https://doi.org/10.15407/frg2023.05.395
UDC 577.121.2:57.04:632.954

# PARTICIPATION OF AUTOPHAGY IN THE RESPONSE OF PLANTS TO THE ACTION OF ABIOTIC STRESSORS

**ZH.Z. GURALCHUK[1], D.S. GURIANOV[1,2]**

*[1]Institute of Plant Physiology and Genetics, National Academy of Sciences of Ukraine*
*31/17 Vasylkivska St., Kyiv, 03022, Ukraine*
*e-mail: azhanna@ukr.net*
*[2]Institute of Molecular Biology and Genetics, National Academy of Sciences of Ukraine*
*150 Akademika Zabolotnoho St., Kyiv, 03143, Ukraine*

The data on the process of plant autophagy, its importance for the functioning of plants under optimal and stressful conditions are reviewed. Autophagy, a highly intricate and conserved process, remains at a basal level under optimal conditions, and is induced when plants fall under stress. This process stands as one of the central mechanisms enabling plants to adapt to adverse environmental factors. The activation of autophagy in response to various abiotic stressors, encompassing extreme temperatures, salinity, drought, nutrient deprivation, and herbicides is discussed. The regulatory mechanisms of autophagy are thoroughly considered, encompassing post-translational protein modifications crucial for its activation and progression, transcriptional regulation, and signaling through phytohormones. The pivotal role of autophagy activation is underscored in eliminating damaged organelles, and providing vital nutrients required for plant functionality and survival when exposed to abiotic stressors. Furthermore, the potential involvement of autophagy in herbicide-induced pathogenesis is discussed, with a dual perspective: its potential role in regulating programmed cell death (PCD), and its role in mitigating herbicide effects through the plant's homeostatic resistance mechanisms.

***Key words:*** autophagy, abiotic factors, herbicides.

Autophagy is a highly conserved process among eukaryotes. Under optimal conditions, it functions at a low basal level, and is essential for maintaining metabolic homeostasis. However, its activation is frequently observed in response to changes in environmental conditions. The triggers for such activation can encompass a range of stressful factors [1]. Autophagy ensures the degradation and recycling of various types of cellular components [1]. This process plays a crucial role in the survival of plants exposed to abiotic stressors. Autophagy is significantly important in plant metabolism and energy balance, and closely linked to maintaining stable plant productivity, especially in suboptimal conditions [2].

The term «autophagy» was initially introduced in 1963 by Christian de Duve to describe the process of degrading cytoplasmic components within the lysosome/vacuole [3]. A pivotal moment in autophagy research was when

Citation: Guralchuk Zh.Z., Gurianov D.S. Participation of autophagy in the response of plants to the action of abiotic stressors. *Fisiol. rast. genet.*, 2023, **55**, No. 5, pp. 395—416. https://doi.org/10.15407/frg2023.05.395

Yoshinori Ohsumi identified the first 15 genes related to autophagy, denoted as *ATG* (autophagy-related genes), through genetic screening using yeast [3—5]. Currently, more than 40 *ATG* genes have been identified in yeast, with the majority of them having close homologues in plants, from algae to vascular plants [6, 7]. Notably, *Arabidopsis thaliana* has approximately 40 *ATGs* [8] (some sources mention 47 [9]), rice (*Oryza sativa* L.) — 33 [10], and tea (*Camellia sinensis* (L.) O. Kuntze) — near 80 [11].

It's worth mentioning that, unlike yeast, many *ATG* genes in plants are organized into small families. It is assumed that genes within these families are induced differently in response to stressful conditions and developmental stages, allowing for finer regulation of autophagy activity [12]. In contrast to the ubiquitin-proteasome system, which involves nearly 1300 genes, constituting about 5 % of the *Arabidopsis* proteome [13], autophagy can target and degrade nearly all substrates or aggregates within cells, including proteins and their aggregates, redundant biomolecules, damaged organelles, and certain invasive microorganisms [14, 15].

There are three types of autophagy in plants: microautophagy, macroautophagy, and megaautophagy [16]. In microautophagy, cytoplasmic material is gathered on the surface of the vacuole, and enters it through the invagination of the tonoplast. Eventually, the tonoplast breaks down to release autophagic bodies, which are intravacuolar vesicles containing cytoplasmic material. In macroautophagy, the predominant and most studied form of autophagy in plants [17], biomaterial is insulated by cytoplasmic vesicles formed de novo. These vesicles originate from the expansion of the cup-shaped phagophore (or isolation membrane) surrounding the cytoplasm, which ultimately condenses to form an autophagosome surrounded by a double membrane [2]. The outer membrane of the autophagosome then merges with the tonoplast to release the inner vesicle as an autophagic body into the vacuole's lumen. In both micro- and macroautophagy, disruption of the autophagic cell membrane exposes its content to vacuolar hydrolases, which cleave this content into its constituent parts, subsequently transported back to the cytoplasm for reuse [18].

In megaautophagy, the tonoplast ruptures or becomes permeable, leading to the release of vacuolar hydrolases directly into the cytoplasm, where they degrade the cytoplasmic material in situ [16]. Megaautophagy often represents the final stage of programmed cell death during development (e.g., during xylogenesis) or in response to pathogen invasion (e.g., during the hypersensitivity reaction) [2].

Autophagy, hereafter referred to as macroautophagy, is a complex, stepwise, and dynamic process involving highly conserved *ATG* genes responsible for the initiation and formation of autophagosomes [19, 20]. ATG proteins can be categorized into four functional groups [20]: 1) The ATG1/ATG13 kinase complex, which triggers the formation of a phagophore in response to the phosphorylation status of ATG13; 2) The phosphatidylinositol-3-kinase complex (PI3K), responsible for incorporating phospholipids into the expanding phagophore; 3) The ATG2—ATG9—ATG18 complex, which plays a role in expanding the phagophore membrane and advancing the autophagosome; 4) The ATG8 and ATG12

conjugation systems, which catalyze the covalent binding of the ATG8 protein to phosphatidylethanolamine (PE) and are crucial for the expansion and maturation of the phagophore [21—27].

*ATG8* plays a central role in plant autophagy. In *Arabidopsis thaliana*, nine homologues of *ATG8* (*ATG8a—ATG8i*) have been described, each with different expression patterns and functions [19, 28]. In maise, there are five *ATG8* homologues [29], while rice has six [10], wheat has 14, and rapeseed (*Brassica napus* L.) has 22 [30].

ATG8-PE conjugates, found on both the outer and inner membranes of the autophagosome, contribute to phagophore expansion and vesicle closure during autophagosome formation. They also play a key role in recognizing autophagic materials, facilitated by adapter proteins and receptors interacting with ATG8 [6, 17, 31].

While autophagy was traditionally viewed as a process for the extensive degradation of cytoplasmic material [32], recent studies have revealed its capacity for selectivity, enabling quality control over specific organelles. These include chlorophagy (chloroplasts) [33], mitophagy (mitochondria) [22, 34], reticulophagy (endoplasmic reticulum) [35—38], pexophagy (peroxisomes) [39—41], ribophagy (ribosomes) [42—44], proteaphagy (proteasomes) [45, 46], as well as the clearance of protein aggregates [47—49]. Autophagy can be triggered both in response to various abiotic stress factors and for nutrient remobilization [1, 2, 50—52], as well as for defense against pathogens [53—56].

The selectivity of autophagy is adjusted by the interaction between ATG8 and specific autophagic receptors that contain ATG8-interacting motifs (AIM) or ubiquitin-interacting motifs (UIM) [17, 45, 57, 58]. These autophagic receptors mediate the selective autophagy of cell organelles, protein aggregates, and even certain pathogens [2].

**The influence of abiotic factors on plant autophagy.** Autophagy is induced by specific environmental factors to maintain a balance between plant growth and stress resistance [59]. It plays a crucial role in plant responses to various abiotic factors such as high and low temperatures, nutrient deficiency, salinity, drought, ultraviolet radiation, and more.

For instance, autophagy can be activated in response to ***elevated temperatures***. Numerous studies have demonstrated the accumulation of autophagosomes in *Arabidopsis* and tomato plants subjected to heat stress [47, 48, 60]. Notably, *Arabidopsis* mutants lacking key autophagy-related genes, *atg5* and *atg7*, displayed increased sensitivity to heat stress, manifested by heightened wilting, elevated electrolyte leakage, and reduced photosynthesis, in comparison to wild-type plants. Furthermore, *atg7* mutant plants exhibited the accumulation of insoluble protein aggregates labeled with ubiquitin [47]. Given that heat stress leads to protein misfolding and denaturation, it is hypothesized that it induces autophagy by triggering endoplasmic reticulum (ER) stress, which results in the accumulation of unfolded proteins within the ER, forming protein aggregates [36, 60]. These aggregates can be degraded by autophagy [48, 49].

It's worth noting that, under heat stress conditions in pepper plants (*Capsicum annuum* L.), the number of regulated *CaATG* genes, and the increase in autophagosomal punctate were more pronounced in the heat-

resistant line R9 compared to the heat-sensitive B6. This observation suggests a strong link between autophagy and tolerance to high temperatures [61].

Following heat stress, plants undergo a thermal recovery phase, during which various processes enable them to return to normal growth and development. It has been observed, that autophagy is induced by thermopriming (moderate heat stress), and remains elevated long after the stressor is removed [62]. Throughout the thermal recovery phase, in the absence of persistent heat stress, the numbers of autophagosomes and autophagic bodies progressively increase in *Arabidopsis thaliana*. This process has been demonstrated to aid in the degradation of HSP during the late stages of the thermal recovery phase, contributing to the resetting of cellular memory regarding heat stress [62].

Recent findings [63] indicate an unexpected role for the ATG8 protein in restoring the vacuolated Golgi apparatus after short-term acute heat stress. ATG8 has been shown to translocate to the extended Golgi apparatus membrane and participate in the recruitment of clathrin light chain 2 (CLC2) to the cisternal membrane, which, in turn, facilitates the reassembly of the Golgi apparatus.

***Low temperatures*** have been shown to increase the expression of *CaATG13* and induce the formation of autophagosomes in pepper plants [61]. Similarly, alfalfa (*Medicago sativa* L.) demonstrates an elevation in *MsATG13* expression in response to cold stress [64]. In tobacco plants overexpressing *MsATG13*, exposure to low temperatures resulted in a larger number of autophagosomes compared to wild-type plants [64]. This increase in autophagy is attributed to the activation of other *ATGs* necessary for autophagosome formation. Additionally, in transgenic tobacco plants with excessive expression of *MsATG13*, the levels of proline content and the activity of antioxidant enzymes increased in response to cold stress, while the levels of reactive oxygen species (ROS) decreased.

