Creating a Model to Understand the Pathogenicity Mechanism of *Candidatus* Liberibacter asiaticus

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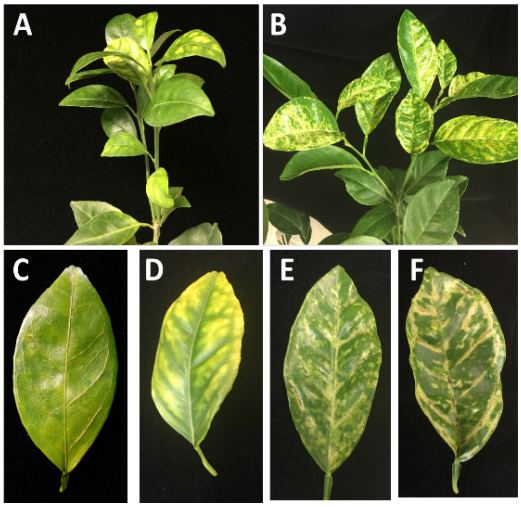
UF/IFAS CREC



HLB symptom-like produced in CTV-tPDS-tδALA plants.

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

Necrosis and chlorosis in CTV-tPDS-tδALA plant leaves.

Take Home Message:

- We created an HLB-mimic tree (appearance and physiology) by silencing genes encoding precursors of the citrus biosynthetic pathways most impacted and targeted by *CLas*.
- The model will be used to screen therapeutic compounds and proteins that enhance tree health.
- Screening for therapeutics that enhance tree health in the absence of the pathogen will allow us to better understand the bacterial pathogenicity to lead to a definitive control strategy.

Effort Statement: The model plant has been entirely created and proved to be an HLB-mimic tree. The trees' physiology is identical to those infected with *CLas*.

Summary: The goal of this research is to better understand the pathogenicity mechanism of *Candidatus* Liberibacter asiaticus (*CLas*) to facilitate other research on developing therapeutic strategies for huanglongbing (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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