Coral Disease in Japan

4

Naohisa Wada, Aki Ohdera, and Nobuhiro Mano

Abstract

While 40 different coral diseases have been described globally since the first report in 1973, the causative agents for most cases have yet to be identified. In Japan, a total of ten coral diseases, including black band disease, brown band disease, white syndrome, pigmentation response, and growth anomalies have been confirmed in the field by epidemiological surveys and monitoring projects. However, disease-induced coral mortality has yet to be reported. A national government-based monitoring project for coral reef conservation has recorded the three major diseases (black band disease, white syndrome, and growth anomalies) in ten areas (Kushimoto, Shikoku, Amakusa, Yakushima, Chichijima, Setouchi, Kerama, Miyako, Ishigaki, and Sekisei Lagoon/Iriomote) of Japan since 2003. Furthermore, a substantial number of studies have contributed to the understanding of coral diseases, identifying ten additional diseases in six coral genera, including a new disease discovered in the temperate coral *Turbinaria peltata* in 2009. In order to elucidate the causative mechanism of coral disease, researchers have utilized a variety of approaches including lipid measurements, molecular technique, and new histopathology methods. However, despite the efforts, further research is required to fully understand the mechanism and impacts of coral disease. This chapter summarizes current knowledge on coral pathogens and discusses the future of coral disease research in Japan.

Keywords

Coral disease • Black band disease • Growth anomalies • White syndrome • White spot syndrome

4.1 Introduction

In the past several decades, the world's coral reefs have shown a decreasing trend in total coral cover, (Hughes et al. 2003; Bellwood et al. 2004; Bruno and Selig 2007), with 32.8% of the 845 species found in reefs currently considered to be in danger of extinction (Carpenter et al. 2008). The drivers of coral reef decline has been attributed to coral bleaching caused by climate change, pollution linked to tourism and industrial development, sediment depositions from estuarine outflow following heavy rainfall, overfishing, and predation by the crown-of-thorns starfish, as well as coral disease (Dustan 1999; Porter et al. 2001; Harvell et al. 2007; Wilkinson 2008; Maynard et al. 2015).

N. Wada (✉)
Biodiversity Research Center, Academia Sinica, No. 128, Sec. 2, Academia Rd, Nangang, Taipei, 11529, Taiwan

Department of Marine Science and Resources, College of Bioresource Science, Nihon University, Fujisawa, Kanagawa, Japan
e-mail: naohisa0308.nw@gmail.com

A. Ohdera
Department of Biology, Eberly College Science, Pennsylvania State University, University Park, PA, USA

N. Mano
Department of Marine Science and Resources, College of Bioresource Science, Nihon University, Fujisawa, Kanagawa, Japan

A. Iguchi, C. Hongo (eds.), *Coral Reef Studies of Japan*, Coral Reefs of the World 13,
https://doi.org/10.1007/978-981-10-6473-9_4

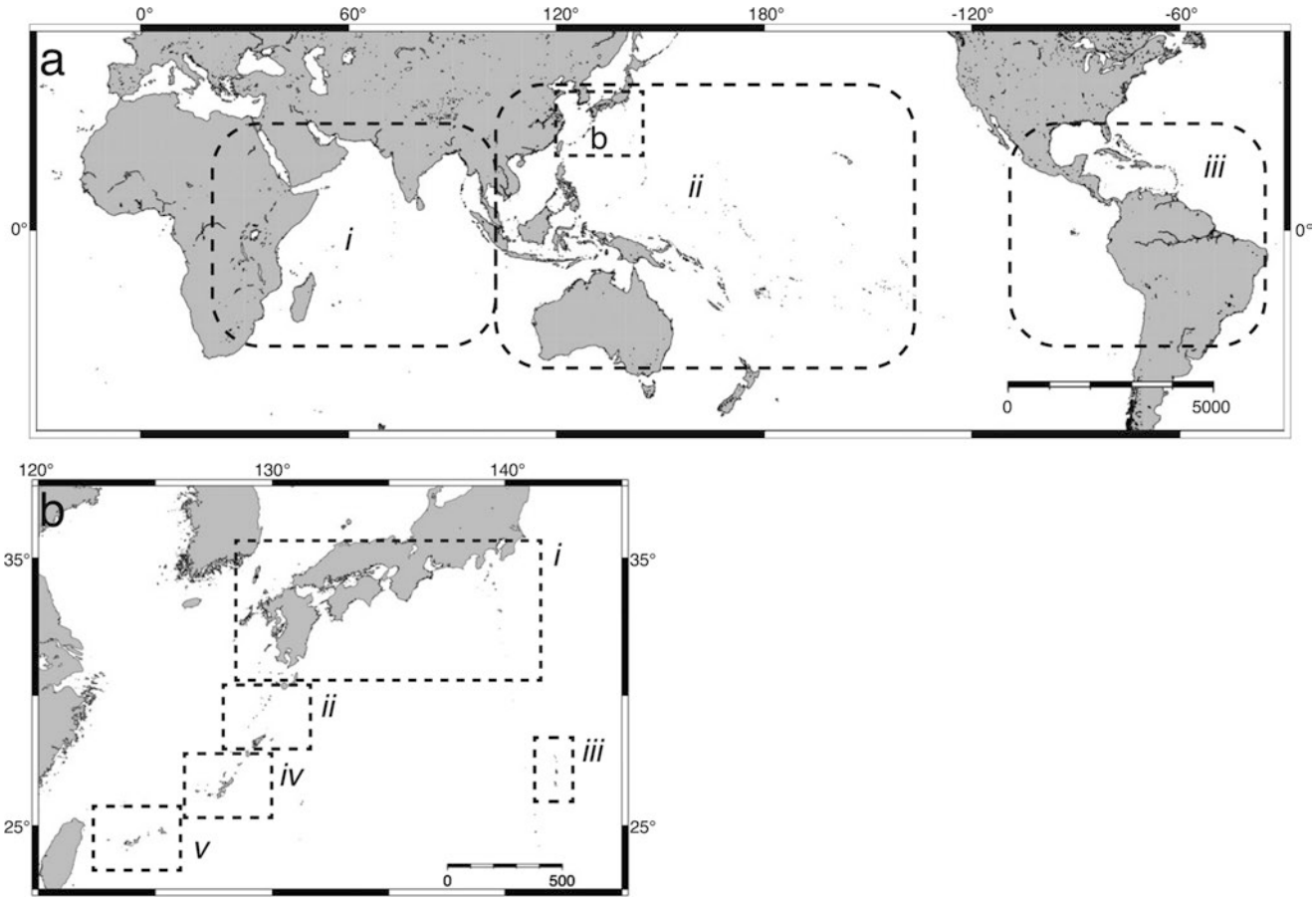

Fig. 4.1 World coral reef distributions are separated into three regions (**a-***i*) Indian Ocean, (**a-***ii*) Caribbean Ocean, (**a-***iii*) Pacific Ocean. Japanese coral reefs are found in five regions within the seas surrounding (**b-***i*) Honshu, Shikoku, and Kyushu, (**b-***ii*) Yakushima to Amami, (**b-***iii*) Ogasawara Islands, (**b-***iv*) Okinawa Main, (**b-***v*) Yaeyama Islands

Since the identification of black band disease (BBD) in Belize in 1973 (Antonius 1973), over 40 new coral diseases and syndromes have been reported from the Caribbean, Indo-Pacific, and Red Sea (Fig. 4.1, Table 4.1; Bruckner 2015), including reports of severe damages to reefs caused by coral disease. Aronson and Precht (2001) suggested white band disease to be responsible for the 95% decline in the Caribbean coral *Acropora palmata* and *A. cervicornis* populations from 1980 to 1990. Due to difficulties of predicting the timing of occurrence and severity of outbreaks, understanding the factors that can lead to and exacerbate the severity of the outbreaks is imperative.

Woodley et al. (2008) has classified diseases of both stony and gorgonian corals into the following six categories:

1. Bleaching through loss or degradation of the dinoflagellate symbiont caused by biotic (bacteria) and abiotic (temperature, UV radiation, salinity, toxicants, etc.) factors
2. Physiological and morphological abnormalities caused by noninfectious environmental stressors such as toxicants, sedimentation, and pollution
3. Physical damage or trauma caused by predators
4. Parasitic infestation caused by protozoans, metazoans, or sponges
5. Growth abnormalities caused by hypertrophy, hyperplasia, neoplasia, or tumors
6. Partial or whole colony infections by bacteria, fungi, virus, or other microorganisms potentially leading to colony mortality

To date, there have been only several cases in which the causative agent has been experimentally linked to an identifiable disease sign, thereby satisfying Koch's postulate.

White pox (WP) found in *A. palmata* has been linked to a rod-shaped gram-negative bacterium, *Serratia marcescens*, which causes distinct white patches throughout an afflicted colony (Patterson et al. 2002). The rod-shaped

Table 4.1 Common coral diseases identified worldwide

Reported year		Disease (acronym)	Coral affected	Location	Location in Fig. 4.1	Source
1970s	1973	Black band disease (BBD)	Massie corals	Belize	A-iii	Antonius (1973)
	1977	White plague type I (WPL I)	*Agaricia agaricites*	Florida	A-iii	Dustan (1977)
		Shut down reaction (SDR)	*Acropora cervicornis*, *Montastrea annularis*, and *Siderastrea siderea*	Belize and Florida	A-iii	Antonius (1977)
1980s	1982	White band disease type I (WBD I)	*Acr. palmata*	Caribbean Sea	A-iii	Gladfelter (1982)
	1983	Growth anomalies (GAs)	*Acr. palmata*	Venezuela	A-iii	Bak (1983)
1990s	1994	Dark spot disease (DSD)	*Orbicella annularis*	Colombian Caribbean	A-iii	Garzón-Ferreira et al. (2001)
		Caribbean yellow band disease (CYBD)	*Orb. annularis* and *Orb. Faveolata*	Florida	A-iii	Reeves (1994)
	1996	White pox (WP)	*Acr. palmata* and *Acr. cervicornis*	Florida Keys	A-iii	Holden (1996)
	1997	Vibrio shiloi-induced bleaching (VSB)	*Oculina patagonica*	Mediterranean coast, Israel	A-i	Kushmaro et al. (1997)
		Yellow blotch/band (YBL)	*Orb. faveolata*	Florida Keys	A-iii	Santavy and Peters (1997)
		White plague type II (WPL II)	*Millepora alcicornis* and *Dichocoenia stokesi*	Florida Keys	A-iii	Richardson et al. (1998)
	1998	White band disease type II (WBD II)	*Acr. cervicornis*	San Salvador and Bahamas	A-iii	Ritchie and Smith (1998)
		Arabian yellow band disease (AYBD)	*Acr. clathrata*, *Acr. pharaonis*, *Acr. tenuis*, *Acr. valida*, *Acr. flouria*, *Porites lutea*, *P. lichen*, *P. nodfera*, *Turbinaria reniformis*, and *Cyphastrea*	Gulf	A-i	Korrubel and Riegl (1998)
	1999	Skeletal eroding band (SEB)	Massive and branching corals	Indo-Pacific	A-i and ii	Antonius (1999)
2000s	2001	White plague type III (WPL III)	*Orb. annularis* and *Colpophyllia natans*	Florida Keys	A-iii	Richardson et al. (2001)
	2002	Pink-line syndrome (PLS)	*P. lutea*	Lakshadweep island	A-i	Ravindran and Raghukumar (2002)
	2003	*Vibrio coralliilyticus*-induced bleaching (VCB)	*Pocillopora damicornis*	Red Sea	A-i	Ben-Haim and Zicherman-Keren (2003)
		Porites ulcerative white spot disease (PUWS)	*Porites* spp.	Philippines	A-ii	Raymundo et al. (2003)
	2004	Brown band disease (BrBD)	Acroporidae	Great Barrier Reef	A-ii	Willis et al. (2004)
		White syndrome (WS)	Acroporidae and Poritidae	Great Barrier Reef	A-ii	Willis et al. (2004)
	2006	Red band disease (RBD)	*Pachyseris speciosa* and *Porites* sp.	Palau	A-ii	Sussman et al. (2006)

gram-negative bacterium *Aurantimonas coralicida* is responsible for white plague type II (WPL II), which causes whitening and tissue mortality at the base of *Dichocoenia* and *Orbicella* colonies (Denner et al. 2003). White plague type I (WPL I) in *Favia* spp. is caused by the rod-shaped gram-negative bacterium, *Thalassomonas loyana*, which exhibits

Table 4.2 Coral diseases identified from Japanese waters

Reported year		Disease (acronym)	Coral affected	Location	Location in Fig. 4.1	Source
2000s	2000	GAs	*Montipora informis*	Sesoko, Okinawa	B-iv	Yamashiro et al. (2000)
	2004	WS	Table *Acropora* spp.	Sekisei Lagoon, Okinawa	B-v	Biodiversity Center of Japan (2004)
		BBD	n.d.	Sekisei Lagoon, Okinawa	B-v	Biodiversity Center of Japan (2004)
	2009	Tissue loss	*Porites cylindrica*	Sekisei Lagoon, Okinawa	B-v	Asoh (2009)
		White spot syndromee (WPS)	*Turbinaria peltata*	Miyazaki	B-i	Yamashiro and Fukuda (2009)
2010s	2012	Pigmenation response	*P. lutea* and *P. lobata*	Zamami and Ginowan, Okinawa	B-iv	Weil et al. (2012)
		PUWS	*P. lutea* and *P. lobata*	Zamami and Ginowan, Okinawa	B-iv	Weil et al. (2012)
		BrBD	*A. nobilis*	Zamami and Ginowan, Okinawa	B-iv	Weil et al. (2012)
		Compromised health conditions (CH)	*Oxypora lacera*	Zamami and Ginowan, Okinawa	B-iv	Weil et al. (2012)
	2017	Skeletal eroding band (SEB)	*Montipora* spp.	Aka, Okinawa	B-iv	Wada et al. (in press)

n.d. no data

similar signs to WPL II (Thompson et al. 2006). White syndrome (WS), which displays rapid loss of tissue leading to exposure of the skeleton beneath, is caused by an infection by a gram-negative *Vibrio* species, affecting *Acropora*, *Montipora*, and *Pachyseris* species (Sussman et al. 2008). Other *Vibrios* have also been identified to cause signs similar to coral bleaching. *V. shiloi*-induced bleaching (VSB; Kushmaro et al. 2001) was identified in *Oculina patagonica*, while *V. coralliilyticus*-induced bleaching (VCB; Ben-Haim and Rosenberg 2002) has been found in *Pocillopora damicornis*. Finally, an infection by a cyanobacterium *Phormidium valderianum*, leads to a pink discoloration of the tissue in *Porites*, known as pink-line syndrome (PLS; Ravindran and Raghukumar 2002).

