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LEMBAR HASIL PENILAIAN SEJAWAT SEBIDANG ATAU PEER REVIEW KARYA ILMIAH : JURNAL ILMIAH BEREPUTASI TERINDEK SCOPUS

Judul Jurnal Ilmiah (Artikel) : **Emerging of white syndrome in echinopora lamellosa at nature reserve Pulau Sempu, Indonesia**

Penulis Jurnal Ilmiah : Rosdianto (**Penulis 1**)

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Halaman 2



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Emerging of white syndrome in *Echinopora lamellosa* at nature reserve Pulau Sempu, Indonesia

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ABSTRACT

Coral reef at nature reserve Pulau Sempu has been provided food for marine biota and became livelihood for fishermen who are living at coast nearby. Coral reef at this island can be found in periphery of island and concentrated on Sempu strait (north part area). Folious coral *Echinopora lamellosa* are distributed well in Sempu bay with local name Kondang Buntung (Depan). The previous study showed the coral percentage of NR Pulau Sempu was stagnant on 26-34% of average of their life coral cover. Coral disease has role to decrease of coral cover in the world one of them is white syndrome that only be reported from Indo-Pacific area. The aim of this research was to observe white syndrome disease growth rate at *E. lamellosa* which has degraded their coral cover at NR Pulau Sempu for 2 months. To calculate white syndrome rate, we used sequence photographed that be taken twice (early and end of month) then calculated wide area using ImageJ. Growth rate of white syndrome was obtained from difference of width area. The result of this research showed the average of white syndrome disease was 0.077 cm²/ day. Environment factor suggested have important role to increase growth rate of white syndrome in this island, increasing sea surface temperature triggered virulence bacteria in coral fast proliferation and caused increase of white syndrome growth rate. White syndrome in *E. lamellosa* from NR Pulau Sempu still on normal categorized compared by other Indo-Pacific area.

Key words : Sendang Biru, Nature reserve Pulau Sempu, Foliose coral, Threat of coral reef, White syndrome

Introduction

Coral reef has been provided food for marine biota (Sawall *et al.*, 2013), antimicrobial substance (Sibero *et al.*, 2018) and became of livelihood for fishermen (Harahap *et al.*, 2019; Kasmini *et al.*, 2018; Nurdin *et al.*, 2016; Madiyani *et al.*, 2018; Suciyo *et al.*, 2019; Nugraha *et al.*, 2020; Twinandia *et al.*, 2011) who living at coastal nearby. Since early 2000's the report of

coral disease in Indo-Pacific has been increasing and became one of factors that responsible to decline of coral reef cover in this area (De'ath *et al.*, 2012; Pandolfi *et al.*, 2003). The great of number of diseases documented in coral of Indo-Pacific, it could be the most detrimental effect and major are those can cause of coral soft tissue loss, is white syndromes (WS). White syndrome is common term suggested by (Willis *et al.*, 2004) for any tissue loss on

scleractinian coral resulted reveal white skeleton in the lack of other disease symptoms causations. WS also reported affects on a diverse of coral species and very different with other coral disease such as trematodiasis and bleaching where these corals may have recovered in next several months, however, WS ensues tissue loss speedily. Consequently, the effect of WS on corals are immediately and irreversible, as demonstrated demography on small and large scale in Indo-Pacific. It eliminated coral dominance of *Acropora* and *Montipora* in Palmyra atoll, Great Barrier Reef and American Samoa, leading to alter in complexity and entanglement of coral reef ecosystem (Roff *et al.*, 2006; Ushijima *et al.*, 2012; Williams *et al.*, 2011; Wilson *et al.*, 2012).

