Journal of the Marine Biological Association of the United Kingdom, 2012, 92(4), 791–801.
doi:10.1017/S0025315411001688

Coral diseases in aquaria and in nature

MICHAEL SWEET[1], RACHEL JONES[2] AND JOHN BYTHELL[1]

[1]School of Biology, Ridley Building, University of Newcastle, Newcastle upon Tyne NE1 7RU, UK, [2]Zoological Society of London, Regent's Park, London, NW1 4RY

Many reef coral diseases have been described affecting corals in the wild, several of which have been associated with causal agents based on experimental inoculation and testing of Koch's postulates. In the aquarium industry, many coral diseases and pathologies are known from the grey literature but as yet these have not been systematically described and the relationship to known diseases in the wild is difficult to determine. There is therefore scope to aid the maintenance and husbandry of corals in aquaria by informing the field of the scientifically described wild diseases, if these can be reliably related. Conversely, since the main driver to identifying coral diseases in aquaria is to select an effective treatment, the lessons learnt by aquarists on which treatments work with particular syndromes provides invaluable evidence for determining the causal agents. Such treatments are not commonly sought by scientists working in the natural environment due the cost and potential environmental impacts of the treatments. Here we review both wild and aquarium diseases and attempt to relate the two. Many important aquarium diseases could not be reconciled to those in the wild. In one case, however, namely that of the ciliate Helicostoma *sp. as a causal agent of brown jelly syndrome in aquarium corals, there may be similarities with pathogenic agents of the wild coral diseases, such as white syndrome and brown band syndrome. We propose that* Helicostoma *is actually a misnomer, but improved understanding of this pathogen and others could benefit both fields. Improved practices in aquarium maintenance and husbandry would also benefit natural environments by reducing the scale of wild harvest and improving the potential for coral culture, both for the aquarium industry and for rehabilitation programmes.*

Keywords: coral, disease, aquarium, bacteria, ciliates

Submitted 7 January 2011; accepted 12 September 2011; first published online 1 November 2011

CORAL DISEASES—'WILD TYPES'

The study of coral diseases encompasses many different disciplines as it involves several aspects of complex host–pathogen interactions within the environment. Diseases and syndromes in corals have increased in number over recent years (Harvell 2007), however since the first observations of diseases affecting reef corals in the late 1970s and early 1980s (Ducklow & Mitchell, 1979; Peters *et al.*, 1983; Rutzler & Santavy, 1983; Antonius, 1985), the research priorities have changed substantially; from simple and general descriptions of disease signs in the field (Rutzler & Santavy, 1983) to microbial studies based on culture and/or non-culturable methods (Lesser *et al.*, 2007; Garren *et al.*, 2009; Kvennefors *et al.*, 2010). Since the early 1990s there has been increasing effort to characterize coral disease, including the application of novel molecular tools to confirm the identities of pathogens and apply Koch's postulates, thereby aiding in the understanding of the mechanisms of the host responses and resistance to particular diseases and pathogenic causal agents (Fredericks & Relman, 1996). Currently 18 coral diseases have been identified (Bourne *et al.*, 2009), yet only a few of these have been attributed to any particular causal agent (Kushmaro *et al.*, 2001; Ben-Haim & Rosenberg, 2002; Cooney *et al.*, 2002; Ben-Haim *et al.*, 2003a; Luna *et al.*, 2007; Sussman *et al.*, 2008), and in some cases the literature is confused with different authors ascribing different causal agents to the same disease (Luna *et al.*, 2007, 2010; Sussman *et al.*, 2008).

White band type II (Denner *et al.*, 2003), white pox (Patterson *et al.*, 2002; Lyndon, 2003; Sutherland & Ritchie, 2004), aspergillosis (Kirkwood *et al.*, 2010) and white plague type II (Richardson *et al.*, 1998; Denner *et al.*, 2003) are believed to be caused by known bacterial pathogens (Richardson *et al.*, 1998; Rosenberg & Ben-Haim, 2002; Weil *et al.*, 2006), and the seasonal bleaching of *Oculina patagonica* and *Pocillopora damicornis* has been proposed to be caused by *Vibrio shiloi* and *V. coralliilyticus* respectively (Rosenberg & Ben-Haim, 2002; Bourne & Munn, 2005), although this is disputed (Ainsworth *et al.*, 2008). Some diseases may be caused by a single agent, which can be amenable to investigation via Koch's postulates (Sussman *et al.*, 2008). However, others appear to be caused by a complex association of microbes. For example, black band disease, found throughout the Caribbean and the Indo-Pacific, appears to contain at least 50 different bacterial types within the disease lesion (Sekar *et al.*, 2006). The current lack of baseline data on coral–microbial associations of healthy corals (Sweet *et al.*, 2011a), coupled with the highly diverse microbial communities often associated with many coral diseases, makes a definitive comparison between coral diseases, often with similar disease signs, very difficult. Historically, the focus of coral disease research has primarily been on bacterial and fungal infections, whereas only recently have other microorganisms been studied. These include the infection of trematodes on *Porites* sp. (Aeby, 2002, 2003, 2007; Palmer *et al.*, 2009) and more frequently the numerous reports of ciliate-associated diseases both in the Indo-Pacific and the

Corresponding author:
M. Sweet
Email: m.j.sweet@newcastle.ac.uk

Caribbean (Croquer *et al.*, 2006a, b; Cooper *et al.*, 2007; Bourne *et al.*, 2008; Page & Willis, 2008; Qiu *et al.*, 2010).

With such a diversity of potential causal agents, and very little research that has located these potential agents to the specific sites and mechanisms of pathogenesis, it is extremely difficult to follow these pathogens through the environment to determine their reservoirs and modes of transmission. Several studies have detected proposed coral pathogens in healthy corals (Ritchie & Smith, 2004; Bourne & Munn, 2005; Klaus *et al.*, 2005; Ritchie, 2006; Sweet *et al.*, 2011b), suggesting that multiple factors in addition to pathogen exposure may be important to disease onset. Further complication arises since diseases are often identified by their symptoms alone leading to confusion over field identification of different disease lesions (Lindop *et al.*, 2008), making comparisons between studies difficult.

Ciliates and other protozoans have only recently been associated with diseases of corals. Skeleton-eroding band (SEB) was not only the first coral disease to be shown to be caused by a protozoan, but the first to be identified as being caused by a eukaryote (Antonius & Lipscomb, 2001). SEB is a progressive disease, widespread throughout the Indo-Pacific with high local prevalence (Page & Willis, 2008). The disease is characterized by a skeletal-eroding lesion with a speckled black band composed of the empty loricae (shell-like housings) of the folliculinid ciliate, *Halofolliculina corallasia* (Winkler *et al.*, 2004). More recently, another ciliate infection in the Indo-Pacific, brown band syndrome (BrB), has been described. BrB is widespread in parts of the Great Barrier Reef and known to effect three major coral families: Acroporidae, Pocilloporidae and Faviidae (Bourne *et al.*, 2008). This ciliate, identified as a member of the subclass Scuticociliatia (Bourne *et al.*, 2008) and only recently described as a novel species *Porpostoma guamensis* (Lobban *et al.*, 2011), has been shown to ingest intact symbiotic algae of the coral and is responsible for the visible symptoms of this disease (a variable brown band). Ciliates have also been shown to invade the tissues of corals after damage by predators, such as the feeding scars left by the crown-of-thorn's star fish *Acanthastar planci* (Nugues & Bak, 2009) and devour the tissues of coral spats (Cooper *et al.*, 2007). These findings further suggest that these organisms have an overall negative effect on coral population dynamics, by increasing post-settlement mortality. In contrast, other protozoans, identified as stramenopile protists, have been shown to be natural associates of corals, found both on the coral surface and within the tissues (Kramarsky-Winter *et al.*, 2006).

