboratory experiments[10,47]. Nevertheless, available evidence from well-studied non-apicomplexan alveolate pathogens (e.g., the case of *Perkinsus marinus* -a member of the Perkinsozoa, Alveolata- in oysters) show that obligate parasites have often evolved strategies to exploit host resources at much broader environmental ranges sometimes overlapping with host physiological optima. For instance, *P. marinus* can induce epizootics along the east coasts of North America from Mexico to Maritime Canada[48], even though prevalence and disease severity are still linked to prevailing environmental conditions. In this framework, investigations reported in this paper are not only timely for understanding the effect of environmental factors on BSM disease outbreaks but also fill a gap of information on the impact of host background and environmental factors on the development of diseases caused by members of the Apicomplexa in marine invertebrates. In the present work, mortality started to ramp up markedly in aquacultured scallops (and to a lesser extent among wild scallops) held at 28°C for 10 days as compared to those maintained at 25 or 22°C (Fig. 6). Field observations also support the role of temperature as a contributing factor to the mortality of infected scallops. For instance, scallops maintained in Flanders Bay systematically showed higher mortality levels than those held in Orient Harbor, in parallel to higher temperatures at the former site throughout the most stressful part of the summer (>2°C warmer temperature at the bottom in over 10% of the deployment period in July and August; Fig. 2). It should be stated that the 28°C applied here during the laboratory experiment is within the high range of what northern bay scallops can tolerate and is unlikely to explain alone observed mortalities (i.e., without BSM infection). For instance, juvenile and adult scallops are considered to be eurythermal, and their temperature tolerance ranges from as low as -6.6°C to 35°C for very short periods (a couple of hours[49–51]);. Their optimal growth temperature, however, hovers around 22/24°C depending on their geographic origin[50,52] and food availability[53–55] and 32°C seems to be a temperature that bay scallop can manage for a limited number of days[52].

However, field conditions are more complex than a laboratory setting and may imply other environmental stressors that can contribute to the mortality of infected scallops. For example, hypoxia is also a threat for marine organisms, particularly when associated with high water temperature. Although the laboratory experiment reported here showed a very limited impact of DO alone on disease dynamics and mortalities (excluding in scallops held in confinement for over 6 weeks), the role of low oxygen levels as a contributing factor to mortalities is possible, rather likely. This is supported by the lower mortality levels found in scallops held in the water column (lantern nets; where temperature is actually slightly higher) as compared to those maintained in ADPI bags where oxygen levels are generally lower. The DO level of 2 mg/L (hypoxia) targeted in this study is generally considered to be stressful to many marine species[56], even though this threshold is not homogenous across marine organisms. For example, Vaquer-Sunyer and Duarte[57] reported a mean LC50 (lethal concentration for 50% of the tested animals) for several common bivalves to be 1.42 mg/L, lower than that of other marine taxa. For example, the LC50 of the juvenile Atlantic surfclam *Spisula solidissima* and the clam *Macoma balthica* was estimated to be 0.5 mg/L[58] and 1.7 mg/L[59], respectively. Consequently, even if a dissolved oxygen level of 2 mg/L was stressful in the laboratory experiment, it did not cause any change in infection levels and scallops were able to survive several weeks before mortality occurred. Interestingly, scallop mortality accelerated at the end of the experiment, when the water temperature slightly increased (>26°C for 1 week). In the field, the highest mortality levels were noted in the ADPI bags deployed in Flanders Bay, which displayed the lowest oxygen and highest temperature levels among all investigated sites. The association of these two stressors likely exacerbated heavily infected scallops, leading to the mortalities reported here. Tomasetti et al.[36] reported the synergistic effect of elevated temperature (26°C and 29°C) and low oxygen level (~1.5 mg/L) which, when combined, were shown to induce heavy scallop mortality (disease was not evaluated in that study even though the experimental scallops originated from a heavily infected cohort). Nevertheless, field data reported here suggest that dissolved oxygen levels below 4 mg/L may be rarely reached in scallop habitats in the Peconic Estuary (Supplementary data 2).

