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A comprehensive glossary of autophagy-related molecules and processes (2nd edition)

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The study of autophagy is rapidly expanding, and our knowledge of the molecular mechanism and its connections to a wide range of physiological processes has increased substantially in the past decade. The vocabulary associated with autophagy has grown concomitantly. In fact, it is difficult for readers—even those who work in the field—to keep up with the ever-expanding terminology associated with the various autophagy-related processes. Accordingly, we have developed a comprehensive glossary of autophagy-related terms that is meant to provide a quick reference for researchers who need a brief reminder of the regulatory effects of transcription factors and chemical agents that induce or inhibit autophagy, the function of the autophagy-related proteins, and the roles of accessory components and structures that are associated with autophagy.

Introduction

One meaning for the term "comprehensive" is to "cover completely or broadly." However, the rapid expansion in the field of autophagy is such that this glossary will be out of date as soon as it is published. On the other hand, "comprehensive" can also indicate, "marked by or showing extensive understanding." If we consider that our knowledge of autophagy has indeed expanded tremendously over the past decade, it may be appropriate to suggest that we are developing an extensive understanding of this subject. Of course we fully expect that our current understanding will undergo substantial change in the coming months and years, and that even the breadth of our knowledge may amount to little more than the tip of the autophagy iceberg. Nonetheless, we think it will be helpful to researchers to catalog the information we have about autophagy in an easily accessible format.

Accordingly, we present here the 2nd edition of a comprehensive glossary for autophagy, which is expanded by more than 62%. The definitions are purposely short; our intent is that the reader can gain a quick, rather than in-depth, understanding of the function or meaning of the relevant word. In addition, the references generally cite the first use of particular terms, or the first cloning of a gene and description of the corresponding protein, or an appropriate review article; due to space considerations, this list is far from being inclusive. We encourage readers to examine the primary literature for additional details about the process of autophagy and the components described herein.

3-MA (3-methyladenine): An inhibitor of class I and class III PtdIns 3-kinase, which results in autophagy inhibition due to suppression of class III PtdIns 3-kinase,[1] but may under some conditions show the opposite effect.[2]

11'-deoxyverticillin A (C42): An epipolythiodioxopiperazine fungal secondary metabolite that is used as an anticancer drug; it triggers apoptotic and necrotic cell death, and enhances macroautophagy through the action of PARP-1 and RIP-1.[3]

12-ylation: The modification of substrates by covalent conjugation to Atg12, first used to describe the autocatalytic conjugation of Atg12 to Atg3.[4]

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14-3-3ζ: A member of the 14-3-3 family of proteins that inhibits macroautophagy; direct interaction with hVps34 negatively regulates kinase activity, and this interaction is disrupted by starvation or C_2-ceramide.[5]

A20: An E3 ubiquitin ligase that also functions as a deubiquitinating enzyme that removes K63-linked ubiquitin from Beclin 1, thus limiting macroautophagy induction in response to Toll-like receptor signaling.[6]

ABT737: A BH3 mimetic that competitively disrupts the interaction between Beclin 1 and Bcl-2 or Bcl-X_L, thus inducing macroautophagy.[7]

Acinus: A protein that in Drosophila regulates both endocytosis and macroautophagy; the *acn* mutant is defective in autophagosome maturation, whereas overexpression of Acn leads to excessive macroautophagy.[8]

AEG-1 (astrocyte-elevated gene-1): This gene encodes an oncogenic protein that induces noncanonical (Beclin 1- and class III PtdIns 3-kinase-independent) macroautophagy as a cytoprotective mechanism.[9]

AEN/ISG20L1 (apoptosis-enhancing nuclease/interferon stimulated exonuclease gene 20 kDa-like 1): A protein that localizes to nucleolar and perinucleolar regions of the nucleus, which regulates macroautophagy associated with genotoxic stress; transcription of ISG20L1 is regulated by p53 family members.[10]

aggrephagy: The selective removal of aggregates by macroautophagy.[11]

AGS3 (activator of G protein signaling 3): A guanine nucleotide dissociation inhibitor for $G\alpha_{i3}$ that promotes macroautophagy by keeping $G\alpha_{i3}$ in an inactive state.[12] AGS3 directly binds LC3 and recruits $G\alpha_{i3}$ to phagophores or autophagosomes under starvation conditions to promote autophagosome biogenesis and/or maturation. See also $G\alpha_{i3}$.

AICAR: Cell permeable nucleotide analogue, aminoimidazole-4-carboxamide riboside that is an activator of AMP kinase; inhibits autophagy[14] through mechanisms that are not related to its effect on AMPK.[14,16]

AIM (Atg8-family Interacting Motif):[17] See WXXL and LIR/LRS.

Alfy (autophagy-linked FYVE protein): An adaptor that targets cytosolic protein aggregates for autophagic degradation.[18] Alfy interacts directly with Atg5 [19] and with p62.[20]

ALR (autophagic lysosome reformation): A self-regulating process in which the macroautophagic generation of nutrients reactivates mTOR, suppresses macroautophagy and allows for the regeneration of lysosomes that were consumed as autolysosomes.[21]

Ambra1 (activating molecule in Beclin 1-regulated autophagy): A positive regulator of macroautophagy. Ambra1 binds Beclin 1 and modulates its activity.[22]

Amphisome: Intermediate compartment formed by the fusion of an autophagosome with an endosome (this compartment can be considered a type of autophagic vacuole and may be equivalent to a late autophagosome, and as such has a single limiting membrane); this compartment has not yet fused with a lysosome.[23]

AMPK (AMP-activated protein kinase): A sensor of energy level that is activated by an increase in the AMP/ATP ratio via the LKB1 kinase. Phosphorylates the mTORC1 subunit Raptor to cause induction of macroautophagy. AMPK also activates the TSC1/2 complex (thus inhibiting Rheb), and binds and directly phosphorylates (and activates) ULK1 as part of the ULK1 kinase complex, which includes mATG13 and FIP200.[24,25] The yeast homolog of AMPK is Snf1.[26,27] Conversely, ULK1 can phosphorylate AMPK through a negative feedback loop.[27]

APMA (autophagic macrophage activation): A collection of autophagy-related processes in cells of the reticulo-endothelial system. APMA includes (1) convergence of phagocytosis and the autophagic machinery, (2) enhanced microbicidal properties of autolysosomes in comparison to standard phagolysosomes, (3) autophagic modulation of pathogen recognition receptor (PRR) signaling, (4) cooperation between immunity-related GTPases and autophagy or Atg proteins in attacking parasitophorus vacuoles and (5) enhanced antigen presentation. APMA is thus recognized as a complex outcome of autophagy stimulation in macrophages, representing a unique composite process that brings about a heightened state of immunological activation.[28]

ARD1 (arrest-defective protein 1): A protein that interacts with and stabilizes TSC2 by acetylation, resulting in repression of mTOR and induction of macroautophagy.[29]

Atg (autophagy related): Abbreviation used for most of the components of the protein machinery that are involved in selective and nonselective macroautophagy and in selective microautophagy.[30]

Atg1: A serine/threonine protein kinase that functions in recruitment and release of other Atg proteins from the PAS.[31] The functional homologs in higher eukaryotes are ULK1 and ULK2.

Atg2: A protein that acts along with Atg18 to mediate the retrograde movement of Atg9 away from the PAS.[32,33]

Atg3: A ubiquitin-conjugating enzyme (E2) analog that conjugates Atg8/LC3 to phosphatidylethanolamine (PE) after activation of the C-terminal residue by Atg7.[34,35] ATG3 can also be conjugated to ATG12 in higher eukaryotes.[4] See also 12-ylation.

Atg4: A cysteine protease that processes Atg8/LC3 by removing the amino acid residue(s) that are located on the C-terminal side of what will become the ultimate glycine. Atg4 also removes PE from Atg8/LC3 in a step referred to as "deconjugation."[36] Mammals have four Atg4 proteins (Atg4A–D), but Atg4B appears to be the most relevant for macroautophagy and has the broadest range of activity for all of the Atg8 homologs.[37,38]

Atg5: A protein containing ubiquitin folds that is part of the Atg12–Atg5-Atg16 complex, which acts in part as an E3 ligase for Atg8/LC3–PE conjugation.[39]

Atg6 (Vps30): A component of the class III PtdIns 3-kinase complex. Atg6 forms part of two distinct complexes (I and II) that are required for the Atg and Vps pathways, respectively. See also Beclin 1.[40]

Atg7: A ubiquitin-activating (E1) enzyme homolog that activates both Atg8/LC3 and Atg12 in an ATP-dependent process.[41,42]

Atg8: A ubiquitin-like protein that is conjugated to PE; involved in cargo recruitment into, and biogenesis of, autophagosomes. Autophagosomal size is regulated by the amount of Atg8. Since Atg8 is selectively enclosed into autophagosomes, its breakdown allows measurement of the autophagic rate.

Mammals have several Atg8 homologs comprising the LC3 and GABARAP subfamilies, which are also involved in autophagosome formation.[43-45]

Atg9: A transmembrane protein that may act as a lipid carrier for expansion of the phagophore. In mammalian cells, mAtg9A localizes to the trans-Golgi network and endosomes, whereas in fungi this protein localizes in part to reservoirs (termed the tubulovesicular cluster) that are localized near the mitochondria, and to the PAS.[46,47] While mammalian mAtg9A is ubiquitously expressed, mAtg9B is almost exclusively expressed in the placenta and pituitary gland.[48]

Atg10: A ubiquitin-conjugating (E2) enzyme analog that conjugates Atg12 to Atg5.[49]

Atg11: A fungal scaffold protein that acts in selective types of macroautophagy including the Cvt pathway, mitophagy and pexophagy. Atg11 binds Atg19, PpAtg30 and Atg32 as part of its role in specific cargo recognition. It also binds Atg9 and is needed for its movement to the PAS.[50]

Atg12: A ubiquitin-like protein that modifies an internal lysine of Atg5 by covalently binding via its C-terminal glycine.[39] In mouse and human cells, ATG12 also forms a covalent bond with ATG3, and this conjugation event plays a role in mitochondrial homeostasis.[4] See also 12-ylation.