Since the first identification of growth anomalies in 2000 (GAs; Yamashiro et al. 2000), ten diseases have been identified in Japan (Table 4.2). This chapter reviews the coral diseases identified through studies conducted by the Ministry of Environment, Government of Japan, and Okinawa Prefecture. We further focus on current researches concerning confirmed growth anomalies and infectious diseases, events and their causes, and the future of disease research on Japanese coral reefs.

4.2 Monitoring Projects for Coral Reef Conservation

Conservation of coral reefs and major habitats in Japanese waters has been mainly led by large-scale national government survey projects, while small-scale projects covering smaller reef areas have been led by local to regional monitoring efforts. For most of these projects, in addition to coral cover and species composition, bleaching severity assessment, crown-of-thorns starfish and mollusk predation, as well as disease prevalence have also been monitored. In order to provide an overview of the disease outbreaks observed in Japanese coral reefs, this section summarizes the abovementioned surveys and their contributions to the coral conservation efforts.

4.2.1 National Government-Based Surveys

Coral reefs are increasingly threatened by global climate change as well the environment destruction caused by anthropogenic impacts. The goal of the national government

surveys is to determine the biological and ecological state of Japanese coral reefs and associated coral communities in order to better develop conservation policies. Since 2003, the Ministry of Environment has been conducting surveys in 24 regions where coral reefs exist, as part of the Monitoring Site 1000 project (Biodiversity Center of Japan 2003–2015). The results of the surveys are summarized according to two geographical regions (Fig. 4.2). The southern distribution of Japanese coral reefs can be encompassed by seven areas (Setouchi, Daito, Okinawa, Kerama, Miyako, Ishigaki, and Sekisei Lagoon/Iriomote) at south of the Tokara Archipelago. High- latitude coral reef communities can be found at north of Yakushima and Tanegashima and encompassed by nine areas (Tateyama, Kushimoto, Shikoku southwest shore, Iki, Amakusa, Southern seacoast of Kagoshima Pref., Yakushima, Kodakarajima, and Chichijima).

Monitoring projects were conducted with between 5 and 125 sites surveyed per area, utilizing the "spot check" method (Nomura 2004): sites in which corals exhibiting signs of growth anomalies (GAs), black band disease (BBD), and white syndrome (WS) have been recorded, in addition to locations in which signs deviating from the abovementioned diseases have also been reported.

The results of the surveys related to disease occurrence have been summarized and shown in Table 4.3. While there are areas in which diseases have not been reported, GAs have been reported from eight areas (Kushimoto, Shikoku southwest shore, Amakusa, Yakushima, Chichijima, Kerama, Miyako, and Sekisei Lagoon/Iriomote). BBD has been reported from two areas (Kerama and Sekisei Lagoon/Iriomote), and WS from eight areas (Kushimoto, Shikoku southwest shore, Amakusa, Chichijima, Setouchi, Miyako, Ishigaki, and Sekisei Lagoon/Iriomote) (Table 4.3). Disease occurrence was highest at Sekisei Lagoon/Iriomote, with all three disease categories being identified from the site since 2003. In 2004, WS was identified in species of *Acropora*; BBD in encrusting *Montipora*, *Echinopora lamellosa*, and *Pachyseris*; and GAs in tabulate or corymbose *Acropora* and *Montipora* species. In 2014, of the 125 sites surveyed, WS was identified from 115 of those (92%), GAs from 35, and BBD from 14. GAs were reported from 2006 in the Chichijima area of the Ogasawara Islands. In 2007 and 2008, a high number of cases of GAs were reported for Acroporid species, including *A. hyacinthus* and *A. florida*. In the Miyako area, only one case of WS was reported in 2006. However, by 2008, WS was identified in most surveyed sites within the area. Since 2008, WS was reported from one or two sites, highlighting the large shift in disease occurrence in some areas.

Other diseases and disease-like (DL) signs distinguishable from the aforementioned diseases have also been reported from survey areas. Since 2004, patch-like patterns of coral tissue necrosis have been reported from *A. hyacinthus* in the Kushimoto area. The occurrence of the disease increased between 2009 and 2014, with the patch-like signs appearing in approximately half of the survey areas. Observations of the lesion front with a microscope revealed a high abundance of a rod-shaped protozoan (length = 500 μm, width 80 μm). This disease may be related to skeletal eroding band (Antonius and Lipscomb 2000) or brown band disease (Willis et al. 2004) but further investigation is required. Colonies of *Turbinaria peltata* in the same area were also discovered exhibiting small white patches (<1 cm circular spots) in 2012 and identified as white spot syndrome (Yamashiro and Fukuda 2009).

4.2.2 Local to Regional-Scale Monitoring

From the summer of 2009 to the spring of 2010, Okinawa Prefecture conducted a coastal survey of the islands, encompassed by nine areas (Fig. 4.3). Each area was surveyed once, employing the manta method or the spot check method. Data on coral cover and abundance of major species, severity of bleaching and disease, and presence/absence of predatory species were collected. A summary of the data is provided in Table 4.4.

Disease surveys were conducted via the manta method (English and Baker 1994), with diseases categorized into BBD, WS, GAs, and others. BBD was only identified in the Miyako area, in 0.1% of corals. WS was identified from six areas, with percent diseased corals ranging from 0.2% in Ihea and Izena Islands to as high as 3.1% in Ie, Aguni, and Tonaki areas. GAs were identified in two regions, Ie/Aguni and Tonaki/Hateruma. 0.6% and 10.9% of corals in the two regions exhibited signs of GAs, respectively.

4.3 Current States

The goal of this section is to outline the current state research concerning coral disease in Japan. It summarizes the five epidemiological surveys that have been conducted. In addition, this section introduces the three pathological studies conducted mainly on three types of coral disease current found in Japan.

4.3.1 Epidemiological Surveys

Of the five epidemiological surveys of Japanese coral reefs that have been conducted, two comprehensive studies focusing on all diseases currently known have been conducted by Weil et al. (2012) and Wada et al. (in press). Three have reported on a subset of known diseases (Asoh 2008, 2009; Yamashiro and Fukuda 2009; Irikawa et al. 2011).

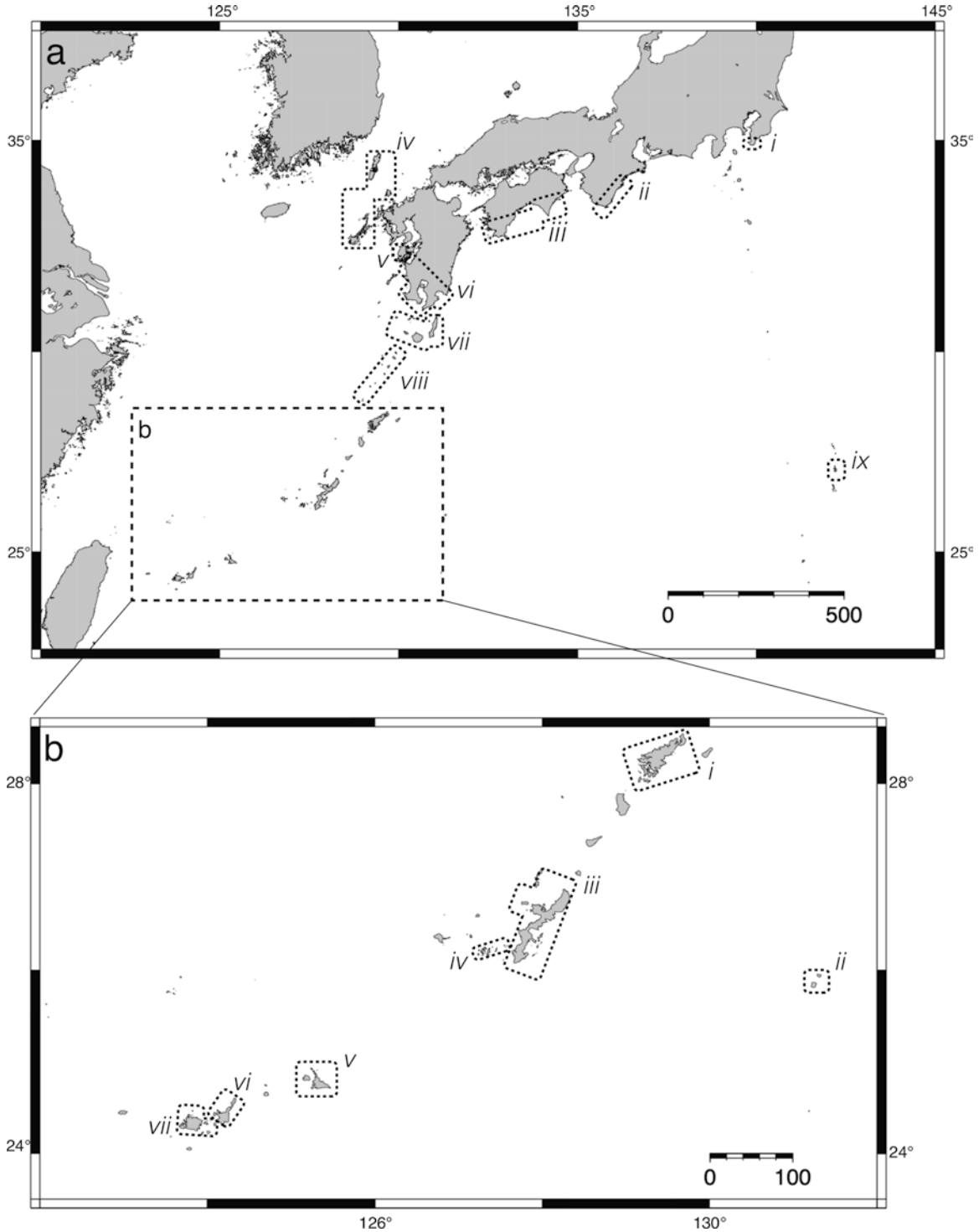

Fig. 4.2 Area surveyed as part of the monitoring sites, 1000 (national government-based survey). (**a**) High latitude coral community areas: (*i*) Tateyama, (*ii*) Kushimoto, (*iii*) Shikoku southwest shore, (*iv*) Iki, (*v*) Amakusa, (*vi*) Southern seacoast of Kagoshima Pref., (*vii*) Yakushima, (*viii*) Kodakarajima, and (**a-***ix*) Chichijima. (**b**) Lower latitude survey area: (*i*) Setouchi, (*ii*) Daito, (*iii*) Okinawa, (*iv*) Kerama, (*v*) Miyako, (*vi*) Ishigaki, and (*vii*) Sekisei Lagoon/ Iriomote

Table 4.3 Confirmed diseases from sites within areas surveyed under the Monitoring Sites 1000 Project (National government survey)

Area (Location in Fig. 4.2)	States	2003	2004	2005	2006	2007	2008	2009	2010	2011	2012	2013	2014
Tateyama (a-i)	Total	n.d.	5	5	5	6	6	6	6	6	6	6	6
	BBD												
	GAs												
	WS												
	Others												
Kushimoto (a-ii)	Total	15	14	14	17	17	16	16	14	18	17	18	15
	BBD												
	GAs										[a]	2	
	WS										[a]	1	
	Others		1	1	1	1	2	8<	1<	11	[a]	15[b]	9
Shikoku southwest shore (a-iii)	Total	11	52	30	29	29	30	30	31	33	31	31	32
	BBD												
	GAs											[d]	
	WS						2[c]					[d]	
	Others											[d]	
Iki (a-iv)	Total	n.d.	4	10	10	14	14	15	15	15	15	15	12
	BBD												
	GAs												
	WS												
	Others				2		1[e]		1				
Amakusa (a-v)	Total	12	15	15	15	15	15	15	15	15	15	15	15
	BBD												
	GAs												
	WS						8	8	[f]	[f]	[f]	[f]	11
	Others												
Southern seacoast of Kagoshima prof. (a-vi)	Total	n.d.	16	18	18	18	18	17	17	16	15	16	15
	BBD												
	GAs												
	WS												
	Others							3					
Yakushima (a-vii)	Total	n.d.	17	18	18	19	19	19	19	19	19	19	19
	BBD												
	GAs										3		
	WS												
	Others	[g]	[g]	[g]	[g]	[g]	[g]						
Kodakarajima (a-viii)	Total	n.d.	n.d.	13	n.d.	n.d.	n.d.	n.d.	23	n.d.	n.d.	n.d.	n.d.
	BBD												
	GAs												
	WS												
	Others												

(continued)

Table 4.3 (continued)

