

Ciliated protozoan occurrence and association in the pathogenesis of coral disease

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Abstract

Various microbial infections have significantly contributed to disease progression leading to the mortality of corals. However, the holobiont and the external surfaces of coral, including the secreted mucus, provide a varied microenvironment that attracts ciliates based on their feeding preferences. Besides, some ciliates (e.g., *Philasterine scuticociliate*) may enter through the injuries or lesions on corals or through their indirect interactions with other types of microbes that influence coral health. Thus, ciliates occurrence and association are described with 12 different diseases worldwide. White syndrome disease lesions have diverse ciliate associations, and higher ciliate diversity was observed with diseased genera *Acropora*. Also, it was described, about sixteen ciliate species ingest coral Symbiodiniaceae and histophagous ciliates for coral tissue loss as secondary invaders. However, the ciliates nature of association with the coral disease remains unclear for primary or opportunistic secondary pathogenicity. Herein, we explore the urgent need to understand the complex interactions of ciliates in coral health.

Keywords: ciliates, corals, disease, heterotrophy, infection, microbes, mixotrophy.

1. Introduction

Eukaryotic marine ciliates transmit directly or indirectly into the diseased corals and devour the damaged tissues [1-8] (Fig. 1). Skeleton-eroding band (SEB) was the first disease described to be caused by the colonial, heterotrich ciliate *Halofolliculina corallasia* [9]. *H. corallasia* is also associated with Black band disease provoking tissue loss in affected corals [10]. In addition, these *Halofolliculina* ciliates were often described from Caribbean reefs, affecting over 26 Caribbean reef-building coral species, afterward termed as Caribbean Ciliate Infection (CCI) disease [11,12]. Another ciliate, *Philaster guamense* (formerly termed as *Porpostoma guamense*) dominate lesions of brown band disease (BrB) [2,7,13-16]. White Syndrome (WS), the most ubiquitous disease, is also characterized by numerous ciliate community associations within their lesion interface [7]. Thereof, various ciliate associations with the varied diseased hosts with multiple diseases such as White Band Disease (WBD), White Plague (WP), Brown Jelly Syndrome (BJS), and other BrB-like syndromes of the Caribbean are described [14,15,17,18]. Thus, earlier reports demonstrated that about 28 ciliate species were associated with varied coral diseases and among which six ciliate species have been described for engulfing the coral symbiotic algae [14]. Even though distinct investigations had related various ciliate species to particular coral disease signs and symbiont ingestion, only a few highlights their approach towards disease progression [4,7-9,14-17,19,20] (Fig. 1). Thus, no distinct role of ciliates in coral pathobiology and their pathogenesis over the immune-compromised tissues are described in detail [18]. For example, although ciliates have been described to be associated with Porites white patch syndrome [21], Koch's postulates has been proven with the bacterial pathogen *Vibrio tubiashii* [21]. The difficulty therefore remains around the mixed ciliate communities often found present and teasing out the roles these individual species play in coral disease (if any) [7,22] (Fig. 2). Moreover, there is no clear experimental demonstration for their possible role as primary or secondary causative agents, even though some ciliates are known for directly consuming symbiotic algae [6,20,23]. Thus, the ciliate diversity with other microbial pathogens was comprehensively reviewed for their possible role in disease progression in various coral diseases described worldwide.

2. Ciliate occurrence and association with coral diseases

2.1 Skeleton-Eroding Band and Caribbean ciliate infection

Skeleton-eroding band (SEB) was the first coral disease where ciliates were identified as important in the disease progression caused by protozoan ciliate from the Indo Pacific reefs [9]. The condition is macroscopically visible as a black line or band that progresses over the healthy coral tissues. However, SEB exhibits slow progress rate of 1 mm per week compare to that of the BBD that

progress rapidly as 1 cm per day [9,24]. The black band formation is due to a dense association of sessile foliicolinid ciliate, *Halofolliculina corallasia* [25-28]. As the *H. corallasia* cell divides, daughter cells move to the front edge of the dark band and secrete a protective shell called lorica. During the secretion of lorica, ciliate kill the polyps and erodes the skeleton [9,10]. The affected tissue is noticeable with small black spots composed of clustered empty lorica, a feature that clearly distinguishes SEB from BBD and leaves behind a bright white skeleton [9]. SEB was demonstrated for its transmission through direct physical contact to the healthy corals [9]. Besides, injured corals were found colonized by *H. corallasia*, which was experimentally confirmed through 100% of artificially created injuries colonized by ciliates within 10 days [3]. SEB affects several massive and branching corals belonging to Acroporidae and Pocilloporidae families of at least 22 genera [10] and 31 species [13] in the Indo-Pacific and the Red Sea [3,9,10,13,14] (Table 1) and had not been reported in the Caribbean [9]. However, a foliicolinid ciliate interacting with over 25 scleractinian coral species was identified as unique and different from that of *H. corallasia* [29,30] (Table 1). Even though the Caribbean ciliate species is yet to be identified, the name Caribbean ciliate infections (CCI) has been given due to the apparent differences in etiology [4,31].

CCI is associated with rapid tissue loss and *Halofolliculina* infection damaging a multitude of the coral host belonging to six families (Poritidae, Faviidae, Meandrinidae, Astrocoeniida, Acroporidae and Agaricidae) of twenty six species [30,32]. CCI are prominent by grey or black band (several mm to 1-2 cm thick) exhibiting salt and pepper appearance on the acute infection [33]. Both CCI and SEB achieve pathogenicity with host tissue mortality with similarities of Caribbean and Indo-pacific ciliates such as their sessile nature, lorica secretion, and clinical signs on their hosts [9,29,30]. Based on these similarities and examination of SEB and CCI infected tissues, it has been reported that both the pathogens are the same, though no evidence to support this conclusion [14,26,29,30] (Fig. 3 and 4; Table 1). However, SEB is more commonly observed in polluted and warmer water [9,10,13], while the Caribbean ciliates were described from oceanic than the human-disturbed coastal reefs [11]. Also, Croquer et al., [30] suggested that transmission of ciliates through the presence of injury in the colony or directly through the water column. The susceptibility also confirmed through field and laboratory based study, and the tissue of ciliate infected *Agaricia tenuifolia* colonies lost ten times faster than the healthy colonies [4]. The ciliates population growth was found higher in coral colonies removed from the deeper depths than at the depths of shallow waters [4]. Besides, tissue mortality of SEB ranged from 0.1 to 1 mm d⁻¹ [9] and from 2 to 3 mm d⁻¹ for the Caribbean infections [3], Page et al., [28] reported that skeletal erosion is often associated with SEB and not with the CCI. Although the

differences with clear etiology and above dissimilarities described CCI as different from SEB [4], more confirmatory studies are required to clarify the confusion between CCI and SEB [26].

2.2 White Band Diseases

White Band diseases (WBD) have been described for their high host specificity, affecting only *Acropora cervicornis* and *A. palmata* in the Caribbean region [34]. However, a substantial number of observations of WBD have been made outside of Caribbean (in eight countries) with thirty three species of massive, branching, and plating corals instead of Elkhorn and Staghorn corals [35]. WBD is an advancing layer of diseased and necrotic tissues that advances rapidly from colony base and up towards branch tips leaving bare, coral white skeleton intact [36]. This disease is classified as WBDI and WBDII, where WBDII is distinguished by a band of bleached tissue that precedes the necrotic tissue [37]. WBDI affects both the *Acropora palmata* and *A. cervicornis* in the Caribbean [38], whereas WBDII has been reported exclusively as *A. cervicornis* in the Bahamas [34,39]. Besides, independent studies have identified different putative WBD pathogens, *Vibrio carchariae* has been identified as a putative pathogen causing WBDII [39,40], while marine Rickettsia-like bacteria [41] and bacteria [17,37] for WBDI. However, no study had satisfied Koch's postulates. Thus, multiple pathogens (viruses, protists) combined with bacteria or singularly could elicit WBD signs [17,41]. Thus, the ciliates observed consistently in the diseased corals and not in healthy corals are described as the likely secondary pathogens [17]. A diverse community of about nine ciliates with WBDI diseased corals (*A. cervicornis*) samples of Caribbean regions [7] (Figs. 3 and 4; Table 1) and about 13 ciliates from Venezuela and Columbia samples were reported from the diseased and not in healthy (Table 1). Although some variations were there in the occurrence of ciliates, a histophagous ciliate, *Philaster lucinda*, is consistently detected in all samples of the diseased in the thirty six cases of Caribbean WBD and sixty seven cases of WS of Indo-pacific [7,14,17,42,43] and within aquaria [44] (Fig. 4; Table 1). Besides, *P. lucinda* is observed to consume intact coral tissues and engulf the symbiotic algae [17]. However, the *P. lucinda* knockdown using metronidazole in WBD and WS experiments suggests histophagous *P. lucinda* for secondary pathogen in WBD, which may be also similar with WS [17,43], contributing significantly for the pathogenesis of WS and WBD [17]. Thus, the histophagic activity and the consistent association of the *P. lucinda* with both WBD and WS indicates these two diseases may be a similar disease [17]. Other dominant histophagous ciliate *Varistrombidium kielum* was not detected consistently with all the diseased samples. Thus, *V. kielum* and other diverse communities of ciliates are described as secondary invaders of WBD [17]. Moreover, most of the WBD ciliates of specific and non-specific associates were detected (Table 1) in corals showing signs of WS, BrB, and PWPS [14] (Figs. 3 and 4).

