1 COMMENTARY

2 The lowdown on breakdown: Open questions in plant proteolysis

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52 Abstract

53 Proteolysis, including post-translational proteolytic processing as well as protein degradation and
54 amino acid recycling, is an essential component of the growth and development of living organisms.
55 In this article, experts in plant proteolysis pose and discuss compelling open questions in their areas
56 of research. Topics covered include the role of proteolysis in the cell cycle, DNA damage response,
57 mitochondrial function, the generation of N-terminal signals (degrons) that mark many proteins for
58 degradation (N-terminal acetylation, the Arg/N-degron pathway, and the chloroplast N-degron
59 pathway), developmental and metabolic signaling (photomorphogenesis, abscisic acid and
60 strigolactone signaling, sugar metabolism, and post-harvest regulation), plant responses to
61 environmental signals (endoplasmic-reticulum associated degradation, chloroplast-associated
62 degradation, drought tolerance, the growth-defense tradeoff)), and the functional diversification of
63 peptidases. We hope these thought-provoking discussions help to stimulate further research.

64 **One-sentence summary:** Expert opinions on major open questions in plant proteolysis research.

65 Introduction

66 **(By Nancy A. Eckardt, Editor)**

67 Proteolysis is Nature's way of keeping house. While some people can function quite happily in a
68 house full of disorganized piles of a lifetime of accumulated stuff, an organism's ability to thrive and
69 reproduce depends on highly functioning proteolytic systems to keep the "stuff" (i.e. proteins) in
70 check. More than just "housekeeping," proteolytic systems serve as "house managers" – not only
71 degrading proteins to prevent their overaccumulation and recycle amino acids but also carrying out
72 regulatory processing of proteins to alter or fine-tune critical pathways in growth, development, and
73 responses to environmental signals. Regulation of protein half-life as well as proteolytic processing
74 as a post-translational modification are prevalent mechanisms that modulate protein function and
75 ensure proper protein stoichiometries throughout the proteome.

76 Proteolytic processing occurs through a wide range of mechanisms in eukaryotic cells. In
77 addition to a plethora of individual peptidases located in different cellular compartments, major
78 routes for protein turnover include the ubiquitin-proteasome system (UPS), which operates
79 principally in the cytosol and nucleus, and the delivery of proteins, protein complexes, and
80 organelles to the vacuole for degradation. The two major routes of delivery of cellular components

to the vacuole are autophagy and endocytosis. Chloroplasts and mitochondria also maintain independent degradation systems (van Wijk 2015). There are numerous associated pathways for targeting and delivering proteins, protein complexes, and whole organelles bound for degradation to the appropriate destination. In this commentary, researchers working on different aspects of plant proteolysis address major open questions in their field of expertise. We acknowledge that the topics covered represent only a small fraction of the proteolytic events taking place in plant cells, and we apologize to readers whose favorite proteases or proteolytic systems were left out.

Questions addressed

Proteolysis and cell biology

- What is the role of the F-box protein FBL17 in the G1/S phase transition in Arabidopsis?
- What is the role of autophagy in the plant DNA damage response?
- How do proteolytic networks regulate mitochondrial function?

N-terminal signals for degradation pathways

- What is the effect of N-terminal acetylation on protein half-life?
- The Arg/N-degron pathways of protein turnover: Boutique or bulk?
- What are the degrons and molecular players in the chloroplast N-degron pathway?

Roles of proteolysis in developmental and metabolic signaling

- How do plants use ubiquitin-mediated proteolysis to regulate photomorphogenesis?
- How does proteolysis of core signaling components occur in different subcellular locations to modulate the ABA pathway?
- How does the D14 receptor function as both receptor and enzyme, linking hormone perception to protein degradation?
- Who takes the lead in the intricate dance between autophagy and sugar metabolism?
- What is the role of proteolysis in fruit ripening regulation?

Roles of proteolysis in plant responses to biotic/abiotic signals

- How does ERAD function in model plants and crops?
- How is chloroplast-associated protein degradation (CHLORAD) regulated in response to developmental and environmental cues?
- How does autophagy contribute to drought tolerance?
- How does the fine-tuning of proteasome regulation impact the trade-off between growth and defense?
- Why are there so many peptidases in plants, particularly in the subtilase family?

The UPS

The UPS tags and delivers proteins to the 26S proteasome, an ATP-dependent, multi-catalytic protease complex that degrades proteins in both the cytoplasm and nucleus (Raffeiner et al. 2023). A specialized pathway of the UPS, endoplasmic-reticulum-associated degradation (ERAD), minimizes the accumulation of damaged or misfolded proteins in the ER. Entry to the UPS begins when a protein is modified by the attachment of Ubiquitin (Ub), a 76 amino acid protein that is highly

119 conserved in all eukaryotes. Target proteins are ubiquitylated through the combined activity of E1
120 Ub-activating enzymes, E2 Ub-conjugating enzymes, and E3 Ub-ligases. The E3 ligase recognizes
121 and binds the target protein. The E1 binds Ub in an ATP-dependent manner and transfers it to an E2.
122 The E2 binds the E3 ligase and transfers Ub to the target protein directly or, for some E3s, Ub is
123 transferred to the E3 and then to the target. Ubiquitylation of a target protein marks it for degradation
124 via the 26S proteasome (Ciechanover et al. 2000). Ub may be attached to a target protein as a
125 monomer or as a linear ubiquitin chain, formed by linkages between one of 7 conserved lysine
126 residues. Polyubiquitylation through Ub lysine residue 48 (K48) is one of the main recognition signals
127 for the UPS.

128 Well over 1000 E3 ligases have been identified in plants (Al-Saharin et al. 2022, Saxena et al.
129 2023). Single subunit E3 ligases can be classified into three or four types: HECT (Homologous to
130 E6AP C-Terminus), RING-finger (Really Interesting New Gene), U-box (sometimes classified as a
131 subset of the RING-type), and RBR (Ring-Between-Ring). Cullin-RING E3 ligases (CRLs) constitute
132 a single large family of multi-subunit E3 ligases. Plants include all of these types, but the largest
133 family is the CRLs. CRLs are further divided into several different types depending on the cullin
134 (CUL) scaffold (Li et al. 2023b). The largest CRL grouping is the SCF complex, composed of an F-
135 box protein (FBP) that functions in target recognition and 3 core subunits (CUL1 as the major
136 scaffold unit, RBX1, which binds the E2 Ub-conjugating enzyme, and Skp1/ASK1/2, which
137 recognizes and binds the FBP). The FBP protein defines the SCF complex, and the large number of
138 SCFs is due to the diversity of FBPs. The FBP family represents one of the largest families of
139 regulatory proteins in plants, with many species including hundreds of FBP-encoding genes. For
140 example, the Arabidopsis (*Arabidopsis thaliana*) and rice (*Oryza sativa*) genomes encode ~700 and
141 970 FBPs, respectively (Saxena et al. 2023).

142 N-degron pathways

143 In general, substrate proteins carry a degradation signal known as a degron that is sufficient for
144 recognition and degradation by the proteolytic machinery. Degrons are heterogeneous sequences
145 that can be located anywhere in the protein, can act in a *cis* or *trans* mode, and can also be
146 generated by post-translational modifications to specific amino acid residues. N-terminal degrons (N-
147 degrons) are the most studied. They are formed by N-degron pathways, previously referred to as "N-
148 end rule pathways." Plants have several different N-degron pathways; some of the best studied are
149 the Arg/N- and Ac/N-degron pathways, involving the creation of an N-degron through arginylation or
150 acetylation of the N-terminal residue, respectively (Holdsworth et al. 2020). N-degrons are
151 recognized by other proteins, called N-recognins, specific to each route of degradation. Many N-
152 recognins are Ub E3 ligases, targeting proteins for degradation via the UPS, but links between N-
153 recognins and autophagy have also been reported (Holdsworth et al. 2020).

154 Vaculoar degradation

155 Vacuolar degradation takes place in the large central vacuole called the lytic vacuole, which typically
156 occupies up to 90% of the plant cell volume (Stefan et al. 2013). Proteins and other cargo molecules

are transported to the lytic vacuole via multiple routes; the major routes of delivery are autophagy of cytoplasmic cargo (Marshall and Vierstra 2018, Tang and Bassham 2018) and endocytosis of plasma membrane proteins (Valencia et al. 2016). In plants, there are two main types of autophagy: microautophagy and macroautophagy. Macroautophagy is the best understood and involves the formation of a membrane structure called a phagophore around cargo proteins, which develops into a double-membrane autophagosome. The autophagosome outer membrane fuses with the tonoplast and releases the cargo into the vacuole for degradation. In microautophagy, cytoplasmic components are taken up by the vacuole through the invagination of the tonoplast. A third type, known as mega-autophagy (also called autolysis), occurs when vacuolar hydrolases are released directly into the cytoplasm (often the final stage of programmed cell death). In addition to its role in the UPS, Ub is involved in autophagy: whereas K48-linked polyubiquitylation targets proteins for the UPS, K63-linked polyubiquitylation is known, among other functions, to mark cargo for degradation by autophagy (Raffeiner et al. 2023).

Endocytosis regulates turnover of plasma membrane proteins (such as receptors and transporters), transporting cargo through the endomembrane system in single-membrane vesicles for delivery to the vacuole or recycling back to the plasma membrane (Fan et al. 2015). There is also substantial overlap between these pathways, for example, cross-regulation between the UPS and autophagy pathways (Su et al. 2020b) and between autophagy and endocytosis (Zhang et al. 2019a; Zhuang et al. 2015).

Peptidases for limited proteolysis

In addition to the proteasome, hundreds of peptidases function in all compartments of the cell including the extracellular matrix, and play roles in almost every aspect of plant development and in the interaction of plants with their biotic and abiotic environment. Among the most abundant peptidases are cysteine, serine, and aspartic proteases, which are named for the amino acid residue that serves as the nucleophile for catalysis, and metalloproteases, which use a polarized water molecule for nucleophilic attack of peptide bonds (van der Hoorn 2008). Some peptidases contribute to protein turnover by non-selective degradation and others perform limited proteolysis of substrate proteins at highly specific sites. Limited proteolysis may result in a loss, gain, or change in activity; it may affect protein assembly and subcellular targeting, and as part of the maturation process, controls the activity of enzymes, regulatory proteins, and signaling peptides (Schaller et al. 2018, Stührwohldt and Schaller 2019). Because peptidases irreversibly modify the structure and function of cognate substrate proteins, their activity is tightly regulated at multiple levels (Fernández-Fernández et al. 2023). Diversification with respect to substrate proteins, cleavage site recognition and mechanism of regulation may have contributed to present-day abundance of peptidases in plants, as discussed below for subtilases, one of the largest families of serine peptidases.

Proteolysis and cell biology: The cell cycle, DNA damage response, and mitochondrial function

194 ## FBL17: A proteolytic engine for the G1/S phase transition?

195 **(By Pascal Genschik and Sandra Noir)**

196 Progression through the cell cycle phases depends on cyclin-dependent kinase (CDK) activity
197 (Budirahardja and Gönczy 2009). In plants, this activity is conferred by A- and B-type CDKs, which
198 are activated by multiple cyclins to permit DNA replication and mitosis (Harashima et al. 2013).
199 CDKA;1 is the main regulator of the G1/S transition, whereas CDKBs are necessary for mitosis
200 (Polyn et al. 2015). At mitotic exit, CDK activity drops and stays low in G1, enabling the licensing of
201 replication origins. This is achieved by several mechanisms working collaboratively, including the
202 selective degradation of mitotic cyclins by the UPS (Mocciaro and Rape 2012). Ubiquitylation of
203 mitotic cyclins is mediated in all eukaryotes by the conserved anaphase-promoting complex or
204 cyclosome (APC/C) Ub E3 ligase (Pesin and Orr-Weaver 2008, Genschik et al. 2014, Willems and
205 De Veylder 2022). CDK activity is also inhibited by the binding of cyclin-dependent kinase inhibitor
206 (CKI) proteins (Besson et al. 2008). In plants, two classes of CKIs carrying distinct functions have
207 been described, called KIP-RELATED PROTEINS (KRPs) and SIAMESE-RELATED proteins
208 (SMRs) (Churchman et al. 2006, Acosta et al. 2011). It was proposed that KRPs mainly play a role in
209 the G1 checkpoint by inhibiting CDKA;1–CYCD complexes, whereas SMR members play a
210 prominent role during endoreplication (Van Leene et al. 2010, Kumar et al. 2015).

211 To re-enter the S phase and release CDK activity, cells need to decrease the level of CKI
212 proteins. In mammals, the UPS plays a fundamental role in cell cycle control and the DNA damage
213 response (DDR) by destroying CKIs. Two families of mammalian CKIs (INK4 and CIP/KIP) play
214 distinct cellular functions and are degraded by diverse types of E3s in both the nucleus and
215 cytoplasm (Starostina and Kipreos 2012). One of them is the SCF^{Skp2} complex, which plays a
216 prominent role in cell cycle control (Carrano et al. 1999, Sutterlüty et al. 1999, Frescas and Pagano
217 2008). The F-box protein Skp2 recognizes many substrates involved in cell cycle control and the
218 DDR (reviewed in Frescas and Pagano 2008). Not surprisingly, with such a repertoire of substrates,
219 Skp2 is involved in multiple aspects of different human cancers and is defined as an oncogene (Chan
220 et al. 2010).

221 With hundreds of publications describing the elaborate multi-task functions of mammalian Skp2,
222 one may wonder whether such a crucial Ub E3 ligase would be conserved in the green lineage. Here
223 we discuss the Arabidopsis F-box protein FBL17, which shows many similarities with mammalian
224 Skp2 (**Table 1**), but for which much work is still required to fully grasp its cellular functions. *FBL17*
225 was initially identified as an essential gene needed for male germ cell division in Arabidopsis, with a
226 phenotype similar to the loss-of-function of *CDKA;1* (Kim et al. 2008, Gusti et al. 2009). FBL17
227 appears to function in the degradation of KRPs, supported by the stabilization of KRP6 in *fbl17* single
228 germ cells (Kim et al. 2008), whereas different *krp* mutations at least partially rescued the *fbl17* pollen
229 phenotype (Gusti et al. 2009, Zhao et al. 2012). The function of FBL17 is however not restricted to
230 germ cells.

231 *FBL17* is expressed in the S-phase in synchronized plant cell cultures (Menges et al. 2003,
232 Trolet et al. 2019). In line with this expression pattern, *FBL17* is a direct transcription target of
233 E2Fa-DPa (a transcription factor associated with cell proliferation) and is repressed by the binding
234 of RETINOBLASTOMA-RELATED 1 (RBR1), an Arabidopsis homolog of the Retinoblastoma (Rb)
235 proteins in mammals, to its promoter (Gusti et al. 2009, Zhao et al. 2012) (**Fig. 1**). Viable *fbl17* null
236 mutant plants were identified at very low frequency, and their molecular and cellular
237 characterization revealed major cell cycle defects (Noir et al. 2015). In particular, *FBL17* loss-of-
238 function drastically reduced cell proliferation and also fully suppressed endoreplication (Noir et al.
239 2015). Such a phenotype could potentially be explained by a strong accumulation of KRPs that can
240 block S-phase CDK activity (Verkest et al. 2005b), and would be consistent with the phenotypic
241 resemblance of *fbl17* and *cdka;1* null mutant plants (Noir et al. 2015, Nowack et al. 2012).
242 Accordingly, the KRP2 protein steady-state level was found to increase in *fbl17* mutants (Noir et al.
243 2015). Another study, using Arabidopsis plants in which *FBL17* function is inhibited by an inducible
244 microRNA, also provided evidence for a degradative role during G2 of the F-box protein in the
245 turnover of free, but not chromosomal bound, KRP4 proteins (D'Ario et al. 2021). Interestingly,
246 meristems with inhibited *FBL17* had abnormally large cells, suggesting that excess free KRP4
247 disrupts cell size homeostasis. This raises the question of whether *FBL17* loss-of-function
248 phenotypes could be explained solely by an impaired degradation of KRPs. The answer is likely
249 no. Strong KRP2-overexpressing lines resemble *flb17* mutant plants in many respects, but also
250 show significant differences. The upregulation of numerous cell cycle and DNA damage genes
251 observed in *fbl17* (Noir et al. 2015, Gentric et al. 2020), also suggests that like mammalian Skp2,
252 FBL17 has a broader range of substrates and functions.

253 In line with this assumption, it was found that *fbl17* mutants are hypersensitive to double-strand
254 break (DSB)-induced genotoxic stress (Gentric et al. 2020). Note that while in mammals the Rb-
255 related protein p130 is degraded by SKP2 (Tedesco et al. 2002), whether FBL17 also targets plant
256 RBR1 remains unknown. Even in the absence of genotoxic stress, *fbl17* mutants exhibit a higher
257 frequency of DNA lesions and increased cell death in the root meristem. It was further shown that
258 FBL17 colocalizes with RBR1 at DNA damage sites, but its substrates and function at this
259 subcellular location remain unknown (Gentric et al. 2020). It is noticeable that in response to DSBs,
260 mammalian Skp2 is required for the activation and recruitment of the Ataxia-telangiectasia mutated
261 (ATM) kinase to DNA damage foci via non-proteolytic K63-dependent ubiquitylation (Wu et al.
262 2012). FBL17 was also recently implicated in DNA replication stress, as it was found that the
263 hypersensitivity to hydroxyurea of a null mutant of the ATM and Rad3-related (ATR) kinase can be
264 suppressed by the *fbl17* mutation (Pan et al. 2021b). Importantly, this study revealed that WEE1, a
265 conserved kinase induced by ATR during replication stress (De Schutter et al. 2007), directly
266 phosphorylates FBL17 and promotes its polyubiquitylation and subsequent degradation by the
267 proteasome (Pan et al. 2021b) (**Fig. 1**). Interestingly, human Wee1 is also able to phosphorylate
268 and destabilize Skp2 at least in a human cell line, supporting the conservation of this mechanism
269 (Pan et al. 2021b). It was later shown that FBL17 is ubiquitylated by the APC/C^{CDC20} E3 (Pan et al.
270 2023) and that WEE1 not only phosphorylates FBL17, but also the APC10 subunit of this Ub E3

271 ligase, enhancing the interaction between the APC/C substrate adaptor, CDC20, and FBL17. As
272 the chemical inhibition of the APC/C also stabilizes FBL17 in the absence of replication stress (Pan
273 et al. 2023), it seems that this Ub E3 ligase plays a broader role in the post-translational control of
274 FBL17.

275 Among the ~700 Arabidopsis F-box proteins known to date, FBL17 is the closest functional
276 homolog to the mammalian Skp2 (Gagne et al. 2002). FBL17 shares with Skp2 many characteristics
277 (**Table 1**). Both are direct targets of E2F/DP for a periodic expression during G1/S, both are required
278 for entry in S-phase likely via their ability to degrade CKI proteins, both appear to be phosphorylated
279 by WEE1 and to be substrates of the APC/C, and finally, both are involved in the DDR. Altogether,
280 this makes FBL17 a fascinating protein for further studies of the plant cell cycle and also beyond.

281 Important questions to tackle in the future include the following. First, and slightly provocative,
282 are KRPs really direct targets of FBL17? Several observations support this conclusion (see above),
283 but to date, none of the *krp* mutations has been reported to suppress the sporophytic phenotype of
284 *FBL17* loss-of-function. This could be tested with higher-order *krp* mutant combinations. Also,
285 biochemical evidence to demonstrate the direct role of the SCF^{FBL17} in KRP turnover is missing. A
286 fully reconstituted ubiquitylation assay would be valuable. To understand how KRPs are
287 recognized by the Ub E3 ligase is also of great interest. It was shown that the stability of some
288 KRPs, such as KRP2, depends on phosphorylation by CDKs (Verkest et al. 2005a), but whether
289 FBL17 binds a phospho-degron and requires the Cks1 cofactor as Skp2 is currently unknown.
290 Obtaining structural data on the interaction of FBL17 with its substrate would significantly advance
291 the field. The ubiquitylation site on KRPs and the topology of Ub chains should also be addressed.

