

**DAF/CD55 (E7G2U) XP<sup>®</sup> Rabbit mAb**

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

Applications:	Reactivity:	Sensitivity:	MW (kDa):	Source/Isotype:	UniProt ID:	Entrez-Gene Id:
W, IHC-Bond, IHC-P	H	Endogenous	78	Rabbit IgG	#P08174	1604

**Product Usage Information****Application**

Western Blotting  
IHC Leica Bond  
Immunohistochemistry (Paraffin)

**Dilution**

1:1000  
1:600  
1:1200

**Storage**

Supplied in 10 mM sodium HEPES (pH 7.5), 150 mM NaCl, 100 µg/ml BSA, 50% glycerol and less than 0.02% sodium azide. Store at -20°C. Do not aliquot the antibody.

For a carrier free (BSA and azide free) version of this product see product #89928.

**Specificity/Sensitivity**

DAF/CD55 (E7G2U) XP<sup>®</sup> Rabbit mAb recognizes endogenous levels of total DAF/CD55 protein.

**Source / Purification**

Monoclonal antibody is produced by immunizing animals with a synthetic peptide corresponding to residues surrounding Lys110 of human DAF/CD55 protein.

**Background**

Decay-accelerating factor (DAF/CD55) is a GPI-linked plasma membrane glycoprotein normally expressed on the surface of vascular endothelial and hematopoietic cells, which are continuously exposed to autologous complement components. In conjunction with other membrane complement regulatory proteins (CD35, CD46, and CD59), DAF/CD55 protects healthy cells from inappropriate complement-mediated lysis (1). DAF/CD55 inhibits activation of the complement cascade by promoting membrane dissociation and inactivation of C3 convertase, which inhibits amplification of the classical and alternative complement cascades (2). Research studies have demonstrated that DAF/CD55 is overexpressed in a variety of solid and liquid tumors, which functions to protect tumor cells from complement-mediated attack (3,4). Given its ability to disable the complement cascade and facilitate immune evasion by tumor cells, DAF/CD55 has received attention as a potential therapeutic target for the treatment of human malignancies. CD55 deficiency is also linked to human disease. The inability to express CD55 on the surface of erythrocytes renders them highly susceptible to complement-mediated lysis, which contributes to the development of paroxysmal nocturnal hemoglobinuria (PNH). PNH is characterized by hemolytic anaemia, pancytopenia, and venous thrombosis (5).

**Background References**

1. Fishelson, Z. et al. (2003) *Mol Immunol* 40, 109-23.
2. Brodbeck, W.G. et al. (1996) *J Immunol* 156, 2528-33.
3. Inoue, T. et al. (2002) *Mol Pathol* 55, 193-9.
4. Niehans, G.A. et al. (1996) *Am J Pathol* 149, 129-42.
5. Brodsky, R.A. (2014) *Blood* 124, 2804-11.

**Species Reactivity**

Species reactivity is determined by testing in at least one approved application (e.g., western blot).

**Western Blot Buffer**

**IMPORTANT:** For western blots, incubate membrane with diluted primary antibody in 5% w/v BSA, 1X TBS, 0.1% Tween® 20 at 4°C with gentle shaking, overnight.

**Applications Key**

**W:** Western Blotting **IHC-Bond:** IHC Leica Bond **IHC-P:** Immunohistochemistry (Paraffin)

**Cross-Reactivity Key**

**H:** Human

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