The first evidence of a coral–protozoan association in the Caribbean was reported in 2002, when a sequence matching with the phylum Apicomplexa was found in tissues of *Montastraea annularis* (Toller *et al.*, 2002). Despite this protozoan being related to coccidians, which are known to be highly virulent parasites, the nature of its interaction with corals remains largely unknown. In 2006, new evidence arose to show that ciliate infections were not exclusive to the Indo-Pacific. Folliculinid ciliates in the genus *Halofolliculina* were reported for the first time affecting over 26 Caribbean reef-building coral species (Croquer *et al.*, 2006b). Although it is still to be determined whether this Caribbean ciliate infection (CCI) is the same as SEB in the Indo-Pacific, their morphology, life cycle and patterns of infection are similar. In terms of pathology, both SEB and CCI have been shown to produce tissue mortality and in the particular case of CCI a negative effect on tissue regeneration (Page & Willis, 2008; Rodriguez *et al.*, 2009). Both diseases have been shown to transmit directly from infected to susceptible hosts (Page *et al.*, unpublished results) with injuries (Page & Willis, 2008; Rodriguez *et al.*, 2009) and temperature (Rodriguez *et al.*, 2009) enhancing transmission rates. Both SEB and CCI are widespread and occur across bioregions (Willis *et al.*, 2004; Winkler *et al.*, 2004; Croquer & Weil, 2009), affecting a wide range of coral hosts which is comparable to the most virulent of the bacterial diseases (Weil, 2004). Thus, increasing evidence indicates that ciliate infections are a significant problem for coral reef health, yet Koch's postulates have not been fulfilled for any of the ciliates associated with coral lesions, further complicating the problem because mixed ciliate communities have been reported thriving upon and/or underneath infected tissues.

A suite of coral pathologies, termed white syndrome (WS) in the Indo-Pacific and 'white' diseases (commonly, white plague and white band disease) are ecologically important and have caused widespread coral mortality. The white syndromes have been correlated with elevated temperature anomalies; however, there is conflicting evidence over the causal agents of these syndromes (Table 1). Despite the prevalence of these diseases/syndromes few types have been satisfactorily characterized (Bythell & Pantos, 2004; Lesser *et al.*, 2007). Despite this classification problem, many attempts have been made to link these diseases with a particular bacterial pathogen (Peters *et al.*, 1983; Barash *et al.*, 2005; Thompson *et al.*, 2006; Efrony *et al.*, 2007, 2009; Sussman *et al.*, 2008). For example, *Aurantimonas coralicida* has been reported to cause white plague Type II disease in the elliptical star coral *Dichocoenia stokesii* (Denner *et al.*, 2003). Another α-proteobacterium, thought to be the causative agent in juvenile oyster disease has been shown to be unique to colonies of the Caribbean coral *Montastrea annularis* exhibiting tissue lesions indicative of a white plague-like disease (Pantos *et al.*, 2003). Many of the most commonly cited bacterial pathogens associated with coral diseases belong to the genus *Vibrio*. Numerous *Vibrio* pathogens have also been associated with WS (Sussman *et al.*, 2008), with *Vibrio harveyi* being the most recent (Luna *et al.*, 2010). Despite the great effort, time and money spent trying to isolate specific pathogens and prove Koch's postulates discrepancies in the final disease outcome are common. Progressive tissue sloughing (tissue detaching from the coral skeleton) such as that described as the main disease sign in these white syndromes for example, has also been ascribed to similar diseases such as shut down reaction, rapid tissue necrosis and stress related necrosis (Borneman & Lowrie, 2001; Luna *et al.*, 2007, 2010; Efrony *et al.*, 2009). The main distinctions between these diseases/symptoms (Table 1) appear to be the rates of progression of the lesion, the species affected and regional separation (most notably those from the Caribbean and the Indo-Pacific). Currently, it is not known how these diseases are related and to date no specific pathogens have been found for these latter diseases.

CAUSATION AND CURE IN THE WILD

A few attempts have been made to cure coral diseases in the wild, notably the use of antibiotics, removal of the microbial

https://doi.org/10.1017/S0025315411001688 Published online by Cambridge University Press

Table 1. Showing the diverse array of coral diseases occurring throughout the world (C, Caribbean; IP, Indo-Pacific; M, Mediterranean; RS, Red Sea) and within aquarium (A), their various attributed names, proposed causal agents, rate of recorded tissue loss and band width (if any):† Kaczmarsky & Richardson (2007) show GA to be transferable and suggest microorganisms as potential causal agent do not always appear as 'white' patterning; * E. Peters (1983) noted the importance of these microorganisms but did not link them directly with the specific disease causation;[$] Luna *et al.* (2007, 2010) noted that *Vibrio* sp. failed to cause white syndromes (WS) in all cases, suggesting WS have multifactorial aetiology and/or a group of diseases caused by more than one pathogen.

Common names used for coral diseases/syndromes	Proposed causal agent (s)	Reference(s)	Location	Rate of tissue loss (cm/d)	Band width (cm)
White band disease Type I (WBDI)	Bacterial	Peters *et al.*, 1983	C, IP, RS	~0.9	~5–8
White b and disease Type II (WBDII)	*Vibrio charcharii*	Ritchie & Smith, 1995	C, IP, RS	~9	~5–8
White plague (WP)	Alpha-proteobacteria—JOD	Pantos *et al.*, 2003	C, IP, RS	~0.1	~0.2
White plague Type II (WPII)	*Sphingomonoas* sp. /*Aurantimonas corallicida*	Zorpette, 1995/Denner *et al.*, 2003/Richardson *et al.*, 1998	C	~1.4	~0.2
White plague Type III (WPIII)	*Sphingomonoas* sp. /*Aurantimonas corallicida*	Richardson *et al.*, 2002	C	~1–10	~0.2
White pox/patchy necrosis	Bacterial	Porter *et al.*, 2001/Patterson *et al.*, 2002	C	Fast	NA
Ring disease	Unknown	Weil, 2001	C	Unknown	NA
Finger coral denuding syndrome	Unknown	Williams & Bunkley-Williams, 2000	C	Unknown	NA
Star coral polyp necrosis	Unknown	Williams & Bunkley-Williams, 2000	C	Unknown	NA
Skeletal eroding band	*Holofolliculina corallasia*	Antonius, 1999/Page & Willis, 2008/Croquer *et al.*, 2006a,b	IP	Unknown	NA
Bacterial bleaching	*Vibrio shiloi*	Kushmaro *et al.*, 1996/Banin *et al.*, 2000	M	Unknown	NA
Bacterial lysis	*Vibrio coralyticus*	Ben-Haim & Rosenberg, 2002	IP	~1–2	NA
Ulcerative white spot disease	*Vibrio* sp.	Raymundo *et al.*, 2003	IP	Slow	3–5 mm round lesion
Growth anomalies (hyperplasia/ neoplasia/blisters)	Micro-organisms (at least in some cases)[†]	Loya *et al.*, 1984/Peters *et al.*, 1986/Kaczmarsky & Richardson, 2007	C, IP, RS	Slow	~1–20
Patchy necrosis	Unknown	Bruckner & Bruckner, 1997	C	~1–2 (1–10 cm/w)	~0.2
Coccidium infection/patchy necrosis	Apicomplexa–*Gemmocystis cylindrus*	Upton & Peters, 1986	C	Unknown	4 X 5
Rapid wasting disease	Fungal /parrotfish bites	Bruckner & Bruckner, 1998	C	Fast	NA
Stress related necrosis	Stress/microparasites (ciliates and amoebas)[*]	Peters *et al.*, 1983/Peters *et al.*, 1997	C, IP, RS	~0.9	~5 - 8
Shut down reaction	Unknown	Antonius, 1985	C, IP, RS	~240 (~10 cm/h)	~0.2
White syndrome	*Vibrio* sp.[$]	Luna *et al.*, 2010; Sussman *et al.*, 2008	IP, A	Unknown	NA
Rapid tissue necrosis/shut down reaction	Bacterial and stress/*Vibrio alginolyticus*/*V. harveyi*	Hormansdorfer *et al.*, 2000/Luna *et al.*, 2007	C, IP, RS, A	Fast	NA
Black band disease	Cyanobacteria	Conney *et al.*, 2002; Frais-Lopez *et al.*, 2003	C, IP	~0.1–2	NA
Brown band syndrome	Ciliate (Scuticociliatia)	Bourne *et al.*, 2008	IP	Fast	~1–2
Atramentous necrosis	Bacterial	Jones *et al.*, 2004	IP	Fast	Variable
Yellow band disease	Bacterial	Kimes *et al.*, 2010	C	~0.02	Variable
Brown jelly syndrome	Ciliate (Scuticociliatia)	Borneman, 2002	A	Fast	~1–5
Red slime algae	Cyanobacteria	Jones, personal communication	A	Unknown	NA
Slow tissue necrosis	Unknown	Luna *et al.*, 2010	A	Slow	NA
Flatworm infestations	Flatworms (e.g. *Amakusaplana acroporae*)	Haapkyla *et al.*, 2007; Rawlinson *et al.*, 2011	C, IP, A	NA	NA

