Atlas of Genetics and Cytogenetics in Oncology and Haematology

OPEN ACCESS JOURNAL AT INIST-CNRS

Gene Section

Review

EEF1A1 (eukaryotic translation elongation factor 1 alpha 1)

Bruna Scaggiante, Giorgio Manzini

Department of Life Sciences, University of Trieste, via Giorgieri, 1, 34147-Trieste, Italy (BS, GM)

Published in Atlas Database: May 2009

Online updated version: http://AtlasGeneticsOncology.org/Genes/EEF1A1ID40407ch6q13.html
DOI: 10.4267/2042/44731

Identity

Other names: CCS-3; CCS3; EEF-1; EEF1A; EF-1-; alpha-1; EF-Tu; EF1A; FLJ25721; RAF-1EF; LENG7; MGC102687; MGC131894; MGC16224; PTI1; eEF1A-1

HGNC (Hugo): EEF1A1

Location: 6q13

Local order: Distal to LOC100129409, proximal to SLC17A5.

DNA/RNA

Description

8 exons, 7 introns (1st intron within 5'UTR), plus a rare optional exon within first intron as found in several ESTs (e.g. emb-CR981691.1, dbj-DC388133.1, dbj-DC406334.1).

Presumably a second promoter, about 800 nt upstream of the most common transcription start, provides an alternative first exon about 320 nt long, as deduced from some ESTs at NCBI (e.g. dbj-DC316623.1, gb-BU173251.1, dbj-DC358918.1).

Introns number 2, 3, 4, 6 are phase 0 (between codons), Introns number 5, 7 are phase 1 (between 1st and 2nd base of codon).

A validated C-G non-synonimous polymorphism has been reported at 1st position of codon 382 (Arg-Gly), plus a few single-hit non-synonimous and some synonymous within CDS. Several others within 3'UTR and introns (SNP source).

Transcription

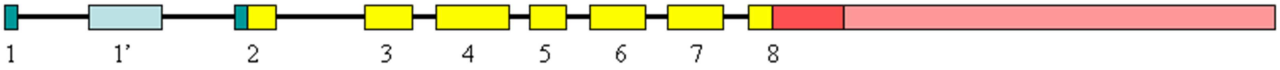
The main processed mRNA encompasses exons 1, 2, 3, 4, 5, 6, 7, 8, this last can be in short or long form. In a few cases also exon 1' is retained. In a few cases exons 1 (and 1') are substituted by the alternative exon from a putative upstream minor promoter, as described above. Moreover, a quite high number of processed transcripts that, after exons 1 and 2, retain intron 2, which introduces a stop codon 22 residues downstream of exon 2 are found (see for instance some of the many ESTs: dbj-DC389722.1, dbj-DC341899.1, dbj-DC414491.1).

Pseudogene

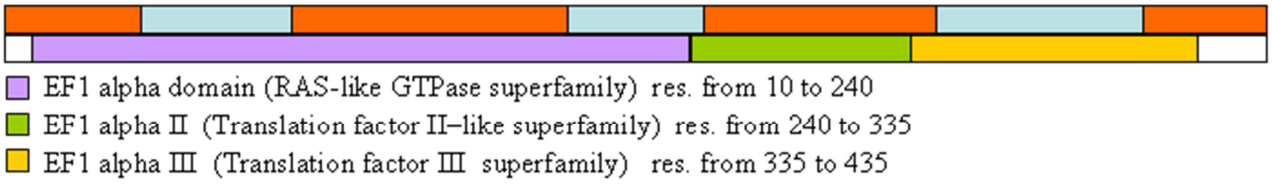
About 20 complete or approximately complete intronless pseudogenes, likely generated by retro-transposition, a few of them exempt from frameshifs and with only a few missenses, are present throughout the genome. Two of them harbour a few hundreds nt long insert each, not related to introns of the expressed gene.

All of them show a higher homology to EEF1A1 than to EEF1A2. Most of the pseudogenes find an orthologous counterpart within the chimpanzee genome.

EEF1AL3 (9q34) Note: highly homologous; EEF1AL4 (7p15.3) Note: highly homologous but with 1 frameshift; EEF1AL5-LOC390924 (19q13.12) Note: contains a 502 nt insert; EEF1AL6 (3q27.1); EEF1AL7 (4q24); EEF1AL8-



Box = exon (blue = 5'UTR, yellow = CDS, light blue = rare optional exon, red = 3'UTR, light red = extended 3'UTR to a downstream polyA signal) Line = intron.



