A human HMG2 cDNA with a novel 3'-untranslated region

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The non-histone chromosomal proteins HMG1 and HMG2 have been shown to affect DNA topology, nucleosomal assembly and transcriptional activation *in vitro* (1). Their DNA binding domains (HMG-box) have been found in several other DNA-binding proteins such as hUBF and SRY (2, 3). HMG1 has been cloned from human and hamster cDNA libraries and is present as different mRNA species due to the usage of different polyadenylation sites during RNA processing (4, 5). The genomic and cDNA sequence of human HMG2 have recently been cloned (6, 7). The human HMG2 cDNA is highly homologous to the pig thymus HMG2 but has an extra 66 nucleotides of 3' UTR (7). It is postulated from both Northern and Southern analyses that HMG2 may have additional exons downstream of its last exon or there may be a highly homologous allele in the genomic DNA (6).

We have cloned a human HMG2 cDNA (HMG2b) sequence during the course of isolation of a transcription factor from a HeLa cDNA library. The coding region of our cDNA sequence and the first 378 bases of the 3' untranslated region (3'UTR) are identical to that of the cloned HMG2 cDNA (HMG2a) (7). However, using pig HMG2 as a reference, the 3'-untranslated region (3'-UTR) of HMG2b differs from that of HMG2a for 126 nucleotides starting from base 1153 (the poly(A) addition site) (8) in the pig HMG2 cDNA (Figure 1). Thus, HMG2b clone is different from the HMG2a clone in the 3'-terminal portion of their 3'-UTRs. Our results support the existence of either different alleles or a differential splicing mechanism for human HMG2.

In addition, we demonstrate, by Northern analysis using the HMG2 coding region as a probe, that in human cell cultures, HMG2 is not inducible by stress conditions such as treatment with calcium ionophore A23187 or Golgi disrupting reagent Brefeldin A (9), but is inducible by the addition of cycloheximide (Figure 2).

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REFERENCES

1. Reeck, G.R. and Teller, D.C. (1985) in Bechor, J. (ed.), *Progress in non-histone protein research* CRC Press, Boca Raton, FL, II, 1-22.
2. Jantzen, H.M., Admon, A., Bell, S.P. and Tjian, R. (1990) *Nature* **344**, 830-836.
3. Sinclair, A.H. et al. (1990) *Nature* **346**, 240-244.
4. Lee, K.L. et al. (1987) *Nucleic Acids Res.* **15**, 5051-5068.
5. Wen, L., Huang, J.K., Johnson, B.H. and Reeck, G.R. (1989) *Nucleic Acids Res.* **17**, 1197-1214.
6. Shirakawa, H. and Yoshida, M. (1992) *J. Biol. Chem.* **267**, 6641-6645.
7. Majumdar, A. et al. (1991) *Nucleic Acids Res.* **19**, 6643.
8. Shirakawa, H., Tsuda, K. and Yoshida, M. (1990) *Biochemistry* **29**, 4419-4423.
9. Liu, E.S., Ou, J.H. and Lee, A.S. (1992) *J. Biol. Chem.* **267**, 7128-7133.

Figure 1. Schematic representation of the 3'-UTRs of pig and human HMG2 cDNAs. Human HMG2 cDNAs have longer 3'-UTR than the pig, as represented by shaded lines. TAA is the stop codon and is boxed; AATAA represents the polyadenylation signal. HMG2b is the clone reported here.

Figure 2. Northern blot analysis of cytoplasmic RNA from human HeLa cells (A panel) or Alex PC cells (B panel) treated with A23187, BFA in the presence or absence of CHX (cycloheximide) as previously described (9).

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