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Chloroplasts as Targets for Pathogens: A Strategy for Defeating Chloroplast Immunity

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SUMMARY

Chloroplast is an indispensable organelle for plants, its function is more than capturing light and converting it to chemical energy. It also plays a crucial role in the arms race between pathogens and plants. Since chloroplast is of great significance to plant immunity, pathogens have evolved many strategies to break through the obstacles set by plants in order to achieve successful infection. They encode effectors to target chloroplasts to weaken the chloroplast-dependent defense, which is widely activated against pathogens.

INTRODUCTION

To circumvent and overcome chloroplast immunity for a successful infection, pathogens have evolved counter strategies. Pathogens produces effectors that directly act on chloroplast to suppress chloroplast mediated immunity, thereby enhancing their efficiency in causing disease. Some viruses have adapted to use the chloroplast envelope membranes as sites for their replication and in this way disturb normal chloroplast function. More commonly, viruses that do not replicate in association with chloroplast membranes encode proteins that interact with specific chloroplast proteins. Many of the identified targets of these viral proteins are part of the photosynthetic machinery.

Effectors Directly Targeting Chloroplasts

To date, there have been numerous reports of viruses interacting with chloroplast proteins to mitigate host defense. Understanding how viruses perturb chloroplasts has set the stage for further investigation into disruption of chloroplast function by other pathogens (Kachroo *et al*., 2021).

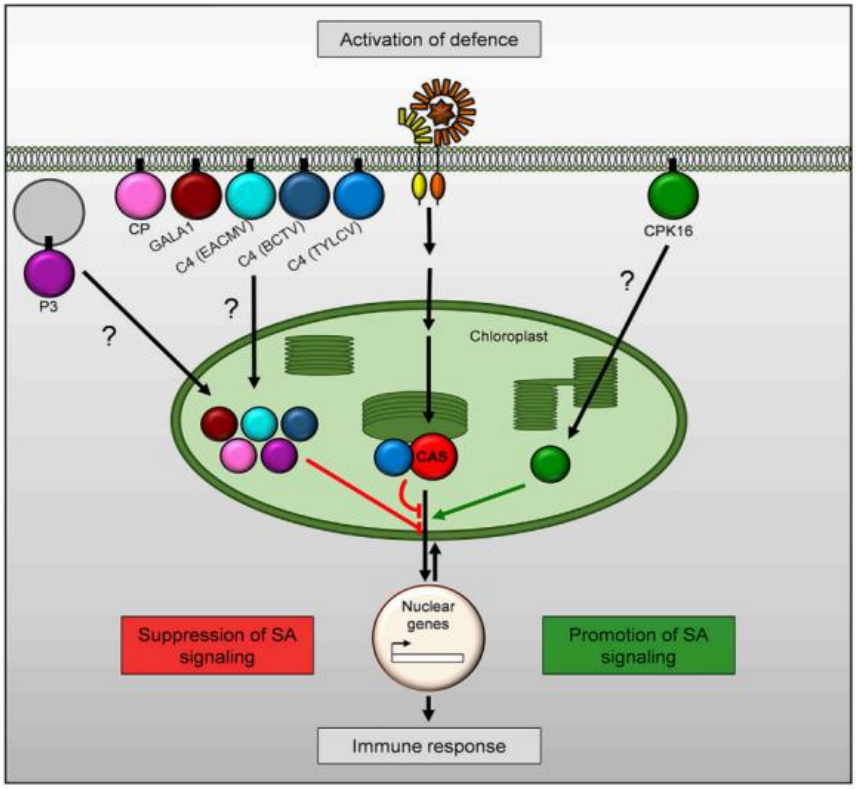

Fig.1: Model of the proposed pathway linking plasma membrane to chloroplasts and activating defense in plants and its co-option by plant pathogens to promote virulence through suppression of SA responses

One example of how the subcellular compartmentalization enabled by protein re-localization can give rise to multifunctionality is illustrated by the C4 protein encoded by TYLCV (Fig.1). At the Plasma membrane, C4 interacts with the receptor-like kinases BAM1 and BAM2 and hinders the intercellular spread of RNAi, following activation of defense, which is triggered by the presence or activity of the virus-encoded Rep protein, C4 is translocated to the chloroplast, where it interacts with CAS and interferes with the CAS-dependent defense responses, including SA biosynthesis. Interestingly, C4 seems to be retained at plasmodesmata, where it interacts with BAM1/2, even after being generally depleted from the PM following activation of defense ; this potential selectivity of the C4 pool undergoing re-localization could ensure that both functions of C4 are maintained throughout the different stages of the infection (Puche *et al*., 2020).

Remarkably, a large proportion of the *P. syringae* type III effector complement physically localizes to chloroplasts, including HopI1, HopN1, HopK1, AvrRps4, HopR1, and HopO1–2, although direct targets are only known for HopI1 and HopN1. The Ralstonia effector RipAL processes a DAD1-like lipase domain and localizes to chloroplasts, where it targets chloroplast lipids. Ectopic expression of RipAL suppressed PTI, inducing JA and JA-Ile as well as JA marker genes and concomitantly reducing both SA and SA-responsive gene expression. Accumulation of JA and suppression of SA were conditional on a functional DAD1-like lipase domain (Nakano *et al*., 2018).

