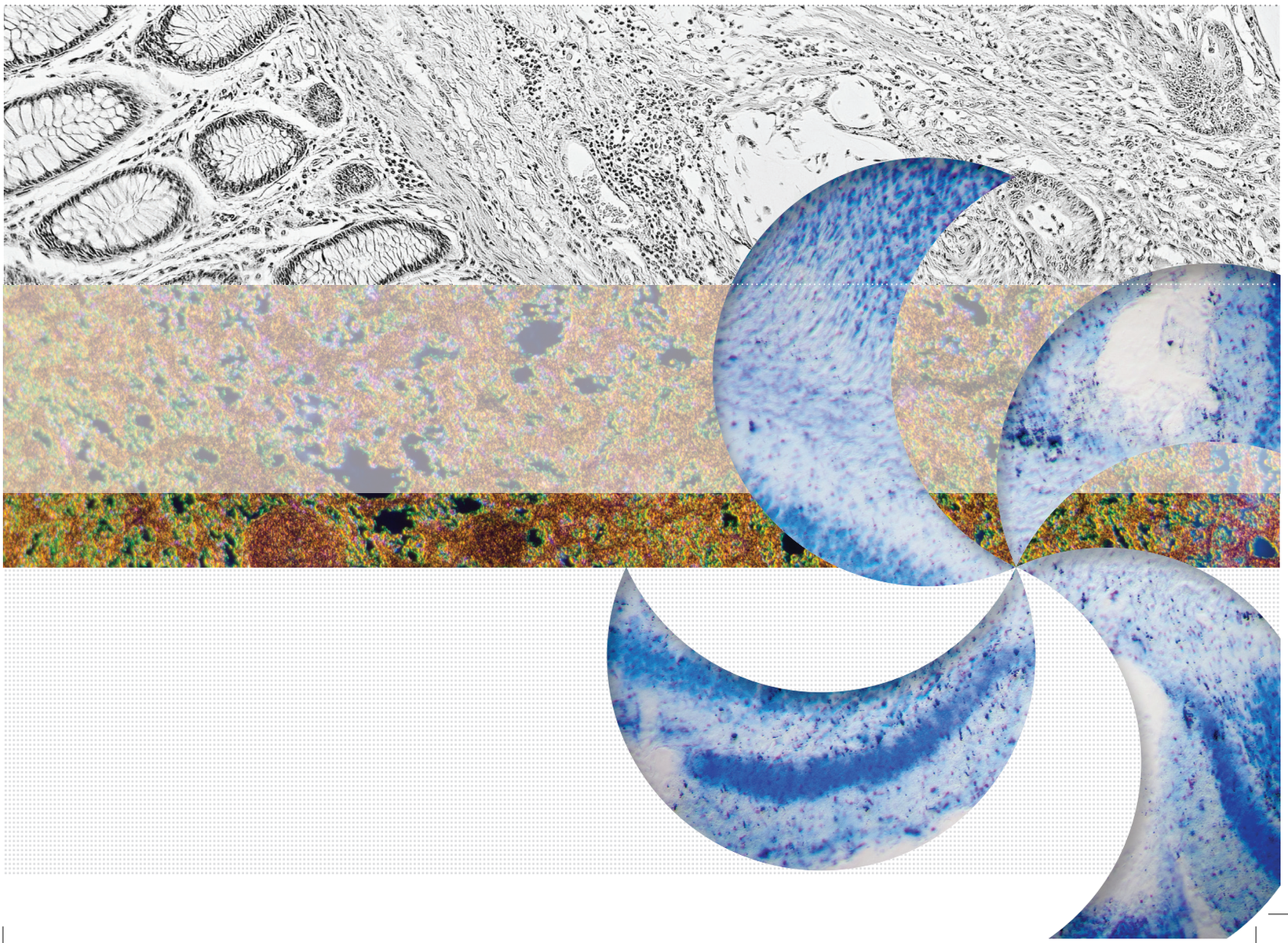
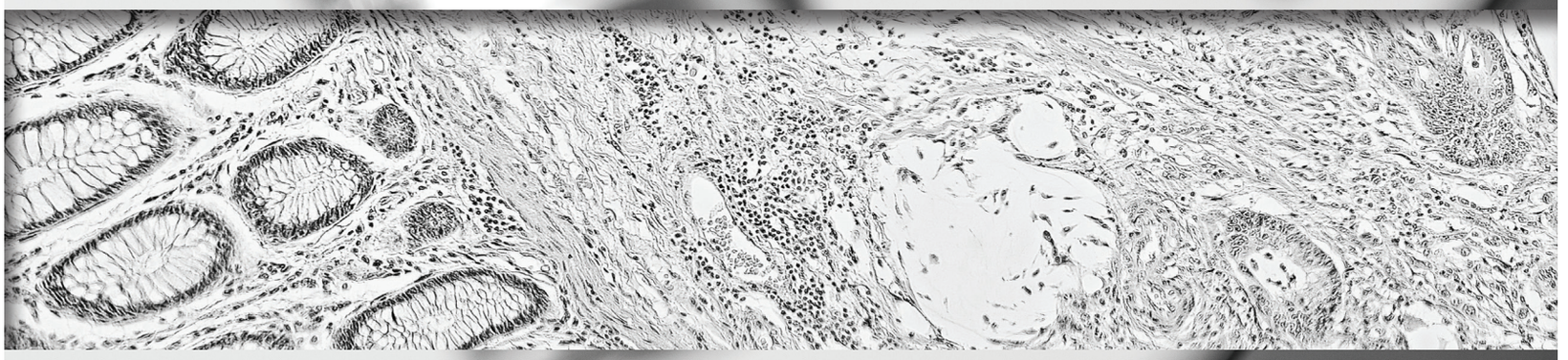
abcepta

