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Host behavior alters spiny lobster–viral disease dynamics: a simulation study

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Abstract. Social behavior confers numerous benefits to animals but also risks, among them an increase in the spread of pathogenic diseases. We examined the trade-off between risk of predation and disease transmission under different scenarios of host spatial structure and disease avoidance behavior using a spatially explicit, individual-based model of the host–pathogen interaction between juvenile Caribbean spiny lobster (*Panulirus argus*) and *Panulirus argus* Virus 1 (PaV1). Spiny lobsters are normally social but modify their behavior to avoid diseased conspecifics, a potentially effective means of reducing transmission but one rarely observed in the wild. We found that without lobster avoidance of diseased conspecifics, viral outbreaks grew in intensity and duration in simulations until the virus was maintained continuously at unrealistically high levels. However, when we invoked disease avoidance at empirically observed levels, the intensity and duration of outbreaks was reduced and the disease extirpated within five years. Increased lobster (host) spatial aggregation mimicking that which occurs when sponge shelters for lobsters are diminished by harmful algal blooms, did not significantly increase PaV1 transmission or persistence in lobster populations. On the contrary, behavioral aversion of diseased conspecifics effectively reduced viral prevalence, even when shelters were limited, which reduced shelter availability for all lobsters but increased predation, especially of infected lobsters. Therefore, avoidance of diseased conspecifics selects against transmission by contact, promotes alternative modes of transmission, and results in a more resilient host–pathogen system.

Key words: agent-based model; disease avoidance; disease transmission; harmful algal blooms; Panulirus argus *Virus 1; sociality*.

Introduction

Sociality brings with it certain biological costs associated with aggregation, including increased vulnerability of the host population to diseases (Alexander 1974, Møller et al. 1993, Altizer et al. 2003, 2011). For directly transmitted diseases, it is usually assumed that the contact rate of the social host is greater than that of the asocial host. Thus, under the mass-action assumption (sensu Anderson and May 1985) the force of infection should be higher in social organisms than in asocial organisms. Similarly, larger group sizes correlate with increased disease prevalence and intensity due to increased contact rates (Cote and Poulin 1995). However, host behavior can also act as a barrier to disease. For example, moribund ants, *Temnothorax unifasciatus*, infected with a communicable fungal pathogen (*Metarhizium anisopliae*) leave their nests, thereby reducing the risk of disease within the colony (Heinze and Walter 2010). Similarly, in the eusocial honey bee, *Apis mellifera*, experimentally sickened individuals remove themselves from the colony and do not return, resulting in altruistic suicide (Rueppell et al. 2010). Most studies have focused on changes in the behavior of infected hosts. But research on humans and a few other social taxa suggest that the behavior of *uninfected* hosts can reduce the transmission of disease.

Whereas a host's immune system constitutes the primary defense against pathogens, some social species have evolved behavioral mechanisms (what has been termed a "behavioral immune system") that may be the first line of defense against pathogen infections (Schaller and Murray 2011). Indeed, a wealth of literature on humans chronicles how xenophobic behavior and "disgust" toward out-groups (Curtis et al. 2004, 2011, Navarrete and Fessler 2006) have evolved as cultural traits that reduce the transmission of disease to in-groups whose immune systems are uninitiated to out-group pathogens (reviewed in Schaller and Murray 2011). Yet only a few examples of a behavioral immune system have been documented in wild animal populations. Chronic Bee Paralytic Virus in eusocial bees (*A. mellifera*) elicits a non-stinging, but aggressive behavior in uninfected hive-mates that is hypothesized to reduce parasite load (Waddington and Rothenbuhler 1976, Drum and Rothenbuhler 1983, 1985). Uninfected female

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mice increase anxiety-associated behaviors in response to the odor of males parasitized by the nematode *Heligmosomoides polygyrus* or the protozoan *Eimeria vermiformis* (Kavaliers et al. 1998). In perhaps the clearest documented case to date, normally gregarious Caribbean spiny lobsters (*Panulirus argus*) avoid cohabitation of communal dens with conspecifics infected by a pathogenic virus, *Panulirus argus* Virus 1 (PaV1), either by abandoning the den or excluding the infected lobster from entry to dens (Behringer et al. 2006, Butler et al. 2008, Lozano-Álvarez et al. 2008, Behringer and Butler 2010). It is this host–pathogen system that is the subject of this study.

Panulirus argus Virus 1 causes disease and mortality in juvenile spiny lobsters (Shields and Behringer 2004) throughout the Caribbean, where it threatens the region's most valuable fishery (Chávez 2009, Ehrhardt et al. 2010, Behringer et al. 2012). The prevalence of the virus in adult lobsters, which are asymptomatic and non-susceptible (Behringer et al. 2010), ranges from 0–15% around the Caribbean (Moss et al. 2013), but its prevalence among juveniles is often higher, with localized outbreaks exceeding 30% (Shields and Behringer 2004, Lozano-Álvarez et al. 2008). In the Florida Keys, the region modeled in this study, prevalence of PaV1 in juvenile lobsters from 2000 through 2010 fluctuated between 2% and 8%, with occasional outbreaks exceeding 60% (Moss et al. 2012). Among juvenile lobsters, mortality approaches 90% within a few months of infection (Butler et al. 2008). The virus is highly contagious among early benthic juveniles (EBJs: lobsters <20 mm carapace length (CL) that live asocially in macroalgae), but susceptibility to infection decreases with lobster size (Butler et al. 2008). Larger, crevice-dwelling juvenile *P. argus* avoid den co-occupancy with infected conspecifics (Behringer and Butler 2010). Although the specific olfactory cue used for detection is unknown (Anderson and Behringer 2013), exposed individuals become detectable and are avoided approximately two weeks before they become infectious (Behringer et al. 2006).

In nature, co-occupancy of dens by infected and uninfected juvenile lobsters is rare (Behringer et al. 2006), but environmental changes can reconfigure nursery habitats and alter the spatial structure of the lobster population in ways that diminish the effectiveness of social aversion in retarding the spread of PaV1. For example, in 1991 and 2007, dense blooms of cyanobacteria (*Synechococcus* sp.) swept over large areas (~500 km^2) of Florida Bay and decimated the sponge community in the region (Butler et al. 2005, Peterson et al. 2006, Stevely et al. 2010, Wall et al. 2012), which is an important lobster nursery. Sponges are the primary shelter for juvenile lobsters in Florida, so following the sponge die-off, the only remaining shelters for lobsters were small coral heads and solution holes into which the juvenile lobsters then aggregated. Consequently, group size increased from 1.74 ± 1.48 lobsters per shelter (mean ± SD) before the sponge die-off to 7.25 ± 4.68 lobsters per shelter afterward (see Plate 1), although healthy and infected lobsters remained segregated (Herrnkind et al. 1997, Butler et al. 2005). Fishing may also alter natural patterns of den co-occupancy by lobsters that may influence disease transmission. In Florida, fishermen manipulate the abundance of sub-legal sized lobsters in traps, which increases disease transmission (Behringer et al. 2012). In other areas of the Caribbean, artificial structures known as "casitas" are deployed on the seafloor to aggregate lobsters for ease of capture, and infected individuals sometimes co-occur with susceptible lobsters within these large structures (Lozano-Álvarez et al. 2008, Huchin-Mian et al. 2013).

In this study, we reconfigured a spatially explicit, individual-based ecological model of the juvenile *Panulirus argus* population of the Florida Keys (Butler et al. 2001, 2005, Butler 2003, Dolan and Butler 2006); the model does not include adult lobster population dynamics nor the effects of fishing on those dynamics. Here we describe how we incorporated PaV1 disease processes and related lobster behaviors into the model and investigated the importance of lobster "behavioral immunity" on the spread of the PaV1 viral disease under different circumstances. We varied the timing of the onset of avoidance of infected lobsters relative to their conversion to the infectious state and contrasted those results with the behavior of the system in the absence of the avoidance behavior. We also modeled two density-independent processes that may contribute to higher prevalence in asocial EBJs. The first model produced infections in EBJs at a constant daily rate, a mechanism representative of an alternate host or environmental exposure to the pathogen. In the second model, post-larvae were randomly infected on their arrival to the coastal system, as reported by Moss et al. (2012), which might be expected if PaV1 is vertically transmitted (i.e., transmission of PaV1 from infected but asymptomatic females to their embryos), or if pelagic larvae are infected while in the plankton or shortly after arrival inshore. Finally, we compared disease dynamics in an environment with an intact habitat structure to that in an altered landscape with few shelters, as is now the case in portions of Florida Bay impacted by sponge-killing, harmful, algal blooms (HABS).

