Chris Aslett, BSc (Hons) MRes

Chris is a Master of Research in Applied Fish Biology graduate with over 20 years experience of keeping marines. He has recently opened a marine aquarium store in Leeds – *The Reef Ranch*.

What the scientists know:

Coral disease – a cause or coincidence

In my last article I described how some land-based microorganisms had been introduced into the marine environment and had adapted to cause disease in corals. I explained how a temperature of 27°C (80.6°F) or more weakens corals and favours coral ¥pathogens. This article consolidates the knowledge of coral diseases amassed by the scientific community over the past 3 decades.

There have been many instances where a change in the environment has led to coral invasion and mortality. Antonius and Ballestros in 1998 surveyed the reefs at Carrie Bow Cay, Belize and Key Largo, Florida. They found incidences of stony coral overgrowth with various species of cyanobacteria. In several cases the skeleton was bare and in some instances eroded[2]. It must be emphasized that coral invasion of this type is not a coral disease, but merely an example of how a change in environment can favour the predominance of one organism at the expense of another[17]. The coral diseases that presently concern scientists have recognised and defined *aetiologies.

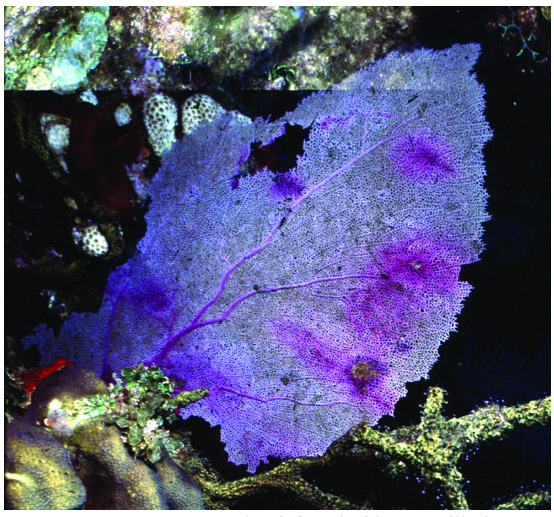
Confirming that a particular microorganism causes a disease is more difficult than one might first imagine. The repeated isolation of a microorganism from a disease **lesion is insufficient evidence to prove that the microorganism is the cause of disease. Scientifically speaking the microorganism has to satisfy Koch's Postulates.

In 1890 Robert Koch a German scientist, recognised that a microorganism must fulfil certain sequential criteria before it can be proven to be the cause of a disease. It must be consistently isolated from a disease lesion, be cultured in a pure form in the laboratory, manifest the same disease lesion when a new ·host is exposed to this culture and be re-isolated from the resultant lesion.

Some coral disease-causing microorganisms have fulfilled Koch's Postulates, but some are unable to be

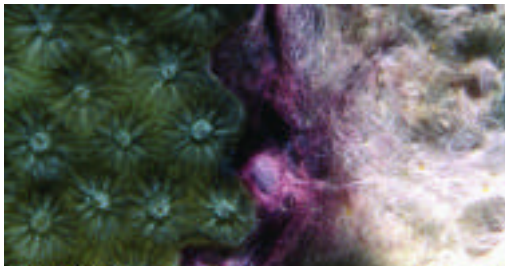
Table 1 Extensively characterised coral diseases

Disease Abbreviation	Causative Agent	Host	References
Aspergillosis	*Aspergillus sydowii*	*Gorgonia* species including *G. ventalina; G. flabellum*	1, 10, 19, 20, 21, 22, 29, 35, 35
Black Band disease, BBD	A microbial consortium including *Phormidium corallyticum*	Hard corals including *Montastrea annularis, M. cavernosa, Diploria strigosa, Porites asteroidies*	9, 13, 14, 15, 24, 35
Bacterial bleaching	*Vibrio shiloi & V. coralliilyticus*	*Oculina patagonica Pocillopora damicornis*	3, 4, 6, 7, 18, 37, 38
Coral Plague CP	A *Sphingomonad*	*Diochocoenia stokesi; Colpophyllia* species; *Mycetophyllia* species	11, 17, 30, 31, 32
White Band	Undetermined possibly, *Vibrio harveyi*	*Acropora* species including *A. palmata; A. cervicornis*	12, 17, 26, 27, 30, 33
White Pox, WP	*Serratia marcescens*	*Acropora* species	16, 17, 25, 28



ANDY BRUCKNER, 1996

Fig. 1 *Gorgonia ventalina* infected with Aspergillosis



ANDY BRUCKNER, 1991

Fig. 2 A Black Band disease infection of *Montastrea annularis*

***aetology:** The cause or origin of a disease as determined by scientific analysis. ****lesion:** A wound or injury. **¥pathogen:** An agent that causes disease, such as a bacterium, virus, or fungus. www.dictionary.com. **·host:** An animal or plant on which or in which another organism lives.

