EEF1E1 (eukaryotic translation elongation factor 1 epsilon 1)

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Abstract

Eukaryotic translation elongation factor 1 epsilon 1, alias EEF1E1, is a protein-coding gene that plays a role in the elongation step of translation. In particular, it is an auxiliary component of the macromolecular aminoacyl-tRNA synthase complex (MARS). Its expression is found frequently altered in human cancer cells and it is considered a putative tumor suppressor gene. This review collects the data on DNA/RNA, the protein encoded and the diseases where EEF1E1 is involved.

Keywords

EEF1E1; eukaryotic translation elongation factor 1 epsilon 1; AIMP3; p18; Translation; Translation elongation factor; protein synthesis; cancer; oncogene; cancer marker

Identity

Other names: P18, AIMP3, ARS-interacting multifunctional protein 3, Multisynthase Complex Auxiliary Component P18, Elongation Factor P18

HGNC (Hugo): EEF1E1

Location: 6p24.3

Figure. 1. EEF1E1 gene and splicing variants/isoforms. The figure shows the locus on chromosome 6 of the EEF1E1 gene (reworked from https://www.ncbi.nlm.nih.gov/gene; http://grch37.ensembl.org; www.genecards.org)
**DNA/RNA**

**Description**

EEF1E1 (eukaryotic translation elongation factor 1 epsilon 1) was identified for the first time by Mao and colleagues in 1998 (Mao et al, 1998). EEF1E1 is a protein-coding gene that starts at 8,079,395 nt and ends at 8,102,595 nt from pter. It has a length of 23,201 bp, counts 5 exons, and the current reference sequence is NC_000006.12. It is proximal to BLOC1S5 (biogenesis of lysosomal organelles complex 1 subunit 5, alias MUTED) gene. Read-through transcription exists between BLOC1S5 gene and EEF1E1 gene with forming the read-through transcript EEF1E1-BLOC1S5 (alias EEF1E1-MUTED) that is a candidate for nonsense-mediated mRNA decay (NMD) and it is unlikely to produce a protein product (He et al, 2018). Near to the genomic sequence of EEF1E1 there is a strong promoter transcriptional element that is located at +1.0 kb. Enhancer transcriptional elements are located at +22.0 Kb and at +18.1 Kb respectively.

**Transcription**

Alternative splicing for EEF1E1 brings to multiple transcript variants. In addition, a read-through transcription is known between EEF1E1 and the neighboring downstream MUTED (muted homolog) gene.

Two main alternative splicing transcript variants for EEF1E1 were detected although several others were reported. In addition, it was speculated the presence of six protein isoforms, but only two are properly described, i.e. the isoform 1 of 174 residues and the isoform 2 that counts 139 residues.

**Pseudogene**

According to Entrez Gene, the analysis of the human genome revealed the presence of an EEF1E1-related pseudogene on chromosome 2. This pseudogene was appointed as eukaryotic translation elongation factor 1 epsilon 1 pseudogene 1, alias EEF1E1P1 and it is classified as a processed pseudogene (http://www.ensembl.org/index.html). Its gene ID is 100130388, its reference is NC_000002.12, and its location is 2q13. EEF1E1P1 starts at 111,887,890 nt and ends at 111,889,485 nt with a length of 1,596 nt.

| Name        | Varian t | RefSeq (1) | Transcript ID | Exons | Type             | Length (bp) | Isomorph | Alias     | RefSeq (2) | Length (aa) | MW (kDa) | pI     |
|-------------|----------|------------|---------------|-------|------------------|-------------|----------|-----------|------------|-------------|----------|--------|
| EEF1E1      | -        | -          | ENST0000048826.2 | 2     | protein coding   | 443         | -        | -         | -          | 94          | (?)      | (?)    |
| EEF1E1      | Var.1    | NM_004280.5 | ENST00000379715.5 | 4     | protein coding   | 1077        | isoform 1| O43324   | NP_004271.1| 174         | 19.8     | 8.5    |
| EEF1E1      | -        | -          | ENST00000507463.1 | 3     | protein coding   | 654         | -        | -         | -          | 150         | 16.6     | (?)    |
| EEF1E1      | Var.2    | NM_001135650.2 | ENST00000429723.2 | 4     | protein coding   | 562         | isoform 2| O43324   | NP_001129122.1 | 139       | 15.5     | 7.9    |
| EEF1E1      | -        | -          | ENST00000502429.1 | 4     | protein coding   | 591         | -        | -         | -          | 136         | (?)      | (?)    |
| EEF1E1      | -        | -          | ENST00000515633.1 | 3     | protein coding   | 460         | -        | -         | -          | 56          | 5.89     | (?)    |

