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Host Parasite Interactions, Coral Disorders and Scientific Unknowns

My previous article contained a detailed description of the extensively researched and characterised coral diseases. I explained that scientifically, to qualify for the classification of disease the disease-causing agent must satisfy Koch's Postulates. This article features some coral aliments that have been discovered in recent years, the majority of which have no known cause.

The coral maladies featured below have all, at one time, been described using the all embracing term "coral disease" and in the most part, in the absence of scientific analysis. Numerous sightings have been made of "coral diseases" in the wild. The discovery of a denuded coral skeleton often prompts the diver to take a closer look. This sighting is invariably logged as the discovery of yet another coral disease. Such a conclusion was made when observing parrotfish predation (**Fig 1**). The condition was given the name Rapid Wasting Disease. Scientific investigations were made into the phenomena that appeared to be caused by a fungus that the parrotfish were distributing[1]. Scientific analysis has since proven that there is no microbial agent involved. The "disease" was simply the result of parrotfish browsing and the fungus was utilising available space on the reef[5].

Monitoring coral disease in the wild is a difficult task because divers are limited by the time they can spend submerged. Therefore in some ways the hobbyist has a superior vantage point. However the observation of a "coral disease" does not tell us anything about the cause, just the symptoms. Any determination of disease causation must be performed under experimental conditions.

Experimental conditions are designed to eliminate all factors that may influence the results of the experiment, save what in theory should occur. The experimenter must first postulate a hypothesis. The experiment is then designed to prove or disprove this hypothesis. The logistics of designing an experiment to determine what is causing a coral disease is a very complex task. Experiments in closed systems such as aquaria are not ideal because no matter how the specimen is housed, it is impossible to determine if these conditions are what the specimen is accustomed to in nature. Any deviation from wild conditions could weaken the coral and thus bias any experimental data acquired.

Fig 2. Hyperplastic growth of *Diploria strigosa* – photograph by Andy Bruckner, 1996

Ideally the experiment must house specimens under the conditions they have evolved to suit. This includes natural seawater, wave action, depth of placement and nutrition. For hermatypic corals that contain zooxanthellae, nutrition is customarily in the form of light. All factors that could influence the experimental data must be eliminated. Even for a scientist with a research grant, the eradication of all the potential influencing factors is near to impossible, so please don't try this at home!

Genuine coral diseases have been observed in aquaria but their occurrence is relatively rare. It is more conventional for a coral to die because it is kept in an environment that is unsuitable for that species. For the reasons stated above,

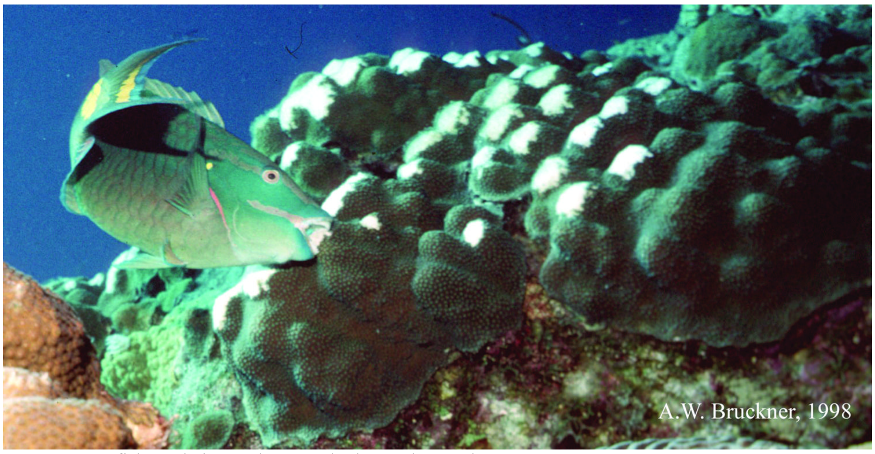

Fig 1. Parrotfish predation – photography by Andy Bruckner, 1998

until scientific research has determined a cause for many of the coral maladies described below, it is not possible to conclude if the observations made by scientists and hobbyist alike, are the symptoms of authentic coral diseases.

