

## DESCRIPTION

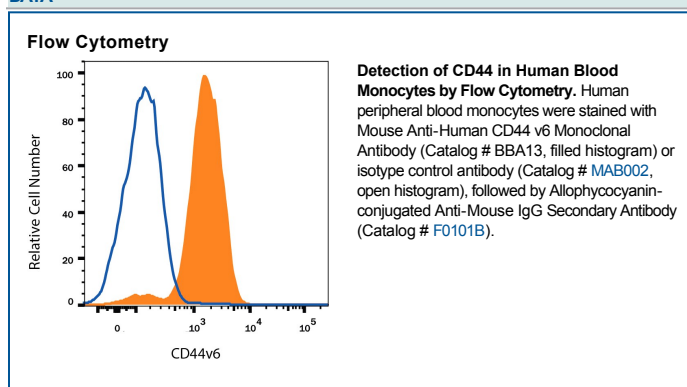
<b>Species Reactivity</b>	Human
<b>Specificity</b>	Detects human CD44v6 in Western blots. By flow cytometry analysis using a panel of CD44-transfected COS cells (9), monoclonal antibody clone 2F10 was deduced to be specific for human CD44 protein isoforms containing variant exon 6 (CD44v6).
<b>Source</b>	Monoclonal Mouse IgG <sub>1</sub> Clone # 2F10
<b>Purification</b>	Protein A or G purified from ascites
<b>Immunogen</b>	Recombinant human CD44 v3-10
<b>Formulation</b>	Lyophilized from a 0.2 µm filtered solution in PBS with Trehalose. See Certificate of Analysis for details.

## 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>	1 µg/mL	Recombinant Human CD44 Fc Chimera (Catalog # 3660-CD)
<b>Flow Cytometry</b>	0.25 µg/10 <sup>6</sup> cells	See Below
<b>Immunocytochemistry</b>	8-25 µg/mL	Immersion fixed human peripheral blood mononuclear cells
<b>Immunoprecipitation</b>	Fox, S.B. <i>et al.</i> (1994) <i>Cancer Res.</i> <b>54</b> :4539.	

## DATA



## PREPARATION AND STORAGE

<b>Reconstitution</b>	Sterile PBS to a final concentration of 0.5 mg/mL.
<b>Shipping</b>	The product is shipped at ambient temperature. Upon receipt, store it immediately at the temperature recommended below.
<b>Stability &amp; Storage</b>	<p><b>Use a manual defrost freezer and avoid repeated freeze-thaw cycles.</b></p> <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

CD44 is a ubiquitously expressed protein that is the major receptor for hyaluronan and exerts control over cell growth and migration (1-3). Human CD44 has a 20 amino acid (aa) signal sequence, an extracellular domain (ECD) with a 100 aa hyaluronan-binding disulfide-stabilized link region and a 325-530 aa stem region, a 21 aa transmembrane domain, and a 72 aa cytoplasmic domain. Within the stem, ten variably spliced exons (v1-10, exons 6-15) produce multiple protein isoforms (1-3). The standard or hematopoietic form, CD44H, does not include the variable segments (1-3). Cancer aggressiveness and T cell activation have been correlated with expression of specific isoforms (1, 3). CD44v6 contains exon 10 and is associated with tumor progression and metastasis in many types of cancer including breast, colon, lung, renal, skin, and ovarian tumors. With variable N- and O-glycosylation and splicing within the stalk, CD44 can range from 80 to 200 kDa (1). Within the N-terminal invariant portion of the ECD (aa 21-220), human CD44 shares 76%, 76%, 86%, 83% and 79% identity with corresponding mouse, rat, equine, canine and bovine CD44, respectively. The many reported functions of CD44 fall within three categories (1). First, CD44 binds hyaluronan and other ligands within the extracellular matrix and can function as a "platform" for growth factors and metalloproteinases. Second, CD44 can function as a co-receptor that modifies activity of receptors including MET and the ERBB family of tyrosine kinases. Third, the CD44 intracellular domain links the plasma membrane to the actin cytoskeleton via the ERM proteins, ezrin, radixin and moesin. CD44 can be synthesized in a soluble form (4) or may be cleaved at multiple sites by either membrane-type matrix metalloproteinases, or ADAM proteases to produce soluble ectodomains (5, 6). The cellular portion may then undergo gamma secretase-dependent intramembrane cleavage to form an A $\beta$ -like transmembrane portion and a cytoplasmic signaling portion that affects gene expression (7, 8). These cleavage events are thought to promote metastasis by enhancing tumor cell motility and growth (1, 5).

## References:

1. Ponta, H. *et al.* (2003) *Nat. Rev. Mol. Cell Biol.* **4**:33.
2. Sreaton, G.R. *et al.* (1992) *Proc. Natl. Acad. Sci. USA* **89**:12160.
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5. Nagano, O. and H. Saya (2004) *Cancer Sci.* **95**:930.
6. Nakamura, H. *et al.* (2004) *Cancer Res.* **64**:876.
7. Murakami, D. *et al.* (2003) *Oncogene* **22**:1511.
8. Lammich, S. *et al.* (2002) *J. Biol. Chem.* **277**:44754.
9. Fox, S.B. *et al.* (1994) *Cancer Res.* **54**:4539.