Coral disease incidence always be traced on imbalanced the interaction of agent, host and environment. Changing environmental condition could trigger the interaction between host and agent (microbial). Increasing sea surface temperature above 1 °C of maximum monthly mean (MMM) affect on coral stress and decrease their immune system, besides microbes in the coral tissue alter more virulence resulted on disease (Work *et al.*, 2008). The agent of white syndrome are presumably various, complex and some part still unconfirmed. However, microorganisms or various bacteria might be responsible for tissue lost in coral and how they work that affects on coral health is still unclear (Work *et al.*, 2008). The acute tissue loss (necrosis) in coral sometimes associates with microorganisms such as: fungi, algae, sponge and cyanobacteria (Work and Aeby, 2011), and in the sub-acute phase these wounds are auto repaired. Hard work to find out of microbial in coral revealed that *Philaster lucinda* produce secondary infection on coral that previously had been infected by nonspecific bacterial (Sweet and Bythell, 2015). In *Montipora* coral some intra specific chimerism has been found invaded gastrovascular canal of coral (called as invasive gastrovascular multicellular structure-IGMS) and associated with tissue loss in basal area of coral (Work *et al.*, 2011).

In Indo-Pacific WS is reported based on the progression rate of tissue loss, i.e. acute (rapid) and subacute (moderate). Several types of coral affected on WS, they were *Acropora* spp (Carpenter *et al.*, 2008; Haapkylä *et al.*, 2007), *Montipora aequituberculata* (Jones, 2004), *Turbinaria mesenterina* (Dalton and Smith, 2006), *Montipora capitata* (Aeby *et al.*, 2010), *Goniastrea* spp, *Porites lutea*, and *Porites*

lutea (Roder *et al.*, 2014; Séré *et al.*, 2013). WS on *Echinopora lamellosa* also reported by (Smith *et al.*, 2014) and (Smith *et al.*, 2015), that form blisters manifested as fluid-filled sacs.

Echinopora lamellosa (Dai and Horng, 2009) was located on Kondang Buntung (Depan) (KBD) of Pulau Sempu (8°26'23.65"S; 112°40'51.81"E)(Luthfi *et al.*, 2014). Colonies of these species only found in KBD laid about 30 m in depth 3-6 m. Taxonomically *E. lamellosa* is under family Merulinidae, that characterized by foliose/ laminar formation of colony, corallite are conical plocoid or beaded like (2-4 mm in diameter), septa are exert, and many little spines between corallite. This research focuses on Nature Reserve of Pulau Sempu because the vulnerability condition of *E. lamellosa* due to many stressors originally come out from both of anthropogenic and natural sources in surrounding of this island. The specific objective of this research was to describe the gross lesion of WS in NR Pulau Sempu to extend report of spreading WS in Indo-Pacific area.

Materials and Methods

Study area and research location

This research was carried out from July to August 2018 at NR Pulau Sempu, Malang, East Java (Figure 1). The survey was conducted in a spot which is

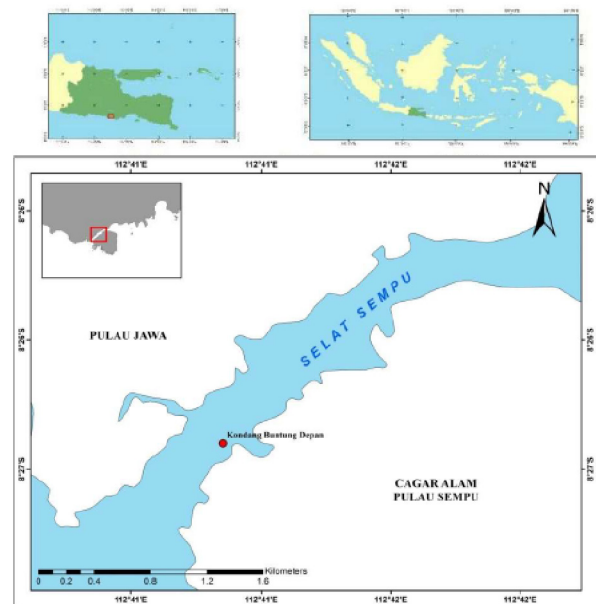


Fig. 1. The research location indicates of red round tips in NR Pulau Sempu, Malang

called as Kondang Buntung (Depan) (KBD). This point of research is located at eastern part of Sempu strait, hence strong current has been influencing it. Besides, local pollution source came from docking activity for traditional boat of fishermen. Coral in KBD is dominated by Echinophora, Porites and Montipora, those were growth from near the thin beach of Pulau Sempu until 7 m depth. No coral reef has been found after 7 m, because of dominance of sand in sharp drop off sea bed.