Autophagy Antibodies





Abcepta: your partner in autophagy research

Abcepta has the most extensive collection of autophagy antibodies. From the hallmark autophagy antibody LC3 to the newest autophagy antibodies such as LAMP and APG1, Abcepta offers the most relevant, qualified antibodies for autophagy research. Its expanding collection includes hundreds of antibodies targeting autophagic proteins.

Autophagy is a catabolic trafficking pathway for bulk destruction/turnover of long-lived proteins and organelles via regulated lysosomal degradation. This process has recently been shown to be important in cancer, neurodegenerative diseases, and cardiovascular diseases. The process consists of sequential signaling, sequestration of cytoplasm, formation of a unique double membrane vesicle (autophagosome), targeting of the completed vesicle to the lysosome followed by docking and fusion, and breakdown. Detailed reviews of autophagy and its associated proteins can be found on page 4.

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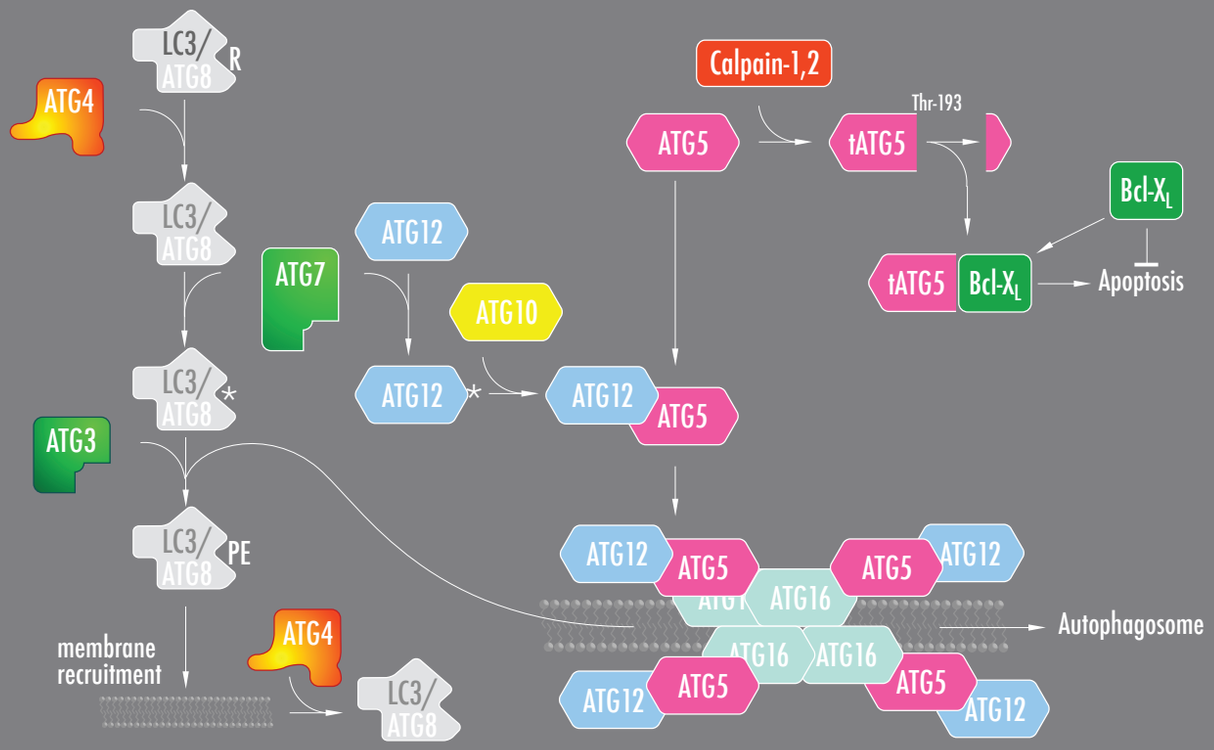
Autophagy Expansion & Apoptosis



Major proteins associated with autophagy and their interactions during the autophagy process.

The network above is from the Abcepta Macro Autophagy wall chart, an overview of the autophagic process. Request a FREE copy at www.abcepta.com.

