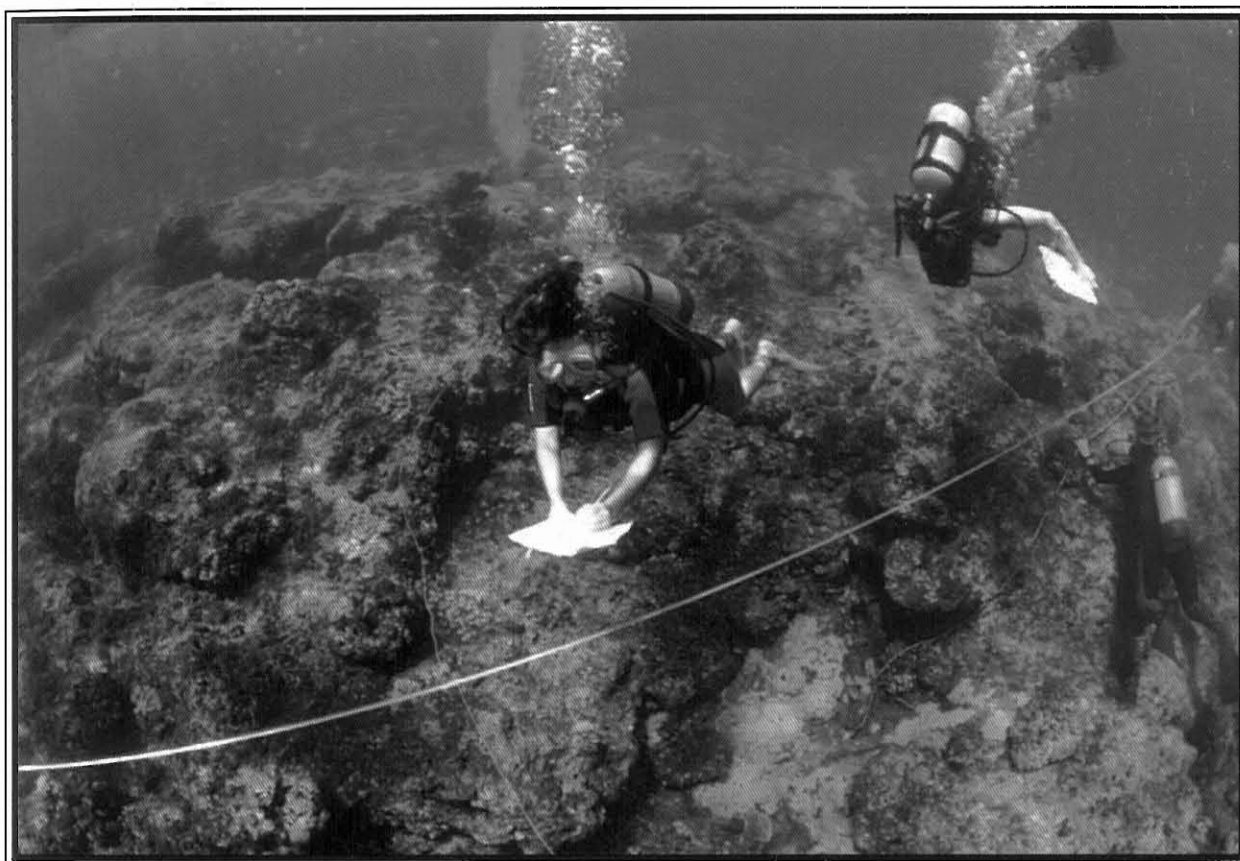


Proceedings of the Hawai'i Coral Reef Monitoring Workshop

June 9-11, 1998 Honolulu, Hawai'i

edited by
James E. Maragos and Rikki Grober-Dunsmore
February 1999



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Chapter 8:

First Records of Coral Disease and Tumors on Hawaiian Reefs

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Abstract

A variety of methods were used to quantify the distribution and temporal progression of tissue necroses and tumors on coral colonies at three sites in Hawai'i. Distribution and abundance of diseased tissues were recorded as well as changes in overall coral and algal abundance through the analysis of permanent macrophotography stations and video transects of survey areas. The incidence and extent of necroses, as well as the amount of algal turf-covered surface area, increased with increasing colony size; the incidence of tumors on coral colonies was independent of colony size. Both coral necroses and tumors usually led to the loss of coral tissue, although recovery and regeneration sometimes occurred from the edges of healthy tissue with time.

Mean coral and algal abundance varied within and among sites, as well as through time. Coral cover declined by 10% during the seven-year period of this study (1991-1998) at Hanauma Bay, Oahu and Honolua, Maui, with a concomitant increase in turf algae. Coral cover increased during the same period at Puako, Hawai'i, while mean algal cover decreased. Incidence of diseased tissues initially declined between 1991 and 1993 censuses at Hanauma Bay and Honolua and has remained relatively constant at all sites from 1993 to 1998. These studies provide the first documentation of disease and tumors in Hawai'i.

Keywords

corals, colony size, disease, tumors, videography

Introduction

In November 1990, a complaint of "sick corals" beyond the fringing reef at Hanauma Bay was brought to the attention of University of Hawai'i scientists by an underwater photographer (D. Schriete) and the Hanauma Bay park manager (A. Hong). Initial field observations confirmed the presence of tissue lesions and tumors on numerous colonies of the dominant scleractinian coral at Hanauma Bay, *Porites lobata*. Although coral diseases have been previously reported in the Indo-Pacific (Antonius 1985), and major reef areas in Florida and the Caribbean have suffered marked losses in living coral over the last two decades as a result of disease epidemics (Gladfelter 1982; Peters and Santavy 1996), there had been no previous reports of disease in Hawaiian reef corals.

A series of studies, funded by the Hawai'i Department of Land and Natural Resources/ Division of Aquatic Resources and University of Hawai'i Sea Grant, were undertaken to ascertain the spatial distribution and temporal progression of these previously undescribed diseases and to develop a preliminary evaluation of the effects of coral disease and tumors on the overall "health" of reefs in Hawai'i.

Methods

General Study Design

Initial selection of study sites was based on areas with similarly high species diversity, range of depths and exposure, and high value as state natural resource areas: Hanauma Bay, Oahu; Honolua Bay, Maui; and Puako, on the Kona coast of the Big Island. Hanauma Bay and Honolua Bay are Marine Life Conservation Districts; Puako reef is a Hawai'i State Fisheries Management Area. Censuses have also been conducted at other reef sites on Maui (Puamana, Olowalu), Hawai'i (Kealakekua), and Molokini.

The impacts of disease and tumors on reef health were assessed at three levels:

- 1) Changes in overall coral abundance on reefs (benthic video transects),
- 2) Colony-level changes (visual surveys of distributions relative to colony size, depth, distance from shore; time-series macrophotography), and
- 3) Histological analysis of diseased tissue

Temporal Changes in Coral Community Structure

Site Stratification

Each of the study sites had slightly different reef community structure and topographic profile. Therefore, within each site, survey areas were stratified based on depth, exposure and reef community characteristics; positioning deliberately avoided large sandy areas and steep slopes.

Four reef areas within Hanauma Bay were surveyed on 22 January 1992; 3 March 1992; 12 July 1992; 7 October 1992; 1 January 1993; 14 April 1993; 4 May 1994; 24 May 1995; 21 May 1997; and 13 May 1998. The defined purpose of the present study was not to assess the direct impact of visitors on the shallow, nearshore reefs of Hanauma Bay, but rather to assess the impacts of disease on overall coral abundance and reef community structure. Selected survey areas were therefore sited beyond the shallow reef crest most utilized by Hanauma Bay visitors to minimize the complicating factors of potential impacts from trampling and other user contact (Fig. 1).

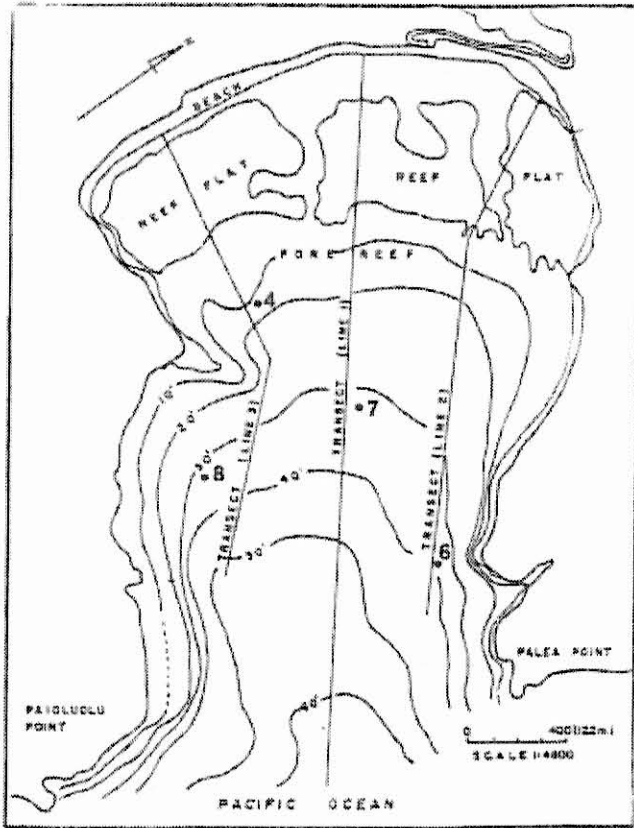


Figure 1. Map of Hanauma Bay, Oahu (from Anderson, 1978), showing study areas #H-4, 6, 7, and 8.

