Complement

Technical
Data Sheet

Monoclonal Antibodies:
Murine Anti-Human S Protein (Vitronectin)

For Research Use Only. Not for use in Diagnostic Procedures.

Background

In contract to membrane-formed C5b-9 complexes, C5b-9 complexes formed in the fluid phase contain an additional protein, the S protein. Subsequently, it was found that isolated S protein inhibited C5b-9 attachment. S protein functions like a "membrane analogue" in that it binds to the binding site generated on assembly of C5b-9 and is required for insertion of the complex into the membrane. As a result, SC5b-7, SC5b-8 or SC5b-9 complexes are formed which have lost their membrane-binding capacity. When the S protein was cloned and sequenced, it was found to be identical to Vitronectin.

Within the complement cascade, Vitronectin not only inhibits attachment of C5b-9 to target cells, but also prevents C9 polymerization. The biological function of Vitronectin is to minimize complement-mediated attack of cells in the vicinity of an ongoing complement attack. This may be relevant, not only during the generation of bactericidal activity in the case of infection, but also in autoimmune disease, immune complex disease, or inflammatory lesions, where complement is readily activated by dead or dying cells, exposed basement membranes or matrix proteins. S protein is present in human plasma at an approximate concentration of 150 µg/mL.

Specificity

The specificity of the monoclonal antibody was established via ELISA and RIA. This antibody was shown to bind S protein (Vitronectin) immobilized in microtiter wells and immunoprecipitates highly purified, radiolabeled S protein (Vitronectin). It binds to the SC5b-9 complex.

Applications

EIA[1]	RIA	IHC[2-3]	WB	FACS
>1:10,000	N/T	>1:1000	N/T	N/T

N/T = Not tested.

Specifications

Catalog Number:	A237
Concentration:	1.0-1.2 mg/ml
Purity:	≥ 95% by SDS PAGE
Volume/Vial:	100 µl
Storage:	
≤ 30 Days	2-8˚C
> 30 Days	≤ –20˚C
Buffer:	Borate Buffered Saline (pH 8.4 ± 0.2)
Isotype:	IgG_1k

Species Cross Reactivity:

None tested.

References

1 On file with Quidel Corporation.

2 Rosoklija, GB. et al. Local activation of the complement system in endoneurial microvessels of diabetic neuropathy. Acta Neuropathol 99(1):55-62 (2000).

3 Verhuis, R. et al. Complement activation in amyloid plaques in Alzheimer's Disease does not proceed further than C3. Virchows Arch 426(6):603-610 (1995).

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