2.3 White Syndrome

White syndrome (WS) is a progressive tissue loss with a sharp distinction between a denuded skeleton and healthy tissues without other disease symptoms [13]. WS disease signs were initially observed in the Indo-Pacific scleractinian coral diseases [13] and described their causes on varied Indo-Pacific corals [45]. Later, it was also observed in Caribbean corals [46,47]. Several etiological agents, including *Vibrio* spp. [13,44,48-50], Cyanobacteria [47], Viruses [47], fungi [47], ciliates [7,51,52], Helminths [52], Intraspecific chimeric parasites [7,52] and apoptosis [53] were described for their association with WS. WS is not due to apoptosis or infection by these organisms as they are deducted in low levels without any consistency [47], but the rapid progression of WS lesions in Hawaiian coral *Montipora capitata* may be due to the associated ciliates [52]. Furthermore, various ciliates association with WS lesions are reported and described [7,14,44,47,51,52] (Fig. 4; Table 1). These ciliates are absent in apparently healthy tissues. Ciliate ribotypes similar to *Diophrys* sp., *Euplotes* sp., *Aspidisca* sp., *Pseudocarnopsis* sp., *Glaucanema* sp., *Philaster* sp., and *Varistrombi* are observed with WS of Heron Islands, Great Barrier Reef (GBR) [44] (Fig. 4; Table 1). The investigation of occurrence of similar bacteria and ciliates of the WS and BrB lesions of *A. muricata*, Heron Islands proposed that the two diseases are same (7). However, ciliates associated with the lesions significantly differentiate the WS from BrB macroscopically with low ciliate densities at the lesion border [45]. Ciliate species such as *Philaster lucinda*, *Holosticha diademata* (Fig. 5), and a *Pseudokeronopsis* were detected in all samples of WS lesions of genera *Acropora muricata* (Fig.3-4), *A. aspera*, and *Pocillopora damicornis* [14,42] (Table 1). Similarly, *Philaster* sp., a *Pseudokeronopsis* sp. and a *Cohnilembus* sp. (Fig. 5) had exhibited association in diseased fragments of *Echinopora lamellose* [43]. However, ciliates are known to occur as a secondary invader after the primary infection of bacteria that weakens the coral defences. Ciliates, *Philaster* sp., *Euplotes* sp., and *Varistrombidium* sp. have been characterized for their Symbiodiniaceae ingestion associated with WS lesions (Table 2).

2.4 White Plague

White Plague (WP) is a rapid tissue loss disease that has affected multiple species of Indo-Pacific and Caribbean corals [54]. Based on the tissue loss progression rates WP is classified into three types WP (I, II, and III) [54-56]. Microbiological studies on causative agents of WP type II were hypothesized as bacterium *Aurantimonas corallicida* in coral *Dichocoenia stokesii* [54,57]. However, the different bacterial association with WP occurred in various regions such as *Thalassomonas loyana* in the Red Sea [58,59], neither *A. corallicida* nor the *T. loyana* was detected (using 16S rRNA gene microarrays) in the WP like diseases of Koh Tao, Thailand [60]. Also, 16S rDNA sequencing indicated that *A. corallicida* is not associated with a WPL-like disease [61] and a coccoid bacterium [62] instead

of *A. coralicida* in Caribbean corals. The above different bacterial associations in other regions suggest that there is indeed more than one causative agent associated with the various WP diseases [34]. Thus, later small circular ssDNA viruses (SCSDVs) [56] and ciliates [3,14,18] association with WP diseased tissue has been reported. *Philaster* sp. *Paraphilaster* sp. and a ciliate belong to order – Prostomatida has been described from the Caribbean [18] and *Halofolliculina* sp. from Indo-Pacific [3] (Table 1). Also, about 14 ciliates are associated with diseased corals of *Colpophyllia natans* and *Orbicella annularis* from Venezuela and Columbia [14] (Fig. 3; Table 1). The genomic sequences of ciliates of Indo-Pacific and Caribbean were slight variations due to the geographic variations [14]. Further, from regions of Venezuela and Columbia reefs, various species of ciliates associated with the disease lesions are identified [14] (Table 1).

2.5 Porites White Patch Syndrome (PWPS)

PWPS has characterized patches (20-100 mm) of discoloration (with no associated tissue loss) on colonies of *Porites lobata* on the GBR [63]. Besides, PWPS was also with patches (50 – 300 mm) of circular to oblong tissue loss on massive colonies of *P. lutea* on Western Indian Ocean (WIO) reefs [64]. 16S rRNA sequencing of Healthy and PWPS-infected *P. lutea* coral colonies revealed several putative bacterial pathogens in the PWPS tissues and no or less in the healthy tissues [65]. In addition [66] suggested that Virus-like particles (VLP) infecting the corals or its symbiont may be the causative agent of PWPS. However, Koch's postulates have been satisfied with the bacterial pathogen *Vibrio tubiashii* [21]. PWPS tissues of three locations were analyzed for the ciliate association and observed for the presence of six species of ciliates [14] (Table 1). Among these, except *Paracineta limbata*, five were also detected in WS tissues [14] (Fig. 4; Table 1).

2.6 Black Band Disease

A black mat of complete tissue degradation characterizes black band disease (BBD). The causative agents include the microbial consortium of a filamentous cyanobacterium (*Phormidium corallyticum*), numerous heterotrophic bacteria [67], marine fungi [68], and both sulfide-oxidizing (*Beggiatoa*) and sulfate-reducing (*Desulfovibrio*) bacteria [55,69]. The cyanobacterial dark bands of 1 mm thick and 1 to 3 mm wide advance rapidly across the coral colony, destroying healthy polyps and leaving behind dead skeletal surface intact [70,71]. Cyanobacteria such as *Leptolyngbya* sp., *Oscillatoria* sp., and *Geitlerinema* sp. encompasses microcystin that exhibits toxicity in tissue damage [71,72]. The infected tissues are further broken by a micro-community that includes bacteria and ciliated protozoans. Thus, decaying coral tissues of *Montastrea annularis* and *Diploria strigma* are devoured by ciliates of genera *Propostoma* and *Philaster* on barrier reefs of Bermuda and Belize, being

prevalent in July, September and May, June respectively [24] (Fig. 4; Table 1). They hover over the *Phormidium* mat and decaying coral tissue mash devouring their Symbiodiniaceae [24] (Fig. 4; Table 1- 2).

Likely, corals of seven species held consistently low ciliate diversity despite the disease size and appeared to have no difference in ciliate diversity between location and species among five ciliated protozoans from the reefs Indo-Pacific and Caribbean (Table 1). *H. diademata* (Fig. 5) and *Protocruzia adherents* are bacterivores colonizers associated with dying coral tissues. At the same time, *Philaster lucinda* grazes over the algal Symbiodiniaceae internally by histophagous activity [14,17] (Table 2). The ciliate *Halofolliculina corallasia* is also associated with 6 % BBD infections on the Great Barrier Reefs [73]. However, their co-occurrence in the BBD mat is unclear to understand its role in disease progression [73].

2.7 Brown Band Disease

Brown band disease (BrB), being reported initially on Great Barrier Reef, Australia, is prone to affect salient coral families such as Pocilloporidae, Acroporidae, and Faviidae (Table 1) [13,16]. BrB is brown-coloured zones of different widths forming the boundary of healthy coral tissue with disease exposed white skeletons [13]. The brown colouration is owed by the aggregation of ciliates in massive amounts with few foraging on coral Symbiodiniaceae (Table 2) [5]. Based on the quantity of ciliates the coloration can range from brown to white, which led to the resemblance of white syndrome ciliates infection [18,42,51]. Also, Sweet and Bythell [7,44] found similar ciliates and bacteria were consistently present in lesion of both WS and BrB on *Acropora* sp. from the GBR. Thus, the authors hypothesized [7] and confirmed [44] that firstly, the similarities of bacterial and ciliates associated with the WS and BrB to consider both the disease are same. Secondly, the ciliate communities appeared responsible for the disease sign i.e. that of rapidly advancing tissue loss leaving behind the white denuded skeleton. Thirdly, authors arrive at two alternative hypothesis, that is, one with bacteria as the primary invaders and the ciliates follow engulfing the dead tissues. While the other with ciliates are the primary casual agents and the bacteria act as the secondary invaders in the stressed corals due to the result of ciliate pathogenesis [7,44]. Besides, physical injury as a result of predation also influence recruitment of pathogens (bacteria and ciliates) in promoting BrB disease [5,8,19,20,23,44]. Colonies of *Acropora cytherea* on the coral reefs of Derawan Island, Indonesia, preyed upon by *Acanthaster planci* (crown-of-thorns starfish (COTS)) are the first linked to feeding scars that showed a high incidence of BrB [5]. Moreover, in an aquarium-based experiment, a gastropod (*Drupella* sp.) feeding the coral is noted to be a highly effective BrB vector exhibiting corallivorous activity that contributes to wounds sufficiently facilitating infection by ciliate as secondary invaders [8]. In another aquarium-

based experiment the pathogenicity of *Philaster guamense* (formally described as *Porpostoma guamense*) on fragments of injured and COTS predation that develops into BrB on the compromised *Acropora hyacinthus* has been demonstrated [19]. These results indicate opportunistic pathogenicity of ciliate that migrate rapidly, colonizing only the compromised coral tissues to rapid coral mortality. Besides, the tropical cyclones, storms, and COTS outbursts are prone to increase coral injury incidence heightening BrB disease and further declining coral cover [19].

Philaster guamensis [7,16] feeds on compromised coral tissues of *Acropora muricata*, *A. aspera*, *A. acuminata*, and *A. surculosa* leaving only intact white skeleton (Figs. 3 and 4). Ciliates enter injury sites resulting in signs of BrB disease within 24 hours and rapid tissue losses within 60 h, claiming rapid tissue loss was by the virulence of *Philaster guamense* (formally described as *Porpostoma guamense*) [19]. An exponential rise in tissue loss was found proportional to a rapid increase in ciliates [18]. This ciliate at high densities often acts as a primary pathogen devoid of around 88% of coral tissues [2,7,13]. Likewise, although varied ciliates occurred with BrB of *A. muricata* in Indo-Pacific reefs [14] (Fig.4; Table 1), the primary ciliates that involved in pathogenesis of BrB reported with different names and conflicting results [2,7,8,16]. Thus, a thorough in situ study assessing how frequently ciliates find and colonise injured corals confirmed that the *Philaster guamensis* can cause extensive tissue loss by rapidly establishing BrB [19,44]. Hence these ciliates of BrB that scavenge on coral tissue leads to swift tissue loss may be resulting in mortality of the matured coral recruits [6,19,44].