292 Second, as Skp2 is reported to target more than a dozen substrates (Frescas and Pagano
293 2008), we might ask how large is the substrate repertoire of FBL17? Addressing this question is
294 challenging. For instance, if FBL17 recruits its substrates through a phospho-degron, a simple
295 yeast-two-hybrid screening approach to identify new targets may fail. Searching for substrates by
296 either pulldown or proteomics approaches may also be challenging. Since *FBL17* expression is
297 mainly restricted to meristematic tissues, interacting substrates may be of low abundance and
298 difficult to detect. In addition, interactions between F-box proteins and their substrates have been
299 described as versatile, often with low affinity for the substrate (Pierce et al. 2009). Therefore, other
300 techniques such as Ub ligase trapping or proximity labeling should be considered (Iconomou and
301 Saunders 2016).

302 Finally, it will be necessary to explore the regulation of *FBL17* at both transcriptional and post-
303 translational levels. Given the feature of the G1/S transition as a critical cell cycle checkpoint where
304 multiple signaling pathways are converging, FBL17, by regulating the stability of a number of
305 important players during this transition phase, appears undeniably in a good position to act as a
306 key regulatory node.

307 Is autophagy a key process in the plant DNA damage response?

308 **(By Poyu Chen, Maren Heese, and Arp Schnittger)**

309 The DNA of plant cells, like the DNA of any other organism, is constantly damaged in various ways,
310 including DNA double-strand breaks (DSBs) and DNA cross-links. Upon the detection of damage, a
311 cell launches a specific response called the DNA damage response (DDR), which depends on the
312 type and level of the damage experienced as well as on the developmental context and the
313 physiological state of a cell (Chen et al. 2019, Szurman-Zubrzycka et al. 2023). If the DNA is mildly
314 damaged, the DDR usually triggers an arrest of cell proliferation (although DNA replication and cell
315 growth can sometimes continue; Adachi et al. 2011), and a DNA repair program is launched. If the
316 DNA is severely damaged and/or if very little damage is tolerated due to developmental
317 constraints, such as in stem cells (Fulcher and Sablowski 2009), terminal differentiation or death of
318 the damaged cell will be induced (Chen et al. 2019). These cellular responses rely on a specific
319 transcriptional response in which the NAC transcription factor SUPPRESSOR OF GAMMA
320 RADIATION1 (SOG1) plays a central role by inducing the expression of, for instance, genes
321 repressing cell division, such as the CYCLIN DEPENDENT KINASE inhibitors SIAMESE
322 RELATED PROTEIN 5 and 7 (SMR5, SMR7), and genes involved in the actual mending of DNA,
323 such as the recombinase RADIATION SENSITIVE 51 (RAD51) and CYCLINB1;1 (CYCB1;1),
324 which both are involved in homologous recombination (HR) repair (Yoshiyama et al. 2009, Ogita et
325 al. 2018, Bleuyard et al. 2005, Weimer et al. 2016, Yi et al. 2014).

326 However, targeted degradation of proteins also plays a pivotal, yet so far not well-studied role
327 in the DDR of plants. In general, the removal of specific proteins can be executed by two different
328 systems: 1) the proteasome, present in the nucleus and the cytoplasm, and 2) selective autophagy,
329 i.e., degradation via lytic compartments such as the vacuole in plants and the lysosome in animal
330 cells, executed in the cytoplasm (**Fig. 2**). Previously, proteasome-mediated protein degradation has
331 been implicated in the DDR of Arabidopsis; for example, the transcriptional repressor MYB3R3,
332 which is involved in cell cycle arrest after DNA damage, is blocked from proteasomal degradation
333 under DNA damaging conditions (Chen et al. 2017b). Conversely, the mitotic regulator CDKB2;1
334 becomes degraded in a proteasome-dependent manner upon DSB induction (Adachi et al. 2011).
335 In contrast, it was not known until recently whether autophagy, which in plants is subdivided into
336 the three forms of microautophagy, macroautophagy, and mega-autophagy (Marshall and Vierstra
337 2018), is involved in a plant's response to DNA damage.

338 Autophagy has emerged as an important regulatory mechanism of cellular homeostasis in
339 many, if not all, eukaryotes and for animals, there is evidence that different types of autophagy are
340 also involved in the DDR. However, the picture is still fragmented, and the identity of the specific
341 autophagy targets remains for the most part enigmatic (Juretschke and Beli 2021). Recently,
342 macroautophagy was identified to play a central role in plant DDR, i.e., during DNA cross-links
343 repair in Arabidopsis, where it was shown to be required for the selective removal of a repressor of
344 HR (Chen et al. 2023b). HR in plants is usually repressed during somatic growth and development
345 by the action of the RTR-complex, homologous to the BLOOM syndrome complex (BTR-complex)
346 in animals. The central components of this complex are a RECQ-type helicase and the
347 topoisomerase TOP3alpha attached to a scaffolding protein named RMI1 (Hartung et al. 2008). To

348 allow for elevated HR after DNA cross-link induction, RMI1 was now shown to be removed in a
349 macroautophagy-dependent manner (Chen et al. 2023b).

350 K63-linked polyubiquitylation (in contrast to K48 polyubiquitin chains that mark proteins for
351 proteasome-dependent degradation) is known, among other functions, to mark cargo for degradation
352 by autophagy (Nathan et al. 2013, Tan et al. 2008). KNOTEN1 (KNO1) of Arabidopsis, a nuclear
353 protein previously implicated in the DDR (Bouyer et al. 2018), was now found to be required for the
354 attachment of K63-linked polyubiquitin to RMI1, which subsequently leads to RMI1 degradation in the
355 cytoplasm in a lytic compartment-derived manner (Chen et al. 2023b) (**Fig. 2**). Interestingly, KNO1
356 itself is also a target of selective protein turnover and was found to be degraded under non-DNA
357 damaging conditions by the proteasome (Chen et al. 2023b). Thus, proteasomal and vacuolar
358 degradation systems appear to be tightly interconnected and collaborate during DDR.

359 Notably, the impact of macroautophagy on DDR likely goes far beyond the regulation of RMI1.
360 Macroautophagy relies on a group of autophagy-related (ATG) proteins that regulate the formation
361 of autophagosomes and promote their delivery to the vacuole (Su et al. 2020). Analysis of
362 Arabidopsis mutants of the central autophagy components *ATG2*, *ATG5*, and *ATG7* revealed that
363 all three mutants are not only sensitive to the DNA cross-link inducing agent cisplatin but also to
364 drugs that cause other types of DNA damage, i.e., hydroxyurea (HU), which interferes with DNA
365 replication and produces single-stranded DNA, as well as zeocin, which induces DNA double-
366 strand breaks (Chen et al. 2023b). Since mutants in *KNO1* are particularly sensitive to DNA cross-
367 linkers, but not to HU or DNA double-strand inducing drugs (Bouyer et al. 2018), it seems likely that
368 *KNO1* independent routes exist, that target proteins to macroautophagy after DNA damage and
369 that several proteins are removed by macroautophagy during the DDR. Autophagy has been found
370 to function not only in a pro-survival (Torii et al. 2016), but also in a cell death-promoting manner in
371 humans (Liu et al. 2018b) and plants (Kabbage et al. 2017, Üstün et al. 2017).Thus it seems
372 possible that autophagy in plants is also involved in a wider context of DDR, e.g., possibly by
373 controlling the cell death response.

374 How do proteolytic networks regulate mitochondrial function?

375 (By Abi S. Ghifari and Monika W. Murcha)

376 Mitochondria are central organelles, responsible for vital biochemical pathways, including aerobic
377 respiration and biosynthesis of amino acids, lipids, and redox cofactors, among many other
378 functions (Spinelli and Haigis 2018). These pathways rely on the homeostasis of thousands of
379 mitochondrial proteins that are maintained through continuous transcription and translation of
380 nuclear and mitochondrial genomes, protein import, assembly, and finally, degradation of damaged
381 and aggregated proteins (Vazquez-Calvo et al. 2020). Proteolysis plays a role at all stages of
382 mitochondrial biogenesis, from the onset, with regards to protein synthesis and assembly, to
383 protein turnover and degradation (van Wijk 2015, Ghifari and Murcha 2022). Evidence suggests
384 that there is a network of proteases with overlapping activities in organelles (van Wijk 2015, Majsec

385 et al. 2017). These proteolytic networks regulate protein function and abundance to maintain
386 protein homeostasis across various mitochondrial compartments and functions (**Fig. 3**).

387 What role do proteases play in protein import and maturation? Most proteins destined for
388 mitochondria are synthesized with cleavable N-terminal targeting peptides that initiate protein
389 translocation through the Translocase of the Outer Membrane (TOM) complex (**Fig. 3**) (Pfanner et
390 al. 2019). Upon import, proteins are matured by various peptidases, such as the mitochondrial
391 processing peptidase (MPP), octapeptidyl peptidase 1 (OCT1), and intermediate cleavage
392 peptidase 55 (ICP55) to cleave the N-terminal targeting peptides and any subsequent unstable
393 residues (Carrie et al. 2015, Huang et al. 2015, Gomes et al. 2017). A distinguishing feature of
394 plant mitochondria is that the a and b subunits of MPP are also integral components of the
395 cytochrome bc_1 complex (Complex III) of the respiratory oxidative phosphorylation (OXPHOS)
396 system (Glaser et al. 1994, Emmermann et al. 1993). The enzymatic activity of MPP/*bc1* is
397 independent of electron transfer (Eriksson et al. 1996) and recent structural studies of Complex III_2
398 have shown that the a and b subunits of MPP form a large cavity allowing for presequence binding
399 (Maldonado et al. 2021). The distinctive dual function of MPP in plants may be a mechanism of
400 regulating protein import with the requirement for substrates, particularly the subunits of the
401 electron transport chain. Proteases have also been implicated in maintaining the abundance of the
402 protein import machinery, which in turn regulates protein uptake rates (Lister et al. 2007, Wang et
403 al. 2012). For example, immunoprecipitation experiments have identified Tim17-2, the inner
404 membrane transporter channel protein as a substrate of Filamentous Temperature Sensitive-H 4
405 (FTSH4) in Arabidopsis (Opalinska et al. 2018).

406 The initial cleavage via MPP generates peptides with the potential to disrupt membrane
407 integrity and inhibit protein import (Zardeneta and Horowitz 1992, Hugosson et al. 1994).
408 Mitochondrial targeting peptides are further degraded in a multi-step peptide processing pathway
409 by numerous matrix-located proteases with overlapping specificity such as the presequence
410 peptidase (PREP) and organellar oligopeptidase (OOP) (Ståhl et al. 2005, Bhushan et al. 2005,
411 Kmiec et al. 2013). Single amino acids are recovered from short peptides by various
412 aminopeptidases (AP) (Kmiec et al. 2018b, Waditee-Sirisattha et al. 2011, Ghifari et al. 2020).
413 Plant mitochondria contain at least 15 individual peptidases involved in the process of removing
414 the targeting signal and processing it to single amino acids (Ghifari et al. 2019). Interestingly, most
415 of these peptidases are dually targeted to both mitochondria and chloroplasts, demonstrating a
416 common bacterial-derived peptidolytic network (Kmiec et al. 2018b), alongside distinct protein
417 import mechanisms. The activities of the dual-targeted intermediate peptidases PREP and OOP
418 are most strikingly observed in chloroplasts whereby functional losses of these peptidases led to
419 an accumulation of peptides of chloroplast origin (Kmiec et al. 2018a, Rowland et al. 2022).
420 However, the effect may be more subtle in mitochondria and has yet to be observed.
421 What proteases are involved in maintaining mitochondrial protein quality control (PQC)?
422 General mitochondrial proteolytic networks primarily composed of ATP-independent proteases
423 such as degradation of periplasmic protein (DEG) and rhomboid-like (RBL) (García-Lorenzo et al.

424 2006) and ATP-dependent such as members of the ATPase-associated with various cellular
425 activities (AAA+) family, which includes FTSH proteases, caseinolytic proteases (CLP), and LON
426 (long filamentous phenotype) proteases (Puchades et al. 2020, Heidorn-Czarna et al. 2022) (**Fig.**
427 **3**). Mitochondrial inner membrane proteins are maintained by the matrix-facing (m-AAA) FTSH3
428 and FTSH10 and the IMS-facing (i-AAA) FTSH4 and FTSH11 (Janska et al. 2010, Kolodziejczak et
429 al. 2007, 2018, Maziak et al. 2021, Heidorn-Czarna et al. 2018). Matrix-located AAA+ proteases,
430 such as CLPP2 and LON1 are active towards both soluble matrix and matrix-facing membrane-
431 bound proteins (Li et al. 2017, Petereit et al. 2020). OMA1 (overlapping activity with m-AAA
432 protease-1) primarily maintains the outer membrane (OM) and the intermembrane space (IMS)
433 proteins (Migdal et al. 2017, Gilkerson et al. 2021).

434 How are OXPHOS complexes turned over? OXPHOS complexes are large, multi-subunit,
435 dynamic complexes of the inner membrane capable of forming larger supercomplex structures
436 (Schlame 2021). Composed of both nuclear- and mitochondrial-encoded subunits, OXPHOS
437 complexes require intricate coordination, assembly, and regulation (Vercellino and Sazanov 2022,
438 Ghifari et al. 2023b). Furthermore, individual subunits are differentially susceptible to oxidative
439 damage exhibiting distinctive protein turnover rates (Li et al. 2017, Szczepanowska et al. 2020).
440 This suggests that submodules and domains are disassembled and degraded in a modular
441 fashion. A recent study demonstrated that the ATPase domain of FTSH3 promotes the
442 disassembly of the Complex I matrix arm domain (Ivanova et al. 2021) by directly interacting with a
443 specific Complex I subunit (Ghifari et al. 2023a). Structures of FTSH3 homologs revealed that this
444 domain can recognize and bind elongated peptides for degradation (Puchades et al. 2017, 2019).
445 When damaged or misfolded, proteins expose their N-terminal peptide, which serves as a
446 degradation signal that can be recognized by the ATPase domain of AAA+ proteases (Rampello
447 and Glynn 2017).

448 This function of FTSH3 has only so far been associated with Complex I, yet all OXPHOS
449 complexes are continually undergoing disassembly and turnover. The challenge lies ahead in
450 discovering the mechanisms of substrate recognition and disassembly. Furthermore, the
451 interconnectivity and how the protease functions are coordinated remains unknown. One of the
452 biggest challenges in using single loss-of-function mutants is that often these knockout plants
453 display mild phenotypic change and subtle biochemical changes, due to overlapping functions or
454 gene duplication (Kmiec et al. 2014, Petereit et al. 2020). Whilst a functional interconnection
455 between proteolytic and peptidolytic degradation has been well demonstrated in chloroplasts
456 (Rowland et al. 2022), the interconnectivity of mitochondrial proteolytic networks needs further
457 experimental confirmation. Identification of substrates, interactors, and proteins in proximity using
458 mass spectrometry-based methods can also provide a more comprehensive view. Trapping
459 approaches, whereby the catalytic function of protease is nullified to trap the protein substrate,
460 have revealed potential substrates and specific activities of various proteases (Heidorn-Czarna et
461 al. 2018, Liao and van Wijk 2019, Opalińska et al. 2017). Proximity-based techniques, such as
462 biotinylation and chemical crosslinking in yeast have also revealed that the prohibitin/m-AAA

protease complex is in proximity to both translation machinery and protein import complexes, demonstrating its importance in determining the fate of newly synthesized and newly imported proteins (Singh et al. 2020, Kohler et al. 2023). A combination of these techniques may provide a more comprehensive view and a better understanding of the modulation and interconnectivity of proteases in plant mitochondria.

N-terminal signals for degradation pathways

To destroy or not to destroy? What is the effect of N-terminal acetylation on protein half-life?

(By Daniel J. Gibbs)

Protein N-terminal (Nt-)acetylation (NTA) involves the transfer of acetyl moieties from acetyl-coenzyme A to the α-amino group of Nt-amino acid residues by enzymes called Nt-acetyltransferases (NATs) (Aksnes et al. 2019, Ree et al. 2018, Giglione and Meinnel 2021). This modification occurs on 60-80% of all proteins in eukaryotes and is assumed to be irreversible, since no Nt-deacetylases are known (Giglione and Meinnel 2021). Until recently NTA was thought to be exclusively and constitutively imprinted during mRNA translation by ribosome-tethered NATs and unlikely to play a significant regulatory role in protein function and signaling. However, recent studies in plants have shown that NTA can occur post-translationally within plastids, and that the activities of certain NATs are linked to abiotic, biotic, and cellular stress responses (Linster et al. 2015, Bienvenut et al. 2020, Huber et al. 2020, Huber et al. 2021). Dual NTA and internal Lysine-acetylation activities have also been reported for some acetyltransferases, broadening our knowledge of acetylation complexity and crosstalk (Bienvenut et al. 2020). Through neutralizing the positive charge of the α-amino group, NTA bestows new biochemical properties that can directly affect protein folding, avidity for protein interaction partners, sub-cellular targeting, and protein stability. Here I discuss current knowledge on the complex relationship between NTA and proteolysis via the UPS, with a particular focus on seemingly contradictory findings as well as key open questions in the field.

In the early 2010s, several studies in yeast and mammals demonstrated that NTA can directly target proteins for degradation, via an acetylation-dependent branch of the N-degron pathway (Ac/N-degron pathway) (Gibbs et al. 2014, Hwang et al. 2010, Shemorry et al. 2013, Park et al. 2015). Here, the N-termini of Nt-acetylated protein substrates are recognized and ubiquitylated by E3 ligases called Ac/N-recognins, which include DOA10/TEB4 and NOT4. A diverse but constrained set of Ac/N-degron pathway substrates was identified, and crucially it was shown that Ac/N-degrons are conditional, since they are usually shielded by protein folding or through intermolecular sequestration (Shemorry et al. 2013). Thus, it was proposed the Ac/N-degrons might contribute to protein quality control by allowing the recognition and rapid elimination of misfolded proteins or excess subunits of multi-protein complexes (**Fig. 4A**) (Nguyen et al. 2018). In contrast to this view, loss of NTA on yeast ribosomal proteins was shown to reduce overall ribosome thermostability, leading to an increase in subunit degradation via the UPS (Guzman et al. 2023).

501 This raises a key question as to whether NTA indirectly influences protein turnover through its
502 effects on protein-protein interaction affinities (**Fig. 4B**).

503 There are several reports of plant proteins that are directly destabilized due to NTA, implying
504 potential conservation of the Ac/N-degron pathway in this lineage. This includes a particular Nt-
505 variant of the immune receptor SNC1 that is acetylated by NATA (Xu et al. 2015b), as well as
506 OsHYPK in rice, itself a substrate, interaction partner, and potentiator of NATA activity (Gong et al.
507 2022). In neither case was the cognate E3 ligase identified. A recent study investigated potential
508 roles for putative Arabidopsis DOA10 homologs as Ac/N-recognins but found no clear connection
509 between DOA10 function and the turnover of Nt-acetylated proteins (Etherington et al. 2023). As
510 such, E3 ligases that recognize Nt-acetylated N-termini in plants await discovery. Interestingly
511 however, cross-species analyses did show kingdom-specific differences in the effect of NATs on
512 the stability of the same protein target through indirectly promoting protein turnover, perhaps
513 through influencing other E3 ligases or the proteasome.