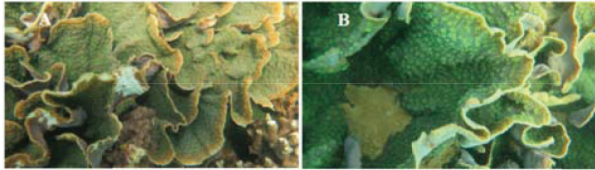


Fig. 2. An illustration of coral colony of *E. lamellosa*, A-Bare coral colonies which be found in NR Pulau Sempu.

Coral Identification

Morphology identification has been used to recognize *E. lamellosa* coral. Most of these colonies are thin leaves, calices have 3-6 mm in diameter that can be clearly seen from the colonies surface. Each calice is spaced around 3-5 m with another. The primary septa are noticeably protrude and carry paliform lobes. Colonies made up of thin laminae set out in whorls or tiers or, rarely, forming tubes. The common color of this coral is amber to greenish (Veron, 2000).

White Syndrome

White syndrome ID

White syndrome (WS) resulted irregular tissue loss in coral, the pattern is not concentrating on coral surface (focal) but diffuse marking of tissue loss that



Fig. 3. White syndrome in *E. lamellosa*

expose bare white skeleton meeting live tissue. The color of syndrome is bare white skeleton to brown because develop of algae. Often deriving from a small lesion front and escalating to a band front across the entire colony (Beeden *et al.*, 2008).

Tagging of WS

Between July and August 2018, we tagged 8 colonies of *E. lamellosa* exhibiting WS with separately (one by one) numbered plastic. The color number tided in coral colonies by a tie cable (Figure 4). Every single colony than photographed using underwater camera (Canon G 16, Japan) and measured in initial time. After 2 months the growth rate of WS measured again and compared the wide area of WS with previous. The area calculation using ImageJ program (NIH, US).

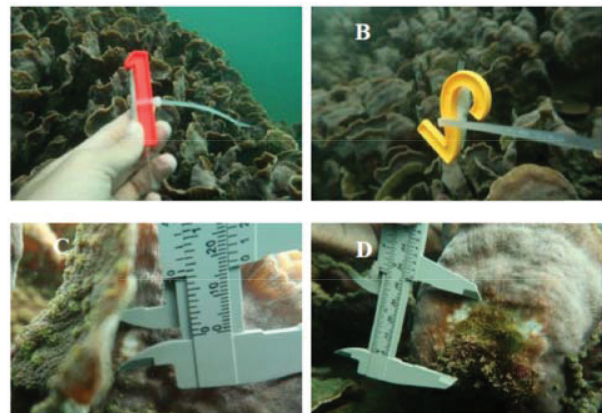


Fig. 4. White syndrome tagging in *E. lamellosa* colonies tagged (A, B) and measured by using a caliper (CD).

Results and Discussion

WS and its prevalence

Gross lesions of WS in *E. lamellosa* was categorized as multifocal with margin of syndrome is irregular. It was clear (Figure 3) tissue of *E. lamellosa* was lose that affected on exposed skeleton of coral. The syndrome may result from some bacteria of genus *Vibrio*, et least five vibrio that been attributed on WS of coral, they were *V. harveyi*, *V. mediterranei*, *V. owensii*, *V. mediterranei* and *V. coralliilyticus* (Smith *et al.*, 2015). *Vibrio* produced specific enzyme zinc-metalloprotease that caused whitening in coral by disturbing and killing zooxanthellae in living coral that carry out photosynthesis and responsible on coral color. Moreover, this enzyme will disrupt of

coral's tissue in cellular level that causes peel of coral tissue (Sussman *et al.*, 2009).

Total number of coral surveyed was 178 colonies, out of which 24 colonies suffered diseased in July 2018 and almost threefold increase (58 colonies) during 1-month period or August 2018. The WS prevalence shows increasing trend from 13.64% in July to 32.58% in the next month (Figure 5). All *E. lamellosa* colonies in NR Pulau Sempu was in shallow water (2-7 m depth) area, they cannot occupy deeper area due to the substrate is sand. (Hobbs and Frisch, 2010) conducted a research in Christmas Island, Australia, compared disease prevalence in shallow and deeper area, and the result showed that shallow water with high densities of coral colonies tended to have higher coral white syndrome prevalence.

