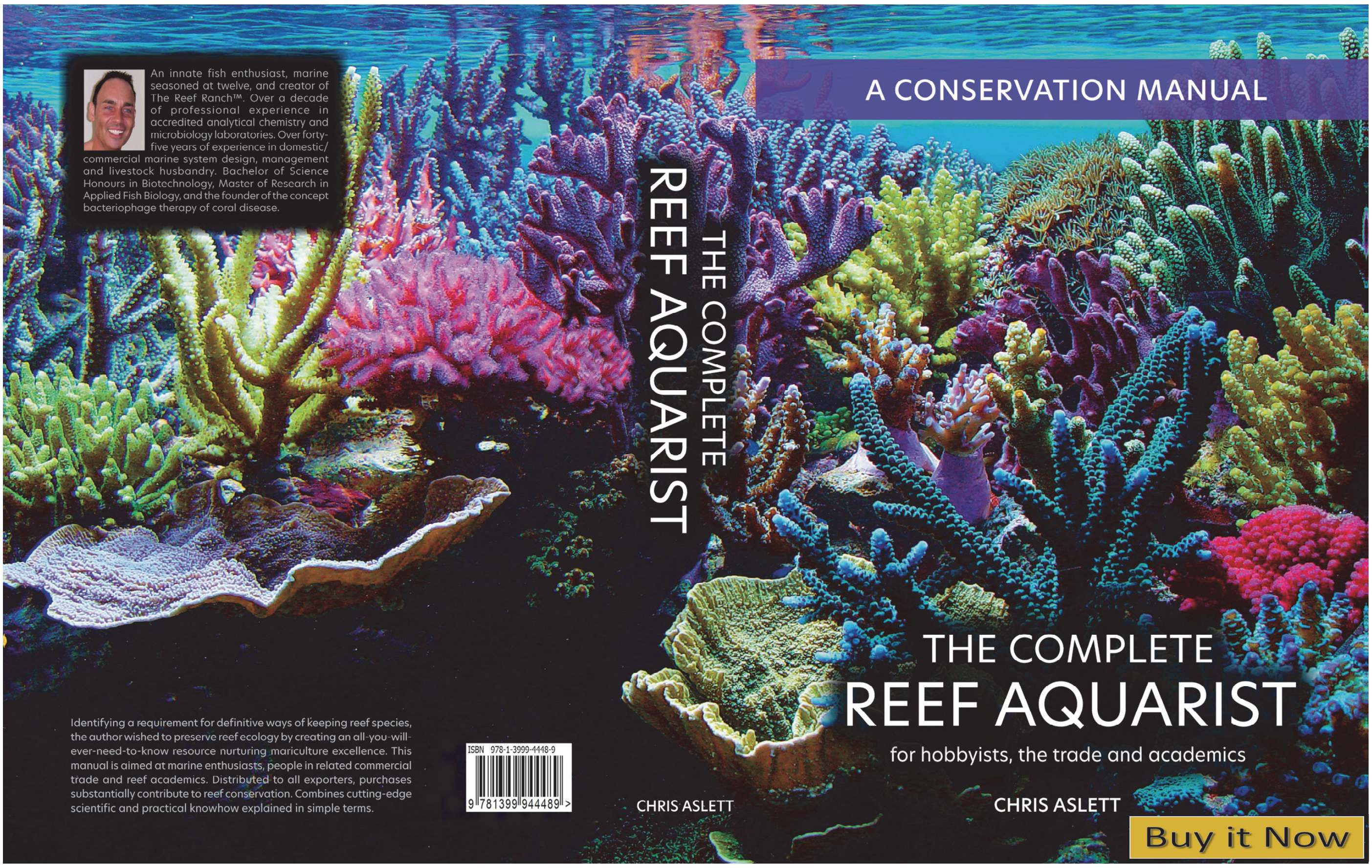

The Complete Reef Aquarist – A Conservation Manual

for the hobbyists, the trade and academics

By

Chris Aslett

Chapter 5: **Diseases of Corals**

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Tropical reefs are threatened by antifouling preparations on shipping, dredging, microplastics, nutrients from nearby pastoral or arable farming, recreational diving, temperature anomalies, sewage discharge, waterborne toxins, and the collection of specimens for the aquarium trade. Excluding anthropogenic activities, a further threat to reef ecology has emerged in the form of coral disease. Reports have been on the increase since the early seventies, and there are few methods of control. Particularly problematic, as they impact hermatypes, pollution has introduced euryhaline microorganisms into the marine environment that gain nourishment from the reef.

Fig 479. A black band diseased boulder of *Pseudodiploria strigosa.*

Bacteria effortlessly acclimate to new environments. Natural selection and Darwinian evolution propose genetic variation enables a species to adapt to environmental change, the time it takes to bear offspring limits acclimation, and those unable to tolerate the new conditions die, which leaves others to thrive. Epigenetics was discussed in a previous chapter, and pathogenic bacteria circumvent immunity by deregulating the transcriptomic biomarkers of coral chromatin (Hamon & Cossart 2012). Bacteria reproduce in minutes, distribute DNA to their neighbours, and replicate their genome in such a manner as to expedite mutations. Moreover, signalling molecules orchestrate microbial agendas and bacteriophages disseminate genes. Prokaryotes have therefore colonised every niche on Earth and most probably beyond.

Serratia marcescens are common human gut commensals that cause white pox in acroporids which were likely inoculated by sewage discharge (Fig 480.; Goreau *et al.* 1998; Lipp *et al.* 2002; Patterson *et al.* 2002; Sutherland & Ritchie 2004; Harding *et al.* 2008; Sutherland *et al.* 2010; Ho 2011; Sutherland *et al.* 2011).

Human intestinal microbes are found in the mucus of healthy corals and the microbial consortium of black band disease (Fig 479.; Frias-Lopez *et al.* 2002; Lipp *et al.* 2002; Lipp & Griffin 2004), whereas the fungi, *Aspergillus sydowii* may cause aspergillosis in Caribbean sea fans (Alcyonacea). Early research suggested the mould was deposited in the Caribbean on particles of north African dust (Shinn *et al.* 2000; Garrison *et al.* 2003), yet Alker and colleagues characterised *A. sydowii* and found it genetically distant from terrestrial strains (Alker *et al.* 2001), which may reflect the degree of variation necessary to fill its current niche. All the same, the findings of a later study suggested the marine *A. sydowii* was unlikely to originate from north Africa (Rypien 2008).

Fig 480. Endangered elkhorn (*Acropora palmata*) exhibiting feeding scars and irregular white patches of necrotic/bleached tissue known as white pox.

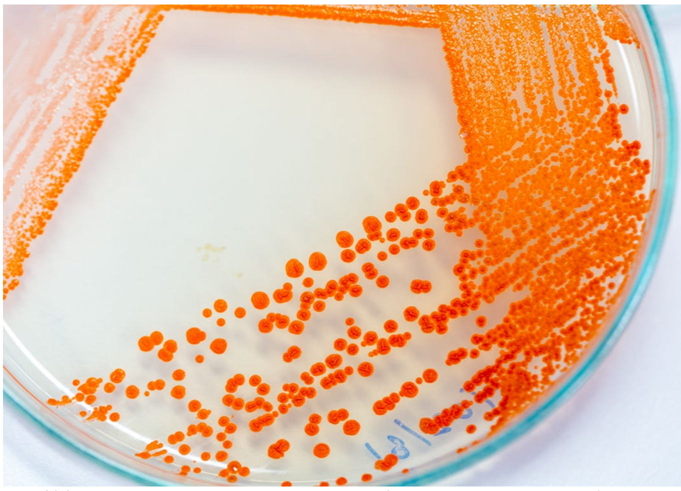


Fig 481. A Petri dish containing a layer of agar upon which the facultative anaerobic bacteria and opportunistic pathogens, *Serratia marcescens* were streaked. Orange prodigiosin is peculiar to several species, where pigmented cells amass ATP and attain greater densities (Haddix & Shanks 2018).

Virulence Factor: A Definition
Virulence factors are manufactured by pathogens to expedite infection, multiplication, dissemination, and the evasion of immunity. Frequently enzymes secreted extracellularly or cell-surface expressed organics, some trigger internalisation into host cells (Peterson 1996).

Quorum Sensing: A Definition
Some bacteria communicate by releasing molecular signals called autoinducers which regulate the genes of others. Quorum sensing impacts virulence factors, growth, and the formation of biofilms and their consortia (Hentzer *et al.* 2003; Vattakaven *et al.* 2006; Tait *et al.* 2010).

Coral Bleaching
There are two kinds: coral flesh can lose all pigment and appear white, whereas colony demise results in the loss of tissue and a white denuded skeleton. During the aetiology of white band disease type II instigated by the bacterium *Vibrio harveyi*,

zooxanthellae are absent a significant while before tissue is destroyed (Fig 482.; Richardson *et al.* 1998a).

Microbiological bleaching was referred to as white syndrome (WS; Borneman 2002; Hobbs & Frisch 2010; Luna *et al.* 2010). However, the idiom has provoked much ambiguity, and thus we later explore WS as defined by Bythell and collaborators who used the term to describe white band disease, white pox, and rapid tissue necrosis (Bythell *et al.* 2004).

Fig 482. Staghorn of the genus *Acropora* putatively infected with *Vibrio harveyi* initiating white band disease type II.

Vertical Transmission: A Definition

Vertical transmission describes the movement of an infective organism from a parent to their offspring, which includes the conveyance of symbionts and commensals to coral planulae (larvae).

Clade: A Definition

Clade describes a group of organisms comprising all the descendants of a common ancestor.

Bleaching Mechanisms

Zooxanthellae's competency for enduring extremes of heat and irradiance is conferred by genotype (Fitt & Warner 1995). Vertical symbiont contagion culminates in the release of infected eggs or planulae (Sharp *et al.* 2011) where various clades were isolated from corals including A, B, C, D, E, F and G (Tonk *et al.* 2014). These now obsolete clades correspond to the genera *Symbiodinium*, *Breviolum*, *Cladocopium*, *Durusdinium*, *Effrenium*, *Fugacium*, and *Gerakladium* (LaJeunesse *et al.* 2018). *Symbiodinium* utilise alternative photosynthetic pathways which disconnect antenna proteins from photosystem II (PSII), so corals with *in hospite Symbiodinium* tolerate temperatures typically associated with impairment (Reynolds *et al.* 2008). Two subtypes of this lineage have a greater or lesser ability to withstand heat (Kramer *et al.* 2012). Mechanisms within symbiont chloroplasts can restore function if temperatures subside; however, sustained adversity forestalls repair, which may trigger the breakdown of the host-symbiont alliance (dysbiosis) synonymous with bleaching (Takahashi *et al.* 2009).

Cladocopium type 3 downregulate the transcription of genes encoding green fluorescent-like pigments (Hume *et al.* 2013), where environmental stressors like enriched nitrate cause zooxanthellae to proliferate which unexpectedly grants a predisposition to bleach (Cunning & Baker 2013; Vega Thurber *et al.* 2014).

Photosynthesis creates reactive oxygen species (ROS) in photosystem I (PSI; Foyer *et al.* 1994; Fitt & Warner 1995; Müller-Moulé 2002), where antioxidant enzymes like superoxide dismutase (SOD) and catalase (CAT) transmute ROS into less harmful by-products (de Grey 2002).

Thermal stress dwindles the photoprotective antioxidants β-carotene, diadinoxanthin, diatoxanthin and peridinin; however, fleeting enriched DIP mitigates antioxidant decline at 31°C (Béraud *et al.* 2013; Ferrier-Pagès *et al.* 2016).

Fig 483. A commensal shrimp on *Oculina robusta* which is a sister taxon of *Oculina patagonica*; however, bleaching caused by *Vibrio mediterranei* has not been described in this species, and despite corallite and colonial morphology being somewhat similar, *O. robusta* and *O. patagonica* are branching and encrusting, respectively. Image courtesy of The Florida Fish and Wildlife Commission©.

The **xanthophyll cycle** quenches the surplus fluorescence of chlorophyll, IR, and excitation energy under intense irradiance, at diminished pH, in the presence of the potent antioxidant, vitamin C (ascorbic acid). The enzyme violaxanthin de-epoxidase converts the carotenoid violaxanthin to antheraxanthin and then to zeaxanthin, whereas violaxanthin is regenerated from zeaxanthin by the enzyme zeaxanthin epoxidase in subdued light. The xanthophyll cycle is induced following the protonation of PSII (Frank *et al.* 2000; Müller-Moulé 2002), while antioxidants become signalling molecules which regulate genes associated with adaptation to abnormal conditions (Frank *et al.* 2000; Müller-Moulé 2002; Ai-Zhen & Fang-Qing 2016).

The regeneration of violaxanthin cannot occur under intense irradiance, while chloroplasts retain a reserve at sunrise which depletes throughout the day. The troughs in the light field occurring during shimmering, are merely a quarter less intense than nominal irradiance, and although meaningful, these brief periods unlike cloud cover, are insufficiently dark or prolonged to reactivate zeaxanthin epoxidase (Stramski & Legendre 1992; Hoegh-Guldberg & Jones 1999; Chiang *et al.* 2008).

A Topical Anecdote

A colony of *Fimbriaphyllia ancora* bleached on one side closest to the glass, while the colony's aggressive nature prohibited relocation. Bleaching was partly attributed to total internal reflection. However, an hour of daybreak sun was observed illuminating the colony seven hours before the lights came on, whereafter an opaque polypropylene cover was fitted to the front of the aquarium each night.

