

DESCRIPTION

Species Reactivity	Human
Specificity	Detects human BAI1 in direct ELISAs. In direct ELISAs, no cross-reactivity with recombinant human BAI3 is observed.
Source	Monoclonal Mouse IgG ₁ Clone # 542515
Purification	Protein A or G purified from hybridoma culture supernatant
Immunogen	Chinese hamster ovary cell line CHO-derived recombinant human BAI1 Met1-Thr879 Accession # O14514
Conjugate	Alexa Fluor 405 Excitation Wavelength: 405 nm Emission Wavelength: 421 nm
Formulation	Supplied 0.2mg/ml in 1X PBS with RDF1 and 0.09% Sodium Azide *Contains <0.1% Sodium Azide, which is not hazardous at this concentration according to GHS classifications. Refer to the Safety Data Sheet (SDS) for additional information and handling instructions.

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.

Immunohistochemistry Optimal dilution of this antibody should be experimentally determined.

PREPARATION AND STORAGE

Shipping	The product is shipped with polar packs. Upon receipt, store it immediately at the temperature recommended below.
Stability & Storage	Protect from light. Do not freeze. 12 months from date of receipt, 2 to 8 °C as supplied

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 αVβ5 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).

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