INDO PAC J OCEAN LIFE
Volume 7, Number 1, June 2023
Pages: 48-53

P-ISSN: 2775-1961
E-ISSN: 2775-1953
DOI: 10.13057/oceanlife/o070105

Prevalence and thermal photo-physiological responses of Skeletal Eroding Band (SEB)-affected *Acropora muricata* from Mauritius Island

SARVESH PANKAJ MUNDIL[1], SHAKEEL YAVAN JOGEE[1], DEEPEEKA KAULLYSING[1,2], RANJEET BHAGOOLI[1,2,3,♥]
[1]Department of Biosciences and Ocean Studies, Faculty of Science & Pole of Research Excellence, Sustainable Marine Biodiversity, University of Mauritius. Réduit 80837, Republic of Mauritius. Tel.: +230-4037916, ♥email: r.bhagooli@uom.ac.mu
[2]The Biodiversity and Environment Institute. Réduit, Republic of Mauritius
[3]The Society of Biology (Mauritius). Réduit, Republic of Mauritius

Manuscript received: 14 October 2022. Revision accepted: 22 November 2022.

Abstract. *Mundil SP, Jogee SY, Kaullysing D, Bhagooli R. 2023. Prevalence and thermal photo-physiological responses of Skeletal Eroding Band (SEB)-affected* Acropora muricata *from Mauritius Island. Indo Pac J Ocean Life 7: 48-53.* The threat to coral reefs due to coral diseases has been on the rise since the late 1990s, and the effects of climate change-driven global warming and coral diseases have yet to be thoroughly investigated. This study aimed to investigate the prevalence of Skeletal Eroding Band (SEB), a coral disease resulting from the ciliate *Halofolliculina corallasia*, in *Acropora muricata*, at two sites, namely Flic en Flac and Belle Mare around Mauritius Island and the thermal photo-physiological responses, in terms of effective quantum yield at photosystem II (ΦPSII), of SEB-affected and non-affected *A. muricata*, measured with a Diving-Pulse Amplitude Modulated (D-PAM) fluorometer. Affected colonies were identified using underwater field guides, and their prevalence was estimated using a random swim method. The prevalence of SEB was highest at Belle Mare during late summer with a prevalence of 24.44 ± 1.93%. The thermal stress experiment consisting of 6 aquaria with 3 at 27°C and 3 at 32°C was set up with both SEB-affected and non-affected *A. muricata* for 19 hours. Both SEB-affected and non-affected *A. muricata* were influenced by high temperatures. However, the SEB-affected samples exhibited a higher susceptibility to 32°C treatment as the ΦPSII declined to almost zero after 6 hours of exposure. These findings suggest that the region of Belle Mare may be potentially at risk if exposed to high temperatures for extended periods and may lose up to 25% or so of its *A. muricata* cover during future thermal severe bleaching events.

Keywords: Ciliate, coral disease, Diving-PAM, ΦPSII, Mauritius, susceptibility, thermal stress

INTRODUCTION

Coral reefs are the most biologically diverse ecosystems worldwide and host over 25% of all marine organisms (Mulhall 2009), while covering <0.1% of the world`s ocean (Moberg and Folke 1999). They have high productivity and are very important as they provide various services and goods such as coastal protection with up to 97% reduction in wave energy (Ferrario et al. 2014), food, and tourist attractions (Wild et al. 2011). Coral reefs exist in various forms, and their estimated income is about USD1 billion per year (Costanza et al. 2014). Coral reefs are very important assets for Mauritius, accounting for around MUR 5 billion of the GDP (Mauritius Tourism Revenues 2020). Fringing reefs surround Mauritius with dominant species such as *Acropora muricata* (Bhagooli et al. 2019).

However, coral reefs have been in considerable decline since the 1990s and are now considered a threatened ecosystem (Coral Reefs 2015) and their loss is estimated to account for $500 billion per year by 2100 (Hoegh-Guldberg et al. 2015). In addition, they are facing several threats impacting their health and the goods and services they provide. Amongst these threats, climate change (Hoegh-Guldberg et al. 2007) is the worst, as the increasing temperatures lead to massive bleaching events that result in mass coral mortality (Hoegh-Guldberg et al. 2007). Other threats include ocean acidification (Erez et al. 2010; Veron 2011), anthropogenic activities, and a relatively novel threat, diseases (Harvell 1999; Peters 2015; Bhagooli et al. 2017). Mauritian reefs have not been spared and are expected to suffer from these threats (Bhagooli and Sheppard 2012; Bhagooli and Kaullysing 2019).

