

DESCRIPTION

Source Human embryonic kidney cell, HEK293-derived
Ala25-Thr880, with C-terminal 6-His tag
Accession # NP_001695

N-terminal Sequence Analysis Ala25

Predicted Molecular Mass 97 kDa

SPECIFICATIONS

SDS-PAGE 109-132 kDa, reducing conditions

Activity Measured by its binding ability in a functional ELISA.
When Recombinant Human C1qL3 (Catalog # 9115-TN) is immobilized at 1 µg/mL, 100 µL/well, the concentration of Recombinant Human BAI3 that produces 50% of the optimal binding response is approximately 0.3-1.8 µg/mL.

Endotoxin Level <0.10 EU per 1 µg of the protein by the LAL method.

Purity >90%, by SDS-PAGE visualized with Silver Staining and quantitative densitometry by Coomassie® Blue Staining.

Formulation Lyophilized from a 0.2 µm filtered solution in PBS with Trehalose. See Certificate of Analysis for details.

PREPARATION AND STORAGE

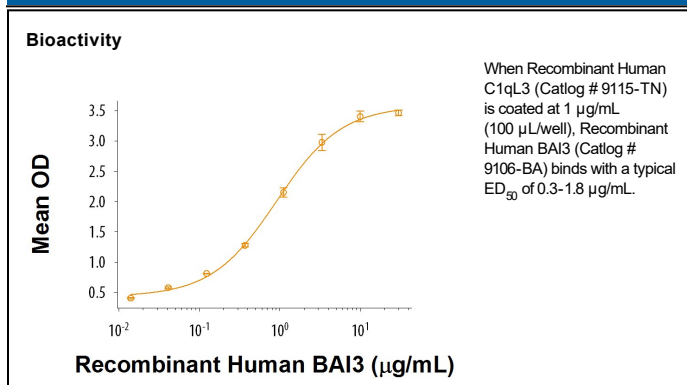
Reconstitution Reconstitute at 1 mg/mL in PBS.

Shipping The product is shipped at ambient temperature. Upon receipt, store it immediately at the temperature recommended below.

Stability & Storage Use a manual defrost freezer and avoid repeated freeze-thaw cycles.

- 12 months from date of receipt, -20 to -70 °C as supplied.
- 1 month, 2 to 8 °C under sterile conditions after reconstitution.
- 3 months, -20 to -70 °C under sterile conditions after reconstitution.

DATA



BACKGROUND

Brain angiogenesis inhibitor 3 (BAI3) is an approximately 180 kDa adhesion GPCR that plays a role in neuronal synapse formation and maintenance. Mature human BAI3 consists of an 856 amino acid (aa) N-terminal extracellular domain (ECD) with one CUB domain, four TSP1 domains, and a GPS protease sensitive linker, followed by a region with seven transmembrane segments and a 376 aa C-terminal cytoplasmic domain (1, 2). Within the N-terminal ECD, human BAI3 shares 98% and 97% aa sequence identity with mouse and rat BAI3, respectively. BAI3 is primarily expressed post-synaptically in the cerebral cortex and on cerebellar Purkinje cells (1-5), and it binds to the TNF-like proteins C1qL1, 2, 3, and 4 (6). BAI3 interaction with C1qL1 is required for the formation and maintenance of excitatory synapses between climbing fibers and parallel fibers with Purkinje cells (4, 5). BAI3 ligation can also reduce excitatory synaptic density in hippocampal neurons, decrease dendrite arborization, and promote axon pruning in climbing fibers (3, 4, 6). It is additionally expressed in developing muscle where it is required for myoblast fusion (7).

References:

1. Shiratsuchi, T. *et al.* (1997) *Cytogenet. Cell Genet.* **79**:103.
2. Kee, H.J. *et al.* (2004) *FEBS Lett.* **569**:307.
3. Lanoue, V. *et al.* (2013) *Mol. Psychiatry* **18**:943.
4. Kakegawa, W. *et al.* (2015) *Neuron* **85**:316.
5. Sigoillot, S.M. *et al.* (2015) *Cell Rep.* **10**:820.
6. Bolliger, M.F. *et al.* (2011) *Proc. Natl. Acad. Sci. USA* **108**:2534.
7. Hamoud, N. *et al.* (2014) *Proc. Natl. Acad. Sci. USA* **111**:3745.