The neuronal-specific Elav-like proteins (HuD, Hel-N, and HuC) contain three RNP-type consensus motifs and bind to AU-rich elements. We have identified and cloned a fourth member of this family (HuR) that is expressed in a wide variety of cell types. The purified recombinant protein binds avidly to the AU-rich element in c-fos and interleukin-3 mRNAs. In the case of the c-fos AU-rich element, HuR binds to a core element of 27 nucleotides that contain AUUUA, AUUUUA, and AUUUUUA motifs. Mutational analysis has shown that all three AU motifs are required for maximal binding.

Immediate early genes are rapidly and transiently expressed on stimulation of cells by a wide variety of agents, including growth factors, cytokines, and neurotransmitters (1). Although such mRNAs have diverse cellular functions, they share the unifying feature of a very short half-life (2). As a result of this short half-life, they can be regulated by alteration of either the rate of transcription or the rate of degradation. The signal transduction pathway that results in the increase in transcription of these genes is relatively well understood (3). Alteration of the rate of degradation is an equally important pathway. The mechanism underlying this pathway is poorly understood. Many immediate early mRNAs are targeted for rapid degradation by virtue of cis-acting elements in their 3′UTR (2, 4). Shaw and Kamen (5) were the first to identify a cis element responsible for regulated mRNA degradation. They showed that a 51-nucleotide AU-rich element (ARE) from the 3′UTR of granulocyte-macrophage-colony-stimulating factor could increase the degradation rate of any mRNA (5). Subsequent work has shown that similar functional ARE elements can also be identified in other mRNAs (6–8). The ARE element in IL-3 mRNA is of particular interest, since the induction of IL-3 expression in stimulated PB-3C cells is solely the result of the inhibition of mRNA degradation (9). There is little primary sequence similarity between AREs from different mRNAs. The unifying feature appears to be AUUUA within an AU-rich background. Recent studies have shown that homologues which are expressed in non-neuronal cells. ARE-mediated destabilization occurs in all cell types. Thus we decided to look for Elav-like homologues which are expressed in non-neuronal cells. Using degenerate oligonucleotides based on the Elav-like protein RNA binding domain, we have cloned a new Elav-like gene that is expressed in all tissues and whose protein product (HuR) binds to AREs with high specificity and affinity.

EXPERIMENTAL PROCEDURES

Materials—HeLa cell total RNA was prepared from a cytoplasmic extract kindly provided by Dr. J. Hurwitz (Program in Molecular Biology, Memorial Sloan Kettering Cancer Center, New York, New York 10021). Materials—Materials were purchased from Clontech. RNase T1 was obtained from Calbiochem. Primers—The primers were used as follows: DG1, 5′-TGTTG(C-T)AT(ACT)T(TCT)CTGTA(TCT)TAA-3′; DG2, 5′-CCG(GA)AA(AATGC)-GC(GA)AA(AATGC)TCT(TT)GA-3′; HuR3, 5′-TGGCC(TGTA(TCT)CCTCCG(TA)-3′; HuR3, 5′-CCAAACATGCCCAGGAGGATC-3′.

PCR Cloning of HuR—Two degenerate primers were designed that spanned residues 263–309 of HuR. This segment was chosen, since it contained two cysteine residues that are peculiar to the Elav-like class of RNA binding proteins. HeLa RNA was converted to cDNA using reverse transcriptase. PCR was carried out with the following conditions: 2 h at 93 °C; 1 min at 33 °C; 2 min at 72 °C; 1 min at 72 °C.
92°C (10 cycles); 1 min at 38°C; 2 min at 72°C; 1 min at 92°C (30 cycles); 2 min at 38°C; 10 min at 72°C. An RT-dependent product of 140 nucleotides was observed and was subcloned. The subclone pRNP49 was sequenced and found to be similar but unique to HuD, HuC, and Hel-N1. This subclone was identical in sequence to clone 33CC12, which was identified by Chris Campbell. We used the insert of clone 33CC12 to isolate a full-length cDNA. 33CC12 DNA was isolated and digested with the EcoRI. The 0.7-kilobase pair EcoRI insert was isolated and labeled by the random hexamer priming method with \([\alpha-32P]dCTP\) (Amersham Corp.). This labeled DNA was used to screen a HeLa DNA library obtained from Stratagene (La Jolla, CA). The recombinant phage library was screened at a density of 1 \(\times\) 10^4 plaque-forming units on 150-mm plates of Escherichia coli strain BB4 (Stratagene). A single clone called pHuR9 was isolated, purified, and converted to pBluescript plasmid by the phage rescue protocol according to the manufacturer's instructions. Restriction digestion of the plasmid DNA with EcoRI revealed an insert of 2.5 kilobase pair. Double-stranded DNA was sequenced on both strands using SK, KS, M13 universal, and reverse primers and internal oligonucleotide primers. Sequences were merged and analyzed for open reading frame and functional motifs with the MacVector analysis software.

