

Journal of Biodiversity and Environmental Sciences (JBES) ISSN: 2220-6663 (Print) 2222-3045 (Online) Vol. 10, No. 4, p. 122-128, 2017 http://www.innspub.net

RESEARCH PAPER

OPEN ACCESS

Sediment promote disease in *Scleractinia* coral at sempu island nature reserve malang, East java, Indonesia

Oktiyas Muzaky Luthfi*

Marine Science University of Brawijaya Malang, Jl. Veteran, Malang, Indonesia

Article published on April 30, 2017

Key words: Sendang Biru, Sempu Island, Coral disease, Disease prevalence and Nature reserve

Abstract

Sempu Island was stated as nature reserve in south Java and the most heavily used due to more 100 tuna fishing boats passing in Sendang Biru port just located < 1 km from Sempu Island. Coral reef in Sempu Island can be found only in northern part (Sempu strait) and in small lagoon inside of island (Segara Anakan), about 10 ha total coral reef covered Sempu Island. Live coral cover percentage on Sempu Island was 22.83% that means coral reef being in the pressure. Pressure or threat might be coming from load sedimentation from terrestrial, run off river, sport water tourism and fishing activities. In light of these the aim this study was to quantify prevalence of diseases affecting coral on these reefs. We used SCUBA to survey on five sites with 1x50 belt transect perpendicular with shore line. Coral were identified on genus level and noted compromised health within each 50x50 cm in belt transect area. The result showed that average of coral disease prevalence was 10.67% with the highest prevalence in Teluk Semut 2 (TS 2) with 15.8%. Compromised health also affected on 8 coral families, they were Acroporidae (0.56%); Agariciidae (0.06%); Dendrophyllidae (0.566%); Faviidae (0.99%); Merulinidae (0.06%); Pocilloporidae (0.06%) and Poritidae (6.05%).

*Corresponding Author: Oktiyas Muzaky Luthfi 🖂 omuzakyl@ub.ac.id

Introduction

Coral reef is a complex building that substantially composed by scleractinians coral and others benthic or non-benthic reef biota (Done, 2011). Coral reef function as 'big house' of many other marine biotas for spawning, feeding and nursery ground. Other functions are providing protein (fish), coastal protection and new biochemical product (Moberg and Folke, 1999; coremap.or.id). Long term survey on coral reef in Indonesia during 1989- 2013 that was conducted by Coremap (Coral reef rehabilitation and management program) has concluded only 5% coral reef on excellent condition (live coral percent cover >75%) and 30% on damage condition (live coral percent cover < 25%) (coremap.or.id). The sustainability of them was impacted from climate change, pollution in land and marine, habitat degradation through sedimentation, over fishing and disease (Raymundo et al., 2008; Tomascik, 1997). In long term these impacts will affect on decreasing of coral percent cover and lead phase shift into algal dominance (Bruno et al., 2007).

Coral disease attracting many coral's researchers in the world due to fast emerging of its spread in among species and regions. Coral disease outbreak was reported from Florida Keys and caused mass mortality on 17 species of scleractinian coral and hydrocoral (Milleporids) in 5 months (Bruckner, 2002; Richardson et al., 1998). In Indo-Pacific area, ulcerative white spot was infected on Porites coral in central Philippine (Raymundo et al., 2005), black band disease, brown band disease, and white syndrome was infected on Acroporids coral and caused of mortality on it (Haapkylä et al., 2009). White syndrome also has been reported infected on 15 species of coral in Great Barrier Reef, Australia and syndrome will increase 50-fold during the summer time (Willis *et al.*, 2004).

The emergence of disease usually involving three factors: host, agent and environment. Coral disease begins with change in environmental such as increasing sea water temperature then lead stress in coral host accompanied by decreasing immune system in coral body, so bacteria inside of coral or pathogens from outside will easy multiply inside of coral tissue and become a syndrome or disease (Lesser *et al.*, 2007). Environments play as driver on coral disease such as temperature (Willis *et al.*, 2004; Ben-Haim *et al.*, 2003), water quality (Voss and Richardson, 2006), rainfall (Haapkylä *et al.*, 2005) and sewage (Harvell *et al.*, 2007).