ANDY BRUCKNER, 1997

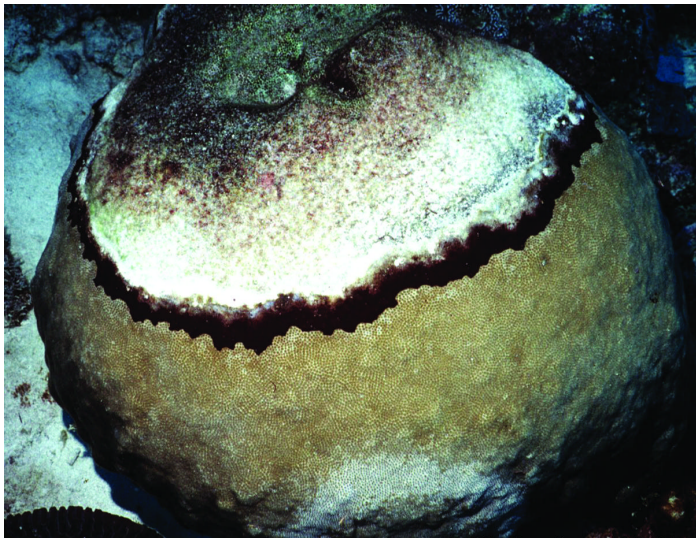


Fig 3. A Black Band disease infection of *Stephanocoenia intersepta*

cultured in the laboratory. Such microorganisms include bacteria of the genera Vibrio that are able to enter a dormant state called viable but non-culturable (VBNC). However advancements in DNA technology have enabled even VBNC microorganisms to be recognised as pathogens.

A number of coral abnormalities have been provisionally categorized as coral diseases, but their cause is yet to be determined. Only the diseases that have been extensively researched and characterised will be included in this article (**Table 1** – page 38).

Aspergillosis

has caused widespread mortality amongst sea fans throughout the Caribbean[36]. The causative agent is *Aspergillus sydowii* a terrestrial fungus[35]. The progression of disease is most rapid at 30°C and *Gorgonia ventalina* is more susceptible than *G. flabellum*[1, 36]. The fungus is deposited on the reef by storms and once established, it can be spread via the water column or by coral-to-coral contact[19, 35]. The disease denudes and erodes the coral skeleton. The gorgonians mount defence mechanisms against the pathogen by increasing the melanin, ‡sclerite and antifungal-chemical content of their cells. The sclerites become so dense, that they cause galling and purpling of the coral tissue (**Fig 1**). The infection is most prevalent in mature specimens and leads to the suppression of reproduction.

As a result gorgonians die and are not replaced by their young. At one time more than 50% of sea fans were lost from the Florida Keys. Aspergillosis in the Keys has since declined because the presence of the disease locally, stimulates the young produced to be more resistant to fungal infections[10, 20, 21, 22, 29].

Black Band disease

occurs when a mixed population of microorganisms form a microbial mat and act in unison to destroy coral tissue. Traditionally the mat consists of the cyanobacteria *Phormidium corallyticum* and bacteria of the genera *Cytophaga*, *Beggiatoa* and *Desulphovibrio*[9, 15]. The infection starts as a focal lesion and radiates concentrically outward forming a band that leaves a bare skeleton in its wake. The band can be several mm wide and migrates over its host at a rate of over a cm per day (**Fig 3**). As the name suggests the band is black to dark red in colouration (**Fig 2**). The microbial community that makes up BBD is typical of the microorganisms found in land runoff and sewage discharge[14]. BBD now infects corals worldwide[13]. During the day the upper layers of the band become supersaturated with oxygen, whereas the lower regions are anoxic and rich in hydrogen sulphide[9]. Bubbles can be seen adhering to the mat and if the infected coral is removed momentarily from the aquarium, there will be a strong smell of hydrogen sulphide. The sulphide is produced below the mat and is the cause of tissue destruction[34]. BBD is capable of infecting a wide range of hard corals but *Montastrea annularis*, *M. cavernosa*, *Diploria strigosa* and

