



Terrestrial runoff influences white syndrome prevalence in SW Madagascar



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ABSTRACT

Terrestrial runoff and sedimentation have been implicated in a variety of impacts on scleractinian corals. However, despite accumulating evidence, little work has been done to investigate their influence on coral disease development. This study examined the role that river runoff and the associated sedimentation could play in affecting the prevalence of the coral disease “white syndrome” in SW Madagascar. Corals from reefs affected by river discharge and terrestrial sediments were more affected by white syndrome than reefs located far from any source of terrestrial runoff. Terrestrial runoff-affected reefs also displayed a wider diversity of coral species affected by this disease. While much evidence has been pointing in the direction of indirect effects of such runoff on coral disease development, our data corroborates earlier suggestions that pathogens are present within the sediments. As such, sediments released on reefs through river discharge could act as reservoirs of coral pathogens.

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1. Introduction

A variety of environmental and anthropogenic stressors have been implicated as cofactors of coral disease development throughout the world. Among environmental stressors, temperature has been pointed out as one of most important drivers of coral disease outbreaks (Bruno et al., 2007; Harvell et al., 2007), increasing coral disease susceptibility (Palmer et al., 2011) and pathogen virulence (Kimes et al., 2012; Kushmaro et al., 1998). Water quality degradation resulting from anthropogenic influences and terrestrial runoff is another major environmental driver of coral disease. Several water quality parameters have been shown to contribute to coral disease outbreaks, including dissolved organic

carbon (Kaczmarek and Richardson, 2011; Kline et al., 2006) and nutrient enrichment (Haapkylä et al., 2011; Vega Thurber et al., 2014).

Sedimentation is also associated with reduced water quality, potentially resulting from terrestrial runoff and/or dredging-associated activity, and has been frequently suggested for its implication in coral disease development. Peters (1984) suggested that wave-induced sediment resuspension might affect black band disease (BBD) incidence in the Caribbean. Both Bruckner et al. (1997) and Voss and Richardson (2006) observed higher BBD prevalence at sites with higher sedimentation levels, and it was proposed that sediments may be involved by acting as a reservoir of coral pathogens (Harvell et al., 2007; Richardson, 1997; Voss and Richardson, 2006). Sediments could be implicated in coral disease outbreaks through a series of both direct and indirect processes. Sedimentation is generally associated with increased turbidity, nutrient and organic carbon loadings, and reduced photosynthetically active radiations (PAR) available to corals (reviewed in Erfteemeijer et al., 2012). Together, these factors may act to reduce

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coral fitness through decreased photosynthesis (Philipp and Fabricius, 2003) and increased microbial and algal development (Kline et al., 2006; Nugues and Roberts, 2003). In turn, reduced coral fitness may result in an increase in bleaching susceptibility (Wiedenmann et al., 2013), thereby further weakening the coral. Sediments may also directly impact coral fitness by causing lesions through abrasion (PIANC, 2010), modifying the microenvironment at the coral surface through sediment accumulation and increased microbial activity (Weber et al., 2012), and inducing coral investment in energy for sediment removal (Riegl and Branch, 1995; Saunders et al., 2005). Though this is debated (Lesser and Jarett, 2014; Rypien, 2008), opportunistic coral pathogens could also originate from the terrestrial environment, as was suggested for *Serratia marcescens* (Sutherland et al., 2011) and *Aspergillus sidowii* (Geiser et al., 1998; Shinn et al., 2000), and sediments could potentially carry such pathogens within their matrices and promote infection by acting as a reservoir. In particular, *S. marcescens* adsorbed on sediments have been shown to successfully infect the coral *Acropora palmata* and cause white pox (Patterson et al., 2002), thereby providing the required evidence to support this hypothesis.

South-western Madagascar has extensive reef systems along its coasts, which once harboured highly diverse and abundant coral communities (Bruggemann et al., 2012; Harris et al., 2010). Unfortunately dramatic declines in coral cover were measured over the years, particularly as a result of increasing human population and pressure on the environment such as reef exploitation and deforestation (Bruggemann et al., 2012). Such declines have been particularly evident for the Grand Récif de Tuléar (GRT), a reef located some 3–5 km off the coast of Tuléar (Tuléar; Fig. 1). These declines were evidenced through extensive surveys conducted in the 1960's–70's (Andréfouët et al., 2013; Bruggemann et al., 2012; Pichon, 1978) which provided the required baseline data to assess changes in coral cover over the years. For example, the coral cover on the outer reef slope of the GRT declined from 30–100% in the 1960's to 15–30% in the 2000's (Bruggemann et al., 2012). Recent works suggested that this decline in coral cover was in part the result of reduced water quality (high nutrients and suspended sediments; Harris et al., 2010). This reduction was probably associated with increases in terrestrial runoff (as indicated by recent increases in sedimentation; Maina et al., 2012) from nearby rivers (Onilahy and Fiherenana, Fig. 1). However, it would appear that, at least on shallow reef flats, the decline in coral cover was driven by