As previously mentioned, gene homologues within families can exhibit varying expression patterns under the influence of different stress factors. For example, in rice, three *ATG6* homologues display distinct responses to high, low temperatures, and drought. All three stress factors increase the expression of *OsATG6c*, while heat stress decreases the expression of *OsATG6a*, and cold stress reduces the expression of *OsATG6b* [65]. In contrast, drought leads to increased expression levels of all three homologues (*OsATG6a—OsATG6c*) in rice. Similarly, in Tatar buckwheat (*Fagopyrum tataricum*), all *FtATG8* homologues are inducibly expressed during drought, low temperature, and salt stress, exhibiting different expression patterns [9]. Among them, *FtATG8a* expression increases under drought conditions, while FtATG8e significantly decreases.

Changes in the expression levels of *CaATG* genes in response to various abiotic factors (high and low temperatures, drought, salt stress) have also been demonstrated in pepper plants [61]. This diversity of expression patterns may suggest multiple functions of homologous *ATG* genes, which could be linked to the variety of motifs present in ATG proteins [28].

Autophagy plays a pivotal role in promoting plant survival by maintaining cellular homeostasis under ***drought conditions*** [66]. Evidence demonstrates that *Arabidopsis atg5* and *atg7* mutants, as well as *RNAi*-

*ATG18a* plants where autophagy is blocked, exhibit heightened sensitivity to drought [47, 67]. Reduced expression of *ATG8d* and *ATG18h* in tomato (*Solanum lycopersicum*), and *ATG6* in wheat (*Triticum aestivum*), also increases their vulnerability to drought [68, 69]. Conversely, the overexpression of *ATG18a* in apple (*Malus domestica*) leads to increased autophagy, and enhanced drought resistance [70].

In apple plants, overexpressing *MdATG8i* under drought conditions, several positive changes were observed. These include a reduction in ROS levels compared to wild-type plants, an increase in the activity of antioxidant enzymes, a decrease in the accumulation of insoluble or oxidized proteins, and higher amounts of amino acids and flavonoids. Additionally, root water absorption improved, resulting in higher water use efficiency, contributing to increased drought resistance [71, 72]. Drought also triggers the activation of autophagy in tomatoes and wheat, leading to increased expression of *ATG* genes and enhanced formation of autophagosomes [68, 69, 73].

In peach (*Prunus persicae* L.), of the 23 autophagy-related genes identified, 20 responded to drought stress. Under the influence of drought, peach leaves exhibited suppressed photosynthetic processes and increased antioxidant enzyme activity. Autophagy and *ATG* expression were notably enhanced, particularly *PpATG8* and some *PpATG18* genes. Interestingly, the activity of antioxidant enzymes displayed an inverse correlation with the expression levels of *PpATG* [74].

*Medicago truncatula* possesses 39 autophagy-related genes (*ATG*), with most of them being strongly induced during seed development and in response to drought, highlighting the essential role of autophagy in both seed development and the plant’s response to drought stress [75].

Autophagy is involved in the degradation of aquaporins belonging to the PIP subfamily (plasma membrane intrinsic protein) during drought conditions, contributing to a reduction in water loss [66]. Using *Arabidopsis* plants as an example, it has been demonstrated that PIP 2;7 is degraded through autophagy, and this process is mediated by the selective autophagy receptor — tryptophan-rich sensory protein (TSPO). When autophagy is blocked using inhibitors or through the use of the *atg5* mutant, both TSPO and PIP 2;7 stabilization occurs [76].

In *Medicago truncatula*, the autophagic degradation of aquaporin PIP 2;7 follows a slightly different pathway. It involves the participation of dehydrin CAS31 (cold acclimation-specific 31), acting as a receptor for the selective autophagic degradation of PIP 2;7 during drought [77].

Recently, a plant-unique protein known as COST 1 (constitutively stressed 1) has been identified. This protein is essential for plant growth but can exert a negative regulatory influence on their resistance to drought through its direct interaction with the autophagy receptor protein ATG8e [78]. In *Arabidopsis thaliana* plants with overexpressed COST 1, autophagy is suppressed, rendering them hypersensitive to drought. Conversely, the *cost 1* mutant displays reduced growth but a heightened level of drought resistance [79].

It has been demonstrated that both MG132, an inhibitor of the 26S proteasome pathway, and concanamycin A, an inhibitor of the autophagy-mediated vacuolar degradation pathway, can hinder the degradation of

COST 1 during dehydration [79]. This observation suggests the involvement of both pathways in the efficient removal of COST 1, which eliminates its inhibition of autophagy and, in turn, enhances drought tolerance [78].

Under conditions of ***salt stress***, the expression of *MdATG10* in apple plants, especially in the roots, is induced [80]. Transgenic apple plants overexpressing *MdATG10* demonstrated increased salt tolerance, characterized by less impaired photosynthesis, improved growth, reduced $Na^+$ accumulation, and a lower $Na^+/K^+$ ratio compared to wild-type plants. Transgenic apple plants with overexpression of *MdATG8i* also exhibited an amelioration of negative effects caused by salt stress [81]. This improvement was accompanied by increased autophagic activity and the accumulation of proline, arginine, and polyamines, which play a crucial role in enhancing plant resistance to salt stress.

In *Arabidopsis*, salinity induces autophagy, with its peak occurring within 30 minutes of salt stress initiation [82]. Autophagy-defective mutants do not display such induction. During a 3-hour salt treatment, the accumulation of oxidized proteins decreases in the wild type; however, this reduction is not observed in autophagy-defective *atg2* or *atg7* mutants. Moreover, it has been demonstrated that autophagy is essential for sequestering $Na^+$ in the central vacuole of root cortex cells under salinity conditions [82].

Autophagy is enhanced under conditions of ***limited nutrient supply***, as it serves to remobilize nutrients in plants and provide cellular material. Depletion of nitrogen, inorganic phosphate (Pi), and limited sugar reserves are known triggers for autophagy in cultured cells, as demonstrated in the case of transformed BY-2 tobacco cells [83—86]. This indicates that such a response occurs at the cellular level [84]. In nutrient-deficient conditions, including Pi deficiency, the induction of autophagy in cultured BY-2 tobacco cells takes approximately 12 hours or even less, whereas intact plants require a significantly longer duration to initiate such a response [85]. Autophagy plays an important role in nitrogen transport and its assimilation. Overexpression of *ATG6* promoted autophagy and biomass accumulation in tomato plants under low nitrogen content in the nutrient medium [52]. At the same time, the nitrogen content in *atg6* mutant plants, the activity of nitrate reductase and nitrite reductase decreased, while their increase was observed in lines with overexpression of *ATG6* under low nitrogen supply.

Experiments conducted using autophagy-defective mutants have provided evidence of autophagy's involvement in nutrient remobilization, particularly in conditions where nutrient supply is limited [19, 51, 87]. In *Arabidopsis* plants, during nitrogen starvation, the expression of most *ATG8* genes is induced, except for *ATG8E* and *ATG8F* [88]. Notably, remobilization of $^{15}N$ in several *Arabidopsis atg* mutants significantly decreased compared to wild-type plants under varying nitrogen availability [87]. Furthermore, the *atg* mutants accumulated higher levels of ammonium, amino acids, and proteins in rosette leaves compared to wild-type plants, while displaying lower sugar content [51]. This observation suggests that

autophagy plays a pivotal role in controlling the C/N status and protein content in *Arabidopsis* leaves.

In the *atg5* mutant of *Arabidopsis*, it has been demonstrated that not only nitrogen but also sulfur remobilization from rosette leaves to seeds is impaired when compared to control lines [89]. Additionally, the efficiency of iron (Fe) translocation from vegetative organs to seeds is significantly reduced in the *atg5-1* mutant, even when *Arabidopsis* plants are adequately supplied with this element during seed formation [90].

In the *atg12* mutant of maize, under conditions of insufficient nitrogen nutrition, increased leaf senescence, impaired nitrogen remobilization, and reduced productivity were observed [91].

Recent studies have revealed that autophagy induced by Pi starvation is regulated by the ER stress response [92]. Additionally, evidence suggests that in *Arabidopsis thaliana*, ER-phagy facilitates Pi recycling during the initial stages of Pi starvation (2—3 days), while chlorophagy replenishes Pi reserves when phosphorus starvation is combined with a substantial (5-fold) nitrate supply to plants and a reduced C/N ratio [93—95].

It has been demonstrated that the expression of *Arabidopsis ATG8* genes (*AtATG8f* and *AtATG8h*) is significantly induced in the roots when exposed to low Pi supply. This regulation is closely associated with their promoter activity and can be suppressed in the phosphate mutant response 1 (*phr1*) [96]. The researchers propose that induction of *AtATG8f* and *AtATG8h* by Pi starvation may not directly facilitate Pi recycling but rather relies on a subsequent wave of transcriptional activation triggered by PHR1. This second phase fine-tunes cell type-specific autophagic activity. Further research is necessary to elucidate the mechanisms of autophagy activation and its role in plant responses to phosphorus starvation.

The lack of zinc also induces autophagy in plants, as demonstrated on the example of *Arabidopsis* [97]. Among all the members of the *ATG8* gene family, *ATG8d* responds most strongly to this factor. Researchers have shown that in the mutants *atg5-4* and *atg10-1*, a more significant increase in $O_2^{\bullet-}$ and $H_2O_2$ levels is observed under zinc deficiency compared to wild-type plants. In other words, autophagy suppresses the accumulation of ROS that form under conditions of limited zinc supply to plants.

Literature evidence suggests that intracellular self-degradation through autophagy in *Arabidopsis* plants replenishes the pool of zinc ions from proteins and organelles to increase the amount of usable zinc when it is scarce [98]. This replenishment of free zinc ions helps mitigate photosynthesis-related chlorosis caused by a Fenton-like reaction during zinc starvation in *Arabidopsis*. Overall, the increased expression of *ATG* genes and the formation of autophagic structures in response to nutrient deficiencies, including nitrogen, carbon, phosphorus, zinc, and others, suggest the involvement of autophagy in plant adaptation to mineral nutrient deficiencies [99].

There is compelling evidence in the literature that autophagy is triggered in photosynthesizing organisms in response to stress, often associated with increased formation of ***reactive oxygen species***, regardless of the source and location of ROS generation within the cell [100, 101]. Elevated ROS production can result from a different abiotic factors, including nutri-

ent deficiencies, drought, salinity, high and low temperatures, intense lighting, and exposure to herbicides. ROS can lead to the accumulation of damaged or toxic materials in cells, and the degradation of these materials occurs through the process of autophagy [102].

