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Review

Diseases in coral aquaculture: causes, implications and preventions

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ABSTRACT

Aquacultured corals are typically reared in dense in situ (mariculture) or ex situ (in aquaria) culture facilities. This high density rearing method makes these corals particularly vulnerable to specific diseases since virulence and communicability of pathogens have been shown to increase with host density. As such, entire production lines may be threatened. Maricultured corals are particularly at risk as the diversity of both diseases and of affected coral species in the marine environment is on the rise. Coral diseases are now a major driver of coral mortality on all reef systems from the Indo-Pacific through to the Caribbean and not only affect species in situ, but can be inadvertently transported into the culture systems. The avoidance of disease outbreaks in culture systems is of upmost importance and the mitigation of diseases in these systems is vital in the maintenance of healthy cultures. Although the study of naturally occurring coral diseases has become a popular and relatively well-studied topic over the last few decades, the effects of these diseases on coral husbandry and aquaculture are still virtually unknown. Aquaculture of corals is a developing industry, both for stocking the ornamental industry and for restoration purposes. This overview outlines what is known about coral diseases in aquaculture; what implications these diseases have on this activity; what may be the causes of the disease outbreaks in these systems, as well as what methods are available for maintenance of healthy stocks and for mitigation once a disease has been observed.

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1. Introduction

Coral reefs have a high economic value as they provide coastal protection, supply food and natural products, and attract tourists from all over the world (Hoegh-Guldberg et al., 2007; Leal et al., 2012; Moberg and Folke, 1999). These highly productive and biodiverse ecosystems also provide hundreds of species for the marine aquarium hobbyists, a trade that has been increasing over the last decades to a point that is currently unsustainable if demand stays at its current rate (Delbeek, 2001; Rhyne et al., 2009; Wabnitz et al., 2003). Corals are among the most lucrative organisms in this trade and their demand has been increasing steadily. During the last few decades there has been a steady increase in demand for corals for basic research, including for natural product research (Leal et al., 2012; Shafir et al., 2006). All these activities demand large quantities of corals, some of which can be cultured in ex situ systems in order to decrease pressure on wild stocks. Coral aquaculture may supply all the activities that rely on wild-stocks, and is, therefore, a practice of growing interest for conservation, research and other economic purposes. Coupled with this rise in demand for corals for these purposes, increasing coral mortality has been observed on reefs worldwide (Hoegh-Guldberg et al., 2007). This has resulted in increased interest in basic coral biology (Weis et al., 2008) as well as on coral reef restoration methods, particularly in transplantation of cultured coral fragments (Jaap, 2000) back into the natural environment where coral cover has been reduced. If the health of cultured fragments including those produced in in situ coral farms is compromised, their use for coral reef restoration may affect the ecosystem as a whole.

Coral aquaculture can be performed in situ or ex situ (in open or closed systems). In situ aquaculture, also known as mariculture, is the culture of organisms (corals in the case of this review) in their local environment, e.g., corals that are cultured in tropical waters near to the reef ecosystems. In these systems, the corals are believed to be both subject to the optimal conditions and benefiting from the elements essential for growth, which are produced within this ecosystem. Despite these advantages, in situ coral cultures suffer from a plethora of natural stressors including predation, competition, pollution, storms and also natural disease outbreaks. Ex situ coral aquaculture in contrast, is the culture and propagation of corals outside of their natural habitat. Ex situ cultures may be performed in either recirculating aquaculture systems (RAS), which may in some cases be very distant from the nearest marine environment, or may be near marine environment, pumping natural sea water into their system and are then usually termed flow-through aquaculture systems (FTAS). The flexibility of ex situ systems is in many instances higher than that of in situ systems, as many features of culture system can be manipulated and controlled to maximize coral production and guarantee the successful husbandry of corals. On the other hand corals reared in FTAS are cultured in natural ambient conditions that are presumably optimal for their growth and health. The water source, and its treatment before reaching each tank, may differ between systems and there is no standard protocol for either FTAS or RAS with regards to use of natural or artificial seawater or a mixture of the two. Nonetheless, FTAS usually use natural seawater, and include certain filtration and/or sterilizing steps before the water reaches the culture tanks. While FTAS discard the water outflowing from the tanks and fresh water is pumped in, RAS reuse a high percentage of water after different filtration steps (Martins et al., 2010). The most common filtering method utilised involves particle removing stages, while sterilisation is usually performed with UV disinfection systems. RAS are usually used in facilities where natural seawater is not readily available and are therefore associated with the use of artificial seawater.

All aquaculture systems have advantages and disadvantages. In situ mariculture has relatively low culture costs, as the environment freely supplies food, water and energy essential to coral growth. Simultaneously, the control of abiotic and biotic factors is fairly limited and is mainly controlled by natural factors, which may have limiting influences due to differences in season, as coral growth may not always be maximised under natural conditions. Furthermore, other natural and anthropogenic features, such as physicochemical environmental impacts, predators, competitors and pollutants may hinder coral production. The introduction of anthropogenic disturbances may therefore affect coral growth and survival in both in situ and ex situ systems using natural seawater without appropriate treatment. In this view, controlled FTAS and RAS have the advantage of providing better control on the water system by manipulation of input water and preventing any potential problem associated with maintaining the corals in natural seawater. Conversely, one common problem with ex situ systems is the possible deterioration of water quality following long term use in completely closed recirculating systems (RAS). If the system is not properly monitored or the water is not properly filtered, water in recirculation systems may become low in nutrients and may have missing elements necessary for coral growth such as strontium, calcium and magnesium. RAS require an initial biological filter adaptation period in order to reach the appropriate physicochemical and biological equilibrium of the culture system, and if this equilibrium is disturbed dramatic consequences in the production may occur, and returning to equilibrium may be challenging.

One feature common to both in and ex situ aquaculture and that may threaten a whole production line is the possibility of contracting certain diseases. The high-density of individuals in the cultures carries risks related to pathogen communicability, and virulence. This threat is so great that any disease outbreak can result in the loss of the entire production. Coral aquaculture is not different from any other aquaculture venture, and prevention and mitigation of diseases play an important role in the maintenance of healthy systems.

Although the study of naturally occurring coral disease aetiology and epidemiology has become a popular and widely-studied topic in the last few decades (Muller and van Woesik, 2012; Pollock et al., 2011; Rosenberg and Kushmaro, 2011; Ruiz-Moreno et al., 2012), the causes and effects of diseases on coral husbandry and aquaculture are still virtually unknown (Sweet et al., 2012). Therefore, recommendations to prevent and treat disease outbreaks are also lacking.

This review outlines the status of current knowledge available on diseases in coral aquaculture. The overall aim of this review is to explore what is known about coral diseases in general and particularly in aquaculture, what implications they may have on this activity, what may be the causes of coral diseases in such systems and how to avoid them or at the very least manage and mitigate their spread. To aid in the understanding of the best practices in coral aquaculture and disease repression, we provide a short review of what is known regarding how corals resist and respond to diseases. Then we describe proposed origins of diseases present within coral cultures and how various factors affect their development. Whenever information is available, all aquaculture systems (mariculture, FTAS and RAS) will be assessed and differences outlined. Particular emphasis will be put on known methods to prevent diseases. However, because prevention is not always successful, treatment of diseased corals is sometimes necessary.

