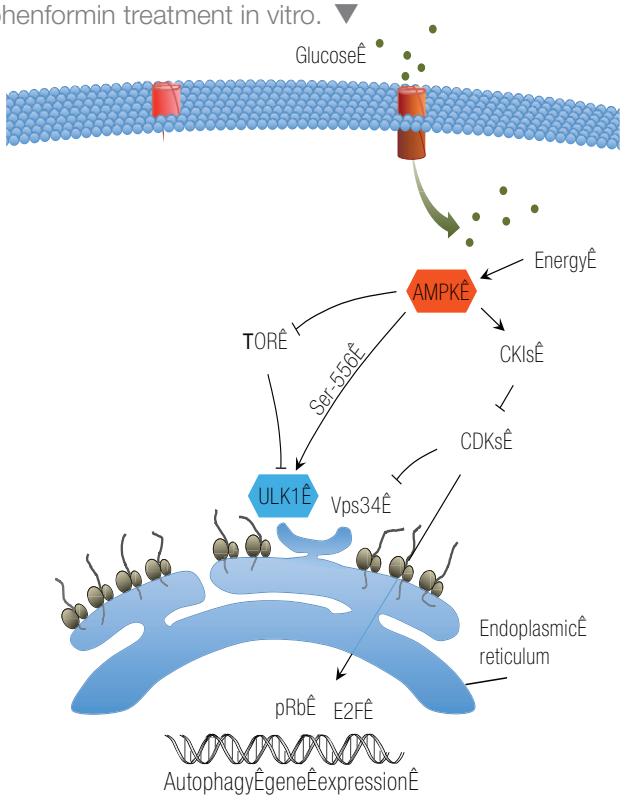
Crown insight

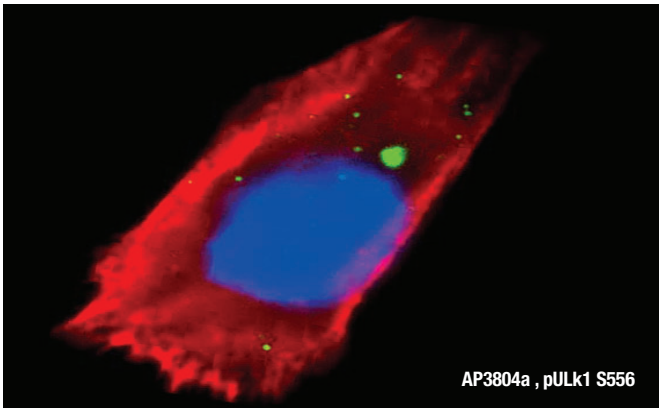
OCTOBER 2013 :

pUlk1

Introduction

Autophagy is induced by growth-dependent import of nutrients, such as amino acids and glucose, as well as through several key signaling molecules such as TOR (or mTOR) and AMPK. Amino acids activate the mTOR pathway, in part, by recruiting mTOR complexes to the lysosomal surface through factors such as the Rag GTPases and p62, and through effects on intracellular lysosomal positioning. The AMP-activated protein kinase (AMPK, red hexagon) responds to glucose uptake, and regulates multiple downstream targets that have effects on autophagy, including mTOR, Atg1 (Ulk1 in mammals, blue hexagon) and CDKs (through cyclin kinase inhibitors; CKIs). Ulk1 has been identified as an AMPK substrate phosphorylated at S467, S556, T574, and S637 after phenformin treatment in vitro. ▼





Fluorescent image of U251 MG cells stained with ULK1 (Phospho S556) Antibody #AP3804a. U251 MG cells treated with Chloroquine (50 µM, 16h) were fixed with 4% PFA (20 min), permeabilized with Triton X-100 (0.2%, 30 min) and incubated with ULK1 (Phospho S556) (1:200, 2hat RT), followed by secondary antibody, conjugated to Alexa Fluor® 488 (green) (1:1000, 1h). Cytoplasmic actin was counterstained with Alexa Fluor® 555 (red) conjugated Phalloidin (5.25 µM, 25 min). Nuclei were counterstained with Hoechst 33342 (blue).

Selected Abgent Products

CAT. #	TARGET NAME
AP3804a	Phospho-Ulk1-S556
AP1817b	Atg16l
AP11448a	Atg4d (N-term)
AP1812a	Atg5 (N-term)
AP1813c	Atg7
AP1814e	Atg9a
AP1814c	Atg9A (C-term)
AM1818a	Beclin 1 (Ascites)
AP1801i	Lc3 (Isoform B)
AP3430a	Phospho-Atg3-Y18
AP3392a	Phospho-Atg4C-S177
AP1817d	Atg16l
AP2183b	Sqstm1 (p62, C-term)
AP1850b	Uvrag (C-term)

Tested in U251 MG cells treated with Chloroquine (50 µM, 16h)

Visual categorization

Target associated (orange)

Autophagy Stem Cell Neurodegeneration

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