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Disease Dynamics in Hard Corals: Transmission Study of *Desulfovibrio salexigens* and *Acinetobacter* sp.

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Abstract

The objective of this study was to analyze the dynamics of spread and tissue damage due to infection with Black Band Disease (BBD) on *Pachyseris* sp. and Brown Disease (BrB) on *Acropora* sp. Additionally, the effect of ambient temperature on transmission rates was investigated. The results demonstrated that BBD on *Pachyseris* sp. caused progressive tissue damage, characterized by zones of necrosis and distinctive black bands separating healthy tissue from dead tissue. At 31°C, the disease transmission rate increased twofold compared to 29°C, with an infection rate reaching 1.72 ± 0.76 cm/day. BrB on *Acropora* sp. showed the highest infection rate, reaching 2.20 ± 0.41 cm/day at 29°C with a bacterial concentration of 10^6 CFU/ml. However, the infection rate decreased significantly at 31°C for all bacterial concentrations tested. The disease propagated linearly along the coral branches, manifesting as yellowish-brown discoloration attributable to symbiont ciliate activity. The virulence of pathogens such as *Acinetobacter* sp. increased at 31°C, accelerating the spread of necrosis through the production of toxins and enzymes that damage the coral epithelium. BrB symptoms appeared within 2 days at 29°C and only 1 day at 31°C. This study confirms that high temperature and sedimentation play a key role in accelerating disease dynamics in corals. Increasing seawater temperatures due to global climate change create ideal conditions for the spread of disease, threatening the sustainability of coral reef ecosystems. To mitigate these challenges, a multifaceted approach involving environmental management, carbon emission reduction, and the development of biotechnology to enhance coral resistance to pathogens is essential.

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INTRODUCTION

The coral reef ecosystem plays a crucial role in marine biodiversity and ecosystem function, offering habitat for a variety of marine organisms and providing fisheries resources. However, the recent surge in diseases affecting corals is a matter of significant concern, attributable largely to the combined impacts of climate change and anthropogenic activities (Harvell *et al.*, 1999). Notable among these diseases are Black Band Disease (BBD) and Brown Band Disease (BrB), which have been identified as significant contributors to coral reef degradation worldwide, including in tropical regions such as Indonesia (Sharma and Ravindran, 2020). The primary causative agents of these diseases are pathogenic bacteria, including *Desulfovibrio salexigens* and *Acinetobacter* sp., which have been observed to exhibit heightened virulence under specific environmental conditions. Increases in sea temperature, for example, have been shown to accelerate bacterial infection rates and exacerbate damage to corals (Boyett *et al.*, 2007). Recent studies have even demonstrated that *Desulfovibrio* can survive and thrive better in sediment-rich environments, posing challenges to coral disease mitigation efforts (Zhou *et al.*, 2023). Nevertheless, the specific mechanisms of transmission of these bacteria in healthy corals still require further exploration.

Coral disease dynamics are influenced by environmental factors, including temperature, water quality, and sedimentation levels. Temperature has been shown to play a crucial role in increasing coral susceptibility to pathogens (Haapkylä *et al.*, 2007). Research indicates that BBD on *Pachyseris* sp. develops more rapidly at temperatures above 30°C, while BrB on *Acropora* sp. shows a similar pattern, with severity influenced by the morphological structure of the coral. In the context of elevated temperatures, bacteria such as *D. salvigens* secrete enzymes that compromise coral tissue integrity. The bacterial concentration within the environment also exerts a significant influence on the severity of the infection (Rosenberg and Ben-Haim, 2002). Furthermore, temperature fluctuations, induced by environmental stress, have been demonstrated to not only augment pathogen activity but also diminish coral resistance to infection (Antsiferov *et al.*, 2017). These factors underscore the pivotal role of temperature in driving the evolution of coral disease dynamics, a process that is further influenced by global climate change.

The theoretical model explaining how temperature affects the virulence of coral pathogenic bacteria includes several key mechanisms. First, increased temperature accelerates bacterial metabolism, increasing the production of proteolytic enzymes and toxins that damage coral tissue. Second, high temperatures activate the expression of virulence genes in bacteria, such as the type III and VI secretion systems (Kimes *et al.*, 2012; Yi *et al.*, 2022). Third, increased temperature increases the secretion of toxins such as hemolysins (Guijarro *et al.*, 2015; Sheikh *et al.*, 2022). Fourth, temperature changes affect the fluidity of bacterial membranes, facilitating the secretion of virulence factors and increasing the invasion of host cells (Lam *et al.*, 2014; Sheikh *et al.*, 2022). Fifth, high temperatures can activate bacterial quorum-sensing systems, which regulate the expression of various virulence factors (Sheikh *et al.*, 2022; Shapiro and Cowen, 2012). Finally, increased temperature increases the efficiency of bacterial effector translocation into coral host cells (Yi *et al.*, 2022; Kimes *et al.*, 2012). This theoretical model underlies the hypothesis that increased temperature will accelerate the rate of infection and tissue damage in the *Pachyseris* sp. and *Acropora* sp. corals tested in this study. This understanding is important for developing more effective coral disease mitigation strategies in the context of global climate change.

