## Overview

<table>
<thead>
<tr>
<th>Product name</th>
<th>Anti-FGFR2 antibody</th>
</tr>
</thead>
<tbody>
<tr>
<td>Description</td>
<td>Mouse monoclonal to FGFR2</td>
</tr>
<tr>
<td>Host species</td>
<td>Mouse</td>
</tr>
<tr>
<td>Tested applications</td>
<td>Suitable for: WB, IHC-P, ICC/IF, Flow Cyt</td>
</tr>
<tr>
<td>Species reactivity</td>
<td>Reacts with: Rat, Human</td>
</tr>
<tr>
<td>Immunogen</td>
<td>Recombinant fragment, corresponding to amino acids 621-724 of Human FGFR2</td>
</tr>
<tr>
<td>Positive control</td>
<td>Purchase matching WB positive control: Recombinant human FGFR2 protein</td>
</tr>
</tbody>
</table>

### General notes

This product was changed from ascites to tissue culture supernatant on 13th Feb 2019. Please note that the dilutions may need to be adjusted accordingly. If you have any questions, please do not hesitate to contact our scientific support team.

## Properties

<table>
<thead>
<tr>
<th>Form</th>
<th>Liquid</th>
</tr>
</thead>
<tbody>
<tr>
<td>Storage instructions</td>
<td>Shipped at 4°C. Upon delivery aliquot and store at -20°C or -80°C. Avoid repeated freeze / thaw cycles.</td>
</tr>
<tr>
<td>Storage buffer</td>
<td>pH: 7.20</td>
</tr>
<tr>
<td></td>
<td>Constituent: PBS</td>
</tr>
<tr>
<td>Purity</td>
<td>Tissue culture supernatant</td>
</tr>
<tr>
<td>Clonality</td>
<td>Monoclonal</td>
</tr>
<tr>
<td>Isotype</td>
<td>IgG2b</td>
</tr>
<tr>
<td>Light chain type</td>
<td>kappa</td>
</tr>
</tbody>
</table>

## Applications

Our Abpromise guarantee covers the use of ab58201 in the following tested applications.

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

Receptor for acidic and basic fibroblast growth factors.

Involvement in disease

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-craniosynostosis, 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 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).

<table>
<thead>
<tr>
<th>Application</th>
<th>Abreviews</th>
<th>Notes</th>
</tr>
</thead>
<tbody>
<tr>
<td>WB</td>
<td>⭐⭐⭐⭐⭐</td>
<td>Use at an assay dependent concentration. This antibody has only been tested in WB against the recombinant fragment used as immunogen. We have no data on the detection of endogenous protein.</td>
</tr>
<tr>
<td>IHC-P</td>
<td>⭐⭐⭐⭐⭐</td>
<td>Use at an assay dependent concentration.</td>
</tr>
<tr>
<td>ICC/IF</td>
<td>⭐⭐⭐⭐⭐</td>
<td>Use at an assay dependent concentration.</td>
</tr>
<tr>
<td>Flow Cyt</td>
<td></td>
<td>Use at an assay dependent concentration. ab170192 - Mouse monoclonal IgG2b, is suitable for use as an isotype control with this antibody.</td>
</tr>
</tbody>
</table>

Target

Function

Receptor for acidic and basic fibroblast growth factors.

Involvement in disease

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.

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Defects in FGFR2 are a cause of lacrimo-auriculo-dento-digital syndrome (LADDS).
LADDs is a form of ectodermal dysplasia, also known as Levy-Hollister syndrome. LADDs is 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 segment 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 Ig-like C2-type (immunoglobulin-like) domains.
Contains 1 protein kinase domain.

Cellular localization
Secreted and Cell membrane.

Images

FGFR2 antibody (ab58201) used in immunohistochemistry at 5ug/ml on formalin fixed and paraffin embedded human stomach carcinoma tissue.
This image was generated using the ascites version of the product.

Western blot against tagged recombinant protein immunogen using ab58201 FGFR2 antibody at 1ug/ml. Predicted band size of immunogen is 38 kDa
This image was generated using the ascites version of the product.
Immunocytochemistry/Immunofluorescence - Anti-FGFR2 antibody (ab58201)
This image is courtesy of an anonymous Abreview.

Staining FGFR2 in Human glioblastoma cell line D54MG by Immunocytochemistry/Immunofluorescence. Cells were fixed in paraformaldehyde and permeabilized in 0.1% Triton X-100 prior to blocking in 0.5% BSA for 30 minutes at room temperature. The primary antibody was diluted 1/50 and incubated with the sample for 16 hours at 4°C. The secondary antibody was TRITC-conjugated Goat anti-Mouse polyclonal, diluted 1/300.

This image was generated using the ascites version of the product.

Flow Cytometry - Anti-FGFR2 antibody (ab58201)
Overlay histogram showing HeLa cells stained with ab58201 (red line). The cells were fixed with 80% methanol (5 min) and then permeabilized with 0.1% PBS-Tween for 20 min. The cells were then incubated in 1x PBS / 10% normal goat serum / 0.3M glycine to block non-specific protein-protein interactions followed by the antibody (ab58201, 2µg/1x10^6 cells) for 30 min at 22°C. The secondary antibody used was DyLight® 488 goat anti-mouse IgG (H+L) (ab96879) at 1/500 dilution for 30 min at 22°C. Isotype control antibody (black line) was mouse IgG2b [PLPV219] (ab91366, 2µg/1x10^6 cells) used under the same conditions. Acquisition of >5,000 events was performed.

This image was generated using the ascites version of the product.

Immunohistochemistry (Formalin/PFA-fixed paraffin-embedded sections) - Anti-FGFR2 antibody (ab58201)
Immunohistochemical analysis of Human gastric adenocarcinoma, staining FGFR2 with ab58201 at 0.2 µg/ml.

This image was generated using the ascites version of the product.
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