Upper boxes, alternating colours: exons (coding part only). Lower boxes: protein domains.

LOC100132804 (7q35); EEF1AL9 (1p21.3); EEF1AL10-LOC644604 (2q12); EEF1AL11 (5p15.1) Note: rather highly homologous; EEF1AL12-LOC647167 (1q31.3); EEF1AL13-LOC100130211 (Xq21.2) Note: lacking about 300 initial codifying nt; LOC124199 (16p12.1) Note: contains a 307 nt insert; LOC387845 (12p12.3); LOC389223 (4q28.3); LOC401717 (12q12); LOC442709 (7q21.13) Note: harbours a 21 nt deletion; LOC645693 (15q21.2); LOC645715 (3p22.1); LOC646612 (3q22.3) Note: lacking about 120 initial codifying nt; LOC728672 (12p12.3); LOC100128082 (5p12).

Protein

Note

The major form of the protein is 462 residues long, composed by three domains, as shown by the diagram, that relates also the protein domains (lower bar) with mRNA coding exons (upper bar).

Description

462 residues, theoretical MW 50140.8 Da, theoretical isoelectric point 9.7 eEF1A1 is one of the alpha subunit forms of the elongation factor 1 complex, that interacts with aminoacylated tRNA and delivers it to the A site of the ribosome during the elongation phase of protein synthesis. The other form is eEF1A2, encoded by a different gene, EEF1A2, located in chromosome 20.

Expression

EEF1A1 is constitutively expressed in all tissues, with the exception of adult brain, heart and skeletal muscle, where EEF1A2 expression is found.

Localisation

Mostly cytoplasmic, but also nuclear.

Function

Canonical function: aa-tRNA delivery to ribo-some in mRNA translation

The eukaryotic elongation factor 1A (eEF1A1, formerly EF-1alpha or eEF1A) protein belongs to the G-protein superfamily, is one of most abundantly expressed protein in mammalian cells and participates to mRNA translation. It carries aminoacyl-tRNA (aa-tRNA) to the A site of the ribosome as a ternary complex eEF1A1-GTP-aa-tRNA. In mammalian, it is ubiquitously expressed with exception of skeletal muscle, heart and brain where during terminal differentiation eEF1A2 is produced (Knudsen et al., 1993).

Moonlighting functions: cytoskeletal remodel-ling, protein folding and degradation, cell signal-ling modulation, control of cell growth, apoptosis and cell cycle

1) eEF1A1 and cytoskeletal remodelling. The most relevant non canonical function of eEF1A1 is the modulation of cytoskeleton organization. eEF1A has activity on microtubule severing and bundling. It has a specific site to bind actin that is different from that for the binding of aa-tRNA (Gross et al., 2005). eEF1A binding to F-actin is modulated by Rho/Rho-kinase pathway. Phospho-rylation by Rho kinase decreases the binding of eEF1A1 to F-actin and F-actin bundling. Myosin phosphatase acts in antagonist fashion on eEF1A1 to modulate actin cytoskeletal organization (Izawa et al., 2000).

2) eEF1A1 and protein degradation and folding. eEF1A controls translational fidelity by binding to uncorrectly folded proteins but not to correctly folded ones. The uncorrectly folded proteins are then directed to degradation pathway (Hotokezaka et al., 2002). eEF1A plays a role in recognition and degradation of co-translationally damaged and ubiquitylated proteins promoting their translocation to proteasome through interaction with proteasome subunit Rpt1 (Chuang and Madura, 2005). eEF1A exhibits chape-rone-like activity by promoting renatura-tion of enzymes such as aminoacyl-tRNA synthetases, likely contributing to maintain the efficiency of translational machinery (Lukash et al., 2004).

