SunLong Biotech Co.,LTD
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# Rabbit Anti-FGFR2 antibody

SL0675R

| | |
|---|---|
| **Product Name** | FGFR2 |
| **Chinese Name** | 成纤维细胞生长因子受体 2 抗体 |
| **Alias** | KGFR; KSAM; Bacteria expressed kinase; BEK; BEK fibroblast growth factor receptor; BFR 1; 332; CD332; CD332 antigen; CEK 3; CEK3; CFD 1; CFD1; Craniofacial dysostosis 1; Crouzon ECT 1; ECT 1; ECT1; FGF receptor; FGFR 2; FGFR-2; FGFR2_HUMAN; Fibroblast growth fa 2; Hydroxyaryl protein kinase; Hydroxyaryl protein kinase; Jackson Weiss syndrome; JWS; JWS SAM; K sam protein; K sam protein; K-sam ; Keratinocyte growth factor receptor 2; Keratinocy factor receptor; Pfeiffer syndrome; Protein tyrosine kinase receptor like 14; TK14; TK25; Tyros kinase; Tyrosylprotein kinase. |
| **Research Area** | Tumour Cardiovascular Neurobiology Signal transduction Stem cells Growth factors and hormo |
| **Immunogen Species** | Rabbit |
| **Clonality** | Polyclonal |
| **React Species** | Human, Mouse, (predicted: Rat, ) |
| **Applications** | WB=1:500-2000,IHC-P=1:100-500,IHC-F=1:100-500,ICC/IF=1:100-500,IF=1:100-500,Flow-C （Paraffin sections need antigen repair） not yet tested in other applications. optimal dilutions/concentrations should be determined by the end user. |
| **Theoretical molecular weight** | 89kDa |
| **Cellular localization** | The cell membrane Secretory protein |
| **Form** | Liquid |
| **Concentration** | 1mg/ml |
| **immunogen** | KLH conjugated synthetic peptide derived from human FGFR2: 21-120/821 <Extracellular> |
| **Lsotype** | IgG |
| **Purification** | affinity purified by Protein A |
| **Buffer Solution** | 1M TBS(pH7.4) with 1% BSA, 3% Proclin300 and 50% Glycerol. |
| **Storage** | Shipped at 4℃. Store at -20 °C for one year. Avoid repeated freeze/thaw cycles. |

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**Attention** This product as supplied is intended for research use only, not for use in human, therapeutic or d applications.

**PubMed** PubMed

**Product Detail**

The protein encoded by this gene is a member of the fibroblast growth factor receptor family, wl acid sequence is highly conserved between members and throughout evolution. FGFR family me from one another in their ligand affinities and tissue distribution. A full-length representative pro of an extracellular region, composed of three immunoglobulin-like domains, a single hydrophob membrane-spanning segment and a cytoplasmic tyrosine kinase domain. The extracellular portic protein interacts with fibroblast growth factors, setting in motion a cascade of downstream signal influencing mitogenesis and differentiation. This particular family member is a high-affinity rece acidic, basic and/or keratinocyte growth factor, depending on the isoform. Mutations in this gene associated with Crouzon syndrome, Pfeiffer syndrome, Craniosynostosis, Apert syndrome, Jacks syndrome, Beare-Stevenson cutis gyrata syndrome, Saethre-Chotzen syndrome, and syndromic craniosynostosis. Multiple alternatively spliced transcript variants encoding different isoforms ha noted for this gene. [provided by RefSeq, Jan 2009]