Sempu Island was stated as nature reserve area, the only one in south of Java sea, that had two important tropical marine ecologies i.e. mangrove and coral reef. This island has covered by 10 ha coral reef that only found in southern part and small lagoon inside of the island. Coral reef in northern part faced directly with utilization zone such as fishing port, residential and tourism area. Many activities that was resulted by human in this area produced threat into coral reef's health. The aims of this study were to (i) quantify the coral disease prevalence, (ii) document and analysts compromised health coral to identify potential environment driver to disease, and (iii) survey on coral cover in Sempu Island.

Material and methods

Study site

Sempu Nature Reserve was located in southern of Malang, East Java (122°45' 32" – 112°47' 30" E; 8°25' – 8°30' S). Coral disease survey was conducted in 5 stations, it was representation of coral cover that dominance in northern part of Sempu Island, they were Kondang Buntung (depan) Sta. 1); Teluk Semut 1 (Sta. 2); Teluk Semut 2 (Sta. 3), Watu Mejo 1 (Sta. 4) and Watu Mejo 2 (Sta. 5) during March-April 2014. Coral reef in this area was found between 2-7 m depth, more than 7 or 8m it was sand and mud.

Coral disease survey

Surveys were conducted using belt transect 50x1m in depth of 5m parallel with coastline (English *et al.*, 1997), to facilitate taking data we used 1x1m quadrat transect and divided it into 4 sub-transects 50x50 cm. And then each coral colony within sub-transect was photographed and recorded as healthy or diseased using underwater camera Canon G 11 (Japan) within sub-transect 50x50 cm. Due to logistic reason transect was performed 1 replication each station. Recorded coral colony than counted and identified possible lesion and compromised health using Coral Disease Card (Raymundo *et al.*, 2008; Beeden *et al.*, 2008). We also measured coral colony area using Image-J version 0.50 (NIH, America) to quantify liver coral cover. Physical-chemical oceanography was quantified using AAQ-1183 (Alec, Japan).

Analysis of disease prevalence

Coral disease prevalence calculated using this formula (Raymundo *et al.*, 2008): $P = \frac{a}{A} \times 100\%$ (1) Where, P= coral prevalence; a= diseased coral and A= total colony coral was surveyed.

Analysis of coral cover

Coral cover was calculated according to total area of living coral by total wide area within quadrat transect (50m²), using formula 2 (English *et al.*, 1997; Hill and Wilkinson, 2004). Measuring area of living coral used Image-J version 0.50. and then coral cover categorized by 4 conditions according percent cover of living coral: excellent (>75% coral cover), good (50-75%), fair (25-50%) and poor (< 25%) (Gomez and Yap, 1988).

$$PC = \frac{Ai}{A}x\ 100\% \qquad (2)$$

Where, PC= Percent cover; Ai= living area of coral species i; A= total wide area (50^{2} m)

Result and discussion

About 1,619 coral colonies was encountered in this research, and total 7 types (Fig. 1.) of diseases has been recorded, they were: atramentous necrosis, black band disease, white syndrome, ulcerative white spot, bleaching (focal and non-focal), trematodiasis and growth anomaly. and 4 compromised health coral were recorded: fish bites, drupella scare. pigmentation response and sedimentation. Coral disease was found in all stations research, the highest disease prevalence found on Teluk Semut 2 (15.8%) and the lowest on Watu Mejo 2 (4.08%) (Fig. 2).

Disease also affected 7 families of coral, Acroporidae and Dendrophylliidae were 0.56%, Agariciidae, Merulinidae and Pocilloporidae were 0.06% respectively, and Poritidae had the highest prevalence about 6.05% (Fig. 3). Poritiidae, Faviidae and Acroporidae were found in almost in every station. Growth anomaly was the most prevalent disease that was infected on 215 coral colonies. Another thing was influenced on coral health were predation (fish bites: 6.8%; drupella: 1.75%), pigmentation response 6.5% and sedimentation was 26.2%.



