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REPORT

Effects of frequent fish predation on corals in Hawaii

D. Jayewardene · M. J. Donahue · C. Birkeland

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Abstract The abundance of lesions from fish bites on corals was quantified at nine shallow reefs in the main Hawaiian Islands. There were on average 117 bite scars m^{-2} on *Pocillopora meandrina* tissue from the barred filefish *Cantherhines dumerilii*, 69 bites m^{-2} on *Porites compressa* tissue, and 4 bites m^{-2} on *Porites lobata* tissue from the spotted puffer *Arothron meleagris*. Across sites, the frequency of *A. meleagris* bites on *P. compressa* per unit area of living coral cover declined exponentially with increasing coral cover. *P. compressa* nubbins in two size classes (1–2 cm and 4–5 cm) were transplanted onto six study reefs. Nubbins in the small size class were entirely removed by bites from *A. meleagris*, while nubbins ≥4 cm were only partially consumed, leaving them able to recover. At sites with abundant *P. compressa*, predation had little effect on transplanted nubbins; at sites where *P. compressa* comprised less than 5% of living cover, all nubbins were preyed upon. *A. meleagris* bite lesions on *P. compressa* were monitored through time and fully recovered in 42 ± 4 days. A model of the risk of over-predation (a second predation event before the first is healed) decreased exponentially with increasing coral cover and increased linearly with increasing lesion healing time. The increased risk of over-predation at low coral cover could indicate an Allee effect limiting the recovery of coral populations if coral cover is substantially reduced by natural or anthropogenic disturbances.

Keywords Coral · Predation by fishes · Corallivory · *Arothron* · *Porites* · Hawaii

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Introduction

Over one hundred species of fishes are known to feed on corals (Rotjan and Lewis 2008). Although fish corallivory is common and widespread (Rotjan and Lewis 2008; Cole et al. 2008), the effects of predation on coral have not been as extensively documented as the effects of fish herbivory on algae. While there are studies that indicate that predation can limit the growth rate, competitive ability, and distribution of corals (Neudecker 1979; Wellington 1982; Cox 1986; Littler et al. 1989; Grottoli-Everett and Wellington 1997), the net effect of fish predation on the coral community has been considered to be less important than the indirect effects of herbivorous fishes on coral (Hixon 1997).

Predation by fishes on corals may be analogous to continuous browsing (partial consumption) on plants in the terrestrial environment by herbivores (Hairston et al. 1960). Thus, corals (=producers) might be limited by competition for resources (e.g., space), not predation, and the corallivores (=primary consumers) might be limited by factors such as predation, shelter and recruitment, not by food (Glynn 1985; Guzman and Robertson 1989). Continuous predation is nonetheless an energy drain for the coral through allocation of resources to tissue regeneration (Meesters et al. 1994), and in the face of recent additional threats to coral reefs and diminishing coral populations,

may become an increasingly influential factor for coral resilience to anthropogenic changes (Rotjan and Lewis 2008).

On Hawaiian shallow reefs it is common to see lesions caused by fish bites on the live tissue of coral colonies where the bare skeleton is exposed. While there is a general perception that parrotfishes are responsible for these lesions, less than 2% of parrotfish bites are inflicted on live coral, and these are generally along the edge where the coral tissue meets the algae (pers. obs., Ong 2007). The majority of bite lesions in the Main Hawaiian Islands are inflicted by the barred filefish (*Cantherhines dumerilii*) and the spotted puffer (*Arothron meleagris*), which have been observed by the authors to remove coral skeleton and inflict lesions when feeding on coral (Jayewardene and Birkeland 2006). They are, however, very skittish and thus seldom seen in action by divers. All but one of Hawaii's 13 species of corallivorous butterflyfishes do not inflict visible lesions because they feed only on the tissue of coral polyps (Randall 2007). The teardrop butterflyfish (*Chaetodon unimaculatus*) is the exception, but it usually has only been seen to bite off small amounts of skeletal material from the rice coral (*Montipora capitata*) (E. F. Cox, pers. comm.).

The goal of this study was to determine the role that corallivory by *C. dumerilii* and *A. meleagris* plays in influencing coral community structure in the Main Hawaiian Islands. The specific objectives were: (1) to quantify the level of fish predation on corals by surveying a range of reefs in the islands for the prevalence of characteristic bite lesions left by *A. meleagris* and *C. dumerilii*; (2) to determine the intensity and immediate effect of predation on coral by exposing two size classes of out-planted *Porites compressa* branches to natural levels of predation on the reef; (3) to establish the healing time for bite lesions inflicted by *A. meleagris* on *P. compressa* by monitoring in situ tissue regeneration; and (4) to examine whether fish predation could pose a risk to coral recovery.

