Ecology and Biology of Coral Disease on the Great Barrier Reef

Thesis submitted by
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THESIS DEDICATION

I dedicate this thesis to my mother, Christine May Page, and to my daughter, Xian Christina Neale. Mum, I agree this thesis should have been completed before now.

ABSTRACT

This study examines the implications of disease for the structure and resilience of Great Barrier Reef (GBR) coral assemblages. Annual disease surveys in the northern and central sections of the GBR between 2004 and 2006 indicate that disease is ubiquitous and persistent throughout much of the GBR. Seven diseases commonly affect a wide range of anthozoan taxa, albeit at relatively low levels of prevalence (~ 3% of scleractinian corals). Nonetheless, values recorded for disease prevalence and for rates of tissue loss and mortality indicate that the acute impacts of diseases such as black band disease (BBD) and skeletal eroding band (SEB) can be similar to those of diseases that have caused significant declines in Caribbean coral populations, highlighting the significant role disease is likely to play in structuring at least some GBR coral assemblages.

Two families, the Pocilloporidae and Acroporidae, consistently ranked highest in a disease susceptibility hierarchy determined for GBR corals. Consistency in spatial patterns in the prevalence of BBD coupled with consistency in disease susceptibility hierarchies over cross- and long-shelf gradients in turbidity, wave action, and water quality, indicate that these environmental factors are not the primary drivers of disease occurrence on the GBR. Instead, concordance in family rankings for susceptibility to disease and susceptibility to other factors that compromise coral health (thermal bleaching, injury from predators, and interactions with macro-algae) suggests that fast growing, branching morphologies of acroporid and pocilloporid corals, and their resultant high abundance in many Indo-Pacific coral assemblages, enhance the vulnerability of these families to a diversity of pathogens, in addition to a range of other biological and physical stressors.

Experimental studies demonstrated the important role that injury is likely to play in the development of SEB. *Halofolliculina corallasia*, the putative pathogen of SEB, rapidly colonised artificial wounds, however, despite initial increases in ciliate densities on wounds, ciliates failed to become pathogenic and cause additional tissue mortality on any of the three coral species tested experimentally. In addition to injury, environmental factors that compromise coral health or the presence of other microbial agents may be required before ciliates become pathogenic. In combination with correlations between family rankings for susceptibility to disease and susceptibility to other factors that compromise coral health, these results highlight the need for management strategies that limit activities and factors that compromise coral health, in order to minimise the spread and transmission of coral diseases.

Partial mortality over three months caused by the three diseases examined in this study ranged from 85.7 ± 1.6% for BBD to 12.9 ± 1.7% for SEB, while rates of whole colony mortality over two years ranged from 84% for BBD to 39% for SEB. Tissue loss was not associated with growth anomalies (GAs) in this study. The combined partial and whole colony mortality caused by BBD was at least two-fold higher than mortality caused by SEB in northern GBR coral populations, however the seven-fold higher prevalence of SEB, combined with further reductions in the growth rates and reproductive output of surviving portions of *A. muricata* colonies, indicate that SEB could have greater fitness consequences for GBR *Acropora* populations than BBD. The potential for failure of gametogenesis in a large proportion of *Acropora* colonies with GAs, indicates that the impact of a chronic outbreak of GAs on the fitness of Indo-Pacific coral populations could rival those of both SEB and BBD.

Sub-lethal impacts of these diseases on growth and reproduction of *Acropora muricata* also varied, suggesting that this species can vary its resource allocation

strategy to maximise contributions to future generations in response to differing levels of disease virulence. Continued investment of resources in colony growth and reproduction in colonies with BBD may represent an attempt to maximise short-term fitness, given rapid rates of tissue loss and the high probability of dying. In contrast, colonies with GAs or slower progressing diseases such as SEB may divert resources from physiological processes not directly linked to colony survival in response to the greater likelihood that some portion of the colony will survive and recover.

Predicted increases in disease with ocean warming and anthropogenic impacts pose the greatest threat to the persistence of the highly susceptible acroporid and pocilloporid corals in this region, taking into consideration both the lethal and sub-lethal impacts of diseases highlighted in this study and the double jeopardy represented by their high vulnerability to other disturbances including cyclones, bleaching and crown-of thorns outbreaks on the GBR.

STATEMENT OF ACCESS

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STATEMENT OF SOURCES

DECLARATION

I declare that this thesis is my own work and has not been submitted in any form for another degree or diploma at any university or other institution of tertiary education. Information derived from the published or unpublished work of others has been acknowledged in the text and a list of references is given.

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