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Coral diseases and associated photosynthetic responses around Rodrigues Island, Western Indian Ocean

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Abstract. *Jogee SY, Anasamy M, Jeetun S, Ricot M, Ramkalam M, Korimbocus A, Kaullysing D, Nascimento B, Casareto BE, Suzuki Y, Wijayanti DP, Bhagooli R. 2025. Coral diseases and associated photosynthetic responses around Rodrigues Island, Western Indian Ocean. Indo Pac J Ocean Life 9: 100-110.* The coral reefs of Rodrigues Island are among the most biodiverse ecosystems in the Western Indian Ocean (WIO) but are increasingly threatened by coral diseases. While disease prevalence has been documented, its physiological impacts remain poorly understood. This study assessed the photo-physiological responses of four reef-building coral species (*Montipora* sp., *Platygyra* sp., *Acropora* sp., *Favia* sp.) affected by Black Band Disease (BBD), Skeletal Eroding Band (SEB), White Band (WB), and White Syndrome (WS). Coral fragments containing both diseased and visually healthy tissues were collected from four sites and analyzed in situ using a Junior-Pulse-Amplitude-Modulated (PAM) fluorometer. Parameters measured included maximum quantum yield (Fv/Fm), maximum relative electron transport rate (rETRmax), maximum non-photochemical quenching (NPQmax), initial slope before saturation (α), post-saturation slope (β), and minimum saturating irradiance (Ik), derived from rapid light curves. Diseased tissues generally exhibited reduced photosynthetic performance, with BBD in *Montipora* sp. and SEB in *Favia* sp. showing the most pronounced declines in Fv/Fm and α, along with altered β and NPQmax values. In contrast, WB and WS had limited or non-significant effects on the measured parameters. These results highlight disease and species-specific vulnerabilities, indicating that some coral-algal symbioses are more susceptible to functional impairment. In the context of climate change, disease-driven stress could accelerate reef degradation, especially in isolated islands like Rodrigues. Integrating photo-physiological monitoring into routine reef health surveys can enhance early detection of functional decline, guide species-targeted restoration, and strengthen adaptive management strategies to safeguard the resilience of Rodrigues' reefs and other WIO coral ecosystems.

Keywords: Chlorophyll fluorescence, coral disease, PAM, photo-physiology, Rodrigues Island, species-specific vulnerability

Abbreviations: α: Alpha (initial slope before the onset of saturation), β: Beta (slope after the onset of saturation), ANOVA: Analysis of Variance, BBD: Black Band Disease, Fv/Fm: F_0: Minimum or Baseline Fluoresence, F_m: Maximum Fluoresence, Maximum Quantum Yield, I_k: Minimum saturating irradiance, NPQ_{max}: Maximum Non-Photochemical Quenching, PAM: Pulse Amplitude Modulation, PAR: Photosynthetically Active Radiation, PWPS: Porites White Patch Syndrome, RLC: Rapid Light Curve, $rETR_{max}$: Relative Maximum Electron Transport Rate, SEB: Skeletal Eroding Band, SEMPA: South East Marine Protected Area. Small Island Developing States: SIDS, WB: White Band, WIO: Western Indian Ocean, WS: White Syndrome, Φ_{PSII}: Effective Quantum Yield

INTRODUCTION

Coral reefs are among the most biologically diverse and productive ecosystems, providing critical ecological goods and services, including habitats for marine biodiversity, shoreline protection, and support for fisheries and tourism, particularly in Small Island Developing States (SIDS) (Hughes et al. 2017; McClanahan et al. 2023). However, coral reefs face numerous global and local stressors, including climate change, ocean acidification, coastal development, overfishing, and coral diseases, which are emerging as significant threats to reef health worldwide (Howells et al. 2020; Good and Bahr 2021; Vega-Thurber et al. 2020). Coral diseases represent a prominent and lethal or sub-lethal threat to coral reef ecosystems globally, contributing to rapid and large-scale coral mortality, biodiversity loss, and ecosystem degradation, with outbreaks documented in various reef systems (Neely and Lewis 2021; Burke et al. 2023). Over the past decade, coral disease incidence has risen sharply, driving substantial declines in reef-building coral populations (Precht et al.

2016; Costa et al. 2021). Understanding the physiological impacts of coral diseases is crucial to assessing their effects on coral resilience and reef stability. Rodrigues Island, situated 600 km east of Mauritius, recently reported multiple coral diseases (Jogee et al. 2023b), yet no research has examined how these diseases affect coral physiology. Rodrigues Island harbors diverse coral communities within its extensive lagoon and fringing reef system but remains highly vulnerable to virulent coral diseases, climate change, and local anthropogenic stressors. While coral bleaching events have been reported in the region (Hardman et al. 2004, 2007), the physiological impacts of coral diseases remain completely unexplored.

Studies on coral diseases primarily focus on field-based identification, prevalence, and distribution patterns (Work and Meteyer 2014). However, fewer investigations have explored the photo-physiological impacts of these factors, which influence coral health and energy acquisition (Lawrence et al. 2015; Grottoli et al. 2018). Coral-algal symbiosis, particularly through the photosynthetic zooxanthellae, is essential for coral survival, as it provides energy through photosynthesis. Disruptions in this relationship, such as those caused by coral diseases, can significantly impair coral function (Higuchi et al. 2015; Suzuki et al. 2015; McClanahan et al. 2023). Pulse-amplitude modulated (PAM) fluorometry is a non-invasive technique widely used to assess photosynthetic performance in corals, including their responses to disease-related stress (Ralph et al. 2015; Bhagooli et al. 2021a).