514 Paradoxically, NTA has also been directly linked to increased stability of specific plant proteins,
515 including SIB1, a positive regulator of salicylic acid–induced cell death, and an alternative N-
516 terminal variant of SNC1 targeted by NATB (Xu et al. 2015b, Li et al. 2020b). This latter finding is
517 particularly intriguing as it highlights how NTA of two different Nt-variants of the same protein can
518 either increase or decrease protein half-life (Xu et al. 2015b, Gibbs 2015). Larger scale studies in
519 yeast, mammalian cells, and plants have also revealed that NATA-mediated NTA is broadly
520 associated with proteome stabilization (Gibbs et al. 2022, Mueller et al. 2021, Guzman et al. 2023).
521 Loss of NATA or HYPK function in Arabidopsis and rice led to increased turnover rates of NATA
522 substrate proteins, which was accompanied by a compensatory increase in translation rates of the
523 same proteins, mediated via the target of rapamycin (TOR) kinase (Linster et al. 2022, Miklankova
524 et al. 2022). This points to the presence of "non-Ac/N-degrons" that are exposed only when NATA
525 activity is downregulated, for example during drought (**Fig. 4C**) (Linster et al. 2015). As such it was
526 posited that regulation of protein NTA might be crucial for rapid proteome turnover to replenish
527 protein pools in response to certain stresses that impact NAT function.

528 How this might occur is yet to be determined, but the concept of "N-degron complementarity"
529 was previously proposed, whereby obstruction of one pathway can redirect a substrate to a
530 different pathway (e.g., a lack of NTA might instead allow targeting via the Arg/N-degron pathway)
531 (Park et al. 2015, Nguyen et al. 2018). Indeed, this was recently demonstrated for different NATB
532 and NATC substrates in mammals, where NTA was shown to prevent degradation by the Arg/N-
533 degron pathway E3-ligase UBR4 (Guedes et al. 2023, Varland et al. 2023). A study in yeast also
534 showed that NTA can stabilize proteins independent of their ubiquitylation, suggesting that
535 additional proteolytic pathways must be considered (van de Kooij et al. 2023). Moreover, different
536 mechanisms are probably at play in different eukaryotic kingdoms. For example, the IAP E3 ligases
537 shown to bind non-acetylated NATA protein substrates in mammalian cells are not found in plants
538 (Mueller et al. 2021).

539 Despite its prevalence, NTA remains a somewhat enigmatic modification without a single
540 defined effect on protein stability, although there is increasing evidence that the “default” effect is to
541 promote stability, while still being able to trigger degradation of a more restricted set of specific
542 proteins. Thus, NTA seems to influence protein half-lives in a substrate and context-specific
543 manner. Several key questions linked to the study of NTA and its effects on proteolysis remain: (1)
544 Does NTA influence protein half-life co-translationally, post-translationally, or both? (2) Are NTA-
545 mediated effects on protein stability direct or indirect, and can different NATs have contrasting
546 effects on substrate turnover? (3) Does NTA catalyzed by other NATs (in addition to NATA) also
547 trigger large-scale protein stabilization in plants, and is this linked to shielding against the Arg/N-
548 degron pathway? (4) Does the partial acetylation observed for some substrates act as a switch to
549 flexibly control protein half-lives? (5) Does NTA have a broader role to play in nascent proteome
550 remodeling in response to signals that affect NAT function? (6) What is the significance of post-
551 translational NTA in plastids, and does it contribute to protein degradation in these or other
552 organelles? By focusing on these questions, the stage is set to provide new insight and further
553 clarity into roles for this widespread protein modification in cellular proteostasis.

The Arg/N-degron pathways of protein turnover: Boutique or bulk?

(By Frederica L. Theodoulou and Hongtao Zhang)

The Arg/N-degron pathway was first defined in the context of arginylation, but now effectively refers to all non-acetylated N-degrons (Varshavsy 2019). Intriguingly, evidence to date indicates that in plants, Arg/N-degron pathways predominantly target short-lived regulatory proteins with unacetylated N-termini, whereas a much wider range of cellular/proteostatic functions has been reported for animals and yeast. Moreover, whilst a seminal study quantifying the half-lives of artificial reporter proteins in yeast established the concept of "stabilizing" and "destabilizing" Nt residues, it is now evident that all 20 proteogenic amino acids can potentially act as Nt degradation signals (N-degrons) in non-plant systems (Bachmair et al. 1986, Varshavsky 2019). This raises the question of how many undetected substrates and processes are regulated by the Arg/N-degron pathways in plants and how these contribute to plant physiology.

Do we know all the players?

Arg/N-degrons are revealed by proteolytic cleavage, or created by subsequent enzymatic modification to produce destabilizing N-termini that are recognized by Ub E3 ligases (known as N-recognins) and targeted for proteasomal degradation (**Fig. 5**). Plastids and mitochondria do not have an internal UPS but are proposed to house discrete N-degron pathways employing the Clp AAA+ protease system (Bouchnak and van Wijk 2019; see next section by van Wijk). The architecture of the nuclear-cytosolic Arg/N-degron pathway and destabilizing residue identities are broadly conserved between yeast, mammals, and plants but plants have a unique complement of N-recognins (Garzón et al. 2007, Graciet et al. 2010). Mammalian N-recognins have overlapping specificity for different classes of destabilizing residues (Type 1, basic; Type 2, aromatic, bulky) and act semi-redundantly (Tasaki et al. 2005). They share a Ub amino-end recognizing (UBR) box but also contain additional motifs involved in substrate recognition and different E3 ligase domains: RING in UBR1 and 2, HECT in UBR5, and a non-canonical hemi-RING E3 domain in UBR4 (Tasaki et al. 2005, Barnsby-Greer et al. 2024).

In contrast, the Arabidopsis homolog of UBR1/2, PROTEOLYSIS6 (PRT6) targets Type 1 N-termini but lacks the ClpS-like domain of UBR1/2 that acts as a recognition domain for Type 2 residues (Garzón et al. 2007). This function has been replaced in the green plant lineage by PROTEOLYSIS1 (PRT1), a unique protein with two RING fingers and a ZZ domain (Potuschak et al. 1998, Till et al. 2019). Although this suggests that separating turnover of Type 1 and 2 substrates could have adaptive value in plants, BIG, an Arabidopsis homolog of UBR4, has recently been implicated in the degradation of substrates with both basic and aromatic N-termini (Zhang et al. 2024a). Plant genomes lack a UBR5 homolog, and it is clear from protein stability reporter studies that further N-recognins remain to be identified, including the elusive Nt-Leu/Ile recognition component(s) (Garzón et al. 2007, Graciet et al. 2010). Whilst genetic evidence strongly supports a role for PRT6 and BIG as N-recognins, biochemical characterization of these

591 very large proteins is challenging and E3 activity has only been formally demonstrated for PRT1
592 (Stary et al. 2003, Mot et al. 2018).

593 An important related question is how N-recognins partner with different E2 enzymes and whether
594 they assemble different Ub linkages, potentially with different cellular outcomes (Orosa-Puente and
595 Spoel 2022, Brillada and Trujillo 2022). Here, reconstitution of the pathway in yeast has provided
596 valuable first insights (Kozlic et al. 2022) and the molecular basis of substrate recognition and
597 ubiquitylation will also be informed by advances in structure determination and predictions (Pan et
598 al. 2021a, Sherpa et al. 2022, Jeong et al. 2023, Barnsby-Greer et al. 2024). Structural studies
599 may also shed light on Arg/N-degron pathway-proteasome complexes recently identified by
600 biochemical approaches (Oh et al. 2020b, Zhang et al. 2024a).

601 ***Do we know all the substrates?***

602 Thus far, only a handful of Arg/N-degron pathway substrates has been identified in plants
603 (Holdsworth et al. 2020). This is in stark contrast to animals and yeast, where the Arg/N-degron
604 pathways participate in cytosolic protein quality control, including degradation of misfolded proteins,
605 mistranslocated proteins, and retrotranslocated organellar proteins, as well as the control of protein
606 subunit stoichiometry (Varshavsky 2019). At present, there is little evidence for this in plants.

607 The majority of substrates confirmed in planta are Met-Cys initiating proteins, comprising Group
608 VII ETHLENE RESPONSE FACTOR transcription factors (ERFVIIs), the polycomb repressive
609 complex 2 subunit, VERNALIZATION2 (VRN2), and the LITTLE ZIPPER 2 (ZPR2) transcription
610 factor (Gibbs et al. 2011, 2018, Licausi et al. 2011, Weits et al. 2019). Following co-translational
611 cleavage of Met1 by aminopeptidases, Cys2 may be converted to Cys-sulfinic acid by PLANT
612 CYSTEINE OXIDASE (PCO) enzymes (Weits et al. 2014, White et al. 2017), rendering the protein
613 susceptible to arginylation and PRT6-mediated proteasomal degradation (**Fig. 5**). Recent evidence
614 suggests the potential presence of further enzymes contributing to complete Nt-Cys oxidation
615 (Zubryzki et al. 2023). Thus, oxygen-dependent turnover of regulatory proteins by the Arg/N-
616 degron pathways plays a central role in environmental and developmental hypoxia sensing
617 (Holdsworth et al. 2020). Characterization of *prt6* and *ate* mutant plants has revealed further
618 functions of the Arg/N-degron pathways in a/biotic stress responses and development;
619 interestingly, the majority of these are attributable to the regulation of ERVIIs (Holdsworth et al.
620 2020).

621 Nevertheless, other substrates must exist: PRT6-dependent, ERFVII-independent control of
622 hypoxia-responsive genes has been reported (Zubryzki et al. 2023), and conservation of arginyl-
623 tRNA-transferase and Nt amidase specificity in plants implies the existence of PRT6 substrates
624 that are not Met-Cys proteins (**Fig. 5**) (Graciet et al. 2010, Vicente et al. 2019). However, these
625 cannot easily be predicted. An N-degron comprises not only the Nt residue but also appropriately
626 positioned Lys residues for Ub conjugation, which must both be sufficiently accessible to N-
627 recognins (Varshavsky 2019). Accordingly, not all Met-Cys proteins are N-degron pathway
628 substrates (Gibbs et al. 2011, Bäumler et al. 2019, Kozlic et al. 2022), nor are all proteins with

629 other Nt destabilizing residues revealed through endopeptidase cleavage (e.g., RIN4; Goslin et al.
630 2019, Kozlic et al. 2022).

631 Mutants impaired in Arg/N-degron function grow like wild-type plants under non-stressed
632 conditions (except for *big* alleles which are pleiotropic), implying that phenotypes- and substrates-
633 may be cryptic. Proteases act as gatekeepers of the Arg/N-degron pathway and offer a largely
634 unexplored route to substrate identification through protein N-terminome ("degradome") analysis
635 (Perrar et al. 2018, Bogaert and Gevaert 2020). Plant genomes encode hundreds of proteases,
636 including metacaspases which are predicted to reveal potential destabilizing residues (Rawlings et
637 al. 2018). Given the conditional nature of proteolytic cleavage, proteomic analyses and other
638 strategies to identify substrates may need to compare N-degron pathway mutant alleles under
639 different environmental conditions to reveal cryptic degrons and also incorporate subcellular
640 fractionation to access low abundance targets. Cell and tissue specificity is also a key
641 consideration for future studies.

642 ***Who regulates the regulators?***

643 The Arg/N-degron pathways do not operate in isolation and a major outstanding question is how they
644 intersect with other signaling pathways. This may be complex, for example, the activity of master
645 regulator substrates such as ERFVIIs is subject to modulation by transcription factors, kinases,
646 membrane association, degron masking, and additional E3 ligases (Licausi et al. 2011, Papdi et al.
647 2015, Lin et al. 2019, Liu et al. 2021, Fan et al. 2023). Furthermore, it is not yet fully understood to
648 what extent different N-terminal modifications compete to influence protein fate in plants (Kats et al.
649 2018, Linster et al. 2022) and it remains to be explored whether plant Arg/N-degron pathways
650 intersect with autophagic pathways as in mammals (Heo et al. 2023). Thus, whilst the importance of
651 plant Arg/N-degron pathways in controlling the lifetime of regulatory proteins is well established, to
652 what extent they contribute to more widely to protein turnover and quality control remains an open
653 question.

654 What are the degrons and molecular players in the chloroplast N-degron pathway? 655 (By Klaas J. Van Wijk)

656 N-degrons are major determinants of protein stability in the cytosol of bacteria and eukaryotes
657 (Dissmeyer et al. 2018, Varshavsky 2019, Holdsworth et al. 2020, Weits et al. 2021), and likely
658 also chloroplasts and non-photosynthetic plastids (Bouchnak and van Wijk, 2019, 2021).
659 Systematic mass spectrometry (MS) analysis of the N-termini of stromal-exposed proteins using N-
660 terminal tagging (with a technique named TAILS) (Rowland et al. 2015) showed enrichment of
661 canonical stabilizing residues A, V, T, S (often in N-α-acetylated form) and avoidance of charged
662 (D, E, R, K) and large hydrophobic residues (*e.g.* W, F, Y, L) that serve as primary or secondary
663 degrons in bacteria and eukaryotic cytosol (Rowland et al. 2015). We therefore postulated that an
664 N-degron pathway exists in chloroplasts and other plastid types (Rowland et al. 2015, Bouchnak
665 and van Wijk 2019) (**Fig. 6**).

666 N-degron pathways in eukaryotes, including plants, typically involve polyubiquitylation and the
667 proteasome (Perrar et al. 2019, Holdsworth et al. 2020, Weits et al. 2021). In contrast, the
668 prokaryotic N-degron pathway depends on the adaptor ClpS (also named N-recognin) for the
669 recognition and delivery of N-degron-bearing substrates to Clp chaperone-protease systems. The
670 first step involves N-degron recognition of hydrophobic residues through a hydrophobic pocket in
671 ClpS followed by docking of the ClpS-substrate complex on the N-domain of the ClpA or ClpC
672 AAA+ chaperone (Kim et al. 2022). The ClpS-substrate complex is then "pulled" into the ClpA/C
673 pore in an ATP-dependent fashion (requiring ATP hydrolysis), and the resulting distortion of the
674 ClpS structure allows release of the substrate inside the ClpA/C pore. ClpS is subsequently
675 released from ClpA/C and unfolding and degradation of the substrate by the Clp protease ring is
676 completed (Kim et al. 2022).

677 Chloroplast ClpS1, a structural and functional homolog of bacterial ClpS, directly interacts with
678 the ClpC chaperones (Nishimura et al. 2013, Nishimura and van Wijk 2015). ClpS1 affinity
679 experiments in Arabidopsis identified several interacting chloroplast proteins, including glutamyl
680 tRNA reductase 1 (GluTR) a key enzyme in tetrapyrrole biosynthesis (heme and chlorophyll)
681 (Nishimura et al. 2013). Follow-up experiments showed that dark-induced degradation of GluTR
682 indeed requires the Clp system (Apitz et al. 2016, Richter et al. 2019). The interaction between
683 ClpS1 and these candidate substrates was dependent on the conserved substrate binding residues
684 in ClpS1 (Nishimura et al. 2013). However, N-degrons in these substrates have not been identified
685 and no obvious canonical N-degrons were found. In vitro ClpS1 affinity assays with selected
686 recombinant N-degron reporters demonstrated that ClpS1 has a restricted N-degron specificity
687 (Montandon et al. 2019a). Furthermore, a high-resolution structure (2 Å) for Arabidopsis ClpS1
688 showed that the N-degron binding pocket of ClpS1 is slightly enlarged compared to that of
689 *Escherichia coli* ClpS (Kim et al. 2021). In addition, amino acid replacement from Val (in *E. coli*) to
690 Ala in ClpS1 caused a reduction in hydrophobic interactions with Leu N-degrons (Kim et al. 2021).
691 Peptide array experiments with recombinant ClpS1 showed that N-terminal acetylation prevented
692 binding of such N-termini to ClpS1 (Aguilar Lucero et al. 2021). Collectively, these in vitro and in
693 vivo data suggest a unique N-degron pathway in chloroplasts. Recent studies show that bacterial
694 ClpS can also recognize non-canonical N-degrons including degrons a few residues downstream
695 of the N-terminus (Gao et al. 2019a, Jin et al. 2021); hence this scenario should also be
696 considered for chloroplasts.

697 ClpF was identified as an interactor of ClpS1 and it was shown that ClpF and ClpS1 mutually
698 stimulate their association with ClpC in vivo (Nishimura et al. 2015). Identified interactions between
699 ClpF, ClpS1, and GluTR suggested a ternary complex, and a testable model was proposed in which
700 ClpS1 and ClpF form a binary adaptor for selective substrate recognition of GluTR (and perhaps
701 other proteins) and delivery to ClpC (Nishimura et al. 2015). Whereas ClpF is a direct interactor to
702 ClpS1 as well as ClpC1, the mechanistic role of ClpF in the N-degron pathway is not understood.

703 To identify additional Clp substrates, an in vivo ClpC1 substrate trap with a C-terminal STREPII
704 affinity tag was expressed in Arabidopsis. This ClpC1 trap has mutated critical glutamate residues

705 (E374A and E718A) in the two Walker B domains of ClpC1 required for ATP hydrolysis (ClpC1-
706 TRAP) (Montandon et al. 2019b, Rei Liao et al. 2022). Based on homology to non-plant ClpB/C
707 chaperones, it is predicted that interacting substrates are not released, i.e. they are trapped (Rei
708 Liao and van Wijk 2019). Affinity purification of the ClpC1-TRAP resulted in more than 50 highly
709 enriched proteins compared to affinity-purified wild-type ClpC1 (Montandon et al. 2019b, Rei Liao
710 et al. 2022). These included >20 small proteins with unknown function/domains and several
711 metabolic enzymes some of which were also identified as ClpS1 interacting proteins or over-
712 accumulated in *clp* mutants (Nishimura et al. 2013). These enriched proteins likely represent Clp
713 protease substrates, some possibly with N-degrons, and/or new adaptors.

714 Despite the significant support for a unique N-degron pathway in chloroplasts that involves
715 ClpS1, and perhaps also ClpF, in vivo demonstrations for ClpS1-dependent substrate selection
716 and delivery to the Clp chaperone-protease system are lacking. For instance, whereas in vitro
717 peptide binding assays for Arabidopsis ClpS1 showed a clear affinity for hydrophobic N-terminal
718 amino acids (in particular F, W, Y), the ClpS1 protein interactome data with stromal proteins (e.g.
719 for GluTR) failed to suggest a canonical N-degron. Furthermore, a direct positive correlation
720 between chloroplast protein N-termini and ClpS1-dependent degradation has not been shown. Key
721 questions about chloroplast N-degron pathways that need to be resolved include: i) does
722 generation of chloroplast N-degrons involve post-translational modifications (e.g. acetylation,
723 phosphorylation, oxidation or amino acid transfer), and are there specific enzymes involved in
724 creating these modifications?, ii) are N-degrons for ClpS1 generated by N-terminally truncation of
725 upstream proteases as has been suggested in *E. coli* (Humbard et al. 2013)? iii) are N-degrons
726 confined to the very N-terminal residue of the substrates or are there more downstream signals
727 (non-canonical N-degrons)?, iv) is ClpS1 aided by co-adaptors such as ClpF?, iv) is there
728 competition between ClpS1 and other adaptors/anti-adaptors – to influence substrate selection and
729 regulation of rates of proteolysis? Novel and innovative in vivo chloroplast tools and approaches
730 are needed to determine the molecular details of ClpS1-dependent mechanisms of N-degron
731 substrate selection and delivery to the Clp chaperone-protease system in chloroplasts.

