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REPORT

Coralline algae disease reduces survival and settlement success of coral planulae in laboratory experiments

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Abstract Disease outbreaks have been involved in the deterioration of coral reefs worldwide and have been particularly striking among crustose coralline algae (CCA). Although CCA represent important cues for coral settlement, the impact of CCA diseases on the survival and settlement of coral planulae is unknown. Exposing coral larvae to healthy, diseased, and recently dead crusts from three important CCA species, we show a negative effect of disease in the inductive CCA species *Hydrolithon boergesenii* on larval survivorship of *Orbicella faveolata* and settlement of *O. faveolata* and *Diploria labyrinthiformis* on the CCA surface. No effect was found with the less inductive CCA species *Neogoniolithon mamillare* and *Paragoniolithon accretum*. Additionally, a majority of planulae that settled on top of diseased *H. boergesenii* crusts were on healthy rather than diseased/dying tissue. Our experiments suggest that CCA diseases have the potential to reduce the survivorship and settlement of coral planulae on coral reefs.

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Introduction

Marine diseases have increased in the past few decades and altered critical ecosystem processes (Harvell et al. 1999). Of particular concern is the decline of reef-building corals, which are the foundation species of coral reefs (Bruno and Bertness 2001). While coral diseases have made a direct contribution to this decline (Aronson and Precht 2006), diseases affecting other reef-associated organisms can indirectly affect coral populations by altering ecological processes such as coral recruitment, competition, and predation. For example, in the early 1980s, an unknown pathogen eradicated most of the long-spined sea urchin *Diadema antillarum* in the Caribbean. The loss of this major herbivore facilitated a coral to algal shift on some reefs (Hughes 1994).

The emergence of diseases has been particularly striking among crustose coralline algae (CCA). In the 1990s, coralline lethal orange disease (CLOD) caused massive mortality in *Porolithon onkodes* in the Pacific (Littler and Littler 1995). During the same period, *Porolithon pachydermum* largely vanished as a result of coralline white band disease (CWBS) in the Atlantic (Goreau et al. 1998). More recently, five CCA disease categories have been described in the Pacific (Vargas-Ángel 2010), and the coralline white patch disease (CWPD) has been reported in Curaçao (Quéré et al. 2015). Although their prevalence remains low, disease hot spots have been found, and a link between coralline fungal disease (CFD) and ocean-warming events indicates that temperature anomalies will increase the susceptibility of CCA to diseases (Williams et al. 2014).

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CCA produce cues enhancing coral larval settlement and metamorphosis (Morse et al. 1988; Heyward and Negri 1999; Negri et al. 2001). However, the impact of CCA diseases on coral recruitment is unknown. After survival through a pelagic phase, larval attachment and subsequent metamorphosis mark the start of the coral benthic life and constitute a critical ecological step to reef recovery and replenishment following disturbances (Vermeij and Sandin 2008; Ritson-Williams et al. 2009). Its success relies on the presence of effective sensory cues (e.g., chemical, spectral) associated with specific CCA and/or microbial films, and the ability of coral larvae to respond to them (Morse et al. 1994; Heyward and Negri 1999; Webster et al. 2004; Mason et al. 2011). CCA diseases could reduce coral larval survivorship and settlement success by inducing shifts in the microbial community and/or the morphogens associated with CCA and disrupting the signal inducing metamorphosis or settlement in coral planulae. It is unclear whether the biofilms present on CCA or the CCA themselves are responsible for the settlement and metamorphosis of corals (Johnson et al. 1991; Webster et al. 2004). However, settlement in marine invertebrates can be hampered when CCA are treated with antibiotics, suggesting that benthic microbes may be necessary to induce settlement and metamorphosis (Johnson et al. 1991; Vermeij et al. 2009). Diseases can change the microbial or chemical profiles of marine organisms. For instance, the production of certain metabolites and the chemical and microbial profiles of the marine sponge *Aplysina aerophoba* were altered when affected by the *Aplysina* Black Patch Syndrome (Webster et al. 2008). Similarly, coral diseases typically induce shifts in the microbial community associated with the coral mucus or tissue (Pantos et al. 2003; Sunagawa et al. 2009). Finally, coral larvae are also sensitive to conditions experienced in the water column prior to settlement (Vermeij et al. 2006). Diseased CCA could impact the survival of coral larvae by releasing noxious chemicals or by acting as reservoir and vector of pathogens.