Coral disease is considered a new and emerging threat (Peters et al. 1986). The coral disease causes the deterioration of vital body functions. Ever since their first documentation, the prevalence of diseases has only been on the increase worldwide (Harvell 1999; Séré et al. 2015; Sweet and Séré 2016). Coral diseases are causing a progressive loss in coral tissue, a reduction in reproductive capacity, a decrease in the diversity of organisms associated with reefs. They also contribute to coral mortality and reduced recruitment (Harvell et al. 2007). There are now over 40 identified and described coral diseases, such as White Plague (WP), White Band (WB), Black Band (BB), Skeletal Eroding Band (SEB), and Growth Anomalies (GA), affecting over 200 reef-building species (Bruckner 2015). Studies carried out in Mauritian waters have shown the presence of GA, SEB, brown band, WP, and WB (Bhagooli et al. 2017) and white syndrome disease in *Acropora* from Saya de Malha on the Mascarene Plateau (Bhagooli et al. 2021). So far, coral diseases have only been briefly described, and most coral diseases' etiology remains unknown (Sutherland et al. 2004; Séré et al. 2015).

SEB is a eukaryotic disease caused by a ciliate protozoan, namely, *Halofolliculina corallasia*. SEB affects the coral skeleton together with the soft tissues and is consequently named Skeletal Eroding Band. SEB was first identified in the Indo-Pacific region, including Mauritius, in 1998 (Antonius and Lipscomb 2000). A similar disease occurring in the Caribbean has also been identified. The causative agent was found to be from folliculinid ciliates (*Halafollunica* sp.), and due to its close resemblance in the way it affects the coral, it has been termed Caribbean Ciliate infection (CCI) (Cróquer et al. 2006). The typical physical trait of an infected coral is the black speckled spots that remain in the skeleton caused by their black lorica that is found in the skeleton itself (Page and Willis 2007) and algae often take over the denuded skeleton.

SEB is one of the most infectious diseases with a minimum of 82 hard coral species affected (Page and Willis 2007). Being extremely virulent and infectious, infecting nearby corals in direct and indirect contact (Antonius and Lipscomb 2000; Rodríguez et al. 2008), SEB is a disease that needs further research, especially on its effect on the health of corals and their response to external stressors such as high temperatures in the wake of a climate change-driven ocean warming phenomenon. Hence, this study aimed to assess the prevalence of and variations in photo-physiology, an established proactive indicator of coral health (Hédouin and Berteaux-Lecellier 2014), in SEB-affected and non-affected *A. muricata* to determine the effects of SEB on the health of corals when subjected to thermal stress.

MATERIALS AND METHODS

SEB Prevalence assessment

SEB field observations were carried out at three stations at each of the two study sites, namely Belle Mare and Flic en Flac, around Mauritius Island, Flic en Flac is a highly touristic site with the reef occurring some 400 m or so from the shore while Belle Mare has a long stretch of the beach and the reefs occur some 1 km from the shore. Belle Mare has some hotels and small-scale agricultural practices along its coasts, while Flic en Flac is packed with hotels and bungalows along its coast. Additionally, Belle Mare is found on the windward side and Flic en Flac on the leeward side of Mauritius (Figure 1).

Corals were identified using the "Field guides to the corals of Mauritius" (Pillay et al. 2002) and "Corals of the World" (Veron 2000). Using underwater cards for disease identification (Weil et al. 2015; Bhagooli et al. 2017) any *A. muricata* colony showing presence of SEB were identified. Since this study focused on diseases that have been studied only once, prevalence of disease was preferred to occurrence (Sutherland et al. 2004) whereby;

Prevalence = (number of diseased coral colonies÷30) x100

The survey consisted of a free swim in a randomly selected direction and observing the first 30 colonies of *A. muricata* encountered. In addition, prevalence studies were carried out in triplicate in 3 distinct regions of each site, namely near the coast, lagoon, and reef, which were selected relative to the distance between the coast and the reef crest. The same observers carried out these surveys in October and November 2019 and January and February 2020.

