SANTA CRUZ BIOTECHNOLOGY, INC.

CD3 (RM0027-3B19): sc-101442

BACKGROUND

The T cell antigen receptor (TCR) recognizes foreign antigens and translates such recognition events into intracellular signals that elicit a change in the cell from a dormant to an activated state. Much of this signaling process can be attributed to a multisubunit complex of proteins that associates directly with the TCR. This complex has been designated CD3 (cluster of differentiation 3). It is composed of five invariant polypeptide chains that associate to form three dimers: a heterodimer of γ and ε chains (γε), a heterodimer of δ and ε chains (δε) and a homodimer of two ζ chains (ζζ) or a heterodimer of ζ and η chains (ζη). The ζ and η chains are encoded by the same gene but differ in their carboxyl-terminal ends due to an alternative splicing event. The γ, ε and δ chains each contain a single copy of a conserved immunoreceptor tyrosine-based activation motif (ITAM). In contrast, the ζ chain contains three consecutive copies of the same motif. Phosphorylated ITAMs act as docking sites for protein kinases such as ZAP-70 and Syk and are also capable of regulating their kinase activity. The crystal structure of the ZAP-70 SH2 domains bound to the ζ chain ITAMs has been solved.

REFERENCES

1. Exley, M., et al. 1991. Structure, assembly and intracellular transport of the T cell receptor for antigen. Semin. Immunol. 3: 283-297.
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3. Chan, A.C., et al. 1994. The role of protein tyrosine kinases and protein tyrosine phosphatases in cell antigen receptor signal transduction. Semin. Immunol. 12: 555-592.
4. Aoe, T., et al. 1994. Different cytoplasmic structure of the CD3 ζ family dimer modulates the activation signal and function of T cells. Int. Immunol. 6: 1671-1679.
5. Ohno, H., et al. 1994. Targeted disruption of the CD3 η locus causes high lethality in mice: modulation of Oct-1 transcription on the opposite strand. EMBO J. 13: 1157-1165.
6. Neumeister, E.N., et al. 1995. Binding of ZAP-70 to phosphorylated T cell receptor ζ and ε enhances its autophosphorylation and generates specific binding sites for SH2 domain-containing proteins. Mol. Cell. Biol. 15: 3171-3178.
7. Weiss, A. 1995. Zapping tandem SH2 domains. Nature 377: 17-18.

SOURCE

CD3 (RM0027-3B19) is a rat monoclonal antibody raised against CD3 positive T cells of mouse origin.

PRODUCT

Each vial contains 100 µg IgG_{2b} in 1.0 ml of PBS with < 0.1% sodium azide and 0.1% gelatin.

STORAGE

Store at 4° C, **DO NOT FREEZE**. Stable for one year from the date of shipment. Non-hazardous. No MSDS required.

APPLICATIONS

CD3 (RM0027-3B19) is recommended for detection of CD3 of mouse origin by immunoprecipitation [1-2 µg per 100-500 µg of total protein (1 ml of cell lysate)], immunofluorescence (starting dilution 1:50, dilution range 1:50-1:500), immunohistochemistry (including paraffin-embedded sections) (starting dilution 1:50, dilution range 1:50-1:500) and flow cytometry (1 µg per 1 x 10^6 cells).

Suitable for use as control antibody for CD3 siRNA (m): sc-29988, CD3 shRNA Plasmid (m): sc-29988-SH and CD3 shRNA (m) Lentiviral Particles: sc-29988-V.

Molecular Weight of CD3: 25 kDa.

Positive Controls: CTLL-2 cell lysate: sc-2242 or TK-1 whole cell lysate: sc-364798.

SELECT PRODUCT CITATIONS

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2. Bei, Y., et al. 2013. Long-term treatment with fasudil improves bleomycin-induced pulmonary fibrosis and pulmonary hypertension via inhibition of Smad2/3 phosphorylation. Pulm. Pharmacol. Ther. 26: 635-643.
3. Semba, T., et al. 2013. The FLS (fatty liver Shionogi) mouse reveals local expressions of lipocalin-2, CXCL1 and CXCL9 in the liver with non-alcoholic steatohepatitis. BMC Gastroenterol. 13: 120.
4. Bei, Y., et al. 2016. RhoA/Rho-kinase activation promotes lung fibrosis in an animal model of systemic sclerosis. Exp. Lung Res. 42: 44-55.
5. Lian, G., et al. 2018. Glutathione *de novo* synthesis but not recycling process coordinates with glutamine catabolism to control redox homeostasis and directs murine T cell differentiation. Elife 7: e36158.
6. Fang, L., et al. 2019. Myelin oligodendrocyte glycoprotein-IgG contributes to oligodendrocytopathy in the presence of complement, distinct from astrocytopathy induced by AQP4-IgG. Neurosci. Bull. 35: 853-866.
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RESEARCH USE

For research use only, not for use in diagnostic procedures.