Table 1: Alternative splicing variants and isoforms of EEF1E1. (reworked from http://grch37.ensembl.org; https://www.ncbi.nlm.nih.gov; https://web.expasy.org/protparam; https://www.uniprot.org). ncRNA = non-coding RNA; nonsense md = nonsense mediated decay; (?) = undetermined; MW = molecular weight; pl = theoretical pl.
It virtually encodes a non-coding transcript of 430 bp named EEF1E1P1-201 (Ensembl Ref: ENST00000446998.2). The real presence of this transcript and its possible role in the cell are totally unknown.

If EEF1E1P1 has any regulatory role in the expression of the respective gene as described for others (Hirotsune et al., 2003), is only speculation in the absence of experimental evidence. Currently, there is no evidence about the involvement of this pseudogene in human cancers or in other diseases.

**Description**

The eukaryotic translation elongation factor 1 epsilon 1 (alias eEF1E1, p18, AIMP3) is the smallest component of the Multiaminoacyl-tRNA Synthetase complex (alias MARS). The exact position of EEF1E1 in the MARS complex is still unknown. However, it seems to be localized on the surface of the MARS complex and it seems to interact with the eEF1H complex (Deineko V.V., 2008).

EEF1E1 is a small globular protein with a length of 174 amino acids and a molecular weight of 19.8 kDa. eEF1E1 shows strong sequence similarity with eukaryotic translation elongation factor 1 beta 2 (EEF1B2) and eukaryotic translation elongation factor 1 gamma (EEF1G) (Quevillon and Mirande, 1996) and with the N-terminal sequence of valyl-tRNA synthetase (Deineko V.V., 2008).

eEF1E1 shows many domains in both isoforms: the amino half terminal is unique for both isoforms and shows an N-terminal-like domain not well characterized followed by a linker domain, while the major differences between the two isoforms are in the carboxyl half terminal. In fact, in the carboxyl half terminal of isoform 1 there are reported two domain overlapping, i.e. a Glutathione S-transferase C-terminal-like domain (GST_C_AIMP3), folded in alpha-helical, and a more general and not well characterized C-terminal domain.

In isoform 2, there is a unique region called C-terminal domain of the Glutathione S-transferase family (GST_C_family). The fold of this domain is alpha-helical (see figure 2).

EEF1E1 interacts with other members of the MARS complex and one interactional model was proposed (Mirande, 2017) although its exact interactions need to be still clarified.

**Post-translational modifications.** Some post-translational modifications are observed, such as phosphorylation and acetylation (https://www.ncbi.nlm.nih.gov).

**Expression**

eEF1E1 is expressed widely in human tissues and normal cells (https://www.genecards.org; https://www.proteinatlas.org/ENSG00000124802-EEF1E1/tissue) while its expression is altered in many cancer types. Frequently it is downregulated in various cancer tissues (Park et al., 2005). Cells that show overexpression of eEF1E1 show an acceleration of senescence and also defects in nuclear morphology (Oh et al, 2010).

**Localisation**

EEF1E1 is located mostly in the cytoplasm but it was also found in the nucleus.
Function

It is well known that in eukaryotic cells the various components of translation machinery are properly organized into two main multienzyme structures: eEF1H (macromolecular eukaryotic translation elongation factor-1 complex), formed by the translation elongation factors (EEF1B2, EEF1D, EEF1G) and VARS (valyl-tRNA synthetase), and MARS (Multiaminoacyl-tRNA Synthetase complex or multi-tRNA synthetase complex, alias MSC), formed by nine aminoacyl- tRNA synthetases (AARSs) specific for amino acids Glu, Pro (EPRS1 (glutaminylprolyl-tRNA synthetase)), Ile (IARS1), Leu (LARS1), Met (MARS1, methionyl-tRNA synthetase), Gln (QARS1), Lys (KARS1), Arg (RARS1), and Asp (DARS1) and other auxiliary non-synthetase protein components, called also aminoacyl-tRNA synthetase (ARS)-interacting multifunctional proteins (AIMPs), i.e. AIMP1 (p43), AIMP2 (p38) and eEF1E1 (AIMP3, alias p18) (Cho et al., 2015; Shalak et al., 2007; Quevillon and Mirande, 1996).

