abcam

Product datasheet

Anti-FGFR3 (phospho Y724) antibody [EPR2281(3)] ab155960

リコンビナント RabMAb

★★★★☆ 1 Abreviews 10 References 画像数 8

製品の概要

製品名 Anti-FGFR3 (phospho Y724) antibody [EPR2281(3)]

製品の詳細 Rabbit monoclonal [EPR2281(3)] to FGFR3 (phospho Y724)

由来種 Rabbit

アプリケーション **適用あり:** Flow Cyt (Intra), Dot blot, WB, ICC/IF, IP
適用なし: IHC-P

種交差性 **交差種:** Human
交差が予測される動物種: Mouse

免疫原 Synthetic peptide within Human FGFR3 (phospho Y724). The exact sequence is proprietary.
Database link: **P22607**

ポジティブ・コントロール WB: MCF7 lysate treated with pervanadate. ICC/IF: MCF7 cells treated with pervanadate. IP: MCF7 cells treated with pervanadate. Flow Cyt (intra): MCF7 cells

特記事項 This product is a recombinant monoclonal antibody, which offers several advantages including:
- High batch-to-batch consistency and reproducibility
- Improved sensitivity and specificity
- Long-term security of supply
- Animal-free production
For more information **see here**.
Our RabMAb® technology is a patented hybridoma-based technology for making rabbit monoclonal antibodies. For details on our patents, please refer to **RabMAb® patents**.

Rat: We have preliminary internal testing data to indicate this antibody may not react with this species. Please contact us for more information.

製品の特性

製品の状態 Liquid

保存方法 Shipped at 4°C. Store at +4°C short term (1-2 weeks). Upon delivery aliquot. Store at -20°C. Avoid freeze / thaw cycle.

バッファー Preservative: 0.01% Sodium azide
Constituents: 40% Glycerol, 0.05% BSA, PBS

精製度	Protein A purified
ポリ/モノ	モノクローナル
クローン名	EPR2281(3)
アイソタイプ	IgG

アプリケーション

The Abpromise guarantee Abpromise保証は、次のテスト済みアプリケーションにおけるab155960の使用に適用されます

アプリケーションノートには、推奨の開始希釈率がありますが、適切な希釈率につきましてはご検討ください。

アプリケーション	Abreviews	特記事項
Flow Cyt (Intra)		1/50.
Dot blot		1/1000.
WB	★★★★☆ (1)	1/1000 - 1/10000. Predicted molecular weight: 88 kDa.
ICC/IF		1/80 - 1/300.
IP		1/15 - 1/50.

追加情報 Is unsuitable for IHC-P.

ターゲット情報

機能 Receptor for acidic and basic fibroblast growth factors. Preferentially binds FGF1.

組織特異性 Expressed in brain, kidney and testis. Very low or no expression in spleen, heart, and muscle. In 20- to 22-week old fetuses it is expressed at high level in kidney, lung, small intestine and brain, and to a lower degree in spleen, liver, and muscle. Isoform 2 is detected in epithelial cells. Isoform 1 is not detected in epithelial cells. Isoform 1 and isoform 2 are detected in fibroblastic cells.

関連疾患 Defects in FGFR3 are the cause of achondroplasia (ACH) [MIM:100800]. ACH is an autosomal dominant disease and is the most frequent form of short-limb dwarfism. It is characterized by a long, narrow trunk, short extremities, particularly in the proximal (rhizomelic) segments, a large head with frontal bossing, hypoplasia of the midface and a trident configuration of the hands.
Defects in FGFR3 are the cause of Crouzon syndrome with acanthosis nigricans (CAN) [MIM:612247]. Classic Crouzon disease which is caused by mutations in the FGFR2 gene is characterized by craniosynostosis (premature fusion of the skull sutures), and facial hypoplasia. Crouzon syndrome with acanthosis nigricans (a skin disorder characterized by pigmentation anomalies), CAN, is considered to be an independent disorder from classic Crouzon syndrome. CAN is characterized by additional more severe physical manifestation, such as Chiari malformation, hydrocephalus, and atresia or stenosis of the choanas, and is caused by a specific mutation (Ala-391 to Glu) in the transmembrane domain of FGFR3. It is proposed to have an autosomal dominant mode of inheritance.
Defects in FGFR3 are a cause of thanatophoric dysplasia type (TD) [MIM:187600, 187601]; also known as thanatophoric dwarfism or platyspondylic lethal skeletal dysplasia Sand Diego type (PLSD-SD). TD is the most common neonatal lethal skeletal dysplasia. Affected individuals display features similar to those seen in homozygous achondroplasia. It causes severe shortening

