The ABCs of SAR

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WHAT IS SAR?

Plants possess an array of defenses that can be actively expressed when challenged by diverse pathogens and non-pathogens. The timing and level of the plant's defense response is critical to determining the outcome of these interactions, which is largely dictated by the plant's ability to recognize the pathogen.

Systemic acquired resistance (SAR) is a state of enhanced resistance as a result of a primary, limited infection by a weak or incompatible pathogen. This type of plant resistance is regulated by the plant signal and salicylic acid, and is associated with numerous defense pathways, as evidenced by the expression of several genes and the accumulation of various antimicrobial compounds.

More than 100 years ago, plant researchers recognized that plants could be preconditioned against a variety of diseases. Early accounts exist of trees infected with a mild disease one season often being more tolerant to other more severe diseases in subsequent seasons, compared to those trees that weren't "challenged" in the first season. The debate at the time was whether this meant that plants had an immune system comparable to animals in what was then referred to as "acquired physiological immunity." Of course, we now know that plants don't possess anything that is strictly analogous to an animal immune system.

It wasn't until the late 1950s when virologists working with tobacco mosaic virus (TMV) demonstrated that not only could a localized infection of tobacco with TMV reduce disease to subsequent TMV infections on the same leaf, but even systemically to adjacent leaves. This observation led to the term, systemic acquired resistance.

Further research in various plant species (monocots and dicots) demonstrated that SAR could be initiated by diverse pathogens (fungal, bacterial and viral), and was in turn effective against the same or a different pathogen. That is, SAR induced by TMV in tobacco could confer protection against some bacterial or even fungal pathogens.

More importantly, researchers found that an induction period following initial pathogen challenge occurred during which resistance steadily increased. This period corresponded with the activation of various defense genes (Pathogenesis-related- or PR-genes are the most common) and the accumulation of metabolic products, including salicylic acid. In dicots, SAR is cyclical, with the resistance level of the plant slowly returning to the pre-SAR state over time. However, it is important to realize that SAR is only one part of the plant's overall defense repertoire.

Other inducible forms of plant resistance exist and act in concert with SAR. These other defense pathways are regulated in the plant by other signaling molecules, like ethylene and jasmonic acid, which interact with salicylic acid, leading to cross-talk among pathways. It is this cross-talk that best explains the dynamic responses plants are able to mount against pathogens and non-pathogens alike. Through the use of certain chemicals that mimic the effect of salicylic acid or stimulate the process that leads to accumulation of salicylic acid, SAR can be triggered in the absence of any biological agent.

PROPERTIES AND MISCONCEPTIONS ABOUT CHEMICALS THAT INDUCE SAR

Several chemicals can induce SAR, activating many of the same molecular genetic and biochemical processes in the plant that are naturally induced by salicylic acid. Chemical inducers also require a period of time between application and activation of the plant's defense mechanisms. The SAR inducer or its metabolites do not exhibit any direct anti-microbial activity as opposed to conventional pesticides which are toxicants and have direct activity on fungi and bacteria.

Two of the best-known SAR inducers are salicylic acid itself and isonicotinic acid, neither of which are used commercially. The most well-known commercial SAR inducer is the product acibenzolar-S-methyl (Actigard, Syngenta Crop Protection). Less well known inducers of SAR are neo-nicotinoid insecticides that are structural relatives of isonicotinic acid. The neo-nicotinoids imidacloprid (Admire, Bayer Crop Science) and thiamethoxam (Platinum, Syngenta Crop Protection) may be drenched into the soil, taken up by the roots and translocated to the leaves. When soil applied, these systemic insecticides provide several weeks' control of citrus leafminers (*Phyllocnistis citrella*) and psyllids (*Diaphorina citri*).

Other products commonly applied to citrus trees as ground applications or foliar sprays do not induce SAR, including various macro- and micronutrients or formulations of phosphites. Although phosphites have potent direct and indirect activity against Phytophthora infection, they do not control bacterial citrus diseases such as citrus canker caused by *Xanthomonas citri* subsp. *citri* (*Xcc*) or Huanglongbing (HLB, or yellow dragon disease or citrus greening) caused by *Candidatus* Liberibacter asiaticus (*Las*).

