

Summary

Production Name	GDF-5 Rabbit Polyclonal Antibody
Description	Rabbit Polyclonal Antibody
Host	Rabbit
Application	WB,IHC,ELISA
Reactivity	Human,Mouse,Rat

Performance

Conjugation	Unconjugated
Modification	Unmodified
Isotype	IgG
Clonality	Polyclonal
Form	Liquid
Storage	Store at 4°C short term. Aliquot and store at -20°C long term. Avoid freeze/thaw cycles.
Buffer	Liquid in PBS containing 50% glycerol, 0.5% BSA and 0.02% New type preservative N.
Purification	Affinity purification

Immunogen

Gene Name	GDF5
Alternative Names	GDF5; CDMP1; Growth/differentiation factor 5; GDF-5; Cartilage-derived morphogenetic protein 1; CDMP-1; Radotermin
Gene ID	8200.0
SwissProt ID	P43026.The antiserum was produced against synthesized peptide derived from the Internal region of human GDF5. AA range:361-410

Application

Dilution Ratio	WB 1:500 - 1:2000. IHC: 1:100-1:300. ELISA: 1:20000..
Molecular Weight	55kD

Background

This gene encodes a secreted ligand of the TGF-beta (transforming growth factor-beta) superfamily of proteins. Ligands of this family bind various TGF-beta receptors leading to recruitment and activation of SMAD family transcription factors that regulate gene expression. The encoded preproprotein is proteolytically processed to generate each subunit of the disulfide-linked homodimer. This protein regulates the development of numerous tissue and cell types, including cartilage, joints, brown fat, teeth, and the growth of neuronal axons and dendrites. Mutations in this gene are associated with acromesomelic dysplasia, brachydactyly, chondrodysplasia, multiple synostoses syndrome, proximal symphalangism, and susceptibility to osteoarthritis. [provided by RefSeq, Aug 2016],disease:Defects in GDF5 are a cause of brachydactyly type A2 (BDA2) [MIM:112600]. Brachydactylies (BDs) are a group of inherited malformations characterized by shortening of the digits due to abnormal development of the phalanges and/or the metacarpals. They have been classified on an anatomic and genetic basis into five groups, A to E, including three subgroups (A1 to A3) that usually manifest as autosomal dominant traits.,disease:Defects in GDF5 are a cause of symphalangism proximal syndrome (SYM1) [MIM:185800]. SYM1 is characterized by the hereditary absence of the proximal interphalangeal (PIP) joints (Cushing symphalangism). Severity of PIP joint involvement diminishes towards the radial side. Distal interphalangeal joints are less frequently involved and metacarpophalangeal joints are rarely affected whereas carpal bone malformation and fusion are common. In the lower extremities, tarsal bone coalition is common. Conductive hearing loss is seen and is due to fusion of the stapes to the petrous part of the temporal bone.,disease:Defects in GDF5 are the cause of acromesomelic chondrodysplasia Grebe type (AMDG) [MIM:200700]. Acromesomelic chondrodysplasias are rare hereditary skeletal disorders characterized by short stature, very short limbs, and hand/foot malformations. The severity of limb abnormalities increases from proximal to distal with profoundly affected hands and feet showing brachydactyly and/or rudimentary fingers (knob-like fingers). AMDG is an autosomal recessive form characterized by normal axial skeletons and missing or fused skeletal elements within the hands and feet.,disease:Defects in GDF5 are the cause of acromesomelic chondrodysplasia Hunter-Thompson type (AMDH) [MIM:201250]. AMDH is an autosomal recessive form of dwarfism. Patients have limb abnormalities, with the middle and distal segments being most affected and the lower limbs more affected than the upper. AMDH is characterized by normal axial skeletons and missing or fused skeletal elements within the hands and feet.,disease:Defects in GDF5 are the cause of brachydactyly type C (BDC) [MIM:113100]. BDC is an autosomal dominant disorder characterized by an abnormal shortness of the fingers and toes.,disease:Defects in GDF5 are the cause of Du Pan syndrome [MIM:228900]; also known as fibular hypoplasia and complex brachydactyly. Du Pan syndrome is a rare autosomal recessive condition characterized by absence of the fibulae and severe acromesomelic limb shortening with small, non-functional toes. Although milder, the phenotype resembles the autosomal recessive Hunter-Thompson [MIM:201250] and Grebe types [MIM:200700] of acromesomelic chondrodysplasia.,disease:Defects in GDF5 are the cause of multiple synostoses syndrome 2 (SYNS2) [MIM:610017]. Multiple synostoses syndrome is an autosomal dominant condition characterized by progressive joint fusions of the fingers, wrists, ankles and cervical spine, characteristic facies and progressive conductive deafness.,disease:Genetic variations in GDF5 are associated with osteoarthritis susceptibility type 5 (OS5) [MIM:612400]. Osteoarthritis is the most prevalent form of arthritis and a common cause of disability.,function:Could be involved in bone formation.,online information:GDF5 entry,similarity:Belongs to the TGF-beta family.,subunit:Homodimer; disulfide-linked.,tissue specificity:Predominantly