Another finding of the present work was that the trajectory of disease development (and by extension, mortalities) directly depends on disease levels at T0 and prevailing environmental conditions. This was noticeable during the temperature experiment as disease intensity markedly worsened in wild scallops held at 28°C as compared to T0 (Fig. 6). In contrast, disease intensity in fact decreased in aquacultured scallops held at 28°C as compared to T0, although differences were not statistically significant. For the dissolved oxygen experiment, the parasite was not microscopically detectable in wild scallops at T0 and BSM scores increased significantly throughout the experiment with the highest disease development reported in scallops held at the lowest oxygen level. In contrast, aquacultured scallops, which started with relatively high BSM scores (2.05 ± 0.69) showed a significant increase in disease intensity only in the most favorable oxygen concentration (2.75 ± 0.55 for the 8 mg/L treatment, which displayed low mortality levels) while the other two treatments yielded more mild increases in BSM intensity but were associated with higher mortality levels. Finally, the field experiment also showed that disease intensity tended to increase through time for wild scallops which started with low intensities and showed low mortalities. In contrast, disease intensity decreased over time for aquacultured scallops, which started with high intensities and displayed high mortalities. These results further underline the

complex interconnectivity between disease levels, mortalities and the environment. Overall, changes in disease levels (i.e., prevalence and/or intensity) over time is the outcome of two opposing processes: (1) increase in disease levels as a result of the development of new cases and/or worsening of previous cases, and (2) a reduction of disease levels as a result of healing or death (i.e., culling) of (most severely) infected individuals. In other words, a selective mortality of most heavily infected scallops may reshape the infection profile of survivors. This is supported by our previous work showing that scallop mortality is directly related to tissue disruptions associated with heavy BSM infections[2]. In parallel, while healing of infected scallops cannot be ruled out, it is unlikely to explain the reduction in disease levels observed here as mortality levels were directly related to infection levels at the beginning of the experiments. In this framework, wild scallops used in this study represent a better cohort to specifically evaluate the role of environmental factors on disease development *per se*. It should be noted that the microscopy-based diagnostic technique used in this study is known to underestimate early infections (small stages of BSM - e.g., sporozoites[2]), hence animals described here to be void of parasite cells (e.g., wild scallops at T0 in the dissolved oxygen experiment) likely had low levels of infection. Overall, evidence reported here strongly suggests that heavily infected scallops are more likely to succumb to mortality and are culled from the population when exposed to stressful conditions as shown for aquacultured scallops used in the laboratory experiments or deployed in Flanders Bay. In contrast, low dissolved oxygen and high temperature conditions appear to favor disease progression in lightly infected scallops as shown in wild scallops used in lab experiments.

Conclusions

Environmental alterations associated with climate change have been linked to multiple outbreaks of infectious disease in terrestrial[60] and marine[61,62] systems. Positive temperature anomalies have been suggested to be linked to disease[2] and mortality[2,36] outbreaks impacting the bay scallop population in the Peconic Estuary. The current study showed high mortality among heavily infected scallops while lightly infected individuals survived long exposures to ecologically-relevant high temperatures and/or low dissolved oxygen. As important, disease levels and subsequent mortalities were different between wild and aquacultured scallops, and a recently published companion study[24] showed that some scallop genotypes in the Peconic Estuary survive the mortality outbreaks better than others. Collectively, these findings spark cautious optimism that the current mortality outbreaks could be managed by optimizing restoration effort via careful consideration of culture practices (e.g., better consideration of the genetic background of broodstock, health management of stocks, etc.) for resource restoration. In nature, the survival of a given host population is driven by its ability to access refuge areas characterized by low disease pressure (unfavorable conditions for parasite development) and/or the spread of genetically resistant individuals (GROUP, 2009). Of interest, breeding programs have been developed to improve resilience against biological and environmental stressors in many bivalve species[34,63–65] including scallops[33] and such strategies can be developed in bay scallops. Despite the lack of a controlled disease transmission protocol for BSM disease (and for most apicomplexan infections in marine organisms), this study shows that it is possible to address relevant questions pertaining to the impact of host genetic background and environmental conditions on disease progress and resulting mortalities. Nevertheless, a better understanding of the effect of temperature on the biology and virulence of the parasite is needed in order to unravel the complexity of host-parasite-environment interactions and develop comprehensive mitigation strategies.

Data availability

All data generated or analyzed during this study are included in this published article and its supplementary files.

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Author contributions

B.A., E.P.E and S.T. designed the study and secured the funding. S.T. and H.T. collected biological samples from the field and M.F.M. conducted laboratory experiments and helped with sample processing. B.A. and E.P.E. generated and analyzed data and drafted the paper. All authors contributed to the editing of the manuscript and approved the final version of the paper.

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Declarations

Competing interests

The authors declare no competing interests.

Ethics

All international, national, and institutional guidelines for organism sampling and experimental use were followed, with all necessary approvals obtained. *Argopecten irradians* is not on any list of species of concern.

Additional information

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