Chichijima (a-ix)	Total	10	12	12	12	12	12	12	12	12	12	12	12
	BBD												
	GAs				1	[h]	[h]	[h]	[h]	[h]	[h]	[h]	[h]
	WS												
	Others					[h]	[h]	[h]	[h]	[h]	[h]	[h]	[h]
Setouchi (b-i)	Total	n.d.	15	15	15	15	15	15	15	15	15	15	15
	BBD												
	GAs					1	1	1	1				
	WS												
	Others												
Daito (b-ii)	Total	n.d.	n.d.	n.d.	n.d.	15	n.d.	n.d.	n.d.	18	n.d.	n.d.	n.d.
	BBD												
	GAs												
	WS												
	Others												
Okinawa (b-iii)	Total	17	94	60	60	65	67	55	69	71	67	51	69
	BBD												
	GAs												
	WS												
	Others												
Kerama (b-iv)	Total	10	10	10	10	10	10	10	12	12	12	12 [j]	12
	BBD												
	GAs				1								
	WS												
	Others				[i]						1	[j]	7
Miyako (b-v)	Total	n.d.	12	14	14	14	14	14	14	14	14	14	14
	BBD												
	GAs				3								
	WS				1	8	9	2<	1	1	1	1	2
	Others												
Ishigaki (b-vi)	Total	75	75	75	75	75	77	77	77	77	77	77	77
	BBD												
	GAs												
	WS					[k]	[k]	[k]					
	Others											[l]	[l]
Sekisei Lagoon/Iriomote (b-vii)	Total	123	123	123	123	123	125	125	125	125	125	125	125
	BBD	4	5	4	6	1	3	8	10	12	11	14	14
	GAs	9	41	34	57	37	50	48	27	49	41	49	35
	WS	31	80	103	113	112	110	109	101	102	106	115	118
	Others												

(continued)

Table 4.3 (continued)

Total total number of sites surveyed, *BBD* black band disease, *GAs* growth anomalies, *WS* white syndrome, *n.d.* no data
[a]WS, GAs, white spot syndrome (WSS), and disease-like (DL) were observed at many sites
[b]DL was found in 12 sites, WSS at 2 sites, what is believed to be *Porites* pink spot syndrome (PPSS) was observed at 1 site
[c]What was thought to be WS was observed at two sites
[d]Of the ten sites in which diseases were confirmed, WS and GA were observed in most. PPSS and white patch syndrome were observed on several coral colonies at a portion of the surveyed sites
[e]Brown band disease-like sign was observed at one site (BrBD)
[f]WS was observed at most sites between 2010 and 2011, but only at a few sites between 2012 and 2013
[g]Observations of DL were continued from 2004, with an increase seen in 2009
[h]GAs and DL were observed on colonies at 9 sites in 2007, 12 sites in 2008, 11 sites in 2009, 11 sites in 2010, 10 sites in 2011, 11 sites in 2012, 10 sites in 2013, and 9 spots in 2014
[i]BBD and DL were observed at eight sites
[j]While DL was observed for several sites, no additional information available
[k]WS identified from 44% of sites in 2007, but numbers were showing decline in 2008 and 2009
[l]DL was observed in 2013, and increases were seen in 2014

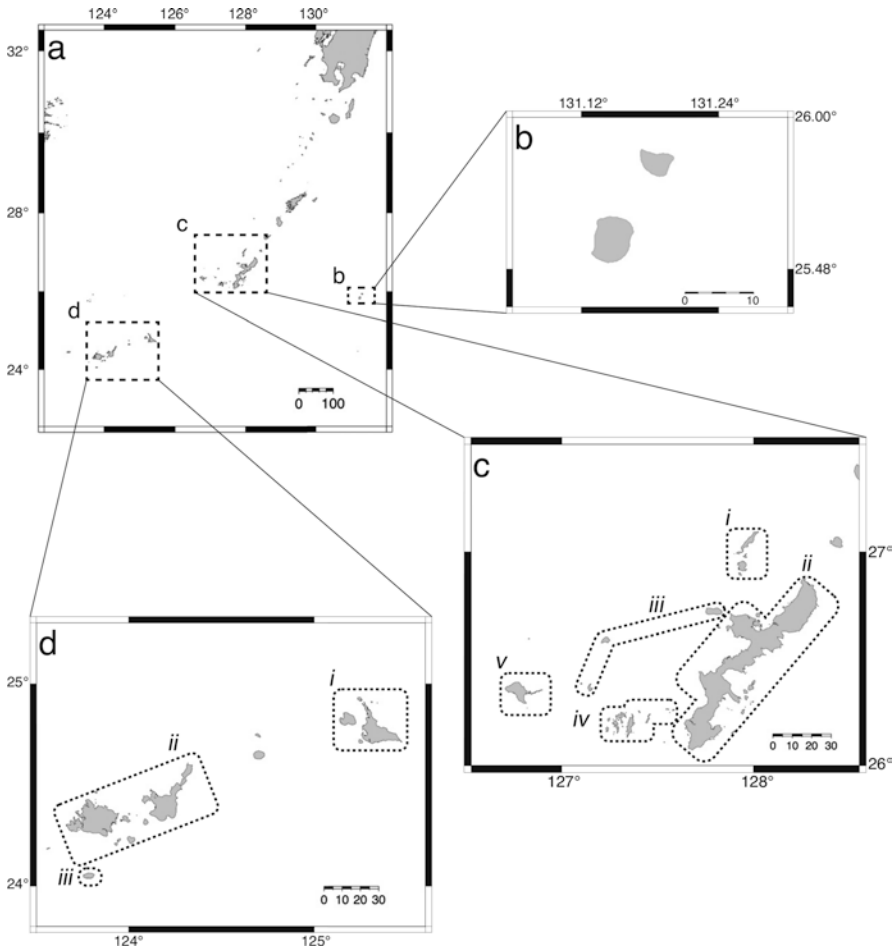

Fig. 4.3 (**a**) Local to regional-scale monitoring survey areas (conducted by the Okinawa Prefecture). (**b**) Daito Island region, and (**c**) Okinawa Islands (*i*. Iheya/Izena, *ii*. Okinawa Island, *iii*. Ie/Aguni/Tonaki, *iv*. Kerama Islands, *v*. Kume Island), and (**d**) Yaeyama Islands region (*i*. Miyako Island, *ii*. Yaeyama Islands, and *iii*. Hateruma)

The study conducted by Weil et al. (2012) surveyed scleractinian corals, soft corals, sponges, as well as crustose coralline algae for signs of disease and was conducted across four reef sites, two at Ginowan-Oyama, Okinawa Island, and two at Zamami, Kerama Islands, in spring and autumn of 2010. The survey identified seven diseases: black band disease (BBD), white syndrome (WS), pigmentation response (PR), *Porites* ulcerative white spots (PUWS), brown band disease (BrBD), crustose coralline white syndrome (CCWS), and compromised health condition (CH) (Fig. 4.4). Of the seven, PR, PUWS, BrBD, and CCWS have never been reported in Japanese reefs previously. First described in the Philippines in 1996, PUWS is characterized by small ovoid areas of bleached or necrotic tissue, leading to a multifocal pattern of skeleton exposure (Raymundo et al. 2003, 2008). Weil et al. (2012) reported colonies of both *Porites lutea* and

Table 4.4 Summarized results of the local to regional-scale monitoring (conducted by Okinawa Prefecture) by area

Area (location in Fig. 4.3)		Okinawa Island (C-ii)	Kerama Islands (C-iv)	Kume Islands (C-v)	Yaeyama Islands (D-ii)	Iheya/Izena (C-i)	Ie/Agunii/Tonaki (C-iii)	Miyako Island (D-i)	Daito (B)	Hateruma (D-iii)
Survey period		Sep. 2009–Jan. 2010	Jun. 2010–Nov. 2010	Sep. 2010–Feb. 2011	Jul. 2010–Mar. 2011	Sep. 2011	Feb. 2010–Jul. 2011	Jun. 2011–Feb. 2012	Aug. 2011	Apr. 2010
Number of survey point		154	56	17	107	18	16	30	2	7
Composition of coral cover										
	<5%	62.3	5.3	11.8	1.9					14.3
	5–10%	27.9	35.1	17.6	21.5	61.1	31.3	16.7	50.0	
	10–25%	9.0	42.1	41.2	27.1	38.9	50.0	63.3	50.0	57.1
	>25%	0.6	17.6	29.4	49.5		18.8	20.0		28.6
Dominant composition of coral species (%)										
	Porites spp.	29.0	18.6	9.9	7.2	3.0	4.3	6.9	12.9	
	Pocillopora spp.	18.9	11.7	19.4	2.9	4.4	18.7	0.3	43.0	2.0
	Acropora spp.	14.2	16.2	28.5	44.0	33.5	28.9	46.8		10.3
	Montipora spp.	1.8	2.0		2.9	2.2		2.0		7.3
	Faviidae	1.5		0.9	0.1			0.5		
	Multiple species	4.4	38.0	38.5	30.7	45.3	45.3	29.4	38.4	63.0
	Others	0.8	7.5	1.6	6.5	1.0	0.8	1.9	1.0	5.8
	Non-dominant[a]	29.4	6.1	1.3	5.7	10.6	39.9	12.2	5.7	11.6
Disease states (%)										
	Healthy	96.1	98.4	97.3	97.4	99.8	96.3	98.3	100.0	89.1
	BBD							0.1		
	WS		0.3	0.7	1.8	0.2	3.1	0.5		
	GA						0.6			
	Others		1.1							10.9
	n.d.	3.9	0.3	2.0	0.7			1.1		

[a]Areas in which a dominant species or coral colonies cannot be identified

P. lobata in their survey sites to be affected with PUWS. Colonies of *P. lutea* were detected the disease at a frequency of 23.1 ± 10.4% in Ginowan-Oyama.

Wada et al. (in press) surveyed all coral colonies found within an approximately 70,000 m^2 area of reef off of Akajima Island, Kerama Islands from May to July in 2010 and 2011, generating a map of disease distribution. BBD, skeletal eroding band (SEB), GAs, WS, as well as coral bleaching were confirmed between the two survey sites (Figs. 4.5 and 4.6). BBD was predominantly confirmed in encrusting *Montipora* spp., GAs in *Acropora* sp. and encrusting *Montipora* spp., and WS in *Acropora* sp. SEB, which was observed in *Montipora* spp. and *Pocillopora* spp., has not been reported in Japan previously. The most commonly encountered disease for both sites was BBD, with the characteristic black band patterns being formed by an aggregation of microbes, including cyanobacteria. In order to determine the pattern of occurrence for BBD, spatial analysis for the distribution of infected encrusting *Montipora* spp. was conducted. Infected colonies were distributed in aggregates, similar to what has been reported for cases of BBD infections of *Favia* in the Gulf of Aqaba (Zvuloni et al. 2009), a yellow band disease outbreak of *Orbicella* in the Gulf of Mexico (Foley et al. 2005), and *Aspergillosis* infections in sea fans in the Florida Keys (Jolles et al. 2002). The mode of transmission of the diseases is likely a result of a water-borne infection. BBD cases in *Montipora* exhibited significant variation in signs, even between colonies found within the same reef site. The rate of progression of the black band front varied from 1.1 to 5.5 mm per day in 2010 ($n = 15$) and 1.2–3.8 mm per day in 2011 ($n = 13$), with variation seen between colonies of the same reef. The band on each colony also changed in coloration and morphology as the diseased progressed.

Asoh (2008) reported multifocal pattern of circular or irregular areas of tissue loss on the branches of *Porites cylin-*

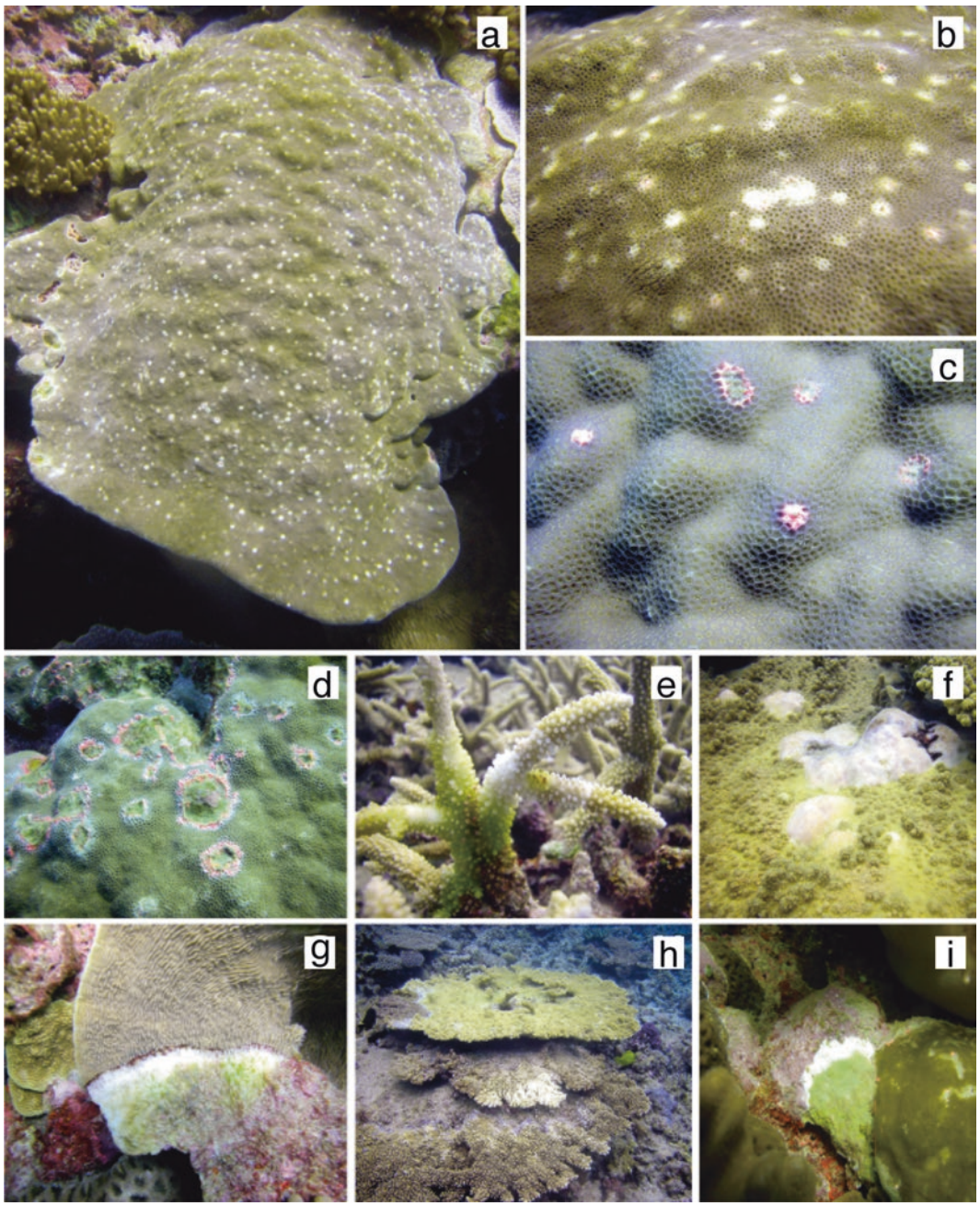

Fig. 4.4 Coral diseases identified by Weil et al. (2012) in 2010 at Kerama Islands and the Ginowan region in Okinawa Prefecture. (**a** and **b**) Colonies of *Porites lutea* showing signs of *Porites* ulcerative white spots (*PUWS*). (**c** and **d**) Pigmentation responses expressed as *pink spot* and *pink lines* on *Porites* colonies. (**e**) Brown band-like disease signs on *Acropora nobilis*. (**f**) Growth anomalies (*GAs*) on *A. cytherea*. (**g**) Black band disease (*BBD*) on *Pachyseris speciosa*. (**h**) White syndrome (*WS*) on *A. florida*. (**i**) Crustose coralline white syndrome on unidentified coralline algae (Photos by E. Weil)

drica in Shiraho reef, Ishigaki Island, Okinawa, in August and September of 2007. Areas of skeleton exposure were characterized by thin tissue with a white opaque border. Line transect surveys were also conducted in September of 2007 and January of 2008 in order to determine the mean proportion of affected *P. cylindrica* branches. As the result, $0.52 \pm 0.16\%$ and $0.08 \pm 0.06\%$ of branches exhibited signs in the two survey months, respectively. Lesion size also varied between the 2 months, with colonies exhibiting larger lesions in higher proportions of the branches (33/34), compared to January of 2008 (5/39), suggesting that severity of the infection increases during the summer months. While the cause of the lesions is unknown, the signs were similar to those