Skeletal abnormalities

have been observed in hard corals and lead to a distortion of the typical coral structure. These abnormalities arise from regions of accelerated growth that result in tumours. There are 2 known types, hyperplasia and neoplasia. Hyperplasms contain zooxanthellae and are made from numerous cell types[6]. Scientific research has revealed that coral hyperplastic cells store fewer fats than normal cells[18] (**Fig 2**).

Coral neoplasms are made from only one type of cell, calicoblastic epithelial cells. These cells lack zooxanthellae and deposit the calcareous material usually found between corallites (**Fig 3**). As the tumour grows it protrudes upwards and covers the normal tissue, killing it in the process[6].

Red Band Disease (RBD)

shares some similarities with Black Band Disease (BBD), described in my previous article. Like BBD, RBD is a cyanobacterial mat colonised by other microorganisms that act in unison to destroy the coral tissue[1]. There are 2 types RBD I, and RBD II. RBD I has been described in *Agaricia*, *Colpophyllia*, *Mycetophyllia*, *Stephanocoenia*, and *Gorgonia ventalina*. The colouration of RBD is more maroon than BBD and the cyanobacterial filaments are often more dispersed. RBD II affects *D. strigosa*, *C. natans, Montastrea annularis*, *M. cavernosa*, *Porites asteroides* and *Siderastrea radians*. During the day RBD II can be distinguished from RBD I because it has a diffuse net like form, which at night compacts into a tight mass[7]. RBD has been observed in the Caribbean, and on the Great Barrier Reef (**Fig 4**).[14].

Fig 4. *G. ventalina* with RBD I – Photography by Andy Bruckner, 1997

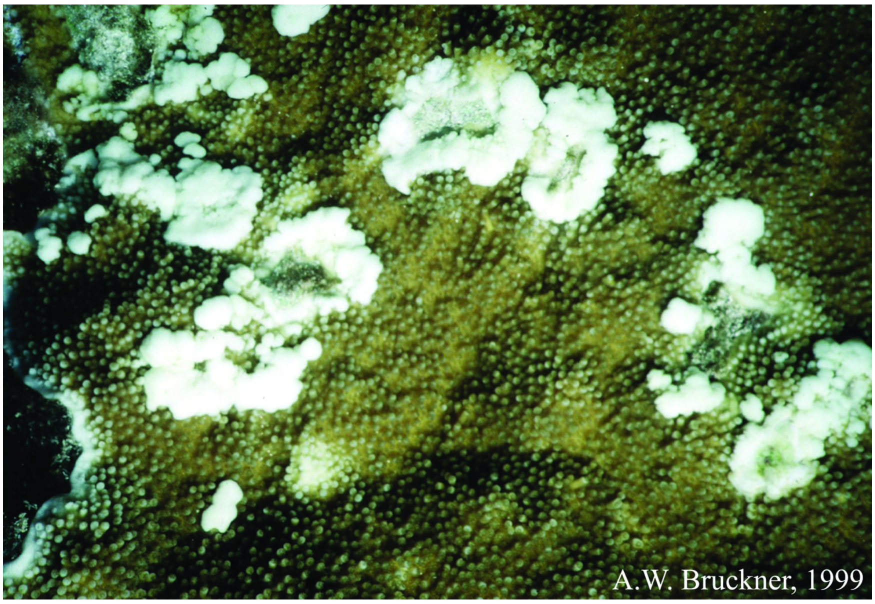

Fig 3. Neoplastic growth of *Acropoa palmata* – photography by Andy Bruckner, 1999

Yellow Blotch Syndrome (YBS)

also known as Yellow Band Disease, is common in *M. annularis* and *C. natans* throughout the Caribbean. Syndrome progression is slow and destroys the coral tissue at a rate of approximately 0.6 cm per month. Ancient colonies appear to be more susceptible to the condition that starts as a foci or band of translucent yellow tissue and radiates concentrically outward[14 cit., 2] (**Fig 5**). An unknown crystalline material has been discovered in the gastric cavity of the affected corals[16 cit., 2]. From 1997 to 1998 90% of Caribbean *M. annularis* had acquired the syndrome that inactivates up to 96% of the coral's zooxanthellae. The symbiotic dinoflagellates are not expelled but develop large void spaces within them, and lack the customary cellular machinery found in healthy zooxanthellae. Scientists have proposed that the syndrome has a primary effect on the zooxanthellae and a secondary effect on the ¥cnidarian host15.