The Mehler-Peroxidase Reaction

Photoreduction in PSI generates the superoxide anion (O_2^-) from dioxygen (O_2) whereupon superoxide dismutase yields hydrogen peroxide (H_2O_2) which is catalysed by vitamin C and ascorbate peroxidase to water, whilst ascorbic acid is replenished by PSI and monodehydroascorbate reductase or NADPH-monodehydroascorbate reductase. This protective mechanism dissipates up to 50 percent of the energy provided by photons under extreme irradiance, yet undermines the typically conserved photosynthetic ratio of one molecule of ATP to one of NADPH. Be that as it may, balance is restored by hitherto unconfirmed pathways, whilst this and the xanthophyll cycle compete for ascorbic acid (Müller-Moulé 2002).

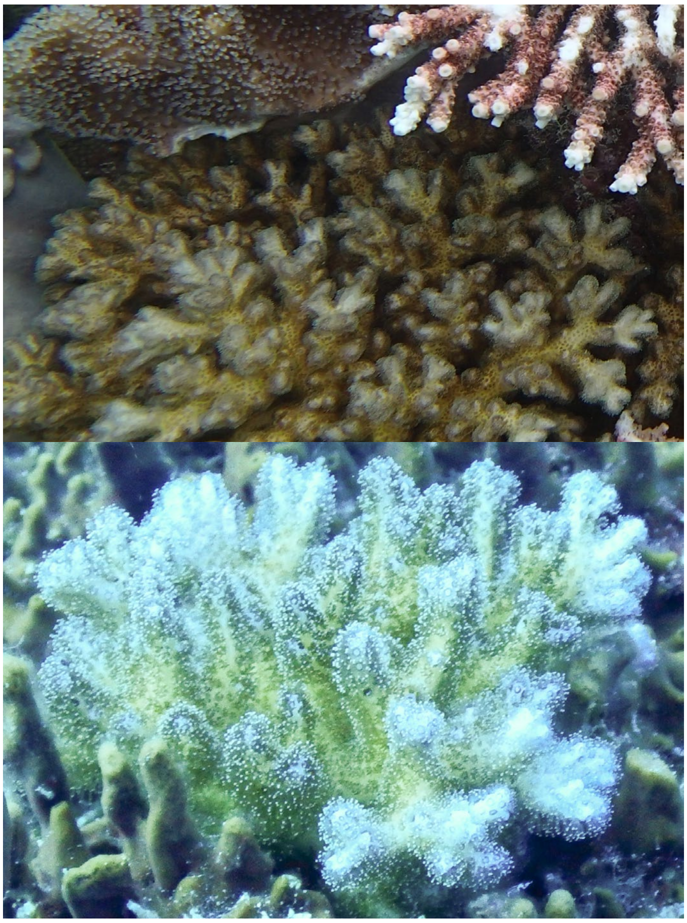


Fig 484. Bleaching of corals of the genus *Pocillopora* putatively instigated by *Vibrio coralliilyticus.*

The above processes downregulate photosynthesis and mitigate ROS, which become ineffectual during immoderations of temperature and/or irradiance. Environmental factors are effortlessly controlled in recirculating systems, and photoperiods may be curtailed on sweltering days, but be sure to reinstate nominal irradiance as soon as temperatures subside.

The milieus of captive reefs can contribute to mortality, and much of this chapter discusses a diseased state arising from the exploits of microscopic pathogens. Nevertheless, several are macroscopic and evade detection using camouflage, which are discussed in Reef Pests.

Global temperatures have risen by an average of 0.6°C in the last 1,000 years (Schmidt 2013), where those of tropical seas are climbing disproportionately (Alexander *et al.* 2018; Levy 2019). Recent spates of coral disease may be symptomatic of advancements in reporting and communication, while research performed on microorganisms has established a link between heat and severity, because corals are weakened and pathogens more virulent at elevated temperatures (Kuta & Richardson 1996; Ben-Haim *et al.* 1999; Banin *et al.* 2000; Banin *et al.* 2001; Israely *et al.* 2001; Ben-Haim & Rosenberg 2002; Oakey *et al.* 2002; Ben-Haim *et al.* 2003; Voss & Richardson 2006; Wang *et al.* 2008; Vega Thurber *et al.* 2014).

Fig 485. A species of *Pocillopora* conceivably bleached by *Vibrio coralliilyticus* inhabited by a commensal crab, *Tetralia* cf. *cavimana*.

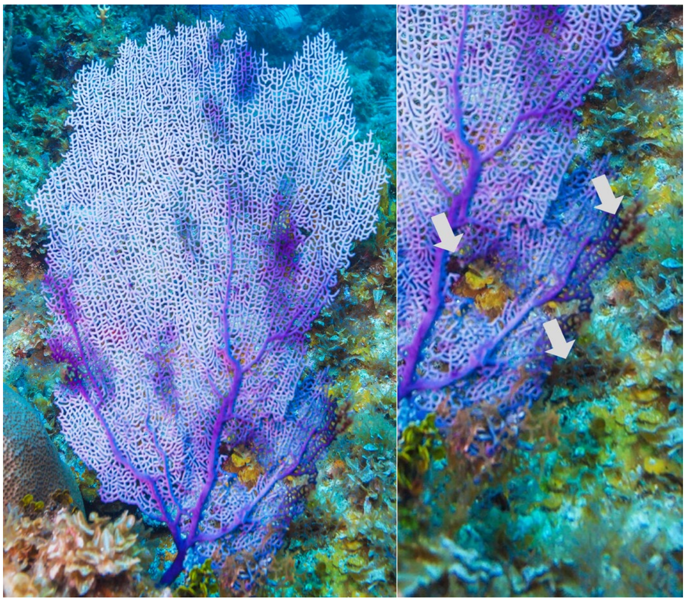


Fig 486. Left - aspergillosis of the Caribbean sea fan, *Gorgonia ventalina*. Right - the same coral invaded by red cyanobacteria redolent of red band disease.

Black band disease was prevalent in the Caribbean, Florida Keys, Bahamas, Indo-Pacific, and on The Great Barrier Reef (GBR), which emerged predominantly in the summer months when sea surface temperatures exceeded 25°C (Carlton & Richardson 1995; Richardson 1997; Dinsdale 2002) while aspergillosis was most severe at 30°C (Alker *et al.* 2001).

The bacteria, *Vibrio mediterranei* (synonym: *Vibrio shiloi*) cause rapid bleaching of *Oculina patagonica*. At higher temperatures, a vibrio cell-surface adhesin binds to a receptor in the host mucus, and remarkably, its availability is determined by symbiont photosynthesis. *V. mediterranei* are either expelled from the coral or destroyed in tissue when temperatures subside in autumn and winter (Ben-Haim *et al.* 1999; Banin *et al.* 2000; Banin *et al.* 2001; Israely *et al.* 2001), and the tentative aetiological agents of white band disease, *Vibrio harveyi*, communicate using quorum sensing but only at low temperature (Tait *et al.* 2010).

Similarly, *V. coralliilyticus* bleach *Pocillopora damicornis* where contagion occurs between 25 and 29°C, when the illness is most severe (Ben-Haim & Rosenberg 2002). The bacteria produce a potent protease that likely contributes to the destruction of tissue, where more enzyme is manufactured at 27 than at 25°C (Ben-Haim & Rosenberg 2002; Ben-Haim *et al.* 2003).

Heat and plastic pollution contribute to coral disease (Ho 2018; Lamb *et al.* 2018), where a three-year dosing experiment enriched nitrogen (N) and phosphorus (P) on a wild reef from 2009 to 2012. Unsurprisingly, nutrient fortification expedited pathogenesis and bleaching (Ho 2013a; Vega Thurber *et al.* 2014). Sustained elevated N and P provoke endogenous competition for DCD, because photosynthate is retained for symbiont proliferation which compromises skeletal accretion (Stambler *et al.* 1991; Langdon & Atkinson 2005). Photosynthesis is curtailed per "alga" which suggests CO_2 becomes limiting even amongst zooxanthellae (Dubinsky *et al.* 1990), whereas most corals recover once oligotrophy is restored (Ho 2013a; Vega Thurber *et al.* 2014).

The concept "One World – One Health" suggests human vigour and well-being are related to our environment, and corals have been likened to coalmine canaries which alert us to climate change (Michael *et al.* 2021).

Fig 487. Red cyanobacteria on denuded cf. *Galaxea*.

Coral disease has been observed in aquaria, where the collaboration of researchers and aquarists could help alleviate the current decline in wild reef ecology. Below we explore the insight amassed by academic studies over the past five decades.

Environmental change can lead to coral invasion and mortality. Antonius and Ballestros surveyed the reefs at Carrie Bow Cay in Belize and Key Largo in Florida, where they found corals overgrown by cyanobacteria. Several skeletons were bare and eroded (Antonius & Ballestros 1998); however, it must be emphasised that incursions of this type are not coral disease but merely examples of how a change in conditions favours the predominance of one kind of organism at the expense of others (Fig 487.; Goreau *et al.* 1998). The coral diseases that alarm conservationists have recognised and defined aetiologies. Proving an organism causes pathology is not a simple affair, insofar as it is not simply a matter of consistently isolating candidates from lesions, at one time they had to satisfy Koch's postulates.

The German scientist Robert Koch devised a set of principles in 1890 that established whether a biological agent caused disease. He recognised that the putative pathogen must be consistently isolated from a lesion, be cultured in a pure form in the laboratory (in vitro), manifest the same disease after infection of a fit host (in vivo), and be re-isolated from that lesion. Some of the aetiological agents of corals have fulfilled Koch's postulates, but some cannot be grown in the laboratory like bacteria of the genus *Vibrio* because they enter a static phase known as viable but nonculturable (VBNC; Vattakaven *et al.* 2006). Some enter dormancy in less-than-ideal conditions and reactivate when things improve (Oliver & Bockian 1995; Sun *et al.* 2008; Colorni & Daimant 2014), whereas contemporary genetic analyses substantiate the infectivity and virulence of VBNC microbes. Several coral abnormalities have been provisionally categorised as diseases, but their aetiology remains poorly understood, and thus we initially explore only extensively researched and characterised idiopathies.

Zoonosis: A Definition

A zoonosis is ordinarily a lethal disease that can be transmitted from animals to humans or vice versa. Influenza is naturally a bird or pig virus, and ebola may be transmitted to humans from bats (Qureshi 2016). The host that transmits or carries the pathogen is referred to as a vector or reservoir.

All the same, the chemical largazole extracted from Floridian cyanobacteria of the genus *Symploca* from Key Largo, slows osteoporosis and induces preprogrammed cell death in human colonic cancers (Taori *et al.* 2008; Lui *et al*. 2010; Garber 2011; Lee *et al.* 2011).

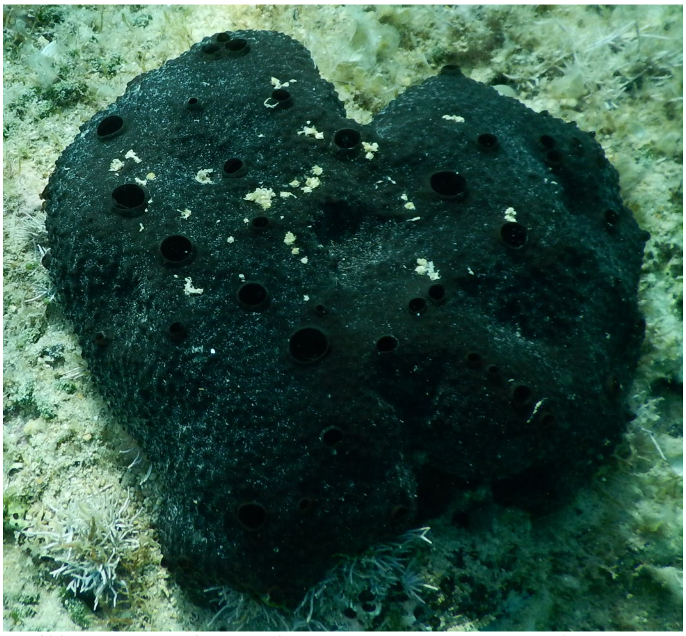


Fig 488. The sponge, *Spongia* cf. *obscura* "shaggy" is a reservoir for *Aspergillus sydowii* (spongeguide.org; Ein-Gil *et al.* 2009).

Aspergillosis

Aspergillosis caused widespread mortalities of sea fans throughout the Caribbean and Florida Keys (Smith *et al.* 1998), where the fungi, *Aspergillus sydowii* were held responsible (Shinn *et al.* 2000). Progression is most rapid at 30°C, and

Gorgonia ventalina are more susceptible than *G. flabellum* (Smith *et al.* 1998; Alker *et al.* 2001). The fungi were thought to be deposited on reefs by storms, where once established they could spread via the water column or by contact (Shinn *et al.* 2000). The mycosis denudes and erodes the skeleton, yet the sea fans (Octocorallia: Alcyonacea) mount a defence by manufacturing an antifungal and elevating the melanin and sclerite components of their tissues, which manifests as galling and purpling. The infection was most prevalent in mature specimens which led to reproductive suppression, and thus sea fans perished and were not replaced (Fig 486.). At one time, more than 50 percent of sea fans were lost from the Florida Keys; however, aspergillosis has since declined, because nearby disease promotes resistance in the rare incidences of enduring larvae (Kim *et al.* 2000a; Kim *et al.* 2000; Dube *et al.* 2002; Petes *et al.* 2003; Kim & Harvell 2004; Bruno *et al.* 2011; Andras 2017).