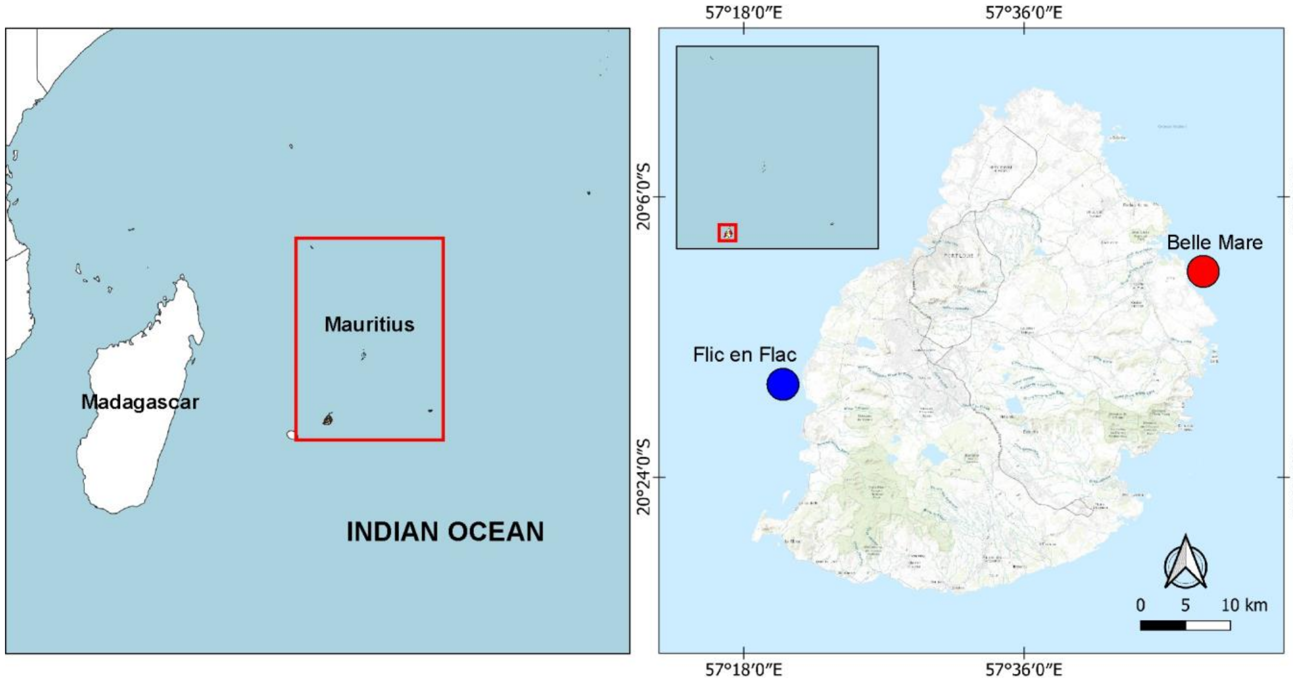

Figure 1. A map of Indian Ocean showing Mauritius (*left*), B Map of study area (*right*), Blue. Flic en Flac, Red. Belle Mare

Collection of samples and thermal experimental trials

In January 2020, 6 nubbins, each 3cm in length, from 3 different colonies of SEB-affected and non-affected corals were collected by snorkeling and identifying corals and then using pliers, nubbins were cut off and placed in ziplock bags. The collected nubbins were transported within 2 hours of the collection, under shaded conditions, in containers filled with seawater to laboratory facilities for thermal experimental trials.

The collected coral samples were put in aquaria fed with a constant air supply at room temperature for 3 hours to acclimatize. The coral samples were distributed in six aquaria with constant air supply and thermostats set for adjusting to the target temperatures. Coral fragments were put in the aquaria, and the thermostat was switched on to reach the desired temperatures of 27℃ and 32℃ in 3 aquaria for each temperature and, this marked the initial time prior to stress, i.e., marking T0. The corals were then continually observed for the 19 hrs duration of the experiment.

Chlorophyll *a* fluorescence measurement

A Diving Pulse-Amplitude-Modulated fluorometer (D-PAM, Waltz) was used to measure each coral fragment's effective quantum yield of photosystem II (ΦPSII). A saturation pulse (4000 µmol quanta $m^{-2}s^{-1}$) was applied by placing the probe flat to the surface of the coral sample, a few cm away from visible SEB lesions, while a probe was placed 3cm from the base for healthy corals while noting the physical appearance of the corals. The D-PAM sent a weak flash of light of <1 µmol quanta $m^{-2}s^{-1}$ at first and determined the fluorescence yield, F_t, and then determined the light adapted maximum fluorescence $F_{m'}$. The ΦPSII was then calculated as follows (Genty et al. 1989):

$$\Phi PSII = (F_{m'} - F_t)/ F_{m'}$$

Readings and observations were carried out at initial (t=0), after 3 hours (t=3), 6 hours (t=6) and 19 hours (t=19). The experiment was set up to minimize all fluctuations due to light variation by maintaining shaded conditions.

Statistical analyses

Normality of all variables was tested using Shapiro-Wilk test. The response in the ΦPSII of the SEB-affected and non-affected *A. muricata*, when exposed to 27°C and 32°C were compared using two-way ANOVA and Tukey HSD test. The SEB disease prevalence throughout summer was compared using Wilcoxonn-Mann-Whitney as data were non-parametric. All tests were performed in R-studio V3.6.2.