2.8 Brown Jelly Syndrome

Brown jelly syndrome (BJS) is an aggregation of brown jelly material afloat on a coral surface. It's a disease of aquarium corals influenced by protozoan ciliates that feed on live coral tissues (Table 1 and 2) [22]. BJS is a fast and contagious tissue loss disease associated with tissue necrosis, extensive visible swelling, and mucus production [22]. In brown jelly, high ciliate densities in *Siderastrea siderea* fragment consumed coral tissues leading to corals death in less than 24 h [18]. This observation was found consistent with rates of tissue consumption by *Philaster* sp. of acroporid corals with BrB disease in Guam [19] and aquaria [2,19]. In other study on aquaria-maintained *Orbicella faveolata* and *S. siderea*, a sequential disease progression with signs of coral diseases resembling white plague, brown-band, and brown jelly appeared with three visually distinct ciliates associations. Among them, two belong to the family Philasteridae and resembled the genera *Philaster* and *Paraphilaster* and one to the order Prostomatida like ciliate [18] (Table 1). In aquarium, WS and BJS were thought to be caused by a ciliate historically referred to as *Helicostoma nonatum*, however, this was miss-identified based on morphological observations alone [13, 74-77] (Figs. 3 and 4; Table 1). Sweet et al., [22] first

indicated that this species was actually more closely related to the genus *Philaster*, and that of *Philaster digitiformis* specifically (Table 1). This has since been further re-classified and named as a novel ciliate species associated with corals in the aquaria and in the wild - *Philaster lucinda* [17] (morphology aligned with genetic evidence). Also, in the aquarium maintained *Acropora* spp., *Philaster digitiformis* (Table 1) was observed beneath diseased coral tissues with the endosymbiotic algae inside the cells (Table 2), which suggest that this ciliate actively consume the coral tissues. Also, no ciliates were observed in the healthy corals [22].

2.9 Caribbean Yellow Band Disease

The Caribbean constitutes a hotspot to the coral disease of 66 % worldwide though it supports only 8 % of coral reefs globally [35]. Caribbean Yellow blotch /band disease (CYBD) affects large reef-building corals throughout the Caribbean and is not similar to yellow band disease [34,78]. CYBD advances into pale yellow blotch over coral tissue, expanding gradually into a band, spreading (0.5 – 1.0 cm) monthly [78,79]. It typically disrupts symbiosis between the coral host and endosymbiotic algae [79]. Their paleness is represented by a lack of Symbiodiniaceae with decreased chlorophyll content, carrying 50 % Symbiodiniaceae density of 75 % reduced chlorophyll pigments a, c2, and 80 % fewer division rates at the instance of raised water temperatures [78]. Hence, it is a disease that affects Symbiodiniaceae [80]. The possible causative agents are described as the consortiums of four *Vibrio* spp. causing symbiont algal dysfunction and disorganization of membrane-bound thylakoid compartments [81,82]. However, only a very few protozoan ciliates of four species are described for their association with CYBD from colonies of *Montastraea annularis* in Venezuela [14] (Figs. 3 and 4; Table 1). Amongst *H. diademata* (Fig. 5), *Trochiloides recta*, and *Protocruzia adherents* are likely bacterivores colonizers associated with dying coral tissues [14]. No ciliates are suspected of harbouring the Symbiodiniaceae. Thereof possibly, these ciliates might be secondary colonizers of the diseased coral fragments.

2.10 Grey-Patch Disease

Grey patch disease (GPD) is a poly-microbial disease with grey biofilms of different-sized thin mats as multifocal lesions over the coral fragment [83]. The diseased colonies range from 5 cm² areas to 1 m² patch over the large affected *Porites* colonies. GPD symptoms on 18 different coral species of 10 other genera associated with cyanobacterial species such as *Symploc*, *Entophysalis*, *Lyngbia*, several *Phormidium* sp., Rivulariaceae, two pathogenic fungi (*Cladosporium* and *Fusarium*), and loricate folliculinid ciliates (Fig. 4; Table-1) [83]. GPD exhibits resemblance with the well-described black band disease [83]. Besides, multiple microbes such as loricate ciliates, fungal hyphae,

and cyanobacteria invade the coral skeleton through diseased tissue lesions hampering the coral defenses leading to disease severity [83].

2.11 Fungal-Protozoan Syndrome

Fungal-protozoan syndrome (FPS) is the loss of Coenenchyme tissue, pigmentation, and increased mucus production in infected corals [34]. The coenenchyma tissue of coral polyps is colonized by fungi leading to dead tissues, further occupied and consumed by an unspecified group of protozoan ciliates [34]. *Trichoderma*, the fungi associated with FPS, although *Cladosporium*, *Humicola*, and *Penicillium* are also consistently associated [84]. However, their role in disease initiation is not hypothesized, and hence, the fungi and protozoan attendants may be opportunistic rather than primary pathogens. Distinct coral species affected by FPS are *Paramuricea clavata*, *Eunicella cavolinii*, and *Eunicella singularis* [84].

The above coral disease associated ciliates distribution have been described in many reefs in both the Indo-pacific and Caribbean regions. In most of the cases, physical injury through abiotic factors due to temperature rise, anthropogenic (recreation, nutrient loading), and invertebrate predation (Starfish and gastropods) are the common factors that influence the ciliate intrusion as secondary invaders in the immunocompromised corals. Although Sweet and his group [7,44] and Katz et al., [19] demonstrated the ciliates for the primary pathogen in the aquaria, which is yet to be confirmed in field conditions. Also, most of the aquaria experimental studies consistently revealed that the ciliate proliferate only in the injured and not in the healthy corals. The present study had revealed that the association of *Philaster lucinda* with eight different coral diseases followed by *Holosticha diademata* with seven different diseases (Fig. 7). The ciliates *Diophrys* sp., *Euplotes* sp. and *Philaster guamensis* are spotted only among the diseases such as BrB and WS of Indo-pacific regions, in Great barrier reef and other islands of Australia (Fig. 7). Hence these ciliates can be considered as region specific and their physicochemical conditions might be the reason for the ciliate prevalence. Also, among the twelve ciliate associated coral diseases, WBD in the Caribbean, WS in Indo-pacific and WP from both regions are described with high number of ciliate invasions (Fig.7, Table 1). Although various investigations on ciliate communities association with coral disease have been characterised through visual descriptions, no ciliate pathogens have been identified for satisfying Koch's postulates, which are used to identify the etiological agent.

3. Ciliates ingestion of symbiotic algae

Various ciliate species were known for engulfing different size categories of prey such as bacteria, pico-, and nanoplanktons, diatoms, dinoflagellates. Also, ciliates were well demonstrated for

their combination of heterotrophy and phototrophy through mixotrophic forms, e.g. *Mesodinium rubrum*. Although the symbionts and the plastids of acquired phototrophs were acquired through specific phagotrophic pathways, most retain only the chloroplasts and not the complete algal cells [85]. Ciliates association with corals were often described for their opportunistic predation on coral dead tissues and microbial communities of coral holobiont (Video S1-S3). However, ciliate ingestion of coral endosymbiont, symbiodiniaceae members are less described from coral tissue either by histophagous or carnivorous [17,85,86]. Nine ciliate ribotypes were found associated with BrB and WS. Among which four ciliates contained Symbiodiniaceae, suggesting coral tissue ingestion, and ciliates such as *Philaster* sp. were also seen underneath tissue lesions [7] (Table 2). A BrB ciliate of class Scuticociliata described for its voracious scavenging on Symbiodiniaceae of *Acropora muricata* and *A. hyacinthus* from diseased polyps with approximate ingestion of more than 50 symbiodiniaceae members per ciliate [16,20,23,87]. Also, decaying coral tissues (Octocorals) and Symbiodiniaceae are devoured by histophagous ciliate, *Philaster* on barrier reefs of Bermuda and Belize [24]. *Philaster guamense*, *Philaster lucinda*, and *Varistrombidium* sp. were also reported for ingestion of coral symbiont through histophagy of *Acropora surculosa*, *A. palmate*, *A. cervicornis* tissues, respectively [2,7,14] (Table 2). *Trachelotractus* sp. and *Condylostoma* sp. are also noticed to forage Symbiodiniaceae of *Acropora* sp., *A. cervicornis*, and *A. muricata* [14] (Table 2). Two other ciliates (*Holosticha* sp. and *Cohnilembus verminus*) (Figs. 5 and 6) described for their voracious feeding on Symbiodiniaceae were also associated with diseased /immunocompromised *Acropora* sp. and *Porites* sp. [15,19] (Table 2) (Video S1-S4). Likewise, marine ciliate *Euplotes uncinatus*, *Paraeuplotes tortugensis*, and a heterotrichea ciliate *Maristentor dinoferus* are reported for their association with symbiodiniaceae (Table 2). Around 50– 800 dinoflagellate species of genus *Symbiodinium* of clade C are observed within a single *Maristentor dinoferus* for symbiotic exchange of photosynthates and nutrients [88-90]. Besides, ciliates ingested Symbiodiniaceae in BrB disease on *A. muricata* remain photosynthetically competent and uncompromised by disease progression. In this scenario, ciliates benefit from photosynthate and photosynthetic oxygen, which alleviates their diffusion limits of oxic respiration over closely populated disease lesions [16,20]. Further, the ingested Symbiodiniaceae remains intact within cytostome without food vacuoles covered and maintained actively inside ciliate for 2-3 days and then digested [2,17,20,82]. Thus, the various reports of ciliate heterotrophy or mixotrophy feeding in coral reefs were the known examples of marine ciliates that host true endosymbionts. However, no precise details are described regarding their parasitic or symbiotic relationship to corals or their prevalence in coral reef ecosystems [85]. Also, various clarifications are yet to be addressed, such as why do ciliates ingest coral endosymbiont on the instant of a disease outbreak and not in a healthy condition?. What type of interactions and effects on coral health ciliates

do participate after engulfing the algae?. Further, it is unclear that ingestion of Symbiodiniaceae by the ciliate occurred during feeding live or dead tissues of coral (carnivorous/histophagous) or attains from elsewhere (algivorous) [16].