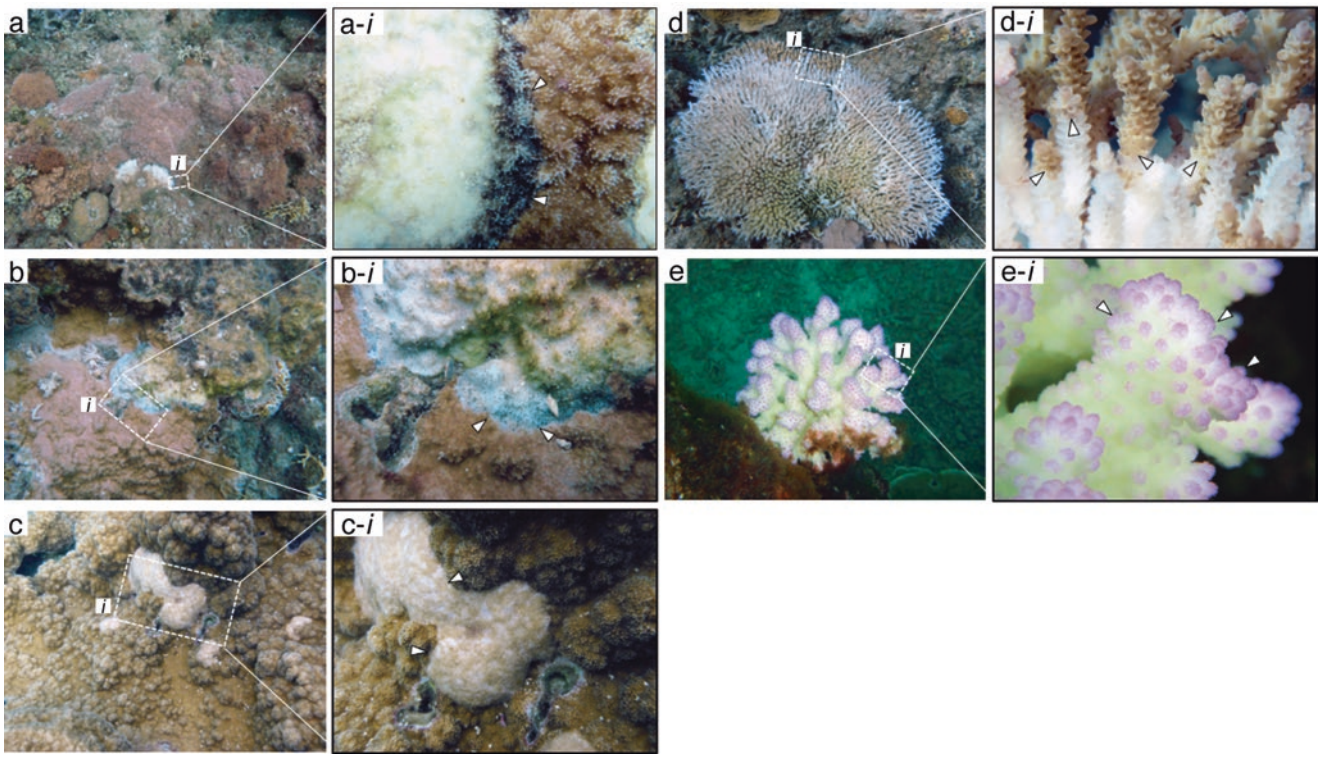

Fig. 4.5 Coral diseases and bleaching identified by Wada et al. (in press) between 2010 and 2011 in Kerama Islands and the region of Okinawa Prefecture. (**a**) Black band disease (*BBD*) on *Montipora* sp. (**b**) Skeletal eroding band (*SEB*) on *Montipora* sp. (**c**) Growth anomalies (*GAs*) on *Montipora* sp. (**d**) White syndrome (*WS*) on *Acropora* sp. (**e**) Bleaching on *Pocillopora* sp. An enlarged view of the box is provided in the corresponding plate labeled (***i***) (Wada et al. in press)

recently reported for cases of WS in *P. cylindrica* in Guam (Lozada-Misa et al. 2015).

In 2008, colonies of *Turbinaria peltata* in the temperate waters around Miyazaki Prefecture were found to have bleached spots approximately 1 cm in diameter on the coenosac (Yamashiro and Fukuda 2009; Fig. 4.7). Named white spot syndrome (WSS), the disease was characterized by gradually increasing spots found covering the colony. A greater than 2/3 reduction in *Symbiodinium* densities was also observed, with the disease eventually causing necrosis of the tissue. Of the 100 colonies surveyed, WSS was confirmed for 1/3 of the coral colonies. Furthermore, colonies of *T. peltata* in Kushimoto, Honshu, were also found to show sign of WSS (Sect. 4.2.1 National Government-Based Surveys), suggesting the potential for future spread of the disease.

In 2011, a survey for the presence of GAs in tabulate acroporids was conducted in Amuro Island of the Kerama Islands. GAs were confirmed for 11 acroporid species (*A. cytherea*, *A. latistella*, *A. hyacinthus*, *A. clathrata*, *A. florida*, *A. valenciennesi*, *A. palifera*, *A. abrotanoides*, *A. samoensis*, *A. digitifera*, and *A. secale*), with *A. cytherea* showing the highest frequency of infection (70/273) in colonies (Irikawa et al. 2011). The researchers found relatively large sizes of *A. cytherea*, with GAs appearing and frequency of occurrence of the disease increasing toward the center of the colony.

4.3.2 Pathological Studies

Pathological studies of coral diseases of Japan have been conducted for GAs, cyanobacteria blooms in gorgonian coral, and BBD.

GAs have been found to affect a variety of coral species from the Caribbean and the Indian Ocean. GAs are tumor-like diseases marked by abnormal skeletal growth (Peters et al. 1997; Sutherland et al. 2004). Since the initial report of tumor-like growth in Japanese corals in 2000 (Yamashiro et al. 2000), several additional cases of the disease have been reported (Yamashiro et al. 2001; Irikawa et al. 2011; Yasuda et al. 2012; Yasuda and Hidaka 2012). Abnormal growth lesions found on colonies of encrusting *Montipora informis* in Sesoko Island, Okinawa showed a reduction in the number of polyps and *Symbiodinium* density in the tissues, as well as finer skeletal structure than normal (Yamashiro et al. 2000). A significant reduction in fecundity was also associated with infected colonies. A threefold reduction in lipid storage relative to dry weight was also seen between healthy (32.2%) and diseased (10.6%) tissue (Yamashiro et al. 2001), as a direct result of reduction in stored wax and triacylglycerol. Furthermore, Yasuda and Hidaka (2012) also reported a lower number of apoptotic nuclei within the oral gastrodermis and calicodermis in disease lesions compared to healthy tissue in *M. informis* and

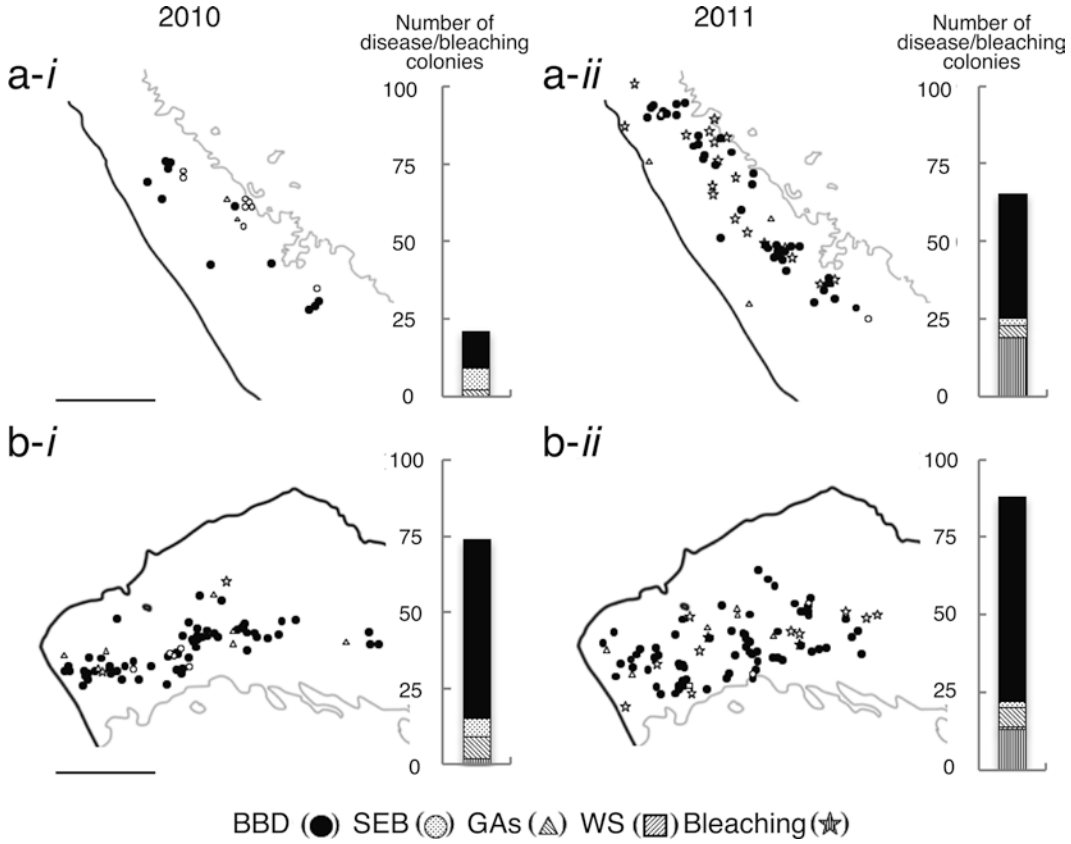

Fig. 4.6 Distribution and prevalence of coral diseases and bleaching identified by Wada et al. (in press) between 2010 and 2011 in Kerama Islands and the region of Okinawa Prefecture. Distribution of colonies showing disease/bleaching signs of black band disease (*BBD*), skeletal eroding band (*SEB*), growth anomalies (*GAs*), white syndrome (*WS*), and bleaching at (**a**) Nishihama and (**b**) Mazyanohama at Kerama Islands, Okinawa in (***i***) 2010 and (***ii***) 2011. *Histograms* indicate the number of diseased and bleached colonies at each site. *Scale bars* indicate 100 m (Wada et al. in press)

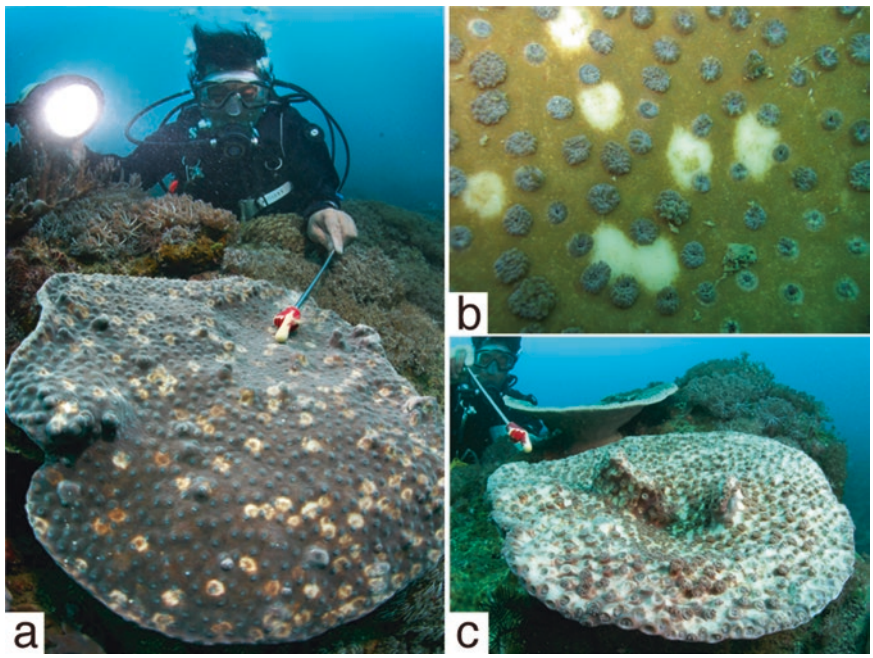

Fig. 4.7 White spot syndrome (WSS) identified by Yamashiro and Fukuda (2009) in 2008 in the waters around Miyazaki Prefecture. (**a**) White spots on the colony of *Turbinaria peltata*. (**b**) Enlarged view of the spots. (**c**) Colony fully covered by WSS