2. Coral diseases — An overview

2.1. Summary of current knowledge

A disease is defined as any impairment to cells or tissues of an organism that results in its dysfunction (Stedman, 1976). A diseased state involves an interaction between an organism, its environment and a disease agent (biotic or abiotic) (Stedman, 1976). Diseases may be either communicative, spreading the agent from one individual to another (e.g. tuberculosis) or not (e.g. cancer). In corals, a number of diseases have been described and classified morphologically, though for most of them the infectious agent is still unknown. Disease identification is carried out for the most part by describing morphological changes (anatomic pathology), which occur during the disease process, then determining the aetiology or cause of the disease and finally ascertaining its mechanism of action (Work et al., 2008). Therefore, when studying coral disease, it is still difficult or at times impossible to differentiate between disease states since many are characterised by the same or very similar morphological signs. A study by Lindop et al. (2008), conducted at the 11th International Coral Reef Symposium in Florida, demonstrated that even experts in coral disease confused symptoms such as fish bites with diseases such as white plague. It is arguable that even the studies which claim to have fulfilled Koch's postulates for a specific disease such as *Aurantimonas coralicida* causing white plague type II and Aspergillosis in sea fans being caused by *Aspergillus sydowii* need revisiting as in the case of *Aurantimonas*, for example, the bacterium has not been associated with WP again since its initial identification (Sunagawa et al., 2009). As for Aspergillosis, *A. sydowii* has more commonly been associated with healthy corals than sea fans showing this disease, raising serious doubt over the named causal agents (Toledo-Hernández et al., 2008).

Most coral diseases are characterised by changes in coloration such as tissue bleaching or spotting, or by loss of tissue confluence, and by massive cellular necrosis (Table 1; Fig. 1). However, very little is known regarding disease aetiology, including parameters such as physicochemical changes in the affected tissues. In some instances, certain functional groups of microorganisms (that may represent different species) always accompany particular disease signs (e.g. black band disease [BBD]; Cooney et al., 2002; Kuta and Richardson, 2002) though Koch's postulate remains to be fulfilled with regards to these diseases as well.

Sweet et al. (2012) reviewed what is known regarding coral diseases in the wild and in captive corals and categorised the diseases as natural and aquarium disease types. Many of the diseases found in the field are also apparent in cultured corals, both in FTAS and RAS systems. Nevertheless, it is almost impossible to confirm whether the same causative agents affect these organisms in the wild and in ex situ culture, even when the visible signs and progression rates of tissue loss may be very similar. It is likely that many of the diseases found in corals in ex situ systems are the result of introduction of relevant pathogens, changes in virulence factors due to improper handling, and/or due to stress factors that were not properly addressed. Most notable among these ubiquitous diseases are the aptly named 'white syndromes' that include many forms of tissue necrosis.

2.2. Issues in elucidating coral disease aetiology

Reported incidence of coral diseases is increasing worldwide and has largely been attributed to climate change, namely increases in sea surface temperatures (Bourne et al., 2009; Hoegh-Guldberg et al., 2007; Vezzulli et al., 2010). As stated above, although many studies have attempted to isolate single pathogens responsible for different diseases (reviewed in Sweet et al., 2012), few if any have fulfilled all stages of Koch's postulates (Fredericks and Relman, 1996). Furthermore, when independent researchers repeat these studies, they often fail to extract similar results (Ainsworth et al., 2007, 2008; Work and Aeby, 2011). Fulfilling Koch's postulates for suspected marine pathogens can be challenging because it necessarily involves culture and isolation steps (Fredericks and Relman, 1996). This can be problematic because certain coral diseases are believed to be caused by a consortia of microorganisms (e.g. black-band disease; Cooney et al., 2002; Frias-Lopez et al., 2003), coupled with most marine bacteria (the group of microorganisms to which most proposed coral pathogens belong) being un-culturable (Ferguson et al., 1984; Hugenholtz et al., 1998). A further complication arises because most studies looking for potential pathogens in wild corals use closed aquaria systems for controlled experiments. Although this is unavoidable in the large part, the differences between the two environments make it very difficult to infer from one environment to the other, making it difficult to link diseases that occur in the controlled aquarium environment to those on the reef system. Indeed, a recent study showed that there is a shift in coral associated flora in healthy corals maintained for even short periods in aquarium systems (Kooperman et al., 2007). Sweet et al. (2012) attempted to link visual signs of pathology of coral diseases occurring in reef systems around the world, to commonly found diseases within aquaria and concluded that although there are certain similarities between diseases such as the white syndromes (found in both aquarium and wild populations; Fig. 1; Table 1) and brown jelly syndrome (currently only reported in aquaria; Fig. 1; Table 1), slight differences in the pathology and aetiology resulted in the inability to reach strong conclusions about their similarities. Thus it is still unclear whether diseases described in one system were those occurring in the other. These differences in pathology may be due to differences in the physiology of the host resulting in microbial and environmental variations between aquaria and the wild. At present, the absence of systematic tests controlling for differences in these factors prevents us from ascertaining characteristic disease pathologies for each system. A further complication, regarding causal agents of diseases arises from the inability to control other non-target microorganisms (e.g. archaea, fungi, ciliates and viruses) during experimental trials. Ciliates for example, have previously been largely overlooked as potential coral pathogens, since they are typically bactivorous. However, recent studies (Ainsworth et al., 2007; Sweet and Bythell, 2012; Work and Aeby, 2011) have shown that certain ciliate species are responsible for coral tissue loss in diseases (at least as far as the pathology of the disease is concerned) such as WS in both the wild (Sweet and Bythell, 2012) and within aquaria (Sweet et al., 2012). Furthermore, although several studies have described increases in viruses associated with stressed corals (Davy et al., 2006; Vega Thurber et al., 2008; Wilson et al., 2005), and the same studies have implied they might be possible causative agents of certain diseases, their role as pathogens has also been largely disregarded. Finally, as opposed to the generally species-specific bacterial communities (Rohwer et al., 2002), corals seem to harbour a highly diverse and generalist archaeal community (Kellogg, 2004; Wegley et al., 2004). Although to date no archaeal pathogen has ever been described (Cavicchioli et al., 2003), Archaea seem to possess several characteristics often found in bacterial pathogens including: the ability to; i) live in association with eukaryotes, (ii) produce potentially toxic molecules, (iii) acquire new genes from bacteria and other archaea and (iv) contain cellular structures which may confer them with the ability to avoid immune defences (reviewed in Cavicchioli et al., 2003; Eckburg et al., 2003).

2.3. How corals resist and fight diseases

Although currently very little is known about coral immunity this particular field is becoming even more popular to study. Although corals seem to lack an adaptive or acquired immune system (no immunological memory) they possess a number of innate immunity mechanisms (Downs et al., 2009b; Kimura et al., 2009; Mydlarz et al., 2009; Palmer et al., 2011; Rinkevich, 2011). These processes provide corals with the ability to resist or overcome disease. However, investment in immune processes is taxon-specific and appears to be largely governed by how much energy and resources each taxon dedicates to constituent

Table 1
Common diseases that may affect corals in mariculture and in ex situ cultures worldwide (A — aquaria and ex situ cultures, IP — Indo-Pacific, M — Mediterranean, RS — Red Sea, WA — Western Atlantic and Caribbean). The information provided therein has been summarised from Raymundo et al. (2008), Sweet et al. (2012), the Global Coral Disease Database (http://coraldisease.org/) and the literature cited within the table.