Previous studies using PAM fluorometry have reported compromised photo-physiology in diseased corals, including reduced maximum quantum yield (Fv/Fm) and lower relative electron transport rates (rETRmax) near disease lesions (Mattan-Moorgawa et al. 2017; Jogee et al. 2023a). For instance, Roff et al. (2008) examined corals affected by White Syndrome, Brown Band, and Skeletal Eroding Band on the Great Barrier Reef, reporting spatial variations in photosynthetic impairment. Burns et al. (2013) documented reduced Fv/Fm in corals with growth anomalies, while Mattan-Moorgawa et al. (2017) found decreased effective quantum yield (ΦPSII) and ETRmax in White Band disease on *Acropora muricata* (Linnaeus, 1758). Similar findings were observed for Black Band, White Band, and Skeletal Eroding Band diseases in Mauritius (Jogee et al. 2023a).

Studies on coral diseases in the Indian Ocean have shown evidence of coral pathologies throughout the region, for example, in the Red Sea (Mohamed and Sweet 2019; Aeby et al. 2021), in the Persian Gulf (Hazraty-Kari et al. 2021; Bharath et al. 2023), in the Atolls of Lakshadweep Islands (Das et al. 2023), in the Republic of Maldives (Bises et al. 2023), and in the Mascarene Islands (Jogee et al. 2023b, 2024). Despite extensive research on coral diseases in the Indo-Pacific, studies in the Western Indian Ocean (WIO)—a region of exceptional coral diversity and endemism—remain limited (McClanahan et al. 2023). Existing WIO research has largely focused on mapping disease prevalence and distribution (Séré et al. 2012, 2015a,b, 2016; Bhagooli et al. 2017, 2021b; Jogee et al. 2023b, 2024), with minimal attention to their physiological impacts. To date, only two studies from Mauritius have assessed photo-physiological responses of diseased corals (Mattan-Moorgawa et al. 2017; Jogee et al. 2023a).

This study addresses this critical knowledge gap by investigating how four prevalent diseases—Black Band Disease (BBD), Skeletal Eroding Band (SEB), White Band (WB), and White Syndrome (WS) affect the photo-physiology of key reef-building corals around Rodrigues Island. Unlike most WIO studies, which focus primarily on disease prevalence and distribution, our approach applies PAM fluorometry, which can proactively detect sub-lethal functional impairment from disease, which often precedes visible tissue loss. PAM fluorometry was used to quantify changes in Fv/Fm, rETRmax, NPQmax, Alpha, Beta, and Ik between healthy and diseased coral tissues. By revealing species-specific physiological vulnerabilities to disease-related stress, this research advances the understanding of coral disease ecology in the WIO; it provides a foundation for adaptive, targeted management strategies to enhance the resilience of Rodrigues' reefs.

MATERIALS AND METHODS

Coral collection sites

This study was conducted around Rodrigues Island (19.7245° S, 63.4272° E), located approximately 600 km northeast of Mauritius Island in the southwestern Indian Ocean. The volcanic island boasts one of the largest lagoons in the WIO region (Chapman 2000) and is surrounded by approximately 90 km of fringing reefs (Soondur et al. 2023). The island has one of the largest marine protected areas in the Indian Ocean, covering an area of around 43 km², known as the South East Marine Protected Area (SEMPA), along with several marine reserves, including Grand Bassin, Passe Demie, Anse aux Anglais, and Rivière Banane. Diseased coral samples were collected through scuba and snorkeling at four sites around the island, namely, Plateau Bénitier, Var Brulée, Pâté Reynieux, and Hermitage Island (Figure 1). These sites were selected following the previous reports of coral diseases observed there in 2020 by Jogee et al. (2023b). With the exception of Plateau Bénitier, located on the north coast of the island at depths of 5-9 m, all the other sites are found in the south, with depths ranging from 1 to 1.5 m.

Coral disease identification and characterization

Coral diseases were identified in the field through visual surveys and using the "Underwater cards for assessing coral health" by Beeden et al. (2008). The gross morphological features of the coral disease-associated lesions were recorded and classified according to the framework proposed by Work and Aeby (2006). Features included the lesion size, lesion color, shape of the lesion, distribution of the lesion on the colony, the margins of the lesion, edges of the lesion, relief of the lesion, and texture of the lesions.

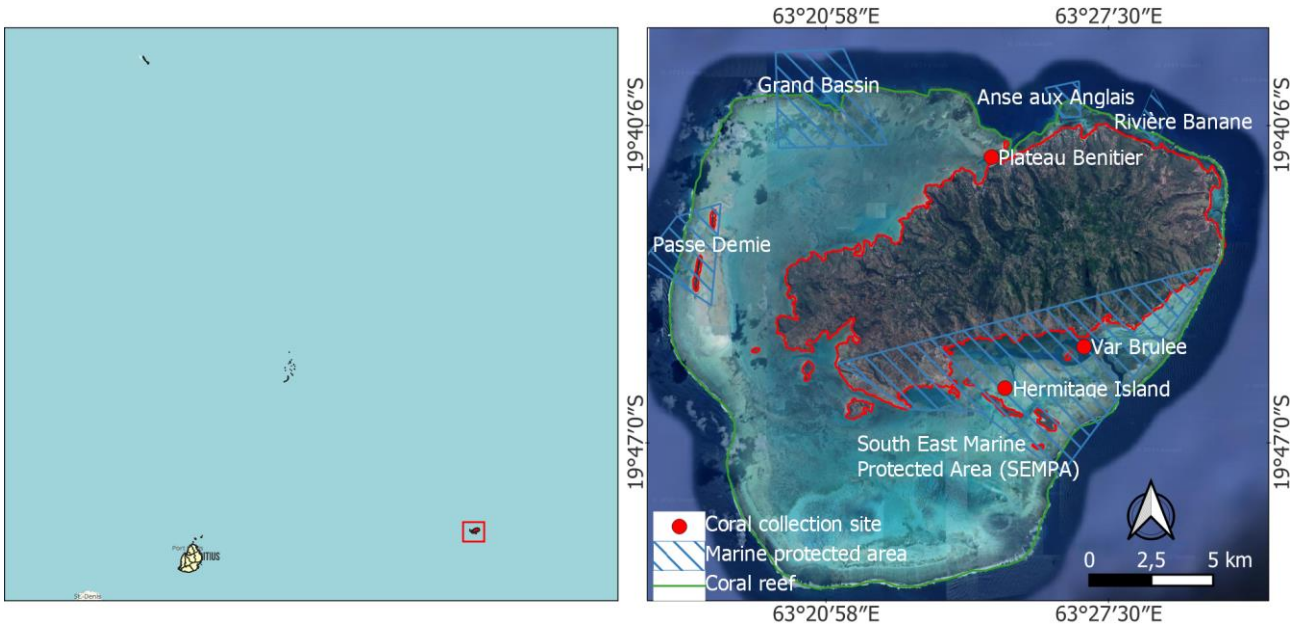

Figure 1. Map of the coral collection sites, marine protected areas, and coral reefs in Rodrigues Island, Mauritius