Methods

Detailed descriptions of the original model are presented in Butler et al. (2001, 2005) and Butler (2003); a complete description of the revised model used for this study can be found in the Appendix. Here, we briefly describe the model's general structure and provide a detailed description of the elements that were altered for these simulations (Table 1), specifically, disease transmission and progression, lobster behavior, and habitat loss. The model was coded in C++ using Microsoft Visual Studio 2005.

TABLE 1. Summary of simulation conditions.

Variable	Treatment conditions
Simulation 1	
Disease avoidance	none
	occurs coincident with infectiousness
	occurs 2 weeks prior to onset of infectiousness
	occurs 4 weeks prior to onset of infectiousness
Density-independent infection of early benthic juvenile lobsters	none
	infection of 0.1% of population daily
Exogenous input of virus from infected post-larval lobsters	none
	present
Simulation 2	
Disease avoidance	none
	occurs 2 weeks prior to onset of infectiousness
	occurs 4 weeks prior to onset of infectiousness
Shelter availability	constant
	declines in response to harmful algal blooms

Notes: Response variables recorded for each simulation: number of lobsters recruiting to 50-mm carapace length; prevalence of PaV1 in each 5-mm lobster size class; incidence of PaV1 in each 5-mm lobster size class.

Lobster population dynamics were simulated within a spatial map of 2792 square, contiguous 1 km^2 habitat cells that corresponded to the primary nursery habitat for *P. argus* in South Florida (Herrnkind et al. 1997) (Fig. 1A). Each cell was designated as seagrass, hard-bottom, open (i.e., unvegetated sand or mud bottom, or land (which included emergent banks), corresponding to the actual spatial distribution of these habitats in the region, based on geographic data from NOAA's Benthic Habitats of the Florida Keys Project (FMRI and NOAA 2000) and on diver-based field surveys of >300 sites throughout the Florida Keys (M. J. Butler IV, *unpublished data*). Each seagrass and open cell in the model was treated as homogeneous habitat with unlimited capacity. However, the habitat in hard-bottom cells contained additional structural details, including unlimited macroalgae for EBJs and realistic densities of several types of benthic structures that are used as shelter by larger juvenile lobsters, including: loggerhead sponges (*Spheciospongia vesparium*), vase sponges (*Ircinia campana*), other sponges (mostly stinker sponges, *Ircinia strobilina*, and grass sponges, *Spongia cheiris*), solution holes, octocoral–sponge complexes, and other shelters (mainly scleractinean corals). Densities of each shelter type were measured using belt transects on 109 sites in 2002 (M. J. Butler IV, *unpublished data*). Ordinary kriging was then used to generate density surfaces that determined the numbers of each shelter type in the model's hard-bottom cells. Each shelter (i.e., individual sponge, coral, solution hole, etc.) was randomly assigned a lobster-holding capacity based on maximum observed group sizes specific to that shelter type from experiments described by Butler and Herrnkind (1997).

The model used a 24-hour time step that was composed of a sequence of processes that mimicked daily activity patterns of real lobsters (Fig. 1B), including arrival and settlement of post-larvae, movement, shelter selection, growth, and mortality. We used the 50-mm carapace length (CL) as the final size for estimation of recruitment, because the dynamics of lobsters larger than that are complicated by Florida's commercial lobster fishery, which uses large numbers of juveniles between 50 mm and 70 mm CL as "live decoys" in traps (Lyons and Kennedy 1981, Hunt et al. 1986, Forcucci et al. 1994). Therefore, this model focuses on juvenile lobster population dynamics and does not include adult lobster dynamics or interactions with the lobster fishery. The details of processes not affected by disease, including influx and settlement, have been presented elsewhere (Butler et al. 2001, 2005, Dolan and Butler 2006). Here we describe how we modeled disease transmission, the time course of the disease in individuals, and the effects of the disease on growth, shelter selection, movement, and mortality of lobsters. We then describe the simulation of harmful algal blooms (HABs), their effect on habitat structure, and their potential indirect effect on lobster mortality.

Contact transmission

The initial PaV1 prevalence in each habitat cell was randomly drawn from a discrete probability distribution function constructed from prevalence values observed at 66 sites from June to August 2002 (Behringer et al. 2011). The disease was then allowed to spread by contact between individuals that shared dens. Contact transmission was modeled by a stochastic function of individual susceptibility to infection and the amount of virus to which a susceptible lobster was exposed during co-occupancy. Little is known about how susceptibility to PaV1 changes with lobster age, although Butler et al. (2008) showed that transmission diminishes linearly with lobster size. Therefore, relative size-dependent susceptibility (S) was modeled by a linear function passing through the point 99% at 5 mm CL and decreasing to

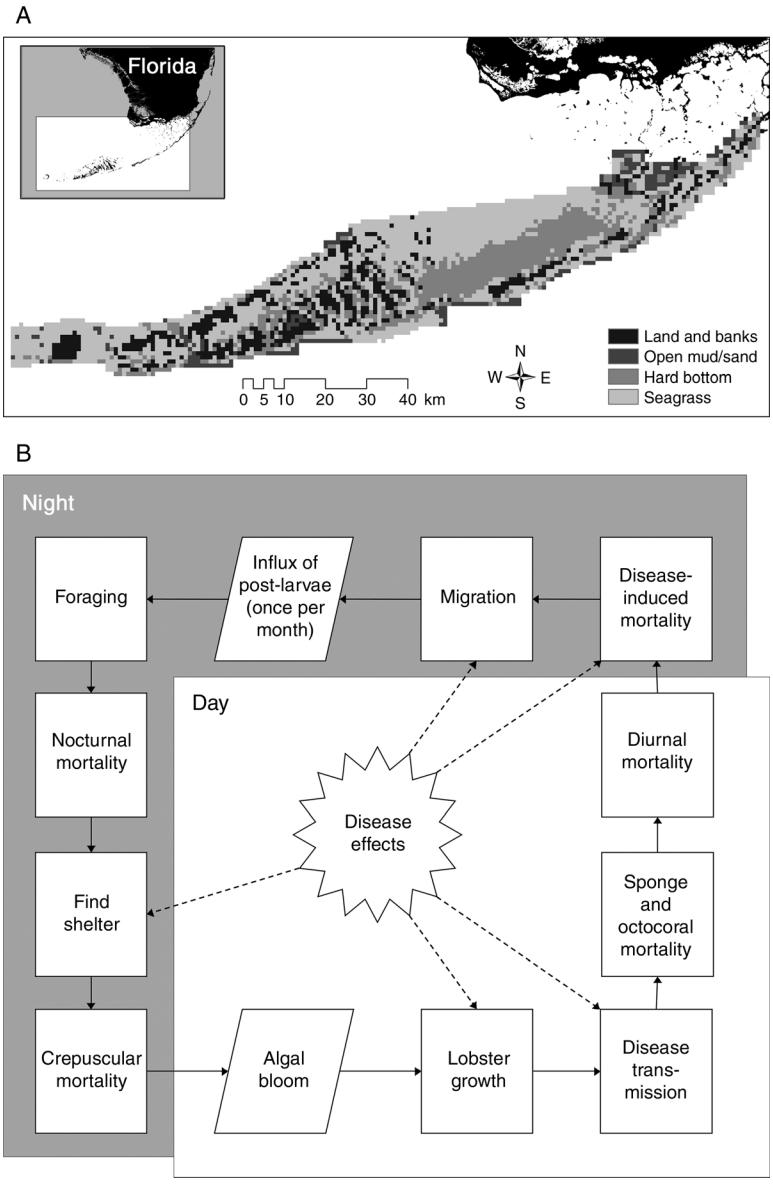

FIG. 1. Model spatial structure and population processes. (A) The spatial structure of the model consisted of a total of 2792 inhabitable cells, of which 846 were hard bottom, 1696 were seagrass, and 250 were open substrate. (B) Population processes were performed in a sequence indicated by solid arrows between process boxes. Dashed arrows indicate processes affected by disease.