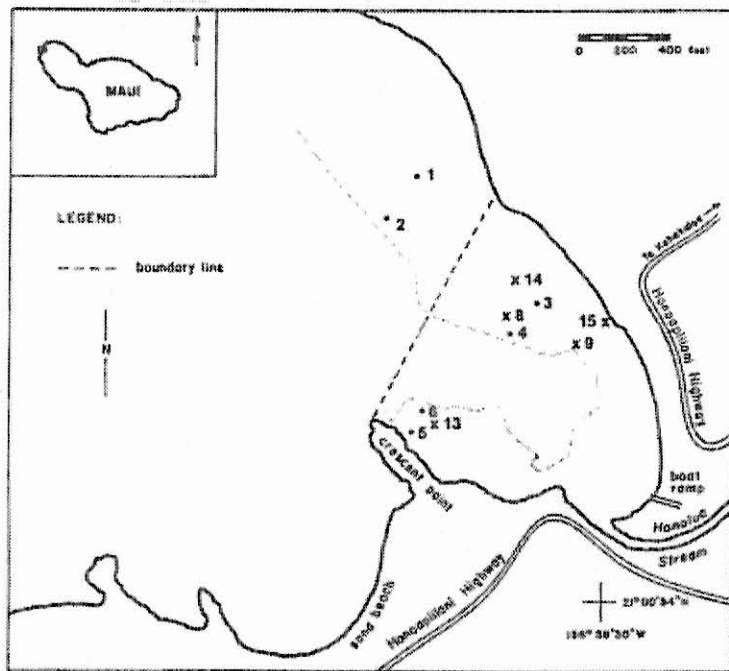


Figure 2. Map of Honolua Bay, Maui (from Torricer et al. 1979), showing study areas #M-1, 2, 3, 4, 5 and 6.

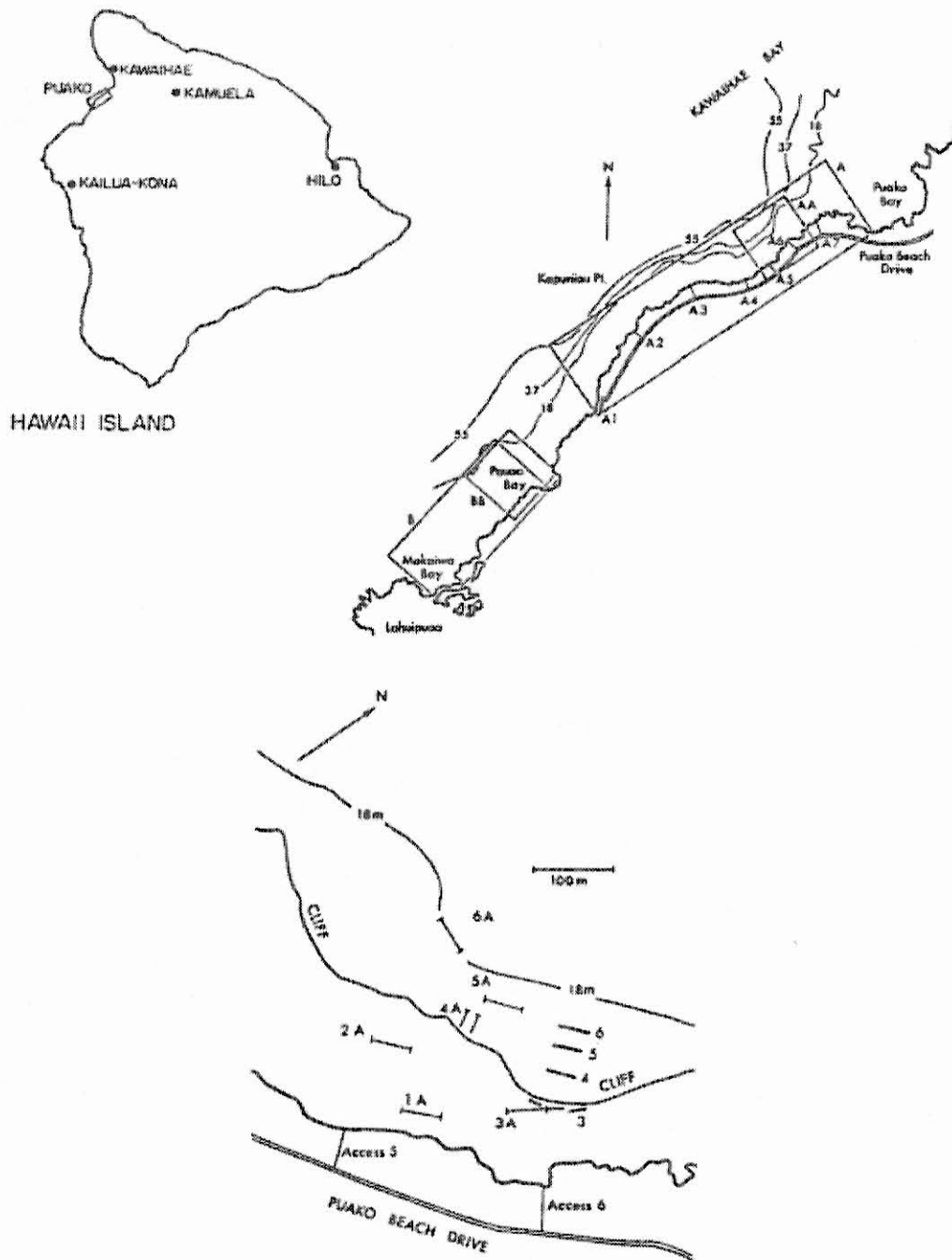


Figure 3. Map of Puako, Hawai'i (from Hayes, et al. 1982), showing study areas #P-3, 4, 5, and 6.

Six areas were selected for monitoring at Honolua Bay (Fig. 2). The majority of coral cover at this site is distributed over a shallow fringing reef at 3 m depth that drops along a fairly steep contour to a shelf at about 6 m before sloping to the sandy bottom at the center of the bay. Therefore, three areas were located at 3 m depth (two "exposed" and one "protected") and three at 6 m (similarly, two were "exposed" and one "protected"). Surveys at the Honolua site were conducted on 2 October 1991; 16 June 1992; 30 October 1992; and 19 May 1997.

The Puako site differs from Hanauma and Honolua in being on a more open coastline. The Puako reef tract is unusual along the Kohala coast of the Big Island in having a fairly extensive subtidal reef flat shoreward of underwater sea cliffs that drop to a gently sloping zone of high coral cover. Four survey areas at this site were established parallel to the shoreline on the reef flat (2 m depth) and below the sea cliffs at 9, 11, and 13 m depth (Fig. 3). Surveys at the Puako site were conducted on 28 September 1991; 24 August 1992; 11 April 1993; 7 April 1995; 26 May 1997; and 30 May 1998.

Installation of Transects

Three parallel transects were marked within each study area (4-6 areas/site). Reconnaissance surveys identified areas predominated by corals, avoiding sand channels and vertical slopes. To randomly select the exact transect locations within each area, a lead weight was dropped blindly from the surface, marking the leeward end of the central of the three parallel transects. The center ("B") transect (10 m in length) was then deployed in a seaward direction along a pre-determined compass heading from that first random point. The "A" and "C" transects (also 10m in length) were aligned 2-4 m in either direction and parallel to the center ("B") transect. The endpoints of each transect were marked with stainless steel eyebolts securely screwed into pre-drilled holes (1/4") in the coral substratum. Eyebolts were collared with 4 x 4 cm plastic tags inscribed with the transect identification codes.

A measure of spatial heterogeneity within each area was obtained by laying a chain (each link=1.42 cm) under the middle transect line, taking care that the chain conformed to the bottom topography. The length of chain necessary to cover a horizontal distance of 10 m under the transect line was then recorded. The ratio of chain length to horizontal distance along the bottom provided an index of vertical relief and estimated spatial heterogeneity at each area (Rogers et al. 1983). Substratum type under each link of the chain was also recorded, providing an additional comparative index of benthic biota and substratum abundance.

Video Surveys

A Yashica TR-81 Hi-8 video camera in a VideoSea underwater housing (JayMar Engineering, San Pedro CA) was used to record benthic biota and substratum abundance at each census period. A diver swam slowly along the length of the transect, holding the camera vertically at a distance of 0.5 m above the transect. Stilled frames from video transects recorded from this height were approximately 20 x 30 cm in area.

Auto-focus and auto-exposure features of the camera provided high-resolution records of the substratum under the transects. The ability of the camera to maximally utilize these features was substantially enhanced by the use of a rainbow-colored nylon line rather than black or white surveyor tapes. The black or white tapes caused over- or under-exposure of the underlying reef features relative to the tapes themselves. The rainbow-colored line, marked and weighted at 10 cm intervals, also provided a color scale within each video frame.

Video Analysis

Hi-8 video records were transferred to sturdier VHS tapes prior to analysis. Original Hi-8 tapes were archived for permanent data storage (University of Hawai'i, Waikiki Aquarium).

Point locations in gridded arrays (10 points/array) were randomly generated and their locations transferred to an acetate sheet cut to fit the video monitor used for analysis of tapes. The positions of ten frames along each 10 m transect were randomly determined prior to viewing the tapes (generated from random numbers between 0-1000 cm at 10 cm increments; overlaps were discarded and regenerated).

Individual video frames were displayed on a VCR monitor. For each transect, the substratum type located under 60 points (6 sets of 10 points) in each of 10 frames was recorded. Percentage data were arcsine transformed prior to statistical analysis. Statistical analysis of results of the first census period at Hanauma Bay (with 6 transects/area) demonstrated that 30 points (3 sets of 10) in 5 frames/transect for 3 transects was sufficient to describe mean coral abundance within an acceptable level of experimental error (<5%). Increasing the number of points, frames, or transects/area did not result in a further decrease in standard error of the means. Subsequent analyses therefore consisted of 30 points x 5 frames x 3 transects/area x 4-6 areas/site resulting in 1800-2700 point-samples per site per survey.

Spatial Distribution of Diseased Corals

The numerical abundance and distribution of diseased colonies of *Porites lobata* in Hanauma Bay were assessed in three surveys on 16-17 April 1992; 23 February 1994; and 27 May 1998. Six areas were selected to represent a range of depths and distances from shore within the bay. A one-time survey of colonies outside of the bay (Paioulu Point area, 13 m depth) was conducted on 6 March 1992.