732 Roles of proteolysis in developmental and metabolic signaling

733 How do plants use ubiquitin-mediated proteolysis to regulate photomorphogenesis?

734 (By Ning Wei and Giovanna Serino)

735 Light signals perceived by photoreceptors are transduced to guide plant growth and development
736 in a process known as photomorphogenesis. Dark-grown etiolated seedlings undergo dramatic
737 changes after exposure to light, including inhibition of hypocotyl elongation, unfolding of the apical
738 hook, expansion of cotyledons, and maturation of chloroplasts (Kendrick and Kronenberg 1994).
739 Genetic screens for Arabidopsis mutants exhibiting longer hypocotyls in the light led to the
740 identification of photoreceptor mutants (such as *long hypocotyl 3/phytochrome B* [*hy3*/*phyB*] and
741 *long hypocotyl 4*/*cryptochrome 1* [*hy4/cry1*]*,* and *hy5, a* mutant of a positive regulator of

photomorphogenesis (Koornneef et al. 1980). Genetic screens for mutants with short hypocotyls and open cotyledons in the dark identified the *constitutively photomorphogenic/de-etiolated/fusca* (*cop/det/fus*) mutants (Chory et al. 1989, Deng et al. 1991, Wei and Deng 1992). As it turned out, all of the corresponding gene products function via the UPS, underscoring its importance in photomorphogenesis, as they encode components of E3 Ub ligase complexes CRL4$^{COP1\text{-}SPA}$ (Ponnu and Hoecker 2021), CRL4^{C3D} (C3D: COP10-DDB1-DET1-DDA1), and of a CRL regulator, the COP9 Signalosome (CSN).The CSN complex regulates all CRLs by de-neddylation, i.e. removing the Nedd8/RUB1 modification of the cullin subunit (Schwechheimer et al. 2001; Qin et al., 2020).. Mounting evidence shows that CRLs play key roles in photomorphogenesis, as they regulate the stability of many components of the light signaling, from photoreceptors to transcription factors (**Fig. 7**).

Regulating photoreceptor stability

Photoactivated phyA (Seo et al. 2004, Debrieux et al. 2013, Saijo et al. 2008), phyB (Jang et al. 2010, Lu et al. 2015, Sheerin et al. 2015), and both CRYs (Chen et al. 2021c, Miao et al. 2022) are ubiquitylation targets COP1-SPA. In addition, under strong red light, phyB is recruited to the CUL3-based ligases CRL3^{LRBs} through LIGHT-RESPONSE BRIC-A-BRAC/TRAMTRACK/BROAD 1 AND -2 (LRB1, LRB2) to be ubiquitylated (Christians et al. 2012, Ni et al. 2014). In the same fashion, CRL3^{LRBs} also mediate the degradation of cryptochrome 1 (CRY1) and CRY2 under high blue light or low temperature (Miao et al. 2022, Chen et al. 2021c, Ma et al. 2021). By lowering the level of phytochromes and cryptochromes, CRL3^{LRBs} serve to prevent over-stimulation and maintain light-signaling homeostasis. In addition, CRL3^{NPH3} targets protein degradation of the phototropin Phot1, a blue light-sensing photoreceptor mediating phototropic responses (Roberts et al. 2011) **(Fig. 7)**.

Regulating PIF stability

The stability of PIFs, which play essential roles in etiolation (skotomorphogenic development in darkness), as well as in shade avoidance and temperature responses under light conditions, is tightly controlled by the UPS. Light exposure results in rapid degradation of PIF proteins to induce de-etiolation. In this process, PIF3 is phosphorylated in response to photoactivation of phyB and then ubiquitylated by CRL1$^{EBF1/2}$ for subsequent degradation (Dong et al. 2017). PIF3, together with phyB, is also an ubiquitylation target of CRL3^{LRBs} (Ni et al. 2014); however, this co-degradation occurs specifically under higher light intensity (Dong et al. 2017). Thus, the two different Ub ligases play opposite roles in phyB signaling in different light environments: CRL3^{LRBs} attenuate light signaling under high light irradiation, while CRL1$^{EBF1/2}$ promotes photomorphogenesis, especially during de-etiolation.

PIF4 plays an important role in plant responses to shade, elevated temperature, and diurnal cycle. CRL3^{BOP2} has been shown to mediate PIF4 ubiquitylation and degradation, and indeed the *bop1 bop2* double mutant is hypersensitive to high temperature-mediated hypocotyl elongation (Zhang et al. 2017). In addition, PIF4 is phosphorylated by the BR signaling kinase BRASSINOSTEROID-INSENSITIVE 2 (BIN2), resulting in its degradation during the diurnal cycle

780 (Bernardo-García et al. 2014). It is probable that PIF4 is also modulated by other E3 ligases, yet to
781 be identified, that are sensitive to PIF4 phosphorylation. Last but not least, light-induced
782 degradation of PIF1 (Zhu et al. 2015), PIF5 (Pham et al. 2018), and PIF8 (Oh et al. 2020) have
783 been shown to involve the CRL4$^{COP1\text{-}SPA}$ complex, arguing for a dual role of COP1 in light signaling.

784 ***Open questions: How does COP1 achieve multifaceted roles within the complexity of light***
785 ***signaling?***

786 There are several open questions as to how UPS regulates light signaling. For example, specific
787 E3s regulating PIF stability in a time-, space- and signal-dependent manner are still to be
788 identified. Here we focus on mechanisms centered around COP1 and its dual role in light signaling.
789 The COP1-SPA complex acts as a central photomorphogenic suppressor by targeting myriad
790 positive regulators of light signaling, such as HY5, HFR, BBX4, photoreceptors, and many more, in
791 the dark or dim light conditions (Ponnu and Hoecker 2021) (**Fig. 7**). However, some of these
792 ubiquitylation targets are also stabilized by COP1 under different conditions. For example, while
793 COP1 targets HY5 degradation in the dark, it stabilizes it during UV-B-mediated
794 photomorphogenesis (Oravecz et al. 2006). While COP1 (as well as DET1) stabilizes PIFs in
795 darkness to ensure etiolated development (Bauer et al. 2004; Dong et al. 2014; Gangappa and
796 Kumar 2017; Ling et al. 2017), it also facilitates de-etiolation through CRL4$^{COP1\text{-}SPA}$-mediated light-
797 induced degradation of several PIFs (see above). Indeed, *cop1* seedlings not only have reduced
798 PIFs levels in darkness, but they also show defects in light-induced PIFs degradation (Pham et al.
799 2018, Zhu et al. 2015).

800 Studies from the last 10 years have also revealed that light signals inactivate COP1 by altering
801 the composition of COP1-associated complexes. UV-B irradiation causes dissociation of COP1-SPA
802 from the CRL4 core complex (Huang et al. 2013) and the subsequent formation of a COP1-UVR8
803 complex (Rizzini et al. 2011; Wang et al. 2022). The binding of monomeric UVR8 to the VP substrate-
804 recognition interface of COP1 dislodges COP1 substrates such as HY5, allowing them to accumulate
805 and thus stimulating downstream light responses (Lau et al. 2019, Huang et al., 2014; Wang et al.
806 2022). Likewise, photoactivated phytochromes and cryptochromes promote the physical separation
807 of COP1 and SPA proteins (Lu et al. 2015, Sheerin et al. 2015; Lian et al. 2011, Zuo et al. 2011;
808 Ponnu et al. 2019). Thus, disassembly of the CRL4$^{COP1\text{-}SPA}$ complex and blocking of the COP1
809 substrate-binding sites seem to be a general strategy in the light-dependent switch of COP1 activity.
810 Similar to those photoreceptor-directed actions, CSU2 suppresses COP1 activity also by binding to
811 the COP1 coiled-coil domain, which interferes with COP1 dimerization and the assembly of COP1-
812 SPA complexes (Xu et al. 2015).

813 However, how COP1 and DET1 stabilize PIFs in the dark remains an outstanding open question
814 (Bauer et al. 2004; Dong et al. 2014; Gangappa and Kumar 2017; Ling et al. 2017). In the absence
815 of COP1 or DET1, PIFs cannot accumulate. Since COP1 has been shown to target EBF1/2 to
816 degradation (Shi et al. 2016), the F-box proteins that mediate ubiquitylation of PIF3 and EIN3 via
817 CRL1$^{EBF1/2}$ ligase, it is possible that *cop1* mutant may accumulate EBF1/2, which would lead to

PIF3 destabilization. Additional mechanisms likely exist, and more rigorous investigations are needed to elucidate how COP1 and DET1 stabilize PIFs.

On the other hand, while it is clear that COP1-SPA serves as a substrate-recognition component in CRL4 $^{COP1\text{-}SPA}$, it remains obscure whether it can function as a stand-alone E3 ligase through the COP1 RING domain. COP1 function also requires CSN and DET1 (Qin et al., 2020; Cañibano et al. 2021), but the physical and functional interactions between COP1- and DET1-associated complexes require further clarification. The CSN complex has pleiotropic functions beyond light signaling, as it regulates most CRL ligases. In light signaling, CSN mediated de-neddylation of CUL1 is necessary for loading the PIF3-EBF1 complex onto the CRL1 ligase, thus assembling the CRL1^{EBF1} holocomplex during light-induced PIF3 degradation (Dong et al. 2024) (**Fig. 7**). Dissecting the functions and dynamic interactions of COP1-, DET1- and CSN-associated complexes remain highly challenging in the coming years.

How does proteolysis of core signaling components occur in different subcellular locations to modulate the ABA pathway?

(By Pedro L. Rodriguez)

ABA is perceived by a family of ABA receptors known as PYRABACTIN RESISTANCE 1 (PYR1)/PYR1-like (PYL)/regulatory components of ABA receptors (RCAR), which leads to inhibition of clade A protein phosphatases type-2C (PP2Cs) and subsequent relief of inhibition of subfamily III Snf1-related protein kinases (SnRK2s) (Cutler et al. 2010). Additionally, B2/B3-type RAF-like MAP3Ks are required to phosphorylate and activate the above SnRK2s (Lin et al. 2021). Proteolytic targeting mediated by E3 Ub ligases is a central component of all phytohormone signaling pathways, including ABA (Blazquez et al. 2020). Pioneering work on E3 ligases that regulate protein levels of transcription factors associated with ABA signaling, such as ABSCISIC ACID INSENSITIVE3 (ABI3) and ABI5, established a link between the UPS and modulation of ABA signaling (reviewed in Stone 2019).

Further work served to identify E3 ligases that regulate protein levels of other core components of ABA signaling, i.e. ABA receptors, PP2Cs, and SnRK2s (reviewed in Ali et al. 2020, Coego et al. 2021). ABA signaling in Arabidopsis involves 14 ABA receptors and 9 PP2Cs, as well as 3 SnRK2s. This is mirrored in the high number of E3 ligases that target these proteins (Ali et al. 2020, Coego et al. 2021). Because these E3 ligases are located in different cell compartments, the connection between plant cell biology and the regulation of ABA signaling is an emerging question for research (**Fig. 8**). Processing at the plasma membrane (PM) leads to cargo degradation into the lytic vacuole, via the endocytic pathway and autophagy (Saeed et al. 2023). Moreover, PM signaling nanodomains might be physiologically connected with the endocytic pathway and E3 ligases targeting ABA signaling components (Yu and Xie 2017, Chen et al. 2023c). The RBR-type E3 ligase RSL1 targets ABA receptors in the PM and promotes their endosome-mediated vacuolar degradation (Bueso et al. 2014). Given that ABA signaling at the PM is critical for the regulation of ion and water transporters,

E3 ligases anchored in the PM through transmembrane domains, myristoylation, or as peripheral proteins might contribute to K63Ub-mediated targeting of core signaling components to the endovacuolar pathway (Bueso et al. 2014, Belda-Palazon et al. 2019, Pan et al. 2020). K63-linked Ub chains not only act as a signal for endocytosis but also might contribute to the autophagic clearance of cargo proteins that act in ABA signaling (Sirko et al. 2021; Saeed et al. 2023).

Proteolytic targeting of all the players in ABA signaling, including ABA receptors, PP2Cs, SnRK2s, and TFs in the nucleus has been reported, affecting ABA transcriptional regulation and likely long-term ABA-induced changes in chromatin arrangement. Multimeric CRL4 E3 ligases regulate ABA receptor and OST1 protein levels in the nucleus through different substrate adaptor modules, involving DDA1 and HOS15, respectively (Irigoyen et al. 2014, Ali et al. 2019). On the other hand, the CRL3^{BPM} or the RING-type COP1 E3 ligases regulate nuclear PP2C protein levels (COP1 also in the cytosol) and affect stomatal function (Julian et al. 2019, Chen et al. 2021a). ABA induces chromatin remodeling in many cell types, affecting, for example, the root, the guard cell, and the mesophyll cell epigenome (Seller and Schroeder 2023). SnRK2s and PP2Cs orchestrate a phosphorylation-based switch to control the SWI/SNF chromatin-remodeling ATPase BRAHMA activity, which might be sensitive to developmental and environmental signals that regulate their protein levels (Peirats-Llobet et al. 2016). A possible memory effect of proteolysis on chromatin remodeling in response to abiotic stress deserves further investigation.

Other E3 ligases that target PP2Cs are located near or associated with the PM, such as PUB12/13 and LOG2/AIRP3 specifically targeting ABI1, or RGLG1/5 for PP2CA (Kong et al. 2015, Wu et al. 2016, Pan et al. 2020). Only in the case of RGLG1 has the subcellular localization of the PP2CA-RLG1 interaction been investigated, and interestingly, it was found that ABA modifies the PM localization of RGLG1 and promotes nuclear interaction with PP2CA (Belda-Palazon et al. 2019). ABA enhances the interaction of the E3 ligase and its target, and elucidation of this ABA-dependent translocation represents an area for further research. In the case of PUB12/13, the ubiquitylation of ABI1 in vitro requires exogenous ABA and the presence of ABA receptors. This suggests that some E3 ligases can recognize the PP2C-Receptor complexes, whose formation requires ABA for the dissociation of dimeric receptors and the assembly of highly stable forms (both for dimeric and monomeric receptors). This model also applies to RGLG1, which forms nuclear complexes with PP2CA and monomeric receptors, such as RGLG1-PP2CA-PYL8 (Belda-Palazon et al. 2019).

However, in another example, ABA protects the PYL8 ABA receptor from degradation (Irigoyen et al. 2014). Thus, when ABA levels increase, the CRL4^{DDA1} complex cannot promote the degradation of PYL8, establishing a positive feedback loop for PYL8-dependent signaling. It is not known whether the assembly of the CRL4^{DDA1} complex is impaired by ABA or if PYL8-ABA-PP2C complexes are resistant to degradation by CRL4^{DDA1}. Finally, the activation of SnRK2 degradation by ABA is postulated to be a negative feedback loop when SnRK2s are phosphorylated by B2/B3-type RAF kinases (Lin et al. 2021), as well as the posttranslational modification of ABA receptors can accelerate receptor degradation (Castillo et al. 2015, Yu et al. 2019). This suggests that some

894 E3 ligases might be sensitive to post-translational modifications of their targets, but the precise
895 mechanism is unknown.

896 In summary, the interaction of different E3 ligases with core components of ABA signaling has
897 been reported in the PM, cytosol, and nucleus, and additionally, the endosomal trafficking pathway
898 plays a key role in the turnover of ABA receptors that have been ubiquitylated at the PM (Belda-
899 Palazon et al. 2016, Yu et al. 2016, Garcia-Leon et al. 2019). ABA plays signaling roles, or its
900 levels are increased, in other subcellular compartments in response to abiotic stress, such as
901 mitochondria or endoplasmic reticulum (Postiglione and Muday 2013, Han et al. 2020). This
902 suggests that ABA perception and regulation of the half-life of certain core signaling components
903 might occur in particular cell regions, and regulation of the local concentration of the core signaling
904 network might be achieved by yet-to-be-discovered E3 ligases. The degradation of key repressors
905 of the ABA pathway (i.e., PP2Cs) is not the only mechanism to activate signaling because of the
906 alternative biochemical inhibition of their activity by ABA receptors (Cutler et al. 2010). This poses
907 unique questions to fully understand ABA signaling open for further research. For example, how is
908 biochemical (reversible) inhibition of PP2Cs intertwined with their proteolytic degradation, either
909 when they are free or in ternary complexes with ABA and ABA receptors? And from a global
910 perspective, is proteolytic degradation involved in signaling, desensitization, or resetting of the ABA
911 pathway?

912 The complexity of the strigolactone signaling pathway: How does the D14 receptor 913 function as both receptor and enzyme, linking hormone perception to protein 914 degradation?

915 **(By Angelica M. Guercio, Malathy Palayam, and Nitzan Shabek)**

916 Strigolactones (SLs), initially identified as root exudates from cotton (*Gossypium hirsutum*) (Cook
917 et al. 1966), were first described to have a role in hormone signaling in the control of shoot
918 branching (Gomez-Roldan et al. 2008, Umehara et al. 2008). Over the years, SLs have been
919 further characterized, and the cohort of diverse processes controlled by SLs is still expanding. SLs
920 can impact plant-environment interactions such as initiating symbiosis with mycorrhizal fungi and
921 stimulating the germination of parasitic plants (Akiyama et al. 2005, Akiyama et al. 2010,
922 Bouwmeester et al. 2003, Gutjahr et al. 2015, Yoneyama et al. 2010). Endogenously, SLs regulate
923 various aspects of plant growth and development, including shoot branching, leaf growth, leaf
924 senescence, secondary stem thickening, the formation of adventitious roots, lateral roots, and root
925 hairs (Bennett and Leyser 2014, Brewer, Koltai and Beveridge 2013, Ruyter-Spira et al. 2013,
926 Smith and Li 2014). Research on the extensive crosstalk between SL signaling and other
927 phytohormones continues to unveil a comprehensive network of cross-hormone regulation and
928 diverse effects in plant physiology.

929 *The crossroads of plant hormones and the Ub system*

930 Similar to other hormone signaling pathways in plants, SL signaling relies on regulated turnover
931 through the UPS (Jiang et al. 2013, Tal et al. 2020, Zhou et al. 2013a). Before the identification of
932 the SL receptor, the F-box protein MORE AXILLARY GROWTH 2 (MAX2) or DWARF3 (D3) in rice,
933 a component of the SCF (Skp1/Ask1- Cullin1-F-box) type E3 Ub ligase, was recognized as a key
934 player in SL-related pathways (Stirnberg et al. 2007). *max2* mutant plants exhibited phenotypes
935 similar to those with mutations in SL biosynthesis pathways. However, unlike other SL mutants,
936 *max2* phenotypes could not be rescued with SL treatment, indicating the involvement of MAX2 in
937 SL signaling. Later, the SL receptor was discovered through mutant analysis (Arite et al. 2009),
938 and similar to GA signaling, the receptor is an α/β hydrolase commonly referred to as D14
939 (DWARF14). It was hypothesized that D14, bound to SL, initiates complex formation with the
940 $SCF^{MAX2/D3}$ which subsequently recruits and orchestrates the polyubiquitylation and proteasomal
941 degradation of the target proteins such as D53 (DWARF53 in rice) or SMXL6/7/8 (SUPPRESSOR
942 OF MAX2 LIKE 6, 7, and 8) (Bennett and Leyser 2014, Jiang et al. 2013, Zhou et al. 2013a, Wang
943 et al. 2015). D53/SMXLs have a weak homology with class I Clp ATPase family proteins and have
944 been shown to regulate SL response genes via their EAR motifs, but their precise functions and
945 transcriptional targets remain elusive (Jiang et al. 2013).

946 ***Decoding the dual role of the SL receptor D14***

947 The SL receptor D14 has a dual role as both a receptor and an enzyme, capable of hydrolyzing SL
948 (Hamiaux et al. 2012). Structural biology has played a vital role in unraveling the intricate
949 mechanisms behind SL perception. Crystal structures of D14 from various plant species have
950 shown a common α/β hydrolase fold with a deep ligand binding pocket formed by a V-shaped lid
951 comprised of four α-helices (Guercio et al. 2023, Hamiaux et al. 2012, Kagiyama et al. 2013). The
952 bottom of the ligand-binding pocket contains a conserved serine catalytic triad, highlighting the
953 hydrolase activity that has been preserved throughout plant evolution (Bythell-Douglas et al. 2017).
954 Studies have further examined the evolutionary history of these receptors, and the co-evolution of
955 the residues lining the ligand-binding pocket to provide specificity for diverse SL molecules
956 (Guercio et al. 2023).