Materials and methods

Study sites

The study was conducted from March 2004 to August 2005 at 12 sites on nine shallow coral reefs located in the Main Hawaiian Islands (Fig. 1). Two sites were "open" sites with unregulated fishing (Portlock and Wawaloli Beach), seven sites had "partial" management prohibiting lay net fishing (Puako, Anaehoomalu Bay, Wawaloli Fisheries Management Area (FMA), Wawaloli $FMA_{shallow}$, Papawai, Keei, and $Keei_{shallow}$), and three sites were "no-take" Marine Life Conservation Districts, in which all fishing was prohibited (Hanauma Bay, $Hanauma\ Bay_{shallow}$, and

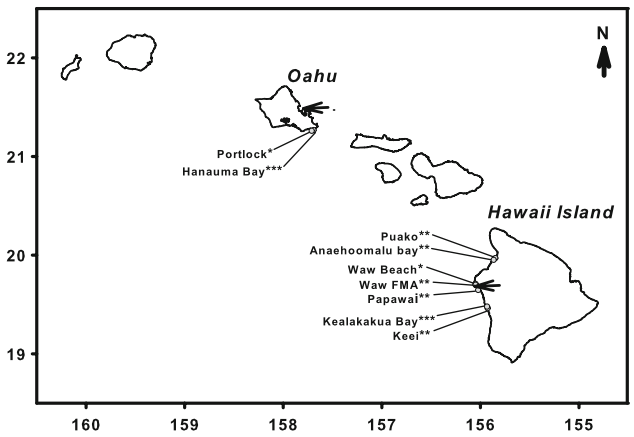

Fig. 1 Map of the Main Hawaiian Islands showing study sites. Sites marked with * indicate "open" sites where fishing was unregulated, ** indicates sites with "partial" protection where lay netting was prohibited, and *** indicates "no-take" sites where all fishing was prohibited. *Arrows* indicate locations where nubbins were collected for transplant experiments

Kealakakua Bay). The shallow areas were included in the study to increase the range in the background cover of *P. compressa* found across study sites. Surveys and experiments at each site were conducted within an area of $\sim$1,000 m^2 overlapping the locations of fixed fish survey transects (Tissot and Hallacher 2003, Jayewardene 2009). The depth of these areas ranged between 6 and 12 m.

Coral abundance

Coral cover within each 1,000 m^2 study site was determined by analyzing fifteen 40×60 cm random photoquadrats along each of 10 randomly located 20 m transects (=150 photoquadrats/site). Photogrid^{TM} image analysis software was used to analyze 20 random points per photoquadrat. Differences in mean coral cover among sites were analyzed using one-way ANOVA.

Abundance of fish bites

Although *C. dumerilii* and *A. meleagris* were observed on study reefs, they were generally seen swimming away at a distance and/or hiding under *Porites lobata* heads. The skittish behavior prevented accurate quantification of the densities of the fishes using either belt transects or the zig-zag survey method used by Guzman and Robertson (1989). As fishes with this behavior are not properly represented by visual count data (Jennings and Polunin 1995), the number of bite lesions found on live coral was used instead as a metric to determine the prevalence of predation on coral by *C. dumerilii* and *A. meleagris*.

Bites were identified as open lesions on live coral with exposed coral skeleton. These were clearly distinguishable

from lesions caused by physical damage on the basis of their appearance and by the presence of identifiable toothmarks. Bite lesions were categorized as inflicted by either *A. meleagris* or *C. dumerilii* and were characterized by observing individuals of each of these species feeding on coral in the field. *A. meleagris* feeding on *P. compressa* was focused on the tips of colony fingers resulting in single concave lesions (Fig. 2a). *A. meleagris* feeding on *P. lobata* occurred across the colony surface resulting in opposing sets of parallel lesions (Fig. 2b) or, when focused in one area, in lesion patches of varying size (Fig. 2c). *C. dumerilii* feeding on *P. lobata* resulted in small indistinguishable lesions, while feeding on *Pocillopora meandrina* focused on the tips of colony branches resulting in single concave lesions (Fig. 2d). Lesions inflicted by corallivorous fishes were easily distinguished from lesions inflicted by parrotfishes, which were thin, more numerous, and haphazard (Fig. 2e).