Significant oxidative stress and induction of autophagy were observed in *Arabidopsis* plants upon exposure to $H_2O_2$ [100], as well as in *Chlamydomonas* [103]. When treated with methylviologen (the herbicide paraquat), which is known to generate ROS, *Arabidopsis* seedlings with impaired autophagy (*RNAi-ATG18a*) exhibited stunted growth and pronounced discoloration compared to wild-type plants [100]. In the *RNAi-ATG18a* plants, unlike in the wild type, there was no accumulation of autophagosomes under the influence of methylviologen, and the level of oxidized proteins in these plants was significantly higher, suggesting the role of autophagy in degrading oxidized proteins during oxidative stress. Similar results were observed when rice mutant *atg10b* was treated with methylviologen [104]. It is worth noting that *Arabidopsis* mutants with overexpression of *ATG5* and *ATG7* showed increased resistance to methylviologen treatment compared to wild-type plants [105]. Under conditions inducing ROS with methylviologen (10 μM), *Nicotiana benthamiana* plants exhibited a 3.25-fold increase in basal autophagy activity, as determined by the number of structures labeled with CFP-ATG8f [106]. In carotenoid-deficient mutants of *Chlamydomonas reinhardtii*, an increase in ROS levels in chloroplasts was accompanied by a significant increase in autophagy [101]. Treatment of *Chlamydomonas reinhardtii* cells with the herbicide norflurazon, which inhibits phytoene desaturases and suppresses carotenoid synthesis, led to autophagy activation only in the presence of light. This suggests that autophagy is associated with photooxidative damage caused by ROS production in the presence of light [102].

Since microtubules are believed to play a role in autophagy, herbicides that cause their depolymerization by binding to tubulins may impact autophagy-related processes in plants. For example, the herbicides amiprofos-methyl and oryzalin significantly reduced the formation of autophagosomes and decreased the level of autophagy by approximately half [106].

It is known, that autophagy can be induced through the interaction of ROS and nitric oxide (NO) [107]. Therefore, data on the effect of the NO donor on the phytotoxic effects of herbicides, such as the acetyl-CoA-carboxylase inhibitor fenoxaprop [108], the protoporphyrinogen oxidase (PPO) inhibitor carfentrazone, and the synthetic auxin 2,4-D [109], provide indirect evidence of the possible involvement of programmed cell death, and specifically, autophagy, in the pathogenesis induced by these herbicides.

In the current literature, three types of programmed cell death (PCD) are considered: apoptosis (type 1), autophagy (type 2), and necrosis (type 3) [110]. While there is literature data on the role of programmed cell death in herbicide-induced pathogenesis [111], it remains unclear whether programmed cell death occurs via necrosis or autophagy. Simultaneously, evidence of the involvement of programmed cell death in herbicide-induced pathogenesis [112] has been obtained for both acetyl-CoA-carboxylase (ACC) inhibitor herbicides, whose action is

mediated by the formation of ROS [113, 114], and for ALS inhibitor herbicides, whose action is independent of ROS.

Direct evidence of the role of autophagy in determining the plants sensitivity to the action of ALS inhibitor herbicides has been obtained. Since ALS is a key enzyme in the biosynthesis pathway of branched-chain amino acids valine, leucine, and isoleucine, the activation of autophagy under the action of ALS inhibitor herbicides may be attributed to a deficiency of these amino acids. For instance, treatment of rapeseed plants with the ALS inhibitor herbicide tribenuron-methyl has been shown to deplete branched-chain amino acids in anthers, ultimately leading to autophagic cell death [115]. Activation of GCN2, a protein kinase that regulates mRNA translation, was observed under the influence of tribenuron-methyl in *Arabidopsis* plants [116, 117]. The activation of autophagy and GCN2 by tribenuron-methyl, as well as their reversal by exogenous valine, leucine, and isoleucine, suggest that the starvation of branched-chain amino acids induced by tribenuron-methyl is responsible for the activation of autophagy and GCN2 [117]. Genetic and biochemical analyses conducted by these researchers revealed a lower proportion of free branched-chain amino acids and more sensitive phenotypes in the single mutants *atg5*, *atg7*, and *gcn2* than in wild-type seedlings after herbicide treatment. Additionally, the lowest proportion of such amino acids and the most sensitive phenotypes were found in the double mutants *atg5 gcn2* and *atg7 gcn2*. It is likely that tribenuron-methyl-induced starvation for branched-chain amino acids activates autophagy, possibly through the inactivation of the target of rapamycin (TOR) [117].

Activation of autophagy in plants treated with herbicides containing ALS inhibitors can lead to an increase in the content of branched-chain amino acids, thereby weakening the phytotoxic effects of the herbicides. This contributes to the development of herbicide resistance, known as homeostatic tolerance [117], in addition to the previously recognized target and metabolic tolerance. Homeostatic tolerance provides cellular protection when exposed to non-lethal stress. However, it should be noted that the long-term effects of herbicides may eventually lead to autophagic cell death.

**Regulation of autophagy under the action of abiotic stresses.** Autophagy is a pivotal process for the adaptation of plants to adverse conditions. Its activation under stressful circumstances is essential for eliminating damaged organelles and recycling essential nutrients to ensure plant survival. This complex process involves a significant number of proteins, receptors, and signaling pathways. To safeguard plant cells, autophagy must be carefully and strictly regulated. The regulatory pathways controlling autophagy activation are still being unraveled. These pathways encompass post-translational protein modifications necessary for initiating and advancing autophagy, control over the protein stability of the autophagy machinery, and transcriptional regulation leading to changes in the expression of autophagy-related genes [20].

Post-translational modifications of key proteins involved in the plant autophagy mechanism include phosphorylation, ubiquitination, lipidization, S-sulfhydration, S-nitrosylation, and acetylation [118]. During these modifications, chemical groups such as phosphate, ubiquitin, persulfide,

acetate, methyl, and others covalently alter specific amino acids in target proteins, enabling changes in their functions, dynamics, and stability [20]. Serine and threonine residues are the most frequently phosphorylated, while tyrosine is less common [119]. In some instances, histidine and arginine residues can also undergo these modifications [120, 121].

A key role in the activation of autophagy is played by kinases, one of which is the TOR, and the other is SNF-related kinase 1 (sucrose nonfermenting 1-related protein kinase 1 (SnRK1)) [122]. TOR functions as a serine/threonine protein kinase and as a nutrient sensor that integrates multiple output signals [123, 124]. The antibiotic rapamycin suppresses its activity. Both of these kinase complexes are evolutionarily conserved among eukaryotes and coordinate many signaling pathways in response to energy deprivation and various stress conditions, including those that activate autophagy [125]. SnRK1 is activated by energy deficiency, often associated with stress, to restore homeostasis, while TOR is activated under nutrient-rich conditions to promote growth [125]. TOR controls the processes of plant growth and development under favorable conditions, while SnRK1 inhibits TOR with low supply of energy and resources, or in stressful conditions [126]. SnRK1 is a master regulator of responses to nutrient and energy deprivation, in particular, as evidenced by the fact that a double mutant in the two catalytic subunits of SnRK1 (KIN10 and KIN11) in *Arabidopsis* is lethal [127]. Transgenic *Arabidopsis* lines overexpressing *KIN10* (*KIN10-OE*) demonstrated induction of autophagy and autophagosome formation by affecting ATG1 phosphorylation, resulting in delayed leaf senescence and increased tolerance to nutrient starvation. In addition, *KIN10-OE* lines were less sensitive to drought and hypoxia compared to the wild type [128]. At the same time, the *kin10* mutant had a basal level of autophagy under normal conditions similar to wild-type plants, but the activation of autophagy by many abiotic stresses was blocked, indicating that SnRK1 is required for the induction of autophagy by a diversity of stress conditions [129].

SnRK1 is a positive regulator of autophagy. It activates this process by directly phosphorylating the ATG1 protein or by inhibiting TOR [128, 129]. Conversely, TOR, which responds to nutrient deficiency, acts as a negative regulator of autophagy in plants [124]. For instance, overexpression of TOR suppresses the induction of autophagy in nutrient-deprived conditions, as well as in the presence of salt and osmotic stress. However, it does not affect autophagy induction in response to oxidative stress or ER stress (which occurs when a large number of unfolded proteins accumulate under unfavorable conditions) [130, 131].

In addition to the TOR and SnRK1 kinases described above, other kinases, particularly which involved in the transmission of signals from brassinosteroids [132—135] or ABA [136—137], can also participate in the phosphorylation of substrates related to autophagy. Furthermore, protein phosphatases may play a role in these processes [138]. Recently, considerable research has been dedicated to persulfidation, a post-translational modification process in which cysteine thiol groups of proteins are converted to persulfide groups [139]. This modification extends to proteins involved in autophagy [140]. According to [141], 5214 proteins that have

undergone persulfide modification were identified in the roots of *Arabidopsis*. In *Arabidopsis*, the deficiency of DES1 (cytosolic L-cysteine desulfhydrase), responsible for cysteine degradation and hydrogen sulfide ($H_2S$) generation, leads to autophagy induction [142].

In *Arabidopsis*, an autophagy-related target protein that undergoes persulfidation is the Cys-protease ATG4, which cleaves the C-terminal extension of ATG8, is crucial for autophagosome formation [143]. As the intracellular ABA level increases, the persulfidation of ATG4 temporarily decreases, contributing to ATG8 processing and autophagy progression [143]. The persulfidation of ATG18a at C103 in *Arabidopsis* regulates autophagy under endoplasmic reticulum stress, and the disruption of this process affects both the number and size of autophagosomes [144]. The literature reports data on the negative regulation of autophagy by sulfide [142, 145—147]. However, the role of $H_2S$ as a negative or positive autophagy regulator may depend on specific abiotic factors and the potential involvement of other regulators in the autophagy signaling pathway [20, 144, 148].

Under the influence of stress factors, post-translational protein modifications ensure the rapid activation of autophagy. To support autophagic activity, plants increase the expression of *ATG* genes [20]. The correct expression of *ATG* genes is mediated by transcription factors. Correlation analysis between the expression of *FtATG8* and related transcription factor genes in buckwheat plants under drought and salt stress indicates that *FtATG8a*, *FtATG8c*, and *FtATG8g* exhibit a strong correlation with numerous stress-related transcription factors, suggesting that *FtATG8* may be regulated by transcription [9]. There is a high degree of correlation between the expression levels of *FtATG8* and most of the stress-related transcription factors, including *FtMYB10, FtMYB21, FtbHLH2, FtNAC2,* and *FtNAC6*, further indicating the potential involvement of *FtATG8* in salt stress and drought in Tatar buckwheat [9].

Spatio-temporal specificity of *FtATG8* response to abiotic factors (cold, drought, salinity) is evident. Some autophagy genes exhibit early responses, while others respond at a later stage. For instance, the maximum expression of *FtATG8a* and *FtATG8e* was observed after 3 hours, whereas other *FtATG8* genes, especially *FtATG8f*, showed their peak expression after 12 hours [9]. It is important to note that autophagy is a long-term regulatory process. In grape plants, for example, certain genes related to autophagy are induced after 20 days of drought [149].

