c-Jun ARE Targets mRNA Deadenylation by an EDEN-BP (Embryo Deadenylation Element-binding Protein)-dependent Pathway*

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In mammalian cells, certain mRNAs encoding cyto- kines or proto-oncogenes are especially unstable, because of the presence of a particular sequence element in their 3'-untranslated region named ARE (AUU-rich element). AREs cause this instability by provoking the rapid shortening of the poly(A) tail of the mRNA. The deadenylation of mRNAs mediated by AREs containing repeats of the AUUUA motif (class I/II AREs) is conserved in Xenopus embryos. Here, we first extend these observations by showing that c-Jun ARE, a representative of class III (non-AUUUA) AREs, also provokes the deadenylation of a reporter RNA in Xenopus embryos. Next, by immunodepletion and immunoneutralization experiments, we show that, in Xenopus, the rapid deadenylation of RNAs that contain the c-Jun ARE, but not an AUUUA ARE, requires EDEN-BP. This RNA-binding protein was previously shown to provoke the rapid deadenylation of certain Xenopus maternal RNAs. Finally, we show that CUG-BP, the human homologue of EDEN-BP, specifically binds to c-Jun ARE. Together, these results identify CUG-BP as a plausible deadenylating factor responsible for the post-transcriptional control of c-Jun proto-oncogene mRNA in mammalian cells.

The control of mRNA translation and/or stability, as a means of regulating gene expression in eukaryotic cells, is now recognized as a mechanism of widespread importance. In a large number of cases, this control is exerted via the 3'-terminal poly(A) tail. In general, mRNAs with a long poly(A) tail are much more actively translated and stable than mRNAs that have a short or no poly(A) tail (reviewed in Refs. 1 and 2). Cytoplasmic activities that alter the length of the poly(A) tail are therefore potent regulators of gene expression. These activities are often modulated by sequence elements that reside within the 3'-untranslated region (3'-UTR)1 of mRNAs.

Among the sequence elements that provoke the shortening of the poly(A) tail (deadenylation), and thereby destabilization, the best known in mammalian somatic cells is probably the family of A/U-rich elements (ARE) (for a review, see Ref. 3).

AREs are present in the 3'-UTRs of many unstable mRNAs such as those encoding proto-oncogenes or cytokines. Based on their sequences, AREs have been divided into three classes. Class I AREs, exemplified by c-Fos ARE, contain several (AUUUA) motifs interspersed within a less defined region. Class II AREs, such as GM-CSF ARE, contain overlapping (AUUUA) motifs. Finally, class III AREs, exemplified by c-Jun ARE, contain no AUUUA motif (3, 4).

It is highly probable that the three classes of AREs act by binding specific factors that target rapid deadenylation. Several class I and II (AUUUA-containing) ARE-binding factors have been identified. Two factors, HuR and hnRNP/AUF1 have been specifically studied. Overexpression of HuR in a variety of cell lines leads to a stabilization of class I/II ARE-containing RNAs (5–7). Cell lines depleted of HuR by an antisense strategy were recently established. This depletion leads to a destabilization of class I/II ARE-containing RNAs (8, 9).

These results strongly suggest that HuR, in binding to AUUUA-containing AREs, stabilizes these RNAs. The function of hnRNP/AUF1 binding to AUUUA-containing AREs is less clear and may be cell type-specific. Overexpression of this protein in K562 cells during hemin-induced differentiation destabilizes class I/II ARE containing RNAs (10), whereas overexpression of the same protein in NIH3T3 cells stabilizes class I/II ARE-containing RNAs (11). To date, no factor involved in the control of the stability of class III, non-AUUUA ARE-containing RNAs has been characterized.

Xenopus embryos are a powerful biological model to identify deadenylation factors for several reasons. First, in Xenopus embryos, mRNA deadenylation and degradation, though functionally coupled, are temporally uncoupled. Deadenylated RNAs are as stable as their polyadenylated counterparts until the blastula stage, several hours after fertilization (12, 13). This allows these two phenomena to be analyzed separately. Second, deadenylation-proficient cell-free extracts can be made (14, 15) that permit several biochemical manipulations. Finally, and most importantly, the functions of several sequences and factors that target rapid deadenylation are conserved between Xenopus and mammals. For instance, AUUUA AREs provoke mRNA deadenylation in Xenopus embryos (13, 15), and the human and Xenopus oocyte poly(A)-specific ribonucleases (PARN) are functionally equivalent (16). Accordingly, studying deadenylation mechanisms in Xenopus embryos should give important clues to understand these mechanisms in mammals.