Atg13: A component of the Atg1 complex that is needed for Atg1 kinase activity. Atg13 is highly phosphorylated in a PKA- and Tor-dependent manner in rich medium conditions. During starvation-induced macroautophagy in yeast, Atg13 is partially dephosphorylated. In mammalian cells, at least Tor and Ulk1/Atg1 phsophorylate Atg13. The decreased phosphorylation of Atg13 that results from Tor inhibition is partly offset by the Ulk1-dependent phosphorylation that occurs upon Ulk1 activation.[51,52]

Atg14: A component of the class III PtdIns 3-kinase complex that is necessary for the complex to function in macroautophagy.[40]

Atg15: A vacuolar protein that contains a lipase/esterase active site motif and is needed for the breakdown of autophagic and Cvt bodies within the vacuole lumen.[53,54]

Atg16: A component of the Atg12–Atg5-Atg16 complex. Atg16 dimerizes to form a large complex.[55] There are two mammalian homologs, Atg16L1 and Atg16L2. The former is one of the risk alleles associated with Crohn disease.[56]

Atg17: A yeast protein that is part of the Atg1 kinase complex. Atg17 is not essential for macroautophagy, but modulates the magnitude of the response; smaller autophagosomes are formed in the absence of Atg17.[57,58]

Atg18: A protein that binds to PtdIns(3)P (and PtdIns(3,5)P_2 via its WD40 β-propeller domain. Atg18 functions along with Atg2 in the retrograde movement of Atg9. Atg18 has additional nonautophagic functions, such as in retrograde transport from the vacuole to the Golgi complex, and in the regulation of PtdIns(3,5)P_2 synthesis; the latter function affects the vacuole's role in osmoregulation.[59] See also WIPI.

Atg19: A receptor for the Cvt pathway that binds Atg11, Atg8 and the propeptide of precursor aminopeptidase I. Atg19 is also a receptor for α-mannosidase, another Cvt pathway cargo.[60,61]

Atg20: A PtdIns(3)P binding protein that is part of the Atg1 kinase complex.[62]

Atg21: A PtdIns(3)P binding protein that is a homolog of, and partially redundant with, Atg18.[63] See also WIPI.

Atg22: A vacuolar amino acid permease that is required for efflux after autophagic breakdown of proteins.[64,65]

Atg23: A yeast protein that transits with Atg9.[66-68]

Atg24 (Snx4): A PtdIns(3)P binding protein that is part of the Atg1 kinase complex.[62]

Atg25: A coiled-coil protein required for macropexophagy in *Hansenula polymorpha*.[69]

Atg26: A sterol glucosyltransferase that is required for micro- and macropexophagy in *Pichia pastoris*, but not in *Saccharomyces cerevisiae*.[70,71]

Atg27: An integral membrane protein that is required for the movement of Atg9 to the PAS. The absence of Atg27 results in a reduced number of autophagosomes under autophagy-inducing conditions.[72]

Atg28: A coiled-coil protein involved in micro- and macropexophagy in *P. pastoris*.[73]

Atg29: A protein required for efficient nonspecific macroautophagy in fungi. Part of an Atg17-Atg31-Atg29 complex that functions at the PAS for protein recruitment.[74]

Atg30: A protein required for the recognition of peroxisomes during micro- and macropexophagy in *P. pastoris*. It binds the peroxin PpPex14 and the selective autophagy adaptor protein PpAtg11.[75]

Atg31: A protein required for nonspecific macroautophagy in fungi. Part of an Atg17-Atg31-Atg29 complex that functions at the PAS for protein recruitment.[76]

Atg32: A mitochondrial outer membrane protein that is required for mitophagy in fungi. Atg32 binds Atg8 and Atg11 preferentially during mitophagy-inducing conditions.[77,78]

Atg33: A mitochondrial outer membrane protein that is required for mitophagy in fungi.[79]

Atg34: A protein that functions as a receptor for import of α-mannosidase during macroautophagy (i.e., under starvation conditions) in yeast.[80] This protein was initially referred to as Atg19-B based on predictions from in silico studies.[81] See also Atg19.

Atg35: The Atg35 protein relocates to the perinuclear structure (PNS) and specifically regulates MIPA formation during micropexophagy in *P. Pastoris*; the *atg35*Δ mutant is able to form pexophagosomes during macropexophagy.[82]

Atg101: An Atg13-binding protein conserved in various eukaryotes but not in *S. cerevisiae*. Forms a stable complex with ULK1/2-Atg13-FIP200 (i.e., not nutrient-dependent), required for macroautophagy and localizes to the phagophore.[83,84]

ATM (ataxia-telangiectasia mutated): A protein kinase that activates TSC2 via the LKB1-AMPK cascade in response to elevated ROS, resulting in inhibition of mTOR and activation of macroautophagy.[85]

autolysosome: A degradative compartment formed by the fusion of an autophagosome (or initial autophagic vacuole/AVi) or amphisome with a lysosome (also called degradative autophagic vacuole/AVd or autophagolysosome). Upon completion of degradation this compartment again becomes a lysosome or residual body.[22,86]

autophagic body: The inner membrane-bound structure of the autophagosome that is released into the vacuolar lumen following fusion of the autophagosome with the vacuole limiting membrane. In *S. cerevisiae*, autophagic bodies can be stabilized by the addition of the proteinase B inhibitor PMSF to the medium. Visualization of the accumulating autophagic bodies by Nomarski optics is a convenient method to follow macroautophagy.[87]

autophagic cell death: A historically ambiguous term describing cell death with morphological features of increased autophagic vacuoles. This term is best reserved for cell death contexts in which specific molecular methods, rather than only pharmacological or correlative methods, are used to demonstrate increased cell survival following inhibition of autophagy.

autophagic stress: A pathological situation in which induction of autophagy exceeds the cellular capacity to complete lysosomal degradation and recycling of constituents; may involve a combination of bioenergetics, acidification and microtubule-dependent trafficking deficits, to which neurons may be particularly vulnerable.[88]

autophagist: A researcher working in the field of autophagy.

autophagosome: A cytosolic membrane-bound compartment denoted by a limiting double membrane (also referred to as initial autophagic vacuole, AVi or early autophagosome). The early autophagosome contains cytoplasmic inclusions and organelles that are morphologically unchanged because the compartment has not fused with a lysosome and lacks proteolytic enzymes. Although in most cases the term autophagosome refers to a double-membrane vesicle, the late autophagosome may also appear to have a single membrane (also referred to as an intermediate autophagic vacuole, AVi/d).[22,86]

autophagy: This term summarizes all processes in which intracellular material is degraded within the lysosome/vacuole and where the macromolecular constituents are recycled.

***Autophagy*:** A journal devoted to research in the field of autophagy (www.landesbioscience.com/journals/autophagy/). Also, a forum (www.landesbioscience.com/journals/autophagy/forum/?nocache = 1887078014) where you can obtain practical information from active researchers regarding the usefulness of commercially available reagents, such as antibodies to specific autophagy-related proteins.

bafilomycin A_1 (BA/BAF): An inhibitor of the vacuolar (V)-type ATPase that alters the pH and membrane potential of acidic compartments, which ultimately results in a block in fusion of autophagosomes with lysosomes, thus preventing the maturation of autophagosomes into autolysosomes.[89,90] Note that the abbreviation for bafilomycin A_1 is not "BFA," as the latter is the standard abbreviation for brefeldin A.

BAG3 (Bcl-2-associated athanogene 3): A stress-induced co-chaperone that interacts with dynein; BAG3 directs Hsp70 misfolded protein substrates to dynein, which targets them to aggresomes, leading to their selective degradation by macroautophagy via a ubiquitin-independent mechanism.[91]

Barkor (Beclin 1-associated autophagy related key regulator): Also known as Atg14L, the mammalian homolog of yeast Atg14, a component of the class III PtdIns 3-kinase complex.[92]

basal autophagy: Constitutive autophagic degradation that proceeds in the absence of any overt stress or stimulus. Basal autophagy is important for the clearance of damaged proteins and organelles in normal cells (especially fully differentiated, nondividing cells), and becomes elevated in response to stress; for example, in certain tumor cells, basal autophagy increases in response to strong oncogenic insults.[93,94]

BATS (barkor/Atg14(L) autophagosome targeting sequence) domain: A protein domain within Atg14L that is required for the recruitment of the PtdInsKC3 to LC3-containing puncta during macroautophagy induction; the predicted structure of the BATS domain suggests that it senses membrane curvature.[95]

Bck1: A MAPKKK downstream of Pkc1 and upstream of Mkk1/2 and Slt2 that controls cell integrity in response to cell wall stress; Bck1 is required for pexophagy[96] and mitophagy.[97] See also Slt2 and Hog1.

Bcl-2 family of proteins: There are two general classes of Bcl-2 proteins; anti-apoptotic proteins include Bcl-2, Bcl-X_L, Bcl-w and Mcl-1 that inhibit macroautophagy, and pro-apoptotic BH3-only proteins include BNIP3, Bad, Bik, Noxa, Puma and BimEL that induce macroautophagy. Interaction of Bcl-2 with Beclin 1 prevents the association of the latter with the class III PtdIns 3-kinase.

Beclin 1 (Bcl-2 interacting myosin/moesin-like coiled-coil protein 1): A mammalian homolog of yeast Atg6/Vps30 that forms part of the class III PtdIns 3-kinase complex involved in activating macroautophagy.[98]

BH3 domain: A Bcl-2 homology (BH) domain that is found in all Bcl-2 family proteins, whether they are pro-apoptotic or anti-apoptotic. A BH3 domain is also present in Beclin 1 and mediates the interaction with anti-apoptotic proteins possessing a BH3 receptor domain (i.e., Bcl-2, Bcl-X_L, Bcl-w and Mcl-1).

BH3-only proteins: A series of proteins that contain a BH3 domain (but not any other Bcl-2 homology, BH, domains). Several BH3-only proteins (BNIP3, Bad, Bik, Noxa, Puma and BimEL) can competitively disrupt the inhibitory interaction between Bcl-2 and Beclin 1 to allow the latter to act as an allosteric activator of PtdIns 3-kinase and to activate macroautophagy.

