**EBAG9 (Estrogen receptor-binding fragment-associated antigen 9)**

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**Identity**

**Hugo:** EBAG9  
**Other names:** EB9; PDAF; RCAS1  
**Location:** 8q23.2

**DNA/RNA**

**Description**
The EBAG9 gene contains 7 exons and 6 introns. It was predicted to span over approximately 24.6 kb of the genomic DNA with mRNA size approximately 1182 bp. The exon 3 was the smallest at 79 bp; the other exons ranged from 92-720 bp. The EBAG9 was isolated from MCF-7, human breast cancer cell library and it has been reported identical with RCAS1 (receptor-binding cancer antigen expressed on SiSo cells) gene from human uterine adenocarcinoma cell line.

**Transcription**
The EBAG9 mRNA is up-regulated by estrogen in MCF-7 cells and its promoter responds to estrogen through the complete palindromic estrogen responsive element (ERE) that was located in the 5'-up stream region of the gene.

**Pseudogene**
One pseudogene located in chromosome 10 associated with RCAS1/EBAG9.

**Protein**

**Description**
The EBAG9/RCAS1 consists of 213 amino acids (aa) corresponding to a molecular weight of 24.4 kDa. The EBAG9/RCAS1 has an N-terminal trans-membrane segment (8-27 aa) and a coiled-coil structure in the C-terminal portion (179-206 aa), indicating that the EBAG9/RCAS1 is a type II membrane protein able to form oligomers through the coiled-coil structure, which is expressed on the surfaces of human cancer cells.

**Expression**
The EBAG9/RCAS1 mRNA is expressed in ovary, testis, prostate, thymus, muscle, and heart. At the protein level the EBAG9/RCAS1 not detected in normal ovary tissues or any of the other above. Neither mRNA nor protein was detected in small intestine, colon, lymph node or peripheral blood lymphocytes.

**Localisation**
Mainly in the golgi, membrane and cytoplasm of cancer tissues, but its expression is very low or hardly detected in normal tissues.

**Function**
The biological functions of the EBAG9/RCAS1 secreted by non-cancerous tissues remain unknown. In cancer cells, the EBAG9/RCAS1 is a ligand for a putative receptor present on various human cell lines and normal peripheral lymphocytes such as T-, B- and natural killer (NK)-cells. The expression of this receptor is enhanced by activation of these lymphocytes. The EBAG9/RCAS1 acts to inhibit the growth of receptor-binding cells and induced apoptotic cell death. Over-expression of the EBAG9/RCAS1 is known to inhibit the growth and induced apoptosis of immune cells. As the results, cancer cells might evade immune surveillance by expressing the EBAG9/RCAS1 and inducing the apoptosis of the EBAG9/RCAS1 receptor-positive immune cells.
**Homology**

Mouse and human EBAG9/RCAS1 shows a high degree of homology at the amino acid level (98%). Mouse (Mus musculus) ebag9 gene spans about 30 kb in genomic DNA and consists of 7 exons. Dog (Canis familiaris) EBAG9/RCAS1 also shows highly homologues to human (96.2%) and to mouse (96.7%). For chimpanzee (Pan troglodytes) 100%, rat (Rattus norvegicus) 94% and chicken-ebag9 (Gallus gallus) 91%.

**Mutations**

**Germinal**

Not known in Homo sapiens.

**Somatic**

Not known in Homo sapiens.

**Implicated in**

**Immunity**

**Note:** During pregnancy, EBAG9/RCAS1 may play a role in the down-regulation of the maternal immune response and may participate in the initiation of the labor. In the healthy women, higher EBAG9/RCAS1 expression was observed in the periovulatory and the secretory menstrual cycle phases than in the proliferation phase. The changes in EBAG9/RCAS1 expression were combined with significant differences in the number of immune cells and their activity. It suggested that EBAG9/RCAS1 endometrial expression may favor the coexistence of active lymphocytes and endometrial cells.

**Disease**

The elevated serum level of EBAG9/RCAS1 reported to be associated with a poor immunological prognosis in HIV-1-infected patients, and also associated with the apoptosis of CD4+ T cells in HIV infection. In addition, the induction and secretion of EBAG9/RCAS1 in HIV-Trans-acting transcriptional activator-stimulated CD4+ T cells and monocytes suggested that EBAG9/RCAS1 may involved in the CD4+ T cell apoptosis observed in HIV-1 infection along with FasL and TRAIL.

**Malignancy.**

**Disease**

The EBAG9/RCAS1 reported to be over-expressed in many human cancers. Among them: breast, female-genital, gastrointestinal, blood, lung, pancreas, liver, renal, biliary-tract, hepatic, prostate, thyroid, gall bladder, and brain cancer.

**Prognosis**

The EBAG9/RCAS1 over-expression could be used as a predictor of poor prognosis in malignant diseases.

**Oncogenesis**

The EBAG9/RCAS1 plays a role in the immune escape of cancer cells. The EBAG9/RCAS1 could help cancer cells to survive and avoid immunosurveillance. This gene over-expression might cause progression, invasion and metastasis. The EBAG9 acts as one of the estrogen responsive genes in estrogen receptor-positive tumors and mediate estrogen function. Overall, the EBAG9/RCAS1 has an etiological role in the development and progression of cancer cells.

**References**


This article should be referenced as such: