Can reef sediments serve as a stony coral tissue loss disease reservoir?

¿Pueden los sedimentos de los arrecifes servir como un reservorio de enfermedades por pérdida de tejido de coral pétreo?

Les sédiments des récifs peuvent-ils servir de réservoir à la maladie de la perte de tissu corallien rocheux?

STUDIVAN MS[1,2], ROSSIN AM[3], RUBIN E[1,2], SODERBERG N[1,2], HOLSTEIN DM[3], ENOCHS IC[2]
[1]*Cooperative Institute for Marine & Atmospheric Studies, University of Miami Rosenstiel School of Marine & Atmospheric Science, 4600 Rickenbacker Causeway, Miami, FL 33149*
[2]*Atlantic Oceanographic & Meteorological Laboratory, National Oceanic & Atmospheric Administration, 4301 Rickenbacker Causeway, Miami, FL 33149*
[3]*Louisiana State University, 1002-Y Energy, Coast & Environment Building, Baton Rouge, LA 70803*
michael.studivan@noaa.gov, aross68@lsu.edu, ewelina.rubin@noaa.gov, nash.soderberg@noaa.gov, dholstein1@lsu.edu, ian.enochs@noaa.gov

EXTENDED ABSTRACT

Since its first appearance off Miami in 2014 following the dredging of Government Cut, stony coral tissue loss disease (SCTLD) has spread rapidly throughout Florida's coral reef tract & to numerous Caribbean territories. It has been shown through transmission experiments & hydrodynamic modeling that the disease is waterborne & highly infectious at both local & regional scales. We conducted a high-replication transmission experiment to determine whether reef sediments can also serve as an SCTLD reservoir. Coral fragments in independent 0.5 L vessels with flow-through seawater were exposed to disease-inoculated reef sediments using entire infected colonies (batch inoculation) or ~5 cm^2 coral fragments (individual inoculation). We identified that reef sediments were able to transmit SCTLD to fragments of the species *Orbicella faveolata* & *Montastraea cavernosa*, albeit at lower rates as direct contact with diseased coral tissue. Transmission rates differed between species & treatments, with the initial onset of SCTLD lesions occurring at 10.3±2.8 & 12.2±1.9 d following exposure to batch diseased sediments. Exposure to individual diseased sediments, however, resulted in disease transmission within 22 h for both species, suggesting temporal/spatial variation in distribution of SCTLD pathogens in sediments. A three-way ANOVA indicated that the time to onset of disease was significantly different between species ($p < 0.018$), among coral colonies ($p < 0.039$), and among disease treatments ($p < 0.001$), with a significant species:treatment effect ($p < 0.046$). Signs of SCTLD infection were similar among disease treatments but varied between species. Early symptoms including discharge of mesenterial filaments, tissue swelling, and tissue paling were common, ranging from ~ 24 h to several days prior to visible lesion formation. Characteristic white lesions and tissue loss (Aeby et al., 2019; Landsberg et al., 2020) were more prevalent in *O. faveolata*, with rapid progression across entire fragments within a few days. Lesions were often less defined in *M. cavernosa* fragments, with smaller patches of necrosis and tissue loss that appeared to progress slowly over several days to a week. In both disease-inoculated sediment treatments, lesion formation was more likely to occur on the vertical sides of the fragment, where there was close contact between coral tissue and the sediments. In these cases, tissue loss was more likely to occur along the fragment margins, often accompanied by excess mucus production in *M. cavernosa*.

Histological examination confirmed the presence of SCTLD across all disease treatments, with gross differences in coral tissues exposed to SCTLD versus those in the healthy sediment treatment. Signs associated with SCTLD (e.g., symbiont exocytosis, body wall breakage, liquifying necrosis; Landsberg et al., 2020; Meiling et al., 2021) were confirmed more commonly in diseased versus healthy treatments, with evidence of subtle variation between species & microbial community profiling using the 16*S V4* region identified 30 bacterial genera significantly differentially abundant among coral conditions, including 16 genera for which the abundance was higher in tissue loss-associated samples. Compared to previous SCTLD microbial studies, we found 15 out the 25 bacterial taxa identified by Becker and colleagues (2021) to be present in our sediment samples, and found several pathogenic taxa in association with disease, including *Vibrio*, *Shimia*, and Rhodobacterales. This experiment demonstrated that reef sediments can in fact transmit SCTLD, and therefore act as a disease vector. The results of this study also pose the hypothesis that sediments may serve as disease reservoirs or vectors at local scales under natural conditions (i.e., in the absence of human disturbance), indicating a critical need to understand the roles of coastal development activities (e.g. port dredging & beach renourishment) on SCTLD transmission, as well as to evaluate management actions to mitigate further spread of SCTLD throughout Caribbean reefs. Sediments may have played a critical role in the uncharacteristically rapid spread of SCTLD throughout Florida's Coral Reef, as well as the persistence of the disease for the past seven years. Therefore, continued disease prediction and mitigation approaches should consider sediments both as a SCTLD vector and potential reservoir. Treatment methods should also be evaluated to reduce disease spread due to natural sediment reservoirs, and following coastal development activities that transport sediments. Effective and comprehensive management strategies in the context of the ongoing SCTLD outbreak are contingent on a holistic understanding of modes of transmission and targeted actions to reduce further disease spread; here we recommend that