RESULTS AND DISCUSSION

Prevalence of SEB in *Acropora muricata*

SEB was observed with varying prevalence across the different stations at both studied sites. Both BM and FEF had no recorded prevalence of SEB at the reef stations. FEF had lower prevalence of SEB with a maximum prevalence of 4.44±1.92%, whereas BM had a maximum prevalence of 24.44±1.93% in February 2020 at its lagoon stations (Figure 2). An increase of 83.33% in SEB prevalence in the lagoon was observed in February 2020 when compared to October 2019 (13.33±3.33%). Overall, the lagoon stations of each studied site had the highest average SEB prevalence when compared to the near coast and reef stations.

Photo physiological response to thermal stress

Significant variations between ΦPSII of corals exposed to 27°C and 32°C ($p < 0.05$) were observed as from 3 hours, with corals exposed to 32°C showing decrease in their ΦPSII. This decrease continued until the corals exposed to 32°C were all bleached. Comparatively, the corals exposed to 27°C had no significant variations ($p > 0.05$) in their ΦPSII (Figure 3). SEB affected and non-affected *A. muricata* all maintained a relatively similar effective quantum yield (ΦPSII) at 27ºC throughout the whole experiment. Overall, there was no significant difference ($p> 0.05$) in the ΦPSII of any coral exposed to 27°C during the 19 hours of exposure (Figure 4).

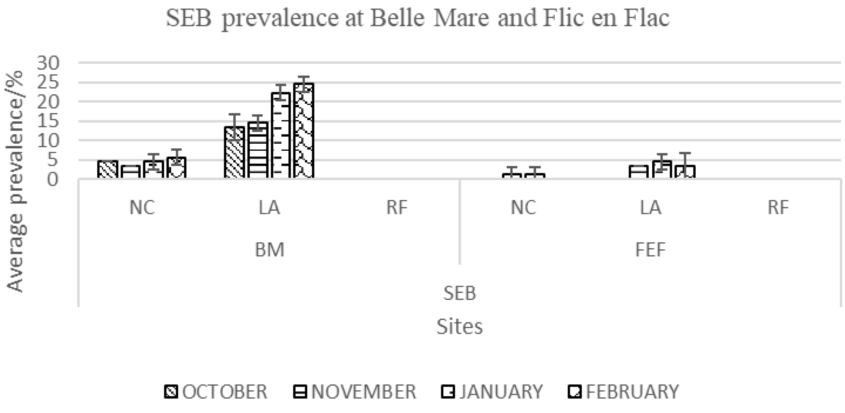

Figure 2. Mean Skeletal Eroding Band (SEB) prevalence across studied stations, Near-Coast (NC), Lagoon (LA) and Reef (RF) at the studied sites, Belle Mare (BM) and Flic en Flac (FEF). Bars represent mean±SD (n=3)

Table 1. The observed conditions of samples exposed to temperatures of 27 and 32°C at 0, 3, 6 and 19 hours

Temperature (°C)	**Condition**	**Exposure Time (hrs)**			
		0	**3**	**6**	**19**
27	Healthy	Normal	Normal	Normal	Normal
	SEB	Normal	Normal	Normal	Normal
32	Healthy	Normal	Paled	Bleached	Dead
	SEB	Normal	Bleached	Dead	Dead

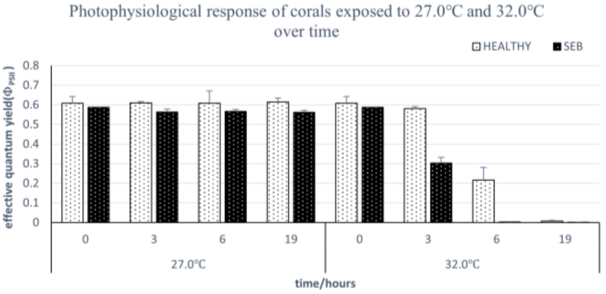

Figure 3. Effective quantum yield at PSII for healthy and SEB affected *Acropora muricata* when exposed to thermal stress. Bars represent mean±SD (n=3)