Disease name	Other common appellations, abbreviations	Proposed pathogen	Coral species affected	Disease description	Regions affected	Key references
Aspergillosis	ASP	*Aspergillus sydowii*	Only octocorals, primarily *Gorgonia*, *Pseudopterogorgia*, *Plexaura*, *Plexaurella*	Focal/multifocal purple rings(s) progressing outwar	WA	Nagelkerken et al. (1997)
Atramentous necrosis	AN	Unknown. May be promoted by environmental factors	Mostly *Montipora* spp., but also *Acropora*, *Echinopora*, *Turbinaria*, *Merulina*	Mutlifocal to irregular lesions exposing bare skeleton followed by grayish-black fouling community	IP	Jones et al. (2004), Haapkyla et al. (2011)
Bacterial bleaching		*Vibrio coralliilyticus*, *Vibrio shiloi*	*Pocillopora* spp., *Oculina patagonica*	Focal/multifocal/coalescing tissue discoloration	IP, M, RS	Ben-Haim et al. (2003), Kushmaro et al. (1998)
Black band disease	BBD	Microbial consortium dominated by cyanobacteria (*Geitlerinema*, *Leptolyngbya*, *Oscillatoria*, *Pseudoscillatoria* spp.)	40+ coral species, primarily *Acropora* spp.	Black/reddish band that radiates outward from colony margin or lesion	WA, IP, RS	Cooney et al. (2002), Richardson (2004), Sussman et al. (2006), Rasoulouniriana et al. (2009)
Brown band disease	BrB	Ciliate (*Philaster* sp.)	Primarily on *Acropora* spp.	Brown band located at the interface between living tissue and exposed skeleton, usually starts from base of the branch	IP	Bourne et al. (2008), Sweet and Bythell (2012)
Brown jelly syndrome	BJS, BJ, brown slime, brown jelly	Ciliate (*Philaster* sp.)	*Euphyllia*, *Acropora*, *Goniopora*, *Xenia* spp.	Fast progressing brown jelly-like mass that seems to "float" above coral tissue	A	Borneman (2002), Sweet et al. (2012)
Caribbean ciliate infection	CCI	Ciliate (*Halofolliculina* spp.)	10+ species including *Dichocoenia*, *Montastraea*, *Acropora* spp.	Speckled to black band, similar to skeleton eroding band	WA	Cróquer et al. (2006)
Dark spots syndrome	DSS, dark spot disease (DSD), DSD type II	Fungi & bacteria (*Vibrio spp.*)	Primarily affects *Montastraea*, *Siderastrea* spp., and *Stephanocoenia*. Also *Agaricia agaricites*	Focal to multifocal, annular to irregular margins, purple to brown in color	WA	Gil-Agudelo and Garzon-Ferreira (2001)
Growth anomalies	Hyperplasia, neoplasia, GA, tumors, calicoblastic epithelioma	Unknown; maybe genetic or caused by environmental stress	*Diploria*, *Colpophyllia*, *Porites*, *Montastraea*, *Agaricia*, *Acropora*, *Dichocoenia*, *Madracis*, *Pocillopora*, *Pavona*, *Fungia*, *Madrepora*, *Montipora*, *Platygyra*, *Goniastrea*	Focal to multifocal, circular or irregular abnormal skeletal growths	WA, IP, RS	Peters et al. (1986)
Rapid tissue necrosis	Shut down reaction, RTN	Bacterial *(Vibrio harveyi, Vibrio alginolyticus)*	Affects primarily *Stephanocoenia intersepta*, *Siderastrea siderea* and *Montastraea annularis*	Rapidly progressing tissue degradation	WA, IP, RS, A	Anthony (2004), Luna et al. (2007)
Red band disease	RBD, red band disease type I, RBD type II	Microbial consortium dominated by Cyanobacteria (*Agaricia* spp.)	Common on octocorals and also affects *Agaricia* spp., *Meandrina*, *Mycetophylia* spp. and other less common species	Diffuse to circular red to dark reddish-brown band progressing from colony margin or lesion	WA	Richardson (1992), Sussman et al. (2006)
Skeleton eroding band	SEB	Ciliate (*Halofolliculina corallasia*)	Primarily on *Acropora* and *Pocillopora* spp., but affects 12 families in total	Black or dark green diffuse/speckled band progressing from recently exposed skeleton or lesion	IP, RS, A	Riegl and Antonius (2003)
Trematodiasis		Trematode	Mainly *Porites* spp.	Focal/multifocal swollen nodules (~1–3 mm)	IP	Aeby (1991)
Ulcerative white spot disease	UWS, *Porites* ulcerative white spot disease	Bacterial (*Vibrio* spp.)	Primarily *Porites* spp.	Focal/multifocal spots (3–5 mm, max 5 cm) of bleached tissue, progressing to multifocal patterns of tissue loss	WA, IP	Raymundo et al. (2003)
White band disease type I	WBD, WBDI	Bacteria (undefined)	*Acropora* spp.	Progressing band of bleached tissue followed closely by necrotic tissue starting from the base of the branch	WA, IP, RS	Peters et al. (1983)
White band disease type II	WBDII	*Vibrio charcharia*	*Acropora cervicornis*	Similar to WBD type I, but tissue loss is preceded by 2–20 cm of bleached tissues	WA, IP, RS	Smith and Ritchie (1995)
White plague type I	WPI, WP	α-Proteobacterium associated with juvenile oyster disease	40+ species of non-acroporid massive and plating corals	Multifocal to coalescing tissue loss starting from base or margin of colony (or algae/sediment interface with colony) and followed by algal colonisation, progressing at 1 mm to >10 cm/day	WA, RS	Richardson et al. (2001), Pantos et al. (2003)
White plague type II	WPII	*Sphingomonas* spp., *Aurantimonas corallicida*	~17 coral species but primarily affects *Dichocoenia stokesi*	Similar to WPI, but much faster rate of tissue loss (~2 cm/day)	WA	Richardson et al. (2001)
White plague type III	WPIII	*Sphingomonas* spp., *A. corallicida*	Primarily affects *M. annularis and Colpophyllia natans*	Similar to WPII but the rate of tissue destruction is extremely high, leaving white skeleton with no turf algae	WA	Richardson et al. (2001)
White pox	White patch disease, patchy necrosis	*Serratia marcescens*	Exclusively affects *Acropora palmata*	Irregular lesions from a few cm^2 to 80 cm^2 that can develop simultaneously on all surfaces of the colony	WA	Sutherland et al. (2010)
White syndrome	WS	*Vibrio* sp., *Arcobacter* sp.	Many coral species including *Turbinaria, Acropora, Goniastrea, Pocillopora, Porites, Pavona, Stylophora, Montipora,* Faviidae	Diffuse areas of tissue loss exposing bare skeleton	IP, RS, A	Luna et al. (2010), Sussman et al. (2008), Sweet and Bythell (2012)
Yellow band disease	Yellow blotch disease, YBD	Bacterial (*Vibrio* spp.)	Primarily *Montastraea* spp.	Focal/multifocal blotches followed by a circular yellow to white margin	WA, IP	Cervino et al. (2008), Weil et al. (2009), Cróquer et al. (2013)

