9051 Mira Mesa Blvd, #910094
San Diego, CA 92191
Tel: 760-390-3989

ACVRL1 Human

Activin A Receptor Type II-Like 1 Human Recombinant
GRF0011

Product Overview

Name	ACVRL1 Human
Description	
Activin A Receptor Type II-Like 1 Human Recombinant	
Accession (Primary)	P37023
Immunogen	
Anti-human ACY1 mAb, is derived from hybridization of mouse F0 myeloma cells with spleen cells from BALB/c mice immunized with recombinant human ACY1 amino acids 1-408 purified from E. coli.	
Synonyms	
ACY1D, ACYLASE, ACY-1, EC 3.5.1.143.	
Introduction	
ACY1 is a cytosolic, homodimeric, zinc-binding enzyme that catalyzes the hydrolysis of acylated L-amino acids to L-amino acids and acyl group, and has been postulated to take part in the catabolism and salvage of acylated amino acids. Defects in ACY1 are the source of aminoacylase-1 deficiency (ACY1D). ACY1D results in a metabolic disorder manifesting with encephalopathy, unspecific psychomotor delay, psychomotor delay with atrophy of the vermis and syringomyelia, marked muscular hypotonia or normal clinical features. Epileptic seizures are a frequent feature.	
Physical Appearance	
Sterile filtered colorless solution.	
Formulation	
1mg/ml containing PBS, pH-7.4, 10% Glycerol and 0.02% Sodium Azide.	
Applications	
ACY1 antibody has been tested by ELISA, Western blot analysis to assure specificity and reactivity. Since application varies, however, each investigation should be titrated by the reagent to obtain optimal results. Recommended starting dilution is 1:1000.	
Type	
Mouse Anti Human Monoclonal.	
Clone	
PAT1E2AT.	
Ig Subclass	

Mouse IgG 2b heavy chain and ? light chain.

Purification Method

ACY1 antibody was purified from mouse ascitic fluids by protein-A affinity chromatography.

Storage Procedures

For periods up to 1 month store at 4°C, for longer periods of time, store at -20°C. Prevent freeze thaw cycles.

Stability / Shelf Life

12 months at -20°C. 1 month at 4°C.

Precautions

ACVRL1 Human is for research use only and not for use in diagnostic or therapeutic procedures.

Target Information: (P37023)

Background

The Physiological Implications and Therapeutic Potential of Activin A Receptor Type II-Like 1 Human Recombinant 1. Abstract This research paper investigates the Activin A Receptor Type II-Like 1 Human Recombinant (ACVRL1), a significant protein involved in the TGF-beta superfamily signaling pathway. We provide an extensive understanding of ACVRL1's structure, signaling mechanism, biological functions, and implications in disease pathology. Additionally, we explore the therapeutic potential of ACVRL1 in various pathological conditions. 2. Introduction ACVRL1, also known as ALK1, plays an essential role in the TGF-beta signaling pathway, which has implications in cellular proliferation, differentiation, and apoptosis. Understanding ACVRL1 and its signaling mechanisms could provide insights into its potential therapeutic applications in various diseases. 3. Structure and Signaling of ACVRL1 ACVRL1 is a type I receptor protein involved in the TGF-beta signaling pathway. It is a transmembrane protein that consists of a ligand-binding extracellular domain and an intracellular domain responsible for signal transduction. Binding of ligands to ACVRL1 triggers phosphorylation events that activate downstream signaling pathways. 4. Biological Functions of ACVRL1 ACVRL1 plays pivotal roles in multiple biological processes, including vascular development, angiogenesis, and maintenance of vascular integrity. It is known to influence cellular processes such as proliferation, differentiation, and apoptosis, thereby implicating it in organogenesis and homeostasis. 5. ACVRL1 in Disease Pathology Mutations in the ACVRL1 gene have been associated with hereditary hemorrhagic telangiectasia (HHT), a genetic disorder characterized by abnormal blood vessel formation. This link underscores the critical role of ACVRL1 in vascular biology and disease. 6. Therapeutic Potential of ACVRL1 Given its crucial role in vascular biology and its link to HHT, ACVRL1 presents a promising target for therapeutic interventions. Modulation of ACVRL1 signaling could potentially

provide treatment options for pathological conditions related to abnormal blood vessel formation and function. 7. Conclusion and Future Perspectives Our understanding of ACVRL1 and its functions has grown significantly in recent years, but there is much yet to be discovered. Continued research into ACVRL1's precise molecular mechanisms and its roles in disease will undoubtedly open new doors for therapeutic development.