mat associated with the lesion and shading of the infected area (Peters *et al.*, 1997; Griffin, 1998). Both yellow band disease (YBD) and white plague (Miller *et al.*, 2003) have been reported to be treatable in the field, involving an application of putty and clay over infected tissue (Riegl *et al.*, 2009). As an alternative to direct treatment of diseased corals, novel biological restoration approaches applied in Florida have been used to enhance recruitment of corals, reduce coral mortality and improve habitat quality. The most notable of these is the biological control and/or mitigation of spread of certain diseases by utilizing the reintroduction of *Diadema antillarum* as a tool to reduce macroalgal cover and induce increased settlement of coral larvae (Miller, personal communication). In response to BBD, Bruckner (1999) tried a variety of different techniques including: (1) the complete removal of the BBD lesion using a syringe, then covering with modelling clay or underwater putty; (2) shading of the BBD lesion using sun screens; and (3) addition of *D. antillarum* to cages containing the diseased corals. Brucker (1999), reported all treatments were successful but to varying degrees, with the highest success rate (>95%) being the use of underwater putty following removal of the microbial mat. This technique along with shading of infected colonies (Griffin, 1998) may be suitable when applied to small scale recovery programmes but remains impractical to treat large numbers of corals over large areas. Added to this, because BBD is believed to be transmitted in water, the removal of the band may liberate potential pathogens and aid in transmission to downstream corals. A recent review by Teplitski & Ritchie (2009), highlighted potential tools for coral disease management, the newest proposed method for curing coral disease *in situ* is the use of phage therapy. Bacterial viruses (bacteriophages or phages for short) have been used for treatment of human diseases with varied success and more recently within aquaculture for treating farmed fish diseases. The main advantages of phage therapy, particularly with corals, are potential host specificity and the fact that viruses self-replicate so any treatment would continue over large time scales with only minimal initial treatment doses, alongside the overall environmental safety of this particular type of treatment. The phage only attacks and destroys specific pathogens, leaving the beneficial bacteria within the coral holobiont unharmed. To date, phage therapy has only been considered for two main types of coral disease: tissue lysis of *Pocillopora damicornis* by *Vibrio coralliilyticus* (Ben-Haim & Rosenberg, 2002; Ben-Haim *et al.*, 2003b); and white plague-like disease affecting several large coral species including *Favia favus*, *Platygyra* sp. and *Goniastrea* sp. thought to be caused by *Thalosomonas loyaeana* (Barash *et al.*, 2005; Thompson *et al.*, 2006). Yet for phage therapy to work in the first place the causal agent must be known to be the sole causal agent and as this is in some debate for all coral diseases (Ainsworth *et al.*, 2008; Lesser *et al.*, 2007), these forms of treatment remain highly controversial. Despite this, Efrony *et al.* (2007) demonstrated that inoculation of specific phages at the same time as the proposed bacterial pathogens did inhibit infection in colonies within the aquarium environment. It was concluded that phage therapy may be a more valuable tool in preventing the spread of diseases, rather than curing infected corals. This was supported by the fact that phage therapy used in a control environment prevented the transmission of a disease from diseased corals to apparently healthy specimens (Efrony *et al.*, 2009). However as with antibiotic treatments, exposure to phages can often select for resistant bacterial mutants. A latter proposal which stemmed from phage therapy was suggested by Teplitski & Ritchie (2009), whereby coral transplants could be inoculated to give them resistance from at least some strains of bacterial pathogens. Seeding them with beneficial bacteria or phages may offer a degree of protection to these young recruits from potential pathogens and stressors (Teplitski & Ritchie, 2009).

In summary, despite the great research effort on wild coral diseases, the definitive causative agents, the (microscopic) co-location of suspected pathogens with sites of pathogenesis, factors contributing to their occurrence and transmission, and consequences on coral populations remain largely unknown or at least incomplete for most if not all coral diseases. As with all animals, diseases of corals are the result of an interaction between host, pathogenic agents and environment, each of which poses its own set of challenges and specific research needs. In addition, specific to coral diseases is the limitation that the pathological signs are limited, so that even experts in their field cannot reliably differentiate between diseases within the field (Lindop *et al.*, 2008), and comparisons between studies are therefore extremely difficult.

CORAL DISEASES—'AQUARIUM TYPES'

Although there is little work reported in the scientific literature on coral diseases in aquaria, a vast amount of 'grey literature' is available on the internet (Table 2). This undervalued information can be used to compile current knowledge of the most common diseases occurring within aquaria. A survey by Coral Zoo (Danovaro & Luna, 2008), reported that the two coral disease types occurring most frequently in aquaria were white syndromes (which comprise nearly 70% of the aquarium diseases on record) and brown jelly syndrome (BJS) (Figure 1). White syndromes (WS) are defined as severe tissue loss from the coral with a sharp demarcation between the apparently healthy tissue and the disease lesion and these signs appear to be equivalent to those reported for WS in the wild. BJS has currently not been reported in the wild

Table 2. Common diseases and syndromes found within aquarium corals and some of the suggested treatments.

Symptoms	Proposed casual agents	Most cited potential cures	Sources
Tissue loss, exposure of skeleton	Bacterial pathogens e.g. *Vibrio harveyi*	Fragmentation, isolation, sealing	Luna *et al.*, 2007; Advancedaqurist.com
Brown jelly like substance	Ciliate e.g. *Helicostoma* sp.	Chloramphenicol	Reefdreams.de
Red band/algal slime	Cyanobacterium	Red Slime Remover™	Aquatichouse.com
Black band	Cyanobacterium	Fragmentation, sealing	Wetwebmedia.com
Red bugs/yellow eggs	*Tegastes acroporanus*	*Milbemycin oxime*	Dorton, 2010; Orafarm.com
Small flatworms/yellow eggs	*Acropora* eating flatworm (AEF)	Salifert's flatworm exit	Reefkeeping.com

https://doi.org/10.1017/S0025315411001688 Published online by Cambridge University Press

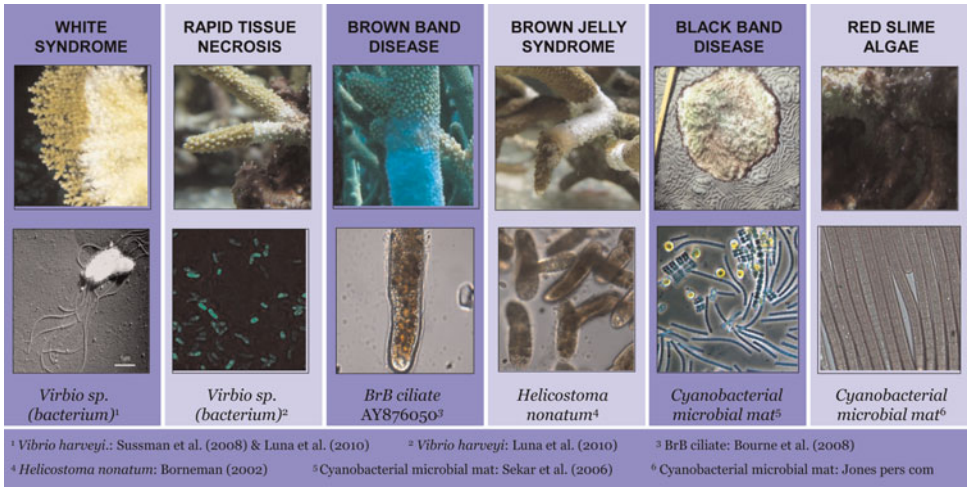

Fig. 1. Showing a selection of diseases affecting corals in the wild (dark blue panels) and within aquaria (light blue panels). Disease morphology is depicted in the top picture and the proposed causal agent is depicted below.