The number of bite lesions on coral was determined by surveying five independent 20 m transects within each 1,000 m^2 area. Ten 0.5 × 0.5 m quadrats were randomly placed along each transect, and discrete bites within each quadrat were identified as either (1) *A. meleagris* lesions on *P. compressa*, (2) *A. meleagris* lesions on *P. lobata*, or (3) *C. dumerilii* lesions on *P. meandrina*. Predation pressure, i.e., number of bites per square meter of reef divided by the abundance of each coral species (bites m^{-2} of coral tissue), was calculated to enable comparisons among species and sites.

Differences in bite density (bite m^{-2}) and predation pressure (bites m^{-2} coral tissue) among sites were analyzed using non-parametric Mann–Whitney and Kruskall–Wallis tests. Differences in *A. meleagris* predation on *P. lobata* versus *P. compressa* were examined using a binomial test, comparing observed bites (total number of bites recorded per species per site) and expected bites (calculated assuming that bites are proportional to coral cover per species per site).

Predation experiment

Predation was experimentally measured by transplanting *P. compressa* branches (nubbins) to eight sites (Wawaloli Beach, Puako, Wawaloli FMA, Wawaloli $FMA_{shallow}$, Keei, $Keei_{shallow}$ Kealakakua Bay, $Hanauma_{shallow}$), exposing the nubbins to ambient levels of predation. To test for differences in predation on different fragment sizes of coral, two size classes of nubbins were set out at each site: small (1 to 2-cm tall) nubbins and large (4 to 5-cm tall) nubbins.

Nubbins transplanted at the Hanauma Bay site on the island of Oahu were collected from the reef at the Hawaii Institute of Marine Biology (HIMB) in Kaneohe Bay (Fig. 1) at 2 to 4-m depth. Nubbins transplanted at sites on Hawaii Island were collected from the reef off Honokohau harbor at 8 to 10-m depth. Sets of four small or four large nubbins were attached to 15.2 × 15.2 cm terracotta tiles using epoxy putty (1 tile = 1 replicate). Nubbins were grouped, rather than individually attached to tiles, to most closely represent a colony of *P. compressa*. Nubbins were maintained in aquaria for 2 days prior to transplantation on to the reefs. Four tiles of each size class were set out at each site totaling eight tiles per site =4 $tiles^{-1}$ size $class^{-1}$ site. Tiles were placed haphazardly within the 1,000 m^2 area among the surrounding coral and monitored once in a week for 3 weeks. Predation was defined as removal of the

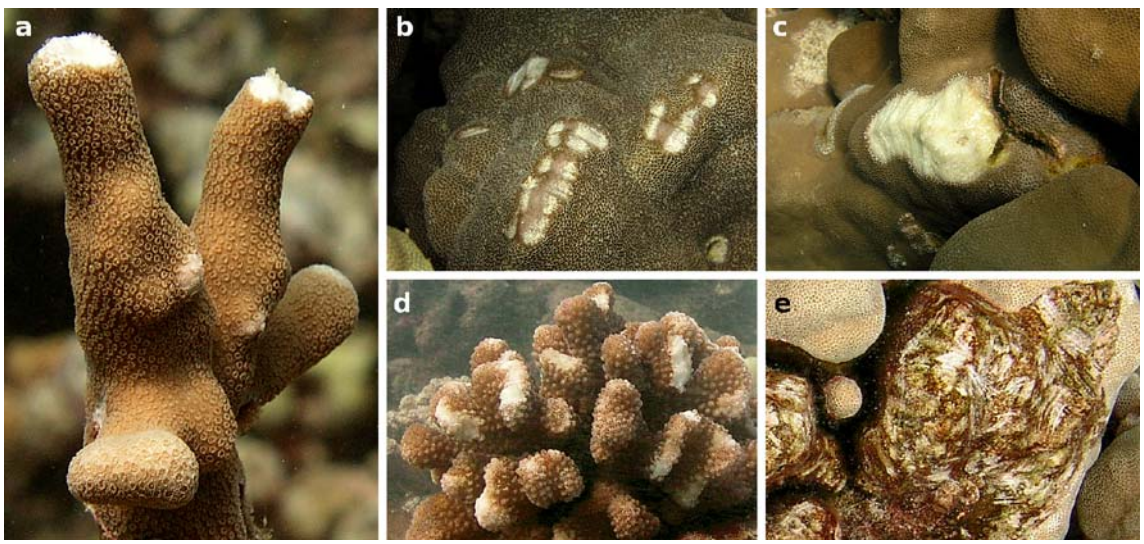

Fig. 2 Bite marks by *Arothron meleagris* on **a** *Porites compressa*, **b**, **c** *Porites lobata*, by *Cantherhines dumerillii* on **d** *Pocillopora meandrina*, and **e** by the bullethead parrotfish (*Chlorurus sordidus*) on non-coral tissue adjacent to *P. lobata*

intact tip of nubbins (=maximum 4 bites replicate tile^{-1}). Differences in predation (bites tile^{-1}) were analyzed using non-parametric Mann–Whitney and Kruskall–Wallis tests.