SOIL APPLICATION OF SAR INDUCERS IS KEY TO THEIR EFFECTIVENESS IN CITRUS

Soil applied neo-nicotinoids control canker in two distinct ways. One

mode of control is through minimizing wounds to the leaf cuticle by the leafminer larvae that promote *Xcc* infection. The second mode of canker control is by SAR induction which reduces bacterial infection and the population of *Xcc* in leaves by 10 to 100 times compared to untreated plants.

In field trials of 3- and 4-year-old Ray Ruby grapefruit trees, soil drenches of Admire, Platinum or Actigard were compared with foliar sprays of copper for percentage of canker-infected leaves on summer and fall flushes. SAR inducers applied once, twice or four times at 30- or 60-day intervals during the growing season were nearly as effective for disease control as 11 copper sprays applied at 21-day intervals. Highest control among SAR inducers was attained with four applications of Actigard during two different growing seasons. Based on efficacy for canker control, Syngenta and Bayer include a 2ee statement on the product labels for Admire Pro and Platinum 75 SG, indicating that these insecticides have activity against canker as soil applications for non–bearing citrus trees.

TRANSGENIC ENHANCEMENT OF SAR IN CITRUS

The *NPR1* gene in the model plant Arabidopsis (*AtNPR1*) is well established as a key regulator of SAR and other defense pathways. Overexpression of *NPR1* enhances resistance to bacterial pathogens in Arabidopsis and is associated with a faster or stronger induction of SAR. Transformation of Duncan grapefruit with *AtNPR1* confers resistance that is correlated with the expression of the gene regulator. The higher the level of *AtNPR1* expression in the transgenic plant, the smaller the number and size of lesions, and the greater the reduction in *Xcc* population. Hence, both chemical and genetic enhancement of SAR in citrus is demonstrated to reduce canker disease.

DOES SAR CONTROL THE SYSTEMIC PATHOGEN CAUSING HLB?

Intuitively, one might expect a systemically expressed resistance to provide control of the pathogen *Las* that infects the phloem from roots to fruits, leading to the disease HLB. However, SAR is most effective for control of non-systemic pathogens like *Xcc* that cause local lesions on leaves, stems and fruits. A recent study of *Las* genome expression by Nian Wang's lab at the Citrus Research and Education Center identified a salicylate hydroxylase (sahA) gene in *Las* which indicates the pathogen could potentially

break down salicylic acid and affect the salicylic acid induction process in *Las*-infected plants. The role that the pathogen may play in the breakdown of salicylic acid possibly explains the lack of response to SAR inducers and, therefore, deserves more study.

Thus far, in greenhouse and field trees treated with soil-applied Admire, Platinum or Actigard, we have not been able to demonstrate an effect of SAR on the development of PCR positive status or HLB symptoms compared to untreated trees. Likewise, screening of *AtNPR1* Duncan grapefruit and Hamlin orange plants with infected psyllids in collaboration with Mike Irey at US Sugar Corp. in Clewiston has not detected an effect of the transgene on the *Las* infection up until now. Finally, a field trial conducted by Bob Rouse at the Southwest Florida Research and Education Center has been evaluating the contribution of a salicylic acid product to the health response of HLB-affected trees on a foliar nutritional program. So far, no difference between trees treated with the salicylic acid product in combination with the nutritional program and those treated with the nutritional program alone has been observed. Hence, the conclusion to date is that salicylic acid does not appear to contribute to limiting the response of HLB-affected trees to *Las* infection.

RAMIFICATIONS OF SAR FOR MANAGEMENT OF HLB

If SAR does not reduce *Las* infection or the incidence and severity of HLB, then SAR-chemical-treated as well as unmanaged trees may act as a pathogen source for psyllid transmission, i.e., trees harbor inoculum of *Las* that is available for regional spread of the pathogen within and between groves. Thus, it is imperative if chemical treatments continue, that they be integrated with other methods for HLB management. These methods include planting disease-free nursery trees, participating in area-wide psyllid management (citrus health management areas or CHMAs), and where possible, surveying groves for HLB symptoms and removing infected trees as soon as feasible. The goal of an integrated program is to reduce inoculum sources as much as possible to reduce *Las* transmission and spread by psyllids.

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