SANTA CRUZ BIOTECHNOLOGY, INC.

Podocin (H-130): sc-21009



BACKGROUND

The onset of autosomal recessive steroid-resistant nephrotic syndrome (SRN1) in humans occurs by early childhood. Characteristics of SRN1 include proteinuria, rapid progression to end-stage renal disease, and focal segmental glomerulosclerosis. The pathological conditions of SRN1 correlate well with mutations at the NPHS2 gene, where expression of a protein known as Podocin occurs. Abnormal or inefficient signaling through Podocin proteindependent networks contributes to the development of podocyte dysfunction and proteinuria. The human NPHS2 gene maps to chromosome 1q25.2 and encodes a 383 amino acid protein. Podocin is an integral membrane protein that appears to fold into a hairpin-like structure with intracellular amino- and carboxy-termini. Transmembrane and cytoplasmic portions of Podocin share homology to the corresponding regions of the stomatin family proteins. Expression of high-order oligomers of Podocin in glomerular podocytes may reflect a scaffolding function that influences proper function of the glomerular filtration barrier, which is necessary for renal stability.

CHROMOSOMAL LOCATION

Genetic locus: NPHS2 (human) mapping to 1q25.2; Nphs2 (mouse) mapping to 1 G3.

SOURCE

Podocin (H-130) is a rabbit polyclonal antibody raised against amino acids 1-130 (deletion 30-61) mapping at the N-terminus of Podocin of human origin.

PRODUCT

Each vial contains 200 μg lgG in 1.0 ml of PBS with < 0.1% sodium azide and 0.1% gelatin.

Available as agarose conjugate for immunoprecipitation, sc-21009 AC, 500 μ g/0.25 ml agarose in 1 ml.

APPLICATIONS

Podocin (H-130) is recommended for detection of Podocin of mouse, rat and human origin by Western Blotting (starting dilution 1:200, dilution range 1:100-1:1000), immunoprecipitation [1-2 μ g per 100-500 μ g of total protein (1 ml of cell lysate)], immunofluorescence (starting dilution 1:50, dilution range 1:50-1:500) and solid phase ELISA (starting dilution 1:30, dilution range 1:30-1:3000).

Suitable for use as control antibody for Podocin siRNA (h): sc-40859, Podocin siRNA (m): sc-40860, Podocin shRNA Plasmid (h): sc-40859-SH, Podocin shRNA Plasmid (m): sc-40860-SH, Podocin shRNA (h) Lentiviral Particles: sc-40859-V and Podocin shRNA (m) Lentiviral Particles: sc-40860-V.

Molecular Weight of Podocin: 42 kDa.

Positive Controls: TE671 cell lysate: sc-2416, Caki-1 cell lysate: sc-2224 or rat cerebellum extract: sc-2398.

STORAGE

Store at 4° C, **DO NOT FREEZE**. Stable for one year from the date of shipment. Non-hazardous. No MSDS required.

RESEARCH USE

For research use only, not for use in diagnostic procedures.

DATA





Podocin (H-130): sc-21009. Western blot analysis of

Podocin expression in TE671 whole cell lysate

Podocin (H-130): sc-21009. Western blot analysis of Podocin expression in TE671 (A) and Caki-1 (B) whole cell lysates and rat cerebellum (C), mouse cerebellum (D) and mouse kidney (E) tissue extracts.

SELECT PRODUCT CITATIONS

- Mao, J., et al. 2006. Expression profile of nephrin, podocin, and CD2AP in Chinese children with MCNS and IgA nephropathy. Pediatr. Nephrol. 21: 1666-1675.
- Toblli, J.E., et al. 2011. Long-term treatment with nebivolol attenuates renal damage in Zucker diabetic fatty rats. J. Hypertens. 29: 1613-1623.
- Boulberdaa, M., et al. 2011. Genetic inactivation of prokineticin receptor-1 leads to heart and kidney disorders. Arterioscler. Thromb. Vasc. Biol. 31: 842-850.
- Chittiprol, S., et al. 2011. Marker expression, behaviors, and responses vary in different lines of conditionally immortalized cultured podocytes. Am. J. Physiol. Renal Physiol. 301: F660-F671.
- Guimaraes-Souza, N.K., et al. 2012. *In vitro* reconstitution of human kidney structures for renal cell therapy. Nephrol. Dial. Transplant. 27: 3082-3090.
- Ito, M., et al. 2012. Glycoprotein hyposialylation gives rise to a nephroticlike syndrome that is prevented by sialic acid administration in GNE V572L point-mutant mice. PLoS ONE 7: e29873.
- Nascimento, F.A., et al. 2012. Maternal vitamin D deficiency delays glomerular maturity in F1 and F2 offspring. PLoS ONE 7: e41740.
- Ndisang, J.F. and Tiwari, S. 2014. Mechanisms by which heme oxygenase rescue renal dysfunction in obesity. Redox Biol. 2C: 1029-1037.
- Tsai, I.J., et al. 2015. Inhibition of Rho-associated kinase relieves C5ainduced proteinuria in murine nephrotic syndrome. Cell. Mol. Life Sci. 72: 3157-3171.

PROTOCOLS

See our web site at www.scbt.com or our catalog for detailed protocols and support products.