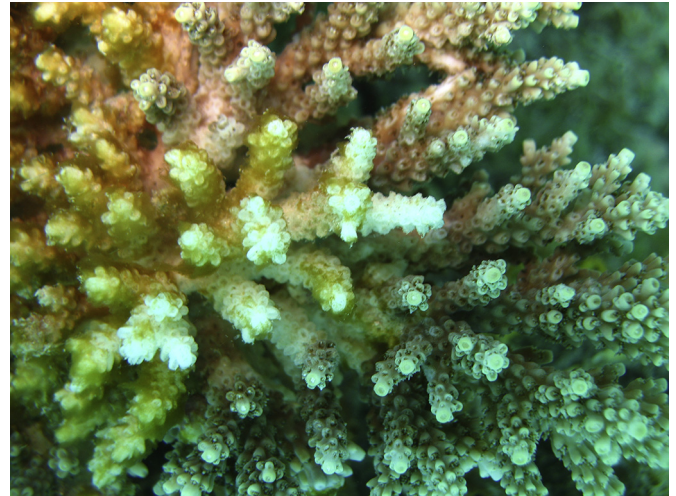


Fig. 2. *Acropora* spp. affected by white syndrome (WS) in SW Madagascar.

damages resulting from local fishing/gleaning pressure (Andréfouët et al., 2013). Also, in view of the oligotrophic water conditions on the GRT (Arfi et al., 2007), nutrients may not be a major driver of this decline. While important declines in coral cover have also been documented for the outer reef slopes of the GRT (Harris et al., 2010), the influence of terrestrial runoff and sediment loads on this decline have not been investigated.

In SW Madagascar, the GRT has undergone severe declines in coral cover (Bruggemann et al., 2012) for reasons as yet poorly understood. Because the GRT is heavily affected by river discharge, particularly during the rainy season (December–April), this work focused on assessing the role of terrestrial runoff, and sedimentation in particular, on the prevalence of white syndrome (WS; Bythell et al., 2004; Willis et al., 2004). WS is a dominant disease (81.3% of recorded coral diseases; Table S1) affecting corals on reefs in SW Madagascar. It is a relatively poorly defined disease (Sweet and Bythell, 2012) affecting scleractinian corals in the Indo-Pacific, and is typically characterised by white lesions resulting from diffuse tissue loss (Fig. 2). This disease has been putatively ascribed to a variety of microorganisms including bacteria (Luna et al., 2010; Séré et al., 2013), ciliates (Sweet and Bythell, 2012;

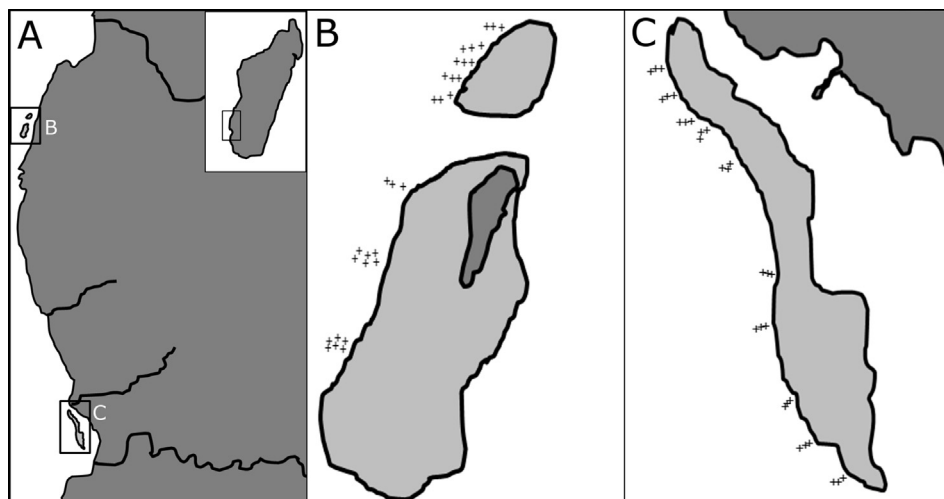


Fig. 1. Location map of the Andavadoaka Reef System (ARS) and the Grand Récif de Tuléar (GRT) in SW Madagascar (A). Disease prevalence survey sites for the ARS (B) and GRT (C) are presented as "+". Coral reefs = light grey areas, land = dark grey areas.

Work and Aeby, 2011) and fungi (Work and Rameyer, 2005). WS abundance is generally positively correlated with host density (Aeby et al., 2010; Hobbs and Frisch, 2010; Muller et al., 2012) and was also recently correlated with dredging-associated sediment plumes (Pollock et al., 2014).

2. Materials and methods

2.1. Study sites

This study was conducted between January and April 2011 during the rainy season on two barrier reef systems in SW Madagascar: the Grand Récif de Tuléar (GRT) and another reef system off the shores of Andavadoaka (Andavadoaka Reef System, ARS; Fig. 1). The GRT is a majorly over-fished (Harris et al., 2010) 18 km long barrier reef system located ~2 km from the shores of Tuléar town and affected by heavy sedimentation loads originating from nearby rivers Fiherenana (~1 km North of the GRT) and Onilahy (1–2 km SE of the GRT). The Onilahy is likely to be the main contributor of sediments affecting this reef as southerly winds tend to drive sediment plumes northward (Maina et al., 2012). In contrast to the GRT, the ARS is not affected by any nearby river runoff, with the nearest river estuary located around 70 km north of the ARS, and terrigenous sediment levels much lower than for the GRT (Nadon et al., 2007; D. Griffiths personal communication). Also, the fishing pressure from local communities of Andavadoaka, Ampasilava and Nosy Hao (tot. pop. 1850) is relatively minor (Nadon et al., 2007) when compared to the intense pressure on the GRT (Andréfouët et al., 2013). The ARS is located 150 km north of the GRT and consists of two barrier reefs surrounding the small islands of Nosy Hao and Nosy Fasy (~5.5 km and ~2.1 km long respectively).

