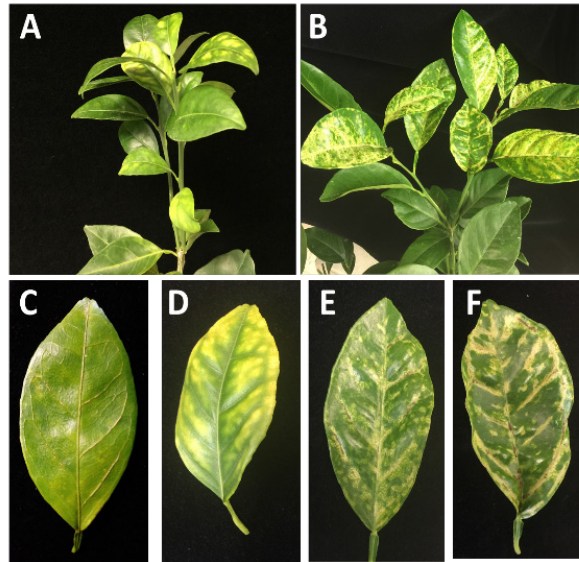


Creating a Model to Understand the Pathogenicity Mechanism of CLas

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HLB symptom-like produced in CTV-tPDS-t ALA plants.

A: CLas-infected plant with symptoms B: HLB-symptom like in the new shoots of CTV-tPDS-t ALA like infected plants. C&D: Vein corking and chlorosis in infected leaves. E&F:

Necrosis and chlorosis in CTV-tPDS-t ALA plant leaves.

The goal of this research is to better understand the pathogenicity mechanism of CLAs to facilitate other research on developing therapeutic strategies for HLB-diseased trees. The putative causal agent, CLAs, initiates a cascade of host plant responses which leads to leaf mottling and twig dieback and other symptoms. We aimed to identify the starting point of this cascade and whether the initiative signal originated from citrus as a response or from the bacteria as a bacterial metabolite. Our studies

led us to generate a hypothetical model for CLAs pathogenicity and the initial citrus defense responses. To test this hypothesis, we silenced genes encoding precursors of the citrus biosynthetic pathways most impacted and targeted by CLAs. Virus-induced gene silencing (VIGS) technology was utilized to silence the target genes. Interestingly, gene-silenced citrus plants showed a phenotype very similar to the typical symptoms of CLAs-infected trees. In addition, the phytohormones and other metabolites were altered in a

pattern similar with those of the infected trees. More importantly, we confirmed that the signal that induces the symptoms (threonic acid; previously identified) is produced by citrus as a response of the presence of CLAs and translocate via the vascular tissues. This approach of constructing a disease without its pathogen will greatly help in understanding the mechanisms of pathogenicity and provide a cleaner model for screening therapeutic compounds that may enhance plant immunity and disease tolerance.

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