In tomato (*Solanum lycopersicum*), silencing *WRKY33*, a representative of one of the largest families of transcription factors in plants [150], results in reduced expression of *ATG5, ATG7*, and *NBR1*. This disruption hampers the formation of autophagosomes, leading to an accumulation of insoluble protein aggregates and decreased plant resistance to elevated temperatures [48]. It is hypothesized that under the influence of elevated temperatures, WRKY33 and ATG proteins collaboratively regulate autophagy. This process is involved in the selective removal of heat-induced protein aggregates, contributing to the plant’s resistance to this abiotic factor. However, the precise interaction between ATG proteins and WRKY transcription factors necessitates further investigation and clarification [151].

WRKY33 can promote the selective degradation and recycling of protein aggregates by enhancing the expression of *NBR1* under elevated temperatures. *NBR1* (neighbour of *BRCA1* gene), the first identified receptor for selective autophagy, interacts with both ATG8 and ubiquitin, facilitating the encapsulation of ubiquitinated protein aggregates in autophagosomes [48, 152].

Another transcription factor, TGA9, serves as a positive regulator of autophagy, upregulating the expression of various *ATG* genes to enhance autophagy in *Arabidopsis* plants under osmotic stress [88]. Overexpression of *TGA9* activates autophagy in both control and stress conditions and amplifies the transcriptional expression of *ATG8B, ATG8E*, and other *ATG* genes by binding to their promoters.

BZR1 (brassinazole-resistant1) is a transcription factor activated by brassinosteroids. It acts as a positive regulator of the brassinosteroid signaling pathway and plays a role in the activation of autophagy [152]. This transcription factor can induce autophagy and the accumulation of NBR1, a receptor facilitating the selective autophagy of protein aggregates, in tomato plants under cold conditions [153]. BZR1 enhances the expression of *ATG2* genes and *ATG6* under the influence of cold and nitrogen starvation, as well as genes *NBR1* (*NBR1a* and *NBR1b*) under cold stress by directly binding to their promoters, thereby promoting autophagy [152, 153]. Consequently, the accumulation of insoluble protein aggregates decreases, resulting in increased resistance of tomato plants to cold and nitrogen starvation [152, 153]. However, it's worth noting that the activation and role of BZR1 in autophagy regulation may depend on the specific abiotic factor and the plant species [20]. Therefore, further research is necessary to draw more comprehensive conclusions.

In experiments with tomato plants, it was demonstrated that under drought conditions, autophagy is activated through the induction of the ethylene response transcription factor 5 (ERF5) [68]. Autophagy, facilitated in part by the binding of ERF5 (ethylene response factor 5) to the promoters of *ATG8d* and *ATG18h*, contributes to the ethylene-mediated drought tolerance of tomatoes [68].

The heat shock transcription factor, HsfA1a, plays a significant role in tomato drought tolerance, particularly by its involvement in the induction of autophagy [73]. HsfA1a-mediated autophagy, enabled by the binding of this factor to the promoters of *ATG10* and *ATG18f*, reduces the accumulation of insoluble ubiquitinated protein aggregates and enhances resistance to drought.

Numerous recent studies have highlighted the significance of hormonal signaling in the regulation of autophagy and plant responses to stress factors. However, it is essential to note that the molecular mechanisms governing such regulation still require further investigation [122].

In summary, autophagy is a complex and highly conserved process that plays a crucial role in the functioning of eukaryotes, both under optimal and stressful conditions. Autophagy is activated in response to various abiotic factors, and it participates in the processing and recycling of damaged biomolecules, organelles, aggregated proteins, and more. This, in turn, enables plants to survive and thrive under adverse environmental conditions.

Literature data indicate that the activation of autophagy occurs not only in response to abiotic factors such as high and low temperatures, drought, salinity, and nutrient deficiency but also in plants treated with herbicides. The action of herbicides, in particular, is associated with the production of reactive oxygen species, disorganization of photosynthesis, and inhibition of specific enzymes. The specific mechanisms by which herbicides influence the activation of autophagy and its role in programmed cell death still require further elucidation.

*REFERENCES*


1. Signorelli, S., Tarkowski, L.P., Van den Ende, W. & Bassham, D.C. (2019). Linking autophagy to abiotic and biotic stress responses. Trends in Plant Sci., 24, pp. 413-430. https://doi.org/10.1016/j.tplants.2019.02.001
2. Marshall, R.S. & Vierstra, R.D. (2018). Autophagy: the master of bulk and selective recycling. Annu. Rev. Plant Biol., 29, No. 69, pp. 173-208. https://doi.org/10.1146/annurev-arplant-042817-040606
3. Ohsumi, Y. (2014). Historical landmarks of autophagy research. Cell Res., 24, pp. 9-23. https://doi.org/10.1038/cr.2013.169
4. Matsuura, A., Tsukada, M., Wada, Y. & Ohsumi, Y. (1997). Apgip, a novel protein kinase required for the autophagic process in Saccharomyces cerevisiae. Gene, 192, pp. 245-250. https://doi.org/10.1016/S0378-1119(97)00084-X
5. Yoshimoto, K. & Ohsumi, Y. (2018). Unveiling the molecular mechanisms of plant autophagy — from autophagosomes to vacuoles in plants. Plant Cell Physiol., 59, No. 7, pp. 1337-1344. https://doi.org/10.1093/pcp/pcy112
6. Liu, Y. & Bassham, D.C. (2012). Autophagy: pathways for self-eating in plant cells. Annu. Rev. Plant Biol., 63, pp. 215-237. https://doi.org/10.1146/annurev-arplant-042811-105441
7. Chen, H., Dong, J. & Wang, T. (2021). Autophagy in plant abiotic stress management. Int. J. Mol. Sci., 22, 4075. https://doi.org/10.3390/ijms22084075
8. Chung, T. (2019). How phosphoinositides shape autophagy in plant cells. Plant Sci., 281, pp. 146-158. https://doi.org/10.1016/j.plantsci.2019.01.017
9. Fang, Y., Wang, S., Wu, H., Li, C., Zhao, H., Chen, H., Wang, X. & Wu, Q. (2022). Genome-wide identification of ATG gene family members in fagopyrum tataricum and their expression during stress responses. Int. J. Mol. Sci., 23, 14845. https://doi.org/10.3390/ijms232314845
10. Xia, K., Liu, T., Ouyang, J., Wang, R., Fan, T. & Zhang, M. (2011). Genome-wide identification, classification, and expression analysis of autophagy-associated gene homologues in rice (*Oryza sativa* L.). DNA Res., 18, pp. 363-377. https://doi.org/10.1093/dnares/dsr024
11. Huang, W., Ma, D.N., Liu, H.L., Luo, J., Wang, P., Wang, M.L., Guo, F., Wang, Y., Zhao, H. & Ni, D.J. (2020). Genome-wide identification of CsATGs in tea plant and the involvement of CsATG8e in nitrogen utilization. Int. J. Mol. Sci., 21, 7043. https://doi.org/10.3390/ijms21197043
12. Yang, X., & Bassham, D.C. (2015). New insight into the mechanism and function of autophagy in plant cells. Int. Rev. Cell Mol. Biol., 320, pp. 1-40. https://doi.org/10.1016/bs.ircmb.2015.07.005
13. Vierstra, R.D. (2003). The ubiquitin/26S proteasome pathway, the complex last chapter in the life of many plant proteins. Trends Plant Sci., 8, No. 3, pp. 135-142. https://doi.org/10.1016/S1360-1385(03)00014-1
14. Svenning, S. & Johansen, T. (2013). Selective autophagy. Essays Biochem., 55, pp. 79-92. https://doi.org/10.1042/bse0550079
15. Hafren, A., Macia, J.L., Love, A.J., Milner, J.J., Drucker, M. & Hofius, D. (2017). Selective autophagy limits cauliflower mosaic virus infection by NBR1-mediated targeting of viral capsid protein and particles. Proc. Natl Acad. Sci. USA, 114, E2026-E2035. https://doi.org/10.1073/pnas.1610687114

16. van Doorn, W.G. & Papini, A. (2013). The ultra-structure of autophagy in plant cells: a review. Autophagy, 9, pp. 1922-1936. https://doi.org/10.4161/auto.26275
17. Bu, F., Yang, M., Guo, X., Huang, W. & Chen, L. (2020). Multiple functions of ATG8 family proteins in plant autophagy. Front. Cell Dev. Biol., 8, 466. https://doi.org/10.3389/fcell.2020.00466
18. Soto-Burgos, J., Zhuang, X., Jiang, L. & Bassham, D.C. (2018). Dynamics of autophagosome formation. Plant Physiol., 176, pp. 219-229. https://doi.org/10.1104/pp.17.01236
19. Su, T., Li, X., Yang, M., Shao, Q., Zhao, Y., Ma, C. & Wang, P. (2020). Autophagy: an intracellular degradation pathway regulating plant survival and stress response. Front. Plant Sci., 11, 164. https://doi.org/10.3389/fpls.2020.00164
20. Agbemafle, W., Wong, M.M. & Bassham, D.C. (2023). Transcriptional and post-translational regulation of plant autophagy. J. Exp. Bot., 26. https://doi.org/10.1093/jxb/erad211
21. Suttangkakul, A., Li, F., Chung, T. & Vierstra, R.D. (2011). The ATG1/ATG13 protein kinase complex is both a regulator and a target of autophagic recycling in Arabidopsis. The Plant Cell, 23, pp. 3761-3779. https://doi.org/10.1105/tpc.111.090993
22. Li, F., Chung, T. & Vierstra, R.D. (2014). AUTOPHAGY-RELATED11 plays a critical role in general autophagy- and senescence-induced mitophagy in Arabidopsis. Plant Cell, 26, pp. 788-807. https://doi.org/10.1105/tpc.113.120014
23. Liu, F., Hu, W., Li, F., Marshall, R.S., Zarza, X., Munnik, T. & Vierstra, R.D. (2020). AUTOPHAGY-RELATED14 and its associated hosphatidylinositol 3-kinase complex promotes autophagy in Arabidopsis. Plant Cell, 32, pp. 3939-3960. https://doi.org/10.1105/tpc.20.00285
24. Bhati, K.K., Luong, A.M. & Batoko, H. (2021). VPS34 complexes in plants: untangled enough? Trends in Plant Sci., 26, pp. 303-305. https://doi.org/10.1016/j.tplants.2021.02.001
25. Zhuang, X., Chung, K.P., Cui, Y., Lin, W., Gao, C., Kang, B.-H. & Jiang, L. (2017). ATG9 regulates autophagosome progression from the endoplasmic reticulum in Arabidopsis. Proc. Natl Acad. Sci., USA, 114, E426-E435. https://doi.org/10.1073/pnas.1616299114
26. Kang, S., Shin, K.D., Kim, J.H. & Chung, T. (2018). Autophagy-related (ATG) 11, ATG9 and the phosphatidylinositol 3-kinase control ATG2-mediated formation of autophagosomes in Arabidopsis. Plant Cell Rep., 37, pp. 653-664. https://doi.org/10.1007/s00299-018-2258-9
27. Feng, Y., He, D., Yao, Z. & Klionsky, D.J. (2013). The machinery of macroautophagy. Cell Res., 24, pp. 24-41. https://doi.org/10.1038/cr.2013.168
28. Slavikova, S., Shy, G., Yao, Y., Glozman, R., Levanony, H., Pietrokovski, S., Elazar, Z. & Galili, G. (2005). The autophagy-associated Atg8 gene family operates both under favorable growth conditions and under starvation stress in Arabidopsis plants. J. Exp. Bot., 56, pp. 2839-2849. https://doi.org/10.1093/jxb/eri276
29. Chung, T., Suttangkakul, A. & Vierstra, R.D. (2009). The ATG autophagic conjugation system in maize: ATG transcripts and abundance of the ATG8-lipid adduct are regulated by development and nutrient availability. Plant Physiol., 149, pp. 220-234. https://doi.org/10.1104/pp.108.126714
30. Kellner, R., De la Concepcion, J.C., Maqbool, A., Kamoun, S. & Dagdas, Y.F. (2017). ATG8 Expansion: a driver of selective autophagy diversification? Trends in Plant Sci., 22(3), pp. 204-214. https://doi.org/10.1016/j.tplants.2016.11
31. Johansen, T. & Lamark, T. (2011). Selective autophagy mediated by autophagic adapter proteins. Autophagy, 7, pp. 279-296. https://doi.org/10.4161/auto.7.3.14487
32. Thompson, A.R. & Vierstra, R.D. (2005). Autophagic recycling: lessons from yeast help define the process in plants. Curr. Opin. Plant Biol., 8, pp. 165-173. https://doi.org/10.1016/j.pbi.2005.01.013
33. Izumi, M., Ishida, H., Nakamura, S. & Hidema, J. (2017). Entire photodamaged chloroplasts are transported to the central vacuole by autophagy. The Plant Cell, 29, pp. 377-394. https://doi.org/10.1105/tpc.16.00637

34. Nakamura, S., Hagihara, S., Otomo, K., Ishida, H., Hidema, J., Nemoto, T. & Izumi, M. (2021). Autophagy contributes to the quality control of leaf mitochondria. Plant & Cell Physiol., 62, pp. 229-247. https://doi.org/10.1093/pcp/pcaa162
35. Liu, Y., Burgos, J.S., Deng, Y., Srivastava, R., Howell, S.H. & Bassham, D.C. (2012). Degradation of the endoplasmic reticulum by autophagy during endoplasmic reticulum stress in Arabidopsis. The Plant Cell, 24, pp. 4635-4651. https://doi.org/10.1105/tpc.112.101535
36. Howell, S.H. (2013). Endoplasmic reticulum stress responses in plants. Annu. Rev. Plant Biol., 64, pp. 477-499. https://doi.org/10.1146/annurev-arplant-050312-120053
37. Chen, Y. & Yu, X. (2023). Endoplasmic reticulum stress-responsive microRNAs are involved in the regulation of abiotic stresses in wheat. Plant Cell Rep., 42, pp. 1433-1452. https://doi.org/10.1007/s00299-023-03040-7
38. Wang, J., Zhang, Q., Bao, Y. & Bassham, D.C. (2023). Autophagic degradation of membrane-bound organelles in plants. Biosci. Rep., 43, BSR20221204. https://doi.org/10.1042/BSR20221204
39. Kim, J., Lee, H., Lee, H.N., Kim, S.H., Shin, K.D. & Chung, T. (2013). Autophagy-related proteins are required for degradation of peroxisomes in Arabidopsis hypocotyls during seedling growth. Plant Cell, 25, pp. 4956-4966. https://doi.org/10.1105/tpc.113.117960
40. Lee, H.N., Kim, J. & Chung, T. (2014). Degradation of plant peroxisomes by autophagy. Front. Plant Sci., 8, No. 5, 139. https://doi.org/10.3389/fpls.2014.00139
41. Young, P.G. & Bartel, B. (2016). Pexophagy and peroxisomal protein turnover in plants. Biochim. Bio. Acta, 1863, 5, pp. 999-1005. https://doi.org/10.1016/j.bbamcr.2015.09.005
42. Floyd, B.E., Morriss, S.C., MacIntosh, G.C. & Bassham, D.C. (2015). Evidence for autophagy-dependent pathways of rRNA turnover in Arabidopsis. Autophagy, 11, pp. 2199-2212. https://doi.org/10.1080/15548627.2015.1106664
43. Bassham, D.C. & MacIntosh, G.C. (2017). Degradation of cytosolic ribosomes by autophagy-related pathways. Plant Sci., 262, pp. 169-174. https://doi.org/10.1016/j.plantsci.2017.05.008
44. Kazibwe, Z., Lium, A.Y., MacIntosh, G.C. & Bassham, D.C. (2019). The ins and outs of autophagic ribosome turnover. Cells, 8, No. 12, 1603. https://doi.org/10.3390/cells8121603
45. Marshall, R.S., Li, F., Gemperline, D.C., Book, A.J. & Vierstra, R.D. (2015). Autophagic degradation of the 26S proteasome is mediated by the dual ATG8/ubiquitin receptor RPN10 in Arabidopsis. Mol. Cell, 58, pp. 1053-1066. https://doi.org/10.1016/j.molcel.2015.04.023
46. Xiong, Q., Feng, R., Fischer, S., Karow, M., Xiong, M., Meβling, S., Nitz, L., Müller, S., Clemen, C.S., Song, N., Li, P., Wu, C. & Eichinger, L. (2023). Proteasomes of autophagy-deficient cells exhibit alterations in regulatory proteins and a marked reduction in activity. Cells, 12, No. 11, 1514. https://doi.org/10.3390/cells12111514
47. Zhou, J., Wang, J., Cheng, Y., Chi, Y.J., Fan, B., Yu, J.Q. & Chen, Z. (2013). NBR1-mediated selective autophagy targets insoluble ubiquitinated protein aggregates in plant stress responses. PLoS Genet., 9, e1003196. https://doi.org/10.1371/journal.pgen.1003196
48. Zhou, J., Wang, J., Yu, J.Q. & Chen, Z. (2014). Role and regulation of autophagy in heat stress responses of tomato plants. Front. Plant Sci., 5, 174. https://doi.org/10.3389/fpls.2014.00174
49. Zhang, Y. & Chen, Z. (2020). Broad and complex roles of NBR1-mediated selective autophagy in plant stress responses. Cells, 9, No. 12, 2562. https://doi.org/10.3390/cells9122562
50. Wada, S., Hayashida, Y., Izumi, M., Kurusu, T., Hanamata, S., Kanno, K., Kojima, S., Yamaya, T., Kuchitsu, K., Makino, A. & Ishida, H. (2015). Autophagy supports biomass production and nitrogen use efficiency at the vegetative stage in rice. Plant Physiol., 168, 1, pp. 60-73. https://doi.org/10.1104/pp.15.00242
51. Guiboileau, A., Avila-Ospina, L., Yoshimoto, K., Soulay, F., Azzopardi, M., Marmagne, A., Lothier, J. & Masclaux-Daubresse, C. (2013). Physiological and metabolic consequences of autophagy deficiency for the management of nitrogen and protein resources

in Arabidopsis leaves depending on nitrate availability. New Phytol., 199, pp. 683-694. https://doi.org/10.1111/nph.12307

52. Cao, J., Zheng, X., Xie, D., Zhou, H., Shao, S. & Zhou, J. (2022). Autophagic pathway contributes to low-nitrogen tolerance by optimizing nitrogen uptake and utilization in tomato. Hort. Res., 9, uhac068. https://doi.org/10.1093/hr/uhac068
53. Liu, Y., Schiff, M., Czymmek, K., Talloczy, Z., Levine, B. & Dinesh-Kumar, S.P. (2005). Autophagy regulates programmed cell death during the plant innate immune response. Cell, 121, pp. 567-577. https://doi.org/10.1016/j.cell.2005.03.007
54. Kwon, S.I., Cho, H.J., Kim, S.R. & Park, O.K. (2013). The Rab GTPase RabG3b positively regulates autophagy and immunity-associated hypersensitive cell death in Arabidopsis. Plant Physiol., 161, pp. 1722-1736. https://doi.org/10.1104/pp.112.208108
55. Leary, A.Y., Sanguankiattichai, N., Duggan, C., Tumtas, Y., Pandey, P., Segretin, M.E., Salguero Linares, J., Savage, Z.D., Yow, R.J. & Bozkurt, T.O. (2018). Modulation of plant autophagy during pathogen attack. J. Exp. Bot., 69, pp. 1325-1333. https://doi.org/10.1093/jxb/erx425
56. Jeon, H.S., Jang, E., Kim, J., Kim, S.H., Lee, M.H., Nam, M.H., Tobimatsu, Y. & Park, O.K. (2023). Pathogen-induced autophagy regulates monolignol transport and lignin formation in plant immunity. Autophagy, 19, pp. 597-615. https://doi.org/10.1080/15548627
57. Farré, J.C. & Subramani, S. (2016). Mechanistic insights into selective autophagy pathways: lessons from yeast. Nat. Rev. Mol. Cell Biol., 17, pp. 537-552. https://doi.org/10.1038/nrm.2016.74
58. Maqbool, A., Hughes, R.K., Dagdas, Y.F., Tregidgo, N., Zess, E., Belhaj, K., Round, A., Bozkurt, T.O., Kamoun, S. & Banfield, M.J. (2016). Structural basis of host autophagy-related protein 8 (ATG8) binding by the irish potato famine pathogen effector protein PexRD54. J. Biol. Chem., 291, 20270-20282. https://doi.org/10.1074/ jbc.M116.744995
59. Wang, P., Mugume, Y. & Bassham, D.C. (2018). New advances in autophagy in plants: regulation, selectivity and function. Seminars in Cell & Develop. Biol., 80, pp. 113-122. https://doi.org/10.1016/j.semcdb.2017.07
60. Yang, X., Srivastava, R., Howell, S.H. & Bassham, D.C. (2016). Activation of autophagy by unfolded proteins during endoplasmic reticulum stress. Plant J., 85, pp. 83-95. https://doi.org/10.1111/tpj.13091
61. Zhai, Y., Guo, M., Wang, H., Lu, J., Liu, J., Zhang, C., Gong, Z. & Lu, M. (2016). Autophagy, a conserved mechanism for protein degradation, responds to heat, and other abiotic stresses in Capsicum annuum L. Front. Plant Sci., 7, 131. https://doi.org/10.3389/fpls.2016.00131
62. Sedaghatmehr, M., Thirumalaikumar, V.P., Kamranfar, I., Marmagne, A., Masclaux-Daubresse, C. & Balazadeh, S. (2019). A regulatory role of autophagy for resetting the memory of heat stress in plants. Plant Cell Environ., 42, pp. 1054-1064. https://doi.org/10.1111/pce.13426
63. Zhou, J., Ma, J., Yang, C., Zhu, X., Li, J., Zheng, X., Li, X., Chen, S., Feng, L., Wang, P., Ho, M.I., Ma, W., Liao, J., Li, F., Wang, C., Zhuang, X., Jiang, L., Kang, B.H. & Gao C. (2023). A non-canonical role of ATG8 in golgi recovery from heat stress in plants. Nat. Plants, 9, pp. 749-765. https://doi.org/10.1038/s41477-023-01398-w
64. Zhao, W., Song, J., Wang, M., Chen, X., Du, B., An, Y., Zhang, L., Wang, D. & Guo, C. (2023). Alfalfa MsATG13 confers cold stress tolerance to plants by promoting autophagy. Int. J. Mol. Sci., 24, 12033. https://doi.org/10.3390/ijms241512033
65. Rana, R.M., Dong, S., Ali, Z., Huang, J. & Zhang, H.S. (2012). Regulation of ATG6/Beclin-1 homologs by abiotic stresses and hormones in rice (*Oryza sativa* L.). Genet. Mol. Res., 11, 3676-3687. https://doi.org/10.4238/2012.August.17.3
66. Tang, J. & Bassham, D.C. (2021). Autophagy during drought: function, regulation, and potential application. Plant J., 109 (2), pp. 390-401. https://doi.org/10.1111/tpj.15481
67. Liu, Y., Xiong, Y. & Bassham, D.C. (2009). Autophagy is required for tolerance of drought and salt stress in plants. Autophagy, 5, pp. 954-963. https://doi.org/10.4161/auto.5.7.9290
68. Zhu, T., Zou, L., Li, Y., Yao, X., Xu, F., Deng, X., Zhang, D. & Lin, H. (2018). Mitochondrial alternative oxidase-dependent autophagy involved in ethylene-mediated drought tolerance in Solanum lycopersicum. Plant Biotechnol. J., 16, pp. 2063-2076. https://doi.org/10.1111/pbi.12939

