Overview

Product name: Anti-FGFR2 antibody [SP273] - N-terminal
Description: Rabbit monoclonal [SP273] to FGFR2 - N-terminal
Host species: Rabbit
Tested applications: Suitable for: Flow Cyt, IHC-P
Species reactivity: Reacts with: Human
Predicted to work with: Mouse
Immunogen: Synthetic peptide within Human FGFR2 aa 1-100 (N terminal). The exact sequence is proprietary.
Database link: P21802

Properties

Form: Liquid
Storage buffer: pH: 7.6
Preservative: 0.1% Sodium azide
 Constituents: PBS, 1% BSA
Purity: Protein A/G purified
Purification notes: Purified from TCS by protein A/G.
Clonality: Monoclonal
Clone number: SP273
Isotype: IgG

Applications

Our Abpromise guarantee covers the use of ab227683 in the following tested applications.
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-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 Ig-like C2-type (immunoglobulin-like) domains.
Contains 1 protein kinase domain.

**Cellular localization**
Secreted and Cell membrane.

**Images**

Flow Cytometry analysis of Kato III (human gastric carcinoma cell
line) cells labeling FGFR2 with ab227683 at 1/400 dilution (green)
compared to a Rabbit IgG negative control (blue).

Formalin-fixed, paraffin-embedded human colon
adenocarcinoma tissue stained for FGFR2 using ab227683 at
1/100 dilution in immunohistochemical analysis.
Formalin-fixed, paraffin-embedded human bladder transitional cell carcinoma tissue stained for FGFR2 using ab227683 at 1/100 dilution in immunohistochemical analysis.

Formalin-fixed, paraffin-embedded human breast ductal carcinoma tissue stained for FGFR2 using ab227683 at 1/100 dilution in immunohistochemical analysis.
Formalin-fixed, paraffin-embedded human cervical squamous cell carcinoma tissue stained for FGFR2 using ab227683 at 1/100 dilution in immunohistochemical analysis.

Formalin-fixed, paraffin-embedded human hepatocellular carcinoma tissue stained for FGFR2 using ab227683 at 1/100 dilution in immunohistochemical analysis.
Formalin-fixed, paraffin-embedded human stomach adenocarcinoma tissue stained for FGFR2 using ab227683 at 1/100 dilution in immunohistochemical analysis.

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