Bif-1 (Bax-interacting factor 1)/endophilin B1: A protein that interacts with Beclin 1 via UVRAG and is required for macroautophagy. Bif-1 has a BAR domain that may be involved in deforming the membrane as part of autophagosome biogenesis.[99]

BNIP3 (Bcl-2/adenovirus E1B 19-kDa interacting protein 3): A member of the BH3-only subfamily of Bcl-2 family proteins. Originally classified as a pro-apoptotic protein, it is required for the HIF-1-dependent induction of macroautophagy during hypoxia by disrupting the interaction of Beclin 1 with Bcl-2.[100,101]

CAL-101: A small molecule inhibitor of the p110d subunit of class 1A PtdIns 3-kinase; treatment of multiple myeloma cells results in macroautophagy induction.[102]

calpains: A class of calcium-dependent, nonlysosomal cysteine proteases that cleave and inactivate ATG5 and the ATG12–ATG5 conjugate, hence establishing a link between reduced Ca^{2+} concentrations and induction of autophagy.[103]

CaMKKβ (calcium/calmodulin-dependent protein kinase kinase-β): Activates AMPK in response to an increase in the cytosolic calcium concentration,[104] resulting in the induction of macroautophagy.[105]

CAPNS1 (calpain small 1): The regulatory subunit of micro- and millicalpain; CAPSN1-deficient cells are autophagy defective and display a substantial increase in apoptotic cell death.[106]

CASA (chaperone-assisted selective autophagy): A degradative process that utilizes the Drosophila co-chaperone Starvin or its mammalian homolog BAG3 to direct the degradation of aggregated substrates through the action of Hsc70, HspB8, the CHIP ubiquitin ligase and p62.[107] The requirement for ubiquitination of, and the absence of a requirement for the KFERQ motif in, the substrates, along with the involvement of the ATG proteins differentiate this process from CMA, which also uses chaperones for lysosome-dependent degradation.

caspases (cysteine-dependent aspartate-directed proteases): A class of proteases that play essential roles in apoptosis (programmed cell death type I) and inflammation. Several pro-apoptotic caspases cleave essential autophagy proteins, resulting in the inhibition of autophagy.[108]

CCI-779 (temsirolimus): A water-soluble rapamycin ester that induces macroautophagy.

Cdc48: A protein that extracts ubiquitinated proteins from the membrane as part of ERAD, but is also required for nonspecific macroautophagy in yeast.[109] See also Shp1 and VCP.

CD46: A cell-surface glycoprotein that interacts with the scaffold protein GOPC to mediate an immune response to invasive pathogens including Neisseria and Group A Streptococcus. Interaction of pathogens via the Cyt1 cytosolic tail induces autophagy, which involves GOPC binding to Beclin 1. CD46 is also used as a cellular receptor by several pathogens.[110]

chaperone-mediated autophagy (CMA): An autophagic process in mammalian cells by which proteins containing a particular pentapeptide motif related to KFERQ are transported across the lysosomal membrane and degraded.[111,112] The translocation process requires the action of the integral membrane protein LAMP-2A and both cytosolic and lumenal hsc70.[113,114]

chloroquine (CQ): Chloroquine and its derivatives (such as 3-hydroxychloroquine) raise the lysosomal pH and ultimately inhibit the fusion between autophagosomes and lysosomes, thus preventing the maturation of autophagosomes into autolysosomes, and blocking a late step of macroautophagy.[115]

c-Jun: A mammalian transcription factor that inhibits starvation-induced macroautophagy.[116]

Clg1: A yeast cyclin-like protein that interacts with Pho85 to induce macroautophagy by inhibiting Sic1.[117]

COG (conserved oligomeric Golgi) Complex: A cytosolic tethering complex that functions in the fusion of vesicles within the Golgi complex, but also participates in macroautophagy and facilitates the delivery of Atg8 and Atg9 to the PAS.[118]

crinophagy: Selective degradation of secretory granules by autophagy.[119] See also zymophagy.

cryptides: Peptides with a cryptic biological function that are released from cytoplasmic proteins by partial degradation or processing through macroautophagy (e.g., neoantimocrobial peptide released from ribosomal protein rps30).[120]

cysmethynil: A small-molecule inhibitor of isoprenylcysteine carboxylmethyl-transferase; treatment of PC3 cells causes an increase in LC3-II and cell death with macroautophagic features.[121]

cytoplasm-to-vacuole targeting (Cvt): A constitutive, biosynthetic pathway in yeast that transports resident hydrolases to the vacuole through a selective macroautophagy-like process.[122]

DAP1 (death-associated protein 1): A conserved phosphoprotein that is a substrate of mTOR and inhibits macroautophagy; inhibition of mTOR results in dephosphorylation of DAP1 and inhibition of macroautophagy, thus limiting the magnitude of the autophagic response.[123]

DAPK (death-associated protein kinase): A kinase that phosphorylates Thr119 of Beclin 1 to activate it by causing dissociation from Bcl-x_L and Bcl-2, thus activating macroautophagy.[124]

Desat1: A Drosophila lipid desaturase that localizes to autophagosomes under starvation conditions; the *Desat* mutant is defective in macroautophagy induction.[125]

DFCP1 (double FYVE domain-containing Protein 1): A PtdIns(3)P-binding protein that localizes to the omegasome.[126] Knockdown of DFCP1 does not result in an autophagy-defective phenotype.

diacylglycerol: A lipid second messenger that contributes to macroautophagic targeting of Salmonella-containing vacuoles.[127]

DJ-1/PARK7: An oncogene product whose loss-of-function is associated with Parkinson disease; overexpression suppresses macroautophagy through the JNK pathway.[128]

DOR (diabetes- and obesity-regulated gene/TP53INP2): A mammalian and Drosophila regulatory protein that shuttles between the nucleus and the cytosol; the cytosolic protein interacts with LC3 and GATE-16 and stimulates autophagosome formation.[129] See also TP53INP2.

DRAM (damage-regulated autophagy modulator): A p53 target that encodes a lysosomal protein that induces macroautophagy.[130]

Draper: A Drosophila homolog of the *Caenorhabditis elegans* engulfment receptor CED-1 that is required for macroautophagy associated with cell death during salivary gland degradation, but not for starvation-induced macroautophagy in the fat body.[131]

Drs: An apoptosis-inducing tumor suppressor that is involved in the maturation of autophagosomes.[132]

E2F1: A mammalian transcription factor that upregulates the expression of *LC3*, *ULK1* and *DRAM* directly, and *ATG5* indirectly.[133] E2F1 plays a role during DNA damage-induced macroautophagy.

eF1α (elongation factor 1α): Multifunctional member of the faimily of G-proteins with different cellular variants. The lysosomal variant of this protein acts coordinately with GFAP at the lysosomal membrane to modulate the stability of the CMA translocation complex. Release of membrane-bound eF1α in a GTP-dependent manner promotes disassembly of the translocation complex and consequently reduces CMA activity.[134]

eIF2α kinase (eukaryotic initiation factor 2α kinase): There are four mammalian eIF2α kinases that respond to different types

of stress. PKR and PERK induce macroautophagy in response to virus infection and ER stress, respectively.[135,136] See also Gcn2, PKR and PERK.

endosomal microautophagy (e-MI): A form of autophagy in which cytosolic proteins are sequestered into late endosomes/multivesicular bodies through a microautophagy-like process. Sequestration can be nonspecific or can occur in a selective manner mediated by hsc70. This process differs from chaperone-mediated autophagy as it does not require substrate unfolding and it is independent of the CMA receptor LAMP-2A.[137] This process occurs during MVB formation and requires the ESCRT I and ESCRT III protein machinery. See also endosome and multivesicular body.

endosome: The endosomal compartment receives molecules engulfed from the extracellular space. It consists of several discrete subcompartments including the early endosome, and the recycling endosome (recycles material back to the plasma membrane). In mammalian cells, early and/or multivesicular endosomes fuse with autophagosomes to generate amphisomes.

***epg* (ectopic PGL granules) mutants:** *C. elegans* mutants that are defective in the macroautophagic degradation of PGL-1, SEPA-1 and/or T12G3.1/p62.[138] The EPG-3 and EPG-7 proteins are homologs of VMP1 and FIP200, respectively, whereas EPG-1 may be a homolog of Atg13.[139]

ERK1/2 (extracellular signal regulated protein kinase 1/2): A kinase that phosphorylates and stimulates Gα-interacting protein (GAIP), which is a GAP for the trimeric G_i3 protein that activates macroautophagy,[140] and which may be involved in Beclin 1-independent autophagy.[141] Constitutively active ERK1/2 also traffics to mitochondria to activate mitophagy.[142]

Everolimus (RAD-001): An mTOR inhibitor similar to rapamycin that induces macroautophagy.

Exo84: A component of the exocyst complex, and an effector of RalB that is involved in nucleation and/or expansion of the phagophore; Exo84 binds RalB under nutrient-poor conditions, and stimulates the formation of a complex that includes ULK1 and the class III PtdIns3K.[143] See also RalB and Sec5.

Exophagy: A process used for protein secretion that is independent of the secretory pathway, and dependent on Atg proteins; acyl-coenzyme A-binding protein (Acb1) uses this route for delivery to the cell surface.[144-146]

Fasudil: A Rho-associated kinase (ROCK) inhibitor that enhances macroautophagy.[147]

FIP200 [focal adhesion kinase (FAK) family interacting protein of 200 kDa/RB1CC1 (retinoblastoma 1 inducible coiled coil-1)]: A putative mammalian functional counterpart of yeast Atg17. FIP200 is a component of the ULK1 complex.[148]

FKBP12 (FK506-binding protein 12): An immunophilin that forms a complex with rapamycin and inhibits mTOR.