The virulence mechanism of coral pathogenic bacteria involves several key

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processes. Bacteria such as *D. salexigens* and *Acinetobacter* sp. produce various toxins and enzymes that play a role in pathogenesis. *D. salexigens*, as a sulfate-reducing bacteria, produces sulfide, which is toxic to coral tissue and also produces proteolytic enzymes that can degrade host tissue (Rahmi *et al.*, 2020). Meanwhile, *Acinetobacter* sp. produces zinc-metalloprotease enzymes that play an important role in its virulence, as well as chondroitin sulfatase or hyaluronidase enzymes that help accelerate host tissue damage (Shadan *et al.*, 2023).

The production of these toxins and enzymes increases at higher temperatures, explaining why bacterial infections in corals are often more severe under warming seawater conditions. Studies have shown that temperatures above 27°C play a direct role in upregulating several *V. coralliilyticus* virulence genes, including factors involved in host degradation, secretion, antimicrobial resistance, and motility (Kimes *et al.*, 2012).

In addition, pathogenic bacteria also have mechanisms to evade the host immune system, such as cell surface modification and host factor mimicry, which allow them to survive and reproduce in coral tissues (Shadan *et al.*, 2023). Increasing temperature also increases the chemotactic ability of pathogenic bacteria, with an increase of more than 60% at temperatures ≥23°C, indicating an increased ability to track chemical cues from the host (Garren *et al.*, 2016).

Recent studies have also revealed the activation of two type VI secretion systems (T6SS) in *V. coralliilyticus* at high temperatures, which allows this bacterium to evade coral host defence mechanisms. Increasing seawater temperatures due to climate change may further exacerbate the virulence of pathogens such as *V. coralliilyticus*, potentially significantly altering coral reef ecosystems (Wang *et al.*, 2024; Tout *et al.*, 2015).

Despite the ongoing expansion of research in the field of coral disease epidemiology, there persists a dearth of knowledge that hinders the formulation of effective mitigation strategies. A significant lacuna in our understanding pertains to the intricate interplay between ambient temperature and pathogen concentration, and their impact on infection rates and coral morphology. Recent studies that have examined the dynamics of *D. salexigens* on *Pachyseris* sp. and *Acinetobacter* sp. on *Acropora* sp. under controlled laboratory conditions have yielded valuable insights. Research indicates that elevated temperatures can accelerate infection rates by more than two orders of magnitude compared to lower temperatures (Berkelmans and Willis, 1999). Furthermore, corals with specific morphological structures exhibit heightened susceptibility to infection due to sediment accumulation, which creates conditions conducive to the proliferation of pathogenic bacteria (Bruno *et al.*, 2007). Recent studies have also demonstrated that temperature variations can expedite pathogen transmission, potentially impacting broad-scale ecosystems (Minz *et al.*, 1999). This study makes an important contribution to future coral reef conservation efforts by uncovering the complex relationships between temperature, pathogen concentration, and coral vulnerability.

METHODOLOGY

Ethical Approval

This study did not require ethical approval as it involved experiments on corals and invertebrate organisms not subject to animal ethics requirements. All coral specimens were collected under local conservation regulations and with proper permits from relevant authorities. The bacterial strains, *D. salexigens* and *Acinetobacter* sp., were isolated and handled following standard microbiological safety protocol.

Place and Time

This study was conducted between June and August 2020 at the Marine Laboratory of the Faculty of Marine and Fisheries Sciences, Hasanuddin University, Makassar, South Sulawesi, Indonesia. Coral fragments of *Pachyseris* sp. and *Acropora* sp.

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were collected from reef sites located within the Spermonde Archipelago (5°03′S, 119°21′E), which represents a typical tropical coral reef environment.

Research Materials

This study used *D. salexigens* (ATCC 14822) and *Acinetobacter* sp. (strain AC-17) cultured in Sea Water Complete (SWC) media. Coral fragments of *Pachyseris* sp. and *Acropora* sp. were collected at depths of 3–5 m and acclimatized in aquaria with filtration and circulation systems. Sterilization was performed using filtered seawater and 1% Povidone-iodine solution. Transmission tests were conducted in controlled aquaria with temperatures (of 29°C and 31°C) maintained by calibrated heaters, and disease progression was documented using high-resolution cameras and digital calipers.

Research Design

This study investigated coral-pathogen interactions using *D. salexigens* and *Acinetobacter* sp., cultured in SWC media (5 g/L bacto-peptone, 1 g/L yeast extract) at 28°C and 140 rpm for 24 hours. Bacterial concentrations (10^2–10^6 CFU/mL) and temperatures (29°C and 31°C) were tested on *Pachyseris* sp. and *Acropora* sp. fragments collected via minimally invasive methods. Coral acclimatization included sterilization with 1% Povidone-iodine and maintenance in aquaria with controlled water flow (1200 L/h) and lighting (150–180 µmol photons $m^{-2}s^{-1}$). A factorial design (2 temperatures × 3 concentrations × 2 coral species) was implemented in 45 × 30 × 30 cm aquaria (5 L seawater), with three replicates per treatment.