4. Future directions

Coral–ciliate interactions must be examined under laboratory conditions, for which ciliate communities associated with corals must be cultured under laboratory conditions. Culturing of these mixotrophic and heterotrophic ciliates may be used as a model in understanding coral ciliate interactions in pathogenesis. These include the ciliate invasion to corals for opportunistic pathogens, histophagous, bacterivores in disease progression, and nutrient cycling of the dead tissues. The mixotrophic and heterotrophic ciliates different feeding patterns and interactions has to be examined to understand their possible beneficial and harmful effects on corals and coral holobiont. This can be achieved by using Epifluorescence microscopy, an important technique that allows the detection of plastids and the presence of specific photosynthetic pigments. Also, easy discrimination between heterotrophic and photosynthetic ciliate forms by this technique will be helpful for the identification of mixotrophic ciliates that interacts with the corals. Thus, examining the organelle-sequestering ciliate cultures may aid in understanding the ciliate's ability to maintain plastids. Ciliate cultures can also be explored to investigate the exchange of nutrients, inorganic and organic material disposal, and genetic exchange. This knowledge could be used to understand the possible roles of ciliates in disease transmission and control of the spread of diseases. Finally, ciliate cultures might also aid to understand the feed specificity of ciliates associated with the corals. This may assist in understanding the host specificity, if any, in the biochemical interactions that affect coral health.

5. Conclusion

Ciliates are mainly demonstrated as secondary opportunistic pathogens colonizing immune-compromised coral tissues leading to coral mortality. However, some ciliates are also demonstrated as the primary pathogen in the aquaria conditions, which were yet to be confirmed under field conditions. Ciliates in the act of scavenging tissue lesions, ingest coral Symbiodiniaceae and keep them intact to be photosynthetically competent so that it serves productive to the host. Thus far, sixteen ciliate species are determined to forage coral Symbiodiniaceae and also 34 different ciliate species are described to intrude over twelve varied coral diseases. Acroporidae, Pocilloporidae, and Faviidae are the most susceptible families of scleractinian corals. Thus, ciliates associated with signs of various coral diseases should be further detailed regarding the possible mode of interactions. Ciliated protozoans interactions with corals are largely underrepresented due to insufficient evidence suggesting the

mixotrophic or heterotrophic ciliate role as an etiological or symbiotic agent. To be clarified, pure ciliate cultures are required for infection of healthy corals. Besides, the study on mixotrophic or heterotrophic ciliates, along with other microbes such as bacteria, fungi, viruses associated with host coral, should yield insights into the causes of the disease.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Figure legend

- Fig. 1. Ciliates association in coral disease progression. Ciliates act histophagous over the onset of physical injury or bacterial infection to coral. Temperature rise, anthropogenic activities (recreation, nutrient loading), and invertebrate predation (Starfish and gastropods) cause bacterium and other microbial infections, steering a debilitated physiological condition to corals (A). Ciliates exhibit these uncompromised coral tissue lesions as secondary invaders devouring dead and decaying coral tissues, causing tissue loss (B). Some ciliates voraciously ingest the coral endosymbiont, Symbiodiniaceae associated with the immunocompromised corals (B, C). Healthy corals were not affected by any ciliates and were observed with intact symbiodiniaceae –host association.
- Fig. 2. Healthy and diseased corals a) Healthy *Acropora* sp. b) Macroalgal overgrowth on the denuded and diseased *Acropora* sp. c) Healthy *Porites* sp. d) Diseased *Porites* sp. with pink line syndrome surrounding the dead tissues e) Healthy *Acropora* sp. f) Macroalgal dominance in the diseased or dead *Acropora* sp.
- Fig. 3. Ciliates association with different diseased corals. Thirteen other coral genera are described to be associated with ciliates. Different ciliate species associated with diseased lesions of the various coral genus such as *Acropora* spp., *Porites* spp., *Orbicella* spp., *Colpophyllia* spp., *Diploria*., *Montastraea* sp., *Goniopora* spp., *Pocillopora* sp., *Siderastrea siderea*, and *Agaricia* sp. Ciliate diversity was observed more with *Acropora* spp.
- Fig. 4. Coral disease-associated ciliate diversity. A different group of ciliates associated with various coral disease outbreaks such as White syndrome (WS), White plaque (WP), White band disease (WB), Skeletal eroding band (SEB), Brown jelly syndrome (BJS), Brown band (BrB), Black band (BB), *Porites* white patch syndrome (PWPS), Caribbean yellow band (CYB) and Caribbean ciliate infection (CCI). Ciliate diversity was observed more with White syndrome.
- Fig. 5. Ciliates and their life stages isolated from diseased *Acropora* sp. and *Porites* sp. Different life stages of ciliates, (a) *Holosticha diademata* adult (b) *H. diademata* larvae (c) *H. diademata* division (d) *Cohnilembus verminus* adult (e) *C. verminus* larvae (f) *C. verminus* division (g) *Uronema marinum* adult (h) *U. marinum* larvae (i) *U. marinum* division (j) *Euplotes* sp. adult (k) *Euplotes* sp. larvae (l) *Euplotes* sp. division. Protozoan ciliate communities such as *Cohnilembus verminus*, *Uronema marinum*, *Holosticha diademata* and *Euplotes* sp. were observed for their association with diseased *Porites lutea*. Diseased *Acropora* sp. were observed with ciliates such as *U. marinum*, *H. diademata* and *Euplotes* sp. No ciliates were observed or isolated from the healthy *Porites* sp. and *Acropora* sp.
- Fig. 6. Devouring of dead and decaying coral tissues along with coral endosymbiont. (a) *Cohnilembus verminus* feeding the dead and decaying coral tissues. (b) *C. verminus* entering beneath the coral tissues in search of feed. (c) *C. verminus* stretches the front tubular portion to engulf the dead and decaying tissues. (d) *C. verminus* was found associated with tissues of endosymbionts (arrows) and also ingestion of symbiodiniaceae (arrows) in the tubular mouth regions.
- Fig. 7: Ciliate associated coral disease in the Indo-Pacific and Caribbean regions. (A) Distribution pattern of ciliates association with different coral diseases in regions such as Indo-pacific and Caribbean. (B) occurrence and association of dominant ciliates with different individual coral diseases of Indo-pacific and Caribbean regions.

Supplementary

Video – S1. *Cohnilembus verminus*- adult *C. verminus* isolated from *Porites* sp. *C. verminus* feeding on dead and decaying coral tissues of *Porites* sp.

Video – S2. *Holosticha* sp. – Adult *Holosticha* sp. isolated from both *Acropora* sp. and *Porites* sp. feeding on the dead and decaying tissues of both *Acropora* sp. and *Porites* sp.

Video – S3. *Uronema marinum* – Adult *Uronema marinum* isolated from both *Acropora* sp. and *Porites* sp. feeding on the dead and decaying tissues of both *Acropora* sp. and *Porites* sp.

Video – S4. *Euplotes* sp.- isolated from both *Acropora* sp. and *Porites* sp.

