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PhosphoPlus[®] ΑΜΡΚα (Thr172) Antibody Duet



Orders: 877-616-CELL (2355)

orders@cellsignal.com

Support: 877-678-TECH (8324)

Web: info@cellsignal.com

cellsignal.com

3 Trask Lane | Danvers | Massachusetts | 01923 | USA

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UniProt ID: Entrez-Gene Id: #Q13131, #P54646 5562, 5563

Product Includes	Product #	Quantity	Mol. Wt	Isotype/Source
Phospho-AMPKα (Thr172) (40H9) Rabbit mAb	2535	100 µl	62 kDa	Rabbit IgG
AMPKα (D5A2) Rabbit mAb	5831	100 µl	62 kDa	Rabbit IgG

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

Description

PhosphoPlus® Duets from Cell Signaling Technology (CST) provide a means to assess protein activation status. Each Duet contains an activation-state and total protein antibody to your target of interest. These antibodies have been selected from CST's product offering based upon superior performance in specified applications.

Storage

Supplied in 10 mM sodium HEPES (pH 7.5), 150 mM NaCl, $100 \mu g/ml$ BSA, 50% glycerol and less than 0.02% sodium azide. Store at -20°C. Do not aliquot the antibody.

Background

AMP-activated protein kinase (AMPK) is highly conserved from yeast to plants and animals and plays a key role in the regulation of energy homeostasis (1). AMPK is a heterotrimeric complex composed of a catalytic α subunit and regulatory β and γ subunits, each of which is encoded by two or three distinct genes (α 1, 2; β 1, 2; γ 1, 2, 3) (2). The kinase is activated by an elevated AMP/ATP ratio due to cellular and environmental stress, such as heat shock, hypoxia, and ischemia (1). The tumor suppressor LKB1, in association with accessory proteins STRAD and MO25, phosphorylates AMPKa at Thr172 in the activation loop, and this phosphorylation is required for AMPK activation (3-5). AMPKα is also phosphorylated at Thr258 and Ser485 (for α 1; Ser491 for α 2). The upstream kinase and the biological significance of these phosphorylation events have yet to be elucidated (6). The $\beta 1$ subunit is posttranslationally modified by myristoylation and multi-site phosphorylation including Ser24/25, Ser96, Ser101, Ser108, and Ser182 (6,7). Phosphorylation at Ser108 of the β1 subunit seems to be required for AMPK activation, while phosphorylation at Ser24/25 and Ser182 affects AMPK localization (7). Several mutations in AMPKy subunits have been identified, most of which are located in the putative AMP/ATP binding sites (CBS or Bateman domains). Mutations at these sites lead to reduction of AMPK activity and cause glycogen accumulation in heart or skeletal muscle (1,2). Accumulating evidence indicates that AMPK not only regulates the metabolism of fatty acids and glycogen, but also modulates protein synthesis and cell growth through EF2 and TSC2/mTOR pathways, as well as blood flow via eNOS/nNOS (1).

Background References

- 1. Hardie, D.G. (2004) *J Cell Sci* 117, 5479-87.
- 2. Carling, D. (2004) Trends Biochem Sci 29, 18-24.
- 3. Hawley, S.A. et al. (1996) *J Biol Chem* 271, 27879-87.
- 4. Lizcano, J.M. et al. (2004) EMBO J 23, 833-43.
- 5. Shaw, R.J. et al. (2004) Proc Natl Acad Sci USA 101, 3329-35.
- 6. Woods, A. et al. (2003) J Biol Chem 278, 28434-42.
- 7. Warden, S.M. et al. (2001) *Biochem J* 354, 275-83.

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