Work Procedure

Bacterial Cultures

This study utilized two types of pathogenic bacteria: *D. salexigens*, which was isolated from aquatic environments with high organic content, and *Acinetobacter* sp., which was obtained from infected corals. *D. salexigens* is an anaerobic bacterium that has been shown to play an important role in the organic degradation process and has been demonstrated to be able to adapt to marine environments with high sediment content (Zhou *et al.*, 2023). *Acinetobacter* sp. is classified as an opportunistic bacterium, capable of thriving in disturbed environmental conditions, including on corals experiencing stress from environmental factors such as elevated sea temperatures (Benavides *et al.*, 2021).

The bacterial isolates were cultivated in SWC (Sea Water Complete) media, which contains bacto-peptone, yeast extract, glycerol, and bactoagar. This medium was selected because it fosters the optimal growth of marine pathogenic bacteria by supplying nutrients that mirror natural environmental conditions (Antsiferov *et al.*, 2017). The culture process was carried out in liquid media using a shaker incubator at 28°C with a speed of 140 rpm for 24 hours, ensuring uniform oxygen distribution to support the growth of facultative bacteria such as *Acinetobacter* sp.

The bacterial concentrations utilized in this study ranged from 10^2 to 10^6 colony-forming units (CFU) per milliliter. The primary objective of this study was to assess the impact of bacterial concentration on the rate of coral infection. The selection of these concentration variations was based on the findings of previous studies, which demonstrated that increasing pathogen concentrations can accelerate infection rates. However, the impact of these variations varies depending on the type of bacteria and the level of host resistance (Minz *et al.*, 1999).In this study, it is hypothesized that this experimental approach will provide deeper insights into the mechanisms of interaction between pathogens and hosts in tropical corals.

Coral Acclimatization

Coral fragments of *Pachyseris* sp. and *Acropora* sp. were collected from the waters surrounding the study site, to ensure ecosystem sustainability and employ minimally invasive collection procedures to avert habitat destruction. After collection, the coral fragments were acclimatized in the laboratory for five days, to facilitate their

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adaptation to the experimental conditions. The fragments were acclimatized in sterile aquaria equipped with adequate water circulation and aeration to maintain water quality and provide a stable environment for the corals (Padilla-Gamiño *et al.*, 2022). This process was implemented to reduce stress due to environmental changes that could potentially affect the study's results.

Before the transmission test, coral fragments underwent a sterilization process using a sterile seawater solution. Additionally, fragments were immersed in a 1% Povidone-iodine solution for 3-4 minutes to remove external microorganisms that could be a source of contamination during testing (Giudice and Rizzo, 2022). Following the sterilization process, the fragments were rinsed with sterile seawater and transferred into treatment aquariums that had been prepared beforehand and met the necessary environmental parameters, including temperature and salinity, to ensure consistent conditions throughout the study. The controlled placement of the fragments in these aquariums was crucial to ensure that the observed effects on the corals were attributable to the specific treatment and not to other environmental factors.

Transmission Test

Tests were conducted to evaluate the effect of temperature and bacteria concentration on disease severity in corals, focusing on the specific responses of *Pachyseris* sp. and *Acropora* sp. A 45 cm × 30 cm × 30 cm aquarium was filled with 5 liters of sterile filtered seawater and equipped with aeration to maintain optimal water quality throughout the experiment (Padilla-Gamiño *et al.*, 2022). Two temperature treatments were applied: 29°C and 31°C. A periodically calibrated aquarium heater was used to stabilize the temperature, ensuring consistency in the experimental environment. This temperature was selected based on previous research, which demonstrated that temperatures above 28°C led to an increase in the virulence of coral-pathogenic bacteria (Giudice and Rizzo, 2022).

To enhance the reliability of the results, each combination of temperature treatment and bacterial concentration (10^2, 10^4, and 10^6 CFU/ml) was replicated thrice, with each aquarium housing a single coral fragment. The incubation period spanned seven days, during which coral morphology was meticulously monitored for indications of bacterial infection. Symptoms such as tissue discolouration, areas of necrosis, and mucus formation on coral fragments were visually observed using a high-resolution underwater camera to ensure accurate documentation (Benavides *et al.*, 2021).

This experimental approach facilitates in-depth analysis of pathogen transmission dynamics, particularly the influence of temperature and bacteria concentration interactions on disease severity. Recent research has demonstrated that a combination of environmental factors, such as temperature and bacteria, can accelerate disease spread in tropical corals, thereby substantiating the relevance of this research design (Zhou *et al.*, 2023).

Coral Infection Rate

The coral infection rate was systematically measured by recording the progression of the infected area on coral fragments at 5-hour intervals using a digital ruler or high-precision caliper. This approach enables consistent monitoring of disease spread dynamics. In *Pachyseris* sp, the presence of Black Band Disease (BBD) was indicated by the spread of distinctive white areas, while in *Acropora* sp., the presence of Brown Band Disease (BrB) was identified through the discolouration of coral tissue (Giudice and Rizzo, 2022). The extent of the infected area was meticulously measured and calculated to ascertain disease severity using a quantitative approach, which is regarded as a pivotal indicator in evaluating the impact of pathogens on coral ecosystems (Benavides *et al.*, 2021).