Photo-physiological assessment

A Junior-Pulse-Amplitude-Modulated (PAM) fluorometer was used to assess the chlorophyll *a* fluorescence of the diseased coral samples collected at various sites around the island. Coral samples were collected using a hammer and chisel or a bone cutter, and the samples were collected in such a way that in included the healthy coral part, the disease lesion part, and the dead and algae-covered part. Coral samples were collected under the Rodrigues Regional Assembly (RRA) Commission for Agriculture, Environment, Forestry, Fisheries, and Marine Parks permit. Measurements were taken at the healthy-looking coral part far (>2-5 cm) from the disease lesion and near (<1 cm) to the disease lesion.

Prior to any PAM measurement using the WinControl Software (v3.34), the coral fragments were dark-adapted for 15 min. The measuring-light intensity was set to between 0.5-1.0 µmol photons m^{-2} s^{-1}, which yielded a stable F_0 without pre-illumination; saturation pulses (~0.8 s) were applied at an intensity of between 3,000-4,000 µmol photons m^{-2} s^{-1} to ensure Fm saturation without overexposure. Rapid Light Curves (RLCs) comprised stepwise actinic PAR increments (0, 66, 90, 125, 190, 285, 420, 625, 845µmol photons m^{-2} s^{-1}; 10-15 s per step), with a saturation pulse at the end of each step. The fluorescence values were zeroed using the F-Offset function, and the gain was brought to 1 prior to taking measurements on a new coral fragment.

The photo-physiological parameters recorded were the Maximum Quantum Yield (Fv/Fm), the maximum relative electron transport rate (rETRmax), and the maximum Non-Photochemical Quenching (NPQ_{max}). A double exponential decay function (Platt et al. 1980) was used to fit curves to the Rapid Light Curves (RLCs) and quantitatively compare the parameters such as α (initial slope of the RLC before the onset of saturation), β (slope after the onset of saturation), I_k (minimum saturating irradiance), and rETRmax (Bhagooli et al. 2021a) between the healthy and diseased coral parts using SigmaPlot software (v.12.0).

Statistical analyses

All statistical analyses were performed at the colony level to compare tissue states (healthy vs near-lesion) within each disease-host combination. Assumptions of normality were assessed using the Shapiro-Wilk test on residuals, and homogeneity of variances was tested using Levene's test. When assumptions of normality or homoscedasticity were violated, data were transformed) before re-testing assumptions. Arcsine square-root transformation was applied since the PAM data are proportion-like values bound between 0 and 1. If assumptions were met, one-way Analysis of Variance (ANOVA) was applied to detect significant differences (α = 0.05) in Fv/Fm, rETRmax, NPQmax, α, β, and Ik among tissue states for each disease-host pairing. All statistical analyses were carried out on R Studio Version 2021.09.0.

RESULTS AND DISCUSSION

Around Rodrigues Island, a total of four coral diseases were observed, identified, and collected for the measurement of their chlorophyll *a* fluorescence. Black Band Disease (BBD) was observed on plating *Montipora* sp. at Var Brulée, located in the south of Rodrigues Island in the SEMPA. BBD in this coral genus was characterized by a thin, narrow, and diffuse black color with undulating annular margins and indistinct edges (Figure 2.A). The lesion was observed on the peripheral part of the coral colony, characterized by tissue loss. The shape of the lesion was irregular, and the edges were indistinct. This disease moderately affected the coral colony, with a range of 25 to 50%. The lesion appeared to be acute due to the presence of a clear, white, denuded coral skeleton, characteristic of recent mortality.

Both the polyp and coenosarc of the coral appeared to be affected by this disease. Although not significant (One-way ANOVA, $p = 0.100$, $F_{1,n} = 3.279$), this might be evident from the much-reduced Fv/Fm values recorded closer to the black band lesion (0.515 ± 0.118) as compared to the healthy-looking coral part (0.620 ± 0.0778) (Figure 3.A). Other observations from key photo-physiological parameters which were not significantly different between the healthy coral part and near the lesion, such as the reduced rETRmax (One-way ANOVA, $p = 0.116$, $F_{1,n} = 3.215$), and α (One-way ANOVA, $p = 0.142$, $F_{1,n} = 2.745$), and the increased NPQmax (One-way ANOVA, $p = 0.794$, $F_{1,n} = 0.072$), and β (One-way ANOVA, $p = 0.071$, $F_{1,n} = 0.307$), might suggest a photo-physiologically compromised health condition near the Black Band lesion on the plating *Montipora* sp. coral, in contrast to the healthy-looking coral parts.