RT-PCR Analysis of HuR Expression—Total RNA (2 \(\mu\)g) from various human tissues (obtained from Clontech) was incubated with 200 units of RT for 60 min at 37°C in a total reaction volume of 20 \(\mu\)l containing the following: 1 mM each dNTP, 28 units of RNase inhibitor, and 2.5 \(\mu\)M random hexamer primers. RT reactions were terminated by incubation at 99°C for 5 min and used directly for subsequent PCR amplification of specific cDNAs. One-twentieth of the RT reaction product was added to a PCR (20 \(\mu\)l final volume; which contained 50 mM Tris (pH 9.5), 1.5 mM MgCl2, 20 mM ammonium sulfate, 0.25 mM each dNTP, \(5 \mu\)Ci of \([\alpha-32P]dCTP\), 0.5 \(\mu\)M each HuR5 and HuR3 primer and 0.5 unit of Taq polymerase (Perkin-Elmer). PCR analyses were performed in an automated DNA clonal cycler (Ericomp) with the following temperature profile: 3 min at 95°C; 30 cycles of 30 s at 65°C, 30 s at 72°C, and 30 s at 95°C; 30 s at 65°C; and 2 min at 72°C. One-fifth of the PCR product was electrophoresed on a 6% acrylamide gel, and the PCR products were analyzed by autoradiography.

Construction and Purification of HuR-GST Fusion Protein—A cDNA encoding residues 2-326 of HuR was generated using PCR with BamHI-linked 5' primer and EcoRI-linked 3' primer. The resultant

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Fig. 1. A, organization of pRNP49, p33cc12 and pHuR9 cDNA clones. The scale denotes nucleotide length in kilobase pairs. The open box labeled open reading frame (ORF) denotes the predicted open reading frame. The triplacate structure of the putative RNA recognition motifs (RRM I, RRM II, and RRM III) is illustrated by the solid black boxes. B, nucleotide sequence and predicted open reading frame of HuR. The accession number of this sequence is U38175.
product was digested and ligated into digested pGEX2T (27). The re-
sultant construct was called pGEX-HuR. An overnight culture of
E. coli BL 21, transformed with pGEX-HuR, was diluted in 1:50 LB medium.
At an A600 of 0.4, the culture was induced with IPTG (0.04 mM). After 4
h of further growth, cells were spun down and resuspended in 10 ml of
buffer A (50 mM Tris (pH 8.0), 200 mM NaCl, 1 mM EDTA). The cells
were lysed by adding lysozyme and Triton to a final concentration of 0.2
mg/ml and 1%, respectively. The lysate was centrifuged at 12,000
3
4
111
g
100
for 30 min. The resultant supernatant was loaded onto a glutathione-
agarose affinity column (13 mg of protein/ml of resin). After washing the
column with buffer B (50 mM Tris (pH 8.0), 200 mM NaCl, 1 mM EDTA,
1% Triton), GST-HuR was eluted with 50 mM Tris pH 8.0/5 mM Gluta-
thione. Protein concentration was measured using the Bradford assay.
Thrombin digestion of the purified fusion protein revealed a 26-kDa
band corresponding to GST and a 36-kDa band corresponding to HuR.
Protein fractions were pooled and stored at -70°C.
Preparation of RNA Transcript—
Plasmid DNAs were digested with
the appropriate restriction enzymes and transcribed in the presence of
[32P]uridinetriphosphate (Amersham) as described previously (23, 28).
pNMUTR was linearized with HindIII and transcribed with T7 RNA
polymerase to yield a transcript of 496 nucleotides of the 3
9
-UTR of
N-myc mRNA. pNMUTR was provided by Dr. Sue Cohn (Northwest-
ern). All transcripts are gel-purified as described previously (28). pIL-3
and pIL-3DAU were linearized with EcoRI and transcribed with T7
RNA polymerase to yield transcripts of 419 and 203 nucleotides, respec-
tively. pIL-3 and pIL-3DAU were provided by Dr. Christoph Moroni
(29). Ribonucleotides were chemically synthesized at a 0.2-
4
mol scale
on a 392 DNA/RNA synthesizer (Applied Biosystems, Foster City, CA)
using RNA phosphoramidites (Glen Research Corp., Sterling, VA). Ri-onucleotides were end-labeled using T4 kinase and [\(\gamma\)-32P] ATP to a
specific activity of 1.5 \times 10^6 cpm/pmole which was then electrophoresed at 40 V for 2.5 hours. The gel was dried on DE-81 paper (Whatman) with a backing of gel drying paper (Hudson City Paper, West Caldwell, NJ) and exposed to XAR5 film (Eastman Kodak Co.) for 6 h at -70°C.
Nitrocellulose Filter Binding Assay—Reaction mixtures (0.02 ml)
contained 50 mM Tris (pH 7.0), 150 mM NaCl, 0.25 mg/ml bovine serum
albumin, 0.25 mg/ml tRNA, 4 fmol of radiolabeled mRNA, and purified
HuR as indicated. After 10 min of incubation at 37°C, the mixtures
were diluted 1:6 with buffer F (20 mM Tris (pH 7.0), 150 mM NaCl, 0.05
mg/ml tRNA) and filtered through nitrocellulose (BA85, Schleicher &
Schuell). After washing the filter twice with buffer F, bound radioac-
tivity was determined by Cerenkov counting.
RNase T1 Protection Assay—Reaction mixtures (0.02 ml) contained
50 mM Tris (pH 7.0), 150 mM NaCl, 0.25 mg/ml bovine serum albumin,
0.25 mg/ml tRNA, 4 fmol of radiolabeled mRNA and purified HuR as
indicated. After 10 min of incubation at 37°C, RNase T1 was added and
the reaction continued for a further 10 min. The mixtures were diluted
1:6 with buffer F and filtered through nitrocellulose (BA85, Schleicher &
Schuell). After washing the filter twice with buffer F, bound radioac-
tivity was determined by Cerenkov counting.
RESULTS
Isolation and Cloning of HuR—We sought to establish
whether there were other Elav-like family members that also
bind to ARE elements and are expressed in non-neuronal cell
types. We designed PCR primers that are derived from a con-
served region adjacent to RNA recognition motif III of HuD