3) eEF1A1 and control of cell cycle, growth and death. eEF1A as ribonucleoprotein complex, containing a non-coding RNA, binds to and mediates activation of heat-shock transcription factor 1 (HSF1) to protect the cell from heat-shock (Shamovsky et al., 2006). Induction of the non-constitutive eEF1A1 expression in cardiomyocytes as response to lipotoxic ER-stress promotes cell death likely by activation of

eEF1A1-dependent cytoske-letal modifications triggering apoptosis (Borradaile et al., 2006). eEF1A1 interacts with the HDM2 gene product at a binding site for eEF1A1 overlaps with that for p53. In normal cells eEF1A1 could promote cell apoptosis by preventing p53 sequestra-tion by HDM2 (Frum et al., 2007). Likely both eEF1A1 and eEF1A2 interacts with the zinc finger protein ZPR1 in response to mitogenic stimuli, redistributing eEF1A1/2 and ZPR1 in the nucleus. This interaction is essential for normal cell proliferation and growth. Thus the interaction eEF1A1/2-ZPR1 is required for normal cell cycle progression (Mishra et al., 2007). eEF1A1 is an interactor of Bood POZ containing gene type 2 (BPOZ-2) that promotes eEF1A1 ubiquitylation and degradation via 26S proteasome. BPOZ-2 inhibits GTP binding to eEF1A1 thus preventing translation. BOPZ-2 is transcriptionally activated by Phosphate and Tensin homologue deleted on chromosome 10 (PTEN). It has been suggested that PTEN exerts growth inhibition effects in cells not only by antagonizing PI3K-Akt signalling path-way, but also inducing BPOZ-2 expression to degrade eEF1A1. In this manner, in normal cells, the transition from growing to resting phases is mediated by BPOZ-2/eEF1A1 interaction, thus leading to prevention of translation and induction of eEF1A1 degradation by 26S proteasome pathway (Koiwai et al., 2008). eEF1A1 is implicated in a novel cell cycle check-point to prevent tetraploidy in binucleated cells. In tetraploids, cell death, preventing aneuploidy malignancies, is mainly controlled in a caspase-independent man-ner by the down-regulation of eEF1A1 levels. eEF1A1 mRNA accumulates in specialized P bodies to reduce the expression of the proteins. The prominent signal in the eEF1A1 mRNA for its translational repression and degradation is in the 5'-UTR. Exogenous expression of eEF1A1 inhibits cell death in tetraploids. Notably, exogenous expression of eEF1A2 whose mRNA 5'-UTR differs from that of eEF1A1 inhibits cell death in tetraploids, thus suggesting another mechanism by which eEF1A2 could promote tumour development (Kobayashi et al., 2009).

4) eEF1A1 and cell signalling modulation. Besides eEF1A2, in adult mouse neurons eEF1A1 is expressed too and it is able to regulate the recycle of M4 muscarinic acetylcholine receptors (mAChR). Thus, eEF1A1 plays a role in locomotor activity of neurons (McClatchy et al., 2006). eEF1A1 modulates the activities of sphingosine kinases (SK1 and SK2). Phosphorylated and non-phosphorylated eEF1A1 forms interact with phosphoryla-ted and non phosphorylated SK1 and SK2 and this results in an increased enzymatic activity of both SK1 and SK2. In this respect, overexpression of eEF1A1 in quiescent cells has been suggested to play a role in oncogenesis by increasing SK1 and SK2 activities (Leclercq et al., 2008). eEF1A1 is involved in the regulation of vascular function mediated by TNF-alpha. eEF1A1 binds to 3'-UTR of the endothelial nitric oxide synthase (eNOS) to regulate post-translational eNOS mRNA stability. In the human endothelial cell line HUVEC, TNF-alpha-mediated eNOS mRNA destabilization involves eEF1A1 to reduce eNOS mRNA levels (Yan et al., 2008).

Homology

Highly homologous over the entire length to EEF1A2 (92% identities);

Moderately homologous over all three domains, higher for the first one, to :

HBS1L, a member of the GTP-binding protein family expressed in erythroid progenitor cells (39% identities);

GSPT1, a GTP-binding protein involved in G1 to S phase transition (38% identities);

GSPT2, a GTP-binding protein involved in G1 to S phase transition (37% identities);

TUFM, Tu translation elongation factor, mitochon-drial (31% identities).

Implicated in

Head and neck cancers

Note

eEF1A1 overexpression is observed in cisplatin-resistant human head and neck cancer cell lines (Johnsson et al., 2000).

Breast cancer

Note

EEF1A1 was found to be upregulated in invasive breast cancer cells derived from snap-frozen adenocarcinoma samples suggesting a role in mediating invasive activity of cancer cells (Zhu et al., 2003). Treatment of the breast human cancer cell line MCF-7 with the histone deacetylase inhibitor sodium butyrate decreases significantly in a dose-dependent manner the eEF1A1 transcription levels. Thus, overexpression of eEF1A1 contributes to breast cancer survival (Gonçalves et al., 2005).

