Calcium Ion Regulation	Antibody	Sample	er	T C	ell Signaling	
Store				Orders:	877-616-CELL (2355) orders@cellsignal.com	
1 Kit (6 x 20 microliters)				Support:	877-678-TECH (8324)	
#8575				Web:	info@cellsignal.com cellsignal.com	
8#			3 Trask Lane	Danvers Mas	sachusetts 01923 USA	
For Research Use Only. Not for Use in Diagnostic Procedures.						
Product Includes	Product #	Quantity	Mol. Wt		Isotype/Source	
ATP2A2/SERCA2 (D51B11) Rabbit mAb	9580	20 µl	114, 140 kDa		Rabbit IgG	

Isotype/Source AT Rabbit IgG 114, 140 kDa υ 20 µi Phospho-Phospholamban (Ser16/Thr17) Antibody 8496 20 µl 6 (monomer); 12, 24 (oligomers) kDa Rabbit Phospholamban (D9W8M) Rabbit mAb 14562 20 µl 12, 24 kDa Rabbit IgG Phospho-PKA C (Thr197) (D45D3) Rabbit mAb 5661 20 µl 42 kDa Rabbit IgG PKA C-α (D38C6) Rabbit mAb 5842 20 µl 42 kDa Rabbit IgG ATP2A1/SERCA1 (D54G12) Rabbit mAb 100 kDa Rabbit IgG 12293 20 µl Anti-rabbit IgG, HRP-linked Antibody 7074 100 µl Goat

Please visit cellsignal.com for individual component applications, species cross-reactivity, dilutions, protocols, and additional product information.

Description	The Calcium Ion Regulation Antibody Sampler Kit provides an economical way to investigate the regulation of calcium ions within the cell. The kit contains enough primary and secondary antibodies to perform two western blot experiments per primary antibody.
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.
Background	Sarcoplasmic and endoplasmic reticulum Ca ²⁺ ATPases (SERCA) are members of a highly conserved family of Ca ²⁺ pumps (1). ATP2A1 (SERCA1) is a fast-twitch, skeletal muscle sarcoplasmic reticulum (SR) Ca ²⁺ ATPase (2). Multiple ATP2A2 (SERCA2) isoforms have been isolated, with ATP2A2a (SERCA2a) found predominantly in the SR of muscle cells and ATP2A2b (SERCA2b) more ubiquitously expressed in the ER of most cell types (3). Post-translational modification of ATP2A2, including phosphorylation and tyrosine nitration, modify Ca ²⁺ -ATPase activity and calcium transport (4,5).
	Phospholamban (PLN) was identified as a major phosphoprotein component of the SR (6). Despite very high expression in cardiac tissue, phospholamban is also expressed in skeletal and smooth muscle (7). Localization of PLN is limited to the SR, where it serves as a regulator of the sarco-endoplasmic reticulum calcium ATPase, SERCA (8). PLN binds directly to SERCA and effectively lowers its affinity for calcium, thus reducing calcium transport into the SR. Phosphorylation of PLN at Ser16 by PKA or myotonic dystrophy protein kinase and/or phosphorylation at Thr17 by Ca ²⁺ /calmodulin-dependent protein kinase results in release of PLN from SERCA, relief of this inhibition, and increased calcium uptake by SR (reviewed in 9,10). It has long been held that phosphorylation at Ser16 and Thr17 occurs sequentially, but increasing evidence suggests that phosphorylation, especially at Thr17, may be differentially regulated (reviewed in 11,12).
	The second messenger cyclic AMP (cAMP) activates cAMP-dependent protein kinase (PKA or cAPK) in mammalian cells and controls many cellular mechanisms such as gene transcription, ion transport, and protein phosphorylation (13). Inactive PKA is a heterotetramer composed of a regulatory subunit (R) dimer and a catalytic subunit (C) dimer. In this inactive state, the pseudosubstrate sequences on the R subunits block the active sites on the C subunits. Three C subunit isoforms (C- α , C- β , and C- γ) and two families of the regulatory subunits (RI and RII) with distinct cAMP binding properties have been identified. Upon binding of cAMP to the R subunits, the auto-inhibitory contact is eased and active monomeric C subunits are released. PKA shares substrate specificity with Akt (PKB) and PKC, which are characterized by an arginine at position -3 relative to the phosphorylated serine or threonine residue (14). PKA phosphorylation is involved in the regulation of Ca ²⁺ channels, including Ca _v 1.1 in skeletal muscle and Ca _v 1.2 in the heart (reviewed in 15).
Background References	 Hovnanian, A. (2007) Subcell Biochem 45, 337-63. Odermatt, A. et al. (1996) Nat Genet 14, 191-4. de Smedt, H. et al. (1991) J Biol Chem 266, 7092-5. Hawkins, C. et al. (1995) Mol Cell Biochem 142, 131-8. Viner, R.I. et al. (1999) Biochem J 340 (Pt 3), 657-69. Kirchberber, M.A. et al. (1975) Recent Adv Stud Cardiac Struct Metab 5, 103-15.



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