2.2. White syndrome prevalence

For each reef system, we selected 10 sites evenly spread (mean distance between sites: ARS = 2.35 ± 0.31 km, GRT = 5.76 ± 0.78 km) along the outer reef slope (Fig. 1). At each site, three 2 m × 50 m belt transects at 5 m, 10 m and 15 m deep and parallel to the reef were surveyed using SCUBA, resulting in a total of 3000 m² of surveyed area for each reef system. Each WS affected colony was characterised following the detailed disease assessment protocol described in Raymundo et al. (2008). Briefly, recorded data included total number of coral colonies per transect, coral species identification, colony size, lesion type, lesion distribution, lesion location, lesion size, lesion colour, lesion margin type, lesion severity (=percentage of each coral colony surface affected by disease) and lesion duration estimation. These data were used as parameters to positively identify lesions as WS, and to calculate lesion severity and WS prevalence (calculated for each transect as the number of WS affected colonies divided by the total number of coral colonies (both healthy and diseased)). Shannon-Weaver coral species diversity indices were also calculated for each reef system.

2.3. Environmental parameters

2.3.1. Sediments

In order to measure sedimentation rate for each reef system, three sediment traps (7.5 cm × 60 cm) were deployed at several outer reef transect sites (5 m, 10 m and 15 m; $n_{\text{GRT}} = 8$, $n_{\text{ARS}} = 5$). Deployment intervals were spread evenly (mean sediment deployment interval: ARS = 6.62 ± 1.72 days, GRT = 8.13 ± 4.69 days) throughout the duration of the coral disease prevalence data collection process (see Section 2.1), and for durations of 3.5 ± 1.3 days. Each trap consisted of a 30 cm PVC tube, a 10 cm ziplock bag

attached to the bottom end of the tube funnelling settling sediments into a 50 ml collection tube forming the bottom end of the trap. Each trap was attached to a 1 m long metal stake fixed into the substrate at least 2 m away from other traps, and so that the mouth of each trap was the culminating point in the water flow and was ~80 cm above the substrate. Sediments accumulated in the 50 ml collection tubes were desalted thrice in ddH₂O before being dried to constant weight at 60 °C. Sediment trap accumulation rates were calculated for each site as the average amount of sediment dry weight (DW) collected by each sediment trap at each site over the duration of deployment. The ash-free dry weight (AFDW) of each sediment sample was determined by combustion of dried sediments at 450 °C for 4 h. Total organic content was determined as the AFDW subtracted from the total DW.

Given the low quantities of collected sediments, grain size distributions were determined using image analysis on thin-sections after a technique similar to Francus (1998). Briefly, sediment samples were added into well plates and embedded with low-viscosity epoxy resin (Araldite 2020). Standard petrographic thin-sections (30 μm thick) were prepared from the hardened block. Digital micrographs (3 replicates/sediment sample) of the thin-sections were captured under a light microscope and processed in ImageJ software (<http://imagej.nih.gov/ij/>) to obtain an estimate of the grain size distribution.

X-ray diffraction (XRD) was used for semi-quantitative analysis of the mineral composition of the sediments. Approximately 1 cm³ of dried sediments were ground and homogenised in triplicate to a grain size of 50 μm and pressed into XRD sample holders. The samples were X-rayed with a Bruker-Siemens D5000 diffractometer operated at 40 kV and 30 mA (CuK α radiation). The XRD spectra were recorded from 4 to 70° 2 θ with a 0.05° 2 θ step length and 3 s counting time per step. Calcite, Mg-calcite, aragonite, quartz and clay minerals (kaolinite) were identified and their relative abundance was approximated by measuring the intensity of their strongest XRD peak. Peak data were then corrected following Brouard (1991) to allow for comparison of mineral content (in weight %) between the different samples.

2.3.2. Water

Temperature was recorded during each transect using a dive computer-based thermometer (UWATEC Smart Pro). Photosynthetically active radiation (PAR) data were obtained for each transect at each depth by means of an Apogee MQ200 quantum meter. The time of PAR measurement was recorded in order to apply a correction for solar elevation angle applied using the algorithm from the NOAA Earth System Research Laboratory – Global Monitoring Division (2014).