Tongue squamous cell carcinoma

Note

A suggestive down-regulation of EEF1A1 expression has been observed in human tongue squamous cell carcinoma with positive lympho-nodes and extracapsular spread. Thus EEF1A1 down-regulation might be involved in the tumour cell progression toward the metastasis (Zhou et al., 2006).

Hepatocarcinoma

Note

eEF1A1 overexpression in human hepatocarcinoma cell lines correlates with an increase of proliferation rate and with the ability to escape apoptosis under suboptimal growth conditions. In particular eEF1A1 overexpression is higher in the more aggressive

phenotype cell line JHH6 with respect to the more differentiated HepG2 and HuH7 cells (Grassi et al., 2007). eEF1A1 regulates the half-life of osteopontin (OPN) mRNA. eEF1A1 is a trans-acting factor that binds to 5'-UTR of OPN. This has strong implications in the invasive process as demonstrated in hepatocellular carcinoma cells, OPN being the major secreted phosphoprotein which is overexpressed by tumour cells in advanced metastatic cancer.

The higher expression of OPN in invasive cancer cells is due to the different localization of eEF1A1: in non invasive Hep3B cells it is mainly bound to G actin whereas in invasive HepG2 type eEF1A1 and G actin association is minimal. Thus eEF1A1 is an indirect regulator of OPN by affecting the OPN mRNA stability through the interaction with G actin. Only F actin-bound eEF1A1 cannot interact with 5'-UTR of OPN (Zhang et al., 2009).

Testicular germ tumours

Note

eEF1A1, as well as eEF1A2, is an interactor of the human testis-specific Y-encoded (TSPY) gene. It has been demonstrated that the binding to TSPY leads to a redistribution of the TSPY-eEF1A1/2 complex in the cell with a nuclear co-localization. A role of the TSPY-eEF1A1/2 complex has been suggested in promoting neoplastic transformation and in sustaining cancer cell growth in human testicular germ tumours, prostate cancer, as well as in other somatic cancers (Kido et al., 2008).

Cervical cancer

Note

A variant form of eEF1A1 named cervical cancer suppressor 3 (CCS-3) lacking the 101 aminoacids at the N-terminal region, has been identified as tumour suppressor that is present in non-trans-formed human cell lines. Its ectopical expression in a cervical tumour cell line leads to cell growth inhibition and apoptosis. CCS-3 seems to act as a co-transcriptional repressor by interacting with the transcriptional regulator PLZF (Rho et al., 2006).

The mRNA of CCS-3 (GenBank Accession AF322220) appears to be a fusion product joining the reverse of the end portion of the 3' UTR of another product (NM_005763, from gene AASS) with most of the normal sequence of EEF1A1 mRNA.

Various tumours including prostate adenocarcinoma, colon adenocarcinoma, pancreatic cancer and gastric cancer

Note

A variant form of eEF1A1 lacking 67 aminoacids at the N-terminal region has been proposed to promote cell transformation and to sustain tumour cells viability. It has been named prostate inducing gene-1 (PTI-1), also known as elongation factor 1A-like 14. Initially identified by differential RNA display screening of cDNA expression library in the human prostate cancer cell line LNCaP, it has been proposed to act as a dominant oncogene in human prostatic adenocarcinoma. Its mRNA contains a 5'-UTR of 630 bp that is highly homologous to part of the 23S rRNA of Mycoplasma sp. fused to most of the CDS of EEF1A1, resulting in the substitution of its first 67 N-terminal residues with Met-Gln-Ser (Sun et al., 1997; Mansilla et al., 2005). Ectopically forced expression of PTI-1 in a mouse cell line induces tumours in nude mice and antisense PTI-1 molecule can reverse malignant phenotype of the transformed cells (Su et al., 1998). Silencing of PTI-1 by specific RNAi not affecting eEF1A1 expression, in a human prostate cancer cell line leads to a reduction of cellular growth, to the block of cell cycle in G1 phase and to the promotion of apoptosis (Yu et al., 2006). It has been hypothesized that PTI-1 could promote cell transformation by causing translational infidelity being in competition with eEF1A1 (Gopalkrishnan et al., 1999). PTI-1 mRNA is detected only in human cancer cells upon Mycoplasma infection. It remains under investigation whether PTI-1 can play a role in the natural history of human prostatic adenocarcinoma upon Mycoplasma infection. The origin of the chimeric transcript of PTI-1 remains to be ascertained (Scaggiante et al., 2008). PTI-1 mRNA has been detected in multidrug-resistance colon cancer cell line LoVoDX (Bertram et al., 1998). By using the detection of a unique PTI-1 region between the 5'UTR and the CDS, PTI-1 mRNA has been found in the human pancreatic cancer cell line AsPC-1, in the human gastric cancer TMK-1 cells, and in the hepatoma Alexander cells, but not in several other pancreatic, gastric and hepatoma human cancer cell lines. Interestingly, in AsPC-1 cells the down-regulation of K-ras mRNA by antisense leads to a reduction of PTI-1 level. PTI-1 mRNA was detected in three of five surgical human specimens of pancreatic cancer (Ohnami et al., 1999).

