

Anti-Alpha Actinin 4 Antibody

Our Anti-Alpha Actinin 4 primary antibody from PhosphoSolutions is mouse monoclonal. It detects huma

Catalog # AN1302

Specification

Anti-Alpha Actinin 4 Antibody - Product Information

Application WB
Primary Accession O43707
Host Mouse
Clonality Monoclonal
Isotype IgG

Isotype IgG
Calculated MW 104854

Anti-Alpha Actinin 4 Antibody - Additional Information

Gene ID 81

Other Names

actinin 4 antibody, Actinin alpha 4 antibody, actinin4 antibody, ACTN 4 antibody, ACTN4 antibody, ACTN4_HUMAN antibody, alpha Actinin 4 antibody, Alpha-actinin-4 antibody, DKFZp686K23158 antibody, F actin cross linking protein antibody, F-actin cross-linking protein antibody, Focal segmental glomerulosclerosis 1 antibody, FSGS 1 antibody, FSGS antibody, FSGS1 antibody, Nonmuscle alpha actinin 4 antibody, Non-muscle alpha-actinin 4 antibody

Target/Specificity

 α -actinin-4 is a member of the actinin protein family comprised of an actin-binding domain in the N-terminus, 4 spectrin-like repeats in the central region, and 2 EF-hand motifs in the C-terminus (Honda et al, 1998). α -actinin-4 and CLP36 form a complex in normal kidney podocytes. CLP36 is dependent on α -actinin-4 for maintenance of its level in podocytes, whereas α -actinin-4 is independent of CLP36. α -actinin-4 is widely expressed in mammalian tissues and organs, while having a high occurrence of genetic mutations in kidney podocytes (Kos et al, 2003). FSGS, focal segmental glomerulosclerosis, is a rare genetic disease that attacks the kidney's filtering units (glomeruli) causing serious scarring which leads to permanent kidney damage and even failure. Three key mutations have been found in α -actinin-4 in people diagnosed with FSGS. R310Q and Q348R, located in the spectrin-like repeats region, and K255E located in the actin-binding region. The R310Q and Q348R mutation significantly inhibits the ability of α -actinin-4 to form the complex with CLP36. The K255E mutation was reversed where it increased the ability to bind CLP36 in the actin-binding region (Liu et al, 2011).

Dilution

WB~~1:1000

Format

Protein G purified

Storage

Maintain refrigerated at 2-8°C for up to 6 months. For long term storage store at -20°C in small aliquots to prevent freeze-thaw cycles.

Precautions



Anti-Alpha Actinin 4 Antibody is for research use only and not for use in diagnostic or therapeutic procedures.

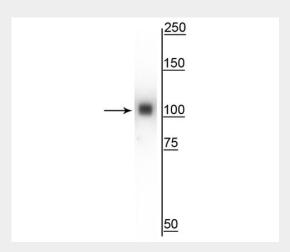
Shipping Blue Ice

Anti-Alpha Actinin 4 Antibody - Protocols

Provided below are standard protocols that you may find useful for product applications.

- Western Blot
- Blocking Peptides
- Dot Blot
- Immunohistochemistry
- Immunofluorescence
- Immunoprecipitation
- Flow Cytomety
- Cell Culture

Anti-Alpha Actinin 4 Antibody - Images



Western blot of mouse whole brain lysate showing specific immunolabeling of the $\sim \! 105$ kDa $\alpha \!$ -actinin 4 protein.

Anti-Alpha Actinin 4 Antibody - Background

 α -actinin-4 is a member of the actinin protein family comprised of an actin-binding domain in the N-terminus, 4 spectrin-like repeats in the central region, and 2 EF-hand motifs in the C-terminus (Honda et al, 1998). α -actinin-4 and CLP36 form a complex in normal kidney podocytes.CLP36 is dependent on α -actinin-4 for maintenance of its level in podocytes, whereas α -actinin-4 is independent of CLP36. α -actinin-4 is widely expressed in mammalian tissues and organs, while having a high occurrence of genetic mutations in kidney podocytes (Kos et al, 2003). FSGS, focal segmental glomerulosclerosis, is a rare genetic disease that attacks the kidney's filtering units (glomeruli) causing serious scarring which leads to permanent kidney damage and even failure. Three key mutations have been found in α -actinin-4 in people diagnosed with FSGS. R310Q and Q348R, located in the spectrin-like repeats region, and K255E located in the actin-binding region. The R310Q and Q348R mutation significantly inhibits the ability of α -actinin-4 to form the complex with CLP36. The K255E mutation was reversed where it increased the ability to bind CLP36 in the actin-binding region (Liu et al, 2011).