Autophagy & Disease

DISEASE	ACTIVATION OF AUTOPHAGY	INACTIVATION OF AUTOPHAGY
Cancer		
Early stages	Blocks tumor growth.	Favors tumor growth. Makes cells unable to enter autophagic cell death after exposure to anticancer treatments.
Late stages	Favors survival of cells in low-vascularized tumors. Favors removal of damaged intracellular macromolecules after anticancer treatments.	Prevents survival of cells in low-vascularized tumors. Increases efficiency of anticancer treatments because damaged macromolecules cannot be eliminated.
Vacuolar myopathies	Promotes elimination of the cytosolic autophagic vacuoles and protein aggregates.	Results in the accumulation of autophagic vacuoles that weaken skeletal and cardiac muscles.
	If hyperactivated could result in muscle waste.	
Neurodegeneration		
Early stages	Favors removal of cytosolic protein aggregates.	Increases accumulation of cytosolic protein aggregates.
Late stages	Destroys irreversibly damaged neurons by autophagic cell death.	Results in accumulation of autophagic vacuoles that alter vesicular trafficking.
Axonal injury	Favors removal of neurotransmitter vesicles and damaged organelles.	Prevents removal of damaged organelles and neurotransmitters vesicles.
		Cytosolic release of neurotransmitters induces apoptosis.
	Provides energy and membranes for regeneration.	Slows down regeneration.
Infectious disease	Contributes to the elimination of bacterial and viral particles.	Offers a survival environment for the bacteria that are able to inhibit autophagosome maturation. Facilitates viral infection.

Occurrences that favor progression of the disease are noted in light gray boxes.

Autophagy, a Survival Guide

Two cellular instruction sets regulating survival/extinction at the individual level of shortlived proteins (the ubiquitin pathway) and at the grand level of the cell (apoptosis) have received great attention over the last fifteen years; the emerging details have advanced both fundamental research and therapeutic initiatives. Autophagy, the third leg on this stool, is a catabolic trafficking pathway for bulk destruction/ turnover of long-lived proteins and organelles via lysosomal degradaton. Newer techniques permitting the identification within the last decade of the full set of autophagy genes in yeast, the discovery of human homologues tied to specific disease states, and the definition of signaling pathways regulating autophagy have accelerated interest in elucidating the full molecular details of this important process.

General Mechanism

Autophagy consists of signaling, sequestration of cytoplasm, completion of vesicle formation, targeting of the completed vesicle to the lysosome/vacuole followed by docking and fusion, and breakdown. In higher eukaryotes, the lysosomal pathway of intracellular degradation is further partitioned into three distinct pathways: macroautophagy, chaperone-mediated autophagy, and microautophagy. Macrophagy is the subject of this brief review.

Macroautophagy begins with formation in the cytoplasm of the autophagosome, a double membrane vescular structure. The autophagosome engulfs cytoplasmic proteins, lipids, and damaged organelles such as mitochondria, endoplasmic reticulum, and ribosomes. The autophagosomal outer membrane fuses with the lysosome in mammalian cells to deliver the sequestered cargo. The inner membrane of the fused structure (autophagolysosome), dissolves, and digestion of interior contents by lysosomal hydrolytic enzymes generates nucleotides, amino acids, and free fatty acids that can be recycled to provide raw materials and energy to the cell.

Morphology

Upon autophagy induction, a membrane cisterna known as the isolation membrane appears and curves around part of the cytoplasm. Sealing of the membrane edges results in the double-membraned autophagosome, visible by electron microscopy. Autophagosomes then fuse with a lysosome, where degradation of the delivered material occurs. Isolation and autophagosome membranes differ from other cellular membranes in having few intramembrane proteins.

Autophagic Proteins/Signaling Pathways

In eukaryotic cells, autophagy occurs at constitutive low levels for housekeeping functions such as destruction of dysfunctional organelles. Upregulation occurs in the presence of external stressors (starvation, hormonal imbalance, oxidative stress, extreme temperature, and infection), and internal needs (generation of source materials for architectural remodeling, removal of protein aggregates). Autophagy is regulated by various kinases, phosphatases, and guanosine triphosphatases (GTPases). For example, mediators of phosphoinositide-3 (PI3) kinase signaling pathways and trimeric G proteins play roles in regulating formation of autophagosomes. Target Of Rapamycin (TOR) kinase, a predominant negative regulator of autophagy, is a significant target for cancer therapeutics. The eukaryotic Initiation Factor 2 (eIF2) kinase Gcn2 and its downstream target Gcn4, a transcriptional transactivator of autophagy genes, induce autophagy under conditions of cellular starvation. The PI3K/Akt signaling pathway inhibits autophagy in response to insulin-like and other growth factor signals. Downstream of TOR kinase, a range of autophagy proteins participate as follows:

- Protein serine/threonine kinase complex that relays upstream signals from TOR kinase: Atg1, Atg13, Atg17
- Lipid kinase signaling complex that engages vesicle nucleation: Atg6, Atg14, Vps34, and Vps15
- Ubiquitin-like conjugation pathways that facilitate vesicle expansion: LC3 (Atg8) and Atg12 networks
- Recycling pathway for removal of autophagy proteins from autophagosomes: Atg2, Atg9, Atg18

LC3 (rat microtubule-associated protein light chain 3), localizes in autophagosome membranes after processing, and is a classical autophagy marker. Following LC3 synthesis, Atg4 cleaves the C-terminus to produce cytosolic LC3-I. LC3-I is converted to LC3-II by Atg7 and Atg3. LC3-II is modified by phosphatidylethanolamine (PE) at the C-terminus and binds to autophagosomal membrane. The amount of LC3-II correlates with extent of autophagosome formation.