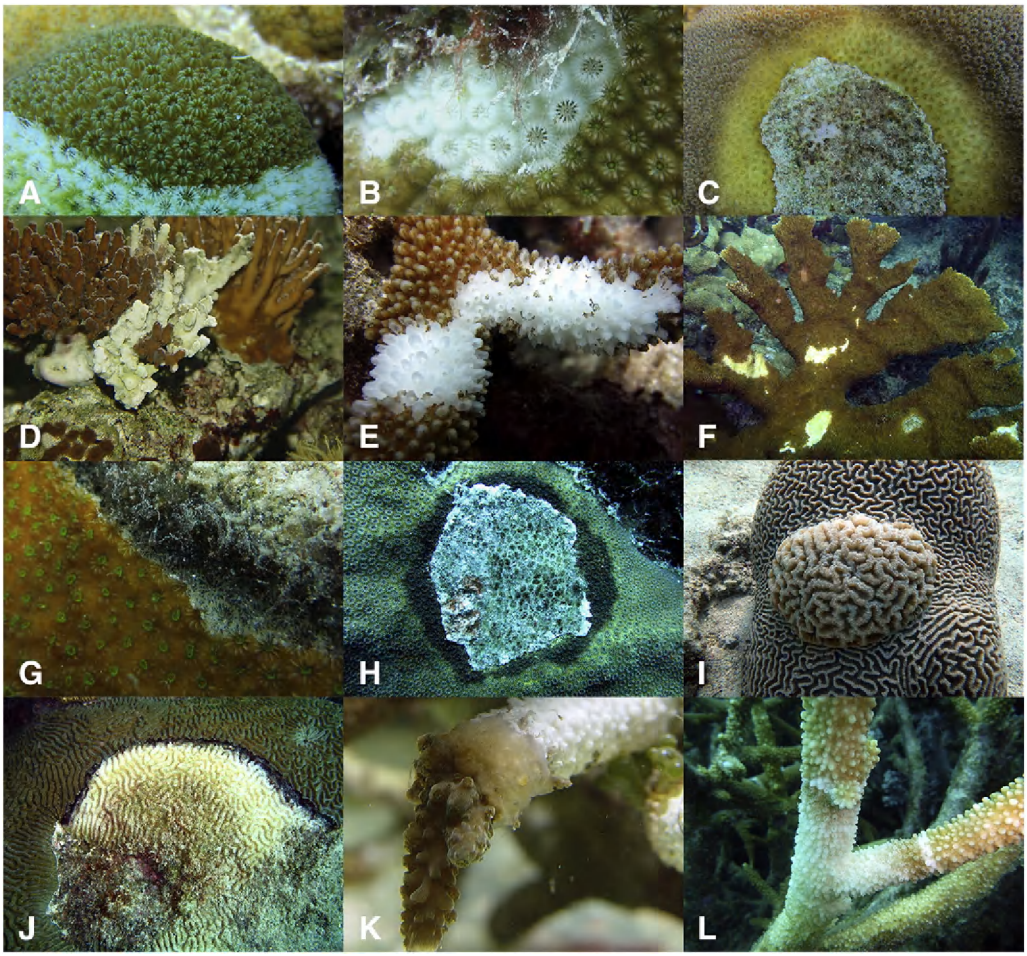

Fig. 1. Major diseases affecting corals worldwide. A — white plague, B — white syndrome, C — yellow band disease, D — rapid tissue necrosis, E — white band disease, F — white pox, G — Caribbean ciliate infection, H — dark spot syndrome, I — growth anomaly, J — black band disease, K — brown jelly syndrome, L — brown band disease. Pictures taken by MS and CS.

immunity when compared to other processes such as reproduction and growth (Palmer et al., 2010). As such, Palmer et al. (2010) showed that coral species with life history strategies aimed towards fast growth and high reproductive output (e.g. Acropora spp.) are more susceptible to disease than those favouring slow growth and low reproductive output (e.g. *Porites* spp.).

Typical stress responses may include gross physiological responses and play an important role in coral immunity. Processes such as mucus production, melanin synthesis (Palmer et al., 2008), the deployment of amoebocytes (Mydlarz et al., 2008), production of antioxidants, antibacterials and free radicals, and processes of xenophagy (Downs et al., 2009a), all partake in innate immune responses of these organisms. In addition, as with higher organisms, the presence of symbionts, including eukaryotes and prokaryotes, may act as a shield in protecting the host by producing antimicrobials, or by filling specific niches, thus preventing invasion by pathogens (Brown and Bythell, 2005; Reshef et al., 2006; Ritchie, 2006; Shnit-Orland and Kushmaro, 2009; Shnit-Orland et al., 2012).

Removal of unwanted microorganisms may occur via active removal using mucus as a trap (Brown and Bythell, 2005; Mydlarz et al., 2010), or once internalized by xenophagy (Downs et al., 2009a). In addition amoebocytic cells that are found in coral tissues were shown to have antimicrobial properties in other marine organisms such as sea anemones and may play a similar role in corals (Hutton and Smith, 1996). Indeed an increasing number of studies have shown that amoebocyte numbers were correlated with a diseased state in gorgonians and in diseased scleractinian coral tissues (Domart-Coulon et al., 2006; Ellner et al., 2007; Mydlarz et al., 2006; Mydlarz et al., 2008).

3. Diseases in coral cultures

3.1. Origins and factors influencing disease development in coral cultures

Knowing where coral disease pathogens originate from and how they affect corals specifically are questions for which no unique and simple answer can be found. The main issue is that in some cases, there might not actually be an original source. For example, the proposed coral pathogens; *Vibrio coralliilyticus* (Ben-Haim and Rosenberg, 2002) and *Vibrio shiloi* (Kushmaro et al., 1996, 1997, 1998) have been found associated with healthy coral tissues (Bourne and Munn, 2005; Vega Thurber et al., 2009) and are thought to only become pathogenic when environmental parameters and/or host immunity changes. Therefore it is possible that diseases develop when stressors such as increased temperature either modify the structure of the coral microbial symbiotic community (Vega Thurber et al., 2009), or trigger the production of virulence factors (Bourne and Munn, 2005). While in most cases the causes and origins of coral disease development remain to be elucidated, a few infection pathways have been described. These tend to be associated with environmental/anthropogenic factors, biological vectors, or a combination thereof. However, the sources and factors influencing coral disease development will be different between coral cultures in the natural environment (in situ) and in artificial environments (ex situ).