At each area, the starting point of a 50 m linear transect was randomly determined by dropping a small lead weight from the surface. From this starting point, a 50 m transect line was deployed approximately parallel to the shoreline. Each colony of *P. lobata* intersecting the transect was measured along and perpendicular to its long axis. The relative proportions of healthy and diseased tissues (necroses and tumors) were estimated for each colony, along with the abundance of algal cover (macroalgae, turf, or coralline) and other features (bleaching, shrimp burrows, or fish bites) on the colony surface.

Temporal Progression of Disease

Diseased areas on *Porites* colonies were marked by drilling two 1/4" holes into the coral skeleton below the affected area. Expandable polypropylene anchor bolts were pushed through plastic tags (2"x2") inscribed with identifying numbers and tapped into one of the holes until flush with the colony surface. A 3/8" x 6" stainless steel peg with a square cross section was tapped into the other hole leaving 2-3 cm of the peg protruding. The top corner of each peg was filed down to indicate orientation of the camera framer. Tags and pegs fit snugly into the drilled holes and have remained in place for more than six years.

A Nikonos V camera with automatic exposure and flash was used for all macro-photography. A 3/8" square socket was affixed to the distal end of a Nikonos close-up kit frame extension bar, allowing an aligned fit with the stainless steel pegs. A label strip with corresponding identification numbers, date, location and a ruler scale was attached to the upper edge of the frame. At each survey, a diver aligned and fitted the socket on the close-up frame over the end of the stainless steel peg below each diseased

Table 1. Summary of chain transect data and indices of spatial heterogeneity measured at "B" transects for all sites.

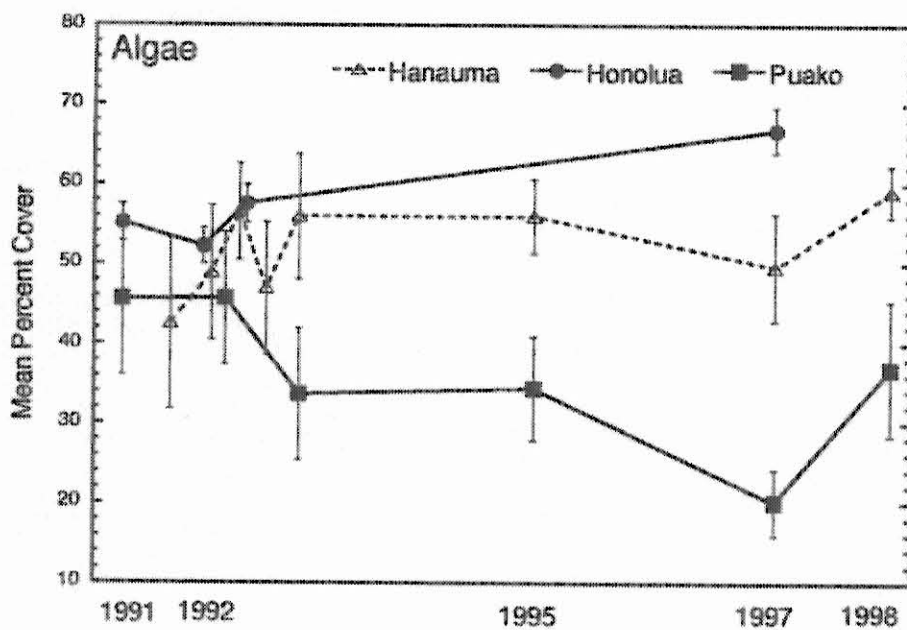
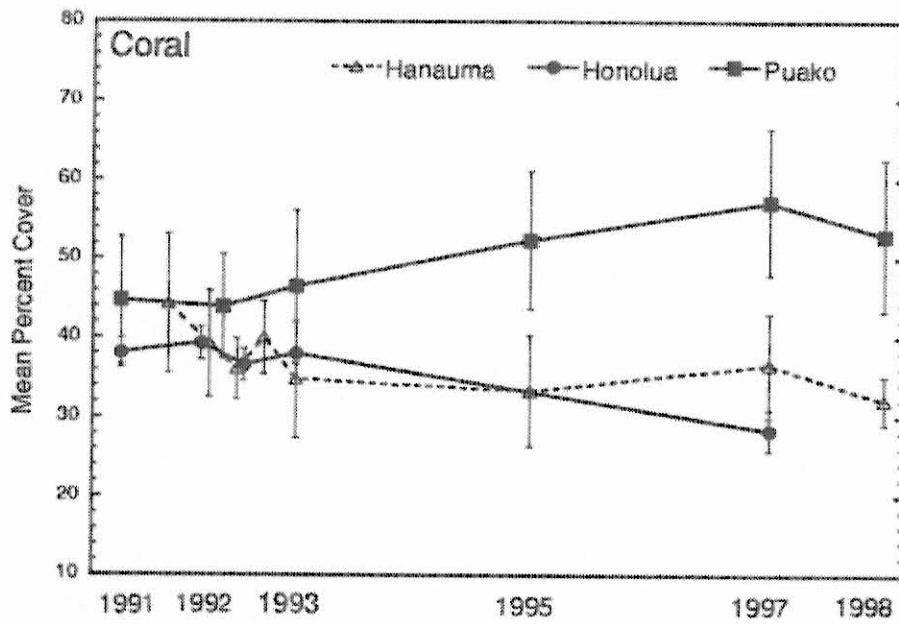
HANAUMA BAY		PL	PC	TURF	CA	PALY	PAV	PAV	POC	MON	MON	MON	MON	ECHIN	HET	DEAD	TOTAL	TRANS	SPATIAL	
							DUER	VAR	MEAN	FLAV	PAT	VERR	DIL	MATT	CENT	PM	SAND	LINKS	LGTH (M)	HET.
SITE 4	SUM	325	30	512	15	6	18	0	0	0	0	0	0	0	2	0	0	908	10.1	1.26
	RATIO	0.36	0.03	0.56	0.02	0.01	0.02	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00		721.0	
SITE 6	SUM	176	228	62	441	0	0	0	0	0	0	0	0	2	0	0	67	976	10.0	1.37
	RATIO	0.18	0.23	0.06	0.45	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.07		714.0	
SITE 7	SUM	321	20	550	12	0	0	0	0	0	0	0	0	43	0	0	5	951	10.0	1.33
	RATIO	0.34	0.02	0.58	0.01	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.05	0.00	0.00	0.01		714.0	
SITE 8	SUM	136	195	355	329	0	0	0	0	0	0	0	0	0	0	0	0	1015	10.4	1.37
	RATIO	0.13	0.19	0.35	0.32	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00		743.0	
HONOLUA																				
SITE 1	SUM	115	0	572	6	0	0	0	21	11	15	5	0	66	0	0	0	811	10.0	1.14
	RATIO	0.14	0.00	0.71	0.01	0.00	0.00	0.00	0.03	0.01	0.02	0.01	0.00	0.08	0.00	0.00	0.00		714.0	
SITE 2	SUM	89	11	426	0	0	0	1	47	0	138	40	5	4	0	40	20	821	10.0	1.15
	RATIO	0.11	0.01	0.52	0.00	0.00	0.00	0.00	0.06	0.00	0.17	0.05	0.01	0.00	0.00	0.05	0.02		714.0	
SITE 3	SUM	333	99	364	10	0	0	7	10	21	112	38	0	0	0	0	0	994	10.5	1.35
	RATIO	0.34	0.10	0.37	0.01	0.00	0.00	0.01	0.01	0.02	0.11	0.04	0.00	0.00	0.00	0.00	0.00		739.0	
SITE 4	SUM	173	312	234	119	0	1	31	0	0	3	64	0	0	0	40	81	1058	10.7	1.41
	RATIO	0.16	0.29	0.22	0.11	0.00	0.00	0.03	0.00	0.00	0.00	0.06	0.00	0.00	0.00	0.04	0.08		753.0	
SITE 5	SUM	73	9	465	25	0	0	6	0	38	75	25	0	11	0	0	75	802	10.0	1.12
	RATIO	0.09	0.01	0.58	0.03	0.00	0.00	0.01	0.00	0.05	0.09	0.03	0.00	0.01	0.00	0.00	0.09		714.0	
SITE 6	SUM	158	63	355	64	2	0	3	12	1	88	143	0	6	0	0	7	902	10.3	1.24
	RATIO	0.18	0.07	0.39	0.07	0.00	0.00	0.00	0.01	0.00	0.10	0.16	0.00	0.01	0.00	0.00	0.01		725.0	
PUAKO																				
SITE 4	SUM	295	93	206	311	0	0	0	0	0	0	3	0	2	4	0	0	914	10.3	1.26
	RATIO	0.32	0.10	0.23	0.34	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00		725.0	
SITE 5	SUM	206	261	228	45	0	0	3	0	0	2	12	0	3	0	0	136	896	10.0	1.25
	RATIO	0.23	0.29	0.25	0.05	0.00	0.00	0.00	0.00	0.00	0.00	0.01	0.00	0.00	0.00	0.00	0.15		714.0	
SITE 6	SUM	155	503	68	105	0	0	2	0	0	0	8	0	3	0	0	64	908	10.0	1.27
	RATIO	0.17	0.55	0.07	0.12	0.00	0.00	0.00	0.00	0.00	0.00	0.01	0.00	0.00	0.00	0.00	0.07		714.0	

area and photographed the framed area using Ektachrome 100 slide film. Total area of photographs within the close-up frame (excluding the label strip) was 11 x 18.5 cm.

A total of thirty-six diseased colonies was photographed over a 6.5 year period at Hanauma Bay (October 1991-May 1998); 18 areas were photographed between October 1991-October 1992 at Honolua and between September 1991-April 1993 at Puako. Images from selected slides were transferred to Kodak compact disc for permanent storage and analysis.

Diagnosis and Pathology of Disease

Coral samples were collected from Hanauma Bay under permit from the State of Hawai'i, Department of Land and Natural Resources. Cores (5 cm diameter x 5 cm thick) were taken from representative diseased areas on four colonies of *Porites lobata* in 1-2.5 m depth at Hanauma Bay. Samples were placed in fresh Helly's fixative immediately upon return to the beach. After 24 h, the cores were thoroughly rinsed in running water and transferred to 70% ethanol for 72 h. Cores were then packed in ethanol-soaked diaper and shipped to the USEPA Environmental Research Laboratory at Narragansett, RI, where they were processed for histological analysis.



