Revision 1

Store at -20C

#8660

PPARγ Regulated Fatty Acid Metabolism Antibody Sampler Kit

1 Kit (7 x 20 microliters)

Cell Signaling TECHNOLOGY®

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

Product Includes	Product #	Quantity	Mol. Wt	Isotype/Source
Phospho-AMPKα (Thr172) (40H9) Rabbit mAb	2535	20 µl	62 kDa	Rabbit IgG
AMPKα (D5A2) Rabbit mAb	5831	20 µl	62 kDa	Rabbit IgG
CBP (D6C5) Rabbit mAb	7389	20 µl	300 kDa	Rabbit IgG
GCN5L2 (C26A10) Rabbit mAb	3305	20 µl	94 kDa	Rabbit IgG
PPARγ (C26H12) Rabbit mAb	2435	20 µl	53, 57 kDa	Rabbit IgG
SirT1 (C14H4) Rabbit mAb	2496	20 µl	120 kDa	Rabbit
RXRα (D6H10) Rabbit mAb	3085	20 µl	53 kDa	Rabbit IgG
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

PPARγ Regulated Fatty Acid Metabolism Antibody Sampler Kit provides an economical means to evaluate PPARγ and related proteins involved in lipid metabolism. This kit contains enough primary antibody to perform two western blots per primary.

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

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 (α1, 2; β1, 2; γ1, 2, 3) (1). 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 phosphorylates AMPKα at Thr172 in the activation loop, and this phosphorylation is required for AMPK activation (2-4). 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 (5).

CBP (CREB-binding protein) is a transcriptional co-activator that associates with PPARγ (6,7). CBP also contains histone acetyltransferase (HAT) activity, allowing it to acetylate histones and other proteins (7).

General Control of Amino Acid Synthesis Yeast Homolog Like 2 (GCN5L2) is a transcription adaptor protein and a histone acetyltransferase (HAT) that functions as the catalytic subunit of the STAGA and TFTC transcription coactivator complexes (8). GCN5L2 is 73% homologous to the p300/CBP-associated factor PCAF, another HAT protein found in similar complexes (9). GCN5L2 acetylates non-histone proteins such as the transcription co-activator PGC1-α (10).

Peroxisome proliferator-activated receptor γ (PPARγ) is a member of the ligand-activated nuclear receptor superfamily and functions as a transcriptional activator (11). PPARγ is preferentially expressed in adipocytes as well as in vascular smooth muscle cells and macrophage (12).

The Silent Information Regulator (SIR2) family of genes is a highly conserved group of genes that encode nicotinamide adenine dinucleotide (NAD)-dependent protein deacetylases, also known as class III histone deacetylases (13). SirT1, the mammalian ortholog of Sir2, is a nuclear protein implicated in the regulation of many cellular processes, including apoptosis, cellular senescence, endocrine signaling, glucose homeostasis, aging, and longevity. Targets of SirT1 include PPARγ (14), and the PPARγ coactivator-1α (PGC-1α) protein (15). Deacetylation of PPARγ and PGC-1α regulates the gluconeogenic/glycolytic pathways in the liver and fat mobilization in white adipocytes in response to fasting (14,15).

The human retinoid X receptors (RXRs) are type-II nuclear hormone receptors encoded by three distinct genes (RXRα, RXRβ, and RXRγ) and bind selectively and with high affinity to the vitamin A derivative, 9-cis-retinoic acid. Nuclear RXRs form heterodimers with PPAR to help regulate transcription during lipid metabolism (16).

Background References

1. Carling, D. (2004) *Trends Biochem Sci* 29, 18-24.
2. Hawley, S.A. et al. (1996) *J Biol Chem* 271, 27879-87.
3. Lizcano, J.M. et al. (2004) *EMBO J* 23, 833-43.

4. Shaw, R.J. et al. (2004) *Proc Natl Acad Sci U S A* 101, 3329-35.
5. Hardie, D.G. (2004) *J Cell Sci* 117, 5479-87.
6. Goodman, R.H. and Smolik, S. (2000) *Genes Dev* 14, 1553-77.
7. Chan, H.M. and La Thangue, N.B. (2001) *J Cell Sci* 114, 2363-73.
8. Candau, R. et al. (1996) *Mol Cell Biol* 16, 593-602.
9. Yang, X.J. et al. (1996) *Nature* 382, 319-24.
10. Lerin, C. et al. (2006) *Cell Metab* 3, 429-38.
11. Tontonoz, P. et al. (1995) *Curr Opin Genet Dev* 5, 571-6.
12. Rosen, E.D. et al. (1999) *Mol Cell* 4, 611-7.
13. Guarente, L. (1999) *Nat Genet* 23, 281-5.
14. Picard, F. et al. (2004) *Nature* 429, 771-6.
15. Rodgers, J.T. et al. (2005) *Nature* 434, 113-8.
16. Gronemeyer, H. et al. (2004) *Nat Rev Drug Discov* 3, 950-64.

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Limited Uses