FKBP51 (FK506-binding protein 51): An immunophilin that forms a complex with FK506 and rapamycin; FKBP51 promotes autophagy in irradiated melanoma cells, thus enhancing resistance to radiation therapy.[149]

FNBP1L (formin binding protein 1-like): An F-BAR-containing protein that interacts with Atg3 and is required for the autophagy-dependent clearance of *Salmonella* Typhimurium, but not other types of autophagy.[150]

FoxO1 (forkhead box O1): A mammalian transcription factor that regulates macroautophagy independent of transcriptional control; the cytosolic form of FoxO1 is acetylated after dissociation from sirtuin-2 (SIRT2), and binds Atg7 to allow induction of macroautophagy in response to oxidative stress or starvation.[151] FoxO1 can also be deacetylated by SIRT1, which leads to upregulation of Rab7 and increased autophagic flux.[152] See also SIRT1.

FoxO3 (forkhead box O3): A transcription factor that stimulates macroautophagy through transcriptional control of autophagy-related genes.[153,154]

FYCO1 (FYVE and coiled-coil domain containing 1): A protein that interacts with LC3, PtdIns(3)P and Rab7 to move autophagosomes toward the lysosome through microtubule plus end-directed transport.[155]

Gαi3: A heterotrimeric G protein that activates macroautophagy in the GDP-bound (inactive) form, and inhibits it when bound to GTP (active state).[156,157] See also AGS3 and GIV.

GABARAP (gamma-aminobutyric acid receptor-associated protein): A homolog of LC3.[158,159] The GABARAP family includes GABARAP, GABARAPL1, GABARAPL2/GATE-16 and GABARAPL3. The GABARAP proteins are involved in autophagosome formation and cargo recruitment.[45]

Gadd34 (growth arrest and DNA damage protein 34): A protein that is upregulated by growth arrest and DNA damage; Gadd34 binds to and dephosphorylates TSC2, leading to mTOR suppression and autophagy induction.[160]

GAIP (Gα-interacting protein): A GTPase activating protein that inactivates $Gα_{i3}$ (converting it to the GDP-bound form) and stimulates macroautophagy.[161] See also $Gα_{i3}$.

GATE-16 (Golgi-associated ATPase enhancer of 16 kDa/GABARAPL2): A homolog of LC3.[158,159]

Gcn2: A mammalian and yeast eIF2α serine/threonine kinase that phosphorylates and activates Gcn4 in response to amino acid depletion, which positively regulates macroautophagy.[135]

Gcn4: A transcriptional activator that controls the synthesis of amino acid biosynthetic genes and positively regulates macroautophagy in response to amino acid depletion.[135]

GFAP (glial fibrilary acid protein): Intermediate filament protein ubiquitiously distributed in all cell types that bears functions beyond filament formation. Monomeric and dimeric forms of this protein associate with the cytosolic side of the lysosomal membrane and contribute to modulating the stability of the CMA translocation complex in a GTP-dependent manner coordinated with eF1α also at the lysosomal membrane.[134]

Gfer (growth factor *erv1*-like): A flavin adenine dinucleotide (FAD)-dependent sulfhydryl oxidase that is part of a disulfide redox system in the mitochondrial intermembrane space, and is also present in the cysosol and nucleus. Downregulation of Gfer results in elevated levels of the mitochondrial fission GTPase Drp1, and decreased mitophagy.[162]

GIV (Gα-interacting, vesicle-associated protein/Girdin): A guanine nucleotide exchange factor for $Gα_{i3}$ that acts to downregulate macroautophagy.[12] GIV disrupts the AGS3-$Gα_{i3}$ complex in response to growth factors, releasing the G protein from the phagophore or autophagosome membrane; $Gα_{i3}$-GTP also

activates the class I PtdIns3K, thus inhibiting macroautophagy. See also $G\alpha_{i3}$.

GOPC (Golgi-associated PDZ and coiled-coil motif-containing protein/PIST/FIG/CAL): Interacts with Beclin 1, and the SNARE protein Syntaxin 6. GOPC can induce autophagy via a CD46-Cyt-1-dependent pathway following pathogen invasion.[110]

Gpnmb (glycoprotein nonmetastatic melanoma B/DC-HIL/osteoactivin): A protein involved in kidney repair that controls the degradation of phagosomes through macroautophagy.[163]

GSK-3β (glycogen synthase kinase-3β): A positive regulator of macroautophagy that inhibits mTOR by activating TSC1/2.

HDAC6 (histone deacetylase 6): A microtubule-associated deacetylase that interacts with ubiquitinated proteins. HDAC6 stimulates autophagosome-lysosome fusion by promoting the remodeling of F actin, promoting the quality control function of macroautophagy.[164-166]

HIF-1 (hypoxia-inducible factor 1): A dimeric transcription factor in which the α subunit is regulated by oxygen; the hydroxylated protein is degraded by the proteasome. HIF-1-mediated expression of BNIP3 results in the disruption of the Bcl-2-Beclin 1 interaction, thus inducing macroautophagy.[167,168]

HMGB1 (high mobility group box 1): A chromatin-associated nuclear protein that translocates out of the nucleus in response to stress such as ROS; HMGB1 binds to Beclin 1, displacing Bcl-2, thus promoting macroautophagy and inhibiting apoptosis.[169] In addition, macroautophagy promotes the release of HMGB1 from the nucleus and the cell, and extracellular HMGB1 can further induce macroautophagy through binding RAGE.[170,171] See also RAGE.

Hog1: A MAPK involved in hyperosmotic stress, which is a homolog of mammalian p38; Hog1 is required for mitophagy, but not other types of selective autophagy or nonspecific autophagy.[97] See also Pbs2, Slt2 and p38.

hsc70 (heast shock cognate of the Hsp70 family): This multifunctional cytosolic chaperone is the constitutive member of the hsp70 family of chaperones and participates in targeting of cytosolic proteins to lysosomes for their degradation via chaperone-mediated autophagy (CMA).[172] The cytosolic form of the protein also regulates the dynamics of the CMA receptor, whereas the lumenal form (lys-hsc70) is required for substrate translocation across the membrane.[173] Recently, this chaperone has been shown to play a role in the targeting of aggregated proteins (in a KFERQ-independent maner) for macroautophagy degradation through chaperone-assisted selective autophagy,[107] and in KFERQ-dependent targeting of cytosolic proteins to late endosomes for microautophagy.[137] See also chaperone-assisted selective autophagy, chaperone-mediated autophagy and endosomal microautophagy.

hsp90 (heat shock protein 90): Cytosolic chaperone also located in the lysosome lumen. The lysosomal form of hsp90 contributes to the stabilization of LAMP-2A during its lateral mobility in the lysosomal membrane.[174]

ICP34.5: A neurovirulence gene product encoded by the herpes simplex virus type 1 (HSV-1) that blocks eIF2α/PKR induction of autophagy.[135] ICP34.5-dependent inhibition of autophagy depends upon its ability to bind to Beclin 1.[175]

IKK (IκB kinase): An activator of the classical NFκB pathway composed of three subunits (IKKα, IKKβ, IKKγ, the latter also called NEMO) that are required for optimal induction of macroautophagy in human and mouse cells.[176]

immunoamphisomes: An organelle derived from the fusion of endosomes/phagosomes with autophagosomes that regulate dendritic cell-mediated innate and adaptive immune responses.[177]

immunophagy: The sum of diverse immunological functions of autophagy.[178]

inositol monophosphatase: An enzyme that regulates the level of myo-inositol-1,4,5-triphosphate (IP_3) levels. Inhibition of inositol monophosphatase stimulates autophagy independently of mTOR.[179]

IP_3R (inositol-1,4,5-triphosphate receptor): A Ca^{2+} channel present in the endoplasmic reticulum that controls the steady-state Ca^{2+} level in the ER. IP_3R is activated by IP_3, regulated by Bcl-2-like proteins, and acts as an inhibitor of autophagy, forming a multiprotein complex that comprises Bcl-2 and Beclin 1.[179]

IRGM (p47 immunity-related GTPase): Involved in the macroautophagic control of intracellular pathogens.[180]

isolation membrane: See phagophore.

JNK1 (c-Jun N-terminal kinase 1): A stress-activated kinase that phosphorylates Bcl-2 at Thr69, Ser70 and Ser87, causing its dissociation from Beclin 1, thus inducing macroautophagy.[181]

Jumpy/MTMR14: A member of the myotubularin family that is a PtdIns 3-phosphatase; knockdown increases macroautophagic activity.[182] Jumpy regulates the interaction of WIPI-1 with the phagophore.

JunB: A mammalian transcription factor that inhibits starvation-induced macroautophagy.[116]

Ku-0063794: A catalytic mTOR inhibitor that increases macroautophagic flux to a greater level than allosteric inhibitors such as rapamycin; short-term treatment with Ku-0063794 can inhibit both mTORC1 and mTORC2, but the effects on flux are due to the former.[183] See also WYE-354.

Laforin: A member of the dual-specificity protein phosphatase family that acts as a positive regulator of macroautophagy probably by inhibiting mTOR, as laforin deficiency causes increased mTOR activity.[184] Mutations in the genes encoding laforin or the putative E3-ubiquitin ligase malin, which form a complex, are associated with the majority of defects causing Lafora disease, a type of progressive neurodegeneration.

LAMP-2 (lysosome-associated membrane protein type 2): A widely expressed and abundant single-span lysosomal membrane protein.[185] Three spliced variants of the *lamp2* gene have been described. Knockout of the entire gene results in altered intracellular vesicular trafficking, defective lysosomal biogenesis, inefficient autophagosome clearance and alterations in intracellular cholesterol metabolism.[186-188]

LAMP-2A (lysosome-associated membrane protein 2A): One of the spliced variants of the *lamp2* gene that functions as a lysosomal membrane receptor for chaperone-mediated autophagy.[114] LAMP-2A forms multimeric complexes that allow translocation of substrates across the lysosome membrane.[174] Regulation of LAMP-2A is partly achieved by dynamic movement into and out of lipid microdomains in the lysosomal membrane.[173]

LC3 (microtubule-associated protein 1 light chain 3): A homolog of yeast Atg8, which is frequently used as a phagophore or autophagosome marker. Cytosolic LC3-I is conjugated to phsophatidylethanolamine to become phagophore- or autophagosome-associated LC3-II.[189] The LC3 family includes LC3A, LC3B and LC3C. These proteins are involved in the biogenesis of autophagosomes, and in cargo recruitment.[45] Vertebrate LC3 is regulated by phosphorylation of the N-terminal helical region by protein kinase A.[190] See also GABARAP.

