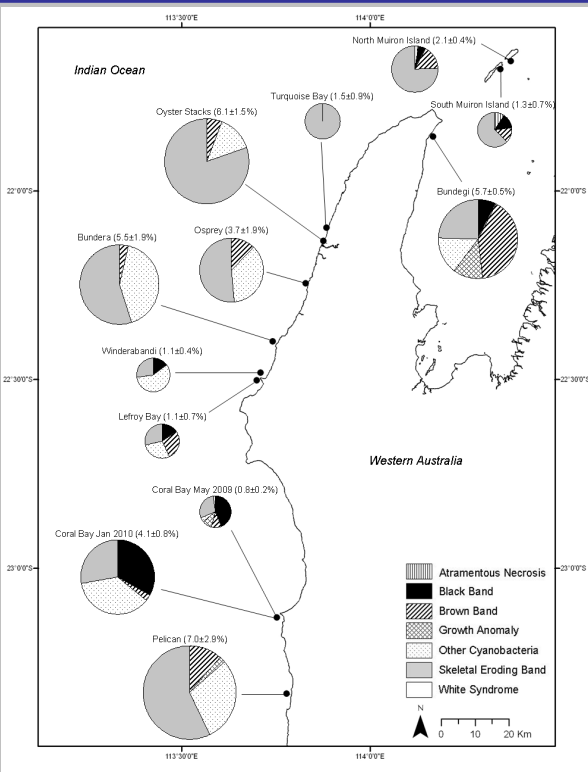


Distribution & drivers of coral disease

of coral disease at Ningaloo Reef

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Disease has emerged as a potential driver of change on coral reefs, particularly among corals. Increasing levels of anthropogenic disturbance on coral reefs and climatic warming highlight the need for baseline information on disease occurrence and factors influencing coral susceptibility to disease, particularly in the Indian Ocean where little coral disease research has been undertaken. This study investigated the prevalence and potential drivers of coral disease across Ningaloo Reef on the Western Australian coast.

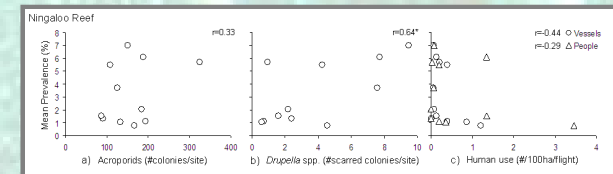
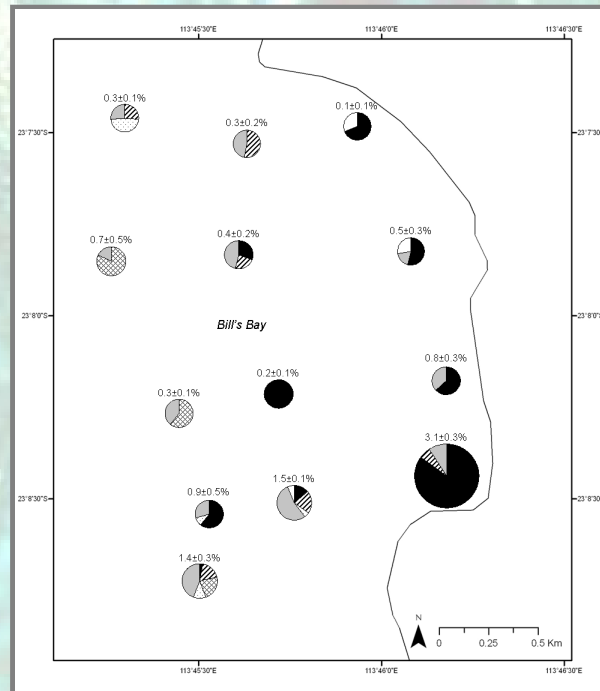


Figure 3. Relationship between disease and a) density of *Acropora* colonies, b) number of *Drupella* feeding scars and c) human use. The density of *Acropora* colonies and *Drupella* scars was assessed during disease surveys, Human usage data was collected from aerial surveys undertaken two to four times annually between 2005 and 2009. A positive relationship was detected between the prevalence of disease and coral colonies exhibiting *Drupella* spp. feeding scars ($F_{1,9} = 6.24, P = 0.033, r = 0.64$). Prevalence of coral disease was not strongly correlated to the abundance of acroporid corals, nor the number of vessels or people using the reef area. A forward stepwise model, which explained 61% of the spatial variation in disease across Ningaloo Reef, did however include abundance of *Acropora* colonies, and density of people in addition to number of *Drupella* spp. feeding scars ($F_{3,7} = 6.20, P = 0.22$).

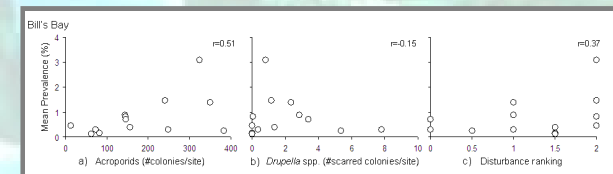


Figure 4. Relationships between disease and a) density of *Acropora* colonies, b) number of *Drupella* feeding scars and c) impact of anoxic events at Bill's Bay. The density of *Acropora* colonies and *Drupella* scars was assessed during disease surveys, impact of anoxic events based on intensity of coral bleaching at each site in 1989, 2002, 2005, and 2008. There are no significant relationships between disease prevalence and *Acropora* density, *Drupella* scars or impacts from anoxic events. However, a forward stepwise model which includes *Acropora* density and impacts of anoxic events explained 54% of the spatial variation in disease prevalence ($F_{2,10} = 7.94, P = 0.009, r^2 = 0.54$).

Figure 2. Disease prevalence at 13 sites in Bill's Bay during May 2009. Sites with the highest percentage of colonies infected occurred in the southeastern corner of the bay and lowest rates of disease prevalence were found in the mid to northern sections of the bay ($F_{12,26} = 3.75, p = 0.023$). Skeletal eroding band was the disease observed most frequently, infecting corals at 85% of sites in Bill's Bay.

Overall, coral disease at Ningaloo is similar to that on other Indo-Pacific reefs with minimal anthropogenic stressors. This study supports previous research that disturbance reduces disease resilience in corals and emphasizes the importance of managing human activities to minimize cumulative stress upon the reef and the effects of disease.



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Figure 1. Prevalence of coral disease at 11 sites along the Ningaloo coastline. During January 2010 all coral colonies within three 20 x2m transects, at each site, were inspected for disease. Overall 2.3% of coral colonies showed signs of disease, although disease prevalence varied significantly among sites, ($F_{10,34} = 2.41, p = 0.028$). The percentage of colonies infected was greatest at Pelican Sanctuary, Oyster Stacks, Bundegi and Bundera and lowest at Lefroy Bay. The most common disease observed along Ningaloo was skeletal eroding band (0.99% of colonies), although other cyanobacterial infections, black and brown band diseases were present at similar densities.

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