

# LKB1 siRNA (h): sc-35816

## BACKGROUND

Peutz-Jeghers syndrome (PJS) is a rare hereditary disease characterized by melanocytic macules lips, gastrointestinal hamartomatous polyps and an increased risk for many classes of cancer. LKB1 (also designated STK11 and PJS) has been identified as the gene mutated in PJS. LKB1 is a 433 amino acid serine/threonine kinase with strong homology to the *Xenopus* cytoplasmic protein kinase XEEK1 and weaker similarity to many other protein kinases. LKB1 is ubiquitously expressed and many frameshift, deletion and splicing mutations have been identified in PJS patients. Despite the increased risk of cancer for PJS patients, LKB1 does not appear to play a major role in colorectal, testicular or breast cancers.

## REFERENCES

1. Jenne, D.E., et al. 1998. Peutz-Jeghers syndrome is caused by mutations in a novel serine threonine kinase. *Nat. Genet.* 18: 38-43.
2. Mehenni, H., et al. 1998. Loss of LKB1 kinase activity in Peutz-Jeghers syndrome and evidence for allelic and locus heterogeneity. *Am. J. Hum. Genet.* 63: 1641-1650.

## CHROMOSOMAL LOCATION

Genetic locus: STK11 (human) mapping to 19p13.3.

## PRODUCT

LKB1 siRNA (h) is a pool of 3 target-specific 19-25 nt siRNAs designed to knock down gene expression. Each vial contains 3.3 nmol of lyophilized siRNA, sufficient for a 10  $\mu$ M solution once resuspended using protocol below. Suitable for 50-100 transfections. Also see LKB1 shRNA Plasmid (h): sc-35816-SH and LKB1 shRNA (h) Lentiviral Particles: sc-35816-V as alternate gene silencing products.

For independent verification of LKB1 (h) gene silencing results, we also provide the individual siRNA duplex components. Each is available as 3.3 nmol of lyophilized siRNA. These include: sc-35816A, sc-35816B and sc-35816C.

## STORAGE AND RESUSPENSION

Store lyophilized siRNA duplex at -20° C with desiccant. Stable for at least one year from the date of shipment. Once resuspended, store at -20° C, avoid contact with RNAses and repeated freeze thaw cycles.

Resuspend lyophilized siRNA duplex in 330  $\mu$ l of the RNase-free water provided. Resuspension of the siRNA duplex in 330  $\mu$ l of RNase-free water makes a 10  $\mu$ M solution in a 10  $\mu$ M Tris-HCl, pH 8.0, 20 mM NaCl, 1 mM EDTA buffered solution.

## APPLICATIONS

LKB1 siRNA (h) is recommended for the inhibition of LKB1 expression in human cells.

## RESEARCH USE

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

## SUPPORT REAGENTS

For optimal siRNA transfection efficiency, Santa Cruz Biotechnology's siRNA Transfection Reagent: sc-29528 (0.3 ml), siRNA Transfection Medium: sc-36868 (20 ml) and siRNA Dilution Buffer: sc-29527 (1.5 ml) are recommended. Control siRNAs or Fluorescein Conjugated Control siRNAs are available as 10  $\mu$ M in 60  $\mu$ l. Each contain a scrambled sequence that will not lead to the specific degradation of any known cellular mRNA. Fluorescein Conjugated Control siRNAs include: sc-36869, sc-44239, sc-44240 and sc-44241. Control siRNAs include: sc-37007, sc-44230, sc-44231, sc-44232, sc-44233, sc-44234, sc-44235, sc-44236, sc-44237 and sc-44238.

## GENE EXPRESSION MONITORING

LKB1 (N-19): sc-8185 is recommended as a control antibody for monitoring of LKB1 gene expression knockdown by Western Blotting (starting dilution 1:200, dilution range 1:100-1:1000) or immunofluorescence (starting dilution 1:50, dilution range 1:50-1:500).

## RT-PCR REAGENTS

Semi-quantitative RT-PCR may be performed to monitor LKB1 gene expression knockdown using RT-PCR Primer: LKB1 (h)-PR: sc-35816-PR (20  $\mu$ l, 521 bp). Annealing temperature for the primers should be 55-60° C and the extension temperature should be 68-72° C.

## SELECT PRODUCT CITATIONS

1. Song, P., et al. 2007. Reactive nitrogen species induced by hyperglycemia suppresses Akt signaling and triggers apoptosis by upregulating phosphatase PTEN (phosphatase and tensin homologue deleted on chromosome 10) in an LKB1-dependent manner. *Circulation* 116: 1585-1595.
2. Choi, H.C., et al. 2008. Reactive nitrogen species is required for the activation of the AMP-activated protein kinase by statin *in vivo*. *J. Biol. Chem.* 283: 20186-20197.
3. Zhang, J., et al. 2008. Identification of nitric oxide as an endogenous activator of the AMP-activated protein kinase in vascular endothelial cells. *J. Biol. Chem.* 283: 27452-27461.
4. Kou, R., et al. 2009. Regulation of Rac1 by simvastatin in endothelial cells: differential roles of AMP-activated protein kinase and calmodulin-dependent kinase kinase- $\beta$ . *J. Biol. Chem.* 284: M808664200.
5. Xie, Z., et al. 2009. Identification of the serine 307 of LKB1 as a novel phosphorylation site essential for its nucleocytoplasmic transport and endothelial cell angiogenesis. *Mol. Cell. Biol.* 29: 3582-3596.
6. Hattori, Y., et al. 2009. Cilostazol inhibits cytokine-induced nuclear factor- $\kappa$ B activation via AMP-activated protein kinase activation in vascular endothelial cells. *Cardiovasc. Res.* 81: 133-139.
7. Zhang, W.B., et al. 2010. Activation of AMP-activated protein kinase by temozolomide contributes to apoptosis in glioblastoma cells via p53 activation and mTORC1 inhibition. *J. Biol. Chem.* 285: 40461-40471.