Fig 489. The corallivorous foureye butterflyfish (*Chaetodon capistratus*) is a recognised vector of black band disease (BBD; Aebu & Santavy 2006).

Contradictory and Supplementary Evidence

Research isolated a strain of *Aspergillus sydowii* in 2009 from a healthy sponge reservoir (*Spongia obscura*; Fig 488.; Ein-Gil *et al.* 2009), while healthy and diseased sea fans were sampled and their fungal communities screened. *A. sydowii* were present in the coral mucus of healthy specimens but not in diseased colonies, and the fungal communities inhabiting coral mucus were more diverse in seemingly healthy Alcyonacea. Such findings infer that *A. sydowii* may not be the causative agents of aspergillosis, where a shift in the composition of the microbial communities of mucus may be largely responsible for disease (Toledo-Hernández *et al.* 2008; Tait *et al.* 2010; Cardenas *et al.* 2011; Egan & Gardiner 2016; Morrow *et al.* 2018). Research has shown that mucosal microbiomes retain significant antimicrobial competencies and are integral components of immunity (Rivera-Ortega & Thomé 2018), whilst a previous study had highlighted *A. sydowii*'s opportunistic nature which has several distinct strains capable of causing disease (Rypien *et al.* 2008). Corroboration of the disease-causing agent of aspergillosis is becoming increasingly moot, because like autoimmune diseases, it may be the manifestation of overcompensating defence.

Black Band Disease (BBD)

The band of BBD is formed from a mixed population of microorganisms that inhabit a network of cyanobacterial filaments which act in unison to destroy coral flesh. The genera *Cytophaga*, *Beggiatoa*, and *Desulfovibrio* inhabit a mat of *Phormidium corallyticum* (synonym: *Roseofilum corallyticum*; Garret & Ducklow 1975; Carlton & Richardson 1995).

Desulfovibrio are classed as sulphate-reducing bacteria which express the product of *dsrA*, sulphite reductase (Müller *et al.* 2015). Comparative sequence analyses of *dsrA* from numerous BBD isolates revealed merely 46 percent homology with that of *Desulfovibrio desulfuricans* (synonym: *Halodesulfovibrio aestuarii*; Barneah *et al.* 2007; Shivani *et al.* 2017) which is redolent of a hitherto unidentified and unrelated lineage.

BBD initiates as a focal lesion which radiates concentrically outward forming a band several millimetres wide that migrates over a cm (0.39") per day to leave a wake of denuded skeleton. Pathology often progresses from a focus of cellular trauma (Aebu & Santavy 2006; Sato *et al.* 2010), where band colouration is provided by the black to dark red light-harvesting cyanoproteins: phycocyanin and phycoerythrin (Fig 479.; Fig 492.; Fig 495.; Rützler & Santavy 1983).

Fig 490. A massive colony of *Montastrea annularis* particularly susceptible to black band disease.

Reef teleosts feed upon the mat which implies they are vectors or bioremediators (Chong-Seng *et al.* 2011), where the findings of an earlier study proposed the consortium was typical of land runoff and sewage discharge (Frias-Lopez *et al.* 2002). With a pantropical distribution, the disease is evident in all warmwater regions (Dinsdale 2002; Frias-Lopez *et al.* 2004; Sato *et al.* 2009).

The upper layers of the band become supersaturated with oxygen during the day, whereas the lower regions remain anoxic and rich in tissue-necrotising hydrogen sulphide (Carlton & Richardson 1995; Rosenberg & Ben-Haim 2002). Bubbles can be seen adhering to the mat, and there is a distinctive smell of "rotten eggs" when the infected coral is exposed to air (Rosenberg & Ben-Haim 2002). BBD infects a variety of Caribbean Scleractinia, yet *Orbicella annularis*, *Montastraea cavernosa*, *Pseudodiploria strigosa*, and *Porites astreoides* are most susceptible (Garret & Ducklow 1975; Frias-Lopez *et al.* 2002). Notwithstanding, corals of the genera *Acropora* and *Pocillopora* are routinely infected on GBR (Fig 491.; Dinsdale 2002).

BBD only infects corals when temperatures exceed 25°C (Kuta & Richardson 1996), whilst Voss and Richardson monitored colony decline when aquaria were dosed with ammonium, nitrite, nitrate, and phosphate, and unsurprisingly, prognoses are poor and aetiologies expedited by enriched nutrients (Voss & Richardson 2006; Vega Thurber *et al.* 2014).

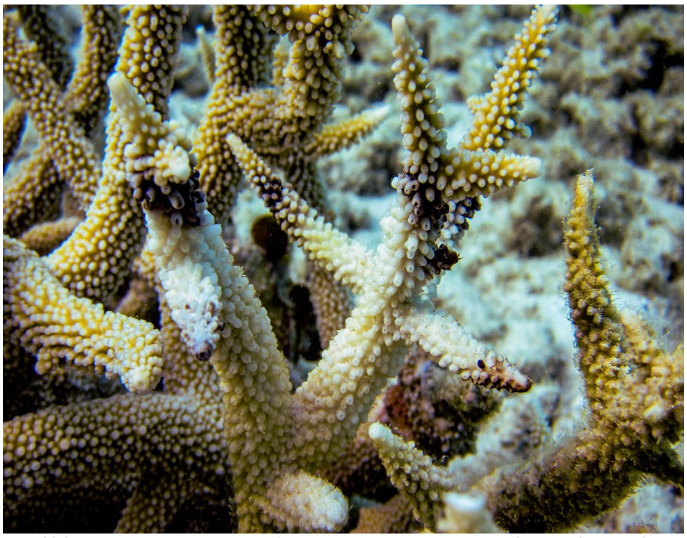


Fig 491. Black band disease of *Acropora*. Ryan McMinds©, the Global Coral Microbiome Project.

Fig 492. Stony coral tissue loss and black band diseases of *Siderastrea siderea*. Image courtesy of The Florida Fish and Wildlife Commission©.

Merely present in bands from these geographic regions, microcystin pervaded lesions in the Florida Keys and the Bahamas which was the first report of this cyanotoxin associated with coral disease (Richardson *et al.* 2007). Microcystin is commonly associated with cyanobacterial blooms in N- and P-enriched freshwater, because the algae lyse which liberates their contents after nutrients become limiting. As a hepato-(liver)-toxin, 60 people died in 1996 after drinking contaminated water from a Brazilian lake (Pouria *et al.* 1998).

Corallivorous foureye butterflyfish (*Chaetodon capistratus*), spread BBD from one coral to another when they feed, whilst teleostean faeces remain credible vectors. BBD was exceedingly virulent in a warm recirculating system where it quickly infected new specimens (Aebu & Santavy 2006), which might be communicated in water (Zvuloni *et al.* 2009), whereas coral-eating snails of the genus *Drupella* contribute to contagion (Onton *et al.* 2011).

Contrasting Research

A later study characterised the microbial communities associated with BBD, whose cyanobacteria were consistently distinct from the previously identified strain of *Phormidium corallyticum* (Cooney *et al.* 2002) which was upheld by Frias-Lopez and colleagues in 2003. None of the identified microorganisms originated from land, humans, or freshwater. Every band was inhabited by species of *Disulphovibrio*, previously undescribed bacteria of the genus *Cytophaga,* and acknowledged γ-proteobacterial pathogens of oysters (Cooney *et al.* 2002). The profile of the microbial community appears attuned to the species and strain of cyanobacterial mat (Frias-Lopez *et al.* 2004a) while the microorganisms remain interdependent like biofilm-constrained communities.

Fig 493. *Porites* cf. *astreoides* susceptible to black band disease where white denuded patches are redolent of yellow band or white syndrome.

Fig 494. Centre - tentative yellow band disease of *Stephanocoenia intersepta.*

In 2012, analyses revealed that lesions of Caribbean BBD are consistently inhabited with cyanobacteria of the subclass Oscillatoriophycideae, which manufacture exceedingly potent microcystin-LR. An isolate was identified and assigned the new genus and species, *Roseofilum reptotaenium*, and experiments revealed the blue-green alga preferentially grew at pH values between 6 and 8 (Casamatta *et al.* 2012), whereas a later study discovered pH 7.7 diminished the density of the consortium and aetiological progression (Muller *et al.* 2017). It is likely that the pH of the microenvironments within the band are under the control of the microbial consortium, insomuch as surface oxygen supersaturation contrasts with sub-mat hydrogen sulphide (Rosenberg & Ben-Haim 2002).

It is ill-advised to intentionally suppress pH and ORP to prevent coral disease (Ho 2017) while the above incongruous studies were carried out in aquaria, where corals in nature can thrive in conditions that might kill those in captivity, so recirculating systems may lack an important unidentified ingredient.

Epizootic: A Definition

An epizootic is like a human epidemic insomuch as it is a widespread outbreak of disease in an animal population.

Bacterial Infections and Bleaching

Bacteria of the genus *Vibrio* are ranked in the class γ-Proteobacteria (Sussman *et al.* 2008), where bleaching is instigated by *V. mediterranei* and *V. coralliilyticus* in respective *Oculina patagonica* and *Pocillopora damicornis* (Fig 483. to Fig 485.). *V. mediterranei* generate a protein fragment that terminates photosynthesis in the presence of ammonia, which originates from the necrosis of tissue elicited by the bacteria (Ben-Haim *et al.* 2000). Furthermore, *V. mediterranei* release a dysbiosis-initiating substance and can adhere to the mucus of an actively-photosynthesising host (Toren *et al.* 1998; Banin *et al.* 1999; Banin *et al.* 2000; Ben-Haim *et al.* 2000).

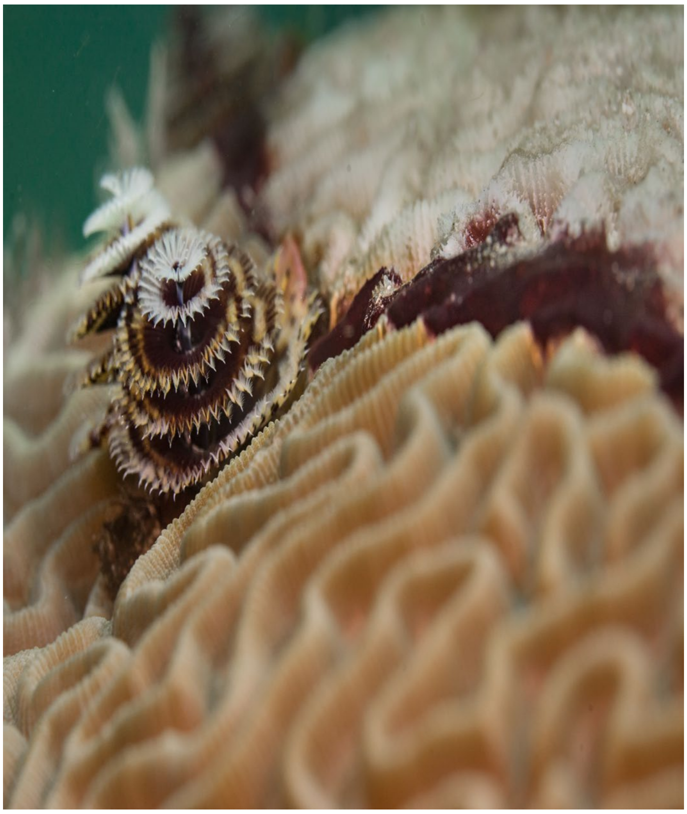


Fig 495. BBD of *Pseudodiploria strigosa.* Image courtesy of Dan Mele©.

Fig 496. The marine fireworm (*Hermodice carunculata)* is a nocturnal polychaete (bristleworm) that devours invertebrates.

V. mediterranei cannot be isolated from the water within which infected corals reside (Banin *et al.* 2000) while the pathogen is contracted during seasonal upturns of sea surface temperature (SST). Hence these bacteria are expelled or killed when ocean warmth subsides, so a reservoir and vector are prerequisites for the following spring's contagions (Israely *et al.* 2001).

Fig 497. Castle corals (*Pachyseris speciosa*) form plating colonies which are susceptible to bleaching caused by *Vibrio coralliilyticus*.

Marine fireworms (*Hermodice carunculata*) are reservoir hosts and vectors, because those of Mediterranean winters carry sizeable inoculums of skin-associated VBNC *V. mediterranei*. All aquarium-confined *Oculina patagonica* fed upon by *H. carunculata* were infected and bleached within six weeks (Fig 496.; Sussman *et al.* 2003; Rosenberg & Falkovitz 2004).

Spread in the water column or through contact, *V. coralliilyticus* instigate rapid bleaching of *Pocillopora damicornis.* These bacteria are undetectable during early contagion (Ben-Haim & Rosenberg 2002) which infect only zooxanthellae and release a zinc-metalloprotease (MP) that inactivates PSII, but destroys the flesh of their host (Ben-Haim *et al.* 2003a; Sussman *et al.* 2008; Sussman *et al.* 2009; Vidal-Dupiol *et al.* 2011). *V. coralliilyticus* are attracted to *P. damicornis* through their mucosal sulphur-rich metabolite, dimethylsulphoniopropionate (DMSP) which leaks during stress and elicits pathogen chemotaxis (Garren *et al.* 2013). Contagious at 25°C yet most virulent at 29, these pathogens tolerate up to 7 percent salt whilst 8 percent is nonpermissive (Ben-Haim & Rosenberg 2002).