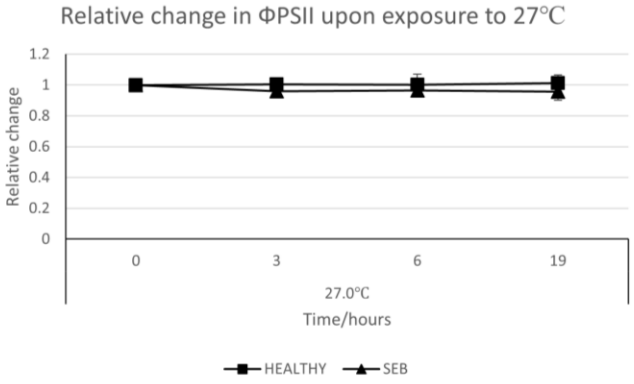

Figure 4. Relative change to initial in effective quantum yield at PSII of healthy and SEB affected *Acropora muricata* under exposure to 32°C. Bars represent mean±SD (n=3)

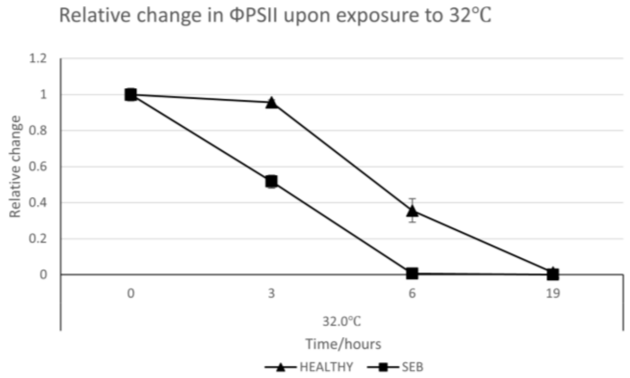

Figure 5. Relative change to initial in effective quantum yield at PSII of healthy and SEB affected *Acropora muricata* under exposure to 27°C. Bars represent mean±SD (n=3)

At 32°C, there was an overall significant decrease in the ΦPSII of both SEB-affected and non-affected corals compared to their initial, with >99% decrease after 19 hours (Figures 3 and 5). After 3 hours, a significant decrease ($p< 0.05$) in ΦPSII of the SEB-affected coral was recorded with a drop of 48.48±12.65%, while in the non-affected corals, the value dropped insignificantly ($p > 0.05$), i.e., by only 4.49±3.12%. However, the ΦPSII of SEB-affected *A. muricata* fell completely with >99% decrease after only 6 hours, while the non-affected coral decreased by 66.8%. The SEB-affected and non-affected corals following 19 hours of treatment had their ΦPSII dropping by >99% (Figures 3 and 5). An overall relative change of <0.01 was observed for both studied samples after 19 hours when exposed to 32°C (Figure 5).

Visual observations during thermal stress experiment

There was no physical change in any samples exposed to 27°C throughout the experiment, with nearly similar color even after 19 hours. SEB-affected *A. muricata* were also pale in color compared to non-affected ones. After 6 hours, partial bleaching and paling occurred in SEB-nonaffected *A. muricata*, while SEB- affected *A. muricata* was completely bleached, accompanied by complete tissue detachment from the skeleton, making the samples turn whitish and the water in the aquaria murky. After 19 hours, the SEB-nonaffected samples suffered from complete bleaching accompanied with tissue sloughing. The physical state of both samples during the experiment is summarized in Table 1.

Discussion

Previous studies found a prevalence of 8.9±1.2% of SEB at Belle Mare (Bhagooli et al. 2017) while this study found the lowest prevalence of 13.33±3.33%. This increase in SEB infection may indicate worsening conditions in the lagoon of BM. There was a nearly 2-fold significant increase ($p < 0.05$) in SEB prevalence in the lagoon of BM from early summer to mid-summer, accompanied by an increase in temperature. In this case, a rise of 3°C from October 2019 to February 2020 was noted. Furthermore, a general observation of disease outbreaks and high thermal anomalies has been noted regardless of the geographic location of these anomalies (Howells et al. 2020). This observation goes hand in hand with other observations, namely at the Great Barrier Reef, where a 2-fold increase in SEB was also observed in summer compared to winter (Harvell et al. 2007) and a similar observation of a 2-fold increase in the Arabian sea (Riegl 2002). A 10% increase in infectivity of ciliates was recorded when the temperature was raised from 26°C to 30°C in a controlled environment (Rodriguez et al. 2009), which could explain the higher increase (83.33% in this study) in situ conditions where temperatures can rise above 30°C and is coupled with the presence of additional stressors.