of the limbs with macrocephaly, narrow thorax and short ribs. In the most common subtype, TD1, femur are curved, while in TD2, straight femurs are associated with cloverleaf skull. Mutations affecting different functional domains of FGFR3 cause different forms of this lethal disorder.
Defects in FGFR3 are a cause of hypochondroplasia (HCH) [MIM:146000]. HCH is an autosomal dominant disease and is characterized by disproportionate short stature. It resembles achondroplasia, but with a less severe phenotype.
Defects in FGFR3 are a cause of susceptibility to bladder cancer (BLC) [MIM:109800]. A malignancy originating in tissues of the urinary bladder. It often presents with multiple tumors appearing at different times and at different sites in the bladder. Most bladder cancers are transitional cell carcinomas. They begin in cells that normally make up the inner lining of the bladder. Other types of bladder cancer include squamous cell carcinoma (cancer that begins in thin, flat cells) and adenocarcinoma (cancer that begins in cells that make and release mucus and other fluids). Bladder cancer is a complex disorder with both genetic and environmental influences. Note=Somatic mutations can constitutively activate FGFR3.
Defects in FGFR3 are a cause of cervical cancer (CERCA) [MIM:603956]. A malignant neoplasm of the cervix, typically originating from a dysplastic or premalignant lesion previously present at the active squamocolumnar junction. The transformation from mild dysplastic to invasive carcinoma generally occurs slowly within several years, although the rate of this process varies widely. Carcinoma in situ is particularly known to precede invasive cervical cancer in most cases. Cervical cancer is strongly associated with infection by oncogenic types of human papillomavirus.
Defects in FGFR3 are the cause of camptodactyly tall stature and hearing loss syndrome (CATSHL syndrome) [MIM:610474]. CATSHL syndrome is an autosomal dominant syndrome characterized by permanent and irreducible flexion of one or more fingers of the hand and/or feet, tall stature, scoliosis and/or a pectus excavatum, and hearing loss. Affected individuals have developmental delay and/or mental retardation, and several of these have microcephaly. Radiographic findings included tall vertebral bodies with irregular borders and broad femoral metaphyses with long tubular shafts. On audiological exam, each tested member have bilateral sensorineural hearing loss and absent otoacoustic emissions. The hearing loss was congenital or developed in early infancy, progressed variably in early childhood, and range from mild to severe. Computed tomography and magnetic resonance imaging reveal that the brain, middle ear, and inner ear are structurally normal.
Defects in FGFR3 are a cause of multiple myeloma (MM) [MIM:254500]. MM is a malignant tumor of plasma cells usually arising in the bone marrow and characterized by diffuse involvement of the skeletal system, hyperglobulinemia, Bence-Jones proteinuria and anemia. Complications of multiple myeloma are bone pain, hypercalcemia, renal failure and spinal cord compression. The aberrant antibodies that are produced lead to impaired humoral immunity and patients have a high prevalence of infection. Amyloidosis may develop in some patients. Multiple myeloma is part of a spectrum of diseases ranging from monoclonal gammopathy of unknown significance (MGUS) to plasma cell leukemia. Note=A chromosomal aberration involving FGFR3 is found in multiple myeloma. Translocation t(4;14)(p16.3;q32.3) with the IgH locus.
Defects in FGFR3 are a cause of lacrimo-auriculo-dento-digital syndrome (LADDS) [MIM:149730]; also known as Levy-Hollister syndrome. LADDS is a form of ectodermal dysplasia, a heterogeneous group of disorders due to abnormal development of two or more ectodermal structures. LADDS is an autosomal dominant syndrome characterized by aplastic/hypoplastic lacrimal and salivary glands and ducts, cup-shaped ears, hearing loss, hypodontia and enamel hypoplasia, and distal limb segments anomalies. In addition to these cardinal features, facial dysmorphism, malformations of the kidney and respiratory system and abnormal genitalia have been reported. Craniosynostosis and severe syndactyly are not observed.
Defects in FGFR3 are a cause of keratinocytic non-epidermolytic nevus (KNEN) [MIM:162900]; also known as pigmented moles. Epidermal nevi of the common, non-organoid and non-epidermolytic type are benign skin lesions and may vary in their extent from a single (usually linear) lesion to widespread and systematized involvement. They may be present at birth or

