Human/Mouse Wnt-5a Antibody

Monoclonal Rat IgG_{2A} Clone # 442625

Catalog Number: MAB645

DESCRIPTION

Species Reactivity	Human/Mouse
Specificity	Detects mouse Wnt-5a in direct ELISAs. In direct ELISAs, no cross-reactivity with recombinant mouse Wnt-1, 2b, 3a, 4, 5b, 8a, 8b, 9b, 10a, 10b, 11, or 16 is observed.
Source	Monoclonal Rat IgG_{2A} Clone # 442625
Purification	Protein A or G purified from hybridoma culture supernatant
Immunogen	CHO-derived recombinant mouse Wnt-5a Gln38-Lys380 Accession # P22725
Formulation	Lyophilized from a 0.2 μm filtered solution in PBS with Trehalose.

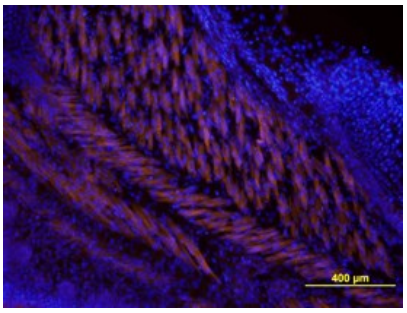
APPLICATIONS

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.*

	Recommended Concentration	Sample
Immunohistochemistry	8-25 µg/mL	See Below

DATA

Immunohistochemistry



Wnt-5a in Mouse Embryo. Wnt-5a was detected in immersion fixed frozen sections of mouse embryo using Human/Mouse Wnt-5a Monoclonal Antibody (Catalog # MAB645) at 10 µg/mL overnight at 4 °C. Tissue was stained using the NorthernLights™ 557-conjugated Anti-Rat IgG Secondary Antibody (orange; Catalog # Catalog # NL013) and counter-stained with DAPI (blue). View our protocol for Fluorescent IHC Staining of Frozen Tissue Sections.

PREPARATION AND STORAGE

Reconstitution	Reconstitute at 0.5 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.** • 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.

Global bio-techne.com info@bio-techne.com techsupport@bio-techne.com TEL +1 612 379 2956
USA TEL 800 343 7475 **Canada** TEL 855 668 8722 **China** TEL +86 (21) 52380373
Europe | Middle East | Africa TEL +44 (0)1235 529449

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BACKGROUND

Wnt proteins are secreted glycoproteins that contain a conserved pattern of 23-24 cysteine residues. Wnts play critical roles in both carcinogenesis and embryonic development for a variety of organisms. Wnts bind to receptors of the Frizzled family, sometimes in conjunction with other membrane-associated proteins such as LRPs or proteoglycans. Downstream effects of Wnt signaling occur through different intracellular components, depending on which pathway is activated. Three pathways have been characterized: the canonical Wnt/β-catenin pathway, the Wnt/Ca^{2+} pathway, and the planar cell polarity (1-2).

Wnt-5a is part of the subgroup of Wnts that are not axis-inducing in *Xenopus* embryos and do not transform C57MG mammary epithelial cells. This subgroup is also implicated in the Wnt/Ca2+ pathway, playing roles in cell movements and cell adhesion (3). This non-canonical Wnt pathway can inhibit canonical Wnt/β-catenin signaling. In Wnt-5a deficient mouse embryos, β-catenin accumulates in the limb bud suggesting that Wnt-5a normally promotes degradation of β-catenin (4). Likewise, in *Xenopus* embryos Wnt-5a antagonizes the ability of the canonical Wnt subgroup to induce a secondary axis (5). Wnt-5a is implicated in various types of cancer and has complex roles. It acts as a tumor suppressor for mammary, B-cell, colon, and uroepithelial cancer cells but is up-regulated in melanomas, where expression levels correlate with severity of metastasis (3). Furthermore, aberrant Wnt-5a signaling results in other diseases such as rheumatoid arthritis (6). Like other developmental growth factors Wnt-5a has diverse roles in development. They are too numerous to enunciate here, as functions span from early anterior-posterior development and gastrulation movements to maintaining hematopoietic stem cell population, lung morphogenesis, and limb outgrowth. Mature Wnt-5a is a 49 kDa protein that shares 99% amino acid identity in mouse, rat and human.

References:

1. Miller, J.R. (2002) Genome Biol. **3**:3001.
2. Roelink, H. and R. Nusse (1991) Genes Dev. **5**:381.
3. Veeman, M.T. *et al.* (2003) Developmental Cell **5**:367.
4. Topol, L. *et al.* (2003) J. Cell Biol **162**:899.
5. Torres, M. *et al.* (1996) J. Cell Biol. **133**:1123.
6. Sen, M. *et al.* (2001) Arthritis & Rheumatism **44**:772.

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