Short title: Coral disease across central Red Sea

Title: A comparative baseline of coral disease across the central Red Sea

Greta Smith Aeby *[1], Amanda Shore[2], Thor Jensen[3], Maren Ziegler[3,4], Thierry Work[5], Christian R.Voolstra[3,6]

[1]Department of Biological and Environmental Sciences, Qatar University, Doha, Qatar

[2]Department of Biology, Farmingdale State College, Farmingdale, NY, USA

[3]Red Sea Research Center, Division of Biological and Environmental Science and Engineering, King Abdullah University of Science and Technology, Thuwal, SaudiArabia

[4] Department of Animal Ecology & Systematics, Justus Liebig University Giessen, Giessen, Germany

[5]US Geological Survey, Wildlife Health Center, Honolulu Field Station, Honolulu, Hawaii, USA

[6]Department of Biology, University of Konstanz, Konstanz, Germany

***Corresponding author**

email: greta@hawaii.edu

Keywords: Red Sea, coral disease, coral bleaching, DHW, white syndrome, black band disease, growth anomalies, temperature stress, salinity stress, land-based pollution

Abstract

The Red Sea is a unique environment for corals with a strong environmental gradient characterized by temperature extremes and high salinities, but minimal terrestrial runoff or riverine input and their associated pollution. Disease surveys were conducted along 22 reefs in the central Red Sea along the Saudi Arabian coast in October 2015, which coincided with a bleaching event. Our objectives were to 1) document types, prevalence, and distribution of coral diseases in a region with minimal terrestrial input, 2) compare regional differences in diseases and bleaching along a latitudinal gradient of environmental conditions, and 3) use histopathology to characterize disease lesions at the cellular level. Coral reefs of the central Red Sea had a widespread but a surprisingly low prevalence of disease (<0.5%), based on the examination of >75,750 colonies. Twenty diseases were recorded affecting 16 coral taxa and included black band disease, white syndromes, endolithic hypermycosis, skeletal eroding band, growth anomalies and focal bleached patches. The three most common diseases were *Acropora* white syndrome (59.1% of the survey sites), *Porites* growth anomalies (40.9%), and *Porites* white syndrome (31.8%). Over half of the coral genera within transects had lesions and corals from the genera *Acropora*, *Millepora* and *Lobophyllia* were the most commonly affected. Cell-associated microbial aggregates were found in four coral genera resembling patterns found in the Indo-Pacific. Differences in disease prevalence, coral cover, amount of heat stress as measured by degree heating weeks (DHW) and extent of bleaching was evident among sites. Disease prevalence was not explained by coral cover or DHW, and a negative relationship between coral bleaching and disease prevalence was found. The northern-most sites off the coast of Yanbu had the highest average DHW values but absence of bleaching and the highest average disease prevalence was recorded. Our study provides a foundation and baseline data for coral disease

prevalence in the Red Sea, which is projected to increase as a consequence of increased frequency and severity of ocean warming.

Introduction

Coral disease is a significant factor impacting coral reefs with localized disease outbreaks occurring worldwide [1,2]. The single most damaging disease outbreak, stony coral tissue loss disease, has been devastating coral reefs throughout the Florida Reef Tract since 2014 [3–5] and has spread to neighboring Caribbean regions [6]. Outbreaks of coral disease have increased through time [7] and have been linked to anthropogenic impacts such as overfishing [8], plastic pollution [9], dredging activities [10], terrestrial runoff [11,12], and increased ocean temperatures [13,14].

Coral reefs face different threats depending on their geographic region. The Red Sea is a unique environment for corals being a partially enclosed body of water with limited exchange with the Indian Ocean, low influxes of freshwater (~30mm/year) and high evaporation rates [15,16]. The majority of coral reefs around the globe live with temperatures usually not exceeding 29°C and salinities around 36 PSU [17]. In the Red Sea, temperature extremes are the norm with temperatures surpassing 32°C in the summer, around 18°C in the winter and with salinities 40 PSU or higher [18,19]. Yet, the Red Sea has extensive and healthy coral reefs with approximately 346 species of reef corals [20,21]. The Red Sea is characterized by natural north to south gradients of temperature, salinity and nutrient availability [22,23]. As example, in the far north, sea surface temperatures (SSTs) average 26℃ (± 1℃) compared to 31.3℃ (± 1.1℃) in the south [22]. Numerous bleaching events have occurred on coral reefs in the Red Sea which also show a latitudinal gradient in coral response. During the recent bleaching in 2015, Osman et al. [24] reported that degree heating weeks (DHW) surpassing the bleaching threshold of 4

(https://coralreefwatch.noaa.gov/product/5km/index_5km_dhw.php) occurred throughout the Red Sea, yet bleaching was restricted to the central and southern Red Sea becoming more severe to the south.

Although coral reefs in the Red Sea face temperature and salinity extremes typically not experienced by corals in other ocean basins, they also receive minimal terrestrial runoff or riverine input and their associated sedimentation, turbidity, and nutrient enrichment. Terrestrial runoff degrades local coral reefs [25] and contributes to increased severity and prevalence of coral diseases [10,26–28]. This creates a unique opportunity to examine coral health in an ecosystem with naturally high temperatures and salinities but minimal terrestrial pollution. We conducted coral disease surveys along the Saudi Arabian coast of the Red Sea in October 2015 which coincided with a bleaching event. Our objectives were to 1) document types, prevalence, and distribution of coral diseases in a region with minimal terrestrial input, 2) compare regional differences in diseases along a latitudinal gradient of environmental conditions and a gradient of bleaching response, 3) use histopathology to characterize disease lesions at the cellular level.

Materials and methods

Disease and bleaching surveys

Coral community structure, disease prevalence and bleaching extent was recorded at 22 sites along the Red Sea coast of Saudi Arabia between October 20 and November 9, 2015 (S1Table; Fig 1). At each site, divers surveyed two replicate 25m belt transects deployed end to end separated by approximately five meters. Corals were identified to the genus level along 25 x 1m belts with the exception of some taxa that are difficult to distinguish in the field. As such, *Favites* and *Dipsastraea* were combined, *Goniopora* and *Alveopora* were combined and

92 *Lobophyllia* and *Symphyllia* were combined. Substrate characteristics (hard coral, soft coral,
93 crustose coralline algae, macroalgae, rubble, sand) and bleaching (color loss) were documented
94 by point-intercept method with the substratum underlying the tape measure recorded at 25 cm
95 intervals and coral cover scored as bleached (pale to total color loss) or healthy. Coral lesions
96 were assessed along wider 25 x 6 m belts. Gross lesions were classified into three lesion types
97 including tissue loss, discoloration and growth anomaly, and nomenclature for lesions was based
98 on the host genus affected and the lesion type (e.g. *Acropora* growth anomaly; [29]). Tissue-loss
99 lesions were further classified based on the lesion size, shape, presence of predators, knowledge
100 of what common predation marks look like and evidence of lesion progression based on degree
101 of algal colonization onto the bare coral skeleton. Transect lengths, widths and numbers were
102 modified as needed when constrained by dive limits.
103
104 **Fig 1.** Sites surveyed for coral disease in three regions along the Saudi Arabian coast within the
105 central Red Sea in Oct-Nov, 2015. Red dots indicate survey sites and blue dots indicate presence
106 of coral reefs.
107

108 Histopathology of coral lesions

109 Paired normal and lesion tissues of coral lesions encountered during surveys were
110 sampled for histopathologic analysis to characterize host response and presence of organisms
111 visible on light microscopy [30]. Fragments were fixed in 20% zinc formaldehyde-seawater
112 immediately after the dive and processed for routine histopathology with hematoxylin and eosin
113 staining of sections. On microscopic exam, host response was categorized as to reversible or
114 non-reversible changes. Reversible cellular changes included atrophy, depletion of

115 zooxanthellae from gastrodermis, wound repair, hyperplasia of basal body wall, and
116 inflammation whereas irreversible changes comprised necrosis and fragmentation. Visible
117 organisms associated with lesions were classified as fungi, bacteria, cyanobacteria, sponges, or
118 algae [30]. Tissue-loss lesions not found associated with obvious micro-organisms were termed
119 'white syndrome' indicating a tissue loss disease of unknown etiology. In addition, all samples
120 collected during surveys for histology were also screened for cell-associated microbial
121 aggregates (CAMA). Certain coral genera in the Indo-Pacific contain CAMAs that are proposed
122 to be facultative secondary symbionts important in coral health [31] and so this was an
123 opportunity to examine whether Red Sea corals also contained CAMAs.
124

125 ## Statistical analyses

126 Underwater time constraints prevented enumeration of all coral colonies within the wider
127 belt transects surveyed for disease. Hence, prevalence of lesions was determined by extrapolating
128 colony counts within the 25 x 1 m transect to the wider 25 x 6 m disease survey area and by using
129 this as the denominator of prevalence calculations (e.g. (number of colonies with lesions/ total
130 number of estimated colonies) * 100). Overall prevalence was the percentage of colonies surveyed
131 that had a particular lesion type with all surveys combined. The frequency of disease occurrence
132 (FOC) reflects the spatial distribution of diseases on reefs and was defined as the number of sites
133 having corals with lesions divided by total number of sites surveyed. The denominator for FOC
134 calculations were limited to sites that had the specific coral taxa exhibiting lesions. All calculations
135 for disease prevalence or FOC were by coral genera (e.g. prevalence of *Porites* growth anomalies
136 was calculated as the total number of affected *Porites* colonies divided by the total number of
137 *Porites* colonies surveyed, multiplied by 100. Percent bleaching was calculated as the number of

points with bleached cover divided by the total number of points (bleached +healthy). Climatology data for each survey site were obtained from NOAA's Coral Reef Watch Product Suite Version 3.1 [32,33] and include the average minimum and maximum sea surface temperatures (SSTs) over the last 25 years (historical SSTs), and the degree heating weeks (DHW) for the 12 week period prior to Oct. 1, 2015.

Data were not normally distributed, even with transformation, so non-parametric analyses were used. A Kruskal-Wallis test and Dunn's post hoc tests were used to examine regional (Al Lith, Thuwal, Yanbu) differences in coral cover, number of coral genera, colony densities, disease prevalence, percent bleaching and degree heating weeks (DHW). Analysis of similarities (ANOSIM) were performed (999 permutations) on weighted (presence and abundance of coral taxa) Bray-Curtis similarity matrices to test for significant differences in coral communities between regions (Al Lith, Thuwal, Yanbu) using PRIMER-E v7 (Primer-E Ltd.). Weighted nMDS plots based on Bray-Curtis similarity matrices were produced to visualize regional differences. Disease susceptibility among coral taxa was examined using a chi-square test for equality of distributions comparing the distribution of the number of diseased versus healthy colonies among the coral genera affected by disease. The relationship between disease prevalence and three potential co-factors: coral cover, percent bleaching and DHW, was examined using a Spearman's rank order correlation. Non-parametric statistics were performed using JMP Pro 13 statistical software (SAS Institute Inc., Buckinghamshire, UK). The map indicating survey locations was created using reefMapMaker [34].

Results

Coral reef characteristics and coral community structure

For the 22 sites surveyed, overall average coral cover was 44.7% (range 3-83%), average soft coral cover was 13.7% (range 0-45%), average crustose coralline algae (CCA) cover was 7.2% (range 0-30%), and average macroalgae cover was <1%. Average colony density was 16.2/m^2 (range 7.3-28.2). Across all sites, 30 coral genera were identified with the three dominant coral taxa being *Porites* (20.6% of the community), *Pocillopora* (14.9%) and *Favites/Dipsastraea* (9.5%) (S2 Table).

Regional differences in coral cover, colony densities and genera richness

There were significant differences among the three regions (Al Lith, Thuwal, Yanbu) in coral cover (Kruskal-Wallis, X^2=11.0, df=2, p=0.004), coral colony densities (Kruskal-Wallis, X^2=7.2, df=2, p=0.03) and number of coral genera (Kruskal-Wallis, X^2=13.1, df=2, p=0.001) (Fig 2). Coral communities were also significantly different among regions (ANOSIM, Global R = 0.13, p = 0.047) (Fig 3). Coral communities in Al Lith differed significantly from Yanbu (ANOSIM, Global R = 0.415, p = 0.013) but Thuwal was not significantly different from Yanbu (ANOSIM, Global R = 0.046, p = 0.301) or Al Lith (ANOSIM, Global R = 0.097, p = 0.169).

Fig 2. Regional differences in coral cover, colony densities and coral genus richness. Letters indicate results of Dunn's multiple group comparison tests. Six sites each were surveyed in Al Lith and Yanbu and ten sites in Thuwal.

183 **Fig 3.** A) Average relative abundance of coral taxa in three regions surveyed for coral disease in
184 2015. Data show the average percent of the coral community represented by each coral genus.
185 B) A non-metric multi-dimensional scaling (nMDS plot) illustrating the differences in coral
186 communities between regions. Six sites each were surveyed in Al Lith and Yanbu and ten sites
187 in Thuwal.
188

189 ## Regional differences in percent bleaching and amount of heat stress

190 There were significant differences among the three regions (Al Lith, Thuwal, Yanbu) in percent
191 bleaching (Kruskal-Wallis, X^2=14.0, df=2, p=0.0009) (Table 1). Sites in the Al Lith region had
192 the highest proportion of bleached coral cover (avg. 33.5%) followed by sites in Thuwal (avg.
193 13.1%) with no bleaching found at sites in Yanbu. There were also significant differences
194 among the three regions in DHW (Kruskal-Wallis, X^2=13.4, df=2, p=0.001)(Table 1). Sites in
195 Yanbu had the highest DHW (avg. 4.9), followed by Al Lith (avg. 4.4) and Thuwal (avg. 3.6).
196 No relationship was found between percent bleaching and DHW (Spearman's rank, Pho=-0.24,
197 p=0.3).
198

199 **Table 1.** Summary of temperature variability and bleaching response in different study sites
200 along the Saudi coast of the Red Sea. Avg. minimum and maximum SSTs over the last 25 years,
201 the Degree Heating Weeks (DHW) for the 12-week period prior to Oct. 1, 2015, and degree of
202 bleaching is reported for each survey site.

		Historical SSTs			
Region	**Site**	**Min (˚C)**	**Max (˚C)**	**DHW (Oct 1, 2015)**	**Bleaching (%)**
Al Lith	Al Lith 3	26.4	31.2	4.0	33.0
Al Lith	Al Lith 2	26.3	31.2	3.8	34.1
Al Lith	Al Lith 1	26.3	31.2	4.3	14.1

Al Lith	Al Lith 6	26.3	31.1	4.8	9.6
Al Lith	Al Lith 4	26.2	31.1	4.6	53.7
Al Lith	Al Lith 5	26.2	31.1	4.7	36.3
Al Lith	**Average**	**26.3**	**31.1**	**4.4**	**33.5**
Thuwal	La Plage	NA	NA	NA	3.4
Thuwal	Abu Madafi	24.7	30.2	2.5	0.0
Thuwal	Al-Mashpah	24.7	30.2	3.1	9.6
Thuwal	Um Alkthal	24.6	30.2	3.7	0.8
Thuwal	Shaab	24.6	30.2	3.8	1.6
Thuwal	Inner Fsar	24.5	30.2	4.1	44.9
Thuwal	Al Fahal	24.5	30.2	3.9	19.5
Thuwal	Tahlah	24.5	30.2	4.1	32.3
Thuwal	Shi'b Nazar	24.5	30.2	2.9	14.1
Thuwal	Qita al Kirsh	24.5	30.2	3.9	5.0
Thuwal	**Average**	**24.6**	**30.2**	**3.6**	**13.1**
Yanbu	Yanbu 3	24.0	29.9	5.3	0.0
Yanbu	Yanbu 2	24.0	29.8	5.2	0.0
Yanbu	Yanbu 1	24.0	29.8	5.1	0.0
Yanbu	Yanbu 5	23.9	29.7	5.1	0.0
Yanbu	Yanbu 4	23.9	29.7	4.5	0.0
Yanbu	Yanbu 6	23.9	29.7	4.5	0.0
Yanbu	**Average**	**24.0**	**29.7**	**4.9**	**0.0**

Disease prevalence, frequency of occurrence and types of diseases

An estimated 75,750 coral colonies were examined for disease and the overall disease prevalence (all sites combined) was 0.17%. A total of 21 diseases in 16 coral taxa were recorded (Table 2). Lesion types included tissue loss diseases of unknown etiology (white syndromes), growth anomalies, distinct focal bleached patches, skeletal eroding band (folliculinid ciliate disease), black band disease (tissue loss due to microbial consortium dominated by filamentous cyanobacteria) and endolithic hypermycosis (purple discoloration due to endolithic fungal infection) (Fig 4). The three most common diseases were *Acropora* white syndrome found at 59.1% of the survey sites, *Porites* growth anomalies found at 40.9% of the sites, and *Porites* white syndrome found at 31.8% of the sites.