LC3-associated phagocytosis: Phagocytosis in macrophages that involves the conjugation of LC3 to single membrane phagosomes, a process that promotes phagosome acidification and fusion with lysosomes.[191] TLR signaling is required for LC3-associated phagocytosis and leads to the recruitment of the Beclin 1 complex to phagosomes. See also NADPH oxidase.

lipophagy: Selective degradation of lipid droplets by lysosomes contributing to lipolysis (breakdown of triglycerides into free fatty acids). In mammals, this selective degradation has been described to occur via macroautophagy (macrolipophagy),[192] whereas in yeast, microlipophagy of cellular lipid stores has also been described.

LIR/LRS (LC3-interacting region/LC3 recognition sequence): This term refers to the WXXL-like sequences found in Atg19, Atg32, NBR1, NIX and p62 that link these proteins to Atg8/LC3 (see also AIM and WXXL-motif).[193]

LKB1 (Serine/Threonine Kinase 1): A kinase that is upstream of, and activates, AMPK.[194]

LLPD (long-lived protein degradation): Macroautophagy is the primary mechanism used by cells to degrade long-lived proteins, and a corresponding assay can be used to monitor autophagic flux;[195] a useful abbreviation for long-lived protein degradation is LLPD.[196]

lucanthone: An anti-schistome compound that inhibits a late stage of macroautophagy; treatment results in deacidification of lysosomes and the accumulation of autophagosomes.[197]

LRRK2 (leucine-rich repeat kinase 2): A large multidomain, membrane-associated kinase and GTPase whose Parkinson disease-associated mutations affect regulation of autophagy.[198,199]

LRSAM1 (leucine-rich repeat and sterile α-motif containing 1): A human leucine-rich repeat protein that potentially interacts with GABARAPL2; knockdown of LRSAM1 results in a defect in anti-Salmonella autophagy.[200]

LY294002: An inhibitor of PtdIns 3-kinases; it inhibits macroautophagy.[201]

lysosome: A degradative organelle in higher eukaryotes that compartmentalizes a range of hydrolytic enzymes and maintains a highly acidic pH.

macroautophagy: An autophagic process involving the formation of an autophagosome, a double- or multiple-membrane cytosolic vesicle of non-lysosomal/vacuolar origin.

MAP1LC3 (microtubule-associated protein 1 light chain 3): See LC3.

MDC (monodansylcadaverine): A lysosomotropic autofluorescent compound that accumulates in acidic compartments such as autolysosomes, and also labels (but is not specific for) autophagosomes.[202,203]

microautophagy: An autophagic process involving direct uptake of cytosol, inclusions (e.g., glycogen) and organelles (e.g., ribosomes, peroxisomes) at the lysosome/vacuole by protrusion, invagination or septation of the sequestering organelle membrane.

MIPA (micropexophagic apparatus): A curved double-membrane structure formed by the PAS that may serve as a scaffold for completion of the sequestration of peroxisomes during micropexophagy; fusion with the vacuolar sequestering membranes encloses the organelles within an intralumenal vesicle.[204]

mitophagy: The selective degradation of mitochondria; can occur by a micro- or macroautophagic process.[205]

Mkk1/2: A MAPKK downstream of Bck1 that is required for mitophagy and pexophagy.[97] See also Bck1 and Slt2.

MTMR3 (myotubularin-related phosphatase 3): This protein localizes to the phagophore and negatively regulates macroautophagy (see Jumpy/MTMR14).[206]

mTOR (mammalian target of rapamycin): The mammalian ortholog of TOR. Together with its binding partners it forms either mTOR complex 1 (mTORC1) or mTOR complex 2 (mTORC2). See also TORC1 and TORC2.

multivesicular body (MVB)/multivesicular endosome: An endosome containing multiple 50- to 80-nm vesicles that are derived from invagination of the limiting membrane. Under some conditions the MVB contains hydrolytic enzymes in which case it may be considered to be a lysosome or autolysosome with ongoing microautophagy.

multivesicular body (MVB) sorting pathway: A process in which proteins are sequestered into vesicles within the endosome through the invagination of the limiting membrane. This process is usually, but not always, dependent upon ubiquitin tags on the cargo and serves as one means of delivering integral membrane proteins destined for degradation into the vacuole/lysosome lumen. ESCRT (endosomal sorting complex required for transport) complexes are required for the formation of MVBs and for autophagosome maturation.

NADPH oxidases (NOX): These enzymes contribute to macroautophagic targeting of Salmonella in leukocytes and epithelial cells through the generation of reactive oxygen species.[207] The NOX2 NADPH oxidase in macrophages is required for LC3-associated phagocytosis.

NAF-1 (nutrient-deprivation autophagy factor-1/CISD2): An integral membrane component that associates with the inositol-1,4,5-triphosphate receptor complex; NAF-1 binds Bcl-2 at the ER, and is required for Bcl-2 to bind Beclin 1, resulting in the inhibition of macroautophagy.[208] CISD2 was reported to be associated with the ER, but the majority of the protein is localized to mitochondria, and mutations in *CISD2* are associated with Wolfram syndrome 2; accelerated autophagy in *Cisd2*$^{-/-}$ mice may cause mitochondrial degradation, leading to neuron and muscle degeneration.[209]

NBR1 (neighbor of BRCA1 gene 1): A selective substrate of macroautophagy with structural similarity to p62. Functions as an adaptor that binds ubiquitinated proteins and LC3 to allow the degradation of the former by macroautophagy.[193]

NDP52 (nuclear dot protein 52 kDa): An adaptor that binds to the bacterial ubiquitin coat and Atg8/LC3 to target invasive

bacteria, including *Salmonella enterica* serovar Typhimurium and *Streptococcus pyogenes* for autophagosomal sequestration.[210]

necroptosis: A form of programmed necrotic cell death;[211] induction of macroautophagy-dependent necroptosis is required for childhood acute lymphoblastic leukemia cells to overcome glucocorticoid resistance.[212]

NFκB (nuclear factor kappa-light chain-enhancer of activated B cells): Activates mTOR to inhibit macroautophagy.[213]

NH_4Cl (ammonium chloride): A weak base that is protonated in acidic compartments and neutralizes them; inhibits the clearance of autophagosomes and amphisomes.

NIX (NIP3-like X; BNIP3L/BNIP3α): A Bcl-2 related protein that is required for selective mitochondrial clearance (mitophagy) during erythroid differentiation.[214]

Nod (nucleotide-binding oligomerization domain): An intracellular peptidoglycan (or pattern recognition) receptor that senses bacteria and induces macroautophagy, involving ATG16L1 recruitment to the plasma membrane during bacterial cell invasion.[215]

noncanonical autophagy: A functional macroautophagy pathway that only uses a subset of the characterized Atg proteins to generate an autophagosome. At present, Beclin 1-independent,[141,216] and Atg5-Atg7-independent[217] forms of macroautophagy have been reported.

OATL1: A Tre2-Bub2-Cdc16 (TBC) domain-containing GTPase activating protein for Rab33B; OATL1 is recruited to phagophores and autophagosomes via direct interaction with the Atg8 family proteins (via a LIR/LRS-like sequence), and it regulates the interaction of autophagosomes with lysosomes by inactivating Rab33B.[218] Overexpression of OATL1 inhibits autophagosome maturation at a step prior to fusion, suggesting that it might interfere with a tethering/docking function of Rab33B. See also Rab33B and LIR.

omegasome: DFCP1-containing structures located at the ER that are involved in autophagosome formation during amino acid starvation.[126]

Omi/HtrA2 (high temperature requirement factor A2): A serine protease that degrades Hax-1, a Bcl-2 family-related protein, to allow macroautophagy induction; knockdown of Omi, or the presence of a protease-defective mutant form, results in decreased basal macroautophagy and may lead to neurodegeneration.[219]

p8 (Nupr1): A transcriptional regulator that controls macroautophagy by repressing the transcriptional activity of FoxO3.[220]

p27 ($p27^{Kip1}$): A cyclin-dependent kinase inhibitor that is phosphorylated and stabilized by an AMPK-dependent process and stimulates macroautophagy.[194]

p38α (mitogen-activated protein kinase isoform p38α): A signaling component that negatively regulates the interaction of mAtg9 and p38IP, and thus inhibits macroautophagy. The yeast homolog is Hog1. See also Hog1.

p38IP [p38α interacting protein/family with sequence similarity 48A (FAM48A)]: A protein that interacts with the C-terminal domain of mAtg9; this interaction is negatively regulated by p38α.[221]

p53: A tumor suppressor. Nuclear p53 activates autophagy, at least in part, by stimulating AMPK and DRAM. Cytoplasmic p53 inhibits autophagy.[222]

p62 (sequestosome 1/SQSTM1): A selective substrate of macroautophagy, that functions as an adaptor protein that links ubiquitinated proteins to LC3. p62 accumulates in cells when macroautophagy is inhibited. p62 interaction with LC3 requires a WXXL or a LIR motif analogous to the interaction of Atg8 with Atg19.[223]

p97: See VCP.

p110β: A catalytic subunit of the class IA PtdIns 3-kinase; this subunit plays a positive role in macroautophagy induction that is independent of mTOR or PKB/Akt, and instead acts through the generation of PtdIns(3)P, possibly by acting as a scaffold for the recruitment of phosphatases that act on PtdIns(3,4,5)P_3 or by recruiting and activating Vps34.[224]

Parkin: An E3 ubiquitin ligase (mutated in autosomal recessive forms of Parkinson disease) that is recruited from the cytosol to mitochondria with a low membrane potential to promote their clearance by mitophagy.[225]

PARL (presenilin associated rhomboid-like): The mammalian homolog of Drosophila rhomboid-7, a mitochondrial protease; regulates the stability and localization of PINK1. A missense mutation in the N terminus has been identified in some patients with Parkinson disease.[226]

PARP-1 (poly [ADP-ribose] polymerase 1): A nuclear enzyme involved in DNA damage repair; doxorubicin-induced DNA damage elicits a macroautophagic response that is dependent on PARP-1.[227] In conditions of oxidative stress, PARP-1 promotes macroautophagy through the LKB1-AMPK-mTOR pathway.[228]

PAS (phagophore assembly site): Also referred to as the preautophagosomal structure. A perivacuolar compartment or location that is the proposed nucleation site for sequestering vesicles (e.g., autophagosomes and Cvt vesicles) used in macroautophagy-like processes in fungi. The PAS may supply membranes during the formation of the sequestering vesicles or may be an organizing center where most of the macroautophagic machinery resides at least transiently. The PAS or its equivalent is yet to be defined in mammalian cells.[229,230]

Pbs2: A MAPKK upstream of Hog1 that is required for mitophagy.[97]

Pcl1: A yeast cyclin that activates Pho85 to stimulate macroautophagy by inhibiting Sic1.[117]

Pcl5: A yeast cyclin that activates Pho85 to inhibit macroautophagy through degradation of Gcn4.[117]

PDK1 (phosphoinositide-dependent protein kinase 1): An activator of PKB/Akt. Recruited to the plasma membrane and activated by PtdIns(3,4,5)P_3 which is generated by the class I PtdIns 3-kinase.