The same disease was also observed to occur on sub-massive *Platygyra* sp. colonies at Plateau Bénitier, located north of Rodrigues Island. The disease in this coral genus was characterized by a large, thick, and black-colored diffuse band that separated the healthy-looking coral tissues from the dead and algae-covered coral skeleton (Figure 2.B). Tissue loss was evident in the region behind the lesion. The lesion was observed on the peripheral part of the colony and had undulating annular margins with distinct edges. Both the polyp and the coenosarc of the coral were observed to be affected as a result of this poly-microbial disease. The shape of the lesion was irregular, and the edges were indistinct. The lesion appeared to be sub-acute due to the absence of a clear, white, denuded coral skeleton, characteristic of recent mortality. A region of algae-covered, dead, and unaffected coral skeleton preceded it. The extent of the disease in this coral genus was severe, with more than 50% of the colony affected. This can be observed from the dissimilarity in the Fv/Fm values between the healthy part of the coral and that near the disease lesion. A significant (One-way ANOVA, $p = 0.047$, $F_{1,n} = 6.904$) drop in the Fv/Fm values (Figure 3.A), which is indicative of reduced photosynthetic capacity, was recorded near the disease lesion (0.469 ± 0.116) compared to the healthy part of the coral colony (0.622 ± 0.0263). Another indication was also evident on the compromised photosynthetic competence of the near lesion coral part as opposed to the healthy coral part from the reduced $rETR_{max}$ values (Figure 2.B), however, all the other photo-physiological parameters including rETRmax (One-way ANOVA, $p = 0.128$, $F_{1,n} = 3.328$), NPQmax (One-way ANOVA, $p = 0.401$, $F_{1,n} = 3.341$), α (One-way ANOVA, $p = 0.115$, $F_{1,n} = 0.65$), β (One-way ANOVA, $p = 0.427$, $F_{1,n} = 3.721$) and Ik (One-way ANOVA, $p = 0.545$, $F_{1,n} = 5.978$) were found to be non-significantly (Tables 1 and 2) different between the healthy coral part and the coral part near the disease lesion.

Skeletal Eroding Band disease was also observed on a *Favia* sp. colony at the same site, Plateau Bénitier, in the north of Rodrigues Island. The disease was characterized by a thin, dark grey line with undulating and indistinct margins, located at the epicenter of the coral colony and separating the healthy-looking tissues from the dead, algae-covered, but slightly eroded coral skeleton (Figure 2.C). Tissue loss was evident from the zone preceding the band. The extent of this disease was moderate, as slightly more than 25%, but less than 50%, of the colony was affected. The mortality associated with this disease was also acute, as evidenced by the white denuded coral skeleton, which is a sign of recent mortality. The shape of the large lesion was irregular, and the polyp, coenosarc, and skeleton appeared to be impacted directly or indirectly by the disease. The impact on all coral features can explain the significantly reduced Fv/Fm values (One-way ANOVA, $p < 0.001$, $F_{1,n} = 205.894$) recorded near the coral lesion (0.482 ± 0.0221) compared to the healthy-looking coral parts (0.652 ± 0.0452). Significantly reduced alpha values (One-way ANOVA, $p = 0.01$, $F_{1,n} = 21.654$) near the disease lesion (0.206 ± 0.0123) compared to the healthy coral part (0.253 ± 0.0126) is an obvious indicator of reduced or compromised photosynthetic performance closer to the disease site (Tables 1 and 2). NPQmax was significantly higher (One-way ANOVA, $p = 0.028$, $F_{1,n} = 16.208$) in the healthy part (0.647 ± 0.084) compared to near the lesion (0.316 ± 0.101). Significantly higher beta values (One-way ANOVA, $p = 0.003$, $F_{1,n} = 40.197$) were also recorded near the disease lesion (0.425 ± 0.001) compared to the healthy parts of the coral 0.0130 ± 0.001. No significant differences were observed in rETRmax (One-way ANOVA, $p = 0.199$, $F_{1,n} = 2.359$) and Ik values (One-way ANOVA, $p = 0.338$, $F_{1,n} = 0.592$).

At the same site, another disease, known as White Band disease, was observed on a branching *Acropora* sp. colony. This disease was characterized by a completely diffuse, white, and denuded band of coral skeleton, which separated the healthy-looking coral from the dead, algae-covered coral skeleton (Figure 2.D). The lesion was wide and located at the basal part of the colony. The lesion margins were smooth, and the edges were distinct with a linear shape. The extent of the disease was mild, as less than 25% of the colony was affected; however, the disease was acute, particularly in the recently exposed denuded coral skeleton. The skeleton at the lesion did not appear to be affected. However, the white color of the skeleton might indicate the loss of the polyp and the coenosarc. However, the data on the photo-physiology of this disease, recorded at Rodrigues Island, do not indicate any statistically significant alteration (Tables 1 and 2) in the photosynthetic capacity of the coral near the disease lesion compared to the healthy coral parts. Fv/Fm was nearly identical between healthy (0.652 ± 0.045) and near-lesion tissue (0.653 ± 0.052) (One-way ANOVA, $p = 0.980$, $F_{1,n} = 0.00$). rETRmax increased slightly from (37.56 ± 24.75) to (45.36 ± 7.52) ($p = 0.478$, $F_{1,n} = 0.55$). NPQmax decreased from (0.582 ± 0.148) to (0.516 ± 0.069). One-way ANOVA, ($p = 0.422$, $F_{1,n} = 0.73$). α increased from (0.123 ± 0.070) to (0.155 ± 0.037) (One-way ANOVA, $p = 0.486$, $F_{1,n} = 0.71$). β declined from (0.0126 ± 0.0072) to (0.0116 ± 0.0053) (One-way ANOVA, $p = 0.896$, $F_{1,n} = 0.02$). Ik decreased slightly from (317.17 ± 236.97) to (314.39 ± 135.81) (One-way ANOVA, $p = 0.993$, $F_{1,n} = 0.00$).

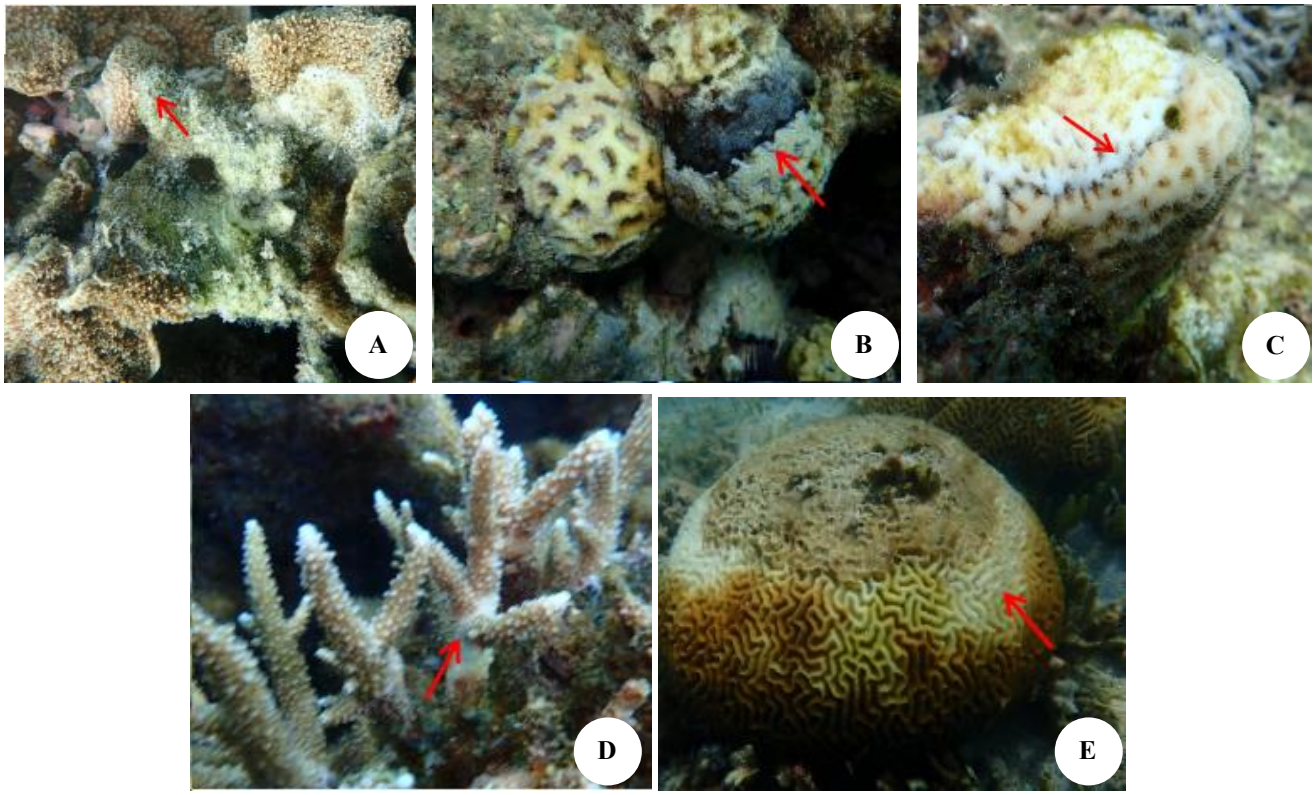

Figure 2. A. Black Band Disease on *Montipora* sp., B. Black Band Disease on *Platygyra* sp., C. Skeletal Eroding Band on *Favia* sp., D. White Band on *Acropora* sp., E. White Syndrome on *Platygyra* sp. (Red arrow shows the location of the disease)

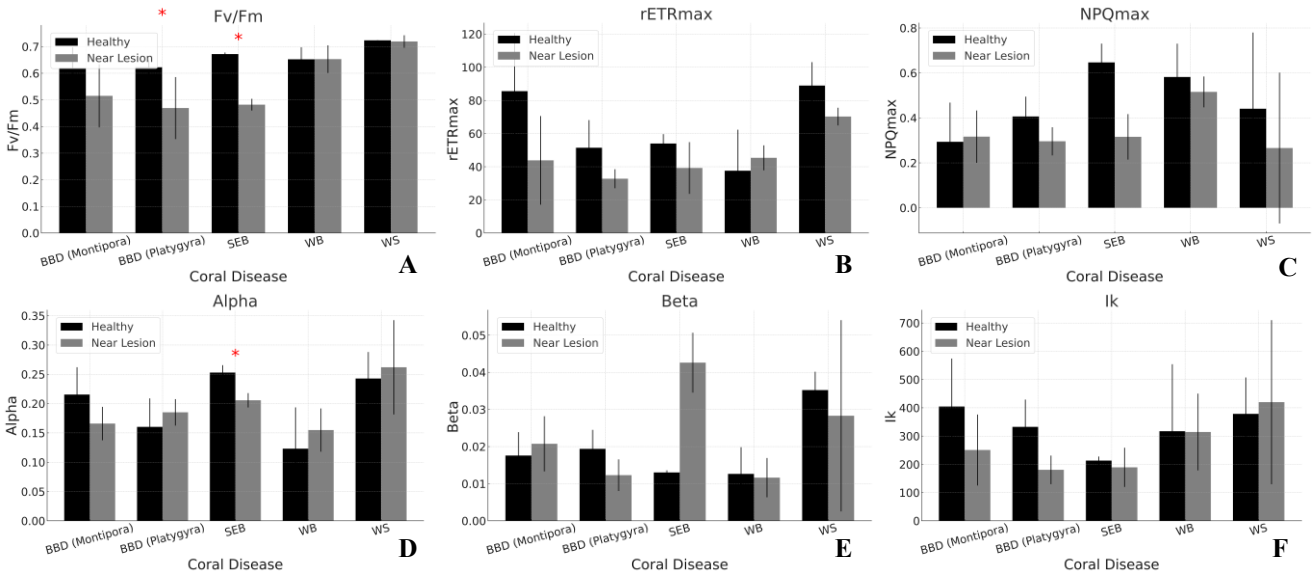

Figure 3. Variation in six photo-physiological parameters between healthy and near-lesion tissues of corals affected by four disease types around Rodrigues Island. A. Maximum quantum yield (Fv/Fm), B. Maximum relative electron transport rate (rETRmax), C. Maximum non-photochemical quenching (NPQmax), D. Initial slope before saturation (α), E. Slope after saturation (β), and F. Minimum saturating irradiance (Ik). Bars represent means ± standard deviation. Red asterisks indicate significant differences between tissue states within a disease-host combination (one-way ANOVA, * - $p < 0.05$). Disease abbreviations: BBD: Black Band Disease, SEB: Skeletal Eroding Band, WB: White Band, WS: White Syndrome