215 **Table 2.** Frequency of occurrence (FOC) and average prevalence of coral diseases found during
216 surveys. FOC represents the proportion of sites containing corals with each respective disease.
217 Average prevalence (standard error in parentheses) calculated exclusively from the sites
218 containing each respective disease. Absence of a standard error indicates the disease was found
219 at a single survey site. Prevalence data includes diseased colonies only within transects and so
220 will differ from frequency of disease occurrence data. WS=white syndrome, GA=growth
221 anomaly, SEB=skeletal eroding band (ciliates), BBD=black band disease, Bl patch=focal
222 bleached area, EH=endolithic hypermycosis. The most common diseases are in bold.
223

Coral Genus	Lesion type	FOC (%)	Average Prevalence (%)
Acropora	**WS**	**59.1**	**2.01 (1.19)**
	GA	9.1	0.95 (0.35)
Astreopora	BBD	13.6	0.90 (0.42)
	WS	9.1	1.43 (0.24)
Echinopora	WS	9.1	4.87 (1.54)
Favites/Dipsastraea	BBD	13.6	2.57 (1.92)
	GA	4.5	0.198
Gardinoseris	BBD	13.6	2.31 (0.54)
Goniastrea	WS	4.5	1.74
Goniopora	BBD	4.5	0
Lobophyllia	WS	4.5	1.85
Millepora	WS	9.1	2.6 (2.0)
	GA	4.5	0.758
	EH	4.5	0.463
Montipora	GA	13.6	0.72 (0.14)
	WS	4.5	6.41
	BBD	4.5	2.33
Pavona	BBD	4.5	10.2
	EH	4.5	0.249
Platygyra	BBD	4.5	1.86
Pocillopora	WS	18.2	2.21 (1.56)
	SEB	13.6	0.29 (0.08)
Porites	**GA**	**40.9**	**1.29 (0.62)**
	WS	**31.8**	**0.40 (0.13)**
	Bl patch	13.6	2.11 (1.70)
Psammocora	WS	4.5	0.08

	BBD	4.5	4.8
	EH	4.5	0
Stylophora	**WS**	**27.3**	**7.57 (6.62)**

Fig 4. Examples of different coral diseases encountered during disease surveys along the Saudi coast of the Red Sea. WS=white syndrome, GA=growth anomaly, EH=endolithic hypermycosis, Bl=bleached. A) black band disease, B) *Acropora* WS, C) *Acropora* GA, D) *Astreopora* WS, E) *Echinopora* WS, F) *Favites* GA, G) *Goniastrea* WS, H) *Lobophyllia* WS, I) *Millepora* WS, J) *Millepora* GA, K) *Millepora* EH, L) *Pavona* EH, M) *Montipora* WS, N) *Montipora* GA, O) *Pocillopora* WS, P) *Pocillopora* SEB, Q) *Porites* WS, R) *Porites* GA, S) *Porites* GA, T) *Porites* Bl patch, U) *Psammocora* WS, V) *Psammocora* EH, W) *Stylophora* WS

Histopathology of coral lesions

Paired normal and abnormal tissues collected from 43 colonies representing 15 coral genera were examined. This included samples from 13 *Porites* colonies (30% of the samples), 6 *Stylophora* (14%), 5 *Pocillopora* (12%), 3 each from *Acropora*, *Astreopora*, and *Psammocora* (7% each), 2 *Gardinoseris* (5%), and 1 each from *Dipsastraea*, *Echinopora*, *Favites*, *Goniastrea*, *Goniopora*, *Leptoseris*, *Montipora*, and *Sarcophyton* (2% each). The most common lesion sampled for histology was tissue loss (67%) (Figs 5A-C), followed by growth anomalies (16.5%) (Figs 5D-E) and discoloration (16.5%) (Fig 5F). Of the 29 colonies with tissue loss, the tissue loss lesions could be further subdivided as subacute (n=14) (Fig 5A), acute (n=10) (Fig 5B), chronic (n=3) (Fig 5C), or a combination of the three (n=2). Histology samples originated from Yanbu (n=13), Al Lith (n=11), and Thuwal (n=19).

A total of 87 tissue samples were collected from colonies manifesting tissue loss (N=29), growth anomalies (n=9) and discoloration (n=7) with the remainder being from grossly

246 apparently normal tissues. Of 29 histology sections from colonies with tissue loss, 12 had
247 necrosis either alone or associated with fungi, cyanobacteria, algae, or sponges, 6 sections had
248 atrophy of tissues with depletion of zooxanthellae, 5 had no evident microscopic changes, and 3
249 had varying degrees of inflammation associated with algae, fungi, or cyanobacteria. For 9
250 samples from growth anomalies, 5 had no evident changes, 3 had hyperplasia of basal body wall,
251 and 1 had necrosis with algae. Of 7 sections with discoloration, all but 2 had necrosis with
252 inflammation, fungi, or algae with the 2 remaining with no evident lesions. Of 42 apparently
253 normal fragments, 16 had no evident changes, 12 had necrosis associated with fungi,
254 cyanobacteria, algae or sponges, 8 had atrophy and depletion of zooxanthellae, 4 had
255 inflammation sometimes associated with algae, and 1 had endolithic fungi. Of organisms
256 associated with host response (inflammation, necrosis), fungi dominated (n=12) followed by
257 cyanobacteria (n=6), algae (n=3), and sponges (n=1).
258
259 **Fig 5.** Representative types of lesions sampled for histopathology. A) Acute tissue loss,
260 *Dipsastraea* sp., B) Acute tissue loss, *Goniastrea* sp., C) Chronic tissue loss with discoloration,
261 *Porites* sp., D) Growth anomaly, *Montipora* sp., E) Growth anomaly, *Porites* sp., F)
262 Discoloration, *Porites* sp.
263

264 Cell associated microbial aggregates (CAMA)

265 A total of 87 coral fragments from 15 genera were examined histologically for CAMAs.
266 CAMAs were found in four coral genera, including 5 out of 6 of the *Stylophora* fragments (83%
267 of the samples examined) (Fig 6A), three out of 13 *Porites* fragments (21%)(Fig 6B), two out of

three *Acropora* fragments (67%)(Fig 6C) and two out of five *Pocillopora* fragments (33%)(Fig. 6D).

Fig 6. Cell associated microbial aggregates (arrows) in epidermis (A, C, D) and gastrodermis (B) of *Stylophora* (A), *Porites* (B), *Acropora* (C) and *Pocillopora* (D).

Differences in disease prevalence among coral genera

Out of 30 coral genera found within transects, 16 had lesions indicative of disease. Disease prevalence diffed among coral genera (X^2=90.3, df=16, p<0.005) with *Acropora* having the highest overall disease prevalence (0.54%), followed by *Millepora* (0.44%) and *Lobophyllia* (0.38%) (Fig 7).

Fig 7. Differences in overall disease prevalence among coral taxa. Data show overall prevalence with all surveys combined.

Differences in disease among regions

Average disease prevalence differed significantly among regions (Kruskal-Wallis, X^2=6.6, df=2, p=0.04) (Fig 8) with differences among specific diseases in the frequency of occurrence and average prevalence (Table 3). Of the three most common lesions (BBD, WS, growth anomalies), there were significant regional differences in black band disease (Kruskal-Wallis, X^2=6.3, df=2, p=0.04), and white syndrome (Kruskal-Wallis, X^2=10.2, df=2, p=0.006) but not growth anomalies (Kruskal-Wallis, X^2=4.6, df=2, p=0.1). Average BBD prevalence was highest in Al Lith (0.33% SE$\pm$0.3), although mainly due to one outbreak site, followed by Thuwal (0.009%

291 SE±0.005), and 0% in Yanbu. Average white syndrome prevalence was highest in Yanbu
292 (0.47% SE±0.15) followed by Al Lith (0.06% SE±0.02) and Thuwal (0.07% SE±0.03). Average
293 prevalence of growth anomalies was 0.11% (SE±0.04) in Yanbu, 0.04% (SE±0.02) in Thuwal
294 and 0.003% (SE±0.003) in Al Lith.
295
296 **Fig 8.** Regional differences in disease prevalence at sites surveyed along the Saudi Arabian
297 coast of the Red Sea in October-November 2015. Letters indicate results of Dunn's multiple
298 group comparison tests. Six sites each were surveyed in Al Lith and Yanbu and ten sites in
299 Thuwal.
300
301 **Table 3.** Regional differences in frequency of occurrence (FOC) and average prevalence
302 (±SEM) of different coral diseases found during surveys in the central Red Sea. Prevalence data
303 includes diseased colonies only within transects and so will differ from frequency of disease
304 occurrence data. WS=white syndrome, GA=growth anomaly, SEB=skeletal eroding band
305 (ciliates), BBD=black band disease, Bl patch=focal bleached area, EH=endolithic hypermycosis.
306 Six sites each were surveyed in Al Lith and Yanbu and ten sites in Thuwal.

Coral Genus	Lesion Type	FOC (%)			Average Disease Prevalence (%)		
		Yanbu	Thuwal	Al Lith	Yanbu	Thuwal	Al Lith
Montipora	BBD	0	0	16.7	0	0	2.33
Favites/Dipsastraea	BBD	0	10	33.3	0	0.5	3.6 (2.8)
Psammocora	BBD	0	0	16.7	0	0	4.8
Gardinoseris	BBD	0	10	33	0	3.3	1.81 (0.29)
Astreopora	BBD	0	10	33.3	0	0.32	1.19 (0.52)
Pavona	BBD	0	0	16.7	0	0	10.2
Platygyra	BBD	0	0	16.7	0	0	1.9
Goniopora	BBD	0	0	16.7	0	0	0

Porites	Bl patch	16.7	20	0	5.1	0.42 (0.19)	0
Millepora	EH	0	10	0	0	0.8	0
Psammocora	EH	0	10	0	0	0	0
Pavona	EH	0	10	0	0	0.25	0
Acropora	GA	0	20	0	0	0.95 (0.35)	0
Montipora	GA	0	10	0	0	0.72 (0.14)	0
Porites	GA	66.7	40	0	2.15 (1.13)	0.56 (0.23)	0
Millepora	GA	0	10	0	0	0.8	0
Favites/Dipsastraea	GA	0	10	0	0	0.2	0
Pocillopora	SEB	33.3	10	0	0.33 (0.12)	0.2	0
Acropora	WS	33.3	80	50	8.4 (7.6)	0.98 (0.42)	0.5 (0.23)
Pocillopora	WS	50	0	16.7	0.87 (0.55)	0	8.3
Montipora	WS	33.3	0	0	6.41	0	0
Porites	WS	50	20	16.7	0.67 (0.04)	0.04 (0.04)	0.15 (0.03)
Echinopora	WS	33.3	0	0	4.86 (1.56)	0	0
Millepora	WS	33.3	0	0	2.6 (2.0)	0	0
Goniastrea	WS	16.7	0	0	1.74	0	0
Psammocora	WS	0	0	16.7	0	0	0.08
Lobophyllia	WS	0	0	16.7	0	0	1.85
Astreopora	WS	0	10	16.7	0	0	1.67
Stylophora	WS	16.7	10	0	1.74 (0.74)	0.19 (0.16)	0.08

Relationship between disease prevalence and environmental factors

We examined the relationship between disease prevalence and coral cover, percent bleaching and degree heating weeks (DHW). No significant relationship was found between disease prevalence and coral cover (Spearman's rank, Pho= -0.06, p=0.79) or DHW (Spearman's rank, Pho=0.35, p=0.12). A negative relationship was found between disease prevalence and coral bleaching (Spearman's rank, Pho= -0.51, p=0.02)(Fig 9).

Fig 9. Relationship between coral disease and level of bleaching for 22 sites surveyed in 2015 along the Red Sea coast of Saudi Arabia.

Discussion

Coral reefs are in decline globally and disease has played a significant factor in that decline [1,35,36]. Comparatively little research has been done on coral disease in the Red Sea and our study presents important information on types of diseases present on coral reefs along the Saudi Arabian Red Sea coast, prevalence of diseases, susceptible coral taxa within this region and a description of the histology of different coral lesions. Baseline data is particularly relevant considering the planned mega-building projects such as NEOM (https://www.neom.com) and the Red Sea project (https://visiontoreality.theredsea.sa), which are expected to exert heavy impacts on surrounding coral reef ecosystems. Twenty-two reefs were surveyed in the Red Sea along the Saudi Arabian coast, and robust hard coral (45%) and soft coral (14%) cover, and very low levels of macroalgae cover (<1%) were found. Thirty hard coral genera were found within transects. Coral reefs had widespread but overall low prevalence of disease (<0.5%) with 20 diseases recorded affecting 16 coral taxa and disease lesions found on corals at all sites surveyed. Coral reefs in the Red Sea had diseases typical of many regions including black band disease, white syndromes, endolithic hypermycosis, skeletal eroding band, growth anomalies and distinct focal bleached patches.

Overview of diseases affecting corals in the Red Sea

Black band disease (BBD)

BBD has been reported from coral reefs across the world [37] including the Red Sea [38,39]. BBD typically remains at low background levels [40,41] with seasonal outbreaks occurring [42–44]. At our sites in the Red Sea, there was a similar pattern with a low prevalence of infected

341 corals (0.02-0.12%) found at seven out of 22 sites. Antonius [45] found similar levels of BBD
342 along the Saudi Arabian coast in the 1980s suggesting BBD levels have not changed
343 significantly along these reefs in the past 30 years. We also documented outbreak levels at one
344 site in the Al Lith region, which has been described elsewhere [38]. In fact, most of the BBD
345 cases were found at sites in Al Lith whereas no cases were found in the northern-most region
346 (Yanbu). BBD is sensitive to water temperatures becoming more common in summer months
347 when water temperature and light levels are higher and usually disappears during colder winter
348 months [46–48]. BBD infections also appear following bleaching events[49,50]. Al Lith has
349 higher average SST ranges (26.3˚C to 31.1˚C) as compared to Yanbu with average SSTs ranging
350 from 24.0˚C to 29.7˚C. Al Lith also had the highest level of bleaching among the regions
351 surveyed whereas no bleaching was found in Yanbu. Thus, higher average SSTs combined with
352 a higher bleaching potential may leave reefs at Al Lith particularly vulnerable to BBD infections.

353 **White syndromes (WS)**

354 Tissue loss diseases of unknown etiology (white syndromes) are commonly found on a multitude
355 of species on reefs throughout the world [51] and white syndromes were found in all three
356 regions affecting 10 coral genera. *Acropora* white syndrome was the most widespread disease
357 occurring in all regions at 13 of the 17 reefs surveyed. In addition, *Acropora* white syndrome
358 occurred at outbreak levels at three sites with the highest level at Yanbu 2 (16% prevalence),
359 followed by Abu Madafi (3.6% prevalence) and Al-Mashpah (1.9% prevalence) (both Thuwal
360 region). We defined a site as having a localized disease outbreak if prevalence was higher than
361 the overall prevalence for this region which was <1%. *Acropora* is an exceptionally vulnerable
362 coral genus to tissue loss diseases throughout the world [51] and the Red Sea is no exception.
363

Endolithic hypermycosis (EH)

Endolithic hypermycosis was an uncommon disease with only three cases noted within three different coral genera (*Millepora, Psammocora, Pavona*). In other regions, this lesion is associated with overgrowth of coral tissue by endolithic fungi [52–54] and the samples we examined also had a consistent histological diagnosis of endolithic fungal invasion. This disease has been reported from American Samoa [55], Hawaii [52], Micronesia [54] and New Caledonia [53] and so the present study extends this disease to the reefs of the Red Sea (biogeographic range extension). There are no prior reports of EH in *Millepora* and so the present report also potentially extends the affected taxa to include *Millepora,* a skeleton-forming hydrozoan. However, although the gross lesion on *Millepora* was consistent with endolithic hypermycosis, histology was not done; therefore, confirming presence of the fungus requires future microscopic examination.

Skeletal eroding band (SEB)

Skeletal eroding band (SEB) was found exclusively on pocilloporids. SEB is caused by folliculinid ciliates with tissue loss occurring when motile larval stages migrate into the tissue edges, and secrete pseudochitinous loricae which embed in the coral skeleton [56]. The disease is characterized by a dark band of varied width, adjacent to the healthy tissue, with the denuded skeleton behind the band littered with discarded black loricae [57]. Folliculinid ciliates readily colonize recently exposed coral skeletons [58] so presence of ciliates on coral skeletons does not necessarily indicate ciliate disease. Hence, we only scored a lesion as SEB if we found ciliates within millimeters of live tissue and did not include tissue loss lesions with loricae in dead skeleton further from the lesion edge. However, as we did not follow tissue loss lesions through

time, we cannot rule out that lesions that we scored as SEB, were opportunistic colonization of ciliates following tissue lost to other processes. Unfortunately, verified SEB infections have not been characterized histologically, so the role of folliculinids in contributing to gross lesions remains speculative.

SEB can be quite common on reefs affecting numerous coral taxa. For example, Page and Willis [58] found SEB at 90-100% of their survey reefs affecting at least 82 scleractinian species across the GBR. Winkler et al. [59] surveyed corals in the Gulf of Aqaba, Red Sea and found 28 coral taxa affected by SEB with an overall prevalence of 29% of the total colonies surveyed. In contrast, we only found SEB affecting pocilloporids, which are one of the most commonly affected coral genera [57] but no other coral taxa had SEB lesions. We also only found SEB infections at 3 of 22 survey sites with a prevalence of <0.5% at affected sites. The low SEB prevalence we found could be due to a more conservative approach to field diagnosis of the disease or the environmental conditions on the reefs we surveyed were not conducive to SEB infections. Transmission experiments conducted by Page and Willis [58] showed that ciliates could not colonize intact coral tissue but infections were initiated in coral with injuries. Page et al. [57] suggested that co-infection involving other pathogens and/or stress under specific environmental factors may be required for ciliates to become pathogenic.

Growth anomalies (GA)

Growth anomalies were found in five coral genera (*Porites, Acropora, Montipora, Millepora,* and *Favites/Dipsastraea*). Prior to our study, the only coral genus reported to be affected by GAs in the Red Sea was *Platygyra* sp. [60]. Our study now expands the biogeographic range of GAs in *Porites, Acropora, Montipora,* and *Favites/Dipsastraea* to the Red Sea. GAs in

Millepora have not been reported elsewhere and so our study also expands the host range of GAs. In many regions of the world, *Acropora* and *Porites* have been found disproportionately affected by growth anomalies in the field [61] and consistent with this, *Porites* was the most common coral taxon affected by GAs at our sites. In contrast, acroporids were the 5th most abundant coral taxa at our sites, yet *Acropora* GAs were only found at two of 22 sites. Aeby et al. [62] found that the environmental predictors of GAs differed between *Porites* and *Acropora* and so the environmental conditions in the Red Sea may be conducive to GAs in *Porites* but not *Acropora*. Alternatively, different species within a single genus can differ in their disease susceptibilities [63] so perhaps the specific *Acropora* species found in the Red Sea are less prone to growth anomalies compared to those in other regions.

Focal bleached patches

Discrete, focal bleached patches were found in *Porites* spp. at three sites. *Porites* bleached patches have been reported from the Persian Gulf and Oman Sea [64], New Caledonia [53], and the GBR [65]. This study now extends this disease to the Red Sea (biogeographic range extension). Bleached patches are thought to be due to a viral infection of symbiotic zooxanthellae [66,67] but little else is known about this disease.

Histology shows healthy tissues compromised and presence of CAMAs

Samples for histology were collected following periods of increased sea temperatures and accumulated heat stress in all three regions surveyed and bleaching occurred in two of the regions. Reflecting this stress, we found that 61% of normal fragments (no gross lesion) had some sort of microscopic lesion, mainly necrosis, half of which were associated with a

433 microorganism or atrophy of tissues with depletion of zooxanthellae (bleaching). Indeed, the
434 breakdown of histologic lesions for apparently normal coral fragments was not much different
435 than those associated with tissue loss lesions. Microscopic lesions in normal fragments is not
436 uncommon and has been documented elsewhere. For instance, in a coral disease survey from
437 Micronesia [54] or New Caledonia [68], ca. 26% and 28% , respectively, of normal fragments
438 had microscopic lesions comprising changes similar to those seen here. Fungi were the
439 dominant organisms associated with tissue loss in Saudi Arabia in most species examined. In
440 contrast, when organisms were associated with tissue loss lesions, ciliates dominated for
441 *Acropora* in the Pacific [69] whereas chimeric parasitic corals dominated for *Montipora* in
442 Hawaii [70]. Cell associated microbial aggregates (CAMAs) were associated with
443 Pocilloporidae, Poritidae, and Acroporidae thus extending a pattern similar to that in the Indo-
444 Pacific where CAMA infect these same coral families [31]. Presence of CAMA in *Stylophora*
445 has not been previously described and adds another genus of Pocilloporidae to the list of
446 members of this family infected with bacterial aggregates. This study also extends the presence
447 of CAMAs in corals to the Red Sea (biogeographic range expansion).
448

449 ## Coral taxa differ in disease susceptibility

450 There were differences in disease prevalence among coral genera with *Acropora,*
451 *Favites/Dipsastraea* and *Millepora* having a higher disease prevalence than expected based on
452 their abundance in the field. This is consistent with other regions of the world where disease
453 susceptibility differs across families or genera [40,55,64,71,72]. Our study differed from other
454 regions, in that, over half of the coral genera within transects had signs of disease (16 out of 30
455 coral genera). In contrast, Aeby et al. [64] found seven out of 25 coral genera with disease signs

456 in the Persian Gulf and Williams et al. [72] found five affected coral genera in the Line Islands
457 which has approximately 31 coral genera on its reefs [73].
458

459 **Regional differences in disease and potential environmental co-factors**

460 The survey sites were spread out along a latitudinal gradient spanning from 19˚ to 24˚N. Among
461 the survey sites, coral cover (a measure of host abundance) ranged from 3% to 83%, degree of
462 heat stress, as measured by DHW, ranged from 2.5 to 5.3 and amount of bleaching ranged from
463 0% to 54%. All three of these co-factors would be expected to affect subsequent disease
464 prevalence and as expected, average disease prevalence varied from 0% to 1.9%. However, no
465 significant relationship was found between disease prevalence and coral cover or DHW. And
466 even more surprising, there was a negative relationship, not a positive one as expected, between
467 percent coral bleaching and disease prevalence. This is in sharp contrast to what has been found
468 on coral reefs in other regions. A positive relationship between host density and disease
469 prevalence is considered a hallmark of the infectious process whereby higher host abundance
470 results in greater rates of transmission and localized increases in prevalence [74]. In the Indo-
471 Pacific, an association between coral cover and coral disease prevalence has been found in
472 numerous regions [13,75–77]. Warmer temperatures, heat stress and bleaching have also been
473 linked with higher disease prevalence [2,13,14,40]. For example, in the Persian Gulf, white
474 syndrome outbreaks coincide with annual thermal heating events [78]. In the Caribbean,
475 bleaching extent was linked to increased disease incidence [79] and tissue-loss disease outbreaks
476 frequently follow bleaching events [80–82]. Our study suggests that a very different pattern is
477 emerging for the Red Sea. The northern-most sites along the coast of Yanbu had the highest

478 disease levels despite no bleaching occurring within transects and although heat stress was
479 higher in this region, DHW alone was not a significant factor explaining disease prevalence.
480 Reef corals in the northern Red Sea have extraordinarily high thermal tolerances in
481 relation to the ambient temperatures they usually experience [24] and our study supports this.
482 Significant bleaching is expected when the DHW value reaches 4˚C-weeks
483 (https:/www.coralreefwatch.noaa.gov/product/5km/index_5km_dhw.php) yet our sites in Yanbu
484 had DHW values over 4 but no bleaching was observed. Thermal tolerance in corals has been
485 linked to host factors [83–87], Symbiodiniaceae partners [88–90] or resident microbial
486 communities [91]. In some coral species, thermal tolerance comes at the expense of increased
487 disease susceptibility [92,93] and this has also been suggested as a possible explanation for high
488 disease levels found in corals in the Persian Gulf [64]. Whether there are trade-offs between
489 disease susceptibility and thermal tolerance in corals in the central Red Sea is a hypothesis worth
490 exploring. No work as yet been done on latitudinal gradients of microbial communities or host
491 adaptations on corals in the Red Sea. However, within the Red Sea, the main Symbiodiniaceae
492 genus in *Porites* changed from *Durusdinium* (D1) at warmer nearshore location to *Cladocopium*
493 (C15) at cooler offshore locations [94] suggesting that differences in Symbiodiniaceae could be
494 influencing spatial patterns of disease occurrence in this region.
495

496 **Disease prevalence is low despite environmental challenges**

497 Compared to other ocean basins, the Red Sea experiences extreme temperature variation and
498 regularly exceeds 32˚C in the summers [18,19,95,96] which are conditions not well tolerated by
499 most other corals. Chronic temperature stress can exert significant energetic costs on corals
500 resulting in reduced growth and reproduction [97–99] and an increased prevalence of coral

501 diseases [2,13]. Yet, we found corals existing in this challenging environment to have
502 surprisingly low disease levels (<1%). Moreover, our surveys were conducted in the midst of a
503 bleaching event. This suggests acclimation/adaptation to prevailing environmental conditions
504 [19,100] and notably, the Red Sea is an arid region with minimal terrestrial run-off and almost no
505 riverine input [18]. At our sites, we saw little evidence of sedimentation, water clarity was good
506 and there was little macroalgae on the reef (<1%). Coastal coral reefs in other regions are
507 increasingly exposed to excess nutrients, sediments, and pollutants discharged from land which
508 are known to degrade local reefs [25] including increasing coral disease prevalence. Haapkyla et
509 al. [11] found a 10-fold greater mean abundance of disease on reefs during the rainy summer
510 months and concluded that rainfall and associated runoff were facilitating disease outbreaks.
511 Laboratory studies showed that the rate of tissue loss from BBD increased with nutrient
512 enrichment [26] and increased BBD prevalence in the field is associated with sewage effluent
513 [101]. An experimental *in situ* nutrient enrichment of reefs was conducted in the Caribbean and
514 corals exposed to chronic nutrient stress suffered a 3.5-fold increase in bleaching frequency and
515 a two-fold increase in prevalence and severity of disease, compared to corals in control plots
516 [27]. Terrestrial run-off also promotes the growth of macroalgae on coral reefs [25] which are
517 major competitors with corals [102,103]. Additionally, macroalgae exude dissolved organic
518 carbon which can disrupt the function of the coral holobiont and promote potential bacterial
519 pathogens [104,105]. Although the coral reefs in the Red Sea have to contend with high
520 temperatures and salinity, these appears to be countered by the lack of terrestrial run-off and all
521 its associated problems. As a comparison, the Persian Gulf is also an arid region but has riverine
522 input, and massive coastal habitat modification by dredging and converting shallow, productive
523 marine areas into land for homes, recreation, and industrial activities [106]. Resuspension of

sediments is an ongoing stress for coral reefs in this region as well [107]. Under these conditions, higher coral disease levels were found and attributed to environmental stress [108]. Compared to our study, reefs along the northeastern Arabian Peninsula show a 6-fold higher disease prevalence with a high number of localized disease outbreaks [64]. Reefs surrounding Kish Island, off the coast of Iran, showed a 20-fold increase in disease [109]. Disease is a serious problem in other world regions [3,12,40] and our study suggests that a reduction in human impacts and improvement in water quality may be effective management strategies giving corals increased capacity to withstand the warming oceans predicted with global climate change.

Acknowledgments

Any use of trade, firm, or product names is for descriptive purposes only and does not imply endorsement by the US Government. We would like to thank the invaluable assistance of CMOR staff who helped with diving operations and boating.

References

1. Harvell CD, Jordan-Dahlgren E, Merkel S, Rosenberg E, Raymundo L, Smith G, et al. Coral disease, environmental drivers and the balance between coral and microbial associates. Oceanography. 2007;20:172–95.

2. Heron SF, Willis BL, Skirving WJ, Eakin CM, Page CA, Miller IR. Summer Hot Snaps and Winter Conditions: Modelling White Syndrome Outbreaks on Great Barrier Reef Corals. PLoS ONE. 2010;5:e12210.

3. Precht WF, Gintert BE, Robbart ML, Fura R, van Woesik R. Unprecedented Disease-Related Coral Mortality in Southeastern Florida. Scientific Reports. 2016;6:31374.

4. Walton CJ, Hayes NK, Gilliam DS. Impacts of a Regional, Multi-Year, Multi-Species Coral Disease Outbreak in Southeast Florida. Frontiers in Marine Science. 2018;5:323.

549 5. Muller EM, Sartor C, Alcaraz N, van Woesik R. Spatial Epidemiology of the Stony-Coral-
550 Tissue-Loss Disease in Florida. Frontiers in Marine Science. 2020;7:163.

551 6. Alvarez-Filip L, Estrada-Saldívar N, Pérez-Cervantes E, Molina-Hernández A, González-
552 Barrios FJ. A rapid spread of the stony coral tissue loss disease outbreak in the Mexican
553 Caribbean. PeerJ. 2019;7:e8069.

554 7. Ward JR, Lafferty KD. The Elusive Baseline of Marine Disease: Are Diseases in Ocean
555 Ecosystems Increasing? PLoS Biology. 2004;2:e120.

556 8. Raymundo LJ, Halford AR, Maypa AP, Kerr AM. Functionally diverse reef-fish communities
557 ameliorate coral disease. PNAS. 2009;106:17067–70.

558 9. Lamb JB, Willis BL, Fiorenza EA, Couch CS, Howard R, Rader DN, et al. Plastic waste
559 associated with disease on coral reefs. Science. 2018;359:460–2.

560 10. Pollock FJ, Lamb JB, Field SN, Heron SF, Schaffelke B, Shedrawi G, et al. Sediment and
561 Turbidity Associated with Offshore Dredging Increase Coral Disease Prevalence on Nearby
562 Reefs. PLoS ONE. 2014;9:e102498.

563 11. Haapkylä J, Unsworth RKF, Flavell M, Bourne DG, Schaffelke B, Willis BL. Seasonal
564 Rainfall and Runoff Promote Coral Disease on an Inshore Reef. PLoS ONE. 2011;6:e16893.

565 12. Aeby GS, Callahan S, Cox E, Runyon C, Smith A, Stanton F, et al. Emerging coral diseases
566 in Kāneʻohe Bay, Oʻahu, Hawaiʻi (USA): two major disease outbreaks of acute *Montipora* white
567 syndrome. Dis Aquat Org. 2016;119:189–98.

568 13. Bruno JF, Selig ER, Casey KS, Page CA, Willis BL, Harvell CD, et al. Thermal Stress and
569 Coral Cover as Drivers of Coral Disease Outbreaks. PLoS Biology. 2007;5:e124.

570 14. Maynard J, van Hooidonk R, Eakin CM, Puotinen M, Garren M, Williams G, et al.
571 Projections of climate conditions that increase coral disease susceptibility and pathogen
572 abundance and virulence. Nature Clim Change. 2015;5:688–94.

573 15. Sheppard C, Price A, Roberts C. Marine ecology of the Arabian region: patterns and
574 processes in extreme tropical environments. London: Academic Press; 1992.

575 16. Berman T, Paldor N, Brenner S. Annual SST cycle in the Eastern Mediterranean, Red Sea
576 and Gulf of Elat: annual cycle of SST. Geophys Res Lett. 2003;30.

577 17. Kleypas JA, Mcmanus JW, Meñez LAB. Environmental Limits to Coral Reef Development:
578 Where Do We Draw the Line? Am Zool. 1999;39:146–59.

579 18. Roik A, Röthig T, Roder C, Ziegler M, Kremb SG, Voolstra CR. Year-Long Monitoring of
580 Physico-Chemical and Biological Variables Provide a Comparative Baseline of Coral Reef
581 Functioning in the Central Red Sea. PLoS ONE. 2016;11:e0163939.

582 19. Voolstra CR, Buitrago-López C, Perna G, Cárdenas A, Hume BCC, Rädecker N, et al.
583 Standardized short-term acute heat stress assays resolve historical differences in coral
584 thermotolerance across microhabitat reef sites. Glob Change Biol. 2020;26:4328–43.

585 20. DiBattista JD, Roberts MB, Bouwmeester J, Bowen BW, Coker DJ, Lozano-Cortés DF, et al.
586 A review of contemporary patterns of endemism for shallow water reef fauna in the Red Sea. J
587 Biogeogr. 2016;43:423–39.