0% at 65 mm CL. Lengths were converted to the equivalent biomass (m), resulting in

$$S = -0.0041m + 0.99. \quad (1)$$

We assumed that the amount of virus shed by any given lobster is dependent on its infectious state and directly proportional to its size. Because actual numbers of virions produced by infectious lobsters have not been measured quantitatively, we used the biomass of infectious lobsters as a relative measure of the doses they would produce. Likewise, we assumed that the dose required to infect a susceptible lobster is dependent on its size. Thus, we modeled the probability of infection, P_I, as

$$P_I = 1 - (1 - S)^{\frac{\sum m_i}{\kappa m_s}} \quad (2)$$

where S is the size-based susceptibility from Eq. 1, m_i is the mass of an infectious lobster to which the focal susceptible lobster is exposed, m_s is the mass of the focal lobster, and κ is a constant of proportionality relating the biomass of a susceptible lobster to the effectiveness of a dose of virions produced by an infectious lobster of the same size. To date, quantification of the dose–response curve has not been possible; the use of κm_s allowed us to

TABLE 2. Parameters for time to infectiousness and time to death functions.

Function	λ	δ	r^2	df
Time to infectiousness	0.02188	25	0.94	39
Time to death	0.01242	41	0.88	39

Note: For each function the fit to empirical data is given by the coefficient of determination, r^2.

test the sensitivity of the model to a range of dose-response relationships. The details of these simulations and graphic depictions of Eq. 2 with different parameterizations are presented in the Appendix.

Transmission to early benthic juveniles

Disease dynamics among early benthic juvenile *P. argus* (EBJs) are unknown in the wild because at this stage lobsters are small, camouflaged, asocial, and sparsely distributed deep within bushy stands of macroalgae. Laboratory experiments have demonstrated that waterborne transmission of PaV1 to EBJs is possible over distances of at least 2 m (Butler et al. 2008). Field experiments (M. J. Butler IV, *unpublished data*) in which we caged uninfected EBJs for two weeks in macroalgae-covered hard bottom are consistent with those laboratory findings, and also indicate that EBJs contract PaV1 infections (9.6% ± 8.9% [mean ± SD]; $n = 731$ EBJs at 25 sites) without direct contact with conspecifics and independently of lobster density. Presumably those infections occurred through virion-laden seawater or by consumption of infected prey tiny enough to move through 15-mm mesh cages. Therefore, we incorporated a constant uniform probability of infection as a background transmission process that represented an unknown density-independent process, such as transmission through the water or by ingestion of infected prey. To determine the best parameter value for our comparisons, we first compared the prevalence values produced using a low 0.1% daily incidence derived from a 2.5% monthly incidence, and a high 0.4% daily incidence derived from a 10% monthly incidence, to the empirically observed prevalence in EBJs from Behringer et al. (2011) and additional data collected after publication. Based on the result of that comparison (see Appendix), we used the low daily incidence (0.1%) for all other simulations that included transmission to EBJs.

Transmission to postlarvae

Numbers of post-larvae entering the model each month were derived from empirical data from a long-term monitoring program conducted in the Florida Keys, Florida, USA, by the Florida Wildlife Conservation Commission. In addition to the infection mechanisms already described, PCR analysis of a preliminary sample of post-larvae collected from Witham collectors placed at Long Key in 2007 revealed that post-larvae may be entering the system already infected with PaV1 (Moss et al. 2012). Therefore, we also simulated exogenous infection of post-larvae using the prevalence values observed in their samples.

Disease progression

For each individual lobster, the time course for the development of disease was characterized by three quantities: the time between infection and conversion to the infectious state, development of a detectable infected cue, and death. The functions governing time to death and time to infectiousness were determined by parametric survival analyses using two-parameter exponential distributions of the form $P = 1 - e^{-(t-\delta)\lambda}$ (Lee and Wang 2003), where P is the probability of the event occurring before a given time t, λ is the shape parameter, and δ is the threshold time before which it can be guaranteed an event will not occur. This particular model was chosen because it is one of the simplest and most commonly used parametric distributions for survival analysis; it fit the data sufficiently well in both cases (Table 2), and is easily manipulated in a program, unlike nonparametric methods like Kaplan-Meier. The models were calibrated to data from Butler et al. (2008). To be certain that time to death was not biased from inclusion of uninfected lobsters, only those lobsters that exhibited disease symptoms or in which infection was later detected by PCR were included in that analysis.

To model the response of uninfected lobsters to the avoidance cue, we assumed that production of the cue, presumably some chemical product of cellular breakdown in the infected lobster, is independent of infectivity, although it seems to occur 2–4 weeks prior to a lobster becoming infectious (Behringer et al. 2006). Therefore, the time at which an infected lobster became detectable was determined by an exponential cumulative density function with the same shape parameter as that determining the time to infectiousness. The threshold value was manipulated to change the mean time between detectability and infectiousness for our sensitivity analysis. The consequences of this choice were not only to increase the variance in timing of detectable infection, but also to skew the resulting distribution such that some infections were not detectable until after the infected individual became infectious (Fig. 2). Thus, the effectiveness of the avoidance behavior was reduced relative to a model using a fixed time difference or otherwise directly manipulating the time between events.

Lobster behavior

The algorithm for shelter selection assigned a probability value to each available structure that was weighted by the rank order of preference of the focal lobster for the shelter type, and the biomass of lobsters in it to account both for the focal lobster's size-specific

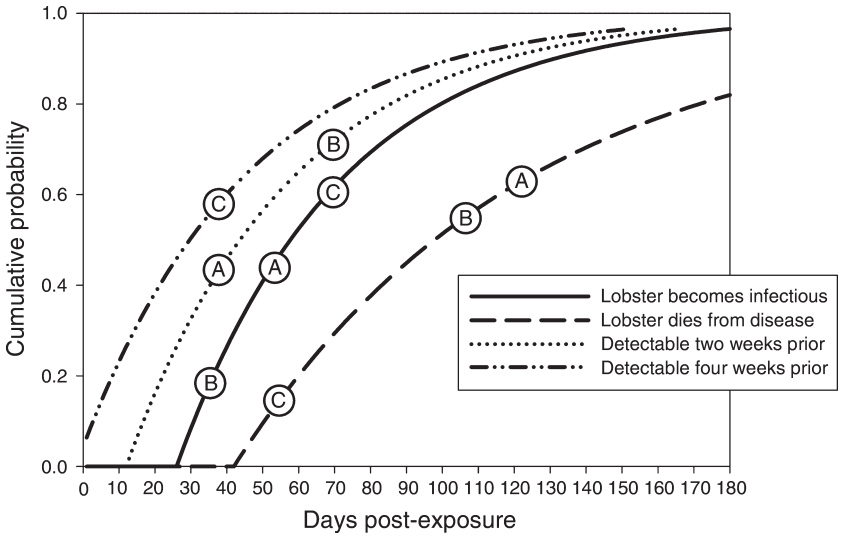

FIG. 2. Examples of possible disease development scenarios for individual lobsters. Lobster A became infected approximately two weeks prior to becoming infectious and died 120 days after being infected. Lobster B became detectable 35 days after becoming infectious and died 45 days later. Lobster C became detectable 35 days before becoming infectious and also died before becoming infectious.

preferences for certain shelter types (Butler et al. 2005) and the attraction to conspecifics (Ratchford and Eggleston 1998, Nevitt et al. 2000). The weighted values were normalized to 100%, and a uniform random draw then determined which shelter the lobster chose. If there were no available shelters, the lobster was assigned to the "open" shelter type, which corresponded to diel observations of lobsters attempting to hide by pressing against structures or clinging to bits of macroalgae.

Once a den was chosen, interactions between the focal lobster and the residents were resolved using a set of behavioral rules that determined whether the focal lobster successfully occupied the den, and, if so, whether the resident lobsters remained. If the susceptible residents of the den chosen by an infected lobster detected the infection, they excluded the infected lobster with a probability of $1 - (1 - P_{\text{excl}})^n$, where P_{excl} is a constant uniform probability of exclusion by an individual resident, and n is the number of susceptible residents whose sizes are within 20 mm CL of the focal lobster's size. If a detectably infected lobster successfully occupied a den containing susceptible conspecifics, or if an infected lobster became detectable while sharing a den with susceptible conspecifics, each susceptible lobster had a constant probability, P_{esc}, of abandoning the shelter. For these simulations, P_{excl} and P_{esc} were set to 30%, which is consistent with behaviors observed during laboratory-based den competition experiments between healthy and diseased individuals (Behringer and Butler 2010). Additional simulations were conducted with different values for the parameters, and are presented in the Appendix.