![Fig. 2. Sequence alignment of HuC, Hel-N2, HuDmex, elR, and HuR. Homology of the amino acid sequences was analyzed by the Geneworks protein alignment program. Shaded residues indicate identity.](http://www.jbc.org/)

![RNA Complex Assay—Reaction mixtures (0.2 ml) contained 50 mM Tris (pH 7.0), 150 mM NaCl, 0.25 mg/ml tRNA, 0.25 mg/ml bovine serum albumin, 4 fmol of labeled RNA, and protein as indicated. Mixtures were incubated at 37°C for 10 min. Following incubation, 5 µl of a dye mixture (50% glycerol, 0.1% bromphenol blue, 0.1% xylene cyanol) was added, and 5 µl of the mixture was immediately loaded on a 0.8% agarose gel in TAE buffer (40 mM Tris acetate, 1 mM EDTA). The gel was then electrophoresed at 40 V for 2.5 hours. The gel was dried on DE-81 paper (Whatman) with a backing of gel drying paper (Hudson City Paper, West Caldwell, NJ) and exposed to XAR5 film (Eastman Kodak Co.) for 6 h at -70°C.](http://www.jbc.org/)
Expression of HuR—We have used the RT-PCR assay to detect HuR mRNA in various human tissues. We designed primer pairs that are specific for HuR and that spanned the putative alternative splice site found in HuD and Hel-N1. With isoforms (23, 33). The sequence of clones p33CC12 and pHuR indicate that HuR is not spliced in this domain and corresponds most exactly with the HuDmex and Hel-N2 isoforms (Fig. 2). This was confirmed by RT-PCR analysis of HuR mRNA (see below).

Expression of HuR—We have used the RT-PCR assay to detect HuR mRNA in various human tissues. We designed primer pairs that are specific for HuR and that spanned the putative alternative splice site found in HuD and Hel-N1. With isoforms (23, 33). The sequence of clones p33CC12 and pHuR indicate that HuR is not spliced in this domain and corresponds most exactly with the HuDmex and Hel-N2 isoforms (Fig. 2). This was confirmed by RT-PCR analysis of HuR mRNA (see below).
these primers, we not only measured the expression of HuR mRNA, but also determined the splice site used. Fig. 3 shows the RT-PCR analysis of HuR mRNA expression. A reverse transcriptase-dependent product of 207 nucleotides was detected using mRNA from all tissues assayed. The size of the RT-PCR product indicates that the major form of HuR mRNA is the structure defined by pHuR9 and p33CC12. Thus HuR most resembles HuDmex and Hel-N2. Additionally, a larger product of 420 base pairs was also detected. Further analysis of this product indicated that it was a PCR-generated dimer of the 207-base pair product.

