Spliced leader RNA and 5S rRNA genes in *Herpetomonas* spp. are genetically linked

Serap Aksoy

Yale University MacArthur Center for Molecular Parasitology, Yale University School of Medicine, Department of Internal Medicine, PO Box 3333 Cedar Street, New Haven, CT 06510, USA

Submitted January 9, 1992

EMBL accession no. X62331

In Trypanosomatidae, the 5'-ends of mature messages contain a conserved nucleotide segment which is not contiguously synthesized with the protein coding sequences. The donor of the 5'-end conserved sequence is a small non-polyadenylated transcript, Spliced Leader RNA (SL RNA) (1). The 5'-end 39 nt segment of the SL RNA is *trans*-spliced to all messages post-transcriptionally. SL RNA gene for the protozoan parasite *Herpetomonas samuelpessoai* has been cloned and sequenced. The 600-bp *Hind*III restriction enzyme fragment was identified as the SL RNA coding segment based on its hybridization to *T. brucei* SL-39-mer-specific oligonucleotide probes in genomic Southern blots (data not shown). The DNA sequence analysis of this fragment shows that it can code for a 98 nt transcript which has the hallmarks of SL RNAs identified in other trypanosomatids (1); a 5'-end 39-nt sequence complementary to other SL RNAs, conserved GT residue following the *trans*-spliced 39-mer, an Sm protein binding site (PuA[U]_nGpu) and a stretch of 10 T residues at the 3'-end of the transcript. The 605-bp fragment also contains a 118 bp sequence homologous to trypanosomatid 5S rRNA sequences starting 120 bp downstream of the SL RNA coding region (2). Southern blot analysis suggests that this arrangement of alternating SL and 5S rRNA genes organized into tandem repeats represents the only locus for these genes in *H. samuelpessoai* genome (data not shown). The genetic linkage of 5S and SL RNA genes has been reported in *T. rangeli* (2), *T. vivax* (2) and in some nematodes (3, 4, 5). The conservation of this linkage in distantly related organisms may suggest a functional significance to this organization.

ACKNOWLEDGEMENTS

This work was supported by USPHS Grant No: 1-PO1 AI 28778 Tropical Diseases Research Unit and MacArthur Foundation Consortium on the Biology of Parasitic Diseases.

REFERENCES

1. Agabian, N. (1990) *Cell* **61**, 1157–1160.
2. Aksoy, S., Shay, G.L., Villanueva, M.S., Beard, C.B. and Richards, F.F. (1992) *Gene* in press.
3. Krause, M. and Hirsh, D. (1987) *Cell* **49**, 753–761.
4. Takacs, A.M., Denker, J.A., Perrine, K.G., Maroney, P.A. and Nilsen, T.W. (1988) *Proc. Natl. Acad. Sci. USA* **85**, 7932–7936.
5. Nilsen, T.W., Schambaugh, J., Denker, J., Chubb, G., Faser, C., Putnam, L. and Bennett, K. (1989) *Mol. Cell. Biol.* **9**, 3543–3547.

Figure 1. Nucleotide sequence of the cloned 600-bp *Hind*III fragment. The *Hind*III restriction site interrupts the SL RNA coding sequence, thus nucleotides 1 to 48 represent the 3'-half of the gene and bases 558 to 605 contain the 5'-region. The 39-mer sequence is underlined. 5S rRNA coding segment is between bases 168 and 285. The orientation of transcription for the SL and 5S rRNAs is schematically shown.