ANDY BRUCKNER, 1993

Fig. 5 A fireworm (*Hermodice caranculata*) predation

PHOTO COURTESY OF NOAA's CHAMP

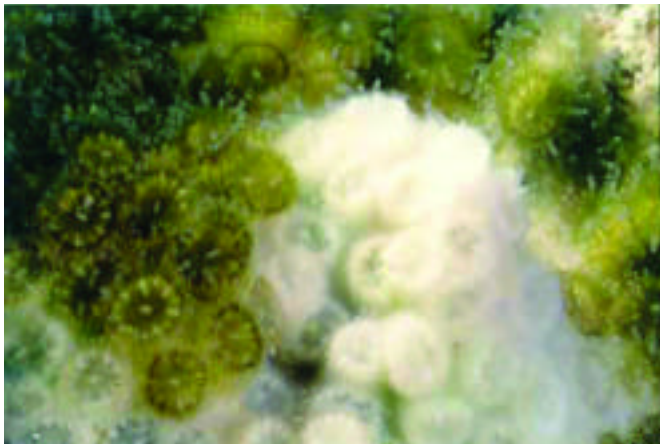


Fig 4. A *V. shiloi* infection of *O. patagonica*

‡ **sclerites:** Microscopic structures like fibres of various shape, mineralized with carbonates. Combined together in various ways, they make up a skeleton in the soft tissues of many marine animals like gorgonians and soft corals. www.dictionary.com

Porites asteroidies are most susceptible[14, 15]. BBD only infects corals when the temperature of the water column exceeds 25°C[24].

Bacterial mediated bleaching

Coral bleaching caused by the bacteria *V. shiloi* and *V. coralliilyticus* has been described in *Oculina patagonica* and *Pocillopora damicornis* respectively (**Fig 4**). *V. shiloi* produces a protein fragment that in the presence of ammonia, stops zooxanthellae from photosynthesising. The ammonia is produced when the bacterium breaks down coral tissue[5]. *V. shiloi* also releases a substance that causes zooxanthellae to be displaced from the coral, and is able to adhere to the mucus of a photosynthesising host[3, 4, 5, 38].

Researchers hypothesized that a lack of *V. shiloi* in the water column surrounding infected corals, implicated a vector for transmission[3]. Further research proved that *V. shiloi* infects corals during the summer months, but when sea temperatures subside in autumn and winter, the bacteria are either expelled or killed. It was proposed that a third-party organism must be responsible for re-infecting the corals in the following summer[18].

The reservoir host was finally identified as *Hermodice caranculata*, the marine fireworm. In winter fireworms were collected from the Mediterranean and were found to be harbouring high numbers of VBNC *V. shiloi* just under the surface of their skin.

The laboratory experiments demonstrated that all corals exposed to the worms nocturnal feeding habits were infected and bleached within six weeks (**Fig 5**)[37].

V. coralliilyticus instigates rapid bleaching of *P. damicornis* and is spread via the water column or by coral-to-coral contact. The bacterium can infect corals at temperatures exceeding 25°C but is at its most †virulent at 29°C. *V. coralliilyticus* is tolerant of salt (NaCl) concentrations up to 7% but 8% NaCl will inhibit its growth. Intriguingly during the early stages of infection, no *V. coralliilyticus* can be detected in the water column[6]. The bacterium infects only the zooxanthellae and it is an enzyme released by the bacterium that destroys the coral tissue[7].

Coral plague

As early as 1977 Phillip Dustan reported a sighting of CP in the Florida Keys[11]. CP is now common throughout the Caribbean. Types I, II and III exist that range from the least to the most virulent respectively. CP infects *Colpophyllia*, *Mycetophyllia* and *Dichocoenia stokesi*. The latter is the most susceptible to the disease, that spreads from the base upwards (**Fig 6**)[17, 30, 31, 32]. The boundary of infection is discretely flanked by healthy coral and bare skeleton indicating that either enzymes or toxins are responsible for the destruction of tissue. A previously unrecognised bacterium of the genus *Sphingomonas* was isolated from a CP lesion and has since satisfied Koch's Postulates[30, 32].

†**virulence:** The capacity of a microorganism to cause disease. www.dictionary.com