Coral bleaching was investigated in Nikko Bay in the Republic of Palau; Nelly Bay on GBR, and around the Majuro Atoll in the

Republic of the Marshall Islands. *V. coralliilyticus* were isolated from bleached *Pachyseris speciosa*, *Montipora aequituberculata*, and *Acropora cytherea* (Sussman *et al.* 2008).

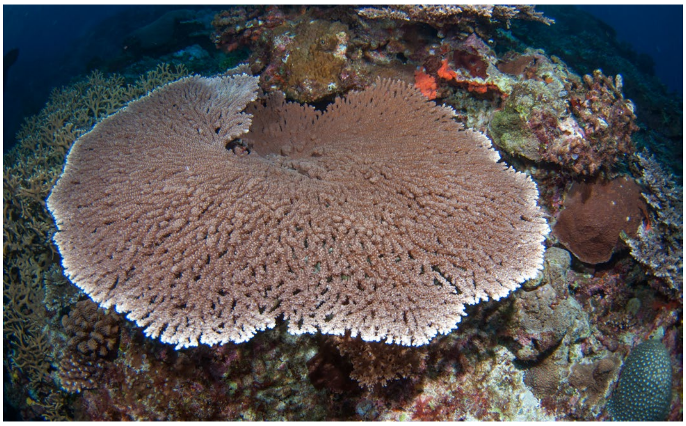


Fig 498. A giant tabular deep-water colony of *Acropora cytherea* vulnerable to bleaching caused by *Vibrio coralliilyticus*.

White Band Disease (WBD) infects corals at Christmas and Cocos Islands in the Indo-Pacific and in the wider Caribbean including the Bahamas and the Florida Keys (Bruno & Selig 2007; Myers & Raymundo 2009; Hobbs & Frisch 2010; Luna *et al.* 2010). Like most coral diseases, outbreaks are associated with the hotter months and anomalously-warm sea-surface temperatures (SSTs; Hoff 2007; Muller *et al.* 2018). All acroporans are vulnerable yet the disease is commonest in endangered *A. palmata* (elkhorn) and scarce *A. cervicornis* (staghorn). A type I infection denudes the skeleton from the base to the tips, whilst type II either starts at the tips and progresses downwards or occurs mid-branch in *A. cervicornis* (Fig 482.; Fig 501.; Fig 502.; Fig 506.; Fig 507.; Goreau *et al.* 1998; Ritchie & Smith 1998; Mayor *et al.* 2006). Type I advances 3 to 6 mm (0.12 to 0.23") per day, whereas type II bleaches tissue prior to destruction and progresses 9 (3.5; Richardson *et al.* 1998; Borneman 2002). Despite *V. harveyi* causing disease in tiger prawns (*Penaeus esculentus*), spiny lobsters (*Panulirus homarus*), pearl oysters (*Pinctada maxima*) and its consistent isolation from the lesions of WBD, the aetiological agents of this condition remain enigmatic (Oakey & Owens 2000).

V. harveyi have lysogenic (temperate) bacteriophages called vibrio harveyi myovirus-like (VHML) which carry genes that encode for bacterial virulence factors (Fig 499.; Oakey & Owens 2000; Oakey *et al.* 2002), whilst their quorum sensing is weakened at higher temperatures (Vattakaven *et al.* 2006; Tait *et al.* 2010). Moreover, these bacteria have been isolated from the lesions of yellow band disease (Luna *et al.* 2010).

Viruses: A Simplified Overview

The scientific perspective is that viruses are "not living", because the majority cannot produce ATP or translate mRNA into proteins and are thus obligate intracellular parasites that cannot reproduce without the machinery of their host (Wessner 2010).

They consist of a three-dimensional capsid constructed from a polymer of one or more subunit proteins that contain and protect the viral nucleic acid. Their genomes are single or multiple segments of RNA or DNA, and when nascent virions bud from the cell, they take with them a section of the membrane that forms their envelope. Conversely, cellular lysis releases a myriad of "naked" virions (Lodish *et al.* 2000).

The terms virion and virus appear somewhat interchangeable which refer to an inert infective particle. A virus infects a cell and either enters the lytic cycle which orchestrates host mechanisms to generate progeny virions, or it enters a form of latency called lysogeny where some of its genome is expressed which often confers auxiliary attributes (Fig 500.; Hershey *et al.* 1952; Bandea 1983; Cohen 2016).

Coral plague (CP)

Coral plague was first reported by Phillip Dustan in the Florida Keys as early as 1977, which spreads from the base upwards and is now prevalent throughout the Caribbean (Dustan 1977; Goreau *et al.* 1998; Richardson 1998; Richardson *et al.* 1998; Richardson *et al.* 2001). Types I, II, and III range from the least to the most virulent, where the disease advances several mm per day with up to 10-cm (3.9") weekly progressions in II and III (Richardson & Aronson 2000; Borneman 2002). CP typically infects corals of the genera *Colpophyllia* and *Mycetophyllia* as well as *Dichocoenia stokesii* which are most susceptible (Fig 503.; Goreau *et al.* 1998; Richardson 1998; Richardson *et al.* 1998; Richardson *et al.* 2001). Healthy tissue and denuded skeleton share a discrete boundary which is indicative of enzymatic or toxin-induced necrosis, where a previously unrecognised bacterium of the genus *Sphingomonas* was isolated from a lesion and satisfied Koch's postulates (Richardson *et al.* 1998; Richardson *et al.* 2001).

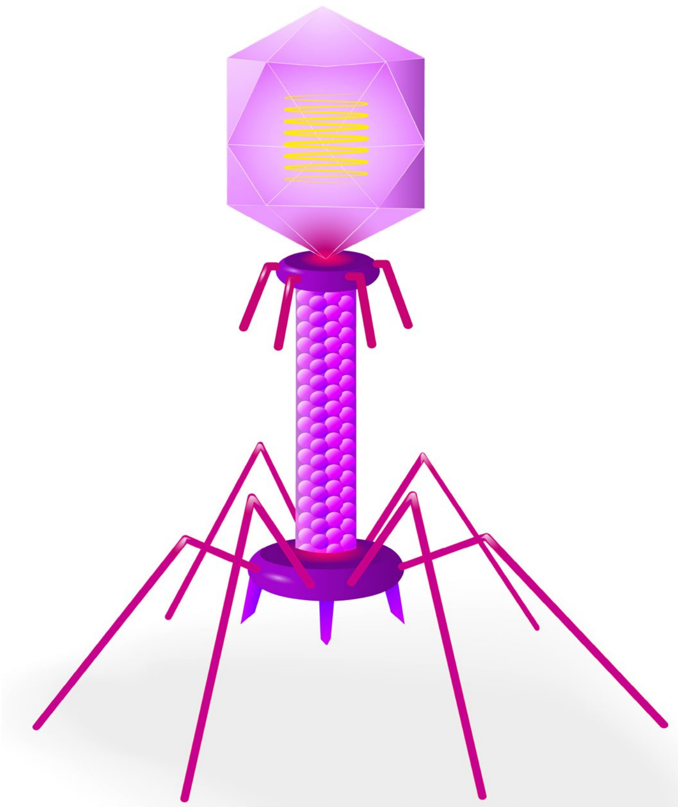


Fig 499. An illustration of a typical myovirus bacteriophage with a genome-enveloping capsid head. The legs firmly attach to the exterior of a bacterium and the base plate penetrates the cell membrane, whereafter nucleic acid is injected by a contraction of the neck.

A member of the phylum Proteobacteria sharing up to 92 percent homology with *Mesorhizobium mediterraneum* was isolated from the lesions of CP, for which the study proposed the new genus and species, *Aurantimonas coralicida* nov. sp. (Denner *et al.* 2003). A strain of the bacterial family Colwelliaceae and genus *Thalassomonas* was discovered in CP's infective boundary in 2006 which was classified as the new species, *Thalassomonas loyana* nov. sp. (Thompson *et al.* 2006). BA_3 bacteriophages specific for *T. loyana* were inoculated to slow the progression and lessen infectivity of CP amongst *Dipsastraea favus* in the Red Sea (Atad *et al.* 2012; Garber 2012), while bacteriophage therapy of coral disease was a comparatively old idea (Aslett 2003; Efrony *et al.* 2007; Efrony *et al.* 2009; Teplitski & Ritchie 2009).

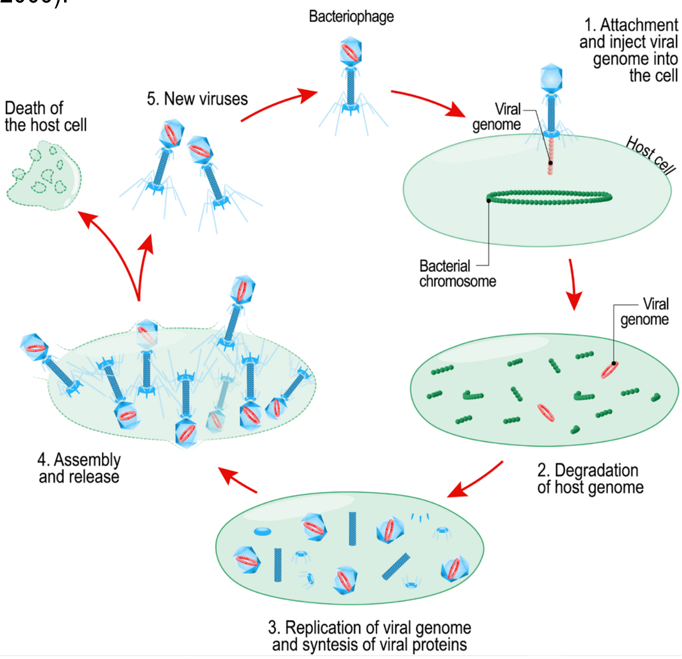


Fig 500. The lytic life cycle of a virulent bacteriophage.

Fig 501. Another example of white band disease type II infecting a species of *Acropora*.

Contrasting Evidence

Aurantimonas coralicida has not been re-isolated from diseased colonies, and CP was renamed white plague (WP; Pantos *et al.* 2003; Sunagawa *et al.* 2009). The community composition of each coral's mucosal microbiome altered before and following an infection, where defence appears inextricably linked to a diverse population of microbiota (Tait *et al.* 2010). A strain of bacteria isolated from lesions of WP closely resembled a known oyster pathogen and the γ-Proteobacteria that inhabit the mat of BBD (Pantos *et al.* 2003). Be that as it may, later research failed to identify pathogenic microbes but isolated a consortium of viruses. The viral affiliates of infected and healthy corals were distinct (Ho 2013; Soffer *et al.* 2013), and this is not an isolated incidence of a substantiated but absent pathogen. Researchers proposed that coral disease may be the manifestation of opportunistic pathogenesis, because coral immunity is diminished or overwhelmed at higher temperatures and temperate 'phage become lytic, which may provide a foothold for commensal or environmental microbes to proliferate and destroy tissue (Lesser *et al.* 2007; Hall *et al.* 2018; Morrow *et al.* 2018).

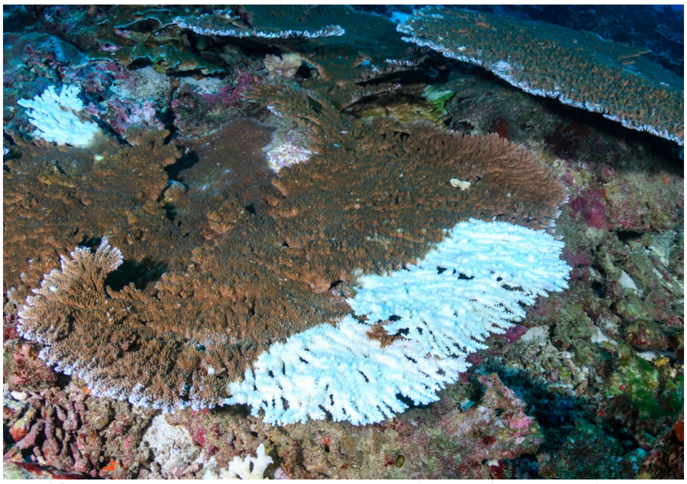


Fig 502. A putative white band disease type II infection of *Acropora* cf. *cytherea* which initiates at the tips and advances towards the base.

Contagions tend to cluster around infected individuals, where yellow band disease, dark spot syndrome, and WP proved atypical. These findings support the hypothesis that weakened corals become susceptible to localised opportunistic microorganisms, and not from the transmission of a primary aetiological agent (Miller *et al.* 2003; Francini-Filho *et al.* 2010; Muller & Woesik 2012; Ruiz-Moreno *et al.* 2012; Hall *et al.* 2018; Morrow *et al.* 2018).

Fig 503. Coral plague (synonym: white plague) of *Pseudodiploria strigosa*, St. Thomas, the US Virgin Islands. Photograph by Dan Mele©.

Epizootics: White Plague

WP type II decimated *Dichocoenia stokesii* in the northern Florida Keys leaving only 75 percent uninfected from 1995 to

2002, while no juvenile colonies were detected throughout the seven years that followed. Merely sizeable colonies prevailed which inferred reproduction had ceased (Richardson & Voss 2005).