Healing of scars

Healing rate of *P. compressa* bite scars was determined by monitoring tissue regeneration across scars inflicted by *A. meleagris* on *P. compressa* colonies in situ. An area approximately 100 m^2 in size was chosen on Keei reef, where *P. compressa* cover was high and bite scars relatively abundant. Eight freshly inflicted bite scars at the tips of *P. compressa* branches were selected haphazardly a few meters distance apart. While some scars were in close proximity to other un-monitored scarred branches, this was deemed unimportant as tissue regeneration is fuelled by a band of tissue directly bordering the lesion (Meesters et al. 1994). Lesions were monitored for the number of days taken for tissue to begin regeneration across lesion (=healing start time) and the number of days taken for tissue to span the entire scar with no remaining sign of the bite (=full recovery time). For each *P. compressa* lesion, healing stage was regressed against time. As the exact time of bite infliction for each scar was unknown, the start time (day 0) was back-calculated from the linear regression. Average healing time was calculated from the average of the regression slopes.

Model of over-predation risk

To evaluate the risk of coral decline due to fish predation, an empirically-based model of over-predation risk as a function of bite frequency and healing time was developed. Over-predation is when a fish bites the same spot of living coral before it has completely recovered from a previous bite. Healing time (h) is the number of days required for a wound to heal. Previous work indicates that healing time is independent of coral head size and other wounds on the same coral head (Jayewardene 2009), but could depend on environmental stress (Meesters et al. 1992; Meesters and Bak 1993; Fisher et al. 2007). Bite frequency (i.e., per capita risk of predation) of *A. meleagris* on *P. compressa* at each site was calculated as area of bites (bites m^{-2} × 0.9 cm^2 per bite, Jayewardene 2009) divided by the cover of *P. compressa.* The relationship between bite frequency and coral cover was analyzed using the regression model: ln(Bite Frequency) $\sim \beta_0 + \beta_1$ *P. compressa* cover. This implies an exponential model of bite frequency, $p(C) = p_0 e^{-p_1 C}$, where p_0 is maximum predation risk at low coral cover and p_1 is the decline in predation risk with increasing coral cover, C. Assuming that bites are independent events, they can be modeled as a binomial random variable: Bites $\sim$ Binomial (h, $p(C)$), where h is the number of days before complete recovery and $p(C)$ is the daily probability of a bite. Over-predation risk is defined as Prob(Bites $\geq$ 2).

Results

Coral abundance

Average coral cover across reefs was 37 ± 4% (mean ± SE), but total coral cover varied among sites ($F_{11,83} = 26.01$, $P < 0.001$, Table 1). The three most common corals across sites were *P. lobata* 20.3 ± 10.2% (mean ± SD), *P. compressa* 10.3 ± 9.6%, and *P. meandrina* 1.5 ± 1.9%, followed by less common *Porites lutea* 0.6 ± 0.7%, *Montipora patula* 0.5 ± 0.9%, *Montipora capitata* 0.5 ± 0.5%, *Pavona varians* 0.3 ± 0.6%, and *Pocillopora eydouxi* 0.1 ± 0.2%. There was no association between coral cover and marine protection status ($F_{2,12} = 2.06$, $P = 0.170$).

Bite density and predation pressure

Based on the characteristics of the bite lesions, *A. meleagris* was responsible for inflicting all bites on *P. compressa* (a uniform concave lesion across entire tip of branch), and *C. dumerilii* for all bites on *P. meandrina* (smaller, sometimes irregular-shaped lesions on tips of branches). While some lesions were not clearly identifiable on *P. lobata*, the majority were inflicted by *A. meleagris*.

The density of fish bites on each of the three coral species (bites m^{-2}) varied among sites (Kruskall–Wallis, *P. compressa*: $H = 32.54$, $P = 0.001$; *P. lobata*: $H = 26.05$, $P = 0.004$, and *P. meandrina*: $H = 29.04$, $P = 0.001$). Bite density was not associated with coral cover (Pearson correlation, *P. compressa*: 0.159, $P = 0.623$; *P. lobata*: 0.484, $P = 0.186$; *P. meandrina*: 0.351, $P = 0.355$) or marine protection status (Kruskall–Wallis: *P. compressa*: $H = 1.44$, $P = 0.487$; *P. lobata*: $H = 0.85$, $P = 0.655$; *P. meandrina*: $H = 3.24$, $P = 0.194$). Predation pressure (bites m^{-2} of coral tissue) differed among coral species (Kruskall–Wallis: $H = 8.17$, $P = 0.017$) and was greatest on *P. meandrina* (117 ± 52 bites m^{-2}of coral tissue, mean ± SE), followed by *P. compressa* (69 ± 19 bites m^{-2} of coral tissue), and *P. lobata* (4 ± 1 bites m^{-2} of coral tissue) (Table 1). Of the two *Porites* species, *A. meleagris* fed preferentially on *P. compressa* (binomial test <0.002, Table 2).