and is characterized by tissue death associated with widespread visible swelling and necrosis of tissues and mucus production (Borneman & Lowrie, 2001; Danovaro & Luna, 2008). WS in the aquarium, like those described in the field, characteristically display extremely variable rates of lesion progression, as well as patterns of tissue loss; which has led to numerous alternative descriptions and definitions, including rapid tissue necrosis (RTN) and shut down reaction (SDR). Coral death occurring within hours is usually referred to as SDR and is thought to be due to stress brought on by any number of factors (handling, temperature, salinity, pH, extreme changes in light or other water quality issues). The slightly slower process of tissue loss, occurring within days or weeks is often referred to as RTN and appears more similar to the wild WS. As a result, RTN is the term more commonly used, specifically for the occurrence of any tissue sloughing in captive corals within aquaria. Borneman (2002) suggested two main potential causes for RTN: (1) a specific pathological agent; and (2) a response to an external stress, such as physical damage, nutrient deficiencies, or temperature fluctuations, both resulting in autolysis and a general breakdown of the immune system. A more likely hypothesis is the combination of both, where the pathogenic agent, often found within healthy corals as well (e.g. within the surface mucus layer) may opportunistically become pathogenic during periods of stress when the coral's immune defences are impaired and therefore causes the disease symptoms such as that of RTN (Kushmaro *et al.*, 1996, 1998; Toren *et al.*, 1998; Ben-Haim *et al.*, 2003b). Despite the large number of cases documented in the grey literature only one study of aquarium diseases has been published in the scientific literature to date (Luna *et al.*, 2007). Luna *et al.* (2007) reported that RTN is readily transmissible from diseased corals to healthy specimens, which supports the hypothesis that pathogenic microorganisms are involved. Luna *et al.* (2007), demonstrate an increase in total *Vibrio* abundance (Figure 1; Table 1) within diseased specimens compared to those of healthy ones. In particular, one known bacterial pathogen, *V. harveyi*, was ascribed as being the main causative agent for RTN in their study (Luna *et al.*, 2007).

Within the aquarium trade, the second most common disease type, BJS, has been associated with a suspected ciliate pathogen, *Helicostoma nonatum* (Figure 1) (Borneman & Lowrie, 2001). Willis *et al.* (2004) first speculated that the ciliate associated with BrB disease was similar to this species or at least a close relative, however they later identified the protozoan associated with BrB as being more closely related to a different species of the class Oligohymenophora, subclass Scuticociliatia (Bourne *et al.*, 2008). There are only a few descriptions of ciliates from the genus *Helicostoma* (Rama Mohan Rao *et al.*, 1980, 1981), and very few refer to the species *H. nonatum* (Kahl, 1931; Purdom & Howard, 1971); although, it is sometimes referred to as '*H. notatum*' (Carey & Carey, 1992). However, there are no gene sequences related to this species on GenBank, so it is impossible to reconcile this ciliate with the one described in BrB by Bourne *et al.* (2008). Other *Helicostomas* such as *H. brudderbucki* and *H. oblongum* are sometimes referred to in the grey literature as the BJS ciliate, but neither fit into the morphological characterization of the BJS ciliate. Further confusion occurs as the taxonomy of this species relates to that of *Porpostoma natatum* (Kahan *et al.*, 1982; Kuhlmann *et al.*, 1996; Song, 2000), recorded in the Australian Antarctica data centre as synonymous with *H. nonatum. Paraturbanella stradbroki* (Hochberg, 2002) has also been cited as being the proper epithet for *Helicostoma nonatum* (Hummon, 2008), having been renamed in 1927, and which has assigned gene sequences in GenBank. This species also appears in databases such as the UNESCO–IOC Register of Marine Organisms, Integrated Taxonomic Information System (ITIS) and World Register of Marine Species (WORMS). To further complicate this issue, recent molecular (sequences submitted to GenBank) and taxonomic research has highlighted the need for restructuring of this particular subclass of ciliates (Scuticociliatia), due to the improvement of molecular characterization and the ability to acquire complete 18S rRNA gene sequences of single cell isolates. Based on a newly submitted sequence to GenBank and the subsequent paper by Zhang *et al.* (2011) we suggest the proper name for the BJS ciliate is a *Philaster* sp. closely related to the newly described species *P. digitiformis* (Zhang *et al.*, 2011). However, further research is necessary to fully understand this.

Despite the confusion in nomenclature, while ciliates are undisputedly present in the brown jelly material associated

https://doi.org/10.1017/S0025315411001688 Published online by Cambridge University Press

with BJS; it remains unclear as to what exact role they play in the disease pathology (i.e. are they the primary or secondary causal agents?), this remains also the case for the wild type disease, BrB. It is feasible they are only present because they are feeding on dead tissue arising from another pathogen or non-pathogenic disease (Borneman & Lowrie, 2001). In support of this, the Zoological Society of London (ZSL) has often but not always isolated *Vibrio* spp. particularly *V. vulnificus* from corals such as *Goniopora* and *Euphyllia* exhibiting disease signs similar to BJS (authors, personal observations), however further work needs to be conducted. Nevertheless, although, the disease signs of BrB and BJS are distinct and ciliates with similar morphologies appear to be involved in both diseases, at least at some level, it remains difficult to conclude if these are two separate syndromes or different visual pathological signs of the same disease caused by the same agent or agents.

Although WS and BJS are the more common diseases in aquaria, many other types of syndromes are often reported, for example, infestations from organisms such as the red bug *Tegastes acroporanus* is often referred to as a disease/syndrome in most of the grey literature. *Tegastes acroporanus* is a predatory micro-crustacean which is specific to acroporids, they are small, ~0.5 mm in length and yellow in colour with a distinctive red dot which gives this species its common name. Poor polyp extension, loss of coloration and overall decline in health have been reported as signs of a red bug infection. Infestations of *T. acroporanus* have not been recorded in the wild to date, although they would be easily overlooked due to their size. Another common infestation of aquarium corals is numerous flatworm species. The reported 'brown rust disease' is attributed to flatworms such as *Convolutriloba* sp. and *Waminoa* sp. (Shannon & Achatz, 2007). *Waminoa* sp. tend to be commensal organisms that live only on corals, while *Convolutriloba* sp. will usually live and grow on any available surface. Despite not being known to cause damage to the corals directly, heavy infestations are reported that can cause disruption to photosynthesis and therefore degrade overall health, particularly in corals such as *Euphyllia* sp. (authors, personal observations). The heavy infestations associated with brown rust have again not been reported in the wild to date. The *Acropora*-eating flatworms (AEF), recently named as *Amakusaplana acroporae* (Rawlinson *et al.*, 2011), however, are a common pathogen of aquarium *Acropora* and have also been described in the wild (Barneah *et al.*, 2007; Haapkylä *et al.*, 2007, 2009). They are usually extremely well-camouflaged and often the only visible signs would be the feeding scars left behind, exposing the coral skeleton, so the extent of this disease may have been under-reported in the wild and disease lesions reported as other syndromes (potentially classed in the WS group). Another common disease within aquaria is known as 'red slime algae'. This disease is most commonly associated with high levels of organic nutrients within the aquarium, which in turn may be influenced by changes in light levels. Despite the common name of this disease, the causal agent is not actually an alga at all, but a consortium of cyanobacteria. Varying from red, black to blue-green, the specific causal agent or agents remain unknown, however there are strong similarities between this disease morphology and that of the cyanobacterial mat of BBD within wild coral populations, and cyanobacterial overgrowth is also commonly reported in the wild, particularly in the Caribbean (Weil, 2004; Weil & Croquer, 2009).

CAUSATION AND CURE IN AQUARIA

Since the main driver to identifying coral diseases in aquaria is the selection of an effective treatment, the lessons learnt by aquarists over what treatments are effective against particular syndromes can provide invaluable evidence for determining the causal agents of these diseases (Table 2). Such treatments are generally not sought by scientists working in the natural environment, due to the cost and potential environmental impacts of the treatments, however the potential to develop, adapt and treat corals in the wild is an important objective. With slight modifications of these proposed cures, coral disease in the wild could potentially even be managed, maintained and/or localized. If a disease can be effectively treated, this can be used as further proof of the causal agent or agents. When corals are transported to aquaria from the wild (about 2 million coral pieces are currently transported legally per year for such uses; Green & Hendry, 1999; Wabnitz *et al.*, 2003), a significant change in the environmental conditions occurs. Thus considerable physiological stress is placed on the corals, from collection, transportation (e.g. transit times from Indonesia are long, with many stages and high potential for delays) and resettlement within aquaria, and it is therefore understandable that large percentages of those collected never reach their destinations and when they do, disease and death is common. In species such as *Catalyphyllia jardinei* and *Goniopora stokesi* mortality rates often approach >80%. In general the most popular species, those of the large single polyp varieties, are vulnerable to physical damage during transport and the onset of rapidly progressing diseases/infections are often seen in these corals. Effective treatments of known diseases and syndromes are therefore important to promote better survival and ultimately minimize the necessity to collect more from natural reefs.