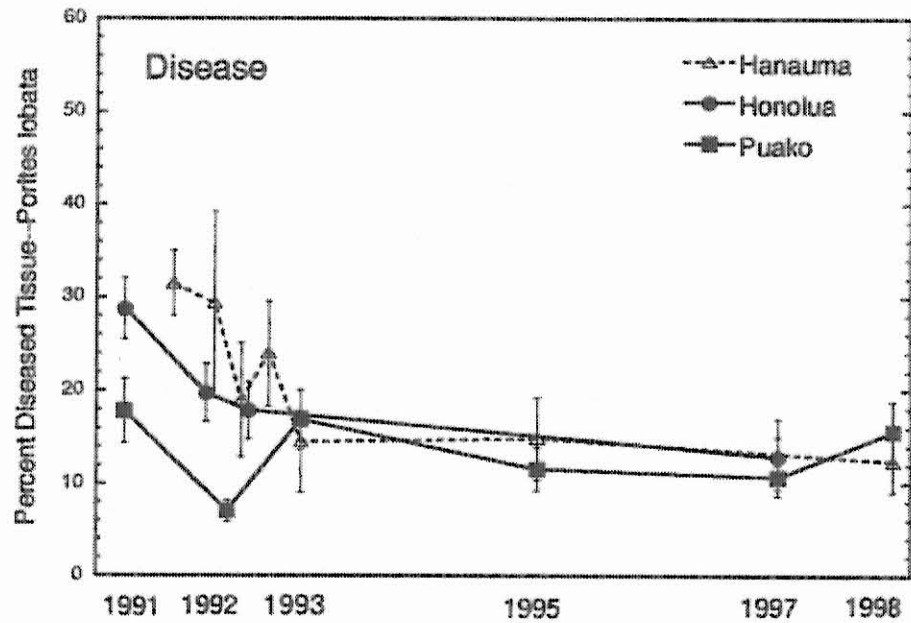
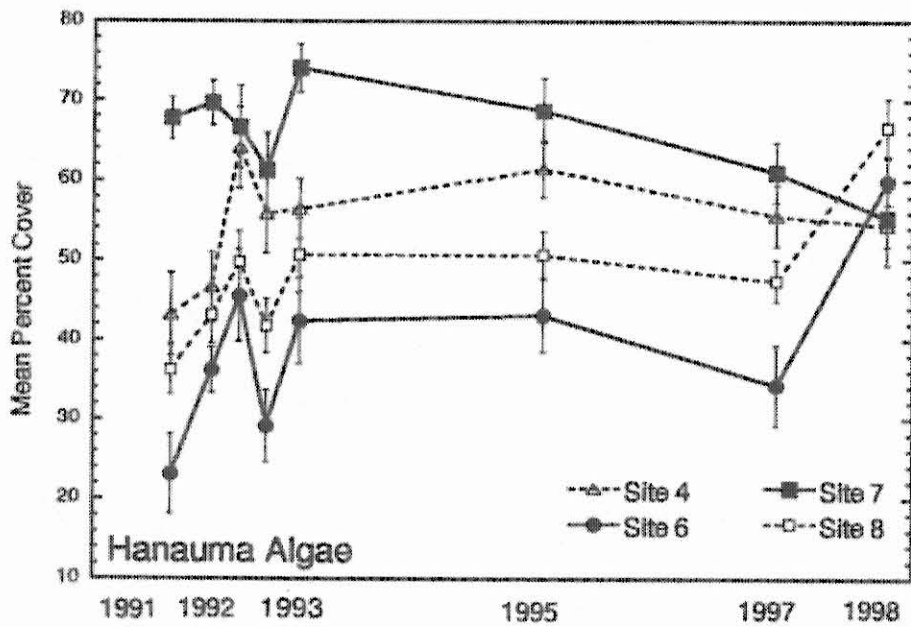
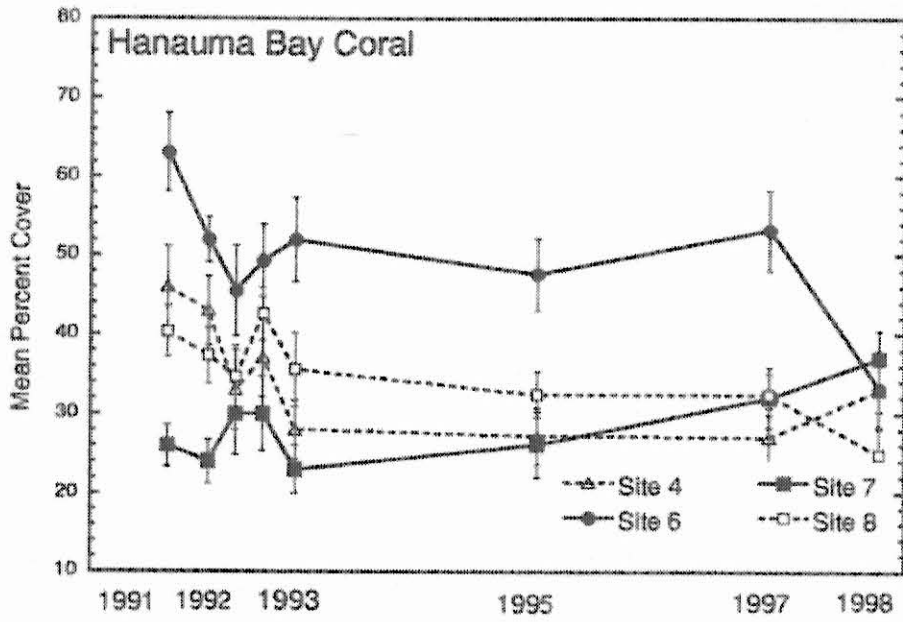


Figure 4. (Opposite page and above) Temporal changes in percent cover of coral, algae, and diseased *Porites* tissues averaged over all sites at Hanauma Bay, Honolulu, and Puako, 1991-1998.



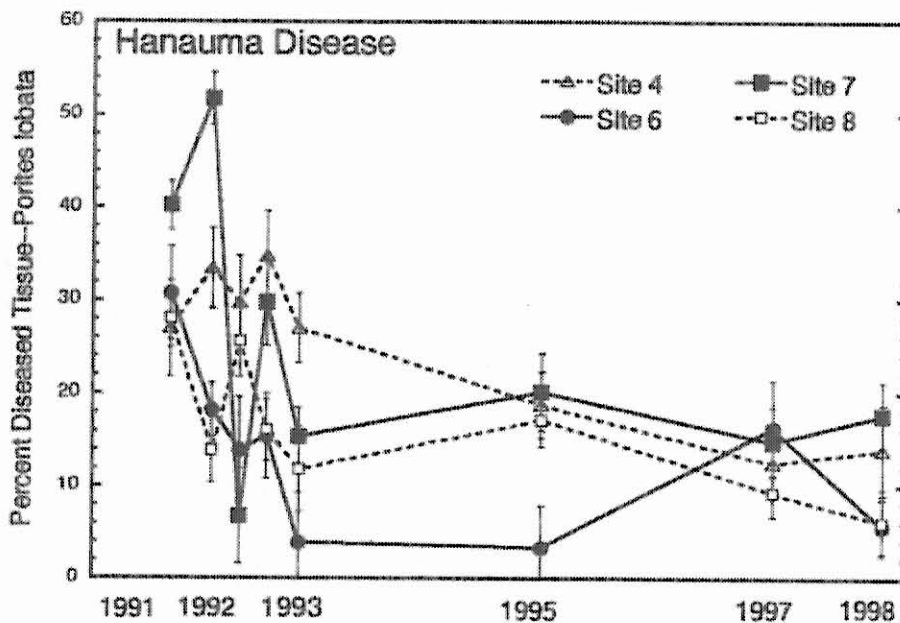
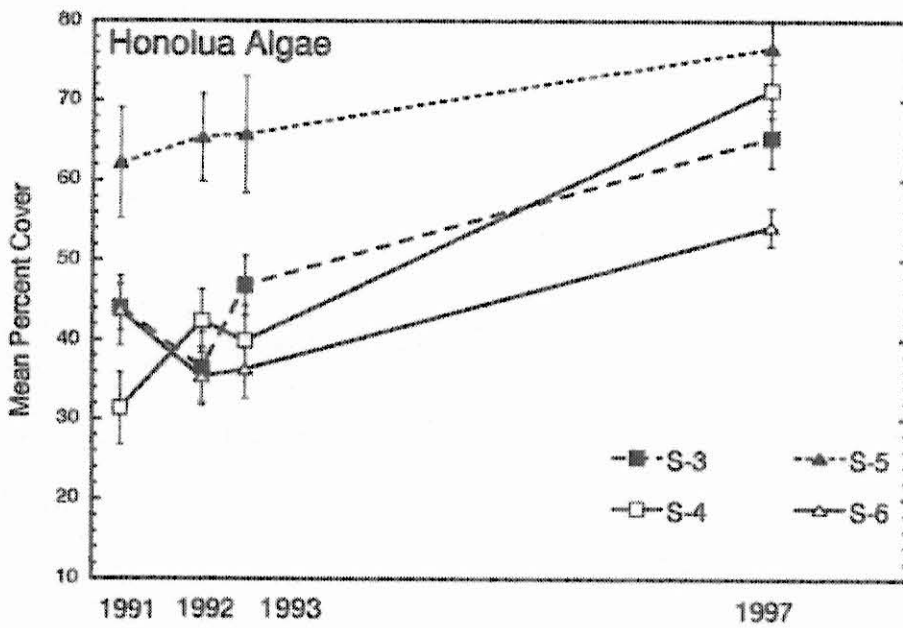
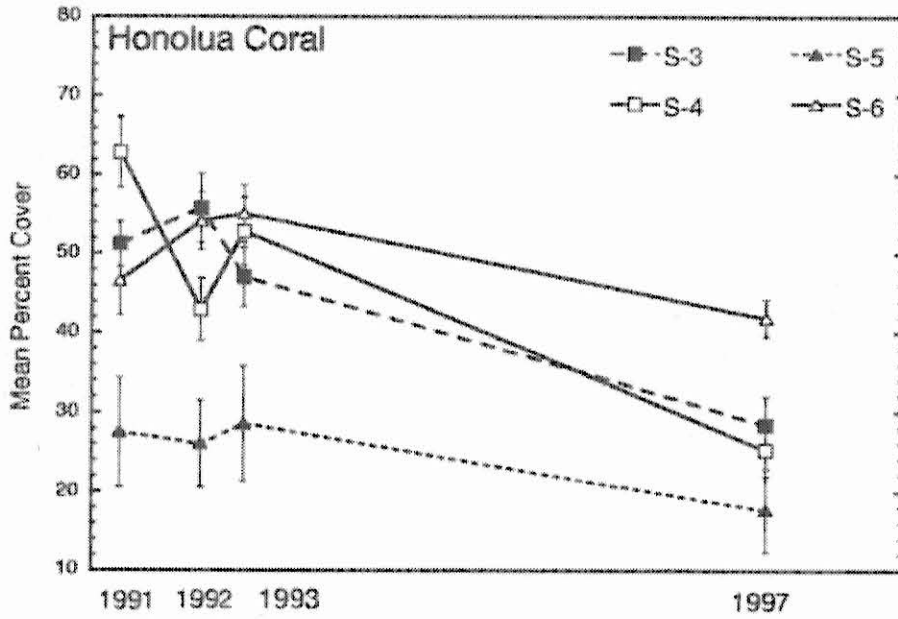