2.4. Identification of potential coral pathogens present within sediments

Sediment (including pore/surrounding water) samples were collected using a sterile pipette from nine sediment traps ($n_{5\text{m}} = 3$, $n_{10\text{m}} = 4$, $n_{15\text{m}} = 2$) on the outer GRT, sonicated for 15 min, cultured on both TCBS and Marine Agar 2216 (BD Diagnostics) media and sub-cultured twice to ensure isolation of pure colonies. Visually different colonies were sampled and fixed in 100% ethanol then stored at –20 °C until analysed. Bacterial DNA was isolated using the Invisorb Spin Tissue Mini kit (Invitex, Berlin, Germany) following manufacturer's instructions. Nearly full length 16S rRNA gene sequences were amplified using either the primers 8F–1492Rm or GM5F–GM8R (Muyzer et al., 1995; Roesch et al., 2007) using PuReTaq™ Ready-To-Go PCR Beads (GE Healthcare) and a Touchdown PCR protocol following Becker et al. (2007). Amplified DNA was purified using the MSB PCRapace purification

kit (Invitex, Berlin, Germany) and sequenced in both directions by Beckman Coulter. Obtained sequences were aligned and checked against the GenBank database to identify related species (sequences deposited on GenBank under accession numbers KJ718923–53). Sequences were considered as potential coral pathogens if the nearest phylogenetic neighbour was either isolated from a coral disease lesion, or other diseased marine organisms.

2.5. Statistical analyses

All statistical analyses were performed using the Kruskal–Wallis rank sum test as the assumptions required for the application of parametric statistics did not hold. All statistical tests were performed using the R-package v3.0.0 (R Core Team 2014).

3. Results

3.1. White syndrome prevalence

Overall WS prevalence was significantly higher within the GRT than in the ARS (Kruskal–Wallis; $H_{(1)} = 15.39$, $p < 0.001$; Fig. 3) with a total of 64 and 12 diseased colonies respectively. Lesion severity was also higher within the GRT when compared to the ARS (Kruskal–Wallis; $H_{(1)} = 10.18$, $p < 0.01$; Fig. 4). WS affected a larger diversity of coral species in the GRT (38 species from 21 genera; Shannon–Weaver index (H) = 3.903 ± 0.01) than in the ARS (13 species from 9 genera; Shannon–Weaver index (H) = 2.668 ± 0.02 ; Table S2). The average total amount of coral colonies per transect (both healthy and diseased) was also higher on the GRT than on the ARS ($n_{GRT} = 191.65$, $n_{ARS} = 126.62$; Kruskal–Wallis $H_{(1)} = 20.52$, $p < 0.001$), giving an overall coral density of $1.91 \text{ coral m}^{-2}$ (GRT) and $1.27 \text{ coral m}^{-2}$ (ARS).

3.2. Environmental parameters

3.2.1. Sediments

While the recorded sediment accumulation rates were not significantly different between the ARS and the GRT ($p = 0.128$), significant differences were observed in their composition. Sediments accumulated in traps deployed within the GRT had an organic content on average 1.6 times higher than those from the ARS (Kruskal–Wallis $H_{(1)} = 8.691$, $p < 0.05$; Fig. 5). Important differences were also observed for sediment mineral compositions, with significantly more clays (kaolinite, Kruskal–Wallis $H_{(1)}$, $p < 0.001$) and less aragonite (Kruskal–Wallis $H_{(1)}$, $p < 0.01$), and to a lesser extent, quartz (Kruskal–Wallis $H_{(1)}$, $p < 0.05$) in the GRT than in the ARS (Fig. 6). Grain size distributions were relatively similar between both reef systems (Fig. 7).

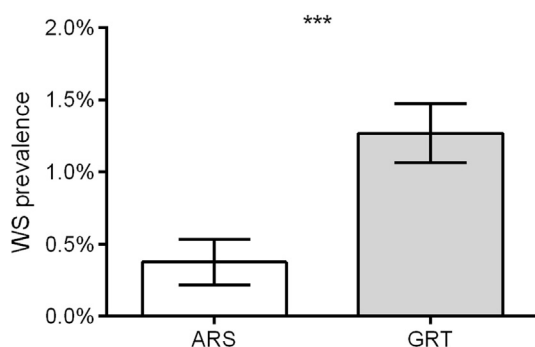


Fig. 3. WS prevalence (\pm SE) in the Andavadoaka reef system (ARS; control) and the Grand Récif of Tuléar (GRT). Significance level: *** = 99.9%.

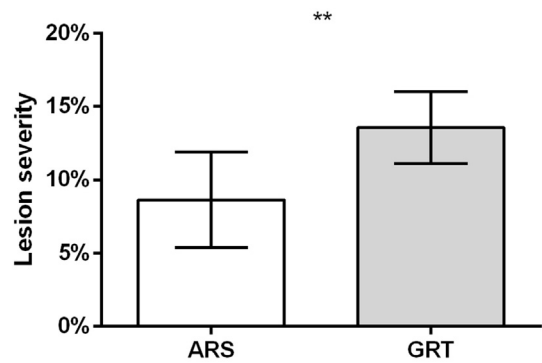


Fig. 4. Lesion severity (\pm SE) as the average percentage of coral colony surface affected by disease in the Andavadoaka reef system (ARS; control) and the Grand Récif of Tuléar (GRT). Significance level: ** = 99%.

3.2.2. Water

The average PAR values across all depths were not significantly different between the ARS and the GRT (Kruskal–Wallis, $p = 0.48$; ARS = $282.95 \mu\text{mol m}^{-2} \text{s}^{-1}$, GRT = $222.22 \mu\text{mol m}^{-2} \text{s}^{-1}$). However, a significant difference was detected between mean PAR_{5m} of the ARS/GRT (Kruskal–Wallis, $H_{(1)} = 4.053$, $p < 0.05$; Fig. 8). Average temperatures across all depths were not significantly different between both reef systems (ARS = 27.59°C , GRT = 27.66°C ; $p = 0.43$).

