abcam

Product datasheet

Anti-FGFR2 antibody ab10647

3 References

Overview

Product name Anti-FGFR2 antibody

Description Rabbit polyclonal to FGFR2

Host species Rabbit

Tested applications Suitable for: IHC-P, IP, WB

Species reactivity Reacts with: Human

Predicted to work with: Mouse, Rat

Immunogen Synthetic peptide:

KLPQYPHINGSVKT

conjugated to KLH by a Glutaraldehyde linker, corresponding to amino acids 809-821 of the

cytoplasmic region of Human FGFR2.

Run BLAST with
Run BLAST with

General notes

The Life Science industry has been in the grips of a reproducibility crisis for a number of years.

Abcam is leading the way in addressing this with our range of recombinant monoclonal antibodies and knockout edited cell lines for gold-standard validation. Please check that this product meets

your needs before purchasing.

If you have any questions, special requirements or concerns, please send us an inquiry and/or contact our Support team ahead of purchase. Recommended alternatives for this product can be

found below, along with publications, customer reviews and Q&As

Properties

Form Liquid

Storage instructions Shipped at 4°C. Upon delivery aliquot and store at -20°C or -80°C. Avoid repeated freeze / thaw

cycles.

Storage buffer pH: 7.40

Preservative: 0.097% Sodium azide Constituents: 0.0268% PBS, 1% BSA

Purity Immunogen affinity purified

Purification notes The antibody is affinity-purified using the immunizing peptide immobilized on agarose.

Clonality Polyclonal

1

Isotype IgG

Applications

The Abpromise guarantee

Our Abpromise guarantee covers the use of ab10647 in the following tested applications.

The application notes include recommended starting dilutions; optimal dilutions/concentrations should be determined by the end user.

Application	Abreviews	Notes
IHC-P		
IP		
WB		

Application notes

IHC-P: Use at an assay dependent dilution, this concentration is determined by indirect immunoperoxidase staining of trypsin or protease-digested human and animal tissue sections. IP: 1/2000. This concentration is determined by immunoprecipitation using a whole lysate of transfected cells expressing recombinant human FGFR2.

WB: 1/2000. This concentration is determined by immunoblotting using a whole extract of transfected cells expressing recombinant human FGFR2. Predicted molecular weight: 110 kDa.

Not tested in other applications.

Optimal dilutions/concentrations should be determined by the end user.

Target

Function

Involvement in disease

Receptor for acidic and basic fibroblast growth factors.

Defects in FGFR2 are the cause of Crouzon syndrome (CS) [MIM:123500]; also called craniofacial dysostosis type I (CFD1). CS is an autosomal dominant syndrome characterized by craniosynostosis (premature fusion of the skull sutures), hypertelorism, exophthalmos and external strabismus, parrot-beaked nose, short upper lip, hypoplastic maxilla, and a relative mandibular prognathism.

Defects in FGFR2 are a cause of Jackson-Weiss syndrome (JWS) [MIM:123150]. JWS is an autosomal dominant craniosynostosis syndrome characterized by craniofacial abnormalities and abnormality of the feet: broad great toes with medial deviation and tarsal-metatarsal coalescence. Defects in FGFR2 are a cause of Apert syndrome (APRS) [MIM:101200]; also known as acrocephalosyndactyly type 1 (ACS1). APRS is a syndrome characterized by facio-cranio-synostosis, osseous and membranous syndactyly of the four extremities, and midface hypoplasia. The craniosynostosis is bicoronal and results in acrocephaly of brachysphenocephalic type. Syndactyly of the fingers and toes may be total (mitten hands and sock feet) or partial affecting the second, third, and fourth digits. Intellectual deficit is frequent and often severe, usually 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 of the skull sutures) with deviation and enlargement of the thumbs and great toes, brachymesophalangy, with phalangeal ankylosis and a varying degree of soft tissue syndactyly. Three subtypes of Pfeiffer syndrome have been described: mild autosomal dominant form (type 1); cloverleaf skull, elbow ankylosis, early death, sporadic (type 2); craniosynostosis, early

demise, sporadic (type 3).

Defects in FGFR2 are the cause of Beare-Stevenson cutis gyrata syndrome (BSCGS)

[MIM:123790]. BSCGS is an autosomal dominant condition is characterized by the furrowed skin disorder of cutis gyrata, acanthosis nigricans, craniosynostosis, craniofacial dysmorphism, digital anomalies, umbilical and anogenital abnormalities and early death.

Defects in FGFR2 are the cause of familial scaphocephaly syndrome (FSPC) [MIM:609579]; also known as scaphocephaly with maxillary retrusion and mental retardation. FSPC is an autosomal dominant craniosynostosis syndrome characterized by scaphocephaly, macrocephaly, hypertelorism, maxillary retrusion, and mild intellectual disability. Scaphocephaly is the most common of the craniosynostosis conditions and is characterized by a long, narrow head. It is due to premature fusion of the sagittal suture or from external deformation.

Defects in FGFR2 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 FGFR2 are the cause of Antley-Bixler syndrome (ABS) [MIM:207410]. ABS is a multiple congenital anomaly syndrome characterized by craniosynostosis, radiohumeral synostosis, midface hypoplasia, malformed ears, arachnodactyly and multiple joint contractures. ABS is a heterogeneous disorder and occurs with and without abnormal genitalia in both sexes.

Sequence similarities

Belongs to the protein kinase superfamily. Tyr protein kinase family. Fibroblast growth factor

receptor subfamily.

Contains 3 lg-like C2-type (immunoglobulin-like) domains.

Contains 1 protein kinase domain.

Cellular localization

Secreted and Cell membrane.

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