This measurement procedure was supported by visual documentation using high-resolution underwater cameras, which allowed for in-depth analysis of changes in coral tissue morphology. Recent studies have

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demonstrated that coral infection rates are influenced by interactions between environmental conditions, such as temperature and water quality, and pathogen concentrations (Zhou *et al.*, 2023). Prior studies have indicated that elevated water temperatures can hasten infection rates by increasing the metabolic activity and virulence of pathogenic bacteria, such as *D. salexigens* and *Acinetobacter* sp. (Padilla-Gamiño *et al.*, 2022). These factors elucidate the variation in infection severity across diverse coral types and environments. This quantitative approach offers a more nuanced understanding of the mechanisms underlying coral disease spread and provides a crucial foundation for developing more effective mitigation strategies in the face of escalating disease incidence associated with global climate change.

Data Analysis

The statistical analysis of the obtained data employed a two-factor test (two-way ANOVA), which facilitated the evaluation of the interactive effect of temperature and bacterial concentration on coral infection rates. This methodological approach enabled the identification of individual effects and interactions between the two independent variables on coral response to infection. Subsequent tests employed the Tukey post-hoc method to compare treatments at a significance level of $p<0.05$, ensuring a more precise and comprehensive interpretation of the results (Benavides *et al.*, 2021).

The infection rate data were presented as mean ± standard deviation, providing a more representative distribution of results and facilitating interpretation of the level of variability between treatments. In addition, descriptive analysis of changes in coral morphology was conducted to support quantitative results with qualitative data. Documentation was conducted through histology photography of coral tissue before and after transmission tests using a high-resolution digital microscope. This approach yielded visual evidence of changes in infected coral tissue, including tissue degradation, mucus formation, and necrosis (Giudice and Rizzo, 2022).

This comprehensive data analysis accounts for the dynamic relationship between environmental variables and pathogenic factors, which is important for understanding the mechanisms of coral disease spread in the context of global climate change. Recent studies have also shown that statistically-based analyses, when coupled with advanced imaging methods, can provide deeper insights into infection mechanisms and their effects on coral tissue (Padilla-Gamiño *et al.*, 2022; Zhou *et al.*, 2023).

RESULTS AND DISCUSSIONS

Tissue changes in *Pachyseris* sp. and *Acropora* sp.

The results showed that Black Band Disease (BBD) infection in *Pachyseris* sp. and Brown Band Disease (BrB) on *Acropora* sp. caused significant morphological changes, with damage patterns influenced by temperature factors and the morphological structure of each coral. In *Pachyseris* sp., the initial BBD infection is characterized by the appearance of a small white area on the coral surface, which develops into a circular zone of necrosis with clear boundaries in the form of black bands. This black band, which is the main sign of BBD, is a mixture of pathogenic microorganisms such as *D. salexigens*, cyanobacteria, and other anaerobic bacteria (Giudice and Rizzo, 2022). The infection rate was doubled at 31°C compared to 29°C, which is consistent with studies showing that high temperatures accelerate the metabolic activity of pathogens and exacerbate tissue damage (Zhou *et al.*, 2023). The decrease in tissue color in *Pachyseris* sp. was also accompanied by an increase in mucus production containing opportunistic microorganisms, which further accelerated the spread of the disease.

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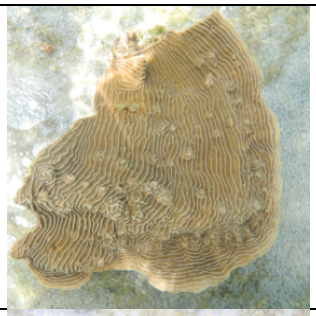
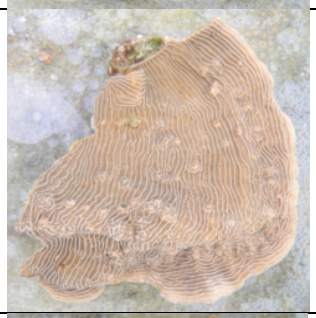
Day post-infection (dpi)	Morphology condition of *Pachyseris* sp. Water temperature (°C) 29	31
1		
2		
3		
4		
5		

