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Rabbit Anti-15 Lipoxygenase 1 antibody

SL6505R

Product Name:	15 Lipoxygenase 1
Chinese Name:	花生四烯酸15脂氧合酶1抗体
Alias:	15 lipoxygenase 1; 15 LIPOXYGENASE RETICULOCYTE ARACHIDONATE; 15 LOX; 15 LOX 1; 15-LOX; 15LOX 1; ALOX15; Arachidonate 15 lipoxygenase; Arachidonate 15-lipoxygenase; Arachidonate omega 6 lipoxygenase; Arachidonate omega-6 lipoxygenase; LOG15; LOX15_HUMAN.
Organism Species:	Rabbit
Clonality:	Polyclonal
React Species:	Human,Mouse,Rat,Pig,Cow,
Applications:	ELISA=1:500-1000IHC-P=1:400-800IHC-F=1:400-800IF=1:100-500（Paraffin sections need antigen repair） not yet tested in other applications. optimal dilutions/concentrations should be determined by the end user.
Molecular weight:	73kDa
Cellular localization:	cytoplasmic
Form:	Lyophilized or Liquid
Concentration:	1mg/ml
immunogen:	KLH conjugated synthetic peptide derived from human ALOX15/15 Lipoxygenase 1:581-662/662
Lsotype:	IgG
Purification:	affinity purified by Protein A
Storage Buffer:	0.01M TBS(pH7.4) with 1% BSA, 0.03% Proclin300 and 50% Glycerol.
Storage:	Store at -20 °C for one year. Avoid repeated freeze/thaw cycles. The lyophilized antibody is stable at room temperature for at least one month and for greater than a year when kept at -20°C. When reconstituted in sterile pH 7.4 0.01M PBS or diluent of antibody the antibody is stable for at least two weeks at 2-4 °C.
PubMed:	PubMed
Product Detail:	Lipoxygenases are a family of enzymes which dioxygenate unsaturated fatty acids, thus initiating lipoperoxidation of membranes and synthesis of signaling molecules, as well as inducing structural and metabolic changes in the cell. The Lox enzymes in mammals

include 12-LO and 15-LO, which are classified with respect to their positional specificity of the deoxygenation of their most common substrate, arachidonic acid. The metabolism of arachidonic acid leads to the generation of biologically active metabolites that have been implicated in cell growth and proliferation, as well as survival and apoptosis. 15-Lipoxygenase (15-LO) acts in physiological membrane remodeling and the pathogenesis of atherosclerosis, inflammation, and carcinogenesis. It is highly regulated and expressed in a tissue- and cell-type-specific fashion. IL-4 and IL-13 play important roles in transactivating the 15-LO gene. Overexpression of 15-LO type 1 in prostate cancer contributes to the cancer progression by regulating IGF-1R expression and activation.

Function:
Oxygenase and 14,15-leukotriene A4 synthase activity. Converts arachidonic acid to 15S-hydroperoxyeicosatetraenoic acid. Also acts on C-12 of arachidonate as well as on linoleic acid.

Subunit:
Homotetramer. Can also form heterotetramers with RYR2. Interacts with CALM; CALM with bound calcium inhibits the RYR1 channel activity. Interacts with S100A1. Interacts with FKBP1A; this stabilizes the closed conformation of the channel. Interacts with CACNA1S; interaction with CACNA1S is important for activation of the RYR1 channel. Interacts with CACNB1. Interacts with TRDN and ASPH; these interactions stimulate RYR1 channel activity (By similarity). Identified in a complex composed of RYR1, PDE4D, PKA, FKBP1A and protein phosphatase 1 (PP1). Repeated very high-level exercise decreases interaction with PDE4D and protein phosphatase 1 (PP1).

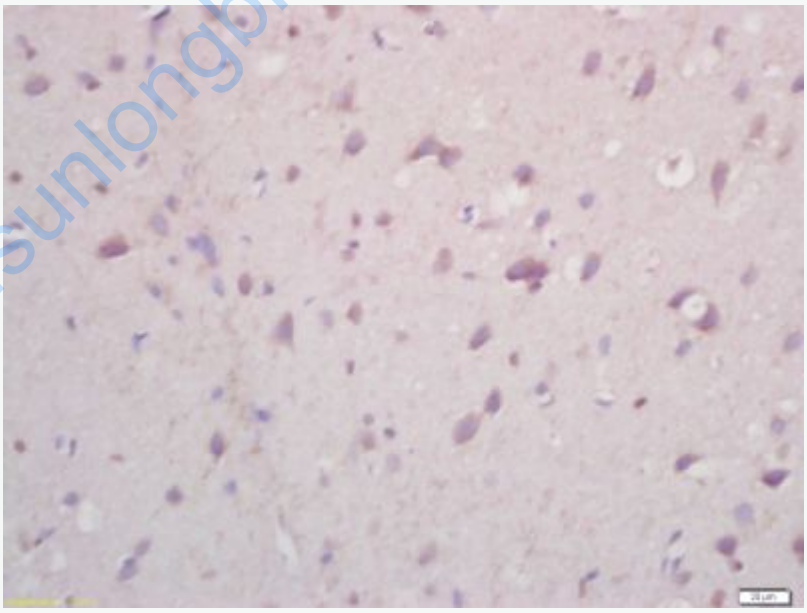
Subcellular Location:
Cytoplasm.