Table 1. Summary of photo-physiological responses of reef-building corals to four coral disease types around Rodrigues Island. Arrows indicate the direction of change in the near-lesion tissue relative to healthy tissue (↓ = decrease, ↑ = increase, - = no directional trend). "ns" denotes non-significant differences ($p > 0.05$). Significant changes ($p < 0.05$) from one-way ANOVA are listed in the final column

Disease type	Host species	Fv/Fm change	rETRmax change	NPQmax change	α change	β change	Ik change	Significant differences ($p < 0.05$)
BBD	*Montipora* sp.	↓ (ns)	↓ (ns)	- (ns)	↓ (ns)	- (ns)	- (ns)	None
BBD	*Platygyra* sp.	↓	↓ (ns)	↑ (ns)	- (ns)	- (ns)	↓ ($p = 0.058$)	Fv/Fm ↓
SEB	*Favia* sp.	↓	- (ns)	↑ ($p = 0.0275$)	↓ ($p < 0.01$)	↑ ($p < 0.01$)	- (ns)	Fv/Fm ↓, α ↓, β ↑, NPQmax ↑
WB	*Acropora* sp.	- (ns)	- (ns)	- (ns)	- (ns)	- (ns)	- (ns)	None
WS	*Platygyra* sp.	↓ (ns)	↓ ($p = 0.0626$)	- (ns)	- (ns)	- (ns)	- (ns)	None

Table 2. One-way ANOVA result of differences in Fv/Fm, $rETR_{max}$, NPQ_{max}, Alpha, Beta, and I_k between the healthy part of the coral and near the lesion of Black Band Disease on *Montipora* sp., Black Band Disease on *Platygyra* sp., Skeletal Eroding Band, White Band, and White Syndrome (* - $p < 0.1$, ** - $p < 0.01$, *** - $p < 0.001$)

	Photo-physiological parameters	df	Sum of Squares	F value	*p* value
Black Band Disease on *Montipora* sp.	Fv/Fm	1	0.03287	3.279	0.100
	$rETR_{max}$	1	3479	3.215	0.116
	NPQ_{max}	1	0.00158	0.072	0.794
	α	1	0.004904	2.745	0.142
	β	1	$1.344x10^{-5}$	0.307	0.609
	I_k	1	47311	1.886	0.212
Black Band Disease on *Platygyra* sp.	Fv/Fm	1	0.04022	6.904	0.0467 *
	$rETR_{max}$	1	597.9	3.328	0.128
	NPQ_{max}	1	0.02088	3.341	0.127
	α	1	0.001059	0.650	0.457
	β	1	$8.581x10^{-5}$	3.721	0.112
	I_k	1	39593	5.978	0.0583
Skeletal Eroding Band	Fv/Fm	1	0.05415	205.9	0.0137 *
	$rETR_{max}$	1	324.9	2.359	0.199
	NPQ_{max}	1	0.13134	16.21	0.0275*
	α	1	0.003351	21.65	0.0464 *
	β	1	0.0013113	40.200	0.0317 *
	I_k	1	848	0.338	0.592
White Band	Fv/Fm	1	0.000001	0.00	0.987
	$rETR_{max}$	1	91.2	0.273	0.629
	NPQ_{max}	1	0.00658	0.492	0.522
	α	1	0.001511	0.478	0.527
	β	1	0.0000015	0.038	0.856
	I_k	1	12	0.00	0.987
White Syndrome	Fv/Fm	1	0.0000241	0.058	0.821
	$rETR_{max}$	1	465.4	6.558	0.0626
	NPQ_{max}	1	0.0366	0.323	0.610
	α	1	0.000493	0.092	0.777
	β	1	0.0000635	0.126	0.740
	I_k	1	2068	0.034	0.866

White Syndromes were observed at two sites in the south of Rodrigues Island in the SEMPA, Hermitage Island, and Pâté Reynieux. This disease was observed primarily on massive *Platygyra* sp. colonies. This disease was characterized by large, diffuse, and oblong areas of tissue loss with focal to multifocal and coalescing distribution (Figure 2.E). Most of the observed lesions occurred at the colony-wide level and were not restricted to a specific part of the affected colony. Areas of dead coral skeletons covered with algae were observed in the center of the lesions. The edges of the lesion were smooth, and the margins were distinct. Most of the colonies were moderately to severely affected by the White Syndromes. The loss of polyp and coenosarc was evident from the bare white coral skeleton. Tissue loss was acute to sub-acute, as evidenced by the large areas of denuded and algae-free coral skeleton. Although there are some indications that the White Syndromes compromise the photo-physiology of the affected coral, as observed from

the slight reduction in Fv/Fm and $rETR_{max}$ values (Figure 3.A), the extensively observed disease around these two sites does not show any significant variation ($p < 0.05$) (Tables 1 and 2) in any of the photo-physiological parameters between the healthy part of the coral and near the disease lesion. Fv/Fm declined slightly from (0.724 ± 0.001) to (0.719 ± 0.023), (One-way ANOVA, $p = 0.553$, $F_{1,n} = 0.40$). rETRmax decreased from (88.93 ± 14.12) to (70.25 ± 5.31) (One-way ANOVA $p = 0.0626$, $F_{1,n} = 6.07$). NPQmax declined from (0.441 ± 0.339) to (0.266 ± 0.336) (One-way ANOVA $p = 0.400$, $F_{1,n} = 0.79$). α increased from (0.243 ± 0.045) to (0.262 ± 0.080) (One-way ANOVA, $p = 0.682$, $F_{1,n} = 0.19$). β declined from (0.0352 ± 0.0050) to (0.0283 ± 0.0258) (One-way ANOVA $p = 0.474$, $F_{1,n} = 0.60$). Ik increased from (378.37 ± 128.82) to (419.89 ± 290.28) (One-way ANOVA, $p = 0.757$, $F_{1,n} = 0.10$).