**Function:**
Tyrosine-protein kinase that acts as cell-surface receptor for fibroblast growth factors and plays a role in the regulation of cell proliferation, differentiation, migration and apoptosis, and in the reg embryonic development. Required for normal embryonic patterning, trophoblast function, limb b development, lung morphogenesis, osteogenesis and skin development. Plays an essential role in regulation of osteoblast differentiation, proliferation and apoptosis, and is required for normal sk development. Promotes cell proliferation in keratinocytes and immature osteoblasts, but promote in differentiated osteoblasts. Phosphorylates PLCG1, FRS2 and PAK4. Ligand binding leads to t of several signaling cascades. Activation of PLCG1 leads to the production of the cellular signalin diacylglycerol and inositol 1,4,5-trisphosphate. Phosphorylation of FRS2 triggers recruitment of GAB1, PIK3R1 and SOS1, and mediates activation of RAS, MAPK1/ERK2, MAPK3/ERK1 and kinase signaling pathway, as well as of the AKT1 signaling pathway. FGFR2 signaling is down- ubiquitination, internalization and degradation. Mutations that lead to constitutive kinase activati normal FGFR2 maturation, internalization and degradation lead to aberrant signaling. Over-expre promotes activation of STAT1.

**Subunit:**
Monomer. Homodimer after ligand binding. Interacts predominantly with FGF1 and FGF2, but c interact with FGF3, FGF4, FGF6, FGF7, FGF8, FGF9, FGF10, FGF17, FGF18 and FGF22 (in v specificity is determined by tissue-specific expression of isoforms, and differences in the third Ig are crucial for ligand specificity. Isoform 1 has high affinity for FGF1 and FGF2, but low affinit Isoform 3 has high affinity for FGF1 and FGF7, and has much higher affinity for FGF7 than isof vitro). Affinity for fibroblast growth factors (FGFs) is increased by heparan sulfate glycosamino function as coreceptors. Likewise, KLB increases the affinity for FGF19 and FGF21. Interacts w GRB2 and PAK4.

**Subcellular Location:**

Cell membrane; Single-pass type I membrane protein. Golgi apparatus. Cytoplasmic vesicle. No
on osteoblast plasma membrane lipid rafts. After ligand binding, the activated receptor is rapidly
and degraded.
Isoform 1: Cell membrane; Single-pass type I membrane protein. Note=After ligand binding, the
receptor is rapidly internalized and degraded.
Isoform 3: Cell membrane; Single-pass type I membrane protein. Note=After ligand binding, the
receptor is rapidly internalized and degraded.

**Post-translational modifications:**
N-glycosylated in the endoplasmic reticulum. The N-glycan chains undergo further maturation t
H-resistant form in the Golgi apparatus.
Ubiquitinated. FGFR2 is rapidly ubiquitinated after autophosphorylation, leading to internalizati
degradation. Subject to degradation both in lysosomes and by the proteasome.

**DISEASE:**
Defects in FGFR2 are the cause of Crouzon syndrome (CS) [MIM:123500]; also called craniofaci
type I (CFD1). CS is an autosomal dominant syndrome characterized by craniosynostosis (premat
the skull sutures), hypertelorism, exophthalmos and external strabismus, parrot-beaked nose, sho
hypoplastic maxilla, and a relative mandibular prognathism.
Defects in FGFR2 are a cause of Jackson-Weiss syndrome (JWS) [MIM:123150]. JWS is an aut
dominant craniosynostosis syndrome characterized by craniofacial abnormalities and abnormalit
broad great toes with medial deviation and tarsal-metatarsal coalescence. Defects in FGFR2 are
Apert syndrome (APRS) [MIM:101200]; also known as acrocephalosyndactyly type 1 (ACS1).
syndrome characterized by facio-cranio-synostosis, osseous and membranous syndactyly of the
extremities, and midface hypoplasia. The craniosynostosis is bicoronal and results in acrocephal
brachysphenocephalic type. Syndactyly of the fingers and toes may be total (mitten hands and so
partial affecting the second, third, and fourth digits. Intellectual deficit is frequent and often seve
being associated with cerebral malformations.
Defects in FGFR2 are a cause of Pfeiffer syndrome (PS) [MIM:101600]; also known as
acrocephalosyndactyly type V (ACS5). PS is characterized by craniosynostosis (premature fusion
sutures) with deviation and enlargement of the thumbs and great toes, brachymesophalangy, with
ankylosis and a varying degree of soft tissue syndactyly. Three subtypes of Pfeiffer syndrome ha
described: mild autosomal dominant form (type 1); cloverleaf skull, elbow ankylosis, early death
(type 2); craniosynostosis, early demise, sporadic (type 3).
Defects in FGFR2 are the cause of Beare-Stevenson cutis gyrata syndrome (BSCGS) [MIM:1237
is an autosomal dominant condition is characterized by the furrowed skin disorder of cutis gyrata
nigricans, craniosynostosis, craniofacial dysmorphism, digital anomalies, umbilical and anogenit
abnormalities and early death.
Defects in FGFR2 are the cause of familial scaphocephaly syndrome (FSPC) [MIM:609579]; als
scaphocephaly with maxillary retrusion and mental retardation. FSPC is an autosomal dominant
craniosynostosis syndrome characterized by scaphocephaly, macrocephaly, hypertelorism, maxil
retrusion, and mild intellectual disability. Scaphocephaly is the most common of the craniosynos
conditions and is characterized by a long, narrow head. It is due to premature fusion of the sagitt
from external deformation.