Figure 1

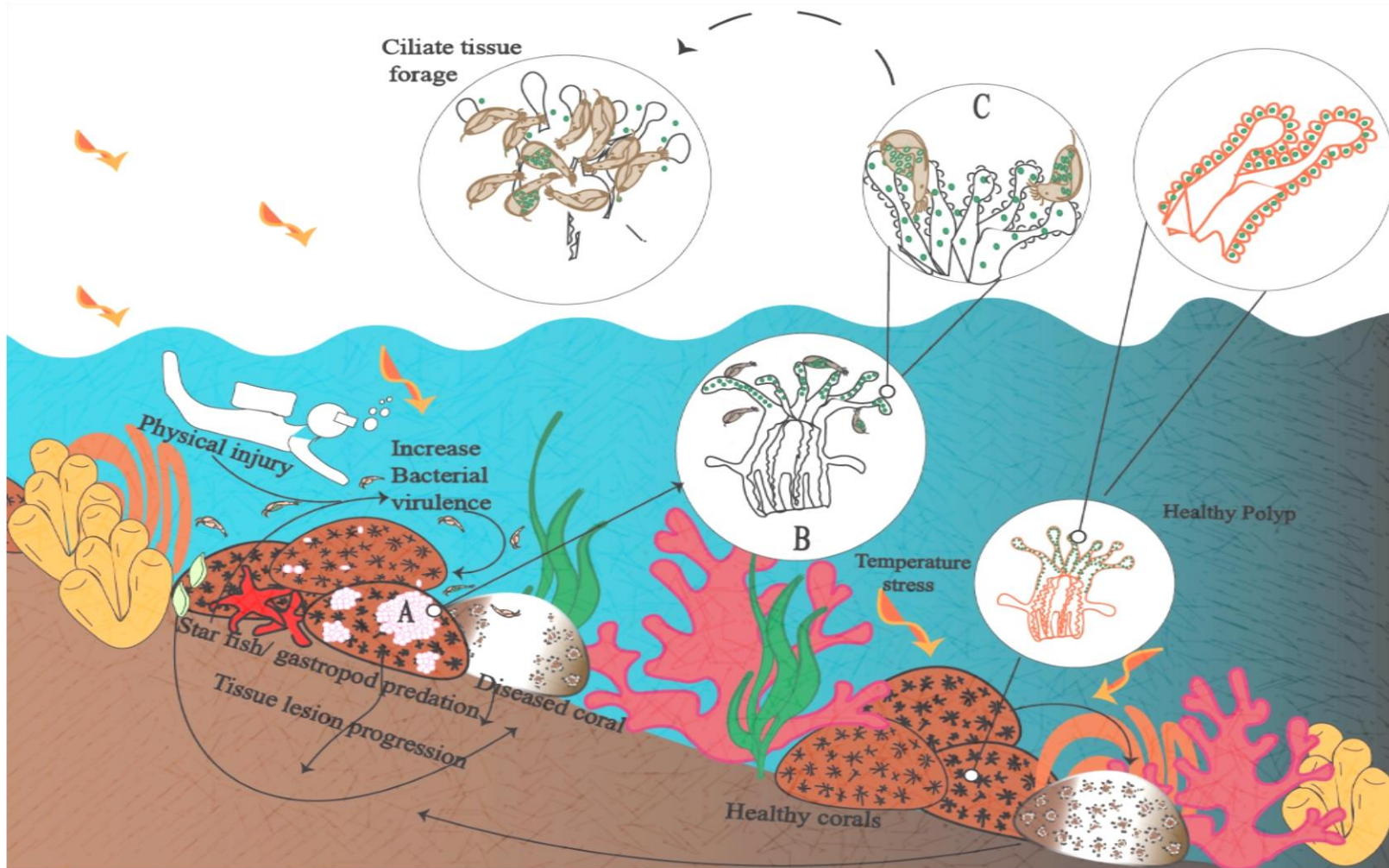


Figure 2

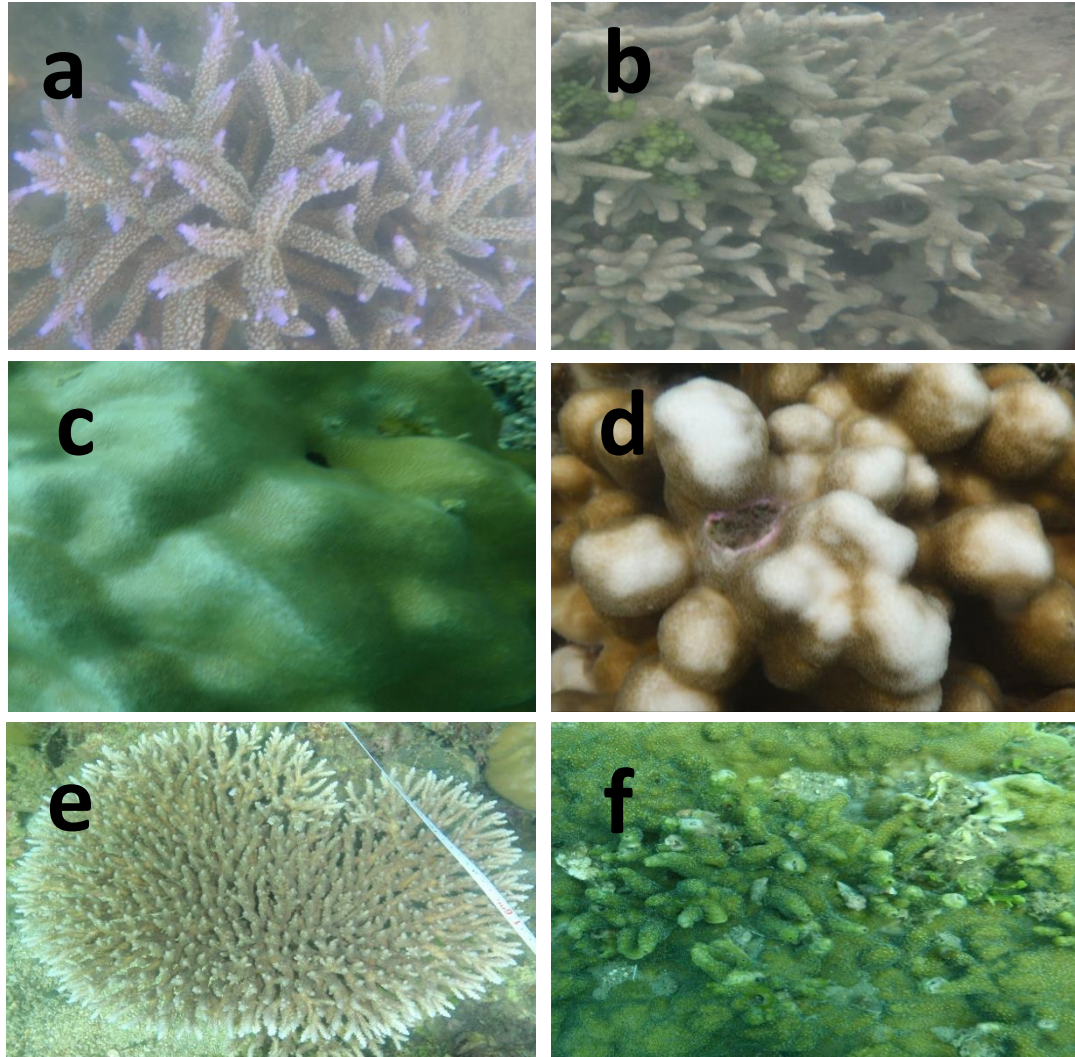


Figure 3

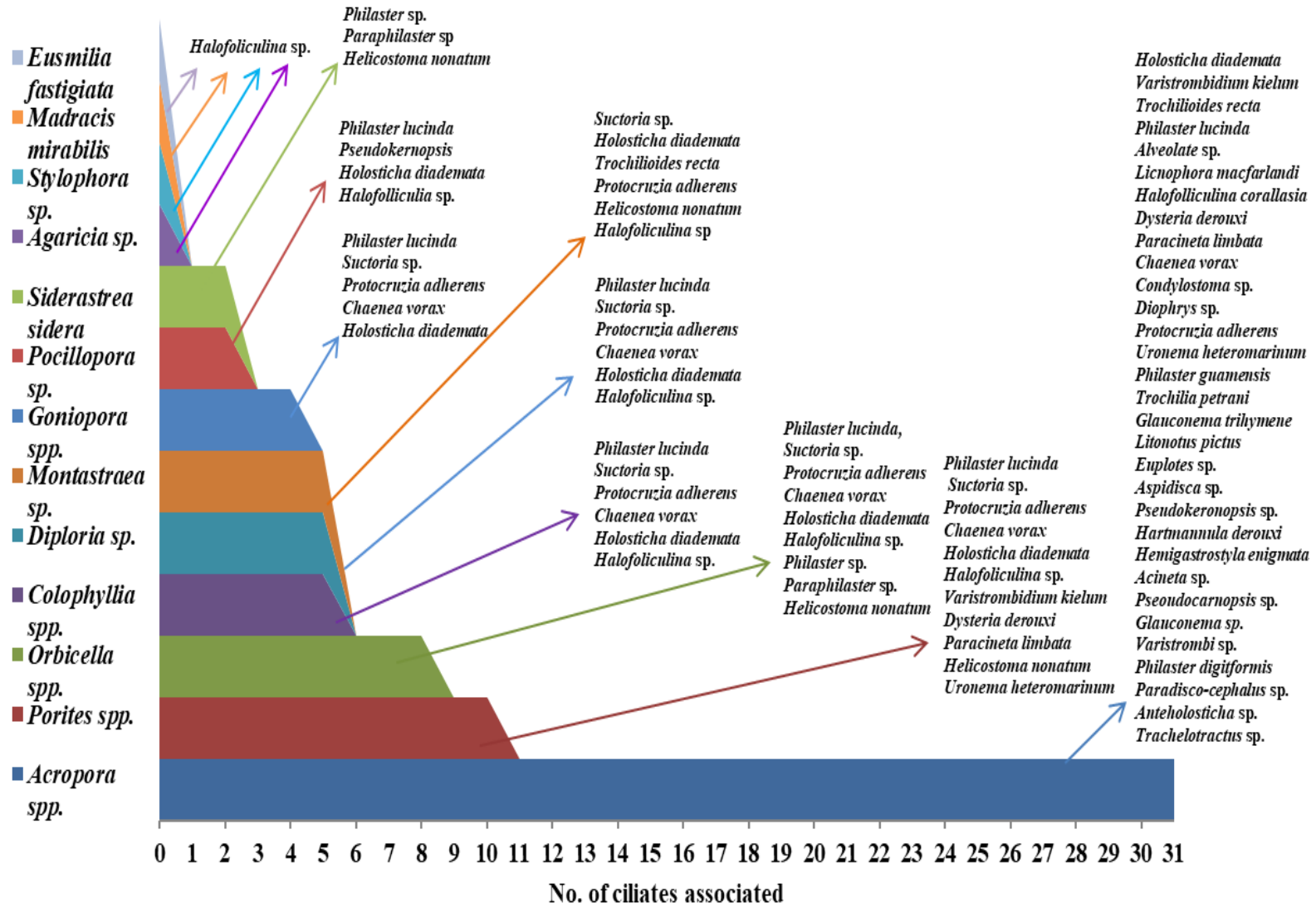


Figure 4

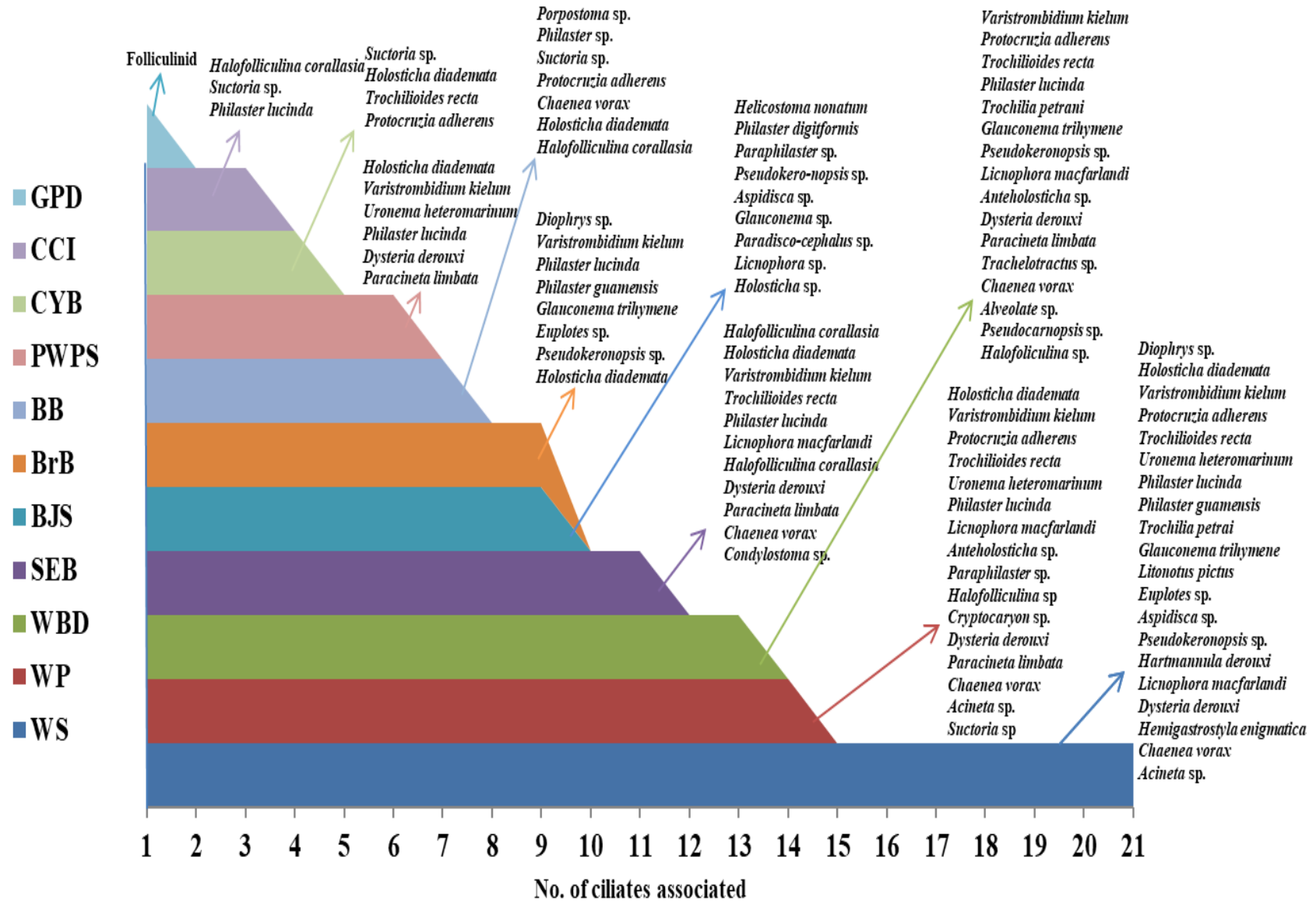


Figure 5

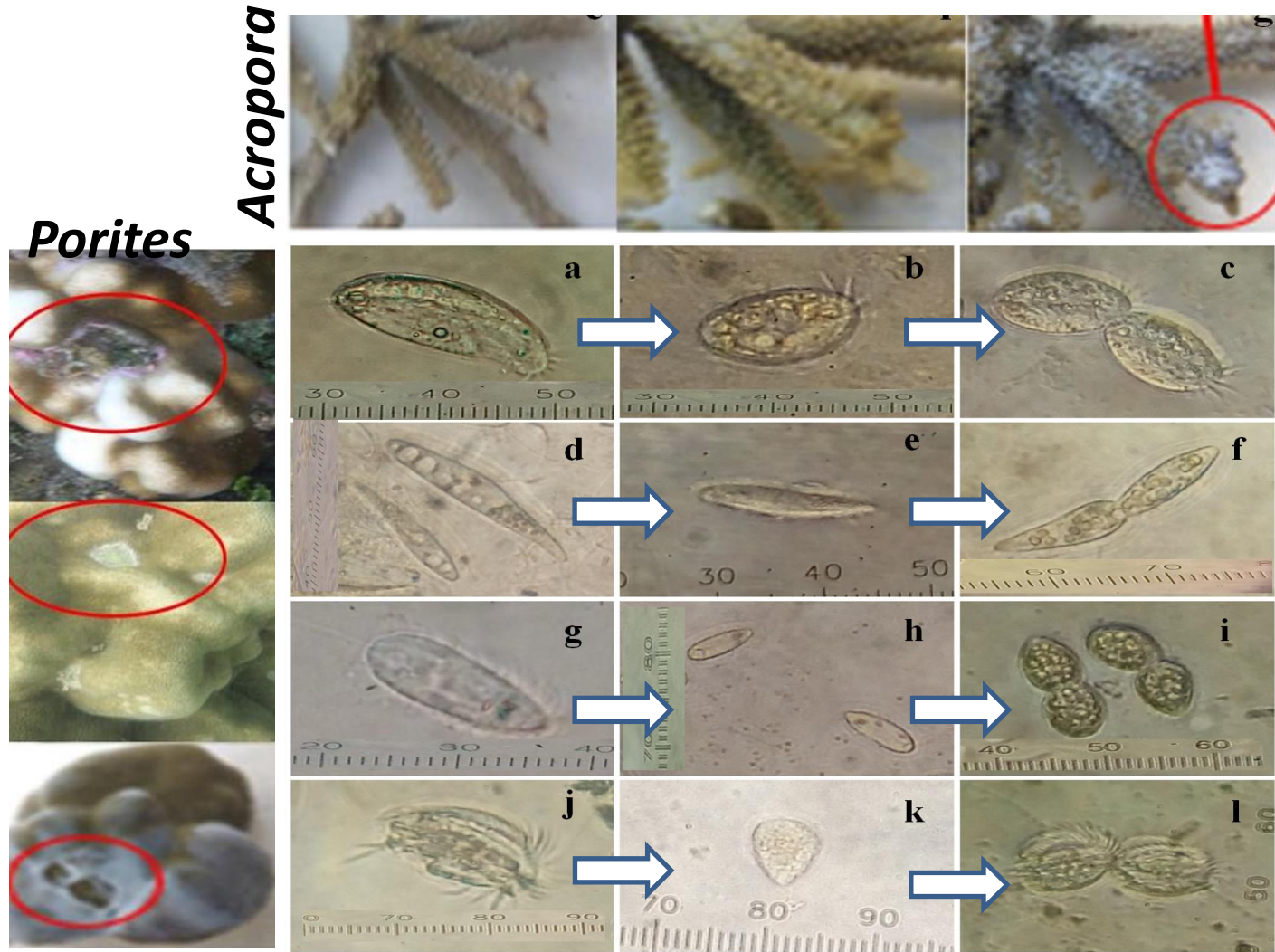


Figure 6

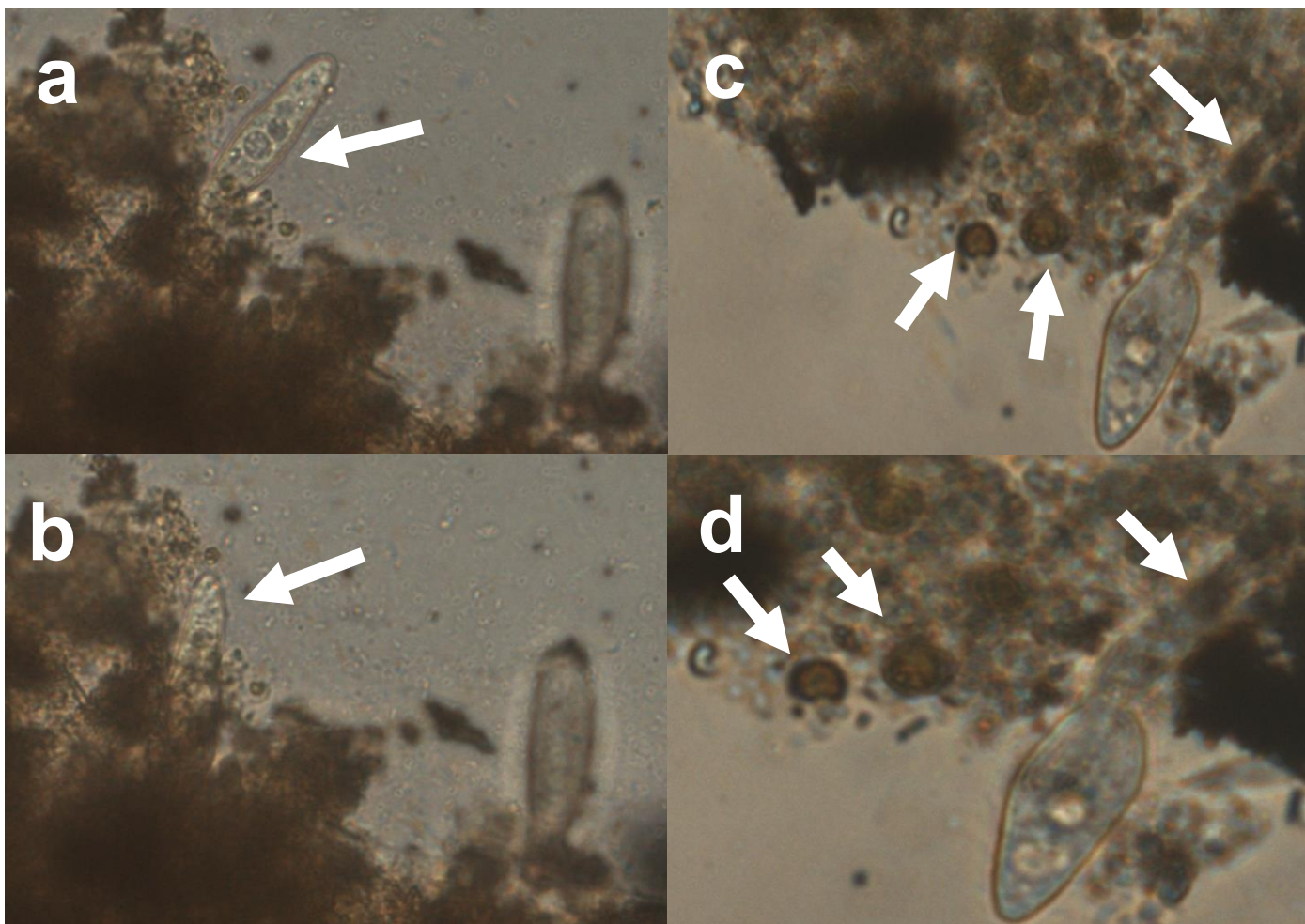


Figure 7

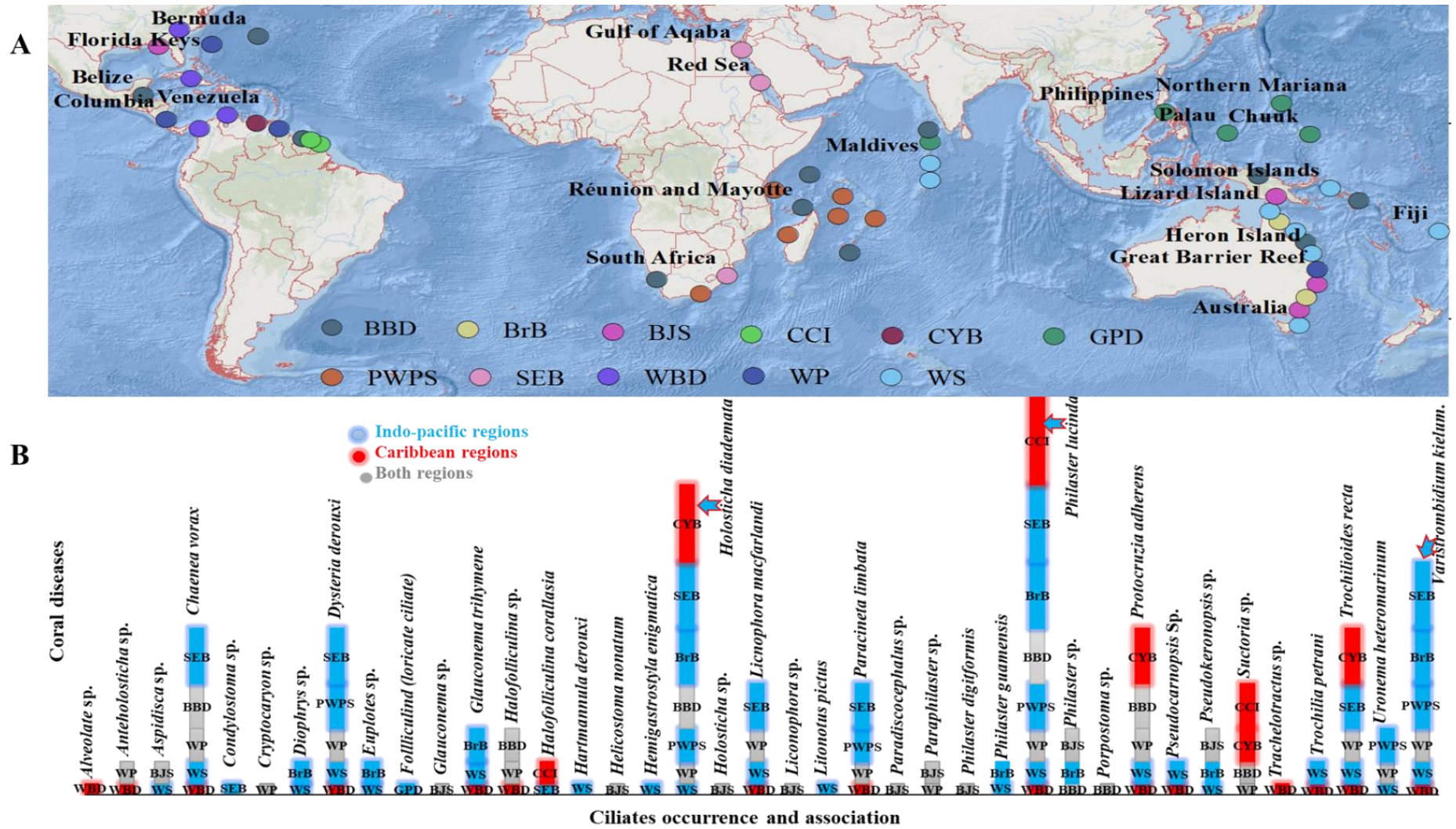


Table 1. Ciliates associated to various disease outbreaks on different coral species.

S No.	Disease	Disease progression	Ciliates associated	Corals	Regions studied	References
1.	White Band	WBDI and II progresses at the colony base and up towards the branch tips leaving bare, coral white skeleton intact. WBDII is also capable of developing at tips of branches and progressing downward (Sutherland et al., 2004).	<i>Alveolate sp. Varistrombidium kielum, Protocruzia adherens, Trochiloides recta, Philaster lucinda, Trochilia petrani, Glauconema trihymene, Pseudocarnopsissp., Licnophora macfarlandi, Anteholosticha sp., Dysteria derouxi, Paracineta limbata, Trachelotractus sp., Chaenea vorax, Halofoliculina sp.</i>	<i>Acropora palmata, A. cervicornis, A. prolifera</i>	A. Caribbean, A. Venezuela and Columbia	Sweet et al. [17], Sweet and Sere [14], Croquer et al. [29].
2	White Syndrome	They are characterised by a progressive tissue loss with a sharp mark.	<i>Diophrys sp., Varistrombidium kielum, Glauconema trihymene, Protocruzia adherens, Trochiloides recta, Philaster lucinda, Trochilia petrani, Litonotus pictus, Aspidisca sp., Euplotes sp., Philaster guamensis, Pseudokeronopsis sp., Holosticha diademata, Hartmannula derouxi, Licnophora macfarlandi, Chaenea vorax, Dysteria derouxi, Hemigastrostyla enigmatica, Uronema heteromarinum and Acineta sp.