69. Li, Y.B., Cui, D.Z., Sui, X.X., Huang, C., Huang, C.Y. & Fan, Q.Q. (2019). Autophagic survival precedes programmed cell death in wheat seedlings exposed to drought stress. Int. J. Mol. Sci., 20, 5777. https://doi.org/10.3390/ijms20225777
70. Sun, X., Wang, P., Jia, X., Huo, L., Che, R. & Ma, F. (2017). Improvement of drought tolerance by overexpressing MdATG18a is mediated by modified antioxidant system and activated autophagy in transgenic apple. Plant Biotechnol. J., 16 (2), pp. 545-557. https://doi.org/10.1111/pbi.12794
71. Jia, X., Gong, X., Jia, X., Li, X., Wang, Y., Wang, P., Huo, L., Sun, X., Che, R., Li, T., Zou, Y. & Ma, F. (2021). Overexpression of MdATG8i enhances drought tolerance by alleviating oxidative damage and promoting water uptake in transgenic apple. Int. J. Mol. Sci., 22, 5517. https://doi.org/10.3390/ijms22115517
72. Jia, X., Mao, K., Wang, P., Wang, Y., Jia, X., Huo, L., Sun, X., Che, R., Gong, X. & Ma, F. (2021). Overexpression of MdATG8i improves water use efficiency in transgenic apple by modulating photosynthesis, osmotic balance, and autophagic activity under moderate water deficit. Hort. Res., 8, 81. https://doi.org/10.1038/s41438-021-00521-2
73. Wang, Y., Cai, S., Yin, L., Shi, K., Xia, X., Zhou, Y., Yu, J. & Zhou, J. (2015). Tomato HsfA1a plays a critical role in plant drought tolerance by activating ATG genes and inducing autophagy. Autophagy, 11, 2033-2047. https://doi.org/10.1080/15548627.2015.1098798
74. Wang, X., Gao, Y., Wang, Q., Chen, M., Ye, X., Li, D., Chen X., Li, L. & Gao, D. (2019). 24-Epibrassinolide-alleviated drought stress damage influences antioxidant enzymes and autophagy changes in peach (Prunus persicae L.) leaves. Plant Physiol. Bio., 135, pp. 30-40. https://doi.org/10.1016/j.plaphy.2018.11.026
75. Yang, M., Wang, L., Chen, C., Guo, X., Lin, C., Huang, W. & Chen, L. (2021). Genome-wide analysis of autophagy-related genes in Medicago truncatula highlights their roles in seed development and response to drought stress. Sci. Rep., 11, 22933. https://doi.org/10.1038/s41598-021-02239-6
76. Hachez, C., Veljanovski, V., Reinhardt, H., Guillaumot, D., Vanhee, C., Chaumont, F. & Batoko, H. (2014). The Arabidopsis abiotic stress-induced TSPO-related protein reduces cell-surface expression of the aquaporin PIP2;7 through protein-protein interactions and autophagic degradation. Plant Cell, 26, pp. 4974-4990. https://doi.org/10.1105/tpc.114.134080
77. Li, X., Liu, Q., Feng, H., Deng, J., Zhang, R., Wen, J., Dong, J. & Wang, T. (2020). Dehydrin MtCAS31 promotes autophagic degradation under drought stress. Autophagy, 16, pp. 862-877. https://doi.org/10.1080/15548627.2019.1643656
78. Bao, Y. (2020). Links between drought stress and autophagy in plants. Plant Signaling & Behavior, 15, 1779487. https://doi.org/10.1080/15592324.2020.1779487
79. Bao, Y., Song, W.-M., Wang, P., Yu, X., Li, B., Jiang, C., Shiu, S.H., Zhang, H. & Bassham, D.C. (2020). COST1 regulates autophagy to control plant drought tolerance. Proc. Natl Acad. Sci., 117, pp. 7482-7493. https://doi.org/10.1073/pnas.1918539117
80. Huo, L., Guo, Z., Jia, X., Sun, X., Wang, P., Gong, X. & Ma, F. (2020). Increased autophagic activity in roots caused by overexpression of the autophagy-related gene MdATG10 in apple enhances salt tolerance. Plant Sci., 294, 110444. https://doi.org/10.1016/j.plantsci.2020
81. Huo, L., Guo, Z., Wang, P., Zhang, Z., Jia, X., Sun, Y., Sun, X., Gong, X. & Ma, F. (2020). MdATG8i functions positively in apple salt tolerance by maintaining photosynthetic ability and increasing the accumulation of arginine and polyamines. Env. Exp. Bot., 172, 103989. https://doi.org/10.1016/j.envexpbot.2020.103989
82. Luo, L., Zhang, P., Zhu, R., Fu, J., Su, J., Zheng, J., Wang, Z., Wang, D. & Gong, Q. (2017). Autophagy is rapidly induced by salt stress and is required for salt tolerance in Arabidopsis. Front. Plant Sci., 8, 1459. https://doi.org/10.3389/fpls.2017.01459
83. Moriyasu, Y. & Ohsumi, Y. (1996). Autophagy in tobacco suspension-cultured cells in response to sucrose starvation. Plant Physiol., 111, pp. 1233-1241. https://doi.org/10.1104/pp.111.4.1233
84. Tasaki, M., Asatsuma, S. & Matsuoka, K. (2014). Monitoring protein turnover during phosphate starvation-dependent autophagic degradation using a photoconvertible fluorescent protein aggregate in tobacco BY-2 cells. Front. Plant Sci., 5. https://doi.org/10.3389/fpls.2014.00172

85. Toyooka, K., Takeuchi, M., Moriyasu, Y., Fukuda, H. & Matsuoka, K. (2006). Protein aggregates are transported to vacuoles by macroautophagic mechanism in nutrient-starved plant cells. Autophagy, 2, pp. 91-106. https://doi.org/10.4161/auto.2.2.2366
86. Takatsuka, Ch., Inoue, Yu., Higuchi, T., Hillmer, S., Robinson, D.G. & Moriyasu, Yu. (2011). Autophagy in tobacco BY-2 cells cultured under sucrose starvation conditions: isolation of the autolysosome and its characterization. Plant Cell Physiol., 52 (12), pp. 2074-2087. https://doi.org/10.1093/pcp/pcr13
87. Guiboileau, A., Yoshimoto, K., Soulay, F., Bataille, M.P., Avice, J.C. & Masclaux-Daubresse, C. (2012). Autophagy machinery controls nitrogen remobilization at the whole-plant level under both limiting and ample nitrate conditions in Arabidopsis. New Phytol., 194, pp. 732-740. https://doi.org/10.1111/j.1469-8137.2012.04084.x
88. Wang, P., Nolan, T.M., Yin, Y. & Bassham, D.C. (2020). Identification of transcription factors that regulate ATG8 expression and autophagy in Arabidopsis. Autophagy, 16, pp. 123-139. https://doi.org/10.1080/15548627.2019.1598753
89. Lornac, A., Haveé, M., Chardon, F., Soulay, F., Clément, G., Avice, J.-Ch. & Masclaux-Daubresse, C. (2020). Autophagy controls sulphur metabolism in the rosetta leaves of arabidopsis and facilitates S remobilization that the seeds. Cells, 9, 332. https://doi.org/10.3390/cells9020332
90. Pottier, M., Dumont, J., Masclaux-Daubresse, C. & Thomine, S. (2019). Autophagy is essential for optimal translocation of iron to seeds in Arabidopsis. J. Exp. Bot., 70, pp. 859-869. https://doi.org/10.1093/jxb/ery388
91. Li, F., Chung, T., Pennington, J.G., Federico, M.L., Kaeppler, H.F., Kaeppler, S.M., Otegui, M.S. & Vierstra, R.D. (2015). Autophagic recycling plays a central role in maize nitrogen remobilization. Plant Cell, 27, pp. 1389-1408. https://doi.org/10.1105/tpc.15.00158
92. Naumann, C., Müller, J., Sakhonwasee, S., Wieghaus, A., Hause, G., Heisters, M., Bürstenbinder, K. & Abel, S. (2019). The local phosphate deficiency response activates endoplasmic reticulum stress-dependent autophagy. Plant Physiol., 179, pp. 460-476. https://doi.org/10.1104/pp.18.01379
93. Yoshitake, Y., Nakamura, S., Shinozaki, D., Izumi, M., Yoshimoto, K., Ohta, H. & Shimojima, M. (2021). RCB-mediated chlorophagy caused by oversupply of nitrogen suppresses phosphate-starvation stress in plants. Plant Physiol., 185, pp. 318-330. https://doi.org/10.1093/plphys/kiaa030
94. Yoshitake, Y., Shinozaki, D. & Yoshimoto, K. (2022). Autophagy triggered by iron-mediated ER stress is an important stress response to the early phase of Pi starvation in plants. Plant J., 110, pp. 1370-1381. https://doi.org/10.1111/tpj.15743
95. Yoshitake, Y. & Yoshimoto, K. (2023). Intracellular phosphate recycling systems for survival during phosphate starvation in plants. Front. Plant Sci., 13, 1088211. https://doi.org/10.3389/fpls.2022.1088211
96. Lin, L.Y., Chow, H.X., Chen, C.H., Mitsuda, N., Chou, W.C. & Liu, T.Y. (2023). Role of autophagy-related proteins ATG8f and ATG8h in the maintenance of autophagic activity in Arabidopsis roots under phosphate starvation. Front. Plant Sci., 14, 1018984. https://doi.org/10.3389/fpls.2023.1018984
97. Eguchi, M., Kimura, K., Makino, A. & Ishida, H. (2017). Autophagy is induced under Zn limitation and contributions to Zn-limited stress tolerance in Arabidopsis (Arabidopsis thaliana). Soil Sci. Plant Nutr., 63, pp. 342-350. https://doi.org/10.1080/00380768.2017.1360750
98. Shinozaki, D., Merkulova, E.A., Naya, L., Horie, T., Kanno, Y., Seo, M., Ohsumi, Y., Masclaux-Daubresse, C. & Yoshimoto, K. (2020). Autophagy increases zinc bioavailability to avoid light-mediated reactive oxygen species production under zinc deficiency. Plant Physiol., 182, pp. 1284-1296. https://doi.org/10.1104/pp.19.01522
99. Wang, J., Miao, S., Liu, Y. & Wang, Y. (2022). Linking autophagy to potential agronomic trait improvement in crops. Int. J. Mol. Sci., 23, 4793. https://doi.org/10.3390/ijms23094793
100. Xiong, Y., Contento, A.L., Nguyen, P.Q. & Bassham, D.C. (2007). Degradation of oxidized proteins by autophagy during oxidative stress in Arabidopsis. Plant Physiol., 143, pp. 291-299. https://doi.org/10.1104/pp.106.092106

