

SGK3 [GST-tagged]

Kinase and Substrate

Alternate Names: Serine/threonine-protein kinase Sgk3, Cytokine-independent survival kinase, Serum/glucocorticoid-regulated kinase 3, CISK, SGKL

Cat. No. 66-0021-050

Lot. No. 30300

Quantity: 50 µg

Storage: -70°C

FOR RESEARCH USE ONLY

NOT FOR USE IN HUMANS



CERTIFICATE OF ANALYSIS Page 1 of 2

Background

Protein ubiquitylation and protein phosphorylation are the two major mechanisms that regulate the functions of proteins in eukaryotic cells. However, these different posttranslational modifications do not operate independently of one another, but are frequently interlinked to enable biological processes to be controlled in a more complex and sophisticated manner. Studying how protein phosphorylation events control the ubiquitin system and how ubiquitylation regulates protein phosphorylation has become a focal point of the study of cell regulation and human disease. The serum- and glucocorticoid-inducible protein kinase (SGK) family is made up of three isoforms, SGK1, 2, and 3, that are phosphatidylinositol-3-kinase (PI3-K)-dependent, serine/threonine kinases, with similar substrate specificity to protein kinase B (PKB). Consequently, the SGK family also regulates similar cell processes to the PKB kinases, including cell proliferation and survival (Bruhn *et al.*, 2013). SGK3 functions in parallel to PKB downstream of PI3-K. It shares ~55% identity with PKB in the kinase domain and is also a direct substrate of 3-phosphoinositide-dependent kinase 1 (PDK1). In contrast to PKB, SGK3 lacks the pleckstrin homology domain but contains a phox (PX) homology domain that binds phosphatidylinositol 3'-monophosphate and targets SGK3 to the early endosome (Wang *et al.*, 2014). SGK3 is unique within the SGK family by containing an N-terminal PX

Physical Characteristics

Species: human

Source: *E. coli*

Quantity: 50 µg

Concentration: 4.2 mg/ml

Formulation: 50 mM Tris/HCl pH7.5, 0.1 mM EGTA, 150 mM NaCl, 0.1% β-Mercaptoethanol, 270 mM sucrose, 0.03% Brij-35, 1 mM Benzamidine, 0.2 mM PMSF

Molecular Weight: ~42.4 kDa

Purity: >95% by InstantBlue™ SDS-PAGE

Stability/Storage: 12 months at -70°C; aliquot as required

Protein Sequence: Please see page 2

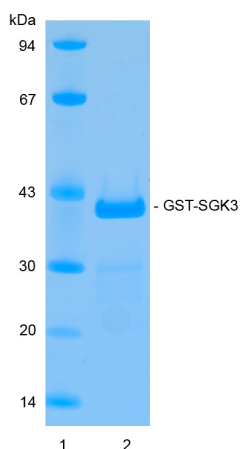
Quality Assurance

Purity:

4-12% gradient SDS-PAGE
InstantBlue™ staining
Lane 1: MW markers
Lane 2: 2.5 µg GST-SGK3

Protein Identification:

Confirmed by mass spectrometry.



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Lot-specific COA version tracker: v1.0.0

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CERTIFICATE OF ANALYSIS Page 2 of 2

Background

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domain which is shown to be important for targeting SGK3 to vesicle-like structures (Bruhn *et al.*, 2013). Cloning of the SGK3 gene was first described by Kobayashi *et al.* (1999). SGK3 (or SGK1) overexpression has been shown to increase phosphorylation of the ubiquitin E3 ligase Nedd4-2, which is known to inhibit Nedd4-2 activity (Lamothe and Zhang, 2013). Activated SGK3 can also promote estrogen/estrogen receptor (ER) dependent transcription and cell survival. Recent studies have established a clinical link between SGK3 and ER, underlining the importance of incorporating SGK3 as a new component in the assessment of breast cancer (Xu *et al.*, 2012).

References:

Bruhn MA, Pearson RB, Hannan RD and Sheppard KE (2013) AKT-independent PI3-K signaling in cancer - emerging role for SGK3. *Cancer Manag Res* 5, 281-292.

Kobayashi T, Deak M, Morrice N and Cohen P (1999) Characterization of the structure and regulation of two novel isoforms of serum- and glucocorticoid-induced protein kinase. *The Biochemical Journal* 344 Pt 1, 189-197.

Lamothe SM and Zhang S (2013) The serum- and glucocorticoid-inducible kinases SGK1 and SGK3 regulate hERG channel expression via ubiquitin ligase Nedd4-2 and GTPase Rab11. *J Biol Chem* 288, 15075-15084.

Wang Y, Xu W, Zhou D, Neckers L and Chen S (2014) Coordinated regulation of serum- and glucocorticoid-inducible kinase 3 by a C-terminal hydrophobic motif and Hsp90-Cdc37 chaperone complex. *J Biol Chem* 289, 4815-4826.

Xu J, Wan M, He Q, Bassett RL, Jr., Fu X, Chen AC, *et al.* (2012) SGK3 is associated with estrogen receptor expression in breast cancer. *Breast Cancer Res Treat* 134, 531-541.

Physical Characteristics

Continued from page 1

Protein Sequence:

MSPILGYWKIKGLVQPTRLLLEYLEEKYEEH
LYERDEGDKWRNKKFELGLEFPNLPYYIDGD
VKLTQSMAIRYIADKHNMLGGCPKERAEISM
LEGAVLDIRYGVSRIRIAYSKDFETLKVDLFL
SKLPEMLKMFEDRLCHKTYLNGDHVTHPD
FMLYDALDVVLYMDPMCLDAFPKLVCFK
KRIEAIPOIDKYLKSSKYIAWPLQGWQAT
FGGGDHPPKSDLEVLVLFQGPLGSMQRDHTM
DYKESCPSVSI PSSDEHREKKRFTVYKVLVS
VGRSEWFVFRRYAEFDKLYNTLKKQFPAMAL
KIPAKRIFGDNFDPDFIKQRRAGLNEFIQN
LVRYPELYNHPDVRAFLQMDSPKHQSDPSE

Tag (**bold text**): N-terminal GST

Protease cleavage site: PreScission™ (LEVLVLFQ▼GP)

SGK3 (regular text): Start **bold italics** (amino acid residues 1-130).

Accession number: NP_037389



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