3.3. Identification of potential coral pathogens present within sediments

A total of 43 bacterial colonies were successfully isolated and sequenced from sediment samples (Table 1). All identified bacteria belonged to either α -proteobacteria (Rhodobacteraceae) or γ -proteobacteria, and were primarily composed of *Pseudoalteromonas* spp., *Vibrio* spp., *Rugueria* spp. and *Pseudovibrio* spp., and 25.6% of all cultured colonies were related to potential coral pathogens. In turn, 72.7% of those were closely related to bacteria directly isolated from diseased corals. One colony in particular was phylogenetically related to a *Vibrio* spp. isolated from a WS affected coral.

4. Discussion

Studies considering the effects of terrestrial runoff on coral disease development have primarily been focussing on the impact caused by nutrient and particulate organic carbon enrichment (Haapkylä et al., 2011; Vega Thurber et al., 2014), but have so far neglected the role that sedimentation might be playing. In this

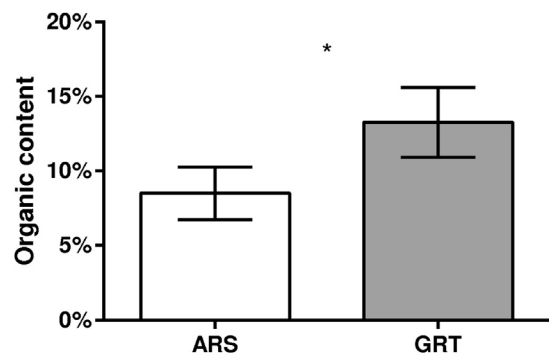


Fig. 5. Comparison of average total organic content (\pm SE) from sediments accumulated within traps deployed on the Andavadoaka reef system (ARS; control) and the Grand Récif of Tuléar (GRT). Significance level: * = 95%.

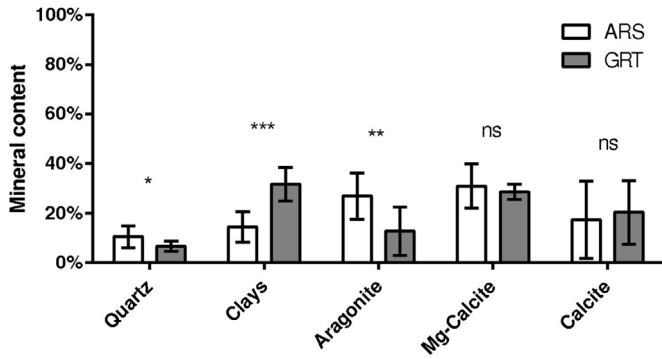


Fig. 6. Mineral class distribution of sediments accumulated in the sediment traps deployed on the Andavadoaka reef system (ARS; control) and the Grand Récif of Tuléar (GRT). Significance level: * = 95%, ** = 99%, *** = 99.9%, ns = non-significant.

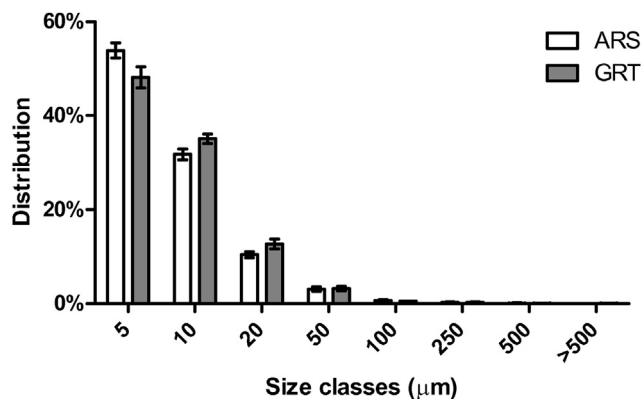


Fig. 7. Grain size distribution of trapped sediments for both Andavadoaka reef system (ARS; control) and Grand Récif of Tuléar (GRT).

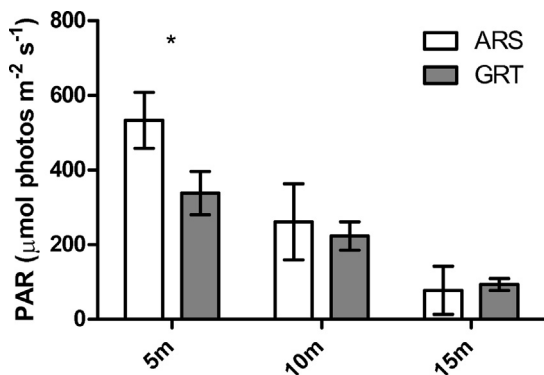


Fig. 8. Average photosynthetically active radiation (PAR) recorded for the ARS and GRT. Data are represented for each depth and **** denotes 95% statistical significance.

study we provide evidence that terrestrial runoff may affect the prevalence of WS in SW Madagascar, and that sedimentation may particularly be involved by acting as a reservoir of coral pathogens as well as a stressor to corals.

