

## DESCRIPTION

<b>Species Reactivity</b>	Human/Mouse/Rat
<b>Specificity</b>	Detects human Vimentin in Western blots. Unconjugated antibody detects mouse and rat Vimentin in immunocytochemistry.
<b>Source</b>	Monoclonal Rat IgG <sub>2A</sub> Clone # 280618
<b>Purification</b>	Protein A or G purified from hybridoma culture supernatant
<b>Immunogen</b>	<i>E. coli</i> -derived recombinant human Vimentin Ser2-Glu466 Accession # P08670
<b>Conjugate</b>	Phycoerythrin Excitation Wavelength: 488 nm Emission Wavelength: 565-605 nm
<b>Formulation</b>	Supplied in a saline solution containing BSA and Sodium Azide. See Certificate of Analysis for details.  *Contains <0.1% Sodium Azide, which is not hazardous at this concentration according to GHS classifications. Refer to the Safety Data Sheet (SDS) for additional information and handling instructions.

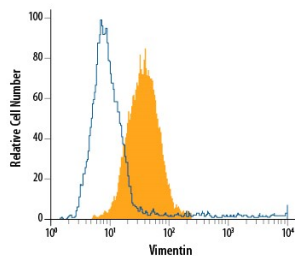
## APPLICATIONS

**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.

	Recommended Concentration	Sample
Intracellular Staining by Flow Cytometry	10 $\mu$ L/10 <sup>6</sup> cells	See Below

## DATA

### Intracellular Staining by Flow Cytometry



**Detection of Vimentin in A172 Human Cell Line by Flow Cytometry.** A172 human glioblastoma cell line was stained with Rat Anti-Human/Mouse/Rat Vimentin PE-conjugated Monoclonal Antibody (Catalog # IC2105P, filled histogram) or isotype control antibody (Catalog # IC006P, open histogram). To facilitate intracellular staining, cells were fixed with Flow Cytometry Fixation Buffer (Catalog # FC004) and permeabilized with Flow Cytometry Permeabilization/Wash Buffer I (Catalog # FC005). View our protocol for [Staining Intracellular Molecules](#).

## PREPARATION AND STORAGE

<b>Shipping</b>	The product is shipped with polar packs. Upon receipt, store it immediately at the temperature recommended below.
<b>Stability &amp; Storage</b>	<b>Protect from light. Do not freeze.</b> <ul style="list-style-type: none"> <li>12 months from date of receipt, 2 to 8 °C as supplied.</li> </ul>

**BACKGROUND**

Vimentin is a 57 kDa class III intermediate filament (IF) protein that belongs to the intermediate filament family. It is the predominant IF in cells of mesenchymal origin such as vascular endothelium and blood cells (1-3). The human Vimentin cDNA encodes a 466 amino acid (aa) protein that contains head and tail regions with multiple regulatory Ser/Thr phosphorylation sites, and a central rod domain with three coiled-coil regions separated by linkers (1, 2). Human Vimentin shares 97-98% aa identity with mouse, rat, ovine, bovine, and canine Vimentin. Sixteen Vimentin coiled-coil dimers self-assemble to form intermediate (10-12 nm wide) filaments (4). These filaments then anneal longitudinally to form non-polarized fibers that support cell structure and withstand stress (4). IF fibers are highly dynamic, and half-life depends on the balance between kinase and phosphatase activity. For example, phosphorylation followed by dephosphorylation drives IF disintegration, followed by reorganization during mitosis (1, 5, 6). Interactions of head and tail domains link IFs with other structures such as actin and microtubule cytoskeletons (7). Vimentin is involved in positioning autophagosomes, lysosomes and the Golgi complex within the cell (8). It facilitates cell migration and motility by recycling internalized trailing edge integrins back to the cell surface at the leading edge (9-11). Vimentin helps maintain the lipid composition of cellular membranes, and caspase cleavage of Vimentin is a key event in apoptosis (8, 12). Phosphorylation promotes secretion of Vimentin by TNF- $\alpha$ -stimulated macrophages (13). Extracellular Vimentin has been shown to associate with several microbes, and appears to promote an antimicrobial oxidative burst (13, 14). Cell-associated Vimentin can also interact with NKp46 to recruit NK cells to tuberculosis-infected monocytes (15).

**References:**

1. Omary, M.B. *et al.* (2006) Trends Biochem. Sci. **31**:383.
2. Ivaska, J. *et al.* (2007) Exp. Cell Res. **313**:2050.
3. Ferrari, S. *et al.* (1986) Mol. Cell. Biol. **6**:3614.
4. Sokolova, A.V. *et al.* (2006) Proc. Natl. Acad. Sci. USA **103**:16206.
5. Eriksson, J.E. *et al.* (2004) J. Cell Sci. **117**:919.
6. Li, Q-F. *et al.* (2006) J. Biol. Chem. **281**:34716.
7. Esue, O. *et al.* (2006) J. Biol. Chem. **281**:30393.
8. Styers, M.L. *et al.* (2005) Traffic **6**:359.
9. McInroy, L. and A. Maata (2007) Biochem. Biophys. Res. Commun. **360**:109.
10. Nieminen, M. *et al.* (2006) Nat. Cell Biol. **8**:156.
11. Ivaska, J. *et al.* (2005) EMBO J. **24**:3834.
12. Byun, Y. *et al.* (2001) Cell Death Differ. **8**:443.
13. Mor-Vaknin, N. *et al.* (2003) Nat. Cell Biol. **5**:59.
14. Zou, Y. *et al.* (2006) Biochem. Biophys. Res. Commun. **351**:625.
15. Garg, A. *et al.* (2006) J. Immunol. **177**:6192.