957 Yet, an open question centers on the necessity of SL hydrolysis for propagating SL signaling.
958 While it was initially proposed that hydrolysis induced conformational changes in D14, allowing it to
959 interface with D3 (Yao et al. 2016), recent research has challenged this view. Mutants of D14
960 incapable of catalyzing SL hydrolysis were shown to bind SL and rescue *d14* mutant phenotypes in
961 an SL-dependent manner, indicating that SL binding, rather than hydrolysis, can initiate the
962 signaling cascade (Seto et al. 2019). The different states of intact SL binding and SL hydrolysis
963 may convey distinct signals, with MAX2 directing the rate of SL hydrolysis, interactions, and the
964 proteasomal degradation of D14 and SMXLs (Marzec and Brewer 2019, Shabek et al. 2018).

965 ***Mounting complexity: Dynamics of E3 Ub ligase influence substrate degradation***

966 Evidence to date demonstrates that D14 binding to MAX2/D3 is required to mediate the
967 degradation of the target substrate. However, the reported crystal structures containing the D14-D3

complex introduce additional complexity. One such structure reveals a significant conformational change in D14 when complexed with D3, presumably in a post-SL hydrolysis state (Yao et al. 2016). This observation is supported by the identification of a covalently linked intermediate molecule (CLIM) formed during SL hydrolysis, indicating that hydrolysis and conformational change play roles in D14-SL-D3 complex formation.

Another crystal structure unveils a stable form of D14 in complex with the C-terminal helix (CTH) of D3. Furthermore, it was demonstrated that D3 exists in multiple functional conformations, characterized by a flexible, highly conserved CTH (Shabek et al. 2018). This CTH plays an essential role in directly binding D14-SL as a dislodged form, leading to allosteric inhibition of SL hydrolysis. The dislodged state of MAX2/D3 can be triggered by a small molecule like citrate by binding to the D-pocket of the MAX2/D3 protein, displacing the C-terminus D720 and inducing a conformational switch. This regulatory action of citrate is crucial for modulating the spatial arrangement of the D14–D3 complex during SL perception and hydrolysis, as evidenced by augmented inhibition of SL hydrolysis by D14–D3 when treated with citrate (Tal et al., 2022). The D3/MAX2 open conformation provides a new interface for the recruitment of D53/SMXLs and their subsequent ubiquitination and degradation by $SCF^{MAX2/D3}$ (**Fig. 9**). Despite its weak binding affinity, citrate's regulatory role is significant due to its dynamic cellular concentrations, influenced by environmental cues, and its potential impact on the fine-tuning of plant developmental processes and responses to stress.

The link between endogenous SL allocations, phosphate-poor soils, and the overproduction of citrate suggests a complex interplay between SL signaling and organic acid metabolism (Brewer et al. 2013, Liu et al. 2018a, López-Bucio et al. 2002, Saeed et al. 2017, Tahjib-Ul-Arif et al. 2021). Moreover, domain analysis of D53 illustrates four major domains (N, D1, M, and D2 domains) also featured by other AAA+ ATPase family members. It was revealed that the rice D53 D2 domain independently establishes a stable complex with D3-D14 and undergoes degradation through the UPS, suggesting that the D2 domain alone is competent for hormone-induced protein turnover catalyzed by D14-SL-$SCF^{MAX2/D3}$ complex (Shabek et al. 2018). Subsequently, other studies have utilized the D2 domain as the best proxy to follow SL signaling and D53/SMXL-dependent degradation, either in a cell-free system, in planta or as a Strigo-D2 biosensor (Shabek et al. 2018, Song et al. 2021, Tal et al. 2022).

Outlook: Receptor turnover and SL catabolism tug of war

The SL receptor D14 undergoes ubiquitylation and degradation by the 26S proteasome in an SL-dependent manner, creating a negative feedback mechanism (Chevalier et al. 2014, Hu et al. 2017). SL induces the rapid degradation of D53 within few minutes, subsequently regulating the expression of SL-responsive genes while elevating D53 expression after about 1-2 hours (Hu et al. 2017). However, the SL-induced degradation of D14 begins approximately 1 hour after exposure and reaches its peak at around 3-4 hours, indicating that precise feedback loops operating at different time intervals effectively modulate the duration and strength of SL signaling (**Fig. 9**). While

1006 further studies are needed to elucidate the precise mode of action of D14 degradation, it is
1007 hypothesized that upon SL hydrolysis, structural changes of the D14 fold occurs while bound to
1008 MAX2/D3, enabling the mediation of ubiquitylation and degradation of D14 (Chevalier et al. 2014,
1009 Hu et al. 2017, Shabek et al. 2018, Tal et al. 2022). The control of D14 levels and, subsequently,
1010 cellular SL levels hold promise as an intriguing new area of research to better understand the
1011 interplay between SL signaling and the UPS.

1012 While D14 can hydrolyze SLs, it acts as a relatively slow enzyme in the regulation of SL
1013 depletion and/or inactivation. Recently, carboxylesterase CXE15 and CXE20 were found in
1014 Arabidopsis to effectively deplete SL levels (Roesler et al. 2021, Xu et al. 2021). Therefore, it is
1015 possible that the role of D14 in breaking down SLs is insignificant compared to carboxylesterase
1016 activity, and the SL hydrolysis process may solely aim to precisely tune up distinct conformational
1017 states of the enzyme and contribute to the regulation of the SL complex.

1018 In conclusion, the perception and signaling cascade of strigolactone is characterized by its
1019 dynamic and complex nature, differing molecularly from other well-characterized phytohormone
1020 signaling pathways. The multifaceted processes governing strigolactone perception and signaling
1021 regulation through proteasomal degradation represent another evolving phytohormone field in the
1022 intricate world of plant hormones.

1023 Who takes the lead in the intricate dance between autophagy and sugar metabolism?

1024 (By Tamar Avin-Wittenberg)

1025 Autophagy is a vital mechanism for recycling nutrients, mitigating the impact of starvation (Takeshige
1026 et al. 1992, Meijer and Codogno 2004). The initial studies characterizing autophagy-deficient (*atg*)
1027 Arabidopsis mutants underscored their sensitivity to carbon and nitrogen starvation (Doelling et al.
1028 2002, Hanaoka et al. 2002). Despite the connection between cellular carbon status and autophagy
1029 and the reciprocal influence of autophagy on metabolite availability, the interplay between carbon-
1030 containing metabolites, particularly sugars, and autophagy remains unclear. I will outline the known
1031 factors, persisting uncertainties, and challenges in exploring this issue.

1032 Several groups have conducted metabolic profiling of *atg* mutant plants under both favorable and
1033 starvation conditions (Izumi et al. 2013, Masclaux-Daubresse et al. 2014, Avin-Wittenberg et al.
1034 2015, Barros et al. 2017, McLoughlin et al. 2018, McLoughlin et al. 2020). Most studies reported
1035 changes in amino acid levels, aligning with autophagy's role as a protein degradation mechanism
1036 (**Fig. 10**). However, some studies also observed alterations in sugar levels. For instance, slight
1037 sucrose accumulation was observed in Arabidopsis and maize (*Zea mays*) *atg* mutants under
1038 favorable conditions (Masclaux-Daubresse et al. 2014, Barros et al. 2017, McLoughlin et al. 2018).
1039 Additionally, Raffinose family oligosaccharides (galactinol, raffinose, and stachiose) accumulated in
1040 *atg* mutants under favorable and carbon starvation conditions (Masclaux-Daubresse et al. 2014,
1041 Avin-Wittenberg et al. 2015, McLoughlin et al. 2018).

1042 Defining a consistent “sugar fingerprint” for autophagy deficiency proves challenging, as many
1043 of the changes in sugar levels are experiment-specific. Starch is also important in the context of
1044 carbon supply, as autophagy was suggested to function in starch breakdown in Arabidopsis (Wang
1045 et al. 2013b), and a cross between starchless and *atg* mutants increased cell death under short-day
1046 conditions (Izumi et al. 2013). Interestingly, maize *atg12* mutants demonstrated increased starch
1047 breakdown under carbon starvation (McLoughlin et al. 2020), while Arabidopsis *atg* mutants
1048 exhibited starch accumulation under carbon starvation (Barros et al. 2017), suggesting that
1049 autophagy-starch regulation is more complex **(Fig. 10)**.

1050 Several factors could explain the variations in sugar phenotypes observed in *atg* mutant
1051 experiments. First, two plant species were analyzed in the aforementioned studies, and the
1052 differences might point to species-specific roles of autophagy. As the study of plant autophagy
1053 expands to more species, additional data may reveal common factors in autophagy and metabolism.
1054 Secondly, the variability in Arabidopsis plant age during experiments adds complexity, potentially
1055 influencing metabolomics results. The variability may imply that autophagy could assume distinct
1056 roles at different stages of plant development, influencing sugar metabolism. Finally, the setup of
1057 carbon starvation may trigger a differential metabolic response. Previous studies have demonstrated
1058 that individual-leaf darkening induces leaf senescence, while whole-plant darkening inhibits it
1059 (Weaver and Amasino 2001). Moreover, a different metabolic response has been observed in both
1060 scenarios (Law et al. 2018). It is plausible that autophagy operates differently under these carbon
1061 starvation conditions, resulting in diverse metabolic outcomes **(Fig. 10)**.

1062 Several studies investigated autophagy and sugar excess. Arabidopsis *atg* mutant seedlings are
1063 less sensitive to elevated glucose and sucrose, but not fructose levels, displaying reduced inhibition
1064 of root growth upon high sugar treatment (Huang et al. 2018, Laloum et al. 2022). Surprisingly, this
1065 reduced inhibition of root growth is not attributed to changes in sugar metabolism or accumulation.
1066 Instead, it is connected to reduced reactive oxygen species (ROS) accumulation in the roots and the
1067 persistence of auxin levels in *atg* mutants, possibly due to reduced pexophagy, allowing for reduced
1068 inhibition of root growth **(Fig.10)** (Huang et al. 2018).

1069 There is another point to consider when studying metabolism in knockout mutants. The metabolic
1070 phenotype may not arise solely from the absence of autophagy but could also stem from pleiotropic
1071 effects of the mutation. Additionally, the role of autophagy in nutrient remobilization from source to
1072 sink tissues adds further complexity to the analysis. For instance, sucrose accumulation was
1073 observed in *atg12* mutant maize seeds (Barros et al. 2023) and *ATG4-RNAi* tomato (*Solanum*
1074 *lycopersicum*) fruit (Alseekh et al. 2022). However, whether this accumulation is a consequence of
1075 autophagy deficiency in the source or sink tissue remains uncertain. Reciprocal crosses between
1076 wild-type and *atg* mutant Arabidopsis plants did not reveal significant differences in sugar and lipid
1077 levels (Erlichman et al. 2023). This suggests that autophagy primarily functions in nitrogen
1078 remobilization rather than carbon remobilization. Thus, it is speculated that sucrose accumulation
1079 may result from localized autophagy effects rather than carbon remobilization from the source.

1080 How are sugar levels involved in the regulation of autophagy? Two key kinase complexes, target
1081 of rapamycin (TOR) and Snf1-related protein kinase 1 (SnRK1), play a crucial role in nutrient sensing
1082 (Janse van Rensburg et al. 2019). TOR promotes plant growth and inhibits autophagy, while SnRK1
1083 induces autophagy in response to starvation. SnRK1 activation of autophagy can occur either
1084 through TOR inhibition or direct activation. TOR is activated by various signals, including cellular
1085 glucose levels, though the specific details of this activation remain unknown (Mugume et al. 2020).
1086 SnRK1 senses energy and nutrient levels through adenine nucleotides (ATP, ADP, or AMP) or sugar
1087 phosphates. Trehalose 6-phosphate (T6P) acts as a negative regulator of SnRK1, representing
1088 cellular sucrose levels (Jamsheer K et al. 2021). Recent research also revealed a connection
1089 between three glycolytic enzymes and autophagy inhibition. These enzymes bind to ATG101, a
1090 regulatory subunit of the kinase ATG1, to restrict its activity (Lee et al. 2023).

1091 Recent findings indicate that sugars can induce autophagy. The stress-related sugar trehalose
1092 accumulates in Arabidopsis during carbon starvation (Barros et al. 2017), and its buildup during
1093 desiccation in the resurrection plant *Tripogon loliiformis* led to autophagy activation (Williams et al.
1094 2015). Additionally, inhibiting trehalose degradation in maize activated autophagy and enhanced
1095 plant biomass (Sun et al. 2022) **(Fig. 10)**. These results align with a growing body of evidence linking
1096 autophagy enhancement with increased plant performance (Minina et al. 2018).

1097 In summary, the complex relationship between autophagy and sugar metabolism poses
1098 challenges in distinguishing cause and effect. The development of innovative tools, including
1099 inducible mutant lines and tissue-specific downregulation, is essential to mitigate the pleiotropic
1100 effects of knockout mutants. Furthermore, uncovering novel sugars that regulate autophagy and
1101 understanding their mode of action will contribute to a better understanding of this elaborate co-
1102 regulation.

1103 What is the role of proteolysis in fruit ripening regulation?

1104 (By Sergey Mursalimov and Simon Michaeli)

1105 Humans and other animals benefit from the ability of plant organs to survive detached while
1106 temporarily maintaining their taste, scent, and nutritional values. However, the lifespan of harvested
1107 leaves, roots, tubers, and fruits is limited without the continued supply of water and nutrients. Hence,
1108 their existence is, by default, subject to stressful conditions of energetic deprivation and dehydration.
1109 Moreover, due to trade and consumer requirements, fresh plant produce will experience extreme
1110 temperature and humidity fluctuations and be subjected to mechanical damage (Pedreschi and Lurie
1111 2015, Al-Dairi et al. 2022). The latter further accelerates the appearance of biotic stressors, mostly
1112 fungi (Prusky and Romanazzi 2023). Changes in oxygen and CO_2 levels during storage can affect
1113 respiration and also lead to oxidative damage (Pedreschi and Lurie 2015). These stressors may
1114 trigger protein misfolding and aggregation (Liu and Howell 2016), negatively affecting fresh produce's
1115 quality and shelf life. Food loss and waste is estimated at around 40% worldwide (Porat et al. 2018)

1116 and is accompanied by economic damage and carbon footprint. Therefore, increasing plant-based
1117 food security requires both increasing crop yields and decreasing food loss waste (Foley et al. 2011).

1118 Proteolysis pathways are pivotal in plant development, senescence, and stress responses and,
1119 as such, may prove to be efficient targets for plant-based reductions in food loss waste. The
1120 postharvest research field is vast. Here, I will focus on climacteric fruit ripening. Climacteric fruits,
1121 such as bananas, mangos, apples, and tomatoes, can ripen postharvest, and their ripening is
1122 accompanied by respiration and ethylene bursts (Cherian et al. 2014).

1123 ***The UPS in ripening***

1124 The UPS is essential in ripening, primarily due to its involvement in the tight regulation of
1125 phytohormones, including auxin, abscisic acid (ABA), and ethylene (Fenn and Giovannoni 2021).
1126 Notably, UPS components identified as hormone modulators in Arabidopsis were later found
1127 necessary in fruit ripening. For example, Ethylene-Insensitive 3 (EIN3)-Binding F-box (EBF)
1128 proteins are known to target EIN3, a key transcription factor in ethylene signaling, for proteasomal
1129 degradation (Guo and Ecker 2003, Potuschak et al. 2003). More recently, the role of EBFs in
1130 tomato and pear ripening (Deng et al. 2018, Wang et al. 2023a, Yang et al. 2010) and carnation
1131 petal senescence (Zhu et al. 2023) was demonstrated. The UPS may also regulate fruit traits
1132 independently of hormonal crosstalk. Fruit color has ecological and postharvest implications,
1133 affecting animal-borne seed dispersal and retail consumption.
1134 In some fruits, such as tomatoes and bananas, color is used to assess the ripening stage and
1135 is determined by the ratio between two plastid types, chloroplasts and chromoplasts (Morelli et al.
1136 2023). With ripening progression, chlorophyll-containing chloroplasts are gradually transformed
1137 into carotenoid-containing chromoplasts (**Fig. 11**). In Arabidopsis, SP1, a RING-type ubiquitin E3-
1138 ligase was found pivotal in the CHLORAD pathway (See the section by Fang, Peixoto, and Jarvis
1139 herein). Recently, the tomato homologs SP1 and SP1-Like 2 (SPL2) were proposed as
1140 instrumental for chloroplast-to-chromoplast transition, suggesting that regulation of the plastid
1141 protein import machinery is vital for this plastid reformation (Ling et al. 2021).

1142 ***Beyond the UPS: Autophagy in ripening***

1143 Autophagy is another crucial cellular quality control and recycling mechanism involved in senescence
1144 and plant responses to stress. Therefore, it is a candidate target for the shelf-life extension of any of
1145 the fresh produce types. Autophagy is generally induced during cellular reprogramming (Rodriguez et
1146 al. 2020). Considering the dramatic cellular transformation during ripening (**Fig. 11**), it is surprising
1147 how little we know about autophagy`s role in this process. Recently, autophagy activity was shown to
1148 fluctuate during pepper and strawberry ripening (López-Vidal et al. 2020, Sánchez-Sevilla et al.
1149 2021), and it was suggested to promote the ripening of the latter. Both these fruits are non-
1150 climacteric, meaning that their ripening is not associated with ethylene and respiration bursts (Perotti
1151 et al. 2023). On the other hand, in the climacteric tomato fruits, we have shown that autophagy
1152 restricts ripening by repressing ethylene production (Kumaran et al. 2023). It is tempting to speculate
1153 that the disparate function of autophagy in climacteric and non-climacteric fruits results from the

1154 differential role of ethylene between these two fruit types. It further highlights our insufficient
1155 knowledge of the interaction of autophagy with phytohormones (Liao et al. 2022). Transmission
1156 electron microscopy of tomato fruit pericarp cells suggests the vacuolar degradation of plastids
1157 during ripening (**Fig. 11C, 11E**). Nonetheless, it still needs to be determined whether this is mediated
1158 via a macro- or micro-autophagy process (Izumi et al. 2017).

1159 ***Proteolysis in postharvest regulation***

1160 Proteolysis pathways are highly selective toward specific targets within an explicit spatiotemporal
1161 environment (Clavel and Dagdas, 2021), ideal for targeting individual traits. Can proteolysis pathways
1162 be harnessed for postharvest trait regulation and fresh produce shelf-life extension? To answer this,
1163 we first need to know whether there are applicable ways to induce or repress specific proteolysis
1164 pathways postharvest, avoiding fitness costs during the plant life cycle. For example, the constitutive
1165 knock-down of tomato *Autophagy-related 4* (*ATG4*) resulted in early leaf senescence and a
1166 considerably low fruit yield (Alseekh et al. 2022). However, when the same silencing was employed in
1167 a ripening-specific manner, these phenotypes were absent, and instead, the role of autophagy in
1168 ripening repression was revealed (Kumaran et al. 2024). This highlights the necessity of examining
1169 proteolysis pathways specifically within a postharvest context, and further, raises the challenge of
1170 uncovering and editing genetic segments that may be exclusively functional during postharvest. An
1171 alternative for genetic manipulation may be using compounds targeting proteolysis pathways that will
1172 be applied to fresh produce. Such compounds would need to be both human- and eco-friendly.

1173 In conclusion, proteolysis pathways are pivotal for fresh produce's quality and shelf life.
1174 Understanding the mechanisms governing these processes during postharvest may advance our
1175 ability to reduce food loss and waste and help ensure access to high-quality and nutritious produce.