Figure 5. (Opposite page and above) Temporal changes in mean percent cover of coral, algae, and diseased *Porites* tissues for four sites at Hanauma Bay, Oahu, 1992-1998.



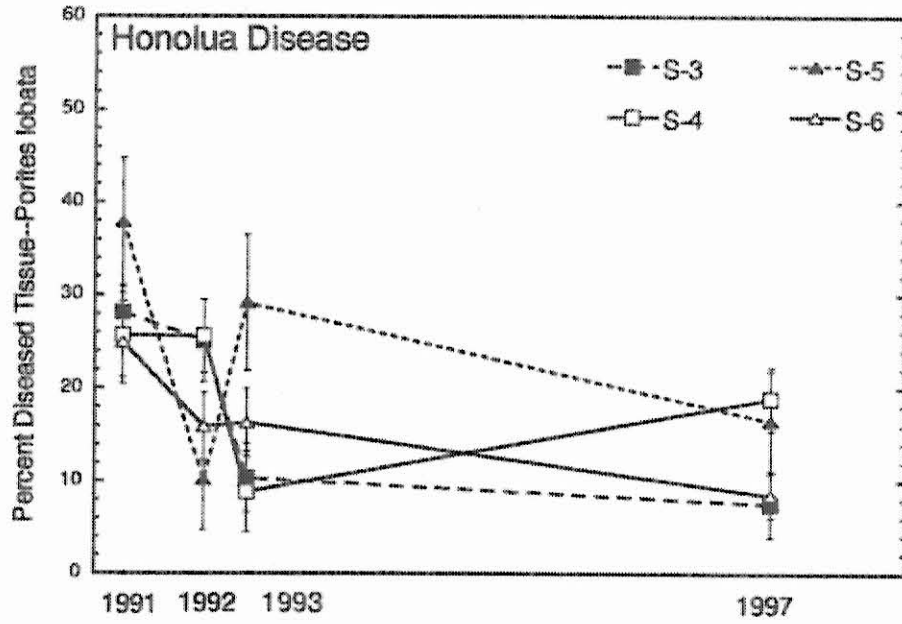
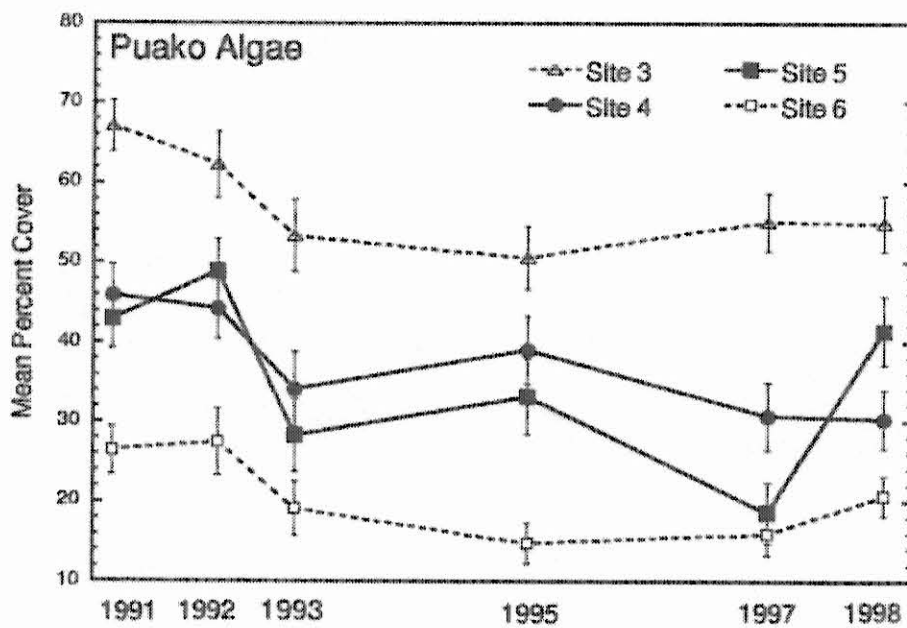
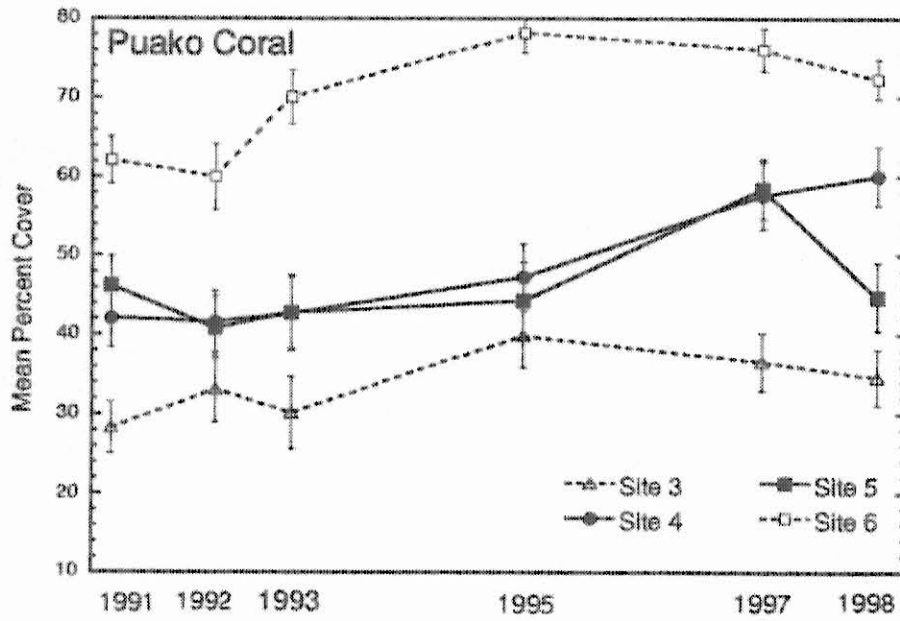


Figure 6. (Opposite page and above) Temporal changes in mean percent cover of coral, algae, and diseased *Porites* tissues for four sites at Honolua Bay, Maui, 1991-1997.



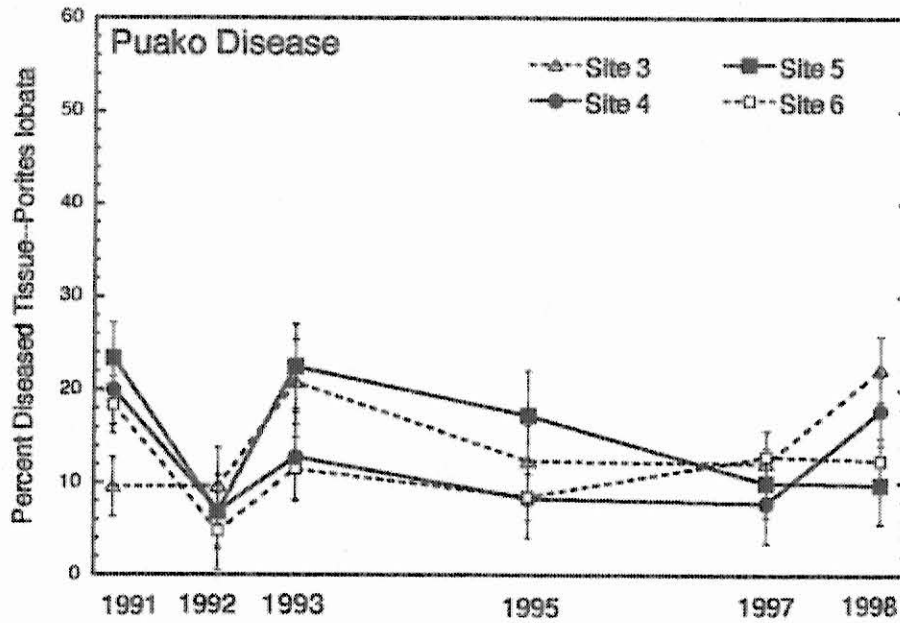


Figure 7. Temporal changes in mean percent cover of coral, algae, and diseased *Porites* tissues for four sites at Puako, Big Island, 1991-1998.