The aim of this study was to test the hypothesis that CCA diseases reduce coral larval survival and settlement success. Survival and settlement of larvae from two major Caribbean reef-building coral species were quantified in no-choice laboratory experiments involving healthy, diseased, and recently dead crusts from three CCA species, which differed in their habitat preference, disease type, and ability to induce coral settlement.

Materials and methods

Coral gametes collection

The experiments were conducted on the island of Curaçao, Southern Caribbean at the Carmabi biological research station. Two common Caribbean reef-building corals were used: *Orbicella* (formerly *Montastraea*) *faveolata* and *Diploria labyrinthiformis*. Both species are broadcast-spawning corals. *O. faveolata* releases sperm–egg bundles during yearly mass spawning between August and November (Vermeij et al. 2006). *D. labyrinthiformis* spawns between April and July (Muller and Vermeij 2011). Planulae were obtained by collecting gametes from eight *O. faveolata* and 11 *D. labytinthiformis* colonies located in the shallow reef (3–8 m) at Seaquarium (12°04′59″N, 68°53′43″W). Gametes bundles from *O. faveolata* were collected at night on September 18, 2011. *D. labyrinthiformis* spawned just before nightfall on May 17, 2012. Gametes were pooled, fertilized, and larvae were reared at Carmabi following Vermeij et al. (2009) until they reached competency on the fourth and second days after spawning for *O. faveolata* and *D. labyrinthiformis,* respectively.

CCA collection

Healthy and diseased fragments of three CCA species (*Hydrolithon boergesenii, Neogoniolithon mamillare, Paragoniolithon accretum*) were collected using hammer and chisel on the reef terrace (5–10 m depth) at Water Factory (12°06′32″N, 68°57′14″W). While *H. boergesenii* has been shown to be an effective coral settlement cue (Morse and Morse 1991; Morse et al. 1994), *N. mamillare* appears less inducive (Ritson-Williams et al. 2014). The potential of *P. accretum* to act as a coral settlement cue has not been tested. *H. boergesenii* commonly grows in cryptic microhabitats between 8 and 20 m depth. *N. mamillare* is abundant at shallow depths (<8 m) and colonizes exposed habitats, while *P. accretum* is less common and grows in cryptic microhabitats. All three species showed frequent signs of disease (Quéré et al. 2015). All collected diseased fragments of *H. boergesenii* and *P. accretum* were affected by CWPD, and all diseased fragments of *N. mamillare* were affected by CWBS. CWBS is characterized by a well-defined, white band progressing over healthy algal tissue (Fig. 1a). The non-pigmented remains of tissue left behind initially appear white but turn greenish as it becomes colonized by endophytic green algae. CWPD is identified by the presence of distinct white patches on an otherwise healthy crust (Fig. 1b). The discolored area is irregularly shaped and can be located peripherally or centrally spreading in a random way, which contrasts with CWBS (see Quéré et al. 2015 for further details on each disease).

Each replicate piece of CCA was selected from an individual patch, and fragments from each health category were placed in individual collecting bags in order to avoid contamination. After collection, healthy and diseased CCA fragments were maintained at Carmabi in separate aquaria with running seawater. A sample of each fragment was