</i>	<i>Acropora muricata, A. aspera, Pocillopora damicornis</i>	A. Australia, Solomons, Fiji, Maldives, UK Aquaria	Sweet and Sere [14]

			<i>Diophrys</i> sp., <i>Euplotes</i> sp., <i>Aspidisca</i> sp., <i>Pseudocarnopsis</i> sp., <i>Glaucanema</i> sp. <i>Philaster</i> sp., and <i>Varistrombis</i> sp.	<i>Acropora</i> sp.	Heron Islands	Sweet et al. [44]
			<i>Philaster</i> sp., <i>Philaster guamensis</i> . <i>Euplotes</i> sp., <i>Diophrys</i> sp., <i>Varistrombidium</i> sp., <i>Pseudokeronopsis</i> sp., <i>Holosticha</i> sp., <i>Glaucanema trihymene</i> , and <i>Hartmannula derouxi</i>	<i>Acropora muricata</i> and <i>A. aspera</i>	Great Barrier Reef and the Solomon Islands	Sweet and Bythell [7]
			Ciliate (unidentified)	<i>Montipora capitata</i>		Work et al. [52]
			Ciliates- 22 in lesion front and 40 in disease region	<i>Acropora hyacinthus</i>	Lizard Island in the northern sector of the Great Barrier Reef (GBR), Australia,	Pollock et al. [47]
3.	White Plague	Progression rates ranging from few mm to 2 cm each day	<i>Holosticha diademata</i> , <i>Protocruzia adherens</i> , <i>Uronema</i> <i>heteromarinum</i> , <i>Anteholosticha</i> sp., <i>Cryptocaryon</i> sp., <i>Dysteria derouxi</i> , <i>Paracineta limbata</i> , <i>Licnophora</i> <i>macfarlandi</i> , <i>Chaenea vorax</i> , <i>Trochilioides recta</i> ,	<i>Colpophyllia natans</i> and <i>Orbicella annularis</i>	Venezuela, Columbia	Sweet and Sere [14]