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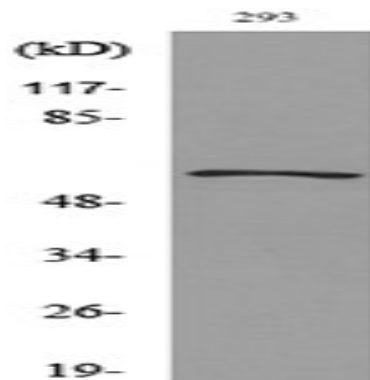


expressed in long bones during embryonic development.,

Research Area

Cytokine-cytokine receptor interaction;TGF-beta;

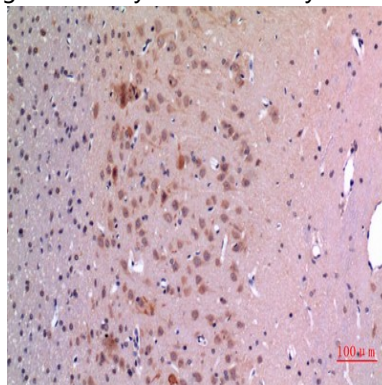
Image Data



Western blot analysis of lysate from 293 cells, using GDF5 Antibody.

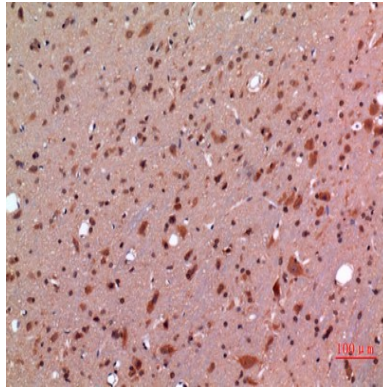


Western Blot analysis of 293 cells using GDF-5 Polyclonal Antibody.. Secondary antibody was diluted at 1:20000

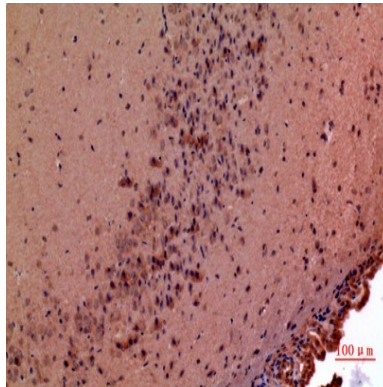


Immunohistochemical analysis of paraffin-embedded rat-brain, antibody was diluted at 1:100

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Immunohistochemical analysis of paraffin-embedded rat-brain, antibody was diluted at 1:100



Immunohistochemical analysis of paraffin-embedded mouse-brain, antibody was diluted at 1:100

Note

For research use only.