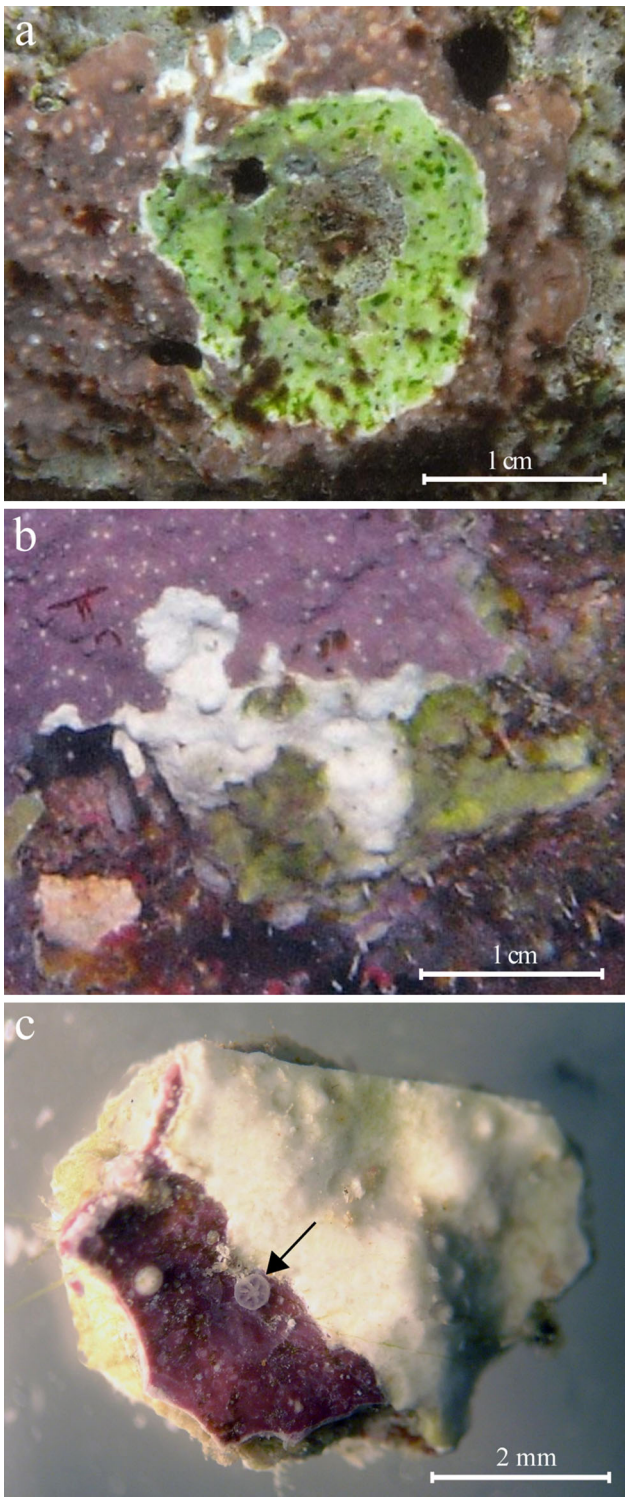

Fig. 1 **a** Coralline white band syndrome (CWBS). **b** Coralline white patch disease (CWPD). **c** Diseased crust (CWPD) showing *Diploria labyrinthiformis* settler (*arrow*) on healthy tissue

kept for taxonomic identification. The pieces used for taxonomic determination were rinsed with freshwater and dried for 6 h in the oven at 60 °C before being checked under a dissecting scope for reproductive and morphological features. The rest of each fragment was chopped into a 0.5 × 0.5 -cm chip using angle pliers and cleaned so that only the crust of the CCA and a thin layer of limestone underneath remained. From the diseased fragment, three chips were chopped: one into the healthy-looking tissue (He-D), one into the diseased tissue leaving ca. 30–50 % healthy-looking tissue (Di-D), and one into the dead tissue (De-D). All chips were used in experiments within 48 h after collection.

Settlement experiments

Larvae from each coral species were exposed in a no-choice experiment to six treatments: (1) a healthy fragment from a healthy CCA (He-H), (2) a healthy-looking fragment from a diseased CCA (He-D), (3) a fragment including an active disease lesion (i.e., the presence of white, non-pigmented zones) from a diseased CCA (Di-D), (4) a recently dead fragment showing greenish tissue from a diseased CCA (De-D), (5) a fragment of dead coral skeleton (dead skeleton control or Sk-Ctrl), and (6) a treatment containing only filtered sea water (0.2 µm FSW; seawater control or Sw-Ctrl). The dead coral skeletons were taken from long-dead and sun-bleached coral colonies lying on shore. The cut pieces were cleaned, rinsed in freshwater, and left to dry under direct sunlight for 24 h.