Cutaneous T-cell lymphoma

Note

In human sera derived from Cutaneous T-Cell Lymphoma (CTCL) patients one of the new tumour antigens was a truncated version of eEF1A1 lacking 77 aminoacids at N-terminal.

Leukemia

Note

In the promyelocytic human leukemia cells the differentiation agent All-Trans-Retinoic Acid (ATRA) induces down-regulation of eEF1A1 thus suggesting a role in contributing to cancer survival in haematopoietic malignancies (Harris et al., 2004).

An isoform of eEF1A1 with a more basic isoelectric point was identified in human haematopoietic cancer cell lines but not in normal lymphocytes raising the possibility that post-translation modifications of eEF1A1 could be involved in cancer development and progression of haematopoietic tumours (Dapas et al., 2003).

Felty's syndrome

Note

Characterized by rheumatoid arthritis, spleno-megaly and neutropenia. eEF1A1 is found as autoantigen (Ditzel et al., 2000).

Skeletal Muscle Trauma

Note

In hypercatabolic traumatized patients eEF1A1 mRNA level significantly rose in skeletal muscle as the result of injury. eEF1A1 expression correlated with overexpression of p66(ShcA) (Bosutti et al., 2007).

Cell transformation

Note

De-regulation of eEF1A1 in rodent cells exposed to chemical and physical carcinogens promotes cell transformation (Tatsuka et al., 1992). Over-expression of eEF1A1 in a non-transformed murine pro-B cell line confers selective resistance to apoptosis induced by endoplasmic reticulum stress, thus providing long-term viability (Talapatra et al., 2000).

References

Tatsuka M, Mitsui H, Wada M, Nagata A, Nojima H, Okayama H. Elongation factor-1 alpha gene determines susceptibility to transformation. Nature. 1992 Sep 24;359(6393):333-6

Knudsen SM, Frydenberg J, Clark BF, Leffers H. Tissue-dependent variation in the expression of elongation factor-1 alpha isoforms: isolation and characterisation of a cDNA encoding a novel variant of human elongation-factor 1 alpha. Eur J Biochem. 1993 Aug 1;215(3):549-54

Sun Y, Lin J, Katz AE, Fisher PB. Human prostatic carcinoma oncogene PTI-1 is expressed in human tumor cell lines and prostate carcinoma patient blood samples. Cancer Res. 1997 Jan 1;57(1):18-23

Bertram J, Palfner K, Hiddemann W, Kneba M. Overexpression of ribosomal proteins L4 and L5 and the putative alternative elongation factor PTI-1 in the doxorubicin resistant human colon cancer cell line LoVoDxR. Eur J Cancer. 1998 Apr;34(5):731-6

Su Z, Goldstein NI, Fisher PB. Antisense inhibition of the PTI-1 oncogene reverses cancer phenotypes. Proc Natl Acad Sci U S A. 1998 Feb 17;95(4):1764-9

Gopalkrishnan RV, Su ZZ, Goldstein NI, Fisher PB. Translational infidelity and human cancer: role of the PTI-1 oncogene. Int J Biochem Cell Biol. 1999 Jan;31(1):151-62

Ohnami S, Matsumoto N, Nakano M, Aoki K, Nagasaki K, Sugimura T, Terada M, Yoshida T. Identification of genes showing differential expression in antisense K-ras-transduced pancreatic cancer cells with suppressed tumorigenicity. Cancer Res. 1999 Nov 1;59(21):5565-71