Any lobster excluded from or abandoning its chosen shelter was allowed to initiate another shelter search, subject to the limitation that no lobster could initiate a shelter search more than three times on any given day. The search limit was required both to restrict the amount of time a lobster could spend searching before sunrise and to prevent an infinite loop of search and exclusion.

The algorithm for the shelter search was further modified if a susceptible lobster attempted to occupy a den but detected a PaV1 infected lobster within it. In this case, the focal lobster had a constant uniform probability, P_{avoid}, that it would reject the shelter and continue searching as suggested by laboratory studies of shelter competition between infected and uninfected lobsters (Behringer and Butler 2010). The rejected shelter was removed from the available shelters, and the indices of attractiveness of the remaining shelters were renormalized to 100%. The searching lobster was allowed to proceed until either an acceptable shelter was found or all available shelters were rejected. If the search did not result in an acceptable shelter, the lobster was placed in the "open" category.

Laboratory-based movement assays suggest that newly infected lobsters move at similar rates as uninfected lobsters, but as the disease progresses, infected lobsters become increasingly sedentary (Behringer et al. 2008). Although these simple assays gauged relative activity levels, not rates of emigration or shelter switching, we assumed that they qualitatively reflected these rates. We further stipulated that movement rates remain unchanged until an infected lobster becomes infectious. After this state change, the probability of emigration was set to zero and the lobster no longer changed shelter unless forced out by another lobster. This is consistent with anecdotal observations of the

behavior of moderately and severely infected lobsters, but potentially introduced a bias in that simulated infected lobsters initially emigrated at higher rates than they would if a smooth function had been used to decrease their movement throughout the course of the disease.

In the late stages of PaV1 infection, lobsters typically become moribund, fail to groom themselves, and cease molting (Behringer et al. 2008). Likewise, moderately and heavily infected lobsters did not molt when held in the laboratory (Li et al. 2008). The exact timing between exposure and cessation of molting is unknown; therefore, we made the simplifying assumption that molting ceases when the lobster becomes infectious.

Habitat loss

To allow lobster aggregation sizes following sponge die-off in the model to approach those observed following the HABs that occurred in 1991 and 2007, the lobster capacities of any undamaged structures remaining after bloom damage occurred were systematically increased as follows. On each day that sponge mortality occurred in a cell, any unused shelter capacity in the surviving structures was subtracted from the amount of loss. The lower value of that result or the number of lobsters in the cell in excess of the current unused capacity was taken as the additional shelter capacity needed. This capacity was then added by iteratively selecting a shelter at random and increasing its capacity by one lobster, until the total capacity needed was reached. This was subject to the limit that no shelter could have a capacity after the sponge die-off that was more than four times its original capacity. If all of the remaining shelters reached their maximum capacity, no additional capacity was added.

Harmful algal blooms in Florida Bay do not affect lobsters directly (e.g., they do not result in hypoxic conditions and are not toxic to lobsters). Instead, by reducing shelter availability, HABs may indirectly increase lobster mortality by leaving them exposed to predators. Natural mortality of lobsters is primarily via predation, as they are not food-limited and relatively few diseases other than PaV1 are known to affect them (Shields et al. 2006). Therefore, predation was modeled as a negative, nonlinear function of lobster size and the degree of protection afforded by the type of shelter each lobster occupied. Predation on lobsters in the latter stages of PaV1 infection is also ~30% higher than on uninfected lobsters, whether sheltering in a den or not (Behringer and Butler 2010). The mortality functions we used are fully described in Butler et al. (2001), Butler (2003), and Butler et al. (2005) as well as in the Appendix.

Simulations

We ran two separate sets of simulations to investigate the effects lobster disease avoidance behavior, different modes of disease transmission, and changes in lobster aggregation associated with habitat loss on the prevalence and persistence of the PaV1 pathogen (Table 1). The response variables monitored for each simulation included the number of lobsters recruiting to 50-mm carapace length, prevalence in each 5-mm size class, and incidence in each size class. The values of each response variable were recorded for each habitat cell each simulated day, and summed over the entire region.

In our first simulation, we varied the timing of the onset of disease avoidance, daily rates of density-independent background infection of EBJs, and the exogenous influx of infected post-larvae in a three-way crossed design. We simulated the timing of behavioral immunity in four ways: never (i.e., no behavioral aversion of diseased lobsters), coincident with infectiousness, two weeks before infectiousness, and four weeks before infectiousness. The timing of the onset of disease avoidance was empirically investigated (Behringer et al. 2006), using inoculated lobsters held in small mesocosms that concentrated the chemical signal produced by an infected lobster, and behavior assays were conducted every two weeks. Though there is compelling evidence for behavior immunity in lobsters, the assay design was too coarse to permit assessment of the potential effects of behavioral immunity in natural settings. For example, in nature the strength of the chemical signal would likely increase with the onset of disease pathology, infected lobster density, and proximity to the source; we took all these factors into account in the model. Although our previous laboratory study (Behringer et al. 2006) indicated that behavioral aversion by lobsters begins two weeks prior to infectiousness, this modeling exercise permitted us to explore a fuller range of possibilities and thus assess whether selection has resulted in the most efficient disease aversion strategy. The density-independent infection rate of EBJs had two levels: none and 0.1% daily. Exogenous infection of post-larvae had two levels: present and absent. Significant differences in recruitment among treatments were analyzed using a three-way model I crossed ANOVA on rank-transformed values. Prevalence and incidence were examined graphically.

Second, we examined the effect of habitat loss (i.e., sponge die-off caused by HAB) and the consequent increase in lobster aggregation on disease prevalence. Sponge die-offs were simulated in the area of the model that corresponded to those areas of Florida Bay that had experienced die-offs in recent years (Butler et al. 1995, Herrnkind et al. 1997, Peterson et al. 2006, Stevely et al. 2010, Wall et al. 2012). For these simulations, HABs either occurred or did not. We compared simulations using three levels of disease avoidance onset: none, onset two weeks prior, and onset four weeks prior to infection. The density-independent infection rate for EBJs was 0.1% per day, and exogenous infection of newly arriving post-larvae was not simulated. Although the entire Florida Keys region was simulated, the response variables were aggregated and compared only

within the HAB-impacted region. Significant differences in recruitment among treatments were analyzed using a two-way crossed ANOVA on log-transformed values, and specific differences were found using the Ryan-Einot-Gabriel-Welsch (REGW) F test. Prevalence and incidence were examined graphically.

Results

Disease avoidance and mode of transmission

In all, 16 scenarios were simulated in a factorial design with four levels of disease avoidance (no avoidance and avoidance with onset coincident with, two weeks prior to, and four weeks prior to infectiousness), two density-independent incidences for EBJs (with and without), and two levels of exogenous infection of newly arriving post-larvae (with and without). There was a significant three-way interaction among the factors ($F_{3, 144} = 62.03$, $P < 0.0001$; also see Appendix: Table A2). Plots of mean annual recruitment (Fig. 3) show that, in the absence of infection in EBJs (i.e., no density-independent infection mechanism and no exogenous infection of post-larvae), the avoidance behavior maximized recruitment at ~4200 recruits·km^{-2}·yr^{-1} regardless of the timing of the onset of the behavior relative to infectiousness. Scenarios that included either density-independent infection of EBJs or an exogenous source of infection of newly arriving post-larvae did not reach this maximum, but increasing the amount of time before infectiousness during which diseased lobster could be detected also increased recruitment.

In the absence of an infection mechanism for EBJs, the disease avoidance behavior drove PaV1 to extinction in the simulated population within five years, regardless of the timing of disease detectability. When avoidance behavior by lobsters was not included in the model, the disease persisted with gradually increasing prevalence. Peaks in prevalence closely followed peaks in lobster abundance within size class, regardless of the timing of the onset of avoidance (Fig. 4). Incidence also tracked with the abundance of susceptible lobsters, indicating that transmission was density dependent in the model. However, the density-dependent signal was much weaker in simulations that included disease avoidance. In all scenarios, prevalence of PaV1 cycled annually with the size of the population of crevice-dwelling lobsters, with peak values occurring between August and October, lagging behind the annual, early-spring peak in post-larval recruitment (Acosta et al. 1997) by 4–6 months. However, there were pronounced annual increases in prevalence corresponding to reductions in lobster abundance in each size class. This was due to growth and recruitment of susceptible lobsters to larger size classes and the cessation of growth of lobsters in late stages of the disease (see Appendix: Fig. A5).

Both density-independent infection of EBJs and exogenous infection of newly arriving post-larvae were sufficient at the levels simulated to produce infections in the largest juvenile lobster size classes (Fig. 5). This was true even when the timing of disease avoidance was set to an unrealistically high level, that is, healthy lobsters could detect and avoid diseased lobsters four weeks (on average) before diseased lobsters became infectious. Although the pulsed introduction of new infections via arriving post-larvae was reflected in similar pulses of prevalence in subsequent lobster size classes, incidence within each size class was not pulsed. Therefore both disease transmission among lobsters and growth of pre-infectious lobsters (i.e., those in the early stages of infection) were important in propagating the disease in larger size classes.

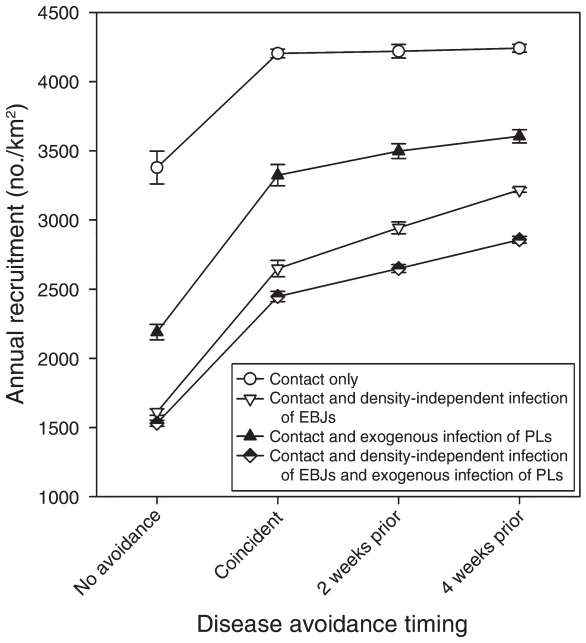

FIG. 3. Simulated annual recruitment per square kilometer for different infection mechanisms and timings of disease avoidance. Simulations were run with different combinations of infection mechanisms: transmission by contact among crevice-dwelling juveniles (Contact); constant, density-independent infection of early benthic phase juveniles (EBJs) at a rate of 0.1%/d (density-independent infection of EBJs); and exogenous infection of newly arrived post-larvae at empirically observed rates (exogenous infection of PLs). Each combination of infection mechanisms was run with one of four different timings for the onset of disease avoidance: no avoidance behavior (No avoidance); avoidance onset coincident with onset of infectiousness (Coincident); avoidance onset two weeks before the onset of infectiousness (two weeks prior); and avoidance onset four weeks before the onset of infectiousness (four weeks prior).

Disease avoidance and habitat loss

We examined the interaction of disease avoidance and habitat loss on lobster recruitment and PaV1 prevalence and persistence in a two-factor, fixed-effects design with three levels of disease avoidance (no avoidance and avoidance with onset two weeks prior to, and four weeks prior to infectiousness) and two levels of habitat loss (HAB occurred or did not occur). For these simulations,

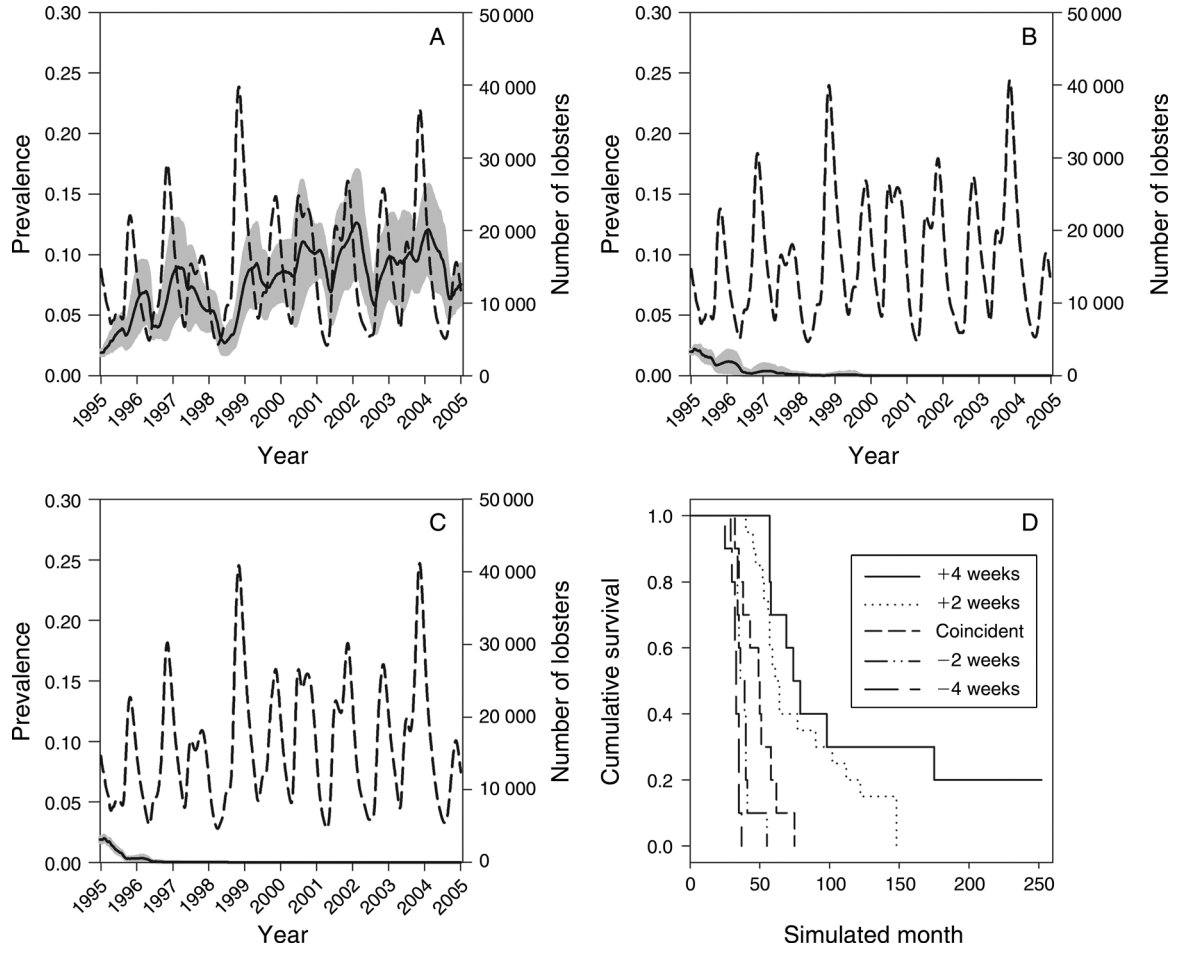

FIG. 4. PaV1 prevalence in juvenile lobsters (proportion of lobsters infected) and lobster abundance predicted in simulations that included only contact transmission. Solid lines are mean daily PaV1 prevalence in juvenile lobster (left y-axis in graphs A–C); dashed lines are lobster abundances (right y-axis in graphs A – C; $n = 10$ simulations; standard error [gray shading]). Simulation results when (A) no disease avoidance was simulated, (B) mean avoidance onset was coincident with mean time to conversion to infectiousness, (C) mean avoidance onset occurred two weeks before conversion to infectiousness. (D) Kaplan-Meier survival curves for PaV1 in the population for different timings of the onset of disease avoidance.