PEA-15/PED (phosphoprotein enriched in astrocytes 15 kDa/phosphoprotein enriched in diabetes): A death effector domain-containing protein that modulates JNK in glioma cells to promote macroautophagy.[231]

PERK (PKR-like ER kinase): A mammalian eIF2α kinase that may induce macroautophagy in response to ER stress.[136]

PES (2-phenylethynesulfonamide): A small molecule inhibitor of HSP70; PES interferes with lysosomal function, causing a defect in macroautophagy and chaperone-mediated autophagy.[232]

pexophagy: A selective type of autophagy involving the sequestration and degradation of peroxisomes; it can occur by a micro- or macroautophagic process (micro- or macropexophagy).[233]

phagophore: Membrane cisterna that is involved in an initial event during formation of the autophagosome. Also referred to as the "isolation membrane," the phagophore is the initial sequestering compartment of macroautophagy.[234]

Pho80: A yeast cyclin that activates Pho85 to inhibit macroautophagy in response to high phosphate levels.[117]

Pho85: A multifunctional cyclin-dependent kinase that interacts with at least ten different cyclins or cyclin-like proteins to regulate the cell cycle and responses to nutrient levels. Pho85 acts to negatively and positively regulate macroautophagy, depending on its binding to specific cyclins.[117] See also Clg1, Pcl1, Pcl5, Pho80 and Sic1.

phosphatidylinositol (PtdIns) 3-kinase (PtdIns3K): A family of enzymes that add a phosphate group to the 3' hydroxyl on the inositol ring of phosphoinositides. The class III PtdIns 3-kinases (see Vps34; also referred to as PtdIns3KC3) are stimulatory for macroautophagy, whereas class I enzymes are inhibitory. So far, nothing is known about the involvement of the class II enzymes in autophagy.

piecemeal microautophagy of the nucleus (PMN)/micronucleophagy: A process in which portions of the yeast nuclear membrane and nucleoplasm are invaginated into the vacuole, scissioned off from the remaining nuclear envelope and degraded within the vacuole lumen.[235,236]

PINK1 (PTEN-induced kinase 1/PARK6): A mitochondrial protein (mutated in autosomal recessive forms of Parkinson disease) that is normally processed in a membrane potential-dependent manner to maintain mitochondrial function,[237] suppressing the need for autophagy.[238] Upon mitochondrial depolarization, this processing is arrested so that PINK1 spans the outer mitochondrial membrane, recruiting Parkin to facilitate mitophagy.[239]

PKA (protein kinase A): A serine/threonine kinase that negatively regulates macroautophagy in yeast,[240] as well as in mammalian cells through direct phosphorylation of LC3.[190] Bacterial toxins that activate PKA can also inhibit autophagy.[241]

PKB (protein kinase B) or Akt: A serine/threonine kinase that negatively regulates macroautophagy.

Pkc1: A protein serine/threonine kinase involved in the cell wall integrity pathway upstream of Bck1; required for pexophagy and mitophagy.[97] See also Bck1 and Slt2.

PKCδ (protein kinase Cδ): PKCδ regulates JNK activation. PKCδ also activates NADPH oxidases, which are required for antibacterial macroautophagy.[242]

PKR (dsRNA-dependent protein kinase R): A mammalian eIF2α kinase that induces macroautophagy in response to viral infection.[243]

PGRP (peptidoglycan-recogntion protein): A cytosolic Drosophila protein that induces autophagy in response to invasive *Listeria monocytogenes*.[244]

PNS (peri-nuclear structure): A punctate structure in *P. pastoris* marked by Atg35, which requires Atg17 for recruitment and is involved in micropexophagy; the PNS may be identical to the PAS.[245]

presenilin: A protease that is part of the γ-secretase complex. Mutations in presenilin 1 result in the accumulation of autophagosomes resulting at least in part from a defect in lysosomal acidification; one of the V-ATPase subunits does not target properly to the lysosome.[246,247]

programmed cell death (PCD): Regulated self-destruction of a cell. Type I is associated with apoptosis and is marked by cytoskeletal breakdown and condensation of cytoplasm and chromatin followed by fragmentation. Type II is associated with macroautophagy and is characterized by the presence of autophagic vacuoles (autophagosomes) that sequester organelles. Type III is marked by the absence of nuclear condensation, and the presence of a necrotic morphology with swelling of cytoplasmic organelles (oncosis).

protolysosomes: Vesicles derived from autolysosomes that mature into lysosomes during autophagic lysosome reformation (see ALR).[20]

PTEN (phosphatase and tensin homolog): A 3' phosphoinositide phosphatase that dephosphorylates PtdIns(3,4,5)P_3, thereby inhibiting PDK1 and PKB/Akt activity.

PTPσ (protein tyrosine phosphatase sigma): A dual-domain protein tyrosine phosphatase that antagonizes the action of the class III PtdIns3K; loss of PTPσ results in hyperactivation of basal and induced macroautophagy.[248]

Rab1: See Ypt1.

Rab7: A small GTP binding protein functioning in transport from early to late endosomes and from late endosomes to lysosomes.[249] Rab7 is also needed for the clearance of autophagic compartments, most likely for the fusion of amphisomes with lysosomes.[250,251] The yeast homolog is Ypt7.

Rab24: A small GTP binding protein with unusual characteristics that associates with autophagic compartments in amino acid-starved cells.[252]

Rab32: A small GTP binding protein that localizes to the endoplasmic reticulum, and enhances autophagosome formation under basal conditions.[253]

Rab33B: A Rab protein of the medial Golgi complex that binds Atg16L1 and plays a role in autophagosome maturation by regulating fusion with lysosomes.[254] Rab33B is a target of OATL1, which functions as a GAP.[255]

RabG3b: A Rab GTPase that functions in the differentiation of tracheary elements of the Arabidopsis xylem through its role in macroautophagy; this protein is a homolog of Rab7/Ypt7.[256]

RAD001 (Everolimus): An orally administered derivative of rapamycin.

Rag (Ras-related GTPase): A GTPase that activates TORC1 in response to amino acids.[257]

RAGE (peceptor for advanced glycation end products): A member of the immunoglobulin gene superfamily that binds the high mobility group box 1 chromatin binding protein (HMGB1); RAGE overexpression enhances macroautophagy and reduces apoptosis.[258] This can occur in response to ROS, resulting in the upregulation of macroautophagy and the concomitant downregulation of apoptosis, favoring tumor cell survival in response to anticancer treatments that increase ROS production.[259] See also HMGB1.

RalB: A GTPase that regulates exocytosis and the immune response; in nutrient-rich conditions RalB-GTP binds Sec5, whereas under starvation conditions, RalB-GTP nucleates phagophore formation through assembly of a ULK1-Beclin 1-VPS34 complex via interaction with the Exo84 protein.[143] RalB may be an analog of yeast Sec4.[260] See also Exo84, Sec4 and Sec5.

rapamycin: TOR (TOR complex 1) inhibitor, which induces autophagy. TOR complex 2 is much less sensitive to inhibition by rapamycin.

Rb-E2F1 (retinoblastoma protein-E2 transcription factor 1): Rb is a tumor suppressor that promotes growth arrest, and protects against apoptosis. E2F1 regulates the transition from the G_1 to the S phase in the cell cycle, and is a pro-apoptotic member of the E2F transcription family. In addition to controlling the cell cycle and apoptosis, the interaction between Rb and E2F1 regulates macroautophagy; RB and E2F1 downregulate and upregulate Bcl-2, respectively, resulting in the induction of macroautophagy or apoptosis.[261]

reactive oxygen species (ROS): These are chemically-reactive molecules that contain oxygen, including hydrogen peroxide, the hydroxyl radical $^{\cdot}OH$, and the superoxide radical $^{\cdot}O_2^-$. Hydrogen peroxide transiently inhibits delipidation of LC3 by Atg4, which is permissive for starvation-induced autophagy.[262] Superoxide is essential for triggering injury-induced mitochondrial fission and mitophagy.[238]

residual body: A lysosome that contains indigestible material such as lipofuscin.[263]

resveratrol: An allosteric activator of Sirtuin 1 that induces macroautophagy.[264]

reticulophagy: The selective degradation of endoplasmic reticulum by a macroautophagy-like process.[265]

Rheb (Ras homolog enriched in brain): A small GTP-binding protein that activates TOR when it is in the GTP-bound form.[266]

Ribophagy: The selective degradation of ribosomes by macroautophagy.[267]

Rim15: A yeast kinase that regulates transcription factors in response to nutrients. Rim15 positively regulates macroautophagy and is negatively regulated by several upstream kinases including Tor, PKA, Sch9 and Pho85.[117,268]

RNS2: A class II RNase of the T2 family that localizes to the lumen of the endoplasmic reticulum (or an ER-related structure) and vacuole in Arabidopsis; RNS2 is involved in rRNA turnover, and *rns2* mutants display constitutive macroautophagy, likely due to a defect in cellular homeostasis.[269]

rubicon (RUN domain protein as Beclin 1-interacting and cysteine-rich CONtaining): Rubicon is part of a PtdIns 3-kinase complex (Rubicon-UVRAG-Beclin 1-hVps34-p150) that localizes to the late endosome/lysosome and inhibits macroautophagy.[270,271]

Sch9: A kinase that functions in parallel with PKA to negatively regulate macroautophagy. Sch9 appears to function in parallel with TOR, but is also downstream of the TOR kinase.[268]

SEA (Seh1-associated) protein complex: A complex found in yeast that includes the Seh1 nucleoporin and the COPII component Sec13 (also a nucleoporin), in addition to Npr2 and Npr3, and four other relatively uncharacterized proteins; the SEA complex associates with the vacuole, potentially acting as a membrane coat and is involved in protein trafficking, amino acid biogenesis, and the starvation response including macroautophagy.[272]

Sec1: Functions with the plasma membrane SNAREs Sso1/Sso2 and Sec9 to form the site for vesicle-mediated exocytosis; as with Sso1/Sso2 and Sec9, temperature sensitive *sec1* mutations also abrogate autophagic delivery of GFP-Atg8.[273] See also Sso1/Sso2.