1176 Roles of proteolysis in plant responses to biotic/abiotic signals

1177 How does ERAD function in model plants and crops?

1178 (By Qian Chen, Qi Xie, and Feifei Yu)

1179 Abiotic and biotic stresses can trigger misfolded protein accumulation in the endoplasmic reticulum
1180 (ER), causing ER stress. The unfolded protein response (UPR), ER-associated degradation
1181 (ERAD), and autophagy are three main mechanisms for relieving ER stress (Liu and Li 2014).
1182 Among them, ERAD involves the ER-located Ub modification system and the cytosolic proteasome
1183 degradation system for protein degradation (Romisch 2005). Traditionally, ERAD is responsible for
1184 identifying misfolded proteins in the ER lumen or membrane and facilitating their degradation
1185 (Romisch 2005). Recent studies have expanded the role of ERAD in the homeostasis of functional
1186 PM and cytoplasmic proteins to modulate various plant biological processes (Zhang et al. 2021, Li
1187 et al., 2023, Pan et al., 2020, Wang et al. 2023b). Here, we focus on progress in the study of ERAD
1188 in plant development and stress responses, broadening insights from model plants to crops.

1189 The role of ERAD in plant abiotic stress response has primarily been studied in Arabidopsis

1190 (**Fig. 12A**). A key ERAD component, UBC32, an ER membrane-anchored Ub-conjugating enzyme
1191 (E2), is transcriptionally induced by salt and drought stresses (Cui et al. 2012). UBC32 plays a role
1192 in the brassinosteroid (BR)-mediated salt stress response. Park et al. (2018) showed that the
1193 soluble ERAD component, ERAD-mediating RING finger protein (AtEMR), forms a complex with
1194 UBC32, negatively regulating salt stress resistance. Additionally, UBC32 cooperates with the
1195 RING-type E3 ligase Rma1 as an E2-E3 pair, enhancing plant drought tolerance by facilitating the
1196 degradation of aquaporin PIP2;1 (Chen et al. 2021b). UBC32, together with its homologs UBC33
1197 and UBC34, also participates in ABA signaling by degrading the phosphorylated ABA transporter
1198 NITRATE TRANSPORTER 1.2/PEPTIDE TRANSPORTER 4.6 (NRT1.2/NPF4.6) (Zhang et al.
1199 2021). Since ABA is crucial in plant drought response, it is still an open question whether UBC32
1200 responds to drought via ABA signaling. Another E2, UBC27, an ortholog of the yeast ERAD
1201 component Ubc1p, interacts with ABA-INSENSITIVE RING PROTEIN 3 (AIRP3). The UBC27-
1202 AIRP3 interaction is enhanced by ABA which leads to ubiquitylation and degradation of ABA co-
1203 receptor ABA-INSENSITIVE 1 (ABI1), thus activating ABA signaling and improving drought
1204 tolerance (Pan et al. 2020). These findings from Arabidopsis indicate that ERAD plays crucial roles
1205 in stress-related hormone signaling and plant adaptation to environmental stress.

1206 Although significant progress has been made in understanding ERAD in model plants, our
1207 understanding of ERAD in crop stress response and growth is limited. In *Medicago falcata*, plant-
1208 specific E3 ligase MfSTMIR participates in the ERAD pathway via interacting with MtUBC32 to
1209 relieve ER stress under salt stress (Zhang et al. 2019b). This finding underscores the significance
1210 of ERAD pathways in crop salt stress response. However, further studies are required to explore
1211 their roles in other stress conditions.

1212 Recent studies have highlighted the role of ERAD in biotic stress resistance in a few crops
1213 (**Fig. 12B**). In rice, overexpression of *OsUBC45*, the ortholog of Arabidopsis UBC32, exhibited
1214 improved resistance to rice blast and bacterial leaf blight by promoting the degradation of
1215 OsPIP2;1, which attenuates disease resistance by mediating the translocation of H_2O_2 from the
1216 cytosol to the apoplast (Wang et al. 2023b). The ERAD related RING type E3 Ub ligase Decreased
1217 Grain Size 1 (DGS1) improved resistance to rice blast, through forming an E2-E3 pair with
1218 OsUBC45 to enhance the degradation of OsPIP2;1 (Wang et al. 2024) . In foxtail millet (*Setaria*
1219 *italica*), the overexpression of the ERAD related RING type E3 Ub ligase Small Grain and Dwarf
1220 (SGD1) also increased blast resistance, though the mechanism remains undefined (Tang et al.
1221 2023). Whether SiUBC32 in millet also contributes to disease resistance needs to be further
1222 explored.

1223 Significant advances have been made in understanding the crucial role of ERAD-related E2-
1224 E3 in grain yield in graminaceous cereals (**Fig. 12B**). In rice, SMALL GRAIN 3 (SMG3), the other
1225 name of OsUBC45, works together with E3 ligase DGS1 to positively regulate grain size by
1226 facilitating the degradation of the misfolded BR receptor BRI1 (Li et al. 2023a). Intriguingly, the
1227 same E2-E3 pair OsUBC45/SMG3-DGS1 also enhances rice yield by targeting GLYCOGEN
1228 SYNTHASE KINASE 3 (OsGSK3), a negative component in brassinosteroid (BR) signaling, for

1229 ubiquitylation-dependent degradation (Gao et al. 2019b, Wang et al. 2023b). In millet, the SGD1-
1230 SiUBC32 pair also boosts yield by strengthening BR signaling. They catalyze Ub attachment to
1231 BRI1 but lead to an accumulation of functional BRI1 rather than its degradation (Tang et al. 2023).
1232 Additionally, the role of SGD1 in regulating seed size is also conserved in wheat (*Triticum*
1233 *aestivum*) and maize (Tang et al. 2023). These studies in crops demonstrate that this specific E2-
1234 E3 pair could contribute to crop yield by enhancing BR signaling through regulating both positive
1235 and negative components involving in BR recognition and signal transduction. Further efforts
1236 should explore the role of DGS1 orthologs in wheat and maize disease resistance and determine
1237 whether the SMG3/OsUBC45/SiUBC32-DGS1/SGD1 pair acts as an E2-E3 pair, targeting different
1238 substrates to improve both yield and disease resistance in staple crops. Moreover, *OsUBC11* in
1239 rice, the ortholog of *AtUBC7* in Arabidopsis, which encodes ERAD components, is implicated in
1240 root development at the seedling stage by affecting auxin signaling (Han et al. 2023), implying the
1241 potential roles of other ERAD components in crop development.

1242 ERAD components play a significant role in phytohormone signaling, including ABA, BR, and
1243 auxin pathways, and crosstalk among these hormones has been known to balance plant
1244 development and stress response (Li et al. 2023, Song et al. 2023, Tang et al. 2023, Wang et al.
1245 2020, Wang et al. 2023b, Yu and Xie 2024, Zhang et al. 2021). It is important to note that although
1246 the function of ERAD is largely conserved across eukaryotes, there may be differences between
1247 model plant and crops. For instance, the mutants of *UBC32* in Arabidopsis show minimal impact on
1248 plant growth and seed size (Cui et al. 2012), whereas its orthologs in rice and millet are essential
1249 factors for both growth and yield (Li et al. 2022, Tang et al. 2023, Wang et al. 2023b), which may
1250 be partially explained by the distinct BR signaling between Arabidopsis and rice. Therefore, it is
1251 crucial to reveal the specific role of other ERAD components, such as the E3 Ub ligase HRD1 and
1252 DOA10 in crop growth and environmental stress interaction, which will provide us the possibility of
1253 utilizing them in crop breeding.

1254 How is chloroplast-associated protein degradation (CHLORAD) regulated in 1255 response to developmental and environmental cues?

1256 **(By Jun Fang, Bruno Peixoto, and R. Paul Jarvis)**

1257 Chloroplasts are essential plant organelles, not only for photosynthesis but also for the biosynthesis
1258 of many important primary and secondary metabolites (Lopez-Juez and Pyke 2005, Sun and Jarvis
1259 2023). Chloroplasts originated through endosymbiosis from an ancient cyanobacteria-like
1260 photosynthetic prokaryote (Reyes-Prieto et al. 2007), and the modern organelles retain a functional
1261 genome with roughly 100 protein-coding genes. However, most endosymbiont genes were
1262 transferred to the host nuclear genome during evolution. Consequently, >90% of the ~3000 different
1263 chloroplast proteins are nucleus-encoded and must be imported following synthesis as precursors in
1264 the cytosol (Li and Chiu 2010, Shi and Theg 2013, Sun and Jarvis 2023).

1265 Import of these precursors into chloroplasts requires translocons in the outer and inner
1266 chloroplast envelope membranes (TOC and TIC, respectively). The TOC complex exists in different,

1267 client-specific subtypes (Li and Chiu 2010, Shi and Theg 2013). The main TOC subtype, comprising
1268 the receptors Toc33 and Toc159 and the channel Toc75, preferentially imports precursors of the
1269 photosynthetic apparatus. In contrast, a minor TOC subtype with a different complement of receptors
1270 (Toc34, Toc132 and Toc120) tends to import non-photosynthetic, housekeeping precursors (Kessler
1271 and Schnell 2009, Li and Chiu 2010). The TOC machinery acts as the gateway controlling entry of
1272 the desired types of precursors. The guardian that regulates the TOC machinery is the so-called
1273 chloroplast-associated protein degradation (CHLORAD) system. CHLORAD is an arm of the UPS,
1274 and it targets TOC proteins for ubiquitylation and degradation, thereby controlling the import of
1275 precursor proteins. A separate system proteolytically removes unimported precursors to prevent their
1276 cytosolic accumulation (Grimmer et al. 2020, Lee et al. 2009, 2016), although whether or how this
1277 system is coordinated with CHLORAD is not known. The CHLORAD system is composed of the
1278 RING-type E3 Ub ligase SUPPRESSOR OF PPI1 LOCUS1 (SP1), an Omp85-type β-barrel protein
1279 SP2, and a cytosolic AAA-ATPase motor CDC48 (Ling et al. 2012, Ling et al. 2019). As we
1280 summarize below, CHLORAD plays important roles both developmentally and under stress
1281 conditions. However, there are major outstanding questions concerning how CHLORAD is regulated
1282 by different developmental and environmental cues (**Fig. 13**).

1283 Chloroplasts are the best-known type of plastid, but there are several other plastid types in
1284 non-green plant tissues. A remarkable feature of these different plastid types is their ability to
1285 interconvert in response to developmental or environmental signals. Such plastid-type
1286 interconversions involve the remodeling of the plastid proteome, which is controlled at least in part
1287 by differential regulation of protein import, particularly at the TOC machinery (Jarvis and López-
1288 Juez 2013, Nellaepalli et al. 2023). CHLORAD degrades TOC proteins to facilitate their
1289 replacement by others, thereby controlling plastid protein import, the organellar proteome, and
1290 plastid transitions.

1291 Work in Arabidopsis showed that loss of SP1 leads to delayed de-etiolation and leaf
1292 senescence, whereas overexpression of SP1 promotes these processes by accelerating plastid
1293 transitions (i.e., etioplast-to-chloroplast and chloroplast-to-gerontoplast transitions, respectively).
1294 Such functions of CHLORAD appear well-conserved among plant species, as its manipulation
1295 similarly affects leaf senescence in tomato, as well as fruit ripening during which chromoplast
1296 formation occurs (Ling et al. 2012, Ling et al. 2021). Recently, two homologs of SP1, namely SP1-
1297 like1 (SPL1) and SPL2, were shown to regulate CHLORAD in an antagonistic manner (Mohd. Ali et
1298 al. 2023). While SPL2 exhibits partial redundancy with SP1, SPL1 negatively regulates SP1
1299 potentially through competitive interaction with other factors (Ling et al. 2021, Mohd. Ali et al.
1300 2023). Both SPL proteins are important for leaf senescence, like SP1. However, it remains unclear
1301 how CHLORAD perceives developmental signals, and how it selectively degrades different TOC
1302 components during different developmental phases.

1303 Different TOC subtypes are regulated transcriptionally in different plant tissues and
1304 developmental stages (Demarsy et al. 2014). In contrast, the *SP1* and *SP2* genes show
1305 comparable expression profiles across different tissues and stages (Ling et al. 2019). Therefore, it

1306 is likely that SP1 and SP2 are regulated post-translationally. Studies have shown that TOC
1307 receptors can be phosphorylated in vitro and in vivo, and such phosphorylation may inhibit TOC
1308 complex assembly, GTP binding, and precursor binding (Demarsy et al. 2014). For example,
1309 physiological analyses suggested that phosphorylation at residue S181 reduces Toc33 activity and
1310 impairs chloroplast biogenesis at early developmental stages, but not later growth (Aronsson et al.
1311 2006, Oreb et al. 2007). A kinase at the outer chloroplast membrane (KOC1) phosphorylates the
1312 A-domain of Toc159 in vitro, and contributes to efficient protein import and chloroplast biogenesis
1313 during de-etiolation (Zufferey et al. 2017). Differential phosphorylation of Toc159 has also been
1314 described when the carbon-sensing kinase SnRK1 (sucrose nonfermenting 1-related protein
1315 kinase 1) is genetically manipulated, with SnRK1α1 gain-/loss-of-function lines showing
1316 higher/lower levels of phosphorylated Toc159, respectively (Cho et al. 2016a, Nukarinen et al.
1317 2016). Moreover, phosphorylation of Toc159 family proteins by SnRK2, and reduced import
1318 efficiency in an abscisic acid (ABA) biogenesis deficient mutant, implies crosstalk between ABA
1319 signaling and protein import regulation (Zhong et al. 2010, Wang et al. 2013a). There is presently
1320 no information on whether CHLORAD components undergo differential phosphorylation at different
1321 developmental stages, although SP1 and SP2 are predicted to have 18 and 15 phosphorylation
1322 sites, respectively (Chen et al. 2023a). Phosphorylation may regulate E3 ligase activity, substrate
1323 recognition, or substrate/ligase interaction (Hunter 2007).

1324 Besides its developmental role, CHLORAD is also critically important for abiotic stress
1325 tolerance in plants (Ling and Jarvis 2015). Under stress conditions, chloroplasts overproduce
1326 reactive oxygen species (ROS), harmful photosynthetic by-products that can oxidize
1327 macromolecules and affect organellar structural and functional integrity (Li and Kim 2022). During
1328 stress, CHLORAD degrades TOC proteins to limit the import of photosynthesis-related proteins,
1329 thereby suppressing photosynthetic activity and reducing ROS production and photo-oxidative
1330 damage (Ling and Jarvis 2015). In addition to their toxicity, ROS also function as signaling
1331 molecules via the redox modification of specific amino acid residues, for example at cysteine thiol
1332 groups (Li and Kim 2022). Evidence suggests that conserved cysteines in TOC components may
1333 be regulated by redox modification, thereby influencing protein import (Kessler and Schnell 2009,
1334 Balsera et al. 2010). Interestingly, Toc75 was found to be oxidized at C219 within its polypeptide
1335 transport-associated (POTRA) domain, after hydrogen peroxide treatment (Doron et al. 2021).
1336 While SP1, SPL1, and SPL2 share several conserved cysteines, it is currently unknown whether
1337 these cysteine residues are essential for function or involved in redox-mediated regulation (Ling et
1338 al. 2012, Ling et al. 2021). It will be interesting to investigate whether the CHLORAD apparatus is
1339 regulated by redox modification directly or by altered affinity towards redox-modified/unmodified
1340 TOC proteins. This might enable rapid limitation of the import of photosynthesis proteins and
1341 further ROS production.

1342 Aside from such direct effects, ROS also induce stress-responsive gene expression changes
1343 by transmitting signals from chloroplasts to the nucleus. This is referred to as retrograde signaling,
1344 and it plays important roles in maintaining cellular homeostasis and acclimation to stressful

1345 environments (Li and Kim 2022). For example, singlet oxygen ($^1O_2$) oxidizes β-carotene to produce
1346 β-cyclocitral, which induces detoxification-related nuclear genes via the scarecrow-like 14 (SCL14)
1347 transcription factor, or salicylic acid (SA)-responsive genes through a chloroplast SA-synthesis
1348 enzyme, isochorismate synthase1 (ICS1) (Lv et al. 2015, D'Alessandro et al. 2018). Chloroplast-
1349 resident executer1 (EX1) protein is also oxidized by $^1O_2$ at its W643 residue, inducing $^1O_2$-
1350 responsive gene expression (Dogra et al. 2019). Thus, the question arises: Does ROS-induced
1351 retrograde signaling play a role in regulating CHLORAD? One possibility is that the expression of
1352 *SP1* and *SP2* is regulated under abiotic stresses. However, differential expression of these genes
1353 was not observed under several abiotic stresses (Hruz et al. 2008, Coolen et al. 2016, Garcia-
1354 Molina and Pastor 2023). Another possibility is that CHLORAD is a target of stress-responsive
1355 proteins regulated by retrograde signaling, for example, proteins involved in stress-related
1356 phytohormone signaling or ROS-triggered responses (Li and Kim 2022).

1357 In addition to ROS, cytosolic calcium, various phytohormones including ABA, and diverse
1358 kinase subfamilies such as type 2C protein phosphatase (PP2C), SnRK2, SnRK1, and mitogen-
1359 activated protein kinases (MAPKs), also play major signal transduction roles during abiotic stress
1360 (Belda-Palazón et al. 2020, Zhang et al. 2022). Therefore, it is conceivable that CHLORAD
1361 perceives stress signals transduced from the PM or cytosol as part of an integrated cellular stress
1362 response. The calcineurin B-like 10 (CBL10) protein, a member of the CBL family that perceives
1363 and transmits Ca^{2+} signals to CBL-interacting protein kinases, was found to interact with Toc34 and
1364 negatively regulate its GTPase activity (Cho et al. 2016b). As mentioned above, ABA signaling can
1365 influence the phosphorylation of Toc159 family proteins. Thus, emerging evidence suggests
1366 possible crosstalk between Ca^{2+} and hormone signaling and protein import regulation, although
1367 whether CHLORAD plays any role in this is unknown. It is clear that post-translational modification
1368 of substrate proteins, such as acetylation, phosphorylation, and SUMOylation, can alter substrate
1369 recognition by RING E3 ligases (Metzger et al. 2014). In this regard, it is noteworthy that the E2
1370 SUMO conjugase SCE1 is implicated in the SUMOylation of TOC proteins to promote their
1371 degradation, possibly through CHLORAD activity (Watson et al. 2021). However, it is unclear
1372 whether SUMOylation affects substrate recognition by SP1 or some other step such as CDC48
1373 recruitment.

1374 Many important questions remain concerning CHLORAD action and the regulation of
1375 chloroplast protein import. We look forward to seeing significant new light shed in this intriguing
1376 area in the future. Because of its importance for plastid development and plant stress responses, a
1377 greater understanding of CHLORAD regulation may prove invaluable in efforts to improve crop
1378 performance concerning yield, quality, and stress resilience.

1379 How does autophagy contribute to drought tolerance?

1380 (By Diane C. Bassham)

1381 Autophagy, a pathway leading to the degradation of cellular components in the vacuole, is
1382 activated by numerous abiotic stresses, including drought (Agbemafle et al. 2023). The activities of

1383 two major kinases are responsible for regulating autophagy under many conditions: the Target of
1384 Rapamycin (TOR) complex, a negative regulator, and Snf1-Related Kinase 1 (SnRK1), a positive
1385 regulator. Downstream of these kinases, a suite of ATG (autophagy-related) proteins function in
1386 the *de novo* production of double-membrane vesicles termed autophagosomes that enwrap the
1387 cargo to be degraded. The cargo is delivered into the vacuole, degraded by vacuolar hydrolases,
1388 and the degradation products are recycled into the cytoplasm (Agbemafle et al. 2023). Disruption
1389 of autophagy by mutating core autophagy genes or by using inhibitors decreases drought tolerance
1390 (Liu et al. 2009). Overexpression in Arabidopsis of *ATG* genes derived from crop species leads to
1391 improved drought tolerance (Fu et al. 2020, Li et al. 2019, 2015, Chen et al. 2022, Yue et al. 2022),
1392 as does overexpression of *ATG18* and *ATG8* homologs in apple (Sun et al. 2018, Jia et al. 2021).
1393 These data indicate a critical function for autophagy in survival during drought, including in
1394 economically important crop species, but the mechanisms by which autophagy acts to allow
1395 survival remain an open question.

