

DESCRIPTION

Species Reactivity	Human/Mouse/Rat
Specificity	Detects human, mouse, and rat DGCR2 in Western blots. Detects human DGCR2 in direct ELISAs.
Source	Monoclonal Mouse IgG _{2B} Clone # 1019908
Purification	Protein A or G purified from hybridoma culture supernatant
Immunogen	Chinese Hamster Ovary cell line CHO-derived human DGCR2 Glu22-Ala349 Accession # P98153
Conjugate	Alexa Fluor 594 Excitation Wavelength: 590 nm Emission Wavelength: 617 nm
Formulation	Supplied 0.2mg/ml in 1X PBS with RDF1 and 0.09% Sodium Azide *Contains <0.1% Sodium Azide, which is not hazardous at this concentration according to GHS classifications. Refer to the Safety Data Sheet (SDS) for additional information and handling instructions.

APPLICATIONS

Please Note: Optimal dilutions should be determined by each laboratory for each application. [General Protocols](#) are available in the Technical Information section on our website.

Western Blot Optimal dilution of this antibody should be experimentally determined.

PREPARATION AND STORAGE

Shipping	The product is shipped with polar packs. Upon receipt, store it immediately at the temperature recommended below.
Stability & Storage	Protect from light. Do not freeze. 12 months from date of receipt, 2 to 8 °C as supplied

BACKGROUND

DiGeorge Syndrome Critical Region 2 (DGCR2), also known as IDD, DGS-C, LAN, and SEZ-12, is an adhesion receptor protein located on the long arm of chromosome 22 (1). The DGCR2 gene encodes an integral membrane protein, consisting of an extracellular domain, a single transmembrane region and a cytoplasmic tail (1). The mature extracellular domain (ECD) of DGCR2 contains both a C-type lectin domain and a cysteine-rich region similar to that of the low density lipoprotein receptor (LDLR) (1, 3). The mature ECD of human DGCR2 shares 93% and 92% amino acid sequence identity with mouse and rat, respectively. DGCR2 is expressed during neurodevelopment in human brain tissues (3). Deletion of the 22q11.2 region results in an extremely variable disorder called 22q11.2 deletion syndrome, with a phenotype ranging from very mild symptoms to severe intellectual disability, facial dysmorphism, heart defects, and urogenital abnormalities (4). Recent studies suggest that DGCR2 regulates critical steps of early cortico-genesis possibly through a Reelin-dependent mechanism. Deletion of DGCR2 has a pathogenic impact on cortical formation by reducing protein expression level, and it plays a critical role in vulnerability to schizophrenia (5). Furthermore, expression of DGCR2 together with USP18 gene may serve as a prognostic marker for muscle invasive bladder cancer survival in patients (5).

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