Coral reefs in SW Madagascar and the GRT in particular have undergone a major decline (both in coral cover and shallow habitat diversity; Bruggemann et al., 2012) in the last 50 years, which has been primarily attributed to both high fishing pressure and seawater eutrophication resulting from increasing terrestrial runoff (Andréfouët et al., 2013; Harris et al., 2010). As well as nutrients, the Onilahy and Fiherenana rivers discharge increasing amounts of fine

sediments on the GRT (Bruggemann et al., 2012; Maina et al., 2012), a stress to which the ARS is not subjected. While the GRT is usually more influenced by sedimentation than the ARS (GRT: Harris et al., 2010; ARS: Nadon et al., 2007; C. Sheridan personal observation), our data remain inconclusive. This inconsistency may however result from sediment resuspension generated by high wind-induced wave surge on the ARS (C. Sheridan personal observations), as supported by significant differences between ARS and GRT organic and mineral contents. In particular, the higher proportion of clays and organic content in GRT sediments suggests that, during the rainy season, the outer reef slopes on the GRT receive much larger inputs of terrigenous sediments than those of the ARS. This difference is also supported by the higher turbidity (as indicated by lower light penetration) observed at shallow (5 m) GRT sites when compared to the ARS. These terrigenous inputs most likely originate from the Onilahy and Fiherenana rivers carrying increasing levels of land sediments as a result of intense deforestation (Bruggemann et al., 2012; Maina et al., 2013). Kaolinite is the only detected clay species and this mineral is known to originate from tropical soils (Thaymuang et al., 2013).

In this study we found that WS was significantly more prevalent and affected a broader range of coral species within the GRT than in the ARS. The larger diversity of WS-affected corals in the GRT is however unlikely to result from a higher number of coral species within this reef system since the ARS reportedly has a higher coral diversity than the GRT (Fenner, 2005; Pichon, 1978). The observed prevalence levels, though relatively low, were in line with observations from other geographical locations (e.g. Indonesia: 0.42%, Haapkylä et al., 2007; Cocos Islands: $0.85 \pm 0.63\%$, Hobbs and Frisch, 2010). Higher WS prevalence levels have however also been reported (e.g. Christmas Islands $13 \pm 5.5\%$, Hobbs and Frisch, 2010), particularly when host density is high (Aeby et al., 2010; Hobbs and Frisch, 2010). The higher WS prevalence observed in the GRT is therefore consistent with its coral density (1.5x higher than in the ARS).

Coral diseases tend to be influenced by a variety of environmental factors (Harvell et al., 2007). Among such factors, thermal stress is generally considered as a major environmental driver of pathogen virulence and disease prevalence (Harvell et al., 2007), particularly for WS (Bruno et al., 2007). However, there was no significant difference in average seawater temperature between these two reef systems. The observed difference in WS prevalence and severity could therefore not be attributed to this stressor. Such differences could be explained by the higher density of coral colonies (Aeby et al., 2010; Bruno et al., 2007) and the higher anthropic (e.g. fishing/gleaning, pollution) pressure within the GRT (Bruggemann et al., 2012; Harris et al., 2010). For example, anthropic pressure on the GRT may have resulted in the measured increases in algal cover (Harris et al., 2010) and in turn, algae may have acted as a reservoir of WS pathogens (Sweet et al., 2013). However, the observed differences in terrestrial runoff and sediment regime, illustrated by higher water turbidity as well as higher sediment organic and terrigenous (clay) inputs on the GRT, provide a viable alternative explanation. In particular the presence of potential coral pathogens (e.g. *Vibrio harveyi*) within the sediments collected on the GRT combined with the potential influence of sediments on coral immunity and energy reserves (Sheridan et al., in press) may explain the observed difference in WS prevalence.

Terrestrial runoff and sedimentation have been shown to affect water quality and stress corals in a variety of ways (reviewed in Erfteimeijer et al., 2012; Risk and Edinger, 2011). In particular, terrestrial runoff and sedimentation have been shown to affect both the photosynthetic efficiency of symbiotic zooxanthellae and coral energy reserves (Flores et al., 2012; Saunders et al., 2005;

Table 1

Nearest phylogenetic neighbour for each bacterial colony successfully isolated from sediments caught using sediment traps on the outer reef slope of the GRT. Potential coral pathogens are indicated by an "X". All E-values were equal to 0.0 and are therefore not provided.

