
Product Name: MCT8 Rabbit Polyclonal Antibody**Catalog #: APRab13743**

For research use only.

Summary

Description	Rabbit polyclonal Antibody
Host	Rabbit
Application	WB,ELISA
Reactivity	Human,Mouse,Rat
Conjugation	Unconjugated
Modification	Unmodified
Isotype	IgG
Clonality	Polyclonal
Form	Liquid
Concentration	1mg/ml
Storage	Aliquot and store at -20°C (valid for 12 months). Avoid freeze/thaw cycles.
Shipping	Ice bags
Buffer	Liquid in PBS containing 50% glycerol, 0.5% protective protein and 0.02% New type preservative N.
Purification	Affinity purification

Application

Dilution Ratio	WB 1:500-1:2000,ELISA 1:20000-1:40000
Molecular Weight	60kDa

Antigen Information

Gene Name	SLC16A2
Alternative Names	SLC16A2; MCT8; XPCT; Monocarboxylate transporter 8; MCT 8; Monocarboxylate transporter 7; MCT 7; Solute carrier family 16 member 2; X-linked PEST-containing transporter
Gene ID	6567.0
SwissProt ID	P36021
Immunogen	The antiserum was produced against synthesized peptide derived from human SLC16A2. AA range:112-161

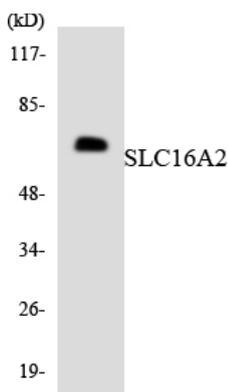
Background

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 diiodothyronine (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],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), triiodothyronine (T3), reverse triiodothyronine (rT3) and diiodothyronine. Does not transport Leu, Phe, Trp or Tyr.,similarity:Belongs to the major facilitator superfamily. Monocarboxylate porter (TC 2.A.1.13) family.,tissue specificity:Highly expressed in liver and heart.,

Research Area

Signal Transduction; Growth Factors/Hormones; Hormones; Neuroscience; Endocrine system; Thyroid axis

Image Data



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