Beclin1, a Bcl-2 interacting protein, stimulates autophagy when overexpressed in mammalian cells. Beclin1 is monoallelically deleted in human breast and ovarian cancers, with reduced expression in those tumors. Beclin1 overexpression promotes autophagy and inhibits tumorigenesis in breast carcinoma cells; conversely, heterozygous disruption of Beclin1 promotes tumorigenesis in mice. Beclin1 associates with the human class III phosphatidylinositol 3- kinase (PI3K), hVps34. The lipid product of Vps34, PI(3)P, is required for autophagy, and also for assembly of proteins involved in endocytosis and trafficking of enzymes from the trans-Golgi network to the lysosomes. Beclin1 is required for hVps34 to function in autophagy.

Autophagy, Apoptosis, and Cell Death

Interplay between autophagy and apoptosis is complex, with autophagy acting either antagonistic, agonist, or independent of canonical programmed cell death via apoptosis.

Examples where autophagy precedes and may even trigger apoptosis include 1) a dramatic increase in autophagy followed by induction of apoptosis in primary sympathetic neurons deprived of neural growth factor, and 2) a similar effect in TNF-a-induced apoptosis of T-lymphoblastic leukemia cell lines, although in this case additional pro-death factors must be present in concert with autophagy to promote apoptosis. Autophagy inhibitors delay apoptosis while caspase inhibitors do not impair autophagy, indicating that autophagy may be a precedent to caspase-dependent cell death.

On the other hand, enhanced induction of apoptosis in autophagic deficient HT-29 colon carcinoma cells by sulindac sulfide highlights that autophagy under certain cases blocks the apoptotic pathway. Destruction of damaged mitochondria by autophagy may serve to delay signaling for the apoptotic cascade.

In other cases, cells switch between cell death via autophagy or apoptosis, providing a backup for selfexecution if the pathway of first choice is corrupted. Physical interactions between autophagic and apoptotic proteins (e. g. beclin1, BNIP3) suggest an intricate system of cell death regulation not previously appreciated.

References

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LC3: A Hallmark Autophagy Protein

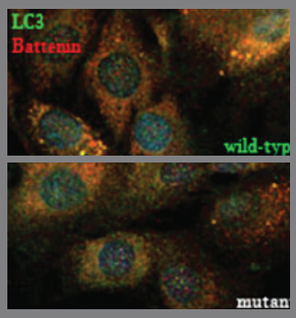

Fig. 1. Detection of LC3 in a Neuronal Mouse Model. LC3 and the CLN3-encoded protein, battenin, co-immunostaining. Wild-type and mutant cerebellar cells were co-stained with #AP1801a, anti LC3-antibody (Red) and a monoclonal anti-battenin antibody (Golabek et al., 2000) (Green), and images were captured by confocal microscopy. Note the numerous larger vesicular structures, typical of mature autophagic vacuoles, stained with both the LC3 and battenin antibodies in wild-type cells, which are rare in mutant cells. Blue=DAPI.

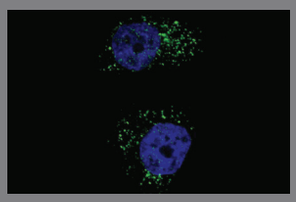

Fig. 2. LC3 antibodies for IF staining. Immunofluorescent (IF) staining of U251 cells treated with Chloroquine using LC3 monoclonal (Cat. #AM1800a).

Autophagy in the Research Spotlight

LC3 is currently the standard marker of autophagosomes. Tracking the conversion of LC3-I to LC3-II, occurring via conjugation to phosphatidylethanolamine, is an indicator of autophagic activity. Due to the transient nature of autophagosomal structures, the ratio of LC3-II to LC3-I can be used to estimate the course of auotophagic activity over time. Under autophagic conditions due to external or internal stressors, western blotting of LC3 usually detects two bands: cytosolic LC3-I (18 kDa) and autophagosome associated LC3-II (16 kDa).

LC3 Monoclonal and Polyclonal Antibodies

Abcepta LC3 antibodies are specific for the hallmark autophagy protein LC3, which associates with autophagosomal vacuoles (autophagosomes) formed upon induction of autophagy. These antibodies have proven to be a successful autophagy detection tool (Fig. 1). LC3 polyclonal (Cat. #AP1801a) and LC3 monoclonal (Cat. #AM1800a) are validated for use in western blotting, immunoprecipitation and immunofluoresence (Fig. 2).

Autophagy Antibodies with the Right Immunogenic Design

Our expanding autophagy line now includes over 150 novel autophagy antibodies directed against human homologs of a series of proteins identified in the autophagy pathway including polyclonal and monoclonal antibodies against a range of epitopes spanning LC3, a marker for the autophagic process. These antibodies are characterized by immunoblot and cell staining of a range of human and rodent lysates.

Abcepta has numerous citations for its LC3 products.

Please visit www.abcepta.com for a full listing.

Select Citations:

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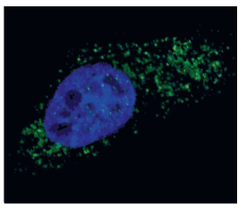
Autophagy Hallmark Target: LC3

Abcepta has developed one of the most validated set of LC3 antibodies available. LC3 is a major marker of the autophagy process, and Abcepta's premium LC3 selection serves as a vital tool for autophagy research.

