

**PhosphoPlus® Estrogen Receptor α
(Ser167) Antibody Duet****Orders:** 877-616-CELL (2355)
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3 Trask Lane | Danvers | Massachusetts | 01923 | USA

For Research Use Only. Not for Use in Diagnostic Procedures.**UniProt ID:**
#P03372**Entrez-Gene Id:**
2099

Product Includes	Product #	Quantity	Mol. Wt	Isotype/Source
Phospho-Estrogen Receptor α (Ser167) (D1A3) Rabbit mAb	5587	100 μ l	66 kDa	Rabbit IgG
Estrogen Receptor α (D8H8) Rabbit mAb	8644	100 μ l	66 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 μ g/ml BSA, 50% glycerol and less than 0.02% sodium azide. Store at -20°C . Do not aliquot the antibody.

Background

Estrogen receptor α (ER α), a member of the steroid receptor superfamily, contains highly conserved DNA binding and ligand binding domains (1). Through its estrogen-independent and estrogen-dependent activation domains (AF-1 and AF-2, respectively), ER α regulates transcription by recruiting coactivator proteins and interacting with general transcriptional machinery (2). Phosphorylation at multiple sites provides an important mechanism to regulate ER α activity (3-5). Ser104, 106, 118, and 167 are located in the amino-terminal transcription activation function domain AF-1, and phosphorylation of these serine residues plays an important role in regulating ER α activity. Ser118 may be the substrate of the transcription regulatory kinase CDK7 (5). Ser167 may be phosphorylated by p90RSK and Akt (4,6). According to the research literature, phosphorylation at Ser167 may confer tamoxifen resistance in breast cancer patients (4).

Background References

1. Mangelsdorf, D.J. et al. (1995) *Cell* 83, 835-9.
2. Glass, C.K. and Rosenfeld, M.G. (2000) *Genes Dev* 14, 121-41.
3. Chen, D. et al. (1999) *Mol Cell Biol* 19, 1002-15.
4. Campbell, R.A. et al. (2001) *J Biol Chem* 276, 9817-24.
5. Chen, D. et al. (2000) *Mol Cell* 6, 127-37.
6. Joel, P.B. et al. (1998) *Mol Cell Biol* 18, 1978-84.

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