It is well known that in eukaryotic cells the various components of translation machinery. Therefore, the main canonical function of eEF1E1 is to play a role as an auxiliary component of the macromolecular aminoacyl-tRNA synthetases complex in the elongation step of translation, in particular, it interacts with several aminoacyl-tRNA synthetases (Tao et al., 2017) and it could contribute to the anchorage of MARS complex to EF1H complex (Quevillon and Mirande, 1996).

Other functions (non-canonical roles): in addition to what has already been said, it seems to play a role in embryonic development of the mammalian face and other structures (Fowles et al., 2003). eEF1E1 has the ability to translocate into the nucleus in response to DNA damage where it has a role in the DNA damage response in association with serine/threonine kinases ATM / ATR and TP53. In fact, it was found a positive relationship between expression levels of eEF1E1 and TP53, i.e. high expression levels of eEF1E1 are correlated with elevated TP53 levels, while eEF1E1 depletion leads to the block of TP53 induction (Park et al., 2005). The eEF1E1 loss-of-function phenotype leads to various kinds of abnormalities: in particular, one allele inactivation increases the susceptibility to spontaneous tumors while the inactivation of both eEF1E1 alleles caused embryonic lethality (Park et al., 2005). The importance of eEF1E1 in embryogenesis is previously reported (Fowles et al., 2003) while in the context of the tumors, eEF1E1 could be a haploinsufficient tumor suppressor (Park et al., 2005) that can accelerate cellular senescence (Kang et al., 2012).

eEF1E1 in involved in the degradation of mature Lamin A (LMNA) which is a major component of the nuclear envelope matrix (Tao et al., 2017).

Homology

eEF1E1 is highly conserved and its homology between the species is reported in Table.2
Mutations

A great number of mutations in the genomic sequence and in the amino acid sequence for EEF1E1 were discovered in cancer cells that are obviously genetically more unstable respect normal ones. However, depletion of EEF1E1 causes itself genomic instability in cells (Kim et al, 2018) and makes the cells susceptible to transformation by single oncogenes (Park et al, 2006).

The genomic alterations observed include also the formation of novel fusion genes. However, there are no sufficient experimental data yet to understand the repercussions on cellular behavior and so the implications in cancer of these alterations.

Implicated in

Top note

A different expression level of EEF1E1 was observed in many cancer types compared to noncancerous control tissue. It is considered as a putative tumor suppressor, in particular for its downregulation in gastric and colorectal cancers (Kim et al, 2011). In fact, high EEF1E1 expression seems to be related to better survival in these two tumor types (Hassan et al, 2018).

However, Hassan et colleagues reported that EEF1E1 is overexpressed in many other cancer types such as breast, lung, gastric, prostate, colorectal and liver tumors and this fact could predict poor survival (breast, lung, liver)(Hassan et al, 2018).

Interesting is the role of inorganic arsenic (iAs) in the epigenetic alteration of DNA methylation in arsenic-induced diseases such as cancer of the bladder, kidney, lung, liver, and prostate. It was revealed that EEF1E1 is one of many genes silenced and involved in iAs related-hypermethylation in an arsenic-methylated tumor suppressorome (Smeester et al, 2011).

In addition, eEF1E1 is involved in some genomic translocations with the creation of numerous fusion genes (Table.3).
Ankylosing spondylitis

It is found that the expression levels of EEF1E1 are significantly upregulated in whole blood of ankylosing spondylitis (AS) patients respect control group and these findings could be attributed to genetic mutations on EEF1E1 gene. This may have an important significance in the pathogenesis of AS because eEF1E1 may be involved in AS-related inflammation by upregulating TP53 and pro-inflammatory cytokines. This may suggest the use of EEF1E1 as an underlying genetic biomarker for the diagnosis of AS but other research are needed to determine the exact role of eEF1E1 overexpression in AS (Fan et al, 2019).

Autism spectrum disorders

EEF1E1 appears in research on developmental delay and autism spectrum disorders focused on deletions in chromosome 6p22.3-p24.3 (Celestino-Soper et al, 2012). However, is still not clear its role in these diseases.

