Store at -20C	Phospho-Akt2 Pair II	(Ser474) Matched	Antibody	ibody Cell Signaling		
					Orders:	877-616-CELL (2355) orders@cellsignal.com	
95					Support:	877-678-TECH (8324)	
#42495	Species Cross Reactivity: H M	UniProt ID: #P31751	Entrez-Gene Id: #208		Web:	info@cellsignal.com cellsignal.com	
#4				3 Trask Lane	Danvers Mas	ssachusetts 01923 USA	

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

Product Includes		Product #	Quantity	Isotype/Source Rabbit IgG	
Phospho-Akt (Ser473) (D9E) XP [®] Rab	bit mAb (BSA and Azide Free)	31957	100 µg		
Akt2 (A5J8O) Mouse mAb (BSA and A	66588	100 µg	Mouse IgG1		
Description	The Phospho-Akt2 (Ser474) Matched An and high throughput ELISA platforms re labeling. Labels include fluorophores, la Matched Antibody Pairs include MSD, Q LANCE, HTRF), and Luminex.	quiring antibody pairs v nthanides, biotin, and b	with specialized eads. Platforms	or custom antibody s requiring conjugated	
	Learn how Matched Antibody Pairs mov antibody-pairs.	e your projects forward	, faster at cst-sc	ience.com/matched-	
Specificity/Sensitivity	ted species, as determir other species.	ned through in-	house testing, but may		
Storage	Store at -20°C. <i>This product will freeze at -20°C so it is recommended to aliquot into single-use vials a avoid multiple freeze/thaw cycles</i> . A slight precipitate may be present and can be dissolved by gently vortexing. This will not interfere with antibody performance.				
Directions for Use	Matched Antibody Pairs consist of capture and detection antibodies that bind to non-overlapping epitopes. For specific identification of the capture and detection antibodies in this pair, please refer to the data figure caption. Optimal dilutions/concentrations should be determined by the end user.				
Formulation	Supplied in 1X PBS (10 mM Na2HPO4, 3 mM KCl, 2 mM KH2PO4, and 140 mM NaCl (pH 7.8)). BSA an Azide Free.				
Background	Akt, also referred to as PKB or Rac, plays a critical role in controlling cell survival and apoptosis (1-3). This protein kinase is activated by insulin and various growth and survival factors to function in a wortmannin-sensitive pathway involving PI3 kinase (2,3). Akt is activated by phospholipid binding and activation loop phosphorylation at Thr308 by PDK1 (4) and by phosphorylation within the carboxy terminus at Ser473. The previously elusive PDK2 responsible for phosphorylation of Akt at Ser473 has been identified as mammalian target of rapamycin (mTOR) in a rapamycin-insensitive complex with rictor and Sin1 (5,6). Akt promotes cell survival by inhibiting apoptosis through phosphorylation and inactivation of several targets, including Bad (7), forkhead transcription factors (8), c-Raf (9), and caspase-9. PTEN phosphatase is a major negative regulator of the PI3K/Akt signaling pathway (10). LY294002 is a specific PI3 kinase inhibitor (11). Another essential Akt function is the regulation of glycogen synthesis through phosphorylation and inactivation of GSK-3 α and β (12,13). Akt may also play a role in insulin stimulation of glucose transport (12). In addition to its role in survival and glycogen synthesis, Akt is involved in cell cycle regulation by preventing GSK-3 β -mediated phosphorylation and degradation of cyclin D1 (14) and by negatively regulating the cyclin-dependent kinase inhibitors p27 Kip1 (15) and p21 Waf1/Cip1 (16). Akt also plays a critical role in cell growth by directly phosphorylating mTOR in a rapamycin-sensitive complex containing raptor (17). More importantly, Akt phosphorylates and inactivates tuberin (TSC2), an inhibitor of mTOR within the mTOI raptor complex (18,19).				
Background References	 Franke, T.F. et al. (1997) <i>Cell</i> 88, 435-7. Burgering, B.M. and Coffer, P.J. (1995) Franke, T.F. et al. (1995) <i>Cell</i> 81, 727-34 Alessi, D.R. et al. (1996) <i>EMBO J</i> 15, 65 Sarbassov, D.D. et al. (2005) <i>Science</i> 33 Jacinto, E. et al. (2006) <i>Cell</i> 127, 125-37 Cardone, M.H. et al. (1998) <i>Science</i> 28 Brunet, A. et al. (1999) <i>Cell</i> 96, 857-68 Zimmermann, S. and Moelling, K. (199 Cantley, L.C. and Neel, B.G. (1999) <i>Pr</i> Vlahos, C.J. et al. (1994) <i>J Biol Chem</i> 2 	<i>Nature</i> 376, 599-602. 5. 41-51. 07, 1098-101. 7. 2, 1318-21. 99) <i>Science</i> 286, 1741-4. <i>oc Natl Acad Sci USA</i> 96,			

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