Algal Galls

have been described in Pseudoplexaura sea fans in the Florida Keys and the Caribbean (**Fig 6**). The galls are produced as a consequence of a skeletal infection by the microalgae *Entocladia endozoica*. The sea fans try to defend themselves by "walling off" the infected area with calcareous sclerites and scleroproteins. Sclerite recruitment encapsulates the microalgae in galls and prevents them from deriving nutrition from the coral tissue. However, the galls have a deleterious effect on the elasticity and tensile strength of host's skeleton. Invariably the skeleton breaks, exposing the microalgae to the high light intensities found on shallow reefs.

The increase in illumination stimulates the microalgae to produce -zoospores that are released into the water column. Eventually the fracture heals and seals the microalgae beneath the coral tissue, enabling the whole process to start again. Scientists believe that the amino acids tyrosine and cytosine extracted by the microalgae from the coral tissue play a key role in regulating the reproductive cycle of the microalgae[3].

¥Cnidarian – "any of the various invertebrate animals of the phylum Cnidaria, characterized by a radially symmetrical body with a saclike internal cavity, including the jellyfishes, hydras, sea anemones, and corals". www.dictionary.com

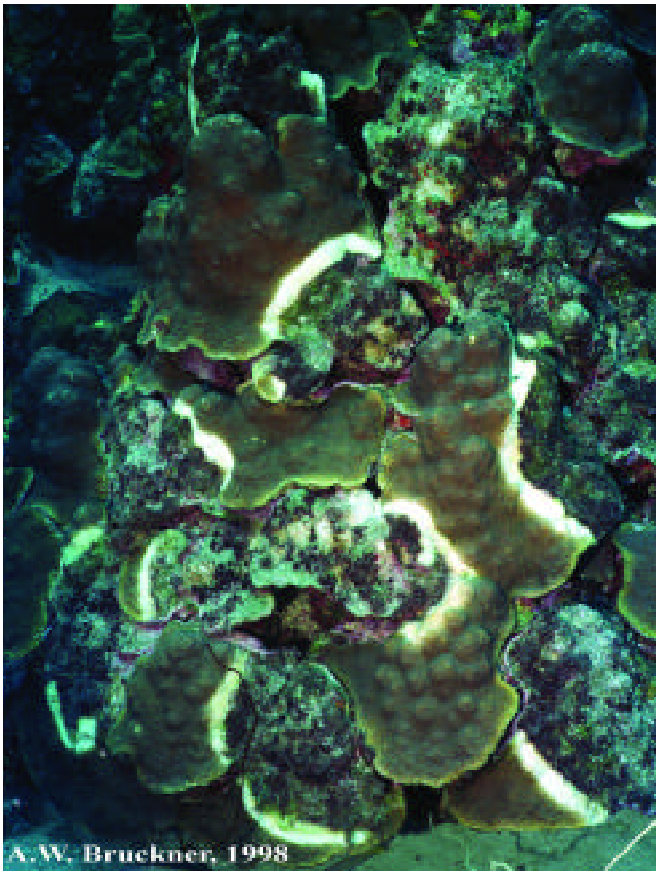

Fig. 5. *M. favolata* with Yellow Blotch Syndrome – photography by Andy Bruckner, 1998.

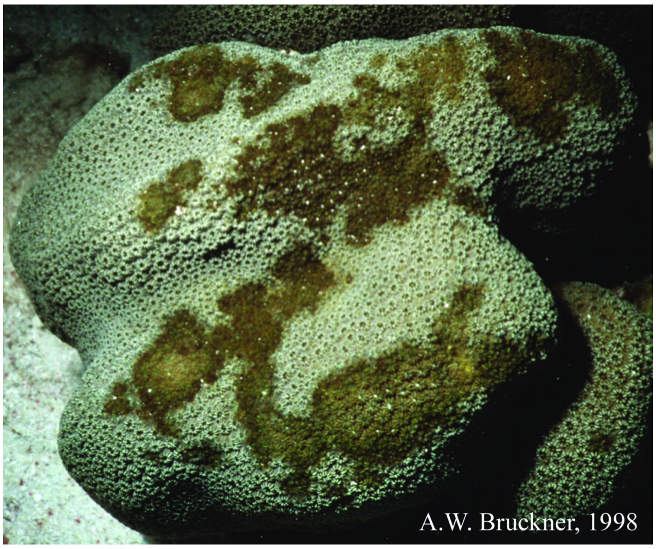

Fig. 7. Dark spot syndrome affecting *M. annularis* – photography by Andry Bruckner, 1998

