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Gene Section

Review

ALCAM (Activated Leukocyte Cell Adhesion Molecule)

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Abstract

ALCAM (Activated Leukocyte Cell Adhesion Molecule), also known as CD166 (cluster of differentiation 166), is a member of a subfamily of immunoglobulin receptors with five immunoglobulin-like domains (VVC2C2C2) in the extracellular domain.

Keywords
ALCAM, CD166, cell adhesion

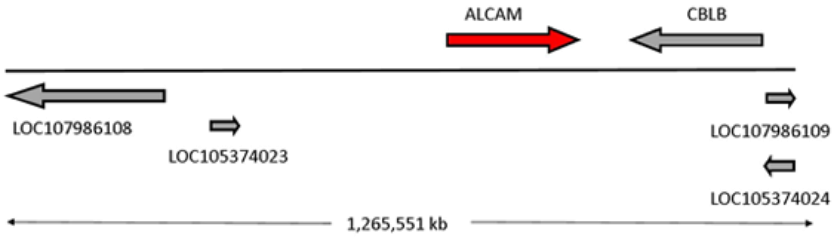
Identity

Other names
CD166 (cluster of differentiation 166), MEMD

HGNC (Hugo)
ALCAM

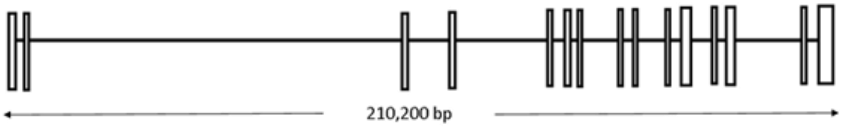
Location
3q13.11

Local order
From centromere to telomere: LOC107986108, LOC105374023, ALCAM, CBLB, LOC107986109, LOC105374024



Local order of ALCAM is shown together with leading and subsequent genes on chromosome 3. The direction of arrows indicates transcriptional direction on the chromosome and arrow sizes approximate gene sizes.

DNA/RNA



Exons are shown as boxes, introns are shown as lines.

Description

ALCAM gene is 210,200 bp long and resides on the positive strand on DNA. The gene has a total of 16 exons.

Transcription

ALCAM gene codes for 9 transcripts, 4 of them are protein coding (4701, 4189, 2496, 1818 bp) and 5 of them are non-protein coding transcripts (2845, 1770, 1242, 783, 564 bp).

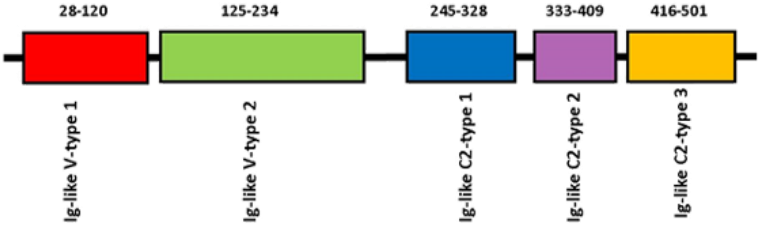
Pseudogene

No reported pseudogenes for ALCAM.

Protein

Note

ALCAM is a glycoprotein of the immunoglobulin superfamily and has an estimated molecular weight of 100-105 kDa.



Domains are shown as bars.

Expression

ALCAM is expressed in almost every tissue. Higher expression is observed in parathyroid gland. Tissues with medium ALCAM expression are cerebral cortex, cerebellum, thyroid gland, tonsil, nasopharynx, bronchus, lung, liver, gallbladder, pancreas, salivary gland, stomach, urinary bladder, kidney, epididymis, prostate, seminal vesicle, breast, cervix, uterine, endometrium and fallopian tube. Tissues with low expression are hippocampus, caudate, adrenal gland, appendix, lymph node, oral mucosa, rectum, duodenum, small intestine, colon, ovary, placenta, soft tissue and skin. Spleen, bone marrow, heart muscle, smooth muscle, skeletal muscle, esophagus, testis, vagina and adipose tissue have no ALCAM expression (The Human Protein Atlas, http://www.proteinatlas.org/).

Localisation

ALCAM protein is localized to the cell membrane (Bowen et al., 1995).

