

Product datasheet for **AM32955PU-N**

CD55 / DAF Mouse Monoclonal Antibody [Clone ID: RDIII-7]

Product data:

Product Type:	Primary Antibodies
Clone Name:	RDIII-7
Applications:	FC, FN, IF, IHC, WB
Recommended Dilution:	Flow Cytometry (Ref.2,3). Western blot: Non-reduced with a band size of 60-70kDa. As positive control CHO-Rat DAF hyper-expressing cells were used (Ref.1-6). Functional Studies: RDIII-7 antibody completely blocked the protective effect of expressed Rat DAF (Ref.2,3,6). Immunofluorescence (Ref.4,5). Immunohistochemistry on Frozen Sections (Ref.3-5). The typical starting working dilution is 1/50.
Reactivity:	Rat
Host:	Mouse
Isotype:	IgG1
Clonality:	Monoclonal
Immunogen:	NIH-3T3 cells expressing GPI-anchored Rat DAF
Specificity:	The monoclonal antibody RDIII-7 recognizes complement decay accelerating factor (DAF), also designated as CD55. RDIII-7 recognizes the common extracellular SCR region of CD55, detecting all isoforms of the protein.
Formulation:	PBS State: Purified State: Liquid 0.2 µm filtered Ig fraction Stabilizer: 0.1% BSA
Concentration:	lot specific
Purification:	Protein G Chromatography
Conjugation:	Unconjugated
Storage:	Store undiluted at 2-8°C. DO NOT FREEZE!
Stability:	Shelf life: one year from despatch.



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Background: Cells express on their surface several proteins which protect against complement attack, namely C receptor 1 (CR1), decay accelerating factor (DAF), membrane cofactor protein (MCP) and CD59. CR1, DAF and MCP regulate the activation pathways of complement by either accelerating decay of the C3 and C5 convertase (CR1, DAF), or acting as cofactors for the serine protease factor I, which cleaves and irreversibly inactivates C3b (CR1, MCP). Rat DAF (CD55) is a 60 kDa transmembrane protein that binds C3b and C4b to inhibit formation and half-life of the C3 convertases. It belongs to the receptors of complement activation (RCA) family. DAF is broadly distributed among cells in contact with plasma complement proteins, including both haematopoietic and nonhaematopoietic cells. Although DAF does not have an essential role in controlling hemolysis of erythrocytes, it has an important role in regulation of the deposition of C3 on nucleated cells. Together with other complement regulators DAF protects self cells from autologous complement-mediated injury. DAF cooperates with CD46 in circumventing autologous C3 deposition, while CD59 inhibits the pathway at the critical end-point.