Larval bioassays were performed in sterile six-well culture plates, with one treatment allocated to an individual well so each plate displayed all six treatments. Eleven coral larvae were added to each well with 10 ml of 0.2-µm FSW. After 48 h, the number of living larvae and settlers (firmly attached and beginning to calcify; Heyward and Negri 1999) was recorded using a dissecting microscope. We also recorded the substrate on which the larvae settled. In each well, except for the seawater control, larvae could settle on three different types of substratum: the top of the fragment (CCA crust or top of the dead skeleton), the bottom of the fragment (limestone rock underneath the CCA or bottom of the dead skeleton), and the plastic of the well. In addition, in the treatment consisting of a fragment with an active disease lesion (Di-D), larvae could settle on the healthy or diseased parts of the CCA crust (Fig. 1c). Ten replicate culture plates were used for each CCA species. The same experiment was repeated for each coral species separately. The experiments were carried out in an indoor laboratory under natural light cycles and with a constant ambient temperature of 29 °C.

Statistical analyses

The percentages of surviving and settled larvae were calculated for each well. Assumptions of parametric testing

were not validated using diagnostic plots in R. Initial homogeneity of dispersion tests (PERMDISP; Anderson 2004) were conducted to ensure that there was no difference in spread among treatments (excluding the two controls). Since the same original diseased fragment was used to make one replicate of three of the six treatments (i.e., He-D, Di-D, and De-D), we conducted a randomized block PERMANOVA to test for differences in larval survivorship and settlement rates for each coral and CCA species combination with treatment as a fixed factor (three levels), 'sample origin' as a random effect (10 levels), and wells as replicates. However, sample origin did not have any significant effect (Electronic Supplementary Materials, ESM, Tables S1, S2); therefore, we did not include this factor in further analyses. A one-way permutation-based analysis of variance (PERMANOVA; Anderson 2001) was used to test for differences in larval survivorship and settlement rates for each coral and CCA species combination with treatment as a fixed factor (six levels), and wells as replicates. Settlement rates on top and bottom surfaces were also analyzed using the same design, but excluding the control treatments since they were often zero. All PERMANOVAs were run on untransformed percentage data using 9999 permutations of raw data from residuals under a reduced model using Euclidian distance. When the effect of the treatment factor was significant ($P_{perm} < 0.05$), we performed individual pair-wise tests to detect which treatments were responsible for significant differences (Anderson 2005). When the number of possible permutations was low, we used the asymptotic Monte Carlo p values (P_{mc}). Analyses were performed in R (v2.15.2; R Development Core Team 2013) and the FORTRAN computer program PERMANOVA (Anderson 2005).

Results

Larval survivorship reached over 66 and 88 % across all treatments for *O. faveolata* and *D. labyrinthiformis*, respectively (Figs. 2a, 3a). Differences in survival rates were found among treatments for *O. faveolata* (all PERMAVOVAs, $P_{perm} < 0.05$), but not for *D. labyrinthiformis* ($P_{perm} > 0.05$; see ESM Table S3 for exact F and P_{perm} values). *Orbicella faveolata* survival in treatments containing *N. mamillare* or *P. accretum* ranged from 66 to 77 %. These values were significantly lower than in the FSW and dead skeleton controls, respectively (PERMANOVA pair-wise tests, $P_{mc} < 0.05$), which had above 81 % survivorship, suggesting a negative effect of these CCA on *O. faveolata* larvae regardless of disease status. In contrast, in treatments containing *H. boergesenii*, *O. faveolata* larvae survived as well in the presence of the healthy fragment of healthy CCA as in the controls, with over 87 % survivorship. However, survivorship declined below 74 % in the presence of diseased and recently dead fragments (Di-D and De-D) and was significantly lower than in the presence of the healthy fragment of healthy *H. boergesenii* (He-H; PERMANOVA pair-wise tests, $P_{mc} < 0.05$), suggesting a negative impact of CCA disease on *O. faveolata* survivorship. The healthy fragment of diseased *H. boergesenii* (He-D) did not differ from the other treatments.

Overall, settlement rates in the controls were null for *O. faveolata* and less than 7 % for *D. labyrinthiformis*, and significantly lower than all other treatments (Figs. 2b, 3b; see ESM Table S4 for overall F and P_{perm} values; PERMANOVA pair-wise tests, $P_{mc} < 0.05$). No difference was detected among the remaining treatments except for the *D. labyrinthiformis* x *H. boergesenii* combination in which settlement in the diseased treatment was significantly lower than in both healthy treatments (He-H and He-D; PERMANOVA pair-wise tests, $P_{mc} < 0.05$). Settlement on dead fragment (De-D) was also lower than on healthy-looking fragments of diseased CCA (He-D).

In each well containing a CCA fragment, larvae had the possibility to settle on the top of the fragment (i.e., the CCA crust), which is the surface available to larvae in natural environment, or on the bottom of the fragment. The crusts of *N. mamillare* or *P. accretum* were clearly avoided by both coral species regardless of disease status (Figs. 2c, 3c). Settlement rates of both species ranged between 0 and 3.6 % on the crust of *N. mamillare* and between 0 and 11.8 % for *P. accretum* with no significant difference in settlement rates among treatments for these CCA species (PERMAVOVAs, $P_{perm} > 0.05$; ESM Table S4). In contrast, with *H. boergesenii*, *D. labyrinthiformis* larvae showed significantly higher settlement on top of both healthy fragments (He-H and He-D; >40 % settlement) than on top of diseased and dead fragments (Di-D and De-D; <20 % settlement; PERMANOVA pair-wise tests, $P_{mc} < 0.05$). *Orbicella faveolata* larvae showed a similar pattern, although only the healthy crust of healthy CCA (He-H) showed a higher settlement rate (15 %) than the crusts of the diseased and dead CCA (<3 % settlement; PERMANOVA pair-wise tests, $P_{mc} < 0.05$). The healthy crust of the diseased CCA (He-D) had intermediate settlement (8 %) and did not differ from the other treatments. Settlement on the bottom of *H. boergesenii* chips generally showed an inverse trend for both coral species (Figs. 2d, 3d). The high overall settlement in *D. labyrinthiformis* on healthy fragments of *H. boergesenii* clearly resulted from increased settlement induced by the healthy CCA crusts.

The importance of healthy *H. boergesenii* tissue was also visible on a smaller scale. When pooling, all *D. labyrinthiformis* planulae that had settled on top of diseased fragments that displayed both healthy and diseased tissue, 15 out of the 20 settlers (75 %) were on healthy tissue (Fig. 1c). Too few