In the present study, we have used Xenopus embryos to study c-Jun ARE, a representative class III, non-AUUUA, ARE (4). We first show that c-Jun ARE-dependent rapid deadenylation is conserved between mammals and Xenopus. Secondly, we show that, in Xenopus embryos, the rapid deadenylation conferred by c-Jun ARE requires EDEN-BP. EDEN-BP (EDEN-binding protein) is a factor which by binding to a specific cis
sequence named EDEN (embryo deadenylation element), targets certain maternal Xenopus mRNAs to rapid deadenylation after fertilization (17). The maternal EDEN-containing mRNAs are in a polyadenylated form in unfertilized eggs because of a cytoplasmic polyadenylation that takes place during oocyte maturation. Cytoplasmic polyadenylation requires a cis element different from the EDEN, and named a CPE (cytoplasmic polyadenylation element) (18, 19). We show that the requirement of EDEN-BP for rapid deadenylation of c-Jun is specific, as inactivating EDEN-BP has no effect on AUUUA ARE-mediated deadenylation. Finally, we show that the human sequence homologue of EDEN-BP, CUG-BP (20), specifically binds to c-Jun ARE, making it a plausible factor to be responsible for c-Jun mRNA rapid deadenylation and degradation in mammalian cells.

**EXPERIMENTAL PROCEDURES**

**Cloning Procedures—**c-Jun ARE (4, 21) was amplified by RT-PCR from HeLa cell RNA with the following primers: sense, GCTCTAGATCAGTAGGGCTTGCTGCTGTCATTC; Human sense, TAGAGGATCCGCCATGAACGGCACCCTGGACC; Xenopus ACAATGGACC; antisense, GTAATGGATCGTTTTATTTATTTATTTA-CTGGCCTGCTTTCGTTAACTGTGTATG; antisense, GTAATGGATCCGAACGGCACCCTGGACC; antisense, GTAGATCGTTTTATTTATTTATTTA-CTGGCCTGCTTTCGTTAACTGTGTATG; Human antisense, GCTCTAGATCAGTAGGGCTTGCTGCTGTCATTC; Human sense, TAGAGGATCCGCCATGAACGGCACCCTGGACC; Xenopus ACAATGGACC; antisense, GTAATGGATCGTTTTATTTATTTATTTA-CTGGCCTGCTTTCGTTAACTGTGTATG; Human antisense, GCTCTAGATCAGTAGGGCTTGCTGCTGTCATTC; Human sense, TAGAGGATCCGCCATGAACGGCACCCTGGACC; Xenopus ACAATGGACC; antisense, GTAATGGATCGTTTTATTTATTTATTTA-CTGGCCTGCTTTCGTTAACTGTGTATG; Human antisense, GCTCTAGATCAGTAGGGCTTGCTGCTGTCATTC; Human sense, TAGAGGATCCGCCATGAACGGCACCCTGGACC; Xenopus ACAATGGACC; antisense, GTAATGGATCGTTTTATTTATTTATTTA-CTGGCCTGCTTTCGTTAACTGTGTATG; Human antisense, GCTCTAGATCAGTAGGGCTTGCTGCTGTCATTC; Human sense, TAGAGGATCCGCCATGAACGGCACCCTGGACC; Xenopus ACAATGGACC; antisense, GTAATGGATCGTTTTATTTATTTATTTA-CTGGCCTGCTTTCGTTAACTGTGTATG; Human antisense, GCTCTAGATCAGTAGGGCTTGCTGCTGTCATTC; Human sense, TAGAGGATCCGCCATGAACGGCACCCTGGACC; Xenopus ACAATGGACC; antisense, GTAATGGATCGTTTTATTTATTTATTTA-CTGGCCTGCTTTCGTTAACTGTGTATG; Human antisense, GCTCTAGATCAGTAGGGCTTGCTGCTGTCATTC; Human sense, TAGAGGATCCGCCATGAACGGCACCCTGGACC; Xenopus ACAATGGACC; antisense, GTAATGGATCGTTTTATTTATTTATTTA-CTGGCCTGCTTTCGTTAACTGTGTATG; Human antisense, GCTCTAGATCAGTAGGGCTTGCTGCTGTCATTC; Human sense, TAGAGGATCCGCCATGAACGGCACCCTGGACC; Xenopus ACAATGGACC; antisense, GTAATGGATCGTTTTATTTATTTATTTA-

**In Vitro Transcription—**Capped, polyadenylated, and radiolabeled GbORF-jun RNA was synthesized in vitro using EcoRI linearized pT7TS vectors and purified antibodies were obtained by in vitro transcription using EcoRI linearized matrices and T7 RNA polymerase with the Promega Riboprobe kit. Large scale uncapped RNA for recombinant proteins production were obtained from EcoRI-linearized pT7TS matrices by in vitro transcription using the Promega Ribomax kit.