Numerous suggestions have been presented by hobbyists for the cure of common aquarium diseases (Table 2), for example, the use of a broad-spectrum antibiotic, chloramphenicol (Tifomycine:flexyx.com, only available in USA) appears the most common to treat corals suffering from BJS. Chloramphenicol is a bacteriostatic antimicrobial, active against both Gram-positive and Gram-negative bacteria and is extremely lipid-soluble for fast effective treatment (Reefdreams, 2010). Doxycycline, oxytetracycline, iodine and freshwater dips (15 ppt) have also been reportedly used to treat BJS with varying levels of success (authors, personal observations). Yet treatment with antibiotics has a variety of significant limitations: (1) they are difficult to administer to the infected individual; (2) antibiotics are often light sensitive and have a short half life therefore requiring a number of repeat treatments; (3) a requirement of no filtration during treatment which in turn causes other water quality issues and the potential for other diseases to occur; (4) they will undoubtedly be harmful to beneficial bacteria within the coral holobiont and the surrounding water; and more often than not (5) by the time the decision is made to treat the disease the progression is so fast that it is too late to be useful. Treatments for diseases such as RTN which encompasses the 'white syndromes' discussed earlier are in much greater debate, primarily due to the causal agent being largely unknown. Fragmentation, isolation, sealing the lesion with epoxy resin and the use of a variety of antibiotics such as doxycycline have all been prescribed as possible treatments (Borneman & Lowrie, 2001; Advancedaquarist, 2002;

https://doi.org/10.1017/S0025315411001688 Published online by Cambridge University Press

Borneman, 2002; Leewis *et al.*, 2009), although no systematic assessment of their effectiveness has been conducted.

A treatment of red bug disease caused by *T. acroporanus*, first developed by Dorton (2010), is the use of *Milbemycin oxime*, an active ingredient in heart worm medication for dogs called Interceptor® within the USA or in a product called Milbemax® in the UK (where *M. oxime* is mixed with praziquantal). This is an indiscriminate drug which kills all crustaceans as well as *T. acroporanus*, so would not be a feasible treatment of wild diseases. An attractive option to this treatment is the introduction of a biological control, the dragonface pipefish, *Corythoichthys haematopterus* for example. These fish are known to anchor themselves to acroporids and feed on crustaceans including *T. acroporanus*. It is also common that secondary infections often follow, initiating from the feeding scars caused by *T. acroporanus*, along with the scars left by nudibranchs, *Drupella* and/or other corallivorous snails.

For the treatment of flatworm infections including AEF, Salifert's flatworm exit, levamisole hydrochloride, freshwater dips (15 ppt for 3 m maximum) and iodine-based dips like Lugol's iodine, Fluke-Tabs™ (Aquarium Products), and Trichlorfon™ (Dylox 80, Bayer A.G.) have all been reported for treatment of infected corals (Carl 2008; Nosratpour, 2008). However, it is important to note that smaller polyp species such as the acroporids can rarely tolerate the use of freshwater dips and often mortality occurs soon after treatment.

Red slime algae is reported to be treatable by commercial products such as Ultralife Red Slime Remover™, Boyd Chemi-Clean™, and Blue Life Red Slime Control™ (Brang, 2010). However, many of these diseases are reported as a sign of poor water quality, so most aquarists propose reassessment and improvement of aquarium water quality (reducing levels of nitrate and phosphate and monitoring light levels and improving flow) as the most effective treatment. There is also a syndrome known in the aquarium trade as 'new tank syndrome', which often manifests itself as blooms of algae/cyanobacteria (e.g. red slime algae). This is thought to be caused by the new silicone within the tank setup increasing nutrient levels which promotes the growth of the algae.

SIMILARITIES BETWEEN WILD TYPES AND THOSE WITHIN THE AQUARIUM

The differences between the natural reef environment and that within the aquarium are obviously great. Despite this, certain similarities can be seen between the diseases found in both cases. Most notable would be the 'white syndromes'. As these diseases include many forms of tissue necrosis exposing the skeleton, it is impossible to confirm whether the same diseases occur without any reasonable doubt, however the visible signs are clearly very similar (Figure 1). Only the progression rate of the lesion is thought to distinguish these particular syndromes from each other (Table 1). In addition, there are significant similarities between the proposed causal agent of one form of white syndrome (ciliated white syndrome; authors, personal observations), and that thought to be the causal agent of BJS (Figure 1), the ciliate in question also being from the same genus as the proposed causal agent in BrB (Figure 1). The differences between these diseases may potentially be explained by the conditions in aquaria compared to the reef. In the natural environment wave action is likely to remove the 'brown jelly like' necrotic tissue associated with BJS in aquaria, revealing a 'white syndrome' type disease lesion. Similarly, black band disease (Figure 1) in the wild is caused by a similar association of cyanobacteria to that of red slime algae (Figure 1); however, the visible disease lesion is also dramatically different. Whilst infestations by other microorganisms like *T. acroporanus* and certain flatworm species appear to be unique to aquaria with no apparent cases in the wild, this may be due to at least in part that these diseases are currently being overlooked within the wild.

CONCLUSIONS AND MAJOR KNOWLEDGE GAPS

In general, many important aquarium diseases cannot be reconciled to those in the wild. In certain cases however, strong similarities can be seen. For example, the ciliate *Helicostoma* sp. as a causal agent of BJS in aquarium corals shows strong similarities with the ciliates associated with BrB in the wild. It is proposed that *Helicostoma* may be a misnomer and this ciliate may be the same as that identified in BrB. Improved understanding of this pathogen and other pathogens of these common aquarium diseases could benefit both fields. Improved practices in aquarium maintenance and husbandry would also benefit natural environments by reducing the scale of wild harvest and improving the potential for coral culture, both for the aquarium industry and for rehabilitation programmes. It is noted that in general, management of diseases within the marine environment and corals in particular is challenging due to the difficulties in controlling habitat and population dynamics and the potentially rapid rates of spread (Bourne *et al.*, 2009). However, without understanding the interactions between causative agents, corals and their environments, management of these diseases in the field will be near impossible. One potential step forward would be trials and adaptations of the cures used within the aquarium trade to those diseases which may be similar in the wild.

FURTHER WORK

(1) A complete study on aquarium diseases and their causal agents needs to be carried out using microbiological techniques, with attempts made to prove Koch's postulates for the proposed causal agents; and
(2) treatment trials to systematically test all treatments proposed for specific diseases (particularly RTN and BJS), within the grey literature and attempts to improve those which work.

REFERENCES

Advancedaquarist (2002) *Coral whisperer*. http://www.advancedaquarist.com/issues/june2002/cwhtm

Aeby G.S. (2002) Trade-offs for the butterflyfish, *Chaetodon multicinctus*, when feeding on coral prey infected with trematode metacercariae. *Behavioral Ecology and Sociobiology* 52, 158–165.

Aeby G.S. (2003) Corals in the genus *Porites* are susceptible to infection by a larval trematode. *Coral Reefs* 22, 216–216.

https://doi.org/10.1017/S0025315411001688 Published online by Cambridge University Press

Aeby G.S. (2007) Spatial and temporal patterns of *Porites trematodiasis* on the reefs of Kaneohe Bay, Oahu, Hawaii. *Bulletin of Marine Science* 80, 209–218.

Ainsworth T., Fine M., Roff G. and Hoegh-Guldberg O. (2008) Bacteria are not the primary cause of bleaching in the Mediterranean coral *Oculina patagonica*. *ISME Journal* 2, 67–73.

Antonius A. (1985) Coral diseases in the Indo-Pacific: a first record. *Marine Ecology* 6, 197–218.