Sec2: A guanine nucleotide exchange factor for Sec4 that normally functions in exocytosis. Upon the induction of macroautophagy, Sec2 function is diverted to promote membrane delivery to the PAS.[260]

Sec4: A Rab family GTPase that normally functions in exocytosis; under macroautophagy-inducing conditions Sec4 is needed for the anterograde movement of Atg9 to the PAS.[260]

Sec5: A component of the exocyst complex; Sec5 binds RalB, Beclin 1, mTORC1, ULK1 and VPS34 under nutrient-rich conditions and prevents these components from interacting with Exo84, thus inhibiting macroautophagy.[143] See also RalB and Exo84.

Sec9: Plasma membrane SNARE light chain that forms a complex with Sso1/Sso2 to generate the target complex of vesicle exocytosis; as with Sso1/Sso2, loss of Sec9 function blocks autophagy at an early stage by disrupting targeting of Atg9 to the tubulovesicular cluster and PAS.[273] See also Sso1/Sso2.

Sec18: Homolog of the mammalian NSF, an ATPase globally responsible for SNARE disassembly. Loss of function inhibits SNARE-dependent early and late events of macroautophagy (i.e., vesicular delivery of Atg9 to the tubulovesicular cluster and PAS,[273] and fusion of autophagosomes with the vacuole[274]).

Sec22: A vesicle SNARE involved in ER and Golgi transport; mutations in Sec22 also block Atg9 trafficking to the tubulovesicular cluster and PAS. Crosslinking experiments suggest Sec22 may be the v-SNARE responsible for the autophagy functions of the ordinarily plasma membrane Sso1/Sso2-Sec9 t-SNARE complex.[273] See also Sso1/Sso2.[273]

SEPA-1 (suppressor of ectopic P granule in autophagy mutants-1): A *C. elegans* protein that is involved in the selective degradation of P granules through macroautophagy.[275]

septin cages: Septins are GTP-binding proteins that assemble into nonpolar filaments (characterized as unconventional cytoskeleton), often acting as scaffolds for the recruitment of other proteins. Septin cages form in response to infection by Shigella; the cages surround the bacteria, preventing intercellular spread, and serve to recruit autophagy components such as p62 and LC3.[276]

Sestrin 2: A protein encoded by a p53 target-induced gene that inhibits mTORC1 and induces macroautophagy, also acting as an AMPK activator.

Shp1/Ubx1: A Ubx (ubiquitin regulatory x)-domain protein that is needed for the formation of autophagosomes during nonselective macroautophagy; Shp1 binds Cdc48 and Atg8–PE, and may be involved in extracting the latter during phagophore expansion.[109]

Sic1: A yeast cyclin-dependent kinase inhibitor that blocks the activity of Cdc28-Clb kinase complexes to control entry into the S phase of the cell cycle. Sic1 is a negative regulator of macroautophagy that inhibits Rim15.[117]

sirolimus: An immunosuppressant also referred to as rapamycin.

SIRT1 (Sirtuin 1): A NAD^+-dependent protein deacetylase that is activated by caloric restriction or glucose deprivation; SIRT1 can induce macroautophagy through the deacetylation of autophagy-related proteins and/or FoxO transcription factors.[277]

SLAPs (spacious listeria-containing phagosomes): SLAPs can be formed by *Listeria monocytogenes* during infection of macrophages or fibroblasts if bacteria are not able to escape into the cytosol.[278] SLAPs are thought to be immature autophagosomes in that they bear LC3 but are not acidic and do not contain lysosomal degradative enzymes. The pore-forming toxin listeriolysin O is essential for SLAPs formation and is thought to create small pores in the SLAP membrane that prevent acidification by the v-ATPase. SLAP-like structures have been observed in a model of chronic *L. monocytogenes* infection,[279] suggesting that autophagy may contribute to the establishment/maintenance of chronic infection.

SLC1A5 (solute carrier family 1 member 5): A high affinity, Na^+-dependent transporter for L-glutamine; a block of transport activity leads to inhibition of mTORC1 signaling and the subsequent activation of autophagy.[280] See also SLC7A5.

SLC7A5 (solute carrier family 7 member 5/SLC3A2): A bidirectional transporter that allows the simultaneous efflux of L-glutamine and influx of L-leucine; this transporter works in conjunction with SLC1A5 to regulate mTORC1.[280]

Slg1 (Wsc1): A cell surface sensor in the Slt2 MAPK pathway that is required for mitophagy.[97] See also Slt2.

SLR (sequestosome 1/p62-like receptor): Receptors that act as autophagic adaptors, and in proinflammatory or other types of signaling.[281]

Slt2: A MAPK that is required for pexophagy and mitophagy.[97] See also Pkc1, Bck1 and Mkk1/2.

smARF (short mitochondrial ARF): A small isoform of $p19^{ARF}$ that results from the use of an alternate translation initiation site, which localizes to mitochondria and disrupts the membrane potential, leading to a massive increase in macroautophagy and cell death.[282]

Snapin: An adaptor protein involved in dynein-mediated late endocytic transport; snapin is needed for the delivery of endosomes from distal processes to lysosomes in the neuronal stroma, allowing maturation of autolysosomes.[283]

spermidine: A natural polyamine that induces macroautophagy through the inhibition of histone acetylases.[284]

sphingolipids: Sphingolipids are a major class of lipids. Some metabolites including ceramide, sphingosine and sphingosine 1-phosphate are bioactive signaling molecules. Ceramide and sphingosine 1-phosphate are positive regulators of macroautophagy.[285,286]

Spinster: A putative lysosomal efflux permease required for autophagic lysosome reformation.[287]

Sqa (spaghetti-squash activator): A myosin light chain kinase-like protein that is a substrate of Atg1 in Drosophila; required for starvation-induced autophagosome formation, and the mammalian homolog ZIPK (zipper-interacting protein kinase) is also involved in mAtg9 trafficking.[288]

Ssk1: A component of the Hog1 signaling cascade that is required for mitophagy.[97] See also Hog1.

Sso1/Sso2: Highly homologous plasma membrane syntaxins (SNAREs) of *S. cerevisiae* involved in exocytosis; the Sso1/Sso2 proteins also control the movement of Atg9 to the tubulovesicular cluster and PAS during macroautophagy and the Cvt pathway.[273]

Syntaxin-5: A Golgi-localized SNARE protein involved in vesicular transport of lysosomal hydrolases, a process that is critical for lysosome biogenesis; syntaxin-5 is needed for the later stages of autophagy.[289]

TAK1 (transforming growth factor-β-activating kinase 1): Required for TRAIL-induced activation of AMPK. Required for optimal macroautophagy induction by multiple stimuli.[290]

TAKA (transport of Atg9 after knocking out *ATG1*) assay: An epistasis analysis that examines the localization of Atg9-GFP in a double mutant, where one of the mutations is a deletion of *ATG1*.[58] In *atg1*Δ mutants, Atg9-GFP is restricted to the PAS; if the second mutation results in a multiple puncta phenotype, the corresponding protein is presumably required for anterograde transport of Atg9 to the PAS.[291] This analysis can be combined with localization of RFP-Ape1 to determine if any of the Atg9-GFP puncta reach the PAS, in which case that punctum would colocalize with the RFP-Ape1 PAS marker.

TASCC (TOR-autophagy spatial coupling compartment): A compartment located at the trans-Golgi where autolysosomes and mTOR accumulate during Ras-induced senescence to provide spatial coupling of protein secretion (anabolism) with degradation (catabolism); for example, amino acids generated from autophagy would quickly reactivate mTOR, whereas autophagy would be rapidly induced via mTOR inhibition when nutrients are again depleted.[292]

Tecpr1 (tectonin domain-containing protein): A protein that interacts with Atg5 and WIPI-2, and localizes to the phagophore (localization is dependent on WIPI-2); Tecpr1 is needed for phagophore formation during macroautophagic elimination of Shigella, but not for starvation-induced autophagy.[293] Tecpr1 also localizes to autophagosomes that target other pathogenic microbes such as group A Streptococcus, to depolarized mitochondria and to protein aggregates, suggesting a general role in selective macroautophagy.

TFEB (transcription factor EB): A transcription factor that positively regulates the expression of genes involved in lysosomal biogenesis (those in the Coordinated Lysosomal Expression and Regulation network), and also several of those involved in macroautophagy (including *UVRAG*, *WIPI*, *MAP1LC3B* and *ATG9B*); the use of a common transcription factor allows the coordinated expression of genes whose products are involved in the turnover of cytoplasm.[294]

TGFβ (transforming growth factor-β): A cytokine that activates autophagy through the Smad and JNK pathways. TGFβ induces the expression of several *ATG* genes including *BECN1*.

TIGAR (TP53-induced glycolysis and apoptosis regulator): A protein that modulates glycolysis, causing an increase in NADPH, which results in a lower ROS level; this reduces the

sensitivity to oxidative stress and apoptosis, but also has the effect of lowering the level of macroautophagy.[295]

Tlg2: Endocytic SNARE light chain involved in early stages of the Cvt pathway[296] and in autophagosome membrane formation.[273] Deletion of *TLG2* results in a modest impairment in Atg9 delivery to the PAS.