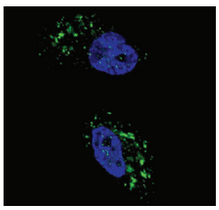
	CAT. #	ANTIBODY	HOST	ISO	APP.	REACT.
1	AP1801a	LC3 (APG8A) (N-term)	Rb	plg	WB,IHC,IF	H,M,R
2	AP1802a	LC3 (APG8B) (N-term)	Rb	plg	WB,IHC,IF	H,M,R
3	AP1800a	LC3 (APG8A)	Rb	plg	WB,IF	H,M
4	AP1801b	LC3 (APG8A) (P45)	Rb	plg	WB,IHC,IF	H
5	AP1805a	Cleaved LC3A	Rb	plg	WB, IHC, ICC, IF	H,M
6	AP3301a	Phospho-LC3C-S12	Rb	plg	WB,DB	H

1

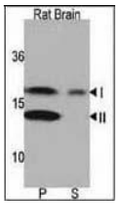


IF analysis of U251 cells treated with Chloroquine using the LC3 antibody.

2

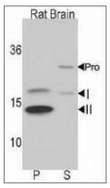


IF analysis of U251 cells treated with Chloroquine using the LC3 antibody.

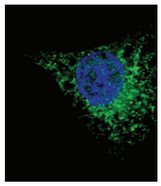


WB analysis of rat brain tissue lysate using the LC3 antibody.

3

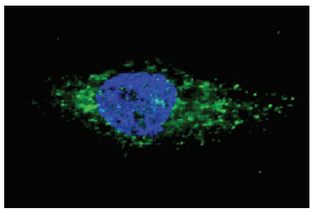


WB analysis of aLC3 antibody in rat brain lysate. Both non-lipidated (arrow, I) and lipidated LC3 (APG8b) (arrow, II) were detected in membrane fraction (P) whereas pro-LC3 (APG8b) and non-lipidated LC3 ((APG8b) were detected in soluble fraction (S).



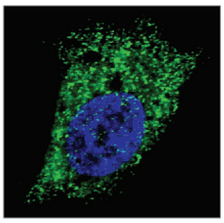
IF analysis of U251 cells treated with Chloroquine using the LC3 antibody.

4



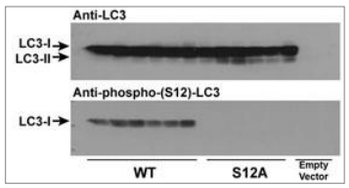
IF analysis of U251 cells treated with Chloroquine using the LC3 antibody.

5



IF analysis of U251 cells using the LC3 antibody.

6



WB analysis of CHO cell line lysates using the LC3 antibody. Native LC3 and LC3 S12 mutant vectors were transfected to show phosphorylation specificity.

Autophagy Antibodies

APG1

	CAT. #	ANTIBODY	HOST	ISO	APP.	REACT.
	AP19250c	APG1 (ULK1) (Center S317)	Rb	plg	WB	H
1	AP8104b	APG1 (ULK1) (Center)	Rb	plg	WB,IF	H
	AP19265c	APG1 (ULK1) (Center S556)	Rb	plg	WB	H
	AP3804a	APG1 (pULK1) (S556)	Rb	plg	IF, DB	H

APG3

	CAT. #	ANTIBODY	HOST	ISO	APP.	REACT.
	AP1807a	ATG3 (N-term)	Rb	plg	WB	H
	AP1807b	ATG3 (C-term)	Rb	plg	WB	H
	AP1807c	ATG3 (C-term K183)	Rb	plg	WB	H,M
2	AP1807e	ATG3	Rb	plg	WB,IHC,IF	H,M

APG4

	CAT. #	ANTIBODY	HOST	ISO	APP.	REACT.
	AP1808a	ATG4A	Rb	plg	WB,IHC	H,M
	AP1808b	ATG4A	Rb	plg	WB,IHC	H
	AP1808c	ATG4A	Rb	plg	WB,IHC	H,M
	AP1809a	ATG4B (N-term)	Rb	plg	WB,IHC	H,M
3	AP1809b	ATG4B	Rb	plg	WB,IHC,IF	H,M
	AP1809c	ATG4B (C-term)	Rb	plg	WB,IHC	H
4	AP1809d	ATG4B	Rb	plg	WB,IHC	H
	AP1810a	ATG4C (N-term)	Rb	plg	WB,IHC	H,M
	AP1810b	ATG4C (Center)	Rb	plg	WB,IHC	H
	AP1810c	ATG4C (C-term)	Rb	plg	WB,IHC	H,M
	AP1810d	ATG4C	Rb	plg	WB,IHC	H

APG4 (cont)

	CAT. #	ANTIBODY	HOST	ISO	APP.	REACT.
	AP1811a	ATG4D (N-term)	Rb	plg	WB,IHC	H,M
	AP1811b	ATG4D (Center)	Rb	plg	WB,IHC	H,M
5	AP1811c	ATG4D (C-term)	Rb	plg	WB,IHC,IF	H
	AP1811e	ATG4D (C-term)	Rb	plg	WB,IHC	H
	AP1811f	ATG4D (center)	Rb	plg	WB	H