Trimmed pieces of decalcified tissues (oral, aboral, and longitudinal surfaces) were dehydrated and cleared, embedded in Paraplast, and sectioned at 6 mm. Slides from each sectioned block were stained with hematoxylin and eosin (to emphasize general tissue and cell structure), Masson's Trichrome (connective tissue), modified Movat's Pentachrome (mucus secretory cells), Gomori's Methenamine Silver (fungi), and Brown-Hopp's stain (gram +/- bacteria). Tissues and slides were sent to the Registry of Tumors in Lower Animals (RTLTA) at the Smithsonian Institution, Washington, DC where they were examined by an expert on disease in Caribbean and Atlantic coral species (EC Peters, TetraTech, Fairfax, VA). RTLTA accession numbers for these tissues are ERLN 51-54.

Results

Temporal Changes in Overall Coral Community Structure

Indices of spatial heterogeneity (Table 1) ranged from 1.13 (shallow reef flat, Honolua) to 1.37 (*Porites compressa*-dominated areas at Hanauma Bay). Overall mean percent coral cover as estimated from video analyses of 1997/1998 surveys were 32% for Hanauma Bay, 28% at Honolua Bay, and 53% at Puako (Fig. 4). Mean algal cover (macroalgae and turf) was 59% at Hanauma Bay, 67% at Honolua, and 34% at Puako. Incidence of diseased tissues in the most recent surveys (measured only for the massive *Porites*) was notably similar at all three sites in 1997/1998, ranging from 11% at Hanauma, 13% at Honolua, and 16% at Puako.

Coral abundance declined 10 to 12% overall from 1991 censuses at Hanauma and Honolua while increasing 8% at Puako (Fig. 4). Specific transect areas showed different responses within each site (Fig. 5; 6; 7). Most of the change in coral and algal cover at Hanauma Bay occurred between 1991 and 1993 censuses, and between 1993 and 1997 at Honolua and Puako. Significant declines in coral abundance occurred at Hanauma Bay for all areas combined between March 1992 and April 1993 (multivariate repeated measures ANOVA; $p=0.002$).

As estimated from video transects, the percent of total *Porites* tissues affected by disease varied considerably among colonies, areas, sites, and survey periods, but appeared to have been fairly constant at all three sites from 1993 to 1998 (Fig. 4; 5; 6; 7). Coral tissues affected by disease decreased by half (31.5% to 15%) over a one year period (1992-1993) at Hanauma Bay and by over a third (29% to 18%) at Honolua.

Spatial Distribution of Diseased Corals at Hanauma Bay

Mean size of massive *Porites* colonies varied from 0.004 m² (Puamana, 5 m depth) to 3.690 m² (Olowalu, 3 m depth) (Table 2). Colony size was significantly larger at shallower depths ($r^2=0.62$, $p<0.01$). However, distributions of diseased corals showed no apparent correlation with depth or distance from shore. The percent of necrotic tissue on colonies was significantly correlated with colony size, although the occurrence of tumors showed no such trend (Fig. 8). Turf algae abundance also increased as a function of colony size, although abundance of calcareous algae did not (Fig. 9).

Necroses and tumors were observed on all aspects of colonies (top, sides, and bottom) suggesting that exposure (e.g., to solar radiation (UV) plays little role in their development. The lack of clumping of diseased or tumorous colonies, along with the observation of distinct lines of demarcation between healthy and diseased tissues between adjacent colonies, suggests that these diseases are not infectious through proximity or direct colony contact.

Table 2. Mean proportion of *Porites* colony surfaces occupied by crustose coralline algae, turfs, alpheid shrimp burrows, fish bites, tumors and necroses for each colony intersecting a 25 m transect at each site (Kealakekua, Puako, Puamana, Olowalu, Molokini, Hanauma 1998) or the first 20 colonies intersecting a 50 m transect line (Hanauma Bay 1992, 1994).

Trans #	Depth (m)	No. Cols.	Mean Col Size (m ²)	Total Col Area (m ²)	Crust	Turf	Shrimp Burrow	Fish Bites	Tumor	Necroses	Tumors			
											% cols w/tumors	Mean No./col	Area (cm ²)	
Kealakekua Bay, May-98														
1	3	17	0.211	3.59	5.6	5.9	5.3	1.5	3.09	0.00	12	1.12	435	
2	3	20	0.886	17.72	8.3	14.8	7.0	4.0	0.05	0.00	5	0.20	13	
4	3	42	0.752	31.57	0.5	13.3	3.1	3.2	0.05	0.12	2	0.19	32	
Puako, May-97														
5	11	41	0.036	1.49	17.3	18.6	0.2	0.4	0.67	0.00	5	0.05	61	
6	12	23	0.101	2.31	7.7	16.1	0.2	0.8	1.10	0.05	26	0.83	107	
Puako, May-98														
5	11	38	0.022	0.83	0.0	2.2	2.2	0.3	0.53	0.79	3	0.03	25	
Puamana, May-97														
1a	5	12	0.004	0.05	nd	nd	nd	nd	0.00	0.00	0	0.00	0	
1b	5	26	0.122	3.18	nd	nd	nd	nd	2.12	0.10	27	1.10	787	
Olowalu, May-97														
1a	3	9	0.052	0.47	nd	nd	nd	nd	4.20	0.00	33	0.89	37	
1b	3	14	3.690	51.67	nd	nd	nd	nd	0.20	0.00	36	1.90	1116	
Molokini, Mar-98														
W-15	5	40	0.037	1.46	0.0	4.3	2.1	0.0	0.03	0.13	3	0.08	6	
W-40	13	44	0.010	0.43	0.0	1.3	0.0	0.0	0.00	0.23	2	0.02	1	
C-15	5	32	0.081	2.58	0.5	2.3	0.6	0.0	0.18	0.31	19	0.41	43	
C-40	13	11	0.068	0.75	0.0	0.5	0.0	0.0	7.27	0.00	36	0.73	27	
Molokini, Oct-98														
C-15	5	23	0.082	1.89	7.4	7.4	4.6	0.9	0.00	1.52	0	0.00	0	
C-40	13	28	0.066	1.85	0.5	1.3	2.1	1.4	0.43	0.71	14	1.04	281	
E-15	5	25	0.015	0.38	0.4	2.6	1.2	0.2	1.00	0.60	4	0.04	12	
E-40	13	9	0.006	0.05	0.0	2.8	0.0	3.3	0.00	0.56	0	0.00	0	
Hanauma Bay Apr-92														
4	3	20	2.368	47.35	0.0	30.0	6.0	11.0	3.00	4.00	35	nd	19581	
6	12	20	0.842	16.84	17.0	3.0	2.0	6.0	5.00	0.00	60	nd	6202	
7	8	20	0.810	16.20	3.0	38.0	3.0	5.0	6.00	3.00	45	nd	11640	
8	10	20	1.075	21.49	14.0	17.0	4.0	11.0	4.00	3.00	35	nd	7220	
RR	9	20	1.072	21.43	11.0	15.0	1.0	7.0	2.00	5.00	30	nd	5560	
Wall	4	20	2.388	47.76	0.0	28.0	4.0	9.0	4.00	5.00	45	nd	9180	
Hanauma Bay Feb-94														
4	3	20	1.801	36.01	2.0	29.0	7.0	5.0	8.00	3.00	25	nd	28260	
7	8	20	0.836	16.72	6.0	27.0	6.0	2.0	3.00	2.00	40	nd	4420	
8	10	20	0.211	4.21	22.0	4.0	4.0	2.0	0.20	0.00	5	nd	30	
RR	9	20	0.554	11.08	3.0	16.0	5.0	2.0	3.00	1.00	45	nd	4420	
Hanauma Bay May-98														
Wall	4	25	0.242	6.05	2.0	30.0	3.2	7.0	1.17	0.61	32	1.24	424	
RR	9	37	0.268	9.91	10.0	14.7	3.0	5.0	2.06	1.20	38	1.57	483	
Mean over all sites			--	0.624	12.6	5.3	13.3	3.0	3.4	2.08	1.10	22.1	0.57	3347