Higher temperatures in mid-summer, combined with degraded water quality due to nutrient inputs, caused a decrease in defense of the corals and made them more prone to disease (Page and Willis 2007). Because of the lowered defense, an increase in microbial beta diversity, thus resulting in a reduced ability of the coral to regulate its microbiome (McDevitt-Irwin et al. 2017; Thurber et al. 2020). This high number of bacteria is a suitable food source for *H. corallasia* and may cause increased SEB prevalence (Antonius and Lipscomb 2000). Another reason for this rise in prevalence in the BM lagoon could be due to an increase in nautical activities owing to the higher number of visitors due to incoming tourists' arrival from December 2019 (Mauritius Tourism Revenues 2020). The corals sustain more damage than in early summer with increased activities such as snorkeling and glass-bottom trips (personal observations). Studies have shown that high touristic zones with nautical activities and diving can create up to a 3-fold increase in the occurrence of SEB (Lamb et al. 2014). This damage sustained to the corals directly exposes them to *H. corallasia,* and it has been shown that damaged corals are easily infected by SEB (Page and Willis 2007).

Compared to other coral samples, which only underwent paling after 6 hours of exposure to 32°C, SEB-affected *A. muricata* bleached completely after only 6 hours. Even though a clear demarcation between infected and non-infected parts exists in SEB-affected *A. muricata*, the ciliate *H. corallasia* could be stressful to the non-affected part of the coral. Some internal stress, not visible and undetected via photo-physiological responses of zooxanthellae, may be present because of *H. corallasia*. This could be because the coral may be using part of its energy to defend itself against the progression of the disease or other stressors and, in doing so, cannot have a fully functional defense against ROS/RNS, produced due to higher metabolic activities both in the host and the zooxanthellae (Traylor-Knowles and Connelly 2017). The communication between the host and zooxanthellae (Baird et al. 2009) could also be affected by the disease and hence cause a cascading effect of increased ROS/RNS production by the zooxanthellae resulting in their release because the host may be unable to cope with the oxidative stress and damage (Buddemeier and Fautin 1993).

This study has shown that SEB is a very virulent disease and in the presence of favorable conditions, there can be as much as a 2-fold increased incidence within only 4 months. Causes may be a temperature rise but also a high level of activities that have the potential to damage corals, such as snorkeling. This implies that well-frequented areas might see a consequent increase in the prevalence of diseases if some of the corals are already infected with SEB. It is noteworthy that SEB-affected corals are more susceptible than non-affected ones. As demonstrated by this study, corals affected by diseases, especially SEB, are more likely to bleach during a high-temperature event. This implies that BM is severely at risk, with 24.4% of SEB infection in its lagoon. In high temperatures, BM may lose about 24% of its lagoonal *A. muricata* in 6 hours, followed by higher numbers if the high temperatures persist for longer.

Future studies need to focus on the extent of the spread of the disease at more sites around the Island, along with other environmental factors such as microbial and nutrient levels of the seawater. The spread of SEB can be stopped with appropriate measures (Lamb et al. 2014). For example, a closure period in areas with intense underwater activities can be implemented to prevent the spread of the disease, or activities should be distributed in such a way that pressure on corals is equally distributed to minimize the impacts as much as possible (Page et al. 2009). Coral diseases are a serious issue. Coupled with the ever-increasing global temperatures, it can become one of the worst problems for coral reefs and the services they provide (Coker et al. 2013). Coral diseases are now distributed worldwide and need immediate action from marine scientists and conservationists (Morais et al. 2022). To prevent further degradation of corals due to coral diseases in the foreseeable future, further studies focusing on the nomenclature, description, characterization, and most importantly methods of transmission (Thurber et al. 2020) and combined effects with other factors such as lowered pH, nutrient input and high temperatures need to be carried out to come out with management solutions for coral diseases eventually.

RERERENCES

Antonius AA, Lipscomb D. 2000. First protozoan coral-killer identified in the Indo-Pacific. Atoll Res Bull 481: 481. DOI: 10.5479/si.00775630.481.

Baird AH, Bhagooli R, Ralph PJ, Takahashi S. 2009. Coral bleaching: The role of the host. Trends Ecol Evol 24: 16-20. DOI: 10.1016/j.tree.2008.09.005.

Bhagooli R, Jogee SY, Kaullysing D, Ramah S. 2021. First report of White Syndrome Disease on branching *Acropora* at Saya de Malha, Mascarene Plateau, Western Indian Ocean. SI - Studies on the Mascarene Plateau. West. Indian Ocean J Mar 1: 189-192. DOI: 10.4314/wiojms.si2021.2.15.

Bhagooli R, Kaullysing D. 2019. Seas of Mauritius. In: World Seas: An Environmental Evaluation. Academic Press, United States. DOI: 10.1016/B978-0-08-100853-9.00016-6.

