Revision 1

Store a

Gluconeogenesis Antibody Sampler Kit



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

Product Includes	Product #	Quantity	Mol. Wt	Isotype/Source	
Enolase-1 Antibody	3810	20 µl	47 kDa	Rabbit	
Enolase-2 (E2H9X) XP [®] Rabbit mAb	24330	20 µl	47 kDa	Rabbit IgG	
FBP1/FBPase 1 (D2T7F) Rabbit mAb	59172	20 µl	39 kDa	Rabbit IgG	
GPI (E2Q8J) XP [®] Rabbit mAb	94068	20 µl	60 kDa	Rabbit IgG	
PCK1 (D12F5) Rabbit mAb	12940	20 µl	63 kDa	Rabbit IgG	
PCK2 (D3E11) Rabbit mAb	8565	20 µl	71 kDa	Rabbit IgG	
PGAM1 (D3J9T) Rabbit mAb	12098	20 µl	28 kDa	Rabbit IgG	
PGK1 Antibody	68540	20 µl	43 kDa	Rabbit	
Pyruvate Carboxylase Antibody	66470	20 µl	130 kDa	Rabbit	
Anti-rabbit IgG, HRP-linked Antibody	7074	100 µl		Goat	

0.02% sodium azide. Store at -20°C. Do not aliquot the antibodies.

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

The Gluconeogenesis Antibody Sampler Kit provides an economical means of detecting select components involved in the gluconeogenesis metabolism pathway. The kit includes enough antibodies to perform two western blot experiments with each primary antibody.

Supplied in 10 mM sodium HEPES (pH 7.5), 150 mM NaCl, 100 µg/ml BSA, 50% glycerol and less than

Storage

Background

Description

Enolase is an important glycolytic enzyme involved in the interconversion of 2-phosphoglycerate to phosphoenolpyruvate. Mammalian enolase exists as three subunits: enolase-1 (α -enolase), enolase-2 (γ -enolase), and enolase-3 (β -enolase). Expression of the enolase isoforms differs in a tissue specific manner (1). Enolase-1 plays a key role in anaerobic metabolism under hypoxic conditions and may act as a cell surface plasminogen receptor during tissue invasion (2,3). Abnormal expression of enolase-1 is associated with tumor progression in some cases of breast and lung cancer (4-7). Alternatively, an enolase-1 splice variant (MBP-1) binds the c-myc promoter p2 and may function as a tumor suppressor. For this reason, enolase-1 is considered as a potential therapeutic target in the treatment of some forms of cancer (8). Research studies have shown elevated levels of neuron-specific enolase-2 in neuroblastoma (1) and small-cell lung cancer (9,10). Fructose-1,6-bisphosphatase 1 (FBP1 or FBPase 1), a rate-limiting enzyme in gluconeogenesis, catalyzes the conversion of fructose-1,6-bisphosphate to fructose-6-phosphate (11). Inhibition of FBP1 expression in basal-like breast cancer (BLBC) cells leads to metabolic reprogramming, including enhanced glycolysis, which leads to increased glucose uptake, biosynthesis of macromolecules, and activation of PKM2 (11). This metabolic reprogramming endows tumor cells with cancer stem cell (CSC)-like properties, thereby increasing their tumorigenicity (11). Depletion of FBP1 was also reported in more than 600 clear cell renal cell carcinoma (ccRCC) tumors, suggesting that FBP1 may inhibit ccRCC tumor progression (12). Glucose-6-phosphate isomerase (GPI) is a multi-functional protein belonging to the glucose phosphate isomerase family (13,14). As an intracellular metabolic enzyme, GPI plays a pivotal role in glycolysis and gluconeogenesis by catalyzing the interconversion of D-glucose-6-phosphate and D-fructose-6-phosphate (15). GPI is also secreted, where it functions as a cytokine (referred to as Autocrine Motility Factor, AMF), acting via the E3ubiquitin-protein ligase AMFR/gp78 (16). In normal tissues, GPI/AMF has been shown to promote both immune cell maturation and neuronal cell survival (17,18). It is also secreted in abundance by some tumor cells (19), where it has been shown to promote tumor cell migration and metastasis (20,21). Phosphoenolpyruvate carboxykinase 1 (PCK1, PEPCK1, or PEPCK-C) is a cytosolic enzyme responsible for the conversion of oxaloacetate to phosphoenolpyruvate (22). PCK1 and PCK2 are involved in controlling the rate-limiting step of gluconeogenesis in the liver, which generates glucose from noncarbohydrate substrates, such as lactate and glycerol (23, 24). PCK2 (PEPCK2 or PEPCK-M) encodes an isoform of phosphoenolpyruvate carboxykinase (PEPCK) that is found in the mitochondria of renal and hepatic tissues (22). Phosphoglycerate mutase (PGAM1) catalyzes the conversion of 3phosphoglycerate to 2-phosphoglycerate during glycolysis (25-29). Research studies have shown