588 21. Berumen ML, Voolstra CR, Daffonchio D, Agusti S, Aranda M, Irigoien X, et al. The Red
589 Sea: Environmental Gradients Shape a Natural Laboratory in a Nascent Ocean. Coral Reefs of
590 the Red Sea Coral Reefs of the World. Springer, Cham; 2019. Available from:
591 https://doi.org/10.1007/978-3-030-05802-9_1

592 22. Sofianos SS. An Oceanic General Circulation Model (OGCM) investigation of the Red Sea
593 circulation: 2. Three-dimensional circulation in the Red Sea. J Geophys Res. 2003;108:3066.

594 23. Sawall Y, Al-Sofyani A, Hohn S, Banguera-Hinestroza E, Voolstra CR, Wahl M. Extensive
595 phenotypic plasticity of a Red Sea coral over a strong latitudinal temperature gradient suggests
596 limited acclimatization potential to warming. Scientific Reports. 2015;9.

597 24. Osman EO, Smith DJ, Ziegler M, Kürten B, Conrad C, El-Haddad KM, et al. Thermal
598 refugia against coral bleaching throughout the northern Red Sea. Glob Change Biol.
599 2018;24:e474–84.

600 25. Fabricius KE. Effects of terrestrial runoff on the ecology of corals and coral reefs: review and
601 synthesis. Marine Pollution Bulletin. 2005;50:125–46.

602 26. Voss JD, Richardson LL. Nutrient enrichment enhances black band disease progression in
603 corals. Coral Reefs. 2006;25:569–76.

604 27. Vega Thurber RL, Burkepile DE, Fuchs C, Shantz AA, McMinds R, Zaneveld JR. Chronic
605 nutrient enrichment increases prevalence and severity of coral disease and bleaching. Glob
606 Change Biol. 2014;20:544–54.

607 28. Shore-Maggio A, Aeby GS, Callahan SM. Influence of salinity and sedimentation on *Vibrio*
608 infection of the Hawaiian coral *Montipora capitata*. Dis Aquat Org. 2018;128:63–71.

609 29. Work T, Aeby G. Systematically describing gross lesions in corals. Dis Aquat Org.
610 2006;70:155–60.

611 30. Work TM, Russell R, Aeby GS. Tissue loss (white syndrome) in the coral *Montipora*
612 *capitata* is a dynamic disease with multiple host responses and potential causes. Proceedings of
613 the Royal Society B: Biological Sciences. 2012;279:4334–41.

614 31. Work TM, Aeby GS. Microbial aggregates within tissues infect a diversity of corals
615 throughout the Indo-Pacific. Marine Ecology Progressive Series. 2014;500:1–9.

616 32. Skirving W, Marsh B, De La Cour J, Liu G, Harris A, Maturi E, et al. CoralTemp and the
617 Coral Reef Watch Coral Bleaching Heat Stress Product Suite Version 3.1. Remote Sensing.
618 2020;12:3856.

619 33. Liu G, Skirving WJ, Geiger EF, Heron SF, Tirak KV, Strong AE, et al. NOAA Coral Reef
620 Watch's 5km Satellite Coral Bleaching Heat Stress Monitoring Product Suite Version 3 and
621 Four-Month Outlook Version 4. 2017;32:7.

622 34. Hume BCC, Voolstra CR. reefMapMaker - convenient creation of user-defined regional
623 maps with coral reef locations (Version v0.1.4). Zenodo. 2021; Available from:
624 http://doi.org/10.5281/zenodo.4415326

625 35. Harvell CD, Kim K, Burkholder JM, Colwell RR, Epstein PR, Grimes DJ, et al. Emerging
626 Marine Diseases: Climate Links and Anthropogenic Factors. Science. 1999;285:1505–10.

627 36. Maynard JA, Anthony KRN, Harvell CD, Burgman MA, Beeden R, Sweatman H, et al.
628 Predicting outbreaks of a climate-driven coral disease in the Great Barrier Reef. Coral Reefs.
629 2010;30:485–95.

630 37. Raymundo L, Weil E. Indo-Pacific coloured-band diseases of corals. Diseases of corals.
631 Hoboken, NJ: Wiley-Blackwell; 2015.

632 38. Hadaidi G, Ziegler M, Shore-Maggio A, Jensen T, Aeby G, Voolstra CR. Ecological and
633 molecular characterization of a coral black band disease outbreak in the Red Sea during a
634 bleaching event. PeerJ. 2018;

635 39. Neave MJ, Apprill A, Aeby G, Miyake S, Voolstra CR. Microbial Communities of Red Sea
636 Coral Reefs. In: Voolstra CR, Berumen ML, editors. Coral Reefs of the Red Sea. Cham: Springer
637 International Publishing; 2019 p. 53–68. Available from: http://link.springer.com/10.1007/978-
638 3-030-05802-9_4

639 40. Willis BL, Page CA, Dinsdale EA. Coral Disease on the Great Barrier Reef. In: Rosenberg E,
640 Loya Y, editors. Coral Health and Disease. Berlin, Heidelberg: Springer Berlin Heidelberg; 2004
641 p. 69–104. Available from: http://link.springer.com/10.1007/978-3-662-06414-6_3

642 41. Page C, Willis B. Distribution, host range and large-scale spatial variability in black band
643 disease prevalence on the Great Barrier Reef, Australia. Dis Aquat Org. 2006;69:41–51.

644 42. Green EP, Bruckner AW. The significance of coral disease epizootiology for coral reef
645 conservation. Biological Conservation. 2000;96:347–61.

646 43. AL-MOGHRABI S. Unusual black band disease (BBD) outbreak in the northern tip of the
647 Gulf of Aqaba (Jordan). Coral Reefs. 2001;19:330–1.

648 44. Aeby GS, Work TM, Runyon CM, Shore-Maggio A, Ushijima B, Videau P, et al. First
649 Record of Black Band Disease in the Hawaiian Archipelago: Response, Outbreak Status,
650 Virulence, and a Method of Treatment. PLOS ONE. 2015;10:e0120853.

651 45. Antonius A. Distribution and dynamics of coral diseases in the Eastern Red Sea. Proceedings
652 of the 6th International Coral Reef Symposium. 1988;2:293–8.

653 46. Kuta KG, Richardson LL. Abundance and distribution of black band disease on coral reefs in
654 the northern Florida Keys. Coral Reefs. 1996;15:219–23.

655 47. Boyett HV, Bourne DG, Willis BL. Elevated temperature and light enhance progression and
656 spread of black band disease on staghorn corals of the Great Barrier Reef. Mar Biol.
657 2007;151:1711–20.

658 48. Sato Y, Bourne DG, Willis BL. Dynamics of seasonal outbreaks of black band disease in an
659 assemblage of *Montipora* species at Pelorus Island (Great Barrier Reef, Australia). Proceedings
660 of the Royal Society B: Biological Sciences. 2009;276:2795–803.

661 49. Lewis CL. Temporal dynamics of black band disease affecting pillar coral (*Dendrogyra*
662 *cylindrus)* following two consecutive hyperthermal events on the Florida Reef Tract. Coral
663 Reefs. 2017;6.

664 50. Kubomura T, Yamashiro H, Reimer J. Appearance of an anomalous black band disease at
665 upper mesophotic depths after coral bleaching. Dis Aquat Org. 2018;131:245–50.

666 51. Bourne DG, Ainsworth TD, Willis BL. White syndromes of Indo-Pacific corals. Diseases of
667 Coral. Wiley-Blackwell; 2016. p. 300–15.

668 52. Work TM, Aeby GS, Stanton FG, Fenner D. Overgrowth of fungi (endolithic hypermycosis)
669 associated with multifocal to diffuse distinct amorphous dark discoloration of corals in the Indo-
670 Pacific. Coral Reefs. 2008;27:663–663.

671 53. Aeby G, Tribollet A, Lasne G, Work T. Assessing threats from coral and crustose coralline
672 algae disease on the reefs of New Caledonia. Marine and Freshwater Research. 2015;67:455–65.

673 54. Work TM, Aeby GS, Hughen KA. Gross and Microscopic Lesions in Corals from
674 Micronesia. Vet Pathol. 2016;53:153–62.

675 55. Aeby G, Work T, Fenner D, Didonato E. Coral and crustose coralline algae disease on the
676 reefs of American Samoa. Proceedings of the 11th International Coral Reef Symposium.
677 2008;7:197–201.

678 56. Antonius AA, Lipscomb D. First protozoan coral-killer identified in the Indo-Pacific. Atoll
679 Research Bulletin. 2001;481:1–21.

680 57. Page CA, Cróquer A, Bastidas C, Rodríguez S, Neale SJ, Weil E, et al. *Halofolliculina*
681 Ciliate Infections on Corals (Skeletal Eroding Disease). In: Woodley CM, Downs CA, Bruckner
682 AW, Porter JW, Galloway SB, editors. Diseases of Coral. Hoboken, NJ: John Wiley & Sons, Inc;
683 2015. p. 361–75. Available from: http://doi.wiley.com/10.1002/9781118828502.ch26

684 58. Page CA, Willis BL. Epidemiology of skeletal eroding band on the Great Barrier Reef and
685 the role of injury in the initiation of this widespread coral disease. Coral Reefs. 2008;27:257–72.

686 59. Winkler R, Antonius A, Abigail Renegar D. The Skeleton Eroding Band Disease on Coral
687 Reefs of Aqaba, Red Sea. Marine Ecology. 2004;25:129–44.

688 60. Loya Y. The coral reefs of Eilat—past, present and future: three decades of coral community
689 structure studies. Coral Health and disease. Springer2004; p. 1–34.

690 61. Work TM, Kaczmarsky L, Peters EC. Skeletal Growth Anomalies in Corals. Diseases of
691 Coral. Wiley-Blackwell; 2016. p. 291–9.

692 62. Aeby GS, Williams GJ, Franklin EC, Haapkyla J, Harvell CD, Neale S, et al. Growth
693 Anomalies on the Coral Genera *Acropora* and *Porites* Are Strongly Associated with Host
694 Density and Human Population Size across the Indo-Pacific. PLoS ONE. 2011;6:e16887.

695 63. Aeby GS, Bourne DG, Wilson B, Work TM. Coral Diversity and the Severity of Disease
696 Outbreaks: A Cross-Regional Comparison of *Acropora* White Syndrome in a Species-Rich
697 Region (American Samoa) with a Species-Poor Region (Northwestern Hawaiian Islands).
698 Journal of Marine Biology. 2011;2011:1–8.

699 64. Aeby GS, Howells E, Work T, Abrego D, Williams GJ, Wedding LM, et al. Localized
700 outbreaks of coral disease on Arabian reefs are linked to extreme temperatures and
701 environmental stressors. Coral Reefs. 2020;39:829–46.

702 65. Roff G, Ulstrup KE, Fine M, Ralph PJ, Hoegh-Guldberg O. S Spatial heterogeneity of
703 photosynthetic activity within diseased corals from the Great Barrier Reef. Journal of Phycology.
704 2008;44:526–38.

705 66. Lawrence SA, Davy JE, Wilson WH, Hoegh-Guldberg O, Davy SK. *Porites* white patch
706 syndrome: associated viruses and disease physiology. Coral Reefs. 2015;34:249–57.

707 67. Kenkel CD, Mocellin VJL, Bay LK. Global gene expression patterns in *Porites* white patch
708 syndrome: Disentangling symbiont loss from the thermal stress response in reef-building coral.
709 Mol Ecol. 2020;29:3907–20.

710 68. Work TM, Aeby GS, Lasne G, Tribollet A. Gross and microscopic pathology of hard and
711 soft corals in New Caledonia. Journal of Invertebrate Pathology. 2014;120:50–8.

712 69. Work TM. Pathology of tissue loss (white syndrome) in *Acropora* sp. corals from the Central
713 Pacific. Journal of Invertebrate Pathology. 2011;5.

714 70. Work TM, Forsman ZH, Szabó Z, Lewis TD, Aeby GS, Toonen RJ. Inter-Specific Coral
715 Chimerism: Genetically Distinct Multicellular Structures Associated with Tissue Loss in
716 *Montipora capitata*. PLoS ONE. 2011;6:e22869.

717 71. Aeby GS, Williams GJ, Franklin EC, Kenyon J, Cox EF, Coles S, et al. Patterns of Coral
718 Disease across the Hawaiian Archipelago: Relating Disease to Environment. PLoS ONE.
719 2011;6:e20370.

720 72. Williams G, Knapp I, Aeby G, Davy S. Spatial and temporal patterns of scleractinian coral,
721 soft coral, and zoanthid disease on a remote, near-pristine coral reef (Palmyra Atoll, central
722 Pacific). Dis Aquat Org. 2011;94:89–100.

723 73. Williams GJ, Maragos JE, Davy SK. Characterization of the coral communities at Palmyra
724 Atoll in the remote central Pacific Ocean. Atoll Research Bulletin. 2008;557:1–32.

725 74. Lafferty KD, Gerber LR. Good Medicine for Conservation Biology: the Intersection of
726 Epidemiology and Conservation Theory. Conservation Biology. 2002;16:12.

727 75. Haapkylä J, Unsworth R, Seymour A, Melbourne-Thomas J, Flavell M, Willis B, et al.
728 Spatio-temporal coral disease dynamics in the Wakatobi Marine National Park, South-East
729 Sulawesi, Indonesia. Dis Aquat Org. 2009;87:105–15.

730 76. Myers R, Raymundo L. Coral disease in Micronesian reefs: a link between disease
731 prevalence and host abundance. Dis Aquat Org. 2009;87:97–104.

732 77. Aeby GS, Ross M, Williams GJ, Lewis TD, Work TM. Disease dynamics of *Montipora*
733 white syndrome within Kaneohe Bay, Oahu, Hawaii: distribution, seasonality, virulence, and
734 transmissibility. Dis Aquat Org. 2010;91:1–8.

735 78. Howells EJ, Vaughan GO, Work TM, Burt JA, Abrego D. Annual outbreaks of coral disease
736 coincide with extreme seasonal warming. Coral Reefs. 2020;39:771–81.

737 79. Brandt ME, McManus JW. Disease incidence is related to bleaching extent in reef-building
738 corals. Ecology. 2009:90:2859-2867.

739 80. Miller J, Muller E, Rogers C, Waara R, Atkinson A, Whelan KRT, et al. Coral disease
740 following massive bleaching in 2005 causes 60% decline in coral cover on reefs in the US Virgin
741 Islands. Coral Reefs. 2009;28:925–37.

742 81. Muller EM, Rogers CS, Spitzack AS, van Woesik R. Bleaching increases likelihood of
743 disease on *Acropora palmata* (Lamarck) in Hawksnest Bay, St John, US Virgin Islands. Coral
744 Reefs. 2008;27:191–5.

745 82. Cróquer A, Weil E. Changes in Caribbean coral disease prevalence after the 2005 bleaching
746 event. Dis Aquat Org. 2009;87:33–43.

747 83. Jokiel PL. Temperature Stress and Coral Bleaching. In: Rosenberg E, Loya Y, editors. Coral
748 Health and Disease. Berlin, Heidelberg: Springer Berlin Heidelberg; 2004. p. 401–25. Available
749 from: http://link.springer.com/10.1007/978-3-662-06414-6_23

750 84. Dove SG, Hoegh-Guldberg O. The cell physiology of coral bleaching. In: Phinney JT,
751 Hoegh-Guldberg O, Kleypas J, Skirving W, Strong A, editors. Coastal and Estuarine Studies.
752 Washington, D. C.: American Geophysical Union; 2006. p. 55–71. Available from:
753 http://www.agu.org/books/ce/v061/61CE05/61CE05.shtml

754 85. Baird AH, Bhagooli R, Ralph PJ, Takahashi S. Coral bleaching: the role of the host. Trends
755 in Ecology & Evolution. 2009;24:16–20.

756 86. Fitt WK, Gates RD, Hoegh-Guldberg O, Bythell JC, Jatkar A, Grottoli AG, et al. Response
757 of two species of Indo-Pacific corals, *Porites cylindrica* and *Stylophora pistillata*, to short-term
758 thermal stress: The host does matter in determining the tolerance of corals to bleaching. Journal
759 of Experimental Marine Biology and Ecology. 2009;373:102–10.

760 87. Howells EJ, Abrego D, Meyer E, Kirk NL, Burt JA. Host adaptation and unexpected
761 symbiont partners enable reef-building corals to tolerate extreme temperatures. Glob Change
762 Biol. 2016;22:2702–14.

763 88. Ulstrup K, Berkelmans R, Ralph P, van Oppen M. Variation in bleaching sensitivity of two
764 coral species across a latitudinal gradient on the Great Barrier Reef: the role of zooxanthellae.
765 Mar Ecol Prog Ser. 2006;314:135–48.

766 89. Berkelmans R, van Oppen MJH. The role of zooxanthellae in the thermal tolerance of corals:
767 a 'nugget of hope' for coral reefs in an era of climate change. Proc R Soc B. 2006;273:2305–12.

768 90. Bay LK, Doyle J, Logan M, Berkelmans R. Recovery from bleaching is mediated by
769 threshold densities of background thermo-tolerant symbiont types in a reef-building coral. R Soc
770 open sci. 2016;3:160322.

771 91. Ziegler M. Bacterial community dynamics are linked to patterns of coral heat tolerance.
772 Nature Communications. 2017;8:1–8.

773 92. Shore-Maggio A, Callahan S, Aeby GS. Trade-offs in disease and bleaching susceptibility
774 among two color morphs of the Hawaiian reef coral, *Montipora capitata*. Coral Reefs.
775 2018;37:507–17.

776 93. Merselis DG, Lirman D, Rodriguez-Lanetty M. Symbiotic immuno-suppression: is disease
777 susceptibility the price of bleaching resistance? PeerJ. 2018;6:e4494.

778 94. Ziegler M, Roder C, Büchel C, Voolstra C. Niche acclimatization in Red Sea corals is
779 dependent on flexibility of host-symbiont association. Mar Ecol Prog Ser. 2015;533:149–61.

780 95. Davis KA, Lentz SJ, Pineda J, Farrar JT, Starczak VR, Churchill JH. Observations of the
781 thermal environment on Red Sea platform reefs: a heat budget analysis. Coral Reefs.
782 2011;30:25–36.

783 96. Chaidez V, Dreano D, Agusti S, Duarte CM, Hoteit I. Decadal trends in Red Sea. Scientific
784 Reports. 2017;7:8144.

785 97. Porter JW, Lewis SK, Porter KG. The effect of multiple stressors on the Florida Keys coral
786 reef ecosystem: A landscape hypothesis and a physiological test. Limnol Oceanogr.
787 1999;44:941–9.

788 98. De'ath G, Lough JM, Fabricius KE. Declining Coral Calcification on the Great Barrier Reef.
789 Science. 2009;323:116–9.

790 99. Cantin NE, Cohen A, Karnauskas K, Tarrant A, McCorkle D. Ocean warming slows coral
791 growth in the Central Red Sea. Science. 2010;329:322–5.

792 100. Voolstra C, Valenzuela J, Turkarslan S, Cardenas A, Hume B, Perna G, et al. Contrasting
793 heat stress response patterns of coral holobionts across the Red Sea suggest distinct mechanisms
794 of thermal tolerance. In Review; 2020 Dec. Available from:
795 https://www.researchsquare.com/article/rs-117181/v1

796 101. Kaczmarsky LT, Draud M, Williams EH. Is There a Relationship between Proximity to
797 Sewage Effluent and the Prevalence of Coral Disease? Caribbean Journal of Science.
798 2005;41:14.

799 102. Tanner JE. Competition between scleractinian corals and macroalgae: An experimental
800 investigation of coral growth, survival and reproduction. Journal of Experimental Marine
801 Biology and Ecology. 1995;190:151–68.

802 103. McCook L, Jompa J, Diaz-Pulido G. Competition between corals and algae on coral reefs: a
803 review of evidence and mechanisms. Coral Reefs. 2001;19:400–17.

804 104. Haas AF, Nelson CE, Wegley Kelly L, Carlson CA, Rohwer F, Leichter JJ, et al. Effects of
805 Coral Reef Benthic Primary Producers on Dissolved Organic Carbon and Microbial Activity.
806 editor. PLoS ONE. 2011;6:e27973.

807 105. Barott KL, Rohwer FL. Unseen players shape benthic competition on coral reefs. Trends in
808 Microbiology. 2012;20:621–8.

809 106. Sheppard C, Al-Husiani M, Al-Jamali F, Al-Yamani F, Baldwin R, Bishop J, et al. The
810 Gulf: A young sea in decline. Marine Pollution Bulletin. 2010;60:13–38.

811 107. Riegl B. Corals in a non-reef setting in the southern Arabian Gulf (Dubai, UAE): fauna and
812 community structure in response to recurring mass mortality. Coral Reefs. 1999;18:63–73.

813 108. Riegl BM, Bruckner AW, Samimi-Namin K, Purkis SJ. Diseases, Harmful Algae Blooms
814 (HABs) and Their Effects on Gulf Coral Populations and Communities. In: Riegl BM, Purkis SJ,
815 editors. Coral Reefs of the Gulf. Dordrecht: Springer Netherlands; 2012. p. 107–25. Available
816 from: http://www.springerlink.com/index/10.1007/978-94-007-3008-3_7

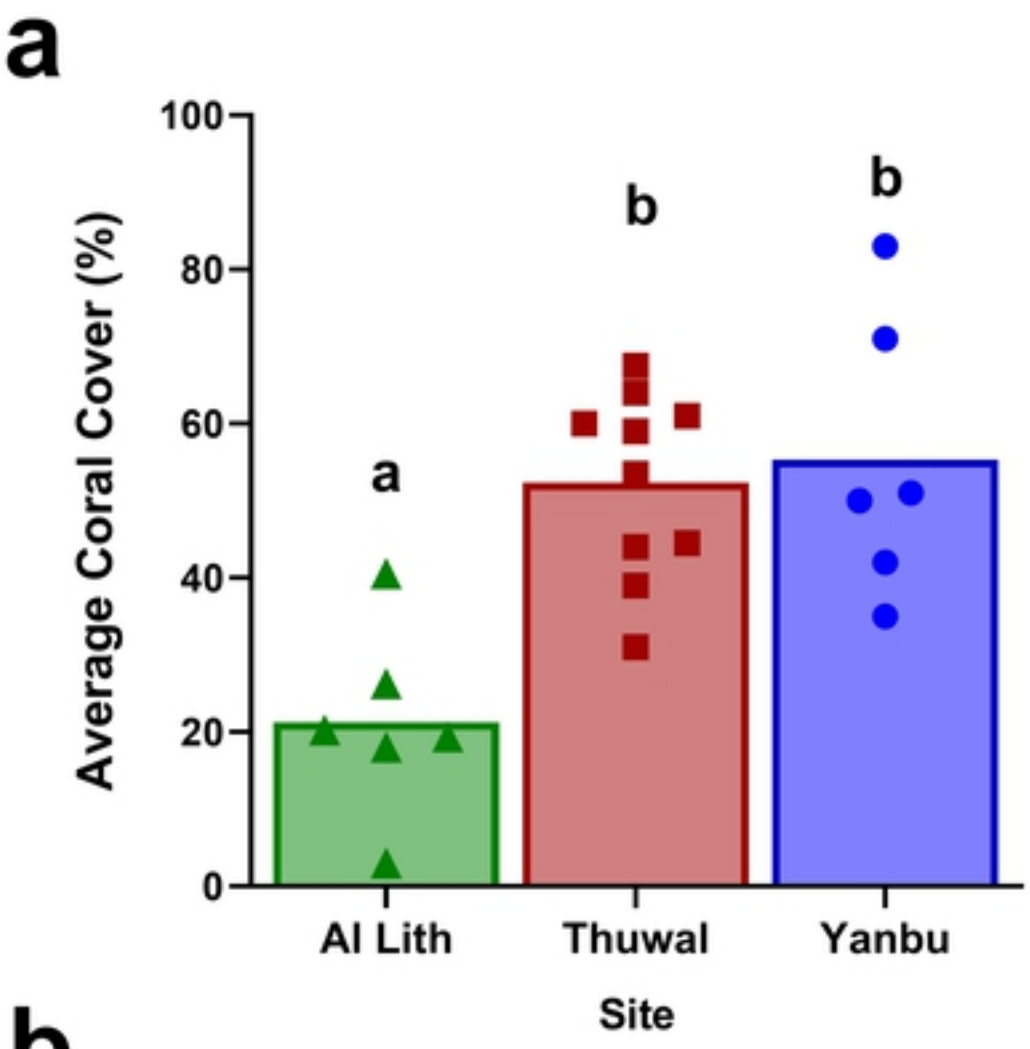
817 109. Alidoost Salimi M, Mostafavi P, Fatemi S, Aeby G. Health status of corals surrounding
818 Kish Island, Persian Gulf. Dis Aquat Org. 2017;124:77–84.