Defects in FGFR2 are a cause of lacrimo-auriculo-dento-digital syndrome (LADDS) [MIM:149
known as Levy-Hollister syndrome. LADDS is a form of ectodermal dysplasia, a heterogeneous
disorders due to abnormal development of two or more ectodermal structures. LADDS is an auto
dominant syndrome characterized by aplastic/hypoplastic lacrimal and salivary glands and ducts
ears, hearing loss, hypodontia and enamel hypoplasia, and distal limb segments anomalies. In add
cardinal features, facial dysmorphism, malformations of the kidney and respiratory system and a
genitalia have been reported. Craniosynostosis and severe syndactyly are not observed.
Defects in FGFR2 are the cause of Antley-Bixler syndrome (ABS) [MIM:207410]. ABS is a mu
congenital anomaly syndrome characterized by craniosynostosis, radiohumeral synostosis, midfa
hypoplasia, malformed ears, arachnodactyly and multiple joint contractures. ABS is a heterogene
and occurs with and without abnormal genitalia in both sexes.

**Similarity:**
Belongs to the protein kinase superfamily. Tyr protein kinase family. Fibroblast growth factor re
subfamily.
Contains 3 Ig-like C2-type (immunoglobulin-like) domains.
Contains 1 protein kinase domain.

**SWISS:**
P21802

**Gene ID:**
2263

**Database links:**

Entrez Gene: 2263 Human

Entrez Gene: 14183 Mouse

Entrez Gene: 25022 Rat

Omim: 176943 Human

SwissProt: P21802 Human

SwissProt: P21803 Mouse

Unigene: 533683 Human

Unigene: 16340 Mouse

Unigene: 12732 Rat

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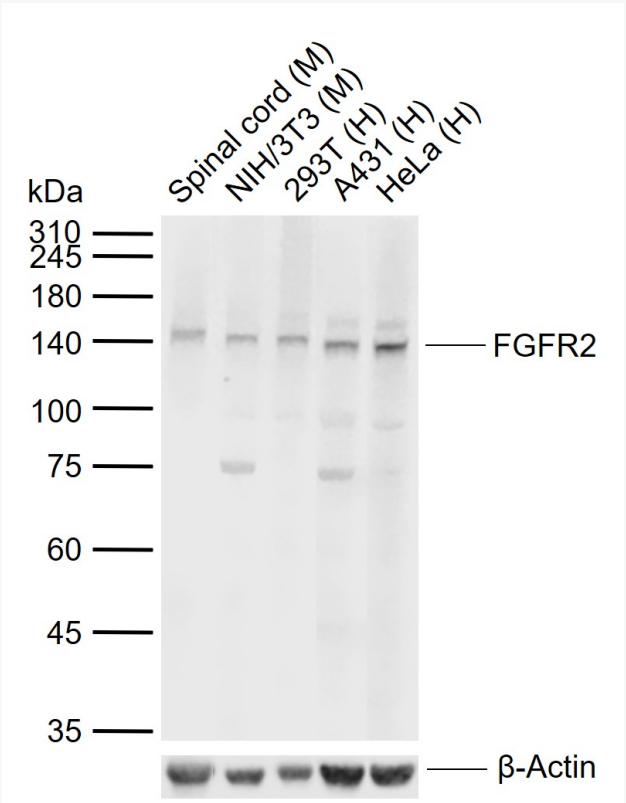
KGFR 又称 FGFR2（Fibroblast Growth Factor Receptor 2）成纤维细胞生长因子受体又称：生长因子受体 2，是 FGFRs 家族的一员。不同的 FGFR 对 FGF 亲和力不同，在组织的分布 FGFR-2 对细胞的增殖、分化、血管生成、胚胎及骨骼发育和在与生长发育相关的进程中要的作用.