Dark Spot Syndrome (DSS)

has been reported throughout the Caribbean. Brown or purple foci accompanied by a lack of polyp extension are symptomatic of the condition (**Fig 7**)[8]. Tissue loss is only experienced at the focus of infection[4]. DSS is commonest in *S. sidereal*, *Stephanocoenia intersepta*, *S. michelinii* and *M. annularis*[8]. Tissue necrosis occurs at a rate of 4.0 cm per month. Like YBS, the zooxanthellae are reduced in number and unable to divide[15].

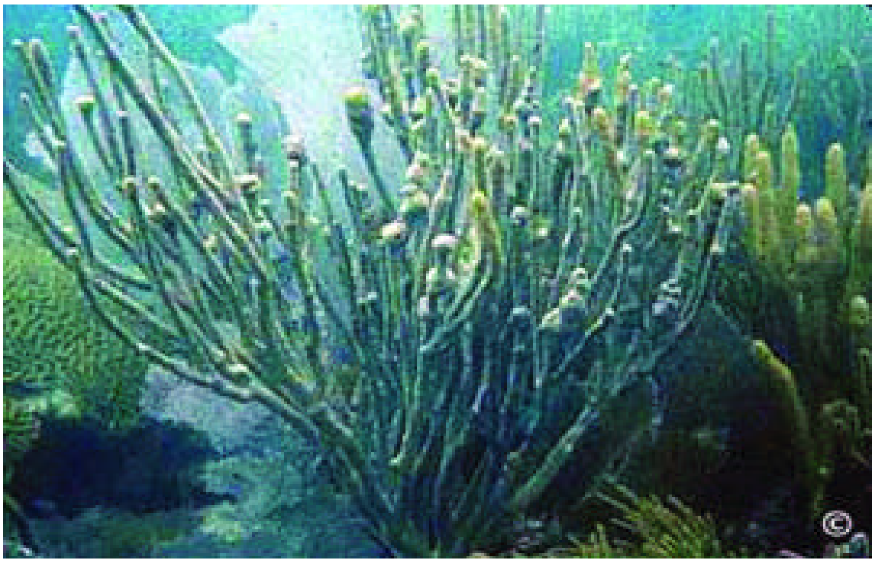

Fig. 6. A *Pseudoplexaura sea* fan exibiting Algal Galls – photography by Laurie Richardson

-Zoospores- "Amotile flagellated asexual spore, as of certain algae and fungi. Also called swarm spore". www.dictionary.com

Brown Jelly

was thought to be a condition only found in aquaria until it was observed in 2003 on the Great Barrier Reef[9]. Brown Jelly can affect all zooxanthellates. Brown Jelly looks exactly how the name suggests. It is a brown jelly-like mass that hovers over and envelopes the coral and upon occasion, releases strings of "jelly" into the water column (**Fig 8**). Microscopy has revealed that the "jelly" is a mixed population of microorganisms consisting of protozoa, bacteria and fungi. Microscopy has revealed that the protozoan *Helicostoma nonatum* ingests the zooxanthellae of affected corals. It has yet to be determined if the organisms are just performing housekeeping duties by consuming a degrading specimen, or if the microbes are involved in an infective process[10]. Outbreaks appear to be sporadic and affect both established and recently introduced specimens.

Rapid Tissue Necrosis (RTN)

or Shut Down Reaction is commonest in SPS corals and denudes the coral skeleton at a rate of approximately 1" per hour. Tissue loss is experienced at such a rate as to allow the observer to watch as the flesh vanishes from the skeleton. The ailment is associated with housing corals in poor conditions. Healthy tissue and bare skeleton share a discrete boundary[11]. The cause of disease is unknown but the bacterium Vibrio vulnificus has been isolated from RTN lesions[13].

Skeletal Eroding Band (SEB)

is the result of an infection by the ciliate

Halofolliculina corallasia. The ciliate infects and grows within the coral, secreting its microscopic shells (loricae) that disrupt the tissue. SEB has been found on the Great Barrier Reef and in the Indo-Pacific region. The families *Pocilloporidae*, *Acroporidae*, *Faviidae*, *Pritidae*, *Fungiidae* and *Merulinidae* are all susceptible[9].