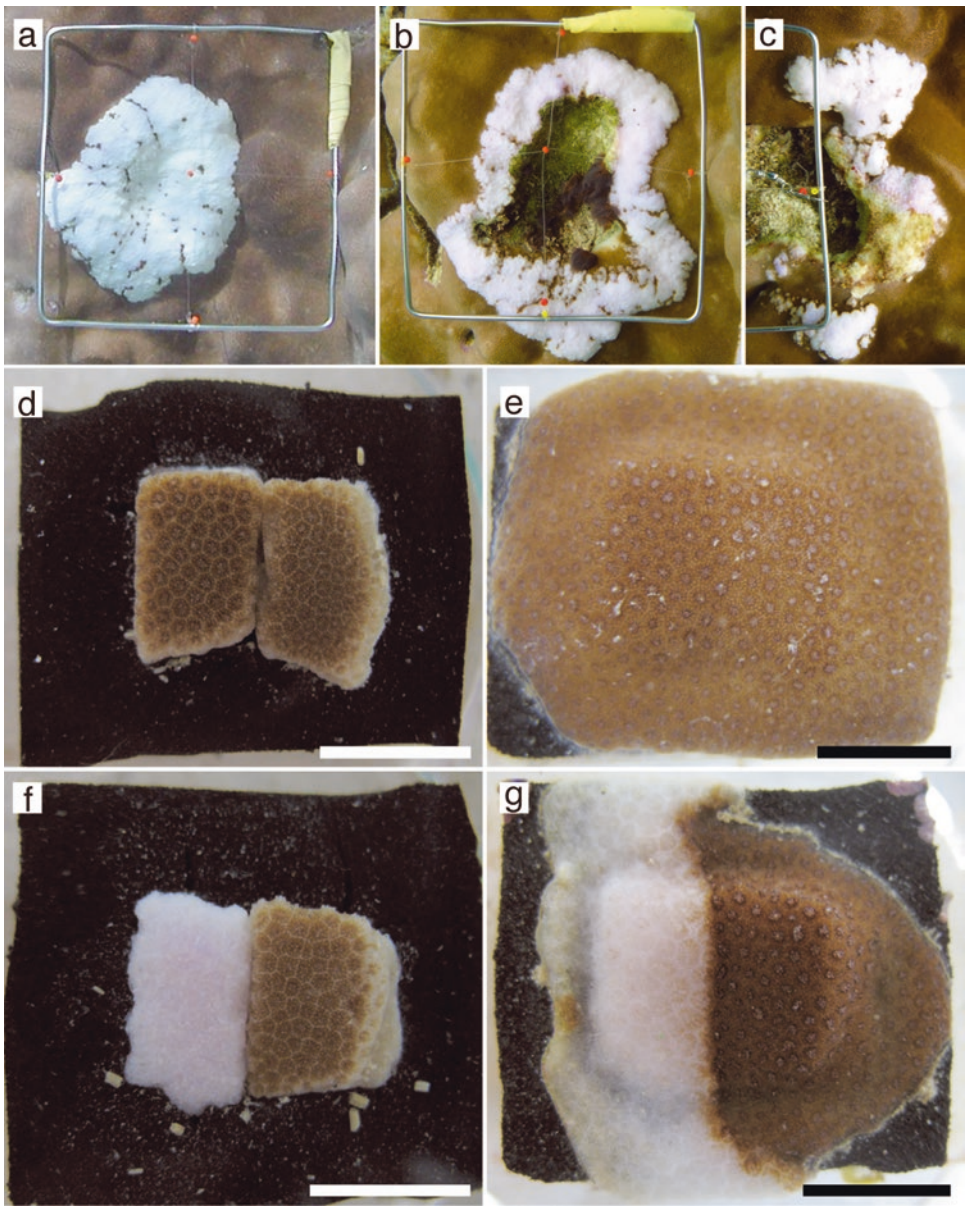

Fig. 4.8 (**a**–**c**) Growth anomalies (*GAs*) identified by Yasuda et al. (2012) from 2003 to 2009. (**a**) GAs on *Porites australiensis* surrounded by healthy tissue. (**b**) GA from plate A observed 4 years later (May 2007) with tissue death occurring from the center outward. (**c**) GAs located at the margin of a colony (April 2009) with tissue death beginning from the margin. (**d**–**g**) Laboratory experiment in which colonies were placed in contact with: (**d** and **e**) healthy tissue before (**d**) and 6 months later (**e**, **f**, and **g**) diseased tissue before (**f**) and 6 months later (**g**). *Squares* and *scale bars* indicate 10 × 10 cm and 1 cm, respectively

Porites australiensis. An increase in BrDU-labeled proliferative cells within the epidermis, gastrodermis, and calicodermis of lesion areas was also seen for both *M. informis* and *P. australiensis*. These results suggest changes to cellular kinetics of disease lesions relative to healthy tissue. Interestingly, disease lesions of *A. cytherea* from Amuro Island, Kerama Islands, and Okinawa showed greater rates of progression in lesions smaller than 15 cm. Lesions greater than 10 cm were characterized by necrosis at the center (Irikawa et al. 2011). Long-term monitoring of *P. australiensis* colonies found in the back reef at Kayo, Okinawa, between 2003 and 2009 showed variable progression of the GAs depending on the location of the lesion (Yasuda et al. 2012). In cases where the disease lesions were located near the colony center, progression into necrosis also began at proximal to the center. However, for lesions toward the colony margins, the tissue necrosis began from the margin of the lesions (Yasuda et al. 2012; Fig. 4.8a–c). The mode of transmission of GA is currently a topic of contention. Field transplant experiments in which healthy *A. cytherea* fragments were placed in contact with disease lesions did not result in transmission of the disease to healthy fragments, even after 2 weeks of contact (Irikawa et al. 2011). Aquarium experiments on *P. australiensis* by Yasuda et al. (2012) also showed GAs to be non-contagious by contact, as healthy fragments in contact with diseased lesions continued healthy growth. Furthermore, the corals continued to show normal growth even after 6 months and fused completely with the healthy and diseased fragments (Fig. 4.8d–g). On the other hand, Kaczmarsky and Richardson (2007) reported contrasting results as successful transmission was established for *Porites* corals.

Aspergillosis, the most commonly reported disease in gorgonian corals (sea fans), has been previously reported

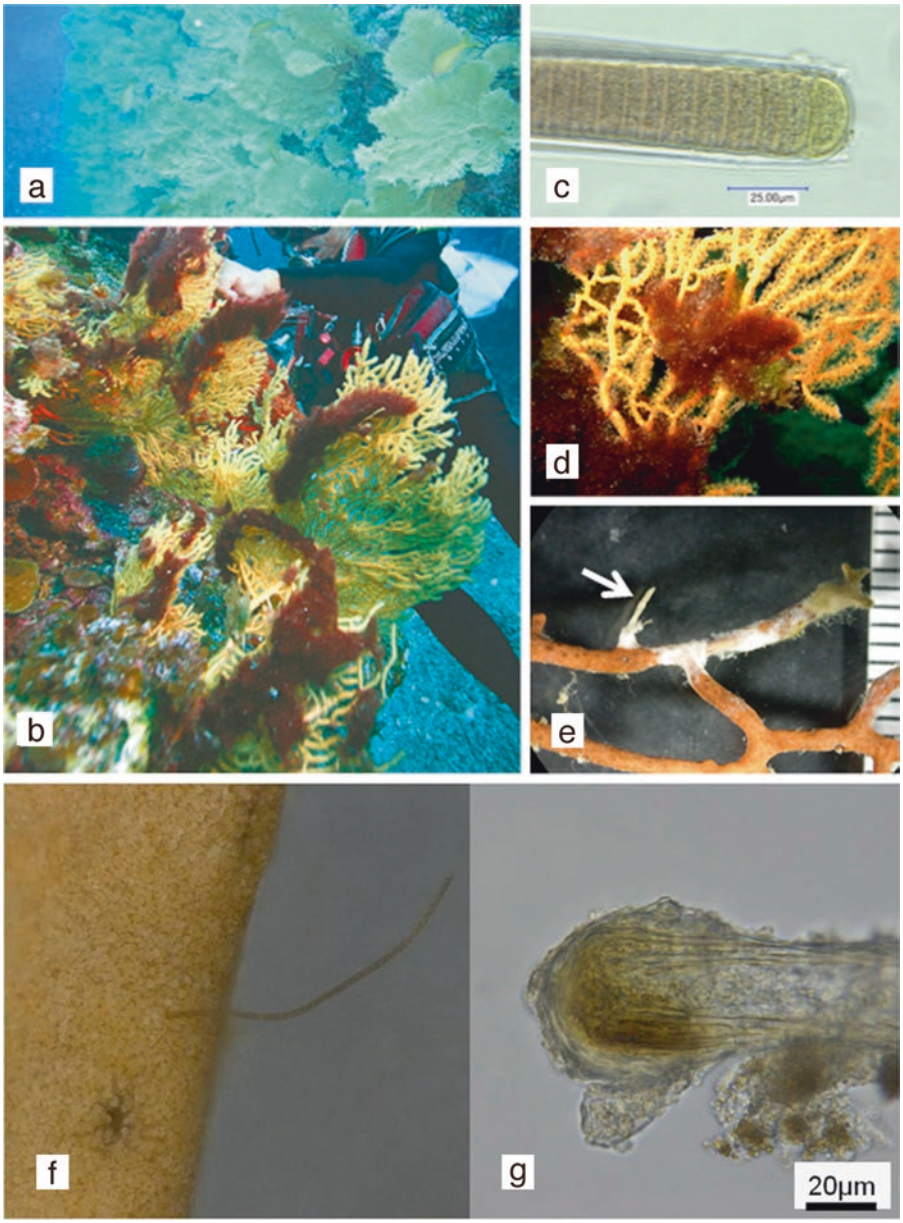

Fig. 4.9 Cyanobacteria bloom on the gorgonian coral *Annella reticulata* in Kerama Islands identified by Yamashiro et al. (2014). (**a**) Colonies of the gorgonian coral *Annella reticulata*. (**b**) Colonies overgrown by the cyanobacterium *Moorea bouillonii*. (**c**) Enlarged view of *M. bouillonii*. (**d**) Close up of coral branches entangled by cyanobacterial mats. (**e**) Removal of cyanobacterial mats revealed white dead branch areas and collapsed branch leaving only a central axis (*arrow*). (**f**) *M. bouillonii* penetrating into host coral tissue. (**g**) Terminal end of *M. bouillonii* showing a swollen structure composed of a multilayer sheath

from the Caribbean in *Gorgonia ventalina* and is caused by the fungal pathogen *Aspergillus sydowii* (Geiser et al. 1998). While a disease has been reported for gorgonians in Japan, the causative agent appears to be cyanobacteria. In March of 2009, branches of *Annella reticulata* colonies, which found at 20 m depth off the coast of Kerama Islands in Okinawa, were covered by a filamentous cyanobacterial mat, which appeared to cause tissue necrosis (Yamashiro et al. 2014; Fig. 4.9). Determination of the bacterial mats by 16S sequencing revealed the biofilm mostly comprised of a microbe closely related to common benthic cyanobacterium *Moorea bouillonii* (Yamashiro et al. 2014). The authors pointed out the anoxic environment at the boundary between tissue and the cyanobacterial mat as the cause of necrosis but also suggested the involvement of cytotoxic macrolides and peptides produced by *M. bouillonii* (Matthew et al. 2010). Observations by microscopy revealed tissue penetration by the cyanobacteria lending strong support to the infection process and resultant necrosis (Yamashiro et al. 2014).

First reported in 1973 in the Caribbean waters off the coast of Belize, BBD has been subsequently identified in corals worldwide (Sutherland et al. 2004). An aggregation of cyanobacteria, sulfate-reducing bacteria, sulfide-oxidizing bacteria, and a multitude of heterotrophic bacteria often characterize the distinguishable black band associated with the disease. Progression of the disease usually leads to tissue necrosis (Richardson 2004; Bourne et al. 2009). Transmission has been demonstrated in experiments in which healthy colonies are placed in contact with those that are diseased, suggesting the disease to be highly contagious (Kuta and Richardson 1996). Since its confirmation in 2003 at Sekisei Lagoon, found between Ishigaki Island and Iriomote Island (Sect.