Predation experiment

There was no significant difference in predation (bites tile^{-1}) between the small and large nubbins

Table 1 Total coral cover (%), coral cover of the three most common species (%), bite density on these species (# bites/m^{-1}) and the intensity of predation on small (1–2 cm) and large (4–5 cm) experimental *Porites compressa* nubbins (number of bites $tile^{-1}$, max = 4 bites $tile^{-1}$) across study sites

Sites	% Coral (±SE)				# Bites/m^{-1} (±SE)			# Bites/tile (±SD)	
	Total	PC	PL	PM	PC	PL	PM	Small	Large
Portlock*	8^{a} (±1)	0 (±0)	5 (±1)	2 (±1)	0.1 (±0.1)	0.4 (±0.3)	3.4 (±2.0)	n/a	n/a
Waw Beach*	42^{de} (±3)	5 (±1)	33 (±2)	2 (±0)	2.1 (±1.0)	0.9 (±0.6)	3.0 (±1.3)	0 (±0)	0.3 (±0.5)
Pauko**	58^{f} (±3)	23 (±3)	29 (±3)	1 (±0)	2.3 (±0.5)	2.6 (±1.2)	0 (±0)	0 (±0)	0 (±0)
A-bay**	39^{cde} (±1)	17 (±1)	17 (±2)	1 (±0)	2.8 (±0.9)	1.8 (±0.9)	3.4 (±2.4)	n/a	n/a
Waw FMA**	44^{e} (±2)	7 (±)	29 (±)	6 (±1)	5.2 (±1.0)	0.6 (±0.3)	1.6 (±0.7)	0 (±0)	0 (±0)
Waw FMA $_{shlw}$**	33^{bcde} (±4)	1 (±1)	25 (±3)	5 (±)	0.9 (±0.3)	0 (±0)	0 (±0)	0.5 (±0.3)	0.3 (±0.3)
Papawai**	31^{bcd} (±2)	1 (±0)	27 (±2)	1 (±0)	0.9 (±0.4)	0.9 (±0.4)	0.7 (±0.5)	n/a	n/a
Keei**	33^{bcde} (±2)	26 (±5)	5 (±1)	0 (±0)	0.3 (±.3)	0 (±0)	0 (±0)	0 (±0)	0 (±0)
Keei $_{shlw}$**	65^{f} (±2)	2 (±1)	63 (±2)	0 (±0)	1.5 (±0.7)	n/a	n/a	1.5 (±1.9)	1.6 (±1.3)
Han***	25^{b} (±2)	3 (±1)	20 (±2)	0 (±0)	1.3 (±0.5)	0.9 (±0.5)	0.2 (±0.2)	n/a	n/a
Han $_{shlw}$***	38^{bcde} (±6)	0 (±0)	19 (±2)	1 (±0)	0.2 (±0.2)	0 (±0)	0 (±0)	4 (±0)	4.0 (±0)
K-bay***	31^{bc} (±2)	11 (±3)	17 (±2)	0 (±0)	3.2 (±1.2)	0 (±0)	0 (±0)	0 (±0)	0 (±0)

Sites marked with * indicate "open" sites where fishing was unregulated, ** indicates sites with "partial" protection where lay netting was prohibited, and *** indicates "no-take" sites where all fishing was prohibited. Total coral covers with same superscript letter are not significantly different (Tukey test, $P > 0.05$). PC *P. compressa*, PL *Porites lobata*, and PM *Pocillopora meandrina*

Table 2 Observed and expected bites on *Porites compressa* (PC) and *Porites lobata* (PL) per site

Sites	Expected # bites		Observed # bites		Direction	
	PC	PL	PC	PL	PC	PL
Han	4	23	16	11	+	−
Portlock	0	6	1	5	+	−
Puako	28	34	29	33	+	−
A-bay	28	30	35	23	+	−
Waw Bch	5	32	26	11	+	−
Waw FMA	14	58	65	7	+	−
Papawai	1	22	11	12	+	−
K-bay	16	24	40	0	+	−
Keei	3	1	4	0	+	−