		<i>Philaster lucinda</i> , <i>Acineta</i> sp., <i>Varistrombidium kielum</i> , and <i>Suctorina</i> sp.		<i>Orbicella faveolata</i>		
		<i>Philaster</i> sp. <i>Paraphilaster</i> sp. and a ciliate belong to order – Prostomatida.			Florida Keys	Randall et al. [18]
		<i>Halofolliculina</i> sp.		<i>Acropora muricata</i> , <i>Pocillopora damicornis</i> and <i>Porites cylindrica</i> .	Great barrier reef	Page and Willis [3]
4.	<i>Porites</i> White Patch Syndrome	Identified to disperse from spherical to rectangle tissue loss, medium to large (50–300 mm diameter), bounded by enlarged white tissue.	<i>Holosticha diademata</i> , <i>Philaster lucinda</i> , <i>Uronema heteromarinum</i> , <i>Dysteria derouxi</i> , <i>Varistrombidium kielum</i> , and <i>Paracineta limbata</i>	<i>Porites lutea</i>	Moyonette, Reunion and South Africa reefs	Sweet and Sere [14]
5.	Black Band	Characterised by a periphery of coloured dark bands from many milli meters to centimetres, at rates of 1 cm each day, destroying healthy polyps and leave behind only the dead skeleton surface intact	<i>Porpostoma</i> sp., <i>Philaster</i> sp., <i>Philaster lucinda</i> , <i>Suctorina</i> sp., <i>Protocruzia adherens</i> , <i>Chaenea vorax</i> and <i>Holosticha diademata</i>	<i>Montastrea annularis</i> and <i>Diploria strigma</i> <i>Porites lutea</i> , <i>Acropora muricata</i> , <i>Goniopora djiboutiensis</i> , <i>Colpophyllia natans</i> ,	Barrier reefs of Bermuda and Belize South Africa, Venezuela Reunion, Maldives,	Rutzler et al. [24] Sweet and Sere [14]