develop early during childhood.
Defects in FGFR3 are a cause of Muenke syndrome (MNKS) [MIM:602849]; also known as Muenke non-syndromic coronal craniosynostosis. MNKS is a condition characterized by premature closure of coronal suture of skull during development (coronal craniosynostosis), which affects the shape of the head and face. It may be uni- or bilateral. When bilateral, it is characterized by a skull with a small antero-posterior diameter (brachycephaly), often with a decrease in the depth of the orbits and hypoplasia of the maxillae. Unilateral closure of the coronal sutures leads to flattening of the orbit on the involved side (plagiocephaly). The intellect is normal. In addition to coronal craniosynostosis some affected individuals show skeletal abnormalities of hands and feet, sensorineural hearing loss, mental retardation and respiratory insufficiency.
Defects in FGFR3 are a cause of keratosis seborrheic (KERSEB) [MIM:182000]. A common benign skin tumor. Seborrheic keratoses usually begin with the appearance of one or more sharply defined, light brown, flat macules. The lesions may be sparse or numerous. As they initially grow, they develop a velvety to finely verrucous surface, followed by an uneven warty surface with multiple plugged follicles and a dull or lackluster appearance.

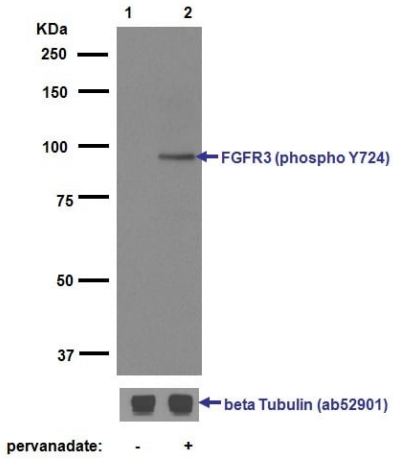
配列類似性

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

細胞内局在

Membrane.

画像



Western blot - Anti-FGFR3 (phospho Y724) antibody [EPR2281(3)] (ab155960)

All lanes : Anti-FGFR3 (phospho Y724) antibody [EPR2281(3)] (ab155960) at 1/20000 dilution (purified)

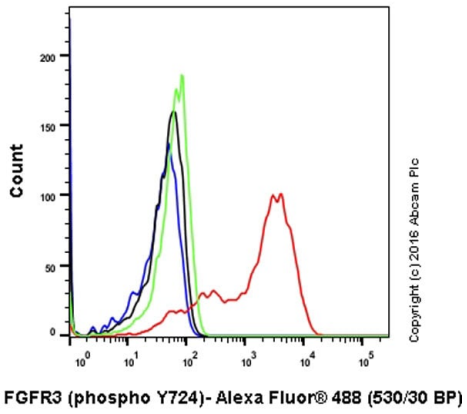
Lane 1 : MCF7 cell lysate - untreated
Lane 2 : MCF7 cell lysate - treated with pervanadate

Lysates/proteins at 10 µg per lane.

Secondary
All lanes : Peroxidase-conjugated goat anti-rabbit IgG (H+L) at 1/1000 dilution

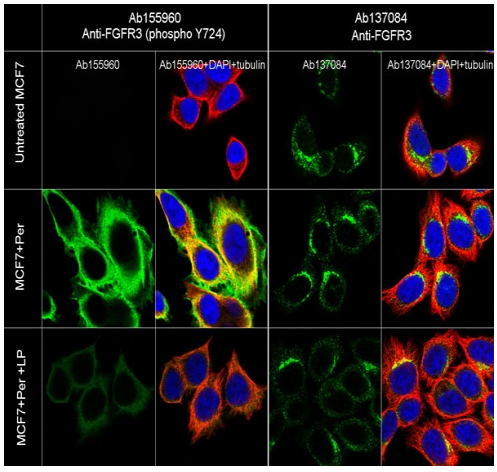
Predicted band size: 88 kDa
Observed band size: 96 kDa

Blocking and dilution buffer: 5% NFDM/TBST.



