This gene belongs to the subfamily of ubiquitously expressed heterogeneous nuclear ribonucleoproteins (hnRNPs). The hnRNPs are RNA-binding proteins and they complex with heterogeneous nuclear RNA (hnRNA). These proteins are associated with pre-mRNAs in the nucleus and appear to influence pre-mRNA processing and other aspects of mRNA metabolism and transport. While all of the hnRNPs are present in the nucleus, some seem to shuttle between the nucleus and the cytoplasm. The hnRNP proteins have distinct nucleic acid binding properties. The protein encoded by this gene has four repeats of quasi-RNA recognition motif (RRM) domains that bind RNAs. This protein binds to the intronic polypyrimidine tracts that requires pre-mRNA splicing and acts via the protein degradation ubiquitin-proteasome pathway. It may also promote the binding of U2 snRNP to pre-mRNAs. This protein is localized in the nucleoplasm and it is also detected in the perinucleolar structure. Alternatively spliced transcript variants encoding different isoforms have been described. [provided by RefSeq, Jul 2008]

In a study, Ji et al. provided evidences for the role of a intergenic long non-coding RNA (LINREP) implicated in the regulation of PTBP1-induced alternative splicing (AS). LINREP interacted with PTBP1 and human antigen R (HuR, ELAVL1) protein complex and protected PTBP1 from the ubiquitin-proteasome degradation. Consequently, a broad spectrum of PTBP1-induced spliced variants was generated by exon skipping, especially for the skipping of reticulon 4 (RTN4) exon 3. Interestingly, LINREP also promoted the dissociation of nuclear UPF1 from PTBP1, which increased the binding of PTBP1 to RTN4 transcripts, thus enhancing the skipping of RTN4 exon 3 to some extent. Besides, HuR recruitment was essential for the stabilization of LINREP via a manner dependent on N6-methyladenosine (m6A) formation and identification. Taken together, our results demonstrated the functional significance of LINREP in human Glioblastoma for its dual regulation of PTBP1-induced AS and its m6A modification modality, implicating that HuR/LINREP/PTBP1 axis might serve as a potential therapeutic target for Glioblastoma [1)].

1)
Ji X, Liu Z, Gao J, Bing X, He D, Liu W, Wang Y, Wei Y, Yin X, Zhang F, Han M, Lu X, Wang Z, Liu Q, Xin T. N6-Methyladenosine-modified lncRNA LINREP promotes Glioblastoma progression by recruiting the PTBP1/HuR complex. Cell Death Differ. 2022 Jul 23. doi: 10.1038/s41418-022-01045-5. Epub ahead of print. PMID: 35871232.