APG5

	CAT. #	ANTIBODY	HOST	ISO	APP.	REACT.
	AP1812a	ATG5 (N-term)	Rb	plg	WB,IHC	H,M
6	AP1812b	ATG5 (C-term)	Rb	plg	WB,IHC	H,M

APG7 & APG9

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	AP1813a	ATG7 (N-term)	Rb	plg	WB,IHC	H,M
	AP1813b	ATG7 (Center)	Rb	plg	WB,IHC	H,M
	AP1813c	ATG7	Rb	plg	WB,IHC	H,M
7	AP1813d	ATG7 (C-term)	Rb	plg	WB,IHC	H
	AP1813e	ATG7	Rb	plg	WB,IHC	H
	AP1814a	ATG9A (N-term)	Rb	plg	WB,IHC	H,M
8	AP1814b	ATG9A (Center)	Rb	plg	WB,IHC	H,M
	AP1814c	ATG9A (C-term)	Rb	plg	WB,IHC	H
	AP1814d	ATG9A	Rb	plg	WB,IHC	H
	AP1814e	ATG9A	Rb	plg	WB,IHC	H
	AP1814g	ATG9A	Rb	plg	WB,IHC	H

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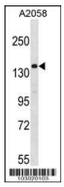

WB analysis of A2058 cell lysate using the APG1 antibody.

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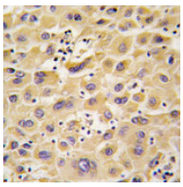
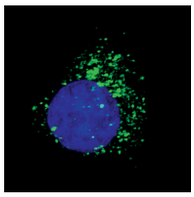


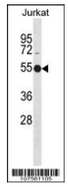
IHC analysis of human hepatocarcinoma tissue stained with ATG3 antibody.

3



IF analysis of U251 cells treated with Chloroquine using the ATG4B antibody.

4



WB analysis of Jurkat cell line lysate with the APG4C antibody.

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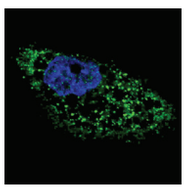
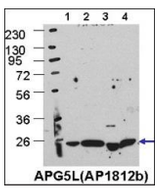


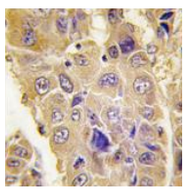
IF analysis of U251 cells treated with Chloroquine using the ATG4D antibody.

6



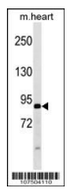
WB analysis of Cos7, HEK293, MEF, and HeLa cell lysates using the ATG5 antibody.

7



IHC analysis of human hepatocarcinoma tissue stained with the ATG7 antibody.

8



WB analysis of mouse heart tissue lysate using the ATG9A antibody.

APG12

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	AP1151a	ATG12 (N-term)	Rb	plg	WB,IF	H,M
9	AP1816a	ATG12 (N-term)	Rb	plg	WB,IHC,IF	H
	AP1816c	ATG12	Rb	plg	WB,IHC	H,M

APG16

	CAT. #	ANTIBODY	HOST	ISO	APP.	REACT.
	AM1817a	ATG16L1 (Ascites)	Ms	IgG1	WB	H
	AP1817a	ATG16L1	Ms	IgG1	WB,IHC	H
	AP1817b	ATG16L	Rb	plg	WB,IHC	H,M
	AP1817c	ATG16L (C-term)	Rb	plg	WB,IHC	H
10	AP1817d	ATG16L	Rb	plg	WB	H

AMBRA, CAMKV, DRAM, GABARAP & PIST

	CAT. #	ANTIBODY	HOST	ISO	APP.	REACT.
	AP1821a	GABARAP (N-term)	Rb	plg	WB,IHC,IF	H
	AP1825b	DRAM (C-term)	Rb	plg	WB,IHC	H
	AP1826a	AMBRA1 (N-term)	Rb	plg	WB,IHC	H
	AP1954a	EIF4E (N-term)	Rb	plg	WB,IHC	H
	AP7118d	CAMKV (N-term D10)	Rb	plg	WB,IHC	H
	AP7250c	MAPK1 (Center)	Rb	plg	WB,IHC,FC	H
11	AP7262b	PIST (C-term)	Rb	plg	WB,IHC	H,M
	AP7262c	PIST (Center)	Rb	plg	WB,IHC	H

Beclin1 (APG6)

	CAT. #	ANTIBODY	HOST	ISO	APP.	REACT.
12	AM1818a	Beclin1 (Ascites)	Ms	IgG1	WB,IHC	H
13	AP1818a	Beclin1	Rb	plg	WB,IHC	H,M
	AP1818b	Beclin1	Rb	plg	WB,IHC,FC	H,M
	AP1818c	Beclin1 (C-term)	Rb	plg	WB,IHC	H,M

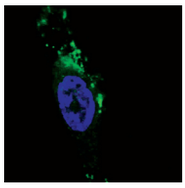
LAMP

	CAT. #	ANTIBODY	HOST	ISO	APP.	REACT.
	AP1823a	LAMP1 (N-term)	Rb	plg	WB,IHC	H,M
	AP1823b	LAMP1 (C-term)	Rb	plg	WB	H
	AP1824b	LAMP2 (C-term)	Rb	plg	WB	H
14	AP1824c	LAMP2 (Center)	Rb	plg	WB	H
	AP1827a	LAMP3 (N-term)	Rb	plg	WB	H