1 Kit (6 x 20 microliters)

	increased PGAM1 expression in cancer (25-28) and mental disease (29). PGK1 (phosphoglycerate kinase) is an essential enzyme in the glycolysis pathway (30). It catalyzes the reversible phospho- transfer reaction from 1,3-diphosphoglycerate to ADP to form ATP and 3-phosphoglycerate. The expression of PGK1 is upregulated in many cancer types and plays an important role in cancer cell proliferation and metastasis (31-34). Pyruvate carboxylase (PC) catalyzes the carboxylation of pyruvate to oxaloacetate to replenish TCA cycle intermediates. It is also critical in regulating gluconeogenesis in the liver (35).
Background References	 Pancholi, V. (2001) <i>Cell Mol Life Sci</i> 58, 902-20. Redlitz, A. et al. (1995) <i>Eur J Biochem</i> 227, 407-15. Jiang, B.H. et al. (2003) <i>Cancer Res</i> 57, 5328-35. Peebles, K.A. et al. (2000) <i>J Surg Res</i> 93, 108-19. Wu, W. et al. (2000) <i>J Surg Res</i> 93, 108-19. Wu, W. et al. (2000) <i>J Exp Metastasis</i> 19, 319-26. Hennipman, A. et al. (1988) <i>Tumour Biol</i> 9, 241-8. Feo, S. et al. (2000) <i>FEBS Lett</i> 473, 47-52. Stern, P. et al. (2013) <i>Cancer Cell</i> 23, 316-31. Li, B. et al. (2013) <i>Cancer Cell</i> 23, 316-31. Li, B. et al. (2000) <i>Biochim Biophys Acta</i> 1480, 235-44. Jeffery, C.J. et al. (2000) <i>Biochim Biophys Acta</i> 1480, 235-44. Jeffery, C.J. et al. (2000) <i>Biochemistry</i> 39, 955-64. Kim, J.W. and Dang, C.V. (2005) <i>Trends Biochem Sci</i> 30, 142-50. Fairbank, M. et al. (1986) <i>Science</i> 234, 576-81. Gurrey, M.E. et al. (1986) <i>Science</i> 234, 566-74. Lucarelli, G. et al. (2015) <i>Medicine (Baltimore)</i> 94, e2117. Liotta, L.A. et al. (1986) <i>Science</i> 234, 566-74. Funasaka, T. and Raz, A. (2007) <i>Cancer Metastasis Rev</i> 26, 725-35. Caton, P.W. et al. (2000) <i>Biol Reprod</i> 83, 859-65. Vander Heiden, M.G. et al. (2010) <i>Science</i> 38, 1302-6. Funasaka, T. and Raz, A. (2007) <i>Cancer Metastasis Rev</i> 26, 725-35. Caton, P.W. et al. (2008) <i>Microvasc</i> Res 76, 89-93. Rens, M.J. et al. (2005) <i>Nat Biotechnol</i> 23, 1303-7. Martins-de-Souza, D. et al. (2009) <i>BMC Psychiatry</i> 9, 17. Beutler, F. (2017) <i>Mature</i> 73, 51-1. Wilson, R.B. et al. (2017) <i>Plepatology</i> 65, 51-528. Cao, H. et al. (2017) <i>Plepatology</i> 65, 51-528. Cao, H. et al. (2017) <i>Plepatology</i> 65, 51-528. Cao, H. et al. (2017) <i>Cancer Chemother Pharmacol</i> 79, 985-994. Cao, H. et al. (2017)
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