3.1.1. Mariculture (in situ)

In the natural environment, environmental and anthropogenic stresses have frequently been correlated to increases in coral disease prevalence (Bruno et al., 2007; Harvell et al., 1999, 2007). The primary

stressors involved are temperature and water quality degradation. Thermal stress (higher than normal seawater temperature) is by far the most referred to of all environmental stressors and has often been described to be a determining factor in facilitating coral disease development and infection by pathogens (e.g. Bourne et al., 2009; Harvell et al., 1999; Kuta and Richardson, 2002; Ward et al., 2007). The effect of thermal stress as a catalyst for coral disease development is particularly likely when coral cover is high (Bruno et al., 2007), as is expected in coral mariculture facilities. Temperature stress can increase chances of coral disease development in two ways: coral holobiont stress (and decreased coral resistance to infection) and/or increased growth and virulence of opportunistic coral pathogens. For example, it was established that the virulence factors associated with infections of *Oculina patagonica* by *V. shiloi* (in the Mediterranean) and of *Pocillopora damicornis* by *V. coralliilyticus* (in the Red Sea and Indo-Pacific) are triggered by elevated seawater temperatures (Ben-Haim et al., 2003; Kushmaro et al., 1998; Toren et al., 1998). Furthermore, increased temperatures have been shown to decrease the production of antimicrobials by symbiotic bacteria in the coral mucus, thereby facilitating the growth of opportunistic and potentially pathogenic bacteria (Ritchie, 2006). However, Ritchie (2006) could not resolve whether a temperature-dependent growth of the population of opportunistic bacteria reduced the population of symbiotic bacteria and consequently the production of antimicrobials, or a temperature-dependent shutdown of antimicrobial production by symbiotic bacteria allowed the development of opportunists. Later work by Shnit-Orland and Kushmaro (2009) supports the theory of temperature-sensitive antimicrobial compounds suggesting that both the quantity produced and their stability decrease past a specific threshold.

Water quality degradation is another important coral stressor, and is usually associated with heavy rainfall, anthropogenic pollution and terrestrial runoff, though the link between anthropogenic stress and disease susceptibility is currently poorly understood (Raymundo and Harvell, 2008). Terrestrial runoff results in higher nutrient and particulate matter concentrations, increased sedimentation and reduced light penetration (reviewed in Fabricius, 2005). Higher than normal nutrient concentrations often found associated with terrestrial runoff have been suggested to be associated with higher coral disease prevalence and severity (Bruckner et al., 1997; Bruno et al., 2003; Kim and Harvell, 2002). However, recent work suggested that their influence on coral disease development is minimal, as opposed to that of dissolved and particulate organic carbon concentrations (DOC and POC, respectively) (Haapkyla et al., 2011; Kline et al., 2006; Kuntz et al., 2005). Consequently, though measures of dissolved nutrient concentrations (nitrate, phosphate, ammonia) are typical components of water quality assays, DOC and POC measurements should also become standard, particularly when monitoring coral health in mariculture facilities. Furthermore, although in the marine environment, terrestrial runoff affects corals primarily through seawater eutrophication and increased sedimentation, their role as a source of potential pathogens should not be disregarded. Human sewage for example, was recently identified as a likely source of the faecal coliform bacteria *Serratia marcescens*, a proposed coral pathogen of white pox (Fig. 1; Table 1) on *Acropora palmata* in the Caribbean (Sutherland et al., 2010).

While environmental factors play a role in coral susceptibility to disease, several corallivorous organisms have also been suggested as vectors of coral pathogens. Initially, many invertebrates were suggested as vectors of specific pathogens. In the Florida Keys for example, the corallivorous snail *Coralliophila abbreviata* has been found capable of transmitting proposed coral pathogens (Williams and Miller, 2005). Similarly, *Hermodice carunculata*, the marine fireworm has been demonstrated to be acting as a reservoir (in the winter) and vector (in the summer) of *V. shiloi*, leading to bleaching of exposed colonies of *O. patagonica* (Sussman et al., 2003). In the Red Sea, an outbreak of the corallivorous snail *Drupella cornus* was correlated with an outbreak of white syndrome (Antonius and Riegl, 1997) and the involvement of the crown of thorn starfish, *Acanthaster planci*, has been suggested in the transmission of Brown Band Syndrome (Fig. 1; Table 1) on *Acropora cytherea* in Indonesia (Nugues and Bak, 2009). Among the vertebrates, Aeby and Santavy (2006) showed that under laboratory conditions, the presence of Foureye butterflyfish (*Chaetodon capistratus*) in aquaria containing colonies of *Montastraea faveolata* (two healthy colonies and one infected with black band disease (BBD; Table 1; Fig. 1)) resulted in infection of the healthy colonies by BBD. However, pathogen transmission has only been shown in the case of *H. carunculata* (Sussman et al., 2003). In all the other aforementioned studies, transmission of coral pathogens could not be distinguished from facilitation (where a corallivorous organism *facilitates* coral infection by pathogens through feeding scars). Considering the large diversity of corallivorous organisms on tropical coral reefs (Rotjan and Lewis, 2008), it is likely that many other species may also play a role in the transmission and/or facilitation of coral diseases.

Finally, the genetic composition of the coral host as well as that of the coral's microbiota is worthy of mentioning here as it can drastically affect disease susceptibility. Different disease susceptibilities may be observed both between coral species and between colonies of the same species. For example, through a combination of field surveys and in situ transmission experiments Vollmer and Kline (2008) found that 6% of *Acropora cervicornis* genotypes were resistant to white band disease (WBD; Table 1; Fig. 1), and suggested that this finding may explain why certain populations of *A. cervicornis* survived the WBD epidemic that started in the 1980's. On the other hand, variation in inter-specific disease susceptibility has been frequently described. For example, Aeby et al. (2011a) showed that different *Acropora* species had varying susceptibility to infection by *Acropora* white syndrome in the Central Pacific. Similar data was obtained in the US Virgin Islands over a range of 12 species of scleractinian corals (Calnan et al., 2008). Although regional variations are likely to occur, a general pattern seems to be occurring, whereby fast-growing corals (like *Acropora* spp. and *Pocillopora* spp.) are typically more susceptible than the slow growers (such as *Porites* spp.;Mydlarz et al., 2010; Palmer et al., 2010). Firstly, one might hypothesise that a species-specific tolerance to environmental stress, as suggested in the case of thermal stress by Fitt et al. (2009), might lead to species-specific susceptibility to infections. This is supported by data from Brandt and McManus (2009), which showed a positive correlation between bleaching extent and coral disease incidence in the Caribbean. In addition, the composition of the microbial communities might also play a role in susceptibility to disease development. It has been shown that corals harbour species-specific bacterial communities, with conspecific corals showing little variation in the composition of their microbial associates over several thousand kilometres, whereas the microbial community structure of different species can be drastically different, even over short distances (Rohwer et al., 2002). Coral microbial populations have also been suggested to evolve according to several stressors (Vega Thurber et al., 2009), and such modifications have been suggested to play a role in the resistance of corals against infections (Reshef et al., 2006; Rosenberg et al., 2007). Finally, as suggested in Mydlarz et al. (2010), different coral species likely manage their energy budgets in a different manner, with fast-growing species (e.g. *Acropora* spp.) dedicating more of their energy to growth and less to immunity (Palmer et al., 2008).