RAB24 & RGS19

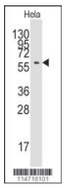
	CAT. #	ANTIBODY	HOST	ISO	APP.	REACT.
	AP1819a	RAB24 (N-term)	Rb	plg	WB,IHC	H,M
	AP1819b	RAB24 (Center)	Rb	plg	WB,IHC	H,M
	AP1819c	RAB24 (C-term)	Rb	plg	WB,IHC	H,M
	AP1819d	RAB24 (Y172)	Rb	plg	WB,IHC	H
	AP1819e	RAB24 (Y17)	Rb	plg	WB,IHC	H
	AP1819f	RAB24 (S95)	Rb	plg	WB,IHC	H
15	AP1820b	RGS19 (Center)	Rb	plg	WB,IHC, IF	H, M
	AP1820e	RGS19 (Y143)	Rb	plg	WB,IHC	H
	AP1820f	RGS19 (S151)	Rb	plg	WB,IHC	H,M

9



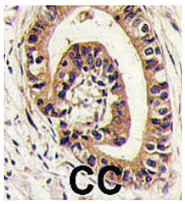
IF analysis of U251 cells treated with Chloroquine using the ATG12 antibody.

10



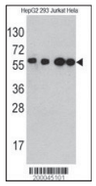
WB analysis of HeLa cell line lysate with the ATG16L antibody.

11



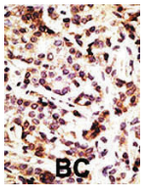
IHC analysis of human colon carcinoma tissue stained with the PIST antibody.

12



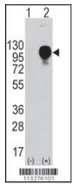
WB analysis of HepG2, 293, Jurkat, and HeLa cell line lysates using the Beclin1 antibody.

13



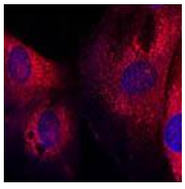
IHC analysis of human breast carcinoma tissue stained with the Beclin1 antibody.

14



WB analysis of non-transfected and transfected 293 cell lysates using the LAMP2 antibody.

15



IF analysis of mouse brain cell lysate using the RGS19 antibody.

Autophagy Antibodies (cont.)

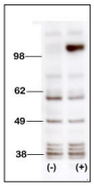
PI3KC3

	CAT. #	ANTIBODY	HOST	ISO	APP.	REACT.
	AP1851b	PI3KC3 (C-term E785)	Rb	plg	WB,IHC	H
16	AP1851c	PI3KC3 (Center K269)	Rb	plg	WB,IHC	H
	AP1851d	PI3KC3 (Center E501)	Rb	plg	WB,IHC	H
	AP1851e	PI3KC3 (S34)	Rb	plg	WB,IHC	H
	AP1851g	PI3KC3 (S164)	Rb	plg	WB,IHC	H
	AP1851h	PI3KC3 (S425)	Rb	plg	WB,IHC	H
	AP1851j	PI3KC3 (S851)	Rb	plg	WB,IHC	H
	AP8014a	PI3KC3 (N-term)	Rb	plg	WB,IHC	H
	AP8016c	PI3KCA (Center)	Rb	plg	WB,IHC	H
17	AP8023d	PI3KR1 (N-term L11)	Rb	plg	WB,IHC	H

SQSTM1 (p62) & UVRAG

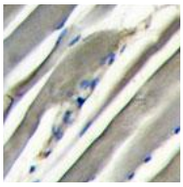
	CAT. #	ANTIBODY	HOST	ISO	APP.	REACT.
18	AP1850b	UVRAG (C-term)	Rb	plg	WB,IHC,IF	H,M
	AP1850c	UVRAG (Center)	Rb	plg	WB,IHC	H,M
	AP1850d	UVRAG (L133)	Rb	plg	WB,IF	H,M
	AP1850e	UVRAG (C-term L555)	Rb	plg	WB	H
19	AP2183b	SQSTM1 (p62) (C-term)	Rb	plg	WB,IHC,IF	H
	AP7239d	STK11 (LKB1) (N-term V34)	Rb	plg	WB,IF,FC	H
	AP8115a	ULK3 (N-term)	Rb	plg	WB,IHC	H

16



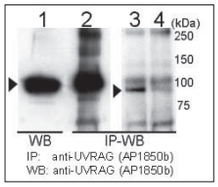
WB analysis of HEK-293 cell lysates using the PI3KC3 antibody.

17



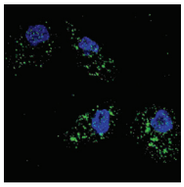
IHC analysis of human skeletal muscle tissue stained with the PI3KR1 antibody.

18



WB and IP analysis of 293T cell lysates using the UVRAG antibody.

19



IF analysis of U251 cells treated with Chloroquine using the SQSTM1 (p62) antibody.

Additional Autophagy Products

Blocking Peptides:

Blocking peptides are available for all Abcepta autophagy antibodies. Check the technical data sheet of any autophagy antibody at www.abcepta.com to locate the companion blocking peptide.

Synthetic Peptides:

Abcepta offers a host of synthetic peptides useful in autophagy research. A selection of synthetic peptides are listed here. For a full list, please visit the Abcepta website.