Function

ALCAM is a type-I transmembrane protein, which belongs to the immunoglobulin superfamily (Bowen et al., 1995). It constitutes 5 immunoglobulin-like domains and a short COOH-terminal cytoplasmic tail (Weidle et al., 2010). ALCAM was first shown as a ligand of CD6 on activated leukocytes (Whitney et al., 1995). Then, it was found on hematopoietic stem cells and myeloid progenitors (Swart, 2002). ALCAM-CD6 interaction mediates T-cell activation or proliferation. In addition, ALCAM-ALCAM homophilic interaction contributes to cellular changes, such as angiogenesis, immune response and cell migration during the neuronal development (van Kempen et al., 2001). The expression of ALCAM was found during organ development in the central and peripheral nervous system, sensory organs, hematopoiesis, endothelial and epithelial lineage (Swart, 2002). Furthermore, a short ALCAM transcript leads to a soluble isoform of ALCAM (sALCAM). This isoform has only one immunoglobulin-like domain (D1), which is important for ligand binding (Ikeda and Quertermous, 2004). The roles of sALCAM were considered to block hemophilic binding of ALCAM and endogenous ALCAM function (Swart, 2002). Moreover, sALCAM was also shown to inhibit tumor progression in melanoma cells (van Kilsdonk et al., 2008).

Mutations

Note

There are only a few reported mutations in ALCAM. One of the known cases is the deletion of ALCAM at 3q13.1 in a keratocystic odontogenic tumor. This mutation is thought to contribute to a matrix metalloproteinase-mediated proteolytic cascade, leading to tumor growth (Heikinheimo et al., 2007). In addition, it was reported that deletion of ALCAM might be related to craniofacial abnormalities (Simovich et al., 2008).

Epigenetics

ALCAM promoter contains several cis-acting elements including RELA (p65 NF-kB) binding motif. This motif has several CpG residues and it is highly methylated in breast cancer cells, resulting

with loss of ALCAM expression (King et al., 2010). Hypermethylation of ALCAM promoter was also observed in preeclampsia placentas (Yeung et al., 2016).

Implicated in

Acute Myeloid Leukemia (AML)

CD166/ALCAM protein levels were reported to be significantly upregulated in AML cell lines and AML blasts of some patients (Strassberger et.al., 2014).

Brain Tumors

ALCAM was reported to be enriched in brain tumor cells to promote invasion (Kijima et.al., 2012). It was suggested that ALCAM/ALCAM interaction may play an important role in early stages of metastasis seeding in the brain (Soto et.al., 2014).

Breast Cancer

Loss of ALCAM expression was found to increase the invasiveness of breast cancer cells and reduced levels of ALCAM was associated with poor prognosis. ALCAM overexpression caused cell growth reduction and a decrease in adhesion to matrix and migration. Low levels of ALCAM in breast cancer cells have been linked to bone matrix adhesion, which leads to bone metastasis (Davies S. and Jiang W.G., 2010). It was suggested that high ALCAM levels were seen in normal and cancerous breast epithelial cells and loss of ALCAM expression is associated with tumor progression, accelerated cell proliferation and decrease in survival (Brandt et.al., 2014). Furthermore, low expression of ALCAM contributes to a more aggressive phenotype among African-American women (Tan et.al., 2014). Interestingly, elevated serum levels of ALCAM was found to be correlated with aggressive tumor behavior in breast cancer patients indicating that serum ALCAM may be used as a biomarker (Witzel et.al., 2012).

Chordoma

ALCAM is a positive biomarker for chordoma and might play an important role for chordoma relapse (Chen et.al., 2015).

Cholangiocarcinoma

ALCAM protein is expressed on the membranes of invasive Cholangiocarcinoma cells so that might be used as a biomarker for diagnosis, prognosis and therapy (Adisakwattana et.al., 2015).

Endometrial Cancer

ALCAM is an early stage endometrioid endometrial cancer biomarker and important in regulation of cell migration, invasion and metastasis of endometrial cancer (Devis et.al., 2016).

Gall Bladder Cancer

SNPs within ALCAM (ALCAM rs1157G>A) gene, may play a role in GBC susceptibility (Yadav et.al., 2015).

Gastric Cancer

Elevated levels of miR-9 may lead to ALCAM mRNA level reduction in SGC-7901 human gastric cancer cells (Ye et.al., 2004).

Colorectal Cancer

PROM1 (CD133) and ALCAM co-localization is seen during early stages of colon tumorigenesis, indicating well differentiation (Margaritescu et.al., 2014). ALCAM expression was detected in colon CSCs in humans and also in premalignant adenomatous polyps (Leavell et.al., 2012). Difference in cellular location of ALCAM cause differences in prognostic value and cytoplasmic expression is associated with worse prognosis outcome and CD166 expression of it, is an indicator of advanced T category and N-positive status in colorectal cancer (Ni et.al., 2013).

Liver Cancer

ALCAM and CD44 promote tumorigenesis of liver cells and might be important for therapy strategies (Ma et.al., 2014). It was also shown that the ALCAM/AKT axis regulates tumorigenesis by post-translational modifications and cytosolic accumulation of FOXO proteins in hepatoma cells (Yu et.al., 2014).