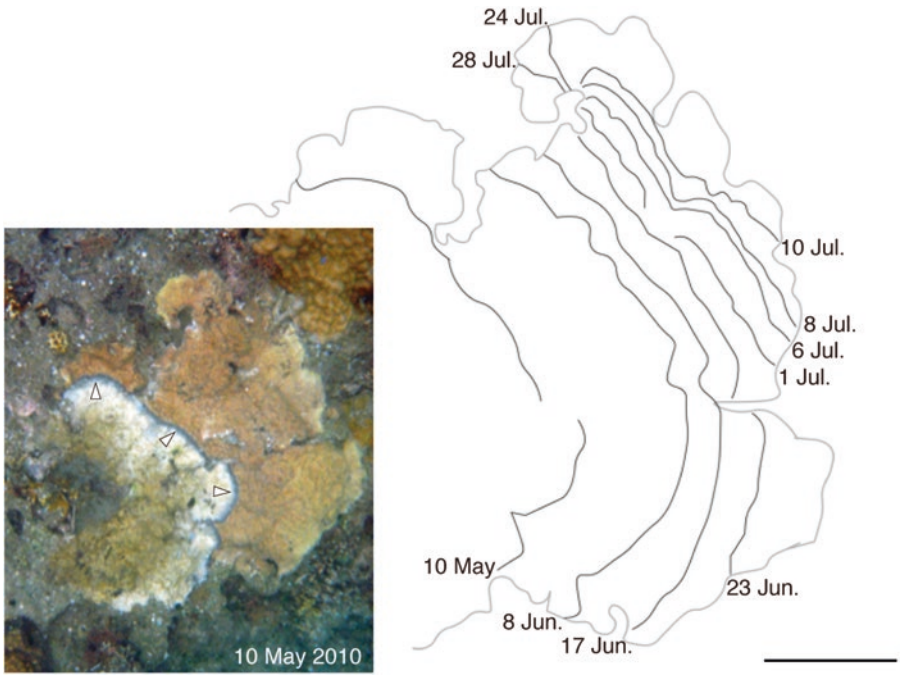

Fig. 4.10 Lesion front (*arrow heads*) of black band disease (*BBD*) observed by Wada et al. (in press) in 2010 at Kerama Island, Okinawa. Illustration shows the progression front of the BBD lesion from May 10th to July 28th in 2010. *Scale bar* indicates 10 cm (Wada et al. in press)

4.2.1 National Government-Based Surveys), additional reports have been made in areas such as Kerama Islands (Wada et al. in press; Weil et al. 2012) and Okinawa (Yamashiro 2014) of Japan. Based on the initial observations of variation in lesion progression of BBD in encrusting *Montipora* spp., a comprehensive survey of *Montipora* colonies off of Akajima Island, Kerama Islands was conducted over a span of 3 months (Wada et al. in press). While the band forming lesion front was found to radiate from a centralized area, as reported by other studies (Fig. 4.10), observations also revealed band coloration to vary within a colony, ranging from grey to white in some areas. Stereomicroscopic observations of samples collected at the end of the study revealed a filamentous cyanobacterium to be highly abundant between the boundary of healthy and diseased tissue (Fig. 4.11a). Additionally, a white filamentous microbe was also found to be associated with areas directly trailing or above the lesion front (Fig. 4.11b). A microbe with two conspicuous peristomial wing, in addition to the ciliate previously identified with SEB, *Halofolliculina corallasia* (Antonius and Lipscomb 2000), were also found to be present in areas of skeleton exposure (Fig. 4.11c). Microscopic observations via decalcification and paraffin sectioning revealed an approximately 3 μm cyanobacterium to dominate around the boundary between healthy and diseased tissue (Fig. 4.11d–e). However, a larger cyanobacterium (approximately 10 μm) (Fig. 4.11e) as well as ciliates (Fig. 4.11f) were also present in some specimens. Furthermore, the increase in sulfate-reducing bacteria with subsequent increase in production of sulfides in sites lesion formation may be related to formation of the black band (Bourne et al. 2011; Glas et al. 2012; Sato et al. 2016). The cyanobacteria associated with BBD is also known to produce the cyanotoxin microcystin (Richardson et al. 2007), which may also play a role in initiating the disease. However, spatial localization of each bacterial taxa associated with the lesion is still unknown, and the mode of progression of the lesion has yet to be determined. Efforts are currently underway to determine the bacterial composition, localization within the lesion, and mode of progression of BBD using techniques that will be discussed in the next section.

4.4 Future Directions

Throughout the archipelago of Japan, corals totaling 415 species have been reported from as far south as the Yaeyama Islands of the Ryukyu archipelago and as far north as Boso Peninsula (Nishihira 2004). Increased sea surface temperature due to anthropogenic climate change and elevated CO_2 levels leading to ocean acidification is major concerns to these coral reef ecosystems (Hoegh-Guldberg 2011). Coral bleaching and diseases caused by high sea surface temperatures are considered major threats to coral reefs (Harvell et al. 1999, 2002; Hoegh-Guldberg 2011). A survey conducted from 1995 to 2009 across 47 reefs in the Great Barrier Reef showed coral bleaching and disease to be the cause of 5.6% and 6.5% reduction in coral cover over the 15 years, respectively (Osborne et al. 2011). The major bleaching event of 1998 caused significant damage to Japanese reefs (Loya et al. 2001; Kayane et al. 2002). The reefs around Okinawa Island and Ishigaki Island experienced two major bleaching events between 1998 and 2009. As a result, major shifts in coral species composition

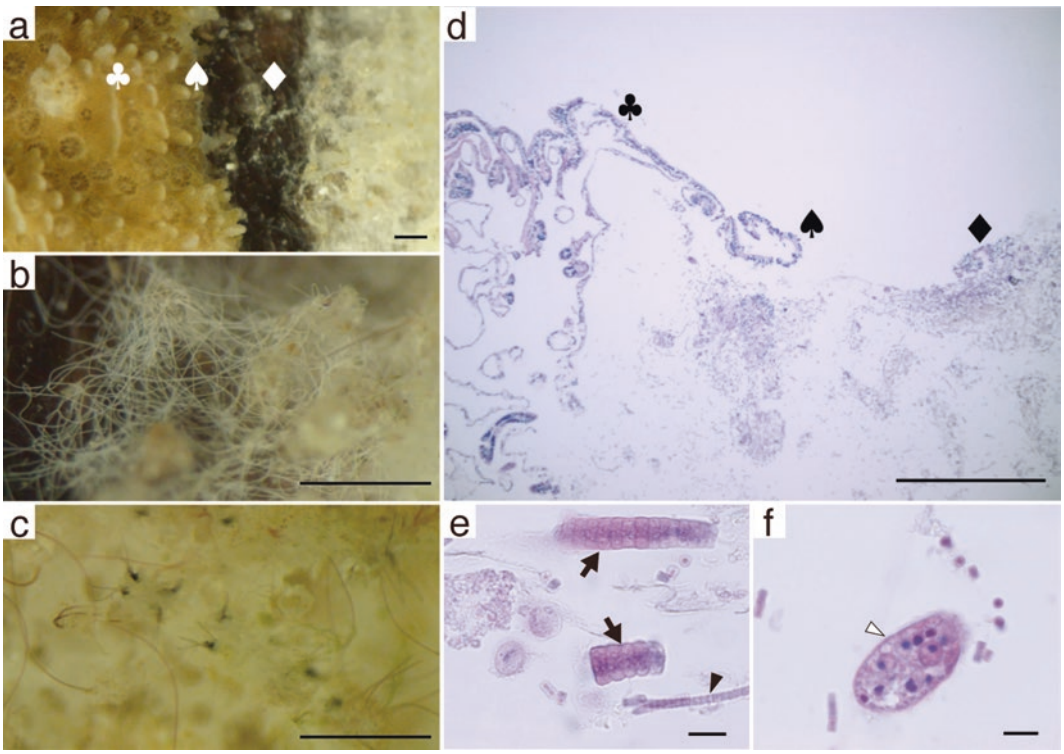

Fig. 4.11 *Montipora* sp. infected with black band disease (BBD) from Kerama Island. (**a**) Overall image of the lesion boundary displaying a black band between tissue and exposed white skeleton. (**b**) White filamentous organisms above the black band surface. (**c**) The ciliate-like *Halofolliculina corallasia* on the skeleton surface. (**d**) Histological section of BBD showed the cyanobacterial mat penetrated into the tissue from right to left. (**e**) Thick (*arrows*) and thin (*arrowhead*) types of the cyanobacteria found in the mats. (**f**) The ciliates containing dinoflagellates found in the mats. The *symbols* indicate the orientation of lesions showing apparently intact coral tissue (♣), lesion front (♠), and cyanobacterial mat (♦). *Scale bars* represent 1 mm (**a**–**c**), 500 μm (**d**) and 10 μm (**e** and **f**).

within the reefs occurred (Hongo and Yamano 2013; Harii et al. 2014). However, little is known on the diversity and impact of coral disease on Japanese reefs. Due to the increasing deterioration of coral reef by infectious diseases worldwide, gathering data concerning types, occurrence, and impact of the coral diseases will be imperative in developing effective conservation strategies in Japan. Efforts toward understanding the causes of these diseases are also necessary.

While there are over 40 different diseases currently identified worldwide (Bruckner 2015), the cause is only known for a handful of infectious diseases, mainly bacteria (Bourne et al. 2009; Pollock et al. 2011). While fulfilling Koch's postulate (Koch 1884; Grimes 2006) has often confounded identification of the causative agent of coral diseases, these difficulties stem from several factors:

(i) The majority of marine bacteria are unculturable (Kogure et al. 1980; Ferguson et al. 1984; Eguchi and Ishida 1990).
(ii) Disease pathogens can be masked by opportunistic infections (Ainsworth et al. 2007).
(iii) There are difficulties in replicating natural conditions in a controlled or laboratory setting (Lesser et al. 2007).
(iv) Some coral diseases are a synergistic effect of multiple causative agents (e.g., BBD is considered a polymicrobial disease) (Sato et al. 2016).

Thus, molecular genomics (Pollock et al. 2011), immunological tools (Palmer et al. 2008), as well as histopathology (Pollock et al. 2011, Work and Meteyer 2014) are important approaches in enhancing our understanding of coral disease. Although a trained eye is required for histopathology work, only a light or fluorescence microscope is necessary to detect the spatial presence of lesions associated with microbes (fungi, protozoa, and bacteria), making it a common diagnostic tool in coral pathology (Work and Meteyer 2014). Conventional histological preparation for coral studies requires removal of skeleton consisting calcium carbonate (i.e., aragonite, Lowenstam and Weiner 1989). However, the specimens treated with a decalcifying agent potentially leave the interactive orientation between tissue, skeleton canals of gastrovascular canal network uncertain (Gladfelter 1983), owing to the vacant spaces on histological sections resulting from skeleton removal. Additionally, disruption of already fragile tissue can result in loss of tissue or associated microbes can be problematic for histopathology (Bourne et al. 2014). In order to circumvent this issue, the authors utilized a non-decalcifying sectioning method for observations of diseased coral tissue (Wada et al. 2016a; Fig. 4.12). While a cryostat and expensive sectioning knives compared to conventional methods are necessary, this method can dramatically reduce pro-

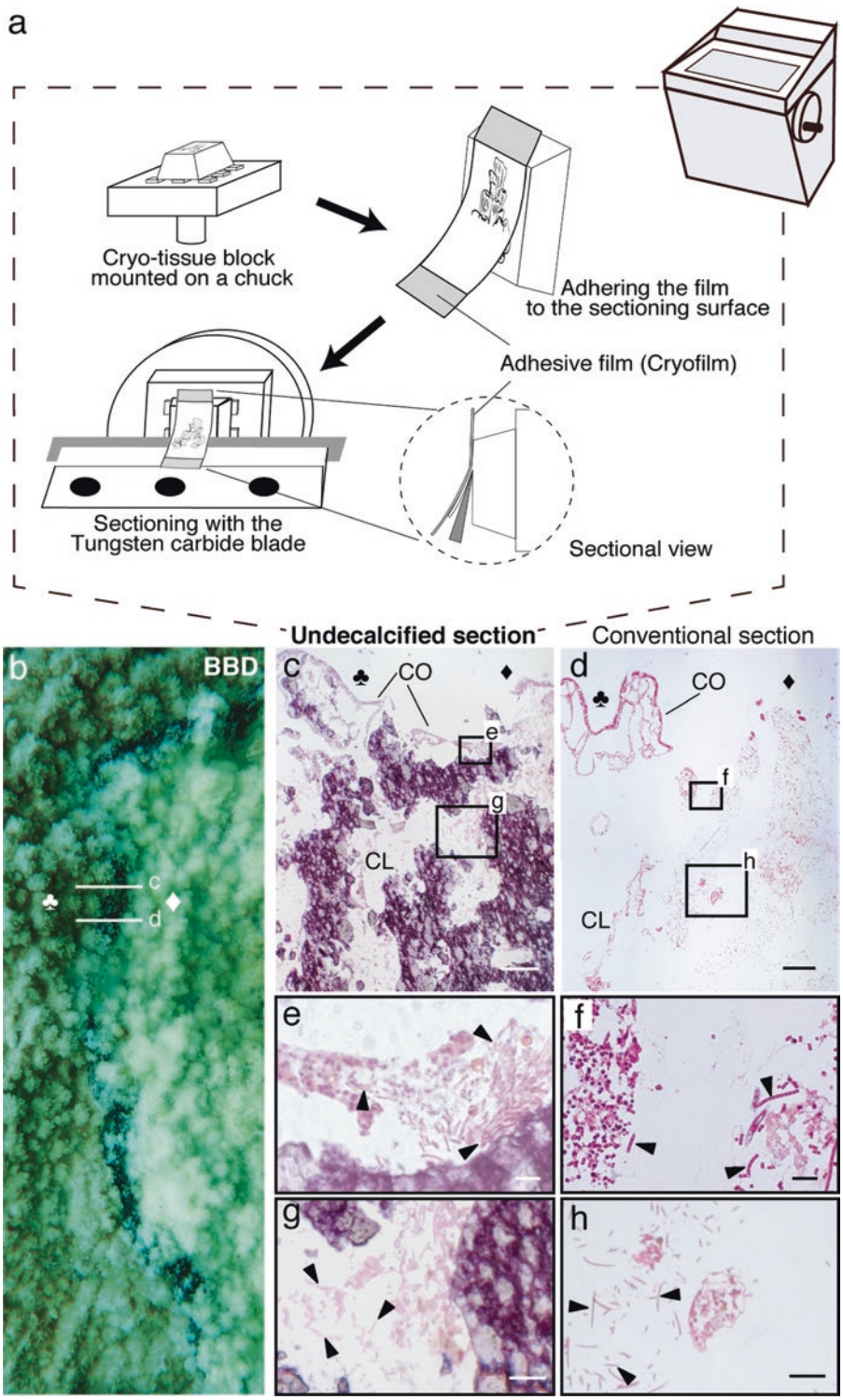

Fig. 4.12 (**a**) Simplified illustration of the un-decalcified sectioning method shown by Wada et al. (2016a). (**b**) *Montipora* sp. infected with black band disease (*BBD*). (**c**, **e** and **g**) *Montipora* sp. disease lesion sectioned via the un-decalcified sectioning method. Dense aggregation of cyanobacterial filaments on the lesion surface as well as between skeletal parts (*black arrowheads*) can be seen clearly. (**d**, **f** and **h**) *Montipora* sp. disease lesion sectioned via the conventional decalcified sectioning method. Cyanobacterial filaments appear loosely associated with BBD-affected tissue in decalcified sections. The *symbols* indicate the orientation of lesions showing apparently intact coral tissue (♣) and exposed skeleton (♦). *Scale bars* represent 200 μm (**c** and **d**), 20 μm (**e** and **f**), and 50 μm (**g** and **h**)

