

Store at -20°C
#8208

PhosphoPlus® AMPKα (Thr172) Antibody Duet



Cell Signaling
TECHNOLOGY®

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

UniProt ID:
#Q13131, #P54646

Entrez-Gene Id:
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 µg/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 ($\alpha 1, 2$; $\beta 1, 2$; $\gamma 1, 2, 3$) (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 AMPK α at Thr172 in the activation loop, and this phosphorylation is required for AMPK activation (3-5). AMPK α is also phosphorylated at Thr258 and Ser485 (for $\alpha 1$; Ser491 for $\alpha 2$). The upstream kinase and the biological significance of these phosphorylation events have yet to be elucidated (6). The $\beta 1$ subunit is post-translationally modified by myristoylation and multi-site phosphorylation including Ser24/25, Ser96, Ser101, Ser108, and Ser182 (6,7). Phosphorylation at Ser108 of the $\beta 1$ subunit seems to be required for AMPK activation, while phosphorylation at Ser24/25 and Ser182 affects AMPK localization (7). Several mutations in AMPK γ 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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