WP emerged in Virginia Key in south-eastern Florida in September 2014 which spread over an expanse of 130 square kilometres (km^2) over the following year. Mortalities ranged from 97 percent of *Eusmilia fastigiata, Meandrina meandrites*, and *Dichocoenia stokesii* to 75 percent of *Colpophyllia natans, Pseudodiploria strigosa, Diploria labyrinthiformis,* and *Orbicella annularis* (Precht *et al.* 2016). Yellow band disease and WP were monitored at La Parguera in Puerto Rico from 2003 to 2007 where 53 percent of corals succumbed (Croquer & Weil 2009; Weil *et al.* 2009).

White Pox (WPX; synonym: Acroporid Serriatosis; APS) manifests at elevated temperatures where the bacteria, *Serratia marcescens* were confirmed as the causative agents using comparative sequence analyses. This was the first substantiated incidence of a human gut commensal causing zoonotic mortalities in corals (Fig 480.; Fig 481.; Patterson *et al.* 2002; Ho 2011). Irregular shaped patches are sloughed from the skeleton where species of *Acropora* are most at risk like elkhorn (*A. palmata*; Goreau *et al.* 1998). *S. marcescens* adhere to coral mucus (Lipp *et al.* 2002; Patterson *et al.* 2002) while outbreaks were reported in Hawai'i (Ho 2012). Elkhorn were annihilated in the Florida Keys where merely 18 percent remained, whilst researchers proposed the coral was a candidate for the endangered species list. *A. palmata* was ranked amongst the US endangered and threatened species in 2006 (Sutherland & Ritchie 2004; Hogarth 2006; Sutherland *et al.* 2011).

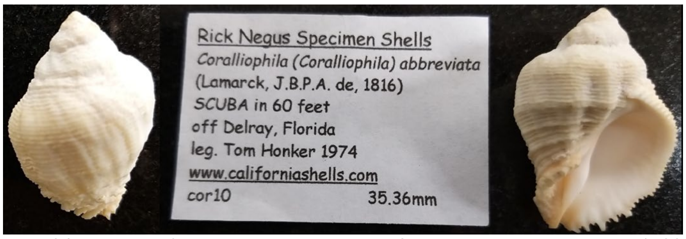


Fig 504. A shell of the corallivorous snail, *Coralliophila erosa* Roding 1798 (synonym: *Coralliophila abbreviata* Lamarck 1816). Responsibly sourced from an established Floridian collection (MolluscaBase 2019; Negus, personal communication).

The corallivorous snails, *Coralliophila abbreviata* Lamarck 1816 were reclassified as *C. erosa* Roding 1798 (Kaullysing *et al.* 2019; MolluscaBase 2019). Cells of the strain PDR60 of *S. marcescens* were isolated from diseased *A. palmata*, the seemingly healthy coral *Siderastrea siderea*, human wastewater, and the snail *Coralliophila erosa* (Fig 504.). WPX was renamed acroporid serriatosis (APS) whilst laboratory experiments satisfying Koch's postulates revealed PDR60 caused APS in *A. palmata*. *Siderastrea siderea* exhibited limited and delayed APS after being exposed to *C. erosa*, which implicated the snails as reservoirs yet refuted them as vectors (Ho 2011; Sutherland *et al.* 2011; Gignoux-Wolfsohn *et al.* 2012).

Elkhorn were surveyed for damage following storm swells in 2008, where 41 percent of injured corals were infested with *C. erosa* and APS by 2009. Those infected were teeming with 46 percent more snails compared with healthy colonies (Bright *et al.* 2016).

Contradictory Evidence

The prevalence of APS and *S. marcescens* were investigated in the Florida Keys. Bacteria were isolated from healthy and diseased colonies, while symptoms of APS were observed sporadically on several species, but no colonies were overwhelmed (Joyner *et al.* 2015). Porter and collaborators made similar observations in 2001, where the commensal microbes of coral polyps confer resilience to PDR60 (Garber 2011a). Diverse populations of mucosal microflora retain antimicrobial properties that render corals impervious to disease (Pantos *et al.* 2003; Tait *et al.* 2010; Rivera-Ortega & Thomé 2018).

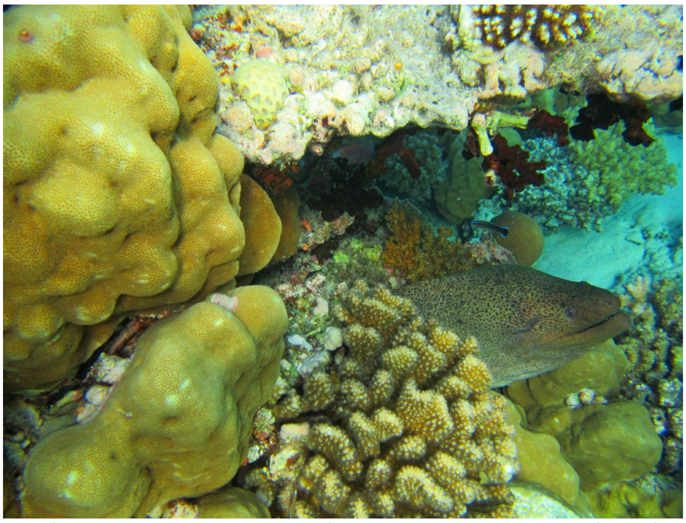


Fig 505. Bottom left - photography of what appears to be *Porites* cf. *nodifera* harbouring corallivorous snails consistent with the observations of Johnston & Miller 2014.

Seasonal bleaching and recovery were evaluated in *Acropora palmata* from 2003 to 2010 at Saint John, the US Virgin Islands, where negligeable sexual and asexual reproduction contributed to nominal recruitment (Rogers & Muller 2012). A study proposed that pathogenesis was a dynamic process in flux, influenced by microorganisms, the coral, and the environment (Sutherland *et al.* 2016), whereas other research observed transmission of APS via coral to coral contact and the corallivory of *C. erosa* (Williams & Miller 2005).

Fig 506. A putative white band disease type I infection of a SPS coral because bleaching is progressing, albeit nominally, from the base upward.

Uncharacterised: Lethal Coral Ailments

The following maladies are not fully described or extensively researched, but remain conspicuous, thanks to their large-scale mortalities.

Yellow Blotch Syndrome (YBS; synonym: yellow band disease) affects *Orbicella annularis* and *Colpophyllia natans* throughout the Caribbean and the waters of Colombia (Santavy *et al.* 1999); nevertheless, an extensive host range embraces *Diploria labyrinthiformis*, *Favia fragum*, *Porites astreoides*, and *Pseudodiploria strigosa* (Fig 493.; Garzón-Ferreira *et al.* 2001; Gil-Agudelo *et al.* 2004). Moreover, an epizootic in the Arabian Gulf diseased *Acropora clathrata*, *A. florida*, *A. pharaonis*, *A. tenuis*, *A. valida*, *Cyphastrea microphthalma*, *Porites lichen*, *P. lutea*, *P. nodifera*, and *Turbinaria reniformis* (Fig 508.; Fig 509.; Korrubel & Riegl 1998).

Fig 507. Elkhorn (*Acropora palmata*). This colony has been stripped of its flesh from beneath, and there is a discrete virtually horizontal line where the putative infection occurred. Seasonal downturns arrest pathology and *A. palmata* is significantly susceptible to white band disease (WBD). The illness may have been WBD type I because much of the neighbouring reef was affected, and remarkably, what appears to be a foureye butterflyfish is nibbling its polyps (Fig 489.).

YBS is chronic insomuch as it advances merely 7 to 10 cm (2.76 to 3.9”) per annum, where ancient colonies appear more susceptible. Initiating as a focus or band of translucent yellow tissue, it radiates concentrically outward (Santavy & Peters 1997; Santavy *et al.* 1999; Borneman 2002; Bruckner & Bruckner 2003). An unidentified crystalline substance pervades the gastrodermis of each polyp, whereas the zooxanthellae cease multiplication, lose their organelles, and vacuolate (Cervino *et al.* 2001). 90 percent of Caribbean *Orbicella annularis* had acquired the syndrome by 1997, which inactivates up to 96 percent of the coral’s algal partners. Studies concluded the condition primarily affects the zooxanthellae while it exerts secondary effects on the host (Bruckner 2001; Cervino *et al.* 2004a).

Virus-like particles were discovered in symbionts, and species of *Vibrio* were isolated from lesions (Cervino *et al.* 2004; Cervino *et al.* 2008; Marhaver *et al.* 2008). *V. harveyi* were implicated (Luna *et al.* 2010) and progression was stemmed by chiselling a skeletal “firebreak” on *O. faveolata* (Randall *et al.* 2018).

Dark Spot Syndrome (DSS) is widespread throughout the Caribbean but does not emerge below 28ºC (Gil-Agudelo & Garzón-Ferreira 2001; Gil-Agudelo *et al.* 2004). Commonest in *Siderastrea siderea, Stephanocoenia intersepta*, and *Orbicella annularis*, corals suffer from a lack of polyp extension and manifest brown or purple foci with necrotised centres. Loss of tissue progresses 4 cm (1.6”) per month, and like YBS, zooxanthellae are fewer and unable to divide (Bruckner 2001). Strains of *Vibrio harveyi* were isolated from lesions but did not replicate symptoms in fit hosts (Fig 510.; Gil-Agudelo *et al.* 2004).

Stony Coral Tissue Loss Disease (SCTLD)

A disease instigating tissue loss in species of *Montipora* was reported in Hawai’i in 2012, and the bacteria *Vibrio owensii* were consistently isolated from lesions but failed to cause the same illness in healthy corals (Ushijima *et al.* 2012). Stony coral tissue loss disease (SCTLD) has been reported in the wider Caribbean since 2014, which presents as multiple cores of dead tissue that advance over the entire colony within several weeks. The disease is exceedingly contagious and spreads via the water column or contact, and persists in the environment for long periods. For instance, Florida’s epizootic has lasted for several years, where more than half the corals have been affected over an area of 150 km^2 (Weinberg 2018). *Dendrogyra cylindrus*, *Dichocoenia stokesii*, *Eusmilia fastigiata*, and *Meandrina* are most vulnerable, whereas *Acropora* appear unaffected (International Coral Reef Initiative 2019). Conservationists remain gravely concerned, where the threat motivated the federal agencies of multiple states to bank Florida’s scleractinian diversity in 2018 (Fig 492.; Fig 511.; Schopmeyer, personal communication). See: Coral Propagation, Reef Conservation.

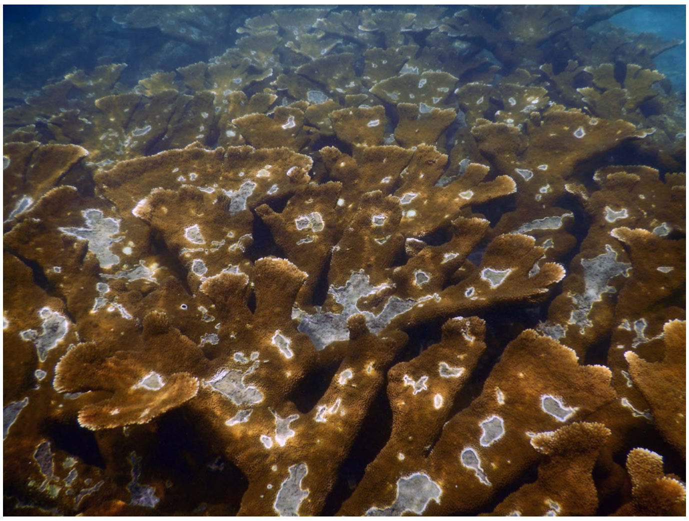


Fig 508. *Acropora palmata* (elkhorn) with what appears to be yellow blotch syndrome, which is widespread amongst acroporids, yet atypical in this species (Korrubel & Riegl 1998).

Fig 509. *Colpophyllia natans* is susceptible to yellow band disease; however, the photograph was captured in Malaysia where YBS is unrecognised, while the discolouration may be necrotic tissue consistent with the findings of Harvel and collaborators from 2007.

Red Band Disease (RBD) is like BBD, insofar as a cyanobacterial mat consisting of filaments of the genus *Oscillatoria* is colonised by microorganisms acting in unison to destroy tissue, but only during the day (Richardson 1993; Santavy *et al.* 1996; Santavy & Peters 1997). The band’s colouration and texture is more maroon and floccose compared to BBD, where types I and II are recognised, with the former observed in the genera *Agaricia, Colpophyllia, Mycetophyllia, Stephanocoenia,* and the species *Gorgonia ventalina* (Fig 486.), whereas RBD II affects *Pseudodiploria strigosa, Colpophyllia natans, Orbicella annularis, Montastraea cavernosa, Porites astreoides*, and *Siderastrea radians.* Moreover, species of *Turbinaria* were most affected in the Gulf of Mannar (Chinnachamy & Patterson 2004). Type II takes on a dispersed net-like form during daylight which condenses into a tight mass at night, where disease has been observed in the Caribbean, the Pacific, the Indian Ocean, and on GBR (Fig 486.; Fig 522.; Rützler & Santavy 1983; Richardson 1992).

Fig 510. Dark spot syndrome and parrot fish predation scars on *Siderastrea siderea.*

Fig 511. Stony coral tissue loss disease (SCTLD) of *Pseudodiploria strigosa*, St. Thomas, the US Virgin Islands. Photograph by Dan Mele©.