Fig. 1. Map showing the study sites where survey on coral disease was conducted in Sempu Island, East Java.

124 | Asrar *et al*.



Fig. 2. All types of disease were infected on coral in Sempu Island.

Black band disease (BBD) (Fig. 3.) was detected only in Watu Mejo 1 (n=1), it was infected on branching Porites coral. BBD characterized by black or dark brown annular band lesion at interface of coral tissue and make obvious border among living coral and dead coral. *Halofolliculina corallasia* is cyanobacteria that infected the coral and made a band (Beeden *et al.*, 2008; Page and Willis, 2006). The number of BBD will increase if the reef has high sedimentation load or close to sewage outflow. Nutrient enrichment also become factor can enhance the BBD progression (Kuta and Richardson, 2002; Voss and Richardson, 2006). Atramentous necrosis/ AtN (n=2) was found affected on folious Montiporids with multifocal bare skeleton that covered by a black sulphurous deposit. Ulcerative white spots/ UWS (n=7) were found in this area may be an early stage of AtN (Beeden *et al.*, 2008). AtN had strong correlation with particulate organic carbon (POC) attribute from terrestrial run off during rainfall season. Rain fall also decreased water salinity and this combination may have reduced immune system in coral body and increased virulence as causative agent (Haapkylä *et al.*, 2005).

Growth anomaly/ GA (n=59) was found in all station and affected on Poritids coral. GA or hyperplasia or tumour is focal or multifocal deformation of coral skeleton and may be caused by presence of organism inside of skeleton or chaotic polyp development (Beeden *et al.*, 2008). GA in Porites was known as PGA (Porites Growth Anomaly) where this incidence was increased may result from runoff sediment from land and lower water quality (Haapkylä *et al.*, 2011). GAs have ability to transmit in other Porites, during seven weeks GA two other Porites coral was infected through direct contact (McClanahan *et al.*, 2009; Kaczmarsky and Richardson, 2007).



Fig. 3. Coral disease prevalence in all station. KB(D): Kondang Buntung (depan); (TS1) Teluk Semut 1; (TS 2) Teluk Semut 2; (WM 1): Watu Mejo 1 and (WM 2): Watu Mejo 2.

125 | Asrar et al.

Mean coral disease prevalence on Sempu Island was 10.67%, it was relative same with other regions in Indo-Pacific. In the Philippine mean coral disease prevalence was 8.3% (Raymundo et al., 2005), in Great Barrier Reef was 10.7% (Willis et al., 2004), and Wakatobi Marine National Park (WMNP), Indonesia was 0.57%. Disease prevalence has associated with environmental factors such as sewage effluent (Kaczmarsky et al., 2005), rainfall and sedimentation (Kuta and Richardson, 2002; Haapkylä et al., 2011), host abundance (Myers and Raymundo, 2009), climate change (Harvell et al., 2007) and human population (Mora, 2008). It was clear why in Sulawesi coral disease prevalence has lower than other, because WMNP located on remote island far from human disturbance and environment relative still in good condition.

North part of Sempu Island is hilly land where recent condition has been change from natural forest to agriculture area. Terrestrial runoff loads nitrate (NO³), ammonia (NH₃), ammonium (NH⁴⁺) and phosphate (PO₄³⁻) into sea water and increased number of algae in water (Scheffer *et al.*, 2001). Algae was competitor for coral and can transmit disease (Hughes *et al.*, 1999). High nutrient in a water also can decrease respiration and calcification rates of coral (Fabricius, 2005). Coral in this area also threated by sedimentation, about 131 colonies of coral was stress because sediment covered its skeleton. Compromised health prevalence that caused by sediment was 32%.

Sediment also influence the coral cover, massive coral from Poritiids and Faviids (Raymundo *et al.*, 2005). Coral in Sempu Island was dominated by massive Porites and Goniastrea that had capability to clean sediment from their skeleton using tentacle. In other way coral also produce mucus to prevent sediment fall inside their polyp. This condition suggested be main reason the coverage of living coral was 22.83%.