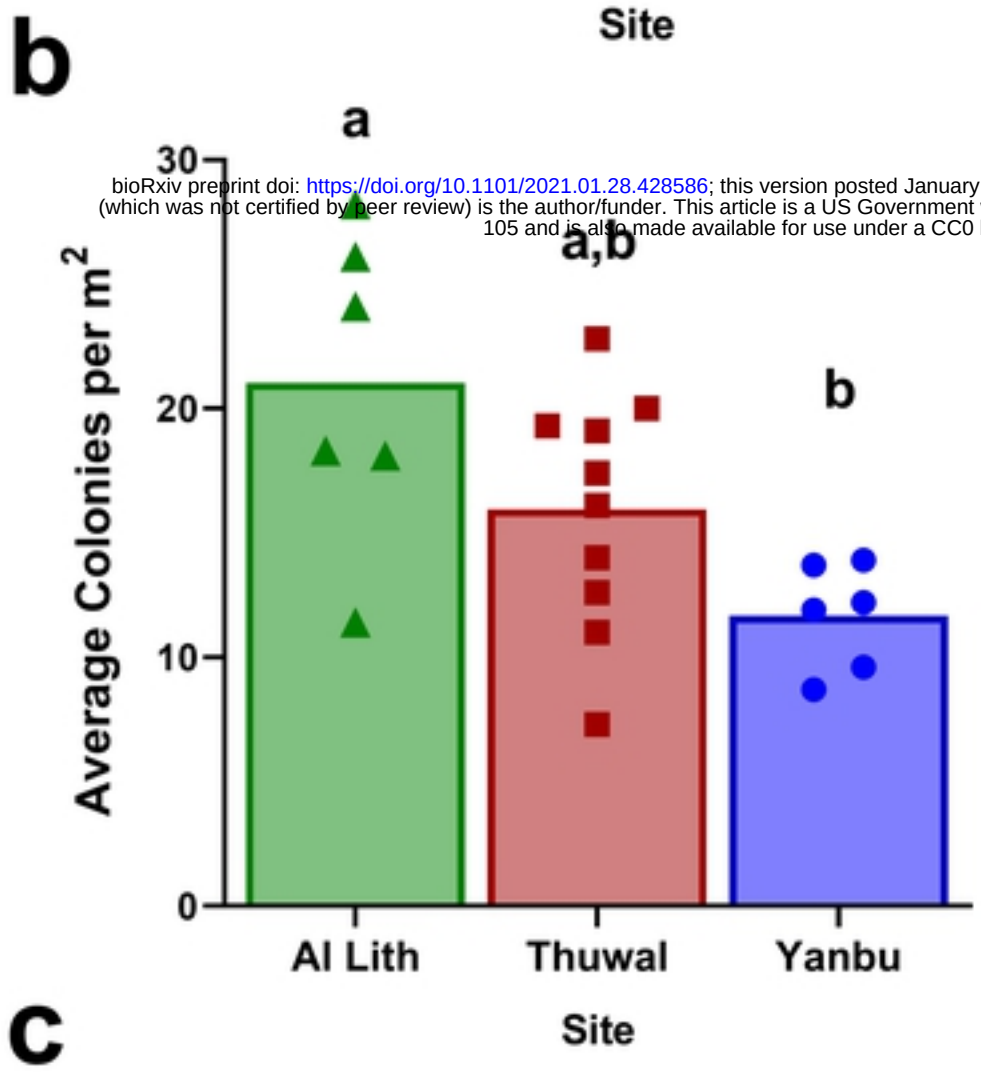
819

820

821

822





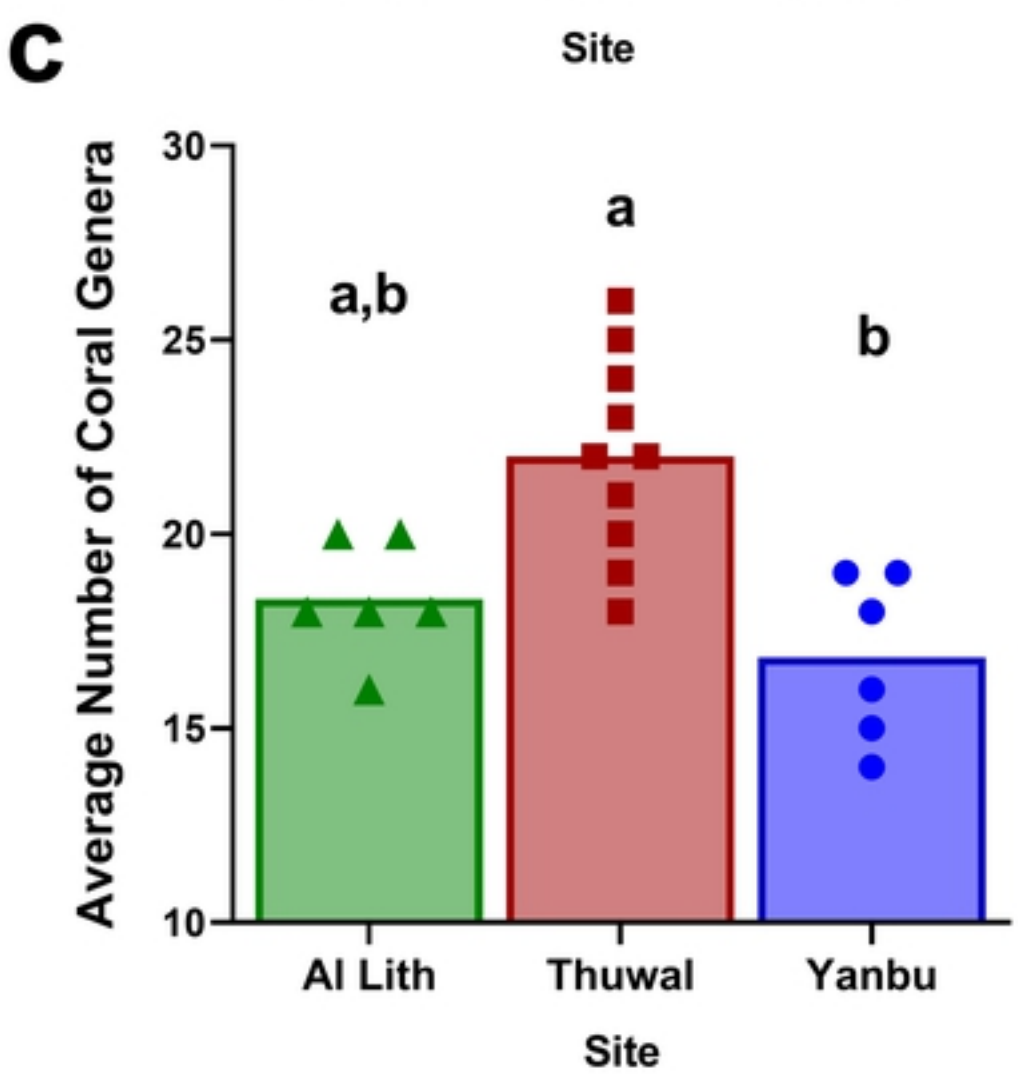


Fig2

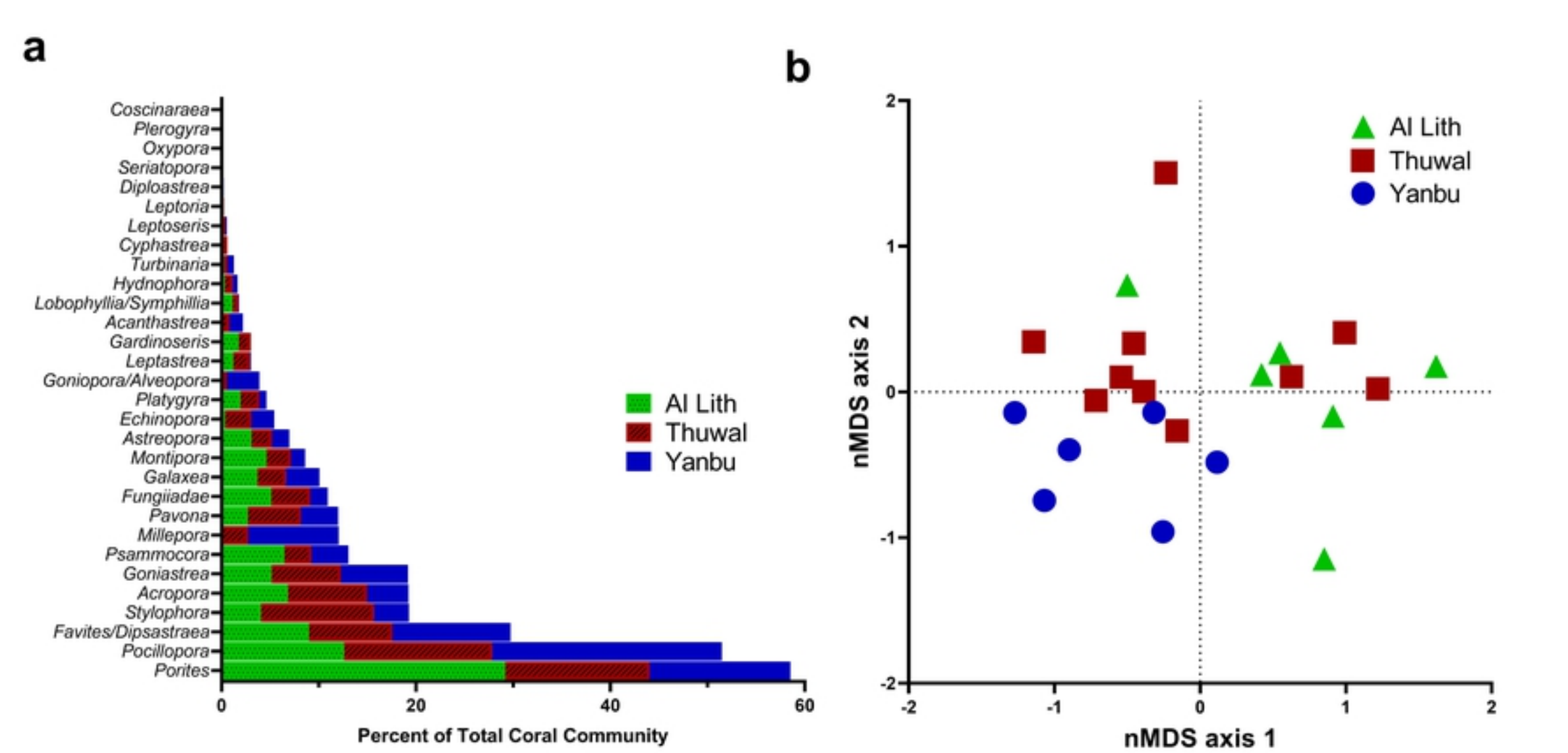

a
Coscinaraea
Plerogyra
Oxypora
Seriatopora
Diploastrea
Leptoria
Leptoseris
Cyphastrea
Turbinaria
Hydnophora
Lobophyllia/Symphillia
Acanthastrea
Gardinoseris
Leptastrea
Goniopora/Alveopora
Platygyra
Echinopora
Astreopora
Montipora
Galaxea
Fungiiadae
Pavona
Millepora
Psammocora
Goniastrea
Acropora
Stylophora
Favites/Dipsastraea
Pocillopora
Porites
0
20
40
60
Percent of Total Coral Community
Al Lith
Thuwal
Yanbu
b
2
1
0
-1
-2
nMDS axis 2
nMDS axis 1
Al Lith
Thuwal
Yanbu

Fig3

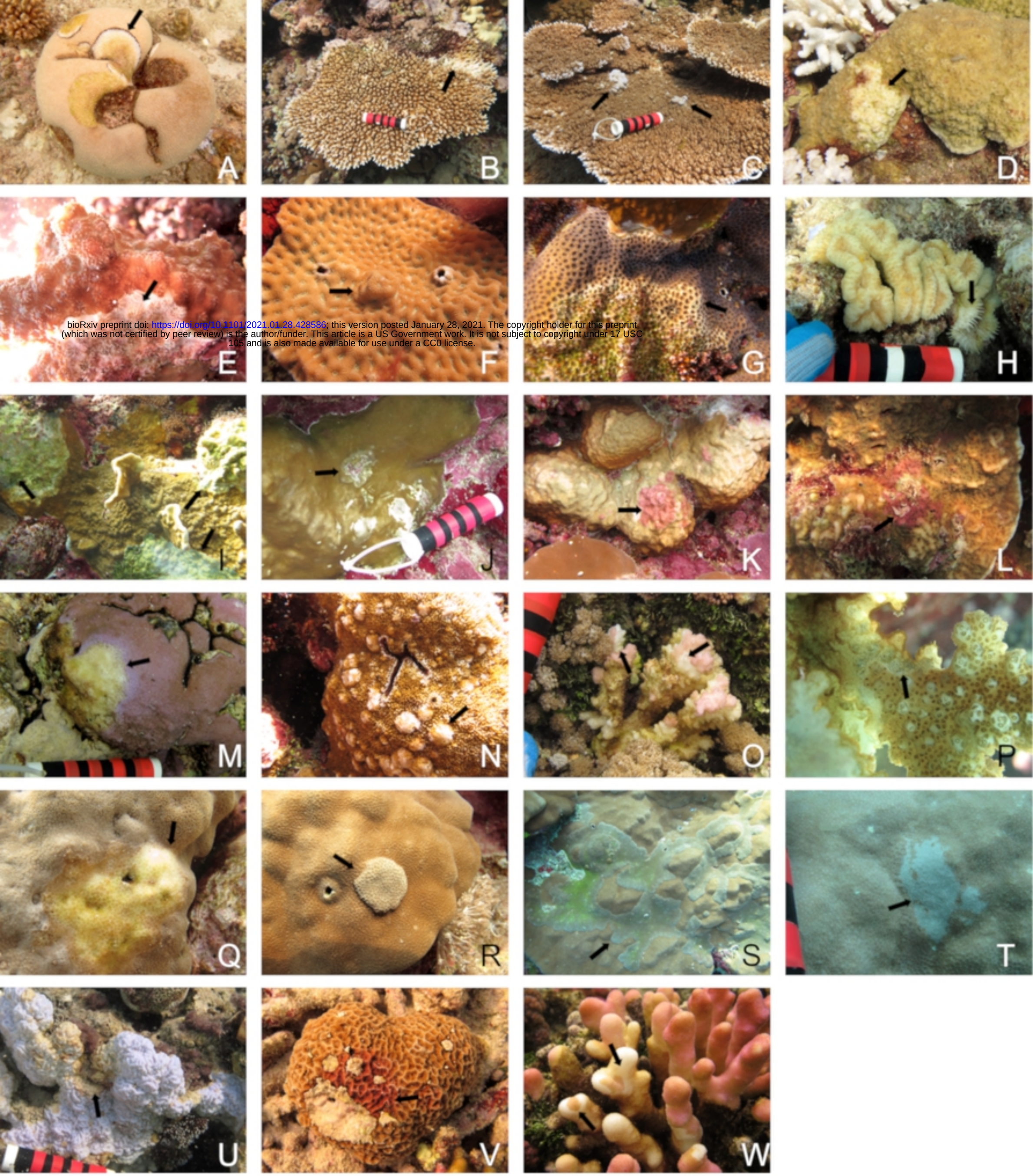
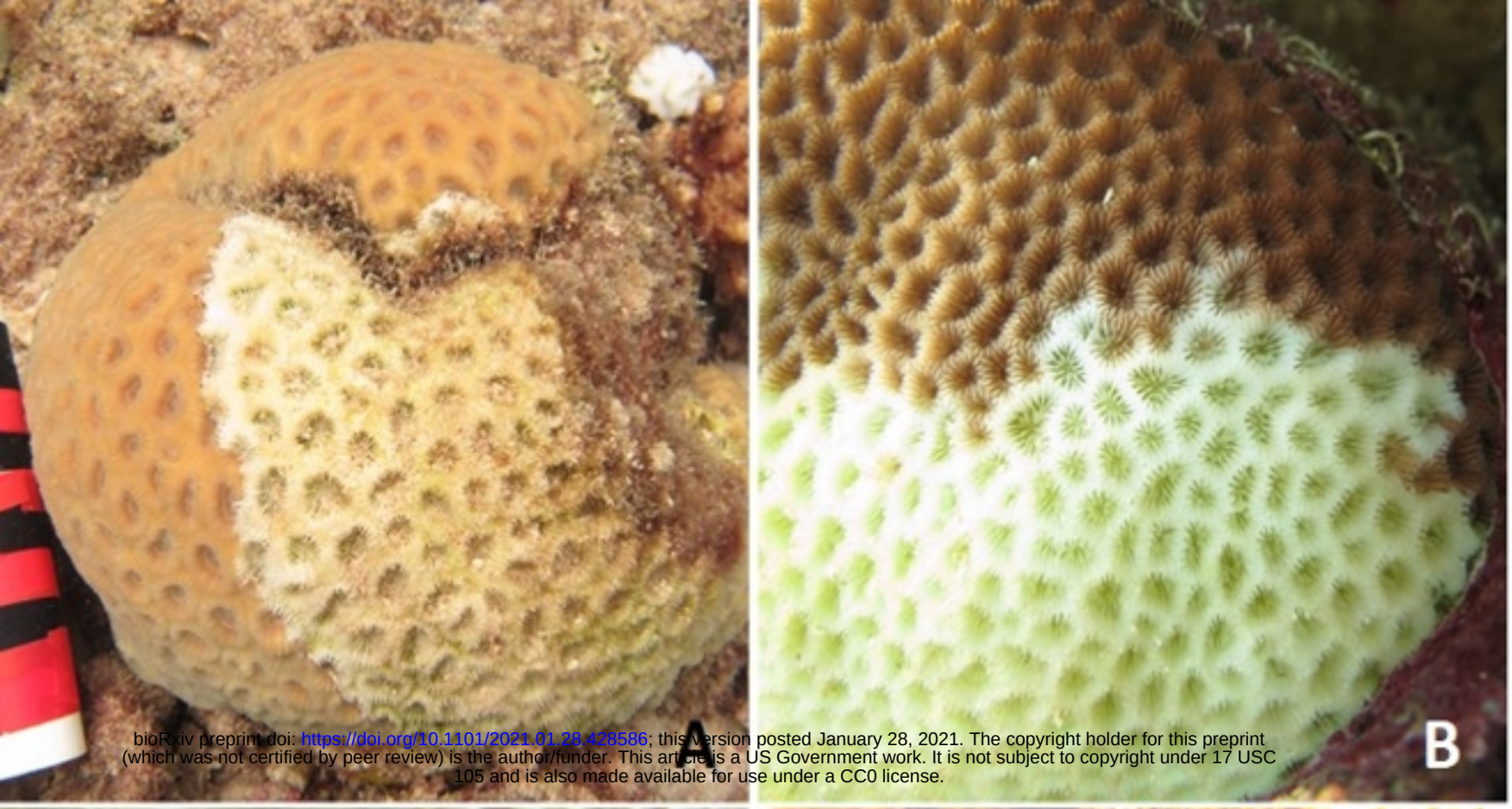
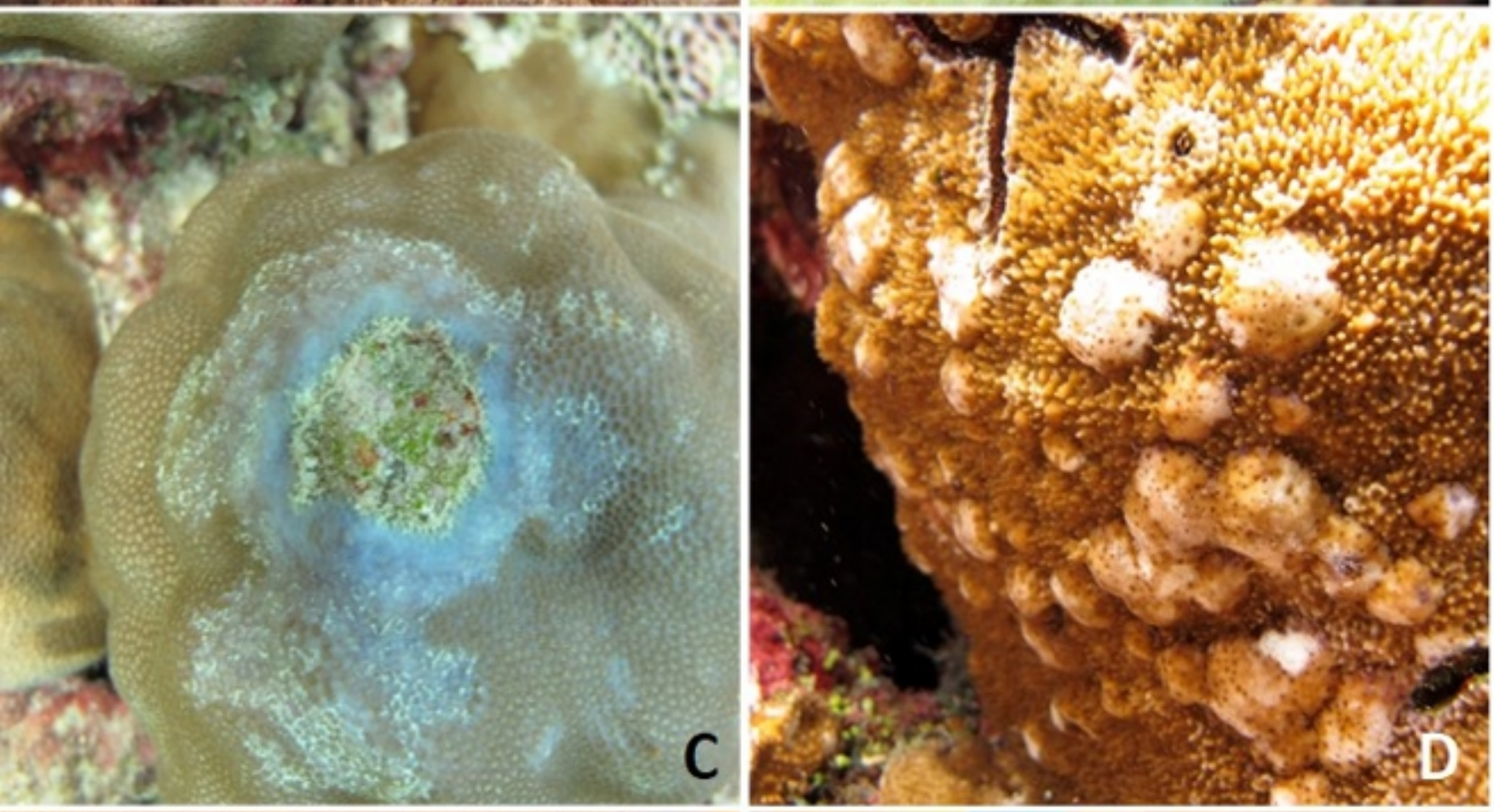