Binomial test: $(½)^9 = 1/512 < 0.002$

Expected number of bites was calculated assuming that bites were distributed in proportion to coral cover. Differences between observed and expected bites were assessed using a binomial test

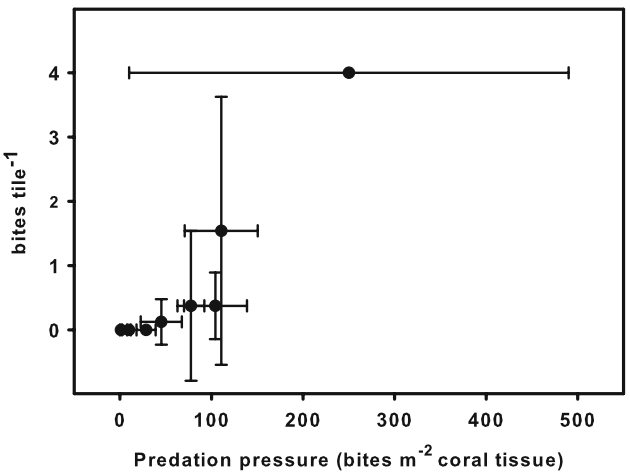

Fig. 3 Experimental predation (# of bites $tile^{-1}$) on transplanted *Porites compressa* nubbins in relation to the observed predation pressure (# of bites m^{-1} *P. compressa* tissue) by *Arothron meleagris* on *P. compressa* in the field at sites. Error bars indicate ±1 SD for *y*-axis and ±1 SE for *x*-axis

(Mann–Whitney: $P = 0.5148$), but there was a significant difference among sites (Kruskall–Wallis: $H = 42.22$, $P < 0.001$). At the $Hanauma_{shallow}$ study site, predation on all transplanted *P. compressa* nubbins occurred within the first week of monitoring. In contrast, after the full 3 weeks of monitoring, no or low levels of predation occurred at the remaining study sites. No bites were inflicted on experimental nubbins at the three sites with the highest cover of *P. compressa* (Kealakakua Bay, Puako, and Keei) (Table 1), while the highest predation (4 bites $tile^{-1}$) was at a site with very low *P. compressa* cover (3%, $Hanauma_{shallow}$). At the sites with low to intermediate abundances of *P. compressa* (Wawaloli $FMA_{shallow}$, $Keei_{shallow}$, Wawaloli Beach, and Wawaloli FMA), number of inflicted bites per tile ranged between 0 and 1.6 bites $tile^{-1}$. Predation was significantly greater on tiles at sites that had greater predation pressure (Pearson correlation: 0.946, $P < 0.001$) (Fig. 3).

While there was no difference in the number of bites inflicted on nubbins between the two size classes, predation on the small nubbins at Hanauma resulted in the entire

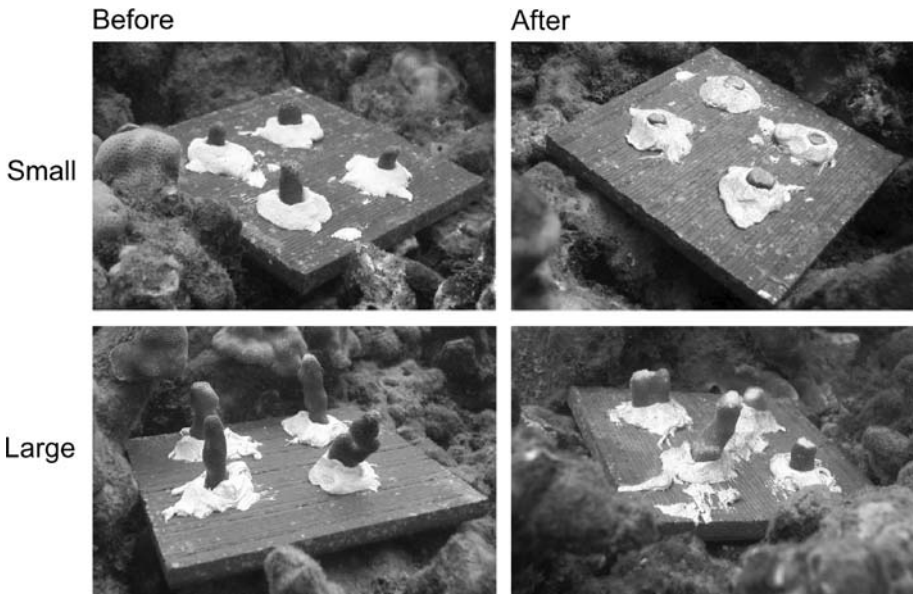

Fig. 4 Examples of experimental tiles from the $\text{Hanauma}_{\text{shallow}}$ study site illustrating complete consumption of small *Porites compressa* nubbins (1 to 2-cm tall) with a bite by *Arothron meleagris* in contrast to partial consumption of large nubbins (4 to 5-cm tall)

biomass of the nubbin being removed, while predation on the large nubbins resulted in only partial consumption (Fig. 4).

