SLC16A3 (solute carrier family 16, member 3 (monocarboxylic acid transporter 4))

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Identity

Other names: MCT3, MCT4, MGC138472, MGC138474
HGNC (Hugo): SLC16A3
Location: 17q25.3

DNA/RNA

Note
SLC16A3 was first cloned from human circulating blood by Price et al. (1998).

Description
11077 bp length, 5 exons.

Transcription
3 transcripts have been described for this gene (all with protein product): SLC16A3-201, (5 exons; 2033 bps transcript length; 465 residues translation length); SLC16A3-202 (4 exons; 4222 bps transcript length; 465 residues translation length); SLC16A3-203 (5 exons; 2054 bps transcript length; 465 residues translation length).

Protein

Description
465 residues; 49469 Da; 12 transmembrane domains; intracellular N- and C-terminals.

Expression
SLC16A3/MCT4 is expressed in tissues such as white skeletal muscle fibres, astrocytes, white blood cells, chondrocytes, testis, lung, placenta, heart and some mammalian cell lines (Halestrap and Meredith, 2004; Meredith and Christian, 2008).

Localisation
Plasma membrane.

Function
Proton-linked monocarboxylate transporter. Catalyzes plasma membrane transport of monocarboxylates such as lactate, pyruvate, branched-chain oxo acids derived from leucine, valine and isoleucine, and the ketone bodies acetoacetate, beta-hydroxybutyrate and acetate.

Homology
Belongs to the major facilitator superfamily (MFS). Monocarboxylate porter (TC 2.A.1.13) family. The SLC16A3 gene is conserved in chimpanzee, dog, cow, mouse, rat, chicken, zebrafish, and M. grisea.

Implicated in

Colorectal carcinoma

Note
SLC16A3/MCT4 protein is overexpressed in colorectal cancer (Pinheiro et al., 2008a).

Cervical cancer

Note
**Bladder cancer**

**Note**
SLC16A3 gene expression was upregulated in some bladder tumours and induced by hypoxia in bladder cancer cell lines, but not in cultures of normal urothelium (Ord et al., 2005).

**Breast cancer**

**Note**
Induction was also seen in two breast cancer cell lines. Expression of SLC16A3 gene is higher in breast cancer distant metastasis as compared to primary tumours or regional metastasis. SLC16A3 gene was then included in the 'VEGF profile' of breast cancer, associated with promotion of vessel formation, survival under anaerobic conditions and loss of dependence upon fibroblasts (Hu et al., 2009).

**Ovarian cancer**

**Note**
SLC16A3 gene expression was described to be downregulated in malignant ovarian tumours as compared to normal ovarian surface epithelial cells. Additionally, the non-tumorigenic cell line TOV-81D presented higher expression that tumorigenic cell lines (Wojnarowicz et al., 2008).

SLC16A3 gene, among other transporter genes, was differentially expressed in a chemotherapy resistant ovarian cancer cell line and tumour tissue as compared to a chemosensitive cell line and tumour tissue. It was suggested that these transporters might be involved in drug influx/efflux, modulating chemotherapy response (Cheng et al., 2010).

**Mitochondrial myopathy**

**Note**
SLC16A3/MCT4 overexpression was described in a patient with a mitochondrial myopathy (Baker et al., 2001).

**Chronic obstructive pulmonary disease**

**Note**
SLC16A3/MCT4 downregulation was described in the vastus lateralis muscle of patients with chronic obstructive pulmonary disease as compared with healthy controls (Green et al., 2008).

**References**


Halestrap AP, Meredith D. The SLC16 gene family—from monocarboxylate transporters (MCTs) to aromatic amino acid transporters and beyond. Pflugers Arch. 2004 Feb;447(5):619-28


This article should be referenced as such: