

## PAI-1 (D9C4) Rabbit mAb



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

Applications: W	Reactivity: H Mk B	<b>Sensitivity:</b> Endogenous	<b>MW (kDa):</b> 48	<b>Source/Isotype:</b> Rabbit IgG	UniProt ID: #P05121	Entrez-Gene Id: 5054
Product Usage Information		<b>Application</b> Western Blotting			<b>Dilution</b> 1:1000	
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.				
Specificity/Sensitivity		PAI-1 (D9C4) Rabbit mAb recognizes endogenous levels of total PAI-1 protein.				
Source / Purification		Monoclonal antibody is produced by immunizing animals with a synthetic peptide corresponding to residues surrounding Arg294 of human PAI-1 protein.				
Background		PAI-1 is a secreted protein that belongs to the serine proteinase inhibitor (serpin) superfamily. It inhibits urokinase and tissue plasminogen activators (uPA and tPA) and thus, reduces the conversion of inactive plasminogen to plasmin (1). PAI-1 regulates fibrinolysis and plays an important role in vessel patency and tissue remodeling. Secreted PAI-1 interacts with the extracellular matrix (ECM) component vitronectin, thereby modulating cell-ECM interactions (2,3). PAI-1 is expressed in a variety of tissues with higher expression in liver, vascular endothelial cells, platelets, macrophages, and adipose tissue (1). Increased levels of PAI-1 are associated with deep vein thrombosis (4). Defects in PAI-1 cause plasminogen activator inhibitor-1 deficiency (PAI-1D), which is characterized by increased bleeding after injury or surgery (5). Research studies have shown that high levels of PAI-1 are associated with obesity, aging, insulin resistance, and type 2 diabetes (6-8). PAI-1 is transcriptionally regulated by TGF-β and mediates TGF-β-induced inhibition of cell migration and invasion in cancer cells (9). Studies have also shown PAI-1 to be involved in fibrosis (10).				
Background References		<ol> <li>Pannekoek, H. et al. (1986) EMBO J 5, 2539-44.</li> <li>Sigurdardottir, O. and Wiman, B. (1994) Biochim Biophys Acta 1208, 104-10.</li> <li>Konstantinides, S. et al. (2001) Circulation 103, 576-83.</li> <li>Baldwin, J.F. et al. (2012) J Vasc Surg 56, 1089-97.</li> <li>Fay, W.P. et al. (1997) Blood 90, 204-8.</li> <li>Pannacciulli, N. et al. (2002) Obes Res 10, 717-25.</li> <li>Juhan-Vague, I. et al. (1991) Diabetologia 34, 457-62.</li> <li>Hashimoto, Y. et al. (1987) Thromb Res 46, 625-33.</li> <li>Humbert, L. and Lebrun, J.J. (2012) Cell Signal 25, 490-500.</li> <li>Zhang, L.P. et al. (1999) J Hepatol 31, 703-11.</li> </ol>				

**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 nonfat

dry milk, 1X TBS, 0.1% Tween® 20 at 4°C with gentle shaking, overnight.

**Applications Key** W: Western Blotting

**Cross-Reactivity Key** H: Human Mk: Monkey B: Bovine

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