abcam

Product datasheet

Human FGFR3 ELISA Kit ab214027

重组 SimpleStep ELISA

6 图像

概述

产品名称 人FGFR3 ELISA试剂盒

检测方法 Colorimetric

精确度

批次内

样品	n	Mean	SD	CV%
Overall	3			3%

批次间

样品	n	Mean	SD	CV%
Overall	5			7%

样品类型 Cell culture extracts, Tissue Extracts

检测类型 Sandwich (quantitative)

灵敏度 61 pg/ml

范围 0.312 ng/ml - 20 ng/ml

回收率

特定样本回收率

样品类型	平均%	范围
Cell culture extracts	100	80% - 115%

检测时间 1h 30m

实验步骤 One step assay

种属反应性 **与反应:** Human

产品概述 Human FGFR3 ELISA Kit (ab214027) is a single-wash 90 min sandwich ELISA designed for the quantitative measurement of FGFR3 protein in cell culture extracts and tissue extracts. It uses our proprietary SimpleStep ELISA® technology. Quantitate Human FGFR3 with 61 pg/ml sensitivity.

SimpleStep ELISA® technology employs capture antibodies conjugated to an affinity tag that is recognized by the monoclonal antibody used to coat our SimpleStep ELISA® plates. This

approach to sandwich ELISA allows the formation of the antibody-analyte sandwich complex in a single step, significantly reducing assay time. See the SimpleStep ELISA® protocol summary in the image section for further details. Our SimpleStep ELISA® technology provides several benefits:

- Single-wash protocol reduces assay time to 90 minutes or less
- High sensitivity, specificity and reproducibility from superior antibodies
- Fully validated in biological samples
- 96-wells plate breakable into 12 x 8 wells strips

A 384-well SimpleStep ELISA® microplate (**ab203359**) is available to use as an alternative to the 96-well microplate provided with SimpleStep ELISA® kits.

说明

FGFR3 is a tyrosine-protein kinase that acts as cell-surface receptor for fibroblast growth factors and plays an essential role in the regulation of cell proliferation, differentiation and apoptosis. FGFR3 plays an essential role in the regulation of chondrocyte differentiation, proliferation and apoptosis, and is required for normal skeleton development. FGFR3 regulates both osteogenesis and postnatal bone mineralization by osteoblasts. FGFR3 promotes apoptosis in chondrocytes, but can also promote cancer cell proliferation. FGFR3 is required for normal development of the inner ear. FGFR3 interacts in vitro with FGF1, FGF2, FGF4, FGF6; FGF8, FGF9, FGF10, FGF17, FGF18, FGF19, FGF20 and FGF23. FGFR3 interacts with KLB. The affinity for fibroblast growth factors (FGFs) is increased by heparan sulfate glycosaminoglycans that function as co-receptors. Likewise, KLB increases the affinity for FGF19 and FGF21. FGFR3 interacts with PIK3R1, PLCG1, SOCS1 and SOCS3. FGFR3 isoform 3 forms disulfide-linked dimers. Ligand binding to FGFR3 leads to FGFR3 dimerization and to the activation of several signalling cascades. FGFR3 phosphorylates PLCG1, CBL and FRS2. 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. FGFR3 plays a role in the regulation of vitamin D metabolism. Mutations that lead to constitutive kinase activation or impair normal FGFR3 maturation, internalization and degradation lead to aberrant signaling. Over-expressed or constitutively activated FGFR3 promotes activation of PTPN11/SHP2, STAT1, STAT5A and STAT5B. Secreted isoform 3 of FGFR3 retains its capacity to bind FGF1 and FGF2 and hence may interfere with FGF signalling.

Abcam has not and does not intend to apply for the REACH Authorisation of customers' uses of products that contain European Authorisation list (Annex XIV) substances.
It is the responsibility of our customers to check the necessity of application of REACH Authorisation, and any other relevant authorisations, for their intended uses.

平台

Pre-coated microplate (12 x 8 well strips)

性能

存放说明

Store at +4°C. Please refer to protocols.

组件	1 x 96 tests
10X Human FGFR3 Capture Antibody	1 x 600µl

组件	1 x 96 tests
10X Human FGFR3 Detector Antibody	1 x 600µl
10X Wash Buffer PT (ab206977)	1 x 20ml
50X Cell Extraction Enhancer Solution (ab193971)	1 x 1ml
5X Cell Extraction Buffer PTR (ab193970)	1 x 10ml
Antibody Diluent 5BI	1 x 6ml
Denaturant	1 x 500µl
Human FGFR3 Lyophilized Recombinant Protein	2 vials
Plate Seals	1 unit
Sample Diluent NS (ab193972)	1 x 12ml
SimpleStep Pre-Coated 96-Well Microplate (ab206978)	1 unit
Stop Solution	1 x 12ml
TMB Development Solution	1 x 12ml

功能

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

组织特异性

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.

疾病相关

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

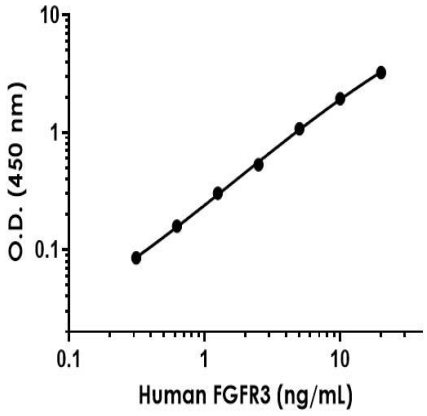
dominant disease and is characterized by disproportionate short stature. It resembles achondroplasia, but with a less severe phenotype.
Defects in FGFR3 are a cause of susceptibility to bladder cancer (BLC) [MIM:109800]. A malignancy originating in tissues of the urinary bladder. It often presents with 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.

序列相似性 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.

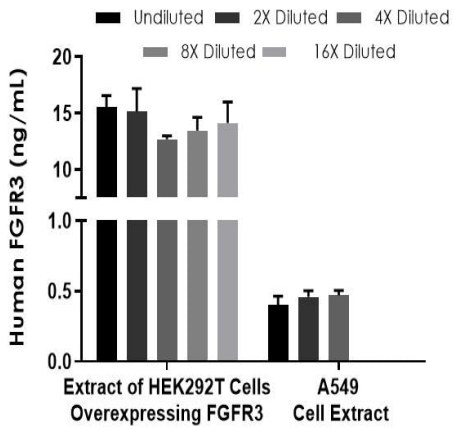
细胞定位 Membrane.

图片



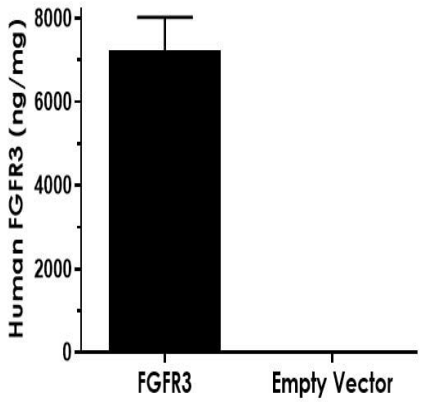
Example of human FGFR3 standard curve.

Background-subtracted data values (mean +/- SD) are graphed.



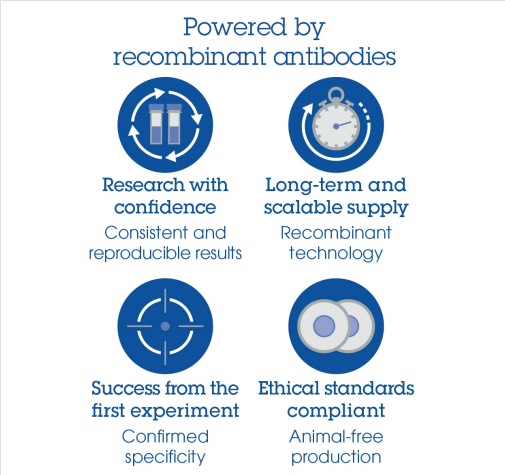
Interpolated concentrations of FGFR3 in extract of HEK293T cells overexpressing FGFR3 samples and native FGFR3 in A549 cell extract samples.

HEK293T cells overexpressing FGFR3 samples based on a 2 µg/mL extract load and native FGFR3 in A549 cell extract samples based on a 300 µg/mL extract load. The concentrations of FGFR3 were measured in duplicates, interpolated from the FGFR3 standard curves and corrected for sample dilution. The interpolated dilution factor corrected values are plotted (mean +/- SD, n=2). Values for A549 cell extract at 8X and 16X diluted had O.D. values below the O.D. values of the MDD. The mean FGFR3 concentration was determined to be 14.2 ng/mL in extract of HEK293T cells overexpressing FGFR3 and 0.44 ng/mL in A549 cell extract.



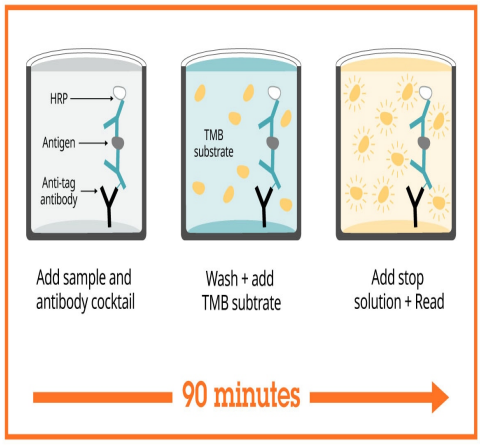
Interpolated concentrations of FGFR3 in extracts of HEK293T cells overexpressing human FGFR3 and HEK293T cells transfected with the associated empty vector.

The concentrations of FGFR3 were measured in three different dilutions in duplicate and interpolated from the FGFR3 standard curve and corrected for sample dilution. The interpolated dilution factor corrected values are plotted in ng FGFR3 per mg of extract (mean +/- SD, n=3). FGFR3 concentration was determined to be 7234 ng/mg in extract of HEK293T cells overexpressing FGFR3 and 56.9 ng/mg in the empty vector.



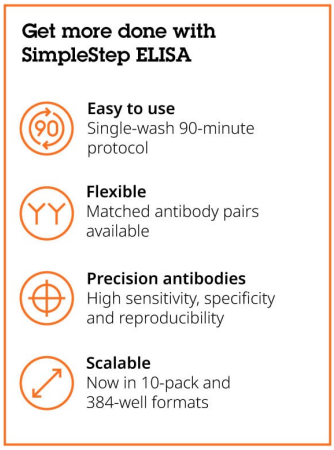
Sandwich ELISA - Human FGFR3 ELISA Kit (ab214027)

To learn more about the advantages of recombinant antibodies see **here**.



Sandwich ELISA - Human FGFR3 ELISA Kit (ab214027)

SimpleStep ELISA technology allows the formation of the antibody-antigen complex in one single step, reducing assay time to 90 minutes. Add samples or standards and antibody mix to wells all at once, incubate, wash, and add your final substrate. See protocol for a detailed step-by-step guide.



Sandwich ELISA - Human FGFR3 ELISA Kit (ab214027)

To learn more about the advantages of SimpleStep ELISA® kits see **here**.

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