

## Product datasheet for **TA388989**

### ACTN4 Mouse Monoclonal Antibody [Clone ID: 93]

#### Product data:

Product Type:	Primary Antibodies
Clone Name:	93
Applications:	IHC, WB
Recommended Dilution:	<b>WB:</b> 1:1000 <b>WB Brain:</b> 1:1000
Reactivity:	Human, Mouse
Host:	Mouse
Isotype:	IgG
Clonality:	Monoclonal
Immunogen:	Fusion protein from the central rod domain of human $\alpha$ -actinin 4.
Specificity:	Specific for endogenous levels of the ~105 kDa $\alpha$ -actinin 4 protein.
Formulation:	10 mM HEPES (pH 7.5), 150 mM NaCl, 100 $\mu$ g per ml BSA and 50% glycerol.
Concentration:	lot specific
Purification:	Protein G purified
Conjugation:	Unconjugated
Storage:	Storage at -20°C is recommended, as aliquots may be taken without freeze/thawing due to presence of 50% glycerol. Stable for at least 1 year at -20°C.
Stability:	After date of receipt, stable for at least 1 year at -20°C.
Predicted Protein Size:	105
Database Link:	<a href="#">O43707</a>



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**Background:**

$\alpha$ -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).  $\alpha$ -actinin-4 and CLP36 form a complex in normal kidney podocytes. CLP36 is dependent on  $\alpha$ -actinin-4 for maintenance of its level in podocytes, whereas  $\alpha$ -actinin-4 is independent of CLP36.  $\alpha$ -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  $\alpha$ -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  $\alpha$ -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).

**Note:**

Protein G purified cultured supernatant.