SANTA CRUZ BIOTECHNOLOGY, INC.

C5 (029-03): sc-58934

SANTA CRUZ BIOTECHNOLOGY

The Power to Question

BACKGROUND

C3α, C4α and C5α are potent anaphylatoxins that are released during complement activation, a system of ligand-surface protein interactions specific to cells of hematopoietic lineage that aids in the elimination of pathogens. Complement C5 precursor contains C5α anaphylatoxin. C3α and C5α secretion correlates with pathophysiological phenotypes such as asthma and bacterial meningitis. Binding of these proteins to their respective G protein-coupled receptors (C3αR, C5αR), which are present on the surface of myeloid leukocytes, induces proinflammatory events such as cellular degranulation, smooth muscle contraction, arachidonic acid metabolism, cytokine release, leukocyte activation and cellular chemotaxis. C5αR utilizes the Ras-Raf-ERK1/2 cascade, couples to G_i/G_{16} proteins, and is prevalent on the surface of hepatocyte, lung, smooth muscle and endothelial cells. Upon activation, C3αR and C5αR are susceptible to rapid GRK-mediated phosphorylation and Clathrin-coated vesicle targeting. The C5 precursor is first processed by the removal of four basic residues, forming two chains, α and β, linked by a disulfide bond. C5 convertase activates C5 by cleaving the α chain, releasing C5α anaphylatoxin and generating C5β.

REFERENCES

1. de Bruijn, M.H., et al. 1985. Human complement component C3: cDNA coding sequence and derived primary structure. Proc. Natl. Acad. Sci. USA 82: 708-712.
2. Buhl, A.M., et al. 1995. Mitogen-activated protein kinase activation requires two signal inputs from the human anaphylatoxin C5a receptor. J. Biol. Chem. 270: 19828-19832.
3. Stahel, P.F., et al. 1997. TNF-α-mediated expression of the receptor for anaphylatoxin C5a on neurons in experimental *Listeria* meningoencephalitis. J. Immunol. 159: 861-869.
4. Langkabel, P., et al. 1999. Ligand-induced phosphorylation of anaphylatoxin receptors C3aR and C5aR is mediated by G protein-coupled receptor kinases. Eur. J. Immunol. 29: 3035-3046.
5. Settmacher, B., et al. 1999. Modulation of C3a activity: internalization of the human C3a receptor and its inhibition by C5a. J. Immunol. 162: 7409-7416.
6. Humbles, A.A., et al. 2000. A role for the C3a anaphylatoxin receptor in the effector phase of asthma. Nature 406: 998-1001.

CHROMOSOMAL LOCATION

Genetic locus: C5 (human) mapping to 9q33.2.

SOURCE

C5 (029-03) is a mouse monoclonal antibody raised against full length native C5 of human origin.

PRODUCT

Each vial contains 100 µg IgG_1 in 1.0 ml of PBS with < 0.1% sodium azide and 0.1% gelatin.

APPLICATIONS

C5 (029-03) is recommended for detection of C5 in normal human plasma (PEG precipitated C5) in non reduced form only of human origin by Western Blotting (starting dilution 1:200, dilution range 1:100-1:1000) and solid phase ELISA (starting dilution 1:30, dilution range 1:30-1:3000); non cross-reactive with plasma from C5 deficient patients.

Suitable for use as control antibody for C5 siRNA (h): sc-42848, C5 shRNA Plasmid (h): sc-42848-SH and C5 shRNA (h) Lentiviral Particles: sc-42848-V.

Molecular Weight of C5: 190 kDa.

Positive Controls: Hep G2 cell lysate: sc-2227.

STORAGE

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

RESEARCH USE

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

PROTOCOLS

See our web site at www.scbt.com for detailed protocols and support products.