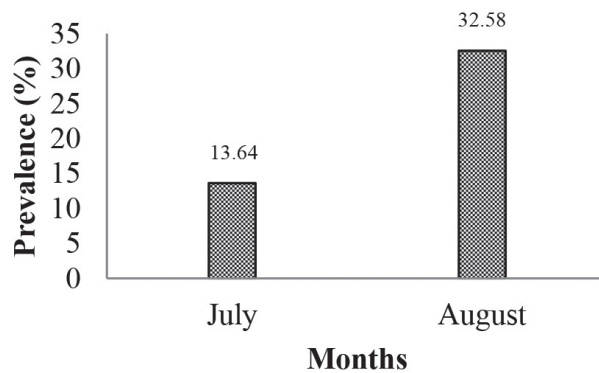


Fig. 5. White syndrome prevalence at NR Pulau Sempu in two months of 2018.

A feasible explanation is that physical and chemical oceanography condition in out-depth water, more light intensity and higher sea surface temperature, encourage the occurrence of white syndrome. Increasing WS prevalence during one-month might be resulted from high coral cover and high density of coral. For instance, the outbreaks of WS on the Great Barrier Reef dependent on high percentage of hard coral cover that are more than fifty percent (Bruno *et al.*, 2007; Willis *et al.*, 2004). Bacterial pathogen such as vibrio can pass through water and spread new disease into others colonies, so the low distance between coral colonies have potential on disease transmission (Hobbs and Frisch, 2010).

Progression rate

White syndrome rate varies among coral colonies, the highest rate was 0.18 cm/ day and the lowest

one was 0.11 cm/day (Figure 6). This tissue loss progression is lower than from the Great Barrier Reef, that WS can result on coral tissue loss, with varying from 1.0 to 124.6 cm² per day (Ainsworth *et al.*, 2007). Tissue loss because WS has been associated by necrotic cell death, where decay progresses on colonies surfaces and associated with long environmental stress or disease.

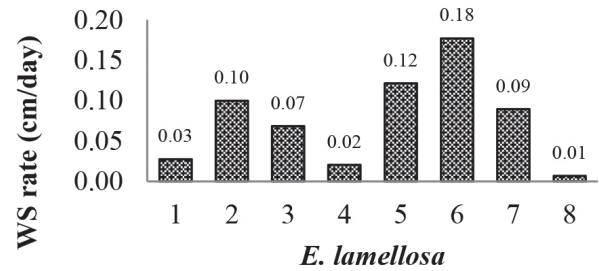


Fig. 6. Progress rate of 8 samples of *E. lamellosa* in NR Pulau Sempu

White syndrome progression also reported from another scleractinian coral such as on *Acropora* and *Montipora* (Aeby *et al.*, 2010; Roff *et al.*, 2011). The progress rate of WS on 3 different *Acroporidae* coral (*Acropora cytherea*, *A. hyacinthus*, *A. clathrata*) in Heron were high, 0 to 1146 cm⁻² week⁻¹, and became the highest WS progress rate in Indo-Pacific (Roff *et al.*, 2011). In addition, other species, *Montipora capitata*, has 3.1% of tissue loss per month in Hawaii water (Aeby *et al.*, 2010). Increased of sea surface temperature can affect on the coral susceptibility as well as increased of number of vibrios. Temperature is one of stressor that common faced by scleractinian coral, because disease always should interact of many stressor (Aeby *et al.*, 2010).

Conclusion

The report of white syndrome in NR Pulau Sempu, is group of coral disease, particularly can increase wide information of massive spreading of this disease. This disease has been resulted on coral mortality and morbidity of family *Acroporidae* in the Caribbean and Pacific. And possibly the number of coral species that are susceptible with kind of disease will be expand in the future. This study shows *E. lamellosa* was infected by WS with high disease prevalence 13-33 % and low progression rate between 0.01 to 0.18 cm/day.

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