Bladder cancers

In general, eEF1E1 is found to be down-regulated in bladder cancers. eEF1E1 is expressed at moderate and high levels in all normal urothelium tissues while only a part of bladder cancers shows this expression's pattern. The loss of EEF1E1 expression is more evident in late-stage (≥ T2) bladder tumours and can be associated with survival in muscle-invasive bladder cancers (MIBC) patients following radiotherapy (Gurung et al, 2015).

Brain and central nervous system (CNS) cancers

EEF1E1 is upregulated in astrocytoma and oligodendroglioma while for glioblastoma and glioma no significant difference in expression levels was observed. High levels of EEF1E1 could be predicted better survival outcomes (Hassan et al, 2018). In addition, one genomic alteration was observed both in astrocytoma and glioblastoma, i.e. the fusion gene t(6;6)(p24;p24) RREB1 /EEF1E1 (Gao et al, 2018; Yoshihara et al, 2015). There are no data about the respective chimeric transcript or protein and so this genomic alteration is still poorly understood.

Breast cancer

Currently, for EEF1E1 there is no significant difference in expression, between breast cancer and normal breast tissues (Hassan et al, 2018; Guglielmi et al, 2015; Gao et al, 2018). There are no reference

Table 3 EEF1E1 rearrangements: translocations and fusion genes

| Name | 5’ end | 3’ end | Loc1 | Loc2 | Description | Type | Disease | Organ | Code | Ref. |
|------|--------|--------|------|------|------------|------|---------|-------|------|------|
| CDYL/EEF1E1 | CDYL | EEF1E1 | 6p24.3 | 6p24.3 | t(6;6)(p25;p24) | Translocation | Malignant melanoma | Skin | SKCM | 1 |
| EEF1E1/AEBP2 | EEF1E1 | AEBP2 | 6p24.3 | 12p12.3 | t(6;12)(p24;p12) | Translocation | - | - | - |
| EEF1E1-BLOC1S5 | EEF1E1 | BLOC1S5 | 6p24.3 | 6p24.3 | Readthrough transcription | Fusion gene | - | - | - |
| EEF1E1/CD2AP | EEF1E1 | CD2AP | 6p24.3 | 6p12.3 | t(6;6)(p24;p12) | Translocation | - | - | - |
| EEF1E1/DSC2 | EEF1E1 | DSC2 | 6p24.3 | 18q12.1 | t(6;18)(p24;q12) | Translocation | - | - | - |
| EEF1E1/EYS | EEF1E1 | EYS | 6p24.3 | 6q12 | t(6;6)(p24;q12) | Translocation | Adenocarcinoma | Breast | BRCA | 2 |
| EEF1E1/KRTDAP | EEF1E1 | KRTDAP | 6p24.3 | 19q13.12 | t(6;19)(p24;q13) | Translocation | - | - | - |
| EEF1E1/NSMCE4A | EEF1E1 | NSMCE4A | 6p24.3 | 10q26.13 | t(6;10)(p24;q26) | Translocation | - | - | - |
| EEF1E1/RAB23 | EEF1E1 | RAB23 | 6p24.3 | 6p12.1 | t(6;12)(p24;p12) | Translocation | - | - | - |
| EEF1E1/REB1 | EEF1E1 | REB1 | 6p24.3 | 6p24.3 | t(6;6)(p24;p24) | Fusion gene | - | - | - |
| INPP4A/EEF1E1 | INPP4A | EEF1E1 | 2q11 | 6p24.3 | t(2;6)(q11;p24) | Translocation | Mesenchymal tumor, NOS | - | - | 1 |
| PSMG4/EEF1E1 | PSMG4 | EEF1E1 | 6p25 | 6p24.3 | t(6;6)(p25;q24) | Translocation | Adenocarcinoma | Breast | BRCA | 1 |
| RREB1/EEF1E1 | RREB1 | EEF1E1 | 6p24.3 | 6p24.3 | t(6;6)(p24;q24) | Fusion gene | Astrocytoma, grade III-IV/Glioblastoma | Central Nervous System | GBM | 2.3 |
| TG/EEF1E1 | TG | EEF1E1 | 8q24.22 | 6p24.3 | t(6;6)(p24;q24) | Translocation | - | - | - |

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t(6;6)(p24;q12) EEF1E1/ EYS and t(6;6)(p25;p24) PSMG4/EEF1E1 (Hu et al., 2018). There are no data about the respective chimeric transcripts or proteins and so these genomic alterations are still poorly understood.