Rapid Tissue Necrosis (RTN) or shutdown reaction was first reported in 1977 (Antonius 1977) which is commonest in SPS corals and denudes the skeleton from 1 to 3.9" (2.5 to 10 cm) each hour (Borneman 2002). The syndrome has been studied in the wild and in aquaria where *Acropora* and *Pocillopora* are most susceptible (Luna *et al.* 2007), while the loss of tissue is so immediate the observer sees the flesh peel from the skeleton (Fig 512.). RTN is associated with housing corals in poor conditions like those of immature systems, whereas healthy tissue and bare skeleton share a discrete boundary. The cause is unknown, yet *Vibrio vulnificus* have been isolated from lesions, which secrete the virulence factors metallo- and serine-proteases (Wang *et al.* 2008). Research suggested RTN may be caused by *V. harveyi* or inorganic toxins (Borneman 2001; Borneman & Lowrie 2001; Luna *et al.* 2007; Calfo 2009; Osinga *et al.* 2012), whilst the author has observed RTN in systems contaminated with hydrogen sulphide.

Fig 512. A species of *Acropora* with rapid tissue necrosis (RTN).

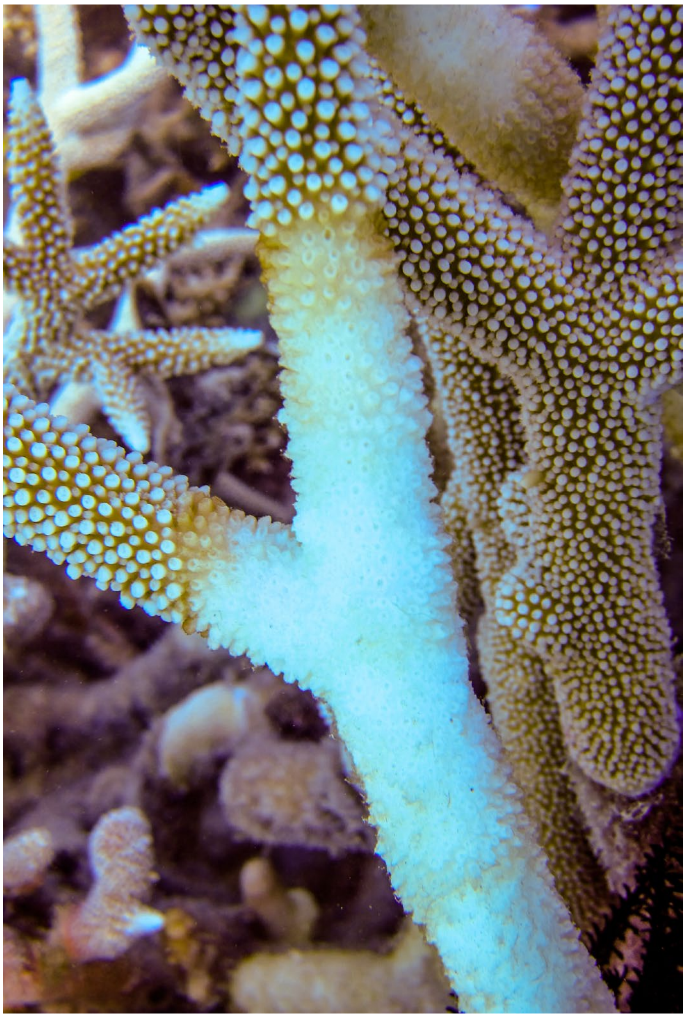


Fig 513. Brown band disease of *Acropora muricata*. Image courtesy of Ryan McMinds©, the Global Coral Microbiome Project.

Brown Band Disease (BrB) has been recorded on GBR where it affects acroporids, whilst vectors include corallivorous snails of the genus *Drupella* (Fig 514.; Willis *et al.* 2004; Joleah & Willis 2011; Nicolet *et al.* 2013). The ciliated chromists *Porpostoma guamensis* consume lysed cells and symbionts (Lobban *et al.* 2011). There are two morphotypes: the first were thought responsible for the disease whilst the second were regarded as opportunistic. Morph I were close relatives of *Philaster digitiformis* which were observed burrowing into and devouring the coral and zooxanthellae, whereas type II were the previously isolated *P. guamensis* (Lobban *et al.* 2011; Sweet & Bythell 2012).

BrB advances over healthy tissue exposing the skeleton, while the infective boundary manifests a cm wide band of ciliates engorged with brown Symbiodiniaceae (Fig 513.; Willis *et al.* 2004; Chong-Seng *et al.* 2011). Most zooxanthellae are brown (Yu 2000) where fish consume ciliates and may thus be vectors or bioremediators (Chong-Seng *et al.* 2011).

The rod-shaped ciliates belong to the class Oligohymenophorea, the subclass Scuticociliatia, and the order Philasterida (Fig 515.; Bourne *et al.* 2008; WoRMS 2020t). BBD, white syndrome, and BrB were surveyed on GBR, and inshore incidences were lessened at locations where line fishing was permitted (Sisney *et al.* 2018); however, unlike other diseases, elevated temperatures do not enhance pathology (Boyett 2006).

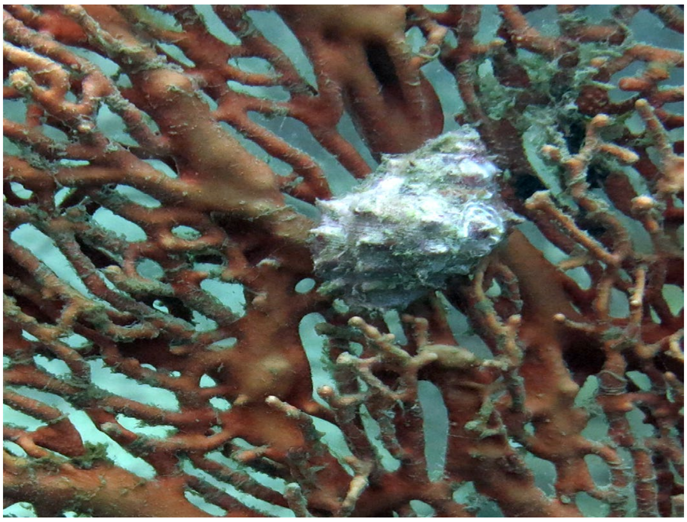


Fig 514. Corallivorous snails of the genus *Drupella* are vectors of brown band disease.

Skeletal Eroding Band (SEB)

The first incidence of a coral disease caused by a sessile ciliated chromist was reported in 2000 (Antonius & Lipscomb 2000). *Halofolliculina corallasia* (Heterotrichea: Folliculinidae) are encased in an excreted black bottle-shaped sheath (lorica) with peristomal wing-like appendages swathed in hair-like cilia akin to flagella, whereas their elongated oral cilia sweep particles into their mouths (Andrews 1914; Antonius 1999).

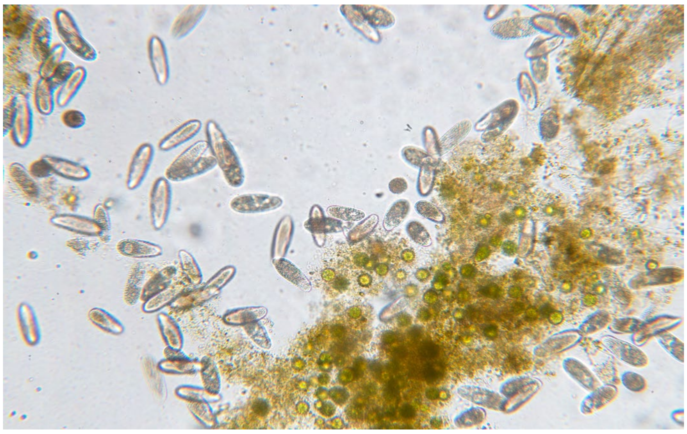


Fig 515. Marine ciliated chromists of the order Tetrahymenida, the class Oligohymenophorea, and the subclass Hymenostomatia surrounding what appears to be cellular debris and coccoid phototrophs. Other Oligohymenophorea affiliates include *Porpostoma guamensis* and *Philaster digitiformis* which are the causative agents of BrB, although they are members of the subclass Scuticociliatia (WoRMS 2020u) and thus scuticociliates.

SEB progresses around 2 mm per day (Page & Willis 2008) where black speckles differentiate it from BBD (Fig 516.; Fig 517.). Several cyanobacteria inhabit lesions yet they do not affect pathology (Antonius & Lipscomb 2000).

Fig 516. A possible example of skeletal eroding band (SEB) of *Siderastrea siderea* consistent with the observations of Borneman from 2002. It appears the band was progressing downward, whereas the active front has since diminished along with the destruction of tissue, and the upper parts of the colony are now inhabited with what appears to be sponge or cyanobacteria. Photography by Dan Mele©.

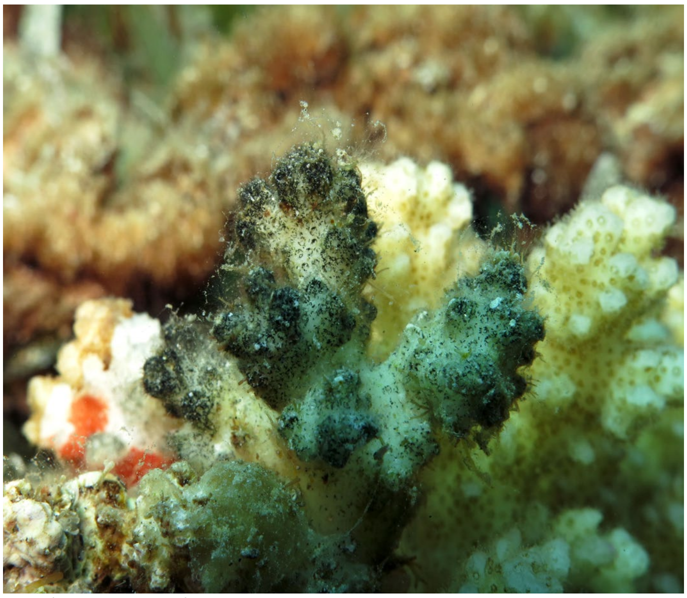


Fig 517. A closeup of what appears to be skeletal eroding band and microalgal invasion of *Pocillopora damicornis*.

Pathology progresses when asexual larvae locate themselves on healthy flesh slightly ahead of an established band, where secretion of loricae throughout larval spinning erodes the tissue and instigates skeletal splintering (Antonius 2000; Antonius & Lipscomb 2000). Branching and massive ancient colonies are affected, yet members of the families Pocilloporidae and Acroporidae are most susceptible (Antonius 1999; Page & Willis 2008). SEB has been reported in the Caribbean, the Pacific, the Red Sea, the Indian Ocean, Hawai'i, on GBR, and in aquaria (Antonius 1999; Antonius & Lipscomb 2000; Palmer & Gates 2010; Adams 2012).

Experiments revealed that *Halofolliculina corallasia* are readily recruited to bare skeleton, yet appear benign to healthy tissue.

The integrity of coral flesh, may therefore, need to be compromised before ciliates can invade (Page & Willis 2008). Numerous corals are sediment-intolerant which is analogous to the deposition of loricae, where the feeding scars of corallivorous snails of the genus *Drupella* may assist colonisation (Page & Willis 2008; Onton *et al.* 2011).

White Syndromes

The term white syndrome is used to describe white band disease, white pox, and rapid tissue necrosis (Bythell *et al.* 2004). White pox and white band disease are somewhat easy to discern because the latter affects only acroporids (Bythell *et al.* 2004). However, exceptions occur. White syndrome has been associated with ciliates, helminths, fungi, algae, sponges, and cyanobacteria (Work & Aeby 2011). Comparative analyses of BrB and white syndrome revealed an almost identical consortium, and researchers postulated they were the same disease (Sweet & Bythell 2012), whereas Nicolet and colleagues asserted they were distinct in 2013.

Fig 518. The Caribbean sea fan, *Pseudoplexaura porosa* is susceptible to algal galls.

The chromists of brown band disease, brown jelly and white syndromes share significant homology which suggests they are caused by *Philaster digitiformis* strain FJ64835 (Sweet *et al.* 2013). Earlier studies of white syndrome revealed biomarkers of preprogrammed cell death (Godar 1996; Ainsworth *et al.* 2007) which is a normal cellular response to an inability to divide (Zhang *et al.* 1999), whereas tissue necrosis is cellular demise resulting from trauma. Hence rapid tissue necrosis and preprogrammed cell death appear identical which can occur without the participation of an infective organism. The corals of Christmas Island have been relentlessly under attack from white syndrome since 1998 (Hobbs & Frisch 2010; Hobbs *et al.* 2015; Preston 2015) where an epizootic occurred in 2008.

Coral and Symbiont Viruses

Heat shocking *Acropora muricata*, *Pavona danai*, and *Stylophora pistillata* yields virus-like particles (Davy *et al.* 2006), while bacteriophages were isolated from corals and symbionts undergoing environmental stress (Correa *et al.* 2016). RNA viruses (retroviruses) infect zooxanthellae (Ho 2012), whilst virion oceanic abundance is in the region of 9×10^8 ml^{-1}. Viral research has the potential to counter reef ecological decline (Wommack & Colwell 2000; Marhaver *et al.* 2008; Garber 2012), where single stranded (ss)DNA viruses were consistently isolated from lesions of white plague (WP; Ho 2013; Soffer *et al.* 2013). Moreover, biomarkers of herpesviruses were found in coral genomes (Garber 2012a; Correa *et al.* 2016).

Fig 519. Brown jelly of torch coral (*Euphyllia glabrescens*). Images courtesy of Aaron Daniels©, Lowestoft, Suffolk, UK.

