

**CD55 / Decay Accelerating Factor (DAF) Antibody - With BSA and Azide**  
**Mouse Monoclonal Antibody [Clone F4-29D9 ]**  
**Catalog # AH11136**

**Specification**

---

**CD55 / Decay Accelerating Factor (DAF) Antibody - With BSA and Azide - Product Information**

Application	,3,4,
Primary Accession	<a href="#">P08174</a>
Other Accession	<a href="#">1604</a> , <a href="#">126517</a>
Reactivity	Human
Host	Mouse
Clonality	Monoclonal
Isotype	Mouse / IgG1, kappa
Calculated MW	70kDa KDa

**CD55 / Decay Accelerating Factor (DAF) Antibody - With BSA and Azide - Additional Information**

**Gene ID** 1604

**Other Names**

Complement decay-accelerating factor, CD55, CD55, CR, DAF

**Storage**

Store at 2 to 8°C. Antibody is stable for 24 months.

**Precautions**

CD55 / Decay Accelerating Factor (DAF) Antibody - With BSA and Azide is for research use only and not for use in diagnostic or therapeutic procedures.

**CD55 / Decay Accelerating Factor (DAF) Antibody - With BSA and Azide - Protein Information**

**Name** CD55

**Synonyms** CR, DAF

**Function**

This protein recognizes C4b and C3b fragments that condense with cell-surface hydroxyl or amino groups when nascent C4b and C3b are locally generated during C4 and c3 activation. Interaction of daf with cell-associated C4b and C3b polypeptides interferes with their ability to catalyze the conversion of C2 and factor B to enzymatically active C2a and Bb and thereby prevents the formation of C4b2a and C3bBb, the amplification convertases of the complement cascade (PubMed: [7525274](http://www.uniprot.org/citations/7525274)). Inhibits complement activation by destabilizing and preventing the formation of C3 and C5 convertases, which prevents complement damage (PubMed: [28657829](http://www.uniprot.org/citations/28657829)).

### Cellular Location

[Isoform 1]: Cell membrane; Single-pass type I membrane protein [Isoform 3]: Secreted [Isoform 5]: Secreted [Isoform 7]: Cell membrane; Lipid-anchor, GPI-anchor

### Tissue Location

Expressed on the plasma membranes of all cell types that are in intimate contact with plasma complement proteins. It is also found on the surfaces of epithelial cells lining extracellular compartments, and variants of the molecule are present in body fluids and in extracellular matrix

### CD55 / Decay Accelerating Factor (DAF) Antibody - With BSA and Azide - Protocols

Provided below are standard protocols that you may find useful for product applications.

- [Western Blot](#)
- [Blocking Peptides](#)
- [Dot Blot](#)
- [Immunohistochemistry](#)
- [Immunofluorescence](#)
- [Immunoprecipitation](#)
- [Flow Cytometry](#)
- [Cell Culture](#)

### CD55 / Decay Accelerating Factor (DAF) Antibody - With BSA and Azide - Images

### CD55 / Decay Accelerating Factor (DAF) Antibody - With BSA and Azide - Background

Recognizes a single chain glycoprotein of 70kDa, identified as CD55 (also known as decay accelerating factor, DAF). This MAbs was clustered in Kobe at the Sixth International Workshop on Human Leukocyte Differentiation Antigens as F429D-9 (N-L120). CD55/DAF is widely expressed on cells throughout the body including leukocytes, erythrocytes, epithelium, endothelium, and fibroblasts. It is a Glycosyl phosphatidylinositol anchored (GPI-anchored) member of the membrane bound complement regulatory proteins that inhibit autologous complement cascade activation. It prevents the amplification steps of the complement cascade by interfering with the assembly of the C3-convertases, C4b2a and C3bBb, and the C5-convertase, C4b2a3b and C3bBb3b. CD55 also serves as receptor for CD97 and for echovirus and Coxsackie B virus. Anti-CD55 can be used as marker for paroxysmal nocturnal hemoglobinuria (PNH).

### CD55 / Decay Accelerating Factor (DAF) Antibody - With BSA and Azide - References

Schlossman S et al. (eds) Leukocyte Typing V. Oxford University Press, Oxford, 1995. Kishimoto T. et al., eds. Leukocyte Typing VI, Garland Publishing, Inc, New York and London, 1997. B.E. Loveland - in Leukocyte Typing VI - Part 6 - Known Non-Lineage CD Antigens - NL11 - CD55 Workshop Panel Report pp519-520, 199