Table 1.	Physical.	chemical	oceanograph	ıv in Semı	ou Island i	nature reserve	during research.
	,,		· · · · · · · · · · · · · · · · · · ·				

	Parameters							
No	Stations	Temp. (ºC)	Salinities (‰)	DO (mg/l)	pН	Depth. (m)	Visibility (m)	
1	KB (D)	27.42	34.2	9.31	8.56	1-8	12.7	
2	WM 1	27.25	34.1	8.92	8.50	1-8	8.97	
3	WM 2	28.41	34.09	9.17	8.33	1-8	8.29	
4	TS 1	27.95	34.12	9.41	8.48	1-8	10.41	
5	TS 2	27.83	34.06	9.36	8.35	1-8	9.31	



Fig. 4. Coral disease prevalence that was infected on 7 families scleractinian coral in Sempu Island nature reserve.

126 | Asrar et al.

Conclusion

Sempu Island Nature reserve was threat by sedimentation and anthropogenic factors. Sediment will cause physically stress in coral also became source of pathogen. High sedimentation will shade the water and disturbing photosynthetic on algal symbiont, in other case sediment will covered the coral polyp and make them die. Combination stress in host, increase pathogen in sea water and decrease of environment factor will cause of emergence of disease. To address what the specific pathogen that carried out by sediment, it need be cultured in the next research. High coral disease prevalence in Sempu Island suggested will increase year by year if the management, researchers and government also all stakeholders not sit together to make a protocol for coral reef conservation.

Acknowledgement

We would like to thanks to Coral Reef Study Center (Acropora) for help taking data in the field. This research is part of PUPT research that found by Ministry of Higher Education of ROI through DIPA Universitas Brawijaya Number: 023.04.2.414989/2014.

References

Beeden R, Willis BL, Raymundo LJ, Page CA, Weil E. 2008. Underwater Cards for Assessing Coral Health on Indo-Pacific Reef. CRTR Program Project Executing Agency, Center for Marine Studies. The University of Queensland. Australia.

Ben-Haim Y, Zicherman-Keren M, Rosenberg E. 2003. Temperature-regulated bleaching and lysis of the coral *Pocillopora damicornis* by the novel pathogen *Vibrio coralliilyticus*. Applied and Environmental Microbiology **69(7)**, 4236-4242.

Bruckner AW. 2002. Priorities for effective management of coral diseases. NOAA Technical Memorandum NMFS-OPR **22**, 54 pp.

Bruno JF, Selig ER, Casey KS, Page CA, Willis BL, Harvell CD, Hugh S, Melendy AM. 2007. Thermal stress and coral cover as drivers of coral disease outbreaks. PLo S biology **5(6)**, e124.

Done T. 2011. Corals: environmental controls on growth. Encyclopedia of Modern Coral Reefs: Structure, Form and Process 281-293.

English S, Wilkinson C, Baker V. 1997. Survey Manual for Tropical Marine Resources. Australia Marine Science Project Living Coastal Resources. Australia.

Fabricius KE. 2005. Effects of terrestrial runoff on the ecology of corals and coral reefs: review and synthesis. Marine pollution bulletin **50(2)**, 125-146.

Gomez ED, Yap HT. 1988. Monitoring reef condition. In: Kenchington, R.A., Hudson, B.E.T. (Eds.), Coral reef management handbook. UNESCO regional office for science and technology for Southeast Asia (ROSTSEA), Jakarta pp. 171-178.

Haapkylä J, Unsworth RK, Flavell M, Bourne DG, Schaffelke B, Willis BL. 2011. Seasonal rainfall and runoff promote coral disease on an inshore reef. PLoS One **6(2)**, e16893.

Haapkylä J, Unsworth RK, Seymour AS, Melbourne-Thomas J, Flavell M, Willis BL, Smith DJ. 2009. Spatio-temporal coral disease dynamics in the Wakatobi Marine National Park, south-east Sulawesi, Indonesia. Diseases of Aquatic Organisms 87(1), 105.

