Linking Heat Stress Cellular Response to Immortality in Hawaiian Corals

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This project aims to link the cellular heat stress response of Montipora capitata to its biological immortality. The amortality of this species is attributed to alternative methods of telomere maintenance. Telomeres are the non-coding, repeated sequence caps at the ends of chromosomes that protect the genome as cells divide. Telomere shortening is the cause of cellular aging and eventual apoptosis (cellular death) in non-ammortal organisms. Telomerase is an enzyme that many cancers use to maintain amortality and telomerase RNA subunits have been found in coral tissues, however, if this enzyme is the mechanism used by corals, it would suggest that they eventually become cancerous, which they do not. Corals were heat stressed on a 5 day heat stress curve and confocal laser microscopy and tissue sampling was performed before and after heat stress. Confocal laser microscopy and visual cues were used to assess bleaching and chlorophyll map disruption as a measure of heat stress. DNA was extracted from the tissues to assess telomere length before and after heat stress and RNA was extracted to assess concentrations and correlation of the two telomersase subunits. Telomeres significantly lengthened in visibly bleached corals and when telomere length was compared to chlorophyll a fluorescence, there was a strong negative trend between chlorophyll fluorescence and telomere length. This effectively links Motipora capitata's methods of telomere maintenance to their cellular heat stress response. Although telomerease subunits were significantly concentrated and correlated in the ambient temperature tissue samples, in the heat stressed samples, telomerase subunits were no longer correlated suggesting that telomerase is not the mechanism that is utilized for telomere maintenance.

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