Discussion

The morphological diagnosis of Black Band Disease on the plating *Montipora* sp. coral aligns with the observations of several other studies (Aeby et al. 2015; Chen et al. 2017; Das et al. 2022). This disease has been previously reported in *Montipora* at various locations worldwide (Aeby et al. 2015; Chen et al. 2017; Das et al. 2022). Although the results of the photo-physiology of the Black Band Disease on *Montipora* sp. did not show any significant difference between the healthy part of the affected coral colony and near the actual disease lesion, declines in parameters such as Fv/Fm and $rETR_{max}$ indicate some level of compromised photo-physiological performance near the lesion compared to the healthy coral part. This observation aligns with that of Roff et al. (2008), who reported reduced Fv/Fm and rETRmax values closer to the Black Band Disease lesion in *Cyphastrea microphthalma* (Lamarck, 1816). However, the same study also highlighted the strong spatial variation and heterogeneity in the photo-physiology of this particular coral disease. This might explain the non-significant differences observed in the photo-physiological parameters between the healthy part and near the disease lesion. Chlorophyll *a* fluorescence should have been recorded at a much finer spatial scale, on the order of mm, as in Roff et al. (2008), in order to accurately decipher variations in the photo-physiology of the Black Band Disease across the coral colony. The reduction in Fv/Fm or $rETR_{max}$ can be attributed to the highly anaerobic and sulfide-rich micro-environment that exists closer to the Black Band (Sato et al. 2017). In addition to the lack of finer spatial resolution for chlorophyll *a* fluorescence measured in this study for the Black Band Disease, the lack of significant difference between the photo-physiology of the healthy part of the coral and that near the disease lesion can be explained by the presence of photosynthetic activities (Sato et al. 2016) close to the lesion by one of the causative cyanobacterial pathogens of this disease, *Phormidium corallyticum* (Rützler and Santavy 1983). The presence of cyanobacteria may influence the accurate measurement of chlorophyll fluorescence in the coral. This finding suggests that monitoring for this disease should include fine-scale (mm-level) PAM measurements using Imaging-PAM near BBD fronts to detect early decline.

The Black Band Disease on *Platygyra* sp. has been previously reported on this genus at several sites worldwide (Thinesh et al. 2014; Hadaidi et al. 2018; Aeby et al. 2020; Bharath et al. 2020; Mohamed et al. 2023). Similar to *Montipora* sp., the morphological diagnosis of Black Band Disease on *Platygyra* Ehrenberg, 1834 has been confirmed by multiple studies (Beeden et al. 2008; Hadaidi et al. 2018; Bharath et al. 2020). The lower Fv/Fm values observed near the lesion of the Black Band Disease, as opposed to the healthy part of the coral, match the observation of Roff et al. (2008) and Jogee et al. (2023a), who both reported a decline in Fv/Fm values closer to the Black Band Disease lesion. This drop in the maximum quantum yield can be attributed to the presence of an anaerobic and sulfide-rich environment, caused by the sulfide-oxidizing bacteria *Beggiatoa* spp. (Sato et al. 2017), which is unfavorable for photosynthesis by zooxanthellae inside the coral tissues. This unfavorable condition can also lead to the expulsion of zooxanthellae, as reported for the impact of zooxanthellae exposure to anaerobic conditions (Howard and Schul 2023). This could help explain the reduction in photosynthesis near the Black Band Disease lesion. However, Roff et al. (2008) reported a significant increase in NPQmax values closer to the disease lesion, whereas our observation showed no such trend. This may be attributed to significantly coarser spatial resolution for data collection in this study compared to the above study, or damage to the photosynthetic apparatus of the zooxanthellae. Similarly to the BBD on *Montipora* sp., this finding suggests that monitoring for this disease should include fine-scale (mm-level) PAM measurements using Imaging-PAM near BBD fronts to detect early decline.

The Skeletal Eroding Band disease on *Favia* sp. has been previously reported in this coral genus (Montano et al. 2012) and described with similar morphological characteristics (Beeden et al. 2008; Page and Willis 2008; Montano et al. 2012). Our observation on the reduction of Fv/Fm corroborates that of Jogee et al. (2023a), but not with that of Roff et al. (2008), who observed no variation in Fv/Fm between the healthy part and the disease lesion. Only an increase in $rETR_{max}$ was observed further away from the lesion by Roff et al. (2008). The decline in Fv/Fm values near the disease lesion can be attributed to the loss of the coral tissues along with the photosynthetic zooxanthellae in that part of the coral as a result of the spinning movement of the causative pathogen, *Halofolliculina corallasia*, which embeds itself in the coral skeleton and disrupts the coral tissue. *Halofolliculina corallasia* has also been observed to engulf zooxanthellae cells (Page et al. 2015), which could explain the reduction in Fv/Fm values closer to the disease lesion compared to the healthy-looking coral tissues. Tissue loss also occurs as a consequence of chemical secretions that are produced by the pathogens of the Skeletal Eroding Band disease during the ciliate's loricae formation process (Page et al. 2015). In addition to the loss of coral tissue and zooxanthellae, the ingestion of photosynthetic zooxanthellae cells by pathogenic ciliates has also been observed (Ravindran et al. 2023). The reduction in alpha values observed near the lesion compared to the healthy part of the coral may be explained by the compromised light-harvesting and

processing efficiency resulting from the eroded corallite structures and, thus, the compromised light microenvironment. These findings indicate that early detection via PAM fluorometry could inform management actions to prevent widespread disease propagation.

