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Roles of H3v in Trypanosoma brucei

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Abstract

African trypanosomiasis, or sleeping sickness, is caused by the parasite *Trypanosoma brucei*, a protozoan that affects the central nervous system. This unicellular eukaryote can be transmitted to mammals by the bite of a tsetse fly. *T. brucei* evades the host's adaptive immune response by carrying out antigenic variation of its protective coat of Variant Surface Glycoprotein which allows the infection to persist and be further transmitted. *T. brucei* lacks the sequence-specific transcription factors found in other eukaryotes, thus chromatin structures at PTU (polycistronic transcription units) boundaries are thought to play important roles in control of gene expression. This paper focuses on identifying and characterizing the roles of the chromatin mark H3v in several cellular processes. H3v plays a key role in DNA replication and transcription, thus studying it will be very beneficial for progress in research about trypanosomes. This research project will focus on creating a library of 63 single point mutations in H3v by site-directed PCR mutagenesis, then cloning them into a vector, transforming the ligated products into *E. coli* competent cells, and finally introducing this H3v mutant library into a trypanosome strain. Mutations will be examined for DNA replication, transcription, and antigenic variation. The non-functioning mutants will be identified to understand the roles of H3v in DNA replication and transcription.