Lung Cancer

ALCAM was reported to be overexpressed in small cell lung cancers (Teicher A.B., 2014). Overexpression of ALCAM leads to malignancy in non-small cell lung carcinomas (NSCLC) (Ishiguro et.al., 2013). ALCAM expression levels were associated with smaller tumors with no lymph node metastasis in primary NSCLC lesions (Tachezy et.al., 2014).

Melanoma

High levels of ALCAM expression in primary tumors indicate poor prognosis and invasive phenotype (Donizy et.al., 2015).

Mesothelioma

ALCAM, was detected as one of the most highly upregulated genes in all 20 cell lines of Mesothelioma which may indicate that ALCAM overexpression is associated with tumor progression and ALCAM may be a target for treatment (Ishiguro et.al., 2012).

Myeloma

It was reported that ALCAM is associated with cell migration and invasion in myeloma cell lines (NCI-H929, JJN3, KMS-11, MM1S, OPM2, RPMI-8226, U266, and U266-LR7 (Paiva et.al., 2015).

Nasopharyngeal Carcinoma

ALCAM is a positive marker of Cancer Stem Cells and expressed in nasopharyngeal carcinoma cells (Chen et.al., 2015).

Ovarian Cancer

ALCAM levels are associated with progression-free survival in ovarian cancer cells as a MIR378A target (Chan et.al., 2014). EGFR was found to control the release of ALCAM from ovarian cancer cells via a metalloproteinase associated mechanism (Lozneanu et.al., 2015). Serum sALCAM levels were found to be significantly higher in epithelial ovarian cancer (EOC) compared to controls and elevated serum levels of sALCAM were associated with MUC16 (CA125) level and more aggressive (type II) tumors (Carbotti et.al., 2013). It was shown that ALCAM is expressed at the surface of epithelial ovarian cancer (EOC) cells and may be internalized after soluble ligand engagement (Nuti et.al., 2013).

Pancreatic Cancer

ALCAM is expressed in most of the pancreatic cancer lesions, but there is not any association between clinical and pathological data (Tachezy et.al., 2012). ALCAM expressing (CD166+) pancreatic cancer cells are reported to be strongly tumorigenic, but interestingly, CD166- pancreatic cancer cells show stronger invasive and migratory activities. (Fujiwara et. al., 2014). ALCAM levels are upregulated in Pancreatic Stellate Cells of pancreatic cancer tissues, which may indicate a role for ALCAM to regulate pancreatic cancer cell-pancreatic stellate cell interaction (Zhang et.al., 2016).

Rectal Cancer

It was found that ALCAM expression is upregulated after radiochemotherapy compared to pre-treatment stage in rectal cancer patients, indicating that radiochemotherapy upregulates stem cell markers in patients with rectal cancer (Deng et.al., 2014).

Salivary Gland Tumor

ALCAM is overexpressed in both benign and malignant salivary gland tumors. ALCAM expression is associated with more aggressive behavior in malignant tumors (Andisheh-Tadbir et.al., 2015).

Thyroid Carcinoma

Elevated levels of ALCAM are associated with aggressive tumors and lymph node metastasis of thyroid carcinoma Chaker et.al., 2013).

Other diseases

Higher ALCAM expression reported in other cases are listed below:

Dental follicles in children (Kang et al., 2016)

Lymphocytes infected with human T-lymphotropic virus type 1 (Curis et al., 2016)

Scrub typhus patients (Otterdal et al., 2014)

Salivary gland epithelial cells of Sjögren's syndrome patients (Le Dantec et al., 2013)

Subcutaneous abdominal adipose tissue of male obese patients (Gonzalez-Muniesa et al., 2013)

Human immunodeficiency virus (HIV)-infected patients with diapedesis compared to HIV seronegative (Williams et al., 2014)

The urine of Type I diabetes patients (Suh et al., 2015)

ALCAM has been implicated in various pathologies other than cancer. For example, multiple sclerosis is speculated to be associated with genetic variations of ALCAM. (Wagner et al., 2013, Wagner et al., 2014).

Elevated ALCAM expression could increase the recruitment of leukocytes into the microvasculature, leading to atherosclerosis (Zimmerman et al., 2006). Another study reported that ALCAM could promote heart injury (Lolyeva et. al., 2013).

Higher ALCAM concentrations in plasma of people with acute ischemic stroke have lower survival rate (Smedbakken et al., 2011).

Single nucleotide polymorphisms (SNPs) in ALCAM were associated with active adult-onset nonallergic asthma, Crohn's disease, rheumatoid arthritis and type I diabetes (Siroux et al., 2014, Eleftherohorinou et al.,, 2009).

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