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Jennifer Kungle
Cleveland State University

John Sabljic
Cleveland State University

Tia Nikova
Cleveland State University

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Characterization of the detailed interaction interface between T. brucei telomere proteins TRF and TIF2

College of Sciences and Health Professions

Student Researchers: Jennifer Kungle, John Sabljic, and Tia Nikova

Faculty Advisor: Bibo Li

Abstract

The protozoan parasite *Trypanosoma brucei* causes fatal African trypanosomiasis in humans and nagana in cattle. *T. brucei* switches its variant surface glycoproteins (VSGs) inside the mammalian host, evading the host immune response. VSGs are expressed monoallelically from subtelomeric expression sites, and telomere proteins regulate VSGs.

We previously found that telomere protein *TbTIF2* interacts with *TbTRF* (TTAGGG-repeat binding factor) and plays important roles in VSG switching regulation. *TbTRF* maintains the telomere terminal structure. *TbTIF2* is essential for subtelomeric integrity and suppresses VSG switching by inhibiting subtelomeric gene conversion. Depletion of *TbTIF2* decreases *TbTRF* protein level. We hypothesize that *TbTRF-TbTIF2* interaction is essential for maintaining *TbTRF* protein level. We test this hypothesis by mapping the interaction between *TbTRF* and *TbTIF2*. *TbTRF* has both N-terminal TRF Homology (TRFH) and C-terminal Myb domains. *TbTRFH* contains seven helices and interacts with the N-terminus of *TbTIF2* (aa 2-190). We found that deleting either of the first two helices in *TbTRFH* abolishes its interaction with *TbTIF2*. Currently we are generating deletion and point mutations within the first two helices of *TbTRFH*, which will be tested for their ability to interact with *TbTIF2* to determine the key residues in *TbTRFH* that are required for interacting with *TbTIF2*.

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