**Product Picture**




Sample:

Lane 1: Mouse Spinal cord tissue lysates

Lane 2: Mouse NIH/3T3 cell lysates

Lane 3: Human 293T cell lysates

Lane 4: Human A431 cell lysates

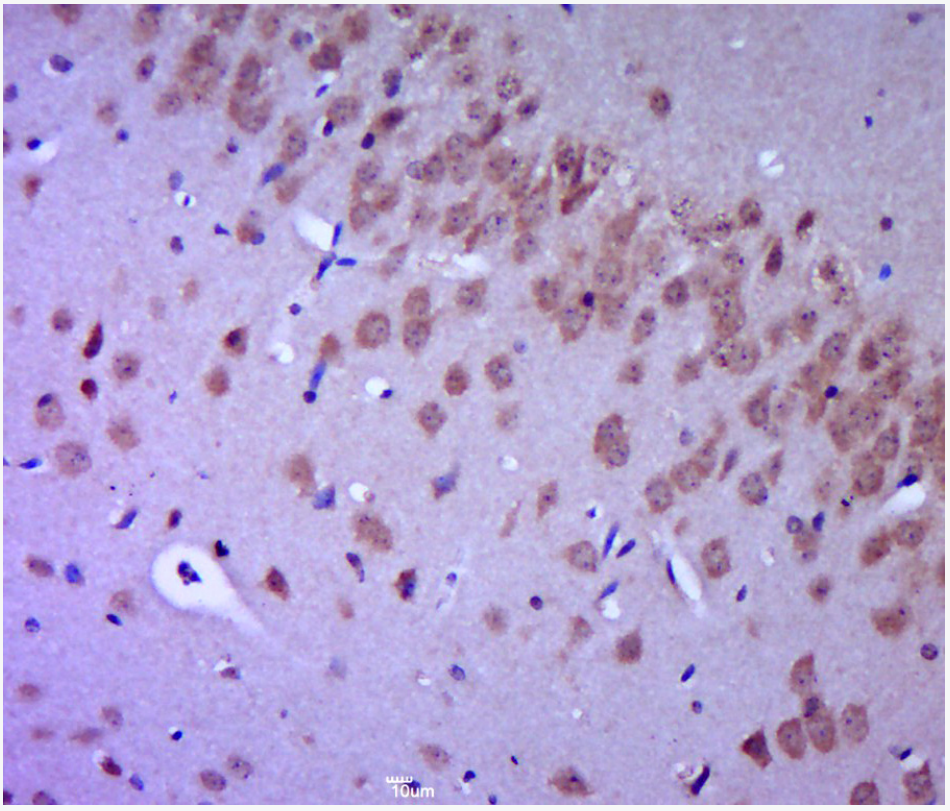
Lane 5: Human HeLa cell lysates

Primary: Anti-FGFR2 (SL0675R) at 1/1000 dilution

Secondary: IRDye800CW Goat Anti-Rabbit IgG at 1/20000 dilution

Predicted band size: 89 kDa

Observed band size: 142 kDa




Paraformaldehyde-fixed, paraffin embedded (mouse brain tissue); Antigen retrieval by boiling
citrate buffer (pH6.0) for 15min; Block endogenous peroxidase by 3% hydrogen peroxide for 2
Blocking buffer (normal goat serum) at 37°C for 30min; Antibody incubation with (FGFR2) P
Antibody, Unconjugated (SL0675R) at 1:400 overnight at 4°C, followed by a conjugated seco
(sp-0023) for 20 minutes and DAB staining.

