Post-transcriptional regulation of the Cell Cycle in the Florida Red Tide dinoflagellate, *Karenia brevis*

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The red tide dinoflagellate, *Karenia brevis*, is responsible for harmful algal blooms in the Gulf of Mexico that cause extensive fish fills, marine mammal mortalities, and human illness on a nearly annual basis. The molecular mechanisms controlling the cell cycle in this dinoflagellate are important because vegetative cell division plays a central role in bloom development. In dinoflagellates, cell division is characterized by novel adaptations including the presence of permanently condensed chromatin throughout the cell cycle, a lack of nucleosomes to aid in chromatin packaging, a permanently intact nuclear envelope, and an extranuclear mitotic spindle that interacts with chromosomes via cytoplasmic channels. Microarray and qPCR studies further suggest that, unlike typical eukaryotes, dinoflagellate cell cycle genes are not regulated at the transcriptional level (Van Dolah et al., 2007). In the current project, we investigated the expression of proliferating cell nuclear antigen (PCNA), an S-phase gene whose expression is typically activated by the E2F transcription factor at the restriction point, which regulates the entry into S-phase. Immunolocalization with anti-*K .brevis* PCNA showed that although PCNA transcripts are constitutively present, PCNA protein is expressed only in cells actively traversing the cell cycle. However, PCNA is present in the nucleus well before S-phase is apparent by flow cytometry. These results suggest that PCNA protein expression is activated by translation initiation at some point prior to S-phase entry. We are currently using immunolocalization of pre-initiation complex MCM proteins to identify precisely when the restriction point occurs, in order to investigate

how *K. brevis* regulates S-phase entry in the absence of transcriptional control of S-phase genes.

Funding Source: Center for Coastal Environmental Health and Biomolecular Research, National Oceanic and Atmospheric Administration, Charleston, SC