The White Band disease observed on the branching *Acropora* sp. colony at Plateau Bénitier is consistent with the morphological descriptions provided in multiple studies conducted worldwide (Lentz et al. 2011; Ainsworth et al. 2015; Nugraha et al. 2019; Gignoux-Wolfsohn et al. 2020). The observation of the bleaching pattern on the *Acropora* sp. coral associated with the White Band suggests that the disease may be of White Band Type II (Ritchie and Smith 1998). As opposed to a study by Mattan-Moorgawa et al. (2017) and Jogee et al. (2023a), who reported lower effective quantum yield and NPQ_{max} in White Band-affected *A. muricata*, no significant difference ($p > 0.05$) in the photo-physiological parameters was reported between the healthy part of the coral and near the disease lesion. Another possible explanation could be the presence of photosynthetically active and competent coral tissues with zooxanthellae cells in the disease lesion where the measurements were recorded. The remaining zooxanthellae cells might still emit a fluorescence signal. This observation warrants the need for visual monitoring paired with molecular diagnostics to proactively detect sub-lethal infection.

White Syndrome is the general name given to a widespread group of observed lesions that are characterized as white patches, bands, or blotches on generally non-acroporid corals (Bourne and Willis 2015; Cróquer et al. 2021). The morphological characteristics of the observed lesions in this study align with those reported by others (Bourne et al. 2015; Howells et al. 2020). This disease has been reported in the *Platygyra* Ehrenberg, 1834 genus by several studies worldwide (Bourne et al. 2015; Muzaki et al. 2017; Raksachon et al. 2017; Howells et al. 2020). White Syndrome is primarily distinguished from other White Diseases by the absence of microbial involvement in the disease progression (Ainsworth et al. 2007). While no photo-physiological assessment has been performed on White Syndromes, observations have been made on White Plague diseases in *A. muricata* and *Porites lobata* Dana, 1846, which have shown contrasting Fv/Fm, rETRmax, and NPQmax values between the near-lesion part of the affected corals and the healthy coral part (Mattan-Moorgawa et al. 2017). This contradicts our findings, which did not reveal any significant differences. However, another study by Roff et al. (2008) on the photophysiology of White Syndrome in *Acropora* made a similar observation to this study, where the tissues near the disease lesion did not appear to be photosynthetically compromised. The explanation of this finding was revealed by the observation of structurally intact zooxanthellae cells with no signs of degradation at the disease lesions (Roff et al. 2008). This might explain our observation of no significant difference in any of the photo-physiological parameters investigated in this study between the healthy part of the coral and the area near the disease lesions in White Syndrome-affected coral colonies. Given the potential for this disease to persist without measurable photosynthetic impairment, management should combine visual prevalence surveys with histological or molecular assays.

Across the diseases investigated, BBD and SEB produced clear and statistically significant declines in photo-physiological performance, particularly Fv/Fm, whereas WB and WS did not show measurable impairment despite visible lesions. This divergence likely reflects fundamental differences in pathogen biology and disease mechanisms. BBD and SEB are driven by active microbial pathogens with direct, destructive interactions with coral tissues and their symbiotic algae. In BBD, the polymicrobial consortium—dominated by cyanobacteria (*Phormidium corallyticum*) and sulfide-oxidizing bacteria (*Beggiatoa* spp.)—creates a hypoxic, sulfide-rich microenvironment at the lesion front (Rützler and Santavy 1983; Sato et al. 2017), which is toxic to zooxanthellae and impairs photosynthetic electron transport. In SEB, the ciliate *Halofolliculina corallasia* physically bores into coral skeletons and ingests zooxanthellae cells (Page et al. 2015; Ravindran et al. 2023), leading to direct loss of photosynthetic tissue. Both diseases, therefore, result in immediate and severe disruption of coral-algal symbiosis and light-harvesting efficiency, producing measurable declines in chlorophyll fluorescence metrics. In contrast, WB and WS lesions did not exhibit significant changes in Fv/Fm, *rETRmax*, or other PAM parameters. For WB Type II, although pathogenic bacteria are suspected (Ritchie and Smith 1998), the lesion margins in this study contained photosynthetically competent tissues, suggesting that the rate of tissue loss may not yet have impaired algal photochemistry. WS, on the other hand, lacks a confirmed microbial pathogen (Ainsworth et al. 2007) and may result from abiotic stressors or secondary colonization of already stressed tissue, allowing zooxanthellae within lesion-adjacent tissues to remain intact and photosynthetically active (Roff et al. 2008). This would explain the maintenance of chlorophyll fluorescence despite visible tissue loss or discoloration. The pronounced differences in physiological impact observed here highlight the importance of integrating pathogen biology into coral disease diagnostics. Microbially mediated diseases such as BBD and SEB produce predictable and rapid declines in photo-physiology, while non-microbial or less aggressive etiologies may leave photosynthetic parameters unaffected until later stages. Recognizing these distinctions is crucial for designing targeted monitoring protocols, selecting early-warning indicators, and prioritizing management interventions.

In conclusion, this study highlights the photo-physiological impacts of coral diseases on reef-building corals around Rodrigues Island, with significant reductions in Fv/Fm for Black Band Disease and Skeletal Eroding Band. While some diseases showed no major physiological effects, limitations in measurement resolution were noted. The identification and monitoring of the progression rates of high-impact diseases and their associated physiological signatures can guide prioritization of intervention sites. The findings of this study support the adoption of a tiered approach: (i) standardized lesion classification (Beeden et al. 2008; Work and Aeby 2008) for field surveys; (ii) targeted PAM fluorometry at lesion margins for early detection of functional decline; and (iii) integration of

environmental monitoring (temperature, light, turbidity) to link disease dynamics with environmental drivers. Such an approach will improve temporal resolution, diagnostic accuracy, and comparability of disease data across WIO reef systems. The results of this study can support the design of adaptive management strategies that incorporate disease surveillance into Marine Protected Area (MPA) management plans, guide restoration site selection under national reef recovery programs, and strengthen regional cooperation on coral health monitoring. The findings highlight the vulnerability of key coral species to disease-driven stress and underscore the need for further physiological and microbiological characterization as well as increased temporal and spatial PAM data collection to inform species-specific conservation and adaptive reef management strategies for Rodrigues Island.

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