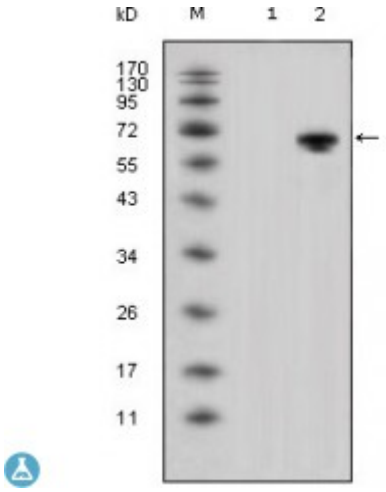
Anti-Wnt-5a antibody



Description	Mouse monoclonal to Wnt-5a.

Model	STJ98453
Host	Mouse
Reactivity	Human
Applications	ELISA, IF, IHC, WB
Immunogen	Purified recombinant fragment of Wnt-5a expressed in E. Coli.
Gene ID	7474
Gene Symbol	WNT5A
Dilution range	WB 1:500-1:2000IHC 1:200-1:1000IF 1:200-1:1000ELISA 1:10000
Specificity	Wnt-5a Monoclonal Antibody detects endogenous levels of Wnt-5a protein.
Tissue Specificity	Expression is increased in differentiated thyroid carcinomas compared to normal thyroid tissue and anaplastic thyroid tumors where expression is low or undetectable. Expression is found in thyrocytes but not in stromal cells (at protein level).
Purification	Affinity purification
Clone ID	6F2
Note	For Research Use Only (RUO).
Protein Name	Protein Wnt-5a
Clonality	Monoclonal
Conjugation	Unconjugated

Isotype	IgG1
Formulation	Ascitic fluid containing 0.03% sodium azide.
Storage Instruction	Store at -20°C, and avoid repeat freeze-thaw cycles.
Database Links	HGNC:12784OMIM:164975
Alternative Names	Protein Wnt-5a
Function	Ligand for members of the frizzled family of seven transmembrane receptors. Can activate or inhibit canonical Wnt signaling, depending on receptor context. In the presence of FZD4, activates beta-catenin signaling. In the presence of ROR2, inhibits the canonical Wnt pathway by promoting beta-catenin degradation through a GSK3-independent pathway which involves down-regulation of beta-catenin-induced reporter gene expression. Suppression of the canonical pathway allows chondrogenesis to occur and inhibits tumor formation. Stimulates cell migration. Decreases proliferation, migration, invasiveness and clonogenicity of carcinoma cells and may act as a tumor suppressor. Mediates motility of melanoma cells. Required during embryogenesis for extension of the primary anterior-posterior axis and for outgrowth of limbs and the genital tubercle. Inhibits type II collagen expression in chondrocytes.
Cellular Localization	Secreted, extracellular space, extracellular matrix.
Post-translational Modifications	Glycosylation is necessary for secretion but not for activity. Palmitoleylation is required for efficient binding to frizzled receptors. Depalmitoleylation leads to Wnt signaling pathway inhibition. Proteolytic processing by TIKI1 and TIKI2 promotes oxidation and formation of large disulfide-bond oligomers, leading to inactivation of WNT5A.