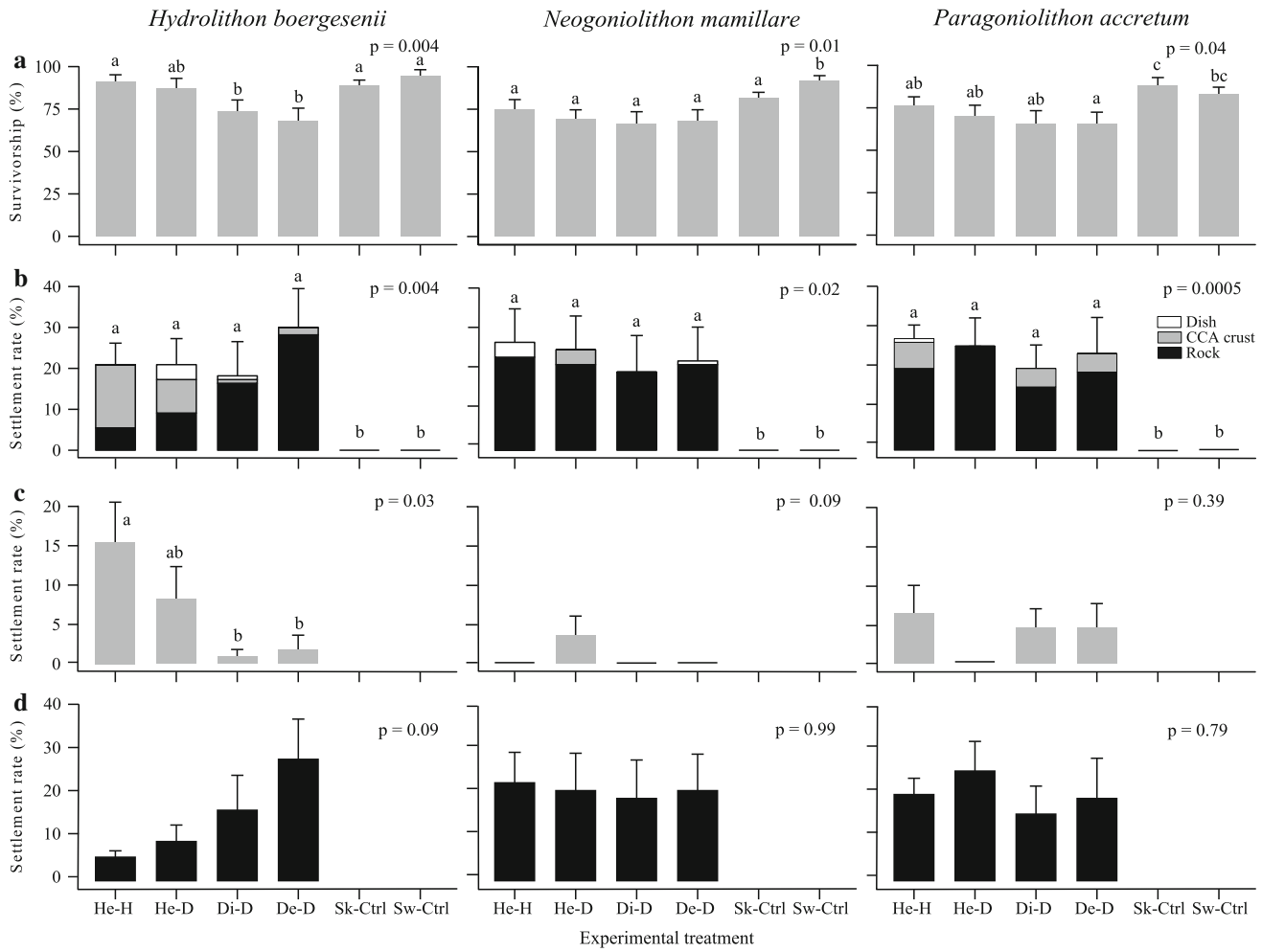

Fig. 2 Survivorship and settlement rates (mean ± SE, $n = 10$) of *Orbicella faveolata* planulae for each crustose coralline algae species (*columns*). **a** Survivorship. **b** Total settlement. **c** Settlement on CCA surface. **d** Settlement on limestone rock underneath the CCA surface. Treatment abbreviations are described in the "Materials and methods". *Letters* indicate homogeneous subgroups determined by pairwise a posteriori comparisons following PERMANOVA. See ESM Tables S3, S4 for full statistical results

O. faveolata larvae ($n = 1$) had settled on top of diseased fragments to make a similar comparison.

Discussion

The survival of *O. faveolata* in the presence of two CCA species (*N. mamillare* and *P. accretum*) was lower than in one of the two controls regardless of their disease status. Although this suggests a negative effect of these CCA on *O. faveolata* larvae, we should consider that this effect was not consistent across both controls for both CCA species, and not observed in *D. labyrinthiformis*. To our knowledge, no negative effect of CCA on the survival of coral larvae has been reported to date. However, in the presence of healthy *N. mamillare* and *P. accretum*, settlement rates in both coral species were also low, especially on the top of the crust, indicating that these two species are ineffective cues for settlement. Negative effects of CCA on coral settlement are well known and result mainly from chemical deterrents used as natural antifoulants (Harrington et al. 2004). An allelopathic substance from the CCA, *Lithophyllum* spp., destroys zoospores from the brown alga *Laminaria religiosa* (Suzuki et al. 1998). It is thus plausible that these two CCA species release chemical deterrents that deter both larval survival and settlement.