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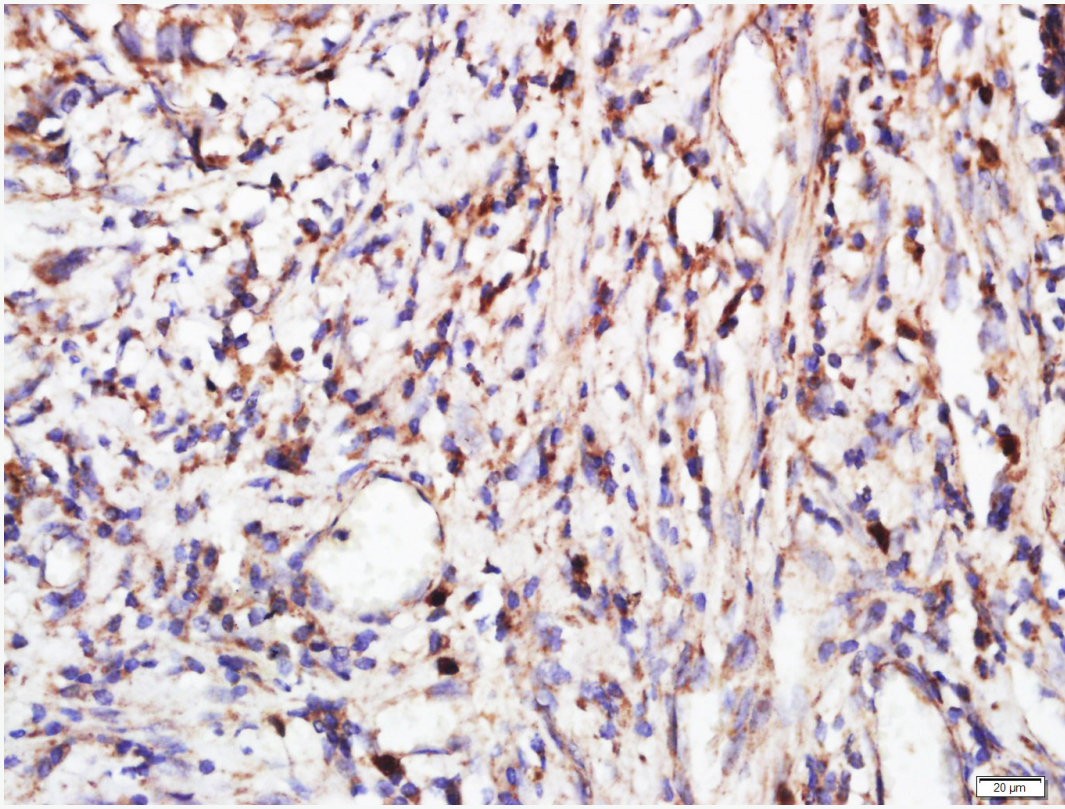


Paraformaldehyde-fixed, paraffin embedded (human stomach cancer); Antigen retrieval by bo
sodium citrate buffer (pH6.0) for 15min; Block endogenous peroxidase by 3% hydrogen perox
minutes; Blocking buffer (normal goat serum) at 37°C for 30min; Antibody incubation with (
Polyclonal Antibody, Unconjugated (SL0675R) at 1:400 overnight at 4°C, followed by a conj
secondary (sp-0023) for 20 minutes and DAB staining.