Bhagooli R, Mattan-Moorgawa S, Kaullysing D, Taleb-Hossenkhan N. 2017. A first field report of coral diseases around Mauritius Island, Western Indian Ocean. West. Indian Ocean J Mar Sci 1: 71-72.

Bhagooli R, Sheppard C. 2012. Prediction of Recurrences of Mass Coral Bleaching / Mortality and Vulnerability of Reef-Building Corals to Climate Change in Mauritian and Japanese Waters. University of Mauritius, Mauritius.

Bruckner AW. 2015. History of coral disease research. Dis Coral: 52-84. DOI: 10.1002/9781118828502.ch5.

Buddemeier RW, Fautin DG. 1993. Coral bleaching as an adaptive mechanism. Biol Sci 43: 320-326. DOI: 10.2307/1312064.

Coral Reefs. 2015. In: IUCN. https://www.iucn.org/theme/marine-and-polar/get-involved/coral-reefs. Accessed 14 October 2022.

Costanza R, de Groot R, Sutton P, van der Ploeg S, Anderson SJ, Kubiszewski I, Farber S, Turner RK. 2014. Changes in the global value of ecosystem services. Glob. Environ Change 26: 152-158. DOI: 10.1016/j.gloenvcha.2014.04.002.

Cróquer A, Bastidas C, Lipscomb D. 2006. Folliculinid ciliates: A new threat to Caribbean corals? Dis Aquat Org 69: 75-78. DOI: 10.3354/dao069075.

Erez J, Reynaud S, Silverman J, Schneider K, Allemand D. 2010. Coral calcification under ocean acidification and global change. In: Dubinsky Z, Stambler N (eds). Coral Reefs: An Ecosystem in Transition. Springer, Dordrech. DOI: 10.1007/978-94-007-0114-4_10.

Ferrario, F, Beck, MW, Storlazzi, CD, Micheli, F, Shepard, CC, Airoldi, L. 2014. The effectiveness of coral reefs for coastal hazard risk reduction and adaptation. Nat Commun 5: 3794. DOI: 10.1038/ncomms4794.

Genty B, Briantais JM, Baker NR. 1989. The relationship between the quantum yield of photosynthetic electron transport and quenching of chlorophyll fluorescence. Biochimica and Biophysica Acta 990: 87-92. DOI: 10.1016/S0304-4165(89)80016-9.

Harvell CD. 1999. Emerging marine diseases--Climate links and anthropogenic factors. Science 285: 1505-1510. DOI: 10.1126/science.285.5433.1505.

Harvell D, Jordán-Dahlgren E, Merkel S, Rosenberg E, Raymundo L, Smith G, Weil E, Willis B. 2007. Coral disease, environmental drivers, and the balance between coral and microbial associates. Oceanography 20: 172-195. DOI: 10.5670/oceanog.2007.91.

Hédouin L, Berteaux-Lecellier V. 2014. Traditional vs new approaches for assessing coral health: A global overview and the paradigm of French polynesia. J Mar Sci Technol 22:25-35. DOI: 10.6119/JMST-013-0813-2.

Hoegh-Guldberg O, Hoegh-Guldberg H, Sumaila R. 2015. Reviving the Ocean Economy: The Case for Action - 2015. WWF - World Wide Fund For Nature (Formerly World Wildlife Fund), Gland, Switzerland.

Hoegh-Guldberg O, Mumby PJ, Hooten AJ, Steneck RS, Greenfield P, Gomez E, Harvell CD, Sale PF, Edwards AJ, Caldeira K, Knowlton N, Eakin CM, Iglesias-Prieto R, Muthiga N, Bradbury RH, Dubi A, Hatziolos ME. 2007. Coral Reefs under rapid climate change and ocean acidification. Science 318: 1737-1742. DOI: 10.1126/science.1152509.

Howells EJ, Vaughan GO, Work TM, Burt JA, Abrego D. 2020. Annual outbreaks of coral disease coincide with extreme seasonal warming. Coral Reefs 39: 771-781. DOI: 10.1007/s00338-020-01946-2.

Lamb JB, True JD, Piromvaragorn S, Willis BL. 2014. Scuba diving damage and intensity of tourist activities increases coral disease prevalence. Biol Conserv 178: 88-96. DOI: 10.1016/j.biocon.2014.06.027.

Mauritius Tourism Revenues. 2020. 1995-2019 Data, 2020-2022 Forecast, Calendar. In: tradingeconomics.com. https://tradingeconomics.com/mauritius/tourism-revenues. Accessed 5 April 2020.

McDevitt-Irwin JM, Baum JK, Garren M, Thurber RLV. 2017. Responses of coral-associated bacterial communities to local and global stressors. Front Mar Sci 4 (1): 1-16. DOI: 10.3389/fmars.2017.00262.