only recruitment within Florida Bay (the area potentially impacted by HABs) was compared. The interaction between habitat loss and disease avoidance had no effect on recruitment ($F_{2,54} = 1.07$, $P = 0.35$; also see Appendix: Table A3). Both disease avoidance and habitat loss significantly ($F_{2,54} = 47.81$, $P < 0.0005$, and $F_{1,54} = 1664.32$, $P < 0.0005$, respectively) affected lobster recruitment. Habitat loss reduced lobster recruitment via increased natural mortality (through increased predation) and emigration of lobsters without shelter. Disease avoidance was equally effective in increasing recruitment, whether the onset of avoidance was two or four weeks prior to infectiousness (REGW F test, $P = 0.82$). The absence of a significant interaction between habitat loss and disease avoidance indicates that disease avoidance operated effectively regardless of the level of shelter limitation. Similar to our previous results, in the absence of EBJ infection, avoidance reduced prevalence to 0% within two years, whereas in the absence of avoidance, prevalence tracked lobster abundance, gradually increasing over time (Fig. 6). In the absence of EBJ infections, prevalence initially increased with the onset of HABs relative to scenarios without HABs; however, total incidence decreased due to loss of susceptible lobsters to predation and migration from the area (Fig. 7). In scenarios that included infection of EBJs, the HAB led to a 77% decrease in mean prevalence. The decrease in prevalence was caused by increased predation on and emigration of susceptible lobsters, and increased predation on infectious lobsters that were excluded from shelters. Overall mortality (for all size classes combined) increased by 15% in the HAB-affected region; unfortunately, this model was not designed to separate mortality by size. The HABs in Florida Bay do not directly affect lobsters. Their effect is to reduce shelter availability, thus increasing the exposure of juvenile lobsters to predators, which would be especially deleterious to late-stage PaV1-infected

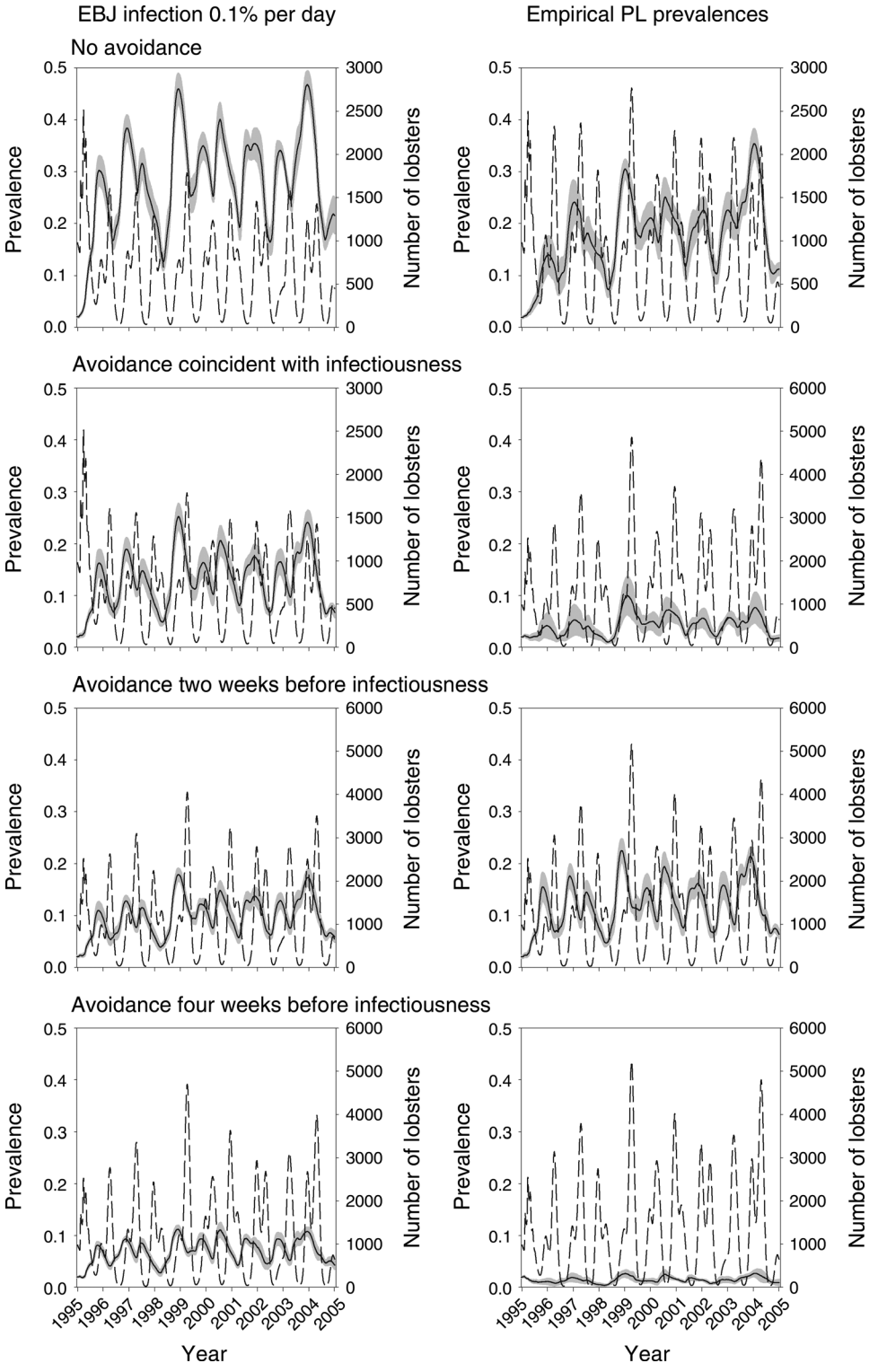

FIG. 5. Effect of density-independent infection of early benthic juveniles (EBJs) and empirical prevalences in post-larval lobsters on PaV1 prevalence in juvenile lobsters (proportion of lobsters infected). The abundance of juvenile lobsters is represented by the dashed line. The solid lines represent mean daily prevalence of PaV1 infection ± SE (gray shading). Note that population levels are not on the same scale in each graph.