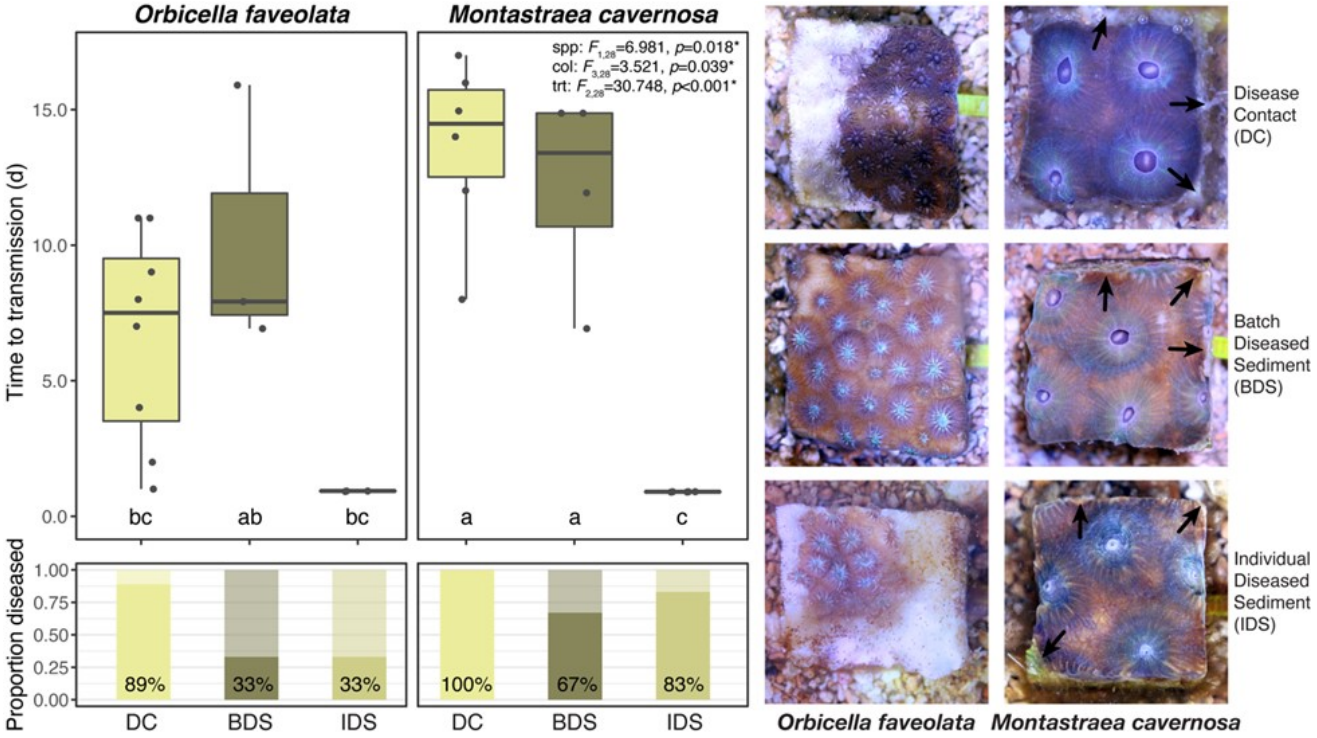

Figure 1. Mean time to transmission (initial observation of lesions) and transmission rates (proportion diseased) by species and disease treatments, with representative photographs. Error bars denote standard error of the mean, test statistics indicate the results of three-way ANOVA among species, colonies, and treatments, and different letters denote significant pairwise differences as generated by Tukey's test. Arrows in photo panel indicate excess mucus production (top right), tissue necrosis (center right), and tissue retraction (bottom right) observed more commonly in *M. cavernosa* fragments.

consideration be given to the impacts that sediments and associated development activities may have on coral reefs in Florida and beyond.

KEYWORDS: disease transmission, disease vector, disease reservoir, sedimentation, microbial communities

LITERATURE CITED

Aeby, G. S., Ushijima, B., Campbell, J. E., Jones, S., Williams, G. J., Meyer, J. L., Häse, C., & Paul, V. J. (2019). Pathogenesis of a tissue loss disease affecting multiple species of corals along the Florida Reef Tract. *Frontiers in Marine Science*, *6*(November), 1–18. https://doi.org/10.3389/fmars.2019.00678

Becker, C. C., Brandt, M., Miller, C. A., & Apprill, A. (2021). Microbial bioindicators of Stony Coral Tissue Loss Disease identified in corals and overlying waters using a rapid field-based sequencing approach. *Environmental Microbiology*. https://doi.org/10.1111/1462-2920.15718

Landsberg, J. H., Kiryu, Y., Peters, E. C., Wilson, P. W., Perry, N., Waters, Y., Maxwell, K. E., Huebner, L. K., & Work, T. M. (2020). Stony coral tissue loss disease in Florida is associated with disruption of host–zooxanthellae physiology. *Frontiers in Marine Science*, *7*, 1090. https://doi.org/10.3389/fmars.2020.576013

Meiling, S. S., Muller, E. M., Lasseigne, D., Rossin, A., Veglia, A. J., MacKnight, N., Dimos, B., Huntley, N., Correa, A. M. S., Smith, T. B., Holstein, D. M., Mydlarz, L. D., Apprill, A., Brandt, M. E., & Neely, K. L. (2021). Variable species responses to experimental stony coral tissue loss disease (SCTLD) exposure. *Frontiers in Marine Science*, *8*(April), 670829. https://doi.org/10.3389/fmars.2021.670829