Flow Cytometry (Intracellular) - Anti-FGFR3 (phospho Y724) antibody [EPR2281(3)] (ab155960)

Intracellular Flow Cytometry analysis of MCF7 (human breast carcinoma) treated with 1 mM pervanadate for 30 minutes cells labeling FGFR3 (phospho Y724) with purified ab155960 at 1/50 dilution (red). The secondary antibody was Goat anti rabbit IgG (Alexa Fluor® 488) at 1/2000 dilution. Green shows untreated MCF7 (human breast carcinoma) cells. A Rabbit monoclonal IgG (Black) was used as the isotype control and cells without incubation with primary antibody and secondary antibody (Blue) were used as unlabeled control.



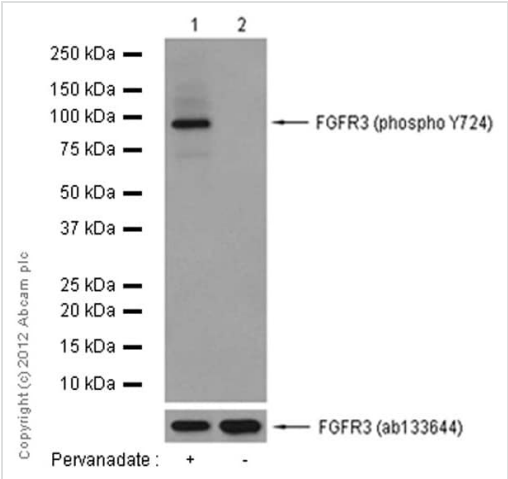
Immunocytochemistry/ Immunofluorescence - Anti-FGFR3 (phospho Y724) antibody [EPR2281(3)] (ab155960)

Immunocytochemistry/Immunofluorescence analysis of untreated, Per treated and Per + LP treated MCF7 cells labelling FGFR3 (phospho Y724) with ab155960 (left) and FGFR3 with **ab137084** (right) both at a dilution of 1/200.

Cells were fixed with 100% methanol. **ab150077**, an Alexa Fluor® 488-conjugated goat anti-rabbit IgG (1/1000) was used as the secondary antibody. DAPI (blue) was used as the nuclear counterstain. **ab7291**, a mouse anti-tubulin (1/1000) and **ab150120**, an Alexa Fluor® 594-conjugated goat anti-mouse IgG (1/1000) were also used.

The image shows increased cytoplasmic staining after Pervanadate (1 mM, 30 min) treatment on MCF7 cells. The LP treatment decreased the cytoplasmic staining caused by Pervanadate.

ab137084 was used as a Pan control for ab155960. The results showed cytoplasmic staining on untreated, Per treated and Per + LP treated MCF7 cells.



Western blot - Anti-FGFR3 (phospho Y724) antibody [EPR2281(3)] (ab155960)

All lanes : Anti-FGFR3 (phospho Y724) antibody [EPR2281(3)] (ab155960) at 1/10000 dilution (unpurified)

Lane 1 : MCF7 whole cell lysate - treated with pervanadate
Lane 2 : MCF7 whole cell lysate - untreated

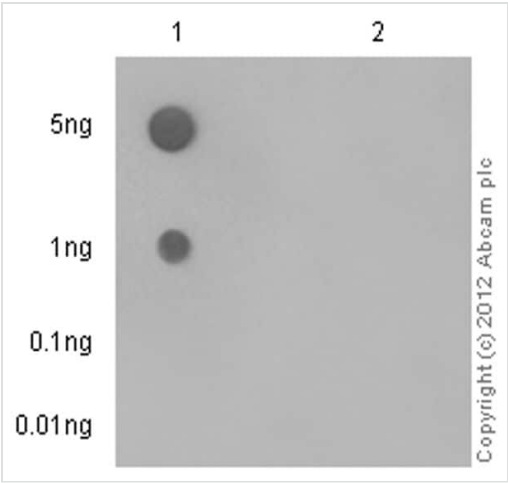
Lysates/proteins at 10 µg per lane.

Secondary
All lanes : Peroxidase-conjugated goat anti-rabbit IgG (H+L) at 1/1000 dilution

Predicted band size: 88 kDa
Observed band size: 98 kDa

Exposure time: 30 seconds

Blocking and dilution buffer: 5% NFDM/TBST.

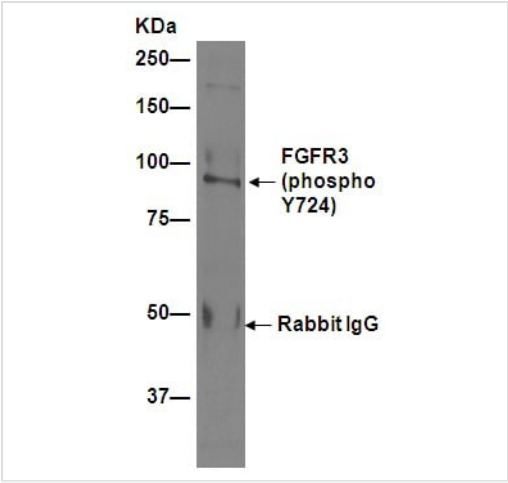


Dot Blot - Anti-FGFR3 (phospho Y724) antibody [EPR2281(3)] (ab155960)

Dot blot analysis of human FGFR3 (pY724) phospho peptide (lane 1) and human FGFR3 non-phospho peptide (lane 2) labelling FGFR2 (phospho Y724) with ab155960 at a dilution of 1/1000. A peroxidase-conjugated goat anti-rabbit IgG (H+L) was used as the secondary antibody (1/2500).

Blocking and dilution buffer: 5% NFDM/TBST.

Exposure time: 10 seconds.

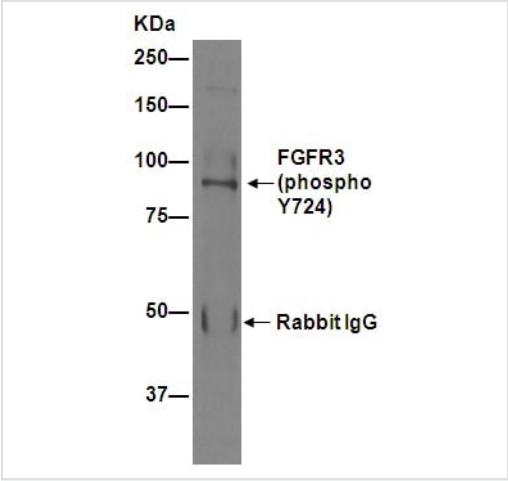


Immunoprecipitation - Anti-FGFR3 (phospho Y724) antibody [EPR2281(3)] (ab155960)

ab155960 (unpurified) at 1/15 immunoprecipitating FGFR3 (phospho Y724) in MCF7 cells treated with pervanadate. For western blotting, a peroxidase-conjugated goat anti-rabbit IgG (H+L) was used as the secondary antibody (1/1000).

Blocking buffer and concentration: 5% NFDM/TBST.

Diluting buffer and concentration: 5% NFDM /TBST.

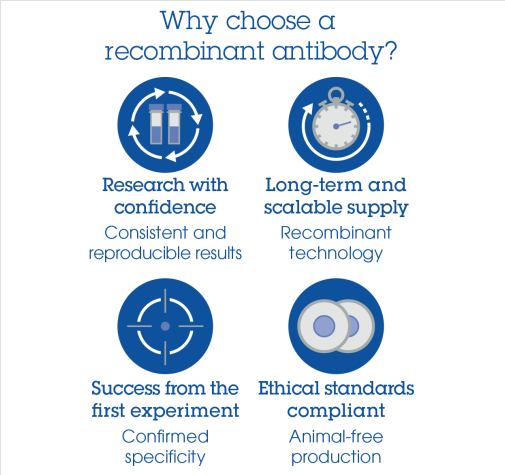


Immunoprecipitation - Anti-FGFR3 (phospho Y724) antibody [EPR2281(3)] (ab155960)

ab155960 (purified) at 1/50 immunoprecipitating FGFR3 (phospho Y724) in MCF7 cells treated with pervanadate. For western blotting, a peroxidase-conjugated goat anti-rabbit IgG (H+L) was used as the secondary antibody (1/1000).

Blocking buffer and concentration: 5% NFDM/TBST.

Diluting buffer and concentration: 5% NFDM /TBST.



Anti-FGFR3 (phospho Y724) antibody [EPR2281(3)] (ab155960)

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