1396 Several pathways by which plants activate autophagy during drought have been identified,
1397 although how they work together remains unclear (**Fig. 14**). In a number of plant species, *ATG*
1398 genes are upregulated by drought, and autophagy activity increases (Tang and Bassham 2022,
1399 Agbemafle et al. 2023); several transcription factors have been identified that can control the
1400 expression of these *ATG* genes during drought (Agbemafle et al. 2023). As for other stresses, TOR
1401 and SnRK1 kinases are critical in activating autophagy during drought, with repression of TOR
1402 activity and increased SnRK1 activity leading to activation (Soto-Burgos and Bassham 2017, Pu et
1403 al. 2017, Chen et al. 2017a). It is becoming clear that sulfide signaling also regulates stress
1404 responses such as autophagy (Jurado-Flores et al. 2023). The core autophagy protein ATG4 is
1405 persulfidated and inactivated upon osmotic stress or ABA treatment, leading to a downregulation of
1406 autophagy (Laureano-Marín et al. 2020), potentially to prevent over-activation and cell death.

1407 While it is well established that autophagy is activated during drought and aids in drought
1408 tolerance, major questions remain of the pathways by which autophagy contributes to tolerance.
1409 Several distinct mechanisms have been proposed, but which ones predominate and how these
1410 mechanisms are integrated is not yet known, and are crucial topics for future research.

1411 ***Degradation of oxidized and/or aggregated proteins.*** Several reports indicate that autophagy is
1412 important for clearing aggregated or oxidized proteins during drought and other stresses (Sun et al.
1413 2018, Zhou et al. 2013b) and that the activities of antioxidant pathways correlate with autophagy
1414 activity (Jia et al. 2021, Li et al. 2019). This potentially could relieve cytotoxic stress caused by
1415 accumulation of damaged proteins and other macromolecules.

1416 ***Regulation of aquaporin activity.*** Aquaporins are channels that control the flux of water and
1417 other small molecules across membranes. In both Medicago (Li et al. 2020a) and Arabidopsis
1418 (Hachez et al. 2014), PM aquaporins are recognized by selective autophagy receptors and
1419 degraded during drought, although the receptors in each species are distinct. This degradation is
1420 proposed to reduce water loss from cells and improve drought tolerance.

1421 ***Regulation of stomatal dynamics.*** Interesting recent findings implicate autophagy in stomatal
1422 dynamics, which are critical to prevent loss of water in conditions of water deficit. Reactive oxygen
1423 species signal in response to environmental stress to inhibit stomatal opening and/or promote
1424 closing; ROS homeostasis in guard cells therefore may be important for drought tolerance.
1425 Autophagy is required for maintaining basal levels of ROS, and Arabidopsis mutants defective in
1426 autophagy have high ROS levels in guard cells, with defects in stomatal movement (Yamauchi et
1427 al. 2019). Interestingly, ABA responses, guard cell opening and ROS homeostasis have all been
1428 linked to regulation by protein persulfidation, suggesting that the integration of drought responses
1429 may involve sulfide signaling (Jurado-Flores et al. 2023).

1430 ***Regulation of growth.*** The transcription factor BES1 controls growth in response to
1431 brassinosteroid signaling. During drought, BES1 is degraded by selective autophagy via the DSK2
1432 receptor, leading to decreased growth and increased drought tolerance (Nolan et al. 2017). In
1433 general, water use efficiency (i.e., the ratio between water used by the plant and that lost by
1434 transpiration) is correlated with leaf area, but unexpectedly not with stomatal density or ABA levels,
1435 suggesting a complex relationship between plant size, water use and drought tolerance (de Ollas
1436 et al. 2023). This is demonstrated in the case of Arabidopsis *cost1* (constitutively stressed)
1437 mutants, which have greatly reduced growth, constitutive autophagy, and are highly drought
1438 tolerant (Bao et al. 2020). The drought tolerance requires active autophagy, and COST1 inhibits
1439 autophagy by interacting with the autophagy machinery. During drought, COST1 is degraded,
1440 releasing the inhibition of autophagy and increasing drought tolerance. Intriguingly, recent work
1441 showed that *cost1* mutants are drought tolerant because they use less water due to their extreme
1442 dwarfism, and that when grown together in the same pot, no drought tolerance is observed
1443 (Ginzburg et al. 2022). However, the *cost1* drought tolerance phenotype can be rescued by
1444 blocking autophagy, but the growth phenotype cannot, indicating that plant size and drought
1445 tolerance can be uncoupled in this mutant (Bao et al. 2020).

1446 These data all indicate an important role for autophagy in drought responses, but also raise
1447 many questions about the precise role of autophagy in these responses, the mechanisms by which
1448 drought is perceived and autophagy is activated, and the integration of stress, growth, and
1449 developmental pathways to allow plant survival. New approaches including the physiological
1450 characterization of higher order mutants in different aspects of the drought response, identification
1451 of additional factors, for example by protein-protein interaction, that link autophagy to drought
1452 tolerance, and non-targeted approaches such as suppressor screens are needed to determine the
1453 relationships between identified pathways and to clarify direct and indirect contributions of proteins
1454 and pathways to drought tolerance. The activity and role of autophagy in the phenotypes of known
1455 drought-tolerant or sensitive mutants also deserve investigation.

1456 How does the fine-tuning of proteasome regulation impact the trade-off between 1457 growth and defense?

1458 **(By Suayib Üstün)**

1459 Trade-offs, situations when a beneficial change in one feature comes with a detrimental change in
1460 another, are inherent to life (Garland 2014). One of the most prevalent examples is the growth-
1461 defense trade-off in plant-microbe interactions. Under changing environmental conditions, and
1462 when resources are scarce, plants must decide between growth or defense. Growth-defense trade-
1463 offs are triggered by changes in the nutrient status and by the activation of pathways with
1464 contrasting functions, e.g., either promoting or limiting growth. Thus, the trade-off between growth
1465 and defense has an enormous impact on plant survival, reproduction, plant fitness, and crop yields
1466 (Huot et al. 2014). As such, it is not surprising that plant hormones, transcription factors, and
1467 kinases that sense the nutrient status of a cell, all play roles in balancing growth-defense trade-offs
1468 (Huot et al. 2014, Lozano-Durán et al. 2013, De Vleesschauwer et al. 2018, Margalha et al. 2019).
1469 Proteostasis, the balance between protein biosynthesis and degradation, has a huge impact on the
1470 growth-defense trade-off. Considering that approximately 80% of protein degradation in plants is
1471 mediated by the UPS, and given the role of the UPS in plant-microbe interactions, cell survival, and
1472 growth (Langin et al. 2023a, Raffeiner et al. 2023), it is evident that the UPS plays a major role in
1473 the growth-defense trade-off. How does the proteasome directly and indirectly influence the trade-
1474 off between growth and defense?

1475 ***The role of the UPS in cellular homeostasis***

1476 The UPS not only recycles proteins as a housekeeper but also has essential roles in controlling
1477 developmental processes and stress responses by fine-tuning the amount of central regulatory
1478 proteins (Raffeiner et al. 2023). Proteasome mutants often display pleiotropic phenotypes, perhaps
1479 related to the role of the UPS in central processes in plant growth and development, such as
1480 balancing cell division and expansion in plants (and see the section above by Bednarek), and plant
1481 response to environmental conditions, including biotic and abiotic stresses (Langin et al. 2023a,
1482 Kurepa et al. 2009, and see above section by Bassham).

1483 Mounting evidence suggests that the UPS is involved in degrading organelle-associated
1484 proteins to alleviate stress conditions (Clavel and Dagdas 2021, and see above sections by
1485 Murcha and van Wijk). As chloroplasts are the most essential energy source in plants, impairment
1486 of proteasome-mediated chloroplast quality control can be expected to have a dramatic effect on
1487 plant fitness. Indeed, severe proteasome stress induced by high concentrations of the proteasome
1488 inhibitor MG132 inhibits root growth (Sheng et al. 2012). However, mild proteasome stress was
1489 shown to enhance root growth (Sheng et al. 2012) and can also have a positive effect on
1490 photosynthesis and plant performance (Grimmer et al. 2020), suggesting that we are missing some
1491 puzzle pieces in our understanding of the role of the UPS in the control of plant growth.

1492 ***Why is the proteasome manipulated during plant-microbe interactions?***

1493 The proteasome might be considered an ideal target for manipulation by microbes to impact as
1494 many pathways and compartments as possible (Langin et al. 2020, 2023a). The proteasome
1495 controls plant immune reactions from pathogen perception to execution and thus is a master
1496 regulator of plant immunity (Langin et al. 2023a, Adams and Spoel 2018). It is known that loss of

various proteasome subunits leads to increased susceptibility towards pathogens (Langin et al. 2023a, Üstün et al. 2016). The inactivation of the proteasome seems to be in general beneficial for most pathogens, although it leads to growth penalties and developmental alterations. Thus, various pathogens, from bacteria to viruses, directly target and inactivate the proteasome to subvert many cellular processes (Langin et al. 2020).

However, there are also contrasting effects on the proteasome during plant-microbe interactions. Although certain pathogens suppress the function of the proteasome to cause disease, the same pathogens also activate the proteasome to degrade central regulators of plant immunity (Langin et al. 2020). How these contrasting functions work together to influence the proteasome remains to be understood but inactivation and activation likely occur in parallel during pathogen attack. Nevertheless, in a simplified scenario, proteasome activation might be the way to combat disease. Indeed, during various pathogen infections or SA treatment, transcription as well as translation of proteasome subunits are induced, which can be explained in two ways: (i) Given the direct role of some subunits in plant defense reactions (Hatsugai et al. 2009, Üstün et al. 2016) proteasome subunits are transcriptionally and translationally induced as a form of defense reaction or (ii) pathogens directly induce the expression of the proteasome to hijack the proteasome. If we think about scenario (i) strengthening the proteasome should lead to resistance without affecting growth. Intriguingly, a recent study discovered a natural allele of proteasome maturation factor UMP1, displaying enhanced proteasome abundance and activity, leading to resistance to multiple pathogens in rice (Hu et al. 2023). While pathogen infection is restricted, defense reactions are increased without any yield penalty. Taken together, activation of the proteasome circumvents the growth defense trade-off. Uncoupling growth and defense trade-offs have been shown in very rare cases, e.g., regulated expression of SA master regulator NPR1 using uORF-mediated translational control (Xu et al. 2017). It appears that utilizing proteostasis seems to be the key to engineering plant disease resistance without fitness costs.

Does proteasome activation balance growth-defense trade-offs?

But would proteasome activation always bypass the growth-defense trade-off? Activation of the proteasome is governed by a chaperone network, including UMP1, that is essential for proteasome assembly and function. Before this, proteasome subunit genes need to be expressed (Marshall and Vierstra 2019). In Arabidopsis, two NAC transcription factors, NAC53 and 78, act in concert to activate the gene expression of proteasome subunits (Gladman et al. 2016). Considering the broad role of NACs in many cellular pathways, it is likely that both transcription factors might have other targets beyond the proteasome. Indeed, both transcription factors have been found to additionally target and repress photosynthesis-associated nuclear genes during proteotoxicity affecting the energy status of the cell (Langin et al. 2023b). The trade-off between proteasome activation and photosynthesis downregulation seems to be a general feature as it occurs in response to various environmental and developmental cues (Langin et al. 2023b). In this scenario, transcriptional upregulation of proteasome subunits might be considered as a defense strategy to restrict pathogens by repressing photosynthesis when pathogens suppress the proteasome (**Fig. 15**).

1536 Although the role of the proteasome in growth and defense has been extensively studied, many
1537 questions remain elusive: 1) Does the magnitude of proteasome activation and de-activation
1538 decide how the growth-defense trade-off is influenced? 2) Does the proteasome act as a trap in
1539 plant-microbe interactions leading to growth penalties to limit pathogens? 3) How can we explain
1540 contrasting effects on the proteasome during pathogen infection? 4) Can we engineer plants that
1541 evade the growth-defense trade-off using the transcriptional activation of the proteasome by
1542 NAC53/78? Addressing these questions in the future will reveal how up- and down-regulation of
1543 the proteasome during plant-microbe interactions integrates different signals to balance the trade-
1544 off between growth and defense.

1545 Why are there so many peptidases in plants, particularly in the subtilase family?

1546 (By Annick Stintzi and Andreas Schaller)

1547 Plants devote a large fraction of their proteome to proteolysis. Adding to the proteasome and the
1548 multi-component UPS, there are several hundred peptidases in plants, for example, 685
1549 peptidases in Arabidopsis (MEROPS database https://www.ebi.ac.uk/merops/). Here, we discuss
1550 whether the expansion of peptidase families is driven by functional diversification or, more
1551 specifically, by the specialization of peptidases for certain substrate proteins, for specific
1552 processing sites, or by distinct mechanisms of regulation. We pose these questions for subtilases
1553 (SBTs), the S8 family of serine peptidases and one of the largest and most studied peptidase
1554 families in plants (Schaller et al. 2018).

1555 *Specialization for substrate proteins*

1556 Plant-specific SBTs (five of the seven SBT clades in tracheophytes) originated from a single event
1557 of horizontal gene transfer from a bacterial donor to streptophyte algae (Xu et al. 2019). Early gene
1558 duplication resulted in two copies, one evolving into the *SBT2* clade, the other one ancestral to
1559 clade1 and clades 3-5. While the *SBT2* lineage remained well-conserved with low copy numbers
1560 throughout land plant evolution, the *SBT1* and *SBT3-5* lineages underwent massive expansion (Xu
1561 et al. 2019). Interestingly, the size of individual clades differs dramatically between angiosperm
1562 taxa, suggesting that some of the gene duplication events occurred comparatively recently.

1563 The *SBT1* clade, for example, is rather small in Arabidopsis with only 9 members, compared to
1564 61 in tomato (*Solanum lycopersicum*) and an average of 21.7 across land plants (Taylor and Qiu
1565 2017, Reichardt et al. 2018). Many *SBT1* genes have been implicated in biotic interactions,
1566 including symbiotic interactions (e.g., arbuscular mycorrhiza and nodulation) as well as pathogenic
1567 interactions with viruses, microbes, insects, and parasitic plants. It was proposed that the *SBT1*
1568 clade expanded by whole-genome and tandem duplications followed by neo-functionalization in
1569 response to the selection pressure from interaction partners (Taylor and Qiu 2017). This would
1570 explain the smaller size of the *SBT1* clade in Arabidopsis, a non-mycorrhizal and non-nodulating
1571 species. Neo-functionalization implies the specialization of SBT paralogs for substrate proteins

1572 specifically involved in the different biotic interactions. However, these substrate proteins have yet
1573 to be identified.

1574 Direct evidence for the diversification of clade 1 *SBTs* in response to pathogen pressure was
1575 obtained for the cluster of 10 monophyletic *P69* genes on tomato chromosome 8, with individual
1576 paralogs contributing to plant defense against different pathogens (Homma et al. 2023, Zhang et al.
1577 2024). Host immune responses rely on P69B which activates the immune protease Rcr3 (Paulus et
1578 al. 2020). Many pathogens produce effector proteins inhibiting P69B to suppress immunity (Homma
1579 et al. 2023). Responding to the resulting selection pressure, paralogs evolved within tomato and
1580 across related Solanum species that show variation mainly at residues located at the
1581 protease/inhibitor interface, thereby escaping effector-mediated P69 inhibition (Homma et al. 2023).

1582 Neo-functionalization with respect to substrate specificity is apparent in the *SBT3* clade, which is
1583 much larger in Arabidopsis, with 18 members compared to only 2 in tomato (Reichardt et al. 2018).
1584 Many of the Arabidopsis *SBT3* copies are found as clusters of tandemly arrayed, monophyletic
1585 paralogs indicating that they originate from tandem duplications in the Brassicales or Brassicaceae
1586 (Taylor and Qiu 2017). Substrates of clade 3 SBTs include PROSCOOPs, a large family of
1587 phytocytokine precursors. PROSCOOP12 and PROSCOOP20 are processed by different SBT3
1588 members to release the corresponding bioactive SCOOP peptides (Yang et al. 2023). Interestingly,
1589 the PROSCOOP family is also restricted to Brassicaceae (Gully et al. 2019), suggesting that co-
1590 evolution with PROSCOOPs may have contributed to the expansion of the SBT3 clade.

1591 ***Specialization with respect to mechanisms of regulation***

1592 Proteases must be tightly controlled, due to their irreversible impact on structure and function of
1593 substrate proteins. This is achieved by diversification with respect to the developmental stage and
1594 cell type in which they occur, the subcellular compartments and conditions under which they are
1595 active, and the mechanisms by which proteolytic activity is terminated. SBTs show highly tissue-
1596 specific and/or stress-responsive expression patterns, likely resulting from sub- or neo-
1597 functionalization at the level of gene regulatory elements. They are produced as pre-pro-enzymes
1598 with an N-terminal signal peptide targeting the proteins to the secretory pathway. The prodomain
1599 serves dual functions, acting as a folding assistant and as an auto-inhibitor of enzymatic activity
1600 (Meyer et al. 2016). Cleavage of the prodomain is critical for enzyme activation and is an
1601 autocatalytic process. In tomato SBT3 it is controlled by pH (Meyer et al. 2016) and occurs late in the
1602 secretory pathway when the pH drops in the trans-Golgi, thereby preventing precocious enzyme
1603 activation. Since SBT activity is required also in earlier compartments of the secretory pathway, as
1604 well as in the apoplast (Stührwohldt et al. 2020b), other regulatory mechanisms for prodomain
1605 processing are likely to exist. Some SBTs are kept inactive, even after prodomain removal, by a self-
1606 inhibitory N-terminal peptide or a flexible β-hairpin occluding the active site. These SBTs require
1607 further processing and homodimerization, respectively, for activation (Janzik et al. 2000, Ottmann et
1608 al. 2009). SBTs also diversified with respect to the pH optimum for catalysis, ranging from pH 4 to 11.
1609 How all these factors relate to physiological function is still largely unresolved.

1610 Protease activity can be terminated by inhibition, degradation, or sequestration. In addition to
1611 pathogen-derived inhibitory effector proteins (Homma et al. 2023), there are also endogenous SBT
1612 inhibitors that are related in structure and function to the SBT prodomain (Hohl et al. 2017). In
1613 Arabidopsis this includes SPI-1, a potent inhibitor with inhibition and dissociation constants in the
1614 picomolar range (Hohl et al. 2017). However, which SBTs are targeted in vivo, and the
1615 physiological consequences of SPI-mediated inhibition, remain to be identified. The activity of
1616 AtS1P in clade SBT7 is regulated by the Serpin1 inhibitor (Ghorbani et al. 2016). Whether plant-
1617 specific SBTs in clades SBT1-5 are inhibited by other members of the large serpin family is still
1618 unknown. As other means of regulation, tomato subtilase P69B is degraded by two matrix
1619 metalloproteinases (Zimmermann et al. 2016), and active phytaspase (AtSBT3.8) is specifically
1620 removed from the apoplast by clathrin-mediated endocytosis (Trusova et al. 2019).

1621 ***Specialization for specific processing sites***

1622 An emerging function of plant SBTs is their predominant role in the formation of peptide hormones
1623 and growth factors, particularly of the post-translationally modified signaling peptides as
1624 extracellular signals for cell-to-cell communication (Stührwohldt and Schaller 2019, Stintzi and
1625 Schaller 2022). The vast numbers of signaling peptides and SBTs in the plant apoplast suggest
1626 that mechanisms are in place both to prevent unwanted degradation of signaling peptides and to
1627 ensure the specificity of peptide precursor processing. Such mechanisms may have arisen by co-
1628 evolution of the signaling peptide and SBT families.