TLR (toll-like receptor): A family of receptors that induces autophagy following binding to a corresponding pathogen-associated molecular pattern (PAMP).

TM9SF1: A protein with nine transmembrane domains that induces autophagy when overexpressed.[297]

TMEM: TMEM74 and TMEM166 are integral membrane proteins that induce autophagy when overexpressed.[298,299]

TOR (target of rapamycin): A protein kinase that negatively regulates macroautophagy. Present in two complexes, TORC1 and TORC2. TORC1 is particularly sensitive to inhibition by rapamycin. TORC1 regulates macroautophagy in part through Tap42-PP2A, and also by phosphorylating Atg13 and Atg1.

TORC1 (TOR complex 1): A rapamycin-sensitive protein complex of TOR that includes Tor1/Tor2 (mTOR), Kog1 (Raptor), Lst8 (mLST8), Rhb1 (Rheb) and Tco89.[300] mTORC1 also includes DEPTOR.[301] In mammalian cells, sensitivity to rapamycin is conferred by Raptor. TORC1 directly regulates macroautophagy.

TORC2 (TOR complex 2): A relatively rapamycin-insensitive protein complex of TOR that includes Tor2 (mTOR), Avo1 (SIN1), Avo2, Avo3 (Rictor), Bit2, Bit61, Lst8 (mLST8) and Tsc11; mTORC2 also includes DEPTOR, FKBP38, PRAS40 and Protor-1.[300-302] The main difference relative to TORC1 is the replacement of Raptor by Rictor. TORC2 is primarily involved with regulation of the cytoskeleton.

Torin1: A selective ATP-competitive mTOR inhibitor that directly inhibits both TORC1 and TORC2.[303]

TP53INP1 (tumor protein 53-induced nuclear protein 1): A stress-response protein that promotes p53 activity; cells lacking TP53INP1 display reduced basal and stress-induced autophagy.[304]

TP53INP2 (tumor protein 53-induced nuclear protein 2): Interacts with VMP1 and is needed for the recruitment of Beclin 1 and LC3 to autophagosomes. Translocates from the nucleus to autophagosomes during autophagy induction and binds VMP1 and LC3 directly.[305] See also DOR.

TRAF6 (tumor necrosis factor receptor-associated factor 6): An E3 ubiquitin ligase that ubiquitinates Beclin 1 to induce TLR4-triggered macroautophagy in macrophages.[6]

TRAIL (tumor necrosis factor-related apoptosis-inducing ligand): Induces autophagy by activating AMPK, thus inhibiting mTORC1 during lumen formation.

TRAPPIII (transport protein particle III): A guanine nucleotide exchange factor for Ypt1 that functions in macroautophagy in yeast.[306] TRAPPIII is composed of Bet3, Bet5, Trs20, Trs23, Trs31, Trs33 and a unique subunit, Trs85.

TRS85: A component of the TRAPPIII complex that is required specifically for macroautophagy.

TSC1/2 (tuberous sclerosis complex 1/2): A stable heterodimer (composed of hamartin and tuberin) inhibited by PKB/Akt and ERK1/2 (phosphorylation causes dissociation of the dimer), and activated by AMPK. TSC1/2 acts as a GAP for Rheb, thus inhibiting TOR.

ubiquilins (UBQLNs): Adaptor proteins that deliver ubiquitinated substrates to the proteasome. Ubiquilins may aid in the incorporation of protein aggregates into autophagosomes, and also promote the maturation of autophagosomes at the stage of fusion with lysosomes.[307]

ubiquitin: A 76-kDa protein that is conjugated to lysine residues. Ubiquitin is traditionally considered part of the ubiquitin-proteasome system and tags proteins for degradation; however, ubiquitin is also linked to various types of autophagy including aggrephagy (see p62 and NBR1).

ULK family (Unc51-like kinase): Homolog of yeast Atg1. In mammalian cells consists of five members, ULK1, ULK2, ULK3, ULK4, STK36/fused (ULK5). ULK1 and ULK2 are required for macroautophagy and ULK3 for oncogene-induced senescence.[308-310]

UVRAG (UV irradiation resistance-associated gene): A Vps38 homolog that can be part of the class III PtdIns 3-kinase complex. UVRAG functions in several ways to regulate macroautophagy: (1) It disrupts Beclin 1 dimer formation and forms a heterodimer that activates macroautophagy; (2) it binds to Bif-1 to allow activation of class III PtdIns 3-kinase to stimulate macroautophagy; (3) it interacts with the class C Vps/HOPS proteins involved in fusion of autophagosomes or amphisomes with the lysosome; (4) it competes with Atg14L for binding to Beclin 1, thus directing the class III PtdIns 3-kinase to function in the maturation step of macroautophagy.[311]

vacuole: The yeast equivalent of the lysosome; this organelle also carries out storage and osmoregulatory functions.

vacuole import and degradation (Vid): A degradative pathway in yeast in which a specific protein(s) is sequestered into small single-membrane cytosolic vesicles that fuse with the vacuole allowing the contents to be degraded in the lumen. This process has been characterized for the catabolite-induced degradation of the gluconeogenic enzyme fructose-1,6-bisphosphatase in the presence of glucose, and sequestration is thought to involve translocation into the completed vesicle. An alternate pathway for degradation of fructose-1,6-bisphosphatase by the ubiquitin-proteasome system has also been described.[312]

Vam3: A syntaxin homolog needed for the fusion of autophagosomes with the vacuole.[313]

VAMP3: A SNARE protein that facilitates the fusion of MVBs with autophagosomes to generate amphisomes.[314]

VAMP7: VAMP7 is a SNARE protein that colocalizes with Atg16L1 vesicles and phagophores, and is required, along with syntaxin 7, syntaxin 8 and Vti1b, for autophagosome formation.[315] VAMP7 is also involved in the maturation of autophagosomes by facilitating fusion with the lysosome.[314]

VAMP8: A SNARE protein that, in conjunction with Vti1b, is needed for the fusion of autophagosomes with lysosomes.[316]

VCP (valosin-containing protein/p97): A AAA^+-ATPase that is required for autophagosome maturation under basal conditions or when the proteasomal system is impaired; mutations of VCP result in the accumulation of immature, acidified autophagic vacuoles that contain ubiquitinated substrates.[317,318] See also Cdc48.

Verteporfin: An FDA-approved drug; used in photodynamic therapy, but it inhibits the formation of autophagosomes in vivo without light activation.[319]

VMP1 (vacuole membrane protein 1): A membrane protein that localizes to the plasma membrane or the ER. Interacts with Beclin 1 and is required for macroautophagy.[320,321]

Vps34: The class III PtdIns 3-kinase that generates PtdIns(3)P, which is required for macroautophagy. In yeast, Vps34 is present in two complexes. Complex I consisting of Vps34, Vps15, Vps30/Atg6 and Atg14 is essential for macroautophagy. Complex II composed of Vps34, Vps15, Vps30/Atg6 and Vps38 acts in the vacuolar protein sorting (Vps) pathway. In mammalian cells there are at least three PtdIns 3-kinase complexes that include Vps34, Vps15, Atg14L and Beclin 1, and combinations of UVRAG, Ambra1 and/or Rubicon.

Vps38: A component of the class III PtdIns 3-kinase complex II, which directs it to function in the vacuolar protein sorting (Vps) pathway.

VSM (vacuolar sequestering membranes): Extensions/protrusions of the vacuole limiting membrane along the surface of peroxisomes that occur during micropexophagy.[322]

Vti1: A soluble SNARE that, together with Sec18/NSF, is needed for the fusion of autophagosomes with the vacuole.[274] In mammalian cells, the SNARE proteins VAMP8 and Vti1b mediate the fusion of antimicrobial and canonical autophagosomes with lysosomes.[323]

WIPI (WD repeat protein interacting with phosphoinositides): The WIPI proteins are putative mammalian homologs of yeast Atg18 and Atg21. There are four WIPI proteins in mammalian cells. WIPI1/WIPI49 and WIPI2 localize with LC3 and bind PtdIns(3)P.[324] WIPI2 is required for starvation-induced autophagy.[325]

wortmannin (WM): An inhibitor of PtdIns 3-kinases; it inhibits macroautophagy.[201]

WXXL motif: An amino acid sequence present in proteins that allows an interaction with Atg8/LC3; the consensus is W/YXXL/I. Also see AIM and LIR/LRS.[326]

WYE-354: A catalytic mTOR inhibitor that increases macroautophagic flux to a greater level than allosteric inhibitors such as rapamycin (and may be used to induce macroautophagy in cell lines that are resistant to rapamycin and its derivatives); short-term treatment with WYE-354 can inhibit both mTORC1 and mTORC2, but the effects on flux are due to the former.[327] See also Ku-0063794.

XBP1: A component of the endoplasmic reticulum stress response that activates macroautophagy. The XBP1 yeast ortholog is Hac1.[328]

Xenophagy: Cell-autonomous innate immunity defense, whereby cells eliminate intracellular pathogens by capture into autophagosomes with subsequent killing.[329]

Xestospongin B: An antagonist of the IP_3R (inositol-1,4,5-trisphosphate receptor) that dissociates the inhibitory interaction between IP_3R and Beclin 1 and induces macroautophagy.[330]

Ykt6: A prenylated vesicle SNARE involved in Golgi transport and fusion with the vacuole (including Cvt vesicle delivery to the vacuole[331]); temperature sensitive *ykt6* mutations also prevent closure of the phagophore.[273]

Ypt1: A yeast GTP binding protein that functions in several forms of autophagy.[306] Ypt1 is needed for correct localization of Atg8 to the PAS. See also TRAPPIII. The mammalian homolog, Rab1, is required for autophagosome formation and for autophagic targeting of Salmonella.[332,333]

Ypt7: A yeast homolog of Rab7, needed for the fusion of autophagosomes with the vacuole.

ZIPK (zipper-interacting protein kinase): See Sqa.[334]

zymophagy: The selective degradation of activated zymogen granules by macroautophagy in a process dependent on VMP1, p62 and the ubiquitin protease USP9x.[335] See also crinophagy.

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