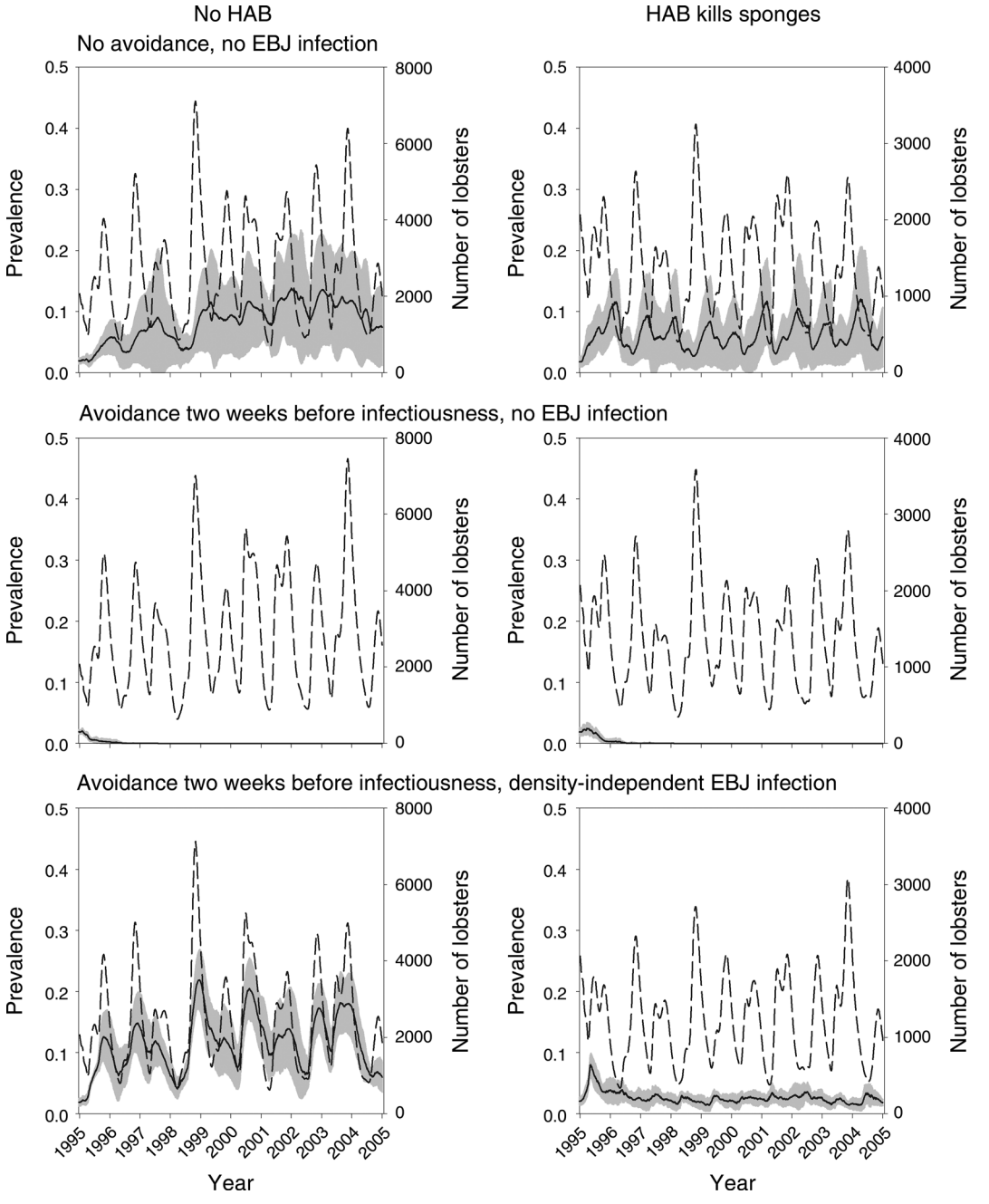

FIG. 6. Effect of harmful algal blooms (HABs) and disease avoidance on PaV1 prevalence (proportion of lobsters infected) in early benthic juvenile lobsters (EBJs). The solid lines are the mean number of infected lobsters ± SE (gray shading). The dashed lines are mean population sizes. Harmful algal blooms did not affect persistence of the disease in the population in the absence of EBJ infection. However, the HAB reduced mean prevalence by 77% when EBJs were infected at 0.1% daily. This reduction was due to increased predation and migration of susceptible lobsters and increased predation of infectious lobsters that were excluded from shelters and could not emigrate.

lobsters that are more susceptible to predation than uninfected lobsters (Behringer and Butler 2010).

DISCUSSION

Using a spatially explicit, individual-based model of the Caribbean spiny lobster–PaV1 virus system we explored how viral disease dynamics are influenced by host behavioral aversion to diseased conspecifics, background sources of infection, and increased host aggregation due to a loss of habitat. In the absence of behavioral immunity, outbreaks of PaV1 occurred rapidly, growing in intensity and duration until, by the

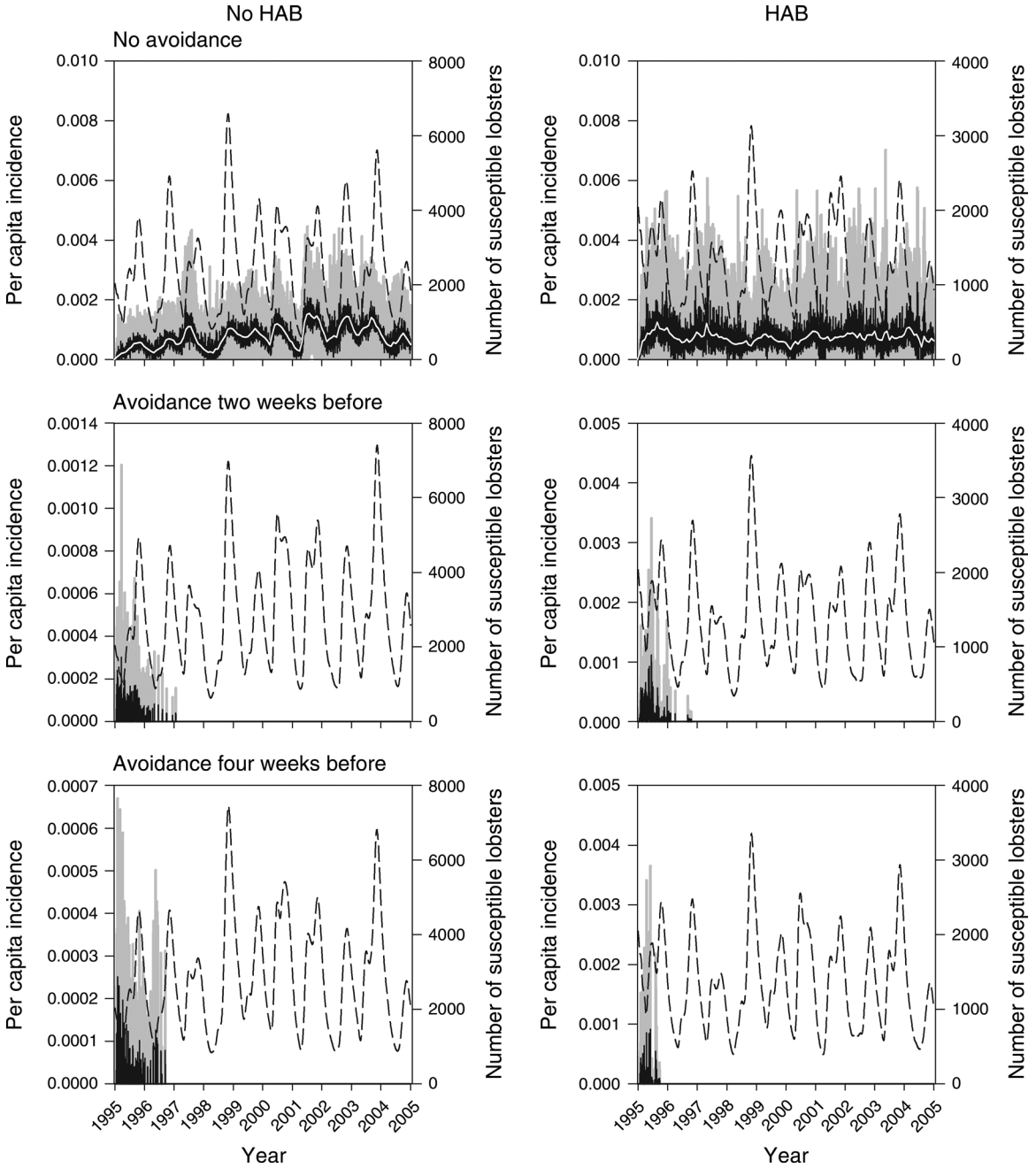

FIG. 7. Effect of HABs and disease avoidance on PaV1 per capita incidence in lobsters >25 mm CL. In graphs A and B, the white line is mean per capita incidence (number of new infections in a given time interval divided by the number of susceptibles at the beginning of the interval) smoothed using a 30-day running average. In all graphs, the black lines are mean per capita incidence ± SE (gray shading). The dashed lines are mean numbers of susceptible lobsters. Density-dependent transmission is demonstrated when per capita incidence increases with increasing population size, which is the case only in the absence of HABs and avoidance of disease. Note that the graphs are plotted with different scales for per capita incidence and for numbers of lobster.

end of the 10-year simulation, the virus was maintained continuously at high levels in the juvenile lobster population. When simulations included avoidance of diseased conspecifics, both the level and duration of PaV1 outbreaks were diminished and, in the absence of other sources of PaV1 infection, the pathogen was driven to extinction within five years. However, when the system included "background" sources of infection, such as density-independent infection of early benthic juveniles or the arrival of infected post-larvae in the system, the disease persisted at levels consistent with empirical observations. If behavioral immunity is as