HuR Binds to the ARE of c-fos mRNA—The open reading frame from pHuR9 was subcloned into the GST vector and the fusion protein purified by affinity chromatography. By virtue of its significant homology to other Elav-like proteins, we anticipated that HuR would bind to AU-rich elements. In these studies we have used transcripts derived from the 3'-UTR of c-fos mRNA, since its ARE has been functionally defined by deletion analysis (8). In particular we have used a 214-nucleotide transcript (called AUFL) which encompasses sequence from residues 568 to 781 downstream of the c-fos stop codon.

Fig. 4 shows that purified HuR quantitatively converts the AUFL transcript to a stable protein-RNA complex that migrates slowly on agarose gel electrophoresis. Complex formation was proportional to the concentration of HuR and was not apparent with high concentrations (480 nM) of GST. The complex formed by HuR migrated faster than the comparable complex formed by HuD. This difference may be due to differences in molecular weight or charge. Significantly less complex formation was observed with a transcript in which the ARE had been deleted (AU1/2) (Fig. 4B, lanes 4–6). Thus like HuD, HuR specifically binds to the ARE of c-fos mRNA (26). The binding of HuR within the ARE was further analyzed by T1 selection analysis. Fig. 5 shows that HuR binds to three fragments of 43, 35, and 20 nucleotides, respectively, with the AUFL transcript. No fragments were selected on incubation with the ARE deletion transcript. Subsequent T1 digestion of the isolated selected fragments revealed that HuR binds predominantly to the 35-nucleotide T1 fragment within the c-fos ARE (Fig. 5B). The 35-nucleotide T1 fragment contains AUUUA, AUUUUA, and the AUUUUUA motifs. The minor 43-nucleotide fragment arises by the protection of the G residues adjacent to the 35-nucleotide fragment in a minority of complexes. The selection of the 20-nucleotide fragment at high concentrations of HuR reveals the presence of a second independent low affinity binding site. We next determined whether the HuR-AUFL complex would be displaced by a molar excess of the unlabeled 35 nucleotide T1 fragment. A 35-mer, a 23-mer corresponding to the low affinity site, and an irrelevant 21-mer were synthesized. HuR-AUFL complex formation was inhibited by a molar excess of the 35-mer, but no inhibition was observed with the 23-mer (the low affinity site) or the irrelevant 21-mer (Fig. 6). From these experiments it appeared that the 35-nucleotide fragment could be the minimal binding site. Accordingly we investigated the binding of HuR to the 35-nucleotide fragment and also to a set of deletions. HuR bound to the 35-nucleotide fragment with similar affinity (apparent Kd is 3 nM) as that displayed with the AUFL transcript (apparent Kd is 2) (compare Fig. 7A with Fig. 8C). Comparable reactivity was also noted with the 27 nucleotide fragment, but substantially less was observed with the 20- and 13-nucleotide fragments. Thus we concluded that the 27-nucleotide fragment is the minimal binding site. The most obvious feature of the 27-nucleotide fragment is the presence of three AU motifs, namely AUUUA, AUUUUA, and AUUUGUU. We tested the requirement of these motifs by synthesizing

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FIG. 7. Mutational analysis of the HuR binding site. The indicated 32P end-labeled RNAs (4 fmol, 1.5 × 106 cpm/pmol) were incubated with HuR at 37 °C for 10 min and filtered through nitrocellulose. The bound radioactivity was determined by Cerenkov counting. A, deletion mutations of the c-fos ARE. B, point mutation in the c-fos ARE. C, sequences of deletions. D, sequences of point mutations.
mutant fragments in which the Us were changed to Gs and Cs (Fig. 7B). Alteration of the AUUUA motif had only a modest effect on binding (the apparent \(K_d\) of the 27-mer is 6 nM, whereas the apparent \(K_d\) of 27-2 is 12 nM). Mutation, however, of either the AUUUUA or AUUUUUA motifs had a significant effect (27-3 and 27-4 exhibited only 29% binding at 100 nM of HuR). Mutation of any two of the AU motifs further decreased binding, and mutation of all three AU motifs completely eliminated binding. Thus we conclude that all three AU motifs are required for maximal binding, but that the AUUUUA and AUUUUUA motifs play the most important role.