				<i>Orbicella annularis</i> and <i>Diplora</i> sp.	and Moyonette	
			<i>Halofolliculina corallasia.</i>	<i>Acropora</i> spp.	Great Barrier Reef Australia	Page and Willis [73]
6.	Brown Band	Characterized by their brown coloured zones of different width forming a boundary of healthy coral tissue with the disease exposed skeletons.	class Oligohymenophorea, subclass Scuticociliatia <i>Porpostoma guamensis</i>	<i>Acropora muricata</i> , <i>A.</i> <i>aspera</i> , <i>A. acuminata</i> , <i>A.</i> <i>surculosa</i>	Great Barrier Reef, Guam, Australia	Bourne et al. [16], Lobban et al., [2], Katz et al. [19].
			<i>Diophrys</i> sp., <i>Varistrombidium kielum</i> , <i>Philaster lucinda</i> , <i>Philaster guamensis</i> , <i>Glaucanema trihymene</i> , <i>Euplotes</i> sp., <i>Pseudokeronopsis</i> sp., <i>Holosticha diademata</i> .	<i>Acropora muricata</i>	Australia	Sweet and Sere [14]
			<i>Philaster guamensis</i>	<i>Acropora muricata</i>	Great Barrier Reef and the Solomon Islands	Sweet and Bythell [7]
			Brown Band ciliate	<i>Acropora hyacinthus</i>	South china sea	Qiu et al., [23]

7.	Brown Jelly Syndrome	An aggregation of brown jelly material floating on the coral surface. Tissue loss is fast and the disease is very contagious.	<i>Philaster</i> sp., <i>Paraphilaster</i> sp. <i>Helicostoma nonatum</i>	<i>Orbicella faveolata</i> , <i>Siderastrea siderea</i> <i>Porites astreoides</i> and <i>Montastraea faveolata</i>	Florida Keys Great Barrier Reef Australia	Randall et al. [18] Willis et al. [13]
			<i>Pseudokero-nopsis</i> sp., <i>Aspidisca</i> sp., <i>Glaucanema</i> sp., <i>Paradisico-cephalus</i> sp., <i>Licnophora</i> sp., <i>Philaster digitiformis</i> and <i>Holosticha</i> sp.	<i>Acropora</i> spp.	Aquaria	Sweet et al. [44]
8.	Skeletal Eroding Band	Affected corals having a black band ranging in width from 1 mm to 80 cm.	<i>Halofolliculina corallasia</i>	<i>Stylophora</i> sp, <i>Acropora muricata</i> , <i>Seriatopora</i> sp., <i>Hydnophora</i> sp., <i>Galaxea</i> sp. <i>Pocillopora</i> sp., <i>Mycedium</i> sp., <i>Montipora</i> sp., <i>Echinopora</i> sp., <i>Lobophyllia</i> sp., <i>Goniastrea</i> sp., <i>Millepora</i> sp., <i>Platygyra</i> sp., <i>Fungia</i> sp., <i>Favia</i> sp., <i>Porites</i> sp., <i>Goniopora</i> sp., <i>Favites</i> sp., and <i>Pavona</i> sp.	Gulf of Aqaba, the Red Sea	Antonius and Lipscomb [9], Winkler et al. [10], Page and Willis [3]

			<i>Holosticha diademata, Chaenea vorax, Philaster lucinda, Dysteria derouxi, Trochilioides recta, Licnophora macfarlandi, Halofolliculina corallasia, Paracineta limbata, Varistrombidium kielum and Condylostoma sp.</i>	<i>Acropora muricata</i>	Moyonette, Reunion, South Africa	Sweet and Sere [14]
9.	Caribbean Yellow Band	Characterised by Pale yellow blotch over coral tissue expanding gradually into a band, spreading at rates of 0.5 – 1.0 cm each month.	<i>Suctorina sp., Holosticha diademata, Trochilioides recta and Protocruzia adherens</i>	<i>Montastraea annularis</i>	Venezuela	Sweet and Sere [14]
10.	Caribbean Ciliate Infection	Characterized by grey or black band diffusing from several mm (0.7-0.9 mm/day) to 1-2 cm thick exhibiting salt and pepper appearance by an acute infection	<i>Halofolliculina corallasia, Suctorina sp., Philaster lucinda</i> <i>Halofolliculina</i> <i>Halofolliculina</i>	<i>Acropora cervicornis</i> <i>Acropora cervicornis, Orbicella annularis, O. faveolata</i> <i>Eusmilia fastigiata, Acropora. cervicornis, A. prolifera A. palmata, Montastraea annularis, M. faveolata, M. franksi</i>	Venezuela Los Roques National Park, Venezuelan coast Venezuela (Morrocoy & Los Roques)	Sweet and Sere [14] Verde et al. [32] Croquer et al. [29,30]

				<p><i>Diploria strigosa</i>, <i>D. labyrinthiformis</i>, <i>Colpophyllia natans</i>, <i>Madracis mirabilis</i>, and <i>Agaricia tenuifolia</i></p> <p><i>Porites lobate</i>, <i>P. australiensis</i>, <i>P. lutea</i>, <i>P. rus</i>, <i>P. vaughani</i>, <i>Laptastrea purpurea</i>, <i>L. transversa</i>, <i>Goniastrea retiformis</i>. <i>Acropora palmarae</i>, <i>Astreopora myriophthalma</i>, <i>Favia elliptica</i>, <i>Favia heliantoides</i>, <i>Montipora cf. acanthella</i>, <i>Pavona frondifera</i>, <i>Psammacora contigua</i> and <i>Montastrea</i> sp.</p>
11.	Grey <i>Porites</i> Disease	Characterized by grey biofilms of varying sized (5 cm ² areas to 1 m ² patches) thin mats occurring as multifocal lesions over the large affected coral fragments of <i>Porites</i> .	Folliculinid ciliate.	Northern Marianas, Philippines, Chuuk, Palau, Maldives and Reunion. Sweet et al. [83]

Table 2. Ciliates invasion in coral diseased lesions to forage Symbiodiniaceae.

S No.	Ciliate	Diseases	Coral genera	References
01.	<i>Condylostoma</i> sp.	Skeletal Eroding Band	<i>Acropora muricata</i>	Sweet and Sere [14]
02.	<i>Porpostoma</i> sp.	Black band disease	<i>Diploria</i> sp.	Rutzler et al. [24]
03.	<i>Philaster</i> sp.	Black band disease	<i>Diploria</i> sp.	Rutzler et al. [24]
04.	<i>Porpostoma guamensis</i> , <i>Philaster guamensis</i>	Brown band disease (BrB)	<i>Acropora surculosa</i> , <i>Acropora hyacinthus</i> <i>Acropora muricata</i>	Lobban et al. [2], Sweet and Bythel [7], Sweet and Sere [14], Katz et al. [19]
05.	<i>Philaster lucinda</i>	White band disease	<i>Acropora cervicornis</i>	Sweet et al. [17], Sweet and Sere [14]
06.	<i>Varistrombidium kielum</i> .	White band disease/BrB	<i>Acropora cervicornis</i>	Sweet and Bythel [7], Sweet and Sere [14], Sweet et al. [17]
07.	<i>Trachelotractus</i> sp.	Black band disease	<i>Acropora cervicornis</i>	Sweet and Sere [14]
08.	Brown band ciliates (class Oligohymenophorea, subclass Scuticociliatia)	BrB	<i>Acropora muricata</i> , <i>Acropora hyacinthus</i>	Ulstrup et al. [20] Bourne et al. [16], Qiu et al., [23]
09.	<i>Holosticha diademata</i> ,	BrB	<i>Acropora hyacinthus</i>	Katz et al. [19], Ravindran et al. [15]
10.	<i>Homalogastra setosa</i>	BrB	<i>Acropora hyacinthus</i>	Katz et al. [19]
11.	<i>Euplotes</i> sp.	White syndrome and BrB	<i>Acropora</i> spp., <i>Pocillopora damicornis</i> .	Sweet and Sere [14], Lobban et al. [89]
12.	<i>Cohnilembus verminus</i>	BrB	<i>Acropora hyacinthus</i>	Katz et al. [19]
13.	<i>Halofolliculina</i> sp.		<i>Acropora palmata</i> , <i>A. cervicornis</i> , <i>A. prolifera</i> , <i>Colpophyllia natans</i> , <i>Montastraea annularis</i> , <i>M. faveolata</i> , <i>M. franksi</i> , <i>Madracis mirabilis</i> , <i>M. decactis</i> ,	Cróquer et al. [30]

Diploria labyrinthiformis, *Porites*
furcata, *P. astreoides*, *Agaricia*
agaricites, *A. tenuifolia*, *A. fragilis*, *A.*
lamarcki, *Siderastrea siderea*, *Scolimia*
cubensis, *Leptoseris cuculata*,
Stephanochoenia intersepta and
Millepora alcicornis

14.	<i>Maristentor</i> <i>dinoferus</i>	-	-	Lobban et al. [89], Lobban et al. [88]
15.	<i>Euplotes uncinatus</i>	-	-	Lobban et al. [89]
16.	<i>Paraeuplotes</i> <i>tortugensis</i>	-	-	Lobban et al. [89]