

Anti-FGFR2 / CD332 Reference Antibody (bemarituzumab)
Recombinant Antibody
Catalog # APR10697**Specification**

Anti-FGFR2 / CD332 Reference Antibody (bemarituzumab) - Product Information

| | |
|-------------------|----------------------------|
| Application | FC, Kinetics, Animal Model |
| Primary Accession | P21802 |
| Reactivity | Human, Mouse |
| Clonality | Monoclonal |
| Isotype | IgG1 |
| Calculated MW | 144 KDa |

Anti-FGFR2 / CD332 Reference Antibody (bemarituzumab) - Additional Information**Target/Specificity**
FGFR2 / CD332**Endotoxin**
< 0.001EU/ µg,determined by LAL method.**Conjugation**
Unconjugated**Expression system**
CHO Cell**Format**
Purified monoclonal antibody supplied in PBS, pH6.0, without preservative.This antibody is purified through a protein A column.**Anti-FGFR2 / CD332 Reference Antibody (bemarituzumab) - Protein Information****Name** FGFR2**Synonyms** BEK, KGFR, KSAM**Function**
Tyrosine-protein kinase that acts as a cell-surface receptor for fibroblast growth factors and plays an essential role in the regulation of cell proliferation, differentiation, migration and apoptosis, and in the regulation of embryonic development. Required for normal embryonic patterning, trophoblast function, limb bud development, lung morphogenesis, osteogenesis and skin development. Plays an essential role in the regulation of osteoblast differentiation, proliferation and apoptosis, and is required for normal skeleton development. Promotes cell proliferation in keratinocytes and immature osteoblasts, but promotes apoptosis in differentiated osteoblasts. Phosphorylates PLCG1, FRS2 and PAK4. Ligand binding leads to the activation of several signaling cascades. Activation of PLCG1 leads to the production of the cellular signaling molecules diacylglycerol and inositol 1,4,5-trisphosphate. Phosphorylation of FRS2 triggers recruitment of

GRB2, GAB1, PIK3R1 and SOS1, and mediates activation of RAS, MAPK1/ERK2, MAPK3/ERK1 and the MAP kinase signaling pathway, as well as of the AKT1 signaling pathway. FGFR2 signaling is down-regulated by ubiquitination, internalization and degradation. Mutations that lead to constitutive kinase activation or impair normal FGFR2 maturation, internalization and degradation lead to aberrant signaling. Over-expressed FGFR2 promotes activation of STAT1.

Cellular Location

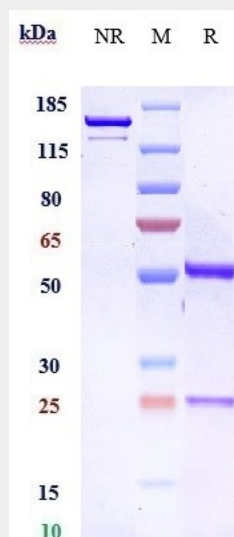
Cell membrane; Single-pass type I membrane protein. Golgi apparatus. Cytoplasmic vesicle. Note=Detected on osteoblast plasma membrane lipid rafts. After ligand binding, the activated receptor is rapidly internalized and degraded [Isoform 3]: Cell membrane; Single-pass type I membrane protein. Note=After ligand binding, the activated receptor is rapidly internalized and degraded [Isoform 13]: Secreted.

Anti-FGFR2 / CD332 Reference Antibody (bemarituzumab) - Protocols

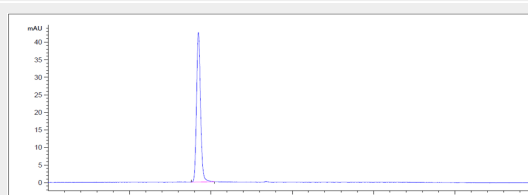
Provided below are standard protocols that you may find useful for product applications.

