Revision 2

Store at -20C

#70751

Autophagy Vesicle Nucleation Antibody Sampler Kit

1 Kit (8 x 20 microliters)

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
PI3 Kinase Class III (D9A5) Rabbit mAb	4263	20 µl	100 kDa	Rabbit
PIK3R4 Antibody	14580	20 µl	153 kDa	Rabbit
Beclin-1 (D40C5) Rabbit mAb	3495	20 µl	60 kDa	Rabbit IgG
Atg14 Antibody	5504	20 µl	65 kDa	Rabbit
UVRAG (D2Q1Z) Rabbit mAb	13115	20 µl	90 kDa	Rabbit IgG
Rubicon (D9F7) Rabbit mAb	8465	20 µl	130 kDa	Rabbit IgG
Bif-1 Antibody	4467	20 µl	42 kDa	Rabbit
Atg9A (D4O9D) Rabbit mAb	13509	20 µl	100-110 kDa	Rabbit IgG
Anti-rabbit IgG, HRP-linked Antibody	7074	100 µl		Goat

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

Description

The Autophagy Vesicle Nucleation Antibody Sampler Kit provides an economical means of detecting target proteins involved in autophagosome formation and maturation. The kit contains enough antibody to perform two western blot experiments per primary antibody.

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 antibodies.

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 PIK3R4/PI3K class III complex interacts with Beclin-1 to play a role during several stages of autophagy. Autophagosome formation is stimulated when Atg14 complexes with PIK3R4, PI3K class III, and Beclin-1. The UVRAG protein competes with Atg14 for Beclin-1 binding, forming a mutually exclusive complex with PIK3R4, PI3K class III, and Beclin-1 that regulates autophagosome maturation. Autophagosome maturation is impaired in the presence of the Beclin-1-binding protein Rubicon (4,5). Co-expression of PIK3R4 is required for PI3K class III activation and regulation by both Beclin-1/UVRAG and nutrient levels (6). Bif-1 directly binds to UVRAG, forming a complex with Beclin-1, resulting in increased PI3-kinase class III/Vps34 activity required for autophagosome maturation (7). Inhibition of GSK-3β, as seen during nutrient deprivation, results in increased expression of Bif-1, and can contribute to autophagic cell death (8). Atg9A is an integral membrane protein that is required for both the initiation and the expansion of the autophagosome (9,10). Recruitment of Atg9A to the autophagosomal membrane is dynamic and transient as Atg9A also cycles between autophagy-related structures known as omegasomes, the trans-Golgi network (TGN), and endosomes, and at no point becomes a stable component of the autophagosomal membrane (9,11). The precise regulation of Atg9A trafficking is not fully clarified, yet it is suggested to involve p38 mitogen-activated protein kinase (MAPK)-binding protein p38IP and the Beclin-1-binding protein Bif-1 (12,13).

Background References

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Limited Uses