Ditzel HJ, Masaki Y, Nielsen H, Farnaes L, Burton DR. Cloning and expression of a novel human antibody-antigen pair associated with Felty's syndrome. Proc Natl Acad Sci U S A. 2000 Aug 1;97(16):9234-9

Izawa T, Fukata Y, Kimura T, Iwamatsu A, Dohi K, Kaibuchi K. Elongation factor-1 alpha is a novel substrate of rho-associated kinase. Biochem Biophys Res Commun. 2000 Nov 11;278(1):72-8

Johnsson A, Zeelenberg I, Min Y, Hilinski J, Berry C, Howell SB, Los G. Identification of genes differentially expressed in association with acquired cisplatin resistance. Br J Cancer. 2000 Oct;83(8):1047-54

Hotokezaka Y, Tobben U, Hotokezaka H, Van Leyen K, Beatrix B, Smith DH, Nakamura T, Wiedmann M. Interaction of the eukaryotic elongation factor 1A with newly synthesized polypeptides. J Biol Chem. 2002 May 24;277(21):18545-51

Talapatra S, Wagner JD, Thompson CB. Elongation factor-1 alpha is a selective regulator of growth factor withdrawal and ER stress-induced apoptosis. Cell Death Differ. 2002 Aug;9(8):856-61

Dapas B, Tell G, Scaloni A, Pines A, Ferrara L, Quadrifoglio F, Scaggiante B. Identification of different isoforms of eEF1A in the nuclear fraction of human T-lymphoblastic cancer cell line specifically binding to aptameric cytotoxic GT oligomers. Eur J Biochem. 2003 Aug;270(15):3251-62

Zhu G, Reynolds L, Crnogorac-Jurcevic T, Gillett CE, Dublin EA, Marshall JF, Barnes D, D'Arrigo C, Van Trappen PO, Lemoine NR, Hart IR. Combination of microdissection and microarray analysis to identify gene expression changes between differentially located tumour cells in breast cancer. Oncogene. 2003 Jun 12;22(24):3742-8

Harris MN, Ozpolat B, Abdi F, Gu S, Legler A, Mawuenyega KG, Tirado-Gomez M, Lopez-Berestein G, Chen X. Comparative proteomic analysis of all-trans-retinoic acid treatment reveals systematic posttranscriptional control mechanisms in acute promyelocytic leukemia. Blood. 2004 Sep 1;104(5):1314-23

Hartmann TB, Thiel D, Dummer R, Schadendorf D, Eichmüller S. SEREX identification of new tumour-associated antigens in cutaneous T-cell lymphoma. Br J Dermatol. 2004 Feb;150(2):252-8

Lukash TO, Turkivska HV, Negrutskii BS, El'skaya AV. Chaperone-like activity of mammalian elongation factor eEF1A: renaturation of aminoacyl-tRNA synthetases. Int J Biochem Cell Biol. 2004 Jul;36(7):1341-7

Chuang SM, Madura K. Saccharomyces cerevisiae Ub-conjugating enzyme Ubc4 binds the proteasome in the presence of translationally damaged proteins. Genetics. 2005 Dec;171(4):1477-84

Gonçalves J, Malta-Vacas J, Louis M, Brault L, Bagrel D, Monteiro C, Brito M. Modulation of translation factor's gene expression by histone deacetylase inhibitors in breast cancer cells. Clin Chem Lab Med. 2005;43(2):151-6

Gross SR, Kinzy TG. Translation elongation factor 1A is essential for regulation of the actin cytoskeleton and cell morphology. Nat Struct Mol Biol. 2005 Sep;12(9):772-8

Mansilla F, Hansen LL, Jakobsen H, Kjeldgaard NO, Clark BF, Knudsen CR. Deconstructing PTI-1: PTI-1 is a truncated, but not mutated, form of translation elongatin factor 1A1, eEF1A1. Biochim Biophys Acta. 2005 Feb 14;1727(2):116-24

Borradaile NM, Buhman KK, Listenberger LL, Magee CJ, Morimoto ET, Ory DS, Schaffer JE. A critical role for eukaryotic elongation factor 1A-1 in lipotoxic cell death. Mol Biol Cell. 2006 Feb;17(2):770-8

