PhosphoPlus [®] SAPK/JNK (Thr183/Tyr185) Antibody Duet					
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For Research Use On	ly. Not for Use in Diagnostic Procedures.				
#P45983	5599				
Product Includes		Product #	Quantity	Mol. Wt.	Isotype/Source
Phospho-SAPK/JNK (Th	r183/Tyr185) (81E11) Rabbit mAb	4668	100 µl	46, 54 kDa	Rabbit IgG
SAPK/JNK Antibody		9252	200 µl	46, 54 kDa	Rabbit
Storage	applications. Supplied in 10 mM sodium HEPE 0.02% sodium azide. Store at –20	S (pH 7.5), 150 mM N)°C. Do not aliquot the	laCl, 100 μg/m e antibody.	I BSA, 50% glyc	cerol and less than
Background	The stress-activated protein kinas activated by a variety of environm inflammatory cytokines, and in so MAPKs, the core signaling unit is mixed lineage kinases (MLKs), wh phosphorylate and activate the S/ GTPases of the Rho family (Rac, and MLKs (3). Alternatively, MKK4 of a germinal center kinase (GCK undergoes alternative splicing, re- translocate to the nucleus and req transcription factors (3,5).	The stress-activated protein kinase/Jun-amino-terminal kinase SAPK/JNK is potently and preferentially activated by a variety of environmental stresses, including UV and gamma radiation, ceramides, inflammatory cytokines, and in some instances, growth factors and GPCR agonists (1-6). As with the other MAPKs, the core signaling unit is composed of a MAPKKK, typically MEKK1-MEKK4, or by one of the mixed lineage kinases (MLKs), which phosphorylate and activate MKK4/7. Upon activation, MKKs phosphorylate and activate the SAPK/JNK kinase (2). Stress signals are delivered to this cascade by small GTPases of the Rho family (Rac, Rho, cdc42) (3). Both Rac1 and cdc42 mediate the stimulation of MEKKs and MLKs (3). Alternatively, MKK4/7 can be activated in a GTPase-independent mechanism via stimulation of a germinal center kinase (GCK) family member (4). There are three SAPK/JNKK genes each of which undergoes alternative splicing, resulting in numerous isoforms (3). SAPK/JNKK, when active as a dimer, can translocate to the nucleus and regulate transcription through its effects on c-Jun, ATF-2, and other transcription factors (3,5).			
Background References	 Davis, R.J. (1999) <i>Biochem Soc</i> Ichijo, H. (1999) <i>Oncogene</i> 18, Kyriakis, J.M. and Avruch, J. (20 Kyriakis, J.M. (1999) <i>J Biol Che</i> Leppä, S. and Bohmann, D. (19 Whitmarsh, A.J. and Davis, R.J. 	c Symp 64, 1-12. 6087-93. 001) Physiol Rev 81, 8 m 274, 5259-62. 999) Oncogene 18, 61. . (1998) Trends Bioche	307-69. 58-62. em Sci 23, 481	L-5.	
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