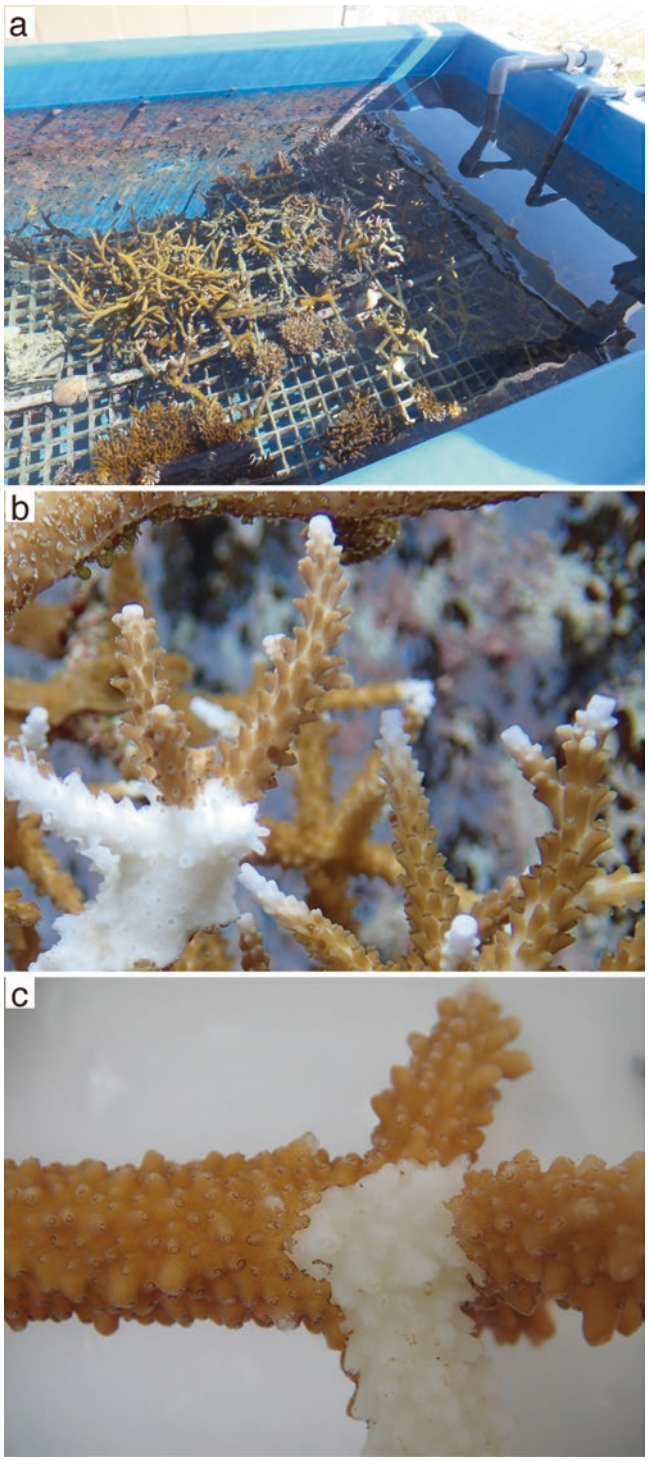

Fig. 4.13 (**a**) Aquarium at a coral aqua culture facility. (**b**) Rapid tissue necrosis (*RTN*; Borneman 2001, Luna et al. 2007) affecting *Acropora* sp. in an aquaculture aquarium. (**c**) Enlarged image of RTN affecting *Acropora* sp. (Photos by R. Nakamura)

cessing time as well as leave tissue near its original state. Research that a part of authors is currently being done on BBD utilizing this technique, in conjunction with identification of bacterial localization using FISH (Wada et al. 2016b), as well as next generation sequencing of the lesion microbiome. This new approach to coral histopathology should become an important tool in determining causative agents for coral disease.

Currently, there have been reported cases of coral diseases in aquariums and coral aquaculture facilities (Sweet et al. 2012). Rapid tissue necrosis (RTN; Borneman 2001, Luna et al. 2007), where sloughing off or death of tissue leading to colony mortality has been common. Cases of RTN have also been confirmed in Japanese aquariums (Wada et al. 2012) and aquaculture facilities (Wada et al. 2015) (Fig. 4.13). Researching diseases affecting cultured corals will also be an important as a holistic approach to coral conservation.

References

Ainsworth TD, Kramasky-Winter E, Loya Y et al (2007) Coral disease diagnostics: what's between a plague and a band? Appl Environ Microbiol 73:981–992. https://doi.org/10.1128/AEM.02172-06

Antonius A (1973) New observations on coral destruction in reefs. 10th Meeting of the Association of Island Marine Laboratories of the Caribbean, University of Puerto Rico (Mayaguez)

Antonius A (1977) Coral mortality in reefs: a problem for science and management. Miami, pp 617–623

Antonius A (1999) *Halofolliculina corallasia*, a new coral-killing ciliate on Indo-Pacific reefs. Coral Reefs 18:300–300. https://doi.org/10.1007/s003380050199

Antonius AA, Lipscomb D (2000) First protozoan coral-killer identified in the Indo-Pacific. Atoll Res Bull 481:1–21

Aronson RB, Precht WF (2001) White-band disease and the changing face of Caribbean coral reefs. In: Porter JW (ed) The ecology and etiology of newly emerging marine diseases. Springer, Netherlands, pp 25–38

Asoh K (2008) Loss of live tissue and exposure of coral skeleton in *Porites cylindrica* at Shiraho, Ishigaki Island, Japan. Galaxea J Coral Reef Stud 10:45–45. https://doi.org/10.3755/galaxea.10.45

Asoh K (2009) Recovery of *Porites cylindrica* from the 2007-summer tissue loss at Shiraho, Ishigaki Island, Japan. Galaxea J Coral Reef Stud 11:27–32. https://doi.org/10.3755/galaxea.11.27

Bak RPM (1983) Neoplasia, regeneration and growth in the reef-building coral Acropora palmata. Mar Biol 77:221–227. https://doi.org/10.1007/BF00395810

Bellwood DR, Hughes TP, Folke C, Nyström M (2004) Confronting the coral reef crisis. Nature 429:827–833. https://doi.org/10.1038/nature02691

Ben-Haim Y, Rosenberg E (2002) A novel *Vibrio* sp. pathogen of the coral *Pocillopora damicornis*. Mar Biol 141:47–55. https://doi.org/10.1007/s00227-002-0797-6

Ben-Haim Y, Zicherman-Keren M (2003) Temperature-regulated bleaching and lysis of the coral *Pocillopora damicornis* by the novel pathogen *Vibrio coralliilyticus*. Appl Environ Microbiol 69:4236–4242. https://doi.org/10.1128/AEM.69.7.4236

Biodiversity Center of Japan (2003) The annual report of the "Monitoring Sites 1000" in Japan: coral reefs. Biodiversity Center of Japan, Ministry of the Environment of Japan, Japan. (in Japanese)

Biodiversity Center of Japan (2004) The annual report of the "Monitoring Sites 1000" in Japan: coral reefs. Biodiversity Center of Japan, Ministry of the Environment of Japan, Japan. (in Japanese)

Biodiversity Center of Japan (2005) The annual report of the "Monitoring Sites 1000" in Japan: coral reefs. Biodiversity Center of Japan, Ministry of the Environment of Japan, Japan. (in Japanese)
Biodiversity Center of Japan (2006) The annual report of the "Monitoring Sites 1000" in Japan: coral reefs. Biodiversity Center of Japan, Ministry of the Environment of Japan, Japan. (in Japanese)
Biodiversity Center of Japan (2007) The annual report of the "Monitoring Sites 1000" in Japan: coral reefs. Biodiversity Center of Japan, Ministry of the Environment of Japan, Japan. (in Japanese)
Biodiversity Center of Japan (2008) The annual report of the "Monitoring Sites 1000" in Japan: coral reefs. Biodiversity Center of Japan, Ministry of the Environment of Japan, Japan. (in Japanese)
Biodiversity Center of Japan (2009) The annual report of the "Monitoring Sites 1000" in Japan: coral reefs. Biodiversity Center of Japan, Ministry of the Environment of Japan, Japan. (in Japanese)
Biodiversity Center of Japan (2010) The annual report of the "Monitoring Sites 1000" in Japan: coral reefs. Biodiversity Center of Japan, Ministry of the Environment of Japan, Japan. (in Japanese)
Biodiversity Center of Japan (2011) The annual report of the "Monitoring Sites 1000" in Japan: coral reefs. Biodiversity Center of Japan, Ministry of the Environment of Japan, Japan. (in Japanese)
Biodiversity Center of Japan (2012) The annual report of the "Monitoring Sites 1000" in Japan: coral reefs. Biodiversity Center of Japan, Ministry of the Environment of Japan, Japan. (in Japanese)
Biodiversity Center of Japan (2013) The annual report of the "Monitoring Sites 1000" in Japan: coral reefs. Biodiversity Center of Japan, Ministry of the Environment of Japan, Japan. (in Japanese)
Biodiversity Center of Japan (2014) The annual report of the "Monitoring Sites 1000" in Japan: coral reefs. Biodiversity Center of Japan, Ministry of the Environment of Japan, Japan. (in Japanese)
Borneman E (2001) Aquarium corals: selection, husbandry, and natural history. Microcosm Limited, Neptune City
Bourne DG, Garren M, Work TM et al (2009) Microbial disease and the coral holobiont. Trends Microbiol 17:554–562. https://doi.org/10.1016/j.tim.2009.09.004
Bourne DG, Muirhead A, Sato Y (2011) Changes in sulfate-reducing bacterial populations during the onset of black band disease. ISME J 5:559–564. https://doi.org/10.1038/ismej.2010.143
Bourne DG, Ainsworth TD, Pollock FJ, Willis BL (2014) Towards a better understanding of white syndromes and their causes on Indo-Pacific coral reefs. Coral Reefs 34:233–242. https://doi.org/10.1007/s00338-014-1239-x
Bruckner AW (2015) History of coral disease research. In: Woodley CM, Downs CA, Bruckner AW et al (eds) Diseases of coral. Wiley, Hoboken, pp 52–84
Bruno JF, Selig ER (2007) Regional decline of coral cover in the Indo-Pacific: timing, extent, and subregional comparisons. PLoS One 2:e711–e711. https://doi.org/10.1371/journal.pone.0000711
Carpenter KE, Abrar M, Aeby G et al (2008) One-third of reef-building corals face elevated extinction risk from climate change and local impacts. Science 321:560–563. https://doi.org/10.1126/science.1159196
Denner EBM, Smith GW, Busse HJ et al (2003) *Aurantimonas coralicida* gen. nov., sp. nov., the causative agent of white plague type II on Caribbean scleractinian corals. Int J Syst Evol Microbiol 53:1115–1122. https://doi.org/10.1099/ijs.0.02359-0
Dustan P (1977) Vitality of reef coral populations off key largo, Florida: recruitment and mortality. Environ Geol 2:51–58. https://doi.org/10.1007/BF02430665
Dustan P (1999) Coral reefs under stress: sources of mortality in the Florida keys. Nat Resour Forum 23:147–155
Eguchi M, Ishida Y (1990) Oligotrophic properties of heterotrophic bacteria and in situ heterotrophic activity in pelagic seawater. FEMS Microbiol Ecol 6:23–30
English S, Baker V (1994) Survey manual for tropical marine resources. Townsv Aust Inst Mar Sci 390:390–390
Ferguson RL, Buckley E, Palumbo A (1984) Response of marine bacterioplankton to differential filtration and confinement. Appl Environ Microbiol 47:49–55
Foley JE, Sokolow SH, Girvetz E et al (2005) Spatial epidemiology of Caribbean yellow band syndrome in *Montastrea* spp. coral in the Eastern Yucatan, Mexico. Hydrobiologia 548:33–40. https://doi.org/10.1007/s10750-005-3619-2
Garzón-Ferreira J, Gil-Agudelo DL, Barrios LM, Zea S (2001) Stony coral diseases observed in southwestern Caribbean reefs. In: Porter JW (ed) The ecology and etiology of newly emerging marine diseases. Springer, Netherlands, pp 65–69
Geiser DM, Taylor JW, Ritchie KB, Smith GW (1998) Cause of sea fan death in the West Indies. Nature 394:137–138. https://doi.org/10.1038/28079
Gladfelter WB (1982) White-band disease in *Acropora palmata*: implications for the structure and growth of shallow reefs. Bull Mar Sci 32:639–643
Gladfelter EH (1983) Circulation of fluids in the gastrovascular system of the reef coral *Acropora cervicornis*. Biol Bull 165:619–636. https://doi.org/10.2307/1541469
Glas MS, Sato Y, Ulstrup KE, Bourne DG (2012) Biogeochemical conditions determine virulence of black band disease in corals. ISME J 6:1526–1534. https://doi.org/10.1038/ismej.2012.2
Grimes DJ (2006) Koch's postulates-then and now. Microbe-Am Soc Microbiol 1:223
Harii S, Hongo C, Ishihara M et al (2014) Impacts of multiple disturbances on coral communities at Ishigaki Island, Okinawa, Japan, during a 15-year survey. Mar Ecol Prog Ser 509:171–180
Harvell CD, Kim K, Burkholder JM et al (1999) Emerging marine diseases – climate links and anthropogenic factors. Science 285:1505–1510. https://doi.org/10.1126/science.285.5433.1505
Harvell CD, Mitchell CE, Ward JR et al (2002) Climate warming and disease risks for terrestrial and marine biota. Science 296:2158–2162. https://doi.org/10.1126/science.1063699
Harvell D, Jordán-Dahlgren E, Merkel S et al (2007) Coral disease, environmental drivers, and the balance between coral and microbial associates. Oceanography 20:172–195
Hoegh-Guldberg O (2011) Coral reef ecosystems and anthropogenic climate change. Reg Environ Chang 11:215–227. https://doi.org/10.1007/s10113-010-0189-2
Holden C (1996) Coral disease hot spot in the Florida keys. Science:5295–5295
Hongo C, Yamano H (2013) Species-specific responses of corals to bleaching events on anthropogenically turbid reefs on Okinawa Island, Japan, over a 15-year period (1995–2009). PLoS One 8:e60952. https://doi.org/10.1371/journal.pone.0060952
Hughes TP, Baird AH, Bellwood DR et al (2003) Climate change, human impacts, and the resilience of coral reefs. Science 301:929–933. https://doi.org/10.1126/science.1085046
Irikawa A, Casareto BE, Suzuki Y et al (2011) Growth anomalies on *Acropora cytherea* corals. Mar Pollut Bull 62:1702–1707. https://doi.org/10.1016/j.marpolbul.2011.05.033
Jolles AE, Sullivan P, Alker AP, Harvell CD (2002) Disease transmission of aspergillosis in sea fans: inferring process from spatial pattern. Ecology 83:2373–2378. https://doi.org/10.1890/0012-9658(2002)083[2373:DTOAIS]2.0.CO;2
Kaczmarsky L, Richardson LL (2007) Transmission of growth anomalies between Indo-Pacific Porites corals. J Invertebr Pathol 94:218–221. https://doi.org/10.1016/j.jip.2006.11.007
Kayanne H, Harii S, Ide Y, Akimoto F (2002) Recovery of coral populations after the 1998 bleaching on Shiraho Reef, in the southern Ryukyus, NW Pacific. Mar Ecol Prog Ser 239:93–103

