Revision 1

Store at -20C

#12589

Adipogenesis Marker 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
Acetyl-CoA Carboxylase (C83B10) Rabbit mAb	3676	20 µl	280 kDa	Rabbit IgG
Adiponectin (C45B10) Rabbit mAb	2789	20 µl	27 kDa	Rabbit IgG
C/EBPα (D56F10) XP® Rabbit mAb	8178	20 µl	42, 28 kDa	Rabbit IgG
FABP4 Antibody	2120	20 µl	15 kDa	Rabbit
Fatty Acid Synthase (C20G5) Rabbit mAb	3180	20 µl	273 kDa	Rabbit IgG
Perilipin-1 (D1D8) XP® Rabbit mAb	9349	20 µl	62 kDa	Rabbit IgG
PPARγ (C26H12) Rabbit mAb	2435	20 µl	53, 57 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

The Adipogenesis Marker Antibody Sampler Kit provides an economical means to evaluate proteins involved in the regulation of adipogenesis. The kit includes enough antibody to perform two western blot experiments with each 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

Adipocytes are the primary cellular component of adipose tissue and play a key role in the storage of triacylglycerol. Adipogenesis is the cellular process where preadipocytes differentiate into adipocytes.

Fatty acid binding proteins (FABPs) act as cytoplasmic lipid chaperones by binding fatty acids and lipids for transport to various cellular pathways (1,2). The predominant fatty acid binding protein found in adipocytes is FABP4.

Adiponectin is an adipokine expressed exclusively in brown and white adipocytes and is secreted into the blood. It exists in three major forms: a low molecular weight trimer, a medium molecular weight hexamer and a high molecular weight multimer (3). Decreased adiponectin levels are seen in obese and insulin-resistant mice and humans (4), suggesting that this adipokine is critical for maintenance of insulin sensitivity.

Peroxisome proliferator-activated receptor γ (PPARγ) is a transcriptional activator preferentially expressed in adipocytes, vascular smooth muscle cells, and macrophages (5,6).

Acetyl-CoA carboxylase (ACC) is a key fatty acid biosynthesis and oxidation enzyme that is responsible for the carboxylation of acetyl-CoA to malonyl-CoA, (7). Phosphorylation of acetyl-CoA carboxylase by AMPK at Ser79 or by PKA at Ser1200 inhibits ACC enzymatic activity (8). ACC is a potential target of anti-obesity drugs (9,10).

CCAAT/enhancer-binding proteins (C/EBPs) transcription factors are critical for cellular differentiation, terminal function, and the inflammatory response (11). Phosphorylation of C/EBPα at Thr222, Thr226, and Ser230 by GSK-3 may be required for adipogenesis (12).

Perilipin localizes to the periphery of lipid droplets and serves as a protective coating against lipases. Evidence suggests that PKA regulates lipolysis by phosphorylating perilipin (13-17), resulting in a conformational change that exposes lipid droplets to endogenous, hormone-sensitive lipases (14). Hence, perilipin plays a pivotal role in lipid storage (14,17).

Fatty acid synthase (FASN) catalyzes the synthesis of long-chain fatty acids from acetyl-CoA and malonyl-CoA. FASN is active as a homodimer with seven different catalytic activities and produces lipids in the liver for export to metabolically active tissues or storage in adipose tissue. In most other human

tissues, FASN is minimally expressed since they rely on circulating fatty acids for new structural lipid synthesis (18).

Background References

1. Tuncman, G. et al. (2006) *Proc Natl Acad Sci U S A* 103, 6970-5.
2. Haunerland, N.H. and Spener, F. (2004) *Prog Lipid Res* 43, 328-49.
3. Kadowaki, T. et al. (2006) *J Clin Invest* 116, 1784-92.
4. Hu, E. et al. (1996) *J Biol Chem* 271, 10697-703.
5. Tontonoz, P. et al. (1995) *Curr Opin Genet Dev* 5, 571-6.
6. Rosen, E.D. et al. (1999) *Mol Cell* 4, 611-7.
7. Castle, J.C. et al. (2009) *PLoS One* 4, e4369.
8. Ha, J. et al. (1994) *J Biol Chem* 269, 22162-8.
9. Abu-Elheiga, L. et al. (2001) *Science* 291, 2613-6.
10. Levert, K.L. et al. (2002) *J Biol Chem* 277, 16347-50.
11. Lekstrom-Himes, J. and Xanthopoulos, K.G. (1998) *J Biol Chem* 273, 28545-8.
12. Ross, S.E. et al. (1999) *Mol Cell Biol* 19, 8433-41.
13. Greenberg, A.S. et al. (1991) *J Biol Chem* 266, 11341-6.
14. Brasaemle, D.L. (2007) *J Lipid Res* 48, 2547-59.
15. Ducharme, N.A. and Bickel, P.E. (2008) *Endocrinology* 149, 942-9.
16. Egan, J.J. et al. (1990) *J Biol Chem* 265, 18769-75.
17. Brasaemle, D.L. et al. (2009) *Mol Cell Biochem* 326, 15-21.
18. Katsurada, A. et al. (1990) *Eur J Biochem* 190, 427-33.

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