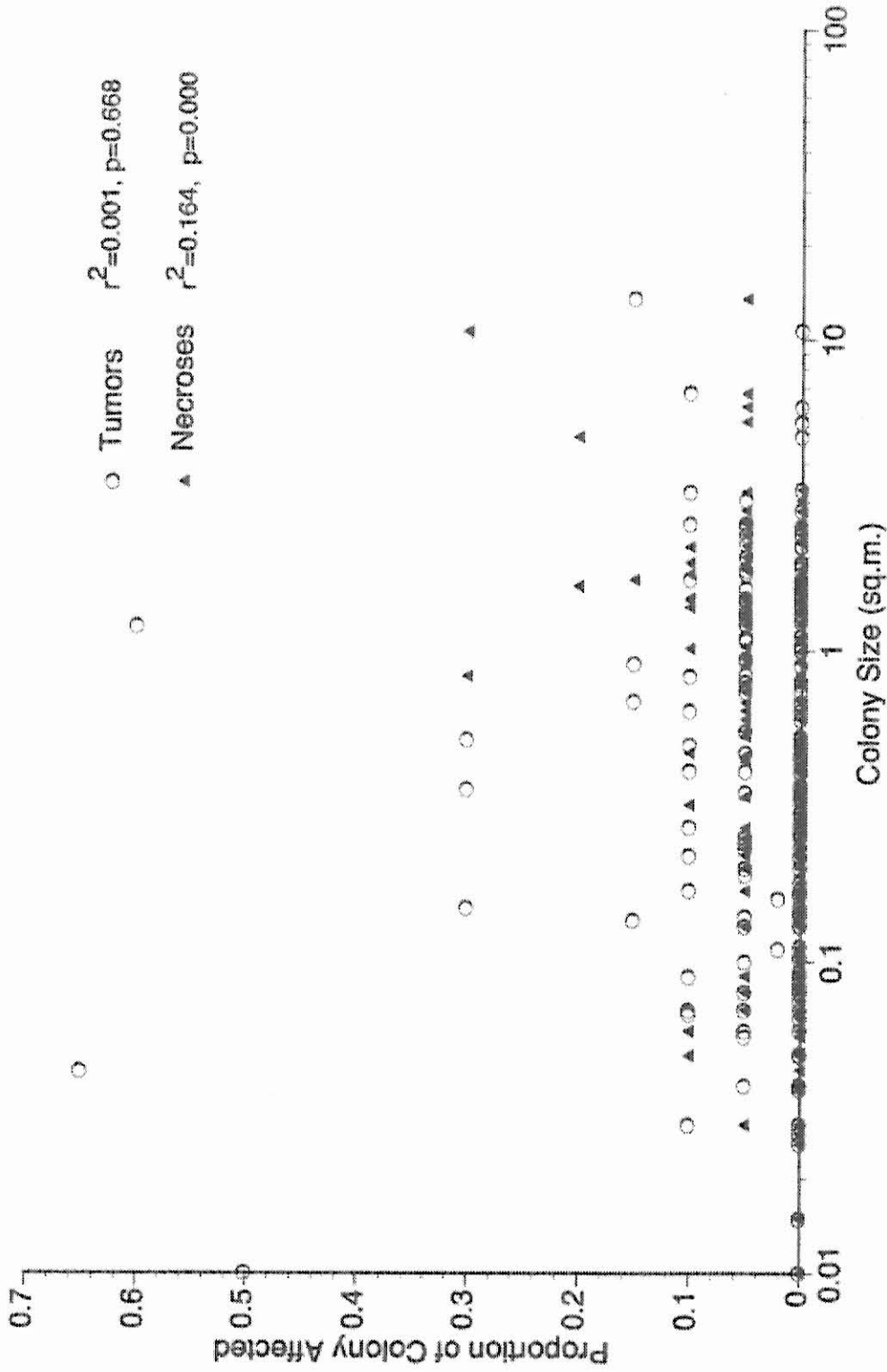


Figure 8. Relationship between colony size and proportion of colonies affected by tumors (open circles) and necroses (triangles) at Hanauma Bay, Oahu.

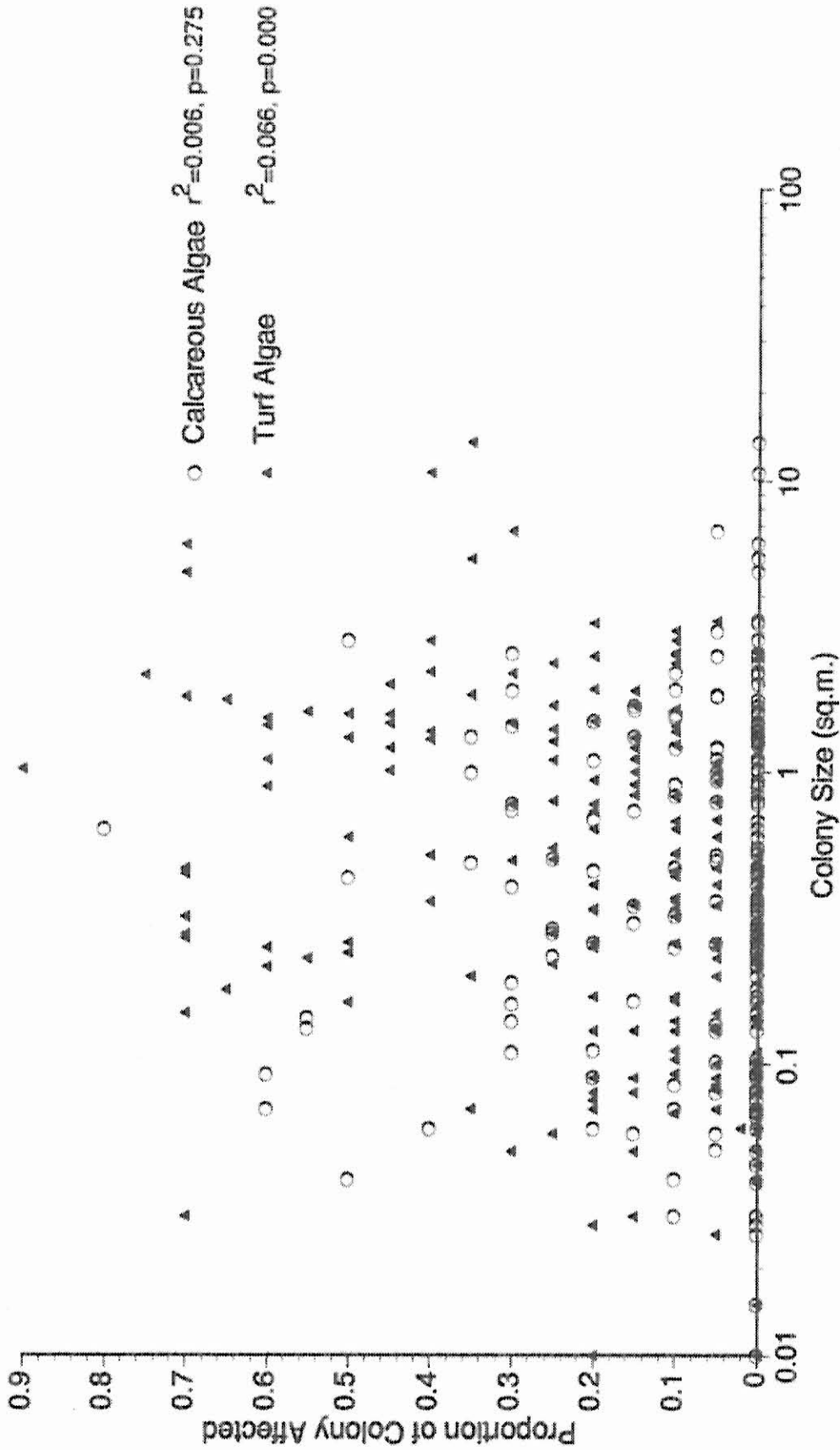


Figure 9. Relationship between colony size and proportion of colonies covered by calcareous algae (open circles) and turf algae (triangles) at Hanauma Bay, Oahu.

Table 3. Change in coral tissue health at macro-sites during initial 2 year study period. + coral tissue increased; - coral tissue decreased; = no measurable change.

Hanauma Bay		Status:		
Macro #	Oct-91	Oct-92	Oct-93	
7	tumor	=	=	
8	necrosis	=	+	
9	necrosis	-	=	
10	necrosis	=	+	
11	necrosis	-	=	
12	tumor	=	=	
13	necrosis	-	-	
14	necrosis	-	=	
15	tumor	-	-	
16	necrosis	-	-	
17	necrosis	-	-	
18	necrosis	+	+	
19	necrosis	+	+	
20	necrosis	-	-	
21	necrosis	+	+	
22	necrosis	-	-	
23	necrosis	+	-	
24	necrosis	-	-	
25	necrosis/tumor	-	-	
26	tumor	-	=	
27	necrosis	-	-	
28	necrosis	-	+	
29	necrosis	+	-	
30	necrosis	-	-	
31	necrosis	-	-	
32	necrosis	-	-	
33	tumor	+	+	
34	tumor	+	=	
35	tumor	-	-	
36	tumor	-	-	
Honolua Bay				
Macro #	Oct-91	Jun-92	Oct-92	
3	sediment covered	-	=	
4	necrosis	-	=	
13	necrosis	-	-	
14	necrosis	-	=	
15	necrosis	-	+	
16	sediment covered	-	-	
17	bleaching	+	-	
Puako				
Macro #	Oct-91	Jan-92	Aug-92	
1	necrosis	-	=	
2	necrosis	+	+	
3	necrosis	=	-	
4	necrosis	-	=	
5	necrosis	-	=	
6	necrosis	-	+	
9	necrosis	-	-	
10	necrosis	=	-	
11	necrosis	-	-	
12	necrosis	-	=	
13	necrosis	=	-	
14	necrosis	=	-	
15	necrosis/tumor*	-	-	
16	necrosis	+	+	
17	necrosis	=	-	
18	necrosis	=	-	

*tumor decreased in size

Temporal Progression of Diseased Tissue

Of the 72 macro-photo areas of diseased tissues initially established, 53 (74%) were followed over the initial three year period of this study (Table 3). Tags were overgrown by algae or otherwise lost for 6 colonies at Hanauma Bay, 11 colonies at Honolua, and 2 colonies at Puako.

Generally, diseased colonies exhibited a progressive loss of tissue within and beyond the framed areas of observation. Necrotic areas were usually rapidly invaded (within days to weeks) by filamentous algae (*Ceramium* sp. and other unidentified spp). A blue-green bacterial mat was often observed to secondarily overgrow the affected areas, persisting from weeks to months before being replaced by filamentous turf. Areas covered by turf algae trapped fine sediments, particularly on the upper (horizontal) surfaces of colonies, perhaps accelerating further tissue loss.

At Puako, 88% (14/16) of diseased areas died (100% tissue loss within the study frames) within the first 18 months of the study period. Similarly, 86% (6/7) of diseased areas died at the Honolua site. During the initial 30 month observation period at Hanauma Bay, 70% (21/30) of diseased areas died. Recovering lesions (14 at Honolua, 12% at Puako, and 23% at Hanauma) replaced <5% of the lost coral tissues.

A "succession" of lesion recovery was observed to entail an initial invasion of fine filamentous or turf algae. This turf was often grazed by fish as exhibited by numerous feeding scars exposing bare coral skeleton. Grazed areas were then apparently invaded by calcareous algal crusts that spread to cover the area "opened" by coral tissue necrosis and death. Over the time of this study, no areas have yet shown signs of coral larval recruitment. Rather, any increases in coral tissue cover of initially diseased areas were the result of apparent remission of necroses followed by regrowth from adjacent healthy tissues.

Coral tumors exhibited a variety of temporal patterns over the period of the study. Of the ten tumors observed (9 at Hanauma, 1 at Puako), 7 increased

in size and exhibited partial (2) or total (5) mortality (Table 3). These tumors became necrotic and tissues were replaced by turf algae. One tumor showed no apparent change in size, shape or coloration over 2.5 years. Two tumors (one at Hanauma, one at Puako) gradually shrank in size and distinctness, and eventually regained normal appearance. Examination of fresh tissue samples from other tumors in July 1993 (not those under long-term observation), showed a lack of any evidence of gonadal development although surrounding "healthy" tissues were ripe with eggs or sperm. However, subsequent samples of tumors from *P. lobata* and *P. compressa* collected in July 1997, showed gonadal development, although number of oocytes per gonad and number of gonads per polyp were reduced up to 60% (Hunter and Field 1998).