3.1.2. Ex situ culture

As mentioned in the beginning, ex situ aquaculture systems come in a number of varieties, from FTAS where sea water is pumped into the culture system and pumped back out to sea (filtered at entrance, exit or both), to RAS using either artificial sea water or natural filtered sea water and a low percentage of water renewal. In FTAS, water is the main potential source of pathogens and contaminants as it comes directly from the natural environment. When these systems use ambient seawater without proper filtration or UV treatment, pathogens may enter

and persist; at this point stressors might cause increases in pathogen virulence and growth (Kimes et al., 2012; Ward et al., 2007) resulting in the decimation of the culture. The origin of diseases previously addressed for in situ coral aquaculture (see previous Section 3.1) is also applicable for ex situ aquaculture system using natural seawater without appropriate pre-treatment. However, the input of natural diseases in an artificial environment may affect the natural behaviour of the disease agent. For instance, high-density of corals is expected in ex situ aquaculture facilities, which will certainly increase the communicability of the pathogen among coral hosts and contribute to the collapse of the coral production. On the other hand, since closed/recirculating systems use either artificial or filtered seawater, these systems are not likely to be contaminated by pathogens entering naturally through the water inlet. Unfortunately, the water entering the culture system is not the only potential source of coral pathogens and even with adequate water treatment, neither FTAS nor RAS are entirely without risk of contamination. Pathogens may enter through a variety of pathways, including the introduction of contaminated coral specimens, pathogen-vector species (e.g. *H. carunculata* fireworms and coral-eating snails as discussed in Section 3.1.1), and live rock. Live rock in particular has been shown to commonly harbour undesired species such as fireworms *Hermodice* spp. (Calado et al., 2007; Carl, 2008). Consequently, disease onset in closed systems suggests a lack of prophylaxis or proper quarantine measures (which are subsequently reviewed in Section 4.2).

Finally, it has been argued that many coral diseases are the result of an increase in abundance of specific opportunistic coral pathogens following changes in environmental parameters (Lesser et al., 2007). Though not subjected to natural or anthropogenic modifications of the environment, closed systems could nevertheless be affected by changes in environmental conditions (e.g. temperature and nutrient loadings) through certain unplanned events such as equipment dysfunction. Such environmental changes have been shown to cause shifts of the coral microbial population from mutualistic/commensal to potentially pathogenic and opportunistic (Vega Thurber et al., 2009).

3.2. Difference in coral associated bacterial communities between natural and aquarium environments

Although aquarium corals all originally stem from wild populations, they will likely undergo strong selection by the time they reach the end user. Many of the more susceptible genotypes would have failed to survive the long transportation from collection, holding in local facilities, international freighting and finally life within the aquaria. Interestingly, *Acropora muricata*, for example, that will undergo stress responses and ultimately bleach and/or contract diseases such as white syndrome when exposed to temperatures of 32 °C in reef systems such as the Great Barrier Reef, can withstand temperatures in excess of 33 °C in the aquarium and suffer no visible ill effects (Sweet pers. obs.). Eventually these aquarium corals when pushed to the limits experience what appears as a rapid tissue loss or what has also been termed as shut down reaction (SDR) in the aquarium trade, rather than a progressive disease syndrome as is often seen in the wild following stressful conditions. In some cases, certain susceptible coral species such as Acroporids, which are vulnerable to increasing temperature stress over much of their range such as the Great Barrier Reef, appear to be more tolerant to high temperatures in so called hot spots such as Thailand. There are two main theories as to why this occurs: 1) the pathogenic agents causing diseases such as WS are not present in Thailand, therefore even if the corals do become stressed they don't subsequently succumb to a disease state or 2) the corals in this area have already undergone a strong selection pressure (similar to that experienced with aquarium corals) and therefore those which remain are more capable of withstanding any potential pathogens. Alternatively, there is a third option whereby the microbial community of the colonies in Thailand includes certain "probiotic" strains that are capable of mitigating these diseases.

Bacteria have often been proposed as the main causal agents of many coral diseases in the wild (Bourne et al., 2009; Cooney et al., 2002; Pantos et al., 2003; Sussman et al., 2008). However, few studies have looked at bacterial communities in aquarium diseases (Kooperman et al., 2007; Luna et al., 2007, 2010). Bacterial associates of healthy aquarium corals from a variety of different species such as *Acropora pulchra*, *A. muricata*, *P. damicornis*, *Seriatopora hystrix*, *Montipora capricornis* and *Euphyllia yaeyamaensis* appear to show homogeneity in their bacterial communities under aquarium conditions. When kept within the same aquaria, healthy corals show similar community profiles between species (Sweet et al. pers. obs.), a result that contrasts with the unique microbial species assemblages associated with particular host species in the wild (Ainsworth and Hoegh-Guldberg, 2009; Ceh et al., 2011; Gil-Agudelo et al., 2007; Guppy and Bythell, 2006; Rohwer et al., 2002). Although Ceh et al. (2011) did note that the variation in bacterial community structure observed in in situ colonies was mainly due to spatial differences rather than differences between species, in most studies a greater distinction occurs between host species than between those with higher spatial distribution (Ainsworth and Hoegh-Guldberg, 2009; Guppy and Bythell, 2006; Kvennefors et al., 2010; Rohwer et al., 2001, 2002). When corals contract WS in aquaria, the bacterial communities change from their healthy state(Sweet et al., in press), as is seen in the wild (Pantos and Bythell, 2006; Rohwer et al., 2002; Sweet and Bythell, 2012), but different species and/or individuals of the same species have no single dominant pathogen in common, suggesting that in this case either the corals sampled were all showing different diseases or that bacteria are not the causal agents of WS in aquaria (Luna et al., 2007, 2010; Sussman et al., 2008; Sweet et al., 2012).

4. Preventing coral diseases

4.1. Policy, management and good practice

It has been argued that poor management both in exportation, importation and regulation in the animal trade has led to some widespread ecological problems. This is particularly the case in the aquarium industry. Two prominent cases include the release of two species of lionfish (*Pterois volitans and Pterois miles*) from aquaria in the Caribbean and the spread of the amphibian fungus *Batrachochytrium dendrobatidis*, which causes Chytridiomycosis (Gahl et al., 2012; Gründler et al., 2012; Rasconi et al., 2012). Zoos, public and private aquaria and hobbyists around the world have been partially implicated for the significant spread in these particular cases, either accidently by releasing captive animals by good natured people or simply by unwittingly transporting diseased animals from site to site (as is thought to be the case with the Chytrid fungus) (Der Sluijs et al., 2011; Lannoo et al., 2011; Puschendorf et al., 2011). Although no coral species are yet officially considered exotic/invasive species, they may also arise several concerns. The corals *Tubastraea coccinea* and *Tubastraea tagusensis*, both originally from the Pacific Ocean, were introduced in Brazil in the 1980s and are threatening local benthic biodiversity (Lages et al., 2011). Coral diseases associated with these introduced species have currently not been reported, however the possibility remains that pathogenic organisms (responsible for certain coral diseases) may have also been transported around the world in a similar manner. Indeed the white diseases (white plague and White Band) found throughout the Caribbean are a relatively recently phenomenon, first being reported in the early 1970s (Aronson et al., 2005; Bythell et al., 2004; Gladfelter, 1982) and spreading rapidly with very high mortality rates following their introduction. This suggests that the causal agents of these diseases may have been introduced into the Caribbean and not be native to the location. With growing evidence linking the pathogenic agents (namely the ciliate; *Philaster* sp.) associated with WS in the Indo-Pacific and the same/similar species found within aquaria worldwide, there is potential that a release of this pathogen may have occurred via aquaria. Lessons should be learned from previous errors, and management of marine invertebrates

in general should be improved. Adhering to strict management practices may help prevent further ecological problems and, therefore, should be implemented at some point between the initial production of the aquacultured organism and its use by the consumer.