**Xenopus Methods—**Microinjections of 20–30 nl of in vitro transcripts into Xenopus two-cell embryos were done following standard procedures. Microinjections of in vitro transcripts with purified antibodies are described elsewhere.2 Deadenylation proficient egg extracts have been described (14). Deadenylation activities were analyzed either by incubating RNAs in the extracts at 22 °C for the indicated time, or by incubating the injected embryos at 22 °C for the indicated times. After incubation, RNAs were extracted, electrophoresed and transferred to a 4% acrylamide-urea gel, and autoradiographed as described (14). UV cross-linking and antibody methods have been described (14, 17).

**RESULTS**

c-Jun ARE Provokes Rapid Deadenylation of a Reporter RNA in Xenopus Embryos—To test whether the capacity of c-Jun ARE to target rapid deadenylation in human cells (4) is conserved in Xenopus embryos, this sequence element was cloned 3′ of the globin open reading frame (ORF). The resulting plasmid was used as a template to synthesize a capped, polyadenylated transcript (GbORF-jun). This transcript was injected into two-cell Xenopus embryos, and its deadenylation behavior was analyzed by denaturing electrophoresis and autoradiography (Fig. 1). A completely deadenuylated form of the GbORF-jun transcript could be detected as early as 1 h after injection (lane 6), and the transcript was predominantly deadenylated 3 h after injection (lane 8). In contrast, and as previously shown (12), no completely deadenylated form of the reporter RNA alone (GbORF) could be detected, even 3 h after injection (lane 4). The deadenylation pattern of the GbORF transcript is evocative of default deadenylation, a slow activity for which the only sequence specificity is an absence of a CPE, which is stimulated during oocyte maturation and persists after fertilization (23–25). These results show that c-Jun ARE targets RNAs for rapid deadenylation in Xenopus embryos, demonstrating a functional conservation of this sequence element in vertebrates.

**c-Jun ARE-dependent Rapid Deadenylation Is Dependent on EDEN-BP in Xenopus—**As a representative class III ARE, c-Jun ARE contains no AUUUA motif (see Introduction). Examination of its sequence revealed the presence of a putative CPE UUUUUAAUU, the element that drives cytoplasmic polyadenylation in Xenopus maturing oocytes (18, 19) and of numerous U/purine dinucleotides (Fig. 2). We have previously shown that an EDEN, the element that drives RNA deadenylation in Xenopus embryos is also enriched in U/purine dinucleotides (17, 26). This suggests therefore that c-Jun ARE acts as an EDEN sequence to target rapid deadenylation in Xenopus embryos. In this case, c-Jun ARE-mediated RNA deadenylation would require active EDEN-BP, the factor that specifically binds to EDEN sequences (17).

In Xenopus cytoplasmic eggs extracts, immunodepletion of EDEN-BP completely abolishes EDEN-dependent RNA deadenylation (17). In Fig. 3A (upper bands) is shown the behavior of GbORF-jun RNA in EDEN-BP- or mock-depleted extracts. In mock-depleted extracts (lanes 1–3), GbORF-jun RNA was deadenylated, as shown by the appearance of a completely deadenylated form of the transcript after 1.5 h of incubation, which was more evident after 3 h of incubation. The deadenylation of GbORF-jun was strongly diminished by EDEN-BP immunodepletion, as no completely deadenylated form of the transcript was detected even after 3 h of incubation (lanes 4–6). In contrast, and as previously shown (17), immunodepletion of EDEN-BP had no effect on the slow, default-type deadenylation of the reporter GbORF transcript (lower bands). Therefore, c-Jun ARE-dependent deadenylation in Xenopus egg extracts specifically requires EDEN-BP.