101. Pérez-Pérez, M.E., Couso, I., Crespo, J.L. (2012). Carotenoid deficiency triggers autophagy in the model green alga Chlamydomonas reinhardtii. Autophagy, 8, pp. 376-388. https://doi.org/10.4161/auto.18864
102. Pérez-Pérez, M.E., Lemaire, S.D. & Crespo, J.L. (2012). Reactive oxygen species and autophagy in plants and algae. Plant Physiol., 160, pp. 156-164. https://doi.org/10.1104/pp.112.199992
103. Perez-Perez, M.E., Florencio, F.J. & Crespo, J.L. (2010). Inhibition of target of rapamycin signaling and stress activate autophagy in Chlamydomonas reinhardtii. Plant Physiol., 152, pp. 1874-1888. https://doi.org/10.1104/pp.109.152520
104. Shin, J.-H., Yoshimoto, K., Ohsumi, Y., Jeon, J.-S. & An, G. (2009). OsATG10b, an autophagosome component, is required for cell survival against oxidative stress in rice. Mol. Cells, 27, pp. 67-74. https://doi.org/10.1007/s10059-009-0006-2
105. Minina, E.A., Moschou, P.N., Vetukuri, R.R., Sanchez-Vera, V., Cardoso, C., Liu, Q., Elander, P.H., Dalman, K., Beganovic, M., Lindberg Yilmaz, J., Marmon, S., Shabala, L., Suarez, M.F., Ljung, K., Novák, O., Shabala, S., Stymne, S., Hofius, D. & Bozhkov, P.V. (2018). Transcriptional stimulation of rate-limiting components of the autophagic pathway improves plant fitness. J. Exp. Bot., 69, 1415-1432. https://doi.org/10.1093/jxb/ery010
106. Wang, Y., Zheng, X., Yu, B., Han, S., Guo, J., Tang, H., Yu, A.Y., Deng, H., Hong, Y. & Liu, Y. (2015). Disruption of microtubules in plants suppresses macroautophagy and triggers starch excess-associated chloroplast autophagy. Autophagy, 11, pp. 2259-2274. https://doi.org/10.1080/15548627.2015.1113365
107. Serrano, I., Romero-Puertas, M.C., Sandalio, L.M. & Olmedilla, A. (2015). The role of reactive oxygen species and nitric oxide in programmed cell death associated with selfincompatibility. J. Exp. Bot., 66, pp. 2869-2876. https://doi.org/10.1093/jxb/erv083
108. Sychuk, A., Radchenko, M. & Morderer, Y. (2013). The increase of phytotoxic action of graminicide fenoxaprop-P-ethyl by NO donor sodium nitroprusside. Sci. Educat. New Dimen.: Nat. Tech. Sci., 2, 21. https://seanewdim.com/uploads/3/4/5/1/34511564/sychuk_a._radchenko_m._morderer_e._the_increase_of_phytotoxic_action_of_graminicide_fenoxaprop-p-ethyl_by_no_donor_sodium_nitrpruside.pdf
109. Ponomaryova, I.G., Khandezhina, M.V. & Radchenko, M.P. (2022). Increase in the phytotoxic action of the protoporphyrinogen oxidase inhibitor herbicide carfentrazone and the herbicide of the class of synthetic auxins 2,4-D when used together with the NO donor sodium nitroprusside. Fiziol. rast. genet., 54, No. 5, pp. 419-428 [in Ukrainian]. https://doi.org/10.15407/frg2022.05.419
110. Lockshin, R.A. & Zakeri, Z. (2004). Apoptosis, autophagy, and more. Int. J. Biochem. Cell Biol., 36, pp. 2405-2419. https://doi.org/10.1016/j.biocel.2004.04.011
111. Morderer, Y.Y., Radchenko, M.P. & Sychuk, A.M. (2013). Programmed cell death in the pathogenesis, induced by herbicides in plants. Fiziol. rast. genet., 45, No. 6, pp. 517-526 [in Ukrainian]. http://dspace.nbuv.gov.ua/handle/123456789/159373
112. Radchenko, M.P., Gurianov, D.S. & Morderer, Ye.Yu. (2022). DNA fragmentation and endonuclease activity under the effect of herbicides acetyl-CoA-carboxylase and acetolactate synthase inhibitors. Fiziol. rast. genet., 54, No. 5, pp. 404-418 [in Ukrainian]. https://doi.org/10.15407/frg2022.05.404
113. Morderer, Ye.Yu., Palanytsia, M.P. & Rodzevych, O.P. (2008). Research on the participation of free radical oxidation reactions in the development of the phytotoxic action of graminicides. Physiol. biochim. cult. rast., 40, No. 1, pp. 56-61 [in Ukrainian].
114. Palanytsia, M.P., Trach, V.V. & Morderer, Ye.Yu. (2009). Generation of active forms of oxygen under the action of graminicides and modifiers of their activity. Physiol. biochim. cult. rast., 41, pp. 328-334 [in Ukrainian].
115. Zhao, L., Jing, X., Chen, L., Liu, Y., Su, Y., Liu, T., Gao, C., Yi, B., Wen, J., Ma, C., Tu, J., Zou, J., Fu, T. & Shen, J. (2015). Tribenuron-methyl induces male sterility through anther-specific inhibition of acetolactate synthase leading to autophagic cell death. Mol. Plant, 8, pp. 1710-1724. https://doi.org/10.1016/j.molp.2015.08.009
116. Lokdarshi, A. & von Arnim, A.G. (2022). Review: emerging roles of the signaling network of the protein kinase GCN2 in the plant stress response. Plant Sci., 320, 111280. https://doi.org/10.1016/j.plantsci.2022.111280

117. Zhao, L., Deng, L., Zhang, Q., Jing, X., Ma, M., Yi, B., Wen, J., Ma, Ch., Tu, J., Fu, T. & Shen, J. (2018). Autophagy contributes to sulfonylurea herbicide tolerance via GCN2-independent regulation of amino acid homeostasis. Autophagy, 14 (4), pp. 702-714. https://doi.org/10.1080/15548627.2017.1407888
118. Qi, H., Xia, F.N. & Xiao, S. (2021). Autophagy in plants: physiological roles and post-translational regulation. J. Int. Plant Biol., 63, pp. 161-179. https://doi.org/10.1111/jipb.12941
119. Champion, A., Kreis, M., Mockaitis, K., Picaud, A. & Henry, Y. (2004). Arabidopsis kinome: after the casting. Functional & Integrative Genomics, 4, pp. 163-187. https://doi.org/10.1007/s10142-003-0096-4
120. Durek, P., Schmidt, R., Heazlewood, J.L., Jones, A., MacLean, D., Nagel, A., Kersten, B. & Schulze, W.X. (2010). PhosPhAt: the arabidopsis thaliana phosphorylation site database. An update. Nucleic Acids Res., 38, D828-D834. https://doi.org/10.1093/nar/gkp810
121. van Wijk, K.J., Friso, G., Walther, D. & Schulze, W.X. (2014). Meta-analysis of Arabidopsis thaliana phospho-proteomics data reveals compartmentalization of phosphorylation motifs. The Plant Cell, 26, pp. 2367-2389. https://doi.org/10.1105/tpc.114.125815
122. Liao, C.-Y. & Bassham, D.C. (2019). Combating stress: the interplay between hormone signaling and autophagy in plants. J. Exp. Bot., 71, pp. 1723-1733. https://doi.org/10.1093/jxb/erz515
123. Hunter, T. (1995). When is a lipid kinase not a lipid kinase? When it is a protein kinase. Cell, 83, pp. 1-4. https://doi.org/10.1016/0092-8674(95)90225-2
124. Wu, Y., Shi, L., Li, L., Fu, L., Liu, Y., Xiong, Y. & Sheen, J. (2019). Integration of nutrient, energy, light, and hormone signaling via TOR in plants. J. Exp. Bot., 70, pp. 2227-2238. https://doi.org/10.1093/jxb/erz028
125. Margalha, L., Confraria, A. & Baena-González, E. (2019). SnRK1 and TOR: modulating growth—defense trade-offs in plant stress responses. J. Exp. Bot., 70, pp. 2261-2274. https://doi.org/10.1093/jxb/erz066
126. Retzer, K. & Weckwerth, W. (2023). Recent insights into metabolic and signaling events of directional root growth regulation and its implications for sustainable crop production systems. Front. Plant Sci., 14, 1154088. 10.3389/fpls.2023.1154088
127. Baena-Gonzalez, E., Rolland, F., Thevelein, J.M. & Sheen, J. (2007). A central integrator of transcription networks in plant stress and energy signaling. Nature, 448, pp. 938-942. https://doi.org/10.1038/nature06069
128. Chen, L., Su, Z.Z., Huang, L., Xia, F.N., Qi, H., Xie, L.J., Xiao, S. & Chen, Q.F. (2017). The AMP-activated protein kinase KIN10 is involved in the regulation of autophagy in Arabidopsis. Front. Plant Sci, 8, 1201. https://doi.org/10.3389/fpls.2017.01201
129. Soto-Burgos, J. & Bassham, D.C. (2017). SnRK1 activates autophagy via the TOR signaling pathway in Arabidopsis thaliana. PloS One, 12, e0182591. https://doi.org/10.1371/journal.pone.0182591
130. Pu, Y., Luo, X. & Bassham, D.C. (2017). TOR-dependent and -independent pathways regulate autophagy in arabidopsis thaliana. Front. Plant Sci., 8, 1204. https://doi.org/10.3389/fpls.2017.01204
131. Pu, Y.T., Soto-Burgos, J. & Bassham, D.C. (2017) Regulation of autophagy through SnRK1 and TOR signaling pathways. Plant Signal. Behav., 12, e1395128. https://doi.org/10.1080/15592324.2017.1395128
132. Nolan, T.M., Brennan, B., Yang, M., Chen, J., Zhang, M., Li, Z., Wang, X., Bassham, D.C., Walley, J., & Yin, Y. (2017). Selective autophagy of BES1 mediated by DSK2 balances plant growth and survival. Develop. Cell, 41, No. 33-46, e7. https://doi.org/10.1016/j.devcel.2017.03.013
133. Montes, C., Wang, P., Liao. C.-Y., Nolan, T.M., Song, G., Clark, N.M., Elmore, J.M., Guo, H., Bassham, D.C., Yin, Y. & Walley, J.W. (2022). Integration of multi-omics data reveals interplay between brassinosteroid and target of rapamycin complex signaling in Arabidopsis. New Phytol., 236, pp. 893-910. https://doi.org/10.1111/ nph.18404
134. Liao, C.-Y., Pu, Y., Nolan, T.M., Montes, C., Guo, H., Walley, J.W. & Yin, Y. (2023). Brassinosteroids modulate autophagy through phosphorylation of RAPTOR1B by

