# Mouse GSK3B Protein (His Tag)

Catalog Number: 50650-M07B



## **General Information**

#### Gene Name Synonym:

7330414F15Rik; 8430431H08Rik; C86142; GSK-3; GSK-3beta; GSK3

#### **Protein Construction:**

A DNA sequence encoding the mouse GSK3B (Q9WV60) (Met 1-Thr 420) was fused with a polyhistidine tag at the N-terminus.

Source: Mouse

Expression Host: Baculovirus-Insect Cells

**QC** Testing

Purity: > 85 % as determined by SDS-PAGE

### **Bio Activity:**

1. The specific activity was determined to be > 20 nmol/min/mg using synthetic Phospho-Glycogen Synthase Peptide-2 (YRRAAVPPSPSLSRHSSPHQpSEDEEE) as substrate. 2. Immobilized His-mGSK3B at 10  $\mu$ g/ml (100  $\mu$ l/well) can bind biotinylated human HG3C-CTNNB1 (cat:11279-H20B), EC<sub>50</sub> of biotinylated human HG3C-CTNNB1 (cat:11279-H20B) is 0.15-0.35  $\mu$ g/ml.

#### **Endotoxin:**

< 1.0 EU per µg of the protein as determined by the LAL method

#### Stability:

Samples are stable for up to twelve months from date of receipt  $\,$  at -70  $\,$   $^{\circ}\mathrm{C}$ 

Predicted N terminal: Me

#### **Molecular Mass:**

The recombinant mouse GSK3B consists of 439 amino acids and has a calculated molecular mass of 49 kDa. It migrates as an approximately 47 kDa band in SDS-PAGE under reducing conditions.

## Formulation:

Supplied as sterile 20mM Tris, 500mM NaCl, 25% glycerol, 0.2mM DTT, pH

Normally 5 % - 8 % trehalose, mannitol and 0.01% Tween80 are added as protectants before lyophilization. Specific concentrations are included in the hardcopy of COA. Please contact us for any concerns or special requirements.

## **Usage Guide**

## Storage:

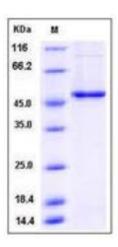
Store it under sterile conditions at  $-20^{\circ}$ C to  $-80^{\circ}$ C upon receiving. Recommend to aliquot the protein into smaller quantities for optimal storage.

## Avoid repeated freeze-thaw cycles.

#### Reconstitution:

Detailed reconstitution instructions are sent along with the products.

#### SDS-PAGE:



## **Protein Description**

GSK3B is a serine-threonine kinase, belonging to the glycogen synthase kinase subfamily. It Contains 1 protein kinase domain, and is expressed in testis, thymus, prostate and ovary and weakly expressed in lung, brain and kidney. GSK3B is involved in energy metabolism, neuronal cell development, and body pattern formation. Polymorphisms in GSK3B gene have been implicated in modifying risk of Parkinson disease, and studies in mice show that overexpression of this gene may be relevant to the pathogenesis of Alzheimer disease. GSK3B participates in the Wnt signaling pathway. It is implicated in the hormonal control of several regulatory proteins including glycogen synthase, MYB and the transcription factor JUN. Phosphorylates JUN at sites proximal to its DNA-binding domain, thereby reducing its affinity for DNA. Phosphorylates MUC1 in breast cancer cells, and decreases the interaction of MUC1 with CTNNB1/beta-catenin. GSK3B also plays an important role in ERBB2dependent stabilization of microtubules at the cell cortex. It prevents the phosphorylation of APC and CLASP2, allowing its association with the cell membrane. In turn, membrane-bound APC allows the localization of MACF1 to the cell membrane, which is required for microtubule capture and stabilization. GSK3B phosphorylates MACF1 and this phosphorylation inhibits the binding of MACF1 to microtubules which is critical for its role in bulge stem cell migration and skin wound repair. It may be required for early embryo development and neuron differentiation.

# References

1.Bergmann C, *et al.* (2011) Inhibition of glycogen synthase kinase  $3\beta$  induces dermal fibrosis by activation of the canonical Wnt pathway. Ann Rheum Dis. 70(12):2191-8. 2.Ban JO, *et al.* (2011) Troglitazone, a PPAR agonist, inhibits human prostate cancer cell growth through inactivation of NF $\kappa$ B via suppression of GSK- $3\beta$  expression. Cancer Biol Ther. 12(4):288-96. 3.Tsukigi M, *et al.* (2012) Re-expression of miR-199a suppresses renal cancer cell proliferation and survival by targeting GSK- $3\beta$ . Cancer Lett. 315(2):189-97.

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