Host/Parasite Interactions

The following conditions have either been investigated by science or were reported by aquarists, which includes anecdotal evidence with examples of pathogenesis in noncoral hosts.

Algal Galls have been described in Floridian and Caribbean sea fans of the genus *Pseudoplexaura* (Fig 518.). They are symptomatic of a skeletal infection by the microalgae, *Ulvella endozoica* which extract the amino acids tyrosine and cytosine from their host. Sea fans mount a defence by "walling off" the infected area with calciferous sclerites and insoluble structural proteins called scleroproteins. Sclerite recruitment encapsulates the microalgae in galls which become dormant, but this strategy compromises the elasticity and tensile strength of their skeleton. The algae's reproductive cycle commences when skeletal fracture and illumination induce spore production and release, while it concludes when the breaches heal which re-encases the algae (NOAA 2014).

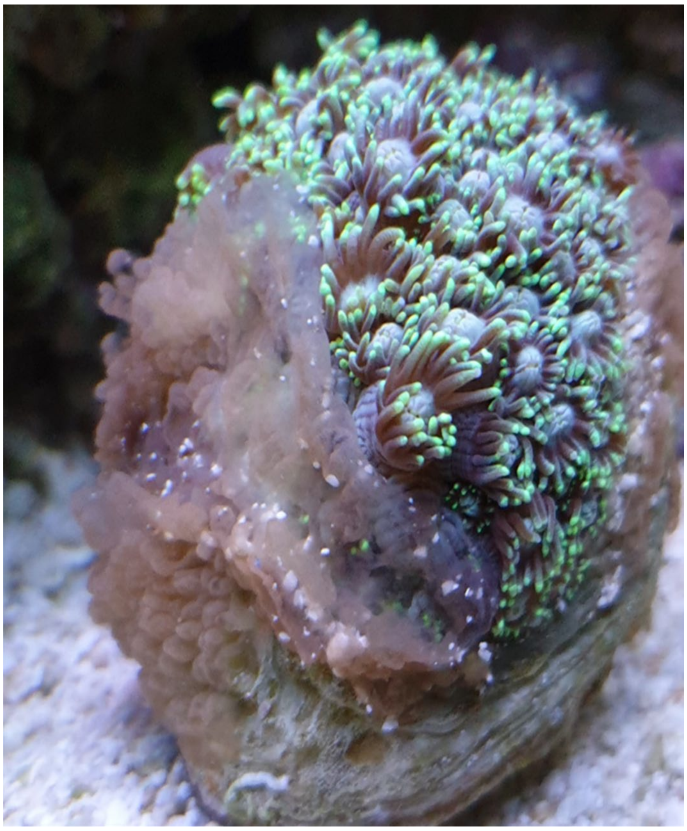


Fig 520. A colony of *Goniopora stokesi* with brown jelly syndrome. Image courtesy of Liam Furlong©, Staffordshire, UK.

With the potential to affect all zooxanthellates, **Brown Jelly Syndrome** was regarded as peculiar to recirculating systems before it was observed on GBR in 2003. Not to be confused with brown stringy exudates from polyp mouths indicative of the winnowing of algal symbionts, the illness appears as a brown jelly-like mass hovering over and enveloping the coral (Fig 519. to Fig 521.). Brightfield microscopy revealed a consortium of bacteria, fungi, and chromists including "zooxanthellae-eating" *Helicostoma notatum*. It was unclear if the chromists were performing housekeeping by clearing cellular debris resulting from apoptosis or whether they were aetiological agents (Borneman 2003).

Necrosis of fleshy LPS corals has occurred in the absence of brown jelly which infers a defined idiopathy, while outbreaks appear sporadic which affect the established and newly introduced. Brown jelly spreads rapidly to neighbouring heads or nearby colonies, and there is a symptomatic smell of putrefaction associated with bacterial sepsis when the coral is removed. The only efficacious strategy is to ruthlessly remove the affected colonies or amputate their diseased heads (Delbeek & Sprung 1994), because syphoning the mass with airline does little to mitigate pathogenesis.

Conflicting Evidence

Researchers analysed brown jelly in the wild and in aquaria and proposed *Helicostoma* was a misnomer (Sweet *et al.* 2012), because genetic analyses of three isolates revealed brown jelly syndrome was induced by the chromists *Philaster digitiformis* strain FJ64835. The ciliates were present in aquaria a week after setup and were lacking in healthy specimens (Sweet *et al.* 2013).

Diseased Sponges

The bacteria *Pseudoalteromonas agarivorans* were isolated from ailing sponges, *Rhopaloeides odorabile*, whose numerous virulence determinants included secretion systems (Choudhury *et al.* 2015). Reports of diseased sponges are comparatively recent, which likely reflects a shift in the holobiont's community compositions and deliberate transitory states of dysbiosis (Pita *et al.* 2018; Munn 2019; Boilard *et al.* 2020).

Fig 521. Brown jelly syndrome of *Fimbriaphyllia ancora*. Image courtesy of Tom Rudge©, Redditch, Worcestershire. www.instagram.com/toms_reef/

Fig 522. Left - a moribund example of *Gorgonia ventalina* which may have been initially infected with red band disease that stripped away the tissue from beneath. Right - filaments of red cyanobacteria. The purpling may be a response to the red band disease, or it may be a legacy of aspergillosis, whereas the lower skeleton is overgrown by a contrasting species. Photography courtesy of Dan Mele©.

Diseases of Echinodermata

An epizootic of the long-spined black sea urchin (*Diadema antillarum*) occurred in Panama in 1983, which rapidly disseminated throughout the wider Caribbean and tropical Atlantic. The reason for their demise remains enigmatic, which were efficient grazers of macroalgae that now monopolise Caribbean reefs (Munn 2019).

In British Columbia in 2013, sea stars of the class Asteroidea were routinely washed ashore degrading with missing limbs. The illness was named sea star wasting disease which manifested along the Pacific coast from Alaska to southern California. The pathology was hastened by the elevated temperatures of 2014 and 2015 where predatory ochre stars (*Pisaster ochraceus*) were one of the worst affected (Munn 2019). Their diet comprised mussels, barnacles, limpets, and snails where their demise initiated a profound shift in nearshore ecosystems (Sanford & Menge 2007; Munn 2019). Suspensions of flesh from diseased sea stars reproduced the same illness when healthy specimens were exposed, whereas virions were suspected but were isolated from asymptomatic starfish. All findings remain inconclusive (Munn 2019).

Coral Microbiomes: The Holobiont

Microbiome refers to all the microorganisms inhabiting an anatomical niche, on and within, another form of life like humans, fish, invertebrates, and corals, which offer several distinct environments that differ in pH, salinity, irradiance, nutrition, temperature, and the availability of oxygen. Similarly, the holobiont encompasses all such anatomies including the host, whose microenvironments are probably impacted by the external circumstances to which they are exposed. The communities that reside on and within Scleractinia, are conceivably distinct and bespoke to colony and reef zone. Yet remarkably, core members occur in several if not all genera (Hernandez-Agreda *et al.* 2018; Munn 2019). Populations of microbes with comparable profiles were isolated from equivalent microbiomes within the same genus living within corresponding conditions, while their core partners undertook roles that significantly advantaged their host (Ainsworth *et al.* 2015; van de Water *et al.* 2016; Hernandez-Agreda *et al.* 2017; Munn 2019). The findings of one study concluded that excluding a mere fraction, the microbial communities on and within corals are diverse, transient, and attuned to the prevailing conditions (Hernandez-Agreda *et al.* 2018). However, metagenomic plasticity and functional redundancy are likely pervasive, where optimal efficacy remains in the face of the loss of nominal taxa (Pollock *et al.* 2019).

Holobionts comprise exceedingly diverse assortments of microorganisms like fungi, viruses, bacteriophages, bacteria, protozoa, chromists, and obligate anaerobes like Archaea (Suchodolski 2012; Munn 2019). The coral secretes nutrition in the form of carbohydrates and proteins which are readily degraded by the extracellular products of each microbiome, which likely reciprocate with co-factors, vitamins, and macronutrients. Manipulation of the biochemicals supplied to each microbiome is likely one means by which corals influence the community compositions of their micro-symbionts and -commensals (Ritchie & Smith 1995). The sponge loop recycles trace elements within coral mucus (DOM) when they transform them to POM, whereafter they are biomineralised by microbes and absorbed by phytoplankton. Hence corals contribute to organic and inorganic C, N, and P within nutritionally-poor oligotrophic milieus (Eppley & Peterson 1979; Munn 2019).

Microbiome Diversity: Deterioration

Varied mucosal communities are vital for colony defence. Shifts in community composition likely reflect dysbiosis, where the host purges microbes in response to dysfunction with an aim of restorative recruitment (Rosenberg *et al.* 2007; Munn 2019; Boilard *et al.* 2020). Hence probiotic therapies can elevate function and restore health (Rosado *et al.* 2018; Munn 2019; Santoro *et al.* 2021).

The paradigms of microbiome flexibility, probiotics, and beneficial microorganisms for corals (BMCs) propose that the plasticity of a microbiome differs according to holobiont lineage; coral-dwelling microorganisms are tailored to suit the prevailing conditions yet remain disposed to probiotic manipulation, and endurance is conferred by symbionts and commensals like the consortium of the mucosal microbiome. Microbial inoculums may therefore mitigate bleaching and disease (Reshef *et al.* 2006; Peixoto *et al.* 2017; Rosado *et al.* 2018; Munn 2019; Voolstra & Ziegler 2020; Santoro *et al.* 2021). The beneficial attributes of microbiota likely include sulphur cycling and detoxification, the manufacture of antioxidants and bacteriocins, nitrogen fixation and cycling, and quorum quenching (Giambiagi-Marval *et al.* 1990; Kalia 2013; Peixoto *et al.* 2017; Lavy *et al.* 2018; NCBI 2019k; Robbins *et al.* 2019; Wirth *et al.* 2020; Maire & van Oppen 2022).

Saltwater microbes derive much carbon and sulphur from the abundant marine compound, dimethylsulphoniopropionate (DMSP), where most metabolise it differently (Wirth *et al.* 2020). Zooxanthellae harbour latent viruses that undergo lytic induction during stress, insofar as corals subjected to nutrient enrichment or increased temperature suffer upsurges of herpes-like virions. Hence bleaching is promoted by temperate viruses (Munn 2019).

Coral Immunity

Transcriptome analyses reveal diseased corals modify the expression of hundreds if not thousands of genes (Traylor-Knowles *et al.* 2021) many of which encode for antioxidants (Jin *et al.* 2016). Immunogenetic products include melanin, collagens, peroxidases, bax-like and fibrinogen-like proteins, transforming growth factor beta (TGF-β), and tyrosine kinases (Traylor-Knowles *et al.* 2021) that transfer the terminal phosphate of ATP to the tyrosine residues of proteins which are vital for intercellular communication and tissue maintenance (Hubbard & Till 2000).

TGF-β are multifunctional signalling cytokines (Chaudhury & Howe 2009) whose expression appears central to the infection of planulae with zooxanthellae. Disrupting the transcription of their mRNA prohibits the recruitment of algal partners and elicits the generation of the cytotoxic immune factor, nitric oxide (Berthelier *et al.* 2017). Inhibition of the TGF-β pathway appears vital for competent immunity (Fuess *et al.* 2020).

Holobiont Metabolism

The metabolomes of coral holobionts likely include the by-products of photosynthesis, vitamins, co-factors, ligands, sulphur, iron, nitrogen, as well as derivatives of nucleic acids, proteins, and polysaccharides. Although their expression is easily perturbed, gene products that modulate quorum sensing and resilience to stress like those associated with DNA repair and antimicrobials are common. Inhabitants include significant cyanobacteria that fix DN_2 to PON, DON, and DIN and γ-Proteobacteria like species of *Vibrio*. Coral holobionts rely upon the proficient metabolism of nitrogen to subsist in nutrient-poor reef ecosystems, whereas the cycling of sulphur and iron and denitrification likely occur in suboxic/marginally-anaerobic deep endolithic microbiomes comprising Archaea (Munn 2019) hitherto classified as archaebacteria.

Fungi including endoliths akin to terrestrial moulds are found in mucus and deep-within skeletal matrices; and disseminated throughout the holobiont are viruses and bacteriophages which are the most abundant. Lytic ‘phage modulate bacterial turnover and composition, whereas lysogeny integrates the genomes of prophage whose expression alters the metabolism and virulence of their host. Heat and chemical stress elicit the lytic cycle in temperate ‘phage that perturbs the composition of microbiome communities by initiating dysbiosis, which may or may not expedite disease (Howard-Varona *et al.* 2017; Munn 2019). Induction of this kind is thus central to the liberation and recycling of nutrients within oligotrophic reef ecosystems (Munn 2019).

Clams

Photosynthetic "giant" clams of the genus *Tridacna* are adapted to nurture and channel irradiance to their symbionts, and excluding the filter feeding of phyto- and microzoo-plankton, their algal partners donate all their carbon in the form of small organic and inorganic molecules (Munn 2019).

Discussion

Naturally, initial scientific investigations focused on identifying definitive biotic agents responsible for each coral ailment, where the exploits of one strain or consortium are responsible for numerous human diseases. Compromised defences permit pathogens to acquire a foothold in an otherwise resistant host, whilst the microbes of coral holobionts appear decisive in the arbitration of immunity (Pantos *et al.* 2003; Garber 2011a; Ho 2013; Soffer *et al.* 2013; Rivera-Ortega & Thomé 2018). Moreover, the populations of nanoplanktonic microbes in aquaria, fluctuate significantly over a matter of hours (Sweet *et al.* 2017), which is likely to be the primary reserve from which holobionts recruit (Brown *et al.* 2023).