1629 To ensure the specificity of precursor processing, SBTs evolved different modes of cleavage
1630 site recognition. Precise processing of the IDA precursor *(*a peptide controlling floral organ
1631 abscission*)* depends on multiple residues on either side of the cleaved bond. These residues are
1632 accommodated in the active site cleft of SBT4.13 with low selectivity, and it is the sum of many
1633 low-affinity interactions that ensures precise recognition of the cleavage site (Schardon et al.
1634 2016). In contrast, the precursors of systemin and PSK (peptides controlling herbivore defense and
1635 flower drop in tomato, respectively) display aspartate residues at their processing sites.
1636 Phytaspases recognize these single aspartates in a highly specific manner, showing little
1637 selectivity for other residues around the cleavage site (Beloshistov et al. 2018, Reichardt et al.
1638 2018, Reichardt et al. 2020). The TWS1 precursor (TWS1 is a peptide controlling embryonic cuticle
1639 development*)* is processed by SBT1.8 and SBT2.4, which act redundantly at the C-terminal
1640 cleavage site (Doll et al. 2020, Royek et al. 2022). Interestingly, SBT1.8 also cleaves at the N-
1641 terminus, but only when a neighboring tyrosine is sulfated. In this case, post-translational
1642 modification of this tyrosine marks the cleavage site for recognition by SBT1.8 (Royek et al. 2022).
1643 The opposite was observed for the precursor of CLE40 *(*a peptide controlling stem cell
1644 maintenance in the root apical meristem*)*. proCLE40 is cleaved by three redundant SBTs at two
1645 sites, the first resulting in the release of the mature peptide, the second producing an inactive
1646 CLE40 fragment (Stührwohldt et al. 2020a). Here, post-translational hydroxylation of a neighboring
1647 proline prevents cleavage at the second site, thereby contributing to the specificity of processing
1648 and CLE40 biogenesis (Stührwohldt et al. 2020a). In these examples, different modes of substrate

recognition by the proteases, and post-translational modifications of the peptide precursors both contribute to the specificity of interaction.

Given the preceding discussion, we can only offer a partial explanation for why there are so many peptidases in plants. Hence, the question is still open, awaiting thorough investigations into the evolutionary forces that propel the expansion of SBT and other peptidase families.

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1687 **Table 1.** Comparison of characteristics of mammalian Skp2 and Arabidopsis FBL17.

Skp2 (mammals)	Characteristic	FBL17 (Arabidopsis)
yes	Expression during S-phase	yes
yes	Transcription targets of E2F/DP	Yes
Viable (mouse)	Loss-of-function	Gametophyte lethal (with sporophytic escapees)
Reduced cell proliferation	Cell cycle defects in mutants	Reduced cell proliferation
Increase in ploidy	Ploidy in null mutants	Suppressed endoreduplication
p21, p27, p57	CKI as targets	KRPs
Cyclins D1 and E, Cdt1, Orc1, Rbl2/p130, E2F	Other cell cycle targets	?
yes	Phospho-degron	?
yes	Requirements of Cks1 cofactor	?
K48, K63, …	Ub chain topology	?
Cdk2, Atk, Wee1	Phosphorylation	WEE1
APC/C^{Cdh1}	Degradation	APC/C^{CDC20}
ATM kinase activation and DNA DSB repair	Role in DDR	Replication stress and DSBs
Brca2, Nbs1,…	Targets in DDR	?

1688

1689

Figure legends

Figure 1. Regulation and possible roles of FBL17 in Arabidopsis. A) During the G1/S phase, E2Fa-DPa directly activates the transcription of *FBL17*. The degradation of KRPs by FBL17-mediated ubiquitylation and degradation would release the activity of CDKA/CYCD. Whether FBL17 recognizes its substrate via a phospho-degron is unknown. **B)** During replication stress, WEE1 phosphorylates FBL17 and the APC10 subunit of the APC/C, which promotes FBL17 ubiquitylation and degradation by the proteasome.

Figure 2. Proteasome- and vacuole-dependent protein degradation in the plant DDR. After DNA damage, a plant cell launches a DNA damage response (DDR), which includes the transcriptional upregulation of certain genes and the targeted degradation of proteins. The two major protein degradation systems, the UPS and the vacuole-dependent system (autophagy) both appear to be involved in the DDR. The diagram shows a prototypical proteasome target (green X). Under non-stress conditions (grey arrows), X is marked by K48 polyubiquitin chains and subsequently degraded by the proteasome. After DNA damage, X becomes stabilized and participates in the DDR. Examples of X are the transcriptional repressor of cell proliferation MYB3R3 and the RMI1 removing factor KNO1. Another and previously not DDR-associated degradation pathway is shown for the orange-marked protein Y, a protein that interferes with efficient DDR. Under non-damaging conditions, Y is present. Under damaging conditions, Y is polyubiquitylated via K63 chains marking it for autophagy-dependent degradation via the cytoplasm. The only example for Y so far is the RTR-complex scaffolding component RMI1, which becomes polyubiquitylated via K63, targeting it for removal to the cytoplasm and degradation via autophagy. The observation that mutants in the macroautophagy pathway (such as ATG mutants) are sensitive to various DNA-damage-inducing drugs indicates that macroautophagy also degrades other proteins after DNA damage and likely plays a major role in the DDR of plants.

Figure 3. Proteolytic networks across different plant mitochondrial compartments. Protein quality control (PQC) (red dotted lines) that includes the disassembly, unfolding, and degradation of proteins and complexes is carried out by various proteases as indicated. PQC is carried out at various pathways including protein import, in organello translation, and assembly. Preproteins are imported from the cytosol through the translocase of the outer membrane (TOM) complex. Preproteins targeted to the outer membrane (OM) and the intermembrane space (IMS) undergo PQC by various IMS-facing proteases, such as overlapping with m-AAA protease OMA1 and rhomboid-like (RBL) protease. Preproteins targeted to the inner membrane (IM) and the matrix are translocated by the translocase of the inner membrane (TIM) complex. Preproteins then undergo the maturation process to remove the N-terminal targeting signal and destabilizing residues by mitochondrial processing peptidase (MPP) embedded within Complex III (CIII) in plants, octapeptidyl peptidase OCT1, and intermediate cleavage peptidase ICP55. Proteins assembled within a complex, such as the oxidative phosphorylation (OXPHOS) supercomplex SC I+III$_2$+IV (or the respirasome) may undergo PQC by the IM-embedded proteases like the matrix-facing FTSH3/FTSH10 and the IMS-facing FTSH4/FTSH11. Nascent polypeptides translated by the mitoribosome may be regulated by FTSH3/10, while PQC of matrix-facing and matrix-located proteins is regulated by the matrix-located CLPP2 and LON1 proteases. Free peptides generated from both targeting peptide cleavage and protein degradation (blue dashed lines) can be further degraded into single amino acids in the peptide processing pathway. This multi-step pathway uses multiple peptidases, including

1732 presequence peptidases (PREP), organellar oligopeptidase (OOP), and various aminopeptidases
1733 (AP), including alanyl aminopeptidase (AAP), leucyl aminopeptidase (LAP), aspartyl aminopeptidase
1734 (DAP), and prolyl aminopeptidase (PAP).

1735 **Figure 4. The relationship between NTA and protein stability. A)** The conditionality of Ac/N-
1736 degrons and their link to protein quality control. Acetylated (Ac) N-termini are often shielded through
1737 internal protein folding (i) or protein-protein interactions (ii) but can be exposed through protein
1738 misfolding or if there is an excess of a particular protein complex subunit. This leads to exposure of
1739 the acetylated N-terminus, which can act as a specific degron for proteasomal degradation via the
1740 Ac/N-degron pathway (Shemorry et al. 2013). **B)** Hypothetical indirect effects of NTA on protein
1741 stability. NTA can increase protein-interaction affinities, to create more stable complexes. A lack of
1742 NTA can lead to reduced thermostability, complex breakdown, and the consequent degradation of
1743 non-bound and potentially misfolded subunits via then UPS (e.g., as has been shown for cytosolic
1744 ribosomes in yeast; Guzman et al. 2023). **C)** NATA-mediated NTA (potentiated by HYPK in plants
1745 and mammals) was shown to promote broad proteome stabilization in diverse eukaryotic taxa. In
1746 plants, drought-induced downregulation of NATA activity leads to reduced NTA of NATA substrates
1747 and an increase in their degradation via exposed ‘non-Ac/N-degrons’ (Linster et al. 2015, 2022).
1748 This suggests that NATs may integrate stress signals to control proteome turnover.

1749 **Figure 5. Enzymes and substrates of plant Arg/N-degron pathways. A)** Schematic of N-recognins
1750 in plants and mammals showing substrate recognition and E3 ligase domains (drawn approximately to
1751 scale; hRING, hemi-RING). **B)** Destabilizing residues and specificity of Arabidopsis N-recognins,
1752 deduced from model substrates (Garzón et al. 2007, Graciet et al. 2010). In these examples, Ub fusion
1753 protein is cleaved in planta by deubiquitylating enzymes (DUBs) to produce glucuronidase (GUS) or
1754 luciferase (LUC) with Nt destabilizing residues (shown in single letter amino acid code). Proteins with
1755 primary destabilizing residues (R,F,L) are targeted for proteasomal degradation by known and unknown
1756 N-recognins. Tertiary destabilizing residues N and Q are converted to D and E by Nt(Asn) amidase
1757 (NTAN) and Nt(Gln) amidase (NTAQ) respectively. These secondary destabilizing residues are then Nt-
1758 arginylated by arginyl-tRNA-transferases (ATEs), enabling PRT6-mediated degradation. **C)**
1759 Physiological substrates of the Arg/N-degron pathway. Met-Cys-initiating proteins VRN2, ZPR2 and
1760 ERFVIIs undergo co-translational Met excision by methionine amino peptidases (MetAPs) to reveal Nt
1761 Cys, a tertiary destabilizing residue. Following Nt oxidation by plant cysteine oxidases (PCOs) and
1762 arginylation by ATEs, they become substrates for PRT6. BIG also mediates the degradation of ERFVIIs
1763 and VRN2.

1764 **Figure 6. Schematic view of the N-degron pathway for degradation by the chloroplast Clp**
1765 **chaperone-protease system.** Proteins can be converted into substrates for the Clp system by
1766 various events including protein complex disassembly and aggregation, different stresses such as
1767 heat and radical oxygen species (ROS) or through metabolic feedback (e.g., known to occur in the
1768 chlorophyll synthesis pathway). These changes to proteins can result in the generation of a
1769 degradation signal known as a degron, either by simply exposing (“unmasking”) the N-terminus of
1770 the protein or by a post-translational modification (PTM). Examples of such PTMs are
1771 phosphorylation, acetylation, oxidation, or the addition of an amino acid to the N-terminus. This N-
1772 degron is then recognized by the ClpS1 recognin (and possibly also ClpF), which delivers the bound
1773 substrate to the ClpC or ClpD chaperones for ATP-dependent unfolding and concommittant threading

1774 into the Clp protease complex. The unfolded substrates are degraded within the Clp proteolytic
1775 chamber resulting in release of degradation products in the form of small peptides. However, the in
1776 vivo nature of these chloroplast N-degrons is yet to be determined. Elucidation of these N-degrons
1777 and the molecular players involved in their generation and recognition is a major challenge to be
1778 addressed.

1779 **Figure 7.** Regulation of light signaling components through the UPS. Photoreceptors, various light-
1780 promoting transcription factors (inside the yellow circle), and light repressors (inside the blue circle)
1781 are regulated by UPS through indicated CRL E3 ligase complexes.

1782 **Figure 8. Proteolysis of core ABA signaling components occurs in different subcellular**
1783 **compartments.** The inset shows that ABA is perceived through dimeric or monomeric receptors
1784 (blue), which triggers the formation of ternary complexes with clade A PP2Cs (red), and relief of
1785 inhibition of SnRK2.2/2.3/2.6 (pink) kinase activity. Nuclear, cytosolic and PM targeting pathways of
1786 core components are indicated. RSL1 illustrates the targeting of ABA receptors at the PM, which
1787 promotes endosome-mediated vacuolar degradation via the ESCRT machinery, whereas PUB12
1788 and AIRP3 might target PP2Cs in the proximity of the PM and follow either cytosolic or vacuolar
1789 degradation pathways. Nuclear degradation of ABA receptors, PP2Cs, and OST1 involves the
1790 multimeric CRL3, CRL4, and RING-type COP1 E3 ligases, among others (see text for details).
1791 Nuclear and cytosolic 26S proteasomes and the vacuole participate in the degradation of core
1792 signaling components, which might influence signaling, desensitization, or resetting of the ABA
1793 pathway. The lytic vacuole also receives cargo for degradation via autophagy but data linking ABA
1794 with autophagy are limited.The figure was created using BioRender (https://biorender.com).

1795 **Figure 9. Regulation of SL signaling through protein degradation** (i) SL (light pink) is
1796 perceived by the receptor D14 (blue). (ii) The activated SL receptor then binds to D3/MAX2 F-box
1797 (grey) as part of the $SCF^{D3/MAX2}$ complex. The presence of citrate or citrate-like molecules (red)
1798 triggers a conformational change in the C-terminal helix, CTH (red) of D3/MAX2 (grey), causing it
1799 to dislodge. (iii)The dislodged CTH of D3/MAX2-D14 complex subsequently loads the
1800 transcriptional repressor, D53/SMXLs (purple), through its D2 domain leading to polyubiquitylation
1801 (Ub, yellow). The binding of D53/SMXLs reactivates the hydrolysis of SL by D14 either during or
1802 after the polyubiquitylation of D53/SMXLs. (iv) The hydrolysis of SLs triggers a conformational
1803 change of D14 and restores the CTH of D3/MAX2 to its engaged conformation and subsequently
1804 triggers the release of polyubiquitylated D53/SMXLs from the D14-D3/MAX2 to proteasomal
1805 degradation. (v) D14 undergoes ubiquitylation and proteasomal degradation completing the
1806 feedback regulation of the SL signaling cascade. Interestingly, before D53/SMXLs are released to
1807 the 26S proteasome, their transient interaction with D3 alters D14 inhibition and gradually restores
1808 SL hydrolysis (Shabek et al. 2018, Tal et al. 2022). This restored activity can effectively “reset” the
1809 SL signal by depleting the hormone and degrading the D14 receptor until the next cue. This E3
1810 ligase domain plasticity provides an additional level of signaling control and represents a unique
1811 mode of targeting substrates for proteasomal degradation in the realm of phytohormone and UPS
1812 signaling.

1813 **Figure 10.** Interplay between carbon-containing metabolites and autophagy.

1814 **Figure 11. Fruit Plastids evolution along ripening progression and their possible delivery to**
1815 **vacuoles. A-E)** Transmission electron micrographs of ultrathin sections of tomato pericarp cells
1816 from three ripening stages as indicated. **A)** Chloroplast (arrow). **B)** Chromoplast (arrow). **C)**
1817 Vacuoles contain structures likely to be plastids (arrowheads) by judging the electron density of the
1818 cytoplasmic plastids in their vicinity (arrows in the inset). **D)** A chromoplast with signs of internal
1819 degradation (arrow). **E)** Vacuolar inclusions of what appear to be plastids remain (judging the
1820 internal plastoglobules). C, Cytoplasm. V, Vacuole lumen. Image credits: S. Mursalimov, A.
1821 Upcher, S. Michaeli.

1822 **Figure 12. The role of ERAD in plant growth, crop yield, and stress response. A**) The role of
1823 ERAD in stress response and phytohormone signaling in Arabidopsis. The leucine-rich repeat
1824 receptor-like kinase (LRR-RLK) CEPR2 phosphorylates ABA importer NRT1.2/NPF4.6, inhibiting its
1825 ability to import ABA. The phosphorylated NRT1.2/NPF4.6 is then transported to the ER for
1826 ubiquitylation and degradation, mediated by UBC32 and its homologs UBC33 and UBC34. ABA
1827 inhibits the CEPR2-mediated phosphorylation of NRT1.2/NPF4.6. ABA receptor PYL recognizes ABA
1828 and initiates the transduction of ABA signaling. UBC27 and AIRP3 act as an E2-E3 pair to activate
1829 ABA signaling and enhance drought tolerance by promoting the ubiquitylation and degradation of
1830 ABI1. Moreover, UBC32 collaborates with AtEMR1 to facilitate the degradation of misfolded BRI1,
1831 thereby influencing BR signaling under ER stress conditions. During drought stress, Rma1 and
1832 UBC32 work together to enhance drought tolerance by promoting the degradation of phosphorylated
1833 aquaporin PIP2;1. **B**) The role of ERAD in crop yield and disease resistance. The ERAD-related E2-
1834 E3 pair, OsUBC45/SiUBC32-DGS1/SGD1 in rice and millet, enhances yield by regulating BR
1835 signaling via distinct mechanisms. They enhance BR signaling by reducing the protein level of
1836 misfolded BR receptor BRI1 (in rice) or increasing the protein level of folded BRI1 (in millet).
1837 Additionally, the E2-E3 pair also promotes the Ub-dependent degradation of OsGSK3, a negative
1838 regulator of BR signaling. Under fungi attack, OsPIP2;1 facilitates the translocation of H_2O_2 from
1839 cytoplasm to apoplast, negatively regulating pattern-triggered immunity (PTI). OsUBC45 and DGS1
1840 promote the degradation of OsPIP2;1, enhancing rice resistance to disease.

1841 **Figure 13. Possible mechanisms of regulation of chloroplast-associated protein degradation.**
1842 CHLORAD is a UPS pathway that selectively degrades chloroplast-resident proteins, including the
1843 TOC apparatus that is responsible for protein import. The SP1 Ub E3 ligase recruits E2 Ub-
1844 conjugating enzyme (via its RING finger [RNF] domain) to direct the ubiquitylation of TOC proteins,
1845 which are then degraded through the combined action of the SP2 retrotranslocation channel, the
1846 CDC48 ATPase motor, and the cytosolic 26S proteasome (26SP); in this, the activity of SP1 is
1847 modulated by the action of SP1-like components (SPLs). Thus, CHLORAD exerts important control
1848 over protein import and the organelle's proteome and functions. Such control is responsive to
1849 developmental and environmental cues through unclear mechanisms. Under different conditions,
1850 phytohormone, Ca^{2+}, or reactive oxygen species (ROS) signaling might regulate the activity of
1851 CHLORAD, and this is possibly mediated through post-translational modification of the CHLORAD

1852 machinery or its TOC apparatus targets, and/or through retrograde signaling and stress-responsive
1853 proteins. Post-translational modifications that are potentially involved in such regulation are
1854 indicated.

1855 **Figure 14. Possible mechanisms by which autophagy regulates drought tolerance.** Autophagy
1856 is activated by drought stress via the TOR complex, SnRK1, and transcriptional pathways. Activation
1857 of autophagy may lead to increased degradation of protein aggregates and aquaporins, and
1858 decreased growth and stomatal aperture, in turn aiding tolerance of drought conditions.

1859 **Figure 15. The proteasome influences the growth defense trade-off. A)** The proteasome
1860 degrades substrates from various cellular compartments and organelles to maintain cell survival and
1861 optimal growth. **B)** Proteins that accumulate due to stress conditions or accumulating preproteins
1862 from organelles or microbes as well as chemical inhibitors can interfere with proteasome function
1863 leading to proteotoxic stress. This will cause growth penalties, impact survival, and in the case of
1864 microbes cause disease. **C)** On the one hand, a natural allele of proteasome maturation factor
1865 UMP1R2115 results in more proteasome abundance and activity improving resistance to multiple
1866 pathogens without growth penalties. On the other hand, proteasome activation can be achieved by
1867 the transcription factor pair NAC53 and NAC78. Whether this transcriptional activation of the
1868 proteasome results in resistance to pathogens and how it impacts plant growth remains to be
1869 discovered. The figure was created with BioRender (Biorender.com).

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1871 ## References

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