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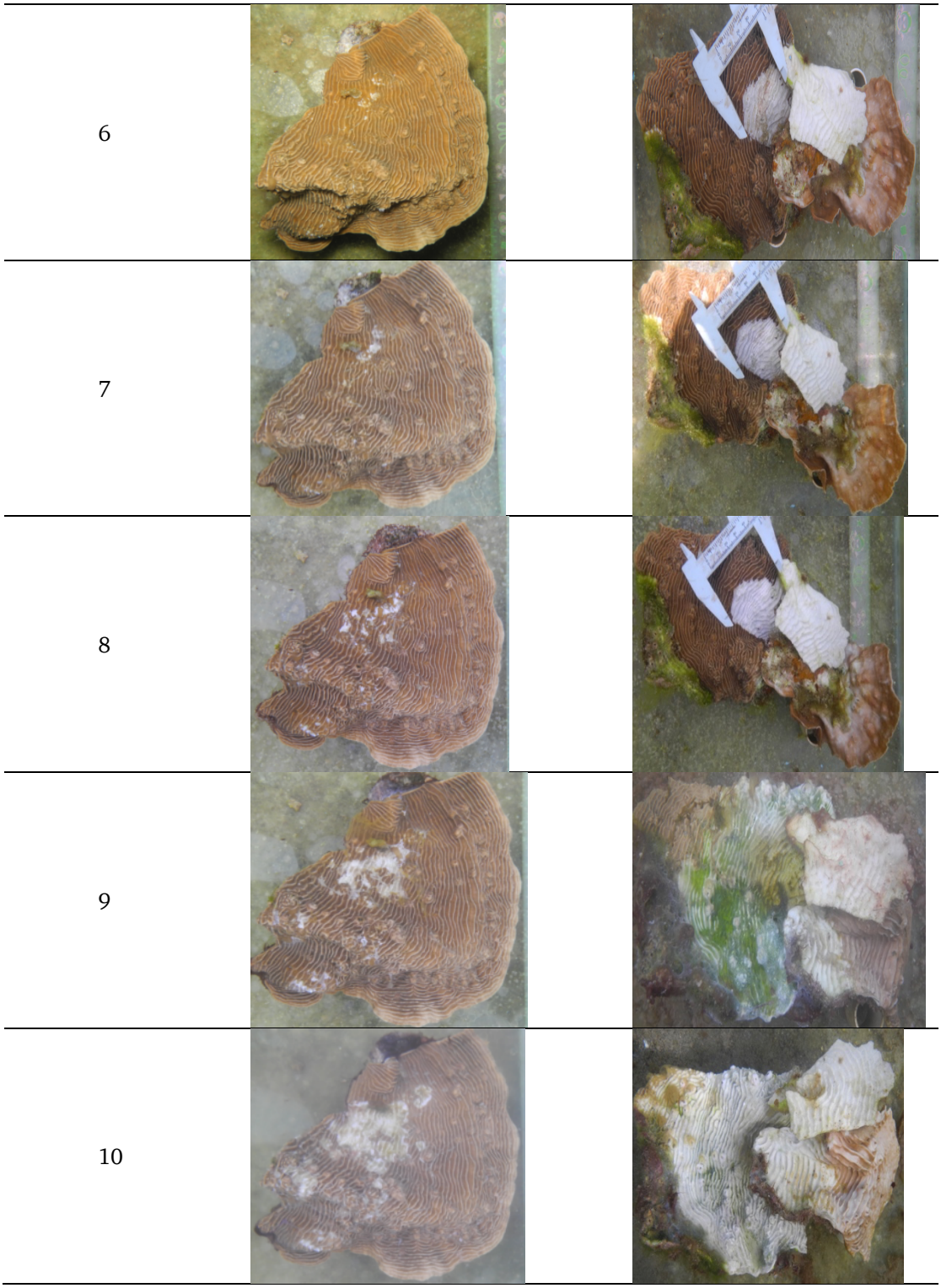

Figure 1. Changes in the condition of coral fragments after infection at temperatures of 29°C (A) and 31°C (B). *Pachyseris* sp. showed symptoms of infection on day 5 (temperature 29°C) where white spots appeared on coral fragments. While at a temperature of 31°C, white spots appeared after infection and coral fragments died on day 7 post-infection (red and yellow marks in the coral fragment area indicate the area of the fragment infected with bacteria).

The mechanism of BBD infection in *Pachyseris* sp. involves the production of proteolytic enzymes and toxins by the pathogen that damage coral tissue and

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facilitate the colonization of other bacteria. Environmental conditions such as high temperature and sedimentation also create an ideal habitat for pathogens to proliferate (Benavides *et al.*, 2021). The foliose morphological structure of *Pachyseris* sp. tends to facilitate sediment accumulation, thus increasing the risk of infection. This finding supports previous studies that coral morphology can influence the degree of disease susceptibility (Padilla-Gamiño *et al.*, 2022).

As illustrated in Figure 1, significant cell damage due to *D. salexigens* bacteria was observed in *Pachyseris* sp. at a temperature of 29°C on the fifth day of observation. Conversely, coral tissue damage was evident at a temperature of 31°C on the first day of observation. It is hypothesized that temperatures of 31°C favour the proliferation of pathogenic organisms, thereby inducing alterations in coral tissue. This finding aligns with the observations reported by Tignat-Perrier *et al.* (2023), who noted that increases in temperature above 30°C can stimulate the activity of pathogenic organisms. Furthermore, studies by previous studies (Rützler and Santavy, 1983; Viehman *et al.*, 2006; Boyett *et al.*, 2007; Sato *et al.*, 2009) and even death for aquatic organisms (Rubio-Portillo *et al.*, 2014).

Day post-infection (dpi)	Morphology condition of *Acropora* sp.	
	Water temperature (°C)	
	29	31
1		
2		
3		

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Figure 2. Changes in the condition of coral fragments after infection at temperatures of 29°C (A) and 31°C (B). *Acropora* sp. showed symptoms of infection on day 2 (temperature 29°C) where white spots appeared on coral fragments. While at a temperature of 31°C, white spots appeared after infection and coral fragments died on day 5 post-infection (red and yellow marks in the coral fragment area indicate the area of the fragment infected with bacteria).

