# abcam

## Product datasheet

## Anti-Connexin 43 / GJA1 antibody ab87645

## ★★★★★ 2 Abreviews 10 References 1 Image

#### Overview

Product name Anti-Connexin 43 / GJA1 antibody

**Description** Goat polyclonal to Connexin 43 / GJA1

Host species Goat

Tested applications Suitable for: WB Species reactivity Reacts with: Rat

Predicted to work with: Rabbit, Guinea pig, Dog, Pig, Cynomolgus monkey

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**Immunogen** Synthetic peptide. This information is proprietary to Abcam and/or its suppliers.

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. Avoid freeze / thaw cycles.

**Storage buffer** pH: 7.30

Preservative: 0.02% Sodium azide

Constituents: 0.5% BSA, Tris buffered saline

Purity Immunogen affinity purified

**Purification notes** ab87645 is purified from goat serum by ammonium sulphate precipitation followed by antigen

affinity chromatography using the immunizing peptide.

**Clonality** Polyclonal

**Isotype** IgG

## **Applications**

1

#### The Abpromise guarantee

Our **Abpromise guarantee** covers the use of ab87645 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		Use a concentration of 0.3 - 1 µg/ml. Predicted molecular weight: 43 kDa.  1 hour primary incubation is recommended for this product.

#### **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

Involvement in disease

Expressed in the heart and fetal cochlea.

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 tipically 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.

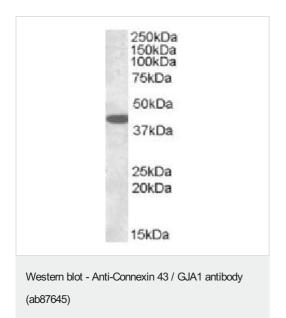
## Sequence similarities

#### **Cellular localization**

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

Cell membrane. Cell junction > gap junction.

#### **Images**



Anti-Connexin 43 / GJA1 antibody (ab87645) at 0.3  $\mu$ g/ml + rat brain lysate (in RIPA buffer) at 35  $\mu$ g

Developed using the ECL technique.

**Predicted band size:** 43 kDa **Observed band size:** 43 kDa

Primary incubation was 1 hour.

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

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- Response to your inquiry within 24 hours
- We provide support in Chinese, English, French, German, Japanese and Spanish
- Extensive multi-media technical resources to help you
- We investigate all quality concerns to ensure our products perform to the highest standards

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