

## ATG4C

**Reactivity:**Human Mouse Rat

**Tested applications:**WB IHC

**Recommended Dilution:**WB 1:500 - 1:2000 IHC 1:50 - 1:200

**Calculated MW:**52kDa

**Observed MW:**Refer to Figures

**Immunogen:**

Recombinant protein of human ATG4C

**Storage Buffer:**

Store at -20. Avoid freeze / thaw cycles. Buffer: PBS with 0.02% sodium azide, 50% glycerol, pH7.3.

**Synonym:**

ATG4C;APG4-C;APG4C;AUTL1;AUTL3;FLJ14867 ;

**Catalog #:**A1123

**Antibody Type:**

Polyclonal Antibody

**Species:**Rabbit

**Gene ID:**84938

**Isotype:**IgG

**Swiss Prot:**Q96DT6

**Purity:**Affinity purification

For research use only.

**Background:**

Autophagy is a catabolic process for the autophagosomic-lysosomal degradation of bulk cytoplasmic contents. Control of autophagy was largely discovered in yeast and involves proteins encoded by a set of autophagy-related genes (Atg) (1). Formation of autophagic vesicles requires a pair of essential ubiquitin-like conjugation systems, Atg12-Atg5 and Atg8-phosphatidylethanolamine (Atg8-PE), which are widely conserved in eukaryotes (2).

Numerous mammalian counterparts to yeast Atg proteins have been described, including three Atg8 proteins (GATE-16, GABARAP, and LC3) and four Atg4 homologues (Atg4A/autophagin-2, Atg4B/autophagin-1, Atg4C/autophagin-3, and Atg4D/autophagin-4) (3-5). The cysteine protease Atg4 is pivotal to autophagosome membrane generation and regulation. Atg4 primes the Atg8 homologue for lipidation by cleaving its carboxy terminus and exposing its glycine residue for E1-like enzyme Atg7. The Atg8 homologue is transferred to the E2-like enzyme Atg3 before forming the Atg8-PE conjugate. During later stages of autophagy, Atg4 can reverse this lipidation event by cleaving PE, thereby recycling the Atg8 homologue (6). Atg4C-deficient mice display a tissue-specific decrease in LC3 lipidation only when under stressful conditions such as prolonged starvation. Mutant mice also exhibit increased susceptibility to the development of chemical carcinogen induced fibrosarcomas suggesting that Atg4C may contribute to events associated with tumor progression (7).

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