NT5E (5'-nucleotidase, ecto (CD73))

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Identity
Other names: CD73, E5NT, NT, NT5, NTE, eN, eNT
HGNC (Hugo): NT5E
Location: 6q14.3

DNA/RNA

Transcription
Two transcript variants encoding different isoforms have been found for this gene.
Variant 1 represents the longer transcript and encodes the longer isoform 1, 9 exons, transcript length 3548 bps, translation length 574 residues; variant 2 lacks an alternate in-frame exon compared to variant 1, 8 exons, transcript length 3384 bps, translation length 524 residues.

Protein

Description
There are two isoforms of NT5E. 5'-nucleotidase isoform 1 preproprotein, 574 amino acids; 5'-nucleotidase isoform 2 preproprotein, 524 amino acids. Isoform 2 has the same N- and C-termini but is shorter compared to isoform 1.
The NT5E preproprotein is further processed into a mature form, which consists of a dimer of 2 identical 70-kD subunits bound by a glycosyl phosphatidyl inositol linkage at its C-terminus to the external face of the plasma membrane.

Expression
CD73 is a cell surface enzyme found in most tissues and many cell types including subsets of lymphocytes, macrophages, dendritic cells, endothelial cells and epithelial cells.

Function
CD73 is an ectoenzyme (ecto-5'-nucleotidase, EC 3.1.3.5).
It catalyzes conversion of AMP to adenosine.
Adenosine exerts its effects via adenosine receptor A1, adenosine receptor A2A, adenosine receptor A2B and adenosine receptor A3.
CD73 has many physiological roles, such as regulation of barrier function, adaptation to hypoxia, ischemic preconditioning, anti-inflammation, leukocyte extravasation.
Expression and activity of CD73 on cancer cells is associated with poor prognosis and may promote metastasis.
CD73 facilitates the adhesion, migration, invasion of human breast cancer cells and proliferation of glioma cells and these process are dependent upon the enzyme's production of adenosine.
The NT5E gene is located on the long (q) arm of chromosome 6 between positions 14 and 21. More precisely, the NT5E gene is located from base pair 86159301 to base pair 86205508 on chromosome 6.

**Implicated in**

**Melanoma**

**Note**
Deregulation of NT5E expression in melanoma occurs via epigenetic changes in the NT5E CpG island. Confirmation of the results in larger clinical series would support the candidacy of NT5E as a clinical biomarker in melanoma, which could be applied in both primary and relapsed disease. Inhibition of NT5E may have therapeutic potential in melanoma, particularly in patients with more aggressive disease metastasis to viscera or the brain.

**Colorectal cancer**

**Note**
CD73 expression in colorectal cancer is significantly higher than in normal colorectal tissues.

**Prognosis**
Patients with high expression of CD73 had a poorer overall survival rate compared with patients with low expression of CD73 in both cohorts. High expression of CD73 can be an independent and useful biomarker for predicting the poor survival of patients with colorectal cancer.

**Chronic lymphocytic leukemia**

**Note**
CD73-generated extracellular adenosine in chronic lymphocytic leukemia increases cytoplasmic cAMP levels by activation of the ADO receptors, inhibiting chemotaxis and limiting spontaneous drug-induced apoptosis of chronic lymphocytic leukemia cells.

**Glioma**

**Note**
Adenosine induced an increase in glioma cell adhesion. Ecto-5'-NT/CD73, an important producer of extracellular adenosine, may modulate glioma cell adhesion and tumor cell-extracellular matrix interactions.

**Breast cancer**

**Note**
CD73 plays an important role in breast cancer growth by affecting cell cycle progression and apoptosis. CD73 overexpression increased cell viability and promoted cell cycle progression, depending on its enzyme activity.

CD73 may facilitate the adhesion, migration and invasion of human breast cancer cells through its enzyme activity of generating adenosine.

Tumor-derived CD73 is a mechanism of tumor immune escape and tumor metastasis, and targeted therapy against CD73 can trigger adaptive anti-tumor immunity and inhibit metastasis of breast cancer.

**References**


This article should be referenced as such: