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Perception of Arabidopsis AtPep peptides, but not bacterial elicitors, accelerates starvation-induced senescence

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Members of the *At*Pep group of Arabidopsis endogenous peptides have frequently been reported to induce pattern-triggered immunity (PTI) and to increase resistance to diverse pathogens by amplifying the innate immune response. Here, we made the surprising observation that dark-induced leaf senescence was accelerated by the presence of Peps. Adult leaves as well as leaf discs of Col-0 wild type plants showed a Pep-triggered early onset of chlorophyll breakdown and leaf yellowing whereas *pepr1 pepr2* double mutant plants were insensitive. In addition, this response was dependent on ethylene signaling and inhibited by the addition of cytokinins. Notably, addition of the bacterial elicitors flg22 or elf18, both potent inducers of PTI, did not provoke an early onset of leaf senescence. Continuous darkness leads to energy deprivation and starvation and therewith promotes leaf senescence. We found that continuous darkness also strongly induced *PROPEP3* transcription. Moreover, Pep-perception led to a rapid induction of *PAO*, *APG7*, and *APG8a*, genes indispensable for chlorophyll degradation as well as autophagy, respectively, and all three hallmarks of starvation and senescence. Notably, addition of sucrose as a source of energy inhibited the Pep-triggered early onset of senescence. In conclusion, we report that Pep-perception accelerates dark/starvation-induced senescence via an early induction of chlorophyll degradation and autophagy. This represents a novel and unique characteristic of PEPR signaling, unrelated to PTI.

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