**Colorectal cancer**

EEF1E1 was found to be upregulated in rectal mucinous adenocarcinoma subtype and, in general in colorectal cancers compared to normal tissues. A reduction of its expression level correlates with a worst prognosis and poor survival (Hassan et al., 2018). Other studies found that normal colon mucosa expressed EEF1E1 in nearly all of the cases while EEF1E1 expression is significantly decreased in the majority of colorectal cancer (CRC) cases. This suggests that the downregulation of EEF1E1 may be related to inactivation of its tumour suppressor function and so might play a role in the development of CRC (Chen et al, 2018; Kim et al, 2011).

**Gastric cancer**

In gastric cancers is found an upregulation of EEF1E1 transcript and this predicts a better overall survival (OS) and first progression (FP)(Hassan et al., 2018). Other studies revealed that normal gastric mucosa expressed EEF1E1 in nearly all of the cases while EEF1E1 expression is significantly decreased in the majority of gastric cancer (GC) cases. This suggests that the downregulation of EEF1E1 may be related to the inactivation of its tumour suppressor function and so might play a role in the development of GC (Kim et al, 2011).

**Head and neck squamous cell carcinoma (HNSC)**

EEF1E1 is found to be overexpressed in head and neck cancers (Hassan et al, 2018). Wiest and colleagues (Wiest et al, 2002) have found an interesting feature in the HPV16 infection in some samples of head and neck cancer in relation to EEF1E1. In detail, the integration site of E6/E7 region of HPV16 falls on chromosome 6 in the proximity of some human genes included EEF1E1. This could contribute to explain the oncogene property of HPV16 or one of the oncogenesis mechanisms of head and neck cancer. However, it is still unclear if the viral integration can affect the regulation of expression of EEF1E1.

**Kidney cancer**

EEF1E1 was found to be downregulated in chromophobe renal cell carcinoma and in kidney clear cell carcinoma (Hassan et al, 2018).

**Liver cancer**

It was documented that EEF1E1 is down-regulated in hepatocellular carcinoma (HCC)(Yu et al, 2017; Du et al, 2012). In particular, there is a high relation between the expression levels of highly up-regulated in liver cancer (HULC) long non-coding RNA and eEF1E1 in HCC. In fact, if HULC is over-expressed the expression levels of eEF1E1 fall down and on the contrary if expression levels of HULC decrease, eEF1E1 is expressed normally. EEF1E1 gene is in close proximity to HULC gene and with high probability the second can mediate the expression levels of the first (Yu et al, 2017). Other authors reported that EEF1E1 expression levels are higher in liver cancer and that this could predict worse survival although they did not tell precisely the cancer type (Hassan et al, 2018).

**Lung cancer**

EEF1E1 expression levels were reported to be high in small cell lung carcinoma, in squamous cell lung carcinoma subtypes, and in large cell lung carcinoma. These high expression levels seem to be correlated with poor overall survival (OS) and first progression (FP) in lung cancers (Hassan et al, 2018). In addition, were reported some somatic mutations for EEF1E1 gene in lung cancer cell lines (Kim et al, 2011).

**Lymphoma and other blood cancers**

EEF1E1 is found to be overexpressed in Burkitt's lymphoma and in diffuse large B-Cell lymphoma. On the contrary, it is found to be downregulated in marginal zone B-Cell lymphoma (Hassan et al, 2018), acute promyelocytic leukemia and chronic myelogenous leukemia (Gurung et al, 2015). In chronic myelogenous leukemia was observed some somatic mutations for EEF1E1 (Kim et al, 2011). In addition, EEF1E1 shows an increased expression in pyothorax-associated lymphoma (PAL), a lymphoma developing in long-standing inflammation (Nishiu et al, 2004).

**Ovarian cancer**

It is detected that EEF1E1 is frequently upregulated in ovarian serous adenocarcinoma (Hassan et al, 2018).

**Pancreatic cancer**

EEF1E1 expression levels were found to be significantly downregulated in pancreatic cancers (Hassan et al, 2008).

**Prostate cancer**

EEF1E1 is significantly overexpressed in prostate cancer. Currently, there is not sufficient data about the prognostic significance of the upregulation of EEF1E1 in prostate cancer (Hassan et al, 2018).

**To be noted**
HIV-1 interactions: It is reported that HIV-1 MA protein interacts with EEF1E1 in human HEK293 and Jurkat cell lines (Jäger et al, 2011) and that EEF1E1 is subject to cleavage by the HIV-1 protease (Impens et al, 2012).

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