Fig4





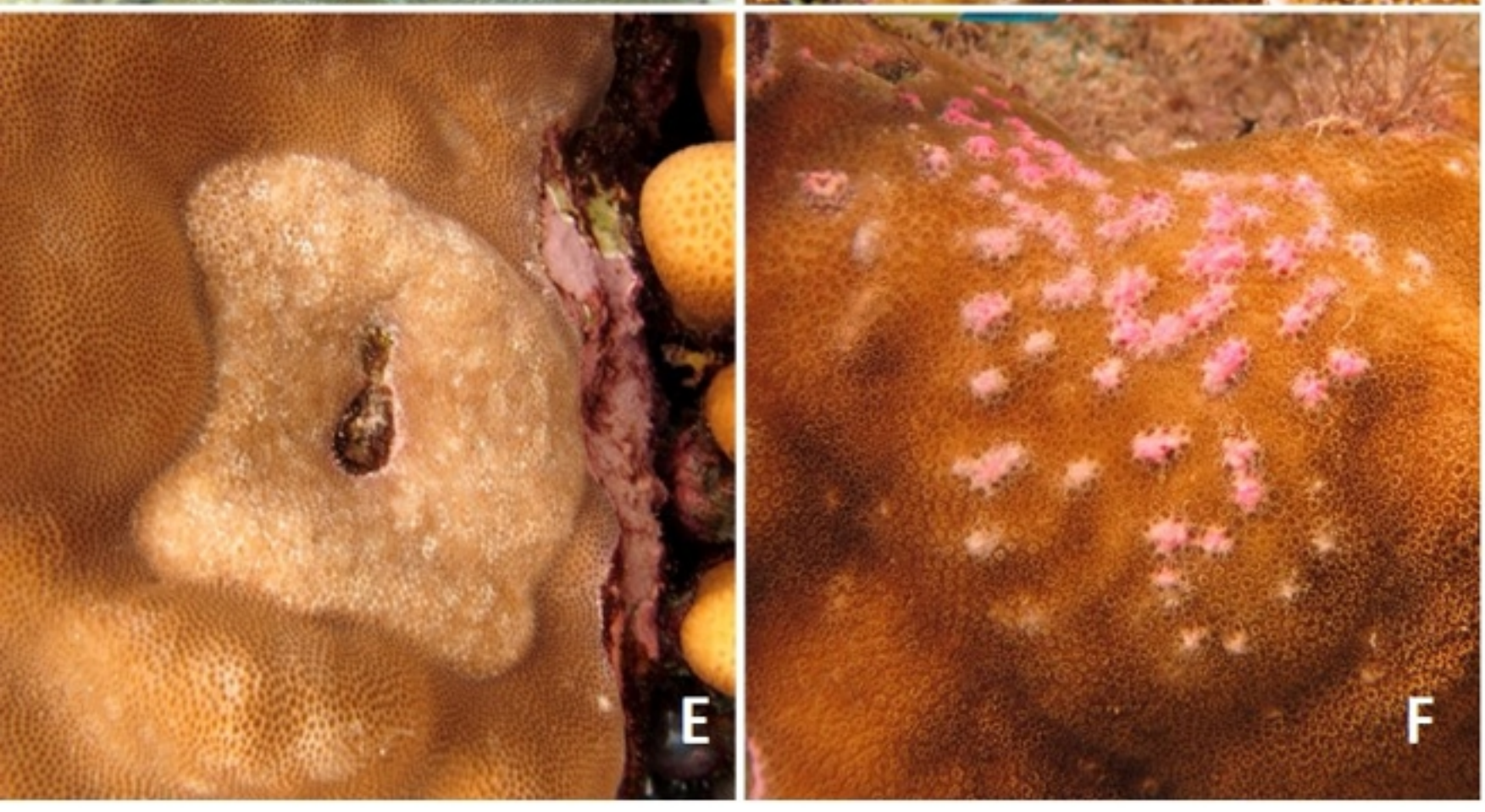
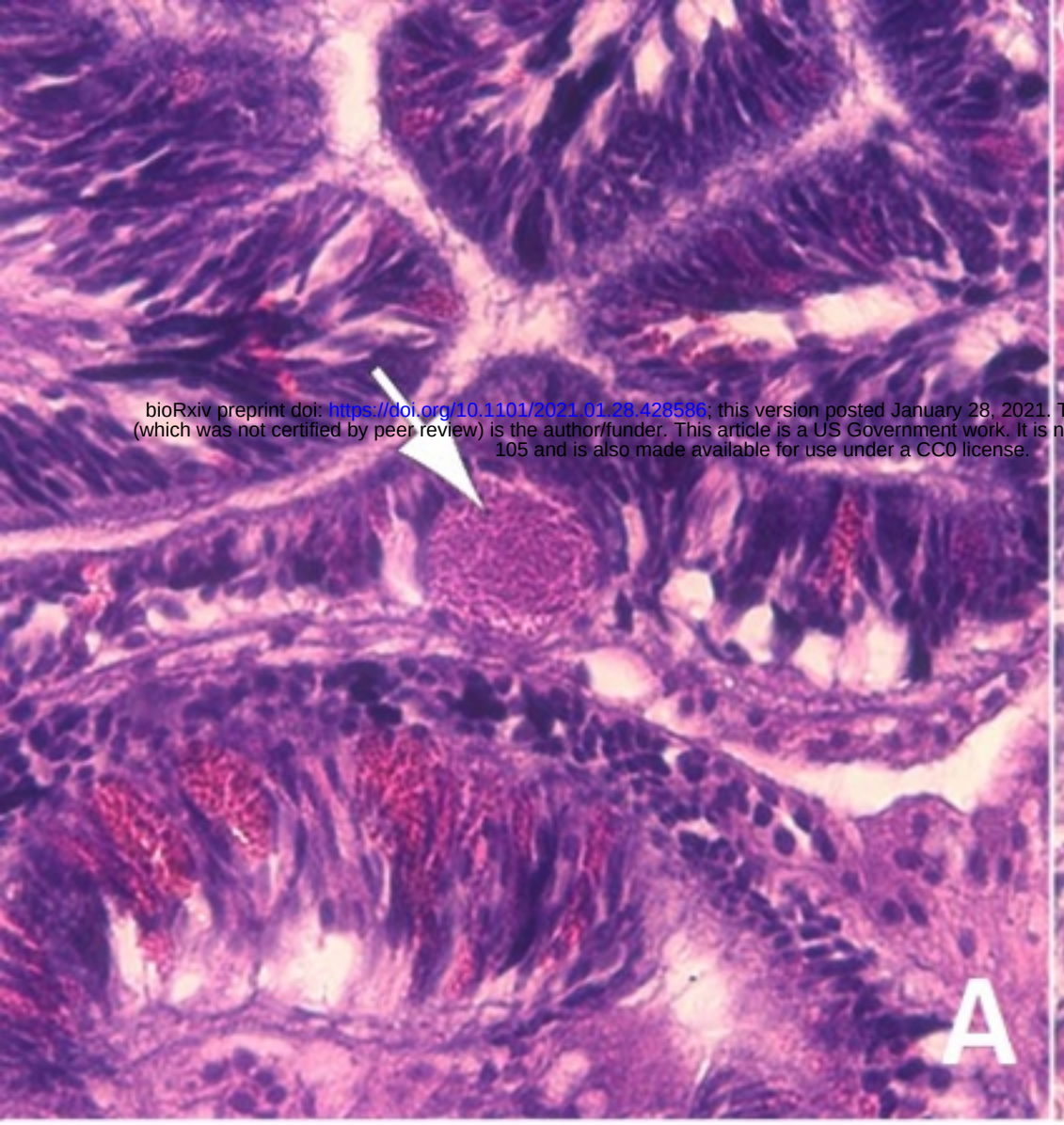
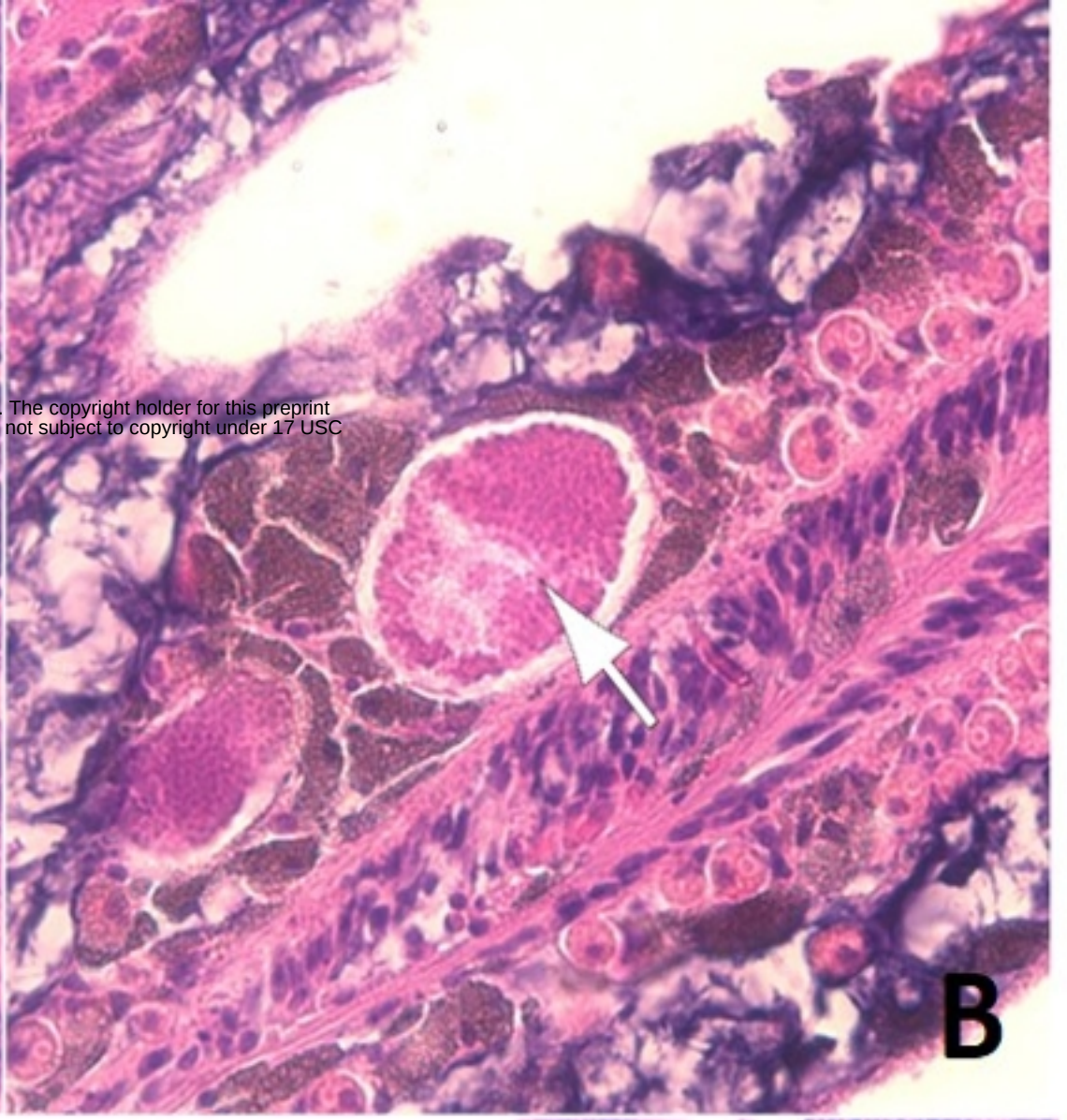
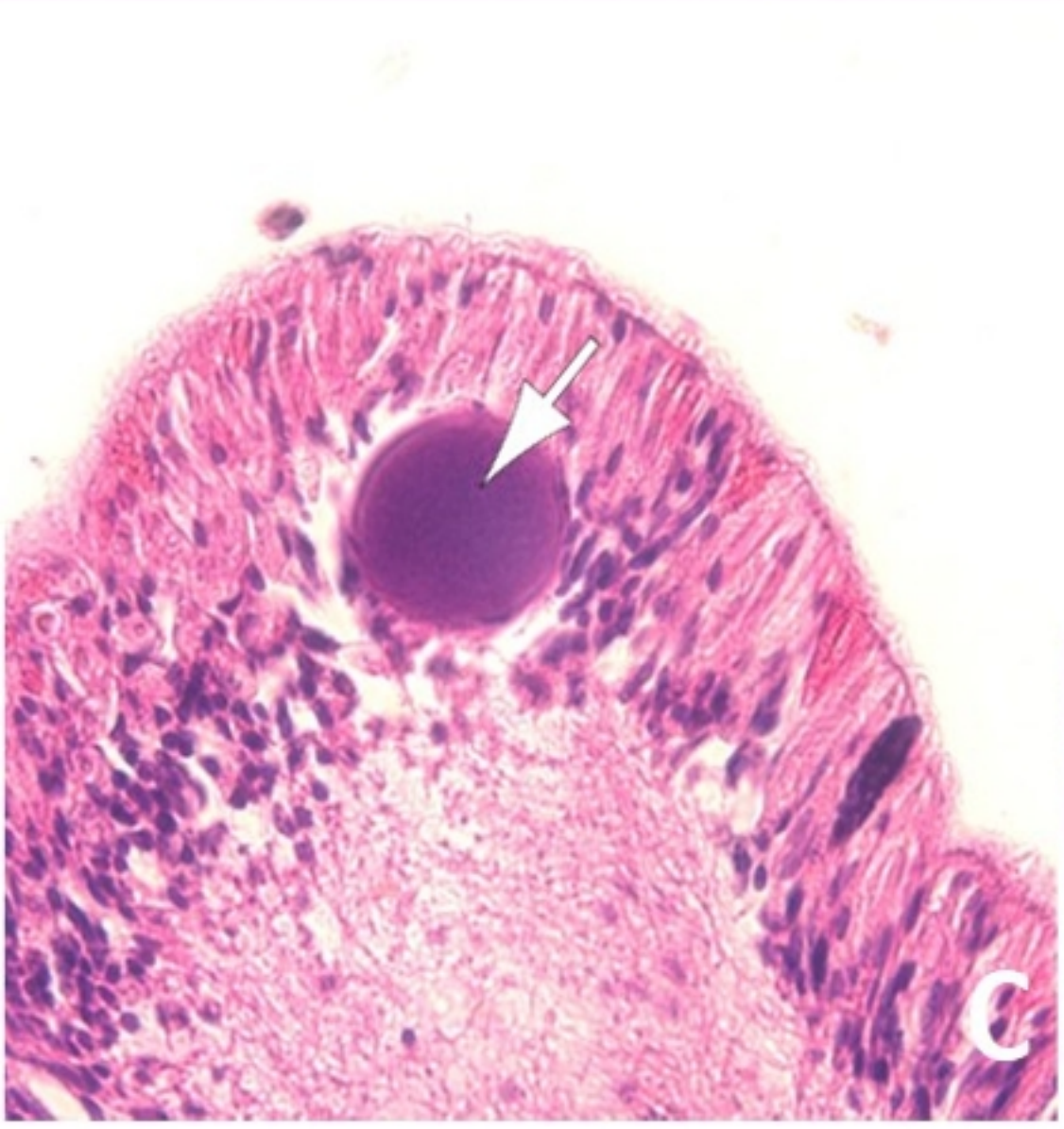