CAT. #	PEPTIDE TARGET	SPECIFICITY
SP1046a	Inducer Control: Beclin1 BH3 domain mutant (Cell Permeable, Human, Mt1-I116a)	H
SP1046b	Inducer Control: Beclin1 BH3 domain mutant (Cell Permeable, Human, Mt2-F123a)	H
SP1047a	Inducer Control: Beclin1 BH3 domain mutant (Human, Mt1-I116a)	H
SP1047b	Inducer Control: Beclin1 BH3 domain mutant (Human, Mt2-F123a)	H
SP1040a	Inducer: Beclin1 BH3 domain (Cell Permeable, Human, Wt)	H
SP1043a	Inducer: Beclin1 BH3 domain (Cell Permeable, Zebrafish, Wt)	H
SP1041a	Inducer: Beclin1 BH3 domain (Human, Wt)	H
SP1044a	Inducer: Beclin1 BH3 domain (Zebrafish, Wt)	H

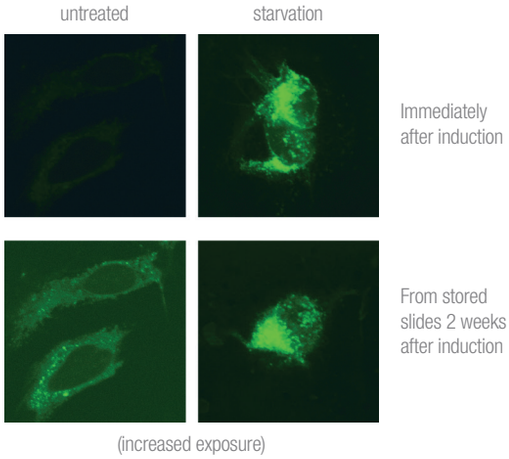
Transfected Cell Lysates:

Transfected cell lysates are available for a number of Abcepta autophagy antibodies. Check the technical data sheet of any autophagy antibody at www.abcepta.com to locate the companion transfected cell lysate.

AutoDOT:

The new autophagy visualization dye! Superior to traditional monodansyl cadaverine staining.

- Faster penetration
- Higher sensitivity/greater signal endurance on stored slides
- Greater resistance to acid



MDHx staining in cerebellar cells

Legends

APPLICATION (APP.)

DB = Dot Blot
FC = Flow Cytometry
ICC = Immunocytochemistry
IHC = Immunohistochemistry
IF = Immunofluorescence
WB = Western Blot

REACTIVITY (REACT.)

H = Human
M = Mouse
R = Rat

ANTIBODY

IgG1= Immunoglobulin 1
plg = Polyclonal immunoglobulin

HOST

Ms = Mouse
Rb = Rabbit

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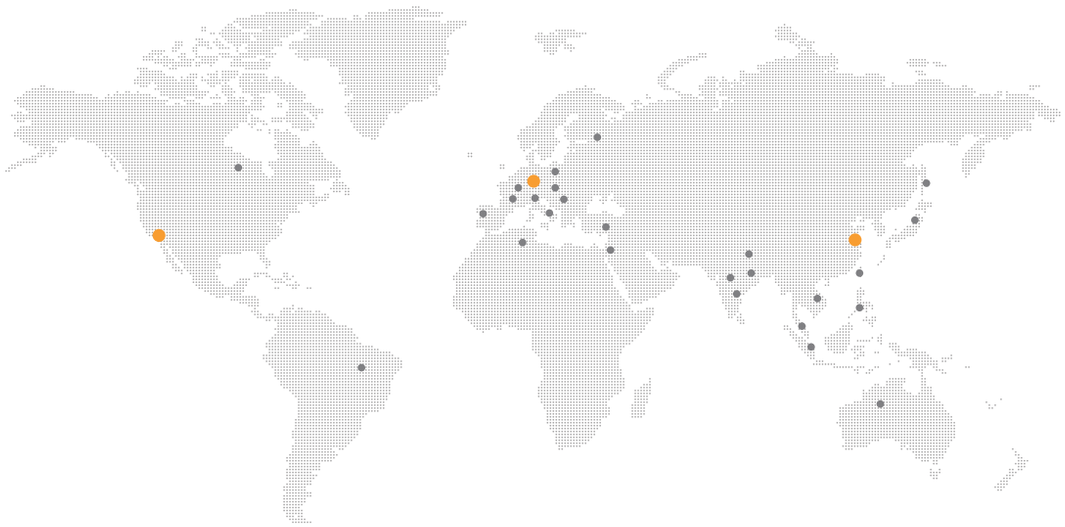
Abcepta truly has a global reach. With offices in the US and China, and a leading network of distributors in Europe and around the world, our products can be at your bench as fast as the next day. Review our list below to find the distributor for your region and take advantage of the products and services that Abcepta has to offer.



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Legends

VALIDATION

ChIP = Chromatin Immunoprecipitation
DB = Phospho-specific dot blot
E = Elisa
IF = Immunofluorescence
IHC = Immunohistochemistry
IP= Immunoprecipitation
WB = Western blot

SPECIFICITY

Gt = Goat
H = Human
M = Mouse
R = Rat

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Legends

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IP= Immunoprecipitation
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