Healing of predation scars

All *P. compressa* scars healed fully without any visible negative effect on the colony branches. Healing started 8 ± 2 days (mean $\pm$ SE) following infliction of bites and lesion closure occurred at 42 ± 4 days (mean $\pm$ SE). During this period, the exposed calcium carbonate within scars was not colonized to any significant extent by algae.

Model of over-predation

Bite frequency of *A. meleagris* on *P. compressa* declined exponentially with *P. compressa* cover from a maximum bite frequency of $p_0 = 0.00964$ (0.00481, 0.0193, 95% CI) at rate $p_1 = 0.136$ (-0.195, -0.0777) ($F_{1,10} = 27.3$, $P < 0.0004$, $R^2 = 0.73$, Fig. 5). The risk of over-predation

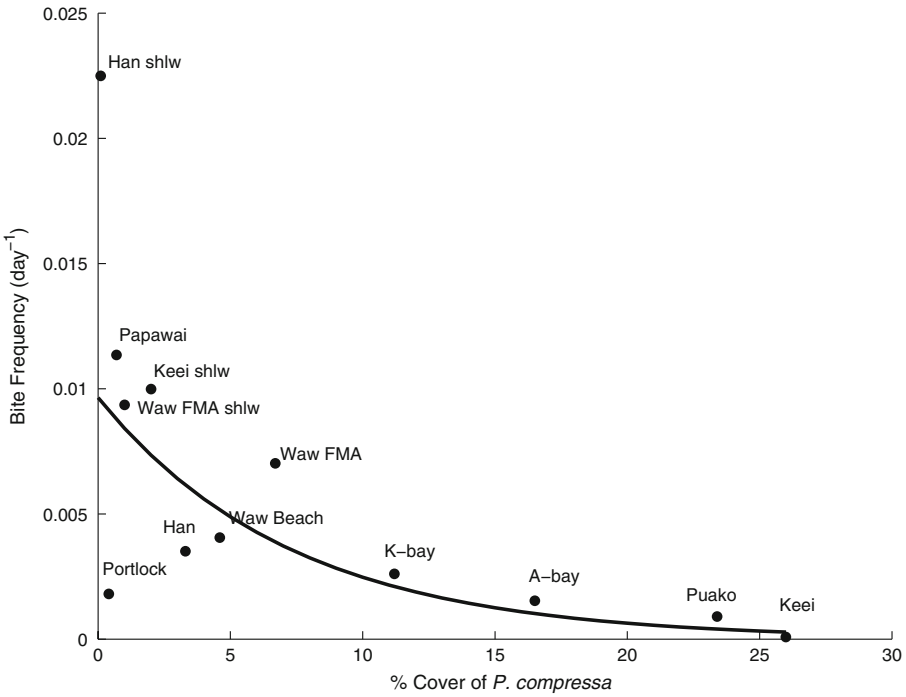

Fig. 5 Bite frequency by *Arothron meleagris* versus percent cover of *Porites compressa*. The line is back-transformed from the regression model: ln (Bite Frequency) $\sim \beta_0 + \beta_1$ % cover of *P. compressa*, ($F_{1,10} = 27.3$, $P < 0.0004$, $R^2 = 0.73$)