The deadenylation of the GbORF-jun transcript is significantly less efficient in extracts than in living embryos (compare Figs. 3A and 1). It could be argued therefore that in living embryos the behavior of this transcript is mainly due to an EDEN-BP-independent deadenylation mechanism. This hypothetical mechanism would be lost while preparing cell-free extracts. To test this hypothesis, we used purified antibodies directed against EDEN-BP to immunoneutralize this protein in Xenopus living embryos. EDEN-BP immunoneutralization specifically inhibits EDEN-dependent deadenylation as measured

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The positions of the polyadenylated (A\textsubscript{+}) RNAs was analyzed by denaturing electrophoresis and autoradiography. The positions of the polyadenylated (A\textsubscript{+}) and deadenylated (A\textsubscript{−}) RNAs are indicated on the left. B and C. Xenopus two-cell embryos were coinjected with 100 ng of either anti-EDEN-BP (lanes 1–4) or GbORF-(AUUUA)\textsubscript{8} (GbORF-AUUUA) transcript (C). RNAs were extracted at the indicated times, and the deadenylation behavior of the injected transcripts was analyzed by denaturing electrophoresis and autoradiography. The positions of molecular weight markers are indicated on the left.

FIG. 3. The rapid deadenylation mediated by c-Jun ARE requires active EDEN-BP. A, capped, radiolabeled GbORF-jun transcripts were incubated for the indicated times in an extract previously immunodepleted of EDEN-BP (lanes 4–6) or mock depleted (lanes 1–3). RNAs were extracted, and the deadenylation behavior of the injected RNAs was analyzed by denaturing electrophoresis and autoradiography. The positions of the polyadenylated (A\textsuperscript{+}) and deadenylated (A\textsuperscript{−}) RNAs are indicated on the left. B and C. Xenopus two-cell embryos were coinjected with 100 ng of either anti-EDEN-BP (lanes 1–4) or nonimmune (NI) antibodies (lanes 5–8) and capped, radiolabeled, polyadenylated GbORF-jun transcript (B) or GbORF-(AUUUA)\textsubscript{8} (GbORF-AUUUA) transcript (C). RNAs were extracted at the indicated times, and the adenylated behavior of the injected transcripts was analyzed by denaturing electrophoresis and autoradiography. The positions of the polyadenylated (A\textsuperscript{+}) and deadenylated (A\textsuperscript{−}) RNAs are indicated on the sides of the gel.

FIG. 4. Human CUG-BP specifically binds c-Jun ARE. Wheat germ extracts programmed to synthesize EDEN-BP or CUG-BP or unprogrammed (U), as indicated, were processed for UV cross-linking to the radiolabeled c-Jun ARE in the presence (lanes 2, 4, 6) or the absence (lanes 1, 3, 5) of a 50-fold excess of unlabeled RNA containing the Eg\textsubscript{5} EDEN. The positions of molecular weight markers are indicated on the right.

EDEN-BP to target an mRNA to rapid deadenylation in Xenopus embryos. To test if this requirement is specific for class III ARE, or if it concerns any ARE, we analyzed the deadenylation behavior of an RNA harboring an AUUUA ARE in EDEN-BP immunoneutralized embryos. It was shown previously that RNAs containing repeats of the (AUUUA) motif were deadenylated in Xenopus embryos, demonstrating the conservation of class I/II ARE mediated deadenylation between Xenopus and mammals (13, 15). Accordingly, when the GbORF reporter RNA containing a eight (AUUUA) repeat (GbORF-AUUUA transcript) was injected with nonimmune antibodies, it was not affected by the injection of anti-EDEN-BP antibodies (lanes 1–4). Therefore, RNAs that contain c-Jun ARE, but not AUUUA-type ARE, are rapidly deadenylated in Xenopus embryos by an EDEN-BP-dependent pathway.

The Human Homologue of EDEN-BP Binds c-Jun ARE—It can be hypothesized that, as EDEN-BP is responsible for c-Jun ARE-mediated rapid RNA deadenylation in Xenopus embryos, a human sequence homologue of EDEN-BP may target c-Jun mRNA for rapid deadenylation in human cells. This hypothesis requires that the human homologue of EDEN-BP should bind to c-Jun ARE. The closest human sequence homologue of EDEN-BP is CUG-BP, which is 88% identical to Xenopus EDEN-BP at the amino acid level (20).