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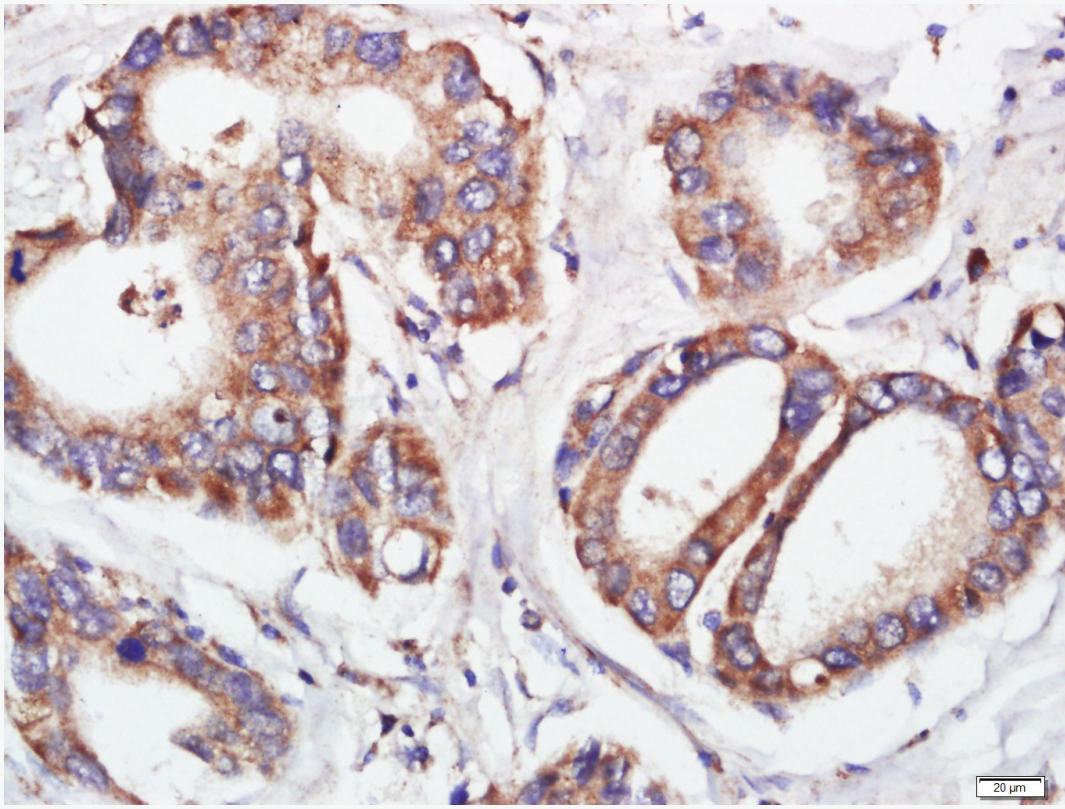


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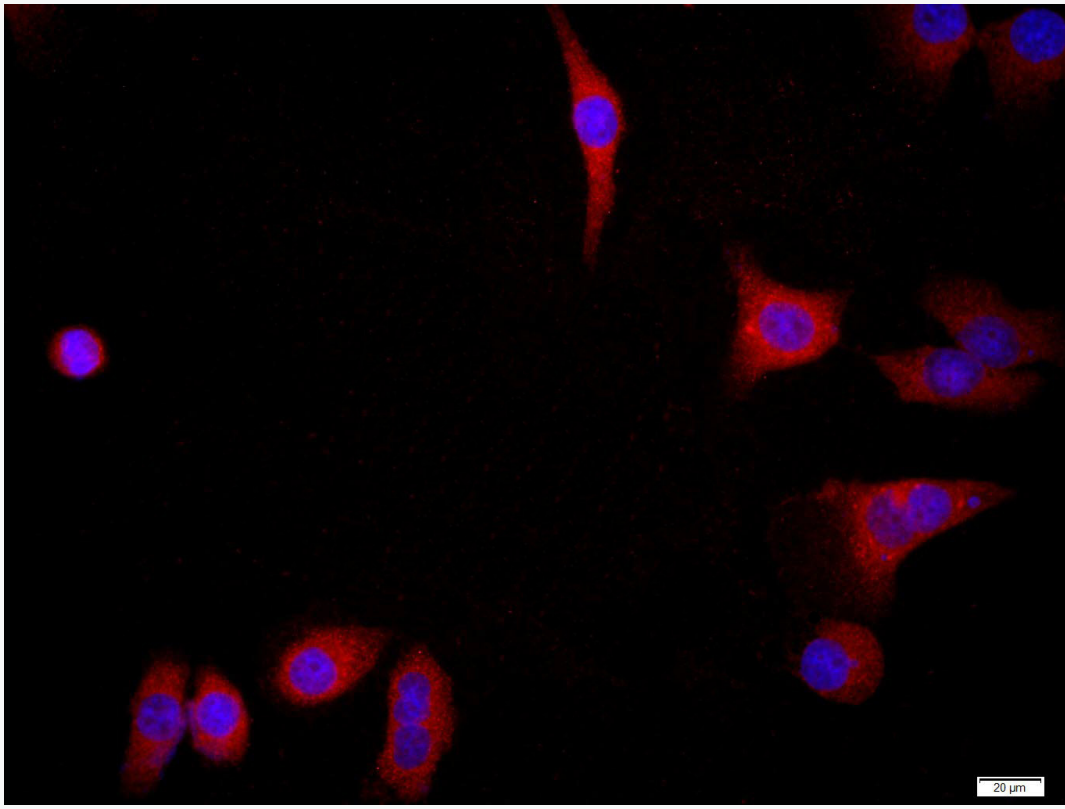


