# abcam

# Product datasheet

# Anti-Connexin 43 / GJA1 antibody [Connexin 43] ab78055

## 9 References

#### Overview

Product name Anti-Connexin 43 / GJA1 antibody [Connexin 43]

**Description** Mouse monoclonal [Connexin 43] to Connexin 43 / GJA1

Host species Mouse

**Tested applications** Suitable for: IHC-Fr, ICC/IF, WB

Species reactivity Reacts with: Mouse

**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 or -80°C. Avoid repeated freeze / thaw

cycles.

Storage buffer Preservative: 0.02% Sodium azide

Constituent: 99.98% PBS

Purity Protein A/G purified

Clonality Monoclonal
Clone number Connexin 43

Myeloma P3x63-Ag8.653

lsotype lgG1

# **Applications**

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#### The Abpromise guarantee

Our Abpromise guarantee covers the use of ab78055 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
IHC-Fr		Use at an assay dependent concentration.
ICC/IF		Use at an assay dependent concentration.
WB		Use at an assay dependent concentration.

#### **Target**

#### **Function**

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.

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

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.

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

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- · Replacement or refund for products not performing as stated on the datasheet
- Valid for 12 months from date of delivery
- · 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

If the product does not perform as described on this datasheet, we will offer a refund or replacement. For full details of the Abpromise, please visit <a href="https://www.abcam.com/abpromise">https://www.abcam.com/abpromise</a> or contact our technical team.

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