Abstract

CTG/CAG trinucleotide repeats were commonly found in the eukaryotic genomes. Expansion of these repeated sequences caused evolutionarily conserved pathogenic effects from *C. elegans* to *human*. Recent evidences indicate that CUG or CAG-binding proteins, such as CUG-BP, Etr-1, and muscleblind，could be involved in the pathogenesis of these diseases. To further elucidate the role of these RNA-binding proteins *in vivo*, we cloned the *C. elegans* homologues of Etr-1 and muscleblind by using RT-PCR. Sequence analysis demonstrated that there were at least four differentially spliced Etr-1 transcripts, designated as Etr-1a, Etr-1c, Etr-1d, and Etr-1e. The cloned Etr-1a has exactly the same DNA sequence as that published in NCBI (GI:1289521). Compared with Etr-1a, Etr-1c lacks exon 7 and has an extra exon (exon 9). Etr-1d lacks exon 5 and exon 7, but with exon 9. Etr-1e lacks both exon 7 and exon 9. In addition, we cloned muscleblind homologues (named as MB-a and MB-b) which were evolutionarily closer to that of Drosophila than that of mammalians. Compared with the published sequence (K02H8.1). MB-a lacks exon 5 and has exon 4, and

MB-b has exon 4 but without exon 5 和 exon 6. Subsequently, we performed RNA interference to knock down Etr-1 and muscleblind transcripts and found that Etr-1 is required for embryonic survival but muscleblind knock down has no visible effects on *C. elegans*. These results indicate that different RNA-binding proteins could have distinct functions in *C. elegans*.