

**PhosphoPlus® Atg14 (Ser29) Antibody Duet**

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

**UniProt ID:**  
#Q6ZNE5

**Entrez-Gene Id:**  
22863

Product Includes	Product #	Quantity	Mol. Wt	Isotype/Source
Phospho-Atg14 (Ser29) (D4B8M) Rabbit Monoclonal Antibody	92340	100 µl	65 kDa	Rabbit IgG
Atg14 (D1A1N) Rabbit Monoclonal Antibody	96752	100 µl	65 kDa	Rabbit IgG

Please visit [cellsignal.com](http://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

Autophagy is a catabolic process for the autophagosomic-lysosomal degradation of bulk cytoplasmic contents (1,2). Autophagy is generally activated by conditions of nutrient deprivation but is also associated with a number of physiological processes including development, differentiation, neurodegeneration, infection, and cancer (3). The molecular machinery of autophagy was largely discovered in yeast and is directed by a number of autophagy-related (Atg) genes. These proteins are involved in the formation of autophagosomes, cytoplasmic vacuoles that are delivered to lysosomes for degradation. The class III type phosphoinositide 3-kinase (PI3K) Vps34 regulates vacuolar trafficking and autophagy (4,5). Multiple proteins associate with Vps34, including p105/Vps15, Beclin-1, UVRAG, Atg14, and Rubicon, to determine Vps34 function (6-12). Atg14 and Rubicon were identified based on their ability to bind to Beclin-1 and participate in unique complexes with opposing functions (9-12). Rubicon, which localizes to the endosome and lysosome, inhibits Vps34 lipid kinase activity; knockdown of Rubicon enhances autophagy and endocytic trafficking (11,12). In contrast, Atg14 localizes to autophagosomes, isolation membranes and ER, and can enhance Vps34 activity. Knockdown of Atg14 inhibits starvation-induced autophagy (11,12).

The serine/threonine kinase ULK1 phosphorylates Atg14 at Ser29 to promote autophagosome formation (13).

## Background References

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