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A novel transcription factor of Entamoebahistolytica EhURE1BP, a homologue to mammalian p100

Mercedes Calixto*, Monica Romero, Esther Orozco, Mario Rodríguez
Cinvestav, Mexico

The phenotypic differences that distinguish the various kinds of cells in a higher eukaryote are largely due to differences in the expression of genes that code for proteins, the expression of these proteins is controlled mainly at transcriptional level. The initiation of mRNA synthesis is a complex and highly regulated process that requires the assembly of general transcription factors and RNA pol II into pre-initiation complex at the core promoter, which is defined as the minimal DNA region that is sufficient to direct low levels of basal transcription. Very little is known about the factors and *cis*-elements involved in the mechanism of transcription of *Entamoeba histolytica*, the protozoan parasite responsible for the human amibiasis. Thus understanding the mechanism of transcriptional regulation of this microorganism is critical in deciphering its molecular modes of gene expression of proteins involved in development, evasion of immune responses and proteins involved in virulence.

The URE1 *cis*-element present in the *hgl5* and *EhrabB* gene promoters activates the transcription of these genes, whose proteins participates in *E. histolytica* pathogenicity.

In this work we identified and characterized the protein that binds to the URE1 element (EhURE1BP). This is a protein of 96 KDa; containing two SNase domains, a Tudor motif and one nuclear localization signal. EhURE1BP has 43% homology to the p100 multifunctional protein. The Ehure1BP encoding gene was cloned in the pGEX-6P-1 vector and the recombinant protein was expressed in *E. coli*. Immunofluorescence assays using antibodies against the recombinant protein showed that EhURE1BP is located in cytoplasm and nuclei. The purified recombinant protein is able of bind to the URE1 sequence in a specific manner and the inhibition of the EhURE1BP expression by siRNA diminishes the phagocytosis of the parasite.

Keywords: transcription factors, Entamoeba histolytica, EhrabB, phagocytosis