Tissue Specificity:
Skeletal muscle and brain (cerebellum and hippocampus).

Post-translational modifications:
Channel activity is modulated by phosphorylation. Phosphorylation at Ser-2843 may increase channel activity. Repeated very high-level exercise increases phosphorylation at Ser-2843.
Activated by reversible S-nitrosylation. Repeated very high-level exercise increases S-nitrosylation.

DISEASE:
Malignant hyperthermia 1 (MHS1) [MIM:145600]: Autosomal dominant pharmacogenetic disorder of skeletal muscle and is one of the main causes of death due to anesthesia. In susceptible people, an MH episode can be triggered by all commonly used inhalational anesthetics such as halothane and by depolarizing muscle relaxants such as succinylcholine. The clinical features of the myopathy are hyperthermia, accelerated muscle metabolism, contractures, metabolic acidosis, tachycardia and death,

	if not treated with the postsynaptic muscle relaxant, dantrolene. Susceptibility to MH can be determined with the 'in vitro' contracture test (IVCT): observing the magnitude of contractures induced in strips of muscle tissue by caffeine alone and halothane alone. Patients with normal response are MH normal (MHN), those with abnormal response to caffeine alone or halothane alone are MH equivocal (MHE(C) and MHE(H) respectively). Note=The disease is caused by mutations affecting the gene represented in this entry. Central core disease of muscle (CCD) [MIM:117000]: Autosomal dominant congenital myopathy, but a severe autosomal recessive form also exists. Both clinical and histological variability is observed. Affected individuals typically display hypotonia and proximal muscle weakness in infancy, leading to the delay of motor milestones. The clinical course of the disorder is usually slow or nonprogressive in adulthood, and the severity of the symptoms may vary from normal to significant muscle weakness. Microscopic examination of CCD-affected skeletal muscle reveals a predominance of type I fibers containing amorphous-looking areas (cores) that do not stain with oxidative and phosphorylase histochemical techniques. Note=The disease is caused by mutations affecting the gene represented in this entry. Multiminicore disease with external ophthalmoplegia (MMDO) [MIM:255320]: Clinically heterogeneous neuromuscular disorder. General features include neonatal hypotonia, delayed motor development, and generalized muscle weakness and amyotrophy, which may progress slowly or remain stable. Muscle biopsy shows multiple, poorly circumscribed, short areas of sarcomere disorganization and mitochondria depletion (areas termed minicores) in most muscle fibers. Typically, no dystrophic signs, such as muscle fiber necrosis or regeneration or significant endomysial fibrosis, are present in multiminicore disease. Note=The disease is caused by mutations affecting the gene represented in this entry. Congenital myopathy with fiber-type disproportion (CFTD) [MIM:255310]: Genetically heterogeneous disorder in which there is relative hypotrophy of type 1 muscle fibers compared to type 2 fibers on skeletal muscle biopsy. However, these findings are not specific and can be found in many different myopathic and neuropathic conditions. Note=The disease is caused by mutations affecting the gene represented in this entry. Note=Defects in RYR1 may be a cause of Samaritan myopathy, a congenital myopathy with benign course. Patients display severe hypotonia and respiratory distress at birth. Unlike other congenital myopathies, the health status constantly improves and patients are minimally affected at adulthood. **Similarity:** Belongs to the lipoxygenase family. Contains 1 lipoxygenase domain. Contains 1 PLAT domain. **SWISS:** P16050 **Gene ID:** 246

	Database links: Entrez Gene: 246 Human Entrez Gene: 81639 Rat Omim: 152392 Human SwissProt: P16050 Human SwissProt: Q02759 Rat Unigene: 73809 Human **Important Note:** This product as supplied is intended for research use only, not for use in human, therapeutic or diagnostic applications.
Picture:	 Tissue/cell: rat brain tissue; 4% Paraformaldehyde-fixed and paraffin-embedded; Antigen retrieval: citrate buffer (0.01M, pH 6.0), Boiling bathing for 15min; Block endogenous peroxidase by 3% Hydrogen peroxide for 30min; Blocking buffer

	(normal goat serum,C-0005) at 37℃ for 20 min; Incubation: Anti-15-LOX Polyclonal Antibody, Unconjugated(SL6505R) 1:200, overnight at 4°C, followed by conjugation to the secondary antibody(SP-0023) and DAB(C-0010) staining