


Anti-Connexin 43 / GJA1 antibody ab47441

1 Image

Overview

Product name	Anti-Connexin 43 / GJA1 antibody
Description	Rabbit polyclonal to Connexin 43 / GJA1
Host species	Rabbit
Tested applications	Suitable for: WB
Species reactivity	Reacts with: Human Predicted to work with: Mouse, Rat 
Immunogen	The antiserum was produced against synthesized phosphopeptide derived from human Connexin 43 around the phosphorylation site of serine 367 (R-A-S ^P -S-R).
Positive control	Extracts from K562 cells.
General notes	<p>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.</p> <p>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</p>

Properties

Form	Liquid
Storage instructions	Shipped at 4°C. Upon delivery aliquot and store at -20°C. Avoid freeze / thaw cycles.
Storage buffer	pH: 7.40 Preservative: 0.02% Sodium azide Constituents: PBS, 50% Glycerol (glycerin, glycerine), 0.87% Sodium chloride
	Without Mg ⁺² and Ca ⁺²
Purity	Immunogen affinity purified
Purification notes	The antibody was affinity purified from rabbit antiserum by affinity chromatography using epitope specific immunogen.
Clonality	Polyclonal

Isotype

IgG

Applications

The Abpromise guarantee

Our **Abpromise guarantee** covers the use of ab47441 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
WB		1/500 - 1/1000. Detects a band of approximately 43 kDa (predicted molecular weight: 43 kDa).

Target

Function

One gap junction consists of a cluster of closely packed pairs of transmembrane channels, the connexons, through which materials of low MW diffuse from one cell to a neighboring cell. May play a critical role in the physiology of hearing by participating in the recycling of potassium to the cochlear endolymph.

Tissue specificity

Expressed in the heart and fetal cochlea.

Involvement in disease

Defects in GJA1 are the cause of autosomal dominant oculodentodigital dysplasia (ODDD) [MIM:164200]; also known as oculodentoosseous dysplasia. ODDD is a highly penetrant syndrome presenting with craniofacial (ocular, nasal, dental) and limb dysmorphisms, spastic paraplegia, and neurodegeneration. Craniofacial anomalies typically include a thin nose with hypoplastic alae nasi, small anteverted nares, prominent columnella, and microcephaly. Brittle nails and hair abnormalities of hypotrichosis and slow growth are present. Ocular defects include microphthalmia, microcornea, cataracts, glaucoma, and optic atrophy. Syndactyly type 3 and conductive deafness can occur in some cases. Cardiac abnormalities are observed in rare instances.

Defects in GJA1 are the cause of autosomal recessive oculodentodigital dysplasia (ODDD autosomal recessive) [MIM:257850].

Defects in GJA1 may be the cause of syndactyly type 3 (SDTY3) [MIM:186100]. Syndactyly is an autosomal dominant trait and is the most common congenital anomaly of the hand or foot. It is marked by persistence of the webbing between adjacent digits, so they are more or less completely attached. In this type there is usually complete and bilateral syndactyly between the fourth and fifth fingers. Usually it is soft tissue syndactyly but occasionally the distal phalanges are fused. The fifth finger is short with absent or rudimentary middle phalanx. The feet are not affected.

Defects in GJA1 are a cause of hypoplastic left heart syndrome (HLHS) [MIM:241550]. HLHS refers to the abnormal development of the left-sided cardiac structures, resulting in obstruction to blood flow from the left ventricular outflow tract. In addition, the syndrome includes underdevelopment of the left ventricle, aorta, and aortic arch, as well as mitral atresia or stenosis.

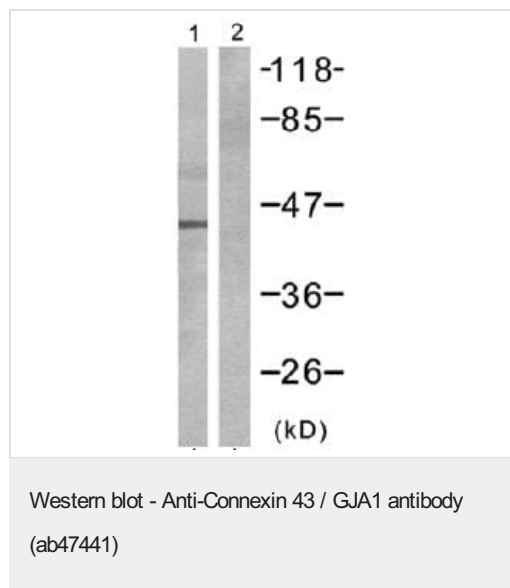
Defects in GJA1 are a cause of Hallermann-Streiff syndrome (HSS) [MIM:234100]. HSS is a disorder characterized by a typical skull shape (brachycephaly with frontal bossing), hypotrichosis, microphthalmia, cataracts, beaked nose, micrognathia, skin atrophy, dental anomalies and proportionate short stature. Mental retardation is present in a minority of cases.

Sequence similarities

Belongs to the connexin family. Alpha-type (group II) subfamily.

Cellular localization

Cell membrane. Cell junction > gap junction.



All lanes : Anti-Connexin 43 / GJA1 antibody (ab47441) at 1/500 dilution

Lane 1 : Extracts from K562 cells, treated with PMA (200ng/ml, 10min). No peptide.

Lane 2 : Extracts from K562 cells, treated with PMA (200ng/ml, 10min). Synthetic peptide present.

Predicted band size: 43 kDa

Observed band size: 43 kDa

Please note: All products are "FOR RESEARCH USE ONLY. NOT FOR USE IN DIAGNOSTIC PROCEDURES"

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