## 🗧 Granzyme M (E7B4W) Rabbit mAb





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Applications: W	Reactivity: H	<b>Sensitivity:</b> Endogenous	<b>MW (kDa):</b> 25-30	<b>Source/Isotype:</b> Rabbit IgG	<b>UniProt ID:</b> #P51124	Entrez-Gene Id: 3004		
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. <i>Do not aliquot the antibody.</i>				ol and less than		
Specificity/Sensitivity		Granzyme M (E7B4W) Rabbit mAb recognizes endogenous levels of total Granzyme M protein. This antibody does not cross-react with human Granzyme A, B, H, or K proteins.						
Source / Purific	cation	Monoclonal antibody is produced by immunizing animals with a synthetic peptide corresponding to residues surrounding Ala141 of human Granzyme M protein.						
Background		Granzymes are a family of serine proteases expressed by cytotoxic T lymphocytes and natural killer (NK) cells and are key components of immune responses to pathogens and transformed cells (1). Granzymes are synthesized as zymogens and are processed into mature enzymes by cleavage of a leader sequence. They are released by exocytosis in lysosome-like granules containing perforin, a membrane pore-forming protein. Granzyme B has the strongest apoptotic activity of all the granzymes as a result of its caspase-like ability to cleave substrates at aspartic acid residues thereby activating procaspases directly and cleaving downstream caspase substrates (2,3). Granzyme M prefers to cleave after a Methionine or Leucine residue, and proteolyzes a restricted set of macromolecular substrates (4-7). Granzyme M is highly expressed in NK, NKT, $\gamma\delta$ T cells, and in differentiated effector CD8 <sup>+</sup> T cells, suggesting a role for Granzyme M in both innate and adaptive immunity (4-11). After perforin-mediated entry into a target cell, various reports show Granzyme M can induce apoptosis and tumor cell death, stimulate LPS-mediated inflammation, or inhibit viral replication (4,11-15).						
Background Re	eferences		<ol> <li>Immunol. Rev. 19</li> <li>Immunol. Rev. 19</li> <li>tton, V.R. (2003) Cu.</li> <li>ovenschen, N. (2014</li> <li>933) J Immunol 151</li> <li>Immunogenetic</li> <li>944) J Biol Chem 279</li> <li>955) Immunogenetic</li> <li>(2010) Mol Immunol</li> <li>17, 117</li> <li>et al. (2013) Cell Death D</li> <li>(2014) Cell Death D</li> <li>et al. (2010) J Immunol</li> </ol>	3, 31-8. <i>rr. Opin. Immunol.</i> 15, 53 4) <i>Cell Death Differ</i> 21, 35 5, 6195-205. 5, 44, 340-50. 5, 54275-82. <i>ics</i> 42, 101-11. <i>ol</i> 47, 903-11. 1419-28. <i>vath Differ</i> 20, 419-29. 1-8. <i>viter</i> 21, 416-26. <i>inol</i> 185, 7605-13.				
Species Reactiv	vity	Species reactivity is de	etermined by testin	g in at least one approve	ed application (e.g.,	western blot).		
Western Blot B	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 K	ey	W: Western Blotting						
Cross-Reactivity Key H: Human								
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