Best Tools for immunology Research

MCT8 Polyclonal Antibody

Catalog No :	YT2686
Reactivity :	Human;Mouse;Rat
Applications :	WB;ELISA
Target :	MCT8
Fields :	>>Thyroid hormone signaling pathway
Gene Name :	SLC16A2
Protein Name :	Monocarboxylate transporter 8
Human Gene Id :	6567
Human Swiss Prot No :	P36021
Mouse Gene Id :	20502
Mouse Swiss Prot No :	O70324
Rat Gene Id :	259248
Rat Swiss Prot No :	Q8K1P8
Immunogen :	The antiserum was produced against synthesized peptide derived from human SLC16A2. AA range:112-161
Specificity :	MCT8 Polyclonal Antibody detects endogenous levels of MCT8 protein.
Formulation :	Liquid in PBS containing 50% glycerol, 0.5% BSA and 0.02% sodium azide.
Source :	Polyclonal, Rabbit,IgG
Dilution :	WB 1:500 - 1:2000. ELISA: 1:40000. Not yet tested in other applications.

Purification : The antibody was affinity-purified from rabbit antiserum by affinity-chromatography using epitope-specific immunogen.

Concentration : 1 mg/ml

Storage Stability : -15°C to -25°C/1 year(Do not lower than -25°C)

Observed Band : 60kD

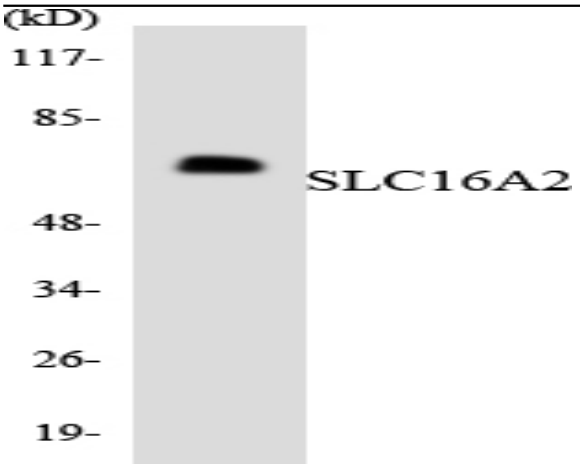
Background : This gene encodes an integral membrane protein that functions as a transporter of thyroid hormone. The encoded protein facilitates the cellular importation of thyroxine (T4), triiodothyronine (T3), reverse triiodothyronine (rT3) and diidothyronine (T2). This gene is expressed in many tissues and likely plays an important role in the development of the central nervous system. Loss of function mutations in this gene are associated with psychomotor retardation in males while females exhibit no neurological defects and more moderate thyroid-deficient phenotypes. This gene is subject to X-chromosome inactivation. Mutations in this gene are the cause of Allan-Herndon-Dudley syndrome. [provided by RefSeq, Mar 2012],

Function : disease:Defects in SLC16A2 are the cause of monocarboxylate transporter 8 deficiency (MCT8 deficiency) [MIM:300523]. MCT8 deficiency consists of a severe form of X-linked psychomotor retardation combined with abnormal thyroid hormone (TH) levels. Thyroid hormone deficiency can be caused by defects of hormone synthesis and action, but it has also been linked to a defect in cellular hormone transport. Affected patients are males with abnormal relative concentrations of three circulating iodothyronines, as well as severe neurological abnormalities, including global developmental delay, central hypotonia, spastic quadriplegia, dystonic movements, rotary nystagmus, and impaired gaze and hearing. Heterozygous females had a milder thyroid phenotype and no neurological defects.,function:Very active and specific thyroid hormone transporter. Stimulates cellular uptake of thyroxine (T4), triiodothy

Subcellular Location : Cell membrane ; Multi-pass membrane protein .

Expression : Highly expressed in liver and heart.

Products Images



Western blot analysis of the lysates from HT-29 cells using SLC16A2 antibody.