Antonius A. (1999) *Halofolliculina corallasia*, a new coral-killing ciliate on Indo-Pacific reefs. *Coral Reefs* 18, 300–300.

Antonius A. and Lipscomb D. (2001) First protozoan coral-killer identified in the Indo-Pacific. *Atoll Research Bulletin* 481, 1–21.

Banin E., Israely T., Kushmaro A., Loya Y., Orr E. and Rosenberg E. (2000) Penetration of the coral-bleaching bacterium *Vibrio shiloi* into *Oculina patagonica*. *Applied and Environmental Microbiology* 66, 3031–3036.

Barash Y., Sulam R., Loya Y. and Rosenberg E. (2005) Bacterial strain BA-3 and a filterable factor cause a white plague-like disease in corals from the Eilat coral reef. *Aquatic Microbial Ecology* 40, 183–189.

Barneah O., Ben-Dov E., Kramarsky-Winter E. and Kushmaro A. (2007) Characterization of black band disease in Red Sea stony corals. *Environmental Microbiology* 9, 1995–2006.

Ben-Haim Y. and Rosenberg E. (2002) A novel *Vibrio* sp. pathogen of the coral *Pocillopora damicornis*. *Marine Biology* 141, 47–55.

Ben-Haim Y., Thompson F.L., Thompson C.C., Cnockaert M.C., Hoste B., Swings J. and Rosenberg E. (2003a) V*ibrio coralliilyticus* sp. nov., a temperature-dependent pathogen of the coral *Pocillopora damicornis*. *International Journal of Systematic and Evolutionary Microbiology* 53, 309–315.

Ben-Haim Y., Zicherman-Keren M. and Rosenberg E. (2003b) Temperature-regulated bleaching and lysis of the coral *Pocillopora damicornis* by the novel pathogen *Vibrio coralliilyticus*. *Applied and Environmental Microbiology* 69, 4236–4242.

Borneman E.H. (2002) *Bacterial infections: a response to recent 'reef notes' columns*. http://www.reefkeeping.com *Reefkeeping* 1(4)

Borneman E.H. and Lowrie J. (2001) Advances in captive husbandry and propagation: an easily utilized reef replenishment means from the private sector? *Bulletin of Marine Science* 69, 897–913.

Bourne D.G., Boyett H.V., Henderson M.E., Muirhead A. and Willis B.L. (2008) Identification of a ciliate (Oligohymenophorea: Scuticociliatia) associated with brown band disease on corals of the Great Barrier Reef. *Applied and Environmental Microbiology* 74, 883–888.

Bourne D.G., Garren M., Work T.M., Rosenberg E., Smith G.W. and Harvell C.D. (2009) Microbial disease and the coral holobiont. *Trends in Microbiology* 17, 554–562.

Bourne D.G. and Munn C.B. (2005) Diversity of bacteria associated with the coral *Pocillopora damicornis* from the Great Barrier Reef. *Environmental Microbiology* 7, 1162–1174.

Brang S. (2010) *How to get rid of reef aquarium pests*. http://kbmarinedepot.com/articleaspx?id=10984

Bruckner A.W. (1999) *Block-band disease (BBD) of scleractinian corals: occurrence, impacts and mitigation*. PhD thesis. University of Puerto Rico, Mayaguez, 286 pp.

Bruckner A.W. and Bruckner R.J. (1997) Outbreak of coral disease in Puerto Rico. *Coral Reefs* 16, 260–260.

Bruckner A. and Bruckner R. (1998) Rapid-wasting disease: pathogen or predator? *Science* 279, 2023–2025.

Bythell J.C. and Pantos O. (2004) *Establishing methods for microbial community structure analysis in bleaching corals*. IOC-GEF/WB Targeted Working Group on Coral Bleaching, Final Report.

Carey P.G. and Carey P.G. (1992) *Marine interstitial ciliates. An illustrated key*. London: Chapman and Hall, 351 pp.

Carl M. (2008) Predators and pests of captive corals. In Leewis R.J. and Janse M. (eds) *Advances in coral husbandry in public aquariums. Public aquarium husbandry series. Volume 2*. Arnhem, The Netherlands: Burgers' Zoo, pp. 31–36.

Cooney R.P., Pantos O., Le Tissier M.D.A., Barer M.R., O'Donnell A.G. and Bythell J.C. (2002) Characterization of the bacterial consortium associated with black band disease in coral using molecular microbiological techniques. *Environmental Microbiology* 4, 401–413.

Cooper W., Lirman D. and Schmale M. (2007) Consumption of coral spat by histophagic ciliates. *Coral Reefs* 26, 249–250.

Croquer A., Bastidas C. and Lipscomb D. (2006a) Folliculinid ciliates: a new threat to Caribbean corals? *Diseases of Aquatic Organisms* 69, 75–78.

Croquer A., Bastidas C., Lipscomp D., Rodriguez-Martinez R.E., Jordan-Dahlgren E. and Guzman H.M. (2006b) First report of folliculinid ciliates affecting Caribbean scleractinian corals. *Coral Reefs* 25, 187–191.

Croquer A. and Weil E. (2009) Spatial variability in distribution and prevalence of Caribbean scleractinian coral and octocoral diseases. II. Genera-level analysis. *Diseases of Aquatic Organisms* 83, 209–222.

Danovaro D. and Luna G. (2008) *Italian Consortium for Marine Sciences (CoNISMa): Research Unit—Coral Zoo*. http://www.coralzoo.org/home/

Denner E.B.M., Smith G.W., Busse H.J., Schumann P., Narzt T., Polson S.W., Lubitz W. and Richardson L.L. (2003) *Aurantimonas coralicida* gen. nov., sp. nov., the causative agent of white plague type II on Caribbean scleractinian corals. *International Journal of Systematic and Evolutionary Microbiology* 53, 1115–1122.

Dorton D. (2010) *Oceans, reefs and aquariums*. http://www.reefsanctuary.com/forums/general-reef-aquarium-discussion/27493-red-bughtml and http://www.orafarm.com

Ducklow H.W. and Mitchell R. (1979) Observations on naturally and artificially diseased tropical corals—scanning electron-microscope study. *Microbial Ecology* 5, 215–223.

Efrony R., Atad I. and Rosenberg E. (2009) Phage therapy of coral white plague disease: properties of phage BA3. *Current Microbiology* 58, 139–145.

Efrony R., Loya Y., Bacharach E. and Rosenberg E. (2007) Phage therapy of coral disease. *Coral Reefs* 26, 7–13.

Frias-Lopez J., Bonheyo G.T., Jin Q.S. and Fouke B.W. (2003) Cyanobacteria associated with coral black band disease in Caribbean and Indo-Pacific Reefs. *Applied and Environmental Microbiology* 69, 2409–2413.

Fredericks D.N. and Relman D.A. (1996) Sequence-based identification of microbial pathogens: a reconsideration of Koch's postulates. *Clinical Microbiology Reviews* 9, 18–33.

Garren M., Raymundo L., Guest J., Harvell C.D. and Azam F. (2009) Resilience of coral-associated bacterial communities exposed to fish farm effluent. *PLoS ONE* 4.

Green E.P. and Hendry H. (1999) Is CITES an effective tool for monitoring trade in corals? *Coral Reefs* 18, 403–407.

Griffin S.P. (1998) The effects of sunlight on the progression of black band disease. *Revista De Biologia Tropical* 46, 175–179.

Haapkylä J., Seymour A.S., Trebilco J. and Smith D. (2007) Coral disease prevalence and coral health in the Wakatobi Marine Park, south-east Sulawesi, Indonesia. *Journal of the Marine Biological Association of the United Kingdom* 87, 403–414.

Haapkylä J., Unsworth R.K.F., Seymour A.S., Melbourne-Thomas J., Flovell M., Willis B.L. and Smith D.J. (2009) Spatio-temporal coral disease dynamics in the Wakatobi Marine National Park, south-east Sulawesi, Indonesia. *Diseases of Aquatic Organisms* 87, 105–115.

Harvell D. (2007) Coral disease, environmental drivers and the balance between coral and microbial associates. *Oceanography* 20, 172–195.

Hochberg R. (2002) Two new species of *Pseudostomella* (Gastrotricha: Macrodasyida) with scaled triancres from the east coast of Australia. *New Zealand Journal of Marine and Freshwater Research* 36, 571–579.

Hormansdorfer S., Wentges H., Neugebaur-Buchler K. and Bauer J. (2000) Isolation of *Vibrio alginolyticus* from seawater aquaria. *International Journal of Hygiene and Environmental Health* 203, 169–175.

Hummon W. (2008) *MEIOFAUNA MARINA, Biodiversity, morphology and ecology of small benthic organisms. Volume 16*. Department of Biological Sciences, Ohio University, USA.

Kaczmarsky L. and Richardson L.L. (2007) Transmission of growth anomalies between Indo-Pacific *Porites* corals. *Journal of Invertebrate Pathology* 94, 218–221.

Kahan D., Wilbert N., Vit G. and Barel T. (1982) Feeding-behaviour of *Porpostoma notatum* (Ciliata). *Journal of Protozoology* 29, 535–535.

Kahl A. (1931) Urtiere oder Protozoa. 1. Wimpertiere order Ciliata (Infusoria). 2. Holotricha. *Dahl Tierwelt Deutschlands Jena Teil* 21, 181–398.

Kimes N.E., Van Nostrand J.D., Weil E., Zhou J.Z. and Morris P.J. (2010) Microbial functional structure of *Montastraea faveolata*, an important Caribbean reef-building coral, differs between healthy and yellow-band diseased colonies. *Environmental Microbiology* 12, 541–556.

Kirkwood M., Todd J.D., Rypien K.L. and Johnston A.W.B. (2010) The opportunistic coral pathogen *Aspergillus sydowii* contains dddP and makes dimethyl sulfide from dimethylsulfoniopropionate. *ISME Journal* 4, 147–150.

Klaus J.S., Frias-Lopez J., Bonheyo G.T., Heikoop J.M. and Fouke B.W. (2005) Bacterial communities inhabiting the healthy tissues of two Caribbean reef corals: interspecific and spatial variation. *Coral Reefs* 24, 129–137.

Kramarsky-Winter E., Harel M., Siboni N., Ben Dov E., Brickner I., Loya Y. and Kushmaro A. (2006) Identification of a protist–coral association and its possible ecological role. *Marine Ecology Progress Series* 317, 67–73.

Kuhlmann H-W., Braeucker R. and Schepers A.G. (1996) *Porpostoma notatum*, a marine scuticociliate with a conspicuous organelle, shows phototactic orientation. *Journal of Eukaryotic Microbiology* 43, 15A.

Kushmaro A., Banin E., Loya Y., Stackebradt E. and Rosenberg E. (2001) *Vibrio shiloi* sp. nov., the causative agent of bleaching of the coral *Oculina patagonica*. *International Journal of Systematic and Evolutionary Microbiology* 51, 1383–1388.

Kushmaro A., Loya Y., Fine M. and Rosenberg E. (1996) Bacterial infection and coral bleaching. *Nature* 380, 396.

Kushmaro A., Rosenberg E., Fine M., Ben-Haim Y. and Loya Y. (1998) Effect of temperature on bleaching of the coral *Oculina patagonica* by *Vibrio* AK-1. *Marine Ecology Progress Series* 171, 131–137.

Kvennefors E.C.E., Sampayo E., Ridgway T., Barnes A.C. and Hoegh-Guldberg O. (2010) Bacterial communities of two ubiquitous Great Barrier Reef corals reveals both site- and species-specificity of common bacterial associates. *PLoS ONE* 5, e10401.

Leewis R., Wijgerde T., Laterveer M. and Osinga R. (eds) (2009) *Working with aquarium corals. A book of protocols for the breeding and husbandry of scleractinian corals*. (available at www.coralzoo.org).

Lesser M.P., Bythell J.C., Gates R.D., Johnstone R.W. and Hoegh-Guldberg O. (2007) Are infectious diseases really killing corals? Alternative interpretations of the experimental and ecological data. *Journal of Experimental Marine Biology and Ecology* 346, 36–44.

Lindop A.M.M., Hind E.J. and Bythell J.C. (2008) The unknowns in coral disease identification: an experiment to assess consensus of opinion amongst experts. *Proceedings of the 11th International Coral Reef Symposium, Ft Lauderdale, Florida, 7–11 July 2008 Session number 7*.

Lobban C.S., Raymundo L. and Montagnes D.J.S. (2011) *Porpostoma guamensis* n. sp., a philasterine scuticociliate associated with brown-band disease of corals. *Journal of Eukaryotic Microbiology* 58, 103–113.

Loya Y., Bull G. and Pichon M. (1984) Tumor formations in scleractinian corals. *Helgoländer Meeresuntersuchungen* 37, 99–112.

Luna G.M., Biavasco F. and Danovaro R. (2007) Bacteria associated with the rapid tissue necrosis of stony corals. *Environmental Microbiology* 9, 1851–1857.

Luna G.M., Bongiorni L., Gili C., Biavasco F. and Danovaro R. (2010) *Vibrio harveyi* as a causative agent of the white syndrome in tropical stony corals. *Environmental Microbiology Reports* 2, 120–127.

Lyndon A.R. (2003) Human gut bug implicated in coral 'white pox'. *Marine Pollution Bulletin* 46, 156–156.

Miller J., Rogers C. and Waara R. (2003) Monitoring the coral disease, plague type II, on coral reefs in St. John, US Virgin Islands. *Revista De Biologia Tropical* 51, 47–55.

Nosratpour F. (2008) Observations of a polyclad flatworm affecting acroporid corals in captivity. In Leewis R.J. and Janse M. (eds) *Advances in coral husbandry in public aquariums. Public aquarium husbandry series. Volume 2*. Arnhem, The Netherlands: Burgers' Zoo, pp. 37–46.

Nugues M.M. and Bak R.P.M. (2009) Brown-band syndrome on feeding scars of the crown-of-thorn starfish *Acanthaster planci*. *Coral Reefs* 28, 507–510.

Page C.A. and Willis B.L. (2008) Epidemiology of skeletal eroding band on the Great Barrier Reef and the role of injury in the initiation of this widespread coral disease. *Coral Reefs* 27, 257–272.

Palmer C.V., Roth M.S. and Gates R.D. (2009) Red fluorescent protein responsible for pigmentation in trematode-infected *Porites compressa* tissues. *Biological Bulletin. Marine Biological Laboratory, Woods Hole* 216, 68–74.

Pantos O., Cooney R.P., Le Tissier M.D.A., Barer M.R., O'Donnell A.G. and Bythell J.C. (2003) The bacterial ecology of a plague-like disease affecting the Caribbean coral *Montastrea annularis*. *Environmental Microbiology* 5, 370–382.

Patterson K.L., Porter J.W., Ritchie K.E., Polson S.W., Mueller E., Peters E.C., Santavy D.L. and Smiths G.W. (2002) The etiology of white pox, a lethal disease of the Caribbean elkhorn coral, *Acropora palmata*. *Proceedings of the National Academy of Sciences of the United States of America* 99, 8725–8730.

Peters E.C., Gassman N.J., Firman J.C., Richmond R.H. and Power E.A. (1997) Ecotoxicology of tropical marine ecosystems. *Environmental Toxicology and Chemistry* 16, 12–40.

Peters E.C., Oprandy J.J. and Yevich P.P. (1983) Possible causal agent of white band disease in Caribbean acroporid corals. *Journal of Invertebrate Pathology* 41, 394–396.

Peters E.C., Halas J.C. and McCarty H.B. (1986) Calicoblastic neoplasms in *Acropora palmata*, with a review of reports on anomalies of growth and form in corals. *Journal of the National Cancer Institute* 76, 895–912.

Porter J.W., Dustan P., Jaap W.C., Patterson K.L., Kosmynin V., Meier O.W., Patterson M.E. and Parsons M. (2001) Patterns of spread of coral disease in the Florida Keys. *Hydrobiologia* 460, 1–24.

Purdom C.E. and Howard A.E. (1971) Ciliate infestations: a problem in marine fish farming. *Journal du Conseil International pour l'Exploration de la Mer* 33, 511–514.

Qiu D.J., Huang L.M., Huang H., Yang J.H. and Lin S.J. (2010) Two functionally distinct ciliates dwelling in *Acropora* corals in the South China Sea near Sanya, Hainan Province, China. *Applied and Environmental Microbiology* 76, 5639–5643.

Rama Mohan Rao P., Hanumantha Rao K. and Shyamasundari K. (1981) *Helicostoma sigmoida* sp. n. from Waltair Coast. *Geobios (Jodhpur)* 8, 188–189.

Rama Mohan Rao P., Hanumantha Rao K. and Shyamasundri K. (1980) On a new species of the genus *Helicostoma* Claparède & Lachmann 1866. *Indian Science Congress Association Proceedings* 67, 164.

Rawlinson K.A., Gillis J.A., Billings R.E.J. and Borneman E.H. (2011) Taxonomy and life history of the *Acropora*-eating flatworm *Amakusaplana acroporae* nov. sp. (Polycladida: Prosthiostomidae). *Coral Reefs* DOI 10.1007/s00338-011-0745-3.

Raymundo L.J.H., Harvell C.D. and Reynolds T.L. (2003) *Porites* ulcerative white spot disease: description, prevalence, and host range of a new coral disease affecting Indo-Pacific reefs. *Diseases of Aquatic Organisms* 56, 95–104.

Reefdreams (2010) http://reefdreams.de/lang_eng/info_o_enghtml

Richardson L.L., Goldberg W.M., Kuta K.G., Aronson R.B., Smith G.W., Ritchie K.B., Halas J.C., Feingold J.S. and Miller S.L. (1998) Florida's mystery coral-killer identified. *Nature* 392, 557–558.

Richardson L.L. and Aronson R.B. (2002) Infectious diseases of reef corals. *Proceedings of the Ninth International Coral Reef Symposium, Bali, 23–27 October 2000. Volume 2*, 1225–1230.

Riegl B., Bruckner A., Coles S.L., Renaud P. and Dodge R.E. (2009) Coral reefs threats and conservation in an era of global change. *Year in Ecology and Conservation Biology 2009* 1162, 136–186.

Ritchie K.B. (2006) Regulation of microbial populations by coral surface mucus and mucus-associated bacteria. *Marine Ecology Progress Series* 322, 1–14.

Ritchie A.E. and Smith G.W. (1995) Preferential carbon utilization by surface bacterial communities from water mass, normal, and white band diseased *Acropora cervicornis*. *Molecular Marine Biology and Biotechnology* 4, 345–352.

Ritchie K.B. and Smith G.W. (2004) Microbial communities of coral surface mucopolysaccharide layers. In Rosenburg E. and Loya Y. (eds) *Coral health and disease*. Berlin and Heildelberg: Spinger-Verlag, pp. 259–263.

Rodriguez S., Croquer A., Guzman H.M. and Bastidas C. (2009) A mechanism of transmission and factors affecting coral susceptibility to *Halofolliculina* sp. infection. *Coral Reefs* 28, 67–77.

Rosenberg E. and Ben-Haim Y. (2002) Microbial diseases of corals and global warming. *Environmental Microbiology* 4, 318–326.

Rutzler K. and Santavy D.L. (1983) The black band disease of Atlantic reef corals 1. Description of the cyanophyte pathogen. *Marine Ecology* 4, 301–320.

Sekar R., Mills D.K., Remily E.R., Voss J.D. and Richardson L.L. (2006) Microbial communities in the surface mucopolysaccharide layer and the black band microbial mat of black band diseased *Siderastrea siderea*. *Applied and Environmental Microbiology* 72, 5963–5973.

Shannon T. and Achatz J.G. (2007) *Convolutriloba macropyga* sp. nov., an uncommonly fecund acoel (Acoelomorpha) discovered in tropical aquaria. *Zootaxa* 1525, 1–17.

Song W.B. (2000) Morphological and taxonomical studies on some marine scuticociliates from China Sea, with description of two new species, *Philasterides armatalis* sp. n., and *Cyclidium varibonneti* sp. n. (Protozoa: Ciliophora: Scuticociliatida). *Acta Protozoologica* 39, 295–322.

Sussman M., Willis B.L., Victor S. and Bourne D.G. (2008) Coral pathogens identified for white syndrome (WS) epizootics in the Indo-Pacific. *PLoS ONE* 3.

Sutherland K.P. and Ritchie K.B. (2004) White pox disease of the Caribbean Elkhorn coral, *Acropora palmata*. *Coral Health and Disease*, 289–300.

Sweet M.J., Croquer A. and Bythell J. (2011a) Bacterial assemblages differ between compartments within the coral holobiont. *Coral Reefs* 30, 39–52.

Sweet M.J., Croquer A. and Bythell J.C. (2011b) Dynamics of bacterial community development in the reef coral *Acropora muricata* following experimental antibiotic treatment. *Coral Reefs* DOI 10.1007/s00338-011-0800-0.

Teplitski M. and Ritchie K. (2009) How feasible is the biological control of coral diseases? *Trends in Ecology and Evolution* 24, 378–385.

Thompson F.L., Barash Y., Sawabe T., Sharon G., Swings J. and Rosenberg E. (2006) *Thalassomonas loyana* sp. nov., a causative agent of the white plague-like disease of corals on the Eilat coral reef. *International Journal of Systematic and Evolutionary Microbiology* 56, 365–368.

Toller W.W., Rowan R. and Knowlton N. (2002) Genetic evidence for a protozoan (phylum Apicomplexa) associated with corals of the *Montastraea annularis* species complex. *Coral Reefs* 21, 143–146.

Toren A., Lanndau L., Kushmaro A., Loya Y. and Rosenberg E. (1998) Effect of temperature on adhesion of *Vibrio* strain AK-1 to *Oculina patagonica* and on coral bleaching. *Environmental Microbiology* 64, 1379–1384.

Upton S.J. and Peters E.C. (1986) A new and unusual species of *Coccidium* (Apicomplexa, Agamococcidiorida) from Caribbean scleractinian corals. *Journal of Invertebrate Pathology* 47, 184–193.

Wabnitz C., Taylor M., Green E., Razak T., Wabnitz C., Taylor M., Green E. and Razak T. (2003) *From ocean to aquarium. The global trade in marine ornamental species*. Cambridge: UNEP–WCMC, 64 pp.

Weil E. (2001) Caribbean coral reef diseases: status and research needs. In McMananus J. (ed.) *Priorities for Caribbean Coral research, Proceedings, Miami*, pp. 1–8.

Weil E. (2004) Coral reef diseases in the wider Caribbean. In Rosenberg E. and Loya Y. (eds) *Coral health and disease*. Berlin and Heidelberg: Springer-Verlag, pp. 35–67.

Weil E. and Croquer A. (2009) Spatial variability in distribution and prevalence of Caribbean scleractinian coral and octocoral diseases. I. Community-level analysis. *Diseases of Aquatic Organisms* 83, 195–208.

Weil E., Smith G. and Gil-Agudelo D.L. (2006) Status and progress in coral reef disease research. *Diseases of Aquatic Organisms* 69, 1–7.

Williams E.H. Jr and Bunkley-Williams L. (2000) Marine major ecological disturbances of the Caribbean. *Infectious Disease Review* 2, 110–127.

Willis B., Page C. and Dinsdale E. (2004) Coral disease on the Great Barrier Reef. In Rosenberg E. and Loya Y. (eds) *Coral disease and health*. Berlin and Heidelberg: Springer-Verlag, pp. 69–104.

Winkler R., Antonius A. and Renegar D.A. (2004) The skeleton eroding band disease on coral reefs of Aqaba, Red Sea. *Marine Ecology—Pubblicazioni Della Stazione Zoologica Di Napoli I* 25, 129–144.

Zhang Q., Miao M., Struder-Kypke M.C., Al-Rasheid K.A.S., Al-Farraj S.A. and Song W. (2011) Molecular evolution of *Cinetochilum* and *Sathrophilus* (Protozoa, Ciliophora, Oligohymenophorea), two genera of ciliates with morphological affinities to scuticociliates. *Zoologica Scripta* 40, 317–325.

and

Zorpette G. (1995) More coral trouble. *Scientific American* 273, 36–37.

Correspondence should be addressed to:
M. Sweet
School of Biology
Ridley Building, University of Newcastle
Newcastle upon Tyne NE1 7RU, UK
email: m.j.sweet@newcastle.ac.uk

https://doi.org/10.1017/S0025315411001688 Published online by Cambridge University Press