The pipeline of coral trade usually follows certain steps: collection, packaging, shipping, receiving/importation by wholesale, retail and finally ends with the private consumer. This long path also creates opportunities to implement management rules to reduce/mitigate these potential threats through general improvement in policy, management and good practice. Currently, coral trade is regulated in 176 countries via the Convention on International Trade in Endangered Species of Wild Fauna and Flora (CITES) which covers >2000 coral species. However, the scope of CITES is limited to regulating the trade of coral species for country/species combinations for which it was found that the trade of specimens could be detrimental. However certain recent developments such as the EU Wildlife Trade Regulation Captive Breeding Database, which was developed for EU CITES authorities (http://captivebreeding.unep-wcmc.org/Home/About, 02/12/12), could be adapted to include information on breeding stocks affected by parasites/pathogens. Besides CITES, other organizations may also have an important role in future management practices, such as the Marine Aquarium Council (MAC). This organization aims to promote a responsible aquarium trade, particularly of marine ornamentals. Already, MAC has in practice several standards and certification processes covering corals, such as the "Collection, Fishing and Holding Standard" (Holthus, 1999), which addresses harvesting, handling prior to export, holding, packaging and transporting. The application of this and other standards yet to be developed can improve management practices throughout the pipeline.

Corals are usually collected from specific sites, most notably in Indonesia, Fiji and the Philippines (Tissot et al., 2010), then transported by freight and held in distribution areas. Sometimes these corals (as is the case for reef fish) are held in quarantine tanks. Although quarantine should last at least thirty days, this is often ignored as it is not a requirement by law. As this step often comes with additional costs it is unfortunately missed out to keep exportation costs at a competitive price. From here, the corals are then distributed to various outlets for further sale. Currently, corals are very much in the shadow of general aquarium trade with most legislation and policies focusing on higher organisms such as the reef fishes. Evidence indicates that collection of some coral reef animals for these trades has caused virtual elimination of local populations and major changes in community/age structure (Tissot et al., 2010). Management and enforcement of collection activities in major source countries, such as Indonesia and the Philippines, remain weak. Strengthening trade laws in countries that import these corals and strengthening of the enforcement capabilities combined with increasing consumer and industry demand for responsible conservation can create strong incentives for improving management in source countries. Implementing local and national management measures is particularly important as the addition of international regulations using existing conventions (e.g. CITES) takes a long time and such proposals may be rejected; for example the addition of the Coralliidae from the Mediterranean Sea was rejected at the 15th CITES Conference of the Parties in Doha (Qatar) (Tissot et al., 2010). Currently, there is a glimmer of positive action being undertaken, with numerous governmental and international agencies working together to develop 'Coral Health Certifications,' yet these are still in their early developmental stages (Berzins et al., 2008; Cato and Brown, 2008).

One further area for improvement is the detection of diseases before transportation. Coral diseases, unlike those in fish (which often manifest themselves quickly), can take days or weeks to show progressive lesions. Most commonly the response by aquarists to disease outbreaks is fragmentation, removal of the dyeing section and/or a water change. Molecular screening for potential pathogens is one way to identify the onset of disease before visual signs are evident as changes in the microbial diversity have frequently been observed in apparently healthy tissues in advance of the disease lesions (Pantos et al., 2003; Sweet and Bythell, 2012). These techniques (e.g. non-culture dependant molecular analysis tools such as polymerase chain reaction (PCR), quantitative PCR, Denaturation Gradient Gel Electrophoresis and/or deep sequencing, coupled with culture dependant techniques) were previously restricted to Universities with specialised equipment and personnel but are increasingly becoming more and more easily achievable for anyone or any business and more importantly to most at significantly lower costs than in previous years. There is the potential that commercial ventures and Universities could offer their services to screen corals for known (potential) pathogens and highlight risks before visual signs are observed. Conducting these trials before transportation or during the quarantine period will significantly reduce the risk of transporting specific pathogens around the world and further reduce the risks of these same pathogens being introduced into the wild. However, as most coral pathogens can be found in both healthy tissues and the disease lesions, as we discussed in Sections 2.1 and 2.2 (Klaus et al., 2005; Ritchie, 2006; Ritchie and Smith, 2004), there is a strong possibility that the pathogens are everywhere. In the light of this, it may be more important to monitor the conditions that the corals are kept in/transported in during the first few steps till retail. Keeping healthy corals will allow the corals themselves to fend off any opportunistic pathogens.

4.2. Technical means

Maintaining an aquaculture facility free of diseases does not mean keeping it pathogen free, as opportunistic pathogens may be found on a variety of environments including healthy corals (Bourne and Munn, 2005; Sunagawa et al., 2009), sediments (Richardson, 1997) and algae (Nugues et al., 2004). However the possibility of disease development from opportunistic pathogens should be taken into account right from the initial design of the aquaculture system. Aquaculture systems should be conceived in such a way as to prevent the spread of disease if it develops in any given section of the system. Keeping corals in replicate systems isolated from one another would therefore inhibit disease spread between systems as many diseases have high virulence with coral colonies nearest to the diseased colony most at risk of contracting the disease (Aeby et al., 2011b; Roff et al., 2011). Coral culture systems could be either completely isolated or semi-isolated (separated water source and culture tanks or culture tanks separated after the filtration system respectively). A completely isolated design would be safer but would also result in higher initial costs and further maintenance costs. A semi-isolated system would be less costly but if a pathogen entered the system and if the filtration/sterilisation equipment was not sufficient, the pathogen has the potential to spread to all culture tanks.

In order to prevent the introduction of opportunistic pathogens into an aquaculture system, it is important to have sufficient filtration and sterilisation of all incoming water and quarantine for all other inputs. Filtration can be chemical (such as activated carbon), mechanical (filters) or biological (live rocks), whereas sterilisation is commonly performed using UV light or through ozonation. Quarantine duration depends largely on the host organism being treated and/or the pathogen of concern; in fish for example quarantine lasts usually between two weeks and 90 days (Kent et al., 2009). However, often even despite quarantine procedures, potential pathogens can slip unnoticed into the aquaculture system as their growth can be controlled by beneficial microbes (Ritchie, 2006) until a stress causes a disturbance favouring pathogen development (e.g. Mao-Jones et al., 2010; Vega Thurber et al., 2009). For the same reasons, the use of prophylactic treatments should be avoided during quarantine, unless pathogen screening has identified a known pathogen for which an effective treatment exists. The careless use of prophylactic treatments might prevent the appearance of disease signs (Kent et al., 2009) and result in the introduction of infected organisms into aquaculture systems which were thought to be safe.