Medium	Taxon	Nearest neighbour	Accession#	Source tissue	Potential pathogen	Seq length	Similarity
Marine Agar	Bacteria	Bacterium 1H215	JF411476.1	Growth anomaly of <i>Platygyra carnosus</i>	X	1330	97%
Marine Agar	Bacteria	Bacterium 2D802	JF411487.1	Diseased coral	X	1302	99%
TCBS	Bacteria	Bacterium 3H101	JF411528.1	Growth anomaly of <i>Platygyra carnosus</i>	X	1408	99%
TCBS	Bacteria	Bacterium 4D713	JF411563.1	Diseased coral	X	1394	99%
Marine Agar	Bacteria	Bacterium MPII.18	JN699099.1	<i>Axinella polypoides</i> (marine sponge)/ Israel		1299	82%
Marine Agar	Bacteria	Bacterium S_D75_164	JX033658.1	Diseased parts of diseased Red Sea sponge <i>Negombata magnifica</i>	X	439	98%
TCBS	Bacteria	Bacterium T12(2011) strain T12	JN119276.1	Coral mucus + tissue		1373	99%
TCBS	γ -Proteobacteria	<i>Photobacterium jeanii</i> strain R-40508	GU065210.1	Mucus of apparently healthy <i>Palythoa caribaeorum</i> (zoanthid)		1405	99%
TCBS	γ -Proteobacteria	<i>Photobacterium</i> sp. JSA04	KC012646.1	<i>Cymodocea serrulata</i> (seagrass)		1398	99%
Marine Agar	γ -Proteobacteria	<i>Pseudoalteromonas citrea</i> strain BF21e	HQ439546.1	Reef surface biofilm, kahenohe bay, Hawaii		537	75%
Marine Agar	γ -Proteobacteria	<i>Pseudoalteromonas luteoviolacea</i>	JN119275.1	<i>Acropora millepora</i> tissue + mucus		1380	99%
Marine Agar	γ -Proteobacteria	<i>Pseudoalteromonas rubra</i> strain AN34	JQ409378.1	Marine habitat		408	80%
Marine Agar	γ -Proteobacteria	<i>Pseudoalteromonas</i> sp. 2ta17	FJ952783.1	Healthy tissue <i>Montastrea annularis</i>		775	100%
Marine Agar	γ -Proteobacteria	<i>Pseudoalteromonas</i> sp. AKA07-7b	AB571948.1	Marine biofilm that induce <i>Acropora</i> larvae settlement, Okinawa, Japan		1402	92%
Marine Agar	γ -Proteobacteria	<i>Pseudoalteromonas</i> sp. J104(2011)	JF314512.1	Isolated from <i>Neogoniolithon fosliei</i> (Heydrich)		766	99%
Marine Agar	γ -Proteobacteria	<i>Pseudoalteromonas</i> sp. NBRC 101808	AB681566.1	Bacterial culture collection National Biological Resource Centre		1363	99%
Marine Agar	γ -Proteobacteria	<i>Pseudoalteromonas</i> sp. PaD1.15a	GQ406584.1	<i>Pseudopterogorgia americana</i> SML; diseased	X	1378	99%
Marine Agar	γ -Proteobacteria	<i>Pseudoalteromonas</i> sp. PP87	JX075060.1	Phyllosoma of <i>Panulirus ornatus</i> , Coral Sea, Australia		568	97%
Marine Agar	γ -Proteobacteria	<i>Pseudoalteromonas</i> sp. PS5	JF309049.1	<i>Paragoniolithon solubile</i> (crustose coralline algae)		724	99%
Marine Agar	γ -Proteobacteria	<i>Pseudoalteromonas viridis</i>	AB681561.1	Bacterial culture collection		1376	99%
Marine Agar	γ -Proteobacteria	<i>Pseudoalteromonas viridis</i> strain C22c	HQ439520.1	Reef surface biofilm, kahenohe bay, Hawaii		654	90%
Marine Agar	α -Proteobacteria	<i>Pseudovibrio</i> sp. 01OK 105-5-5	AB612240.1	Ascidian, coral reef, Okinawa Japan		1318	99%
Marine Agar	α -Proteobacteria	<i>Pseudovibrio</i> sp. 01OK 105-5-5	AB612240.1	Ascidian, coral reef, Okinawa Japan		1275	73%
Marine Agar	α -Proteobacteria	<i>Pseudovibrio</i> sp. 01OK 105-5-5	AB612240.1	Ascidian, coral reef, Okinawa		1332	99%
Marine Agar	α -Proteobacteria	<i>Pseudovibrio</i> sp. AM56	JQ342682.1	Cultured coral (<i>Briareum</i> sp.)		240	94%
Marine Agar	α -Proteobacteria	Rhodobacteraceae bacterium BSML-FTL-6w	KF148595.1	<i>Montastraea cavernosa</i> ; afflicted with black band disease	X	443	98%
Marine agar	α -Proteobacteria	<i>Ruegeria</i> sp. AH10	JX075061.1	Isolated from <i>Appendicularia</i> sp. Australia		1262	83%
Marine Agar	α -Proteobacteria	<i>Ruegeria</i> sp. AH10	JX075061.1	Isolated from <i>Appendicularia</i> sp. Australia		1309	97%
Marine Agar	α -Proteobacteria	<i>Ruegeria</i> sp. CBMAI 1067	JN615415.1	Sargassum sp.		411	100%
Marine Agar	α -Proteobacteria	<i>Ruegeria</i> sp. K2	JX075063.1	Larviculture water from <i>Panulirus ornatus</i> cultur, AIMS		396	96%
Marine Agar	α -Proteobacteria	<i>Ruegeria</i> sp. K2	JX075063.1	Larviculture water, AIMS		435	99%
Marine Agar	α -Proteobacteria	<i>Ruegeria</i> sp. S1611	FJ460027.1	Copepod		1317	85%
Marine Agar	α -Proteobacteria	Uncultured <i>Pseudovibrio</i> sp. clone 0307_BHM1_39	AB612240.1	<i>Montastraea faveolata</i> healthy SML		1320	100%
Marine Agar	α -Proteobacteria	Uncultured <i>Ruegeria</i> sp. clone CF27	JX523669.1	Beach sand		424	97%
Marine Agar	γ -Proteobacteria	Uncultured <i>Vibrio</i> sp.	AM930498.1	<i>Tripneustes gratilla</i> body wall lesion (Madagascar)	X	1326	92%
TCBS	γ -Proteobacteria	<i>Vibrio alginolyticus</i> strain 0015KARWAR	KC178716.1	Mandovi Estuary sediment		437	99%
TCBS	γ -Proteobacteria	<i>Vibrio brasiliensis</i> strain 0017KARWAR	KC178717.1	Open sea sediment, India		437	99%
TCBS	γ -Proteobacteria	<i>Vibrio harveyi</i> strain HL19	JQ948038.1	White shrimp pathogen strain	X	1414	99%
TCBS	γ -Proteobacteria	<i>Vibrio owensii</i>	JX280419.1	Marine environment		378	97%
Marine Agar	γ -Proteobacteria	<i>Vibrio</i> sp. PaD1.34	GQ406601.1	Diseased <i>Pseudopterogorgia americana</i> SML	X	403	88%
Marine Agar	γ -Proteobacteria	<i>Vibrio</i> sp. PaH3.36c3	GQ406799.1	<i>Pseudopterogorgia americana</i> ; healthy, SML		438	100%
Marine Agar	γ -Proteobacteria	<i>Vibrio</i> sp. PP05	JX075052.1	<i>Chaetognatha</i> sp. (arrow worm), Coral sea, Australia		1393	99%
TCBS	γ -Proteobacteria	<i>Vibrio</i> sp. S10	EU372920.1	WS infected <i>Pachyseris speciosa</i> , sample from disease lesion interphase	X	1398	99%