In contrast, the survival of *O. faveolata* larvae was similar in the presence of healthy *H. boergesenii* than in both controls, suggesting no negative effect of this CCA species on coral larvae, but declined in the presence of diseased and dead fragments of diseased *H. boergesenii*, suggesting a negative impact of CCA disease on larval survival. *Orbicella faveolata* larvae could be sensitive to microbes associated with the disease and/or metabolites

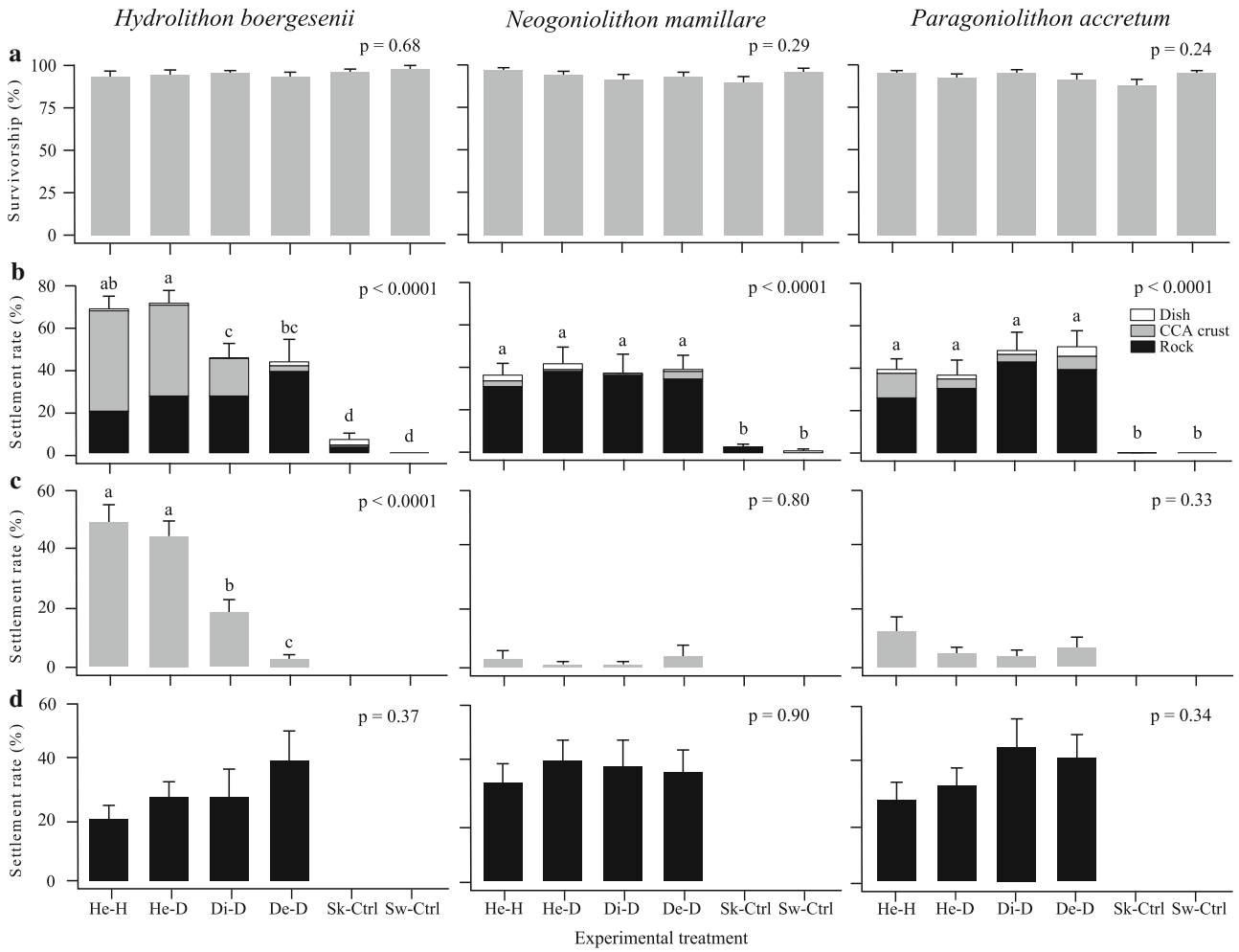

Fig. 3 Survivorship and settlement rates (mean ± SE, $n = 10$) of *Diploria labyrinthiformis* planulae for each crustose coralline algae species (*columns*). **a** Survivorship. **b** Total settlement. **c** Settlement on CCA surface. **d** Settlement on limestone rock underneath the CCA surface. Treatment abbreviations are described in the "Materials and methods". *Letters* indicate homogeneous subgroups determined by pair-wise a posteriori comparisons following PERMANOVA. See ESM Tables S3, S4 for full statistical results