Moberg F, Folke C. 1999. Ecological goods and services of coral reef ecosystems. Ecol Econ 29: 215-233. DOI: 10.1016/s0921-8009(99)00009-9.

Morais J, Cardoso APLR, Santos BA. 2022. A global synthesis of the current knowledge on the taxonomic and geographic distribution of major coral diseases. Environ Adv 8: 100231. DOI: 10.1016/j.envadv.2022.100231.

Mulhall M. 2009. Saving the rainforest of the sea: An analysis of international efforts toconserve coral reefs. Duke Environ Law Policy Forum 19: 321-352.

Page C, Baker D, Harvell C, Golbuu Y, Raymundo L, Neale S, Rosell K, Rypien K, Andras J, Willis B. 2009. Influence of marine reserves on coral disease prevalence. Dis Aquat Org 87: 135-150. DOI: 10.3354/dao02112.

Page CA, Willis BL. 2007. Epidemiology of skeletal eroding band on the Great Barrier Reef and the role of injury in the initiation of this widespread coral disease. Coral Reefs 27: 257-272. DOI: 10.1007/s00338-007-0317-8.

Peters EC, Halas JC, McCarty HB. 1986. Calicoblastic neoplasms in *Acropora palmata*, with a review of reports on anomalies of growth and form in corals. J Natl Cancer Ins 76: 895-912. DOI: 10.1093/jnci/76.5.895.

Peters EC. 2015. Diseases of coral reef organisms. Coral Reefs in the Anthropocene 147-178. DOI: 10.1007/978-94-017-7249-5_8.

Pillay R, Terashima H, Venkatasami A, Uchida H. 2002. Field guide to corals of Mauritius. Albion Fisheries Research Centre, Ministry of Fisheries, Albion, Petite Riviere, Mauritius.

Riegl B. 2002. Effects of the 1996 and 1998 positive sea-surface temperature anomalies on corals, coral diseases and fish in the Arabian Gulf (Dubai, UAE). Mar Biol 140: 29-40. DOI: 10.1007/s002270100676.

Rodríguez S, Cróquer A, Guzmán HM, Bastidas C. 2008. A mechanism of transmission and factors affecting coral susceptibility to *Halofolliculina* sp. infection. Coral Reefs 28: 67-77. DOI: 10.1007/s00338-008-0419-y.

Séré M, Chabanet P, Turquet J, Quod J, Schleyer M. 2015. Identification and prevalence of coral diseases on three Western Indian Ocean coral reefs. Dis Aquat Org 114: 249-261. DOI: 10.3354/dao02865.

Sutherland K, Porter J, Torres C. 2004. Disease and immunity in Caribbean and Indo-Pacific zooxanthellate corals. Mar Ecol Prog Ser 266: 273-302. DOI: 10.3354/meps266273.

Sweet MJ, Séré MG. 2016. Ciliate communities consistently associated with coral diseases. J Sea Res 113: 119-131. DOI: 10.1016/j.seares.2015.06.008.

Thurber RV, Mydlarz LD, Brandt M, Harvell D , Weil E, Raymundo L, Willis BL, Langevin S, Tracy AM, Littman R, Kemp KM, Dawkins P, Prager KC, Garren M, Lamb J. 2020. Deciphering coral disease dynamics: Integrating host, microbiome, and the changing environment. Front Ecol Evol 8: 1-18. DOI: 10.3389/fevo.2020.575927.

Traylor-Knowles N, Connelly MT. 2017. What is currently known about the effects of climate change on the coral immune response. Curr Clim Change Rep 3: 252-260. DOI: 10.1007/s40641-017-0077-7.

Veron J. 2000. Corals of the World, 1st ed. Australian Institute of Marine Science and CRR Ald Pty Ltd, Australia.

Veron J. 2011. Ocean acidification and coral reefs: An emerging big picture. Diversity 3: 262-274. DOI: 10.3390/d3020262.

Weil E, Raymundo LJ, Beeden R, Page CA. 2015. Underwater Cards for Assessing Coral Health on Indo-Pacific Reefs. Coral Reef Targeted Research and Capacity Building for Management Program. Currie Communications, Melbourne.

Wild C, Hoegh-Guldberg O, Naumann MS, Colombo-Pallotta MF, Ateweberhan M, Fitt WK, Iglesias-Prieto R, Palmer C, Bythell JC, Ortiz J-C, Loya Y, van Woesik R. 2011. Climate change impedes scleractinian corals as primary reef ecosystem engineers. Mar Freshw Res 62: 205. DOI: 10.1071/mf10254.