**HuR Binds to N-myc, c-myc, and IL-3 mRNAs**—The current classification of AU-binding proteins distinguish those activitiess that solely bind to cytokine AREs versus those that bind to all AREs. Fig. 8 (A and B) shows that HuR forms a stable complex with the 3' UTR of N-myc, c-myc, and IL-3 mRNAs. No detectable complex formation was detected with an irrelevant globin transcript or an IL-3 transcript (IL-3 \(\Delta\) AU) in which the ARE element had been deleted. Quantitative analysis using a nitrocellulose binding assay confirmed that HuR displays a similar high affinity (\(K_d = 2 \text{ nM}\)) with the ARE-containing mRNAs, but significantly less affinity with ARE deletions (\(K_d\) for AU1/2 = 70 nM; only 10% binding to IL-3\(\Delta\)AU at 100 nM) or the irrelevant globin transcript (no detectable binding at 100 nM). A significant difference between the ARE of cytokine and oncogene AREs is the arrangement of the AUUUA motifs. The IL-3 ARE contains eight AUUUA motifs (labeled I-VIII, Fig. 9B) in a complicated arrangement. Fig. 9A shows that two major T1 fragments were selected by HuR. No fragments were selected from an IL-3\(\Delta\)AU transcript. In addition, no T1 fragments were selected from a partial digest of IL-3\(\Delta\)AU. This confirms the specificity of HuR binding, in that it does not simply select large T1 fragments. The major selected T1 fragments correspond to AUUUA motifs I and VI, VII, VIII. Mutational analysis has shown that motifs VI, VII, and VIII are essential for the destabilizing activity of the IL-3 ARE (34).

**DISCUSSION**

It is clear that the destabilization of mRNA by cis-acting AREs is an important control point in gene expression. The mechanism underlying this regulation is not completely understood. In the absence of a cell-free system that reconstitutes this reaction, one approach is to study the trans-acting factors that bind to AREs. Previously we have shown that the Elav-like protein HuD binds specifically and avidly to AREs (23, 26). This protein is only expressed in neurons (31, 32). In this paper we have cloned and characterized a more distant Elav-like family member called HuR, which we have found is expressed in a wide variety of cell types. HuR contains three RNA recognition motifs in an identical arrangement to the other Elav-like proteins. We have found that the RNA binding specificity of HuR is very similar to HuD and Hel-N1. This suggests that HuR, HuD, Hel-N1, and HuC perform a similar function, albeit in different cell types. The marked diversity of Elav-like pro-
HuR does not resemble the mRNA decay system (22) and AUH was affinity-purified using a characterized AUF1 was identified by its requirement in a cell-free system that are bound by AUF1 and AUH have not been elucidated. It is possible that they bind to a different subset than HuR and that all three proteins are required for the assembly of the complex that degrades mRNA. It is also possible that AUF1, AUH, and HuR respond to different signal transduction pathways.

A number of groups have identified AU binding proteins using gel retardation and UV cross-linking assays (13–18). HuR most resembles the activity identified by Vakalopoulou et al. (17). There is close agreement in molecular weight (32 versus 36 kDa), and both activities require a central AU motif and flanking U residues. In this respect HuR may also correspond to the 32-kDa AU binding activity that binds to herpes virus saimiri small RNAs (36). HuR binds to oncogene mRNAs and to cytokine mRNAs. Thus HuR would appear to belong to the AU-A class of RNA binding proteins (13). However in contrast to the previously described AU-A activities, it binds to AREs with high affinity (the apparent Kd is 2 nM). This high affinity is comparable with the affinity of the previously described AU-B activity for AUUUA multimers (14). Although we have shown that a basal level of HuR is expressed in all cells, it will be interesting to investigate whether it is induced by factors that regulate cytokine mRNA stabilization.

In summary the properties of HuR indicate that it plays a role in ARE-dependent mRNA degradation. As we have found with other Elav-like members, HuR does not have any detectable specific endonuclease activity. Thus it is likely that the binding of HuR to mRNA may facilitate the binding of other factors that degrade mRNA. It is also equally possible that HuR may inhibit the interaction of ARE-containing mRNAs with such degradation factors. An in vitro system capable of reconstituting ARE-dependent degradation will be required to distinguish these possibilities. The HuR protein described here may facilitate the generation of such an in vitro system and permit the purification of other factors required for ARE-dependent mRNA degradation.

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Cloning and Characterization of HuR, a Ubiquitously Expressed Elav-like Protein
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