Tissue/cell: MCF7; 4% Paraformaldehyde-fixed; Triton X-100 at room temperature for 20 mi
buffer (normal goat serum, C-0005) at 37°C for 20 min; Antibody incubation with (FGFR2) P
Antibody, Unconjugated (SL0675R) 1:200, 90 minutes at 37°C; followed by a conjugated Go
Anti-Rabbit IgG antibody (SL0295G-FITC) at 37°C for 90 minutes, DAPI (blue, C02-04002)
stain the cell nuclei.

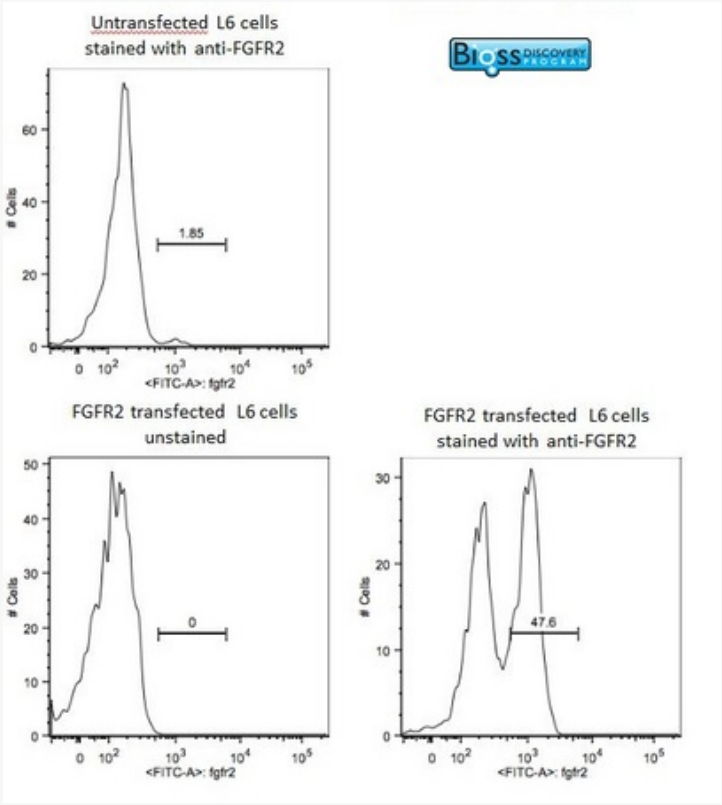


Image was kindly submitted by Dr. Uthaman from Yale University. L6 cells were transfected FGFR2, and stained with RABBIT ANTI-FGFR2 POLYCLONAL ANTIBODY, conjugated (SL0675R-FITC) at 1:100 dilution