

C5b-9 (aE11): sc-58935

BACKGROUND

The complement component proteins: C2, C3, C4 and C5 are potent anaphylatoxins that are released during complement activation. Binding of these proteins to their respective G protein-coupled receptors, C3aR, C1R and C5aR, induces proinflammatory events, such as cellular degranulation, smooth muscle contraction, arachidonic acid metabolism, cytokine release, leukocyte activation and cellular chemotaxis. Activation of the complement system leads to the formation of C5b-9 terminal complex, and while C5b-9 can promote cell lysis, the sublytic assembly of C5b-9 on plasma membranes causes an opposite result and induces cell cycle activation and survival. C5b-9 can rescue oligodendrocytes from FAS-mediated apoptosis by regulating caspase-8 processing via PI 3-K signaling. C5b-9 may play a pro-inflammatory role in the acute phase of multiple sclerosis, but may also be neuroprotective during the chronic phase of the disease.

REFERENCES

1. Cybulsky, A.V., et al. 1999. Complement C5b-9 induces receptor tyrosine kinase transactivation in glomerular epithelial cells. *Am. J. Pathol.* 155: 1701-1711.
2. Soane, L., et al. 1999. Inhibition of oligodendrocyte apoptosis by sublytic C5b-9 is associated with enhanced synthesis of Bcl-2 and mediated by inhibition of caspase-3 activation. *J. Immunol.* 163: 6132-6138.
3. Montinaro, V., et al. 2000. Renal C3 synthesis in idiopathic membranous nephropathy: correlation to urinary C5b-9 excretion. *Kidney Int.* 57: 137-146.
4. Putzu, G.A., et al. 2000. Immunohistochemical localization of cytokines, C5b-9 and ICAM-1 in peripheral nerve of Guillain-Barre syndrome. *J. Neurol. Sci.* 174: 16-21.

SOURCE

C5b-9 (aE11) is a mouse monoclonal antibody raised against purified C5b-9 of human origin.

PRODUCT

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

APPLICATIONS

C5b-9 (aE11) is recommended for detection of a neoepitope exposed in C9 when incorporated into C5b-9 in both membrane bound (MAC) and fluid phase (SC5b-9) complexes of human and porcine origin by immunofluorescence (starting dilution 1:50, dilution range 1:50-1:500) and solid phase ELISA (starting dilution 1:30, dilution range 1:30-1:3000); not recommended for detection of the native components.

RECOMMENDED SUPPORT REAGENTS

To ensure optimal results, the following support reagents are recommended: 1) Immunofluorescence: use m-IgGκ BP-FITC: sc-516140 or m-IgGκ BP-PE: sc-516141 (dilution range: 1:50-1:200) with UltraCruz[®] Mounting Medium: sc-24941 or UltraCruz[®] Hard-set Mounting Medium: sc-359850.

STORAGE

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

SELECT PRODUCT CITATIONS

1. Wen, X.F., et al. 2011. Activation of the lectin complement pathway on human renal glomerular endothelial cells triggered by high glucose and mannose-binding lectin. *Afr. J. Biotechnol.* 10: 18539-18549.
2. Berends, E.T., et al. 2013. Distinct localization of the complement C5b-9 complex on Gram-positive bacteria. *Cell. Microbiol.* 15: 1955-1968.
3. Malik, R., et al. 2014. Transient hyperkemia in the setting of neuromyelitis optica (NMO). *Muscle Nerve* 50: 859-862.
4. Woehl, J.L., et al. 2017. The structural basis for inhibition of the classical and lectin complement pathways by *S. aureus* extracellular adherence protein. *Protein Sci.* 26: 1595-1608.
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8. Paganelli, F.L., et al. 2018. Group IIA-secreted phospholipase A₂ in human serum kills commensal but not clinical *Enterococcus faecium* isolates. *Infect. Immun.* 86: e00180-18.
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10. Arroyo-Olarte, R.D., et al. 2018. Complement system contributes to modulate the infectivity of susceptible TcI strains of *Trypanosoma cruzi*. *Mem. Inst. Oswaldo Cruz* 113: e170332.
11. Tradtrantip, L., et al. 2019. CD55 upregulation in astrocytes by statins as potential therapy for AQP4-IgG seropositive neuromyelitis optica. *J. Neuroinflammation* 16: 57.
12. Mulfaul, K., et al. 2020. Toll-like receptor 2 facilitates oxidative damage-induced retinal degeneration. *Cell Rep.* 30: 2209-2224.e5.
13. Chaturvedi, S., et al. 2020. Complement activity and complement regulatory gene mutations are associated with thrombosis in APS and CAPS. *Blood* 135: 239-251.
14. Langereis, J.D., et al. 2021. Complement factor D haploinsufficiency is associated with a reduced complement activation speed and diminished bacterial killing. *Clin. Transl. Immunology* 10: e1256.

RESEARCH USE

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