As illustrated in Figure 2, *Acropora* sp. exhibited signs of cellular damage due to the presence of *Acinetobacter* sp. bacteria at temperatures of 29°C and 31°C on the first day of observation. However, the extent of cellular damage was found to be more pronounced at 31°C compared to 29°C. It is hypothesized that the higher temperature of 31°C fosters the proliferation of pathogenic organisms, thereby causing alterations in the cellular composition of coral organisms. It has been established that alterations in temperature, surpassing 30°C, can amplify the activity of such pathogenic organisms (Tignat-Perrier *et al.*, 2023). Moreover, an escalation in water temperature, exceeding 28°C, has been demonstrated to expedite the propagation of diseases within aquatic ecosystems (Rutzler and Santavy, 1983, Viehman *et al.*, 2006, Boyett *et al.*, 2007, Sato *et al.*, 2009) and even death for aquatic organisms (Rubio-Portillo *et al.*, 2014).

In *Acropora* sp., BrB infection has been observed to result in accelerated tissue degradation when compared with BBD in *Pachyseris* sp. The initial phase of infection is marked by a discolouration of the tissue, characterized by a yellowish-brown hue, attributable to the activity of the ciliate symbiont responsible for BrB. This damage subsequently progresses to a state of widespread necrosis along the coral branches. The study found that at 31°C, the rate of disease spread increased up to three times faster than at 29°C, suggesting that high temperatures not only increase the metabolic activity of pathogenic bacteria but also decrease host resistance (Giudice and Rizzo, 2022). The branching structure of *Acropora* sp. facilitates water flow but also increases exposure to ciliates and pathogenic bacteria. These infections are exacerbated by toxins and enzymes produced by pathogens, which damage coral epithelial tissue and facilitate the colonization of opportunistic microorganisms (Benavides *et al.*, 2021).

The results of this study confirm that changes in ambient temperature play an important role in increasing infection rates and disease severity in tropical corals. The difference in response between *Pachyseris* sp. and *Acropora* presents the importance of morphological factors and pathogen type in

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determining disease dynamics. This study underscores that environmental stressors, such as elevated temperatures and sedimentation, generate optimal conditions for disease propagation, thereby amplifying the risk of damage to coral reef ecosystems (Padilla-Gamiño *et al.*, 2022; Zhou *et al.*, 2023). Consequently, coral disease mitigation strategies must encompass environmental management, including the mitigation of the impacts of global warming and anthropogenic activities, to ensure the sustainability of marine ecosystems.

Tissue damage patterns of *Pachyseris* sp. and *Acropora* sp.

Black Band Disease (BBD) infection on *Pachyseris* sp. and Brown Band Disease (BrB) on *Acropora* sp. exhibited progressive tissue damage patterns, with severity influenced by temperature and the morphological structure of each coral species. In *Pachyseris* sp., BBD infection initiates with the formation of a circular zone of necrosis in the infected area, characterized by a distinctive black band separating healthy tissue from dead tissue. This black band is comprised of a consortium of anaerobic bacteria, such as *D. salexigens*, along with cyanobacteria and other microorganisms that collaborate in the damage to coral tissue (Giudice and Rizzo, 2022).

It has been demonstrated that elevated temperatures, particularly at 31°C, enhance the metabolic activity of the pathogens, thereby accelerating tissue damage by up to twofold compared to 29°C (Zhou *et al.*, 2023). In response to infection, *Pachyseris* sp. increases mucus production, which, although it may appear to favour the growth of opportunistic bacteria, in fact, worsens tissue conditions (Benavides *et al.*, 2021). The foliose structure of *Pachyseris* sp. facilitates sediment accumulation, creating ideal environmental conditions for pathogens to thrive (Benavides *et al.*, 2021). In contrast, BrB infection in *Acropora* sp. caused more rapid and extensive tissue damage than BBD. Early symptoms include yellowish-brown discolouration due to the activity of symbiont ciliates associated with the pathogen. The spread of necrosis occurred linearly along the coral branch, with infection rates increasing up to three times at 31°C compared to 29°C.

High temperatures have been shown to not only increase the activity of pathogens such as *Acinetobacter* sp. but also weaken host defences through decreased levels of antimicrobial compounds in coral mucus (Padilla-Gamiño *et al.*, 2022). The branching structure of *Acropora* sp., although allowing better water flow, increases exposure to pathogens in the water column. The infection process involves the production of toxins and enzymes by pathogens, which damage the coral epithelium and facilitate the colonization of opportunistic microorganisms.

This disparity in damage levels between the two coral types underscores the pivotal role of morphological characteristics and pathogen type in shaping infection dynamics. The foliose structure of *Pachyseris* sp. augments the risk of sediment accumulation, while *Acropora* sp., with its branching structure, exhibits heightened susceptibility to pathogen exposure in column water. Moreover, elevated temperatures, a preeminent environmental factor, intensify infection dynamics through amplified pathogen activity and diminished host resistance (Zhou *et al.*, 2023). The consequences of environmental stresses, such as elevated temperatures and sedimentation—which are frequently initiated by anthropogenic activities—present considerable challenges for coral reef ecosystems.

