

DESCRIPTION

Species Reactivity	Human
Specificity	Detects human BAI1 in direct ELISAs. In direct ELISAs, less than 2% cross-reactivity with recombinant human BAI3 is observed.
Source	Polyclonal Sheep IgG
Purification	Antigen Affinity-purified
Immunogen	Chinese hamster ovary cell line CHO-derived recombinant human BAI1 Ala31-Thr879 Accession # O14514
Endotoxin Level	<0.10 EU per 1 µg of the antibody by the LAL method.
Formulation	Lyophilized from a 0.2 µm filtered solution in PBS with Trehalose. See Certificate of Analysis for details. *Small pack size (-SP) is supplied either lyophilized or as a 0.2 µm filtered solution in PBS.

APPLICATIONS

Please Note: Optimal dilutions should be determined by each laboratory for each application. *General Protocols* are available in the *Technical Information* section on our website.

Neutralization	Measured by its ability to neutralize the the enhancement of adhesion of BCE C/D1b bovine cornea derived endothelial cells induced by BAI-1. The Neutralization Dose (ND50) is typically 2-8 µg/mL in the presence of 0.5 µg/mL Recombinant Human BAI-1.
-----------------------	--

PREPARATION AND STORAGE

Reconstitution	Reconstitute at 0.2 mg/mL in sterile PBS.
Shipping	The product is shipped at ambient temperature. Upon receipt, store it immediately at the temperature recommended below. *Small pack size (-SP) is shipped with polar packs. Upon receipt, store it immediately at -20 to -70 °C
Stability & Storage	Use a manual defrost freezer and avoid repeated freeze-thaw cycles. <ul style="list-style-type: none"> ● 12 months from date of receipt, -20 to -70 °C as supplied. ● 1 month, 2 to 8 °C under sterile conditions after reconstitution. ● 6 months, -20 to -70 °C under sterile conditions after reconstitution.

BACKGROUND

Brain Angiogenesis Inhibitor 1 (BAI1) is a 170 kDa 7-transmembrane domain G protein-coupled receptor (GPCR) that has a large N-terminal extracellular region with an RGD motif, five thrombospondin type I repeats, and a juxtamembrane GPS (GPCR proteolytic cleavage site) (1). Within the extracellular domain (ECD) up to the GPS (amino acids 31-879), mature human BAI1 shares 94% amino acid sequence identity with mouse and rat BAI1. BAI1 is preferentially expressed on brain neurons but also is found on astrocytes and macrophages and in the pancreas, stomach, and colon (1-8). BAI1 can be cleaved within the GPS to release a 120 kDa fragment termed Vasculostatin which corresponds to nearly the entire N-terminal ECD (9). Generation of additional soluble fragments suggests the cleavage of BAI1 at multiple sites (9, 10). BAI1 fragments interact with Integrin α V β 5 or CD36 on microvascular endothelial cells to inhibit cell proliferation and migration (10, 11). Overexpression of BAI1 in glioblastoma or pancreatic adenocarcinoma cells inhibits their tumorigenicity and the development of tumor-associated neovascularization (6, 12). Fragments of the ECD, including Vasculostatin, also suppress *in vivo* angiogenesis and tumor growth (1, 9, 11). BAI1 is down-regulated in glioblastoma, carcinomas of the pancreas, colon, and stomach and also in experimental ischemia (2, 4, 6-8). Its expression is inversely correlated with tumor vascularity in colorectal and pulmonary carcinomas (8, 13). On macrophages and astrocytes, BAI1 mediates the phagocytosis of apoptotic cells through recognition of cell surface phosphatidylserine (5).

References:

1. Nishimori, H. *et al.* (1997) *Oncogene* **15**:2145.
2. Koh, J.T. *et al.* (2001) *Brain Res. Mol. Brain Res.* **87**:223.
3. Mori, K. *et al.* (2002) *Neurosci. Res.* **43**:69.
4. Kaur, B. *et al.* (2003) *Am. J. Pathol.* **162**:19.
5. Park, D. *et al.* (2007) *Nature* **450**:430.
6. Duda, D.G. *et al.* (2002) *Br. J. Cancer* **86**:490.
7. Lee, J.H. *et al.* (2001) *Int. J. Oncol.* **18**:355.
8. Fukushima, Y. *et al.* (1998) *Int. J. Oncol.* **13**:967.
9. Kaur, B. *et al.* (2005) *Oncogene* **24**:3632.
10. Koh, J.T. *et al.* (2004) *Exp. Cell Res.* **294**:172.
11. Kaur, B. *et al.* (2009) *Cancer Res.* **69**:1212.
12. Kang, X. *et al.* (2006) *Cancer Gene Ther.* **13**:385.
13. Hatanaka, H. *et al.* (2000) *Int. J. Mol. Med.* **5**:181.