Disease development is often the result of degradation of environmental conditions which either affects the coral host directly or indirectly through modification of its environment or of its microbial/microalgal

symbioses. For example Raymundo et al. (2009) suggested that functionally diverse fish communities minimise coral disease development through the control of corallivorous fish which exacerbates disease development. Furthermore, increases in algal prevalence, as a result of environmental degradation for example, can also induce coral mortality through the release of compounds enhancing microbial activity (Smith et al., 2006). As such, maintaining healthy environments can provide an inexpensive prophylaxis measure. It is therefore crucial to monitor water quality parameters (e.g. temperature, pH, nutrient content) regularly. Furthermore, as highlighted by Kline et al. (2006) on top of "common" nutrient concentration measurements (e.g. nitrate, phosphate, ammonia), water quality monitoring should include measures of DOC and POC as these tend to drive coral disease development.

4.3. Genetic selection

Genetics play a key role on inter- and intra-specific resistance to disease, and this is a particularly important issue as aquacultured corals are usually reproduced by fragmentation of mother colonies, resulting in genetically identical clones. Coral susceptibility to infection of disease has been discussed in Section 3.1.1 along with the genetic aspect of variations between species and colonies specifically. As there is no guidebook with the "strongest" coral species, or what genotypes are more resistant to diseases, it is recommended to conduct an initial screening, searching for candidate colonies with enhanced immunity and growth. However, investigations aiming to identify immunity genes in corals are only just emerging (Hayes et al., 2010). Nevertheless, since immunity is often enhanced at the expense of growth and reproduction (Sadd and Schmid-Hempel, 2009), selecting fast growers to enhance coral production might be a risky strategy.

The widespread propagation by fragmentation of cultured corals has another important implication. Since fragmented colonies are genetically identical, genetic diversity will tend to decrease over time, and this can in turn negatively affect coral disease resistance (Nunes et al., 2009; Spielman et al., 2004). Managers will therefore need to weigh the advantages of selecting for coral genotypes with enhanced immunity against the drawbacks of seeing disease resistance decreasing with time as a result of the loss of genetic diversity.

5. Treating coral diseases

Various attempts have been made to treat coral diseases in ex situ cultures using a number of "home remedies" (e.g. tinctures of iodine, fresh water dips, addition of antibiotics) (Sweet et al., 2012). These are usually trialled by either hobbyists and reported in blog sites or by zoos and public aquariums. The most common method reported in the grey literature to eliminate a disease is fragmentation of the coral, which eliminates the diseased fraction. However, this does not necessarily mean that the pathogen will be eliminated from the aquaculture system as with regard to bacteria for example, apparently healthy tissue in advance of the lesion interface has been shown to have changeable diversity compared to healthy corals and may contain the pathogens specific to one or more disease (Cróquer et al., 2013; Sweet and Bythell, 2012). Little work is currently done on systematically trialling these potential cures in a scientific setting. However, work at Newcastle University (Sweet pers. obs.), in collaboration with the aquarium at Horniman Museum and Gardens in London is currently underway, with the aim of systematically testing suggested cures and developing a treatment for a range of coral diseases usable within aquaculture. Potential cures are currently being trailed against potential pathogens such as the bacteria, *Vibrio harveyi*, and an *Arcobacter* species, as well as ciliates, such as *Philaster* sp., all of which have previously been linked to diseases such as WS, BrB and BJS (Table 1; Fig. 1), in both aquaria and in the wild (Luna et al., 2007, 2010; Sussman et al., 2008; Sweet and Bythell, 2012).

Another treatment option currently being tested is the use of pro-biotic bacteria, which may provide a health benefit to the coral host (Teplitski and Ritchie, 2009). Probiotic bacteria may interfere with pathogens through interference of cell–cell signalling (Defoirdt et al., 2011; Hunt et al., 2012; Skindersoe et al., 2008), pathogen exclusion (Mao-Jones et al., 2010; Reshef et al., 2006) and the production of antimicrobials (Ritchie, 2006; Shnit-Orland and Kushmaro, 2009). As a result, Teplitski and Ritchie (2009) proposed that inoculating corals with beneficial bacteria initially isolated from the reef or bacterial viruses (known as phages) could protect them against pathogens and stressors.

Finally the most recent option is the use of phage therapy, which uses lytic bacteriophage viruses to highly selectively target specific coral pathogens (Efrony et al., 2007, 2009). However, their high specificity is a double-edged sword; on one hand, the property which makes them relatively risk free for environmental usage, namely their high specificity to certain bacterial hosts means that there is little risk of having the phage targeting non-pathogenic bacteria in symbiosis with the coral host. In contrast, this specificity means that this method can only be used for diseases for which the aetiology has been resolved and consists of a single pathogen, as many of the proposed pathogens are under great debate the usefulness of this technique is therefore limited at current times. That said, phage therapy has been applied successfully in the case of two coral diseases, 1) bacterial bleaching of *P. damicornis* by *V. coralliilyticus* (Efrony et al., 2007) and 2) white plague disease of *Favia favus* caused by *Thalassomonas loyana* (Atad et al., 2012; Efrony et al., 2009). These studies provide the only strong evidence linking specific causal agents to certain diseases/syndromes to date. Furthermore, Atad et al. (2012) took this process to the next level and were the first to successfully prevent both progression and transmission of a coral pathogen (*T. loyana* in this case) within the natural environment, a result showing high promise for the future treatment of coral diseases in both mariculture and ex situ aquaculture.

6. Conclusion and future studies

Diseases are a common problem of all aquaculture ventures and coral aquaculture is no exception. While much information is available on the origin and treatment of diseases in aquaculture species such as fish and crustaceans (Bondad-Reantaso et al., 2005; Defoirdt et al., 2011; Kent et al., 2009), our current understanding of coral diseases is still insufficient to provide managers with a comprehensive guidebook to prevent and/or treat diseases in coral aquaculture. While several promising treatments are being looked into, so far the best option to deal with diseases in coral aquaculture remains through prevention. Most importantly, it is imperative to develop a multifunctional toolbox for assessing disease state in corals. As stated by Work et al. (2008), methods applied in biomedical sciences, including rigorous anatomical description, use of appropriate diagnostic methods and controlled experimental design, should be applied in investigating coral diseases. Such a toolbox would be of particular use to aquaculture as it may allow for the discrimination of healthy and diseased colonies prior to introduction into culture systems. Molecular biology in particular may provide promising tools with potential applications in disease outbreak identification, as well as genetic selection of candidate coral species and/or colonies to culture. However, because the use of fragmentation techniques to propagate corals is still the most popular method in aquaculture facilities, managers will have to consider the costs of seeing disease resistance decreasing with the loss of genetic diversity (Nunes et al., 2009; Spielman et al., 2004). Nevertheless, progress is continuously being made to use sexual reproduction techniques to create large coral numbers to use in aquaculture (Petersen, 2008). Compared to fragmentation, this is a much more efficient method to produce a large numbers of corals. Furthermore, sexual reproduction will retain and even increase genetic variability within the cultured organisms.

Despite undeniable knowledge gaps, research into coral disease understanding, prevention and treatment is advancing quickly. However, as highlighted in this review, our understanding of these processes in coral aquaculture is currently rather limited, which underlines the need

for increased investment in collaborative research between scientists and hobbyists/aquarists. Such collaborations might allow for the development of significant improvements in coral culture system design and management.

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