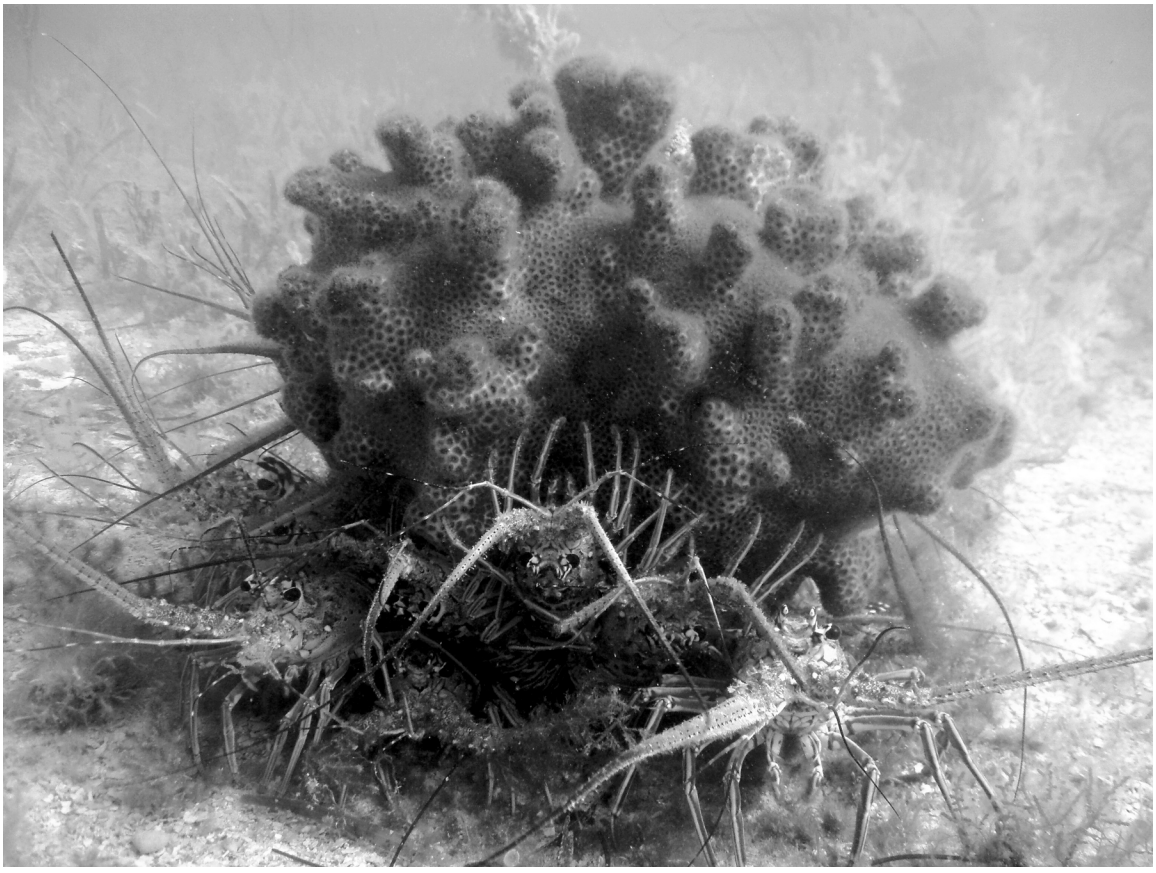

PLATE 1. A large aggregation of juvenile Caribbean Spiny Lobster (*Panulirus argus*) under a coral head (*Solenastrea hyades*) in the Florida Keys following the 2007 sponge die-off, which reduced shelter availability in the lobster nursery and resulted in unusually large aggregations of juvenile lobsters, thus increasing the potential for spread of the contagious PaV1 virus. Photo credit: M. J. Butler III.

effective in reality as it is in the model, it is a strong selective force against contact transmission and should favor pathogen strains that use other modes of transmission. The fact that viral prevalence appears stable in real populations indicates that other modes of transmission that surmount the host's behavioral immunity are probably important in the Caribbean spiny lobster–PaV1 virus host–pathogen relationship.

More generally, this study serves as an example of the use of a fine-scale, detailed epidemiological model to explore the intricacies of host-pathogen dynamics such as host behavior and the changing spatial structure of natural habitats and, hence, host populations. Traditional epidemiological models do not handle transmission dynamics well if complicated by changes in habitat structure or quality, host behavior, or ontogeny that alter patterns of disease transmission (reviewed in Ostfeld et al. 2005). Yet understanding transmission under such circumstances is the key to predicting the spread of pathogens in changing natural environments. The modeling of viral and bacterial diseases has remained firmly tied to theoretical population-based formulations, with transmission based on particle diffusion linked to state-based transition probabilities. Although more complex mass-action functions have surfaced, evidence from experiments and observations of disease outbreaks place in question their general applicability (see McCallum et al. 2001). Alternative approaches that explicitly recognize spatial proximity, host behavior, nonuniform host characteristics, and changes in the local environment that alter transmission efficiency are necessary.

Moreover, there is concern that the terrestrial-based epidemiological models that dominate the literature may not be applicable in the sea. There are fundamental differences between terrestrial and marine systems in host and pathogen life history and modes of pathogen dispersal (Harvell et al. 2004, McCallum et al. 2004). Perhaps foremost among those differences as they pertain to disease transmission is the strongly advective physical environment of the sea, capable of long-distance dispersal of not only planktonic larvae, pollutants, and chemicals but also pathogens. Among the consequences for highly connected marine metapop-

ulations are the potential for the rapid spread of the disease over large geographic areas (McCallum et al. 2003), the apparent density-independent maintenance of the disease in local populations (Gurarie and Seto 2009), and the promotion of pathogen virulence (Ferdy 2009).

Our simulations indicate that the behavioral immunity exercised by Caribbean spiny lobsters is quite effective at preventing epizootics. Absent behavioral immunity, the PaV1 virus spreads in the population, eventually infecting >70% of the population. Our simulations also indicate that selection has fine-tuned the onset of social aversion relative to infectiousness so as to maximize its effectiveness in preventing the spread of disease. That is, the aversion of diseased lobsters by healthy conspecifics was equally effective in sustaining a healthy lobster population, whether the onset of avoidance was two or four weeks prior to infectiousness. Behavioral immunity is well documented in humans where its importance has been linked to such fundamental processes as the evolution of sexual reproduction (Hamilton and Zuk 1982, Zuk 1992), mate choice (Gangestad and Buss 1993, Gangestad et al. 2006), and xenophobic behavior in a variety of cultures (Schaller and Murray 2011). In contrast, beyond the PaV1–spiny lobster example, only a few nonhuman studies of domesticated animals (mice [Kavaliers and Colwell 1995, Kavaliers et al. 1998]) and wild animals (chimpanzees [Goodall 1986]; tadpoles [Kiesecker et al. 1999]) have documented the existence of behavioral aversion as a mechanism to slow the spread of disease. None of those studies investigated how "optimal" the development of behavioral immunity might be in a particular social system, as we have done here.

In fact, avoidance behavior was so effective in reducing the spread of the PaV1 virus in our simulations, that other modes of transmission were needed to overcome extinction of the pathogen; for example, a source such as the influx of infected recruits (post-larvae) from outside the local system. Like many marine species, the Caribbean spiny lobster has a planktonic larval stage that remains in the plankton for several months (Goldstein et al. 2008) and thus is capable of long and complex patterns of dispersal throughout the Caribbean (Butler et al. 2011, Kough et al. 2013). The Florida Keys, in particular, appear to be an ecological "sink" in terms of *P. argus* larval connectivity (Kough et al. 2013). Some post-larvae arrive inshore in the Florida Keys infected with PaV1 (Moss et al. 2012), which they have acquired through an unknown mechanism such as vertical transmission or waterborne infection while in the plankton or upon entry to the coastal zone.

Other possible routes of PaV1 transmission include waterborne transmission and ingestion of infected tissue (Butler et al. 2008). If by water-borne transmission, then it likely operates on a very short timescale, as laboratory and field studies indicate that the virus only remains viable and capable of infecting cell cultures and EBJs for a few days (M. J. Butler IV, *unpublished data*). Ingestion of PaV1-infected prey or through alternate/intermediate hosts is another potential indirect route of transmission; however, no alternate hosts for PaV1 are known (Butler et al. 2008). Still, our modeling indicates that another mechanism for PaV1 transmission probably operates as yet undetected in the background and is necessary for maintenance of the PaV1 virus in the lobster population given the effectiveness of the host's behavioral immunity.

The efficacy of the behavioral immunity displayed by lobsters is particularly compelling when examined in the context of the effects of habitat loss and changes in host distribution. In spite of larger aggregations of lobsters that occur following the loss of sponge shelters killed by HABs, our simulations predicted that the prevalence of PaV1 should decrease, not increase as one might expect when hosts become highly aggregated. Yet, our empirical studies corroborate these unexpected modeling predictions. Surveys reveal that PaV1 prevalence remained steady in Florida's lobster population before, during, and at least five years after the last HAB to hit the region (Behringer et al. 2011, 2012). Again, we suspect that some undetected mode of PaV1 transmission exists in nature that allows the virus to persist. Without such a mechanism, model projections are that a smaller lobster population (due to increased mortality of infected lobsters and greater emigration by healthy lobsters) coupled with disease avoidance should drive PaV1 to extinction within five years. Thus, our search continues for an additional mode of viral transmission, the elusive evolutionary "counter-punch" to the effectiveness of behavioral immunity.

Acknowledgments

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Supplemental Material

Appendix

Supplemental testing, sensitivity, and results for the *Panulirus argus*-PaV1 model (*Ecological Archives* E095-208-A1).

Supplement

C++ code for the *Panulirus argus*-PaV1 spatially explicit, individual-based model (*Ecological Archives* E095-208-S1).