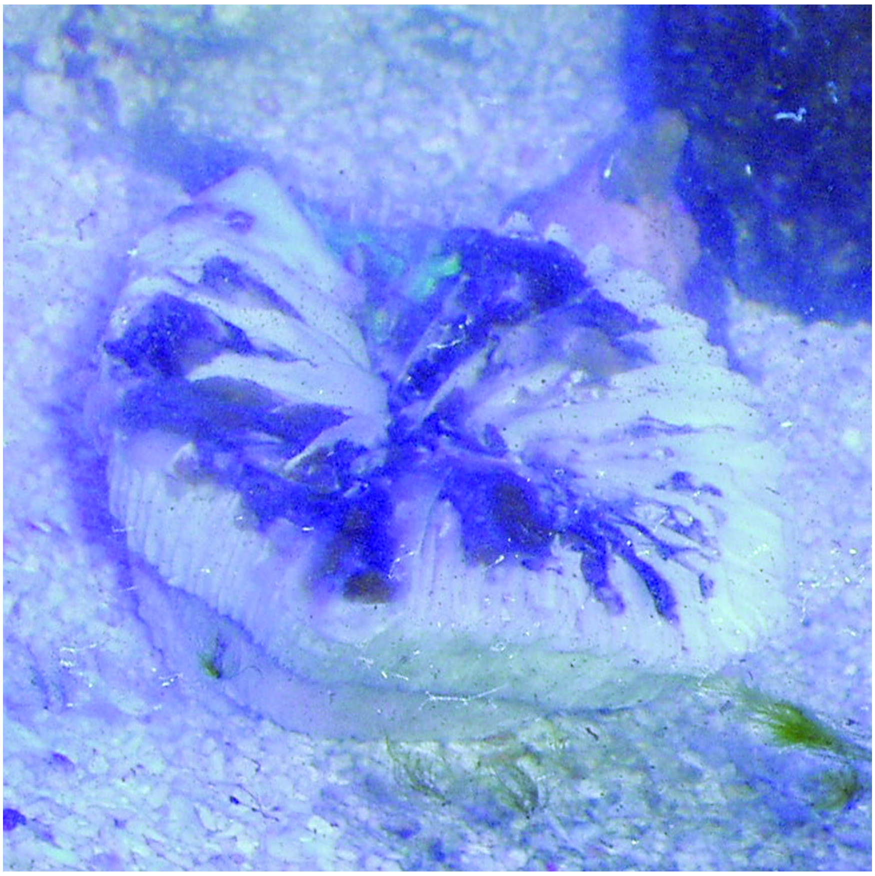

Fig 8. *Euphyllia cristata* affected by Brown Jelly – photograph by Chris Aslett ,2005

Black Necrosis Syndrome

affects Gorgonia on the northern Great Barrier Reef. Black patches of tissue slough away from the skeleton. The condition is caused by an unidentified fungus[9].

Pink Spot

has been observed on the Great Barrier Reef. The lifecycle of the parasitic flatworm *Podocotyloides stenometra* is comprised of 2 larval stages of development, prior to adulthood. During the larval stages the flatworm uses a mollusc for its first host and the coral *P. compressa* for its second. Maturity is only achieved within butterflyfish. An infected coral polyp becomes pink and swollen and therefore more vulnerable to predation by butterflyfishes. When the butterflyfish eats the polyp it then becomes host to the flatworm. After the infected polyps have been consumed normal polyps can regenerate[9].

Conclusion

Excluding the above, there have been many more reports of coral ailments that require scientific investigation. The majority of the conditions detailed here are likely to be a consequence of shifts in the ecological balance of the reef environment, caused by [§]anthropogenic activity. If you have witnessed any "coral diseases" in your aquaria and wish to share your experiences, please feel free to contact me through my web site (http://www.reefranch.co.uk). My next article will inform the hobbyist of the measures that can be taken to prevent coral diseases in their aquarium, and what is the best way to deal with an outbreak when it occurs.

[§]Anthropogenic- "of, relating to, or resulting from the influence of human beings on nature". www.dictionary.com

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