These factors also augment the risk of disease propagation, jeopardizing the long-term viability of tropical coral reefs. The findings of this study underscore the imperative for comprehensive environmental management strategies to mitigate coral diseases. These strategies encompass the reduction of global warming and sedimentation impacts, the regulation of ocean temperature and water quality, and the advancement of biotechnologies to

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enhance coral resistance to pathogens. This research provides critical evidence for the development of more effective conservation policies to deal with coral disease threats in the context of global climate change.

The transmission rate of *Pachyseris* sp. and *Acropora* sp.

The study demonstrated that disease transmission rates in *Pachyseris* sp. due to Black Band Disease (BBD) and in *Acropora* sp. due to Brown Band Disease (BrB) were considerably influenced by temperature and pathogen type. In *Pachyseris* sp., BBD infection initiates with the formation of a circular zone of necrosis in the infected area, distinguished by a distinctive black band separating healthy tissue from dead tissue. This black band contains a consortium of anaerobic bacteria, including *D. salexigens*, cyanobacteria, and other microorganisms, which synergistically accelerate coral tissue destruction (Giudice and Rizzo, 2022).

Furthermore, the study observed a significant increase in disease transmission rates at 31°C compared to 29°C, suggesting a correlation between elevated temperature and enhanced pathogen metabolic activity. This heightened metabolic activity has been linked to an upsurge in the production of proteolytic enzymes and toxins, which inflict substantial damage to coral tissue (Zhou *et al.*, 2023).

Table 1. Infection rate of *Pachyseris* sp. due to infection with *D. salexigens* bacteria at different temperatures and concentrations.

Water Temperature (°C)	Concentration	Infection Rate								
		1	2	3	4	5	6	7	8	9
29	10^2	0.96±0.15^{a}	0.82±0.05^{a}	0.81±0.04^{a}	0.83±0.07^{a}	1.07±0.29^{a}	1.30±0.35^{a}	1.24±0.39^{a}	1.27±0.40^{a}	1.29±0.64^{a}
	10^4	1.16±0.21^{a}	0.92±0.06^{a}	1.01±0.03^{a}	0.76±0.10^{a}	0.84±0.31^{a}	0.83±0.38^{a}	0.92±0.48^{a}	0.93±0.43^{a}	1.12±0.68^{a}
	10^6	1.02±0.07^{a}	0.88±0.06^{a}	1.04±0.29^{a}	0.80±0.10^{a}	0.85±0.26^{a}	0.84±0.38^{a}	0.95±0.43^{a}	0.95±0.39^{a}	1.14±0.58^{a}
31	10^2	1.00±0.13^{a}	0.82±0.04^{a}	0.77±0.03^{a}	0.86±0.05^{a}	1.12±0.27^{b}	1.33±0.47^{a}	1.37±0.49^{a}	1.49±0.56^{a}	1.72±0.76^{a}
	10^4	0.85±0.19^{a}	0.81±0.07^{a}	0.90±0.04^{a}	0.81±0.05^{a}	0.77±0.29^{a}	1.03±0.38^{a}	0.90±0.49^{a}	0.96±0.60^{a}	1.21±0.63^{a}
	10^6	0.90±0.08^{a}	0.89±0.09^{a}	0.98±0.20^{a}	0.87±0.05^{a}	0.82±0.02^{ab}	1.20±0.22^{a}	1.07±0.21^{a}	1.20±0.18^{a}	1.08±0.49^{a}

Data are presented as mean ± SD, different superscript letters in the same column indicate significantly different results in Tukey's test ($p<0.05$).

Table 2. Infection rate of *Acropora* sp. due to infection with *Acinetobacter* sp. bacteria at different temperatures and concentrations.

Water Temperature(°C)	Concentration	Infection Rate			
		1	2	3	4
29	10^2	1.09±0.19^{a}	1.01±0.15^{a}	0.95±0.34^{a}	1.45±0.08^{a}
	10^4	1.41±0.14^{a}	1.47±0.19^{a}	1.31±0.08^{a}	2.00±0.43^{a}
	10^6	1.45±0.15^{a}	1.28±0.16^{a}	1.32±0.10^{a}	2.20±0.41^{a}
31	10^2	0.27±0.27^{a}	0.32±0.32^{a}	0.23±0.23^{a}	0.47±0.47^{a}
	10^4	0.19±0.19^{a}	0.48±0.48^{a}	0.31±0.31^{a}	0.47±0.47^{a}
	10^6	0.09±0.09^{a}	0.23±0.23^{a}	0.09±0.09^{a}	0.19±0.19^{a}

Data are presented as mean ± SD, different superscript letters in the same column indicate significantly different results in Tukey's test ($p<0.05$).