Harvell D, Eric JD, Susan M, Eugene R, Laurie R, Garriet S, Ernesto W, Willis B. 2007. Coral disease, environmental drivers, and the balance between coral and microbial associates. Oceanography **20**, 172-195.

Hill J, Wilkinson C. 2004. Methods for ecological monitoring of coral reefs. Australian Institute of Marine Science, Townsville 117.

http://coremap.or.id/Kondisi-TK

Hughes T, Szmant AM, Steneck R, Carpenter R, Miller S. 1999. Algal blooms on coral reefs: what are the causes? Limnology and Oceanography **44(6)**, 1583-1586. **Kaczmarsky L, Richardson L.** 2007. Transmission of growth anomalies between Indo-Pacific Porites corals. Journal of Invertebrate Pathology **94(3)**, 218-221.

Kaczmarsky LT, Draud M, Williams EH. 2005. Is there a relationship between proximity to sewage effluent and the prevalence of coral disease. Caribb J Sci **41(1)**, 124-137.

Kuta K, Richardson L. 2002. Ecological aspects of black band disease of corals: relationships between disease incidence and environmental factors. Coral Reefs **21(4)**, 393-398.

Lesser MP, Bythell JC, Gates RD, Johnstone RW, Hoegh-Guldberg O. 2007. Are infectious diseases really killing corals? Alternative interpretations of the experimental and ecological data. Journal of experimental marine biology and ecology **346(1)**, 36-44.

McClanahan TIM, Weil E, Maina J. 2009. Strong relationship between coral bleaching and growth anomalies in massive Porites. Global Change Biology **15(7)**, 1804-1816.

Moberg F, Folke C. 1999. Ecological goods and services of coral reef ecosystems. Ecological economics **29(2)**, 215-233.

Mora C. 2008. A clear human footprint in the coral reefs of the Caribbean. Proceedings of the Royal Society of London B: Biological Sciences **275(1636)**, 767-773.

Myers RL, Raymundo LJ. 2009. Coral disease in Micronesian reefs: a link between disease prevalence and host abundance. Diseases of aquatic organisms **87(1-2)**, 97-104.

Page C, Willis B. 2006. Distribution, host range and large-scale spatial variability in black band disease prevalence on the Great Barrier Reef, Australia. Diseases of aquatic organisms **69**, 41-51. Raymundo LJ, Couch CS, Harvell CD, Raymundo J, Bruckner AW, Work TM, Ernesto Weil, Cheryl M, Woodley, Eric Jordan-dahlgren, Bette L, Willis, Yui Sato and Aeby GS. 2008. Coral Disease Handbook Guidelines for Assessment, Monitoring and Management.

Raymundo LJ, Couch CS, Harvell CD. 2008. Coral Disease Handbook: Guidelines for Assessment, Monitoring and Management. Coral Reef Targeted Research and Capacity Building for Management Program. The University of Queensland Australia.

Raymundo LJ, Rosell KB, Reboton CT, Kaczmarsky L. 2005. Coral diseases on Philippine reefs: genus Porites is a dominant host. Diseases of aquatic organisms **64(3)**, 181-191.

Richardson LL, Goldberg WM, Carlton RG, Halas JC. 1998. Coral disease outbreak in the Florida Keys: plague type II. Rev. Biol. Trop, 46 (Supl 5), 187-198.

Scheffer M, Carpenter S, Foley JA, Folke C, Walker B. 2001. Catastrophic shifts in ecosystems. Nature **413(6856)**, 591-596.

Tomascik T. 1997. The ecology of the Indonesian seas. Oxford University Press.

Voss JD, Richardson L. 2006. Nutrient enrichment enhances black band disease progression in corals. Coral Reefs **25(4)**, 569-576.

Voss JD, Richardson L. 2006. Nutrient enrichment enhances black band disease progression in corals. Coral Reefs **25(4)**, 569-576.

Willis BL, Page CA, Dinsdale EA. 2004.Coral disease on the Great Barrier Reef. In Coral health and disease (pp. 69-104).Springer Berlin Heidelberg.