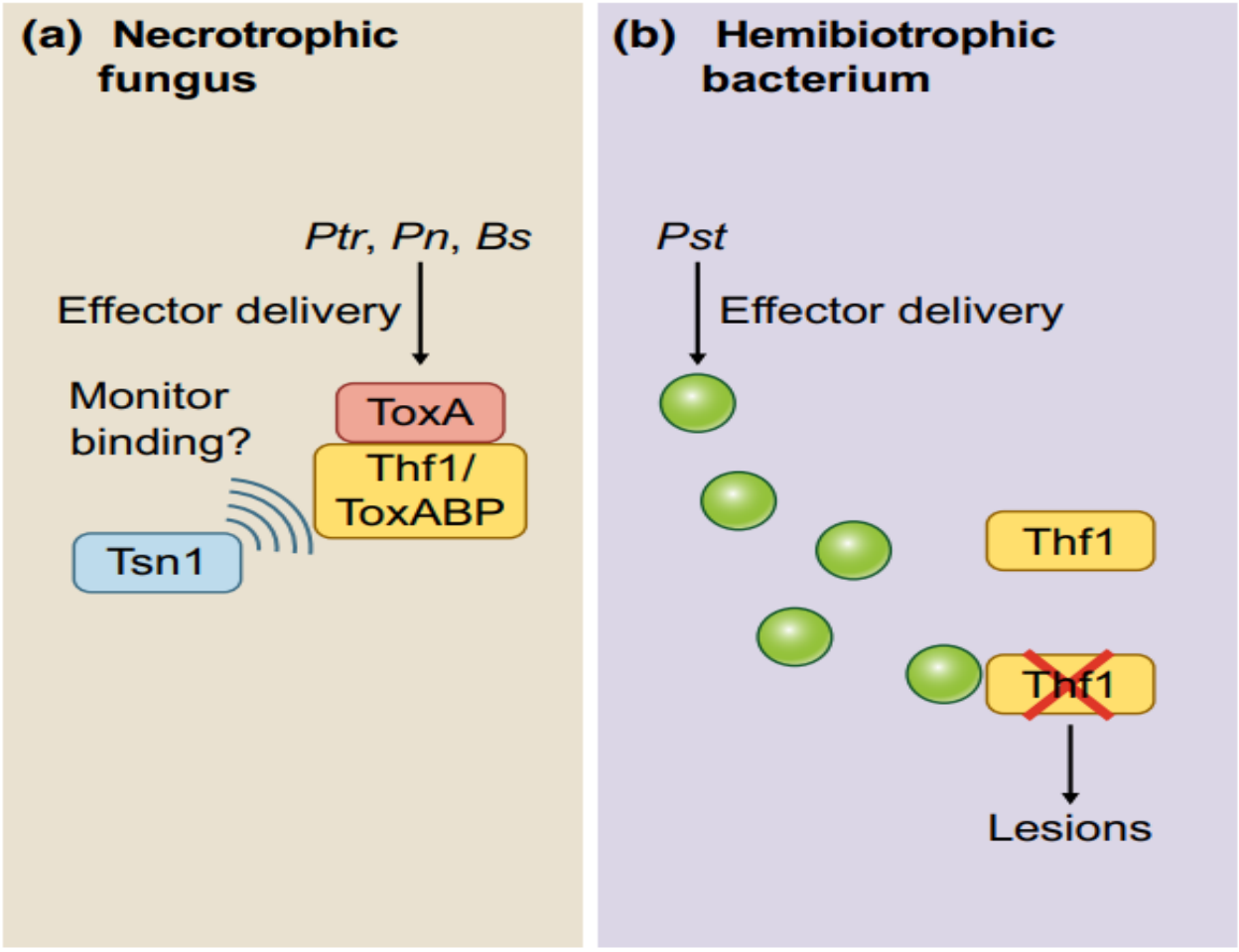

Fig.2: Convergent targeting of Thylakoid formation 1 (Thf1), a negative regulator of cell death, by diverse pathogens.

Thf1 plays an important role in photosystem II (PSII) – light harvesting complex II dynamics and is targeted by necrotrophs, biotrophs and viruses.

The effector protein ToxA found in a variety of necrotrophic wheat fungal pathogens, *Parastagonospora nodorum*(Pn), *Pyrenophora tritici-repentis*(Ptr) and *Bipolaris sorokiniana* (Bs), targets the wheat Thf1 orthologue, ToxA Binding Protein 1 (ToxABP), inducing necrosis via ROS accumulation through reduction in PSI and PSII protein complex abundance.

The hemibiotrophic bacterium *Pseudomonas syringae* pv. *tomato* (Pst) delivers effectors (yellow circles) which appear to disrupt Thf1 function, again leading to enhanced lesion formation, although it remains to be determined whether this is by direct or indirect interaction (Fig.2, Littlejohn *et al*., 2021).

CONCLUSION:

In recent years, an increasing number of studies have supported that chloroplast is not only the energy producer for plants but also plays a key role in plant defense against pathogen infection. Chloroplast-dependent immunity may be interfered by effectors released by pathogens, suppressing the chloroplast-mediated immunity to promote infection.

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