

Synonym

CD19,B4,CVID3,MGC12802

Source

Alexa Fluor 647-Labeled Human CD19 (20-291), His Tag (CD9-HA2H6) is produced via conjugation of AF647 to Human CD19 (20-291), His Tag with a new generation site-specific technology under Star Staining labeling platform. Human CD19 (20-291), His Tag is expressed from human 293 cells (HEK293). It contains AA Pro 20 - Lys 291 (Accession # P15391-1).

Molecular Characterization

CD19(Pro 20 - Lys 291) P15391-1

Poly-his

B

This protein carries a polyhistidine tag at the C-terminus

The protein has a calculated MW of 47.2 kDa.

Conjugate

AF647

Excitation Wavelength: 640 nm

Emission Wavelength: 672 nm

Endotoxin

Less than 1.0 EU per µg by the LAL method.

Purity

>95% as determined by SDS-PAGE.

Formulation

Lyophilized from 0.22 µm filtered solution in PBS, pH7.4 with trehalose as protectant.

Contact us for customized product form or formulation.

Reconstitution

Please see Certificate of Analysis for specific instructions.

For best performance, we strongly recommend you to follow the reconstitution protocol provided in the CoA.

Storage

For long term storage, the product should be stored at lyophilized state at -20°C or lower.

Please protect from light and avoid repeated freeze-thaw cycles.

This product is stable after storage at:

- -20°C to -70°C for 12 months in lyophilized state;
- -70°C for 3 months under sterile conditions after reconstitution.

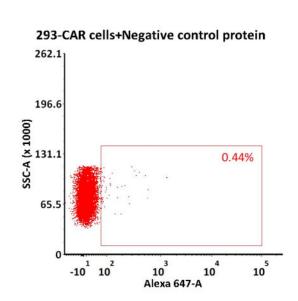
Evaluation of CAR expression

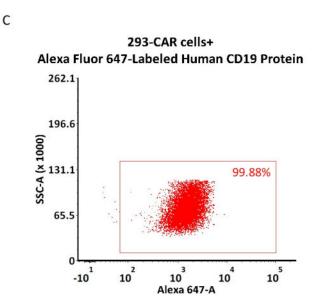
FACS Analysis of Anti-CD19 CAR Expression 293 cells+ Alexa Fluor 647-Labeled Human CD19 Protein 262.1 196.6 SSC-A (x 1000) 0.21% 105

10

Alexa 647-A

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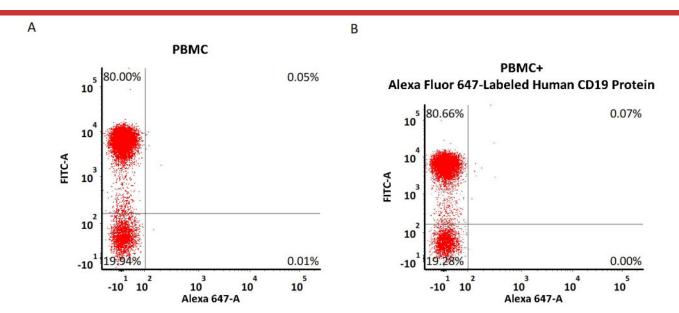
5e5 of 293 CAR cells transfected with anti-CD19-scFv were stained with 100 μL of 3 μg/mL of Alexa Fluor 647-Labeled Human CD19 (20-291), His Tag (Cat. No. CD9-HA2H6) and negative control protein respectively (Fig. C and B), and non-transfected 293 cells were used as a control (Fig. A), Alexa 647 signal was used to evaluate the binding activity (QC tested).

FACS Analysis of Non-specific binding to PBMCs

Alexa Fluor™ 647-Labeled Human CD19 (20-291) Protein, His TagStar Staining

Catalog # CD9-HA2H6





5e5 of PBMCs were stained with Alexa Fluor 647-Labeled Human CD19 (20-291), His Tag (Cat. No. CD9-HA2H6) and anti-CD3 antibody, washed and then analyzed with FACS. FITC signal was used to evaluate the expression of CD3+ T cells in PBMCs, and Alexa 647 signal was used to evaluate the non-specific binding activity to PBMCs (QC tested).

Background

B-lymphocyte antigen CD19 is also known as CD19 (Cluster of Differentiation 19), is a single-pass type I membrane protein which contains two Ig-like C2-type (immunoglobulin-like) domains. CD19 is expressed on follicular dendritic cells and B cells. In fact, it is present on B cells from earliest recognizable B-lineage cells during development to B-cell blasts but is lost on maturation to plasma cells. It primarily acts as a B cell co-receptor in conjunction with CD21 and CD81. Upon activation, the cytoplasmic tail of CD19 becomes phosphorylated, which leads to binding by Src-family kinases and recruitment of PI-3 kinase. As on T cells, several surface molecules form the antigen receptor and form a complex on B lymphocytes. The (almost) B cell-specific CD19 phosphoglycoprotein is one of these molecules. The others are CD21 and CD81. These surface immunoglobulin (sIg)-associated molecules facilitate signal transduction. On living B cells, antimmunoglobulin antibody mimicking exogenous antigen causes CD19 to bind to sIg and internalize with it. The reverse process has not been demonstrated, suggesting that formation of this receptor complex is antigen-induced. This molecular association has been confirmed by chemical studies. Mutations in CD19 are associated with severe immunodeficiency syndromes characterized by diminished antibody production. CD19 has been shown to interact with: CD81, CD82, Complement receptor 2, and VAV2.

Clinical and Translational Updates

Please contact us via <u>TechSupport@acrobiosystems.com</u> if you have any question on this product.