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Editorial

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Leaf Senescence and Pathogen Interaction: A Complex Balance Between Aging and Defense

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Introduction

Leaf senescence is a genetically programmed and highly regulated developmental process that marks the final stage of leaf growth. During senescence, nutrients such as nitrogen, carbon, and minerals are remobilized from aging leaves to developing organs like seeds and young tissues. While senescence is essential for plant fitness, it also influences plant–pathogen interactions. Many pathogens exploit senescing tissues to enhance infection, whereas plants may use senescence as a defense strategy to limit pathogen spread. Understanding the interaction between leaf senescence and pathogen attack is important for improving crop health and yield stability [1,2].

Discussion

Leaf senescence is controlled by complex signaling networks involving plant hormones, transcription factors, and environmental cues. Hormones such as ethylene, abscisic acid, and jasmonic acid promote senescence, while cytokinins delay it. Interestingly, these same hormones are also involved in plant immune responses, creating an overlap between senescence regulation and pathogen defense [3,4].

Pathogens often manipulate host senescence pathways to favor their own growth. **Necrotrophic pathogens**, which feed on dead tissue, actively induce senescence or cell death to access nutrients. They secrete toxins and effector proteins that accelerate chlorophyll degradation and membrane breakdown, weakening plant defenses. In contrast, **biotrophic pathogens**, which require living host cells, tend to suppress senescence to maintain a viable environment for prolonged infection [5].

Plants, however, can use senescence strategically as part of their defense. **Localized or premature senescence**, similar to the hypersensitive response, can restrict pathogen spread by depriving invaders of nutrients and limiting colonization. Senescing leaves often accumulate defense-related proteins, pathogenesis-related enzymes, and antimicrobial compounds that enhance resistance. Reactive oxygen species (ROS), which increase during senescence, also play a dual role by signaling defense responses and directly inhibiting pathogen growth.

At the molecular level, senescence-associated genes (SAGs) interact with immune-related genes through shared transcription factors such as WRKY and NAC proteins. These regulators coordinate nutrient recycling with defense activation. Recent research has shown that epigenetic modifications and small RNAs further fine-tune the balance between senescence progression and immune responses during pathogen attack.

Conclusion

Leaf senescence and pathogen interaction represent a dynamic balance between plant aging and defense. While pathogens may exploit senescence to enhance infection, plants can also harness senescence-related processes to restrict pathogen growth and redistribute resources efficiently. A deeper understanding of this interaction can support the development of crops with delayed senescence and enhanced disease resistance, contributing to improved yield, resilience, and sustainable agricultural systems.

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