fig5







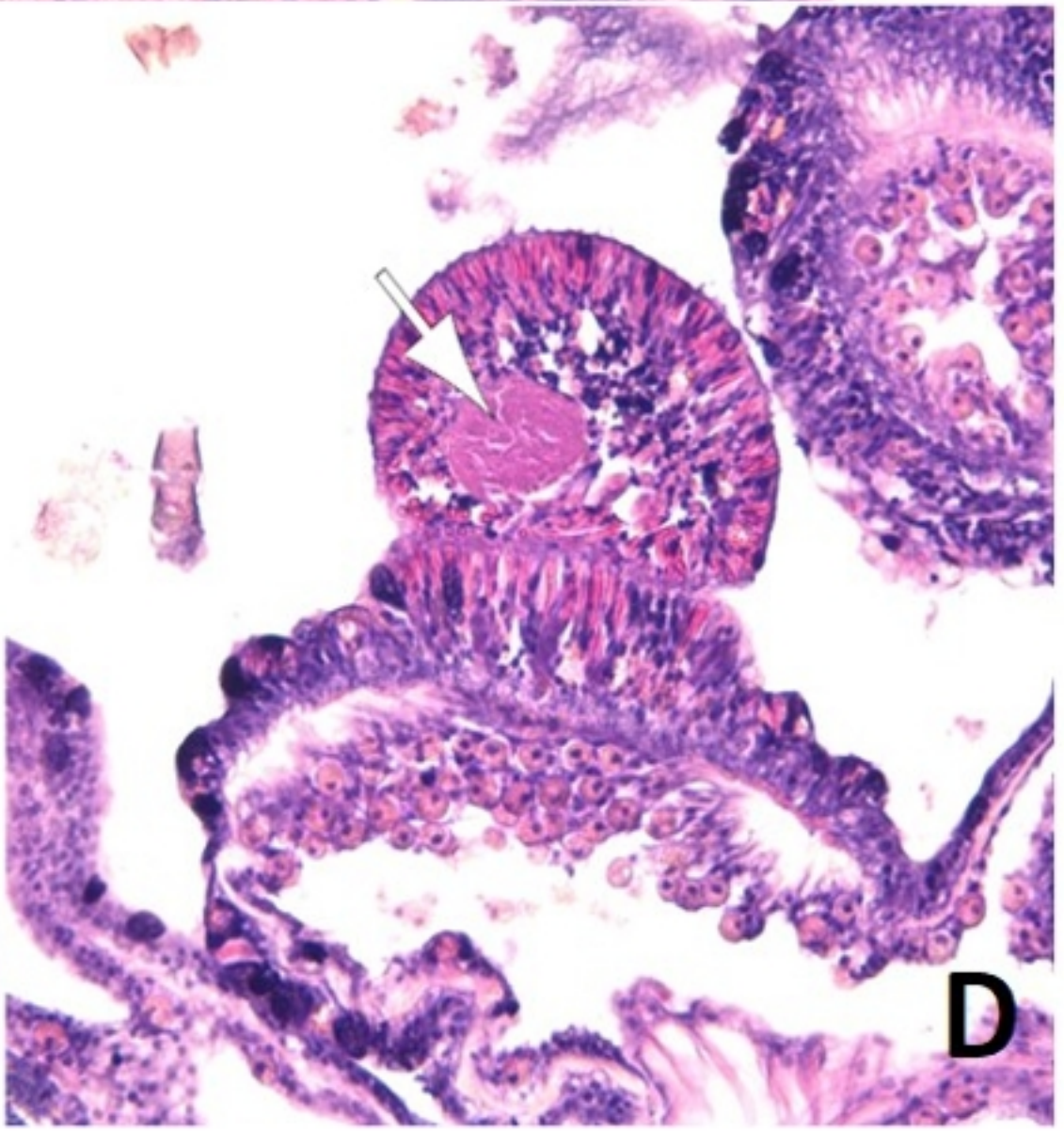


fig6

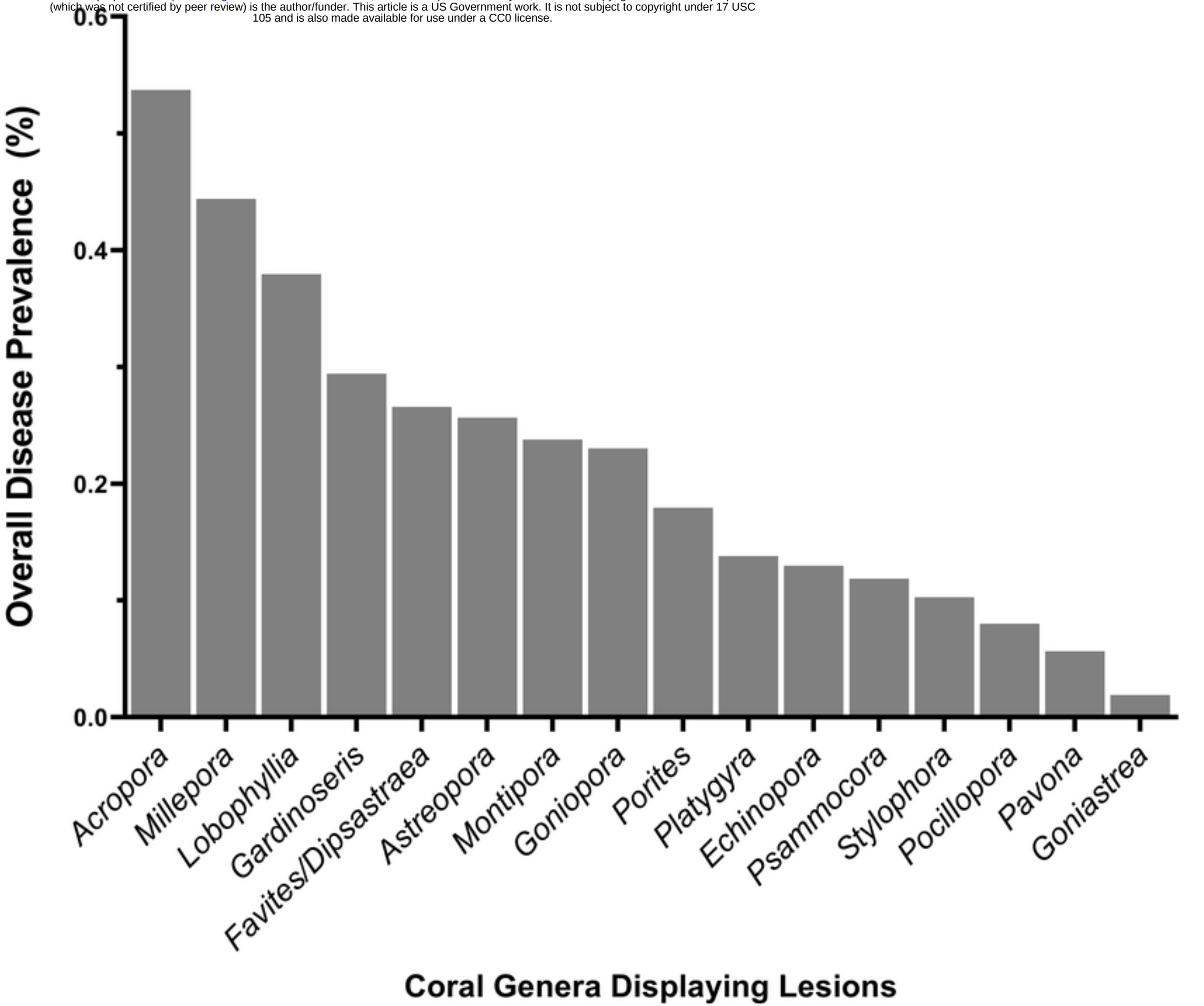


fig7

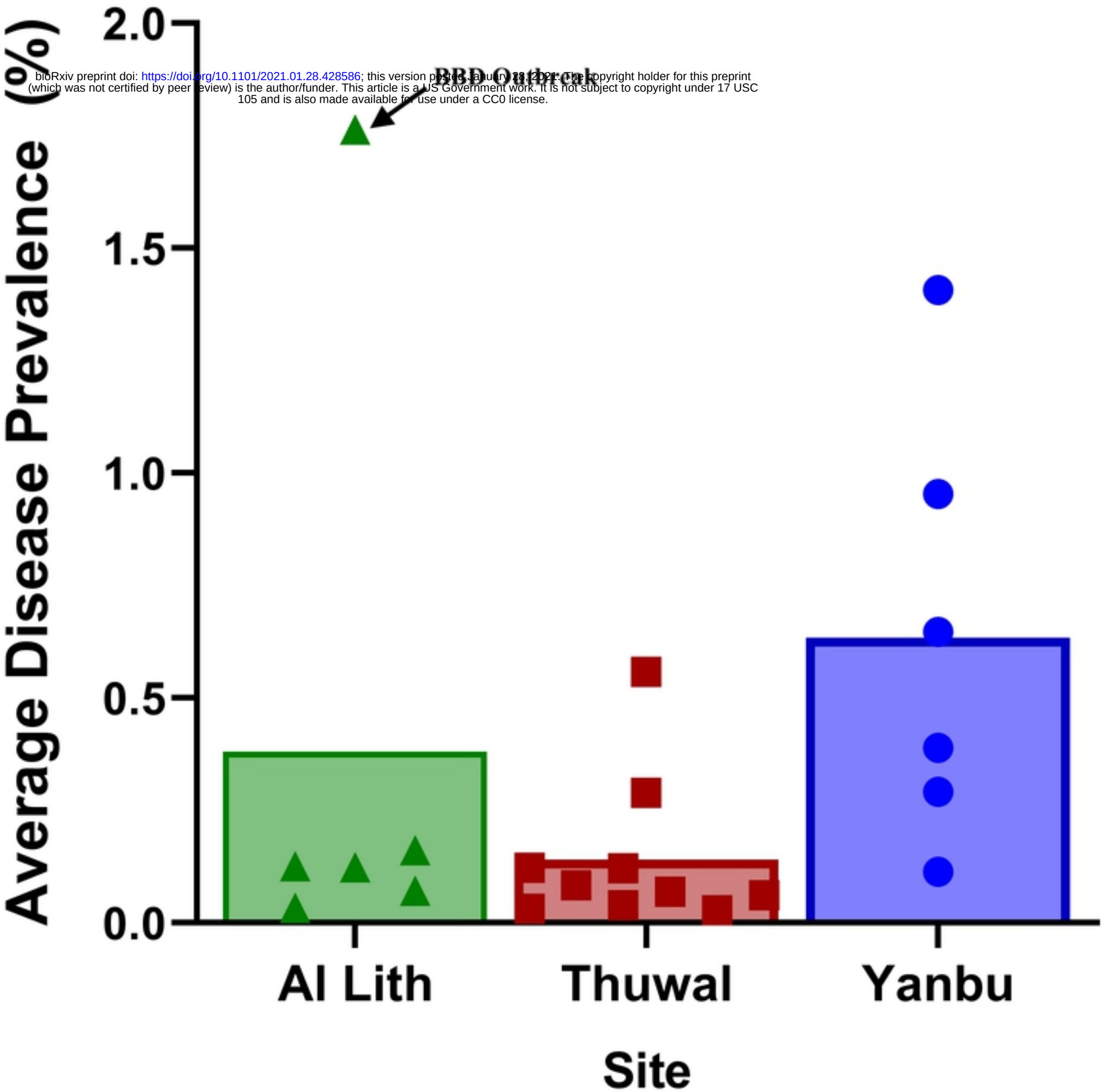


fig8

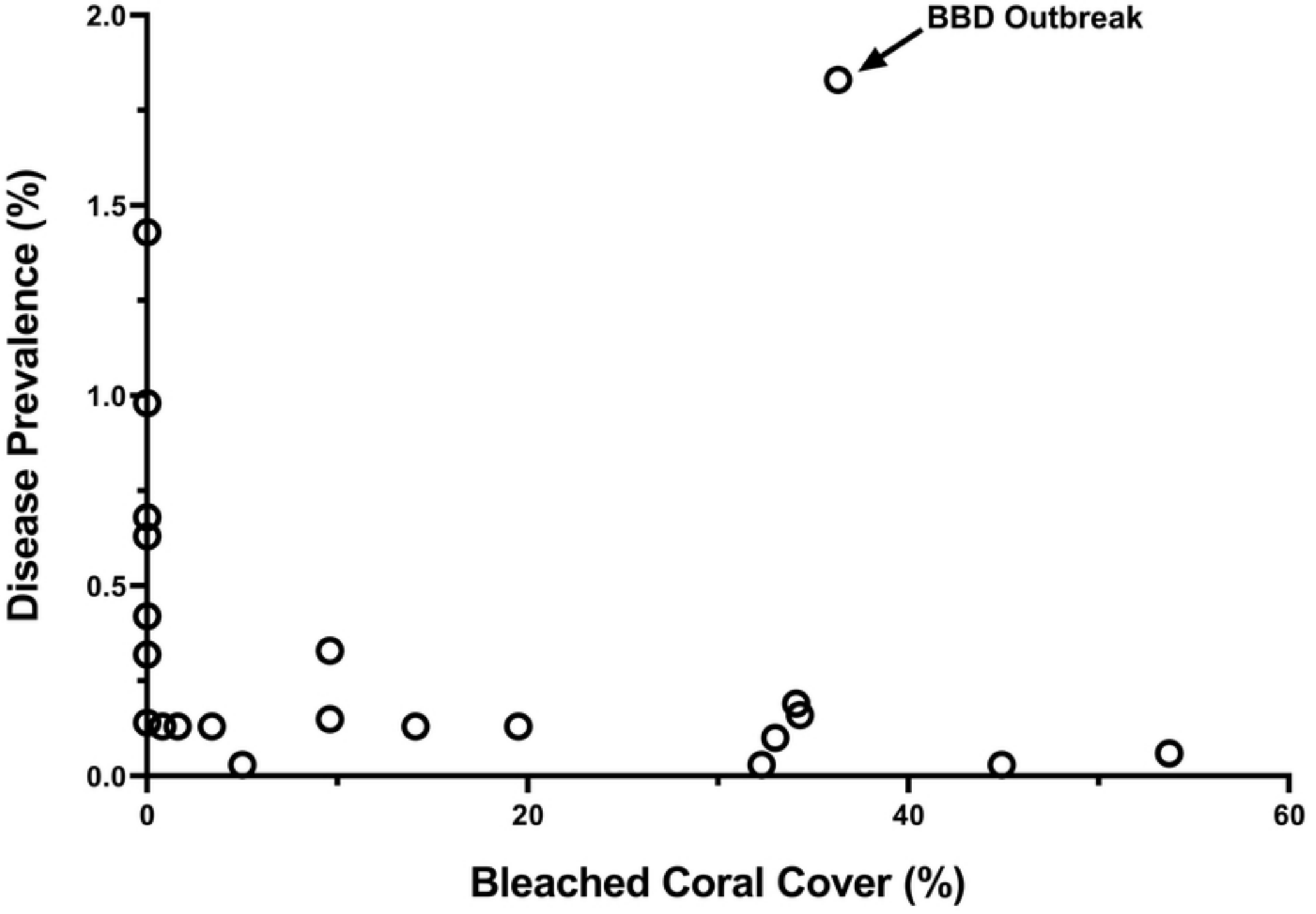

BBD Outbreak
Disease Prevalence (%)
2.0
1.5
1.0
0.5
0.0
0
20
40
60
Bleached Coral Cover (%)

fig9

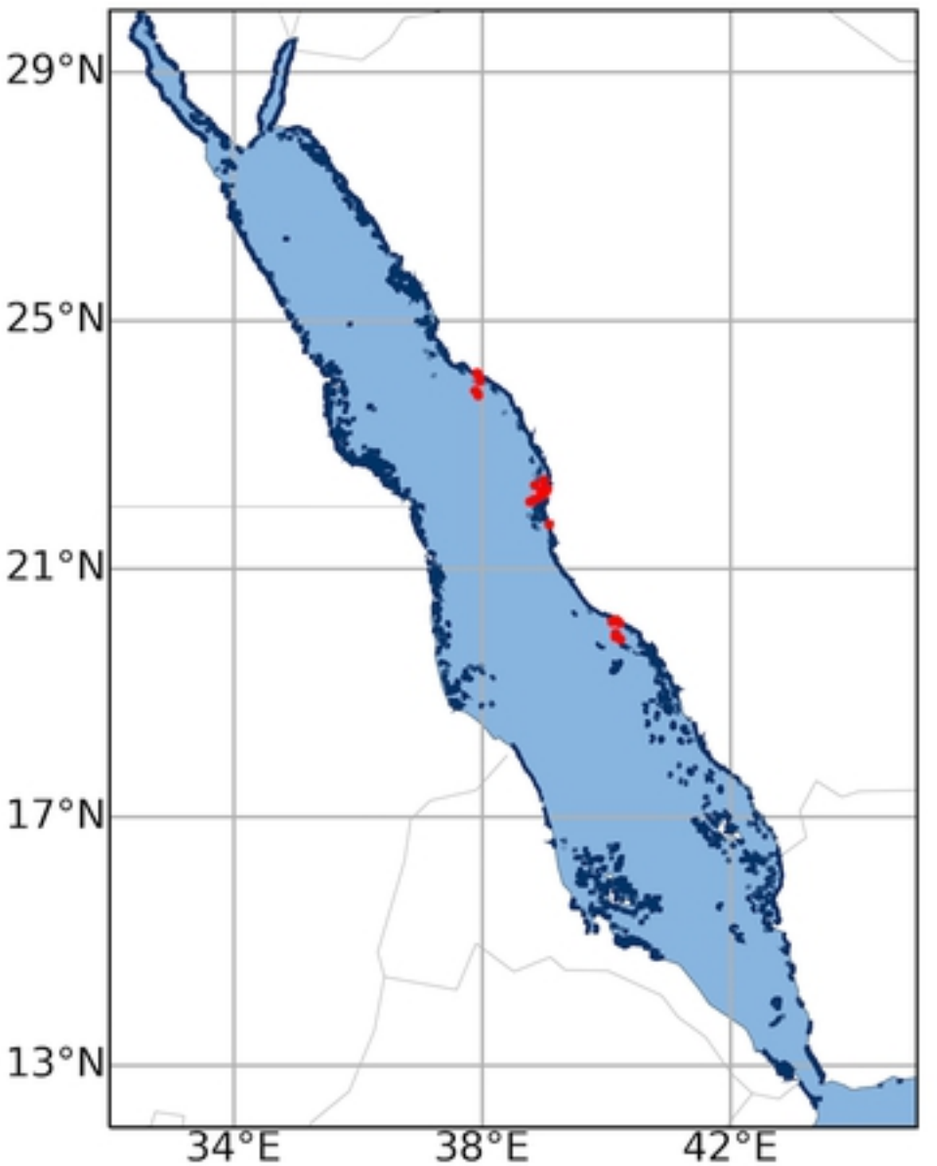


Fig1