Elevated temperatures weaken the coral animal and may damage the photosynthetic apparatus of zooxanthellae (Gardner *et al.* 2017), induce the lytic cycle in prophage (Howard-Varona *et al.* 2017; Munn 2019), and augment the virulence of pathogens (Kuta & Richardson 1996; Ben-Haim *et al.* 1999; Banin *et al.* 2000; Banin *et al.* 2001; Israely *et al.* 2001; Ben-Haim & Rosenberg 2002; Oakey *et al.* 2002; Ben-Haim *et al.* 2003; Voss & Richardson 2006; Wang *et al.* 2008; Vega Thurber *et al.* 2014). Host (Gardner *et al.* 2017), and to a lesser extent zooxanthellar metabolism (Nielsen *et al.* 2018) generate reactive oxygen species (ROS) which accumulate in coral flesh whose cells robustly induct antioxidant pathways (Gardner *et al.* 2017). The shielding communities of the mucosal microbiome are destabilised, which provokes adaptive and remedial shifts in their metabolites and community compositions known as metagenomic plasticity (Boilard *et al.* 2020). Abiotic insult that exceeds their restorative potential, demands participation of immunity. The coral instigates intended transitory states of microbial purging and dysbiosis (Oakey & Owens 2000; Wommack & Colwell 2000; Oakey *et al.* 2002; Davy *et al.* 2006; Marhaver *et al.* 2008; Garber 2012; Ho 2012; Correa *et al.* 2016; Bass *et al.* 2019; Boilard *et al.* 2020), to an end of recruiting and re-establishing a wealth of exceedingly-diverse and functionally-redundant populations of microbial commensals and mutualists (Fautin & Buddemeier 2004; Gardner *et al.* 2017; Palmer 2018; Munn 2019). Perceptible bleaching represents an acute example of this kind of microbial purge (Boilard *et al.* 2020).

Eutrophy destabilises mucosal and zooxanthellar communities and impairs coral defence (Cruz-Piñón *et al.* 2003; Ho 2013a; Vega Thurber *et al.* 2014). Enrichment of nitrate elicits zooxanthellar overproliferation (Houlbrèque & Ferrier-Pagès 2009; Angelo & Wiedenmann 2014) which predisposes corals to a loss of pigment, whereas the exchange of nourishment between the animal and its algal partners is disrupted, and cross-holobiont nutrients become scarce (Balling *et al.* 2008; Cunning & Baker 2013). Transient elevated nitrogen may mitigate ROS, symbiont loss, and bleaching (Béraud *et al.* 2013; Ferrier-Pagès *et al.* 2016). Elevated phosphorus expedites skeletal deposition at the expense of strength and abnormalities (Cruz-Piñón *et al.* 2003; Dunn *et al.* 2012; Ferrier-Pagès *et al.* 2016), or slows accretion and compromises integrity (Ezzat *et al.* 2015).

The scientific hypotheses that underpin experimentation must exclude all variables bar one, and the extensive and ubiquitous nature of marine micro- and macro-biota introduce an inordinate number of variables that confound scientific enquiry. A shortage of ecological niches is the catalyst for change, because space and nutrition are swiftly utilised. A paradigm has emerged over the last decade and a half that describes the sequence of events that lead to dysbiosis and the suppression of defence (Pantos *et al.* 2003; Vattakeven *et al.* 2006; Tait *et al.* 2010; Garber 2011a; Ho 2013; Soffer *et al.* 2013; Rivera-Ortega & Thomé 2018).

Soft tissue trauma, apoptosis, and induction of temperate 'phage liberate cellular debris and in- and -organic molecules that are exploited by numerous nearby microbes (Oakey & Owens 2000; Oakey *et al.* 2002; Lesser *et al.* 2007; Hall *et al.* 2018; Morrow *et al.* 2018), yet their presence and proximity remain weak indicators of pathogenicity.

Probiotics applied to the mucosal microbiomes of heat stressed corals restructured their microbial communities which instigated genetic and metabolic alterations that ameliorated bleaching and mortality. Beneficial microbes for corals (BMCs) applied to thermally stressed hermatypes elicited holobiont reorganisation and the modification of host gene expression that favoured recuperative cellular repair, immunity, and thermal protection. Furthermore, the revitalising bacterial consortium proved a biocide for *Vibrio coralliilyticus* strain V1 and *Vibrio alginolyticus* V2 (Santoro *et al.* 2021).

BMC isolates were sourced from healthy corals, yet remarkably, the mucosal consortia of the probiotic-treated colonies did not reflect the profile of the inoculum, whilst the communities of non-heat stressed corals did not alter when exposed. However, the transcriptome of each host coordinated with the community composition of their mucosal microbiome, which inferred immunity and the holobiont communicate via receptors and molecular exchange (Santoro *et al.* 2021). These findings lend credence to crosstalk and hologenomics (Rosenberg & Zilber-Rosenberg 2018), whereas recuperation coincided with the reversion of each mucosal microbiome to its pre-stressed consortium (Santoro *et al.* 2021).

Preventative Measures and Treatments

Immunity is Nature's way of ensuring an organism thrives which is usually compromised by stress (Tort 2011). There is no substitute for optimal design and exceptional husbandry, where sustaining the stability of parameters commensurate with this volume promotes animal health.

Wild efficacious approaches included chiselling to create a skeletal "firebreak", sealing lesions with two-part reef epoxy, antibiotics, and virulent bacteriophages (Borneman 2002; Aslett 2003; Garber 2011a; Atad *et al.* 2012; Garber 2012; Randall *et al.* 2018). Skeletal grooves may be helpful for arresting progression on massive boulders, but chisels haphazardly fragment hobby-sized specimens. We explored the dangers of two-part epoxy earlier in this volume, and disease outbreaks are customarily the result of stress, and thus epoxy will exacerbate rather than ameliorate. Reef putties contain bisphenol A which is a polychlorinated phenolic that negatively affects the quality of sperm in men (Pyrek 2016; Vitku *et al.* 2016) which initiates lethality in aquatic ecosystems at less than 2 mg l^{-1} (ppm) after 96 hours (Alexander *et al.* 1998). However, all hard-cured epoxy products remain significantly inert. See: System Maintenance, Two-Part Reef Epoxy: Precautionary Notes.

Apart from brown jelly syndrome and brown band disease, prevention is simply a matter of keeping the temperature stable and below 25°C (Kuta & Richardson 1996; Voss & Richardson 2006; Vega Thurber *et al.* 2014) which is challenging without air conditioning or a chiller. Here we sanction evaporative cooling in a microbe-free trickle tower, yet harmful fluctuations are "ironed out" by increasing warmth above foreseen ambientes. The temperature of choice must be below 29°C, and preferably sub-27. All the same, decrease the hours of illumination on sweltering days (Fitt & Warner 1995).

Fig 523. Irrigation of an infected lesion.

We must be unwavering custodians of water quality, therefore, promptly remove rotting and degrading specimens. Coral fragmentation is one technique used to save partial colonies (Borneman 2002) which often stresses the remainder and induces loss, whereas doing nothing enriches N and P which affects all the inhabitants. The coral can be removed and placed in quarantine, which is less robust and low in nitrogen-cycling microbes. Hence the coral invariably dies which annihilates water quality, and thus, ruthlessly discard decomposing animals.

Corals with microbial infections may be immersed in a US gallon (3.79 litres) of aged aquarium water dosed with 2 ml of Lugol's iodine for 10 to 15 minutes, which is initially minimally stressful, yet it may render corals defenceless by partially sterilising their mucosal microbiome (Oakey & Owens 2000; Oakey *et al.* 2002; Lesser *et al.* 2007; Hall *et al.* 2018; Morrow *et al.* 2018). Akin to human gut microflora, "good" bacteria are essential for health, immunity, and wellbeing (Isolauri 2001), whereas Lugol's iodine also contains non-sterilising and beneficial iodide.

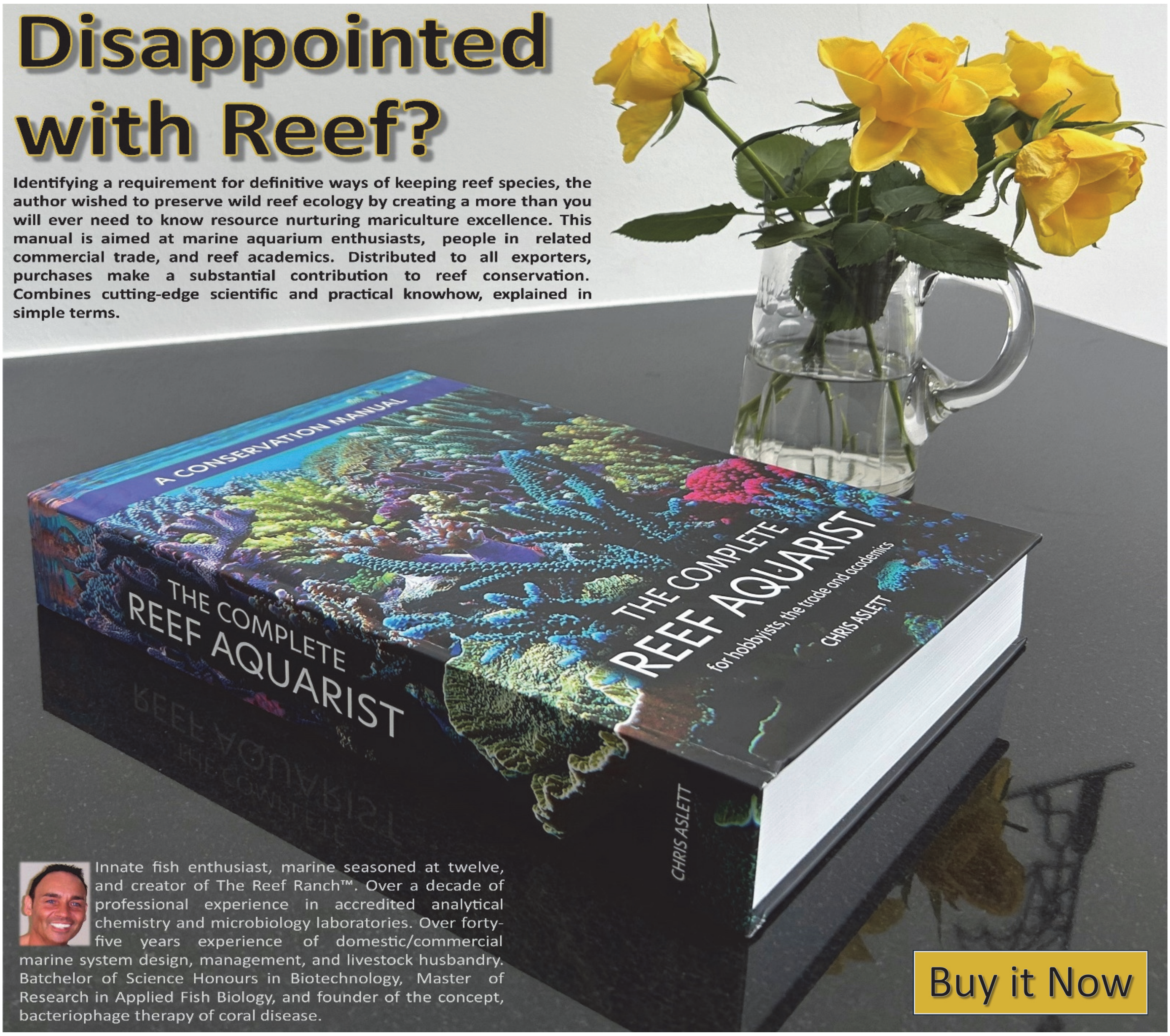
Freshwater kills marine bacteria via osmotic lysis and dislodges macroscopic parasites, where administering freshwater to a coral lesion irrigates and treats only the infected area and surrounding tissue. The remainder of the colony is only suspended out of water for two minutes, and corals are routinely exposed to air on spring tides (Fadlallah *et al.* 1995).

The irrigation technique uses a disposable Pasteur pipette and two small buckets of warm pre-aerated reverse osmosis (R/O) water, where 2 ml US gal^{-1} (3.79 l^{-1}) of Lugol's iodine is administered to one. The colony is removed from the aquarium and held with the infected area facing downward over a bucket containing the solution of iodine, and the lesion and surrounding tissue are continuously irrigated for 140 seconds using the Pasteur pipette. The infected area is then rinsed for a further 20 seconds in pure freshwater to purge molecular iodine, and the coral is returned to the system (Fig 523.).

Beneficial Microbes for the Mucosal Microbiome

The author foresees several preparations claiming to be beneficial microbes for corals (BMCs). Samples from healthy live specimens were incubated on agar, purified, and characterised (Santoro *et al.* 2021), where incubated Petri dishes and bottled environments are very different. Hence proprietary probiotic inoculums exhibiting analogous efficacies to those in coral research likely remain out of reach, yet warrant further investigation.

Ocean Alchemist supply the wave action resistant ointment **CoralCure™ base2B** which is combined with antibiotics to mitigate disease in wild corals. It is administered directly to the boundary of infection or impregnated into rope (Gay 2021; Eaton *et al.* 2022). However, it is *not* intended for use in aquaria because antibiotics negatively impact beneficial microbes including biofilter consortia.



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