Sheridan et al., in press). Sedimentation (whether associated with terrestrial runoff or not) may also cause physical lesions on corals resulting from abrasion, sediment accumulation and increased microbial activity (Flores et al., 2012; PIANC, 2010; Weber et al., 2012). The energy depletion resulting from investment in immunity and sediment rejection mechanisms (Sheridan et al., in press) might increase coral disease susceptibility to pathogens while sediment-induced abrasion lesions may facilitate infection by opportunistic pathogens.

Terrigenous clays, as those found in large quantities in GRT sediments, can act as reservoirs of large amounts of adsorbed substances, trace metals and other contaminants (Risk and Edinger, 2011). Similarly, sediments could act as reservoirs of opportunistic coral pathogens as initially suggested by Richardson (1997) and Voss and Richardson (2006). In the present study, we isolated several potential coral pathogens from sediment samples from the outer reef slope of the GRT, including *V. harveyi* and several other *Vibrio* spp., a taxonomic group frequently involved in pathogenesis in a variety of organisms (Vezzulli et al., 2013), and recently implicated as causative agents of WS (Luna et al., 2010; Sussman et al., 2008). Though potential pathogens may also be present in both surrounding and pore water, and bacterial cultures offer only a selective snapshot of environmental bacterial communities (only 0.001–1% of the microbial diversity may be successfully cultivated using such methods; Zengler et al., 2002), these results may provide additional evidence supporting the role of sediments a reservoir of potential coral pathogens. Future studies should therefore investigate the presence of such potential pathogens in sediments affecting corals worldwide, as well as their potential involvement in coral disease development. In particular, microbiological studies comparing bacterial communities associated with sedimentation from sites affected by terrestrial runoff and non-affected sites would help to ascertain the influence of terrestrial runoff on the presence of potential coral pathogens within sediments.

Overall, the potential role of sediments as either a source of opportunistic pathogens or a substrate for their development may be particularly important for reefs where inputs of terrigenous sediments have been predicted to increase (as is the case of the GRT in SW Madagascar (Maina et al., 2013)). More importantly, both the abundance of such opportunistic pathogens within natural pathogen reservoirs (Vezzulli et al., 2013) and pathogen virulence (Kimes et al., 2012) have been shown to increase with rising seawater temperature. In view of the predicted increases in seawater temperature (Hoegh-Guldberg et al., 2007), the role of environmental stressors such as terrestrial runoff and sedimentation as agents promoting coral disease may become further exacerbated.

In conclusion, as illustrated by this study as well as earlier work (Haapkylä et al., 2011; Pollock et al., 2014; Voss and Richardson, 2006), the mounting evidence that terrigenous sediments might be implicated in coral reef decline by promoting disease development calls for increasing terrestrial runoff management in coastal management plans. Such changes in runoff management are particularly relevant as improving water quality through management efforts can reduce the impacts of climate change on reef corals (Wooldridge and Done, 2009). For example, a reduction in terrestrial runoff-associated dissolved inorganic nutrients inputs on inshore reefs of the GBR has been suggested to improve coral thermal tolerance (Wooldridge, 2009), and therefore coral adaptability to expected changes in seawater temperature (Hoegh-Guldberg et al., 2007). Improving terrestrial runoff management may consequently have beneficial impacts on coral survival and tolerance to a variety of environmental stressors.

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Appendix A. Supplementary data

Supplementary data related to this article can be found at <http://dx.doi.org/10.1016/j.marenvres.2014.08.003>.

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