ICAM1 (intercellular adhesion molecule 1 (CD54), human rhinovirus receptor)

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Published in Atlas Database: July 2008


DOI: 10.4267/2042/44489

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Identity

Other names: BB2; CD54; ICAM-1; P3.58
HGNC (Hugo): ICAM1
Location: 19p13.2

DNA/RNA

Description
ICAM1 gene maps on chromosome 19p13.3-p13.2 scanning 15512 bp.

Transcription
7 exon; mRNA 3249 bp.

Protein

Description
A transmembrane glycoprotein of 532 amino acids, which has a molecular mass ranging from 75 to 115 kDa. ICAM-1 is a member of the immunoglobulin supergene family, and contains five extracellular immunoglobulin-like domains.

Expression
ICAM-1 is expressed on various cells of both hematopoietic and non-hematopoietic cells, including endothelial cells, leukocytes other than basophilic granulocytes, T cells, B cells, fibroblasts, and cancer cells.

Function
ICAM-1, a member of adhesion molecules, binds to two integrins belonging to the beta2 subfamily CD11a/CD18 (LFA-1) and CD11b/CD18 (Mac-1) on the surface of leukocytes. ICAM1 is a key molecule in immune-mediated and inflammatory processes and function as a co-stimulatory signal which is important for the trans-endothelial migration of leukocytes and the activation of T cells. ICAM-1 can be induced under inflammatory condition by TNF-alpha in a process that involves IKK-beta. ICAM1 on target cells leads to recruitment of the MHC-I proteins to the contact area and enhances presentation of cognate peptide MHC-I complex to cytotoxic T cells. ICAM-1 interacts with important factors involved in many kinds of human cancers. ICAM-1 is involved in transmembrane signal transduction in the regulatory process of cell proliferation through the mitogen-activated protein kinase (MAPK) pathway and eventually the AP-1 pathway (Springer, 1990; Dustin et al., 1998; Simmons et al., 1988; Caras et al., 1987; Rothlein et al., 1986; Dustin et al., 1986; Tsujisaki et al., 1991; Gardiner and D'Souza, 1999).

Implicated in

Melanoma

Disease
ICAM-1 expresses with a dose- and time-dependent increase in human malignant melanoma cells on stimulation of TNF-alpha. Inhibition of ICAM-1 expression on melanoma cells reduces the metastatic ability of the melanoma cells, indicating an important role of ICAM-1 in metastasis (Miele et al., 1994).

Hepatocellular carcinoma

Disease
Serum concentration of ICAM-1 (sICAM-1) in patients with hepatocellular carcinoma is a marker for disease progression and prognosis. Higher sICAM-1 levels are more frequently observed in those patients with multiple lesions and intra-hepatic metastasis (Mei et al., 2000).

**Prostate cancer**

**Disease**

Two polymorphisms and a common haplotype within the ICAM-1 gene cluster were correlated with prostate cancer risk among men with family history of the disease (Chen et al., 2006).

**Colon cancer**

**Disease**

ICAM-1 expression is decreased in colorectal cancer (Berndt et al., 2008).

**Gastric cancer**

**Disease**

Decreased ICAM expression leads to lymph node metastasis, suggesting that ICAM-1 gene transfection can inhibit lymph node metastasis (Tanaka et al., 2002; Yashiro et al., 2005).

**Head and neck cancer**

**Disease**

sICAM-1 shows a significant elevation in the sera of patients with nasopharyngeal carcinoma compared to healthy controls (Liu et al., 1999).

**Leukemia**

**Disease**

A high expression of ICAM-1 may identify high-risk MDS (Graf et al., 2005).

**Breast cancer**

**Disease**

ICAM-1 down-regulation at the mRNA and protein levels led to a suppression of breast cells invasion (Rosette et al., 2005).

**References**


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Miele ME, Bennett CF, Miller BE, Welch DR. Enhanced metastatic ability of TNF-alpha-treated malignant melanoma cells is reduced by intercellular adhesion molecule-1 (ICAM-1, CD54) antisense oligonucleotides. Exp Cell Res. 1994 Sep;214(1):231-41


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