Revision 1

#8217 Store at -20C

PhosphoPlus® Stat1 (Tyr701) Antibody Duet

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
Stat1 (D1K9Y) Rabbit mAb	14994	100 µl	84, 91 kDa	Rabbit IgG
Phospho-Stat1 (Tyr701) (D4A7) Rabbit mAb	7649	100 µl	84, 91 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

The Stat1 transcription factor is activated in response to a large number of ligands (1) and is essential for responsiveness to IFN-α and IFN-γ (2,3). Phosphorylation of Stat1 at Tyr701 induces Stat1 dimerization, nuclear translocation, and DNA binding (4). Stat1 protein exists as a pair of isoforms, Stat1α (91 kDa) and the splice variant Stat1β (84 kDa). In most cells, both isoforms are activated by IFN-α, but only Stat1α is activated by IFN-γ. The inappropriate activation of Stat1 occurs in many tumors (5). In addition to tyrosine phosphorylation, Stat1 is also phosphorylated at Ser727 through a p38 mitogen-activated protein kinase (MAPK)-dependent pathway in response to IFN-α and other cellular stresses (6). Serine phosphorylation may be required for the maximal induction of Stat1-mediated gene activation.

Background References

1. Heim, M.H. (1999) *J Recept Signal Transduct Res* 19, 75-120.
2. Durbin, J.E. et al. (1996) *Cell* 84, 443-50.
3. Meraz, M.A. et al. (1996) *Cell* 84, 431-42.
4. Ihle, J.N. et al. (1994) *Trends Biochem Sci* 19, 222-7.
5. Frank, D.A. (1999) *Mol Med* 5, 432-56.
6. Wen, Z. et al. (1995) *Cell* 82, 241-50.

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