Diagnosis and Pathology of Disease

The most common type of disease in Hawaiian *Porites lobata* [ERLN 51] was characterized by numerous gram negative bacterial aggregates in the gastrodermal cells of tentacles (sometimes deeper in the polyp) and was diagnosed as "stress-related necrosis" (Peters 1984). Necrotic tissues are evidenced by lysed nuclei and cell death. Similar bacterial aggregates have been identified in acroporid corals with "white band disease" in the Caribbean, but their association and/or pathogenicity have not been definitively confirmed.

Two other samples of necrotic tissues [ERLN 53 & 54] showed few or no bacterial aggregates, but secondary invasions of filamentous algae. One of the algal species was identified by K. McDermid (University of Hawai'i-Hilo) as *Ceramium* sp., a filamentous red alga with tiny rootlets that embed in coral epidermis. Such algal invasions may be caused by damselfish bites on the coral tissues resulting in cell lysis or by elevated nutrient loads in the water column increasing the potential for algal growth.

The fourth sample [ERLN 52] was a coral tumor characterized by larger polyps and lighter pigmentation than the surrounding normal tissues. Cell architecture of the tumor appeared normal, but there were numerous bacterial aggregates in the tentacles and coenosarc. In addition, *Nematopsis*-like cysts (gregarine sporozoans) were identified in this sample, the first record of the latter in coral tissues in the Pacific. There was no evidence of infection by fungi or the blue green bacterium *Phormidium corallyticum* in any of the samples. *P. corallyticum* has been shown to be associated with Black Band Disease in reef corals and gorgonians in the Caribbean.

Discussion

This study represents the first record and pathological diagnosis of diseased corals on Hawaiian reefs. Diseased and/or tumorous tissues in corals appeared to be chronic and relatively frequent on *Porites lobata* at all three sites in the present study, although they are particularly apparent at Hanauma Bay, both in number and proportion of coral tissue affected.

Although the potential causes of coral necroses are still conjectural, bacterial aggregates found in tissues of *Porites lobata* from Hanauma Bay were similar to those found in *Porites astreoides* in the Caribbean (Peters et al. 1983). Peters and Santavy (1996) suggest that these bacteria may be mutualistic in healthy corals but become opportunistic pathogens when corals have been environmentally stressed. Potential stresses may include elevated nutrient levels that encourage algal overgrowth; mechanical abrasion from shifting sand or fish predation; smothering of tissues from episodically high levels of sedimentation, and/or other factors that are currently unidentified. Rates of colonization and persistence of invading algae in the newly "opened" (dead) areas perhaps govern consequences of disease.

The cause and cellular basis of tumors in *Porites* corals is also unknown. Some exhibit a form of "gigantism" (Peters et al. 1986), similar to those reported in *Platygyra* in Australia by Loya et al. (1984), while others show uncontrolled or "hyperplastic" growth. A unique feature of the *Porites* tumors noted in the present study is the apparent ability of some to recover a normal appearance. Whether cells in these tumors are eventually replaced by normal cells or actually mutate back to a "normal" condition is an area of great interest. Of additional interest is the long-term impact of tissue loss on the coral community from tumors that become necrotic.

An outbreak of black band disease at Looe Key, Florida, in 1986 was found to be associated with high temperatures, eutrophication, and sedimentation (Peters and Santavy 1996). However, outbreaks of other diseases do not appear to be consistently associated with known anthropogenic stressors (e.g., pollutants, proximity to urban centers). Background rates of tissue loss as a result of disease (i.e., non-outbreak occurrence levels) and the relationship to subsequent algal overgrowth are currently unknown for any reef ecosystem. Nutrient and toxic contaminants input levels are unknown for most Hawaiian reef sites. Estimates of shoreline water column concentrations of nitrates, ammonium, phosphates, chl *a*, and enterococcus are monitored biweekly at Hanauma Bay by the Hawai'i State Department of Health, but may not reflect absolute rates of input nor amounts of uptake of nutrients or toxic contaminants by the ecosystems.

Previous studies provided a basis for comparison of present coral and algal abundance with past conditions as Hanauma Bay (Anderson 1978), Honolua Bay (Torricer et al. 1979), and Puako (Hayes et al. 1982). The maps provided from the previous Puako survey (and the fairly homogeneous nature of the reef areas at this site) allowed reasonably accurate comparisons. Exact locations of study areas within each site were not available from the previous studies at Hanauma Bay and Honolua Bay. Coral abundance at three of the four Hanauma Bay reef areas was remarkably similar over a 16-year period (Table 4). The fourth area (Area 6 in the present study) showed a substantial decline in coral abundance between 1976 and 1992. Of note is the apparent increase in algal abundance in 1992 when compared to 1976 levels at all areas surveyed. Whether this increase in algal cover is real or the result of differences in scoring by individual investigators cannot be resolved. Such questions emphasize the need for permanent visual records of reef sites.

The importance of long-term, quantitative studies of temporal and spatial variability of coral reef ecosystems has become increasingly apparent over the past few years (Knowlton 1992; Hughes 1993). Symposia and workshops at national and international meetings have reiterated the present lack of information necessary to make scientifically sound decisions as to whether potential environmental changes (e.g., increased pollution, seawater temperatures, sea level, UV radiation) are causing or will cause significant changes in coral reef vitality. It is hoped that the data presented here will provide the basis and framework by which long-term changes on Hawaiian reefs might be evaluated and managed.

At least three general types of coral disease commonly occur on Hawaiian reefs (necroses, band diseases, tumors; Hunter pers. obs.). Diseased coral tissue is rapidly invaded by unidentified fine filamentous (turf) algae and cyanobacteria. There appear to be three resultant outcomes of this invasion: 1) a successional change from turf to crustose coralline algae on which new coral recruits may become established, 2) recovery and overgrowth by healthy, adjacent coral tissues, or 3) persistence of the turf community and a change from coral to algal domination of the affected coral colony.

The change from an ecosystem dominated by reef-building corals to one dominated by non-reef-building algae is usually gradual and difficult to detect with statistical rigor using traditional repeated measures assessments of macro-scale surveys of reefs. In future studies, diseased tissues will be photographed

Table 4. Comparison of percent coral and algal abundance data from present study to data from previous studies. * refers to site designations of previous studies

<u>Hanauma Bay</u>		
Mean Coral Abundance	Anderson, 1978*	present study
	Sep-76	Oct-92
Area:		
10', p.293-94*, 4	28.10	33.90
25', p.254-55*, 7	36.30	30.22
30', p.301-02*, 8	37.65	34.65
35', p.277-78*, 6	64.76	45.51
<u>Mean Algal Abundance</u>		
Area:		
10', p.293-94*, 4	29.00	64.07
25', p.254-55*, 7	31.50	66.67
30', p.301-02*, 8	29.00	49.78
35', p.277-78*, 6	17.00	45.58
<u>Puako</u>		
Mean Coral Abundance	Hayes, et al., 1982*	present study
	Spring. 1990	Apr-93
Area:		
3A*, 3	16.00	30.13
4A*, 4	65.00	42.72
5A*, 5/6	63.00	42.78/70.11
<u>Mean Algal Abundance</u>		
Area:		
3A*, 3	73.00	53.34
4A*, 4	29.00	34.05
5A*, 5/6	31.00	28.22/19.11
<u>Honolua Bay</u>		
Mean Coral Abundance	Torriger, et al., 1979*	present study
	Jun-76	Jun-92
Area:		
9* (6m), 4 (6m)	30.80	42.93
13* (2-8m), 6 (3m)	36.40	54.16
15* (1-6m), 3 (3m)	15.90	55.78
<u>Mean Algal Abundance</u>		
Area:		
9* (6m), 4 (6m)	5.50	42.40
13* (2-8m), 6 (3m)	43.40	35.40
15* (1-6m), 3 (3m)	16.00	36.56

using a higher magnification close-up lens. Time-series photographs will be digitized and the process of change recorded at each survey interval. Algal taxa involved in each successional stage will be characterized within and among study sites, and principal component analyses will be used to assess any taxonomic and/or geographic trends in the outcomes of disease on Hawaiian coral reefs.

Acknowledgments

Numerous people have assisted in various aspects of the development and implementation of the research objectives of this project since its inception: G. Aeby, R. Ames, M. Awai, K.O. Beckman, E. Brown, B. Carlson, B. Carman, E. Chornesky, S. Coles, M. Cottone, N. Craig, J. Culp, F. Coopersmith, G. DeCouet, C. Evans, S. Field, A. Friedlander, M. Gassel, D. Gochfeld, R. Grigg, D. Gulko, B. Hannum, J. Hardy, S. Hau, P. Hendrix, J. Holland, A. Hong, L. King, R. Kosaki, B. Larson, M. Lesser, K. Lowe, J. Maragos, K. McDermid, O. McMillan, K. Meier, R. Nishimoto, D. Pence, E. Peters, D. Potts, B. Rinkevich, S. Romano, S. Russell, D. Schricte, S. Scott, C.M. Smith, J.E. Smith, N. Stambler, D. Stellar, K. and U. Stender, M. Stephenson, R. Trench, L. Udall, R. Vago, P. Vroom, S. Wells. Particular thanks are due to S. Folsom for the majority of the video analyses; A. Hong for providing research access to Hanauma Bay; E. Brown for diving and logistical support on Maui; B. Carman and the DAR crew for diving and logistical support on the Big Island; D. Schricte for identifying the initial question and providing great photographs; M. Stephenson for enormous assistance in every aspect of the study; and Hawai'i DAR and UH Sea Grant for funding support. While little would have been accomplished without the contributions of those listed above, all errors and omissions reside with the author.

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