

## DESCRIPTION

<b>Species Reactivity</b>	Human
<b>Specificity</b>	Detects human CD55/DAF in direct ELISAs and Western blots.
<b>Source</b>	Polyclonal Goat IgG
<b>Purification</b>	Antigen Affinity-purified
<b>Immunogen</b>	Mouse myeloma cell line NS0-derived recombinant human CD55/DAF Asp35-Ser353 Accession # P08174
<b>Formulation</b>	Lyophilized from a 0.2 µm filtered solution in PBS with Trehalose. See Certificate of Analysis for details. *Small pack size (-SP) is supplied either lyophilized or as a 0.2 µm filtered solution in PBS.

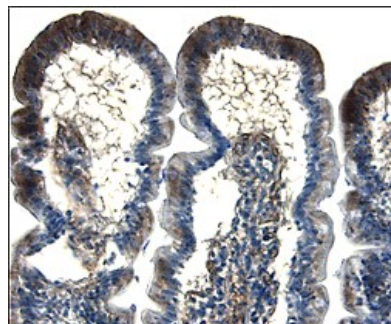
## APPLICATIONS

**Please Note:** Optimal dilutions should be determined by each laboratory for each application. *General Protocols* are available in the *Technical Information* section on our website.

	<b>Recommended Concentration</b>	<b>Sample</b>
<b>Western Blot</b>	0.1 µg/mL	Recombinant Human CD55/DAF (Catalog # 2009-CD)
<b>Immunohistochemistry</b>	5-15 µg/mL	See Below

## DATA

### Immunohistochemistry



**CD55/DAF in Human Colon Cancer Tissue.** CD55/DAF was detected in immersion fixed paraffin-embedded sections of human colon cancer tissue using 15 µg/mL Goat Anti-Human CD55/DAF Antigen Affinity-purified Polyclonal Antibody (Catalog # AF2009) overnight at 4 °C. Tissue was stained with the Anti-Goat HRP-DAB Cell & Tissue Staining Kit (brown; Catalog # CTS008) and counterstained with hematoxylin (blue). View our protocol for [Chromogenic IHC Staining of Paraffin-embedded Tissue Sections](#).

## PREPARATION AND STORAGE

<b>Reconstitution</b>	Reconstitute at 0.2 mg/mL in sterile PBS.
<b>Shipping</b>	The product is shipped at ambient temperature. Upon receipt, store it immediately at the temperature recommended below. *Small pack size (-SP) is shipped with polar packs. Upon receipt, store it immediately at -20 to -70 °C
<b>Stability &amp; Storage</b>	<b>Use a manual defrost freezer and avoid repeated freeze-thaw cycles.</b> <ul style="list-style-type: none"> <li>● 12 months from date of receipt, -20 to -70 °C as supplied.</li> <li>● 1 month, 2 to 8 °C under sterile conditions after reconstitution.</li> <li>● 6 months, -20 to -70 °C under sterile conditions after reconstitution.</li> </ul>

**BACKGROUND**

CD55, also known as DAF or decay-accelerating factor, is a 70-75 kDa member of the RCA family of proteins. Human RCA (regulators of complement/C' activation) proteins are products of chromosome 1 genes that are ubiquitously expressed on cells exposed to plasma complement proteins (1-4). A hallmark of RCA proteins is the presence of four to 30 SCRs (short consensus repeats; also called CCPs for C' control protein modules) in their plasma-exposed regions. SCRs are characterized by a 60-65 amino acid (aa) module that contains a highly conserved Trp residue and two internal disulfide bonds that create a  $\beta$ -barrel structure (1). Human CD55 is synthesized as a 381 aa precursor that contains a 34 aa signal sequence, a 319 aa mature region and a 28 aa C-terminal prosegment (5, 6). The mature region contains four SCR modules and a C-terminal O-glycosylated extension (7). Following cleavage of the prosegment, a serine is exposed that serves as an anchor for a GPI-linkage (8). Multiple polymorphisms are found in the molecule. Alternate splicing also exists. One form that may not be translated shows an intron insertion in the prosegment, resulting in a 79 aa substitution for the standard C-terminal 20 aas of the prosegment (6). Another form generates a truncated 199 aa precursor that cannot be membrane-bound and may not be secreted (9). Mature CD55 is 53% and 84% aa identical to mouse and monkey CD55, respectively. CD55 is known to bind CD97 via the first SCR (4). It also binds physiologically-generated C3 convertases with its second and third SCRs (7, 10). Binding results in an accelerated "decay", or dissociation of active C3 convertases, thus blocking the development of C' attack complexes on nonforeign cells (1, 2). Finally, viruses and bacteria are also known to utilize multiple SCR sites for infection (4).

**References:**

1. Herbert, A. *et al.* (2002) *Biochem. Soc. Trans.* **30**:990.
2. Miwa, T. and W-C. Song (2001) *Int. Immunopharmacol.* **1**:445.
3. Hourcade, D. *et al.* (2000) *Immunopharmacology* **49**:103.
4. Lea, S. (2002) *Biochem. Soc. Trans.* **30**:1014.
5. Medof, M.E. *et al.* (1987) *Proc. Natl. Acad. Sci. USA* **84**:2007.
6. Caras, I.W. *et al.* (1987) *Nature* **325**:545.
7. Lukacik, P. *et al.* (2004) *Proc. Natl. Acad. Sci. USA* **101**:1279.
8. Moran, P. *et al.* (1991) *J. Biol. Chem.* **266**:1250.
9. Lublin, D.M. *et al.* (1994) *Blood* **84**:1276.
10. Williams, P. *et al.* (2003) *J. Biol. Chem.* **278**:10691.