ACSL4 (FACL4) Antibody (Center)

Catalog_no： AB2083

Reactivity： H

Category： 抗原抗体

Size： 100μL/50μL

Immunogen： HUMAN:236-267

Specificity： This ACSL4 (FACL4) antibody is generated from rabbits immunized with a KLH conjugated synthetic peptide between 236-267 amino acids from the Central region of human ACSL4 (FACL4).

Dilution： IHC-P,1:25;WB,1:1000;IF,1:10~50;

Purification： Purified polyclonal antibody supplied in PBS with 0.09% (W/V) sodium azide. This antibody is purified through a protein G column, eluted with high and low pH buffers and neutralized immediately, followed by dialysis against PBS.

Other_name： Long-chain-fatty-acid--CoA ligase 4, Long-chain acyl-CoA synthetase 4, LACS 4, ACSL4, ACS4, FACL4, LACS4

Isotype： Rabbit Ig

Background： Long chain acyl-CoA synthetase (LACS), or long chain fatty acid-CoA ligase (FACL), converts free long chain fatty acids into fatty acyl-CoA esters, key intermediates in the synthesis of complex lipids. The FACL4 gene encodes a form of LACS and is expressed in several tissues, including brain. FACL4 cDNA from brain encodes a gene product that shows preference for arachidonic acid as a substrate when expressed in mammalian cells.1 The sequence of the predicted 670-amino acid human protein is 97% identical to that of rat ACS4. FACL4 is highly expressed in adult human brain, especially in the cerebellum and hippocampus, similar to the mouse.2 A strong cytoplasmic staining was found in the Purkinje and granular cells of the cerebellum and the pyramidal layer of hippocampus, indicating that FACL4 is specifically expressed in neurons and not in glial cells. Two patients with Alport syndrome, elliptocytosis, and mental retardation carried a large deletion of the COL4A5 region that included FACL4.3 The absence of FACL4 might play a role in the development of mental retardation or other signs associated with Alport syndrome. Two point mutations, 1 missense and 1 splice site change, were reported in the FACL4 gene in 2 families with nonspecific mental retardation.2 Analysis of enzymatic activity in lymphoblastoid cell lines of affected individuals revealed low levels compared with normal cells, indicating that both mutations are null mutations.