- [Western Blot](#)
- [Blocking Peptides](#)
- [Dot Blot](#)
- [Immunohistochemistry](#)
- [Immunofluorescence](#)
- [Immunoprecipitation](#)
- [Flow Cytometry](#)
- [Cell Culture](#)

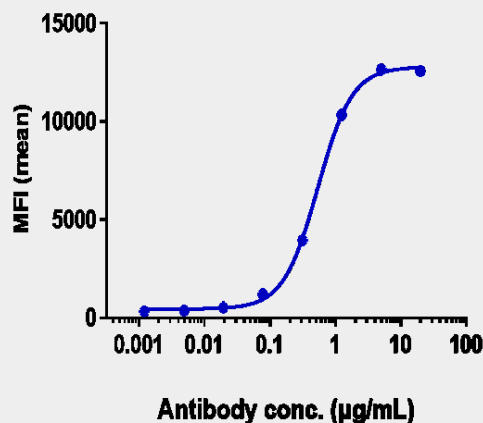
Anti-FGFR2 / CD332 Reference Antibody (bemarituzumab) - Images



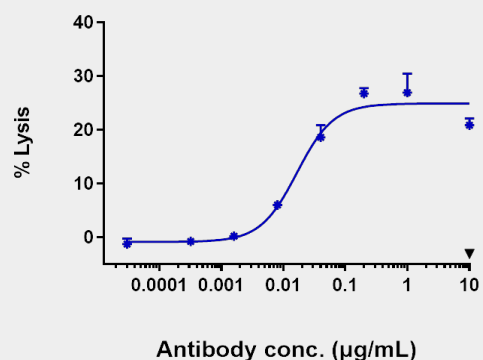
Anti-FGFR2 / CD332 Reference Antibody (bemarituzumab) on SDS-PAGE under reducing (R) condition. The gel was stained with Coomassie Blue. The purity of the protein is greater than 95%



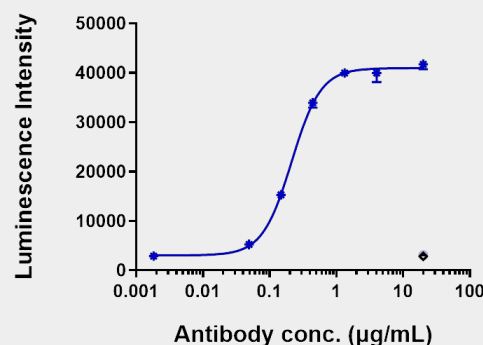
The purity of Anti-FGFR2 / CD332 Reference Antibody (bemarituzumab) is more than 100%, determined by SEC-HPLC.



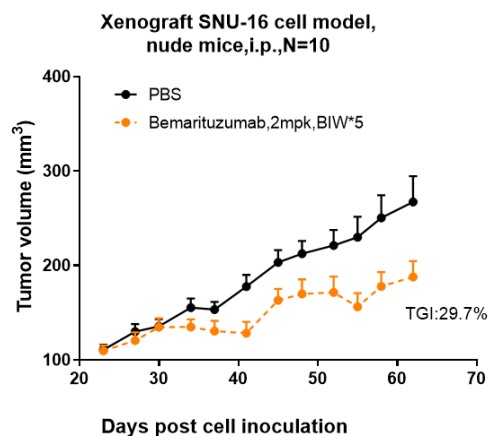
SNU-16 cells were stained with Anti-FGFR2 / CD332 Reference Antibody (bemarituzumab) and negative control protein respectively, washed and then followed by PE and analyzed with FACS, EC763=0.5368 µg/mL



Anti-FGFR2 / CD332 Reference Antibody (bemarituzumab) induced ADCC activity was evaluated using Human FGFR2 HEK293 Reporter Cell. The max induction fold was approximately 25.



Anti-FGFR2 / CD332 Reference Antibody (bemarituzumab) -ADCC luciferase Assay on KATOIII cells. The maximum suppression factor is approximately 14.



Bemarituzumab inhibited the tumor growth of SNU-16 on Balb/c nude mice. The result showed significant anti-tumor effects, with an tumor inhibition rate (TGI) of 29.7% at 2 mpk at D62.