

Store at
-20C
#94905**PhosphoPlus® Glycogen Synthase
(Ser641) Antibody Duet**
Orders: 877-616-CELL (2355)
orders@cellsignal.com

Support: 877-678-TECH (8324)

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For Research Use Only. Not for Use in Diagnostic Procedures.
UniProt ID: #P13807
Entrez-Gene Id: 2997

Product Includes	Product #	Quantity	Mol. Wt	Isotype/Source
Phospho-Glycogen Synthase (Ser641) (D4H1B) XP® Rabbit mAb	47043	100 µl	85-90 kDa	Rabbit IgG
Glycogen Synthase (GYS1/GYS2) (15B1) Rabbit mAb	3886	100 µl	84 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

Glycogen is a polysaccharide of glucose and serves as an energy storage in mammalian muscle and liver (1). Glycogen synthase catalyzes the rate-limiting step of glycogen biosynthesis and has two major isoforms in mammals: muscle isoform (glycogen synthase 1, GYS1) and liver isoform (glycogen synthase 2, GYS2), respectively (1). Glycogen synthase kinase-3α (GSK-3α) and glycogen synthase kinase-3β (GSK-3β) phosphorylate glycogen synthase at multiple sites in its C-terminus (Ser641, Ser645, Ser649, and Ser653), inhibiting its activity (2,3). Hypoxia alters glycogen metabolism including temporal changes of GYS1 expression and phosphorylation in cancer cells, suggesting the role of metabolic reprogramming of glycogen metabolism in cancer growth (1).

Background References

1. Favaro, E. et al. (2012) *Cell Metab* 16, 751-64.
2. Mora, A. et al. (2005) *FEBS Lett* 579, 3632-8.
3. Jensen, J. et al. (2012) *Am J Physiol Endocrinol Metab* 303, E82-9.

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