ANDY BRUCKNER, 1994

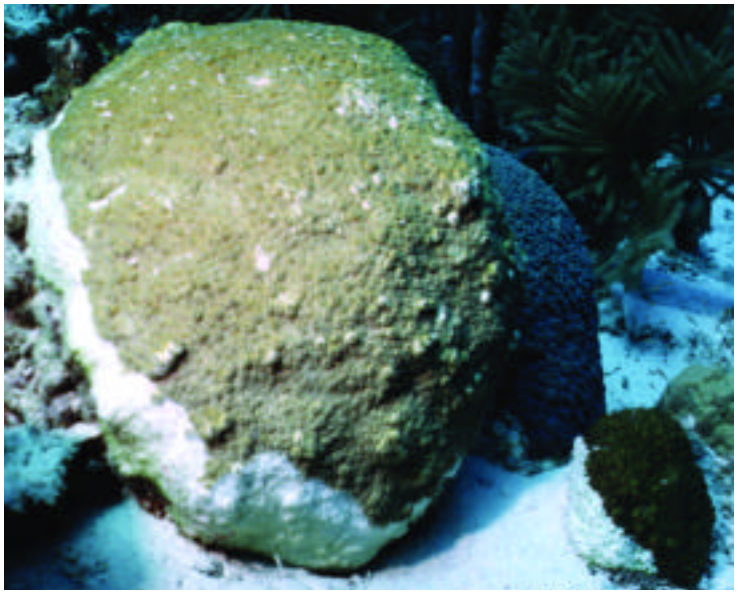


Fig 6. Coral Plague infections of *Dichocoenia stokesii* (left) and *Montastrea cavernosa* (right)

ANDY BRUCKNER, 1994

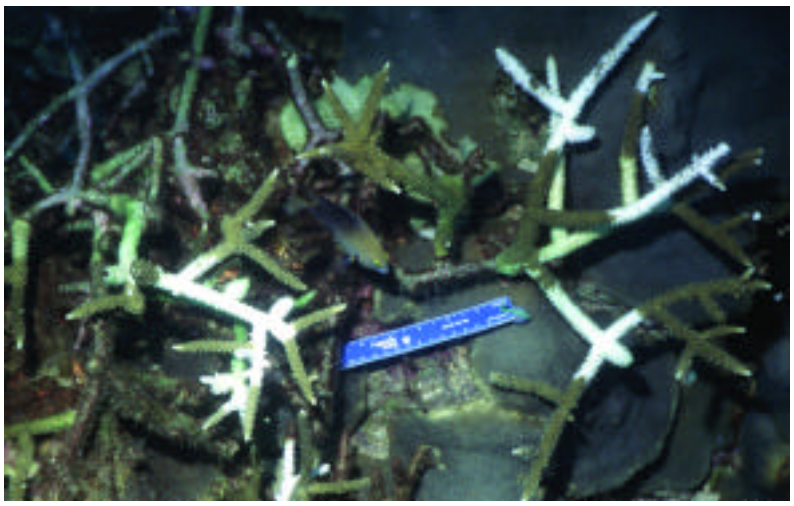


Fig 7. *Acropora cervicornis* infected with White Band disease type II

ANDY BRUCKNER, 2000

Fig 8. A White Pox infection of *Acropora palmata*

White Band disease

infects corals in the Caribbean including the Bahamas and the Florida Keys. All acroporids are vulnerable but WBD is commonest in *Acropora palmata* and *A. cervicornis*. Types I and II exist. A type I infection denudes the skeleton from the base to the tips, but a type II infection either starts at the tips and progresses downward to the base, or occurs mid-branch in *A. cervicornis* (**Fig 7**)[17, 26, 33]. In type II infections the margin between ††necrotic and healthy tissue is indistinct, because the tissue is bleached prior to destruction[30]. Despite *V. harveyi's* capacity to cause disease in the tiger prawn (*Penaeus esculentus*), the spiny lobster (*Panuliris homarus*) and the pearl oyster (*Pintata maximus*), and the bacterium's consistent presence in WBD lesions, the cause of WBD remains uns ubstantiated[12, 27].

White pox

The bacterium *Serratia marcescens* was proven to be the causative agent of WP by the utilisation of DNAtechnology (**Fig 8**)[16, 28].

During a WP infection irregular shaped patches of tissue are lost from the skeleton. *Acropora* species are most at risk, particularly *A. palmata*[17]. *S. marcescens* is carried by humans in their gut and has a propensity to adhere to coral mucus[25, 28].

In conclusion

It is our duty as conservation minded marine hobbyists to use the above knowledge to help prevent the needless loss of diseased corals in our aquaria. Nevertheless as a marine hobbyist of many years I realise that a disruptive treatment such as a coral dip in the absence of a proper diagnosis, could prove detrimental. If a specimen is looking "off colour" or even suffering from tissue loss, it may simply need relocating to a more suitable place in the aquarium. It is surprising what results can be achieved by improving and/or maintaining water quality, and providing the correct lighting.

From my articles to date it should be apparent that one parameter that will affect coral health and contribute to disease is temperature. One should endeavour to keep aquarium temperatures stable and below 77°F (25°C). I prefer to keep the temperature of my aquaria between 70°F-72°F (21°C-22°C). Naturally any parameter adjustments that are made to an established aquarium should be made gradually. For those of you without chillers, I will outline disease treatments in a later article before the summer arrives. Next time I will describe the not so well characterised coral abnormalities, often described using the all-embracing term coral disease.

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