In the context of *Acropora* sp., BrB infection showed a higher transmission rate

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at 29°C compared to 31°C. The disease spread linearly along the coral branches, with the highest infection rate reaching 2.20 ± 0.41 cm/day at 29°C with a bacterial concentration of 106 CFU/ml. However, the infection rate decreased significantly at 31°C for all bacterial concentrations tested. The initial symptoms of BrB appear as a yellowish-brown discolouration, which is caused by the activity of the ciliate symbiont associated with the pathogen. Furthermore, it has been observed that elevated temperatures enhance the virulence of Acinetobacter sp., which secretes enzymes and toxins that compromise the coral epithelium, thereby expediting the progression of necrosis (Padilla-Gamiño *et al.*, 2022). The branched structure of *Acropora* sp. offers enhanced water circulation but concomitantly amplifies exposure to pathogens in the surrounding water column, thus resulting in accelerated disease propagation.

The results of this study show different infection patterns between BBD in *Pachyseris* sp. and BrB in *Acropora* sp. This pattern is similar to *Vibrio coralliilyticus* infection in *Pocillopora damicornis* which also shows increased virulence at high temperatures (Munn, 2015). However, it is different from White Plague Disease caused by various *Vibrio* spp., which attack zooxanthellae and reduce chlorophyll concentrations (Mhuantong *et al.*, 2019). Another study on *Acropora tenuis* showed that the effects of thermal stress depend on complex interactions between members of the coral holobiont (Munn, 2015). This study confirms that the dynamics of coral disease are influenced by complex interactions between pathogen types, coral morphology, and environmental factors such as temperature.

The disparity in transmission rates between *Pachyseris* sp. and *Acropora* sp mirrors the impact of coral morphology and pathogen type on disease dynamics. In *Pachyseris* sp., foliose structures enable sediment accumulation, which fosters pathogen colonization but curtails the initial propagation of infection. Conversely, the branching architecture of *Acropora* sp. permits direct exposure to pathogens, thereby expediting the dissemination of disease. Environmental factors such as elevated temperatures were the predominant catalysts in escalating transmission rates in both coral types. These studies suggest that increased water temperature not only accelerates pathogen metabolism but also weakens host defences, such as decreased levels of antimicrobial compounds in coral mucus (Benavides *et al.*, 2021; Padilla-Gamiño *et al.*, 2022).

Environmental stresses, such as increased temperatures and sedimentation caused by human activities, create ideal conditions for the spread of coral diseases. This underscores the need for comprehensive mitigation strategies, including the implementation of environmentally sustainable development practices, such as reducing carbon emissions to mitigate global climate change and enhancing water quality to reduce sedimentation that fuels pathogen colonization (Benavides *et al.*, 2021; Padilla-Gamiño *et al.*, 2022).

The findings of this study emphasize the importance of comprehensive mitigation strategies to address coral disease threats in the context of global climate change. Some strategies that can be implemented include: environmental management to reduce the impacts of global warming and human activities, regulating sea temperatures and improving water quality to reduce stress on corals, and reducing sedimentation that can trigger pathogen colonization. Implementation of environmentally sustainable development practices and reducing carbon emissions are also important to mitigate global climate change. Development of marine probiotics to increase coral resistance to pathogens. In addition, the development of biotechnology to increase coral resistance to pathogens and the implementation of marine protected zones can support the recovery of coral reef ecosystems. These strategies aim to reduce environmental factors that trigger the spread of coral diseases and increase the

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resilience of coral reef ecosystems to future disease threats. Furthermore, additional research is necessary to engineer biotech solutions that enhance coral's resilience to pathogens.

CONCLUSION

This study demonstrated that coral disease dynamics due to infection with Black Band Disease (BBD) on *Pachyseris* sp. and Brown Band Disease (BrB) on *Acropora* sp. were significantly influenced by environmental factors, particularly temperature, as well as coral morphology and pathogen type. In *Pachyseris* sp., BBD resulted in progressive tissue damage, with transmission rates increasing twofold at 31°C compared to 29°C. The study identified that factors such as foliose structure and sediment accumulation created an ideal environment for pathogen colonization, which exacerbated disease spread. Meanwhile, *Acropora* sp. showed a higher BrB infection rate at 29°C compared to 31°C. However, the infection rate decreased significantly at 31°C for all bacterial concentrations tested. The branching structure of *Acropora* sp. facilitates exposure to pathogens in the water column, accelerating the spread of infection. This study emphasizes the importance of environmental management to reduce the risk of coral disease, including reducing sedimentation and mitigating climate change. For further research, it is recommended to investigate the role of probiotics in preventing coral disease and develop biotechnological strategies to increase coral resistance to pathogens. In addition, research on reducing sedimentation and other environmental management can help reduce the risk of coral infection.

CONFLICT OF INTEREST

The authors declare that there is no conflict of interest among all authors upon writing and publishing the manuscript.

AUTHOR CONTRIBUTION

Rahmi was responsible for the formulation of the research idea (conceptualization), development of the methodology, implementation of the investigation, data curation, and preparation of the initial manuscript draft. Jamaluddin Jompa provided overall supervision of the research process, validated the research findings, and contributed to the critical review and editing of the manuscript. Khairun Nisaa carried out formal analysis, performed laboratory work, developed data visualizations, and assisted in the review and refinement of the manuscript. Akmal supported the research by providing resources, conducting field data collection, managing project administration, and securing funding for the implementation of the study. All authors have read and approved the final version of the manuscript before submission.

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