Koch R (1884) Die Aetiologie der Tuberkulose. Klin Wochenschr 2:1–88. https://doi.org/10.1007/BF01765224
Kogure K, Simidu U, Taga N (1980) Distribution of viable marine bacteria in neritic seawater around Japan. Can J Microbiol 26:318–323
Korrubel JL, Riegl B (1998) A new coral disease from the southern Arabian Gulf. Coral Reefs 17:22–22. https://doi.org/10.1007/s003380050088
Kushmaro A, Rosenberg E, Fine M, Loya Y (1997) Bleaching of the coral *Oculina patagonica* by Vibrio AK-1. Mar Ecol Prog Ser 147:159–165. https://doi.org/10.3354/meps147159
Kushmaro A, Banin E, Loya Y et al (2001) *Vibrio shiloi* sp. nov., the causative agent of bleaching of the coral *Oculina patagonica*. Int J Syst Evol Microbiol 51:1383–1388
Kuta K, Richardson L (1996) Abundance and distribution of black band disease on coral reefs in the northern Florida Keys. Coral Reefs:219–223
Lesser MP, Bythell JC, Gates RD et al (2007) Are infectious diseases really killing corals? Alternative interpretations of the experimental and ecological data. J Exp Mar Biol Ecol 346:36–44. https://doi.org/10.1016/j.jembe.2007.02.015
Lowenstam HA, Weiner S (1989) On biomineralization. Oxford University Press, Oxford, p 324
Loya Y, Sakai K, Nakano Y, Woesik RV (2001) Coral bleaching: the winners and the losers. Ecol Lett 4:122–131. https://doi.org/10.1046/j.1461-0248.2001.00203.x
Lozada-Misa P, Kerr A, Raymundo L (2015) Contrasting lesion dynamics of white syndrome among the scleractinian corals *Porites* spp. PLoS One 10:e0129841. https://doi.org/10.1371/journal.pone.0129841
Luna GM, Biavasco F, Danovaro R (2007) Bacteria associated with the rapid tissue necrosis of stony corals. Environ Microbiol 9:1851–1857. https://doi.org/10.1111/j.1462-2920.2007.01287.x
Matthew S, Salvador LA, Schupp PJ et al (2010) Cytotoxic halogenated macrolides and modified peptides from the apratoxin-producing marine cyanobacterium *Lyngbya bouillonii* from Guam. J Nat Prod 73:1544–1552. https://doi.org/10.1021/np1004032
Maynard J, van Hooidonk R, Eakin CM et al (2015) Projections of climate conditions that increase coral disease susceptibility and pathogen abundance and virulence. Nat Clim Chang. https://doi.org/10.1038/nclimate2625
Nishihira M (2004) Hermatypic corals of Japan. In: Tsuchiya M, Nadaoka K, Kayanne H, Yamano H (eds) Coral reefs of Japan. Ministry of the Environment and Japanese Coral Reef Society, Tokyo
Nomura K (2004) Appendix 3 spot check method for coral reef monitoring. In: Tsuchiya M, Nadaoka K, Kayanne H, Yamano H (eds) Coral reefs of Japan. Ministry of the Environment and Japanese Coral Reef Society, Tokyo
Okinawa Prefectural Government (2009) Report on the coral reef resources information preparation program. Nature Conservation Division, Department of Environmental Affairs, Okinawa Prefectural Government, Japan. (in Japanese)
Okinawa Prefectural Government (2010) Report on the coral reef resources information preparation program. Nature Conservation Division, Department of Environmental Affairs, Okinawa Prefectural Government, Japan. (in Japanese)
Okinawa Prefectural Government (2011) Report on the coral reef resources information preparation program. Nature Conservation Division, Department of Environmental Affairs, Okinawa Prefectural Government, Japan. (in Japanese)
Osborne K, Dolman AM, Burgess SC, Johns KA (2011) Disturbance and the dynamics of coral cover on the great barrier reef (1995–2009). PLoS One. https://doi.org/10.1371/journal.pone.0017516
Palmer CV, Mydlarz LD, Willis BL (2008) Evidence of an inflammatory-like response in non-normally pigmented tissues of two scleractinian corals. Proc Biol Sci 275:2687–2693. https://doi.org/10.1098/rspb.2008.0335
Patterson KL, Porter JW, Ritchie KB et al (2002) The etiology of white pox, a lethal disease of the Caribbean elkhorn coral, *Acropora palmata*. Proc Natl Acad Sci U S A 99:8725–8730. https://doi.org/10.1073/pnas.092260099
Peters EC, Gassman NJ, Firman JC et al (1997) Ecotoxicology of tropical marine ecosystems. Environ Toxicol Chem 16:12–40. https://doi.org/10.1002/etc.5620160103
Pollock FJ, Morris PJ, Willis BL, Bourne DG (2011) The urgent need for robust coral disease diagnostics. PLoS Pathog 7:e1002183. https://doi.org/10.1371/journal.ppat.1002183
Porter J, Dustan P, Jaap W, Patterson K (2001) Patterns of spread of coral disease in the Florida keys. Hydrobiologia:1–24
Ravindran J, Raghukumar C (2002) Pink line syndrome (PLS) in the scleractinian coral Porites lutea. Coral Reefs 21:252–252
Raymundo LJH, Harvell CD, Reynolds TL (2003) Porites ulcerative white spot disease: description, prevalence, and host range of a new coral disease affecting Indo-Pacific reefs. Dis Aquat Org 56:95–104. https://doi.org/10.3354/dao056095
Raymundo LJ, Couch CS, Bruckner AW, Harvell CD (2008) Coral disease handbook guidelines for assessment. Coral reef targeted research and capacity building for management program
Reeves L (1994) Newly discovered: yellow band disease strikes keys reefs. Underw USA 11:16
Richardson LL (2004) Black band disease. In: Rosenberg PE, Loya PY (eds) Coral health and disease. Springer Berlin, Heidelberg, pp 325–336
Richardson LL, Goldberg WM, Carlton RG, Halas JC (1998) Coral disease outbreak in the Florida Keys: Plague Type II. Rev Biol Trop 46:187–198
Richardson LL, Smith GW, Ritchie KB, Carlton RG (2001) Integrating microbiological, microsensor, molecular, and physiologic techniques in the study of coral disease pathogenesis. Methods 460:71–89. https://doi.org/10.2307/176940
Richardson LL, Sekar R, Myers JL et al (2007) The presence of the cyanobacterial toxin microcystin in black band disease of corals. FEMS Microbiol Lett 272:182–187. https://doi.org/10.1111/j.1574-6968.2007.00751.x
Ritchie KB, Smith GW (1998) Type II white-band disease. Rev Biol Trop 46:199–203
Santavy DL, Peters EC (1997) Microbial pests: coral disease research in the western Atlantic. In: 8th int. coral reef symp., pp. 607–601
Sato Y, Civiello M, Bell SC et al (2016) Integrated approach to understanding the onset and pathogenesis of black band disease in corals. Environ Microbiol 18:752–765. https://doi.org/10.1111/1462-2920.13122
Sussman M, Bourne DG, Willis BL (2006) A single cyanobacterial ribotype is associated with both red and black bands on diseased corals from Palau. Dis Aquat Org 69:111–118
Sussman M, Willis BL, Victor S, Bourne DG (2008) Coral pathogens identified for white syndrome (WS) epizootics in the indo-Pacific. PLoS One 3:e2393. https://doi.org/10.1371/journal.pone.0002393
Sutherland K, Porter J, Torres C (2004) Disease and immunity in Caribbean and Indo-Pacific zooxanthellate corals. Mar Ecol Prog Ser 266:273–302. https://doi.org/10.3354/meps266273
Sweet M, Jones R, Bythell J (2012) Coral diseases in aquaria and in nature. J Mar Biol Assoc U K 92:791–801
Thompson FL, Barash Y, Sawabe T et al (2006) Thalassomonas loyana sp. nov., a causative agent of the white plague-like disease of cor-

als on the Eilat coral reef. Int J Syst Evol Microbiol 56:365–368. https://doi.org/10.1099/ijs.0.63800-0

Wada M, Nishihara G, Isomura N (2012) Probing stony coral rapid tissue necrosis with optical oxygen sensors. In: 12th international coral reef symposium, Cairns, Queensland, Australia

Wada N, Shouhei I, Shinpei S et al (2015) Vibrio spp. isolated from scleractinian coral *Acropora muricata* with rapid tissue necrosis (RTN) and reared in aquarium. BioMicroWorld 2015, Barcelona

Wada N, Kawamoto T, Sato Y, Mano N (2016a) A novel application of a cryosectioning technique to undecalcified coral specimens. Mar Biol 163:117. https://doi.org/10.1007/s00227-016-2895-x

Wada N, Pollock FJ, Willis BL et al (2016b) In situ visualization of bacterial populations in coral tissues: pitfalls and solutions. PeerJ 4:e2424. https://doi.org/10.7717/peerj.2424

Wada N, Mano N, Yanagisawa Y, Mori T (in press) Occurrence of coral diseases in Akajima, Okinawa, Japan in 2010 and 2011. GALAXEA, Jpn Coral Reef Soc

Weil E, Irikawa A, Casareto B, Suzuki Y (2012) Extended geographic distribution of several Indo-Pacific coral reef diseases. Dis Aquat Org 98:163–170. https://doi.org/10.3354/dao02433

Wilkinson C (2008) Status of the coral reefs of the world: 2008. Global Coral Reef Monitoring Network, Townsville

Willis BL, Page CA, Dinsdale EA (2004) Coral disease on the great barrier reef. In: Rosenberg PE, Loya PY (eds) Coral health and disease. Springer Berlin, Heidelberg, pp 69–104

Woodley CM, Bruckner AW, McLenon AL et al (2008) Field manual for investigating coral disease outbreaks. National Oceanic and Atmospheric Administration, Silver Spring

Work T, Meteyer C (2014) To understand coral disease, look at coral cells. EcoHealth 11:610–618. https://doi.org/10.1007/s10393-014-0931-1

Yamashiro H (2014) Black band disease in Sesoko Island, Japan. The 3rd Asia-Pacific Coral Reef Symposium, Kenting, Pingtung, Taiwan

Yamashiro H, Fukuda M (2009) White spot syndrome of *Turbinaria peltata* in the temperate region of Japan. Coral Reefs 28:893–893. https://doi.org/10.1007/s00338-009-0514-8

Yamashiro H, Yamamoto M, van Woesik R (2000) Tumor formation on the coral *Montipora informis*. Dis Aquat Org 41:211–217. https://doi.org/10.3354/dao041211

Yamashiro H, Oku H, Onaga K et al (2001) Coral tumors store reduced level of lipids. J Exp Mar Biol Ecol 265:171–179. https://doi.org/10.1016/S0022-0981(01)00333-1

Yamashiro H, Isomura N, Sakai K (2014) Bloom of the cyanobacterium *Moorea bouillonii* on the gorgonian coral *Annella reticulata* in Japan. Sci Rep. https://doi.org/10.1038/srep06032

Yasuda N, Hidaka M (2012) Cellular kinetics in growth anomalies of the scleractinian corals Porites australiensis and *Montipora informis*. Dis Aquat Org 102:1–11. https://doi.org/10.3354/dao02530

Yasuda N, Nakano Y, Yamashiro H, Hidaka M (2012) Skeletal structure and progression of growth anomalies in *Porites australiensis* in Okinawa, Japan. Dis Aquat Org 97:237–247. https://doi.org/10.3354/dao02408

Zvuloni A, Artzy-Randrup Y, Stone L et al (2009) Spatio-temporal transmission patterns of black-band disease in a coral community. PLoS One 4:e4993–e4993. https://doi.org/10.1371/journal.pone.0004993