the GSK3-like kinase BIN2 in Arabidopsis. Autophagy, 19, pp. 1293-1310. https://doi.org/10.1080/15548627.2022.2124501
135. Zhang, B., Shao, L., Wang, J., Zhang, Y., Guo, X., Peng, Y., Cao, Y. & Lai, Z. (2021). Phosphorylation of ATG18a by BAK1 suppresses autophagy and attenuates plant resistance against necrotrophic pathogens. Autophagy, 17, pp. 2093-2110. https://doi.org/10.1080/15548627.2020.1810426
136. Wang, P., Zhao, Y., Li, Z., Hsu, C.C., Liu, X., Fu, L., Hou, Y.J., Du, Y., Xie, S., Zhang, C., Gao, J., Cao, M., Huang, X., Zhu, Y., Tang, K., Wang, X., Tao, W.A., Xiong, Y. & Zhu, J.K. (2018). Reciprocal regulation of the TOR kinase and ABA receptor balances plant growth and stress response. Mol. Cell, 69, pp. 100-112, e6. https://doi.org/10.1016/j.molcel.2017.12.002
137. Rodriguez, M., Parola, R., Andreola, S., Pereyra, C. & Martínez-Noel, G. (2019). TOR and SnRK1 signaling pathways in plant response to abiotic stresses: do they always act according to the «yin-yang» model? Plant Sci., 288, 110220. https://doi.org/10.1016/j.plantsci.2019.110220
138. Ahn, C.S., Ahn, H.-K. & Pai, H.-S. (2015). Overexpression of the PP2A regulatory subunit Tap46 leads to enhanced plant growth through stimulation of the TOR signaling pathway. J. Exp. Bot., 66, pp. 827-840. https://doi.org/10.1093/jxb/eru438
139. Aroca, A., Serna, A., Gotor, C. & Romero, L.C. (2015). S-sulfhydration: a cysteine posttranslational modification in plant systems. Plant Physiol., 168, pp. 334-342. https://doi.org/10.1104/pp.15.00009
140. Aroca, A., Benito, J.M., Gotor, C. & Romero, L.C. (2017). Persulfidation proteome reveals the regulation of protein function by hydrogen sulfide in various biological processes in Arabidopsis. J. Exp. Bot., 68, pp. 4915-4927. https://doi.org/10.1093/jxb/erx294
141. Jurado-Flores, A., Romero, L.C. & Gotor, C. (2021). Label-free quantitative proteomic analysis of nitrogen starvation in arabidopsis root reveals new aspects of $H_2S$ signaling by protein persulfidation. Antioxidants (Basel), 10, 508. https://doi.org/10.3390/antiox10040508
142. Gotor, C., García, I., Crespo, J.L. & Romero, L.C. (2013). Sulfide as a signaling molecule in autophagy. Autophagy, 9, pp. 609-611. https://doi.org/10.4161/auto.23460
143. Gotor, C., Aroca, A. & Romero, L.C. (2022). Persulfidation is the mechanism underlying sulfide-signaling of autophagy. Autophagy, 18, pp. 695-697. https://doi.org/10.1080/15548627.2021.1936357
144. Aroca, A., Yruela, I., Gotor, C. & Bassham D.C. (2021). Persulfidation of ATG18a regulates autophagy under ER stress in Arabidopsis. Proc. Natl Acad. Sci. USA, 118, e2023604118. https://doi.org/10.1073/pnas.2023604118
145. Álvarez, C., García, I., Moreno, I., Pérez-Pérez, M.E., Crespo, J.L., Romero, L.C. & Gotor, C. (2012). Cysteine-generated sulfide in the cytosol negatively regulates autophagy and modulates the transcriptional profile in Arabidopsis. Plant Cell, 24, pp. 4621-4634. https://doi.org/10.1105/tpc.112.105403
146. Laureano-Marín, A.M., Moreno, I., Romero, L.C. & Gotor, C. (2016). Negative regulation of autophagy by sulfide is independent of reactive oxygen species. Plant Physiol., 171, pp. 1378-1391. https://doi.org/10.1104/pp.16.00110
147. Jurado-Flores, A., Aroca, A., Romero, L.C. & Gotor, C. (2023). Sulfide promotes tolerance to drought through protein persulfidation in Arabidopsis. J. Exp. Bot., 74, pp. 4654-4669. https://doi.org/10.1093/jxb/erad165
148. Xuan, L., Wu, H., Li, J., Yuan, G., Huang, Y., Lian, C., Wang, X., Yang, T. & Wang, C. (2022). Hydrogen sulfide reduces cell death through regulating autophagy during submergence in Arabidopsis. Plant Cell Rep., 41, 1531-1548. https://doi.org/10.1007/s00299-022-02872-z
149. Shangguan, L., Fang, X., Chen, L., Cui, L. & Fang, J. (2018). Genome-wide analysis of autophagy-related genes (ARGs) in grapevine and plant tolerance to copper stress. Planta, 247, pp. 1449-1463. https://doi.org/10.1007/s00425-018-2864-3
150. Chen, X., Li, C., Wang, H. & Guo, Z. (2019). WRKY transcription factors: evolution, binding, and action. Phytopathol. Res., 1, 13. https://doi.org/10.1186/s42483-019-0022-x
151. Song, I., Hong, S. & Huh, S.U. (2022). Identification and expression analysis of the Solanum tuberosum StATG8 family associated with the WRKY transcription factor. Plants (Basel), 11, 2858. https://doi.org/10.3390/plants11212858

152. Wang, Y., Cao, J.-J., Wang, K.-X., Xia, X.-J., Shi, K., Zhou, Y.-H., Yu, J.-Q. & Zhou, J. (2019). BZR1 mediates brassinosteroid-induced autophagy and nitrogen starvation in tomatoes. Plant Physiol., 179, pp. 671-685. https://doi.org/10.1104/pp.18.01028
153. Chi, C., Li, X., Fang, P., Xia, X., Shi, K., Zhou, Y., Zhou, J. & Yu, J. (2020). Brassinosteroids act as a positive regulator of NBR1-dependent selective autophagy in response to chilling stress in tomato. J. Exp. Bot., 71, pp. 1092-1106. https://doi.org/10.1093/jxb/erz466

Received 26.10.2023

## УЧАСТЬ АУТОФАГІЇ У РЕАКЦІЇ РОСЛИН НА ДІЮ АБІОТИЧНИХ СТРЕСОРІВ

*Ж.З. Гуральчук[1], Д.С. Гур'янов[1,2]*

[1]Інститут фізіології рослин і генетики Національної академії наук України
03022 Київ, вул. Васильківська, 31/17
e-mail: azhanna@ukr.net
[2]Інститут молекулярної біології і генетики Національної академії наук України
03143 Київ, вул. Академіка Заболотного, 150

Розглядаються процеси, пов'язані з аутофагією рослин, її значення для функціонування рослин за оптимальних і стресових умов. Аутофагія є складним висококонсервативним процесом, активність якого знаходиться на базовому рівні за оптимальних умов та індукується за стресових. Вона є одним із ключових процесів адаптації рослин до дії несприятливих чинників середовища. Описується активація аутофагії у рослин у відповідь на дію різних абіотичних чинників (високих і низьких температур, засолення, посухи, нестачі поживних речовин, гербіцидів). Обговорюються механізми регуляції аутофагії, пов'язані з посттрансляційними модифікаціями білків, необхідних для активації та прогресування аутофагії, регуляцією транскрипції та фітогормональною сигналізацією. Підкреслюється значення активації аутофагії для видалення пошкоджених органел і реутилізації поживних речовин, необхідних для функціонування і виживання рослин за дії абіотичних чинників. Припускається можлива участь аутофагії в індукованому гербіцидами патогенезі. Зазначається, що роль аутофагії в індукованому гербіцидами патогенезі, з одного боку, може бути пов'язана з її участю у регуляції програмованої загибелі клітин (ПЗК), з іншого — з послабленням їх дії, зумовленим гомеостатичною резистентністю рослин.

*Ключові слова:* аутофагія, абіотичні чинники, гербіциди.

**ORCID**

**Zh.Z. GURALCHUK** — https://orcid.org/0009-0009-8869-7378

**D.S. GURIANOV** — https://orcid.org/0000-0003-0594-2286