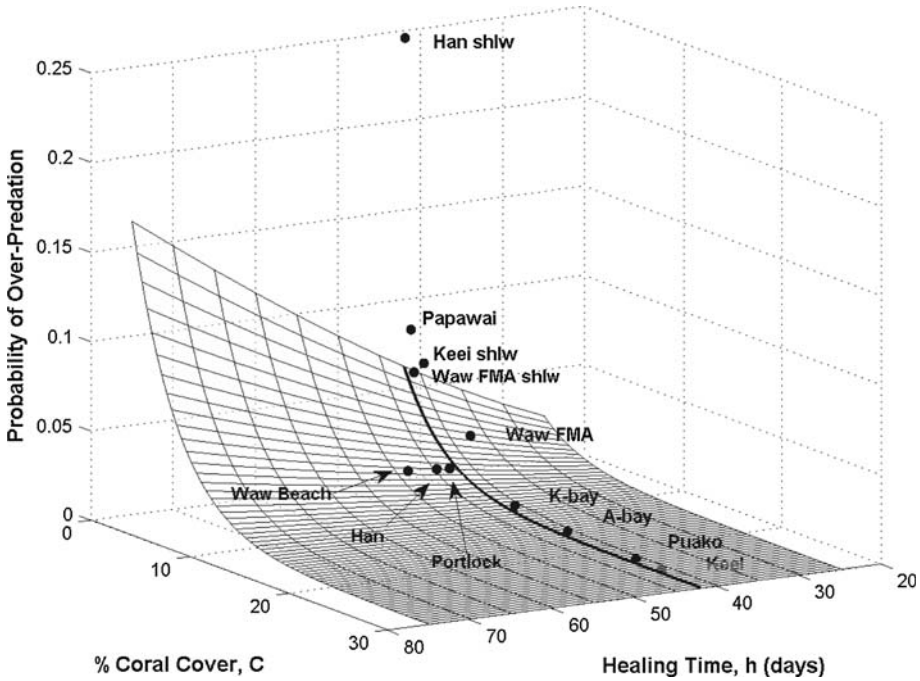

Fig. 6 Probability of over-predation versus coral cover (C) and healing time (h). The surface is the model prediction as a function of C and h, the line is the model prediction for the measured healing time $h = 42$ days. The points are the site-specific probabilities of over-predation, based on site-specific bite frequencies (Fig. 5) and $h = 42$ days

decreased exponentially as coral cover increased and increased linearly with healing time (Fig. 6).

Discussion

This study determined that corallivory by fishes is widespread and often intense on the three most common species of coral in Hawaii. The density of fish bites on the reef, a reflection of the abundance of corallivorous fishes, was not related to coral cover or marine protection status, indicating that these fish populations are not tightly regulated by prey abundance or fishing pressure. Observations coupled with surveys indicated that *C. dumerilii* is responsible for bites on *P. meandrina*, and *A. meleagris* for bites on *P. lobata* and *P. compressa.* While it is possible that corallivorous fishes other than *A. meleagris* and *C. dumerilii* are responsible for inflicting some of the surveyed bite lesions, there has been no documentation found for this on Hawaiian reefs. The spotted knifejaw (*Oplegnathus punctatus*) has been observed preying on *Pocillopora eydouxi* in the Northwestern Hawaiian Islands (Musburger pers.com.), but individuals are extremely rare in the Main Hawaiian Islands.

A. meleagris preferentially fed on *P. compressa*, a branching endemic species which provides essential habitat for various reef fishes in Hawaii (Walsh 1987; DeMartini and Anderson 2007; Ortiz and Tissot 2008). Despite each bite involving substantial removal of *P. compressa* skeletal material, tissue regeneration across lesions was efficient and complete. Even very small *P. compressa* fragments (1-cm tall) have been found to heal successfully without suppressing growth of the coral (Jayewardene 2009). The transplant experiment, however, indicated that intense predation may have detrimental cropping effects on small fragments (<1 to 2-cm tall) through removal of their entire biomass when the abundance of *P. compressa* in the community falls below a threshold (<5%). Although predation on experimental fragments may have been unrealistically high due to the novelty of transplant nubbins on the reef, the relative differences in predation intensity between sites and the outcome of whole fragment removal remains relevant.

Bite frequency decreased exponentially with increasing abundance of coral prey (Fig. 5). This is similar to a Type II functional response, in which per capita predation risk declines with increasing prey abundance. The risk of over-predation decreased rapidly with increasing coral cover and increased with healing time (Fig. 6). While corallivory is inconsequential to population growth when *P. compressa* is above 10–15% cover and lesions heal efficiently, it may limit population growth on reefs where coral cover is low and/or healing rate is compromised by environmental stress (Meesters et al. 1992; Meesters and Bak 1993; Fisher et al. 2007). This increased risk of corallivory at low coral cover could indicate an Allee effect (a decline in population growth rate at low density), limiting the recovery potential of coral populations. In Panama and Jamaica, for example, sustained levels of predation following events that caused extensive coral mortality had a substantial negative impact on the coral (Glynn 1985; Knowlton et al. 1990). While corallivorous fish populations are likely to be negatively

affected by declining coral cover in the long term (Jones et al. 2004, Berumen and Practchett 2006), predation may remain high in the short term if fish populations respond more slowly than coral to environmental stressors (Glynn 1985; Guzman and Robertson 1989; Knowlton et al. 1990). As a result, predation may accelerate the decline of impaired reefs and pose a management challenge to reef recovery.

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