To test if CUG-BP can bind to c-Jun ARE, recombinant human CUG-BP and Xenopus EDEN-BP were expressed in wheat germ extracts. The capacity of these recombinant proteins to bind to c-Jun ARE were assayed by UV cross-linking (Fig. 4). A strong UV cross-linking signal was detected with wheat germ expressing either EDEN-BP (lane 3) or CUG-BP (lane 5). In contrast, no similar signal was observed with unprogrammed wheat germ extracts (lane 1), demonstrating that the observed signals are due to the recombinant proteins. To test the specificity of the interaction between c-Jun ARE and EDEN-BP or CUG-BP, cross-linking to c-Jun ARE was assayed using an Eg2-derived probe (data not shown). When the GbORF-jun transcript was injected together with anti-EDEN-BP antibodies, the deadenylation behavior of this RNA was abrogated (Fig. 3B, lanes 5–8). Indeed, in EDEN-BP immunoneutralized embryos, the GbORF-jun transcript was further polyadenylated, as evidenced by the reduction of its electrophoretic mobility. This is probably due to the action of the putative CPE that is present in c-Jun ARE (see Fig. 2). In contrast, the deadenylation of the GbORF-jun transcript was maintained when this transcript was injected with control, nonimmune antibodies (Fig. 3B, lanes 5–8).

The above data show that c-Jun ARE requires an active
in the presence of a 50-fold molar excess of unlabeled RNA containing the Eg5 EDEN (17). The cross-linking signal was strongly diminished by this excess of competitor (lanes 4 and 6). Hence, both EDEN-BP and CUG-BP can bind specifically to c-Jun ARE.

**DISCUSSION**

In the present article, we show that the ability of c-Jun (class III) ARE to provoke rapid RNA deadenylation is conserved between human somatic cells and Xenopus embryos. Similar results on AUUUA (class I/II) AREs have been published (13, 15). Hence, the function of the three classes of AREs is conserved between mammalian somatic cells and Xenopus embryos. It should be noticed however that ARE-mediated deadenylation leads to mRNA degradation in mammalian somatic cells, but not in early Xenopus embryos. Deadenylated mRNAs are stable in Xenopus embryos until the blastula stage, several hours after fertilization (this study and Refs. 12 and 15).

Next, we used immunodepletion and immunoneutralization experiments to show that c-Jun ARE-mediated deadenylation in Xenopus embryos required EDEN-BP, both in vitro and in vivo. Furthermore, we have shown that neutralizing EDEN-BP did not affect the deadenylation of a AUUUA-containing reporter RNA. By overexpressing hnRNPD/AUF1, Xu et al. (11) recently showed that the different isoforms of this ARE-binding protein have different destabilizing effects on class I and class II AREs. However, the deadenylation mediated by c-jun ARE was not affected in these experiments. Together therefore, these results show that mRNAs containing different classes of AREs are regulated by different trans-acting factors, and it is at least theoretically possible for the cell to regulate the deadenylation of the various ARE-containing mRNAs independently. This observation may be especially important considering that c-Fos proto-oncogene mRNA contains several AUUUA motifs (3), and that c-Fos and c-Jun proteins are subunits of the AP-1 transcription complex (27). Differential post-transcriptional regulation of c-Jun and c-Fos would be a way to alter the composition of the AP-1 complex, which may have important implications on its targets.

Having shown the requirement of EDEN-BP for c-Jun ARE-mediated deadenylation in Xenopus embryos, it was tempting to hypothesize that the closest human sequence homologue of EDEN-BP, CUG-BP (20), is responsible for the post-transcriptional regulation of c-Jun mRNA in human cells. In support of this hypothesis, we show that CUG-BP indeed binds to c-Jun ARE. c-Jun ARE is enriched in dinucleotides U/purine. This characteristic of the cis-element bound by CUG-BP is in agreement with data obtained using a tri-hybrid assay (28). In addition, in a UV cross-linking experiment, CUG-BP binds BRE sequences that consist mainly of U/purine repeats (29). It remains to be demonstrated conclusively that CUG-BP is a deadenylation factor in human cells, where it would regulate the expression of c-Jun proto-oncogene. This may be attempted using an antisense RNA strategy similar to that used for HuR protein (8, 9).

A role for CUG-BP in class III ARE-dependent deadenylation could appear contradictory with the described nuclear function for this protein as an alternative splicing regulator of the cardiac troponin T and the insulin receptor pre-mRNAs (30, 31). However, CUG-BP has been detected both in the nucleus and the cytoplasm (32). Moreover, most ARE-binding factors are nucleus-cytoplasm shuttling proteins, with different functions in these two compartments (reviewed in Ref. 33). Accordingly, a dual role for CUG-BP as both a splicing regulator and a deadenylation factor is conceivable.

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