produced by secondary invaders colonizing the dead CCA crust killed by the disease. To date, the causal agents responsible for CWBS and CWPD are unknown, but some CCA diseases are associated with bacteria and fungi (Littler and Littler 1995; Williams et al. 2014), which are well-known coral pathogens (Sutherland et al. 2004). In addition, dead CCA are rapidly colonized by a wide variety of microorganisms (Tribollet and Payri 2001; Ghirardelli 2002). A comparison between live and dead thalli in coralline red algae revealed a more abundant and diverse community of microorganisms (cyanobacteria, chlorophyta, and fungi) in dead thalli (Ghirardelli 2002). Algal turfs, macroalgae, and benthic cyanobacteria can negatively impact coral larval survival, most likely due to chemicals (e.g., allelochemicals or dissolved organic carbon) and/or pathogens associated with algae (Kuffner et al. 2006; Vermeij et al. 2009; Paul et al. 2011; Olsen et al. 2014).

The detrimental effect of CCA disease on larval survival was not found in *D. labyrinthiformis* larvae. *D. labyrinthiformis* reached competency after two days and exhibited high settlement rates (>37 %), whereas *O. faveolata* reached competency after 4 days and exhibited lower settlement rates (≤30 %). In a comparable study, *O. faveolata* settlement rates reached about 42 % in the presence of healthy *H. boergesenii* and 37 % in the presence of healthy *N. mamillare* (Ritson-Williams et al. 2014), while in our study, the rates reached only 21 and 26 % for the same species, respectively. It is thus possible that larvae of *D. labyrinthiformis* were healthier than larvae of *O. faveolata*. Alternatively, coral species' sensitivity toward CCA disease could vary. It has been demonstrated that coral species are not equal in the face of environmental stress (Miller et al. 2009; Hartmann et al. 2013; Miller 2014). This is the first report of in vivo larval survival and

settlement rates in *D. labyrinthiformis*. The percentage of settlement reached over 60 % in the presence of healthy *H. boergesenii*, which is in the same range as the rates obtained with seven-day old larvae from *Acropora palmata* in a comparable study (Ritson-Williams et al. 2010).

Our results confirm that *H. boergesenii* is a strong settlement inducer for corals, which is consistent with earlier studies (Morse et al. 1994; Ritson-Williams et al. 2010, 2014). However, they also show that the ability of this species to induce settlement is reduced when affected by disease. It is unclear whether this is the result of a loss in morphogens, bacteria, or spectral signature associated with healthy CCA tissue (Morse et al. 1994; Heyward and Negri 1999; Webster et al. 2004; Mason et al. 2011). However, the lack of differences in settlement between healthy fragments of healthy and diseased crusts and the preference of larvae for healthy tissue when offered diseased fragments suggest that the mechanism operates on a millimeter scale.

Disease did not affect settlement rates in *N. mamillare* and *P. accretum*. Contrary to *H. boergesenii*, these two species may not possess the morphogen(s) responsible for the activation of coral larvae metamorphosis and settlement. Nevertheless, settlement still occurred on diseased and dead *H. boergesenii* as well as on the other less inductive species (though mostly on the bottom side of the fragments) regardless of their disease status. Thus, living CCA are not essential to induce settlement. Microbes not associated with living CCA have been shown to induce coral metamorphosis (Negri et al. 2001; Webster et al. 2004). They may induce settlement without being influenced by the health of their underlying substrata.

The results of this study are experimental and need to be tested in situ. However, this study adds a further concern for the maintenance and recovery of coral reefs. Little is known about CCA communities in situ (Aeby et al. 2008; Vargas-Ángel 2010; Tribollet et al. 2011; Quéré et al. 2015). Disease outbreaks could lead to profound changes in these communities with cascading effects on coral recruitment (Doropoulos et al. 2012; Miller et al. 2013). Information on the status of CCA in the field, particularly the long-term effect of diseases on their populations, is also needed to understand their potential for coral recruitment and the trajectory of reef communities in the face of climate change.

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