

**CERTIFICATE OF ANALYSIS – TECHNICAL DATA SHEET**

<b>Product name</b>	DAF, Rat, clone RDII-24	<b>Expiry date</b>	-
<b>Catalog number</b>	HM3036-20UG		
<b>Lot number</b>	-	<b>Amount</b>	20 µg
<b>Volume</b>	200 µl	<b>Concentration</b>	100 µg/ml
<b>Formulation</b>	0.2 µm filtered in PBS+0.1%BSA	<b>Conjugate</b>	None
<b>Host Species</b>	Mouse IgG1	<b>Purification</b>	Protein G
<b>Endotoxin</b>	<24 EU/mg		
<b>Storage</b>	4°C		

**Application notes**

	IHC-F	IHC-P	IF	FC	FS	IA	IP	W
Reference #	2			1,2	2	3		1,2
Yes	•			•	•	•		•
No								
N.D.		•	•				•	

N.D.= Not Determined; IHC = Immuno histochemistry; F = Frozen sections; P = Paraffin sections; IF = Immuno Fluorescence; FC = Flow Cytometry; FS = Functional Studies; IA = Immuno Assays; IP = Immuno Precipitation; W = Western blot

Dilutions to be used depend on detection system applied. It is recommended that users test the reagent and determine their own optimal dilutions. The typical starting working dilution is 1:50.

- W: non-reduced with a band size of 60-70kDa. As positive control CHO-rat DAF hyper-expressing cells were used.

**General Information**

<b>Description</b>	The monoclonal antibody RDII-24 recognizes complement decay accelerating factor (DAF), also designated as CD55.. Cells express on their surface several proteins which protect against complement attack, namely C receptor I (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.
<b>Immunogen</b>	NIH-3T3 cells expressing GPI-anchored rat DAF
<b>Aliases</b>	Complement Decay Accelerating Factor, CD55
<b>References</b>	<ol style="list-style-type: none"> <li>1. Spiller, B et al; Efficient generation of monoclonal antibodies against surface-expressed proteins by hyperexpression in rodent cells. J Immunol Methods 1999, 224: 51</li> <li>2. Spiller, B et al; Tissue distribution of the rat homologue of decay accelerating factor. Immunol 1999, 97: 374</li> <li>3. Harris, C et al; Coupling complement regulators to immunoglobulin domains generates effective anti-complement reagents with extended half-life in vivo. Clin Exp Immunol 2002, 129: 198</li> </ol>
<b>Storage&amp;stability</b>	Product should be stored at 4°C. Under recommended storage conditions, product is stable for at least one year.

**Precautions**

For research use only. Not for use in or on humans or animals or for diagnostics. It is the responsibility of the user to comply with all local/state and federal rules in the use of this product. Hycult Biotech is not responsible for any patent infringements that might result from the use or derivation of this product.

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We hereby certify that the above-stated information is correct and that this product has been successfully tested by the Quality Control Department. This product was released for sale according to the existing specifications. This document has been produced electronically and is valid without a signature.

Approved by Manager of QC  
Brenda Teunissen

Date  
11/01/2021

Do you have any questions or comments regarding this product? Please contact us via [support@hycultbiotech.com](mailto:support@hycultbiotech.com).