McClatchy DB, Fang G, Levey AI. Elongation factor 1A family regulates the recycling of the M4 muscarinic acetylcholine receptor. Neurochem Res. 2006 Jul;31(7):975-88

Rho SB, Park YG, Park K, Lee SH, Lee JH. A novel cervical cancer suppressor 3 (CCS-3) interacts with the BTB domain of PLZF and inhibits the cell growth by inducing apoptosis. FEBS Lett. 2006 Jul 24;580(17):4073-80

Shamovsky I, Ivannikov M, Kandel ES, Gershon D, Nudler E. RNA-mediated response to heat shock in mammalian cells. Nature. 2006 Mar 23;440(7083):556-60

Yu L, Wu G, Wang L, Wang H, Zhang G. Transient reduction of PTI-1 expression by short interfering RNAs inhibits the growth of human prostate cancer cell lines. Tohoku J Exp Med. 2006 Jun;209(2):141-8

Zhou X, Temam S, Oh M, Pungpravat N, Huang BL, Mao L, Wong DT. Global expression-based classification of lymph node metastasis and extracapsular spread of oral tongue squamous cell carcinoma. Neoplasia. 2006 Nov;8(11):925-32

Bosutti A, Scaggiante B, Grassi G, Guarnieri G, Biolo G. Overexpression of the elongation factor 1A1 relates to muscle proteolysis and proapoptotic p66(ShcA) gene transcription in hypercatabolic trauma patients. Metabolism. 2007 Dec;56(12):1629-34

Frum R, Busby SA, Ramamoorthy M, Deb S, Shabanowitz J, Hunt DF, Deb SP. HDM2-binding partners: interaction with translation elongation factor EF1alpha. J Proteome Res. 2007 Apr;6(4):1410-7

Grassi G, Scaggiante B, Farra R, Dapas B, Agostini F, Baiz D, Rosso N, Tiribelli C. The expression levels of the translational factors eEF1A 1/2 correlate with cell growth

but not apoptosis in hepatocellular carcinoma cell lines with different differentiation grade. Biochimie. 2007 Dec;89(12):1544-52

Mishra AK, Gangwani L, Davis RJ, Lambright DG. Structural insights into the interaction of the evolutionarily conserved ZPR1 domain tandem with eukaryotic EF1A, receptors, and SMN complexes. Proc Natl Acad Sci U S A. 2007 Aug 28;104(35):13930-5

Kido T, Lau YF. The human Y-encoded testis-specific protein interacts functionally with eukaryotic translation elongation factor eEF1A, a putative oncoprotein. Int J Cancer. 2008 Oct 1;123(7):1573-85

Koiwai K, Maezawa S, Hayano T, Iitsuka M, Koiwai O. BPOZ-2 directly binds to eEF1A1 to promote eEF1A1 ubiquitylation and degradation and prevent translation. Genes Cells. 2008 Jun;13(6):593-607

Leclercq TM, Moretti PA, Vadas MA, Pitson SM. Eukaryotic elongation factor 1A interacts with sphingosine kinase and directly enhances its catalytic activity. J Biol Chem. 2008 Apr 11;283(15):9606-14

Scaggiante B, Bonin S, Cristiano L, Siracusano S, Stanta G, Dapas B, Giansante C, Fiotti N, Grassi G. Prostate-tumor-inducing gene-1 analysis in human prostate cancer cells and tissue in relation to Mycoplasma infection. Cancer Invest. 2008 Oct;26(8):800-8

Yan G, You B, Chen SP, Liao JK, Sun J. Tumor necrosis factor-alpha downregulates endothelial nitric oxide synthase mRNA stability via translation elongation factor 1-alpha 1. Circ Res. 2008 Sep 12;103(6):591-7

Kobayashi Y, Yonehara S. Novel cell death by downregulation of eEF1A1 expression in tetraploids. Cell Death Differ. 2009 Jan;16(1):139-50

Zhang J, Guo H, Mi Z, Gao C, Bhattacharya S, Li J, Kuo PC. EF1A1-actin interactions alter mRNA stability to determine differential osteopontin expression in HepG2 and Hep3B cells. Exp Cell Res. 2009 Jan 15;315(2):304-12

This article should be referenced as such:

Scaggiante B, Manzini G. EEF1A1 (eukaryotic translation elongation factor 1 alpha 1). Atlas Genet Cytogenet Oncol Haematol. 2010; 14(4):377-382.